Third Edition



Medicine and Surgery of CAMELIDS

MURRAY E. FOWLER



WILEY-BLACKWELL

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Murray E. Fowler, DVM

In collaboration with P. Walter Bravo, DVM, PhD, on Chapters 17 and 21

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A John Wiley & Sons, Inc., Publication

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Dedication

The third edition is once again lovingly dedicated to my wife Audrey, who has been an intimate participant in all of the editions. She has not only tolerated the hours spent away from the family but has read, corrected, and reread the manuscripts more times than either of us would like to remember. This revision could not have been brought to fruition without her help.

Preface to the Third Edition

The purpose of a preface is to provide an overview of the book. The second edition was published in 1998. The medical challenges that have arisen since then have kept pace with the advances made in veterinary medicine in general. West Nile virus encephalitis entered the United States and has become endemic in all the mainland states. Ionophore coccidiostat toxicity is new to camelids as is red maple, *Acer rubrum*, poisoning which heretofore has only been reported in horses.

The scope of this edition has broadened materially, as reflected in the new title. Information on Old World camelids has been added. While much of the anatomy and medical information is applicable to all camelids, where specific differences occur they will be discussed more fully.

The world literature has been scanned to provide an international approach to camelid medicine. Hundreds of new citations from the world literature provide details for the interested reader. References from sources that are less readily available to most readers have been deleted.

South American camelids are popular in the private sector in many countries in the world. They may become exposed to new infectious and parasitic diseases, such as West Nile virus disease, eastern equine encephalomyelitis, and encephalomyocarditis. In Europe, camelids have contracted Borna disease.

Camels suffer from a unique form of pox, which is the scourge of camels in their native countries. South American camelids may be experimentally infected, but are not known to be clinically infected naturally. Camels are also subject to surra (trypanosomiasis), which is caused by a protozoan parasite in countries where native camels exist.

The opportunities for continuing education in camelid medicine are numerous. Annual four-day workshops are now rotated between Ohio State University in Columbus and Oregon State University in Corvallis. Another conference is held at Kansas State University in Manhattan, Kansas. Sporadic seminars and conferences are held in conjunction with local camelid association meetings and at large regional veterinary conferences. Similar meetings are held in Australia, the United Kingdom, Ireland, and European countries.

The quantity and quality of research on camelid medical challenges have expanded exponentially. A significant milestone in camelid research is the completion of the alpaca genome project, which has great potential for genetic studies. Research studies on camelid medicine are reported in many professional journals. The *Journal of Camel Practice and Research* is devoted entirely to camelids.

Color has been added to the third edition, with more than 500 color images and 250 black and white photos and diagrams. The author's aim is to provide medical information on all camelids for clinical veterinarians, research scientists, students, and others interested in the health and well-being of these special animals that share this Earth with us.

Preface to the Second Edition

It has been ten years since the first edition of Medicine and Surgery of South American Camelids was written. Much has transpired during that time period. The population of llamas in North America has grown to over 120,000. Fewer than 200 llamas are exhibited in zoos, the rest being in private ownership. The astronomical prices paid for llamas a decade ago have dropped to a more reasonable level. Alpacas have become more popular, and over 2,000 animals have been imported from South America, bringing the North American alpaca population to greater than 10,000. Countries on other continents are experiencing a growth in South American camelids (SACs) as an alternate livestock enterprise; they include Australia, New Zealand, England, Scotland, France, and Germany.

Numerous regional and national organizations offer membership conferences, shows, sales, and other activities to maintain and stimulate interest in these unique animals. Llamas are now being used to guard flocks of sheep and to pack supplies and equipment for the U.S. Forest Service. Cottage industries making use of both llama and alpaca fiber abound. Camelid industries in North America are strong, and there is continued interest in learning about health care, hence the need for a second edition of this book.

Approximately 1,000 references from the world literature were cited in the first edition. In the intervening ten years the author has found another 1,300 references to add to that list. Considerable clinical experience has been gained by practitioners all over the world. A number of institutions are conducting basic investigations to solve problems. The camelid industries have contributed generously to fund research, but much more support is required to bring camelid veterinary medicine into the twenty-first century on a par with other livestock industries.

Articles appear regularly in professional veterinary journals. Books have been published dealing with medicine, reproduction, feeding, neonatology, and infectious and parasitic diseases. Veterinarians are no longer able to say that nothing is known about these animals. Likewise, owners should not say that veterinarians don't know how to treat the disorders of camelids. Not only are there books and professional journal papers but also numerous state, regional, and national veterinary meetings that offer half to two days of information on camelid medicine to attendees.

A two-day annual workshop on "the cutting edge of llama and alpaca medicine" has alternated between the veterinary schools at Fort Collins, Colorado, and Davis, California, with forty to seventy veterinarians attending. Other veterinary schools have offered special programs for veterinarians and, in some cases, for owners/breeders. Similar programs for veterinarians have been held in Australia, England, Scotland, Italy, France, and Germany. Veterinary schools in California, Colorado, Oregon, and perhaps other states have offered special elective llama and alpaca medicine and surgery courses to undergraduate veterinary students.

The format of the second edition is similar to that of the first, with two new chapters added on conformation and disaster management. Some chapters did not warrant much change, but others were revised significantly and brought up to date. Considerable basic research has been conducted on reproductive physiology during the past ten years. The author invited Dr. P. Walter Bravo to contribute to the chapter on reproduction. Topics that were omitted or glossed over superficially in the first edition have been expanded in keeping with newer knowledge that has been acquired.

As with the first edition, numerous individuals provided input in the form of inspiration, encouragement, shared clinical experiences, and basic research. A

second edition would not have been considered without the support of the reading public. Again, my special thanks to Dr. La Rue Johnson, a true friend, who has been willing to discuss philosophies, ideas, failures, and successes within a framework of mutual respect and admiration. I feel blessed to have shared so many good years with owners and managers of llamas and alpacas. I have learned from them and their animals. I hope this book will help to return the favor of friendship and support.

The basic stimulus for preparing this book has not been the owners/breeders or colleagues but the author's desire to contribute to the longer, healthier life of the versatile camelids of this world.

Murray E. Fowler

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1

General Biology and Evolution

The domesticated camelids of the world have had a significant impact on Old and New World cultures. Populations of camels and South American camelids (SACs), also called New World camelids, declined in the latter part of the nineteenth and early twentieth centuries. Governments neglected them as being an unimportant component of the life of indigenous people and tried to replace them with other domestic animals. Only in the last few decades have these animals been recognized as a valuable resource and efforts made to research their unique physiology and adaptation to hostile environments.[1,4]

TAXONOMY

Linnaeus placed the llama, alpaca, and Old World camels in a single genus, Camelus, in 1758. Other taxonomists proposed separate genus status for SACs in the early nineteenth century, but none of this work was accepted by the International Commission on Zoological Nomenclature. The genus name Auchenia was proposed by Illiger for SACs in 1811 and is frequently seen in print even today in the South American literature. However, Auchenia had been applied earlier to a genus of insects and thus was not a valid name for any other animal. In 1827, Lesson published an acceptable paper classifying the New World Camelidae in the genus Lama. In 1924, Miller assigned the vicuña to a separate genus, Vicugna.[13,14,17]

The systematic classification of Old World camels has never been controversial. The one-humped camel (dromedary) is named *Camelus dromedarius* (Figure 1.1) and the two-humped camel (Bactrian) *C. bactrianus* (Figure 1.2). Recently there has been acceptance of a third species of camel, the wild Bactrian camel of Mongolia, *Camelus bactrianus ferus*[12,14,21] (Figure 1.3).

The classification of SACs has been controversial. One system classifies the guanaco, llama, and alpaca within the genus Lama and vicuña as a single species in the genus Vicugna. Another system classifies all SACs within the genus Lama. Others classify the llama and alpaca as subspecies of L. *guanicoe guanicoe*. Recent DNA studies have determined that llamas and guanacos are appropriately species of *Lama*, and that the alpaca and vicuña belong in the genus *Vicugna*.[12]

The family Camelidae was previously designated as an infraorder, Tylopoda, under the suborder Ruminantia,[18,23,26] but the most authoritative and current classification gives Tylopoda suborder status.[8,10,17,22,25,28,29,30] Some modern taxonomists have deleted the suborder designation and use only family names. This book will follow the classification for camelids as listed in Table 1.1.

Collectively, llamas, alpacas, guanacos, and vicuñas are called South American camelids or New World camelids (NWC), although the term "auquenidae" is often found in older South American literature. Both camels and SACs are included in the term "camelid."

Alpacas and llamas exist only as domestic species. Guanacos and vicuñas are wild species. It is generally accepted that the alpaca shares some characteristics with the vicuña, e.g., incisor teeth with an open pulp cavity and continuous eruption into adulthood.

GENERAL BIOLOGY

The karyotype of all camelids is 2n = 74. All SACs have produced fertile hybrids. Dromedary and Bactrian camels also produce fertile crosses.[9] In Israel a cross between an alpaca male and a dromedary female by artificial insemination produced a stillborn full-term fetus. In the United Arab Emirates scientists have succeeded in producing four living OWC and NWC offspring (two males, two females). It has yet to be determined if the hybrids are fertile. See Chapter 22

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Figure 1.1. Dromedary camel.

Figure 1.2. Bactrian camel.

Figure 1.3. Wild Bactrian camel from the Gobi Desert in Mongolia. Used by permission from George B. Schaller, Wildlife Conservation Society.

Table 1.1. Classification of camelids and other artiodactylids.

Class—Mammalia
- Order—Artiodactyla
  - Suborder—Suiformes—Hippos, swine, peccaries
  - Suborder—Tylopoda—Camelids
- Old World genus and species
  - *Camelus dromedarius*—Dromedary camel
  - *C. bactrianus*—Bactrian camel
  - *C. bactrianus ferris*—Wild bactrian camel
- New World genera and species
  - *Lama glama*—Llama
  - *L. guanicoe*—Guanaco
  - *Vicugna pacos*—Alpaca
  - *V. vicugna*—Vicuna
    - *V. vicugna mensalis* (Peruvian)
    - *V. vicugna vicugna* (Argentine)
  - Suborder—Ruminatia—Cattle, sheep, goats, water buffalo, giraffes, deer, antelopes, bison

for illustrations (Dr. J. Skidmore, personal communication, Dubai, United Arab Emirates, April 2009).

Despite size differences, the anatomy of all species of camelids is similar. SACs have similar behavioral patterns (Chapter 3).[26] The camels completed Pleistocene evolution in a semidesert environment in southern Asia, the Middle East, and North Africa and developed sophisticated adaptations for dealing with heat and dehydration (Chapter 9). The SACs became adapted to South American habitats, especially the high altitude lands of the Andes.

Camelids have a complex, three-compartmented stomach. Gastric digestion is similar to, but not analogous with, ruminant digestion. The two suborders separated from each other 65 million years ago when primordial species were simple stomached. Both groups used fibrous forage and developed similar foregut fermentation systems by parallel evolution (Chapter 13). Camelids regurgitate and rechew ingested forage, as do ruminants, but are more efficient than ruminants in extracting protein and energy from poor quality forages (Chapter 2). It is important to understand that camelids are not ruminants, pseudoruminants, or modified ruminants. See Table 1.2 for other differences between camelids and ruminants.[5]

SACs establish communal dung piles, and in large herds there may be multiple dung piles, as animals in a herd segregate themselves into smaller units. Llamas and alpacas that have been imported into the United States following extended quarantine are more likely to urinate and defecate at random. Camels defecate at random. Feces are pelleted in both groups and used for fuel by people who share their habitat. Camelids have a unique reproductive cycle (Chapter 17).

Table 1.2. Differences between South American camelids and ruminants.

South American camelids	*Ruminants*
Evolution	
Evolutionary pathways diverged 40 million years ago	Evolutionary pathways diverged 40 million years ago
Blood	
Red blood cells elliptical and small (6.5 µ); predominant white blood cell is neutrophil; leukocytes up to 22,000	RBCs round and larger (10 µ); predominant WBC is lymphocyte; leukocytes up to 12,000
Foot	
Foot has toenails and soft pad. Second and third phalanges are horizontal.	Foot has hooves and sole. Second and third phalanges are nearly vertical
Digestive system	
Foregut fermenter, with regurgitation, rechewing and reswallowing	Same (parallel evolution)
Stomach—3 compartments, resistant to bloat	Stomach—4 compartments, susceptible to bloat
Dental formula—I 1/3, C 1/1, PM 1–2/1–2, M 3/3 × 2 = 28–32[a]	Dental formula—I 0/3, C 0/1, PM 3/3, M 3/3 × 2 = 32
Reproduction	
Induced ovulator	Spontaneous ovulation
No estrous cycle	Estrous cycle
Follicular wave cycle	No follicular wave cycle
Copulation in the prone position	Copulation in standing position
Placenta diffuse	Placenta cotyledonary
Epidermal membrane surrounding fetus	No epidermal membrane on fetus
Cartilaginous projection on tip of penis	No cartilaginous projection on tip of penis
Ejaculation prolonged	Ejaculation short and intense
Respiratory system	
Soft palate elongated; primarily a nasal breather	Soft palate short; nasal or oral breather
Urinary system	
Kidney smooth and elliptical	Kidney smooth or lobed
Suburethral diverticulum in female at external urethral orifice	No suburethral diverticulum
Dorsal urethral recess in male at junction of pelvic and penile urethra	Dorsal urethral recess in some species
Parasites	
Unique lice and coccidia; share some gastrointestinal nematodes with cattle, sheep, and goats	Unique lice and coccidia; share GI nematodes
Infectious diseases	
Minimally susceptible to tuberculosis; no known natural bovine brucellosis; mild susceptibility to foot-and-mouth disease; rare clinical disease with other bovine and ovine viral diseases	Highly susceptible to tuberculosis, bovine brucellosis, and foot-and-mouth disease

[a] I = incisors, C = canines, PM = premolars, M = molars.

SOUTH AMERICAN CAMELIDS

The guanaco (Figure 1.4) has the broadest distribution, both historically and currently, of the four SACs. Four geographic subspecies of guanaco have been described,[6] ranging from sea level in Tierra del Fuego at the southernmost tip of South America to 4,600 m in the Andes. The northernmost populations exist at latitude 8° south in Peru.[3,11] Guanacos live in both migratory and sedentary groups.[4] Captive-born guanacos may be tamed and handled similarly to llamas; the Incas used them as pack animals.[11] All guanaco subspecies share uniform coloration, with a dark brown upper body, neck, and limbs; whitish fiber on the underside of the neck and belly; and a grayish to black face.

Vicuña distribution is limited to the puna (Quechua for highland) life zone of the Andes (elevation 4,200 to 4,800 m).[4,6,26] The vicuña (Figures 1.5, 1.6) is the smallest of the SACs and has the finest fiber coat. It has a cinnamon-colored coat, white underparts, a pale cinnamon face, and a bib of white hair on the chest. There

Figure 1.4. Guanaco.

Figure 1.5. Peruvian vicuña (long bib hair).

Figure 1.6. Argentine vicuña (short bib hair).

Figure 1.7. Heavily fibered huacaya alpaca.

are two geographic subspecies of vicuña: the Peruvian, with long white hairs on the bib, and the Argentinian, with shorter hair on the bib. The vicuña was considered the property of the Inca kings, and only royalty were allowed to wear garments made from the fiber.

The two breeds of alpaca, huacaya (Figures 1.7, 1.8) and suri (Figure 1.9), have both become popular in the United States. Alpacas are separated on the basis of fiber coat characteristics: 90% in Peru are of the huacaya breed. Huacaya fiber is shorter than that of the suri breed and is crimped and spongy, giving it the appearance of Corriedale sheep wool.[6,16,19] The coat of suri alpacas consists of long fibers with no crimp that hang down alongside the body in ringlets. A type of llama is now being bred that has suri fiber characteristics. Alpaca coloration varies from white to black with intermediate shades and combinations. The alpaca is the primary SAC fiber producer of the Andean highland.

The llama is the largest of the SACs; however, among individuals there is marked variation in size, overlapping sizes of other species. The llama has been a beast of burden since its domestication. Two breeds are recognized in Peru: the more woolly varieties are

Figure 1.8. Huacaya alpaca female.

Figure 1.9. Suri alpaca.

Figure 1.10. Woolly-necked llama.

Figure 1.11. Llama.

called "ch'aku" in Quechua (Figure 1.10), and those with less fiber on the neck and body are called "q'ara" (woolless) (Figure 1.11). Llamas were killed as sacrificial offerings in the Inca culture, and the practice continues to the present.[4]

Both alpacas and llamas are slaughtered for meat in Andean countries. Lack of refrigeration necessitates immediate consumption, or the meat may be sun dried for storage. Dried meat is called "charqui," from which the North American word "jerky" originated for a similar product.

OLD WORLD CAMELIDS

Dromedary camels are especially adapted to life in hot, arid areas of the world, notably the Middle East, North Africa, and India, with a large feral population in Australia. Dromedaries have been used since ancient times for transport of people and goods, warfare, food, fiber, and companionship. With the advent of modern transportation and the establishment of paved highways, camels have become somewhat obsolete. However, country leaders have deemed it advisable to treasure some of the traditional uses of dromedaries and have fostered camel racing as a sport.

The Bactrian camel is adapted to the cooler, arid climates of Mongolia, southern areas of the former Soviet Union, China, and south central Asia. It is a beast of burden for carrying goods throughout its native lands and was the cargo carrier of goods along the Silk Road from inner China to the Mediterranean. Bactrian camels have a heavy fiber coat to cope with the cooler arid climate and provide the bulk of the fiber used in the manufacture of camel hair garments.

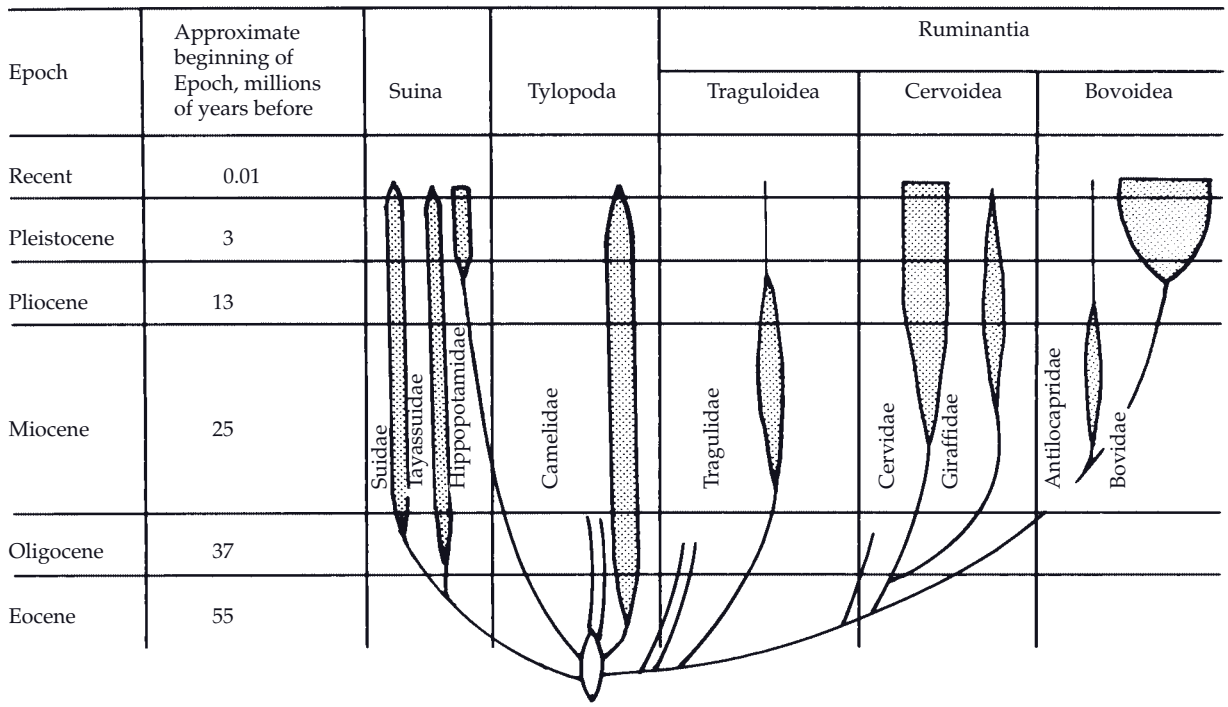

Figure 1.12. Suggested evolution of recent artiodactylids. Adapted from Romer 1966, Simpson 1980.

EVOLUTION

Camelid

Camelid evolution began in North America over 82 million years ago in the early Eocene epoch.[32] Geologic and paleontologic time scales are estimates and subject to revision. Figure 1.12 provides a diagram of the relationships of the various artiodactylid families since the Eocene epoch. Webb published a definitive work on the evolution of Pleistocene camelids based on paleontologic evidence.[23] He suggested three major tribes, separated as early as the Eocene epoch. This family tree (Figure 1.13) is sketchy, with insufficient fossil records to trace lines accurately (postulates indicated by broken lines).[8,23] Two tribes, Camelopini (Camelops) and Camelini (Camelus), evolved in North America only west of the Mississippi River. The tribe Lamini (Lama) was also found in Florida.[11,29]

The Pleistocene epoch was characterized by a series of periods of extreme cold and glaciation in northern North America and Europe.[18] The last glacial retreat occurred about 10,000 years ago, marking the beginning of the recent epoch. It was during the Pleistocene epoch that the camelidae flourished (Figure 1.14).[7,9,19,22] Many genera in the family Camelidae became extinct, for unknown reasons, before the recent epoch.

Figure 1.13. Skeletons of various prehistoric camelids removed from the La Brea tar pits in Los Angeles, California.

Camel

Asia and Alaska are now separated by the 90-km (56-mile)-wide Bering Strait. However, during the height of one of the early Pleistocene glacial periods, the sea level was lowered sufficiently to expose a wide land bridge.[2] Plant and animal species moved back and forth across this bridge; the camel line of Camelidae migrated from North America into Asia to continue the evolutionary process, dying out in North America.

Once in Asia, Camelus radiated through eastern Europe (Rumania and southern Russia), the Middle East, and North Africa as far west as the Atlantic and as far south as Tanzania. It is likely that the dromedary

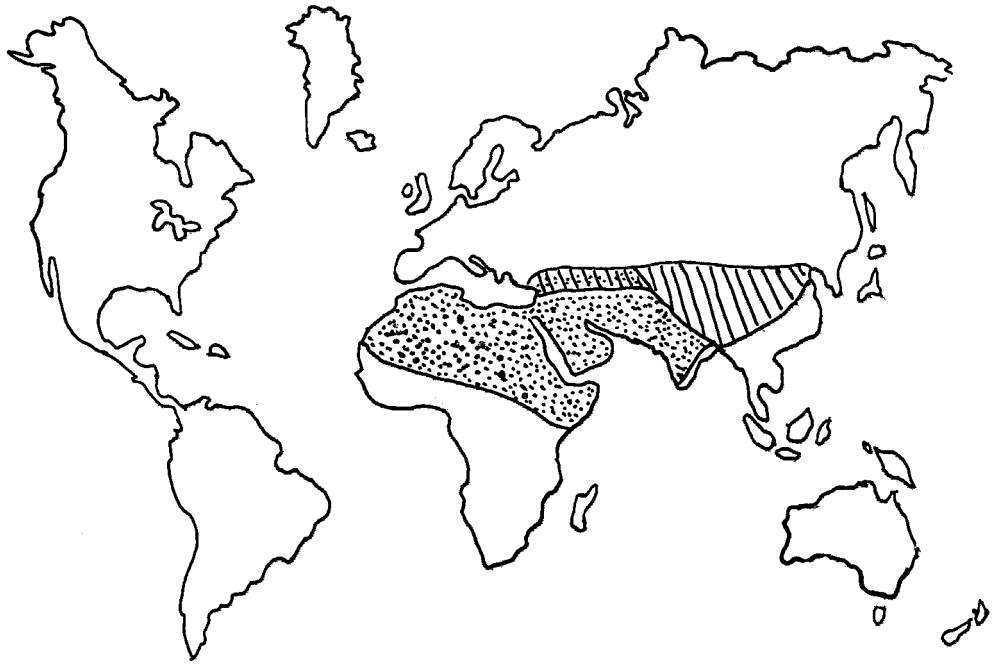

Figure 1.14. Historic world distribution of camels.

evolved from the Bactrian camel, although the hump(s) may be an acquired characteristic of domestication. Wild camels had become virtually extinct in North Africa before historic times (3000 B.C.). Only *C. bactrianus ferus* now exists in the wild state in one small area in the Trans-Altai Gobi Desert on the border of Mongolia and China, with a limited population of fewer than 1,000 animals.[12,14,22]

South American Camelids

The first SACs migrated to South America at the beginning of the Pleistocene epoch (approximately three million years ago) when an open land connection between North and South America developed.[17,24] The Isthmus of Panama was formed by volcanic eruptions from the ocean floor, forming a series of islands that ultimately became interconnected (the Caribbean land bridge). The major earliest SAC genus appearing in South America was Hemiauchenia (Tanupoloma), which radiated throughout the flatter regions east of the Andes. During the middle Pleistocene, the genera Palaeolama, Lama, and Vicugna developed from the long-limbed, flatland-adapted Hemiauchenia. These genera had shorter limbs, which more easily adapted them to the mountainous Andes.

Various species of Palaeolama migrated back to North America. Fossils associated with North American Hemiauchenia have been found along the Gulf Coast and Florida (Figure 1.15).[28]

Table 1.3. Approximate number of years that certain animals have been domesticated.

Reindeer	14,000	Llama and alpaca	6,000–7,000
Dog	12,000–15,000	Horse	5,500
Goat	11,500	Dromedary camel	5,000
Sheep	11,000	Bactrian camel	4,500
Cattle	9,000	Asian elephant	4,000
Pig	9,000	Cat	3,000–4,000

DOMESTICATION

Camels

The approximate time of domestication of a few animals is listed in Table 1.3. The precise time and location of domestication of the Bactrian camel is unknown, but it is thought to have occurred sometime prior to 2500 B.C. on the border of Turkmenistan and Iran on the east side of the Caspian Sea.[14] The name Bactrian is derived from a place name, Baktria, on the Oxus River in northern Afghanistan. Strangely, this is not the place of origin of the domestic two-humped camel, nor is the species even found in this area at present.

Domestic Bactrian camels had spread north into southern Russia by 1700 to 1200 B.C. and were in western Siberia by the tenth century B.C. Bactrian camels were used in China as early as 300 B.C. as the

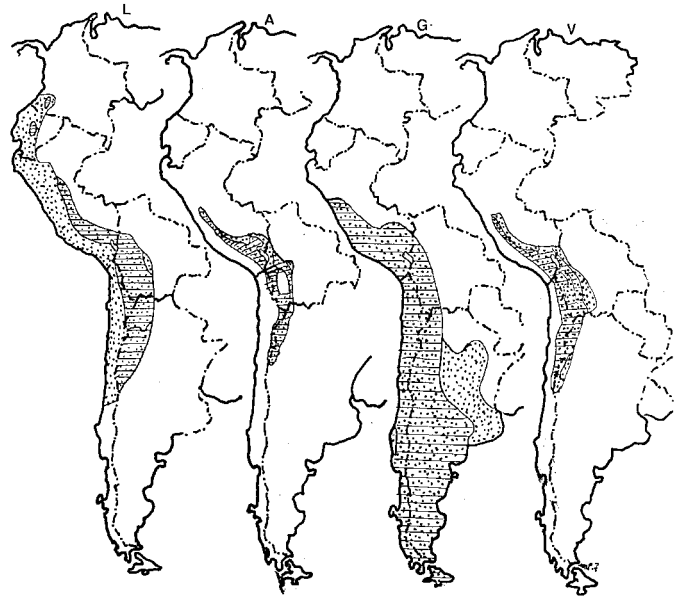

Figure 1.15. Historic and current distribution of NWC in South America.

original Silk Route camels but were later replaced by crossbreeds of the dromedary and Bactrian camels.[14]

Domestication of the dromedary occurred prior to 3000 B.C. in the Arabian peninsula. The term "dromedary" is derived from dromos (Greek for road) and thus is only directly applicable to the riding or racing dromedary camel. However, dromedary is the name used throughout the world for this species, which existed in historic times only as a domesticated animal. Dromedaries were first associated with nomadic Semitic cultures and never became important until the rise of the Arabian culture.[14]

Dromedaries were reintroduced into North Africa in the third century B.C. More were brought into Egypt during the Roman period, after the third century A.D., but became important domestic animals only with the Moslem conquests of Egypt in the seventh to eleventh centuries A.D. A comparison of camel characteristics is found in Table 1.4.

Llamas and Alpacas

The cradle of llama domestication is the Andean puna (elevation 4,000 to 4,900 m), probably around Lake Titicaca, at approximately 4000 to 5000 B.C. Alpaca domestication probably occurred elsewhere, perhaps near Telarmachay. Alpaca-type incisors have been found in middens at Telarmachay, dated at 4000 to 3500 B.C.[15,16,20,31] Once domesticated, llama and alpaca herding economies spread beyond the limits of the puna and became important in the economy of the Andean people from sea level to high mountain elevations.[17,27]

The Inca empire was dependent upon the llama and alpaca for food, fuel, clothing, transport of goods (the wheel was not introduced to South America before the conquest), and religious ceremonies. All SACs were the property of the government, and production of domestic species was rigidly controlled. The fiber from vicuñas was for royal usage only. The maximum numbers and broadest distribution of SACs developed under Inca rule. After the Spanish invasion of 1532 and the introduction of European breeds of livestock, numbers and distribution of SACs declined. However, llamas and alpacas survived because they are essential to Andean culture. They are the most reliable source of food, fiber, and fuel in the high, cool Andean environment. SAC ownership is the primary source of wealth for indigenous people.[17,31]

In 1970 the Peruvian government initiated an agrarian land reform. The large ranches, owned by wealthy, absentee landlords, were confiscated and returned to the *campesinos* or pastoralists who had been working the land. Some groups of *campesinos* formed cooperatives, and the better ones hired skilled people to manage the cooperatives, some with herds as large as 40,000 alpacas. During that time, however, most of the llamas and 80% of alpacas in Peru were under the

Table 1.4. Characteristics of camels.

Characteristic	*Dromedary* Camelus dromedarius	*Bactrian* Camelus bactrianus	*Wild Bactrian* C. bactrianus ferus
Breeds/types	50 different breeds recognized Draft type: heavy body, stocky legs Riding type: Slim body, long legs Racing type: Similar to riding	Geographical differences	Single type
Weight kg/lb	300–650*/661–1432	450–700/992–1543	450–690/992–1521
Weight of newborn kg/lb	26–45/57–100	35–54/55–120	?
Height at shoulder cm/in	180–210/71–83	180–195/71–77	180–200/70.8–78.7
Body length cm/in	120–200/40–80	120–200/40–80	140–156/55–61
Shape	1 firm, upright hump	2 large humps, may be flopped over	2 small, conical humps
Color	Cream to tan to dark brown	Cream to tan to dark brown	Cream to gray–brown
Unique anatomy	Male has a soft palate diverticulum (dulaa) which may protrude from mouth	No dulaa, ears 15 cm	No dulaa, prominent toenail, small foot and flat sole for rapid gait to escape predation. Able to drink salty water, face narrow, ears 10 cm
Fiber/hair	Diameter 20–50 µ	Diameter 10–40 µ, long staple, primary source for camel hair garments	Short fiber
Special adaptations	Adapted to heat, aridity, and sparse vegetation	Adapted to cooler, arid environments	Adapted to the deserts of northern China and Mongolia.
Running speed	21.6–40.3 kph (13.4–25 mph)	15–20 kph (9.3–12.4 mph)	40 kph (24.9 mph)

*Males, castrated as juveniles, may grow taller and larger than 1,134 kg (2,500 lb).

control of traditional pastoralists[4,6,17], who maintained small to moderate herds (thirty to 1,000) grazing on communal lands. The large cooperatives were disbanded in 1988, partly as a result of the activities of the *Sendero Luminosa* (Shining Path) terrorists and because the new president and his advisors felt the cooperatives were not functioning as they should. Currently llamas and alpacas are raised by traditional pastoralists.

In the nineteenth century SACs were exported to other countries from South America as zoo animals. Peru enacted legislation in 1843 prohibiting the export of live alpacas. Approximately seventy years ago, all of the Andean countries banded together to prevent exploitation of SACs by other countries. No legal exportations occurred from then until the 1980s, when the ban was lifted from alpacas and llamas.[4]

North American llamas have expanded from the small population imported from South America prior to 1930 and a few animals that had been imported from other countries. Current numbers of North American llamas are estimated to be between 100,000 and 120,000, and alpacas are thought to number more than 150,000. Sporadic importation of llamas and alpacas from Chile into the United States began in 1984 after the U.S. government periodically recognized Chile as free of foot-and-mouth disease. Importations have also originated in Bolivia and Peru, but these must meet special quarantine protocols for animals deriving from countries where foot-and-mouth disease is endemic. A few animals have been imported from England, New Zealand, Canada, and Australia.

A general overview of the similarities and differences among the four South American species is listed in Table 1.5. Sizes of SACs are listed in Table 1.6. Additional biological information is provided at the beginning of other chapters.

USES OF CAMELIDS

Camels are an important part of the culture of the nomadic peoples of Asia and the Middle East, supplying food (meat and milk), fuel (the fecal pellets), fiber

Table 1.5. Characteristics of South American camelids.

Characteristic	*Vicuña*	*Alpaca*	*Guanaco*	*Llama*
Types/breeds/ subspecies/ races	**Peruvian**—Apron or bib on frontal chest **Argentine**—No chest bib	**Huacaya**—Fibers with light crimp. Fleece at right angles to body. **Suri**—Fiber lacks crimp. Fleece hangs in ringlets.	May be as many as 4 subspecies or races	South American breeds: Heavy neck fiber (chaku, lanuda, tapada) Short neck fiber (ccara, pelada). No North American breed standards. Alpaca and Llama Show Association show classification: Light, medium, and heavy wool divisions.
Conformation				
Topline	Straight with rounded rump	Straight with rounded rump	Straight or slightly rounded from withers to tail	Straight from withers to tail
Head and neck carriage				
Alert	Vertical	Vertical	Vertical	Vertical
Resting	Vertical	15 degrees from vertical	Vertical	Vertical
Ears	Short, curved on both borders	Short, spear-shaped, curved on both borders. Huacaya—sharp tip. Suri—rounded tip.	Medium length, curved on both borders	Long, inner border straight or curved inward (banana ears)
Face	Short	Short	Medium	Long
Pastern	Sloped	Almost vertical	Sloped	Sloped
Fiber				
Diameter in microns (avg.)	10–30 (13–14)	Huacaya—16–40 (22) Suri—16–35 (23)	18–24	Undercoat—16–40 (26) Guard hair—40–150 (70)
Quality	Finest of all SACs. Staple short.	Excellent. Staple long.	Inner coat is excellent; juvenile pelts are used for fur garments. Staple short.	Variable; inner coat may make excellent garments. Staple may be long or short.

Coverage	Uniform over body, head, and upper legs. No topknot.	Topknot present. Fiber extends below the knee and hock.	No topknot. Uniform over body, head, and upper legs.	Fiber usually doesn't extend much below the knee or hock or onto the face
Guard hair	The bib and lower body fiber are guard hairs.	Highest-quality animals have no guard hair.	Concentrated on lower parts of body and limbs	Numerous
Color	Color pattern similar to that of guanaco, but the basic body color is a yellowish light brown. The white in front of the rear limbs may extend to the top of the back. Argentine subspecies has long white guard hairs on the chest, but this is extended into a bib (8–14 in./20–35 cm) in Peruvian subspecies.	22 solid colors recognized, ranging from white to black. Multicolored fleeces also produced.	Basic body color light to dark reddish brown (cinnamon) above, whitish hair below (countershading). White extends up behind the foreleg and in front of the rear leg, around the perineum, inside of legs, and up the bottom of the neck. Front and outside of the upper limbs are body color or grey. Head, face, and ears are dark grey to black, with the darkest color over top of the head and bridge of the nose.	Numerous solid colors from white to black. Multicolors (pinto, appaloosa) also seen.
Teeth	Incisors long, narrow (sides parallel) and continue to grow throughout life. Enamel present only on labial side of the incisor teeth.	Incisors elongated and continue to grow into adulthood. Enamel present only on labial side of incisors.	Incisors broad, spatulate-shaped and do not continue to grow. Enamel surrounds the crown of the incisors.	Incisors similar to those of guanaco
Feeding strategy	Grazer/browser	Prefers to graze succulent forage in marshes and moist places but also utilizes drier grasses and shrubs	Uses broad range of habitat for grazing and browsing	Browser by preference but grazes grasses, even dry, harsh species

Table 1.6. Weights and sizes of South American camelids.

Characteristic	*Vicuña*	*Alpaca*	*Guanaco*	*Llama*
Weight (lb/kg)				
Adults	99–121/45–55	121–200/55–90	220–265/100–120	250–550/113–250
Birth weight	9–13/4–6	13–20/6–9	18–33/8–15	18–40/8–18
Height at withers				
Adults (in./cm)	34–38/86–96	30–38/76–96	43–45/110–115	40–47/102–119 Some imports 38 in.

Figure 1.16. Dromedary camel as a riding camel for tourists.

Figure 1.17. Dromedary camels as draft animals.

(clothing, ropes), leather, transportation (packing, riding; Figure 1.16), and racing. Special breeds of dromedaries were developed for riding and became important in the mobilization of military expeditions. Camels are also used for draft purposes, pulling plows and wagons (Figure 1.17). Camels were used heavily in Australia prior to the development of modern vehicles for packing and pulling heavy and awkward items into the Outback. The feral camels of Australia are descendants of escapees. Camels also were used in the United States as part of a military experiment in the hot, desert environment of the Southwest. The Camel Corps was soon disbanded for various reasons.

Figure 1.18. Racing dromedary camels in the United Arab Emirates.

Camel racing has become a popular sport in Kuwait, Saudi Arabia, Qatar, Oman, and the United Arab Emirates (Figure 1.18). Adult female camels over seven years of age are generally used for major racing. Male camels are less easily managed. Younger camels may participate in short races but the major races are 4 to 10 km. The track is a huge oval and camels are quickly out of sight of the central grandstands. The race is viewed by remote television from vehicles that accompany the racers on a paved track on the inside of the race course.

The camels are ridden by lightweight jockeys (formerly by children) sitting behind the hump. They are now sometimes ridden by mechanical robot jockeys. The start of major races is a raucous affair with as many as fifty camels at the starting gate. The camels move out at a gallop and may reach speeds up to 40.32 kph (25.05 mph) for the first 2 km. Then they settle down to a fast pace at an average speed of 32.4 kph (20.1 mph).

Camels may change gaits (pace to gallop, and vice versa) during the course of the race, much like the flying change of leads in a galloping horse. The camel gallop is slightly faster than the pace and is often used in the dash to the finish line. Wagering (betting) is not allowed in Moslem cultures. Prize money is awarded

down to the tenth place. The top prizes in major races may be elite 4-wheel-drive sport utility vehicles.

Llamas have a long history of interaction with Andean people, supplying meat, leather, and fiber (garments, blankets, rope, costales [burlap bags]); transporting goods; and serving as sacrificial animals.[4] In North America, llamas fill numerous niches, including, but not limited to, breeding, showing (Figure 1.19), parades, companion animals, packing (Figure 1.20), driving, and fiber (cottage industry). Well-trained, quiet llamas are ideal for taking to schools and convalescent hospitals and for interacting with emotionally and physically disadvantaged children and adults. Another important niche is the use of llamas to guard sheep and goats from predation by coyotes.

Alpacas are the premier fiber-producing animals in the Altiplano region of the Andes. They are also harvested for meat in South America, and the leather is used to make ropes. The pelts of crias are used to make fine rugs and wall hangings; they are sewn together in intricate patterns and designs. In North America, alpacas serve as companion animals and are used for breeding and showing. The use of their excellent fiber is currently a cottage industry, but has the potential for commercial production.

Figure 1.19. Showing alpacas.

Figure 1.20. Packing llamas on trek.

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2

Feeding and Nutrition

Camelid owners and managers develop strong views on what constitutes appropriate feeding, and newcomers to the industry are bombarded with, "This is the way you have to feed." There are no absolutes in feeding practices for camelids. Veterinarians may be unable to clarify the issues without basic information. It is not what you feed but how you feed it!

To understand the basis for feeding practices and nutritional physiology, knowledge of the anatomy of the digestive system is required. Therefore, study of this subject should be preceded by a review of the anatomy of the camelid digestive tract (Chapter 13). Furthermore, to knowledgeably feed camelids when managed in captivity, it is necessary to know how they subsist in their native countries. In South America, SACs are never fed concentrates or supplements and are rarely fed cured hay. They feed on the native grasses and forbs. Llamas/alpacas are concentrated in the Altiplano, which is an area of broad, high-altitude valleys and plateaus at elevations over 3,800 m (12,500 feet). The climate of this area consists of a long, dry season and a short, wet season, with 75% of the rainfall occurring from December through March. The dry season is from May through October.

The growing season (wet) is characterized by low temperatures (more than 300 nights of frost per year) and intense solar irradiation. Daytime temperatures reach a maximum of 18.3°C (65°F); nighttime temperatures fall to a low of –12°C (10°F) (Figures 2.1, 2.2). Available forage varies from the wet to dry seasons, but the animals adapt to the "feast and famine" routine by depositing layers of fat in the subcutaneous, muscular, and retroperitoneal tissue during the wet season and mobilizing these reserves in lean times. This adaptation to good followed by seasonally deplorable feeding conditions is usually completely forgotten by North American managers. Because there is never a lean time, animals often become obese.

Camels are highly adapted to the consumption of dry, harsh forage. Dromedary camels foraging in their native environment prefer to browse during the wet season and graze during the dry season. Camels do not exhibit the feast and famine syndrome, as described for SAC, but do store fat in their humps. The well nourished camel has a firm upright hump, whereas an emaciated camel's hump is smaller and may flop to the side, particularly in Bactrian camels. If Bactrian camels are overfed when maintained in captivity, the humps may also bend over to the side because of the excessive weight. There may also be a genetic component to the hump's fibrous infrastructure that is not strong enough to cope with extra weight.

Since the first edition of this book, considerable research has been conducted on the feeding and nutrition of camelids.[9,10,45,47,72,112] Grazing and feeding behavior in South American camelids has been studied,[21,67,75,93,94] adding to knowledge gained from previous studies.[72,73,88,89,106] Some experience in managing the feeding of camelids in zoos and private ownership has been reported.[13,29,59,104,122]

This chapter provides background on the feeding behavior of camelids in their native habitat, presents a review of the literature concerning digestive physiology as it relates to nutrition, and makes some recommendations for suitable rations based on current knowledge of nutrient requirements.

FEEDING BEHAVIOR[8,9,21,35,48,55,85,93,95,104,127]

The vicuña is one of two species of wild camelids. It inhabits the high Andes at elevations of 3,700 to 4,800 m. This is a harsh environment of semiarid grassland and barren pampas, with cold temperatures and sparse vegetation.[36] The vicuña is a grazer of forbs and grasses.

Medicine and Surgery of Camelids, Third Edition by Murray E. Fowler

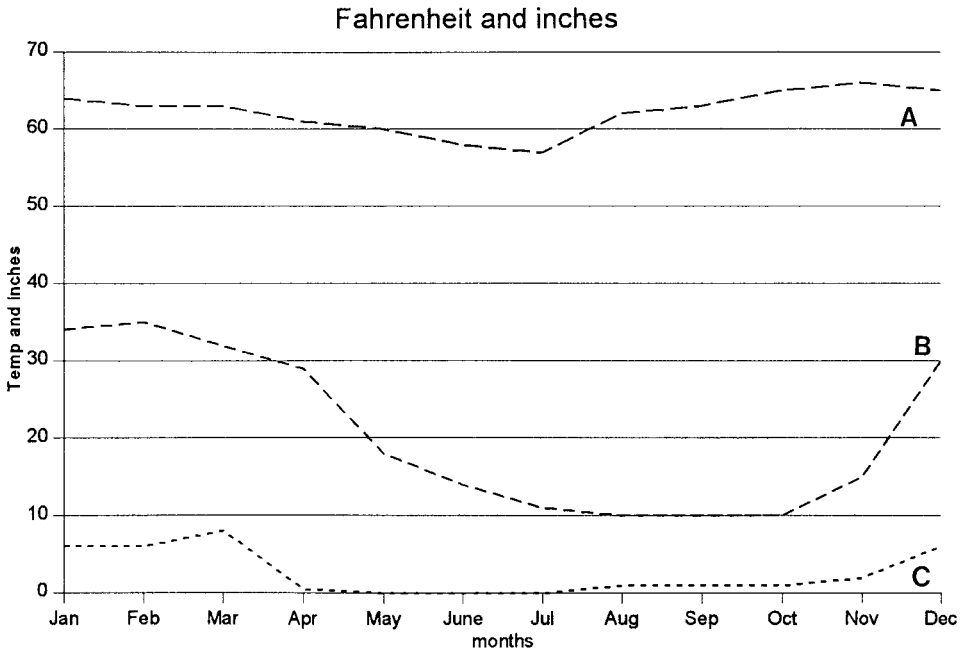

Figure 2.1. Average ambient maximum (A) and minimum (B) temperatures (°F) and rainfall in inches (C) at a station in the Altiplano.

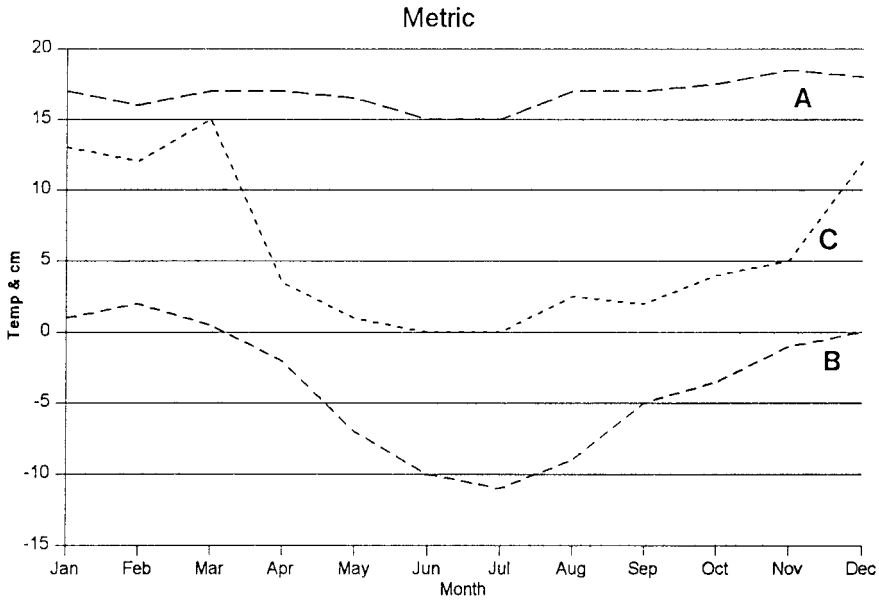

Figure 2.2. Average ambient maximum (A) and minimum (B) temperatures (°C) and rainfall in centimeters (C) at a station in the Altiplano.

The guanaco, also a wild species, has the broadest geographic and altitudinal distribution, ranging from sea level to 4,250 m. The guanaco is both a grazer and a browser, inhabiting desert grassland, savanna, and shrub land; it may even be seen in forests. It is found in one of the driest deserts of the world, the Atacama in Chile, and also in the wet archipelago of Tierra del Fuego, where rain falls year-round.[86]

The llama is found at moderate elevations of 2,300 to 4,000 m. This distribution pattern may be more cultural than biologic, since the llama has shown ready adaptability to diverse altitudinal and geographic habitats in locations outside South America. The llama is both a grazer and a browser, preferring to pasture on dry tablelands and slopes, feeding on tall, coarse bunchgrasses, which are collectively called "ichu" (Stipa, Festuca, Calamagrostis) and dominated by fescue (*F. doli chophylla*). Llamas also browse available shrubs and trees. A number of excellent studies have been conducted to determine the nutrient composition

Figure 2.3. Alpacas grazing on bofedales. The distant slopes are used by llamas.

of alpaca/llama forages at different seasons of the year. These studies support the concept that, to have evolved and become domesticated in this harsh environment, llamas/alpacas must be hardy, resilient, and efficient in extracting the maximum amount of nourishment from forages.

The alpaca is found at elevations of 4,400 to 4,800 m and is a grazer, preferring the bottomland vegetation of meadows and marshes collectively called "bofedales" (Figure 2.3).[36,90] The nutrient composition of Peruvian plants varies greatly seasonally, by altitude, and according to soil type.[55] In dry seasons the vegetation is sparse and of poor nutrient quality. However, proximate analysis of Peruvian plants (Table 2.1) shows that at least during part of the year the nutrient quality may be quite high. Even though camelids have become evolutionarily adapted to survival in their harsh environment, it is important to recognize that they may grow better and be more fertile if given optimal nutrition, at least for a major portion of the year.

A select list of plant species eaten by dromedary camels is found in Table 2.2. As the table shows, there is considerable variation in the nutrient composition of the plants but this is true of plants available to livestock throughout the world.

NUTRITION ANATOMY AND PHYSIOLOGY

Prehension

All camelids have incisor teeth that are firmly fixed in the mandible, similar to the dentition of sheep and goats but in contrast to the loose attachment of bovine teeth. These teeth, pressing against the upper dental pad, shear vegetation. These animals are able to graze short plants and cut them close to the ground, but their general behavior is to move about a feeding area, picking a mouthful here and there, not mowing any one spot clean unless they are tethered.

The lips of camelids are unique. The upper lip is split by a labial cleft. Each side of the lip can be manipulated independently to investigate a potential food item and draw it to the teeth. SACs rarely consume foreign bodies because of this fastidious checking of feed with the lips, but camels may.

The tongue does not participate in prehension. Because the tongue rarely protrudes from the mouth, camelids do not, as a rule, lick themselves, their young, or salt blocks. Some may lick at a salt block long enough to obtain some supplement, but usually camelids chew at the block rather than lick it.

Mastication

Initial chewing is cursory, sufficient only to mix feed with saliva to form a bolus to be swallowed. The amount of time required by a camelid to complete sufficient ingestion of feed varies with the quality and availability of the forage, but in most cases it occupies a third of the daylight time. Camelids are not active in the dark.

Mastication is also a part of the rumination cycle. Koford[61] described the rumination cycle in the vicuña. While resting, the vicuña brings up a bolus and begins chewing, with the mandible scribing a horizontal figure-eight arc. A deer or a bovine masticates with a unilateral elliptic movement of the jaw. The vicuña

Table 2.1. Composition of Peruvian llama and alpaca pasture plants.

Feed–Pasturage South America		*Dry matter (%)*	*Protein (%)*	*Digestible energy (Mcal/kg)*	*Fiber (%)*	*Calcium (%)*	*Phosphorus (%)*	*Ca:P*
Muhlenbergia, aerial part, fresh	Early	100.0	6.8	2.60	31.2	0.37	0.13	2.8
Muhlenbergia fastigiata	Mature	100.0	5.8	2.86	27.5	0.41	0.13	3.2
Reedgrass, aerial part, fresh	Early	100.0	6.2	2.59	33.8	0.38	0.11	3.5
Calamagrostis antonia	Mature	100.0	4.6	2.34	32.0	0.64	0.07	9.1
Fescue, aerial part, fresh	Midbloom	100.0	8.3	2.12	41.3	0.09		
Festuca dolichophylla	Mature	100.0	2.5	1.21	44.3	0.20	0.04	5.0
Spikesedge, aerial part, fresh	As fed	21.7	3.4	0.59	7.4	0.07	0.04	1.8
Eleocharis geniculata	Dry	100.0	15.5	2.73	34.1	0.32	0.19	1.7
Plantain, aerial part, fresh *Plantago* sp.	Dry	100.0	14.7	3.05	35.4	0.16	0.26	0.6
Crimson clover, aerial part, fresh	As fed	17.6	3.0	0.49	4.9	0.24	0.05	4.8
Trifolium incarnatium	Dry	100.0	17.0	2.81	27.7	1.38	0.29	4.8
Ladysmantle, aerial part, fresh *Alchemilla pinnata*	Dry	100.0	10.9	2.79	28.9	1.90	0.24	7.9

Source: McDowell et al. 1974.

Table 2.2. Percent digestibility of two diets in pony, sheep, guanaco, and llama.

Feed	*Nutrient*	*Pony*	*Sheep*	*Llama/guanaco*
Alfalfa pellets	Dry matter[a]	64.8	63.8	71.5
	Acid detergent fiber	46.7	50.5	61.0
	Cellulose	51.2	64.8	77.6
	Crude protein	65.7	69.7	74.7
Complete pelleted diet	Dry matter	69.7	71.7	78.0
	Acid detergent fiber	29.3	34.0	38.7
	Cellulose	34.5	39.4	47.2
	Crude protein	68.8	63.1	69.2

Source: Hintz et al. 1973.
[a] The animals consumed between 1.7 and 2% of their body weight in dry matter.

retains the cud in the mouth for fifteen seconds, during which time it chews twenty-five to thirty-five times. That bolus is then swallowed and the process repeated. Other camelids chew with a similar pattern.

Saliva[22,82]

The gross and microscopic anatomy of the salivary glands are similar to those of other artiodactylids. Salivary secretion is necessary to moisten and lubricate feed so that it can be swallowed. In one study,[76] the prefeeding flow of the parotid saliva of the alpaca was 140 ml/hour. The pH was 8.6, and the composition was as follows: 121 mEq/L HCO_3, 33.5 mEq/L HPO_4, 164.8 mEq/L Na, and 13.7 mEq/L K.

While feeding, the flow was 202 ml/hour, pH was 8.59, composition was 127.8 mEq/L HCO_3, and other ion concentrations were similar to prefeeding levels. At postfeeding, the flow remained higher than during prefeeding, at 159 ml/hour, and the pH stayed at 8.58. Other parameters returned to prefeeding levels.

Stomach[25,74,121,123,124]

The digestive strategy of both camelids and ruminants is similar. Both taxa utilize coarse forages that require a fermentation chamber to convert plant nutrients to molecules that may be absorbed by the animal. Millennia of parallel evolution provided similar anatomic and physiologic characteristics. Both regurgitate and rechew forage (rumination), but there are other species, including some primates and marsupials, besides ruminants that have that behavior.

Each group has distinct differences and it is not appropriate to call a camelid a pseudoruminant or a modified ruminant. Nor is it appropriate to call a ruminant a true ruminant. They are unique in and of them-

selves and it is important to understand this when considering feeding these animals.

Compartments one and two (C-1 and C-2) of the three-compartmented stomach are anaerobic fermentation chambers, hosting the microbial flora and fauna necessary for utilization of coarse, fibrous herbage.[113] Nonetheless, there are numerous and significant differences in the anatomy and physiology of the camelid stomach as compared with the stomach of a ruminant.[34,119,121]

Stomach motility is markedly different. In camelids at rest, three to four contractions of C-1 per minute are average.[117,119] If the animal has eaten recently, the rate is faster. The contraction wave of C-1 is from caudad to craniad, in contrast to the craniad-caudad wave of the ruminant. It is generally not possible to palpate the contraction in the left paralumbar fossa, as can be done with cattle and sheep; rather, it is necessary to listen with a stethoscope. Because fermentation takes place only in the forestomach of camelids, there should be little gas formation, hence borborygamous, except from the stomach.

In ruminants, the rumen contents are stratified with a gas layer dorsally, a solid layer of ingesta in the middle, and a more liquid and small particulate matter layer ventrally. In camelids, the ingesta is homogeneous and relatively dry. This causes some difficulty when attempting to aspirate ingesta to transfaunate another animal. Bloat is rare in camelids, and the homogeneity of the ingesta may be a factor in avoiding frothy bloat.

The reticular groove of camelids is not nearly as well defined as it is in cattle, sheep, and goats. Nonetheless, in the neonate camelid, milk is shunted past C-1 into C-2 and then into C-3. In a study by Vallenas,[115,116,118] the response to chemical stimulation was more like that of a bovine than of an ovine. There was good to fair response with 10% solutions of sodium sulfate, sodium chloride, and sodium bicarbonate, but no reaction to a copper sulfate solution.[116]

The same study evaluated glucose absorption in alpacas.[116,118] In the alpaca neonate, the plasma glucose level was 121 mg/dl, whereas in the adult alpaca it is 72 to 99 mg/dl. In the bovine, it is generally believed that all carbohydrates are acted upon by ruminal microorganisms and converted to volatile fatty acids. This requires a functional rumen. Thus, in the bovine neonate up to about three months of age, glucose reaches the abomasum and intestine, where it is absorbed as such. In the adult, there is no glucose absorption from the rumen and less available glucose reaches the abomasum and intestine, so plasma levels are lower. In the alpaca a similar situation exists. Blood glucose levels in llamas (125 ± 38 mg/dl) must be evaluated carefully because elevated levels may be the result of the excitement caused by the restraint necessary for blood collection, stimulating catecholamine release.

Urea metabolism in camelids is similar to that in the ruminant in that urea can be recycled and utilized by stomach microorganisms for the synthesis of protein.[2,49,50,53] In general, blood urea levels are low in adult ruminants (8 to 30 mg/dl). This is also true in llamas, with adults at 24 ± 13 mg/dl and neonates at 14 ± 8 mg/dl.

When a llama fasts for three to four days, plasma urea elevates because of a significantly lower turnover and transfer of urea to the gastrointestinal (GI) tract.[23] Tissue catabolism may also be a factor. The specific mechanism involved is not known, but it is likely that decreased permeability to urea has developed in the stomach mucosa.[51] The foregoing has relevance to a llama that is ill and anorectic. Elevated blood urea nitrogen levels may not necessarily indicate a primary renal problem.

In one study, it was noted that llamas were able to hydrolyze more urea per unit of time (mmol/hour/kg) in C-1 than cattle and sheep can in the rumen. Therefore, in the llama, more urea would be available for protein synthesis by microorganisms.[50]

A number of studies have investigated camelids' superior increased efficiency in extracting energy and other nutrients from coarse forages.[13,83,118,132] Some early studies indicated that alpacas were 50% more efficient than sheep in digesting fiber.[51] Later and more sophisticated studies demonstrated greater efficiency, but to a lesser degree, in experiments involving poor quality forage, and no difference in those involving high-quality forage.[53] Table 2.3 provides data from a study comparing an alfalfa hay and a pelleted complete ration.

A greater efficiency of fiber and protein digestion may be the result of a number of factors, including more rapid forestomach contractions that provide for better maceration, mixing, and absorption. Early studies indicated that a more effective buffering system in C-1 neutralizes short-chain volatile fatty acids (VFA), which in turn encourages increased production of VFA.[92,114] More sophisticated studies, using a Pavlov pouch, did not corroborate those findings but rather suggested that the glandular areas provided more complete and more rapid absorption of VFA, thus stimulating further production of VFA.[100]

The degree of digestive efficiency may reflect the quality of forage eaten.[11,100] More studies in a variety of situations and with a variety of forages are needed to establish the facts.

The end products of carbohydrate fermentation in camelids are VFA, just as in the true ruminant.[18] In the llama and guanaco, the peak production of VFA occurs approximately one and a half to two hours after feeding, and normal levels return within five to six

Table 2.3. Chemical composition of selected plants eaten by camels.

Plant	*Part eaten*	*Dry matter %*	*Crude protein %*	*Crude fiber %*	*Ash %*
Acacia nilotica	Leaves, stems	48	17.50	33.70	5.70
Acacia tortilis	Browse	72.62	9.28	38.33	8
Anachis hypogaea	Mature peanut foliage without nuts	100	DP 10.5	20.3	—
Aristida kelleri	Aerial part		4.90	27	34.90
Artemesia campestris	Browse	31	17.30	24.80	8.80
Capparis decidua	Browse		4.90	31.60	13.50
Cassia spp.	Browse	39	10.80	16.70	5.70
Chenopodium album	Leaves	95.60	21.20	8.30	32
Chloris gayana	Rhodesgrass hay	91	10.1	46.7	9.6
Cyprus esculentus	Aerial part		6.50	21.90	25.40
Ephedra aeta	Browse	95.20	10.40	45	7.50
Hordeum glaucum	Aerial part	94.1	3.70	36	9
Lolium spp.	Aerial part	91	9	3.90	11.30
Prosopis africana	Leaves	54	14.20	34	8.60
Phoenix dactylifera	Fruit	80	3	3.60	4
Opuntia vulgaris	Fruit	15	9	42.90	3.20
Tribulus terrestris	Aerial part	310	17.20	45.10	17.70
Salsola kali	Aerial part	21	15	23.70	20

Source: Wardeh 1991.

hours.[120] There is a negative correlation of increase in VFA with pH. In cattle, peak production occurs two to three hours after feeding, and the level is maintained longer. The pH drops faster and lower, presumably because cattle lack the more effective absorption system of the camelid.[51]

In a study conducted by Vallenas et al.,[123] VFA were found all along the GI tract, but compared with sheep, cattle, and deer, there was a lower concentration of VFA caudal to the stomach in llamas and alpacas, indicating either more efficient absorption of these nutrients from the stomach or less microbial activity beyond the stomach.

The camelid forestomach is characterized by the presence of glandular mucosal areas in all compartments.[22,121] In C-1, in both cranial and caudal sacs, areas of saccules lined with a mucinous glandular epithelium evert during stomach contraction, expelling the contents into the lumen of C-1.

The glandular epithelium of C-2 covers all but the small area of the lesser curvature. The cells are not as deep as those of the saccules of C-1. The reticular pattern of C-2 is superficially like the pattern of the reticulum of the true ruminant, but these structures are not analogous.

STOMACH MICROBIOLOGY[26,105]

The microflora and -fauna of ruminants have been intensely studied and found to be highly complex. Hundreds of species of bacteria and protozoa inhabit the rumen and participate in the fermentative process so important to the nutrition of these species. Both Gram-positive and Gram-negative organisms are present, but all are anaerobic. Each species, or perhaps group of species, acts on certain components of the diet. Specific bacteria digest cellulose, hemicellulose, starches, sugars, acids, lipids, and proteins. Other bacteria produce ammonia, or methane, and still others synthesize vitamins. Several bacteria have multiple functions.

The species present and relative proportions of each may depend upon numerous factors. Each species has its own special substrate requirement, and any disease- or human-induced change in the environment may destroy a specific population of bacteria, upsetting the nutrition of the animal.

In addition to bacteria, dozens of species of ciliated and flagellated protozoa have been found. The species present in a given animal may vary according to diet, season of the year, and geographic location.[12]

Few reports have been published of studies of the species of protozoa found in camelids.[25,105,106] Speciation is a complicated and specialized process. Practically, it is important to know that protozoa are present and that sudden changes in diet should be avoided so as not to destroy the resident population. Camelids can adapt to new forages, but introduction should be gradual.

One study compared the volume and variety of protozoan species in goats and guanacos.[40] In the goat, from one million to two million cells per gram of ingesta were found. Approximately 90% were in the small protozoa category. In the guanaco, there were about 1.3 million cells per gram of ingesta, and all the protozoa corresponded to the small species found in goats. It was estimated that the total volume of protozoa amounted to 9.4% of the rumen ingesta of the goat

(wet weight) and 5.3% of C-1 ingesta in the guanaco. The health of stomach microorganisms is vital to the nutritional status of camelids. This may have practical relevance when treating a sick llama that has been anorectic over a period of time with stomach atony. It is likely that much of the stomach microflora and -fauna has been destroyed. Transfaunation is commonly practiced in ruminants and has also been performed in llamas.

The question arises as to whether rumen contents from a bovine or ovine would be efficacious, or only the contents of a camelid stomach. Given a choice, it is best to transfaunate llamas with llama stomach contents, but bovine ingesta from fistulated donors have been used in llamas with apparent success. No studies have been conducted to determine the efficacy of commercial desiccated rumen microorganisms for reestablishing the flora of a llama stomach. Feces are not recommended for transfaunation, because stomach microorganisms will have been digested in the intestine and not be viable in feces.

Intestinal Function[12,14,18,19,24,28,64]

Intestinal digestion and absorption in the camelid are apparently similar to the process in ruminants. Camelids lack a gallbladder, so bile may flow continuously. The cecum and large colon do not function as primary fermentative chambers. The large intestine forms a spiral colon that diminishes in diameter by two-thirds within the coils. Fecal pellets are formed approximately halfway through the spiral. The diminution of size contributes to the formation of impactions in llamas. The transit time of particles through the gastrointestinal system varies with particle size but averages approximately four days.[13,43,44]

NUTRIENT REQUIREMENTS OF CAMELIDS[6,72,76]

The nutrient requirements of camelids are similar. Few detailed nutritional studies have been conducted. What is known is based on observations from feeding practices and extrapolation from data accumulated by studies of sheep, goats, and cattle. Because camelids may have a more efficient digestive system than ruminants, some of the figures may be in error. However, this purported efficiency may make a greater difference when the diet is poor. In the recommendations being made, a fair to good quality diet is assumed, so the margin of error in the recommendations may not be great.

Tables 2.4 to 2.7 provide a summary of estimated basic nutrients required by SAC. Perhaps the most significant column is the final one, which gives the amount of feed required to supply the general needs of a llama of a given weight. Feed amounts must, however, be correlated with forage composition (see Tables 2.15 and 2.16 later in the chapter) to ensure that adequate levels of all nutrients are provided.

The nutrient requirements for camels are listed in tables 2.8 to 2.11. The difference between OWC and NWC is the availability of the various forages.

Energy[103]

In a recent study of the energy requirements for llamas, metabolizable energy (ME) for maintenance was calculated to be 84.5 kcal/body weight $(BW)^{0.75}_{kg}$.[11] A previous study of maintenance energy was reported by Schneider et al. in Germany as being 61 kcal $ME_m/BW^{0.75}_{kg}$.[103] The investigators concluded that the daily maintenance digestible energy requirement was $71 \times BW^{0.75}_{kg}$ = kcal digestible energy (DE). The difference between these studies may be a difference in the method of extrapolation to zero energy retention for estimating metabolizable energy for maintenance or a difference in the quality of the diets fed during the studies. The range may be expected because the ME for maintenance for sheep is 72 to 107 kcal. Cattle have a higher ME value (110 kcal). Nutritionists should consider the 84.5 value as a more accurate estimation for

Table 2.4. Nutrient requirements for adult SAC, maintenance*,.**

Body weight kg/lbs	*Metabolic weight* $W^{0.75}_{kg}$	*Metabolizable energy*		*Crude protein g/d*	*TDN kg/d*	*Ca g/d*	*P g/d*	*Vitamin A IU/d*	*DM intake*	
		Mcal/d	*Mjoules/d*						*kg/d*	*% BW*
60/132	18.80	1.57	0.38	75	0.44	1.6	1.2	1884	0.82	1.37
80/176	26.74	1.95	0.54	94	0.54	1.9	1.5	2512	1.02	1.27
100/220	31.60	2.30	0.63	111	0.64	2.2	1.8	3140	1.21	1.21
120/275	37.40	2.64	0.71	127	0.73	2.4	2.0	3768	1.38	1.15
140/308	42.90	2.97	0.78	143	0.82	2.7	2.2	4396	1.55	1.11
175/385	47.50	3.28	0.96	158	0.91	2.9	2.4	5024	1.71	1.07
200/440	53.20		1.08	141		3.1	2.5			
225/495	58.10		1.17	154		3.3	2.7			

*Forage caloric density 1.91 kcal ME/d.
**Source: Fowler, 1998, and Anonymous, 2007, Nutrient Requirements for Camelids.

Table 2.5. Nutrient requirements for SAC during pregnancy.

Body weight kg/lb	*Metabolic weight* $W_{kg}^{0.75}$	*Metabolizable energy*		*Crude protein g/d*	*TDN kg/d*	*Ca g/d*	*P g/d*	*Vitamin A IU/d*	*DM intake*	
		Mcal/d	*Mjoules/d*						*kg/d*	*% BW*
8th month										
60/132	21.55	1.85	0.44	96	0.51	3.8	2.2	2730	0.47	1.61
80/176	26.75	2.33	0.57	119	0.65	4.2	2.5	3640	1.22	1.53
100/220	31.60	2.79	0.67	141	0.77	4.5	2.9	4550	1.46	1.46
120/265	37.40	3.24	0.77	161	0.90	4.8	3.2	5460	1.69	1.41
140/309	42.90	3.66	0.87	181	1.02	5.1	3.5	6370	1.92	1.37
160/353	44.48	4.08	0.98	200	1.37	5.4	3.8	7280	2.13	1.33
9th month										
60/132	21.55	2.16	0.52	117	0.60	3.7	2.1	2730	0.91	1.51
80/176	26.75	2.75	0.66	146	0.76	4.1	2.4	3640	1.15	1.44
100/220	31.60	3.32	0.79	172	0.92	4.9	3.2	4550	1.74	1.74
120/265	37.40	3.87	0.92	197	1.07	5.3	3.6	5460	2.02	1.68
140/309	42.90	4.40	1.05	221	1.22	5.7	4.0	6370	2.30	1.64
160/353	44.48	4.92	1.18	245	1.36	6	4.4	7280	2.58	1.61
10th month										
60/132	21.55	2.63	0.63	145	0.73	3.8	2.1	2730	0.92	1.53
80/176	26.75	3.34	0.80	180	0.93	4.4	2.8	3640	1.40	1.75
100/220	31.60	4.02	0.96	213	1.11	4.8	3.2	4550	1.68	1.68
120/265	37.40	4.68	1.12	244	1.30	5.2	3.5	5460	1.96	1.63
140/309	42.90	5.33	1.27	274	1.48	5.6	3.9	6370	2.23	1.59
160/353	44.48	5.97	1.43	303		5.9	4.3	7280	2.50	1.56

Table 2.6. Nutrient requirements for SAC, lactation*,.**

Body weight kg/lb	*Metabolic weight* $W_{kg}^{0.75}$	*Metabolizable energy*		*Crude protein g/d*	*TDN kg/d*	*Ca g/d*	*P g/d*	*Vitamin A IU/d*	*DM intake*	
		Mcal/d	*Mjoules/d*						*kg/d*	*% BW*
50/110	18.80	2.04	0.48	106	0.57	3.5	2.3	2675	1.07	2.14
80/176	26.75	2.42	0.58	124	0.67	3.8	2.6	4280	1.27	1.58
100/220	31.60	2.78	0.66	141	0.77	4.1	2.9	5350	1.45	1.45
120/265	37.40	3.11	0.74	157	0.86	4.3	3.1	6420	1.63	1.36
140/309	42.90	3.44	0.82	173	0.95	4.6	3.3	7490	1.80	1.28
160/353	44.48	3.75	0.90	188	1.04	4.8	3.5	8560	1.96	1.23

*Milk yield = 0.5 kg/d.
**Source: Anonymous, 2007, Nutrient Requirements of Camelids.

Table 2.7. Nutrient requirements for SAC, growth*,.**

Body weight kg/lb	*Metabolic weight* $W_{kg}^{0.75}$	*Metabolizable energy*		*Crude protein g/d*	*TDN kg/d*	*Ca g/d*	*P g/d*	*Vitamin A IU/d*	*DM intake*	
		Mcal/d	*Mjoules/d*						*kg/d*	*% BW*
40/88	15.90	1.52	0.36	84	0.52	3.9	1.9	4000	0.66	1.64
60/132	18.80	2.30	0.55	104	0.64	4.3	2.3	6000	0.96	1.60
75/165	25.50	2.67	0.64	122	0.74	4.9	2.9	8000	1.40	1.86
100/220	31.60	3.03	0.72	139	0.84	5.1	3.2	10000	1.58	1.58
120/275	37.40	3.37	0.81	155	0.93	5.4	3.4	12000	1.76	1.47
140/308	42.90	3.69	0.88	171	1.02	5.6	3.7	14000	1.93	1.42

*Weight gain 100 g/d.
**Caloric density at 1.91 kcal/kg.

Table 2.8. Nutrient requirements for SAC, pregnancy.

Body weight kg/lb	*Metabolic weight* $W_{kg}^{0.75}$	*Metabolizable energy*		*Crude protein g/d*	*Ca g/d*	*P g/d*	*Vitamin A 1000 IU/d*	*DM intake*	
		Mcal/d	*Mjoules/d*					*kg/d*	*% BW*
9th and 10th months									
300/661	72.1	157.42	37.66	234	16	14	25	4.29	1.43
350/771	80.9	167.45	40.18	263	21	16	27	4.81	1.37
400/882	89.4	202.86	48.53	290	23	18	30	5.32	1.33
450/992	97.7	211.80	50.67	317	26	20	34	5.80	1.29
500/1102	105.7	230.86	55.23	343	29	22	38	6.29	1.26
550/1216	112.6	247.83	59.29	368	31	24	42	6.75	1.23
600/1323	121.5	264.59	63.30	393	34	26	46	7.20	1.20
11th month									
300/661	72.1	188.98	45.21	292	26	20	34	5.36	1.79
350/771	80.9	220.87	52.84	328	29	22	38	6.10	1.74
400/882	89.4	243.99	58.37	363	31	24	42	6.64	1.66
450/992	97.7	266.52	63.76	396	34	26	46	7.26	1.61
500/1102	105.7	288.59	69.04	429	36	28	50	7.86	1.57
550/1216	112.6	311.05	74.14	462	39	30	53	8.44	1.53
600/1323	121.5	317.81	76.03	492	42	32	57	9	1.50

Table 2.9. Nutrient requirements for dromedary camels, maintenance*.

Body weight kg/lbs	*Metabolic weight* $W_{kg}^{0.75}$	*Metabolizable energy*		*Digestible protein g/d*	*Ca g/d*	*P g/d*	*Vitamin A 1000 IU/d*	*DM intake*	
		Mcal/d	*Mjoules/d*					*kg/d*	*% BW*
200/440	53.2	96.7**	23.14	144	8	7	9	2.50	1.48
250/550	62.9	114.3	27.35	169	10	9	11	2.96	1.18
300/660	72.1	130.8	31.3	195	12	10	13	3.39	1.11
350/772	80.9	147.3	35.23	218	14	11	15	3.80	1.08
400/882	89.4	162.6	38.91	241	17	13	17	4.20	1.05
450/992	97.7	177.7	42.51	264	18	14	19	4.59	1.02
500/1102	105.7	192.4	46.02	285	20	15	21	4.97	0.99
550/1216	112.6	206.5	49.41	307	21	16	23	5.34	0.97
600/1323	121.2	220.5	52.76	327	22	17	26	5.70	0.95

*Source: Wardeh, M.F., Nutrient Requirements of Camels.
**Energy and protein requirements may be increased by 25% to 40%, respectively, when camels work two to four hours/d.

Table 2.10. Nutrient requirements for dromedary camels, lactation, 5.0 kg/d (4.2% fat).

Body weight kg/lbs	*Metabolic weight* $W_{kg}^{0.75}$	*Metabolizable energy*		*Digestible protein g/d*	*Ca g/d*	*P g/d*	*DM intake*		*Vitamin A 1000 IU*
		Mcal/d	*Mjoules/d*				*kg/d*	*% BW*	
300/661	72.1	251.8	60.25	470	26	20	6.55	2.2	13
350/772	80.9	269.9	64.56	493	28	21	7	2	15
400/882	89.4	290	69.62	516	31	23	7.56	1.9	17
450/992	97.7	303.9	72.72	539	32	24	7.90	1.8	19
500/1102	105.7	320.4	76.65	560	34	25	8.33	1.76	21
550/1213	113.6	336.3	80.46	582	35	26	8.74	1.59	23
600/1323	121.2	352.4	84.31	602	36	27	9.15	1.5	26

Source: Wardeh, M.F., Nutrient Requirements of Camels.

Table 2.11. Nutrient requirements for dromedary camels, growth.

Body weight kg/lbs	*Metabolic weight* $W_{kg}^{0.75}$	*Gain/d g*	*Digestible protein g/d*	*TDN kg/d*	*Ca g/d*	*P g/d*	*DM intake*		*Vitamin A 1000 IU*
							kg/d	*% BW*	
100/220	31.6	500	249	27.7	15	9	2.75	2.75	6
150/331	42.9	500	298	35.5	16	10	3.46	2.30	9
200/441	53.2	750	394	50.54	21	15	4.64	1.86	12
250/551	62.9	750	427	57.99	22	17	5.78	1.93	14
300/661	72.1	750	454	65.23	23	18	6.50	2.17	15
350/772	80.9	1000	528	84.68	30	21	8.43	2.41	19
400/882	89.4	1000	542	92.68	31	24	9.23	2.31	19
450/992	97.7	1000	549	100.58	29	26	10.02	2.23	20
500/1102	105.7	1000	550	108.37	30	27	10.79	2.16	23

Source: Wardeh, M.F., Nutrient Requirements of Camels During Pregnancy.

North American feeding conditions.[11] Another study in South America arrived at 71 kcal/$BW_{kg}^{0.75}$.[102]

A clear understanding of the concept of metabolic weight is needed to follow the computations of Tables 2.4 to 2.11. In early studies of metabolism, researchers found that many reactions were not proportional to body mass but rather to body surface area. Smaller animals have a higher proportion of surface to mass than larger animals. The surface-to-mass ratio was calculated for many species and found to generally conform to the body weight in kilograms raised to the 0.75 power. Therefore, in nutritional calculations, metabolic weight is expressed as $BW_{kg}^{0.75}$, which simply means that a large animal has a smaller nutrient requirement per unit of body weight than a small animal.

Protein

The protein requirement for any animal has a direct relationship to the requirement for energy. The figure used here is the same as for sheep and goats: 31 g protein/Mcal DE.

Camelids may be able to digest protein and fiber more efficiently than ruminants. Efficiency has been demonstrated in some studies of forage with high fiber content and low protein levels, in other words, when a poor ration was consumed. On adequate diets, camelids apparently digest fiber and protein at an efficiency rate similar to that of sheep. In making recommendations for the nutrition of llamas, an optimum ration, not a marginal one, should be the goal.

Studies conducted in Colorado demonstrated that protein intake of 10% (100% DM) is adequate for maintenance in llamas. Higher levels, up to 16%, may be required for growth, lactation, and late gestation.[58,59]

Dry Matter[11]

All figures in Table 2.3, except those in the last two columns, are based on 100% dry matter. The amount actually fed depends on the moisture content of the forage eaten. For most sun-cured hay the moisture content is 10% to 12%, and for pasture plants, 50% to 75%.

Increased levels above adult maintenance are required for growth, lactation, late pregnancy, work, and inclement weather. None of these requirements have been ascertained specifically for llamas or alpacas. Extrapolating from the data on goats, the following recommendations may be made. For the last four months of pregnancy, 0.093 Mcal DE/$BW_{kg}^{0.75}$ should be added to maintenance requirements. For growth, based on 8.92 kcal DE/g gain, the figure is 1.78 Mcal DE/day. For lactation, based on 1,533 kcal DE/kg milk, if a 20 kg cria is consuming 10% of its body weight, the female must produce 2 kg of milk. The added energy requirement for that female would be 3.06 Mcal DE/day. A working pack animal may require a 75% increase in energy over the adult maintenance requirement.

Tables 2.4 to 2.11 should be used only as an estimate and until more specific data are reported in the literature. Variations in the composition of feeds may occur when the forage is grown on different soils and under different climatic conditions. Newer cultivars of forages may vary slightly in composition. Furthermore, biologic variation among individual animals must be considered. Observation and adjustment by the feeder is necessary to maintain a sound nutritional basis for feeding practices.

Water

The drinking behavior of SACs is to suck in water with the mouth slightly opened. Camelids have learned

to drink from all types of waterers and containers but are slow to become accustomed to automatic waterers, especially if it is necessary to press a lever to cause the water to flow. Running streams and ponds are natural for them. They are fastidious and may refuse to drink polluted water. This is interesting because packing llamas on the trail are inclined to urinate and defecate in a stream while they are drinking. Trail llamas will surprise the novice in that they frequently do not drink during the day, even with ample access to water.

Like all animals, camelids require adequate amounts of good quality water to sustain life, reproduce, work, and produce fiber and milk. Although Old World camelids are legendary in their ability to subsist on little or no water for long periods, they nevertheless need an adequate balance.

Only limited studies have been conducted on the water requirements of SACs.[99] It has been determined that the water content of the body of the llama is approximately 67%, compared with the goat at 60%. This varies with age and body fat content. In the same study, it was found that goats and llamas have similar water requirements. In an indoor experiment in a neutral environment, the water turnover rate (water intake by drinking plus food and oxidative water) was 62.1 ml/$BW_{kg}^{0.82}$/twenty-four hours in the llama and 59 ml in the goat. The amount of water consumed was 42.5 ml/$BW_{kg}^{0.82}$ in the llama and 40 ml in the goat. With a 40% reduction in food intake, water consumption of the llama decreased by 18%. When water was restricted, the llama was able to maintain food consumption. In similar studies of llamas on pasture, water turnover rates were double those of the indoor experiments: 122.2 ml/$BW_{kg}^{0.82}$.[91] Water requirements are directly related to caloric requirements (1 ml of water per 1 kcal of required energy). This later value is useful in calculating water requirements for sick animals.

Basic water requirements for camelids based on these studies are indicated in Table 2.12. This correlates quite closely with water requirements of other ungulate species. Water requirements can be met by free water consumption, moisture in feed, and water produced by the oxidative processes associated with energy metabolism. Camelids on lush pasture may obtain a majority of the water requirement from feed. Dry feed necessitates greater water intake. Salt and other mineral content of forage may affect the water requirement as well. Camelids will not break through ice to obtain water. The waterer must be heated in cold climates (Figure 2.4).

Water is lost through urine, milk, perspiration, and evaporation from the respiratory tract. Situations resulting in an increased production of any of these fluids will increase water needs.

Lactation places a significant demand on water consumption. No studies have been conducted on camelids, but in the goat, it is recommended that 1.43 L water be provided for each kilogram of milk produced.

Elevated ambient temperatures result in extra demands for water in SACs because they lack the ability of Old World camelids to withstand elevation

Table 2.12. Water requirements for llamas and alpacas.

Body weight (kg/lb)	*Body weight ($kg^{0.82}$)*	*Water turnover in a controlled environment (42.5 ml/$kg^{0.82}$/24 hr) (L or qt)*[a]	*Water requirements: 2 × turnover in controlled environment (Approx. water consumption in a moderate environment) (ml/kg/24 hr)*	*Water turnover on pasture (125 ml/$kg^{0.82}$/24 hr) (L or qt)*	*Water requirements: 2 × turnover on pasture (Approx. water intake in a moderate environment, including water in forage) (ml/kg/24 hr)*
10/22	6.61	1.0[b]	NA	NA	NA
20/44	11.66	2.0[b]	NA	NA	NA
40/88	20.59	0.88	44	2.57	129
50/110	24.73	1.05	42	3.09	124
75/165	34.48	1.47	39	4.31	115
100/220	43.65	1.86	37	5.46	109
125/275	52.42	2.23	36	6.55	105
150/330	60.87	2.59	35	7.61	101
175/385	69.07	2.94	34	8.63	99
200/440	77.06	3.28	33	9.63	96
225/496	84.88	3.61	32	10.61	94
250/550	92.54	3.91	31	11.57	93

Source: Grace et al. 1994.
[a]Actual consumption may be 1.5 to 2.5 times this figure, depending on weather, activity, and forage consumption.
[b]Based on milk consumption at 10% of body weight. Not calculated.

Figure 2.4. A heated waterer.

of body temperature. Old World camelids, particularly the dromedary, have evolved special physiologic mechanisms to deal with water deprivation. The camel can control the water content of feces and the concentration of urine, which reduces excretion of vital fluids during heat stress. Contrary to popular belief, the camel does not store water any more than any other species, but because of its ability to conserve body fluids, it need not drink water for considerable periods.

The dromedary is able to tolerate extreme dehydration and has been known to survive the loss of body water equal to 40% of its body weight. In contrast, a human losing 12% of the body's weight in water would be close to death. The small oval erythrocyte of the camel continues to circulate despite increased blood viscosity. Even after severe dehydration, the camel is able to drink sufficient water at one session to make up the deficit. Immediate intake of this amount of water would cause severe osmotic problems in humans or other animals of temperate climates. In the camel, water is absorbed from the stomach and intestines slowly, allowing establishment of equilibrium. The erythrocytes are able to avoid osmotic problems by swelling to 240% of their initial volume without rupturing. SACs may share this ability with Old World camels. In other species, erythrocytes can swell only to 150%.

Camels also can endure a wide range of diurnal fluctuation of body temperature, from 36.5°C to 42°C. The body acts as a heat sink during the hot times of the day, thus conserving vital water that would otherwise be lost through evaporative cooling. The excess heat is dissipated by conduction and radiation during the cool desert nights.

SACs should be given access to unlimited free-choice consumption of clean water. Intake will be adjusted to body needs. If water is restricted below the requirement, feed consumption will decrease, lactation will slow down or cease, and, in the extreme, hyperthermia may result.

Table 2.13. Mineral mixes for llamas and alpacas.

1. General mineral supplement
   50 lb trace mineral salt
   50 lb dicalcium phosphate
   50 lb dry powdered molasses
   10 lb ZinPro* 100
2. General + Se/Vitamin E
   To the general add
   Se at 90 ppm
   5 lb Vitamin E (227,000 IU/lb)
3. High phosphorus supplement
   50 lb trace mineral salt .
   25 lb dicalcium phosphate
   50 lb dry molasses
   25 lb monosodium phosphate
   10 lb ZinPro* 100
4. High phosphate + Se/Vitamin E
   To the High phosphate add
   Se at 90 ppm
   5 lb Vitamin E (227,000 IU/lb)

*ZinPro is a highly absorbable zinc supplement.
Source: Developed by Dr. LaRue Johnson, Fort Collins, Colorado.

Macrominerals[8,38,77,113]

Basic mineral metabolism is the same in both OWC and NWC.

In spite of the harsh environment and the sparse vegetation of some areas of the world where camelids exist, there are no reports of vitamin and mineral deficiencies or toxicities in camelids in their native habitat. This does not mean that they do not occur or that deficiencies may not develop in locations outside of their native lands. The problem may be a lack of diagnostic facilities or observation, or a failure to report. Camelids evolved with their environment and have developed the ability to cope with native soil types, climates, and forage quality. Out of the native habitat, they may not be able to cope. Most North American owners supply a granular mineral mix. A variety of mixes are available commercially, but formulations recommended by Dr. LaRue Johnson are excellent (Table 2.13).

A word of caution: In 1997 the U.S. Food and Drug Administration (FDA) banned the use of mammalian protein and bone meal products in feed for ruminant animals. This ban was enacted to prevent the inclusion of abnormal prion protein in ruminant feeds, potentially causing bovine spongiform encephalopathy (BSE). Camelids were not included in this act as ruminants, and there is no reason to believe that camelids are susceptible to BSE. However, prudence may dictate that where steamed bone meal is called for in a formula that an alternate source of calcium and phosphorus be used, such as dicalcium phosphate.

Calcium and Phosphorus

Calcium (Ca) and phosphorus (P) requirements are listed in Anonymous, 2007, Nutrient Requirements of New World Camelids, Table 2.6.[6] These are the two most important minerals for all vertebrates; 99% of body Ca and 80% of P are found in the skeletal system.[84] In order for Ca requirements to be met, the final diet should contain more than 0.3% Ca on a dry weight basis, and the Ca–P ratio (Ca:P) should not be less than 1.2:1.

It is unlikely that these minerals will be deficient in animals given free access to temperate climate pastures or hays, which usually contain ample Ca and P, with a satisfactory Ca-P ratio. In tropical climates, however, P deficiency is common in livestock on pasture.[71] Grains are deficient in Ca, and the Ca:P is poor. When concentrates are added to the diet, it is necessary to evaluate the diet to ensure that adequate Ca and P levels have been maintained.

Ca-P ratios of less than 1:1 and above 7:1 have been shown to decrease growth and feed efficiency in ruminants,[71] though ruminants tolerate more variation in Ca:P than other livestock. Excesses of either Ca or P in a ration may inhibit the availability of certain trace elements.

Trace Minerals[38,42,67,91,131]

There has been no scientific investigation of mineral nutrition and metabolism in alpacas and llamas. Laboratory analysis of whole blood or serum, and in some instances organ tissue, has been performed on groups of animals to help establish reference ranges, but the effects of deficiencies or toxicities are not precisely known. The following discussion is based on a literature review of pertinent minerals in cattle, sheep, and goats, plus what has been determined by veterinarians collecting laboratory samples from llamas/alpacas. Signs in camelids may differ as they do in many livestock species, horses, and companion animals. There is much to learn, but the basic information on similar species may be applied to beginning work with camelids.

Trace and macrominerals interact profoundly. Much is known about some relationships, such as that of copper (Cu) and molybdenum (Mo), or selenium (Se) and vitamin E. When a deficiency or toxicity of a mineral is suspected, the entire diet should be evaluated to determine if excesses or deficiencies of other minerals may be involved in the syndrome. Table 2.14 provides a conversion from conventional to Système International units (SI) and vice versa. The units are those in common usage in North America.

Cobalt[70,92]

Function

Cobalt (Co) is an essential component of vitamin B_{12} (cyanocobalamins), which is synthesized by rumen microorganisms and presumably by the microorganisms in the camelid stomach. B_{12} is involved in the metabolism of propionate to form glucose; thus, a deficiency may cause ketosis.

Table 2.14. Conversion of conventional units to Système International units for blood, plasma, serum.

Analyte	*Conventional units*	*Multiply by*		*SI units*
		Conventional to SI units	*SI units to conventional*	
All enzymes	U/L	0.0167	60	µKat/L
Albumin	g/dl (g%)	10	0.1	g/L
Copper	µg/dl (µg%)	0.1574	6.35	µmol/L
Iron	µg/dl (µg%)	0.1791	5.58	µmol/L
Lead	µg/dl (µg%)	0.04826	20.72	µmol/L
Magnesium	mg/dl (mg%)	0.4114	2.43	mmol/L
Mercury	µg/L	4.985	0.20	
Zinc	µg/dl (µg%)	0.1530	6.54	µmol/L
Calcium	mg/dl (mg%)	0.2495	4.01	mmol/L
Phosphate	mg/dl (mg%)	0.3229	3.10	mmol/L
Energy	calorie	4.184	0.239	Joule (J)

*Note: Additional conversion factors:
1 ppm (part per million) = 1 mg/kg = 1 µg/g = 0.454 mg/lb = 0.91 g/ton
1 ppm = 0.0001% = 0.002 lb/ton
0.1 ppm = 0.000011% = 0.0002 lb/ton = 0.09 g/ton = 0.0454 mg/lb
1 mg/g = 100 ppm
0.001 ppm = 1 ppb (part per billion)
1 fl oz = 29.6 ml
1 oz = 28.35 g
1 kg = 2.2 lb
1 pt = 473.2 ml = 1 lb (approx.)
1 qt = 946.4 ml = 1 L (approx.)
1 microgram (mcg or µg) = 0.000,001 g
1 nanogram (ng) = 0.000,000,001 g
1 picogram (pg) = 0.000,000,000,001 g

Interactions

A deficiency of Co eventually leads to thiamine and ascorbic acid deficiency and reduced glucose and ATP levels. Cobalt reduces the storage of Cu in the bovine liver. There may be possible antagonism from manganese (Mn), zinc (Zn), and iodine (I).

Signs of Deficiency

Serum levels of B_{12} are lowered, followed by loss of appetite and elevated pyruvate levels in ten to fourteen weeks. Other signs include ocular discharge, listlessness, anemia, ketosis, loss of condition, weakness, rough hair coat, and reduced rates of conception.

Signs of Toxicity

As with many of the trace minerals, signs of toxicity are similar to those of a deficiency and include slow growth rate, incoordination, rough hair coat, increased hemoglobin, and packed cell volume (PCV).

Overdosage with Co salts used in therapy or prevention of phalaris staggers may cause cardiomyopathy, gastroenteritis, and nephrosis. In an accidental overdose of $CoSO_4$ in alpacas, one cria became ill within minutes with colic, eructation, recumbency, and sweating. Death occurred within twelve hours. A second cria also died within twelve hours.

Diagnosis

Cobalt levels are not considered a satisfactory guide for the detection of deficiency and/or toxicity. Vitamin B_{12} levels are more accurate.

Treatment for Deficiency

A number of Co salts, including chloride, nitrate, and carbonate, provide ionic cobalt for therapy. Cobalt oxide is less easily absorbed and is used in Co bullets for intrarumenal deposition for long-term release. Cobalt sulphate monohydrate is also used and was the product involved in the accidental overdosage.

Cobalt is administered orally on a continuous basis. Rumen bullets are the most effective in cattle and sheep and may function for more than a year if not excreted. A new soluble glass bullet containing Co, Cu, and Se is proving to be effective in sheep and cattle. The anatomy and motility pattern of the camelid stomach precludes long-term retention of a bullet. Free-choice mineral/salt mixes must contain 0.005% to 0.02% Co but are effective only if the animal consumes the mix daily.

Calves from Co-deficient cows require supplementation within one to three months. A single injection of 2 mg B_{12} will suffice for up to twenty weeks.

Treatment for Toxicity

An overdose of Co used for therapy can only be dealt with by catharsis or by chelating the Co.[65] Dimercaptopropanol (BAL) and D,L-penicillamine were not effective as Co chelators. N-acetyl-L-cysteine (NAC), L-cysteine, glutathione, and 2,3-dimercaptosuccinic acid (DMSA) had some effect and could be considered as alternative Co chelators but are not readily available to the veterinary clinician. Calcium EDTA and diethylenetriaminepentaacetic acid (DTPA) were the most efficacious in preventing Co toxicity in rat studies.[5] Calcium EDTA (used to treat lead poisoning) is readily available and should be administered at a dose of 35 mg/kg intravenously TID for five days. Skip two days and repeat the series if necessary.

Acute Co toxicity causes cardiomyopathy and nephrosis; therefore, the animal should be monitored with a hemogram, and a serum chemistry panel should be done to detect cardiac muscle necrosis and decreased kidney function.

Prevention

Top-dressing a pasture with 0.35 to 5 kg of $CoSO_4$/hectare (0.14 to 2 kg/acre) is effective for a year but is expensive.

Rumen bullets (Co oxide, soluble glass, acid-base reaction cement) have all been effective. Salt/mineral mixes are satisfactory if the animals consume the material daily. Sheep have been drenched with Co chloride or sulphate (7 mg Co/week).

Copper[1,37,88,89,92,134]

Function

Copper (Cu) is an essential trace mineral for all mammals. Copper-containing enzymes (metalloenzymes) are essential for energy utilization, bone formation, digestion, detoxification, production of melanin, utilization of iron (Fe) in hemoglobin formation, amino acid metabolism, cellular respiration, and connective tissue formation. Excess Cu is stored in the liver.

Interactions

Adequate dietary levels of beta carotene are essential for absorption of Cu from the small intestine. Excessively high dietary intake of Ca may depress Cu absorption. Absorption may also be impaired if the diet contains excessive Zn (>100 ppm), Fe, cadmium (Cd), Se, Mo (>1 ppm), and sulfur (S) (>2,000 ppm). Lush growth of forages, particularly on alkaline soil, impairs absorption.

The inverse relationship of Cu and Mo must be understood and evaluated. Excessive Mo in the diet causes a deficiency of Cu. Copper salts may be used to

prevent the effects of excessive levels of Mo. A dietary ratio of Cu/Mo should be 6:1 to 10:1.

Signs of Deficiency

Signs of deficiency vary slightly if the problem is an absolute Cu deficiency or induced because of excessive intake of Mo. The signs include ataxia, anemia, depigmentation of the hair coat (dark animals become grayish; all hair colors lose brilliance and appear to be faded), immunosuppression, chronic diarrhea (only occurs in Mo excess), lameness (bone growth inhibited), decreased growth rate, infertility in both male and female, and decreased milk production. Cows tend to have more retained placentas and increased prevalence of abomasal ulcers.

Calves and lambs born to Cu-deficient dams may suffer from demyelinization of the spinal cord (enzootic ataxia, swayback), causing inability to suckle, incoordination, stiff gait, opisthotonus, and recumbency (lateral). Copper deficiency in sheep causes wool to lack normal crimp, have less tensile strength, and lose color in those breeds with dark wool.

Copper deficiency has been diagnosed in alpacas and llamas; however, when sheep and alpacas/llamas were grazed side by side in certain Cu-deficient areas of South America, sheep were affected but alpacas/llamas were not. Camelids may select forages that contain higher levels of Cu, may have lower requirements, or may have a different response to Cu deficiency. However, some suspect that excessive shedding and stringy hair fibers are a result of Cu deficiency. Having said the above, it is important to realize that subclinical Cu deficiency may have effects that have not been recognized.

Signs of Toxicity

Acute toxicity is usually caused by ingestion of $CuSO_4$ (foot baths, sprays, or accidental overdose). A severe gastroenteritis is produced, with hemorrhagic diarrhea the principle sign.[67]

Chronic toxicity is caused by long-term ingestion and liver storage of Cu. The actual syndrome is peracute. Sheep are notorious for Cu accumulation. If toxic levels continue to be ingested, the capacity of the liver is exceeded and excessive Cu enters the circulation, causing severe hemolysis and resulting in anemia, icterus, hemoglobinuria, hemoglobinemia, nephrosis (impaction of kidney tubules with hemoglobin), renal failure, and death. It is important to recognize that copper toxicity in SAC may not result in hemolysis and icterus, rather there is hepatic necrosis and signs of hepatic insufficiency. Additionally, liver storage capacity may be diminished by plant toxin ingestion or liver fluke damage to the liver.

High levels of Cu may inhibit respiratory cilia function in calves, leading to increased prevalence of respiratory disease. Copper toxicity has been reported in the llama. The clinical signs reported included anorexia, icterus, and hemoglobinuria with obvious hemolysis. Blood did not clot properly. The plasma Cu level was 0.83 µg/ml, and the AST (SGOT) was 1,500+. However, camelids do not appear to be as sensitive to Cu toxicity as sheep.

Diagnosis

Antemortem determination of plasma Cu levels is more precise than the determination of levels in serum because some Cu is lost into the clot. Plasma levels are not suitable for evaluating impending toxicity because excess plasma Cu is sequestered into the liver until hours before the acute hemolytic crisis occurs. Young calves are more susceptible than adults. Liver Cu levels may be obtained by liver biopsy. Use plasma for Cu determination rather than serum. Postmortem, hepatic necrosis and nephrosis are the primary lesions.

Treatment

Copper deficiency in cattle may be treated by feeding free-choice trace mineral/salt mix (0.1% to 0.5% Cu) or by adding Cu to the total diet at a rate of 10 mg Cu/kg of total ration. Sheep mineral/salt mixes should contain only 0.03% to 0.05% Cu. Total Cu ingestion should be 10 to 12 g/head/day. The recommended level of Cu in the total diet is 5 to 10 ppm. Oral Cu oxide granules, 20 to 50 g, or Cu needles, 10 to 20 g, last for twelve months. Soluble glass bullets are also effective. Injectable Cu glycinate (400 mg) subcutaneously lasts for two to three months but may produce a severe local reaction.

Once an acute hemolytic crisis occurs, there is little that can be done to treat Cu toxicity. Blood transfusion is indicated but may be ineffective if irreversible hemoglobin impaction of the kidney tubules has occurred.

Prevention

Prevention is the same as for treatment. In cattle, the minimum recommended level is 10 ppm, dry weight, total diet. Pugh[88,89] recommends 5 to 10 ppm in the diet for llamas. The dietary ratio of Cu/Mo should be 6:10. Pastures may be top-dressed with 2 kg $CuSO_4$/hectare (0.8 kg/acre).

Iron[92]

Function

Iron (Fe) is an integral component of hemoglobin.

Interactions

Cobalt, Cu, Se, and Zn deficiency may be induced by elevated Fe levels. Iron deficiency increases Co, lead (Pb), and Zn absorption. Infectious diseases that cause fever may reduce serum Fe.

Signs of Deficiency

Iron deficiency rarely occurs in adult animals. However, in an evaluation of the causes of anemia, Fe must be considered. Hemoglobin and PCV levels are not specifically diagnostic of Fe deficiency. Tests for Fe are more suitable (see diagnosis).

Iron deficiency was once thought to be a factor in the failure to thrive syndrome in alpacas and llamas because those animals tend to be anemic. That line of investigation has been discontinued. Milk contains relatively little Fe.

Signs of Toxicity

The author is aware of one instance of chronic Fe toxicity in horses fed alfalfa hay that had been sprinkle-irrigated with water containing excessive Fe. The signs were anorexia and poor growth. Ruminants seem to be quite resistant to Fe toxicity. Overdosage with $Fe_2(SO_4)_3$ may result in a heavy metal toxicity in horses, characterized by inappetence, depression, icterus, sweating, central nervous system (CNS) disturbance, dyspnea, and tremors with staggering.

Diagnosis

The most sensitive index of stored Fe is percent saturation of serum transferrin (1 ng/ml serum ferritin represents 8 mg of stored Fe). Iron-binding capacity may also be used to evaluate Fe deficiency. The anemia produced is microcytic-hypochromic.

In normal calves, the total Fe-binding capacity is 300 to 400 µg/dl and normal unbound Fe is 200 to 270 µg/dl. Elevated serum Fe occurs with Pb toxicity and hemolytic, hypoplastic, and aplastic anemias. Decreased serum Fe occurs with Fe deficiency, infections, and active erythropoiesis. Elevated total Fe-binding capacity occurs with blood loss and pregnancy, while decreased total Fe-binding capacity occurs with infections, hepatic fibrosis, uremia, and neoplasia.

Treatment

Incorporate $Fe_2(SO_4)_3$ into a mineral mix.

Prevention

Same as for treatment.

Selenium[17,30,46,80,87,90]

Function

Selenium (Se) is important as an antioxidant in tissues, in concert with vitamin E. The enzyme responsible for this effect is glutathione peroxidase (GSH-Px), which is a selenoprotein. The precise biochemical pathway is not understood and needn't concern the clinician. Suffice it to say that insufficient levels of GSH-Px affect growth, fertility, muscle, liver, and pancreas.

Interactions

Both Se and vitamin E have similar actions. Either may be individually deficient, and each may be protective against a deficiency of the other, but not completely. The author is aware of at least one llama that had a primary vitamin E deficiency. Excessive levels of a number of minerals, including silver (Ag), arsenic (As), Cd, Cu, mercury (Hg), Pb, Zn, and S, may induce Se deficiency.

Dietary Ca more than 0.8% may reduce Se absorption. Growing cattle on low-protein diets have a higher than normal Se requirement. Selenium deficiency and/or abundance in soil is a geologic phenomenon and is definitely regional.

Signs of Deficiency

On marginally deficient diets, cattle may have retained placentas; abortions; weak, stillborn, or lethargic calves that are unable to stand or suckle; reduced female fertility; cystic ovaries; metritis; delayed conception; erratic, weak, or silent estrus; poor fertilization; decreased male fertility; reduced growth rate; and immunosuppression.

Severe deficiency may result in myonecrosis of skeletal muscles (diaphragm, intercostals), resulting in dyspnea; the tongue, resulting in inability to eat; limb muscles, resulting in stiffness, paresis, and paralysis; and cardiac muscle (sudden death). Deficiency may also result in hepatic necrosis and diarrhea.

Periodontal disease involving the molars is unique to sheep. Other signs and syndromes in sheep are similar to those of cattle.

Llamas and alpacas develop signs and syndromes similar to those of cattle and sheep. Heat stress seems to increase the Se requirement; thus, animals on a marginal Se intake are more prone to develop hyperthermia. In areas where meningeal worm is endemic, ataxia and paralysis could be confused with the effects of white-muscle disease.

To summarize, it is evident that Se deficiency affects the reproductive process in all species, causing abortion, early embryonic death, retained placenta, and weakened neonates. In addition, muscle damage causes weakness, ataxia, and paralysis. Sudden death will result if cardiac myonecrosis occurs. Chronic diarrhea with poor growth and a decreased ability to mount an immune response may occur, resulting in decreased resistance to infection and parasites.

Signs of Toxicity

Signs of acute Se toxicity may be similar to the signs of deficiency, including poor conception rates, abortion, and developmental defects. Polioencephalomalacia in a llama with toxic levels of Se in the blood has been reported.[27]

Diagnosis

Clinical signs are not specific and may mimic numerous other diseases. Diets containing less than 0.1 mg/kg Se are considered inadequate in most large animal species. Whole blood Se concentrations of more than 0.1 µg/ml are considered adequate for large animals. A study was conducted using fifteen yearling llamas fed alfalfa hay. The hay contained 0.2 mg/kg of Se. Whole blood Se concentration was 0.17 ± 0.032 µg/ml. Glutathione peroxidase (GSH-Px) activity was 25.76 ± 6.53 mU NADPH oxidized/minutes/mg of hemoglobin.

In an excellent study[46] comparing serum Se concentrations in llamas at two different locations in North America, the researchers found "normal" levels in pregnant females to be 213 ng/ml (range 162 to 264 ng/ml). At parturition the levels were slightly lower, but not statistically significant.

Serum Se concentrations associated with healthy crias under North American farm conditions have a mean of 111 ng/ml (range 85 to 143 ng/ml). This may not be the lowest level for good health because the study didn't include crias that were obviously Se deficient, but the above levels are adequate.[48]

In herds of six or fewer llamas, researchers recommend that all animals be tested. Concentrations lower than 160 ng/ml in an individual animal should prompt supplementation. In larger herds, a herd mean of lower than 190 ng/ml should signal supplementation.[48]

Various laboratory determinations are used to evaluate the Se status of an individual or herd. Glutathione peroxidase contains the major active form of Se, and some laboratories prefer to analyze for this enzyme. Selenium is found primarily within the erythrocytes, so whole blood determination of Se provides maximum Se blood concentration. The third method is to determine serum Se concentrations. Any of the three methods are valid if the clinician is cognizant of the method of analysis used and evaluates accordingly. Each method has favorable and unfavorable attributes. A disadvantage of measuring glutathione peroxidase or whole blood Se is that GSH-Px and whole blood Se are added to the erythrocyte in the hematopoietic tissue before cells are released into the peripheral circulation, and none is added thereafter. The life span for erythrocytes is approximately 100 days. Thus, evaluating GSH-Px and whole blood Se concentrations provides a picture of the long-term Se status. On the other hand, serum Se concentrations respond quickly as Se is added to or subtracted from the diet, giving an indication of current Se intake. It is important to know which method of determination was used and to evaluate accordingly.

Treatment

The intramuscular or subcutaneous administration of selenium selenite may be indicated in the face of acute deficiency, but once myonecrosis occurs, treatment is of no value. The prognosis will be determined by the muscle groups affected and the extent of the necrosis.

Prevention

Provision of adequate Se levels in the diet of a herd may take a number of forms. Chemical analysis will establish whether or not purchased hay was grown on soil containing adequate levels of Se.

1. If hay is grown on the ranch, Se-deficient fields may be fertilized with sodium selenate. Pasture may also be fertilized to increase the Se content of the forage.
2. A mineral mix may be provided for free-choice consumption. The amount of Se in the mix should be determined by the degree of deficiency of soil Se in the region where the forage is grown or grazed. Mineral mixes must be protected from rain and snow to prevent leaching of the minerals from the mix. The problem with free-choice mineral mixes is that not all animals will consume a sufficient quantity to provide the necessary amounts of Se. Note that trace-mineralized salt may contain variable amounts of Se and other minerals. Check the bag label or obtain an analysis from the supplier or manufacturer.
3. Se can be incorporated in a grain mix. Most llamas/ alpacas do not require grain in the diet; however, many owners feed a token amount to ensure vitamin and mineral intake. Dr. James Vickers,[a] Michigan State University, stated, "Combining selenium with a grain mix and feeding it continuously is probably the best way to supplement selenium. When calculating dietary Se intake, only the supplemental amount of Se fed is taken into account."

The present recommendations for supplemental Se intakes are as follows:

Selenium (daily intake)	*Geldings and males*	*Breeding females*
Mildly deficient plants	0.5 mg	0.5–0.75 mg
Moderately deficient	1 mg	1–1.50 mg
Severely deficient plants	1.5 mg	2–2.25 mg

If a llama is fed approximately one lb. of grain mix per day, to provide 1.5 mg of Se from 1 lb. of grain mix, the mix must contain approxi-

mately 3 ppm Se. Only a few commercial llama supplements containing this concentration of Se are available. To formulate such a mix yourself, concentrated Se supplements must be added to existing mix formulas. Se supplements containing either 90 or 270 mg of Se/pound are available. Ten pounds of the 270-mg/pound product must be added to a ton (2,000 pounds) of feed to provide 1.5 mg Se/pound of grain, or use 30 lbs. per ton of the 90-mg/pound product. Both these products contain 3 ppm Se, which may make mill operators reluctant to mix the feed, because the maximum Se content in livestock feeds is 0.3 ppm. The reason for the concentration difference is not that the llamas require more Se, but they eat a far lower proportion of their diet as grain than do many other species.

4. Injectable Se/vitamin E products (BoSe [1 mg/ml, 2 ml/cria at birth], MuSe [5 mg/ml, 1 ml/100 kg]) may be administered periodically, but anecdotal information indicates that levels achieved by injection persist less than a month, and furthermore, in llamas there is usually a significant reaction at the injection site. Such use is not recommended as a long-term solution to the problem.
5. A few owners, in regions where the soil is extremely deficient in Se, feed each animal a measured amount of Se selenite in a sweet grain mix, to provide 1 to 3 mg of Se/day (Figure 2.5).

Zinc[66,92]

Function

Zinc (Zn) is an essential trace mineral that is a component of a number of zinc metallo-enzymes, including carbonic anhydrase, alcohol dehydrogenase, alkaline phosphatase, carboxypeptidase, RNA and DNA polymerases, and thymidine kinase. Deficiency of Zn affects protein metabolism, DNA and nucleic acid synthesis, skin and hair, growth, spermatogenesis, female fertility, and possibly skeleton abnormalities. Zinc is essential for normal wound healing and synthesis of collagen in bone.

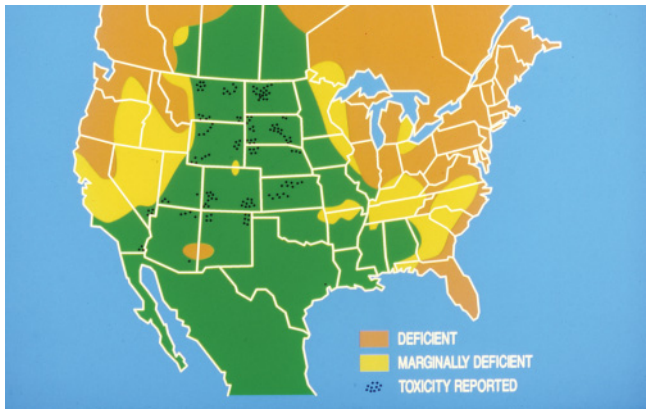

Figure 2.5. Regional selenium levels in North American forages.

Interactions

Excessive Zn adversely affects Ca metabolism and vice versa. Cadmium in quantities greater than 300 ppm in the diet reduces Zn absorption in calves. Zinc, Cu, and Fe are mildly antagonistic. Zinc deficiency inhibits utilization of vitamin A stores.

Plant uptake is influenced by soil Zn and moisture content, degree of compaction, nitrogen (N) and P levels, and pH. Zinc levels decrease as plants mature. Grasses accumulate more Zn than legumes when grown under the same conditions. Liver Zn is elevated by Cu excess.

Signs of Deficiency

Nutritionists have been unable to reproduce in the laboratory some of the clinical conditions associated with low Zn intake. Some go so far as to not label the signs as those of a deficiency, but rather as Zn-responsive conditions.

In cattle, effects are clinically more noticeable in juveniles than in adults. Reduced feed intake and growth rate, lethargy, reduced immune response, and parakeratosis have been reported. In adult cattle, subclinical effects are more likely to be seen. Signs include reduced conception rate, severely impaired maturation of sperm, and weak hoof horn, with increased susceptibility to interdigital dermatitis.

In addition to the signs noted in bovines, swine develop a severe form of parakeratosis and anemia. Skeletal effects include shortening and thickening of long bones.

Sheep tend to develop skeletal disorders, including enlarged joints, ankylosed hocks and knees, arched backs, and bowed hind limbs. They also suffer from impaired reproduction, parakeratosis, weak hoof horn, reduced food consumption, anemia, weight loss, wool loss, and wool eating.

In llamas/alpacas certain forms of dermatitis are Zn responsive. Llamas and alpacas are susceptible to many dermatologic conditions, some being steroid responsive, some Zn responsive, some antimicrobial responsive, and others that defy definitive diagnosis and/or therapy. Zinc-responsive parakeratosis is diagnosed on the basis of serum Zn levels and characteristic histopathology.

Signs of Toxicity

Calves are more susceptible to Zn toxicity than adults. Affected calves drink excessively and have

polyuria, diarrhea, and pica early in the course, followed by anorexia as they become emaciated, and develop pneumonia, ocular discharges, bloat, cardiac arrhythmias, and, ultimately, nystagmus, tonic clonic convulsions, and death.

In adult cattle, increased prevalence of arthritis and milk fever have been reported. High Zn levels interfere with Ca metabolism. Pancreatitis may develop if levels of dietary Zn are greater than 1,600 ppm.

In pigs signs include growth depression, enteritis, arthritis, and osteochondrosis.

In sheep, signs include reduced feed intake, diarrhea, weight loss, weakness, dehydration, pancreatitis, abomasitis, and reduced fat deposition.

In llamas/alpacas signs of toxicity have not been observed or reported.

Diagnosis

Serum alkaline phosphatase activity is reduced by Zn deficiency. No single sign, lesion, or chemical parameter is definitive. Zinc interacts with so many other trace minerals that it is difficult to determine which signs are attributable to a given mineral. Current recommendations are that dairy cattle should be provided with a diet containing more than 45 ppm dry matter intake with 0.3% Ca.

Treatment

Dietary intake should be greater than 0.05 mg Zn/kg body weight. Requirements increase during pregnancy and lactation. Males have a higher dietary requirement than females.

For cattle, provide free-choice or 40 g/head/day of trace mineralized salt containing 0.5% to 0.8% Zn. Although most salts of Zn are utilized by livestock, a form of Zn chelated to methionine (ZinPro 100)[b] is reported to make the Zn more available. A short-term loading dose may be accomplished by providing 250 ppm of zinc sulphate or carbonate in the diet. Zinc oxide bullets and implants have been used in sheep.

For llamas/alpacas, various Zn salts are incorporated into trace mineral mixes. Not all trace mineralized salt mixtures contain Zn, and they may contain variable amounts of other minerals. The bag label should be checked or an analysis obtained from the supplier or manufacturer.

Iodine

Function

Iodine (I) is an essential element for the synthesis of the thyroid hormones (thyroxine and triiodothyronine). Hypothyroidism has profound effects on a number of organ systems, especially the central nervous system, reproductive organs, and skeletal system. Thyroid dysfunction may take many forms; not all of them produce an enlargement of the thyroid gland (one form of goiter).

Interactions

Thyroid activity and the pituitary gland function in concert with each other. Hyperthyroidism causes diminished pituitary activity and vice versa.

Signs of Deficiency

Nothing is known about the signs of I deficiency in llamas or alpacas. However, in all other domestic animals, signs include mental retardation, stunted growth, lack of libido, delayed maturation of the genitalia, failure of production of ova, and arrested fetal development. There may or may not be visible or palpable goiter.

Diagnosis of Deficiency

An evaluation of thyroid function is necessary. It is important to know that normal T_3 and T_4 levels in SACs are approximately ten times that of cattle and sheep. The normal background serum concentration of I is 25 to 50 µg/dl.

Signs of Toxicity

Iodine has been used therapeutically for centuries. Most cases of toxicity are the result of overzealous treatment. Iodine is essentially stored only in the thyroid, and cessation of ingestion results in rapid return to normal. Excessive ingestion of I increases the secretions of the respiratory tract fluid, causing a nonproductive cough and a seromucous nasal discharge. Lacrimation, lameness, and desquamation of the skin epithelium are other signs of I toxicity.

Diagnosis of Toxicity

Serum I concentrations from 1,600 to 5,000 µg/dl have been recorded in toxic cases.

Treatment

The best treatment for deficiency is to provide iodized salt, either in a mineral mix or grain mix. In the case of toxicity, preventing ingestion of I salts will clear up the condition rapidly.

Prevention

Avoid excessive therapy with I-containing medications. Iodized salt should be provided. Llamas and alpacas are at the same risk as other livestock species in I-deficient areas of North America.

VITAMINS

No vitamin deficiency diseases have been reported in the South American literature. The North American situation may be entirely different. Veterinarians

should consider vitamin deficiency diseases that affect other domestic livestock.

B-Complex Vitamins

Vitamins in the B-complex category are usually produced in the gastrointestinal tract of ruminants and presumably in that of camelids as well. Little attention has been paid to these vitamins except when requirements are not met. Thiamine (B_1) and cyanocobalamin (B_{12}) may both be in this category. Thiamine is often administered therapeutically in diseases that affect the central nervous system. Thiamine deficiency is implicated in polioencephalomalacia cases in cattle and sheep. Polio has been diagnosed in a llama, but in that case it was ascribed to Se toxicosis.[27]

Vitamin K

Vitamin K is essential for production of clotting factors II, VII, IX, and X in the liver. A deficiency of vitamin K results in a prolonged prothrombin time. The main cause of vitamin K deficiency is the ingestion of warfarin-derived rodenticides. Another cause is the ingestion of moldy sweet clover hay, *Melilotus* spp. During spoilage, coumarin is converted to dicoumarol, which in the body acts in the same manner as warfarin. Llamas and alpacas develop a hemorrhagic syndrome just as other livestock species do.

Vitamin A (Retinol)[17]

Function

Vitamin A is necessary for normal growth, vision, and reproduction. Keratinization of the skin and mucous membranes, failure of regeneration of rhodopsin in the retina, and interference with osteoblast and osteoclast activity occur during hypovitaminosis A.

Interactions

Carotenes, found in plant material, must be converted in the small intestine to vitamin A activity. Species vary in the efficiency of that conversion. Cattle are poor converters. The status of llamas or alpacas in this regard is unknown.

Signs of Deficiency

Night blindness, fetal resorption, failure of spermatogenesis, stunted growth and defects in growth, and differentiation of epithelial tissues leading to keratinization are signs. In calves, deficiency results in increased intracranial pressure causing intermittent convulsions and depression.

Signs of Toxicity

Acute hypervitaminosis A has occurred in polar explorers who ate the liver of polar bears or seals. Acute poisoning causes increased intracranial pressure, resulting in headaches, vomiting, diplopia, visual field defects, fatigue, and anorexia in people. Chronic hypervitaminosis A has been observed in people and animals and is usually caused by overzealous vitamin A therapy. Signs include lethargy, colic, bone and joint pain, restlessness, brittle hoofs and nails, alopecia, and dry scaly skin, signs similar to those of a deficiency.

Diagnosis

In a feeding trial with fifteen yearling llamas fed alfalfa hay, the following data were recorded.[19] The hay contained 0.9 mg/kg of provitamin A. The vitamin A concentration in serum was 74.8 ± 5.5 µg/dl. Normal serum concentration of vitamin A in cattle is 25 to 60 µg/dl with less than 20 µg/dl considered indicative of deficiency. Concentrations more than 100 µg/dl may be indicative of toxicosis.

Treatment

Vitamin A requirements for all species range between 40 and 80 IU/kg of body weight (1 IU = 0.4 µg). The daily dietary requirement, in the form of carotene, is 0.12 mg/kg. Cattle with clinical signs are given 440 IU of vitamin A parenterally, then 4,000 IU/kg of the diet, orally daily.

Prevention

Fresh green pastures provide an abundance of provitamin A (carotenes, cryptoxanthins). Carotenes are lost during processing and storage. Vitamin A may be stored in the liver so daily ingestion is not needed unless there is no liver reserve. Vitamin A deficiency has not been reported in camelids; however, it should be given consideration because the situations leading to low vitamin A intake are present in camelid management. Llamas or alpacas will be deficient in vitamin A precursors if they are fed nothing but grass hay in a drylot situation or are fed alfalfa hay that has been stored for more than a year or that has been subjected to high temperatures or humidity. A dietary supplementation of 6,600 to 11,000 IU of vitamin A/kg of the diet has been recommended for llamas.[59]

Vitamin D[129]

Function

Vitamin D is the generic term for closely related steroids that function with Ca and P in bone metabolism. Vitamin D occurs in two forms. Vitamin D_2 (ergocalciferol, D_2), is produced by irradiation of ergosterol in plant material. There is little or no D_2 in pasture plants, but sun-cured hay is a good source of D_2. All mammals, except New World nonhuman primates, are able to utilize D_2. Vitamin D_3 (cholecalciferol, D_3) is found only in animal tissues (fish oils, liver). It is

produced by ultraviolet irradiation of 7-dehydrocholesterol (derived from cholesterol) in the skin of animals when exposed to sunlight.

D_2 and D_3 are the precursors to ultimate hormonal vitamin D activity. Each form of vitamin D follows its own metabolic pathway, but with similar chemical reactions. The first step is the conversion, in the liver, to the 25-hydroxy derivative (25-OHD). Under the action of parathormone, 25-OHD is converted to 1,25-dihydroxy cholecalciferol (the active form of vitamin D) in the kidney. Hypofunction of either the liver or kidney may lead to hypovitaminosis D.

The active form of vitamin D produces an effect at three sites:

1. The epithelial cells of the intestine, which are involved in the active transport of Ca ions across the intestinal barrier into the blood. It also is involved in the activation of the intestinal epithelial cell transport system, which increases absorption of both Ca and PO_4.
2. Within the epithelial cells of the renal tubules to bring about resorption of PO_4 and perhaps Ca.
3. In bone, to mobilize Ca. Vitamin D activity is responsible for maintaining the circulating levels of Ca in the blood, and vitamin D and parathormone acting together mobilize Ca and PO_4 from bone.

Milk does not naturally contain vitamin D. Thus, sucklings deprived of exposure to sunlight (because they are kept inside or there is prolonged inclement weather) may be at risk for hypovitaminosis D. This may be the pathogenesis of rickets in suckling or weanling alpacas and llamas.

Signs of Deficiency

Rickets is the syndrome caused by an absolute deficiency of vitamin D in growing animals.[31,47,71,129] In adult animals, metabolic bone disease results in various manifestations (see discussion later in this chapter). Alpaca and llama crias generally develop clinical rickets from four to seven months of age. Early signs include lameness, reluctance to run and play, cessation of growth, and excessive recumbency (Figures 2.6 to 2.11). Angular limb deformity is seen in some animals, but not all. The carpi, tarsi, and fetlock joints are swollen, as are the costochondral junctions of the ribs.

Signs of Toxicity

As with vitamin A, ingestion of excessive vitamin D causes serious to lethal poisoning. Hypervitaminosis D is usually caused by overzealous therapy. Vitamin D is stored in the liver, but when the storage capacity is exceeded, metastatic calcification may occur in tendons, ligaments, cardiac muscle, kidneys, and greater vessel walls. Signs of poisoning include lameness, a heart murmur, weight loss, and polyuria. Areas of calcification in muscles and tendons may be

Figure 2.6. An alpaca cria with rickets, standing humpbacked and reluctant to walk.

Figure 2.7. An alpaca cria with rickets, spending most of her time recumbent and reluctant to play with other pen mates.

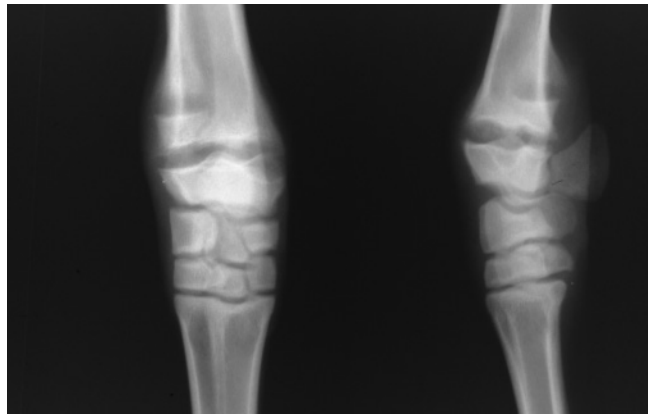

Figure 2.8. Radiograph of the carpus of a rachitic alpaca shows wide physis, metaphyseal flaring, and thinning of the cortices.

Figure 2.9. Rachitic rosary at the costochondral junction in rickets.

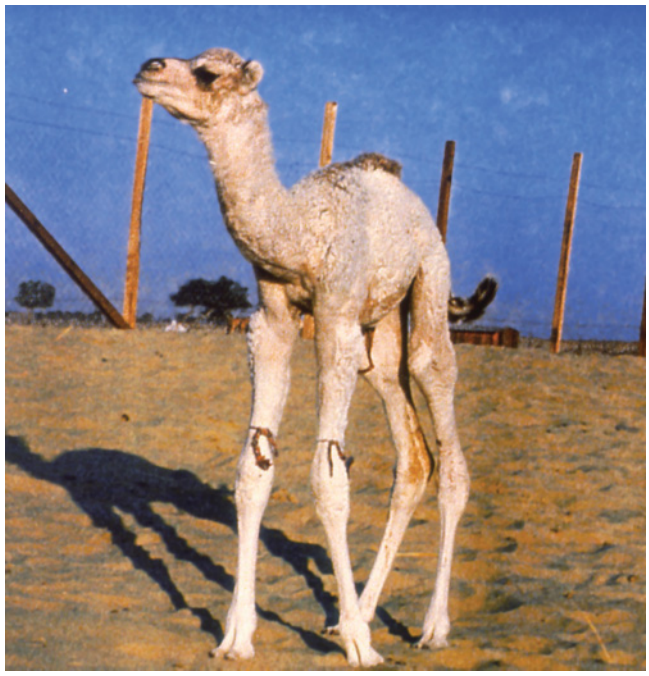

Figure 2.10. Camel calf with rickets.

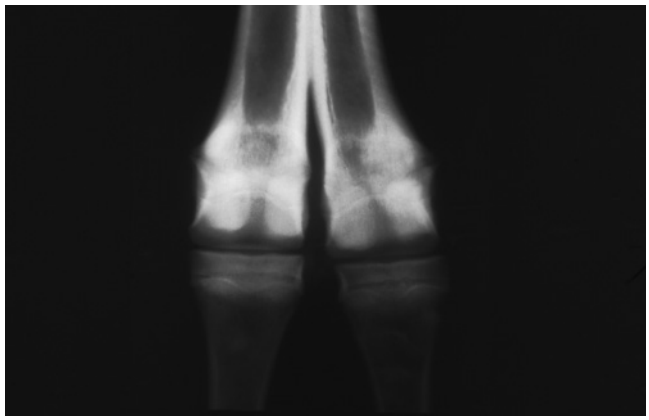

Figure 2.11. Radiograph of the fetlock of a camel calf with rickets. Note the widened physes and flaring.

palpated. Calcification lesions may be seen on radiographs.

The definitive diagnosis is the measurement of serum concentrations of 25-hydroxyergocalciferol or 25-hydroxycholecalciferol or 1,25-dihydroxycholecalciferol. Treatment is not effective once severe calcification has occurred.

Diagnosis

Signs are highly suggestive, along with the age of the cria and the weather patterns of the previous few weeks. Rickets is common in regions of the country with prolonged inclement weather and constant cloud cover. Radiographs are definitive (Figures 2.8 and 2.11). The physes are wider than normal. There is metaphyseal flaring, cortical thinning, and, possibly, angular limb deformity. Radiographs should be taken of the carpus and tarsus. Serum vitamin D (25-hydroxycholecalciferol) concentrations of less than 15 nmol/L are supportive of a diagnosis of hypovitaminosis D.[129]

Not all forms of angular limb deformity are caused by rickets. Comparing the radiographic picture of rickets with Figure 22.5 illustrates the difference. The serum concentration of P and Ca should be evaluated to determine whether or not P supplementation is indicated.

Treatment of Deficiency

A rachitic cria should be given an intramuscular or subcutaneous injection of a repositol, aqueous emulsion of vitamin D_3. Crias up to 30 kg should receive 1 to 2.5 ml, and weanlings 2.5 to 5 ml.[56] Although this therapy may be sufficient in many cases, if there is an absolute deficiency of P, that must be corrected by daily oral administration of sodium phosphate in corn syrup or molasses (1/8 to 1/4 teaspoonful [0.5 to 1 g]).

Prevention

Oral administration of 15,000 to 30,000 IU of vitamin D every two weeks during winter or periods of inclement weather has proven effective in preventing rickets in llamas and alpacas.[71] A product that is used in the Pacific Northwest of the United States is a vitamin A, D, and E gel containing 500,000 IU of vitamin A, 100,000 IU of vitamin D_3, 100 IU of vitamin E, and 3,000 µg of vitamin B_{12}/15 ml of the gel. The dosage for llama and alpaca crias is 5 ml orally every two weeks. The problem is usually solved once the cria is consuming well-cured hay or is exposed to adequate sunlight. Ingestion of a mineral mix incorporating sodium phosphate will provide adequate P if the cria eats it.

An alternative preventive modality is to prepare a pelleted supplement that contains 6,000 IU of vitamin D_3/pound of pellets. This is then supplemented at a level of 0.5 lb. of pellets/100 lbs. of body weight. During the winter months, one veterinarian also administers

an injectable vitamin D (1 ml) to crias shortly after birth and then administers an oral paste every two to three weeks.[c]

Vitamin E (α Tocopherol)

Function

Vitamin E serves as a biologic antioxidant to help maintain cellular integrity. It works within the cell membrane to prevent formation of lipid hydroperoxide and subsequent lipid peroxidation of the cell membrane.[17]

Interactions

Vitamin E and Se have much the same ultimate action; however, Se, as a component of GSH-Px, converts hydrogen peroxide and lipoperoxides that are already formed to less harmful alcohols and water.[17] Thus, vitamin E prevents formation of free radicals while Se corrects free radicals. The two compounds function synergistically.

Signs of Deficiency

Same as for Se deficiency.

Diagnosis

It is difficult to separate vitamin E and Se deficiencies unless laboratory data can be obtained. Data were collected on fifteen yearling llamas being fed alfalfa hay containing 5 mg/kg of α tocopherol. The serum α tocopherol concentration was 128 ± 41.7 µg/dl. Normal α tocopherol plasma concentration of large animals is 100 to 200 µg/dl. Necrosis of cardiac and skeletal muscle groups is observed at necropsy.

Treatment

Therapy does not correct muscle necrosis but may prevent further necrosis. The most active form of α tocopherol is d-l α tocopherol acetate, which is the form in most injectable vitamin E preparations. However, the amount present in commercial injectable formulae may be insufficient to correct a primary vitamin E deficiency.

Prevention

Fresh green pasturage supplies sufficient vitamin E; however, the vitamin E content of forage diminishes rapidly with plant maturity and is markedly reduced by harvesting, heat, moisture, and processing. Vitamin E is not stored in the liver as are vitamins A and D, so daily ingestion is necessary. The National Research Council recommends 15 to 60 IU (1 mg of d-l α tocopherol acetate = 1 IU of vitamin E activity) of vitamin E/kg of dry matter for beef and dairy cattle. The hay fed to the yearling llamas in the study reported above had 5 mg/kg (IU) of α tocopherol on a dry matter basis, so the diet was probably deficient in vitamin E.

Although vitamin E and Se work synergistically and a deficiency of one may be nullified by adequate amounts of the other, it is possible to have an absolute deficiency of either. Although the end result is the same, from a management standpoint, it is vital to know which supplement is indicated.

FEEDING CAMELS[73,79,131,133]

Feeding Racing Camels

Camel racing has become a popular sport in the Middle East countries and Australia. These animals are bred for speed and require a special diet to cope with the energy demands of racing. Races are 3 to 10 K long. Usually only females are raced.

The diet during the racing season in the Middle East usually consists of cereal grains, hay, dried dates, fresh cow's milk, and fresh alfalfa (*Medicago sativa*) (Table 2.15). The combination usually contains 15% crude protein, 45% fat, 21% acid detergent fiber, and 10 Mj (41.8 Mcal) metabolizable energy.[63]

An interesting side note is that at the beginning of the racing season camels are purged, allegedly to clear the intestines of toxins. Traditionally, Bedouins feed a halophyte plant with the local name of "harm" (*Zygophyllum qatarense*). The plant is fed for one or two weeks. The purgative action of the plant may be related to its high sulfur content (6.03%).[133] The purging is repeated at the end of the racing season. Alternatively, a single dose of Epsom salts (Magnesium sulfate) (1 kg/camel) is administered.

North American Forages

An informed veterinarian needs to know the type and quality of forages locally available and the variations that may take place seasonally. No locale is precisely the same as another. The best variety for each location is determined by the grower, but in the United States basic information may be obtained from Cooperative Extension agents associated with the land grant institution in the state. Each region originally had native grasses, but many of these were overgrazed or poorly managed into oblivion. Now pasture managers have introduced grasses from the far parts of the world, and decades of development have produced cultivars that are highly productive. The composition of forages is found in United States-Canadian Tables of Feed Composition, Atlas of Nutritional Data on Composition of United States and Canadian Feeds, and Latin American Tables of Feed Composition.[2,3,70]

Eleven grasses and three legumes that provide forage for llamas and alpacas were selected for the following discussion. Table 2.16 provides percentages for selected nutrients in grasses used in pastures and

Table 2.15. Nutrient composition of feeds for racing camels.

Ingredient	*Dry matter g/kg*	*Net energy*		*Digestible protein g/kg*	*Crude fiber g/kg*	*Ash g/kg*	*Form*
		Mcal/kg	*Mj/kg*				
Barley (*Hordeum* spp.)	860	2.8	11.78	73.96	43	21	Whole grain
Green alfalfa (*Medicago sativa*)	240	0.47	1.96	31.20	4.67	24.70	Early flowering
Fresh milk	903	0.62	2.59	32	0.13	7.45	Cow's milk 3.5% fat
Dates (*Phoenix* dactiflora)	903	2.83	11.83	17.16	128.20	26.19	Dried fruit, seedless
Rhodes grass (*Chloris gayana*)	850	1.42	5.95	32.30	289	66.30	Mature, hay
Pellets	900	2.57	10.75	95	183	85	Camel racing pellets
Mixed grain	880	2.63	11	97.40	44	20.24	Cracked

Table 2.16. Composition of selected grasses used for pasture and hay for llamas and alpacas.

Grass	*Type*	*Dry matter (%)*	*Crude protein (%)*	*TDN (%)*	*Digestible energy (Mcal/kg)*	*Crude fiber (%)*	*Ca (%)*	*P (%)*	*Ca:P*	*Mg (%)*	*Cu (%)*	*Co (%)*
Bahiagrass	Pasture											
Paspalum notatum	Vegetative	30	2.6	16	0.70	9.0	0.14	0.06	2.3:1	—	—	—
	Dry	100	8.9	54	2.38	30.4	0.46	0.22	2.1:1	—	—	—
	Hay											
	Sun cured	91	7.4	46	2.05	29.2	0.46	0.20	2.3:1	—	—	—
	Dry	100	8.2	51	2.25	32.0	0.50	0.22	2.3:1	—	—	—
Bluegrass,	Pasture											
Kentucky	Early veg.	31	5.4	22	0.98	7.8	0.12	0.12	1:0	0.06	5.0	—
Poa pratensis	Dry	100	17.4	72	3.17	25.3	0.33	0.34	1:0	0.17	14.0	—
	Mature	42	4.0	23	1.03	13.4	—	—	—	—	—	—
	Dry	100	9.5	56	2.47	32.2	—	—	—	—	—	—
	Hay											
	Sun cured, full bloom	92	8.2	52	2.32	29.9	0.29	0.22	1:3	0.14	9.0	—
	Dry	100	8.9	57	2.51	32.5	0.25	0.25	1:3	0.16	10.0	—
Brome, smooth	Pasture											
Bromus inermis	Early veg.	30	6.3	22	0.95	6.7	0.16	0.13	1:2	0.09	—	—
	Dry	100	21.3	73	3.22	22.8	0.45	0.45	1:2	0.32	—	—
	Mature	55	3.3	29	1.28	19.1	0.09	0.09	1:6	—	—	—
	Dry	100	6.0	53	2.34	34.8	0.16	0.16	1:6	—	—	—
	Hay											
	Sun cured, midbloom	90	13.2	51	2.23	28.8	0.25	0.20	1:3	0.17	9.0	0.08
	Dry	100	14.6	56	2.47	31.8	0.28	0.22	1:3	0.18	10.0	0.09
	Sun cured, mature	93	5.4	48	2.12	29.8	—	—	—	—	—	—
	Dry	100	5.8	52	2.29	32.2	—	—	—	—	—	—
Fescue, tall	Pasture											
Festuca	Vegetative	29	4.2	19	0.85	7.1	0.15	0.11	1:4	—	—	—
arundinacea	Dry	100	14.5	67	2.91	24.6	0.51	0.37	1:4	—	—	—
	Hay											
	Sun cured, early	91	18.4	58	2.57	21.5	—	—	—	—	—	—
	Dry	100	20.2	64	2.82	23.6	—	—	—	—	—	—
	Sun cured, mature	90	8.3	50	2.22	29.3	—	—	—	—	—	—
	Dry	100	9.2	56	2.47	32.6	—	—	—	—	—	—

Table 2.16. ***Continued***

Grass	*Type*	*Dry matter (%)*	*Crude protein (%)*	*TDN (%)*	*Digestible energy (Mcal/kg)*	*Crude fiber (%)*	*Ca (%)*	*P (%)*	*Ca:P*	*Mg (%)*	*Cu (%)*	*Co (%)*
Oats	Hay											
Avena sativa	Sun cured	91	8.5	56	2.46	27.8	0.22	0.20	1:1	0.24	14.0	0.07
	Dry	100	9.3	61	2.69	30.4	0.24	0.22	1:1	0.26	15.0	0.07
Orchardgrass	Pasture											
Dactylis glomeratus	Early veg.	23	4.3	17	0.74	5.8	0.13	0.13	1:0	0.07	2.0	—
	Dry	100	18.4	72	3.17	24.7	0.58	0.54	1:1	0.31	7.0	—
	Milk stage	35	2.9	19	0.82	12.3	—	—	—	—	—	—
	Dry	100	8.4	53	2.34	35.2	—	—	—	—	—	—
	Hay											
	Sun cured, early	89	13.4	58	2.55	27.6	—	—	—	—	—	—
	Dry	100	15.0	65	2.87	31.0	—	—	—	—	—	—
	Sun cured, late	91	7.6	49	2.16	33.6	0.35	0.32	1:1	0.15	12.0	0.42
	Dry	100	8.4	54	2.38	37.1	0.39	0.35	1:1	0.17	13.0	0.46
Ryegrass, perennial	Pasture											
	Fresh veg.	27	2.8	18	0.80	6.2	0.15	0.07	2:1	—	3.0	0.02
Lolium perenne	Dry	100	10.4	68	3.00	23.2	0.55	0.27	2:0	—	13.0	0.06
	Hay											
	Sun cured	86	7.4	55	2.43	26.1	0.56	0.28	2:0	—	—	—
	Dry	100	8.6	64	2.82	30.3	0.65	0.32	2:0	—	—	—
Sudangrass	Pasture											
Sorghum sudanense	Early veg.	18	3.0	12	0.55	4.1	0.08	0.07	1:1	0.06	—	—
	Dry	100	16.8	70	3.09	29.0	0.43	0.41	1:0	0.35	—	—
	Hay											
	Sun cured, full bloom	91	7.3	51	2.25	32.8	0.50	0.28	1:8	0.47	—	—
	Dry	100	8.09	56	2.47	36.0	0.55	0.30	1:8	0.51	—	—
Timothy	Pasture											
Phleum pratense	Late veg.	26	4.8	19	0.04	8.5	0.10	0.08	1:3	0.04	3.0	0.01
	Dry	100	18.0	72	3.17	32.1	0.33	0.28	1:2	0.14	11.0	0.04
	Hay											
	Sun cured, early	90	13.4	59	2.61	25.1	0.59	0.30	2:0	0.13	—	—
	Dry	100	15.0	66	2.91	28.0	0.66	0.34	2:0	0.14	—	—
	Sun cured, milk	92	6.4	47	2.10	31.0	0.26	0.17	1:5	0.11	—	—
	Dry	100	7.0	52	2.29	33.9	0.28	0.18	1:6	0.12	—	—
Wheatgrass, crested	Pasture											
	Early veg.	28	6.0	21	0.92	6.2	0.13	0.10	1:3	0.08	—	—
Agropyron desertorum	Dry	100	21.5	75	3.31	22.2	0.46	0.34	1:4	0.28	—	—
	Hay											
	Sun cured	93	11.5	49	2.17	30.5	0.31	0.20	1:6	0.15	—	—
	Dry	100	12.4	53	2.34	32.9	0.33	0.21	1:6	0.16	—	—

Note: Data taken from *US and Canadian Tables of Feed Composition*, National Academy Press, Washington, D.C., 1982. These data are averages of thousands of samples. Forage may vary regionally. These figures should be used only as a guide. Forage analysis is necessary for accurate assessment.

Early veg. = early vegetative stage, late veg. = late vegetative stage, midbloom = hay cut during midflowering stage, sun cured = dried in the field, milk stage = cut when seeds are first formed, dry = plant material converted to 100% dry matter.

as hay in Canada and the United States. The table compares the forages in the pasture and at 100% dry matter. Most pasture grasses are only 30% dry matter, pastured legumes are 20% to 25% dry matter, and hays are approximately 90% dry matter. Table 2.17 supplies similar information on three legumes for comparison.

Protein levels in legumes are generally higher than in grasses, but protein content drops in all forages in both mature pasture stands and late-cut hay. Total

Table 2.17. Composition of legumes for pasture and hay for llamas and alpacas.

Grass	*Type*	*Dry matter (%)*	*Crude protein (%)*	*TDN (%)*	*Digestible energy (Mcal/kg)*	*Crude fiber (%)*	*Ca (%)*	*P (%)*	*Ca:P*	*Mg (%)*	*Cu (mg/kg)*	*Co (mg/kg)*
Alfalfa	Pasture											
Medicago	Late veg.	21	14.3	13	0.59	4.9	0.48	0.07	6.9:1	0.07	2.0	0.03
sativa	Dry	100	20.0	63	2.78	23.0	1.90	0.30	6.3:1	0.27	10.0	0.13
	Full bloom	25	3.5	14	0.61	7.7	—	—	—	—	—	—
	Dry	100	14.0	55	2.43	31.0	—	—	—	—	—	—
	Hay											
	Sun cured, late bloom	90	12.6	47	2.06	28.8	1.13	0.20	5.7:1	0.28	13.0	0.29
	Dry	100	14.0	52	2.29	32.0	1.25	0.22	5.7:1	0.31	14.0	0.33
	Sun cured, mature	91	11.7	46	2.01	34.4	1.03	0.17	6.1:1	0.24	13.0	0.08
	Dry	100	12.9	50	2.21	37.7	1.13	0.18	6.3:1	0.27	14.0	0.98
	Sun cured, leaves	89	20.6	64	2.84	15.8	2.27	0.24	9.5:1	0.36	10.0	0.19
	Dry	100	23.1	72	3.17	17.7	2.54	0.27	9.4:1	0.40	11.0	0.22
Clover, white,	Pasture											
ladino	Early veg.	19	5.2	13	0.58	2.7	0.27	0.08	3.4:1	0.08	—	—
Trifolium	Dry	100	27.2	68	3.00	14.0	1.27	0.35	3.6:1	0.37	—	—
repens	Hay											
	Sun cured	90	19.7	58	2.57	19.1	1.21	0.28	4.3:1	0.43	—	—
	Dry	10	22.0	65	2.87	21.2	1.35	0.31	4.4:1	0.48	—	—
Clover, red	Pasture											
Trifolium	Early bloom	20	3.8	12	0.60	4.6	0.30	0.07	4.3:1	0.09	2.0	0.03
pratense	Dry	100	19.4	68	3.04	23.2	1.64	0.36	4.6:1	0.51	9.0	0.14
	Hay											
	Sun cured	89	14.2	49	2.15	25.5	1.35	0.22	6.1:1	0.38	10.0	0.14
	Dry	100	16.0	55	2.43	28.8	1.53	0.25	6.1:1	0.43	11.0	0.16

Note: Early veg. = early vegetative stage, late veg. = late vegetative stage, sun cured = dried in the field, dry = plant material converted to 100% dry matter.

Table 2.18. Nutrient composition of legumes cut at 1/10 bloom.

Legume	*Productivity (lb DM/acre)*	*TDN (%)*	*Crude protein (%)*	*Crude fiber (%)*	*Fat (%)*	*Calcium (%)*	*Phosphorus (%)*	*Potassium (%)*
Alfalfa	4632	57.4	17.9	33.9	1.54	1.41	0.28	1.42
Red clover	4008	62.9	15.9	26.2	1.54	1.36	0.22	1.36
White clover	2044	77.4	21.8	17.4	1.72	1.55	0.28	1.96

Note: 1/10 bloom based on 100% of dry matter.

digestible nutrient percentage (TDN) is similar in both legumes and grasses. Crude fiber is similar or slightly higher in grasses. The Ca-P ratio of grasses is closer to the recommendation of 2:1.

Grasses[3,4,5,15,20,33,43,62,94,96,97,98,101,102,110]

Grasses are the most dominant and by far the most economically important plant family in the world (600+ genera, 9,000 to 12,000 species). All camelids depend upon grass, either as a standing crop or as cured hay, for the majority of their nutritional needs. These highly adaptable animals are capable of utilizing grass wherever it is found. A comparison of grasses cut at the same stage of maturity is found in Table 2.18. The relationship between composition and maturity is illustrated in Figure 2.12.

Bahiagrass *Paspalum notatum.* Bahiagrass is a deep-rooted, warm season perennial that is native to South America. It was introduced into North America in 1913. It is principally adapted to the coastal area of the southern United States, as are coastal bermudagrass and dallisgrass. However, bahiagrass tolerates a broader range of soil conditions than other southern-adapted grasses. Bahiagrass seed head stems may grow to a height of 60 cm (24 in.), but more commonly, the leaves spread out near the ground.

Coastal Bermudagrass, *Cynodon dactylon* (Wiregrass, Devilgrass, Vinegrass, Dogtoothgrass). Bermudagrass was

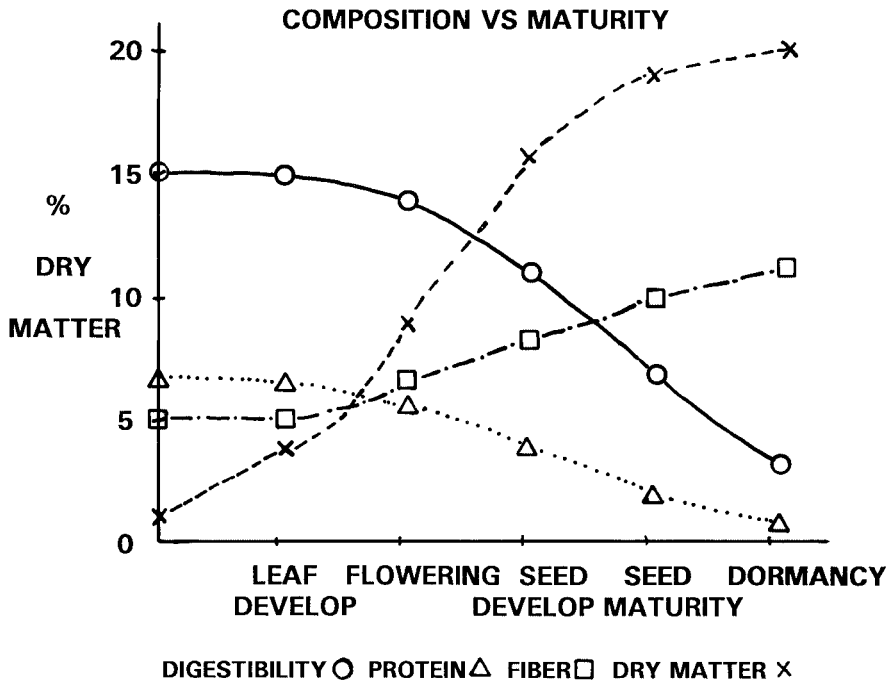

Figure 2.12. A diagram illustrating the change in composition with the stage of maturity of grass. Legumes have a similar change, although they have different values for the parameters.

introduced from the Old World and is a hardy perennial that forms heavy sod by means of extensive creeping rhizomes and stolons. It is popular as lawn sod in areas of heavy human traffic, though it becomes dormant and brown during the winter. Coastal bermudagrass (a variety) is a popular pasture forage in the southern and southeastern United States and is harvested for hay.

Kentucky Bluegrass, *Poa pratensis.* Kentucky bluegrass is sometimes thought to be native to the limestone soils of the northeastern United States but was actually introduced from Europe. It is a cool season, long-lived perennial grass growing 30 to 75 cm (14 to 30 in.) in height. The most popular lawn grass in the Unites States, it is also grown extensively as a pasture grass in the northeast and north central states.

Smooth Brome, *Bromus inermis.* Smooth brome was introduced from Europe. It is a cool season, sod-forming perennial that is adapted to temperate climates from the middle of the United States northward to Canada and Alaska. With irrigation, it is planted throughout the United States. Smooth brome is frequently planted with a legume.

Tall Fescue, *Festuca arundinacea.* Tall fescue has become the most widely grown pasture grass in the United States (approximately 14.16 million hectares [35 million acres]). Tall fescue grows to a height of 60 to 200 cm (24 to 79 in.) and is a deep-rooted, perennial grass that forms a dense sod through spreading short, underground rhizomes. Tall fescue is capable of withstanding environmental extremes, including drought; flooding; extremes of heat and cold; soils varying in texture, moisture, salinity, and pH; and heavy traffic. Cool, moist conditions are optimal for growth, so surges occur in the spring and fall.

Oats, *Avena sativa.* Oats is an annual that is harvested as grain or as hay in the Pacific Coast states but is rarely used as a pasture grass. The stage of maturity when cut has a profound influence on the nutritive value of the hay. The best quality hay is obtained if cut when the flowers are in the boot, but the greatest yield of TDN results from cutting when seed heads are in the soft-dough stage. If allowed to go past maturity, the food value of oats rapidly declines to almost nothing as the oats become straw.

Orchardgrass, *Dactylis glomeratus.* Orchardgrass is a perennial, cool season grass introduced into the United States from Europe more than 200 years ago. Cultivars are grown in all states except Hawaii. It may grow to heights of 60 to 200 cm (2 to 6.5 ft). Orchardgrass has become one of the most popular pasture grasses for llama and alpaca managers because of its adaptability to many areas of the United States and its palatability.

Ryegrasses. Various species of ryegrass have become naturalized in most temperate and subtropical climates of the world. Perennial ryegrass (*Lolium perenne*) is grown as a pasture plant and harvested as hay from millions of hectares; it is the second most popular pasture grass in the United States. Perennial ryegrass

is a short-lived perennial that grows to a height of 25 to 50 cm (10 to 20 in.). Annual ryegrass (*L. multiflorum*) may grow to a height of 1 m (3 ft). Both species are native to Europe.

Sudangrass, *Sorghum sudanense*. Sudangrass and other sorghums were introduced from Africa in the early part of this century. The sorghums are extremely important as forage for animals (pasture, green chop, silage, hay, grain) and as a seed crop for human consumption. Sudangrass is a highly productive annual.

Timothy, *Phleum pratense*. Timothy is a perennial bunchgrass native to Eurasia but now distributed throughout temperate and subarctic climates of the world. It is a cool-season grass and doesn't grow well under drought conditions. It is grown primarily as a hay crop in the northeast, north central, and northwestern United States. Timothy is the hay of choice of horse owners throughout the country. It grows to a height of 80 to 110 cm (30 to 43 in.). When planted for pasture, it is usually mixed with other grasses and legumes.

Crested Wheatgrass, *Agropyron desertorum*. Crested wheatgrass is a native of Eurasia. Crested wheatgrass is a winter-hardy, drought-resistant perennial bunchgrass, primarily used as pasture forage. It has become a popular grass for seeding rangelands in the northern great plains and the dry northern intermountain area.

Legumes[3,39,43]

Alfalfa, *Medicago sativa* (Lucerne). Alfalfa is a herbaceous perennial legume. It has become the most important forage crop in the United States ("queen of the forages") and is grown in every state. Alfalfa probably originated near Iran, but similar species are found throughout central Asia and Siberia. It has been introduced all over the world by invading armies, explorers, and missionaries. Alfalfa was described as an excellent forage for horses and other animals by Pliny the Elder as early as 490 B.C. More than 12 million hectares (49 million acres) are planted to alfalfa in the United States. Alfalfa is also the most important source of nectar for bees in the United States.

A mature alfalfa plant may have from five to twenty-five stems growing to a height of 60 to 90 cm (15 to 36 in.). Alfalfa is most commonly harvested for hay because ruminants that graze alfalfa pasture tend to bloat. Camelids are resistant to bloat.

Like other legumes, alfalfa is able to convert atmospheric N to nitrate by a symbiotic relationship with Rhizobium meliloti, an efficient N-fixing bacterium. Colonies of Rhizobium are found in nodules on the roots (Figures 2.13, 2.14). Seed must be inoculated with the bacteria before sowing to initiate the process.

Figure 2.13. Alfalfa root nodules containing colonies of nitrogen-fixing bacteria (*Rhizobium* spp.).

Figure 2.14. Alfalfa root nodule containing colony of nitrogen-fixing bacteria (*Rhizobium* spp.).

Alfalfa is harvested one to ten times a season, depending on the climate and availability of irrigation. First-cut hay is leafier and has fewer stems than later cuttings. The first cutting is the premier dairy cattle feed, but is not the first choice for feeding camelids.

A close relative of alfalfa, burclover (*M. polymorpha*) has a procumbent growth, so it is rarely harvested for hay. It is, however, an important component of open ranges in California and other regions of the west. It is of value only during the wet season and before the curled pods (with spines) mature and become entangled in fiber.

Alfalfa has received unwarranted negative publicity as a feed for camelids. The following statements have been made to discredit alfalfa hay for SACs.

Statement: Alfalfa has an excess of Ca that is detrimental to mineral nutrition by being deleterious to absorption of P, Zn, and Mg.

Facts: The result of chemical analysis of Ca and P in alfalfa hay, full bloom, is Ca 1.25% and P 0.22%. This produces a Ca-P ratio of 5.7:1, which seems to be out of balance with the recommended ratio of 1.5:1 or 2:1. However, other factors must be considered. Chemical analysis reports the total quantity of Ca in the feed stuff but does not provide information about the availability of the Ca for absorption. A significant level of oxalate in alfalfa forms insoluble Ca oxalate crystals.[41] Digestive studies conducted in horses, cattle, and sheep indicate that 50% to 70% of the Ca in alfalfa hay is tied up in insoluble Ca oxalate. Thus, the available Ca-P ratio in mature alfalfa hay is more like 1.7:1, which is an acceptable ratio. Notice in Table 2.8 that the Ca-P ratio of alfalfa leaves is 9.4:1, a less than desirable ratio that supports the concept that llamas should be fed less leafy hay and not be offered more hay until all the stems are eaten.

The interrelationship of Ca, P, and vitamin D is complex. The dietary ratios of these nutrients are important, but scientific studies of these nutrients and their actions in camelids have not been reported. Dogmatism at this time is unwise.

Statement: It is recommended that alfalfa hay be restricted in certain near-term dairy cows because of its high Ca content.

Facts: Veterinarians know that high-producing dairy cows are prone to hypocalcemia. The surge of milk production at the beginning of lactation places a heavy drain on body Ca, which is not quickly replaced by the cow's metabolism. It has been found that one of the predisposing factors to postparturient hypocalcemia is that the parathyroid glands are not primed to mobilize Ca from the bones rapidly enough to supply the needs of heavy lactation. If the cow has been on an adequate to high Ca intake before parturition, there has been little need for parathormone production by the parathyroid gland. By cutting back on Ca intake for a few days prior to parturition, the parathyroid is stimulated to return to the production of parathormone; thus, mobilization of Ca after parturition is more easily accomplished.

This phenomenon is seen only in high-producing dairy cows. Llamas and alpacas are not bred for milk production, and postparturient hypocalcemia has not been reported in any of the camelids. This argument is false.

Statement: Alfalfa is too high in total digestible nutrients, thus fostering obesity in llamas.

Facts: There is a danger in overfeeding alfalfa hay, not because of excess protein or improper Ca-P ratios, but because of too much energy intake. Llamas become obese if fed too much, whether alfalfa hay or any other suitable hay. It is not an inherent fault of the alfalfa but rather lack of appreciation of how much to feed.

Statement: Alfalfa contains a higher level of protein than is necessary or desirable for llamas/alpacas.

Facts: Alfalfa hay does contain higher levels of protein than is required by llamas/alpacas. The question is whether or not there is a detrimental effect on the animals. Excess protein is deaminized and the nitrogen is excreted via the urine while the other moieties are converted to energy. In a feeding trial using different levels of protein in the diet, the level of serum urea nitrogen (SUN) in llamas consuming a diet containing 10% protein was 19.3 mg/dl, and on a diet containing 16% protein the SUN was 29.6 mg/dl.[60,64] No evidence was presented to indicate that the higher level of SUN was detrimental. "Protein poisoning" was once thought to be a factor in swine production, but that was debunked years ago. The only valid argument against alfalfa hay is that protein is the expensive nutrient in most forages.

In the final analysis, considering the present lack of basic nutrition studies on mineral metabolism, there is little evidence to support a dogmatic statement that alfalfa is not a good feed for llamas/alpacas. Alfalfa has been an outstanding forage in numerous species of ungulates for centuries, yet it is not a perfect forage, because no single forage is perfectly balanced.

Red Clover, *Trifolium pratense*. Red clover is the most widely grown true clover, planted extensively in the Pacific Northwest, California, Idaho, Nevada, Utah, and the entire eastern half of the United States east of the Mississippi River. Red clover is a perennial legume and shares many characteristics with alfalfa. Cultivars are adapted to either single or multiple cuttings per year. Red clover is a common component of mixed pastures.

White Clover, *Trifolium repens* (Ladino Clover). White clover is closely related to red clover and has a slightly broader distribution than red clover in the western and northwestern United States. White clover is a common component of irrigated mixed pastures. Alsike clover, *T. hybridum*, is an intermediate between red and white clover.

Table 2.19. Comparison of the nutrient compositions of grasses cut at the same growth stage.

Grass	*Protein*	*Crude fiber*	*TDN*[a]
Orchardgrass	9.3	31.4	53
Bromegrass	10.9	29.4	53
Timothy	7.9	30.3	63
Reed canarygrass	13.2	26.4	65
Kentucky bluegrass	12.3	27.6	56
Tall fescue	14.5	24.6	67
Perennial ryegrass	10.4	23.2	68

Note: Growth stage of grasses determined by percent of dry matter.
[a]TDN = total digestible nutrients.

Miscellaneous Legumes. Numerous species of legumes are adapted to specific regions of the United States. There are more than 250 species of the genus Trifolium worldwide, including strawberry clover (*T. fragiferum*), crimson clover (*T. incarnatum*), and subterranean clover (*T. subterraneum*). Additional legumes used for forage in the United States include birdsfoot trefoil (*Lotus corniculatus*), lespedezas (*Lespedeza cuneata or L. striata*), and hairy vetch (*Vicia villosa*). A comparison of the composition of selected legumes cut at 1/10 bloom stage is found in Table 2.19.

Forage Evaluation[53]

Forages may be grown on the farm or ranch, but most often they are purchased. The purchaser must be able to judge the quality of the forage or rely on the integrity of the supplier. Usually the owner/manager evaluates the forage, but veterinarians should be able to determine the quality of forages and recommend a change or supplementation as appropriate.

The quality of hay is determined by the stage of maturity when it is cut; the method of curing and conditions of storage; color; odor (sweet, musty); and the presence of foreign material such as weeds (particularly poisonous plants), dirt, sticks, unpalatable grasses, grass awns, or wire and nails.

Grass Hay Characteristics

Premium quality grass hay is cut in the boot stage (seed heads emerging from the leaf roll) or early head stage. The color should be bright green. This is not the stage that produces the greatest tonnage per hectare, so often the grass is allowed to grow longer. At midgrowth, the grass should still be greenish and there should be no mature heads. This stage produces the most tonnage. Mature grass contains ripe seeds and large stems with yellowish brown leaves.

Legume Hay Characteristics

Leaf-stem ratio is important. Leaves predominate in early growth; as the legumes mature, stems grow longer and coarser (stemmy), and the lower (older) leaves fall off. Faster growing plants tend to be more stemmy, which occurs during midsummer (when it is warmest). Leaf loss during harvesting increases as maturity is reached.

Legumes should have a bright green color if cut early and cured properly. Sun bleaching produces light golden yellow that is usually streaked because the bleaching occurs on the surface of the windrows. If hay is too moist when it is baled, fermentation begins and turns the hay brown, and often it has a musty or tobacco odor. If legumes are soaked by rain, the hay turns dark brown or even black, the stem harsh and brittle.

Alfalfa. When cut in the prebud stage, there is a high percentage of leaves, with fine pliable stems. Protein content is highest at this stage, but tonnage per hectare is low. Premium quality alfalfa is cut at 1/10 bloom (a few purple flowers) with more robust stems. Protein levels are adequate. At full bloom, alfalfa has large stems, a number of purple flowers, and some mature seed pods. Protein levels may drop to half that of the prebud stage.

Clover. Premium quality clover hay is cut at 1/4 to 1/2 bloom. The leaf-to-stem ratio is high. At full bloom a large number of flowers are present, and the leaf-to-stem ratio is low. At full maturity, brown flowering heads are present along with mature seed pods; leaf-to-stem ratio is poor, and stems are large.

Diet Evaluation

Adequate evaluation of the diet of a llama or alpaca requires analysis of the forage. A number of hay samplers are available commercially. An excellent sampler is called the Penn State Forage Sampler, which is a 45-cm (18-in.) stainless steel tube, 2.86 cm (11/8 in.) in diameter with adapters to fit either a hand brace and bit or an electric drill. The sampler is bored to a depth of 20 cm (8 in.) into the ends of twenty to thirty bales (Figure 2.15). All of the cores should be placed into a single plastic bag to make a composite sample, which is then submitted to the laboratory. States with Cooperative Agricultural Extension offices are likely to have a list of laboratories that perform analysis of forages and soils. Samples may also be collected from pastures by grabbing handfuls of the predominant plants and processing as above for analysis.

The evaluation of concentrate feeds (grains, meals) and processed feeds may be difficult. Poor-quality forage may be compressed into cubes or pellets, making it impossible to evaluate the quality of the ingredients. Grains should be free of molds and have a sweet smell.

Total diet evaluation requires a list of the components of the diet, how much of each is fed and how

Figure 2.15. Obtaining a core sample of hay for analysis of nutrient composition.

much is eaten (fed – eaten = wastage), and the weight of the animals fed. Nutritionists and some veterinarians have computer software programs that contain the proximate analysis of individual feeds, and calculations may be made about the percentage of various nutrients. This is then compared with the requirements of the animals, to the best of current knowledge. Recommendations may then be made for appropriate supplementation or change in the diet.

North American Feeding Practices[6,7,16,32,59,73,111,112,126,129,130]

Pasture[45,78,81,108,109]

There are nearly as many different pasture feeding practices as there are llama owners. Llamas and alpacas have been grazed on every conceivable type of pasture, from native pastures in Texas or Arizona to improved pastures in Michigan or British Columbia. The tables of composition of feed items indicate relative values of some forages (Tables 2.16, 2.17). Information on feeds not mentioned may be found in the references appended.[3,4,36,43,76] The nutrient content of feeds may vary from region to region, so these figures cannot be considered as absolutes.

Adult llamas may be maintained on most pastures if sufficient forage is available for ingestion within a reasonable amount of time. Some pastures are not of sufficient quality to sustain pregnant and lactating females without weight loss or to supply growing animals with adequate nutrition. Forage quality should be compared with the requirements of different classes of llamas.

As an example, if a llama weighing 100 kg was kept on a ryegrass pasture, the animal would have to consume 5.54 kg/day (ryegrass contains 0.80 Mcal DE/kg, as fed, and the animal requires 4.43 Mcal/day). This amounts to 5.54% of the body weight. To consume sufficient forage of this type, the animal must graze for much of the day. If this llama was a pregnant female or nursing a baby, the pasture would not provide sufficient nutrients, and supplementation would be necessary to maintain her weight.

It should be remembered that pasturage is all that is available to camelids in their native lands. Animals living in particularly harsh areas suffer accordingly. There is considerable seasonal variation in nutrient intake, hence periodic weight gain and loss.

Although pasture management is not the usual expertise of a veterinarian, a knowledge of basic principles may be helpful in working with an owner to establish appropriate feeding practices. The basic principles are

1. The appropriate grass or pasture mix should be selected for the area.
2. If possible and economically feasible, irrigation should be used to prolong the productive season.
3. Natural or artificial fertilization is important.
4. The pasture should be groomed. If grass is allowed to go to seed, the stand may go into seasonal dormancy. Some native bunchgrasses cure standing and may provide adequate forage, but most grasses dry up and lose most

of the nutrient value. Periodic mowing or clipping may be practiced.

5. Owners with large pastures should consider sectionalizing to allow rotational grazing. This may result in more efficient use of resources and may allow for part of the pasture to be used for hay.
6. If there is uneven grazing of grass in a multispecies pasture, adding additional animals such as sheep, cattle, or horses to the pasture may be helpful. There may be two benefits. First, other animals may graze different grasses, and second, each animal will avoid grazing near defecation and urination sites of its own species but will graze the grass near other species' feces.
7. There are as many different fences in use as there are salespersons to tout them. Most llama/alpaca owners tend to over fence. Electric fences are useful for sectionalizing a pasture.
8. Pastures should not be overgrazed. A stand of perennial grass may be destroyed if grazed too closely over a period of time.
9. Palatability must be factored into any grass or pasture mix. Check with local breeders to see what their animals prefer.
10. Weeds in the pasture should be controlled. Weeds compete with forages for soil nutrients. Weed seeds may be carried via irrigation systems. Weeds often take over in abused pastures because they tend to be more hardy than introduced grasses.

Hay

A large percentage of North American camelids are fed hay for at least part of the year. Numerous legumes and grasses may be cured and stored in a variety of ways to preserve the nutrients for feeding at a later time. Climate greatly affects the quality of hay. Hay subjected to rain during the curing process loses much of its nutrients.

Tables 2.16 and 2.17 provide the composition of some the plants and combinations of plants fed as hay in North America. Most of these have been used at one time or another for llamas and alpacas. Probably any of them are satisfactory if appropriate supplementation is provided according to the needs of the animals. A comparison of the composition of selected grasses, cut at the same growth stage, is found in Table 2.19, and selected legumes in Table 2.18. Llamas may be considered to be picky eaters. Those who feed alfalfa will have observed that llamas prefer leaves and fine stems and avoid the large stems. This results in significant wastage if unlimited free-choice feeding is practiced.

Some owners subscribe to the theory that if a "cafeteria" of various feed items is provided, a llama will select a diet that meets individual requirements. This is neither economically, behaviorally, nor nutritionally justified. As in all species, llamas eat not to satisfy basic nutrient requirements but to satisfy appetite (i.e., taste and smell). A wise manager will learn the animal's requirements and develop a feeding regimen that coincides with those requirements.

As an example of how to use the tables to determine the quality of a hay ration, consider a 100-kg llama with a DE requirement of 4.43 Mcal/day. Alfalfa hay has a caloric density of 2.29 Mcal DE/kg. Therefore, a llama consuming 1.94 kg of alfalfa hay would satisfy the energy requirement. Other nutrient requirements would also be met. Because this amount is only 1.94% of the body weight, it would easily be consumed by this animal. If the same llama was kept on a ryegrass pasture, the animal would have to consume 5.54 kg/day (ryegrass contains 0.8 Mcal DE/kg, as fed). This amount is 5.54% of its body weight. To achieve this the animal must graze for much of the day. If this llama were a pregnant or lactating female, the pasture would not provide sufficient nutrients, and supplementation would be necessary.

Fertilization

Optimum fertilization of forages used for pasture or grown for hay is a discipline of its own and warrants careful consideration by owners/managers who are fortunate enough to have land to grow forage. Veterinarians may be asked, "Can I use llama or alpaca manure to fertilize my pastures?" Camelid manure is usable as a fertilizer.[43] There may be concern that parasite ova could be spread with the manure, but the benefits outweigh the risks. Pellets should be ground before spreading. Sunlight and drying destroy most parasite ova. The actual nutrient composition of llama and alpaca manure is far below that of commercial fertilizers (Table 2.20), but the addition of organic matter has merit. Just as forage analysis is appropriate, soil analysis may be necessary to determine the most suitable fertilization regimen to use on a given area. Plants can't manufacture trace elements; they must be absorbed from the soil (Figure 2.16).

Processed Feeds[52]

Current agricultural practices supply livestock growers with hays and concentrates in various forms, such as bales, cubes, and pellets. Forages may also be harvested green and preserved as silage or haylage, which have been used to feed llamas but probably do not constitute an important source of feed for them.

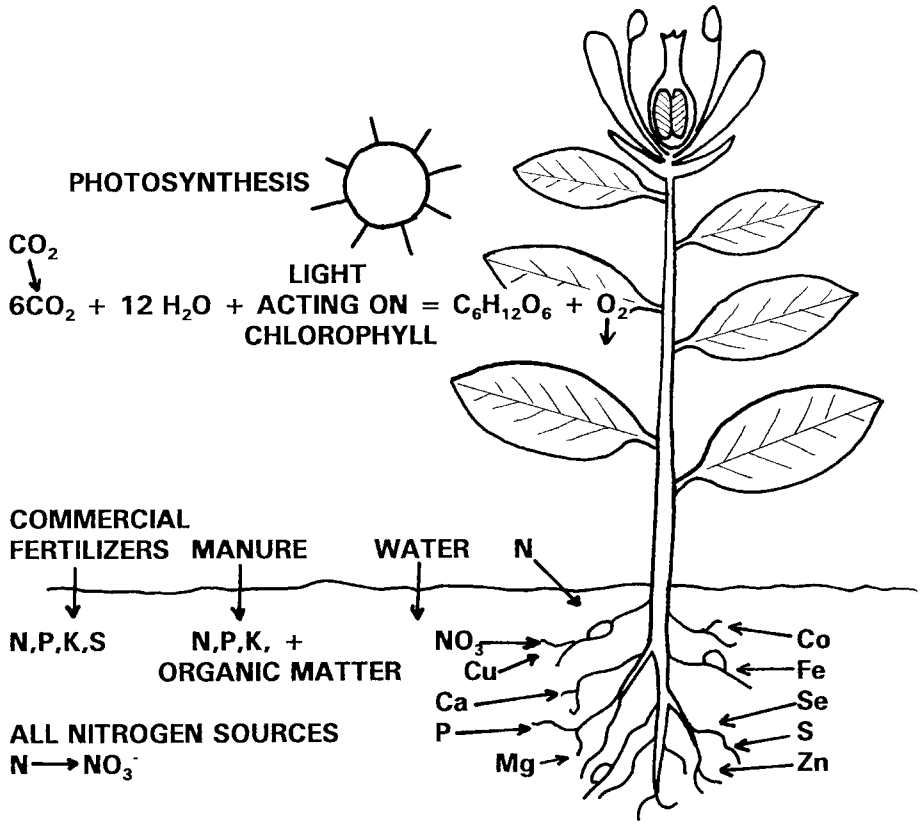

Figure 2.16. Source of nutrients for a plant.

Table 2.20. Commercial fertilizers and manure of various species.

Fertilizer	*Nitrogen (%)*	*Phosphorus (%)*	*Potassium (%)*
Commercial 16-16-16	16	16	16
Commercial 16-20-0	16	20	0
Manure			
Dairy cow	2.38	0.48	2.2
Sheep	3.05	0.83	3.1
Poultry	4.71	1.58	1.4
Llama I[a]	14.2	1.2	0.4
Llama II[b]	2.7	1.3	1.2
Alpaca[c]	1.49	0.23	1.06

[a] Johnson 1992—Feces taken directly from rectum.
[b] Fowler—Unpublished data.
[c] Hart, 1991.

Pelleted feeds, both simple hay and complete diets, have been fed to camelids in private ownership and in zoos for a long time. These are somewhat expensive products, but wastage can be kept to a minimum; thus in the long run, these may be less expensive than other types of feed. Camelids generally consume pellets satisfactorily, but choke has occurred. The larger the pellet, the better. It has been suggested that more fiber may be needed in the diet than is provided by pellets. In one study, an increase in soil ingestion by llamas was observed when pellets were fed, even though the pellets were not fed on the ground.

Concentrates

Some SACs avoid ingestion of grains or mixed sweet feeds, while others willingly eat them. Individual animals may prefer one type of grain over another, but this is probably a learned response, for most of the grains are acceptable. All grains supply increased energy and, in some cases, protein. They are deficient in Ca, P, and trace minerals. Concentrates are usually more expensive than hays and should be used only when needed to supply additional energy. Mixtures of grains, protein supplements, vitamins, and minerals are commonly bound together by blackstrap molasses to form a sweet, palatable, high-energy protein supplement. These are satisfactory for camelids if the extra nutrients are needed. Camelids are able to metabolize urea as an N source for synthesis of protein, but suitable levels for inclusion of urea in a ration have not been determined.

NUTRITIONAL PROBLEMS[125]

Starvation/Inanition

There are enough new, inexperienced camelid owners that veterinarians may see a starved animal (Figures 2.17, 2.18). A thick fiber coat masks a loss of condition, and unless the owner can and does routinely feel the backbone, there will be little to indicate weight loss. Many large operations regularly monitor the weights of animals. Owners should be encouraged to weigh their animals regularly, either on a purchased farm scale (Figure 2.19) or at a public weigh station. In the absence of a scale a second choice is to perform a "body score." This is a process, devised by Dr. LaRue Johnson, of observing and palpating selected regions of the body (Figure 2.20) to assign an overall score from 1 to 10, with 1 being emaciated, 5 being desirable, and 10 being severely obese.[60,61]

Having found a thin animal, it is necessary to determine if weight loss is caused by a nutritional problem or an infectious or parasitic disease. An evaluation of feeding practices may help in making a diagnosis. Appropriate remedial measures should be instituted. At necropsy, a starved animal will lack body fat, and

Figure 2.17. A thin llama, whose condition is obscured by the fiber coat.

Figure 2.18. The vertebral column of the previous thin llama showing atrophy of the longisimus dorsi muscles.

Figure 2.19. Scale.

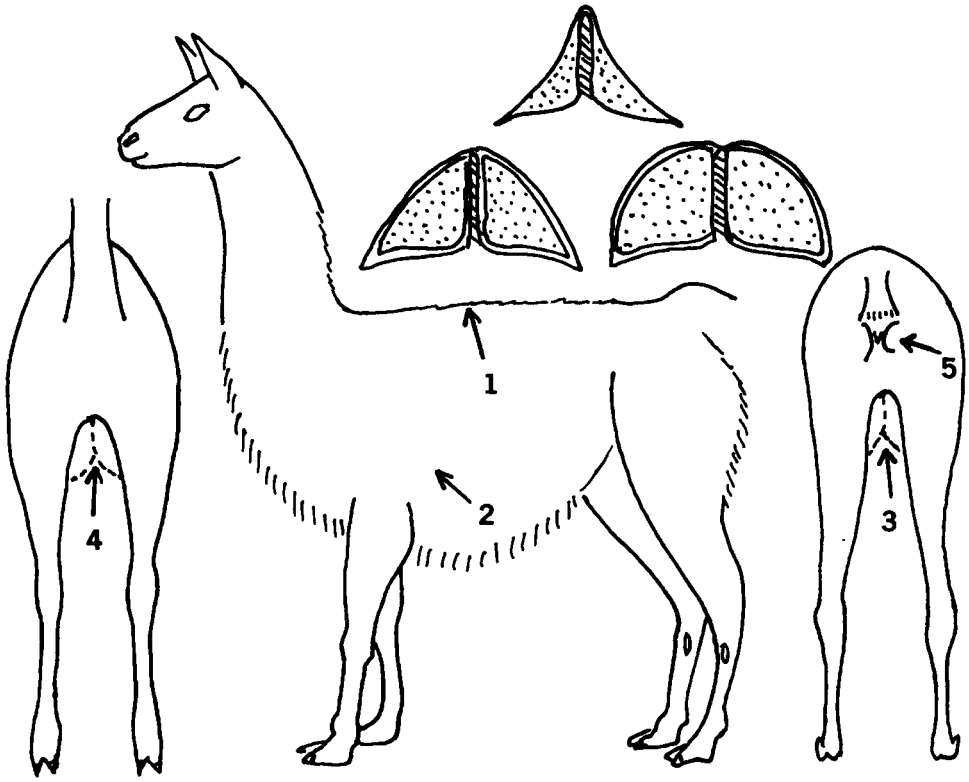

Figure 2.20. Key locations for evaluating body condition, in order of examination: (1) withers, (2) fiberless area behind the elbow, (3) between the rear legs, (4) chest (between the front legs), and (5) perineum. Score from 1 to 10, with 1 = thin, 5 = ideal, 10 = obese.

serous atrophy of fat around the heart and in the peritoneal cavity may be seen. Bone marrow will be watery rather than gelatinous. Fatty infiltration of the liver is often observed.

Failure to Thrive/Wasting Syndrome[107]

A number of llamas have been observed to become anorectic, lose weight, and ultimately die (Chapter 9). Growth of crias is not uniform throughout the growing period, but weight gain should be regularly evaluated.[57] Juvenile SACs that are persistently infected with bovine virus diarrhea virus (BVDV) usually fail to thrive and usually die by one year of age.

Obesity

Probably a more frequent problem than underfeeding, especially among new, overly solicitous owners, is overfeeding. This easily occurs if concentrates are used routinely without regard to actual requirements. Overfeeding is not only costly, but obese animals are more likely to be infertile and develop hyperthermia more easily than those of normal weight (Figures 2.21 to 2.23).

There is no question that some animals become obese more readily than others. The recommended requirements should be adjusted to fit individual needs. If an animal gains undesired weight when

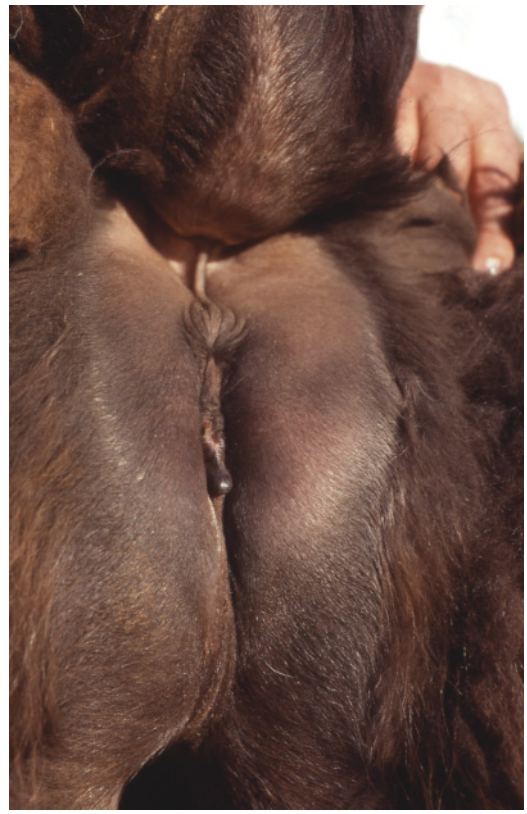

Figure 2.21. Obese female llama.

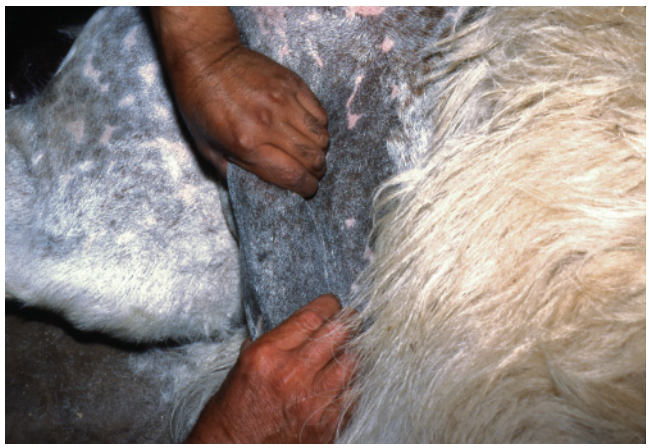

Figure 2.22. Roll of fat on the flank/abdomen of an obese llama.

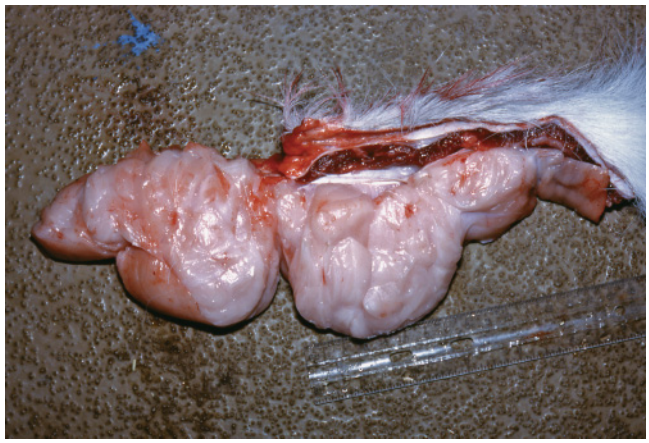

Figure 2.23. Accumulation of retroperitoneal fat from an obese llama.

fed recommended amounts, the amount of feed given should be decreased until a correct balance is achieved.

Metabolic Bone Disease[29,47,68,69,75,84,128]

Metabolic bone disease (MBD) is a collective term used to describe various manifestations of the complex syndrome produced by inadequate Ca, inadequate P, improper Ca–P, and/or nonexposure to ultraviolet light or lack of vitamin D in the diet.[31] Other factors that may have an impact on the syndrome include protein deficiency and primary diseases of the kidney, liver, and intestine, which affect the conversion of vitamin D to the active hormone and absorption of minerals from the intestine.

The clinical signs of MBD exhibited by an individual animal depend on the age of the animal, which component is deficient, and duration and degree of the deficiency. Signs include lameness, fractures, deformity of bones, painful joints, and reluctance to move. Anorexia results from painful mastication, caused by loosening of teeth. Growth of young animals is slowed. In lactating females, milk production decreases. Severe Ca deficiency may result in tetany. Some species develop fibrous osteodystrophy, but it is not known if this occurs in camelids. Any, some, or all of the above signs may be seen in an affected animal.

Some llamas develop angular limb deformities, the etiology of which has not been determined. Considering that this condition may have a nutritional basis, some owners suspect alfalfa and its high Ca content (1% to 2.5% on a dry matter basis) (see discussion of alfalfa in this chapter).

Poisons in Feed

Several poisonous plants have been identified as toxic to camelids (Chapter 23). In connection with feeding, it is important to recognize that processed feeds present a special hazard for all animals. Given a choice, a camelid may not select and eat a toxic plant. However, when included in a cube or a pellet, poisonous plants may be undetectable. Unfortunately, cubes and pellets may be made to use poor quality forage or hay containing poisonous plants. The reputation of the supplier is the only safeguard available.

Other Diseases

Infectious and parasitic diseases place a stress burden on the animal. This may be reflected in increased nutrient requirements, paradoxically, at a time when an animal may be disinclined to eat. Protein and energy requirements may be significantly increased in the febrile state or when an animal is suffering from nutrient-losing diarrhea or pneumonia. Postsurgical wound healing and fracture repair may require 150% of the usual maintenance needs.

Many disease conditions diminish or destroy stomach microorganisms. Without fermentation, digestion and absorption of nutrients is impaired. Hydration and reestablishment of essential flora and fauna may be crucial to recovery.

The type of diet fed may change some serum and urine values.[64] Animals on a high-protein diet (16% CP) have higher SUN values.

SPECIAL FEEDING SITUATIONS

Feeding on the Trail

Llamas are popular pack animals; therefore, feeding on the trail is necessary. Feeding is limited to grazing and feed that can be carried in addition to needed items for humans. Llamas make good use of available plants at rest stops and in camp. They eat many types of forbs, grasses, sedges, shrubs, and small trees

without stripping an area clean of all vegetation. Llamas may be hobbled and allowed to run free or tethered to picket lines or, individually, to trees, shrubs, rocks, or stakes.

Poisonous plants may be a hazard. Although llamas in South America may have learned to avoid native poisonous plants, this may not be true of animals in North America. The packer must avoid tethering an animal where there is little to eat but a poisonous plant. A near tragedy occurred when llamas were tied to the mountain laurel shrub, *Leucothoe davisaii*.

Hard-working pack llamas may have energy requirements that are double the amount needed for maintenance. Time must be allowed for them to graze and/or consume carried feed.

One successful, experienced packer feeds a mixture of alfalfa pellets and a mixed sweet feed at a rate of approximately 0.45 kg (1 lb) per animal, night and morning, in addition to permitting as much grazing as possible. Other packers feed alfalfa pellets and COB (a mixture of cracked corn, oats, and barley).

Cold Weather

Camelids are superbly adapted to cool weather but, surprisingly, not to the extremely cold weather of high elevations in North America. Temperatures rarely dip below –10° to –20°C, even at high elevations in the Andes where camelids live. In North America, temperatures may drop to 40°C (40°F) and be accompanied by a wind chill factor that drops the effective temperature even lower.

A paradox occurs in feeding herbivores. A high-fiber diet yields more heat for warming the body. A high-concentrate diet provides more energy, but it is not as efficient in warming the animal. However, a growing animal may not be able to obtain sufficient energy on a high-fiber diet to meet its requirements for growth.

Total Parenteral Nutrition

Total parenteral nutrition (TPN) is feasible and useful in selected situations.[54] The protocol is as in horses and cattle.

Table 2.21. Glossary of terms used in feeding and nutrition.

Joule	The work done when the point of application of a force of one newton is displaced through a distance of one meter in the direction of the force.
One joule	0.239 calorie
Kilojoules	1,000 joules
Megajoules	One million joules
Energy density	The amount of energy in a feed (Kcal/kg or Mj/kg).
Calorie	The amount of heat required to raise the temperature of 1 gram of water from 14.5°C to 15.5°C at standard pressure. The standard calorie used for expressing the chemical energy in feeds.
One small calorie	4.18 joules
Kcal	1,000 small calories
Mcal	One million small calories
Dry matter	The nutrient composition of a feed minus the moisture.
As fed	The nutrient composition of a feed as consumed as hay, pasture, or a concentrate. Includes moisture.
Metabolic weight	Body weight in kilograms raised to the 0.75 power ($W_{kg}^{0.75}$).
Basal metabolic rate	The energy used by an animal to maintain body temperature in a thermoneutral environment ($Kcal/d = C \times W_{kg}^{0,75}$), C = A constant for a group of animals.
Gross energy	The amount of heat that is released when a substance is completely oxidized in a bomb calorimeter containing 25 to 30 atmospheres of oxygen.
Digestible energy	Gross energy minus fecal energy.
Metabolizable energy	Food intake gross energy, minus fecal energy, minus energy in the gaseous products of digestion, minus urinary energy.
Net energy	Metabolizable energy minus losses from digestive fermentation and nutrient metabolism.
Parts per million (ppm)	mg/kg
Concentrate	A feed used with another feed to improve the nutritive balance of the total and intended to be diluted and mixed to produce a supplement or a complete feed.
Forage	Aerial plant material, primarily grasses and legumes containing more the 18% crude fiber on a dry basis, used as animal feed.
Supplement	A feed used with another feed to improve nutritive balance or performance.
Photosynthesis	The formation of carbohydrates from the action of sunlight on plant chlorophyll, plus water from the soil and carbon dioxide from the atmosphere ($6CO_2 + 12\ H_2O$ + Action of sunlight on chlorophyll = $C_6H_{12}O_6$ (carbohydrate) + O_2).
Halophyte	A plant that tolerates high salinity in the soil, in irrigation water or salty spray, such as near an ocean.

A glossary of terms used in feeding and nutrition is found in Table 2.21.

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[a] Dr. James Vickers, Michigan State University, personal communication.

[b] Sources of ZinPro 100: ZinPro Corporation, Minneapolis, MN, (612) 944 2736; Janos Chemical Co., Forbes, N.S.W. Australia (68) 521044. Source of powdered molasses: Feed Specialties, 1877 NE 58th Ave., Des Moines, IA, 50313, (515) 262–3261.

[c] Dr. William Barnett, Washington, D.C., personal communication, Sept. 1996.

3

Restraint and Handling

The procedures described herein are those that allow access to the animal to carry out diagnostic and therapeutic procedures. The author is well aware of the outstanding techniques that are now being employed in the training of SAC that may make it unnecessary to use some of the methods described.[2,3,22,23] It would be desirable that all SAC be trained to allow veterinarians to use minimum restraint when caring for an animal, however this is not yet possible. It takes time and skill to train a camelid or any other animal. Not all owners/keepers are willing or able to train each and every animal on the farm or in a zoo, nor is it the responsibility of the veterinarian to do the training. Nonetheless, a veterinarian should assess from the owner/keeper the degree of training given an individual to be handled and act accordingly. Having said that, the veterinarian must be able to handle animals considering safety to themselves, any assistants and the animal.

Llamas, alpacas, and camels have been domesticated for thousands of years. When accustomed to being handled, they are docile and pleasant. Only the rare individual is aggressive or a "spitter." Guanacos and vicuñas are wild animals and may not be as easily handled (i.e., chemical immobilization may be necessary). However, guanacos can be tamed and handled similarly to llamas if procedures are carried out slowly and quietly.

CAMELID BEHAVIOR[4,15,17,19,20]

Prior to beginning a discussion of restraint it is important to have a basic understanding of camelid behavior. Behavior, as used here, refers to an animal's total activity, especially that which can be externally observed. It is not the intent of the author to discuss all aspects of behavior, rather to emphasize those actions that have a bearing on the successful handling of camelids. Being able to evaluate an animal's behavior also provides important clues about its medical well-being, and allows recognition of the early signs of disease. More information may be found in Chapter 4 and in the references provided.[2,3,22,23]

Camelids communicate with one another and humans through body language and vocalization. Although all camelids share some basic behavioral patterns, SACs and OWCs are sufficiently different that they will be discussed separately.

New World Camelid Behavior

Ear and tail position are sure indicators of the mental state of an SAC. Owners/handlers should understand these signals to minimize stress for both animals and humans. The ears of a contented, unaroused SAC are in a vertical position and turned forward (Figure 3.1B). In the alert animal, the ears are cocked forward (Figures 3.2, 3.3). Relaxed SACs may allow the ears to lie horizontal to the rear. This is a normal position and should not be considered aggressive, unless other signs of aggression are present. In some individuals the ears may appear to spread laterally from the top of the head (Figure 3.4).

Various degrees of aggression are communicated between herd mates by ear, head, and tail position, usually displayed in concert. Ear and tail positions may be in a continual sate of flux, especially when feeding stations lack adequate space for all herd members. Mild to moderate aggression is signaled by the head held horizontal with (1) the ears positioned above the horizontal (Figure 3.1C), (2) the ears horizontal (in the same plane as the head, Figure 3.1D) and (3) the ears below the horizontal (Figure 3.5), and (4) the ears flattened against the neck (Figure 3.6). Intense aggression is exhibited by the nose being

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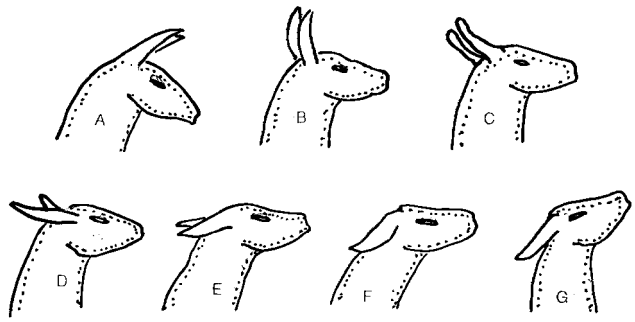

Figure 3.1. Ear and head positions of llamas. (A) Alert, (B) normal ear position, (C) resting position, (D) resting position and listening toward rear, (E to G) various degrees of aggression.

Figure 3.4. Llama with ears spread laterally, resting.

Figure 3.2. Llama in an alert position, ears forward and tail slightly elevated.

Figure 3.5. Alpaca with head and ears horizontal, resting or listening rearward.

Figure 3.3. Alpaca with ears vertical.

Figure 3.6. The llama in the foreground is exhibiting mild to moderate threat, with head horizontal, ears back against the neck.

Figure 3.7. An aggressive llama, exhibiting threat behavior, with nose in the air and ears against the neck.

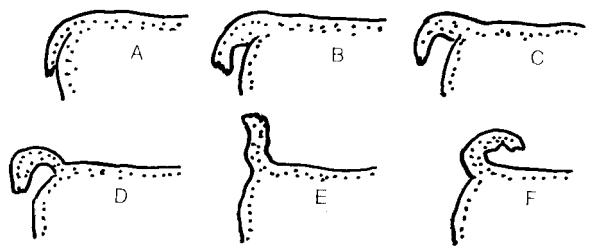

Figure 3.8. Diagram of tail positions of a llama. (A) Normal, non-aroused, (B and C) alert, (D and E) degree of threat or aggression, (E) submissive.

pointed in the air and the ears flattened against the neck (Figure 3.7).

Tail positions also communicate social information (Figure 3.8).[2–5] In the non-aroused SAC, the tail lies flat against the perineum (Figure 3.8A). Mild aggression or alertness is indicated by the tail being slightly elevated, but below horizontal (Figures 3.8B and C). As the degree of agitation escalates, the tail may be carried horizontal, curled above horizontal or vertical (Figures 3.8D,E). Basically, the higher the tail, the higher the level of aggression. The tail may also be seen to wave from side to side, especially in males that are slightly agitated. These aggressive behaviors are employed by social animals to minimize outright fighting.

Submissiveness in the llama, guanaco, and alpaca is indicated by curving the tail forward over the back, with the head and neck held low, the ears in a normal to above horizontal position, and the front limbs slightly bent (Figure 3.9). This behavior is frequently seen in SACs that have been given too much human attention early in life. The submissive crouch of a vicuña is with the tail curved forward but with the head curved back over the body.

Figure 3.9. Submissive llama posture.

Vocalization

Although SACs are not highly vocal, they do have a repertoire of sounds. Alpacas are generally more vocal than llamas. A gradient of sounds has been described as humming (bleating). The pitch and tone of the humming is significant in SAC communication. Franklin describes the contact hum as an auditory contact between herd members and especially between a mother and her cria. Status humming is a deeper tone that communicates contentedness, tension, discomfort, pain or relief. The interrogative hum (question) is higher pitched and has an inflection at the end. Other variations in intonation are described as a separation hum and a distress hum.[4]

Llamas emit a snort characterized by a short burst of air through the mouth with loose lips. The snort indicates mild aggression. A clicking sound can be made with the tongue, which also indicates mild aggression. A grumbling threat is emitted when a feeding animal is approached too closely by another, or when an aggressor is about to regurgitate on an offender.

Screaming indicates extreme fright. Some llamas and alpacas scream continuously when restrained for diagnostic or therapeutic procedures. Screeching is a loud squealing sound, usually made by males chasing one another during a territorial dispute or when males are fighting with each other.[4]

The SAC alarm call is emitted when a male or female perceives danger to be near. The approach of strange dogs or other predators may trigger an alarm call. The sound is a high-pitched staccato series of sounds and

has been described as whistling or neighing, and by some as similar to the braying of a hoarse donkey. When the alarm call is sounded other SACs within hearing become alerted and turn toward the source of the sound.

Offensive and Defensive Behaviors

Spitting behavior is, unfortunately, one of the few characteristics of camelids known to the general public. In reality, llamas and alpacas are generally placid around people and it is rare that the behavior will be exhibited. However, spitting is the ultimate response in social intercourse between SACs, if mild threat displays are disregarded. The material spewed out of the mouth may be saliva or feed material if it happens to be in the mouth at the time.

It is interesting to watch an annoyed cria spew out a vapor of saliva. The reflex response is present, but the function of the first compartment of the stomach has not yet been initiated, so there is no ammunition other than saliva.

The behavioral sequence of spitting begins with the ears laid back against the neck and a gulping or gurgling sound will be heard in the throat region, followed by regurgitation of a bolus of ingesta from compartment one of the stomach. Ingesta is then spewed out of the mouth in a diffuse pattern and may spray as far as 1 or 2 m.[2,3] An unfortunate human recipient will find that the obnoxious odor persists until after a shower and shampoo.

It has been the author's experience that alpacas are more prone to spitting than are llamas. It is also recognized that individual llamas may develop a dislike for a particular person. Veterinarians are often selected for such disfavor.

South American camelids normally urinate and defecate at communal dung piles (Figure 3.10). The ritual begins when first arising in the morning at daybreak. This may be the only time that urine samples and fresh fecal samples may be collected. The dung pile is a social gathering site. Males, as well as females, partially squat and project urine rearward, clear of the hind limbs. Frequency, position, and duration of urination and defecation are important indicators of incipient illness.

Another characteristic behavior is dusting and rolling (Figure 3.11). This must be differentiated from the rolling that may occur with colic.

Offensively, male SACs bite, charge (Figure 3.12), chest butt (Figure 3.13), and rear up and strike down on another male. Properly reared and trained, male llamas are as safe and as easy to handle by humans as female llamas. Imprinted males are a different matter. An SAC usually kicks with a forward and outward thrust (commonly called cow-kicking). Certain individuals are more prone to kicking than others and alpacas are notorious for their kicking. SACs are not limited to cow-kicking. The author has been kicked when pressing a pack llama too closely from behind.

Figure 3.11. Llama rolling (dusting).

Figure 3.10. Defecation and urination posture, alpaca.

Figure 3.12. Llama male charging.

Figure 3.13. Llama males chest butting.

Figure 3.15. Llama in normal sternal recumbency, legs out in front.

Figure 3.14. Llama in resting, sternal recumbency.

Figure 3.16. Llama in normal resting lateral recumbency.

Recumbency

Sternal recumbency is the most common position for rest and relaxation for llamas and alpacas (Figure 3.14). In fact, that position is considered the default position for them when faced with an unpleasant situation such as toe-nail trimming or blood collection. When lying sternally, the front legs are usually folded beneath the chest, but SACs have the unique capability to lie with the forelimbs extended forward (Figure 3.15). South American camelids have a pronounced calosity over the sternum and they may remain recumbent sternally for hours to days without compromising the circulation of the limbs. Lateral recumbency (Figure 3.16) is also a normal position, with the animal apparently sleeping or sunning itself via the thermal window (Figure 3.17). An evaluation of the forms of recumbency is important in disease diagnosis.

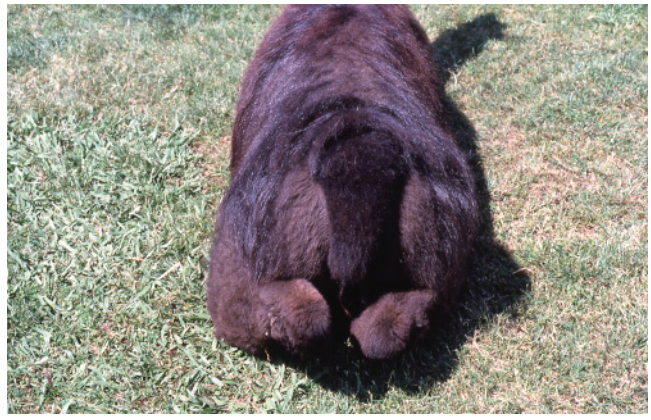

Figure 3.17. Llama sternal, hocks elevated to lift body for air circulation over thermal window.

Old World Camelid Behavior[4,6,7,8,16,21]

Camel ears are rather short compared with SAC, but ear position still reflects mood in OWC. The usual ear position for a relaxed camel is with the ears vertical on the head and rotated laterally (Figures 3.18, 3.19). A camel alert to some activity nearby cocks its ears forward. A frightened or angry camel positions the ears rearward. The more anger, the more rearward the position.

Figure 3.18. Head of a relaxed camel, ears vertical and turned laterally.

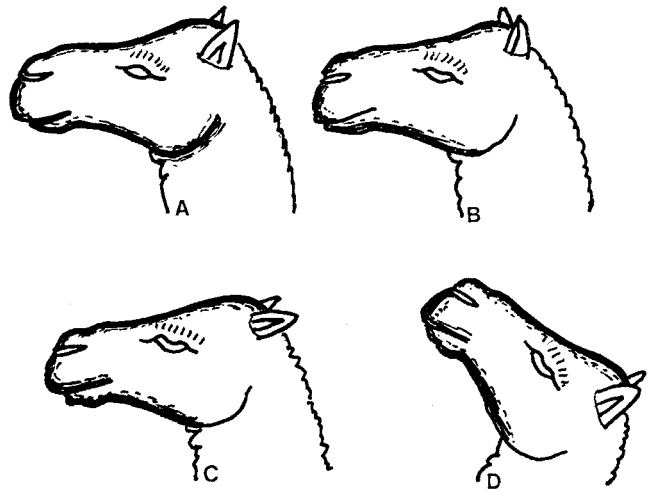

Figure 3.19. Diagrams of camel ear and head positions. (A) Usual ear position, (B) alert or attentive position, (C) upset or angry position, (D) threat display or flehmen.

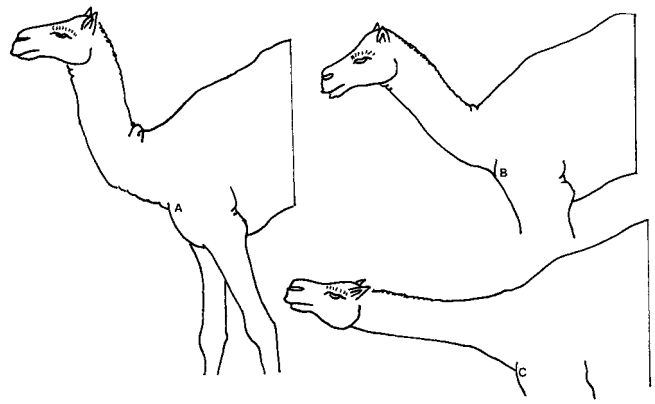

Figure 3.20. Diagrams of camel head and neck positions. (A) Alert stance, (B) relaxed or walking stance, (C) charging or running downhill stance.

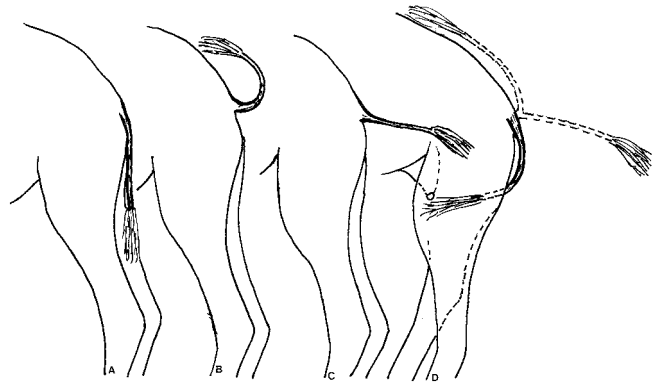

Figure 3.21. Diagrams of the tail set of camels. (A) Normal position, (B) excited, angry, or pregnant, (C) pregnant, (D) tail flipping of male camel in rut.

Figure 3.22. Camel stance for urination and defecation.

Head and neck position also reflects emotional state. A relaxed camel holds its head at approximately the same height as the hump (Figure 3.20). An alert or attentive camel positions the head 20 to 38 cm (8 to 15 in) above the hump. When running or charging, the head is held low and extended.

The tail of an agitated camel is elevated (Figure 3.21). The tail of a male camel in rut is constantly wet with urine. In rut, the tail is flipped between the legs as the male dribbles urine (Figure 3.22). Then the tail is flipped up over the back. Soon the back of the rutting camel is encrusted with a foul-smelling mixture of urine, dirt, and the secretion from the poll gland.

Vocalization

Females and their offspring exchange a variety of "mmms" or hums depending on the situation. The vocalization of adult females interacting with each other varies from a growl, to a roar, or bellow, depending on whether they are contented, angry, or frightened. The older females in a group are usually less vocal than the younger ones. Camels are chronic complainers when being handled or restrained against

Figure 3.23. Dromedary bull with dulaa protruding.

their will. Although camels do not have an alarm call as the South American camelids do, a camel may emit a bark-like sound when excited or concerned about activities around them.

Bulls in rut utter a unique blubbering or gurgling vocalization produced by blowing air through the dulaa (diverticulum off the ventral aspect of the soft palate, that is protruded through the mouth) (Figure 3.23). Saliva is also produced in abundance and this is whipped into a froth that further modifies the sound.

Offensive and Defensive Behaviors

Camels have been domesticated for thousands of years and are easily managed if accustomed to being handled by people. Improperly trained adult camels (zoo camels, privately owned camels, feral camels) may inflict serious to fatal injuries to an unsuspecting handler. Even well-trained adult bulls may become belligerent and dangerous while in rut.

Spitting. Although camels, particularly young camels, may project saliva from the mouth, more commonly stomach contents are spewed. Spitting is not used to intimidate or threaten herd mates, as it is in South American camelid societies, but both Bactrian and dromedary camels are capable of spewing first-compartment stomach contents at people when they become angry or frustrated. While one may predict the projectile pathway in SACs, thus allowing a handler to point the head away from people, not so with camels. They are able to spit out of the side of the mouth to spray a handler standing at their side.

Biting. Camels are able to open the mouth wider than any other ungulate, making them capable of grasping any part of the human body, including the head. Adult male camels have a formidable array of caniniform teeth. A prelude to biting may be the snapping together of the teeth, which is part of a threat display.

When a male camel bites, he clamps on the object and begins to shake his head. This may cause considerable contusion and severe lacerations. Children have been grasped by the head, lifted off the ground and the neck fractured in the ensuing shaking.

A bull Bactrian camel grabbed a nursing calf by the back, perforating the thorax and lacerating the diaphragm, producing a pneumothorax and a diaphragmatic hernia. When adult males in rut resort to serious fighting, they may bite their opponent on the head, neck, legs, and scrotum.

While a male in rut may be normally aggressive to other camels and humans, abnormal aggressiveness of a non-rutting male has been experienced by the author. A calf was orphaned because he had marked laxity of the limb joints, preventing him from standing to nurse. As a result his mother abandoned him. He was given a name, taken into the owner's home, and raised as a companion animal, without contact with other camels. The owners failed to recognize undesirable actions, thinking them charming, and even encouraged him to misbehave. In a camel society he would have been severely disciplined as he stuck his nose into everything and wanted to be the center of attention.

After weaning, the juvenile was placed into a camel herd. As he matured, he became overly pushy. He would be the first to approach a person entering the corral and would demand to be fed first. Ultimately he was moved to another farm.

After he became an adult, I was inside the corral with him and his herd mates, and he singled me out and charged me in the manner of a male in rut. His mouth was open and he was bellowing. Fortunately, I had a lead rope in my hand at the time and was able to deflect his first charge, but he persisted, trying to push me into the fence. My only escape route was over the tall fence. I had to continually flail at him with the rope to prevent him from biting me while I scrambled over the fence. Had I not had the rope, I could have been severely injured or even killed.

Such behavior is all too common in South American camelid males that become maladjusted to humans at an early age (berserk male syndrome, imprinted male). When maladjusted males become sexually mature, they may treat a human just as they would another

male. It appears that camel raisers should also allow camel calves to live in a camel society. If necessary, milk may be supplied by caretakers, but caresses and tenderness should be avoided.

KICKING. All four feet and legs may become formidable weapons. The front legs may strike out in any direction. The rear legs are able to reach forward to scratch the head. Thus, there is literally no safe place to stand around an untrained camel, as there is in the horse (at the side of the withers). One must also appreciate the length of the swing of a camel's limb. The lack of a skin fold forming the flank provides extreme mobility for the rear limb.

A Bactrian camel tied within a shed became angry and kicked out with a rear limb. His foot struck and shattered a 10-cm × 10-cm (4-in × 4-in) solid wooden post that was serving as a roof support. One should readily appreciate what would happen to a person's leg, hit in such a way.

Other aggressive behaviors include charging and bumping a person. If the victim is knocked down, which is most likely to happen, an aggressive camel may stomp on the person as they would do to another camel that had been knocked down during a fight.

PHYSICAL RESTRAINT

There is a distinct handling difference between llamas, alpacas, and camels.[9,10] Each will be discussed separately.

Llamas

Experienced llama owners recommend that a small catch pen be associated with a pasture. This area can incorporate a shed or feeding area so that camelids become accustomed to entering it. Designs of such facilities are as variable as the mind can imagine. If advising a client about the design of future facilities, appropriate principles of design for catch pens for cattle and sheep may provide guidance.

If no catch pen is available, it is relatively easy to restrict one or many llamas with ropes, poles, or even humans with outstretched arms. Two or more people can corner a llama by approaching it slowly with arms outstretched (Figure 3.24). Only one person should signal the team to move forward or retreat.

Two people may hold a rope (minimum of 10 m [32.8 ft]) taut between them 1 m (3.3 ft) above the ground (Figure 3.25). The intended captive should be moved along a fence line to a corner. It is easier to contain all members of a group at once. Most llamas will not challenge the rope. Occasionally, an individual will run under the rope or try to charge through it. If a member of a group escapes capture, others will also attempt to escape. When the group is cornered, the rope should be shortened until the animals are completely restricted. One end of the rope may be tied to the fence to free a handler to move among the llamas to place a rope or halter on the animal selected.

Figure 3.24. Directing and containing with outstretched arms.

Figure 3.25. Containing llamas with a rope.

In a large pasture, with more help, two ropes may be used in a crisscross pattern. With this method, a llama group can be restricted along a fencerow instead of in a corner. A modification of this technique employs long bamboo poles or plastic pipes, each carried by a single person in the manner of a tightrope walker (Figure 3.26). Two or more of the poles are used to herd and contain the llamas.

The method used by llameros in the Andes to load packs is as follows: pack llamas are driven into a group; llameros surround the group with llama fiber ropes (Figure 3.27). While the rope ring is in place, other llameros work their way into the herd and with a shorter rope tie three animals together by their necks (Figure 3.28). The rope is looped around the neck of the first llama, and the ends given one or two twists. Then they are looped around another and twisted, and

Figure 3.26. Containing a llama with plastic pipes.

Figure 3.27. Rope corral around Peruvian llamas.

Figure 3.28. Three llamas tied together with a neck rope.

finally a third llama is included before the ends are tied together. Once all the triads are roped up, the perimeter rope is dropped. Because three llamas tied together will not all go in the same direction, they stand still. Packs may then be placed on their backs.

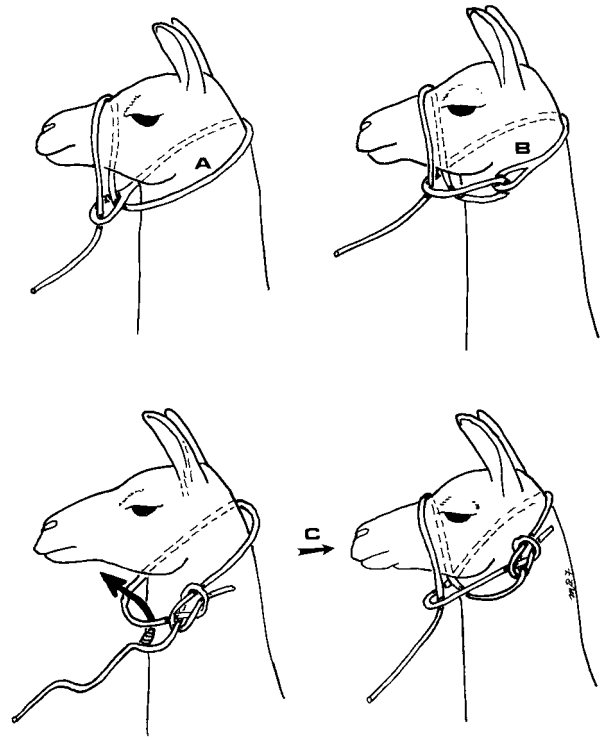

Figure 3.29. Diagrams of temporary rope halters.

A handler may not be able to enter an enclosure housing an aggressive, imprinted male. It may be necessary to rope such an animal to gain initial contact. A halter may then be placed or a temporary rope halter fashioned from the loop around the neck (Figure 3.29).

Llamas generally dislike having their heads touched or scratched, unless gentle desensitization has been carried out. Trainers usually state that llamas are sensitive about the head because every time someone touches them something less than pleasant happens. To place a halter, the llama should be approached from the left side at the withers. The right arm should reach over the neck while the halter's poll strap is pushed under the neck to be grasped with the right hand. The nose loop should be slowly moved up and positioned over the nose. The llama may try to dodge this action. With the nose loop in place, the poll strap can be buckled or snapped to the cheek ring. If placed correctly, the nose loop should not slip down over the bridge of the nose, should the llama pull back against the restraint.

Halters used on llamas resemble pony-sized horse halters. Most llama owners prefer not to leave a halter on a llama when it is free in a corral or pasture for fear a strap may catch on something and the llama be injured. Some llama owners leave a leather or fiber neck band in place to facilitate capture.

In a restricted area the llama may be slowly approached and one arm placed around the chest and

neck while the opposite hand grasps the tail. This is similar to the way a sheep is handled. It may be difficult to hold a llama weighing more than 150 kg (330 lbs.) in this manner unless it can be quickly pushed against a wall or fence.

None of the foregoing techniques are suitable for single-handed containment of a spitter, since the llama can easily turn its head and spray the handler. With a spitter, a second handler must quickly grasp the head or ears to direct the spray away from the handlers, then place a "spit rag" over the muzzle. With some obstreperous individuals, the approach may have to be with a garbage can lid or a large plastic bag slipped over the head for protection (eye and arm holes must be cut out).

Earing

The llama and alpaca respond to "earing," as does the horse.[5] If this hold is applied correctly, the animal will not become "ear shy" or be more difficult to capture another time. Before grasping an ear, a handler should explain the procedure to the owner and inquire if this is acceptable. Some owners do not understand the process and may resent the "rough" handling.

The procedure for earing a llama is similar to that for earing a horse.[5] The handler should stand on the left side of the withers, place the right arm over the neck, and work the opened palm of the hand up the neck to surround the base of the ear (Figure 3.30). The handler should squeeze firmly because the llama may try to pull free at this point. Frequently, the llama will jerk its head toward the left and the handler to escape pressure on the right ear and use its head as a battering ram. One handler suffered a broken nose when an obstreperous llama slammed its head into her face. The left ear may be grasped as well for additional restraint.

An individual llama in a herd may be restrained for quick procedures by one handler grasping the ears and another grasping the tail. Greater restraint is achieved by pushing the llama against a fence or wall. Care should be taken to ensure that the llama does not put a foot or leg through a net fence or under or through a wooden fence or wall.

Figure 3.30. Earing a llama.

Chutes and Stocks

Tractable llamas may be cross-tied in a narrow alleyway or in a conventional equine stock. Semi-permanent pipe stocks are available, but animals must be trained to be handled in a pipe stock. A number of injuries to llamas and handlers have occurred in such facilities. Stout, aggressive llamas can lift the stock if it is not bolted to a floor or platform. Once, such a stock was lifted and dropped on a handler's foot, fracturing the metatarsal bone. The jaws of the llama may be traumatized when anchored to the padded yoke of such stocks. Both back bands and belly-bands may be used, but one llama ruptured its bladder while struggling against such restraint. Too often, a novice llama handler uses maximum rather than the minimum restraint needed to accomplish a task. The pipe stock is frequently used to clamp a llama so tightly that it can't move, when instead an appropriate sedative or anesthesia should be used.

Many homemade stocks function admirably. Some provide more flexibility in use than others, and there is wide variation in cost and ease of construction. A few designs are illustrated.

Fowler Design. This is a simple, easily constructed chute/stock (Figure 3.31). The neck is readily accessi-

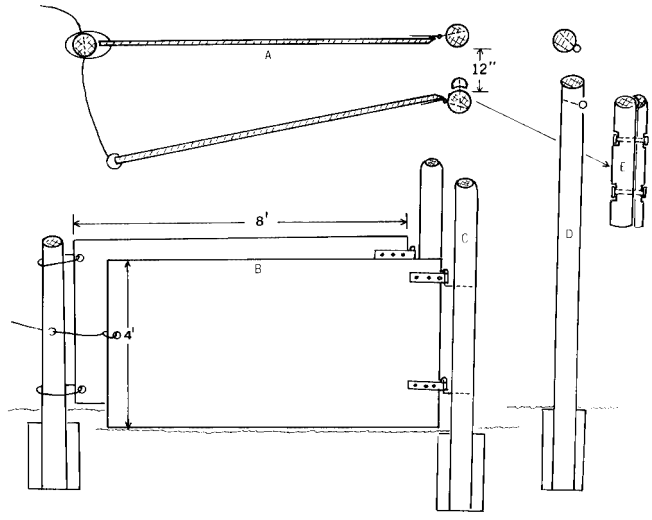

Figure 3.31. Diagram of the Fowler-designed squeeze chute for llamas. (A) Top view, (B) side view, (C) shoulder post, (D) tie post, (E) shoulder post with detachable scab for small llamas.

Figure 3.32. Fowler-designed llama chute, front view.

Figure 3.33. Fowler-designed llama chute, rear view.

ble for blood collection. Reproductive tract examinations can be conducted with the squeeze applied, and other examinations may be carried out with the squeeze released. Figures 3.32 to 3.33 illustrate the construction and actual use of this chute. A narrow space is allowed through which the head and neck may pass but which holds the shoulders. The head should be pulled forward and upward and secured to a recessed ring on a wall or post placed forward of the shoulder poles, as indicated.

The chute may be constructed against the solid wall of a barn (Figure 3.34), or both sides may be left free to swing on hinges at the shoulder posts. Sheets of 2.54 cm × 1.22 m × 2.44 m (1 in × 4 ft × 8 ft) plywood are hung on heavy-duty hinges. A back band may be used to prevent rearing, but the llama should be able to lie down if necessary or desirable.

Ebel Design. This is an excellent, versatile design (Figure 3.35).[11] It requires more space for construction than the above, but may be portable. Basically, this is a three-sided, reinforced plywood box, open on the top and at both ends. Adjustable shoulder poles prevent the llama from moving forward. The head is crosstied to the sides of the box. Access is provided to both sides, the front, and the back of the llama.

Imprinted Males

Mother-reared male SACs are usually no more difficult to handle than females, in contrast to bulls, stallions, and rams. Bottle-fed orphan males or neonates given too much human attention may imprint on humans and develop a dangerous behavioral pattern. Essentially, an imprinted male treats a human as if he or she is another male SAC.

This behavior may be anticipated in the male youngster that approaches and pushes its nose into a person's face or gallops up and pushes the owner around with its neck or chest. Peculiarly, overt aggression may not manifest itself until maturity, and then a particular person may bear the brunt of the aggression. A number of people have been severely injured by vicious attacks of otherwise handleable males.

In a full attack, the ears are laid back against the llama's neck. He will charge and attempt to knock a

Figure 3.34. Fowler-designed llama chute constructed at the side of a barn.

Figure 3.35. Ebel-designed llama chute.

person down by butting with his chest. If this fails, he may rear up and slam down on the person. A person who falls will be trampled with the llama's forefeet and bitten. A victim who remains standing may be bitten at the neck, knees, or any other spot within reach. When male llamas fight with other males, they bite the neck, hind limbs, and scrotum.

Several anatomic adaptations in male SACs protect against fatal injuries during such encounters. The skin of the upper cervical region is as much as 1 cm thick. Instead of lying superficially in a jugular furrow, the SAC jugular vein lies deep, in juxtaposition to the carotid artery. Finally, a ventral projection of the transverse process of the cervical vertebrae forms an inverted U channel that protects vital cervical structures.

Aggressive males are ferocious, uttering loud vocalizations and attempting to climb fences to get at intended victims. One male alpaca attempted to climb up onto the seat of a tractor to reach a young driver. Once this behavior has been exhibited, contact with that SAC is no longer safe for any person. A few individual animals have been deconditioned with negative stimulation, but usually safety has been achieved only for the individual deconditioning them. Others remain at risk.

Not all imprinted males develop aberrant behavior, and some may become obnoxious but not dangerous. It is also possible to hand raise an orphaned male SAC without it becoming imprinted, if no loving attention is given while it is nursing and it is returned to the herd after feeding to be socialized as an SAC. Be aware that hand-reared females may also develop aggressive tendencies, but this is less common than in males.

Castration of adult males is not effective in changing abnormal behavior, as it is in bulls and stallions. It is recommended that orphaned males that have been given extensive human attention be castrated by two months of age or, at the latest, before weaning.

Alpacas

Alpacas are social animals with a strong flock orientation, which may be an advantage when driving

the animals into a smaller enclosure for close observation or capture.[3] The docility of individual alpacas may be assessed by observation of the ear set and tail position.[4] The aggressive or agitated alpaca pulls the ears rearward over the neck, much as does an agitated llama. Although the alpaca has shorter ears, the position is similar to that seen in a hostile llama. Alpacas often protest vociferously during restraint procedures. Behaviorists recognize two types of vocalization during restraint and handling. A frightened alpaca screams, but the extremely angry alpaca may actually screech. Most vocalization during handling consists of screaming. Screeching is heard more when two males are fighting. Alpacas tend to jump more than llamas when physically restrained, or conversely, they go into a kushed position more readily.

The most common human injury caused by an alpaca is a bloody nose or thick lip, produced when the alpaca raises the head explosively, striking the handler in the face. The alpaca is not purposefully trying to injure the handler; rather, this is a normal reflex. When being approached in a group, an animal may have its head down, as if to hide, may get behind another animal, or may go under a fence. When a handler grabs an alpaca, its normal response is to lift the head quickly.

It is always desirable to use the least amount of restraint necessary to perform a task. Alpacas are the most social of the camelids, and a herd should be handled as a unit until selected individuals are grasped. Many alpacas have been taught to halter and lead. Untrained alpacas are best controlled by pulling the head and neck close to the chest with one hand while the other hand rests on the top of the shoulders (Figure 3.36). Alternatively, the tail may be grasped by the second hand (Figure 3.37). A second person, if available, may grasp the tail close to its base to prevent the rear end from swinging around. If a person is working alone, a lead rope may be half looped around a fence post and the animal pressed against the fence. The alpaca must be prevented from poking a leg under or through the fence.

Alpacas may be herded into a narrow alleyway or catch pen. They may also be baited into a catch pen with hay, pellets, or grain, or on a warm day, by spraying water. Alpacas may also be herded into a corner with ropes or poles or by humans with outstretched arms. The arms should not be waved exuberantly because this may alarm the animals.

Optimal veterinary care of alpacas requires suitable restraint facilities. Confined areas (alleyways or stocks) should be provided to allow work on the animals to be done safely and efficiently. Blood collection, diagnostic examination, and, particularly, reproductive tract examination may necessitate a chute to control the head and prevent excessive movement of the rear quarters. Chutes designed for llamas are generally not suitable for alpacas, and many alpaca managers are opposed to the use of chutes. While most procedures may be performed by manual restraint, a thorough examination for infertility is much more safely done in a chute. Training for standing in a chute is just as necessary as halter training. Alpacas should never be left unattended in a chute.

A simple chute that may be adapted to an animal of any size is described in the llama section under Fowler Design.

Figure 3.36. Restraint of an alpaca by holding the head close to the chest and pressing a hand on the withers.

Figure 3.37. An alternate method of restraining an alpaca.

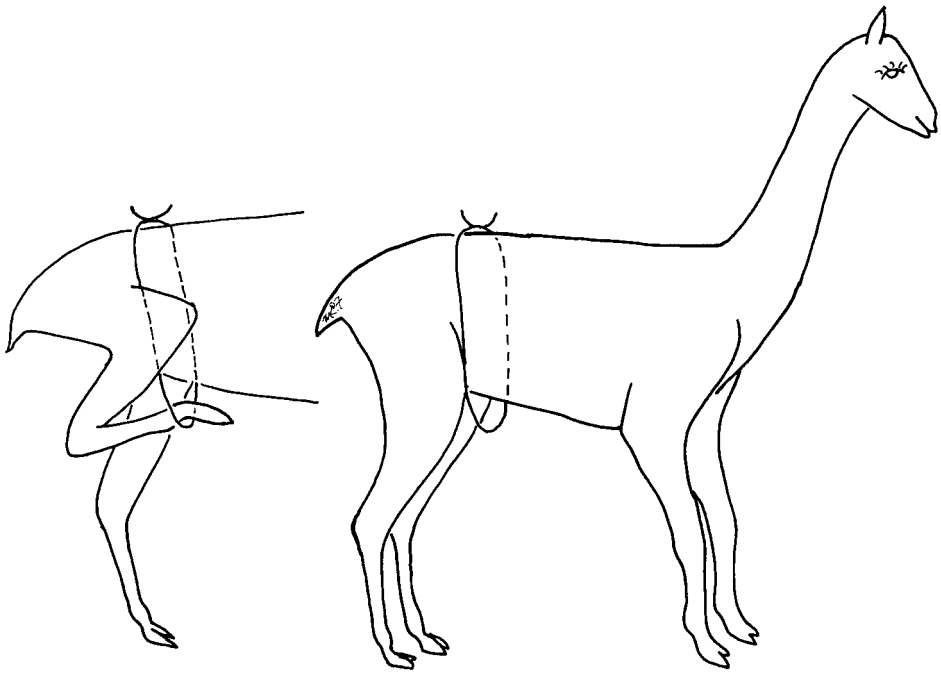

Figure 3.38. A diagram illustrating chukkering.

Chukkering

Alpacas may be held in the kushed (recumbent) position by restricting the hind limbs. A loop of soft cotton rope (llama fiber rope is used in South America) with approximately 15 cm (6 in.) of slack is tied around the body just ahead of the pelvis (Figure 3.38). The handler reaches across the back and lifts the opposite rear leg and places the foot in the loop under the abdomen; then the other foot is placed in the loop. This causes the alpaca to sit down and, eventually, kush (Figure 3.39). The front legs may be restricted by placing a loop over the flexed carpus as is done with the camel. Alpacas have been carried in the cabin of a private small airplane under this form of restraint.

Figure 3.39. A chukkered alpaca.

Guanacos and Vicuñas

Guanacos may be tamed if reared in association with humans and handled during maturation. Such animals may be handled in a similar manner as llamas or alpacas, but it should be recognized that guanacos may be more easily frightened or excited. Free-ranging newborn guanacos may be hand captured within the first few hours of birth, but thereafter they can run as fast as their dams.[13]

More vigorous manual restraint is needed to control wild guanacos or vicuñas. Two or more people may be needed to catch an animal within a restricted space such as a small stall. Most of these animals will not tolerate being tied with a halter and will fight if placed in one of the chutes designed for their domestic cousins.

A less traumatic and stressful method of restraint is intramuscular chemical sedation via a blow gun or stick pole syringe (Chapter 5). Neither the guanaco nor the vicuña has a heavy fleece to obstruct insertion of the needle into the muscle mass of the upper rear limb or triceps. The stick pole syringe method may also be

used to sedate an extremely aggressive male llama. For more specific handling and training procedures, the reader is advised to consult training texts.[12–14]

Camels

Information on the restraint of camels is needed because these animals are now being raised by private individuals in North America.[10] Male camels have five pairs of large canine teeth, which are employed in intraspecies aggression with other males vying for a female. Bites from Old World camels may be fatal to humans (Figures 3.40, 3.41). Handlers must be continually alert when working around even tame camels, because they may kick in any direction. A camel has great flexibility—it can reach up and scratch its head with a hind foot.

Psychological Restraint[6,10]

The degree of psychological restraint that may be employed is a factor of the taming and training that has been done. Ear position is not as apparent as in llamas or alpacas, because the ears are not as long or as expressive, but they reflect mood. The farther the ears are pulled rearward, the higher the degree of agitation. The tail of an agitated camel is elevated. Vocalization is a dead giveaway of a camel's displeasure. Restraint of even a mild degree is likely to elicit a chronic complaint roar. Unaroused camels emit a low-pitched grumble, but when annoyed, the pitch rises to a whine or roar. Handlers should become accustomed to the change in pitch and be aware of the subsequent action that may follow.

The voice of a handler reflects her confidence and/or fear of a camel. Other body language changes also may be involved. Voice changes in the handler may be so subtle as to go unnoticed by many people, but the camel may perceive the change. People with a musical ear are more likely to notice a slight change in pitch.

Confinement

To capture a single animal in a herd, it may be wise to take advantage of the social behavior of camels and move them as a group to a smaller enclosure, alleyway, box stall, or catch pen.

Physical Barriers

Numerous types of chutes and stocks have been used to restrain camels. The design and construction of stocks is as variable as the designer. The author has handled many camels in a standard equine stock (Figures 3.42, 3.43). In this design, both the front and rear gates are padded to minimize trauma should the animal kick or strike and to protect the examiner. A heavy, soft cotton rope stretched between the bars and attached to the loops provided may restrict forward and rearward movement. Sturdy pipe could also be used. This stock will accommodate the average female camel.

The following design was constructed at a private camel farm (Figure 3.44). The heavy pipe construction provided adequate strength for containing the camels, but its original design with double gates in front and back with 91 cm (36 in.) between the side frames allowed too much movement within the stock. It also

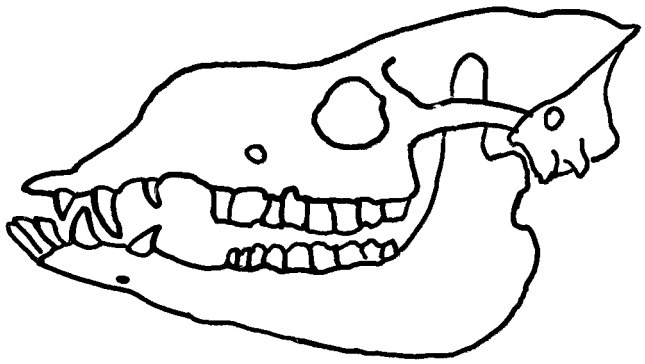

Figure 3.40. Prominent canine teeth in the camel premaxilla and mandible.

Figure 3.41. Camels can open their mouth widely.

Figure 3.42. Camel in an equine stock.

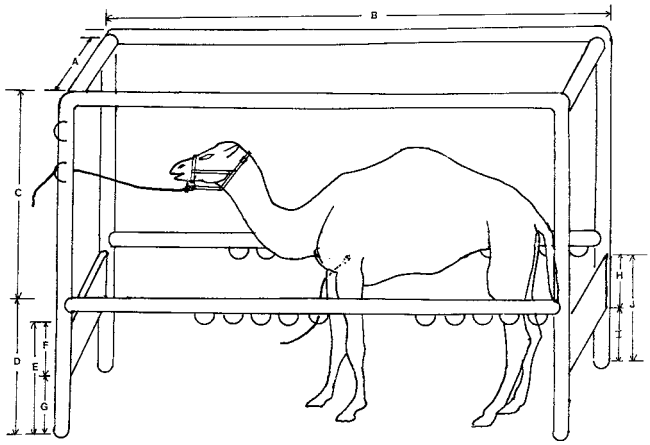

Figure 3.43. Diagram of an equine stock, including measurements. (A) 76 cm (30 in.), (B) 2.1 m (84 in.), (C) 1.8 m (70 in.), (D) 1.2 m (48 in.), (E) 1 m (41 in.), (F) 53 cm (21 in.), (G) 51 cm (20 in.), (H) 53 cm (21 in.), (I) 36 cm (14 in.), (J) 89 cm (35 in.). Pipe is 7 cm (3 in.). Front and back gates padded.

Figure 3.44. Camel in California-designed stock. There is too much room in which to move around.

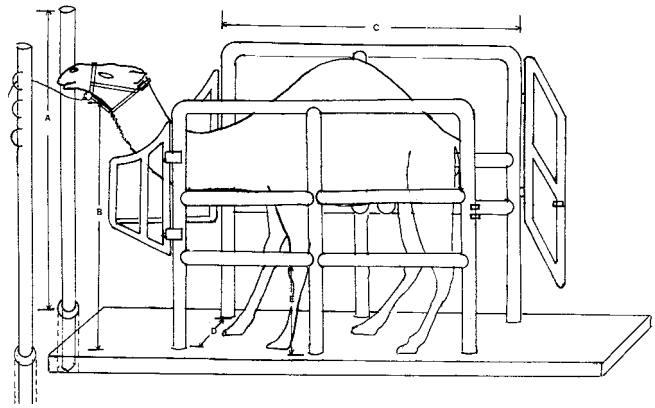

Figure 3.45. Diagram of a modified California-designed stock, including measurements. (A) 2.1 m (84 in.), (B) 1.6 m (64 in.), (C) 2.1 m (84 in.), (D) 76 cm (30 in.), (E) 61 cm (24 in.). Pipe, main frame, 8 cm (3 in.); gates, 6.4 cm (2.5 in.).

Figure 3.46. Stock used for embryo transfer studies in Al Ain, United Arab Emirates.

did not provide adequate access to the rear of the animal for rectal examination. The author recommends a modification of the original design, with a single swinging gate at the rear and a curved front gate (Figure 3.45). I would suggest only 76 cm (30 in.) between the side panels. I realize that some extremely large animals may have difficulty in fitting into that narrow space, but for a stock to be effective, side-to-side motion must be limited.

A stock used in a camel facility in the United Arab Emirates is illustrated in Figure 3.46. These were designed primarily to examine the reproductive tract and specifically to conduct embryo transfers.

The author's preferred stock is a simple design that does not require the use of pipe. The Fowler design may be constructed next to a barn or solid wall, as illustrated in Figure 3.47. A heavy post is set

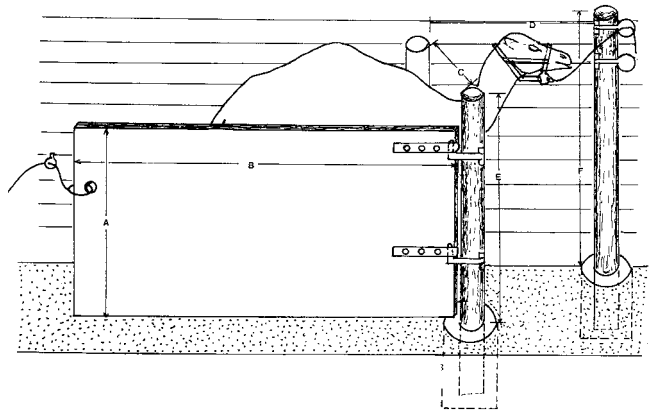

Figure 3.47. Diagram of Fowler-designed stock. (A) 1.2 m (48 in.), (B) 2.4 m (96 in.), (C) 46 cm (18 in.), (D) 91 cm (36 in.), (E) 1.8 m (72 in.), (F) 2.1 m (84 in.). Posts 15 cm (6 in.) in diameter.

approximately 45 cm (18 in.) from another post that is next to the wall. A 2.5-cm (1-in.) thick sheet of marine plywood 1.2 m (4 f) by 2.4 m (8 f), is hung on the post with heavy bolt hinges. An alternative type of construction is with both sides of the chute hung on posts so that access may be from both sides (Figure 3.48). Arrangements may be made to attach another post on the inside of one post to narrow the opening for smaller camels. A post set in the front allows the animal to be tied, thus restricting backward movement. Once the camel is tethered, the gate may be swung closed, restricting side motion. Bales of hay or straw may be placed behind the camel if a rectal examination is necessary. Figure 3.49 illustrates the principle of this design using an aluminum gate, with the camel against a wire fence. The chance of an injury to the camel is greater with the aluminum gate. Any of the open sided stocks may be made more handler kick-proof by lining the stock with plywood sheeting. Bales of hay or straw protect the examiner from kicking.

The best-designed stock or chute is useless unless the camel will enter it. The key is the placement of the stock or chute in relation to the corral design. If the chute is located in an area that is strange to the camel, or if the camel is not trained to walk into the chute, handlers will be unable to coerce an adult camel to enter.

Untamed camels are a special problem. This is often the situation in zoos or breeding facilities where little attention is paid to the animals except when something is to be done that is unpleasant for them. A special catch pen should be provided in these facilities to allow the animals to be brought from a larger enclosure or pasture to a more manageable enclosure. Preferably, the camels have been fed at least part of their feed in the catch pen, so they are willing and accustomed to entering it.

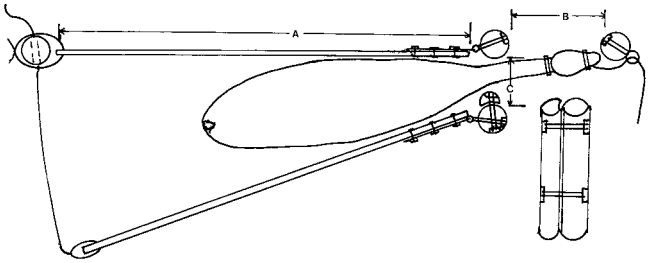

Figure 3.48. Fowler design, top view. (A) 2.4 m (96 in.), (B) 91 cm (36 in.), (C) 46 cm (18 in.).

Physical Restraint

Positioning a camel in sternal recumbency (kushing) provides an opportunity to closely examine or collect laboratory samples without risk of being kicked or struck. Obviously this necessitates a camel being trained to kush on command. The camel may be secured in the sternal position by using a body harness (described below). A camel may jump up quickly if painful procedures are performed without benefit of a sedative or local anesthesia. A handler may augment the restraint by pressing a foot on the top of the radius.

A body harness has been used for centuries in camel-using countries. Either a leather strap or rope is used to place a loop or figure-8 around the front limb when the leg is flexed at the knee (carpus). Using only the loop around the front leg, the camel may still rise to its knees. Most camels are trained to allow hind-limb physical restraint as follows: A person on either side of the camel brings a soft cotton rope up behind the hind limbs as the camel is being directed to kush (lie down); optimally the rope should be below the fetlock (Figure 3.50). Once recumbent, the rope is placed medial to the stifles and tied tightly over the back behind the hump (Figure 3.51). Rising on the

Figure 3.49. Temporary stock using an aluminum gate to press the camel toward a fence. Bales of straw protect the examiner.

Figure 3.50. The rope is brought up behind the pasterns or rear legs.

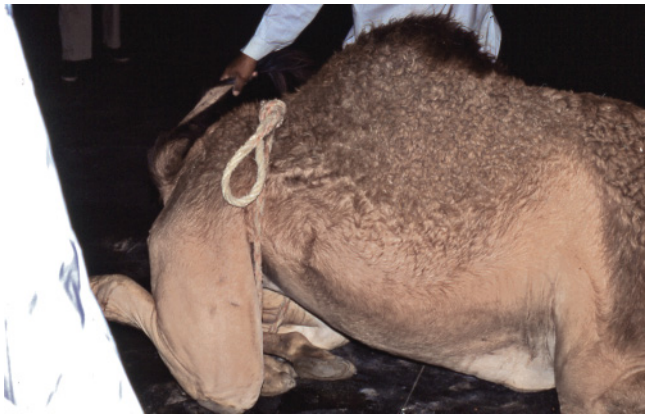

Figure 3.51. The rope is placed beneath the pasterns and on the medial side of the stifles, then tied over the back.

Figure 3.53. Wooden nose plug.

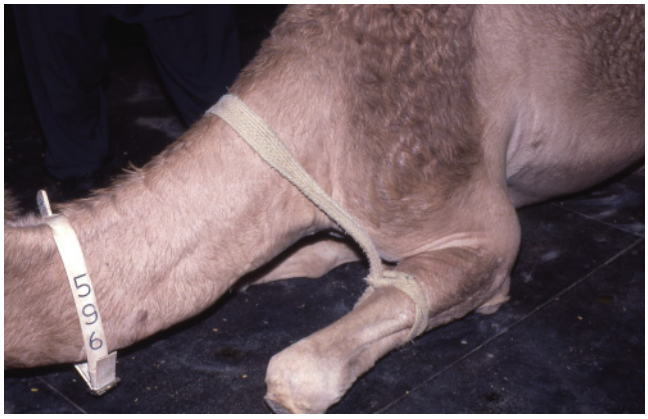

Figure 3.52. The strap is placed around both front legs and over the top of the neck.

Figure 3.54. Nose plug in place.

forelimbs is prevented by placing a loop over a flexed limb, extending the rope over the top of the neck, and securing the opposite fore leg in the same manner (Figure 3.52).

Head Control. The method of catching a camel and controlling the head varies with the experience and culture of those carrying out the procedure. Camels may be roped with a lariat, but the roper needs to keep the loop as high on the neck as possible to provide better control of the head.

A loop may be placed around the neck to pull the head toward a barrier, thus allowing placement of some type of head restraint. In countries where camels have been beasts of burden for centuries, a head restraint may consist of a wooden peg placed through the side of the nostril (Figures 3.53, 3.54). A small cord is attached to the peg. Pressure on the peg signals to the camel a desired response. Usually, the tactile contact is accompanied by a verbal command which may vary with the trainer, but common English terms include "gee" (right), "haw" (left), "go" (move-out, step-up, hup), "stop" (whoa), and many others.

If the peg is pulled too vigorously, it may be pulled through the skin. Sometimes the wooden peg is replaced with a short rope loop, left permanently in place, to which a lead rope may be attached.

Another means of head restraint is to secure a loop of rope around the lower jaw, caudal to the canine teeth and under the tongue. The foregoing restraint practices may not be acceptable in some cultures, if they are looked upon as inhumane or abusive. In reality, they are no more inhumane than piercing the ears, as practiced in people, or the use of a twitch on a horse.

In North America, South America, and Europe, the head is usually restrained by placement of some type of halter. Camel handlers should learn how to tie a bowline knot because it is fundamental to producing a non-slip or non-tightening loop in many restraint situations.

A camel should never be secured to a solid object with a lariat loop or any other knot, except a bowline tied around the neck. The risk of a camel becoming frightened, pulling back, and strangulating is too great.

Figure 3.55. Various snaps used on lead ropes. (A) Standard snap, (B) heavy-duty snap, (C), quick-release snap.

Halter designs are as varied as the individual designer. Halters may be constructed from rope, synthetic fibers, or leather. The halter should be sized and positioned to have the nose piece high on the bridge of the nose, near the eyes, such that if the camel pulls back, the nose piece will not slip over the premaxilla and occlude the nasal passages. Camels are obligate nasal breathers and may suffocate if the nasal passageways are constricted. The cheek pieces should be positioned so as to not rub against the orbit. The halter material and all buckles, snaps, and other attachments must be strong enough to hold the camel should it be come frightened or pull back. Snaps are often the weakest link in the system. Quick-release snaps that can be released while under pressure are most suitable (Figure 3.55).

Halters are rarely left on camels constantly for fear that they may catch on a post or other protruding object. Neck bands may be left in place, serving as a means of identifying an animal or as a way of catching, especially if a short segment of rope dangles from the neck band.

Bridle. The use of a bridle is controversial. The presence of the canine teeth allows a camel to grasp the bit, decreasing its function of producing pressure on the commissure of the mouth. The bit banging against the teeth may annoy the camel and cause it to continually shake its head. One owner, who exhibits an eight-camel hitch, uses a bridle with the chin strap over the bridge of the nose to hold the bit near the roof of the mouth (Figure 3.56). This eliminated head shaking in his camels.

Tethering. Camels should be trained to stand quietly when tied to a post or other solid object. Handlers should learn to tie a quick-release knot, such as the halter tie, that allows release of the knot even while under pressure (see Figure 3.68, later in text). Camels may be tethered from the halter, or by a front leg. A leather or fiber strap is attached to the leg above the ankle, and the tether is attached by means of a "D" ring on the hobble. The opposite end of the rope may be attached to a post, stake, or metal ring embedded in the ground. Commercial camel trekkers usually tether to a metal ring that is augured into the ground by means of a spiral coil. It is also possible to tether to a picket line (a rope stretched between two secure ends), either on the ground or elevated at 1 m (3.3 ft) above the ground.

Figure 3.56. Bridle on a driving camel. The strap from the bit over the bridge of the nose keeps the bit away from the lower canines.

Figure 3.57. Rope hobble on front legs.

In horses and llamas, the ears may be grasped to provide additional restraint of the animals. Camel ears are too short to be useful in this regard; however, a variation, grasping the lower or upper lip, functions as does the twitch used in equine restraint.

Hobbles. Hobbles are employed routinely to minimize straying when animals are released to graze while on trek (Figure 3.57). Usually only the forelegs are secured to one another, but some camels may learn to gallop using a short stride, with only the front legs hobbled. In that event, it may be necessary to hobble one forelimb to a hind limb.

Hobbles may be leather straps with buckles that surround each ankle and attach to the opposite leg by a short length of chain. They may also be fashioned from soft cotton rope, (1.5 m [5 ft] long and 1.27 cm [0.5 in.] in diameter). The rope is folded in half around one front leg. The two ends are twisted together for three or four turns, then the ends are tied around the opposite leg using a square-bow knot.

The use of a hobble may be important in preventing a camel from striking a handler when protective barriers are not available. A hobble may also be placed between the hind legs, but extreme caution must be exercised to avoid being kicked while applying the hobble.

PROCEDURES COMMON TO ALL CAMELIDS

Neonates

The camelid neonate may be handled by methods similar to those used for a foal or calf. One person working alone can easily handle a neonate. One arm is placed around the chest while the tail is grasped with the opposite hand (Figure 3.58). Prolonged pressure on the tail will cause the neonate to slump to the ground.

A cria of less than 20 kg (45 lbs) may be lifted with one arm around the chest and the other behind the rear legs (Figure 3.59). Larger crias may struggle and push themselves from the handler's arms. An arm in front of the rear legs has a natural calming influence on the neonate and reduces struggling (Figure 3.60).

The neonate may be restrained in lateral recumbency by casting as illustrated in Figure 3.61 and then by laying an arm over the neck and grasping the arm of the down forelimb. The other arm should pass over the body in front of the hips to grasp the thigh of the down rear limb (Figure 3.62). Folding the limbs and straddling the body in a kneeling position will force

Figure 3.59. Lifting a neonate or juvenile incorrectly by grasping behind the legs.

Figure 3.58. Restraining a cria by holding the neck and tail.

Figure 3.60. Lifting a neonate or juvenile correctly.

Figure 3.61. Method used to place a cria into lateral recumbency.

Figure 3.62. Holding a cria in lateral recumbency.

the neonate into sternal recumbency. Pressure can be adjusted as appropriate (Figure 3.63). The handler should not sit on the neonate.

From the sternal position a single person can perform venipuncture, pass an orogastric tube, or give an enema. See Chapter 21 for details.

Weanlings and Yearlings

This age group is the most difficult to restrain for blood collection, general examination, passing an orogastric tube, or other diagnostic or therapeutic procedures. These animals are too large to force into sternal recumbency, as described for neonates, and too small to put into one of the described chutes.

Many animals of this age group have not been halter trained and may struggle excessively if tied for the first time. Some llamas have suffered neck injuries,

Figure 3.63. Restraining a neonate in sternal recumbency.

sustained while "fish tailing" on the end of a lead rope. The most satisfactory method is to hold the youngster with one arm under the chest and to grasp the tail with the other hand. Even with this mild restraint, animals of this age are inclined to jump.

Dealing With a Spitter

The initial catch of a spitter is difficult. It may be roped, or if it is to be caught by hand, the catcher may be protected against ingesta spewing by wearing a plastic garbage sack with cutout arm and face holes or by using the lid of a garbage can.

A spitter may be dealt with by several methods once it is in hand. The head can be directed away from the handlers, or a towel or rag may be tucked into the nose piece of the halter and draped over the nose and mouth (Figure 3.64). The accumulation in the towel of stomach contents seems to deter further spitting, since the llama seems to dislike contact with the ingesta as much as humans.

A more permanent muzzle can be fashioned that will snap into the cheek rings of the halter (Figures 3.65, 3.66). Such muzzles are often fitted on camels that have a propensity to bite. Once a spitter is caught, the upper and lower lips can be grasped and held together, much as a hand nose twitch is applied to a horse. The llama ceases to spit because it is unable to clear the ingesta from its mouth.

Leading

It is surprising how quickly an untrained llama may learn to be led. In the beginning, it is helpful

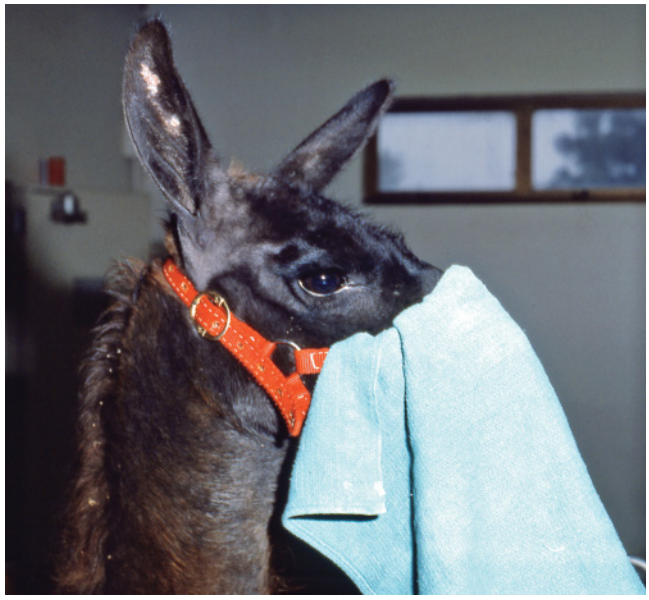

Figure 3.64. Spit rag.

Figure 3.65. A muzzle may be used to prevent spitting, biting or ingestion of feed.

Figure 3.66. Muzzle for a llama.

Figure 3.67. Tail loop used to teach a cria to lead.

for a second person to walk behind, encouraging forward progression. A single person may encourage the llama to lead by pulling forward on a loop over the rump, a technique often used to teach a foal to lead (Figure 3.67).

Llamas can be trained to jump into and out of high-level trailers and even pickup trucks. Some caution is necessary, particularly if transporting a sick llama to a clinic, because it may not have normal coordination. Even healthy llamas have fallen as they have jumped out of a truck bed.

Hospitalization

SACs use communal dung heaps for defecation and urination. Camels defecate and urinate at random. It may be difficult to induce them to defecate in strange surroundings. If trailered to the hospital, the camelid will usually have a full bladder and rectum. The animal should be given access to a dirt or grass area for voiding before confinement in a concrete or wood-floored stall.

Many types of bedding may be used for stalls. Wood shavings and sawdust work into the fleece and are extremely difficult to remove. A healthy animal unaccustomed to straw may consume more of it than is desirable, causing indigestion. However, straw is more satisfactory than shavings.

When automatic waterers are used in a stall, an alternative water source should be provided until it is certain that the animal is using the automated source.

Knots and Rope Work

Following is a review of knots and rope work for handling camelids.[9,10,11] The important knots are the halter tie (Figure 3.68), bowline (Figure 3.69), and slip-

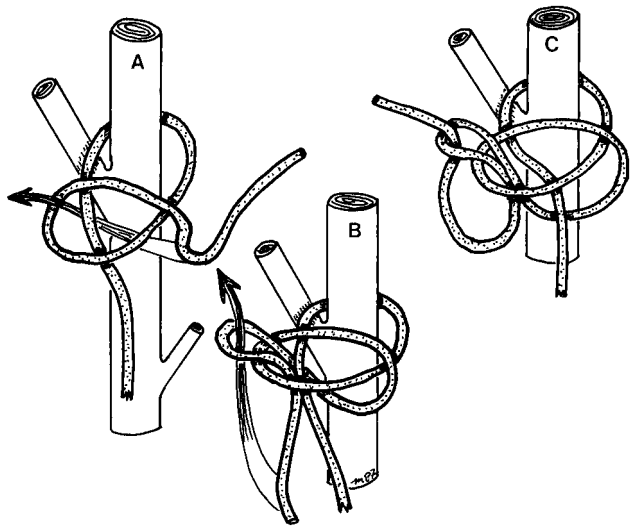

Figure 3.68. Tying the halter tie.

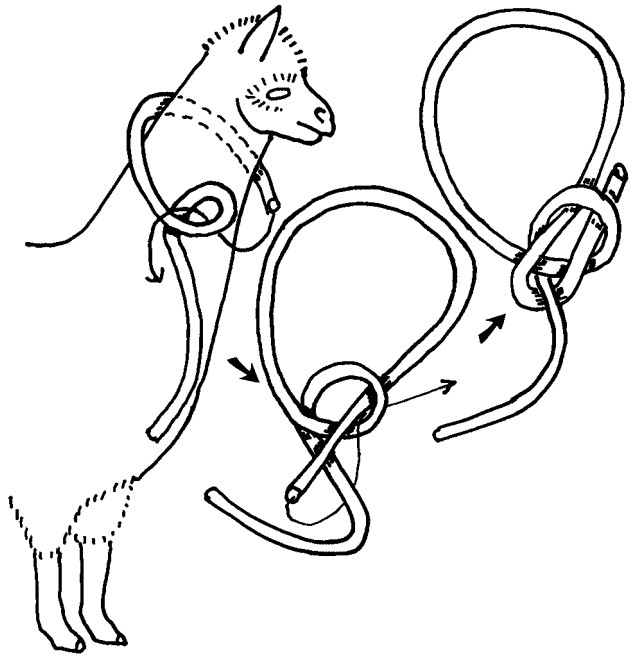

Figure 3.69. Tying a bowline knot.

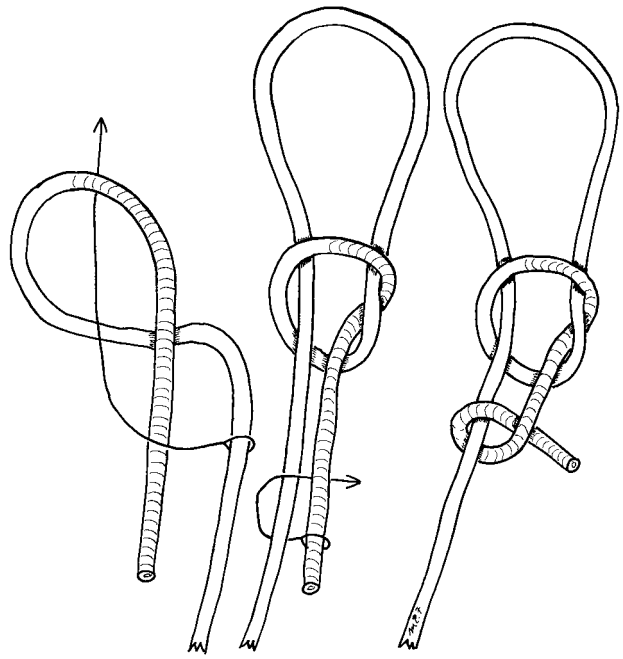

Figure 3.70. Tying a slipknot for attaching a rope to a sheet of material.

Figure 3.71. Using a stone to tie a slipknot to the corner of a towel (the stone is folded in the towel).

knot (Figure 3.70 to 3.72). For more information on knots and rope work, see the references.[9,10]

South American llameros use a llama leather riata (lariat) to capture vicuñas. North American cowboys may attempt to rope a llama or alpaca, but SACs quickly learn to dodge a tossed lariat. They may also stand close to a fence, making it difficult to lay the loop over the head. Even an experienced roper may be unable to catch some llamas. Once a llama or alpaca feels a rope around its neck, it usually submits to handling. It should be mentioned that in a group including an adult male, the male will attempt to mount the captured llama.

Transporting[1,11,18]

Llamas and alpacas in North America are highly mobile and generally travel well. The majority lie down soon after the beginning of a journey and remain recumbent while moving; however, traveling is stressful for most SACs whether or not they exhibit overt signs of stress. It is the responsibility of the transporter to minimize stress, and it is obviously in the best interests of the owner to know which stressors are acting and how problems may be minimized. SACs do not need to be tied within a truck or trailer. At least two fatalities have been associated with transporting tied llamas by trailer. In the first accident, a person

Figure 3.72. Completed slipknot to corner of a towel.

borrowed a llama to use in a parade. The borrower disregarded instructions and tied the llama in the trailer with a lead rope. The llama became entangled in the rope and suffocated.

In the second accident, the llama was tied in a trailer constructed with a horizontal bar across the front, at a height corresponding to the midcervical region on the llama. The llama was tied with a rope over the bar. At the destination, the llama was found dead, partially hanging from the bar. At necropsy, a massive hematoma was located in the pharynx, which obstructed the glottis. Apparently the llama had bumped against the horizontal bar at some point in the journey, rupturing a vessel in the pharynx. As the llama pulled back on the rope and halter, the mouth was forced shut so that blood in the pharynx could not flow out the mouth or nostrils.

Reasons for Transporting

New purchases must be moved to the new owner's premises. Other movements include to and from shows, sales (auction and private), public appearances (schools, parades, rest homes), veterinary care facilities, and outside breedings.

Types of Conveyances

Llamas and alpacas are small enough and adaptable enough to be moved in numerous different types of vehicles including automobiles, private vans, pickup trucks, livestock trucks, trailers (open and closed), and commercial vans. The type used may depend on the age and size of the animal and what is available, but weather must also be considered.

Camels generally require larger vehicles or trailers, but pickup trucks are used in the Middle East.

Pretrip Requirements

Only healthy camelids should be moved, unless the animal is being transported to a veterinary facility. Appropriate documentation should accompany the animal. If a camelid is to be moved across a state line, it is necessary to obtain a veterinary inspection and a health certificate (valid for thirty days). It is the responsibility of the owner (usually in consultation with a veterinarian) to determine what special tests or other documentation may be required at the point of destination. A frequent error is to procrastinate finding out what is required until there is insufficient time to carry out some of the tests or to obtain the permit from the state of destination. Seven to ten days should be allowed to collect samples, send them to the laboratory for analysis, and have a report returned. The author is well aware that even though a state may require a health certificate, there may be no inspection station at the border. Some owners may say, "why bother?" However, the owner may be asked to produce the documents at any time. A driver who is stopped by any law officer, becomes involved in a vehicular accident, or has mechanical difficulties requiring assistance may be cited if appropriate documentation is not available.

A few states require an approved import permit before the animal is allowed into the state. This requires that a health certificate be approved at the state veterinarian's office in the state of origin and forwarded to the state of destination before an import permit may be issued. It may be weeks before such a permit is secured.

Vehicle and trailer licenses must be current as well. If animals are insured, establish whether there is coverage for transportation; if not, explore the possibility of acquiring trip insurance. Llamas and alpacas should be accustomed to the vehicle in which they are to travel. Certainly this should include halter training and leading them into it or a similar conveyance. Even if an animal is not going to be shipped, training should be practiced to prepare for evacuation in the event of a natural disaster.

It is easy to procrastinate routine maintenance of vehicles and trailers, but it is vital to ascertain that exhaust fumes do not flow past the heads of the SACs. Brakes, tires, batteries, and lights should also be checked.

Selecting an Appropriate Time

Most trips are undertaken at the convenience of the people involved, but the needs of the animals should be the major consideration because the animal's life may be at stake. People usually ride in an air-conditioned vehicle. While air-conditioned trailers may be used by people who routinely ship animals,

most animals are moved without the benefit of special cooling or heating. More than one SAC has died as a result of heat stress while being trailered. Climatic conditions must be considered. Most airlines that accept live cargo refuse to carry an animal if daytime temperatures reach above 29.4°C (85°F) anywhere that the plane lands. It is necessary to know weather conditions where stopovers may be made, as well as at the destination. Road conditions should be investigated, including any construction detours that may cause delays. Storms should be anticipated.

Commercial Shippers

Numerous companies advertise capability for moving animals about the country. Some have experience and own appropriate vehicles. However, no license is required for animal transport other than for the vehicle. Owners must rely on the reputation of the shipper. Important questions to ask the shipper include: Is there insurance coverage for the animals? (If there is any doubt, ask to see the policy.) What will be the route of travel? How will layovers and rests be handled? What will be the time of pickup and delivery? What is the size of the vehicle and/or trailer? It may be necessary to move animals to an accessible location if the vehicle is a large semitrailer. Some shippers require that animals be haltered in the event that it becomes necessary to quickly off-load. All shippers require that the animal be trained to halter. Who will supply halters and lead ropes? What type of bedding will be used? What type of feed is to be offered? It is best to provide hay to which the camelid is accustomed.

A written agreement should be obtained on liability, feeding and watering responsibility, whom to call in the event of an emergency, and cost. The owner is responsible for obtaining all required documents, but these must be carried by the driver.

Trailers and Trailering

Trailers used for animal transport vary from home-constructed rigs for carrying one animal to luxurious, air-conditioned multianimal types. Some have built-in living space for people. Using an underpowered or inappropriate vehicle is dangerous. The trailer hitch should be installed by a competent mechanic and be rated for a specific drawbar weight. The hitch should be solidly attached to the frame of the vehicle. Hitches that attach only to the bumper are not appropriate for animal trailers unless they are attached to heavy-duty steel bumpers on pickups and larger trucks. Safety regulations require that the trailer be chained to the vehicle. All running lights, turn signals, and brake lights must be functioning, even for daytime travel.

Trailers may be constructed of steel or aluminum. Obviously, towing weight will differ. A single-axle trailer is not as safe as a double-axle trailer. A blown tire on a single-axle trailer may be fatal. A goosenecked or fifth-wheel trailer provides the most balanced ride for the animal but necessitates a truck that is dedicated to that trailer. Some trailers have a small ramp leading up to the floor of the trailer for loading and unloading. Others require that the animal step up to load. Llamas and alpacas prefer to step up rather than walk up a ramp. It is unwise to pull or push a llama or alpaca into a step-up trailer. A leg may slip beneath the floor of the trailer and be fractured if the animal lunges forward. Appropriate training prior to loading obviates problems of this nature.

Trailer flooring must be considered. Although camelids are surefooted and can generally walk on smooth surfaces, it is not appropriate to leave either a metal or wood floor of a trailer bare. Rubber mats are most desirable because they provide footing, insulation from both heat and cold, and comfort for alpacas who usually lie down as soon as a journey begins. Some owners use indoor/outdoor carpeting. Straw is often used, but though it may provide insulation in cold weather, it does not provide footing. It does absorb urine. It is unwise to use straw for bedding on long journeys, especially if no feed is supplied. Hungry or bored animals may eat the straw and, if unaccustomed to it, may develop digestive upsets. Sand may be used, but sawdust or wood shavings become embedded in the fiber coat and are difficult to remove.

Special precautions must be taken when traveling in subzero weather. When an SAC lies down, its thermal window is in contact with the floor, which is usually the coldest part of the trailer. The floor should be padded with a rubber mat, layers of indoor/outdoor carpeting, or, less desirably, a deep layer of straw.

Avoid tying a camelid when trailering. Accidents have occurred when animals have become entangled in a tied lead rope and strangled, unbeknownst to the driver. If a covered horse trailer is used, the area above the rear doors should be blocked off with netting to preclude the unlikely event of an animal attempting to jump or climb out.

Trailers that are vented for horses may not provide adequate ventilation for SACs. Ventilation is important not only to provide air for breathing but also to provide air movement to dissipate heat.

Shipping by Air

Air shipment by commercial airlines is quick but expensive. Preplanning is a must. Regulations regarding crate size and other shipping conditions are detailed in a handbook of the International Air Transport Association.[1] Not all airlines will carry live cargo, and each airline may impose its own restrictions. Alaskans Janet and Jim Faiks have shared their experiences with air shipping of alpacas.[5]

General Procedures

Sufficient hay and supplements should be carried to sustain the animal through the trip and allow gradual introduction of a new forage. Abrupt diet changes may be upsetting to some individuals. It is usually more difficult to carry sufficient water. SACs may be trained to use different containers than may be used on the farm. Water SACs with plastic or metal buckets for a few days before beginning a trip. Animals may be reluctant to drink sufficient new source water, which in hot weather may be disastrous. The odor and/or taste of new water may be disguised by pretraining the animal to drinking water containing two drops of vanilla extract or oil of eucalyptus.[4] New water can then be similarly treated. Sufficient buckets, blankets, halters, lead ropes, pitchforks, shovels, brooms, and net hay feeders should be carried.

Emergency Supplies

Emergency supplies and equipment should include flashlights, highway flares, a first aid kit, fire extinguishers, a thermometer in an insulated container to monitor body temperature in the event of heat stress, and directories to enable contact with local breeders for assistance.

Dung Pile

Alpacas are more willing to defecate and urinate in a strange environment than are llamas. A periodic opportunity should be provided for them by stopping for a rest so that they may stand or, if off-loading is feasible, to do so. It may be desirable to carry a container of dung so that at a rest stop or at the destination a few pellets may be spread in an appropriate spot and the SAC led to investigate them. Those who transport alpacas in vans for long distances indicate that if an animal has to urinate or defecate it will begin humming and the humming will become more intense the longer the driver disregards the signal.

Many shows now have a communal dung pile near the back gate of the show ring to encourage defecation outside the ring. Owners taking an SAC to a veterinarian for a reproductive examination are advised to give it an opportunity to empty its bladder before the examination begins.

Transporting the Sick or Injured Camelid[11]

An ill llama or alpaca may refuse or be unable to remain in a kushed position. It may roll to its side. If possible, the SAC should lie on its right side to minimize the likelihood of passive regurgitation with subsequent inhalation of stomach contents. The jarring associated with lying on its side in the back of a pickup or in a trailer may be stressful. The animal may be cushioned by using an inflated air mattress or sacks filled with straw. Alternatively, the tires may be partially deflated.

Avoid giving feed and water while traveling. Make certain that the SAC is able to cope with ambient temperature and humidity conditions and keep in mind that an ill animal may not be able to thermoregulate as well as a healthy one.

Be cautious when unloading an ill SAC. A healthy animal may be able to comfortably jump from a pickup bed, but the ailing animal may fall. The SAC may be so weak that it is unable to off-load; a canvas tarp or piece of plywood may be used to lift it from the conveyance.

As stated before, if the ill SAC is able to stand, take it to a grassy area so that it may urinate or defecate. This is particularly important if a rectal examination is to be performed.

Dealing with Fractures

Fractures of the long bones of the lower leg should be immobilized before attempting to transport a camelid to a veterinary facility. Receptionists or veterinary technicians should be taught how to give instructions to owners about dealing with fractures. Splint material may be polyvinyl plastic pipe cut lengthwise, strips of plywood, metal rods, branches from a tree, or rolled up newspapers. No attempt should be made to straighten malaligned bones; they should simply be stabilized in the position found. Padding may be cotton or newspaper. The splints should be bound to the limb with Vetrap, a gauze bandage, an Ace bandage, or strips of cloth torn from a worn-out bedsheet. Alpacas may be forced into the kushed position by chukkering, but this technique should be avoided when dealing with a hind limb fracture. It is difficult to stabilize fractures of the upper limbs except by flexing the lower leg and binding the entire limb to the body.

It may be appropriate to rig a temporary sling inside a trailer or truck using a large bath towel or piece of canvas. Cut two holes near the center on one side approximately 15 to 20 cm (6 to 8 in.) apart. Gently lift a forelimb and insert the foot through one hole and then repeat with the other forelimb. The side closest to the hole should be toward the head. Fasten a small rope to each corner by first folding the corner of the towel over a small rock (2.4 cm, or 1 in.); then tie a slipknot around the cloth and the rock and pull it tight (Figures 3.71, 3.72). The ropes are then tied to the side of the trailer or partition. The feet should remain on the floor of the trailer, and a person should ride beside the animal. A lifting sling may be constructed from four loops (Figure 3.73).

Moving a Recumbent SAC

Moving a recumbent SAC may be accomplished using one of the following techniques. A sheet of

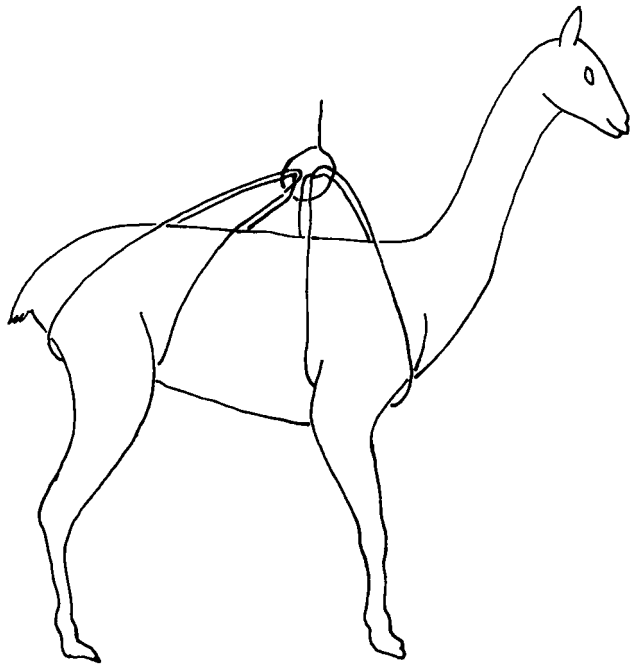

Figure 3.73. A temporary sling constructed of four loops around the legs.

Figure 3.74. Using a piece of plywood as a sled for moving a recumbent llama.

plywood may be used by drilling holes in two of the corners and making loops of rope or wire (Figure 3.74). A tow rope may then be attached to pull the animal to a more convenient location. If sufficient people power is available, the plywood sheet with the animal on it may be lifted into a truck or trailer.

A plastic or canvas tarp that is longer than the length of the animal may be rolled up on one side and placed next to the animal that is moved into the kushed position. Do not roll the animal on its back onto the tarp; rolling may cause regurgitation and possibly inhalation pneumonia. Rather, partially roll the animal to sternal recumbency and push the roll under it.

Figure 3.75. First step in placing a llama on a tarp: roll the animal up to sternal recumbency and place a rolled tarp next to it.

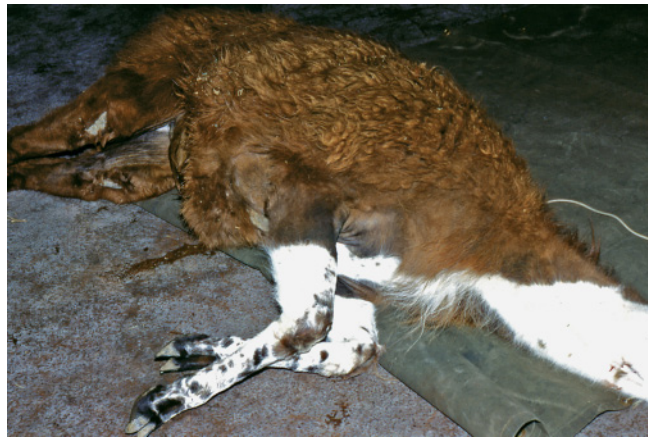

Figure 3.76. Second step: allow the llama to roll back to lateral recumbency.

Allow the animal to resume lateral recumbency so that the tarp may be unrolled beneath the SAC. Then both sides of the tarp may be rolled up to form a handhold for those who will lift and carry the animal. The head and forequarters should be kept higher than the rear quarters to avoid passive regurgitation (Figures 3.75 to 3.78).

The third method is to fashion a travois. The equipment required includes a plastic or canvas tarp and two heavy wooden poles or pipes that are long enough to extend beyond the tarp. Place the poles on the expanded tarp and fold them over each other as if making a human stretcher, as explained in first aid manuals (Figures 3.79, 3.80). Pull the animal onto the travois and lift the end that holds the head and forequarters and drag the travois to the new location.

Alpacas may be lifted in a stretcher by two people. Two people may also physically lift and carry an

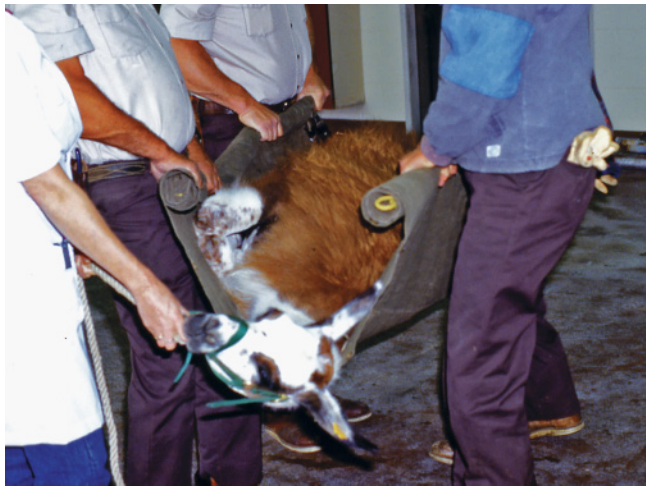

Figure 3.77. Third step: lift the animal and move to the desired location.

Figure 3.78. Construction of a stretcher from a tarp and poles.

Figure 3.79. Llama on a stretcher.

Figure 3.80. Using a stretcher as a travois.

Figure 3.81. Two-person lift of an alpaca.

alpaca by standing on either side of the alpaca and facing each other. Reach arms beneath the animal, with one arm just behind the front limbs and the other just in front of the stifles (Figure 3.81). Some people place the forward locked arms in front of the forelimbs, but this may put pressure on the trachea and is less desirable.

Medical Problems

Medical problems that have occurred while transporting SACs include trauma, choking, colic, infection, inhalation of carbon monoxide fumes, strangulation, and paralysis of a forelimb from lying on one side too long.

How to minimize medical problems:

1. Ship only healthy animals, or if the animal is known to be ill or injured, take appropriate precautions.

Figure 3.82. Camel in a pickup truck.

2. Select appropriate weather for shipping.
3. Maintain the vehicle and/or trailer in optimum operating condition.
4. Carry emergency supplies and equipment.
5. Train SACs to halter, load, unload, and remain quietly in a trailer.
6. Avoid tying SACs in a trailer.
7. Make preparations to prevent heat and cold stress.
8. Avoid feeding except on long hauls and then avoid bedding with straw.
9. Separate variable-sized animals or animals of different hierarchical status.
10. Always separate adult males.
11. Have emergency phone numbers to call.

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4

Clinical Diagnosis: Examination and Procedures

The basic procedures of clinical diagnosis in camelids are similar to those used for livestock, horses, and even small animals, but anatomic differences necessitate some modifications. Unique characteristics and normal data are discussed.

PHYSICAL EXAMINATION

A physical examination is required for obtaining insurance and interstate or international health certificates, performing a pre- or post-purchase examination, conducting a soundness examination, or conducting an examination on a sick animal.[3,6,7] The concepts are the same as for other species of domestic animals. Table 4.1 is a checklist of basic information that should be obtained on every examination. Table 4.2 is a checklist for a physical examination, patterned after the checklist used for evaluation of llamas or alpacas for acceptability for registration, but could be used on any camelid.

Conformation

All species of South American camelids (SACs) are similar in conformation, with differences primarily in size. Camels are larger still and different restraint procedures are required. Veterinarians conducting soundness examinations and evaluations for lameness should have an understanding of the morphology of the animal. See Chapter 24 for details and illustrations of SAC conformation. The high-head carriage is immediately noticeable, and it contributes to the functional balance of these unique animals. In a healthy llama, the neck is held almost vertical, while the neck carriage of an alpaca is at approximately 70 degrees. With weakness or depression, the head is held lower, and in a sick, laterally recumbent animal, the head and neck are usually positioned back over the thorax in what would be considered opisthotonos in other species.

Gaits of Camelids

Camelids have four natural gaits: the walk, pace, trot, and gallop. See Chapter 24 for details.

Body Condition

Every physical examination should include an evaluation of body condition (see Chapter 2).

Tables 1.4 and 1.6 show comparative weights for OWCs and NWCs, respectively. There are marked variations in size among domestic camelids. Male and female SACs vary little, but this is not true of Old World camelids. It is a widely believed myth that North American llamas are 20% to 40% larger than their South American counterparts, but large llamas may be found in Peru, and small or even dwarfed llamas may be seen in the United States. It is wise to know approximate weight for age.[8,11,13]

Body Temperature

The resting body temperature of adult camelids varies depending on the environment (Tables 4.3, 4.4). Normal body temperatures of neonate camelids fluctuate in a wider range because thermoregulatory mechanisms are not yet as sophisticated as those of adults. SACs evolved in harsh, cool climates and are well able to adapt to cold. Extremely hot and humid climates are less well tolerated, and special cooling systems should be incorporated into management programs to help them cope with heat stress (Chapter 9).

During hot summer seasons, some llamas are able to allow body temperatures to elevate and remain at 40°C (104°F). It is disconcerting to see a heavily fleeced llama lying peacefully in full sunshine on a hot summer

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Table 4.1. Checklist of basic data to collect for any examination.

Identity of the individual Date _____
Name _____
Registration number _____, Microchip number _____
Owner's name and address _____
_____
Age _____, Sex _____, Weight _____
Color: Body _____, Head _____, Neck _____,
Legs _____; Distinguishing markings _____
Sire _____, Dam _____

History
How long has the animal lived at the present premises? _____
Previous owner(s) _____
_____
Location of previous farms _____
_____
Type of housing: Barn _____, Pens _____, Pasture _____
Other animals on the farm: Horses _____, Cattle _____, Sheep _____, Goats _____
Other _____
How many other llama/alpacas on the farm? _____
Previous illness in this animal _____
_____

Basic information
Body temperature _____°F, _____°C
Heart rate _____, Respiratory rate _____
Body condition: Thin _____, Obese _____, Normal _____, Body score _____

Temperament
Easily caught: Yes _____, No _____
Easily haltered: Yes _____, No _____

Table 4.2. Checklist for physical examination of llamas and alpacas.

Date _____

Identification
Name _____
Registration number _____, Microchip number _____
Color _____, Age (yrs) _____
Sex: Male _____, Female _____, Neutered _____

Movement
Normal (Y/N)
Free-moving gait _____, Stiff gait _____, Lameness _____
Leads easily: Yes _____, No _____

Front limbs
Normal (Y/N)
Front view: Base wide _____, Base narrow _____; Carpal valgus: Slight (<5°) _____,
Moderate (<10°) _____, Severe (>15°) _____, Bowed out at carpus _____, Splay feet _____,
Pigeon-toed _____, Polydactyly _____, Syndactyly _____
Side view: Camped forward _____, Camped behind _____
Angulation: OK _____, Too straight _____, Too flexed _____, Buck-kneed _____,
Calf-kneed: Slight (<175°) _____, Moderate (170–175°) _____, Severe (<165°) _____
Cocked ankle (pastern angle >90°) _____, Down in fetlock (pastern angle <30°) _____
Comments

Rear limbs
Normal (Y/N)
Rear view: Base wide _____, Base narrow _____
Cow-hocked: Slight (<5° rotation) _____, Moderate (5–10°) _____, Severe (>10°) _____
Bowed out at hock _____, Splay footed _____, Pigeon-toed _____

Table 4.2. ***Continued***

Side view: Camped forward _____, Camped rearward _____
Angulation: Post-legged _____, Too much flexion _____
Sickle-hocked: Slight (hock angle <135°) _____, Moderate (hock angle <130°) _____, Severe (hock angle <125°) _____
Cocked ankle (pastern angle >90°) _____, Down in fetlock (pastern angle <30°) _____
Comments

Head
Normal (Y/N)
Wry face: Slight (<2°) _____, Moderate (2–5°) _____, Severe (>5°) _____
Nostrils: Air movement both nostrils _____
Ears: Normal _____, Long _____, Short _____, Gopher _____, Frostbitten _____, Curled (fused) _____
Eyes: Entropion _____, Ectropion _____, Laceration _____, Tearing (evidence of blocked tear duct) _____,
Corneal opacity _____, Cataract _____, Dilated pupil _____, Constricted pupil _____, Evidence of blindness _____
Teeth: Superior brachygnathism (undershot jaw): <0.5 cm _____, >0.5 cm _____
Inferior brachygnathism (parrot mouth, overshot jaw): <0.5 cm _____, >0.5 cm _____
Retained deciduous incisors _____, Permanent canine teeth erupted _____
Comments

Neck and Body
Normal (Y/N)
Throat latch: Swelling _____
Cervical spine: Symmetrical _____, Scoliosis _____
Thoracic and lumbar spine: Scoliosis _____, Lordosis _____, Kyphosis _____
Tail: Twisted tail _____, No tail _____
Height, alpacas: Juvenile <74 cm (29 in.) _____, Adult <86 cm (34 in.) _____, Adult >102 cm (40 in.) _____
Weight, alpacas: Juvenile: >48 kg (106 lb) _____, <48 kg _____
Alpaca adults (central incisor erupted): Must be above 48 kg (106 lb) sheared or 50 kg (110 lb) unshorn _____
No height or weight standards for llamas
Comments

Reproductive system
Normal (Y/N)
Male
Testicles: Both testicles in scrotum _____, Cryptorchid _____
Size (length): **llama** <3.4 cm (1 yr) _____, <4.2 cm (2.5 yr) _____
alpaca: <1.8 cm (1 yr) _____, <2.4 cm (2 yr) _____, <3.0 cm (adult) _____
Consistency: Hard _____, Too soft _____
Scrotal edema _____
Female
Position of vulva: Vulvar slit <0.6 cm _____, Vertical _____, Horizontal _____
Clitoris enlarged (evidence of intersex) _____
Comments

Pulmonary system
Normal (Y/N)
Respiratory rate: _____ bpm; Abnormal lung sounds _____
Exercise tolerance _____; Upper airway sounds _____
Comments

Cardiovascular system
Normal (Y/N)
Heart: Murmur _____, Arrhythmia _____, Heart beat rate _____ bpm
Comments

Miscellaneous characteristics
Body temperature: _____°F, _____°C
Teats: Normal (4) _____, <4 _____, >4 _____
Hernias: Umbilical (>1 cm) _____, Scrotal _____
Toenails: Normal _____, Elongated _____, Curled _____, Abnormal horn _____
Other defects: _____
_____

day. However, it should be remembered that fleece insulates from heat as well as from cold.

The llama that is forced to remain recumbent because of trauma or disease is more subject to heat stress, even in neutral environments, because the underside is a fleece-free area of the body and the site for heat dissipation. If the llama is continually recumbent, neither normal nor fever-induced body heat can be dissipated.

The dromedary is able to endure diurnal fluctuations of body temperature from 36.5°C to 42°C (97.7°F to 107.6°F). The body acts as a heat sink during the heat of the day, thus conserving vital water that would otherwise be lost through evaporative cooling. During the cool night, body heat is dissipated by conduction and radiation. It should be obvious that evaluation of a fevered state in the dromedary is difficult. The vital signs for camelids are listed in Table 4.4.

Cardiac Assessment

The heart rate of camelids are listed in Table 4.4. Pulse evaluation is not used in camelids, because there are no readily accessible arteries. The heart is accessible for auscultation as in other mammals. By reaching under the fleece at the elbow, the stethoscope may be placed on a fleece-free area caudal to the triceps, which allows for both cardiac and thoracic auscultation.

A number of congenital cardiac anomalies may persist into adulthood (Chapter 22). A thorough assessment of the heart should be made in every physical examination. Sinus arrhythmia is a common finding, and a llama or alpaca should not be faulted for such arrhythmia.

Table 4.3. Conversion of degrees Celcius to degrees Fahrenheit.

C	*F*	*C*	*F*	*C*	*F*	*C*	*F*
25.0	77.0	36.4	97.5	39.0	102.2	41.6	106.9
26.0	78.8	36.6	97.9	39.2	102.6	41.8	107.2
27.0	80.6	36.8	98.2	39.4	102.9	42.0	107.6
28.0	82.4	37.0	98.6	39.6	103.3	43.0	109.4
29.0	84.2	37.2	98.9	39.8	103.6	44.0	111.2
30.0	86.0	37.4	99.3	40.0	104.0	45.0	113.0
31.0	87.8	37.6	99.7	40.2	104.4	46.0	114.8
32.0	89.6	37.8	100.0	40.4	104.7	47.0	116.8
33.0	91.4	38.0	100.4	40.6	105.1	48.0	118.4
34.0	93.2	38.2	100.8	40.8	105.4	49.0	120.2
35.0	95.0	38.4	101.1	41.0	105.8	50.0	122.0
36.0	96.8	38.6	101.5	41.2	106.2		
36.2	97.2	38.8	101.8	41.4	106.5		

Thoracic Cavity Assessment

The resting respiratory rate of camelids is listed in Table 4.4. The thoracic area of SACs is bound by the caudal border of the triceps muscle, a line extending caudally for 20 to 25 cm (8 to 10 in.) below the top of the ribs and triangled back to the olecranon. The line of diaphragmatic reflection is more caudal than this area, but lung sounds are not heard in the caudal thorax. The auscultation area for a camel is similar, but larger than in SACs.

Normal lung sounds are muted in camelids and may be difficult to hear. With excitement and more rapid breathing, the sounds vesicular rather than bronchiolar. The respiratory rate is best established by placing the stethoscope over the trachea at the thoracic inlet.

Abdominal Auscultation

The major fermentative process of camelid digestion takes place in compartment one (C-1) of the stomach, which occupies the entire left side of the abdomen. Gastrointestinal sounds are primarily associated with gas/liquid churning; therefore, these sounds are usually heard only on the left side.

Gastric motility in camelids differs from that in the ruminant (Chapter 13). Palpation is usually not possible, and a stethoscope is required to hear the subdued sounds. Normal gastric motility rate is three to four per minute, which increases slightly after feeding. A fleece-free abdominal area is located just cranial to the

Table 4.4. Vital signs of camelids.

Parameter	*New World camelids*	*Old World camelids*
Body temperature, adults in moderate environment (°C/°F)	37.5–38.9/99.5–102	36–37/96.8–98.6, morning; 38/100.4, afternoon
Body temperature, adults in hot summer afternoon (°C/°F)	Up to 40/104	36.5–38/97.7–100.4, morning; 39.5–40.5/103.1–104.9, afternoon; 42/107.6 maximum
Body temperature, crias/calves	0.5–1°C higher than adults	0.5–1°C higher than adults
Heart rate, beats per minute	60–90	32–36, relaxed; 44, nervous
Respiratory rate, breaths per minute	10–30	5–8, cool weather; 10–12, summer
Feces	Pellated, use dung pile	Pelleted, void at random
Urine	Clear, pale yellow to amber	Clear, pale yellow to amber, dark brown and syrupy when dehydrated

thigh muscles of the hind limb. To expose the area for auscultation, it is necessary to reach under the fleece in what would be the flank area in other species and lift it up.

Eye

One of the more attractive features of the camelid is the large, expressive eye with long eyelashes. The iris of SACs has a large corpus nigrum (granula iridica) on the dorsal aspect and a smaller one on the ventral, the function of which is unknown. Unilateral or bilateral nonpigmentation of the iris (blue eye, glass eye, wall eye, or watch eye) is seen. The retina is characterized by a pronounced vascular pattern and no fovea. There is no tapetum or eyeshine. See Chapter 19 for more details on eye anatomy, function, and abnormalities.

The nasolacrimal duct originates approximately 5 to 7 mm from the medial canthus of both the upper and lower lids. The duct terminates within the nares on the cutaneous side of the mucocutaneous border, a centimeter or two dorsal to the floor of the ventral meatus on the lateral wall over the ridge formed by the premaxilla. The orifice is 2 to 3 mm in diameter.

A slight ectropion of the lower eyelid may be seen on some individuals when they are excited, frightened, or agitated. This condition should not be confused with anatomic ectropion, which may be either congenital or acquired.

Ear

The ears of llamas are long, variably shaped pinnae that are used to express mood (see Figure 3.1 in Chapter 3). Alpacas have shorter spear-shaped ears. Close inspection of the pinna and the external ear canal is difficult, because llamas and alpacas resent having their ears touched. Camels have even shorter ears but on close inspection they too are indicative of the mood of the camel.

The external ear canal is small (3 to 5 mm in diameter), and a bend along the course of the canal precludes visualization of the tympanic membrane in many adults (Chapter 19). Foxtails (*Hordeum* spp.) have become lodged in the canal and in one case penetrated the tympanic membrane, middle ear, and temporal bone to lodge in the base of the brain. Facial paralysis is usually seen with ear infections. Spinose ear ticks (*Otobius megnini*) and other species of ticks have been seen in the ear.

Lacerations of the ear are frequently observed when multiple males with intact canines are housed together. Frostbite, with variable shortening of the ear, has been seen, but so also has congenital absence of the pinna.

Mouth

SACs are unable to open the mouth wide enough to make possible a more than cursory examination of the oral cavity. The incisor and canine teeth can be viewed and should be checked in every physical examination, especially for over- or underbite. Congenital malocclusion is prevalent in SACs (Chapter 22). The tongue rarely protrudes from the mouth. Nonpigmented gingiva may be used to determine a capillary refill time. The mouth of the camel opens more widely than does the SAC mouth.

DIAGNOSTIC PROCEDURES

Blood Collection

Blood analysis is vital to differential diagnosis of many diseases. Venipuncture and collection of blood is not as simple a task in SACs as it is in most other domestic animals. SACs have evolved a number of protective mechanisms to prevent exsanguination from bites by males fighting with one another. In all locations of the neck, extreme caution must be exercised to prevent accidental cannulation of the carotid artery when taking blood samples.

Two major sites are suitable for jugular venipuncture: low on the neck near the thoracic inlet and high, near the ramus of the mandible (Figure 4.1). The site selected may depend on restraint facilities, assistance available, desires of the client, and experience of the operator. Both locations have advantages and disadvantages.[1]

It is necessary to have an understanding of the anatomy of the vessels of the cervical region.[3] The

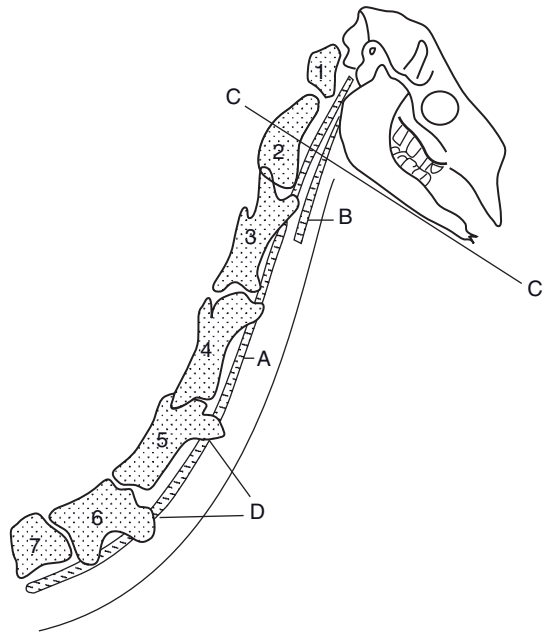


Figure 4.1. Sites for blood collection in a camelid. (A) Jugular vein, (B) tendon of the sternotrachealis muscle, (C) site for upper collection, (D) sites for lower collection.

jugular vein is formed by the confluence of the lingual, facial, and maxillary veins, approximately 1 cm caudad to the ramus of the mandible. Superficially, the vein is covered by the skin and platysma muscle. This junction and the first few centimeters of the jugular vein are embedded within the ventral border of the parotid salivary gland. The jugular vein will, at this point, be on the lateral surface of the sternomandibularis muscle tendon as it inserts on the mandible. The vein then courses slightly dorsal around the tendon to go deeper into the neck, medial to the sternomandibularis muscle (Figures 4.2, 4.3).

The jugular vein lies superficial to the omohyoideus muscle, which separates the vein from the carotid artery. The omohyoideus muscle and its extended fascia form the deep border of the jugular furrow in horses and cattle, which extends throughout most of the cervical region. In camelids, the muscle lies in a different position (Figure 4.4). It is narrow (10 cm) and serves as a separation between the artery and the vein for a distance of only 14 cm caudal to the ramus of the mandible.

The jugular vein continues on toward the thoracic inlet, coursing deep to the sterno-mandibularis muscle, and is incorporated in the same fascial sheath with the carotid artery and the vagosympathetic nerve. All of these structures lie on the ventrolateral surface of the trachea. There is no jugular furrow in camelids.

The vessels are further protected from accidental laceration by the ventral projection of the transverse process of the cervical vertebrae, which forms an inverted semi-U-shaped channel (Figure 4.5). The jugular vein lies just medial to this projection. The ventral projections on the sixth cervical vertebra are prominent and easily palpated, serving as landmarks for venipuncture low on the neck. In the male, the thickness of the skin of the neck varies from 1 cm near the mandible to 0.5 cm near the thoracic inlet.

In long-necked animals such as horses, giraffes, and camelids, valves in the jugular veins prevent backflow of blood to the head when it is lowered for feeding and drinking. In the llama, four or five of these valves are distributed from the confluence of the veins that form the jugular to the thoracic inlet. The valves may be bicuspid or tricuspid.

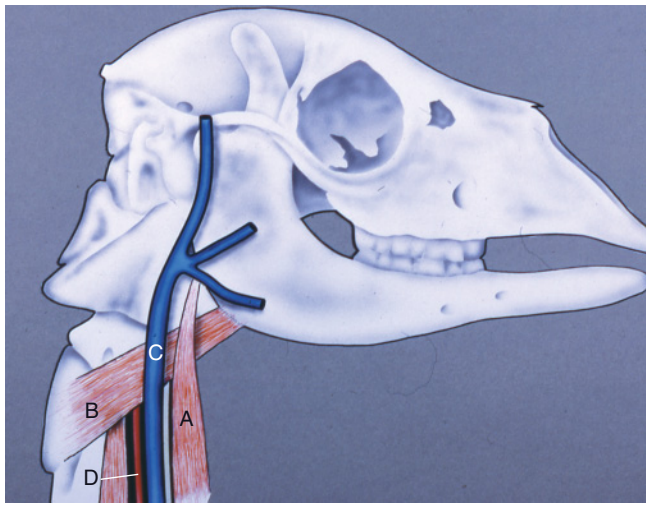


Figure 4.2. Diagram of the anatomy of the jugular vein. (A) Sternomandibularis muscle, (B) omohyoideus muscle, (C) external jugular vein, (D) common carotid artery.

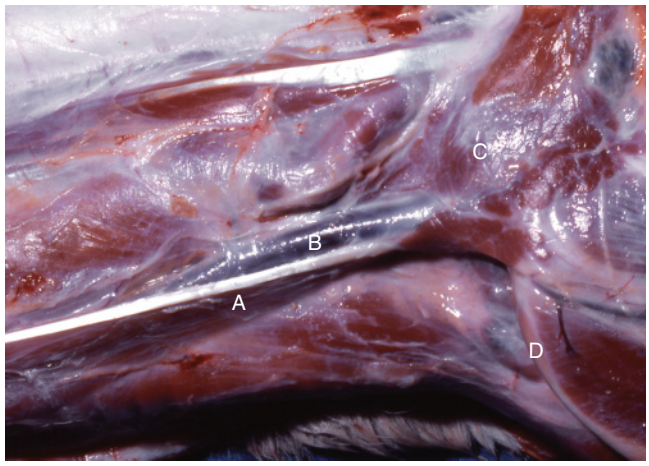


Figure 4.3. Anatomic dissection of the jugular vein. (A) Tendon of the sternomandibularis muscle, (B) jugular vein, (C) parotid gland, (D) mandible.

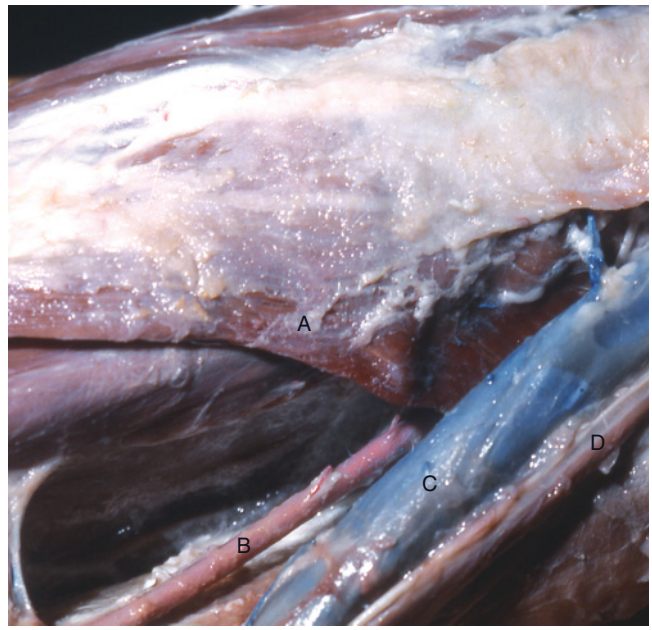


Figure 4.4. Anatomic dissection of the jugular vein. (A) Omohyoideus muscle, (B) common carotid artery, (C) jugular vein, (D) tendon of the sternomandibularis muscle.

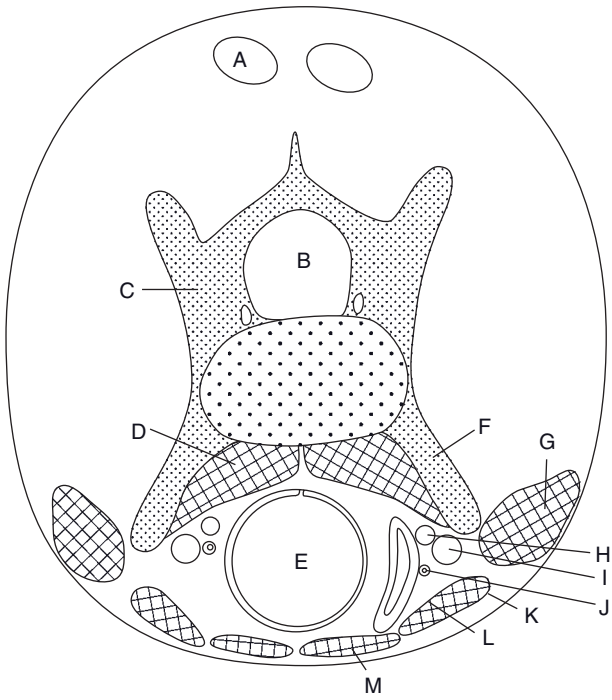


Figure 4.5. Cross-sectional diagram of the camelid cervical region at the level of the sixth vertebra. (A) Ligamentum nuchae, (B) vertebral canal, (C) vertebra, (D) muscle, (E) trachea, (F) ventral extension of the transverse process of the vertebra, (G) brachiocephalicus muscle, (H) carotid artery, (I) jugular vein, (J) vagosympathetic trunk, (K) sternomandibularis muscle, (L) esophagus, (M) sternothyrohyoideus muscle.

A set of valves is situated just caudad to the major veins that form the jugular, approximately 1 cm from the angle of the mandible. Another valve is located 5 cm caudal to the first, at the level of the thyroid vein (Figure 4.6). This valve may interfere with venipuncture when the upper site is selected. The remaining valves are spaced 15 to 20 cm (6 to 8 in.) apart along the jugular vein (Figure 4.7). One of the valves may be located near the ventral process of the sixth cervical vertebra (the site for a low venipuncture).

High-neck Venipuncture

The advantage of high-neck venipuncture is that the jugular vein is more superficial at this location and is separated from the carotid artery; thus, there is less likelihood of arterial penetration.

The disadvantage is that the skin is thickest at this location, and visualization is not possible in adults, making it necessary to rely on landmarks and ballottement of the vein for venipuncture.

It is neither necessary nor desirable to clip a patch of fiber for collecting a blood sample from a camelid. It may take a year to eighteen months for the fiber to grow back, and owners are not happy to have a bare patch on the side of the neck. An imaginary line should be scribed along the ventral border of the mandible onto the neck, with the head held in a slightly flexed position. The tendon of the sternomandibularis muscle should be palpated to locate the site of penetration, just dorsal to the intersection of those two lines. The vein should be occluded by deep, firm pressure at the ventrum of the vertebrae. By stroking the area of the vein toward the occluding hand, a wave may be felt

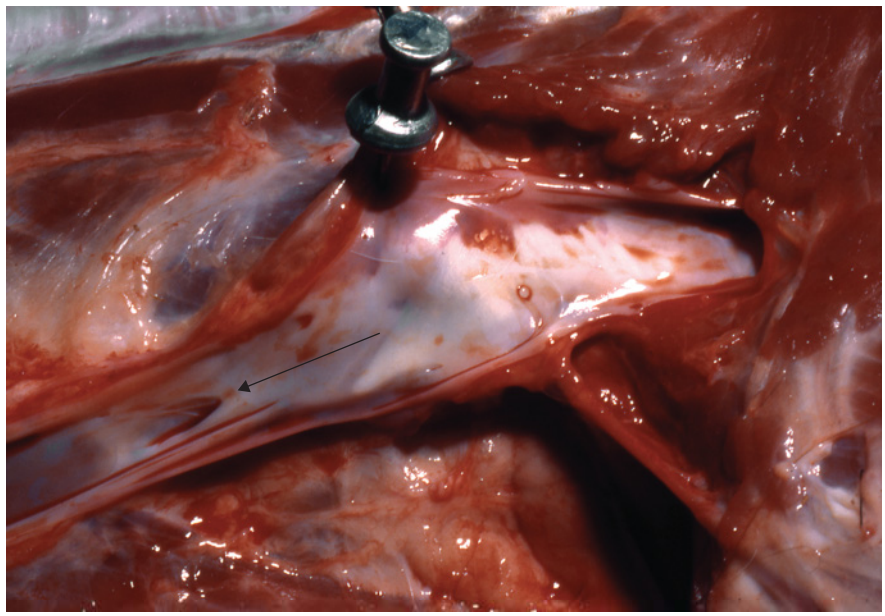


Figure 4.6. Valves in the jugular vein in the upper neck.

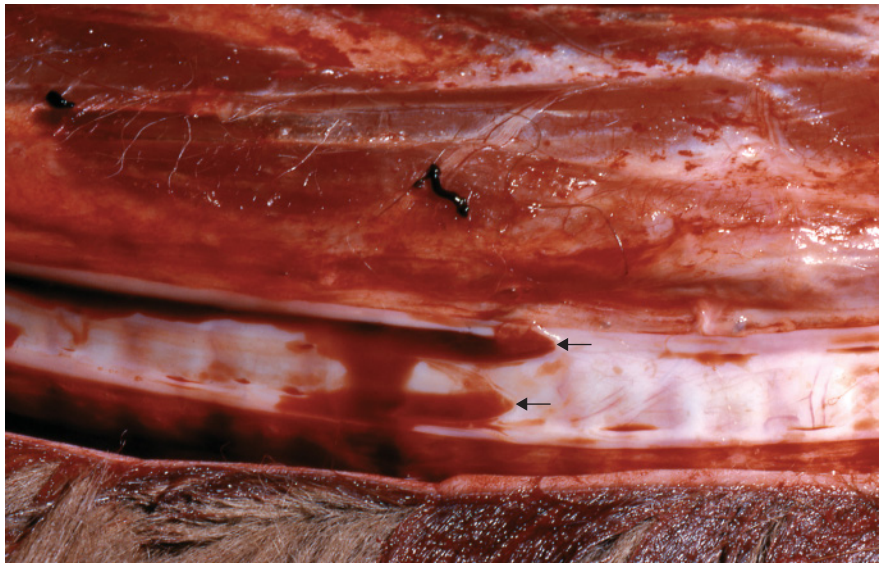


Figure 4.7. Valves in the jugular vein low on the neck.

against the fingers. Alternatively, correct selection of the site may be tested by placing a finger over the suspected location of the vein and releasing the occlusion to see if the dilated vessel empties.

Positioning of the head and neck is important to achieve success with this procedure. The author prefers to perform venipuncture from the right side. An assistant should stand on the left to grasp the right ear while pressing his body against the left neck to produce a slight bow, with the convex side toward the operator. The nose should also be pulled toward the left, making certain that the halter straps do not press on the vessels proximal to the penetration site.

Low-neck Venipuncture

The advantage to low-neck venipuncture is that the skin is thinner, and movement of the head is less disruptive.

The disadvantage is that the lower neck is usually more heavily fleeced than the upper. Because the jugular vein and carotid artery are in juxtaposition at this location, there is a greater chance of arterial cannulation. Some type of stock or chute is desirable to fix the head upward to prevent the llama from pushing forward.

The head should be elevated. The operator should palpate for the ventral projection of the transverse process of the sixth cervical vertebra and occlude the vessel at this site by wrapping the fingers around the projection. The pulsation of the carotid artery may be felt. The needle should be inserted slightly medial to the projection, toward the center of the neck.

Restraint for venipuncture in a neonate up to four months of age may be difficult. Fortunately, the skin of the neonate is thin, and the jugular vein can usually

Figure 4.8. Position for venipuncture in a cria.

be distended and visualized. Select a clean, dry location and place a clean towel beneath a newborn to protect the umbilicus. A simple method that enables a single person to perform venipuncture begins with straddling the neonate and folding the front legs with the hand as the cria is pushed down (Figures 4.8, 4.9). The head should be pushed down with the other hand. As the cria lies down on the forelegs, a slight pressure on the rump encourages it to assume sternal recumbency. The cria should never be collapsed by sitting on it.

Alternatively, it may be lifted off its feet and tipped to lie on its side. Then the legs will fold up beneath it

Figure 4.9. Alternate position for venipuncture in a cria.

as the handler straddles it. While kneeling over the cria to keep it in sternal recumbency, the left arm should be placed on the right side of the neck to push the head and neck to the left. The left hand should also be used to occlude the vessel while the right hand inserts the needle to collect blood or give medication. Venipuncture may be accomplished at either the low or high position.

Yearlings are often more difficult to restrain for venipuncture than neonates or adults. Many are not accustomed to being tied or having their heads restrained. They frequently rear up and struggle vigorously. Chutes and stocks are not usually designed for animals smaller than adults. Nonetheless, the basic principles apply.

Venipuncture may be performed at other locations as well. The saphenous vein is superficial and easily palpated on the medial aspect of the stifle in a recumbent animal. The artery and vein lie contiguous to each other, with the vein more craniad.

Small volumes of blood may also be collected from the middle coccygeal vein on the ventrum of the tail. In the camelid, the coccygeal vein is superficial, lying just beneath the skin. The tail should be elevated, but pushing it too hard up over the back should be avoided, because this will occlude the venous return. The procedure for blood collection from the tail of a camelid is easier than it is in the bovine, in which the vein is situated deep, near the body of the coccygeal vertebrae. Insert the needle through the skin, then establish negative pressure in the syringe, and continue the insertion until blood flows into the syringe. It is unwise to administer medication in the coccygeal vein because

Figure 4.10. Ear veins on a llama.

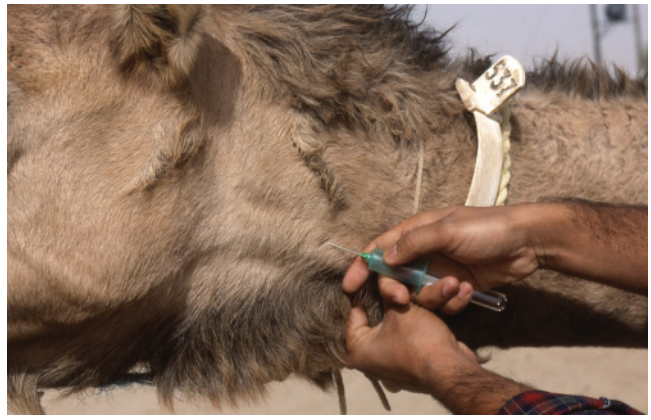


Figure 4.11. Blood collection from the jugular vein of a camel.

the long bevel on most needles may span the vessel and allow perivascular deposition of the medication.

There are also accessible veins on the ear (Figure 4.10). Llama owners may nick the caudal border of the ear to collect small samples for progesterone analysis. A vein can also be cannulated with a 23-gauge needle attached to a pediatric catheter. The largest vein is located on the outer caudal border of the pinna. A temporary tourniquet (heavy elastic band) may be placed at the base of the ear to raise the vein.

A brachial vein is present on the cranial forearm just as in a dog or cat. This vein may be useful if the fiber covering is not too dense. The medial saphenous vein is accessible on the medial aspect of the stifle in a laterally recumbent camelid.

Jugular venipuncture in the dromedary camel is easier than in SACs if the head is restrained and side-to-side movement of the camel is controlled (stock or chute). The jugular vein may be distended using digital pressure as in the SAC (Figure 4.11), or a rolled up towel may be placed on the ventrum of the neck, near the thoracic inlet, and held tightly with a cord or rope.

Figure 4.12. Lateral thoracic vein of a camel, an alternate site.

The towel serves as a temporary tourniquet. The distended jugular vein is visible in the upper cervical region, and the distention progresses on to the facial vein on the lateral aspect of the mandible. Venipuncture may be performed at any location on the distended vein. During the winter months, the fiber of dromedaries increases in length and may diminish the visibility of the vein. The vein is easily palpated. Bactrian camels often have heavy neck fiber like the dromedary in cold weather. Venipuncture is performed the same in Bactrians as in dromedaries. The lateral thoracic vein is also accessible in camels, but the animal must be restrained to avoid kicking (Figure 4.12).

Hemogram and Blood Chemistry

Evaluation of hemogram and blood chemistry data used in conjunction with clinical examination is crucial to making a diagnosis in many disease conditions.[2,4,10,12] A full discussion of normal and abnormal clinical pathology findings is found in Chapter 15.

Evaluation of Blood Loss

Acute blood loss from laceration of major vessels is potentially life threatening because of the development of hypotension and inability of the decreased blood supply to carry sufficient oxygen to the brain and other vital organs. Chronic blood loss leads to anemia (Chapter 15). The blood volume of SACs has been calculated to be 6.5% to 8.6% of the body weight in an animal with normal body condition. Presuming that camelids are similar to other animals, they may lose up to 25% of their blood volume without succumbing to blood loss. A hemogram may not help in establishing acute blood loss because both cells and plasma are lost and the ratio of the remaining blood remains the same until compensatory fluid is drawn from tissue and other fluid sources. Many days are required for cellular replacement. Table 4.5 provides an overview of blood volume and dangerous blood loss.

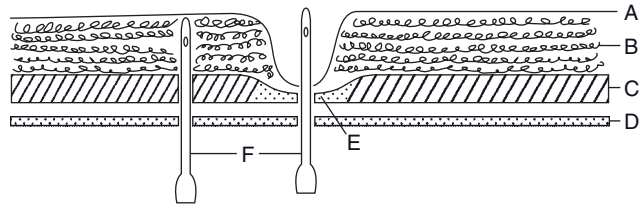


Figure 4.13. Diagram of the abdominocentesis location. (A) Peritoneum, (B) abdominal fat, (C) abdominal muscles, (D) skin, (E) linea alba, (F) teat cannula.

Table 4.5. Nonlethal hemorrhaging.

Body weight (lb/kg)	*Blood volume 8.6% body weight[a] (L/qt)*	*Nonlethal blood loss (25% blood volume) (L/qt)*
30/13.6	1.17	0.29/0.61
50/23.0	1.98	0.50/1.06
60/27.6	2.37	0.59/1.25
90/41.0	3.53	0.88/1.86
100/45.0	3.87	0.97/2.05
120/54.4	4.69	1.17/2.47
200/90.0	7.74	1.94/4.10
330/150.0	12.90	3.23/6.82

[a] Body weight of a normal conditioned animal.

Abdominocentesis

An evaluation of peritoneal fluid is a vital component of differential diagnoses of digestive and other abdominal disorders. Peritoneal fluid aspirate may be obtained from the ventral midline, just caudal to the umbilicus. The retroperitoneal fat layer of SACs may be as much as 6 cm (3 in.) thick and 14 cm (6 in.) broad lying on either side of the linea alba (Figure 4.13). Only on the precise midline is it possible to penetrate the peritoneal cavity with certainty. The sample may be obtained from either the standing or recumbent llama. The site should be clipped and surgically prepared and local anesthesia injected. A stab incision should be made with a number 12 scalpel blade for insertion of a 6-cm (3-in.), 14-gauge teat cannula with a quick thrust.

In adult camelids, the glandular saccule area of C-1, covered by the greater omentum, is located on the ventral midline craniad to the umbilicus. Because the omentum may occlude the ports of a teat cannula, it is recommended that abdominocentesis be performed caudal to the umbilicus.

An alternative site for collecting peritoneal fluid in SACs is a paracostal approach. The site is located caudal to the last rib and approximately one-third to

one-half of the distance from the ventral midline to the dorsal midline. The site of penetration in alpacas is 1 cm dorsal and 3 cm caudal to the costochondral junction of the last rib, and in llamas it is 2 cm dorsal and 5 cm caudal to the costochondral junction of the last rib.[3]

In a normal animal, insufficient abdominal fluid may preclude collection. Unless there is 150 ml or more of fluid in the cavity, it will not flow freely. If it does not, attaching a syringe and establishing slight negative pressure while repositioning the needle tip in the cavity may yield a sample. If the fluid accumulation is localized, successful aspiration will be fortuitous.

The technique for collection of peritoneal fluid from adult camels is described as follows: The site is 10 cm (4 in.) caudal to the xyphoid cartilage and 10 cm (4 in.) left of the midline.

Orogastric Intubation

Gastric intubation is accomplished via the oral cavity. The nasal cavity is narrow and precludes passage of any except small tubes. See Chapter 12 for description of the nasal cavity. Passage of the orogastric tube differs little from the procedure in cattle and sheep. The cheek teeth are sharp, so a speculum is necessary to guide the gastric tube through the oral cavity and into the oropharynx. A standard cattle Frick speculum is suitable for camels but too large except for the largest llama. A 20- to 25-cm (8- to 10-in.) segment of rubber garden hose slightly larger in diameter than the orogastric tube makes an excellent guide. A similar length of polyvinyl chloride pipe, wrapped with adhesive tape to prevent accidental shattering, is also suitable. Neonates may be intubated while in sternal recumbency (Figure 4.14). The appropriate size for a stomach tube is listed in Table 4.6.

The head should be secured with a halter that allows opening the mouth sufficiently to insert the speculum over the base of the tongue. The head should be slightly flexed. Once the tip of the tube is in the oral pharynx, gentle pressure should be exerted to stimulate the animal to swallow. Indiscriminate jabbing should be avoided, but gently rotating the tube or adjusting the position of the head will encourage the llama to swallow the tube. There should be a slight resistance to passage of the tube.

The tube should be palpated as it traverses the left ventral cervical region to ensure intubation of the esophagus rather than the trachea. Alternatively, the free end of the tube may be placed into a container of water to check for bubbles. The tube may be inserted into the lumen of C-1. However, if it is desired to bypass deposition of fluid or medication into C-1, the tube should be kept within the esophagus. This is particularly important when feeding orphan neonates via stomach intubation. Milk deposited in C-1 may remain and ferment rather than be digested in C-3.

Gastric intubation may stimulate regurgitation (Figure 4.15). This is not serious, but it does make the process disagreeable and may necessitate starting over again.

Urinary System

The easiest way to collect a urine sample is to make a free catch while the animal is urinating. Camelids use a communal dung heap for both urination and defecation. Both the male and female eject urine caudally from a partial squat position. Unless extremely wild, most llamas will permit an approach to catch the urine in an open cup, or the cup may be attached to a stick approximately 1.2 m (4 ft) long. Complete urination requires thirty to sixty seconds, allowing ample time to obtain a sample.

A free catch is the only available method of obtaining a urine sample in an adult male because a recess,

Figure 4.14. Positioning for gastric intubation of a neonate llama.

Table 4.6. Orogastric tube sizes for llamas and alpacas.

Body weight	*French gauge*	*Outside diameter (mm)*	*Outside diameter (in.)*
4–8 kg (8–18 lb)	18–22	6–7 (Stallion catheter or feeding tubes)	1/4
9–20 kg (19–44 lb)	24–30	8–10	1/3 to 3/8
30–90 kg (66–198 lb)	30–40	12–13 (Equine stomach tubes)	1/2
100–200 kg (220–440 lb)	40–45	13–16 (Equine stomach tubes)	1/2 to 5/8

located dorsal to the urethra at the ischial arch, makes catheterization virtually impossible. It would be most difficult to prevent insertion of the catheter into the diverticulum, even if digital pressure were applied through the wall of the rectum to aid direction of the catheter into the pelvic urethra.

It is possible to catheterize the female. The external urethral orifice, a groove, is easily palpated on the floor of the vulva at the region of the hymen. However, a suburethral diverticulum in the female may also complicate catheterization (Figure 4.16). After the vulva has been cleansed, a sterile gloved finger should be inserted into the vulva to palpate the orifice. Withdrawing the finger slightly and inserting the catheter dorsal to the finger aids in avoiding penetration into the more ventral blind diverticulum. A 1.66 (number 5 French) polypropylene catheter is suitable for urine collection. The bladder should be found at a depth of 25 cm from the lips of the vulva. For urinalysis data see Chapter 18.

Cystocentesis has been performed on SAC neonates and juveniles. The bladder is situated intrapelvically on the floor of the pelvis. The site for penetration of the abdomen is just cranial and slightly lateral to the pelvic brim. Clip a spot of fiber and disinfect the area before inserting the needle. The procedure may be performed on a standing animal or one in lateral recumbency.

Figure 4.15. Regurgitation accompanying gastric intubation.

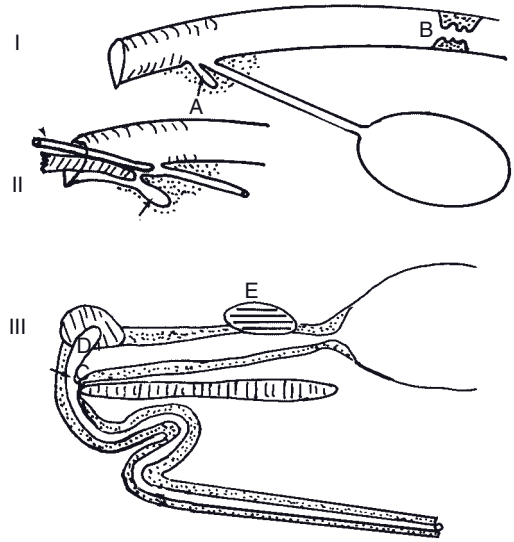
Miscellaneous Procedures

Enema

An enema is indicated in cases of constipation, heat stress, or dehydration. In the juvenile, a feeding tube may be used attached to a funnel for the fluid (Figure 4.17), or with a syringe (Figure 4.18). Adults may be given an enema with an orogastric tube and pump or a garden hose. It is not necessary to insert the tip of the hose into the rectum. The tip is pressed against the anus and held with a hand to allow water to run in and out as the stimulus to evacuate occurs (Figures 4.19 to 4.22.)

Thoracocentesis

Thoracocentesis may be indicated to obtain samples of pleural fluid for diagnostic purposes, remove



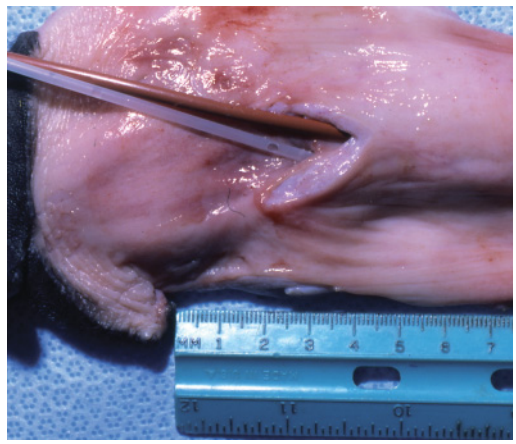


Figure 4.16. (Left) Diagrams of the urethra. I. Schematic of the female urethra. (A) Suburethral diverticulum, (B) cervix. II. Covering the suburethral diverticulum to catheterize the female. III. Schematic of the male urethra. (C) Urethral recess, (D) prostate gland, (E) pubic bones.

Figure 4.17. Giving an enema to a juvenile with a tube and funnel.

Figure 4.18. Giving an enema to a juvenile with a tube and syringe.

Figure 4.19. Using a garden hose to give an enema to an adult, light flow of water.

Figure 4.20. Hose to the anus but not inserted.

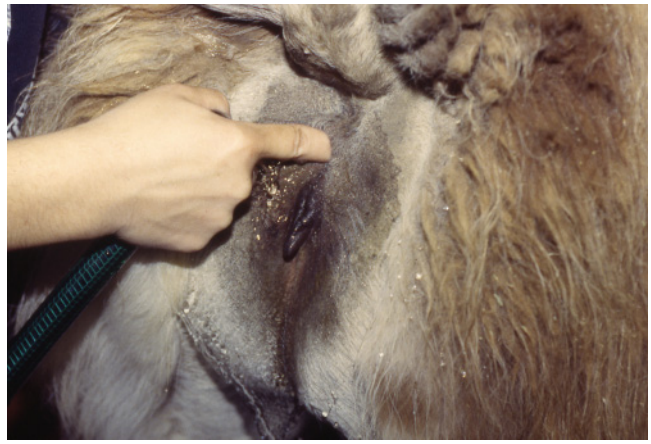


Figure 4.21. Using a finger to open the anus.

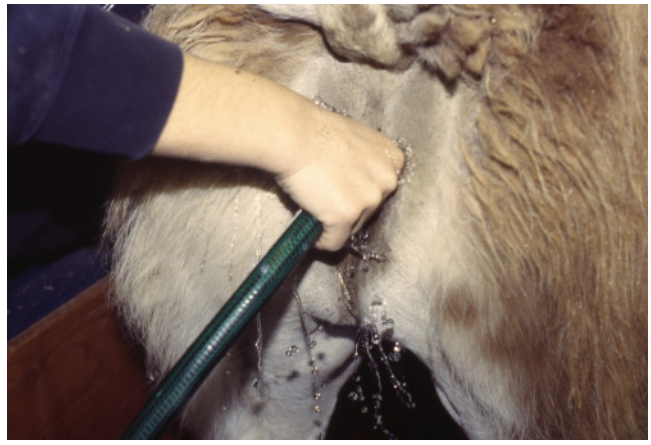


Figure 4.22. Holding the hose next to the anus to allow water to flow in and out of the rectum.

excess fluid or exudate, or remove air from the pleural spaces. Penetration can be made from either side. The preferred site is at the sixth or seventh intercostal space (there are twelve ribs), 10 to 15 cm (4 to 6 in.) dorsal to the ventrum of the sternum or 2 to 4 cm dorsal to the costochondral junction of the ribs. The area should be clipped and prepared for aseptic surgery.

Selection of the needle or cannula to be used is determined by the purpose for the thoracocentesis. If fluid is expected, a 14- to 16-gauge, 5-cm (2-in.) needle should be inserted near the cranial border of the rib to avoid the intercostal vessels. The pleural cavity may be penetrated at an approximate depth of 2 to 3 cm (1 to 1.5 in.). Fluid should flow from the needle. A spinal needle and stylet may be selected if air is to be removed.

If the lung has collapsed, a more dorsal and caudal position should be selected. Entrance should be made at the eighth or ninth intercostal space, approximately 25 cm from the dorsum of an adult llama. Distances for smaller individuals must be reduced proportionately. With a standard technique an indwelling catheter may be placed within the pleural space.

Penis

The penis is situated in the ventral pelvic area and has a sigmoid flexure similar to that of other artiodactylids. The prepuce is directed caudally in the unaroused male. The orifice of the prepuce is under the control of two sets of muscles: the cranial prepucial muscle, which pulls the prepuce cranially, and the caudal prepucial muscle, which pulls it caudally. When urinating, the male squats to spread the rear limbs.

The accessory sex glands include a small prostate gland and a pair of bulbourethral glands but no seminal vesicle. The erect penis is less than 2 cm in diameter and has a cartilage projection at the tip of the glans (Chapter 17). The urethral opening lies alongside the process. The terminal urethra is only 3 to 5 mm in diameter, allowing passage of only a number 5 French catheter. Urinary stones have been found lodged approximately 6 to 10 cm from the tip of the glans.

Foot

The foot of the llama is unique, with two digits on each foot (Chapter 6). The bearing surface of each digit has a soft, pliable sole and a digital cushion that is composed of fibroelastic tissue similar to the tissue of the digital cushion of the equine. Between the sole and the digital cushion is a layer of undifferentiated fibrous tissue that is thinnest near the toe and thickest at the bulb of the heel. The suborder name for camelids is Tylopoda, meaning "padded foot." A small, true nail tips each digit, and phalanges 2 and 3 lie horizontally above the cushion.

Old World camels have a single sole and pad beneath the digits, while SACs have a pad beneath each digit. In the camel, P-1 is essentially vertical, P-2 is approximately at 45 degrees, and P-3 is horizontal.

Bone Marrow Aspiration

Bone marrow is collected from one of the sternebrae. The wing of the ilium is too thin to allow penetration of the marrow cavity, and the rib marrow is less consistent in its location than the marrow of the sternebrae. An appropriate site is approximately 3 to 4 cm dorsal to the callosity on the ventrum of the sternum (Figure 4.23). Some clinicians prefer to insert the needle vertically through the sternal callosity. The author prefers the lateral approach. The needle[a] should be directed medially and slightly dorsally to engage the bone. Significant pressure exerted on the needle is required to enter the bone. A grating may be felt with the thrust. The needle should not be twisted during the insertion or the bevel will be damaged.

Figure 4.23. Needle position for collecting a bone marrow sample.

Marrow may be obtained at a depth of approximately 2.5 cm in an adult SAC, and 3 to 5 cm in camels. After penetration, the stylet should be removed and a 12-ml syringe attached. Withdrawing the plunger with a pumping action should yield a sample. Marrow in the llama is diluted with blood and is more fluid than that of cattle and horses. A smear made of marrow shows fat droplets. Smears may be made directly, or the sample may be placed in an anticoagulant vial. The sample must be mixed thoroughly with the anticoagulant before the needle is removed from the vial to ensure collection of a proper sample.

If no marrow is obtained, the stylet should be replaced and penetration continued. After two or three unsuccessful attempts at aspiration, the needle should be withdrawn and moved 2 to 3 cm cranial or caudad, assuming that the initial insertion was made into an intersternebral space.

Liver Biopsy

In the camelid the liver lies entirely on the right side of the abdomen. The biopsy needle may be directed

via ultrasonography, or a landmark approach may be used. The technique for obtaining a biopsy entails penetration of the caudal thorax, through the pleural space and the diaphragm, into the liver. Clipping a large area may be avoided by parting the fibers and fixing them out of the operative site with masking tape. An area 2.54 cm (1 in.) square should be clipped and prepared for aseptic surgery (Figure 4.24). Using local anesthesia, a liver biopsy may be done with the animal in either a standing or a sternally recumbent position.

The recommended site for performing a liver biopsy is at the ninth intercostal space and approximately 20 to 22 cm (9 to 10 in.) from the top of the back. The needle should be directed toward the midline, caudally and slightly ventrally (Figure 4.25). The chest wall is thin. The diaphragm is located immediately adjacent to the chest wall. To check proper site, let go of the needle. If the needle is in the diaphragm, it will move forward and back in synchrony with respiration. Because the liver lies immediately medial to the diaphragm, pushing the needle 2 to 3 cm deeper will penetrate the liver. The sample should be collected as indicated for the type of needle being used.

If the sample is desired for histologic examination, a wide choice of needles[b] is available. Disposable needles used in human medicine are satisfactory (Figure 4.26). If larger samples are required for nutritional studies, special types of trocars and cannulas are required.

Figure 4.24. Masking a small area for sterile collection of a liver biopsy.

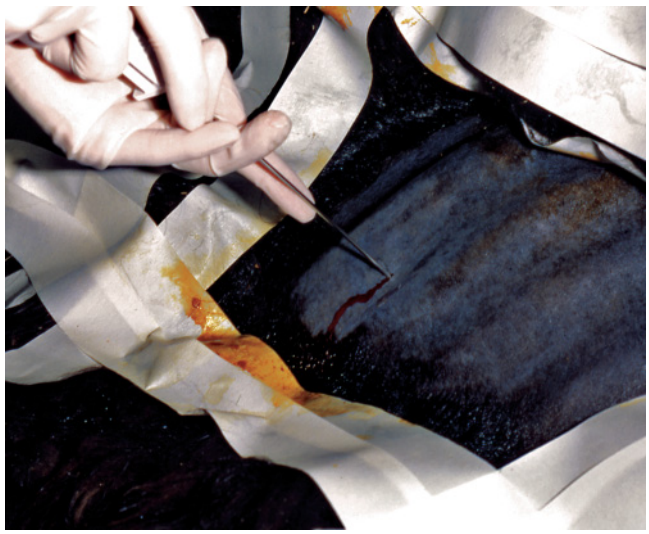


Figure 4.25. Insertion of needle for collection of liver biopsy.

Collection of Cerebrospinal Fluid[9]

INDICATIONS. Penetration of the subarachnoid space is performed to collect cerebrospinal fluid (CSF) for laboratory analysis, for conducting a myelogram, and to ascertain CSF pressure.

ANATOMY. The subarachnoid space may be entered either at the lumbosacral space or at the atlantooccipital space. The relationship of the subarachnoid space to the meninges, vertebrae, and spinal cord is the same in camelids as in livestock and horses. In the llama, the spinal cord ends at the level of the second sacral vertebral segment. The site should be prepared for aseptic surgery and appropriately draped.

ANESTHESIA. The type of anesthesia should be determined by the site and the procedure to be carried out. Myelography requires general anesthesia. Collection of fluid from the lumbosacral space may be done under local anesthesia, but collection from the atlantooccipital space should be done under general anesthesia to avoid uncontrolled movement and possible trauma to the spinal cord.

ATLANTOOCCIPITAL SPACE. With the head in a flexed position, the wings of the atlas should be palpated to locate the narrowest width at the cranial border (Figure 4.27). An imaginary line from wing to wing should be established and the skin penetrated directly on the midline, perpendicular to the cervical vertebrae.

The needle will penetrate the funicular ligamentum nuchae and then the dorsal atlantooccipital membrane. The cervical dura mater lies closely adjacent to the membrane, and with the head held in tight flexion, the membrane and dura will be tense. The needle should

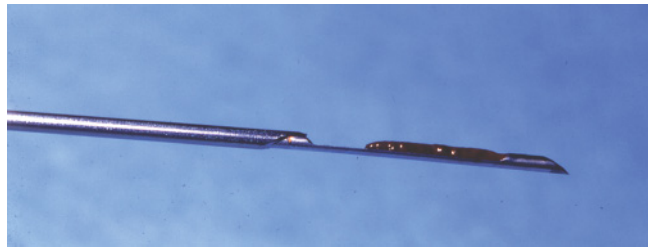


Figure 4.26. Liver biopsy sample collected with True-cut needle.

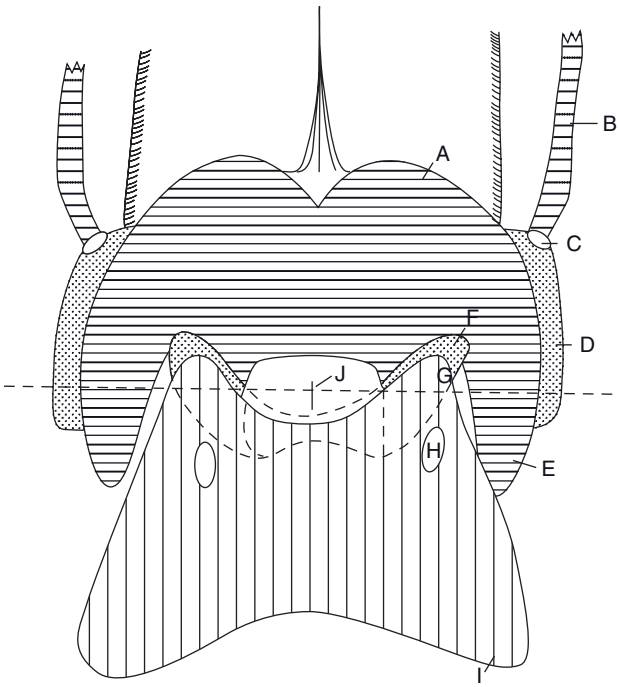


Figure 4.27. Diagram of the atlantooccipital space for cerebrospinal fluid collection. (A) Nuchal crest, (B) zygomatic arch, (C) osseous ear canal, (D) mastoid process, (E) jugular process, (F) occipital condyle, (G) cranial wing of the atlas, (H) alar foramen, (I) caudal wing of the atlas, (J) site for needle insertion.

penetrate the subarachnoid space with an audible or palpable "pop."

The stylet should be withdrawn to check for fluid. If none is present, the stylet should be reinserted and the needle rotated 90 degrees and again a check made for fluid. If still no fluid is encountered, the stylet should be reinserted and the needle advanced 1 or 2 mm deeper. This may be continued until fluid can be withdrawn. The needle should never be manipulated without the stylet in place, otherwise the spinal cord may be lacerated or a plug of neural tissue removed.

A 6.35-cm (2.5-in.), 20-gauge spinal needle is appropriate for this location. The depth of the subarachnoid space in an adult llama is approximately 4 cm (2 in.).

LUMBOSACRAL SPACE. The landmarks for this location are formed by the tuber sacrale of the pelvis and the dorsospinous process of the last lumbar vertebra (L-7). It is not difficult to palpate the dorsospinous process of L-7 because the dorsal processes of the sacrum are short. The correct site is 2 cm caudal to the dorsospinous process of L-7 on the midline, perpendicular to the vertebral column. The interarcuate space between L-7 and the first sacral vertebra (S-1) is large (2 cm cranial caudal and 4 cm wide) (Figures 4.28 to 4.30).

The tissues penetrated by the needle are the skin, thoracolumbar fascia, interspinous ligament, interarcuate ligament, dura mater, and arachnoid. A pop will not be felt when passing through the interarcuate

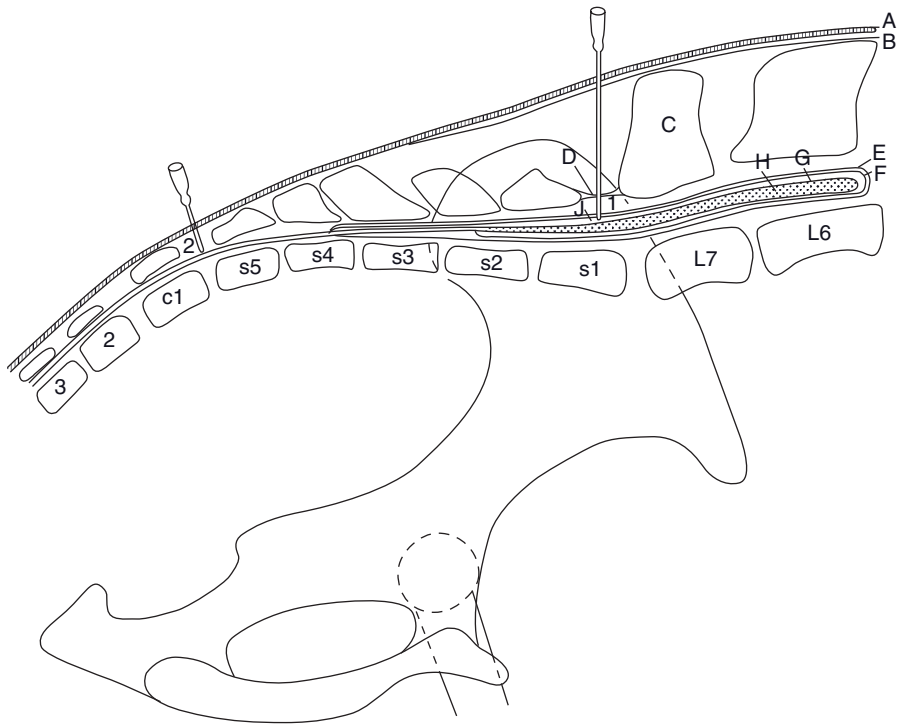


Figure 4.28. Diagram of the spine, with locations for (1) collecting cerebrospinal fluid and (2) administering epidural anesthesia.

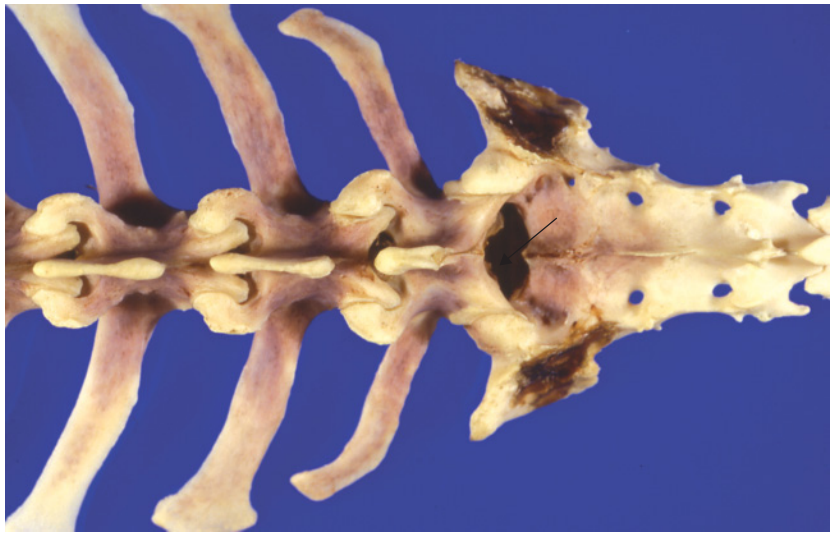


Figure 4.29. Lumbosacral space, dorsal view.

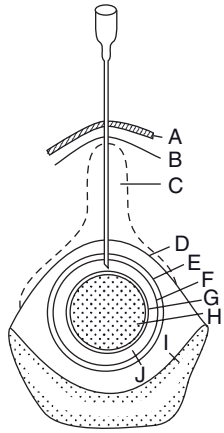


Figure 4.30. Diagram of a cross section at the lumbosacral space to illustrate layers penetrated to collect cerebrospinal fluid or administer spinal anesthesia. (A) Skin, (B) lumbodorsal fascia, (C) dorsal spine of the sixth lumbar vertebra, (D) interarcuate ligament, (E) dura mater, (F) arachnoid meningeal layer, (G) pia mater, (H) spinal cord, (I) body of the vertebra, (J) subarachnoid space.

Figure 4.31. Placement of a spinal needle.

ligament as in the atlantooccipital position, but decreased resistance should be noted.

A check for fluid should be made with each change of resistance. As previously indicated, the needle should never be moved without the stylet in place. If no fluid flows, the jugular may be occluded to increase intraspinous pressure (Figure 4.31). If still no fluid is encountered, the needle may be advanced through the conus medullaris of the cord to the floor of the spinal canal. Then the needle should be withdrawn, a millimeter at a time, until fluid flows.

The depth of the subarachnoid space is 6 to 6.5 cm in an adult llama and 2 to 2.5 cm in a neonate. The floor of the spinal canal is approximately 8 cm in an adult and 3.5 cm in a neonate. An 18- to 20-gauge, 9-cm (3.5-in.) spinal needle is necessary to reach the subarachnoid space in an adult llama. The depths are correspondingly less in the alpaca.

Table 4.7 lists the normal values for CSF along with a differential for various disorders.

Radiography

Radiography is an important component of clinical diagnosis of the skeletal system and is discussed in detail in Chapter 11.

Ultrasonography

Ultrasonography is now employed in evaluating the reproductive system, heart, and digestive system,

Table 4.7. Cerebrospinal fluid analysis in selected diseases.

	Normal	*Nonpurulent encephalomyelitis*	*Purulent encephalitis*
Pressure	<250 mm H_2O	Normal to elevated	>250
Transparency	Clear	Clear to cloudy	Cloudy with strings, coagulates
Color	Colorless	Colorless, whitish	White to reddish
Specific gravity	1.005–1.010	↑	↑
Refractive index	<1.3350	↑	↑
Erythrocytes	<35	?	?
Nucleated cells	<20	30–400	200–600
Neutrophils	None to few	±	+++
Lymphocytes	95% +++	++	±
Protein (mg/dl)	<80	< 200	>200–1000
Glucose (mg/dl)	<70	?	?

and is used to direct biopsy needles to the liver and kidney.

Endoscopy

Endoscopy has become a significant discipline in all facets of medicine. The same procedures and techniques that are used in livestock and horses may be applied to camelids.

Laparoscopy

The applications of this advanced technology are endless. In llamas and alpacas it has been employed to evaluate the reproductive and digestive tracts intra-abdominally and intraluminally and to visualize the esophagus, nasal cavity, and nasopharynx. Arthroscopy has also been used to diagnose osteochondrosis of the shoulder joint.

Computerized Tomography

Computerized tomography (CT) scan units are now being used in many veterinary school teaching hospitals.[5] Some have the capability of scanning only the head, but some units are able to support the entire body. The author has used the tool to diagnose meningocele, congenital agenesis of facets on the cervical vertebrae, and choanal atresia. It has application for diagnosis of otitis media and interna and certain dental problems. Deep anesthesia is required to perform this procedure.

Magnetic Resonance Imaging

Currently relatively few portable magnetic resonance imaging (MRI) units are available for veterinary use, and the cost of the examination is high. Anesthesia is required.

Blood Gas Analysis

A camelid's acid-base balance may be determined by analyzing venous blood (pH, HCO_3, base excess). Arterial blood is analyzed during anesthesia to monitor blood gases (pO_2, pCO_2, oxygen saturation).

Arthrocentesis

Arthrocentesis should be performed under strict asepsis. Anesthesia may be either sedation with xylazine HCl (0.1 to 0.4 mg/kg) or the use of a local anesthetic. A needle with a short rather than a regular bevel should be used. It may be difficult to obtain synovial fluid from a normal joint. In SACs the joints may be accessed as follows.

Shoulder. To aspirate the shoulder joint, palpate the acromion process of the scapula and the tendon of the infraspinatus muscle as it attaches to the greater tubercle of the humerus. The greater tubercle has a notch just cranial to the tendon. The needle should be inserted through the notch horizontally and slightly caudal to the vertical axis of the body.

Elbow. The needle should be inserted on the lateral side of the olecranon and directed ventrally in a line on the axis of the olecranon. The needle should be kept close to the olecranon.

Carpus. Either the radiocarpal or carpal metacarpal articulation can be palpated by flexing the carpus and separating the carpal bones. Insertion of the needle through the extensor tendons should be avoided. They may be palpated on the dorsal surface of the carpus. A 3- to 4-cm, 20-gauge needle should be used.

Stifle. Aspiration of the stifle joint capsule may be accomplished from three locations. It is unknown at the present just how these complicated joint capsule compartments intercommunicate, but joint fluid may be removed from an obviously distended compartment. The distal compartment may be entered by inserting the needle just proximal and slightly lateral to the tibial tuberosity. This requires at least an 8-cm needle. The interfemoral tibial fat pad is freely moveable and may inhibit penetration of the joint capsule unless the joint is distended.

As in the horse, another site for aspiration is alongside the lateral digital extensor muscle tendon as it passes over the tibial groove on the craniolateral border of the proximal end of the tibia.

A third location for aspiration is from the dorsolateral aspect of the patella. The needle should be inserted beneath the patella. The femoral patella compartment is large and, if excessive fluid is present, provides the easiest access to stifle joint fluid.

The anatomy of the stifle is unique in the llama. The tendons of the vastus lateralis and vastus medialis muscles form a sheet that inserts partly on the dorsal aspect of the patella, but also a free portion extends around the patella to insert on the tibial tuberosity. Instead of one, two, or three patellar tibial ligaments, there is a sheet of tendons composed of a combination of the quadriceps group plus the tendons of the biceps femoris muscle.

Hock. The needle should be inserted ventral to the lateral malleolus of the tibia and directed medially and ventrally to enter the tibiotarsal joint sac.

The metacarpal, tarsal, phalangeal, and interphalangeal joints should be penetrated on the dorsolateral or medial aspect in areas devoid of tendons, vessels, and major nerves, all of which can be easily palpated beneath the skin.

Placement of an Intravenous Indwelling Catheter

The procedure is commonly employed in premature or septic neonates to replace fluids and electrolytes, supply nutrients, and allow frequent intravenous medication. In mature animals with dehydration or septicemia, continuous fluid therapy is indicated.

The site for placement of an indwelling venous catheter is usually the right jugular vein at either the upper or lower position. Once the site is chosen, the area must be clipped, cleansed, and draped for aseptic insertion of the catheter.

In companion animals, horses, and livestock, it is common to clip the hair from an extensive area. This is not acceptable in camelids because it takes so long for the fiber to grow back. An adequate surgical preparation may be accomplished by a combination of tape drapes, clipping, removing the tape drapes, and redraping with tape before cleansing the site.

Locate the vessel by palpation or using landmarks. Separate the fiber parallel with the vessel (Figure 4.32). Stick strips of masking or duct tape to the parted fiber. Clip the fiber the width of the clipper blade (sideways), first one direction and then the other. This provides a window approximately 2.5 cm (1 in.) wide and 5 cm (2 in.) long. Pull the strips away, carrying with them the clipped fiber. Redrape the site, holding the fiber away from the site and using four strips of tape, as shown in Figure 4.33. Clean and disinfect the site.

The area may be draped further with a fenestrated cloth or paper drape or with sterile towels. This clipping and draping procedure is applicable for collecting cerebrospinal fluid, liver biopsy, or penetrating any other body site.

A bleb of local anesthetic agent is injected subcutaneously at the desired site of insertion, unless the animal is sedated or under general anesthesia. Do not attempt to thrust a catheter through the skin of a mature camelid; the skin in the upper cervical region may be as much as 1 cm (1/2 in.) thick. Even with a

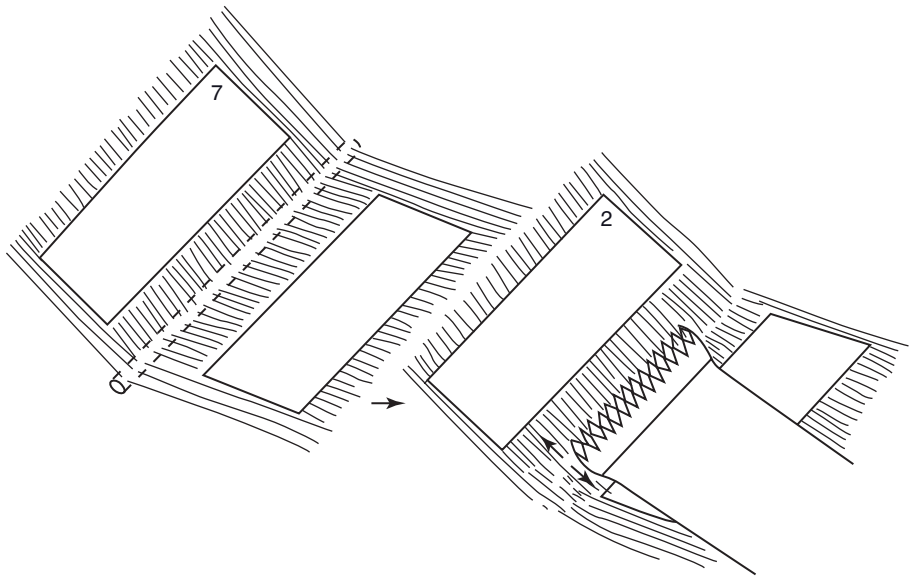


Figure 4.32. Diagram of a method of clipping a small area for preparing a field for aseptic procedures. Clip and remove the cut fiber with the tape.

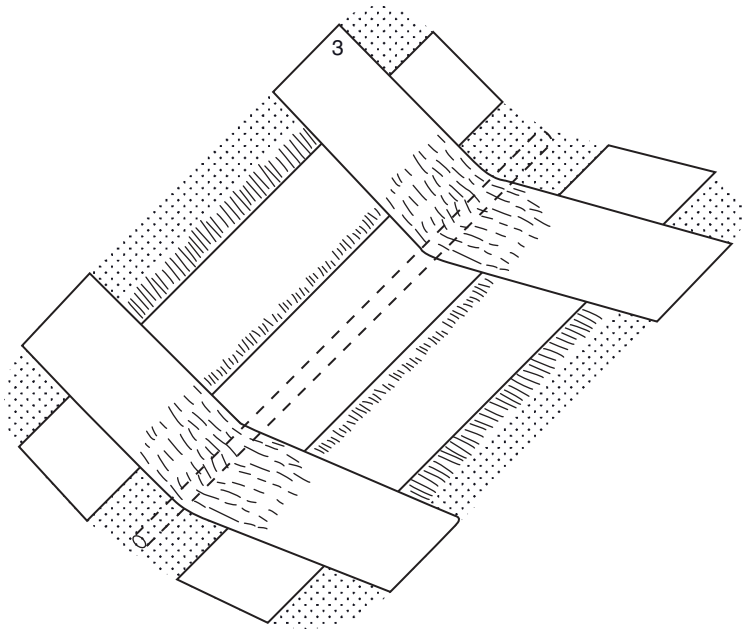


Figure 4.33. Diagram of a method of clipping a small area for preparing a field for aseptic procedures. Initial drape with tape.

neonate, it is easier to place the catheter following a skin incision.

With a number 12 scalpel blade, make a stab incision through the skin, taking care not to penetrate the vein. Distend the vein using digital pressure and insert the needle, pushing the catheter into the vein following the flow of blood from the needle. Selection of an appropriate catheter depends on the purpose for the placement and the length of time the catheter is to remain in place. Attach an adapter segment onto the catheter with a PRN port and flush the catheter with a heparin solution.

Once the catheter is fully inserted, it should be secured to the skin to avoid accidental removal. One technique is to place adhesive tape tabs on the hub of the needle or catheter. Place a 2.5-cm (1-in.) strip of tape behind the needle and place another 2.5-cm (1-in.) strip over the first. Tabs will project on either side of the hub. With simple sutures those tapes are then used to attach the catheter to the skin. Alternatively, the hub may be sutured directly to the skin. In either case, the use of superglue to close the skin incision and to anchor the catheter to the skin stabilizes the catheter greatly. If the skin incision is long, it may be desirable to place a simple interrupted suture, taking care not to penetrate the catheter. A sterile compress should be placed over the site and the neck bandaged after removing the draping tape, leaving the adapter catheter exposed.

When the catheter is no longer needed, gently withdraw it and apply pressure to the wound for a few moments. With the dressing gone, the fiber may completely cover the clipped area.

Additional Resources to Aid in Diagnosis

Camelid veterinarians have been extremely helpful with colleagues who request help on a difficult case. In the United States, there are at least three Internet listed servers where one is able to post a question which is usually answered in a short time:

1. International Camelid Institute (ICI) at Ohio State University, Columbus, Ohio, email: info@llamabanner.com
2. American Association of Small Ruminant Practitioners (AASRP), email: aasrp@aasrp.org, website: www.aasrp.org.
3. Lama Medicine list server, Oregon State University, Corvallis, Oregon, email: lama_med@lists.oregonstate.edu.

There may be similar internet accesses in other countries of the world.

There is an annual three- or four-day meeting of camelid veterinarians held in alternate years at Columbus, Ohio, and Corvallis, Oregon.

Journals containing significant papers dealing with camelids include:

1. Journal of Camel Practice and Research, Bikaner, India.
2. Australian Veterinary Journal, Australia.

3. Journal of Wildlife Diseases, United States.
4. Journal of Zoo and Wildlife Medicine, United States.
5. Journal of the American Veterinary Medical Association, United States.

There are several textbooks on camelid medicine as listed in the references for each chapter. There are thousands of scientific papers in various journals published during the last fifteen years. A "Google" search can retrieve many of these papers. Also, a search of bibliographic databases such as CAB Abstracts or PubMed will yield access to the papers or abstracts.

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[a] Monoject bone marrow biopsy needle, 5 cm (2 in.), 16 gauge, Sherwood Medical, St. Louis, MO, 63103.

[b] Tru-cut disposable biopsy needle, 15.2 cm (6 in.), 14 gauge, Travenol Laboratories, Deerfield, IL 60015; ABC actuated biopsy needle, 15.2 cm (6 in.), 14 gauge, Monoject (Sherwood Medical), St. Louis, MO, 63103; Silverman biopsy needle, 15.2 cm (6 in.), 14 gauge, many medical suppliers.

5

Anesthesia

LOCAL ANESTHESIA

In camelids, infiltration of a surgical site with a local anesthetic agent is a routine practice for lancing abscesses, suturing lacerations, laparoscopy, abdominocentesis, and thoracocentesis. Local anesthesia may also be used in combination with mild sedation for such surgical procedures as castration or a standing exploratory laparotomy.

Regional nerve blocks are used in SACs and OWCs, particularly in their native countries.[16,22,24] Regional nerve blocks are less often selected in SACs because the course of the nerves is not well documented, but what is known indicates wide variation from other livestock species.[24] For instance, the major nerve supply to the foot is located only on the medial aspect of the metacarpus and metatarsus rather than bilaterally, as in the equine and bovine. An inverted "L" local block is appropriately used for a flank laparotomy incision to avoid interference in wound healing as a result of anesthetic agent infiltration of the operative site.

Any of the standard local anesthetic agents are suitable. Selection may depend on the rapidity of onset and duration of anesthesia desired or the experience of the operator (Tables 5.1, 5.2). Any local anesthetic agent may be toxic if the administered dose is excessive.

For example, a two-month-old llama was sedated with xylazine at 0.4 mg/kg and the surgical site for an umbilical herniorrhaphy was infiltrated with 40 ml of 2% lidocaine. The cria died while exhibiting respiratory distress and tachycardia.[10] Assuming that the cria weighed approximately 25 kg at two months, the dose of lidocaine was 32 mg/kg. The lethal dose reported for horses is 4.5 mg/kg.

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Overdosage of lidocaine and most other local anesthetic agents causes central nervous system (CNS) stimulation, with muscle twitching and convulsions. Additional signs may be hypotension, nausea, vomiting, tachycardia, and dyspnea.[11]

Epidural Anesthesia

Indications: Epidural anesthesia is indicated for any type of perineal surgery (suturing lacerations, penetration of a persistent hymen), replacement of a prolapsed rectum or vagina, and prevention of excessive straining during rectal palpation.

Preparation: Epidural anesthesia should not be performed without surgical preparation of the site, which requires clipping the fiber. Minimal disfigurement results if a 1-cm^2 patch around the site is clipped with scissors. The remaining fibers may be parted and taped away from the site with masking tape. The site should be scrubbed and disinfected.

Technique: The tail should be grasped with one hand and rocked up and down to locate the sacrococcygeal intervertebral space. The five sacral vertebrae (S-1 to S-5) are fused in most individuals, so the first moveable intervertebral space would be between S-5 and coccygeal 1 (C-1) (see Figure 4.29 in Chapter 4). In a few individuals, S-5 is free and moveable. The space available between S-4 and S-5 for penetration into the neural canal is minimal. If difficulty is encountered when attempting an epidural, move caudally to the second moveable space to facilitate the procedure.[12,17]

A 1.5-in., 20-gauge needle should be inserted perpendicularly or slightly cranial to the slope of the tailhead, directly on the midline. The depth of the spinal canal is 1.5 to 2 cm in a 130-kg llama and deeper for camels. If the needle is positioned properly, the fluid should flow and drain from the hub of the needle when the syringe is detached and a drop of the anesthetic agent is put on the hub. The needle should be

Table 5.1. Comparison of injectable local anesthetic agents.

Agent	*Trade names*	*Company*	*Formulation*	*Onset (min)*	*Duration (hr)*	*Single–dose toxicity*
Procaine HCl	Procaine, Novocaine	Various, Winthrop	1–2% sol. 1% and 10% sol.	2–5 (2% sol.)	0.25–0.5 (2% sol.)	
Lidocaine HCl	Lidocaine, Xylocaine	Various, Astra	1–2% sol. 2% sol.	0.5–1 (1% sol.)	0.5–1 (1% sol.)	>4.5 mg/kg
Lidocaine HCl with epinephrine	Lidocaine	Various	1% sol.	0.5–1	2–6	>7 mg/kg
Mepivacaine	Carbocaine	Winthrop	1–2% sol.	3–5	0.75–1.5	
Bupivacaine	Bupivacaine, Marcaine	Abbott Winthrop	0.25–0.75% sol.	5 (0.25% sol.)	2–4 (0.25% sol.)	

Note: Facts and comparisons are from Drug Information Service, J.B. Lippincott, St. Louis, MO 63246.

Table 5.2. Topical local anesthetic agents.

Agent	*Trade name*	*Company*	*Formulations*	*Indications*
Proparacaine HCl	Ophthaine, Ophtheltic proparacaine	Squibb Allegram, various	0.5% sol.	Ophthalmic
Tetracaine HCl	Tetracaine, Pontocaine	Various, Astra	0.5% sol. 0.5% ointment	Ophthalmic
Lidocaine HCl	Lidocaine, Xylocaine	Various, Astra	5% ointment 2% jelly 10% sol.	Mucous membrane, endotracheal intubation, urethral catheterization
Benzocaine HCl	Benzocaine, Americaine	Various, American	20% gel 20% gel	Oral and laryngeal, mucosa, endotracheal intubation, endoscopy

Note: Facts and comparisons are from Drug Information Services, J.B. Lippincott, St. Louis, MO 63246.

left in position until motor control of the tail is lost, in approximately one to two minutes.

Solutions containing preservatives should not be used for epidural or spinal anesthesia. One to 2 ml 2% lidocaine HCl, without epinephrine, provides anesthesia for more than an hour and allows a llama to remain standing. Higher volumes may allow cranial migration of the agent, involving the nerves to the hind limbs, resulting in incoordination and recumbency.

In a study conducted in Peru, 2% lidocaine HCl was used epidurally in doses of 1 to 5 ml. Anesthesia was accomplished within five minutes, persisting as long as five hours. Higher doses may cause paralysis of the hind limbs.[14]

Analgesics

Phenylbutazone[8] (Butazolidin)

Oxyphenbutazone is similar.

Pharmacology: The analgesic effect of phenylbutazone is produced by its anti-inflammatory action. It has been used extensively in a variety of species, including humans, to treat musculoskeletal disorders and reduce soft tissue inflammation. It also has an antipyretic effect.

Indications: Phenylbutazone is recommended for the same types of conditions for which it is useful in horses and livestock. The oral or intravenous (IV) dose is 2 to 4 mg/kg once daily. Perivascular injections of phenylbutazone may result in phlebitis and adjacent skin slough. There has been insufficient usage of phenylbutazone to enable drawing of conclusions as to its efficacy in various disorders. In general, however, it seems to be more effective in relieving musculoskeletal pain than for soft tissue and colicky pain.

Phenylbutazone is supplied in 50- and 100-ml bottles (200 mg/ml); as 1-g, 2-g, and 4-g boluses; in 8-g packages of granules; and as a paste that can be measured in grams of phenylbutazone.

Side Effects and Precautions: Phenylbutazone has been reported to be ulcerogenic in the equine. There are no convincing reports of ulcers in llamas following its use. There has been no evidence of hematologic disturbance in camelids, as has been reported in humans.

Flunixin Meglumine[7] (Banamine)

Pharmacology: Flunixin is a nonsteroidal anti-inflammatory agent that has analgesic and antipyretic

effects. It has proven to be an effective analgesic for colicky pain.

Indications: Flunixin has been used extensively in camelids for analgesia in musculoskeletal disorders. It seems to be more effective than phenylbutazone in the relief of gastrointestinal (GI) spasms. However, automatic administration of this drug early in a colicky disorder may mask clinical signs, delaying differential diagnosis and selection of appropriate therapy.

Flunixin is supplied in granular form in 250-mg packets and as a solution in a concentration of 50 mg/ml. The recommended IV dosage for llamas is 1.1 mg/kg, given once daily.

Side Effects and Precautions: Although never reported in camelids, intraarterial injection of flunixin has resulted in ataxia, rapid breathing, muscle weakness, and hysteria in other species. As stated before, flunixin may mask critical signs.

Aspirin[8]

Formerly acetylsalicylic acid.

Pharmacology: Aspirin is a nonsteroidal anti-inflammatory analgesic. It is often administered to animals but is not used as extensively as it is in humans. Aspirin has a variety of pharmacologic actions that have little relevance to camelids.

Indications: The primary indications for aspirin are to relieve mild musculoskeletal postsurgical pain when the cost of more potent analgesics is prohibitive or if therapy is contemplated for a long time.

There are no studies upon which to base a sound dosage regimen. Extrapolating from cattle, an oral dose of 5 to 100 mg/kg given twice daily may be appropriate.

Side Effects: None reported.

Sedation, Tranquilization, Chemical Immobilization

Many procedures can be carried out on nervous or apprehensive camelids by dulling the sensorium with tranquilizers or low doses of injectable anesthetic agents (Tables 5.3, 5.4). In some instances the animal remains standing, at other times recumbency may be necessary. Chemical immobilizing agents are routinely used to render wild, dangerous zoo animals immobile. Immobilization of obstreperous or vicious male camelids may be necessary to carry out even such minor procedures as nail trimming. Chemical immobilization of vicuñas or untrained guanacos may be preferable to physical restraint.

Individual drugs are described below, followed by a discussion of applications, either individually or in combination.

Etorphine Hydrochloride (M99)[8,11]

Pharmacology: Etorphine is a highly potent narcotic analgesic, producing pharmacologic effects similar to those of morphine, namely, depression of the respiratory and cough centers, decreased GI motility, elevated blood pressure, tachycardia, and behavioral changes. In camelids, low doses of etorphine cause CNS stimulation with muscle rigidity, tremors, and possibly convulsions. At higher doses, paradoxically, the CNS is depressed.

Administration: Etorphine is readily absorbed from an intramuscular (IM) site. Onset of anesthesia occurs five to fifteen minutes after IM injections. If no antidote is administered, recovery is slow, requiring up to three hours. When the antidote is injected, the animal becomes ambulatory within two to ten minutes. Etor-

Table 5.3. Chemical restraint agents for New World camelids.

Generic name	*Trade name*	*Sedative dose mg/kg*	*Immobilization dose mg/kg*	*Reversal agent mg/kg*
Xylazine HCl	Rompun	0.25–0.30	1–2	Yohinbine 0.125–0.25, Tolazsoline 0.5–1.5
Xylazine/ketamine	Rompun/vetalar		0.4/5 IM 0.25/3–5 IV	Tolazoline for xylazine
Xylazine/ketamine/butorphanol	Rompun/vetalar/torbugesic		0.1/2–3/0.05–0.1 IM	Tolazoline/none/naltrexone
Xylazine/butorphanol	Rompun/torbugesic	0.2/0.05		Tolazoline
Acepromazine	Promace	0.05–0.1		None
Diazepam	Valium	0.05–0.3		Flumazenil 1.0–2
Diazepam/ketamine	Valium/vetalar		0.2–0.3/5–8 IM 0.1–0.2/3.0–5 IV	
Meditomidine/ketamine	Dormitor/vetalar		0.06–0.08/2–4 IM	Atipamezole 4–5 × dose of meditomidine IV or 0.1–0.15 mg/kg
Detomidine/ketamine	Dormosedan/vetalar		0.02–0.04/2–4 IM	Atipamezole
Tiletamine/zolazepam	Telazol, tilazol		4–6	Flumazenil for zolazepam

Table 5.4. Chemical restraint agents for camels.

Generic name	*Trade name*	*Sedative dose mg/kg*	*Immobilization dose mg/kg*	*Reversal agent mg/kg*
Xylazine HCl	Rompun	0.25–0.50	1–2	Yohinbine 0.125–0.25 Tolazsoline 0.5–1.5
Xylazine/butorphanol	Rompun/torbugesic	0.2/0.05		Tolazoline
Acepromazine	Promace	0.05–0.1		None
Diazepam	Valium	0.05–0.3		Flumazenil 1–2
Xylazine/ketamine	Rompun/vetalar		0.25–1/1–3	Tolazoline for xylazine
Xylazine/ketamine/ butorphanol	Rompun/vetalar/ torbugesic		0.1/2–3/0.05–0.1	Tolazoline/none/ naltrexone
Xylazine/diazepam/ butorphanol	Rompun/valium/ torbugesic		0.1/0.2/0.05	Tolazoline/flumazenil/ naltrexone
Meditomidine/ketamine	Dormitor/vetalar		0.06–0.08/2–4	Atipamezole 4–5 × dose of meditomidine IV or 0.1–0.15 mg/kg
Meditomidine	Domitor	0.01	0.02–0.03	Atipamezole
Detomidine	Dormosedan		0.03–0.06	Atipamezole

phine should not be mixed with atropine, because atropine reduces its solubility.

Side Effects: Inhibition of respiratory centers may directly or indirectly influence blood gases and acid-base balances. Etorphine is extremely dangerous to humans. If injected accidentally, medical help should be sought immediately. Naloxone or the specific antidote, diprenorphine (M50-50), should be administered intravenously. Equipment for artificial respiration should be kept available to deal with possible respiratory arrest.

Etorphine is readily absorbed through mucous membranes and may be absorbed through abraded or lacerated skin. It is important to avoid inhalation, ingestion, or contamination of the skin, particularly of the hands, which might touch the mouth.

Antidote: Diprenorphine (M50-50) is a specific antidote for etorphine. The standard dose is double the amount of etorphine injected. If diprenorphine is unavailable, naloxone or naltresone may be used.

Application in Camelids: Etorphine has been recommended for llamas, but the author has not been favorably impressed with its use. In one trial, a 135-kg llama gelding was given 0.5 mg etorphine HCl intramuscularly. In five minutes the llama began to stiffen and fell onto its side. The llama was rigid, the legs stiffly outstretched with some jerking. The head was raised, as if the llama were trying to rise. The preinjection heart rate (HR) was 62 and the respiratory rate (RR) was 20. Ten minutes after the first injection, another 0.5 mg M99 was given intramuscularly. Additional relaxation was achieved, but it was not complete. At minute 15, the HR was 66 and the RR was 4. At minute 20, the HR had increased to 110, with the RR remaining at 4 or 5.

In another trial, 1 mm etorphine was administered to a 130-kg llama. The animal became recumbent in ten minutes but remained rigid. This dose was insufficient to completely immobilize the llama. Diprenorphine was administered, and within two minutes the llama was standing.

Heck[11] immobilized adult guanacos with etorphine at 1.4 mg total dose and adult llamas with 1.5 mg total dose. No mention was made of rigidity as a complication. Merilan used 1.5 mg etorphine combined with 20 mg xylazine in adult camelids for electroejaculation (see Chapter 17).

Prior experience with numerous zoo species has led the author to believe that the dosage being reported for camelids is low and that a minimum of 2 mg should be given to an adult llama. Also, as in many other species, such as the equines, it is preferable to combine etorphine with xylazine or acepromazine to minimize the CNS stimulation effected by etorphine.

Carfentanil (Wildnil)[8]

Pharmacology: Carfentanil is similar to etorphine and is the main narcotic used in place of etorphine. The purchase of either etorphine or carfentanil requires a special license in addition to a standard Drug Enforcement Administration (DEA) license (contact a local DEA office).

Indications: The drug may be used in any application for which etorphine is applicable. It is primarily used in large ungulates, such as camelids. Carfentanil is usually combined with xylazine or other α_2 agonists or tranquilizers.

Administration: Carfentanil may be administered intravenously, intramuscularly, or subcutaneously.

Side Effects and Precautions: Same as etorphine.

Antidote: Diprenorphine (M50-50), naloxone, naltrexone, nalmefene.

Thiafentanil (A 3080)[8]

Pharmacology: Thiafentanil oxalate is a synthetic derivative of fentanil. Its action is similar to carfentanil, but it has a shorter duration and a greater margin of safety.

Indications: It is used in place of other immobilizing narcotics, but has been shown to be especially useful for the giraffe, eland, and kudu. There are no reports of conditions in camels for which its use may be indicated.

Administration: Thiafentanil may be administered by any parenteral route.

Side Effects and Precautions: Similar to etorphine and carfentanil, but renarcotization is less common than with the other opiate restraint drugs.

Antidote: Naltrexone is the antagonist of choice. Reversal should occur in less than a minute.

Butorphanol Tartrate[8] (Torbugesic)

Pharmacology: Butorphanol is a centrally acting narcotic agonist-antagonist analgesic.

Indications: Butorphanol has been used extensively in camelid clinical practice, either alone or in combination with other sedatives, to calm an excitable animal for examination or minor surgical procedures. In horses, it is used to alleviate abdominal pain caused by obstruction of the intestine, spasmodic colic, tympanitic colic, and postpartum pain.

Administration: Butorphanol may be administered intravenously, intramuscularly, or subcutaneously in a dosage range from 0.02 to 0.1 mg/kg. The drug is supplied in vials containing 10 mg/ml.

Side Effects: Slight ataxia initially following IV administration.

Antidote: Butorphanol has narcotic antagonistic action approximately equivalent to that of nalorphine. A marked overdose of butorphanol may be reversed by a larger dose of nalorphine because nalorphine has no agonistic effect.

Ketamine Hydrochloride[8,15] (Ketalar, Vetalar, Ketaject, Ketanest)

Pharmacology: Ketamine is a nonbarbiturate dissociative anesthetic agent. The animal usually retains normal pharyngeal-laryngeal reflexes. This desirable effect minimizes accidental aspiration of food or ingesta. However, endotracheal intubation is difficult when ketamine is the only agent used.

Ketamine produces an increased RR with a decrease in the tidal volume. If ketamine is given intravenously at a too-rapid rate, apnea may be produced.

Ketamine does not produce skeletal muscle relaxation, rather a catatonia. There is profound analgesia at medium to high dosages, although analgesia of the visceral peritoneum may be less than optimal. Excessive salivation can be alleviated with atropine.

Animals experience transitory pain upon IM injection of the solution. Because ketamine crosses the placenta, anesthetic effects are noted in the fetus when ketamine is used as a sedative for cesarean section or dystocia.

Ketamine produces a fixed expression in the eyes. The eyelids stay open, yet the cornea usually remains moist. Occasionally, corneal ulceration has resulted from prolonged exposure. Palpebral reflexes persist. Ketamine is detoxified in the liver, and metabolites are excreted via the urine.

Administration: Ketamine is supplied as a solution in 20-mg/ml and 100-mg/ml concentrations.

Side Effects: Tonic-clonic convulsions are produced in some species but have not been observed in camelids except upon accidental intracarotid injections. Ketamine is not known to produce abortion. Regurgitation has occurred following ketamine use in camelids. It is difficult to say whether this was an effect of the drug or simply passive regurgitation from immobilization. Ketamine is rarely used as the sole immobilizing or anesthetic agent in camelids. Side effects are generally ameliorated by the combined drug.

Antidote: There is no known clinical antidote for ketamine.

Tiletamine HCl and Zolazepam HCl[8,15,28] (Telazol—Fort Dodge, Iowa; Zoletil—Palmvet, South Africa and Europe)

Pharmacology: Telazol is a combination of equal parts by weight of tiletamine base (an analog of ketamine) and zolazepam, a diazepinone tranquilizer related to diazepam. The combination produces the rapid dissociative anesthesia typical of ketamine and with the relaxing action of zolazepam.

Indications: Telazol is used for chemical immobilization, anesthesia induction, sedation, and analgesia for minor surgical procedures. Indications for its use are the same as for ketamine, but less rigidity is associated with telazol administration. In practice, telazol functions similarly to a combination of xylazine and ketamine.

Administration: The 5-ml vial contains 50 mg/ml of tiletamine base and 50 mg/ml of zolazepam. The administered dose is calculated from the combined (100 mg/ml) activity. Free-ranging adult male guanacos were immobilized with 4 to 6 mg/kg doses. The first signs of the drug's effect is incoordination, beginning at 2.9 minutes, with recumbency occurring at a mean time of 7 minutes (1.5 to 53 minutes). Immobilization lasts for 15 to 15.6 minutes. Characteristically, the higher the dose the longer the recovery period.

Side Effects and Precautions: In guanacos there is some muscle twitching and rigidity at lower doses. Hypersalivation was common and pronounced in some individuals. Chewing movements, retching, and vocalization have been observed.

Antidote: There is no antidote for tiletamine. Zolazepam effects may be countered with flumazenil (Mazicon)(see Table 5.8, later in the chapter).

Xylazine (Rompun)[2,4,5,13–15,18–22,24,29,31,32]

Pharmacology: Xylazine is not a narcotic; it is a sedative, analgesic, and muscle relaxant, producing its effect by stimulating both central and peripheral presynaptic α_2 adrenoceptors. Under the influence of xylazine, animals appear to be sleeping. Other actions include depressed thermoregulation; hyperglycemia; decreased heart rate, cardiac output, and aortic flow; temporary increase in blood pressure followed by hypotension; and respiratory depression.

Administration: Xylazine is supplied in 20-mg/ml and 100-mg/ml solutions and may be given intravenously or intramuscularly. Immobilization occurs within three to five minutes following IV injection or ten to fifteen minutes after IM injection. Analgesia lasts from fifteen to thirty minutes, but the sleeplike state is maintained for one to two hours. Painful procedures should not be performed after thirty minutes.

Side Effects and Precautions: Stimulation during the induction stage may prevent optimum sedation. Seemingly sedated animals have roused explosively, negating the sedation. Occasionally, muscle tremors, bradycardia, and partial atrioventricular block occur with standard doses. Salivation is pronounced in camelids; atropine (0.04 mg/kg) should be given to counter cardiac effects and diminish salivation.

Intracarotid administration produces transient seizures and collapse.

Xylazine has been used extensively in llamas at all stages of pregnancy with no apparent abortions, as have been reported in the bovine.

Antidote: Yohimbine HCl (0.125 to 0.25 mg/kg) reverses the effects of sedation in camelids, presumably by blocking α_2 adrenoceptors, as it does in experimental animals; however, other reversal agents are being used now, such as tolazoline and atipamezole.

Detomidine HCl (Dormosedan—SmithKline Beecham)[1,6,8,15,27]

Pharmacology: Detomidine is a xylazine clone that is longer acting and more potent than xylazine. It is a non-narcotic sedative and analgesic α_2 agonist. Bradycardia, diuresis, and hypersalivation are noted.

Indications: Detomidine may be used in all situations in which xylazine is appropriate.

Administration: Detomidine is supplied in vials of 0.1 mg/ml. It may be administered intravenously or intramuscularly in doses of 0.02 to 0.04 mg/kg. The highest dose is necessary for analgesia. When administered intravenously the onset is two to four minutes and when given intramuscularly three to five minutes.

Side Effects and Precautions: Side effects and precautions are the same as for xylazine (hypertension, bradycardia, piloerection, sweating, and, rarely, excitement rather than sedation).

Antidote: The most satisfactory antidote is atipamezole.

Medetomidine HCl (Dormitor)[8,15,30]

Pharmacology: Medetomidine is the newest and most potent in the line of α_2 adrenoreceptor agonists, with action more specifically on receptors associated with sedation and analgesia. The drug is rarely used alone; it is usually combined with ketamine.

Indications: It is primarily used as an immobilizing agent in combination with ketamine.

Administration: Medetomidine may be administered intravenously or intramuscularly at dosage of 40–80 µg/kg. Induction is two to five minutes.

Side Effects and Precautions: Medetomidine is one of the safest immobilizing agents being used today. Although bradycardia is an inherent action of α_2 agonists, this is countered by ketamine. Regurgitation is rare in animals with multi-compartmented stomachs, and abortion has not been reported.

Antidote: Atipamezole (Antisedan V) is the specific antidote.

Acepromazine Maleate[3,8,15]

Pharmacology: Acepromazine maleate is a potent tranquilizing agent that depresses the CNS. It produces muscular relaxation and reduces spontaneous activity, exhibiting antiemetic, hypotensive, and hypothermic properties.

Indications: Acepromazine maleate is rarely used singly but is usually combined with ketamine or etorphine. Its muscle-relaxing characteristic is of particular value when combined with ketamine. Acepromazine maleate has been used in llamas. A dose of 0.15 mg/kg quieted an aggressive male that was subsequently anesthetized using mask induction with halothane. Barrie reported that an adult female guanaco (approximately 100 kg) was tranquilized for an eye examination with 3 mg (0.03 mg/kg).

Administration: Acepromazine is supplied in solution in a 10-mg/ml concentration that may be injected intravenously, intramuscularly, or subcutaneously. When given intravenously, effects are noted within one to three minutes. Intramuscularly, fifteen to twenty-five minutes are required for full effect. Reports of use are insufficient to establish a standard dose in camelids.

Side Effects and Precautions: Acepromazine should be used cautiously in combination with other hypotensive agents. Occasionally, instead of producing CNS depression, it acts as a stimulant, and hyperexcitability ensues. Acepromazine is a phenothiazine derivative and may potentiate the toxicity of organophosphate parasiticides, so inquiry should be made about the prior use of these products before administration. Acepromazine is contraindicated for the control of convulsions in progress. While the drug may prevent convulsions, it also reduces the threshold for convulsion stimuli.

Antidote: There is no known antidote.

Diazepam[8,15] (Valium, Tranimal, Tranimul)

Pharmacology: Diazepam acts on the thalamus and hypothalamus, inducing calm behavior. It has no peripheral autonomic blocking action, unlike some other tranquilizers. Transient ataxia may develop with higher doses as muscle relaxation progresses. Spinal reflexes are blocked. Diazepam is an effective anticonvulsant.

Indications: Diazepam prevents the convulsive effect of ketamine. If injected intravenously, it effectively controls convulsive seizures in progress. It can also be used as preanesthetic medication to calm an excited animal.

Administration: Diazepam is supplied in solution in a concentration of 5 mg/ml and administered at a dose of 0.1 to 0.5 mg/kg. Onset is within one to two minutes when given intravenously. If given intramuscularly, it takes effect in fifteen to thirty minutes, depending on the dose. Diazepam is metabolized slowly in the normal liver. Usually, clinical effects disappear within sixty to ninety minutes.

Side Effects and Precautions: Diazepam may be chemically incompatible with other immobilizing agents and it should not be mixed with them in the same syringe nor in IV solutions. Some pain is associated with IM injection, and a transient inflammatory reaction may develop at the site. Diazepam is contraindicated in patients with suspected glaucoma.

Antidote: Flumazenil (Mazicon) is a specific antagonist for benzodiazepine tranquilizers.

Midazolam HCl[8,15] (Versed)

Pharmacology: Midazolam is a short-acting benzodiazepine central nervous system depressant, similar to diazepam, but more potent.

Indications: Same as for diazepam.

Administration: Any parenteral route. Midazolam is a schedule IV drug in the Controlled Substance Act of 1970.

Side effects and precautions: Prolonged sedation, incoordination, nausea, vomition, and coughing may be seen.

Antagonists: Flumazenil (Mazicon)

Muscle Relaxants

Atracurium Besylate (Tracurium)[8]

Pharmacology: Atracurium is a neuromuscular blocking agent developed for use in large animals.

Indications: Atracurium is indicated to obtain muscular relaxation in an animal that is already under general anesthesia. It has application in orthopedic procedures and in ocular surgery where muscular relaxation is vital. The drug should be used only by a qualified anesthesiologist.

Administration: An initial bolus of 0.15 mg/kg is administered intravenously. Relaxation should occur within one minute and lasts for at least seven minutes. Repeated injections of atracurium must be given or an IV drip set up to administer 0.4 mg/kg/hour.

Side Effects and Precautions: Respiratory paralysis occurs, and assisted respiration is necessary to avoid hypoxia.

Antidote: There is no specific antidote. Relaxation time is short, and assisted respiration is all that is required.

Succinylcholine Chloride[8] (Suscostrin, Anectine)

Pharmacology: Succinylcholine chloride is a depolarizing muscle relaxant, with no analgesic or anesthetic properties.

Indications: Although the drug has been reported as having been used as an immobilizing agent for capturing guanacos, it is not recommended as a drug for dealing with camelids except during general anesthesia for orthopedic procedures.

Side Effects: This drug suppresses respiration, and suffocation will ensue without assisted respiration.

Guaifenesin[8]

Guaifenesin (Gecolate, guaiphenesin, glycerol guaiacolate) is a muscle-relaxing agent that is usually combined with the short-acting barbiturates xylazine or ketamine to provide analgesia and/or anesthesia. These combinations are used extensively in equine anesthesia. An anesthetist accustomed to working with equine species may prefer these combinations for injectable anesthesia. It has been found to be safe and effective in llamas. The dosage currently recommended in the llama is 100 to 150 mg/kg (1 to 1.5 ml/kg of a 10% solution). A short-acting barbiturate such as sodium thiamylal or sodium thiopental at a dose of 4.5 mg/kg may be added to the solution before administration to provide fifteen to twenty minutes of

anesthesia, which may be supplemented if the llama begins to awaken.

A disadvantage to the use of guaifenesin is the volume required to induce and maintain anesthesia. An IV catheter should be placed in the jugular vein before attempting to use this agent.

GENERAL ANESTHESIA

General anesthesia may be accomplished by the use of higher than sedation doses of some drugs used for sedation and immobilization. Gaseous agents may be used to induce and maintain anesthesia. It is difficult to stabilize the depth of anesthesia in llamas. When general anesthesia is selected for a camelid, the patient must be continuously monitored by a qualified person.

Preanesthetic Considerations

For elective surgery, the llama should be fasted for twenty-four to forty-eight hours and water withheld for eight to twelve hours. This will not completely empty compartment one (C-1) of the stomach but will decrease the volume and diminish gas production. To remove feed debris the mouth should be irrigated immediately before applying a mask or attempting tracheal intubation.

Both passive and reflex regurgitation are potential sequelae to general anesthesia in llamas. The primary factors predisposing to passive flow of ingesta from the stomach are relaxation of the gastroesophageal sphincter, surgical positioning, pressure buildup in the stomach (either gaseous or external), and a large volume of ingesta in the stomach.

In an anesthetized camelid, continuing contraction of C-1 of the stomach moves ingesta toward the cardia. In a normal cycle, the cardia relaxes and ingesta is propelled to the mouth for rechewing. This cycle is a hindrance during anesthesia. These mechanisms are under parasympathetic control; thus, atropine may diminish stomach contractility.

Laryngeal stimulation results in reflex closure of the glottis, which in turn causes high negative intrathoracic pressure during inspiration. This induces passage of ingesta into the esophagus. It is essential that the laryngeal reflex be abolished before attempting endotracheal intubation.

Positioning for Anesthetic Procedures

Camelids may be placed in any conceivable position for surgery, but selection should be made after consideration of various factors. Regurgitation is more likely in left rather than right lateral recumbency. Prolonged surgery in either right or left lateral recumbency may produce postsurgical radial paralysis. However, paralysis can be prevented by properly padding the shoulder and by pulling the lower limb forward to avoid direct pressure from the rib cage on the midhumeral region.

Dorsal recumbency should be avoided unless it is possible to place an endotracheal tube. If regurgitation has occurred during the surgery, special care must be taken while removing the tube. The head should be lowered and the mouth gently irrigated to remove any particulate matter remaining in the oropharyngeal cavity. The cuff should remain inflated while withdrawing the tube from the trachea, though if resistance is noted at the larynx, it may be necessary to deflate the cuff slightly. The inflated cuff will scoop any fluid or particulate matter out of the trachea. Excessive coughing may be noted in an animal that has inhaled fluid or feed particles.

Dorsal recumbency also shifts the weight of the abdominal viscera toward the diaphragm and the lungs, restricting inflation, which causes decreased functional residual capacity and closure of the small airways. Pressure is also applied to the greater abdominal vessels. There is decreased cardiac output and change in pulmonary blood-flow patterns, especially in the dependent areas of the lung. All of these cardiopulmonary changes may lead to hypoxemia.

A suitable method for maintaining a llama in dorsal recumbency is the simple cradle illustrated in Figure 6.16 in Chapter 6. Alternatively, the llama could be supported between two bales of straw or hay. Overflexion or overstretching of the limbs when clearing the limbs from the operative site with ropes should be avoided.

A llama may be kept in sternal recumbency to facilitate perineal surgery. However, keeping an anesthetized animal in this position for two or more hours may result in ischemia of the limbs. Ischemia does not occur in a conscious animal, even one that is down and unable to rise for various reasons.

Often, the forequarter is tilted downward during abdominal surgery to allow the viscera to drift out of the caudal abdomen and give better exposure. A camelid should be positioned like this only if an endotracheal tube is in place, because passive regurgitation is likely to occur.

Tracheal Intubation[26]

To provide positive support for respiration and avoid complications if the camelid should regurgitate, tracheal intubation is recommended whenever general anesthesia is selected. Intubation is also necessary to inflate a collapsed lung and to correct pulmonary edema.

Endotracheal intubation is difficult in SACs because the restricted space in the oropharynx impairs visualization of the glottis with a laryngoscope while the endotracheal tube is manipulated into position. The restricted space is the result of a combination of factors,

including the narrow space between the rami of the mandible, inability to open the mouth widely, the elevated mound on the dorsum of the caudal aspect of the tongue, and the elongated soft palate that may be situated either ventral or dorsal to the epiglottis (Figures 5.1, 5.2).

Camels are able to open their mouths widely so there is less difficulty with intubation. In adult camels the anesthetist can insert a hand and arm to the larynx and carry the endotracheal tube with the arm and hand.

Tubes designed for small animals are too short for use in llamas. Tubes designed for cattle and horses are too long. In adult llamas, the tube should be 50 cm long, with a diameter appropriate to the size or weight of the animal (Table 5.5). Use appropriately sized equine endotracheal tubes in camels.

To prevent reflex regurgitation, chewing and swallowing reflexes must be abolished before attempting to insert an endotracheal tube. Relaxation may be accomplished by use of a xylazine-ketamine or guaifenesin-barbiturate combination or by masking the animal for induction with an inhalant agent.

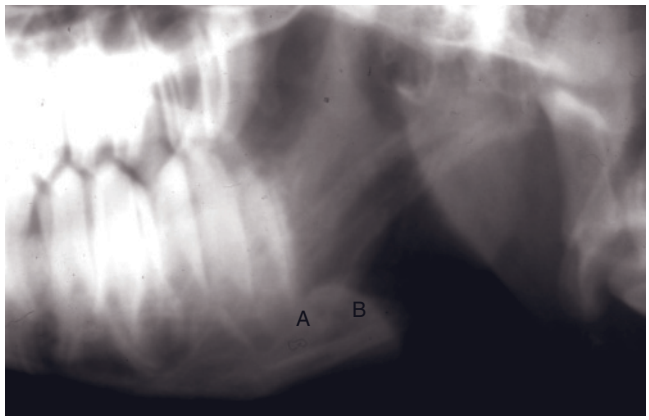

Figure 5.1. Lateral radiograph of the oropharyngeal region of a llama. The soft palate is ventral to the epiglottis. (A) Soft palate, (B) epiglottis.

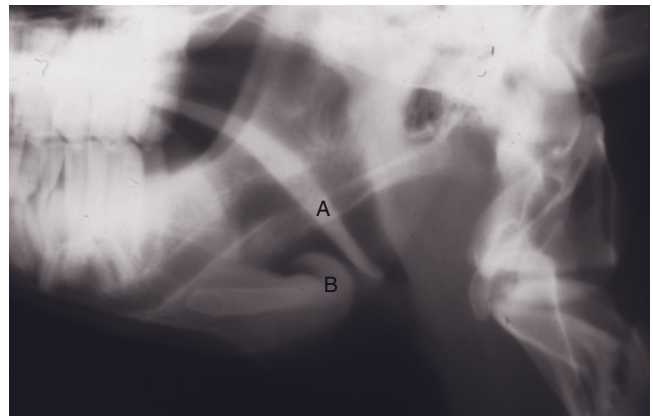

Figure 5.2. Lateral radiograph of the oropharyngeal region of a llama with the soft palate dorsal to the epiglottis. (A) Soft palate, (B) epiglottis.

Table 5.5. Endotracheal tubes for camelids.

Body weight (kg/lb)	*Inside diameter (mm)*	*Outside diameter (mm)*	*Length (cm)*	*French (size)*
9–60/20–132	3	4.7	30	14
	4	6	30	16
	5	7	35	22
	6	8.7	35	26
65–160/143–350	7	10	50	30
	8	11	50	33
	9	12.3	50	37
	10	14.7	50	44
	11	15.3	50	46
	12	17	50	51
160+/350+	14	19	50	57
	16	21	50	63
Adult camel				
700–1400/1543–3086	18	23.6	50	71
	20	31.8	50	76
	26	34.1	50	105

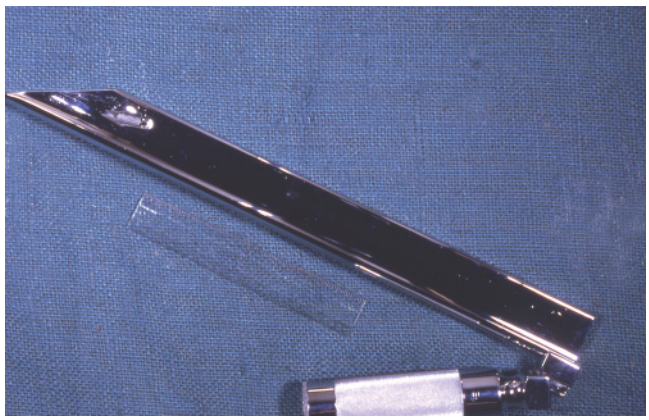

Figure 5.3. Long-bladed laryngoscope used for endotracheal intubation of camelids.

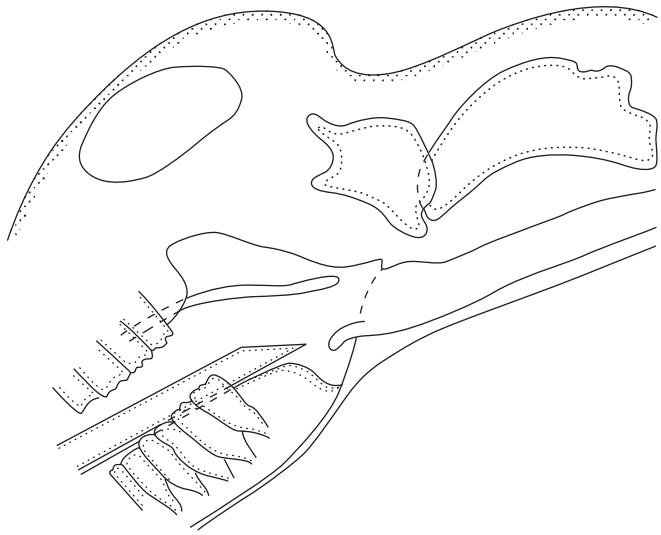

Figure 5.4. Diagram of a method of tracheal intubation. The laryngoscope blade is used to depress the tongue, providing visualization of the glottis.

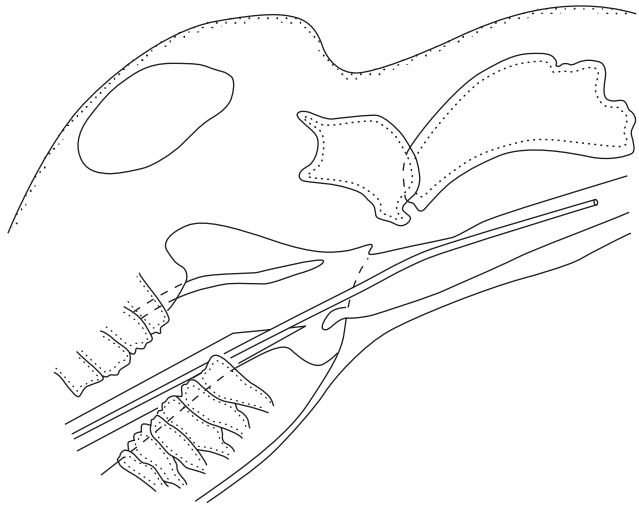

Figure 5.5. Diagram of a method of tracheal intubation. A plastic catheter is inserted into the trachea.

Placement

One method is to insert the endotracheal tube while visualizing the larynx with a laryngoscope (Figure 5.3). Some anesthetists prefer this method, even though visualization is impaired. In some animals, the limited space makes it impossible to visualize the laryngeal opening and insert the endotracheal tube at the same time. The mouth should be held open with gauze loops and the head extended maximally. The laryngoscope blade should be inserted to the base of the tongue which is pressed down to visualize the glottis. An aluminum rod stylet should be placed in the endotracheal tube to keep the tube rigid. The tube may be inserted through the laryngeal opening at the time of maximum opening. The stylet should be removed and a check made for air flow through the tube.

The author's preferred method is the insertion of a small catheter into the larynx over which the endotracheal tube is threaded. To do this, two 8- to 10-French, 50-cm stiff polyethylene catheters should be coupled with tape. A long-bladed (45 cm) laryngoscope is desirable, but a 19-cm blade can be used if the unit is inserted up to the commissure of the mouth.

The head should be extended maximally. It may be desirable to hold the mouth open with gauze loops and gently pull the tongue rostrally. The laryngoscope blade may be inserted and the epiglottic cartilage depressed (Figure 5.4). If the epiglottic cartilage is not visible, the soft palate may be lifted from beneath the epiglottis with the tip of the blade. The polyethylene catheter is inserted into the trachea (Figure 5.5) and the laryngoscope blade removed. The endotracheal tube should be threaded over the catheter and pushed gently between the cheek teeth into the trachea (Figure 5.6).

Some difficulty may be encountered in threading the endotracheal tube through the laryngeal opening. If anesthesia is too light, there will be reflex closure of the laryngeal opening as the tube touches the epiglottic or arytenoid cartilage. The endotracheal tube should be gently pressed against the larynx. The head may be repositioned slightly, or it may be helpful to fix the larynx from the exterior. If these adjustments fail, a slightly smaller tube should be tried (Table 5.5).

A check should be made for air movement through the endotracheal tube before the catheter is removed. The cuff should be inflated and the tube tied or taped to the lower jaw.

Camel Endotracheal Intubation

Camel anesthesia is essentially the same as for SACs. However, the oropharyngeal cavity is much

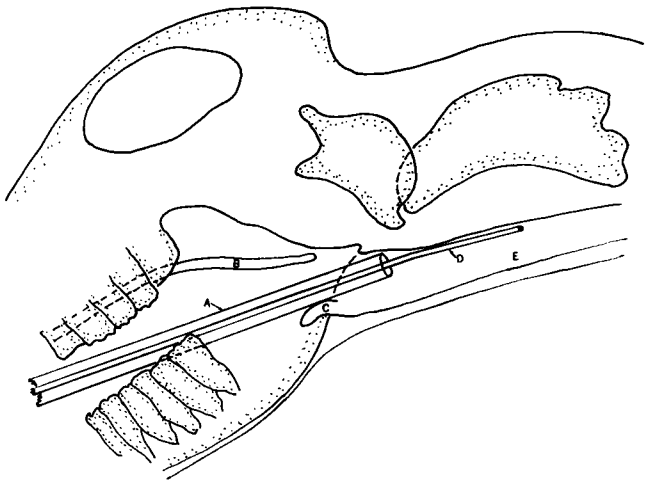

Figure 5.6. Threading the endotracheal tube over the catheter.

Figure 5.7. Positioning a llama for endotracheal intubation following chemical immobilization.

larger and the mouth will open widely. Another consideration is that in the male dromedary there is a diverticulum of the ventral aspect of the soft palate called the dulaa (also called the dulah, doolah, or doola) that may protrude from the mouth during the process of intubation. This may startle the novice camel anesthetist, but should merely be replaced or pushed aside to complete intubation.

Induction of an Inhalant Anesthetic

Sedation is advisable and the selection of the sedative may depend on the drugs available, the tameness of the camelid, the restraint facilities available, and the experience of the anesthetist. Xylazine has been used extensively as a sedative in camelids, but several other regimens are available. The method used at the veterinary school of the University of California, Davis, is as follows:

The right jugular area is prepared for surgical asepsis to allow placement of an IV catheter attached to a three-way stopcock. When ready to induce anesthesia, either diazepam 0.2 to 0.4 mg/kg or midazolam 0.1 to 0.2 mg/kg is administered IV. Several minutes are allowed for sedation to occur.

The camelid is then positioned near the site for the surgery. Ketamine HCl 2 to 4 mg/kg is administered IV. The animal should become recumbent. An assistant keeps the animal in a sternal position by straddling it at the withers. The head is extended maximally and the mouth is held open by gauze strips or small cords (Figure 5.7). An alternative to ketamine is the administration of propofol 1 mg/kg IV.

The anesthetist inserts a long bladed laryngoscope to the base of the tongue to visualize the glottis. If the soft palate is below the epiglottis it is lifted with the blade. At this point it is recommended that the arytenoid cartilages be sprayed with 2 ml of 2% lidocaine before attempting either the insertion of the directional catheter over which the endotracheal tube is threaded or the direct insertion of the endotracheal tube. The cuff is then inflated.

Although assessment of the pulse is not a usual clinical parameter, superficial arteries may be palpated in the anesthetized individual. The largest and most accessible artery is the saphenous, which lies on the medial aspect of the stifle, but the caudal articular and digital arteries may also be palpated. Ocular reflexes are used to assess depth of anesthesia. The ventral eyelid palpebral reflex is suppressed when anesthesia is adequate, but the upper lid will likely retain the response to tactile stimulation. Nystagmus rarely occurs. The administration of ketamine may complicate assessment of ocular reflexes until the inhalant anesthetic agent takes effect.

NEONATE ANESTHESIA

General anesthesia is usually reserved for invasive orthopedic procedures and laparotomy. The neonate will be less stressed if physical restraint and mild sedation or local anesthesia is selected whenever possible. For general anesthesia, inhalation anesthesia is preferred, with mask induction being the simplest. Regurgitation is not a major problem in the neonate, as it is in the adult llama.

Injectable Anesthesia

Numerous injectable anesthetic agents or combinations of agents have been used in camelids (Table 5.3). Refer to the pharmacodynamic discussions of the agents for details. In this section, applications for use in camelids are described.

Xylazine

Doses of 0.1 to 0.2 mg/kg xylazine HCl provide sedation, enabling excited or wild animals to be examined or certain special procedures to be carried out. A higher dose (0.25 mg/kg) will induce calm animals to lie down, especially if administered intravenously. This dose, in conjunction with a local anesthetic, provides sufficient analgesia to lance abscesses and suture lacerations.

The author's standard immobilizing dose is 0.25 mg/kg intravenously or 0.35 to 0.45 intramuscularly. Many diagnostic procedures may be performed under this degree of sedation and analgesia. Induction occurs within five minutes and lasts for no more than twenty minutes. Procedures that may be performed include abdominocentesis, thoracocentesis, bone marrow aspiration, radiography, and blood collection.

Some have recommended that higher doses of xylazine (0.4 to 0.7 mg/kg) may permit longer procedures, but the author prefers to combine drugs or shift to an inhalant anesthetic for more prolonged procedures.

Xylazine in Combination with Other Agents. Following are combinations that have been used by the author:

1. Xylazine (0.25 mg/kg) and ketamine (2 to 5 mg/kg). A similar combination has been successful in the equine, but Heath has seen difficulties with this combination in goats and thus does not recommend it for llamas. The author, on the other hand, finds this combination useful for minor surgery, dental procedures, preanesthetic induction, and endotracheal intubation. This combination has been used experimentally in our laboratory with extensive monitoring and has been found to be a safe anesthetic combination.

   Supplemental doses of either drug may be given intravenously without complicating recovery. Supplemental IM doses prolong recovery and should not be administered.
2. Xylazine (0.1 mg/kg) and butorphanol (0.051 mg/kg). This combination will allow most llamas to remain standing.

Diazepam and Ketamine

If use of xylazine is not desired, a combination of diazepam (0.2 to 0.5 mg/kg) and ketamine (2 to 5 mg/kg) provides adequate anesthesia for procedures in a variety of domestic and wild ungulates, including the llama.

Barbiturates

In two different studies of anesthesia in alpacas, sodium pentobarbital was used in a 6.5% solution (65 mg/ml). In one, the IV dose was 14 mg/kg and in the other, 20 mg/kg (7). Induction occurred in three to four minutes and lasted for approximately one hour. Both investigators considered the anesthesia to be excellent. The author recommends a lower dose of pentobarbital (10 mg/kg) (18). Ultrashort-acting barbiturates such as sodium thiamylal or sodium thiopental may be used as a single anesthetic agent in a dose of 8 to 11 mg/kg given only intravenously. This dose provides anesthesia for only ten to fifteen minutes.

Inhalation Anesthesia

Indications: Inhalation anesthesia has many advantages (Figures 5.8 to 5.10). With this type, it is possible to control the depth of anesthesia, provide respiratory support, and reduce the risk of toxicity to the patient.

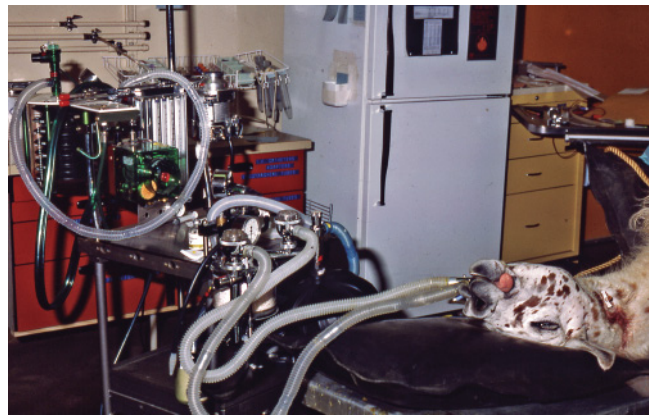

Figure 5.8. Inhalation anesthesia in a llama.

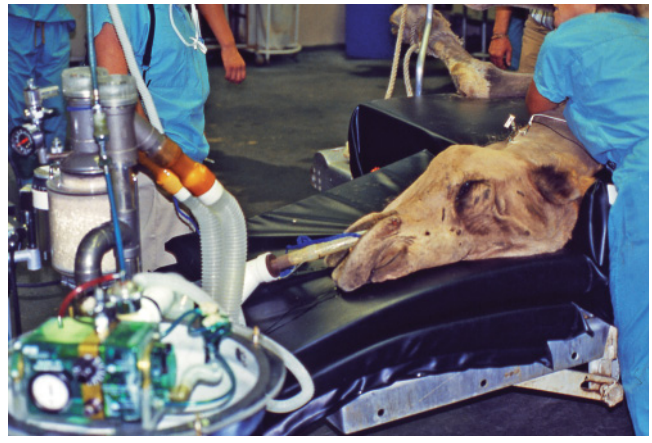

Figure 5.9. Inhalation anesthesia in a camel.

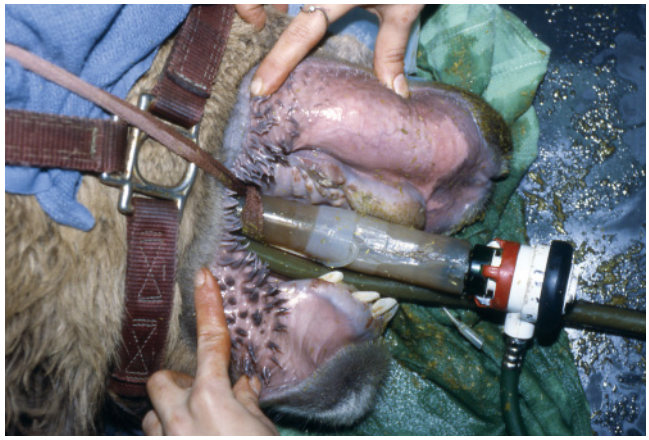

Figure 5.10. Intubation of a camel.

Table 5.6. Inhalant anesthetic agents for camelids.

Generic name	*Commercial name*	*Source*
Isoflurane	Arrane	Anaquest, Madison, WI, 53713
Sevoflurane	Sevorane, SevoFlo	Abbott Laboratories, Chicago IL, 60064
Halothane	Fluothane	Not available, but may be stockpiled

The disadvantages are the cost of the agent, the expense of specialized equipment for administration, and the requirement for an anesthetist.

Equipment: Llamas weighing up to 140 kg (300 lbs.) may be managed on a small animal anesthetic unit with a 5 to 6 L rebreathing bag. The soda lime canister must be monitored constantly to assure that the system is not overburdened with carbon dioxide. Larger animals require a rebreathing bag of up to 78 L in size and a correspondingly larger soda lime system. A large animal anesthetic unit is preferred for larger llamas.

Agents

Isoflurane is the agent of choice, but servoflurane and halothane (if available) are also suitable agents (Table 5.6).

Isoflurane[8,15] (Arranae). Isoflurane has been used in numerous cases in the author's clinic. The flow rates for oxygen are the same as for halothane. An isoflurane vaporizer is required and set at 4.5% for the first five minutes and 1% to 1.5% for maintenance. Induction and recovery is much more rapid with isoflurane anesthesia.

Halothane[7,8–11] (Fluothane). Halothane has been the most commonly used inhalant anesthetic agent. It depresses many cardiopulmonary functions, so the patient should be carefully monitored. The degree of depression is related to the dose or the depth of anesthesia. Functions affected include cardiac output, stroke volume, arterial pressure, and muscle blood flow. The rate and depth of breathing may also be depressed, and respiratory acidosis develops after prolonged anesthesia.

Oxygenation is poorest when the patient is in dorsal recumbency. Hypoxemia is common during the recovery stage, so administration of supplemental oxygen is desirable for ten to fifteen minutes.

Nitrous oxide has been used in combination with other inhalant gases in llamas, but the author does not recommend it because of its propensity to accumulate in obstructed loops of bowel, one of the more common indications for general anesthesia.

Atropine (0.04 mg/kg) may be given, but some anesthesiologists do not use atropine for fear of secondary paralysis of peristalsis.

Physiology

The tidal volume of an adult llama is 10 ml/kg/minute. Oxygen flow rates of 20 ml/kg/minute are recommended for induction. In a controlled ventilation system, this can be reduced to 10 to 12 ml/kg/minute for maintenance (2 L/minute for a 175-kg llama). Flow rates for non-rebreathing systems are three times the foregoing (6 L/minute).

In a study inducing llamas with xylazine and ketamine followed by halothane maintenance, both spontaneous and controlled ventilation were used.[10] After initial induction and endotracheal intubation, general anesthesia was accomplished while the llama was breathing spontaneously by setting the halothane vaporizer at 4.5% for five minutes and at 3% to 3.5% for the remainder of a 120-minute experiment. The actual end tidal alveolar halothane concentration was measured at 1.21% to 1.28%.

A lower concentration of halothane was sufficient when the llama was under controlled ventilation with a respirator. The setting was 3% for the first five minutes and 1.75% to 2.25% for the remainder of the procedure. The end tidal alveolar concentration was 0.68% to 0.72%.

In these same studies, cardiovascular and pulmonary function parameters were measured using intravascular catheters and blood gas analysis. Parameters of cardiovascular function such as the HR, mean arterial pressure, and cardiac output were satisfactorily maintained when the animal was breathing spontaneously, while parameters of pulmonary function such as PaCO2 and arterial pH were markedly altered. Conversely, with controlled respiration, parameters of cardiovascular function changed more. It was concluded that the safest physiologic state was maintained with controlled ventilation.

The mean arterial blood pressure of an unanesthetized llama varies from 130 to 170 mm of mercury (Hg). Intraarterial catheters may be placed in an auricular or saphenous artery. Measurement of blood pressure, using a Doppler unit, can be made over the medial aspect of the distal metacarpus or metatarsus or on the ventrum of the tail. Doppler measurements should be considered only approximations of the actual pressure, but are sufficiently accurate to indicate changes.

During both spontaneous and controlled ventilation halothane anesthesia, arterial blood pressure dropped from 140 to a low of less than 60 mm Hg at approximately one hour into surgery. Then the pressure steadily returned to about 110 after two hours.

Recovery time from halothane anesthesia varies with the physiologic state of the patient. Debilitated animals remain affected longer than vigorous patients. In general, a llama should be able to remain in sternal recumbency within sixty minutes following cessation of administration of the agent and be able to stand within ninety minutes, but may be depressed for several hours.

The body temperature of a llama will drop during general inhalation anesthesia. In experimental two-hour procedures, body temperature dropped from 38.3°C to 36.5°C (100.9°F to 97.7°F). Body temperature did not return to normal for one to three hours after recovery from surgery. These animals were kept in a thermoneutral environment, and no steps were taken to warm the llamas.

SUPPORTIVE THERAPY DURING ANESTHESIA

HR, RR, and body temperature should be assessed periodically during anesthesia. If available, a pulse oximeter attached to the tongue is excellent for monitoring the oxygen status of the patient. Hypothermia may be countered by the application of heating pads to the inner thighs, abdomen (if accessible), and axilla. In extreme cases, administration of warm IV fluids or warm water enemas is indicated.

An IV catheter should be placed and appropriate fluids administered during the procedure. An arterial catheter is desirable if facilities for arterial pressure assessment or blood gas analysis are available.

The head should be positioned to avoid overextension in order that saliva and stomach contents may be appropriately drained. Artificial tears should be instilled into the conjunctival sac and measures taken to protect the prominent eye from positional trauma.

If an injectable anesthetic is used, body and head positions are even more important. If a procedure is prolonged, the patient will benefit from supplemental oxygen, administered by placing a small tube up a nostril at a flow rate of 1 L/minute.

Sophisticated electrocardiographic monitoring is desirable but not required, especially if an anesthetist is continually monitoring the patient.

Anesthetic Support Drugs

Anesthetic support drugs are shown in Table 5.7.

Atropine Sulfate[8]

Pharmacology: Atropine is a parasympatholytic drug with action equivalent to blockage of the parasympathetic autonomic nervous system. It decreases salivation, sweating, gut motility, bladder tone, and gastric and respiratory secretions. Vagal blockage produces tachycardia. Mydriasis occurs.

Table 5.7. Emergency drugs for camelid anesthesia.

Drug concentration	*Dose*	*Indication*	*Volume*			
			20 kg	*100 kg*	*200 kg*	*500 kg*
Atropine LA 2 mg/ml	0.04 mg/kg	Decreased heart rate	0.4	2	4	10
Epinephrine 1:1,000 (1 mg/kg)	0.01 mg/kg	Cardiac arrest, anaphylaxis	0.2	1	2	5
Calcium gluconate 23% 8 mEq/ml 10% 0.43 mEq/ml	0.7 mEq/ml	Hypocalcemia	0.2	1.1	2.2	5.5
Bicarbonate, Na 1 mEq/ml	0.5 mEq/ml	Metabolic acidosis	10	50	100	250
Doxopram 20 mg/ml	0.1 mg/kg	Respiratory depression	0.1	0.5	1	2.5
Lidocaine 20 mg/ml	0.5 mg/kg	Ventricular arrhythmia	0.5	2.5	5	10
Diazepam (Valium) 5 mg/ml	0.1–0.5 mg/kg	Seizures	2	10	20	125
Dexamethasone 4 mg/ml	2 mg/kg	Shock	5	50	100	250

Indications: Atropine diminishes excessive secretions induced by ketamine. It is also commonly used as a preanesthetic medication to prevent reflex vagal stimulation of the heart (cholinergic bradycardia) during induction.

Administration: Large animal formulations are supplied as solutions of 2 mg/ml and small animal formulations as 0.5 mg/ml. Atropine can be given orally or parenterally at dosages of 0.04 mg/kg. Atropinization occurs within one to fifteen minutes, depending on the route of administration.

Side Effects and Precautions: Dilated pupils should be protected from direct sunlight to prevent retinal damage. Atropine is contraindicated for patients with glaucoma.

Antidote: Parasympathomimetic drugs may aid in counteracting the effects of atropine, but atropine is difficult to reverse.

Doxopram HCL[7,23] (Dopram)

Doxopram is a respiratory and cardiac stimulant that may be indicated when an immobilized animal experiences respiratory depression. It is not a reversal agent or antagonist to any drug. Recommended dosage is 1 mg/kg administered intravenously.

Reversal Agents (Antidotes, Antagonists)

Reversal agents are tabulated in Table 5.8.

Diprenorphine[8] (M50-50)

Pharmacology: Diprenorphine is a narcotic antagonist used to reverse the effects of etorphine. It acts as a depressant on the CNS, and if used in excessive dosages, may complicate recovery from etorphine administration.

Administration: Diprenorphine is injected intravenously if possible, otherwise, intramuscularly. The recommended dose is double the injected dose of etorphine. When injected intravenously, reversal occurs within one to four minutes. IM injection may require fifteen to twenty-five minutes to accomplish reversal.

Antidote: Naloxone acts as an antidote for diprenorphine.

Naltrexone HCl[8,15]

Pharmacology: Naltrexone competitively binds to opiate receptors in the CNS, thus preventing both exogenous and endogenous opiates from acting. It is particularly effective in preventing renarcotization when using carfentanil.

Indications: It is one of the more effective opiate antagonists. It is now considered the drug of choice for treatment of accidental injection of opiates in humans.

Administration: May be administered IM, IV, or IP.

Side Effects and Precautions: At usual doses it is relatively free of adverse effects.

Naloxone Hydrochloride[7] (Narcan)[19,21,24]

Pharmacology: Naloxone is a true narcotic antagonist, with none of the agonistic effects that some other narcotic antagonists may produce.

Indications: Naloxone is used solely as an antagonist to narcotic immobilizing agents.

Administration: Intravenous, intramuscular, and subcutaneous routes for injection are satisfactory. Induction time for naloxone is within two to three minutes when injected intramuscularly. The effective dose of naloxone is 0.006 mg/kg. For a human accidentally injected with etorphine or carfentanil, an initial injection of 1 mg of naloxone should be administered intravenously, followed by injections of 0.4 to 0.8 mg every two to four minutes until the victim is under hospital management.

Antagonist: None.

Tolazoline HCl[8,23] (Tolazine)

Pharmacology: Tolazoline is a competitive α_1 and α_2 adrenergic blocking agent. It also relaxes vascular smooth muscles, producing a peripheral vasodilatation.

Indications: In restraint it is used to antagonize α_2 adrenergic agonists (xylazine, medetomidine, detomidine).

Administration: IV or IM.

Side effect and precautions: Transient tachycardia, gastrointestinal hypermotility.

Antagonist: None.

Table 5.8. Reversal agents used to antagonize sedatives and chemical immobilizing agents.

Generic name	*Trade name*	*Reversal for*	*Concentration*	*Dose*
Yohimbine HCl	Antagonil, Yobine	α_2 agonists	5 mg/ml	0.25 mg/kg
Tolazoline HCl	Tolazine	α_2 agonists	100 mg/ml	0.5–5.0 mg/kg
Atipamezole HCl	Antisedan	α_2 agonists	5 mg/ml	300–500 µg/kg
Diprenorphine HCl	M50-50	Narcotic	2 mg/ml	Double the dose of etorphine
Nalorphine HCl	Narcan	Narcotic	0.4 mg/ml	0.006 mg/kg
Naltrexone HCl	Trexan	Narcotic	50 mg/ml	100 × dose of carfentanil
Nalmefene		Narcotic	50 mg/ml	10 × dose of etorphine
Flumazenil	Mazicon	Diazepam, zolazepam	0.1 mg/ml	10 × dose of diazepam

Yohimbine[8,24,25] (Antagonil)

Pharmacology: Yohimbine was retrieved from the discarded drug locker when it was found to have α adrenoceptor antagonist activity and became the first drug to be used for reversing the effects of xylazine. It is relatively nonselective in action and is not effective in all species.

Indications: Yohimbine has been given to numerous species to reverse respiratory depression caused by xylazine, and ultimately to assist in complete recovery of the animal from immobilization. The variable responses of individual species will be addressed in animal group discussions.

Administration: Yohimbine may be administered intramuscularly, but a more immediate reaction occurs with intravenous injection.

Side Effects and Precautions: There are few side effects. The major weakness of yohimbine is failure of effect in some species.

Antagonist: None.

Atipamezole[8,15]

Pharmacology: Atipamezole competitively inhibits α_2 adrenergic receptors and is thus a reversal agent for α_2 adrenergic agonists such as medetomidine, detomidine, and xylazine.

Administration: It should be administered intravenously for optimal response, but may be given intramuscularly in doses four to five times the dose of medetomidine.

Side Effects and Precautions: There are no contraindications for this drug, but it is not recommended that atipamezole be used in pregnant or lactating animals.

Flumazenil[8] (Maloxicam)

Pharmacology: Flumazenil is a competitive blocker of benzodiazepines at receptor sites in the CNS.

Indications: In restraint, it is used as an antagonist for benzodiazepines (diazepam, midazolam).

Administration: IM or IV.

Side Effects or Precautions: Slight pain at the site of injection. Rarely produces seizures with overdose.

EUTHANASIA IN CAMELIDS

Injectable Agents

Toxic barbiturates are the drugs of choice for euthanizing dogs, cats, other small animals, and SACs. Barbiturates have the advantages of being less expensive than many other agents, rapid action, and causing minimal discomfort to the animal. The disadvantages are that these drugs must be administered intravenously or intraperitoneally, and are not readily available except to veterinarians. The cost of barbiturates may be prohibitive for use in an animal the size of an camel.

Because immobilizing agents are now available in all but the most remote locations on the globe, the humane method of euthanasia for camels is initial immobilization with an agent that renders the camel totally unconscious (etorphine, carfentanil, xylazine, detomidine). A catheter should be placed in an accessible vein immediately following recumbency. Incise down to the vein if blood pressure drops or there is a problem with inserting the catheter through the skin. Then a saturated solution of potassium chloride (KCl) should be injected intravenously. The heart stops in a few seconds and death is instantaneous.

Powdered KCl is readily available from chemical companies or a pharmacy without a prescription. It need not be medical grade, and sterility is not necessary. A saturated solution is prepared by adding 180 g of KCl to 600 ml of hot water, which provides 0.3 g/ml of solution. The dose required to stop the heart is 44 mg/kg (20 mg/lb). For instance, to euthanize a 500-kg camel, 6.6 ml of the 0.3 g/ml saturated solution is required. In practice, the solution is usually administered in excess, so it is appropriate to double or triple the quantity of KCl solution to account for possible spillage (15 to 20 ml). This method of euthanasia is humane, suitable for any large domestic or wild animal, and is in compliance with the American Veterinary Medical Association's Guidelines for Euthanasia.[5] The animal must be anesthetized and fully unconscious before injecting the KCl solution.

Alternative Euthanasia

Camelids may also be euthanized by a bullet or a blow to the head in the location illustrated in Figure 5.11.

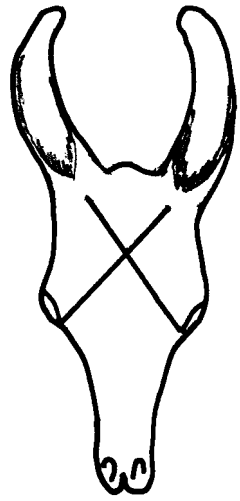

Figure 5.11. Correct placement of a blow or bullet for euthanasia.

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6

Surgery

Surgery in the camelid is fundamentally the same as surgery in livestock. Variations in anatomy that have surgical implications are emphasized in this chapter.[14,35] Though details of every surgical procedure that has been performed or may be performed are not discussed, unique conditions are described, including indications for surgery, pertinent anatomy, clinical signs, diagnosis, presurgical recommendations, anesthesia, positioning, method, postsurgical care, and complications. If appropriate the reader is directed to standard surgical texts.[21,35,56]

HEAD

Abscesses

Etiology

Abscesses of the head and neck are common clinical entities (Figures 6.1A,B). In the author's practice, the organism most often isolated from camelid abscesses is *Arcanobacterium pyogenes*. However, streptococci, staphylococci, bacteroides, actinomyces, *Corynebacterium pseudotuberculosis*, and *Escherichia coli* have also been isolated. It has been suggested that these organisms may be present in the normal flora of the oral mucosa and skin of camelids and opportunistically invade the tissue via a break in the epithelium. Two isolates of *Nocardia asteroides* have been made in the author's clinic.

Pathogens may also be introduced via the hematogenous route, in which case infection is likely to be found in a lymph node. Abscesses may be located in the oral and pharyngeal submucosa, any lymph node of the head and neck region, the salivary glands, or the subcutaneous tissues of the head and neck.

When male llamas fight, they bite the throat and neck. Abscessation may be a sequel to such encounters. Lacerations of the labial mucosa may result from sharp enamel points on the teeth. Grass awns and other harsh forages may traumatize the mucosa, providing a portal of entry for opportunistic bacteria.

Clinical Signs

Signs vary with the anatomic site of the abscess. Swelling usually calls attention to an external abscess (Figures 6.1A,B). Other swellings that may be confused with abscesses include hematomas, tumors, cellulitis, and edema.

Oral or deep pharyngeal abscesses may not be observable without the aid of a laryngoscope or endoscopic equipment. Usually, impairment of prehension or deglutition resulting in anorexia is observed.

Though localized abscesses rarely produce a febrile response or a change in the hemogram, progressive enlargement of a developing abscess may obstruct venous drainage, causing edematous swelling. Some abscesses develop slowly over a period of days or weeks; others mature in two to six days.

The degree of fluctuation of an abscess depends on its stage of maturity and the extent of encapsulation. *Corynebacterium* spp. and streptococcal organisms tend to produce minimal encapsulation, while abscesses caused by *Actinomyces* spp. develop a thick capsule and little lumen.

Abscesses located on the lateral face may impinge on the facial nerve or the parotid salivary duct. Special care must be taken to isolate and reflect these structures from the incision site when an abscess is lanced or extirpated.

Position

Unless the animal is extremely fractious, lancing an abscess can be accomplished in the standing position

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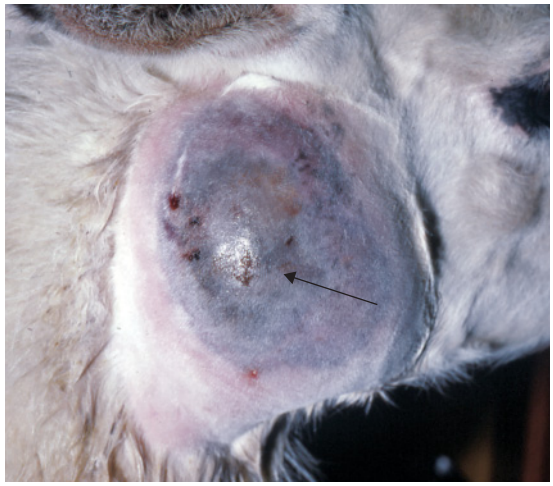

Figure 6.1A. Abscess of the parotid area.

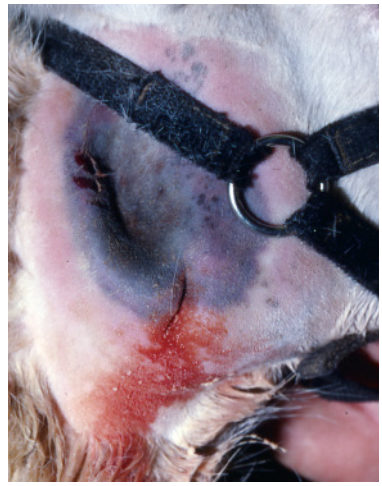

Figure 6.1B. Abscess lanced for drainage.

by using a combination of physical restraint and local anesthesia. The use of a stock is recommended to better control the head, but it should not be employed to overpower the animal or to omit appropriate sedation or anesthesia.

Presurgical Preparation

The area should be clipped and cleansed. Surgical asepsis is superfluous, but it is important to collect the exudate upon opening the abscess. Even though the infective organism may be ubiquitous and opportunistic, the exudate should be collected or drained onto a surface that can be cleansed and disinfected. Gloves should be worn by the operator to prevent self-infection.

Anesthesia

If the abscess has fully matured, to the point of forming a head (producing mucosal necrosis or a necrosed spot of skin), anesthesia is not necessary. If a deep abscess is suspected, infiltration of the intended incision site with a local anesthetic agent is recommended. If the skin is closely adhered to the capsule of the abscess, diffusion of the anesthetic agent may be impeded or prevented. If cellulitis has developed, the local anesthetic agent may be neutralized by a change in pH of the inflamed site. If extirpation of the abscess and its capsule are anticipated, especially in the region of such vital structures as nerves, vessels, or salivary glands, sedation or general anesthesia is indicated.

Surgery

Surgery is frequently preceded by aspiration with a diagnostic needle to confirm the presence of an exudate or other fluids. The point of insertion of the needle should be selected to avoid vital structures and should be as ventral as possible. The needle should be left in place while a vertical incision is made through the skin, alongside the needle. Further penetration may be accomplished by blunt dissection with either a finger or Mayo scissors, inserted closed but opened as they are withdrawn. If the capsule is thick and fibrous, it may be necessary to stab with a scalpel guided by the shaft of the needle. This incision should be small enough to allow insertion of a hemostat or needle forceps to bluntly enlarge the incision to avoid the risk of lacerating a vessel or nerve.

The lumen of the abscess should be explored with forceps or finger. The orifice should be enlarged sufficiently to establish good drainage. If the initial incision site lies far dorsal, it may be appropriate to establish a more ventral second drainage site, again avoiding vital structures.

The exudate may be viscous or inspissated to the degree that it must be removed by curettage. The lumen should be irrigated thoroughly with dilute povidone-iodine solution. Acute abscesses may require no further treatment to induce healing. Chronic abscesses with thick capsules may produce an inner abscess membrane that inhibits healing, similar to the inner lining of a fistula. In such cases, the lumen of the abscess should be treated with a caustic solution, such as 7% tincture of iodine, to destroy the inner lining and allow healing to commence.

Extirpation is possible only if the abscess is accessible, with a well-developed capsule and no surrounding cellulitis. For this procedure, surgical asepsis is desirable so that the wound may be closed following surgery.

Postsurgical Care

The wound should be irrigated with a disinfectant solution once a day until granulation tissue fills the lumen and it is no longer possible to insert an irrigator.

If two incisions were made, a seton may be placed using 5 or 7.6 cm (2 or 3 in.) gauze. This keeps the incisions open, and irrigation should be continued. An ointment such as zinc oxide should be applied to prevent maceration of the skin ventral to the incision.

Antibiotics are not indicated for either nonsurgical or postsurgical management of localized abscesses. Though the action of antibiotics may slow development of an abscess, it will rarely halt such development. Encapsulation prevents penetration of the abscess by parenterally administered antibiotics, and locally applied antibiotics are immediately washed out by the exudate. An exception is an abscess caused by *Actinomyces* spp. (as yet unnamed but unique to SACs). These are likely to reseed themselves and reappear after supposed healing has taken place. If this organism is isolated or suspected, a six-week course of parenteral penicillin or other antibiotics to which Gram-positive organisms are sensitive may be necessary to effect healing.

Healing must proceed outwardly by granulation from within. Once this begins, the process should not be inhibited by excessive cautery. The lumen may be kept free of exudation by maintaining patency and irrigation.

Complications

Multiple abscesses may be seeded in an area in various stages of development. Initial lancing of a large abscess may miss some of the smaller seeds, which will continue to develop. If incisions are too small or they close prematurely, the abscess may recur. The most serious complication is damage to vital contiguous structures, either as a result of the inflammatory process or from trauma during surgery.

Lacerations[16]

Lacerations may occur on the lips, cheeks, face, body, or limbs. A large male camelid may bite a smaller male, inflicting lacerations anywhere on the head, ears, neck, or scrotum. Aggressive males in a frenzy to reach an opponent may traumatize themselves on fences. Lacerations may also result from dog bites (Figures 6.2A,B). Severe disfigurement as a result of dog bites has occurred; e.g., the lower lip has been stripped away from the mandible.

Restraint or transport of camelids may result in lacerations from sharp obstructions. The tongue may be lacerated by sharp enamel points of the teeth or by rough manipulation when the tongue is pulled out for tracheal intubation.

Presurgical Preparation

The wound area should always be clipped or shaved and cleansed as thoroughly as possible. Clipping tends to deposit short fibers in the wound unless a moist compress is applied over the laceration before begin-

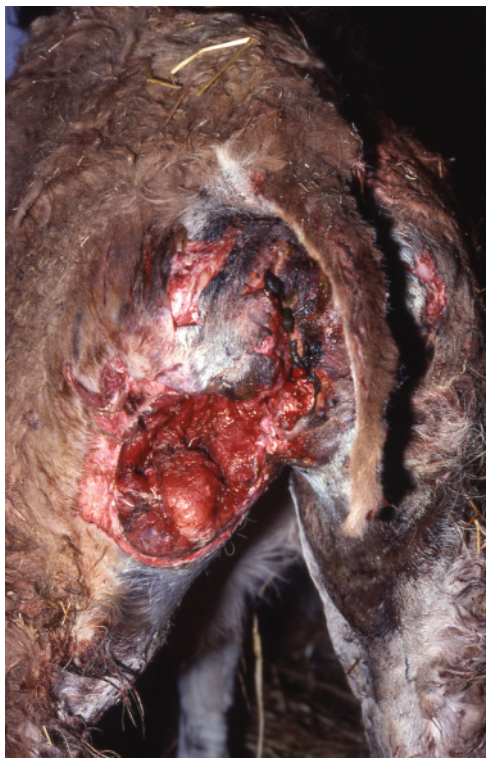

Figure 6.2A. Laceration from a dog bite.

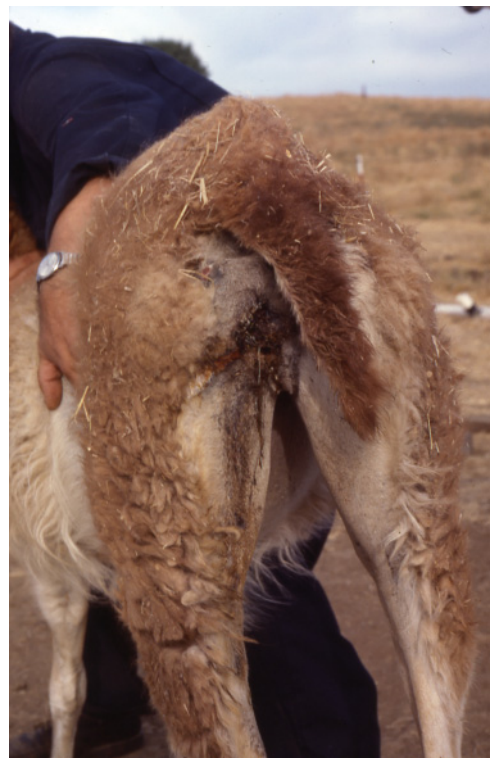

Figure 6.2B. Healed dog bite laceration.

ning. Alternatively, lubrication jelly may be applied to the wound.

Anesthesia

Once the wound is cleansed, subcutaneous infiltration along the margins of the laceration with a local anesthetic agent usually provides sufficient analgesia. General anesthesia may be necessary to repair multiple lacerations or lacerations of the lips or eye structures.

Surgery

Debridement of a wound is a basic essential surgical procedure for removing foreign material or devitalized tissue from the wound. The wound should be irrigated with povidone-iodine solution, diluted 1:10. A water pick may be improvised, using a 20– to 35-ml syringe and a 22– to 25-gauge needle. A pulsating spray can be achieved by periodic pressure on the plunger. The spray penetrates the recesses of the wound and flushes out particulate matter.

The skin of camelids is relatively thicker than that of other species; thus, infolding is not a serious problem. However, there is less flexibility in camelid skin, and it is more tightly adhered to the underlying structures than in other species, making reconstructive surgery more difficult. Tension sutures may be used as appropriate.

An attempt should be made to replace the torn gingiva and labial mucosa when repairing an injury in which the lower lip has been stripped away from the mandible. Little gingival tissue may be left to which the labial mucous membrane may be reattached. If this is the case, a nonabsorbable suture may be laced around the roots of the incisors to form a latticework to which sutures may be attached. If the wound is fresh and thoroughly cleansed and debrided, the subcutaneous tissue may heal to the periosteum and minimize the potential defect from such a laceration.

Postsurgical Care

Antibiotics are indicated only if vital structures (such as articulations) are exposed; in puncture wounds (as a precaution against tetanus); or when devitalized tissue must be left in a wound for fear of traumatizing vessels, nerves, or ducts during debridement. Otherwise, routine postsurgical care should be followed.

Choanal Atresia[11,13,24]

Choanal atresia is a congenital defect wherein there is either a membranous or osseous separation of the nasal and pharyngeal cavities at the level of the choanae. The condition may be unilateral, bilateral, partial, or complete. The paired choanae are separated by the caudal border of the vomer bone. Embryologically, the nasal cavity forms from a caudad-directed invagination of the nasal membrane and the rostrum of the embryo. A corresponding invagination moves forward from the pharyngeal pouch. The two cavities are separated by the buccopharyngeal membrane, which ultimately ruptures in normal embryologic development.

Other nasopharyngeal deformities may be associated with choanal atresia. The nasal bones may be shortened or situated abnormally close to each other, narrowing the nasal passageway. There may be total absence of the nasal cavity and nostrils.

Complete bilateral choanal atresia is a life-threatening condition. Neonate camelids are obligate nasal breathers. They are able to breathe through the mouth with some difficulty, but when doing so, they are unable to nurse. Without surgical intervention, the neonate will either starve or develop aspiration pneumonia.

It is not possible to state with assurance that choanal atresia is hereditary; however, there are indications that heredity is a factor. Therefore, surgery should not be performed on a neonate destined for breeding. At the present, surgical correction is indicated only for allowing affected animals to reach adult status and be used for experimental breeding to establish whether or not the condition is hereditary.

Clinical Signs

Respiration is labored. In the characteristic breathing pattern, upon inspiration, the mouth opens slightly, air is drawn in, the lips close, and the cheeks puff out. The air in the oral pharynx is then forced around the caudal border of the elongated soft palate into the larynx and trachea. Expiration is prolonged and labored because air must again be forced around the soft palate, in reverse.

With complete choanal atresia, no air movement through the nares is possible. It is common for air entrapped in the nasopharynx to be swallowed, resulting in gastric tympanitis. The affected neonate fails to gain weight and weakens rapidly.

Diagnosis

Plain film radiography may be suggestive, and definitive diagnostic radiographs can be made by depositing 5 to 10 ml of a radio-dense liquid, such as Renografin or barium sulfate, into the nasal cavity with a 3– to 5-French catheter. The llama should be placed in sternal recumbency, with the head elevated, so that the contrast medium may accumulate in the caudal nasal cavity before making radiographs.

Anesthesia

General anesthesia is required.

Surgery

A tracheostomy must be performed before attempting correction of the atresia. The junction of the hard and soft palates should be palpated with an index finger, and with the other hand, a 3.5– to 6-cm (1/8– to 1/4-in.) trocar-tipped intramedullary pin is inserted into the nostril through the ventral meatus, with the tip directed toward the finger within the mouth. Resistance will be felt as the tip of the pin meets the membrane. If an osseous plate is present, resistance will be strong, but the pin should be pushed through the plate. The pin may be felt in the nasopharynx through the soft palate.

A silastic tube must replace the intramedullary pin to maintain patency while healing. In one technique, a silastic tube with an inside diameter slightly larger than the pin is lubricated internally, threaded over the pin, and pushed into the nasopharynx. Then the pin is withdrawn.[21]

Alternatively, the original pin is withdrawn and inserted into a silastic tube of a slightly greater diameter than the pin and reinserted within the silastic tube sheath. When the tube is positioned properly, the pin is withdrawn. In either case, the tube must be sutured to the ventromedial aspect of the nostril and cut off flush to prevent the tube from catching on objects during feeding. The process is repeated on the opposite side. The tubes must be left in place for three to four weeks to allow epithelium to cover the newly created orifices. The tracheostomy must be maintained to allow breathing until the tubes can be removed.

Another approach to the obstructed choanae is via a midline trephine opening through the nasal bones directly dorsal to the choanae.[a,21] The choanal area is visualized through the nasal cavity on either side of the nasal septum. The choana is penetrated with a scalpel and the orifice enlarged using rongeur bone forceps.

Complications

The prognosis for such surgery is guarded to poor. The risk of aspiration pneumonia is high. The procedure has not been standardized. The defect is highly variable and is often accompanied by secondary defects that may complicate recovery. Even if surgery is successful, the nasal openings may be less than optimum for adequate air passage.

DENTAL DISEASE

Dental anatomy is addressed in Chapter 13. That section should be studied before considering dental surgery.

Malocclusion

Malocclusion describes abnormal positioning of a tooth or teeth that prevents appropriate fitting and wear with corresponding teeth in the opposite jaw. Both superior and inferior brachygnathism frequently occur in llamas and alpacas (Chapter 22), resulting in either elongated incisors or excessive pressure and wear on the dental pad.

Causes

Malocclusion may be caused by trauma to the teeth, the bones supporting the teeth, or the temporomandibular articulation.[39] Chronic pain in any of these tissues may result in chewing abnormalities, which in turn cause asymmetric wear of the teeth and, ultimately, malocclusion.

A more frequent cause of malocclusion in South American camelids is a congenital anomaly: shortening of the premaxilla, lengthening of the mandible, or a combination of both. Bustinza and Jahuira[9] and Sumar[49] believe that these are hereditary conditions (Chapter 22). A third cause may be fluorine poisoning, which causes softening of the enamel and uneven wear of the teeth.

Clinical Signs

It is easy to determine whether or not the incisors are aligned. In the normal animal, the incisors fit against the rostral tip of the dental pad. It is more difficult to ascertain occlusion of the cheek teeth. Gross abnormalities may be palpated through the buccal membrane. SACs have a narrow mouth that cannot be opened widely enough to allow clear visual inspection without great difficulty. A thorough inspection of the mouth necessitates sedation or anesthesia.

Sharp enamel points on the buccal surface of the upper and lingual surface of the lower cheek teeth are normal in SACs. This condition should not be confused with malocclusion. However, if either the upper or lower jaw is shifted rostrally in relation to the other, the end cheek teeth will not wear against their opposites and will become elongated.

Animals with malocclusion suffer from varying degrees of dysfunction in prehension and chewing. Severe malocclusion may result in malnutrition and poor body condition. Neonates with severe malocclusion are unable to nurse.

Indications

Radiographic evaluation of the dental structures may help in making a diagnosis. A detailed film series, including oblique studies, is required to visualize each jaw without superimposition of the opposite jaw.

The condition should also be evaluated in terms of potential genetic transmission. Congenital malocclusion should be viewed as a hereditary defect until proven otherwise, and such animals should not be included in breeding herds. It is unethical for a veterinarian to surgically correct such a defect on an animal

intended for sale as a breeding animal. Otherwise, appropriate action should be taken to correct interference with prehension and chewing.

Position

SACs do not tolerate oral examination or submit to dental work without resistance. It may be possible to file off a small tooth projection with the unsedated animal in a standing position, but most dental work necessitates sedation, recumbency, and, perhaps, general anesthesia. Lateral recumbency is the recommended position.

Anesthesia

As appropriate.

Surgery

Minor dental elongations may be leveled using standard equine dental floats. The enamel points on the cheek teeth should not be leveled as in the horse. If a cheek tooth has been lost, with corresponding elongation of the opposing tooth, the elongated tooth should be shortened. Teeth at both ends of each dental arcade should be checked carefully and leveled to the rest of the arcade.

A Hauptner bovine dental speculum, set on the narrowest opening, is suitable for holding the mouth open for inspection and surgery. A narrow wooden block, inserted between the cheek teeth of the jaw opposite to the jaw being corrected, makes a satisfactory substitute for a speculum.

Elongated incisors may be cut off with obstetric wire, a hacksaw, a Stryker orthopedic saw, or a circular saw mounted on a hobby drill unit (Dremel Mototool). A Gigli surgical wire saw is too fragile and will usually break during the procedure. In a correctly trimmed incisor, the pulp cavity is not opened and the nerve is neither exposed nor stimulated. However, the nerve is affected by the vibration and heat generated during the cutting, making this a painful procedure that should not be performed without administration of appropriate analgesia.

It should be reiterated that the incisor teeth of the vicuña and, to a lesser extent, the alpaca, continue to erupt until late adulthood. Incisor malocclusion precludes even wear, resulting in a chronic problem of overgrowth of the incisors.

Retained Deciduous Incisors

In the normal eruption process, the permanent tooth bud begins development at the base of the deciduous tooth and pushes it out as eruption progresses. The permanent tooth bud for a single tooth, or for each of the incisors, may develop in an abnormal position and fail to push out the deciduous tooth, resulting in a double set of incisors.

Clinical Signs

A double set of teeth is readily apparent. The permanent set is on the lingual surface (Figure 13.23, Chapter 13). The deciduous teeth are usually smaller, are worn on the occlusal surface, and may be loose in the jaw, attached only by a short root.

Anesthesia

Usually sedation and analgesia are all that are necessary.

Surgery

Retained incisors may be so loose in the jaw that they can be removed with the fingers. If not, the attachment of the gingiva to the tooth should be loosened with a dental elevator. The alveolar periosteal membrane may also be broken down with the elevator, if necessary. Damage to the attachment of the permanent tooth is a hazard. The tooth may be grasped with small animal dental forceps and rotated gently on its vertical axis while exerting slight traction. As the tooth loosens, the degree of rotation and the traction should be increased until the tooth is extracted.

Postsurgical Care

The alveolar sockets may be left open. Hemorrhage is usually minimal, and neither antibiotics nor disinfectants are recommended for use in the mouth. The mouth may be inspected for two to three days following surgery to remove any feed material that is trapped in the socket. Otherwise, the wounds should be allowed to heal by second intention. The animal usually continues to eat, with no evidence of discomfort.

Disarming the Canine Teeth[26]

All of the camelids have canine teeth, which are especially well developed in the male. It is a common practice in North America to blunt these teeth in some manner to avoid severe lacerations of the ears, throat, limbs, and scrotum when two males fight.

The essential anatomy of the canine teeth is described in Chapter 13. Additional anatomic considerations for surgery follow: (1) The most ventral curve of the mandibular canine is immediately adjacent to the mental foramen and nerve. Manipulation in that area should be avoided. (2) The alveolus of the third incisor is immediately adjacent to the rostral border of the mandibular canine. Rough elevation and leverage may damage the root of the incisor. Roots of the canine are located rostral to the caudal border of the mandibular symphysis (Figure 6.3). This site provides added structural strength for applying leverage to the canine or when it is necessary to remove the bony plate. Even here, application of too much force may

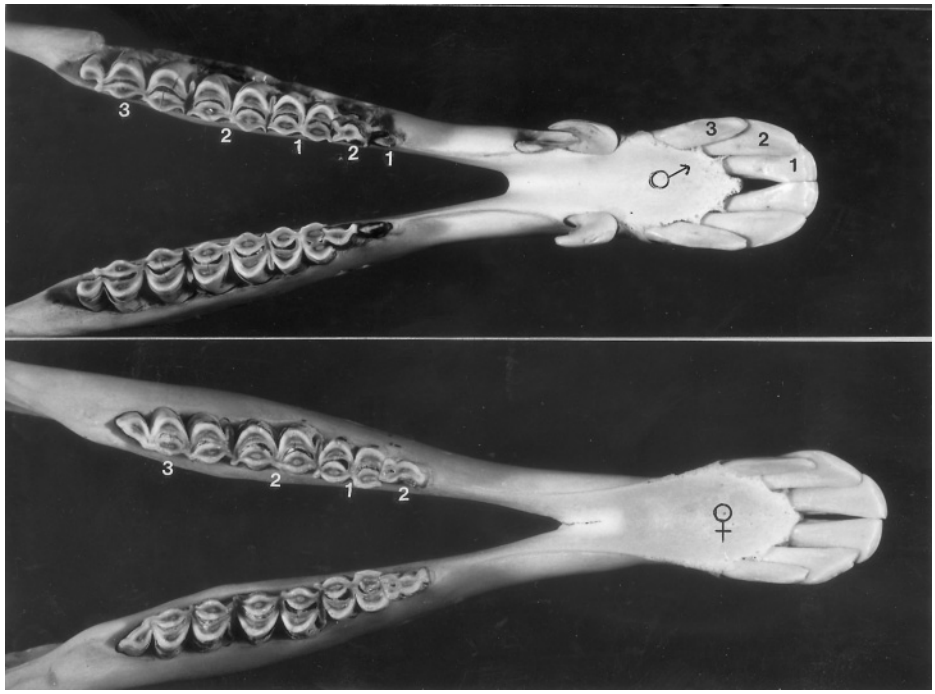

Figure 6.3. Inferior cheek teeth, lower incisors, and lower canine teeth of a male and female llama, dorsal view.

fracture the mandible or cause separation of the symphysis.

Two methods are recommended to disarm canine teeth: total extraction, cutting off the crown above the gingiva, and cutting off the crown at the mandible and covering the stub with the gingiva. The advantages and disadvantages of each of these alternatives are described. Other procedures have been reported by Kock.[22]

Extraction

INDICATIONS. This procedure is not recommended for routine disarming because the roots of the canines are curved and large (Figure 6.4). However, a fracture of the tooth and subsequent infection of the pulp cavity may necessitate extraction. Extraction is also indicated if a pulp cavity abscess has developed following removal of the crown.

SURGERY. The gingiva should be incised rostrally and caudally to the crown. A second incision should be made diagonally from the crown toward the root apex. The gingiva may be reflected from the bone and the crown with a dental elevator or chisel.

A plate of bone should then be removed from the lateral aspect of the upper canine tooth root and the dorsolateral aspect of the mandibular canine. The bony plate may be carefully chiseled off or cut with a surgical saw. Once the plate is removed, the root may be separated from the alveolus with a dental elevator.

The crown should be grasped with a small animal dental forceps and a gentle rocking motion, medial to lateral, used to further loosen the tooth. It is not possible to rotate canine teeth on the vertical axis because of the curved root. The gentle elevation and forceps movements should be continued until the tooth is released. This process should not be hurried, because some time is required to loosen the root. Excessive torque may fracture the root, complicating complete extraction.

The extracted tooth should be closely examined to ascertain that the entire root has been removed. Any spicules of the alveolar plate or fractured segments of the root should be removed with a small curette.

Cutting Off the Crown Above the Gingiva

INDICATIONS. This procedure requires the least amount of time, instrumentation, and materials. It is especially applicable when it is desired to blunt the tip of the canines while they are still erupting. Also, if the deciduous canines are particularly long and sharp and the young male is overly aggressive with pen mates, the tips of these teeth can be blunted with this technique.

ANESTHESIA. The procedure has often been performed by the breeder by securing the head in a stock or over a yoke and cutting off the teeth without anesthesia. Omitting anesthesia may be acceptable if no more than the tip is to be cut off, but if the tooth is to be cut off near the gingiva, the pulp cavity and nerves will be exposed. It is not justifiable to subject an animal to that degree of pain simply because the animal can be physically overpowered.

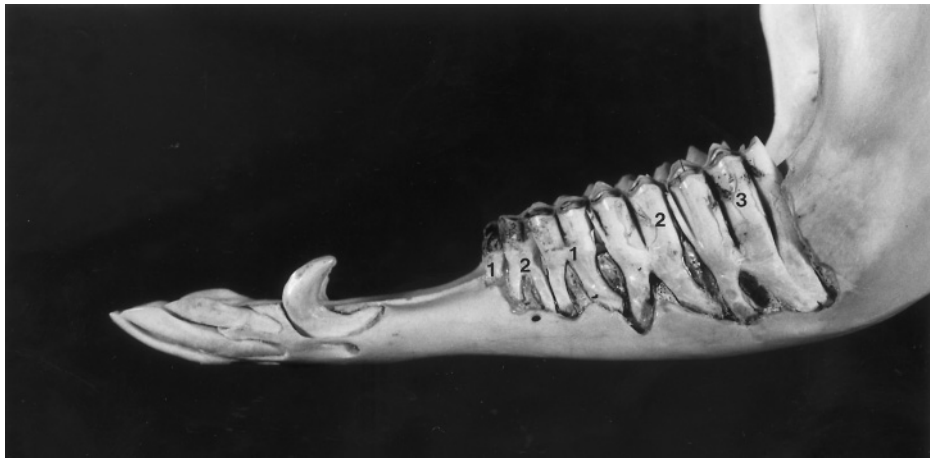

Figure 6.4. Sculptured mandible exposing the roots of the cheek teeth, incisors, and canine tooth of a male llama, lateral view.

Intravenous (IV) xylazine HCl (0.4 mg/kg) will sedate the animal and provide analgesia for the short time necessary to complete the surgery. Butorphanol (0.1 mg/kg) may also be used.

SURGERY. The mouth should be held open with a speculum. Obstetric wire is usually used to cut off the tooth; however, a circular saw on a hobby drill is also suitable. The cut should begin 2 to 3 mm above the gingiva. A rapid sawing motion with the wire generates heat, searing the pulp cavity as the crown is removed. Less pressure should be exerted on the wire as the cut nears completion to avoid fracturing the edge of the tooth.

If only the tip is cut off, the pulp cavity will not be exposed. With a deeper cut, exposing the pulp cavity and development of pulpitis and/or apical abscess are risks, but hundreds of llamas have been operated on by owners and veterinarians using this technique with only a few reports of postsurgical infection. Apparently, the pulp cavity usually seals itself, with a dentine layer forming quickly at the exposed surface. Nonetheless, if the pulp cavity and nerves are exposed, discomfort is associated with this procedure. No postsurgical care is necessary.

Anatomy of the Cheek Teeth

Superior Cheek Teeth

Each upper arcade has one or two premolars and three molars (Figure 6.5). These are frequently designated as "cheek teeth" and are numbered 1 to 5 cranial to caudal. Cheek tooth 1, when present, is small and triangular shaped (1 × 1 × 1 cm), with three roots and no infundibulum, but the occlusal surface is folded on its caudal border (Figure 6.6).

Cheek tooth 2 is rectangular (1.5 × 1 cm), with one medial and two lateral roots and one infundibulum. The roots of this tooth are immediately ventral to the infraorbital foramen.

Cheek tooth 3 (molar 1) is rectangular (2 × 1.5 cm), with four roots and two infundibuli. Cheek tooth 4 is the largest tooth in the mouth (2.5 × 1.5 cm), rectangular in shape, with four roots and two infundibuli. Cheek tooth 5 is slightly smaller than the fourth but also has four roots and two infundibuli. The roots of the upper molars (cheek teeth 3 to 5) lie within the maxillary sinus.

Inferior Cheek Teeth of SACs

The inferior cheek teeth are narrower than the superior cheek teeth, and the mandibular arcades are closer together. In a normal resting position, the upper arcade extends approximately 1 cm labially to the lower arcade, and the lower arcade extends 3 to 4 mm lingually to the upper arcade. This fosters the development of sharp enamel points on the lingual surfaces of the lower cheek teeth and the buccal surfaces of the upper cheek teeth. Development of such points is normal, and unless they become exaggerated or cause labial lacerations, the enamel points need not be floated.

The inferior cheek teeth have the following characteristics (Figures 6.3, 6.4, 6.7, 6.8): lower cheek tooth 1 (premolar 1), if present, is conical in shape (0.8 × 0.5 cm), with two roots lying closely together and no infundibulum. Cheek tooth 2 (premolar 2) is triangular (1 × 0.6 cm), with two divergent roots and no infundibulum. Cheek tooth 3 (molar 1) is rectangular (1.7 × 1 cm), with two roots and two infundibuli. Cheek tooth 4 (molar 2) is rectangular (2.4 × 0.2 cm), with two divergent roots and two infundibuli. Cheek tooth 5 (molar 3) is rectangular (3 × 1.2 cm), with three roots, the caudal two fused, and two infundibuli.

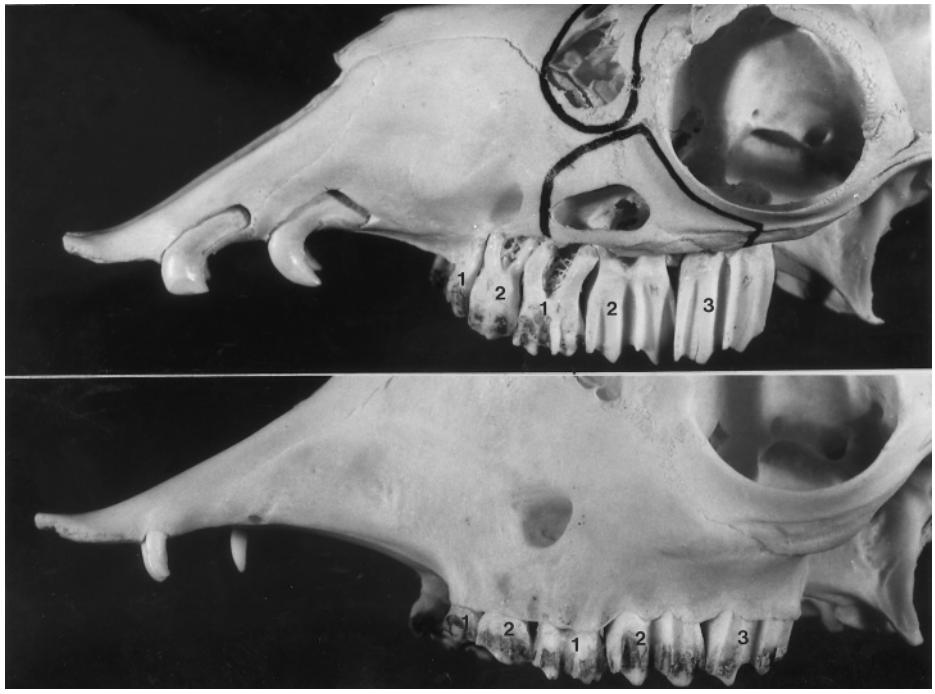

Figure 6.5. (Top) Sculptured upper jaw exposing the roots of the canine teeth and superior cheek teeth, lateral view. (Bottom) Canine teeth and maxillary cheek teeth, lateral view.

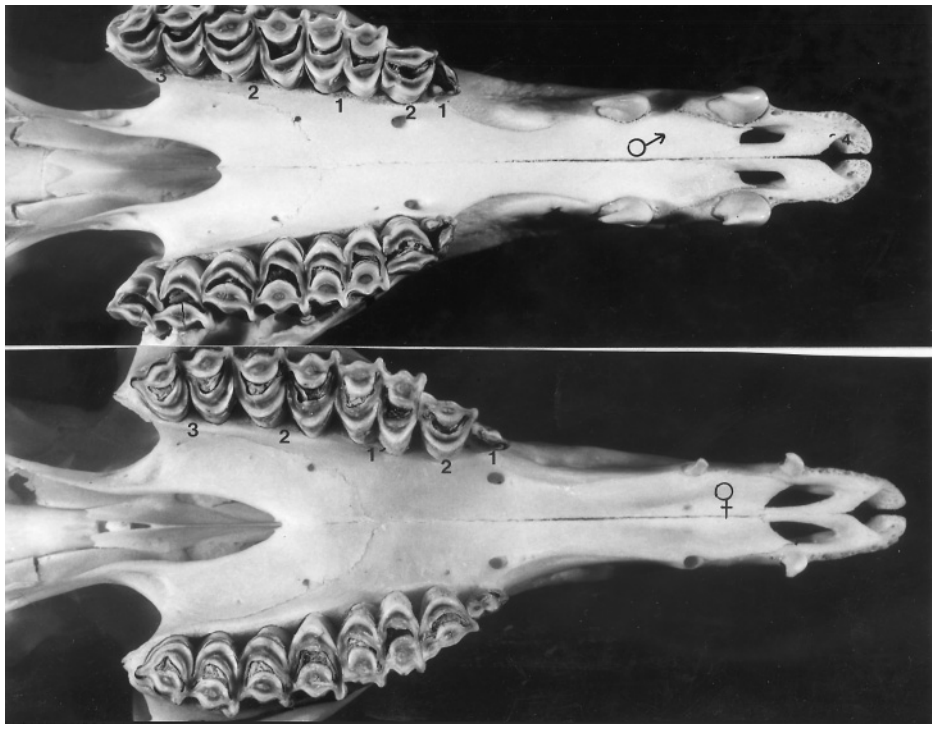

Figure 6.6. Superior cheek teeth and canine teeth, ventral view.

Infection of the Cheek Teeth (Premolars and Molars)

Pulpitis and alveolar periostitis or osteitis may result from infection of the alveolar space through a break in the gingiva, fracture of a tooth exposing the pulp cavity, or decay within the infundibulum. A compound fracture of the mandible or maxilla may also provide a portal of entry for infection. Various species of opportunistic bacteria may be

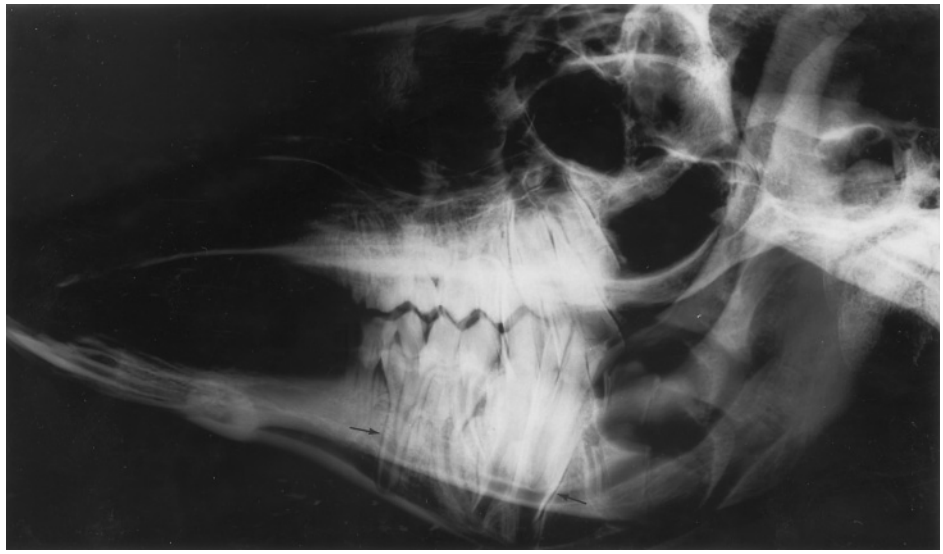

Figure 6.7. Lateral oblique radiograph of a mature normal llama mandible.

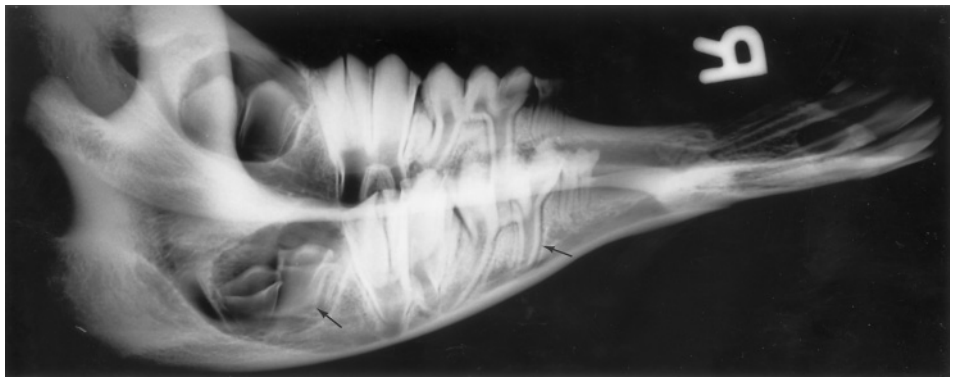

Figure 6.8. Lateral oblique radiograph of an immature llama mandible with erupting molars.

involved, but *Actinomyces* spp. is common in the author's practice.

Clinical Signs

The classic signs of an infected tooth are swelling over the root, retraction of the gingiva from the crown, and reluctance to eat or peculiar chewing behavior, indicating an attempt to avoid pressure on the tooth. With involvement of the upper molars, a malodorous unilateral nasal discharge, smelling like necrotic bone, will emanate from the maxillary sinus.

Chronic periosteal infection of the inferior arcades usually proceeds to osteitis. Ultimately, a fistulous tract will break through to the exterior, with exudation from the ventral border of the mandible. This exudate also has an odor like that of necrotic bone. Any drainage from the ventral border of the mandible should be considered to be associated with a dental infection until proven otherwise.

Diagnosis

A detailed examination of the cheek teeth via the oral cavity necessitates sedation or anesthesia. Although clinical signs may be suggestive, a definitive diagnosis should be based on radiographic evaluation. Radiographs must be of high quality, and right and left oblique views must be taken so that roots in both arcades can be evaluated without superimposition of the roots of the opposite arcade (Figures 6.7 to 6.11).

Anesthesia

General anesthesia is required and inhalant anesthesia is preferred, even though an endotracheal tube diminishes the space available for oral manipulation. With a cuffed endotracheal tube in place, there is less chance for aspiration of blood from the oral cavity or ingesta from regurgitation. If IV anesthesia is used, an IV catheter must be placed for the administration of

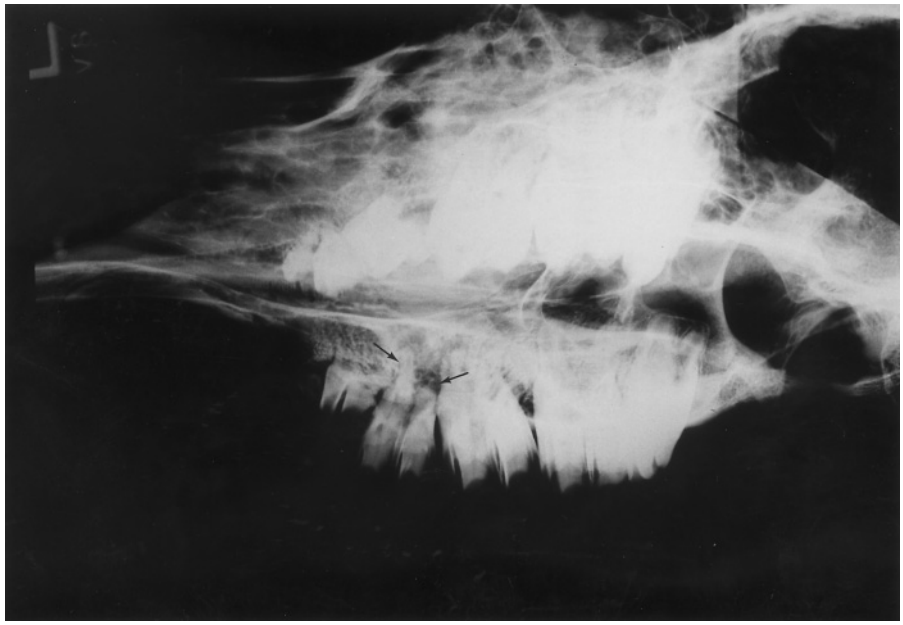

Figure 6.9. Lateral oblique radiograph of maxillary alveolar periostitis.

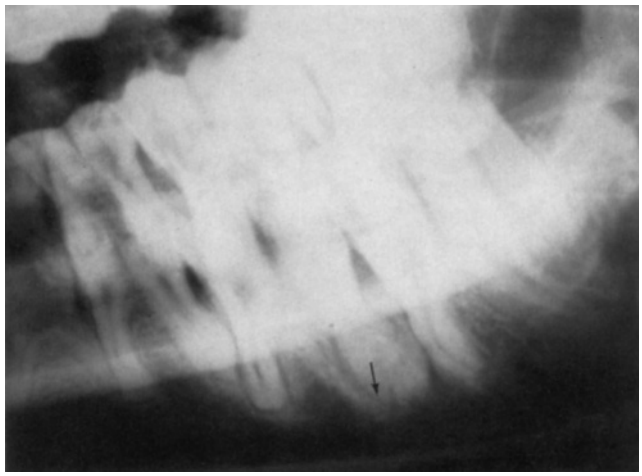

Figure 6.10. Lateral oblique radiograph of mandibular alveolar periostitis.

supplemental doses of anesthetic agent, should it be necessary. Controlling the depth of anesthesia is difficult with IV agents, and inhalation of foreign material is a possible complication.

Surgery

Endodontic procedures are usually not indicated to save infected camelid teeth; however, recently surgeons have been performing root canal procedures and longitudinal transection of a tooth to remove only the infected root. If a definitive diagnosis of an infected tooth is made, the usual therapy is to remove the tooth, by extraction or repulsion.

Extraction of Cheek Teeth

Dental surgery in SACs presents special challenges. The mouth does not open widely, and the interarcade space is narrow. Further, these animals are too small for the efficient use of equine dental instruments and too large for the effective use of instruments designed for small animal dentistry. Both sizes may be required.

Both upper and lower cheek teeth 1 are accessible and may be loosened with a dental elevator and grasped with a small animal right-angle dental forceps to rock the tooth medially and laterally or to rotate the tooth on its vertical axis, beginning with minimal torque. When the tooth loosens, a slooshing noise may be heard as the alveolar membrane begins to break down. Tooth extraction is a laborious process and should not be hurried, lest with the leverage of an equine extractor, the crown be broken from the root.

To extract cheek tooth 2, the mouth should be held open with a dental speculum (a Hauptner bovine speculum works well) and the gingiva separated from the crown with a sharp dental pick or scalpel. The tooth may be grasped with a small animal right-angle dental forceps and manipulated as described for tooth 1.

Extraction of the remaining teeth is difficult. The extractor occupies nearly all of the space between the opposing jaws, even with the mouth fully opened, leaving little room to apply sufficient leverage to pull a tooth from its socket. If the tooth is loose, it may be possible to manipulate the forceps to either the labial or lingual side of the opposing arcade and to apply slight tension. Otherwise, rotation must be continued until the entire alveolar membrane has been broken

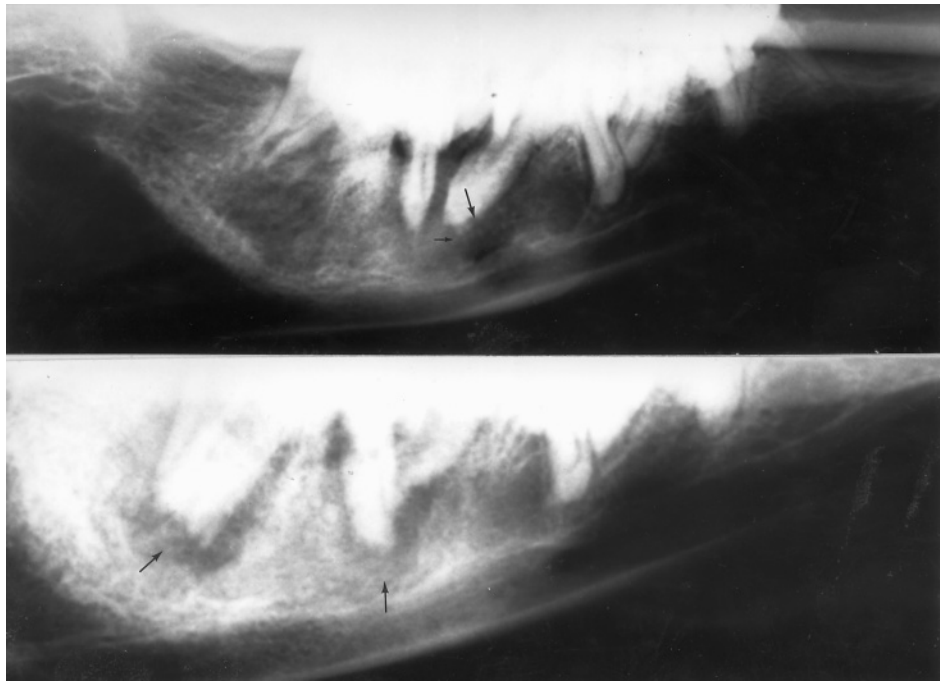

Figure 6.11. Lateral oblique radiograph of mandibular alveolar periostitis.

down and the tooth may be lifted from the socket with fingers or a small canine forceps.

Repulsion of the Cheek Teeth

A trephine opening for repulsion of the upper teeth should be made just dorsal to the facial crest and below a line scribed from the medial canthus of the eye to the infraorbital foramen. This places the operative site ventral to the osseous lacrimal canal and the canal for the infraorbital nerve. Even with the trephine opening properly located, it is possible to damage the osseous canal of the infraorbital nerve, because it lies between the medial and lateral roots of the molars. The punch must be carefully placed over the lateral aspect of the roots.

The third upper molar (cheek tooth 5) is extremely difficult to remove. Extraction is impossible because of space limitations. The roots lie within the maxillary sinus but so far caudad that the alveolus for the tooth forms the ventral floor of the orbital socket. Repulsion can be done only through a small trephine opening through the malar (zygomatic) bone just ventral to the facial crest and at the lowest point of the orbit. It is necessary to separate the masseter muscle from the facial crest to make the trephine opening.

Once the anatomic site for the trephine hole is located, a circular incision the approximate size of the trephine should be made in the skin. Muscles, blood vessels, nerves, and the parotid duct should be reflected and the incision continued through the periosteum. The parotid duct enters the buccal cavity at the level of the junction between upper cheek teeth 2 and 3. Caution must be exercised to avoid injury to the duct when operating in this area.

A 13- or 16-mm (1/2- or 5/8-in.) Galt trephine is generally used, but a 9-mm (5/6-in.) Michelle trephine may also be satisfactory. The center bit of the Galt trephine should be extended 2 mm past the circular saw to fix the trephine to the bone. As the trephine is swiveled back and forth, a circular groove is cut into the bone. The center bit may then be retracted and the cut through the bone continued to remove a disc. Excessive pressure on the trephine should be avoided to prevent fracturing the bone, especially when opening a sinus. If a trephine is not available, a window can be cut in the bone over the root of the tooth with a small osteotome, sterilized wood chisel, or bur on a surgical drill.

A speculum should be used to open the mouth to identify the affected tooth. The gingiva may be cut from the crown of the tooth with a sharp dental pick, curved sharp bistoury, or curved scalpel blade. The affected tooth should be grasped with the thumb and forefinger of one hand and the punch directed with the other hand. It may be necessary to improvise the punch. It should be about 6 mm in diameter. The punch should be carefully placed over the root(s) of the tooth encased in the alveolar bone plate. A mallet is used to strike the punch gently to break down the alveolar bone. As solid pressure is exerted on the tooth, the

vibrations will be felt on the tooth crown within the mouth. It is important to align the punch with the vertical axis of the tooth. The punch should be struck with gradually increasing intensity until the tooth begins to erupt past the table surface of the arcade. Thereafter, gentle strikes of the punch will finish repelling the tooth.

The removed tooth must be examined to ascertain the completeness of the repulsion. Any remaining segments of the alveolar plate or roots should be removed with a curette or small bone chisel.

The mandible of the camelid is fragile. It is not uncommon to cause a fracture of the mandible during the repulsion process. An alternative is to make a vertical incision through the skin over the long axis of the affected tooth. Once an incision is made through the skin, the facial artery and nerve traversing the incision site at a right angle must be identified. Reflect the vessels and nerve before incising through to the oral cavity. A plate of bone is lifted from the lateral surface of the affected tooth. From this position, the affected root may be visualized and the tooth transected if desired. A dental elevator may be used to loosen and repel the tooth from the alveolar plate.

Postsurgical Care

The socket must be plugged to prevent feed from packing into the site and inhibiting healing. Traditional postsurgical care is to insert a gauze pack into the tooth cavity and tie it to another gauze pack at the external trephine opening. This technique has been supplanted by plugging the socket with methylmethacrylate, hoof acrylic, or dental impression material (Optisil) applied at the time of the initial surgery.[25] In this method, wire is placed around the teeth contiguous to the socket and a cross strut is strung between the wires to help form a latticework to support the acrylic. The softened acrylic is placed in the defect between the teeth and extended approximately 5 mm past the gingiva into the socket. The hardened acrylic plug prevents feed penetration and allows granulation to fill in the socket. Irrigation and drainage of the socket is accomplished through the trephine opening. The pack will ultimately be pushed out by the granulation tissue.

Complications of Trephination and Tooth Repulsion

Trephination and repulsion are not without hazard. Improper placement of the punch may result in penetration of the hard palate; damage to contiguous tooth roots; fracturing of either the medial or lateral aspect of the mandible or maxilla; and/or damage to the infraorbital nerve, facial nerve, nasolacrimal duct, or parotid salivary duct.

Tracheostomy

Indications

Camelids are primarily nasal breathers; thus, any lesion causing obstruction of the nasal or nasopharyngeal airways produces dyspnea and may warrant a tracheostomy. Surgery performed in the region of the nasal cavity, such as for correction of choanal atresia (Chapter 22), requires that a preoperative tracheostomy be done first. Oral surgery may necessitate tracheal intubation through a tracheostomy to provide room in the oral cavity and visualization of the surgical site. Rattlesnake bites on the muzzle require first aid insertion of a short tube into the nostril of the victim or tracheostomy to prevent suffocation. Surgical repair of tracheal stenosis has been described.[27]

Position

The surgeon should be prepared to perform this surgery in all conceivable positions (standing, sternal, lateral, or dorsal recumbency).

Presurgical Preparation

In an elective tracheostomy, standard surgical asepsis should be practiced. A site on the ventral midline of the cranial third of the neck should be prepared with sufficient fiber clipped off to preclude the remaining fiber from dangling over the orifice of the tube. In a heavily fleeced llama or alpaca, this may require feathering away from the site.

Anesthesia

An emergency tracheostomy may be performed on a semiconscious or unconscious patient without anesthesia or surgical preparation. In an elective tracheostomy, either infiltration of the incision site with local anesthesia or sedation with xylazine HCl is appropriate.

Surgery

To avoid hemorrhage, the incision should be made on the midline. The paired sternohyoideus muscles should be separated by blunt dissection. The underlying fascial layer may be separated with blunt Mayo scissors or incised with a scalpel. Care should be taken to avoid scoring the tracheal rings.

The trachea of the llama or alpaca lies deeper within the cervical structures than it does in ruminants or horses. The external jugular veins, common carotid arteries, vagosympathetic trunks, and esophagus (on the left side) are in close apposition to the trachea. Nonetheless, if strict adherence to a midline approach is used, hemorrhage will be minimal.

The trachea should be opened by incising the annular ligament between two tracheal rings. Avoid

incising more than half the circumference. Additional space for insertion of a tube may be obtained by removing a half-moon-shaped segment from adjacent rings, but the rings should not be transected.

Various types of tracheostomy tubes may be inserted; the most satisfactory are those consisting of an outer sleeve and an inner tube that may be removed for cleaning. The inside diameter of the trachea is approximately 1 cm in a neonate SAC and 3 cm in a 175-kg adult SAC. The size of the camel trachea is larger, allowing the use of equine tracheostomy tubes. The procedure followed at this point is determined by the type of endotracheal tube chosen and the objective of the surgery. For long-term placement, the skin should be sutured around the tube, and the tube itself may be sutured to the skin. For short-term placement, the incision may be left open.

Postsurgical Care

It is extremely important to monitor the patency of the tube at frequent intervals to prevent obstruction from exudate or mucus. Exudate or dried blood and mucus should not be pushed into the trachea. When the tube is no longer needed, it should be removed and the wound allowed to heal by granulation. The wound should be debrided twice daily with a povidone-iodine-soaked gauze sponge until healing is complete.

Tracheostomy tubes should be removed, cleaned, and replaced as often as necessary to maintain patency. Alternatively, the tube can be cleaned by suction.

Soft Palate Surgery

All camelids are obligate nasal breathers because of an elongated soft palate that may cover the glottis during deglution or be positioned beneath the epiglottis to allow inhalation of air. A congenital condition of the head region is a cleft palate. Surgical repair of this defect has been reported, but is rarely done.

Both male and female dromedary camels have a unique diverticulum on the ventral aspect of the soft palate. The diverticulum (dulaa, dulla, dulah) is more highly developed in the male and may be inflated and protruded from the oral cavity during rut, or when excited or angry (Figure 6.12). Bactrian camels do not have a dulaa.

Indications for Surgery

Several conditions of the dulaa may require surgical intervention including lacerations, hematomas, erosions, ulcerations, necrosis, impaction, and paralysis. The dulaa may be lacerated by bites from other combative males or from its own canine teeth while the dulaa is protruded. If the dulaa remains protruded for a prolonged period of time it may be traumatized by objects in the enclosure or dry out and become infected or necrotic. The dulaa may become impacted with feed, preventing protrusion.

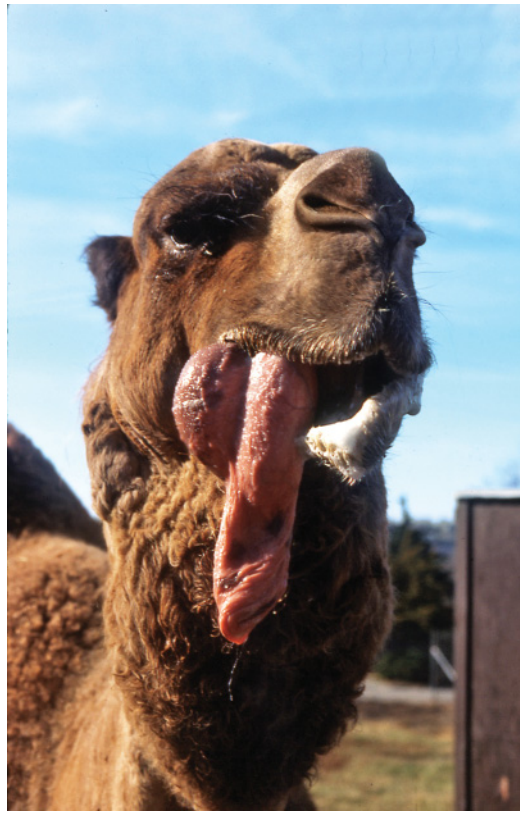

Figure 6.12. Dulaa of a male dromedary camel.

The dulaa may be amputated in racing camels because of real or perceived interference with respiration while racing.

Clinical Signs

Signs of trauma that may be observed when the dulaa is protruded include erosions, ulcers, hematomas, and necrosis. Impaction interferes with swallowing feed and drinking water. Large impactions may interfere with breathing. The swelling may be observed or palpated upon oral examination. Impaction may cause a swelling in the throat region that is visible or palpable.

Management

Superficial lacerations and erosions may be managed with routine wound care. More serious conditions may necessitate amputation. Several methods for amputation of the dulaa are described in the literature.

Presurgical Preparations

The camel should be heavily sedated and maintained in sternal recumbency, preferably with a body rope harness in place to prevent the camel from rising

during the procedure. Some surgeons prefer to have the camel under general anesthesia.

The mouth is held open with a Hauptner cattle speculum or other speculae used for livestock. The oral cavity should be lavaged with water and finished with a disinfectant solution such as chlorhexidine or povidone-iodine solution. A tracheostomy should be performed to prevent interference with respiration while working in the oral cavity.

Four methods are described for amputation of the dulaa. In each of them, visible blood vessels should be ligated or cauterized by electrocautery.

1. Pull the dulaa from the mouth and spread the tissue. Cut the dulaa close to its attachment to the soft palate with a curved Mayo scissor. The wounds are left open to heal by second intention.
2. Clamp the tissue close to the soft palate with a large Carmalt forcep or an angiotribe. Incise on the distal side of the forcep with a scalpel. Leave the clamp in place for a minute. Then release the clamp and replace it on the next segment to be incised. Repeat the process until the entire dulaa is amputated.
3. The dulaa is stretched maximally and incised with a long Mayo scissor. Vessels must be ligated to prevent hematoma formation following closure. The margins of the mucous membranes are approximated and closed using a Cushing infolding suture pattern.
4. Impactions may be managed by mechanical evacuation of the impacted material. In severe cases it may be necessary to use a blunt hook to pull the dulaa forward to allow amputation.

Postsurgical Care

The administration of a broad-spectrum antibiotic such as long-acting tetracycline is recommended. The oral cavity should be irrigated with dilute hydrogen peroxide, chlorhexidine solution, or povidone-iodine solution for three to five days. Feed soft feeds for two weeks.

CELIOTOMY (LAPAROTOMY)[16]

Anatomy

To perform abdominal surgery, the anatomy of the abdominal musculature must be understood for both NWCs and OWCs. The cutaneous trunci muscle is limited to the specialized preputial muscles in the male and a few fibers on the ventral midline in the female. The abdominal tunic (deep fascia) is less well developed in SACs than in the horse or cow. It is comprised of fibroelastic tissue that adds support to the muscles and their aponeuroses. The tunic covers the external abdominal oblique muscle dorsally and cranially and is an integral part of the aponeurosis of the external oblique muscle ventrally and caudally. The abdominal tunic extends into the inguinal area and helps to form the superficial inguinal ring.

A specialized section of the abdominal tunic becomes the medial suspensory ligament of the mammary gland. The suspensory ligaments of the mammary gland are paired, but the right and left halves are not as easily separated at surgery as they are in cattle.

The external abdominal oblique muscle is the most superficial of the abdominal muscles. Its fibers are directed caudad to slightly ventrad. This muscle originates on the caudal border and lateral aspect of the last few ribs and the fascia over the intercostal muscles. The body of the muscle is comprised of short fibers and is approximately 1 cm thick. An extensive aponeurosis inserts onto the tuber coxae, prepubic tendon, and linea alba.

The internal abdominal oblique muscle lies beneath the external abdominal oblique muscle (Figure 6.13). Its fibers are directed ventrad, craniad, and mediad. This muscle has its origin on the tuber coxae and the deep lumbar fascia. The body of the muscle is comprised of short fibers and is 1 to 2 cm thick. Ventrally and cranially an aponeurosis blends with the aponeurosis of the external oblique muscle. These combined aponeuroses (abdominal tunic, internal and external abdominal oblique muscles) insert on the caudal border of the last rib and the linea alba and are the main fibrous support for the abdominal organs. They form the outer sheath of the rectus abdominis muscle.

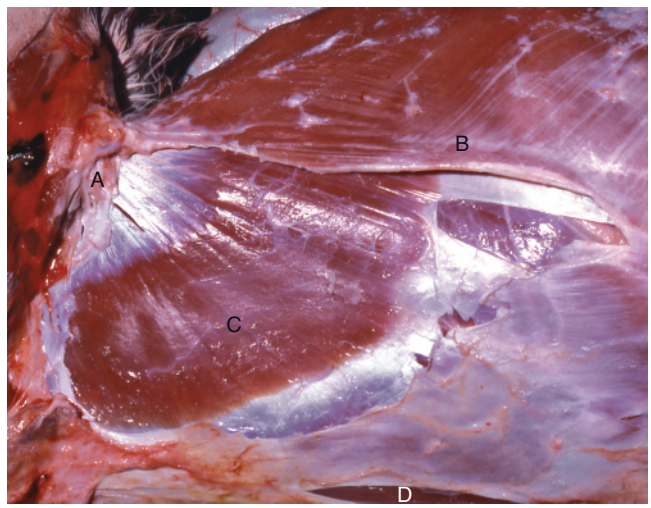

Figure 6.13. Position of the internal abdominal oblique muscle. (A) Tuber coxae, (B) external abdominal oblique muscle, (C) internal abdominal oblique muscle, (D) rectus abdominis muscle.

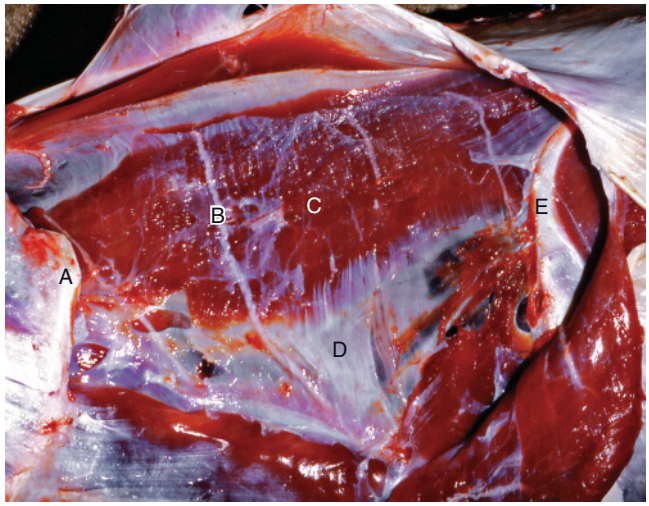

Figure 6.14. Position of the transversus abdominis muscle. (A) Tuber coxae, (B) lumbar spinal nerve, (C) body of the transversus muscle, (D) ventral aponeurosis of the transversus muscle, (E) costal arch.

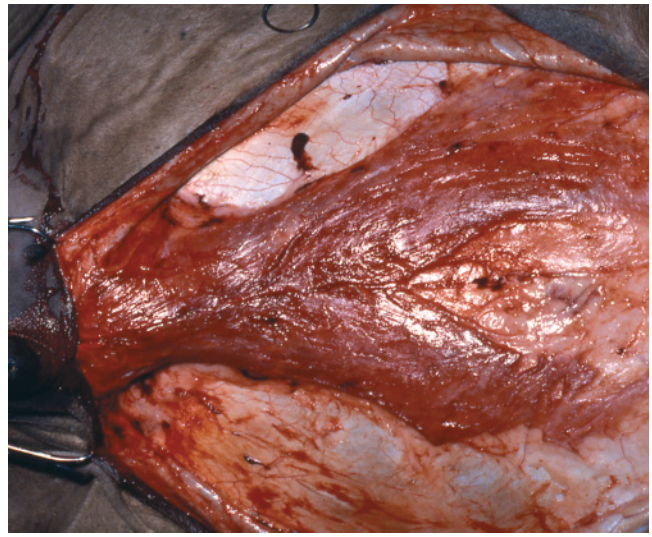

Figure 6.15. Preputial muscles of the llama.

The fibers of the transversus abdominis muscle are directed ventrad and mediad (Figure 6.14). The aponeuroses of the transversus muscle lie dorsally and ventrally. The dorsal aponeurosis originates in the deep lumbar fascia and the transverse processes of the lumbar vertebrae and the costal arch; it is 10 cm long cranially and 4 cm long caudally. The length of the body of the muscle is only 9 cm, continuing on as the ventral aponeurosis, which inserts on the linea alba deep to the rectus abdominis muscle.

The abdominal wall of the camelid has unique features. There is no fold of skin from the lateral abdominal wall to the limb, above the stifle (flank). The paralumbar fossa is not prominent. The only muscle fibers over the area bordered by the last rib, the transverse processes of the lumbar vertebrae and the tuber coxae, are those of the external abdominal oblique muscles at the cranial aspect and some of the internal oblique muscle fibers caudally. Immediately dorsal and lateral to the rectus abdominis muscle, caudal to the costal arch, there is no muscle fiber, only aponeurosis. Therefore, in the camelid, this area is not suitable as a site for an abdominal incision.

In the female camelid, the mammary gland is situated cranial to the pubic attachment of the rectus abdominis muscle. A caudal ventral midline incision to perform an ovariohysterectomy necessitates elevation and reflection of the gland caudally to allow adequate exposure.

In the male camelid, the prepuce and penis are located in a similar area. The external preputial orifice of a 175-kg male is located 15 cm cranial to the rim of the pelvis. The prepuce opens caudally in a sexually unaroused male. The prepuce is under the control of two pairs of muscles that are modifications of the cutaneous trunci muscle (Figure 6.15). The cranial preputial muscle pulls the preputial orifice forward when the male is aroused sexually. The caudal preputial muscle pulls the preputial orifice caudally as arousal subsides.

It is necessary to transect one of the cranial preputial muscles when making a ventral midline approach to the caudal abdomen for cystotomy. The muscle segments must be accurately re-apposed and sutured during closure to avoid subsequent malfunction.

The umbilicus is located slightly cranial to the midpoint of the distance between the xiphoid cartilage and the pubis. The linea alba is indistinct in the llama, particularly caudal to the umbilicus.

Position

A ventral midline approach requires dorsal recumbency. Although elaborate surgical tables are employed in university and private clinics, a simple cradle may suffice to hold a llama in the correct position (Figures 6.16, 6.17). The llama may be lifted onto an elevated platform composed of bales of straw to avoid back strain in the surgeon from bending over and working on the floor. Bales of hay or straw may also be used to brace the llama, but they have the disadvantage of diminishing exposure.

The llama should be in lateral recumbency for a high-abdominal-wall approach.

Presurgical Preparation

Feed should be withheld for twenty-four to thirty-six hours and water for twelve hours prior to elective surgery on an adult SAC. Feed and water should not be withheld from neonates. Precautions should be

Figure 6.16. Cradle used to restrain llama in dorsal recumbency.

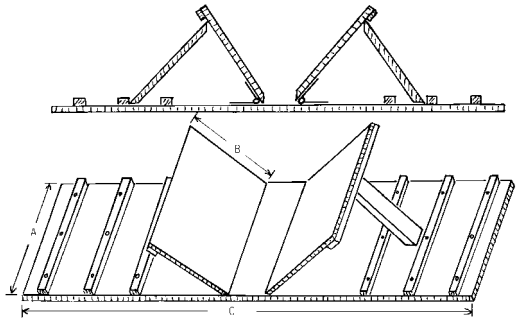

Figure 6.17. Diagram of camelid cradle. (A) 30 cm, (B) 38 cm, (C) 125 cm.

taken to deal with regurgitation, which is common when the patient is in dorsal recumbency but which may also occur in other positions.

Anesthesia

General anesthesia is usually necessary.

Midline Approach[57]

The tissue layers penetrated vary with the site of the incision. Cranial to the umbilicus, the layers encountered are the skin, subcutaneous areolar tissue, a single layer of fascia comprised of the aponeuroses of the abdominal muscles, deep abdominal fat, and the peritoneum.

Caudal to the umbilicus, the layers found are the skin, subcutaneous tissue, cutaneous trunci muscle, superficial fascia (aponeuroses of the external and internal abdominal oblique muscles), deep fascia (thin aponeurosis of the transversus abdominis muscle), deep abdominal fat, and peritoneum. Lateral to the midline, the two fascial layers are separated by the paired bodies of the rectus abdominis muscle. The incision must be made directly on the midline to avoid incising the muscles. In addition, deep abdominal fat will complicate entrance if the incision is off the midline.

Closure also varies with the incision site. Cranial to the umbilicus, the suture layers include the peritoneum, fascia, and skin. Caudal to the umbilicus, it is important to ensure inclusion of all the fascial layers into one or more suture layers.

The body of the rectus abdominis muscle is usually included in the suture of the deep fascia, but it should be understood that the fascia is the tissue that provides support for the abdominal wall.

The author prefers placement of a cruciate (Figure-8) pattern tension suture, using sutures of number 2 polyglactin 910 (vicryl) on the fascial layers (Figure 6.18). Postsurgical dehiscence and herniation are more frequent complications with the more caudal midline incisions than when alternate surgical sites are selected.

Vicryl or gut is suitable for buried sutures. Any nonabsorbable suture or polyglactin 910 is suitable for suturing skin.

Mid- to High-Flank (Paralumbar Fossa) Approach

Indications

The paralumbar approach is recommended for first compartment gastrotomy from the left side. A vertical incision should be made midway between the tuber coxae and the last rib and 6 to 8 cm ventral to the transverse processes of the lumbar vertebrae. If maximal intraabdominal exposure is required, the horizontal fibers of the external abdominal oblique muscles must be incised. The internal oblique and transversus muscles are separated longitudinally in the direction of their muscle fibers. The deep abdominal fat layer, which should not be too thick at this location, may be bluntly dissected and the peritoneum picked up with thumb forceps. The peritoneum should be held taut while it is penetrated with a digit or Mayo scissors.

The peritoneum may be closed with either a continuous or simple interrupted pattern made with absorbable sutures. If a grid incision was made, the muscle layers may be closed with simple interrupted sutures. If the muscle masses were incised, they should be accurately apposed and the outer muscle fascial layer included to provide more strength for the suture to optimize healing.

Other Approaches

A third site for a laparotomy incision is cranial and slightly ventral to the tuber coxae. This approach is indicated for simple ovariohysterectomy or intraabdominal orchidectomy. A diagonal incision should be made 6 to 10 cm ventral to the tuber coxae, ventrad and craniad in the direction of the fibers of the internal abdominal oblique muscle. The fibers of the external

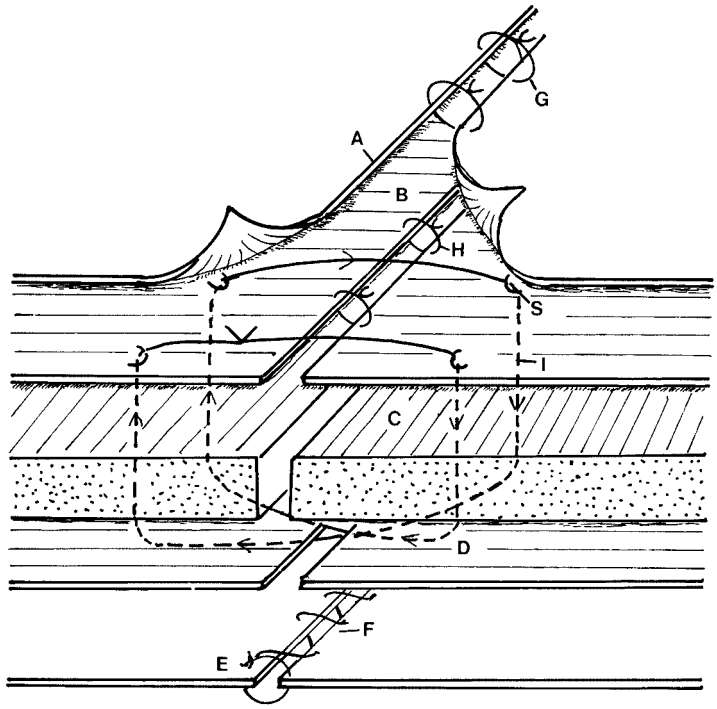

Figure 6.18. Diagram of the placement of a cruciate (Figure-8) suture. (A) Skin, (B) superficial fascia, (C) rectus abdominus muscle, (D) deep fascia, (E) peritoneum, (F) continuous suture to close peritoneum, (G) interrupted sutures to close skin, (H) interrupted sutures to close superficial fascia, (I) cruciate suture pattern, (S) suture.

abdominal oblique muscles are minimal or absent at this site. The incision will penetrate the abdominal tunic, fibers of the internal abdominal oblique muscle (blunt dissection), aponeurosis of the transversus abdominis muscle, and peritoneum. Closure should follow the same sequence as for the high-flank approach.

Another approach for ovariohysterectomy is termed "parainguinal" and advocated by surgeons at Colorado State University.[41] The animal is placed in lateral recumbency with the pelvic limb flexed and abducted. A 10– to 15-cm skin incision is made approximately 8 cm cranial and dorsal to the inguinal canal. Sharp dissection is continued to the internal abdominal oblique muscle, which is blunt dissected in the direction of the long axis of the muscle fibers. The transversus abdominis muscle and peritoneum are penetrated in the plane of the muscle fibers. This approach is similar to the grid approach used in the horse. The uterus and ovaries are identified and withdrawn from the incision for evaluation and/or amputation.

Herniorrhaphy

All types of hernias that occur in other species have been seen in SACs, but umbilical hernias occur most frequently. Although not yet proven in SACs, a congenital umbilical hernia is likely to be of genetic origin. Appropriate recommendations should be made to owners who ask about breeding affected individuals.

Temporary patency of the abdominal wall around the umbilical vessels is normal in many neonate SACs, but this ring should be closed within a month after birth. Usually, the tip of a finger can be inserted through the ring. Rings allowing insertion of two or more fingers or persisting for longer than a month are unlikely to close and should be regarded as hernias. Herniorrhaphy may be delayed for two to three months until the abdominal wall has become firmer to provide better retention for sutures.

Ventral hernias may be caused by trauma. One male adult llama eviscerated itself on a metal post while rearing in an attempt to reach another male. Such hernias may be complete, as in this case, or result only in a rent through the muscle and fascial layers.

Iatrogenic hernias occur from failure of suture lines to hold following laparotomy. Although rare, postcastration inguinal herniation may be prevented by transfixion ligation of the entire spermatic cord.

Anesthesia

General anesthesia is appropriate for this surgery.

Surgery

Techniques, including clamps, appropriate for this surgery in large domestic animals are applicable to SACs. All of the methods illustrated in Figure 6.19 have been successfully employed. If the defect is less than 4 cm in diameter, the peritoneal cavity should not

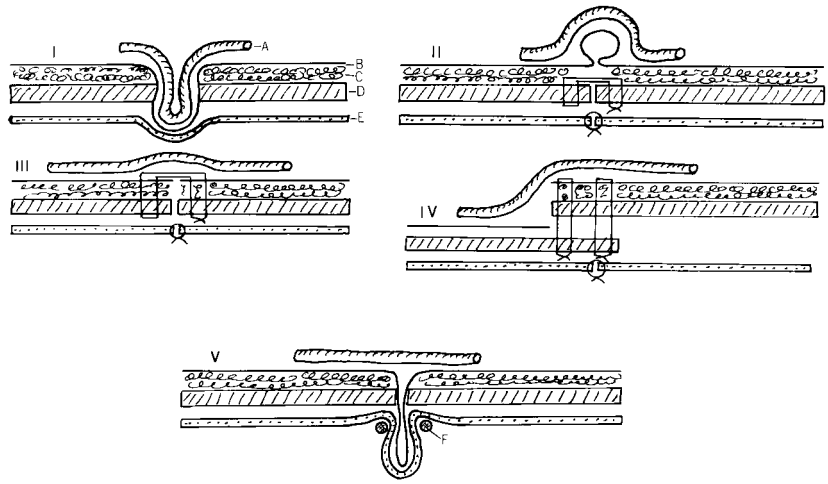

Figure 6.19. Diagrams of closure techniques used in herniorrhaphy. I. Intestinal loop in hernial sac, II. closure without entering peritoneal cavity, III. apposition of muscle-fascial ring, IV. overlap of abdominal wall, V. clamp closure. (A) Intestine, (B) peritoneum, (C) deep abdominal fat, (D) muscle and fascial layers, (E) skin, (F) clamp or constricting band.

be opened; the edges of the ring in the abdominal musculature should be scarified and mattress sutures placed to approximate the edges of the ring.

If the size of the ring is 5 to 10 cm, the closure illustrated in Figure 6.19(I) is recommended. The hernial sac may be excised with caution to avoid adhered viscera. The author has rarely chosen the technique depicted in Figure 6.18(IV), because there is usually insufficient slack in the abdominal wall to allow pulling one side over the other. However, this technique provides the most security against recurrence.

Incarceration of a loop of bowel complicates the surgery. Reference to standard surgical texts is suggested for methods of dealing with this rare complication.

The organ most likely to be found immediately adjacent to an umbilical hernia in the adult llama is compartment one (C-1) of the stomach. Although the greater omentum is small in camelids, it is present at the region of the umbilicus and may protrude into the hernial sac. In neonates and nursing infants, the small intestine may occupy the ventral most position, and it is possible for a loop of bowel to pass into the hernial sac.

Inguinal hernias are less common than umbilical hernias in SACs.[43,60] Techniques recommended for cattle or swine are satisfactory for repair.

Iatrogenic hernias present varying problems unique to each situation. It may be necessary to deal with infection, adhesions, lack of tissue orientation, or fragmented tissue. Incorporation of a teflon mesh is recommended to close a defect when it is impossible to appose the abdominal wall. If possible, heavy tension sutures should be placed, anchored well lateral to the incision line. Number 2 polyglactin 910 placed in a cruciate pattern is recommended. If appropriate, the suture may be doubled. Some or all of the sutures should be placed loosely before tightening. Excessive tension on individual sutures may be avoided by pulling several sutures taut at the same time, minimizing the chance that a tension suture may pull out. A second row of simple interrupted sutures of gut or vicryl should be placed between the tension sutures to make a tighter closure.

It is not necessary to attempt to close the peritoneum when correcting an iatrogenic hernia. Subcutaneous fascia should be approximated, as appropriate, to eliminate dead space. Finally, the skin may be closed with either simple interrupted or vertical mattress pattern sutures. Absorbable vicryl or nonabsorbable sutures are appropriate for closing the skin.

Diaphragmatic hernia has been reported in llamas and camels. Description of the technique for surgical correction of an operable diaphragmatic hernia is beyond the scope of this book. Standard surgical texts should be consulted.

Postsurgical Care

Generally, antibiotics are not indicated for postsurgical care of a routine herniorrhaphy. In iatrogenic or complicated hernial repair, administration of antibiotics is necessary.

During recovery, the animal should be kept where the footing is secure to avoid slipping, stretching, or falling. An attendant may soothe the animal to keep it in sternal recumbency until all effects of the anesthesia have dissipated.

An elastic body support bandage (Elastoplast) should be applied to the abdomen of any camelid having a midline or high-flank celiotomy. A tempo-

rary support bandage, applied while the animal is recumbent, may provide security while it is gaining its feet. In the recumbent animal, relationships of body surfaces are different than in the standing animal, so a permanent support bandage should be applied only while the llama is standing. The abdomen of a llama narrows toward the rear, making it difficult to keep a support bandage in place. Vetrap tends to gather, making it unsuitable. A clean piece of muslin or bed sheeting laid over the top of the fleece before application may prevent difficulty in removing the elastic bandage.

While healing, the llama's activity should be restricted to a small, clean enclosure or box stall. Some movement should be encouraged to aid in the dispersement of edema.

DIGESTIVE SYSTEM

Esophageal Fistula

An esophageal fistula may be performed experimentally to collect swallowed feed, may occur from a laceration of the cervical area, or may develop following prolonged esophageal obstruction and necrosis of tissue through the skin.[15]

Gastrostomy

A gastrostomy is indicated to remove foreign objects (hair balls, concretions, pieces of plastic) from C-1 or to relieve a concentrate overload. Experimental gastrostomy is performed to study gastric physiology.[42] The immediate emptying of C-1 may be crucial to the recovery of a llama that has consumed oleander, Nerium oleander (Chapter 23), or other toxic substances.

This surgery is usually performed under general anesthesia with a ventral midline approach, but it is also possible to perform a C-1 gastrostomy via a left paralumbar fossa approach. This procedure is similar to a rumenotomy in a cow.

Llamas have a tendency to lie down when they are in pain. The llama may be quieted with butorphanol (0.05 to 0.1 mg/kg intramuscularly) and the incision site desensitized with local anesthetic, using an inverted L block or local infiltration.

A 15-cm incision should be made in the left flank beginning 15 cm (6 in.) ventral to the transverse processes of the lumbar vertebrae, midway between the last rib and the tuber coxae, to avoid the pleural reflection, which may extend 2 to 3 cm caudal to the last rib. The incision should penetrate the abdominal tunic, and the fibers of the external oblique muscle should be separated as they traverse the incision diagonally, ventrocaudad. If present, the fibers of the internal abdominal oblique muscle should also be separated in the direction of the fibers. The fibers of the transversus abdominis are vertical and should be separated in that direction. The peritoneum may be penetrated with a finger or Mayo scissors.

The rings and shrouds used to perform rumenotomies in cattle are too large for use in llamas. A more adaptable but more time-consuming technique is to suture C-1 to the skin prior to incising the wall of the compartment. This technique precludes contamination of the peritoneal cavity while exploring and evacuating C-1.

Ingesta should be removed as appropriate. In the case of ingestion of oleander, C-1 should be washed out with water to remove all ingesta possible. Even small particles of leaves may be sufficient to produce toxicity.

The exposed wall of C-1 should be thoroughly cleansed with physiologic saline and povidone-iodine solution, diluted 1:10. The compartment wall incision should be closed with a Cushing or Connell pattern before the skin/wall stay sutures are released. The abdomen should be closed as for a laparotomy.

Permanent C-1 Fistulation

This surgery is usually done as an experimental procedure for basic nutrition studies, but it may also be indicated for maintenance of a donor animal for transfaunation of other llamas with digestive disorders. The basic incision is made as for gastrostomy. A half circle should be removed from the skin on each side of the incision. The resultant circular opening should be the size of the plastic fistula to be inserted later.

The dorsal aspect of the caudal sac of C-1 should be pulled up into the skin incision and the serosa of the stomach sutured to the skin with a single continuous suture. The seal at the ventrum must be secure. It may be appropriate to scarify the serosa of C-1 prior to the suturing to enhance the adhesion of the stomach wall to the skin.

The exposed serosa of C-1 should be covered with a moist dressing to preclude drying. One to two weeks should be allowed for healing before the stomach is incised for insertion of the plastic fistula.

Surgery of the Spiral Colon

The spiral colon is a frequent site of intestinal impaction. If obstruction of the spiral colon is encountered during an exploratory laparotomy, it should be evaluated and treated as follows.[41,42] The spiral colon may be seen near a ventral midline incision unless it is displaced by gaseous or fluid distention of other segments of the intestine or by torsion of the mesenteric root.

Intestinal coloration varies with the severity of the lesion, ranging from normal in a simple impaction or

mild ulceration to congested and cyanotic in torsions or other conditions with vascular compromise. Necrosis of the bowel wall may also be encountered. The proximal colon and the distal small intestine may become distended in colonic impaction.

If a torsion is found, the direction and degree of the twist should be established and the appropriate correction made. The spiral colon may be exteriorized through the incision to aid in evaluating torsion or impaction.

It may be possible to gently knead a semisolid impaction and reduce it to particles small enough to be moved through the remainder of the intestine by normal peristalsis. However, if the wall is ischemic and friable, more harm may be done by using this technique than by performing an enterotomy.

Before an enterotomy can be performed, the segment of colon containing the obstruction must be isolated from other segments of the spiral. The centrifugal coils are deeply embedded within the mesentery that binds the segments of the spiral together. Blood vessels should be avoided, but adequate exposure is necessary to allow manipulation of the colon to achieve a simple enterotomy or intestinal resection.

It is usually necessary to decompress loops of bowel distended by gas or fluid prior to complete examination of the digestive tract and, surely, before enterotomy or resection.[25] One of two methods may be chosen for decompressing the bowel. Gas and fluid may be withdrawn via a large-gauge needle or a rubber tube inserted through a stab incision and surrounded by a purse-string suture. A more rapid and thorough decompression can be accomplished using a technique frequently employed in equine colic surgery. In this technique, the distended bowel is laid on a sterile tray inclined to a collecting basket that, in turn, is connected to the surgery's drainage system. A longitudinal incision is made along the antimesenteric border of the bowel. Regular culinary water may be used to wash ingesta down the tray and wash the surface of the intestine. A common cause of ileus is failure to adequately decompress the intestinal segments before closure. This intestinal incision should be closed using an inverting pattern, preferably a Cushing pattern, before progressing further with surgery.

If enterotomy is necessary, a small longitudinal incision should be made over the mass, which is then withdrawn with forceps and discarded. The incision should be closed with a single-layer inverting pattern. Infolding of the bowel wall should be strictly limited to minimize the potential for future stricture. All exteriorized loops of intestine should be rinsed with sterile physiologic saline and replaced in the normal position. The abdominal wall should be closed as for any laparotomy.

Rectal Prolapse

Cause

Chronic enteritis resulting in persistent diarrhea is the primary cause of rectal prolapse. However, the rectum may also evert during tenesmus associated with prolapse of the vagina or uterus. A weak or relaxed anal sphincter may allow a few folds of the rectal mucosa to prolapse, especially while a female in an advanced stage of pregnancy is lying down.

Clinical Signs

The protrusion of the mucosa and wall of the rectum is evident. A protrusion of 5 to 15 cm may not compromise the vascularity of the rectum, but if the prolapse is 20 to 30 cm, ischemic necrosis of the bowel may occur from compression of the vessels. The protruded rectum will exhibit varying degrees of edema, trauma, and necrosis of the mucosa.

Position

The standing position is most desirable, but because camelids have a propensity to lie down, it may be necessary to operate with the animal in that position.

Anesthesia

Epidural anesthesia (Chapter 5) provides analgesia and relaxation sufficient to replace the prolapse.

Surgery

The mucosa must be cleansed and all foreign material physically removed. The cleansed tissue may be cupped in the palms of the hands and the prolapse gently replaced. If edema is severe, it may be desirable to wrap the prolapse tightly with a moistened towel or sheet until the swelling is reduced. Internal telescoping may be reduced by gentle insertion of a lubricated plastic or cardboard vaginal speculum.

A purse-string suture should be placed in the anus (Figure 6.20). Four or five loop sutures should be placed approximately 2 to 3 cm peripheral to the center of the anal orifice (Figure 6.19). The loops may be laced through with 3-mm umbilical tape, which is tied in a bow, leaving sufficient space for feces to be passed.

The author has not had occasion to perform a submucosal resection or amputation of a necrotic segment of the rectum in a camelid, but presumably this could be done with techniques recommended for cattle or sheep.[17]

Postsurgical Care

The patient should be observed for passage of feces and persistent tenesmus. The bow may be released when straining ceases, but the loops should be left in place in case the prolapse recurs.

Figure 6.20. Placement of purse-string suture for prolapsed rectum.

Complications

Stricture of the rectum or anus may occur from trauma to the mucosa, but stricture more frequently develops following amputation. Peritonitis, resulting from dehiscence of the suture line, is a possible complication.

Rectal Laceration

Causes

Laceration of the rectal mucosa or of the entire wall occurs rarely during rectal palpation. Veterinarians with large hands or arms may have difficulty in conducting a thorough rectal examination because of the small size of the pelvis, rectum, and colon of SACs. Llamas are not as prone to laceration as equine species nor as resistant to rectal trauma as cattle. Incidents of rectal laceration and perforation have been reported at meetings.

In one instance, an epidural had been given to allow a more thorough examination. While the operator's hand was in the colon, a spasm of the colon clamped the wall around the wrist. Any attempt to withdraw the hand met with resistance, and had force been applied, the mucosa would surely have split. Ten minutes elapsed before the spasm relaxed.

Strong peristaltic waves may push against the hand. The palpator must give and take with these waves. A sudden movement of the animal, such as falling or rearing, when the rectum is under tension may result in a perforation.

Clinical Signs

The palpator may be able to feel the split occur, but usually the first indication is when the hand is withdrawn and a large quantity of blood is seen on or in the hand. It is not unusual or dangerous to see a few streaks of blood on the back of the wrist when the hand is withdrawn, especially with a large hand. The anus may be the limiting orifice, and if a rectal examination proceeds without adequate lubrication or relaxation with an epidural, the anal epithelium may be stretched beyond its flexibility, resulting in tiny fissures. After a prolonged examination, the mucosa may be slightly abraded and a small quantity of blood will be seen on the fingers. Neither of these conditions should be confused with the large quantity seen if a laceration has occurred.

If a laceration is not detected at the time of palpation, the progression of the syndrome depends on the severity of the lesion. A small mucosal laceration may heal without incident, or streaks of fresh blood may be seen on some of the fecal pellets. In mares, a mucosal laceration may enlarge and penetrate both the muscularis and the serosa. Insufficient numbers of llamas have been studied to know whether this occurs in llamas.

If the laceration perforates the colonic or rectal wall, peritonitis will ensue. Signs indicative of peritonitis appear within twenty-four to thirty-six hours. Without treatment the animal will die in five to seven days.

If a laceration is suspected, it is unwise to reenter the rectum to investigate. An epidural and an equine plastic or cardboard vaginal speculum may be used to visualize the rectum without imposing excessive further stretching of the mucosa.

Presurgical Preparation

Rectal laceration is an emergency. Parenteral administration of broad-spectrum antibiotics should be begun as soon as a laceration is suspected.

Anesthesia

Evaluation and suturing via the anus necessitates epidural or general anesthesia.

Surgery[48]

The peritoneal reflection is located approximately 4 cm from the anus. A penetration caudad to that location will be retroperitoneal. The rectum and colon cranial to the reflection have an outer serosal layer, and this is more likely to be the area of the bowel that has been traumatized. Fecal pellets should be gently removed from the rectum and the rectum packed cranially with cotton or gauze to avoid subsequent contamination of the field. The mucosa should be carefully but gently cleaned with physiologic saline and precautions taken to avoid enlarging the laceration. A dilute (1:10) povidone-iodine solution may be used to provide some degree of asepsis.

If it is determined that the laceration penetrates only the mucosa and is less than 1 cm in length, no treat-

ment may be needed other than parenteral administration of broad-spectrum antibiotics for seven to ten days. Lacerations longer than 1 cm or penetrating the muscle layers should be sutured. It is difficult to obtain exposure via the anus. If the laceration is within 10 to 15 cm of the anus, a human plastic vaginal speculum, with a spoon length of 10 cm, may provide access to the surgical site.

A mucosal laceration may be closed with simple interrupted sutures using swedged, taperpoint needles and vicryl suture. Picking up the underlying muscle layer adds strength to each suture.

If the laceration has penetrated the bowel wall, a laparotomy should be performed to close the laceration and to evaluate the peritoneal cavity for evidence of fecal contamination. This is an emergency procedure; therefore, it is not be possible to fast the llama to partially empty the stomach. Exposure of the surgical site is the major problem. A high-flank incision would put the surgeon in the closest proximity to the colon and rectum, but unless both the external and internal abdominal oblique muscles are transected, there is not sufficient exposure to operate. If the patient is a pregnant female, especially if she is in advanced pregnancy, the flank approach is mandated.

The caudal colon and cranial rectum may be approached from a ventral midline incision. If there is extensive fecal contamination or peritonitis is already present, the prognosis is unfavorable.

Placing an intestinal clamp (Carmalt) on the laceration may prevent further contamination during the procedure. Particulate matter should be removed manually and the abdominal cavity flushed with physiologic saline in sufficient quantities to remove any residual contamination. A final flushing with povidone-iodine solution (diluted 1:10) is important.

The defect should be closed with an infolding Lembert or Cushing pattern using absorbable suture. The abdominal wall may be closed as for any laparotomy. Broad-spectrum antibiotics should be administered for seven to ten days post surgery.

Postsurgical Care

Little can be done to soften the stool without giving a laxative, which is contraindicated, because it would stimulate increased peristaltic activity. Enemas are also contraindicated, because the fluid could seep through the incision and the pressure would be detrimental to healing.

The patient should be monitored for signs of peritonitis. A hemogram should be performed at least every other day for six days.

Complications

Peritonitis is the most serious sequel. A rectal stricture is also possible.

Atresia Ani/Coli/Ilei

Atresia ani or coli is a congenital/hereditary defect of the neonate in which the anal opening is lacking or there is a stricture in the rectum or colon. Both forms have been reported in llamas and alpacas. This may be a hereditary condition, so the implications should be discussed with the client. These animals should not be used as breeders.

Clinical Signs

Abdominal distention and straining to defecate are the primary signs. In atresia ani, the absence of the anal opening is apparent, and there is usually a bulge at the anus. The signs are noted within a few days of birth.

Atresia coli is more difficult to detect, depending on how far cranially the lesion is located. Digital palpation may locate a lesion in the rectum, but a barium enema may be required to determine the site of a lesion farther forward in the colon. It is possible for an older animal to acquire a rectal or anal stricture and to develop the same clinical signs.

Anesthesia

Sufficient anesthesia for the correction of atresia ani may be achieved by infiltration of local anesthetic into the operative site. General anesthesia is required for repair of atresia coli, which necessitates a laparotomy.

Surgery

With atresia ani, a circular disc of skin approximately 2 cm in diameter should be removed from the center of the bulge. Feces may be allowed to escape by puncturing the rectal mucosa. The opening may be enlarged as seems appropriate. The mucosa should be sutured to the skin using vicryl or chromic gut, Figure 6.21.

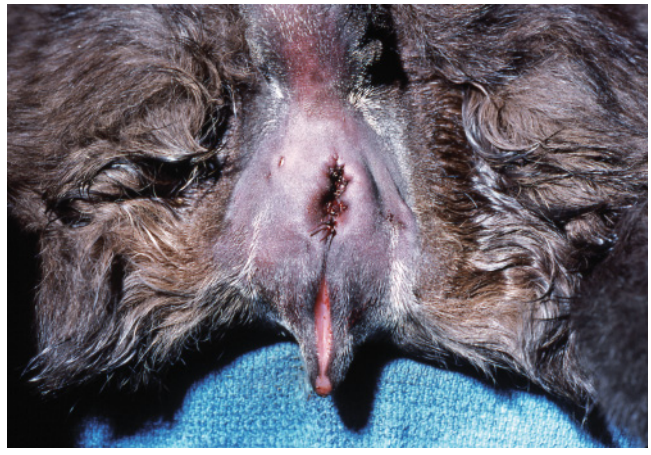

Figure 6.21. Repair of an atresia ani.

Strictures within the rectum are difficult if not impossible to reach surgically. Strictures farther forward in the colon may be corrected by laparotomy and intestinal resection.

Postsurgical Care

To maintain patency the orifice should be stretched at least every other day for two weeks.

Complications

Colonic or anal stricture is the most important adverse sequel. If there is no anal sphincter, the animal will be incontinent.

Miscellaneous Soft Tissue Surgery[18,30,46]

As veterinarians gain more experience with camelids, most of the surgical procedures performed on livestock and horses will be done. Procedures that have been described in the literature include Janeway gastrostomy,[42] silicone T-tube to treat tracheal stenosis,[26] inguinal hernia repair,[56] esophageal fistulation,[19] mastectomy,[16] and oral flaps for correction of cleft palate.

REPRODUCTIVE SYSTEM

Cesarean Section[35,40]

Indications

A cesarean section is indicated when the fetus is unable to traverse the birth canal. The fetus may be enlarged (emphysema or anasarca) or malformed (ankylosis of carpus or tarsus, schistosomas reflexus).

If an immature female is bred accidentally (cases are on record in which a female has become pregnant at five months), parturition will occur before the pelvis and the genital tract have reached maturity. The cervix may fail to relax and dilate properly, or the uterus may clamp around the fetus so tightly that forced extraction will rupture the uterine wall. A fetal mummy lacks flexibility, and the cervix may not relax sufficiently to allow expulsion of a mummified fetus.

Narrowing of the birth canal may occur as a result of previous trauma, causing fibrosis and stenosis, or from pelvic tumors. Correction of an acquired vaginal stenosis resulted in a passageway that allowed penetration by the penis, but not sufficient space to allow natural delivery.[b] Uncorrected uterine torsion mandates a cesarean section. In addition, prolonged manipulation may jeopardize the life of the fetus, and a cesarean may be appropriate to obviate the threat.

Clinical Signs and Diagnosis

Prolonged labor without progress is the major sign. There are no unique clinical manifestations or diagnostic procedures.

Position

Dorsal recumbency is required for the ventral midline approach. For a flank incision, lateral recumbency with the animal lying on its right side is appropriate.

Presurgical Preparation

A cesarean section is usually an emergency operation, thus preparation is limited by time and available facilities. The best possible standard of surgical preparation should be followed.

Anesthesia

General anesthesia is necessary for a cesarean section.

Surgery

Left-Flank Approach. Because more than 95% of camelid pregnancies are in the left horn, there is a gravitational twist counterclockwise that pulls the uterus toward the right side. If the right side is selected, it is more difficult to exteriorize the uterus for extracting the fetus because the uterus is already pulled toward the right side. Furthermore, if the uterus contracts during suturing, the uterus will pull away from the right incision. The low-flank area is devoid of muscle mass and should be avoided. A midflank incision is appropriate. The laparotomy technique has been described earlier. There is no greater omental sling to bypass, so it is easy to grasp and exteriorize the pregnant uterine horn.

The uterine wall should be incised on the greater curvature, avoiding large vessels. The fetal membrane arrangement in camelids differs from that of other artiodactylids (Chapter 17). With a cranial presentation, the hind limbs are encountered first in the uterus; they may be grasped to deliver the fetus. A clamp should be placed on the umbilicus. The uterine-chorial attachment is minimal, and it may be possible to deliver the placental membranes through the uterine incision. If much pressure must be exerted to free the placenta, it should be left in situ for later vaginal delivery. The second horn of the uterus should always be examined for the presence of a twin. Any uterine torsion should be corrected and the uterus palpated externally for possible uterine rupture.

The uterine incision may be closed with one or two layers using a Lembert or Cushing infolding suture pattern and any absorbable suture. The uterus should be replaced in the abdominal cavity and the cavity flushed with physiologic saline if there has been contamination with infected fetal fluids. The presence of uninfected amniotic or allantoic fluid in the peritoneal cavity is not harmful.

The abdominal wall may be closed as for a laparotomy.

Ventral Midline Approach. The initial incision should be made from the cranial border of the mammary gland forward for a distance of 30 cm. The operation may then proceed as for the flank approach.

Postsurgical Care

If dystocia was prolonged, or significant manipulation occurred before the decision to perform a cesarean section was made, it may be necessary to treat for endometritis or metritis. Oxytocin (30 units) should be administered intravenously to stimulate uterine contraction, expel the placenta, and assist in hemorrhage control. Oxytocin should not be administered until the uterus has been sutured and replaced inside the abdomen; 1 to 2 g tetracycline powder may be inserted in the uterine horn at the time of surgery. Boluses are contraindicated, because these may stimulate straining. Excessive soft-tissue trauma or microbial contamination may indicate the administration of parenteral antibiotics for five days post surgery.

Complications

Retained placenta, metritis, endometritis, peritonitis, and herniation at the incision site are potential complications.

Ovariohysterectomy

Indications

A uterine rupture with severe trauma to the uterus may be an indication for hysterectomy. In one case, small fetal mummies were identified by rectal palpation. A hysterotomy was performed, and three partially developed fetal mummies were found firmly embedded in the uterine mucosa. These were removed with difficulty. Postsurgically, this female suffered from a prolonged chronic metritis and, ultimately, hysterectomy was required.

One of the more common congenital anomalies of the reproductive tract of llamas and alpacas is segmental aplasia of the tubular tract anywhere along its length, preventing egress of normal uterine secretions. In such cases, uterine fluids accumulate, resulting in enlargement of the uterine horn, simulating pyometra. Eight to 10 L of milky fluid have been drained from some long-standing cases (Figure 6.22). Ovariohysterectomy is indicated to enable use of the animal for a pet, a packer, or fiber production.

Various experimental protocols may require either ovariectomy or ovariohysterectomy.

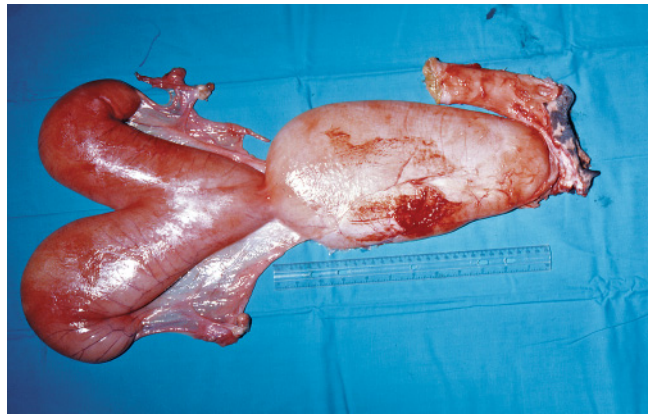

Figure 6.22. Mucometria, ventral view, emphasizing vascular distribution.

Position

Dorsal or lateral recumbency may be appropriate, but dorsal recumbency is required if the cervix is to be extirpated.

Presurgical Preparation

As in other elective procedures, the animal should be fasted for twenty-four to forty-eight hours.

Anesthesia

General anesthesia is required.

Surgery

Surgery should be from the ventral midline approach. The skin incision should begin just cranial to the mammary gland and extend forward 30 to 40 cm. The mammary gland should be reflected caudally for better exposure for penetration of the abdominal wall as close to the pelvis as possible. Both uterine horns should be exteriorized.

Fluid may be drained from the uterus with a large-bore needle attached to a flexible tube for gravity drainage or attached to a suction apparatus. More rapid drainage may be accomplished by inserting a suction head through an incision in the uterine wall. For this procedure, a purse-string suture is placed at the intended site. With the suction head ready, a stab incision is made and the head quickly inserted through the opening. The purse string should be tightened around the suction head with a single throw and the tension held with a hemostat. Following removal of the uterine contents, the suction head should be removed and the purse-string suture pulled tight.

The uterus may then be manipulated to isolate vessels and the cervix. The ovarian and cranial uterine arteries must be identified and double ligated. Other uterine vessels are found in the broad ligament and on the ventral aspect of the uterus (Figure 6.20). These

arteries and veins should also be secured by double ligation.

Following ligation of the vessels, the uterine ligaments may be transected to expose the vagina just distal to the cervix. Even with marked enlargement of the uterus, the cervix lies close over the brim of the pelvis or within the pelvis. Visualization of the vagina is enhanced if the animal can be tilted forward by raising the hindquarters, but it is difficult to achieve the desired exposure.

The vagina should be closed with overlapping mattress sutures and number-2 or -3 gut or other absorbable sutures of a similar size. Amputation of the reproductive tract proximal to the mattress sutures is best. Simple interrupted sutures may be used to approximate the serosal surfaces of the stump. A final check for hemorrhage should be made before closing the abdomen as described previously.

Routine monitoring during surgery is necessary, because shock may ensue from the removal of such a large organ.

Postsurgical Care

Standard postsurgical regimens are recommended, as for any laparotomy. Since uterine fluid is not infected, there is no indication for parenteral antibiotics if surgical asepsis is maintained.

Persistent or Imperforate Hymen

Diagnosis

It is critical that this condition be differentiated from segmental aplasia of the vagina. A persistent hymen may be only one manifestation of a continuum of congenital anomalies of the tubular genital tract of the llama and alpaca. Because such anomalies are considered to be hereditary in certain breeds of cattle,[31] the veterinarian should consider the ethical implications of performing surgery to permit breeding, especially if it is likely that the female is to be sold as a breeder.

Persistence of a segment of hymen may present a stricture at the vulvovaginal junction. This may be discovered by digital palpation. The orifice may be enlarged by gentle finger pressure. The urethral opening on the floor of the vulva should be identified either by digital palpation or inspection with a vaginal speculum before the hymen is manipulated. A small heifer speculum may then be used to enlarge the orifice further to visualize the cervix.

An imperforate hymen may be indicated by a bulge in the vulva caused by the accumulation of fluid in the vagina and uterus. On palpation, this may resemble palpation of a fluid-filled balloon. It may be thought that the hymen should break at breeding, but the male llama does not have a vigorous pelvic thrust, and penile penetration may be easily deterred by even a slight obstruction. In at least one case, the penis was deflected into the urethra. When penetration by the male was checked for by the breeder, it was noted that the urethra was markedly dilated. The female lacked a vagina; there was no passageway beyond the hymen.

Indications

An imperforate hymen results in the accumulation of uterine secretions, just as in segmental aplasia of the vagina. Rupture of the hymen and maintenance of patency allows drainage of the fluid and avoidance of the complications of a prolonged distention of the uterus.

Position

The animal should be standing, with the tail secured up over the back or to the side, out of the perineal area.

Anesthesia

Usually, none is necessary, but an epidural using 1 to 2 ml 2% lidocaine provides anesthesia if required.

Surgery

The membrane may be easily ruptured by a quick thrust with a finger. A membrane too thick to rupture with finger pressure requires surgical intervention. The lips of the vulva may be spread, or a short speculum may be used to allow visualization of the hymen. A stab incision, enlarged with a finger or forceps, is recommended. Palpation proximal to the hymen should ascertain the size of the lumen. The orifice may be enlarged by lateral incisions in the form of a cross or by a circular incision to remove a segment of the hymen.

Postsurgical Care

It is important to maintain the patency of the orifice by continual stretching during the healing process. Otherwise, contraction and scarring will negate the benefits of the surgery. Stretching may be accomplished by inserting a heifer vaginal speculum once or twice daily for ten days.

Complications

Reclosure and stenosis of the vulvovaginal junction are the major complications. It may be possible to make an opening in the tract large enough to allow penetration by the male to inseminate the female. If, however, stenosis develops at the hymenal area, dystocia is probable.

Prolapse of the Vagina

Causes

Vaginal prolapse is usually associated with pregnancy. In cattle, it often occurs at two to three months

of pregnancy, at about the stage in which the placenta begins to secrete estrogens, inducing relaxation of the pelvic structure. Insufficient records have been kept to correlate similar happenings in camelids.

Prolapse occurs more frequently in multiparous females, indicating that stretching or injury to the pelvic structures as a result of a previous birth may predispose to vaginal prolapse. In cattle and some other species, it is thought that a hereditary factor may be involved and also that some animals may produce more estrogens than others.[31]

Obesity, with excessive pelvic fat, may produce increased abdominal pressure while an animal is lying down. A preexisting relaxation of the pelvic structure may also contribute to vaginal prolapse.

Vaginal prolapse is a complication of parturition, particularly if dystocia has resulted in excessive manipulation of the fetus.

Clinical Signs

A slight prolapse of the mucous membrane of the vagina is sometimes seen during pregnancy near term, especially when the female is lying down. The membrane is usually pink, moist, and glistening, with no evidence of pathology. If the bulge disappears when the female rises, no treatment is indicated. However, a continually exposed membrane may become traumatized by the tail or become dry and necrotic.

The entire vagina may be exteriorized by persistent tenesmus. Edema or pressure may obstruct the urethra and cause urine retention. Other caudal abdominal organs may prolapse through the vulva via rents in the uterine or vaginal wall.

Position

The llama should be standing, if possible, with the hindquarters elevated.

Presurgical Preparation

An extensively prolapsed vagina should be wrapped with a moistened bath towel and encased in a plastic bag to keep the membrane moist and to avoid trauma and infection.

Anesthesia

Epidural anesthesia is recommended.

Surgery

The extent of the prolapse and degree of trauma to the mucous membrane determine treatment. The membrane should be thoroughly cleansed with physiologic saline and povidone-iodine solution (diluted 1:10). Any foreign material must be physically removed, and obviously necrotic tissue should be debrided. The vagina may be cupped in the hands and gently pushed back into place with the palms. Point pressure with the fingers should be avoided. The invagination must continue past the lips of the vulva. A person with small hands may be able to push beyond the vulva with bent fingers. A plastic or cardboard mare speculum may be gently inserted to complete the inversion process if the operator's hand and arm are too large to enter the female.

It is usually necessary to suture the lips of the vulva closed to prevent recurrence until the edema and inflammatory response have dissipated. The vulva is small, and a simple purse-string suture may be sufficient. However, the author prefers a lacing technique similar to that used on cattle.

In this technique, two or three loop sutures are placed on either side of the vulva, 2 cm lateral to the vulvar slit. Excessive slack should not be left in these loops (Figure 6.23). The loops are threaded like a shoe is laced, using 3-mm (1/8-in.) umbilical tape, and tied in a bow. The animal will be able to urinate. The threaded strand may be loosened or even removed to ascertain the degree of healing but can be easily replaced if the prolapse threatens to recur.

If this procedure is carried out on a pregnant female that is nearing the end of term, she must be closely observed so that the lacing can be removed as parturition begins.

Sprinkling the swollen membrane with sugar to reduce edema of a prolapsed vagina is a time-honored practice, and there is little doubt that fluid can be removed in this manner. However, theriogenologists believe that, as well as being hygroscopic, the sharp sugar granules traumatize the friable mucous membrane, allowing the escape of vital fluid.[31]

It may be desirable to insert a catheter to instill a solution containing powdered tetracycline into the vagina, but an antibacterial bolus should not be inserted, because it may stimulate tenesmus.

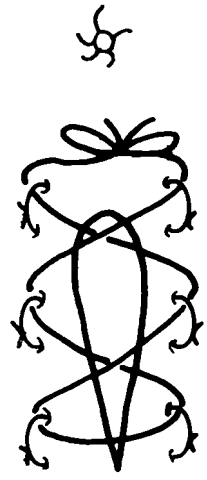

Figure 6.23. Truss (shoelace) pattern for retention of vaginal prolapse.

Postsurgical Care

The animal should be observed closely for signs of continuing tenesmus or significant exudation.

Complications

The major complication is recurrence of the prolapse; however, vaginitis and even metritis may follow a prolapse. If tenesmus persists, the vagina should be reexamined to ensure that the prolapse was completely repositioned.

Prolapse of the Uterus

Causes

Much of the discussion relative to vaginal prolapse applies to uterine prolapse. Prolonged and intense tenesmus induced by a retained placenta is a common cause. The uterus has also been known to prolapse during attempted delivery of the placenta immediately following parturition. Following an exhausting dystocia, a completely atonic uterus may prolapse through the relaxed vagina.

Clinical Signs

The protrusion of the inverted uterus is obvious. In camelids, there are no cotyledons on the placenta. It may be somewhat difficult to differentiate uterine from vaginal mucosa, but it should be possible to identify the cervical rings.

Position

Same as for vaginal prolapse.

Anesthesia

Same as for vaginal prolapse.

Surgery

The procedures outlined for vaginal prolapse should be followed. However, it may first be necessary to deliver the placenta. Any portion of the placenta that remains should be delivered. The attachment is minimal, and delivery should not be difficult. It is more difficult to reduce the telescoping, because of the small size of the canal, but immediately after parturition, even a person with large hands can manually reposition the uterus, taking care to evert both horns. If the prolapse occurs at any other time, it may be repositioned only by a person with small hands and arms. As soon as the uterus is replaced, 30 to 50 units of oxytocin should be administered intravenously or intramuscularly to stimulate contraction of the uterine muscle.

A truss suture pattern may be employed to ensure retention of the uterus, but in many cases this is not necessary. The animal should be observed closely for tenesmus and reexamined if it persists.

Uterine Torsion[20]

Uterine torsion is discussed in Chapter 17, Reproduction.

Rupture of the Uterus

Causes

In SACs, rupture of the uterus is usually associated with dystocia. Prolonged, strong contractions of the uterine musculature against an immovable fetus may result in a rent in the wall of the uterus. Other causes include uterine torsion and an oversized fetus. A limb may be pressed through the wall if a carpus or tarsus has become ankylosed and inflexible. Chronic peritonitis associated with uterine adhesions may result in a tear in the uterine wall when the contractions of parturition begin. Severe trauma to the abdomen near term may cause a rupture of the uterus.

Clinical Signs

External signs may be minimal and consist only of knowledge that dystocia has occurred. Frequently, the rent will be palpated after the fetus has been delivered and the uterus is being checked for a twin. If the rupture is not detected at parturition, it may be indicated as signs of peritonitis develop.

Position

Dorsal recumbency is the preferred position for surgery.

Anesthesia

General anesthesia is required.

Surgery

The surgical approach to correct a uterine tear is similar to that used for hysterectomy. Hemorrhage in the peritoneal cavity or peritonitis may be present. Because the tear may be in the body of the uterus near the cervix or in the uterine horn, careful palpation is necessary. A tear in the body is more difficult to visualize and suture.

A Lembert or Cushing infolding suture, as in a cesarean section, is satisfactory for closing the tear. The abdominal cavity should be lavaged with physiologic saline and treated as appropriate to prevent or alleviate peritonitis. The abdominal incision is closed as described previously.

A novel technique for suturing a uterine tear in cattle has been described but not yet tried in a llama. With this technique, if the rent is detected during or immediately following parturition, a hand and arm are inserted into the tip of the lacerated horn. An assistant administers 4 to 6 ml 1 : 1,000 epinephrine HCl intravenously to induce relaxation of the uterine musculature. When the surgeon feels relaxation begin, the tip of the

horn is grasped by the fingers and slowly everted and pulled through the vagina. The laceration may then be sutured outside the body and the uterus returned to its proper position. Oxytocin (20 units) should be administered to stimulate contraction of the uterus following the surgery.

Postsurgical Care

Standard laparotomy protocol is appropriate. A five- to ten-day course of antibiotics, given parenterally, is indicated to prevent or treat metritis and peritonitis.

Complications

If one of the large uterine arteries is ruptured concurrently, intraabdominal hemorrhage may result in exsanguination or severe anemia. Anemia would not be detectable via a hemogram for a few days after the incident. Peritonitis is a common sequel, along with metritis.

Laparoscopy[62]

Indications

Laparoscopy has been used to visualize the reproductive tract, especially ovarian function.[50,52] It is also used to evaluate other abdominal organs and for guided biopsy of the liver or kidney and for vasectomy.[7] It may also have application in diagnosis of chronic infertility in conjunction with ultrasonography, rectal palpation, and hormone profiles.

Position

Laparoscopy in North America is usually conducted in the standing position from the right flank. In South America, experimental animals are physically restrained in dorsal recumbency on a surgical table that can be tilted vertically, with the animal in a head-down position. With the llama in dorsal recumbency, the penetration site is on the ventral midline near the umbilicus or 3 to 5 cm cranial to the mammary gland.[50] Two penetration sites may be necessary, one for the fiberoptic scope and the other to admit forceps to manipulate structures.

Anesthesia

Mild sedation with xylazine HCl (0.1 mg/kg), followed by butorphanol (0.05 to 0.1 mg/kg), will quiet the animal yet allow it to remain standing. The insertion site for the laparoscope should be infiltrated with a local anesthetic.

Surgery

For the standing approach, the right flank is selected because C-1 of the stomach obscures the field on the left side. The appropriate site is 10 to 15 cm ventral to the transverse processes of the lumbar vertebrae and 10 to 15 cm cranial to the tuber coxae. The kidney may be traumatized if the penetration site is too far dorsal to the recommended site. The insertion site should be clipped and aseptically prepared for surgery.

Standard laparoscopy procedures are employed, which vary with the instruments available. Inflation of the peritoneal cavity with an inert gas (nitrogen) is necessary to visualize abdominal organs. The anatomy of the camelid reproductive tract should be reviewed before initiating laparoscopy (Chapter 17). The camelid ovary is enveloped within a bursa that must be lifted from the ovary before it can be visualized.

Observation of the left ovary is more difficult but is possible from the right side. In dorsal recumbency, the ovaries are equally accessible.

Castration[54]

Indications

In addition to the usual indications for castration as seen in livestock, castration is highly recommended for llama or alpaca male crias that are hand raised with poor socialization with the herd. It should be understood that a cria may be bottle raised, but it should be given no special human attention other than feeding followed by return to association with other camelids to be properly socialized. Bottle-raised crias (both male and female) or crias with close human contact may become imprinted on humans and become pushy or aggressive as adults. Such animals should be castrated before sexual maturity, preferably before two months of age. The gonads are usually present in the scrotum at birth, and castration may be performed as early as two weeks of age or any time thereafter.

Position

For a right-handed surgeon, lateral recumbency with the right side up is recommended. The upper leg should be tied to the neck and the tail tied to the fleece over the back.

Anesthesia

Xylazine (0.3 mg/kg IV), with local anesthetic infiltration of the skin 3 to 5 cm cranial to the scrotum on either side of the median raphe, is satisfactory. If sedation is insufficient, IV ketamine (25 mg/kg) may be administered.

Surgery

Numerous methods have been employed in castrating camelids.[6,8,10,38,51,54] Any standard method recommended for livestock is suitable. The author's preferred method is described first, with variations of the technique to follow.

With the animal in lateral recumbency, two 4-cm incisions should be made 3 to 5 cm cranial to the scrotum and 1 cm to the right and left of the median raphe. Blunt dissection may be used to isolate the spermatic cord. The penis should be palpated to avoid exteriorizing it. A finger may be placed under the cord to lift it out of the incision and pull the testicle forward from the scrotum. The testicle should be grasped in one hand and the fat stripped from the tunic as far craniad as possible (toward the external inguinal ring).

A transfixation ligature should be placed around the tunic near the external ring and the cord transected with either an emasculator or a scalpel. Alternatively, an incision may be made through the common tunic longitudinally to isolate the vessels and the ductus deferens, which should be ligated separately, transected, and released. Then the common tunic should be ligated.

After both testicles have been removed, the incisions should be plucked to remove any strings of fat that may protrude from the incisions after the animal rises. Any pockets may be eliminated to ensure unimpaired drainage. Incisions are not sutured.

Postsurgical Care

Administration of antibiotics is unnecessary, and equine-origin tetanus antitoxin serum is not recommended. Swelling is rare. Llamas do not lie in their own feces; therefore, the incision remains clean. It is recommended that the llama be returned to pasture or kept in a clean, straw-bedded box stall.

Alternative Techniques

1. The same procedure may be followed, except incisions are sutured as in a canine castration. Surgical asepsis must be employed throughout.
2. The initial incision may be made through the scrotum. Using a closed technique, the testicle and surrounding tunics are isolated and separated from the surrounding fascia as far craniad as possible. An open technique may also be performed, in which case the common tunic is incised, freeing the testicle, epididymis, and ductus deferens. The ductus deferens and other vessels should be ligated prior to transection. The common tunic is left in situ.
3. In standing castration, the camelid should be placed in a narrow stock or squeeze chute, with the tail held or secured up over the back. The area should be surgically prepared, with local anesthesia administered subcutaneously over each scrotal sac. Additional local anesthetic may be injected into the spermatic cord, craniad to the scrotum, or an open procedure is employed, with incisions left unsutured.
4. In single incision, the animal is anesthetized and placed in lateral recumbency. A rope is placed around the upside fetlock and the rope threaded over the back to the opposite hock and tied at the fetlock.[34] This method of restraint places the rear limbs in a frogleg flexed position. A testicle is pushed craniad as far as possible (10 to 15 cm from the scrotum). A 5-cm incision is made over the testicle, approximately 3 cm lateral to the midline, to the common tunic. The testicle is grasped and stripped of areolar tissue and the cord ligated as close to the inguinal ring as possible. The cord is then transected distal to the ligature. The opposite testicle is pushed craniad, dorsal (beneath in this position) to the penis, and extracted from the same incision. The skin is closed with the surgeon's preferred suture pattern

Early Castration—Pro or Con

It has been known since the time of Aristotle that boys castrated in infancy (eunuchs) developed elongated limbs when they grew up. The same is true for many other species of animals, but the degree of effect varies from species to species. In llamas and alpacas, castration before puberty may delay physeal closure of the long limb bones, allowing growth to continue.[22]

The physiology behind physeal closure should be understood. In males the physes of the humerus, radius, metacarpus, femur, tibia, and metatarsus begin closure with the onset of testosterone production. In females, estrogen production stimulates closure of the physes. If hormonal stimulation is delayed or prevented by neutering, physeal closure is delayed. The limbs continue to elongate, which changes the normal balance between body depth and limb length conformation (Chapter 24).

Reasons for early castration:

1. Prevention of aberrant behavior in human-imprinted males.
2. Elimination of intermale aggression so that males may be housed together or the gelded male may run with females.
3. Avoidance of accidental breeding of inferior males to receptive females.
4. Prevention of the use of inferior males, sold for pets or to packers, as breeding males.
5. Preference of packers for a tall llama.

Reasons to avoid early castration:

1. Avoidance of long-legged adults.
2. Tendency of long-legged camelids to be post legged on both the fore and hind legs, which may predispose them to patellar subluxation, especially if a shallow trochlear groove is present.
3. Potential for development of dropped fetlock, especially if the animal becomes obese, and arthritis or ligamentitis.
4. Concern of veterinarians with food animal experience about the possibility of urolithiasis in gelded animals. To date this has not been a problem; most urolithiasis cases have occurred in intact males.

Recommendations

Young males with aberrant-behavior tendencies should be castrated prior to the onset of puberty. If facilities for maintaining intact males are available, it is desirable to delay castration until libido begins and the canine teeth have erupted fully, usually between two and three years of age.

Although it has not been experimentally confirmed, the parenteral administration of testosterone to a gelded male at the approximate time of maturation may initiate physeal closure.

Be aware that hypo and agonadal females may also develop elongated limbs due to lack of estrogen production.

Cryptorchid Castration[36]

Cryptorchidism is one of many genetic malformations of the camelid male reproductive system (see Table 22.5 in Chapter 22). Only rarely has the cryptic gonad been retained in the abdominal cavity. More frequently, the gonad has been located in the fascia, anywhere from the external inguinal ring to near the scrotum and alongside the penis. In a few cases, the gonad has been located in the subcutaneous fascia on the medial aspect of the rear limb. The cryptic gonad may be difficult to palpate, because it may be only 0.5 to 1 cm in diameter and may be confused with local lymph nodes.

Because cryptorchidism is a genetic trait, such an animal should not be used for breeding, and both the cryptic and normal testicles should be removed. Bilateral cryptorchidism is extremely rare.

Surgery

The cryptic gonad should be located and an incision made where necessary to amputate. If the gonad is intraabdominal and the side can be identified, a paralumbar fossa grid approach may be used after incising the skin. If it is impossible to determine the appropriate side, the abdomen may be entered via the ventral midline approach.

Vasectomy

Indications

The primary purpose for vasectomy is to facilitate study of reproductive physiology. Vasectomized males can be used to check females for pregnancy or to induce ovulation without fertilization.

Surgery

The scrotum is not pendulous, so the spermatic cord is not as accessible as it is in the bull or ram. An incision is made cranial to the scrotum, similar to a castration incision. The spermatic cord is isolated and exteriorized. The tunic is incised and the ductus deferens isolated. Ligatures are placed 2 cm apart, and the segment between the sutures is excised. The tunic should be sutured with simple interrupted observable sutures.

Alternatively, a 2-cm segment of the ductus deferens may be excised via standard laparoscopy using forceps scissors.[7,50]

URINARY SYSTEM[58]

Ruptured Urinary Bladder

Indications

A rent in the wall of the urinary bladder may result from overdistention of the bladder, usually caused by partial or complete obstruction of the urethra. This is most likely to occur in the male and has been diagnosed in male llamas as young as six weeks of age as well as in adults. It is not known whether seepage of urine from a grossly distended bladder occurs in llamas as it does in cattle.

Another major cause of bladder rupture is trauma, especially if it occurs when the bladder is full. Two instances have been reported. In the first, a female llama had been transported in a trailer for a considerable distance and immediately used as a model to demonstrate a portable restraining stock, without being given an opportunity to urinate. The stock included two belly bands and a band over the back to prevent the llama from rearing or lying down. During the course of the demonstration, the llama struggled vigorously against the restraint. The llama died two days later despite treatment for depression and a supposed digestive disturbance. At necropsy, a rent in the bladder wall was observed.

The second instance of bladder trauma occurred during rectal palpation for fertility evaluation. On

initial entry, it was determined that the bladder was distended, making it difficult to palpate the genital tract. Digital pressure was exerted over the dorsal surface of the bladder through the rectum in an attempt to manually express urine. The clinician felt the distention suddenly dissipate and immediately realized that the bladder had ruptured. Almost instantaneously, the llama became extremely disturbed, began to vocalize, climbed out of the restraining stock, and threw herself to the floor, jumping up again after rolling. The clinical signs were those of violent colic; apparently, the rapid flow of urine into the peritoneal cavity was excruciatingly painful.

In retrospect, it seems possible that the violent struggling of the first llama, restrained by bands in the stock, occurred after the bladder had ruptured, not before.

Diagnosis

Uremic signs in the llama are not definitive. Except at the initial flush of urine into the peritoneal cavity, as previously described, signs of colic have not been seen. Depression and anorexia, with cessation of stomach motility, may be seen. Blood urea nitrogen levels become elevated to 50 to 100 mg/dl as the condition worsens. The abdomen is not likely to be distended, but fluid will be obtained by abdominocentesis, if done correctly.

The most precise method of determining the presence of urine in the peritoneal cavity is by analysis of fluid aspirate for potassium. Normal peritoneal fluid or exudate has a potassium concentration of 4 to 5 mEq/L, similar to that of serum. Urine has a concentration of 50 to 150 mEq/L. Odor and color are not sufficiently precise indicators, but urea nitrogen determinations are indicative. Another positive indication is detection of urate crystals in the peritoneal fluid.

Position

The animal should be placed in dorsal recumbency. Although the distended bladder is intraabdominal, the contracted bladder will lie partially within the pelvis or at the brim of the pelvis. A caudal midline approach is necessary to expose all surfaces of the bladder.

Presurgical Preparation

Standard surgical asepsis should be practiced. The patency of the urethra must be determined either preceding or during surgery. (See Chapter 18 for descriptions of anatomy and catheterization.)

Anesthesia

General. The uremic patient is an anesthetic risk and should be monitored closely. Hyperkalemia may predispose the development of cardiac arrhythmia, particularly if halothane inhalation anesthesia is chosen. IV fluid should be saline rather than Ringers solution.

Surgery

The abdominal wall should be penetrated as near the pubis as possible. All surfaces of the bladder must be inspected for rents, because multiple rents are possible. The rents previously described were found on the dorsal aspect of the body of the bladder, but experience in other species indicates that ruptures may occur at the neck or elsewhere in the body of the bladder.

Before closing the defect, the patency of the urethra should be established via retrograde catheterization of the external urethral orifice or by passing a catheter distally through the bladder. A double layer, continuous infolding suture pattern, using •• gut, is recommended to close the bladder wall.

The abdominal cavity should be irrigated with warm physiologic saline to remove all traces of urine. Unless secondarily infected, urine will not cause peritonitis.

The abdominal incision should be closed as previously described. This surgical procedure is also recommended for removal of cystic calculi and tumors.

Postsurgical Care

Postsurgical care is routine. Urine output should be monitored.

Urethral Obstruction

Clinical Signs

The severity of the clinical signs associated with obstruction of the urethra are determined by the duration of the obstruction. In early stages, with distention of the bladder, the animal will strain as if to defecate (Figure 6.24). The posture for urination and defecation are essentially the same in a camelid. Other colicky signs may be noted as well.

The enlarged, tense bladder should be obvious on rectal palpation. It is not likely that obstructions of the urethra can be palpated. If the bladder has ruptured, initial signs of severe colic will be followed by general uremia.

Diagnosis

In most livestock species, urethral obstruction is primarily a problem in castrated males, but in camelids it is frequently the intact, breeding males that are most commonly affected. In animals less than six months of age, it is difficult to locate the urethral opening because of preputial adhesions to the glans penis. Catheterization is difficult.

The hemogram in early stages of the disorder is normal. If the obstruction persists, creatinine and blood urea nitrogen levels will become elevated and a

Figure 6.24. Stance of a llama cria with urethral obstruction from a urolith.

leukocytosis and left shift may be evident in the differential cell count.

Radiography of the pelvic area is indicated, but some uroliths are not radiopaque. In one of the author's cases, the stone was composed of urate salts and could not be seen on radiographs, either of the animal or when the stones were placed on a cassette after removal from the urethra. In addition to the urate stone, the author has also removed a stone composed of unoriented crystals of 90% hydroxyl apatite and 10% struvite and silicate.

Stones may be located at the narrow opening between the pelvic and penile urethra at the level of the ischial arch, or where the urethra narrows as it enters the glans penis. In a three-month-old llama, this point was 7 cm from the external urethral orifice, while in an adult male it may be as much as 12 cm from the orifice. This location coincides with the area of preputial reflection.

In a six-week-old neonate, a 3.5 French catheter was passed to the ischial arch but could not be forced beyond it. Radiographs were taken, but no urolith was seen. A laparotomy was performed and a rupture of the bladder detected and closed. Signs of urinary obstruction recurred. A pelvic urethrostomy was performed. Urine flowed freely from a catheter placed in the bladder. A 3.5 French catheter was passed distally past the external urethral orifice, followed by passage distally of a 5 French catheter. Resistance was noted as the larger catheter reached the location of the narrowing of the urethra, but it was possible to continue on to the orifice. In the process, a urate urolith was pushed out of the urethra. The 3.5 French catheter had passed around the urolith.

Presurgical Preparation

The perineal area over and ventral to the ischial arch should be prepared for aseptic surgery.

Anesthesia

In cattle and sheep, this procedure is usually performed under epidural anesthesia, supplemented with local infiltration if necessary. This technique could be attempted in the camelid, but general anesthesia is more suitable to permit the various manipulations necessary for location and surgical correction of the obstruction.

Surgery

If the obstruction is located in the distal urethra, the incision should be made directly over the urethra. A catheter should be inserted up to the obstruction to identify the approximate incision site. If the bladder has ruptured or become atonic, urine flow may not resume immediately following removal of the obstruction. The catheter should be passed as far as possible to preclude the possibility of multiple uroliths. The urethra should not be sutured, to avoid inducing a urethral stricture.

Ischial urethrostomy may be performed as a temporary measure to aid in diagnosis or removal of a stone, or a permanent opening may be established if the urethra is severely traumatized and strictured distally. The incision must be made directly on the midline beginning at the ischial arch, extending ventrally 6 cm. The paired retractor penis muscles should be separated. The paired ischiocavernosus muscles are united on the midline by a firm fibrous layer that must be incised directly on the midline to minimize hemorrhage.

Insertion of a polypropylene catheter (5 French) as far as the ischial arch may be helpful in locating the urethra. At this point, the urethra is surrounded by the bulbospongiosus muscle and the corpus spongiosum urethra. The incision should continue into the lumen of the urethra, staying on the midline, and the urethra should be fixed with fingers or forceps to avoid incising alongside the urethra into the corpus cavernosum.

A catheter may then be inserted into the bladder or passed distally to locate the site of the obstruction. If necessary, an additional incision may be made over the distal obstruction to remove a urolith that will not pass with gentle catheter manipulation.

If the obstruction can be relieved, the urethrostomy should be considered temporary and not sutured. The skin incision must be more ventral than the opening into the urethra to prevent accumulation of urine in the subcutaneous tissue.

If the urethrostomy must be kept open, either for a period of time or permanently, the urethral mucosa should be sutured to the skin. If urine flow resumes, the urethra/skin sutures can be removed to permit healing.

Postsurgical Complications

The condition(s) allowing formation of the urolith(s) may not have been corrected, or be correctable, with the result that obstruction may recur. Urethral stricture is a common ailment in SACs.

In all cases, it is important to establish whether or not the bladder is intact. If rupture has occurred, a laparotomy, with appropriate surgery, must be performed. Peritonitis is always a possible result of urinary leakage, especially if there is a concurrent bacterial cystitis. Urine may or may not be sterile, so leakage from a stretched or ruptured bladder may or may not initiate peritonitis.

Postsurgical Care

Urine scald of the skin may be prevented by daily cleaning and applying zinc oxide ointment surrounding the incision or in the direction of urine flow. Systemic antibiotics may be indicated. Close monitoring of urine flow and observation for signs of reobstruction and uremia are recommended.

Patent Urachus

Indications

Persistent patent urachus may be congenital, characterized by failure of the urachus to close at the time of birth. However, it may also be caused by reopening of the urachus as a result of omphalophlebitis.[31] This distinction is critical to the prognosis. Perhaps it should be assumed that omphalophlebitis is present, with the patient treated accordingly with systemic antibiotics.

Anatomy

The urachus connects the fetal bladder with the allantoic cavity of the fetal membranes, which serves as the receptacle for fetal urinary excretory products until after birth. The urachus is a thin-walled duct lined with columnar epithelium. Within the umbilical cord, the urachus is surrounded by two arteries and two veins. All four vessels are equally thick walled, and it is not possible to differentiate them grossly except by dissection to their origin within the neonate body.

Position

Both lateral and dorsal recumbency are appropriate.

Presurgical Preparation

The umbilical stump should be cleansed and disinfected with povidone-iodine solution.

Anesthesia

This procedure is frequently performed with physical restraint only. Infiltration of local anesthetic agents produces edema and possibly obstructs details of the surgical site. Furthermore, inflammatory response at the surgical site may negate the effects of local anesthetic agents.

Sedation with xylazine, or even general anesthesia, may be necessary if the umbilical stump has ruptured close to the body wall and it is necessary to explore into the abdominal cavity to locate and ligate the urachus.

Surgery

If the urachus remains patent at birth, it is possible to ligate the entire umbilicus or localize the urachus and ligate it directly. A patent urachus that develops a few days after birth is an indication that an umbilical infection has reopened the urachal stump. Vigorous therapy and surgery are then necessary to avoid the complications of omphalophlebitis and septic arthritis.

The neonate should be prepared for general anesthesia and the umbilicus evaluated. The surgeon should be prepared to perform a laparotomy to remove all infected tissue. The urachus should be traced to the bladder and amputated. The opening at the bladder should be closed with a double-layered Lembert or Cushing pattern.

Umbilical vessels should be identified and ligated proximal to healthy tissue and the diseased segments removed. Infection may have traveled along the umbilical veins to the liver. In this case, it is recommended that the infected tract be marsupialized to provide drainage and access for irrigation with povidone-iodine solution. Broad-spectrum antibiotics are appropriately administered when infection is present. The neonate should be observed for evidence of urethral patency. If the owner observes urine issuing from the penis or vulva, no further treatment is indicated, but urine output should be carefully monitored following ligation to ensure that urethral flow is adequate.

Catheterization should be avoided, especially through the umbilicus, because the area is always contaminated and the risk of introducing potential pathogens into the abdomen is great. Also, the risk of trauma caused by catheterization of the neonate outweighs possible benefits.

MUSCULOSKELETAL SYSTEM

Space limitation precludes detailed descriptions of all the orthopedic procedures that may be performed on camelids. Even though few procedures have been described in camelid literature, experience and discussions with colleagues make it apparent that many procedures are performed routinely, using techniques described in standard surgical textbooks for horses, cattle, and dogs. The approach in this chapter is to describe unique problems, especially to emphasize anatomic variations of SACs from other species.[29,59]

Limb Amputation[39,41]

Indications

Both fore and hind limb amputations have been performed on llamas. The usual indication for such surgery is irreparable trauma to the limb, such as may be caused by shatter fractures, dog bites or other severe lacerations, inoperable tumors, nonresponsive osteomyelitis, or vascular lesions resulting in gangrene of the distal limb.

Surgery

Limb amputation is not described in any of the current large animal surgery texts. However, an excellent illustrated description of such surgery in the dog is found in Newton and Nunamaker.[33,43]

Insufficient numbers have been operated on to establish precise recommendations of preferred sites for amputation of the camelid limbs. In general, the author recommends the midhumerus as the site for forelimb amputation and that the hind limb be amputated at the distal third or midfemur. In large animals, amputations below the carpus or tarsus usually result in trauma of the stump caused by the animal trying to step down onto the shortened limb. Little muscle mass is available to provide padding around the end of the bone if the amputation site is the radius or tibia.

Postsurgical Care

Easy access to feed and water is necessary until the animal becomes accustomed to three-legged ambulation. That camelids adapt well to amputation is indicated by the fact that one pregnant female successfully carried a fetus to term following a hind limb amputation.

Complications

The contralateral limb may be incapable of withstanding the additional weight and extra burden of locomotion. If so, the fetlock may overextend and the collateral ligaments of the fetlock carpus or tarsus may become weakened. Infection and wound dehiscence are also possible complications if asepsis is inadequate.

Fracture Repair[53]

Fractures in camelids are caused by the same types of trauma reported in livestock, horses, and small animals.[32] Any of the fixation techniques practiced in other domestic animals are applicable in appropriate situations in camelids.[23,24] The size of llamas and alpacas places them between small animals and horses in terms of appropriate techniques. Camelids tolerate orthopedic surgery and application of various orthopedic devices well, so selection of a particular procedure should depend upon the bone involved and the nature of the fracture, available anesthesia, equipment and instrumentation, and the skill and experience of the surgeon.

Although sophisticated procedures such as compression bone plating and various pinning techniques are valuable for many fracture repairs, they may be contraindicated in certain cases in which less sophisticated procedures are valid and effective. The Schroeder-Thomas splint has been used successfully on radial fractures. A walking bar, incorporated into a coaptation splint, may be considered for fractures below the carpus or tarsus.

Anatomic Considerations for Orthopedic Surgery

Though surgical approaches to the long limb bones are basically similar to those performed on dogs, horses, and cattle, although there are slight variations.[23,32,41,44,47] A description of recommended approaches follows.

Humerus

The distal humerus should be approached from the lateral aspect of the limb, directly over the bone. The skin and fibrous sheath over the muscles should be incised. The depression between the lateral head of the triceps brachii muscle and the brachialis muscle should be palpated to incise the fascia or reflect the muscles away from the bone with blunt dissection.

The radial nerve curves diagonally around the caudal and lateral aspects of the distal third of the humerus. Prolonged pressure on this location with the animal in lateral recumbency may cause postsurgical radial paresis or paralysis.

The proximal humerus may be approached from the craniolateral aspect of the limb just cranial to the deltoid tuberosity of the humerus. The biceps brachii muscle may be reflected away from the cranial border of the humerus. If the caudal border must be exposed, it is necessary to transect the attachment of the deltoideus muscle from the deltoid tuberosity.

The major blood supply to the forelimb is the median artery, which lies on the medial aspect of the

humerus but is protected from it by muscle tissue. Fractures of the humerus are less likely than femoral fractures to result in transection of major blood vessels.

Radius

The radius is superficial throughout its length on the medial aspect of the limb. To expose the bone, incise the skin and the heavy subcutaneous fascial sheath and reflect the extensor carpi radialis muscle from the cranial border of the radius along with the major vessels and nerves to the lower limb. The flexor carpi radialis muscle is firmly attached to the caudal border of the radius.

The medial aspect of the radius is rounded, providing a suitable surface for attaching a plate or inserting a pin. The proximal medial surface is narrow. If a plate is needed here, it must be placed on the cranial surface of the proximal radius.

Metacarpus

Both the medial and lateral aspects of the metacarpus are superficial. However, the major blood supply to the foot lies on the medial side; thus, this is the approach that should be used to allow visualization of the vessels. The extensor tendons may easily be reflected out of the way. The flexor tendons, suspensory ligament, vessels, and nerves are encased in a heavy fibrous sheath, which must be incised to expose the bone. The two nutrient foramina, one for each marrow cavity, are located slightly proximal to the middle of the bone.

A large deep palmar metacarpal vein lies between the palmar aspect of the metatarsus and the suspensory ligament. When the suspensory ligament is to be transected surgically, such as to release contracture of the ligament, this vein should be isolated and reflected away from the ligament before an incision is made.

Femur

To expose the femur, the skin should be incised on the lateral aspect of the limb directly over the femur. The fascia lata lies immediately beneath the skin and should be incised in the same direction. The fascial sheet should be identified and incised between the biceps femoris muscle (caudal) and the vastus lateralis muscle (cranial). The biceps femoris muscle fibers run diagonally to the long axis of the limb, and those of the vastus lateralis run vertically.

The femur may be palpated at the distal end, but more proximally it is covered by the vastus intermedius muscle on the lateral and caudal borders. To completely expose the femur, it is necessary to sever the attachment of the vastus intermedius muscle on the lateral and cranial borders of the bone.

The sciatic nerve lies 3 to 5 cm caudal to the femur, well out of the way of the surgical site. The femoral artery and veins lie medial to the caudal border of the femur. Because of the close proximity of these vessels to the femur, they are at high risk of laceration in a fracture of the femur. Exsanguination into the soft tissue and death have resulted from lacerations caused by a femoral fracture in the horse, and presumably, this could occur in camelids.

Closure should include placement of sutures in each layer incised, especially in the fascia lata.

Tibia

The tibia should be approached from the medial aspect. The tibia is free of muscle covering throughout its length on the medial side of the limb. Incising the skin and fascial layer exposes the bone. The cranial tibial muscle lies on the cranial border of the tibia and can be reflected out of the way. The deep digital flexor muscle is firmly attached to the caudal border of the proximal two-thirds of the tibia. This attachment must be released to completely free the tibia.

The major blood vessels and nerves to the lower hind limb lie in the fascia between the gastrocnemius muscle and the deep digital flexor muscle and thus are well out of the way. The medial aspect of the tibia is rounded in the distal half but flattened in the proximal half, providing a suitable site for attachment of bone plates or the insertion of Kirschner/Ehmer pins.

Metatarsus

Both the medial and lateral aspects of the metatarsus are superficial and accessible for surgery. However, the major blood and nerve supply lies on the medial side, so this approach is more desirable.

Exposure may begin by incising the skin and reflecting the extensor tendons dorsally. The flexor tendons, suspensory ligament, blood vessels, and nerves of the lower limb are firmly attached to the plantar aspect of the metatarsus by a heavy fibrous sheath. The sheath must be incised to expose the bone. The major blood vessels lie on the medial side in the groove between the suspensory ligament and the deep digital flexor tendon. At the level of the junction between the middle and distal third of the metatarsus, the vessels and nerves leave the sheath and swing around to the plantar aspect of the flexor tendons, continuing distally in the space between the paired tendons, ligaments, and digits of the foot.

There appears to be only one metatarsus, as metatarsi 3 and 4 are fused. However, there are two marrow cavities, with a nutrient foramen for each, slightly dorsal to the middle of the metatarsus on the plantar aspect.

Tendon/Ligament Contraction[1]

Indications

Trauma to a tendon or muscle may cause contracture and subsequent extension or flexure of the joint involved. The types of contraction most often encountered involve the digital flexor tendons and carpal flexors. Congenital carpal flexure has been observed in neonates with multiple birth defects. In one neonate, an intrauterine tibial fracture had apparently been caused by a severe contracture of the tarsal flexor tendon.

Diagnosis

The posture of the animal is disturbed. Contraction of the carpal flexor tendons prevents proper extension of the carpus and results in a buck-kneed stance. Contraction of the digital flexor tendons may also produce a slight flexion of the carpus or act in concert with the carpal flexors to accentuate the problem.

The deep digital flexor tendon inserts on the flexor process of phalanx (P) 3. Contracture causes the fetlock to be flexed, and the llama must walk on the tips of the toes.[51]

The superficial digital flexor tendon inserts on the distal end of P-1 and the proximal end of P-2. Contracture increases the flexion of the fetlock, causing the pastern to become more vertical, but the llama need not walk on the tips of the toes.

Diagnosis is enhanced by palpation of the tendons under maximum flexure or extension. The degree of tautness can be felt.

In one case, the tendon of the interosseous muscle (suspensory ligament) of the hind limb developed a contracture.

Anesthesia

General.

Surgery

The flexor tendons of the lower limb in both fore and hind limbs should be approached from the medial aspect. The major blood vessels lie on the medial aspect of the metacarpus and metatarsus. The flexor tendons and the suspensory ligament are encased in a heavy fibrous sheath, which must be incised to provide access to the tendons and the ligament. Blood vessels and nerves should be reflected from the surgical field.

A blunt, straight bistoury should be inserted beneath the affected tendon and the cutting edge turned outward to transect the tendon while it remains under pressure. The tendon should be transected only to the degree necessary to release the contracture to allow the limb to resume a normal position. The skin is sutured and a pressure bandage applied for twenty-four hours.

If both the digital flexor tendons must be severed to correct the flexure, other techniques may be required (see equine surgery texts). Arthrodesis of the metacarpalphalangeal joint has been performed in a llama.

Angular Limb Deformity[28,55]

See Chapter 22 for a discussion of this condition as a congenital defect.

Indications

This surgery should not be performed to correct a defect on a breeding animal unless there is good evidence that the condition is the result of trauma or a nutritional disorder. Excessive bowing of the limb may incapacitate an animal that otherwise could be a suitable pet or packer. To be effective, this surgery must be performed during the active growing phase, while the physis is still open. Although the distal radial physis may be open as shown radiographically until after 2.5 years of age surgery after fifteen months of age is unlikely to be effective. The normal neonate carpus is illustrated in Figure 6.25.

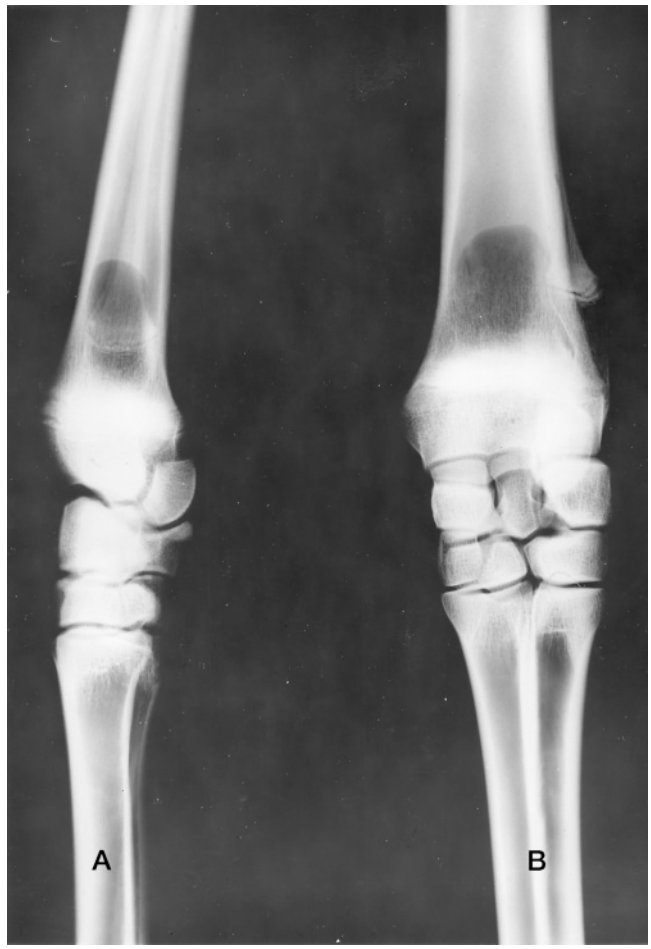

Figure 6.25. Lateral and dorsopalmar radiographs of a normal neonate llama carpus. (A) Ulnar physis, (B) radial physis.

Presurgical Preparations

Current radiographs should be available to aid in localizing the surgical site.

Anesthesia

General.

Surgery

Two methods have been used to correct angular limb deformities. One involves the placement of cortical bone screws on either side of the physis, bridging across the physis with stainless steel wire.[12] The other is periosteal stripping.[1–5] Both techniques were developed in equine surgery, and both have been used effectively in llamas. Each is described.

Transphyseal Bridging[37]

Advantages

The surgeon has control of the degree of correction. No tendons or major blood vessels are involved at the surgical site.

Disadvantages

Special instruments and screws are required. A second surgery is necessary to remove the screws. Either the physis or the carpal joint may be traumatized by improper placement of the screws.

Surgery

The physis is the most prominent palpable landmark. A 4-cm vertical incision is begun 2.5 cm dorsal to the medial physis and extended to 1.5 cm ventral to the physis. The skin and subcutaneous tissue are incised to the periosteum. The tendon of the flexor carpi radialis muscle lies immediately caudal to the surgical site and, abutting it, courses the cephalic vein. The medial collateral ligament of the carpus originates just dorsal to the physis. The incision should have penetrated the origin of the ligament parallel to its fibers. The tendon of the long digital abductor muscle crosses diagonally, cranial and ventral to the site.

The metaphyseal screw should be placed approximately 1 to 1.5 cm above the physeal prominence. After the hole is drilled, the depth should be checked with a depth gauge.

The required size of the cortical bone screw varies with the size of the llama. In animals less than three months old, the screws should be 3.5 mm in diameter and 20 to 22 mm long. The hole for this size screw should be drilled with a 2-mm drill and tapped with a 3.5-mm tap. Larger and older llamas require a 4.5-mm screw that is 22 to 24 mm long. The drill should be 3.2 mm and the tap 4.5 mm.

The epiphyseal screw is placed centrally in the epiphysis. The holes should be drilled slightly toward the physis to minimize the possibility of penetrating the carpus and to add strength to the set of the screw. Proper placement of the screws should be confirmed with a dorsopalmar radiograph of the carpus. After both screws are loose-set in position, stainless steel wire (0.89 mm in diameter) is used to place a Figure-8 wrap around the screws. The wires are twisted together, with the twisted tips of the wires bent to lie parallel with the strands of the wire. The screws are then tightened, and the tapered head of the screw pulls the wires taut. If the wire is not taut, growth must take up the laxity, prolonging the time necessary to straighten the leg. Proper placement of the screws is illustrated in Figures 6.26 and 6.27.

A layer of simple interrupted absorbable sutures should be placed in the subcutaneous fascia to cover the screw heads. The skin may be closed with nonabsorbable suture using a simple interrupted pattern.

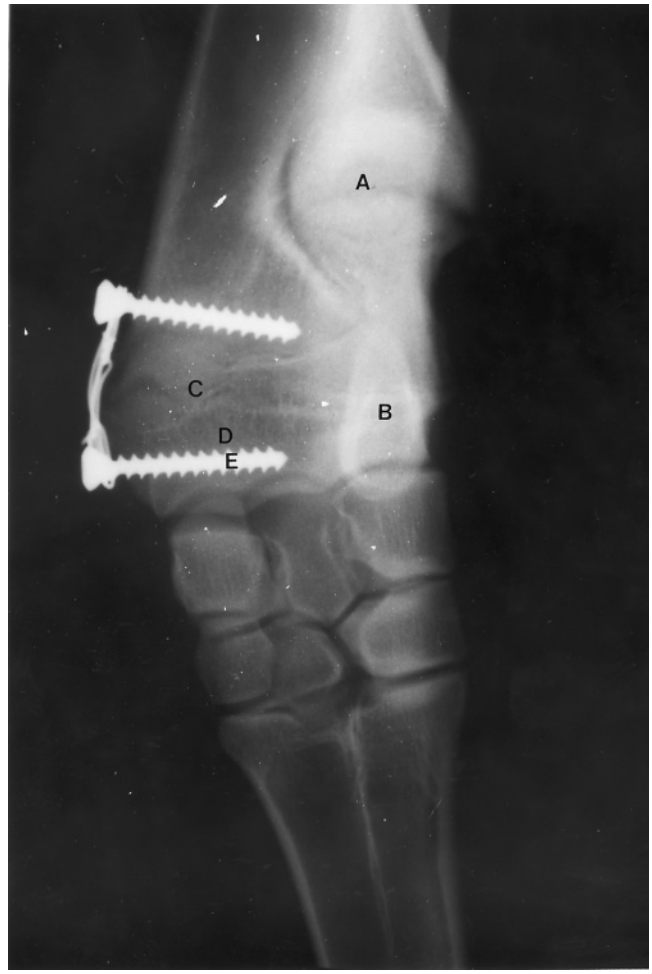

Figure 6.26. Dorsopalmar radiograph illustrating proper placement of cortical bone screws. (A) Clubbed ulnar physis, (B) accessory carpal bone, (C) radial physis, (D) radial epiphysis, (E) cortical bone screw.

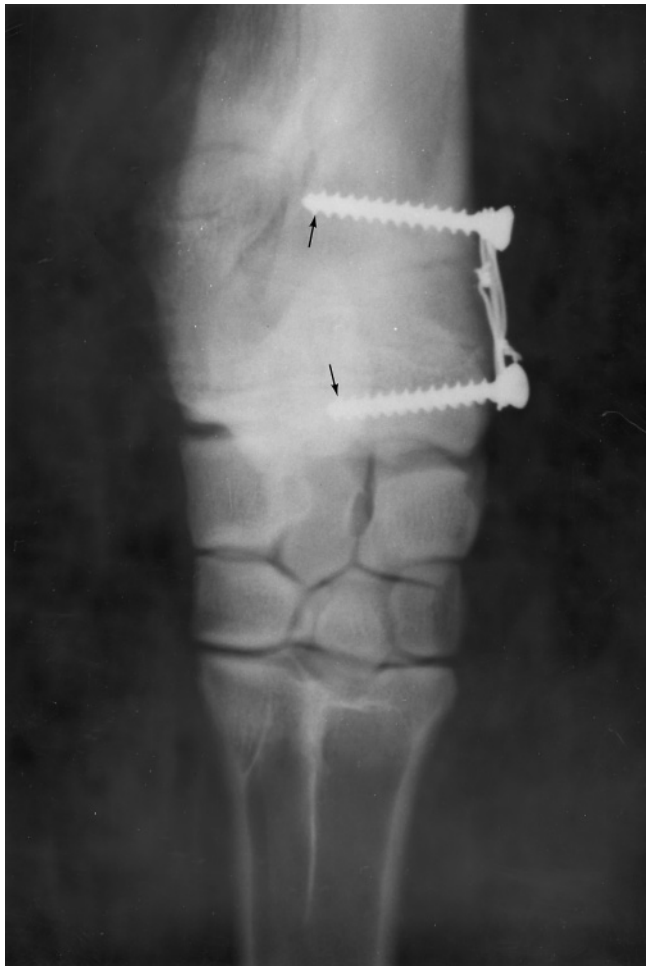

Figure 6.27. Normal divergence of cortical bone screws left in place for thirty to sixty days in a case of carpal valgus.

Postsurgical Care

The leg(s) should be bandaged to protect the incision for seven to ten days. With severely bowed legs, the carpi are in contact while walking, which will abrade the incision site if the legs are not bandaged. A standing bandage on the lower leg will aid in keeping the carpal bandage in place. Antibiotics are not necessary.

The screws should be removed when the angular limb deformity has been corrected. The time involved may be as short as six weeks in a two-month-old llama or as long as six months in an older animal. The limb should not be allowed to overcorrect. The llama must be sedated or anesthetized to remove the screws, and aseptic techniques should be followed. A stab incision over the head of each screw permits insertion of the hexagonal-tipped screw driver to twist out the screws. After both screws have been removed, the wires may be retrieved with a needle forceps. Each incision should be closed with simple interrupted sutures.

Complications

Little or no correction will take place if physeal growth has halted. If the animal is not monitored, overcorrection is possible. A seroma may form at the incision, or the wound may become infected. In either case, removal of one or two of the ventral sutures may establish drainage. The incision should not be fully opened to expose the heads of the screws.

No postsurgical pain or lameness should occur. If pain or lameness is observed, penetration of the carpal joint by the lower screw should be suspected. Epiphysitis may result if either of the screws penetrates the physis.

In one case, the owner failed to notice overcorrection.[12] The tension on the wire avulsed the epiphysis into the physis and initiated epiphysitis. The distal screw and the wire became embedded in the inflammatory fibrous reaction, making removal of the screw extremely difficult.

Periosteal Stripping[1,3,4,5,12]

In 1982, a new technique, called hemicircumferential transection of the periosteum and periosteal stripping, was reported for the correction of angular limb deformities in foals.[2] The basic premise postulated that tension across the physis may be brought about by using the periosteum as a fibroelastic tube, uniting the proximal and distal epiphyses.[2] If greater tension is exerted on one side than on the other, the growing leg will bow away from the side with the least growth. A bowed leg can be produced experimentally by performing a periosteal transection in a healthy foal.[1]

This technique has been successfully applied to llamas. However, the anatomy of the radius and ulna of the llama differs from that of the horse. In most but not all llamas, the distal ulnar epiphysis is fused with the radial epiphysis. The ulnar physis is approximately 35 cm dorsal to the radial physis. A thin section of the ulnar epiphysis wraps around the lateral radius and fuses with it. Radiographically, the radial physis appears to extend entirely across the bone; dissection has shown that this is not so.

The ulnar physis is abnormal in angular limb deformity and seems to be the structure that inhibits normal growth of the limb.

Carpal angular limb deformity is the most common form seen in llamas and alpacas, but angulation of other joints should be evaluated to determine whether the following methods have application. If there are significant changes in the joint surfaces or of the bones in a joint, surgery may be contraindicated.

Advantages

No specialized instruments or screws are required. Surgery can be performed quickly, and a second

surgery to remove screws is not necessary. There is less risk of infection with this technique because there are no bone implants.

Disadvantages

There is less control over the degree of correction. To date, insufficient numbers of llamas have been operated with this technique to accurately evaluate the extent of transection or stripping that is desirable. The common and lateral digital extensor muscle tendons lie in the immediate vicinity of the surgical field.

Surgery

The incision should be made dorsally over the prominence of the ulnar physis on the lateral aspect of the radius for a distance of 6 cm between the tendons of the common and lateral digital extensor muscles. A hemicircumferential inverted T incision is made through the periosteum approximately 1 cm dorsal to the physis. A periosteal elevator is used to strip the periosteum away from the bone, but no periosteum is actually removed.

The ulna has no weight-bearing function in the llama, and in several cases, the ulna was transected with a surgical saw following periosteal elevation. A bone chisel was used to physically separate the distal ulnar metaphysis from the radius beyond the ulnar physis. Ulnar physectomy has been a satisfactory procedure to augment periosteal stripping.

The periosteum is left open. The subcutaneous fascia is closed with a simple continuous pattern using absorbable sutures. The skin may be closed with a simple interrupted or vertical mattress pattern using nonabsorbable suture or vicryl. A light bandage should be placed over the carpus and changed in three days. The bandage may be removed in seven to ten days.

Complications

The main complication is failure of adequate correction of the angulation. If this occurs, a second periosteal stripping can be performed, or cortical bone screws may be used. In severe deformities, both physeal stripping and transphyseal bridging may be necessary to achieve correction.

Wedge Osteotomy

Severe angular limb deformity in animals past the growing stage has been made serviceably sound by wedge osteotomy.[34,45]

Fracture of the Mandible in Camelids

Etiology

Transverse fractures of the mandible, rostral to the cheek teeth, are most commonly seen in male dromedary camels during the rutting season. They may be caused by other male camels biting the victim or from the victim biting inanimate objects. Fractures may also be caused by automobile accidents, falls when landing on the jaw, or other external traumas in both male and female camelids.

Clinical Signs

There is an obvious malalignment of the incisor teeth and lower lip with the caudal mandible. Usually the fracture is compounded. The camel is unable to prehend feed. Abnormal movement of the jaw and crepitation confirm the diagnosis. Radiographs should be taken to establish the severity of the fracture and determine the presence of comminuted bone fragments.

Anesthesia

The selection of an anesthetic agent and the degree of anesthesia are determined by the docility of the camel and the method to be employed in immobilizing the fracture.

Management of Mandibular Fractures

The management protocol may range from a simple cradle slung beneath the mandible and strapped to the head to sophisticated orthopedic procedures using bone plates and screws to immobilize the bone segments. The selection of a given procedure may be based on the temperament of the camel, ability of the caretaker to provide after-care, access to orthopedic surgery, willingness of the owner to assume the expense of a procedure, and the skill and experience of the surgeon. Additionally, the presence of a compound, comminuted fracture and the length of time between the injury and obtaining medical assistance may be factors.

Slings for supporting the fracture have been constructed of wooden sticks, light metal, plastic casting material, and heavy fabric or canvas with straps over the maxilla and behind the ears.

Two methods of wiring have been used.

1. The mouth is cleansed appropriately and the camel sedated in the sternal position. Either stainless steel or copper wire, (1 mm in diameter and one m long) is used. A small intramedullary bone pin is used to drill a hole between the first and second cheek teeth just above the gum line. The wire is folded double and the free ends are inserted through the hole. Then the wire is inserted between the base of the central incisors.

   The bone segments are reduced and the wire ends brought around the intermediate and corner incisors. The two ends of the wire are twisted together and tightened. The excess wire

is trimmed and flattened against the corner incisor. The wire is left in place for fifty to sixty days.

2. Another wiring technique is as follows: One hole is drilled through the rostral segment of the bone from one side to the other and another hole through the caudal segment rostral to the cheek teeth. A stainless steel mattress suture is used to bring the bone fragments together. A second series of holes may be used to reinforce the first suture. The mucous membranes are sutured with an absorbable suture material.

Assume that infection is present with any compound fracture. Preoperative preparation must include a thorough cleansing of the operative site, preferably with a water pick, to dislodge debris. A temporary water pick may be constructed using a 20- or 60-ml syringe and a blunted 22- to 24-gauge needle. Alternate pressure and release on the plunger gives a pulsating spray that is highly effective in cleansing tissue.

Postsurgical Care

A person with much experience in dealing with these fractures recommends feeding 10 to 12l of cow's milk for nourishment for two weeks, then gradually adding green feed. Ramadan, another surgeon, recommends feeding green feed, bran, pellets soaked in water, or alfalfa meal and molasses. A broad-spectrum antibiotic should be administered. Deal appropriately with abscesses that may develop.

The oral cavity should be irrigated daily to rinse out residual feed particles.

Compression bone plating may be used. It requires specialized equipment and supplies in the hands of a qualified orthopedic surgeon.[39]

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[b] Dr. Art Kennel, M.D., Rochester, Minn., personal communication, June 1996.

7

Infectious Diseases

Infectious diseases play a prominent role in the production of camelids.[58–61,74,85,91,92,99,100,140,152,184,207] Prevention, diagnosis, and treatment are vital to the management of these animals.

It is appropriate to reiterate here that camelids are not ruminants and should not be so classified by regulatory officials. There are considerable differences in the susceptibility of camelids and ruminants to many of the diseases of concern to world animal health officials. As an example, although clinical foot-and-mouth disease (FMD) has been reported in both Old World camelids (OWC) and New World camelids (NWC), it is rarely seen, even in countries with endemic FMD. There is no evidence that camelids are carriers of the virus for more than a few days and there are no recorded incidents in which camelids have been the source of infection for cattle, sheep, goats, or swine, all of which are highly susceptible to FMD. This has been demonstrated by experimentally-produced FMD in OWC[209] and NWC.[116,117,185]

VIRAL DISEASES OF CAMELIDS

Table 7.1 is a list of abbreviations used in this chapter.

The prevalence of viral diseases in camelids is unknown. A few clinically important viral diseases have been reported. Investigators have reported positive serologic test results, indicating exposure and antigenic response to several viruses.[152,164]

Whether or not camelids are susceptible to a number of important viral diseases of cattle and sheep is still unknown.[58] Perhaps camelids have not been exposed to these viruses, or clinicians and researchers may not have conducted adequate virologic testing to discover them. For example, rinderpest has not been reported in South American camelids (SAC). Rinderpest does not occur in South America, but llamas have been experimentally challenged and found susceptible to the rinderpest virus. It is known that Old World camels are susceptible to rinderpest infection, and there is reason to believe that all members of the order Artiodactyla are susceptible to this virus, at least to some degree.

Viral diseases of cattle not known to occur in camelids, and without known positive serology, include malignant catarrhal fever (bovine herpesvirus III), bovine leukemia (bovine leukemia virus), cowpox, pseudorabies, and bovine papilloma. Similarly, diseases of sheep that have not been reported in camelids include ovine progressive pneumonia (ovine leukovirus), sheep or goat pox, balanoposthitis, sheep and goat papilloma, and scrapie.

Table 7.2 provides an overview of known viral diseases of camelids. Current knowledge of camelid viral diseases is rudimentary. Comparison of SACs with camels is important to indicate possible familial susceptibility. Clinicians should be alert to recognize the presence of undiagnosed viral diseases.

Rabies

Rabies is a viral encephalitic disease endemic in many areas of the world. Like all other mammals, camelids are susceptible to this rhabdovirus. The virus has been studied extensively because of its zoonotic aspect and its high mortality rate once clinical signs appear. While rabies does not occur in Australia, other lyssaviruses do and they may cause a syndrome similar to that of rabies.

Epidemiology

Rabies virus is primarily spread by bites from infected animals (Table 7.2). The virus has been found in saliva and other body excretions, but it cannot

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Table 7.1. Abbreviations used with infectious diseases.

CP	Camelpox
CE	Contagious ecthyma
VS	Vesicular stomatitis
FMD	Foot-and-mouth disease
BT	Bluetongue
HD	Hemorrhagic disease
BVD	Bovine viral diarrhea
EHV-1	Equine herpesvirus type 1
EEE	Eastern equine encephalitis
TB	Tuberculosis
JD	Johne's disease
EMC	Encephalomyocarditis
WND	West Nile disease
AF	Alpaca fever
FA	Fluorescent antibody staining
IHC	Immunohistochemistry
EM	Electron microscopy
PCR	Polymerase chain reaction
ELISA	Enzyme linked immunosorbent assay
CELISA	Competitive ELISA
MAP	Monoclonal antibody panel
USDA	United States Department of Agriculture
APHIS	Animal and Plant Health Inspection Service
PPD	Purified protein derivative
OT	Kock's old tuberculin
PMP	Preventive medical program
IM	Intramuscular
SC	Subcutaneous
ID	Intradermal
IP	Intraperitoneal
IV	Intravenous
CF	Complement fixation
AGID	Agar gel immunodiffusion
SRID	Single radial immunodiffusion
GI	Gamma interferon
VI	Virus isolation
VN	Virus neutralization
RT-PCR	Reverse transcriptase PCR

penetrate the unbroken skin. Infected mouse brain suspensions have been instilled into the conjunctival sac of alpacas without producing disease.[182,197]

Reservoir hosts vary with location. In Peru, the dog and fox are considered to be reservoir hosts. In tropical South America and Mexico, the vampire bat (*Desmodus rotundus*) is a serious threat to livestock production. In the United States, rabies is controlled in dogs and cats by vaccination, but wild species serve as a reservoir, Table 7.3. The striped skunk (*Mephitis mephitis*) is the major wildlife host in the western United States. In the Southeast and along the eastern seaboard to the New England states, the raccoon (*Procyon lotor*) has become a serious threat to domestic and zoo animals and humans. In the Midwest, there are foci in which two species of fox, red fox (*Vulpes vulpes*) and grey fox (*Urocyon cinereoargenteus*), are the reservoir hosts. Thirty-nine species of insectivorous bats inhabit the United States. The rabies virus has been isolated from thirty of these species. It is unlikely that all thirty species serve as vectors, but it does emphasize the fact that the rabies virus is circulating in bat populations.

In many areas of the United States and Canada, rabies is endemic in one or more reservoir hosts. Because camelids are susceptible, they should be protected against infection by appropriate vaccination.

In one instance in Peru, twenty alpacas from a herd of 160 were bitten by a rabid dog; thirteen died or were euthanized in extremis. The incubation period was as short as fifteen days in one animal, twenty-two days in two, twenty-four days in one, and thirty-one to thirty-four days in nine animals. Affected animals died six to eight days after the development of clinical signs.

In another instance, twenty-nine of a herd of 330 alpacas developed rabies. Dogs were implicated in this case as well. Dogs were not seen biting the alpacas, but rabid dogs were known to have been in the area and in one instance had attacked a herder. The losses occurred over a three-month period beginning about fifteen days after the suspected exposure. One neonate was involved in this outbreak. Transmission of rabies from alpaca to alpaca as a result of bites has also been reported.[48] Experimental rabies has been produced.[185]

The author is aware of six llamas that have died of rabies in the United States.[9,10,11,109,111,160] It is significant that rabies has developed from exposure to all of the major strains of the virus in the United States.

Rabies has been reported in dromedary camels in all dromedary raising countries. The reservoir host and transmitting host are not always known, but are presumed to be the dog and red fox (*Vulpes vulpes*) in the United Arab Emirates (UAE). There is no mention of the possibility of bat-associated rabies.

Clinical Signs[63]

Early signs of rabies in camelids include lameness, ataxia, and posterior paresis. Early signs are followed by either an aggressive syndrome (furious rabies) or a paralytic syndrome (dumb rabies).

In the aggressive form, a normal body temperature is usually recorded until the animal becomes aggressive and there is excessive muscular activity. The major signs in this form are attacks on pen mates, offspring, people, and themselves. The rabid animal may also bite inanimate objects, chew foreign objects, and become self-destructive by pushing into and over obstacles. Vocalization changes (alarm cries without cause) and other signs that have been observed during the course of the disease include bloat, pruritus, muscle tremors, aimless running, sexual hyperactivity (spontaneous ejaculation), recumbency, convulsions, coma, and death in one to four days. Females may refuse to

Table 7.2. Viral diseases of camelids.

Virus	*Family*	*Disease*	*SACs*		*Camels*	
			Clinical disease	*Serologic response*	*Clinical disease*	*Serologic response*
Orthopox virus	Poxviridae	Camel pox	Experimental	+	+	+
Parapoxvirus	Poxviridae	Contagious ecthyma, sore mouth, orf	+	+	+	+
Rabiesvirus	Rhabdoviridae	Rabies	+	+	+	+
Vesicular stomatitis	Rhabdoviridae	Vesicular stomatitis	+	+	?	?
Bovine herpesvirus 1	Herpesviridae	Infectious bovine rhinotracheitis	0	+	?	?
Equine herpesvirus 1	Herpesviridae	Equine rhinopneumonitis	++	+	0	0
Foot and mouth disease virus	Picornaviridae	Foot and mouth disease	+	+	+	+
Rinderpest	Paramyxoviridae	Rinderpest	0	0	+	+
Orbivrus	Reoviriade	Bluetongue	?	+	0	+
Bovine virus diarrhea	Togaviridae	Bovine virus diarrhea	+	+	+	+
Phlebovirus	Bunyaviridae	Rift Valley fever	0	0	0	+
Influenza A	Orthomyxoviridae	Influenza A	0	+	+	+
Parainfluenza III	Parmyxoviridae	Pneumonia	0	0	0	+
Borna virus	Bornaviridae	Borna disease	+	+	0	0
Papilloma virus	Papomaviridae	Papilloma, warts	0	0	+	+
Rotovirus	Reoviridae	Virus diarrhea	0	+	0	0
Coronavirus	Coronaviridae	Virus diarrhea	+	+	?	?
Adenovirus	Adenoviridae	Pneumonia	+	+	?	?

+ = susceptible, 0 = not reported, ? = no information.

Table 7.3. Rabies in llamas in the United States.

State	*Vector*	*Virus strain*	*Clinical syndrome*
Alabama	Raccoon	Raccoon *(Procyon lotor)*	Aggressive
Minnesota	Skunk	Skunk *(Mephitis mephitis)*	?
Oklahoma	?	Skunk *(Mephitis mephitis)*	Aggressive
New York	?	Red fox *(Vulpes vulpes)*	Aggressive
Texas	?	Grey fox *(Urocyon cinereoargenteus)*	Aggressive
Washington	Bat	Silver-haired bat *(Lasionycteris noctivagans)*	Aggressive

permit nursing. Camels characteristically yawn in the terminal recumbency stage.

Paralytic rabies in camelids is characterized by anorexia, depression head droop, ptosis, tenesmus, salivation, circling, facial paralysis, mild fever (39.4°C [103°F]), flaccid muscles (face, anus, bladder), and pharyngeal/laryngeal paralysis.

Regulatory officials have expressed concern about the public health aspects of rabid camelids "spitting" on people, but apparently camelids suffering from either form of rabies are unable to regurgitate and spew stomach contents or saliva.

Diagnosis[110,111]

The rabies virus causes a nonsuppurative encephalitis with perivascular cuffing by mononuclear cells. Neuronal degeneration is seen. Camelids develop Negri bodies (intraneuronal, eosinophilic, cytoplasmic inclusions) in neurons of the hippocampus and other locations in the brain. Fluorescent antibody staining of neural tissue is usually the first screening test performed in most laboratories. Some of these have been positive in llamas. Previous experience with various wild animals leads to the conclusion that no single test should form the basis of a definitive diagnosis until experience has demonstrated its accuracy and reliability. Rodent inoculations with brain tissue are appropriate. Various viral strains are identified by a monoclonal antibody panel (MAP). Texas and New York conduct MAPs at the state level; other states submit samples of tissue to the Communicable Disease Center in Atlanta, Georgia. The coyote strain and grey fox strains are identical on a MAP but may be differentiated by polymerase chain reaction (PCR) or DNA sequencing.

Differential diagnosis should include head trauma, neural abscesses, meningeal worm, tick paralysis, other encephalitic diseases, hepatoencephalopathy, protozoal encephalitis, and copper deficiency.

Prevention

In the United States there has been a conflict between camelid owners and some state regulatory officials. In one New England state, camelids were essentially quarantined to the farm because there was no officially recognized vaccine for use in camelids. However, killed rabies vaccines have been used and are appropriate for immunization of camelids. Clinical research has demonstrated that llamas respond to killed vaccines with titers that are considered protective in other species (Table 7.3).[a] Owners should be informed that no vaccines have been officially approved for camelids, but Trimune (Fort Dodge) and Imrab (Rhone-Merieux) have been administered to camelids and are, at least, safe. Annual revaccination is recommended. Knowing that llamas have contracted rabies in a number of different endemic areas makes it unwise to refuse to vaccinate. A modified live virus (MLV) rabies vaccine should never be given to any animal unless it has been specifically approved for that species.

Following an outbreak of rabies in alpacas in Peru, a herd of 290 alpacas was vaccinated with an MLV vaccine.[48] Thirty of the alpacas (10%) developed post-vaccination paralysis within fourteen to thirty days.

Herpesvirus[206]

Herpesviruses are highly evolved and usually well adapted to one or more hosts. Interestingly, no herpesviruses unique to camelids have been identified. When herpesviruses infect a nonadapted host, serious disease or death is likely to result, which has occurred in llamas, alpacas and OWCs.[21]

Bovine herpesvirus type 1 was implicated in one llama ill with bronchopneumonia, but the authors reporting the case caution that further studies are necessary to confirm the etiologic diagnosis.[210]

Equine herpesvirus type 1 (EHV-1) produces rhinopneumonitis and abortion in horses. The virus is endemic in most equine populations within the United States, and vaccination is routinely practiced to control the infection. In 1984, blindness was diagnosed in twenty-one alpacas and llamas of a herd of approximately 100 animals. The condition was ultimately attributed to infection with EHV-1.[94,159,193]

Epidemiology

The affected herd had been imported into the United States from Chile and had undergone the prerequisite federal quarantine at the point of origin and further quarantine at the U.S. Department of Agriculture (USDA) quarantine station in Florida. All affected

animals had been kept in a large barn during a specific time period. Also housed in the barn during that time were two zebras that were sold shortly before the infection developed in the camelids. It was not possible to subsequently trace the whereabouts of the zebras to obtain confirmation of their involvement. However, the zebras were the only possible equine exposure. The means of transmission was unknown.

Three llamas were experimentally infected with an isolate of equine herpesvirus type 1 that had been isolated from the brain of a clinically affected alpaca.[88] Two of the llamas became clinically ill; one died and the other was euthanized. The virus was isolated from one llama, and lesions were typical of equine herpesvirus type 1 infection. The llama that lived developed antibodies typical of a primary immune response.[68]

Equine herpesvirus type 1 has been reported from a Bactrian camel and it should be assumed that dromedaries are also susceptible to the virus.

Clinical Signs

Blindness, with nonresponsive dilated pupils, was the primary manifestation in animals in the affected herd. Retinal degeneration and optic nerve atrophy were seen on ophthalmoscopic examination. Blindness was complete and irreversible. Four animals exhibited neurologic signs in addition to blindness. One animal died of encephalitic complications. Camels exhibit neurologic signs but not blindness.

Diagnosis

A herpesvirus, indistinguishable from EHV-1 virus, was isolated from four animals, and acute and convalescent serum titer evaluations demonstrated active infection in the blind animals. All individuals in the herd with a four-fold elevation in titer eventually developed blindness.

The lesions noted at necropsy included retinal detachment and hemorrhage, retinitis, choroiditis, vitreitis, and optic nerve degeneration. The presence of intranuclear inclusion bodies, typical of herpesvirus infection, was the first clue that led to the diagnosis.

Treatment

Antiviral therapeutic agents have been used in human herpesvirus infections and also in elephants, but to the author's knowledge these agents have not been administered to camelids. Although steroids and other medications were administered to llamas and alpaca, the retinal lesions were unresponsive.

Prevention

It is significant to note that this was an equine virus infection in an artiodactylid. With increasing production of camelids in North America and increased opportunity for association with horses, not only this virus, but other equine viral infections, should be considered in a differential diagnosis.

Vaccines are routinely used in horses. Killed vaccines were given to exposed camelids in the case described. The value of blanket vaccination of camelids with equine vaccines is questionable. Before such a step is recommended, a controlled trial should be conducted to determine both the safety and efficacy of the vaccine.

Each vaccine is prepared for a specific animal. Each animal responds characteristically. Both bovine and sheep vaccines have routinely been given to camelids, and it is more logical to use vaccines developed for ruminants than to use an equine vaccine without first exploring the ramifications of such a step.

Bluetongue[80]

Bluetongue was first described in domestic sheep but now is known to affect many species of ruminants.[57] There is serologic evidence that camelids respond to BTV with the formation of antibodies, but clinical disease is questioned.

Etiology

Bluetongue is an insect-transmitted viral disease caused by bluetongue virus (BTV) (*Orbivirus* sp. Family Reoviridae). BTV is antigenically related to the epizootic hemorrhagic disease virus of deer. BTV exists in a multiplicity of serotypes. The degree of cross-protection against heterologous serotypes has been shown to be limited and variable.

Epidemiology

Small, blood-feeding gnats of the genus Culicoides biologically transmit BTV. After a female gnat has taken an infective blood meal, the virus replicates in the salivary glands. Ten days later, BTV may be transmitted by the gnat while taking another blood meal. The gnat may live for twenty-eight days or more, feeding every three to five hosts.

BTV antibodies have been found in numerous species of wild animals and in alpacas,[156] but there is no evidence that alpacas are a reservoir for the disease in sheep. Twenty percent of the alpacas on one ranch in Peru had a positive antibody response to BTV.

BTV is probably present in most regions where sheep, cattle, and camelids coexist. This disease should be included in a differential diagnosis.

Clinical Signs

Clinical manifestations of bluetongue vary widely according to species. In sheep, it is characterized by fever, nasal discharge, pneumonia, congestion of the lips, oral and nasal mucosa and epithelium of the tongue, necrosis of the dental pad, edema of the ears, and necrosis of skeletal and cardiac muscle. Mouth

lesions may progress to shallow ulcers on the lips and gums. In a few individuals, a similar inflammatory response will occur on the coronary bands of the hoofs. Lameness is an early clinical sign in flocks of infected sheep on pasture.

Cattle may show fever, a stiff gait, drooling from the mouth, small ulcers on the oral and nasal mucous membranes and the dental pad, and/or nasal discharge. The muzzle may be hyperemic. The coronary bands may become inflamed and swollen, resulting in lameness similar to that seen with sheep. Reproductive abnormalities such as abortion, deformed calves, or unthrifty newborn calves have been associated with BTV infections in cattle. Also, many cattle apparently acquire an infection that is undetectable or not observed. Cattle usually recover seven to ten days after clinical signs appear.

The author has seen one suspected case in a llama. A pregnant female had an episode of respiratory distress followed by an abortion. Paired serum samples taken after the abortion demonstrated a four-fold increase in BTV antibody titer. A case of mortality in an alpaca was attributed to bluetongue in the United Kingdom.[80]

Diagnosis

Virus isolation and serologic tests are necessary for diagnosis. Gross and microscopic lesions are suggestive but must be differentiated from vesicular diseases and rinderpest. A competitive ELISA test was developed and tested in two llamas that had been experimentally infected with BTV. Both animals remained clinically normal but developed titers within two weeks postinoculation that remained high for the ten weeks of the experiment. The authors suggested that a 50% inhibition would be indicative of exposure to the virus in llamas in New Zealand.[4]

Treatment

Supportive therapy and administration of antibiotics to prevent secondary infections are recommended.

Prevention

Sheep are routinely vaccinated. Currently, available MLV vaccines are strain-specific and have not been tested for camelids. The use of these vaccines in camelids, with the present state of knowledge, is not recommended.

Contagious Ecthyma

Contagious ecthyma (CE) (contagious pustular dermatitis, sore mouth, orf) is a parapox viral disease (*Parapoxvirus ovis*, Family Parapoxviridae). CE primarily affects the epidermal structures of the nose and lips of sheep and goats. Other susceptible species include musk-oxen (*Ovibos moschatus*), wild sheep (*Ovis canadensis*), Rocky Mountain goats (*Oreamnos americanus*), camelids, and humans.[78,79]

Epidemiology

The natural reservoir host is probably the sheep. The virus may remain viable in crusts from a lesion for longer than a year. Transmission may be by either direct contact or insect vectors.

CE is a zoonosis that produces severe ulcerating lesions on the fingers, limbs, or face of affected people. A clinician should always wear rubber gloves while examining a suspected case. One case involved a llama farm employee who had recently returned from working on a sheep ranch in New Zealand. Although she had a finger lesion at the time, it was not recognized as CE. A month later, a number of llamas at the farm developed lesions.

Clinical Signs

Clinical CE occurs in both OWCs and NWCs. In Peru, classic lesions have been seen in alpacas at two to four months of age. Young camels are also typically susceptible animals. Typical proliferative epidermal lesions at the commissures of the mouth have been seen in camelids in North America and New Zealand (Figures 7.1 to 7.3) and in the UAE. The lesion may be chronic, characterized by thickening of an area of the skin of the face or the perineum. Such lesions must be differentiated from those of sarcoptic mange or ringworm.

A nursing cria or camel calf may develop lesions on the lips and transmit the virus to the teats. Because of the pain associated with a lesion on the teats, the dam may refuse to allow nursing.

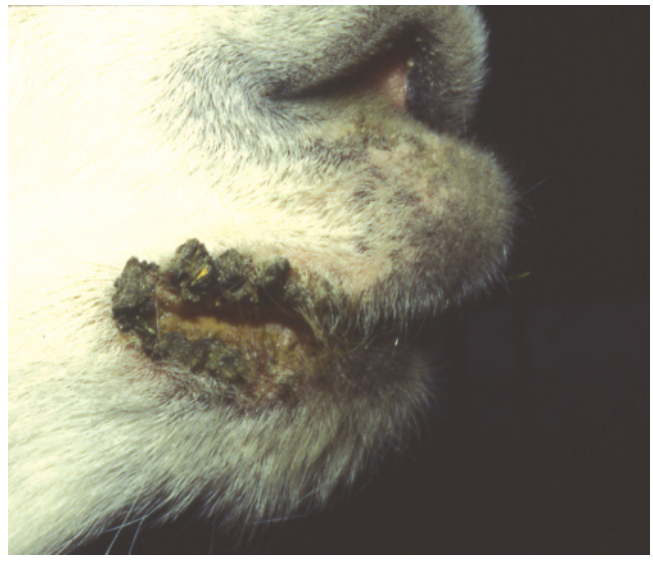

Figure 7.1. Contagious ecthyma in an alpaca.

Figure 7.2. Contagious ecthyma in an alpaca.

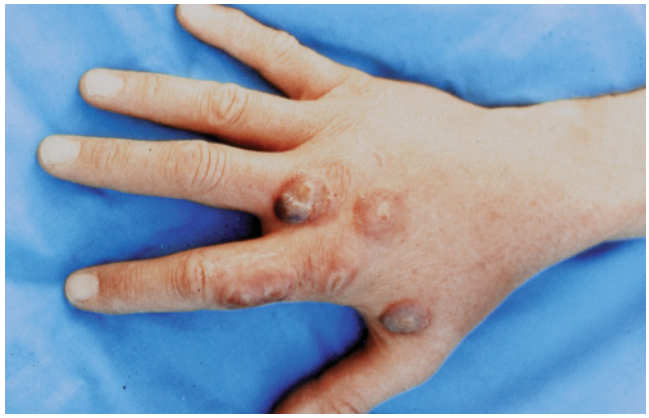

Figure 7.3. Contagious ecthyma (orf), a zoonosis.

In sheep and goats, CE is a self-limiting disease. In camelids, the lesions may persist for months.

Diagnosis[114]

The CE virus grows in tissue cultures but grows poorly on the chorioallantoic membrane of chick embryos. Microscopic lesions are diagnostic in early and acute cases.[80] Cytoplasmic inclusion bodies (4 to 8 μ) are found in swollen epidermal cells but disappear in older (six days or more) lesions.[80] Chronic lesions seen in camelids may be difficult to diagnose. One of the forms of zinc-responsive dermatosis is a hyperkeratotic lesion of the lips, and this condition should be considered in a differential diagnosis in SACs. Dermatophylosis should also be included in a differential diagnosis.

Treatment

No specific treatment is recommended. Nonspecific immunostimulation with such drugs as levamisole have been attempted.

Prevention

Modified live virus (MLV) vaccines are routinely used in sheep and goats, but these vaccines are risky and may be unwise to use on camelids. Faced with an outbreak of CE in a herd, a clinician would have to weigh risks against possible benefits.

Any sheep or goats kept with camelids should be vaccinated and all traces of vaccinal lesions gone before such animals are allowed to mix with camelids.

Foot-and-Mouth Disease

Foot-and-mouth disease (FMD) (aftosa, aphthous fever, hoof-and-mouth disease) is a highly contagious viral disease, primarily of cattle, sheep, swine, and goats, but also affecting other artiodactylid domestic and wild animals. It is characterized by vesicular lesions and, subsequently, erosions of the epithelium of the lips, gums, soft palate, nares, muzzle, coronary bands, interdigital spaces, teats, and rumen pillar. Degenerative necrotizing lesions may have been observed in the myocardium of calves.

Etiology

FMD is caused by *Aphthovirus* sp. (Family Picornaviridae). Seven immunologically distinct types of FMD virus (FMDV) are known. Within the seven types, more than sixty subtypes have been identified over the years by complement fixation tests.

Epidemiology[56]

FMD occurs in many countries but not in the United States and Canada. Mexico and other Central American countries have stringent regulations to prevent introduction of FMDV. FMD is of interest to camelid owners because the virus is present in many of the countries in which camelids are indigenous. Many species of artiodactylid are susceptible to FMDV, but they vary markedly as to whether or not clinical manifestations develop. Some species (e.g., the African buffalo [*Syncerus caffer*]) may harbor the virus in the pharynx for as long as twenty-eight months, but vesicular lesions do not develop.[57]

Many species of wild animals seem to be affected when an epizootic of FMD occurs in an area of Africa, but no susceptibility studies have been conducted. There are anecdotal reports, but no confirmed records, of natural infection in camels.[188] Experimental infections of FMD in camels have been reported.[144] However, in an epizootic of FMD in Ethiopia, where camels were in intimate contact with cattle, they neither developed lesions of FMDV nor produced antibodies to the strains involved.[212]

During a severe epizootic of FMD in Peru approximately fifteen years ago, Peruvian regulatory

veterinarians dealing with cattle reported that similar lesions had been seen in alpacas.[123] Also in Peru, a case of FMD in alpacas was confirmed by laboratory test (type A24) in 1971.[106] A similar type of isolation made in llamas in Peru that same year was not reported in the literature.

One study of camelid susceptibility to FMDV was conducted in 1952.[123] One vicuña, nineteen alpacas, and sixteen llamas were exposed to FMDV by various methods, including scarification of the tongue, intradermal inoculation of the tongue, intramuscular (IM) and intravenous (IV) injection, and cohabitation with animals infected by other routes.

Animals became infected from all routes of exposure. Four animals died in the groups given IM or IV injections. The incubation period from exposure to the development of lesions was forty-eight to seventy-two hours. The first manifestations were vesicles on the tongue, with generalized vesicular formation occurring elsewhere within three to four days.

The four llamas exposed by cohabitation developed no lesions, but these same animals became infected when inoculated intramuscularly. Cattle exposed by cohabitation suffer 100% morbidity. One of the alpacas in the cohabitation experiment developed lesions in three days, another in five days, and a third in ten days. Two other alpacas failed to develop lesions through cohabitation, but did so twenty days later when injected intramuscularly.

It was concluded from this Peruvian study that camelids are susceptible to the virus, but less susceptible than cattle and sheep. In a study conducted at the USDA National Foreign Animal Disease Laboratory at Plum Island, New York,[b] transmission was shown to be possible from the bovine to llamas and vice versa.[91,92,93] The virus could not be isolated from llamas after fourteen days following infection.

A more definitive study involving dozens of llamas was conducted in Argentina as a cooperative project with the Argentine government and the U.S. Department of Agriculture. Previous findings were confirmed, demonstrating that llamas are quite resistant to infection with the FMD virus and do not remain carriers.

The route of field transmission may be by respiratory aerosol, direct contact with infected animals, or exposure to contaminated feed and water. FMDV may be inhaled in aerosol droplets by people working around infected animals. The virus lodges in the pharyngeal mucosa and may be exhaled for as long as twenty-four hours post exposure. Thus, humans may contribute to the spread of FMD. Actual FMDV infection in humans is rare, and the disease is not considered to be a public health risk.

Clinical Signs

The incubation period varies from three to five days. In cattle, early signs include fever, depression, and anorexia. Stomatitis causes salivation, and lameness is caused by lesions on the coronary bands and interdigital spaces. Pain may cause the animal to tread, shake or kick out the feet, or lie down. Ultimately, the vesicles rupture, forming erosions, and a mucopurulent nasal discharge may develop. Pregnant cows may abort, and calves may die acutely. The overall mortality rate in cattle is usually less than 5%, but 50% of affected calves may die from myocardial degeneration.

Experimentally infected camelids in Peru developed fever up to 40°C (104°F). This was the first sign noted in all affected animals. They became totally anorectic approximately twenty-four hours following inoculation and chose to lie down most of the time. Vesicle locations and progress of the lesions followed patterns seen in cattle and sheep.

In the North American study, the affected llamas were depressed but had no fever. Vesicles formed on the tongue, lips, dental pad, coronets, and interdigital glands. When the vesicles ruptured, the erosion healed rapidly. Footpads became undermined.

Diagnosis

The diagnosis of any suspected vesicular disease is under the control of state and federal regulatory veterinarians in the United States and comparable authorities in Canada, Mexico, and other countries. Suspected cases must be reported promptly. Suspect facilities must be quarantined until a diagnosis is confirmed or disproved by a variety of sophisticated laboratory tests. A provisional diagnosis will be made within twenty-four hours of receipt of lesion material at the diagnostic laboratory. Specific vesicular diseases cannot be differentiated by clinical signs nor at necropsy. Rinderpest must also be included in a differential diagnosis.

Pathology

Vesicles, followed by erosions, may occur on the tongue, dental pads, gums, cheeks, hard and soft palates, lips, nostrils, muzzle, coronary band, interdigital space, teats, and stomach pillars. Degenerative, necrotizing myocarditis may cause death, especially in young animals. Because camelids are also susceptible to experimental inoculation with vesicular stomatitis virus, this disease must be considered in a differential diagnosis.

Treatment

There is no treatment for FMD. By law in the United States, both infected and exposed animals must be

euthanized. This practice is also followed in Canada, Mexico, and Central America.

Prevention

Vaccines are used to control FMDV in livestock in countries in which FMD is enzootic. Vaccines are strain-specific and must be prepared for a given geographic area. Vaccination is illegal in North America.

Vesicular Stomatitis

Vesicular stomatitis (VS) (erosive stomatitis, pseudo aftosa) is another vesicular disease; it has a broader host range than FMD, including horses and omnivores. VS is enzootic in a number of locations in the western hemisphere.

VS virus is classified as *Rhabdovirus* sp.(Family Rhabdoviridae rhabdovirus). There are two major types, New Jersey and Indiana. There is no known reservoir host. It is possible that this virus primarily attacks a plant or an insect and only secondarily infects mammals.

Epidemiology[172,208]

Definitive information on the epidemiology of VS is not complete. Epizootics occur seasonally in various geographic locations, affecting horses, cattle, swine, and deer in the United States. One natural case of VS in camelids has been reported. Experimentally, alpacas and llamas are susceptible to intradermal inoculation into the dorsum of the tongue, which produces localized vesicles.[73] Other methods of inducing infection were unsuccessful. Attempted exposure routes included IM injection, rubbing the virus on the surface of the tongue, and cohabitation with infected animals. Only 50% of those given intradermal inoculations developed vesicles (in forty-eight to ninety-six hours).

Fluid taken from vesicles of camelids was infective to susceptible cattle. Camelids that were unaffected in the cohabitation trials were subsequently susceptible to intradermal tongue inoculation a month later.

No reports exist on VS in OWCs.

Clinical Signs

Clinical signs of VS are indistinguishable from those of FMD.[189] In experimental llama cases, a transient, slight elevation in temperature (0.5°C or 0.9°F) occurred twenty-four to forty-eight hours after inoculation, followed by anorexia. The animals became recumbent and developed vesicles. No foot lesions were seen. In the naturally infected llama, signs were similar to those described for experimental cases.

Pathology, diagnosis, and treatment are the same as for FMD. Animals should be treated supportively.

Borna Disease

Borna (equine encephalomyelitis) is a viral disease primarily of horses and donkeys and is restricted to localities in central Europe. This disease was diagnosed in a group of SACs that died at a zoo in Erfurt,[4,5] and at the Leipzig zoo[171] in Germany. OWCs may also be affected. The Borna virus (*Bornavirus* sp., Family Bornaviridae) is an arbovirus that may be transmitted by the tick *Hyalomma anatolicum*. Oral transmission in horses has also been demonstrated, and the virus has been found in saliva, urine, feces, milk, and nasal secretions.

The host range is unknown, but young horses appear to be the most susceptible, and donkeys are more resistant. Sheep and cattle have been involved in natural outbreaks. Animals may be viremic for long periods with inapparent infections. No reservoir host has been identified.

Clinical Signs

Llamas and alpacas at the Erfurt zoo exhibited anorexia and weight loss. Death occurred acutely or within three weeks.[6] Horses developed ataxia, pharyngeal paralysis, muscle tremors, spasms, and blindness. The anorexia shown by SACs may have been neurologically induced.

Diagnosis

In the outbreak at the Erfurt zoo, camelids developed no neurologic signs, and cerebral spinal fluid and the brain were not examined in early cases of the disease. Diagnosis was confirmed by histopathologic evaluation of later cases. Histologic changes are typical of a nonsuppurative encephalomyelitis.[100] Intranuclear inclusion bodies, known as Joest-Degen bodies, are diagnostic and are especially prevalent in the hippocampus. Diagnosis may also utilize virus isolation, intracerebral inoculation in rabbits, immunohistochemistry, and indirect immunofluorscence in infected cell cultures.

Treatment and Prevention

Treatment was not effective in the sick camelids. An MLV vaccine was administered to all SACs at the Erfurt zoo in 1972.[6] A month later, an alpaca died, and Borna disease was confirmed by histopathology. It is impossible to determine whether the death of this alpaca was caused by a natural infection or was vaccine induced. However, all SACs at Erfurt now receive yearly vaccinations, with no outward reaction.

Rinderpest[23]

Rinderpest is a highly contagious disease of artiodactylids characterized by fever, lymphocytopenia, erosive stomatitis, gastroenteritis, and diarrhea.

Etiology

Rinderpest is caused by *Morbillivirus* sp. (Family Paramyxoviridae), closely related to the viruses that cause canine distemper and human measles. Several different strains vary in virulence, but all are immunologically indistinct.

Epidemiology

OWCs are susceptible to rinderpest. Rinderpest has never appeared in North America and only once in South America, in Brazil in 1921. It is a devastating disease, and veterinarians dealing with such animals as camelids that may be shipped around the world must have a basic understanding of rinderpest.

Natural hosts for rinderpest virus probably include all members of the order Artiodactyla. Natural rinderpest has never been reported in SACs. Limited experimental studies at the Federal Animal Disease Diagnostic Laboratory, Plum Island, have shown that llamas develop a mild febrile response, running a short clinical course, with recovery in three to five days.[c] Old World camels are susceptible. Primary hosts for this virus on a worldwide basis are cattle, water buffalo, and domestic swine, but no reservoir host has been determined.

Transmission of rinderpest is by direct contact. All animal discharges contain the virus, and the infection route is either via the respiratory tract or by ingestion. No carrier state of rinderpest has been established in cattle. Clinically affected animals shed the virus for two to three weeks if they survive.

Clinical Signs

In cattle, rinderpest is characterized by a high fever (41°C or 105.8°F), anorexia, inflammation, and necrosis of the mucous membranes, with hyperemia of buccal mucosa and development of erosions but not vesicles or ulcerations. There is little bleeding from the erosions. Diphtheritic membranes may develop on the muzzle. Constipation precedes a severe, fetid diarrhea. Pregnant animals may abort.

The incubation period is three to nine days. The disease course is three to four days in acute cases and up to twelve days in less severe cases. Animals may recover after a prolonged convalescence.

Diagnosis

Rinderpest is a reportable disease. Clinical signs may be confused with FMD or VS. Suspect cases should be reported to governmental authorities, after which differential diagnosis will be carried out under their direction. Isolation of the virus, animal inoculation, and serologic procedures are required for definitive diagnosis.

Pathology

Erosions and ulcerations occur throughout the digestive tract. Hemorrhage of serosal surfaces occurs with the septicemic form of the disease. Microscopically, destruction of lymphocytes and epithelial cells of the digestive tract may be seen.

Treatment and Prevention

In North America, affected and exposed animals must be euthanized. Vaccines are used for disease control in other enzootic areas but are illegal in North America.

Bovine Virus Diarrhea

Bovine virus diarrhea (BVD) is a serious disease of cattle, sheep, goats, and wild ruminants. Heretofore BVD was considered to be of little consequence to camelids[d] but recent findings that both SAC crias and OWC calves may become infected in utero and become persistent carriers has changed that perception.

Etiology

BVDV is a small RNA virus (Family Flaviviridae). The genus is *Pestivirus* sp. Other species of *Pestivirus* are the causal agents of border disease in sheep and classical swine fever (hog cholera).

Epidemiology[189,199]

If a pregnant female is exposed to the virus during the first trimester of pregnancy the fetus may become infected.[18] The infection may kill the fetus and cause abortion or cause congenital birth defects, stillbirth, low birth weight, or weakened crias or calves. If the cria survives it becomes persistently infected and viremic. Though it does not show signs of BVD, it is a source of infection for herd mates.[18,32,34,50–53,126,142,190,202]

Postnatal infection with BVDV is by ingestion or inhalation of contaminated material.

Clinical Signs

Abortion is the primary clinical sign of BVD in camelids. Additionally, stillbirths, weak neonates, failure to thrive, congenital deformities, respiratory distress with a chronic mucopurulent nasal discharge, and gastroenteritis with diarrhea have been reported.

Diagnosis

Laboratory techniques include immunohistochemistry, virus isolation, and RT-PCR. Serologic tests such as ELISA detect antibodies against BVDV and may be used for screening a herd of cattle or camelids for determining whether BVDV is active in the herd.

Management

Prevention of BVD necessitates strict biosecurity. The practice of taking a female with her cria to a farm

for breeding is risky. Both the dam and cria should be subjected to a battery of tests to detect antigens and antibodies. Persistently infected crias are a source of infection for any comingled animals naive to BVDV.

Both live and inactivated vaccines are used in dairy and beef cattle herds. Live vaccines are not recommended for camelids, but inactivated vaccines have been used in NWCs.

Camelpox

Camelpox is the most frequently seen infectious disease of both dromedary and Bactrian camels,[205] and has been produced experimentally in NWCs. The disease primarily affects younger animals.

Etiology[201]

The infective agent is *Orthopoxvirus cameli* (Family Poxviridae, Subfamily Chordovirinae). Camelpox virus is a large, complex DNA virus having a brick-shape. There are numerous strains of the virus with differing degrees of virulence.

Epidemiology

Transmission is via viral exposure to abrasions or breaks in the skin, aerosols, or mechanical transmission by biting insects or other arthropods (mosquitoes, ticks). Recovery from the disease confers life-long immunity.

Camelpox is found throughout the Middle East, Asia, North Africa, Pakistan, Russia, and India, but not in the United States nor in the feral camel population in Australia.

Clinical Signs

The incubation period is nine to thirteen days. In mild cases pustules develop on the muzzle, nostrils, eyelids, oral mucosa, and nasal mucosa. The pathogenesis of the pox lesion starts as an erythematous macule, progressing into a papule and then a vesicale (Figure 7.4A). The vesicle changes into a pustule with a depressed center and a raised erythematous border. When the pustules rupture they become encrusted.

More severe cases are characterized by pox lesions over the entire body and limbs, accompanied by fever, anorexia, depression, and diarrhea (Figures 7.4B to 7.7). Lesions may occur in the trachea and on the surface of the lung. Abortion has also been reported. Acute camelpox has been produced experimentally in a guanaco (Figure 7.8). Secondary infection may complicate the clinical picture. Healed lesions leave patches of alopecia or a scar (Figure 7.9).

Chronic camelpox may occur when a mild case develops in an immunodeficient individual. The lesions may take four to six weeks to heal with or without scars.

Figure 7.4A. Camelpox, acute, disseminated. Photo courtesy of Dr. U. Wernery, U.A.E.

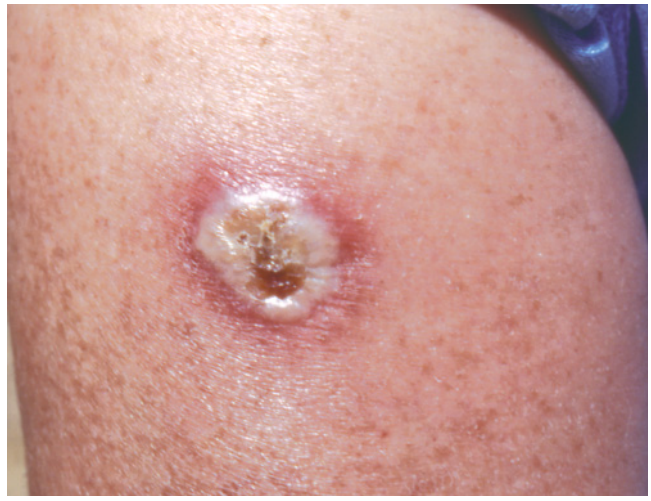

Figure 7.4B. Individual pox lesion.

Diagnosis

The clinical signs are characteristic, but a differential diagnosis must include lesions caused by a species-specific papilloma virus (warts) and parapoxvirus (contagious ecthyma). Laboratory diagnosis may include inoculation of embryonated chicken eggs, cell cultures, and inoculation into rabbits. Newer methods include ELISA, monoclonal antibody panels, and DNA restriction enzyme analysis and DNA probes.[202] A definitive diagnosis may be accomplished by electron microscopy, immunohistochemistry, and PCR technology.

Management

There is no specific treatment for camelpox. In the generalized form it is recommended to administer

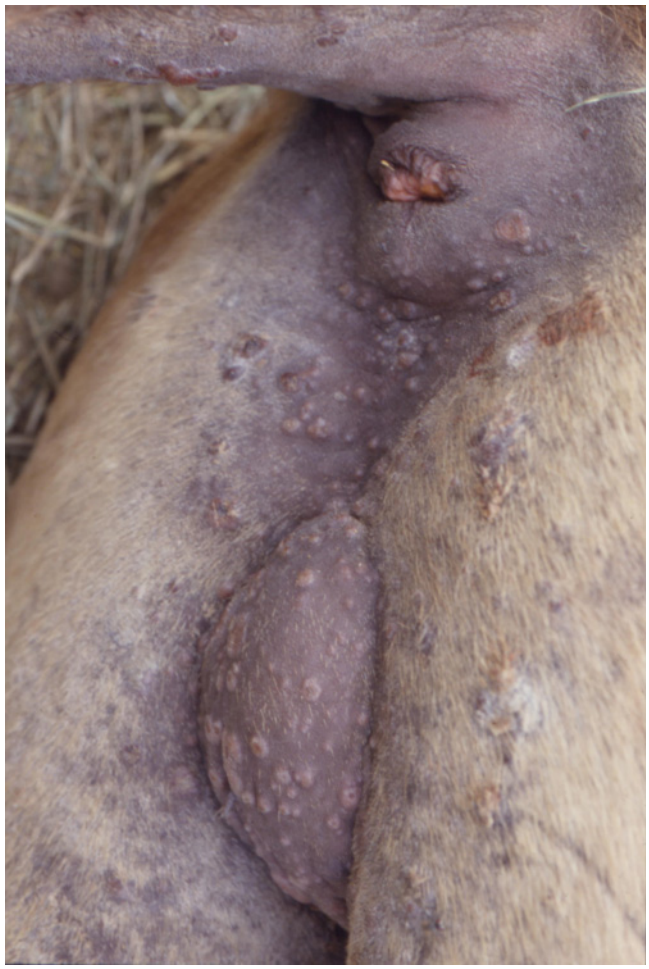

Figure 7.5. Camelpox, acute, scrotum. Photo courtesy of Dr. U. Wernery, U.A.E.

Figure 7.7. Camelpox, subacute.

Figure 7.8. Camelpox, healing lesions.

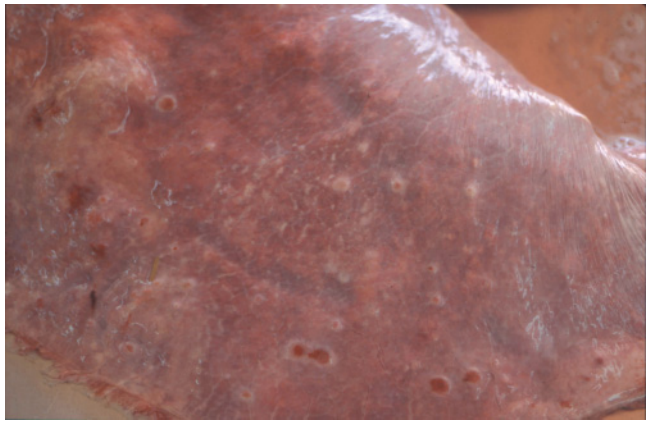

Figure 7.6. Camelpox, acute, lung lesions. Photo courtesy of Dr. U. Wernery, U.A.E.

Figure 7.9. Camelpox experimentally produced in a guanaco.

broad-spectrum antibiotics to minimize secondary infections.

A vaccine, Ducapox®, has been developed and used successfully in the United Arab Emirates to prevent camelpox. Recombinant DNA technology may be used in the future to produce vaccines.

Papillomatosis

Papillomas (warts) occur on a variety of animals worldwide. In humans, the papillomavirus initiates cervical cancer. In most animals the lesions are a benign neoplastic proliferation of the skin epithelium and mucous membranes. Warts usually occur in camels less than two years of age.

Etiology

Papillomas are caused by a species-specific papillomavirus (Family Papoviridae).

Epidemiology

Papillomas have been reported only in OWCs. The disease is rare, and is of little economic significance in camels. The lesions may be difficult to differentiate from camelpox and contagious ecthyma in the early stages of the disease.

Papillomatosis is found in the Middle East,[105] North Africa, and India. Transmission is by direct contact of abrasions of the skin or mucous membranes with active lesions on other animals. Handling equipment may become contaminated if used on infected animals. Insects may be responsible for mechanical transmission.

Clinical Signs

The lesion is characterized as a round cauliflower pedunculated mass varying in size from 0.3 to 4 cm in diameter. The papilloma do not affect the general health of the camel. The mature wart is not likely to be confused with camelpox or contagious ecthyma, but the early lesion is a hyperemic elevation of the skin similar to the macula stage of camelpox.

Diagnosis

Microscopically the lesion is a hyperplastic epithelium with excessive folding. Electron microscopy may identify viral particles. An immunohistochemistry method has become the definitive diagnostic technique.[105]

Management

The disease is generally mild and self-limiting and no treatment is necessary. However, in a herd outbreak, an autogenous vaccine was prepared and administered. Healing time was markedly diminished.

TOGAVIRUS INFECTIONS

Eastern Equine Encephalitis

Etiology

Eastern equine encephalitis (EEE) is caused by *Alphavirus sp.* (Family Togaviridae).

Epidemiology[145]

The EEE virus is transmitted by mosquitoes. The disease affects many species of mammal, including horses, humans, and NWCs. The virus is endemic in reservoir hosts which are birds, especially those found in freshwater swamplands in the western hemisphere. EEE is primarily seen in the eastern states of the United States, in Central and South America, and the Caribbean. Horses, humans, and NWCs are dead-end hosts.

Clinical Signs

Signs include fever, lethargy, ataxia, seizures, recumbency, torticollis or opisthotonus, and vestibular signs. Note that the signs are similar to any other encephalitic condition. With EEE there is no evidence of blindness in NWCs,[16] but mortality is high in NWCs.

Diagnosis

Antemortem diagnosis may be based on the seasonal occurrence (late summer, fall), signs, and prior history of EEE in the area. At necropsy PCR and immunohistochemistry are definitive. A differential diagnosis should include West Nile encephalitis, listeriosis, rabies, polioencephalomalacia, and other encephalitides. Regionally, meningeal nematodiasis must be considered.

Management

Supportive treatment is indicated. Mosquito control is helpful in some situations. Vaccines are used in horses, but no vaccines have been approved for camelids.

West Nile Virus Encephalitis (WNVD)[44,211,214]

Etiology

The etiologic agent is *Flavivirus* sp. (Family Flaviviridae).

Epidemiology

WNVD was first introduced into the United States in 1999. The disease has been present in Africa and the Middle East for decades. The virus is transmitted by mosquito bites. The reservoir hosts are wild birds. Dead-end hosts include horses, humans, camelids, and several other mammalian species, as well as zoo and domestic birds.

Clinical Signs

Camelids may have an inapparent infection, but when clinical disease occurs the mortality is high. Signs are similar to those occurring with EEE including inappetence, fever, lethargy, ataxia, stiff gaits, seizures, recumbency, torticollis or opisthotonus, and vestibular signs. Affected animals usually are unable to right themselves from lateral recumbency.[112]

Diagnosis

ELISA and hemaglutination inhibition (HI) are used antemortem and PCR at necropsy. Differential diagnosis should include EEE, listeriosis, rabies, polioencephalomalacia, and other encephalitides. Regionally, meningeal nematodiasis must be considered.

Management

Supportive treatment is indicated. There is no specific treatment. Mosquito control may help. Vaccines are used effectively in horses and humans. Killed vaccines have been used off label in alpacas.

Louping-Ill[40,121]

Etiology

Louping ill (*Flavivirus* sp.) is in the family Flaviviridae.

Epidemiology

The louping-ill virus is endemic to specific regions in Britain and the contiguous coast of continental Europe.

The infection is seen in many species of wild birds and mammals including sheep, humans, horses and camelids. The virus is transmitted by ticks, primarily *Ixodes rincinus*.

Clinical Signs

Louping-ill virus produces an encephalitis characterized by anorexia, lethargy, hyperexcitability, ataxia, goose-stepping gait, and progressive paralysis.

Diagnosis

Virus isolation and serology are used antemortem with immunohistochemical stain of CNS tissue at necropsy.

Management

Tick control by dipping and spraying is used in sheep as are vaccines. The disease is rare in camelids and no treatment regimen has been established.

Influenza A

Influenza is an ever-present threat to many species of birds and mammals including humans and camelids. The possibility of a global pandemic is of great concern to world health authorities.

Etiology

The influenza A viruses are in the Family Orthomyxoviridae. There are numerous subtypes and strains classified according to the animal most commonly affected and by twelve hemagglutinins (H) and nine neuroaminidases (N). Mutation of the virus is common and recombinations of strains is important in determining virulence.

Epidemiology

Volumes have been written on this subject. Suffice it to say that Bactrian camels in Mongolia have been infected with swine influenza A. Influenza has also been suspected in dromedary camels in the Middle East. It is likely that any camelid could be affected if exposed under the right circumstances.

Clinical Signs

Signs have been described in Bactrian camels including fever, anorexia, weakness, prostration, dyspnea, coughing, and ocular and nasal dischares. Severe case may progress to pneumonia, pulmonary edema, and death.

Diagnosis

The virus is isolated on tissue culture or embryonated eggs. Strains are identified by serology and DNA tests.

Management

No vaccines are available for camelids.

MISCELLANEOUS VIRAL DISEASES[142]

Adenoviruses have been isolated from five llamas and one alpaca with diarrhea in Oregon.[125] Another llama developed pneumonia and hepatitis associated with an adenovirus.[66] A coronavirus was isolated from an outbreak of enteritis in young llamas, and has been diagnosed in camels in the Middle East.[210]

Suspected Virus Disease

During the summer and fall of 2007 a new respiratory disease occurred in llamas and alpacas in the United States. The disease was given several names including camelid respiratory disease, alpaca respiratory disease, and upper respiratory disease. Owners called it "snots" based on the prominent nasal discharge.

Epidemiology

Outbreaks on farms and ranches were associated with animals returning from shows or sales. The out-

breaks were clustered in areas of the United States with high populations of SACs.

Clinical Signs

The rapid spread and highly contagious nature of the disease suggested a viral etiology. Generally the disease was mild and characterized by fever, nasal discharge, coughing, and increased breathing rate. Recovery was rapid. Signs of a more severe form included dyspnea, pulmonary edema, foaming from the nostrils, pneumonia, abortion, stillbirth, and death. The severe form was considered to result from secondary bacterial infection.

Management

Treatment was supportive and symptomatic. Prevention consisted of quarantine of ill animals and strict biosecurity.

Minor infectious diseases of OWCs and NWCs are tabulated in Tables 7.4 and 7.5.

FUNGAL INFECTIONS[146]

Dermatophytosis (Ringworm)[163,173]

Various dermatophytes produce superficial infection of the epidermis in many different species of animals. Fungal dermatitis is uncommon in camelids, but occurs in both NWCs and OWCs.

Etiology

Two species have been isolated from llamas at the author's clinic. *Trichophyton verrucosum* (Figure 7.10) is the common cause of ringworm in cattle and goats and more rarely in horses, donkeys, dogs, sheep, camels, and llamas. Arthroconidia are seen lining the surface of hairs in potassium hydroxide (KOH) preparations of material scraped from lesions. The arthroconidia are approximately 5µ in diameter. Microscopic chains of chlamydospores are typical (Figure 7.10).

T. mentagrophytes var. *mentagrophytes* (Figure 7.11) has also been isolated from skin lesions in llamas as well as from cats, dogs, cattle, sheep, pigs, horses, rodents, and monkeys. The organism has a ubiquitous geographic distribution. *T. mentagrophytes* var. *interdigitale* is the most common organism of human athlete's foot. Arthroconidia on the surface of hairs tend to cluster. Growth on artificial media is characterized by microconidia; macroconidia; coiled hyphae; and branched, septate hyphae. *Microsporum* spp. have also been isolated from llamas.

Species that have been isolated from OWCs include *Trichophyton verrucosum, T. mentagrophytes, T. schoenleinii, T. sarkisovii, T. dankaliense, Microsporum gypseum,* and *M. canis.*

Clinical Signs

Clinical cases may simply be alopecic areas with limited distribution on the head and neck or more generalized over the body and limbs. In the clinical cases seen, the lesions in the llama caused by both organisms were similar to those observed in cattle, with raised, crusty, circular plaques around the poll and face. The age of the lesions was unknown in the case reported, but the plaques varied from 2 to 4 cm in diameter. The extensive lesions illustrated in Figure 7.12 are ringworm superimposed on another dermatologic problem.

Epidemiology

The epidemiology of ringworm in camelids is as yet unexplored, but direct and indirect contact are the usual modes of transmission of dermatophytes. The chlamydospores of both *T. verrucosum* and *T. mentagrophytes* may remain viable for up to 4.5 years in hair and cellular debris scraped off the animal and left attached to barn walls, fence posts, trees, feed bunks, halters, blankets, packs, brushes, and combs. Other camelids and humans may become infected from contact with these fomites. Both organisms may be zoonotic, causing an acute inflammatory response on the scalp and other areas of skin, with possible scarring.

Pathology

Early lesions have not been observed in camelids, but in other species, the dermatophyte produces toxins and allergens that evoke an inflammatory response in the susceptible host, with erythema, exudation, and alopecia resulting. The organism tends to move peripherally toward unaffected tissue, enlarging the lesion in a circular pattern. In cattle, the lesion becomes thicker as it enlarges peripherally.

Diagnosis

Direct microscopic examination of hair and crusts taken from the periphery of a lesion is the most rapid method of diagnosis. The material should be placed on a slide and a few drops of 10% KOH solution applied and allowed to react for ten to fifteen minutes, preferably accompanied with gentle heating to facilitate clearing. Fungal spores may be observed clinging to hairs when viewed microscopically at 100×, and irregular, septate hyphae may be identified.

Both *T. verrucosum* and *T. mentagrophytes* may be cultured on Sabouraud's dextrose agar. In contrast with most dermatophytes, *T. verrucosum* grows better at 37°C (98.6°F) than at room temperature. Growth is slow, requiring ten to fourteen days before colonies become evident. Chlamydospores and mycelia are produced on unenriched media, but numerous

Table 7.4. Minor or rare infectious diseases of OWC, may only develop antibody titer.

Disease	*Etiology*	*Distribution*	*Species affected*	*Epidemiology*	*Signs*	*Diagnosis*	*Management*
Akabane virus disease	Orthounyavirirus	Australia	Camels and ruminants	Spread by midges	CNS anomalies of fetus		Insect control
Rift Valley fever	*Phlebovirus* sp., Family Bunyaviridae	Africa and Arabian Peninsula	Camels, livestock, humans, wildlife	Arthropods, heavy rainfall and flooding	Sudden abortion storms, fever, anorexia, nasal exudate, diarrhea, sudden death	RT-PCR, serology	RVF is a zoonosis and potential bioterrorism agent. Vaccination is used in livestock
Hemorrhagic septicemia	*Pasteurella multocida*	Worldwide	OWCs	Transmitted by direct contact, inhalation, ingestion	Dyspnea, fever, anorexia, diarrhea	Culture of the organism	Broad-spectrum antibiotics
Rinderpest	Paramyxovirus, closely related to canine distemper and measles		All species of artiodactylids including camels	Transmitted by direct contact, inhalation, ingestion	Fever, erosions of mucous membranes	Government will do it, differentiate from FMD, VS	Affected animals must be euthanized in U.S.,
Encephalitis blindness	Equine herpesvirus type 1, Family Herpesviridae	Worldwide	Horses primary, NWCs, OWCs	Camelids are an aberrant host, camelids with equids	Blindness, nystagmus ataxia, paralysis	VI, IHC, ELISA	No treatment, killed vaccines used
Adenovirus	Coronavirus				Pneumonia, diarrhea, hepatitis		
African horse sickness	*Orbivirus* sp., Family Reoviridae	Africa, Middle East, Spain	Equids, OWCs	Transmission by midges (*Culicoides* spp.) and ticks (*Hyalomma dromedarii*)	Fever, dyspnea, pulmonary edema, heart failure, usually subclinical in OWCs	VI, ELISA, AGID	No treatment, insect control
Bluetongue	*Orbivirus* sp., Family Reoviridae	Tropics, subtropics worldwide, now in Europe and U.S.	Primarily sheep, OWCs, and NWCs	Transmission by midges (*Culicoides* spp.)	Subclinical or rare, abortion, respiratory infection, hyperemia of MM	VI, ELISA, AGID	No treatment, control midges
Influenza	Influenza virus (Orthomyxoviridae)	Various strains worldwide, H1N1 human	Has only been reported in Bactrian camels	Inhalation	Fever, dyspnea, pneumonia	Virus isolation, DNA tests, serology	Isolation, symptomatic treatment
Plague	*Yersinia pestis*	Europe, Middle East, Asia	Camels have had bubonic, pulmonic, and septicemic form	Transmitted by fleas, ticks, and inhalation	Fever, buboes, dyspnea, pneumonia	Culture from lymph nodes, nasopharnyx	Broad-spectrum antibiotics, insect control
Melioidosis	*Burkholderia pseudomallei*	Tropical, moist areas in Australia, Indonesia	OWCs and alpacas; it is a zoonosis	Direct contact	Necrotic pneumonia and lung abscesses	Culture	Broad-spectrum antibiotics

Table 7.5. Minor infectious diseases of SACs.

Disease	*Etiology*	*Distribution*	*Species affected*	*Epidemiology*	*Signs*	*Diagnosis*	*Management*
Encephalo myocarditis	*Cardiovirus,* Family Picornaviridae	Worldwide	OWCs, NWCs, elephants and numerous wild animals, rarely humans	Rodents are the vectors, transmitted by fecal contamination of feed	Lethargy and reluctance to move, sudden death is most common	Signs, VI, SN, necropsy, myocarditis	Rodent control, sanitation, vaccination?
Eastern equine encephalitis	*Alphavirus*, Family Togaviridae	North, South, and Central America; Caribbean	Primarily equine, humans, SACs	Transmitted by mosquitos, and by aerosolation	Lethargy, ataxia, seizures, recumbency, fever, torticollis, opisthotonus	Signs, neutropenia, RT-PCR, IHC, histopathology, always consider rabies	Symptomatic therapy, vaccination with killed vaccine
West Nile disease	*Flavivirus* sp., Family Flaviridae	Africa, U.S., South America	NWCs, horses, humans	Transmitted by mosquitos	Lethargy, ataxia, seizures, fever	ELISA, VI	Potential bioterrorism agent, symptomatic, mosquito control
Pseudo-tuberculosis	*Yersinia pseudo-tuberculosis*, related to *Y. pestis*	Worldwide	SACs, birds, cats, rodents	Rodents are reservoir, transmission by ingestion	Lymphadenitis, gastroenteritis	Culture, may be in mixed cultures	Broad-spectrum antibiotics, rodent control

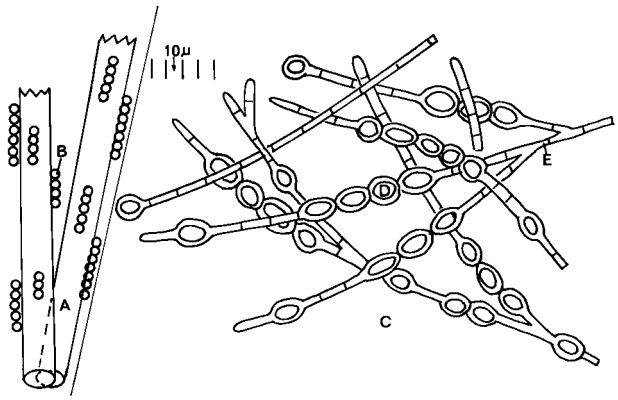

Figure 7.10. Diagrams for identification of *Trichophyton verrucosum*. (A) Fibers, (B) arthroconidia on the surface of the fibers, observed on KOH preparation of skin scrapings, (C) growth on artificial media, (D) chlamydospores, (E) septate hyphae.

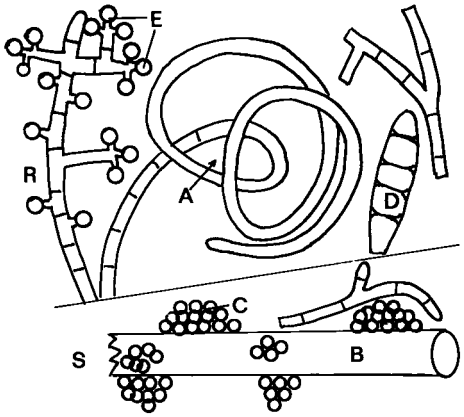

Figure 7.11. Diagrams for identification of *Trichophyton mentagrophytes*. (A) Coiled hypha, (B) fiber, (C) arthroconidia, (D) macroconidia, (E) microconidia, (R) growth on artificial media, (S) characteristics observed on KOH preparation of skin scrapings.

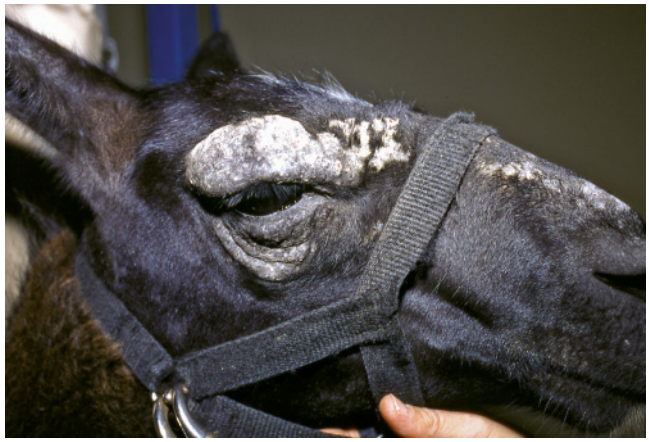

Figure 7.12. Ringworm in a llama.

microconidia and more regular mycelia grow on thiamine-enriched media.

A number of nonspecific and generally undiagnosed keratin-proliferative dermatoses have been seen in SACs. Any thickened lesion should be checked carefully, with multiple deep skin scrapings for dermatophytes and sarcoptic mange mites (Chapter 8).

Treatment

Lesions of ringworm are usually self-limiting in most species. Iodine (2% tincture) may be applied directly to a lesion daily for two weeks. A less caustic povidone-iodine preparation (diluted 1:4) may be equally effective. This product is used extensively to treat ringworm in livestock, horses, and dogs, and has been effective in treating camelids.

Captan[e] is a fungicide for ornamental plants. As a caution, captan has not been approved for use on animals intended for food, and rubber gloves should be worn by people mixing and applying the solution. Captan may be purchased as a 50% wettable powder from nursery suppliers. The recommended dilution is 2 tablespoons of the powder to 4 L (1 gal) of water. The mixture is stable for one week after mixing. The solution should be applied liberally to the lesion(s) and the surrounding area twice weekly for two weeks. The animal should not be rinsed off after application of the mixture.

Thiabendazole has antifungal action and may be applied as a 2% to 4% ointment to lesions of ringworm at three-day intervals. A popular but expensive treatment for ringworm is the application of Tresaderm solution.[f] The author has no experience with the use of oral griseofulvin in camelids. Vaccines are used in OWCs.

Candidiasis

Candidiasis (candidosis, moniliasis, thrush) is a common, sporadic disease of the digestive tract caused by a yeastlike fungal infection. The disease is seen in poultry and swine and, rarely, in dogs, cats, horses, and wild animals.[160,167] In humans, the disease causes glossitis in infants, skin infections, and vaginitis. In cattle, both mastitis and abortion have been reported. One case of gastric candidiasis has been reported in a neonatal llama in Europe.[59]

Etiology[108,113,168]

Candida albicans is the usual agent of infection, but other species have been isolated. Candida has a yeastlike phase, usually encountered when the infection is on the surface of a mucous membrane, and a mycelial or pseudomycelial form that occurs when the infection becomes invasive. Candida is not highly pathogenic and may be isolated in the normal flora of the digestive

tract. Candidiasis is probably an opportunistic infection.

Epidemiology

C. albicans is normally present on the mucous membranes of the alimentary and genital tracts of animals. The organism may be isolated from many lesions, in which it may be present as a commensal or secondary infection. The isolation of *C. albicans* should not be construed as establishing a diagnosis of candidiasis. In many species, the development of candidiasis frequently involves an immune-deficient host (neonate, chronic disease) or follows long-term antibiotic therapy. Transmission may be via ingestion of contaminated food or water, but most cases are probably endogenous, the agent having been acquired during or shortly after birth.[180]

One case has been reported in a neonate llama in a European zoo.[76] Another case was a chronic dermal infection in a gelded eight-year-old llama.

Clinical Signs

In the neonate the llama was anorectic, with a yellowish diarrhea of three days' duration prior to death at five days of age. The llama had been treated for enteritis with antibiotics, fluids, and electrolyte replacement therapy.

The adult llama had extensive areas of thick, coalescing crusts on the axillary, inguinal, and perineal areas and on the muzzle. The crusts could be removed, leaving the underlying skin moist and red and with a foul odor. Within the area of the removed crusts there were scattered small pustules. This llama was weak, had poor body condition, and was depressed.

Diagnosis

Antemortem diagnosis is difficult to establish. The organism can be isolated from normal animals, and clinical signs are nonspecific. At necropsy, a mucosal scraping stained with lactophenol cotton blue, new methylene blue, Wright's, Giemsa, or a standard Gram stain may show characteristic rounded or oval budding cells (blastospores) 3 to 6µ in diameter. The budding cells may be linked, forming a chain (pseudohyphae), with indentations between the buds. True mycelia may also be found.

Candida may be cultured in Sabouraud's dextrose broth or on Sabouraud's agar at either room temperature or 37°C (98.6°F). Standard texts describe other methods of culture for identification to species.

A differential diagnosis should include *Coccidioides immitis* and *Conidiobolus coronatus*.

Necropsy

In enteric infection, the walls of compartments 1 and 2 (C-1, C-2) of the stomach may be thickened and edematous. In one case, a grayish-white pseudomembrane in an irregular pattern 1 to several millimeters thick was found. A whitish fluid containing necrotic debris from the pseudomembrane was found within the stomach lumen. No pseudomembrane was seen in C-3. The mucosa was hyperemic to mildly inflamed.

Microscopically, the epithelium of the mucous membrane was necrotic and invaded by masses of pseudohyphae and budding yeast cells. The invasion rarely progressed beyond the basement membrane of the epithelium, and then only where deep ulceration had occurred. Yeast cells were best demonstrated in the tissue with a periodic acid-Schiff stain.

Treatment

In an adult llama with dermatitis, nystatin and chlorhexidine acetate ointments were applied topically to the affected areas. Healing occurred in sixty days. Because antemortem diagnosis is difficult and the disease rare, it seems unlikely that a treatment regimen can be properly developed. However, nystatin has been recommended for intestinal infection in pigs.

Prevention

Prevention of candidiasis can best be accomplished by minimizing predisposing factors such as stress and prolonged antibiotic therapy. Lack of response to antibiotic therapy after three to four days should prompt reevaluation of the treatment regimen. Cleanliness in the management of orphan camelids is crucial.

Aspergillosis[70,154,177,213,216]

Aspergillosis is an opportunistic fungal infection, rare in mammals but common in birds. The disease is characterized by an inflammatory granulomatous lesion, usually of the respiratory system. The disease has been reported in a disseminated form in an alpaca[151] and in OWCs.

Etiology

Aspergillosis is caused by *Aspergillus fumigatus* and other *Aspergillus* spp. An extensive body of literature deals with Aspergillus spp. The hyphae are 4 to 6µ in diameter and septate. Identification depends upon the presence of conidiophores, which are characteristic of the genus.

Epidemiology

A. fumigatus is a ubiquitous organism. Infection is rare in mammals, usually occurring when a patient is immunodepressed or has been under intense or prolonged stress. Transmission is by inhalation of fungal spores from a contaminated environment. The disease is not contagious.

Clinical Signs[31]

A young female alpaca in which disseminated aspergillosis was diagnosed had been imported into the United States and held in quarantine for a total of ten weeks before being examined for an apparent blindness (head tilt, intermittent circling, searching gaze, and widely dilated pupils with no pupillary light reflexes).[151,181] Retinal degeneration was identified by ophthalmoscopy. Further examination and laboratory evaluations failed to determine the etiology. Nonspecific steroid (30 mg prednisone daily) and amoxicillin therapy was administered for sixteen days, with gradual reduction of the steroid during the last ten days. The alpaca became anorectic and lost weight during the therapy, and the day after therapy was concluded, the alpaca became moribund and died.

Necropsy

A perforating ulcer was found in C-1, with ingesta in the greater omental sac. The lungs contained multifocal firm nodules ranging in size from pinpoint to 3 to 4 mm in diameter. Similar lesions were observed in the caudal pole of a kidney.[151]

Microscopically, septate hyphae were identified in the lung and kidney lesions and in the necrotic retina, ciliary body, and posterior lens capsule. No hyphae were identified in sections taken from the margin of the gastric ulcer.

Diagnosis

In the case described, the cause of death was probably peritonitis associated with the perforated gastric ulcer. Typically, mycelial elements in tissue sections may be observed microscopically under 100×, using hematoxylin and eosin stains, but are more easily identified with Gomori's methenamine silver stain. Impression smears from suspected granulomas may be treated with KOH to clear debris from the fungal elements before staining.

Aspergillus spp. can easily be cultured on Sabouraud's dextrose agar incubated at room temperature.

Treatment and Prevention

Treatment of aspergillosis has been unsatisfactory. As a stress-related disease, it is frequently associated with prolonged steroid or antibiotic therapy or an immune-deficient patient. Predisposing conditions must be corrected before therapy can be effective. Amphotericin B is the antibiotic of choice, but it has not been used to treat aspergillosis in camelids.

Coccidioidomycosis

Coccidioidomycosis (San Joaquin Valley fever, valley fever) is a fungal infection primarily of the respiratory tract of humans and susceptible animals.[62] It may also appear in a disseminated form or as a dermatitis.

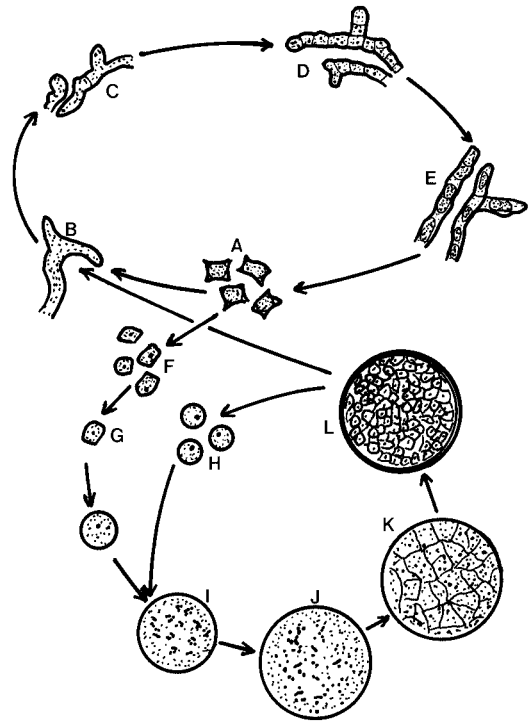

Figure 7.13. Life cycle of *Coccidioides immitis*. (A) Arthroconidia, (B) emerging fungal hypha, (C) nonseptate hypha, (D) septate hypha, (E) arthroconidia with hypha, (F and G) maturation of arthroconidia to spherule, (H) endospores, (I to K) development of spherule, and (K and L) endosporulation of spherule.

Coccidioides immitis was first described as a protozoa, similar to coccidia, but it is actually a dimorphic fungus (Figure 7.13). Its life cycle is usually completed in the absence of animals. The literature on this organism is extensive because of human infection, but this disease is not a zoonosis. There are no reports of coccidioidomycosis in OWCs.

Epidemiology

SACs appear to be highly susceptible to coccidioidomycosis, which is acquired by inhalation of arthrospores from a contaminated environment. Arthrospores are the infective stage; they convert to the tissue-invasive spherules seen in animal tissues. The thick, refractile-walled spherule sporulates and becomes filled with endospores. When the spherules rupture, endospores are released, spreading the infection.

Coccidioidomycosis is an infectious disease, but it is not transmitted by direct contact from animal to animal. Infection is generally considered to be restricted to specific geographic areas, but in camelids, with extensive movement of animals from one location

to another, the disease may be diagnosed in any area.

Jones[g] reported a case of coccidioidomycosis in a one-year-old llama in 1952. A number of cases have been presented to the veterinary hospital of the University of California. Ingram, in Arizona, has also dealt with cases.[h] Whereas most other artiodactylids seem to have a low susceptibility to clinical infection with *C. immitis*, the llama, in contrast, seems to be highly susceptible. Llamas and horses frequently develop a disseminated form.

Llamas and alpacas maintained in endemic areas are at risk. The known areas in which *C. immitis* is endemic include the southern desert areas and the San Joaquin Valley of California; the southernmost parts of Utah and Nevada; and most of southern Arizona, New Mexico, and southwestern and west-central Texas. The climatic conditions of these hot, arid areas of the lower Sonoran life zone are conducive to maintenance of the fungus.[122]

The arid regions of northern Mexico are known to harbor *C. immitis*. In South America, endemic areas are located in northwestern Venezuela and the Gran Chaco Pampa region of Argentina and Paraguay, with possible extension into southern Bolivia. Endemic areas have also been identified in Honduras, Guatemala, and Nicaragua in Central America. Infection originating in Europe and Asia has never been authentically confirmed. It is likely that such infection has been carried to the Old World by infected animals or fomites.

Disruption of the soil, such as for road building or excavation for construction, exposes the organism to winds, creating dusty aerosols suitable for inhalation of the arthrospores. In California, heavy winds have transported infective dust from southern areas of the state to northern California, causing an epidemic in humans and animals in areas not normally exposed to coccidioides.

Transplacental infection has occurred in a llama. Previously, this was only known to occur in humans.

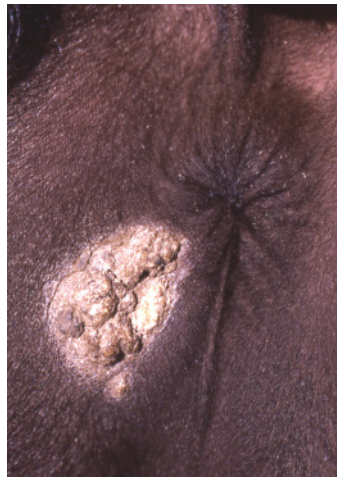

Figure 7.14A. Dermal coccidioidomycosis.

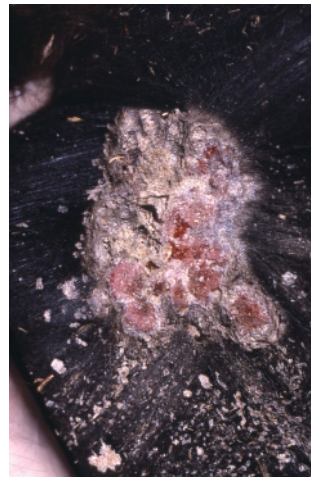

Figure 7.14B. Dermal coccidioidomycosis.

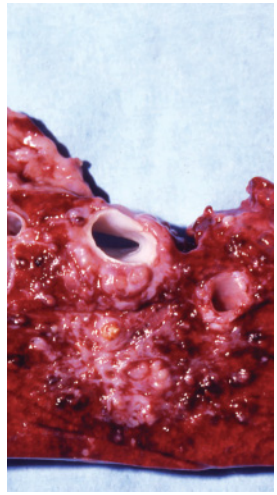

Figure 7.15A. Lung granulomas caused by *C. immitis*.

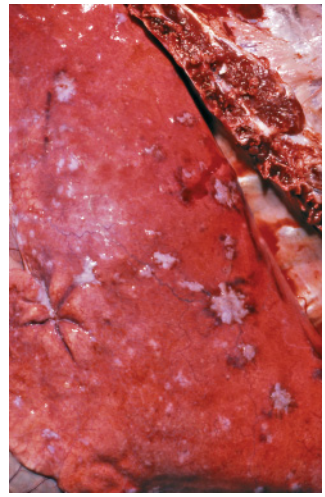

Figure 7.15B. Lung granulomas cause by *C. immitis*.

Clinical Signs

Clinical signs vary with the location of the lesion. One llama had a posterior paresis, with slightly impaired hind limb neural reflexes.[143] A radiographic change was discernable at the level of the tenth thoracic vertebra (T-10). Although disseminated lesions were seen at necropsy in the heart, lungs, liver, and all lymph nodes, primary signs were neurologic. In other cases, respiratory signs have predominated, i.e., dyspnea and coughing.

A dermal form is characterized by nodular lesions from 1 to 3 cm in diameter or extensive raised plaques (Figures 7.14A and B). Lesions have been observed over most of the body surface, e.g., on the perineum, face, and limbs. Pruritus is not evident.

Pathology

In less severe forms of coccidioidomycosis, granulomas may be restricted to the thoracic cavity and lymph nodes and within lung tissue. These lesions may be incidental findings at necropsy. Most likely, animals in enzootic areas have inhaled the organism and developed this inapparent mild infection.

In the disseminated form, every organ or tissue of the body is a potential site for granuloma formation (Figures 7.15A to 7.17). Though only a few reports have been documented in North America, granulomas affecting the pericardium, myocardium, endocardium,

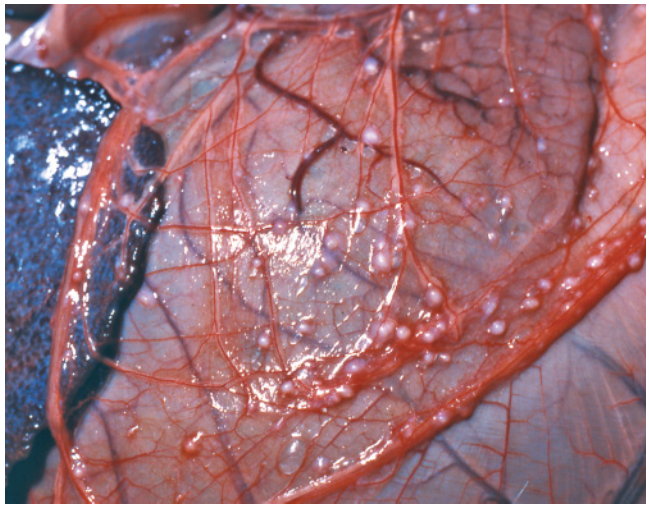

Figure 7.16. Lesions in the peritoneal cavity caused by *C. immitis*.

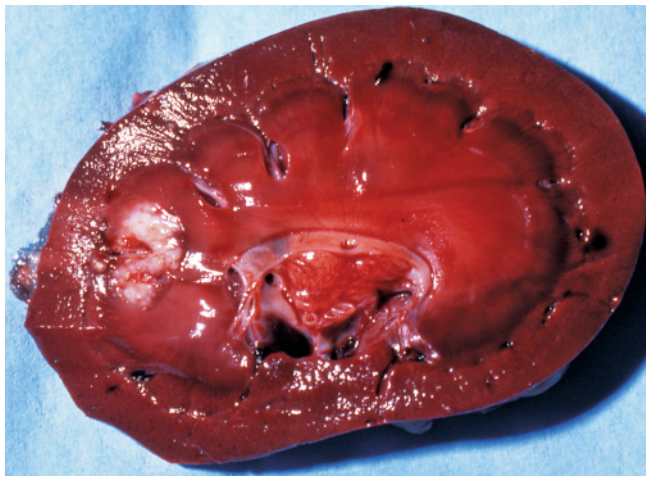

Figure 7.17. Renal granuloma caused by *C. immitis*.

spleen, liver, kidney, thyroid, lungs, bone, central nervous system, visceral and peripheral lymph nodes, skin, and buccal mucosa have been seen. Lesions may appear as discrete granulomas ranging from 1 to 5 cm in diameter or coalesce in large, irregularly shaped masses. The nodule is usually gray and firm. The gross lesion is nondifferentiable from those of mycobacteriosis. The microscopic lesion is characterized by granulomas or pyogranulomas, with numerous spherules present.

Diagnosis

A presumptive clinical diagnosis is difficult because of the numerous organ systems involved. Diagnosis is facilitated if a lesion is accessible because the organism can be readily identified by direct microscopic observation and identification of fungal spherules (10 to 80 m in diameter) from respiratory secretions, pleural and peritoneal fluids, needle aspirates from abscesses and skin nodules, or biopsy samples. In the llama with posterior paresis, the cerebrospinal fluid sample collected from the lumbosacral space was normal. The granuloma at T-10 had been encapsulated, preventing development of an inflammatory response that could be detected in the cerebrospinal fluid.

The interpretation of serologic data from llamas is difficult because, as yet, insufficient numbers of individuals and herds have been tested to establish sound normal data as a basis for comparison.

The organism may be cultured on selective media such as cycloheximide-chloramphenicol agar incubated at room temperature. On artificial media, hyphae will be seen sprouting from the spherule. The hyphae are septate. Growth is rapid (three to five days). Arthrospores develop within hyphal segments. An investigator should refer to standard texts for details of safety precautions before attempting to culture *C. immitis* because of the risk of infection from the arthrospores produced in culture.

Serologic and immunologic tests used in the study of coccidioidomycosis include precipitin, complement fixation, fluorescent antibody, latex particle agglutination, and agar gel diffusion precipitin tests. Some of these tests are presently being used to evaluate the status of infection in herds in endemic areas of Arizona.[i] Intradermal tests, at dilutions of 1:5, 1:10, and 1:100, have been negative in known infected animals.

Lesions within the thoracic cavity are detectable by radiography but cannot be differentiated from abscesses, tumors, or other granulomatous lesions.

Treatment with Amphotericin B

On October 30, 1985, a female llama given a prepurchase physical examination was pronounced pregnant. She was losing weight according to the owner, but no other clinical signs were evident. On January 30, 1986, a biopsy obtained from a dermal lesion proved to be coccidioidomycosis. Thoracic radiographs indicated extensive granulomatous lesions in the lungs. An immunodiffusion test was positive for complement-fixing-type antibodies, and the complement fixation quantitative test was 4+ at 1:16 and 2+ at 1:32. Pregnancy was confirmed with the fetus estimated to be four months of age.

In view of the value of the fetus, a decision was made to attempt treatment to carry the mother through pregnancy. The llama weighed 115 kg. The jugular vein was catheterized and the catheter remained in position for six weeks. A test dose of amphotericin B (1 mg) was given intravenously to check for adverse reactions.

The therapeutic regimen was to be 1 mg/kg amphotericin B, administered intravenously every forty-eight hours for six weeks. The regimen was begun with 34.8 mg amphotericin B mixed in 500 ml of 5% dextrose in water solution. This was administered over a four-hour period, followed by 3 L lactated Ringer's solution containing 1.5 ml vitamin B complex solution (Bsol) and 20 mEq potassium chloride (KCl). The supplemental lactated Ringer's solution, B complex, and KCl were given to stimulate diuresis and prevent hypokalemia, a side effect of amphotericin therapy.

The dosage of amphotericin was increased by 10 mg per treatment until the maximum dose of 115 mg or 1 mg/kg was reached. The llama was monitored carefully and laboratory tests (urinalysis, hemogram, and serum chemistry) performed every three to five days. There was no evidence of nephrotoxicity. The llama tolerated the therapy well, with anorexia appearing only during the last week of therapy.

This therapy regimen was expensive (approximately \$33/dose × 21 = \$693). After completing therapy, the female stabilized, was discharged, and ultimately delivered a male cria on July 9, 1986. The neonate was examined carefully for evidence of coccidioidomycosis, including thoracic radiographs. On initial examinations, parameters were evaluated as normal. Approximately a month later the cria had become dyspneic. A thoracic radiograph indicated a cystic lesion in the lung; serologic tests were positive for coccidioidomycosis. The female's complement fixation titer was positive at 1:256. Euthanasia was elected, and both the neonate and the female were euthanized on September 19, 1986. Extensive diffuse lesions were found throughout the thorax and abdomen of the female. Lesions were also found in the lungs of the cria. It was concluded that therapy was not successful in eliminating the infection from the female nor in preventing transplacental passage of the organism to the fetus.

Several other antifungal imidazole drugs or combinations of drugs are used to treat coccidioidomycosis in humans and other animals. These include fluconazole, ketoconazole, miconazole, and itraconazole. The author could not find references to the use of these drugs in camelids, nor dosages for large animals. The dose for humans with disseminated valley fever is 400 mg/kg once daily orally for ten days.

Prevention

Vaccines are being developed to protect humans who must work in high-risk areas. Similar vaccines have also been used in nonhuman primates in highly endemic areas with apparent success. Vaccines for camelids have not been evaluated but may become important if camelids are to be kept in endemic areas.

Cryptococcosis

Etiology

Cryptococcus neoformans is a yeast-like fungus. It does not form hyphae. The organism is not contagious but is infectious.

Epidemiology

C. neoformans is distributed worldwide and is capable of infecting any mammal. The organism is found in surface dust and dirt in contaminated areas, particularly where pigeons (*Columbia livia*) congregate, and pigeons are a primary reservoir. Transmission to mammalian hosts is usually by inhalation. Predisposing factors include immunosuppression such as stress and preexisting immunodeficiency or intensive exposure.

Clinical Signs

Infection often localizes in the central nervous system and signs vary with the location of the infection.[22] Blindness and chorioretinitis may result from eye infection. Mastitis may be seen in cattle.

Diagnosis

Yeasts may be visualized on direct examination after staining with India ink. The organisms may be cultured on blood agar or Sabouraud's agar.

Management

The source of the infection should be ascertained and steps taken to eliminate the hazard. Masks should be used by personnel involved in cleaning up pigeon debris. Fluconazole is the treatment of choice.

Mucormycosis

Etiology

There are numerous genera within the order Mucorales. The disease produced by any of these is called "mucormycosis." Only one genus, *Rhizopus* spp., has been isolated, from a disseminated, multisystemic infection in a llama. Rhizopus is a complex genus. The isolate from the llama was not speciated.

Clinical Signs

The llama was maintained in a herd used for teaching and research. The first sign of disease was bilateral facial paralysis (cranial nerve VII) that developed following an episode of struggling in a restraint chute. It was believed that the halter straps had traumatized the facial nerves. The eyelids were paralyzed at first, and in a few days the nictitating membrane failed to be pushed over the cornea, indicating an inability for the bulb to be retracted deep into the orbital socket by

paralysis of the abducent (cranial nerve VI) nerve, which supplies the retractor bulbi muscle.

Ultimately, prehension and swallowing became impossible (cranial nerve IX, glossopharyngeal). The llama began to lose weight. It became apparent that multiple cranial nerves were involved, which precluded the diagnosis of trauma to superficial nerves.

The llama was anesthetized for more detailed diagnostic procedures. An endoscopic examination of the nasal cavity revealed a black membrane flecked with irregular white patches on the surface of the turbinates and the nasal mucous membrane. There was no prior nasal exudate.

Diagnosis

The llama died and was necropsied. *Rhizopus* spp. were isolated from nodules (0.5 to 2 cm) in the lung parenchyma. The nodules were firm and black on the cut surface. Filamentous growth was present on the surface of a necrotic rhinitis, including the turbinates and cribriform plate.

The meninges on the ventral aspect of the brain were inflamed, and granulomas were present in the region of the cranial nerves, indicated by the clinical signs. Significant lesions caused by *Rhizopus* included multifocal bronchopneumonia, necrotizing rhinitis, and severe, multifocal meningoencephalitis.

Miscellaneous Fungal Infections[107,124,213]

Infection caused by *Entomophthoramycosis conidiobolae* has been reported.[132] Histoplasmosis has been reported in llamas.[209]

The normally saprophytic fungus *Conidiobolus coronatus* has produced localized infection in humans, horses, and one llama. The lesion consisted of a nodular dermatosis involving the external nares, which had been present for three years. In addition to the nares, the face and muzzle were involved.[65] Encroachment of the nares had caused dyspnea to develop. The mass obstructing the nostril was excised, but total extirpation was not possible. The llama lived another three years following the surgery.

BACTERIAL DISEASES[68]

Clostridial Diseases

The major clostridial diseases of domestic animals are listed in Table 7.6. Many of these are ubiquitous and would be serious threats to livestock production if bacterin/toxoids were not available. Camelids may acquire some of these diseases.

Clostridia are Gram-positive, rod-shaped, anaerobic bacilli. All form spores that may persist in the soil for months or years. Some of these organisms may be found in the normal flora of the digestive tract and become pathogenic only if accessible tissue is damaged as a result of deep penetrating trauma to the muscle bundles or a compromised gastrointestinal mucosa.

Clostridial organisms produce potent exotoxins that are primarily responsible for the disease these agents cause. The toxins are metabolites that are produced as the organism grows in the host tissue, except for botulinum toxin, which may be ingested preformed. A given organism may produce single or multiple toxins, each with a different effect on the host. Botulinum and tetanus toxins are neurotoxic only. Most other clostridial organisms produce toxins with both local and systemic effects, including hemolysis and local tissue necrosis.

Botulism

C. botulinum is distributed worldwide. One or more of the types (A to G) is probably toxic to all vertebrates. No cases have been reported in NWCs but has been reported in a group of dromedaries in Chad.[203] The clinical diagnosis is difficult and may have been simply overlooked. There is good reason to believe that all camelids may be susceptible.

The syndrome is believed to be similar to that seen in most other mammals, consisting of a progressive paralysis of all skeletal muscles. Initially, incoordination, muscle weakness, and recumbency are seen, leading finally to flaccid paralysis of all muscles, including respiratory muscles. Body temperature is not elevated. The pupils of the eyes become dilated. Salivation is decreased, and mucous membranes become cyanotic.

Diagnosis. Because *C. botulinum* may be cultured from a normal digestive tract, isolation of the organism is not diagnostic. A definitive diagnosis can be made only by the injection of filtrates of suspected feed materials or gut contents into mice or guinea pigs. Control animals are given simultaneous injections of protective doses of specific antitoxin.[24]

Treatment. Once signs have developed, little can be done other than to support respiration. Antitoxins (toxin-type specific) used to treat human cases are not available for animals.

Prevention. No toxoid bacterins against botulism are available for protection of animals or humans.

Tetanus

C. tetani occurs worldwide as a soil saprophyte, but it can also be found in the feces of horses, humans, and cattle. Tetanus is more common in tropical regions than in cold climates. There is wide variation in the susceptibility of animal species to tetanus toxin. Horses, nonhuman primates, and swine are highly susceptible, with cattle, sheep, goats, and humans less so and dogs and cats quite resistant. The degree of susceptibility of camelids is not known. In camels, tetanus is considered to be insignificant. Two cases of

Table 7.6. Differential diagnosis of clostridial diseases in camelids.

Disease	*Species*	*Unique clinical signs*	*Differential pathology*	*Epidemiology*	*Occurrence in SACs*	*Occurrence in camels*
Botulism	*C. botulinum*	Flaccid muscle paralysis	None	Ingestion of toxin	NR	R
Tetanus	*C. tetani*	Tetanic muscle spasms	None	Anaerobic wound contamination	N, Mi	N, Mi
Blackleg	*C. chauvei*	Fever, hemorrhagic swelling, gas formation in muscles	Serosanguinous myositis with gas	Unknown in SACs	E, N	N, Mi
Malignant edema	*C. septicum*	Edematous swelling around wound, no gas, sudden death	Edematous subcutaneous cellulitis	Wound infection	N, Mi	N, Mi
Black disease	*C. novyi* Type B	Sudden death, dyspnea, ataxia	Hepatic necrosis	Spores and vegetative forms in liver of healthy animals, liver damage allows initiation	NR	NR
Bacillary hemoglobinuria	*C. haemolyticum*	Sudden death, hemoglobinuria, anemia	Hepatic necrosis, intravascular hemolysis	Same as above	NR	NR
Type A enterotoxemia	*C. perfringens* Type A	Sudden death, depression, colic, convulsions, no diarrhea	Well muscled, hemorrhages on serosae, intestine distended with fluid and gas	In best conditioned neonates, 8–35 days	N, Mi	N, Mi
Type c enterotoxemia	*C. perfringens* Type C	Sudden death, severe diarrhea, distended abdomen, gas, colic prostration, paddling	Similar to Type A, with more gastrointestinal hemorrhage and enteritis, cerebral edema	In U.S., otherwise similar to type A, occurs in years with abundant grass	N, Mi	N, Mi
Type D enterotoxemia	*C. perfringens* Type D	Sudden death, convulsions, circling, prostration, posterior paralysis, some diarrhea	Hemorrhages extensive on serosae, epicardium, and endocardium, no gas formation	Not common in neonate, usually in animals on heavy feeding schedules	N, Mi	N, Mi

Note: N = natural infection, NR = not reported, Mi = minor disease, R = rare.

tetanus in alpacas in Peru have been reported,[137] one case of tetanus in a llama in Argentina,[194] and several cases in llamas, one being a neonate, in the United States.[103,115]

Tetanus develops when wounds have been contaminated with soil or feces containing *C. tetani* spores. Contaminated deep wounds with devitalized tissue are most at risk. Such wounds are poorly aerated, providing optimum conditions for growth of the anaerobic organism. It was suspected that the llama cria acquired tetanus via a navel infection.

Clinical Signs.[191,192,203,204] Both of the affected alpacas in Peru had purulent rear limb wounds. Signs included prostration, muscle rigidity, dyspnea, tonic muscular contractions, joint stiffness, a fixed stare, erect ears, a locked jaw, and a fever of 41.5°C (107°F).[102,137] In Argentina, a llama with infected wounds on the feet was diagnosed as having tetanus. The llama remained standing in a "sawhorse" stance, with the limbs base-wide. The jaw was closed and rigid, with drooling saliva, and the facial expression was wooden (Figure 7.18). There was dyspnea, erect ears, an elevated and rigid tail, and a protruding nictitating membrane.[194]

The author has treated two tetanus cases in llamas and followed the course of another case managed by a nearby colleague. The signs noted in the Argentine llama were present in these llamas. An additional sign was that the animals were unable to kush. If they did finally fall down, they could not remain sternal and were unable to right themselves.

Treatment. One llama was given the treatment recommended for tetanus in cattle: antibiotics were administered, and the wounds were debrided and cleansed. Tetanus antitoxin was administered at a dose of 225 units/kg body weight; half was given in an IV dose and half in an IM dose. Anaphylactic shock is a hazard of this therapy because tetanus antitoxin is a horse serum product. The llama was placed in a nonstimulating environment and was tranquilized with chlorpromazine (2.2 mg/kg/6 hours).

Figure 7.18. Tetanus in a llama.

Within a few hours, the llama began to eat, drink, and move about the stall. Medication was discontinued in thirteen days, with the llama recovering fully.[194]

One llama was stiff and unable to eat or drink for two weeks. In such a case supportive care must include oral or rectal fluids and nourishment. The author has used a slurry of quick-cooking rolled oats. The quantity of the breakfast cereal selected is put into boiling hot water, allowed to cool, and then diluted to a consistency that may be pumped through a stomach pump. Total parenteral nutrition could also be used.

Prevention. Tetanus toxoid vaccines are readily available. Studies have been conducted that demonstrate that llamas respond to toxoid vaccination with a rise in titer, but challenge studies have not been conducted.[j,150]

Blackleg

Blackleg (quarter evil, quarter ill, black quarter, symptomatic anthrax) is a disease of ruminants characterized by high fever, serohemorrhagic swellings, and gas formation in the heavy muscles of the body and limbs.[107] The toxin is produced by *C. chauvei*. There are no reports of natural infections in SACs, but blackleg has been produced experimentally in alpacas. The alpaca seems to be more resistant than the bovine to infection.[133] The disease has been reported to occur in the camel.

Blackleg is mentioned here because the disease must be differentiated from anthrax. Diagnosis is based on a positive fluorescent antibody test or isolation and identification of the organism from infected tissue.

Treatment. A recommended treatment regimen consists of antibiotic administration (penicillin, tetracycline) for five to eight days. Clostridial toxoid vaccines contain all multiple species, including *C. chauvei*, and induce effective protection in cattle. However, the low to nil prevalence of this disease in camelids does not warrant vaccination.

Malignant Edema[193]

Malignant edema (gas phlegmon, gas edema, bradsot, braxy) occurs worldwide in a broad host range, including domestic livestock, horses, humans, dogs, and cats.[134] Wild herbivores are considered equally susceptible. Malignant edema is an economically important disease in alpacas in Peru. Cases of

malignant edema have been associated with rattlesnake bite in llamas in Colorado.

C. septicum produces a toxin that causes severe edema. It is basically a soil organism but has been found in both spore and vegetative forms in the intestines of healthy animals.

The organism invades tissue through a necrotic, deep wound that provides anaerobic conditions. Oral wounds and bruises are common entrance sites in alpacas. The organism may also gain access to body tissue from disruption of the stomach epithelium. As the organism grows, toxins are produced, and clinical signs appear within one to three days. Two types of syndromes develop in SACs. One is the typical wound infection and edema. The other is an acute systemic disease similar to braxy of sheep. The Spanish name for this disease is "muerte súbita" (sudden death), which characterizes the primary finding. With wound infections, clinical signs include a rapidly spreading, edematous swelling in the subcutaneous tissue surrounding the wound. Little, if any, gas forms in this disease, in contrast with blackleg. Other signs include fever, rapid pulse, anorexia, depression, and weakness. *C. septicum* affects animals of all ages. Death may occur twelve days after signs develop.

Diagnosis, treatment, and prevention are the same as for blackleg.

Clostridial Hepatitis[49]

Two different diseases are caused by two different strains of *C. novyi*. Black disease is produced by *C. novyi* type B (in the United States this strain is designated *C. hemolyticum*), and bacillary hemoglobinuria (red water, icterohemoglobinuria) is produced by type D. Neither of these diseases is known to occur in camelids, but some aspects of the pathogenesis of the diseases warrant consideration.

The type B organism is a common contaminant of soils in the western hemisphere and may also be a normal constituent of the microflora of the digestive system of domestic livestock. Spores may be transported to the liver, where they lie dormant until conditions are suitable for vegetative growth and invasion of the hepatic parenchyma. Favorable conditions include any necrotizing or tissue-damaging insult to the liver, such as that caused by migration of immature liver flukes (*Fasciola hepatica*), chemical toxins, plant toxins, liver abscesses, or trauma (liver biopsy).

The germinating spores produce the highly necrotizing toxin, which compromises the liver further. Recognizing the likelihood that spores of *C. novyi* are present in the livers of camelids kept with cattle and sheep, and that liver biopsy is a routine diagnostic procedure, it may be wise either to immunize SACs against *C. novyi* or administer long-acting benzathine penicillin at the time a liver biopsy is performed.

Clinical signs of black disease in cattle are those of a peracute to acute toxemia, with death occurring in many cases before signs are noticed. In subacute cases, signs include dark discoloration of the skin as a result of severe venous congestion (hence the name), severe depression, anorexia, dyspnea, ataxia, and recumbency, with death occurring within a few hours.

Bacillary hemoglobinuria is also an illness of short duration (eighteen to thirty-six hours). Initial signs may be similar to those of black disease, but if the animal survives for twenty-four hours, severe intravascular hemolysis with anemia and hemoglobinuria will develop. Death usually results from hypoxemia and respiratory depression.[49]

The mortality rate for both diseases is more than 90%, and treatment is of little avail because of the severe hepatic necrosis. Definitive diagnosis is based on isolation and identification of the organism in the laboratory using immunofluorescent antibody tests.

Enterotoxemia[179]

C. perfringens is an anaerobic, spore-forming bacterium. Small numbers of the organism may be found in the gastrointestinal tract of healthy individuals. The organism proliferates only if the environment in the intestine deteriorates.[169] The spores of *C. perfringens* are common soil inhabitants, and infections tend to recur year after year once an area is seeded with the organism.

Five types of *C. perfringens* are known to affect animals. Collectively, the types are found worldwide, but individual types have a specific geographic distribution. There is marked variation in species susceptibility to each type. The types are designated as A, B, C, D, or E, according to the toxin produced. Camelids are known to be susceptible to types A and C. Type D is also highly suspect. Each of the three types will be discussed separately.

Type A Enterotoxemia.[47,156] **Epidemiology**: *C. perfringens* organisms (both spores and vegetative forms) are ingested in feed or water contaminated with infected soil or the feces of carrier animals.

The predisposing factors for type A enterotoxemia and the clinical syndrome have occurred in North America, but the precise etiologic diagnosis has eluded diagnosticians. Type A enterotoxemia is the most serious disease of neonate alpacas in Peru.[89,139,] Epizootics may occur during the birthing period or prolonged rainy periods or as a result of poor sanitary conditions. In different districts of Peru, annual mortality rates from this disease vary from 10% to 70% of alpaca crias. Even in carefully controlled conditions, such as those that prevail at La Raya Research Station, mortality from enterotoxemia varied from 1% to 56% between 1973 and 1979.

Death losses from type A enterotoxemia occurred in crias from three to eighty days of age, with more than 85% of the losses occurring between eight and thirty-five days. Paradoxically, crias in the best condition were most likely to be affected.

Type A toxins are poor immune responders, and currently there is no vaccine available to protect against type A enterotoxemia.

Another important factor in the development of enterotoxemia is the serum protein level in the cria, especially of the globulin fraction. It is naturally low at birth (<5.2 mg/kg). With ingestion of colostrum, the protein level elevates to 5.5 to 6.2 mg/kg by four or five days. The globulin fraction begins to decrease, because the neonate is not yet immunologically competent and is not producing sufficient immunoglobulin G (IgG) to compensate for the diminishing of colostral IgG. The lowest level of globulin occurs between two and three weeks of age, which also corresponds to the age of highest death losses from type A enterotoxemia.

Clinical Signs: Sudden death may be the only overt manifestation. Signs, and their intensity, depend upon the quantity of toxin produced. Rectal temperatures may be subnormal, normal, or slightly elevated. The cria soon becomes recumbent, with the head stretched forward, eyes closed, ears directed backward, and legs stretched. Movement and vocalizations are indicative of colic. The abdomen is frequently distended, with gas tympany in the intestinal tract. The cria is anorectic and dyspneic. Diarrhea is not a sign of type A enterotoxemia but may be seen in mixed infections with *Escherichia coli* or other microorganisms. Constipation is more likely to occur in pure type A enterotoxemia.

As the disease progresses, central nervous system (CNS) disorders become apparent, indicated by convulsions and opisthotonos. Finally, the animal may become comatose and die. Death occurs too rapidly for the development of hematologic alterations.

Pathology: The carcass is usually in good muscular condition. Hyperemia and petechia of the subcutaneous tissue may be seen. Lungs are congested, and petechia of the pleural surface may occur. The bronchi contain fluid and foam, indications of pulmonary edema, and ingesta, aspirated terminally. Thoracic lymph nodes may be edematous and hemorrhagic. The thymus may be congested, with surface petechiae. Variable amounts of excess serosanguineous pericardial fluid have been found. The coronary arteries are dilated, and petechiae are present on the epicardium of the auricles. Thrombocytopenia prevents blood coagulation that may be observed when the ventricles are opened.

The glandular saccule areas of the stomach are markedly congested. The intestines are typically distended with watery fluid and gas, which have a disagreeable odor. The small intestine, particularly the jejunum and ileum, is congested. The large intestine may also be congested, and Peyer's patches are prominent.

There is congestion of the renal cortices, and the capsule of the kidney peels away with difficulty. The urinary bladder is usually distended as a result of paralysis from the effects of the toxin. The brain is congested, and there is an excessive amount of cerebrospinal fluid.

Prevention: Climatic conditions may make it difficult to avoid cool, wet weather during the birthing season, but special precautions should be taken to keep neonates uncrowded and as dry as possible. During an outbreak at a ranch in California, metronidazole (Flagyl) was administered orally from day 2 postdelivery to one month of age. Metronidazole is effective against anaerobic bacteria. Prior to administration of metronidazole, 100% of the crias died. Crias born during inclement weather at that ranch are automatically treated with metronidazole.

Type C Enterotoxemia.[48,138,203] **Epidemiology:** Type C enterotoxemia resembles type A and is included with type A as an economically important disease in Peru,[122] but recently researchers have concluded that of the two, type A is much more important. Cases of type C enterotoxemia have been reported in North American camelids, but confirmation to toxin is rarely carried out. Clinicians and pathologists base diagnoses on experience with type C enterotoxemia in lambs and calves. Not all strains of type C are infective for camelids.

In Peru, enterotoxemia outbreaks occur in years of heavy rainfall, when grass is lush and abundant and milk production by the females is high. Types A and C both affect alpacas, llamas, and vicuñas, but alpacas ten to forty days of age are more often affected. Some researchers feel that alpacas are much more susceptible to enterotoxemia than llamas.[k] Unsanitary conditions enhance the possibility of infection.

Clinical Signs: Some animals die with no clinical illness having been observed. Animals seen to be perfectly healthy may be moribund four hours later, with subnormal temperatures, 35°C to 36°C (95°F to 97°F), and subsequent death shortly thereafter. Disinterest in nursing and depression are seen early in both acute and chronic cases. In chronic cases, the temperature may be elevated, up to 40°C (104°F), and a watery diarrhea develops.[118] The abdomen becomes distended, and vocalizations indicate colic. Some animals ingest great quantities of water or develop pica.

South American researchers indicate that some animals with severe diarrhea seem to recover better than those with minimal diarrhea, presumably because the toxin is flushed from the intestine by the rapid fluid expulsion.[109] The morbidity rate in some herds

may reach 100%, and the mortality rate may be as high as 60%.

Experience with type C enterotoxemia in North American camelids is of a different nature. No epizootics have been reported. Incidence of the disease has been sporadic. Infection has been most frequently observed in the cria less than two weeks of age that is nursing a dam with high milk production. Clinical signs are prostration, paddling (CNS involvement), and watery diarrhea. Some animals have been found dead without illness having been previously observed.

Pathology: In Peru, the lesions are essentially the same as for type A enterotoxemia.[118] In North America, the usual necropsy findings are of a hemorrhagic enteritis, with blood-stained intestinal contents. The intestines are distended with gas and are intensely congested. Pulmonary interstitial edema and hydropericardium are often seen. There may be cerebral edema and neuronal degeneration in the brain.[169]

Type D Enterotoxemia. **Epidemiology**: Type D enterotoxemia (overeating disease) has not been diagnosed in SACs in Peru. Sporadic cases have been reported in North America. Type D is a serious disease of feedlot cattle and sheep, animals on lush pastures, or those being overfed with grains. *C. perfringens* type D produces two major toxins: an α hemolytic toxin, which, in turn, aids the more damaging epsilon toxin to cause necrosis of the intestinal wall; the epsilon toxin is then absorbed and produces similar necrotic lesions in the brain.

Clinical Signs: Type D enterotoxemia is characterized by CNS signs such as convulsions, circling, prostration with opisthotonos and paddling, posterior paralysis, and coma.[20] Sudden stimuli may initiate a convulsive seizure. A slight fever, drooling, and diarrhea are other signs.[183] As with most of the enterotoxemias, sudden death is common.

Pathology: Necropsy findings listed here are those seen in cattle and sheep, since the lesions in camelids have not yet been adequately described. Petechial and ecchymotic hemorrhages may be found on any serosal surface, and both epicardial and endocardial hemorrhages are common. Excessive amounts of pericardial fluid may contain fibrin clots. Gastroenteritis is present, but there is no significant gas formation. Additional findings in animals that survive the peracute stage include intramuscular hemorrhage, pulmonary congestion and edema, edematous mesenteric lymph nodes, and focal necrosis and edema of the brain.[183]

Diagnosis of Enterotoxemia. There is sufficient overlap of epidemiology, clinical signs, and pathology of the various enterotoxemias that laboratory assistance is necessary to make a definitive diagnosis. *C. perfringens* can be isolated from intestinal contents of the ileum in 100% of the affected camelids.[127] The organism may also be isolated from the blood, lungs, liver, spleen, and mesenteric lymph nodes in septicemic cases.

The isolation and identification of the organism, including the type, is carried out by standard microbiologic procedures. Ileal contents are filtered through a millipore filter. Either in vitro or in vivo techniques may be used to identify the strain. A diagnosis may also be made by identifying the toxins using immunologic procedures.

A differential diagnosis must include other diseases that cause peracute signs, severe diarrhea, and death. Failure of passive transfer of immunoglobulins to the neonate may predispose the cria not only to enterotoxemias but to other opportunistic bacterial and viral infections as well. Nutritional diarrheas are common in neonates, especially if they have been orphaned, and *E. coli* septicemias are not rare and may be acquired in utero. Coccidiosis is a parasitic disease that should be considered in a differential diagnosis.

Treatment. Treatment has been unrewarding in clinical cases. Administration of broad-spectrum antibiotics may arrest the growth of the organism and production of toxins, but usually severe organ damage has already occurred. Supportive treatment with fluids is indicated, but the prognosis is grave. In herd outbreaks in Peru, workers have found that chloramphenicol is most effective against type A enterotoxemia. In North America, herd enterotoxemias have been managed by adding chlortetracycline to the feed at a rate of 22 mg/kg feed.[183]

Prevention. Feeding, sanitation, and general husbandry practices must be evaluated periodically and changed, if necessary. Toxoid administration is commonly practiced in cattle, sheep, and llamas in North America. The risk for development of enterotoxemia in camelids is greatest during the first few weeks of life. Neonates have immune systems that are able to mount an adequate response to *Clostridium* spp. or other pathogens, but sufficient time is required (two weeks plus). The preferred approach is to administer toxoids to the dam approximately two months before parturition, with a booster at one month prior to parturition.[37]

Type A toxin produced by *C. perfringens* is a poor antigen[169] and is not included in any of the multispecies clostridial preparations. Toxoids produced from type A toxins were not effective in controlling enterotoxemia in Peruvian alpacas.[145,146] Types A, B, C, D, and E toxins are used in typing the strain but may not be the major disease producers. Researchers are presently attempting to enlarge the toxin base for subsequent protection trials.

Tuberculosis[81]

Tuberculosis is a chronic, granulomatous, bacterial, infectious disease characterized by the development of

tubercles (small avascular nodules containing giant cells) in various organs of the body. Camelids are not highly susceptible to the infection, but both natural and experimental infections have been reported.[33,37,] Currently tuberculosis in camelids is of concern to regulatory officials.

Four major species of acid-fast staining mycobacteria (*Mycobacterium bovis, M. tuberculosis, M. avium, M. paratuberculosis*) affect livestock, and all four have been reported from camelids, either as an experimental or a natural infection. *M. microti* has been reported in a zoo vicuña,[149] and a single case of *M. kansasii* has been reported.[96] M. *tuberculosis*, *M. bovis*, and *M. avium* also infect humans. Additionally there are numerous atypical mycobacteria or soil-saprophytic mycobacteria that may share sufficient antigens to complicate a testing program.

Epidemiology[39,141,147,195,196]

Tuberculosis is a worldwide disease that usually develops slowly, providing ample opportunity for dissemination of the organisms. The disease was thought to be under control in the human population in the United States, but recently there has been an upsurge in cases, particularly serious because many strains have become resistant to medication. It is a serious problem in immunocompromised individuals. Lesions are commonly found in the respiratory and digestive systems, and excretions and secretions may be contaminated with the organism. Infection may be acquired by inhalation or ingestion.

Llamas are not particularly susceptible to tuberculosis. On an Iowa game farm, a llama died and diffuse granulomas were found at necropsy. Tuberculosis was diagnosed in cervids at this same farm. Ultimately, the herd of ten or eleven llamas were purchased and sent to Ames, Iowa, for detailed necropsy. No lesions were found in any of those animals even though they had been in direct contact with the tubercular llama that died. Similar findings were noted on a game farm in Canada that kept cervids and llamas running together. Many of the cervids and one of the llamas had granulomas, but the rest of the llamas were clean.

There are only rare reports of confirmed natural cases of tuberculosis in camelids in South America, even though tuberculosis is common in cattle, sheep, and humans in the areas shared with SACs.[115] In North America, *M. bovis* was isolated from eight llamas during a five-year period at the Veterinary Services Laboratory of the USDA.[190]

In 1957, 390 alpacas in Peru were tested with intradermal injections of mammalian tuberculin. Another sixty were injected subcutaneously to ascertain body temperature changes. None of the 450 alpacas developed positive reactions to the tuberculin. Unfortunately, the degree of exposure to tuberculosis was not mentioned in the report. However, this report is of interest because others have reported that camelids are prone to exhibiting false positive and negative reactions to tuberculin, and these alpacas showed none.

Clinical Signs

The signs of tuberculosis vary widely, depending on the organ system involved. Tuberculosis is a debilitating disease, so weight loss and emaciation are typical. Diarrhea and dyspnea may accompany lesions in respective organ systems,[118] although dyspnea may not be exhibited unless forced exercise gives evidence of exercise intolerance. Tuberculosis is a highly chronic disease that may allow an animal to live for years before succumbing to the disease.

Healthy camelids may withstand exposure to a few mycobacteria. Others may acquire a minimal infection, with the lesion healing without progressing to overt disease. Tuberculosis should be considered in the differential diagnosis of illnesses involving chronic weight loss or emaciation in camelids.

Diagnosis[62,81,178,182,186,187,189,198,199]

The antemortem diagnosis of tuberculosis presents a challenge. Intradermal tuberculin testing with Koch's old tuberculin (OT) is the classic diagnostic test, but modern diagnostic methods include lymphocyte stimulation tests, competitive ELISA serologic testing, DNA probes, culturing the organisms, and intradermal testing with balanced, purified protein derivatives (PPD) of *M. avium* and *M. bovis*. None of these tests are infallible, but all may aid in diagnosis of tuberculosis in camelids.[43]

Tuberculin testing in Bactrian camels resulted in a number of false positive reactions, both with Koch's OT and balanced bovine and avian PPD tuberculins. Lymphocyte stimulation tests in this herd were also positive, but no tubercles were observed at necropsy, and no mycobacteria were isolated.[104]

The author tuberculin tested llamas during an outbreak of tuberculosis at a zoo. A significant reaction to avian PPD was observed in one llama, but a retest one month later was negative.

In a Peruvian experiment, alpacas were exposed to virulent organisms of *M. tuberculosis, M. bovis, and M. avium* by both oral and subcutaneous routes.[36] Subsequently, the animals were tuberculin tested using mammalian OT intradermally. This test resulted in a mixed response, with negative reactors, suspects, and positive reactors, even to *M. avium*, inoculated subcutaneously. At necropsy, localized lesions were found at the inoculation sites except for one animal given a subcutaneous inoculation of *M. tuberculosis*, in which case generalized tuberculosis developed that caused death.

A definitive diagnosis requires the culturing and speciation of the organism. It is evident that camelids are susceptible to mycobacterial infections and that they develop hypersensitivity responses. However, there may be a problem with both false positive and negative tuberculin reactions if sites other than the axillary space or lateral cervical region are used. Animals with disseminated tuberculosis may not respond to the small amount of antigen injected for a tuberculin test.

Tuberculosis is a serious challenge to deer farming in New Zealand. Microbiologist researchers there developed what is called "the blood test for tuberculosis" (BTB), combining a lymphocyte stimulation test, an ELISA test, and an inflammatory test (haptoglobin). In deer, this combination of tests has a sensitivity of more than 95% and a specificity of 98% for the diagnosis of *M. bovis* infection. The blood test has been applied to alpacas in New Zealand and compared with standard intradermal tuberculin tests with bovine and avian PPD.[62] Preliminary findings indicate that the BTB has a higher sensitivity and specificity than the skin test and can be used to differentiate between avian and bovine infections.

Differential diagnosis should include coccidioidomycosis, pulmonary abscesses, aspergillosis, Johne's disease, neoplasia, and any chronic wasting disease.

Gross lesions are suggestive, but other granulomatous diseases may be confused with tuberculosis. Acid-fast staining of tissue sections is conclusive for the presence of mycobacteria but does not identify species. Tuberculosis lesions may have only a few acid-fast organisms, while in paratuberculosis lesions, numerous organisms are present. Granulomas are usually present somewhere in the body of infected animals, but atypical infections may be lesion free. In some cases, specific lymph nodes are harvested, homogenized, and cultured.

Tuberculosis is a reportable disease, and results of tuberculin testing must be reported to appropriate state and federal agencies in the United States. The management of a tuberculin-positive individual and its herd falls under control of these governmental agencies.

Radiographs of the chest may indicate increased density lesions, but these cannot be differentiated from coccidioidomycosis or other abscesses of the lungs. A positive culture from a tracheal wash or fecal sample would be definitive, but culturing may take weeks or months. *M. paratuberculosis* generally grows much more slowly (sixteen weeks) than *M. bovis* (four to eight weeks).

USDA, Animal and Plant Health Inspection Service (APHIS) is the federal government agency responsible for tuberculosis programs in the United States. No official testing site has been mandated because there is no Uniform Methods and Rules (UM&R) for llamas and alpacas. However, the preferred site is the relatively fiberless area in the axillary space, and it is recommended that this site be used for either a single intradermal test or a comparative balanced bovine PPD and avian PPD test. The midlateral cervical area may be used, but that area requires clipping, and it is more difficult to pull up a fold of skin there to measure a response. Although the caudal aspect of the pinna has been used in Argentina and Australia, the study conducted by USDA in Ames, Iowa, found results of pinna injections were variable. Calipers should be used to measure the response.

Considerable research has been conducted during the last five years to establish a suitable tuberculosis diagnostic regimen. One of the first projects was conducted at Colorado State University in Fort Collins. It was there that the axillary space was first determined to be a sensitive site. Another experiment was conducted in Mexico on animals that were artificially infected. That study concluded that the axillary site was sensitive, but the response was more diffuse and slightly more difficult to interpret than that at the lateral cervical area. Canada had closed its borders to the importation of camelids in 1991. Following that closure, the Canadian government conducted some experiments on a few animals at Nepean, Canada.

In 1994, USDA investigators intratracheally infected four llamas at the National Veterinary Services Laboratory in Ames, Iowa, with 400 colony-forming units. Three of the animals became infected and were then followed with diagnostic procedures:

1. A drop of bovine PPD was instilled into the conjunctival sac. The body temperature was monitored at three, six, nine, twelve, and twenty-four hours to determine any elevation in temperature. The test was considered to be 80% sensitive, and specificity was 50% (because half of the animals developed conjunctival ulcer, probably from a reaction to phenol).
2. A comparative axillary intradermal tuberculin test was conducted. Based on a 3-mm increase in the double-fold skin thickness, the sensitivity was 66% to 75% and specificity 100%.
3. A tuberculin test was conducted on the caudal aspect of the pinna. Responses were variable, and it was concluded that the site on the ear was not suitable.

It must be reiterated that because of the small number of animals (four) used in this study, the results provide an indication but are not conclusive. Serologic testing on the USDA animals has not yet been reported.

Scientists at Iowa State University and Colorado State University are continuing to work to perfect ELISA tests.

A rather extensive investigation has been carried out in Argentina using large numbers of animals. Twenty-four llamas were infected intratracheally with live *M. bovis*. Twenty-four llamas were sensitized with *M. avium* antigen. Twelve llamas served as controls. The sites used included the axillary space, midcervical region, and pinna. A positive response was = 5 mm. Each of the tests demonstrated 100% sensitivity with slightly lower specificity. The comparative test could differentiate between avian and bovine infections. Blood was collected for serologic studies, which have not yet been reported.

Government Regulations

Personnel of regulatory agencies, with responsibility to control infection in cattle and to prevent human infection, don't want other animals moving freely about the country that could be carriers of mycobacteria. Thirty states require a negative tuberculin test within thirty days of shipment for interstate transport: Alaska, Arkansas, Colorado, Connecticut, Georgia, Hawaii, Indiana, Kentucky, Maine, Maryland, Massachusetts, Mississippi, Montana, Nebraska, New Hampshire, New Mexico, New York, North Dakota, Ohio, Oregon, Pennsylvania, Rhode Island, South Carolina, South Dakota, Utah, Vermont, Virginia, Washington, Wisconsin, and Wyoming. Pennsylvania also requires that animals moving within the state must have a negative tuberculin within 180 days and must be permanently identified (ear tag or tattoo). There are no current provisions for microchips. Currently, camelids are not included in any federal program for tuberculosis control; however, this could change quickly. A UM&R for the control of tuberculosis in cervids is in operation.

The llama and alpaca industries are caught in the middle of a disease challenge. Llamas and alpacas are not serving as a nidus for infection in other species, but camelids are being treated the same as cervids. It is not valid, but the sway of opinion is that camelids may constitute a threat to the tuberculosis-free status of a state.

Llamas seem to rarely develop tuberculosis. There is no absolutely reliable test to identify carriers. Regulatory agencies have the power to establish laws that would:

1. Prevent inter- or intrastate movement of llamas/alpacas.
2. Quarantine and test herds if there appears to be a threat to cattle or humans.
3. Confiscate and destroy positive tuberculin reactors.

Cooperation between regulatory agencies and representatives of the llama and alpaca industries has supported research that is slowly demonstrating that tuberculosis is an unimportant disease in camelids and that camelids are not a threat to livestock.

Treatment

Treatment is not allowed in the United States. In the past, permission has been granted to treat valuable zoo artiodactylids with isoniazid (5 to 10 mg/kg/day). It should be understood that isoniazid is not a mycobactericidal drug. The infection may be controlled and the tuberculin reaction suppressed, but if treatment is discontinued, the tuberculin response may reappear because the infection is still present.

Prevention

Tuberculosis vaccines are illegal in the United States.

Johne's Disease[131,169,188,200,203]

Definition and Etiology

Johne's disease (JD) (paratuberculosis) is a chronic, granulomatous disease caused by an acid-fast staining bacterium, *Mycobacterium avium paratuberculosis*, which is a ubiquitous organism throughout the world in temperate, subtropical, and tropical countries, is found in every state in the United States. *M. avium* spp. *paratuberculosis* is an obligate intracellular acid-fast rod found in clumps within macrophages in the mesenteric lymph nodes, lamina propria of the cecum and colon, and feces of infected animals. *M. avium* spp. paratuberculosis shares common cell wall antigens with other species of mycobacteria and other genera, including *Nocardia* spp. and *Corynebacterium* spp.[171] The organism is differentiated from saprophytic and other infectious mycobacteria by its slow growth in vitro and its requirement for complex media supplementation (mycobactin).

Various strains of *M. avium* spp. paratuberculosis exist. Sheep strains are notoriously difficult, if not impossible, to culture. Diagnosis must be made on the basis of other diagnostic tests. The strains cultured from camelids are presumed to be of cattle origin because they have cultured satisfactorily. Johne's disease occurs in NWCs and in OWCs. Bactrian camels are more commonly affected than dromedaries, possibly because of the different environments in which these animals are kept.

Epizootiology

Significant losses in milk production and mortality occur annually in dairy cattle in the United States and throughout the world. The organism may survive up to eleven months in manure and soil, and five

to nine months in pond water.[171] Exposure of the organism to sunlight, heat, and acid conditions, such as urine decomposition to ammonia, decreases survival. The chronicity of the diseases and the shedding of millions of organisms from infected cattle make it an insidious disease. An infected cow may shed up to 5×10^{12} bacteria per day. Antemortem diagnosis is difficult, particularly in an animal with a subclinical infection, yet such an animal may be a prolific shedder.

Transmission of the organism is primarily by the fecal/oral route, but the organism is also found in milk and is known to cross the placental barrier. It is a disease of poor sanitation, concentration of animals, and fecal contamination of feed and water.

The original source of infection in camelids has not been determined. Possible sources that have been considered include occupation of pastures and corrals previously inhabited by cattle or sheep, use of goat colostrum from infected goats, and direct contact with other infected animals (sheep, goats, cattle, deer, other camelids). None of these means of transmission have been verified as occurring in camelids.

It has been more than ten years since the original outbreak in Australian alpacas. No new cases have appeared, and thousands of agar gel immunodiffusion (AGID) tests have been conducted on alpacas in the state of Victoria, where the outbreak occurred. The two cases in Colorado were part of a herd of 200 llamas. No new cases have appeared, and the rest of the herd has remained negative to repeated testing for fifteen years.

Clinical Signs

The disease is characterized by a delayed onset, and variable expression of signs. The disease in adult cattle is characterized by chronic diarrhea and weight loss. In sheep, camelids, and deer, chronic weight loss is the primary sign, with diarrhea appearing in the terminal stage of the disease. Clinically affected animals are usually afebrile and have a good appetite until the terminal stages of the disease. The diarrhea is without blood or fibrin and usually has no special odor. In cattle, the clinical onset ranges from four months to fifteen years but is typically from two to five years. Once clinical signs appear, the course of the disease may last for two weeks or as long as six months.[170] Temporary remission may occur, especially during pregnancy, but stress may precipitate the onset of clinical disease.

JD has been reported in a number of species of zoo ruminants,[80] in numerous free-ranging ruminants, and in camelids.[14,17,155] The death of four llamas from paratuberculosis has been reported in the United States, and at least ten alpacas have been diagnosed in Australia. One zoo llama was diagnosed as having JD in England. Bactrian camels in Russia are susceptible, and it has been reported sporadically in dromedaries in the Middle East, Africa, and India.

Syndromes in Camelids

The majority of clinical cases of JD in camelids have been in young animals, less than two years of age. It is rare for the disease to become clinical in such young animals of other species.

LLAMAS. In the zoo llama in England, the clinical signs were acute, with severe diarrhea, weakness, prostration, and death in six days. It may be difficult to detect weight loss and emaciation if the animal is not palpated. The fiber coat hides the true body condition.

In Colorado a twelve-year-old female llama was found recumbent in a pasture, unable to rise. When examined at a hospital, she was dyspneic and had a hemogram indicating a degenerative left shift on the WBC. She was hypoproteinemic (3.9 g/dl), hypokalemic (3.2 mg/dl), ketonuric (2+), and hypoxemic, and had a metabolic acidosis. She was nine months pregnant, so a tentative diagnosis of pregnancy toxemia was made and treatment instituted. The second animal was a sixteen-month-old male llama that presented as a septicemia approximately a year following the first case. He was emaciated and had persistent diarrhea. He too was hypoproteinemic (3.7 g/dl) and ketonuric (2+).

A six-year-old llama was diagnosed as having JD in Oklahoma. She had a loose stool in late 1992 and delivered a cria in early 1993. She weaned the cria, but her condition deteriorated, and approximately a month following weaning she was euthanized. In the final two to three weeks she became extremely weak and then recumbent and developed a noticeable diarrhea.

The fourth U.S. case was another six-year-old female llama from a herd in Minnesota, consisting of six llamas and ten fallow deer. Over a period of three months, she became debilitated and weak, lost weight, and developed terminal diarrhea. JD was diagnosed by culture and histopathology at a diagnostic laboratory in South Dakota. A complicating factor in this case is that the remaining five llamas also died during the time described. These llamas lost weight and became debilitated but did not develop diarrhea. None of these animals were submitted for necropsy. The deer suffered no weight loss or diarrhea.

ALPACAS IN AUSTRALIA. JD was diagnosed in ten alpacas (eight: one to two years of age, one: six-year-old female, and one: four-year-old male).[161] Five of the cases had a history of weight loss, poor growth, and, terminally, diarrhea. The others showed no clinical signs but were positive on fecal culture and caprine AGID assay. Clinically affected alpacas had hypoproteinemia.

Diagnosis

Antemortem diagnosis may be difficult. Body condition is not easily determined by casual observation of the camelid because of the fluffy fiber coat. The definitive diagnostic procedure is culture and identification of *M. paratuberculosis* from feces or tissue. Unfortunately, the organism is slow growing (four to sixteen weeks, and it requires special enhanced media [Herrold's egg yolk medium containing mycobactin J and 4 g/L sodium pyruvate]). Other diagnostic tests used to screen herds and make a diagnosis in individual livestock species include AGID, complement fixation, competitive ELISA, histologic pattern of a granulomatous reaction, Ziehl-Neelsen staining (acid-fast) of tissue and feces, polymerase chain reaction (PCR), and DNA probes.

Differential Diagnosis

Salmonellosis, coccidiosis, intestinal nematodiasis, tuberculosis, eosinophilic enteritis, enterotoxemia, endotoxemia, and pregnancy toxemia should be excluded.

Necropsy

At necropsy of the London llama, the carcass was emaciated, lymphoid patches (Peyer's patches) were prominent in the intestine, mesenteric lymph nodes were enlarged, and, on cut sections, areas of caseous necrosis were evident. Histologic sections of the lymph nodes and lymphoid patches of the intestine contained numerous colonies of acid-fast-staining bacteria that were morphologically identical to *M. avium* spp. paratuberculosis.

Necropsy findings on the two cases encountered in Colorado included thickening (up to four times) of the caudal jejunum, ileocecal junction, and proximal large intestine; lack of body fat; and enlarged mesenteric lymph nodes. Large clumps of acid-fast rods were noted in the macrophages of the lamina propria and in the mesenteric lymph nodes. One of the llamas was a pregnant twelve-year-old. Cultures were negative on the nine-month-old fetus (transplacental transmission is known in cattle).

Necropsy findings in the ten Australian alpacas were essentially the same as those of the Colorado llamas. In addition to the intestinal mucosa and lymph nodes, granulomatous lesions were also observed in the liver, lungs, and lymphatics of the peritoneal serosa. PCR was employed to confirm gross and microscopic findings. The organism was cultured.

Treatment

No treatment is effective in reversing a clinical case or in clearing a carrier animal. Once a diagnosis of JD is made, a camelid should be euthanized to avoid continued contamination of the environment.

Prevention

Suspected infected animals should be isolated until a diagnosis can be made. The management of a herd in which a JD diagnosis has been made should be similar to the recommendations for managing an infected goat herd. The herd should be screened for subclinical infection. Culture-positive animals should be euthanized. Dung piles must be scrupulously cleaned daily, avoiding fecal contamination of feed and water (use separate forks, shovels, rakes, and brooms for cleaning and feed handling). Water should be provided in containers that can be cleaned and sanitized. The density of animals in an enclosure or pasture should be decreased.

Government Regulations in the United States

JD is not a reportable disease, and no national eradication program is in effect. Management compliance is voluntary except in such states as New York that restrict the movement of a culture-positive animal to anywhere but slaughter.

Considerable conflict has been generated since 1994 between the llama industry and some government officials over the ban of pack llamas from Canyonlands National Park in Southern Utah because of a supposed threat of transmission of JD from llamas to desert bighorn sheep (*Ovis canadensis nelsoni*). Although the weight of scientific evidence is contrary to that supposition, there is much misinformation and lack of understanding. Here is a summary of the currently known facts about JD in camelids:

1. Camelids can become infected and die from clinical JD.
2. The clinical syndrome in camelids is characterized by weight loss and terminal diarrhea.
3. The lesions of JD in camelids are similar to those observed in cattle, sheep, and goats.
4. The epidemiology of JD in camelids is virtually unknown.
5. In cattle, sheep, and goats, transmission of *M. avium* spp. paratuberculosis depends on the ingestion of large numbers of organisms by young animals within the first few months of life or continuous exposure of older animals to high doses of the organism. Casual exposure is not considered a source of infection.
6. Food and water, contaminated by feces from a shedding infected animal, is the most likely source of infection.
7. JD is an important disease in dairy cattle but not in beef cattle, presumably due to a

decreased opportunity for transmission on pasture or range conditions.

8. Llamas with clinical JD are likely to be debilitated and weakened and would be unable to withstand use as packers.
9. Llamas and alpacas have not been identified as responsible for transmission of *M. avium* spp. paratuberculosis to any other animal.
10. Typically, only animals affected with advanced clinical disease shed high numbers of the organism. Such individuals are unfit for packing.

Camelids do not pose a threat for transmission of JD to any other animal, according to the scientific literature and statements by knowledgeable scientists.

Anthrax

Anthrax is an acute, septicemic disease of many species of mammals, including camelids.[41] It is caused by *Bacillus anthraces*, a spore-forming, Gram-positive rod that is a constituent of the normal flora of many soil types worldwide. Spores may persist in soil for years. Infection is usually acquired by ingestion of feed or water that has been contaminated with soil containing the spores. Outbreaks are usually sporadic and may follow marked climatic changes such as heavy rainfall, flooding, or drought.[101]

Clinical Signs

Cases of anthrax have been reported in SACs both in Peru and the United States. Insufficient numbers of cases have been studied to establish the incubation period in camelids, but manifestation of the disease appears to be uniform throughout most species. The incubation period ranges from one to fourteen days, with the more common length being three to seven days. The first sign of anthrax is fever, as high as 42°C (108°F), but this may be missed. The general signs of total anorexia, stomach stasis, colic, hematuria, and hemorrhagic diarrhea may be more evident.

Sudden death may occur without premonitory signs being observed. If an animal survives for twenty-four hours, subcutaneous swellings may be seen on various parts of the body. Hemorrhagic discharges may exude from all body openings. Dyspnea indicates pulmonary involvement. Ultimately, the animal becomes severely depressed, convulsive, or comatose and may die in one to three days.

Diagnosis[35]

Anthrax may be confused with other diseases that cause sudden death or produce septicemia. *B. anthraces* is easily cultured from the tissues of the carcass. If anthrax is suspected, it is prudent to avoid a complete necropsy to preclude further contamination of the soil with the organism. A small quantity of blood is sufficient for the laboratory to make a direct smear or culture. A fluorescent antibody test is also available.

Treatment

If a rapid diagnosis is made, *B. anthraces* is susceptible to many antibacterial agents, including penicillin and tetracyclines. Therapy should be continued for five or more days, depending on the response.

Prevention

Live-spore bacterins are routinely used to protect cattle, sheep, goats, swine, and horses in endemic areas. Similar bacterins have been used in camelids, but they should be used carefully.[148,157] The dose of vaccine must be adjusted to the size of the animal. The live spores in the bacterin must germinate and grow in the animal's body to stimulate antibody production, but it is possible for the body's defenses to be overwhelmed and for overt infection to result from the bacterin.

Anthrax was diagnosed in a herd of llamas, and live-spore bacterin was administered to all other animals in the herd, including the neonates. Two nursing babies died within two weeks following inoculation, with signs and lesions of anthrax. The infection was thought to be bacterin induced. Bacterin-induced anthrax has also been reported in foals.[26]

The Sterne strain of anthrax bacterin[1] is currently recommended. The dose for sheep is suggested for adult SACs, and a fourth to a half dose is recommended for neonates and weanlings.[101] A second dose is recommended two to four weeks following the first.

It is inappropriate to administer antibiotics simultaneously with the bacterin, because this will inhibit or prevent development of antibodies.

Brucellosis[69]

Brucellosis is not a major disease of camelids, but they have been proven to be susceptible to *Brucella melitensis* type 1.[1,114] One significant outbreak in a herd of alpacas in Peru has been described, with classic signs of brucellosis. In the study, of 1,449 alpacas tested, 20.9% had plate agglutination titers greater than 1:25. More than 25% of the seventy-nine people caring for the animals also developed positive titers, some with active undulant fever.

B. melitensis is the primary species found in goats and sheep. It was felt that sheep were the source of infection in the Peruvian herd.[1] Transmission in camelids is assumed to be the same as in cattle, sheep, and goats, with the usual route being ingestion of feed or water that has been contaminated with the body excretions of infected individuals. The placenta and fetal fluids of infected individuals are a significant source

of the organism. The organism is also found in the milk.

Three llamas died at the London zoo a few weeks after contact with newly imported camels from Moscow.[55] Serum titers for *B. melitensis* were greater than 1:1,000, indicating active infection. Camels have been shown to be affected by both *B. abortus* and *B. melitensis*. Nothing is known of the natural susceptibility of camelids to *B. abortus*, *B. ovis*, or *B. suis*. *B. abortus* infection (including abortion) has been produced experimentally in llamas, but no natural cases have been reported.

Clinical Signs

Abortion may occur in the last third of gestation, at nine or ten months. Affected crias may be born dead or die shortly after birth. The extent to which brucellosis interferes with fertility in camelids is not known. There were no reports of epididymitis or orchitis in the alpaca outbreak.[2]

Alpacas did not develop retained placentas, as is common in cattle and sheep. This may be a result of the difference in the placental attachment (Chapter 17).

Pathology

Mortality is nil except for fetuses. Lesions of the fetus and placenta of SACs are similar to those found in cattle specimens.

Treatment

B. melitensis is a Gram-negative coccobacillus, sensitive to many broad-spectrum antibiotics. Camelids with positively diagnosed brucellosis should be treated for two to three weeks to eliminate development of the carrier state in the female. Regulations in areas in the United States that are certified brucellosis free may require euthanasia of positive reactors.

Prevention

Any preventive program must be integrated with the programs of regulatory authorities in a given area (see preceding note). Strain 19 bacterin used to control brucellosis in cattle is inappropriate for camelids because the bacterin contains MLV antigens.

Government Regulations

Personnel of the USDA have similar concerns for brucellosis as they do for tuberculosis. There is no UM&R for brucellosis in camelids, but studies have been conducted by USDA scientists to establish the validity of bovine testing procedures in camelids. Twelve llamas were donated by the llama community. Animals were separated into four groups:

1. Low-dose group (9×10^5): viable *B. abortus* strain 2308 instilled into the conjunctival sac.
2. High-dose group (9×10^8): viable *B. abortus* was given.
3. Killed antigen group (9×10^5): heat-killed *B. abortus* was used.
4. Diluent group.

Blood samples for serology and cultures from various locations were collected at appropriate times. All of the llamas were euthanized at the conclusion of the studies and detailed necropsies performed.

Cultures were positive on one or more tissues in animals that had been infected with viable organisms. The aborted fetus from a pregnant animal yielded *B. abortus*. All infected llamas developed high serologic titers by five weeks postinoculation that were detectable on the buffered acidified plate antigen (BAPA) test, card test, standard plate test (SPT), and Rivanol test. The particle concentration fluorescence immunoassay test (PCFIA), standard tube test (STT), complement fixation test (CFT), and D-tec ELISA were less consistent and less reliable indicators of infection.

A brucellin intradermal response was also measured, but results were highly variable, and this test was not considered suitable.

The study summary stated that llamas are susceptible to experimental infection by *B. abortus* and that infected llamas develop positive serological titers using conventional bovine diagnostic tests (BAPA test, standard plate test, card test, and Rivanol test). Llamas develop histological lesions similar to those found in cattle, sheep, and goats, and they develop a delayed type hypersensitivity reaction to brucellin.

Listeriosis[42,47,64,77,127,136,194]

Listeriosis (circling disease, listerellosis, silage disease) is caused by *Listeria monocytogenes*, a Gram-positive nonspore-forming coccobacillus that has a worldwide distribution. The disease is infectious but not highly contagious, causing sporadic occurrence in a broad range of animal species, including NWCs and OWCs. The herd morbidity is low, but the mortality rate of affected individuals is high, approaching 100%.

L. monocytogenes may be isolated from the intestinal tract of healthy animals and may remain dormant in the soil for years. The organisms may be saprophytic, living in a plant-soil environment, and infection may develop in domestic and wild animals or humans at any time.[33] Infection does not become clinical unless resistance has been impaired by stress, concurrent disease, or pregnancy.

Clinical Signs[128,176]

Camelids develop an encephalitic syndrome, similar to that seen in cattle, with unilateral facial paralysis, circling, trembling of the head, running into objects,

erect ears that may each be pointed in a different direction, salivation, depression, and a fever as high as 41.5°C (107°F).[23,101,120,174] Death occurs two to five days after onset of the first signs of illness. Abortion in late gestation was not described in the case in Peru, but it is a common occurrence in all other species studied.

Listeriosis was responsible for the deaths of six of the eight llamas in a German zoo. Other species of animals were also involved in an apparent epizootic. Because of the explosive outbreak, MLV bacterins were given to all ungulates in the zoo, including llamas, with no apparent untoward effects. Two adult llamas contracted encephalitic listeriosis in New York.[30] Signs included abortion, facial paralysis, ataxia, depression, and recumbency.

Pathology

No gross lesions have been described in camelids. Histopathologically, miliary abscesses and perivascular cuffing, with large mononuclear cells, are especially prominent in the medulla oblongata.

Treatment and Prevention

Treatment of clinical cases has been unsuccessful to date. The disease appears to be rare in camelids; therefore, no vaccination program is recommended.

Leptospirosis

Leptospirosis is caused by various serovars of the genus Leptospira, a spirochete that may be visualized by use of special staining techniques. Fortunately, only a few of the more than 100 serovars of Leptospira cause disease in North American domestic animals.[60] However, worldwide, the genus affects a broad range of hosts. There are reports of leptospirosis in both OWCs and NWCs.[119]

Epidemiology

The epidemiology of the disease in camelids is unknown but is presumably similar to that of other species. One or more primary hosts maintain each serovar as a reservoir for nonadapted species. The organism is shed to the environment from primary hosts and infected secondary hosts via the urine and can remain viable in ponds for as long as three months.

The infection is acquired from contaminated surface water through abrasions on the skin or through exposed mucous membranes.[60] The organisms invade the liver, producing necrotic foci (leptospiremia), and secondarily affect the kidney, lung, reproductive organs, and brain.

Clinical Signs

The effects of leptospirosis are not clearly defined in camelids. One ranch experienced an abortion epizootic. From studies considering a number of diseases, it was concluded that *L. grippotyphosa* was implicated.[m]

Leptospirosis has been diagnosed at necropsy in a zoo guanaco.[84] This animal was dyspneic and refused to rise. On closer examination, the guanaco was icteric, anuric, and passed no feces. Serum creatinine was 5.5 mg/dl, and the blood urea nitrogen was 80 mg/dl. These values are not markedly elevated but were significant in the overall evaluation of the case. The guanaco died forty-eight hours after the first signs were observed.

Customary signs noted in cattle and sheep include fever, hemoglobinuria, icterus, anemia, encephalitis, mastitis, pulmonary congestion, orchitis, and abortion. The kidneys and reproductive organs are most often involved.

Pathology

Lesions are consistent with the signs noted. Gross lesions may be minimal but may include edema of the lungs and icterus of the fat, mucous membranes, and fascia. Histologically, there may be acute tubular and interstitial nephritis. The organisms may be observed in tissue sections if special stains are used.

Diagnosis

Serologic testing, coupled with clinical signs, is the most accurate diagnostic method. Titers of more than 1:100 using the microscope agglutination technique are considered significant. Acute sera and convalescent sera collected two weeks following the first sample are most helpful, because it is difficult to interpret a titer from a single sample.

Leptospira may be isolated from the living animal but only during and shortly after the acute stage of the disease.

Treatment

Leptospira are susceptible to many broad-spectrum antibiotics. The success of therapy depends on the degree of tissue and organ damage done prior to treatment. Symptomatic and supportive treatment is indicated. Renal function and urine output must be continuously monitored and fluid and electrolyte therapy adjusted accordingly.

Prevention

Multiple-serovar leptospiral bacterins are commercially available. These have been administered to camelids with apparent safety and efficacy, although no experimental challenge studies have been conducted.[82,153] Bacterins containing appropriate serovars for the area, as determined by diseases being diagnosed in livestock, should be used.

Necrobacillosis[135]

Etiology and Epidemiology

Fusobacterium necrophorum (Sphaerophorus necrophorus) is a Gram-negative, anaerobic bacterium. The organism has worldwide distribution and has been implicated in numerous disease processes in a broad host range of domestic and wild animals. *F. necrophorum* is not highly tissue invasive. Usually, a break in healthy epithelium or devitalized tissue is required to provide a portal for entry.

There is evidence to support the concept that *F. necrophorum* forms a symbiotic relationship with other organisms such as *Arcanobacterium pyogenes*, the toxins of each enhancing the effects of the other. Multiple organisms are often isolated from necrobacillosis lesions, particularly from abscesses in the lungs or articulations.

Necrobacillosis usually occurs sporadically. If climatic and other environmental factors cause long-term stress, outbreaks of the disease may result.

Clinical Signs

Clinical signs depend on the location of the lesions, which may develop on the lips, tongue, palate, pharynx, larynx, interdigital space, sole of the foot, stomach, or mandible or maxillary bones. Young animals are usually more at risk. Oral and pharyngeal lesions are characterized by fever (40.5°C or 107°F), anorexia, salivation, excessive drinking of water, and depression. Dyspnea and open mouth breathing accompany laryngeal lesions. Oral necrobacillosis lesions produce a characteristic foul odor that emanates from the mouth and nostrils of affected individuals. Infected particles from the mouth and throat may be aspirated, causing pneumonia.

Ulcers and diphtheritic membranes may be observed in the rostral oral cavity. The pharynx and larynx can be inspected with a laryngoscope or a fiberoptic scope only after the animal has been sedated.

Lameness may result from necrotic lesions in the interdigital space and on the footpad (infectious pododermatitis) (Chapter 10). Foot lesions are extremely resistant to therapy and, if cleared up, tend to recur with little provocation.

Diagnosis

F. necrophorum may be isolated from lesions using standard anaerobic microbiologic methods. The necrotic lesions are characteristic, with ulcerations containing variable amounts of surface debris. Raised diphtheritic lesions are typically observed on or within the larynx and the stomach.

Treatment

The author has had little success in treating necrobacillosis in camelids as well as in other species. In vitro, the organism is susceptible to many antibiotics, but the nature of the lesion precludes penetration of antibiotics.

Environmental stressors must be alleviated, which may be difficult. In a llama with infectious pododermatitis, prolonged topical therapy and systemic antibiotics resulted in healing of the pad lesions, but when the animal was returned to its original environment, the lesions recurred.

Prevention

No bacterins have been developed that protect against this disease. Environmental stressors should be minimized.

Osteomyelitis of the Mandible

Etiology and Epidemiology

Inflammatory swellings of the mandibles are seen in mammals throughout the world. Such swellings may be caused by dental anomalies or infections, tumors, trauma, or infection of the bone by osteolytic or proliferative bacteria. It has been suggested that the organisms gain access to the bone via disruptions in the mucous membranes of the mouth.

There may be regional differences in the organism responsible for the infection. In Australia, marsupials develop osteolytic lesions of both mandible and maxillary bones, and researchers there have incriminated *Fusobacterium necrophorum* as the primary etiology. Marsupials in North America also suffer from mandibular lesions, but the osteitis is usually proliferative, and the implicated organism is *Actinomyces* spp. Both Actinomyces and Fusobacterium should be considered in a differential diagnosis in camelids.

A new species of Actinomyces has been identified by microbiologists at the University of California. It has been designated *A. lamae*, but that name is not yet official. It is awaiting confirmation from DNA studies. *A. lamae* causes abscesses in a variety of soft and osseous tissues and has been isolated from lumpy jaw cases.

Necrobacillosis and cornynebacteriosis lesions have been observed in both domestic[29] and wild camelids (Figures 7.19 to 7.21).[32] The occurrence is sporadic and not of major economic importance.

Clinical Signs

Firm swellings along the rami of the mandible are easily observed and palpated.[119] Little or no pain may be associated with palpation. General signs of anorexia, fever, and depression are usually absent. Variable

Figure 7.19. Mandibular osteomyelitis in a llama.

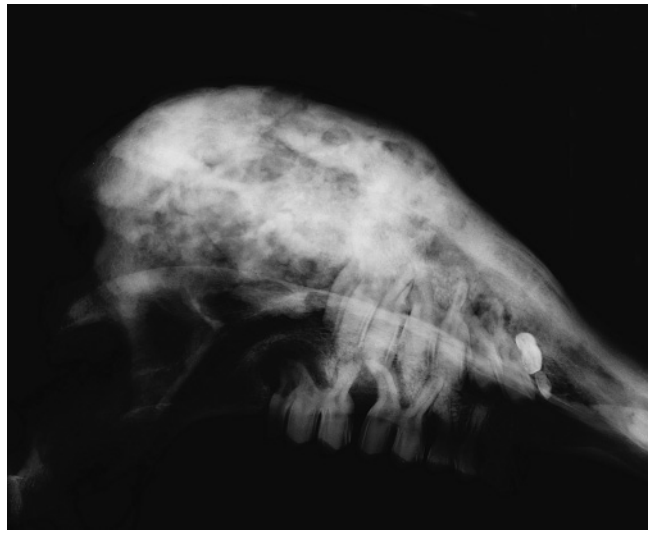

Figure 7.20. Radiograph of a llama mandible with osteomyelitis.

Figure 7.21. Jaws of alpacas affected with alveolar osteomyelitis, presumably caused by *Fusobacterium necrophorum*.

degrees of soft-tissue involvement are encountered, and a fistulous tract may lead from the lesion to the ventral border of the jaw. The odor of the exudate of a dental infection or a necrobacillosis lesion is foul, but little or no odor may emanate from infections with other organisms. If swellings are in the vicinity of the teeth, the examination should investigate dental involvement (Chapter 6). Swellings are usually unilateral, but bilateral involvement has also been observed.

Diagnosis

Radiographic evaluation of the lesion is imperative to determine whether the lesion is lytic or proliferative and if the teeth are involved. Isolation and identification of the infective microorganism may be difficult but are necessary for definitive diagnosis. Surgical extirpation and microscopic examination of the lesions may aid in the diagnosis.

Treatment and Prevention

Therapy is entirely dependent upon the etiologic agent and the nature of the lesion and may include surgery, administration of antimicrobials, and supportive therapy. No effective preventive measures have been developed.

Streptococcosis[38,75,98,164,175,212,215]

Etiology

The following species of Streptococcus have been isolated from alpacas in Peru and are considered to be constituents of the normal flora of mucous membranes: *S. zooepidemicus, S. pyogenes, S. faecalis, S. uberis, and Streptococcus group E*. Various species of Streptococcus have been isolated from abscesses in camelids.[22]

S. equi subsp. *zooepidemicus* is the etiologic agent of "la fiebre de las alpaca" (alpaca fever) in Peru.[14,16,17,51,52,111,116,137,140–142] This organism is also responsible for a variety of syndromes in livestock and horses worldwide. Streptococci are also responsible for diarrhea in crias.[58,154]

Epidemiology

S. equi subsp. *zooepidemicus* is the causal agent of strangles in horses and is ubiquitous in horse populations. Alpaca fever is one of the most important diseases of SACs in the Altiplano of South America. The morbidity may be as low as 5% to 10%, but the mortality rate of those affected varies from 50% to 100%. Transmission is via oral ingestion from contaminated objects or direct contact from other animals. The systemic forms are frequently associated with a stressor such as inclement weather or malnutrition.

Clinical Signs[98]

Alpaca fever occurs in acute, subacute, and chronic forms. The acute and subacute forms are usually seen in young animals. Signs include anorexia, recumbency, depression, and fever as high as 41.2°C (106.4°F). The morbidity rate in a herd is only 5% to 10%, but the mortality rate in those affected may be 50% to 100%.

If infection becomes systemic there is a polyserositis involving the lungs and serosae of the thoracic and abdominal cavities. The signs of polyserositis include dyspnea; colic; a tense, tender abdomen; and cessation of defecation. Meningitis was seen in an outbreak in alpacas in Kansas.[98] Death may occur four to eight days after signs are observed.

Wound, preputial, and mammary gland infections result in local, painful edema and cellulitis. Chronic forms, more commonly seen in adults, are essentially abscesses or focal infections. Abscesses may be either external or internal.

Diagnosis

Diagnosis involves the correlation of signs and lesions and isolation and identification of the organism. The lesions of acute and subacute cases include significant quantities of fibrinopurulent exudation of the thoracic and abdominal cavities along with pleuritis, peritonitis, pneumonia, and petechial and ecchymotic hemorrhages of all serosal surfaces.[30] Balanitis and mastitis have also been observed. Isolation and identification of the organism is definitive; however, mixed infections occur.

Treatment and Prevention

Localized abscesses should be lanced and irrigated. Antibiotic therapy is not indicated, except for systemic forms. Streptococci are responsive to antibiotic therapy, but sensitivity patterns should be developed to avoid prolonged therapy with antibiotics to which the organism may be resistant. It is important to correct any predisposing factors.

Although mixed streptococcal bacterins are available, there is little evidence of their efficacy.

Colibacillosis

Etiology And Epidemiology

Escherichia coli is a ubiquitous, Gram-negative, enteric bacterium. The organism may be a constituent of the normal flora of the intestinal tract, but under favorable conditions, it may become a pathogen. *E. coli* is the most common cause of uterine infection seen at the author's clinic and has been isolated in pure culture from septicemic neonates. In Peru, an atypical diarrhea of crias is felt to be a result of colibacillosis. In contrast to enterotoxemia, in which the best-conditioned animals are involved, in colibacillosis the thin, undernourished animal is most likely to contract the disease. Malnourishment, as a result of decreased maternal milk production, may predispose, as does poor sanitation.

Transmission occurs via ingestion or through the umbilicus.

Clinical Signs

The most serious disease of SACs involving *E. coli* in the United States is neonatal septicemia, followed by metritis, mastitis, and abscesses. The typical signs of illness in alpaca crias are profuse diarrhea (colored whitish, yellowish, or greenish) lasting for five to twenty days, weight loss, abdominal distention, no fever, pica, and debility.[109]

Diagnosis

Clinical signs and lesions are not diagnostic. Isolation and identification of the organism is necessary for a confirmed diagnosis. Colibacillosis may also be seen

as a secondary infection accompanying such diseases as enterotoxemia.

Lesions are consistent with the organ systems involved in the infection.

Treatment and Prevention

Successful treatment of *E. coli* septicemias requires intensive supportive and antibiotic therapy. The aminoglycoside antibiotics are most often effective and should be begun immediately, until results of sensitivity testing are available, when more precise therapy may be instituted.

Melioidosis[20]

Etiology

Burkholderia pseudomallei is the etiologic agent. This is an aerobic, Gram-negative, motile rod.

Epidemiology

The organism is found in soil and water in endemic subtropical and tropical areas around the world. The disease has been reported in camels maintained in swampy or wet environments in northern Australia. Affected hosts include many species of mammals, humans, and camelids. Transmission is by direct contact with the organism.

Clinical Signs

The disease may infect different organ systems in various hosts. Primarily the infection is in the lungs, joints, uterus, mammary glands, and lymph nodes. The camels had pneumonia and septicemia.

Diagnosis

The organism may be cultured. PCR technology is used to differentiate specific serotypes.

Management

No vaccines are available. Antibiotic therapy should be based on culture and sensitivity. Tetracyclines and fluoroquinoilones are generally used.

Pasteurellosis

Etiology

Pasteurella spp. and *Mannheimia* spp.[45] are common causes of respiratory infection in numerous species of animals. The species of concern in camelids are *Pasteurella multicida* in OWCs and *Mannheimia haemolytica* reported in NWCs. These organisms are small Gram-negative rods or coccobacilli, nonspore forming and facultative anaerobes. These organisms produce several proteinaceous exotoxins that are important in the pathogenesis of the disease.

Epidemiology[183]

These organisms may be normal inhabitants of the respiratory mucous membranes. Stress is a major predisposing factor. Transmission may occur from direct contact, inhalation, or ingestion.

Clinical Signs

Infection is typically associated with the respiratory system, with septicemia a common sequel. The syndrome is characterized by fever, nasal discharge, lacrimation, dyspnea, diarrhea, congestion of mucous membranes, and lymph node swelling.

Diagnosis

Pasteurella and Mannheimia may be cultured from the exudate from the respiratory tract and infected tissue. DNA probes and PCR technology are used to identify specific strains.

Management

Broad-spectrum antibiotics are indicated based on culture and sensitivity. Vaccines are used in ruminants, but have not been reported to be used in camelids.

Ehrlichiosis/Anaplasmosis

Etiology

Ehrlichia spp. and *Anaplasma* spp. were formerly classified as parasites. They have been reclassified as tiny bacteria in the Order Rickettsiales. Other name changes have complicated the determination of which organism causes which disease. *Anaplasma* spp. infect erythrocytes, while *Ehrlichia* spp. are intracellular infections of the leukocytes. *Ehrlichias* are obligate intracellular organisms that have a cell wall but are extremely tiny (<4 μm)

Ehrlichia (formerly *Cowdria*) *ruminantium* is the etiologic agent of African heart-water disease of wild and domestic ruminants. An ehrlichia was observed in the blood of a sick llama in California. DNA technology showed that the species was related to the *Ehrlichia phagocytophilia* geno-group.[15]

Epidemiology

Ehrlichias are transmitted by tick bites. The organism is found primarily in subtropical and tropical climates around the world. Various reservoir hosts have been identified. In the case of the California llama the reservoir host was likely coastal black-tailed deer (*Odocoileus hemionus*). The organism was also isolated from ticks from the llama farm.

There have been no reports of naturally occurring cases of anaplasmosis as a result of *Anaplasma marginale* infection in camelids. However, alpacas have been

infected experimentally with blood from infected cattle.[54] Anaplasma bodies were observed in the blood twenty-nine to 120 days following inoculation, but the infection was subclinical, even though hemoglobin and erythrocyte levels were reduced in the affected alpacas.

Clinical Signs

Clinical signs in the llama included inappetence, lethargy, and slight ataxia. Ultimately the llama became recumbent. On hematology there was a mild lymphopenia, monocytosis, and eosinophilia. Intracytoplasmic inclusion bodies of ehrlichia were observed in the neutrophils.[n]

Diagnosis

Antemortem diagnosis is by indirect immunofluorescence and PCR assay of the blood.

Management

Tetracycline antibiotics are effective early in the course of the disease.

Mycoplasmosis

Etiology

Mycoplasma haemolamae, previously classified as a protozoan parasite (*Eperythrozoon* spp.) is now in the bacterial family Rickettsiae.[46,90,97,129,130] The organism is closely related to *Anaplasma* spp. *M. haemolamae* is unique to SACs.

M. haemolamae is a small bacterium and devoid of a cell wall. Organisms are generally <1 µm in diameter and have a coccoid, rod, or ring shape. The organisms are on the surface of erythrocytes but do not penetrate the cell wall.

Epidemiology

The organisms are presumably transmitted by bloodsucking arthropods, although intrauterine transmission is recognized.

Clinical Signs

No overt disease may be evident, but fever, mild to severe anemia, depression, icterus, infertility, edema, poor growth, and mild to severe hypoglycemia may be seen.[5]

Diagnosis[158]

The organisms may be observed on blood smears made soon after drawing the blood. Otherwise the organisms fall away from the erythrocytes and appear as cellular debris on the smear. There is a cyclic bacteremia, so not finding the organisms on a smear is not definitive for a negative diagnosis. A PCR assay is definitive.

Management

Several antibiotics have been used experimentally. Enrofloxicin is ineffective. Oxytetracycline suppresses infection, but doesn't eliminate it. Blood transfusion may be necessary when the PCV is less than 10%.

Miscellaneous and Minor Bacterial Infections[72,170]

Numerous bacteria have been isolated from single cases of infection involving camelids.[83,86] Infection is sporadic, possibly opportunistic, and may be of no economic importance. Isolation of an organism from mucous membranes may not determine etiology.[159] Enterococcus has been reported as the etiology of septicemia in a llama.[29]

Other Gram-negative bacterial isolates include various species of the genera Citrobacter, Enterobacter, Pseudomonas,[3] Campylobacter,[120] Proteus, *Klebsiella pneumoniae, and Actinobacillus* spp.[3]

Salmonellosis does not appear to be a major disease in either crias or adults. However, two cases of septicemic salmonellosis have recently been reported in the literature.[7] One was characterized by a generalized septicemia, with fibrinopurulent pleuritis, pericarditis, and peritonitis caused by *Salmonella choleraesuis* var. *kunzendorf*. This species is generally considered to be host-specific to swine, and the affected llama had been in contact with swine. The other case was a premature neonate, infected by *S. typhimurium*. The signs included weakness, listlessness, dehydration, and recumbency. At necropsy there was petechiation of mucous membranes, hydropericardium, hydrothorax, pulmonary congestion, and gastrointestinal hemorrhage.[6]

The genus Corynebacterium is important in camelid infections. *Arcanobacterium* (*Corynebacterium) pyogenes* is frequently isolated from superficial abscesses in llamas and may also be a primary pathogen in endometritis, mastitis, and internal abscesses. *Arcanobacterium pyogenes* abscesses are thin walled and filled with thick, nonfetid exudate. (Recall that *Arcanobacterium pyogenes* may act synergistically with *Fusobacterium necrophorum* to facilitate necrobacillosis.)

Corynebacterium (Rhodococcus) equi is primarily an equine pathogen, but chronic granulomatous lymphadenitis has been reported in other species. Multiple caseous abscesses have been found in the lungs, liver, and spleen of a llama at the University of California and is also reported from the literature (Figure 7.22).[87,] In Peru, *Corynebacterium pseudotuberculosis* has been reported as an isolate from abscesses in camelids (Figures 7.23A and B).[71]

In a single case of keratoconjunctivitis in a llama, *Staphylococcus aureus* was isolated from the conjunctival sac, while *Moraxella liquefaciens* was isolated from the cornea.[28] The clinical signs were photophobia; blepherospasms; lacrimation; conjunctivitis; miosis;

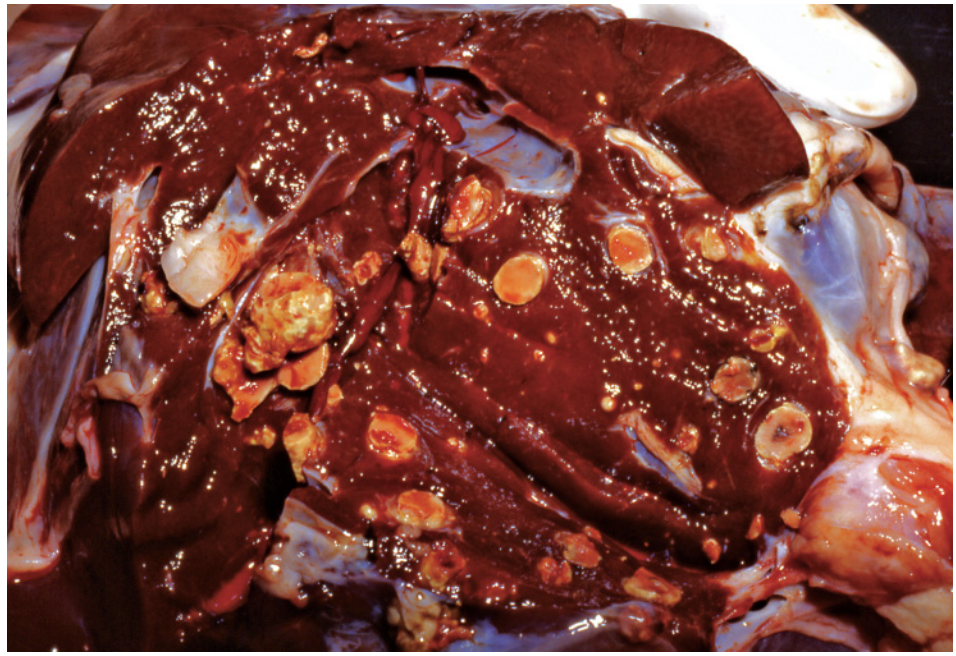

Figure 7.22. ***Corynebacterium (Rhodococcus) equi*** **abscesses in a llama liver.**

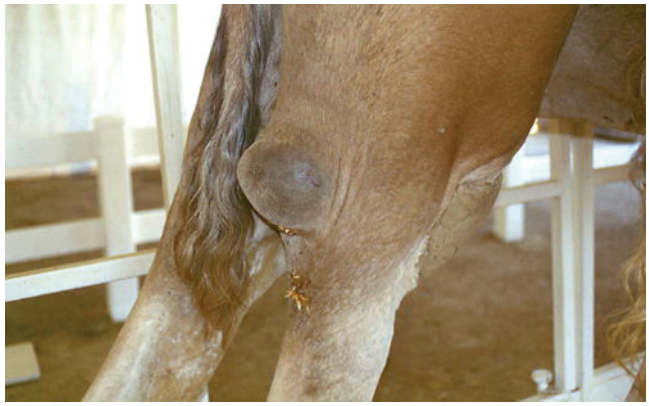

Figure 7.23A. ***Corynebacterium psuedotyberculosis*** **abscess in a camel.**

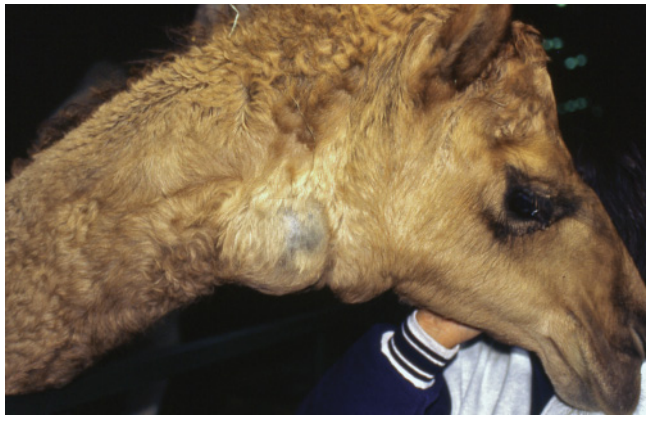

Figure 7.23B. ***C. pseudotuberculosis*** **abscess in a camel.**

and focal, unilateral corneal opacity. A 5-mm central corneal ulcer retained fluorescein stain.

The mixed infection was treated with a subconjunctival injection of benzathine penicillin (300,000 units) plus systemic administration of antibiotics and atropine. The corneal ulcer healed completely in thirty days.

Staphylococcus aureus has also been isolated from an alpaca with dermal abscesses. The diagnosis from the surgically extirpated lesion was botryomycosis, which is defined as a chronic purulent granulomatous infection (Figures 7.24A and B). Botryomycosis was first thought to be a fungal infection, hence the name, but was subsequently identified as a local skin infection caused by *S. aureus*. The abscesses in the alpaca varied from 0.5 to 4 cm in diameter and were located on the medial aspect of the thigh (Figures 7.24A and B). Treatment was successful and consisted of surgical extirpation of the abscesses followed by broad-spectrum antibiotic therapy for four weeks.

Lyme disease caused by *Borrelia burgdorferi* has affected humans and many animal species (both domestic and wild) since it was first reported in 1975. *B. burgdorferi* is a bacterial spirochete that is transmitted via tick bites. Tick species and their hosts vary from region to region in the United States. There is only circumstantial evidence that camelids have been infected. In an area where Lyme disease is endemic, the disease should be considered in a differential diagnosis if animals have arthritic, cardiovascular, or neural involvement.

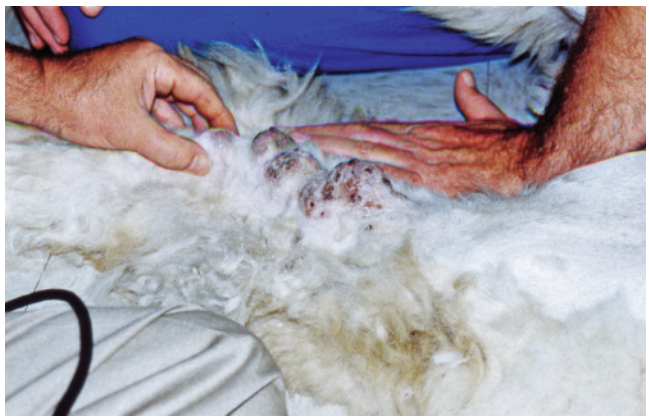

Figure 7.24A. Botryomycosis (*staphylococcosis*) in an alpaca.

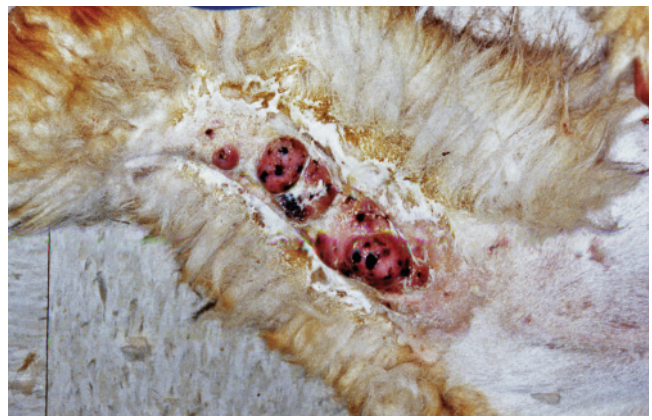

Figure 7.24B. Botryomycosis in an alpaca.

Bacteroides fragilis is a large, Gram-negative, nonmotile, anaerobic rod. It is the most common anaerobe causing infections in humans and recently is being increasingly isolated from animals.[93] *Bacteroides fragilis* was isolated in pure culture from an internal abscess in a llama. In the author's clinic, *Bacteroides* spp. have also been cultured along with *F. necrophorum* from a llama with infectious pododermatitis.

Pododermatitis—A Case Report

In April 1985, a gelding llama developed erosions and ulcerations of the footpads. A practitioner prescribed application of astringent solutions to the feet with and without bandages. In November, the llama was referred to the veterinary hospital of the University of California for evaluation and consultation.

Ulcerative lesions were present on all four footpads and interdigital spaces. The lesions were debrided and biopsied, and specimens were cultured. Initial therapy consisted of soaking the feet in dilute povidone-iodine solution (1:4) for fifteen minutes once daily, followed by application of a formalin solution (1:10). The feet were then bandaged.

Reports of the first biopsy supported a diagnosis of severe suppurative necrotizing pododermatitis, accompanied by periarteritis and arteriolar hypertrophy. Several colonies of bacteria grew on the original cultures, including two unspeciated Gram-negative aerobes and streptococci. Anaerobic cultures yielded *F. necrophorum* and *Bacteroides* spp. Bacteroides is highly resistant to most antimicrobial therapy but has been found to be sensitive to dimetridazole (Flagyl). A seven-day course of medication with dimetridazole was initiated at a dosage rate of 7.5 mg/kg administered intravenously four times daily. Foot soaks were continued. The llama tolerated the therapy well, with the exception that salivation and drooling occurred with each treatment. The ulcers became quiescent, and epithelialization began at the periphery. The topical medication was changed to an astringent solution (white lotion, zinc sulfate, and lead subacetate), and the llama was discharged with some of the smaller lesions completely healed and the larger ones obviously healing. The owner was instructed to continue application of the white lotion and bandaging until healing was complete.

In June 1986, the llama was returned to the hospital, severe pododermatitis having recurred on all four footpads. Although IV therapy with dimetridazole had been clinically effective, the expense of the medication ($120 per day) was now prohibitive for the client, and topical medication was elected. The feet were soaked in a 5% tannic acid solution for fifteen minutes, followed by application of dilute povidone-iodine solution (1:4) and pine tar under a bandage. Although the llama was docile and easily handled, the bandage changing was painful, and it was necessary to sedate the llama. Xylazine (0.04 mg/kg) and butorphanol (0.04 mg/kg) were administered intravenously. Initially, the treatment regimen was carried out daily. After a month it was conducted every other day, and at the end of a four-month period, every third day. Healing waxed and waned, but the ulcers progressively epithelialized, and by October the llama was discharged once again.

Unfortunately, the pododermatitis recurred a third time, and in January 1987, a decision was reached to euthanize the llama. Radiographs of the severely affected feet indicated no bone involvement. At necropsy, the infection was found to be restricted to the sole and adjacent corium of the foot and the skin and subcutaneous tissue of the interdigital space. The digital cushion was not invaded. Sections were reported as epidermal necrosis and ulceration.

Actinomyces has been mentioned in connection with osteomyelitis of the mandibles. An atypical species of Actinomyces given a temporary name of *Actinomyces lamae* was isolated from a thick-walled abscessed tract in the cheek of a llama. Repeated lancing and irrigation failed to clear up the infection. Only with radical extirpation of the entire lesion was therapy successful.

Dermatophilosis (streptothrichosis, streptotrichosis, mycotic dermatitis, lumpy wool disease) is caused by the actinomycete *Dermatophilus congolensis* (*Streptothrix congolensis*).[19] Practitioners have reported cases of dermatophilosis in llamas and alpacas at meetings, but there are no published reports of clinical cases. The organism is known to cause a severe form of dermatitis in camels.[211] The disease is characterized as an ulcerative, exudative dermatitis.[19] The organism is found worldwide, but infection is more prevalent in areas of high rainfall and in tropical and subtropical climates. Administration of antibiotics, including penicillin and tetracyclines, has been highly effective in treating dermatophilosis.

For centuries, camelids were thought to be endemically infected with syphilis, *Treponema pallidum*.[163] European invaders sought to discredit indigenous people and their animals, and when syphilis appeared in the Andean countries, wreaking havoc with the nonresistant indigenous people, the colonizers blamed the epidemic on the camelids, rather than accept the responsibility for having introduced the infection themselves.

Although modern medical authorities have discounted the legend of an animal source of syphilis, it was not until 1970 that experiments proved that alpacas were not susceptible to infection with *T. pallidum*.[162,163]

Nocardia asteroides has been isolated from two abscesses in llamas at the author's clinic and in a llama with granulomatous abscesses in the lung.[o]

ABSCESSES

Abscesses are local collections of purulent exudate buried in tissues, organs, or other confined spaces. They occur commonly in all animals including camelids. Abscesses may occur in any organ system but are most common in subcutaneous tissue, lymph nodes, and the parenchyma of the viscera. Infection may arise from breaks in the epidermis or from hematogenous sources.

Etiology

Many different bacterial species have been isolated from abscesses. *Arcanobacterium* (formerly *Corynebacterium*) *pyogenes* and *Corynebacterium pseudotuberculosis* are the most common isolates. Other species that may be isolated include *Staphylococcus aureus, Streptococcus* spp., *Rhodococcus equi, Pseudomonas aeruginosa, salmonella* spp., *Nocardia asteroides, Actinomyces* sp. *Actinobacillus* spp., *Corynebacterium ulcerans, Escherickia coli, Enterococcus* spp., *Proteus vulgaris, Fusobacterium necrophorum, Bacteroides* spp., *and Klebsiella* spp. Many of these organisms are opportunistic in that they may be normal inhabitants of the skin or mucous membranes, and only become pathogens when an animal is stressed, there is a break in the skin, there are combinations of organisms that enhance each other, or there is an overwhelming contamination.

Epidemiology

Abscesses occur worldwide. An abscess may be a pure culture of one of the species mentioned, but mixed infections occur. *Arcanobacterium pyogenes* infection in combination with *Fusobacterium* or *Bacteroides* is common.

Clinical Signs

Abscesses may be small (pustules, 1 mm) or large 12 to 15 cm, (5 to 6 in.), soft and fluctuant, firm, cold or hot to the touch, thin- or thick-walled or painful or not to palpation. The exudate may be thin, thick, caseated, colored (whitish, yellowish, greenish, reddish), or have an odor or no odor.

Diagnosis

Superficial abscesses may be observed or palpated. A large bore needle (12 or 16 gauge) may be used to aspirate exudate. Appropriate culture and sensitivity procedures should be performed. Sometimes the exudate is inspisated and may not be aspirated. Occasionally the exudate is sterile, the infection being in the wall of the abscess.

Internal abscesses may be difficult to detect. Ultrasound examination may help if this is available. Leukocyte counts may be elevated, but more commonly they are normal on well-localized abscesses.

Management

Most superficial abscesses are treated by surgical incision and lavage. It may be necessary to bring the abscess to maturity with a counter irritant such as hot compresses or ichthammol ointment. The use of antimicrobial therapy is debatable unless vital organs are nearby. Frequently antibiotic therapy will diminish abscess activity but when the antibiotic is discontinued the activity will resume. This is particularly true of thick-capsuled *Corynebacterium pseudotuberculosis* abscesses. The antibiotic simply doesn't reach the organisms. Sensitivity patterns should always be used to establish the appropriate antibiotic.

Corynebacterium pseudotuberculosis and *c. ulceran* abscesses should be handled with care.[1,8,25,26,27] The organism is contagious and steps should be taken to

isolate the animal and the exudate for appropriate disinfection. In a study involving sheep and goats, three methods were evaluated for treatment:

1. Typical lancing and lavage plus a single dose of procaine penicillin G (20,000 U/kg) SC.
2. Closed lavage and intralesional installation of tulathromycin (2.5 mg/kg) antibiotic.
3. Closed lavage and SC administration of tulathromycin (2.5 mg/kg) antibiotic.

Group 1 abscesses were lavaged with betadine solution after lancing, and lavage in groups 2 and 3 consisted of inserting a 16-gauge needle into the abscess and instilling normal saline to thin the exudate. Then the exudate was aspirated and additional saline instilled and aspirated to remove all the of exudate. The abscesses resolved within a month on both groups with tulathromycin therapy.[101]

Autogenous vaccines have been used to deal with extensive prevalence on a farm or ranch.

PREVENTIVE MEDICINE FOR CAMELIDS[165,166]

Preventive medicine is sometimes assumed to be little more than periodic vaccination or worming of animals. A preventive medicine program (PMP) encompasses all aspects of management that enhance the health and well-being of an animal. Surely, it can be as Spartan or as elaborate as the people who develop a plan want it to be. The topic is located in this chapter because immunoprophylaxis against infectious diseases is a major component of a PMP. Llama and alpaca owners/managers may not be familiar with livestock management programs; therefore, information on husbandry factors is included.

Components of a PMP

Management is the foundation upon which a program is built. A preventive medicine program consists of both husbandry and health-related factors. Husbandry factors include commitment, knowledge, adequate feeding, environmental control, provisions for water, a sound breeding program, record keeping, insurance, and preparation for emergencies. Health-related factors include sanitation, parasite control, vaccination, quarantine, disease diagnosis, special examinations, and performance of routine care.

Husbandry Factors

Commitment to a PMP. Owners/managers must be convinced of the desirability of developing a plan. Once the plan is in place, reminders of the dates for appropriate procedures may be given by the veterinarian.

Knowledge/Information. Considerable practical and scientific information is available about camelids specifically, and even more information that is directly applicable to camelids, or may be applicable with minor modification, is available from other livestock industries.

Team Approach. Camelid owners are encouraged to have a team approach to the development and implementation of a PMP. The team may consist of the owner/manager, veterinarian, nutritionist, geneticist, or others as deemed appropriate. Having more than one person providing input into a plan is a sound practice. Owners may not have an overview and may require assistance. The plan should be written down, and copies given to all concerned.

Sound Genetic Program. A veterinarian may have only casual interest in bloodlines and genetics, but none can deny that health has a genetic component over and above the production of congenital/hereditary birth defects. The camelid owner should be encouraged to be diligent in acquiring sound animals and/or to develop a breeding program that maximizes strengths of animals in possession and replaces animals that do not enhance the program.

Adequate Feeding and Nutrition. Camelids are capable of adapting to many different forages. It is not necessary to feed the same items that are fed in another part of the country, but it is necessary to adhere to sound feeding principles. Malnutrition is a major stressor in the life of any animal. Consideration must be given to the special needs of various age groups of animals and to special physiological periods such as growth, late pregnancy, lactation, and fiber production.

Potable Water Source. Water may be piped from culinary sources, private wells, streams, lakes, ponds, or other standing water. Alpacas play in water, but they also drink the same water. Marshes, bogs, or slow-moving water are prime habitats for snails (secondary hosts for flukes) and other parasites. In cold climates waterers must be heated. Equipment should be of the highest quality possible and should be correctly installed. Auxiliary power should be available.

Environmental Control. Shelter from cold in northern climates and provision for dealing with heat stress (water, shade) in hot climates or seasons are important. No region of the country is free from potential disaster situations (earthquakes, floods, tornadoes, hurricanes, blizzards). Fire (grass, forest, barn) is an ever-present hazard (see Chapter 25 for further discussion on disasters). Emergency procedures should be included in the plan.

Buildings should be constructed in locations that minimize destruction by hazards and have sufficient doors to allow escape for entrapped animals. Local building codes should be scrupulously followed lest insurance policies be voided. Great care should be given to electrical installation and maintenance. Wires may be torn loose in high winds and/or flooding, pre-

cipitating sparks and subsequent fire. A master power switch should be installed to enable the quick disruption of power during heavy storms.

Fencing must be adequate and appropriate for camelids. Camelid owners tend to overfence, but there are many different types and styles. Some are elaborate and expensive, designed to keep dogs and deer out. Others are as simple as a single strand of electrified, smooth wire. Generally, barbed wire is not recommended because there is a risk of lacerations and fiber being torn from the fleece.

When new fences are installed or renovated, the fencing should be attached to the posts on the side next to the camelids. This provides added strength when animals press against the fence to reach for food outside the enclosure or fight over a fence in the case of males. It is particularly important to use this method of attaching fencing in alleyways or other areas where animals are likely to run or jump while trying to elude capture, to prevent them from crashing against protruding posts.

Sanitation. Sound sanitation practices minimize exposure to disease by decreasing the numbers of microorganisms in the environment. Factors to consider include general cleanliness in handling feed and feeding containers, fly control, rodent and vermin control, and management of feces and urine. Some owners may not understand what is appropriate and inappropriate sanitation in a livestock situation.

Records. Memories of owners or veterinarians are not records. Records should be written or maintained on a computer. Enlightened long-term management depends upon knowing what has gone on before. Complete records (including timely updates) should be maintained on each animal.

Evaluation. Periodic consideration of a PMP is a must to update or modify it as situations change.

Medical Components[95]

Parasite Control. Owners/managers of camelids should understand that it is unlikely that parasites can be entirely eliminated from their animals. The key word is control. To control parasites it is necessary to know what parasites are present. Having such information, a protocol can be developed in accordance with a priority of importance, and a monitoring schedule set up to periodically assess the level of parasitism.

Biological control should be considered first, including disrupting the life cycle of a parasite, and may be simple, such as rotational grazing or routine fecal cleanup, or more complex, including therapy at specific seasons to decrease parasite levels in the dam prior to birthing. Camelids may be the secondary host to parasites of cats (toxoplasmosis) and white-tailed deer (meningeal worm). Parasite ova require a period of time to mature before they become infective when ingested by another animal. Some parasite ova persist in the environment for long periods of time, while others become noninfective if exposed to sunlight and cold.

Immunoprophylaxis (Vaccination). Current levels of livestock production in the United States and most countries of the world would be impossible to achieve without vaccination against catastrophic diseases. Hundreds of biologics (vaccines) are produced for livestock. Each must undergo testing by regulatory agencies for efficacy and safety. When used in species not listed on the label (extra-label), there is no assurance of efficacy or safety. This is particularly important to understand when using vaccines that contain living organisms. No vaccines have been approved for use in camelids in the United States.[12]

It is not possible to vaccinate against every conceivable disease agent that could infect a llama or alpaca. The animal's body could not cope with all the immunologic demands imposed upon it. Veterinarians must determine which diseases pose a risk to the camelid population in a given area and weigh the desirability of using extra-label products to reduce risks.[158]

Eight diseases are caused by bacteria in the genus *Clostridium*. All have been produced experimentally in camelids, but from a practical standpoint only three or four are important, and the risk of even these may be negligible in some regions. Administration of seven- and eight-way clostridial vaccines became the established norm as the llama/alpaca industry began to expand, but there are some veterinarians, including myself, who feel that use of such vaccination is not necessary and may be inappropriate. The camelid may not be able to respond properly to the multiple antigens contained in these vaccines.

The following vaccines are recommended as a basic foundation: C. *perfringens* type C and D toxoid and tetanus toxoid. In rabies-endemic areas, a killed rabies vaccine should be administered. Likewise, if leptospirosis is a problem in livestock species in an area, the appropriate serovar bacterin should be selected and administered. Malignant edema, caused by C. *septicum*, is usually the result of wound infection and has been implicated as a complication with rattlesnake bite in Colorado.

Numerous other vaccines have been administered to camelids in the United States. The Association of Small Ruminant Practitioners has listed eighteen different products that are used for extra-label purposes in llamas.[10] A word of warning: A modified live virus vaccine or live bacterin should be used with caution on any animal unless that species is listed on the label!

Llamas and alpacas have developed retinal degeneration (blindness) following infection by equine herpesvirus type 1. A killed virus vaccine (pneumabort-K) has been administered to camelids in regions sur-

rounding an outbreak. It is not appropriate to use the vaccine routinely.

Much has been written and said on when to vaccinate, but the fact is that no one knows the appropriate schedule because no studies have been conducted in which camelids have been vaccinated at specific ages and then challenged with the disease organism. This is what is required for obtaining government approval for the use of a vaccine in a given species. Veterinarians with livestock experience usually recommend a regimen similar to that followed with other species. Some veterinarians may begin a vaccination series at two to four weeks of age while others recommend beginning at five to six months of age. My own recommendation is to vaccinate crias at eight to twelve weeks of age, followed by a booster in three to four weeks. Begin antirabies vaccination at twelve to sixteen weeks. Adults should be vaccinated annually. Many veterinarians, including myself, recommend a booster four to six weeks prior to the anticipated birth of a cria to ensure maximum production of antibodies for the colostrum. Near-term mothers should be handled gently to avoid stress.

Quarantine. A valuable tool, quarantine is frequently neglected, creating an extremely weak link in the chain of preventive medicine for most camelid owners. Newly purchased animals are usually put in with a herd or are in intimate contact with other camelids on the farm immediately on arrival. Animals are taken to shows and returned to the herd with little regard for possible exposure to disease agents. Keeping new arrivals isolated from resident llamas or alpacas for a period of fourteen to thirty days is a good business practice.

Females may be accepted for breeding or boarding without the benefit of a screening health examination. No owner/manager should accept outside females for breeding or boarding unless the animal is accompanied by documents certifying that it has tested negative for those diseases listed in the regulations for entry into the state of origin. See the documents for such tests. The state of origin may refuse re-entry to an animal that subsequently tests positive. This should also be required for legal protection, because without such documentation someone may be able to claim that a disease was acquired on a client's premises.

Deal with Disease. Owners/managers should understand the importance of making valid attempts to diagnose any disease that appears in the herd. Any animal that dies should be necropsied. This is an absolute requirement for collection of insurance. Unfortunately, a precise diagnosis is not always forthcoming from a necropsy, but necropsy may be crucial for the protection of the rest of the herd and the acquisition of baseline information on causes of death in llamas and alpacas.

It is appropriate to mention the potential of bioterrorism in camelids in regard to dealing with diseases. Several potential infectious and toxic diseases of livestock and humans are listed in Table 7.7. These should also be considered in the differential diagnosis of sudden, unknown diseases of camelids.

Pre- or Postpurchase Examination. Preventive medicine begins with a sound, healthy animal. Without that, a preventive medical program is off to a rough start.

Protocols for Special Circumstances. Written protocols for events such as parturition, neonatal care, dealing with failure of passive transfer, prematurity, or evacuation in the event of a disaster are desirable.

Routine Procedures. Routine evaluation and care of teeth and toenails may extend the productive life of a camelid. It is appropriate to analyze blood samples from normal animals to serve as baseline information should disease develop later on.

GOVERNMENT REGULATIONS AND INFECTIOUS DISEASES

In the United States, the llama and alpaca communities are intimately involved in cooperating with governmental agencies in conducting research on diseases that are or have been perceived as involving camelids. Llamas and alpacas have not been responsible for transmission of any known infectious disease to domestic ruminants.

Llamas and alpacas are required to conform to interstate regulations in the same manner as for cattle or in some instances sheep and goats. A few states still classify camelids as exotic animals, under the jurisdiction of game and fish agencies. The camelid industries have produced documentation about why llamas and alpacas should be treated as a separate category, but changes in the regulations have been slow in coming.

Diseases that have caused concern in various governmental agencies include foot-and-mouth disease, bovine tuberculosis, bovine brucellosis, and Johne's disease. (See elsewhere in this chapter for details of these diseases and discussion regarding the lack of evidence that llamas and alpacas pose any threat of spreading the diseases to domestic ruminants.) Still, more than half of the states in the United States require negative tests for tuberculosis and brucellosis prior to interstate shipment. Nine or ten states also require negative tests for bluetongue virus and anaplasmosis.

Importation Requirements[13]

From Areas Having Endemic Foot-and-Mouth Disease or Rinderpest

Currently included areas are eastern Europe; Asia; Africa; and Peru, Bolivia, and Argentina in South America.

Table 7.7. Potential agents of bioterrorism against livestock, camelids, and people.

Agent	*Etiology*	*Clinical Signs*	*Pathogenicity for camelids*	*Diagnosis*	*Exposure*
Foot-and-mouth disease	*Aphthovirus* sp. (*Picornaviridae*)	Vesicles on muzzle, oral cavity, feet, teats, erosions	+/−	Gov. diagnostics, DNA tests, serology	Direct contact with infected animals
Rinderpest	Rinderpest virus (*Paramyxoviridae*)	Fever, leukopenia, erosions and ulcers of muzzle, oral cavity, and entire G.I. tract, diarrhea	+, Only camels reported	Gov. diagnostics, DNA tests, serology	Direct contact with infected animals
Yellow fever	*Flavavirus* sp. (*Flaviviridae*)	Hepatic insufficiency, icterus	None	Gov. diagnostics, DNA tests, serology	Mosquito transmission
Hemorrhagic fever	Ebola virus (*Filoviridae*)	Extensive hemorrhages throughout the body, death	None	Gov. diagnostics, DNA tests, serology	Direct contact, insect transmission
Influenza	Influenza virus H1N1 and other strains (*Orthomyxoviridae*)	Fever, weakness, dyspnea, coughing, pneumonia, myalgia	+, Only Bactrian camels reported	ELISA, virus isolation	Inhalation
Plague	*Yersinia pestis*	Fever, dyspnea, diarrhea, buboes, meningitis	+++, Both Bactrian and dromedaries	Culture	Fleas, ticks, cats are susceptible and cat fleas may transmit, direct contact
Botulism	*Clostridium botulinum* toxin	Muscle paralysis, suffocation	++	Animal inoculation with ingesta filtrate	Ingestion
Anthrax	*Bacillus anthracis*	Septicemia, hemorrhages from body orifices	++	Culture of bacteria	Direct contact with infected animals
Ricin poisoning	*Ricinus communis* toxin	Diarrhea, fever, leukopenia, pronounced heartbeat, colic, weakness, convulsions, shock	++++	Fluid-filled intestines, hemorrhages, isolation of toxin	Inhalation, water
1080 poisoning	Sodium monofluroacetate	Vomiting, muscle twitching, convulsions, tachycardia, nystagmus, death	++++	Peracute death, analysis	Inhalation, ingestion
Cyanide poisoning	HCN	Muscle twitching, dyspnea, convulsions, death	+++	Bright red blood	Inhalation, ingestion

+ = Minimal susceptibility, ++ = moderate susceptibility, +++ = high susceptibility, ++++ = extreme susceptibility.

1. Camelids may enter the United States only following a ninety-day quarantine at the USDA's maximum security facility (Harry S Truman Animal Import Center [HSTAIC]) near Key West, Florida.
2. Camelids are subject to a pre-embarkation quarantine of sixty days within the country of origin under the direct supervision of USDA veterinarians. During the pre-embarkation quarantine, all animals are subject to a battery of tests conducted by or under the supervision of USDA. Camelids are tested for the following diseases:
   a. Tuberculosis: Intradermal tuberculin test using bovine PPD antigen (0.1 ml) in the axillary space.
   b. Bovine brucellosis: Tube-agglutination test, negative in a 1:25 dilution.
   c. Foot-and-mouth disease: Virus neutralization, virus infection associated antigen (VIAA), and probang test (virus isolation in tissue culture of any animal found positive on either of the first tests).
   d. Vesicular stomatitis: Virus neutralization for five serotypes.
   e. Trypanosoma vivax: Indirect immunofluorescent antibody test.
   f. Bluetongue: Agar gel immunodiffusion serological test.

   Although not tested for, there must be no evidence of trichomoniasis, Vibrio fetus, leptospirosis, or Johne's disease in the herd(s) of origin in the past five years.
3. When released from pre-embarkation quarantine, the animals must be transported directly by air or ship to HSTAIC to complete the ninety-day quarantine there.
4. During HSTAIC quarantine, the animals are subject to additional testing and are housed with sentinel cattle and swine.
5. The HSTAIC quarantine facility is used for importation of ruminants, swine, and camelids. Requests for importation permits always exceed the space available. Selection of permittees is done on a lottery basis. Approximately 400 animals may be accommodated per quarantine, and only three periods are available per year. All expenses for maintaining the pre-embarkation and HSTAIC quarantine are borne by the importer.
6. The USDA inspects animals for external parasites and treats them appropriately, usually with ivermectin, but does not have a program for controlling gastrointestinal parasites. Ivermectin should not be administered less than fourteen days prior to collection of blood for serologic testing.

From Non-Foot-and-Mouth Disease and Nonrinderpest Areas

Procedures are as follows:

1. Permits must be obtained from the USDA and require an official health certificate supplied by a veterinarian employed full-time by the government veterinary service agency of the exporting country. In the case of Chile, this is Servicio Agricola Ganadero (SAG) (Livestock Agricultural Service).
2. The Chilean government must certify that the country is free of FMD, that the animals have not been vaccinated for FMD, and that the sire and dam of the intended export animals were born and raised in Chile. It is also stated in the regulations that there must be no Johne's disease on the premises for the two previous years.
3. A sixty-day quarantine is required in a facility supervised by a SAG veterinarian and approved by the USDA.
4. During the pre-embarkation quarantine, tests are conducted for the same diseases as listed for maximum security quarantine. The tests may be conducted in the country of origin, but this must be approved by the USDA.
5. Animals are sent to the Animal Import Center (NYAIC), Stewart Airport, Newburgh, New York. This quarantine lasts for a minimum of thirty days, during which additional tests may be required.

VALIDITY OF LABORATORY TESTING FOR INFECTIOUS DISEASES

The management and regulation of infectious diseases in livestock, horses, and companion animals depends on the use of data from serologic and other laboratory diagnostic procedures. Many of these tests are important to diagnose disease and determine antemortem exposure to infectious agents in camelids. Transposition of diagnostic tests to species other than the species for which they have been validated may be unwise because the assumption that a serological test will perform identically in camelids as in livestock may be incorrect.[67] There may be differences in pathogenic strains, serovars, host responses, and exposure to organisms of similar antigenic structure that produce cross-reacting antibodies. Furthermore, some assays require species-specific reagents/test components that might not be commercially available, and most assays have not been standardized. Many of the diagnostic tests used in livestock have, by tradition, been accepted as valid, but in reality most have not been adequately validated.[53]

The validity of a test involves establishing the sensitivity and specificity of the test for a given species of animal. Sensitivity is the probability that a test will correctly identify animals that have been exposed to a given pathogen (true positive). A test with a high sensitivity provides maximum assurance that the animal has been exposed to the organism, but false negatives may occur when (1) a recently infected animal has a low concentration of antibodies, (2) an animal is immunologically tolerant to infection, (3) the cutoff for the test was set too high, or (4) the test was not correctly performed.

Specificity is the probability that a test will correctly identify animals that have not been exposed to a given pathogen (true negative). False-positive reactions may occur under any of the following conditions: (1) the presence of specific and nonspecific antibodies following vaccination, (2) errors in handling and testing in the laboratory, (3) excessively low assay cutoff values, and (4) cross-reactions with antibodies produced by antigenically related organisms.[53]

Of particular concern to camelid veterinarians are the testing procedures imposed on camelids moving internationally or intrastate. Each state is empowered to determine the tests that are required. The majority of states require negative tests for tuberculosis and brucellosis (*Brucella abortus*). Less than a dozen test for bluetongue and anaplasmosis. International requirements vary widely.

A study on the application of brucellosis serologic tests to llamas, conducted by the USDA in cooperation with the International Llama Association, revealed that the standard screening tests that do not require species-specific reagents are valid for testing llamas (standard plate test, card test, Rivanol test, and BAPA).

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[a] Dr. Robert Pollard, personal communication.
[b] Juan Lubroth, personal communication.
[c] Juan Lubroth, personal communication.
[d] Eric Sharpnack, personal communication.
[e] Orthocide Ortho-Chevron, San Francisco, CA 94120-7144.

[f] Tresaderm (15 ml plastic bottle: thiabendazole 40 mg/ml, dexa methasone 1 mg/L, and neomycin 3.2 mg/ml), Merck, Sharp, and Dohm, Rahway, NJ, 07065.

[g] Unpublished laboratory report, California Livestock and Poultry Pathology Lab., San Gabriel, Calif., 1952.[97]

[h] I. Ingram, Scottsdale, Ariz., personal communication.

[i] I. Ingram, Scottsdale, Ariz., personal communication.

[j] J. Paul Murphy, Davis, Calif., personal communication.

[k] W. Bravo, personal communication.

[l] Anthrax spore bacterin, Colorado Serum Co., Denver, Colo.; Thraxol-2, Bayvet Div., Cutter Labs., Shawnee Mission, Kans.

[m] P. Long, Corvallis, Oreg., personal communication.

[n] Dr. Jeffrey Barlough, Davis, Calif., personal communication, Sept. 1996.

[o] C.-O. Chang, Auburn, Ala., personal communication.

8

Parasites

This chapter is not meant to be a definitive discussion of all the ramifications of the parasites of camelids; however, sufficient information is discussed to allow general identification, provide the life cycle if known, and aid in the management of parasitism in OWCs and SACs. Parasitism is a limiting factor in the production of fiber and meat in South America, and losses reach more than $1.5 million annually in Peru.[110,111]

The classification of parasites is in constant flux. Therefore, a recent reliable author should be selected as a source for taxonomy. In this book, the classifications of Levine for nematodes[67] and protozoa[68] are followed. For other parasites, Soulsby[100] and Bowman[9] are the sources. Taxonomy is not of great importance to the clinician except to know that closely related species tend to share similar life cycles, which aids in more effective management planning. For instance, if a particular parasite is known to be a trichostrongylid, plans to control the parasitism can be based on knowledge of the probable life cycle and susceptibility to certain anthelmintics.

A classification of parasitic genera reported to affect NWC and OWC follows.[9,28,35,67,68,100]

- Phylum Arthropoda
  - Class Insecta
    - Order Mallophaga—biting lice
      - *Damalinia (Bovicola) breviceps*
    - Order Siphunculata [Anoplura]—sucking lice
      - Family Linognathidae
        - *Microthoracius cameli*
        - *M. praelongiceps*
        - *M. mazzai*
    - Order Siphonaptera—fleas
      - *Vermipsylla* spp.
    - Order Diptera—flies
      - Suborder Nematocera
        - Family Culicidae—mosquitoes, miscellaneous genera
        - Family Simuliidae—black fly
      - Suborder Brachycera
        - Family Tabanidae
          - *Tabanus* spp.—horse fly, deer fly
      - Suborder Cyclorrhapha
        - Family Muscidae
          - *Musca domestica*—house fly
          - *Musca autumnalis*—face fly
          - *Stomoxys calcitrans*—biting stable fly
        - Family Calliphoridae—blow flies
          - *Calliphora* spp.—blue blow fly
          - *Cochliomyia hominivorax*—primary screw worm
          - *Phaenicia* spp.—green blow fly
          - *Phormia* spp.—black blow fly
        - Family Oestridae—bot flies
          - *Oestrus ovis*—sheep nasal bot
          - *Cephenemyia* spp.—deer nasal bot
          - *Cephalopsis titillator*
  - Class Arachnida
    - Order Acarina
      - Suborder Metastigmata—ticks
        - Family Argasidae—soft-bodied ticks
          - *Otobius megnini*—spinose ear tick
        - Family Ixodidae—hard-bodied ticks, various genera
      - Suborder Mesostigmata—mites
        - Family Sarcoptidae
          - *Sarcoptes scabiei*
        - Family Psoroptidae
          - *Psoroptes ovis*
          - *Chorioptes bovis*

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Phylum Protozoa
Subphylum Sarcomastigophora—flagellates
Trypanosoma spp.
Tritrichomonas spp.
Giardia spp. (OWC, NWC)
Subphylum Apicomplexa (Sporozoa)
Eimeria lamae (NWC)
E. alpacae (NWC)
E. punoensis (NWC)
E. macusaniensis (NWC)
E. peruviana (NWC)
E. aburnensis
Cryptosporidium parvum
Sarcocystis aucheniae
S. tilopoidi
Toxoplasma gondii (OWC, NWC)

Phylum Platyhelminthes (Flat Worms)
Class Trematoda—flukes
Order Digenea
Fasciola hepatica (OWC, NWC)
F. gigantica (OWC)
Fascioloides magna (NWC)
Eurytrema pancreaticum (OWC)
Dicrocoelium dendriticum (OWC, NWC)
Class Eucestoda—tapeworms
Family Taeniidae
Echinococcus granulosus (OWC, NWC)
Family Anoplocephalidae
Moniezia expansa (OWC, NWC)
Thysaniezia spp. (NWC)

Phylum Nemathelminthes
Class Secernentea (Phasmida)
Order Strongylida
Superfamily Trichostrongyloidea
Trichostrongylus axei (OWC, NWC)
T*richostrongylus* spp.
Ostertagia ostertagi (OWC, NWC)
Ostertagia spp.
Marshallagia (Ostertagia) marshalli (OWC, NWC)
Camelostrongylus mentulatus (OWC, NWC)
Graphinema aucheniae (NWC)
Haemonchus contortus (OWC, NWC)
Haemonchus spp. (OWC, NWC)
Lamanema chavezi (NWC)
Spiculopteragia peruvianus (NWC)
Nematodirus lamae (NWC)
N. battus (OWC, NWC)
Cooperia spp. (OWC, NWC)
Teladorsagia spp.
Dictyocaulus viviparus (NWC)
Dictyocaulus filaria (OWC, NWC)
Superfamily Metastrongyloidea
Family Protostrongylidae
Parelaphostrongylus tenuis (NWC)
Angiostrongylus cantonensis (NWC)
Superfamily Strongyloidea
Family Strongyloidea
Oesophagostomum spp. (OWC, NWC)
Chabertia ovina (OWC, NWC)
Superfamily Ancylostomatoidea—hookworms
Family Ancylostomatidae
Bunostomum spp. (OWC, NWC)
Order Spirurida
Superfamily Thelazoidea
Family Thelaziidae
Thelazia californiensis (NWC)
Thelazia spp. (OWC, NWC)
Superfamily Spiruroidea
Gongylonema spp. (OWC, NWC)
Class Adenophorea (Aphasmidia)
Order Enoplida
Superfamily Trichuroidea
Trichuris tenuis
Capillaria spp.

Following is a list of the parasites of OWC not listed in the previous list. For a description of life cycle, epidemiology, clinical signs, diagnosis, and management, refer to related species under SAC descriptions or Bornstein.[8]

Phylum Arthropoda
Class Insecta
Order Diptera
Family Sarcophagidae—flesh flies
Wohlartia magnifica, W. nuba
Sarcophaga dux
Family Calliphoridae—blow flies
Lucia cuprina
Chrysoma bezziana
Family Oestridae—bot fly
Cephalopina titillator
Family Glossinidae—tsetse fly
Glossina spp.
Family Tabanidae—horse flies
Haemalopota spp.
Chrysops spp.

Phylum Pentastomida
Linguatula serrata
Class Arachnida
Family Ixodidae—hard-bodied ticks
Hyaloma spp.
Amplyoma spp.
Boophilis decoloratus
Rhipicephales spp.
Family Argasidae
Ornithodorus spp.

Phylum Saarcomastigophora (Flagillates)
- Order Kinetoplastida
  - *Trypanosoma evansi*
  - *T. simiae*
  - *T. Brucei*
  - *T. Congolense*
  - *T. Vivax*
- Order Trichomnadida
  - *Tritrichomonas foetus*

Phylum Apicomplexa
Class Sporozoa
- Family Eimeria
  - *Eimeria bactriani*
  - *E. cameli*
  - *E. Dromedarii*
  - *E. Pellerdyi*
  - *E. rajasthani*
- Family Cryptosporidiidae
  - *Cryptosporidium* spp.
- Family Sarcocystidae
  - *Sarcocystis cameli*
- Family Toxoplasmatidae
  - *Besnotia* spp.
  - *Isospora cameli, I. orlovi*
  - *Neospora caninum*
  - *Hammondia heydorni*

Subclass Piroplasmia
- *Babesia* spp.
- *Theileri* spp.

Phylum Ciliophora
- Order Trichostomatida
  - *Balantidium coli*

Phylum Nemathelmintha
Class Nematoda
- Order Strongylida
  - Family Strongylidae
    - *Trichostrongylus probolurus*
    - *T. vitrinus*
    - *T. falculatus*
    - *T. affinus*
    - Cooperia onchophora
    - C. pectinata
    - C. surnabada
    - *Impalaia tubeculata*
    - *I. nadeaollis*
    - *I.aegyptiaca*
  - Family Molineidae
    - *Nematodirus spathiger*
    - *N. mauritianicus*
    - *N. abnormalis*
    - *N. Dromedarii*
    - *N. helvetianus*
    - *Nematodirella dromedarii*
    - *N. Cameli*
  - Family Chaberiidae
    - *Oesophagostomum columbianum*
    - *O. Venulosum*
    - *O. Vigintimembrum*
    - *Chabertia ovina*
  - Family Ancylostomatidae—hookworms
    - *Bunostomum trigonocephalum*
  - Family Trichuridae
    - *Trichuris ovis*
    - *T. globolus*
    - *T. affinis*
    - *T. raoi*
    - *T. skrjabini*
    - *T. cameli*
- Order Spirurida
  - Family Gongylonematidae
    - *Gongylonema pulchrum*
    - *G. verracusum*
  - Family Habronematidae
    - *Parabronema skrjabini*
  - Family Thelaziidae
    - *Thelazia leesei*
    - *T. rhodesi*
- Order Filarida
  - Family Onchocercidae
    - *Dipelatonema evansii*
    - *Onchocerca armillata*
    - *O. Fasciata*
    - *O. gulurosa*
- Order Rhabtitida
  - Family Strongyloididae
    - *Strongyloides papillosus*

Phylum Platyhelmintha
Class Cestoda
- Family Taeiniidae
  - *Taenia multiceps*
  - *T. hydatigena*
  - *T. Hyaenae*
- Family Anaplocephalidae
  - *Monezia benedeni*
- Family Avittellinidae
  - *Stilesia centripunctata*
  - *S. globipuctata*
  - *S. vittata*
  - *Avitellina woodlandi*
  - *Thezaniezia ovilla*

Class Digenea
- Family Fasciolidae
  - *Fascioloides magna*
- Family Paramphistomatidae
  - *Paramphistomum* spp.
- Family Schistostomatidae
  - *Schistosoma bovis*
  - *S. mattheei*

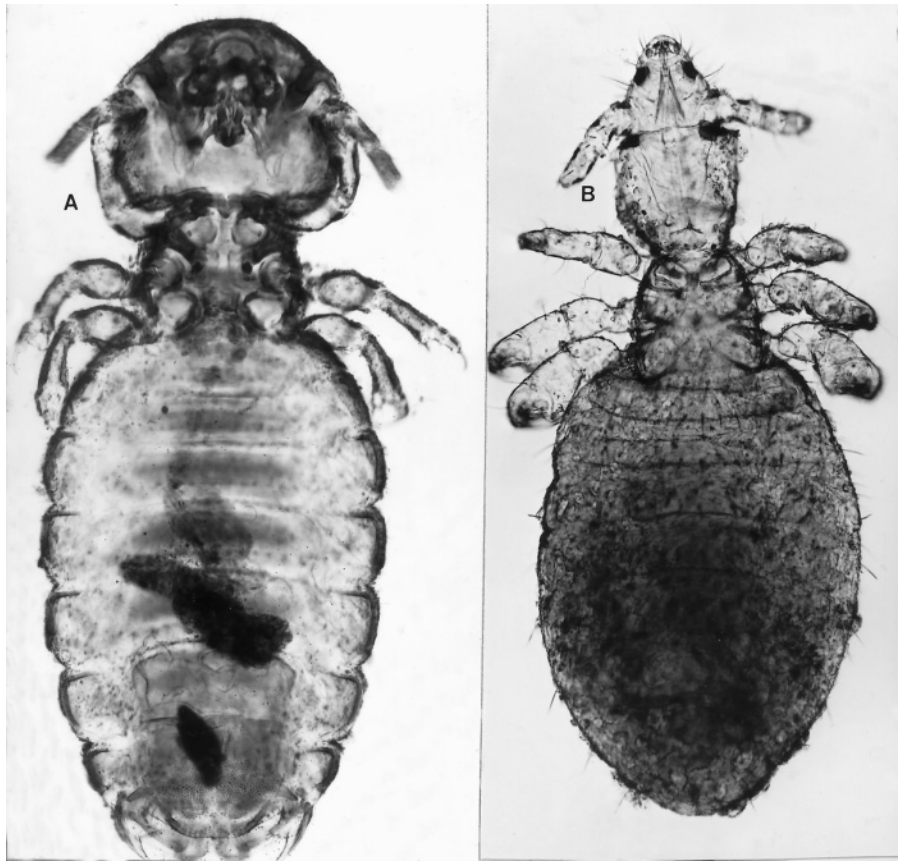

Figure 8.1. Camelid lice. (A) Biting louse (*Damalinia breviceps*), (B) sucking louse (*Microthoracius cameli*).

Only a few gastrointestinal (GI) parasites of SACs have been identified for species in North America. Thus, it is not known whether the species found in SACs are the same as those found in cattle, sheep, and goats, or if they are different species from the same genera.[31] Though important from an epidemiologic standpoint, information will not be available until classic taxonomists of parasites take an interest in SACs. Fortunately, that is now occurring.

The parasites of herd or flock importance in South America have been studied intensively. Those of lesser importance are rarely investigated. The lack of reported instances of parasitism from major taxonomic groups may only reflect a failure to observe, identify, or report. As yet, only seventy-five species of parasites have been reported to cause clinical disease in camelids.[16,24,57,69,77,80,82,87,92] SACs share no nematode genera with equids, and there are no reports of ascarids in SACs.

EXTERNAL PARASITES[70,71]

Lice[17,106]

Identification

Lice are wingless insects that live a complete life cycle on a single host.[100] They are generally host specific; i.e., lice found on livestock and pets do not spread to camelids nor will camelid lice transfer to other species.[107] Both biting (*Damalinia breviceps*) and sucking (*Microthoracius minor*) lice and *M. mazzai, M. praelongiceps, and M. cameli* (Figure 8.1) have been identified on camelids.[9,12]

Biting lice vary in size from 0.5 × 1.2 to 1.5 × 4 mm. They may be white or light tan. People with average vision can see these lice without magnification, but a hand lens (10×) assists the observer. Unless recently groomed, a camelid fleece contains detritus, which may be confused with lice. Lice move when disrupted by parting of the fiber and are found near the surface of the skin. Biting lice have a blunted head. This characteristic can usually be seen without magnification, but precise identification requires microscopic evaluation.

Sucking lice of SACs are approximately two-thirds the size of biting lice. Sucking lice are more sedentary than biting lice and thus are more difficult to see. They have elongated mouth parts, but these are so small that microscopic identification is necessary.

Life Cycle

The life cycle of lice is simple (Figure 8.2). Adult lice copulate, and the female deposits fertilized eggs (nits)

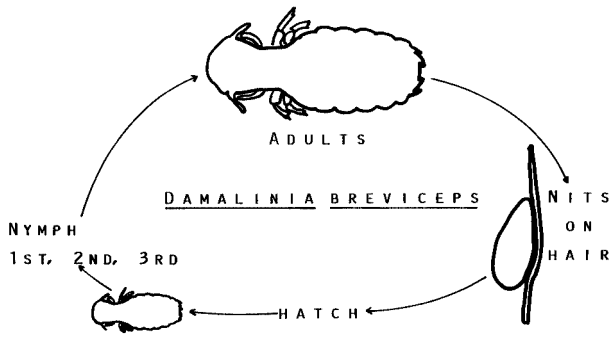

Figure 8.2. Life cycle of a louse.

on fibers and cements them in place. The eggs hatch within one to three weeks, and tiny replicas of adult lice emerge. As the louse matures, it undergoes two or three molts, but in each case it is growth, not a change of body structure. The maturation phase lasts one to two weeks. The entire life cycle may be completed in as little as two to five weeks. Adults live for fifteen to forty days.

Epidemiology

Lice may complete the life cycle on a single animal. There is no free-living stage. Transmission from one camelid to another is by close body contact, as may occur with maternal care of infants, during breeding, or when individuals lie down touching each other. Transmission can also occur by the use of communal grooming equipment such as combs, brushes, and carding combs or by group use of blankets, scratching posts, dust bath areas, narrow doorjambs, and feed bunks.

Populations of lice tend to build up during the colder months of the year. Although the cycle may be completed in as little as two to five weeks, it may be extended many months by arrested development in the nit stage, or a few lice may lie dormant until environmental conditions become conducive to rapid development of a population.

Clinical Signs

The syndromes of the two types of lice are different.

BITING LICE. The fiber coat lacks luster and has a ragged appearance. Heavy infestation results in matting and loss of fibers. The llama may bite at itself or rub against fences, trees, shrubs, poles, or buildings. In one case, the animal rubbed raw a spot over the dorsal spine of the vertebra.

The areas of the body most likely to be infested are around the base of the tail, along either side of the vertebral column, on the side of the neck, and along the sides of the body. To search for lice, part the fibers down to the skin, use a bright light, and observe for movement of the tiny specks. If no lice are seen, change locations and repeat the procedure. The egg cases (nits) may be observed attached to the fibers.

SUCKING LICE. Of the two types of lice, sucking lice are more pathogenic. In addition to the signs noted for biting lice, sucking lice may cause anemia because they extract blood and tissue fluid from the host. Anemic llamas are more susceptible to cold stress and secondary infections.

Sucking lice prefer sites around the flanks, head, neck, and withers. Lice may be found clinging to the fibers close to the skin or actually embedded in the skin taking a blood meal.

Treatment

Lice may be destroyed by direct contact with numerous insecticides, including pyrethrins, chlorinated hydrocarbons, carbamates, and organic phosphates. The problem is establishing contact with the lice. Dusts and sprays applied superficially do not reach them. Drenching sprays or dips are not practical for temperate climates, especially because the problem is more pronounced in the winter.

Ivermectin is not effective against biting lice but has excellent activity against sucking lice at a dose of 0.2 mg/kg body weight administered subcutaneously. It should be noted that although ivermectin has not been approved by the Federal Drug Administration (FDA) for use in llamas, it has been used extensively as a general anthelmintic in llamas and has proven to be safe and effective. This should be discussed with a client before this drug is administered.

The author has had poor results using "pour-on" organic phosphate insecticides for biting lice, but others have recommended its use. One method of applying a pour-on insecticide to the skin is to attach an artificial insemination pipette with rubber tubing to a syringe. The pipette is inserted through the fiber coat over the tail head, then pushed forward against the skin as far as possible. The pipette is withdrawn while ejecting the selected dose from the syringe.[a]

In the author's experience, an effective product is a 50% wettable powder of methoxychlor (Marlate, obtainable at orchard and garden supply companies). The powder can be dispensed from a large shaker-top container like those used for salt in food establishments. The fibers should be parted down the topline and the powder shaken next to the skin. Manipulating the wool aids in distributing the powder. More powder should be applied 5 to 7.6 cm (2 to 3 in.) to the side with fibers parted as before. This process should be continued until all of the problem areas have been covered. It is a laborious process but effective during cold winter months. Carbaryl powder is also effective but is likely to be more expensive than methoxychlor.

Fleas

Identification

Fleas are wingless insects with laterally compressed bodies.[100] They vary in size from 1.5 to 4 mm. Fleas are not host specific; thus, several different types may be found on camelids. No specific reports of flea infestations have appeared in the literature on SACs, but the genus Vermipsylla contains a number of species that infest camels. Llama owners have described flea infestation to the author.

Life Cycle

Adult fleas copulate, and the female lays approximately twenty eggs at a time in detritus on the host or in dust/dirt in the llama's environment. Larvae hatch in two to sixteen days and feed on dried blood, feces, or other organic matter. The larval stage is completed in seven to ten days, and the insect enters the pupal stage, which may last for ten to seventeen days or remain dormant for months. The adult emerges from the pupal case to seek a suitable host.

Epidemiology

Fleas may be transmitted from one llama to another by close body contact. Fleas are active and may jump prodigious distances. Contamination of the environment with flea eggs and pupae enables infestation to be acquired by simply walking through any area previously exposed to animals with fleas. The life span of an individual flea may be more than one year. During this time the female may lay as many as 400 eggs.

Fleas are notorious vectors for infectious and parasitic diseases; however, no such instances have been documented in SACs.

Clinical Signs

Adult fleas consume only blood and may cause anemia. In massive infestations, exsanguination from fleas may be fatal. While biting the host, fleas deposit saliva, which may stimulate an allergic response varying from mild irritation to marked pruritus, hyperemia, swelling, and dermatitis.

Treatment

Same as for lice.

Mosquitoes

Identification

Mosquitoes are slender-bodied, long-legged, winged insects with spherical heads and a long slender proboscis, which is used to cannulate capillaries to ingest blood meals from the host.[112] The numerous species differ in habitat preference but have no significant host specificity. Camelids in mosquito territory will be bitten.

Life Cycle[112]

The gravid female lays eggs in water. The eggs hatch in less than a week to become air-breathing larvae. Larvae undergo four molts over a period of two weeks, progressing to the pupal stage that lasts two to seven days. The adult emerges from the puparium and must dry itself before being able to fly off within twenty-four hours. The entire cycle requires approximately a month, less in warm, moist habitats.

Epidemiology

Because mosquitoes fly, they can easily reach hosts. They do not remain on the host but, rather, light, ingest a blood meal, and leave. A swarm of mosquitoes can be annoying and may stimulate a bite response similar to that of fleas. Like fleas, mosquitoes are vectors for numerous infectious diseases, serving as the intermediate host for some and as mechanical vectors for others.

Clinical Signs

Mosquito bites, even in large numbers, will not likely cause anemia. Individual bites may be pruritic, hyperemic, and swollen.

Treatment

Reducing mosquito numbers requires management of the environment (decreasing breeding habitat, insecticide treatment). Mosquito repellents used by humans are effective for a short period and are recommended for those who pack with llamas.

Black Flies (Buffalo Gnats)

Identification

Simulid flies are closely related to mosquitoes but have a short, piercing proboscis and a hump over the thorax. The legs are not as long as those of mosquitoes.

Life Cycle[100]

The female black fly lays eggs only in running water, either on the surface or on submerged stones, twigs, or vegetation. The eggs may remain in the water for months, overwintering in this state, or hatch in a few days. Larvae are mobile, with the lopping type of ambulation typical of an inchworm. Larvae molt six times before entering the pupal stage, which floats near the surface so that a special respiratory tube can take in the necessary air. Adults emerge from the puparium and may swarm in dense clouds if the population is large.

Epidemiology

Black flies are found throughout the world but are concentrated in warmer climates. Flies are active in the morning and evening. They do not live on the hosts but take blood meals from them.

Clinical Signs

Swarms of black flies are annoying to the animals and may inhibit feeding. In sensitive animals, the bites may simulate those from mosquitoes.

Treatment

Same as for mosquitoes.

Tabanids (Horse Flies, Deer Flies)

Identification

Tabanids are medium to large flies with powerful wings and large eyes.

Life Cycle[100]

Females lay eggs in damp soil or decaying organic matter. The eggs are glued in masses near a water source so that larvae, hatching in about one week, will fall into the water to continue maturation. Larvae and subsequent pupae may burrow into the mud at the bottom of a pond or stream and overwinter there. Adults emerge from the puparium and require a blood meal soon to complete the life cycle, which requires a minimum of four months or may extend to the next season.

Epidemiology

Tabanids are diurnal and are especially active on hot, bright, humid days. Only the females take blood meals every three or four days. The bite from a tabanid is painful. Tabanids may be responsible for transmission of parasites and infectious agents, but this has not been documented for SACs. Tabanids are an important intermediate host for *Trypanosoma evansi*, the cause of surra in camels and other artiodactylids in Africa and Asia. There is circumstantial evidence of *T. evansi* infection in South America, with a potential for infection in SACs.

Clinical Signs

The large mouth parts may pierce capillaries, which continue to flow after the tabanid has departed, attracting other non-biting flies. Sensitive individuals may develop erythema and swelling at the bite site. The flies may annoy the animal.

Treatment

No treatment or prevention is available other than swatting the fly if observed near or on the animal.

Miscellaneous Flies

Identification

Llamas and other camelids are plagued with the same types of flies that afflict domestic livestock. The house fly (*Musca domestica*) has four dark stripes on the thorax and yellow spots on the side of the abdomen and is approximately 7 mm long.[80] The face fly (*M. autumnalis*) is slightly larger than the house fly and lacks the yellow color on the abdomen. The biting stable fly (*Stomoxys calcitrans*) is also annoying to llamas. It is the same size as the house fly but has a long, stiff proboscis and gray-brown spots on the abdomen. Other species of flies may annoy or afflict llamas. The species that are present depend on the environment.

Life Cycle[100]

House Fly. The female lays eggs on manure or decaying organic matter. The larvae hatch in less than twenty-four hours, then grow and molt twice in a few days to become third-stage larvae, which move to a drier area and pupate. Adults emerge in two to three weeks, climb to the surface, and spread their wings to dry. Adults live for six to eight weeks, during which time a female will lay 2,000 eggs. The entire cycle takes three to five weeks.

Face Fly. The life cycle is similar to that of the house fly (Figure 8.3), except that the female lays eggs in fresh cattle feces, where hatching, larvation, and pupation occur. Hence, face flies are a problem only where camelids are housed contiguously to cattle.

Biting Stable Fly. The life cycle is similar to those of house and face flies. Eggs are laid in decaying organic matter or manure. Both sexes suck blood and feed once or twice daily.

Epidemiology

House flies are found both inside and outside of barns and sheds. Face flies usually do not enter buildings, so llamas may gain respite by going inside;

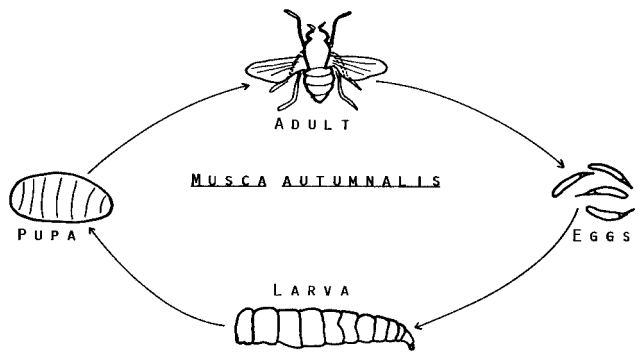

Figure 8.3. Life cycle of a fly.

however, face flies may enter buildings to escape cold weather. They may overwinter as adults, becoming active on days of unseasonably warm weather. All flies are more active on warm days than on cool days and cease activity at dark.

Clinical Signs

Flies are annoying to camelids. Biting flies may cause additional irritation. Flies are particularly irritating if there is excessive lacrimal secretion. They may exacerbate the problem until conjunctivitis is produced.

Management

Fly control is a never-ending challenge to managers of any animal enterprise. All the methods employed by those who raise livestock have application with camelid enterprises. A visor of cloth or leather strips, similar to those used on horses, may be employed to keep flies from the eyes.

Blow Flies

Identification

Many species of flies deposit eggs in fresh or necrotic wounds of animals. Camelids are equally susceptible to the attacks of these flies. Some examples of blow flies follow.

The blue blow fly (*Calliphora* spp.) may be as long as 11 mm. The thorax is dull colored and the abdomen is blue-green. The green blow fly (*Phaenicia* spp.) is approximately 7 mm long, with green to bronze coloration of the thorax and abdomen. The black blow fly (*Phormia* spp.) is approximately 8 mm long and is bluish-black. The primary screw worm (*Cochliomyia hominivorax*) is a special problem because it invades healthy tissue.

Life Cycle

The female fly of the primary screw worm lays eggs (200) in a fresh wound. The eggs hatch in twenty-four hours and the larvae feed on flesh, invading contiguous tissue. Mature larvae leave the wound in seven days to pupate. Adults emerge in one to several weeks. The entire life cycle may be completed in as little as two and a half weeks or continue for several. The primary screw worm could be considered an obligatory parasite.

The facultative parasitic calliphorids have a similar life cycle but do not invade fresh wounds. They are attracted to suppurating or necrotic wounds or areas soiled with feces and urine. The neonatal umbilicus is a favored site. Downer animals may develop decubitus on soiled areas and be subject to attacks.

Epidemiology

Primary screw worms have been eradicated from the United States but may be present in other areas of the New World. The other flies are ubiquitous.

Clinical Signs

Larvae are seen crawling and feeding in the wound.

Treatment

The wound should be cleaned and debrided to remove necrotic tissue. Fiber should be clipped to allow proper drainage of the wound and exposure to the air. Larvae may be removed mechanically or killed by the instillation of insecticides into the wound. The vehicle used to solubilize the insecticide may be irritating to the tissue and will be absorbed into the system much more readily than through healthy skin, so only as much fluid as is absolutely necessary to destroy the larvae should be applied. Chloroform, ether, and hydrogen peroxide also cause the larvae to retreat from crevices and cavities. Once larvae are destroyed, the wound should be properly treated and dressed. The wound should be monitored to remove missed larvae or deal with reinvasion. Ivermectin is also effective.

Bot Flies

Identification

Only three species of bot flies have been reported from camelids: the sheep and goat nasal bot (*Oestrus ovis*), various species of the deer nasopharyngeal bot fly (*Cephenemyia spp.*), and the camel bot (*Cephalopina titillator*). The first two species are important for SAC owners and are discussed separately.

Oestrus Ovis

IDENTIFICATION. The adult fly averages 12 mm in length, about the size of a small honeybee. Soulsby describes the fly as having a dark gray color with tiny black spots that are especially prominent on the thorax.[100] First instar larvae are whitish, translucent, and less than 2 mm long. As the larvae mature, dark transverse bands develop on the dorsal aspect of the segments. Mature larvae are approximately 3 cm long, with a long, tapering anterior end and a flat posterior end. There are two black stigmal plates on the posterior surface (Figure 8.4), black oral hooks on the anterior end, and rows of small spines on the dorsal surface.

LIFE CYCLE. The fly deposits larvae around the nostrils of the camelid. Flies cause considerable annoyance to the animal, and it may take evasive action by pressing the muzzle close to the ground or against other animals. The larvae instinctively crawl upward into the nasal cavity. Larvae may stay in the turbinate area or crawl

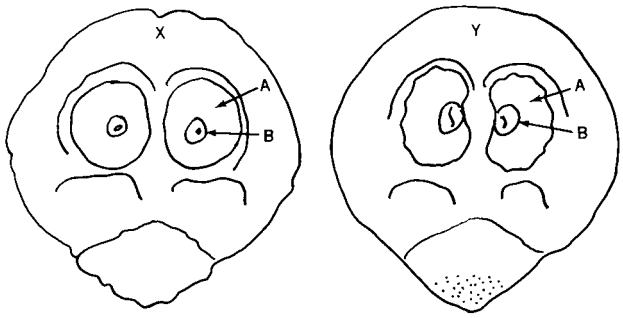

Figure 8.4. Third instar larvae. (X) *Oestrus ovis*, (Y) *Cephenemyia* spp., (A) peritreme, (B) button, posterior view.

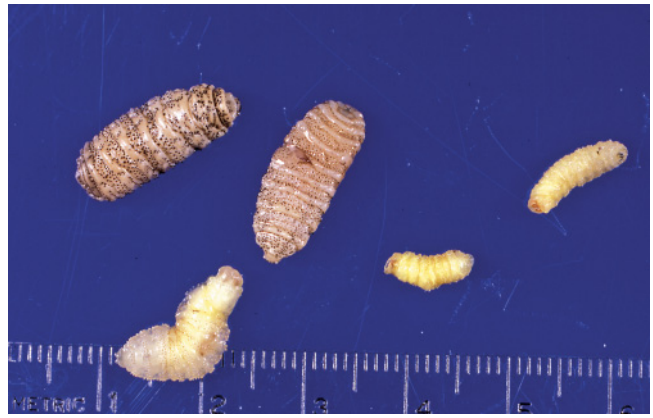

Figure 8.5A. Third instar larva of *Cephenemyia* sp.

Figure 8.5B. Third instar larva of *Cephenemyia* sp.

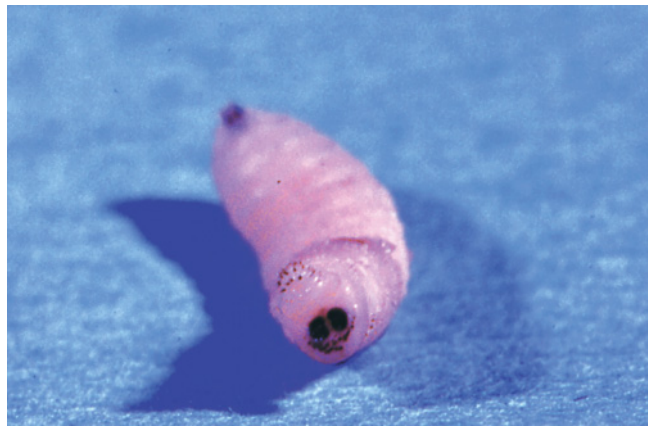

Figure 8.5C. Posterior peritremes of *Cephenemyia* sp.

into a sinus. The cycle in the llama has not been defined as to the location or length of time necessary to complete maturation, if, indeed, the larvae reach maturity in this species. In sheep, the first instar progresses to the second in two weeks to as long as nine months. The second instar matures in twenty-five days or more. Mature larvae crawl out of the nasal cavity, fall to the ground, and pupate for three to six weeks in warm weather or remain viable in the pupal stage for months if protected from freezing.

EPIDEMIOLOGY. The fly is active during the day but spends most of its time resting in cracks or crevices. In temperate climates, the fly is active in all but winter months, and in warm climates throughout the year. This fly is a serious parasite in sheep and goats in many areas. It has been only circumstantially identified from a camelid. Proper identification should be pursued on any nasal larvae found in a camelid, because the more common larva found has been the deer bot.

CLINICAL SIGNS. The syndrome seen in llamas has not been reported in the literature. Affected llamas exhibit signs similar to those seen in sheep, with nasal exudation, sneezing, and head shaking prominent. Visualization of the larvae with endoscopic examination would be fortuitous because the larvae are usually in the sinuses.

TREATMENT. Ivermectin therapy, at doses of 0.2 mg/kg, has been successful.

Cephenemyia Spp. (Deer Nasopharyngeal Bot, Throat Bot, Deer Nasal Bot)

IDENTIFICATION. The adult fly superficially resembles a small bumblebee and is approximately 14 mm in length.[29] First instar larvae are approximately 1 mm long and may molt up to 3 mm. Second instar larvae vary in length from 3 to 13 mm. Third instar larvae vary from 12 to 40 mm. The puparium is 16 to 20 mm long. Third instar larvae of all species are similar, making it difficult to identify the species. First instar larvae are most easily identified but are rarely seen or recovered.

A third instar larva removed from the nasopharynx of a llama had the following characteristics: cream color, 2.5 cm long, and approximately 0.7 cm in diameter (Figure 8.5A to 8.5C). The peritremes (stigmal

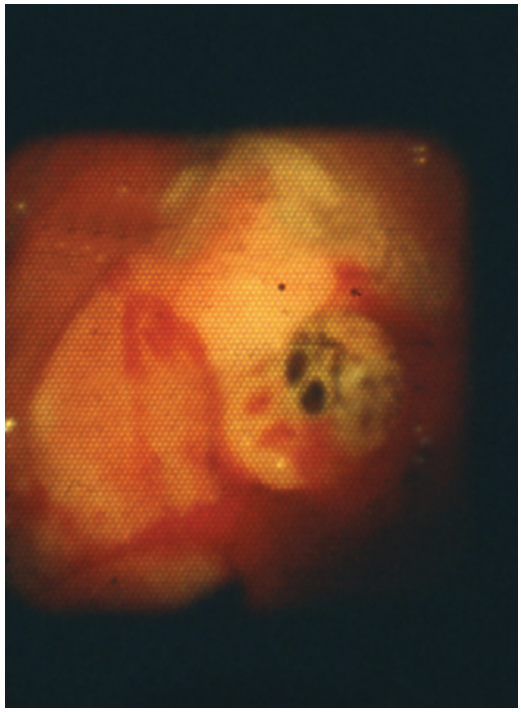

Figure 8.6. Endoscopic view of a cephenemyia larva in situ.

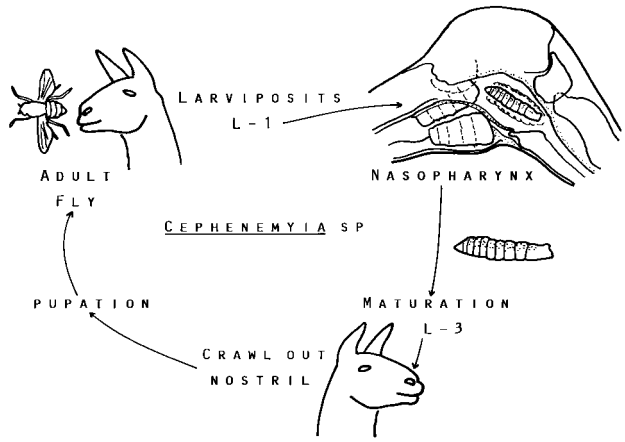

Figure 8.7. Life cycle of *Cephenemyia* spp.

plates) were crescent shaped, with the outer margins slightly sinuous. The button was on the inner margins (Figure 8.4). In contrast, the peritremes of the third instar *O. ovis* larva are circular with a central button (Figure 8.4).

LIFE CYCLE. Primary hosts for *Cephenemyia spp.* are cervids. Whether the cycle is completed in camelids is unknown. In deer, the adult fly lives for two to three weeks without feeding. Larvipositing by the female is carried out by spraying larvae into the nostrils of the host (Figure 8.6). Larvipositing evokes a behavioral response in deer. The head is jerked back, and sneezing and snorting ensue, accompanied by head shaking. Older animals may lower the head, pressing the nose against the ground. A few animals will run a few yards immediately following a strike.[29]

The larvae may remain for a time in the nasal passageways but ultimately migrate to the nasopharynx, where they take up residence in the retropharyngeal pouches found in cervids and develop to the second and third instar. At maturity, the third instar larvae migrate or are sneezed out through the nostrils, fall to the ground, pupate, and after sixteen to thirty-one days emerge as adult flies (Figure 8.7).

EPIDEMIOLOGY. A number of llamas have been infested with the deer nasopharyngeal bot.[29] Camelids cohabiting pastures with deer are at risk. Various species of flies of this genus can be found in any area of North America where there are cervids. Camelids are aberrant hosts for this parasite, and it is not known whether the cycle is completed in the llama.

CLINICAL SIGNS. In deer, the oral hooks and spines of the larvae irritate the mucosa of the nasopharynx, inducing secretion of a viscous mucoid exudate that apparently provides nourishment for the larvae. Affected deer may have a nasal exudation and sneeze periodically.

Because the camelid is an aberrant host, there is a more significant reaction, with the development of a granulomatous swelling in the nasopharynx and nasal cavity.[73] None of the affected llamas have died, and no biopsies have been taken to establish the histologic picture of this mass. However, the larvae can be seen embedded in the surface of the mass with endoscopic examination.

Prominent signs in the llama are sneezing and coughing, with or without a nasal discharge. The lesion is in the nasopharynx, and any exudation may be swallowed, not pushed out through the nasal cavity. Owners have reported that llamas are short of breath or fail to keep up with others on the trail. Though the llama is an obligate nasal breather, if the lesion becomes too obstructive, the llama will be forced to mouth breathe, with all its debilitating effects (Chapter 22). Audible breathing will also be heard. There is usually no febrile response or change in the hemogram. This is significant in differential diagnosis to rule out respiratory infectious diseases.

DIAGNOSIS. The characteristic clinical syndrome, observed in a llama in an area harboring deer, would be primary evidence. The lesions and perhaps the larvae may be seen via endoscopic examination in an anesthetized individual, Figure 8.6. The mass appears as a radio-dense area in a radiograph (Figure 8.8). A final diagnosis may be made on the basis of response to treatment.

TREATMENT. Although too few cases have been seen and treated to be conclusive, there is evidence

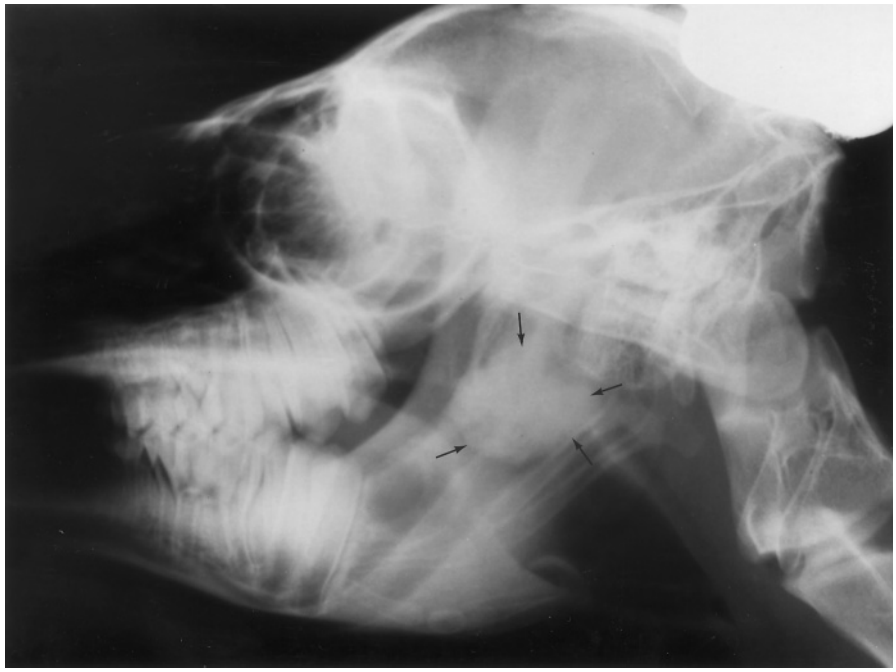

Figure 8.8. Lateral radiograph of the head of a llama with a Cephenemyia granuloma in the nasal pharynx.

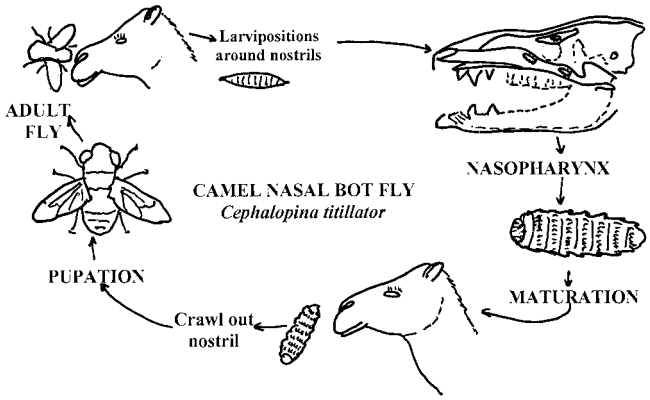

Figure 8.9. Life cycle of *Cephalopina titillator*.

that ivermectin is effective at double or triple the usual 0.2 mg/kg given subcutaneously (0.4 or 0.6 mg/kg).

Camel Nasal Bots

ETIOLOGY. The larval stages of the camel nasal bot fly (*Cephalopina titillator*) (family Oestridae) are common inhabitants of the nasopharynx of OWC.[8]

LIFE CYCLE. The female fly deposits first stage larvae in the nostrils. The larvae are small, 0.7 mm long. They migrate to the nasopharynx and attach to the mucosa. The larvae molt twice and mature up to eleven months before detaching from the mucosa and being sneezed out of the nostril. Larvae may also be seen in the labyrinth of the ethmoidal bone. Pupation occurs in the soil (Figure 8.9).

EPIDEMIOLOGY. The prevalence of *C. titillator* infestation is high in countries maintaining a sizable camel population, varying from 47% to 100%.

CLINICAL SIGNS. The adult flies do not annoy camels like the deer bot fly does SACs, and they may even be seen in large numbers resting on the heads of camels. Usually camels exhibit no clinical signs of infestation except when mature larvae are migrating from their attachment sites to exit the nostril, where sneezing is common. Local inflammation may occur at the site of attachment and heavy infestations may cause nasal exudation. Mortality is rare and is usually associated with larval penetration of the ethmoturbinates, resulting in meningitis. The camel is the primary host of *C. titillator* and not an aberrant host as is cephenomia in SACs; thus, there is no granulomatous reaction with potential obstruction of the airways.

DIAGNOSIS. Larvae may be seen exiting the nostrils. Larvae are frequently found in the nasopharynx at necropsy when camels die from other diseases. The larvae may be observed in situ by endoscopic examination.

MANAGEMENT. The larvae may be killed with ivermectin, 0.2 mg/kg SC, or other parasiticides.

Ticks

Identification

There are two major groups of ticks: hard-bodied ticks (family Ixodidae) and soft-bodied ticks (family Argasidae). There are numerous genera of hard-bodied ticks. Although no reports in the literature

list the species found in SACs, it is likely that those that are not host specific are identified according to the locality inhabited by SACs.[25] Tick paralysis (discussed later) has been reported in a llama as a result of hard-bodied tick attachment.

The only soft-bodied tick that has caused a problem in llamas is the spinose ear tick (*Otobius megnini*). The adult tick, which is not parasitic, is about 8 mm long and has a constriction at the middle, giving it a fiddle shape.[112] The larvae are pear shaped to spherical and about 2 to 3 mm long. The nymph, which is the primary parasitic stage, molts twice, and when engorged measures 7 to 10 mm in length.

Life Cycle

SPINOSE EAR TICK. Adult ticks do not feed and may live for six months in cracks and crevices of barns, sheds, and fences or under feed bunks or stones (Figure 8.10). The female may oviposit 500 to 600 eggs in the previously mentioned areas during her lifetime. The eggs hatch to six-legged larvae, ready to attach to a host in ten days. The larvae migrate to the ear canals and begin to feed on lymph. Larvae survive only two to four months without a suitable host.[100]

The eight-legged nymphs molt twice within the ear canal, remaining on a single host for one to seven months, feeding on blood. The mature nymph climbs out of the ear canal, drops to the ground, and molts to the adult stage. Usually only one mating cycle is completed each year.

HARD-BODIED TICKS. Some species complete the entire cycle using a single host species, others require two hosts, and a few, three separate hosts, one for each of the life stages. The female oviposits only once, producing thousands of eggs. Larvae feed only once, and there is only one nymph stage, taking a single feeding. Hard-bodied ticks are more likely to be encountered in open grassland or shrubby areas rather than near sheds and barns, as is the case with soft-bodied ticks.

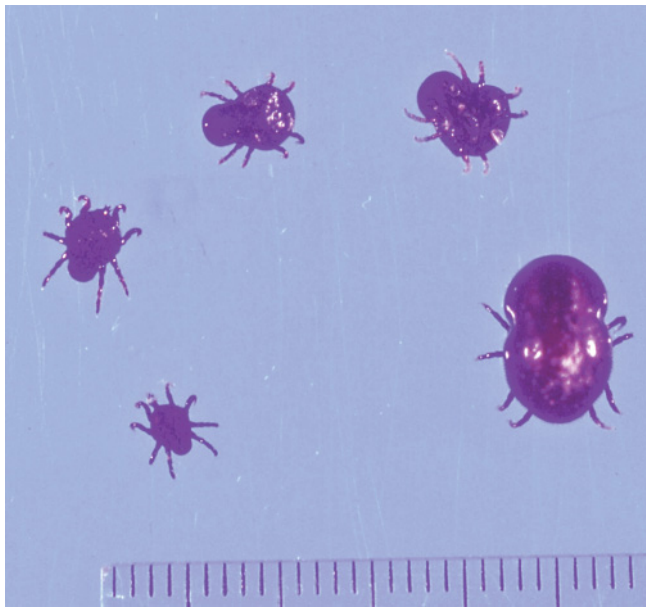

Figure 8.10. Spinose ear tick nymphs.

Epidemiology

Spinose ear ticks are a problem in llamas and alpacas in localized pockets in the western United States. Animals kept entirely on pasture or open range are less likely to encounter the ticks because the preferred habitat of these ticks is around buildings, sheds, wooden fences, and rough-barked trees, e.g., Eucalyptus spp.

Hard-bodied tick infestation is a problem in much of the West, especially with llamas used for packing into wilderness areas.

Clinical Signs

The two prime signs of spinose ear tick infestation are head shaking and exudation from the external ear canal. Examination should reveal the nymphs within the ear canal. Anemia may result in young llamas with heavy infestations.

Treatment

Ivermectin (0.2 mg/kg) administered subcutaneously is effective for both the larvae and nymphs of the spinose ear tick. The individual ear canal may be physically cleaned with standard techniques and an insecticide solution instilled into the ear canal. The problem is to prevent reinfestation. There is little hope of eradicating the tick from an environment wherein the adults hide in crevices and under objects difficult to reach with sprays. Periodic inspection and treatment is necessary to prevent a buildup of infestation.

Tick Paralysis[27]

EPIDEMIOLOGY. Tick paralysis has been recognized for more than fifty years, and forty-three species in ten different genera of ticks have been incriminated.[26] Usually only female hard-bodied ticks produce toxin, which is localized in the saliva and is injected into the host when the tick ingests a blood meal. No information is available as to why a given tick produces the toxin.

In the United States, the neurotoxin from *Dermacentor* spp. interferes with acetylcholine liberation at the neuromuscular endings, causing a lower motor neuron paresis. The bite from a single tick is sufficient to kill an animal if the tick is not removed. Based on work in other species, there is variable host susceptibility. There may also be seasonal or annual variability, since there have been reports of epidemics of paralysis in some years and none in other years.

Clinical Signs. Tick paralysis has been diagnosed in the llama.[25,64] A one-year-old male llama was observed as normal on one day and the following day was totally paralyzed. A diffuse lower motor neuron disease was suspected. *Dermacentor* spp. ticks were found.

The llama was sprayed with a pyrethrin insecticide, and ivermectin (0.2 mg/kg) was administered parenterally. Muscle tone reappeared within twelve hours, and the llama was standing in thirty-six hours.

An affected camelid would probably follow a similar progressive course as noted in other species. Signs may not develop for five to seven days after the tick begins to feed. Paresis and paralysis are progressive, beginning in the rear quarters and moving cranially. Ataxia develops, followed by a loss of all motor functions. Pain perception remains, according to human victims.

The signs may progress rapidly over a few hours or more slowly over a period of twenty-four to forty-eight hours. Paralysis ascends to the forelimbs, neck, head, and face. In early stages, the victim is bright and alert, able to eat and drink, but as the paralysis ascends, difficulty in chewing and swallowing develops. The cause of death is respiratory arrest from involvement of the respiratory centers of the brain.

Treatment. No drug can counter the effects of the neurotoxin. The cure is to remove the tick from the animal. The difficulty lies in finding a tick on an unshorn llama or alpaca. Because only one tick may be sufficient to cause the paralysis, finding it is crucial. In one tragic death in a human infant, the tick was located within the vulva.

Removal of the tick may reverse the signs in two to twelve hours. Assuming inability to find the offending tick, a standard insecticide dip may be employed. Amatraz (Mitaban, UpJohn, Kalamazoo, Michigan) may be applied as a dip in a concentration of 250 ppm of the active drug (10.6-ml bottle/2 gal water).

In Australia, Ixodes holocyclus is responsible for a serious form of tick paralysis. Even if the offending tick is found and removed, recovery may not occur unless specific tick antiserum is administered.

If dyspnea and respiratory arrest occur, the patient must be intubated and maintained on artificial respiration until the effects of the neurotoxin wear off.

Mites[58]

Three types of mange mites have been reported from camelids: sarcoptic, psoroptic, and chorioptic. Of these, the mite that causes sarcoptic mange is the most common and troublesome. The true identity of the mite must be established to institute proper therapy and determine whether or not the case must be reported to regulatory authorities. Psoroptic mange (scab) is a reportable disease in the United States, and all forms of mange are reportable in certain states.

Transmission

Direct contact is usually necessary for a new animal to acquire any of the mange mites. However, objects such as clothing, bedding, and grooming tools may serve as mechanical vectors. Dust bathing areas are also sources. Adult mites may survive off the host on epithelial debris for a maximum of thirty days. Eggs hatch in foru to ten days, and larvae must feed within ten days or die.

Sweatman has shown that *Psoroptes* spp. and *Chorioptes bovis* are not host specific, and cross transfers are theoretically possible.[103,104] Sarcoptic mites are thought to be host specific. No cases of zoonosis from camelid sarcoptic mange have been reported, but it has been reported from camels in Australia.

Diagnosis

Any of the mange mites may be seen (but not identified) at 30× with either a dissecting or a compound microscope. At 100×, the mite will fill the field of view, and most of the identifying characteristics can be seen; however, the segmentation of the pedicel attached to the suckers in *Psoroptes* spp. can be clearly seen only at 400×.

Scrapings for mange mite identification should be taken at the periphery of a lesion. When scraping lesions, it is necessary to penetrate deeply enough to draw blood. The debris should be scraped into a glass tube or plastic container, placed on a slide, bathed with 10% to 15% potassium hydroxide solution warmed to boiling, and examined for mites.

Treatment

Psoroptic and chorioptic manges respond readily to insecticide dusts, sprays, and dips, because mites live on the surface of the skin. Sarcoptic mange is more difficult to treat topically, but ivermectin (0.2 mg/kg) given subcutaneously has proven to be highly effective.[63] Severe cases may require repeated treatment or an increase in the dose.

Sarcoptic Mange Mite[34,63,75,105,109]

Identification. *Sarcoptes scabiei* is morphologically identical in all animals. The identifying characteristics of the mite are a globose or round body with short legs.[20] The caudad two pairs of legs do not extend beyond the margin of the body. There are bell-shaped suckers (caruncles) on the long, nonsegmented stalks (pedicels) on the tarsi of all the legs in the male but only on the anterior two pairs in the female mite. The male has no adanal suckers (copulatory discs). The capitulum (head or false head) is broad (Figure 8.11).

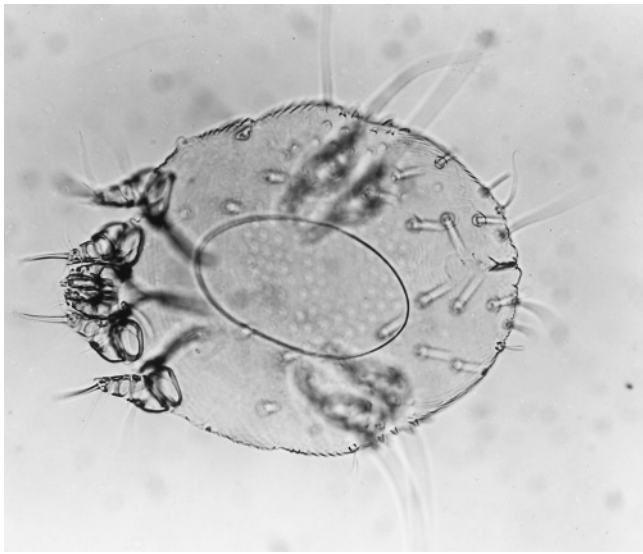

Figure 8.11. Gravid *Sarcoptes scabiei*.

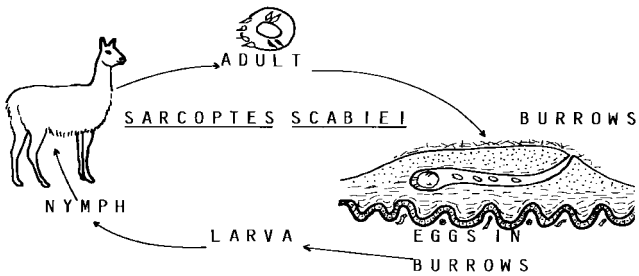

Figure 8.12. Life cycle of *Sarcoptes scabiei*.

The maximum size of the female mite is about 0.5 mm and of the male, 0.3 mm.

Life Cycle.[100] The entire life cycle is completed in the skin of the camelid (Figure 8.12). Adult male and female mites copulate on the surface of the skin. The fertilized female burrows into the skin, depositing eggs behind her as she tunnels along. The eggs hatch in three to eight days. Larvae migrate to the skin surface and mature through the nymph stage to become adults in four to six days. The entire life cycle is completed in seven to fourteen days. Males and unfertilized females may also burrow or may follow the tunnels of the fertile female.

Clinical Signs. The burrowing mite causes hyperemia, papules, and pustules, which become encrusted.[26] The skin becomes thickened in affected areas and loses its vitality, thus becoming susceptible to secondary bacterial infection, with subsequent exudation. Pruritus is common. Lesions are usually found on the limbs (between the toes), medial thighs, ventral abdomen, chest, axilla, and perineum of the female and prepuce of the male. This distribution is similar to that seen with chorioptic mange, but the thickening of the skin is the differential characteristic of sarcoptic mange. In severe cases of infestation, the entire head, body, and limbs may be involved (Figures 8.13 to 8.18).

Figure 8.13. Sarcoptic mange on the lips of an alpaca.

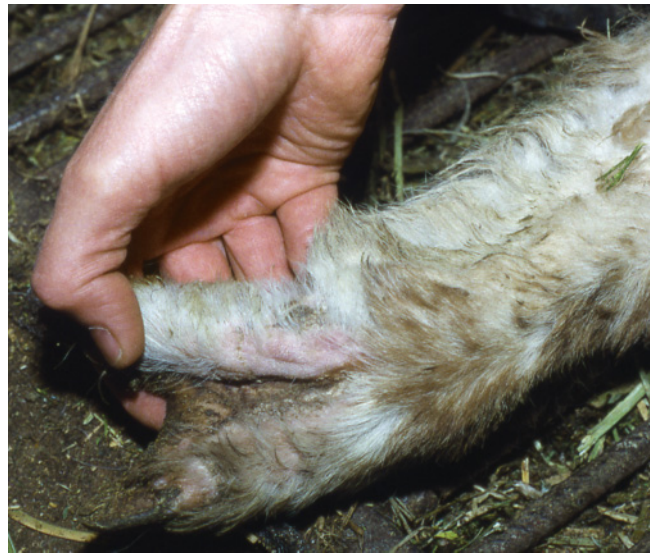

Figure 8.14. Sarcoptic mange in the interdigital space of an alpaca. There is a scent gland in this area that may have an accumulation of debris on it normally.

Psoroptic Mange Mite

Identification.[100,104] The body of *Psoroptes* spp. is more oval shaped than that of *Sarcoptes scabiei*, and the legs are longer, projecting beyond the margin of the body (Figure 8.19). The pedicels of the tarsal suckers are long and composed of three segments (Figure 8.20). In the female mite, the third set of legs end in bristles instead of suckers (Figure 8.21). In the male mite, all legs have suckers, and the fourth set of legs (caudal set) are much shorter than the third. The male mite also

Figure 8.15. Sarcoptic mange of the caudal abdomen and inguinal region in an alpaca.

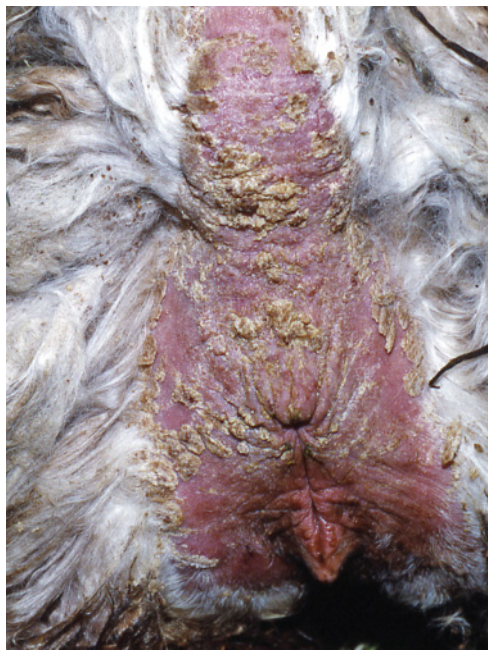

Figure 8.16. Sarcoptic mange in the perineal region of an alpaca.

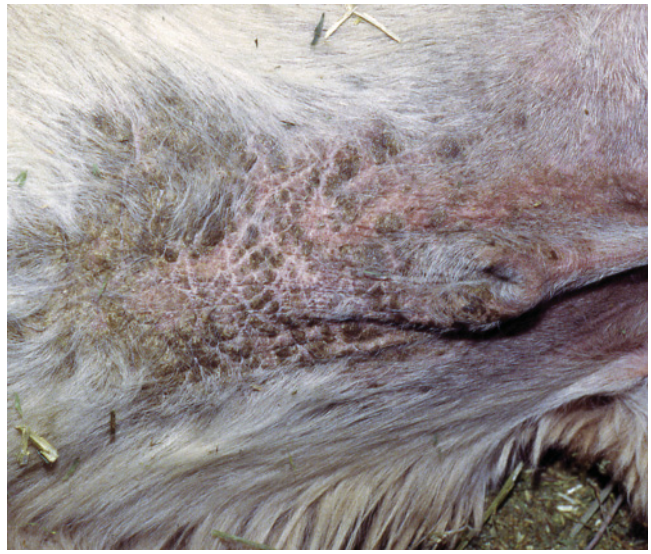

Figure 8.17. Sarcoptic mange of the prepuce of an alpaca.

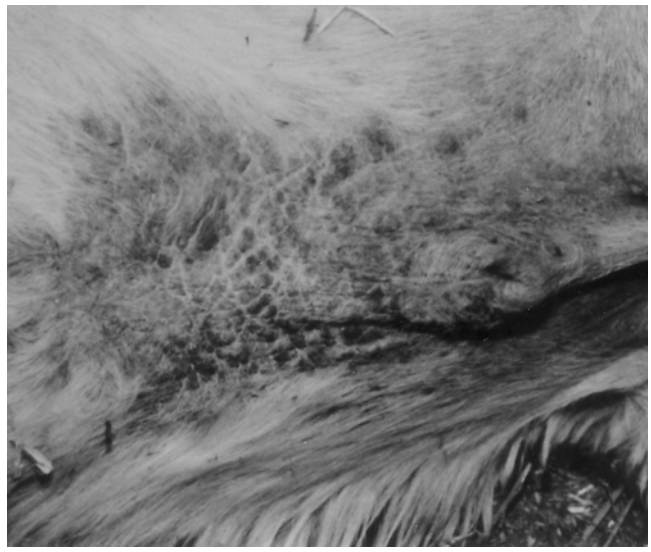

Figure 8.18. Healing sarcoptic mange of the prepuce and abdomen of an alpaca.

has a pair of adanal suckers. The posterior margin of the abdomen of the male is bilobed, and the capitulum is elongated. The maximum size of the male mite is 0.7 mm, that of the female about 0.8 mm.

LIFE CYCLE. The entire cycle is completed on the host. Eggs are deposited on the skin at the periphery of the lesions and hatch in one to three days if the eggs remain in contact with the skin. If separated from the skin by crusts, hatching becomes delayed until four to five days, and if detached from the body along with wool, the eggs will either hatch in ten days or die. The larval stage lasts for two to three days, during which time feeding occurs. The nymphal stage lasts from three to four days. Adults copulate soon after maturation. A female mite lives for thirty to forty days, laying about five eggs daily. The cycle may be as short as ten days but is usually about three weeks.

CLINICAL SIGNS. The mouth parts of mites penetrate the epidermis to suck lymph and in so doing stimulate a local inflammatory reaction that exudes serum. The

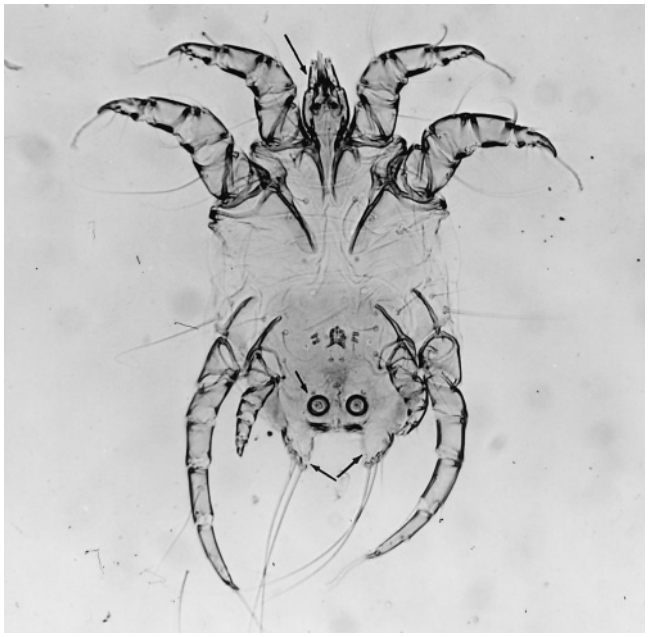

Figure 8.19. Male *Psoroptes* sp. mite. Note elongated capitulum, adanal suckers, and bilobed abdomen.

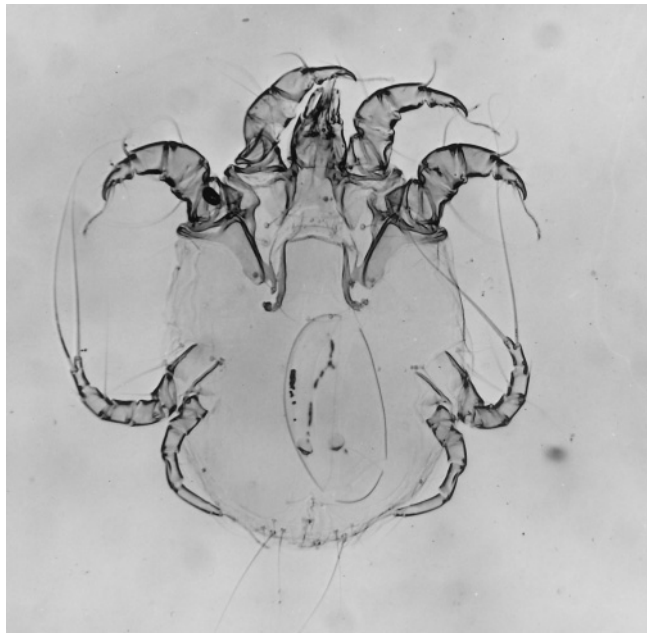

Figure 8.21. Gravid female *Psoroptes* sp. mite.

Figure 8.20. High-power magnification (400×) of the segmented tarsal pedicel of the tarsal suckers of *Psoroptes* sp.

serum coagulates to form crusts (hence the name "scab"). The dermatitis produced causes pruritus and fiber loss. Psoroptic mites have been found only on the pinna or within the ear canal in the United States, causing head shaking and incoordination.[30] Other reports indicate that the mite prefers the heavily fibered areas of the body. When fibers are pulled, rubbed, or fall out, the mites move to the margins of the lesion. This habit should be kept in mind when scrapings are collected to check for the presence of mites.

Lesions are usually located around the shoulder and along the back, sides, and tail head. An early lesion or one at the margin of an established infestation consists of small papules about 5 mm in diameter. The papules are yellowish and have a moist surface. A mite is likely to be found in the center of a papule. Within four to five days, the serum will exude, congeal, and form the crusts characteristic of scab. Although dermatitis is produced, the skin does not become thickened to the extent observed in sarcoptic mange.

EPIDEMIOLOGY. Little is known about the seasonality of psoroptic mange in camelids, but in sheep, it is most active during the fall and winter. Psoroptic mange is a reportable disease in the United States. One case in llamas, involving a cria and his dam, has been reported in the United States, and only a few reports have come from South America. Identification to species was not possible in the case occurring in the United States.

Chorioptic Mange Mite[103,113]

IDENTIFICATION. *Chorioptes bovis* closely resembles *Psoroptes* spp. except that the tarsal suckers are on short, unsegmented pedicels. The capitulum is shorter and blunter than that of *Psoroptes* spp. (Figure 8.22). Female chorioptic mites are approximately 0.4 mm long, and males, 0.35 mm (Figure 8.23); larvae are 0.2 mm.

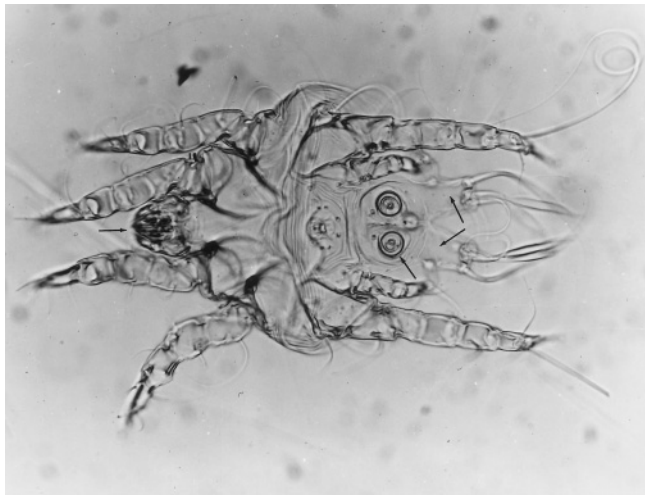

Figure 8.22. Male *Chorioptes bovis* mite from a llama. Note the broad, shortened capitulum, adanal suckers, and bilobed abdomen.

Figure 8.23. Copulating adult male (left) and nymphal female *Chorioptes bovis* mites.

CLINICAL SIGNS. Chorioptic mites feed on epidermal debris on the skin surface. These mites have short chelicerae adapted for chewing, in contrast with the penetrating chelicerae of psoroptic mites. In some cases diagnosed by the author, lesions were found on the ventrum of the tail, around the anus and vulva (Figure 8.24), and extending ventrally on the inner sides of both hind legs to the ventral abdomen (Figure 8.25). The lesions extended laterally up to more heavily fibered areas. Lesions were also present in the axilla, again involving lightly fibered areas. Other sites included the tips and inner surfaces of the ears and between the digits, extending up to the fetlock. The basic lesion of chorioptic mange is similar to that of psoroptic mange. The skin is hyperemic and covered with incrustations 0.5 to 1.5 cm in diameter (Figure 8.26). The pathogenesis of the lesion is unknown.

EPIDEMIOLOGY. Chorioptic mange is supposedly more ubiquitous and less debilitating to the host than

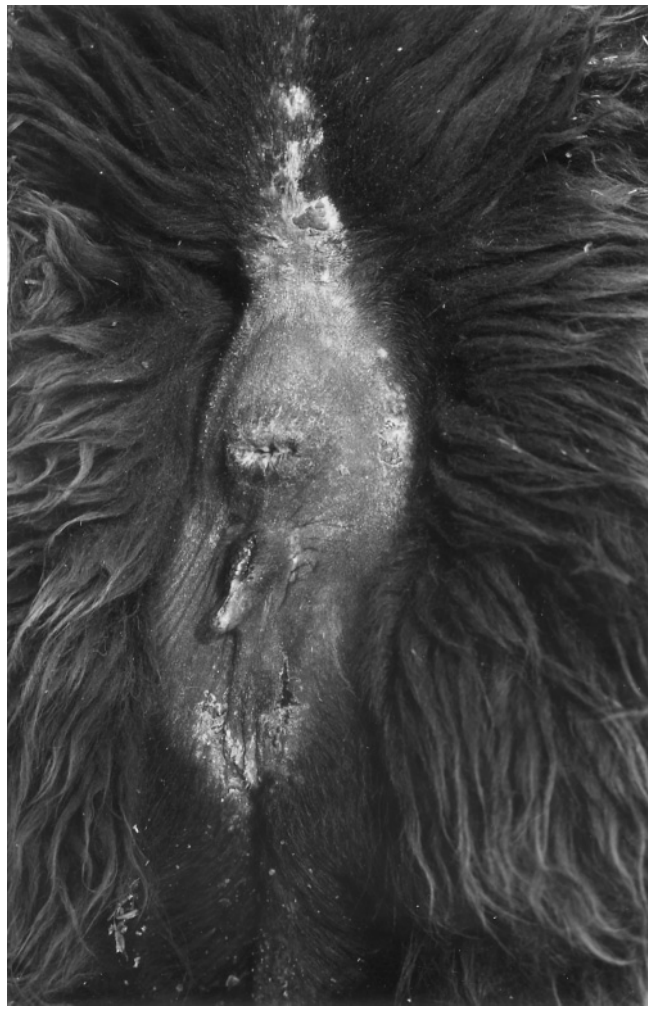

Figure 8.24. Chorioptic mange on the perineal area of a llama.

Figure 8.25. Chorioptic mange on the ventrum of a llama.

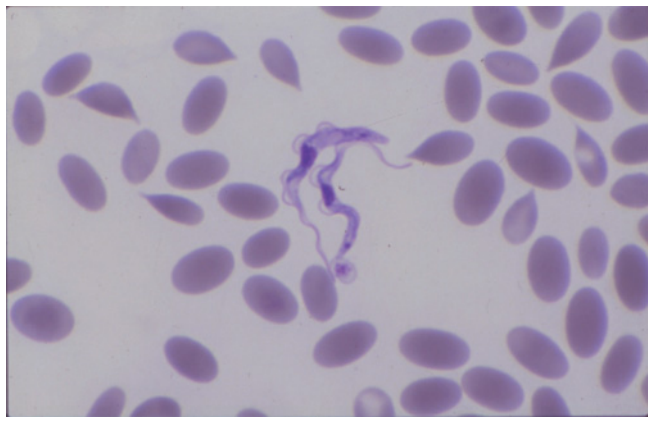

Figure 8.27. *Trypanasoma evansi* in a camel.

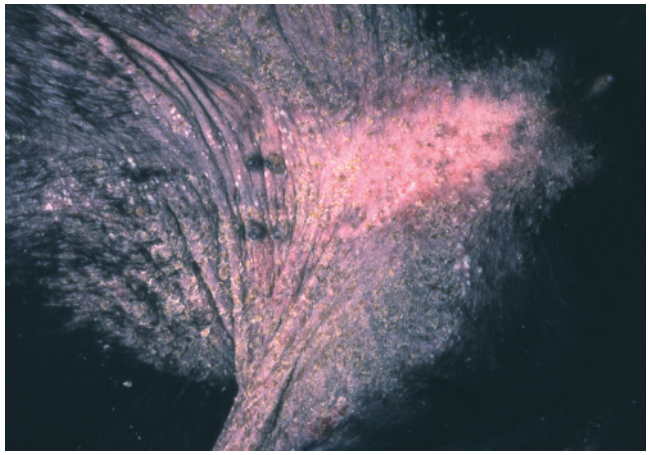

Figure 8.26. Encrustations typical of both psoroptic and chorioptic mange.

the other two manges. Chorioptic mange is rarely reported in llamas, but the author has diagnosed cases and it has been reported in South Africa[113] and by Sweatman.[103]

INTERNAL PARASITES

Protozoa[68]

Protozoal diseases are important throughout the world. Eperythrozoans were first classified as protozoa[74] but have since been reclassified as rickettsial organisms (Chapter 7). Some blood parasites have yet to be precisely identified.

Trypanosomiasis (Surra)[8]

IDENTIFICATION. Old World camels are highly susceptible to infection by a number of species of Trypanosoma. Camels are not commonly used in the region called the Tsetse Belt. Illness and death caused by *T. brucei*, causing nagana, may be the key factor of their absence from the area. *T. evansi* is the major trypanosome affecting camels, causing a disease called surra (Figure 8.27).[28] Surra is an Indian word meaning rotten. *T. evansi* may have some significance in SACs for reasons to be enumerated.

Trypanosome species are similar, and species identification may be impossible without animal inoculation and other sophisticated diagnostic procedures. They have a leaf-like shape, with a single flagellum attached to the cell by an undulating membrane.[112]

LIFE CYCLE. *T. evansi* does not require a period of maturation in an insect vector and thus has a noncyclic transmission. Any biting or blood-sucking insect or tick may serve as a vector. Flies of the genera Tabanus and Stomoxys are commonly implicated. Mechanical transmission by contaminated hypodermic needles is also possible. In species that do not require an intermediate host, the trypanosomes are deposited in the blood by bloodsucking insects. In the camelid host the organisms multiply by longitudinal binary fission in blood, lymph, and cerebrospinal fluid. When that fly takes another blood meal it becomes a mechanical vector.

T. brucei and *T. congolensis* are the primary cause of human sleeping sickness and are transmitted by the tsetse fly (*Glossinia* spp.) which serves as an intermediate host. OWC may be infected with these organisms as well. When the tsetse fly takes a blood meal from an infected animal the organisms multiply within the hind gut of the fly. There they undergo a metamorphosis and migrate to the salivary glands and reach an infective state that is then transmitted when the fly takes another blood meal.

EPIDEMIOLOGY. Trypanosomiasis (surra, sleeping sickness, Chagas disease, nagana)is a blood parasitism affecting humans and numerous species of mammals including camelids. *T. evansi* is the primary species affecting OWC and is widespread throughout North Africa, Asia Minor, countries of the former USSR, Pakistan, Afghanistan, India, Burma, Malaya, Indochina,

South China, Indonesia, and the Philippines.[27] It has also been reported from Central and South America, though trypanosomiasis has not been reported in SACs in these areas, despite the presence of the organism. Trypanosomiasis caused by *T. cruzi* is an important disease of humans and animals in the New World. Unidentified trypanosomes have been isolated from llamas imported into the United States from Chile.[b]

T. evansi is transmitted mechanically by biting flies, but has no intermediate host.

Clinical Signs. Acute surra is characterized by fever, progressive anemia, depression, weakness, ventral edema, and paralysis (Figure 8.28). The presence of pulmonary edema may contribute to the development of secondary pneumonia. Females may abort, and the milk of lactating females may become caseous. Large numbers of trypanosomes are seen in peripheral blood samples. Death may occur within a few weeks. The subacute form is characterized by fever, edematous plaques, emaciation, high mortality, and death within a few weeks.

Chronic surra is characterized by intermittent episodes of fever, anemia, pendant edema, and emaciation. Between episodes of fever, the parasite may be absent from peripheral blood vessels. A camel may live for three or four years, depending on the care provided.

Figure 8.28. A camel ill with trypanosomiasis.

Diagnosis. Finding the organism in the peripheral blood of infected camels may be highly problematic because of the low numbers of the organisms and fluctuating parasitemia. The development of a suitable ELISA serological test has been found to be effective in decreasing the economic losses associated with surra. Other DNA technologies are being used in endemic countries.

Toxoplasmosis

Toxoplasma gondii is a ubiquitous protozoan parasite.[15,22,61,93] Abortion has been diagnosed in SACs, and serum titers have been reported,[22,93] and it is likely that other manifestations of the disease will be reported in time.[93] Much has been written on this parasitic disease, and the reader is referred to recent reports.

Coccidiosis[13,38,39,43,44,46,48,49,60,94,97]

Identification. The species of eimeria that parasitize camelids are unique to camelids and are not transferable to livestock species or vice versa. Likewise, the species of Cryptosporidia and Sarcocystis are unique to camelids.[25,101] Other species of protozoan parasites may be shared by the other species of farm animals. Five species of the genus eimeria have been described from alpacas (Table 8.1).[39,60] All five species have also been identified by fecal flotation in llamas from Oregon, Wyoming, Colorado, Washington, Iowa, and California, so llamas and alpacas are hosts to the same species of eimeria.[31,91,95,100] Eimerian oocysts are frequently encountered on routine fecal flotations, and cases of coccidiosis have been reported. Coccidia identified from camels are listed in Table 8.2.

Life Cycle. The life cycle of coccidia includes both sexual and asexual phases (Figure 8.29). The asexual cycle is called "schizogony" or "merogony." Sporulated oocysts are ingested by the animal and pass along the digestive tract to the small intestine. The oocyst frees sporozoites, which invade the epithelial cells. The sporozoite changes shape and becomes a trophozoite, which, in turn, grows larger and forms a schizont (merzont). Within the schizont, merozoites form and ultimately rupture the cell and escape to infect other cells. This process may be repeated two or three times.

Table 8.1. Coccidia of SACs.

Species	*Size of oocyst (μm)*	*Shape of oocyst*	*Micropyle*
Eimeria alpacae	22–26 × 18–21	Ellipsoidal	+
E. lamae	30–40 × 21–30	Ovoid to ellipsoidal	+
E. macusaniensis	81–107 × 61–80	Ovoid	+
E. punoensis	17–22 × 14–18	Ellipsoidal or oval	+
E. peruviana	28–37 × 18–22	Ovoid	–

Source: Soulsby 1982.

Table 8.2. Coccidia of camels.

Species	*Size of oocyst (μm)*	*Shape of oocyst*	*Micropyle*	*Sporulation time (days)*
Eimeria bactriani	32 × 25–27	Spherical-ellipsoidal	+	10
E. cameli	81–100 × 63–94	Truncate	+	10–15
E. dromedarii	22–33 × 20–25			15–17
E. pellerdyi	23.2 × 12.6			5

Source: Soulsby 1982.

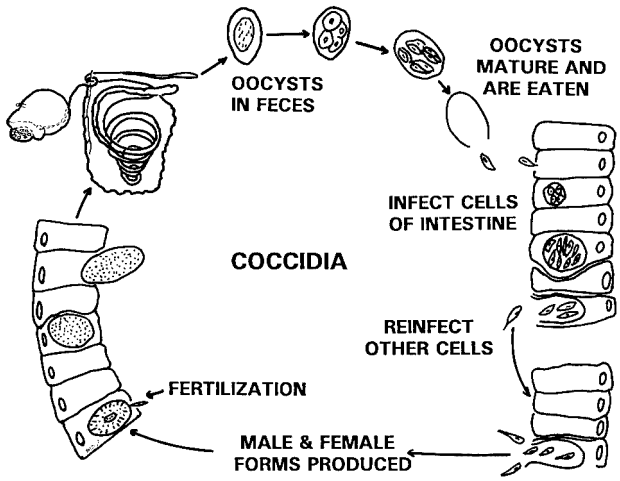

Figure 8.29. Life cycle of *Eimeria* spp.

The sexual cycle is called "gametogony." The merozoites produced by the last schizogony cycle infect a cell and develop into either male (microgametocyte) or female (macrogametocyte) gamonts. The male gamont fertilizes the female gamont while it is still in the cell, producing a zygote. The zygote matures to become an oocyst, which ruptures from the host cell and is shed in the feces. The oocyst sporulates in one to two days to become infective.

EPIDEMIOLOGY. Coccidial infections are generally self-limiting because asexual reproduction is repeated only two or three times.[112] Unless reinfection takes place, only one cycle of development can occur, but in a contaminated environment, reinfection is the rule, and heavy buildups can occur that may kill the host. Knowledge of the concept is important to the clinician, who must manage coccidiosis.

Ruminant livestock have a definite immune response to infection with coccidia. Whether or not this occurs in camelids is unknown. It is not uncommon to see eimerial oocysts on fecal flotation, but coccidiosis is uncommon in camelids in North America. It is a serious problem of recently weaned llamas and alpacas in Peru.

CLINICAL SIGNS. Coccidia invade the epithelial mucosa of the small intestine, causing enteritis and diarrhea. The diarrhea may be mild catarrhal or more serious hemorrhagic.[31,83,97]

TREATMENT.[21] Numerous coccidiostats are used in ruminants. Their use in camelids in North America has been recommended. Monensin and other ionophores should not be used until testing is carried out, because there is species sensitivity to these drugs. Horses have been killed by feeding from the same feed bunks as cattle being given medicated feed.

Cryptosporidiosis[6,25,80,95]

IDENTIFICATION. *Cryptosporidium parvus* and *C.* spp. are coccidial protozoan parasites that are of great concern to livestock managers and veterinarians. The organism inhabits the distal small intestine and large intestine. C. muris has been isolated from Bactrian camels.

LIFE CYCLE. The life cycle is similar to eimeria, however the small oocysts (4 to 4.5μm) sporulate within the host and thus are infective to other animals immediately upon passage in the feces. Autoinfection is possible. Transmission is via contaminated feed and water.

EPIDEMIOLOGY. Both OWC and NWC have been infected. Bactrian camels seem to be more susceptible than dromedaries. Predisposing factors include failure of passive transfer of immunoglobulins and concurrent infection. This parasite is a zoonosis.

CLINICAL SIGNS. The signs are those of a gastroenteritis. Severe diarrhea, tenesmus, dehydration, and emaciation may lead to death.

DIAGNOSIS. Oocysts may be seen in smears made of fresh feces. Modified Ziehl-Nielsen stain is commonly used with counterstaining with carbo-fuchsin. At necropsy there is villous atrophy leading to malabsorption. In crias and camel calves with severe diarrhea, other enteric pathogens must be considered, including *Clostridium perfringens* (enterotoxemias), *E. coli*, *Salmonella* spp., *Coccidia* spp., coronavirus, rotavirus, and bovine virus diarrhea virus.

MANAGEMENT. Dehydration and acidosis are the main consequences of severe diarrhea. Both of these must be dealt with by rehydration with intravenous fluids and appropriate administration of sodium bicarbonate. Cryptosporida are resistant to all commonly available, commercial antimicrobial agents and most disinfectants. Oocysts may persist in the environment for months to years.

Sarcocystiasis[11,78]

IDENTIFICATION. *Sarcocystis* spp. have an interesting two-host life cycle. Sarcocystis is a genus of coccidia, closely related to the eimeria. The primary host is a carnivore, in which gametogony, fertilization, and sporulation occur. Camelids serve as an intermediate host in which only schizogony and encystment occur. Two species have been reported from the camelid. *Sarcocystis aucheniae* has been reported from Bolivia and Peru[36,54] in the llama and alpaca and *Sarcocystis tilopoidi* from the guanaco in Argentina.[81]

Equine protozoal myeloencephalitis has only been recognized as a disease entity for thirty years, and the definitive etiology (*Sarcocystis falcatula*) established in 1994.[26] The horse is a dead-end host for this parasite that normally cycles between wild birds and opossums in the eastern United States. There is circumstantial evidence that protozoal myeloencephalitis occurs in llamas in the same area.[c]

LIFE CYCLE. Life cycles for camelid sarcocysts have not been established.[42] Whether dog, fox, cat, or human serves as the primary host is unknown. In general, the carnivore ingests the muscle of the herbivorous intermediate host (Figure 8.30). Zoites are released in the intestine and invade the epithelial cells. Gametogony is completed in the wall of the intestine of the carnivore up to and including the sporulated oocysts, which are excreted in the feces. This cycle differs from the eimerian cycle, in which nonsporulated oocysts must undergo sporulation outside the primary host.

The camelid ingests the sporocyst; sporozoites are released in the intestine and invade vascular endothelial cells. One or more asexual cycles are completed in these cells before the merozoites enter muscle cells and form sarcocysts.

EPIDEMIOLOGY. Sarcocysts have been encountered as a secondary finding at necropsy in North American llamas, but no cases of clinical disease have been described. In Peru, it has been reported that in certain areas, more than 50% of the camelids over three years of age are infected with sarcocysts.[d] Meat is downgraded as a result of the infection.

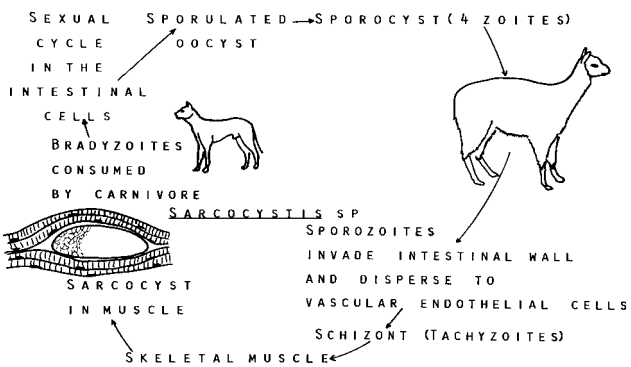

Figure 8.30. Life cycle of *Sarcocystis* spp.

A carnivore or omnivore must serve as the definitive host, passing the sporulated oocysts with the feces. The camelid serves as the obligatory intermediate host.

CLINICAL SIGNS. Little or no damage is done to the definitive host. No signs may be noted in light infections in camelids. In high numbers, the schizogonous cycles in endothelial cells may produce acute febrile disease, resulting in abortion and death. Sarcocysts may cause mild myositis with myalgia and interference with muscle function.

Protozoal myeloencephalitis in horses is characterized by nonspecific lameness, ataxia, and weakness, particularly of the hind limbs and atrophy of the gluteal muscles. The cardinal "A" signs include asymmetric ataxia and atrophy. Horses may also develop laryngeal hemiplegia and become dyspneic. A similar syndrome has been described in llamas, but meningeal worm disease also occurs in the same area.

TREATMENT. No treatment is available for the muscular form. Prevention requires disruption of the life cycle from the carnivore host. Treatment for protozoal myeloencephalitis in horses is pyrimethamine (1 mg/kg) once daily and sulfadiazine (20 mg/kg) once or twice a day, orally via a dose syringe. Treatment must be continued for four months. The regimen has been administered to llamas, but efficacy has not been verified.

Giardiasis

Giardia spp. cysts have been detected in immature, poor-conditioned llamas with soft stool or diarrhea.[59,95]

Trematode

Fascioliasis[18,85]

IDENTIFICATION. The common liver fluke, *Fasciola hepatica*, is locally ubiquitous and found throughout tropical and temperate regions of the world. This fluke is leaf-shaped (Figure 8.31) and reaches a maximum size of 30 × 13 mm. *Fascioloides magna* (large American fluke) is a parasite of deer in North America, and immature specimens of this fluke have been found in hepatic cysts in a llama.[14]

LIFE CYCLE.[99] Adult flukes live in the bile ducts (Figure 8.31). Eggs are discharged into the bile duct, carried to the intestine, and excreted in the feces. The eggs must fall into water for maturation to the ciliated miracidium stage to take place. This takes ten to twelve days. The miracidium bores into one of many species of snail. Lymnaea truncatula is one of the more common intermediate hosts, but other species of Lymnaea may also act as hosts. While in the snail, the miracidium loses its cilia and matures to become a sporocyst, then

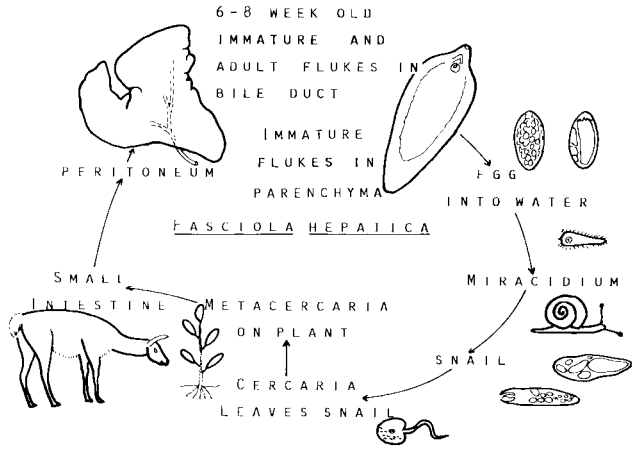

Figure 8.31. Life cycle of *Fasciola hepatica*.

a redia, and finally a cercaria. This phase requires four and a half to seven weeks.

The cercaria leaves the snail and is free swimming for a few minutes to two hours, attaches to a plant just below surface level, loses its tail, and becomes a metacercaria, which is the infective stage for the camelid. Metacercaria are ingested, and immature flukes (marita) are released into the duodenum; they penetrate the wall of the intestine, enter the peritoneal cavity, and migrate to the liver. By three to seven days after infection, the majority of the young flukes will have reached the liver, where they penetrate the capsule. Following this, a migratory period of some five to six weeks in the liver parenchyma occurs before the flukes enter the bile duct and mature.

The prepatent period is about eight weeks, but development may be retarded, delaying maturity another two months. Adult flukes may live for nine months or longer.

EPIDEMIOLOGY.[100] The completion of the fluke life cycle depends on the presence of snail intermediate hosts, which, in turn, depend on an aquatic environment. The eggs will not develop at temperatures below 10°C, and there is a direct correlation of development time with temperature (at 13°C, sixty days; at 15°C, forty days; and at 26°C, twelve days).

Some snail hosts are capable of estivation for as long as thirteen months under certain circumstances, e.g., during drought conditions. The infection may be maintained while the snail is buried in the dried mud.

Metacercaria can persist for a few days to a few weeks while encysted on the plant. Some of the cysts may fall to the bottom of the water and be stirred up and ingested when an animal walks into the water to graze or drink. The cyst may survive for as long as eight months on moist hay, but the usual drying process shortens life to a few weeks.[99]

Liver fluke infection in camelids has been reported from South America[3,12,56] and occurs in localized problem areas in the western and southern United States.[33,55,88,90] If flukes are endemic in local cattle and sheep, and llamas or alpacas inhabit similar swampy, poorly drained pastures, it is reasonable to assume that the camelids will have a fluke problem also.

CLINICAL SIGNS. Both acute and chronic forms of fascioliasis have been seen. The acute form occurs with overwhelming infections that produce signs of hepatic insufficiency similar to those caused by other agents (Chapter 13). The chronic form is more often seen. Chronic stasis of the bile, caused by flukes obstructing the ducts, produces a hepatic fibrosis, which ultimately causes an elevation of intrahepatic blood pressure. A hyperplastic cholangitis, which allows leakage of plasma protein, causing hypoproteinemia, also occurs. Adult flukes suck blood, causing intrabiliary hemorrhage, which results in anemia.

The affected llama becomes anorectic. Mucous membranes may be pale, and pendant edema may be seen. The fiber becomes dry and brittle, with "breaks" associated with intense periods of the disease. Depression and emaciation follow anorexia. Either diarrhea or constipation may be seen.

TREATMENT. Many different drugs have been used to treat fluke infestation. Currently, the only recommended drug in the United States is Clorsulon (Curatrem) at a dose of 7 mg/kg body weight. This is given per os, twice at forty-five- to sixty-day intervals. Albendazole is also effective.[21]

The unhealthy, fluke-infested liver may be susceptible to secondary bacterial infection from clostridial organisms or enterics. One disease of concern to livestock people is Black's disease, caused by *Clostridium novyi*. Vaccination with a multiple-antigen clostridial toxoid may prevent complications from this disease.

Tapeworms

Hydatid Disease[96]

IDENTIFICATION. Hydatid disease, caused by *Echinococcus granulosus*, is worldwide in distribution. The adult tapeworm resides in the intestine of a carnivore, with dogs, foxes, coyotes, and wolves all potential definitive hosts. This is a small tapeworm, 2 to 7 mm long, consisting of only three or four proglottids, which usually disintegrate in the intestine, so only eggs are seen in the feces.[96]

LIFE CYCLE.[100] The feces of the carnivore host contaminate the feed of the camelid (Figure 8.32). The eggs are immediately infective to any of many ungulate intermediate hosts, the camelid being one. The eggs hatch in the intestine, releasing oncospheres, which penetrate the intestinal venules or lymphatics and migrate to the organ of predilection for the species (liver, lung).

The hydatid cyst (metacestode) (Figures 8.33A and 8.33B) develops slowly over several months,[2,100] reach-

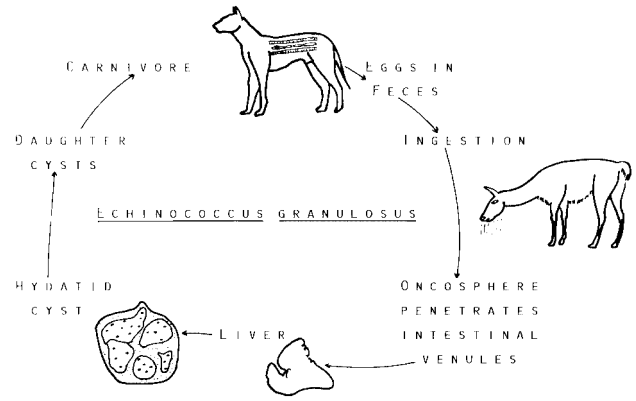

Figure 8.32. Life cycle of *Echinococcus granulosus*.

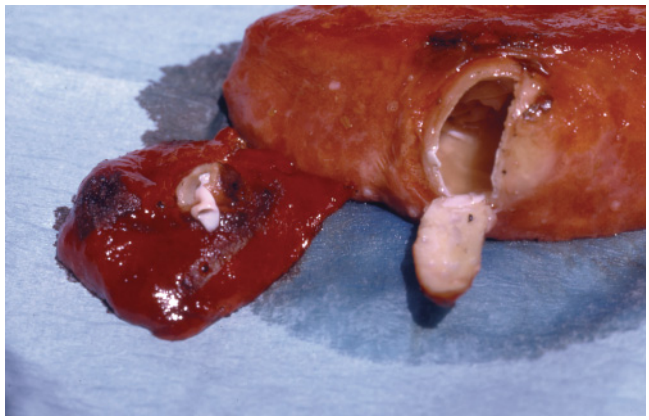

Figure 8.33A. Lesion of *E.granulosus* in the lung of a camel.

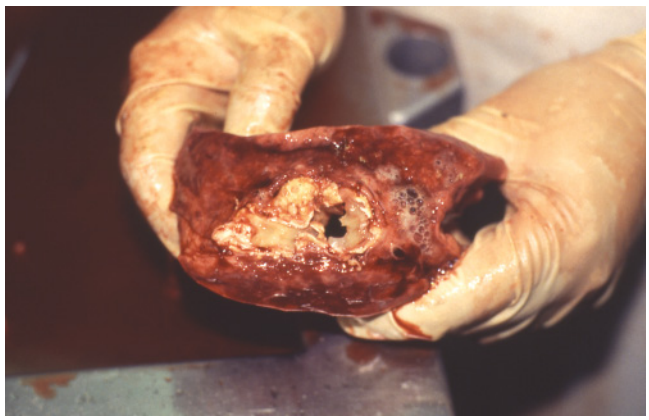

Figure 8.33B. Lesion of *E. granulosus* in a llama.

ing a diameter of 8 to 10 cm. The inner membrane of the cyst or bladder is composed of germinal epithelium and is surrounded by fluid enclosed in a capsule of inflammatory cells. Brood capsules, each with their own scolex, may develop within the outer capsule. Some of the brood capsules rupture, releasing scolices into the fluid ("hydatid sand"). The fluid is infective if the outer capsule is ruptured.

The hydatid cyst is consumed by the carnivore when it scavenges a carcass or is fed offal. The protoscolices are released in the intestine, evaginate, and penetrate between the villi into the crypts of Lieberkühn, developing to maturity in about forty-seven days. Dogs may maintain an infection for two years.

EPIDEMIOLOGY. Only one species of *E. granulosus* has been identified, but in the field, apparently, various strains or cycles exist between a particular species and a certain carnivore (sheep/dog, cattle/dog, camel/dog, wallaby/dingo [Australia], deer/coyote [California], moose/wolf). The cycle in camelids in South America is camelid/dog or camelid/fox.

CLINICAL SIGNS. The adult tapeworm is usually harmless to the carnivore, and except under unusual circumstances, a mild infestation in a herbivore produces no clinical signs. The cysts usually develop in the liver or lungs, but in humans and other hosts, they may develop in the brain or heart as well. In any case, malfunction of any organ will occur if large numbers of cysts develop or if a cyst develops in a vital area. If the outer wall of a brood cyst ruptures, metastasis may occur, with numerous new cysts developing as a result.

TREATMENT. No treatment can destroy the hydatid cyst in the intermediate host. Cysts can be removed surgically after the cyst has been injected with formalin to obviate accidental rupture of the cyst and subsequent contamination of the surgical site. Prevention consists of ridding the carnivore of the tapeworm. This may be successful in a controlled situation involving a pet dog cycle but is not possible in sylvatic cycles or areas where feral dogs roam.

Monieziasis

IDENTIFICATION. *Moniezia expansa* has been recovered from camelids in the United States, and *M. expansa* and *M. benedeni* are significant problems in some areas of South America. Adult tapeworms have been found in the small intestine of camelids. An intact tapeworm may reach 600 cm in length and be 1.6 cm wide. The scolex is 0.36 to 0.8 mm and has prominent suckers and no hooks.[100]

LIFE CYCLE. Mature proglottids separate from the rest of the tapeworm and, along with the contained eggs, are passed with the feces. Cysticercoid development occurs in oribatid mites, which are a true intermediate host. Infective stages develop in approximately four months.

Camelids acquire the infection by consuming forage contaminated with the mites. Further maturation to the adult tapeworm in the small intestine takes thirty-seven to forty days.

EPIDEMIOLOGY. Moniezia occurs worldwide but is usually not a major disease-producing parasite. Heavy infestations could obviously impair nutrition and cause debility or even intestinal obstruction. There may be a seasonality of infestation in temperate climates because of the overwintering of the oribatid mites. Neonate camelids begin to nibble on grass early in life and could acquire an infestation and be passing proglottids by the age of six weeks.

CLINICAL SIGNS. Diarrhea and unthriftiness may accompany heavy infestations.

TREATMENT. Praziquantel (Droncit) at a dose of 2.5 to 10 mg/kg given orally or as an injection is probably effective. Fenbendazole at 10 to 15 mg/kg is effective in cattle and sheep and may be effective in camelids.

Thysanieziasis

Tapeworms of *Thysaniezia* spp. have been reported in llamas. Little is known about the life cycle or epidemiology. Adult tapeworms of the genus have been found in the small intestine of sheep, goats, and cattle. Little or no clinical significance seems to be associated with this parasite.

Taenia Helicometra, T. Hidatigena

Infections with larval stages of these two *Taenia* spp. have been reported in alpacas and vicuñas in South America.[9]

Nematode Parasites[24,41,59,67,100]

Nematodes are the most numerous and most detrimental of the camelid parasites.[57,105] The taxonomic outline at the beginning of this chapter lists all the nematodes that have been reported from camelids. Most of these parasites are located in the GI tract. Many aspects of GI parasitism are similar, regardless of which species of parasite is involved.[66,69] The following introductory remarks obviate the need to repeat the same information for each species.

Pathogenesis

Most GI parasites produce a protein-losing gastroenteropathy. In severe cases, hypoalbuminemia may develop. Enteritis induces changes in the secretory status of the gut. Appetite and utilization of the feed consumed is reduced, depriving the body of vital nutrients. Absorption of calcium and phosphorus is depressed, causing, in turn, arrested skeletal development in the young animal. Selenium uptake is also retarded. Young animals are at greatest risk when affected by parasitism because no resistance has been developed to the invading organisms.

Clinical Signs

There are peracute, acute, and chronic forms of most parasitisms. Death may be caused by overwhelming invasion of an organ or system, but usually parasitism results only in debilitation in varying degrees. Over a period of time, the body loses the ability to resist minor infectious agents, and a secondary infection may take the animal's life. Some degree of unthriftiness usually accompanies parasitism. The fiber coat may lack luster.

Emaciation may be seen in longstanding cases, a result of inappetence, leading to complete anorexia, combined with poor food utilization. Inappetence and poor food utilization also inhibit growth and maturation of parasitized young animals. Diarrhea is the most prominent sign of enteritis, but it is important to recognize that diarrhea need not always be present in parasitism, especially when larvae invade such tissues as the liver or lungs.

Anemia may be seen in heavy infestations, even with parasites that are not blood suckers. The cutting mouth parts used for attachment may result in leakage of plasma and cells from capillaries.

In adult animals, production and quality of fiber and milk are depressed. Thus, a baby may be doubly jeopardized, both by its own parasite load and lack of nourishment as a result of the effect of parasitism on the mother.

Diagnosis

The presence of one or more of the signs noted above should direct attention to a differential diagnosis, including parasitism. Unless adult parasites have already been seen in the feces, some type of fecal examination should be conducted to begin the process of diagnosis. Standard texts provide an explanation of the methodology for fecal examinations.[100]

Table 8.3 lists sizes of male and female adult parasites, larvae, and eggs and location of the internal parasites in the body.[21] Table 8.4 lists external parasites. Figures 8.34 to 8.37 illustrate the eggs for identification when recovered from the feces. Identification in this situation is possible only to the genus level and maybe only to the family. However, this usually suffices to indicate methods of management and therapy.

A direct smear is used as a quick preliminary procedure to determine the presence of nematode eggs, but it is even more important for detecting motile protozoan parasites such as giardia or trichomonas. The feces must be freshly passed so that the parasites are still alive and moving.

Various types of differential centrifugation or flotation are used to identify eggs, which are separated by the specific gravity unique to each parasite ova. Likewise, various counting procedures allow estimation of the parasite burden. Interpretation of these counts should be done by experienced people.

Special methods, such as a Baermann apparatus, are required for detection of parasites that pass larvae in

Table 8.3. Internal parasites of llamas.

Scientific name	*Common name*	*Size (mm)*				*Anatomic location*		*Intermediate host*	*Prepatent period (days)*
		Adult male	*Adult female*	*Larva (μm)*	*Egg (μm)*	*Adult*	*Immature*		
Fasciola hepatica	Liver fluke	13–30			130–150 × 63–90	Bile ducts	Small intestine, peritoneum, liver	Snail, *Lymnaea* sp.	56
Echinococcus granulosus	Hydatid	2–7			32–36 × 25–30	Intestine of carnivores	Lungs, liver	SAC (primary host is dog)	
Moniezia expansa	Tapeworm	600 cm × 1.6 cm wide			56–67	Small intestine	Small intestine	Oribatid mite	37–40
Thysaniezia spp.	Tapeworm	200 cm × 1.2 cm wide				Small intestine	Small intestine	?	
Trichostrongylus colubriformis	Stomach worm	4–5.5	5–7	620–790	79–101 × 39–47	Stomach, small intestine		None	20
Ostertagia ostertagi	Medium brown stomach worm	6.5–7.5	8.3–9.2	L-3 797–910	80–85 × 40–45	C-3		None	21
Ostertagia (Marshallagia) marshalli	Stomach worm	10–13	12–20		178–217 × 78–100	C-3, duodenum		None	
Camelostrongylus mentulatus	Stomach worm	6.5–7.5	8.3–9.2		75–85 × 40–50	C-3		None	
Bunostomum spp.	Hookworm	12–17	19–26	500–678	79–97 × 47–50	Small intestine		None	30–56
Skrjabinema ovis	Pinworm	2.3–3.7 × 110–180 μm	5–10 × 350–500 μm		47–63 × 27–36	Colon, rectum	Small intestine	None	17–25
Parelaphostrongylus tenuis	Meningeal worm		39–91	348		Central nervous system (may never mature in llama)	Small intestine, spinal cord		90
Thelazia californiensis	Eye worm	17 (11–19.5)				Conjunctival sac, lacrimal duct	Conjunctival sac, lacrimal duct	*Musca autumnalis*, face fly	
Gongylonema spp.	Cattle gullet worm	30–62 × 150–300 μm	80–145 × 300–500 μm		50–70 × 25–37	Esophagus, C-3	?	Beetles	?

Table 8.3. ***Continued***

Scientific name	*Common name*	*Size (mm)*				*Anatomic location*		*Intermediate host*	*Prepatent period (days)*
		Adult male	*Adult female*	*Larva (μm)*	*Egg (μm)*	*Adult*	*Immature*		
Trichuris spp.	Whipworm	50–80	35–70		70–80 × 30–42	Cecum, large intestine	Small intestine	None	28–35
Capillaria spp.		8–13	12–20		45–50 × 22–25	Small intestine		None	
Eimeria spp.	Coccidia					Small intestine			
Sarcocystis spp.	Sarcocyst					Muscle			
Toxoplasma gondii	Toxoplasma				(oocysts) 11–14 × 9–11	Sexual cycle in cats	Multiple organs	SAC	2–7 in cats
Haemonchus contortus	Large stomach worm	10–20	18–30	650–750	70–85 × 41–48	C-3	C-1	None	15
Cooperia mcmasteri		4.5–5.4	5.8–6.2	780		Small intestine		None	14–21
Nematodirus	Thread-necked strongyle	10–15	15–23	922–1120	175–260 × 106–110	Lumen of small intestine	Mucosa of small intestine	None	15
Graphinema aucheniae	Stomach worm	5.5–7.8	9–12		80–90 × 40–45	C-3		None	
Lamanema chavezi		10–13	14–18		150–170 × 70–80	Small intestine	Liver	None	
Spiculopteragia peruvianus	Stomach worm	6.7–7.7	8.4–10.3		81–95 × 45–49	C-3		None	
Dictyocaulus viviparus	Lung worm	40–55	60–80	L-1 300–360	82–88 × 33–38	Bronchi	Small intestine, mesenteric lymph node, thoracic duct	None	30
Oesophagostomum columbianum	Nodular worm	12–16.5	15–21.5	771–923	73–89 × 34–45	Small intestine, large intestine		None	41
Chabertia ovina	Strongyle	13–14	17–20	710–789	90–105 × 50–55	Large intestine	Small intestine	None	49

Table 8.4. External parasites of South American camelids.

Scientific name	*Common name*	*Size (mm)*			*Anatomic location*	
		Adult	*Larva*	*Pupa*	*Adult*	*Immature*
Culex, Anopheles Aedes	Mosquitos				Fiberless areas of head, neck, limb	Water
Vermipsylla spp., others	Fleas	1.5–4	<6	4 × 2	Same	Soil
Tabanus spp.	Horse fly Deer fly				Same	Same
Musca domestica	Housefly	6.5–7.5	10–12		Around eyes, ears, nostrils	Feces, organic debris
Musca autumnalis	Face fly	Slightly larger than housefly			Same	Cattle feces
Stomoxys calcitrans	Stable fly	11			Same	Same
Calliphoridae (various spp.)	Blow flies	8			Free-living flies	Infected and necrotic wounds
Oestrus ovis	Sheep botfly	12	L-1 = 2 L-3 = 30		Same	Nasal cavity, sinuses
Cephenemyia spp.	Deer nasobot fly	14	L-1 = 1–3		Same	Naso-pharynx
Otobius megnini	Spinose ear tick	8	2–3	Nymph 7–10	Around farm and ranch structures	External ear canal
Ixodidae (various spp.)	Hard ticks				Attached to body	Depends on species
Sarcoptes scabiei	Sarcoptic mange mite	0.3–0.5			In tunnels in epidermis	Same
Psoroptes bovis	Psoroptic mange mite	0.7–0.8			At periphery of scab	Same
Chorioptes ovis	Chorioptic mange mite	0.35–0.4	0.2		Same	Same

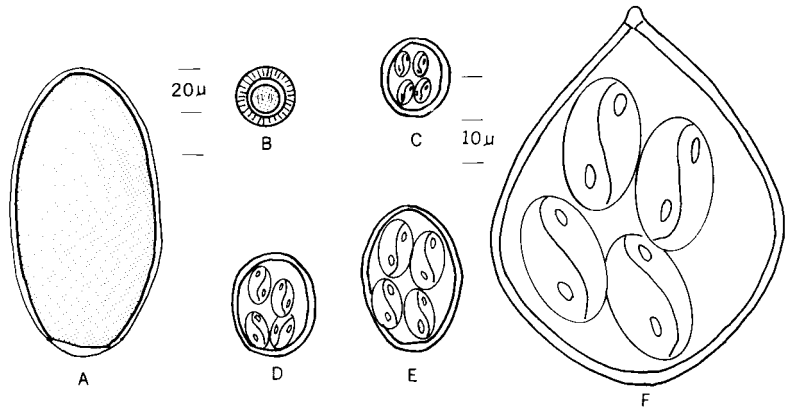

Figure 8.34. Miscellaneous parasite ova of camelids. (A) *Fasciola hepatica*, (B) *Echinococcus* spp., (C) *Eimeria punoensis*, (D) *Eimeria alpacae*, (E) *Eimeria lamae*, (F) *Eimeria macusaniensis*. (Eimeria scale = 10μ; others = 20μ).

the feces instead of eggs (*Dictyocaulus* spp.). A final diagnostic tool is response to treatment. The clinician may choose to treat on the basis of suspicion and previous experience.

Management and Treatment

The basic principles involved in the management and treatment of parasitism are identification of the parasite(s), at least to genus, and review of the life cycle. The same management procedures may be used as have been outlined for similar parasitism in ruminants. The nutritional status of the herd should be evaluated and appropriate changes made.

Numerous anthelmintics are safe and effective against GI nematodes in ruminants (Table 8.5).[1,52,53,106,108] All of these have been used in camelids, but the

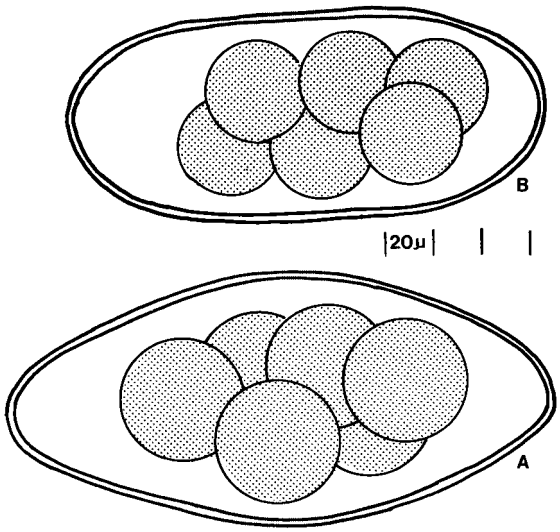

Figure 8.35. Nematode ova from camelids. (A) *Nematodirus* spp., (B) *Ostertagia (Marshallagia) Marshalli*.

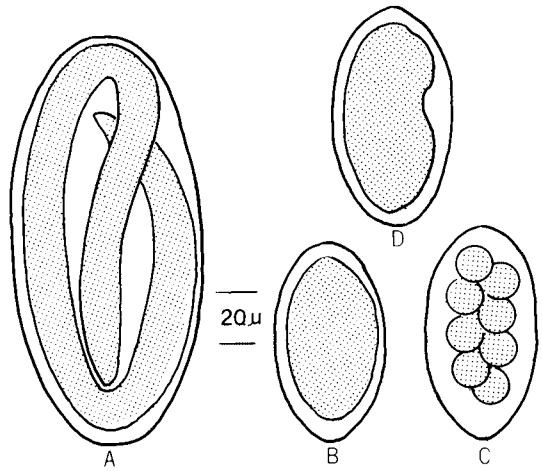

Figure 8.37. Nematode ova of South American parasites. (A) *Lamanema chavezi*, (B) *Camelostrongylus mentulatus*, (C) *Graphinema aucheniae*, (D) *Spiculopteragia peruvianus*.

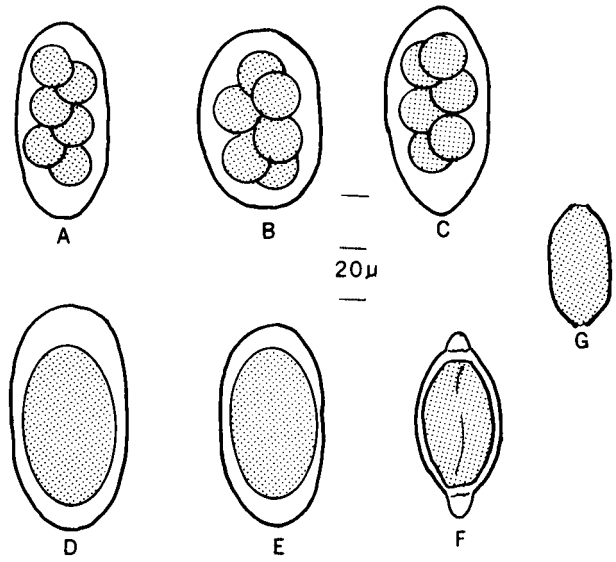

Figure 8.36. Nematode ova from camelids. (A) *Cooperia* spp., (B) *Haemonchus contorta*, (C) *Oesophagostomum columbianum*, (D) *Trichostrongylus* spp., (E) *Ostertagia ostertagi*, (F) *Trichuris ovis*, (G) *Capillaria* spp.

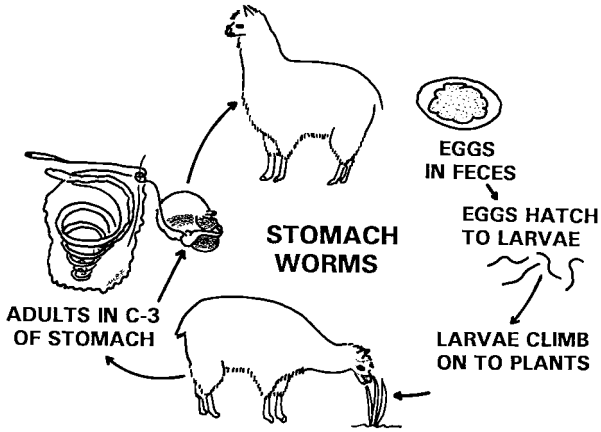

Figure 8.38. Life cycle of *Trichostrongylus* spp.

pharmacodynamics of anthelmintics in camelids is unknown. Until such information is available, the clinician should use ruminant doses and dosing intervals.

As in ruminants, local populations of parasites in camelids may develop resistance to an anthelmintic or class of anthelmintics. For instance, the failure of ivermectin to control previously sensitive parasites has been reported to the author by practitioner colleagues.

When an animal is suffering from severe dermatitis associated with external parasitism, chemicals are more likely to be absorbed through the damaged skin, which may lead to the development of toxicosis. It is suggested that such animals be treated in stages.

A final management recommendation is that fecal samples be monitored semi-annually to evaluate the effectiveness of chemotherapy. The examination should coincide with known exacerbation of parasite loads for the area.

Trichostrongylus spp.,[23] *T. axei*,[12,64,86] *T. vitrinus*,[64] *T. colubriformis*,[12,64] *T. longispicularis*[41]

LIFE CYCLE.[100] A generalized life cycle for *Trichostrongylus* spp. is as follows (Figure 8.38). The female pro-

Table 8.5. Anthelmintics used in camelids.

Anthelmintic		*Dose mg/kg*		*Route of administration*
Generic name	*Trade name in US*	*NWC*	*OWC*	
Albendazole	Valbazen	10.0 15.0 for *Fasciola magna*	5.0–7.5	Oral
Amprolium	Corid	5	?	Orally × 21
Chlorsulon	Curatrem	7	?	Oral
Decoguinate	Deccox	0.5	?	Orally × 28
Fenbendazole	Panacur	5–10 15 for *Trichuris*		Oral
Oxfenbendazole	Oxfenbendazole	5	5	Oral
Levamisole	Levasole	5–8	7.5	Oral
Thiabendazole	Equizole	100	100–150	Orally × 1–3
Mebendazole	Telmin	22	5	Orally × 3
Pyrantel pamoate	Strongid	18	25	Orally × 3
Ivermectin	Ivomec	0.2 0.4–0.6 for *Trichuris* and *Cephenemyia*	0.2	Oral or SC
Doramectin	Dectomax	0.2	0.2	SC or IM
Moxidectin	Cydectin	0.4	0.4	Oral
Toltrazuril	Baycox	20	20	Oral; no data for use in camelids
Diclazuril	Decocci	1	1	IM, SC; no data for use in camelids
Metronidaxole	Flagyl	25 Twice a day		Orally × 5

Data from Richard 1992, Bornstein 2002, and personal experience.
Caution! Do not use ionophore coccidiostats (monensin salinomycin) in camelids.

duces eggs containing embryos in the morula stage. The eggs are passed in the feces, and the morula mature to become first-stage (L-1) larvae within the egg case. Under ideal conditions, hatching occurs in one to two days. The free-living L-1 larvae feed on microorganisms in the feces and molt to L-2 and again to L-3, which is the infective stage, taking four to six days.

L-3 larvae migrate out of the feces in about one week and climb onto vegetation. The life cycle is direct, and the camelid ingests grass containing L-3 larvae, which mature through L-4 and L-5 to become adults in the third compartment (C-3) of the stomach or the intestine. The prepatent period is about twenty days.

EPIDEMIOLOGY. The length of time necessary for maturation of free-living larvae depends on climate, season, and temperature. Some larvae will survive a mild winter in protected areas. Drought and desiccation are detrimental to the survival of most larvae, but some species have evolved in such environments and have developed adaptations for survival.

The clinician faced with a trichostrongyle problem in a group of camelids should review one of the standard texts on the subject in ruminants and apply the same principles.

Ostertagia ostertagi,[12,55,64] *O. circumcincta*,[12,64] *O. lyrata*[12,41]

LIFE CYCLE. There are two phases or types of cycles in these species (Figure 8.39). Type I is typical of a trichostrongyle life cycle, with larvae maturing to adults without passing through a developmental arrest stage. Type II involves developmental arrest in the mucosa of the stomach. Larvae become arrested in early fall and begin development again in winter in northern temperate regions. In southern temperate regions with dry summers and winter rainfall, arrest occurs in late winter and spring, and development begins again in late summer and fall. Larvae in the glands of the wall of the stomach stimulate formation of grayish-white nodules (Figure 8.39). Maturation of previously arrested larvae can cause a buildup of pathogenic adults, which, in turn, cause gastritis and heavy egg levels in the feces.

EPIDEMIOLOGY. The life cycle of *O. ostertagi* may vary according to the climate and species affected, i.e., cattle, sheep, or SACs. The cycle described for cattle in a given area may not be the cycle that occurs in cattle in another area and is even less likely to be the cycle followed in llamas in yet a third area or another country. Until information has been obtained by

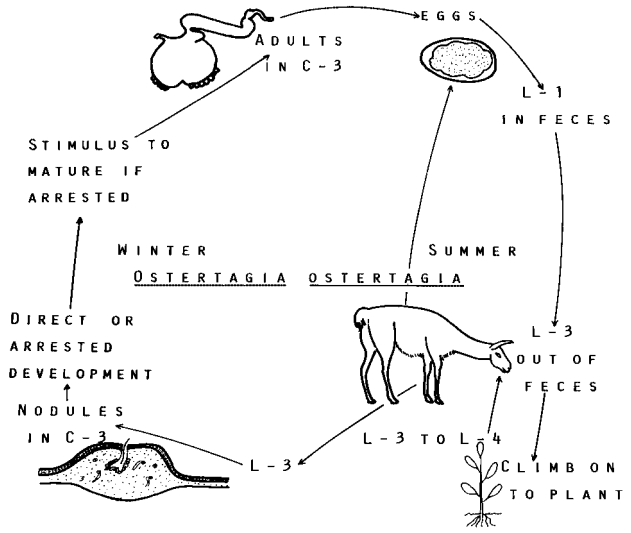

Figure 8.39. Life cycle of *Ostertagia ostertagi.*

detailed studies, however, reliance must be placed upon information that is known of ruminants of a given area.

Marshallagia (Ostertagia) marshalli[12,64]

LIFE CYCLE. This species has been shifted back and forth from its own generic status to that of a species of *Ostertagia*. It is closely related to *Ostertagia* and has a similar life cycle. When ingested, the larvae penetrate into the gastric mucosa of C-3 and produce a nodule that is 2 to 4 mm in diameter. Instead of one larva per nodule, as in ostertagiosis, each nodule contains two or three larvae that will mature in fifteen to eighteen days. The prepatent period lasts for up to three weeks, but there may also be arrested development in this species. The eggs of this parasite are large and frequently confused with that of *Nematodirus* spp. (Table 8.3).

EPIDEMIOLOGY. Marshallagia has a limited distribution, but pockets of infection are located in the western United States. It has been seen in llama herds in northern California.

Teladorsagia spp.

Teladorsagia spp. is an *Ostertagia*-like nematode that is usually found only in sheep.[41] This parasite was found in the third compartment of the stomach in a llama that was necropsied for other reasons. The llama had been pastured with sheep. Nodules were found in the stomach that had the gross and histologic appearance of pre-Type II ostertagiosis. No adults were observed, and it is not known whether or not the larvae would resume development and produce clinical disease.

Camelostrongylus Mentulatus[12,64,65]

This is another species that is closely related to *Ostertagia*. It is a common stomach (C-3) parasite of camels in the Middle East, but it is also found in Australia, South America, and the United States.[73] The life cycle and epidemiologic patterns are similar to those of *Ostertagia*.

Graphinema aucheniae[30,39]

This trichostrongyle has been reported only from South America. Adult parasites are found in C-3. The life cycle and epidemiology are similar to those of other trichostrongyles.

Haemonchus contortus[30,41,64]

LIFE CYCLE. This parasite is similar to other trichostrongyles in the preparasitic stage. Infective larvae are present four to six days after the eggs have been passed in the feces. Eggs in the prehatch stage are more resistant to freezing and desiccation than the larvae, but all stages of the eggs and larvae can tolerate some degree of desiccation and low temperature.

Once the infective larvae have been ingested, they may enter an arrested development stage resembling that of *Ostertagia*. The nodules in C-3 are not so pronounced as in ostertagiosis. Redevelopment begins when environmental conditions suitable for preservation of the eggs and larvae in the free-living state occur.

EPIDEMIOLOGY. In ruminants there is a breed-associated resistance to infection with *H. contortus*. Whether or not camelids may be resistant is unknown. Other species of *Haemonchus* may have slightly different cycles and be adapted to either hot and dry climates or moist and cool climates. Infection in ruminants may stimulate an immune response. Conversely, animals that are stressed have lowered immunity, and infection and disease may be more debilitating.

A preparturient relaxing of immunity that may be accompanied by a buildup of the parasite load in a camelid has been reported by Soulsby.[112] Any period of stress should be monitored for the possibility of parasitism override.

CLINICAL SIGNS. *H. contortus* differs from other trichostrongyles in that it is a blood sucker. The trauma to the mucosa produced by this parasite also allows seepage of blood into the lumen of the intestine. The amount of blood loss caused by each nematode has been calculated to be 0.05 ml/day.[112] Blood may appear in the feces six to twelve days following infection.

In addition to erythrocyte loss, there is a significant loss of plasma protein through such seepage. Some compensation for the protein loss may be achieved by increased albumin synthesis, but ultimately,

protein reserves are depleted and hypoalbuminemia develops.

There are hyperacute, acute, and chronic forms of haemonchiasis. The chronic form is the more important in camelids; affected animals become unthrifty, weak, and emaciated. The fecal egg count cannot be used as a gauge of the severity of the disease. Neither can the degree of anemia or hypoproteinemia, since the animal may have the capacity to compensate for the loss of cells and plasma as described above.

Lamanema chavezi[5,47,50,51,98,102]

IDENTIFICATION. *Lamanema chavezi* is one of the more important parasites of camelids in the Andes. Fortunately, no instances of parasitism outside of South America have been reported, but camelids being imported from South America should be carefully examined. The first description of this parasite in SACs was based on adult male nematodes.[5] A redescription of the egg and adult male and female parasite was made by Sutton[102] in Argentina, from specimens recovered from the mountain viscacha (*Lagidium viscacia boxi*) (Table 8.3).[5,35] Considering that the syndrome of parasitism in SACs is so severe, it is highly likely that the definitive host is the viscacha and the alpaca or llama is an aberrant host.

LIFE CYCLE. The life cycle of *Lamanema chavezi* is not well understood (Figure 8.40). The adults are found in the small intestine rather than in C-3. The stages, up to ingestion of infected larvae, are probably similar to those of other trichostrongyles. Ingested larvae penetrate the intestinal wall and migrate to the liver and lungs. It is in the liver that the most serious damage occurs. Ultimately, maturation is completed with migration back to the small intestine via the trachea.

EPIDEMIOLOGY. This is a dangerous parasite for recently weaned camelids. In one study, four-month-old alpacas were given 200,000 larvae. Some alpacas began dying within twenty days. Because of the severe effect of this parasite on camelids, it is likely that camelids are aberrant hosts.

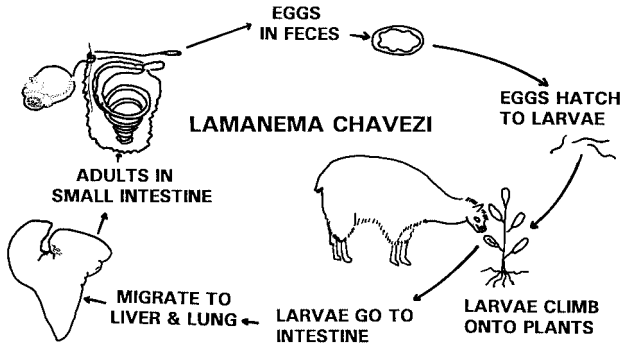

Figure 8.40. Life cycle of *Lamanema chavezi.*

CLINICAL SIGNS. Massive infections cause hepatic and respiratory failure from involvement of the liver and lungs.

PATHOLOGY. The migration of the larvae through the wall of the intestine produces a catarrhal, hemorrhagic enteritis, with areas of necrosis of the mucosa. In acute cases, with recent migrations, the liver is congested, with multiple foci of coagulative necrosis, petechial hemorrhage, and punctate abscessation. There are also areas of congestion in the lungs.

Following maturation and remigration of the larvae, the liver lesions become fibrotic and may calcify. These residual foci have been seen in adult alpacas slaughtered for meat.

Spiculopteragia peruvianus[30,41]

IDENTIFICATION. This trichostrongyle nematode is found in C-3 of llamas, alpacas, and vicuñas on the Altiplano, near Lake Titicaca, in Peru. Little is known about the biology of this parasite other than its description (Table 8.3). It was mistaken for other trichostrongyles for a long time.[41]

Nematodirus lamae,[5] *N. battus*,[7,64] *N. spathiger*,[12,41] *N. filicollis*,[64] *N. lanceolatus*,[64] *Nematodirus* spp.[3,40]

LIFE CYCLE.[99] Adult nematodes are located in the small intestine. Eggs are passed in the feces and undergo slow development (over two or three months) to L-3 larvae. If eggs at this stage are ingested in late summer, the larvae are infective, but this is not the normal route of exposure. Rather, the larvated eggs usually overwinter, and cold temperatures are required to ultimately induce the eggs to hatch and release the larvae. In the spring, when soil temperatures begin to rise, the mobile larvae climb onto herbage and are ingested.

Following ingestion, infective L-3 larvae penetrate the intestinal mucosa, where they molt to L-4 and L-5. L-5 larvae leave the mucosa and become adults in the lumen of the small intestine. The prepatent period is fifteen days under optimum circumstances. Adult parasites live for only a few weeks. Diagnosis of parasitism is based on observation of the ova on fecal flotation and descriptions of the adults.[82]

EPIDEMIOLOGY. Because the larvated eggs overwinter, an infection in a given year depends on contamination of a pasture the previous year. The major disease problem is in young animals with no prior exposure to the parasite because animals develop some degree of resistance to the parasite over a period of time. A few older juveniles and adults harbor some parasites and contribute to contamination of the pastures, but overt disease is usually not seen in adults.

The degree of severity of the problem depends on the timing of the birth of the baby and the hatching of the eggs to free the infective larvae. Once freed, larvae

live for only a few weeks. Species differences may also account for variation in pathogenicity. *N. battus* is infrequently encountered, but may cause serious enteritis.

CLINICAL SIGNS. Most of the damage occurs as the larvae embed in the intestinal mucosa, causing enteritis with its characteristic syndrome.

Cooperia zurnabada (*C. mcmasteri*),[30,57] *C. oncophora*[9,38,71]

Cooperids are small trichostrongyles living in the small intestine of ruminants and camelids throughout the world. The life cycle and epidemiology are similar to those of *Trichostrongylus* spp. The infective larvae are able to survive on pastures nine to twenty-six weeks and even overwinter. There may be an arrested development stage in the wall of the intestine.[100] Peruvian workers believe that *Cooperia zurnabada* and *C. mcmasteri* are separate species, based on size differences.[37]

Dictyocaulus filaria,[43,66] *D. viviparus*,[19] *Dictyocaulus* spp.[57]

IDENTIFICATION. These lungworms are included in the superfamily Trichostrongyloidea because the life cycle is direct. See Table 8.3 for measurements.[45]

LIFE CYCLE.[100] Adult parasites live in the lumen of the bronchial tree (Figure 8.41). The female lays a larvated egg, which is coughed up and swallowed. Hatching usually occurs during passage through the digestive tract. The free-living larvae, unlike other trichostrongyles, live on stored nutrients rather than on microorganisms. The larvae develop from L-1 to the infective L-3 in five days or more. Larvae then climb onto herbage and are ingested. The larvae penetrate the wall of the small intestine and migrate to the mesenteric lymph nodes and thence, via the blood vessels or lymphatics, to the lungs. About four weeks are required for maturation of L-5 to the adult nematode. The prepatent period is four to five weeks.

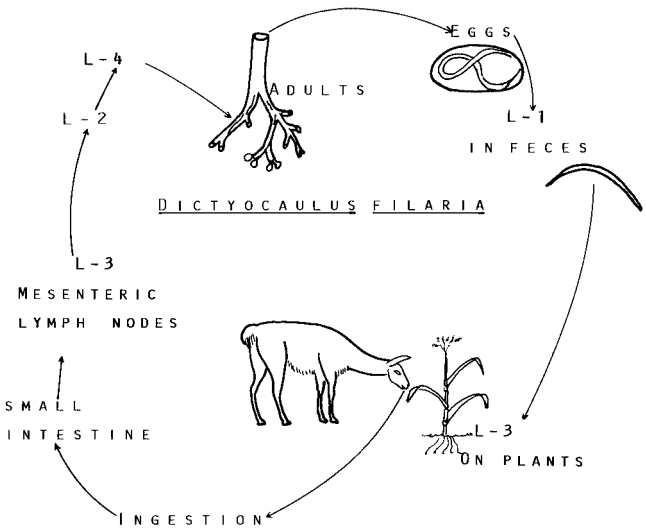

Figure 8.41. Life cycle of *Dictyocaulus filaria.*

EPIDEMIOLOGY. Larvae require a moist environment, and therefore are not a problem in hot, dry regions. Infective larvae can overwinter, but the more serious problem seems to arise in the fall, after a summer buildup of infection. The larvae are not usually evident on a standard fecal flotation. A Baermann apparatus is necessary to evaluate feces for lungworm larvae. It is also important to remember that clinical disease may be seen before larvae appear in the feces. Larvae developing in the lungs produce a marked catarrhal bronchitis. Although this is a common parasite of SACs in South America, it is rarely encountered in the United States.

CLINICAL SIGNS. The cardinal signs are cough, dyspnea, and nasal exudate. Not all of the signs need to be present at the same time. The body temperature is normal unless a secondary pneumonia develops.

Oesophagostomum spp.,[3] *O. venulosum*[64]

IDENTIFICATION. The nodular worm (*Oesophagostomum venulosum*) is a strongyloid nematode found in the small and large intestines of camels and ruminants. None of the parasites found in SACs have been identified as to species. A similar strongyloid, found in sheep, is *Chabertia ovina*, which has also been reported in camels and in the vicuña in South America. See Table 8.3 for measurements.

LIFE CYCLE.[100] The life cycle of camelid oesophagostomums has not been established. Based on *O. columbianum* or *O. venulosum*, it is probably as follows. The thin-walled eggs are passed in the feces and require six to seven days to reach the infective larval stage. The larvae climb onto herbage, are ingested, and exsheath in the small intestine. Larvae penetrate the wall of the intestine according to species predilection and may or may not produce nodules. L-4 and L-5 maturation takes place in the wall of the intestine; then the larvae migrate back to the lumen and become adults, taking up residence in the large intestine. The prepatent period of *O. columbianum* is forty-one days, and twenty-one to thirty-eight days for *O. venulosum*. *O. venulosum* has less tendency to form nodules and thus is less pathogenic.

EPIDEMIOLOGY. Nothing is known about this parasitism in SACs.

Bunostomum spp.

IDENTIFICATION. *Bunostomum* are hookworms that are rare in camelids; they are found only in SACs living in warm, tropical climates. See Table 8.3 for measurements.

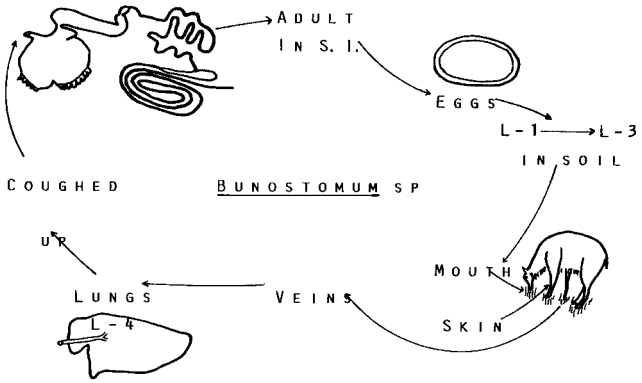

Figure 8.42. Life cycle of *Bunostomum* spp.

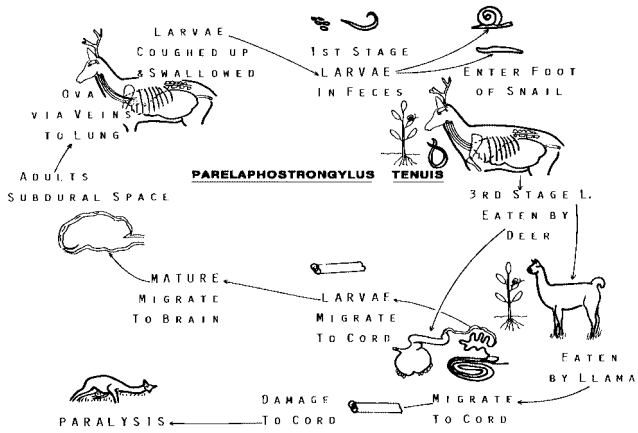

Figure 8.43. Life cycle of *Parelaphostrongylus tenuis.*

LIFE CYCLE.[100] The life cycle is direct (Figure 8.42). Adults are attached to the mucosa of the small intestine and are blood suckers. The eggs require a few days after passage in the feces before infective larvae are produced. These larvae may enter the body via the mouth or through the skin. If via the skin, the larvae migrate to the lung via the venous or lymphatic vessels and mature to L-3. These are coughed up and swallowed. L-4 larvae migrate to the intestine. The prepatent period is thirty to fifty-six days.

EPIDEMIOLOGY. Infective larvae are susceptible to desiccation, so this parasite is a problem only where there is permanent moisture or high humidity. There is no encystment in the muscles nor is there transmammary migration as occurs in ancylostomiasis.

Skrjabinema ovis[64]

IDENTIFICATION. This parasite is the sheep pinworm. It has been reported in the guanaco in Argentina. The males are 2.3 to 3.7 mm long and 110 to 180 µm wide, making them wider than most other GI nematodes.[77] The eggs are slightly flattened on one side and are 47 to 63 × 27 to 36 µm.

LIFE CYCLE. The life cycle is direct. The adults live in the rectum, but the female traverses the anal sphincter to deposit eggs around the anus at night.[67] Infective larvae develop within the eggs. The eggs drop off and contaminate feed and water. When ingested, eggs hatch in the small intestine, and the larvae migrate to the large intestine where they become mature worms in seventeen to twenty-five days.

EPIDEMIOLOGY. The parasite probably does little harm to the host. Nothing is known about environmental requirements for persistence of the eggs.[67]

CLINICAL SIGNS. Clinical signs include possible anal pruritus. Eggs will not usually be seen in a fecal flotation. Scotch tape applied to the anus and then applied to a glass slide is the customary diagnostic technique.

Parelaphostrongylus tenuis[32]

IDENTIFICATION. The meningeal worm, *Parelapho strongylus tenuis* (*Pneumonstrongylus tenuis*), is in the Protostrongylidae family. The white-tailed deer (*Odocoileus virginianus*), is the primary host for this parasite. See Table 8.3 for measurements.[4,10,20] Canadian authorities tried to make a case for considering *Elaphostrongylus cervi* as a potential parasite in alpacas because of its taxonomic relationship with *P. tenuis*, but *E. cervi* has never been found in any noncervid host, and there is absolutely no reason to even remotely consider it as a camelid parasite.

LIFE CYCLE.[20] The life cycle in the deer is described first (Figure 8.43). It is not known whether the cycle is completed in the llama. Adult nematodes are located in the veins and sinuses of the dura mater of the brain. The eggs are deposited there and may or may not hatch. Eggs and larvae pass via the blood vessels to the lungs, and L-1 larvae break out into the alveoli. The larvae are then coughed up, swallowed, and passed in the feces. The larvae are picked up by terrestrial snails and slugs, *Deroceras graciles, D. reticulatum, D. laeve, Cionella lubrica, Arion circumscriptus*, *Zonitoides nitidus*, or *Z. arboreus*.

Development continues within the snail for three to four weeks. The snail or slug is then ingested by the deer. Larvae are released in the stomach, penetrate the wall of the peritoneal cavity, and migrate to the spinal cord, usually via the spinal nerves, within ten days. The larvae mature in the dorsal horns of the gray matter for twenty to thirty days. The adults then move to the spinal subdural space and migrate to the brain. They move into their ultimate location by penetrating the dura mater and passing into the venous sinuses. The prepatent period is eighty-two to ninety-one days.[112]

EPIDEMIOLOGY. The camelid is an aberrant host for this parasite. A number of instances of affected llamas have been reported.[4,10,32,62,72,76,79,80,89,91] The white-tailed deer

has a broad distribution in the southern, eastern, and northern United States. The parasite is generally found wherever deer are found. Llamas cohabiting in pastures with white-tailed deer are at risk. It should be mentioned that other ungulate species may also be aberrant hosts for this parasite, e.g., moose (*Alces alces*), wapiti (*Cervus canadensis*), caribou (*Rangifer tarandus*), black-tailed deer (*O. hemionus*), red deer (*Cervus elaphus*), and sheep and goats.

CLINICAL SIGNS. The parasite is well adapted to the primary host, in which little or no clinical disease develops. In aberrant hosts, such as the llama, migration of the larvae in the spinal cord produces neurologic deficits commensurate with the location of the larvae, including lameness, ataxia (Figure 8.44), stiffness, circling, blindness, hypermetria, paraplegia, paralysis, and abnormal positions of the head.

DIAGNOSIS. No definitive antemortem diagnosis is possible. There may or may not be a peripheral eosinophilia or increased eosinophils in the cerebrospinal fluid. One report indicates an increased eosinophil concentration in cerebrospinal fluid.[72]

TREATMENT. No definitive treatment and preventive programs have been established. Contact camelid veterinarians for the latest recommendation. The development of anthelminic resistance has complicated therapy considerably. Ivermectin is effective against the stages prior to entering the spinal cord. Ivermectin does not readily penetrate the intact blood-brain barrier, but some clinicians report improvement and explain the results on the basis that penetration of the larvae through the meninges opens the blood-brain barrier. A few management procedures may be helpful in protecting valuable camelids from this parasite. A deer-proof fence could exclude white-tailed deer from camelid pastures. A molluscicide could be used to destroy the snails and slugs that serve as intermediate hosts, and monthly treatment with ivermectin could be established during the spring, summer, and fall.

Figure 8.44. Posterior ataxia from meningeal worm.

Angiostrongylus cantonensis

An alpaca in quarantine, preparatory to importation to Australia, died, and at necropsy *Angiostrongylus cantonensis* nematodes were found in the lungs. This species is a lungworm of rats in the Pacific Basin. The alpaca is an aberrant host.

Thelazia californiensis, Thelazia spp.

IDENTIFICATION.[100] The eyeworm, *Thelazia californiensis*, is a spirurid nematode found in the conjunctival sac of deer, elk, cattle, sheep, dogs, cats, foxes, rabbits, llamas, and humans. See Table 8.3 for measurements.

Larvated ova are found in the lacrimal secretions (Figure 8.45). Muscoid flies, such as the face fly (*Musca autumnalis*), ingest the ova or larvae. L-2 larvae migrate to the fly's ovarian follicles and mature to L-3 in fifteen to thirty days. L-3 larvae migrate from the follicles to the labia of the fly, and infective larvae are deposited in another eye when the fly feeds on lacrimal secretions. Maturation to the adult nematode takes sixteen to twenty days, the prepatent period.

EPIDEMIOLOGY. The life cycle also describes the epidemiology. The parasite is locally common and is transmitted only during the fly season.

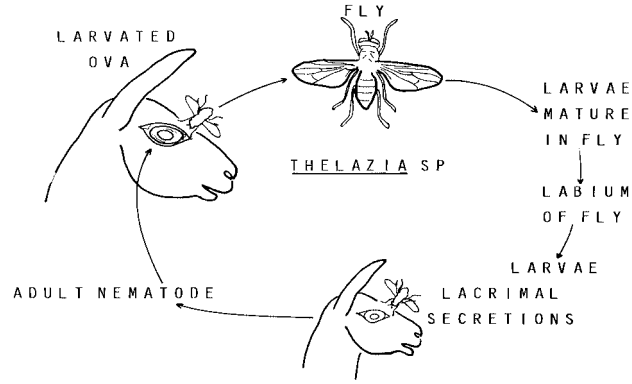

Figure 8.45. Life cycle of *Thelazia californiensis.*

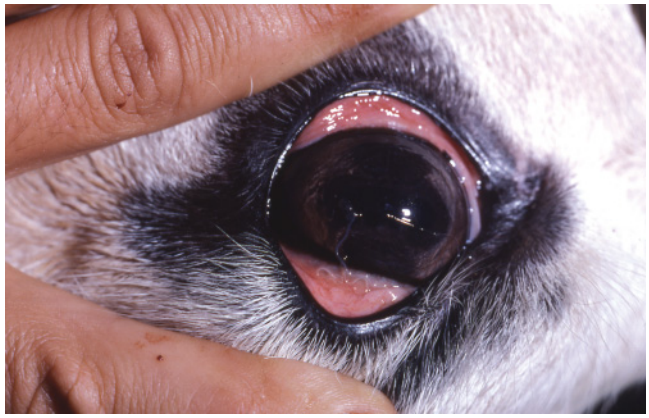

Figure 8.46. *T. californiensis*, in conjunctival sac.

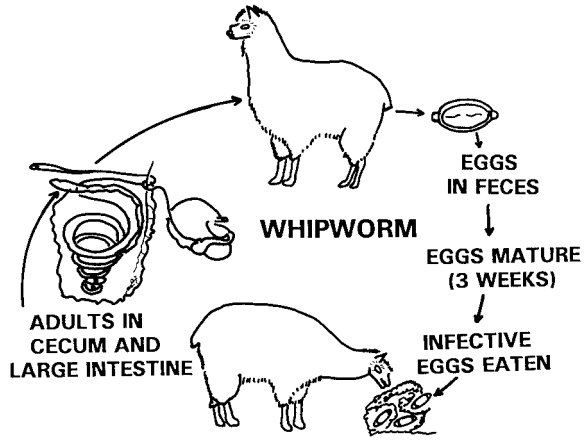

Figure 8.47. Life cycle of *Trichuris tenuis*.

CLINICAL SIGNS. Excessive lacrimation may be the only sign noted. Nematodes may be seen on the surface of the cornea or in the conjunctival sac (Figure 8.46). They may lodge beneath the nictitating membrane or be found in the nasolacrimal duct. With large numbers, a mild conjunctivitis may develop.

TREATMENT. The parasites can be mechanically removed under sedation with a local anesthetic. Diethylcarbamazine in a concentration of 2 mg/L may be instilled into the conjunctival sac. Ivermectin drops also may be instilled into the conjunctival sac.

Gongylonema spp.

IDENTIFICATION. *Gongylonema pulchrum* has been reported from alpacas in Peru.[12] This is a spirurid nematode. *G. pulchrum* has a broad host distribution and is called the "cattle gullet worm." No speciation was done on specimens collected from the alpacas. Male *G. pulchrum* are 30 to 62 mm long and 150 to 300 µm wide; females are 80 to 145 mm long and 300 to 500 µm wide. The eggs are 50 to 70 × 25–37 µm.[67]

LIFE CYCLE. The life cycle requires an intermediate host. The adults are embedded in the mucosa or submucosa of the esophagus and stomach. Eggs are passed in the feces and ingested by beetles. The eggs hatch and mature to infective larvae in about four weeks.[67] The camelid must then ingest the beetle to complete the cycle. The migration route of the larvae to the esophagus is not known.

EPIDEMIOLOGY. Unknown, other than that beetles serve as intermediate hosts.

CLINICAL SIGNS. These parasites are nonpathogenic to cattle, and no reports of signs or pathology in camelids have been published.

Trichuris tenuis,[3,49] *T. ovis*[12,16,84]

IDENTIFICATION. Whipworms (*Trichuris* spp.) are significant parasites of camelids and are resistant to treatment. They are characterized by a long, slender anterior and a thicker posterior segment. See Table 8.3 for measurements. The eggs are doubly operculated and thus are easy to identify, but they must be differentiated from those of *Capillaria* spp. Adult parasites are found in the cecum and large intestine. Trichurids identified from camels include *T. globulosa, T. cameli, and T. skrjabini.*[99]

LIFE CYCLE. South American parasitologists report that *T. ovis* is the species affecting SACs. *T. tenuis* has been found more frequently in the Pacific northwest.[49] However, trichurids all have a similar cycle, which is direct (Figure 8.47). Eggs are passed in the feces, requiring about three weeks to become infective. The infective eggs are ingested by the camelid and hatch. Larvae penetrate the wall of the anterior small intestine. Maturation proceeds for two to ten days, and the larvae migrate to the cecum and large intestine and become adults.[99] The prepatent period for *T. ovis* is seven to nine weeks.

EPIDEMIOLOGY. The persistence of infective eggs in the environment makes management of this parasitism difficult.

CLINICAL SIGNS. These nematodes tunnel into the intestinal mucosa with the slender anterior ends (Figure 8.48A and 8.48B). They traumatize vessels, producing a catarrhal enteritis and causing hemorrhage, which is ingested by the parasites. The syndrome is similar to that of haemonchiasis.

Capillaria spp.[12,64]

IDENTIFICATION. Capillarids are closely related to *Trichuris* but lack the slender and thick variation of the body. Little is known about the species found in SACs, but eggs identified in the laboratory are presumed to be identical to species found in ruminants.

LIFE CYCLE. Not known.

EPIDEMIOLOGY. Not known.

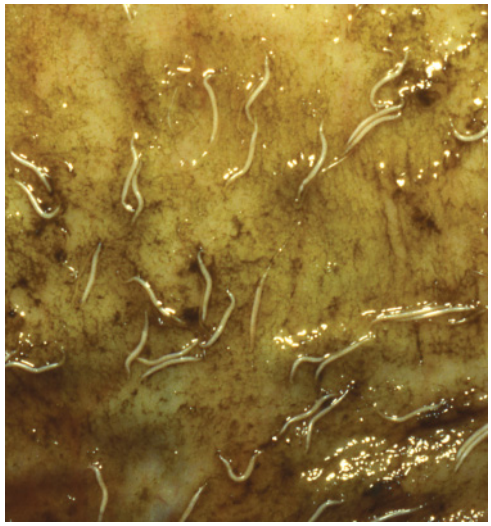

Figure 8.48A. Whipworms in the colon of a llama. Ingesta washed away.

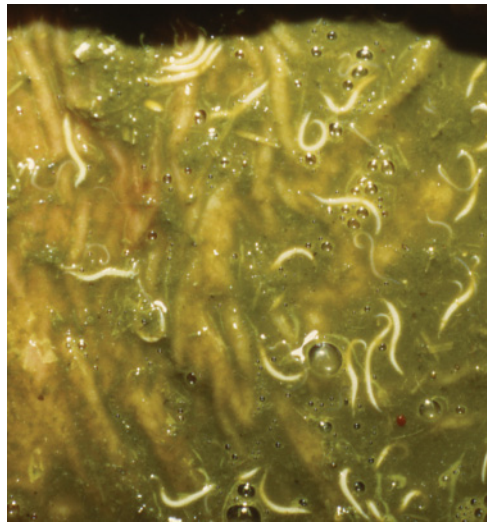

Figure 8.48B. Ingesta surrounding parasites.

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[d] W. Bravo, personal communication, 1986.

9

Multisystem Disorders

This chapter deals with noninfectious and nonparasitic disorders affecting multiple organ systems.

NEOPLASIA

Although relatively few tumors have been reported in camelids, the basic premise is that if a determined search were made through a sufficient population of camelids, neoplasia would be noted in all organ systems.[1,5,18,20,23,29,38,58,59] The more thorough the necropsy, the more likely the detection of a lesion. Many necropsies of camelids are conducted by people without specialized training in pathology. Therefore, many subtle lesions may go unnoticed, hence unreported. This is especially true of neoplasia that may be incidental to the actual cause of death.

The etiology of neoplasia in camelids is unknown, as it is for most tumors of domestic and wild animals.

Lymphosarcoma was diagnosed in a four-year-old breeding male llama,[25] kept with approximately thirty other llamas. Clinical signs included self-imposed isolation from the herd, anorexia, emaciation, weakness, stumbling gait, stomach atony, and dyspnea. Thoracic radiographs indicated pleural effusion. Cytologic examination of the sterile serosanguineous fluid from the pleural space demonstrated large numbers of immature, pleomorphic, nondifferentiated lymphoid-type cells. Similar cells were observed from a bone marrow aspiration.

The llama was euthanized and necropsied. A large lymphoma ($10 \times 15 \times 20$ cm) was found in the region of the pancreas, duodenum, and adjacent loops of colon (Figure 9.1A). The liver was infiltrated. There was fibrinous pleuritis, with lymphomas on the parietal pleural surfaces, within the lung, and in mediastinal lymph nodes. Both external and internal lymph nodes were enlarged. A nodule was found in a kidney. The microscopic lesions were similar to those observed in other domestic animals with lymphosarcoma. The primary nidus was not determined.

Carcinomas may occur in numerous organs and tissues. Carcinoma of the larynx and trachea is illustrated in Figure 9.1B.

In another llama, a mass on the caudal ventral aspect of the right mandible was noted to enlarge rapidly, accompanied by a significant weight loss. Bronchial lymph nodes and the ventral lungs were invaded by tumor cells. Approximately 2,800 ml of serosanguinous fluids containing tumor cells were aspirated from the pleural spaces.

Lymphoid tissue tumors reported in the literature since the second edition include multicentric T-cell lymphoma in an alpaca,[53] B-cell lymphosarcoma,[43] acute myeloid leukemia in an alpaca,[57] and lymphosarcoma.[31] Tumors of the integumentary system include basal cell carcinoma in a camel,[2] mammary carcinoma,[4] multiple trichoepethelioma of the eyelid, cutaneous melanocytoma, a metastatic malignant melanoma, and a cutaneous carcinoma.[41] Tumors of the musculoskeletal system include rhabdomyocarcinoma in a camel,[64] osteogenic melanoma, ossifying fihroma,[33] vertebral osteoma, and an amelanoblastic odotoma.

Newly reported tumors of the respiratory system are metastatic carcinoma and pulmonary neoplasia. A urinary tract tumor is a urethral sarcoma.

Other tumors have been observed in llamas and reported by clinicians at seminars and conferences.

CONGENITAL DISORDERS

Congenital defects may be observed in single or multiple organ systems.[5,9] See Chapter 22 for details.

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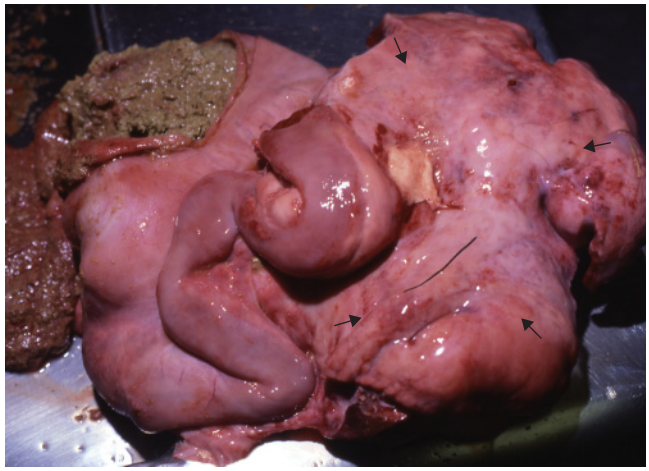

Figure 9.1A. Lymphosarcoma of abdominal organs.

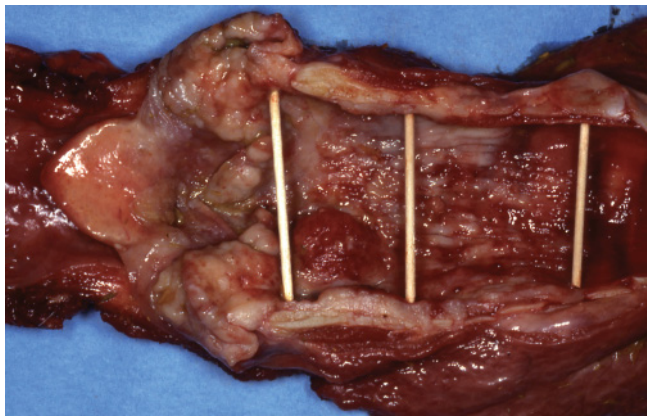

Figure 9.1B. Carcinoma of the larynx and trachea.

STRESS

Definition of Terms

Stress is the cumulative response of an animal to interaction with its environment via receptors.[11,12,50–52] Stress is an adaptive phenomenon. All responses are primarily directed at coping with environmental change. Each reaction to a stressor has adaptive significance. Intense or prolonged stimulation induces detrimental responses that may be fatal in the camelid.

A "stressor" (stress-producing factor) is any stimulus that elicits a nonspecific response when perceived by an animal. Potential somatic stressors (stimulation of physical senses) acting on camelids include temperature changes; strange sights, sounds, touches, or odors; stretching of muscles during restraint procedures; close confinement; thirst; and hunger.

Psychological stressors include anxiety, fright, terror, anger, rage, and frustration. Closely allied are behavioral stressors, including overcrowding, lack of social contact, hierarchial upsets, unfamiliar surroundings, trailer transport, and lack of habitual foods. It is becoming more and more important to recognize that stimulation of visual and auditory senses have a marked bearing on accumulative stress. These stressors may operate prior to, during, and after restraint and may lack adaptive context.

Miscellaneous stressors include malnutrition, toxins, parasites, infectious agents, burns, surgery, drugs, chemical and physical immobilization, and confinement.

Selye described responses as either specific or nonspecific.[44] Modern interpretation makes no distinction between these types of responses because there is marked species variation in how organisms process and act upon stimuli.[35–37,61] There may even be varying responses within an individual, depending upon which stimuli are acting upon it at a given time (experience, adaptation, hierarchial status, nutrition).

Animals respond in appropriate ways to stimulation of specific receptors. For instance, when cold receptors are stimulated, the body experiences a sensation of coolness. Various somatic and behavioral changes occur that conserve heat and stimulate increased heat production. The animal is adjusting to a new situation (homeostatic accommodation).

Other responses may be mediated through the autonomic nervous system and neuroendocrine pathways.

Prolonged and intense stimulation by stressors may overwhelm the body's ability to respond in a normal manner. Some neuroendocrine responses may produce reactions that are not evident immediately, but effects of stressors may be cumulative and potentially detrimental to an animal.

Body Response to Stress Stimulation

Response to the stimulation of a receptor may follow one of three pathways: voluntary motor, autonomic (usually adrenal medulla), and neuroendocrine (e.g., hypothalamic adenohypophyseal adrenal, or hypothalamic adenohypophyseal gonadal)[11] (Figure 9.2). The alarm response is described in Table 9.1 and illustrated in Figure 9.3.

Selye's concept of cortisol being responsible for most of the nonspecific reactions held sway for a number of years, but modern researchers of the stress phenomenon have discarded that concept for the broader view that stressor stimulation may affect a number of endocrine pathways.[28,35–37,61] This is not to say that cortisol is not involved, but the picture is more

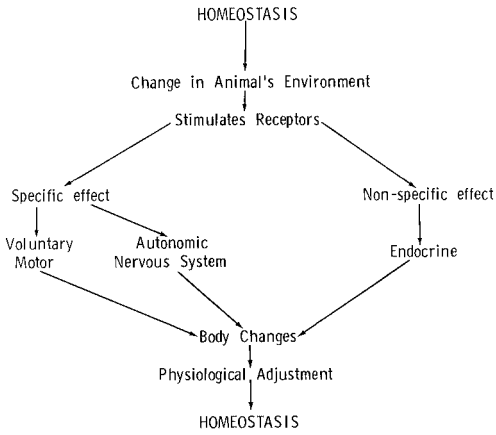

Figure 9.2. Homeostatic mechanisms.

Table 9.1. Clinical signs of the alarm response.

Vasoconstriction skin and intestine	Bronchodilation
Vasodilation muscles and heart	Pilocrection
Blood glucose ↑	Mydriasis
Metabolic rate ↑	Coagulation time ↓
Muscle fasciculation	Pain threshold ↓
Alertness↑	Blood pressure ↑
	Secretions ↓

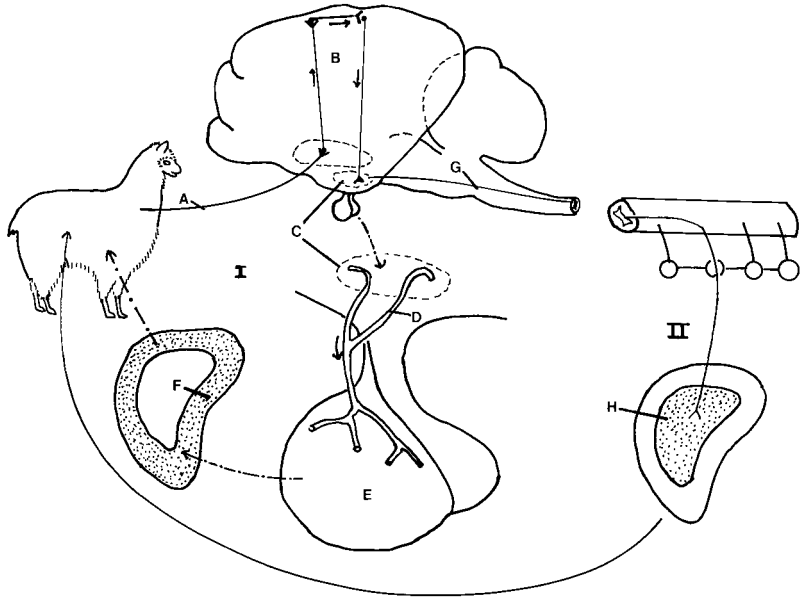

Figure 9.3. Diagram illustrating the pathways for the cortisol stress reaction (I) and the alarm response (II). (A) stimulus, (B) cortex, (C) hypothalamus, (D) blood flow from hypothalamus to pituitary, (E) pituitary gland, (F) adrenal cortex, (G) sympathetic pathway, (H) adrenal medulla.

complex than once supposed. More detailed information may be found in the references cited.

Continuous adrenal cortex stimulation and excessive production of cortisol elicit many adverse metabolic responses (Figure 9.3). Psychological as well as physical changes occur. The clinical syndromes of adrenocortical stimulation have been identified in some species (human, dog, horse, laboratory animals). There is much still to learn about the effects of hypercorticism in camelids. However, the basic biologic effects of cortisol should be understood.

Protein catabolism and lipolysis contribute to the pool for glyconeogenesis. Slight to moderate hyperglycemia has a diuretic effect, producing polyuria and polydipsia. Prolonged hyperglycemia stimulates the β cells of the pancreas to produce more insulin.

Cortisol reduces the heat, pain, and swelling associated with the inflammatory response, an effect useful in the treatment of many diseases. The anti-inflammatory action of cortisol is brought about by reducing capillary endothelial swelling, thus diminishing capillary permeability. Additionally, capillary blood flow is decreased by the action of cortisol. Both of these actions are helpful in shock therapy.

The integrity of lysosomal membranes is enhanced by cortisol. Under such circumstances, bacteria and other particulate matter are engulfed by phagocytes, but hydrolytic enzymes (which would destroy the organisms) are not released from the lysosomes.

Within a few hours of cortisol stress response, reduction in the number of circulating lymphocytes is 50% or greater. Lymphocyte levels return to normal

within twenty-four to forty-eight hours following cessation of stress. The effect of stress on the total leukocyte count varies with the species, and depends upon the normal relative leukocyte distribution. Species with normally high percentages of lymphocytes, such as mice, rabbits, chickens, and cattle, respond with a lymphopenia and neutrophilia and a decrease in total leukocytes. Dogs, cats, horses, camelids, and humans having relatively low lymphocyte counts respond with an increase in leukocytes.[27]

Eosinophil production decreases in response to elevated levels of cortisol. Eosinophil production is directly related to histamine production, such as occurs in the event of tissue injury or allergic reactions. Cortisol neutralizes histamines and inhibits regranulation of mast cells, thus further reducing histamine production. The elevated production of cortisol during stress results in eosinopenia. Catecholamines also cause eosinopenia; thus, emotional stress may also elicit a stress hemogram.[27] In addition, cortisol stimulates increased production of circulating erythrocytes. Serum calcium (Ca) levels decrease as well, through inhibition of Ca absorption from the gastrointestinal tract.

Interference with DNA synthesis causes atrophy of lymphoid tissue throughout the body. Cell-mediated immune responses are diminished,[22] an effect that may interfere with tuberculin-testing programs. Neutrophilia, eosinopenia and lymphopenia are produced. Leukocytosis, monocytosis, and an absolute lymphopenia and eosinopenia are the characteristics of a stress hemogram of a camelid because of the preponderance of neutrophils in the leukocyte series.

Gastric and intestinal ulcers are common in llamas. No etiologic agent has been identified. Stress ulceration of the gastrointestinal system is a well-known syndrome in humans, rats, and marine mammals. Whether or not stress is a factor in llama ulcers is unknown, but one should be mindful of the basic effect of cortisol on the digestive system. Most of the studies have been performed on humans and laboratory animals, and because there may be significant species differences, direct extrapolation is unwise. The pathogenesis of gastric stress ulcers in humans and marine mammals is multifactorial. Hypercortisolism causes hypersecretion of acid and digestive enzymes. A duodenal reflux introduces substances from the duodenum into the stomach (lysolecithin) that reduce the effectiveness of the mucous membrane barrier. A third factor is vasoconstriction of the vasculature of the stomach, which in turn causes local hypoxia and a deficiency of adenosine triphosphate. These also contribute to the reduction of the mucous membrane barrier. Whether these factors are operating in the llama is unknown but should be considered.

Ulcers also occur in compartment 1 of the llama stomach (no gastric enzymes or acid production) and the colon. A different mechanism is the likely explanation of the pathogenesis of ulcers in these locations.

Catecholamines (epinephrine) contribute to the production of gastric secretions, so stimuli mediated via the sympathetic nervous system (fear, anxiety, frustration, anger) may have a potential effect on ulcerogenesis.

Hemorrhagic gastroenteritis and ulceration are sequelae to severe trauma (head injuries, burns, multiple fractures, spinal surgery) in humans, dogs, and other mammals.[27,61] A juvenile alpaca with a congenital cervical vertebrae defect developed hemorrhagic enteritis twenty-four hours following an extensive myelographic examination.

The lesions produced by harmful stress are difficult to document. Pathologists often negate a diagnosis of death caused by stress. Many of the effects of stress are functional, leaving no definitive lesion to mark their presence. Nonetheless, it is known that tissues and organs are weakened by prolonged insult, lowering resistance to disease. Classic lesions are lymphoid tissue atrophy, adrenal cortical hyperplasia, and gastrointestinal ulceration. Though the actual cause of death may be pneumonia, parasitism, or starvation, stress may have paved the way for development of these terminal ailments.[27]

Veterinarians providing health care for camelids should consider stress as a contributory factor to specific diseases. Husbandry practices should be evaluated and those that may be harmful corrected. Camelids are social animals. Isolation for therapy or recuperation may be counterproductive. Malnutrition is a stressor, as are repeated restraint episodes. More detailed information about stress may be obtained in the references.

THERMAL STRESS

Physiology of Thermoregulation and Water Balance[28,34,40,53]

SACs are especially adapted for dealing with a cool environment, but they have been introduced to a wide variety of habitats in North America and other countries and have demonstrated a general adaptability. The ventral regions of the body are sparsely covered with hair, providing a thermal window for dissipation of heat. Evaporative cooling efficiency is determined by skin temperature, insulation, and radiant heat (sun, heated surrounding surfaces). The fiber coat of llamas and alpacas is a highly efficient insulating layer against cold, and it also inhibits radiant heat from reaching the skin. The fiber layer may serve as a positive or negative barrier for evaporative cooling. Evaporative cooling involves the conversion of water to vapor, which

requires 580 calories per gram of water converted. If the vapor is not dissipated promptly, it will re-condense into water, so if the fiber coat is dirty, matted, excessively long, or wet, heat dissipation is inhibited.

Behavioral activities that aid SACs in thermoregulation include seeking shade, orientation to minimize radiant heat gain when lying in direct sunlight, recumbency when in sunlight to cover the thermal window, standing in a stream or pond, or cooling by standing over a sprinkler.

SACs have epitrichial sweat glands, probably distributed over much of the body but especially numerous on the relatively fiberless areas of the ventrum.[2] In a study of evaporative water loss in the llama, there was no detectable loss when the llama was maintained at 20°F to 25°C (68°F to 77°F).[11,37] When the ambient temperature was raised to 40°C (104°F), vapor loss became evident and gradually increased to 100 to 240 g/m^2/body surface/hour.[3] Intravenous (IV) injection of epinephrine caused an increased loss of water vapor similar to that caused by exposure to high ambient temperatures.[5]

Only one water balance study has been made in the llama, in which the llama was compared with the goat.[15,43] Water turnover rates were calculated under conditions of ad libitum water intake with adequate feed intake, water deprivation and adequate feed, and reduced feed intake with ad libitum water intake. Nondesert-adapted species usually lower feed intake when water is restricted. The llama continued to ingest feed when water was restricted, thus making oxidative water available to cope with potential dehydration. Both the llama and goat were able to increase the osmolarity of urine under conditions of feed and water restriction, indicating an ability to conserve moisture by diminishing renal excretion.[43]

The energy metabolism of an SAC under restricted food intake lowers from 61 kcal/kg[75]/24 hours to 52 kcal/kg[48,75] This is associated with a concomitant decrease in water requirements.

In a study of guanacos, it was determined that these wild SACs can deal with heat stress and dehydration, but to a lesser degree and with different mechanisms than the camel.[41] Water was withheld from a female guanaco for five days, while feed was supplied ad libitum. A 14.8% loss of body weight resulted. Body temperature remained within a normal range.

Water was withheld from a male guanaco for four days in an area with an ambient temperature of up to 28°C (84°F). A 23.4% loss of body weight occurred, but there was no elevation in body temperature.[36] At the conclusion of the foregoing period, the male was subjected to an ambient temperature of 45°C (113°F) for six hours. Even with heat stress added to the marked dehydration, body temperature elevated less than 1°C (1.8°F). The heart rate of the heat- and water-stressed guanaco was not elevated, in contrast to the response of domestic animals and humans, who exhibit a significant increase in heart rate when under such stress.[41]

The packed cell volume (PCV) of the 23.4% dehydrated guanaco increased from 30.6% to 43.6% and the hemoglobin (Hb) from 13.9 to 18.9 g/dl. This indicated an approximate 40% reduction in blood plasma.[36] The camel has only a 2.3% reduction in blood plasma during severe dehydration and thus is better adapted to endure dehydration without ill effects.

The respiratory rate was not elevated above normal when guanacos were heat stressed, but when the animals were dehydrated and then subjected to heat stress, the respiratory rate increased two- or three-fold. This may be explained as follows. Because there is no body temperature elevation in un-dehydrated but heat-stressed guanacos, thermal balance must be accomplished by dermal evaporative cooling. In the dehydrated guanaco, there is a reduction in plasma volume and a diminished ability to sweat; when heat stress is added, hyperpnea provides an alternative to dermal evaporative cooling.

Guanacos, like camels and donkeys, are able to correct severe dehydration quickly when offered water. A 23.4% dehydrated guanaco was able to drink 9 L water in eight minutes, restoring 66% of the deficit.[36]

The dromedary camel is one of the most well adapted animals to hot arid climates.[17] Contrary to popular opinion, the camel does not store water any more than any other species, yet it need not drink water for days. The camel is able to tolerate extreme dehydration and has been known to safely lose body water equal to 40% of its body weight. Such a water loss would be lethal in any other animal. In the camel, plasma volume is maintained at the expense of tissue fluid; thus, circulation is not impaired. The small oval erythrocyte of the camel continues to circulate despite increased blood viscosity.

Even after severe dehydration, the camel is able to drink sufficient water at one session to make up the deficit. This amount of water would cause severe osmotic problems in humans or other animals. In the camel, water is absorbed from the stomach and intestines slowly, allowing equilibrium to be established. The erythrocytes are able to avoid osmotic problems by swelling to 240% of their initial volume without rupturing. In other species, erythrocytes can swell only to 150%.[22] SACs share some of these characteristics with camels.

The camel is able to endure a diurnal fluctuation of body temperature, from 36.5°C to 42°C (97.7°F to 107.6°F).[22,45–47] The body acts as a heat sink during the heat of the day, thus conserving vital water that would otherwise be lost through evaporative cooling. During the cool night of the desert, excess body heat is dissipated by conduction.

The kidney of the camel is capable of concentrating urine markedly to diminish water loss. The urine becomes as thick as syrup, and salt content may be increased to twice the concentration of salt in sea water. Water is extracted from the fecal pellets to such a degree that they can be used for fuel immediately upon voiding. All these unique physiologic characteristics must be considered when evaluating clinical signs in a diseased camel.

Hyperthermia (Heat Exhaustion, Heat Stroke, Sun Stroke, Heat Stress)[6,10,22,25,33,39,59]

Hyperthermia is a significant problem in llamas and alpacas in many areas of the southern and western United States. The South American camelids' later evolutionary development occurred in a cool climate, thus they are better able to deal with cold than excessive heat and humidity.

Etiology

The predisposing factors for development of hyperthermia in SACs include prolonged, high environmental temperatures and humidity, muscular exertion, fever, dehydration, mycotoxins that inhibit thermoregulation, and drugs that depress thermoregulation. Activities that may contribute to the production of body heat include packing, racing, breeding, fighting, being transported, prolonged restraint, being chased by dogs, or breeding males pacing a fence.

The normal core body temperature of adult SACs ranges from 37.5°C to 38.6°C (99.5°F to 101.5°F). Normal temperature of neonates may be a degree higher than that of the adults. It is not uncommon for adult animals in a hot climate to maintain a daytime core body temperature of 40°C (104°F). This is quite close to other species' temperatures that are considered to be the risk threshold for potential damage to organs and tissues (40.1°C to 41.1°C [104.2°F to 106°F]).

Both the camel and the guanaco are able to sustain a 25% body weight loss as a result of dehydration without observable ill effects. Furthermore, both were able to rehydrate themselves immediately when given free access to water. A dehydrated human must be rehydrated slowly to avoid rupture of the erythrocytes as a result of too rapid water absorption. Camelids have an elliptical erythrocyte that allows swelling to 240% without rupture. A human may sweat 1 L/hour if heat stressed but hydrated. It is unknown what the sweat production rate is in llamas or alpacas.

The degree of hyperthermia and the effects of hyperthermia on organs and tissues may vary according to the duration of exposure to excessive heat and humidity and the presence of other conditions, such as metabolic acidosis, cardiovascular dysfunction, chronic disease, excessive fiber covering, and/or the presence of saddle pads and packs.

Body temperatures in llamas and alpacas rarely are noted above 43.3°C (110°F); in fact, animals in severe heat stress may have temperatures of only 41.1°C to 42.2°C (106°F to 108°F). Protein denatures at temperatures of 45°C to 47°C (113°F to 116.8°F), causing immediate death of tissues and organs.

Effects on Organ Systems

It is often assumed that when a hyperthermic animal has been cooled, all organ systems begin functioning again at their normal capacity. That may be true if the heat stress has been of short duration and moderate intensity. If heat stress has been severe or prolonged, many residual effects may alter organ function and even kill the animal long after the core body temperature has returned to normal. A veterinarian must understand the effects of heat stress on organ systems to make an accurate diagnosis, treat a case effectively, and give a prognosis.

Central Nervous System. One of the systems most sensitive to hyperthermia is the central nervous system (CNS). Effects on the CNS may be initiated by direct effects of heat, causing coagulative necrosis of neurons; by secondary factors, such as hypotension, causing cerebral hypoxia; or by electrolyte alterations, causing neurotransmission dysfunction. Lesions in the CNS may also be caused by hyperthermic damage to the cardiovascular and hemic systems (hemorrhage, disseminated intravascular clotting [DIC]). Signs exhibited are determined by the area of the CNS damaged, but generally there is decreased mental function and there may be convulsions. Damage to the thermoregulatory centers may predispose animals to relapses or subsequently increased sensitivity to heat.

A frequently overlooked but serious consequence of heat stress in a pregnant female is fetal CNS damage, resulting in various congenital anomalies or even death of the fetus. Congenital CNS defects associated with prenatal prolonged hyperthermia in humans and other animals include exencephaly, anencephaly, encephalocoele, microencephaly, spina bifida, hydrocephaly, and neurogenic arthrogryposis. These anomalies are the result of excessive heat acting on the embryonic cells of the CNS at a crucial time. In humans, the critical time is between forty and forty-four days following fertilization. The crucial period in camelids is unknown, but is likely approximately the same.

Reproductive System. Heat stress may have a marked effect on the adult female, including diminished intensity of receptivity and anestrus. During pregnancy, the more profound effects are seen as fetal damage, including inhibition of embryonic cleavage and implantation, initiation of teratogenesis, and abortion.[19,36,37]

General effects on the fetus may result in reduced birth weight, which may be caused by placental retar-

dation. Fetal effects have been noted in other species when the core body temperature of the dam rises above 40.1°C (104.2°F) for prolonged periods. Recall that these are temperatures routinely recorded in clinically unaffected llamas and alpacas during hot weather. Hyperthermic effects on an embryo depend on the degree of hyperthermia, duration of hyperthermia, and the stage of development of the embryo.

Abortion may be the result of placental necrosis, direct effects on the fetus causing death (microvascular leakage, edema, hemorrhage), or, in near-term fetuses, a stress response causing elevated cortisol levels.

In males excessive heat is spermicidal, at the primary spermatocyte stage. In much of the southern and western United States it is common for males to become infertile during the hot weather of summer. The scrotum is not pendulous as it is in most domestic ruminants, thus core body temperature changes have a rapid and profound effect on developing spermatids. Although not yet studied in llamas or alpacas, at least thirty-five days up to sixty days are required in other species for new spermatogenesis to produce mature viable sperm once the heat stress has decreased. Scrotal edema is a classic sign of hyperthermia in male SACs (Figure 9.4A and B).

Respiratory System. A 1°C (1.8°F) rise in the body temperature increases the requirement for oxygen by 10% to maintain normal function of the energy systems of the body. If the body temperature rises to 41°C (105.8°F), the respiratory system can no longer supply sufficient oxygen by normal respiration. Heat stress causes tachypnea, respiratory acidosis, and open mouth breathing (this sign alone is inconclusive, as any altercation, particularly between males, results in a pouting expression with open mouth that appears to be open mouth breathing).

Digestive System. Signs of colic are commonly seen in heat-stressed SACs. Elevation of the core body temperature initiates a shift in the blood supply from the viscera to the skin. Decreased blood flow to the stomach and intestine causes decreased digestive function. Gastrointestinal motility is decreased, as is rumination.

Hypoxia of hepatocytes and decreased hepatic function results from decreased blood flow to the liver. In severe cases there is a failure of production of elements in the coagulation cascade. Persistent low-intensity hyperthermia may cause decreased digestive function, which in turn may cause poor growth rates in juveniles and poor appetite and less efficiency in feed utilization in adults.

Cardiovascular System. Hyperthermia causes dilatation of peripheral arterioles and a shift of blood from the viscera to the skin. Heart rate is increased and central venous pressure decreased, along with a relative decrease in blood volume (potential for hypotension and hypovolemic shock). Animals with previous cardiac conditions (ventricular septal defect) may be at greater risk of dying.

Hematopoietic System. Hyperthermia causes hemoconcentration, electrolyte imbalances, increased fragility of erythrocytes, leucocytosis, and metabolic acidosis. Platelet counts are decreased. Effects on the

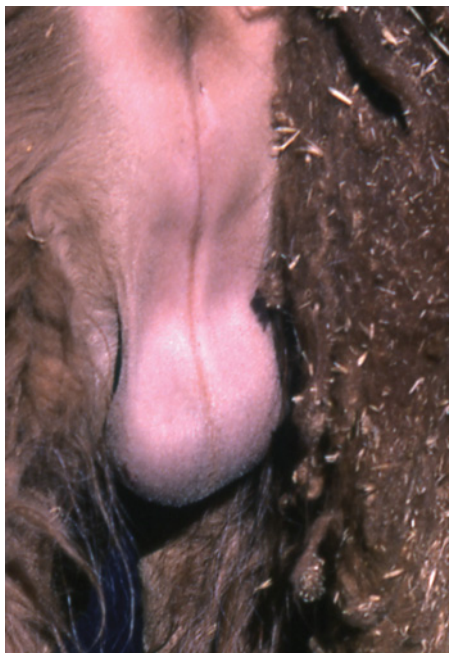

Figure 9.4A. Scrotal edema caused by hyperthermia.

Figure 9.4B. Scrotal swelling caused by an inguinal abscess.

coagulation cascade may be profound and lethal. Prothrombin time is increased, and there is an increased consumption of coagulation factors and fibrin split factors, resulting in hemorrhage and, potentially, DIC.

URINARY TRACT. Hypovolemia may result in decreased glomerular filtration and loss of kidney function (prerenal uremia), followed eventually by renal shutdown, possibly complicated by DIC. Generalized hemolysis overloads the kidney with hemoglobin, which exacerbates any kidney malfunction or may be the direct cause of kidney malfunction.

Miscellaneous Defects Caused by Prolonged Hyperthermia

In addition to effects on the CNS, the following congenital defects have been associated with hyperthermia in other species: microphthalmia, exophthalmos, coloboma, talipes, facial clefting, cardiac defects, and renal agenesis.

Sequence of Events During Hyperthermia

As may be ascertained from the foregoing discussion, many changes in organ systems have a bearing on diagnosis, clinical signs manifested, and diagnostic tests that may be employed to assess organ system function and potential for residual effects that may complicate recovery or result in the death of the animal some days later. Following is the probable sequence during a severe hyperthermic episode in a llama or alpaca.

1. Elevation of the core body temperature
2. Accelerated heart rate
3. Increased respiratory rate
4. Redness of skin surface
5. Sweating
6. Hemoconcentration
7. Body fluid shift from viscera and muscle to skin
8. Decreased glomerular filtration
9. Dehydration
10. Decreased central venous pressure
11. Effects on the CNS, including cerebral hypoxia and coagulative necrosis
12. Effects on the embryo and fetus
13. Coagulation defects (DIC)
14. Other organ system damage

Differential Diagnosis

Primary hyperthermia must be differentiated from infectious diseases producing a fever. Additional diseases that should be on a differential list include polioencephalomalacia, tick paralysis, meningeal worm parasitism, primary digestive system disturbance (colic), and gastric ulcers.

Necropsy

Any delay in conducting a necropsy on a hyperthermic SAC will result in a pathology report stating that the carcass was autolyzed. The insulating fiber coat prevents dissipation of normal body heat, and when this is complicated by hyperthermia, autolysis is rapid. Thus, necropsy of an animal that died and was undetected for six hours in hot weather will provide little useful information.

Gross lesions suggestive of death from hyperthermia include hemorrhages and DIC. Intact males may show scrotal edema (Figures 9.4A, 9.4B). Coagulation necrosis of the neurons of the CNS and necrosis of the liver and kidneys would be characteristic histopathologic findings.

Therapy

Successful resolution of hyperthermia requires early recognition, rapid cooling, supplying supplemental oxygen, fluid administration to deal with hypovolemia, and correction of acidosis.[50] The patient should be monitored through sequential hemograms and serum chemistry profiles measuring hemoconcentration, electrolytes, pH, platelets, and the coagulation cascade elements.

Soaking the fleece of heavily fibered animals is not effective in cooling. Cold water should be applied to the ventral abdomen, including the axillary and inguinal spaces. Cold water enemas are highly effective, but the ability to monitor the body temperature is lost, unless the animal is a female and the vulva can be used to monitor temperature. See Chapter 4 for illustrations of giving an enema.

Basic maintenance fluid requirements for an adult llama are approximately 30 to 40 ml/kg/day. Crias require 80 to 120 ml/kg/day. Depending on the degree of dehydration, two to five times the basal requirement may be necessary. Normal saline or lactated Ringer's may be used to supply fluid requirements. Electrolyte imbalance must be dealt with according to serum chemistry findings. Acidosis may be controlled by using sodium bicarbonate intravenously. The use of dipyrone to reduce the temperature may be useful in fever conditions, but the author does not recommend it for hyperthermia.

Prevention

Managers must provide shade and possibly water for cooling. Sprinklers that spray only the surface of the coat may do more harm than good, though sprinkling may lower the ambient temperature. It should be obvious that plenty of potable water must be available. A pond is an excellent refuge for llamas and alpacas in hot weather (Figure 9.5). A shady, sandy spot that can be moistened is desirable; the animal can lie upon

Figure 9.5. Hyperthermic alpaca cooling herself in a pond.

this and become cool through the thermal window. Llamas and alpacas have been observed lying down in full sunshine through most of the day. Presumably they have selected a spot that cooled down during the night, and as long as the animals remain recumbent, they may cool through the thermal window.

Restraint procedures should be avoided on warm days. Clients should be advised against heavy work for packers during the afternoons on hot days. Feeding concentrates and high-protein forages should be avoided, because llamas excrete excessive nitrogen byproducts via the urine, thus increasing fluid requirements.

Many North American llama owners have resisted shearing their animals because months are required for the fiber coat to grow back and make them presentable. Fortunately, that trend is changing, and many have found that shearing is mandatory for the well-being of their animals. Alpaca owners have long understood the need for shearing (Figure 9.6). Total shearing does not automatically allow the SAC to thermoregulate better; in fact, it may be undesirable to closely shear a SAC at the approach of the hot season. Partial shearing of a heavily wooled animal, leaving 8 to 10 cm (3 to 4 in.) of fiber, is an appropriate course of action, especially for the hyperthermia-prone llama.

Some animals are at increased risk of hyperthermia because of low heat tolerance. Older animals may have decreased sweat gland activity, be in poor physical condition, or have deterioration of cardiovascular function. Obese animals are generally less physically fit than those of normal weight. Evaporative cooling is determined by the number of sweat glands per square meter of skin surface. Metabolic heat is produced in

Figure 9.6. Alpaca with the body fiber clipped.

proportion to body mass but is dispersed in proportion to skin surface area. Thus, obese animals are at a distinct disadvantage in hot weather. These animals must be observed frequently and special steps taken to ensure their ability to remain cool. Some owners have provided fans and even air-conditioned stalls in particularly oppressive conditions.

Hypothermia

Predisposing Factors

Although SACs are adapted to the cool temperatures of their native lands, temperatures there do not reach the extremes found in areas of North America where winter nighttime temperatures may dip to minus 40°C (–40°F). When combined with wind chill, the effective temperature may be –73°C (–100°F). Without shelter, SACs have difficulty coping with such severe cold.

Neonates are particularly susceptible to hypothermia because they have poorly developed thermoregulation mechanisms and a higher metabolic rate. Their relatively greater proportion of skin surface allows for rapid dissipation of heat. Neonates lack a shivering reflex. Even adult SACs under anesthesia or in shock are prime candidates for hypothermia if in a cold ambient environment.

Insufficient food intake reduces metabolic heat production. Restricted muscular activity prevents heat generation. A poor fiber coat has less insulation capacity and contributes to heat loss. Any or all of these factors predispose SACs to hypothermia.

Signs

Clinical thermometers record body temperatures only to as low as 33.3°C (92°F). With more sensitive thermometers, temperatures as low as 29.4°C (85°F) have been recorded in living SACs. Other signs include depression progressing to coma. In contrast to hyperthermia, the hypothermic SAC may live for hours.

A decrease in body temperature is accompanied by a decrease in cardiac output, heart rate, blood pressure, and glomerular filtration rate. Blood viscosity and hematocrit levels increase. Signs noted with temperatures below 30°C (86°F) include slow and shallow breathing, metabolic acidosis, "sludging" in the microcirculation, ventricular arrhythmias leading to fibrillation, and coagulation disorders.

Therapy

Total body immersion in warm water 40.5°C to 45.5°C (105°F to 14°F) is the fastest way to warm an animal. Total body immersion is impossible with an adult llama but possible with a neonate. Running the fingers through the fiber coat keeps warm water flowing over the skin. If the whole body cannot be immersed, warm water should be applied to the legs and the legs massaged.

A warm water enema is highly effective, but the ability to monitor the body temperature is temporarily lost. A hair dryer may be helpful in warming a neonate. Covering the animal with blankets helps conserve heat, but if the temperature is below 32.3°C (90°F), metabolic heat production is proportionately reduced, and endogenous rewarming is slowed.

IV infusions of warm saline are effective. Surgical exposure of a suitable vein may be necessary to effect IV administration because of vasoconstriction. Circulating water-type heating pads are effective in preventing hypothermia in neonates during surgery and in treating accidental hypothermia, but electric heating pads have caused skin burns and sloughs. Hypothermic and shock patients normally suffer from skin vasoconstriction and exhibit a reduced ability to carry heat away from the skin. Be cautious when applying heat directly to the skin. Measure the temperature between the skin and the pad and keep the temperature below 42°C.

Hot water bottles may be used to raise the ambient air temperature in a small enclosed area. Plastic milk cartons or plastic bags may be substituted for hot water bottles. The air surrounding the patient may be warmed with infrared heat lamps, forced-air driers, or electric floor heaters.

Prevention

Shelter from wind and rain should be provided. SAC fiber is quite resistant to moisture penetration, but wet face, ears, and limbs may allow significant heat loss. Deep straw bedding may minimize heat loss from the thermal window in extremely cold climates. A shelter should be small enough to be warmed by a group of animals huddling together. A box stall may be made smaller by blocking with bales of hay or straw. Insulated and heated barns may be required in particularly harsh, cold climates.

High-quality feed should be provided, including concentrates if the animals are used to eating them. Water must be available. SACs are not likely to break through ice and are reluctant to drink sufficient amounts of icy water. A stock tank heating unit may be required.

For birthing in cold weather, a maternity stall with provisions for supplemental heat should be available.

SACs generally lie down when being transported. If trailering in cold weather, the trailer floor should be deeply bedded with straw to avoid hypothermia from lying on a cold or freezing surface.

Frostbite

Hypothermia causes peripheral vasoconstriction to conserve energy to maintain the core body temperature. Intense or prolonged skin vasoconstriction of exposed structures may result in ischemic necrosis and gangrene. The ears are the primary site of gangrene in llamas and alpacas. Neonates are most frequently affected; however, at least one ill recumbent adult llama lost ears to frostbite during a cold spell in the north central Midwest.

SIGNS. The skin of the ear becomes devitalized. A sharp line of demarcation separates healthy tissue from the necrotic tip. The skin and the cartilage become hardened and leathery, then slough.

Frostbite must be differentiated from congenital shortening of the pinna. Frostbite usually produces a squared tip rather than the tapered tip of the congenitally shortened ear. The healed margin produced by frostbite is scar tissue, devoid of hair. The margins of congenitally shortened ears are haired. See Chapter 19 for an illustration of frostbite.

THERAPY. Once freezing has occurred, no treatment will halt the process. The necrotic tissue may be amputated, but it is not necessary or advisable because more of the ear may be saved by nature than by the surgeon. Antibiotic therapy may be indicated for five to eight days to prevent tetanus in animals not otherwise immunized against it.

PREVENTION. All the measures suggested for prevention of hypothermia are appropriate.

DEHYDRATION

The mammalian body is composed of 60% to 80% water. High percentages of body fat decrease the percentage of water.[11,12,17,21,34,42,44–47] Desert-adapted animals, such as certain species of antelope and rodents, have developed methods of water conservation similar to those previously described for the camel. Mammals adapted to temperate climates require water in amounts of approximately 40 ml/kg/body weight daily to maintain normal water balance in a basal metabolic state. Exact water requirements are based on metabolic weight rather than actual body

weight (1 ml of water for each kcal of required energy). The basal amount is equivalent to fluid lost in urine and feces and through insensible evaporation via skin and lungs. The actual amount required is increased by activity and a rise in ambient temperature. As already described in the section on the physiology of heat stress and dehydration, camelids are able to tolerate degrees of dehydration that would be fatal to humans or other temperate climate-adapted species. Water requirements of camelids are listed as ml/kg in Table 2.4 in Chapter 2.

Etiology

Dehydration is usually caused by water deprivation brought about by failure of a newly acquired animal to recognize the water source, a frozen water source, failure to use automatic waterers, insufficient water supply during hot weather, overheating, prolonged muscle exertion, severe diarrhea, hemorrhage, or loss of fluid resulting from burns.[16]

Signs

Signs include muscle weakness, depression, enophthalmus (eyeball retracted into the orbit), tacky or dry oral mucous membranes, decreased urine production, low blood pressure leading to prolonged capillary refill time, cold extremities, elevated heart rate, decreased pulse pressure, and difficulty in distending a vein for intravenous therapy. Decreased skin turgor is mentioned for evaluating dehydration in companion animals and horses, but thickness of the skin precludes the use of this parameter in camelids, except in neonates. Skin turgor is obscured by obesity and exaggerated by emaciation.

In one study, a guanaco experienced a 23.4% weight loss from four days of water deprivation yet exhibited no clinical signs of dehydration.[36] He continued to eat and had no circulatory deficits. There was hemoconcentration, with the PCV elevating from 30.6% to 43.6%. It is important to note that the normal PCV is lower in camelids than in other domestic animals, and though a 43.6% PCV would cause no alarm in other species, a rise of that magnitude represents a plasma volume loss of as much as 40%. The hemoglobin level in the blood elevated from 13.9 to 18.9 g/dl. The respiratory rate may increase in dehydrated camelids, especially if under heat stress, because of the loss of plasma volume and the inability to sweat or thermoregulate.

Laboratory Findings

Dehydration results in an elevated PCV, Hb, erythrocyte count, plasma protein, serum urea nitrogen (SUN), and creatinine. Blood chemistry values for sodium (Na), potassium (K), chlorine (Cl), pH, and bicarbonate may vary with the underlying disease process.

Therapy

Lost body fluids must be replaced. Camelids are able to rapidly restore a deficit by oral fluids without causing the osmotic upsets seen in temperate climate-adapted species. Gastric intubation may be necessary to relieve moribund animals. Fluid is readily absorbed from the colon, so enemas are effective in rehydration. IV administration of physiologic saline may be lifesaving in severe dehydration. Surgical exposure of a vein is often necessary because of low blood pressure.

Prevention

Attention to all the factors discussed in the section on etiology will prevent dehydration. Special attention must be given to the camelid with persistent vomiting (rhododendron poisoning) or severe diarrhea. Electrolyte imbalances must also be alleviated.

FLUID THERAPY IN LLAMAS AND ALPACAS

Fluid therapy is a fundamental component of medical and surgical therapy in many diseases. Fluids may be administered orally, intravenously, intraperitoneally, subcutaneously, and via the rectum. All of the fluids used to treat other species of domestic animals are appropriate for camelids. Following is a discussion of some salient features of fluid therapy.

Total Body Water

Water comprises approximately 60% to 70% of an adult camelid's body weight. In the neonate or cria body, water content varies from 75% to 85%. The majority of the fluid is intracellular. Extracellular fluid is primarily in plasma but may also be found in intercellular spaces, within the lumen of the gastrointestinal tract, and in other spaces (peritoneal cavity, thoracic cavity). K is the primary anion in the intracellular space, and Na in the extracellular spaces.

Various disease processes cause a shift of fluids and electrolytes among compartments. Plasma volume is often altered in fluid balance changes. Decreased plasma volume results in poor venous return, decreased cardiac output, inadequate tissue perfusion, and, ultimately, shock. Decreased cellular perfusion leads to anaerobic metabolism, accumulation of lactate, decreased renal perfusion, and metabolic acidosis, all of which may have profound effects on various organ system functions.

Indications for Fluid Therapy

The following conditions warrant careful consideration for the administration of fluid, with or without electrolyte supplementation: maintaining homeostasis during surgery, diarrhea, vomiting, dehydration (may be caused by inadequate intake or a fluid-losing disease), hyperthermia, hemorrhage, paralytic ileus,

peritonitis, renal failure, esophageal obstruction, and intestinal obstruction. Dehydration may be seen as a primary disorder, but it is usually associated with other disease processes. It is important to recognize and treat dehydration because its effects may so alter metabolism that medical or surgical therapy for other conditions is negated.

Degree of Dehydration

The critical method of determining the degree of dehydration is to evaluate hemogram and blood chemistry data.

Neonates

The calculated basic fluid requirement is 80 to 100 ml/kg/day, but in practice 80 to 120 ml/kg/day is given. Neonates are at a higher risk for infection than adults, so asepsis must be strict.

A normal neonate has a higher urine output and a lower fecal output as compared with a normal adult. Body fluid compartmentalization differs between neonates and adults, with total body fluid, plasma volume, and extracellular fluid volume relatively higher than in an adult. Premature crias have an even higher relative volume of fluid.

Neonates have a higher surface-to-body-weight ratio and thus have a greater insensible fluid loss than adults. Fluid and energy requirements are linked to an all-milk diet. Anything that inhibits intake has a profound effect on energy and fluids.

Adult Llamas or Alpacas

The basic maintenance fluid requirement is 30 to 40 ml/kg/day. A rough approximation of fluid requirements for replacement in a dehydrated camelid may be calculated from the following formula: Fluid deficit (L) = (% clinical dehydration × body weight in kilograms) + maintenance requirement. (An example: 8% dehydration × 75 kg = 6 L + 3 L for maintenance for twenty-four hours. Total = 9 L.)

If an animal has an ongoing fluid loss, such as having diarrhea, hyperthermia, or vomition, additional fluid must be administered. It is common to underestimate the volume of fluid necessary to rehydrate an animal. A 150-kg llama with 10% dehydration would require 15 L fluid to make up the deficit. With 20% dehydration, 30 L (8 gal) are required.

GERIATRIC MEDICINE IN LLAMAS AND ALPACAS[14,24,26,32]

Some SACs are entering their golden years. In other livestock enterprises, aging, nonproductive animals are usually culled from the herd, because they are not profitable. SAC owners who are not willing to accept culling as an alternative now consult with veterinarians to deal with the special problems of aging animals. Many owners also desire to extend the productive life span of both males and females to capitalize on their investment.

Population dynamics data for llamas and alpacas are unavailable in both North and South America. North American breeders are unable to call upon the experience of South American producers because there is no older population of llamas or alpacas in South America. Nonproductive animals are eaten.

The question as to what constitutes an old llama was asked of a number of experienced llama/alpaca breeders who have large herds. The response was surprisingly uniform (fifteen to eighteen years). Figure 9.7 is a diagram comparing the life stages of llamas with those of humans. The data are only approximate. The description of characteristics of old llamas is based on personal experience, plus experiences of longtime breeders of llamas. It is also possible to extrapolate from other animals that have been allowed to live out their life spans and to apply basic principles to augment meager knowledge.

Age Changes of Medical Importance

All organ systems age, but changes in some are more dramatic than in others. This discussion is limited to a few systems, including dental, skeletal, reproductive, immune, and special senses.

Dental

There are two stages in the life of an SAC when dental problems are likely to occur: when the permanent cheek teeth erupt (three and a half to five years) and after a lifetime of chewing and grinding that wear teeth. The precise age when teeth become worn varies but is determined by the type of feed provided over the years, inherent dental quality, and occlusion of teeth. (See the discussion on teeth in Chapter 13 for further details.)

Skeletal System

Llamas used for packing may experience more wear and tear on the skeletal system than breeders. Animals with basic conformational weakness are more prone to unsoundness in later life. Expect to see changes in conformation of older animals. A female that has produced numerous crias may develop lordosis in her old age. The lordosis may intensify as term nears, and her body may return to near normal conformation following delivery. Likewise, the stretching of tendons and ligaments may cause an SAC to be over at the knees (buck kneed) or down in the fetlock.

It is important to evaluate such conformation defects in a proper light, related to circumstance. Such faults present in a young female should raise concern in regard to breeding this female and perpetuating a fault. It should be understood, however, that the

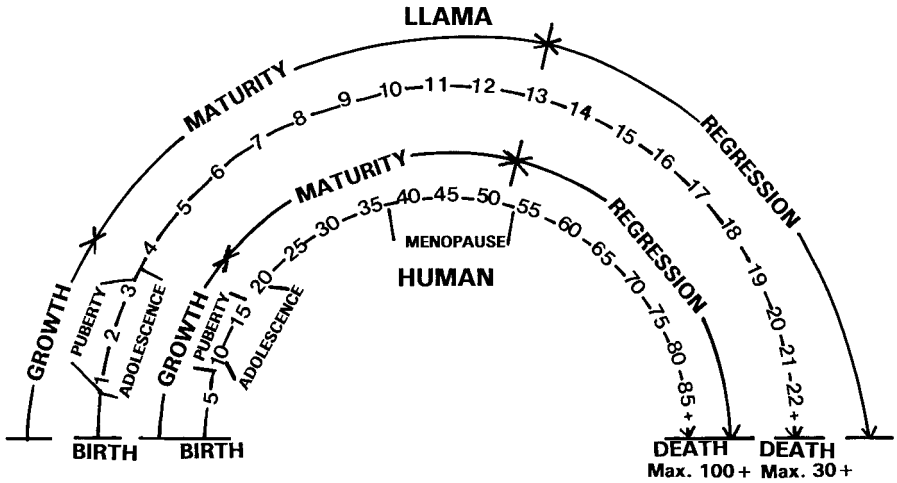

Figure 9.7. Diagram comparing life stages of an SAC with those of a human.

results of normal wear and tear are not usually genetic in origin.

Each skeletal condition should be evaluated as to impact on function, presence of pain, and whether or not corrective measures are possible. Degenerative changes and bony spurs may occur in any joint, including those of the spine (spondylosis). Spondylosis may be present as an incidental finding, but trauma and/or Ca:phosphorus (P) imbalance may exacerbate the condition. Young animals with conformation faults are more prone than others to suffer degenerative changes and calcification of collateral ligaments as they age because of asymmetrical wear on joint surfaces or tangential forces from improper angulation. Older SACs may move about more slowly or stiffly as a result of pain, but they may also be naturally more sedate than youngsters and prefer to conserve energy.

There may be reluctance to roll in the dust bath, or the head or neck may be held in peculiar positions. They may be stiff when first arising in the morning or after recumbency, have unnecessarily frequent and/or prolonged periods of recumbency, or resist turning one direction or another when being led. The head may not be held upright. Lameness, frequent resting of the head and neck on fences or feed bunks, and constant shifting of weight from one leg to another may be observed. Affected SACs may graze while lying down.

Management. Attempt to identify specific conditions and deal with them, if treatable. However, in many cases there is little a veterinarian can do. Use caution when placing camelids on long-term anti-inflammatory or anti-arthritic drugs, which may produce significant untoward side effects (production of stomach ulcers). However, some llamas have been on long-term phenylbutazone therapy for years.[a] The animal should be made comfortable. Bedding, or sandy or grassy spots in cool, shaded areas, may be provided.

From a preventive standpoint, select animals that are well conformed to begin with and keep feet trimmed properly. Animals that must compensate for mechanical instability are more prone to develop skeletal unsoundness.

Vision and Hearing

Vision and hearing may diminish. Senile cataracts occur in most species but have not yet been reported in llamas or alpacas. Llamas and alpacas are both visual and auditory in their social communication, yet animals with impaired vision and/or hearing may function admirably in a familiar enclosure. Problems have been noted when such animals have been shifted to unfamiliar enclosures or grouped with strangers. Advise clients to be perceptive and aware of changes that may occur in their animals.

Immunity, Metabolism, and Organ Function

Little is known about the immune system of camelids,[30] and even less about immunology of the aging camelid. In humans and other domestic animals, old individuals have shown diminished resistance to disease. What might be a mild, easily treated infection in a five-year-old camelid may become severe illness in a twenty-five-year-old. In human medicine, old age may be the most common form of immune deficiency. Drugs may not be metabolized as readily and thus may be either less effective or more toxic.

Greater caution must be exercised with anesthesia because diminished liver and kidney function may slow metabolism of anesthetic agents. Elderly humans

may have lost approximately 30% of kidney function without ever having experienced specific kidney disease. The cardiovascular system is not as efficient in old animals, nor is respiratory function.

Feeding and Nutrition

Aside from the feeding problems associated with dental disorders, older animals have lower requirements for energy and protein. Overfeeding is a risk for older camelids. On the other hand, requirements for vitamins may be slightly higher. Periodic weighing and diet modification may help to keep older camelids in optimum condition.

In addition to free-choice forage a diet of equal parts alfalfa pellets, whole cottonseed, and flaked corn, with 1% molasses, has proven to be an effective geriatric supplement when it is up to 50% of the dry matter intake.[b]

Reproductive Performance

Theoretically, a healthy llama or alpaca female could produce a cria every year from the time she is two until she is twenty, but that is not likely to happen. A few female llamas have continued to produce into their twenties. A few females have produced seventeen crias, but a more realistic average is eight to twelve. Many factors determine productivity, and the aging process is one of them. Llamas and alpacas do not experience a menopause, so pregnancy may occur into old age. In old age, camelids may die from some other cause, with a fetus in the uterus. However, ovarian function may wane or cease entirely. The age at which this may occur is highly variable and may be genetically influenced in llamas and alpacas, as it is in all other domestic animals.

The signs of senile infertility are not different from signs of infertility in younger animals. More time and effort may be necessary to evaluate an older animal to establish whether or not a specific health problem exists, or if the reproductive tract is fully functional. A single visit evaluation is not likely to provide any meaningful information for management of the older female. Repeated, or even daily, examination may be required to evaluate the function of the ovaries. Evaluation of hormone levels is now possible. See Chapter 14 on endocrinology.

Crias born to aged dams may be denied adequate milk because mammary gland function may also diminish with age, as a result of scarring and inadequate hormone balance. The quality and quantity of colostrum produced by aged animals may be suspect because of diminished immune function.

ELECTROCUTION

Electrocution is the passage of electric current through or on the surface of the body to the ground.

Causes

There are two major causes of electrocution: lightning strike and contact with a high-voltage electrical current.

A lightning strike may be a direct hit, or the charge may spread through the roots of a tree or wet soil to an animal some distance away. Oak trees are prone to receive lightning strikes. An electric charge caused by lightning may also follow a metal fence or envelope a building. Unfortunately, camelids are prime targets for lightning strikes because of their high head carriage in either the standing or kushed position.

Camelids may chew through exposed electrical wires, be exposed to bare high-tension wires that fall during storms, or contact short-circuited electrically powered equipment (heating pads, electric blankets, heated waterers) or improperly grounded electrical devices. Also, faulty electrically powered water pumps may electrify metal water lines.

Clinical Signs

Camelids, like people, may survive a lightning strike. Unless the strike is witnessed, it may be difficult to make a diagnosis, but the following signs may be noted: depression, blindness, nystagmus, paralysis, and temporary unconsciousness. Characteristically, there is evidence of lightning damage in the area. Linear singe marks are definitive if present, but lightning may kill a llama without producing burns. One llama was struck while kushed in an open pasture. When the body was moved, the green grass had been singed where the llama had been lying. A single lightning strike may kill more than one animal, especially if a group has congregated under a tree for shelter. Lightning may kill so quickly that there is feed still in the mouths of grazing animals.

Camelids are sensitive to electricity. Usually, after the first contact, they avoid an electric fence. It is important to realize that a solid contact with the standard 100-volt current or household and barn electrical service is sufficient to kill animals. The amount of damage done depends upon how well the camelid is grounded (wet surfaces are more dangerous) and the duration of the contact with the current.

Electrocution usually shocks the heart, causing cardiac arrest or ventricular fibrillation. Electrical current often follows the course of nerves or blood vessels and may destroy those vital structures by the heat generated. Electrical current stimulates the nervous system and causes the muscles to contract, the strongest muscles overpowering the weaker ones. A serious problem in people who touch electrical wires is that the shock causes the hand to close and grasp the wire, thus continuing the contact with the current. An electrician always touches a suspect wire with the back

of his or her hand so that if the wire is hot, the shock will pull the hand away from the wire.

A source of annoying but rarely lethal electrical current is called "stray voltage." A llama owner reported an incident in which llamas in a herd of forty animals were reluctant to drink from the waterers provided. The ranch had a sprinkler system operating, and he noticed that the llamas were drinking from the sprinklers rather than the waterers. The owner observed one llama attempt to drink from a waterer, but it jerked its head upward just as the lips contacted the water. He put his hand into the water and felt a mild shock.[c] Using a voltage meter, he measured 11 to 22 volts between the waterer and the ground. He immediately contacted his local power company and ultimately the problem was minimized.

Stray voltage may arise from (1) faulty, improper, or outdated wiring; (2) poor grounding; (3) unbalanced 120-volt loads; (4) dirty or damaged electrical boxes; (5) neutral conductors that are too small; (6) poor connections on neutral or ground wires; (7) incorrectly grounded trainers or fences; (8) heavily loaded power lines serving a farm; or (9) power loads at neighboring farms.

Correction of the problem, or at least minimizing it, involves proper wiring and grounding. Help may be obtained from a utility company.

Prior to administering assistance, always make certain that the victim is disconnected from any electrical current. If the victim is still touching the downed power line, quickly determine if it is possible to turn off the current. Call the emergency line of the local utility company. Do not attempt to remove or cut the wires. If the voltage is too high, readily available insulation may not be sufficient to prevent electrocution of the rescuer.

Once the victim is freed from the electrical current, determine whether the heart is beating and/or the victim is breathing. Cardiopulmonary resuscitation is the only first aid assistance that may be effective. If the victim is in ventricular fibrillation, cardiac massage will not correct the problem, but massage may circulate sufficient blood to keep the victim alive until an electrical defibrillator can be applied in a hospital.

FAILURE OF PREDICTED GROWTH AND WEIGHT LOSS

Normal Growth[13,54,55]

The growth of seventeen llama crias from birth to one year of age was studied in Oregon. Weight was correlated with thoracic circumference, body length, and body height, but only body weight is illustrated in Table 9.2. Growth is most rapid during the first month. Daily gain from birth to seven weeks was 0.42 kg (doubling birth weight in thirty days). Daily gain from seven to twenty-four weeks was 0.29 kg, and from twenty-four to seventy-two weeks, 0.16 kg. The curve flattens, but there is no plateau; growth consistently increases.

Table 9.2. Growth of the llama.

Age (months)	*Mean body weight (kg)*	*Age (months)*	*Mean body weight (kg)*
1	21.9	13	87.5
2	31.9	14	91.1
3	39.7	15	94.5
4	46.3	16	97.9
5	52.3	17	101.1
6	57.7	18	104.3
7	62.7	19	107.4
8	67.3	20	110.4
9	71.8	21	113.3
10	75.9	22	116.2
11	80.0	23	119.0
12	83.8	24	121.8

Source: Smith et al. 1992.

In a second study at the same institution, growth was followed from birth to maturity in 270 llamas.[57] Full-term crias varied from 11 to 15 kg (average, 12.6 ± 1.3). Others have reported cria birth weights from 8 to 20 kg. Body height reached its maximum by eighteen months, but adult body weight was not reached until thirty-six months. Adult body weight (thirty-six months) in the study was 108.9 to 210.9 kg (average, 151.5 kg). Data from South America list adult weights ranging from 130 to 155 kg (average, 108.5 ± 20 kg). Growth is not constant except for the first few months of life. Later, periods of rapid growth are followed by periods in which growth is slowed.

Abnormal Growth

Various syndromes with failure to grow or weight loss as the primary clinical sign have been described in llamas and alpacas. Names applied to the syndromes include failure to thrive, ill-thrift, wasting disease, and "Ain't doin' well." Forms of growth failure may occur at specific ages. Various etiologies have been suggested, including failure of passive transfer of antibodies, hypothyroidism, anemia, congenital and acquired immunodeficiency, virus-induced immunodeficiency, parasitism, rickets, and malabsorption.

If abnormal weight loss is detected in a camelid adult, a detailed physical examination and laboratory evaluation is necessary to arrive at a diagnosis. Conditions to consider in a differential diagnosis include chronic disease, bacteremia, eperythrozoonosis, dental abscesses, neoplasia, and any disorder accompanied by anorexia. In juveniles, differential diagnosis should include inadequate milk production

by the dam, failure to convert to a solid diet, rickets (hypovitaminosis D and hypophosphatemia), parasitism (coccidiosis, gastrointestinal nematodiasis), immunodeficiency, anemia (eperythrozoonosis), chronic infection, protein-losing conditions, chronic blood loss, and dental disorders.

Efforts have been made to find a single etiologic agent causing these syndromes. A paper was published implicating a retroviral agent, but other retrovirologists have questioned the validity of that report. To date, no viral agent has been demonstrated in tissue or cultured from camelids with these syndromes. Although suspected, an inherited immune deficiency has not been verified. Other studies have implicated thyroid dysfunction or iron deficiency anemia. These may contribute to a syndrome but have not been proven to be the primary cause.

Immunodeficiency

An excellent study reported out of Colorado describes a syndrome characterized by an array of signs, laboratory findings, and lesions that are consistent with immunodeficiency as described in other species.[30] Conditions that are suggestive of an immunodeficiency disorder include repeated infections (poor response to therapy), high susceptibility to low-grade pathogens, systemic illness following vaccination with live-virus vaccines, lymphoid hypoplasia/atrophy, and infections with organisms rarely observed in immunocompetent animals.

Twelve juvenile llamas with suspected immunodeficiency disorders were examined and detailed laboratory investigations conducted. All the llamas died, and necropsies were performed. Deaths occurred from 9.4 to 29.3 months of age.

Clinical Signs

Weight loss or failure to gain were the most consistent signs, despite generally good appetites. Animals were presented with variable depression, nasal discharge, ocular problems, and lameness. Ages varied from four to 15.3 months. Body temperatures were normal to slightly elevated.

Diagnosis

Four of the twelve llamas had eperythrozoans on the erythrocytes. Anemia was pronounced in these animals but was not marked in others. All the llamas had hypoalbuminemia. Other serum biochemical evaluations were normal. Lesions included marked thymic hypogenesis/atrophy, lymph node hypoplasia, chronic inflammatory lesions in various organs, pneumonia, dermatitis, arthritis, orchitis, myocarditis, nephritis, and oral ulcerations. Lymphocyte blastogenesis assays indicated a severe defect in both T-cell and B-cell lymphocyte proliferative responses.

It was not possible to establish whether these animals were primary (genetic) immunodeficient or secondary (acquired). This group was evaluated between 1988 and 1990. A protocol was designed for a prospective study, but similar clinical cases have not been available.

RECUMBENCY[7,8]

SACs that are unwilling or unable to stand present a special challenge to owners and veterinarians who attend these animals. An SAC may be found in varying forms of recumbency. Recognition of the various forms allows the veterinarian to focus more precisely on etiology, resulting in more efficient therapy. Following are variations in the recumbent state.

1. Sternal, bright and alert, able to hold the head up, appetite good.
2. Sternal, anorectic, won't drink, slight depression.
3. Sternal, head and neck held back over the thorax.
4. Lateral recumbency, able to right self into sternal position, depression, head and neck positioned vertical to body.
5. Lateral recumbency, opisthotonos, marked depression, unable to right self into sternal position.
6. Body condition fair to good.
7. Body condition thin or emaciated.

Checklist of Conditions Resulting in SAC Recumbency

Nervous System

Head trauma (concussion, contusion, fracture, hematoma), space-occupying lesion (tumor, abscess, hematoma, cerebral edema), spinal lesion—trauma (cervical fracture, cervical luxation, thoracolumbar spinal trauma), peripheral nerves—trauma/degeneration (brachial plexus, radial nerve).

Diseases

Infectious diseases (equine herpesvirus type I, listeriosis, coccidioidomycosis, mucormycosis, rabies, encephalitis, extension of otitis interna, abscess in brain, beryllioses), parasitic diseases (toxoplasmosis, *Parelaphostrongylus tenuis* myelitis, tick paralysis, eperythrozoonosis), noninfectious diseases (copper deficiency, toxic agents).

Musculoskeletal System

Fracture of limb bones, fracture of pelvis, myopathy, foreign body in a foot, infectious pododermatitis, arthritis, borreliosis, spondylosis, discospondylitis, rickets.

Digestive System

Malnutrition, colic, perforated ulcer, intestinal obstruction, gastric ulcers, hepatopathy, severe gastrointestinal parasitism, acute abdomen, toxic plants (rhododendrons, yew, oleander).

Urinary System

Colic, urinary obstruction, ruptured bladder, uremia.

Cardiovascular System

Congenital defects (ventriculo septal defect, transposition of aorta and pulmonary artery, tetralogy of Fallot), arterial thrombosis (iliac), endocarditis, congestive heart failure, cardiomyopathy, shock.

Metabolic Disorders

Hypocalcemia, hypoglycemia, hypomagnesemia, hyperacidity (exertional stress), electrolyte imbalance (hypokalemia), malnutrition, hypoproteinemia, anemia, neoplasia, hypothermia, hyperthermia, grain overload.

General Septic/Toxic Conditions

Escherichia coli endotoxemia/septicemia, klebsiellosis, mycotoxicosis, pneumonia, enterotoxemia (*Clostridium perfringens*), septic mastitis/metritis, omphalophlebitis, snakebite.

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[a] L. Johnson, personal communication.
[b] L. Johnson, personal communication.
[c] D. Goodyear, personal communication.

10

Integumentary System

The integumentary system consists of the skin, hair (fiber), mammary glands, adnexal glands (sebaceous, sweat, scent), and toenails.

SKIN

The functions of the skin are manifold.[32,48] Primarily, the skin serves as a protective barrier between the body and the external environment and is an important organ for homeostasis, preventing excessive water loss and invasion of the body by pathogenic microorganisms. The flexibility and elasticity of the skin allow motion and provide shape and form. The skin also plays a vital role in thermoregulation and sensory perception.

The skin contains both sebaceous and sweat glands, which serve a secretory function. Melanin provides the variety of colors found in camelids. Precursors for vitamin D_3 synthesis in the skin are acted upon by solar ultraviolet light to produce the vitamin that is later converted to 1, 25 dihydroxycholecalciferol, the active hormone, via metabolism in the liver and kidney. Special appendages of the skin include hair, mammary glands, and nails.

The skin of camelids is thick and nonpliable. Scent glands are located in the interdigital spaces and the medial and lateral metatarsal regions. Callosities form over the sternum, carpus, and stifle in response to recumbency patterns. The anatomy of the skin of the llama and alpaca is similar and will be considered as an entity.

Anatomic studies of camelid skin are few in number, but a detailed study of the skin and adnexa has been published by Atlee.[2,3] This information is needed because the skin of camelids is unique among domestic animals, and few dermatopathologists understand the anatomy of normal skin, which complicates evaluation of pathologic states.

The layers of the skin are illustrated diagrammatically in Figure 10.1. The epidermis usually consists of four layers, rather than the five described in other animals. The *stratum corneum* (horny layer) represents one-half to three-fourths of the total epidermal thickness. This layer consists of anuclear remnants of flattened, fully keratinized cells pushed up from basal layers. A *stratum lucidum* is occasionally seen in sparsely haired skin as a dense eosinophilic layer beneath the *stratum corneum*. The *stratum granulosum* (granular layer) is a single layer of cells in some areas and discontinuous in others. The nuclei are pycnotic, and most of the cytoplasm has been replaced with keratin.

The *stratum spinosum* (prickle layer) is reduced in camelids but is composed of daughter cells of the basal layer and is one to three cells thick. These cells are viable and nucleated and actively synthesize keratin.[32] The *stratum basale* (basal layer, or *stratum germinativum*) is the deepest layer of the epidermis and consists of a single layer of cuboidal or columnar cells, most of which are keratinocytes with a few melanocytes.

Melanocytes contain melanin pigment in pseudopods distributed between epidermal cells of the skin and hair. Skin color is determined by the number, size, arrangement, and dispersion of melanin granules.[15,44] In chronic dermatitis, there may be overproduction of melanin, which causes darkening of the skin. Conversely, depigmentation may result from trauma, burns, or infection of the skin. Albinism occurs in camelids as it does in all species.

The dermis (corium) of camelids is thick (up to 1 cm in the cervical region of a mature male) and consists of a superficial layer comprised of loose connective tissue interdigitating with undulations in the epidermis and the deep dermis, which is comprised of dense

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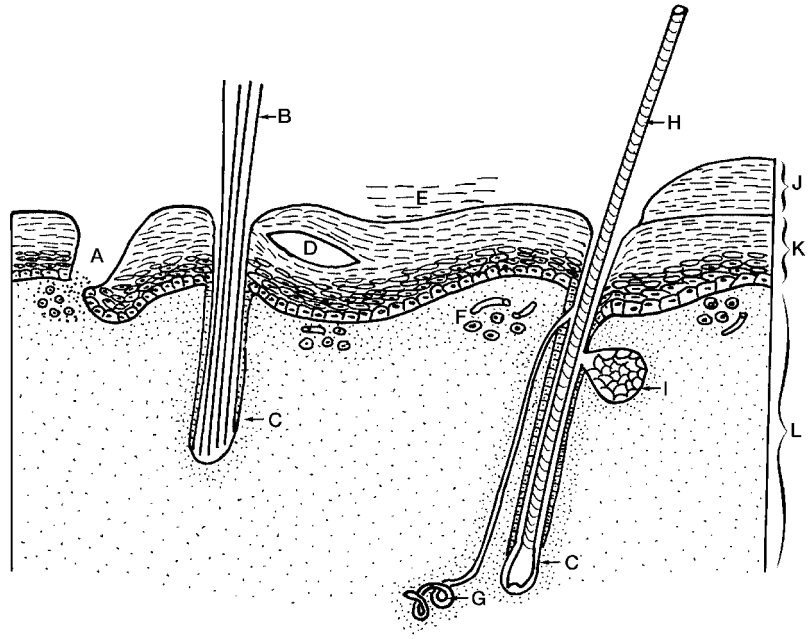

Figure 10.1. Diagram of the skin. (A) Laceration, (B) multiple fibers from a single follicle, (C) follicle, (D) vesicle, (E) desquamated epithelium (dandruff), (F) dermal blood vessels, (G) sweat gland, (H) fiber, (I) sebaceous gland, (J) hyperkeratosis, (K) epidermis, (L) dermis.

fibrous tissue. The dermis contains hair follicles, blood and lymph vessels, nerves, and sebaceous and sweat glands. The middermis of camelids is characterized by a proliferation of blood vessels, in contrast to that in other domestic animals. Vessel walls are hyalinized. This normal histologic picture is frequently misinterpreted as an abnormal vascular proliferation by the pathologist. There is variation in the thickness of the epidermal layers in various areas of the body. Furthermore, the degree of vascularity and mononuclear infiltration may vary.

The hypodermis (subcutis) is comprised of loose connective tissue, which attaches the skin to the underlying bones or muscles. Some sweat glands extend into the hypodermis.

HAIR (FIBER COAT, WOOL)[1,7,11,15,17,25,27,31,41,47]

Wool is the fine, soft, wavy hair forming the fleece of sheep characterized by its property of felting, made possible by the overlapping of minute surface scales.[1,7] Technically, the term "wool" should be restricted to the fiber from sheep. Although wool is commonly used to designate camelid hair in North America, in South America the term used is alpaca or llama fiber, but hair will be used in this chapter. Table 10.1 lists the characteristics of camelid and other domestic animal wool and hair. Table 10.2 defines terms used to describe hair.

The hair coat of a llama consists of two types of hairs, the coarse guard hairs and a finer undercoat. The coat of most alpacas consists of only undercoat hairs. Llama guard hairs comprise approximately 20% of the fleece and are 140–150 µ in diameter.[19] Guard hairs must be removed from the fleece prior to spinning and garment manufacture.

In Peru, alpacas are sheared every year. Previously, the fleece was allowed to grow up to eighteen months, but it has been found that fiber production is increased with more frequent shearing. Alpaca or llama hair is slow growing, and this fact should be kept in mind when contemplating clipping the coat from an area for diagnostic or therapeutic purposes. In North America, llamas are rarely sheared to harvest the fiber; rather, the fiber is harvested by brushing. Llamas have only a partial annual shed. Wild NWCs and camels shed annually. Shearing is becoming more common as a management tool to help llamas cope with hot, humid weather. Alpacas are commonly sheared annually, especially in warm to hot climates.

The density of the hairs varies with the location on the skin, with the thickest coat over the back and sides of the animal.[2] Fiber management and harvesting are important factors in alpaca management, but this has little medical significance other than to deal with hyperthermia. More detailed information about fiber production is found in The Alpaca Book.[19]

Hair follicles are tubular invaginations of the epidermis into the dermis. In camelids, there are two types: simple follicles containing a single coarse hair and complex follicles containing multiple fine hairs. Both sebaceous and sweat glands empty into the hair follicles.[2,3]

Table 10.1. Wool and hair characteristics.

	Diameter (μ)	*Cross section of hair shape*	*Medulla*	*Shed*
Sheep				
Merino (fine wool)	10–30	Circular to oval	Rare in fibers	None
Lincoln (coarse wool)	30–50		<35 μ	
Kemp fibers, guard hair	>70	Dumbbell (bilobed)	Heavy	Annual
Angora goat				
Mohair		Circular	Rare	
Kids	10–30			
2 years	20–50			
4 years	50–60			
Down goat				
Cashmere		Circular		Annual
Kashmir, Pashu	14–17			
Camel				
Bactrian	5–40		Interrupted	Annual
Dromedary	20–50			
Llama				
Undercoat	(26)	Circular to elliptical	Most are medullated	Partial
(woollike crimped)	10–40			
Outer coat	(70)			
(straight hairs)	40–150		Strongly	
Alpaca	(22)			
Huacaya	16–40	Smooth margin, staple crimped	Strongly	
	(23)			
Suri	16–35	Crenated margin	Strongly	
Vicuña	(13–14)	Circular to elliptical	Fibers less than 18 rare	
	10–30			
Guanaco	18–24	Circular to elliptical	Strongly	
Horse hair	(150)			

Note: Numbers in parentheses denote averages.

The fine hairs of the undercoat of llamas and alpacas have three distinct layers: the medulla, cortex, and cuticle.[12,29] In contrast to sheep hair, camelid hairs are strongly medullated, except for most of the guard hairs. The cortex is circular to elliptic in cross section. The cuticular layer is evident within the follicle but becomes flattened as the hair emerges.

Crimp is the natural curl or wave in a hair. The crimp and scaled cuticle of sheep wool hairs provide excellent binding of the hairs as they are spun into yarn. The crimp also provides resiliency, the garment conforming to changing shapes as it is worn.

There are two breeds of alpaca. The huacaya (Figure 10.2A) has a shorter fleece coat with moderate crimping of the hairs, while the suri breed (Figure 10.2B) has a longer fleece with no crimping of the hairs. A pure alpaca hair garment does not resume its original shape if stretched. Special procedures must be used to spin alpaca hair, and frequently a percentage of fine sheep wool is mixed with it to ease spinning and knitting and to improve garment usage. Llama hair is coarse and has no crimp. Garments made from llama hair have a harsher feeling than those made from alpaca hair.

A cottage industry for making llama-hair garments has developed in North America, while in South America llama hair is handled locally by the indigenous people and used to make blankets, ropes, bags (corresponding to burlap or jute bags) called costales, and inexpensive garments. Indigenous people say that llama hair sweaters are warmer than those made from alpaca hair, but recognize that llama hair is harsher and may cause the skin to itch.

Fine garments are made from pure alpaca hair or alpaca hair mixed with fine sheep wool or synthetic fibers. The bulk of alpaca hair is sold to companies that sort, grade, clean, and spin it into yarn. Although in years past the yarn was shipped to England for garment

Table 10.2. Definition of terms used to describe hair, wool, and camelid fibers.

Cotting—The entanglement of fibers into an irreversible matt.
Crimp—The natural curl or wave in a wool fiber.
Felting—Producing a compact, irreversibly entangled structure when subjected to friction in a soluble medium.
Fiber—The hair of animals other than sheep.
Fleece—The wool that covers a sheep or similar animal. The quantity of wool cut from a sheep at one time.
Kemp—Kemp fibers, guard hairs are coarse, relatively flattened fibers with a long tapering tip. They are periodically shed.
Luster—Silver luster from fine merino wool, silk luster in medium to long wool, and glass luster in mohair and other goat fibers.
Staple—(1) Locks or tufts of wool shorn from a sheep. (2) Natural fibers or cut lengths from filaments, also called staple length.
Suint—That part of the new fleece that is soluble in cold water. The dried residue of the sweat glands.
Wool—The fine, soft, curly hair forming the fleece of the sheep characterized by the property of felting due to the overlapping of minute surface scales.
Wool wax—The fatty product obtained from wool. Also known as wool fat or wool grease. Purified form known as lanolin. Produced by the sebaceous glands.

manufacture, much of the manufacturing is presently done in South America.

Hair Quality

Hair quality may be modified by such environmental factors as day length, temperature, elevation, and general nutrition, but the dominant factor in determining quality is heredity.

Hair Loss[33]

It is often difficult to establish the etiology of patchy or generalized loss of hair in SACs. Certain specific diseases, such as mange, cause alopecia, but pediculosis does not, unless the animal rubs the hair from such locations as over the spinous processes of the vertebrae. Alopecia may also occur at other sites of excessive wear, such as alongside the necks of animals fed in a slatted manger. Mention has already been made of alopecia over the bridge of the nose and on the inside of the pinnae.

Large patches of hair may be lost with no apparent inciting cause. There may be no evidence of an inflammatory response of the skin, yet when the skin surface is examined carefully, it will be noted that the hairs have broken off. This is likely similar to a condition called "wool break" in sheep. The term "break" may be used to describe the actual breaking of the hair as a result of excessive thinning or the visible line on a fleece caused by the thinning of the diameter of the hairs. Studies have not been conducted to establish breaking as a phenomenon occurring in llamas and alpacas, but it is reasonable to assume that it does.

Figure 10.2A. Hucaya fiber showing crimp.

Figure 10.2B. Suri fiber.

To explain this phenomenon, it is necessary to understand that such a change in the diameter of the hair can occur only in animals that have a long growth cycle (anogen) (sheep, llamas, alpacas). Wild sheep and most other domestic animals shed wool and hair annually, and the fleece grows back to a genetically predetermined length. In anogen, various nutritional, metabolic, and environmental factors may influence the development of the hair while it is still in the follicle.

Sheep wool grown during the winter is finer (the hairs are thinner in diameter) than that grown in summer. Studies have shown that this is not a result of cold weather but rather a response to the shorter day length. Poor general nutrition may also cause thinning. If both the foregoing are combined, the hairs may become fragile and break easily.

Sheep that have been stressed by illness or fever develop a break line in the hair that is growing in the follicle during that time. The entire fleece may be lost in one or two weeks. Thinning of the hairs can be produced by the administration of cortisol parenterally, indicating that any stressor may cause thinning of the hair and a potential break.

Lack of any essential nutrient in the diet may contribute to the production of hairs that are uneven and fragile. A deficiency of dietary copper weakens wool hairs. Because molybdenum has an inhibitory effect on copper metabolism, animals fed forage grown in areas known to have high concentrations of molybdenum may develop a copper deficiency. Low-protein diets, especially if deficient in sulfur-containing amino acids such as cysteine, result in thinning of the hairs.

Thyroid hormone stimulates growth (both length and diameter) of hair. Estrogens tend to promote finer hairs, and testosterone, coarser ones.

In sheep, specific bacterial and fungal diseases of wool cause the hairs to deteriorate. Such diseases have not been identified in camelids.

MAMMARY GLAND[18,26,42,43,46,49]

There are four nipples on the mammary gland of both Old and New World camelids. There may also be supernumerary teats, either cranial or caudal to the normal teats, some of which may connect with glandular tissue. In SACs each nipple has two streak canals that enter into separate teat and gland cisterns (Figures 10.3, 10.4). Camels have three streak canals. Variable numbers and sizes of milk ducts collect milk from the gland and empty into the gland cistern.

Although the glands and milk ducts draining into a single teat overlap and interdigitate with one another, they are separate and distinct, with no anastomosis as determined by evaluation of latex cast preparations (Figure 10.4) and contrast radiography. Thus, in effect, there are eight mammary glands. Various teat malformations occur (Figures 10.5, 10.6). Double teats have been identified (Figure 10.7).

The streak canals of camelid teats are short (2 mm) and have such a narrow diameter that

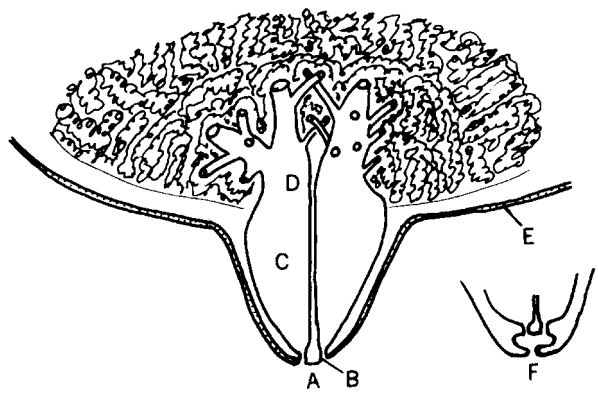

Figure 10.3. Diagram of a camelid teat and collecting system. (A) Streak canals opening on tip of teat, (B) streak canal, (C) teat cistern, (D) mammary gland cistern, (E) skin, (F) alternate morphology—streak canals emptying into a sinus with a single external orifice.

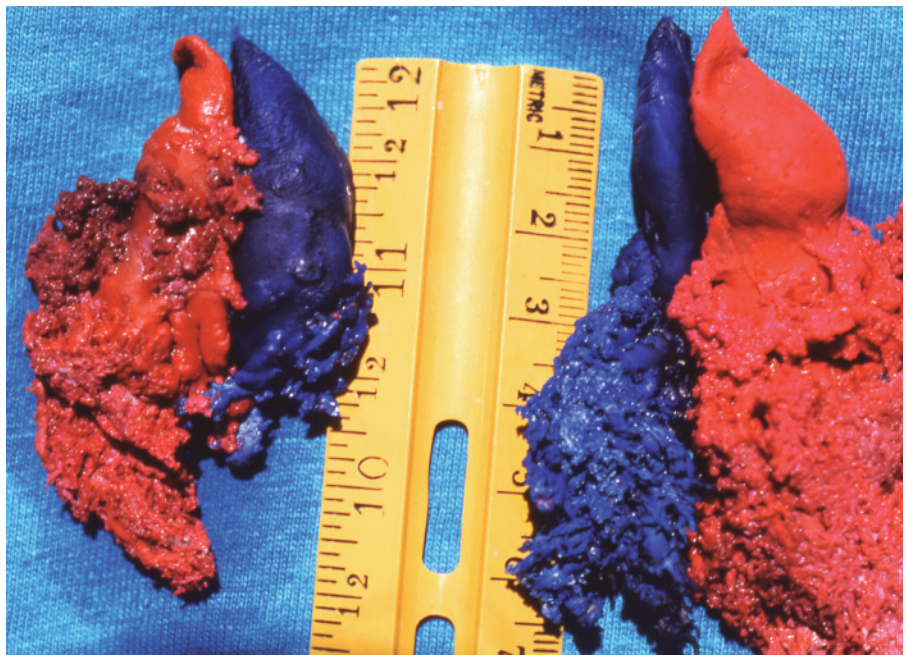

Figure 10.4. Latex cast of mammary gland-collecting system.

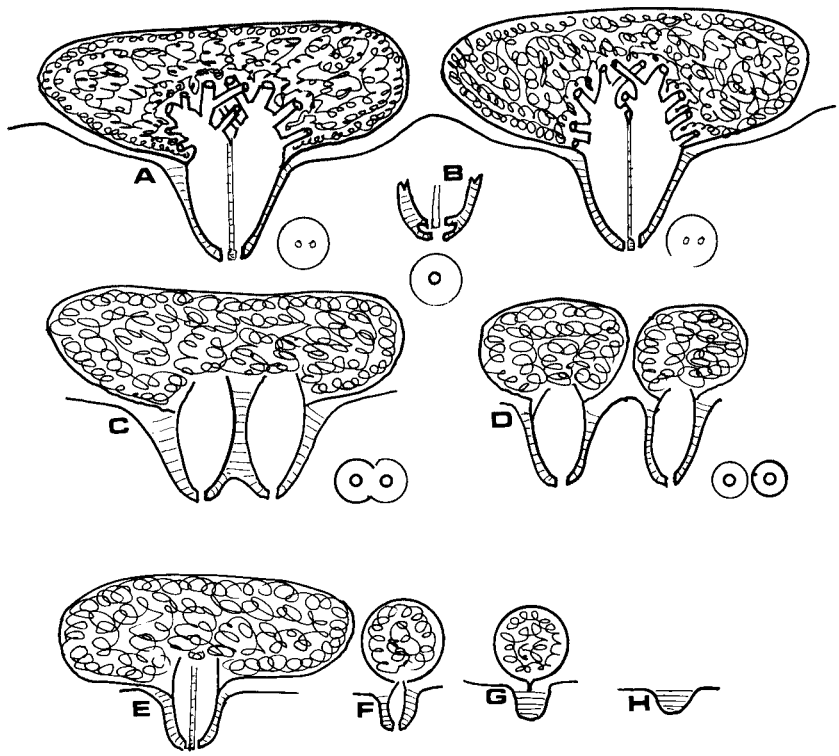

Figure 10.5. Diagrams of the side view of teats: (A) Normal, (B) alternate orifice, (C) partial separation of teats at a normal location, (D) double teats at a normal location, (E) small teat, may be one of the four normal teats or a supernumerary teat, (F) supernumerary teat with a functional gland, (G) supernumerary blind teat associated with glandular tissue, (H) supernumerary teat with no attachment to glandular tissue.

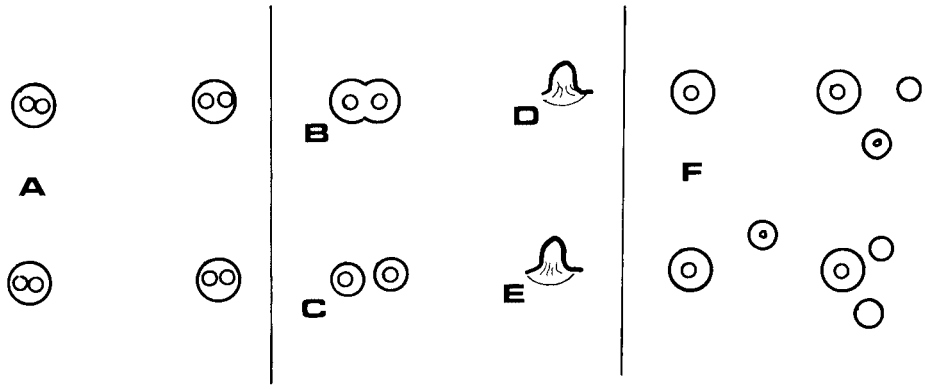

Figure 10.6. Diagram of the ventral view of teats of a camelid: (A) normal configuration, two openings at the tip of each teat, (B) fused double teat, (C) separate double teat, (D) lateral deviation from the vertical axis, (E) medial deviation from the vertical axis, and (F) possible locations of supernumerary teats.

conventional tubes used to instill antibiotics into the udder of a cow or sheep are too large. A 1-mm (3.5 French) catheter is about the maximum usable size. The streak canals may exit the teat on the conical surface of the tip of the teat, or both canals may exit into a sinus at the tip of the teat, giving the erroneous impression that there is only one orifice (Figure 10.3). The teat and gland cistern are not separated. The combined cistern is approximately 2.5 cm long and 1 cm in diameter.

The glands are compound tubuloalveolar glands similar to those of cattle. In SACs the right and left halves of the mammary gland are separated by an incomplete suspensory ligament of the udder. The front and rear quarters cannot be visually or surgically separated, but there is no connection between the collecting systems of the two quarters. The udder of the camel is similar, except that there are two or three teat ducts with independent teat cisterns leading to the gland cistern.

Figure 10.7. Double teat in a llama.

The recognition that there are eight separate glands is important clinically in the management of mastitis. If infusion of antibiotics into the gland is prescribed as a treatment for mastitis, it is crucial that the appropriate gland be infused.

Hand milking a llama is a challenging task because of the short teat. At best, a thumb and one or two fingers are all that can be used. Even then, some llamas have such short teats that they are difficult to grasp. The cria may fail to grasp such short teats as well. This is particularly true if there is edema of the udder at the time of parturition. A technique using a 10- or 20-ml plastic syringe as a breast pump has been described.[37]

Old World camels are milked routinely to provide milk for people. A dairy industry is developing in the Middle East to make cheese, ice cream, and other dairy products.

Supernumerary Teats

Supernumerary teats (polythelia, accessory teats, rudimentary teats) are those in excess of the normal number of four for camelids. They occur in all species of livestock, companion animals, and humans. In SACs, two glands and duct systems are associated with each teat, so there are eight distinct mammary glands. Old World camels may have two or three openings on the teat end, but this author is not aware of the details of the distinctness or the separation of individual glands. Although quite rare, a lesser number of teats may be present (subnumerary teats, hypothelia). Supernumerary teats have been determined to be an inherited, simple, recessive trait in cattle and are assumed to be recessive in other species.[26] Other types of teat anomalies are seen (Figures 10.5 and 10.6):

1. Short teats
   a. Functional, associated with glandular tissue
   b. Nonfunctional, associated with glandular tissue, but no teat canal
2. Introverted (innies)
3. Excessively long or bulbous
4. Small dermal enlargement (rudimentary)
5. Divergence from the vertical axis
6. Partial separation of paired teats at a normal location (Figure 10.6)
7. Complete separation of paired teats at a normal location

Camelid mammary gland embryology has not been reported. A brief discussion of the embryology of the bovine mammary gland may assist in a greater understanding of the development of supernumerary teats.[4] The mammary gland is a modified sweat gland. The site for future development is first noticed on the bovine embryo at 2.5 cm, by two milk lines of ectoderm that run caudally from the umbilicus. The cell layers at specific places on this line proliferate and become the mammary hillock, which in turn enlarges to become an ovoid mammary bud (Figure 10.8). By 19 cm, the mammary bud elongates (duct anlage) into the mesenchymal tissue, and a lumen develops at the tip of the duct anlage, which is to become the duct system. The tip of the developing teat is an epidermal cone with no teat canal present. By 35 cm the proliferation of the duct system has progressed to form the gland cistern and the teat cistern. The epidermal cone begins to separate, from the inside, to form the teat canal.

Once the duct system is formed, further development is arrested until the hormonal changes (estrogen) associated with puberty stimulate glandular development. The ultimate development of the glandular tissue is determined by genetics, nutrition, presence of excessive fat in the gland area, and hormonal stimulation during the terminal stages of gestation.

Supernumerary teats may develop in all stages of the embryologic progression. Most of them will be associated with a column of tissue (duct anlage), which, when palpated, gives the impression of glandular tissue. The separation of the epidermal cone, to become the teat canal, may or may not be completed. When examining immature animals, it is difficult to differentiate between functional and nonfunctional supernumerary teats.

An interesting sidelight for North American breeders is that a high plane of nutrition (particularly excess energy) just prior to puberty results in lower mammary secretory tissue weight and is associated with low milk production in subsequent lactation.

The total weight of the SAC udder in a nonpregnant mature female is approximately 250 g. Udder enlargement may begin approximately two months before parturition, especially in a primiparous female. The udder of a fully lactating female is relatively small,

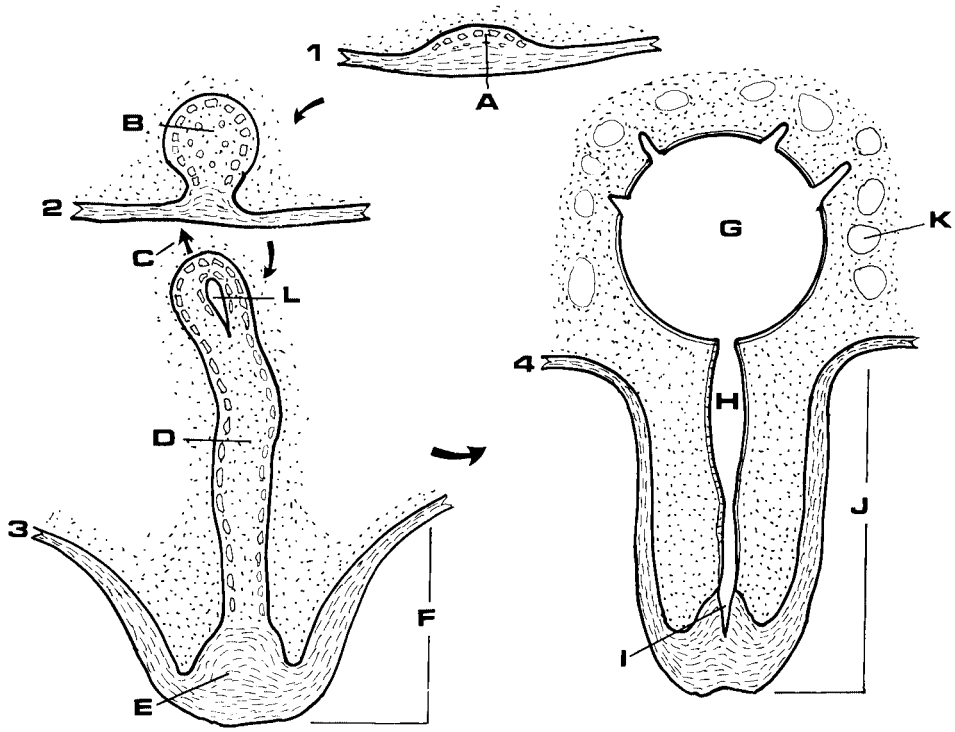

Figure 10.8. Diagrams of early embryologic development of the bovine mammary gland and teat. (1) Embryo size 2.5 cm, (2) embryo size 3.8 cm, (3) embryo size 5 cm, and (4) embryo size 27.5 cm. (A) Mammary hillock, (B) mammary bud, (C) direction of growth, (D) duct tissue, (E) skin cone, (F) developing teat, (G) gland sinus (milk cistern), (H) teat sinus (teat cistern), (I) teat canal (lactiferous duct, streak canal), (J) developing teat, (K) developing fat tissue, and (L) beginning of formation of the duct system. Diagram modified from Hibbitt et al. 1992.

weighing only 450 g, and may appear empty all the time if nursed properly. As in all species of mammals, a genetic component determines mammary gland size and milk production. Breeding female llamas and alpacas have not been evaluated on the basis of milk production, nor is any information available on evaluation of the udder.

Reports of camelid milk composition in the past have been variable. Recent studies, using large numbers of llamas from different regions of the United States, give more credibility to milk analysis.[29] More detailed information can be found on other species.[6,9,10,12,20–23,30]

Alpaca and llama milk is similar and may be considered together.[9,45] Moro describes alpaca milk as having a white porcelain color and an odor similar to that of cow's milk. Alpaca milk has a sweetish taste and is more viscid than cow's milk. The fat content varies from 0.7% up to 5.7% during the course of a lactation period, but with marked variation from day to day. Morin did not find such variation in llama milk in his study.[41] Alpaca milk has a pH of 6.4 to 6.8 and is more acidic than cow's milk. The volume of milk production ranges from 40 to 1,200 ml per day, but most animals produce less than 320 ml.

Consult Table 10.3 for composition values for the milk of camelids and other species. Numerous factors affect the composition of milk, including genetic predisposition, nutrition, stage of lactation, age of the female, season, ambient temperature, and presence of chronic infection of the glands.

It may be of interest to compare the milk of SACs with that of their Old World cousins.[12] Camel milk is used extensively for human food. The Koran speaks of God answering the prayers of the desert people by giving them the she camel that they may drink her milk. Camels are uniquely adapted to subsistence on harsh, dry, desert herbage and intermittent supplies of drinking water while continuing to produce milk that is suitable for camel calves and human infants alike. Mammals adapted to a temperate climate cease to lactate when subjected to drought and dehydration. Not so the camel.[52,53]

The normal composition of camel milk is similar to that of SAC milk (Table 10.3). Milk yields of camels vary from 3.5 to 14 kg per day, with lactation yields (nine to eighteen months) totaling 1,000 to 4,000 kg. Camel milk has a sweet, sharp taste and sometimes is salty. Milk taste and odor are modified by plant ingestion and other environmental conditions. Camel milk

Table 10.3. Milk composition (as fed).

Species	*Water (%)*	*Total solids (%)*	*Protein (%)*	*Fat (%)*	*Lactose (%)*	*Kcal/L*
Llama	86.9 (79–89.5)	13.1	3.4	2.7	6.5	822
Goat	87	13	(2.1–4.4)	(1.3–5.9)	(5.2–7.7)	1680
Sheep	81	19	6.2	7.9	4.8	1138
Cow	87.3	12.7	3.3	3.6	4.8	653
Camel	86.5 (85.02–88.5)	13.6	3.6 (3.01–4)	4.5 (2.87–5.38)	5 (3.36–5.8)	

Source: Morin et al. 1995a and Yagil 1982.

is rich in vitamin C, three times higher than cow's milk and 1.5 times higher than human milk. It is an important source of vitamin C for humans in a vitamin-impoverished environment. The fat droplets in camel milk are small and do not rise to the top of standing milk.[51,52]

Cattle, sheep, and goats produce milk with a higher than normal percentage of total solids when water is scarce. Such milk is contraindicated for the health of nursing young. The camel, on the other hand, produces milk with 4.5% fat and 84% water when provided with optimal water, but when chronically dehydrated, the fat content drops to 2%, and the water content elevates to 90%. Such milk admirably serves the needs of camel calves and humans alike as a source of both moisture and nourishment.

The secretion of dilute milk by a hot desert-adapted camel is explained on the basis of the effect of the pituitary antidiuretic hormone on secretion of water by the mammary gland (the mammary gland is a modified sweat gland). Camels have minimal sweat glands otherwise, which is one of the many physiologic adaptations that aid them in dealing with high temperatures and dehydration.[52,53]

OTHER SKIN GLANDS

Adnexal Glands

In camelids, sebaceous (holocrine) glands are associated with each hair follicle, but the production of sebum (wool wax, wool fat, wool grease, lanolin) is low in comparison to that of sheep. Sebum is a mixture of waxes, cholesterol, and cholesterol esters that, when mixed with sweat, coat the skin and wool hairs to enhance water repellency, inhibit microorganism penetration, and inhibit dehydration via the skin.

Clippers used to shear sheep are naturally lubricated with wool wax as shearing proceeds. The lack of wool wax in camelids causes rapid overheating of sheep clipper heads. Clippers used to shear llamas and alpacas must be dipped alternately into oil and water for cooling. The blades oscillate at a rate of approximately 300 per minute in contrast to sheep clippers that oscillate at about 1,000 per minute.

Sweat (apocrine) glands are generally found widely distributed over the surface of the skin but are more dense on the ventrum, which is sparsely covered with hair (thermal window). The sweat glands are poorly developed in llamas and alpacas and consist of simple, tubular, or unilobular glands; sheep, on the other hand, have multilobular glands.[2,3]

Metatarsal Glands

Camelids have unique, oval-shaped, hairless patches on both the medial and lateral surfaces of the metatarsal regions of the rear limbs (Figure 10.9A).[2,3] Associated with the patches are multilobulated holocrine glands, with ducts emptying on the surface. The dermis is markedly papillated, corresponding to invagination into the epidermis. The function of these glands is probably the excretion of alarm pheromones, perceived as "burned popcorn" odor to humans. Glandular secretions solidify upon excretion into leathery sheets that can be peeled off the surface of the skin. Some references describe these structures as chestnuts, but histologically, they are not comparable to the chestnuts of horses.[2,3,40]

Interdigital Gland

Interdigital glands are found on all four feet of SACs. The structure and specific function of these glands is unknown, but they are probably associated with individual and group identification (Figure 10.9B).

Poll Glands of Camels

Camels have a scent gland on the poll, between and just caudal to the ears. The gland is quiescent except when the male is in rut, at which time the gland secretes a brownish-black fluid with an offensive odor (to humans). When in rut the male camel may roll and rub the back of the head on the ground to scent mark.

Figure 10.9A. Metatarsal scent glands of the llama.

Figure 10.9B. Interdigital scent gland in a llama foot.

FOOT[13]

The camelid foot is unique, with two digits on each foot. The plantar surface is covered with a soft, cornified layer of epithelium similar to that of the bulb of the heel in sheep and goats (Figure 10.10). This is called the slipper. In SACs there is a separate slipper for each digit, while in the camel a single slipper covers the entire bearing surface. Deep to the slipper is a layer of dense connective tissue, the corium, containing blood vessels and nerves.

Camelids are modified digitigrades. In SACs phalanx 2 (P-2) and P-3 lie horizontally within the foot and P-1 upright at approximately 45 degrees (Figures 10.11 to Figure 10.14). In camels, P-2 and P-3 are more vertical. A small, nonweight-bearing nail, similar to a human nail, is located at the extremity of each digit and closely attached to P-3 via the corium.[20] Primary nail growth occurs at the coronary band.

The suborder name for camelids is Tylopoda, meaning padded foot, so named because of the digital cushion interspersed between the slipper, corium, and P-2 and P-3.[22] The digital cushion consists of a poorly vascularized meshwork of collagenous and elastic fibers with interspersed masses of fat and, occasionally, cartilage tissue, all encased in a fibrous sheath (Figure 10.15).[38,39] The toenail has laminae which attaches the nail to P-3, Figures 10.16A and B and 10.17. The camel foot is illustrated in Figures 10.18 and 10.19.

DIAGNOSIS OF DERMATOLOGIC CONDITIONS

A general protocol for differential diagnosis of dermatologic conditions should include the following in the approximate order of listing:

1. Conduct a complete physical examination to exclude systemic diseases that may produce a secondary dermatosis.

Figure 10.10. Bottom view of llama foot.

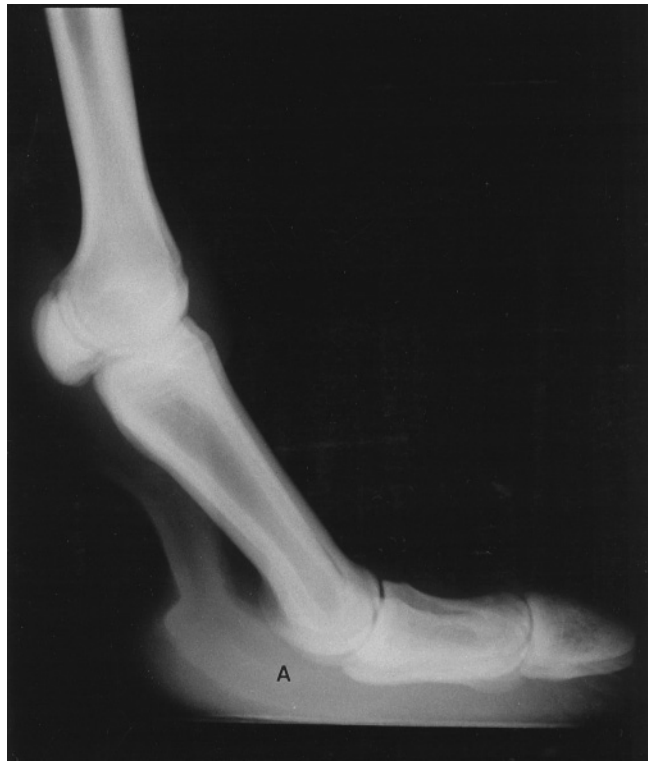

Figure 10.12. Lateral radiograph of the foot, pastern, and fetlock of a llama. (A) Digital cushion.

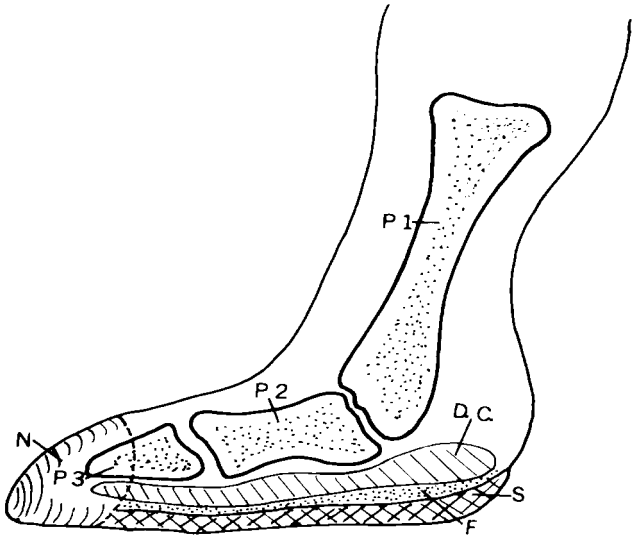

Figure 10.11. Diagram of SAC foot and pastern. (N) Toenail, (DC) digital cushion, (F) sole corium, (S) sole (slipper).

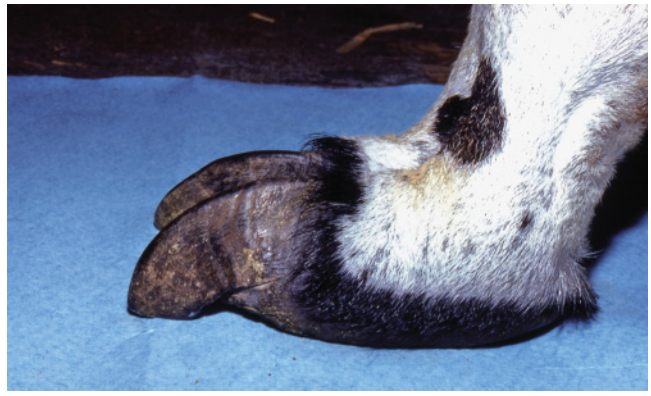

Figure 10.13. Normal llama foot, lateral view.

2. Obtain a complete history. In addition to information collected in the general examination, determine how long the dermatosis has existed. Is one animal or are multiple animals affected? Are the lesions enlarging or spreading to other areas of the body, face, or limbs? Is the animal pruritic? Dermatoses that cause pruritus include sarcoptic and chorioptic mange, pediculosis, food allergies, inhalant allergies, contact dermatitis, and zinc-responsive dermatosis.
3. Establish and record the distribution and size of the lesions.
4. Pluck hair samples from the margin of the lesion and prepare a hot KOH preparation.
5. Perform a deep skin scraping from a number of sites to check for presence of mange mites.
6. If pyoderma is suspected, collect skin cultures as per the same from dogs or horses.

Figure 10.14. Normal llama foot, dorsal view.

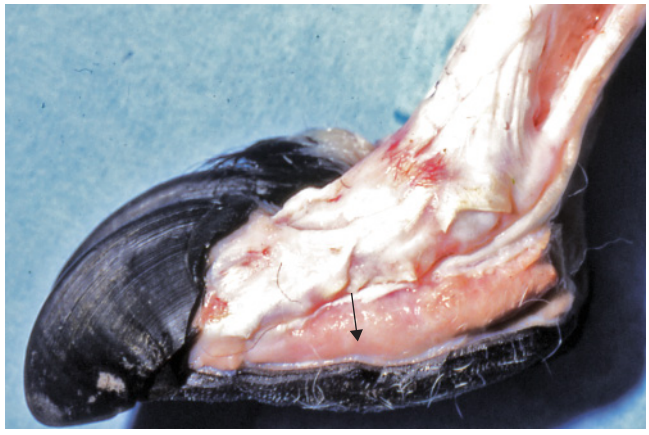

Figure 10.15. Digital cushion, lateral view.

7. Culture skin scraping material on Sabouraud's media.
8. Collect multiple skin biopsies from active and less active lesions.
9. Treat and observe the response to treatment.

Many camelid dermatoses are undiagnosed as to etiology. (Figures 10.20, 10.21). Alopecia and chronic dermatitis over the bridge of the nose (most commonly

Figure 10.16A. Toenail of a camel.

Figure 10.16B. Lamina of a toenail of a camel.

of dark-skinned llamas and alpacas) frequently occur (Figure 10.22). The etiology may be multifactorial, including a vice of rubbing the nose, fly bite irritation, dermatophytosis, solar dermatitis, or an extension of some of the more generalized dermatoses.

DISEASES OF THE INTEGUMENT[14,16,34]

Definition of Terms

The dermatopathologist uses special terminology to describe lesions of the integument. Following are definitions of a few terms, from Muller et al.,[32] that have relevance to the discussion of integumental diseases in camelids.

Acanthosis is diffuse hypertrophy of the prickle cell layer of the epidermis. Acantholysis is loss of cohesion

Figure 10.17. Lamina of toenail of a llama.

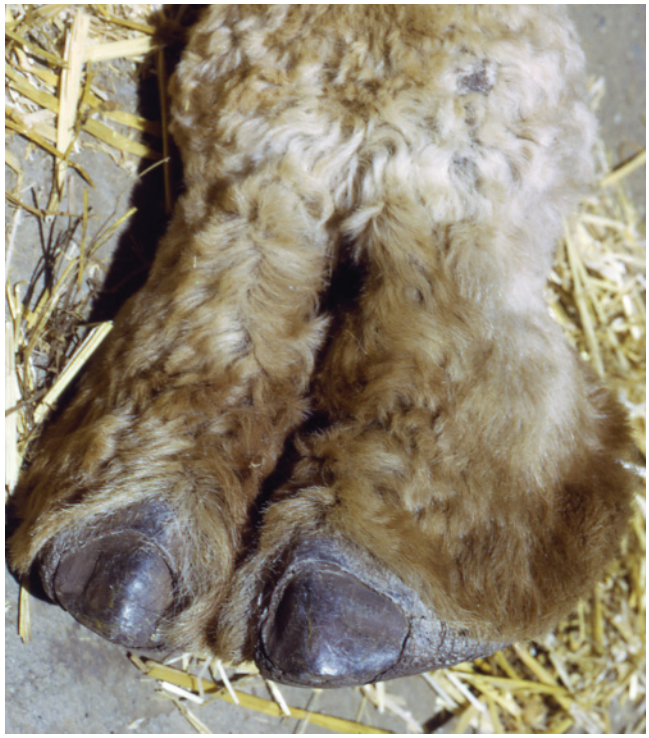

Figure 10.19. Camel foot dorsal view.

between epidermal cells. Hypergranulosis is increased thickness of the *stratum granulosum*. Hyperplasia is increased thickness of the noncornified epidermis. Hyperkeratosis is increased thickness of the *stratum corneum*. In normal skin of most species, the epidermal renewal time is thirteen to twenty-two days. With inflammation, the renewal time may be reduced to three to five days, resulting in thickening (stacking) or

Figure 10.18. Bottom of a camel foot.

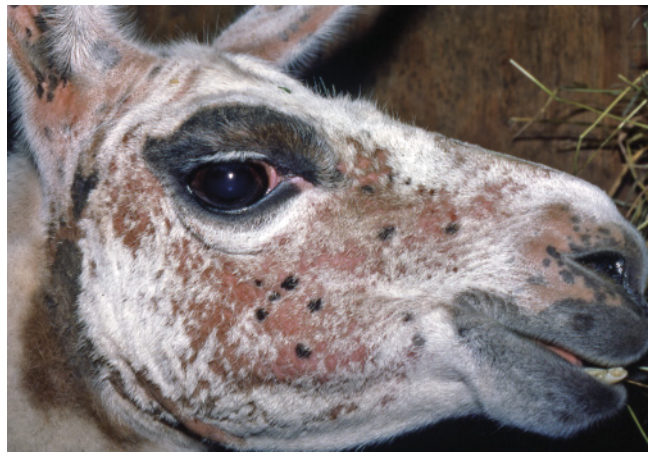

Figure 10.20. Acute facial zinc-responsive dermatitis.

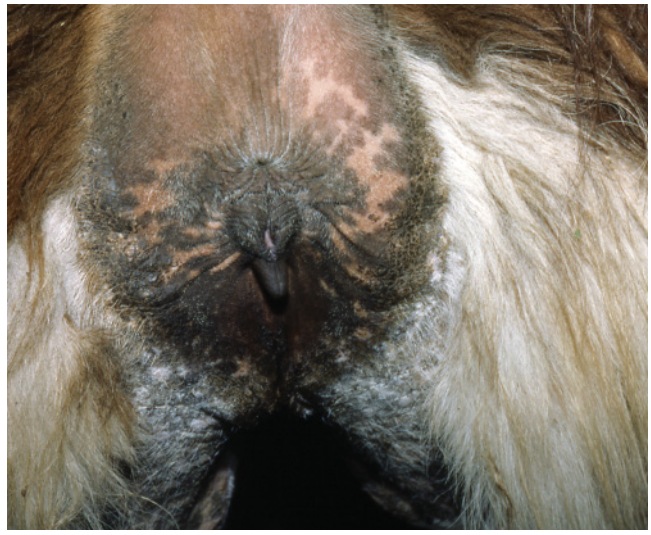

Figure 10.21. Acute nonspecific dermatitis.

Figure 10.22. "Dark nose" dermatitis in an alpaca.

excessive scaling. Parakeratosis is abnormal retention of keratinocyte nuclei in the *stratum corneum*.

Traumatic Lesions of the Skin

Lacerations, contusions, abrasions, and puncture wounds of the skin occur in camelids as they do in other animals. The management of such injuries is similar to that used for livestock and companion animals. Camelids should be immunized against tetanus. If an individual has not been immunized, whether or not tetanus antitoxins should be administered may be controversial. Several cases of tetanus in camelids have been reported, and one of these was produced experimentally. Apparently, camelids are much less susceptible to the organism than horses. Tetanus antitoxin is prepared in horse serum; therefore, there is a risk of anaphylaxis when the antitoxin is administered to animals other than the horse. Even if there is no immediate reaction, the camelid may become sensitized to horse serum, and anaphylaxis is a possibility if such a product is administered in the future. It is recommended that tetanus antitoxin not be used; rather, tetanus toxoid and benzathine penicillin should be administered at three-day intervals until the wound heals. Tumors of the integument are not common. See Chapter 9 for details.[8,35,36]

Parasitic and Infectious Diseases

Parasitic diseases of the skin include mange (sarcoptic, chorioptic, psoroptic, demodectic), pediculosis (*Damalinia* spp., *Microthoracius* spp.), and myiasis (Chapter 8). The few infectious diseases of the skin include staphylococcal folliculitis, dermatophytosis, contagious ecthyma, and coccidioidomycosis. These clinical syndromes are described in Chapter 7. Dermatophilosis (*Dermatophilus congolensis*) has been diagnosed, particularly in hot, humid regions of the United States. Dermal plaques are frequently seen on the surface of the pinnae caused by *Candida albicans*.

Zinc-Responsive Dermatosis (Idiopathic Hyperkeratotic Dermatosis)

Zinc deficiency dermatosis has been described in swine, sheep, goats, and laboratory animals. Parakeratosis is a prominent feature of the disease in swine and laboratory animals but not in sheep and goats. In SACs, the lesions ascribed to zinc deficiency have been hyperkeratosis with variable perivascular infiltrates of lymphocytes, plasma cells, macrophages, and eosinophils. The condition may be seen in SACs of any age but is usually found in mature adults.

The clinical syndrome in SACs is characterized by alopecia, dermal thickening, scaling (Figure 10.20), and hyperpigmentation involving the face, ventral abdomen, medial thorax, and thighs. Animals may be mildly pruritic or nonpruritic. Black SACs may have a predilection for the disease. A tentative diagnosis may be made by finding plasma zinc levels of less than 0.5 mg/dl and evaluation of a biopsy.

Care must be taken in obtaining samples for zinc analysis. Erythrocytes contain large amounts of zinc, so if cells are allowed to hemolyze, false elevated plasma levels will be found. Collect blood in a heparinized tube (green top), and mix gently by rolling without allowing the blood to contact the stopper. Keep the tube upright, spin immediately, and harvest the plasma for analysis. Zinc levels were determined in twenty-one adult llamas in Colorado. The mean was 0.51 ppm with a range of 0.27 to 0.81 ppm.[39]

Zinc deficiency may be the result of an absolute dietary deficiency, poor absorption from the intestine, or the binding of zinc with calcium in the intestinal tract. Zinc is a required trace element. Pharmacologically, it is an immune system modulator and has some anti-inflammatory effects as well.

Zinc-responsive dermatoses are treated by supplying zinc sulfate in the diet at a dosage rate of 2 g/day. Improvement requires at least thirty days of supplementation. A more easily assimilated zinc supplement is zinc chelated to methionine (ZinPro) at 4 g/day (Chapter 2). Nonspecific dermatoses of camelids are often treated empirically by adding supplemental zinc to the diet. Colleagues report variable success as a result of this treatment, possibly because a precise diagnosis has not been made and all dermatoses are treated alike.

Idiopathic Superficial Neutrophilic/Necrolytic/Hyperkeratotic Dermatosis

As the name "idiopathic superficial neutrophilic/necrolytic/hyperkeratotic dermatosis" suggests, this is

a catchall dermatosis that may have multiple etiologies.

Clinical Signs

The lesions are generally in the axilla, inguinal area, ventral abdomen, distal extremities, perineum, and face (Figures 10.21). Animals are usually not pruritic. Erythema, crusting, and vesiclelike structures indicate necrotic epidermis. The severity of the lesions may periodically improve, only to become worse.

Diagnosis

Microscopic lesions include ballooning degeneration of the superficial epidermis followed by a neutrophil infiltration. Microabscesses may develop in the substratum corneum. Additional lesions include orthokeratotic and parakeratotic hyperkeratosis with a tendency to palisade. Nuclear and cellular debris may alternate with hyperkeratotic debris.

Therapy

Secondary bacterial infection must be handled with antibiotics. Some of these cases respond to oral corticosteroid therapy. A suggested regimen is to administer prednisone 2 mg/kg/day orally for one to two weeks. If responding well, decrease to 1 mg/kg/day orally for an additional two weeks, then administer this dose every other day for an additional two weeks, and then discontinue therapy.

Burns

Burns are caused by contact with dry heat (fire, friction), moist heat (steam, hot liquid), chemicals (caustic or corrosive substances), electricity (current or lightning) or electromagnetic energy (sunburn, X-rays). In camelids, burns are most likely sustained in barn or forest fires or automobile accidents, or they result from entanglement and struggling in ropes. Light-skinned camelids are at risk of sunburn after shearing. The same is true of animals that have undergone a surgical procedure necessitating close clipping of an area that may be exposed to sunlight.

Clinical Signs

Burns range in severity from reddening of the skin (first degree burn), to blistered, swollen skin (second degree burn), to charred skin with damage to deeper structures (third degree burn). The surrounding fiber may become matted with fluid escaping from the injured skin. Burned areas are painful to the touch. In first degree burns the superficial layers of the skin will slough in three to five days.

Severe burns open the skin to the invasion of infectious agents and the loss of fluids. The danger depends on the area of the body affected and the degree of damage.

Management

If the burn is witnessed, cooling the area by spraying or immersing in clean cold water may be beneficial. The application of an ice pack is indicated if available. Second and third degree burns should be covered with a nonsticking dressing to minimize fluid loss and prevent contamination and infection of devitalized tissue. Do not open blisters.

Chemical burns present different challenges. Any residual substances should be removed with clean water. Avoid using soap or disinfectant solutions unless the specific caustic chemical is known. Phenolic disinfectant burns may be neutralized with rubbing alcohol. Silver nitrate, found in some styptic sticks, should be neutralized with a saline solution.

Miscellaneous Dermatoses[28]

Congenital ichthyosis has been reported in a llama.[5] Photosensitization may occur with hepatopathy (Figures 10.23, 10.24) or ingestion of primary photosensitizing agents. Other conditions include neoplasias (papilloma, mast cell tumors) and dark nose alopecia.

Foot Diseases of Camelids

Overgrowth of the toenail is the most common disorder of the SAC foot (Figures 10.25 to 10.29). The cause may be insufficient wear or congenital curling of the toenail in which the nail is pushed out of position for normal wear. The toenails should be trimmed as needed, using Burdizzo sheep hoof trimmers, hand pruning shears, equine hoof nippers, or small trimmers designed especially for camelids (Figure 10.30). Some SACs have a heavy ridge on the forward surface

Figure 10.23. Photosensitization of the muzzle associated with fascioliasis.

Figure 10.24. Photosensitization of the ears.

Figure 10.25. Elongated toenails on a llama.

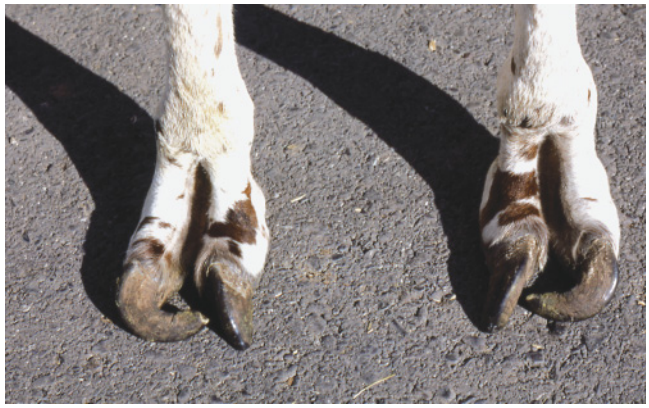

Figure 10.26. Elongated toenails on a llama.

Figure 10.27. Elongated toenails, both curved to one side.

Figure 10.28. Elongated toenails, both curved outward.

Figure 10.29. Elongated toenails, both curved inward.

of the nail. This appears to contribute to the curling. The author recommends that this ridge be rasped off to give the nail a little more flexibility.

The toenail of a llama may be avulsed from the digit (Figures 10.31A and B, 10.32). The nailbed must be protected by a light bandage until the nail regrows, usually within two months.

Onychia is inflammation of the corium beneath the nail. Paronychia involves the tissue at the margins of

Figure 10.30. Hoof-trimming tools. (A) Burdizzo sheep nail trimmer, (B) hoof knife, (C) pruning shears, (D) equine hoof nipper.

Figure 10.31A. Avulsed toenail of a llama.

Figure 10.31B. Avulsed toenail of a llama.

the nail. Both conditions may occur in camelids as a result of contusion or laceration of the nail. Neglected toenail trimming may allow infection to migrate dorsally under the nail. Drainage should be established and treatment continued with local disinfectant medications.

The footpads are subject to laceration, contusion, foreign body penetration, erosion, and ulceration. Subfootpad abscessation may result in complete undermining of the pad. To repair this ailment, the detached slipper must be removed and the sensitive underlying tissue protected by bandaging until recornification takes place.

Infectious pododermatitis occurs in llamas (Figures 10.33A and B). In South America, it is felt that *Fusobacterium necrophorum* is the primary etiologic agent, but a variety of anaerobic organisms have been isolated from pad ulcerations in North America. *Bacteroides* spp. are particularly difficult to eradicate because they are resistant to most antibiotics (Chapter 7).

Povidone-iodine, diluted 1:4, is excellent for disinfection underneath a bandage. Another antimicrobial

Figure 10.32. Healed avulsed toenail of a llama.

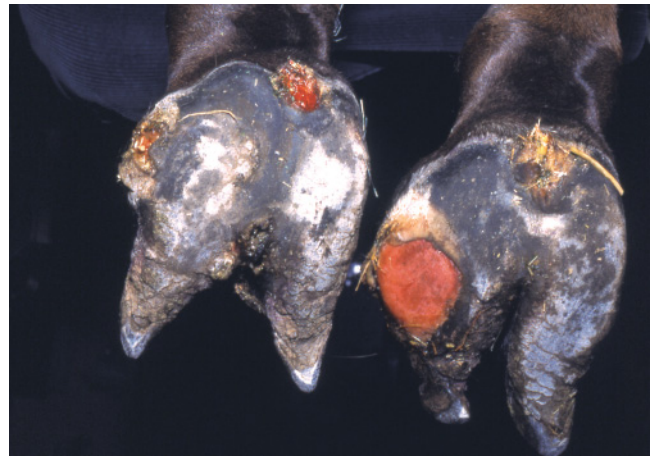

Figure 10.33A. Infectious pododermatitis.

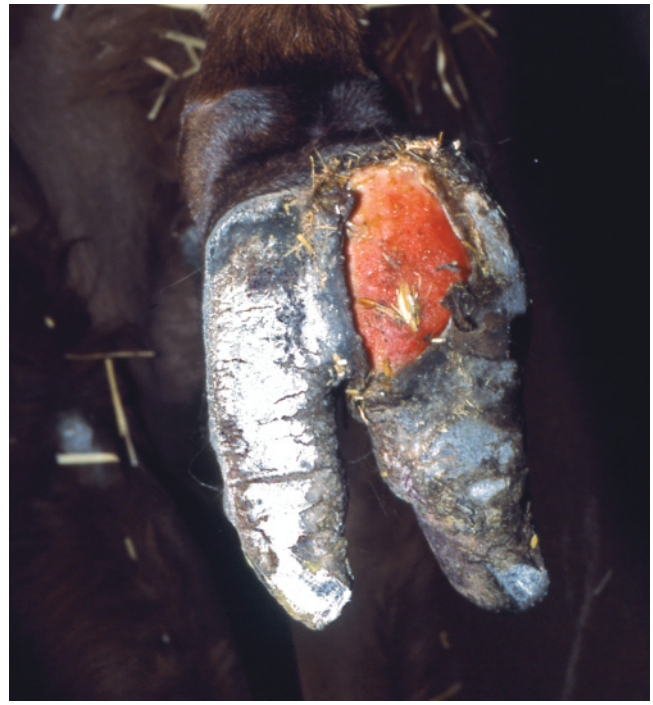

Figure 10.33B. Infectious pododermatitis.

medication, copper naphthenolate (Kopertox, Ayerst Labs, New York), can be sprayed onto the exposed surface. It adheres readily. A light bandage aids in keeping the medication from wearing away.

Mastitis[24]

Mastitis is not common in SACs or camels, but when it does occur, prompt attention is necessary to avoid loss of function in one or more quarters or even death. Camelid mastitis occurs in the same forms as seen in dairy cattle, namely, subclinical, chronic, acute, and peracute.

Predisposing Factors

In camelids, the udder is not pendulous, and the teats are relatively short, so trauma is minimized. Nonetheless, trauma (laceration, contusion, abrasion) of the glands and teats is possible if a female attempts to jump a fence or is mauled by dogs. Trauma to the tip of the teat may weaken the sphincter of the streak canal, which in the normal state tends to prevent access to the gland of pathogenic bacteria. Even though the location of the udder minimizes exposure to filth, when the female is recumbent, she may be forced to lie down in mud if there is no access to dry, clean areas.

Milk is an excellent medium for bacteria, and any condition that allows milk to stagnate within the gland fosters growth of any organism that may have gained temporary entrance.[50] Failure of the cria to nurse, blockage of a streak canal with a waxy plug, pre- and postparturient udder edema, and stricture of the streak canal are factors that may cause stagnation.

Etiology

There are reports on specific bacteria that cause mastitis in camelids.[49] It might be anticipated that all of the organisms causing mastitis in cattle could do likewise in camelids because those organisms have been isolated from other disease conditions in camelids. Isolates from peracute camelid mastitis have included *Escherichia coli, Klebsiella pneumoniae*, and *Aerobacter enterobacterium*, which are the same organisms causing peracute mastitis in cows.

Clinical Signs

Subclinical mastitis may be detected only by culturing for an organism in the milk or testing the milk with one of the mastitis tests such as the California mastitis test (CMT). Indurated areas may be palpable within a

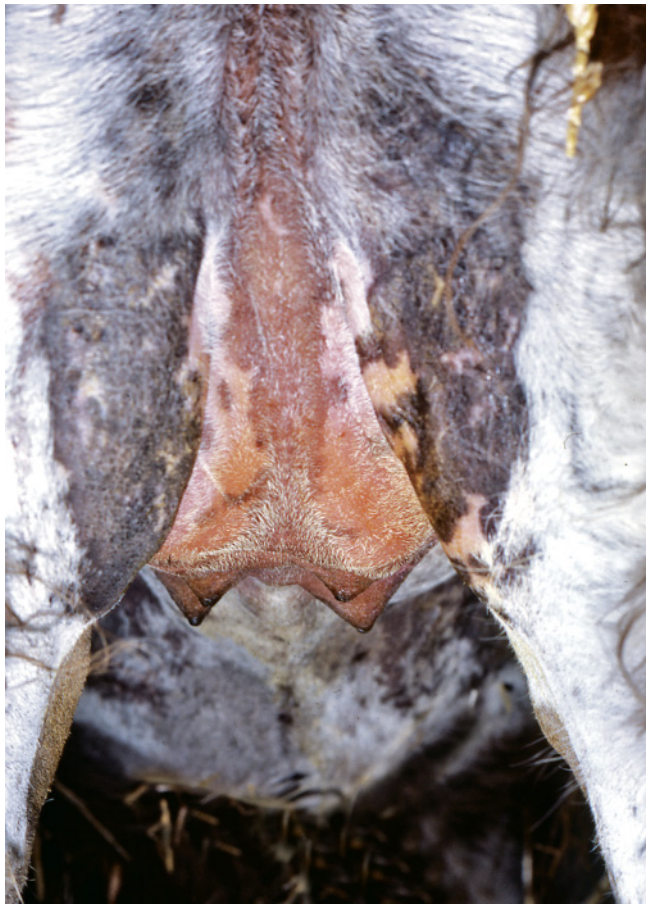

Figure 10.34. Acute mastitis and dermatitis caused by *E. coli* infection.

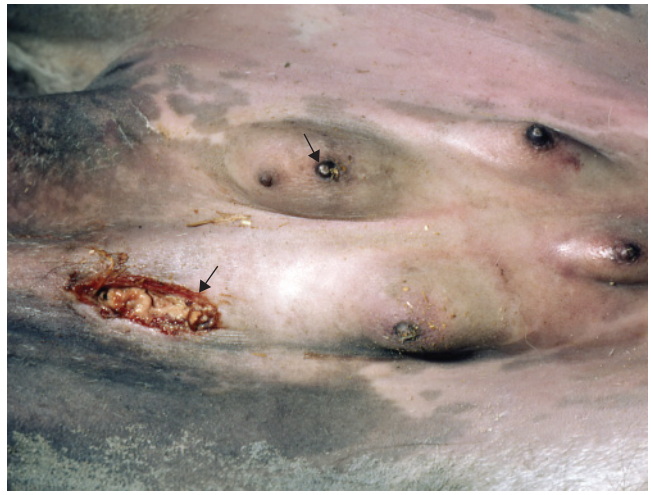

Figure 10.35. Acute mastitis with abscessation.

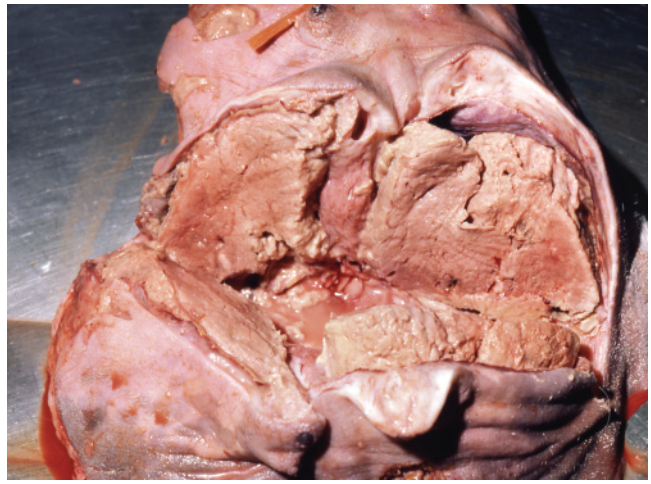

Figure 10.36. Necrotic mastitis caused by *E. coli* infection.

gland, but there are no systemic signs, and the gland is not swollen or hot. Chronic mastitis results in periodic changes in the quality of the milk.

Acute mastitis is usually seen just before or within a few days after parturition and is characterized by heat, swelling, hardness of the affected gland, and evident pain on palpation. The secretion may be watery, hemorrhagic, thickened, stringy, or odorous. The female may refuse to allow the cria to nurse because of the pain, or the cria may refuse to nurse because of the unpalatable secretion. The cria may also develop gastroenteritis or septicemia from ingestion of the pathogen. The female may be anorectic and have a low-grade fever.

Peracute mastitis may have all of the signs of acute mastitis (Figure 10.34) plus severe depression and toxemia, and may even lead to gangrene (Figures 10.35, 10.36), septicemia, and death. Peracute mastitis is usually seen within a few days of parturition.

Diagnosis

The diagnosis is based on a thorough physical examination; evaluation of the secretion for consistency, color, viscosity, presence of debris, and sediment; culture and sensitivity; and evaluation with the CMT. All quarters should be examined, not just the obviously affected gland. Ultrasonography may be used to locate walled-off abscesses.

Pre- and postparturient edema occurs in camelids and must be differentiated from mastitis. Edema is symmetric and uniformly distributed over the gland. The swelling is not hot or painful, and the secretion quality is not altered. Edema may cause swelling of the teats, which may mechanically interfere with the cria's ability to grasp the teat in its mouth. Massage and manual milking may be necessary to reduce the swelling. Colostrum should be saved and offered to the cria via a bottle or by tube feeding. Continued engorgement of the gland may predispose toward mastitis.

Therapy

The objectives of treatment are to save the life of the female, restore the function of the gland, and improve milk quality.[70] These objectives are achieved by removing the cause of the mastitis, providing systemic support, locally infusing the glands with antimicrobial agents, and promoting the healing of the damaged tissue.

The veterinarian must be fully aware of the anatomy of the camelid mammary gland and that a noncommunicating double gland is associated with each teat. If local infusion is selected, both streak canals must be cannulated simultaneously to ensure treatment of the affected portion. The streak canals are tiny, and 1-mm (3.5 French) tomcat catheters are required. The streak canal is easily traumatized. Do not attempt to infuse with bovine teat cannulae or commercial infusion tubes.

Peracute and possible acute mastitis cases require parenteral administration of antibiotics along with local infusion. In both instances, the selection of an antibiotic should be based on culture and sensitivity. The toxemia should be treated aggressively with steroids and intravenous fluids. The exudate should be removed from the gland three to five times daily. Alternating hot and cold packs and gentle massage will facilitate removal of the exudate.

Following is the protocol used for successful treatment of a case of peracute mastitis. The left rear quarter became hot and swollen. Milk production in that quarter ceased and was replaced by a hemorrhagic fluid containing clots of debris. The secretion was CMT positive throughout the treatment period. A culture and sensitivity was conducted, yielding *E. coli* and *Klebsiella* spp., with sensitivities to gentamicin, trimethoprim sulfas, and third-generation cephalosporins.

Gentamicin (1 mg/kg) was administered intramuscularly three times daily for ten days. The unaffected quarters were stripped every four hours. The affected quarter was infused with a solution of polymyxin B (250,000 units diluted in 40 ml sterile saline) at 8 a.m. and 8 p.m.; 20 ml were infused simultaneously, using tomcat catheters, into each streak canal. The medication was left in the quarter for four to six hours, following which the affected quarter was stripped every two hours day and night. Infusion therapy was continued for ten days.

At completion of the treatment, gland secretion had returned to milk of normal appearances, with a weak CMT positive reaction. Heat and swelling had disappeared.

In acute and peracute cases, it is imperative that the cria be temporarily orphaned as a protection to itself and to minimize painful manipulation of the affected gland and teat.

Prevention

Careful attention to the nursing behavior of the cria and observation and palpation of the udder allow early detection of problems. A weak or premature cria will not remove sufficient milk to prevent stagnation. The female should be milked regularly until the cria is strong enough to nurse unassisted. The cria generally removes milk from all four teats at each nursing episode. A single swollen teat should be investigated.

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11

Musculoskeletal System

ANATOMY

No text describes the anatomy of the musculoskeletal system of SACs. There are several references in the periodic literature.[5,7,10–13], However, an excellent modern anatomy text of the dromedary camel is available.[27] Obviously, there are many differences, but much of the information is applicable to SACs. Only those aspects of anatomy that are unique to camelids and clinically important are discussed here. Figure 11.1 is a diagrammatic representation of the skeletal system of an SAC. The anatomy of certain sections of the musculoskeletal system is described in other chapters, such as Chapter 6, Surgery. Anatomy of the head is described in the discussions of disorders of the teeth (Chapter 13) and congenital disorders of the head (Chapter 22). The anatomy of the limbs is discussed in Chapter 6 in the section dealing with orthopedic surgery of the long bones.

RADIOGRAPHY[1,23]

Vertebrae

Cervical

Camelids have a highly mobile neck, and in SACs, it is covered by thick skin (up to 1 cm). The seven cervical vertebrae are elongated, and from C-3 caudad, a ventral projection of the cranial segment of the transverse process forms an inverted U-shaped osseous channel on the ventrum of the neck (Figures 11.2, 11.3). The channel protects the vital structures of the neck from accidental laceration during inter-male aggressive bouts. Figures 11.4 to 11.8 are of cervical vertebrae.

More pronounced ventral projections (2.5 cm) are seen on C-4 and C-5. There are ventral projections on both the cranial and caudal segments of the transverse processes of C-6. This vertebra is easily recognized on a radiograph. Because C-7 lies deep within the neck, it is difficult to visualize radiographically. It has no ventral projections and is the shortest of the cervical vertebrae. The approximate length of each cervical vertebra is as follows: C-1, 5.5 cm; C-2, 11 cm; C-3, 10 cm; C-4, 9 cm; C-5, 9 cm; C-6, 8 cm; and C-7, 5 cm.

Thoracic

There are twelve thoracic vertebrae. The dorsal spinous processes are easily identified, but the bodies, articular facets, and spinal canal are more difficult to visualize because of the heavy vertebral muscle mass.

Lumbar

There are seven lumbar vertebrae (Figure 11.8). The transverse process of L-7 is shortened. The dorsal spinous processes are vertical.

Sacrum

There are five sacral vertebrae. The dorsal spinous process of S-1 is minimal. This clear demarcation of the lumbosacral space marks the preferred site for placement of the spinal needle (Chapter 4).

Coccygeal

The number of coccygeal vertebrae varies from ten to fifteen in the camelid. No references were found describing the coccygeal vertebrae, but observations on skeletal preparations indicate that the first three or four coccygeal vertebrae possess neural arches, providing a neural canal. Likewise, those same vertebrae have transverse processes. The shape changes from an irregular square, to an upside down pear, to a linear rectangle. The first

Medicine and Surgery of Camelids, Third Edition by Murray E. Fowler

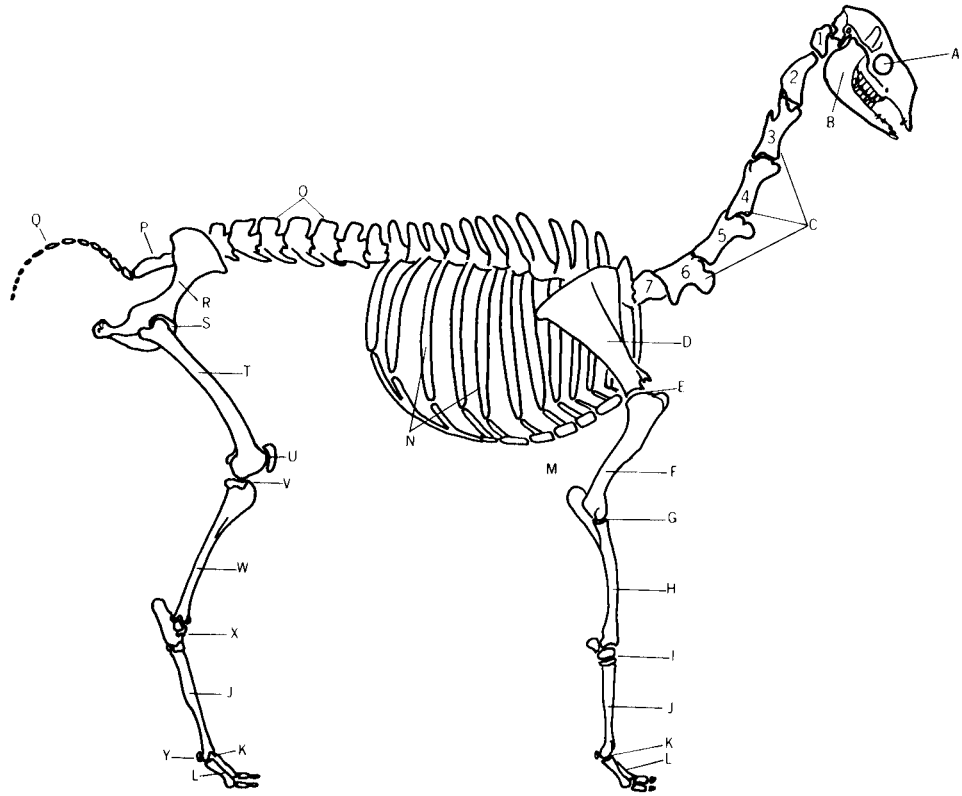

Figure 11.1. Diagram of a llama skeleton. (A) Orbit, (B) mandible, (C) cervical vertebrae, (D) scapula, (E) shoulder, (F) humerus, (G) elbow, (H) radius, (I) carpus, (J) metacarpus, (K) fetlock, (L) pastern, (M) sternebrae, (N) ribs, (O) vertebrae, (P) sacrum, (Q) coccygeal vertebrae, (R) ilium, (S) hip, (T) femur, (U) patella, (V) stifle, (W) tibia, (X) hock, (Y) sesamoids.

Figure 11.2. Llama cervical vertebrae (C-1 to C-6), dorsal view.

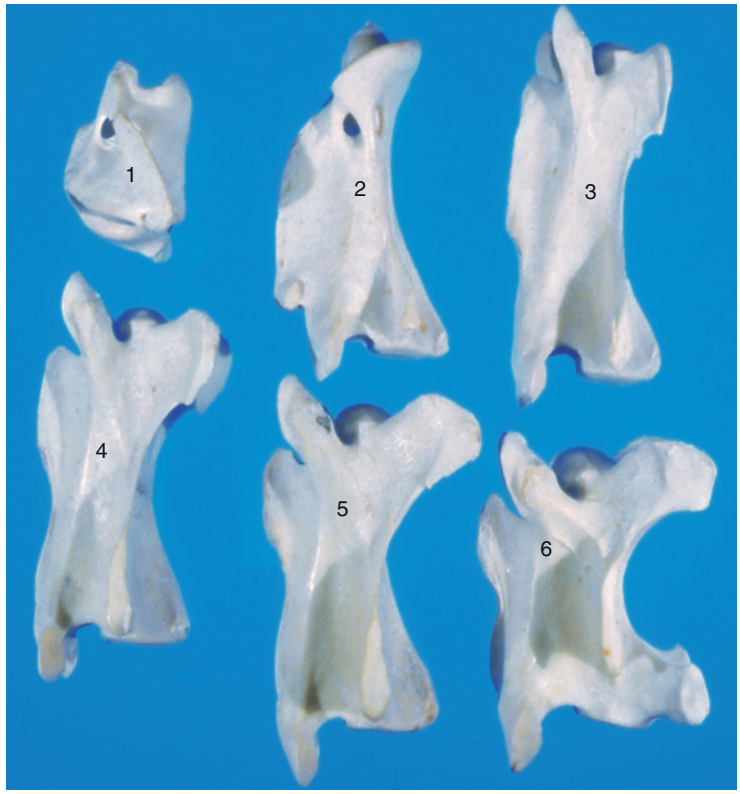

Figure 11.3. Llama cervical vertebrae (C-1 to C-6), right lateral view.

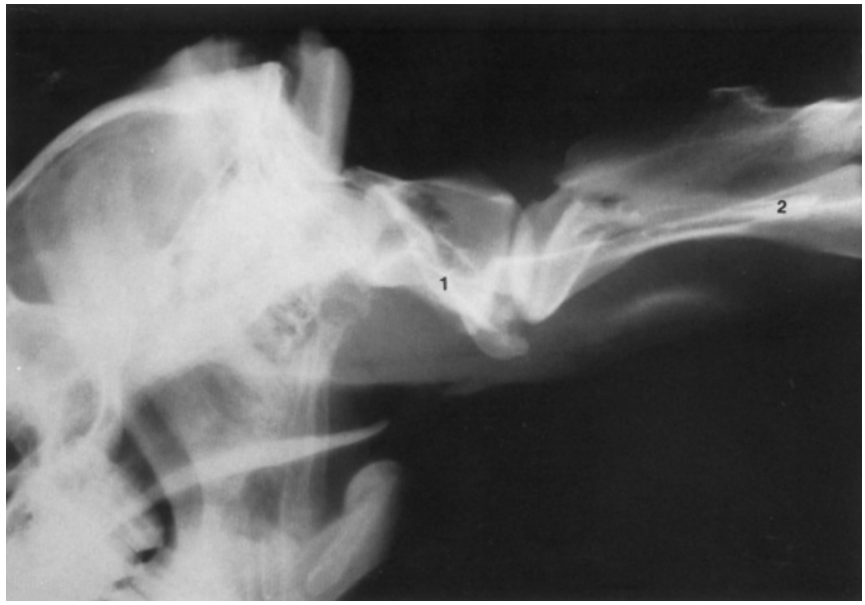

Figure 11.4. Lateral radiograph of caudal skull, C-1, and C-2 (fractured).

coccygeal vertebra may fuse to the sacrum, particularly in older subjects.

Other Bones of the Trunk

There are twelve pairs of ribs. The camelid pelvis is not unique. The sternebrae are flattened from side to side, as is characteristic of many artiodactylids. The skin over the sternum of an adult is highly calloused, giving an erroneous impression of soft-tissue pathology on a radiograph (see Figure 11.52, later in the chapter). The sternum of camels has a ventral osseous extension called the pedestal (see Figure 11.53, later in the chapter).

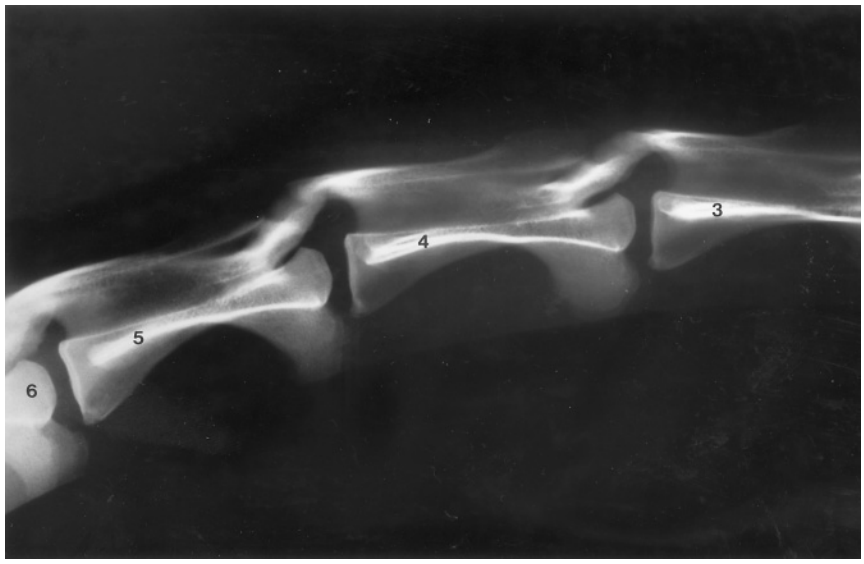

Figure 11.5. Lateral radiograph of cervical vertebrae, C-3 to C-6.

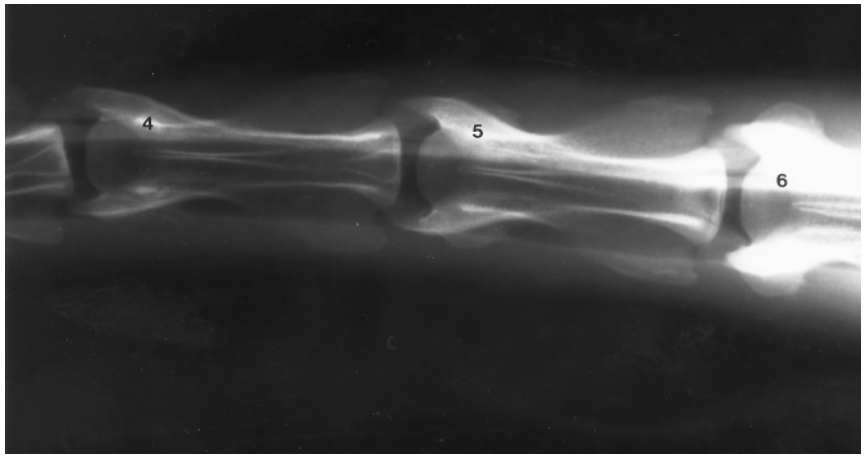

Figure 11.6. Dorsoventral radiograph of cervical vertebrae, C-4 to C-6.

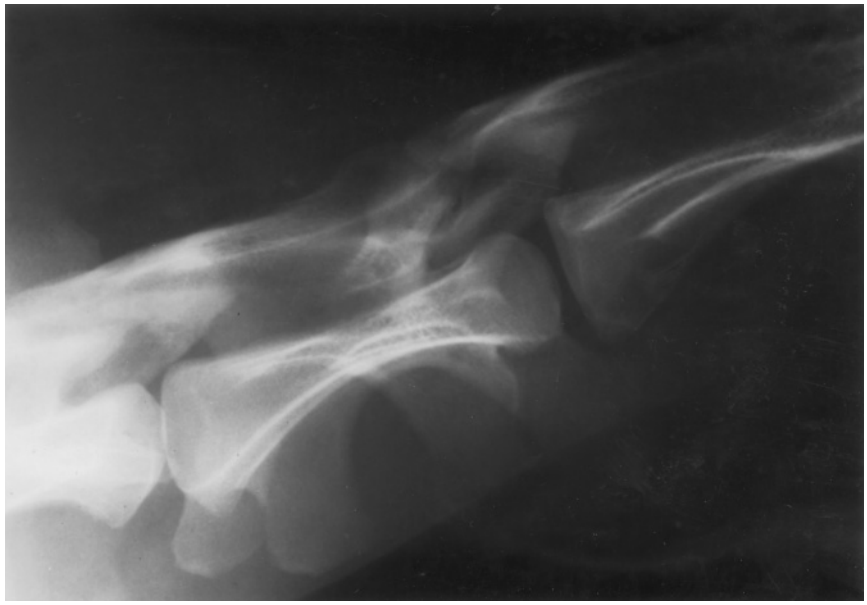

Figure 11.7. Lateral radiograph of C-6, illustrating pronounced ventral projections of the transverse processes.

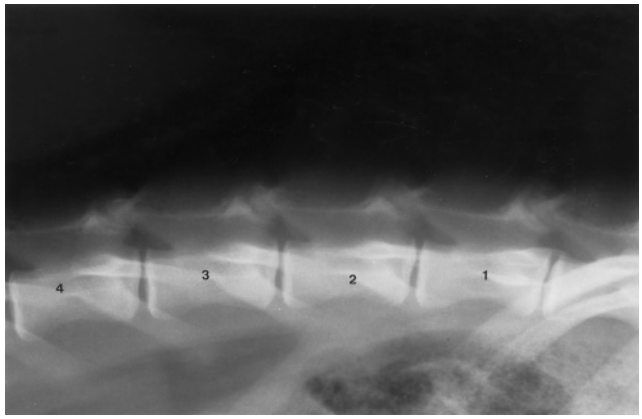

Figure 11.8. Lateral radiograph of lumbar vertebrae.

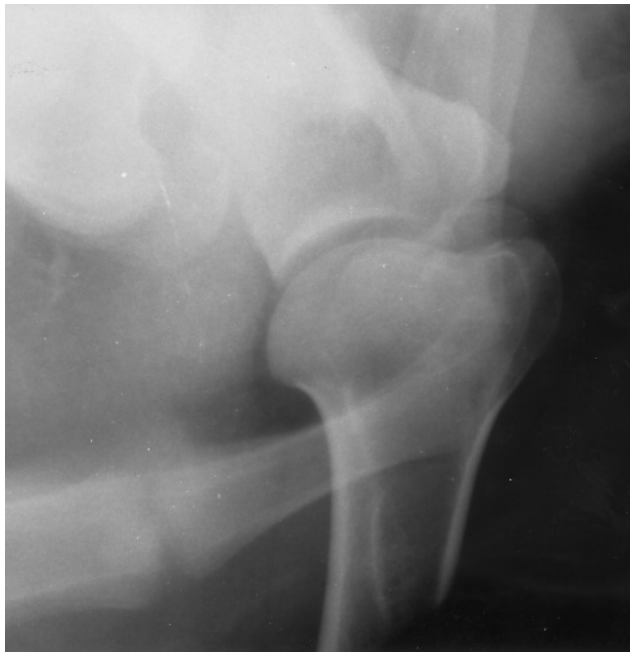

Figure 11.9. Lateral radiograph of a llama shoulder.

Limb Articulations[24,25,30]

The radiographic appearance of the articulations of the forelimb is follows: shoulder (Figures 11.9 to 11.11); elbow (Figures 11.12 to 11.15); carpus (Figures 11.16 to 11.18); metacarpal phalangeal, and phalangeal joints (Figures 11.19 to 11.22). Note that two sesamoid bones are associated with each first phalanx (P-1). For more details on the carpus, see Chapter 22.

The coxofemoral articulation of the adult is illustrated in Figures 11.23 and 11.24. The animal should be anesthetized for dorsoventral radiography. The femorotibial articulation is seen in Figures 11.25 to 11.30. The relationship of the tarsal bones to the tibia and metatarsus is unique (Figures 11.31 to 11.34). The lower limb articulations are similar to those of the

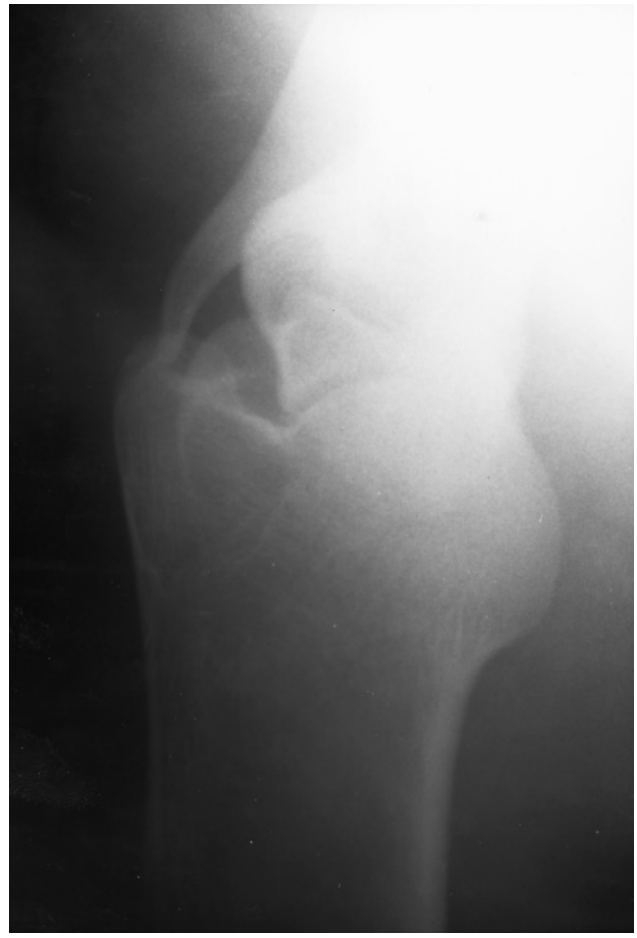

Figure 11.10. Craniocaudal radiograph of a llama shoulder.

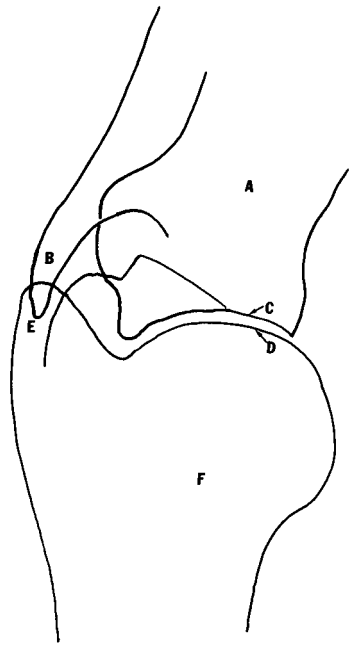

Figure 11.11. Diagram of craniocaudal radiograph of a llama shoulder. (A) Scapula, (B) acromion process, (C) glenoid cavity, (D) head of humerus, (E) major tubercle of humerus, (F) humerus.

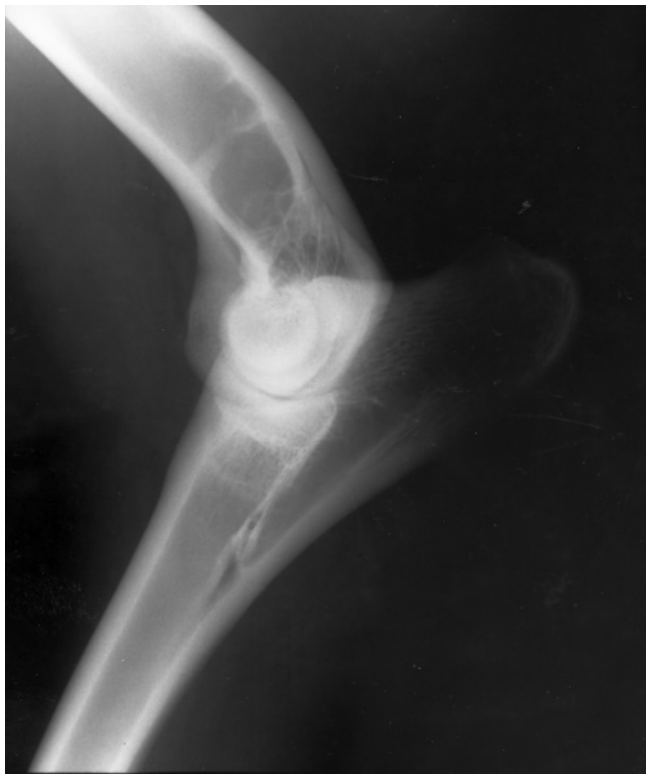

Figure 11.12. Lateral radiograph of a llama elbow.

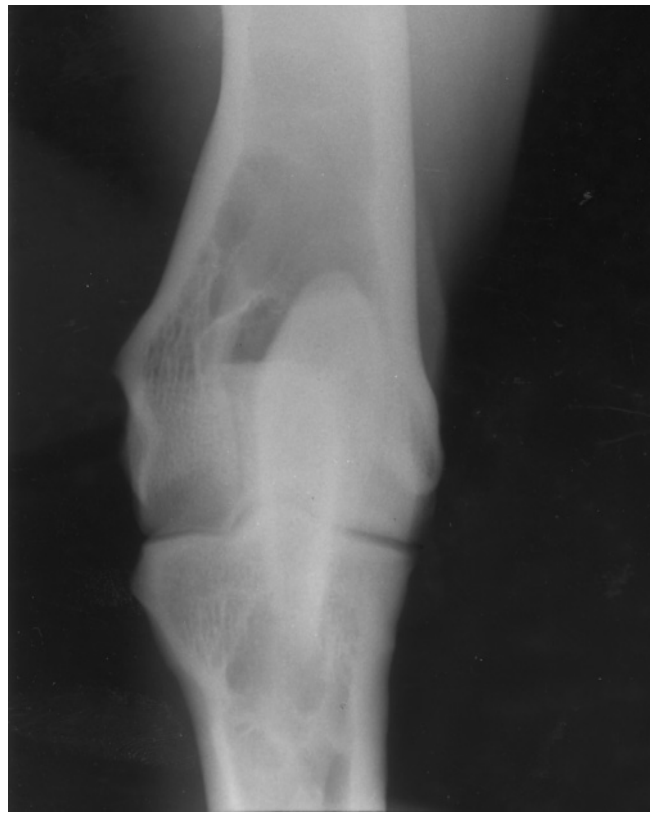

Figure 11.14. Craniocaudal radiograph of a llama elbow.

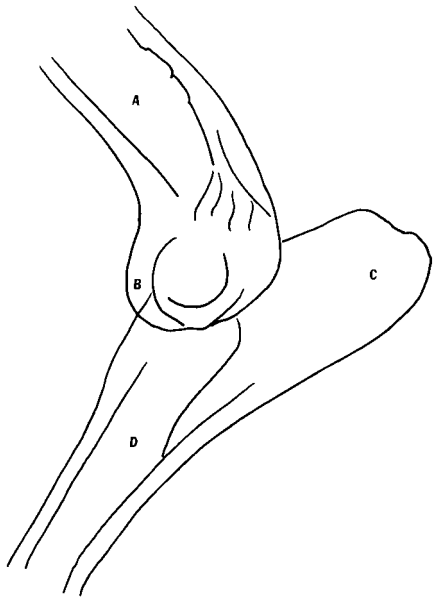

Figure 11.13. Diagram of lateral radiograph of the elbow of a mature llama. (A) Humerus, (B) trochlea of the humerus, (C) olecranon, (D) radius.

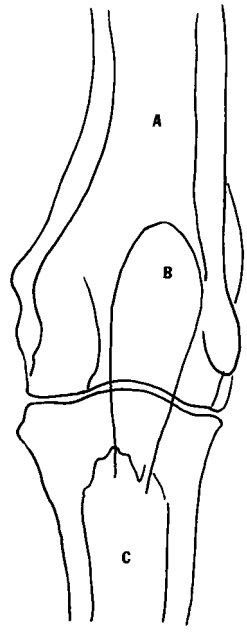

Figure 11.15. Diagram of craniocaudal radiograph of the elbow of a mature llama. (A) Radius, (B) olecranon, (C) radius.

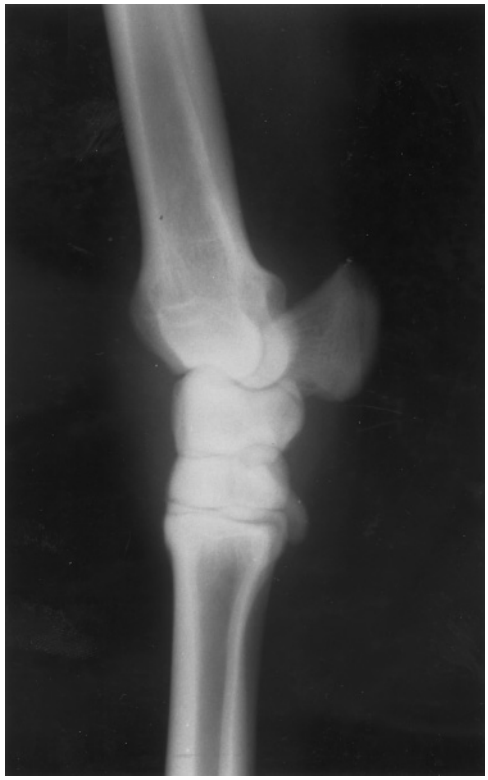

Figure 11.16. Lateral radiograph of a llama carpus.

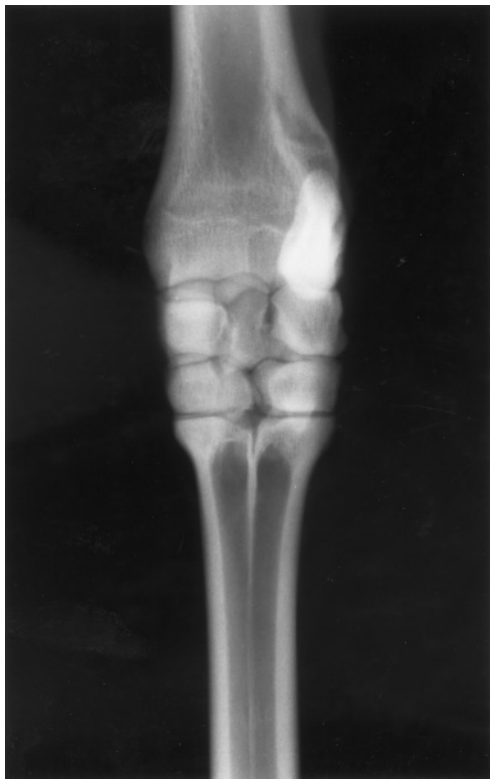

Figure 11.17. Dorsopalmar radiograph of a llama carpus.

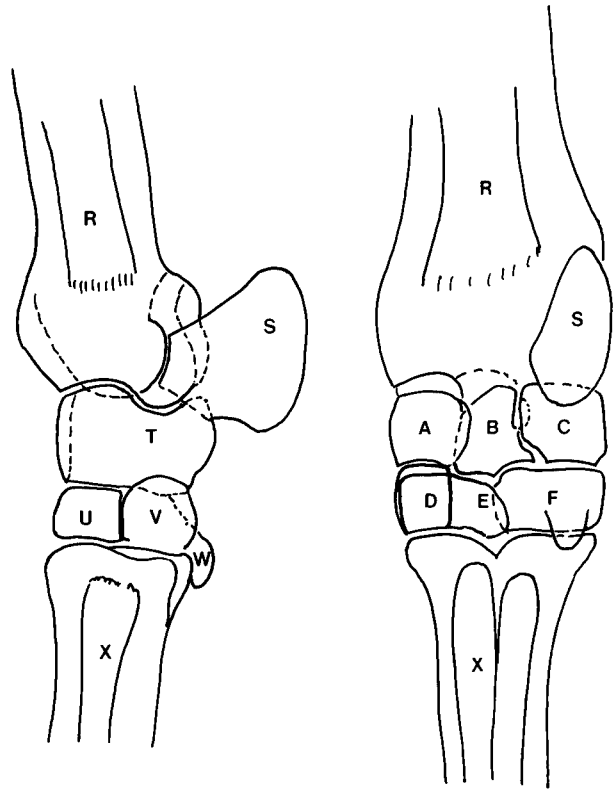

Figure 11.18. Diagram of a mature llama carpus. (R) Radius; (S) accessory carpal bone; (T) radial, intermediate, and ulnar carpal bones superimposed; (U) third carpal bone; (V) second carpal bone; (W) projection of the fourth carpal bone; (X) metacarpus; (A) radial carpal bone; (B) intermediate carpal bone; (C) ulnar carpal bone; (D) second carpal bone; (E) third carpal bone; (F) fourth carpal bone.

forelimb. Figures 11.36 to 11.51 illustrate growth centers on neonate llamas.

The synovial fluid obtained from normal healthy SACs was similar to that of other large domestic animals except that total protein levels were higher in SACs (2 to 3.8 g/dl compared to 1.5 mg/dl in livestock and horses).[30]

Radiographic Technique

Table 11.1 lists techniques used to obtain the foregoing radiographs. The film was 3-M, XDL general-purpose radiographic film in Kodak Lamex regular screen cassettes. The focal film distance was 100 cm (40 in.) and the exposure 1/10 second. Newer digital radiography techniques use different technology. See Chapter 4.

The Sternum

The skin over the camelid sternum develops a pronounced callosity as a response to the animal spending considerable time in sternal recumbency (Figure 11.52). In the SACs there is no modification of the underlying

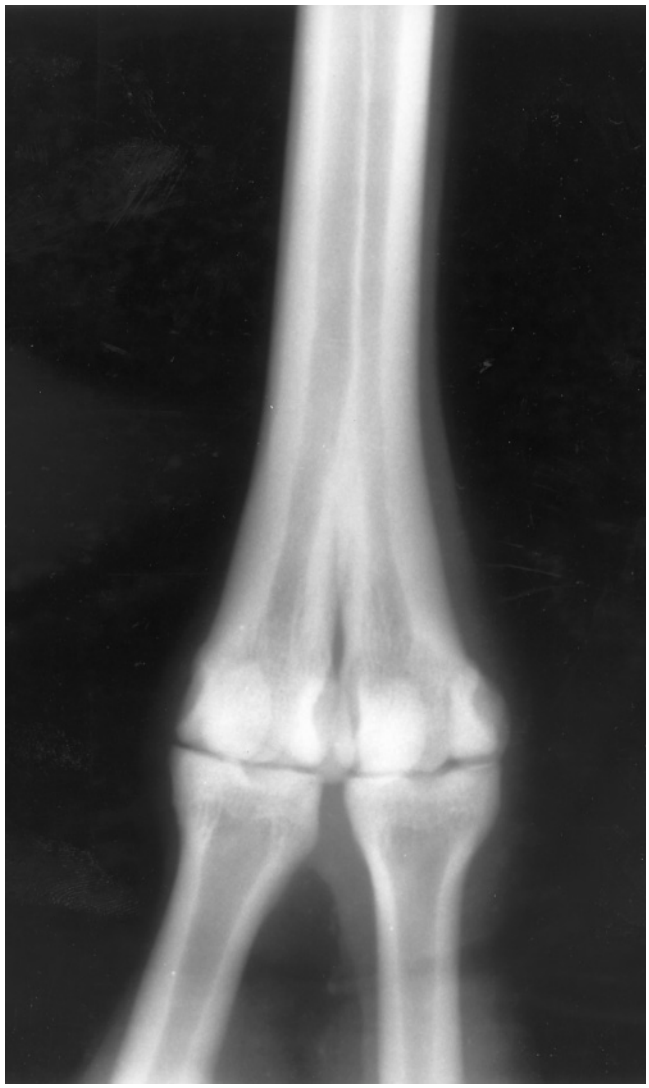

Figure 11.19. Dorsopalmar radiograph of a llama fetlock.

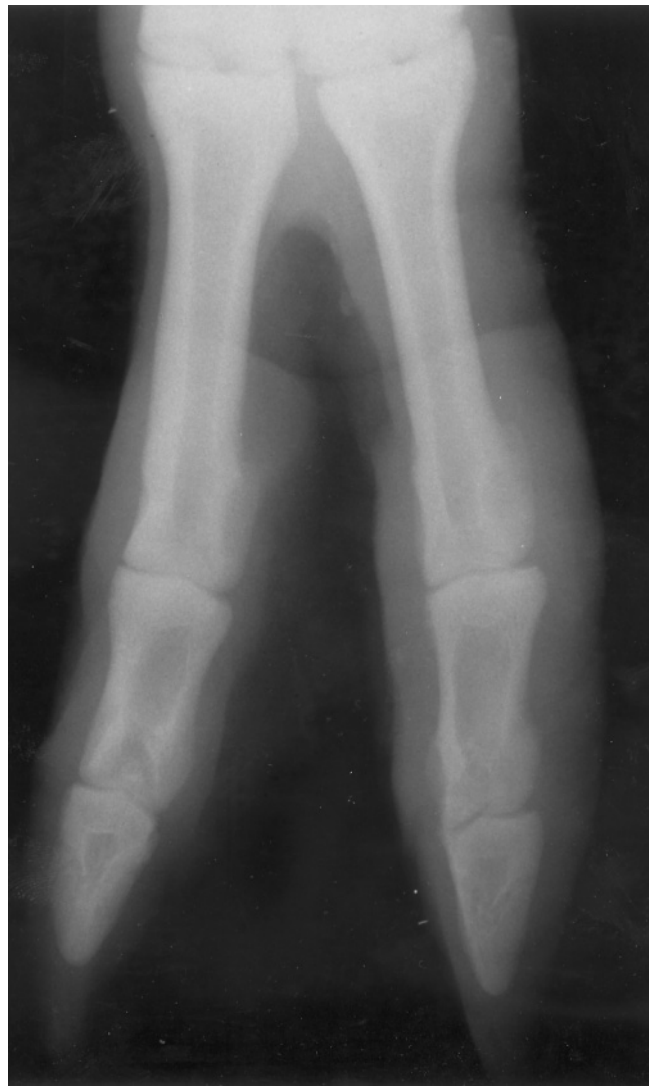

Figure 11.20. Dorsopalmar radiograph of a llama pastern and foot.

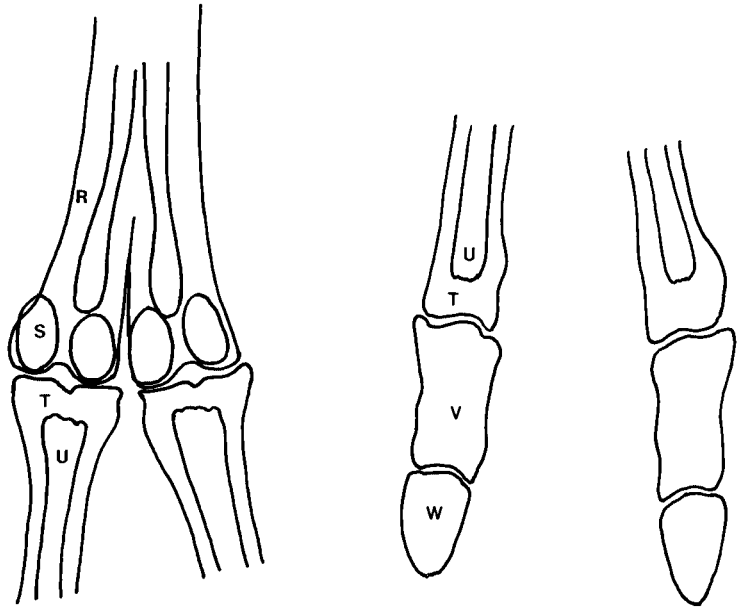

Figure 11.21. Diagram of the fetlock and phalanges. (R) Metatarsus, (S) sesamoid bone, (T) P-1, (U) marrow cavity of P-1, (V) P-2, (W) P-3.

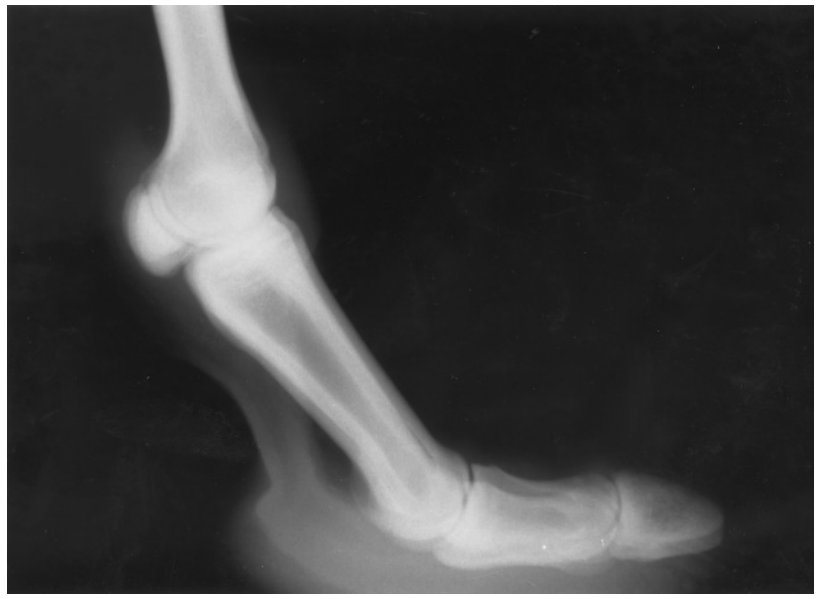

Figure 11.22. Lateral radiograph of a llama fetlock and phalanges.

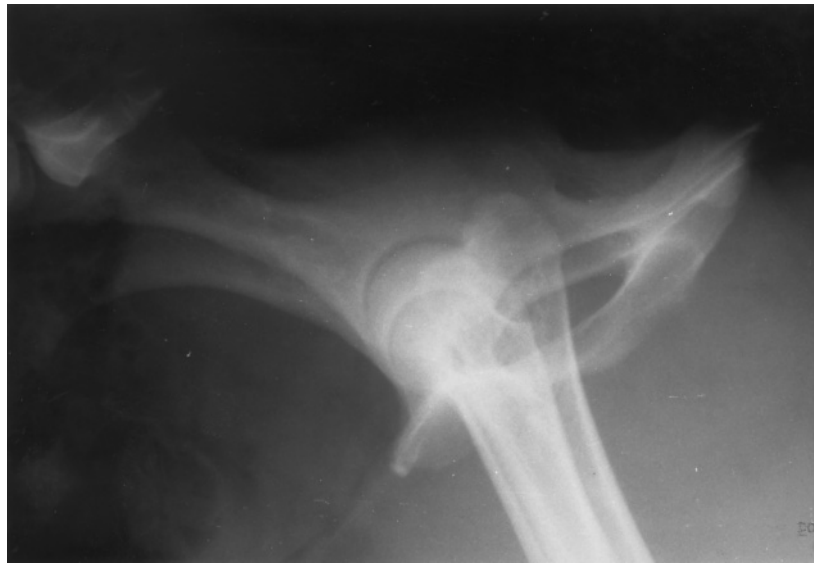

Figure 11.23. Lateral radiograph of a llama pelvis and hip.

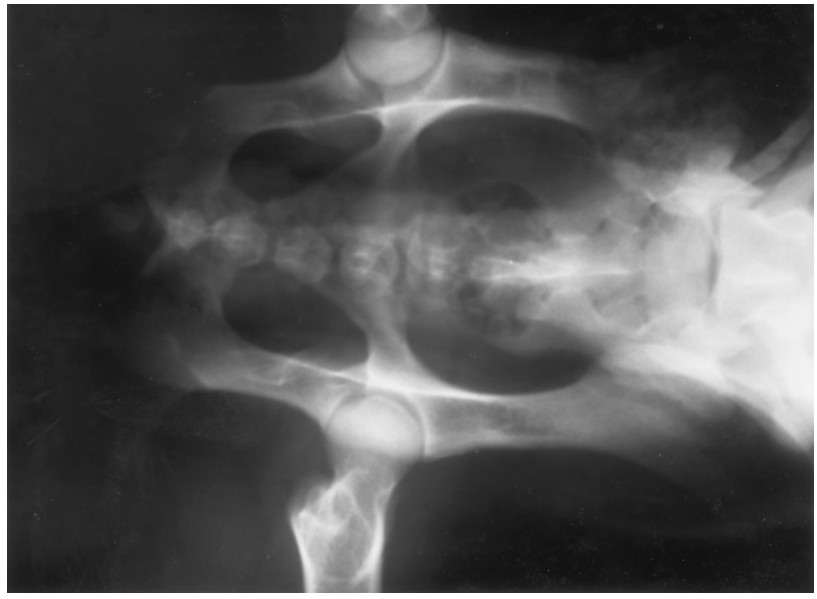

Figure 11.24. Dorsoventral radiograph of a llama pelvis and hips.

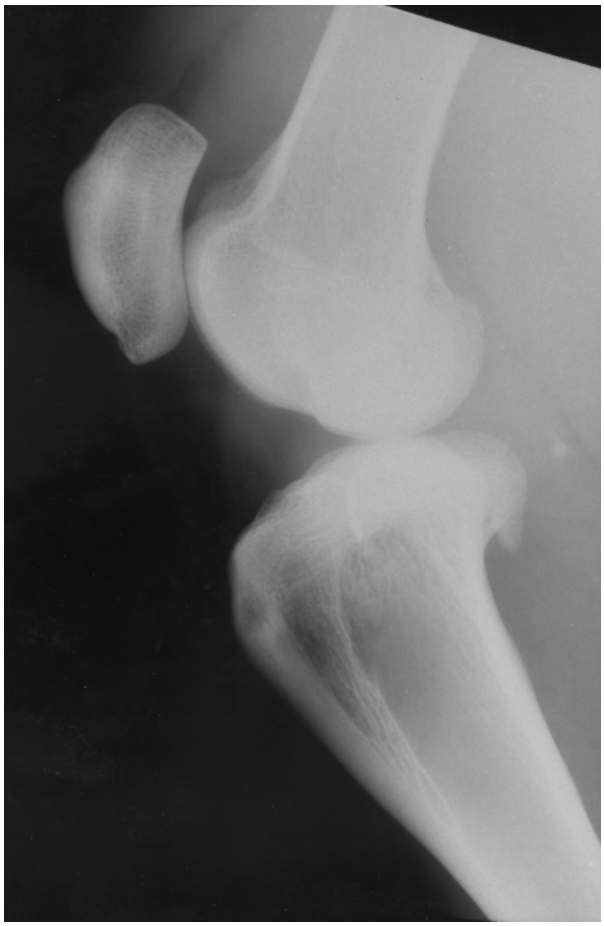

Figure 11.25. Lateral radiograph of a llama stifle (patella in normal position).

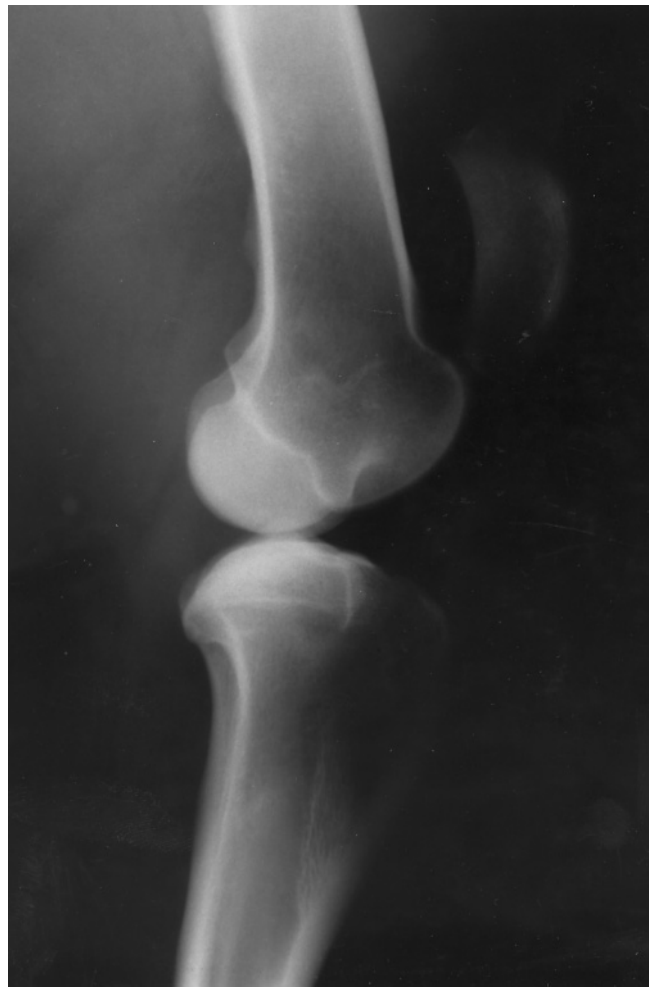

Figure 11.27. Lateral radiograph of a llama stifle (patella riding too high on trochlea).

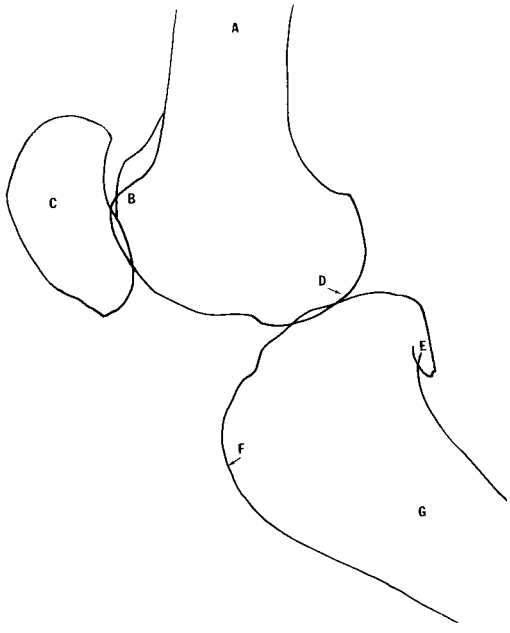

Figure 11.26. Diagram of Figure 11.25. (A) Femur, (B) femoral trochlea, (C) patella, (D) femoral condyle, (E) fibula, (F) tibial crest, (G) tibia.

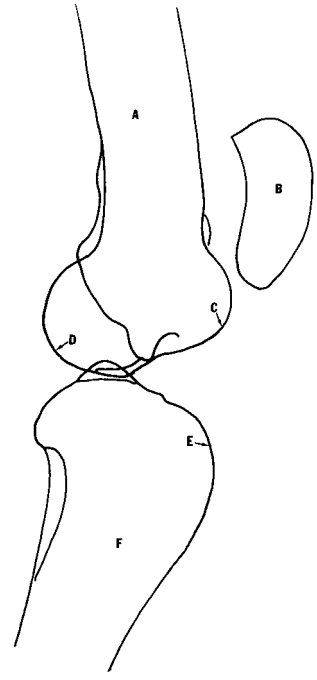

Figure 11.28. Diagram of Figure 11.27. (A) Femur, (B) patella, (C) femoral trochlea, (D) femoral condyle, (E) tibial crest, (F) tibia.

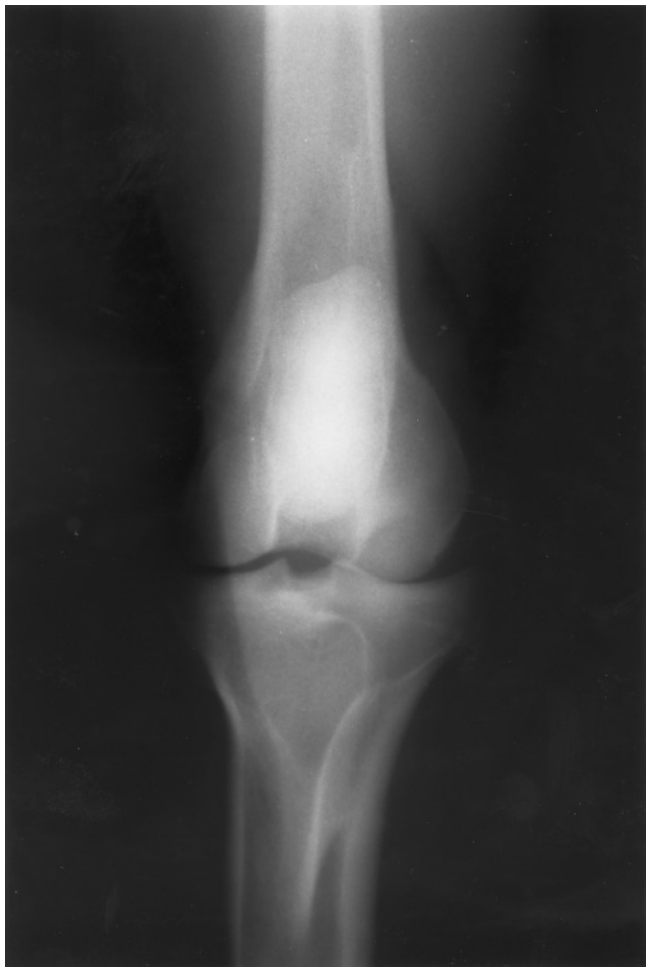

Figure 11.29. Craniocaudal radiograph of a llama stifle.

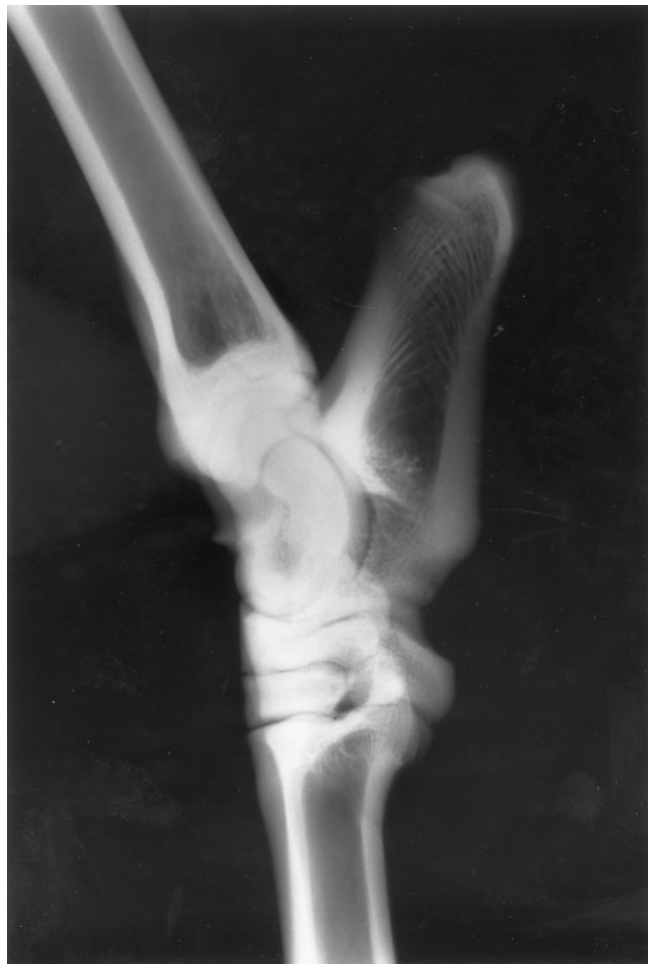

Figure 11.31. Lateral radiograph of a mature llama tarsus.

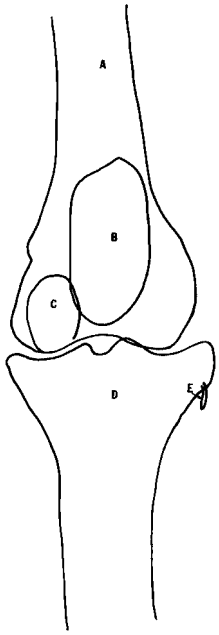

Figure 11.30. Diagram of Figure 11.29. (A) Femur, (B) patella, (C) medial condyle of femur, (D) tibia, (E) fibula.

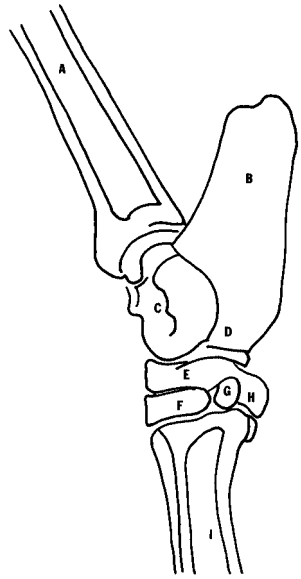

Figure 11.32. Diagram of Figure 11.31. (A) Tibia, (B) tuber calcis, (C) tibial tarsal bone (talus), (D) fibular tarsal bone (calcaneus), (E) central tarsal bone, (F) second and third tarsal bones, (G) first tarsal bone, (H) fourth tarsal bone, (I) medullary space.

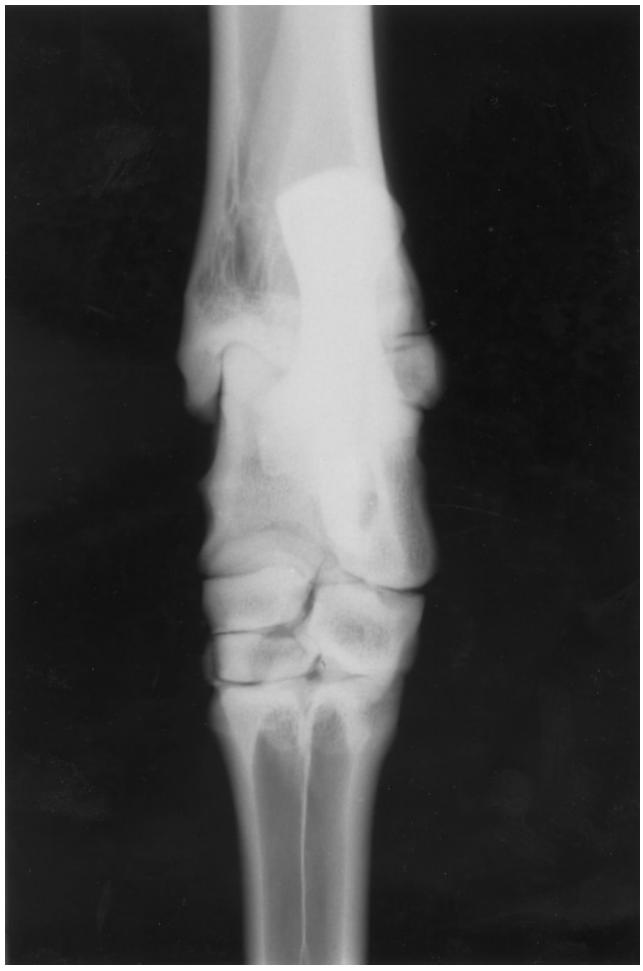

Figure 11.33. Dorsoplantar radiograph of a mature llama tarsus.

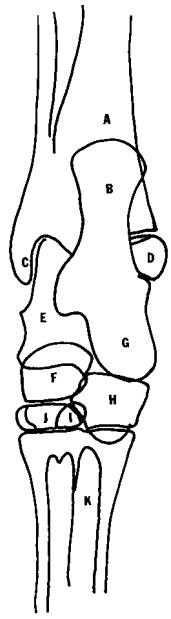

Figure 11.34. Diagram of Figure 11.33. (A) Tibia, (B) tuber calcis, (C) lateral malleolus (distal tip of fibula), (D) medial malleolus, (E) tibial tarsal bone (talus), (F) central tarsal bone, (G) fibular tarsal bone (calcaneus), (H) fourth tarsal bone, (I) first tarsal bone, (J) second and third tarsal bones, (K) medullary space.

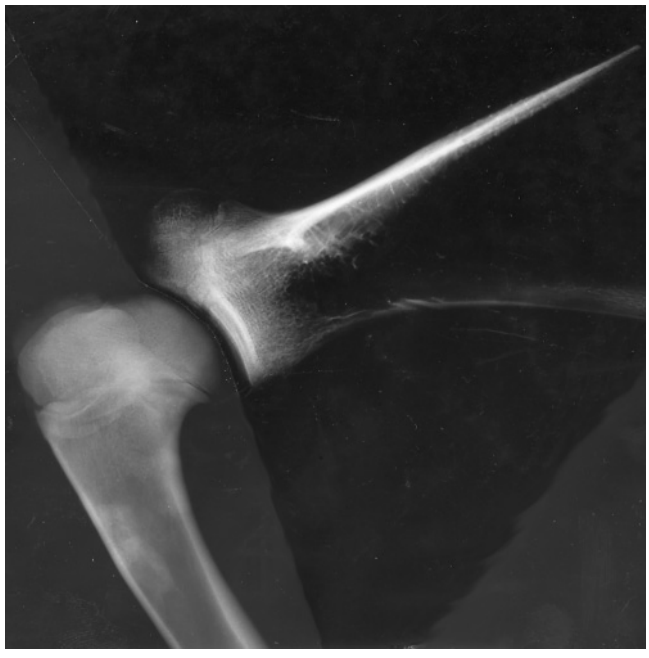

Figure 11.35. Lateral radiograph of a neonate llama shoulder.

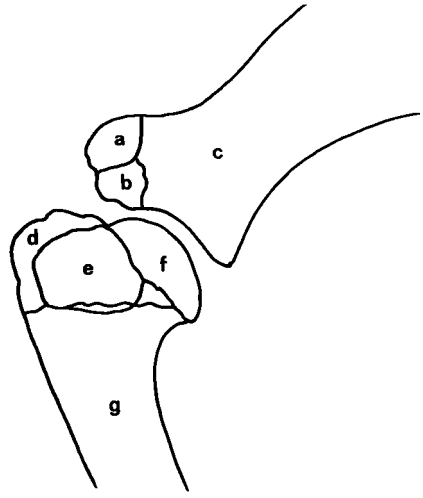

Figure 11.36. Diagram of lateral view of a neonate llama scapulohumeral articulation. (A) Epiphysis of the supraglenoid tubercle, (B) epiphysis of the cranial glenoid cavity, (C) neck of the scapula, (D) lateral tuberosity of the humerus, (E) medial tuberosity of the humerus, (F) proximal epiphysis of the humerus, (G) diaphysis of the humerus.

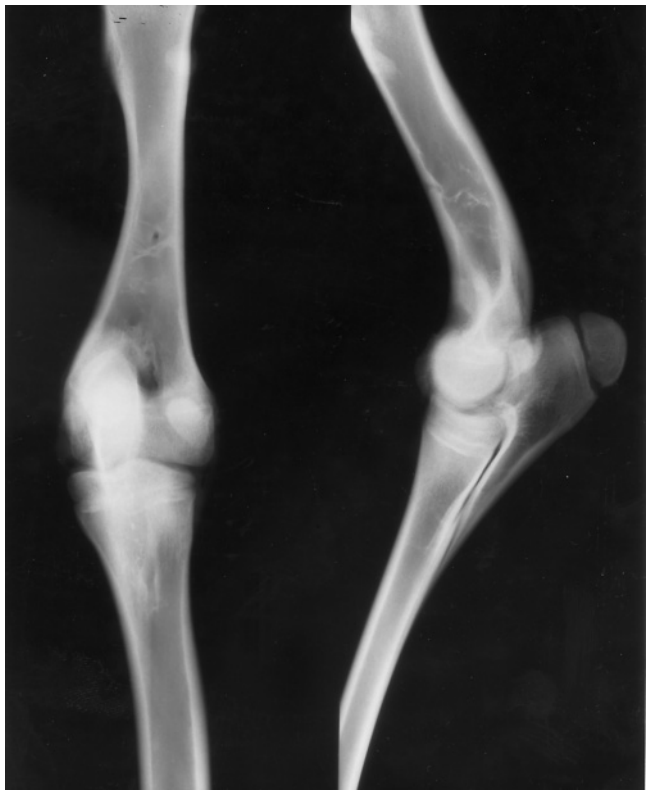

Figure 11.37. Lateral and craniocaudal radiographs of a neonate llama elbow.

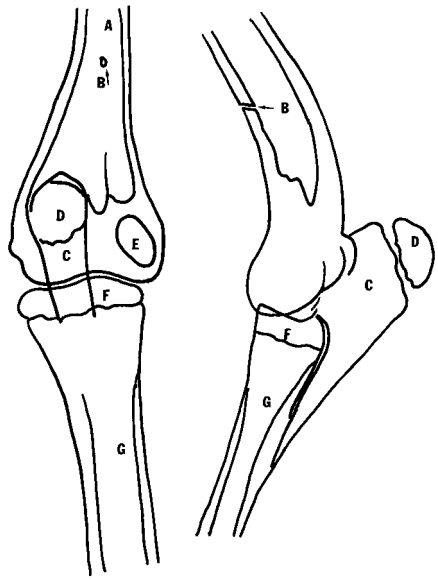

Figure 11.38. Diagram of Figure 11.37. (A) Humerus, (B) nutrient foramen, (C) olecranon, (D) olecranon epiphysis, (E) humeral trochlea, (F) proximal radial, (G) radius.

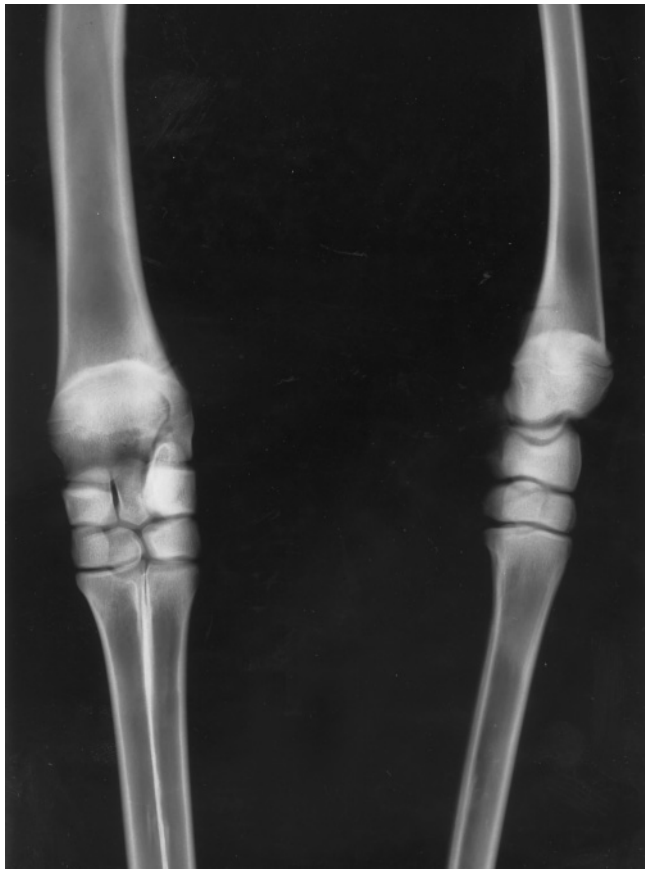

Figure 11.39 Lateral and dorsopalmar radiographs of a neonate llama carpus.

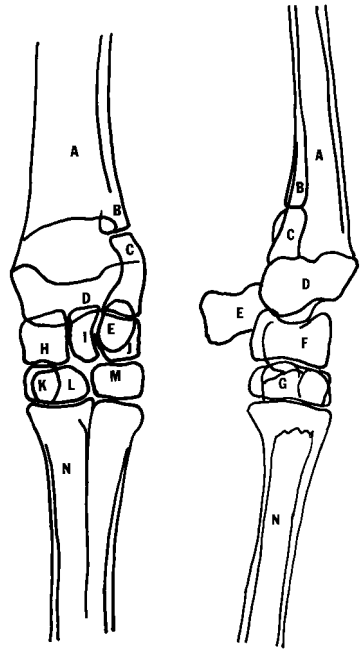

Figure 11.40. Diagram of Figure 11.39. (A) Radius; (B) ulna; (C) ulnar epiphysis; (D) radial epiphysis; (E) accessory carpal bone; (F) combined ulnar, intermediate, and radial carpal bones; (G) combined second, third, and fourth carpal bones; (H) radial carpal bone; (I) intermediate carpal bone; (J) ulnar carpal bone; (K) second carpal bone; (L) third carpal bone; (M) fourth carpal bone; (N) metatarsus.

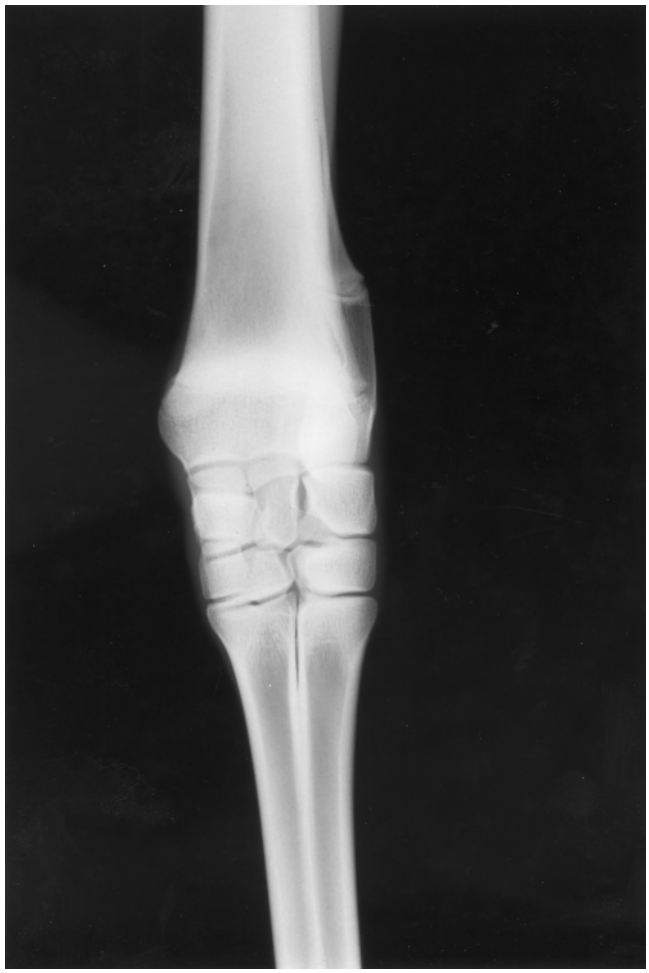

Figure 11.41. Dorsopalmar radiograph of a six-week-old llama carpus.

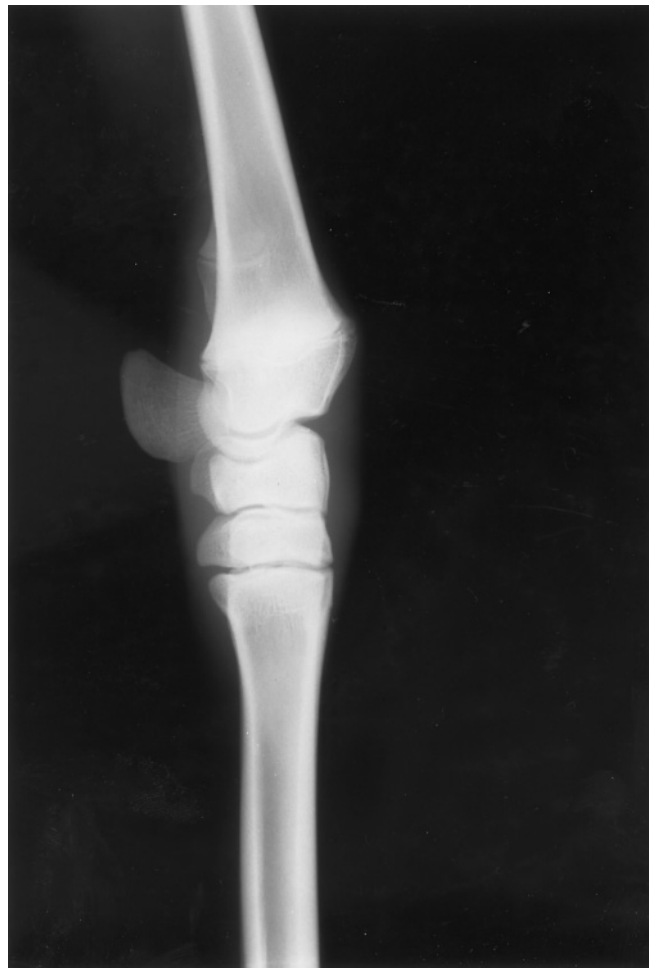

Figure 11.42. Lateral radiograph of a six-week-old llama carpus.

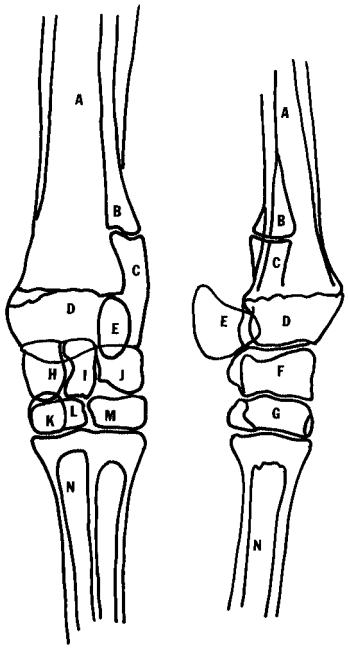

Figure 11.43. Diagram of Figures 11.41 and 11.42. (A) Radius; (B) ulna; (C) ulnar epiphysis; (D) radial epiphysis; (E) accessory carpal bone; (F) combined ulnar, intern, and radial carpal bones; (G) combined second, third, and fourth carpal bones; (H) radial carpal bone; (I) intermediate carpal bone; (J) ulnar carpal bone; (K) second carpal bone; (L) third carpal bone; (M) fourth carpal bone; (N) metatarsus.

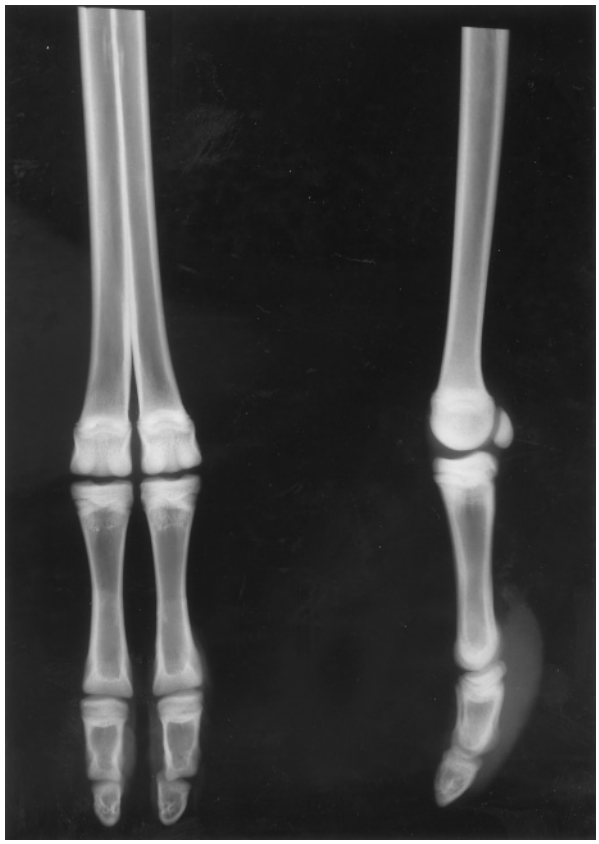

Figure 11.44. Lateral and dorsopalmar radiographs of a neonate llama fetlock and foot.

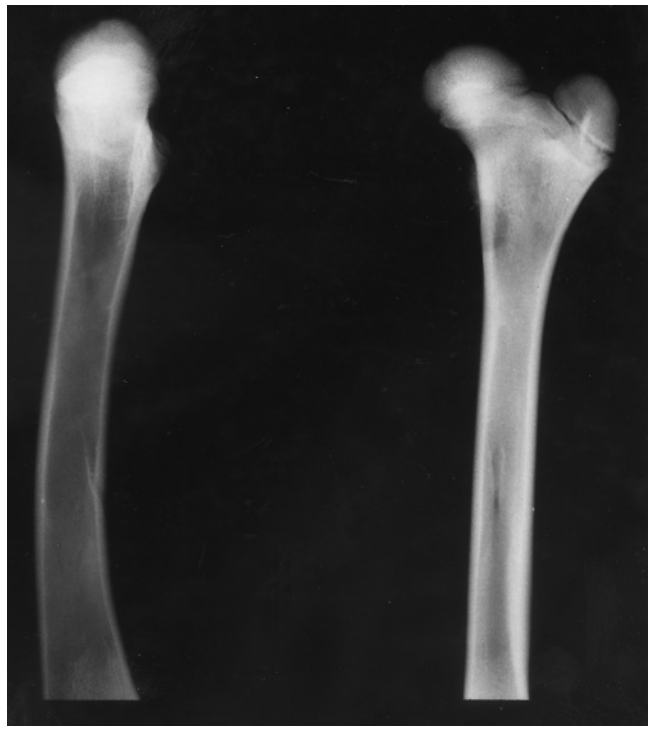

Figure 11.46. Craniocaudal and lateral radiographs of a neonate llama proximal femur.

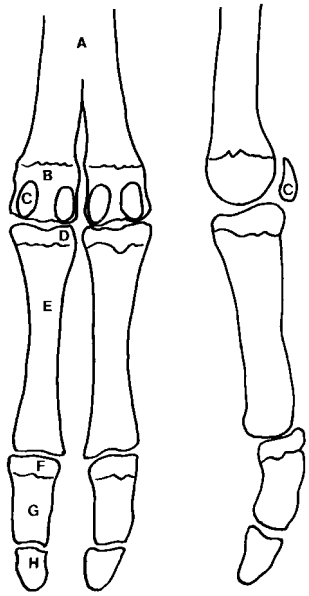

Figure 11.45. Diagram of Figure 11.44. (A) Diaphysis of metacarpus, (B) distal epiphysis of metacarpus, (C) sesamoid bone, (D) proximal epiphysis of P-1, (E) diaphysis of P-1, (F) proximal epiphysis of P-2, (G) P-2, (H) P-3.

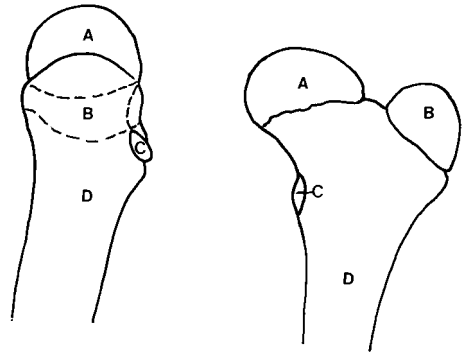

Figure 11.47. Diagram of Figure 11.46. (A) Proximal epiphysis of femur, (B) trochanter major, (C) trochanter minor, (D) diaphysis of femur.

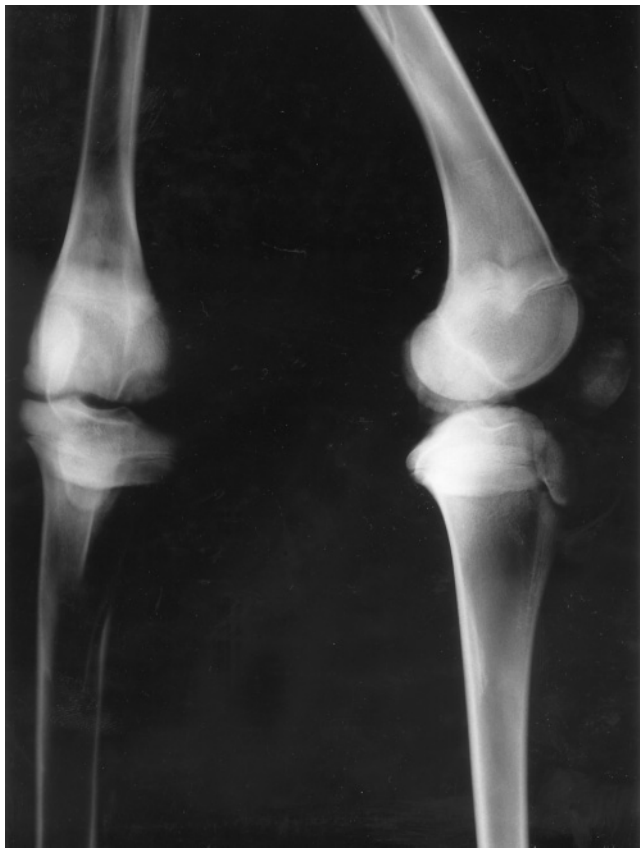

Figure 11.48. Craniocaudal and lateral radiographs of a neonate llama stifle.

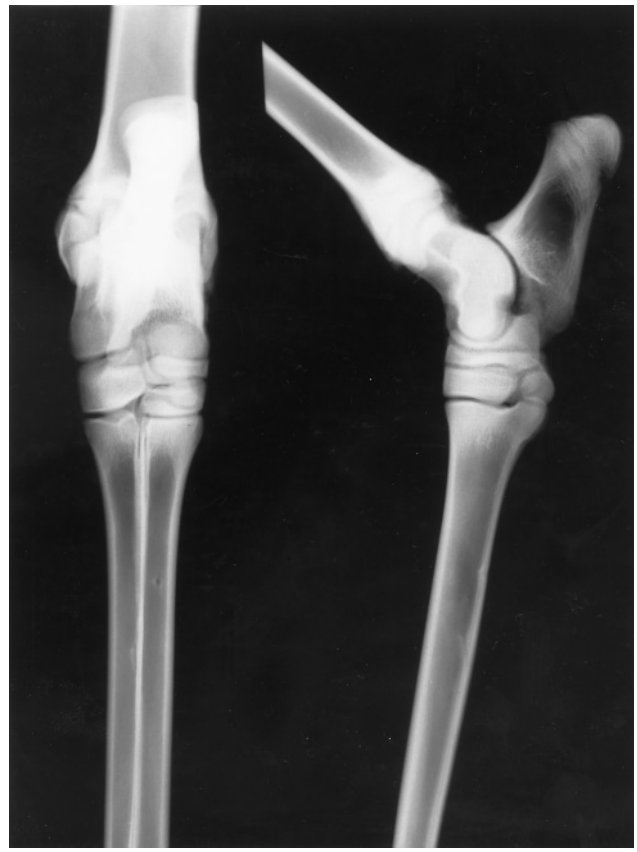

Figure 11.50. Dorsoplantar and lateral radiograph of a neonate llama tarsus.

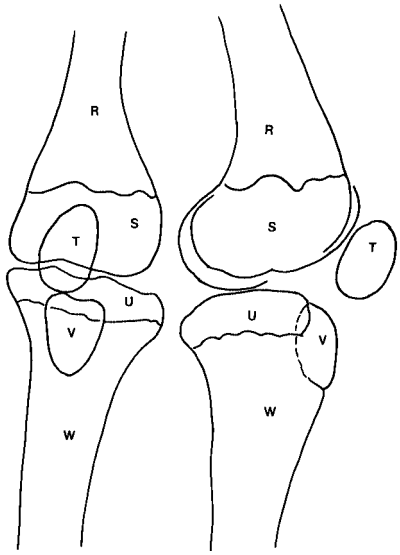

Figure 11.49. Diagram of Figure 11.48. (R) Diaphysis of femur, (S) distal epiphysis of femur, (T) patella, (U) proximal epiphysis of tibia, (V) tibial tuberosity, (W) diaphysis of tibia.

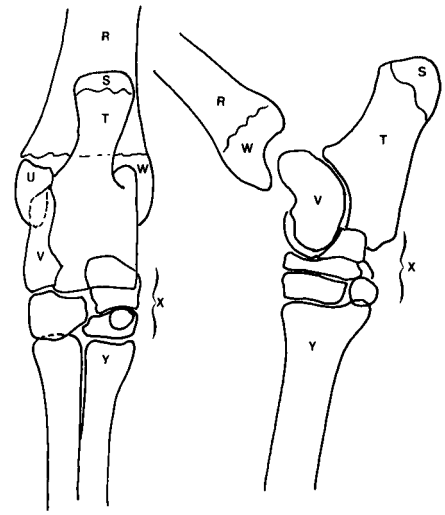

Figure 11.51. Diagram of Figure 11.50. (R) Diaphysis of tibia, (S) epiphysis of fibular tarsal bone, (T) fibular tarsal bone, (U) medial malleolus of tibia (distal epiphysis of fibula), (V) fibular tarsal bone, (W) distal epiphysis of tibia, (X) tarsal bones, (Y) metatarsus.

Figure 11.52. The pedestal callosity in a camel.

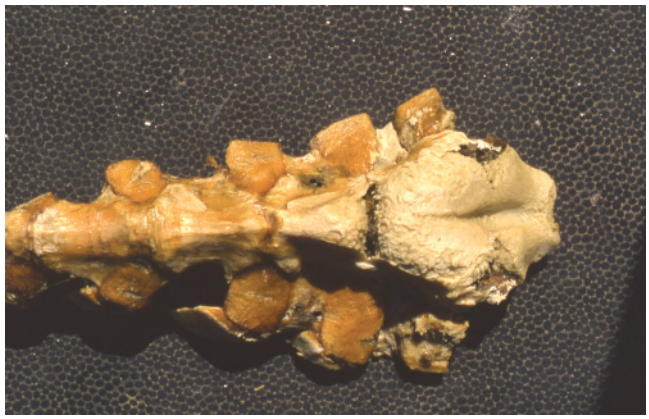

Figure 11.53. The osseous pedestal of the sternum of a camel.

osseous sternum, but in the OWCs the cranial sternabra has a pronounced osseous protrusion (pedestal) that helps to support the larger animal (Figure 11.53).

MUSCULOSKELETAL DISORDERS

Congenital disorders of the musculoskeletal system are discussed in Chapter 22 and include angular limb deformities,[21,22,26] choanal atresia, facial deformities, shortened long limb bones, arthrogryposis, polydactyly, syndactyly, medial luxation of the patella,[2,8] dysgenesis of the maxilla and mandible, hemivertebrae, vertical talus, and contracted tendons. Nutritional myopathy and calcium-phosphorus-vitamin D imbalance are discussed in Chapter 2.

Sternal Trauma in the Camel

The camelid sternal callosity may be subject to laceration, contusions, abrasion, ulceration, osteomyelitis and tumor formation (Figure 11.54, 11.55).

Arthritis

No unique arthritic conditions have been reported in camelids. Traumatic arthritis has been diagnosed in

Figure 11.54. Chronic trauma to the camel pedestal.

Table 11.1. Radiographic technique used on adult llamas.

Region	*MAS*	*KVP*	*Approximate thickness in animals 115–127 kg(cm)*
Head			
L-rostral	100	62	9
L-caudal	100	70	15
DV-rostral	100	70	13
DV-caudal	100	72	18
Vertebrae			
Cervical			
L	200	80	10
DV	100	66	15
Thoracic			
L	100	80	29
Lumbar			
L	200	84	23
DV	200	96	
Pelvis			
L	200	96	30
DV	200	96	30
Articulations			
Scapulohumeral	100	68	34
			23
Humeral-radioulnar			
L	100	66	9
DP	100	66	13
Carpus			
L	100	52	8
DPL	100	52	9
Metacarpal phalangeal	100	50	
Coxofemoral-DV	200	96	30
Femorotibial			
L	100	64	13
DPL	100	64	20
Tarsus			
L	100	50	7
DPL	100	68	9
Thorax–soft tissue	100	76	38

Note: L = lateral; DV = dorsoventral; DP = dorsopalmar; DPL = dorsoplantar.

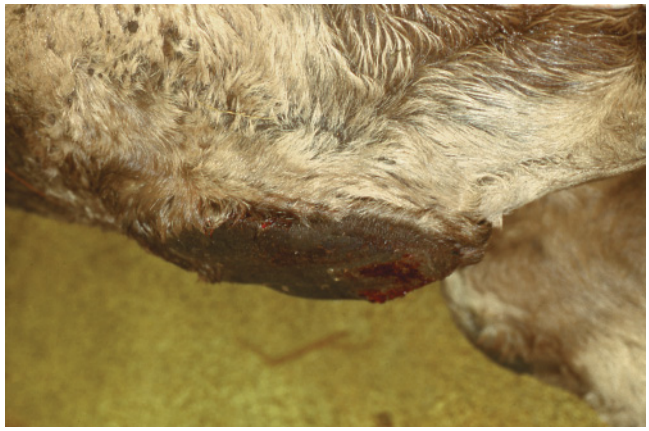

Figure 11.55. Ulceration of a camel pedestal.

the author's practice. Degenerative osteoarthritis may be a sequel to angular limb deformity (Figure 11.56) or result from other unknown etiologies. Infectious arthritis may follow omphalophlebitis or lacerations of the joint capsule or be caused by opportunistic bacteria (Figure 11.57). No specific infectious arthritides have been reported.

Overextension of the Metacarpophalangeal Articulation

Overextension of the metacarpalphalangeal/metatarsaophalangeal articulation (dropped fetlock) has been thought to be caused by a variety of etiologic agents. The only known objective study of these conditions concluded that most of the cases in their study (n12) were not related to degeneration or trauma, but rather caused by a secondary copper deficiency coupled with a zinc excess.[24]

Fractures

Several papers in the periodic literature deal with the prevalence and management of fractures in camelids.[16,19,20,28,29,31] It is evident that most fractures of the long bones are the result of direct trauma to the limbs.[19,20]

It is also noted that SACs are considered to be excellent patients for orthopedic procedures because of their relative low body weight, tolerance of external fixation coaptation devices, and tolerance for prolonged recumbency.[20] The application of fracture repair techniques commonly used in livestock and equine surgery are routinely used in camelids.[18–20,27,28,30]

Vertebral Fractures

Etiology

Vertebral fractures are uncommon in camelids. The probable cause of trauma to the cervical vertebrae and

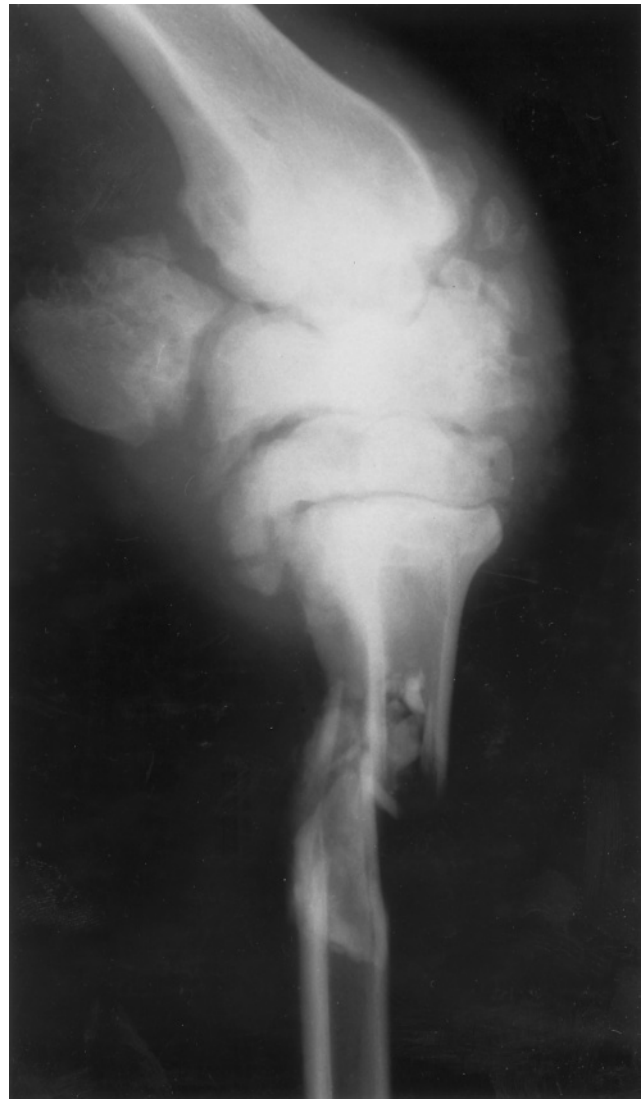

Figure 11.56. Radiograph of chronic osteoarthritis of a carpus and recent metacarpal fracture.

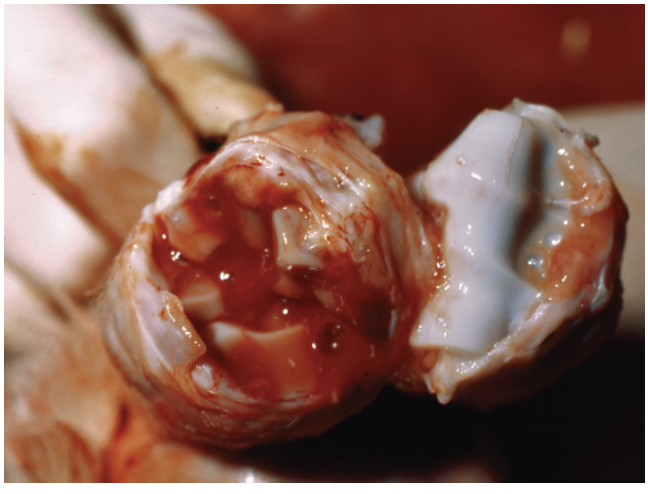

Figure 11.57. Suppurative carpitis.

associated ligaments is a blow to the neck or a struggle against restraint when first being haltered and tied. Lumbar vertebral fractures may be caused by another large animal rearing up and striking down on the back of the victim during inter-male aggression or dominance behavior. A large breeding male may injure a small female as he attempts to force her to recumbency.

Clinical Signs

Variable degrees of ataxia, incoordination, paresis, and paralysis have been observed caused by trauma that involves the vertebral canal and produces compression on the spinal cord or emerging nerves. Fractures of the articular facets of the cervical vertebrae (Figure 11.58) cause the head to be held in an abnormal position. There may be a palpable deformity at the fracture site and evidence of pain on digital pressure. Trauma to the intervertebral ligaments, intervertebral discs, and contiguous muscles produces signs indistinguishable from those caused by osseous lesions.

Diagnosis

Radiography is required for definitive diagnosis. Figures 11.59 and 11.60 illustrate a luxation of the intervertebral disc. A complete fracture of the body of C-2 (Figure 11.61) resulted in total paralysis, necessitating euthanasia.

Management

Vertebral fractures in large animals are difficult to manage. The cervical vertebrae of camelids have no flat surface suitable for plating. Pressure on the spinal cord may be treated with steroids and analgesic anti-inflammatory agents. Activity must be restricted, but camelids do not tolerate slinging well.

Spondylosis[32]

Spondylosis is periarticular hyperostosis with potential bridging between the vertebrae. Extensive spondylosis was seen at necropsy in a twenty-one-year-old female llama (Figures 11.62 to 11.64B). Figures 11.62 and 11.63 are radiographs taken postmortem. This llama also had degenerative carpitis and thus was lame and spent most of the time in sternal recumbency. Whether the spondylosis contributed to reluctance to ambulate is unknown. Spondylosis has been found as an incidental lesion in llamas as young as nine years old.

Rickets[15,16]

Rickets is discussed in detail in Chapter 2.

Carpal and phalangeal valgus is discussed in detail in Chapters 6 and 22.

Myopathy

Etiology

Nutritional myopathy is discussed in Chapter 2. The cellular necrosis caused by nutritional myopathy cannot be differentiated from the lesions of exertional myopathy. Although camelids are usually not willing to exert themselves beyond endurance as pack animals

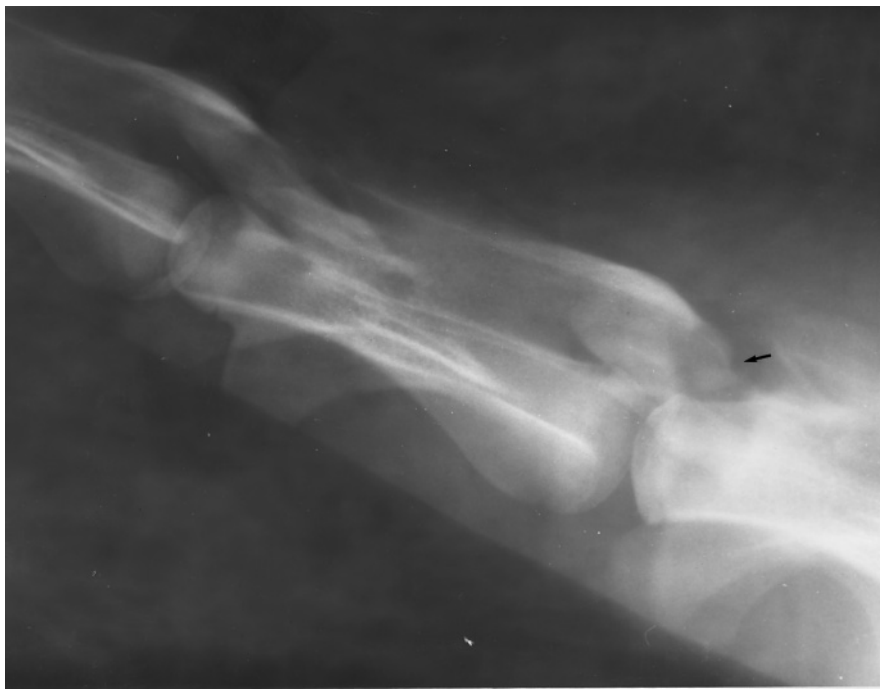

Figure 11.58. Radiograph of a fracture of the articular facet of C-6.

Figure 11.59. Lateral radiograph of the lumbar area with chronic luxation of the intervertebral disc.

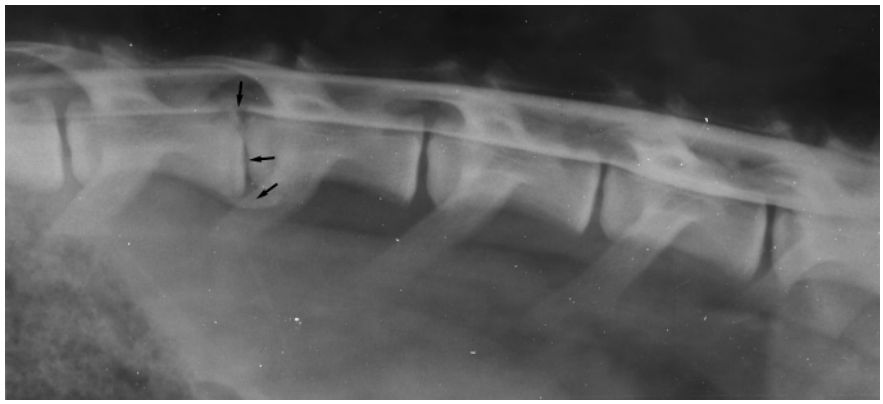

Figure 11.60. Lateral radiograph of the lumbar area with contrast media in the subarachnoid space. Compare with Figure 11.59.

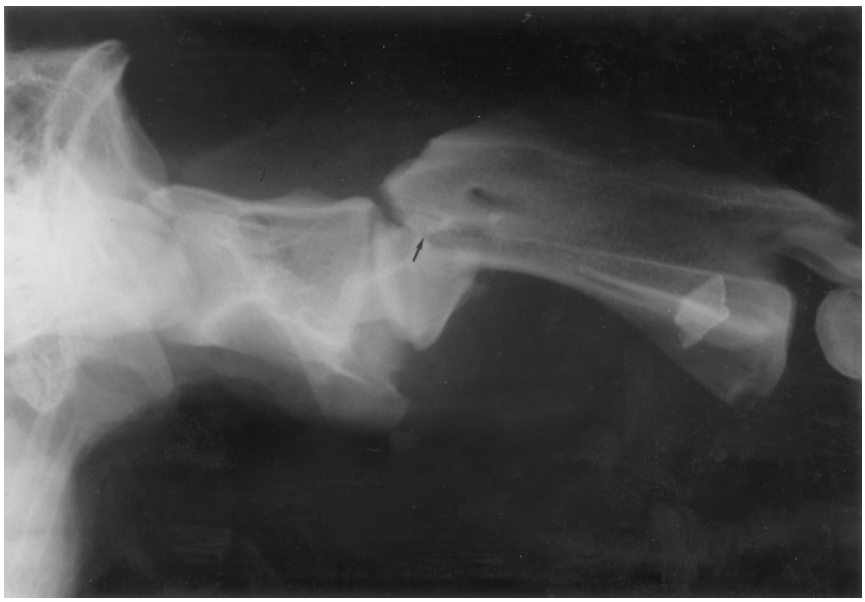

Figure 11.61. Radiograph of a fracture of the dens of C-2.

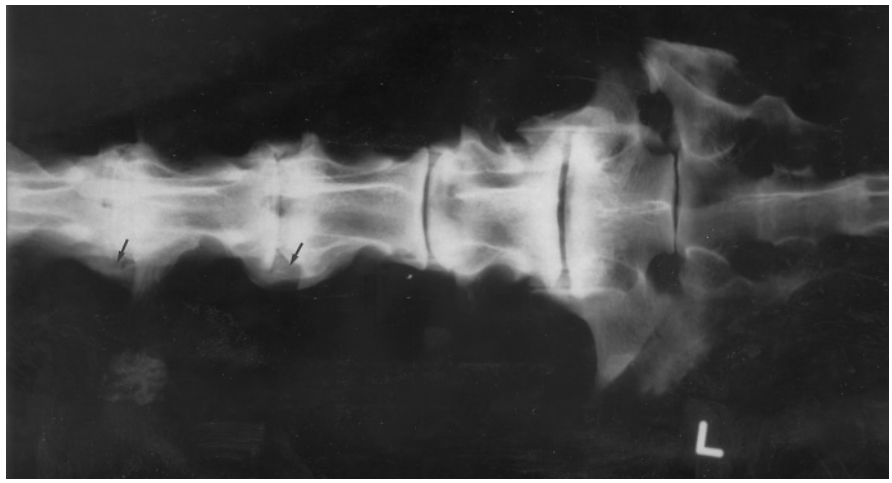

Figure 11.62. Dorsoventral radiograph of lumbosacral spine with spondylosis.

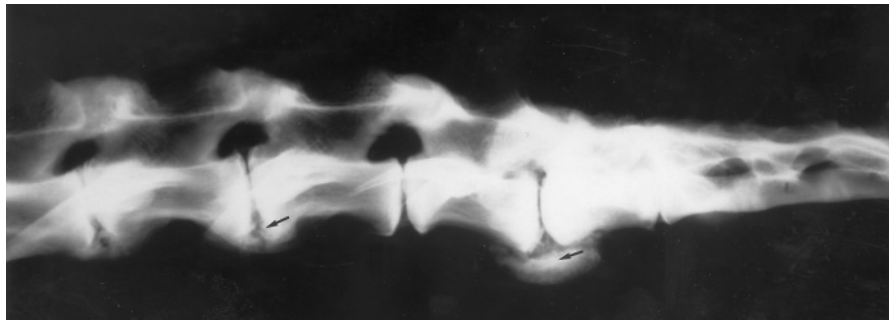

Figure 11.63. Lateral radiograph of lumbosacral spondylosis.

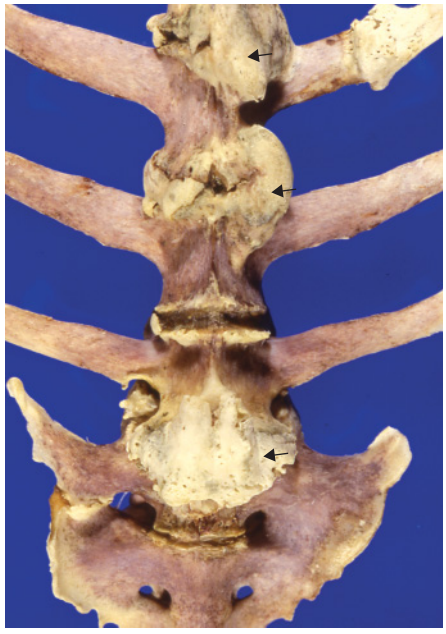

Figure 11.64A. Spondylosis of a llama lumbosacrum ventral view.

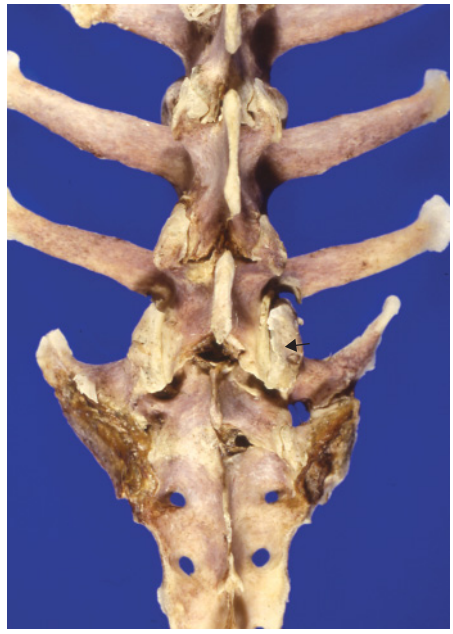

Figure 11.64B. Spondylosis of a llama, dorsal view.

or in a capture/restraint situation, an injured camelid may overexert in a struggle to rise.[14]

Muscle necrosis is frequently diagnosed on gross necropsy. Such a diagnosis should be made only after careful evaluation because postmortem autolysis occurs quickly, giving muscles a whitish appearance. Muscle necrosis may ensue as a sequel to prolonged recumbency of a weakened llama (ischemic necrosis) or from a reaction to an intramuscular injection. Parasitic myositis is caused by *Sarcocystis aucheniae* (Chapter 8).

Clinical Signs

Varying degrees of paresis or paralysis may be seen. Muscles may be hot, swollen, and painful on palpation. Chronic lesions may cause a loss of flexibility and resiliency of the muscle.

A common syndrome has been described as the "downer llama," in which the animal refuses to rise and is frequently hyperthermic. Prolonged recumbency in a camelid prevents cooling via the thermal window (Chapter 9). Muscle biopsies frequently indicate a mild to moderate nonspecific myositis. Some of these animals die despite intensive therapy and support. Few other lesions have been seen at necropsy.

Diagnosis

Muscle-specific serum enzymes may be elevated. However, prolonged recumbency and repeated intramuscular injections cause a mild to moderate elevation in serum enzymes. Diagnosis is made on the basis of clinical signs.

Therapy

Nutritional myopathies may be prevented by appropriate use of selenium/vitamin E preparations, but these are not useful for therapy once the muscle has necrosed. Corticosteroid therapy is indicated for acute, noninfectious myositis. Hot packs applied over the affected muscles enhance circulation and healing. Physical therapy is appropriate, but exercises must be done gently.

Luxation of the Patella

Etiology

Both medial and lateral congenital patellar luxations have been diagnosed in camelid crias. As in the horse, upward fixation of the patella also occurs, but with a different anatomic predisposition. Camelids have a broad patellar ligament rather than the three ligaments of the horse. Fixation in the camelid likely occurs when the patellar ligament is stretched or the bones of the rear limb are overly straight. This is accompanied by a stretching or rupture of the medial or lateral femoropatellar ligament, which allows the patella to move medially or laterally to catch the dorsal aspect of the ridges of the trochlea. Acquired medial and lateral patellar luxation usually follows a traumatic incident that ruptures the medial or lateral femoropatellar ligament.

Camelids tend to have minimal angulation of the upper rear limb bones, but this may be exaggerated to produce a straight or post leg, which causes the patella to ride high on the trochlea. Trauma to the patellar ligament may cause weakening and stretching, which, in turn, allows the patella to ride higher. Such injuries have occurred when a llama attempted to jump a fence and the hind limbs failed to clear it. Stifle injuries may also occur as a result of twisting during chest-butting episodes if one male catches the other slightly off balance. One llama jumped off a simulated bridge in an obstacle course of a show and traumatized the stifle.

Clinical Signs

Medial and lateral patellar luxation is evident on palpation. Lameness caused by pain or mechanical impairment is present. Congenital bilateral patellar luxation causes the cria to assume a crouched position, making ambulation difficult. Heat and swelling may be present. The various compartments of the stifle joint capsule may be distended, depending on the severity of the trauma.

If the patella is locked dorsally, the limb will be fixed in extension, and if forced to move, the llama must drag the limb (Figure 11.65 to 11.66B). With intermittent upward fixation, the signs may vary from an audible click as the patella momentarily locks during ambulation to a lock prolonged for a few seconds, followed by an exaggerated flexion of the limb as the lock is released. Lameness may be evident during periods of quiescence, depending on the cause of the upward riding of the patella.

Diagnosis

Clinical signs and palpation are the primary means of diagnosis. Radiographs add little more than can be determined by palpation.

Treatment

Upward fixation may be released by extending the limb fully while manipulating the patella. Mild upward fixation may be controlled by restricting activity to permit a stretched patellar ligament to heal. Surgical imbrication may be required in severe cases.

Acquired medial and lateral patellar luxations usually result from rupture of respective contralateral femoropatellar ligaments. Surgical correction may be necessary.

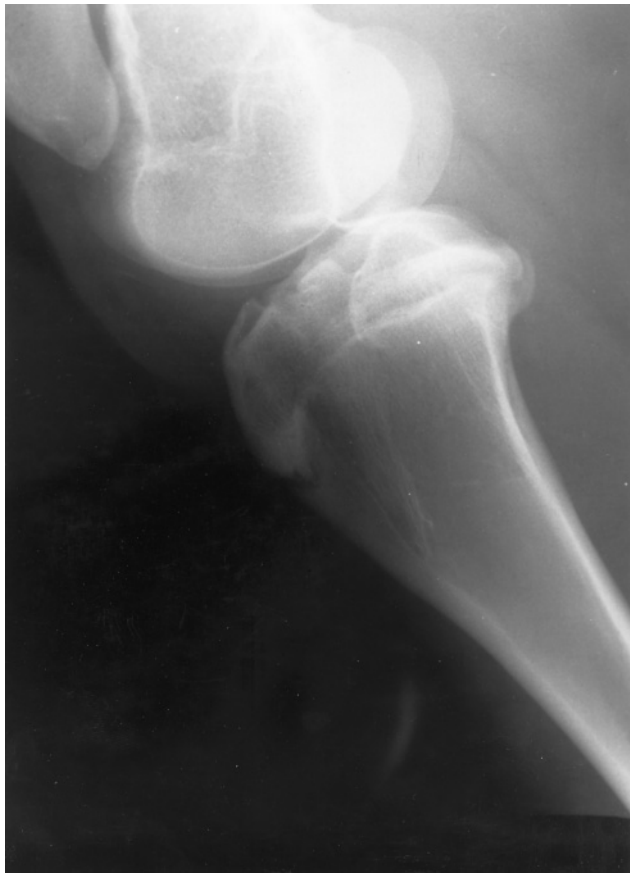

Figure 11.65. Lateral radiograph of upward subluxation of the patella.

Figure 11.66A. Luxation of the patella.

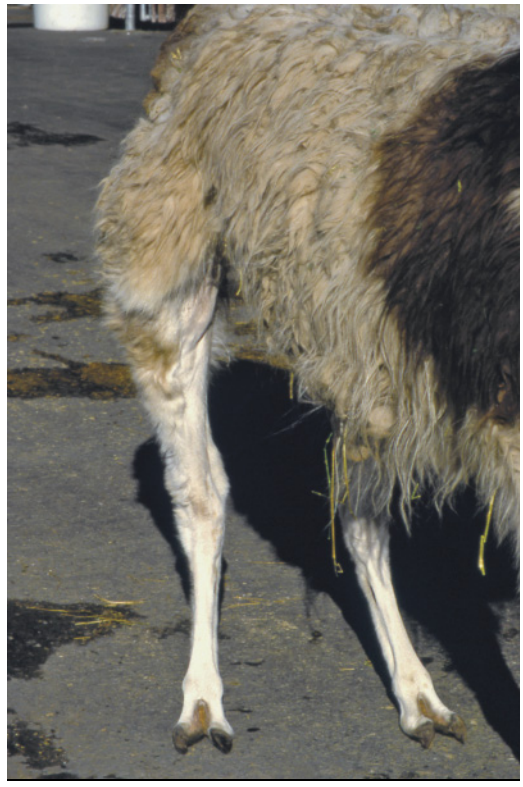

Figure 11.66B. Stance of stifle instability of a llama.

Disorders of the Tail

The tail is an important tool of communication in camelid society. Most desirably, the tail should be straight and fully functional so that neural impulse transmission results in the tail moving appropriately. Just as conformation of the limbs varies from animal to animal, so does the shape and size of the tail. The fiber covering the tail often hides any hint that a tail has a curvature (wry-tail, crooked tail, deviation of the tail, kinked tail, bent tail); however, when the tail is held erect, during a period of increased alertness, the tail may be noticed to deviate to the left or the right. When such a tail is palpated, the bend may be readily apparent or seem to be perfectly normal because the bend is of neural or muscular origin. A curvature may be present at the time of birth, but it is rarely recognized by an owner because interest is centered on more pressing concerns (Figure 11.67).

The Alpaca Registry, Inc., and the Canadian Alpaca Registry have both indicated that a crooked tail is a reason for denying registration to animals being screened, yet there are many possible causes of crooked

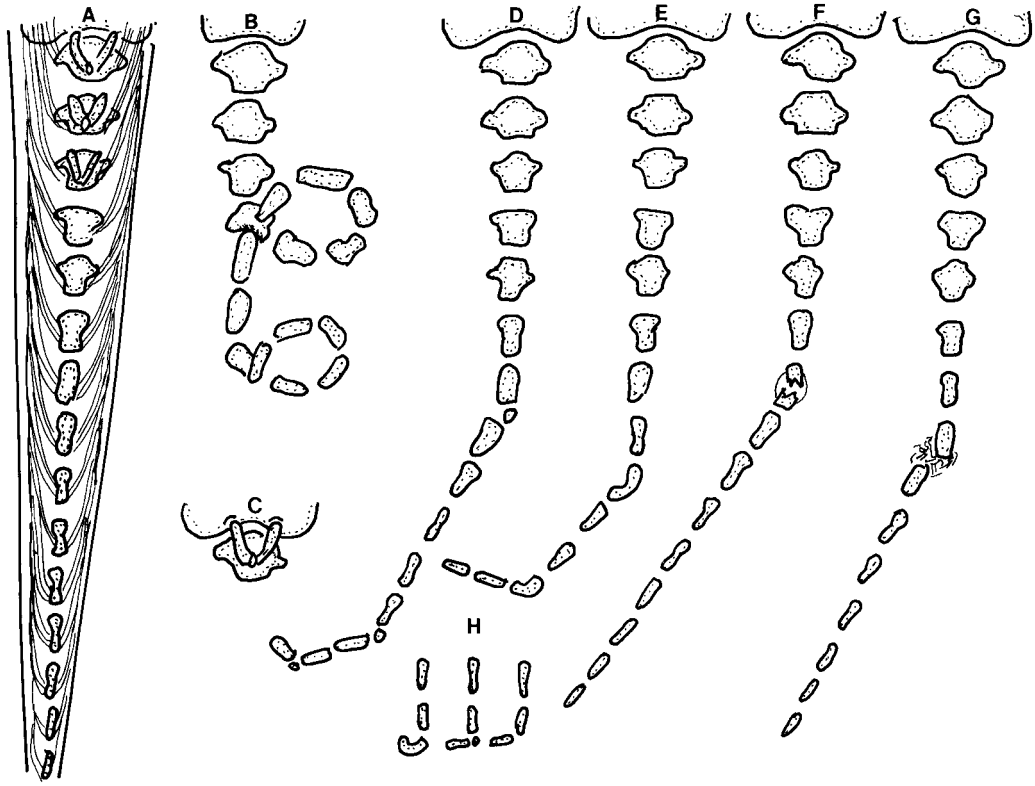

Figure 11.67. Diagrams of the normal and abnormal configuration of the coccygeal vertebrae. (A) Normal, (B) spiral tail, (C) agenesis of the tail, (D) deviation of the tail caused by hemivertebrae, (E) deviation of the tail caused by C-shaped vertebrae, (F) deviation of the tail caused by a fractured vertebra, (G) deviation of a tail caused by luxation of an intervertebral joint, (H) variations in a J tail tip.

tail (Table 11.2). The great concern is that crooked tails may be hereditary. In fact, the first question asked when it seems that a llama or alpaca has a crooked tail is, "Is it hereditary?" It is important to understand that a crooked tail has as much chance of being acquired through an injury as it does through heredity. What difference does it make if a llama or alpaca has a crooked tail? Complete absence of the tail precludes tail posture as a means of conveying certain behavioral information. Malformations may cause postures that convey erroneous information. Disfigurement of the tail may be considered unattractive, but no animal has perfect conformation. A crooked tail has had a significant impact in certain legal cases.

Diagnosis

Paralysis of the tail may result in inability to lift the tail during defecation and urination so that the tail becomes soiled. Manipulation of the tail that allows straightening probably indicates a neural etiology. When the curvature cannot be reduced, radiographs should be taken to identify causes, such as healed fractures, traumatic lesions, or the presence of hemivertebrae (Table 11.3, Figure 11.68). Evidence from other species may indicate that deviation caused by the presence of hemivertebrae may be hereditary, but this has not been verified in llamas or alpacas. Anesthesia is not required, but extremely nervous individuals may require tranquilization. Conduct a thorough examination to eliminate the presence of multiple anomalies.

Scoliosis

Scoliosis may be caused by congenital defects (hemivertebrae), cervical trauma (fractures, subluxation), or myopathy (Figure 11.69).

Tumors

Several osseous tumors have been reported,[3,4,9,14,16] and it is likely that, given time, all types may be diagnosed.

Bone Sequestration

Bone sequestration may been seen in appendicular skeleton bones and in the mandible. Cases have been diagnosed in SACs in Australia and North America. Most cases of bone sequestration in other species is

Table 11.2. Causes of deviation of the tail in llamas and alpacas.

Damage to or malfunction of nerves
Paralysis of the nerves of one side of the tail
Meningeal worm larval migration in the spinal cord
Laceration that severs nerves
Space-occupying lesions in the neural canal (abscess, tumor, hematoma)
Dislocation or fractures of the thoracic or lumbar vertebrae
Nerve impulses stronger on one side of the tail than the other, causing the tail to curve. When such an affected tail is palpated, it may feel perfectly normal.
Damage to or malfunction of the muscles of the tail
Trauma to the tail, sufficient to crush muscles. Scar tissue may shrink and pull the tail to one side.
Mechanical interference with tail posture
Fracture of a coccygeal vertebra with malalignment of the healed bone segments
Partial or complete luxation of a joint between coccygeal vertebrae. The resultant scar tissue may cause the tail to be canted to one side, or chronic pain may cause the animal to favor one side or the other.
Lacerations
Congenital disorders (may or may not be hereditary)
Hypogenesis or agenesis of one or more coccygeal vertebrae
Hemivertebra (partial development of a coccygeal vertebra)
Spiral curvature of the tail. It may be so severe that the tail curls similar to that of a pig or certain breeds of dogs.
C-shaped coccygeal vertebrae

Table 11.3. Some causes of trauma to the tail.

Improper grasping of the tail during restraint procedures
Lifting an animal by the tail
Male biting another male or female during intraspecies aggression
A blow to the tail head from a door closing or a kick from a horse
An animal rearing up and throwing itself backward during a restraint procedure
Infection of the nerves or spinal cord
Vehicle accidents
Dog bite

caused by a traumatic incident, but in those cases seen in SACs there is no evidence of trauma. Hematogenous infection is thought to be a factor.[25]

Clinical Signs

Unexplained lameness is the major presenting complaint. The lameness may be mild or non-weight

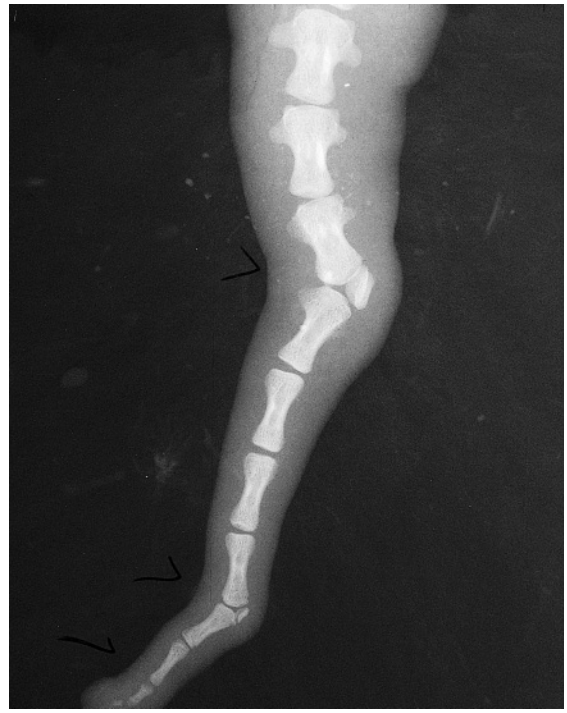

Figure 11.68. Radiograph (dorsoventral view) of deviated coccygeal vertebrae caused by the presence of hemivertebrae.

Figure 11.69. Scoliosis in an alpaca.

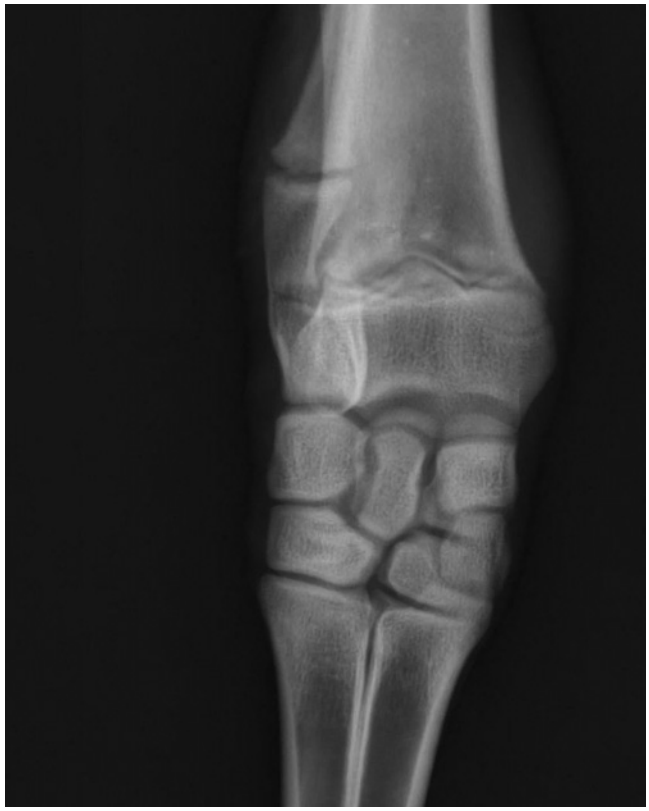

Figure 11.70. Necrotic bone sequestrum in a llama.

bearing. A firm swelling of a bone may be palpated later in the course of the condition. Occasionally a fistulous tract to the bone may be seen.

Diagnosis

Radiographs should be obtained of the affected limb (Figure 11.70). It is important to have multiple views, as clear demarcation of the separated segment may be difficult to see. Fistulous tracts may be cultured, but often culturing will be negative.

Radiographic evaluation is necessary to ensure the removal of the bone segment will not compromise the stability of the bone. If this is threatened the surgeon should be prepared to apply stabilization.

Surgery

The method for removal of the sequestrum depends on its location, size, and presence of contiguous periosteal and new cortical bone formation. Curettage should be used to eliminate any evidence of osteomyelitis. It may be necessary to leave a drain in the wound. Broad-spectrum antibiotics are appropriate for seven to ten days.

Miscellaneous Skeletal Conditions

Hypertrophic osteopathy is a rare condition in camelids, but has been diagnosed in an alpaca.[6] Panosteitis was seen in a camel.[17]

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12

Respiratory System

ANATOMY

The anatomy of the respiratory tract of camelids has not been adequately described in the literature. No texts and only a few papers have been published.[1–9,12,13,18–29,31–35,38] The best anatomic description of the camelid lung is found in Smuts and Bezuidenhout.[36] This presentation does not provide a complete anatomy; however, clinically important anatomic characteristics are described.

Nostril and Nasal Cavity[39]

Nostrils of SACs are not unique. Unlike their Old World cousins, the camels, SACs are unable to completely close the nostrils to exclude dust (Figure 12.1). The nasal cavity has a ventral, middle, and dorsal meatus (Figures 12.2, 12.3). Conchae (turbinates) are arranged in a pattern similar to those of cattle and sheep. The ventral aspect of the ventral meatus is 2 to 3 mm wide. A bulge in the septal mucosa partially occludes the lumen. The dorsal section of the ventral meatus is only 0.7 to 0.8 mm wide. The turbinates are delicate and easily traumatized, so passage of a nasogastric tube should be performed carefully. Small fiber optic endoscopes may be inserted carefully to visualize the nasopharynx.

The nasal orifice of the nasolacrimal duct is located approximately 1 cm dorsal from the floor of the nostril at the junction of the mucocutaneous junction. The hard palate ends approximately 6 cm caudad to the leading edge of the first cheek tooth. Depending upon the stage of breathing or swallowing, the soft palate is elongated in camelids and may lie either dorsal or ventral to the epiglottis (Figures 12.4, 12.5). See Chapter 5 for a more detailed discussion. Camelids are obligate nasal breathers.

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Sinuses

The maxillary and frontal sinuses are delineated in Figure 12.6.

Larynx

The larynx and hyoid bones (with their dimensions) of the llama are illustrated diagrammatically in Figure 12.7.

Trachea and Bronchi

The trachea and bronchi are not unique.

Lungs

Camelid lungs are most similar to those of the horse. A cardiac notch separates the apical portion, but there are no lobes except for the small accessory lobe on the right lung that surrounds the caudal vena cava. Each main stem bronchus divides into an apical bronchus, a cardiac bronchus, and the larger diaphragmatic bronchus. The bronchus to the azygous lobe arises from the diaphragmatic bronchus.

The Clara cell is a nonciliated epithelial cell that occurs in the bronchioles of animals. It appears to be a secretory cell, but its function is unknown. The number of Clara cells per millimeter of bronchiolar epithelium is markedly different between llamas kept at high elevations (55 to 106/mm) and those kept at sea level (8/mm). Mountain air is dry and cold. The secretion of these cells may be of protective value in animals living at high altitude.[19,20]

The mediastinum is complete. The line of diaphragmatic pleural reflection follows a line approximately 23 cm cranial to the costochondral junction and crossing the midportion of the twelfth (last) rib to finish dorsally approximately 4 cm caudal to the twelfth rib.

PHYSIOLOGY

A study of anesthesia in five llamas provided baseline data on the following respiratory parameters: PaO_2 127 ± 8.9 mm Hg, $PaCO_2$ 34.1 ± 1.1 mm Hg, excess base –2.3 ± 1.1 mEq/L, and bicarbonate 21.3 ± 1.1 mEq/L.[16]

The tidal volume of a 105-kg llama was 0.6 L and the calculated dead space 0.33 L. The ratio of dead space volume to the tidal volume of a llama is 0.55 compared with human 0.30, giraffe 0.36, and camel 0.25.[23] The minute volume of the 105-kg llama was 8.5 L/minute.

Figure 12.1. Nostril of a camel. The orifice can be closed tightly to exclude dust.

Camelids are known to be well adapted to the hypobaric environment of the high Andes.[11,24,25,26,28,35,37] Numerous physiologists have studied this phenomenon in an attempt to establish the mechanism(s) for this adaptation. It is probable that a combination of mechanisms involving the respiratory, cardiovascular, muscular, and hemic systems is involved. Some of these mechanisms may have relevance to clinical syndromes and clinical evaluation of the respiratory system.

SAC ADAPTATIONS TO ALTITUDE

1. High hemoglobin concentration of blood.
2. Large numbers of erythrocytes.
3. Small ellipsoid, thin erythrocytes (greater surface area).

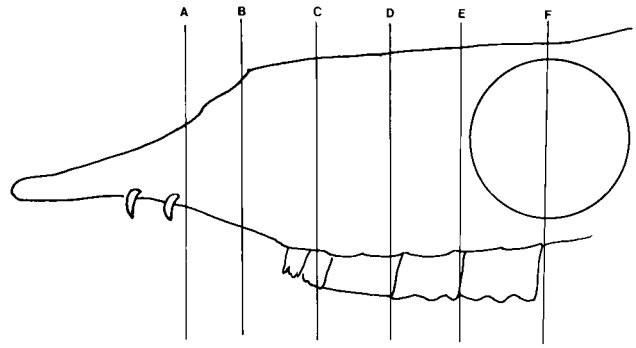

Figure 12.2. Locations of cross-sectional cuts shown in Figure 12.3.

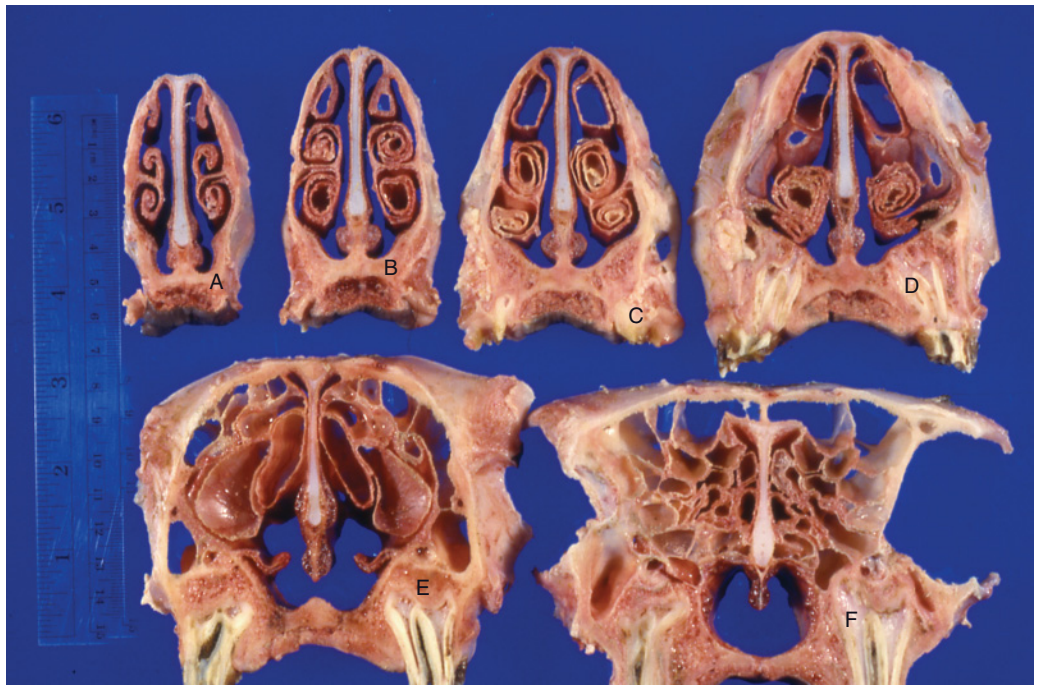

Figure 12.3. Serial cross sections of the nasal cavity of a llama. (A–F) Locations are from Figure 12.2.

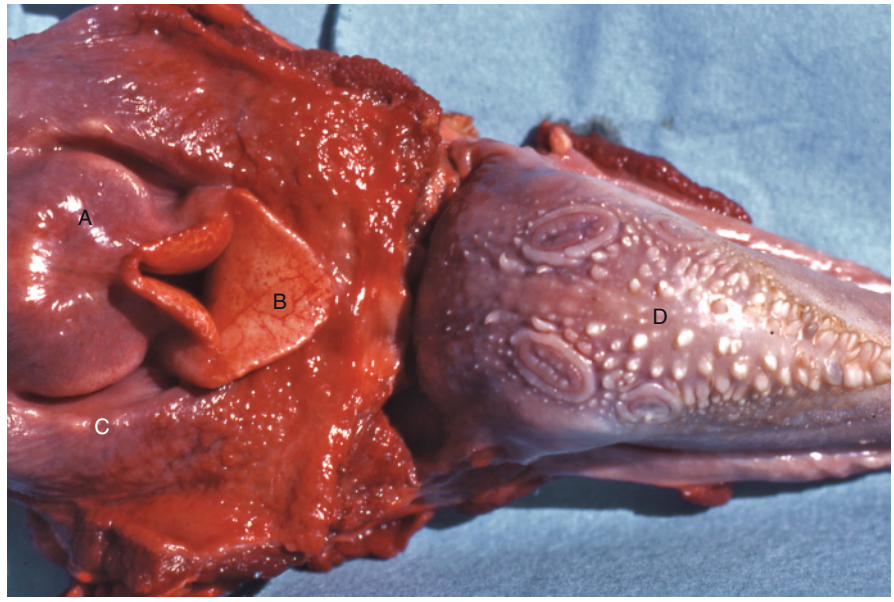

Figure 12.4. Soft palate situated ventral to the epiglottis. (A) Larynx, (B) epiglottis, (C) margin of soft palate, (D) base of the tongue.

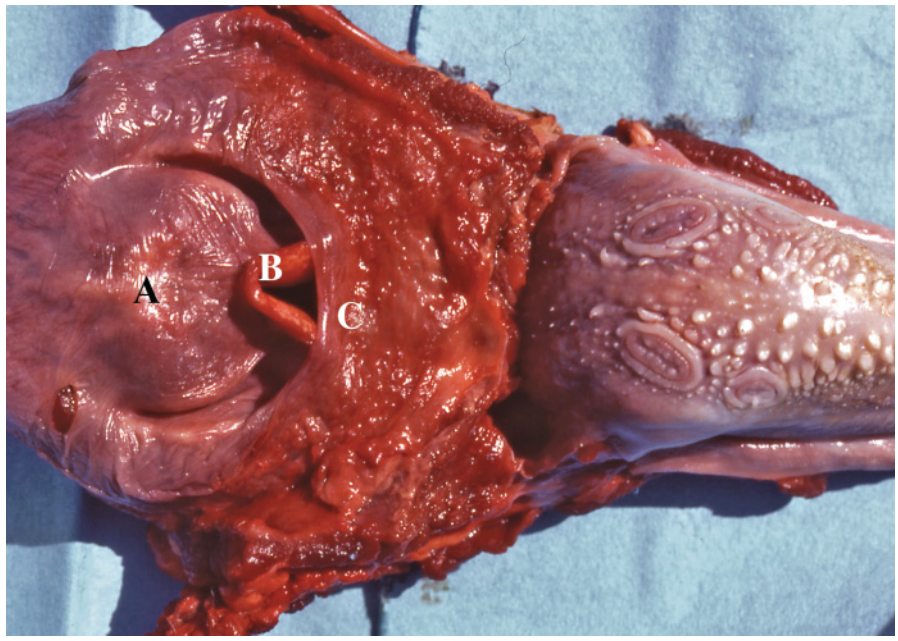

Figure 12.5. Anatomic dissection of the relationship of the soft palate dorsal to the epiglottis. (A) Larynx, (B) arytenoid cartilage, (C) caudal margin of the soft palate.

4. High mean corpuscular hemoglobin concentration, 45%.
5. Mild to moderate degree of pulmonary hypertension at altitude, which does not progress to right heart hypertrophy or thickening of the pulmonary arteries.
6. High affinity of SAC hemoglobin for oxygen.[2]
7. Efficient utilization of oxygen by the tissue (more efficient myoglobin).
8. Oxygen dissociation curves of the blood of SACs are shifted to the left more than in species adapted to low altitudes.[12,13]

DIAGNOSTIC PROCEDURES[17]

Auscultation is described in Chapter 4. The area available for auscultation is much more restricted than would be anticipated by the more caudal line of pleural

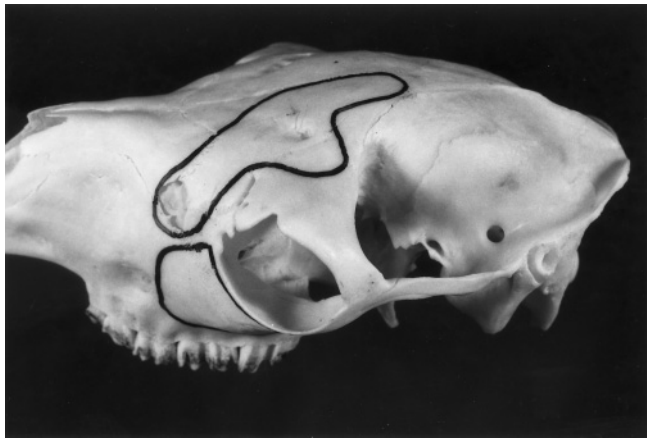

Figure 12.6. Skull, illustrating the outlines of the maxillary and frontal sinuses.

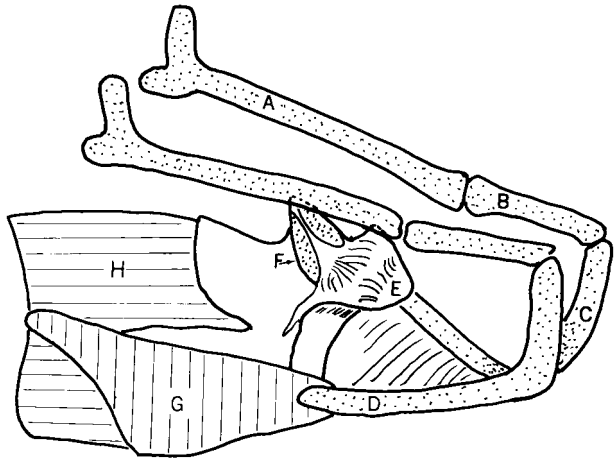

Figure 12.7. Diagram of larynx and hyoid apparatus of a llama (approximate size in an adult). (A) Stylohyoid, 7 cm; (B) epihyoid, 4 cm; (C) ceratohyoid, 5.5 cm; (D) thyrohyoid, 5 cm; (E) epiglottis, 3.5 × 3.5 cm; (F) arytenoid cartilage, 2.5 × 1.5 cm; (G) thyroid cartilage, (H) cricoid cartilage.

reflection. Normal respiratory sounds are muted and may not be audible in the resting animal. Audible sounds are described as bronchovesicular (similar to those of cattle).

Thoracocentesis is described in Chapter 4.

Radiographic evaluation of the thorax is limited to the lateral view in the adult (Figures 12.8, 12.9). Both lateral and dorsoventral projections are possible in neonates (Figures 12.10, 12.11). The technique varies with the area of concern. When radiographing the dorsocaudal area, use an ffd 152 cm (60 in.), 1/50th second, milliamp seconds (MAS) 10, and kilovolt peak (KVP) 100. In the cranial area use the same ffd, time 1/25th second, 20 MAS, and 120 KVP. At Davis, a grid is placed on a Dupont Quanta 3 screen cassette, and Dupont Chronex 7 film is used.

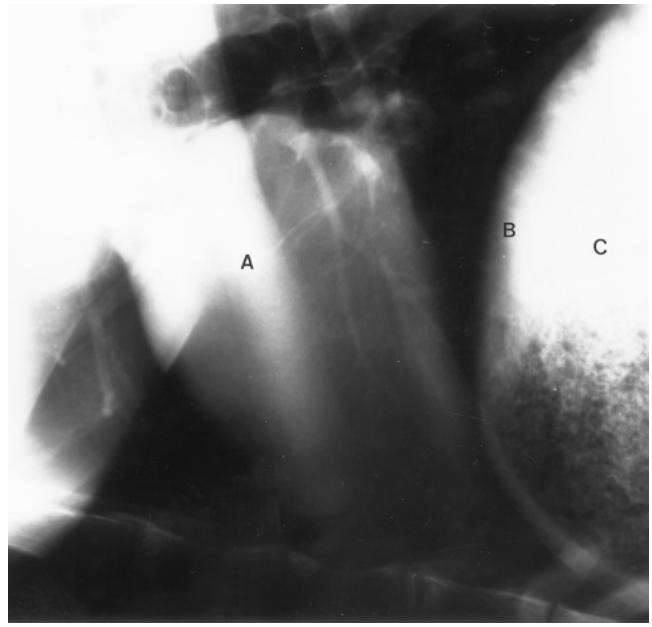

Figure 12.8. Lateral radiograph of the cranial thorax of a llama. (A) Heart, (B) diaphragm, (C) C-1 of stomach.

INFECTIOUS DISEASES

Infectious diseases of the respiratory system include tuberculosis, coccidioidomycosis, actinobacillosis, and nonspecific ailments such as fusobacteriosis (Figure 12.12) caused by opportunistic bacteria. Discussions of these diseases are found in Chapter 7. Viral diseases of the camelid respiratory system are rare. There are reports of infectious bovine rhinotracheitis (IBR) occurring in llamas, and titers indicate that llamas respond to antigenic stimulation. The acute upper respiratory syndrome is thought to be of viral origin (see Chapter 7).

PARASITIC DISEASES

Camelids are susceptible to lungworm (*Dictyocaulus viviparous*) and nasopharyngeal bots (*Cephenemyia* spp.), both of which are described in Chapter 8. The lungs may also serve as a migratory route for larval parasites such as the meningeal worm (*Parelaphostrongylus tenuis*).

CONGENITAL DISORDERS

Congenital disorders include facial deformities, choanal atresia, and cleft palate, all discussed in Chapter 22.

MISCELLANEOUS DISEASES[10,15,30]

Trauma

Etiology

Injuries to the muzzle (dog bite, contusion) or nasal cavity causing swelling of the tissue may occlude the

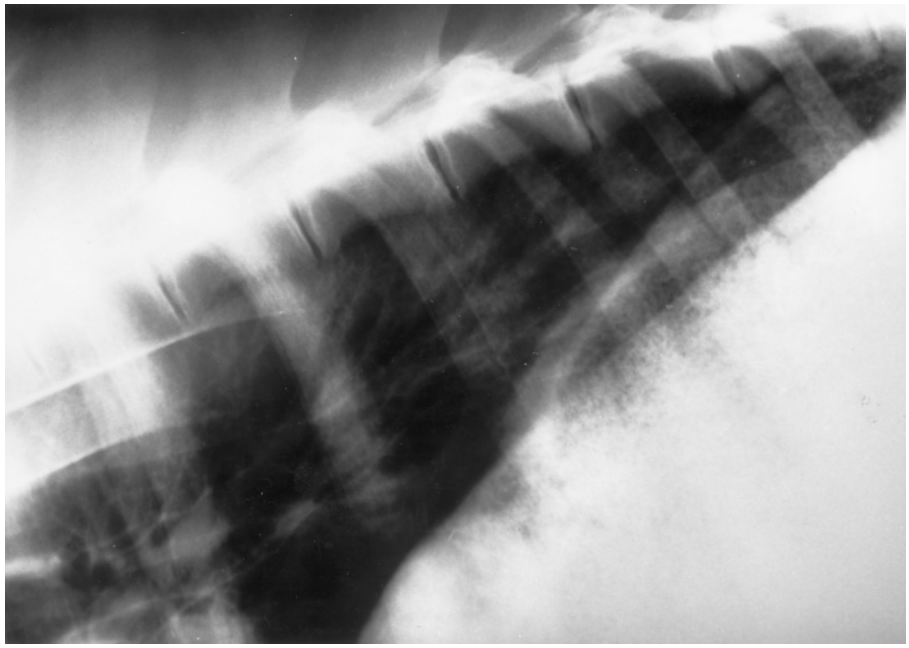

Figure 12.9. Lateral radiograph of the caudal thorax of an adult llama.

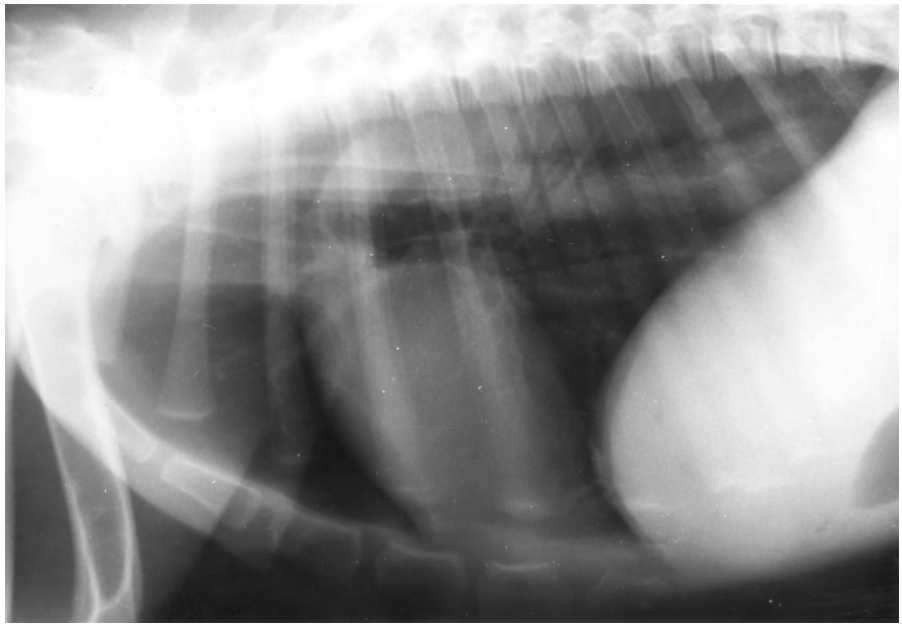

Figure 12.10. Lateral radiograph of a neonate llama thorax.

passageways, resulting in dyspnea. Rattlesnake bites of the nose cause edema and occlusion of the nares. Llamas have strangled from occlusion of the nostril or trachea when they have become entangled in the halters and ropes used to secure them in trailers for transport. It is not desirable to tie camelids during transport.

One llama strangled as a result of a strange accident during trailering. The llama was tied over a bar at the front of a horse trailer. At some time during the trip the llama apparently was thrown forward, hitting the bar at the level of the larynx and upper trachea. The llama was found dead, hanging with the halter tightly occluding the nostrils and the mouth. A massive hematoma was found in the pharynx and larynx. The llama had apparently pulled backward, occluding the nostrils and mouth and restricting the flow of blood from the contused site.

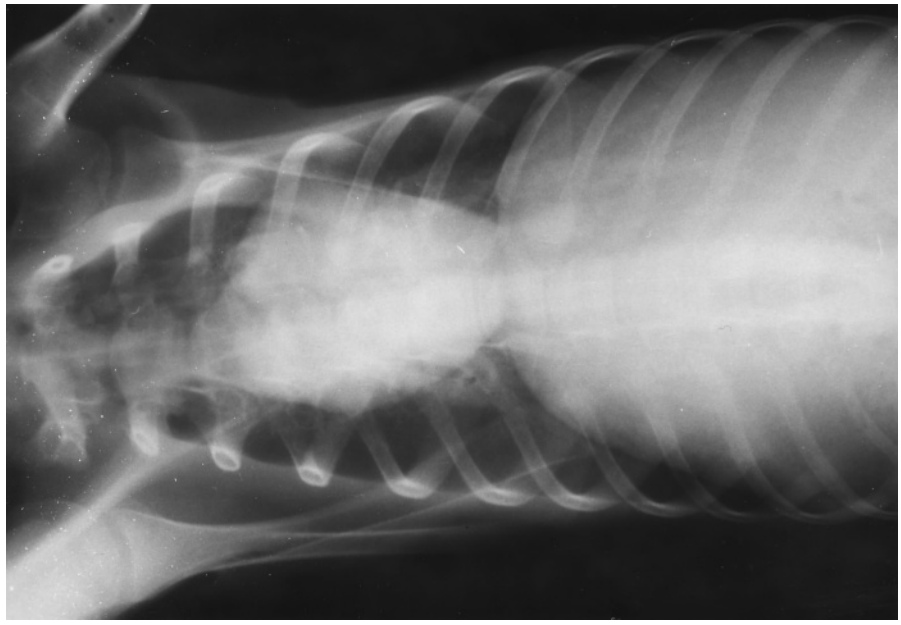

Figure 12.11. Dorsoventral radiograph of a neonate llama thorax.

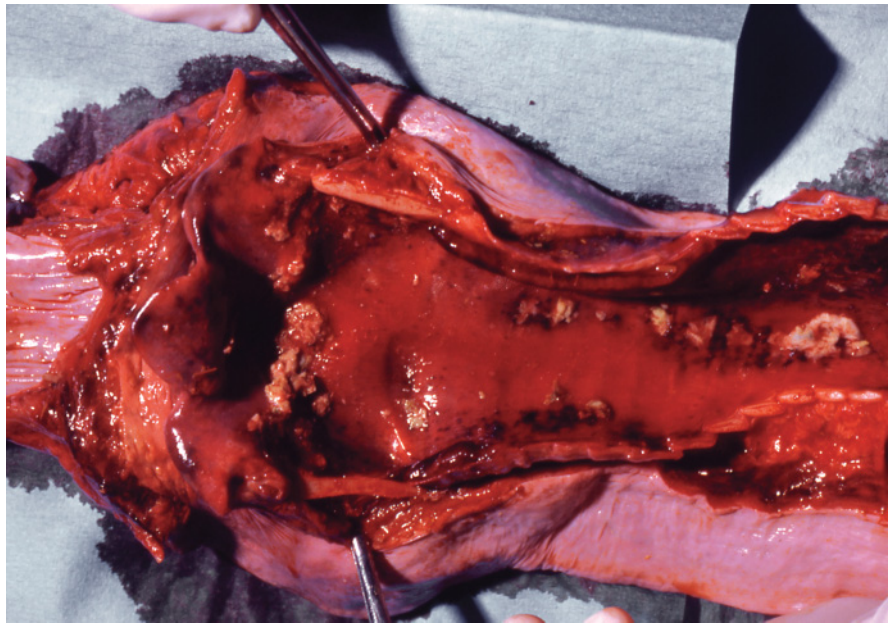

Figure 12.12. Necrotic laryngotracheitis.

Lacerations, contusions, and foreign-body penetration of respiratory organs are similar to these accidents in other species. Llamas have been found upside down in ditches and other declivities, unable to extricate themselves. If left in this position for more than a few minutes, bloat or passive regurgitation and aspiration of stomach contents may occur, strangling the animal.

Bloat is rare in camelids, but one case has been reported of esophageal obstruction caused by ingestion of a whole apple, with death resulting from bloat and internal pressure on the diaphragm and lungs.

Clinical Signs

Because camelids are primarily nasal breathers, any impairment of air passage from the nostrils results in dyspnea and open mouth breathing. Partial occlusion of the upper airways may result in stertorous breathing.

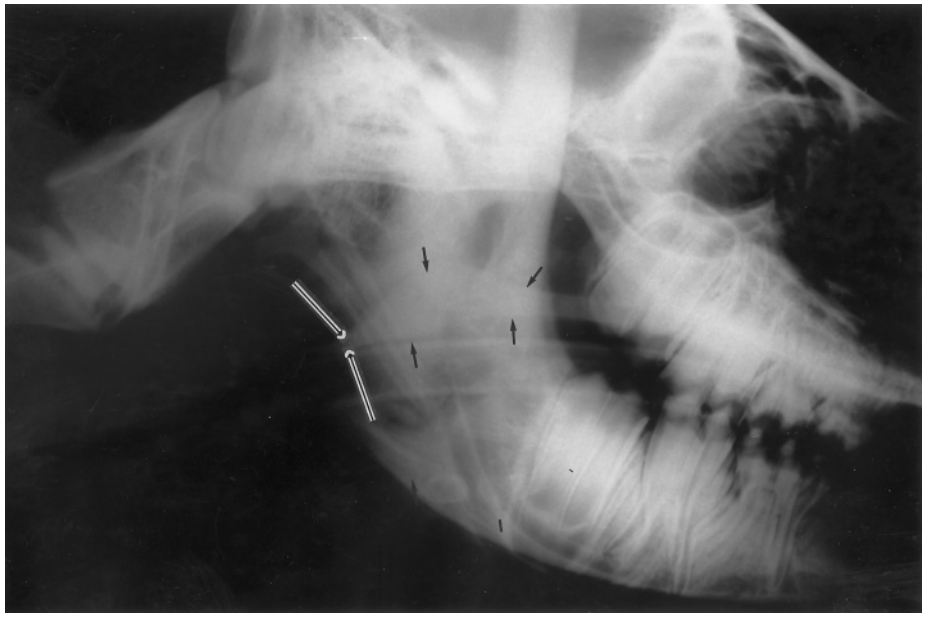

Figure 12.13. Hyperplasia of soft palate.

Management

Occlusive lesions may necessitate tracheostomy to preserve the life of the animal (Chapter 6). The primary lesion must be dealt with as in any animal species.

Pneumonia

Pneumonia is often a presumptive diagnosis in llamas, especially neonates, based on increased respiratory sounds and some degree of dyspnea. Pneumonia is also a frequent diagnosis at a gross necropsy because the normal lung tends to be slightly edematous and hyperemic. That pneumonia occurs there is no doubt, but a thorough examination and evaluation are necessary to exclude diseases of other organ systems. Pulmonary edema is a common terminal lesion seen in animals dying from numerous diseases (Figure 12.13).

Etiology

The causal bacterial agents of camelid pneumonia are similar to those causing pneumonia in livestock and horses. Most infectious cases result from opportunistic bacteria. Septicemic animals usually develop pneumonia, and the most common agent isolated in the author's practice has been *Escherichia coli*. Other causes of pneumonia include inhalation of toxic vapors. *Actinomyces lamae* may produce abscessation of the lung.

Aspiration of stomach contents occurs in the orphaned neonate being fed from a bottle or while being stomach tubed. Passive regurgitation during anesthesia is a significant risk. If surgery entails prolonged left lateral or dorsal recumbency, it is advisable to intubate the trachea with a cuffed tube and to position the head and upper neck so that stomach contents can flow freely from the mouth.

Clinical Signs

Signs are exaggerated in the neonate and include dyspnea, coughing, elevated body temperature, variable nasal exudation, depression, and anorexia. Sounds heard at auscultation vary with the degree of exudation and consolidation.

Diagnosis

A hemogram should be done. With bacterial or fungal infections, there will be an elevated leukocyte count and left shift. Radiographic evaluation is useful. A transtracheal wash can be used to collect material for culture and sensitivity. To do this, an area over the trachea in the midcervical region is prepared aseptically and a 15-gauge needle inserted between the tracheal rings. A sterile catheter is threaded through the needle. Position the neck to horizontal and flow 5 to 10 ml of nonbacteriostatic water or saline into the trachea with a pumping action of the syringe to aspirate exudate. Collected material should be examined for cytology and cultured.

Therapy

Broad-spectrum antibiotic therapy is recommended until sensitivity results are available, because Gram-negative organisms are frequently involved. General nursing care and supportive treatment are indicated. Nebulization may be helpful in mobilizing exudates. Fifty percent dimethylsulphoxide (DMSO)

in aqueous solution has been efficacious in the author's practice.

Pleural Effusion

Etiology

Pleural effusion is usually secondary to pleuritis, pericarditis, or right heart insufficiency. Generalized lymphosarcoma with mediastinal lesions resulted in incapacitating pleural effusion.[14]

Clinical Signs

Inspiratory dyspnea is the most prominent sign. The absence of sounds in the lower thorax and a dull sound on percussion in the same area are diagnostic. A definitive diagnosis is based on radiography and thoracocentesis. It is imperative that the nature of the pleural fluid be determined, because it may be a modified transudate or exudate.

Management

Therapy is determined by the etiology. Excess fluid may be removed via thoracocentesis, but the critical factor is to prevent recurrence. The prognosis for a neoplasm is grave. Infectious pleuritis should be treated with broad-spectrum antibiotics until results of culture and sensitivity tests are known.

Hypertrophic Osteopathy

Hypertrophic osteopathy has been reported in an alpaca. The definitive identification of the inciting cause was not determined; however, at necropsy a chronic pneumonia was found in the ventral aspect of the left apical lobe of the lung. There were also sarcocystis lesions in skeletal muscle.[14]

Exercise Intolerance

Etiology

The llama was domesticated as a beast of burden in the high Andes.[22] Many llama owners have purchased animals for the purpose of packing with them in the wilderness. A few llamas have either refused to carry packs or have been unable to exert themselves sufficiently to make worthwhile pack animals.

A number of defects within the respiratory system may contribute to exercise intolerance. Crias afflicted with congenital bilateral choanal atresia usually die, but unilateral or partial stenosis of the choanae could restrict air flow later in life. Congenital narrowing of the nasal cavity is recognized and causes stertorous breathing, particularly if the animal is excited or exercised. Lesions within the nasopharynx such as polyps, lymphoid hyperplasia, or granulomatous lesions caused by *Cephenemyia* spp. have caused restricted air flow. Hyperplasia of the soft palate (Figure 12.13) of unknown etiology results in reduced air flow. Previous pneumonic episodes may have left areas of the lung fibrotic and nonfunctional.

Other nonrespiratory causes should be considered, such as ventricular septal defects, anemia, hepatic insufficiency, renal insufficiency, arthritis, and myopathy.

Clinical Signs

The primary sign is the inability of the animal to work up to the potential for its stage of training. Most llamas simply slow down or stop if the exertion is beyond their capability. Some animals may show open-mouth or audible breathing if the lesion is in the upper airway. Careful consideration should be given to the weights expected to be carried. Small animals (100 kg) should not be expected to start with more than 20 to 25 kg and should carry only 36 kg when trained and fit. Larger animals may carry 45 kg.

Diagnosis

With the diversity of causes, a thorough physical examination is necessary, possibly including hematology, serum biochemistry, and radiography of the head and thorax. Lesions within the nasal cavity and the nasopharynx may be explored by endoscopy.

Management

Many of the lesions causing exercise intolerance are either congenital or chronic in nature. Little can be done to correct these, but an appropriate prognosis may be given. Infections with *Cephenemyia* spp. are treated with ivermectin (0.2 to 0.6 mg/kg). Surgery may be appropriate to correct hyperplastic lesions of the nasal cavity or nasopharynx.

Drowning

Description

Camelids are able to swim, but circumstances may arise in which they become trapped in deep water, sink, and inhale water into the lungs.

Etiology

Natural aggression resulted in the drowning of two male llamas. They were pastured on opposite sides of an arm of a lake. When the water froze, the two animals attempted to cross the ice to fight with each other. They both broke through the ice and could not be rescued before they became exhausted, sank into the water, and drowned. A heavy fleece is a serious detriment to a llama trapped in water over its head. Floods have trapped and drowned camelids.

Management

The first step is to quickly remove the animal from the water, which may be extremely difficult with a

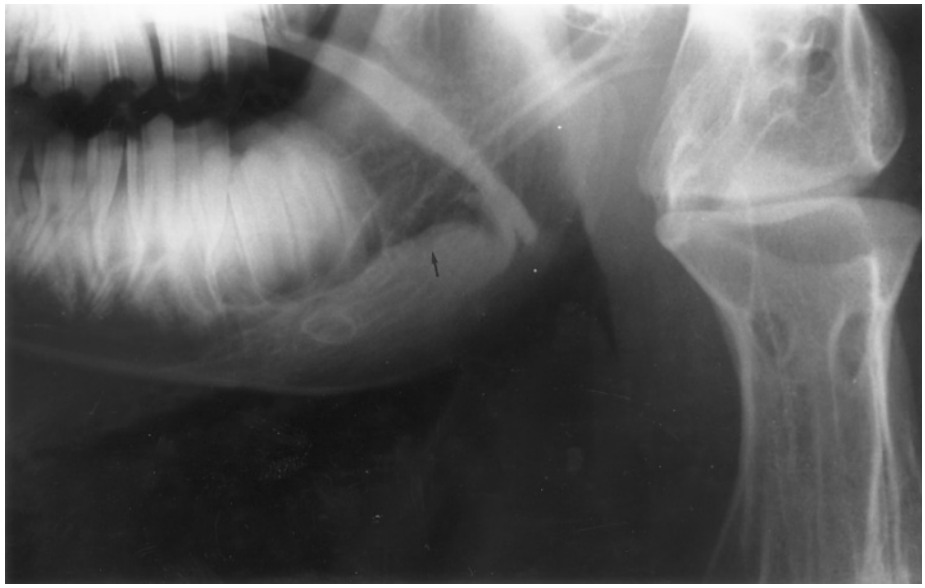

Figure 12.14. Subepiglottic mass.

large llama. Place the camelid on its right side with the head and neck lower than the body so that water can flow out. There is a risk of producing regurgitation in this position, but the need to remove the water from the lungs must take precedence. Carry out artificial respiration (Chapter 4). If immersion lasted for more than two to three minutes, the heart may have stopped, making cardiac massage necessary. Massage and artificial respiration must be continued until the victim responds or is perceived to be dead. Although people have been resuscitated after being under water for twenty to thirty minutes, especially if immersed in cold water, it is highly unlikely that resuscitation will be successful in a camelid that has been immersed for more than five minutes.

Other Miscellaneous Conditions

The following conditions have been diagnosed, but no etiologic agent was identified: necrotic laryngotracheitis (Figure 12.13), hyperplasia of the soft palate (Figure 12.14), and subepiglottic mass (Figure 12.15).

ARTIFICIAL RESPIRATION

Artificial respiration is indicated for any case of apnea, drowning and electrocution. The technique is as follows: with the animal in lateral recumbency, stand at the withers and reach over the body and grasp the last rib and lift the rib cage. This expands the chest and pulls air into the lung. Expiration is accomplished by pressing on the chest wall just behind the front leg. Do not press on the rib cage near the last rib because this puts pressure on C-1 of the stomach, with the risk of forcing ingesta up the esophagus (Figures 12.16 and 12.17).

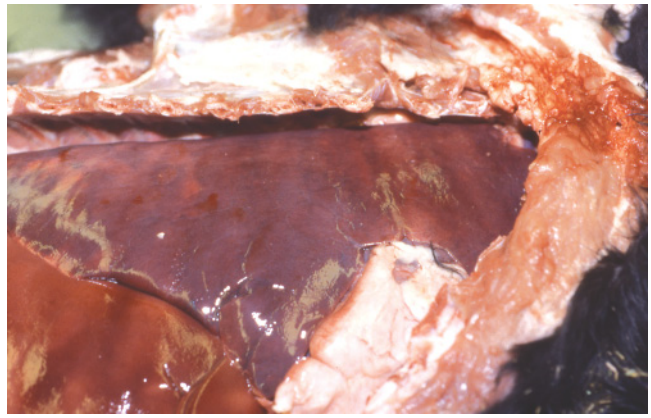

Figure 12.15. Pulmonary edema.

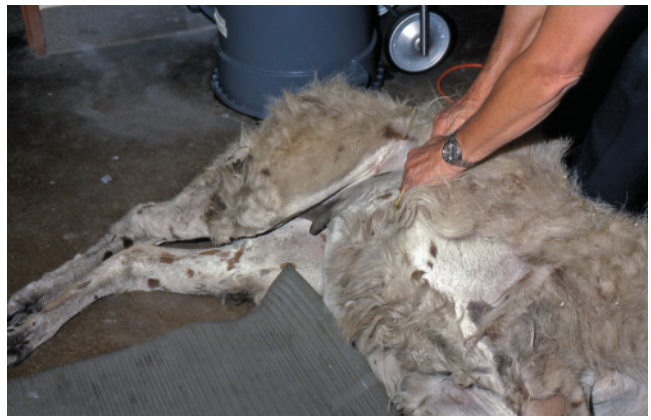

Figure 12.16. Artificial respiration. Lifting rib cage for inspiration.

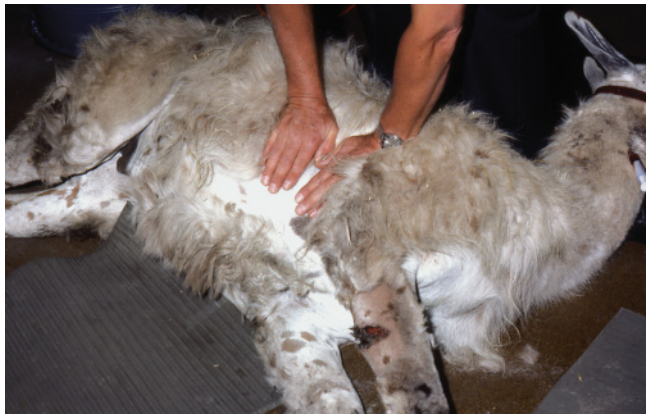

Figure 12.17. Artificial respiration. Pressing close to the front leg for expiration.

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13

Digestive System

The digestive and reproductive systems of camelids have been the most intensely studied of any of the organ systems. Both systems have unique characteristics, which constitute some of the major differences that separate camelids from ruminants in the order Artiodactyla. Both Old and New World camelids evolved in harsh but differing environments. The two environments had in common a characteristic of sparse, poor quality forage for at least part of the year.

It appears that the common ancestor of both camelids and ruminants was simple stomached, but in camelids, a foregut fermentation system and a rumination cycle evolved in parallel with the digestive systems of ruminants. Numerous other morphologic differences of the digestive system affect diagnosis, treatment, and management of diseases in these animals.

ANATOMY AND PHYSIOLOGY[84]

Lips

The upper lip of camelids is split by a philtrum (labial cleft) (Figure 13.1). Each side of the lip can be manipulated independently by the elevator nasolabialis muscle under the control of the facial nerve. The upper lip is highly tactile, useful for fine discrimination, and the camelid uses it as a sense organ to investigate potential feed. As a result of this fastidious feeding behavior, gastric foreign bodies are rarely found in SACs. Old World camels are not so discriminating. The lower lip has no unique characteristic, but it is less mobile than the lower lip of the caprine or ovine.

Mouth

The oral cavity of the llama is small. The rami of the mandibles are set close together, and the tongue occupies the ventral space. The lips are tightly opposed to the teeth. It is rare for a cud to become trapped in the buccal cavity. Camels have a larger oral cavity and the mouth is capable of opening widely.

Tongue

The tongue of camelids is relatively immobile. It is rarely extended beyond the lips and is not used for prehension of feed. The camelid does not lick the neonate nor does it remove any attached fetal membrane. In a 150-kg llama male, the tongue was 20 cm long, 3 cm wide, and 2 cm deep in the anterior two-thirds. The caudal third had a pronounced dome (5 cm) (Figure 13.2). The camel tongue is proportionately larger.

Oropharynx

The major morphologic traits of this area that have clinical significance are the narrow oropharyngeal space, the elongated soft palate, and the domed base of the tongue. Camelids are obligate nasal breathers, with inefficient mouth-breathing capabilities. With nasal obstruction, simultaneous eating and breathing are difficult. Breathing takes immediate precedence, and the animal will slowly starve if nasal obstruction persists.

Esophagus

The camelid esophagus is similar to that of ruminants. It is not known where the stimulus arises, but camelids are more prone than the bovine to regurgitation during passage of a stomach tube.

Teeth[16,54,58,63,64,117]

The dental anatomy of camelids sets them apart from ruminants. A comparison of the deciduous and permanent dental formulas for camelids and bovines is given in Table 13.1. The tabulation presents only part of the picture. The single upper incisor of both SACs

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and camels has migrated caudally and evolved to a caniniform shape and function. With the mouth closed, this tooth rests just caudad to the corner lower incisor. All four canine teeth are present in adult males. Therefore, male SACs appear to have two upper and one lower canine on each side (Figure 13.3).

Camels have more premolars than SACs, but the first upper and lower premolars also have migrated forward in the jaw and become caniniform. Thus, the camel appears to have three upper and two lower canines on each side (Figure 13.4). In contrast, cattle, sheep, and goats appear to have four lower incisors and no canines, but the corner incisor is actually a canine tooth that has migrated forward and become incisiform.

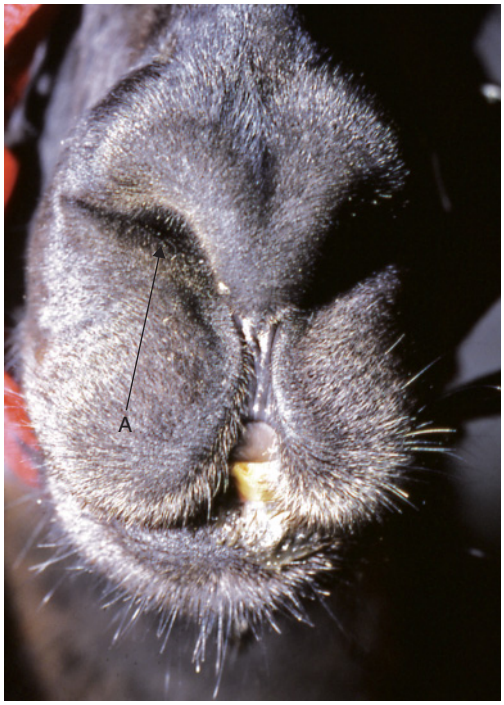

Figure 13.1. Philtrum in a llama. (A) Approximate location of the nasolacrimal duct orifice.

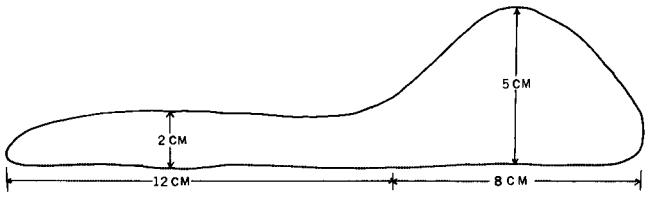

Figure 13.2. Diagram of the tongue of a llama.

Incisors

Camelid incisors are anchored firmly into the alveolus, as in sheep, but in contrast to cattle in which the teeth are movable in the alveolus. All SACs have the same dental formula, but vicuña incisors differ in shape and structure from those of other artiodactylids; vicuña incisors are more like the continuously erupting incisors of rodents. The permanent incisors of the vicuña are parallel sided, 10 times as long as they are wide, with a square cross section. The crown enamel is confined to the labial surface, extending down the root to within 2 to 3 mm of the wide, open base (Figure 13.5). The occlusal surface of the vicuña incisor is comprised of dentine, which is softer than the labial rim of enamel; thus, the tooth remains shaped like a chisel.

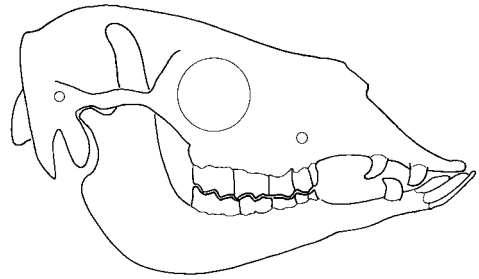

Figure 13.3. Diagram of the lateral view of a male llama skull.

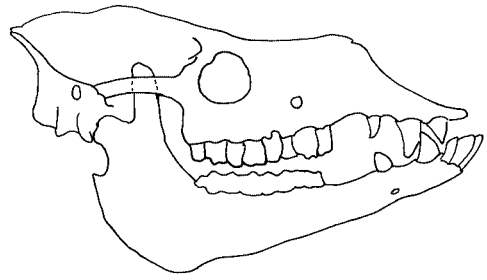

Figure 13.4. Diagram of the lateral view of a male camel skull.

Table 13.1. Dental formulas of some artiodactylids.

	SACs	*Camels*	*Cattle, sheep, goats*
Deciduous	I 1/3, C 1/1 PM 2–3/1–2 × 2 = 20–22	I 1/3, C 1/1 PM 3/2 × 2 = 22	I 0/3, C 0/1 PM 3/3 × 2 = 20
Permanent	I 1/3, C 1/1 PM 1–2/1–2, M 3/3 × 2 = 30–32	I 1/3, C 1/1 PM 3/2, M 3/3 × 2 = 34	I 0/3, C 0/1 PM3/3, M 3/3 × 2 = 32

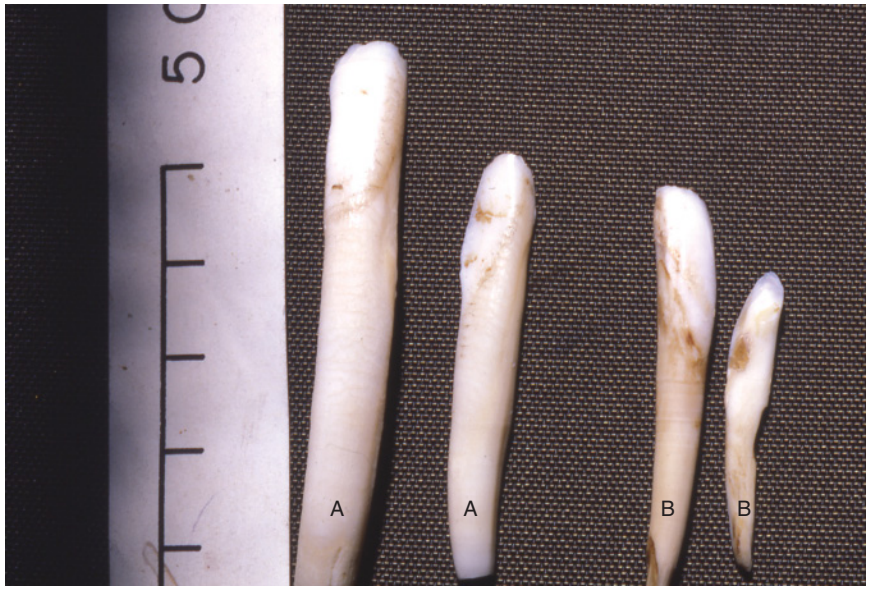

Figure 13.5. Incisor teeth of (A) vicuña, (B) alpaca.

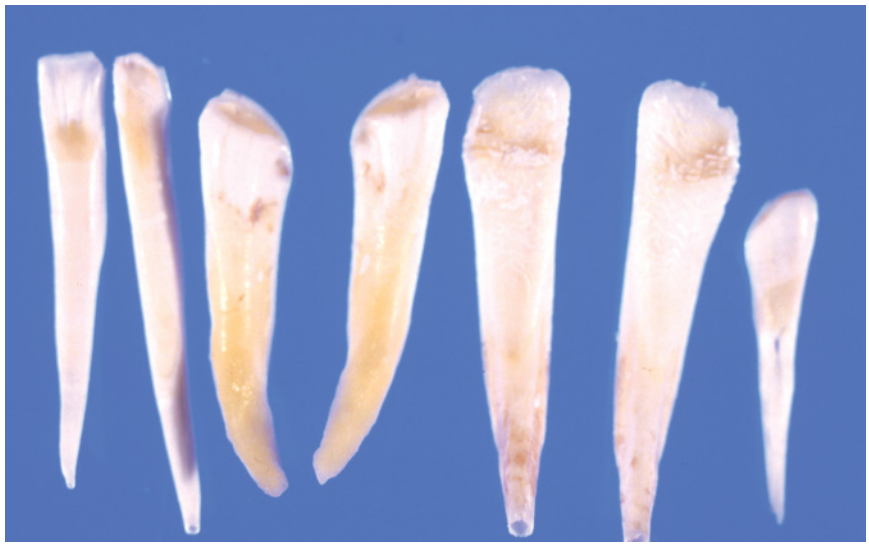

Figure 13.6. Incisor teeth of a mature llama.

The deciduous teeth of vicuñas are more elongated than those of the llama, but they are spatulate. The base of the incisor remains open. There is thin enamel on the lingual surface of the tooth, and the enamel of the labial surface extends halfway down the tooth.[77]

Incisors of the llama and guanaco are spatulate, with the greatest width ranging from one-fifth to one-fourth the greatest length (Figure 13.6). The root tapers rapidly to a closed base. Both labial and lingual surfaces of the crown are enameled, and on the lingual side the enamel extends one-third the length of the tooth. Deciduous and permanent incisors are similar in shape and structure but can be differentiated by size and amount of wear.

Incisors of alpacas seem to be intergrade between those of the vicuña and the llama (Figure 13.5). Enamel is present on both the labial and lingual surface of the tooth, and the tooth is narrow. Dental structure and DNA evidence demonstrates that alpacas have evolved from vicuñas and are now classified in the genus Vicugna. The adult alpaca incisor is nonspatulate but has a rectangular cross section rather than the square cross section of the vicuña. The apex of the incisor remains open, and there is good clinical evidence that the incisor continues to grow well into maturity. Alpaca deciduous incisors are spatulate shaped, but as the animal begins to grow and wear off the spatulate crown, the teeth assume more of the shape of an adult

alpaca incisor. As in the vicuña, alpaca incisors retain a chisel shape on the occlusal surface. If the occlusal surface is flattened and has a rectangular or square shape, it is an indication that such teeth have been trimmed to avoid having them protrude past the lips.

Examiners may experience some difficulty in telling the difference between the deciduous incisors of a one-and-a-half-year-old and a full-mouthed five-year-old.[116] The following characteristics may aid in differentiation. Deciduous incisors are slightly smaller and have a chalky color, in contrast with the permanent incisors that have a translucent sheen. Deciduous incisors taper at the gum line, whereas permanent teeth have parallel sides into the gum. Some clinicians identify vertical ridges on permanent alpaca incisors by running a fingernail across the labial surface of the crown. If permanent canines are present in a male, it is an indication that the animal is mature and the incisors are permanent.

Nondental indications of age include characteristics of the teats of a female. A five-year-old will likely have had one or two crias, and the teats will be from 1 to 2 cm long. A yearling is usually smaller than a five-year-old, but that variation is not clear in some instances.

More dental anomalies have been reported in the alpaca than in other camelids. Overgrowth of the incisor teeth is common, caused primarily by malocclusion. If the incisors occlude properly with the dental pad, the teeth will wear off and not grow out past the lips. (See Chapter 22 on congenital anomalies.)

The dental formulas for camelids are found in Table 13.1.

Canines

Permanent canine tooth eruption in males is associated with puberty and a surge of testosterone. The average time for the testosterone surge is between twenty-two and twenty-four months. A few precocial males may have testosterone activity at twelve to fifteen months of age. On the contrary, some don't reach hormone maturity until three years of age. Thus, if permanent incisors are erupted, it generally indicates that the male is more than two years old.

The canines of the camelids are adapted to intraspecies aggression, especially between breeding males. The deciduous canines are quite small and peg-shaped. They erupt rarely in females and in only about 5% of the males. The large canines of the mature male are formidable weapons (Figure 13.7). Females and geldings vary as to whether or not the permanent canines erupt. The teeth are sharp and are anchored into the jaw by a curved root.

Cheek Teeth

The cheek teeth are selenodont (cusps have crescentic outline). It is sometimes difficult on clinical examination to identify where a tooth begins and ends. The table surface of the tooth exhibits the sharp ridges and points typical of a herbivore that is accustomed to eating harsh grasses, shrubs, and forbs (Figure 13.8). The cheek teeth of the lower jaw are slightly more narrow than those of the upper, but this does not seem to foster the excessive development of sharp enamel points on the lingual surfaces of the lowers and labial surfaces of the uppers as it does in the equine. The

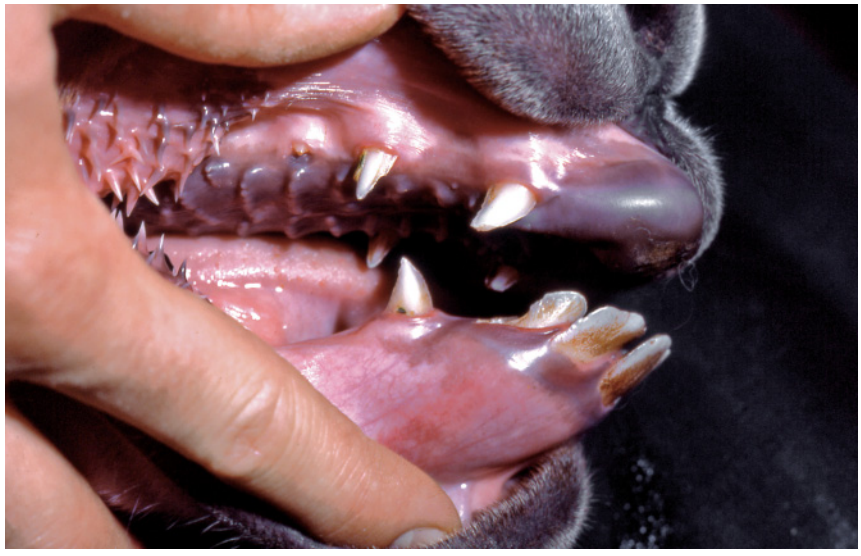

Figure 13.7. Male canine teeth.

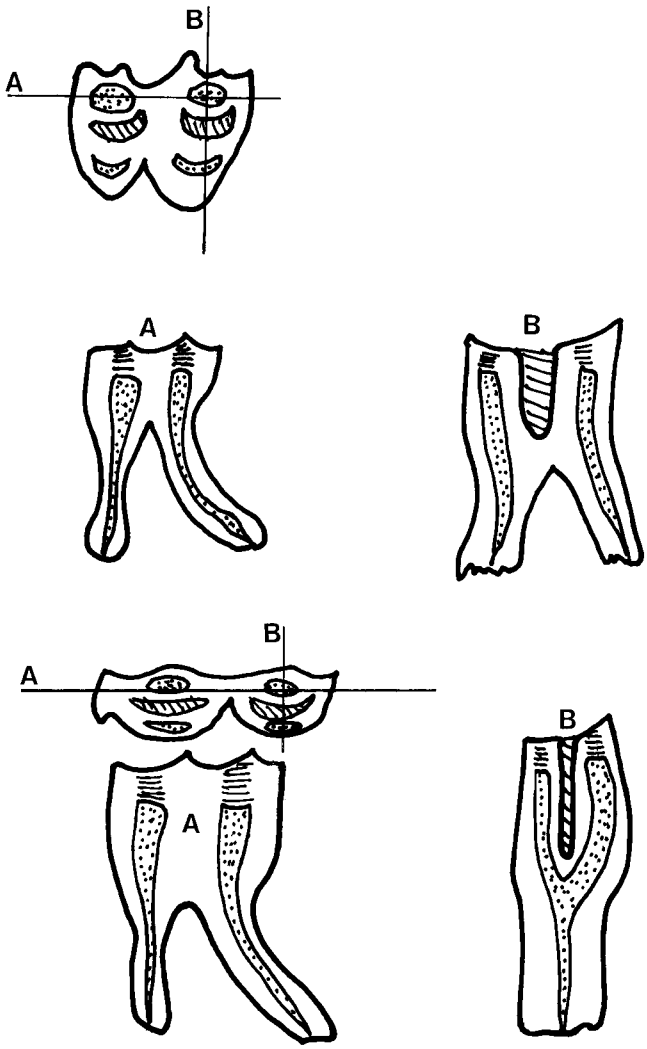

Figure 13.8. Diagrams of the upper and lower cheek teeth of an SAC. (A) Longitudinal direction, (B) cross direction.

Table 13.2. Eruption of SAC teeth.

Deciduous teeth	
All three incisors are usually present in full-term neonates.	
In premature neonates I_2 and I_3 may be delayed up to ninety and 107 days, respectively.	
Canines–only visible in 5% of males.	
Premolars–present at birth.	
Permanent teeth	
I_1	2–2.5 years
I_2	3–3.25 years
1_3	3.1–6 years
C	2–7 years, but average is 2.5–3.5 years
P_3	3.5–5 years
P_4	3.5–5 years
M_1	6–9 months
M_2	1.5–2 years
M_3	2.75–3.75 years

Table 13.3. Eruption of camel teeth.

Deciduous	
D I_1	2 weeks
D I_2	5 weeks
D I_3	6–12 weeks
Permanent	
I_1	4 years
I_2	5 years
I_3	6–7 years
C	7–8 years
PM_2	5–6 years
PM_3 (upper caniniform)	5–6 years
M 1/1	12–15 months
M 2/2	24–36 months
M 3/3	5–5.5 years

Dental formula for camels: 2(I 1/3, C 1/1, PM 3/2, M 3/3) = 34.

camelid chews with a lateral motion that seems to keep wear even. The roots of the cheek teeth are closed, and there is no continuous growth.

Aging

Determining age by dental eruption times and wear of the teeth has been studied in alpacas and llamas only in South America.[56] Eruption times may be controlled by a genetic trait, but wear depends on the harshness of feedstuffs and the degree of grinding necessary to masticate them. An approximation of age in the living animal can be derived by incisor eruption up to about five years (Table 13.2). After that, it is extremely difficult.[33] Figures 13.9 through 13.37 illustrate, by diagrams and photographs, the aging of llamas and alpacas. The eruption times of camel teeth are found in Table 13.3.

Salivary Glands[73]

There are three pairs of major salivary glands (parotid, mandibular, and sublingual). Four other glandular regions are less well defined but also contribute to salivary secretion (buccal, palatine, lingual, and labial).[11,45]

The salivary glands of camelids are similar in location, number, and histology to those of cattle, sheep, and goats. The parotid gland is much larger than the others and produces only a serous secretion,[69,77] while the other salivary glands are both serous and mucous producers. For a detailed discussion of the histologic and histochemical anatomy of the salivary and intestinal glands, see Luciano et al.[60,61] For a discussion of secretion, composition, and volume of salivary fluids, see Chapter 2.)

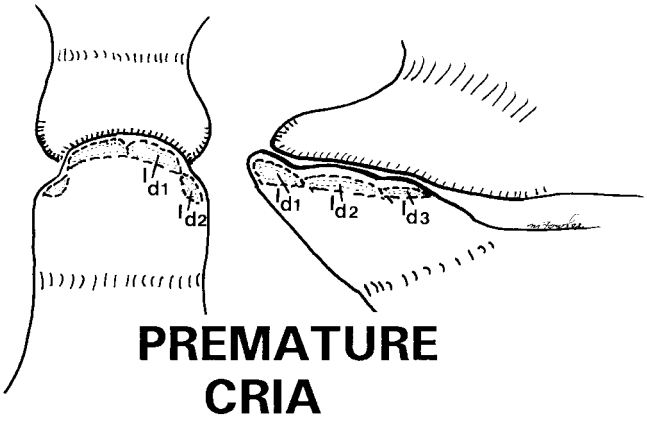

Figure 13.9. Diagram of the teeth of a premature cria.

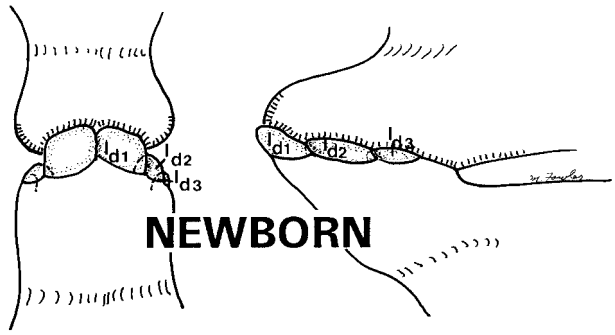

Figure 13.10. Diagram of the teeth of a newborn llama.

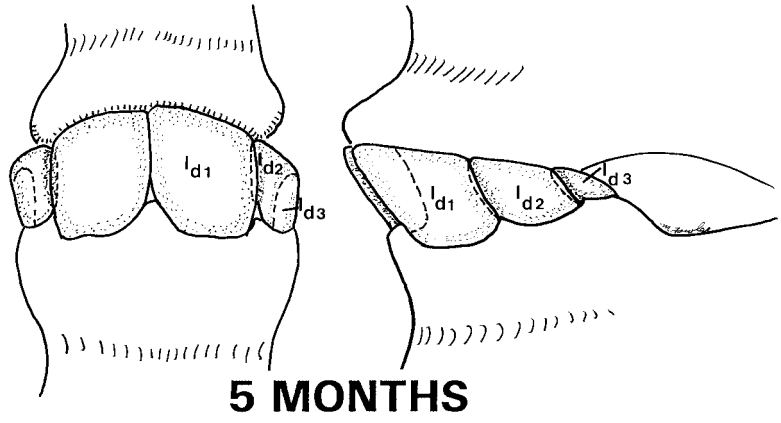

Figure 13.11. Diagram of the teeth of a five-month-old llama.

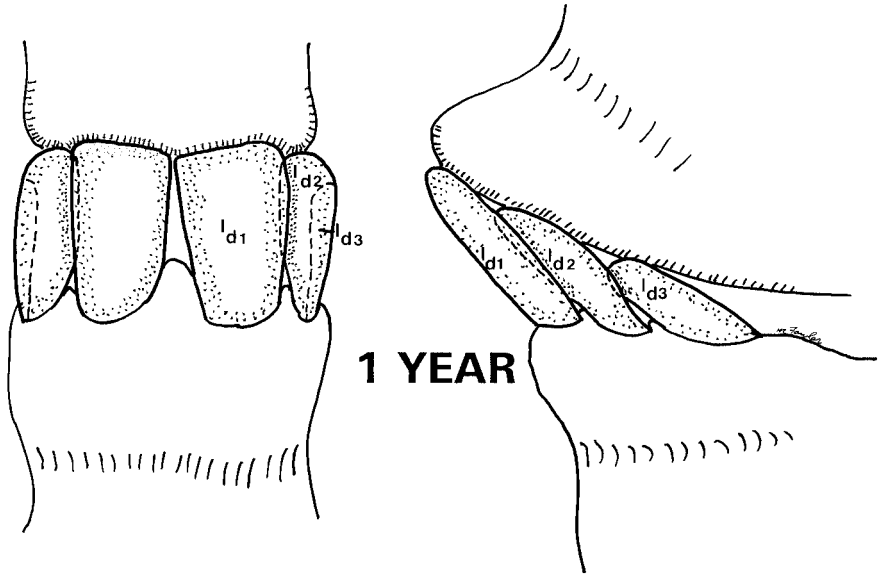

Figure 13.12. Diagram of the teeth of a one-year-old llama.

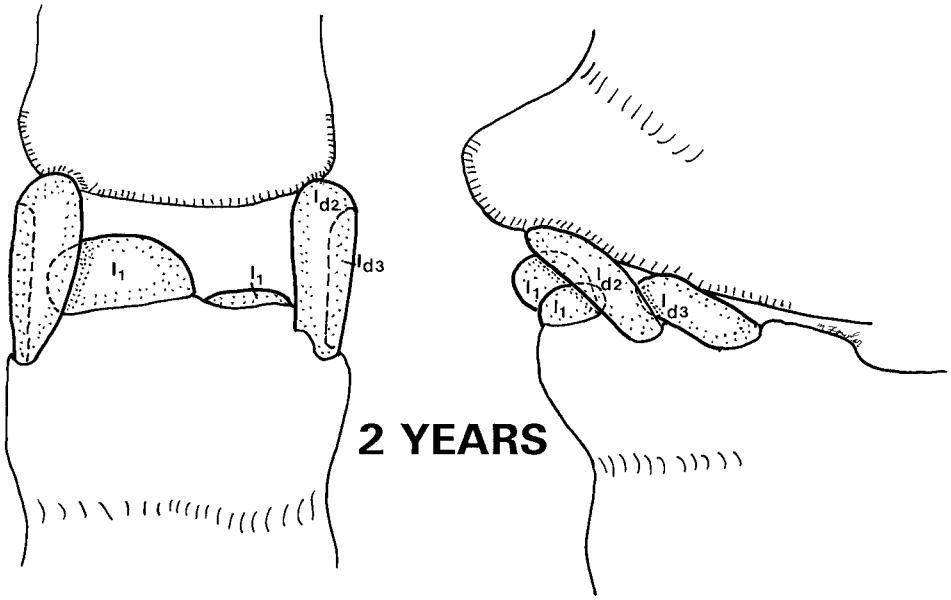

Figure 13.13. Diagram of the teeth of a two-year-old llama.

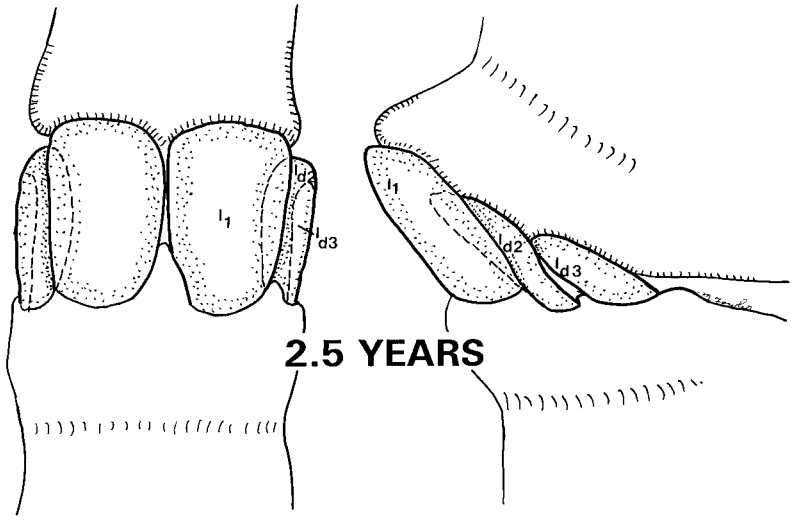

Figure 13.14. Diagram of the teeth of a two-and-a-half-year-old llama.

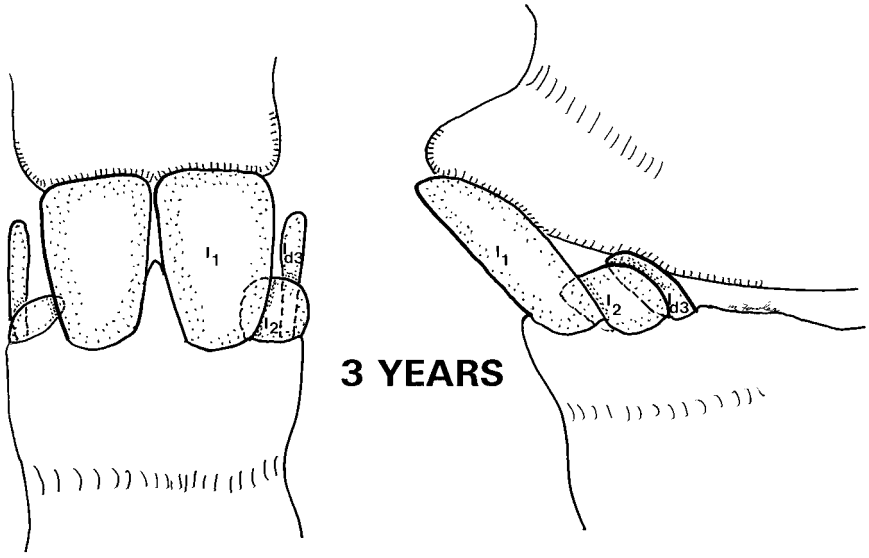

Figure 13.15. Diagram of the teeth of a three-year-old llama.

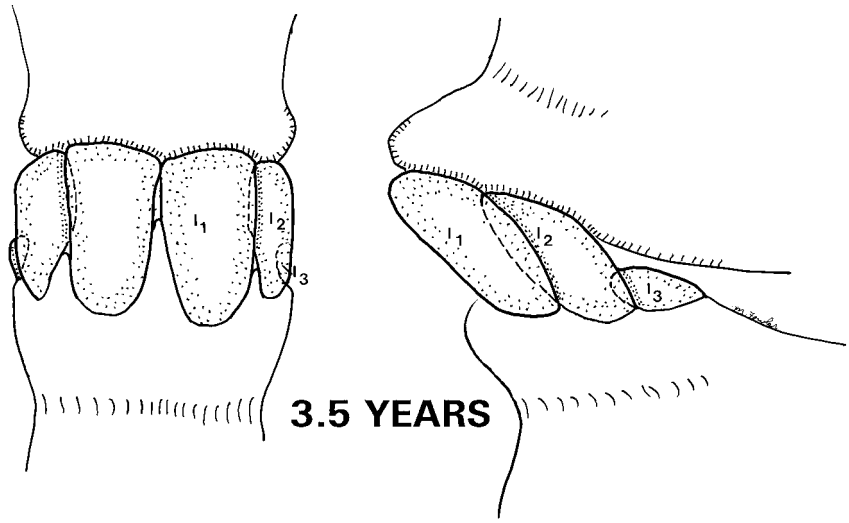

Figure 13.16. Diagram of the teeth of a three-and-a-half-year-old llama.

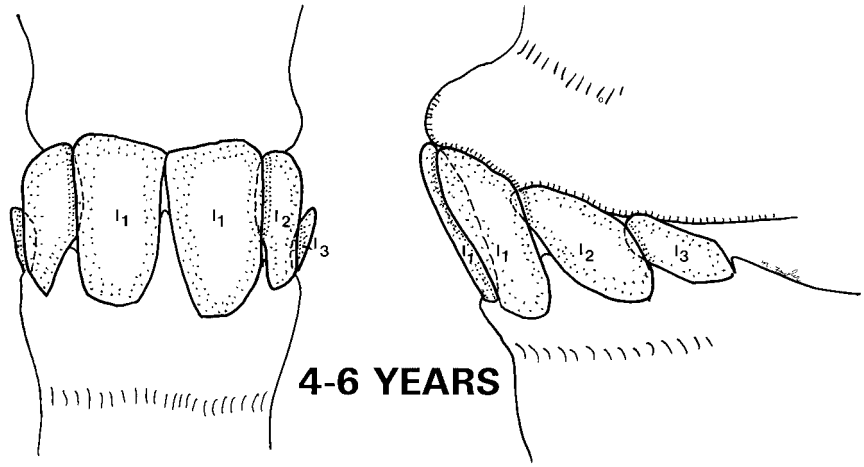

Figure 13.17. Diagram of the teeth of a four- to six-year-old llama.

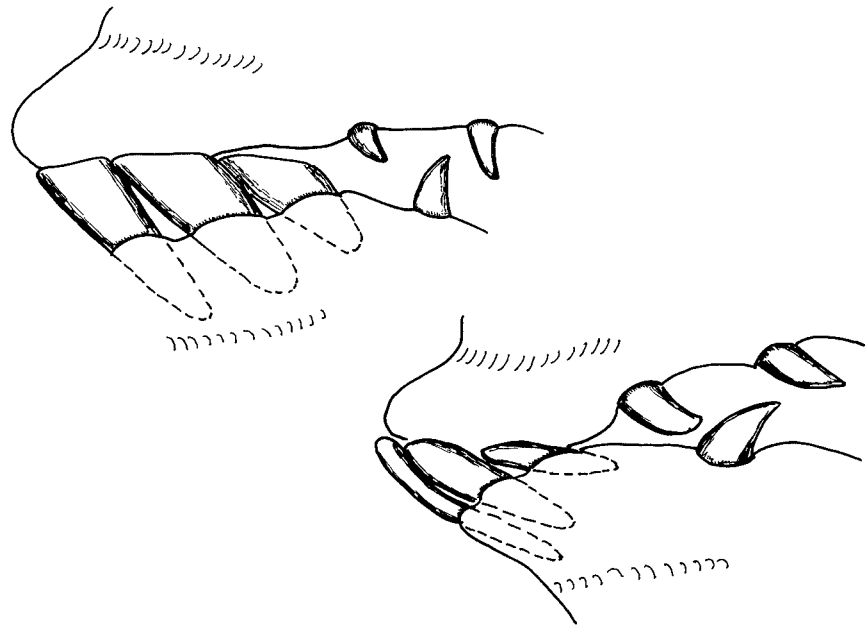

Figure 13.18. Diagram of the teeth of a seven-year-old llama (left) and an eleven-year-old (right).

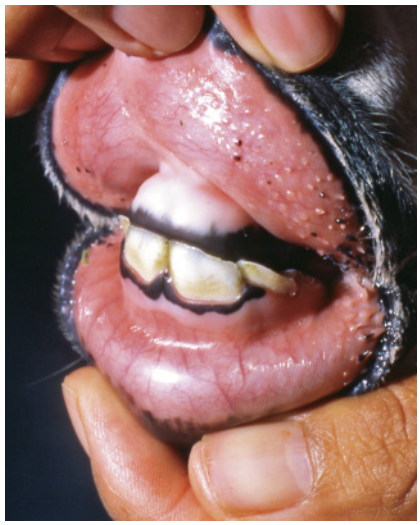

Figure 13.19. Llama, two months.

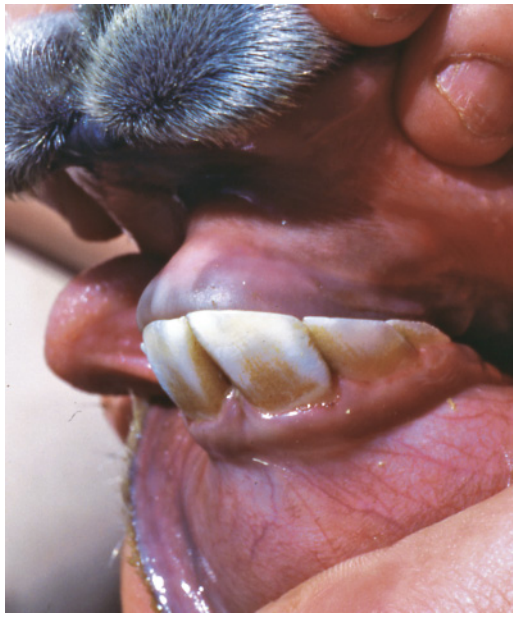

Figure 13.20. Llama, five months.

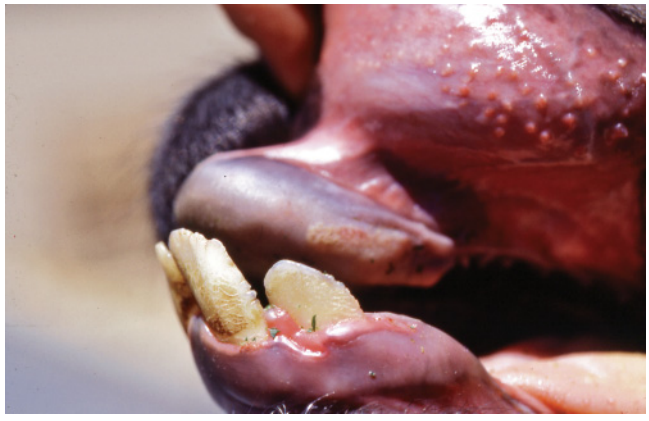

Figure 13.21. Llama, three years.

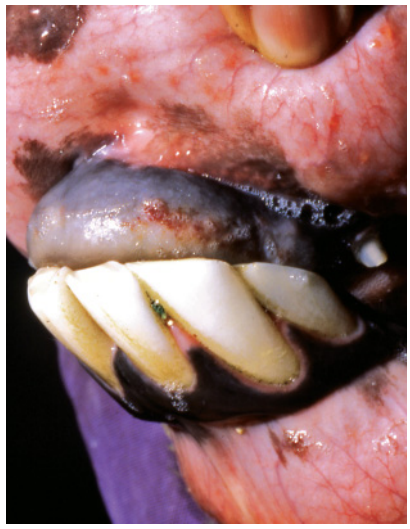

Figure 13.22. Llama, four years.

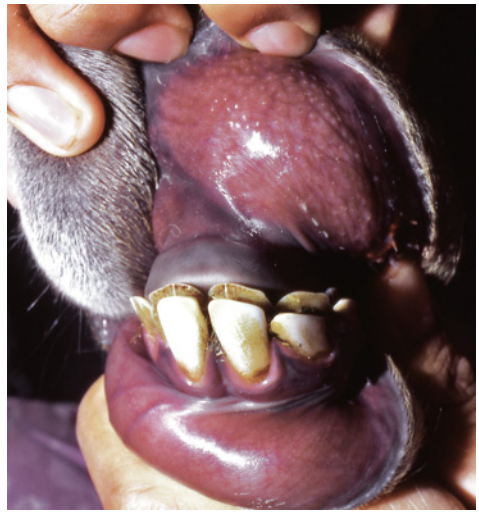

Figure 13.23. Llama, four years. Retained deciduous incisors.

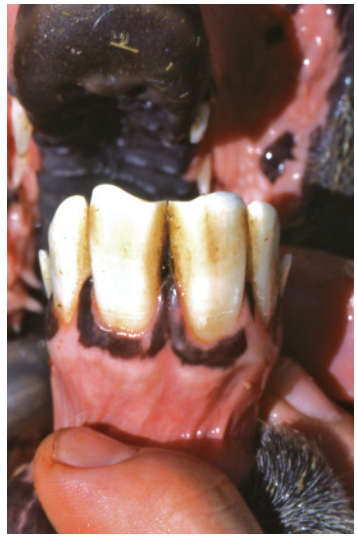

Figure 13.24. Llama male, five years.

Figure 13.25. Llama, seven years.

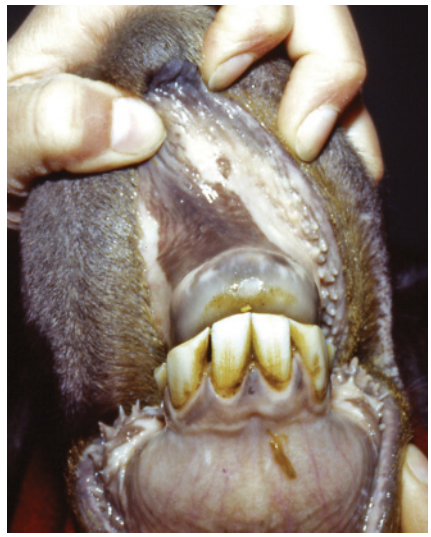

Figure 13.27. Llama, twelve years.

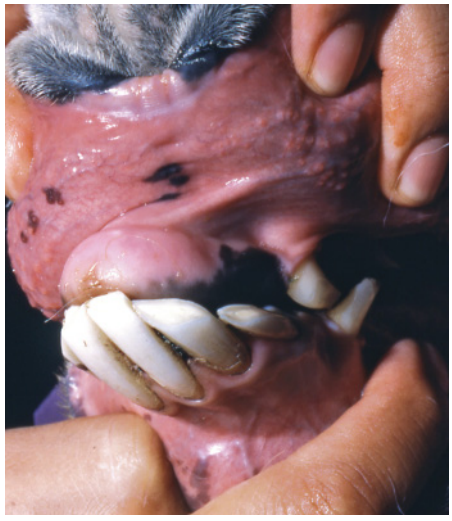

Figure 13.26. Llama, eleven years, abnormal wear.

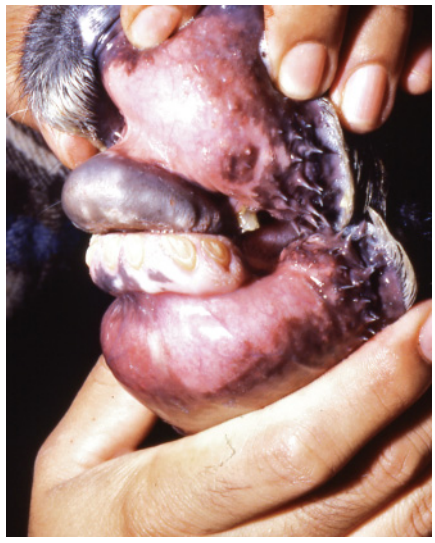

Figure 13.28. Llama, sixteen years, abnormal wear.

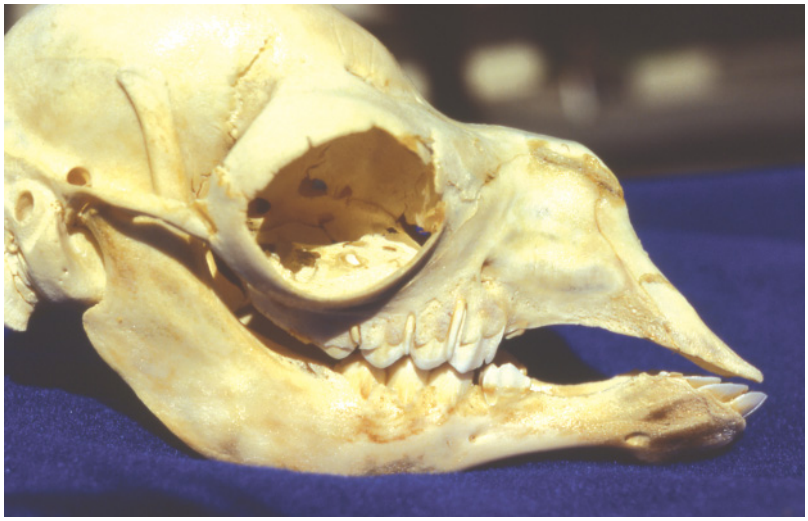

Figure 13.29. Lateral view of the skull of a neonate alpaca.

Figure 13.30. Dorsal view of the mandible and incisors of a neonate alpaca.

Figure 13.31. Alpaca, three months.

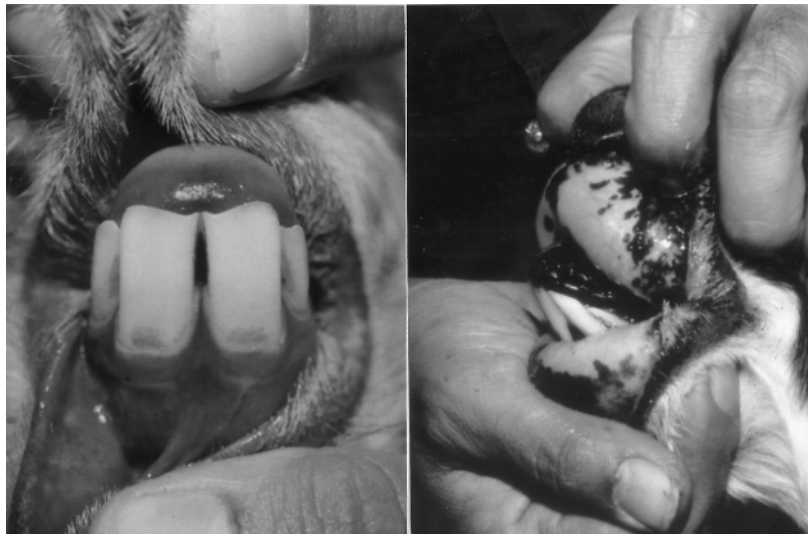

Figure 13.32. Alpaca, one year (left), one-and-a-half years (right).

Figure 13.33. Alpaca, two years (left) with retained deciduous incisors.

Figure 13.35. Alpaca, four years.

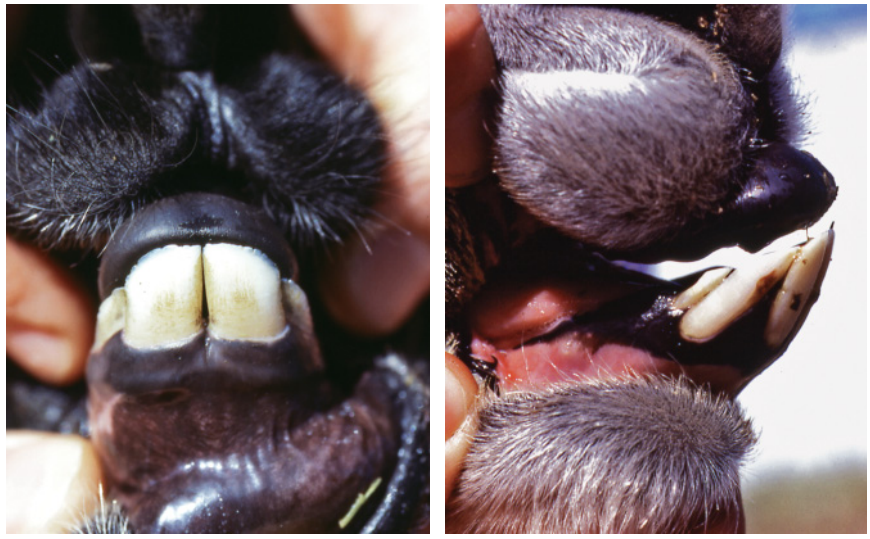

Figure 13.34. Alpaca, three years.

The parotid duct arises on the ventral rostral border of the parotid salivary gland. The duct traverses the side of the face mesial to the platysma muscle, approximately 1 to 1.5 cm dorsal to the facial vein (Figure 13.38). The parotid duct empties into the oral cavity through an orifice on a flattened papilla that is located 1 cm dorsal to the gingiva at the junction of upper cheek teeth 2 and 3 (Figure 13.39). The parotid duct of the camel empties opposite the fourth upper cheek tooth. In both groups of animals a line dropped from the rostral margin of the orbit gives the approximate location of the orifice. The duct may be cannulated with a 3.5 French catheter, but it is difficult to access it from the oral cavity because the mouth cannot be opened widely enough. The mandibular duct empties at the sublingual caruncle, and the numerous ducts of the sublingual gland open alongside the tongue.

Salivary calculi (sialoliths) are composed of calcium carbonate and are common in horses but rare in camelids. Salivary calculi may form in the gland and be carried through the duct system. If the calculus is tiny it may pass into the mouth and cause no problem. A calculus may lodge in a narrow segment of the duct and continue to enlarge. With total obstruction, the salivary gland may swell because of the backed-up saliva. The duct may dilate as well, causing a cystic formation called a ranula. Ultimately the affected salivary gland will atrophy.

GASTROINTESTINAL TRACT[106,107]

The gastrointestinal tracts of all the *camelidae* are basically the same except for size. A diagrammatic representation of the digestive tract is illustrated in Figure 13.40.

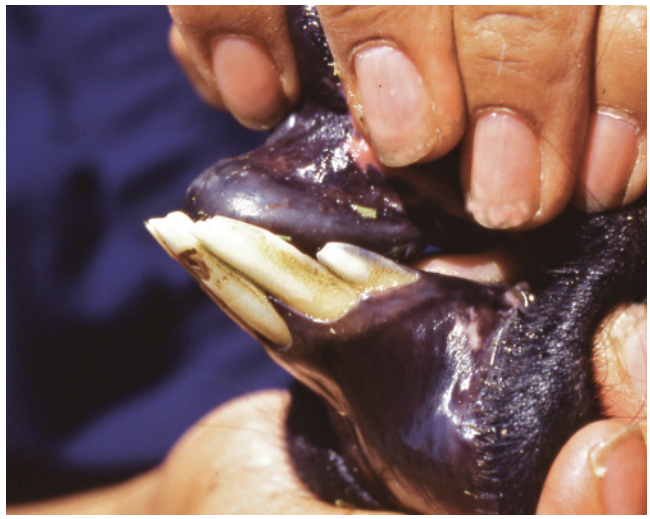

Figure 13.36. Alpaca, five-year-old incisors.

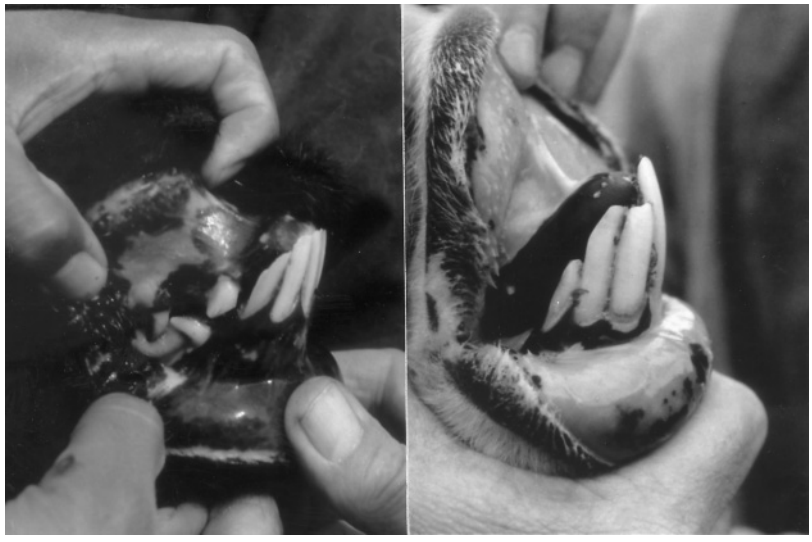

Figure 13.37. Alpaca, five-year-old male (left) and seven-year-old (right).

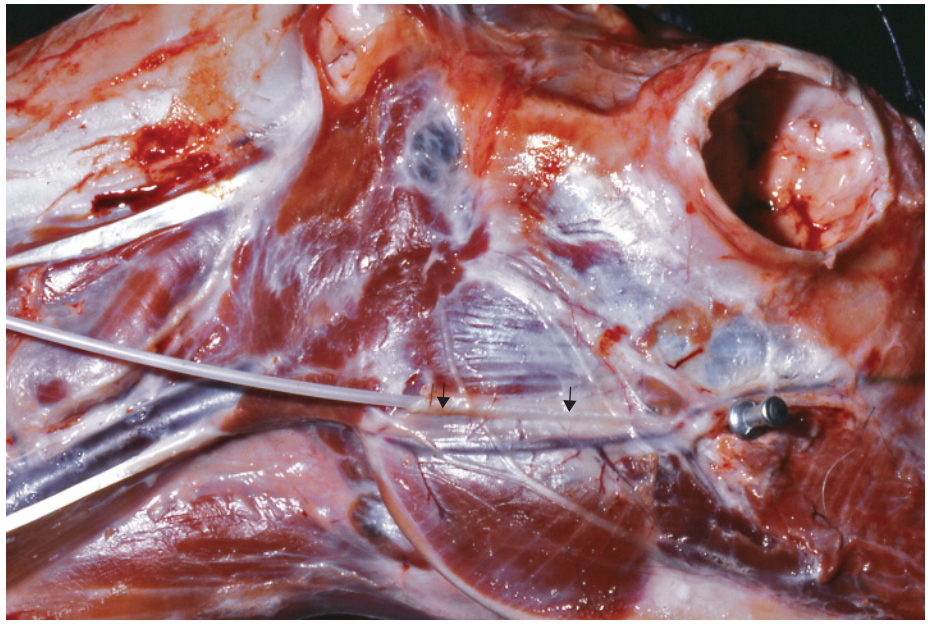

Figure 13.38. Course of the parotid salivary gland duct.

Figure 13.39. Parotid duct opening opposite upper cheek tooth 3.

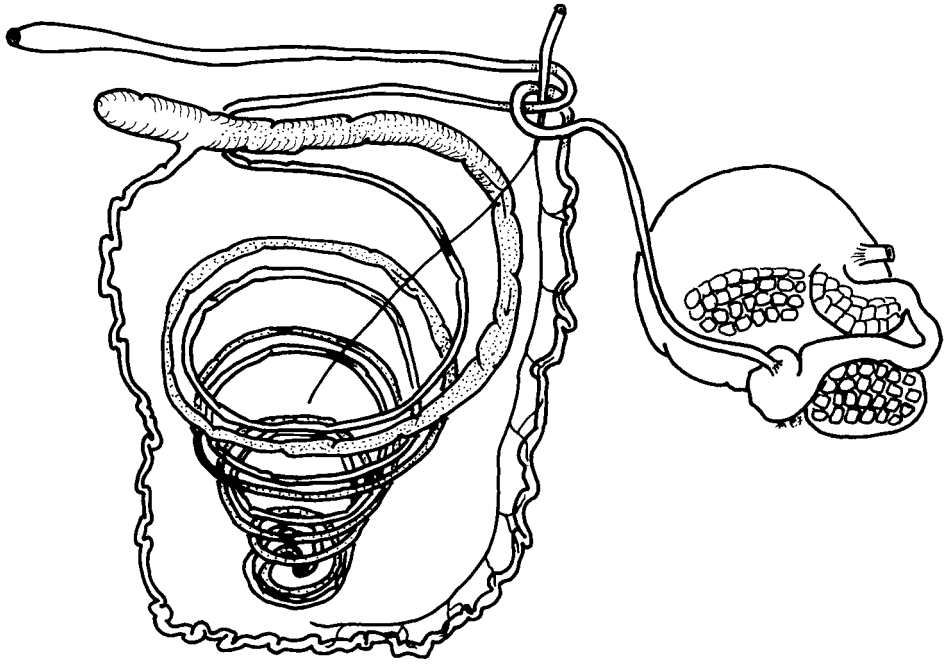

Figure 13.40. Diagram of the gastrointestinal tract of a camelid.

Stomach[28,37,61,62,80,97,100,114,119]

No uniform nomenclature for the stomach of camelids has been adopted. Various anatomists have used different terminology.[84] The author has chosen to use the terminology of Vallenas et al.[104] The anatomy of the forestomach of camelids differs significantly from that of ruminants (Tables 13.3 to 13.6).[8,9,19,31,37,53,56,57,76,99,105] The three compartments of the camelid stomach (C-1, C-2, C-3) contrast with the four-compartmented stomach of the ruminant. The compartments of the camelid stomach are not analogous, morphologically, to the rumen, reticulum, omasum, and abomasum.[30,102,107] There are no papillae in C-1 and C-2, but all three compartments have glandular areas. Motility patterns are markedly different in camelids, and the timing of rumination and eructation during the motility cycle also varies from that of the ruminant stomach.[25,40,99]

The evolution of the various regions of the stomach can be traced from the primordial simple stomach by the structure of the tunica muscularis (Figure 13.41).[51]

Basically, the anatomy of the stomach and intestines is the same in all camelids (Figures 13.42 to 13.44). Similar to the ruminant, the camelid neonate has a poorly developed first compartment and a large true stomach (Figure 13.43).

Table 13.4. Comparison of ruminant and camelid stomachs.

Ruminant	*Camelid*
4 compartments	3 compartments
Esophagus enters between rumen and reticulum	Esophagus enters C-1 only
Pillars pronounced, horizontal	Pillars minimal, horizontal
Glands in abomasum only	All compartments glandular
Esophageal groove double lipped	Esophageal groove single lipped
Rumen papillated	C-1 nonpapillated
Motility pattern 2–4/min	Motility pattern 3–5/min
Rumen—epithelium keratinized, stratified squamous	C-1—epithelium nonkeratinized, squamous
Abomasum—entirely covered with enzyme and acid-secreting epithelium	C-3—only distal one-fifth covered with enzyme and acid-secreting epithelium

Source: Bohlken 1960.

C-1 occupies the greater part of the left abdomen (Figure 13.43). A cranial and caudal sac are weakly divided by a horizontal pillar. The esophagus enters C-1 on its craniodorsal midline. C-2 is situated on the right craniodorsal surface of C-1. The tubular C-3 arises on the cranial mesial aspect of C-2. It curves to the right, caudal and ventral to the liver, and lies on the right surface of C-1 in the right midventral abdomen.

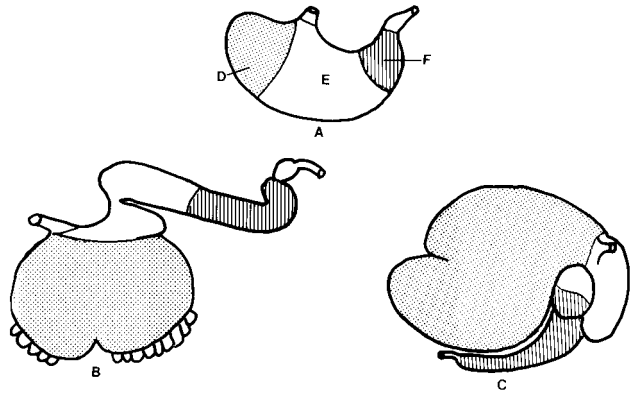

Figure 13.41. Comparative origins of different regions of the stomach. (A) Simple stomach, (B) camelid stomach, (C) ruminant stomach, (D) fundal region, (E) body region, (F) pyloric region.

Table 13.5. Information about llama stomach compartments.

Compartment	*pH*	*Body weight of contents (%)*	*Retention time (hr)*			*Volume of stomach ingesta (%)*	*Function*
			Liquid	*Particles*			
				<0.2 cm	*>0.2 cm*		
C-1	6.4–7.0			20.3	>40	83	Fermentation, absorption of water, VFA and other solutes
C-2	6.4–7.0	10–15	9.6	20.3		6	Fermentation, absorption of water, VFA and other solutes
C-3	6.5 cranial <2–3 caudal	1–2	5.7	9		11	Absorption of water and solutes Proximal 4/5, digestive Distal 1/5, enzymes, acid

Table 13.6. Compartments of the ruminant stomach.

Compartment	*Epithelial covering*	*Volume of ingesta (%)*	*pH*	*Function*
Rumen	Keratinized, stratified squamous, papillated	Cow—64 Sheep—69 Lamb—31	5.8–7.0	Fermentation chamber, major contractions 1–2/min
Reticulum	Stratified squamous		5.8–7.0	Fermentation chamber
Omasum	Stratified squamous	Cow—25 Sheep—8 Lamb—8	5.8–7.0	Reduce the particulate matter of the content of the omasum to a finer state
Abomasum	Glandular	Cow—11 Sheep—23 Lamb—61	3.0	Secretion of digestive juices that continue the digestive process

The nonglandular mucosal surface of C-1 and C-2 is not papillated and is comprised of unkeratinized, stratified squamous epithelium.[21,60,61] The ventral surfaces of both the cranial and caudal sacs of C-1 contain glandular saccules (Figure 13.45). The epithelium varies slightly from the orifice to the depths of the saccule, but it is generally a mucinous glandular epithelium. In the camel, glandular epithelium is found only on the bottom and lower walls of the saccules.

In earlier studies, it was reported that this glandular epithelium was the source of sodium bicarbonate,[21] but later studies, using a Pavlov pouch, did not confirm this.[26,75,79] Current studies indicate that the glandular epithelium also provides for rapid absorption of water and solutes.[13,63] For more information on the physiology of the stomach, see Chapter 2 and the references.[22,24,25,29,46,62,105]

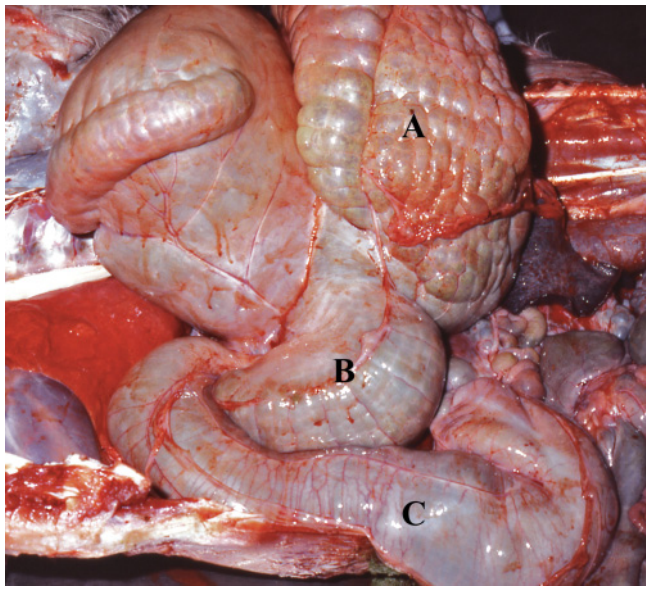

Figure 13.42. Stomach of a llama. (A) C-1, (B) C-2, (C) C-3.

The mucosal surface of C-2 is glandular except for a small area on the lesser curvature that constitutes the esophageal or ventricular groove (Figure 13.46). The glandular area is subdivided by a series of intersecting crests that produce a retiform pattern and is covered by a papillated glandular mucosa. The retiform pattern is not analogous to the pattern seen in the reticulum of ruminants.[102,108] The primary crest margins are covered by stratified squamous epithelium extending from the ventricular groove, but the secondary crests are covered with glandular mucosa.[102,105] The depressions of C-2 are not deep and do not evert during the contraction phase of the motility cycle of the stomach.

The mucosa of C-3 is entirely glandular (Figure 13.47). There are three pattern areas and two types of mucosa. On the lesser curvature of the first one-fifth is a retiform pattern with short crests and shallow depressions. On the greater curvature of the first one-fifth are nonpermanent folds. The mucosa of the middle three-fifths consists of permanent longitudinal pleats (approximately fifty). The epithelium of the proximal four-fifths is a mucinous glandular tissue, similar to the glandular tissue of C-1 and C-2.

The terminal one-fifth of C-3 contains the true gastric glands. The mucosa is reddish-brown, in contrast to the lighter pink of the proximal four-fifths. The wall of this area is thickened and the mucosal surface smooth. Digestive enzymes and acid are secreted by these glands. The pH in the cranial segment of C-3 is

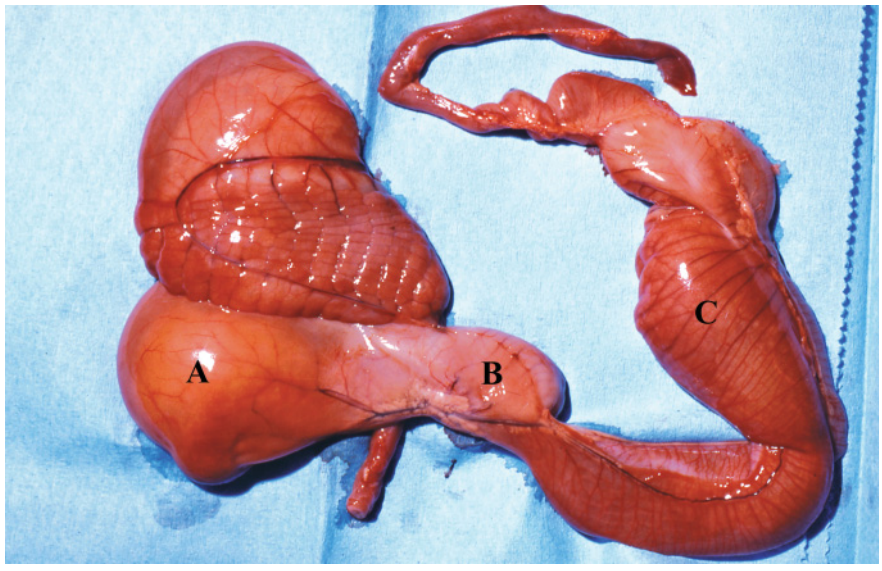

Figure 13.43. Stomach of a neonate llama. (A) C-1, (B) C-2, (C) C-3.

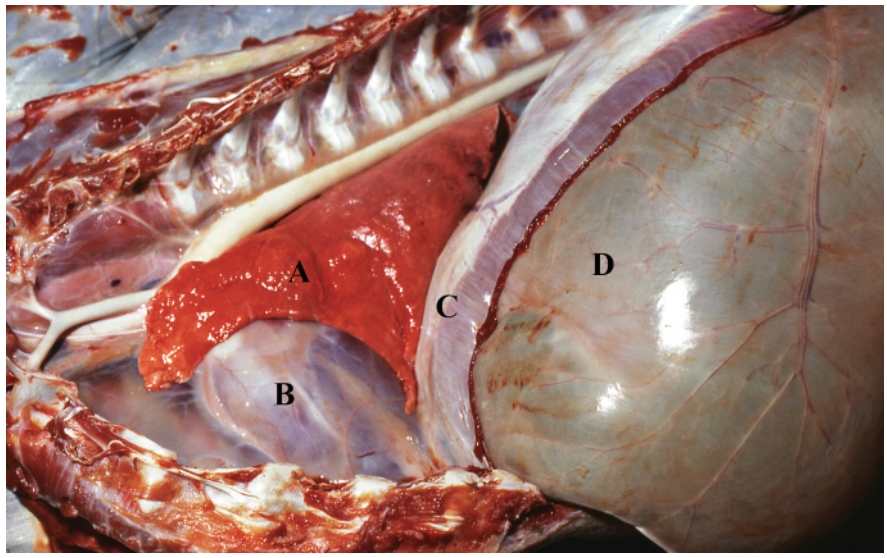

Figure 13.44. Relation of stomach to other organs. (A) Lung, (B) heart, (C) diaphragm, (D) C-1.

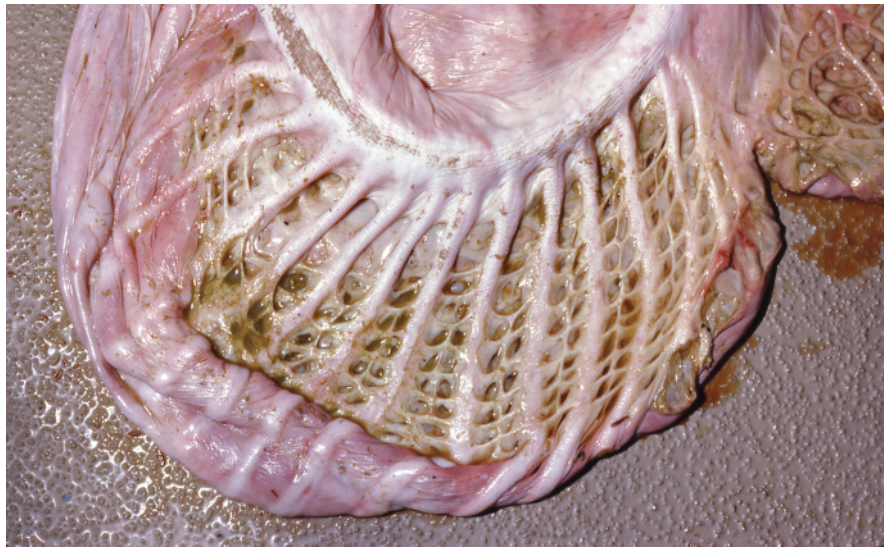

Figure 13.45. Glandular saccules of C-1.

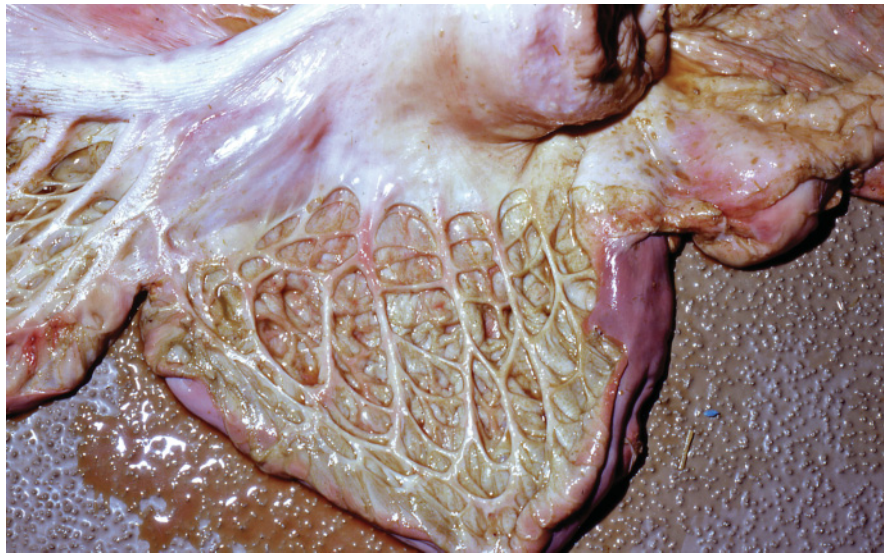

Figure 13.46. Glandular divisions of C-2.

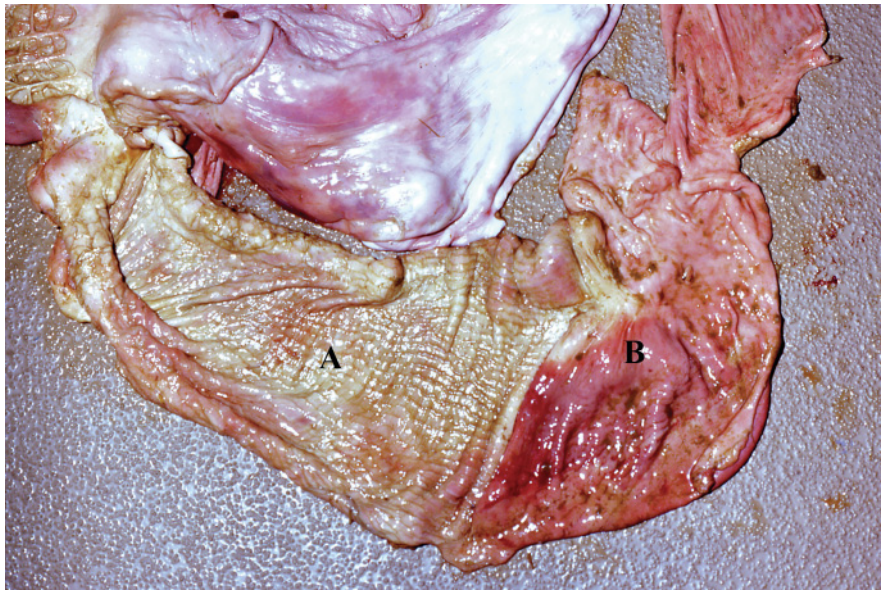

Figure 13.47. C-3 of stomach: (A) general glandular mucosa, (B) true stomach.

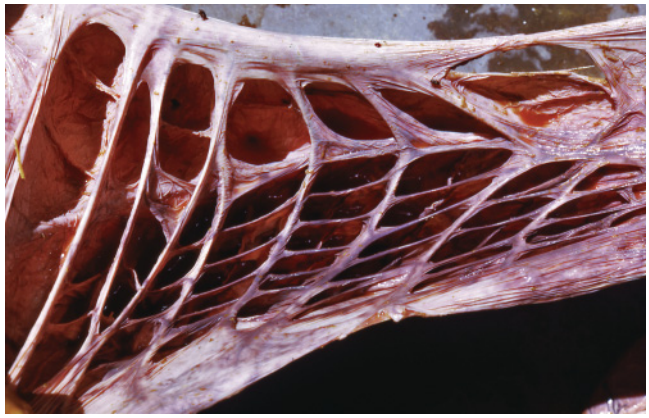

Figure 13.48. Camel stomach, C-1.

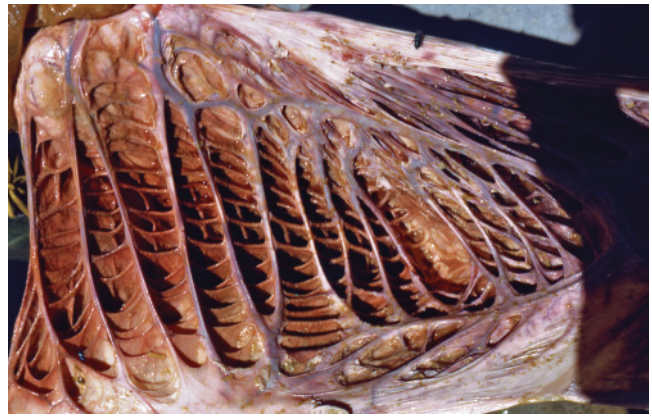

Figure 13.49. Camel stomach, C-2.

6.5, which decreases to less than 2 in the terminal fifth.[29] In Old World camels, the mucosa of the terminal fifth of C-3 may have coarse rugae.[105]

The camel stomach is basically the same as that of the SAC except for size (Figures 13.48 to 13.50).

The basic contraction cycle of the stomach of the llama and guanaco was established by Vallenas.[99,103] A contraction cycle is initiated by a single rapid contraction in C-2, followed quickly by a contraction of the caudal sac of C-1. The direction of the contraction is from caudad to cranial. Next, the cranial sac of C-1 contracts in the reverse direction, cranial to caudad.

In the resting llama, the sequence of contractions of C-1 is repeated six to eight times before another contraction of C-2 occurs. The cycle is from one contraction of C-2 to the next (of C-2), and the average length of a cycle is 1.8 ± 2 minutes.

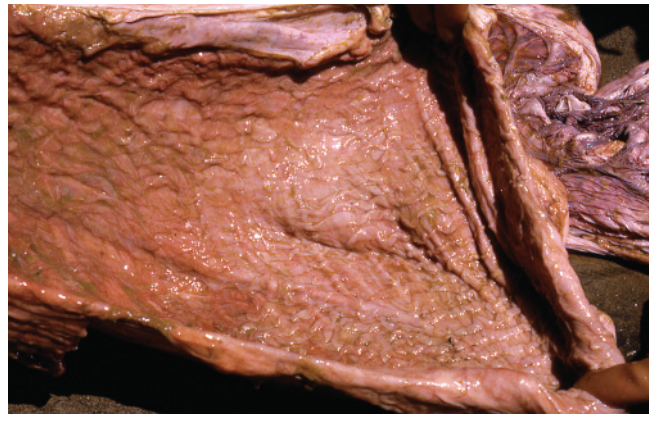

Figure 13.50. Camel stomach, C-3.

To the clinician, this equates to three or four audible contractions per minute in a resting animal and four to five contractions during feeding or the immediate postfeeding state. The amplitude of the contractions also increases during feeding.

The contents of the glandular saccules of the caudal sac are extruded just prior to the contraction of the caudal sac of C-1. The contents of the caudal sac and dorsal area of the cranial sac of C-1 are quite dry (see Figure 13.51). The more fluid and smaller particulate matter is found in the ventral cranial sac. The motility pattern tends to dump ingesta back and forth between the cranial and caudal sacs. This is a highly efficient mechanism for mixing the ingesta and enhancing the fermentation process. The ingesta in C-3 is rather dry, a result of the water absorption that takes place in the cranial four-fifths.

Volatile fatty acids (VFA) are rapidly absorbed from C-1, perhaps at two to three times the rate of absorption from the rumen of sheep and goats.[27,28,29,101] Similar absorption of VFA occurs from the proximal four-fifths of C-3.

C-1 microflora and fauna are described by Harmeyer and Hill[47] and Sillau et al.[89] and in Chapter 2.

Camelids are capable of adapting to low-protein diets by recycling the urea produced as an end product of nitrogen metabolism.[48,49] Urea is used by stomach bacteria to form their own protein. The bacteria are subsequently digested and the protein absorbed further along the intestinal tract and utilized by the camelid. The presence of VFA enhances the efficiency of urea recycling; thus, it is desirable for the camelid to consume a source of carbohydrate, if at all possible, when on a low-protein diet. When food is withheld from a llama for forty-eight hours, the plasma-urea (BUN) concentration will increase.

Figure 13.51. Homogeneous ingesta, characteristic of C-1.

The esophageal groove of the camelid is not as well developed as in ruminants.[99] There is only a single lip as contrasted with two in the ruminant. The functional groove extends from the cardia to and through the lesser curvature of C-2. See Vallenas[98] and Chapter 21 for a discussion of chemical stimulation and closure of the esophageal groove.

Transit times for ingesta have been studied,[10] but no conclusions have been reached. A study on eructated gas expulsion in alpacas was conducted in 1968.[22,86]

Omentum

The greater omentum is relatively smaller in the camelid than in cattle and sheep. In the adult ruminant, the greater omentum usually conceals all the intestines in a sling except for the descending duodenum. Again, in the ruminant, the omentum does not support the weight of the intestines but merely rests on the abdominal floor.

In the camelid there is no omental sling. The greater omentum is attached along both the lesser and greater curvatures of C-2 and C-3 and along the right surface of C-1.[55] The epiploic foramen enters the sac formed by the greater omentum ventral to the liver, near the entrance of the post cava into the liver. Epiploic herniation of the jejunum has caused death in a llama.

Intestine

Small Intestine[118,119]

There is a dilated ampulla at the entrance of the duodenum. The duodenum courses dorsally on the right side of C-1 to the dorsum of the abdominal cavity just caudad to the liver. The jejunum is folded around the root of the mesentery, in the right caudal abdomen. The ileum begins ventrally and courses mesially and dorsally to enter the large intestine at the cecocolic orifice.

Large Intestine[15,105]

The cecum lies approximately on the midline and is directed caudally toward the pelvic inlet, or it may curve ventrally and laterally to the left. The camelid colon is similar to that of the bovine. The ascending colon begins as a proximal loop that courses cranially and ventrally to enter the spiral loop (Figures 13.52, 13.53). The most proximal loop of the ascending colon is loosely attached by a mesentery to the more compact spiral colon. There are five and a half centripetal coils (including the proximal loop) and four and a half centrifugal coils. There are only two coils in bovines and

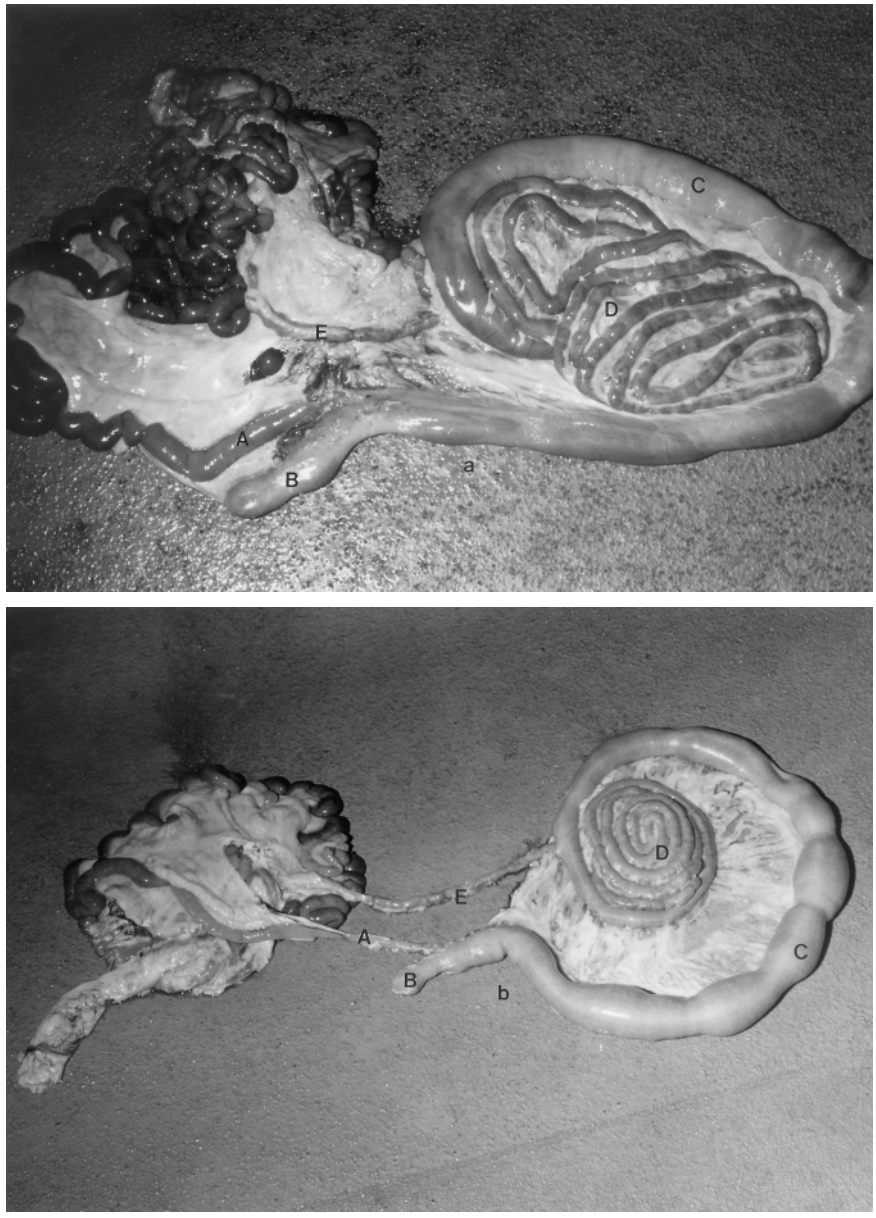

Figure 13.52. Intestines of a llama. (A) Ileum, (B) cecum, (C) proximal loop of spiral colon, (D) spiral colon, (E) transverse colon.

three in ovines. The centrifugal coils are buried within the spiral, deep to the centripetal coils. The spiral colon lies on the midventral abdominal wall and is likely to be the first organ encountered on a ventral midline laparotomy.

The colon narrows from a 5-cm diameter at its beginning to 2 cm within the first centripetal coil of the spiral loop. The spiral colon is the primary site of fecalith impaction in camelids.

The distal loop of the ascending colon is juxtaposed to the proximal loop. The transverse colon passes from right to left cranial to the mesenteric artery and continues on as the descending colon to the rectum and anus.

Length of Intestinal Segments

The measurements of the intestines of a 140-kg llama were as follows: The small intestine was 11.5 to 12 m, of which the duodenum was less than 1 m, the jejunum 9.5 to 10 m, and the ileum 1 m in length. The large intestine was 7.5 m long, of which the cecum was 10 cm long and 5 cm in diameter, the large colon was 1.5 m long, diminishing in diameter from 5 to 2.5 cm in its course, and the small colon was 6 m long and approximately 2.5 cm in diameter.

The camel small intestine is approximately 40 m (131 ft) long, and the large intestine 19.5 m (64 ft).[24]

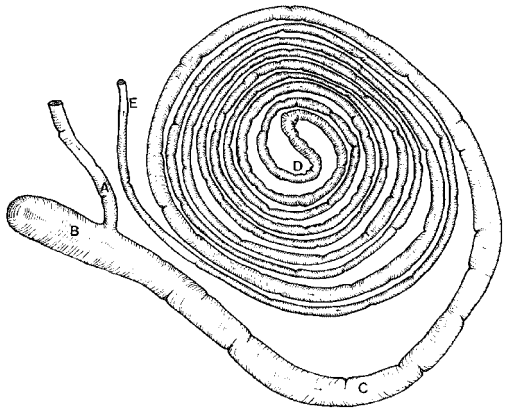

Figure 13.53. Diagram of the spiral colon of a llama. (A) Ileum, (B) cecum, (C) proximal loop of spiral colon, (D) spiral colon, (E) transverse colon.

Pellet Formation

Camelids void feces in pellet form.[44] These begin to form in the proximal spiral colon. Desiccation of the pellet continues throughout the colon. Old World camels have the capacity to pass totally desiccated feces when water intake is restricted. In the Arabian desert during the summer, freshly passed feces can be used for fuel. The fecal pellets of SACs are also used for fuel, but they must be allowed to dry further.

The llama fecal pellet varies with the size of the animal, from 7 × 12 to 20 × 30 mm (Figure 13.54A). There is generally a sharp teat on one end of the pellet. Some individuals pass teardrop-shaped pellets (7 × 12 × 20 mm). The pellets of a healthy llama may be voided as separate pellets or in a compressed cylin-

Figure 13.54A. Fecal pellets from llamas.

Figure 13.54B. Fecal pellets from a camel.

dric mass, which easily breaks apart into individual pellets. The color of the stool varies with the diet but is generally greenish-brown, blackening after voiding.

The stool of the dromedary camel is passed in irregularly shaped pellets. A medium-sized female may void pellets that range in size from 15 × 15 × 15 to 20 × 25 × 25 mm. A large male may pass pellets as large as 25 × 30 × 35 mm (Figure 13.54B).

SACs generally use communal dung piles (Figure 13.55). In inclement weather, a dung pile may be begun in a barn and the use of it continued into the summer. Numerous methods have been tried by owners to change the location of a dung heap. Old World camels do not use a dung heap.

More details of the anatomy of the digestive system, including vascular supply, may be found in Langer,[57] Vallenas,[96] Cummings et al.,[19] and Galotta and Galotta.[37]

Figure 13.55. Dung pile used by llamas for urination and defecation.

Liver[2,5,110–112]

The liver is located entirely on the right side (Figure 13.56).[14] The dorsocaudal border extends caudad to the cartilage of the last rib. The liver covers C-2 and C-3 of the stomach, although there is a notch on the craniоventral border where a small portion of C-3 is visible. The caudal border of the liver is strikingly fimbriated (Figure 13.57).

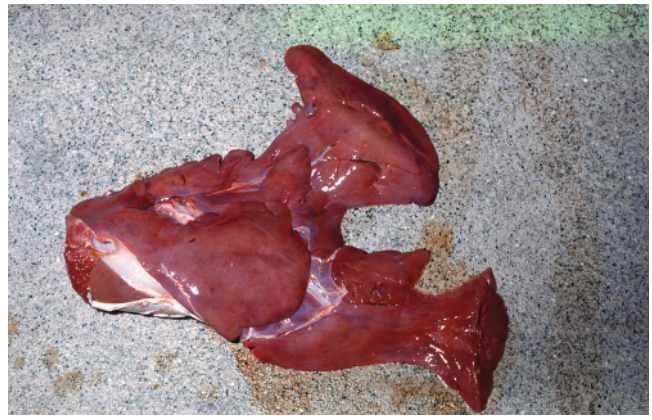

Figure 13.56. Liver of a llama.

Bile Duct

The collecting system ends in the major bile duct, 4 mm in diameter, at the caudal aspect of the liver. The duct is 3 to 4 cm long and penetrates the antimesenteric surface of a loop of duodenum that courses toward the liver. The opening into the duodenum is 16 to 20 cm

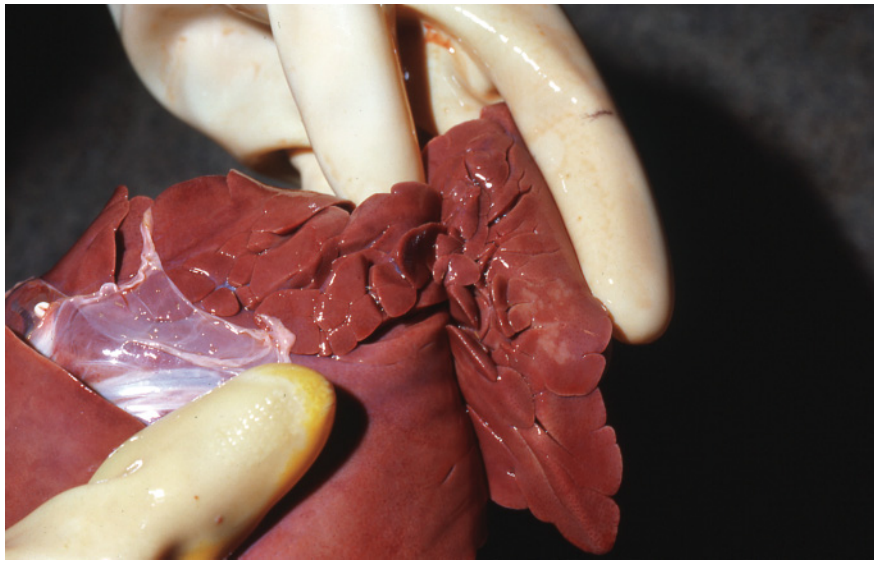

Figure 13.57. Fimbriated caudal border of an SAC liver.

from the pylorus. The gallbladder is absent except in rare instances.

DIGESTIVE DISORDERS

Signs Associated with Digestive Disorders

None of the signs suggesting disease of the digestive system are definitive. Nondigestive system diseases may produce similar signs. An overview of these signs and their multiple causes helps establish a tentative diagnosis and aid in formulating a diagnostic plan.

Anorexia/Inappetence

Anorexia is the lack or loss of appetite for food. Other words that are used, somewhat synonymously, to describe this condition include "inanition" (the physical condition resulting from the complete lack of food) and "inappetence" (lack of desire or appetite for food). It is difficult at times to determine whether there is a genuine lack of desire to eat or if there is an inability to eat because of weakness or an organic disorder that prevents eating. An attempt should be made to determine whether or not anorexia stems from an internal disorder or from external factors.

Internally caused factors include electrolyte imbalance, dental pain, stomatitis, temporomandibular arthritis, oral or pharyngeal abscesses, glossitis, colic, gastritis, central nervous system (CNS) injury, anemia, septicemia, and pyrexia. External factors that may contribute to anorexia include psychologic ostracism and unsuitable feed (not appropriate to the individual, moldy, too stemmy, or containing foreign material).

Herbivores normally consume feeds with low levels of sodium and an excess of potassium. The bovine kidney has a high concentration of renin to aid in the conservation of sodium. Potassium is poorly conserved because it is usually amply provided in the diet.

Anorexia results in low potassium intake and, subsequently, development of hypokalemia, especially if the llama continues to drink or is given parenteral fluids lacking in potassium. Hypokalemia may also result from loss of ingesta via vomition or diarrhea or from prolonged diuretic or corticosteroid therapy. Hypokalemia may contribute to or result from an alkalosis.

Clinical signs of hypokalemia include muscular weakness, depression, and altered cardiac function. These are nonspecific signs often seen in an ailing llama, and a case must be carefully evaluated to determine the cause. Serum chemistry analysis may be necessary.

Prehension Difficulties

This sign may be closely related to anorexia, since anything that interferes with a llama's ability to take in food may result in anorexia. Some of the causes of prehension difficulty include facial nerve paralysis, glossal trauma, labiaitis, dental pain, CNS disturbance, temporomandibular arthritis, trauma to the mandible/premaxilla/maxilla, infection or tumors of the same bone, congenital defects (brachygnathism of either the mandible or the maxilla), and foreign bodies in the oral cavity.

Dysphagia

Reluctance or inability to swallow may be caused by hypoglossal or glossopharyngeal nerve paralysis, pharyngitis, esophagitis, pharyngeal abscesses, pharyngeal tumors, esophageal obstruction, stomach overload, bloat, infectious diseases (rabies, botulism), or congenital defects (agenesis of the soft palate, cleft soft palate). With choanal atresia, the infant cannot eat and breathe simultaneously. Fracture of the hyoid apparatus and a variety of CNS disorders may also cause dysphagia.

Signs of dysphagia include drooling of saliva (sialosis), retention of feed in the oral cavity, a fetid odor from the oral cavity, gagging, and retching.

Regurgitation and Emesis

It is important to differentiate between regurgitation and emesis. Regurgitation may be voluntary in camelids. It is used as a threat or as a defense against aggression between males or by the female if she is pregnant or unwilling to submit to the male. This behavior can be anticipated if the ears lie back over the neck, followed by a gurgling noise in the pharyngeal region as the camelid prepares to eject the bolus. On the left side, the bolus can be seen traversing the cervical region. Stomach contents can be spewed a distance of 3 m quite accurately.

Active regurgitation may occur as a result of esophageal stimulation as a gastric tube is passed or from laryngopharyngeal stimulation when an endotracheal tube is inserted. Passive regurgitation may occur from relaxation of the stomach cardia during anesthesia or from improper positioning during recumbency. If possible, the llama should lie on its right side. Abdominal pressure, either externally applied or as a result of bloat or other abdominal distention, may also cause regurgitation.

Emesis is the involuntary projection of stomach contents by a reflex action and consists of an initial deep inspiration followed by closing of the glottis to prevent aspiration of ingesta. At the same time, the soft palate should be automatically elevated to seal off the nasopharynx. Ingesta is ejected by a strong contraction of all the abdominal muscles and antiperistaltic contractions of the esophagus. In the llama, as in the horse, emesis is a grave sign because of the elongated soft palate. If the soft palate fails to seal off the nasophar-

ynx, it is relatively easy for ingesta to be pushed into the nasal cavity, causing rhinitis, obstruction, or aspiration.

Some of the causes of emesis include overloading of C-1, gastritis, diaphragmatic hernia, arsenic poisoning, partial esophageal obstruction, and the ingestion of poisonous plants. A list of such plants includes oleander (*Nerium oleander*), corn lily (*Veratrum californicum* or *V. viridis*), lily of the valley (*Convallaria majalis*), foxglove (*Digitalis purpurea*), sneezeweed (*Helenium* spp.), and various members of the Ericaceae family, all containing the same andromedotoxin (a glycoside), including rhododendrons and azaleas (*Rhododendron* spp.), laurel (*Leucothoe* spp.), labrador tea (*Ledum* spp.), and kalmia (*Kalmia* spp.).

The clinical evidence of regurgitation or emesis is obvious. The medical management of these conditions depends on the primary cause. Any obstruction should be corrected and irritants removed from the stomach, which may require gastrostomy. In addition, C-1 electrolyte and microbial balance should be reestablished.

Abdominal Distention (Bloat)

Abdominal distention may be caused by excessive fat, presence of a fetus, excessive gas (flatus), accumulation of feces, or abdominal fluid (five Fs of abdominal distention). A thorough physical examination may lead to identification of the cause, but in camelids, a thorough rectal examination is not possible.

Abdominal distention is frequently benign, caused by overeating. However, excessive or prolonged abdominal pressure pushes the diaphragm forward, causing dyspnea. Compression of the abdominal vessels interferes with circulation, and shock may ensue (Figures 13.58A and B).

Figure 13.58A. Bloat in an alpaca.

Figure 13.58B. Bloat in an alpaca.

Tenesmus

Tenesmus is ineffectual straining to defecate. Nondigestive disorders may mimic this straining. Potential causes include diarrhea, parturition, rectal or vaginal prolapse, urethral calculus, cystitis, vaginitis, proctitis caused by irritation from a rough rectal examination, CNS derangements (spinal cord compression), and rabies.

The signs of tenesmus are obvious. Management requires correction of the primary cause. An epidural anesthesia may give temporary relief and possibly interrupt the painful cycle that perpetuates the tenesmus. Alcohol epidural blocks have proved to be efficacious when treating cattle with chronic tenesmus.

Diarrhea

Diarrhea may be defined as the abnormally frequent passage of feces that are irregularly formed and more moist or fluid than usual. Diarrhea is such a frequently observed clinical sign that it cannot be considered to be definitive in differential diagnoses. However, some of the diseases for which it is important for making a tentative diagnosis should be discussed.

In neonates the major cause is dietary upset, usually seen in orphan neonates experiencing rapid changes in the milk supplied. Overfeeding is also a factor. Infectious causes include colibacillosis, coronavirus, and rotavirus infections. Coccidiosis may be seen within the first few weeks of life. Poor sanitation contributes to the exposure of the neonate to opportunistic or pathogenic microorganisms. If the neonate failed to receive the passive transfer of immunoglobulins, such an animal is a prime target for infection. Septicemic neonates usually have diarrhea.

In mature camelids, enteritis is the most common cause of diarrhea and may be initiated by clostridial toxemia, parasitism, stomach overload (C-1 acidosis), or plant or other poisoning (arsenic, copper, lead, molybdenum). Additional causes are overhydration, consumption of too much lush feed, and sudden change in feed. Nondigestive-tract causes of diarrhea include excitement, hepatopathy, and poisoning from organic phosphate insecticides. Infectious diseases

causing enteritis, hence diarrhea, include salmonellosis, paratuberculosis, colibacillosis, rinderpest, and tuberculosis.

An overview of the pathogenesis of diarrhea may help in visualizing the many factors that may be contributors. There are five basic causes of diarrhea: (1) osmotic pressure imbalance, (2) inhibition of absorption of electrolytes, (3) derangement of the secretory function of the intestinal mucosa that may likewise adversely change the electrolyte balance, (4) changes in mucosal permeability caused by denudation of the epithelium or circulatory changes, and (5) derangement in the motility cycle of the intestine.

The signs of diarrhea are obvious. There are, however, numerous types of diarrhea, each of which has a bearing on the diagnosis. General signs include depression, dehydration (sunken eyes, dry mucous membranes, loss of skin pliability), fever in some cases, colic, tenesmus, weight loss, hypothermia, and loss of nursing reflex in crias. The odor of diarrheic feces may be quite offensive. A detailed discussion, including the management of diarrhea, can be found under Enteritis or specific diseases with diarrhea as the major clinical entity.

Constipation

Constipation is the abnormally infrequent or difficult evacuation of feces that have remained in the rectum and are usually more firm or dry than usual. The term "obstipation" is sometimes used instead, but the precise meaning of this term is intractable constipation. In the usual clinical sense, constipation refers to any slowness or absence of passage of feces. It is important to differentiate between true constipation, in which feces pass into the colon and rectum, but the rate of passage is slow and fluid is absorbed, making the feces hard and dry, and the more serious obstipation, in which an obstruction or other cause prevents passage of feces into the terminal colon.

Because camelids pass a relatively dry fecal pellet, the dryness of feces may be a difficult sign to interpret. The constipated llama is not likely to be straining. When feces are collected from the rectum, the pellets or the fecal mass may be covered with mucus, giving the feces a shiny appearance.

Some of the causes of constipation include malnutrition or starvation, dehydration, painful condition around the anus, pelvic abscess, pelvic fracture, pelvic tumors, acorn poisoning (*Quercus* spp. tannins), paralytic ileus, general debility, spinal injury, lack of exercise (downers), and toxicosis (lead, fluorine, chronic zinc poisoning).

Ileus

Ileus refers to intestinal obstruction. True, or complete, ileus may be caused by external compression of the intestine from peritoneal adhesions, tumors, advanced pregnancy, torsions, or internal hernias. Such obstructions are referred to as mechanical ileus. Dynamic ileus may be spastic, caused by a constriction around an impaction or enterolith, or paralytic, a result of intrinsic disorders of bowel motility. Intestinal atony may be caused reflexively postoperatively as a result of peritonitis, ischemia, or overstretching of the intestine from ingesta or gas. Metabolic changes may also initiate an ileus (acidosis, hypokalemia).

Stomach Atony

Stomach atony does not occur as a primary disease but rather is a clinical sign caused by numerous disorders including grain overload (rare in llamas), simple indigestion, sudden changes of feed, prolonged antimicrobial therapy, spoiled feed, gastric ulceration, and obstruction of the cranial intestinal tract.[85] Other causes might be hypomagnesemia, septicemia, and other types of colic.

Microflora and fauna activity is crucial to proper stomach activity, and any condition that destroys normal microorganisms predisposes to gastric atony. Atony is usually accompanied by anorexia.

Lack of motility is generally ascertained by auscultation with a stethoscope. The contractions of C-1 are not as pronounced as contractions of the rumen in the ruminant, so palpation in the left paralumbar fossa is not helpful. The rate and rhythm of C-1 contractions vary with the proximity to feed ingestion. If the llama is eating or has recently consumed feed, the contractions will be frequent, two to four per minute, with no intervals between. If no rumination is taking place, the rate is slower. In the normal llama, the only abdominal sounds are from C-1. With obstruction or delay of movement of ingesta along the intestinal tract, gas formation may mix with fluid and produce intestinal borborygmus.

Stomach atony is usually only a sign of other clinical problems, so medical management of atony should deal with the primary disorder. However, prolonged atony of C-1 (forty-eight to seventy-two hours) may initiate a change in the pH of the ingesta, which in turn will destroy the microflora and fauna. Ultimate reestablishment of gastric motility may necessitate transfaunation.

Colic[13]

Much has been written about equine colic. The pathophysiology has been intensively studied, and great advances have been made in dealing with digestive disorders causing colicky or abdominal pain. Ruminants also have digestive disorders that cause obstruction and distention of the gastrointestinal (GI) tract, but ruminants react to pain differently. Camelids seem to be more like the horse in expression of the pain

of colic, but the digestive disorders of the camelid resemble those of the ruminant because of the closer similarity of the digestive tract anatomy.

Normal GI mucosa is insensitive to touch, cutting, pinching, and tearing. GI pain arises from stretching of the muscular layer and serosa, as occurs with distention of the viscus or with powerful muscular contractions.[53]

The sensory response is via the afferent visceral fibers accompanying the sympathetic pathway.[53] An acute inflammation or vascular engorgement decreases the pain threshold. As a result, mild stimuli that would not initiate a pain response in the normal bowel cause pain. Tissue hypoxia also decreases the threshold; thus, obstructive lesions compromising circulation cause significant pain. Excessive production of acid in C-1 may produce a direct inflammation of the gastric mucosa and initiate a pain response. Acid may also stimulate muscle spasms.

Most digestive tract disorders, at one time or another during the course of the disease, produce colicky pain. An umbilical abscess caused colic in a three-month-old cria. What appears to be GI pain may arise from disorders of other organ systems, so careful evaluation in the initial examination is important. Uterine torsion, cystitis, urethral obstruction, liver diseases, back pain, and certain infectious diseases (salmonellosis, rabies, anthrax) should be considered. Even thirst or starvation may, in the early stages, cause stomach contractions that will be expressed as painful colic.

Signs of colic in the camelid are similar to those seen in the equine and include groaning, grinding of the teeth, getting up and down, rolling, refusing to get up, kicking at the belly, peculiar stances, pressing into a corner, looking back at the belly, frenzy, depression, anorexia, arched back, tenesmus, tense abdomen, stomach atony, increased heart rate, frequent passage of small amounts of urine, tenseness of facial muscles, and a pained expression of the eyes (Figures 13.59 to 13.61).[36] Signs of colic in the neonate may be caused by retained meconium, atresia ani or atresia coli, and incarceration of an intestinal loop in an inguinal or umbilical hernia.

Other conditions that may mimic signs of colic include normal rolling behavior, weakness, incoordination (insecticide and heavy metals poisoning, head injuries, spinal cord compression), septicemia, and arthritis. Other sources of abdominal pain may be pleuritis, peritonitis, ruptured liver, and ruptured bladder.

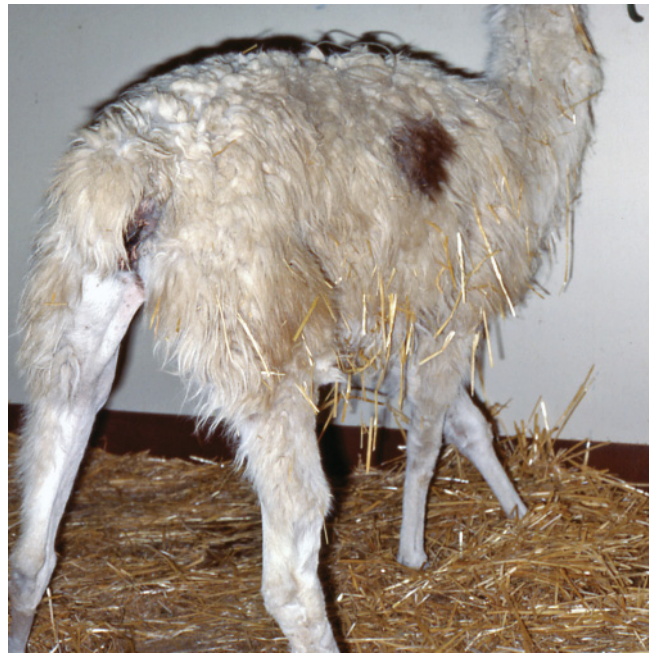

Figure 13.59. Abnormal stance of a llama with colic.

Figure 13.60. Pressing and abnormal position of a llama with colic.

Figure 13.61. Depression and recumbency of a llama with colic.

DISEASES OF THE DIGESTIVE SYSTEM[4,20,50,85]

Lip Disorders

Laceration of the lips may be caused by barbed wire or other sharp objects in the environment or

by dog bites. The lips may also be lacerated by the incisor teeth as a result of a blow to the muzzle. Puncture wounds occur similarly. Labial swelling may result from dependent edema from hypoproteinemia or anemia, cellulitis, a tumor, constriction of venous drainage, contusion, or rattlesnake bite (Chapter 23).

Erosions and ulcers of both the external and internal surfaces of the lips may be caused by vesicular diseases (foot-and-mouth disease, vesicular stomatitis), rinderpest, or grass awns (yellow bristle grass, *Setaria lutescens;* foxtails, *Hordeum* spp.) (Chapter 23). Caustic materials are known to cause labial ulcers in livestock and horses, but camelids are more cautious about ingestion of such materials.

Proliferative lesions of the lips may be caused by contagious ecthyma (Chapter 7). Paralysis of the lips may follow damage to the facial nerve, as in listeriosis (Chapter 7).

Stomatitis

The term "stomatitis" encompasses the inflammatory response to injury or disease of any oral tissue. Designation of a specific organ may be made, such as glossitis or gingivitis.

Etiology

Any of the factors causing lip problems may also affect the mouth. Additional damaging agents may include foreign bodies, candidiasis, actinobacillosis, trauma from gastric tube passage, cellulitis arising from focal infections within the mouth, and developmental defects (malocclusion).

Signs

Signs of oral ailments include inappetence, peculiar chewing motions, excessive salivation, odorous breath, hyperemia of the mucosa, erosions, ulcers, and vesicles.

Diagnosis

Careful physical examination of the mouth usually allows visualization of the lesions, although it may be difficult in SACs because the mouth is narrow and will not open widely. The camelid usually resents manipulation of the mouth, so sedation may be necessary to fully examine the mouth and carry out diagnostic procedures, including inspection, cultures, and biopsy. Because some oral diseases are foreign animal diseases, a definitive diagnosis is mandatory. Any case suspected of being a vesicular disease should be reported immediately to state or federal regulatory agencies. Procedures necessary to arrive at a definitive diagnosis will then be conducted by them.

Treatment

Correct the primary condition. Provide soft, palatable feed. Using a dose syringe with a long nozzle, flush the mouth with water to cleanse it of feed particles or exudate.

Tongue Disorders

No diseases specific to the tongue of the camelid have been reported. Glossitis may be a part of a general stomatitis. Hypoglossal nerve paralysis may occur as a result of a blow to the intermandibular space or from fractures of the mandible and hyoid apparatus, abscesses at the base of the tongue, or lesions in the brain (encephalitis, rabies, tumor).

The tongue is rarely lacerated because it does not protrude from the mouth. Sharp dental enamel points could traumatize the tongue, but this is rare. The large canines in the male or the incisors of both sexes may puncture or lacerate the tongue if the llama should fall or strike its head in an attempt to flee from an aggressive enclosure mate or restraint attempts. The tongue may also be lacerated by overzealous pulling on the tongue when inserting an endotracheal tube.

Diagnosis

Physical examination is necessary to establish the presence of a tongue disorder. In the instance of paralysis, more intensive investigation is necessary to ascertain the reason for the paralysis. This usually involves a neurologic evaluation.

Deglutition is usually inhibited with any lesion on the tongue. Bilateral hypoglossal nerve injury would allow the tongue to protrude from the mouth and possibly be secondarily traumatized.

Treatment

Lacerations should be sutured (probably requiring general anesthesia); otherwise, general nursing care is recommended.

Disorders of the dulaa of male camels is discussed in detail in Chapter 6.

Oral Abscess

Etiology

Oral abscesses may be caused by numerous opportunistic bacteria. The infection may enter through a break in the oral mucosa or be blood borne. *Arcanobacterium pyogenes* is the organism most frequently cultured from oral abscesses in the author's practice, but streptococci, staphylococci, actinobacillus, fusobacterium, and actinomyces have also been encountered.

In one case, an abscess on the lateral buccal surface ventral to the eye was repeatedly lanced. The organism isolated was *Actinomyces* sp. (Chapter 7). Because the

abscess complex was situated in the region of the passage of the facial nerve and the parotid duct, massive surgical extirpation was avoided until the failure of other measures to resolve the problem prompted drastic action. The abscessation had spread dorsally and caudally toward the ramus of the mandible. Radical surgery, followed by packing and irrigation with hydrogen peroxide and organic iodine, finally eradicated the problem.

Necrobacillosis is a serious problem in SACs in South America. Necrobacillosis causes severe abscesses of the bones of the mandible and maxilla, but soft-tissue abscesses occur as well. See Chapter 7 for a full discussion of this disease.

Dental Problems

Diagrams of dental eruption in the llama are illustrated in Figures 13.9 to 13.37. Alpaca eruption is similar. The eruption time for camel teeth is tabulated in Table 13.3

Etiology[63,77,79]

All of the dental problems seen in ruminants have been seen or are potential problems in camelids.[16] Conditions diagnosed by the author include dental plaque; pigmentation of the teeth; alveolar periostitis; pulpitis; fractures of the incisors, canines, or molars (Figure 13.62); fractures of the mandible; sharp enamel points caused by malocclusion; excessive wear; elongated teeth associated with superior brachygnathism; and other developmental defects causing malocclusion. The most common dental surgery is for canine tooth removal.[54,55]

Figure 13.62. Fractured mandibular cheek tooth.

Dental examination and care have become a discipline in veterinary medicine. A chart, similar to those used by dentists, may be appropriate for recording findings (Figure 13.63).

Signs

The signs of dental problems include inappetence, weight loss, abnormal chewing, dropping of a cud, swelling over the dental roots, mandibular fistulae, and sinusitis (superior molar roots are contiguous to the maxillary sinus).

Diagnosis

Physical examination of the teeth is not easy and frequently necessitates anesthesia. Radiography is necessary, but high-quality, properly positioned films are required if radiography is to be of benefit.[66] Oblique shots of the cheek teeth must be taken to prevent override from the opposite jaw. Radiographs of dental lesions of camelids are evaluated in the same manner as those of other animals. Loss of the lamina dura dentes, radiolucent areas with abscessation, fractures and increased density with sclerotic bone, and osteomyelitis are conditions that may be seen. Figure 13.63 is a chart for recording dental problems.

Treatment

Plaque rarely interferes with oral function and does not require removal unless it is causing gingivitis. Pulpitis and alveolar periostitis may necessitate extraction or repulsion of the tooth (Chapter 6). If a dental fracture opens the pulp cavity, a pulpotomy may be done. If the pulp has become infected, the tooth should be removed.

Genetic defects can be eliminated only by a stringent breeding program, including culling. Elongated incisors may be cut off with a circular saw or an obstetric wire under anesthesia. The pulp cavities are rarely exposed. This should not be done to prepare a llama or alpaca for a show or sale. Elongated incisors are an unsoundness that should bar them from participation. For more details, see Chapter 6.

Salivary Gland Disorders

Etiology

The salivary glands of camelids are affected by problems similar to those of ruminants. Abscesses within or next to the salivary glands are most common. *Arcanobacterium pyogenes* is usually isolated from abscesses.

Generalized parotiditis has not been described. Salivary fistulae may occur from lacerations of the side of the face or abscessation and rupture of a duct. A laceration of the body of the gland is possible, but the skin thickness over the neck and face is such that a severe

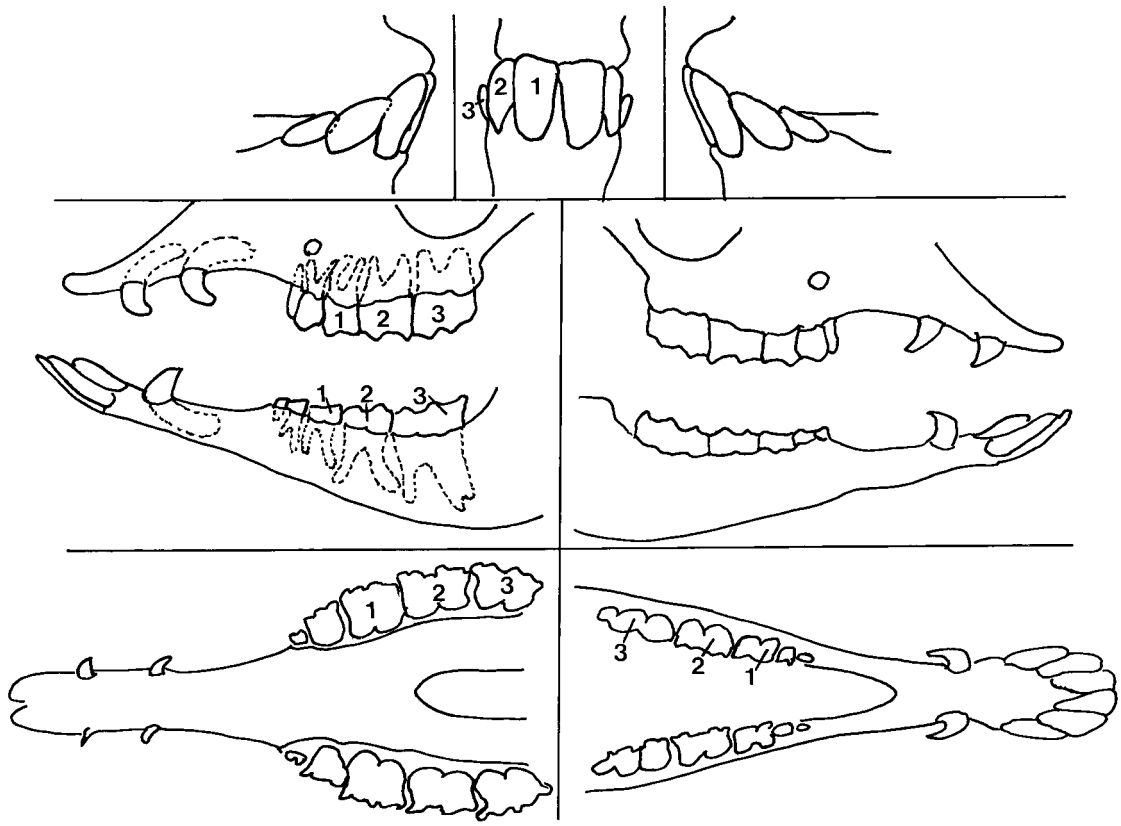

Figure 13.63. Diagram for dental examination.

slashing blow would be required to penetrate the skin. A parotid salivary fistula may occur from a facial laceration. Sialiths have not been reported but are likely to occur.

Signs

The primary sign is swelling over the gland, either firm in the case of cellulitis or parotiditis or fluctuant with an abscess. The area is hot and the skin hyperemic. The llama is likely reluctant to eat because of the pain in the angle of the jaw.

In the case of a salivary fistula, an excessive flow of saliva is observed, especially while the llama is feeding or ruminating.

Diagnosis

A physical examination usually discovers the problem. A needle may be inserted into a fluctuant swelling to establish the presence of an exudate, serum, or blood.

Treatment

Primary parotiditis requires broad-spectrum antimicrobial therapy. However, this is of little value in abscesses. It may be desirable to wait for an abscess to mature and localize before lancing, but otherwise a needle should be inserted at the ventral aspect of the abscess. When the exudate begins to flow, the needle should be left in place and an incision made along the needle in a vertical direction through the skin. If possible, blunt dissection should be continued with a hemostat to open the abscess. The cavity may be explored with a gloved finger and irrigated with a 1:4 dilution of povidone-iodine solution. The irrigation should be continued daily until the cavity has healed from the inside out.

Pharyngitis

Pharyngitis is any inflammation of the mucous membrane and surrounding tissue of the oro- or nasopharynx.

Etiology

Pharyngitis may be caused by acute or chronic bacterial infections, especially those of the respiratory system. Causes also include trauma from inept gastric intubation, abscesses, *Cephenemyia* spp., larvae infestation of the nasopharynx (Chapter 8), and developmental defects such as agenesis of the soft palate or cleft soft palate (Chapter 22).

Signs

Signs of pharyngitis include anorexia, weight loss, nasal discharge, coughing, fluctuant swelling behind the ramus of the mandible, extended head, pain response to palpation of the throat region, and hyperemia found in the mucous membrane when it is examined with a laryngoscope or a Frick-type speculum.

Diagnosis

With the llama under anesthesia the oropharynx may be visualized by insertion of a long-bladed laryngoscope. Anesthesia is necessary because if examination is attempted without sedation or anesthesia, the llama may reflexively regurgitate. A Frick speculum is too large to insert into the mouth of any but the largest llama. The principle, however, may be applied by substituting an appropriately sized, smooth-ended piece of polyvinyl chloride pipe for the speculum. The nasopharynx may be explored with an endoscope via the nasal cavity, and the oropharynx, via the oral cavity.

Treatment

Soft, palatable feed and suitable quantities of water should be provided. Dehydration may be more of a problem than lack of food. If the problem is not resolved within a couple of days, with resumption of normal eating habits, it is necessary to institute intravenous (IV) feeding. Parenteral antibiotics may be indicated.

It is usually necessary to lance abscesses. Sweating helps to bring an abscess to maturation. This may be done by frequent applications of moistened hot towels or by smearing the area with an irritant such as ichthammol. This requires that the wool in the upper neck be clipped, which most llama owners resist.

Esophagitis

Etiology

Primary esophagitis is rare in camelids. There are no reports of rinderpest in SACs, but it has been reported in Old World camels, so lack of exposure is the probable reason the disease has not been reported in SACs.

Esophagitis has been observed following repeated gastric intubation, especially with a rough-ended tube. Zinc deficiency or prolonged therapy with NaEDTA may result in esophageal parakeratosis.

Signs

Esophagitis is usually indicated by anorexia and a reluctance to swallow and/or regurgitate.

Diagnosis

Diagnosis is difficult unless large-animal endoscopic equipment is available.

Treatment

The primary cause should be eliminated, then time allowed for healing.

Esophageal Obstruction (Choke)

Obstruction of the esophagus is less common in camelids than in equines or bovines because of the ability of camelids to regurgitate freely. Camelids have more voluntary control of the esophagus than equines. However, choke related to apples and alfalfa pellets has been reported in the llama.[37] Obstruction may be partial or complete. Any foreign body that becomes lodged in the esophagus will initiate muscular contractions with the purpose of moving the object one way or the other. If neither peristaltic nor antiperistaltic action is effective, the end result may be a spasm on either side of the obstruction that complicates the situation further and which may turn a partial into a complete obstruction.[39,47,83,116]

Etiology

A reported instance of choke in a llama involved the ingestion of apples falling from a tree in an orchard where the llama was corralled. In cattle, various feed items such as carrots, potatoes, or even boluses of grain have become lodged in the esophagus. The problem usually arises when a greedy animal bolts a mouthful of feed and attempts to swallow it without chewing. A periesophageal hematoma formed following venipuncture was responsible for external compression of the esophagus and subsequent choke.[116] Neural dysfunction may also cause interference with esophageal peristalsis.

Signs

Choke usually initiates alarming behavior that indicates the animal is in discomfort. The signs could be confused with colic, since they include retching, coughing, dysphagia, head shaking, salivation, nasal discharge, anxiety, and, if a blockage is prolonged, dehydration. If the animal continues to eat, food may pack in the pharyngeal area, and subsequent retching may drive feed particles into the nasopharynx and out the nose. Aspiration of feed material into the trachea and lungs is a common sequel to choke.

The obstruction may be lodged anywhere from the origin of the esophagus to the cardia. If the object is in the cervical region, swelling may be observed over the esophagus. The esophagus of the camelid lies deeper than that of other species and is somewhat obscured by the ventral tuberculum of the transverse process of

the cervical vertebrae. Palpation may be necessary to locate the obstruction. The object causing the obstruction need not be larger than the bolus the animal would normally ingest or regurgitate for rechewing. The exact reason for initiation of a spasm causing an obstruction is not entirely known.

Diagnosis

Clinical signs are characteristic in the early stages, but if the blockage persists, the animal will become depressed, and retching and coughing will diminish or cease. If blockage is suspected, a large gastric tube should be inserted into the esophagus and gently advanced to determine the patency of the esophagus.[108] Vigorous force should not be used to push the object along. Radiography, both plain and contrast, is helpful, particularly in diagnosing obstructions within the thoracic esophagus. The diagnosis may also be aided by response to spasmolytic therapy.

Treatment

Objects in the upper esophagus cannot be retrieved by hand, as they may be in cattle. The narrowness of the throat of the llama does not allow this. Spasmolytics (atropine, regelen) should be given to relax the esophageal musculature and, hopefully, allow peristaltic activity to resume. Analgesics may be given, because this condition is certainly painful. Once the esophagus is relaxed, a large equine stomach tube may be used to gently push the object onward.

The foregoing stimulation of the pharynx and esophagus may initiate a reflex regurgitation attempt, and sedation or anesthesia may be necessary to continue. Aspiration of feed and saliva into the lungs is always a risk, and endotracheal intubation may be desirable when dealing with a difficult case.

Complete obstruction of the esophagus prevents eructation, which may lead to the development of bloat. Bloat may be relieved by inserting a large-bore needle into C-1 through the left paralumbar fossa, similar to the treatment of bloat in the ruminant.

Surgical intervention is rarely required to release an obstruction, but if it is necessary, reference to the equine literature will provide details of the technique.

Esophageal Dilatation (Megaesophagus, Paralysis of the Esophagus and Esophageal Achalasia, Cardiospasm)[115]

Megaesophagus has been diagnosed in the author's clinic, and a retrospective study of fifteen cases at one university has been reported.[114] The disease is characterized by dilatation of all or a part of the esophagus, especially the intrathoracic segment. In dogs, there may or may not be an associated failure or relaxation of the esophagogastric junction.[46]

Etiology

Watrous et al. list more than forty potential causes for megaesophagus in the species that have been studied (humans, horses, dogs, cats, cattle, sheep, and pigs).[114] The cause of esophageal dilatation (ED) is unknown in the llama. The disorder may be congenital, but no hereditary transmission has been proven. Although clinical signs may be observed shortly after birth, it is more likely to be observed at about the time of weaning. ED may be acquired as an adult for a variety of reasons, including an episode of choke that may traumatize the esophageal mucosa and ultimately lead to a fibrotic stenosis.

Signs

Hypersalivation is a frequent sign, but recurring regurgitation is the major sign. The material regurgitated may not have the normal odor of C-1 contents because it has never reached the stomach. Regurgitation is most likely to occur shortly after feeding and when the head is lowered to feed or drink. An affected animal fails to grow or loses body condition. Additional signs include dysphagia, anorexia, esophageal waves, fetid breath, and reduced libido. Aspiration pneumonia is a common sequel in the dog and foal and, presumably, in the camelid. Dehydration may be a factor as well.

Diagnosis

A greatly dilated esophagus is observed on plain radiographs of the thorax (Figure 13.64). More definitive delineation is obtained by using contrast radiography. Fluoroscopy may also be used to evaluate the peristaltic activity of the esophagus. Figures 13.65A and B shows the dilated esophagus in situ at necropsy. Mild rhabdomyolysis of the skeletal muscle component of the distal esophagus was present on histologic examination.

Treatment

Insufficient cases have been managed to arrive at any conclusions as to treatment. Young dogs may improve as they mature. In dogs, more frequent ingestion of smaller quantities of food has been suggested to avoid overloading the esophagus.

Dietary management has been used in llamas, but the prognosis should be guarded to unfavorable. The presence of pneumonia may necessitate antimicrobial therapy. In foals, the cardiospasm is treated with promazine hydrochloride and spasmolytics such as dipyrone (Novin). Any attempted treatment should include feeding the animal on an inclined plane so the fore-

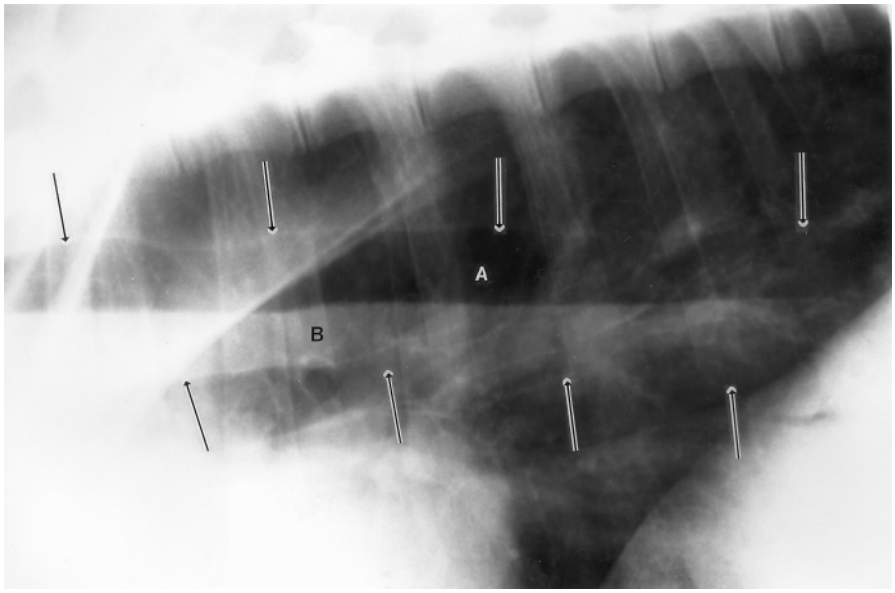

Figure 13.64. Lateral radiograph of dilated thoracic esophagus (arrows outline the wall of the esophagus). (A) Gas, (B) food.

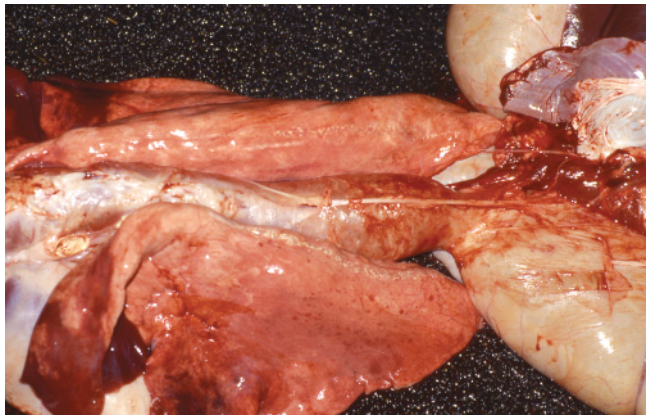

Figure 13.65A. Thoracic esophageal dilatation in a llama.

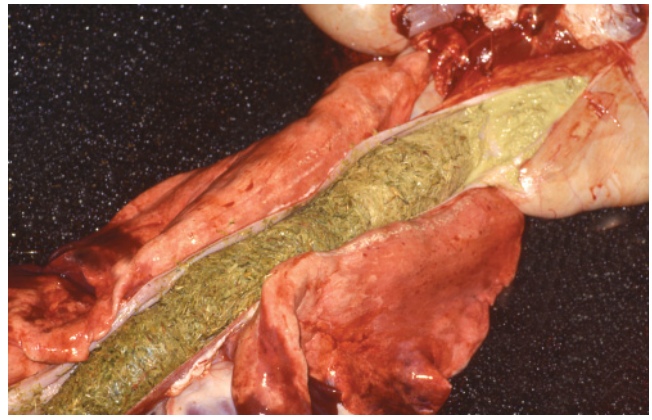

Figure 13.65B. Megaesophagus opened.

quarters are elevated. Gravity then helps the esophagus to empty.

Stomach Disorders[1]

Disorders of the stomach may be primary or secondary to other diseases of the gastroenteric or other systems.[92] The conditions seen by the author include simple indigestion (stomach atony), gastritis, parasitism (Chapter 8), and gastric ulceration with or without perforation. A llama will rarely engorge itself on grain or other concentrates, and bloat is not common. SACs are not troubled with displaced C-3 or traumatic gastritis, nor has vagal indigestion been described.

Gastric Ulcers[78,96]

Primary gastritis is not usually diagnosed as a clinical condition but may be seen as a lesion at necropsy. Gastric ulceration, a common disorder, is not easily diagnosed but may contribute to serious problems in camelids. Gastric ulcers may be seen in any of the stomach compartments. In C-1 and C-2, the ulcers are likely to involve the margins of the saccules in the glandular areas (Figures 13.66, 13.67).

Ulceration of the mucous membrane of C-3 is common. The ulcers in the proximal four-fifths of C-3 tend to be linear along the ridges of the longitudinal pleats (Figure 13.68). Ulcers of the distal one-fifth are more focal, deeper into the submucosa, and more likely to perforate (Figures 13.69 to 13.72). Bleeding ulcers like those seen in ruminants, resulting in anemia and melena, have not been reported. Occult blood tests have been uniformly negative in the author's practice.

Etiology. The etiology of ulcers is unknown. The role of stress in the development of the syndrome is

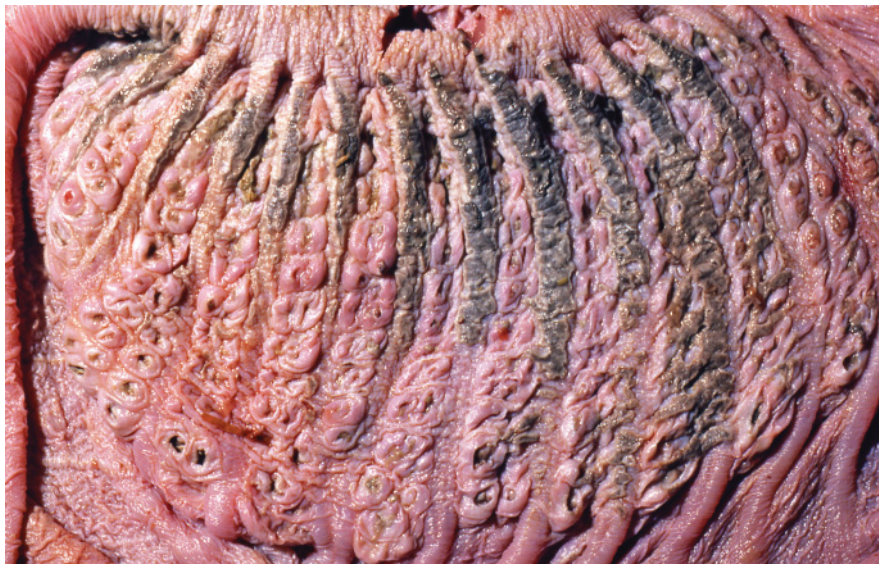

Figure 13.66. Mucosal ulceration of C-1 in a llama.

Figure 13.67. Mucosal ulceration of C-2 in a llama.

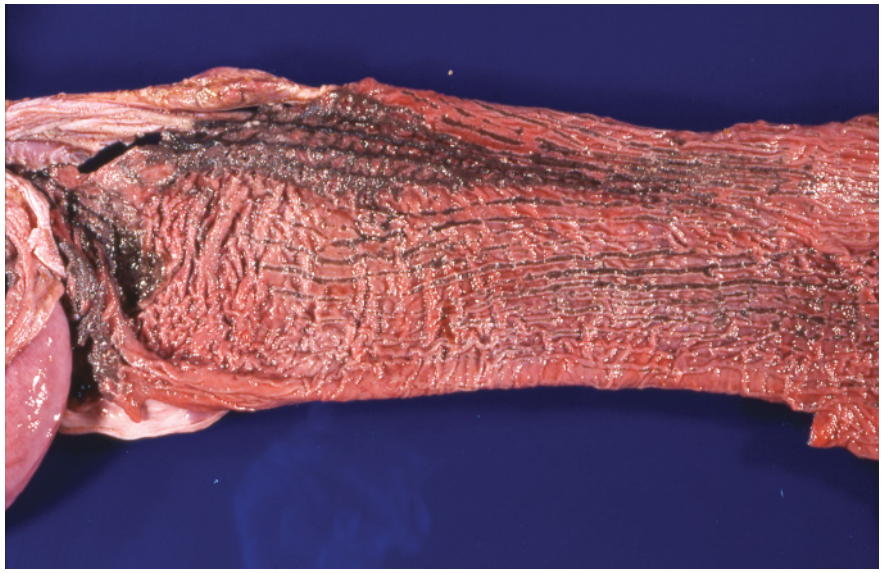

Figure 13.68. Linear mucosal ulceration of C-3 in a llama.

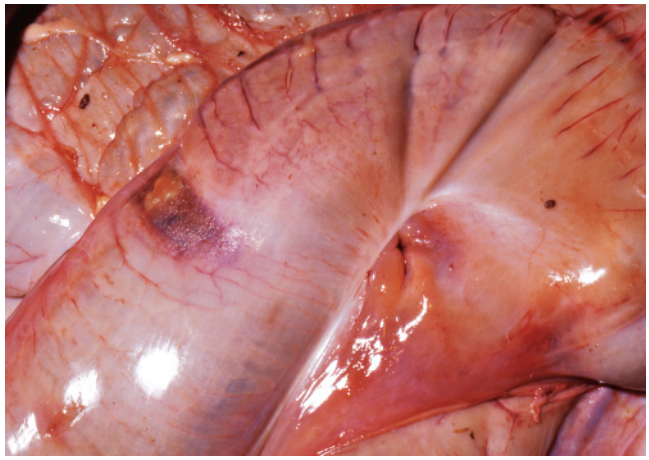

Figure 13.69. Serosal surface of C-1 with gastric ulcers.

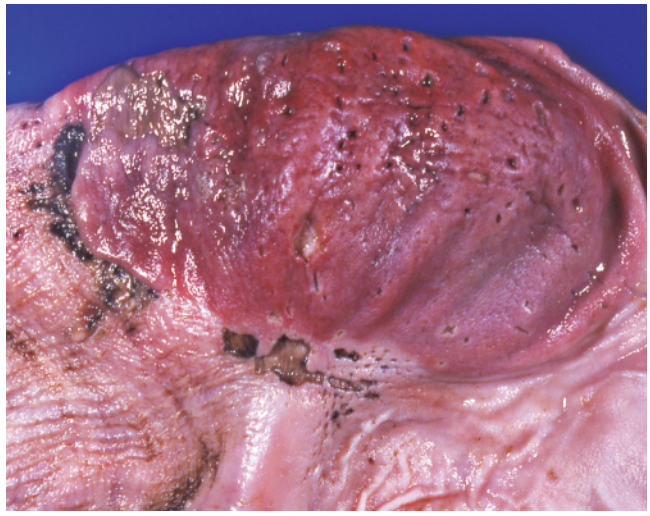

Figure 13.70. Ulceration of C-1.

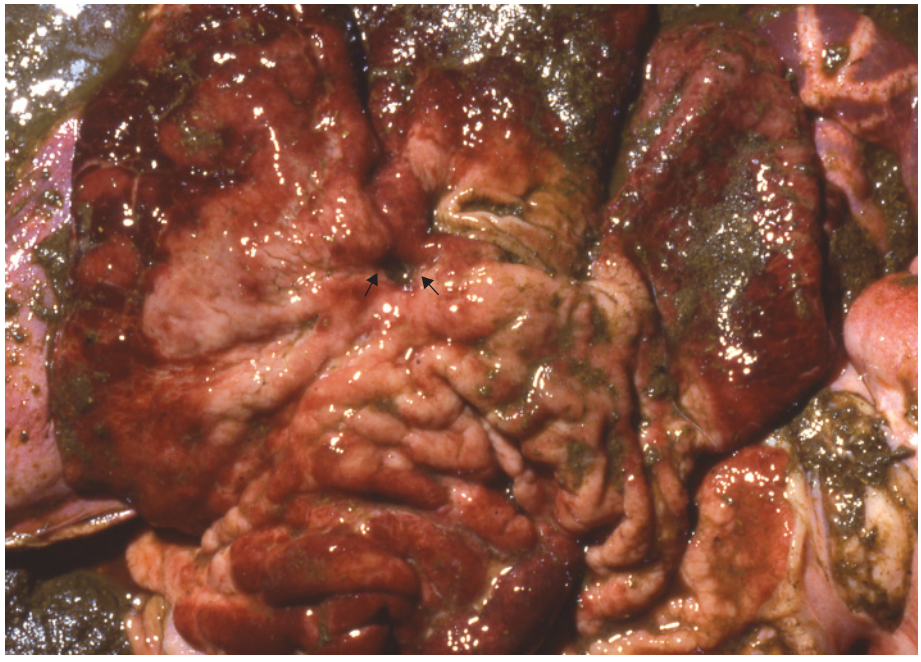

Figure 13.71. Perforating gastric ulcer in C-3.

unknown as well (Chapter 9). The condition has been diagnosed in animals on good rations as well as in those on poor rations. Some have been infested with parasites, while tests of others have been negative for parasitism.

Signs. Atony of C-1, with inappetence and scanty feces, may be the major syndrome. Nonperforating ulcers do not stimulate a hematopoietic tissue response or change the composition of the peritoneal fluid or blood serum. The temperature, heart rate, and respiratory rate are usually normal. The llama may be depressed and stand off from the herd, but colic is not a prominent sign. The owner will frequently comment that the llama is by itself, lying down, and not coming up to feed in the usual manner. Unfortunately, these are the preliminary signs for most of the digestive and respiratory disorders. Animals exhibiting these mild signs should be given a thorough physical examination. If an ulcer has perforated, the signs of peritonitis will be seen.

Diagnosis. Simple gastric ulceration can be diagnosed only by elimination of other, more serious digestive

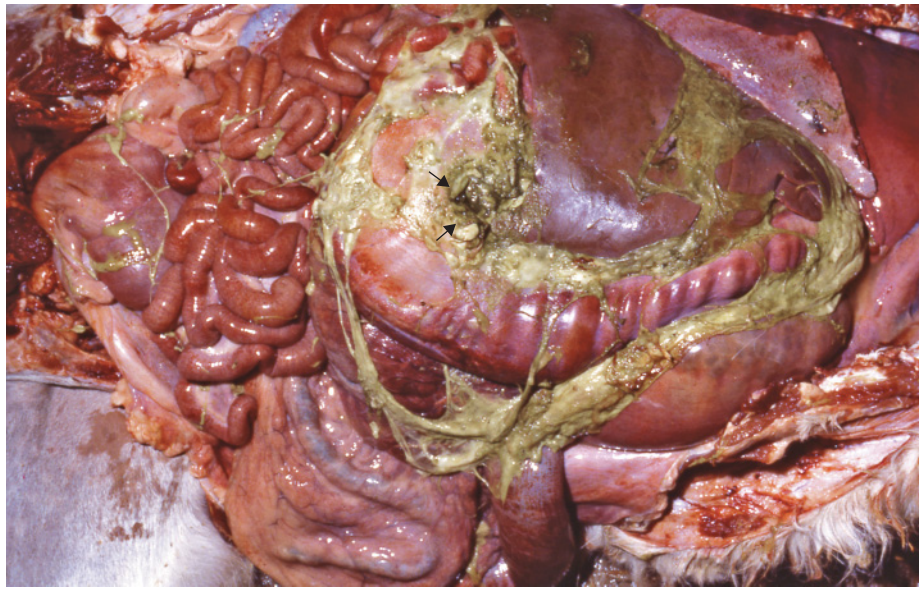

Figure 13.72. Acute, fibrinous peritonitis caused by perforating gastric ulcer.

Table 13.7. Systematic examination of a colicky llama.

Obtain a complete history—diet, stressors, pregnancy, recent acquisition
Clinical examination
Temperature
Heart rate—there are no good arteries to evaluate the pulse
Respiratory rate and thoracic auscultation
Palpation (if possible)—umbilicus, scrotum, abdomen (tenseness), rectal
Auscultation of the abdomen
Evaluation of colicky pain
Special diagnostic procedures
Abdominocentesis—midline near the umbilicus
Hematology
Serum chemistry and electrolytes
pH of C-1
Response to therapy

disorders. Tables 13.7 to 13.11 provide values for differential diagnosis.

TREATMENT. No studies have been conducted to establish the response of camelids to most of the medications used in the treatment of ulcers. The effects of antacids on the camelid stomach are unknown. Pharmacologic studies on ruminants indicate that these species do not have the same type of acid receptors in the stomach as do simple-stomached animals. Therefore, ruminants should not be responsive to cimetidine. The type of acid receptor in camelids is not known, but empirical experience indicates that they do respond to cimetidine therapy. Drew et al. studied the effects of flunixin meglumine and cimetidine in the llama and found that neither of them changed the pH of the third compartment to any significant extent;[23] however, clinical experience dictates that cimetidine be used. The recommended dosage regimen is 2.2 mg/kg, given twice daily subcutaneously. Oral cimetidine therapy in the adult ruminant is contraindicated because the drug is destroyed in the rumen. It is assumed that this is also true in llamas. If there is prolonged anorexia, no response to treatment, and no production of feces, it is the author's recommendation that an exploratory laparotomy be performed.

Different regimens may be indicated for treating a case of ulcers in C-1 as compared with treatment of ulcers in C-3. Oral medication given via stomach tube may take a long time to reach C-3.

Nourishment and fluid must be provided as appropriate. An IV catheter may be placed to hydrate the llama and give minimal nourishment. Electrolyte imbalance is not a major factor in gastric ulceration.

Gastric Overload

Although it is an uncommon disease of camelids, when gastric overload is suspected, it must be dealt with immediately. Other names for this condition include acute gastric impaction, C-1 acidosis, grain overload, and engorgement toxemia.

ETIOLOGY. In one instance, a llama broke into a feed room and consumed a large quantity of grain.[a] The

Table 13.8. Differential peritoneal aspirate parameters in acute abdominal disease in llamas.

	Normal	*C-3 perforation*	*Strangulated jejunum*	*Ruptured bladder*
Gross appearance	Clear	Cloudy	Hemorrhagic	Cloudy
Protein g/dl	<2.0	4.2	3.8	<2.0
Specific gravity	1.0110	1.0185	1.0178	1.024
Leukocytes/µl	1–10	9700	1950	1–10
Potassium mmol/L	4.3 ± 0.78	4.3	4.3	185–400

Table 13.9. Differential hemograms.

	Normal	*C-3 perforation*	*Colonic perforation*	*Strangulated jejunum*	*Ruptured bladder*
Erythrocytes × 10/µl	12 ± 2	16	20	19	18
Hematocrit, %	33 ± 6	42	49	46	51
Fibrinogen, mg/dl	327 ± 96	100	800	300	700
Leukocytes/µl	14,000 ± 3,000	7,600	4,300	3,000	11,100
Metamyelocytes	0%	0	387	90	1,776
Bands	0–4%	3,192	645	930	3,330
Neutrophils	56–87%	2,356	989	690	1,554
Lymphocytes	3–22%	1,520	1,634	960	3,330

Table 13.10. Differential blood chemistry.

	Normal	*Stomach perforation*	*Strangulated jejunum*	*Ruptured bladder*
Blood urea nitrogen mg/dl	22 ± 6	26	54	153
Sodium mmol/L	150 ± 3	152	155	
Potassium mmol/L	24 ± 0.78	3.3	2.7	
Chloride mmol/L	114	113	111	
Creatinine mg/dl				12.5

Table 13.11. Differential diagnosis of acute abdominal diseases of llamas.

Definitive diagnostic parameters	*Stomach atony*	*Intestinal obstruction*	*Stomach ulcers without perforation*	*Perforated ulcers*	*Retained meconium*	*Urinary obstruction without bladder rupture*	*Bladder rupture*
Pain	–	+ + + +	+	+ + early + late	+ + +	+ + + +	±
Gastric atony	+ + + +	+ + + +	+ +	+ + + +	+	+	+
Shock	–	– early + late	–	+ + + +	–	–	+ +
Abdominocentesis	Normal	Transudate, hemorrhagic, protein 3.8 g/dl leukocytes +	Normal	Exudate cloudy may have intestinal contents, protein 4.2 gm/dl, leukocytes +	Normal	Normal	Protein <2, few leukocytes, potassium 100 mmol/L
Hemogram	Normal	Leukocytes, left shift	Normal	Leukocytes, degenerative left shift	Normal	Normal or stress response	Leukocytes normal or elevated, left shift
Blood chemistry							
BUN mg/dl	Normal	54	Normal	Normal	Normal	Normal	100 +
Potassium mmol/L	Normal	Decreased	Normal	Decreased	Normal		Decreased

clinical signs were similar to those of a cow suffering from bloat.

The overingestion of highly fermentable carbohydrates, usually concentrates, may lead to lactic acidosis, dehydration, and depression. It is also possible to overingest highly fermentable proteinaceous feeds, such as soybean derivatives, resulting in excessive ammonium ion production, which causes alkalosis. Alkalosis produces signs of excitement and hyperesthesia. The latter syndrome has not yet been described in camelids, but in the right circumstances, it could occur.

Pathogenesis. Diagnosis, therapy, and management of gastric overload require an understanding of the pathogenesis. The following discussion is based on extrapolation from studies on cattle. The normal microflora of C-1 ferments carbohydrates, with short-chain VFA (acetic, propionic, butyric) as end products, which are absorbed and used for energy. If the llama consumes an overabundance of readily digestible carbohydrates, the fermentation pattern changes, a new microflora emerges as predominant, and lactic acid and other long-chain fatty acids are produced.

Lactic, formic, valeric, and succinic acids change the osmolarity of C-1, resulting in fluid being drawn in from the circulatory system and other tissues. Thirst also results in accumulation of excessive water in C-1. The pH of C-1 may drop to 4, and there is gastric stasis and destruction of the normal C-1 microflora. The excess fluid drawn into the GI tract produces a profuse, watery diarrhea. The acidic condition in C-1 also causes gastritis and permits bacterial invasion of the wall of C-1. A bacteremia may develop that may lead to development of abscesses on the liver.

Signs. Signs of toxic indigestion may not develop until twelve to thirty-six hours after engorgement. The timing depends on the nature of the food ingested and the amount consumed. This delay is important in the management of known engorgement, since it allows time to attempt to alleviate the problem. Signs of engorgement include weakness, incoordination, depression, anorexia, C-1 atony, colic (grunting, grinding teeth), abdominal distention, dehydration, a fetid diarrhea, and recumbency. The respiratory rate increases because of the acidosis. Without treatment, death may occur in twenty-four to forty-eight hours. Some of the signs will persist during recovery. The mucosa of C-1 has received a severe insult, and time is required to relieve the gastritis. Camelids are not likely to develop laminitis as in equines, but the other effects of grain overload occur in camelids as in ruminants and equines.

Diagnosis. The history of access to concentrates is vital to the diagnosis; however, it may be difficult to establish the amount consumed. Clinical signs are valuable in making a diagnosis. The ingesta from C-1 should not test below pH 5.5. If the pH of the first compartment of the stomach of a camelid drops below 5.5 as a result of either ketoacidosis or grain overload, microorganisms cease to grow, necessitating transfaunation to re-establish C-1 function.

Treatment. The administration of antacids to correct the acidosis may suffice in mild cases. Magnesium carbonate and magnesium hydroxide may be given in large volumes of warm water so as to disperse throughout C-1; 3 L may be used in an adult llama. The dose of the magnesium powder should be 1 g/kg in early acute cases and 0.5 g/kg if the stomach has been evacuated. Additional therapy should include thiamine HCl, IV fluids, antibiotics, and flunixin meglumine.

In consideration of the value of llamas and alpacas, a gastrotomy to remove the grain from the stomach may be prudent. The technique is the same as for rumenotomy. Parenteral therapy is also indicated to correct both acidosis and dehydration.

Gastric Dilation (Bloat)

All forms of overproduction of gas or eructation failure are rare in camelids, but do occur (Figures 13.58A, 13.58B). The reasons for this are not understood. Obstructive lesions of the esophagus or neuromuscular disorders could theoretically cause gastric dilation, but such a case has not yet been reported. If the problem should arise, it may be handled as if in a ruminant.

Intestinal Disorders

Ailments of the intestinal tract of camelids are similar in etiology to those of ruminants. However, the camelid's response to colicky pain is more like that of the equine. Conditions that have been diagnosed in llamas include enteritis, obstruction, ulceration with and without perforation, colitis, proctitis, rectal prolapse, and GI parasitism. Fiber optic endoscopy has become an important diagnostic tool. Disorders of the nasal cavity, nasopharynx, larynx, trachea, mouth, oropharynx, and esophagus are amenable to endoscopic examination. Laparoscopy has been used extensively to evaluate the reproductive tract of small animals, but recently, its use has been described for evaluating the abdominal viscera in the llama.[92]

Enteritis

The inflammatory response of the camelid intestinal mucosa varies from hyperemic to catarrhal and hemorrhagic, progressing to necrosis of all the layers of the intestinal wall. Specific names may be given in reference to the specific area that is involved (duodenitis, jejunitis, ileitis, cecitis [typhlitis], colitis, and proctitis).

Etiology. Enteritis may be primary or secondary to many other diseases. Infectious diseases that may involve enteritis include salmonellosis, colibacillosis, paratuberculosis, tuberculosis, rinderpest, and clostridial enterotoxemias.

Numerous parasitisms cause enteritis (Chapter 8). All of the agents causing diarrhea may produce enteritis.

Secondary enteritis may occur with any of the obstructive lesions and also with septic metritis and mastitis. Proctitis may result from a too vigorous rectal examination. The ultimate trauma associated with rectal abuse is a perforated rectal wall.

Signs. The primary manifestation of enteritis is diarrhea. The nature of the feces aids in establishing the location and/or severity of the enteritis. The feces may have a fetid odor and contain excessive mucus, blood, undigested feed particles, and even shreds of sloughed mucosa.

Other signs of enteritis are dehydration, electrolyte imbalance, tenesmus, anorexia, colic, depression, stomach atony, plus or minus fever, and increased intestinal borborygmus. No intestinal sounds can be heard in the healthy llama. If sounds are present, fermentation has begun, resulting in a mixture of gas and fluid in the intestine.

Diagnosis. Clinical signs should determine diagnosis. An etiologic diagnosis requires cultures, fecal examination for cytology and parasitic ova, and hematology and blood chemistry evaluations. Colitis and proctitis may be evaluated by endoscopic examination. Colonic biopsies may be a diagnostic aid.

Treatment. In the management of enteritis, it is crucial to monitor and correct any fluid and/or electrolyte imbalance. It is particularly important to provide glucose and sodium. Acidosis is common, so giving sodium bicarbonate is usually necessary. Intestinal protectants are frequently given, but their value is questionable. These include kaolin, pectin, bismuth, and subsalicylate (Pepto-Bismol). Lactobacillus acidophilus therapy has not been found to be effective in ruminants. Broad-spectrum antibiotic therapy is indicated, not only to treat a primary infection, but to treat or prevent generalized secondary infections, since the permeability of the intestinal wall may be increased by enteritis, allowing invasion of the body by enteric bacteria.

Obstruction

Etiology. Either partial or complete obstruction of the intestine is a frequently seen clinical condition.[17,34,41,52] While all the possible causes of obstruction have not been observed or diagnosed in camelids, it is reasonable to assume that any of the types seen in ruminants may appear in camelids.

Specific causes already described in camelids include obstipation, impaction (sand, phytobezoars, enteroliths, fecaliths) (Figure 13.73 to 13.75), torsion (Figure 13.76), intussusception, compression from the gravid uterus, strangulation from hernias, neoplasia, abscessation, infarction, and congenital defects (atresia coli and atresia ani).[59]

Signs and Diagnosis. The signs of blockage vary with the location and completeness of the obstruction, the rapidity of development, the degree of enterotoxemia produced, the degree of vascular occlusion, and the production of gas. The major sign is colic, with little or no passage of feces.

When an obstruction involves interference with circulation, the ensuing sequence of events significantly alters the integrity of the bowel. In a partial vascular compression, the venous drainage is adversely affected.

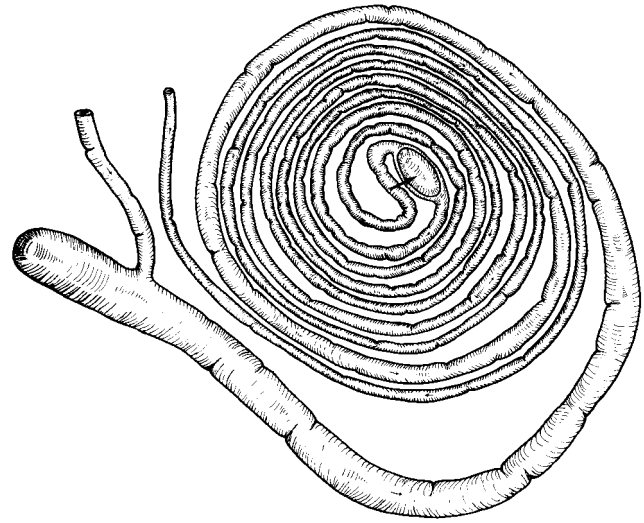

Figure 13.73. Diagram of spiral colon impaction in a llama.

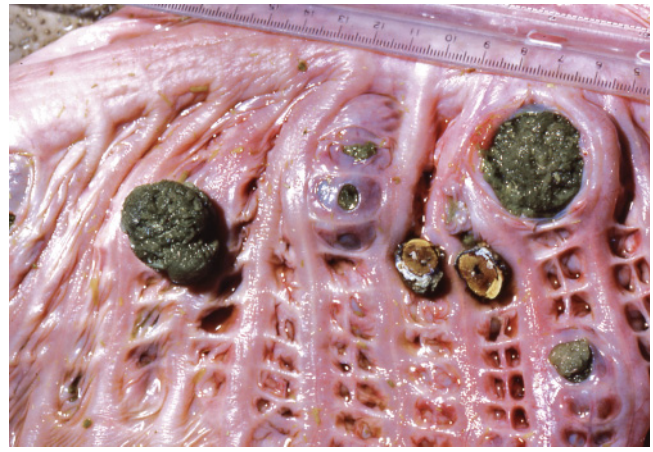

Figure 13.74. Gastroliths in the glandular saccules of C-1 in a llama.

Figure 13.75. Gastroliths removed from the glandular saccules of C-1 in a llama.

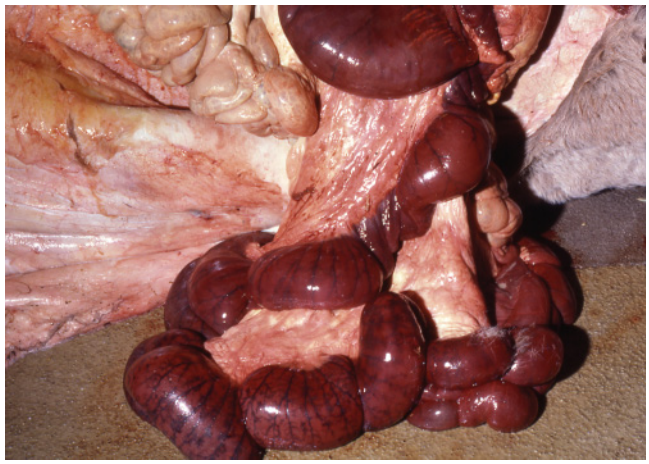

Figure 13.76. Lateral radiograph of gastroliths in situ.

Arterial blood continues to flow into the area, causing edema. If compression is complete, there will be tissue anoxia and increased capillary permeability, followed by intramural and mucosal hemorrhage. This is accompanied by edema and loss of the integrity of the epithelium, which allows bacterial invasion of the intestinal wall and, ultimately, bacteremia and peritonitis. Ischemia becomes complete and necrosis occurs. Tissue break-down products from the necrosis may be absorbed from the peritoneal cavity and contribute to cardiovascular collapse (shock).

This is an important clinical entity, characterized by a syndrome called the acute abdomen; therefore, four case reports are presented to illustrate the signs, diagnostic procedures, and management of these cases.[12]

Case 1. Strangulated jejunum

History: The patient was a three-year-old pregnant female. Thirty-six hours before admission, she was noticed to be depressed and anorectic. She was generally recumbent, with her head pulled back over the side. This clinical sign should not be given any specific clinical importance, since it is the usual position assumed by a weak or sick llama. When encouraged to stand, she did so reluctantly and was somewhat ataxic. The owner reported that she frequently groaned. She refused water for twenty-four hours. Normal fecal pellets were excreted up to four hours before admission. Another female llama in this herd had died of a perforated gastric ulcer a few months previously.

Signs: When presented, the llama was recumbent and refused to rise. She was depressed, but respiration appeared to be normal. Rectal temperature was 36.1°C (97°F) and the heart rate 132 per minute. GI motility was absent. Mucous membranes of the mouth were pale.

The llama was in sternal recumbency most of this time. Intermittently, she would roll to lateral recumbency and throw her head back into opisthotonos. If stimulated, she would roll back up to the sternal position but was reluctant to hold her head up for more than a minute. Otherwise, the head and neck would curve around to the side. Periodically, the llama would struggle to her feet and wander around the stall in an ataxic manner. The head was carried low while standing. Then she would flop back into sternal recumbency.

Diagnosis: A standard battery of procedures was performed and the results tabulated (Tables 13.7 to 13.10).

Management: Therapy for shock was instituted, and on the basis of the results of abdominocentesis and the hemogram, an exploratory laparotomy was begun. The llama died during preparation for surgery.

Necropsy: Serosanguineous fluid was found in both peritoneal and pericardial sacs. The omental bursa had incarcerated approximately 2 L of fluid, which also contained floating specks of fibrin. Tags of fibrin were loosely adhered to the surface of C-1.

A 0.5-m segment of the proximal jejunum had herniated through the omental foramen and had become incarcerated and strangulated. The intestine was hemorrhagic and necrotic, but it had not ruptured. The colon was congested but contained normal fecal pellets.

Case 2. Perforated gastric ulcer

History: The llama was a two-year-old male kept in a dry pasture with other llamas. On the morning of admission he had refused to get up, groaning as if in pain. The abdomen was distended.

Signs: Rectal temperature was 37.2°C (99°F); heart rate, 120 per minute; and respiratory rate, 50 per

minute. Mucous membranes were cyanotic, and capillary refill time was prolonged. There was no gastroenteric motility. The llama was unable or unwilling to stand.

Diagnosis: The high protein content (4.2 g/dl) and cloudy appearance of the abdominal aspirate were indicative of an acute abdominal disorder. Therapy for shock was instituted and an exploratory laparotomy was begun. Upon encountering GI contents in the peritoneal cavity, the llama was euthanized.

Necropsy: A perforating ulcer was found in C-3, accompanied by a generalized peritonitis. The ulcer was located 7 cm craniad from the pylorus. The serosal opening was approximately 4 mm, while on the mucosa there was a 10-mm craterous ulcer, with the surrounding mucosa thickened and hyperemic.

The thickened mucosa surrounding the ulcer was hyperplastic and was composed of a type of acid-secreting cells. Multifocal infiltrations of eosinophils and mononuclear cells were seen in the lamina propria. Excessive mucus and cellular debris covered the mucosal surface, and in this layer were embedded several adult trichostrongylid nematodes. Larval nematodes were noted in glands of the mucosa. The parasites were identified as *Ostertagia* spp. The remaining mucosa of C-3 was chronically inflamed and atrophic.

Case 3. Spiral colon impaction[7]

History: An adult female llama was noticed by a caretaker to refuse feed one evening, and since she was alone in an enclosure, the caretaker also noticed she was not passing feces.[44] Treatment with milk of magnesia and Banamine was not successful. Depression deepened, and the llama was admitted to the veterinary medical teaching hospital.

Signs: The llama was reluctant to rise and, when forced, would move slowly. The rectal temperature was normal, as were heart and respiratory rates. Mucous membranes were normal. Abnormalities noted were a decrease in stomach motility, lack of feces, and depression. There were no overt signs of colic.

Hemogram and serum chemistry parameters were normal. Abdominocentesis was performed, but no fluid was obtained. Standard symptomatic treatment was begun with Banamine and cimetidine for three days. No feces had been passed for six or seven days. On day six, rumen contents (1 L) from a cow were given via stomach tube along with magnesium hydroxide. Another hemogram and chemistry panel was performed. All parameters remained normal.

The llama continued to be anorectic. She did drink water, and it was noted that she had urinated. The temperature remained normal.

She spent most of the time in sternal recumbency. She would get up if harassed by treatment regimens but was reluctant to move around. Her abdomen was somewhat tense, and a general feeling of abdominal discomfort was noted.

Therapy: Seven days after the illness was noted, an exploratory laparotomy was performed (Chapter 6). The small intestine and the proximal large intestine were distended with gas and ingesta. Gas was withdrawn from the distended intestine with a 16-gauge needle attached to an IV administration set. The terminal colon was empty of fecal pellets. The color of the viscera was normal. A small, hard mass (7×3 cm) was located within one of the loops of the spiral colon (Figure 13.73). The segment of intestine could not be visualized without incising through a layer of mesentery. The mass was exteriorized and packed off and an enterotomy performed.

The mass was not too large to have passed normally along the intestine. Why such a mass will impact is difficult to understand. It was composed primarily of dry, matted food particles, including a few large strands of straw, and was enveloped in a layer of mucus. Apparently, the consistency of the mass did not stimulate normal peristalsis but rather induced spasticity of the intestine on both sides of the mass. This phenomenon has also been seen in the horse and the hippopotamus. There was no evidence of ischemic necrosis of the intestinal wall as sometimes occurs in such cases.

The llama began to eat the day following surgery. Loose fecal material was passed within twelve hours, and within forty-eight hours pellets were being passed. Trimethoprim sulfa was administered daily for four days. The llama was discharged one week after admission.

Case 4. Perforating ulcer of the colon

History: The patient was a one-and-a-half-year-old male kept with other llamas in a dry pasture. He had been ill for five days prior to admission. He was colicky, as evidenced by restlessness, continual recumbency, and vocalization.

Signs: Groaning was marked. Mucous membranes were cyanotic.

Diagnosis: Ingesta in the abdominal fluid indicated a grave prognosis, and the llama was euthanized. Laboratory parameters are listed in Tables 13.7 to 13.9.

Necropsy: Fibrinous adhesions were present over the serosal surfaces. Fecal material coated much of the intestine in the caudal abdomen, including the spiral colon. A thick-walled sacculation containing formed feces was found on the surface of the spiral colon. A 2-cm perforation was present just caudad to the sacculation. Another outpouching of the colonic wall was found in the inflamed area.

The sacculation was covered with a serosal membrane that was thickened by inflamed fibrovascular tissue. It is supposed that a previous perforation had penetrated through the mucosal and muscle layers of the colon and had then dilated but not ruptured the serosa. Fecal material had accumulated in the sacculation.

The laboratory data for this and the preceding cases, and a case of ruptured bladder are presented in Table 3.10. It is important to remember that an etiologic diagnosis of any of the acute abdominal diseases cannot be made on the basis of clinical signs alone. The results of additional diagnostic procedures must be analyzed. These should include abdominocentesis and hemogram and blood chemistry evaluations.

Treatment. Surgery is often required for therapy for many of the diseases causing the syndrome of acute abdomen. The key is early diagnosis. Exploratory surgery and supportive therapy should not be delayed in order to establish the precise location of the lesion. The circulatory, electrolyte, and toxemic changes associated with obstructive lesions must be alleviated immediately. If the bowel perforates, death is inevitable unless the omentum successfully walls off the inflammatory response. The inability to conduct a thorough rectal examination precludes obtaining the diagnostic information that would be available in making a diagnosis in a cow or horse. In the camelid, such information can be obtained only by exploratory surgery.

Supportive therapy may be supplied by cannulation of the jugular vein for administration of appropriate fluids. See Chapter 6 for details of the surgery.

Recommended procedures for conducting a complete physical examination of a camelid with a suspected abdominal ailment are outlined in Tables 13.7 to 13.11.

Ulceration

Etiology. Ulceration of the intestinal mucosa may follow any severe enteritis and develop at any location from the duodenum to the rectum. The most common site is the small colon within the spiral colon. In many cases no indication of the cause can be determined, even at necropsy.

A unique ulcer is that seen resulting from ischemic necrosis caused by an intestinal spasm around an impaction of dried fecal matter. Multiple ulcers may develop in such a case because the intestine may temporarily relax sufficiently for the impacted mass to move on, only to spasm again. This is sometimes seen in small-colon impactions in horses, when chemotherapy results in temporary relaxation of the muscles. Such ulcers frequently necrose through the entire wall, perforating the intestine.

Diagnosis. It is extremely difficult to diagnose a primary ulcer. Elimination of other intestinal disorders should precede the tentative diagnosis, but an antemortem diagnosis of intestinal ulceration is always questionable.

Rectal Prolapse

Protrusion of the mucous membrane or the entire rectum through the anus is not common in camelids, but it has been reported.

Etiology. The most common cause is tenesmus, associated with prolonged, severe diarrhea. A mild protrusion, lasting only a few minutes, may be observed following defecation. A slight protrusion of both the rectal and vulvar mucosae has been noted in females near term, especially when lying down; pressure from the large fetus pushes organs into the pelvic canal.

Signs. A protrusion is obvious. The extent of the protrusion is important because of the potential disruption of the blood supply to the invaginated segment. A llama with a prolapse was referred to the Veterinary Medical Teaching Hospital at the University of California. When it arrived at the clinic, 6 feet of the colon had prolapsed, and an additional 3 feet protruded within the next few minutes. The llama was euthanized, and at necropsy it was seen that the mesentery and vascular supply had been stripped from the colon. Prior to that case the author would have said that a rectal prolapse was not a life-threatening emergency. Not so.

In less severe cases, the mucosa may be traumatized by laceration or abrasion from the tail or dirt when the animal is lying down. Edema is a likely result, and necrosis of the mucosa may occur.

Diagnosis. Obvious.

Treatment. This condition can be handled as it would be for a ruminant. The inciting cause should be corrected. The mucous membrane should be cleaned and the edema reduced by surrounding the protruding tissue with a towel soaked in warm water. Gentle pressure should be exerted on the towel and tissue. Previous recommendations have been to apply sucrose to the prolapse to draw fluid through the mucosa, which it does; however, investigations have demonstrated that the sugar granules actually traumatize the mucosa. When the swelling is reduced, the prolapsed tissue should be gently replaced, avoiding point source pressure. It may be necessary to use epidural anesthesia to allow replacement. It is essential that the invagination be completely corrected. An equine glass or plastic vaginal speculum inserted into the rectum works well. A purse-string suture may be placed if necessary, but it should be remembered that a camelid excretes pelletized feces and may need a proportionally larger orifice than a cow.

The camelid should be observed for recurrence of the problem or the build-up of feces behind a purse-string suture. Chronic rectal prolapse may require a submucous resection, similar to the procedure for cattle. If the protruding segment is necrotic, it may be amputated, using the same techniques as used in cattle (Chapter 6).

Rectal Laceration[87]

ETIOLOGY. A rectal tear may occur during breeding, but camelid copulation is not accompanied by as vigorous a pelvic thrust as is seen in cattle or horses. Prolonged dystocia, especially with manual manipulation, may traumatize the rectal wall[87] and allow either immediate or delayed rupture of the wall. The most likely cause of a rectal tear is an accident at the time of a rectal examination. The pelvis of even a large llama is small, and the size of the rectum is commensurate with the size of the animal. People with a glove size of over 7.5 will have difficulty performing a rectal examination in any but the largest animals.

Straining against an inserted hand and arm may be lessened by using epidural anesthesia or by mixing xylocaine with the lubricant. Even under epidural anesthesia, a colonic spasm may trap an arm caudad to the wrist. It is necessary to wait until the spasm relaxes before removing the arm to avoid splitting the wall of the colon.

DIAGNOSIS. The mucosal split may be felt at the time it is happening, but it is more likely that blood will be seen on the hand when it is withdrawn. Camelids are intermediate between mares and cows in terms of susceptibility to trauma of the rectal mucosa. As a result of tiny splits in the anal mucosa, it is not uncommon to see traces of blood on the back of the hand or wrist when finishing an examination. Copious quantities of blood in the palm of the hand or on the fingers should alert the clinician to the possibility of a mucosal tear.

If a laceration is suspected, the rectum should be carefully explored with a speculum, under epidural anesthesia, to determine the extent of the laceration. The rectum should not be explored with a digit, because this may enlarge a tear.

TREATMENT. Lacerations that penetrate deeper than the mucosa should be sutured.[89] This may be done through the anus, as described for diagnosis, if the laceration is caudal enough to be reached and there is sufficient space to manipulate the instruments. Tears occurring more craniad require suturing through a laparotomy. It is important to be aware that the pelvic reflection of the peritoneum extends approximately two-thirds the length of the pelvic canal, ventral to the uterus and bladder, but only half the length between the uterus and the rectum.

Gastrointestinal Concretions[90]

Anciently, people marveled at the "stones" that animals were thought to have swallowed. These stones took on the aura of magic in some cultures or protection against poisons in other cultures. Concretions are technically thought to be mineral in origin, but many consist of combinations of minerals, plant, and even animal tissue (hair).

"Concretion" derives from a Latin word meaning to grow together. "Bezoar" is an ancient word derived from the French word bézoard or the Arabic word bazahr or the Persian word padzahr—all meaning an antidote. (Gastrolith = stomach stone; enterolith = intestinal stone; phytobezoar = plant stone; and trichobezoar = hair ball.)

Anciently, poisoning was a common form of murder, and bezoars were thought to be antidotes to poisons. Royalty tried to protect themselves from poisoning by keeping bezoars about their persons. The Spanish invaders of the Altiplano (Pizarro, 1532) noticed that the native Incas ascribed magical and curative properties to the small stones that they found in the stomachs of all of the South American camelids. A historian reports that the superstitious Spaniards coveted these stones,[75] "which led to the mass slaughter of alpacas and llamas, and even the hunting of vicuña and guanacos for the stones. These prized stones were made into amulets to protect the wearer from evil and witchcraft. They are also used today for ritual purposes and are laid out on the tables containing the offerings to the Pachamama and the Apus. They are much sought after when an alpaca or llama is killed."

ETIOLOGY. Most of the concretions found in the stomach or intestine of camelids are the result of precipitation of consecutive layers of mineral matter (phosphates) around a central kernel of plant fiber or seed awn in saccules of the first compartment of the stomach. Not all of these spherical objects are made up of mineral matter. Three types of bezoars have been found in the stomach and intestines of camelids: (1) mineral stones (concretions, gastroliths, enteroliths), (2) compacted plant fiber balls (phytobezoars, fecaliths, impaction) and (3) hair balls (trichobezoars, zootrichobezoars). A fourth type may be a combination of one or more of the other three. The accumulation of sand in a segment of the intestine may be closely allied to the "stones" and cause similar signs of distress. All of these types of concretions have been detected in the digestive tracts of camelids, ruminants, horses, and humans.

Mineral stones are common in the saccules of the camelid stomach (Figures 13.74, 13.75, 13.77).[10,32,35,38,43,50,55,60,64,65,67,68] Plant fiber balls become major intestinal obstructions when they develop in the

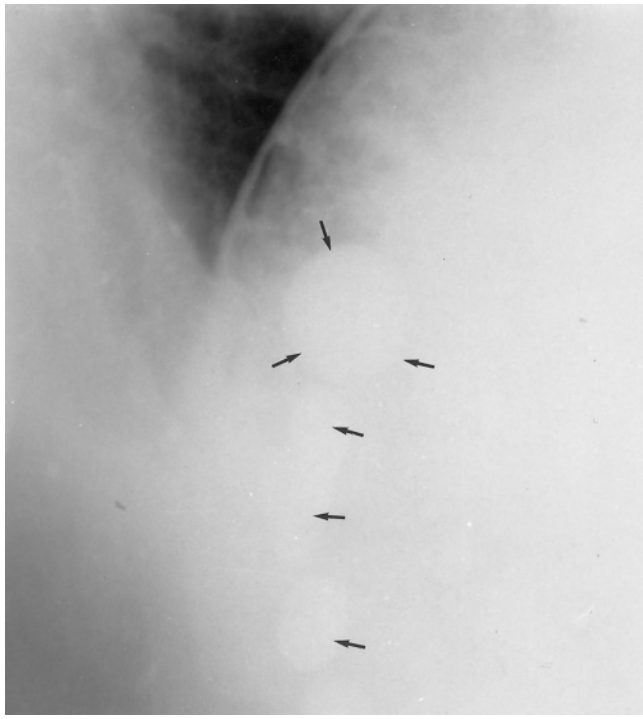

Figure 13.77. Intestinal torsion.

pellet-forming portion of the spiral colon. The diameter of the intestine decreases by five times in the spiral colon, and any large, firm object may completely occlude passage of normal pellets to the rectum. Cats and rabbits are notorious for developing hairballs because of their habit of licking their fur during grooming. Camelids seldom lick anything, so hairballs are rare, but they have been found in crias that develop a habit of chewing the fiber coat of their mothers. Most commonly this is observed in crias that suffer from nasal obstruction (choanal atresia) and have difficulty breathing while eating normally. Such crias stand with the head and neck extended and chew fiber to satiate hunger.

Sizes of the gastroliths vary from 1 mm (1/32 in.) in diameter to objects the size and shape of a small to medium chicken egg (2.5 to 4 cm × 4 to 5 cm [1 to 1.6 × 1.6 to 2 in.]). The surface of the gastrolith may be smooth or rough. The mineral composition of llama gastroliths is not known, but similar objects in other species have yielded struvite, magnesium ammonium phosphate, on analysis. Usually when a stone is cut through the middle, a kernel of plant fiber may be seen that served as the nucleus for precipitating the mineral from the normal stomach contents. The mechanism that triggers the process is unknown. Concretions may form directly in the intestine of horses and perhaps other species, but in camelids the likely source of intestinal concretions is the stomach.

The saccules of the first compartment of the camelid stomach may be an ideal place for concretions to form. The secretions from the glands of the saccules have been intensively studied by physiologists and nutritionists. An important component of the secretion is bicarbonate, which produces a slightly basic reaction. Bicarbonate is also secreted by the salivary glands, and concretions may form in the salivary ducts of horses and people. The basic reaction of the secretion within the saccule is conducive to the precipitation of phosphates and carbonates, forming concretions over a period of months or years.

The composition of GI enteroliths is more complex than that of uroliths. Preliminary studies show that the mineral composition includes various forms of calcium and phosphate. Factors that may contribute to the formation of phosphate concretions in the intestinal tract may include diets high in magnesium or high pH of the ingesta. Formation of trichobezoars may be associated with ingestion of plants that have tiny hairs on the surface of leaves or ingestion of hair (aberrant behavior or "pica" in crias).[90]

Signs. Concretions located in the saccules of C-1 cause no impairment of gastric function, as far as is known. The stones are seen as incidental findings on radiographs of the midventral abdomen or at necropsy. Other concretions may cause obstruction, ulceration, and perforation of the viscus wall.[11] The clinical signs of bezoar obstruction of the stomach or intestine are the same as for any other type of obstruction. Signs include anorexia, cessation of fecal passage, colic, refusal to drink, and depression.[29,35] Without surgical treatment, the animal may die in two to five days. A large, mineral-encrusted phytotrichobezoar caused obstruction of C-3 of the stomach of a zoo llama and ultimately killed it.

Diagnosis. Concretions in C-1 may be seen on a radiograph of the abdomen (Figure 13.77). Small concretions lying in a segment of the intestine overlaid by C-1 will be obscured. Many digestive tract disorders (impaction of C-3, ulceration of C-3, impaction of the intestines, torsion, and ileus) produce similar signs.[29] Furthermore, uterine torsion and obstructive urolithiasis produce the same signs. Any animal with colic and depression should be given a thorough examination.

Clinical Pathology. Hematologic and blood chemistry findings may vary from normal to those of an inflammatory syndrome in the face of peritonitis or severe gastritis. Dehydration is reflected in an elevated packed cell volume and hyperproteinemia. Evaluation of abdominal fluid obtained via abdominocentesis is a valuable adjunct, particularly in establishing the presence of peritonitis.

Pathology. At necropsy, the obstructing concretion is usually evident, except for phytobezoars located at the reflection of the spiral colon as it begins its centripetal coiling. Such obstructions may be easily overlooked

Figure 13.78. Acute peritonitis, with fibrin tags.

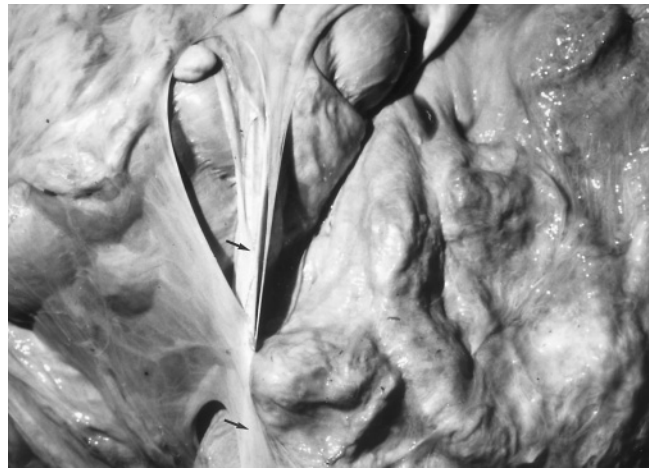

Figure 13.79. Chronic peritonitis, with fibrous bands that may incarcerate a loop of bowel.

because the intestine is hidden by the fibrous tissue that binds the spiral colon together.

THERAPY. Concretions in C-1 require no therapy. If the concretion obstructs the intestine, it must be removed surgically.[72,109] Recommended treatment for sand impaction is repeated doses of mineral oil or dioctyl sulfonate and neostigmine.

Peritonitis[91,113]

Peritonitis is inflammation of the serosal surface of the abdominal viscera or the wall of the abdomen. The inflammation may be focal or diffuse. Adhesions are a natural consequence of an inflamed serosa.

Etiology

There are numerous causes of peritonitis, among which are uterine tears, ruptured bladder, abscessation, and GI perforation.

Signs

Signs include colic, tense abdomen, stomach atony, ileus, weakness, plus or minus fever, diarrhea, painful movement, recumbency, and death in four to forty-eight hours.

Diagnosis

The collection of abdominal fluid by abdominocentesis is of major diagnostic help. All forms of peritonitis produce fluid, so if the lesion is focal or regional, the results of abdominocentesis may be negative. A diffuse peritonitis will cause a hemogram response.

At necropsy, peritonitis may be acute or chronic (Figures 13.78, 13.79) and must be differentiated from serous atrophy of fat.

Treatment

The prognosis for diffuse peritonitis is grave, especially if accompanied by perforation of the bowel. When that diagnosis is made, the camelid should be euthanized. Otherwise, administration of broad-spectrum antibiotics and supportive therapy, including IV fluids, are recommended.

Intraabdominal Hemorrhage

Etiology

Hemorrhage may result from the rupture of any major vessel in the abdominal cavity or from extensive capillary oozing. Common sources of hemorrhage are a ruptured liver or spleen from abdominal trauma. The cranial uterine artery is in the freely moveable broad ligament and may be subject to trauma, particularly late in pregnancy. The umbilical arteries may rupture too close to the body wall in the neonate and retract into the abdomen without closing. If the spermatic cord is under excessive tension at the time of transection of the cord with an emasculator, the artery may retract into the abdomen and continue bleeding.

Signs

The severity and rapidity of development vary with the rate of blood loss. The llama first becomes weak. The heart rate increases. There may be dyspnea and pallor of the mucous membranes and cool extremities. A hemogram is of little help until eight to ten hours have passed.

Treatment

Even though results of a test for packed cell volume do not aid in initial diagnosis, it should be done to provide a base for monitoring to evaluate the response to therapy. Also, the blood should be checked for possible clotting defects. If blood loss is thought to be caused by rupture of an abdominal vessel, it may be

necessary to investigate the site via laparotomy. In one case, the liver had been ruptured, and it was possible to correct this at surgery.

Blood transfusions are possible in camelids. Numerous blood types have been identified in the llama and alpaca, but any llama's blood can be transfused to another, once. After that, there is risk of an incompatibility reaction.

Neoplasia[6,88]

Neoplasia is discussed more fully in Chapter 9. Digestive tract neoplasms have been reported from the liver and stomach. Lymphosarcoma may have a generalized distribution in the body.

Hepatopathy[3,42,74,82]

Disorders of the liver occur in all species of animals. Periodic reports of hepatic disorders of South American camelids have appeared in the literature. During 1996 and 1997 several clusters of sick SACs associated with hepatic lipidosis and high mortality prompted a closer look at the pathophysiology of hepatic disorders in camelids. The problems are not fully understood as this volume goes to press, but some background information follows. More input from clinicians, physiologists, pathologists, microbiologists, and epidemiologists will be required to solve the challenges.

Hepatic Insufficiency

Hepatic insufficiency (HI, liver failure) is a syndrome common to all vertebrates. The liver has great reserves (80% of the hepatocytes must be damaged before the liver fails) and great regenerative ability. However, once the critical threshold has been reached, a process begins that may cause the death of the animal within thirty-six to forty-eight hours. Lesions of the liver may be toxic, degenerative, proliferative, or inflammatory.

Etiology. The causes of hepatic injury are many and include parasites (*Fasciola hepatica*, *Lamanema chavezi*[81]), infections (adenovirus, *Rhodococcus equi*, tuberculosis, coccidioidomycosis), metabolic disorders (hepatic lipidosis, hyperthermia), obstructive processes (fibrosis, cholelithiasis[94]), toxic agents (anesthetic agents, elemental phosphorus, halogenated hydrocarbons, copper,[51] pentachlorophenols, tannins, cresols, lupinosis, blue-green algae, alsike clover, lantana, mushrooms, cocklebur, sneezeweed[41]), and neoplasia[18,36,71] (adenoma, carcinoma of the biliary system, lymphosarcoma). Additional causes include hematogenous abscesses, gastritis (C-1), septic metritis and mastitis, peritonitis, rupture of the liver, malnutrition (cobalt deficiency, selenium deficiency[19]), and mycotoxins (sporidesmin, aflatoxin). Portal hypertension caused by cardiac insufficiency also causes decreased hepatic function.

Signs. The syndrome produced by HI is essentially the same, regardless of the initial cause. There are slight species differences. The cardinal signs of HI are depression, recumbency, and weight loss. Additional signs that may be seen at certain stages of the syndrome or in various species include anorexia, decreased growth rate, unthrifty appearance, icterus, photosensitization, hemorrhage, hepatic encephalopathy (incoordination, aimless walking, head pressing, coma), tenesmus, rectal prolapse, hemoglobinuria, and diarrhea.

Hepatic insufficiency syndrome in an SAC resembles that seen in the ruminant rather than in the horse. In the horse the central nervous signs are more exaggerated, with violent pushing, ataxia, aimless walking, leaning on objects, and falling. Chewing at objects or attempting to grasp feed or a fence with the mouth opened in a yawn is also characteristic of hepatic insufficiency in the horse. All of these CNS manifestations should cause alarm in rabies-endemic areas.

The ruminant and camelid appear to be depressed. Coma, rather than violent, active signs, may be associated with hepatoencephalopathy. When a camelid becomes weakened and toxic, it will lie down and refuse to rise rather than stumble about and push against objects in its environment, although head pressing is seen.

Photosensitization is a sign seen in some hepatopathies. Normally, the liver degrades chlorophyll to phylloerythrin, which is further degraded to inactive substances. In hepatic insufficiency, the liver fails to decompose phylloerythrin, which then accumulates in the peripheral circulation. Phylloerythrin is a photodynamic agent. For an SAC to be photosensitized, nonpigmented skin must be exposed to the ultraviolet rays of sunshine. Heavily fibered areas of the body are protected. The clinical signs of photosensitization are similar to those of sunburn. Photosensitization is only one sign of hepatic insufficiency and is relatively insignificant in the overall management of the case. Placing SACs inside a barn prevents progressive skin damage from the effects of sunlight.

Hemoglobin, bile pigments, or both may be seen in the urine of animals suffering from hepatic insufficiency. Hemolysis is not of a magnitude to produce anemia.

Although icterus is a consistent sign associated with hepatic insufficiency, it may be absent in a small percentage of terminal cases and is rarely observed in simple hepatic lipidosis. Diseases other than primary hepatic disease, such as hemolytic conditions and obstructive intestinal disorders, also produce icterus.

Diagnosis. Signs are not pathognomonic. Routine hematology and serum biochemistry may be negative but also may be highly indicative. It is usually not possible to establish an etiologic diagnosis based on gross

necropsy findings. The liver may be large, small, soft, firm, yellowish, darkened, or mottled. Postmortem decomposition begins quickly in SACs if veterinarians do not cool the first organs to undergo autolysis. It is not uncommon for a snap diagnosis of hepatopathy to be changed after microscopic examination (Figures 13.80 to 13.82).

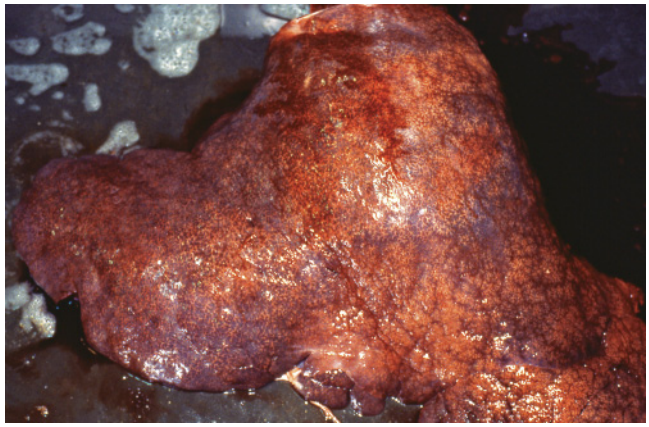

Figure 13.80. Hepatitis in a camel.

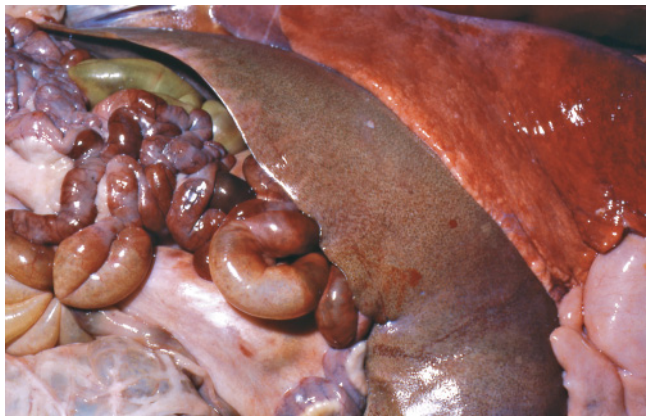

Figure 13.81. Hepatic necrosis in a llama.

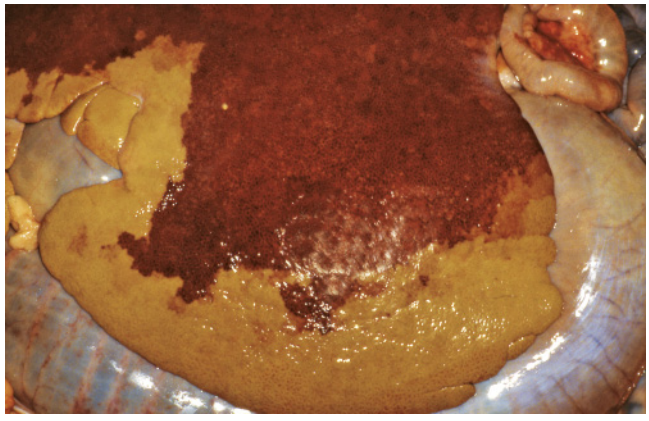

Figure 13.82. Hepatic lipidosis in a llama.

The cardinal signs of liver disease (depression, recumbency, weight loss) should more correctly be termed the cardinal signs of a sick SAC. Thus many diseases mimic the syndrome of hepatic insufficiency. Furthermore, the liver may be secondarily affected by pansystemic diseases (septicemia, enterotoxemia, endotoxemia). A differential diagnosis should include consideration of the following conditions: parasitism, dental disorders, gastric ulcers, partial obstruction of the gastrointestinal tract, or chronic wasting diseases (tuberculosis, Johne's disease, coccidioidomycosis, pneumonia, chronic nephritis, neoplasia).

Evaluation of clinical pathology parameters is a primary diagnostic tool.[81] It should be mentioned that studies to establish which of the enzymes are specific for hepatic injury in SACs have not been done. Extrapolation from ruminant data is currently being used. Enzyme changes are likely to be seen only when there is active necrosis. Urinalysis and liver biopsy may be definitive in establishing the etiology.

Hepatic Lipidosis[70,82,93,95]

Hepatic lipidosis is not a specific disease but rather the end result of one or more of the metabolic processes:

1. Increased mobilization of depot fat
2. A decreased rate of oxidation of mobilized fat
3. An increased formation of fat
4. A reduced rate of removal from the liver

Accumulation of lipids (primarily triglycerides) in hepatocytes is caused by mild injury. More severe injury results in necrosis.

Etiology. Hepatic lipidosis occurs in cattle, sheep, cats, dogs, and, to a lesser extent, horses. The mechanisms of development vary from species to species. In order to begin to understand the cause of hepatic lipidosis in South American camelids, it is necessary to know how they subsist in their native countries. In South America, these animals are never fed concentrates or supplements and rarely are fed cured hay. They feed on native grasses and forbs. Llamas/alpacas are concentrated in the Altiplano, which is an area of broad, high-altitude valleys and plateaus at elevations over 12,500 ft (3,800 m). The climate of this area consists of a long dry season and a short wet season, with 75% of the rainfall occurring from December through March. The dry season is from May through October.

The growing season (wet) is characterized by low temperatures (300+ nights of frost per year) and intense solar irradiation. Daytime temperatures reach a maximum of 18.3°C (65°F) with nighttime temperatures of –12°C (10°F). Available forage varies from the wet to dry seasons, but the animals adapt to the "feast and famine" routine by depositing layers of fat in the

subcutaneous, muscular, and retroperitoneal tissue during the wet season and mobilizing these reserves in lean times. This is an evolutionary adaptation to good foraging followed by seasonally deplorable feeding conditions, and this adaptation is usually completely forgotten by North American managers. It is generally assumed by owners that SACs consume only enough to satisfy their requirements. This is not true, and some animals may become obese because of overfeeding.

The generic causes of hepatic lipidosis include starvation, low-protein diet, high-fat diet, deficiency of insulin, deficiency of thyroxine, deficiency of pituitary hormones, and stress. Toxins may also initiate or contribute to lipidosis (bacterial toxins, phytotoxins, chemical toxins). Prolonged hypoxia or anoxic episodes may be involved (hypovolemic shock, hyperthermia, severe anemia).

SIGNS. Signs may not point directly to liver malfunction. Icterus is not a sign of uncomplicated lipidosis. Approximately thirty pregnant llamas died over a period of two months on one farm. The first signs noted were blindness and staggering. The animals became recumbent but continued to eat and drink up to the time of death. Most of the llamas were in fair to good condition at the onset of signs, but if they lived for days to weeks, they gradually lost weight. Later, in the course of the outbreak, abortion became a factor in the syndrome. Forty-five females aborted; eight were among the dead llamas. Three stillbirth deliveries occurred.

Clinical pathology findings in many of the cases were not helpful. A few llamas had hyperlipemia.[2] Table 13.11 provides normal values for selected biochemical parameters and compares levels found in other species. Table 13.12 lists values for animals with hyperlipemia. Glucose levels were generally elevated (300 to 650 mg/dl), but this is true with any SAC that is anorectic. The CNS signs are explainable on the basis of hepatoencephalopathy generally thought to be caused by increased blood ammonia levels. Ammonia levels were measured in one of the above llamas and found to be elevated (550 µg/ml [normal, <5 µg/ml]). The only consistent lesion found at necropsy was hepatic lipidosis and renal necrosis. If renal function and hepatic function were diminished, the prognosis was grave. Late in the course of the disease outbreak, hypoinsulinemia was considered. Histopathology of the pancreas revealed a paucity of β cells. Glucose tolerance tests on a couple of animals resulted in a noninsulin-responsive flat curve.

Table 13.12. Selected biochemical parameters and blood gases from SACs with hyperlipemia.

Parameter	*Alpaca*	*Llama*
Glucose mg/dl	436	635
Creatinine mg/dl	6.3	4.8
S urea N mg/dl	27	132
Potassium mEq/L	2.6	1.3
Phosphate mg/dl	0.5	1.3
Triglyceride mg/dl	1564	5658
Cholesterol mg/dl	193	297
AST U/L	1331	888
SDH U/L	37	
GGT U/L	217	
Blood pH	7.36	
PO_2 mmHg	30	
PCO_2 mmHg	30	
Bicarbonate mEq/L	16.9	
Base excess mEq/L	–6.5	

TREATMENT.[95] The prognosis for acute hepatic necrosis is unfavorable. In mild cases, the therapeutic rationale is to correct any dietary deficiency and electrolyte imbalance and to follow with the administration of glucose. Vitamin B complex is also indicated. Broad-spectrum antibiotic therapy is indicated because secondary bacterial infection is frequently seen in any hepatopathy. Recent studies indicate that hyperglycemia and hypoinsulinemia are common in the hepatic lipidosis syndrome. Parenteral insulin therapy (20 to 50 IU daily for three days) has been beneficial in the early stages of hepatic lipidosis. Studies in cattle found sodium propionate, niacin, and nicotinic acid had beneficial effects.

Pathophysiology of Hepatic Disorders

The following discussion attempts to explain the pathogenesis of the primary signs and conditions associated with HI.

Anorexia (inappetence, reduced feed intake): An SAC may be unable to eat because of mechanical interference with chewing or swallowing (temporomandibular arthritis, oral or pharyngeal abscesses, esophageal obstruction). Pain may prevent ingestion of feed (dental, stomatitis, glossitis).

An SAC may lack a desire to eat. A major factor here may be hypokalemia. Additional factors include gastritis (colic), CNS deficit, CNS injury, septicemia, pyrexia, psychologic ostracism, and unsuitable feed.

Hepatic encephalopathy (hyperammonemia): Hepatic encephalopathy (hepatic coma) is a common terminal syndrome associated with hepatic insufficiency. The precise mechanisms are not known, but elevated levels of ammonia are characteristic. Ammonia is absorbed from the gut into the portal system and carried to the liver, where it is converted to urea. Urea is commonly recycled in SACs, as in ruminants, to serve as a nutrient for forestomach microorganisms. Consult the references if more detail is desired on the biochemistry of urea metabolism. If the liver is not functioning,

ammonia builds up in the circulatory system, ultimately contributing to the hepatic encephalopathy syndrome.

Signs in SACs include depression, weakness, stumbling, and head pressing. These are all signs of the hepatic insufficiency syndrome.

Weight loss: The primary cause of weight loss is reduced caloric intake, which may in turn be caused by anorexia or dental disorders. Other causes for weight loss include too rapid a passage of ingesta (diarrhea), malabsorption, parasitism, chronic wasting diseases (tuberculosis, Johne's disease, coccidioidomycosis), chronic depression, excessive muscular activity, disuse atrophy, hypoproteinemia, and neurogenic muscular atrophy.

Clinical Pathology

Hypokalemia: Potassium (K^+) is not stored in the body. There must be a constant intake of K^+. Anorexia results in a low K^+ intake, especially if drinking continues. Vomition and diarrhea contribute to a loss of K^+. Other causes for hypokalemia include prolonged diuretic therapy, prolonged corticosteroid therapy, and alkalosis. The normal serum level of potassium in SACs ranges from 3.6 to 6.2 mEq/L.

The signs of hypokalemia include muscular weakness (recumbency), depression, and altered cardiac function (tachycardia, P-wave depression).

Hyperglycemia: SACs may have a unique glucose metabolism. Normal glucose levels are 86 to 163 mg/dl, and elevated levels up to 300 mg/dl are common in any SAC that is anorectic. Glucose is not the primary energy source in SACs, as is also the case with ruminants. A little glucose may be stored as glycogen in the liver, but this is quickly used up during high-energy expenditure episodes. Volatile fatty acids and ketone bodies are the primary energy sources (80%) in ruminants and presumably in SACs as well. Glucose is required for energy metabolism in the brain, erythrocytes, leukocytes, renal medulla, and peripheral nerves.

The pathogenesis of hyperglycemia associated with anorexia may be explained as follows. When an animal becomes anorectic, metabolism is changed to mobilize fat for energy. A hormone-sensitive lipase increases lipolysis, which in turn frees glycerol, which is then converted to glucose. Hormone-sensitive lipase is activated by epinephrine and norepinephrine (excitement, sympathetic stimulation, heavy exercise), glucocorticoids (from the adrenal), and corticotrophin (from the anterior pituitary). Thus, any disease with stress as an accompanying factor causes hyperglycemia.

Ketosis: Ketosis is a metabolic disorder in which the level of ketone bodies (acetone, acetoacetic acid, β hydroxybutyric acid) in body fluid is elevated. The mechanism of action and syndromes produced vary from species to species. Ketosis is reported in SACs, but mechanisms have not been studied.

The syndrome in dairy cattle may be primary—a result of decreased energy intake—or secondary to other disease processes (retained placenta, metritis, hardware disease). The signs of dairy cattle ketosis include gauntness, dull appearance, inappetence, hindquarter incoordination, occasional excitability, decreased milk production, weight loss, and an acetone odor to the breath.

The basic mechanism is an energy deficit in early, heavy lactation that causes the mobilization of fat and the production of free fatty acids (FFA). The FFA are changed to ketone bodies in the liver. Ketone bodies are excreted in urine and milk, and excessive amounts accumulate in the blood. Low glucose + low insulin + lipase yields elevated cyclic AMP, which acts on hepatocytes and fat stores to release lipids. There is an overall increase in hepatic lipids resulting in hepatic lipidosis, a common condition in ketosis.

Normal blood ketone body levels in cows are acetone(0.0 to 10 mg/dl); acetoacetate (0.0 to 1.1 mg/dl); and β hydroxybutyrate (0.0 to 9 mg/dl).

The best management for cattle ketosis is prevention by the administration of oral propylene glycol (175 to 250 grams BID for five to ten days). Other therapies include sodium propionate, niacin, and nicotinic acid.

Pregnancy toxemia of sheep (lambing sickness, twin lamb disease, pregnancy ketosis) differs from cattle ketosis in that the syndrome is seen during the last month of multiple-fetus pregnancies. Signs include depression, lack of appetite, general weakness, and sometimes a fever because of concurrent infection. The basic mechanism seems to be low energy intake and stress. Glucose levels are down, ketone levels up, and hepatic lipidosis is marked. About the only effective treatment is to remove the fetuses.

Enzymology: The liver-specific enzymes of SACs have not been delineated. Enzymes found to be useful in ruminants are as follows: GGT (γ glutamyltransferase [normal in SACs 16 IU/L, range 3 to 28], the most useful enzyme for diagnosis of hepatic disorders in ruminants); SDH (sorbitol dehydrogenase [5 IU/L, range 0 to 10]); LDH (lactate dehydrogenase [356 IU/L, range 10 to 695]); and GOT (AST) (aspartate aminotransferase [289 IU/L, range 128 to 450]).

Elevation of hepatic enzymes is not likely in chronic hepatopathy. Release from the hepatocytes occurs when cells are necrosed.

Creatinine: Small quantities of creatinine are ingested from animal products. Most derives from the degeneration of creatine. Creatinine is freely filtered through the glomerulus. Any disease that damages the glomerulus causes a buildup of creatinine in the blood. This

is the most sensitive indicator of renal impairment in ruminants.

Volatile fatty acids (VFA): VFA include propionate (required for gluconeogenesis), butyrate, pyruvate, lactate, and acetate. Volatile fatty acids are absorbed from the stomach and small intestine into the portal system and transported to the liver, where they are metabolized to provide energy or are stored as triglycerides in the hepatocytes.

Urea metabolism: Urea is a product of protein metabolism. It is recycled in ruminants and camelids to serve as a source of nonprotein nitrogen for microbial synthesis.

$$CO\begin{matrix}NH_2\\NH_2\end{matrix} + H_2O = CO_2 + NH_3$$

$$NH_3 + \text{Forestomach bacteria} = \text{Bacterial protein}$$

Excessive NH_3 in the stomach and small intestine is absorbed and carried to the liver, where it is reconverted to urea and excreted from the body via the kidneys. Blood NH_3 concentration should be 0.1 to 0.2 mg/dl.

Energy: The signs of energy deficiency include weight loss, delayed puberty, diminished libido and estrous behavior, decreased fiber production, decreased immune function, and poor wound healing. Prolonged severe energy deficiency may result in death, caused by hypoglycemia and protein depletion.

Excessive energy intake leads to obesity, reduced fitness, and reduced reproductive efficiency in both males and females, and may lead to ketosis and hepatic lipidosis.

Protein: Signs of protein deficiency include diminished hair coat quality, decreased immunocompetence, and poor wound healing. These are similar to the signs of energy deficiency. In fact, in the face of energy deficiency, the body mobilizes body protein and metabolizes it for energy purposes.

No signs are associated with excessive protein intake, and there is no such thing as protein poisoning. However, unused protein is metabolized, yielding urea. Llamas on a high-protein diet may have elevated serum levels of urea nitrogen. Urea is excreted by the kidneys, so such llamas may drink more and urinate more than llamas consuming more appropriate levels of protein.

Pancreatitis is discussed in chapter 14.

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[a] S. Taylor, personal communication.

14

Endocrine System

ANATOMY AND PHYSIOLOGY

A few anatomic and physiologic studies describing the endocrine glands of camelids have been published.[1–23] Since the first edition of this book, significant work has been carried out on reproductive endocrinology (Chapter 17).

Pituitary Gland

The pituitary gland of camelids is not unique. It is located at the base of the brain in the pituitary fossa of the basic sphenoid bone, similar to the location in bovine. The gross anatomy, histology, and histochemistry of the gland have been studied.[12,14] The growth hormone of the alpaca has been isolated and characterized and found to be chemically similar to other mammalian growth hormones.[2] Unfortunately, this hormone has not been studied in a clinical setting to determine if it is involved in the failure to thrive syndrome.

Thyroid Gland

The paired thyroid glands are situated on the dorsolateral surface of the trachea. They are approximately 4 cm long and 2 cm wide and occupy the space from the cricoid cartilage of the larynx to the third or fourth tracheal ring. Blood is supplied by a cranial and caudal thyroid artery directly from the carotid artery.

Reference ranges for serum triiodothyronine (T_3) and thyroxine (T_4) in llamas have been established using radioimmunoassay techniques (Table 14.1).[10,18,19] These levels are higher by six to ten times than those reported for any other species in which values have been determined. There is considerable variation in serum T_3 and T_4 levels at different ages (Table 14.1). Serum T_3 levels are high in crias less than two days of age, increase slightly by day four, then decrease over the next ninety days to adult levels. Serum T_4 levels are highest at birth and slowly decrease over the next ninety days to adult levels.

What is the significance of the high T_3 and T_4 levels in camelids in general, but crias especially? The first part of the question has no answer, but the second may be answered by consideration of the biochemical physiology of the thyroid hormones. High neonatal serum T_3 and/or T_4 levels are reported in other species.[11] High thyroid hormone serum levels may be important for rapid development and function of organ systems including the nervous and musculoskeletal systems. Thyroid hormones play a major role in thermogenesis by their influence on brown fat.[11] Although catecholamines are the major stimuli for increased thermogenesis from brown fat, thyroxine inhibits monoamine oxidase degradation of catecholamines in brown fat and thus aids in thermogenesis.

Camelids are adapted to cool to cold climates, and the neonate cria is not licked dry or stimulated by licking by the dam. Therefore, the neonate camelid cria, more than any other species, may depend on high neonatal thyroid hormones to stimulate adequate thermogenesis and neuromuscular activity.[11] The clinical significance of these findings has not been evaluated.

Parathyroid Glands

The parathyroid glands are embedded in the caudal pole of each thyroid gland, as they are in other ungulates. No parahormone levels or disorders of parathyroid function have been reported.

Adrenal Glands

The right adrenal gland is elongated (1 × 4.5 cm) and lies medial and 34 cm craniad to the cranial pole of the right kidney. The left adrenal gland lies in

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Table 14.1. Comparison of T_3 and T_4 values from various studies.

Age	*McLaughlin et al. 1993*				*Smith et al. 1989*				*Fowler and Zinkl 1989*			
	T_3 ng/dl[a] U.S. units	*T_3 nmol/L SI[b] units*	*T_4 ng/dl[c] U.S. units*	*T_4 nmol/L SI units*	*T_3 ng/dl U.S. units*	*T_3 nmol/L SI units*	*T_4 ng/dl U.S. units*	*T_4 nmol/L SI units*	*T_3 ng/dl U.S. units*	*T_3 nmol/L SI units*	*T_4 ng/dl U.S. units*	*T_4 nmol/L SI units*
0–2 d	513 (280–750)	7.7 (4.2–11.3)	61.0 (39.2–82.8)	785 (505–1066)	—	—	—	—	—	—	—	—
3–4 d	554 (400–700)	8.3 (6.0–10.5)	56.3 (50.2–62.4)	725 (646–803)	—	—	—	—	—	—	—	—
5–6 d	543 (470–620)	8.1 (7.1–9.3)	35.0 (19.1–51.0)	450 (246–656)	—	—	—	—	—	—	—	—
7–8 d	422 (380–460)	6.3 (5.7–6.9)	42.1 (19.3–64.9)	542 (248–835)	—	—	—	—	—	—	—	—
9–10 d	427 (290–560)	6.4 (4.4–8.4)	28.9 (16.7–41.1)	372 (215–529)	—	—	—	—	—	—	—	—
11–30 d	327 (80–560)	4.9 (1.2–8.4)	20.6 (15.6–25.6)	265 (201–329)	—	—	—	—	(274–686)	(4.1–10.3)	(18–39)	(232–502)
31–90 d	156 (90–220)	2.3 (1.4–3.3)	14.5 (9.6–19.2)	187 (124–247)	—	—	—	—	(42.0–326)	(0.63–4.9)	(10.0–25.9)	(129–333)
<1 yr	—	—	—	—	—	—	—	—	(63.0–321)	(0.94–4.8)	(10.0–20.0)	(129–257)
Adult Y	—	—	—	—	145 (45–414)	2.2 (0.67–6.2)	12.4 (7.0–22.0)	160 (90–283)	(108–277)	(1.6–4.2)	(7.8–18.7)	(100–241)
Adult X	—	—	—	—	136 (45–412)	2.0 (0.67–6.2)	8.8 (4.1–18.9)	113 (52.7–243)	(70.0–197)	(1.1–2.9)	(12.1–19.8)	(156–255)
Pregnant X	—	—	—	—	145 (48–440)	2.2 (0.72–6.6)	10.0 (3.3–30.6)	129 (42–394)				

Note: These studies were all conducted using radioimmunoassay kits, but all the kits were from different manufacturers, hence the variable values.
[a] Triiodothyronine.
[b] SI = Système International.
[c] Thyroxine.

approximately the same position relative to the left kidney but has a squarish shape (2.5 × 3 cm and 1 cm thick). Camelid adrenal function has had little research attention.[15–17,20,21]

Pancreas

The pancreas is both an endocrine (insulin) and exocrine (digestive enzymes) gland. In SACs it lies caudal to the liver. It is a flattened gland, not well encapsulated in the duodenal mesentery. It is approximately 15 to 20 cm long and 3 to 6 cm wide. The pancreatic duct exits the cranial aspect of the gland and unites with the bile duct about 3 cm before it enters the intestine. Diabetes mellitus has been reported in camelids. Transient hyperglycemia, especially in neonates, has been treated with insulin.

Insulin production is the endocrine function of the pancreas. No studies on basal reference levels of insulin in different age groups and physiologic states have been reported. The status of diabetes melitensis in adult camelids is controversial. For more details refer to Chapter 13 (hepatic lipidosis and pancreatitis). A few clinicians use insulin in cases in which serum glucose levels are inordinately high (500 mg/dl). A California clinician with considerable llama experience has documented response to parenteral insulin in neonates with elevated glucose levels.[a]

Gonads

Gonadal anatomy and physiology are discussed in Chapter 17.

DISEASES

Diseases of the gonads are discussed in Chapter 17. Stress, as it relates to camelids, is discussed in Chapter 9.

Failure to Grow

Failure to grow or chronic weight loss is an important clinical syndrome in camelids. See Chapter 9 for a full discussion.

Pancreatitis[11]

The pancreas has both an endocrine function and produces enzymes necessary for digestion. Pancreatitis is uncommon in camelids. This may reflect failure in diagnosis or in documenting diagnoses, but pancreatic disease is also rare in cattle. Ongoing work with the hepatic lipidosis syndrome has revealed that in some cases there has been a depletion of the β cells in the pancreas. In one outbreak in the northeastern United States, a virus was isolated from the pancreas of affected llamas.[b] These animals had hyperglycemia, hypoinsulinemia, ketosis, and lipemia.

Diabetes Mellitus[1,4, 5,6,13,22]

Etiology

Only a few cases of diabetes mellitus have been reported in camelids. The precise etiology in camelids is unknown, but in other species causes include pancreatitis, obesity, infection (viral?), stress, estrus, and autoimmunity.

Signs

Signs include polydipsia, polyuria, hyperglycemia, glycosuria (200 mg/dl threshold), ketonemia, lipemia, ketoacidosis, ketonuria, and weight loss.

Diagnosis

Diagnosis of diabetes mellitus (DM) in simple-stomached animals is based on high fasting blood glucose, acidosis, hyponatremia, hypokalemia, and response to insulin. An intravenous glucose tolerance test (IVGTT) may be performed by administering 50% glucose intravenously at a dose of 0.5 g/kg body weight (BW) over a two- to three-minute period. Samples are collected at 0, five, fifteen, twenty-five, thirty-five, forty, and sixty minutes. In diabetes mellitus, glucose levels peak but the one-half-time return toward normal is prolonged (normal T 1/2 = 15 to 45 minutes, in DM T 1/2 = > 70 minutes). The serum insulin level for the cow is <5μ units per ml; the dog is 5.8 ± 1.6μ units per ml.

SACs normally show a higher serum glucose (100 to 200 mg/dl) than cattle (45 to 75 mg/dl). During many digestive diseases, glucose levels may elevate to 200 to 300 mg/dl. This may result in some erroneous diagnoses of diabetes.

Ketoacidosis is a common finding in DM in simple-stomached animals. Glucose levels above 200 mg/dl in the blood are excreted via the urine, causing a mobilization and utilization of glycogen stored in the liver. Replacement glucose is derived from protein breakdown and gluconeogenesis. The oxidation of fatty acids is accelerated with the production of ketone bodies that are acidic. An acetone odor may be detected on the breath. Excessive numbers of ketone bodies cause excretion of sodium and potassium in the urine. Excessive glucose causes osmotic diuresis, resulting in dehydration.

Electrolyte imbalance + ketoacidosis + dehydration = collapse, coma, and death

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[a] Dr. Robert Pollard, Sonora, Calif., personal communication, March 1994.

[b] Dr. Susan Stehman, Ithaca, N.Y., personal communication.

15

Hemic and Lymphatic Systems

ANATOMY AND PHYSIOLOGY

Numerous investigations on camelid blood have been carried out. The unique oval erythrocyte of camelids was first described more than 100 years ago, and ever since, researchers have been attempting to establish the role played by blood constituents in the adaptation of camels to life in a desert environment and of South American camelids (SACs) to life at high altitudes. Though a complete physiology of hemic systems is not reviewed here, facts relevant to evaluating a diseased state or to therapy are discussed.

HEMATOLOGY

Since publication of the first edition, numerous studies on hematology and clinical chemistry have been conducted in North America and elsewhere.[1,2,12–16,21,22,26,27,32,35,37,39,43–45,51,59,62,63] Reference ranges for camelids are found in Table 15.1. Previous studies carried out in South America have reported results for llamas, alpacas, and vicuñas, which are so similar that hematologic values for one species may be assumed to be applicable to the others (Tables 15.2 and 15.3).[48] Camelids are unique and were evolutionarily separated from other ungulates for millions of years. They developed adaptations to harsh environments (deserts for camels[49,50,61] and high altitudes for SACs[7,23,24,42,46,57,64]), and hematologic changes were part of that adaptation. Comparative hematology of selected parameters are listed in Table 15.4. All hematologic and serum chemistry values are reported in conventional units generally used in the United States. Système Internationale conversions are listed in Tables 15.5 and 15.6.

Hemoglobin values for SACs are higher than those for cattle and the same as for horses. Camelid erythrocytes are small (Table 15.4) and ellipsoid[18,19,56,58] and circulate in larger numbers than in other mammalian species.[25,47] The small size and shape result in a lower packed cell volume (PCV). Camelid erythrocytes are oriented with the long axis in the direction of the blood

Abbreviations Used in Hematology

Mass = kg
RBC = Erythocytes
Hb = Hemoglobin
PCV = Packed cell volume (hematocrit)
MCV = Mean corpuscular volume
MCH = Mean corpuscular hemoglobin
MCHC = Mean corpuscular hemoglobin concentration
L = Liter
g/dl = grams per deciliter
mg/dl = milligrams per deciliter
µg/dl = Micrograms per deciliter
WBC = Leukocytes
Bands = Band neutrophils
M = Meter
Platelet = Thrombocyte
BUN = Blood urea nitrogen
CSF = Cerebrospinal fluid
PF = Peritoneal fluid
SI units = Systemè Internationale
Conv. US units = Conventional (US)
AST (GOT) = Aspartate amino transferase
ALT (GPT) = Glutamate pyruvate transferase
ALP = Alkaline phosphatase
SDH = Sorbital dehydrogenase
CK = Creatine kinase
LDH = Lactate dehydrogenase
NWC = New World camelid
SAC = South American camelid
OWC = Old World camel
AC = Arabian camel
Bact. = Bactrian camel

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Table 15.1. Hematologic parameters in camelids.

Parameter/units Conventional/US	*South American camelids*				*Racing camels*		*Bactrian camels*		*Parameter/unit International/SI*
	<6 Months		*Adults*		*Adults*		*Adults*		
	US	*SI*	*US*	*SI*	*US*	*SI*	*US*	*SI*	
Erythrocytes/10^6/mm^3	9.6–17.2	9.6–17.2	10.5–17.2	10.5–17.2	7.5–12	7.5–12	10.2–13.2	10.2–13.2	Erythrocytes/10^{12}/L
Hemoglobin/g/dL	10.1–18.1	101–181	11.9–19.4	119–194	12–15	120–150	11.72–13.68	117–137	Hemoglobin/g/L
PCV/%	24–28.5	24–28.5	27–45	27–45	26–38	26.0–38	36.5–42.7	36.5–42.7	PCV/L/L
MCV/µL	21.5–29	21.5–29.9	22.2–29.9	22.2–29.9	26–34	26–34			MCV/fL
MCH/µg	9–11.9	9–11.9	10.1–12.7	10.1–12.7					MCH/pg
MCHC/g/dL	39.4–44.9	39.4–44.9	39.3–46.8	39.3–46.8					MCHC/g/L
Leukocytes/10^3/mm^3	7.1–22.9	7.1–22.9	8–21.4	8–21.4	6–13.5	6–13.5	10–15.8	10–15.8	Leukocytes 10^9/L
Neutrophils %	34.9–63		41.7–72.9		50–60		28–83		Neutrophils %
Band neut. %									Band neut.%
Lymphocytes %	18.3–41.9		9.2–25.2		30–45.0		19–56		Lymphocytes %
Monocytes %	0–5.2		0–4.6		2–8		0–7		Monocytes %
Eosinophils %	0–9.5		2.2–21.4		0–6		0–18		Eosinophils %
Basophils %	0–1		0–14		0–2		0–3		Basophils %
Thrombocytes 10^3/mm^3	268–912	268–913	200–600	200–600	200–700	200–700			Thrombocytes
Reticulocytes/% of RBCs	0–7.5		0–0.4						Reticulocytes/% of RBCs

Table 15.2. Reference ranges for hematologic parameters in llamas and alpacas.

Parameter	*Fowler and Zinkl 1989*		*Van Houten et al. 1992*	*Weiser et al. 1992*
	<1 year	*Adults*	*Adults*	*Adults*
Erythrocytes × 10^6/µl	10.2–17.1	10.5–17.2	11.3–16.9	11.3–17.6
PCV (%)	25–40	27–45	28–39	29–39
Hemoglobin (g/dl)	10.4–17.4	12.5–19.2	12.6–17.8	12.8–17.6
MCV (fl)	21.9–28.1	22.8–29.9	20.1–27.5	21–28
MCH (pg)	9.2–11.9	10.1–12.7		
MCHC (g/dl)	38.9–45.3	39.3–46.8	43.3–46.5	43.2–46.6
Leukocytes/µl	8,000–23,800	8,000–21,400		7,500–21,500
Neutrophils/µl	2,502–13,411	4,711–14,868		4,600–16,000
Band neutrophils/µl	0–91	0–147	0–360	0–350
Lymphocytes/µl	1,762–7,900	689–4,848	1,000–7,800	1,000–7,500
Eosinophils/µl	0–5,934	647–4,867	0–3,200	0–3,300
Monocytes/µl	0–1,462	0–1,009	0–1,000	50–800
Basophils/µl	0–383	0–298	0–400	0–400
Platelets/µl			200,000–600,000	
Reticulocytes/µl	0–4.8	0–0.4		
Nucleated RBC/µl	0–8	0–2		
Plasma protein (g/dl)	4.8–7.0	5.1–7.9		
Fibrinogen (g/dl)	100–400	100–500		

Table 15.3. Leukocyte factors for South American camelids in Peru.

Parameters	*At 4200 m*[a]			*At 3900 m*[b]	
	Llama (12)	*Alpaca (12)*	*Vicuña (12)*	*Alpaca (113)*	*Vicuña (12)*
Leukocytes × 10^3/µl	11.7 ± 1.20	11.6 ± 0.85	12.2 ± 0.81	15.79 5.68–28.48	12.76 8.08–22.76
Neutrophils (%)	59.0 ± 3.90	58.5 ± 3.90	46.8 ± 3.10	52.24 25.5–86.00	55.16 41–67
Lymphocytes (%)	27.7 ± 4.30	33.5 ± 4.20	33.8 ± 3.00	36.21 11.8–69.00	28.81 17.50–42.50
Monocytes (%)	3.3 + 0.50	3.0 ± 0.60	2.4 ± 0.30	1.50 0–9.80	6.85 1–26.80
Eosinophils (%)	10.0 ± 2.90	5.0 ± 1.10	14.6 ± 2.20	8.24 0–28.00	8.49 0.5–22.50

[a] Data from Reynafarje et al. 1968.
[b] Data from Copaira 1949, 1953.

Table 15.4. Hematologic parameters in selected species.

Parameter	*Horse*	*Cow*	*Sheep*	*Lamoid*	*Camel*
Erythrocytes × 10^6/µl	6.5–12.5 (9.5)	5–10 (7)	9–15 (12)	9.9–17.7 (13.8)	7.22–11.76
Size (µm)	5.7	4–8 (5.5)	3.2–6 (4.5)	3.2 × 6.5	
Shape	Round	Round	Round	Elliptical	Elliptical
Life span (days)	140–150	160	140–150	60–225	
Hemoglobin (g/dl)	11–19 (15)	8–15 (11)	9–15 (11.5)	11.5–19.5 (15.5)	7.8–15.9
PCV (%)	32–52 (42)	24–46 (35)	27–45 (35)	25–46.5 (35)	25–34
MCV (fl)	34–58 (46)	40–60 (52)	28–40 (34)	22–30 (26)	35–60
MCH (pg)	12.3–19.7 (15.9)	11–17 (14)	8–12 (10)	9.8–12.7 (11.2)	17–22
MCHC (g/dl)	31–37 (35)	30–36 (33)	31–34 (32.5)	37.7–49 (43)	36.5–50.9
Leukocytes/µl	5,500–12,500 (9,000)	4,000–12,000 (8,000)	4,000–12,000 (8,000)	8,900–22,000 (16,200)	11,200–16,500
Neutrophil/lymphocyte ratio	1.1	0.48	0.5	1.54	1.45
Thrombocytes × 10^5	1.0–3.5 (2.5)	1.8–8.0	2.5–7.5 (4)		

Table 15.5. Conversion between conventional and Système International (SI) units, hematology.

Analyte	*Conventional units*	*Multiply by*		*SI units*
		Conventional units to SI	*SI units to conventional*	
Erythrocytes, RBC	$10^6/mm^3$	10^6	10^{-6}	$10^{12}/L$
PCV, hematocrit	%	0.01	100	L/L
Hemoglobin	g/dl (gm%)	10.0	0.1	g/L
MCV	μ^3	No change	No change	fl
MCHC	g/dl (gm%)	10	0.1	g/L
MCH	µg	No change	No change	pg
Leukocytes, WBC	$10^3/mm^3$	10^6	10^{-6}	$10^9/L$
Platelets	$10^3/mm^3$	10^6	10^{-6}	$10^9/L$
Protein, total	g/dl	10.0	0.1	g/L
Albumin	g/dl	10.0	0.1	g/L
Globulins	g/dl	10.0	0.1	g/L

Table 15.6. Conversions between conventional and Système International (SI) units, blood and fluid chemistry.

Analyte	*Conventional units*	*Multiply by*		*SI units*
		Conventional to SI	*SI to conventional*	
Albumin	g/dl	144.9	0.007	µmol/L
Albumin	g/L	14.49	0.069	µmol/L
Ammonia	µg/dl	0.59	1.69	µmol/L
Bicarbonate	mEq/L	No change	No change	mmol/L
Bilirubin	mg/dl	17.1	0.059	µmol/L
Calcium (serum)	mg/dl	0.25	4.0	mmol/l
Calcium (urine)	mg/24hr	0.025	40.0	mmol/24hr
Carbon dioxide	mEq/L	1.0	1.0	mmol/L
Chloride	mEq/L	1.0	1.0	mmol/L
Cholesterol	mg/dl	0.026	38.67	mmol/L
Copper	µg/dl	0.157	6.35	µmol/L
Creatinine (serum)	mg/dl	88.4	0.011	µmol/L
Creatinine (urine)	g/24hr	8.84	0.113	g/24hr
Enzymes	U/L	0.017	60.0	µkat/L
Fibrinogen	mg/dl	0.01	100.0	g/L
Globulins	g/dl	10.0	0.1	g/L
Glucose	mg/dl	0.056	18.0	mmol/L
Iron	µg/dl	0.179	5.58	µmol/L
Iron-binding capacity	µg/dl	0.048	20.7	µmol/L
Lactate	mg/dl	0.111	9.01	mmol/L
Magnesium	mg/dl	0.41	2.43	mmol/L
Magnesium	mg/dl	0.882	1.216	mEq/L
Myoglobin	mg/dl	0.585	1.71	µmol/L
pO^2	mmHg	0.133	7.5	kPa
Phosphate	mg/dl	0.323	3.1	mmol/L
Potassium	mEq/L	1.0	1.0	mmol/L
Protein (total)	g/dl	10.0	0.1	g/L
Protein (CSF)	mg/dl	0.01	100.0	g/L
Protein (urine)	mg/24hr	0.01	100.0	g/24hr
Sodium	mEq/L	1.0	1.0	mmol/L
Thyroxine (T_4)	µg/dl	12.87	0.777	nmol/L
Triiodothyronine (T_3)	ng/dl	0.015	65.11	nmol/L
Triglycerides	mg/dl	0.011	88.5	mmol/L
Urea nitrogen (UN)	mg/dl	0.357	2.81	mmol/L
Uric acid	mg/dl	59.48	0.017	µmol/L
Zinc	µg/dl	0.153	6.54	µmol/L

flow; this makes it possible to traverse small capillaries, resulting in fewer problems of sludging when the viscosity of the blood increases during dehydration.[56] One study determined that alpaca blood has a large oxygen-carrying capacity and a low viscosity, which ideally suits the alpaca to living in an environment with low oxygen tension.[64,65] Numerous studies have been conducted on erythrocyte fragility and biochemistry.[33,29,30,31,39] The proteins in erythrocytes provide a means of verifying parentage according to blood groups (Chapter 22).

The normal mean corpuscular volume (MCV) of SACs is lower than that found in livestock species because of the smaller erythrocyte size. The mean corpuscular hemoglobin concentration (MCHC) is higher (44.5) than in livestock (30 to 35). The MCHC measures the ratio of the weight of hemoglobin (Hb) to the total volume of the erythrocytes. Normal Hb levels in SACs are higher than those of livestock species and the PCV is slightly lower; therefore, a higher MCHC index results. A low MCHC in SACs is indicative of a hypochromic anemia.

The mean corpuscular hemoglobin (MCH) expresses the weight of Hb in an average erythrocyte. Normal MCH levels of SACs are slightly lower than those of other livestock species because the erythrocytes are smaller. An excessively low value indicates anemia.

The leukocyte count is significantly higher in SACs than in other domestic mammals. The neutrophil-lymphocyte ratio is similar to that of the horse, with the majority of cells being neutrophils[34] (Figures 15.1, 15.2A, 15.5). High eosinophil numbers have been reported in the European, North American, and South American literature. Attempts have been made to correlate this with parasite burdens, but no controlled studies have been conducted. The definitive reason for high eosinophil counts is not known.

SAC eosinophils contain homogenous granules, similar to those of horses and cattle (Figure 15.8), in contrast to eosinophils of dogs and cats that have crystalloid granules. Eosinophils originate in bone marrow,[5] and in cattle they mature from eosinophilic myelocytes in three to six days. The life span of an eosinophil is eight to fifteen days following entrance into the peripheral blood. Eosinophils leave the blood quite rapidly to concentrate beneath the epithelial surfaces of intestine, subcutaneous tissue, and the respiratory tract. The cells do not re-enter the circulatory system.

Eosinophils function to detoxify histamine and inhibit the edema-producing properties of serotonin and bradykinin. Minimal phagocytosis is performed. Eosinophilia is thought to be the result of parasitism, allergic and anaphylaxis reactions, or severe hypoxemia. In all of these conditions there is histamine release in the tissue, and eosinophils respond. The eosinophilic response to parasitism most often accompanies larval migration or parasites that attach to the mucosa and extract blood. An antigen/antibody reaction of the parasite and host attracts eosinophils. Elevated eosinophil numbers in cerebrospinal fluid is considered, by some, to be diagnostic of spinal cord migration of the larvae of *Parelaphostrongylus tenuis* (meningeal worm).

Eosinopenia accompanies hypercortisolism as seen in stress, Cushing's syndrome, and acute infection. Corticosteroid therapy has a neutralizing effect on histamine, which in turn decreases eosinophil attraction.

Basophils are also produced in bone marrow and have a life span of ten to twelve days in peripheral blood (Figures 15.2A, 15.2B). Basophils are related to mast cells and contain a large amount of histamine. Basophils are in zero to low numbers in SAC peripheral blood.

Monocytes originate from stem cells in bone marrow, (not from lymphocyte series) (Figure 15.3). Monocytes are related to macrophages and require approximately nine hours to generate and then stay only about twenty-two hours in blood before entering the tissue. The life span is only a few days. Monocytes are phagocytic, engulfing bacteria, damaged tissue cells, and antigen/antibody complexes.

Lymphocytes are produced in bone marrow and lymph nodes (Figures 15.1 and 15.4). In cattle, lymphocytes require only two hours to mature and have a life span of two to 100 days, depending on the cell type. T-cell lymphocytes are associated with cellular immunity and delayed hypersensitivity. B-cell lymphocytes are responsible for humoral responses. A few large lymphocytes contain azurophilic granules in the cytoplasm, similar to cattle. Lymphocytes move freely from blood to lymph nodes and vice versa. Lymphocytes are vital to the competency of the immune system. Various tests are conducted to measure the immune status of SACs (Chapter 22).

Hemogram reports are often accompanied by statements such as toxicity of neutrophils or degenerative left shift. Following are descriptions of the characteristics used in such a classification.

Regenerative left shift is a neutrophilia and is considered slight when bands appear in peripheral blood, moderate when bands and metamyelocytes are seen, and marked when myelocytes and progranulocytes appear.

A degenerative left shift is characterized by neutropenia when young neutrophilic granulocytes exceed mature neutrophils, indicating that bone marrow is unable to produce mature cells. This condition is common in septicemia.

Toxic neutrophils contain vacuoles and diffuse basophilia. Toxic granules are blue-black or large red discs.

Table 15.7. Leukocytosis response to bacterial infection.

Degree	*Horse*	*Cow*	*SAC*
Moderate	15,000–25,000	4,000–12,000	23,000–30,000
Marked	25,000–30,000	15,000–25,000	30,000–35,000
Extreme	35,000+	25,000+	40,000+

Leukocytic response to a few disease conditions follow: antigen/antibody reaction—eosinophilia, chronic infection or inflammation—monocytosis (Figure 15.8); virus infection—leukopenia; bacterial infection—early septicemia, leukocytosis, or degenerative left shift (Table 15.7, Figures 15.6, 15.7, and 15.9). With pus formation there is a marked neutrophilia.

Noninfectious causes of neutrophilia include malignancy, chemical intoxication, uremia, hemolytic crisis, and significant blood loss. Panleukopenia is seen in ehrlichiosis, bacterial endotoxemia, bacterial toxemia, and advanced septicemia.

SERUM BIOCHEMISTRY[3,36,67]

Serum biochemistry reference ranges reported by various investigators are found in Table 15.8. Most biochemical parameters of SACs are similar to those of cattle, with the exception of serum glucose, which is twice that of cattle (Table 15.9).[20] There are no established reference ranges for a number of parameters (liver function other than enzymes and the liver-specific enzymes, kidney function, glucose tolerance, amylase, xylose absorption). Serum enzyme patterns for llamas are listed in Table 15.10.

Serum protein levels in alpacas have been studied in South America (Table 15.11).[41] Protein electrophoresis patterns have been performed on North American llamas.[27,45,60] Table 15.12 provides means and ranges for various age groups on a limited number of llamas.

Precolostrum neonates have low total protein and relatively low globulin.[11] With the absorption of colostral immunoglobulin, the A:G ratio is lowered, and total protein and globulin levels are elevated. Globulin levels decrease at about three weeks of age, as passively acquired globulin wanes. Levels remain low until production of globulins by the maturing immune system of the cria makes up the loss. This period may be a critical time in the life of the SAC neonate, since it may be more susceptible to infectious diseases at this stage.

In a study of 188 alpacas in Peru, calcium levels ranged from 7.6 to 11.6 mg/dl, and phosphorus, 4.2 to 9.7 mg/dl. The investigator noted that alpacas did not become hypocalcemic shortly before or following parturition, as do other domestic ungulates. No cases of clinical postparturient hypocalcemia (milk fever) in SACs have been reported. In one study, neonate alpacas, up to three weeks of age, had higher calcium levels than adults.

Serum inorganic phosphorus values show a wide range of reported normals, particularly at the lower end of the scale. This author believes that those low values do not represent a true normal range (including those reported in his own published paper) because it is now known that hypophosphatemia is a common clinical finding in llamas and alpacas during long periods of inclement weather when animals are deprived of exposure to sunlight. Phosphorus levels below 4.5 mg/dl should be considered hypophosphatemic.

Accurate phosphorus determination also depends on proper handling of blood specimens from collection to analysis. Cells should be separated from serum as quickly as possible to avoid hemolysis. Rupture of the cell membrane allows escape of organic phosphorus molecules, which then become included in the inorganic phosphorus determination to give a false normal to high reading. Blood for biochemical evaluation should not be allowed to become heated (e.g., on the dashboard of a vehicle) because this also causes hemolysis.

Values for iron evaluation were determined in thirty-eight healthy llamas[10] and included serum iron concentration 101 ± 21 µg/dl (70 to 148), total iron-binding capacity 300 ± 39 µg/dl (230 to 370), and transferrin saturation 34 ± 6.6% (22 to 50). (Additional information on iron is found in the references.).

Miscellaneous biochemical parameters for SACs kept at 4,200 m altitude are listed in Table 15.13.[4,6,13,52,53,55,65] Prenursing neonate biochemical and hematologic values differ from those of adults. See Chapter 21 for a full discussion of important parameters. Comparative serum biochemistry values are listed in Table 15.9.

Coagulation factor reference ranges have been reported in only one llama and one guanaco.[35] Clotting time was ten seconds for the guanaco and 14.5 seconds for the llama, prothrombin time was 120 seconds for the llama, partial thromboplastin time was 36.4 seconds for the guanaco and 26.4 seconds for the llama, and the platelet count in the llama was 370,000, with a platelet diameter of 2 mm.[38,40]

There is disagreement between two studies regarding the life span of erythrocytes in SACs. In one study using erythrocytes labeled with 51 chromium in vitro, the life span was approximately sixty days in twelve llamas, twelve alpacas, and twelve vicuñas.[48] In another study of two guanacos, using glycine-2-^{14}C, the life span was 225 days.[9] The life span of erythrocytes in humans is 120 days, horses 140 to 150 days, and cattle 135 to 162 days.[28] The shorter life span in the Reynafarje et al.[48] study may be the result of adaptation to

Table 15.8. Serum biochemistry parameters in camelids.

Parameter/units Conventional/US	*South American camelids*				*Racing camels*		*Bactrian camels*		*Parameter/unit International/SI*
	<1 Year		*Adults*		*Adults*		*Adults*		
	US	*SI*	*US*	*SI*	*US*	*SI*	*US*	*SI*	
Total protein/g/dL	4.9–7.1	49–71	5.1–7.8	40–78	5.7–7.5	57–75	5.47–7.37	54.7–73.7	Total protein/g/L
Albumin/g/dL	3.4–4.5	34–45	3.1–5.2	31–52	3–4.3	30–43.0	3.66–5.3	36.6–53	Albumin/g/L
Fibrinogen/mg/dL	100–400		100–500		250–400		268		Fibrinogen/mmol/L
Urea nitrogen/mg/dL	12–28	4.28–18	9–34.0	3.21–12.14	3–21.0	1.07–7.5	12.3–17.7	4.38–6.33	Urea nitrogen/mmol/L
Creatinine/mg/dL	1.3–2.4	114.9–212.2	1.4–3.2	123.8–282.9	0–2.2	0–194.5	0.2–4	17.7–35.4	Creatinine/Mmol/L
Glucose/mg/dL	108–156	5.99–8.66	74–154	4.11–8.55	0–110	3.89–6.11	37–67	2.05–3.72	Glucose/mmol/L
Calcium/mg/dL	8.6–10.7	2.15–2.68					2.2–2.68	0.60–0.67	Calcium/mmol/L
Phosphorus/mg/dL	5.1–10.2	1.65–3.29	2.6–7.3	0.84–2.36	3.5–6	1.13–1.94	1.6–2	0.52–0.68	Phosphorus/mmol/L
Sodium/mmol/L	149–153	149–153	148–158	148–158	150–160	150–160	129–161	129–161	Sodium/mmol/L
Chloride/mEq/L	102–114	102–114.0	102–120	102–120	90–110.0	90–110.0			Chloride/mmol/L
Potassium/mEq/L	4.4–7		3.7–6.1		3.5–5.5		6–6.1		Potassium/mmol/L
Magnesium/mg/dL	2–2.3		2–3.5				1.8–2.9		Magnesium/mmol/L
Iron/mg/dL	70–148		70–148				49–57		µmol/L

Table 15.9. Comparative serum biochemistry of the llama, camel, cow, and horse.

Parameter	*Llama*	*Camel*	*Cow*	*Horse*
Total protein (g/dl)	4.7–7.3	6.3–8.7	6.7–7.5	5.2–7.9
Albumin (g/dl)	2.9–5	3–4.4	3–3.6	2.6–3.7
Globulin (g/dl)	1.1–3	2.8–4.4	3–3.5	2.6–4
A:G ratio	1.1–1.6:1		0.84–0.94:1	0.62–1.46:1
Calcium (mg/dl)	7.6–10.9	6.3–11	9.7–12.4	11.2–13.6
Phosphorus (mg/dl)	1.6–11	3.9–6.8	5.6–6.5	3.1–5.6
Sodium (mEq/L)	148–158	129.3–160.7	132–152	132–146
Potassium (mEq/L)	3.6–6.2	3.6–6.1	3.9–5.8	2.4–4.7
Chloride (mEq/L)	98–120		99–109	97–111
Total CO_2 (mm/L)	14–34		24–32	21.2–32.2
T_3 (ng/dl)	0–423			
T_4 (µg/dl)	9.8–30		4.2–8.6	0.9–2.8
SGOT (IU/L)	128–450		78–132	226–366
SGPT (IU/L)	0–14		14–38	3–23
SGGT (IU/L)	3–28			3–13.4
SDH (IU/L)	0–15		4.3–15.3	1.9–5.8
LDH (IU/L)	10–695		692–1445	162–412
ALP (IU/L)	0–610		0–488	143–395
CPK (IU/L)	0–137		4.8–12.1	2.4–23.4
Creatinine (mg/dl)	0.9–2.8	1.2–2.8	1–2	1.2–1.9
Urea N (mg/dl)	9–36	15.7–48.5	20–30	10–24
Cholesterol (mg/dl)	0–128	20.8–79.2	80–120	75–150
Glucose (mg/dl)	76–176	37–67	45–75	75–115

Table 15.10. Serum enzyme parameters in camelids.

Parameter/units Conventional/US	*South American camelids*				*Racing camels*				*Parameter/unit International/ SI*
	<1 Year		*Adults*		*Calves*		*Adults*		
	US	*SI*	*US*	*SI*	*US*	*SI*	*US*	*SI*	
AST (GOT)/IU.L	137–547	137–547	127–420	127–420	60–120	60–120			AST (GOT)/IU/L
ALT (GPT)/IU/L	5–33	5–33	9–29	9.9–29			7.3–5.9		ALT (GPT)/IU/L
ALP/IU/L	0–671	0–671	27–132	27–132	60–140	60–140			ALP/IU/L
SDH/IU/L	1–24	1–24	1–62	1–62					SDH/IU/L
CK/IU/L	12–130	12–130	14–238	14–238	40–120	40–120			CK/IU/L
LDH/IU/L	144–750	144–750	82–670	82–670	400–775	400–775			LDH/IU/L

Table 15.11. Serum protein and fractionation in alpacas.

	Adults (100)	*Crias*				
		1 day	*8 days*	*15 days*	*22 days*	*120 days*
Total protein	5.22–8.92 (6.79)	6.36	5.70	5.82	5.95	5.92
Albumin	2.05–4.78 (3.19)	2.32	2.69	3.21	3.13	1.19
Globulin	1.76–5.47 (3.55)	4.03	3.00	2.60	1.27	4.72
Alpha	0.18–2.70 (1.24)	1.56	1.42	1.08	2.82	3.34
Beta	0.03–2.79 (1.04)	1.21	0.59	0.75	0.62	0.55
Gamma	0.66–2.32 (1.42)	1.37	0.98	0.76	0.79	1.02
A:G ratio	0.44–1.80 (0.90)	0.52	0.90	1.28	1.14	0.26

Source: Vallenas 1957.

Table 15.12. Serum protein electrophoresis patterns in llamas.

Age group	*Parameter*	*Total protein (g/dl)*	*Albumin (g/dl)*	*Globulin (g/dl)*	*Alpha (g/dl)*	*Beta (g/dl)*	*Gamma (g/dl)*	*A:G ratio*
<1 month	Mean	5.6	3.3	2.3	0.7	1.0	0.7	1.4:1
n = 6	Range	5.1–5.9	3.1–3.5	1.9–2.7	0.5–0.9	0.8–1.1	0.4–1.1	1.1–1.7:1
1–5 months	Mean	5.9	3.7	2.2	0.7	0.9	0.6	1.7:1
n = 6	Range	5.4–6.4	3.3–4.0	1.4–2.7	0.4–1.0	0.7–1.1	0.3–1.0	1.4–2.8:1
6–18 months	Mean	6.1	3.8	2.3	0.7	0.9	0.8	1.6:1
n = 11	Range	5.5–6.9	3.1–4.4	1.8–3.0	0.5–0.8	0.6–1.1	0.4–1.1	1.3–2.4:1
Mature male and female	Mean	6.3	3.5	2.9	0.7	1.0	1.1	1.2:1
n = 7	Range	5.9–6.8	3.1–3.7	2.3–3.3	0.5–0.9	0.9–1.1	0.8–1.5	1.1–1.6:1
All llamas >1 month	Mean	5.9	3.5					
n = 24	Range	5.4–6.9	3.1–4.4					

Note: n = number of animals tested.
Source: Author's unpublished data.

Table 15.13. Miscellaneous biochemical and hematologic parameters of mammals maintained at altitude (4200 m).

Parameter	*Llama*	*Alpaca*	*Vicuña*	*Horse*	*Cow*	*Human*	*Camel*
Plasma iron (µg/dl)	192 ± 11[a]	156 ± 11	113 ± 6.6	73–140	57–162 (62–133)	94 ± 3.8	98.5
Iron uptake (%)	84.8 ± 3.6	82.4 ± 4.4	90.6 ± 3.3	74–77	55	92 ± 2.4	
Plasma iron turnover rate (mg Fe/day/kg)	0.59 ± 0.12	0.52 ± 0.04	1.11 ± 0.32	0.45–0.65	0.27	0.39 ± 0.01	
Red cell iron turnover rate (mg Fe/day/kg)	0.52 ± 0.10	0.43 ± 0.04	1.03 ± 0.34				
Total blood volume (ml/kg body weight)	65.2 ± 4.1	72.0 ± 5.3	86.6 ± 2.1	68.8–109.6	62.5–81.1	88.1 ± 1.5	
Plasma (ml/kg body weight)	40.2 ± 2.1	45.3 ± 3.4	56.0 ± 1.4				
Erythrocytes (ml/kg body weight)	25.0 ± 1.4	24.3 ± 4.2	31.3 ± 0.9				
Hemoglobin (ml/kg body weight)	9.9 ± 0.8	9.7 ± 0.8	11.9 ± 0.3				
Arterial (%) oxygen saturation	80.2 ± 2.31	91.5 ± 0.1					
Bilirubin							
Direct mg/dl	0.07 ± 0.004	0.10 ± 0.015	0.06 ± 0.004				
Indirect mg/dl	0.06 ± 0.01	0.04 ± 0.01	0.02 ± 0.01				
Total mg/dl	0.13 ± 0.03	0.14 ± 0.01	0.08 ± 0.01				

[a] Standard error.

chronic hypoxic conditions, which are also reflected in the higher rates of red cell iron turnover in SACs as compared with other domestic animals.[48]

Bone marrow biopsies were performed on six alpacas, eight llamas, and twelve vicuñas kept at 4,200 m.[48] The myeloid-erythroid (M:E) ratio was approximately 0.5:1. In humans living at sea level, the M:E ratio is 3:1, and for those living at high altitudes, 1:1. In cattle, the M:E ratio is 0.71:1 and in horses 2.43:1. In the author's clinic, healthy llamas usually have an M:E ratio of 1:1 or 1.2:1. In a more recent, detailed study of the cellular components of bone marrow in llamas, an M:E ratio of 0.9:2.9 was considered normal.[5]

Blood volumes for SACs have been calculated to be between 6.5% and 8.6% of body weight.

LYMPHATICS

Lymph vascular patterns and sites of the lymph nodes in SACs are the subjects of two dissertations in Peru, but otherwise, little is known about the lymphatic system.[8] The authors report that the distribution of lymph nodes is similar to that of cattle and sheep. On the basis of anatomic and pathologic dissection by this author, the nodes of SACs are small and may be difficult to locate. Multiple small nodes may be found, rather than a single large node at certain sites such as the superficial inguinal or prefemoral locations. It is possible to palpate these nodes on emaciated animals. The mediastinal and mesenteric nodes are present but are small and may be easily overlooked. Aggregations of lymphoid tissue (Peyer's patches) are located along the antimesenteric border of the large intestine.

DIAGNOSTIC PROCEDURES

Constituents of the blood are easily measured in the laboratory, facilitating diagnoses. Standard procedures are employed for SACs, except that automated erythrocyte counting systems must be calibrated for camelid erythrocytes. Reference ranges for most parameters have been established. The technique for bone marrow aspiration is described in Chapter 4.

DISEASES

Infectious Diseases

Infectious diseases of hemic systems are discussed in Chapter 7 and include leptospirosis, ehrlichiosis, borreliosis, eperythrozoonosis, bacillary hemoglobinuria, and septicemia[51] caused by miscellaneous bacteria.

Parasitic Diseases

The only parasitic disease of the blood of camelids is trypanosomiasis (Chapter 8). Immature stages of other parasites may be found in the blood, as a stage of the life cycle or while being transported to preferred locations within the body. *Parelaphostrongylus tenuis* matures in the venous sinuses of the meninges of the white-tailed deer, and ova are transported to the lungs via the jugular vein. This strongyle is an aberrant parasite of llamas and may not reach maturity in this host (Chapter 8).

Noninfectious Diseases

Lymphosarcoma is the only neoplasia as yet reported from the hematopoietic or lymphatic system (Chapter 9). With more critical necropsies and more complete reporting, it is probable that tumors similar to those affecting other domestic animals will be found.

Anemia

Anemia has become an important clinical finding in llamas. Generally, anemia is a secondary disease caused by numerous primary agents. Anemia is a reduced ability of the blood to supply oxygen to the tissues and may be the result of a reduction of erythrocyte numbers, hemoglobin concentration, or hematocrit. See Table 15.1 for normal values.

ETIOLOGY. Basically, anemia is caused by either an excessive loss of erythrocytes and/or hemoglobin or by decreased production of vital blood constituents. Excessive loss of blood constituents may be caused by hemorrhage (lacerations, hematoma, extensive contusion, gastrointestinal ulceration, coagulopathy), parasitism, ingestion of spoiled yellow sweet clover hay or anticoagulant rodenticides, hemolytic crisis (copper toxicity, leptospirosis, bacillary hemoglobinemia), iron deficiency, hepatopathy, or chronic hemolysis (hypophosphatemia).

Iron deficiency is the most common cause of decreased hemoglobin production, but other nutrient deficiencies (copper, vitamin B_{12}) may contribute. Bone marrow suppression may be caused by renal disease, irradiation, myelotoxins (bracken fern toxicity), mild chronic inflammatory diseases (infections), or hypothyroidism.

Hepatopathy may interfere with serum protein production, causing hypoproteinemia and changes in the osmolarity of the blood. Two toxicities inhibit oxygen transport and utilization, but they are not considered to cause anemia. Methemoglobinemia prevents oxygen binding to hemoglobin, hence oxygen transport. The most frequent cause of methemoglobinemia is the ingestion of excessive nitrate, which is reduced to nitrite in the forestomach and absorbed, producing the toxic effect. There is one report of vicuñas dying from nitrate poisoning following consumption of silage.

The cyanide ion interferes with the cytochrome oxidase enzyme system and inhibits utilization of oxygen at the tissue level. The body becomes starved for oxygen, and death ensues quickly if immediate therapy is not forthcoming.

CLINICAL SIGNS. Anemic animals exhibit variable signs, depending on the etiology.[10,47] Most affected SACs are underweight or have had a recent weight loss. Basically, dyspnea, depression, and pallor of mucous membranes are seen. If anemia is caused by a hemolytic crisis, additional signs will include icterus, hemoglobinuria, and elevated body temperature (a result of hemolysis). Anemia associated with hypoproteinemia results in ventral edema, with no inflammatory component.

Chronic blood loss may produce basic signs of anemia, but acute blood loss has a more serious prognosis.[66] Loss of one-third of the blood volume is serious

in all animals, and further loss may result in death. Acute blood loss may produce hypovolemic shock, with tachycardia and dyspnea. Hemorrhage inside the calvarium may cause paresis, ataxia, and sudden death. Pericardial hemorrhage may produce cardiac tamponade. Hemorrhage into any organ may interfere with the function of that organ and result in signs of organ malfunction.

Chronic to mild blood loss may be reflected in a regenerative anemia response, demonstrated in hematologic parameters and bone marrow evaluation. Acute blood loss is not easily evaluated in the laboratory (hours elapse before the hematocrit changes, because the body attempts to reestablish blood volume by shifting tissue fluid to the blood). Clinical signs provide the vital clues to establish a diagnosis of peracute hemorrhage.

Methemoglobinemia causes cyanosis, with a chocolate discoloration of the blood and mucous membranes. Cyanide poisoning causes ataxia, muscle tremors, convulsions, and rapid death. Venous blood is bright red because the tissues are unable to accept oxygen bound to hemoglobin. Diagnosis from this characteristic should be made with caution, and only with corroborating factors, because SAC venous blood is normally a brighter red than that of other domestic animals.

Two papers have recently been published that deal with anemia.[44,54] In the first paper, eight llamas were evaluated. They were a subset of anemic llamas presented to a university veterinary clinic from a larger group of twenty-three llamas with failure to thrive syndrome.[25] All the anemic llamas had been normal at birth and during the nursing period. Anemic llamas were thin, with arched backs and dull hair coats, and seven of the eight llamas had angular limb deformity. In contrast with llamas suffering from iron deficiency anemia, none of these llamas were dyspneic.

Hematologic findings included erythropenia, anisocytosis, poikilocytosis, cytoplasmic extensions at the poles of erythrocytes, uneven distribution of hemoglobin, and folded cells. Erythrocyte indices were PCV < 26%, MCV 27.5 ± 5.4 fl (16.3 to 19.5 fl), MCHC

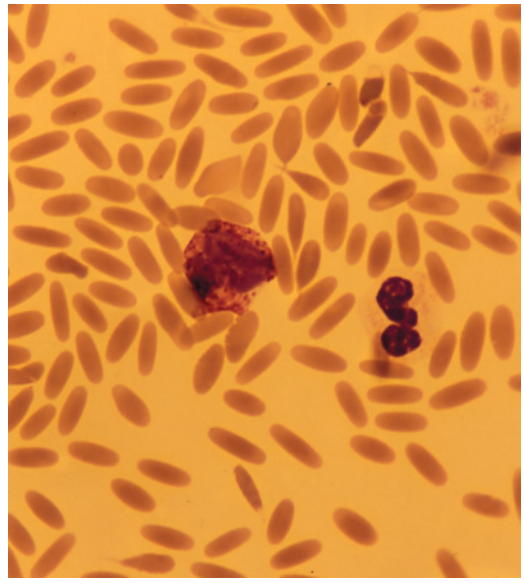

Figure 15.2A. Basophil (left) ad neutrophil (right) in a llama.

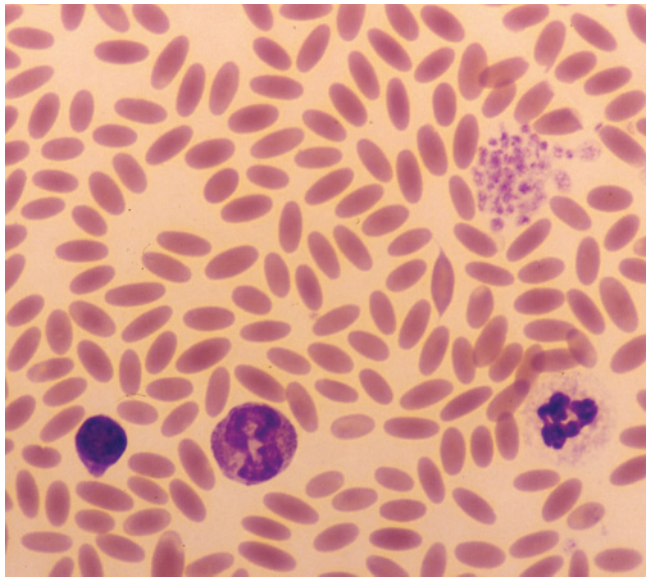

Figure 15.1. Lymphocyte, eosinophil, thrombocytes, and neutrophil in a llama (from left to right).

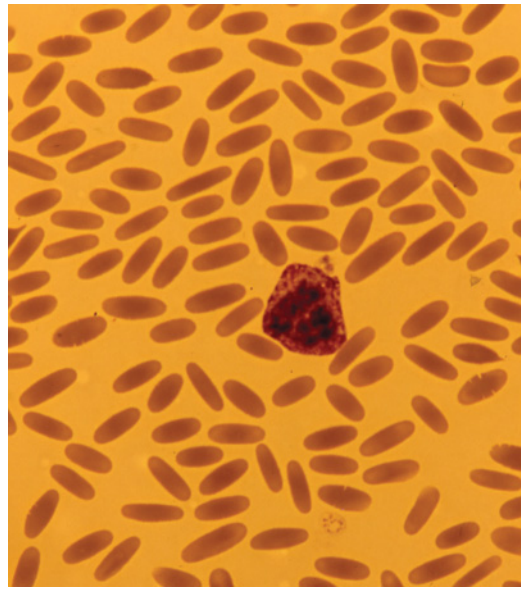

Figure 15.2B. Basophil in a llama.

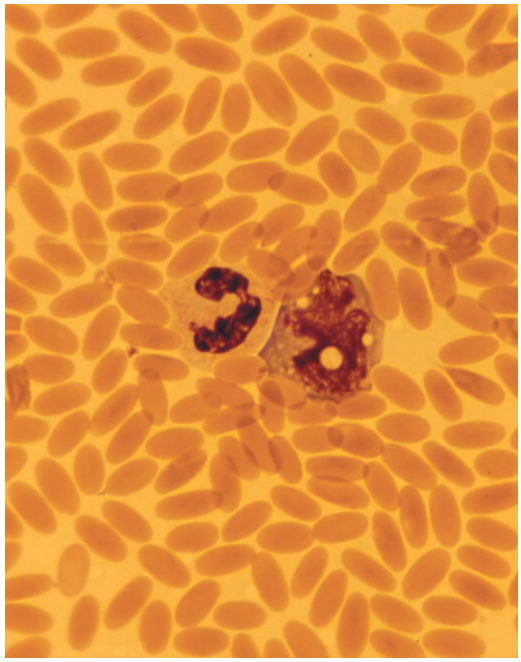

Figure 15.3. Neutrophil (left) and monocyte (right) in a llama.

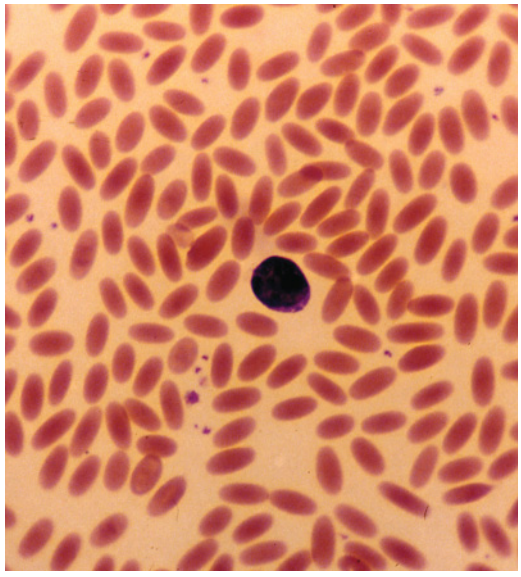

Figure 15.4. Lymphocyte (intermediate) in a llama.

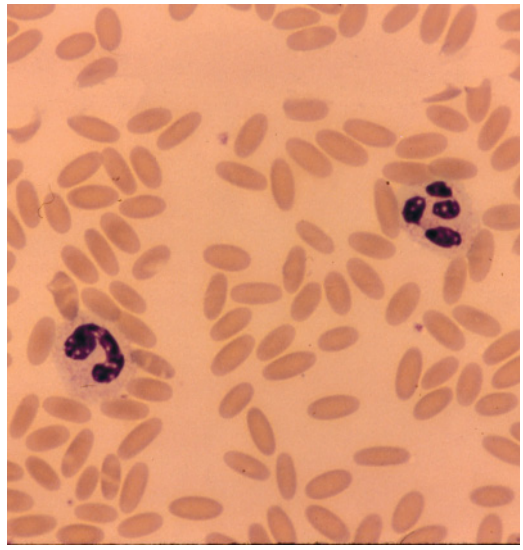

Figure 15.5. Neutrophil.

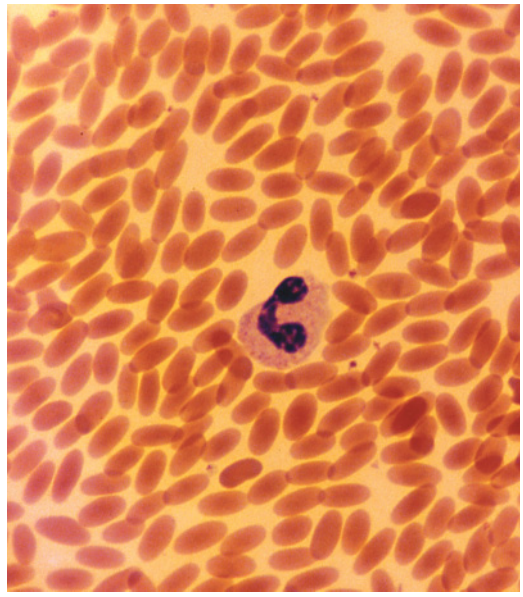

Figure 15.6. A young neutrophil in a llama.

44.5 ± 5.4 g/dl (33 to 54.3 g/dl), and MCH 14 ± 5 (9.5 to 27.7 pg). See the tables for reference ranges. Five llamas were hypophosphatemic, and all eight had low normal T_4 levels.

Investigators considered hypothyroidism, iron deficiency, and other deficiencies (selenium, vitamin E, copper, and zinc). Three of the eight llamas were sisters, so a familial component was considered but not verified. Oral supplementation with iron was unsuccessful in correcting the progressive deterioration of these llamas. Neither was thyroprotein therapy helpful. Six of the llamas died or were euthanized.

Another paper described the anemia syndrome in three llamas with iron deficiency anemia. All three llamas had moderately low PCV (18% to 22%), microcytosis (MCV 15.3 to 18 fl), and decreased MCHC (36 to 41 g/dl). Erythrocytes were centrally hypochromic with frequent folded cells and dacrocytes (hemoglobin concentrated at a pole). All llamas had a neutrophilia, monocytosis, and lymphopenia (stress hemogram). Decreased serum iron concentrations (20 to 60 µg/dl) were present in all llamas, along with decreased transferrin saturation (3.5% to 14.5%) and normal

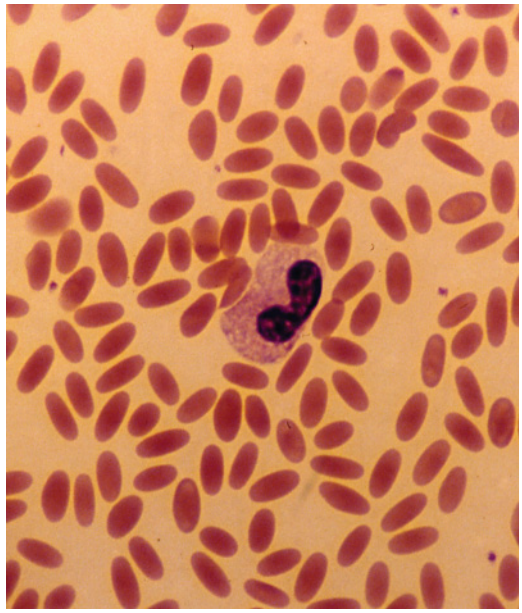

Figure 15.7. Toxic band neutrophil in a llama.

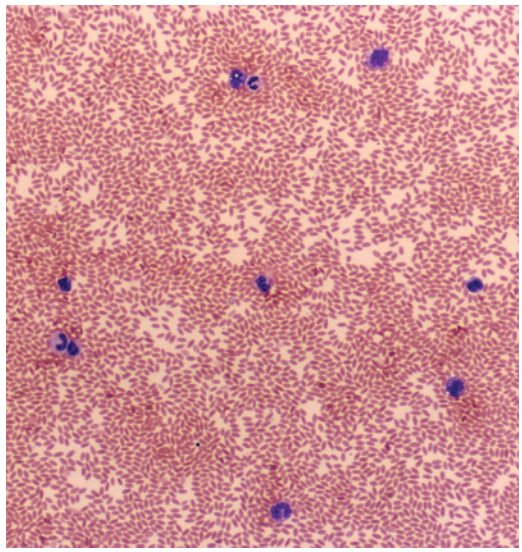

Figure 15.8. Monocytosis in a llama.

or increased total iron-binding capacity (265 to 565 µg/dl).

THERAPY. The underlying cause of the anemia should be corrected. Proper nutrients should be supplied in the diet or given parenterally in acute cases. Blood transfusion is indicated in life-threatening situations (i.e., a hematocrit level less than 15%). Blood compatibility testing is possible, but in an emergency, any SAC blood may be given once with little risk. Thereafter, the risk of a transfusion reaction increases. Two of the three llamas with iron deficiency anemia responded to administration of parenteral iron dextran.[44]

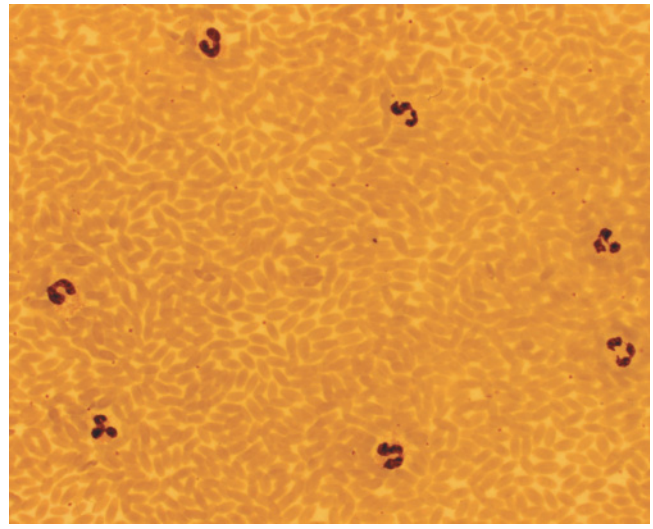

Figure 15.9. Leukocyteosis in a llama.

Polycythemia

Polycythemia (erythrocytosis) is reported in dogs, cats, horses, humans, and a llama.[17,64]

ETIOLOGY. Polycythemia may result from dehydration (relative polycythemia), excessive bone marrow proliferation (primary absolute polycythemia), renal disease, neoplasia, hypoxemia caused by living at high altitudes, or pulmonary or cardiac disease (secondary polycythemia). A llama was diagnosed with secondary absolute polycythemia associated with respiratory pathology.

CLINICAL SIGNS. A female llama became tachypneic with open mouth breathing when exercised. She became extremely distressed when being bred. Pulmonary auscultation revealed harsh lung sounds, and a diagnosis of pneumonia was made and the llama treated accordingly.

DIAGNOSIS. Major laboratory findings were a packed cell volume varying from 50.8% to 66% (the PCV reached 74 just prior to death). Hemoglobin levels were 21.8 to 24.2 g/dl, and the albumin was 5 g/dl. Serum urea nitrogen was 48.6 mg/dl and the creatinine 4 mg/dl. Azotemia developed late in the course of the disease.

Elevated serum erythropoietin levels were attributable to hypoxemia. Erythropoeitin values were obtained on this animal (104 ± 6 mU/ml). Two clinically normal llamas had erythropoeitin levels of 19.6 and 39.8 mU/ml.

THERAPY. One liter of blood was removed from the circulation and physiologic saline returned. The PCV was only slightly reduced and the level returned to pretreatment values in seventy-two hours. No attempt was made to be more aggressive at hemodilution. The animal died.

NECROPSY. Grossly, the liver and lungs were congested. Histologically, pulmonary vessels were thick walled, and hemosiderosis was present in the pulmonary parenchyma. Fibrosis and thrombosis of vessels in the kidney and liver were noted.

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16

Cardiovascular System

ANATOMY

The cardiovascular anatomy of SACs is similar to that of the dromedary camel, as described in detail in Smuts.[35] The heart and arterial system are not unique in camelids.[4,7,18,19,24,26,27,31,35,36] However, the arterial supply and venous drainage of the lower limb are different than in horses and cattle. The major vessels lie on the medial aspect of the metacarpus and metatarsus and are then directed to the palmar and plantar surfaces of the tendons and are distributed to the digits from the interdigital space.[10,11,21,31,27,37] The vascular anatomy of the intestine is unique,[23,33,39] as is the vasculature of the head region.[30,35] The jugular vein is unique. See Chapter 4 for a discussion of superficial veins used for collection of blood samples and intravenous medication.

PHYSIOLOGY

A study of anesthesia in five llamas provided baseline data on the following cardiovascular parameters:[12]

Cardiac output 8.2 ± 0.8 L/minute; stroke volume 146 ± 20 ml/beat; mean systemic arterial pressure 137 ± 8 mm Hg; mean pulmonary arterial pressure 14 ± 0.8 mm Hg; mean right atrial pressure 2.6 ± 0.7 mm Hg; total peripheral resistance 1,470 ± 183 dyne. seconds/cm^3; left ventricular work 15.7 ± 0.9 kg m/minute; cardiac output per kg 73 ± 9 ml/minute/kg; PaO_2 127 ± 8.9 mm Hg; $PaCO_2$ 34 ± 1.1 mm Hg; base excess 2.3 ± 1.1 mEq/L; and bicarbonate 21.3 ± 1.1 mEq/L.

Cardiovascular adaptations contribute to hypobaric tolerance in camelids.[1,5,6,13,15–17,25] Lowland-adapted species, including humans, respond to high-altitude hypoxia with pulmonary artery constriction, resulting in pulmonary hypertension and subsequent right heart hypertrophy. Although llamas and alpacas may have a light to moderate pulmonary hypertension at high altitude, they do not respond with pulmonary arterial hypertrophy and right heart hypertrophy. SACs also do not respond to chronic hypoxia by enlargement of the carotid bodies and changes in their cellular components as do lowland-adapted animals.

The blood volume in llamas and alpacas approximates 63.5 ml/kg body weight, or 6.35% (6.35% to 8.65%) of body weight. This is lower than in other mammals.

A number of electrocardiographic studies have been reported since the first edition. With slight variations of patterns,[3,28,34] camelid electrocardiographs are similar to those of ruminants.

SPECIAL DIAGNOSTIC PROCEDURES

The diagnosis of cardiovascular disorders requires special procedures, including auscultation, electrocardiography,[3,8,22] angiography, cardiac catheterization,[38] cinefluoroscopy, and ultrasonography. The principles are the same in camelids as in other species. Employment of these procedures requires special technical help, special equipment, and interpretation by a specialist.

DISEASES

Congenital Defects

Congenital defects of the heart include ventricular septal defects (Figure 16.1), atrial septal defects, patent ductus arteriosus, transposition of the aorta and pulmonary artery, persistent aortic trunk, and persistent right aortic arch (Chapter 22). Septal defects are not unusual, and a familial tendency may be suspected, but proof of such is lacking. The author has seen two

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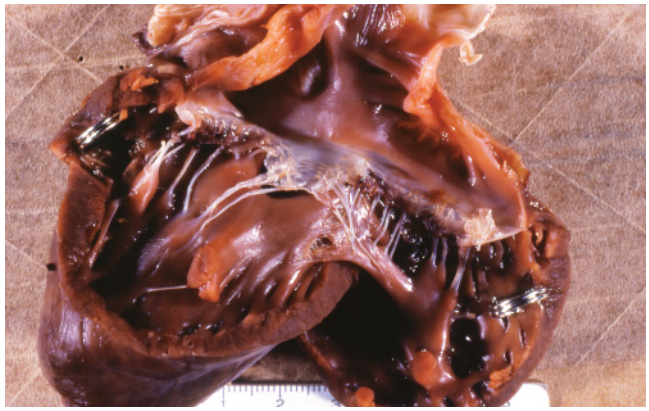

Figure 16.1. Ventricular septal defect in a llama.

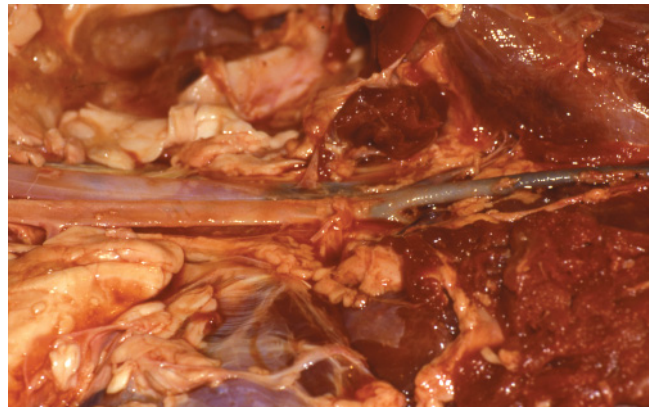

Figure 16.3. Iliac thrombosis in a llama.

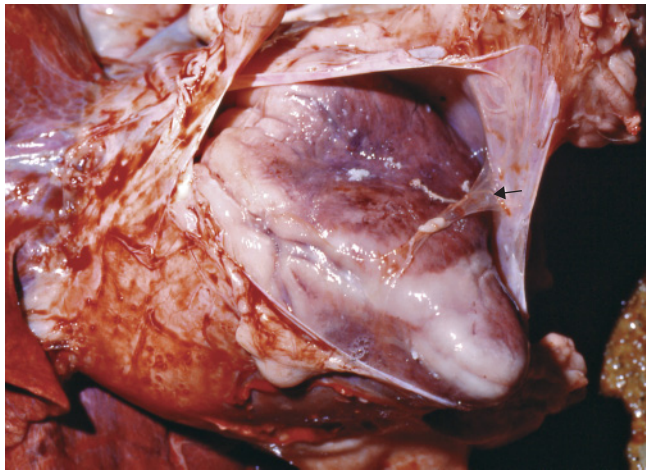

Figure 16.2. Fibrinous pericarditis and epicarditis in a llama.

transpositions of the aorta and pulmonary arteries, a rare defect in any other species.

Infectious Diseases

No unique infectious diseases of the cardiovascular system have been reported. Valvular endocarditis has been diagnosed, but the causal organism was not isolated. Fibrinous pericarditis and epicarditis occur in septicemic conditions (Figure 16.2). Viral encephalomycarditis is discussed in chapter 7.

Parasitic Diseases

Cysts of *Sarcocystis* spp. have been found in the myocardium (Chapter 8). Larval forms may be found in the vascular system transiently. Llamas may be infested with larvae of *Parelaphostrongylus tenuis*. The adult parasites are found in the subdural venous sinuses of the brain of the white-tailed deer. It is not known whether the parasite completes its life cycle in the llama, an aberrant host.

Miscellaneous Diseases

Selenium/Vitamin E Deficiency

Selenium/vitamin E deficiency may cause necrotic myocarditis with cardiac failure or fibrosis, which may decrease the efficiency of the heart (Chapter 2). Ischemic gangrene of the extremities, including the tail, ears, and limbs, may occur with frostbite or ingestion of ergot. Iatrogenic myocardial infarction has been reported.[29]

Iliac Arterial Thrombosis (Saddle-block Thrombus)

Etiology. Although thrombi may form in situ, the most likely source of the thrombus is from vegetative valvular endocarditis (Figure 16.3).

Clinical Signs. A female llama developed unilateral caudal paralysis. The left rear limb was cool to the touch, and no pulse could be detected in any of the arteries of the limb. If occlusion of an iliac artery is complete, the limb will begin to necrose in a few minutes to hours (Figure 16.3).

Diagnosis. Clinical signs are highly suggestive. Definitive diagnosis must include angiography.

Therapy. The source of the thrombus must be determined. A clotting panel should be conducted. If vegetative endocarditis is present, it must be treated with broad-spectrum antibiotics and anticoagulant agents (coumarin). A thorough examination is necessary to detect other areas of the body that may have been affected by release of multiple thrombi. Surgical removal of the thrombus is employed in dogs and cats if the thrombus is detected early.

Endocarditis

Endocarditis is relatively uncommon in camelids; however, cases of thrombolic endocarditis have been reported in alpacas.[9] (Figures 16.4A and B)

Clinical Signs. No signs were pathognomonic for endocarditis. Abdominal distention caused by ascites

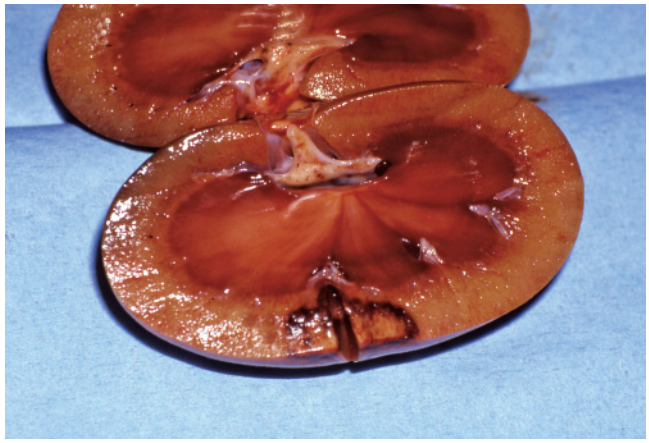

Figure 16.4A. Hepatic thrombosis caused by endocarditis.

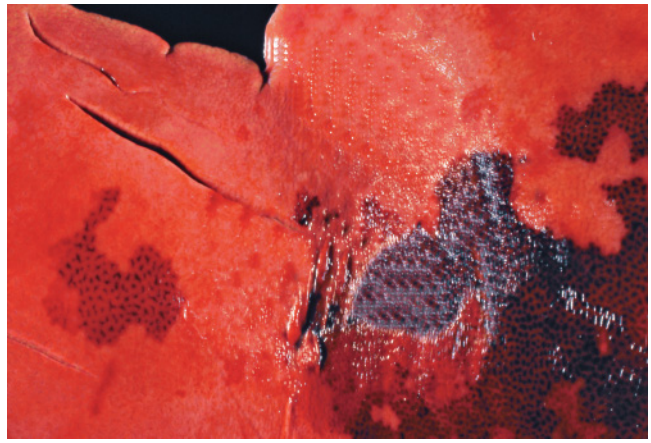

Figure 16.4B. Renal thrombosis caused by endocarditis.

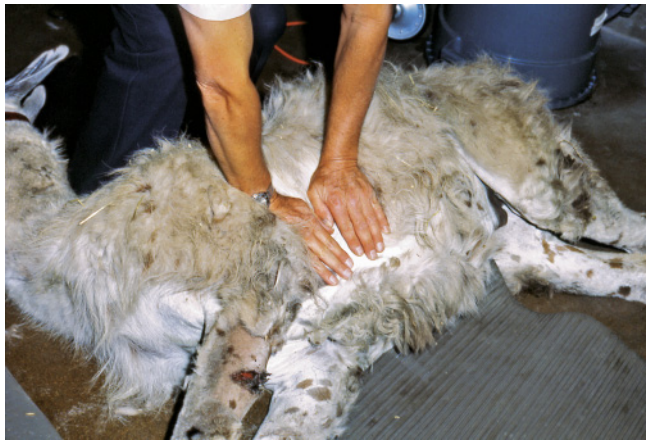

Figure 16.5. Site for cardiac massage in a llama.

was the most consistent sign in a series of cases. Ultrasound examination confirmed the presence of peritoneal and pleural fluid and hepatomegaly (eight of ten animals had liver fluke infestation *Fasciola magna.* Only two of ten animals had cardiac murmurs. Hematology and serum biochemistry values were variable and non-diagnostic. Serum enzyme values reflected hepatic involvement consistent with liver fluke infestation.

Diagnosis. Careful cardiovascular examination including echocardiographic evaluation are the most definitive antemortem diagnostic procedures. At necropsy, the lesions of endocarditis are evident, including fibrinous thrombotic material adhered to the endocardium and underlying endocarditis. Diagnostic procedures may not provide an etiologic diagnosis.[14]

Pericarditis[2,20,32]

Etiology. Pericarditis may be caused by bacterial or viral infection, trauma, neoplasia, and immune-related disorders. Septicemic diseases may have pericarditis as a secondary response. Extension of pulmonary infection may cause pericarditis.

Clinical Signs. Signs include tachycardia, muffled heart sounds, and tachypnea. Thoracic radiographs reveal a diffuse opacity of the thoracic cavity, dorsal deviation of the trachea, and lack of a cardiac silhouette.[2] An echocardiogram revealed pericardial effusion with alterations in the ventricles indicative of cardiac tamponade. Pleural effusion was also evident.[2] Gross lesions are seen in Figure 16.2.

Diagnosis. Signs, radiographs, ultrasonography, and echocardiography are indicated but the definitive diagnosis is confirmed by pericardiocentesis. The fluid is likely to be a modified transudate unless the etiology is of an infectious nature.

Management. Treatment may depend on the etiology of the condition. Broad-spectrum antibiotics are indicated, but removal of the pericardial fluid is vital to prevent cardiac tamponade and congestive effects on circulation. Pericardial lavage was successfully employed in a llama.[2] Pericardectomy was accomplished in a llama cria with chronic pericardial effusion, but the treatment failed to overcome complicating cardiac anomalies.[32]

CARDIAC MASSAGE

Cardiac massage is rarely required in camelids, but if indicated, place the animal preferably on its right side. Pull the left leg forward and press on the chest behind the triceps muscle, Figure 16.5.

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17

Reproduction

Murray E. Fowler, DVM, and P. Walter Bravo, DVM, PhD

South American camelids (SACs) occupy some of the most inhospitable habitats in the world. The progenitors of the four species now found in the Andean countries developed unique reproductive strategies that enabled them to survive and flourish. Reproductive anatomy and behavior are similar in both SACs and OWCs. During the last few years there has been an increasing interest in studying camelid reproductive biology, and as a result useful information is available to students and clinicians.

Consideration of the reproductive strategies of vicuñas and guanacos may aid in understanding problems of llamas and alpacas. The anatomy and physiology of the reproductive system will be addressed, followed by a discussion of infertility.

REPRODUCTIVE STRATEGIES

Vicuñas

Vicuñas are limited to ranges at high elevations in the Andes of Peru, Bolivia, and the border between Argentina and Chile. They are nonmigratory. Family groups are highly territorial. The dominant male defends a family feeding territory against intrusion by strangers, either male or female. The borders of the territory are strictly delineated. The family group also maintains a sleeping territory, to which the family retreats in the evening. Corridors between sleeping and feeding territories may be neutral territory for several family groups.

Vicuñas are seasonal breeders in Peru, with 90% of all births occurring between mid-February and early April.[69,120,123,126,127,129] Though females will be bred within two weeks of parturition, only approximately half of the females will give birth the following year.[15]

Vicuña social structure consists of family groups of two to five females and their crias, which are dominated by a single breeding male, and male groups (bachelor herds), varying from two to 100 animals. Both male and female offspring are driven from the family group by the dominant male (males at four to nine months of age, females at ten to twelve months). Females rarely conceive before they are two years old, but a few become pregnant at twelve to fourteen months.[69]

Males driven from the family group form temporary associations with other males and do not attempt to establish their own territories until four years of age. Juvenile females that are driven from the family group may attempt to enter another family group, but they may be repulsed by the male so as to maintain the optimum size of the group. Unattached females ultimately become part of a new male's family group.

In the Andes, more than 90% of vicuña births occur in the morning hours. This is probably an evolutionary adaptation to the weather patterns of that area.[69] The frequent afternoon storms could interfere with adequate drying of the cria before the nightly drop to near-freezing temperatures occurs.

Guanacos[56,69]

Reproductive strategies and the social organization of guanacos are similar to those of the vicuña. However, guanacos have a broader distribution in more diverse habitats, from sea level to 3,000 m.[134] Guanacos are more flexible than vicuñas; some populations are sedentary, whereas others are migratory. Migratory behavior necessitates some modifications of family group and male group patterns. In addition to these, aggregates of females may form during the winter, while each breeding male remains alone in the family group territory. There may even be mixed groups of

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males and females that come together during the winter.

Reproduction takes place only within the family group. Juvenile males and females are evicted from the family group, as in the vicuña strategy, but the age differs at which dispersal occurs. In guanacos, juvenile males and females are weaned at approximately seven months but begin nursing again when the next cria is born, continuing until evicted at thirteen to fifteen months of age. Thus both juveniles and the crias of the year are present in a family group for a few months. It is thought that enhanced social maturity is fostered by this biologic strategy.[134]

Wild Bactrian Camels

Wild Bactrian camels inhabit some of the most inhospitable areas of the world, with climactic extremes, sparse vegetation, and only salty water to drink. The males are highly territorial, controlling 12 ± females and their offspring. Males fight one another for breeding rights, and such fights may be vicious. Breeding and birthing is seasonal to coincide with the availability of forage for mother and calf.

NORMAL REPRODUCTION[62,65,109]

Male Llamas and Alpacas[24,34,78]

Anatomy[24,60,97]

The penis of the male SAC has a prescrotal sigmoid flexure (Figures 17.1 to 17.2B). The nonerect penis of a small llama, 135 kg (300 lbs.), may vary in length from 36 to 45 cm (14 to 18 in.), measured from the tip to the ischial arch. The diameter is relatively small, 0.8 to 1 cm (0.3 to 0.4 in.), at the region of the preputial reflection, increasing to 1.2 to 2 cm (0.5 to 0.8 in.) at maximum diameter near the ischial arch. The penis does not expand appreciably in diameter during erection but becomes firm and elongated. The erect alpaca penis varies from 35 to 40 cm (14 to 16 in.) in length.[111]

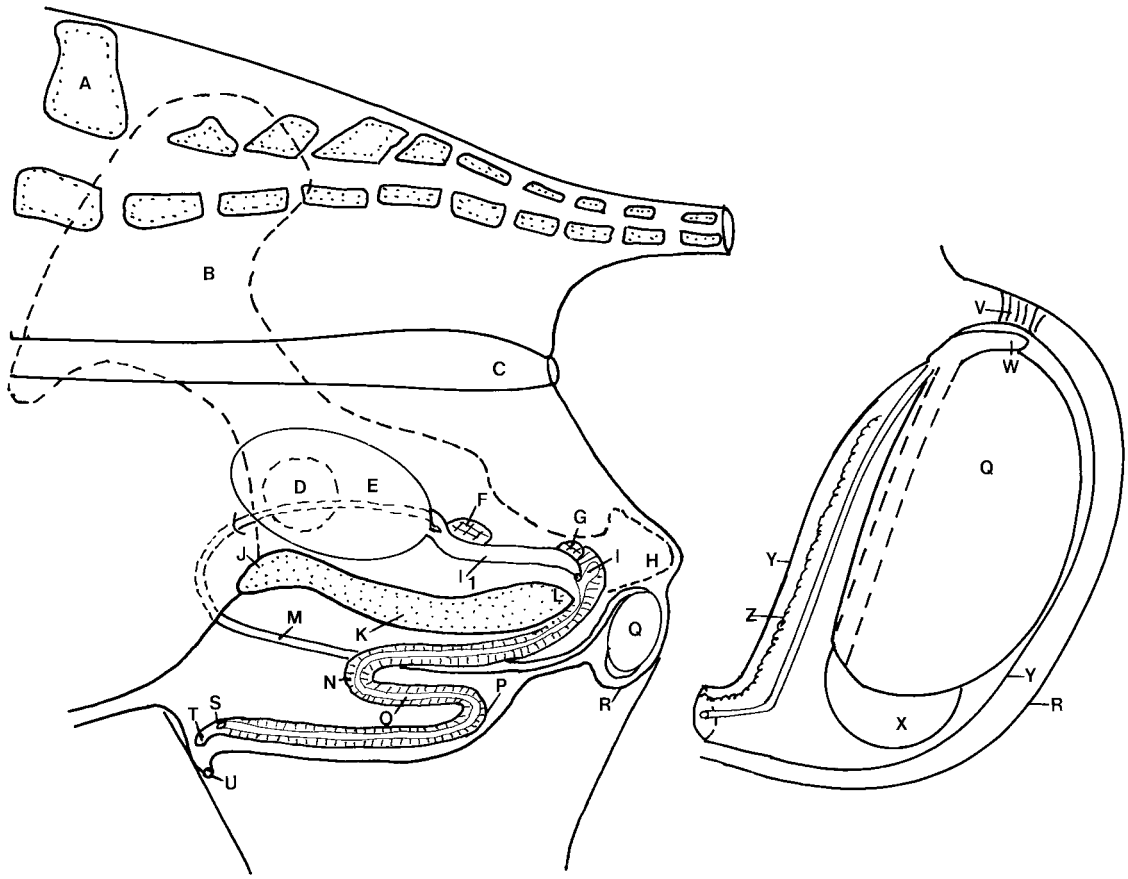

Figure 17.1. Diagram of lateral view of male camelid genitalia. (A) Spinal column, (B) ileum, (C) rectum, (D) acetabulum, (E) bladder, (F) prostate gland, (G) bulbourethral gland, (H) ischiatic tuberosity, (I) dorsal urethral recess, (I_1) pelvic urethra, (J) brim of pelvis, (K) pubic bone, (L) ischiatic arch, (M) ductus deferens, (N) corpus cavernosum penis, (O) penile urethra, (P) sigmoid flexure of the penis, (Q) testicle, (R) scrotum, (S) urethral orifice, (T) cartilaginous tip of the penis, (U) prepucial orifice, (V) scrotal ligament, (W) tail of epididymis, (X) head of epididymis, (Y) tunica vaginalis communis, (Z) pampiniform plexus.

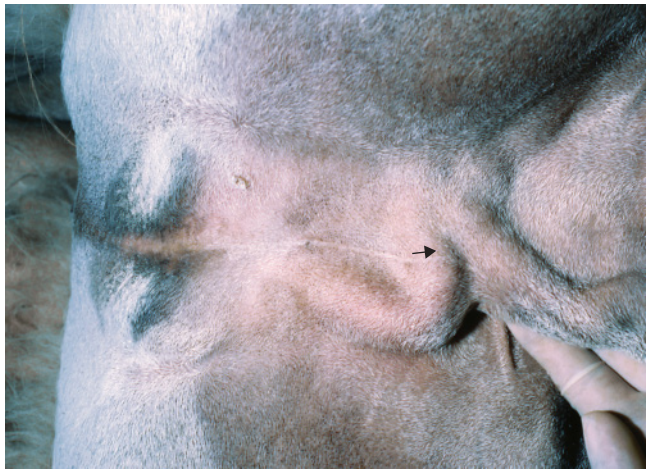

Figure 17.2A. Sigmoid flexure of a llama penis.

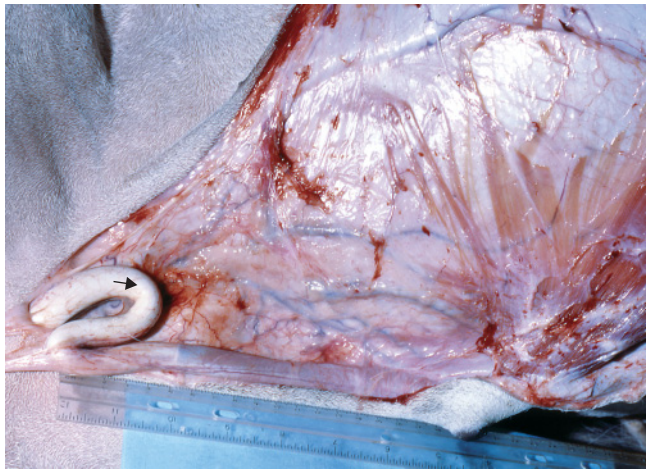

Figure 17.2B. Sigmoid flexure of a llama penis.

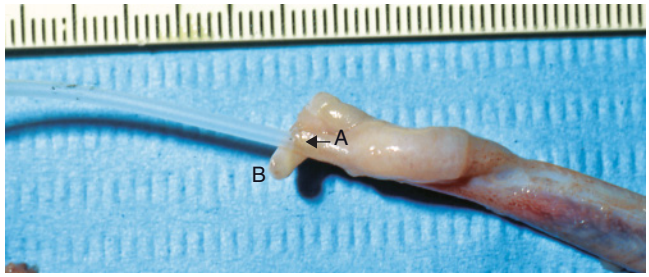

Figure 17.3. Tip of penis.

In the adult male, the glans is long (9 to 12 cm) and tapers to the tip, merging into a firm cartilaginous projection that has a slight clockwise curvature (Figure 17.3). The distance from the tip to the preputial reflection is approximately 11 cm (4.3 in.). The penis penetrates the cervix and deposits semen into the uterine horns.[132] The cartilaginous tip may be an adaptation to facilitate threading the penis through the ringed cervix.

The prepuce of the SAC is triangular and nonpendant. In an unaroused male, the prepuce is directed caudally, and urine is projected backward between the hind limbs from a semisquatting position. When the male is sexually aroused, the cranial preputial muscles pull the prepuce cranially, and the penis is extruded in the same manner as in the bull. Movement is controlled by a double set of preputial muscles, cranial and caudal. Both arise from the cutaneous trunci muscle and lie on either side of the linea alba. These muscles are relatively larger in SACs than in domestic livestock.

The prepuce of the neonate and juvenile male adheres to the glans penis, making it impossible to extrude the penis from the prepuce until as late as two to three years of age (Figures 17.4 to 17.7). In a study of male alpacas, 8% to 12% were free of adhesions at one year of age, 70% to 78% were free at two years, and by the age of three, all were free of adhesions.[24] When selecting future alpaca sires in Peru, early maturation is desirable, and yearlings that are free of adhesions are preferred. If a male is castrated prior to the onset of testosterone production, these adhesions may not be released or may be only partially released (Figure 17.8).

Urethral catheterization is difficult in the SAC male neonate because the glans cannot be extruded. Under sedation, the urethral opening may be protruded through the folds of the prepuce. In neonates, the cartilaginous process of the adult male glans penis is not present, and the tip of the penis is blunt. Attempts to peel the prepuce from the glans usually causes hemorrhage. Successful catheterization may be accomplished by gently probing for the urethral orifice with a number 3 French urinary catheter.

In the adult, the urethral opening on the glans is located at the base of the cartilaginous projection (Figure 17.3) and will accommodate passage of a number 5 or larger French catheter. The pelvic urethra is approximately 7 mm in diameter, narrowing as it enters the penile segment. The urethra narrows further at approximately the level of the preputial reflection. Uroliths may lodge at this site.

It is impossible to pass a catheter through the tip of the penis into the bladder because of a dorsal urethral diverticulum just cranial to the ischial arch because the catheter would automatically be directed into the diverticulum (Figure 17.9). Even with a finger inserted into the rectum to deflect the catheter ventrally, bypassing this recess would be strictly fortuitous.

Accessory sex glands are limited to a pair of bulbourethral glands and a small prostate (Figure 17.10). There are no seminal vesicles. The bulbourethral glands lie dorsally and laterally to the pelvic urethra

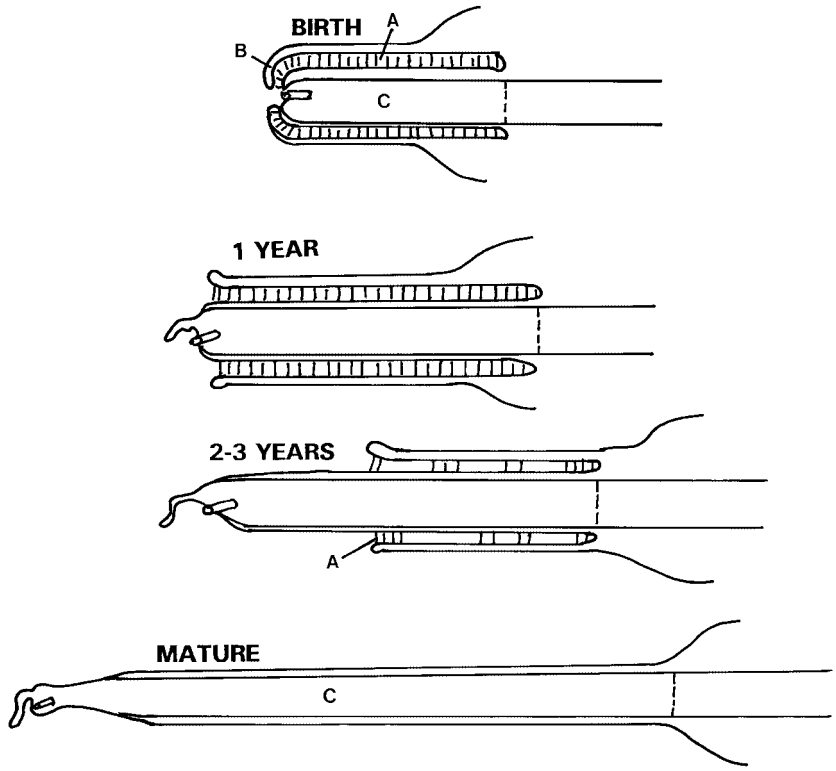

Figure 17.4. Diagram of prepucial adhesions of the camelid. (A) Prepucial adhesions, (B) prepuce, (C) penis.

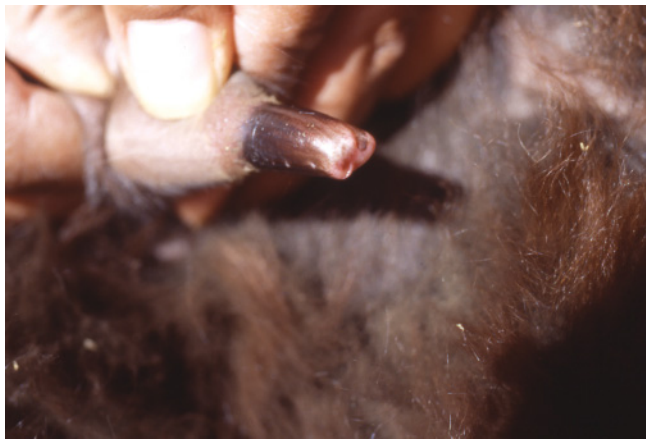

Figure 17.5. Inability to protrude penis, three-month-old alpaca.

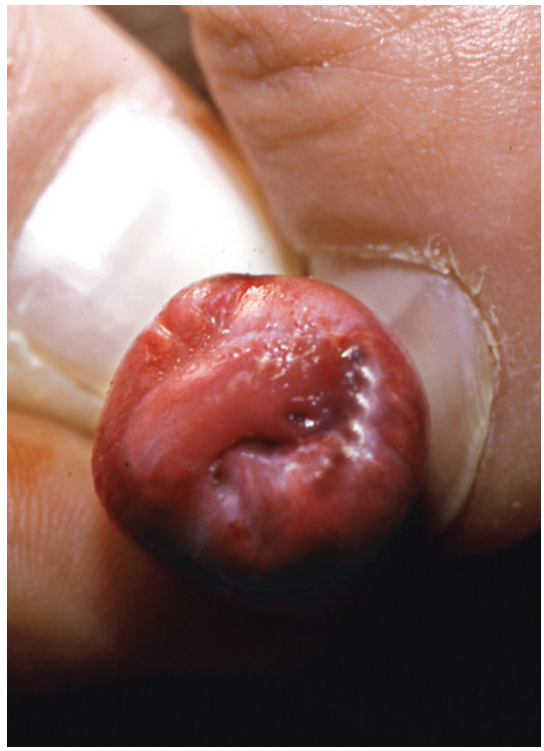

Figure 17.6. Prepucial adhesions of a yearling llama.

at the ischial arch and are approximately 2 cm (0.8 in.) in diameter. The prostate gland is small (3 × 3 × 2 cm) and situated on the dorsum of the urethra near the trigone of the bladder.

The retractor penis muscles arise from the anal sphincter muscle and pass around the ischial arch in close association with the body of the penis. In a pelvic urethrostomy, the paired muscles must be parted or pulled to one side to enhance exposure of the urethra. The distal insertion of the muscle on the tunic of the penis is at the level of the preputial reflection.

The testicles of llamas and alpacas are roughly ovoid (Figure 17.11), but a few may be globose. There is great variation in the size of the testicles.[122] The ultrasonographic anatomy of the testicles is important

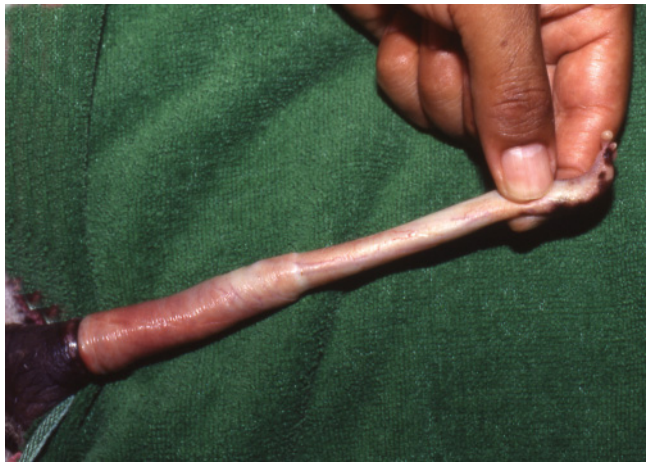

Figure 17.7. Adhesions of prepuce to the glans penis.

because this technique is used daily and it provides a clear view of the testicular parenchyma. The testicular width and depth may be adequately measured, but not so the length because the length of the probe may not be enough to cover the whole testicle. The most important feature to look for is the echogenecity of the parenchyma. Figure 17.5 Depicts a tranversal view of a normal testicle. The parenchyma's appearance is homogenous and hypoechoeic without any hyperechoeic and nonechoeic spots. It is useful to move the ultrasound probe along the length of the testicle to see any variation in echogenicity. This sequence gives a better assessment of the testicular parenchyma. In a longitudinal view the testicular mediastinum may be clearly assessed and observed. Testicular size influences fertility of the male SAC (Table 17.1). Forty-five

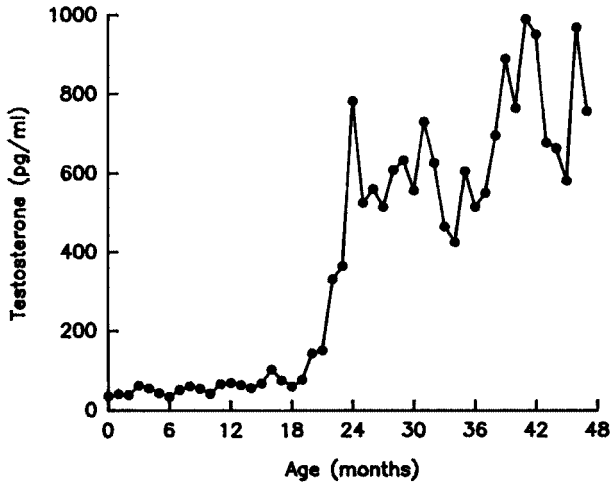

Figure 17.8. Testosterone concentration in the male llama from birth to maturity. (Bravo et al. 1992, Proc. 12th ICAR 3:789).

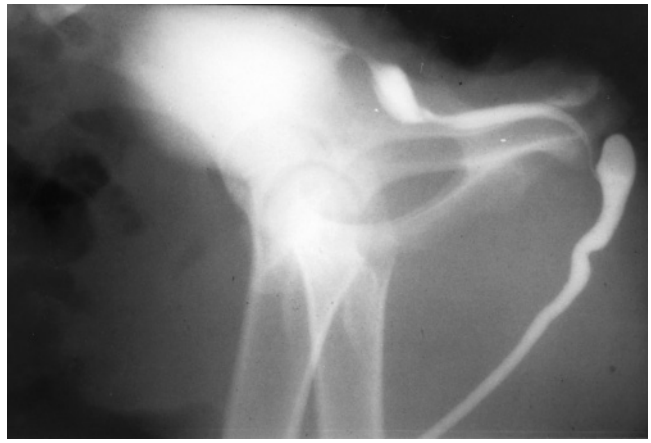

Figure 17.9. Radiograph of male urethra containing contrast medium. Notice the dorsal urethral recess at the ischial arch.

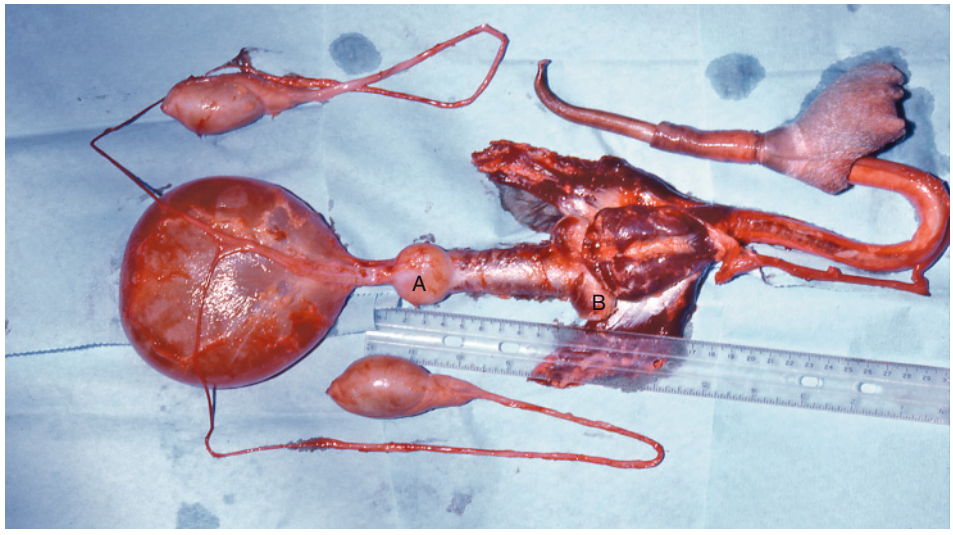

Figure 17.10. Male camelid genitalia. (A) prostate gland, (B) bulbourethral gland.

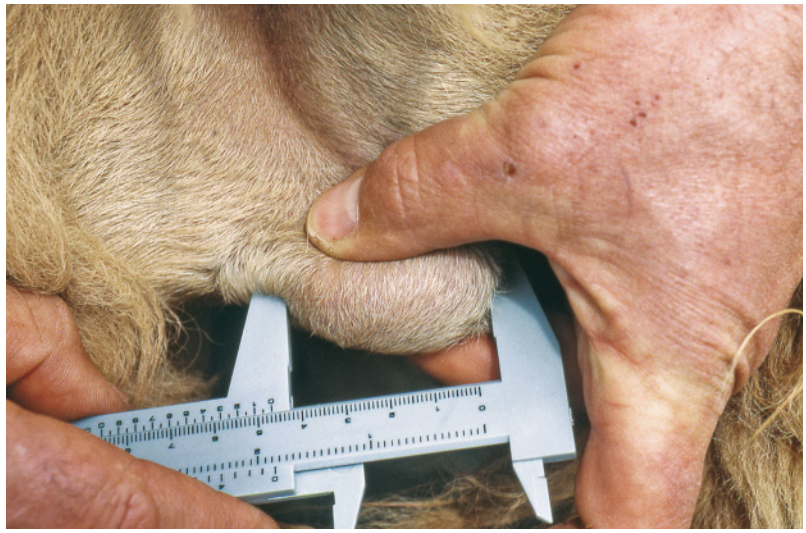

Figure 17.11. Measuring the length of a testicle.

Table 17.1. Mean testicular size (length × transverse axis) and weight (g) in llamas, alpacas, and vicuñas of different ages (n = 158 alpacas, 54 llamas, 6 vicuñas).

Age (months)	*Llama*[a]		*Alpaca*[b]		*Vicuña*[c]
	Size (cm)	*Weight*	*Size (cm)*	*Weight*	*Size (cm)*
6	2.4 × 1.4	na[d]	na	na	na
12	3.4 × 2.3	5.1	2.3 × 1.5	2.9	na
18	3.5 × 2.6	14.0	2.8 × 1.9	6.6	na
24	3.9 × 2.3	17.4	3.3 × 2.2	9.9	na
30	4.4 × 2.5	17.8	3.6 × 2.3	13.9	na
36	4.5 × 2.7	18.2	3.6 × 2.4	13.6	na
Sires	5.4 × 3.3	na	3.7 × 2.4	17.2	3.3 × 1.9

[a] From Bravo et al. 1992[40].
[b] Source: Bravo, unpublished data.
[c] From Urquieta et al. 1991[262].
[d] na = not available.

percent of females became pregnant when bred to males with small testicles, in contrast to 75% pregnancies in females resulting when bred to males with normal size testicles.[24] Testicular size is also important in bulls. In Peru, future alpaca sires are selected at one year of age. The length of the testicles at this time ranges from 1.1 to 1.4 cm. The males with larger testicles are chosen.

The nonpendant scrotum is situated just ventral to the ischial arch (Figures 17.12A and B). The testicles and the scrotum protrude only slightly from the surrounding surface. Having the testicles closely apposed to the body may be an important adaptation to prevent accidental castration during male-male fighting. Also, the skin over the scrotum is thick, providing additional protection for the testicles. Scars are often seen on the scrotum, a result of lacerations from the bites of other males.

The testicles are usually in the scrotum at birth but are tiny, flabby, and difficult to palpate. Adult testicles should be turgid, not mushy, and freely moveable within the scrotum. The epididymis is closely attached to the testicle, and the head, body, and tail can be palpated after some practice. The head of the epididymis is cranial-ventral and the tail caudal-dorsal as it is in the boar but not in the bull, ram, and stallion, where the head of the epididymis is dorsal. The epididymis should be resilient, with no nodules.

The microscopic characteristics of the testicles and epididymis are essentially the same as for other livestock species.[61] The diameter of seminiferous tubuli is between 174 and 237 microns.[37,92] The shape of SAC spermatozoa is similar to that of livestock. Characteristics of SAC semen, from some limited studies, are noted in Table 17.2.[35] The cycle of the

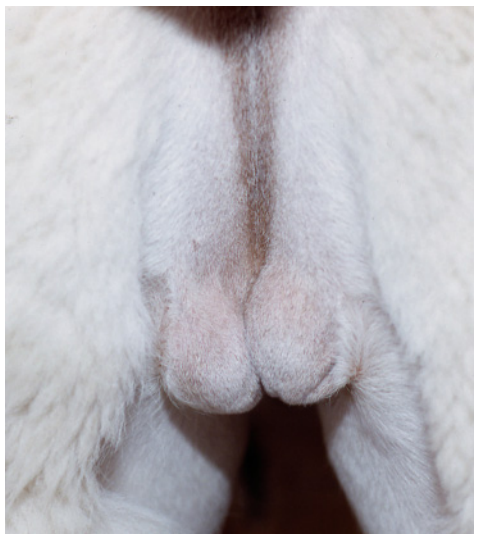

Figure 17.12A. Scrotum of an alpaca.

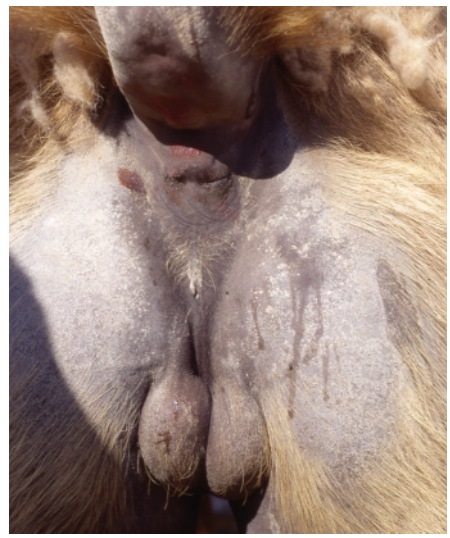

Figure 17.12B. Scrotum of a camel.

Table 17.2. Semen characteristics of llamas and alpacas.

Characteristics	*Llama*	*Alpaca*	*Range*
Volume (ml)	1.6 ± 0.8	1.9 ± 0.4	0.8–3.1
Concentration (number/mm^3)	1,388,000 ± 54,000	147,500 ± 7894	82,500–250,000
Total concentration (millions)		289,000 ± 15,200	61,785–750,000
Motility (%)	61.7 ± 23.5	85 ± 5.2	69.0–91.1
Live sperm (%)	82.7 ± 7.3	69.6 ± 3.7	58.0–83.1
Normal sperm (%)	na[a]	75.9 ± 2.1	70.6–84.1
Abnormal heads (%)	na	6.7 ± 1.4	2.6–12.6
Abnormal tails (%)	na	12.3 ± 0.8	9.4–15.2
Cytoplasmic droplets (%)	na	3.8 ± 0.8	1.3–7.3

[a] na = not available.

seminiferous epithelium for the male llama has been described as eight stages with relative frequencies as 9.8%, 12.5%, 17.7%, 14.1%, 5.8%, 8.1%, 13%, and 18.9%, respectively.[63]

Physiology[74,118]

To understand SAC breeding behavior and physiology, the general biology of the wild species should be reviewed (also see Chapter 1). Wild SAC males are basically territorial polygamists.[69] A successful breeding male will defend a territory and a set number of females against all intruders, male or female. Fights between males may be vicious. Although domestication of the llama and alpaca has diminished territorial behavior, if alpaca or llama males are kept near vicuñas, they revert to wild behavior and become difficult to manage as a domestic herd.

Testicular Function. The testicle has two functions: spermatogenesis and hormone production. As in other mammals, testosterone is responsible for the development of secondary sexual characteristics such as greater muscular mass, appearance, and libido. Testosterone concentrations in the male llama and alpaca have been determined from birth to adulthood (Figure 17.8).[32] Basal concentrations of testosterone are observed from birth up to nineteen to twenty months of age, after which testosterone levels increase rapidly, reaching adult values when males are approximately two years old. This increase in testosterone coincides with exacerbated sexual libido in male llamas and alpacas.

When male alpacas were separated from females as a management practice, testosterone concentration dropped to 3,900 pg/ml in contrast to 9,000 pg/ml during the breeding season. Similar observations were made for male vicuñas: 288 pg/ml of testosterone in the nonbreeding season in contrast to 1,009 pg/ml in the breeding season.[128] This apparent change in testosterone concentration has not been observed in the United States in llamas maintained year-round

with females. Testosterone concentrations fluctuated between 900 and 1,200 pg/ml throughout the year.[32]

Assessment of spermatogenesis is difficult. When SAC semen contacts air, it is converted to a gelatinous mass, which defies attempts to produce a satisfactory smear for evaluating motility and morphology. It is now known that the gel appearance of semen is due to the presence of a thick gelatinous material secreted mostly by the bulbo-urethral glands. Several protocols for semen collection have been devised; the most successful and suitable for clinical purposes is collecting it from the vicinity of the external cervical os of the female after breeding has taken place. For research purposes the most adequate method of semen collection is using an artificial vagina (AV), suitable for camelids, which has enabled collection of reliable semen samples. Briefly, this modified AV has two important characteristics: a stricture on the latex liner simulating the cervix and an electric heating pad to maintain liner warmth for the length of copulation.[28]

Semen is whitish-cream in color, becoming reddish when collected after copulation from the female, with a pH between 7.2 and 7.8. Most of the biochemical components of the seminal plasma of alpacas[70] and llamas are similar to those of other livestock species, with the exception of small amounts of fructose (5 mg/dl) and citric acid (4.3 mg/dl), perhaps as a result of the absence of seminal vesicles. Fructose concentrations did not vary among semen samples collected from February through September,[71,100] nor between males of three and six years of age. Semen characteristics of alpacas and llamas appear in Table 17.2.[35]

Ovulation does not take place for at least twenty-four hours after copulation in SACs. The gelatinous semen mass probably provides some protection to the sperm until liquefaction of semen takes place. It is not known when liquefaction (liquefaction is the process of a solid becoming liquid, and since ejaculated semen is not solid, rather a gel, the correct term should be degelification) takes place in vivo, but in vitro semen samples begin to liquefy eight hours following collection. The influence of season on seminal production has been partially defined in llamas. Male alpacas lose libido when maintained in continuous association with females. Also, in the northern hemisphere, sperm production in llamas and alpacas decreased to nil values after undergoing heat stress during the summer, then later returned to normal. Normal spermatozoa morphology was around 74% during spring in contrast to 24% in summer, rising again to 71% during the autumn. In addition, the proportion of abnormal spermatozoa increased during a period of approximately one to one and a half months. Thus, a temporary loss of fertility during the summer may be normal.

Sexual Behavior[38]

Male llama crias (neonates is a term only for crias for the first week) begin play fighting at a few weeks of age (Figure 17.13). Some precocious weanlings will rear up on females and, if they can catch a nonpregnant female lying down, may attempt to mount her. Weanling males and females should be kept separate from each other because, in a few instances, males less than a year of age have impregnated females. Llama breeders in North America may tend to push young males into breeding too soon. Some individuals are anatomically capable of breeding when they are one and a half to two years old, but until they are three years old, most individuals lack sufficient libido and necessary body weight to force the female down and to breed vigorously. In some very young males, the testicles do not produce viable sperm, and there may still be preputial adhesions that prevent full extension of the penis.

Guanaco males do not become territorial until they are four to six years of age.[69] This does not mean that male guanacos are physically incapable of breeding earlier than this, but social and behavioral restrictions prevent earlier breeding.

In Peru, male alpacas are put into breeding service at three years of age;[122] however, it would be unwise to pronounce a male llama or alpaca as infertile (precluding any anatomic defects) before the age of three and a half to four years.

Both male and female (to a lesser extent) camelids exhibit flehmen, but in a slightly different manner than other mammals (Figures 17.14A and B). Many males

Figure 17.13. Young male llamas play fighting.

Figure 17.14A. Alpaca male exhibiting flehmen.

Figure 17.14B. Llama exhibiting flehmen.

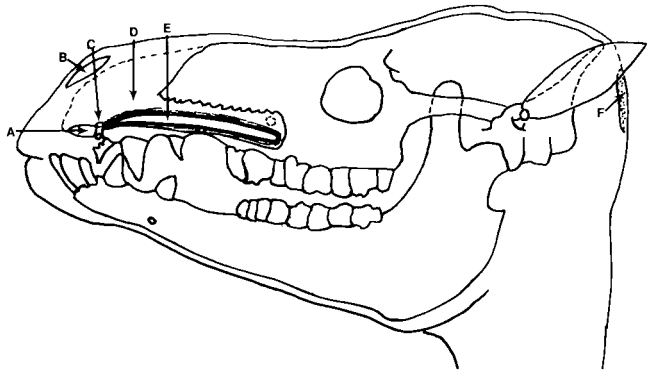

Figure 17.15. Diagram of the vomeronasal organ in a camel. (A) Palate, (B) opening to the vomeronasal organ, (C) nostril, (D) nasal septum, (E) Vomeronasal organ, (F) poll gland.

approach a dung heap recently used by a female. He sniffs the area and then lifts his head and elevates the upper lip and opens the mouth slightly. The upper lip does not curl back as it does in other artiodactylids. Some, but not all, males sniff the perineal area of the female. The volatile pheremone enters the duct of the vomeronasal organ for evaluation (Figure 17.15).

The usual breeding behavior of a male SAC placed in the same enclosure with a receptive female begins with pursuit of the female. Some females become recumbent immediately, but most move away from the male until he rears up and puts pressure on the hindquarters in an attempt to force the female to lie down (Figures 17.16A and B). He may do this repeatedly if she continues to move away. The rearing is not an attempt to copulate but merely to force the female to lie down. The size and body weight (not fat) of the male is important in successful copulation, as is the libido of the male. A male with good libido will chase a female for at least five minutes before giving up.

Once the female is recumbent, the male positions himself at her rear, in a half-sitting position, and begins intromission (Figures 17.17 to 17.19). The pelvic thrust of SACs is not as vigorous as it is in other domestic livestock. It may be difficult to ascertain whether or not intromission has actually occurred unless the penis is palpated or the fiber coat (wool) is pulled to the side to allow observation. With some experience, it is possible to perceive whether or not the male is in proper position. The male may be too far behind the female's pelvis. This is often observed when the male first mounts a recumbent female. At this stage, erection begins, and the tip of the penis may be visible. As the male begins to adjust his position by shifting from side to side or forward, working closer to the female, erection is completed. The perineal area of the female will be probed with the penis, almost as if the male is exploring to find the vulva. Upon close observation of the penis at this stage, it should be noted that the glans may alternately assume a corkscrew and a straight shape. This alternate configuration of the glans also occurs during electroejaculation and should not be construed as penile malformation. Perhaps this shape

Figure 17.16A. Male llama attempting to force a female to sternal recumbency prior to copulation.

Figure 17.16B. Beginning copulation, not close enough for intromission.

changing aids in penetration of the vulva and in threading the penis through the cervix.

When intromission has occurred and effective copulation is underway, the male will be positioned with his pelvis close to the pelvis of the female, and the hocks of the male are side to side to the hocks of the female. A practical approach to assess penis intromission into the genital tract of females is following the penis of the male and if it is positioned into the vulva of the female. This is done from behind the animals copulating. It could be done from the side, but the presence of the legs of the male and his heavy

Figure 17.17. Proper breeding position in alpacas.

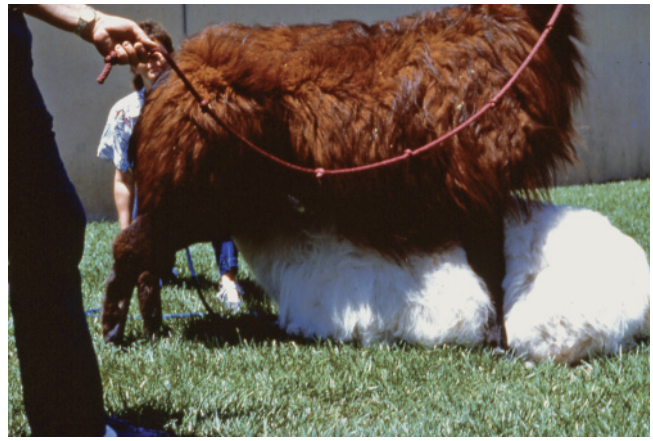

Figure 17.18. Inexperienced llama male attempting to breed a female.

coat make it more difficult to evaluate than from behind.

The male constantly vocalizes during copulation with a guttural sound that is called "orgling."[a] The exact source of the sound is unknown, but it may be the result of vibrations of the relaxed, elongated soft palate as air rushes past during rapid breathing.

Copulation may continue from five to fifty minutes, with an average time of twenty to thirty minutes. The elapsed time of copulation and conception rates have not been correlated. It is impossible to ascertain exactly when ejaculation occurs, because ejaculation is continuous from two to three minutes after intromission until the end of copulation. There is no tail flagging, as in the stallion, or hyperpelvic thrust, as in the bull. It is known however, that ejaculation is a continuous

Figure 17.19. Camels breeding.

process (pulsating ejaculation). It is common to see the male reposition himself during copulation by moving slightly backward, then shifting right or left before moving forward again. It is probable that he is changing the position of the penis from one uterine horn to ejaculate into the other. It has been demonstrated that the penis reaches the uterine horns.[132]

Occasionally, a male will cease copulation for no apparent reason, stand up, and begin the positioning sequence once again. When copulation is completed, the male rises and wanders off. The penis will have been retracted into the prepuce. The female may remain recumbent for a few minutes or may also rise and move away.

If the male and female are left together, copulation may be repeated the same day but usually not until the next day, and perhaps it will be repeated on a third day. If ovulation has occurred, the female will refuse to lie down and will spit at the male to discourage him. This is called "spitting off."

If other receptive females are in the enclosure, a vigorous male may breed as many as five or six females in one day. Reports from Peru indicate that a vigorous alpaca male may breed as many as eighteen times in one day, for the first three days of the breeding season, but this activity quickly drops off on subsequent days, and if the male is left with the females for ten to fourteen days, he will ignore newly receptive females.[24] The length of copulation decreases when males are subjected to consecutive breeding on the same day. Males will copulate for sixteen minutes for the first two or three matings, in contrast to twelve minutes for a fourth mating and ten minutes for a sixth.[38] In addition, the length of copulation also decreases on consecutive days of breeding. Males copulate for seventeen minutes on the first day, thirteen minutes on the third day, eleven minutes on the seventh day, and six minutes on the eighth and ninth days of breeding. The number of copulations affects the fertility rate of the male: the percentage of pregnant females was 77% for males copulating two to four times a day, in contrast to 59% for males copulating six times a day. Likewise, the fertility of the male decreases with the progressive number of days of breeding: 88% of females became pregnant on the first day of breeding, 84% on the third day, 71% on the seventh day, and no pregnancies resulted from breedings on the ninth day.

A male SAC may take a dislike to a particular female and refuse to breed her. He may even become somewhat aggressive with her. Varying degrees of aggressiveness have been observed in breeding males. Some will even "rape" pregnant females. Others are so shy that they almost have to find a female lying down before they attempt to copulate. Shy males may be intimidated by dominant females. Libido may be an inherited trait, so it may be undesirable to use a shy breeder.

Intermale aggression can be violent and damaging to both combatants, especially if each has a full complement of canine teeth. The objective of the fight is to establish dominance. The males may run at each other, rear up, and ram chests, each trying to knock the other off balance. If an advantage has been gained, the stronger will try to position himself to the side, with his neck over the neck of his opponent, in an attempt to force him to the ground. If the weaker animal cannot dislodge the stronger, he may reach around and bite the leg of his opponent. This "necking" may continue for some time, with each alternating at being the stronger.

Males may position themselves side by side in a head-to-tail position. Each male may try to bite the other on the hind legs and the scrotum. As one male is being bitten, he may lean into the aggressor and almost sit down on the aggressor's head. Severe wounds may be inflicted by intact canine teeth.

Particularly aggressive males may charge from the side in an attempt to knock an opponent down or rear up and come down on the body to force recumbency. They may also bite at the neck, ears, or jaws of an opponent. During the fight, the animals may constantly spew stomach contents at one another.

The interaction is over when one of the males remains recumbent in a subordinate posture. Both males will stand with their mouths partly open for several minutes after breathing has returned to normal (Figure 17.20). This is not a form of open mouth breath-

Figure 17.20. Open mouth stance (pouting) assumed by male llama after aggressive encounter with another male.

ing, necessary to increase air flow into the lungs, but rather a behavioral trait exhibited after any aggressive or disagreeable interaction. The authors have also observed this behavior after a strenuous restraint episode.

The anatomy and basic reproductive physiology of the male camel is similar to that of NWCs.[67,133] However, both Bactrian and dromedary male camels undergo a seasonal rut period characterized by heightened aggression, secretion from the poll glands, dribbling of urine, flicking of the tail to splash urine onto the back, and vocalization. Weight loss is usual because the male expends so much energy in establishing and defending his harem. Inappetence may also be a factor in weight loss. Rut may last for weeks or months.

The poll gland is a scent gland that is unique to OWC males. The gland secretes a brownish to black fetid fluid that mixes with dirt and urine during rut to mat the hair on the back. If allowed to do so, a male camel in rut may roll and attempt to rub the poll gland against the dirt to scent-mark. The testicles increase in size during rut, accompanied by an increased testosterone level in the blood.

Male dromedary camels have a diverticulum on the lingual aspect of the soft palate (dulaa, dulla, dulah) which may be inflated with air and be extruded from the oral cavity during rut. The camel produces a gurgling sound accompanied with excessive salivation and foaming. This behavior is a threat to other males. The dulaa may be protruded at times other than rut, when the camel is excited or angry. It may also come out of the mouth during anesthesia induction and may cause alarm to the novice camel anesthetist. At such time the dulaa may simply be pushed aside and intubation completed.

Sexual behavior may be evident in dromedary males at two years of age, but puberty usually doesn't occur until the animal is two to five years old. Dromedary males are usually not used for breeding until they are five years of age. Breeding may continue for ± twenty years.

Bactrian males mature more slowly, with sexual behavior beginning at three years of age and puberty at five to six years of age. Copulation is in the recumbent position as in SACs. In dromedaries the duration of copulation may be seven to twenty-two minutes; in the Bactrian the duration is only an average of three minutes. In contrast, alpacas copulate for five to fifty minutes and llamas for five to sixty-five minutes.

Camel semen is similar to that of SACs and is white and creamy to grayish. Camel semen develops the same gelatinous consistency shortly after ejaculation as does that of the SAC. The volume of semen produced varies from 1 to 15 ml. Semen pH is alkaline (7.6 to 8). Motility varies from 40% to 80%.

Disease Conditions in Male Camels

These are similar to those seen in SAC males. Specific conditions noted in the references include scrotal laceration, orchitis,[11] testicular hematoma, testicular hypogenesis, cryptorchism,[101] neoplasia, and epididymal cysts.

Female Llamas and Alpacas

Anatomy[10,20,21,57]

See Table 17.3 for a comparison of measurements of llama and alpaca reproductive organs.

Vulva. The labia of the vulva lie in a slightly slanted to vertical position approximately 4 to 6 cm ventral to the anal orifice (Figure 17.21). In aged or emaciated llamas or those with congenital malformations of the reproductive tract, the labia may lie in a more horizontal plane (vulvar shelving) (Figure 17.22). The vulvar orifice (a slit) is approximately 3 to 5 cm long. The labia do not swell during the reproductive cycle. The depth of the vestibule (vulva to the hymen) is approximately 3 to 4 cm.

Vagina. The vagina, from the hymen to the cervix, varies in length from 15 to 25 cm (6 to 10 in.) and is approximately 5 cm in diameter.

Cervix. The llama cervix is 2 to 5 cm long and 2 to 4 cm in diameter. The cervix has two to three ringlike structures (Figures 17.23A and B). The cervix relaxes with estrogen stimulation and opens for several days, then closes and remains closed, which precludes the insertion of uterine catheters and or insemination pipettes.

Uterus. The SAC uterus is bicornate. The body of the uterus is short (3 to 5.5 cm) and approximately the same diameter throughout. Externally, the length is

Table 17.3. Measurements of female SAC reproductive organs.

	Llama[a] *(cm)*	*Alpaca*[b] *(cm)*
Vulva		
Length of labia	5	
Depth of hymen	6–8.5	
Vagina		
Hymen to cervix		
Length	15–21	13.4 ± 2
Diameter	5	3.4 ± 0.7
Cervix		
Length	2–5	
Diameter	2–4	
Rings	2–3	2–3
Body of uterus		
Length to septum	3–5.5	3.05 ± 0.71
Diameter	3–5	
Horns of uterus		
Tips of septum to tip of horn	21–22.5	
Bifurcation to tip of horn	8.5–15	7.9 ± 1.3
Diameter	2.5–4	
Oviduct	10.5–18.3	20.4 ± 4.2
Ovary		
Right		
Length	1.3–2.5	1.6 ± 0.3
Depth	1.4–2	1.1 ± 0.2
Width	0.6–1	1.1 ± 0.2
Weight		1.87 ± 0.94 g
Left		
Length	1.5–2.5	1.6 ± 0.3
Depth	1.5–2.5	1.1 ± 0.2
Width	0.5–1	1.1 ± 0.2
Weight		2.4 ± 1.34 g

[a] Measurements collected at University of California, Davis.
[b] Data from Bravo and Sumar 1984, Some anatomical parameters of the reproductive tract in alpacas, Resumenes Invest., Univ. Nac. Mazor, San Marcos (Lima).

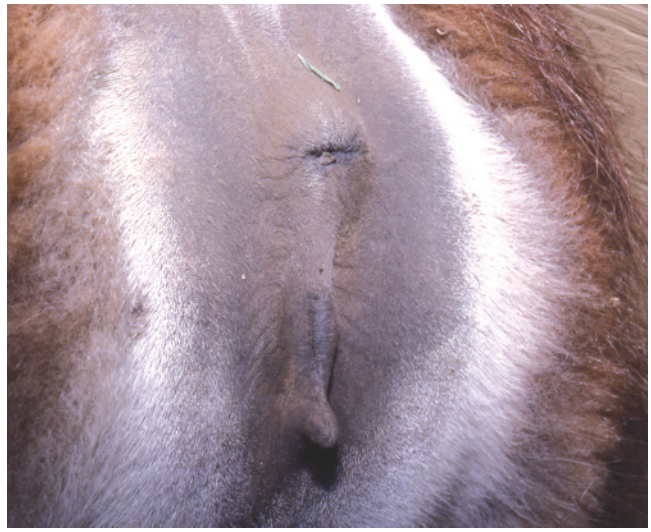

Figure 17.21. Llama perineum.

Figure 17.22. Perineum of llama with abnormal, near horizontal relationship of vulva to anus (shelving).

not apparent because the horns are fused together for a short distance, being separated by a septum (Figures 17.24 to 17.26).

The left uterine horn may be slightly larger than the right, especially after the first pregnancy. The uterine horns of a llama, from the end of the septum to the tip of the horn on the convex surface, vary from 20 to 22.5 cm in length; in alpacas the length is 6 to 7 cm. The proximal segments of the horns diverge at approximately 180 degrees. The distance from the notch at the divergence to the tip of each horn varies from 8.5 to 15 cm (3.3 to 6 in.). The diameter of the horns varies from 2 to 3 cm with age and the number of previous pregnancies. The tip of the uterine horn is rather blunt.

Uterine mucosa is similar to that of other species, consisting of a columnar epithelial layer. The fibrous tissue of the submucosa is more dense than in the mare, and there are fewer and smaller uterine glands.[106] There are no glands in the cervical submucosa, and the epithelial layer is more cuboidal. This information is necessary to enable differentiation of the site from which a biopsy specimen has been taken.

The penis penetrates the uterine horns. Thrusting during copulation causes mechanical friction and may initiate an inflammatory response in the uterine endometrium, including hemorrhage and edema in some cases.[44] This inflammatory reaction is normal, but if a female is bred more than once during a follicular

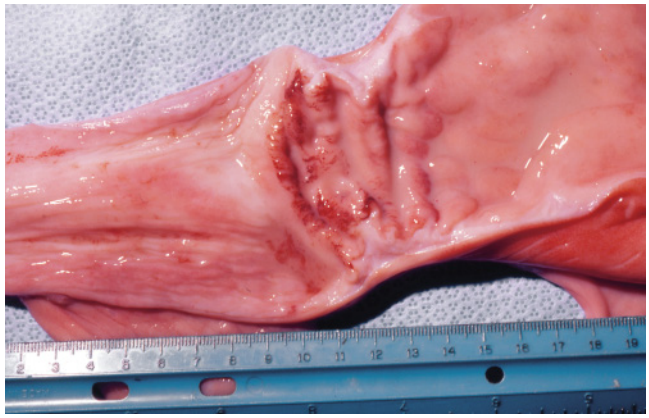

Figure 17.23A. Llama cervix showing the rings.

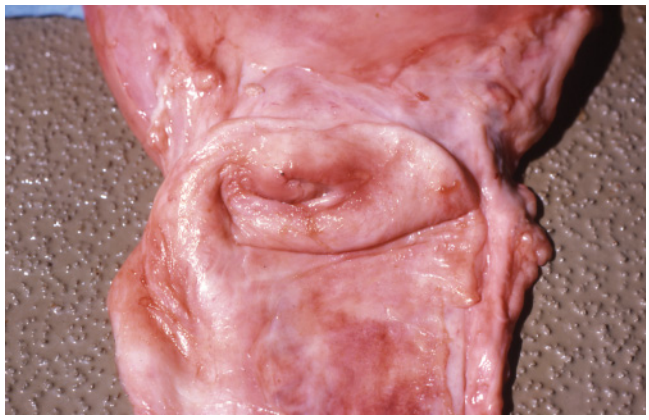

Figure 17.23B. External cervical os.

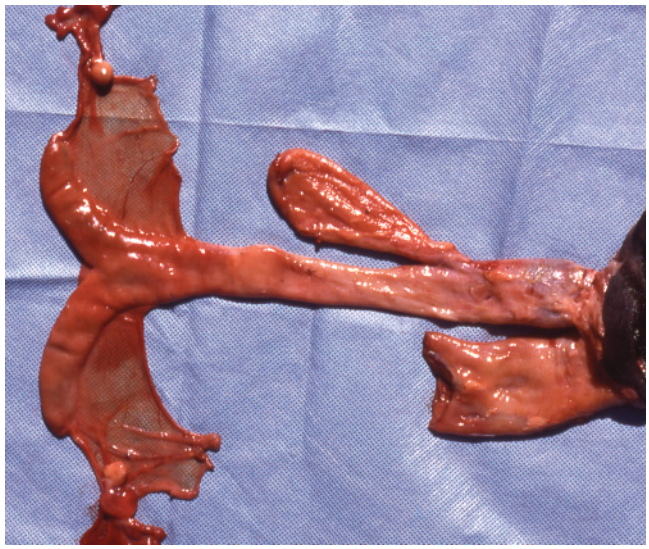

Figure 17.24. Female reproductive tract of a nulliparous alpaca.

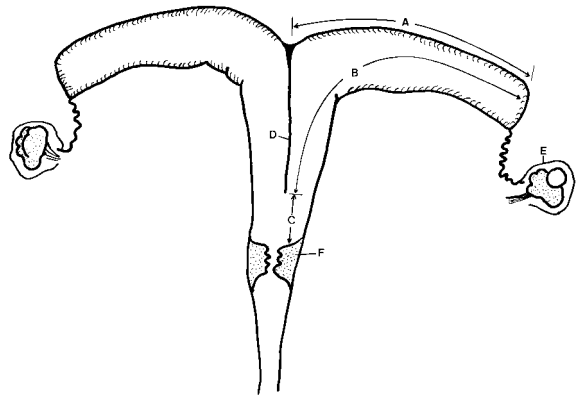

Figure 17.25. Diagram of camelid female reproductive tract. (A) Palpatable length of uterine horn, (B) actual length of uterine horn, (C) body of uterus, (D) intercornual septum, (E) ovarian bursa, (F) cervix.

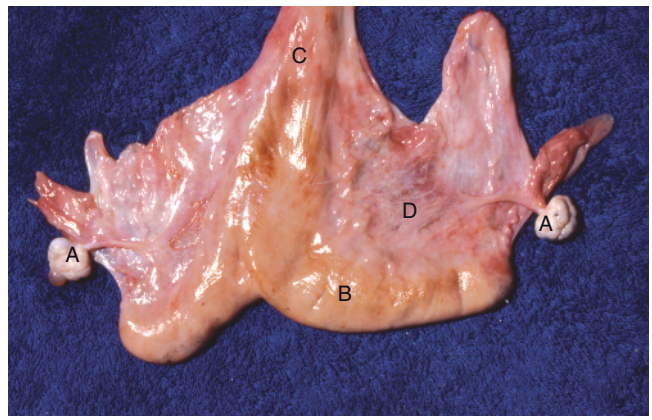

Figure 17.26. Uterus and adnexa of a multiparous llama. (A) ovary encased with the ovarian bursa, (B) left uterine horn, larger than the right, (C) cervix, (D) mesosalpinx.

phase, the damage to the uterus may be correspondingly greater. Histologically, there may be lymphocyte infiltration, plus plasmocytes and eosinophils. Increased branching of the endometrial glands of the endometrium may also be observed.

Oviducts. The oviducts are rather long and tortuous and are embedded within the mesosalpinx. The duct measured 10.5 cm in one llama, with a diameter of 3 mm. Bravo and Sumar measured the ducts of alpacas at 20.4 ± 4.2 cm.[55] The oviduct empties into the tip of the uterine horn on a papilla that contains a sphincter (Figure 17.27).[24] It is impossible to flush fluid retrograde into the oviduct from the uterus, but the reverse is possible.

The ovarian bursa is large (2.5 × 2.5 × 5 cm) and completely surrounds the ovary. Ovarian structures must be palpated through the bursa, or the bursa may be gently moved to expose the surface of an ovary. At laparoscopy, the bursa is lifted off the ovary with forceps.

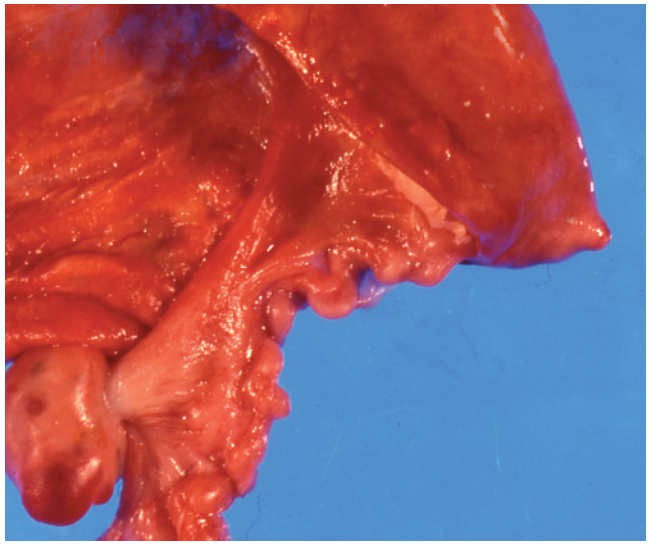

Figure 17.27. Tip of uterine horn everted to expose oviduct papilla.

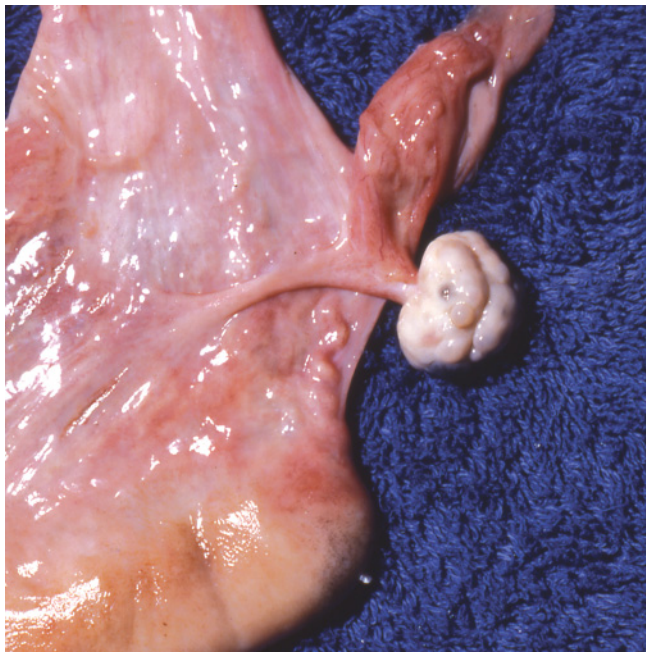

Figure 17.28. Inactive llama ovary.

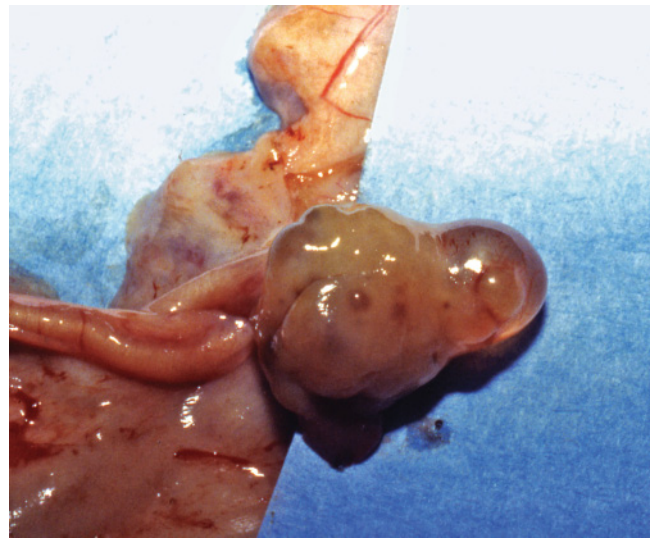

Figure 17.29A. Llama ovary with an ovulatory follicle.

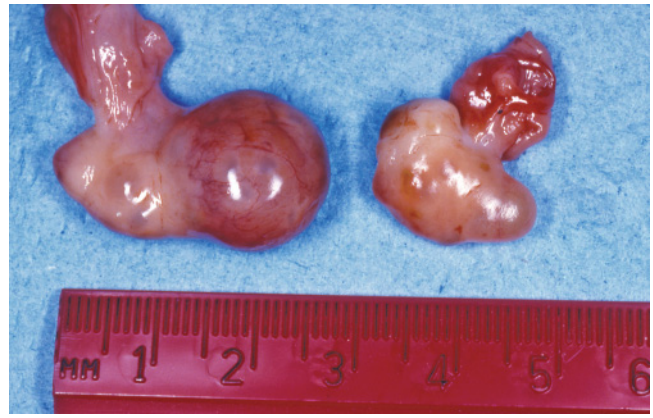

Figure 17.29B. Llama ovary with a corpus luteum.

Ovary.[29,57] The ovaries of the llama are ellipsoid to globular, while those of the alpaca are more globular. Numerous follicles, varying in size from 2 to 10 mm, can be seen on the surface of the mature, normal ovary (Figures 17.28, 17.29A and B).[10] A follicle ready for ovulation may reach 12 mm. Any follicle larger than 13 mm is considered to be pathologic.[6,40,112] Measurements of the ovaries from six llamas were as follows: length, 1.3 to 2.5 cm.; depth, 1 to 2 cm; and width, 0.5 to 1 cm. These measurements may be compared with those of alpacas (Table 17.3). Llama ovaries have not been weighed, but alpaca ovaries weigh approximately 2 g. A composite of sonograms of the reproductive organs of a female alpaca has been assembled to give the reader a real perspective of the organs visualized using ultrasonography (see Figure 17.52 later in chapter).

Puberty

The assessment of puberty in female SACs is difficult because of the lack of an estrous cycle. Nonetheless, some progress has been made in assessing onset of sexual maturity based on body weight and ovarian activity. Female alpacas twelve to thirteen months of age with body weights of 33 kg successfully delivered and cared for crias. Under South American conditions in which nutrition is based on native pastures, 60% of yearlings attained weights of 33 kg, and of these, 70% conceived following copulation. At the next breeding season, there was no difference in the body weights of these females compared with those of older females.

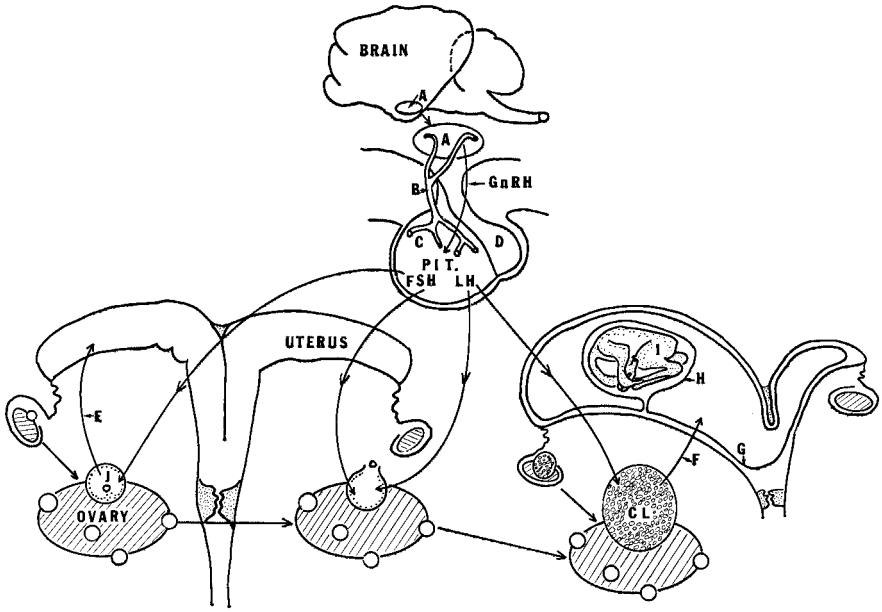

Figure 17.30. Diagram of the endocrine relationships of the female reproductive tract. (A) Hypothalamus, (B) blood flow from the hypothalamus to the pituitary, (C) anterior pituitary, (D) posterior pituitary, (E) estrogen stimulation of the uterus, (F) progesterone maintaining pregnancy, (G) chorioalantois, (H) amnion, (I) fetus, (J) ovarian follicle, (CL) corpus luteum.

Percentages of animals reaching maturity based on body weight may be greater where cultivated pastures and supplementation provide better nutrition. Yearlings did not differ from adults in sexual behavior.

Investigations of ovarian activity began with alpacas at ten months of age.[57] Laparoscopic studies of ovarian follicular development showed little difference between yearlings and adults: 65 follicles 6 mm in size were counted in yearlings, and 80 mature follicles in adults.

Follicle development may begin earlier than ten months. When follicle activity was monitored by measuring concentrations of estrone sulfate in urine samples collected weekly from female llamas, beginning with youngsters at five months of age, intermittent increases in estrone sulfate were apparent in the majority of females at six months of age.[32]

Physiology[22,23,25,26,33,35,36,42–45,49,52,65,90,103,105,113,115]

The endocrine relationships to the female reproductive tract are complex. The miracle is that in the vast majority of instances the correct correlation occurs (Figure 17.30).

Camelids have a unique ovarian cycle; they are induced ovulators but their hormonal physiology differs from that of other induced ovulators such as the rabbit or cat.[21,30] The typical follicular phase of the ovarian cycle is charted in Figures 17.31 and 17.32.

The ovarian cycle of a noninduced ovulation species is illustrated in Figure 17.33. Unlike these species, SACs do not have an estrous period. Females are gen-

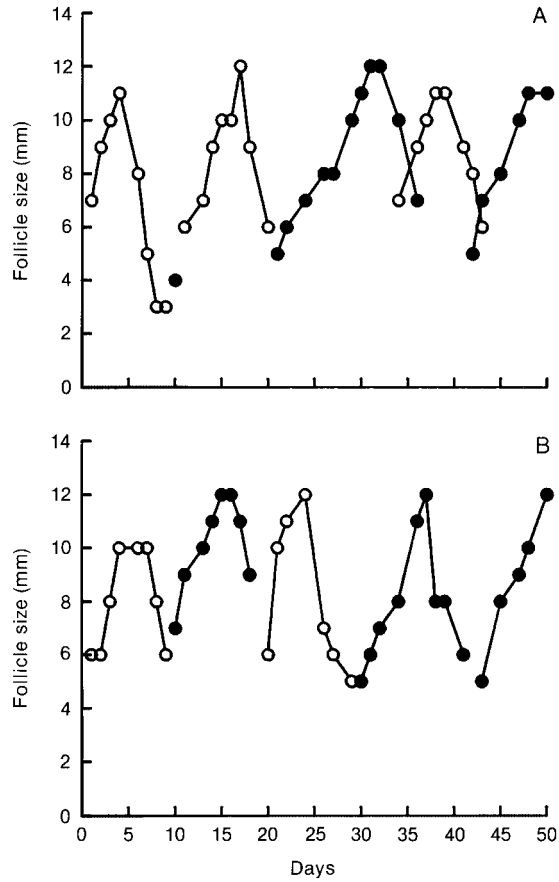

Figure 17.31. Ovarian follicle size in llamas (A) and alpacas (B) for a fifty-day period without contact with a male. (Used by permission from W.B. Saunders Company.)

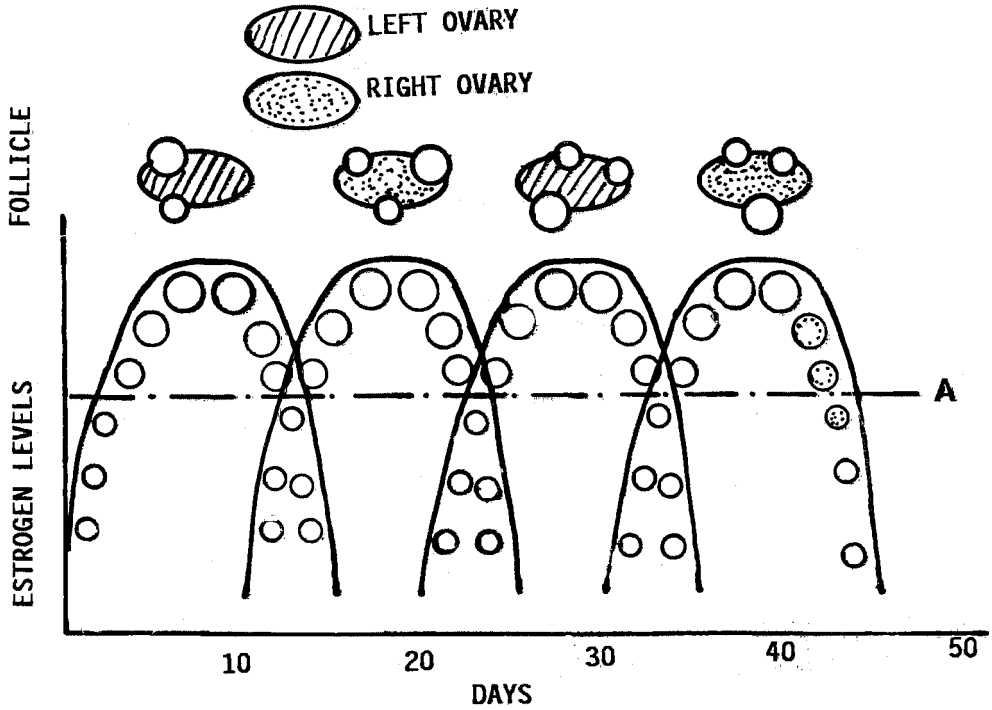

Figure 17.32. Diagram of the follicular wave cycle of an SAC female.

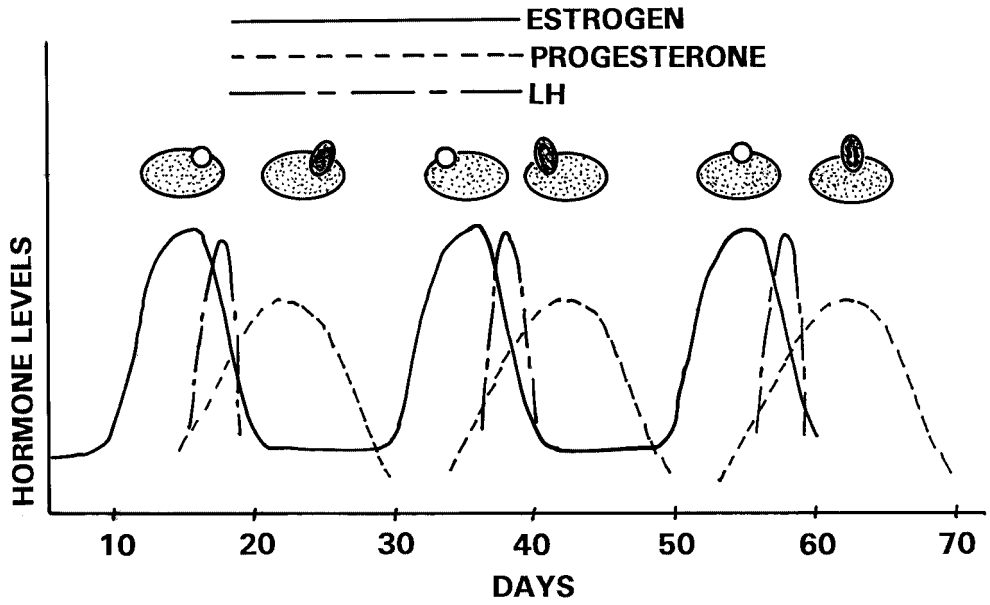

Figure 17.33. Diagram of the hormonal relationships of a noninduced ovulatory mammal. (Bravo et al. 1990a, Biol. Reprod. 43:579–85).

erally receptive to a male unless they have been recently bred or are pregnant. Camels are also induced ovulators and have follicular waves, but the wave interval is longer, so the waves don't overlap to produce estrogen, which would signal constant receptivity (Figure 17.34). Thus, camels exhibit periodic receptivity that appears to be an estrous cycle; however, the follicular dynamics are markedly different from those of a noninduced ovulator species.[2]

Follicular development in llamas and alpacas is similar.[25,26,30,43,47] Follicles grow and regress in an average time of 10–18 days, with ovaries alternating in 85% of the cases in which the presence of mature follicles were observed. Studies of ovarian follicle

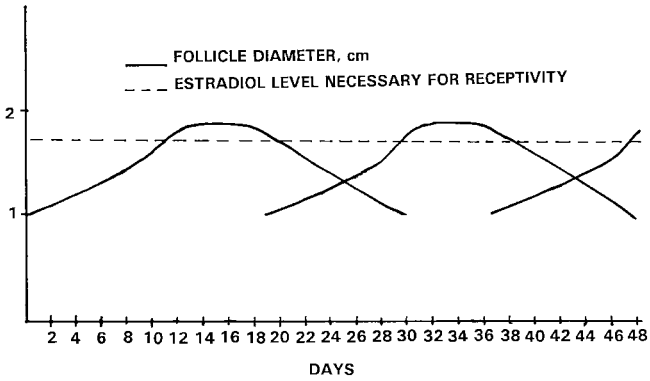

Figure 17.34. Diagram of the follicular wave of a camel. Dips indicate periods of nonreceptivity.

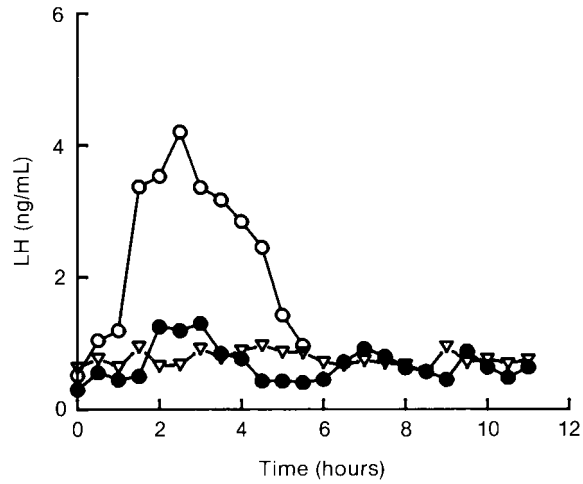

Figure 17.35. Luteinizing hormone response following copulation in a llama. Open circle = ovulating females, closed circle = nonovulating females, and triangle = control females that were not bred. (Bravo et al. 1992. Biol. Reprod. 47(5):884–88).

dynamics using laparoscopy and ultrasonography, correlated with hormone concentrations, revealed that follicles grow, mature, and regress in an overlapping wavelike pattern (follicular wave) (Figure 17.32). These waves are similar in adult and yearling females,[48,49] and apparently these waves extend into the first half of pregnancy. The follicular phase resumes early in the postpartum period of the females, as early as 5–7 days.[7] There was little difference in the number of follicular waves between the nonbreeding[56] and breeding seasons in South America.

For ovulation to occur, a follicle must be >7 mm in diameter. In a female with small follicles (<6 mm) copulation did not trigger ovulation; neither were follicular waves altered. If females with follicles between 7 and 12 mm in diameter were bred, ovulation was induced.[119] If females are bred during the regressive stage, copulation may initiate luteinization of the follicle, without ovulation. Progesterone is secreted, resulting in a short luteal phase lasting five to six days. In contrast, the luteal phase lasts ten to twelve days when ovulation occurs but pregnancy doesn't follow. A short luteal phase may explain why some females refused the male on day 6 or 7 after copulation but were sexually receptive on day 9 or 10 after copulation.

Follicles of seven to nine mm in diameter are mature. Any follicle >13 mm is considered to be cystic. A puzzling aspect of normal reproductive physiology in the female SAC is that sexual behavior or sexual receptivity is not related to the size of follicles. There was no difference in sexual receptivity between females with small follicles and those with mature follicles.

Ovulation in the female SAC may be induced by copulation or the administration of a luteinizing hormone.[31,116] Stimuli for ovulation may include penile penetration through the cervix, orgling of the male, and the male clasping his forelimbs around the body of the female. Peripheral reflex stimulation is transmitted to the hypothalamus, which in turn secretes gonadotropin-releasing hormone (GnRH). GnRH acts on the anterior pituitary to initiate release of luteinizing hormone (LH), which subsequently acts on the follicle, causing ovulation (Figure 17.35).[1] Ovulation usually occurs twenty-six hours following copulation, but sequential observation of the ovaries revealed that ovulation may occur between twenty-four and forty-eight hours following copulation. It is also apparent that a single ovulatory input, either through copulation or administration of GnRH, is enough to induce ovulation. A second copulatory period and/or GnRH administration at six, twenty-four, or even forty-eight hours does not stimulate more release of LH from the pituitary.[51] After the initial surge, the pituitary gland becomes refractory to GnRH stimulation. It should also be noted that a small percentage of females may ovulate spontaneously, complicating breeding management.

If conception does not occur, the corpus luteum (CL) enlarges to its maximum size in seven to eight days and then regresses in response to the action of prostaglandin $F_{2\alpha}$ ($PGF_{2\alpha}$).[114] Pulsatile release of $PGF_{2\alpha}$, detected as its metabolite 15-keto-13,14-dihydro$PGF_{2\alpha}$, begins at days 9 through 12 after copulation. While the CL is active, the development of ovarian follicles is suppressed. As the CL regresses, follicle size increases, returning to normal by three to four days following the demise of the corpus luteum. The female becomes receptive to a male and remains in this state until copulation and ovulation occur again. More rarely, spontaneous ovulation may occur, or an ovarian quiescent state ensues.

Hormone concentrations of estradiol follow follicular dynamics. Estradiol 17-β and estrone sulfate in

plasma or urine are related to follicular waves. Concentrations of 10 to 15 pg/ml of estradiol, 20 to 30 ng/mg of urinary estrone sulfate, correlate with follicle sizes of 7 to 11 mm.

Pregnancy

The ovarian bursa completely encases the ovary so that transport of the ovum to the oviduct is easily accomplished. In livestock species, the ovum reaches the ampulla (midportion) of the uterine tube within two hours, where fertilization takes place and the zygote begins the cleavage process. The zygote remains in the uterine tube for three to six days. South American researchers have indicated that the alpaca embryo reaches the uterus by day 6 or 7 after copulation.

Once the zygote reaches the uterus, it will probably implant in the left horn. More than 95% of successful llama and alpaca pregnancies are implanted in the left horn. Some studies have indicated that 99% to 100% of pregnancies are implanted in the left horn.[121] Because there is an equal distribution of CLs in both the right and the left ovaries, and because each ovary is encased within a bursa, the ovum and subsequent zygote from the right ovary must migrate from the right horn to the left horn or be absorbed.

The precise time of fetal membrane attachment to the uterine mucosa in camelids has been reported recently. The approximation of embryo membrane to the uterus is initiated by twenty to twenty-one days, which coincides with elevated levels of estrone sulfate between twenty-one and twenty-seven days after copulation.[45] In addition, appearance of the embryonic heartbeat twenty-four to twenty-five days after copulation may indicate implantation.

Early embryonic death occurs more frequently in alpacas and llamas than in livestock species.[69] Estimates of 15% to 20% have been reported in North America; however, in South America 50% embryo loss within the first thirty days is seen. Late work involving ultrasonography and progesterone determination during the first forty-five days of pregnancy indicates that 48% of embryo loss occurred in females that had previously failed to conceive for a year and 29% to 30% of embryo loss in lactating or maiden females. The precise cause of early embryonic death is unknown, but is suspected to be heavily related to progesterone. About 42% of embryonic loss was correlated to a drop in progesterone levels. A second 21% of embryo loss occurred when progesterone concentrations dropped two to three days before the disappearance of the embryo (progesterone insufficiency). And, a third 37% of embryo loss occurred when progesterone concentrations remained elevated (persistent corpus luteum). In addition, failure of the right horn to maintain a suitable environment for the conceptus, or chromosomal aberrations may be factors. Infections have not been identified as abortifacients in SAC.

Fetal development proceeds in SACs as it does in other livestock species.[50] Prenatal growth in alpacas has been described.[51] It was found that 85% of fetal weight was gained after 210 days of gestation. The usual length of gestation ranges from 335 to 360 days,[92] with the occasional female carrying a fetus for more than a year. Gestations as long as thirteen months have been reported; however, the authors have dealt with a number of female llamas suspected of pregnancies of more than a year, only to ultimately find a discrepancy in breeding dates. Twinning is rare.[111,125,128]

The growth of the embryonic vesicle is illustrated in Figure 17.36. The growth of the fetus is illustrated in Figures 17.37 and 17.38. The crown-rump length progresses uniformly, with an increase in rate during the last three and a half months. The primary increase in fetal weight and development occurs during the last three and a half months of pregnancy.

Fetal Membranes.[97,103,110,120] Placentation of camelids has been classified as diffuse and epitheliochorial, similar to that of the mare and sow. However, camelid fetal membranes are unique. Early embryonic development of the fetal membranes, from the blastocyst stage to attachment to the uterus, is not unusual. Between seventy and ninety days, the surface of the chorion becomes dotted with numerous half-circular, domed projections that fit into corresponding depressions in the uterine mucosa (Figures 17.39, 17.40). At 20×, a capillary network may be seen on the surface of the domed placentomas. The attachment is tenuous, and the chorion may be peeled from the mucosa with no resistance.

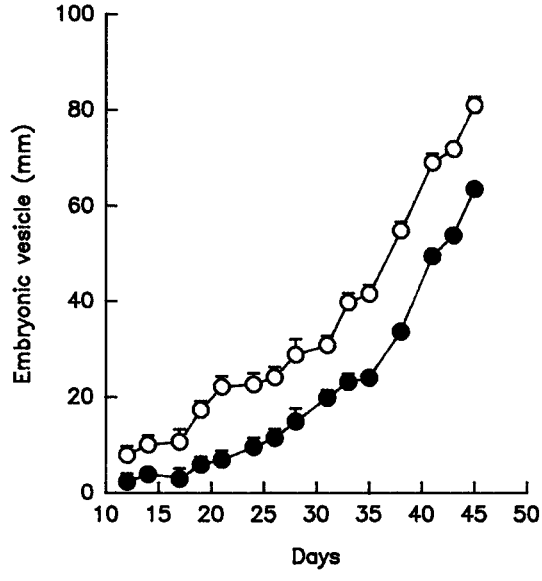

Figure 17.36. Growth of the embryonic vesicles in two alpacas from the day of detection to day 45.

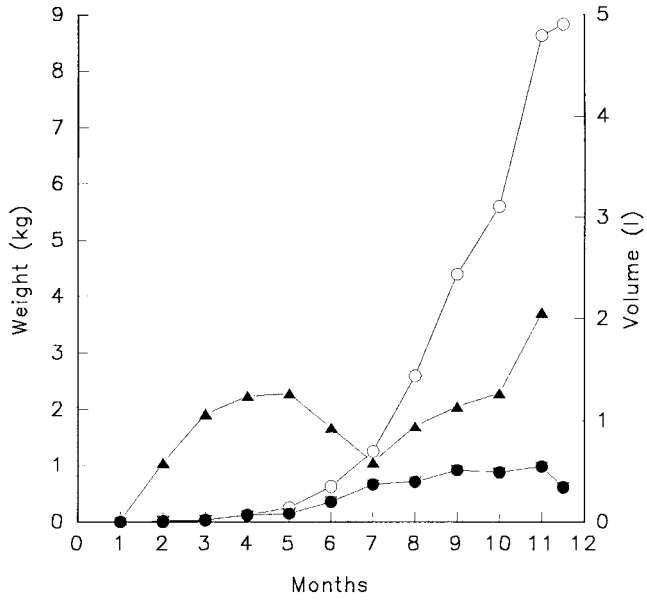

Figure 17.37. Fetal growth (open circle), amniotic fluid volume (closed circle), and weight of the placenta (triangle) during pregnancy. (Bravo and Varela 1993, Anim. Reprod. Sci. 32:245–52).

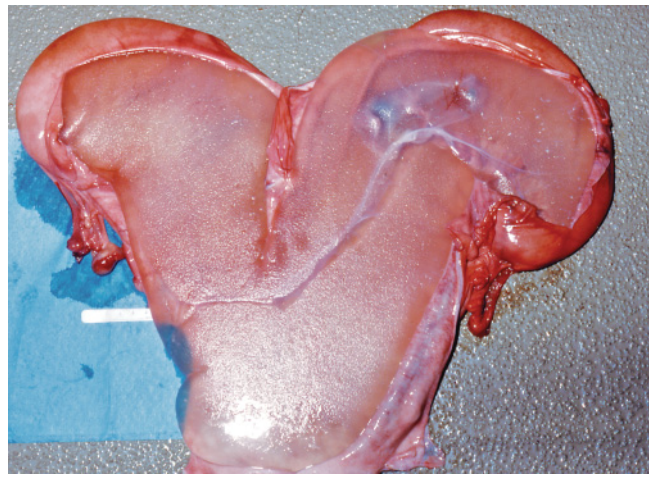

Figure 17.39. Llama placenta, in situ, approximately sixty days gestation.

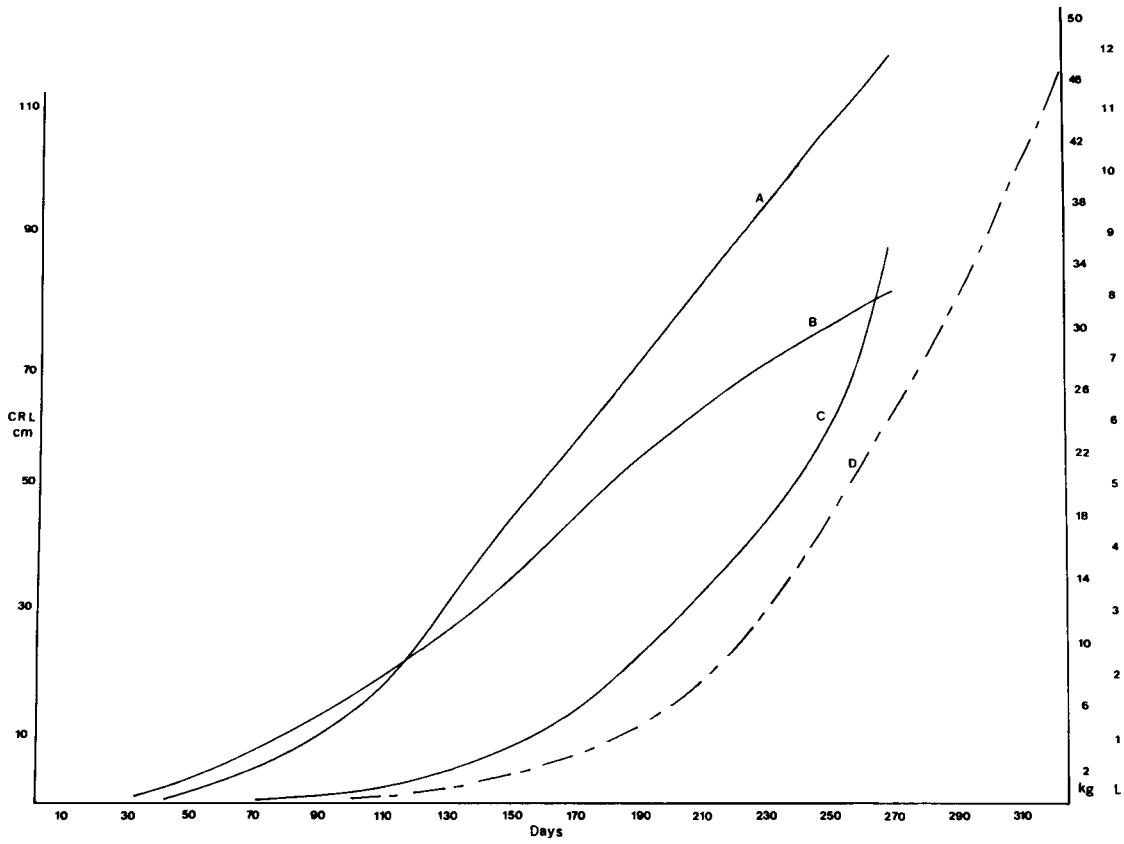

Figure 17.38. Fetal growth curves. (A) Bovine crown-rump length (CRL), (B) bovine amniotic fluid volume, (C) bovine fetal weight, (D) equine fetal weight.

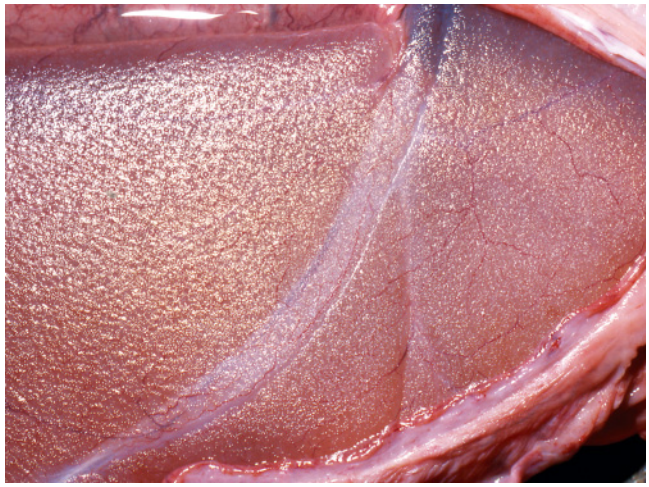

Figure 17.40. Surface of a llama chorion at four months gestation.

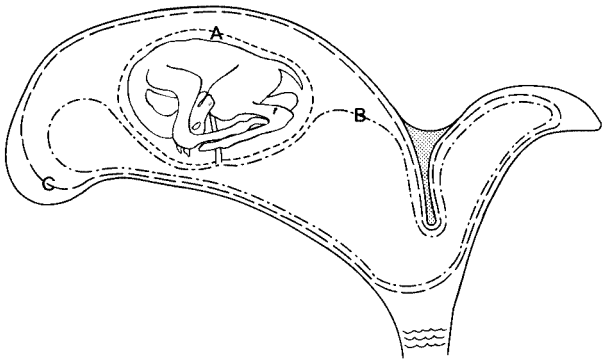

Figure 17.41. Diagram of early camelid placenta. (A) Amnion, (B) allantois, (C) chorion.

At this stage, the embryo is encased within an amniotic vesicle that is free-floating within the allantochorion, attached to the allantois by the umbilical cord and vessels (Figure 17.41).[96] This relationship is maintained until the last two months of gestation.

The allantochorion contains the fetal vascular supply. The surface of the chorion gradually changes from the domed placentoma to a multifolded papilla. It appears that each dome elongates, producing folds that expand to provide a greater surface area. The tip of each tuft is larger than the neck. If the placentoma becomes edematous as a result of disease, it looks like a miniature mushroom. The papillae fit into corresponding crypts in the uterine mucosa.

The papillae vary in size and shape, but generally the base is smaller than the tip. They may be columnar, rectangular, or shaped like an inverted cone and are 0.25 to 1.5 mm long. The density of the papillae is greatest in the patches, but a few papillae are found in the interspaces.

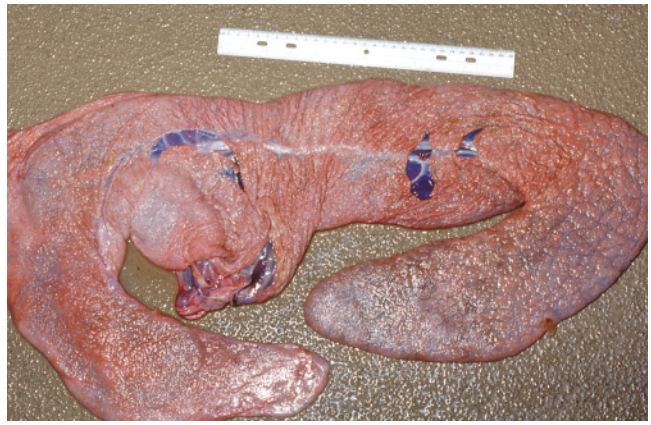

Figure 17.42. Full-term llama placenta, chorion outside.

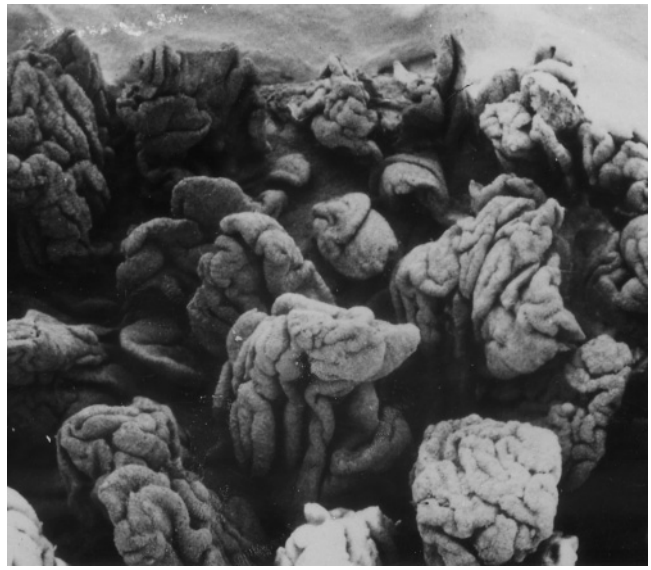

Figure 17.43. Scanning electron microscope picture of the endometrial face of the chorion in a full-term llama placenta (Magnification 0.025 KX).

On visual inspection, reddish, irregularly shaped patches occupy most of the surface of the chorion, with lighter-colored interspaces (Figure 17.42). The surface appears to be a roughened, undulating membrane grossly, but when viewed through a dissecting microscope at 20×, hundreds of multifolded papillae per square centimeter may be seen. An electron microscopic picture emphasizes the multiple folds (hence more surface area) (Figure 17.43). Patches of papillae are most dense on the greater curvature and toward the center of the crescent-shaped placenta. The tips of the placenta have lighter patchiness, and a strip 2 to 3 cm wide, extending the length of the lesser curvature in juxtaposition to the large vessels of the allantochorion, is nearly devoid of papillae. A branching pattern of vessels may be traced from the large vessels arising

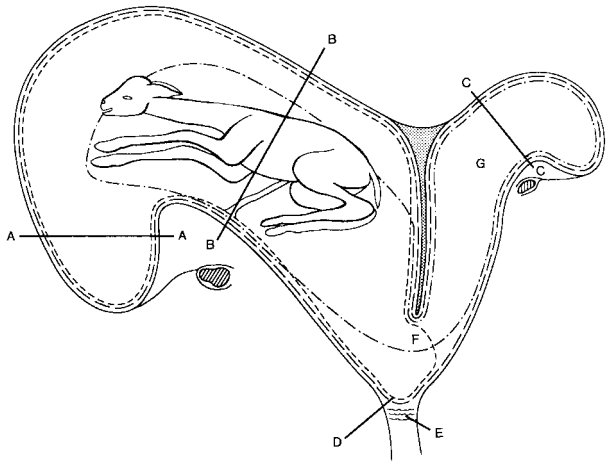

Figure 17.44. Diagram of full-term llama placenta: (AA, BB, CC) cross section (see Fig. 17.40), (D) site of amniochorion dehiscence, (E) cervix, (F) body of uterus, (G) nonpregnant horn.

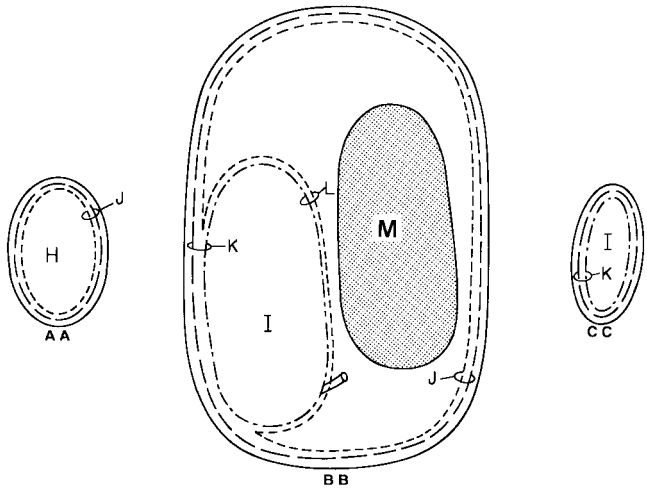

Figure 17.45. Diagram of cross section of full-term fetal membranes: AA, BB, and CC correspond to locations from Figure 17.39, (H) amniotic cavity, (I) allantoic cavity, (J) amniochorion, (K) allantochorion, (L) amnioallantois, (M) fetus covered with epidermal membrane.

Figure 17.46. Epidermal membrane attached to the nostrils and lips.

Figure 17.47. Epidermal membrane attached to pads and nails.

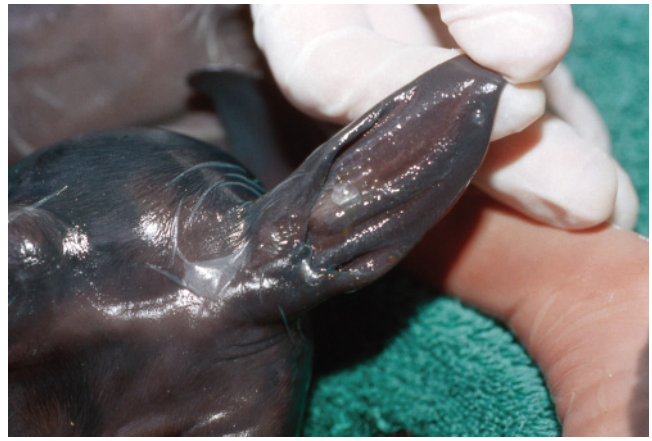

Figure 17.48. Epidermal membrane over the pinna.

from the lesser curvature of the placenta to each patch of papillae. The relationship of the membranes to the fetus is illustrated in Figures 17.44 and 17.45.

The most unusual characteristic of the camelid fetal membranes is the development of an extra membrane of fetal epidermal origin. This membrane was described in the dromedary camel thirty years ago[95] but was not described in SACs until 1988.[68,93,108]

The epidermal membrane (EM) is an opaque whitish membrane, approximately 1 to 2 mm thick, covering the surface of the fetal body, head, neck, and limbs at near full term (Figures 17.46 to 17.49). The EM is attached to the neonate at the mucocutaneous junctions such as at the lips, anus, vulva, and prepuce. The EM is also attached at the junction of the skin and footpad, coronet of the nail, and umbilicus.

Figure 17.49. Epidermal membrane over the eye and eyelids.

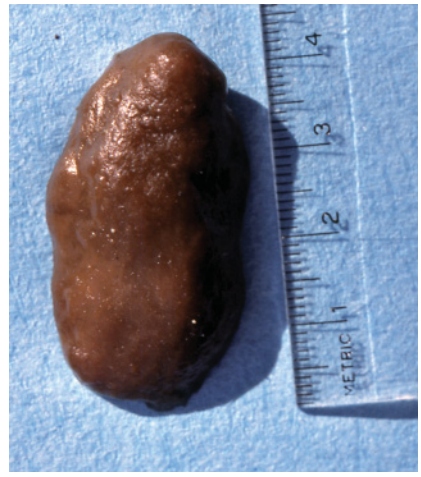

Figure 17.50A. Hippomanes from llama allantoic cavity.

Figure 17.50B. Hippomanes from llama allantoic cavity.

The EM is friable and easily torn or brushed from the surface of the neonate with only slight friction. Even with little movement or abrasion, it dries out and withers away soon after parturition. This is in contrast to the amnion of most mammals, which may completely envelope the nose and mouth, causing suffocation of a weak, nonstruggling neonate. Histologically, the EM consists of a layer of stratified squamous epithelial cells lying next to the fetus and an outer keratinized layer with indistinct cell outlines and no nuclei.[130] The fetus is usually delivered with the EM intact. However, if parturition is prolonged, with erratic or intense labor, or if manual assistance is necessary, the EM will split and disintegrate.

The precise function of the EM is unknown. The fetuses of noncamelid placental mammals float freely in the amniotic fluid. In the later stages of gestation in camelids, the EM separates the skin from the amniotic fluid. All of the SAC body orifices are unaffected by the EM, so excretory products from the digestive tract of the fetus are discharged into the amniotic sac, as in other mammals.

The amniotic fluid of the llama remains watery throughout gestation, as does that of the camel, producing a slippery surface on the EM that facilitates delivery.[105] In contrast, the amniotic fluid of the mare and cow becomes mucoid toward the end of gestation; this also lubricates the fetus and facilitates delivery.

The EM originates from the epidermis of the fetus. The precise embryology of the membrane is not known in SACs, but the time of its development has been studied in the camel.[104] Microscopic changes could be identified with a fetal crown-rump length (CRL) of 17 cm, or approximately midgestation. Gross appearance of the membrane did not occur until just before the development of the hair, which seemed to elevate the membrane away from the skin surface. In llamas, the EM has not been observed grossly until the last two months of gestation. Prior to this time, aborted fetuses have been surrounded by the amnion, which had not become adherent to the chorion.

At full term, the SAC fetus is situated within the pregnant horn and the body of the uterus. The amniotic sac extends to the tip of both the pregnant and nonpregnant horns and occupies all the space not occupied by the allantoic sac. The amnion may be easily peeled from the fetal side of the chorion. The chorionic attachment to the allantois is more secure. There is a minimal amount of fluid in the amniotic sac.

The allantoic sac extends into the nonpregnant horn, though not to its tip, and occupies all of the space in the pregnant horn. One or more hippomanes (allantoic calculi) are present in the allantoic sac, varying in size from $1 \times 1 \times 1.5$ cm to $1 \times 2 \times 5$ cm and weighing 2 to 10 g (Figures 17.50A, 17.50B).

PREGNANCY DETERMINATION. Methods used to determine pregnancy in SACs include behavior, progesterone analysis, rectal palpation, ballottement, and ultrasonography.[9]

Behavior: Usually, a mature, nonpregnant female will accept mating by a breeding male. In South America, refusal of sexual advances of the male by a female is considered to be a positive diagnosis of pregnancy. Refusal may be a good preliminary indication of pregnancy, with an 84% accuracy rate, but many factors may modify this behavior, and for the SAC owner or manager in North America, additional information must be obtained to verify pregnancy. Certain pathologic conditions simulate pregnancy (pyometra, retained CL, mummified fetus).

Breeding strategies that continually leave a male with females may foster development of a male that ignores even receptive females. Also, the male and one or more females may simply be incompatible.

An extremely aggressive male may overpower a timid female and force copulation, even though the female is pregnant. There is some danger if this should happen, since the male inserts the penis through the cervix and even into the horns of the uterus during copulation. Physical entry into the uterus after ninety days may traumatize the developing fetal membranes or introduce infection into the uterus. In either case, abortion may ensue.

Hormone Analysis[1,4,64]: Serum progesterone analysis has been promoted as the definitive method for pregnancy determination in SACs. While it is true that elevated levels of progesterone indicate the presence of a CL, this is not always confirmation of pregnancy. If this fact is kept in mind, progesterone levels may be useful as a management tool not only for determining pregnancy but also for evaluating various types of infertility problems. Elevated progesterone (>2 ng/ml) at day 7 after copulation is an indication of ovulation and presence of an active CL. A concentration level of progesterone >1 ng/ml (depending on the laboratory) twenty-one days post-copulation is an indirect indication of pregnancy.

Two new reproductive hormones have been studied: estrone sulfate and relaxin.[44] Peaks in levels of estrone sulfate in plasma and/or urine are observed twice during pregnancy (Figure 17.51), the first at twenty-one to twenty-seven days after copulation (45 ng/ml in serum and 75 ng/mg of creatinine in urine) and the second at the end of pregnancy. At this time, concentration levels of 40 ng/ml in serum and 900 ng/mg of creatinine in urine have been reported. The first peak probably indicates the beginning of implantation, and the second is an indication of fetal viability. The second measurement is useful in cases of prolonged pregnancy, as the amount decreases precipitously at the time of parturition.[41]

Elevated levels of relaxin concentrations in the pregnant llama or alpaca may also indicate pregnancy. At two months of pregnancy the values are 26 ng/ml (basal, 2.4 ng/ml). The maintenance level is 5 ng/ml, increasing to >25 ng/ml by the end of pregnancy, making relaxin an accurate indicator of pregnancy at specific times.[46] Analysis of relaxin is currently experimental.

Radioimmunoassay (RIA) and enzyme immunoassay (EIA) are standard analytic techniques for determination of progesterone levels. There is some variation between laboratories, but as long as results are consistent and the same laboratory is used for all determinations, this should not cause great concern.

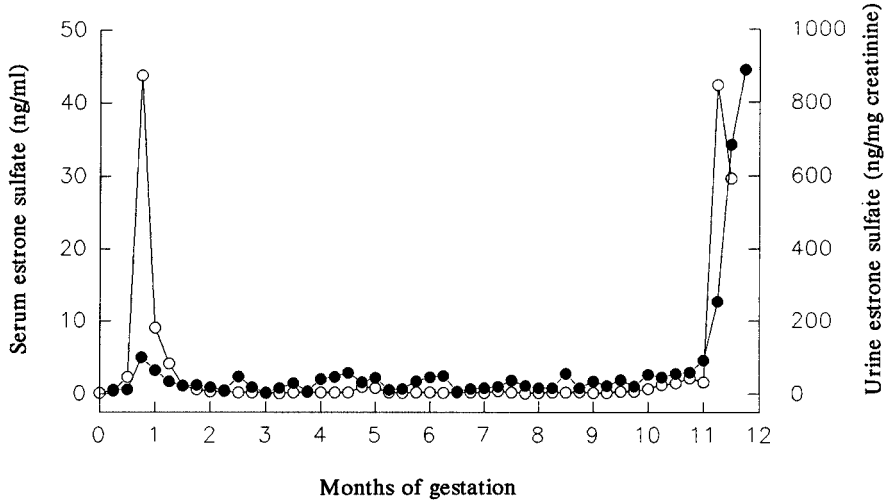

Figure 17.51. Estrone sulfate concentration in serum (open circle) and urine (closed circle) of the pregnant llama/alpaca. (Bravo et al. 1996, J. Am. Vet. Med. Assoc. 208:2027–30)

Some laboratories using RIA report that levels of 2 ng/ml are indicative of the presence of the CL. Higher levels, 5 to 8 ng/ml, have been reported but do not correlate with better maintenance of the CL. A different laboratory may use 1 ng/ml as the cutoff point. It should be noted that cases of successful pregnancies have been reported in which progesterone levels were less than 0.5 ng/ml. At the other extreme, progesterone levels of over 2 ng/ml have resulted from a retained CL, with no fetus present.

Rectal Palpation: Rectal palpation has been the method of pregnancy determination in horses and cattle for decades. The smaller size of llamas and alpacas makes rectal palpation more difficult, but it is a valid technique and may be done safely if certain precautions are observed.

Adequate restraint is a prerequisite for rectal palpation and ultrasound examination. Several different types of chutes prevent side-to-side motion of the animal (Chapter 3). Some SACs lie down when subjected to an unpleasant situation; others constantly alternate between lying down and jumping up. The latter behavior makes examination difficult and dangerous. Some clinicians prefer to perform a llama rectal examination with the animal in sternal recumbency, because abdominal pressure pushes the pelvic viscera into a more accessible location. To ensure continued recumbency, it may be necessary to sedate the SAC with 0.15 mg/kg xylazine intravenously.

People with large hands (glove size larger than 7) or large forearms will find it difficult to examine any except the largest animals. Adequate lubrication is the key to success. Fecal pellets should be gently removed from the rectum and methyl cellulose or other lubricants instilled into the rectum with an irrigation-tipped 60-ml syringe or dose syringe. The rectum should be slowly dilated, with the hand held in a cone position, palm facing dorsally. More lubricant should be added as needed.

Some examiners find it helpful to use a lubricant containing xylocaine to diminish mucosal irritation and reflex straining. Commercial xylocaine lubricants are available, but a less expensive mixture may be prepared by mixing 10 ml injectable 2% xylocaine with 100 ml methyl cellulose.

The anus may be the most restrictive site in the examination process. The anal epithelium may tear slightly during dilation. Excessive anal tone may be diminished by epidural anesthesia (Chapter 5). Xylocaine (1 to 2 ml, 2%) provides adequate relaxation of the sphincter but does not prevent peristaltic activity in the rectum. While a rectal examination using epidural anesthesia was being conducted, the female contracted the rectum around the author's wrist. The spasm did not subside for about ten minutes. Nothing could be done except wait because any vigorous attempt to withdraw the hand would have split the rectal wall.

SACs are not as susceptible to rectal trauma as mares but are more sensitive than cows. See Chapter 6 for a discussion of rectal laceration.

The cranial half of the uterine horns diverge at approximately 180 degrees from each other. Judicious palpation allows measurement and evaluation of the horns and the ovaries. It is important to remember that the ovaries are encased within an ovarian bursa, which may hinder evaluation. The relaxed ovary is ventral to the broad ligament. Retraction of the uterine horn aids in delivery of the ovary for palpation.

Examiners familiar with rectal palpation of the pregnant mare or cow may find slight differences in the uterine tone of an SAC. At sixty to ninety days, the left uterine horn is taut, resembling an inflated balloon, and is the key feature differentiating a nonpregnant from a pregnant female SAC.

At thirty days, pregnancy of a primiparous female may be determined through rectal palpation done by experienced SAC palpaters, who expect a unilateral increase in the diameter of the pregnant horn. In multiparous females, the left horn is usually larger than the right. If determination is to be limited to a single examination, it is preferable to examine at forty to forty-five days, at which time the pregnant horn should be 7 to 8 cm in diameter (palm width). Beyond ninety days, the uterus is positioned cranial and ventral to the brim of the pelvis, making it difficult to reach the uterus for evaluation. After eight months, the fetus may be accessible for palpation.

Signs such as the inability to palpate the uterus, a flattened, taut vagina, and a firm cervix may be suggestive of pregnancy but not conclusive evidence. There are too many congenital anomalies that produce mucometria, especially in a nulliparous female. The middle uterine artery develops fremitus during the last trimester of pregnancy, but this is not the basis for determining pregnancy or differentiating between pregnancy and pyometra, as it is for the bovine. It is not possible to slip the fetal membranes in llamas. The relative quantity of placental fluids is much less in SACs than in other livestock species. Some examiners have even made the mistake of concluding the presence of a mummified fetus because of the lack of fluids.

Determining the age of the fetus via rectal palpation is difficult after ninety days, even late in pregnancy, when various parts of the body and limbs may be palpable. Increases in the CRL are gradual, even during the last trimester, while the fetal body weight increases exponentially during the last three and a half months.

Ballottement: External signs of pregnancy may be lacking in female llamas. Udder development may be minimal, especially in a nulliparous female. The body contour changes in most females, but the fiber coat

tends to mask changes. As late as eight months, the fetus weighs only 3.5 kg. By ten to eleven months, the fetus may be balloted from the right side. Compartment 1 of the stomach occupies the entire left side of the abdomen, so the pregnant uterus is diverted to the right side. Ballottement is carried out as in cattle and may be done in two positions. In a standing animal, a clenched fist is pressed up against the abdominal wall, and a short vigorous push is exerted while the fist is held in place. If the female is pregnant, the push will cause the fetus to bounce away from the push, being cushioned in the fetal fluids, but it then drifts back and bumps the fist. In the lateral position, as practiced in South America immediately after shearing, pressure of both hands is applied on the abdominal wall, and the fetus bounces, indicating pregnancy and that the animal must be immediately separated from open females. The procedure should be repeated at a number of locations on the right side before the conclusion is reached that the female is not pregnant. At time of ballottement, the development of the mammary glands should also be inspected. However, mammary glands do not become enlarged with colostrum until twenty to thirty days before parturition, and some may not be enlarged then.

Ultrasonography: The use of ultrasound techniques for evaluation of the tubular reproductive tract and the ovaries has added a new dimension to veterinary medicine. The rapid improvements in technology in this field parallel those of the computer industry. Units are currently available that allow the transducer to be carried into the rectum and manipulated by the hand of the palpator. This is the most desirable method to use on pregnancies of less than 120 days.[5,8,16,76]

More sophisticated units allow transabdominal evaluations, which are more applicable for pregnancy determination later in the gestation period. In one instance, repeated rectal palpation and rectal ultrasound evaluation led a clinician to conclude that a female was not pregnant, and he was ready to institute hormonal therapy. A last examination using the transabdominal approach detected a fetal heartbeat in the abdomen, ventral to the stomach.

Earlier, Doppler ultrasound, which detects the heartbeat of the fetus, was used to attempt pregnancy diagnosis in llamas and alpacas. The accuracy of the method varied between 80% and 90% at day 70 of pregnancy but decreased to 60% to 70% by day 150 of pregnancy.

The application of real time rectal or transabdominal ultrasound has a great advantage, especially for early detection of pregnancy. The presence of the embryonic vesicle, as a nonechogenic circular structure within the uterus as early as twelve to fourteen days after copulation, is a positive indication of pregnancy. Selected ultrasonograms of early pregnancy in llamas appear in Figure 17.52. Figure 17.37 provides information on the growth rate of an alpaca embryonic vesicle from its detection until day 45 of pregnancy.[b]

Adequate restraint is a prerequisite for ultrasonography (see earlier rectal palpation discussion). When manual palpation is not possible, the rectal probe should be rigid. The attachment of a polyvinyl chloride (PVC) pipe eases the process. An alternative is the use of a prostatic probe, as in humans and mares. The resolution of the scanner is also greatly enhanced when a probe of 7.5 MHz is used. Details on formation of membranes, heartbeat, and placental development are evident using the latter probe. To determine fetal age and possible due date, the biparietal distance may be measured transabdominally. There is a strong positive correlation between gestational age and the biparietal distance. The equation is formulated as follows: GA = 18.8 + 3.79 BPD, where GA is the gestational age and BPD is the biparietal distance.[72]

Conclusions: All of the techniques described in this section have application for pregnancy determination in SACs. None of the procedures are 100% accurate, and some may be limited to certain stages of pregnancy and to individual examiners of limited size. It is likely that employment of more than one system will be necessary to provide optimum management of a breeding herd of SACs. A combination of rectal palpation or ultrasonography with progesterone determination is desirable.

Parturition

The endocrinology of parturition in llamas and alpacas has been partially defined. It has been reported that progesterone levels decrease rapidly two days before parturition. The same profile was observed when urine samples were assayed for pregnanediol glucuronide, a metabolite of progesterone. A different profile is seen when estrone sulfate is evaluated. Estrone sulfate in urine decreased precipitously only at time of parturition, i.e., when the fetal-placental unit was disrupted.[39]

Trying to predict the imminence of parturition is frustrating. Though the abdomen enlarges, growth may be hidden by the heavy fiber coat of many llamas and alpacas. Field biologists are able to assess pregnancy in guanacos and vicuñas by body contour, but these wild camelids lack the thick fiber coat of the alpaca and llama.[c]

Pregnant females within a month of parturition change behavior and appear restless and uncomfortable, with more frequent sniffing at the dung pile and possibly more voiding. Some females develop ventral edema, but this is not the problem that it is in mares and cows. Udder development is not necessarily correlated with nearness of parturition. The udder of a primiparous female may show little or no develop-

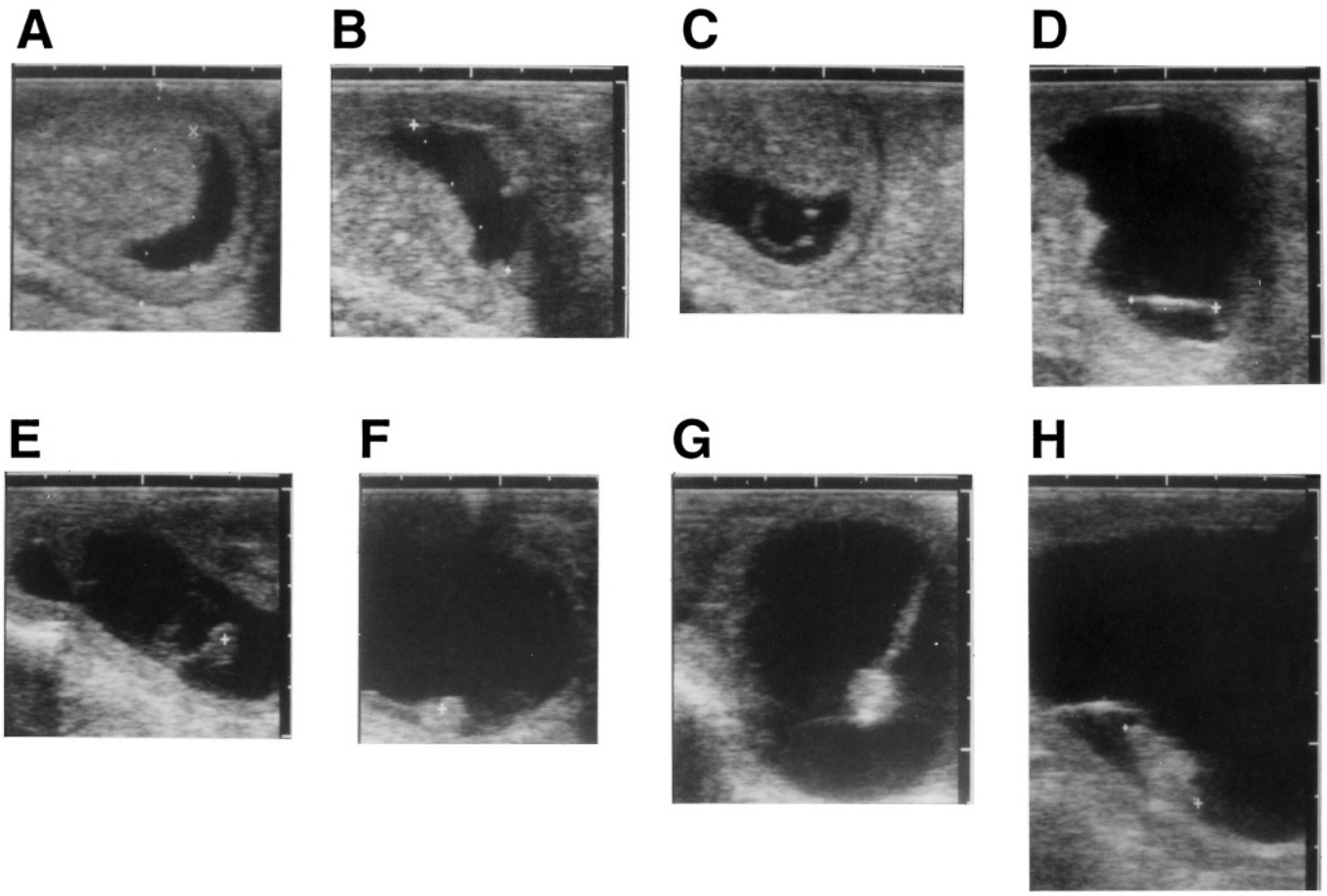

Figure 17.52. Ultrasound images of pregnant llamas. (A) seventeen days, (B) twenty days, (C) twenty-four days, (D) thirty days, (E) thirty-five days, (F) forty days, (G) fifty days, (H) sixty days.

ment until after parturition. The udder of a multiparous female usually enlarges two to three weeks before delivery, and the nipples may swell three to four days before delivery. Wax may form on the end of the teat, but its absence is not evidence that parturition is not imminent.[33]

Normal parturition is initiated by hormonal changes and is divided into three stages. Stage one begins with cervical relaxation and uterine contractions that propel the fetus toward the pelvis, dilating the cervix. This stage may last from two to six hours and tends to be longer in primiparous females. The signs of stage one include slight discomfort, excessive vocalization (humming), and repeated defecation and urination.

Stage two is the expulsion of the fetus. The amniochorion may dehisce within the birth canal, or the chorion only may dehisce and the amnion protrude through the vulva as a fluid-filled sac. In either case, a little fluid is expelled, usually dripping from the vulva. It is possible that the fluid-filled sac may be a pouch of the epidermal membrane, pushed ahead of the fetus. At this stage the perineum will begin to protrude (Figures 17.53, 17.54).

Figure 17.53. Parturition, protrusion of the amniotic sac.

The most common normal presentation is cranial longitudinal, with a dorsosacral position and the head lying dorsal to the extended limbs (Figure 17.55). A slight variant of this may occur, with the limbs positioned over the top of the head (Figure 17.56). The posterior longitudinal presentation, dorsosacral position with the hind limbs extended, is also normal but

Figure 17.54. Delivery of the fetus.

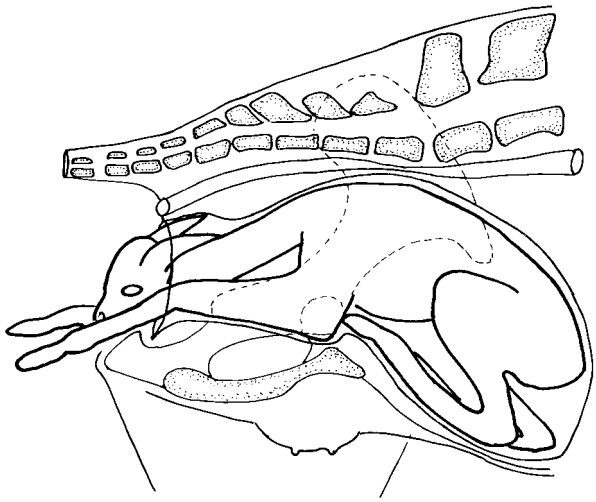

Figure 17.55. Normal llama delivery, cranial presentation, dorsosacral position.

much less common (Figure 17.57). The risk to the life of the fetus is greater in a caudal presentation dystocia because of the danger of occlusion of the umbilical vessels as a result of stretching or pressure.

Uterine contractions initially occur every ten minutes, increasing in frequency and intensity as expulsion progresses. At this stage, the female is definitely uncomfortable. She may lie down and rise

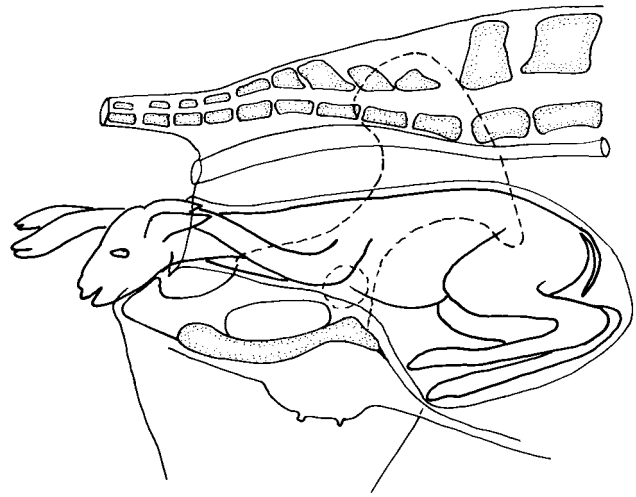

Figure 17.56. Normal llama delivery, with legs above the head.

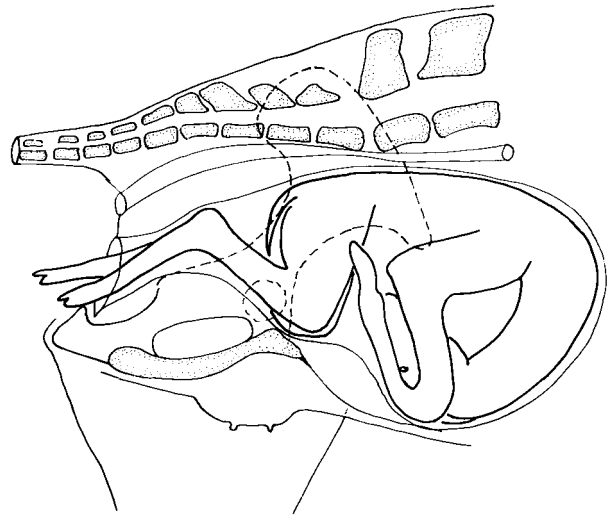

Figure 17.57. Normal but uncommon llama delivery, caudal presentation, dorsosacral position.

repeatedly, lie on her side, or even roll. If the fetus has been partially delivered, it may be traumatized by such actions. The female may look back at her side, vocalize, and exhibit other signs of colic. In an uncomplicated parturition, the unassisted female should deliver the fetus within eight to twenty-five minutes (24 minutes in llamas in the Altiplano). The majority of females deliver the fetus while in the standing position. In observations made on alpaca parturition in Peru, 65% to 73% of the fetuses were delivered while the female was standing. An additional 20$ to 24% were delivered from sternal recumbency and 7% to 12% while the female was lying on her side.

The umbilical cord usually ruptures 15 to 20 cm from the abdomen. Maternal care is minimal. The

mother does not attempt to remove the epidermal membrane or lick the cria to dry it. There is usually nose-to-nose touching, with humming that apparently functions to establish the mother-cria bond.

The majority of births take place during daylight hours, with the preponderance occurring in the morning.[69,83] However, llamas in North America have been known to give birth at night. SACs seem able to regulate parturition as a voluntary activity. Too much attention and excitement may actually delay parturition.

Stage three of parturition is expulsion of the placenta. The epitheliochorial placentation releases readily, and delivery of the placenta should occur within two hours. Retention of the placenta is rare in SACs. Dystocia or uterine inertia predisposes retention of the placenta. Failure to expel the placenta in six hours should be considered abnormal, but at this point the only recommended therapy is the administration of oxytocin (20 to 30 units intramuscularly). If a segment of the placenta is protruding from the vulva, it should be grasped gently by a gloved hand and slight tension applied. Frequently, the membranes are sitting loosely in the vagina and the body of the uterus, and a slight tug is all that is needed to effect delivery.

It has never been necessary to manually separate the placenta from the uterine mucosa. If the placenta has not delivered by twelve hours, the protocols used for dealing with the problem in a mare are recommended.

The SAC placenta is crescent shaped. In the majority of cases, the membranes are reddish in color, with the chorion on the outside. In a few instances, the placenta is turned inside out, with the amnion exposed, and have a whitish, glistening surface. Each placenta should be inspected to determine the presence of the two tips, a good indication of complete expulsion.

The average llama placenta, exclusive of fluids, weighs approximately 3 kg (range 0.74 to 1.44 kg). The greater curvature measures 196 to 280 cm (77 to 110 in.), and the concave curvature measures 96 to 210 cm (40 to 83 in.).

Female Camel Reproduction[2,124]

Female camel anatomy and physiology are similar to those of SACs with due consideration for a larger size. Puberty may occur at two to four years of age, but breeding is usually not recommended until four years of age. Camels are seasonally polyestrous. The breeding season usually coincides with the rut in males. After the breeding season the ovaries become inactive. Anestrous may extend up to six months.

The ovarian cycle is a follicular wave, similar to that of the SAC female. Camels are also induced ovulators. Contrary to SAC females, which are essentially receptive to the male or they are pregnant, the camel follicular cycle is spread out, which allows estrogen levels to become low enough to cause the female to become un-receptive and later to become receptive again. This type of cycling is frequently but mistakenly, called an estrous cycle. There is still only a follicular wave and no ovulation unless the female camel is bred at an appropriate time.

The induction of ovulation is a neurohormonal stimulation brought about by copulation. Non-penile mechanical stimulation is not effective in initiating ovulation in Bactrian camels. It is thought that an unknown chemical substance in camel semen is the stimulus for ovulation. In alpacas, it is thought that ovulation is initiated by a combination of penile intromission, vocalization (orgling), legs clasping the body, and deposition of the semen.

As in SACs, fertilization of the ovum occurs in the oviduct. The embryo enters the uterus at six and a half to seven days post breeding in camels. This is similar to the migration in llamas, but in alpacas the embryo may reach the uterus in three to four days. Organ differentiation occurs in forty-five to sixty days. A heart beat may be detected by twenty-two days. Gestation length varies from 355 to 419 days.

Camel Pregnancy

Camel caretakers claim the pregnancy is proven when the female extends her tail or holds it upward when teased by a male. Definitive diagnosis is established by rectal palpation or ultrasound examination as in SACs.

Signs of impending parturition include distention of the abdomen, mammary gland enlargement, relaxation of the pelvic ligaments, and edema of the vulva a few days prior to parturition. Parturition is similar to that of SACs. Uterine involution is complete in ± twenty days.

Postpartum Complications[87]

Failure to Deliver the Placenta. See discussion on parturition in previous section.

Prolapse of the Uterus and Vagina. See Chapter 6. Too vigorous pulling on the placenta immediately after delivery of the fetus may evert the uterus. Replacement of the uterus at this time is easily accomplished.

Uterine Infection. Metritis is rare except in cases of dystocia in which trauma has been inflicted by manipulations. A few cases have been associated with the obese female, postpartum.

Postpartum Hemorrhage. Hemorrhage can arise only from lacerations of the uterus or vagina. A persistent flow of blood should be investigated immediately (Chapter 6).

Uterine Tear. See Chapter 6.

Agalactia. Lactation failure following parturition is common in SACs (see chapters 10 and 21). The problem is seen more often in primiparous females, but it also occurs in multiparous females. Lactation in SAC is presumably controlled by the same systems as in other livestock species. If the fetus is delivered prematurely, hormonal preparation of the mammary gland to begin secretion may not have been completed. Inadequate milk let down may be a factor. Placement of the neonate in the nursing position may stimulate the dam to allow milk let down, or administration of intramuscular oxytocin in repeated doses of 20 units every two hours is appropriate therapy. Both actions should be accompanied by gentle massage of the udder with warm water and stripping milk from the teats.

A careful watch of the baby is necessary to ensure that sufficient milk is being consumed. Otherwise, the cria must be supplemented or orphaned until an adequate milk flow has been established.

Rejection of the Cria. Rejection of the cria is extremely rare in SACs; in fact, female alpacas and llamas often accept crias other than their own. In one zoo herd, a particular female would accept every cria in the herd long after its own mother had weaned it. Vicuñas and guanacos do not accept any cria other than their own.[d]

Involution of the Uterus.[29] Discharge from the vulva postpartum (lochia) is scanty and is usually absent after six to eight days. Postpartum involution of the uterus occurs rapidly in SACs. In a study conducted with alpacas, the average weight of the uterus was 883 g twenty-four hours after parturition, with the pregnant horn being approximately 15 cm in diameter. Ten days postpartum, the uterus weighed only 155 g, and the pregnant horn was 3.5 cm, and in twenty days, 83 g and 3 cm, respectively. Such a rapid involution is necessary to maintain a twelve-month birthing interval. Female llamas will accept copulation within two to three days postpartum, but the uterus is not involuted, and it is not desirable that breeding take place until twenty days following parturition.[43]

A male may attempt to copulate with a female when she is recumbent and parturition is imminent.

Obstetric Procedures[102]

Dystocia. Dystocia refers to any parturition that is more prolonged than expected. Breeders report a low prevalence of dystocia in SACs as contrasted with sheep and cattle.[79] In observations of large numbers of animals in Peru, it was determined that the prevalence of dystocia in alpacas was less than 2.4% and that the entire parturition process in the alpaca should be completed within three and a half hours.[24] Causes of dystocia may be related to fetal or maternal factors (Table 17.4).

Malposition. The factors that orient the fetus to the normal parturient presentation and position are unknown, as are those factors that cause malpositioning. Several malpositions are described and illustrated.

Table 17.4. Causes of fetal and maternal dystocia.

Fetal	*Maternal*
Malposition	Uterine torsion
Congenital defects	Uterine inertia
Schistosomus reflexus	Failure of cervix to dilate
Ankylosis of major joints	Malformed pelvis
Hydrocephalus	Stenosis of the vagina
Edema and maceration of fetus	Stenosis of the vagina

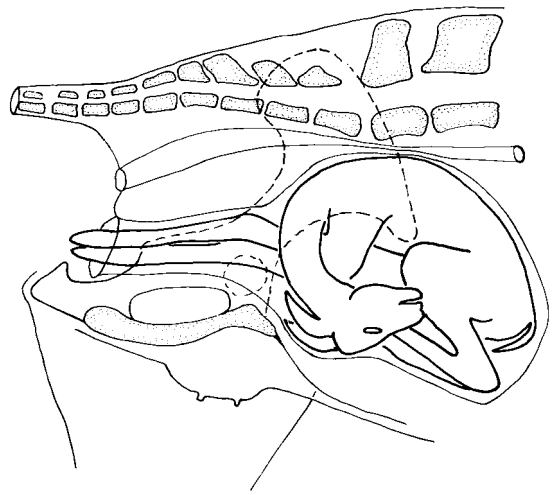

Figure 17.58. Camelid dystocia, cranial presentation, dorsosacral position, with head retained to one side.

The most common malposition is cranial longitudinal presentation in the dorsosacral position, with the limbs extended but the head and neck flexed backward to the side (Figure 17.58). Others include (1) cranial longitudinal presentation, dorsosacral position, with the head and one limb extended but the other front limb retained (Figure 17.59); (2) cranial longitudinal presentation, dorsosacral position, with one limb extended but the head and the other limb retained (Figure 17.60); (3) cranial longitudinal presentation, dorsosacral position, with the limbs extended but the head retained between the limbs (Figure 17.61); (4) cranial longitudinal presentation, dorsosacral position, with the head extended but both limbs retained (Figure 17.62); (5) cranial longitudinal presentation, dorsosacral position, with both limbs and head retained (Figure 17.63); (6) caudal longitudinal presentation, dorsosacral position, with the limbs retained (breech presentation) (Figure 17.64).

Other, less common dystocias are cranial longitudinal presentation, sacropubic position, with head and

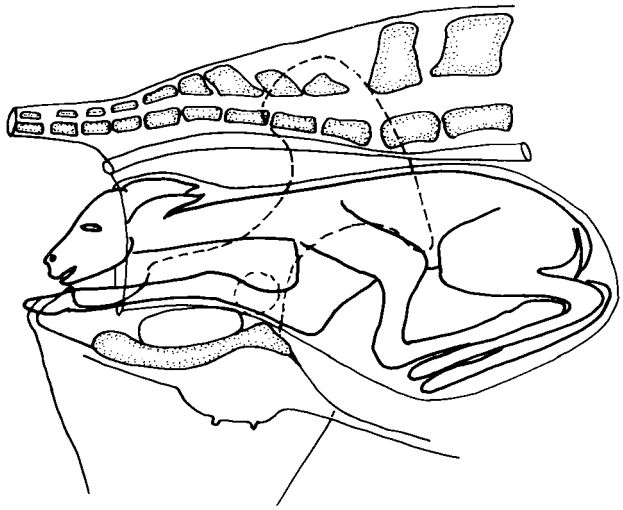

Figure 17.59. Camelid dystocia, cranial presentation, dorsosacral position, one limb retained.

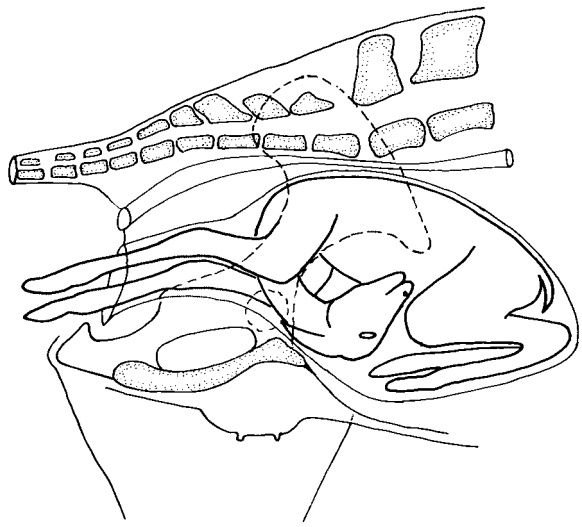

Figure 17.61. Camelid dystocia, cranial presentation, dorsosacral position, head retained between legs.

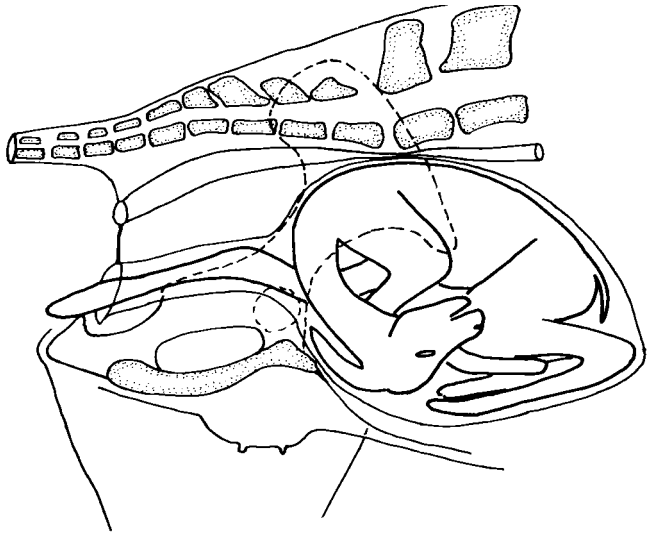

Figure 17.60. Camelid dystocia, cranial presentation, dorsosacral position, head and forelimb retained.

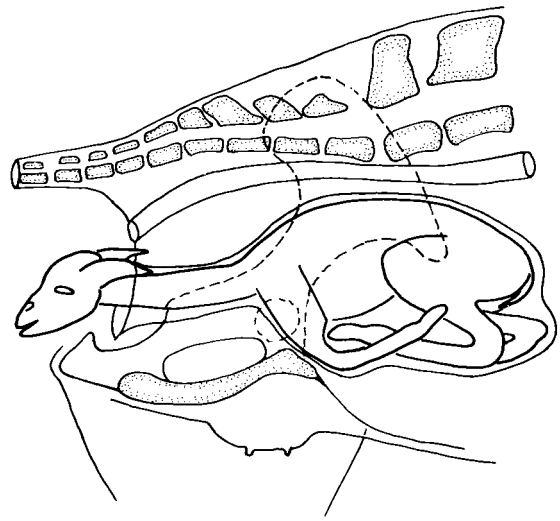

Figure 17.62. Camelid dystocia, cranial presentation, dorsosacral position, both forelimbs retained.

forelimbs extended (Figure 17.65), and transverse ventral presentation, dorsoilial position, with all four limbs and the head extended (Figure 17.66).

Manipulation of Dystocias. Space does not permit detailed discussions of obstetric procedures. The reader is referred to a standard veterinary obstetrics textbook.[89] A few unique facts should be kept in mind. SACs are small and there is little room for manipulation, even by a person with small arms and hands. It may be necessary to draft the smallest person available to perform any necessary internal manipulation, under appropriate direction. If manipulation by a large person is essential, epidural anesthesia should be administered to provide analgesia; additional relaxation may be produced with butorphanol (Chapter 5).

The limiting dimensions of the birth canal are not at the pelvic inlet but rather at the level of the caudal end of the sacrum and the ischiatic spines of the ilia (Figures 17.67, 17.68). The measurements are of one adult female weighing approximately 140 kg. On the cranial view, the vertical distance is 11 cm; the width, 10.5 cm; and the diagonal, 12 cm. The caudal view shows the relationship of the ischia ilia and sacrum. The vertical distance is 12.5 cm, the narrowest width, 8.5 cm; and the diagonal, 14 cm. For comparison, Figures 17.69 and 17.70 illustrate cranial and caudal views of the pelvic inlet of a male.

Some ligamentous relaxation occurs at the time of parturition to allow the sacrum to tilt up slightly, but

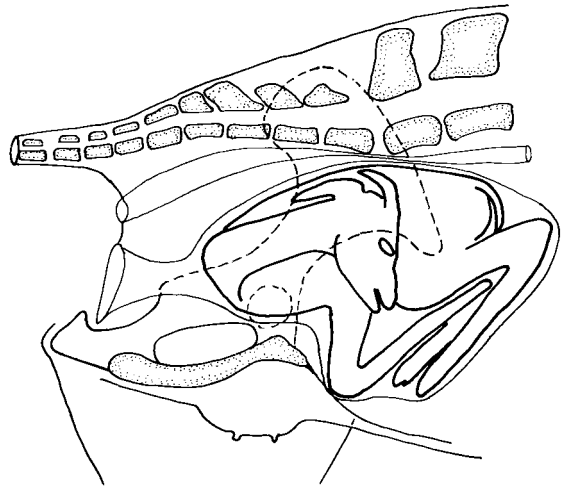

Figure 17.63. Camelid dystocia, cranial presentation, dorsosacral position, head and both forelimbs retained.

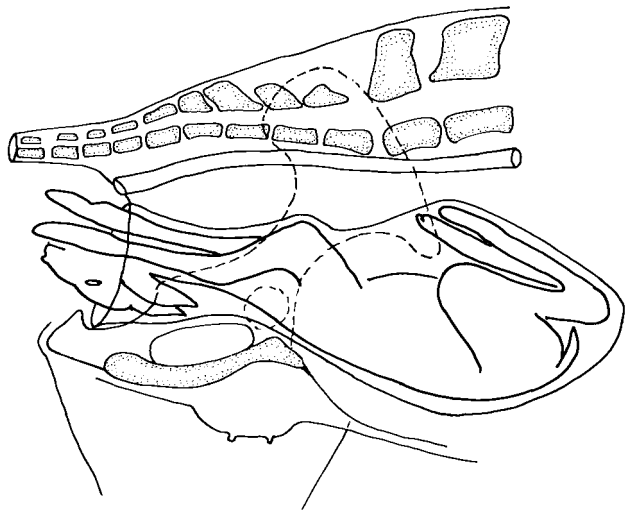

Figure 17.65. Camelid dystocia, cranial presentation, sacropubic position, head and forelimbs extended.

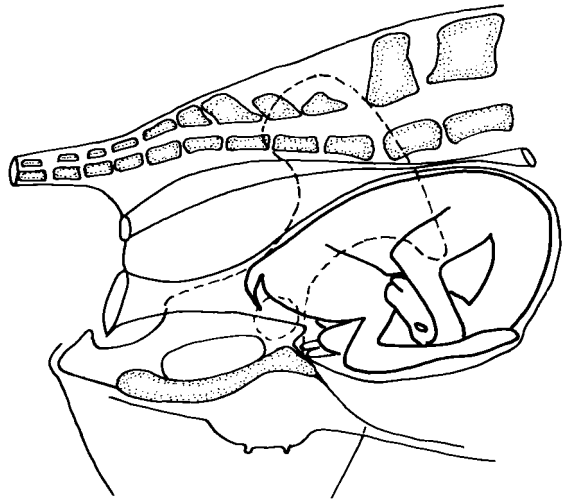

Figure 17.64. Camelid dystocia, caudal presentation, dorsosacral position, hind limbs retained (breech).

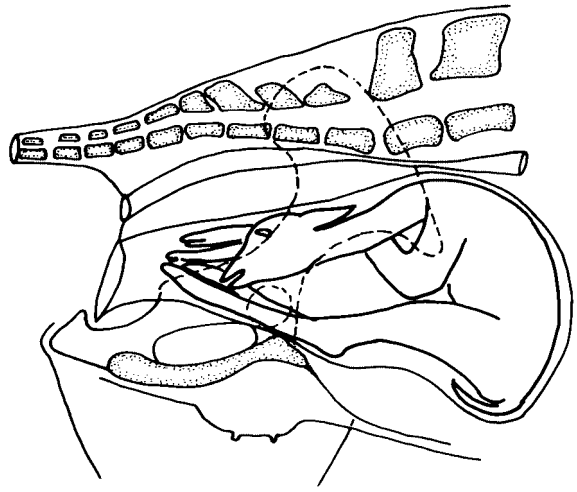

Figure 17.66. Camelid dystocia, transverse presentation, dorsoilial position, four limbs and head extended.

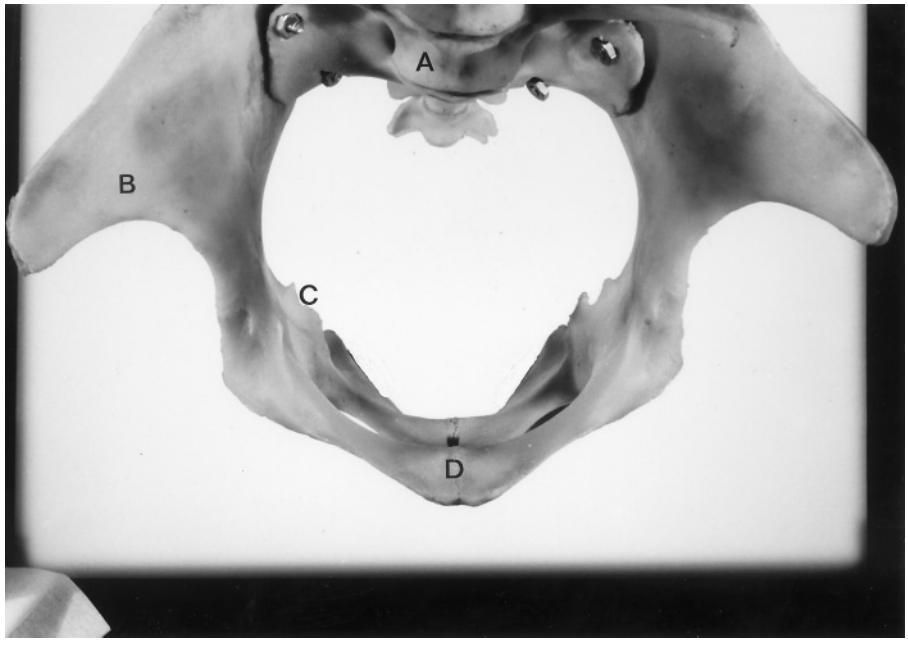

Figure 17.67. Female llama pelvis, cranial view. (A) Sacrum, (B) ileum, (C) ischiatic spine, (D) pubis.

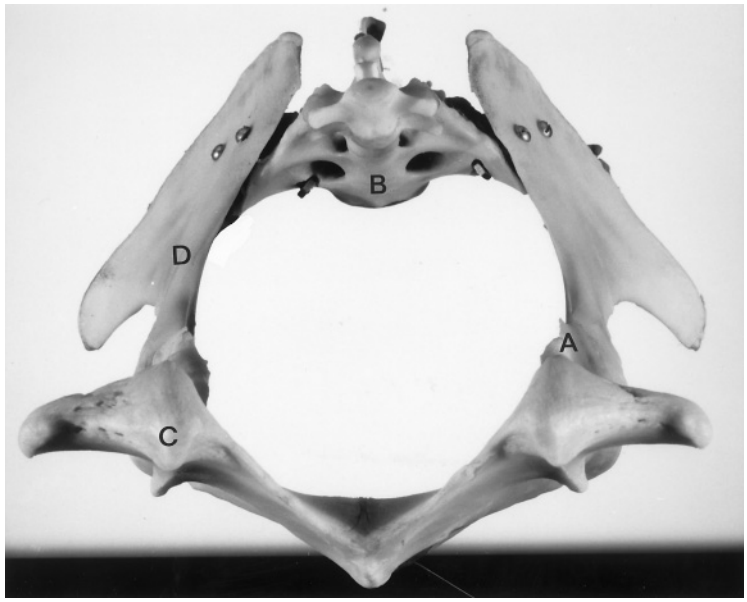

Figure 17.68. Female llama pelvis, caudal view. (A) Ischiatic spine, (B) sacrum, (C) ischium, (D) ileum.

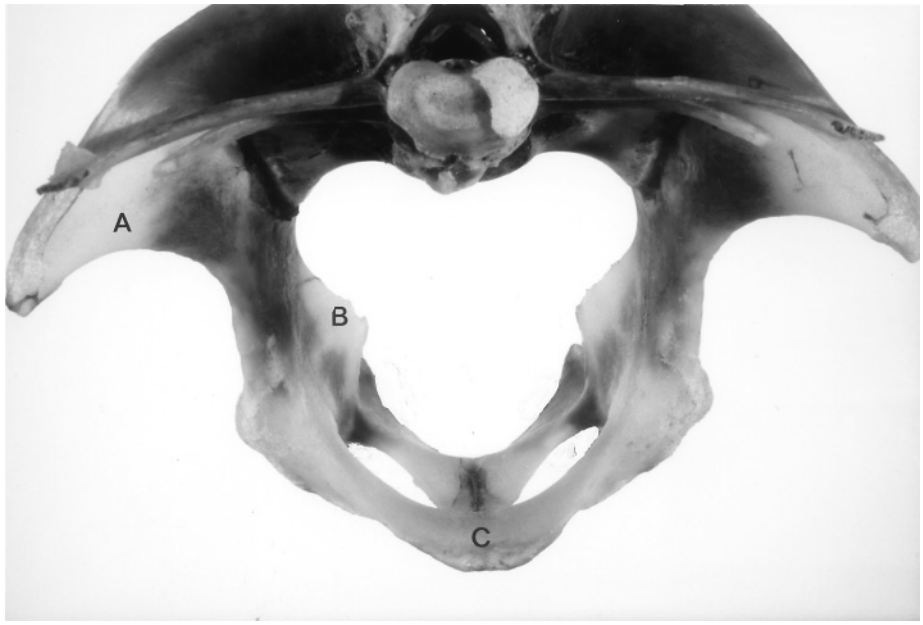

Figure 17.69. Male llama pelvis, cranial view. (A) Ilium, (B) ischiatic spine, (C) pubis.

it is not likely that there will be any give in the width. The authors have noted that the ischiatic spines may be so close that the hand must be inserted in a vertical plane to pass this anatomic obstruction.

Amniotic fluid is scanty, and if the epidermal membrane has been rubbed off by manipulation, lubrication will be needed to facilitate delivery. Soap is contraindicated, because it is irritating to the mucosa and dissolves surface-protecting oils. Methylcellulose is excellent.

In most livestock species, the fetus is likely to impact at the hips. In the llama, the shoulder girdle is more likely to impact. From a normal delivery presentation and position, before the shoulders reach the pelvic canal, it is helpful to twist the fetus 45 degrees and gently pull on one leg at a time. The adult pelvic girdle is basically a vertical rectangle, with the greatest cross-sectional dimension on the diagonal.

The use of obstetric chains is not warranted. Usually, only manual grasping is necessary. If repulsion is nec-

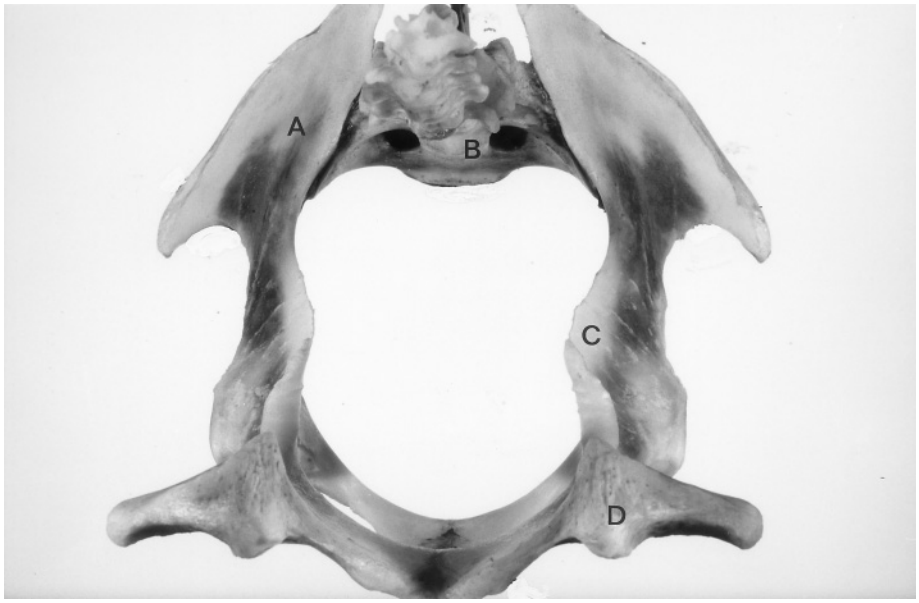

Figure 17.70. Male llama pelvis, caudal view. (A) Ilium, (B) sacrum, (C) ischiatic spine, (D) ischium.

essary, a clean, disinfected lightweight nylon cord may be secured to a limb to allow later access to the limb.

If the hips lock, the fetus should be repelled and twisted on its longitudinal axis 45 to 90 degrees before traction is applied dorsally. The fetal pelvis is wider than it is deep, and the twist serves to coincide the fetal pelvis with the greatest vertical dimension of the dam's pelvic girdle. Other manipulations are similar to those recommended in cattle and sheep dystocias.

Any of the caudal presentations carry more risk of fetal death with prolonged delivery. The umbilical circulation is quickly compromised because of the caudal position of the umbilicus on the abdomen. It is wise to advise any client who is conducting preliminary obstetric examinations that caudal presentations usually require professional assistance and should be considered emergencies.

Delivery from the caudal presentation must be quick and efficient. There must be adequate lubrication, and the cervix must be fully dilated before traction is applied. If the clinician feels that more than ten minutes will elapse before the fetus can be delivered, a cesarean section may be preferable.

Many dystocias caused by congenital anomalies require delivery by cesarean section, or in the hands of a skilled operator, fetotomy may be performed.

Maternal Dystocia. Uterine torsion occurs in llamas and alpacas. The causes are unknown. The torsion may be in either direction. Ranges of 90 to 360 degrees rotation in the vagina have been reported. Rectal examination, manual vaginal examination, or a vaginal speculum examination will identify the problem. The primary clinical sign of torsion is repeatedly assuming the sternal position and staying there for a little while, then standing and not eating. The female may look at her flanks and act colicky. The cervix may partially dilate, but relaxation cannot be completed because of mechanical inhibition.

The management of uterine torsion depends on the degree of rotation, the size of the female, and the distress experienced by the female. When rolling the female, she should be put in lateral recumbency on the side where the uterine torsion is directed, i.e., if the torsion is clockwise, the female should be in lateral recumbency on her right side. If the torsion is counterclockwise, the female should be put in lateral recumbency on her left side and correction of the uterine torsion begun (Figure 17.71).

A rectal and/or vaginal manual palpation is conclusive if the uterine torsion has been corrected, or there was an over correction. Rolling the female while pressing against the uterus through the wall of the abdomen with fists or a board may be of help in some cases (Figure 17.71). Some degree of sedation may be necessary. The torsion may be corrected by the female herself through such colicky maneuvers as getting up and down or rolling. One alpaca became upset while being restrained in a chute, reared up, and fell over backward. After she scrambled to her feet, reexamination showed that the torsion had been corrected.[e] Manual maneuvers should not be continued for more than ten to fifteen minutes. If the torsion persists longer than this, a cesarean section may be necessary to save the life of the fetus and the dam.[75] A practical observation is that after correcting the uterine torsion, the female will get up and go with other females and start eating. Uterine rupture is a possible sequel to torsion.

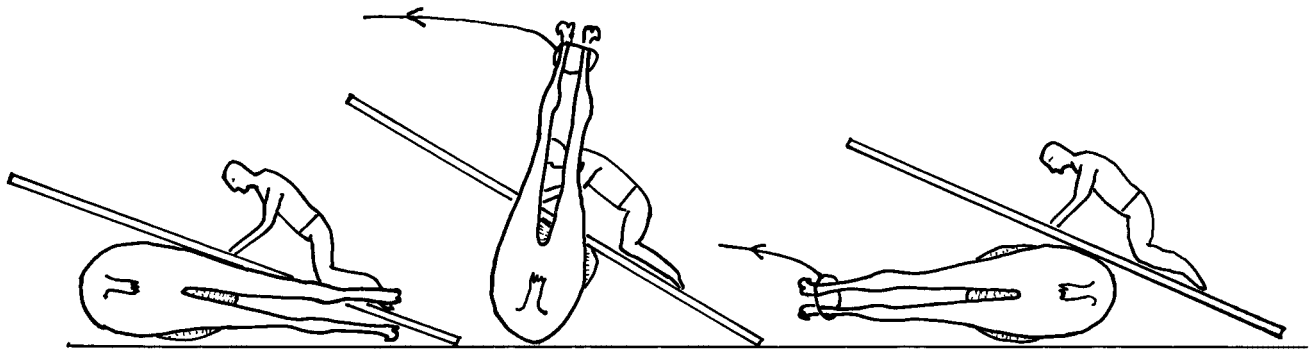

Figure 17.71. Diagram of the mechanical method of correcting a counterclockwise torsion of the uterus.

Uterine inertia is usually caused by prolonged, unproductive effort during stage one or two of parturition. Following correction of malpositioning, neither uterine nor abdominal contractions will continue if the female is exhausted. In such cases, though delivery will be more difficult without the assistance of contractions, it is unwise to administer oxytocin until after the fetus is delivered.

Failure of the cervix to dilate is difficult to differentiate from false labor. A small female carrying a large fetus may experience colicky signs during the last month of pregnancy; the colic is caused by partial obstruction of the digestive tract by the massive uterus. The relaxation of the cervix is under hormonal control, which may be abnormal. Good breeding records, careful observations, and a physical examination are necessary to precisely identify this problem.

Prostaglandins ($PGF_{2\alpha}$) are the trigger for cervical relaxation and may be administered subcutaneously at a 5- to 10-mg total dose. However, if a mistake has been made in the breeding date, this drug will cause abortion. Relaxation occurs within twenty-four hours following the administration of $PGF_{2\alpha}$. If the SAC is showing signs of stage one, it may be necessary to manually dilate the cervix.

Pelvic stenosis may be caused by abscesses, tumors, or healed but malaligned fractures. A cesarean section may be necessary to relieve the dystocia prior to correction of the basic disorder.

Cesarean Section. This surgical procedure is a valid alternative when manual manipulation to deliver the fetus is impossible, the fetus is too big, or the pelvic canal of the dam is too narrow. The general principles of cesarean section in other livestock species should be followed. Cesarean section is performed with the female in lateral recumbency or in sternal position; a C-section of a llama in standing position has even been reported. The uterus should be incised at the mesometrial angle, avoiding large blood vessels. If available, the use of a vacuum pump is helpful to avoid any spilling of fetal fluids within the abdominal cavity of the patient. The placenta may be left inside the uterus, which should be expelled later through the vagina within eight hours after performance of the C-section. In addition, the placenta may be to separated for a couple of centimeters along the incision side at the mucosal level of the placenta. This is helpful because the uterine wall is not caught when the double suture is placed. A person knowledgeable of neonatology should care for the neonate and assist it if it does not begin breathing and/or correct any other lack of vital signs.

Pregnancy Termination[27]

The termination of pregnancy in SACs may be indicated for the same reasons as in other livestock. Accidental mating is the most common indication, but suspected prolonged gestation may be considered sufficient reason for the induction of parturition.

Early Gestation. Two major methods have been recommended to induce abortion. After the impregnated ovum has reached the uterine horn, presumably after seven days, uterine irrigation with 25 ml 2% Lugol's iodine solution, 250 mg of tetracycline in 25 ml saline, or 25 ml 70% ethyl alcohol is effective in destroying the ovum and flushing it from the uterus.

Alternatively, $PGF_{2\alpha}$ (lutalyse), at a total dose of 10 mg, may be administered once intramuscularly from six to 100 days following copulation. The six-day lag allows time for the CL to begin formation. SACs that have been administered prostaglandins should be constantly observed for at least an hour to detect reaction. Should the llama become dyspneic or colicky, oxygen should be administered immediately.

Midgestation. In cattle, after 150 days gestation it is recommended to induce abortion with a combination of dexamethasone and prostaglandin. Dexamethasone, even in doses of 5 mg, caused fetal death with delivery of a stillborn within seven days of administration, and females had a retained placenta. Placenta retention was not observed in $PGF_{2\alpha}$ administration.[21]

Late Gestation. Observations indicate that dexamethasone, estradiol, and oxytocin did not induce parturition within ten days of the due date. By contrast, $PGF_{2\alpha}$ was the most efficient way to induce parturition. Delivery occurred within twenty-one hours after $PGF_{2\alpha}$ was administered. The primary indication for late gestation termination or induction of parturition is a female suspected of prolonged gestation. The female should always be examined for pregnancy before induction is begun. Clients have been embarrassed to find that the female they thought to be showing all the signs of impending parturition was just becoming fat.

A veterinarian should be cautious about acquiescing to a client's desire to induce parturition. Numerous instances of incorrect breeding dates have been reported. Furthermore, valid thirteen-month gestations are known. Prolonged gestation, as recognized in Holstein cows, is not known in SACs. The fetus does not overgrow the ability of a normal female to deliver it.

INFERTILITY[82]

Female Llamas and Alpacas

Early breeding management strategies for alpacas and llamas in Peru yielded approximately 70% live births. The strategy used then was similar to that recommended for the management of sheep. The reproductive physiology of SACs is unique, not at all like that of sheep, and sheep methods were not successful with SACs. With better management the fertility rates of llama and alpaca herds and the individual animal has markedly improved, but numerous problems remain to be solved. Data are still incomplete on the precise causes of infertility in both males and females.

North American experiences with therapy and management are equally limited. A detailed discourse on these matters would lack factual foundation and simply be an extrapolation from knowledge of cattle, horses, sheep, and goats. Therefore, this discussion only outlines what is known about infertility in SACs and suggests some approaches to the problems. Experimental data is introduced when appropriate.

Etiology

Congenital defects[12,13] account for much infertility in the female llama and alpaca, both in North America and in the Andes. In one study in Peru, one or more anomalies that could compromise fertility were seen[112] in more than 10% of the females examined at slaughter. Buyers should be encouraged to require pre-purchase examinations of breeder animals if they are old enough that such an examination may be conducted. Otherwise, a written agreement relative to congenital anomalies should be part of the buyer-seller contract.

Following is a list of the anomalies that have been seen or reported in the female llama and alpaca reproductive tract: ovarian aplasia, ovarian hypoplasia, tubular hypoplasia, segmental aplasia (oviduct, uterine horn, cervix, vagina), persistent hymen, uterus unicornis, double cervix,[17] and intersex. (See Chapter 22 for a full discussion.) In addition, the presence of numerous small follicles, hypoplastic ovaries, occurrence of cystic follicles, and failure to ovulate compromise fertility. Numerous small follicles resemble a bunch of grapes with no follicle reaching maturity. This anomaly may last for fifteen days and then resolve itself.

Hypoplastic Ovaries. Hypoplastic ovaries (10×13 mm) represent 17% of the total anatomical abnormalities. Follicles may grow sporadically, but follicular waves are irregular, and follicles do not mature beyond a size of 6 mm. Estrone sulfate levels are lower than in females with normal ovarian follicles.[40]

Cystic Follicles. The presence of cystic follicles, 13 to 25 mm in diameter, is common in llamas and alpacas.[40] Cysts may persist for an average of nine days (range four to fifteen days). Once a cystic follicle is present, the contralateral ovary is quiescent. Attempts to treat cystic follicles have had variable results. Some may be ruptured manually and others may or may not respond to hormonal treatment. Breeding females with cystic follicles has variable results. Some females respond to copulation, and LH is secreted; however, luteinization of the follicle ensues, resulting in a short luteal phase (described above). Other females do not respond to copulation, and cystic follicles may increase in size. Erratic sexual behavior of the female may be seen, with some females refusing to permit copulation.

Ovulation Failure. Failure to ovulate occurs in approximately 15% of females. Some develop mature follicles but do not ovulate after copulation or the administration of GnRH.[40]

Endometrial Cysts. Endometrial cysts are rare. Ultrasonography indicates they are irregularly shaped, nonechogenic structures, and randomly located in the endometrium. Early treatment with prostaglandins has eliminated cysts. Females with endometrial cysts do not become pregnant, even when ovulation occurs.

Failure to Maintain a CL. Failure to maintain a CL has been recognized as a clinical entity, and progesterone implants are being used to support pregnancy, with some reported successes. Synchromate B is a progesterone product used to synchronize estrus in dairy cattle, and it has been administered to llamas that continue to suffer early embryonic loss. To avoid perpetuation of poor reproductive performance in a possibly genetically inferior female, the product is recommended only for females that have successfully carried a fetus to term. An implant is inserted with a special needle gun at the base of an ear. Three weeks later another implant is inserted in the opposite ear, and

three weeks after that, the implant from the first ear is replaced. The procedure is repeated every three weeks until a fetus is delivered.

A second source of exogenous progesterone is Hydroxyprogesterone caproate (commonly known as Hypoval), which is administered intramuscularly. This is a good alternative to maintain pregnancy in a female that is diagnosed pregnant by ultrasonography and that has generally lost the pregnancy within the first month of gestation. The caveat of using this drug is that when used up to term it sometimes induces an extension of the pregnancy period and may result in a dead fetus that must be delivered by C-section. Records of a clinic (Walter Bravo) indicate that of twelve females given an exogenous progesterone supplementation to maintain pregnancy, five delivered a dead fetus after the pregnancy was prolonged beyond a year, four of which required a C-section to deliver a dead fetus. Exogenous progesterone should only be administered up to ten months of pregnancy, under strict veterinarian supervision. The role of a veterinarian is critical. Some owners who opted to administer exogenous progesterone to all females that failed to conceive induced a serious disturbance of the ovarian cycle of the female. The author (Bravo) observed that ten females did not resume ovarian activity for two to three months after an indiscriminate administration of exogenous progesterone. Most females do not need exogenous progesterone. Before a decision is made to administer exogenous progesterone, an ultrasound examination should confirm the presence of an embryonic vesicle and most important, weekly doses of progesterone should be less than 0.7 ng/mL.

An important side note is that synthetic progesterone in the commercial product is not detected by standard laboratory progesterone analysis; thus, the only way to monitor effectiveness of treatment is the use of ultrasound periodically to confirm the presence of embryonic vesicle, embryo, fetus, or fetal components.

Miscellaneous Conditions. Physical abnormalities should be noted. A horizontal vulva (shelving) may be seen in old, emaciated, or poorly conformed females. The labia may lack tone. Nothing is really known as to how or whether these may contribute to infertility. They are of concern in the mare or cow.

Inflammation of the reproductive tract includes vaginitis, cervicitis, metritis, endometritis, and pyometra. True venereal diseases are rare in SACs. Brucellosis (*Brucella melitensis*) has been reported in South America but not in North America (Chapter 7).

The normal flora of the vagina and uterus is not known. Numerous species of bacteria have been cultured from infertile females, but Koch's postulates have not been demonstrated.[53] Perhaps, as in other females, the organisms are opportunistic pathogens. It should be kept in mind that in about one-third of the infertile females cultured, samples show no bacterial growth simply because the cause of infertility is not a uterine infection, but rather some other disturbance involving the ovary or even the pituitary. This may be either accurate or a result of technique error.

In the clinic of the author (Walter Bravo) some years ago, cases of acute and peracute metritis were usually associated with a hemolytic *Escherichia coli* infection. *Arcanobacterium pyogenes* was the second most prevalent organism cultured. In 2006 the bacteria most often isolated from uterine culture were *Escherichia coli*, *Enterococcus* spp., and *Klebsiella* spp. In 2007, 55% of isolates were *Pseudomonas aeruginosa*, 18% *Enterobacter* spp., 9% *Escherichia coli*, and 9% *Streptococcus* sp. The remaining 9% yielded no bacteria. In 2008, 51% were *Pseudomonas aeruginosa* and *Pseudomonas fluorescens*, 11% *Escherichia coli*, 6% *Arcanobacterium pyogenes*, 6% *Streptcoccus* spp., alpha hemolytic, 6% *Proteus* spp., and 20% did not yield any bacteria. More than 50% of isolates are Pseudomonas. This bacterium is a normal inhabitant of the reproductive flora, but it may become virulent and wreak havoc in the endometrium. Unfortunately, it is resistant to most antibiotics. Some cultures showed sensitivity to Amikacin, Gentamycin, and Enrofloxacin, but others showed resistance to at least one of those antibiotics. Thus, a treatment protocol should be delayed until results from the laboratory are available.

After treatment with the appropriate antibiotics, a second culture may show the continued presence of Pseudomonas, requiring a second round of treatment. It is recommended to rest the female for two to three weeks following treatment to allow the uterine defense mechanism to rejuvenate.

Abortion

Abortion is defined as the loss of a fetus from forty-five days gestation to term.[113] Prior to forty-five days, the loss of a conceptus is considered embryonic mortality.[80,81,84]

Signs of Abortion. Signs of abortion include a placenta protruding from the vulva, presence of a dead fetus in a paddock or pen, or indications of receptivity to a male.

Prevalence. In general, the percentage of abortion after sixty days gestation is about 2%. Recently, a study in New Zealand reported a 5% fetal loss after 100 days of gestation. In a separate study, a 10% to 17% loss was observed in alpacas.

Causes. Infectious causes reported include *Brucella melitensis*, listeriosis,[91] chlamydiosis,[78] toxoplasmosis,[78] leptospirosis,[78] and equine rhinitis virus.[130] Additional causes include stress, hormonal imbalances, and administration of corticosteroids.[27,99] Dozens of other causes of abortion have been identified in horses and

livestock, and given time, SACs will likely become the victims of many of these.

Unfortunately, a precise diagnosis will be made in less than 50% of abortions. Nonetheless, every effort should be made to collect samples and begin recording data.

TREATMENT. Every female that aborts should be examined to exclude an intrauterine infection. In most early gestation abortions, no special therapy is indicated.

Late term abortions may require more attention. Often there is a discharge from the vulva that may vary from odorless to foul smelling. A foul-smelling exudate may be associated with necrotic tissue. If the placenta or any part of it remains in the uterus, 20 IU of oxytocin should be administered, followed by voluminous flushing with physiological saline. Antibiotics (parenteral and intrauterine) may be indicated.

Infertility Examination

It is vitally important that systematic, thorough examinations be conducted and that detailed records be kept. It is only through such records that it will be possible to amass sufficient data to serve as a sound basis for rational evaluation. Table 17.5 is a suggested examination form.

The examination should be preceded by a detailed history. Unfortunately, infertile females are frequently shifted from one owner to another, often without the new owner being aware of the problem. Females with no ovaries have been "guaranteed" to have had two or three crias.

The vaginal examination may be conducted using a heifer glass vaginal speculum (2.5 cm outside diameter, 25 cm long). Specula that are constructed for use in goats are too short to reach the cervix (15 cm). In larger and multiparous females, a standard equine glass or cardboard speculum may be used, but it is longer than necessary (4 cm in diameter, 25 cm long). A piece of polyvinyl chloride plastic pipe of suitable diameter may be smoothed off on the end or fired and used as a speculum after sterilization. A last alternative is the use of a human proctoscope which accommodates itself very well to the vulva and vagina of the female alpaca. This vaginal speculum could be attached to a light source to facilitate examination of the cervix. It should be remembered that the cervix opens when there is an ovulatory-sized follicle in the ovary, and then remains closed. A swab culture from the cervix should be taken when the cervix is open, rather than forcing the insemination rod and/or the tip of a swab culture and damaging the cervix. In some cases of uterine infection the cervix is closed and there is no discharge from the uterus. It is wise to wait until the cervix opens to observe the nature of the discharge. If discharge is seen its presence may be noted on the ventral aspect of the female's tail. Any thick, yellowish dry matter should be removed, because during the next examination, if the tail is has the yellowish matter the female is still infected.

It is possible, in multiparous females examined by a small-handed person, to manually palpate the vagina after suitable cleansing and gloving. Rectal palpation is a valid and necessary procedure for evaluation of the reproductive tract. It can be done safely on all but the smallest females and virginal alpacas. Obesity greatly restricts movement in the pelvic canal and may preclude an adequate examination. A detailed description of rectal palpation has been given previously. It is unfortunate, but some owners have been swayed by exaggerated rumors of the dangers of rectal palpation and will not allow such an examination to be made. It may then be impossible to evaluate an infertile female and recommend a course of action.

Ultrasonography is a valuable aid not only for pregnancy diagnosis but also for conducting the infertility examination. The rectum should be lubricated with the appropriate gel to facilitate the conductivity of radio waves. A systematic and organ-by-organ ultrasound should be conducted. As a landmark, the urinary bladder is visualized, and immediately the presence of urine as a nonechogenic structure serves as point of beginning. As the probe is introduced gently the cervix becomes apparent, and if the animal is pregnant the cervix shows two or three distinct hyperechogenic areas (rings of the cervix). Then the body of the uterus is visualized, and by moving the probe both uterine horns, as round structures which appear as hypoechogenic, homogenous, should appear. The diameter of the uterine horns should be observed. In maiden females, they should be around 4 to 5 cm. When there is infection in the uterine horns, the presence of discrete and irregularly shaped hyperechogenic structures are apparent. In addition, there are lymphatic vessels of irregular shape at the bottom of the uterine body. In the early postpartum female, lymphatic vessels are pronounced and manifested. Ovaries are usually located on the side of the uterine horns, but sometimes they are in the center. The presence of follicles, which appear as rounded structures with fluid and are nonechogenic, serves as a basis to identify the ovaries. The presence of a cystic follicle (greater than 13 mm in diameter) is unmistakable. Sometimes ovaries are not found easily because of the presence of large cystic follicles; the weight of these follicles drops the ovary to the bottom of the abdominal cavity.

A corpus luteum will appear as large as the ovary itself, and as a clear hypoechogenic rounded structure at the periphery of the ovary. When the female is losing its embryo, the embryonic vesicle appear with irregular hyperechogenic structures, and the delimiting outside border is disrupted and not well defined.

Table 17.5. Llama reproduction examination—female.

Identification Date _____

Name_____ Age _____ Owner _____

ISIS No. _____, Llama Registry No. _____

Color

Body _____ Neck _____

Head _____ Legs _____

History

Previous pregnancies—0, 1, 2, 3, 4

Life births—0, 1, 2, 3, 4

Abortions—0, 1, 2, 3

Breeding dates during past 6 months _____

_____

Number of males used _____

Fertility of male—known _____ unknown _____

Response to male, past 2 months _____

_____

Previous medical problems _____

_____

Previous fertility treatments and response _____

_____

Fertility rate of the herd _____

_____

Rectal examination

Cervix diameter _____ cm, length _____ cm

Cervix tone _____

Right horn

Diameter _____ cm, length _____ cm

Left Horn

Diameter _____ cm, length _____ cm

Consistency _____

Right ovary _____ × _____ × _____ cm

Left ovary _____ × _____ × _____ cm

structures present

Pregnant _____ yes, _____ no, _____ months

Vaginal examination

Vulva

Conformation _____

Discharge _____

Hymen _____

Vaginal mucosa

Color _____ Exudation _____

Cervical os

Cytology _____

Exudation _____

_____

Cervical culture _____

Uterine culture _____

Uterine cytology _____

Uterine biopsy _____

Hormone assay _____

Progesterone _____ ng/dl

Ultrasound evaluation _____

_____

Other diagnostic procedures—laparoscopy, laparotomy _____

_____

Conclusions _____

Uterine cytology,[106] endometrial cultures, and sensitivities are vital for selection of proper therapy for metritis. An equine uterine culture swab (Tiegland) is recommended. It is useless, in fact misleading, to obtain a culture from the vagina or external os of the cervix. Although it is difficult to thread a tube through the cervix, by gentle persistence it can be done. The cervix relaxes only when a follicle is present in the ovary and allows access to the uterus.

Two approaches may be used. A small-handed person may enter the vagina with a gloved hand. A finger should be used to locate the cervical os and begin dilatation. The tube should be inserted alongside the arm, up to the finger, and gently pushed forward with periodic changes of direction to move over the rings. In another method, a short vaginal speculum is pressed around the os of the cervix. The culture tube is inserted through the os and the manipulation continued by visual contact, or the speculum may be withdrawn and allowed to rest on the tube while the other hand is inserted into the rectum to grasp the cervix to aid in the threading process. There is usually insufficient room for both a speculum in the vagina and an arm in the rectum in the SAC. Endoscopy is now being used to guide pipettes and swabs.

Cultures should be interpreted in relation to the cytology. If no inflammatory cells or discharge are present, the organism cultured may not be a pathogen.

Endometrial Biopsy. The histologic evaluation of the uterine mucosa has become a standard diagnostic technique in mares and cows suffering from infertility. Much less experience has been gained in SACs, but the technique and histologic picture of the endometrium are known. The technique is similar to passing the swab for a culture. The tip of the biopsy forceps must be threaded through the cervix, and with a hand in the rectum to fix the uterus, a snip of tissue may be pinched off the mucosa. Some of the larger equine forceps have jaws that may be too large for an SAC; consequently, a heifer forceps is most appropriate for llamas and alpacas. Injudicious use may penetrate the uterine wall. Cultures can be obtained from the biopsy tissue before placing it in fixative.

Each clinician should be consistent in the general location from which the specimen is collected. Unless a uterine mass must be biopsied directly, the lateral wall of the left horn is an appropriate location.

The uterine mucosa consists of an epithelial layer of tall columnar cells and a connective tissue layer containing the glands. The density of the connective tissue is greater and the number of glands in the mucosa much less in an SAC than in a mare, leading to misinterpretation by inexperienced pathologists. Other lesions of inflammation are similar in both species.

Pigmented macrophages have been observed in the endometrium. This may be suggestive of abortion, parturition, or other causes of hemorrhage, as it is in mares. Fibrosis of the uterine glands is an unfavorable lesion. SACs do not cycle as most mammals do; therefore, a variable histologic picture associated with stages of the estrous cycle cannot be described.

Laparoscopy.[54,73,117,120] Laparoscopy has been used effectively in experimental work to document ovarian activity during various stages of the reproductive cycle. It is possible to visualize the ovaries, oviducts, and uterine horns via a right-flank approach, with the animal in a standing position. Mild sedation with butorphanol and local anesthesia is used. The abdominal cavity must be inflated with an inert gas, and the distention may cause discomfort in the patient.

In the male, intraabdominal vasectomy may also be done using laparoscopy. The male is fasted for at least twenty-four hours. A general anesthetic is administered, and then the area ventral and lateral to the penis is prepared aseptically. The male is positioned in Trelenderburger position, with the rear legs up and the head down. Two incisions are made; one is used to insert a trocar cannula which is then replaced by the rigid telescope. The other is for a biopsy and/or cutting forceps. The abdominal cavity is insufflated moderately until the vas deferens are located on the sides of the male. They are white and glistening. Using the cutting forceps, a 2-cm segment of each vas deferens is removed. Surgical material is withdrawn, air is pushed out by manual pressure, and the two skin incisions are sutured with simple interrupted sutures.[48,73]

Hormone Analysis. Progesterone levels have been used extensively to determine pregnancy in llamas, but it should be reiterated that positive levels indicate only the presence of a CL. Repeated or periodic progesterone samples helpful in evaluating certain infertility problems such as recurrent early embryonic death. Levels of progesterone have been detected in milk and urine.[f] Estrone sulfate and relaxin have also been studied in pregnant llamas and alpacas. Estrone sulfate shows a bimodal peak, the first during the first twenty-one to twenty-seven days of gestation and the second during the last month of pregnancy. Relaxin, though still in the experimental stage, may prove to be an accurate indicator of pregnancy after month 2 of pregnancy (see description under Pregnancy Determination).

Treatment of Infertility and Metritis

Techniques currently being used to treat infertile mares and cows are being applied to SAC. Statistics are not available as to the efficacy of various therapies.

Sexual rest is an important management tool. Animals that have experienced any difficulty with par-

turition should be given at least thirty days for optimal uterine involution before being exposed to a male.

Various hormones have been administered to livestock and horses to correct deficiencies based on actual measurement of hormone levels or on the basis of clinical signs and palpation examinations. No baselines have been established in llamas or alpacas for most of the hormones, so hormone therapy must be based on extrapolation from regimens developed for other species. Before these drugs are prescribed, it is important to be certain that the basic reproduction cycle of SACs is understood. It is important to use caution in dosages of the hormones.

Discussion of therapy for metritis usually includes a list of antibiotics that have proven useful. Such a list is inappropriate for SACs because no consistency of therapy has been established. Clinicians in different regions of North America administer the antibiotics they use in mares or cows, but the data have not been correlated to determine efficacy.

The trend is to minimize the use of uterine flushes with disinfectants or antimicrobials and instead to use physiologic saline to thoroughly wash out all exudate. One therapeutic regimen is to flush the uterus as described, finishing with a uterine infusion of llama plasma.

Infertility Conditions of Female Camels

Infertility conditions of female camels are basically the same as those in SAC females. Tibary discusses the following conditions in detail: ovarian hypoplasia, ovarian bursal adhesions, ovarian neoplasia,[94] laceration of the cervix, rectovaginal fistula, rectovaginal prolapse,[77] uterine infection, retained CL, endometritis, early embryonic death, cystic ovaries, ovarian inactivity, uterus unicornus, infantile uterus, vaginal prolapse, retained placenta, uterine prolapse, and vulvar and vaginal lacerations.[14] Surgical conditions are described in the chapter on surgery for SACs. The procedures are similar for camels. Camel obstetrical procedures are the same as for SACs.

Male Llamas and Alpacas

Etiology

Little investigation has been carried out to delineate the various causes of infertility in male SACs. Congenital defects are some obvious causes and include such conditions as cryptorchidism, ectopic testicle, hypogonadism,[122] persistent frenulum, prepucial adhesion, small penis, curvature of the penis, and intersex.

Hyperthermia. See Chapter 9 for a detailed discussion of this important topic.

Hydrocele. The author has seen two llamas with scrotal hydrocele. In one case, the cause was an abscess at the external inguinal ring, obstructing fluid flow into the peritoneal cavity. In the other case, the etiology was not determined. Sterile, normal fluid was contained between the visceral and parietal tunics. There was no evidence of an inflammatory response. Ultrasonic examination confirmed the presence of nonpurulent fluid. Differentiate hydrocele from scrotal edema, which is a characteristic sign of hyperthermia in male SACs.

Trauma. The prepuce, scrotum, and testicles may be traumatized during aggressive intermale encounters. The wounds should be managed as any other lacerations (Chapter 6). Unattended lacerations of the prepuce may result in posthitis and, ultimately, stenosis and phimosis (inability to extrude the penis).

A previously successful breeding male began to fail to complete copulation. He chased the females and assumed his usual breeding position, but at the time intromission should have occurred, progress stopped, and he arose and left. Upon examination, it was observed that a stricture prevented full extension of the penis. Attempts to forcibly extrude the penis elicited a pain response, even though the llama was sedated. This male exhibited a strong libido, but because erection had become painful, he simply quit.

Another source of trauma to the penis is entangling fibers from the female's perineal region. North American llama owners are reluctant to clip fleece from their animals, but the perineal area of all breeding females should be clipped sufficiently to preclude fibers from obstructing copulatory efforts. One or two caught fibers may lacerate the glans and cause balanitis. If fibers wrap around the glans and become twisted, ischemic necrosis may occur.

Prepubertal adhesions may persist into adulthood.[98] Stenoses of the prepuce are serious impediments to successful breeding; surgical correction is usually necessary. Standard techniques used on bulls have been successfully employed in SACs and may include multiple triangular release reconstruction surgery or removal of circumferential collar and resuturing of the preputial mucosa. Infectious diseases of the male reproductive system are either rare or have not been reported. Brucellosis, caused by *Brucella melitensis*, has been reported in South America but not in North America. Trichomoniasis and campylobacteriosis are unknown. Two cases of breeding males with Pseudomonas infection have been observed by Bravo. In both cases, a female was found infected with the same bacteria. Lavage with antibiotics was done in the following manner: The antibiotic was diluted with saline solution and 50 cc of the mixture was placed inside the prepuce. The opening was held tight for about five minutes. This procedure was repeated for five consecutive days. A second culture from the glans penis

was negative to bacterial infection, and the male returned to the breeding program.

No specific abscesses have been observed in the testicle. Infections are frequently a sequel to lacerations and severe contusions.

Hormonal Deficiencies. Hypogonadism is likely the result of low levels of testosterone, but little is known about normal levels in male SACs. Certainly, some males lack libido, but whether this is caused by a hormonal defect or is a behavioral defect is unknown.

Hormonal treatment of hypogonadal testicles in adult males is questionable. If the testicles are small, there could be a genetic component which makes the male with small testicles a poor breeder prospect. It should be also considered that in South America only 5% of the males are selected as sires, which means that only outstanding males in every sense of the word should be sires. The same pressure of selection has been used for many years in other livestock species. Moreover, there is a relationship between fertility and testicular size. See text on page.

Behavioral Abnormalities. Males may be overly timid or hyperaggressive. Such behaviors may be inherited, as in other livestock species, or they may be developed. A young male that is just starting to breed may be intimidated by an aggressive female. The major known behavioral anomaly is that of the human-imprinted (rogue) male (Chapter 3). Such behavior does not make the male infertile, but it may preclude his usefulness as a breeding male because of the danger to humans who must handle him.

Infertility Examination

A fertility examination should begin with a complete history and a thorough physical examination to identify problems that may preclude breeding, such as arthritis, or that may inhibit mounting and copulation. In the fertility examination, the use of a chart such as that illustrated in Table 17.6 facilitates completeness. The individual should be identified by registration number (the number should be seen) or by color and markings.

An examination for breeding soundness varies somewhat according to the age of the animal and the reason for the examination. An examination for sale of a breeding male should encompass a complete set of parameters. The examination of a weanling or yearling as a potential breeding male must, of necessity, be limited. The examination of a male with identified or suspected fertility problems is complex.

Breeding males may also be examined for insurance purposes. The veterinarian must ascertain whether the insurance is for mortality only or if the client has contracted for breeding assurance, in which case much more detail is required in the way of examination and evaluation.

History is vital. Has the male bred previously? Has breeding resulted in living crias? Is he aggressive or shy? Is he intimidated by dominant females? What is the status of his libido? It would be desirable to see the male actually breeding a female, but most of the history must be obtained by judicious questioning of the owner. The owner may or may not have been able to determine whether or not the male has made penetration.

A thorough examination of the male reproductive organs should be conducted, including evaluation of the testicles for size and consistency. The testes should be measured. The epididymis should be palpated. Measurements of the penis can be made only with the penis extruded from the prepuce. This requires tranquilization or anesthesia. Some information may be obtained by palpation through the skin. Rectal palpation should enable examination of the accessory sex glands, especially the bulbourethral glands.

Semen Collection.[28,66,88] A complete fertility examination should include semen evaluation. This is difficult in SACs, since no method has been effective in collecting semen from every animal. Artificial vaginas have been employed to collect semen from stallions, bulls, rams, and alpaca males. Males must be trained to use the dummy female, so this method is applicable only for research projects.

Electroejaculation has been of limited effectiveness. The male must be sedated and even lightly anesthetized. Various types of ejaculators have been used. The exact power, pulse frequency, and duration needed have not been established. In the author's clinic, a variable-voltage bovine unit has been used, attached to a three longitudinal electrode ram probe. Before proceeding, the feces should be removed from the rectum. The technician must be prepared to collect semen at this point and also when the probe is inserted, since either stimulation may cause premature ejaculation. It is most desirable to manually extrude the penis from the prepuce prior to any rectal stimulation, so that semen may be collected from the penis rather than from the prepuce. Contamination of semen with urine and absence of a well-developed ampulla are the factors that make electroejaculation less than optimal as a technique for semen collection.

Under clinical situations, the most reliable and effective method of semen collection is retrieving it from the vaginal fundus. This method takes advantage of three features of breeding. First, ejaculation is constant throughout copulation. Ejaculation begins after two minutes of penis insertion into the female reproductive tract, and ends right before male withdrawal. Second, semen is gelatinous and seminal plasma holds spermatozoa. Third, semen is also left outside the uterus when the male is repositioning between the two

Table 17.6. Llama reproduction examination—male.

Identification Date _____
Name _____ Age _____ Owner _____
ISIS No. _____, Llama Registry No. _____

Color
Body _____ Neck _____
Head _____ Legs _____

History
Born at present ranch—yes _____, no _____
Age at time of purchase _____
Pastured or housed alone—yes _____, no _____
Number and sex of other animals in the group _____

History	Prior to purchase	After purchase
Number of crias sired	1, 2, 3, 4, 5, +	1, 2, 3, 4, 5, +
Number of females bred	1, 2, 3, 4, 5, +	1, 2, 3, 4, 5, +
Age at time of first breeding	1, 2, 3, 4, 5	1, 2, 3, 4, 5

Physical examination
General body condition Good _____, thin _____, fat _____
Conformation faults _____
Testicles _____
Right _____ × _____ × _____ cm
Left _____ × _____ × _____ cm
Consistency _____ firm _____ soft _____ hard _____
Nodules _____ Other _____
Epididymus _____
Abnormalities _____ Cryptorchid—yes _____ no _____
Penis
Length _____ cm, diameter _____ mm
Abnormalities
Preputial adhesions _____, curvature _____

Semen evaluation
Volume _____ ml, color _____
pH _____, density _____
Concentration _____ million/ml
Motility _____
Live/dead _____ %
Abnormal forms _____

Behavior
Hand reared—yes _____, no _____
Aggressive to people—yes _____, no _____
Precopulatory behavior
Aggressive _____, timid _____
Copulatory behavior
Proper position—yes _____, no _____
Penetration—yes _____, no _____

uterine horns. Hence, following copulation and with the aid of a warmed vaginal speculum and a warmed insemination pipette attached to a syringe, semen may be retrieved from the posterior os of the cervix. A phase contrast microscope and a prewarmed glass slide are needed to determine motility. Then the preparation may be stained for live/dead evaluation and morphology.

Concentration is difficult to assess but not impossible. Motility, live/dead, and morphology of spermatozoa may be determined. The presence of large numbers of red blood cells may impair a fair assessment of sperm morphology. Live spermatozoa may be determined using a drop of Hancock's stain. Table 17.7 collates the results of examinations of twenty-two alpaca and eight llama sires.

Table 17.7. Sperm analysis of 22 alpacas and 8 llamas.

Species	*Motility (%)*	*Live spermatozoa (%)*
Alpacas	68 (50–90)	85 (70–95)
Llamas	71 (50–90)	78 (68–90)

Statistics are unavailable on the desirable ratio of normal to abnormal sperm, but in a fertile male, at least 70% of the sperm should be normal.

The morphology of the sperm should be evaluated at 1000×. It may be necessary to dilute the semen to separate the seminal fluid, which can be done during the staining process by mixing the sperm with a larger volume of stain A prior to making the smear. A variety of stains may be used.

Evaluation of the motility of SAC sperm in the viscid semen may be misleading. SAC spermatozoa are much slower than those of rams or bulls because of the thick seminal fluid. It is imperative that the semen be maintained at a constant temperature of 37°C from the time of collection until motility is evaluated; otherwise, motility should not be considered in the fertility examination.

Advanced Reproductive Techniques[28,37,107,133]

Artificial insemination, embryo transfer, oocyte recovery from ovaries, and in vitro fertilization have been reported, but the techniques need more investigation and development if they are to become practical procedures.

Artificial Insemination[58,107,132–134]

INDICATIONS. Artificial insemination (AI) is used primarily to increase the production of crias from a clearly superior male. It may also be used in cases of physical incapacitation of the male or female.

APPLICATION IN SOUTH AMERICAN CAMELIDS. AI was first performed in Peru as early as 1968.[101] Subsequently, AI has been investigated as a possible adjunct to natural breeding in both North and South America.[89] The question as to the characteristics of a clearly superior SAC remains unresolved. The following account is a summarized discussion of the state of artificial insemination in llamas and alpacas in Peru, and mimics the procedure of semen extension and preservation as has been developed in other livestock species.

Semen degelification was achieved using mainly two enzymes, trypsin and collagenase. Semen dilution has also been accomplished with phosphate buffer, egg-yolk citrate, skimmed milk, and Tris buffer with egg yolk. The best extender is Tris buffer with egg yolk citrate. Spermatozoa were maintained alive for more than twenty-four hours under refrigeration. Glycerol has been used successfully for semen freezing and a concentration of 7% of glycerol is adequate. Semen was also frozen successfully using vapors of liquid nitrogen but freezing must be done slowly. A rapid freezing killed spermatozoa. The survival rate of spermatozoa in frozen-thawed semen is between 30% and 50%. Insemination with fresh diluted semen and thawed semen have both resulted in the birth of live crias. Semen preservation requires considerably more experimentation and improvement before AI becomes practical.

Embryo Transfer

INDICATIONS. Embryo transfer (ET) is primarily valuable for enhancing the production of a superior female. It is suggested that female recipients could be of poorer quality or could have nonreproductive system congenital defects.

APPLICATION IN SOUTH AMERICAN CAMELIDS. ET was first attempted by surgical recovery of embryos in 1968 by Novoa and Sumar. Subsequently a number of investigators have used both surgical and nonsurgical procedures with variable success.[3,17–19,51,55,59,85,86,131] Jane Vaughn in Australia, in 2008, succeeded in producing more than 1,000 crias through embryo transfer with fresh embryos. In this case, embryos were collected after breeding and without manipulation of the ovaries.

Protocols to induce production of multiple follicles in the ovaries are many in other livestock species. In llamas and alpacas, FSH and eCG have been used effectively. Embryos have been collected surgically as reported by Novoa and Sumar, but the best option is to flush the uterus with a catheter inserted through the cervix. In this sense, it is easy to make the ovaries grow multiple follicles; these follicles will also ovulate after breeding with an intact male plus the administration of luteinizing hormone. Freezing embryos is being reported by scientists not only in South America but also in other countries of the world. Dr. Paul Taylor, a llama breeder in the United States, feels that this is the coming technology of the llama industry.[g]

In-vitro maturation, fertilization, and culture of SAC oocytes have been also explored. Protocols from other species have been extrapolated to llamas and alpacas. It is an exciting technology and the future will bring tangible results of the application of these technologies for llamas and alpacas.

SELECTION OF BREEDING CAMELIDS

A prepurchase examination of a breeding animal should include a complete soundness examination, with special emphasis on the reproductive system. A veterinarian is likely to be restricted to noninvasive procedures, but the checklists provided should guide the clinician in obtaining pertinent information (Tables 17.8 and 17.9).

Table 17.8. Checklist for a female intended as a breeder.

Breeding History

Previous pregnancies: 0, 1, 2, 3, 4, _____
Live births: 0, 1, 2, 3, 4, _____
Date of last birth _____
Abortions/Stillbirths: 0, 1, 2, _____
Breeding dates during past months _____
_____
Number of males used _____
Fertility of male: Known _____, unknown _____
Response to male _____
_____
Previous medical problems _____
_____
Previous fertility examinations, treatments and response: _____
_____
_____
Fertility rate of the herd _____

Noninvasive examination

Conformation of the vulva

Vertical _____, horizontal _____; Prominent clitoris _____
Discharge from vulva _____
Hymen: Present _____, absent _____

Mammary gland

Number of teats present _____, size of teats _____
Palpable swelling _____
Secretion from gland _____

Pregnancy status

Pregnant _____, nonpregnant _____
Method of determination: Rectal palpation _____, ultrasound _____, progesterone _____

Detailed examination

Rectal examination

Cervix: Length _____ cm
Right horn
Diameter _____ cm, length _____ cm
Left horn
Diameter _____ cm, length _____ cm
Tone of uterus _____, pregnant: yes ____, no ____
Right ovary
Size _____ cm; structures present _____
Left ovary
Size _____ cm, structures present _____

Ultrasound examination

Pregnant: yes _____, no _____
Abnormalities _____
_____

Vaginal examination

Partial persistent hymen _____; tags _____
Vaginal mucosa
Normal: yes _____, no _____; Discharge _____
Cervical opening
Normal: yes _____, no _____; Discharge _____

Note: The extent of the examination depends upon an agreement between the buyer and seller, the expertise of the examining veterinarian, and the purchasing situation. A purchase at an auction rarely allows a detailed examination prior to purchase. This is a distinct disadvantage. I would push for an agreement that would be contingent upon a thorough examination either prior to purchase or immediately upon arrival at the new owner's premises.

Table 17.9. Checklist for a male intended for a breeder.

Behavior
Hand reared: yes _____, no _____
Aggressive to other llamas: yes _____, no _____
Aggressive to people: yes _____, no _____
Precopulatory behavior
Aggressive _____, timid _____
Copulatory behavior
Proper position: yes _____, no _____
Penetration: yes _____, no _____

Physical examination
Testicles
Right: Size_____cm, consistency_____
Left: Size _____cm, consistency_____
Abnormalities _____
_____
Penis
Length _____cm, diameter _____mm
Abnormalities _____
_____

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* Dr. P. Walter Bravo is a reproductive endocrinologist with vast experience in the study of reproduction and reproduction problems in South American camelids both in North and South America.

[a] The term used in Peru is cutuneo.

[b] P.W. Bravo et al. 1997, unpublished data.

[c] W.L. Franklin, personal communication.

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18

Urinary System

ANATOMY

Kidney

Camelid kidneys are nonlobulated, shaped like those of a sheep (Figure 18.1). Both kidneys are the same size (approximately 5 × 9 cm) and fixed in the dorsal abdominal area. The left kidney is located ventral to the transverse processes of the fifth to seventh lumbar vertebrae. The right kidney is located slightly more cranial, beneath the fourth to sixth transverse processes of the lumbar vertebrae. Kidney weight varies from 120 to 170 g in llamas weighing from 74 to 120 kg. Microscopic anatomy has been described.

Ureters and Bladder

The ureters and bladder are like those of cattle and sheep.

Urethra

The male urethra is described in Chapter 17. A dorsally situated urethral recess at the ischial arch of SAC males prevents passage of an urethral catheter into the bladder (Figure 18.2).[31] The author doesn't know if the recess is present in camel males. Camel anatomy texts do not describe it. It is possible to pass a urinary catheter into the bladder of the camel male, which would indicate the lack of a recess. The female urethra of camelids is large, with the external orifice located at the ventral border between the vagina and vestibule. A ventral diverticulum at the external orifice makes female urethral catheterization difficult but not impossible.

CHARACTERISTICS OF CAMELID URINE[1,9,28,29]

The composition of urine is known.[1,6,15] The urine of 138 alpacas maintained at an elevation of 4,200 m was analyzed in Peru (Table 18.1).[3,4] In general, there was insignificant variation with age, sex, or physiologic state except for ketones. Acetoacetic acid was detected in 42% of the nonpregnant alpacas and in 100% of the pregnant alpacas. Acetone was detected in only 2% of the nonpregnant alpacas but in 88% of the pregnant alpacas. The significance of urinary ketones is unknown because it is an uncommon clinical finding. It may be detected in hepatic lipidosis cases.[1]

Urea is excreted by adult alpacas at 6.08 g/L urine. Urea excretion is slightly higher in youngsters as compared with adult animals, and in pregnant animals it is higher yet. Animals consuming diets containing high levels of protein (>15%) have higher levels of serum urea nitrogen (SUN) and likewise have higher urinary nitrogen levels. In general, alpacas have higher rates of urea excretion than any other animal except humans. The amount of ammonia excreted (0.13 g/L) does not vary with age or sex.[6] Renal function has been discussed for camelids.[5,6,34] Urine collected from normal llamas in California had similar characteristics to those of alpacas. Table 18.2 compares urine characteristics from camelids, bovids, equids, and canids. Table 18.3 provides data for selected urinary tract diseases.

Camel urine is different than SAC urine.[21] The physiology of urine production, storage, and voiding are important considerations for the camel clinician. The composition of camel urine is highly variable depending on the availability of water, dehydration status and environmental factors such as ambient temperature and availability of plants that are resistant to drought.

Camel urine is normally clear and pale yellow in color. The pH varies from 6 to 9. The specific gravity is highly variable depending on the degree of dehydration. Extreme dehydration results in a urine of a syrupy consistency. Calcium oxalate and calcium hydrogen phosphate crystals may be observed in urine sediment.

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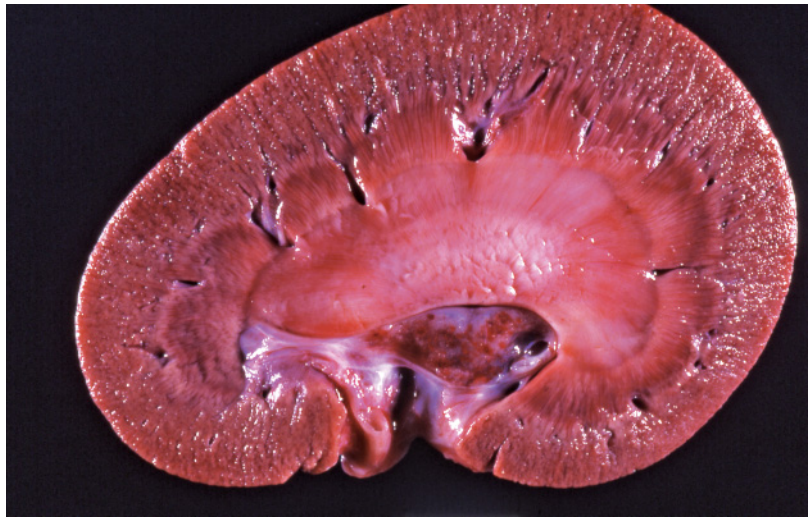

Figure 18.1. Longitudinal section of a camelid kidney.

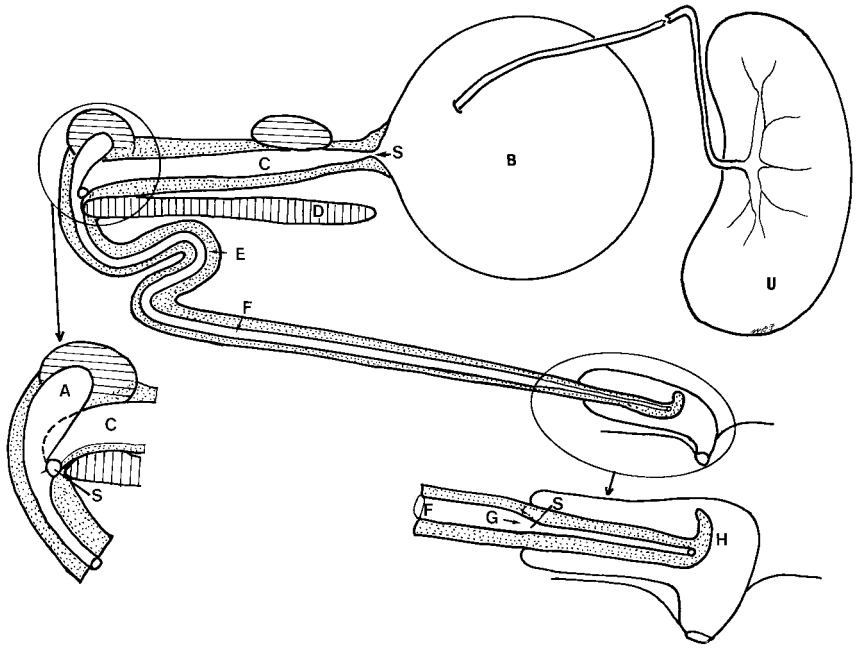

Figure 18.2. Diagram of the urinary tract of a male camelid. (A) Urethral recess, (B) bladder, (C) pelvic urethra, (D) pubic bone, (E) sigmoid flexure, (F) penile urethra, (G) beginning of the glans penis, (H) cartilaginous projection at the tip of the penis, (S) point of narrowing (potential site for urethral obstruction), (U) kidney.

DIAGNOSTIC PROCEDURES

Urine may be collected as a free catch during urination at a communal dung heap, by urethral catheterization in the female, and by cystocentesis in both the female and the male SAC by fixing the bladder per rectum and inserting a 10– to 15-cm (4– to 6-in.) 18-gauge needle on the midline just cranial to the pubis. The area is surgically prepared, and in the male the penis and prepuce are pushed laterally to allow the midline insertion of the needle.

Urinalysis may be conducted in the field using dipsticks to evaluate common parameters (glucose, pH, ketones, proteins, blood). A refractometer may be used to determine specific gravity. More sophisticated procedures are done in the laboratory, including electrolyte levels, quantifying protein and other solutes, and cytology.

Urinary tract diseases are diagnosed by a combination of clinical signs, urinalysis, serum biochemistry, hemogram, and special procedures such as renal angiography, radiography, ultrasonography, and cystoscopy in the female.

Table 18.1. Normal camelid urinalysis.

Parameter	*Alpaca kept at 4,200 m N = 138*	*Llamas kept at sea level N = 15*	*Dromedary camels*
Volume (L/24 h)	0.125–3.8 (1.06)		1.5–5.0
Color	Clear, yellow to amber	Clear, light yellow to amber	Clear, light yellow to dark yellow
Specific gravity	1.010–1.048 (1.021)	1.013–1.048 (1.023)	1.022–1.070
PH	Alkaline 94%, acidic 2%	7–8.5	4.5–8.7
Protein	Negative	Negative	Negative
Glucose	Negative	Negative	Negative
Ketones	50% + at 1	Negative	Negative
Urobilinogen	52% = at 1	Negative	Negative
Bilirubinogen	45% = at 1	Negative	Negative
Indican	45% +	Not run	Negative
Blood	Negative	Negative	Negative
Sediments	Triple phosphates common, ammonium urate rare	Calcium oxalate common in alkaline urine, uric acid rare	Calcium oxalate, calcium hydrogen phosphate, ammonium urate, triple phosphate

Table 18.2. Characteristics of normal urine.

Parameter	*Camelid*	*Bovine*	*Equine*	*Canine*
pH	7.0 to 8.5, alkaline	Alkaline	Alkaline	Acidic
Specific gravity	1.010 to 1.048	1.015 to 1.045	1.020 to 1.050	1.020 to 1.045
Color	Clear, yellow to amber cloudy standing	Clear, but becomes	Cloudy	Clear
Solids	Calcium oxalate, uric acid rarely	Phosphates	Calcium carbonate	Uric acid

Table 18.3. Urinalysis in selected diseases.

	Dehydration	*Urinary tract hemorrhage*	*Chronic nephritis*	*Cystitis*
Gross appearance	Clear	Clear	Clear	Cloudy to hemorrhagic
Specific gravity	↑	↑	↓	↑
Refractive index	↑±	↑	↓	↑
Protein	0	+1–4	+1–2	+1–4
Color	Yellowish-amber	Reddish	Yellowish	White to reddish
Glucose	0	+1	+1–2	0– +1
Ketones	0	0	0– +1	0
Cells	±	±RbC	0– +1	Neutrophils-epithelial
Bacteria	0	0	0– +1	+ +

A number of electrolyte excretion and clearance tests are used in human and other animal species to evaluate renal function. Reference ranges for camelids are now available for consideration by clinicians and diagnosticians. These tests include total excretion of electrolytes and creatinine, fractional excretion of the same substances, and electrolyte excretion compared with creatinine as an index to avoid having to collect twenty-four-hour urine samples.

The presence of certain enzymes in urine, including γ-glutamyltransferase (GGT) and N-acetyl-β-D-glucosamidase (NAG), indicate tubular activity such as leakage or necrosis.

SUN is often used as an index for kidney function. Causes for an elevated SUN may be prerenal in origin (reduced renal perfusion, hypovolemia, dehydration, starvation, high-protein diet, fever, burns, steroid administration, anabolic steroids, infection) or the direct effect of kidney malfunction (acute renal failure, chronic renal failure). Postrenal elevated SUN may arise from urinary tract obstruction or ruptured bladder. A mildly elevated SUN may simply be a reflection of anorexia and is seen as an ancillary finding in many sick camelids.

Low SUN concentrations are seen in neonatal animals and in camelids suffering from hepatic insufficiency.

Elevated serum creatinine levels are caused by the same factors that cause elevated SUN levels. The creatinine level is a better guide to renal failure because

SUN is influenced more by diet. An elevated SUN associated with an elevated creatinine is significant.

Percutaneous renal biopsy may be accomplished by guiding the needle with ultrasonography.

URINATION BEHAVIOR

SACs use communal dung piles for both defecation and urination. Camels defecate at random, but assume the same stance as SACs to urinate. With stimulation to void, both male and female SACs seek out a dung pile. At the approach, the animal will sniff the pile and, if satisfied, turn around and assume a squatting position, with the hind legs spread apart and brought forward under the body. Defecation usually occurs first, followed immediately by urination. A llama or alpaca will usually urinate two to four times a day but while carrying a heavy load in the wilderness may urinate only upon arising in the morning and again in the evening.

An adult camel, with unlimited access to water, voids up to 7 liters of urine per day in aliquots of 450 ml. The camel urination stance is similar to that of SACs, with the hind legs apart and in a semi-crouch position. Male camels may vary this position during heat stress and stand erect, spraying the hind legs, apparently to produce evaporative cooling.

A llama may refuse to urinate while being transported. This must be considered when a llama is transported by trailer to a clinic. If the bladder is distended with urine, any restraint method employing bands around the body may cause rupture of the bladder if the animal should struggle (Chapter 6). Likewise, it is wise to proceed with caution when conducting a rectal examination on an animal having a distended bladder: rupture of the bladder has occurred from application of too much pressure. A newly hospitalized llama may refuse to urinate or defecate for a few hours if on a concrete or wooden floor. The animal should be moved to a lawn or dirt area to give it a chance to void before concluding that there is oliguria or no passage of feces.

Dysuria is characterized by abnormal posture at the dung pile or by straining as if to urinate but with urine dribbling or no urine flow. It may be difficult to differentiate dysuria from tenesmus.

DISEASES

Congenital renal agenesis and persistent urachus are discussed in Chapter 22. Urolithiasis and rupture of the bladder are discussed in Chapter 6.[2,17] Primary infectious diseases of the urinary system are discussed in Chapter 7 and include leptospirosis, clostridial diseases, anthrax, coccidioidomycosis, tuberculosis, and septicemia. No parasitic diseases of the camelid urinary system have been reported.

The prevalence of acute renal failure or nephritis is low in the author's practice, but other clinicians have reported this as a frequent cause of death.[16] Secondary nephritis may be more common. Insufficient numbers of cases have been studied clinically and at necropsy to categorize nephritis (glomerulonephritis, interstitial nephritis, pyelonephritis), as has been done for other species. Iatrogenic toxicity from the administration of gentamicin has accounted for the majority of acute renal failure cases.

Other noninfectious causes of acute renal failure include hydronephrosis, urinary tract obstruction, neoplasia, autoimmune disorders, toxicities (heavy metals, oxalates, tannins, ethylene glycol), hemolytic crisis, and crotalid snakebite.

Nephrosis

Nephrosis is a degenerative disorder of either the glomeruli or tubules and may be caused by endotoxemia, renal ischemia, or nephrotoxic agents (oak tannins, oxalates, ethylene glycol, lead, arsenic, aminoglycoside antibiotics, myoglobin, hemoglobin). Renal ischemia is associated with shock and may be exacerbated by general anesthesia. In horses and dogs, surgical manipulation of the abdominal viscera results in a reflex decreased blood flow to the kidneys. A similar phenomenon may occur in camelids. Partial or complete vascular occlusion to a segment of the intestine results in a buildup of endotoxins in the lumen. If the obstruction is corrected surgically, endotoxic shock and hypotension may occur as a result of the rapid absorption of endotoxins once circulation is restored. The animal may die from the direct effects of the shock or develop renal failure from renal ischemia.

Severe, prolonged dehydration, as may occur with diarrheal diseases, may predispose to ischemic nephrosis. Excessive quantities of myoglobin and hemoglobin in the urine produce degeneration of the kidney tubules. Myoglobinuria may be seen following crushing injuries or violent struggling or it may be associated with white muscle disease. Hematuria or hemoglobinuria may be observed in renal trauma (automobile accident), copper poisoning, blood transfusion reactions, severe burns, neoplasia, dicoumarol poisoning, or urolithiasis, or following urethral catheterization, or it may be associated with infectious diseases (cystitis, pyelonephritis, urethritis, prostatitis, septicemia).

Oak Toxicity

Oak toxicity was diagnosed in a llama that had access to large numbers of acorns. The affected llama was a 134-kg female in the fifth month of gestation. Eighty other llamas were in the same pasture with access to the acorns, but this llama was the only one affected. This is characteristic of the syndrome as seen in cattle. Certain animals acquire a taste for the acorns.

Clinical Signs

The llama was anorectic, depressed, weak, and recumbent when first examined. She exhibited signs of colic (rolling, refusal to rise) and no gastric motility. A large amount of mucoid material was excreted from the rectum, but feces weren't. The fetus was aborted. Temperature, pulse, and respiration were normal. She was anuric and had submandibular and perineal edema.

Laboratory Findings[20]

SUN (136 mg/dl), creatinine (11.5 mg/dl), and potassium (2.8 mEq/L) levels were elevated, and sodium (149 mEq/L) and chloride (117 mEq/L) levels were decreased. A metabolic acidosis was present. Urinalysis results showed a specific gravity of 1.023, a pH of 8.5, a trace of protein, no glucose, a trace of ketones, and initially a 1+ blood.

Treatment

She responded to the administration of lactated Ringer's solution at 80 ml/kg/day (two times basic) to which was added 26 mEq/L of KCl. Ampicillin 10 mg/kg and cimetidine 2.2 mg/kg were administered twice daily.

Gentamicin Toxicity

Two cases of gentamicin toxicity have been reported, and the author has had a similar case.

Clinical Signs

Signs were similar to those seen in the oak toxicity, with the addition of weight loss, dehydration, bruxism, loose stool, and gait abnormalities.

Laboratory Findings

SUN and creatinine levels were markedly elevated (SUN 280 and 396 mg/dl, creatinine 21.5 and 24 mg/dl). Serum phosphorus was 13.6 and 33 mg/dl; sodium, 133 and 140 mEq/L; chloride, 89 and 105 mEq/L; and a severe acidosis, HCO_3 11 and 11.1 mEq/L. The urine was isosthenuric at 1.013 with a pH of 5.5.

Chronic Renal Failure

Some of the foregoing signs may be seen in chronic renal disease, but additional signs include anorexia, weight loss, depression, straining, and mild anemia. A low-grade fever of 40°C to 41°C (104°F to 106°F) may be seen with suppurative nephritis. The urine may contain bacteria, leukocytes, blood, and elevated levels of protein. Concentration of the urine varies, as does the quantity of urine produced, but usually the concentration is low and the volume output is high.

Uremia is the terminal syndrome of renal failure and is characterized by anorexia, weight loss, depression, muscular weakness, muscular tremors, dyspnea, oliguria, albuminuria, hyperemic mucous membranes, uremic odor from the breath, tachycardia from dehydration, recumbency, coma, and death.

Nephritis

Inflammatory lesions of the kidneys are usually caused by infectious agents, which may produce focal or diffuse glomerulonephritis, interstitial nephritis, pyelonephritis, or embolic nephritis. Clinical signs are the same as for other acute and chronic renal failures. Treatment should include antimicrobial therapy.

Treatment

Renal failure must be treated early and intensively to avoid permanent damage to the glomeruli and tubules. The animal must be carefully rehydrated to overcome renal hypotension, renal ischemia, and oliguria. Urine production should be monitored to ensure that the kidneys are able to function.

Furosemide (0.5 to 1 mg/kg) may be given intramuscularly twice daily to cause diuresis. However, furosemide is ineffective if tubular nephrosis is severe. Any electrolyte imbalance must be corrected and administration of fluids continued until the animal is able to drink adequate quantities of water.

Appropriate parenteral antibiotics are indicated for infectious nephritis and may prevent secondary infection in nephrosis. Once the animal is stabilized and eating, protein intake should be minimized by offering grass hay.

Cystitis and Urethritis

Cystitis and urethritis are more common in the female because of a shorter urethra and the possibility of retrograde invasion by opportunistic bacteria. Cystitis and ureterolithiasis traumatize the mucous membrane, predisposing to infection. In one female llama with segmental agenesis of the vagina, repeated breeding by a male resulted in penetration of the urethra by the penis and traumatic urethritis. Bladder paresis and urine stagnation predispose to cystitis. One case of a tumor of the bladder has been reported to have caused cystitis.

Clinical Signs

Urination is painful and frequent, with only small quantities of urine produced. The urine may contain blood, leukocytes, bacteria, and excessive numbers of epithelial cells in the sediment.

Diagnosis

Clinical signs and urinalysis usually suffice to confirm this diagnosis. Urine should be cultured and a sensitivity test performed.

Treatment

Diuresis and appropriate parenteral antibiotic therapy for ten to fourteen days are usually successful.

Posthitis

Posthitis is an inflammation of the prepuce.

Etiology

No specific infectious diseases cause this condition in camelids. Trauma, caused by biting during aggressive male interaction, is the usual cause. Lacerations may be minimal or severe. In male camels posthitis may be caused by masturbation in the hot desert sand. See Chapter 17.

Clinical Signs

Lacerations are evident. Heat, swelling, exudation, and dysuria may also be observed (Figure 18.3). The swelling may obstruct urine flow.

Treatment

Recent lacerations may be cleansed, debrided, and sutured. Infected wounds should be treated locally by applying disinfectants (povidone-iodine) and alternating hot and cold packs to disperse the edematous inflammatory response. Parenteral, broad-spectrum antibiotics may be administered.

Concretions of the Urinary Tract

Urinary calculi (urolithiasis, uroliths, nephrolith, bladder stone, cystolith) are formed in either the calices of the kidney, or more commonly in the urinary bladder. Small uroliths may enter the ureter or urethra and cause partial or complete obstruction of urine flow.[3,4,7–9,13,14,17–19,22,23,24,25,27–30,32] No specific studies on the pathogenesis of formation of urinary calculi in camelids have been reported. There are clinical reports of disease caused by the obstruction produced by the calculi. Camelid veterinarians must rely on information extrapolated from studies of cattle, sheep, and goats, which have urine of similar composition (Table 18.2). The composition of urinary calculi recovered from llamas at the University of California is listed in Table 18.4. Urethral obstruction may also occur with non-mineral concretions.

Urinary calculi are formed in males and females equally, but the bore (diameter) of the female urethra generally allows free passage of a calculus that may enter the urethra. Thus, obstructive urolithiasis is rare in the female. Urolithiasis has been associated with a diet high in concentrated feeds, such as are often used in zoos. Cattle pastured on grasses containing high levels of silicates may sometimes develop silicate urolithiasis, and presumably camelids grazing on such pastures may also be at risk.

A basic understanding of the camelid urethra is required to locate sites of possible obstruction and develop approaches to management. Figure 18.2 is a diagram of the camelid urethra and associated structures. The prostate gland does not surround the pelvic urethra as in carnivores. The pelvic urethra is expansive, but at the reflection around the ischium, only a tiny orifice allows passage of urine beyond this point. The anatomy of this area is further complicated by a dorsal urethral recess, which precludes any possibility of passing a catheter into the bladder from the tip of the penis. Whereas the sigmoid flexure is the probable site of the majority of bovine urethral obstructions, this is not the case in camelids. The orifice from the pelvic urethra into the penile urethra is a common site of obstruction; another is where the penile urethra narrows as it enters the glans penis.

Clinical Signs

Uroliths in the bladder or calices of the kidney rarely cause discomfort, although large and rough-surfaced uroliths may initiate a cystitis. The signs of urethral obstruction caused by calculi vary with the stage of the disorder.[6,10,15] Signs prior to bladder rupture include colic, straining stance to urinate (Figure 18.4),

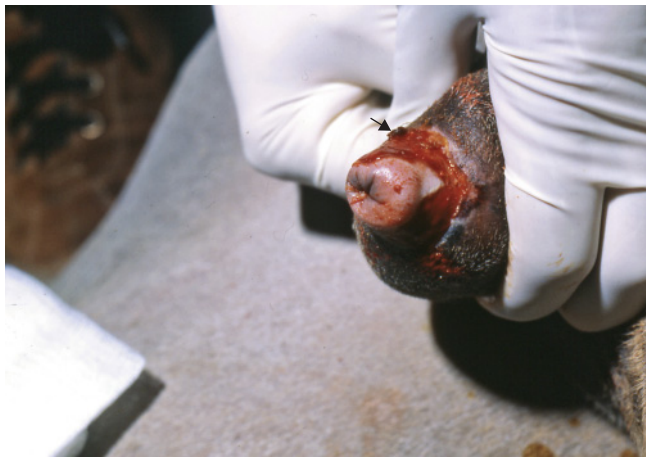

Figure 18.3. Posthitis in a llama.

Table 18.4. Composition of urinary calculi recovered from llamas at the Veterinary Medical Teaching Hospital, University of California.

Silicon dioxide (SiO_2)	Crystobalite
Magnesium ammonium phosphate ($MgNH_4PO_4 \cdot 6H_2O$)	Struvite
Basic calcium phosphate ($Ca_5(PO_4)_3(OH)$)	Apatite
Calcium carbonate ($CaCO_3$)	Carbonate
Uric acid ($C_5H_4N_4O_3$)	Urate

Figure 18.4. Stance of a llama cria with urethral obstruction.

dribbling urine, blood-tinged urine, anuria, distended bladder, and possible pulsation of the urethra. Signs after bladder rupture are no colic, depression, anorexia, anuria, and uroperitoneum, with possible distention of the abdomen and uremia (muscular weakness, dehydration, dyspnea, tremors, uremic odor in the breath, tachycardia, recumbency, coma, death).[30]

Uroperitoneum may be caused by trauma when the bladder is distended or from rupture of the bladder following urethral obstruction. In the llama, the immediate response to urine flushing into the abdominal cavity is excruciating pain. Llamas become frenzied and thrash about violently. This initial pain subsides, and the pain associated with a distended bladder disappears. Urine in the abdomen may arise from a single or multiple tears in the bladder wall but also from seepage through the stretched-thin bladder wall. A ruptured urethra has been reported in camels.[3,11] A ureter may also rupture, but such a condition has not been diagnosed in a camelid.

Diagnosis

Diagnosis is based on assessment of clinical signs, pertinent history, and special diagnostic tests. A differential diagnosis should include any disease condition in which colic is a sign. The major hematologic and serum chemistry changes found in camelids with obstructive urolithiasis include hemoconcentration, elevated blood urea nitrogen, hypophosphatemia, hypercalcemia, hypermagnesemia, hyperkalemia, hypercreatininemia, and hypochloremia.[27,32]

It is not always easy to identify the source of fluid in the abdominal cavity. Urine should have the odor of ammonia, but it may be necessary to heat the fluid to concentrate the odor before it becomes perceptible. The ability of individuals to smell ammonia varies. Abdominal fluid caused by urine has a creatinine concentration of >15 mg/dl (normal serum <3 mg/dl). Urine potassium is >185 mmol/L (normal serum <5 mmol/L).

Urethral catheterization and double-contrast radiography are routinely used to diagnosis urethral calculosis in carnivores. It is impossible to pass a catheter in a camelid male because of the dorsal urethral recess and restrictive diameter of the urethra. The value of radiography and ultrasonography has been less than satisfactory. Uroliths that don't contain calcium may be radiolucent, especially when surrounded by an inflammatory reaction.

The narrow urethra may predispose the accumulation of organic debris. A llama developed a swelling of the prepucial area that was treated initially as a trauma and later as a cellulitis. At necropsy, the urethra had ruptured proximally to an obstruction at the glans penis caused by a plug of nonmineralized organic material. The urethra may also become obstructed by external pressure on the urethra caused by a hematoma of the corpus cavernosum urethra following penile trauma.

Urinary tract tumors are rare, but a urethral sarcoma[12] and a renal teratoma[26] have been reported.

Treatment

In most species, urethral obstruction caused by uroliths is more common in castrated males, but unfortunately, in llamas, breeding males are most frequently the victims. The first task is to relieve the obstruction and to restore urine flow if the bladder has not already ruptured. Traditional therapy for feedlot steers is to perform a urethrostomy and/or penile amputation just ventral to the ischium and bring the penile stump out through the incision and suture it in place. In breeding male llamas an attempt is made to localize the obstruction and perform a urethrostomy over the urolith.[30,33] Unfortunately, the prognosis for return to breeding condition is poor to unfavorable. Dietary management in herbivores is not highly successful.

Potential sequelae of urolithiasis include ruptured bladder,[2] ruptured urethra, urethral stricture caused by trauma from the calculus, urethral stricture caused by scarring following surgical manipulation, chronic cystitis, and recurrence of calculi.

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19

Organs of Special Sense

EYE

Anatomy[33]

The basic anatomy of the eye is similar to that of other mammals (Figure 19.1). Camelids have keen vision.[9,12,27] Large, expressive eyes, with long eyelashes, are set at the sides of the skull, providing excellent peripheral vision with a broad field of view (Figure 19.2). Binocular vision is limited. The pupil is horizontally oblong, and this shape is accentuated by the corpora nigra located on the dorsal and ventral borders of the iris (Figure 19.3). The cornea and lens resemble those of other ungulates. Vascularity of the chorioretina is pronounced and similar to that of the bovine. The vessels converge on the optic disc, which is located in the medial ventral position. Both rods and cones are present in the retina, so presumably camelids have color vision. There is no fovea. Camelids are diurnal and have no need for a tapetum to intensify light. The red eyeshine seen under intense light at night, or from a photoflash, is a reflection of the fundus. Although llamas are not usually active at night, packers who have found themselves on the trail at night know that llamas remain agile and have night vision equal to theirs. Distant vision is probably as acute as that of any artiodactylid that depends on movement perception at some distance to detect stalking predators.

The aqueous humor has been studied in SACs and was found to be similar to that of other ungulates.[2] The normal flora of camels has been reported.[20]

The nasolacrimal duct originates as paired canaliculi connecting the punctae on the upper and lower eyelids; there is approximately 5 mm from the medial commissure to the lacrimal sac (Figure 19.4). The duct traverses the osseous lacrimal canal within the lacrimal bone for a distance of 2 cm in close association with the osseous canal of the infraorbital nerve (Figure 19.5), then exits the bone and continues submucosally within the nasal cavity to exit within the nares near the mucocutaneous junction, approximately 1 cm dorsal from the floor of the nostril (Figure 13.1).[7,22,30] The orifice is approximately 2 to 3 mm in diameter.

Ophthalmic Diagnostic Procedures

Ophthalmic examinations of camelids are carried out in a similar manner to those performed on other domestic large animals.[12,15,27,32,37] Reflexes, such as pupillary and palpebral, are similar. Reflection of the conjunctival mucous membranes is more difficult than in the horse but easier than in the cow.

Diseases[11,12,15,18,32]

Infectious diseases involving the eye are discussed in Chapter 7 and include aspergillosis, moraxellosis,[6] and equine herpesvirus type 1.[28] The normal flora of the conjunctival sac has been studied, and numerous species of opportunistic bacteria have been isolated; however, mycoplasmas have not been isolated.[13,14] Fungal keratitis has been described and should be considered in keratitis that is nonresponsive to antibiotic therapy. Panophthalmitis may be seen in septicemias.

Parasitic diseases are discussed in Chapter 8 and include those caused by *Toxoplasma gondii*, *Thelazia californiensis* (Figure 19.6) and various species of flies that are attracted to lacrimal secretions. Congenital eye anomalies are discussed in Chapter 22 and include agenesis of the eyelids, microphthalmia, cataracts,[3] blindness, entropion, ectropion, dermoids, and cyclopian.

Noninfectious ophthalmic conditions include trauma to the eyelids or cornea (laceration, abrasion,

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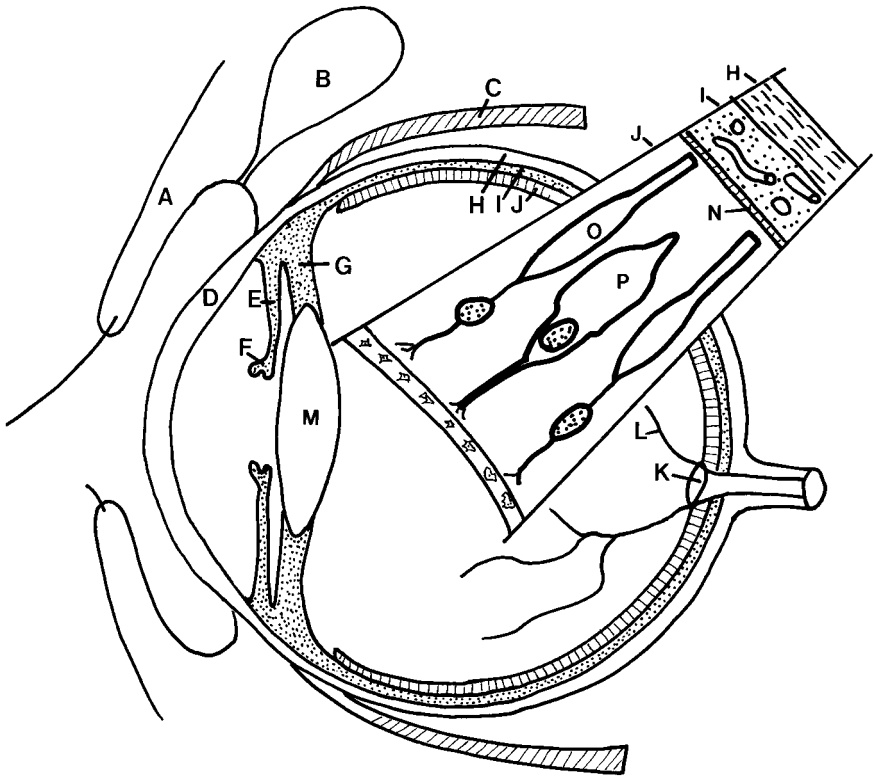

Figure 19.1. Diagram of a longitudinal section through the eye of a camelid. (A) Upper eyelid, (B) lacrimal gland, (C) ocular muscle, (D) cornea, (E) iris, (F) corpus nigrum, (G) ciliary body, (H) scleral layer of the bulb, (I) chorioid layer of the bulb, (J) retinal layer of the bulb, (K) optic disc, (L) retinal vessels, (M) lens, (O) cone, (P) rod.

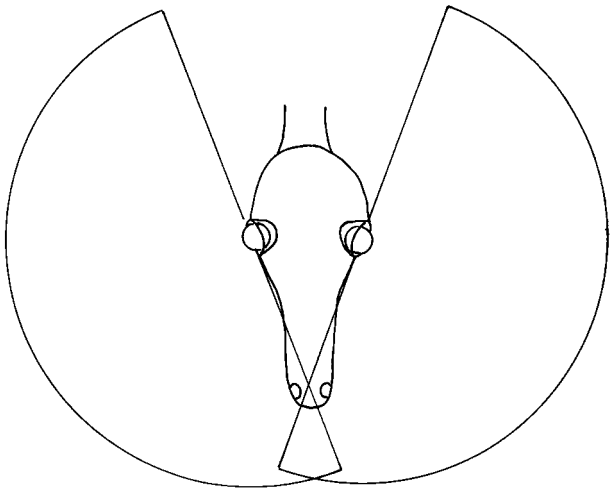

Figure 19.2. Field of view of a camelid.

ulceration,[23,38] foreign body penetration), intraocular hemorrhage, tumors,[3] retinal detachment, and acquired cataracts.[16,17] Recurrent uveitis is frequently seen in immunosuppressed llamas and is considered a grave sign. Uveitis is responsive to standard therapy, but the underlying condition may be refractory to treatment. Enrofloxacin-induced retinopathy has been reported.[21] Corneal epithelial inclusion cysts have been reported in llamas.[28]

Obstruction of the nasolacrimal duct has been diagnosed.[31] The etiology may be a temporary plug of mucus or swelling of the duct from inflammation. Prolonged inflammation may result in fibrosis and permanent occlusion of the duct. Clinical signs are epiphora, with or without concomitant conjunctivitis. The nasolacrimal duct may be cannulated with a 1-mm catheter at the nasal orifice or with a 22-gauge lacrimal irrigation needle at the eyelid.[26] The duct may then be flushed with normal saline to establish patency. Alternatively, a fluorescein dye may be instilled into the eye and observations made to see if and when the dye appears at the nasal orifice.

Congenital conditions should be considered including micro-ophthalmia (Figure 19.7), blindness (Figures 19.8A and B), eyelid defects (Figure 19.9) and congenital agenesis of the nasolacrimal duct. Exophthalmia is reported, but a cautionary note should be made. Male llamas that have been in an altercation with another male may appear to evert the lower eyelids, giving the impression of exophthalmia. This is actually a part of the pouting syndrome caused by constriction of the facial muscles that puts tension on the lower eyelid which in turn exposes the conjuctiva (Figure 19.10).

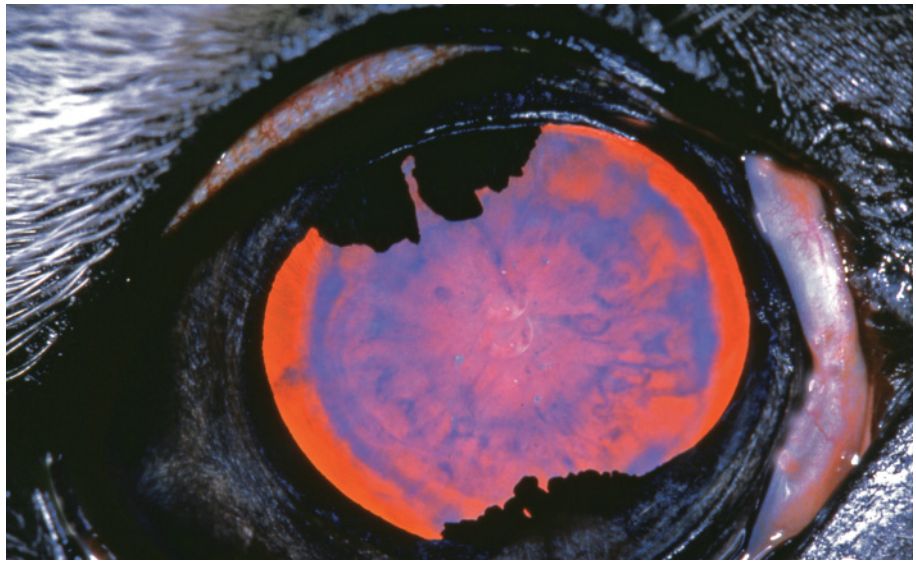

Figure 19.3. Granula iridica (corpora nigrum) of a llama.

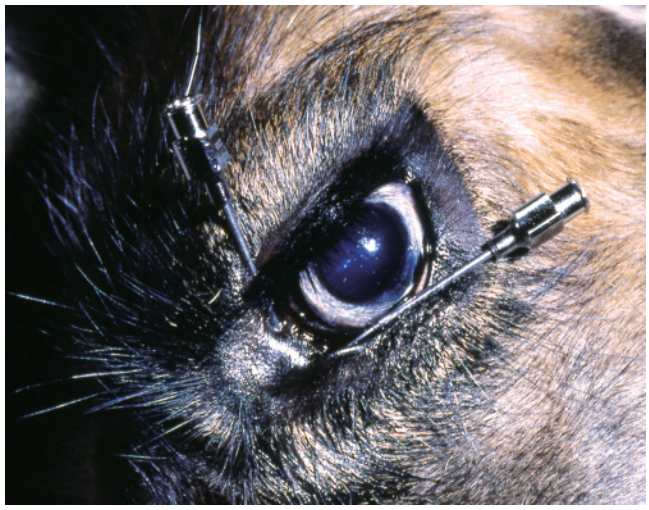

Figure 19.4. Cannulae in the nasolacrimal ducts.

Apigmentation of the iris (glass eye, blue eye, watch eye), deserves special mention (Figures 19.11A and B). Eye color is the result of varying amounts of melanin in the iris. Blue eye color is caused by lack of pigment in the iris and is the result of refraction of light through the iris, similar to our perception of a blue sky. As in all species, eye color is hereditary. Owner perception of the desirability of blue eyes varies. Esthetics seem to be the major concern for many. South American veterinarians feel that blue eye coloration predisposes camelids to snow blindness and that such animals suffer from the intense ultraviolet light at high altitudes. None of these concerns have been validated by scientific studies. However, in recent years, a more serious ramification has been suggested. Lack of pigment in the iris (blue eye, glass eye) is associated with deafness in white cats, white rabbits, Dalmatian dogs, and Australian shepherd dogs with merle coloration. The same type of deafness has been recognized in white llamas and alpacas. However, not all white, blue-eyed llamas and alpacas are deaf, nor are colored animals with blue eyes.

The deafness is a degenerative response in the inner ear, not a dysgenesis. The basic lesion is a deficit in endolymph production, predisposing the vital structures to excessive vibrations.

Deaf llamas and alpacas compensate for lack of auditory sense by increased visual acuity and tactile sensation, and by sensing herd dynamics. It may be difficult to assess deafness. Subjective evaluation by clapping the hands or shouting outside the animal's visual field is used. The definitive determination of deafness is by conducting a brain stem auditory evoked response (BAER). BAER[1,36] requires general anesthesia and placement of appropriate electrodes. The wave form generated corresponds to particular anatomic structures within the ear. This test cannot be performed on immature animals. BAER is the only means of definitively diagnosing unilateral deafness.

Albinism has not yet been reported, but it is seen in many species of mammals, birds, and reptiles and, in time, is likely to be seen in camelids. In humans, the trait is a simple recessive.

Bilateral liquid keratopathy may be seen in hepatic lipidosis.[29]

The diagnosis and management of ophthalmic conditions are similar to the procedures employed when dealing with similar problems in an equine or bovine eye. Treatment should be begun immediately and carried out intensively to avoid loss of vision and, perhaps, loss of an eye.[5,26,37]

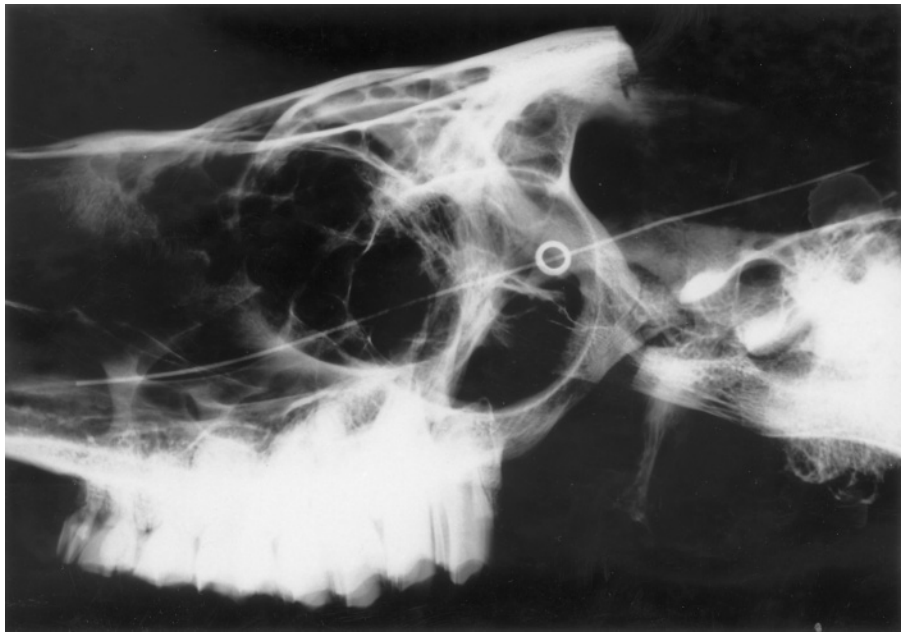

Figure 19.5. Lateral radiograph of a radiopaque cannula in the nasolacrimal duct illustrating the relationship to the roots of the maxillary cheek teeth.

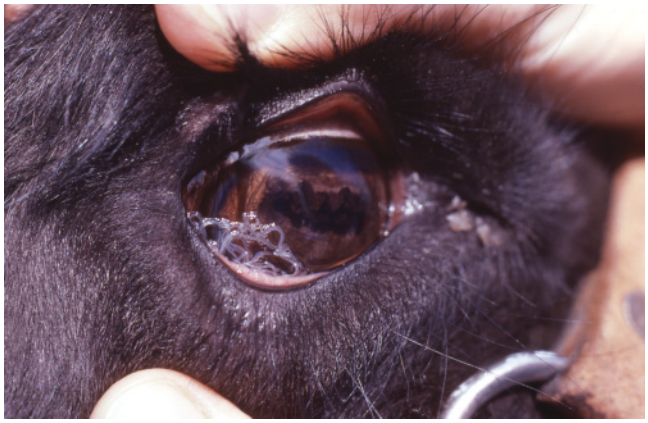

Figure 19.6. *Thelazia* sp. in the conjunctival sac.

Figure 19.7. Micro-ophthalmia in a llama cria.

A method that has been found useful in treating llamas with ocular conditions requiring intensive, repeated medication is insertion of an indwelling catheter into the nasal orifice of the nasolacrimal duct. Insert the catheter gently until the llama blinks, indicating the cornea or conjunctiva has been touched. Then withdraw the catheter 1 to 2 cm. Anchor the catheter in place by affixing tape to the catheter and suturing the tape to the skin near the nostril. The eyelids are then sutured closed to protect the cornea from light and repeated movement of the conjunctiva over the cornea. Medication can then be instilled into the conjunctival sac, as required, without stressing the llama.[26] A word of caution about treating camelid ophthalmic conditions with atropine. Camelids are supersensitive to atropine instilled intraconjunctivally. Pupillary dilatation may last for days or even weeks following a single instillation. Be certain to restrict a camelid treated with atropine to a darkened

Figure 19.8A. Blindness in a llama cria.

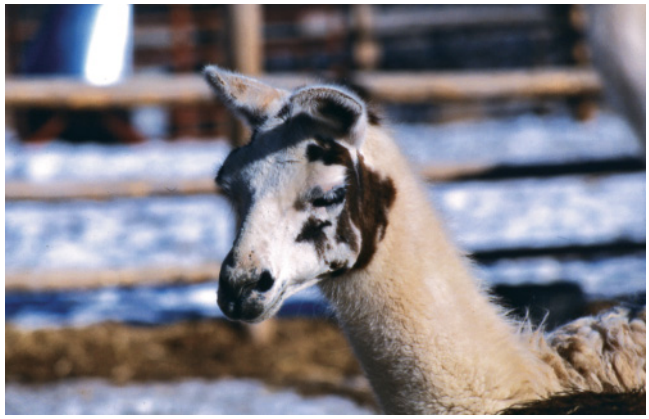

Figure 19.8B. Blindness in an adult llama.

Figure 19.9. Eyelid defect in a llama.

Figure 19.10. Exposed conjunctiva caused by pouting.

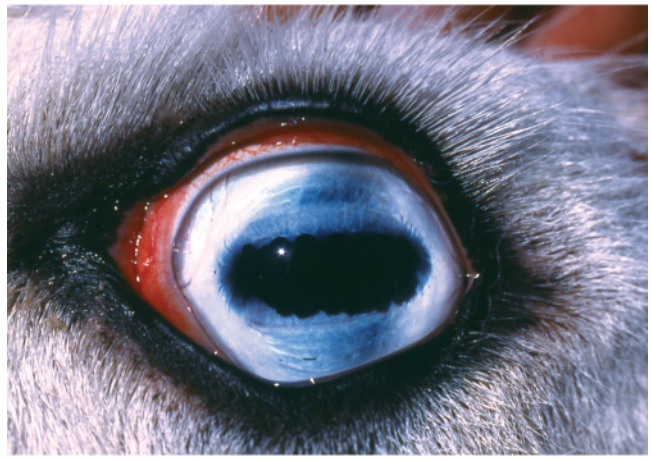

Figure 19.11A. Apigmentation (blue eye) of the iris.

Figure 19.11B. Blue eye in a llama cria.

Figure 19.12A. Pinnae of a llama, banana ears.

Figure 19.12B. Pinnae of a llama, straight ears.

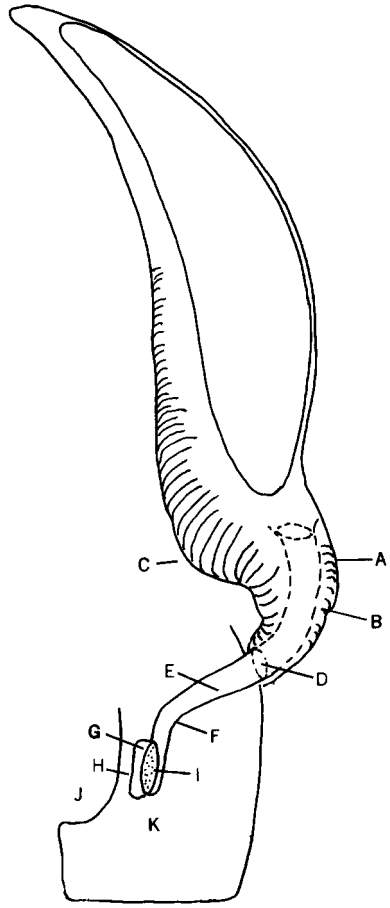

Figure 19.13. Diagram of the relationship of the pinna, external ear canal, tympanic membrane, and middle ear. (A) Lateral side of the left pinna, (B) annular cartilage of the pinna, (C) conchal eminence, (D) external orifice of (E) osseous ear canal, (F) bend of canal, (G) middle ear, (H) inner ear, (I) tympanic membrane, (J) cranial cavity, (K) tympanic bulla.

environment, and avoid remedication as long as the pupil remains dilated.

It is beyond the scope of this book to delve into the intricacies of diagnosis and therapy of ocular diseases.[5] Detailed discussions of cattle ocular diseases are found in Whitley and Moore.[36]

EAR

Anatomy[7,8,22]

The external pinna is elongated and highly mobile (Figures 19.12A and B). The ears are expressive of health and emotional state. The auditory canal is located on the lateral aspect of the annular cartilage of the pinna. When inserting an otoscope cone, it is easy to mistake the conchal eminence for the ear canal.

The osseous ear canal enters the petrous temporal bone immediately ventral to the zygomatic process of the temporal bone.[8] In the adult, the canal is directed slightly rostrad and ventrad. At a depth of 1.8 cm, the canal bends ventrally at an angle of approximately 120 in a percentage of llamas (Figure 19.13) and continues on for a distance of 1 cm. The irregularly shaped tympanic membrane is located on the medial side of the distal canal and is approximately 0.8 cm in diameter.

The canal is essentially straight in a neonate. Apparently, the external auditory canal is partially composed of cartilage in the neonate and ossifies at an unknown later time. The osseous ear canal is extremely narrow in the llama. The inside diameter of the external orifice, measured from the skeletal preparation of a large male, was 6 mm, narrowing to 4 mm at the bend. Addition of the epithelial lining further restricts canal size.

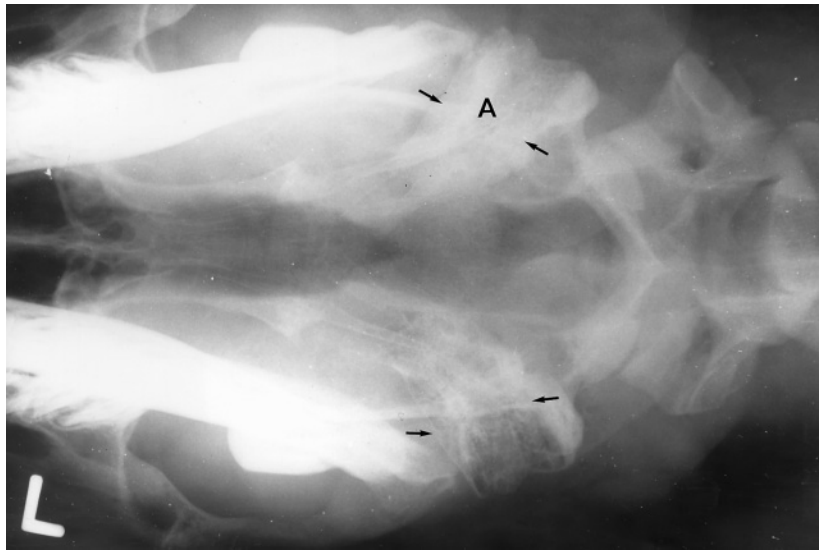

Figure 19.14. Dorsoventral radiograph of the skull with sclerosis of the tympanic bulla. (A) Affected bulla.

It is not possible to view the tympanic membrane in approximately 50% of adult llamas because of the bend and narrowness of the external ear canal. Edema and inflammation of the canal also obstruct viewing. The tympanic bulla is large and extensively honeycombed, but there is no fundic cavity (Figure 19.14). The inner ear lies between the middle ear and the cranial cavity.

Diagnostic Procedures[4]

An unsedated camelid is reluctant to allow even a casual examination of the ear. Deep examination of the canal is virtually impossible without sedation. If exudate oozes from the ear, it is important to exclude the presence of foreign bodies (grass awns) or ticks. Radiographic examination demonstrates sclerosis of the tympanic bulla of the petrous temporal bones (Figure 19.14). Clinical evaluation of cranial nerve deficits in camelids is the same as for other mammals.[11]

Diseases

Lacerations

Lacerations of the pinna are common in adult males because of inter-male aggression. If the event causing the laceration is observed and immediate attention can be given, such wounds may be cleansed, debrided, and sutured with reasonable success. Lacerations of the poll may transect the ear muscles, preventing motor control of the pinna. Every effort should be made to realign severed ends of the muscles of the pinna.

Other Diseases of the Pinna

Dermatophytes may infect the pinna. Sarcoptic and psoroptic mange and demodectic mites have been detected on camelid pinnae (without causing a dermatitis). Hematoma of the pinnae is not common but may be seen in association with head shaking in ear tick infestation. A blow to the ear may cause a similar lesion.

Otitis Externa

Etiology. Inflammatory lesions of the external ear canal are common, but the cause of the inflammation is not always readily apparent. The spinose ear tick, *Otobius megnini*, may be a regional problem (Chapter 8). Grass awns may lodge in the canal and scarify the epithelium, providing a portal of entry for opportunistic pathogens.

Clinical Signs. The signs of otitis externa are head shaking, scratching the ear with a hind foot or against a post or barn, head tilt, peculiar positioning of the pinna, and exudation from the external ear canal.

Diagnosis. Diagnosis is based on clinical signs and a thorough physical examination of the outer ear canal following cleansing of the ear and removal of all debris and ear wax. The epithelial surface may be hyperemic and ulcerated. Cultured samples taken from the external ear canal are not likely to be helpful, since the normal flora may harbor many opportunistic pathogens.

Treatment. The external ear canal should be cleansed and irrigated with povidone-iodine solution twice daily, followed by instillation of a broad-spectrum antibiotic ointment.

Otitis Media and Interna

Etiology. Infection of deeper structures is usually caused by a direct extension of infection in the external

ear canal.[24,25,35] Also, grass awns may penetrate the tympanic membrane and initiate infection. *Listeria monocytogenes* was isolated from an otitis interna infection that subsequently spread to the brain.[35]

Clinical Signs. Otitis media is usually associated with facial nerve paralysis (Figures 19.15, 19.16) and Horner's syndrome (Chapter 20).[8] The cardinal signs of Horner's syndrome are slight ptosis of the upper eyelid, inability of the pupil on the affected side to dilate in subdued light, and the nictitating membrane pushing out over the bulb as a result of retraction of the bulb deeper into the orbit, which makes the eye appear to be smaller (microphthalmia). This syndrome, plus facial paralysis, is virtually pathognomonic evidence of a lesion in the middle ear because the sympathetic fibers involved in Horner's syndrome lie in juxtaposition to the facial nerve only near the middle ear.

Additional signs may include disorientation, ataxia, and circling.

Figure 19.15. Facial paralysis.

Figure 19.16. Chronic facial paralysis.

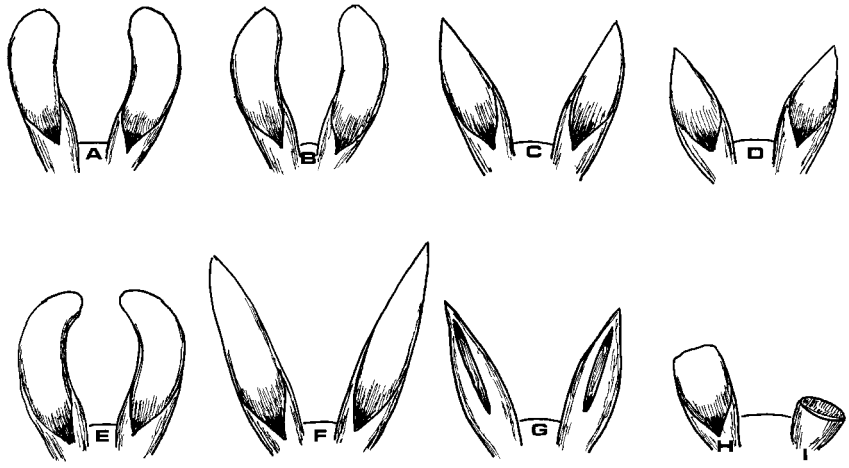

Figure 19.17. Diagrams of llama ear shapes. (A) Normal banana ear, (B) sharp-pointed banana ears, (C) straight medial border, (D) short ears, (E) exaggerated banana ears, (F) elongated, spear-shaped ears, (G) curled ears, (H) congenital shortened ear, (I) congenital gopher ear.

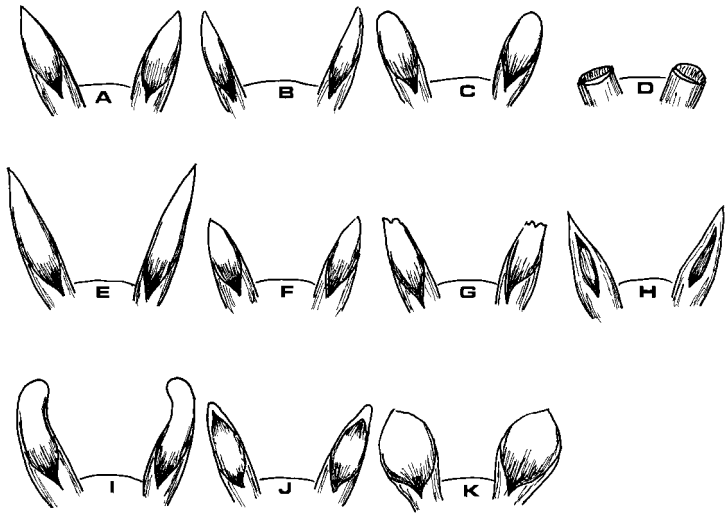

Figure 19.18. Diagrams of alpaca ear shapes. (A) Normal spear-shaped ears, (B) straight medial border, (C) round-tipped ears, (D) congenital gopher ears, (E) elongated ears, (F) short spear-shaped ears, (G) congenital-tip anomaly (multiple configurations), (H) curled ears, (I) banana-shaped (llama ears), (J) tented tip, (K) cauliflower ears.

Figure 19.19. Gopher ear.

Figure 19.20A. Pinna defect, shortened and notched.

Miscellaneous Conditions

The shape of the ears is a hereditary trait. Figures 19.17 and 19.18 illustrate some ear pinna shapes. A novel device has been applied to the measurement of length and angulation of the ears.[2] Congenital diseases of the ear include agenesis of the pinna (gopher ear, Figure 19.19), shortened pinnae, and curled ear (tented, Figures 19.20A to C). Bent ears (floppy pinna, airplane ears, helicopter ears) may be congenital or acquired (Chapter 22). In the neonate, a temporary floppy pinna may be the result of premature birth. This condition corrects itself as the cartilage strengthens with growth. It may be desirable to support the floppy ear with a partial roll of gauze bandage on the concave side and the pinna wrapped to the roll.

The most common noninfectious, nonparasitic disease is frostbite, Figure 19.21 (Chapter 9). Tumors have been reported, including sebaceous carcinoma of the eyelid,[1] retinoblasoma,[10] and tricoepithelioma.[34]

Figure 19.20B. Pinna defect, curled tips.

Figure 19.20C. Pinna defect, fused tip (tented).

Figure 19.21. Frostbite ears.

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20

Nervous System

ANATOMY

There are no published comprehensive treatises on the anatomy of the nervous system of any of the camelids. A few references can be found in the periodic literature, and anatomic subjects are popular as theses for students.[3,9,14,15,17,19,21–23,25,30,32–34,35,38,40] Unfortunately, South American theses are not readily accessible. Most papers deal with the specific nerves of a limited region of the body or of the brain.

The general morphology of the brains of camelids does not differ from that of other domestic ungulates, nor does the basic distribution of peripheral nerves.[22,36] The locations of nerves that are important in surgical procedures have been discussed in the appropriate sections of this book. The spinal cord terminates at the midsacral region (Figure 20.1).

DIAGNOSIS

Clinical diagnosis of neural function should follow the same pattern as for other domestic animals.[6,18] The purpose of a neurologic examination is to establish the presence and location of a neural lesion(s), and diagnostic procedures are used to determine the cause of the lesion(s). The neurologic examination is similar to that for cattle and sheep, although camelids tend to respond more slowly to many of the stimuli. Special procedures include radiography, fluoroscopy, and myelography for evaluation of compression of the spinal cord or brain. Cerebrospinal fluid may be obtained from the occipitoatlantal space or the lumbosacral space (Chapter 4).[24,41]

All of the new, sophisticated techniques for evaluating neural function are applicable to and have been performed on camelids. Computerized tomography, muscle and nerve biopsies, and the various electrodiagnostic procedures (electromyography, nerve conduction velocities, evoked potentials) are particularly useful after normal values have been established.

Cerebrospinal fluid analysis is also an important diagnostic procedure.[20] For the technique of obtaining cerebrospinal fluid, see Chapter 4.

DISEASES

Congenital Diseases

Hydrocephalus is the most frequently seen congenital deformity of the nervous system. The cause is unknown. A diagnosed internal hydrocephalus (dilatation of the lateral ventricles) in a llama was reported to be caused by partial stenosis of the cerebral aqueduct.[7] Clinical signs included inability to stand, constant adduction of the right front leg, wry neck to the left, erect ears pulled to the left, and a domed forehead. At necropsy, bilaterally symmetric dilatation of the lateral ventricles of the cerebrum was seen. The brain seemed to expand upon removal of the calvarium. A large cardiac ventricular septal defect was also noted.[7]

A definitive diagnosis of hydrocephalus is not easily made on the basis of clinical signs unless there is gross enlargement of the head. Many normal llamas and especially alpaca neonates have a slightly domed forehead. Doming may become exaggerated when deformities of the facial bones occur. Plain film radiography may be of little assistance in diagnosis. Computerized tomography is the quickest and most accurate method for diagnosing hydrocephalus. Another condition seen in our clinic is cerebral meningocele (Figure 20.2).

Congenital disorders affecting the spinal cord may be primary, such as meningocele or myelomeningocele associated with spina bifida, or the spinal cord may be affected secondarily by compression from vertebral anomalies such as hemivertebrae (Chapter 22), or

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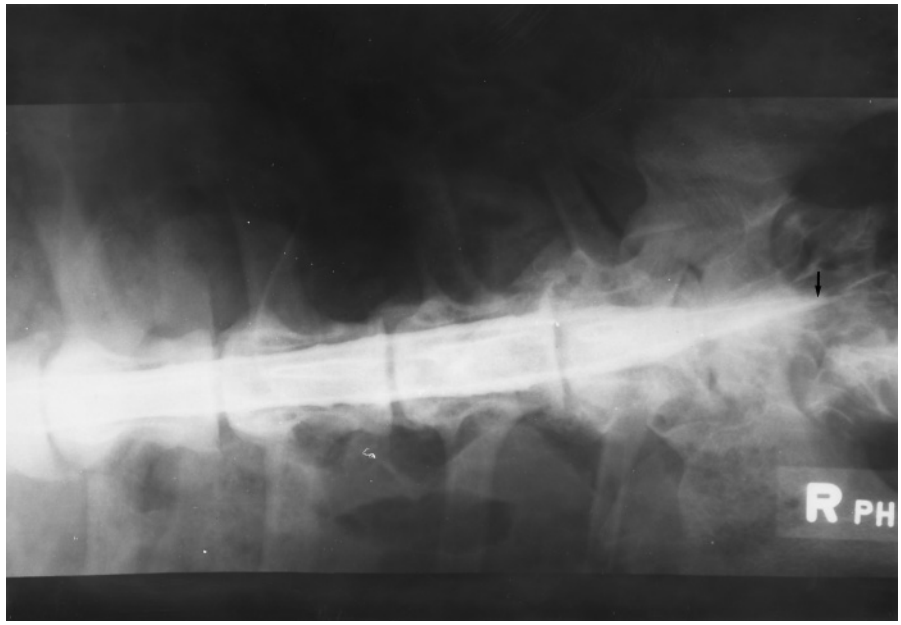

Figure 20.1. Dorsoventral radiograph of the terminal spinal cord with contrast media in the subarachnoid space. The meninges terminate at the midsacral region.

Figure 20.2. Cerebral meningocele in a llama cria.

Figure 20.3A. Agenesis of the spine and spinal cord.

complete agenesis of the spine and spinal cord (Figures 20.3A and B).

Infectious Diseases

Infectious diseases involving the central nervous system are discussed in Chapter 7. These include rabies, tetanus (Figure 20.4), facial paralysis from middle ear infection, neural abscesses, and Borna disease.[1]

Parasitic Diseases

Parasitic diseases involving the nervous system include tick paralysis,[5] toxoplasmosis, sarcocystosis, and meningeal worm (Figure 20.5) (Chapter 8).

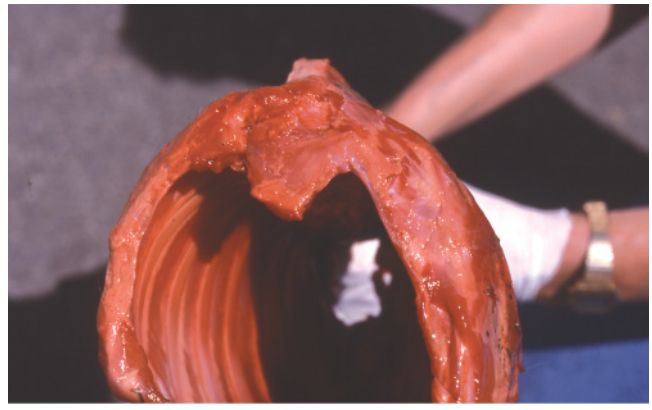

Figure 20.3B. Agenesis of the spine and spinal cord.

Figure 20.4. Tetanus in a llama.

Figure 20.5. Ataxia caused by meningeal worm infestation.

Noninfectious Diseases

Spinal cord neuronal degeneration causing ataxia, paresis, and paralysis has been reported in llamas.[26,27–29] Similar degeneration has been diagnosed in the author's clinic. Although a definitive etiologic diagnosis has not yet been determined, copper deficiency is suspected (Chapter 2). Polioencephalomalacia has been reported.[23] Neoplasia is rare, but an astrocytoma has been diagnosed.[13]

Figure 20.6. Postsurgical radial nerve paralysis.

Trauma

Concussion and contusion of the brain have resulted from kicks by horses and from running into solid objects when agitated or frightened. Laceration of peripheral nerves may occur with laceration of the skin and muscles or internally from sharp fragments of bone fractures. Nerves may be incised or contused during surgical procedures. One llama lost motor control of the lower lip following trephine removal of a cheek tooth. Temporary loss of motor or sensory function may occur from direct contusion of a nerve or from pressure on a nerve from an edematous or inflammatory lesion.

Partial radial paralysis may be a sequel to prolonged lateral recumbency during anesthesia if the forelimb is not pulled forward properly or is not padded (Figure 20.6). Hot packs over the humerus may hasten reduction of the contusion, and the lower limb should be padded or wrapped to prevent abrasion of the fetlock during recuperation.

Injury to the spinal cord usually follows vertebral fractures or luxations. The cervical cord of one male llama was transected by a fracture of cervical vertebra 2. The llama was attempting to return to a group of females from which he had been separated and ran headlong into a fence. For a discussion of other vertebral fractures, see Chapters 6 and 11.

Llama Downer Syndrome

Ailing llamas may be reluctant to rise from sternal recumbency. In many instances, the animal may be physically unable to rise. It is difficult to establish whether the nervous system, muscular system, skeletal system, or a metabolic disorder is the cause. Ketosis and postparturient hypocalcemia have been reported in camelids but are rare. Electrolyte imbalance or uremia may contribute to malaise and depression.

Prolonged recumbency in a camelid is hazardous because covering the ventral abdomen impairs thermoregulation. A moderate fever frequently accompanies the downer syndrome.

There may be many ultimate causes for the downer syndrome. A thorough physical examination is necessary to exclude the obvious, but some cases defy diagnosis and therapy.

Cranial Nerve Dysfunction

Published clinical reports of cranial nerve (CN) dysfunction in camelids have involved only a few of the twelve CNs.[11,31] Space does not permit nor is sufficient information available to provide a detailed discussion of CN dysfunction. An abbreviated review is provided.

Etiology. Cranial nerve dysfunction may arise within the brain from meningoencephalitis (listeriosis, toxoplasmosis, rabies), toxins (botulism), neoplasia, contusion, or concussion. Peripheral CNs may be traumatized from basilar skull fractures and inflammatory lesions in the areas traversed by the nerves. Facial nerve paralysis is a prime example of this. A llama developed multiple CN dysfunction (CN V-XII) following an extensive mycotic infection of the caudal nasal cavity and nasopharynx.

A blow to the throat may result in fracture of the hyoid apparatus and subsequent hypoglossal nerve paralysis. Excessive tension on the tongue during endotracheal intubation or dental surgery may cause temporary paralysis of the tongue as a result of stretching of the hypoglossal nerve. Other causes of CN dysfunction include any space-occupying lesion (hematoma, abscess, tumor) in the vicinity of a nerve.

Clinical Signs Associated with Individual Nerves.[8]

CN I (olfactory): loss of sense of smell.

CN II (optic): blindness. In llamas and alpacas optic nerve degeneration and retinal degeneration have followed infection with equine herpes type I virus.

CN III (oculomotor): this nerve, as well as CNs IV and VI, innervates the muscles of the globe. Various eye position abnormalities occur, depending on the species. Nothing is known of the specific positional problems associated with paralysis of CNs III, IV, and VI in camelids.

CN IV (trochlear): see CN III.

CN V (trigeminal): dropped jaw that cannot be closed, difficult prehension, food in the oral cavity.

CN VI (abducent): see CN III.

CN VII (facial): see detailed discussion in Chapter 19.

CN VIII (vestibulocochlear, acoustic): loss of hearing and equilibrium.

CN IX (glossopharyngeal): paralysis of soft palate, pharynx, and cervical esophagus.

CN X (vagus): laryngeal muscle paralysis and, potentially, vague autonomic dysfunction due to loss of parasympathetic input to the viscera, particularly with bilateral vagal lesions.

CN XI (spinal accessory): paralysis of portions of the brachiocephalicus, sternocephalicus, and trapezius muscles.

CN XII (hypoglossal): paralysis of muscles of the tongue. Interferes with deglutition, prehension, and mastication.

Diagnosis. Diagnosis requires detailed neurologic evaluation.

Treatment. The primary cause must be eliminated, if possible. Anti-inflammatory agents, antimicrobials, and supportive treatment are indicated. Prognosis for recovery of cranial nerve function depends on the cause and severity of the lesion as well as prompt diagnosis and appropriate therapy.

Facial Nerve Paralysis[12]

Etiology. The clinical syndrome associated with facial nerve paralysis in llamas is known, and a detailed discussion follows. The facial nucleus may be damaged by intracranial lesions as already described, but more often, the nerve becomes involved in an extension of otitis media and interna as the facial nerve passes through the facial canal in the petrosal bone and emerges through the stylomastoid foramen.[6] Individual branches of the nerve may be traumatized by blows to the face, space-occupying lesions (hematoma, abscess,[37] tumor), lacerations (bites, surgical incisions), edema or cellulitis from snake bite, direct trauma from prolonged pulling against a halter (training and trailering accidents), or lying in lateral recumbency on a surgery table without adequate padding of the head. Trauma to the zygomatic arch may involve only the auriculopalpebral branch of the nerve, in which case only the ear and eyelid muscles are involved.

Clinical Signs. The facial nerve supplies motor function to all of the muscles of facial expression (ear, eyelid, nose, cheek, lip, and caudal portion of the digastric muscle).[8] Loss of function results in unilateral or bilateral ear drooping and inability to position the ear (Figure 19.16). This is a dramatic sign in llamas, because the ears are so expressive. In unilateral dysfunction, the nose is pulled toward the unaffected side (Figure 19.16). In cases of chronic facial paralysis, the paralyzed muscles atrophy. This may result in the nose being pulled back toward the affected side, complicating a differential diagnosis (Figure 19.17). The lips on the affected side may droop, allowing saliva to drip. Feed may also fall from the lips because the llama cannot move feed into the teeth for chewing and sub-

sequent swallowing. The nostril on the affected side fails to dilate symmetrically with the opposite nostril upon inspiration.

Paresis of eyelid muscles results in failure of closure of the palpebral fissure and excessive drying of the cornea, progressing to corneal ulceration. In one such case, it eventually became necessary to enucleate the eye.

Head shaking or scratching at the ear with a hind foot may be seen. An exudate may be present in the affected ear, but not always.

Diagnosis. Diagnosis is usually based on clinical signs. The external ear canal should be examined for exudation and foreign bodies. Sedation is usually required for a thorough examination. Sclerosis of the tympanic bulla may be evident on a radiograph (Figure 19.15).

Treatment. In cases of trauma, administration of anti-inflammatory agents is indicated along with hot packs to restore circulation and encourage drainage of edema. Space-occupying lesions should be dealt with as appropriate. Culture and sensitivity tests should be done to aid in appropriate antibiotic therapy.

Otitis media is treated with broad-spectrum antibiotics (gentamicin sulfate 1 mg/kg three times daily) and irrigation of the external ear canal with dilute povidone-iodine solution.

Polioencephalomalacia[8,10]

Etiology. Polioencephalomalacia (PEM, cerebrocortical necrosis, polio, sleeper, brainer) is a softening of areas in the cerebral cortex. There may be several causes including altered thiamine metabolism, excessive ingestion of sulfur, water deprivation, salt poisoning, amprolium-induced necrosis in a camel,[42] lead, and selenium toxicosis. The condition is seen in both SACs and camels.

Signs. Signs are variable depending on the area of the cerebral cortex affected and may include blindness, dullness, head pressing, anorexia, muscle tremors, opisthotonos, recumbency, trismus, salivation, nystagmus, and clonic convulsions. The prognosis for acute PEM is unfavorable, but subacute and chronic cases may respond to therapy.

Therapy. Administer thiamine at a dosage of 10 to 20 mg/kg either IM or SC for three to seven days. Dexamethasone 1 to 2 mg/kg may be beneficial in reducing cerebral edema.

Vertebral Osteomyelitis (Diskospondylitis)[6]

Vertebral osteomyelitis is an inflammation of the vertebral bodies and associated intervertebral disks.

Etiology. Usually septic from a variety of organisms.

Signs. Signs include vertebral column pain, progressive paresis, and ataxia. No fever or hemogram alterations are present.

Diagnosis. Radiographs of the vertebral column at a suspected site reveal irregular bony proliferation in all directions, which may occlude the neural canal. Lytic lesions may be observed.

Therapy. Long-term antibiotic therapy is indicated based on a sensitivity test or the use of a broad-spectrum antibiotic.

Intervertebral Disk Extrusion (Slipped Disk)

Etiology. Trauma.

Signs. Hypertonia, hyperreflexia, paresis, ataxia, pain.

Diagnosis. Radiography, myelogram, CT scan.

Therapy. Conservative medical management.

Myelitis[2,8]

Etiology. The cause is unknown.

Signs. A nine-year-old male llama that had been used for breeding was noticed to be unable to copulate with a female. More careful observation revealed that he had rear limb weakness and ataxia. Extensive clinical and radiographic examination provided no clues as to the etiology. He acted as if he wanted to breed a receptive female but was reluctant to do so, and it appeared to observers that it was painful for him to go into sternal recumbency. Over a period of weeks he became recumbent and depressed, lost weight, and ultimately died.

Diagnosis. At necropsy, there were no gross findings that could explain the hind limb weakness and ataxia. Microscopic examination of the cord indicated a mild myelopathy consisting of Wallerian degeneration of the cervical, thoracic, and lumbar spinal cord. There was also mild neuronal necrosis in the lumbar segments of the cord. The sciatic nerve had mild Wallerian degeneration, but this was thought to be a result of the prolonged recumbency. There were elevated liver and kidney copper levels, the significance of which was not determined. No cause of death was determined.

Therapy. Analgesics failed to provide adequate relief from pain sufficiently for him to resume breeding activity. Acupuncture was performed without success.

MISCELLANEOUS CONDITIONS OF THE NERVOUS SYSTEM

Phrenic nerve degeneration resulted in diaphragmatic paralysis in a llama.[4] An intracranial teratoma is reported in an alpaca.[16] Cerebral injury has occurred as a result of accidental intracarotid injection.[39]

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21

Neonatology

Murray E. Fowler, DVM, and P. Walter Bravo, DVM, PhD

CHARACTERISTICS OF THE CAMELID NEONATE[1,2,4,14,32]

The Spanish word for a baby SAC is cria (birth to weaning), and it is the designation most often used for baby SACs by North American owners as well. The term is used in this book. A camel neonate is called a calf. While the following discussion is based on the SAC cria, the same general principles of care and conditions that may occur are the same in camels.

The body weight of newborn alpaca crias varies from 3.6 to 10.4 kg (8 to 23 lb), and llama crias vary from 8 to 20 kg (18 to 45 lb). Birth weight is determined by genetic factors, age and size of the mother, degree of maturity, and nutrition of the dam during gestation. The neonate is not likely to gain weight during the first three days of life; in fact, it may lose up to 0.5 kg (1 lb).[22,28] Greater weight loss than this may indicate dehydration or lack of milk intake. After the first few days, weight gain should be about 250 g (0.5 lb) per day for the first two weeks and 0.5 kg (1 lb) after that for llama crias, and 125 to 250 g (0.25 to 0.5 lb) for alpaca crias. Weight of the cria at birth is also influenced by age of the dam. The heaviest crias (8 to 9 kg) are born from dams that are between eight and ten years old. Dams between two and seven years have crias weighing between 7 and 8 kg. As nutrient conditions change in the Andes, dams older than eleven to twelve years old produce crias weighing less than 8 kg. These weights may be increased in other countries wherein health and nutrition has been greatly improved.

The neonate is usually covered by a thin, semitransparent epidermal membrane that is attached at the mucocutaneous junctions, at the coronets of the nails, and at the umbilicus (Figures 21.1, 21.2) (Chapter 17). The camelid fetus is not encased in the amniotic membrane in a normal birth. The eyelids are open, and most of the time the incisor teeth are erupted.

The body temperature of the recent newborn should be the same as that of the dam, 37.7°C to 38.9°C (100°F to 102°F) (Figure 21.3). The cria's rectal temperature drops to 35.5°C (95.9°F) during the first thirty to forty-five minutes after birth, then rises to the homeothermic stage (37.5°C, 99.5°F) Once the cria has stabilized to the ambient environment, body temperature may fluctuate more in a cria than in an adult and may rise to 39.2°C (102.5°F) normally. The heart rate varies from sixty to ninety beats per minute and the respiratory rate from ten to thirty breaths per minute. Camelids are obligate nasal breathers, so any upper respiratory obstruction causes dyspnea.

IMMEDIATE CARE OF THE NEWBORN[11,28,29,31]

Camelid owners vary greatly in the care given to the neonate. Some allow the entire process to proceed naturally, while others wish to be present at each delivery, assisting in the parturition and lavishing care on the cria. The weather may have a significant bearing on whether or not special care must be given, but with the value of camelids, it is logical to give maximum care in all cases. The following instructions are based on the owner/manager or veterinarian being present at the time of parturition. An evaluation of the cria should be made shortly after birth (Table 21.1).[3,33,34]

At birth, make certain that the cria is breathing and clear the airways. A simple and gentle squeeze of the nostrils eliminates any fluids present in the distal nasal cavity. Epidermal membranes may be removed from the muzzle by gentle rubbing with a towel. Insert a finger into the mouth to remove any collected mucus. The stimulus of the finger in the mouth may initiate a nursing reflex or stimulate the cria to move. Suction is the most satisfactory method for removing excess

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Table 21.1. Cria scoring system.

Score foreach sign	*Cardiac rate*	*Respiratory effort*	*Irritant response*	*Activity/Muscle tone*
0	Undetectable	Undetectable	Unresponsive	Limp, lying on side
1	Less than 80 beats/min.	Irregular, noisy, open mouth, flared nostrils	Grimace, with mild rejection	Some tone, lying on side, but with effort can move to sternal
2	Over 80 beats/min.	Steady, nonlabored	Sneeze/cough, pulling away from irritant	Sternal or actively trying to get sternal

Source: courtesy of L.B. Walker
Note: Scoring*
0–3 Critical—Notify veterinarian of an emergency.
4–6 Marginal—Notify veterinarian if second score at 10 minutes has not improved.
7–8 Normal—Give usual supportive care.
*A total score is the accumulation of the signs observed in each of the four columns. Example: If a cria's heart beat was less than 80 (1 point), was limp and lying on side (0 points), breathing steady (2 points), and pulled away from an irritant (2 points), the total score would be 5 (marginal).

Figure 21.1. Newborn alpaca with epidermal membrane intact.

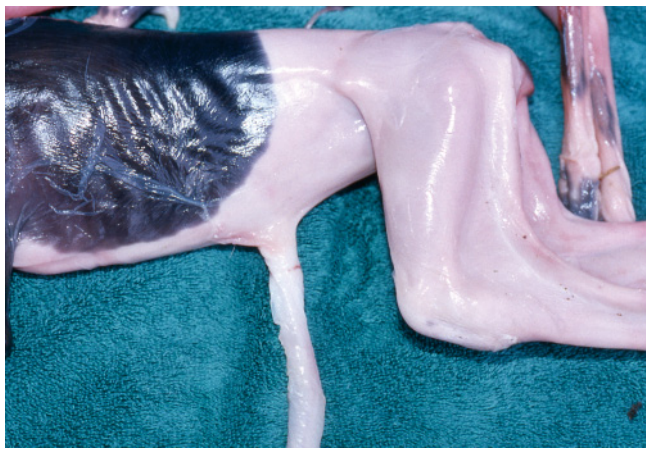

Figure 21.2. Late-term aborted fetus showing epidermal membrane and umbilicus.

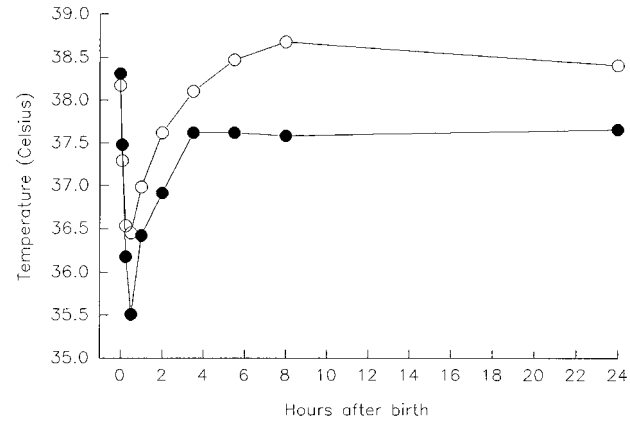

Figure 21.3. Rectal temperature in neonate alpacas for the first twenty-four hours following birth. Normal crias (open circles), hypothermic crias (closed circles).

fluids from the nostrils and mouth, but in a field situation, the cria can be held upside down for a few seconds or gently swung in an arc to develop mild centrifugal action to clear the passageways. Camelids have long necks and long legs, so care must be taken to avoid traumatizing the head. It may be advisable to grasp both a hind and foreleg before spinning.

If the cria has still not begun to breathe, artificial respiration must be initiated. The most beneficial method is mouth to nose resuscitation, since the camelid is an obligate nasal breather. Chest massage and compression are also useful. In a hospital situation, administration of oxygen via a nasal tube or endotracheal intubation is appropriate. Also examine the mucous membranes of the mouth and check for capillary refill time to ascertain the status of circulation. Cyanotic gums calls for immediate oxygen administration and detailed assessment of its cause.

The camelid mother does not lick the cria to dry it, nor does she nudge or otherwise stimulate the cria to arise. The mother may nuzzle the cria and vocalize with a humming sound (Figure 21.4). Some llama owners call this "kissing" and relate it to bonding between a human mother and baby. A quick physical

Figure 21.4. Bonding between alpaca neonate and mother.

Figure 21.5. An alpaca cria.

examination of the cria is appropriate. It is more accurate to weigh crias at twenty-four hours after birth rather than immediately, because the amount of fluid at birth varies widely between crias. It is better to weigh the cria the day after birth). The examination should include taking the rectal temperature, assessing heart sounds to determine the presence of murmur, and recording the pulse and breathing pattern. Listen to the trachea for indications of the presence of fluids so corrective steps may be taken to expel excess fluids. Observe for patency of the anus and vulva in females. In addition, the shape and direction of the ears should be assessed. Some crias may have the pinna bent and even completely folded. Straightening it requires a bandage and even the case of a 6- or 10-ml syringe for support for two to three days. If only the tip the ear is bent a simple bandage should correct it. The presence of any birth defect should be noted.

The cria should immediately begin to struggle, attempting to get up. A normal cria is able to stand, albeit unsteadily, within an hour, and will immediately attempt to make contact with the mother. Directional instincts may be poor initially, but the cria will soon locate the udder and begin nursing.

Little has been written on llama and alpaca nursing behavior.[19] In a study of a few animals, first nursing occurred at about one hour.[24] The cria must learn to semi-crouch and extend the head upward to reach the udder and assume the correct posture for suckling. The suckling posture is described as reverse parallel, with the cria standing at the side of the mother, its tail directed toward the mother's head. Nursing may be stimulated, if the cria is standing, by tickling its tail. Frequency of nursing varies with the age of the cria. During the first ten days of life, it will nurse two to three times per hour during the daylight. Up to a month of age, nursing frequency may be twice per hour (Figures 21.5, 21.6).

The time elapse at each nursing episode varies from five seconds to three minutes, but the majority of episodes last less than thirty seconds. The newborn cria will spend about 5% of its time nursing; this drops to about 3.5% by the age of one month. Llama crias begin nibbling at hay or grass at ten to fourteen days. As the intake of solid food increases, nursing episodes and time spent nursing decrease.[24]

Of primary importance is for the cria to suckle the colostrum.[3,13] As previously stated, it requires an hour or two for the cria to be able to stand and search for the udder. In one study, approximately 7.5% of alpaca crias achieved suckling on the first attempt. At the second attempt, 33.4% were successful; 53% required three efforts. Suckling occurred every half hour for the first four hours after birth, and then reduced to every hour; suckling lasted for about four minutes. Crias suckled about ten times per day for the first seven days, after which periods decreased to eight hours.[21,24]

It is neither necessary nor desirable to give an enema to every camelid neonate. The cria should be observed for passage of the meconium, which usually occurs

Figure 21.6. Camel calf nursing.

within twelve to twenty-four hours. In addition, the presence of meconium is an indication of the laxative effect of colostrum. Failure of passage of the meconium in a nursing cria by twenty-four hours after birth, especially if straining is evident, may indicate need for an enema. Warm water (200 to 500 ml) may be inserted via a syringe, emptied fleet enema container, enema can, funnel, or water bottle system. Avoid rigid tubing and be gentle with the insertion.

All of the initial care procedures may be carried out in the presence of most camelid mothers. Rarely are they aggressive, especially if eye contact is avoided. Once initial care has been completed, it is best to leave the mother and cria undisturbed.

In a herd or pasture situation, a good deal of socialization occurs, with other females gathering to nose the newborn.[18,24] Some owners prefer to isolate mother and newborn for a few hours in a stall with clean straw stall to minimize distractions for both mother and cria. This is a must for the disinterested mother.

Although human interference should be limited to essentials, the cria should be kept under constant observation to note when it can stand and when it begins nursing and for how long. The position of the head is a good indicator of a cria's well being. A head up and with attentive ears indicates that the cria is well. A cria that has not nursed by six hours following birth requires intervention.

Care of the Umbilical Cord

If the female delivers while standing, the umbilical cord usually ruptures a few inches from the body wall during the descent to the ground or, if the female is recumbent, as soon as the cria begins to struggle or the female stands up. If the cord does not rupture, it should be squeezed and cut off 15 cm (6 in.) from the body of the cria. It is not necessary nor desirable to ligate (tie a string or suture around) the umbilical cord unless a pulsing stream of blood continues to flow. Even then, it is better to apply temporary pressure to the cord. A ligature may prevent normal drainage from the cord, and the fluid is an excellent medium for the growth of bacteria. If the cord is covered with dirt or other debris, it should be gently cleaned and disinfected. If hemorrhage is evident the umbilical cord can be ligated with umbilical tape as used in foals, or with dental floss.

Several disinfectants have been employed, into which the cord is dipped or with which it is sprayed, presumably to prevent infection. Proponents are often vociferous in proclaiming the benefits of one or another solution over others, usually based on anecdotal evidence only. Recently, a study conducted on numerous foals compared the bacterial counts obtained from the umbilical cord before and following treatment with commonly recommended solutions (7% tincture of iodine, 2% tincture of iodine, tamed iodine [betadine, povidone-iodine], and chlorhexidine [Nolvasan]). The results were interesting.

Nolvasan (chlorhexidine) was the only solution that was shown to be both safe and effective in reducing the bacterial count. Although it is blue, it does not stain the hair or fingers, nor is it caustic when diluted appropriately (at full strength it is very caustic). Perhaps a psychological drawback to its use is that the cord remains soft and pinkish, giving the appearance that nothing has been done. Nonetheless, following the use of Nolvasan, the umbilical cord dried and healed within an appropriate time frame, and most importantly, umbilical infection prevalence was reduced. A one-time treatment with Nolvasan provided residual disinfection that none of the other solutions provided. A bottle of diluted, ready to use, Nolvasan solution (0.5%) is made by pouring 112 ml (4 oz) of Nolvasan into 338 ml (12 oz) of clean tap water. The diluted Nolvasan should be kept in a dark bottle, and any unused solution should be discarded after two weeks.

A small quantity for single use may be prepared by placing 15 ml (1/2 oz or 1 tablespoon) of Nolvasan in 45 ml (1 1/2 oz or 3 tablespoons) of water. A yet smaller amount may be prepared by using 5 ml (1 teaspoon) of Nolvasan in 15 ml (3 teaspoons or 1 tablespoon) of water.

Care Given the Dam

As soon as a neonate is stabilized, the dam should be examined to determine that no serious lacerations of the vulva have occurred and to determine the status of the udder. In the experience of the author (Walter Bravo), which includes delivering thousands of crias, there was never an occasion to perform an episiotomy; however, some veterinarians and owners encourage an episiotomy to facilitate passage of the head of the fetus through the birth canal. The udder should be checked for development of glandular tissue and any

Figure 21.7. Wax accumulation on the teats of a llama.

excessive swelling (edema), because edema may make it difficult for the cria to grasp the teat. Check for the presence of mastitis. The teats should be squeezed lightly to remove wax or other plugs (Figure 21.7). This procedure also allows an assessment of the quality and quantity of colostrum. Good quality colostrum gives an appropriate reading of IgG. (Figures 21.6, 21.7) Too vigorous and persistent stripping robs the cria of vital colostrum.

The parturient dam vocalizes with a guttural sound like clicking to communicate with her cria. Sometimes this sound is a humming. The dam is always close to the neonate and when the cria is suckling, the dam may turn her head and sniff the perineum of the cria. This is supposedly a way of identifying her own cria. Some dams make an episodic cluster of hummings when the crias are in their proximity and especially when they contact the head of the cria.

Agalactia is a common postpartum disorder and may be temporary or permanent. The etiology of agalactia may be as simple as failure of milk letdown because of lack of oxytocin stimulation, or it may be the result of lack of development of the glandular tissue or lack of patency of one or more teats. Other causes of agalactia or dysgalactia in camelids include hyperthermia, toxemia, mastitis, pain (lameness, arthritis), digestive upsets, ergot ingestion, stress, and genetic factors. If parturition is premature, glandular tissue lacks maturity. The degree of immaturity depends on the stage of gestation. Mastitis is more likely to occur in a female having had more than one cria but it may occur in a first-time mother.

Simple failure of milk letdown may be treated by stimulation of the udder with a warm water bath and massage or by intramuscular injection of oxytocin, which may be repeated at three- to four-hour intervals if necessary. Oxytocin, or any other drug, does not increase milk secretion in a gland that is nonfunctional.

Pain caused by mastitis, udder edema, or simple engorgement with milk may cause the female to move away, preventing the cria from nursing. Sometimes a female may be a poor mother and refuse to stand still for the cria to nurse.

Mastitis is rare in llamas and alpacas. The author (Walter Bravo) has detected only five cases of mastitis that were confirmed by the laboratory. Three cases involved two teats and the remaining cases a single teat. A manual palpation of the udders revealed the presence of hard tissue within the quarters affected. Bacteria isolated were *Escherichia coli* in three cases and *Staphylococcus aureus* in two cases. Edema of the udder after giving birth is also present in some animals. A gentle massage with udder balm two times per day for three days has been effective.

Treatment of agalactia may also involve the use of Domperidone orally twice a day for three to four days. As an antidopaminergic, Domperidone stimulates the secretion of prolactin, the hormone of milk production. Domperidone should be used cautiously, because sometimes dams produce enough milk but in the absence of a nursing cria, letdown of milk is not present.

Another treatment for agalactia is a constant stimulation of milk production. This can be done by a gentle massage of the udder and milking up to four times on a daily basis. Milking of the llama and alpaca udder requires patience. Only two fingers are used, and the production of milk yields as much as 120 ml. Repetitive milking is advised to continue milk production and when the cria is ready there should be no problems in milk production and milk letdown.

When collecting blood samples to determine IgG concentrations, serum may be different colors, varying from transparent to yellowish. A yellowish serum indicates presence of bilirubin which in turn is an indication of the turnover of fetal red blood cells to mature red blood cells.

Other procedures that were not performed during the last two months of pregnancy can be done; they may involve administering anthelmintics, trimming toe nails, and trimming incisors. Some of these procedures may have been postponed because females were close to giving birth and to avoid any stress and consequently abortion.

PREMATURITY[6]

Intensive care of the premature infant has become commonplace in humans, horses, cattle, and companion animals. No studies have been reported on the characteristics of a premature camelid cria, but sufficient observations have been made to establish certain factors, which, combined with extrapolation from

known care for other premature ungulates, may save the life of a premature cria. Prematurity may be determined on the basis of birth weight, length of gestation, observable characteristics of the neonate, and laboratory analysis of blood. There is considerable variation in the length of gestation of llamas and alpacas (330 to 360 days, with an occasional pregnancy continuing for more than a year). Such variation makes it difficult to establish prematurity on the basis of the age of the fetus. Furthermore, an accurate breeding date is sometimes difficult to establish. When pasture breeding is practiced, unobserved early embryonic death may be followed by breeding within a few days. The degree of maturity should not be based entirely on the number of days in the uterus. Foals born at full term, with known breeding dates, have nevertheless shown signs of immaturity.

Signs of Prematurity

Unusually low birth weight is the most evident characteristic of lack of maturity. Generally, if a llama cria weighs less than 8 kg (18 lb), there should be concern; if it weighs less than 7 kg (15 lb), some degree of prematurity should be assumed. Alpaca crias should weigh at least 3.6 kg (8 lb). However, an absolute weight cannot be considered as the sole determinant. For instance, if the expected delivery weight of a mature fetus is 16 kg (35 lb), a cria delivered at 11 kg (25 lb) may be immature.

Premature crias are weak and may be unable to stand or hold the head up to nurse (Figure 21.8). Overextension of the fetlocks, from tendon and ligament immaturity, causes the cria to walk on the fetlocks (Figures 21.9A and B). The sucking reflex may be weak or entirely absent.

Premature crias have floppy ears, a result of immaturity of the aural cartilage (Figure 21.10). However, not all tipped or floppy ears are caused by prematurity; genetic factors may also be involved. An immature fiber coat has a silky feel, especially over the back and rear quarters. The eyelids may be stuck together in crias that are a month or more premature.

The rubbery covering over the ungulate hoof is also present on the camelid fetal nail (Figure 21.11). In a mature cria, the rubbery mass is removed from the toe within six to twelve hours as the cria struggles to stand and takes its first steps. The covering persists for one to two days in premature crias.

The epidermal membrane is thin, friable, and easily rubbed off a full-term fetus within a few hours after

Figure 21.8. Depressed premature newborn llama.

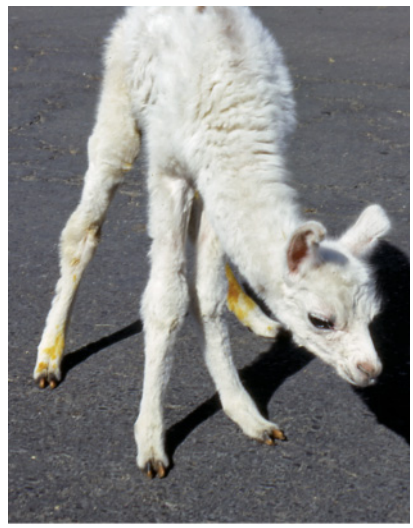

Figure 21.9A. Relaxed tendons/muscles.

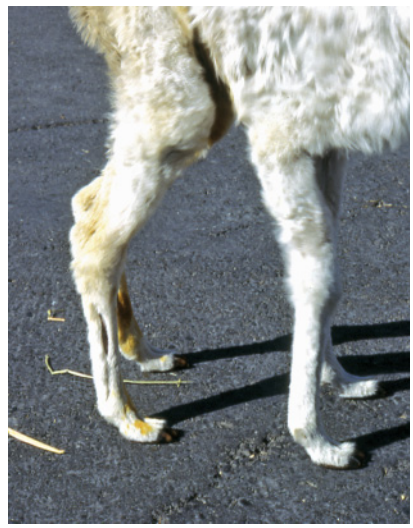

Figure 21.9B. Angular limb deformities.

Figure 21.10. Floppy ears of a premature cria.

Figure 21.11. Persistent rubbery covering on the toenail of a premature cria.

birth. In the premature cria, the membrane is thicker, and remnants may cling for twenty-four hours.

The incisor teeth have erupted in the normal, full-term SAC fetus (Figures 21.12A to C). Failure of these teeth to erupt for days or weeks after birth is a primary indication of prematurity. Another sign of prematurity is that premature crias sleep most of the time. They even adopt a lateral position and it may be thought that the cria is dead. Premature crias may sleep for up to three days and then may become more active and interact with other crias.

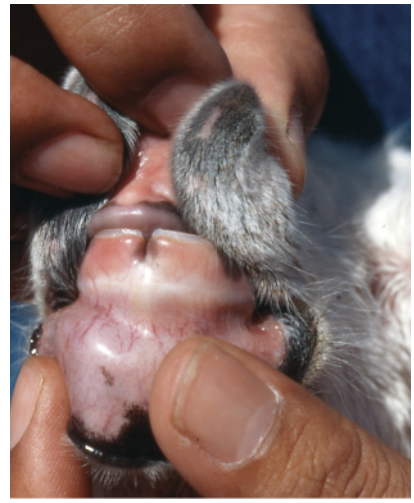

Figure 21.12A. Dental eruption at the time of birth, normal.

Figure 21.12B. Premature cria, incisors covered with membrane.

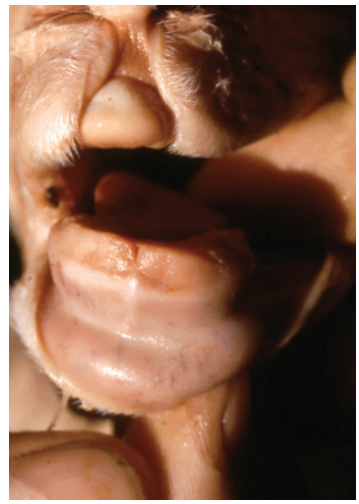

Figure 21.12C. Membrane over all the premature alpaca's incisors at birth.

Feeding a premature cria involves the use of oral intubation. The tube should be passed slowly through the pharynx to make sure that is the esophagus. The volume of milk fed a premature cria is also less than for a normal cria.

Bradypnea (less than 10/min) is common in the premature cria. There is an absolute hypercapnia, but this fails to initiate respiratory effort, and PaO_2 is low. Although pulmonary surfactants have not been studied in camelids, it is assumed that conditions present within the alveoli of the llama lung are similar to those of foals, calves, and humans and that the absence of surfactants in the premature cria decreases oxygen transfer across the alveolar membrane.

Mature neonates may be hypoglycemic at birth and for a few hours thereafter.[9] Premature crias may remain hypoglycemic and lack the energy to stand or nurse. The premature cria has poor thermoregulatory ability and is thus at great risk of hypothermia. The body temperature of a cria suspected to be premature must be carefully monitored. Leukopenia, caused by an absolute neutropenia, is characteristic of prematurity. The immature adrenal cortex is unable to respond to adrenocorticotrophic hormone stimulation to produce cortisol and deal with stress.

The intestinal mucosa of the premature cria is refractory to absorption of immunoglobulin (Ig) macromolecules.[12]

Premature foals have incomplete formation of the carpal and tarsal bones, predisposing to angular limb deformities.[17] This phenomenon has not been investigated in premature camelids, but it warrants consideration, because angular limb deformities are common in llamas and alpacas.

It is important to recognize that the fully mature cria may exhibit one or more of the characteristics of immaturity. The overall morphology and activity of the cria must be evaluated on an individual basis. Intrauterine septicemia may simulate many of the signs of prematurity, as may congenital heart defects. A neonatal maladjustment syndrome has been reported in foals wherein the foal fails to adapt to extrauterine life, even though fully mature. This situation has not been reported in camelids but should be considered.

Prematurity is life threatening. Prompt diagnosis and intensive care is mandatory.[15] The veterinarian should work closely with clients to educate them on early recognition so that prompt, effective remedial action may be taken.

A crucial component in the survival of the neonate is the concentration of immunoglobulin G (IgG). This specific immunoglobulin is the main source of antibodies for the neonate. Camelid crias are born without IgG (agammaglobulinemic) because the camelid placenta is refractory to the passage of IgG from the mother to the fetus. Crias must acquire antibodies from colostrum. Upon ingestion of colostrum, IgG levels increase dramatically to 2,500 mg/dl in serum. This value is maintained for the first ten days after birth, and then it decreases slowly to 1,500 mg/dl during the next ten days.[10] There is no difference in the IgG concentrations between llama and alpaca crias.

"Dismaturity" is a term used for crias that have a complete time of gestation but nonetheless are still weak. They may lack a suckling response and the administration of colostrum is imperative to avoid a failure of passive transfer. These crias may become normal after three to four days and join other crias in socializing.

IMMUNOGLOBULINS

Production of Immunoglobulins

In the cow, preparation of the mammary gland for colostrum production begins about five weeks prepartum. The stimulus is probably rising estrogen levels. Special receptors on the epithelial cells of the mammary gland selectively bind serum IgG, which is, in turn, taken into the cell and ultimately transported to the lumen and hence the colostrum.[25] Factors that influence the amount of IgGs in bovine colostrum are:

1. Failure to allow the mammary gland to make preparation for colostrum production. In camelids this may result if the previous cria is allowed to continue to nurse for a year or if the female serves as a wet nurse for multiple crias.
2. Genetic factors such as breed differences.
3. Number of offspring. Multiparous cows produce more and higher-quality colostrum than uniparous cows.
4. Diet. Energy-deficient diets result in a decrease in the volume of colostrum produced but do not alter the composition.
5. Milking. Ig content of the first milking is double that of the second milking, and content is halved with each succeeding milking.[25]
6. Premilking, which dissipates IgG.
7. Leakage of colostrum from an engorged udder.
8. Administration of long-acting corticosteroids over a period of nine to nineteen days to induce parturition.
9. Premature birth.
10. Prenatal mastitis.
11. Immune status of the female prior to parturition. In llamas and alpacas, research indicates that there is no transfer of IgG from maternal blood into the mammary glands. Rather the mammary gland synthesizes IgG before parturition, which starts approximately thirty days before parturition. The concentrations of IgG of the pregnant dam do not vary prior to parturition.

Table 21.2. Colostrum composition (as fed).

Species	*Water (%)*	*Protein (%)*	*Fat (%)*	*Kcal/L*
Llama	74.0	16.5	0.95	1341
Goat	73.5	12.0	6.0	1064
Sheep	58.8	20.1	17.7	2064
Cow	70.0	17.0	3.0	991

Colostrum Composition and Absorption

Colostrum differs widely from milk.[5,17,20,27] Colostrum is high in protein (immunoglobulins [IgG, IgA, IgM]), high in chloride, low in fat, low in carbohydrates (lactose), low in calcium and phosphorus, and higher in minerals and vitamins than milk. It contains a high number of white blood cells that are vitally involved in providing protection against infection (macrophages, B and T lymphocytes, neutrophils), and it is high in another substance called "complement," which inhibits infection of the lining of the stomach and intestines.[11] Camel and SAC colostrums are similar in fat percentage and are white and watery in contrast to the thick and creamy colostrum of cattle (Table 21.2). The high protein content of colostrum is the result of the presence of IgGs.

Camelids, like other ungulate species, obtain passive protection from IgGs via intestinal absorption of macromolecules in colostrum. Failure to ingest colostrum during the first twenty-four hours of life is probably the most important predisposing cause of mortality in camelid neonates. In Peru, serum IgG concentrations of alpaca crias that died were significantly lower than those of crias that lived.[10,11] Colostral intake serves two purposes: (1) a source of IgGs for absorption into the circulatory system across the intestinal mucosa and (2) local effects, protecting the mucosa against endotoxins and microorganisms. The first requires ingestion within twenty-four hours of birth, while the second benefits the animal for a number of days and is brought about by local action of the specific IgGs, nonspecific complement, transferrins, and lactoferrins.

Recent work in Peru also indicates that the time of suckling colostrum by the cria is important to achieve more than 1,000 mg/dL at forty-eight hours. The highest IgG concentration happens when crias start getting colostrum between two and four hours after birth. Suckling colostrum at six or eight hours after birth also provides marginal but close to 1,000 mg/dL at forty-eight hours (Figure 21.13b). Furthermore, three teats with good colostrum provide enough IgG at forty-eight hours. This may happen when one of the teats is missing or an accident happens at time of shearing.

The IgGs contained in colostrum are large-molecule proteins. Normally such proteins cannot be absorbed from the intestinal tract but are digested to amino acids, which can then be absorbed. During the first twenty-four hours of life, the wall of the intestine remains open to absorb IgGs but then closes to prevent infections from also gaining entrance to the body. The mechanism of absorption of IgGs is a biological marvel (Figure 21.14). The cells lining the intestine contain vacuoles. The protein molecule and the vacuole line up at the cell wall. The protein passes through the wall and is engulfed by the vacuole and thus is spared the usual response of the cell to such a foreign body. The vacuole containing the IgG moves to the opposite end

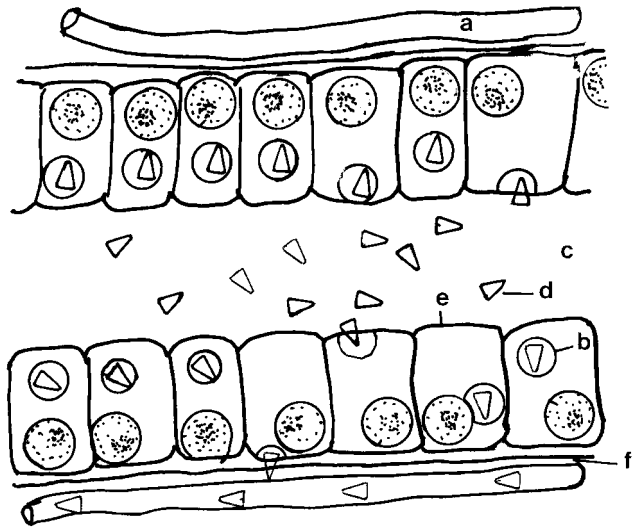

Figure 21.13. Diagram of the transfer of immunoglobulins through the mucosa of the intestine. (A) Lymph vessel, (b) IgG molecule in a vacuole, (c) lumen of the small intestine, (d) IgG molecules in colostrum, (e) epithelial cells of intestinal mucosa, (f) basement membrane.

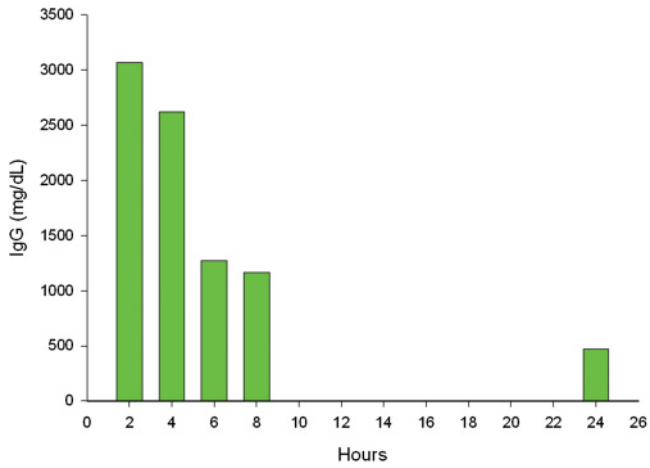

Figure 21.14. Concentrations of IgG in crias at forty-eight hours when suckling of colostrum begins at two, four six, eight, and twenty-four hours after birth.

of the cell; the IgG passes out of the cell into the lymphatic vessels and then to the general circulation.

Information on the initiation of closure of the intestinal wall to IgG is extrapolated from studies conducted on cattle, sheep, and horses. In a normal full-term cria, closure begins soon after the first feeding and certainly by six hours postpartum, whether or not the cria has nursed. Fifty percent closure has taken place by nine hours. A progressive change occurs in the cells of the intestinal wall that begins to destroy the IgG. The newborn has little protein digestion ability until a few hours after birth at which time the IgG, which is a protein, begins to be digested. Stomach acid is necessary to curdle milk preliminary to digestion. The newborn stomach contains little acid until a few hours postpartum.

The intestinal mucosa of the premature cria is not prepared for absorption of Ig macromolecules, so even though colostrum is administered via stomach tube, there may be a failure of passive transfer of Ig (Figures 21.13, 21.14). This is an extremely important factor, necessitating intravenous or intraperitoneal administration of plasma to avoid mortality from neonatal infections.

Colostral proteins are absorbed in the distal small intestine. IgGs of milk deposited in compartment 1 (C-1) of the stomach are delayed, or IgG may be denatured before it ever reaches the small intestine. A number of other macromolecules may be absorbed along with IgGs, including alkaline phosphatase and G glutamyltransferase. Elevated levels of the two enzymes in the neonate may not be indicative of a disease state.

In cattle, a number of behavioral factors modify the ultimate levels of IgGs in the circulation of the calf. Mothering has a beneficial effect, with higher levels attained when the calf is kept with the mother and allowed to nurse freely, as contrasted with calves allowed to nurse periodically but kept separate from the mother. Lowest of all are levels reached in bottle-fed calves, even when given equal or greater volumes of true, first-milk colostrum. It is not known how these factors affect camelids. Although the llama female does not lick the cria as does a cow her calf, a bonding and nursing response nevertheless occurs. Other factors that may affect camelid immune response follow. Cold weather decreases cria vigor and hence overall colostral intake and absorption. Heat stress likewise inhibits Ig absorption efficiency.[25] A cria delivered following a dystocia is not likely to have a depressed Ig uptake, but one delivered by cesarean section will. Such animals must be watched carefully (Figure 21.15).

Absorption is directly related to the amount of IgGs ingested. There seems to be better absorption from a first ingestion with a higher concentration of Ig than from the same total quantity of IgG in a larger volume of milk.

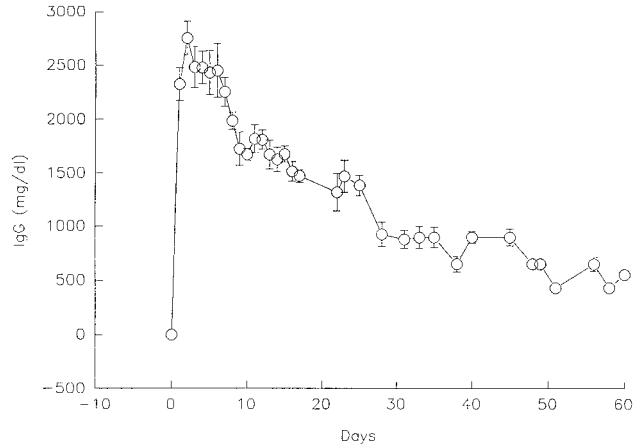

Figure 21.15. Immunoglobulin concentrations in the alpaca cria from birth to day 60. (Bravo et al. 1996. Immunoglobulin G concentrations in periparturient llamas, alpacas, and their crias. Small Ruminant Research.)

Using experience from calf rearing as a guide, it can be assumed that crias deprived of all colostrum are at risk of dying from colisepticemia within three to four days of birth. If some Ig has been absorbed, the risk of disease is still great but will occur later in the form of diarrhea, pneumonia, or chronic polyarthritis. If the dam has not been vaccinated against nor exposed to a specific pathogen, the cria remains at risk from that pathogen. Even crias absorbing adequate IgGs may succumb to an overwhelming infection.

FAILURE OF PASSIVE TRANSFER (FPT) OF IMUNOGLOBULINS[1]

Causes of the failure of immunoglobulin transfer are listed in Table 21.3.

Detection of FPT

Even with intense management, it is thought that 10% to 30% of calves are hypo- or agammaglobulinemic as a result of failure of passive transfer of IgG. This is significant in neonatal morbidity and mortality.[3,5] Veterinarians dealing with llamas and/or alpacas must assume this to be true in camelids also and act accordingly.

Early recognition of FPT is crucial to the well-being of the cria. A history of no ingestion of colostrum during the first twenty-four hours is prima facie evidence of FPT, but this may not always be known. The cria may have appeared to nurse without obtaining colostrum for reasons enumerated in the agalactia discussion. Also, the amount of colostrum intake is important.

Single radial immunodiffusion is the most specific laboratory test to determine the level of IgG in calves

Table 21.3. Causes of failure of passive transfer of immunoglobulins.

Conditions associated with the dam
Rejection of the cria
Not immune to common diseases
Poor colostrum production
Obesity (Inhibits mammary gland development and may make it difficult for the cria to grasp the nipple)
Sore udder
Udder edema
Tiny nipples
Plugged teat ducts
Overabundance of milk may cause dam discomfort and reluctance to allow nursing. Heavy producers may also begin to leak colostrum from the teats because of the pressure.
No milk letdown for 24–48 hours; in the meantime the intestinal tract closes to the absorption of immunoglobulins
Mastitis—blood, pus, clumps, odor, color change.
Older females may begin to have diminished colostrum and milk production.
A pregnant female may allow a juvenile to continue nursing during the 3–4 weeks prior to parturition.
Premature delivery—dam may not have produced IgG yet.
Causes—unknown, heat stress, excessive stress of the pregnant female (physical restraint, medical procedures [vaccination, deworming, nail trimming, shearing], transporting, social stress [regrouping, new arrivals]), and prenatal infection
Conditions associated with the cria
Weakness—unable to stand (prematurity, fatigue from prolonged labor, prenatal infection)
Congenital defects
Facial (dental malocclusion, wry face, choanal atresia)
Limbs (carpal contraction, fused joints [arthrogryposis])
Trauma during delivery
Prenatal septicemia/endotoxemia, *Escherichia coli* "dummy syndrome" cria
No nursing reflex (prematurity, septicemia, endotoxemia, hypoxemia)
Conditions associated with human interference
Too vigorous unplugging of teats
Failure to prevent late nursing of older offspring
Failure to properly immunize the dam prior to birth

and foals.[8,16] Other reliable methods include refractometer measurement of total protein, serum electrophoresis, zinc sulfate turbidity, and zinc sulfite precipitation. All of these methods have been used in camelids, but except for total protein evaluation and electrophoresis, the results have been equivocal. Electrophoresis is good but time-consuming, and action must be taken prior to obtaining results. This leaves total protein evaluation of the blood as the only practical test available, but one that has not yet been evaluated sufficiently. Total protein levels of less than 5 g/dl suggest FPT. Levels between 5 and 6 g/dl are equivocal, and levels over 6 g/dl indicate probable successful passage of IgG.

Management of FPT

The management of FPT depends on the stage at which one becomes involved. Involvement at parturition is ideal.[28] If the cria has failed to nurse by six hours, an attempt should be made to offer it colostrum obtained directly from the mother, or stored, frozen camelid or goat colostrum. Mare and cow colostrum have been used, but goats are more likely to have been immunized for the diseases of most concern in camelids. Furthermore, goat colostrum and milk composition are most nearly like those of camelids. It is virtually impossible to milk sufficient colostrum from the postparturient female to supply the needs of the cria. It is probably be more satisfactory to rely on frozen goat colostrum. However, the source of the colostrum must be scrutinized. The desired colostrum is the first-drawn milk only. Many livestock producers operate on the principle that since the milk is not normal for up to seventy-two hours after parturition, all fluid withdrawn during that time is colostrum. Unless it is specified that only first-drawn milk is to be used, producers may not supply suitable colostrum. Furthermore, it is important to determine that the herd is free of disease and that the colostrum is collected and stored under sanitary conditions.

Treatment of FPT[30]

Clinical diseases following FPT vary, but the most critical are bacteremia, septicemia, and enteritis. These require intensive care with appropriate antibiotics and

fluid therapy. Total parenteral nutrition may be necessary.

The most effective method of treating FPT is administration of plasma to crias. Plasma may be given by three methods: intravenously, intraperitoneally, and orally. Intravenous administration of plasma requires the insertion of a cannula into the jugular vein and then the plasma is administered slowly, which may take up to two hours. Crias are susceptible to lung edema and plasma should be administered slowly.

When administering plasma intravenously to an extremely weak, debilitated, hypovolemic, and dehydrated cria, the placement of the intravenous cannula into the jugular vein may be facilitated if the cria is in a lateral position, with the head at the same level. Digital pressure over the jugular allows the vein to distend so the cannula may be inserted with only one attempt. Use a catheter that is 3 or 4 inches long.

Intraperitoneal plasma administration may be done faster. The left flank of a cria is prepared aseptically for a small 5-mm skin incision. A blunt cow teat canula is inserted. Warmed plasma is administered in a matter of five to ten minutes, even under field conditions. The skin incision may be sutured and the cria returned to its mother. One of the authors (Walter Bravo) has not observed any adverse reactions (anaphylaxis) to this procedure, neither was the kidney perforated nor the intestines. If the needle is too superficial plasma will be deposited either intramuscularly or subcutaneously and the depth of insertion should be improved to allow plasma enter the peritoneal cavity.

Plasma also may be given orally by stomach tube. The decision to administer plasma orally is made when the dam does not have any colostrum or there is an impaired udder.

Facts about FPT

1. Some crias with IgG levels less than reported "normals" or with minimum levels survive and remain healthy. For success, sanitation and hygiene must be excellent.
2. Crias with high levels of IgG may succumb to infection. The body may not be able to cope with overwhelming exposure to an infectious microorganism. Furthermore, immunoglobulins (antibodies) are produced in response to specific antigens, so if the dam was not vaccinated appropriately, the colostrum will lack certain protective antibodies, even though IgG levels are high. In addition, not all infectious agents stimulate the production of IgG.

CARING FOR THE ORPHANED CAMELID[14,15]

The cria may be orphaned at birth as a result of rejection by the mother, disease, injury, or death of the mother, or because of a variety of factors previously discussed in this chapter. Successful rearing of an orphan camelid is an art. Certain individuals within the camelid industry have made tremendous contributions to the knowledge of the rearing of orphans. Their experience and expertise should be sought when faced with the challenge of long-term hand feeding. However, the veterinarian should be able to deal with short-term problems and make recommendations on how to deal with FPT and neonatal infections.

Some basic information follows. Camelids rarely learn to drink milk from a bucket. Many crias refuse to allow supplementation of mother's milk by nursing on a bottle and seem to desire one or the other. There are exceptions, but it requires much patience to succeed. If the female is slow in coming into lactation, it may be necessary to supplement by intubation rather than bottle-feeding. It is important not to overfeed, or the cria will have no stimulus to attempt nursing.

If a cria is bottle-fed, the bottle should be held level, even with the head, not above it, to avoid aspiration (Figure 21.16). The hole in the nipple used for a weak baby should be tiny, then enlarged with a cross cut as strength is gained. A human baby nipple or a lamb nipple with a cross cut, rather than a round hole for an orifice, is recommended for feeding the cria. Offer 120 ml (4 oz), or as much as the cria will take, every two to three hours up to twenty-four hours or until the cria is able to stand and nurse on its own.

If the cria refuses or is unable to suckle an artificial nipple, it is necessary to intubate the cria. Intubation is easily carried out by straddling the recumbent cria in a kneeling position. Many different tubes and methods are available to administer colostrum (Figure 21.17).[10]

A tube with a 10- to 13-mm outside diameter (24 French) and an end port, not a blind tip,[a] is recom-

Figure 21.16. Bottle position for feeding an orphan llama.

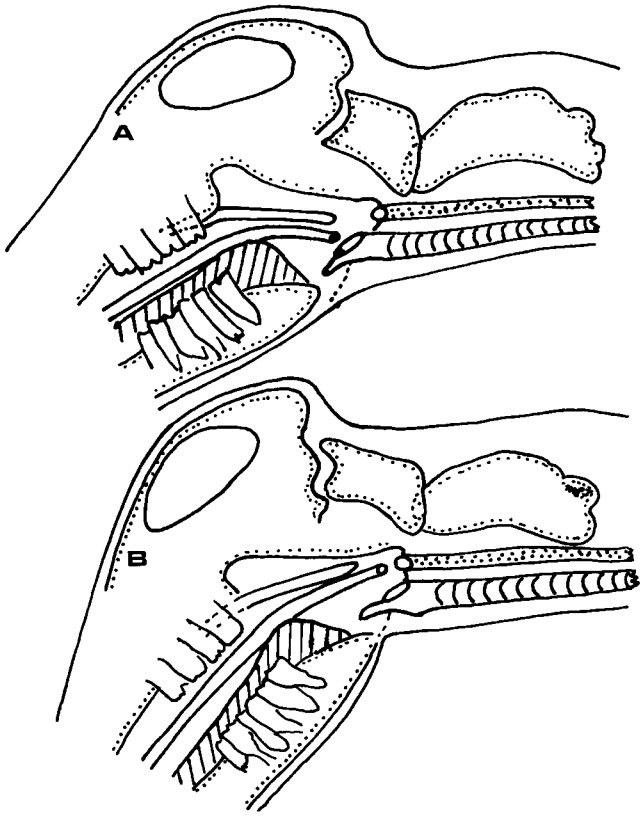

Figure 21.17. Orogastric intubation. (A) Head too straight, allowing insertion into the trachea, (B) proper head position.

mended. Larger tubes may cause interference with cardiac function. The tube should be lubricated with water. Hold the head in a semiflexed position to insert the tube into the mouth. No speculum is necessary. Gently push the tube to the pharynx and allow the cria to swallow it. Palpate the left cervical region for esophageal placement and continue insertion only to the thoracic inlet. Fluid traversing the thoracic esophagus may cause reflex closure of the esophageal groove and shunting of the colostrum to C-3 of the stomach, where it belongs. Do not insert the tube into the stomach, because this will ensure placement of the colostrum into C-1 of the stomach. Milk in C-1 will ferment, contributing to digestive upset.

The first tube feeding should be given by twelve hours postpartum in the amount of approximately 1% of the body weight, 112 ml (4 oz) for an 11.34-kg (25-lb) cria. Repeat the tube feeding every two hours until 10% of the body weight is reached. The stomach capacity of most mammals is 25 to 50 ml/kg of the animal's body weight. For an 11.34-kg llama cria, this amounts to 280 to 560 ml, but it is undesirable to use the higher volumes until the stomach has expanded and is prepared to accept such amounts. Otherwise, the fluid may back up and spill into C-1 or, worse, cause regurgitation with the attendant risk of aspiration. Intubation should be repeated at fifteen and twenty hours if necessary.

If twenty-four hours have elapsed since birth, it must be assumed that the intestine is refractory to absorption of IgGs. At this time, the only recourse is to administer blood, plasma, or serum, preferably obtained from the dam. Blood typing is in its infancy in camelids, but erythrocyte factors are known, and plasma is preferable, at a volume of 10 to 20 ml/kg body weight. No studies have been conducted to determine the most suitable volume for camelids, so extrapolations have been made from livestock studies. For an 11.34-kg (25-lb) cria, the recommended volume is 120 to 240 ml. Plasma may be administered intravenously, intraperitoneally, intramuscularly, or subcutaneously. However, the volume is quite high for the latter two, and even with dispersal in a number of sites, the cria will experience distress and possibly lameness for a few days. The preferred technique is to give plasma intravenously, but it is slower than intraperitoneal administration.

Another method for dealing with an orphan cria is adoption by another dam. In various instances dams without crias may be available, especially when parturition occurs in a definitive season, and dams that lose their crias are good candidates to adopt an orphan cria. By nature SACs are good mothers and they may adopt another cria if they have lost their own cria recently. In some areas of the world, the skin of the dead cria is put on the top of the orphan cria and then maintained with the surrogate dam separated from other animals. Some people try to rub the perineal area of the orphan cria close to the nose of the dam, and it may smell and adopt the cria. Others just put the orphan cria with the surrogate mother and let the cria nurse from the adopting dam. Hopefully the new mother will accept the cria and feeding the cria with milk replacement is not necessary. The manager or owner must be patient during this process.

Milk Replacement

A variety of milk substitutes (pasteurized, homogenized cow's milk; pasteurized goat's milk; commercial lamb and kid milk replacers) have been used successfully by caregivers who understand basic principles. Owners faced with the daunting task of having to provide milk for a cria are often given conflicting recommendations by other owners who may have tremendous or minimal experience themselves. Camelid owners sometimes feel that veterinarians confuse clients with conflicting information about what to feed and how to mix milk replacers for crias.

The optimal quantity of colostrum for the cria is 10% of its body weight, to be given over twenty-four hours, preferably eighteen hours. Obviously, it is most

desirable that this be the mother's colostrum, but if, for various reasons, the cria is unable to suckle, or the mother can't provide the colostrum, substitute colostrum should be given. There is no exact match for camelid colostrum, but Table 21.2 provides information on three other colostrums. Both cow and goat colostrum are used. Sheep colostrum is too high in fat and will likely make a camelid cria develop diarrhea.

There is no difference between the composition of llama and alpaca milk, so the term "llama" applies to both.[24] Table 21.4 provides an approximate composition of selected components of various milks. It should be evident that camelid, goat, and cow milks are quite similar in composition. Either goat or cow milk makes a satisfactory substitute for camelid milk, but some experienced orphan rearers recommend the use of goat milk because it is the most nearly like camelid milk. Because goat milk fat is naturally homogenized, it may be more easily digested than the larger fat globules of the milk of other species. Any substitute fresh milk should be pasteurized. Orphans also satisfactorily tolerate kid and lamb milk replacers. Once a substitute is chosen, it is unwise to switch back and forth between milk products.

Consideration should be given to a cria's nutrient requirement. A 10-kg (22-lb) cria has a basic energy requirement (BMR) of 394 kcal/day to perform body functions in an optimal environment. Its maintenance requirements are BMR plus 1.5 BMR or 985 kcal/day. Camelid milk has approximately 822 kcal/L, and Land O'Lakes Lamb Milk Replacer, mixed with 1.5 pints of water, has 864 kcal/L (qt). Thus, a 10-kg (22-lb) cria requires approximately 1 L of milk per day (1 qt [32 oz]) or 10% of its body weight.

Commercial Milk Replacers

Numerous products are advertised as being the ideal milk replacer. Most commercial products are satisfactory if basic principles are understood and followed.[9] Companies supplying milk replacers for camelids are listed in Table 21.5.

Unless you have sound data and/or experience to direct you, always follow the directions provided on the label. Even these may be confusing. Several products supply a plastic measuring cup to measure the powder. Directions are given to mix the powder with a certain quantity of water, usually listed in pints. Some people who are aware that there are 2 cups (240 ml each) in a pint proceed to mix the solution using the powder measuring cups to measure the water. However, the powder measuring cup may hold 270 ml instead of 240 ml of water (Table 21.6).

The mixing directions on the label of Land O' Lakes Lamb Milk Replacer provide the appropriate reconstituted composition for lambs, but it makes a more concentrated milk than is normal for the camelid. Using 1.5 pints of water instead of 1 pint provides the closest approximation to camelid milk, but crias have been reared on replacer mixed according to the label directions. Note that kid milk replacer mixed according to label directions is close to camelid milk in composition (Table 21.7).

Another consideration is that product ingredients may vary. Also, proximate analysis of some commercial products has been performed by users, and the levels as stated on the label have not always been accurate. Palatability factors may also enter into acceptance of any product.

WEANING[25]

Most llama crias wean themselves by six months of age. A few persist in nursing for more than a year if the female will tolerate it. However, in these instances, the amount of milk consumption may be negligible, and the nursing has become a behavioral pattern. Some females may kick the cria away at four to five months. In vicuñas, the crias are weaned at approximately six months and expelled from the herd at nine to twelve months. Guanacos have a slightly different weaning pattern, and yearlings may still be found with the mother while a cria of the year is nursing.

If a cria has converted to pasture and hay, there is little advantage to continued nursing after six months, and there may be a decided disadvantage to the female if she is pregnant again. Some owners wean at five months, and this is entirely satisfactory if the cria is well adapted. Orphaned crias may be able to make it on their own at two months of age, but there is less risk of stunting if the nursing period can be prolonged.

Table 21.4. Milk composition (as fed).

Species	*Water (%)*	*Protein (%)*	*Fat (%)*	*Kcal/L*
Llama	86.9 (79–89.5)	3.4 (2.1–4.4)	24.5.7 (1.3–5.9)	822
Camel	86.4 (85.02–88.5)	3.6 (3.5–5.2)	2.9–5.5	—
Goat	87	3.3	4	680
Sheep	81	6.2	7.9	1,138
Cow	87.3	3.3	3.6	653

Table 21.5. Some companies manufacturing lamb and kid milk replacers in the USA.

Land 0′ Lakes 2827 8th Ave. S. Fort Dodge, IA 50501 Phone: (800) 369-3060 FAX: (515) 576-2685	Products:	Ultrafresh Lamb Milk Replacer Instant Kid Milk Replacer
Merrick's Inc. 2415 Parview Rd. P.O. Box 620307 Middleton, WI 53582 Phone: (608) 831-3440 FAX: (608) 836-8943	Products:	Super Lamb Milk Replacer Super Kid Milk Replacer
MannaPro Milk Products 707 Spirit Park Dr. Suite 150 Chesterfield, MO 63005 Phone: (800) 690-9908 FAX: (636) 681-1799	Products:	Lamb Milk Replacer Kid Milk Replacer
Calva Products, Inc. 4351 Winery Rd. Acampo, CA 95220 Phone: (209) 339-1516 FAX: (209) 339-1517	Products:	Lamb Lac—Lamb Milk Replacer Kid Lac—Kid Milk Replacer
Cuprem, Inc. 202 North Smith Kenesaw, NE 60956-0147 Phone: (800) 228-4253 FAX: (402) 752-3397	Product:	Costro Cria Milk Replacer
Purina Mills, LLC 100 Danforth Dr. Gray Summit, MO 63039 Phone: (800) 227-8941 or (536) 742-6100	Product:	Purina Kid Milk Replacer

Table 21.6. Cup volume variations.

Measurer	*Volume of water (ml)*	*Weight of powder (g)*
Kitchen cup	240	
Land O′ Lakes—Lamb, red	270	114
Land O′ Lakes—Kid, light blue	240	114
Merrick's—Lamb, white	230	114
MannaPro—Lamb	270	114

NEONATAL DISEASES[26]

Neonatal septicemia has been discussed in Chapter 7. Congenital/hereditary diseases are discussed in Chapter 22 or in the appropriate organ system discussion.[23] Defects that should be considered include choanal atresia, atresia ani[7,22] and atresia coli, persistent urachus, and cardiac anomalies. Acquired conditions include urethral obstruction, ruptured bladder, and retained meconium, all of which are discussed in appropriate sections.

Umbilical Infection

Acute Umbilical Infection

DEFINITION. Navel ill (omphalophlebitis) is inflammation of the umbilical stump and associated structures, including the urachus, arteries, veins, and surrounding tissues (Figure 21.18). The infection may be localized in the region of the umbilical stump or spread to the bladder via the urachus, the liver via the umbilical veins, or the general circulation to become a septicemia, or it may spread directly to the peritoneum causing peritonitis.

The majority of infections result from contamination of the cord with ubiquitous organisms in the immediate environment. This is especially true if failure of passive transfer of IgG has occurred, rendering the neonate susceptible to otherwise nonpathogenic organisms. A variety of opportunistic Gram-negative organisms are the usual cause.

SIGNS. If infection is limited to the base of the umbilical stump, there may be swelling, heat, pain on palpation, and an exudate from the stump. Infection of the

Table 21.7. Composition of Land O' Lakes milk replacers and llama milk.

Product	*Protein (%)*	*Fat (%)*	*Kcals ME/L*[a]
Lamb Powder	24	35	
114 g/454 g (1 pt)	27 g/454 g = 5.95	40 g/454 g = 8.8	1152
114 g/681 g (1.5 pt)	27 g/681 g = 3.96	40 g/681 g = 5.9	864
Kid Powder	26	20	
114 g/681 g (1.5 pt)	29.6 g/681 g = 4.3	22.8 g/681 g = 3.3	
Llama Milk	2.1 to 4.4	1.3 to 5.9	822

[a] Kilocalories of metabolizable energy per liter.

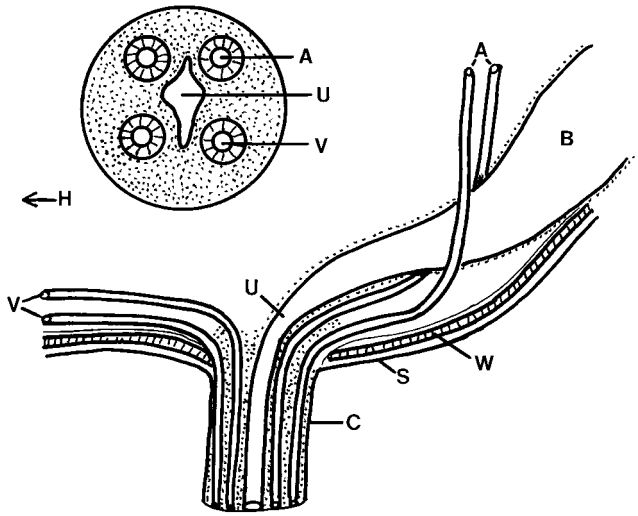

Figure 21.18. Diagram of the relationship of the bladder and vessels. (A) Umbilical artery, (B) bladder, (C) umbilicus, (H) cranial direction, (U) urachus, (V) umbilical veins, (W) body wall.

umbilical arteries, veins, urachus, or peritoneum may have no external manifestations, but the animal may exhibit colic, depression, or anorexia and spend an inordinate amount of time recumbent.

Diagnosis. Signs and physical examination are primary methods of diagnosis, but hematology and ultrasound examination of the umbilical region may be helpful.

Therapy. If infection is detected early (within twenty-four hours), broad-spectrum antibiotics and supportive care may suffice to arrest it. If infection is allowed to spread or if abscessation occurs, it is usually necessary to remove the infected tissue and structures surgically.

Chronic Infection

Definition and Etiology. Chronic infection may take many forms and thus has a variety of common names including navel ill, joint ill, septic physitis, septic polyarthritis (multiple joints), and septic epiphysitis. Basically, the cause is the same as for acute infection, but the manifestation of disease is delayed for varying reasons, including innate resistance of the animal, slow-growing abscesses, or localization of infection in various organs and tissues.

Signs. Signs are highly variable depending on the site of infection and may be delayed for three to six weeks. Urinary tract abscess may result from progression of infection from the urachus. Liver abscesses may occur. Septicemia may spread infection to the kidney, joints, physes, and bone. Sudden lameness in one or more legs is the usual initial sign. Joints may be swollen, hot, and painful upon manipulation. Arthritis caused by trauma is rare.

Diagnosis. Arthrocentesis and radiography are used to establish joint infection. Cultures may be negative even in an infected joint. Blood cultures may be more useful.

Therapy. The prognosis of septic arthritis is guarded to grave. Aggressive therapy is crucial for satisfactory resolution. Systemic and intra-articular broad-spectrum antibiotic therapy is necessary. Antibiotic therapy must be continued for four to six weeks. Joint lavage with normal saline is used to remove fibrin and debris to minimize destruction of the articular cartilages and synovial membranes.

Diarrhea

Diarrhea may occur when the cria ingests too much colostrum and/or colostrum becomes too acidic. The cria shows a yellowish and/or orange watery diarrhea that lasts for a day. Sometimes the diarrhea is so severe that the cria becomes recumbent from pain. This type of diarrhea is generally temporarily and the cria has recovered on the following day. Most of the time no treatment is necessary. If the diarrhea continues it may be caused by bacteria or the dam has mastitis; consequently, the cria is also feeding itself with bacteria that will cause diarrhea. In this case, the administration of oral antibiotics may be necessary.

Dummy Syndrome

This condition is observed when crias, instead of approaching the udder of the dam to suckle, look in

other places, such as the corner of a room, the feeder, or the hinge of a door. It seems that these crias are so disoriented that they wander around, walking with the head up, and no matter what is done, they always return to those places. The author (Walter Bravo) has observed that in these cases, dams do not call or hum for the cria; rather, they continue grazing. Patience must be exercised because it is frustrating to watch a cria looking for food in odd places and not finding the udder. After two to three days, crias find the udder of their dams and this behavior stops.

A similar behavior has been also described in foals, and is related to hypoxia during the last months of pregnancy or at time of parturition.

Infections may cause this syndrome. It is well recognized in the foal that the brain blood barrier is not well established yet, and consequently any bacteria may enter the brain and cause an inflammation and edema, which in turn causes this behavior. That could be the case for camelids as well. The treatment encompasses administering colostrum and/or milk, even by intubation if there is no suckling response. Depending on a blood test, administration of glucose may also help. Because there may be hypoxia, administration of oxygen to the cria may prevent development of this syndrome.

IMMUNOPROPHYLAXIS

The most protective regimen for the camelid cria is to make certain that the dam is immunized at least two months prior to parturition and that a booster vaccination is given four to six weeks before parturition. This ensures maximum production of specific IgGs in the colostrum.

The immune system of the neonate lacks full maturity, and the neonate may not be able to respond to vaccination at an early age. Nonetheless, veterinarians with sheep experience recognize that enterotoxemia extracts a high toll in lambs that are not vaccinated by two weeks of age. Knowledge of clostridial diseases in camelids in North America is meager, but prudence dictates that vaccinations be administered within the first month of life, with boosters a month later and again at weaning.

The specific vaccine recommended depends upon the region and prevalence of diseases in local cattle, sheep, and goats. Basically, it is recommended that *Clostridium perfringens* types C and D toxoids and *C. tetani* toxoid be given. Vaccines for other diseases may better be administered shortly before weaning. If leptospirosis is a problem in the area, a multiple serovar bacterin should be given. In rabies-endemic areas, a killed rabies vaccine should be given, and anthrax spore bacterins are indicated in certain areas. Crias should be given only one-fourth to one-half the dose of anthrax spore vaccine recommended for the adult (Chapter 7).

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[a] Harris flush tube, K 45, 24 French; Pharmaseal Toa Alta, Puerto Rico 00758.

22

Congenital/Hereditary Conditions

Congenital problems of llamas and alpacas occur more frequently than in livestock, companion animals, horses, and humans. The emotional trauma associated with the realization that an animal has a congenital defect should prompt veterinarians providing service to owners/breeders to give these problems special attention. Misconceptions and misunderstanding are prevalent. Unfortunately, little factual information is available to guide a breeder in managing a breeding program. Yet much is known about similar or identical conditions in livestock. While direct extrapolation to camelids may be unacceptable, the principles of herd management are the same for all ungulates.

Though not all congenital conditions are of equal importance, clients should be advised of the presence of any defect and provided with information on the potential impact on breeding and performance. It is then the responsibility of the client to determine the disposition of the animal.

This chapter surveys the entire problem of congenital anomalies. Information is presented about various causes of such anomalies, and special attention is focused on potentially hereditary conditions. Reported information is sparse; therefore, veterinarians should participate in the information-gathering process. Keeping detailed records is vital to (1) obtaining crucial information for the benefit of the llama/alpaca industry and (2) providing protection to the veterinarian in the event that the animal is sold or has defects that affect a future breeding program. It is important to properly identify the animal and, if possible, obtain information on genealogy.

Illustrations for some of the defects are found in the organ system chapters.

Medicine and Surgery of Camelids, Third Edition by Murray E. Fowler

TERMINOLOGY

Various terms used to describe certain conditions are often erroneously used interchangeably by breeders and veterinarians. A congenital condition is one that is present at the time of birth. Unfortunately, some congenital defects may not be readily apparent at the time of birth. In humans and livestock, certain biochemical defects may not become apparent until later in life. Conformation characteristics may not become apparent until after the cria has grown. Yet, in these instances, the foundation for the characteristic is present at the time of birth.

A hereditary condition is genetically transmitted from parent to offspring. The manifestation of the condition may be present at the time of birth or develop subsequently. The term "genetic" is frequently used interchangeably with "hereditary," but they are not synonymous. Certain genetic disorders may cause serious defects in a single individual, but the disorders are not passed on to subsequent generations. However, reproduction may be impossible because of the nature of the defects.

CAMELID GENETICS[19,39,62,89]

All camelids have a diploid chromosome number of 74.[7,10.116] Fertile hybrids have been produced among all four species of South American camelids (SACs).[24] Bactrian and dromedary camels interbreed. In Israel a cross between a male alpaca and a female dromedary by artificial insemination produced a stillborn full-term fetus. In the United Arab Emirates, scientists have succeeded in producing four living OWC and NWC offspring (two males, two females). It is yet to be determined if the hybrids are fertile.[107,108]

There are three pairs of submetacentric autosomes and thirty-three pairs of acrocentric autosomal chromosomes in camelids. The X chromosome is the largest submetacentric chromosome, and the Y chromosome

Glossary of selected terms[79,96]

Allele: One of several different forms of a gene. Slight differences may produce changes in the end product of gene function (eye color, hereditary diseases, resistance to a microorganism).

Biodiversity: The sum total of all life on earth.

Bioinformatics: The process of using a computer to search through massive biological data bases.

Chromosome: A linear or circular strand of DNA that contains genes. Each animal has a specific number of paired chromosomes; in the case of camelids, 37, or as usually written, 2n = 74.

Congenital defect: Abnormalities of structure or function that are present at birth. Not all of these are genetic defects, since other physical, chemical, and infectious agents may affect the fetus.

DNA: An acronym for deoxyribonucleic acid. It is the genetic material that comprises the genes, chromosomes, and the genome. DNA is in the form of a double helix (spiral) as reported by Watson and Crick in 1953.

DNA fingerprinting: Much like the fingerprint used in human identification, but done with the unique DNA characters for each individual animal. Commonly used in forensic medicine (crime solving).

DNA sequencing: Establishing the anatomy of DNA by chemical analysis

Embryogenesis: A complex, marvelously integrated process. The wonder is that the majority of offspring are normal. Many factors may influence the well-being of the fetus, and numerous agents other than genetic factors may disrupt organogenesis. The science of **teratology** ("monster" in Greek) deals with overall birth defects. A **teratogen** is any agent that causes abnormal development of the fetus. The furor over inadequate testing of drugs that may be prescribed for pregnant human women was spawned by the thalidomide disaster of the 1970s. Both physical and chemical effects on the fetus were known before that time, but now an entire discipline of medicine and biology deals with such topics. **Teratogenesis** is the process by which teratogens exert their effect.

Gene: The fundamental unit of heredity; a specific section of DNA within a chromosome; the unit of information in DNA that specifies the translation of a particular protein. Mammals have 20,000 to 35,000 distinct genes in their genome.

Genetic defect: Sometime called a hereditary defect. Certain genetic disorders may cause serious defects in a single individual, but the disorders will not be passed on to subsequent generations.

Genetic diversity (variation): Variation that occurs in a group of interbreeding organisms (camelids) by the frequency of alleles appearing in a population or the frequency of genotypes.

Genetic engineering: The direct manipulation of genes to alter the physical appearance of an animal.

Genotype: The entire genetic makeup of an individual.

Genome: A full-length copy of an individual's genetic endowment. A genome is the sum total of all the genes, DNA, and genetic information, neatly compiled in two distinct copies (one from each parent), in every cell of the body.

Hereditary defect: A defect that is passed from one generation to the next by the parents.

Karyotype: A microscopic picture of the chromosomes.

Mendelian genetics[52]: Simple inheritance based on dominant and recessive traits that segregate according to mathematical ratios. Gregor Mendel was an Austrian monk who used plant breeding and direct observation to establish the ratios. He is considered the father of modern genetics.

Microsatellite: A stretch of DNA that is repeated several times in a row. All mammals examined so far have 100,000 to 200,000 such repeats. These are located at random throughout a chromosome. The variation in these markers between individuals allows for parentage verification and is a tool in the forensic community for matching blood and semen left at a crime scene. These microsatellite markers are given names and numbers.

Molecular biology: The study of the biochemical and biophysical aspects of the structure and function of genes and other sub-cellular entities.

Phenotype: The observable expression of the genotype of an individual (structure, color).

Recombinant DNA technology: A process of finding a gene on a chromosome, snipping it out of its original location, and inserting it into a new location (another organism). Currently used in the production of safe and efficient vaccines for animals and humans.

is a very small acrocentric chromosome. Some confusion over the classification of camelid chromosomes has arisen among investigators, perhaps because of variations in staining procedures and evaluations at different phases of meiosis.[10] Chromosome banding patterns and nucleolus organizer regions have also been identified, but a complete discussion of karyology is beyond the scope of this book.

A milestone of camelid genetics was the bringing to fruition the alpaca genome project.[13,14,29,47,52,70,88,120] The alpaca was selected for genomic sequencing of nuclear DNA by a committee of scientists from the United States National Cancer Institute, Laboratory of Genomic Diversity, in cooperation with other scientists from Harvard University, Massachusetts Institute of Technology, and Washington University.

The alpaca joins other species that have had their genomes sequenced, namely, humans, mice, rats, dogs, cats, horses, cows, and several insects. The human genome project took sixteen years to complete at a

cost of $3 billion (U.S. dollars). The alpaca genome project only took two years and at a fraction of the cost. Mitochondrial DNA has been sequenced in Bactrian camels.

Genomic research was fostered by a knowledge of the chemical structure of DNA as described by Watson and Crick in 1953 to be a double helix. The development of the computer was a necessity, with mainframe computers developed during the late 1940s and early 1950s. Personal computers came along in the early 1980s. Finally, instrumentation to automate DNA chemical analysis coupled with software to manipulate the voluminous data has made it possible for the work to go forward.

Genomic information is of inestimable value to camelids and their owners and managers. It will aid in solving some of the congenital disease riddles. Of particular relevance to camelid medicine is the opportunity to determine which gene(s) controls resistance or susceptibility to infectious or parasitic diseases. The ability to manipulate genes will enhance the production of vaccines. The ultimate goal is to improve herd management, which will in turn improve the well-being of camelids throughout the world.

TERATOGENESIS

Etiology

The causes of congenital/hereditary defects are manifold (Table 22.1). Genetic factors are discussed at length, and the effects of infectious diseases deserve special mention. Although no specific congenital defects caused by infectious agents have been reported, it seems likely that they occur. Consider a few examples from other species.

A number of virus infections are teratogenic in humans, cattle, sheep, goats, swine, cats, and ferrets. The ultimate effects on the fetus are determined by the species involved, strain of the virus, and stage of pregnancy at the time of exposure to the teratogen. Bovine virus diarrhea virus (BVDV) has caused cerebellar dysplasia, ocular defects, inferior brachygnathia, alopecia, internal hydrocephalus, and impaired immunologic competence in calves and lambs.[64]

Bluetongue virus (BTV) has been shown experimentally to cause central nervous system defects (hydrocephalus, cerebral hypoplasia, dysplastic spinal cord), retinal dysplasia, and arthrogryposis in lambs. Exposure of pregnant heifers to BTV resulted in abortion, arthrogryposis, prognathia, and a "dummy calf" syndrome.[64] It is important to note that modified live virus (MLV) BTV vaccines may also exert teratogenic effects on the fetus of the pregnant ewe. The use of any MLV vaccine in any species other than those for which the vaccine was prepared is hazardous.

Both hog cholera virus and swine influenza virus are teratogenic. Feline panleukopenia virus (FPLV) causes cerebellar hypoplasia in kittens and ferrets.[64] It is interesting that mature ferrets are refractory to overt infection with FPLV, yet teratogenesis occurs. In humans, examples of teratogenesis include congenital syphilitic blindness and congenital deafness from prenatal infection with German measles virus.

Chemically induced teratogenesis is being intensively studied in humans, livestock, and laboratory animals. No chemically induced teratogenic defects have been identified in camelids. However, such effects are known to occur in all other species studied, so it should be expected that chemical teratogenesis will ultimately be identified in camelids. Some congenital defects identified in camelids are induced by chemical teratogens in other livestock species. It should be noted that these defects are also known to be inherited traits in one or more species (Table 22.2). Veterinarians should investigate both possibilities when congenital deformities occur.

Some general principles should be understood: (1) The degree of susceptibility to the effects of a teratogen is determined by the genotype of the animal. Not all species are equally affected. (2) The teratogen, to affect the fetus, must pass through the placenta in the metabolically active form. (3) The nature of the deformity is dose dependent. High doses of a certain teratogen at a critical time result in resorption. Slightly lower levels result in dead, deformed fetuses; still lower levels in living, deformed fetuses; and at the lowest levels in normal, live offspring. (4) The fetus must be exposed to the teratogen at a specific period during gestation. Knowledge of embryology and, especially, the time and sequence of organogenesis, is fundamental to understanding teratogenesis. (5) Chemically dissimilar teratogens may produce identical effects on the fetus.

Table 22.1. Etiology of birth defects.

Genetic
Mutant genes
Familial characteristics
Chromosomal aberrations
Infectious agents damaging fetus
Physical effects on the fetus
Trauma
Hyperthermia
Irradiation
Chemical
Drugs
Poisonous plants
Malnutrition
Excesses
Deficiencies

Table 22.2. Congenital conditions in lamoids and their inheritability in other domestic animals and humans.

Condition	*Bovine*	*Equine*	*Ovine*	*Caprine*	*Porcine*	*Canine*	*Feline*	*Human*
Skeletal								
Ankylosis, carpus		U						
Angular limb deformity								
Carpal valgus		S	S					Y
Carpal varus			U					
Femorotibial valgus								
Metacarpophalangeal valgus								
Arthrogryposis	Y	Y	Y		Y			Y
Femur, shortened							Y	
Hemivertebra	S				Y	U		
Spinal agenesis								
Metacarpal, shortening						U	Y	
Patella, medial luxation						Y		Y
Polydactyly	Y	U	Y	Y	S	Y	Y	Y
Scoliosis	U		U					Y
Syndactyly	Y		U		U	Y		Y
Tail, agenesis			U		U	U	Y	
Talus, vertical								
Tendon contracture								Y
Carpus			U					
Stifle			U					
Head/face								
Cerebellar hypoplasia	Y	S	S		U	S	Y	Y
Choanal atresia								Y
Cyclopia	U	U	U	U	U		U	U
Encephalomeningocele			U				U	U
Facial bones								
Agenesis								
Lateral deviation		U						
Hydrocephalus, internal[a]		U	U		S	Y	Y	Y
Mandible								
Brachygnathia	Y	Y	Y	Y	U	Y		
Micrognathia			Y		U			
Prognathia		Y				Y		Y
Maxilla								
Brachygnathia					U	Y		
Prognathia		Y				Y		Y
Nares, agenesis								
Nasal passages, stenosis						U		
Palate								
Agenesis								
Palatoschisis (cleft)	Y	U	Y		U		Y	Y
Teeth, retention of deciduous								
Reproductive system								
Cervix								
Agenesis								
Double	Y							
Fallopian tubes, segmental agenesis	Y				U			Y
Hymen, imperforate	S				U			Y
Intersex, pseudohermaphrodite	U	Y	Y	Y	Y	U	U	U
Ovary								
Agenesis	Y						U	Y
Hypoplasia	Y						U	
Penis								
Corkscrew	U							U
Curvature								
Hypoplasia	U	U						U
Persistent frenulum	Y				U			

Table 22.2. ***Continued***

Condition	*Bovine*	*Equine*	*Ovine*	*Caprine*	*Porcine*	*Canine*	*Feline*	*Human*
Testes								
Cryptorchidism	Y	Y	Y	Y	Y	Y	U	Y
Cystic structures								
Ectopic	Y						U	
Hypoplasia	Y		Y				U	Y
Twinning	Y	Y	Y	Y				Y
Uterus								
Segmental agenesis	Y				U		U	
Unicornis								
Vagina, segmental agenesis	Y		U				U	Y
Digestive system								
Atresia ani	Y	U	Y		Y			Y
Atresia coli	U	U						Y
Megaesophagus		U				S	S	
Pyloric stenosis								
Cardiovascular								
Atrial septal defect		U			U	U	U	U
Aortic arch, persistent, right[b]		U				Y	U	
Ductus arteriosus, patent		U			U	Y	S	Y
Portocaval shunt								
Tetralogy of Fallot	U	U				Y	U	U
Transposition of great vessels		U			U		U	
Ventricular septal defect	Y	U	Y		U	Y	U	U
Eye								
Blindness, cause not determined	U					Y		
Cataract[c]	Y	Y				Y	Y	Y
Ectropion		U				U		
Entropion	Y	U	Y		U	U	U	Y
Eyelid, hypogenesis							U	Y
Iris, nonpigmented (glass)	Y	U			Y	Y	U	
Miscellaneous								
Dwarfism	Y	Y	Y	Y	Y	U		Y
Ears, short				Y	S			
Hernia								
Diaphragmatic		U	U		S	Y	Y	Y
Inguinal	U	U			Y	U	U	
Umbilical[d]	Y	Y	Y		Y	Y	Y	U
Renal agenesis	U	U	U	U	U	S	U	Y
Polythelia, supernumerary teats	Y	Y	Y	U	Y	U	U	Y
Teat agenesis								
Toenails, crooked	Y	Y	Y		Y			
Urachus, patent	U		U		U		U	

Sources: Bovine, Leipold et al. 1983; equine, Huston et al. 1977; ovine, Dennis and Leipold 1979, Saperstein et al. 1975; caprine, Leipold and Dennis, 1984; porcine, Huston et al. 1978; canine, Erickson et al. 1977; feline, Saperstein et al. 1976; human, Elsas and Priest 1979, McKusick 1986.

[a] Brown 1973.

[b] Kolaczkowski and Soboncinski 1971.

[c] Ingram and Sigler 1983.

[d] Fowler 1987.

Note: Y = inheritance confirmed; S = inheritence suspected; U = occurs but etiology unknown; blank = no information.

Table 22.3. Plants with known teratogenic effects in livestock.

Plant name		*Species affected*	*Stage of gestation, days*	*Type of defect*
Scientific	*Common*			
Astragalus spp. *Oxytropis* spp.	Locoweed	Cattle, sheep	1–100	Flexure of carpus, abortion
Conium maculatum	Poison hemlock	Cattle	40–70	Arthrogryposis
Datura stramonium	Jimson weed	Swine	60–120	Arthrogryposis
Leucaena leucocephala	Koa haole, lead tree	Swine, rats		Fetal resorption, polypodia
Lupinus spp.	Lupine, blue bonnet	Cattle	40–70	Arthrogryposis, scoliosis, cleft palate
Nicotiana tobaccum	Tobacco	Swine, rats		Twisted limbs, dorsal flexure of hind digits
Sorghum vulgare	Sudan grass	Horses		Carpal ankylosis
Trachymene cyanantha	Wild parsnip	Sheep		Crooked legs
Veratrum californicum	False hellebore, corn lily	Sheep, SACs	14	Cyclopia

Poisonous plant ingestion by a pregnant camelid is an ever-present hazard to the fetus.[54] Camelids are fastidious in their eating habits, rarely consuming large amounts of strange plants, but they do investigate and try new plants. A low-dose intake may be a saving factor in camelids. Table 22.3 lists plants known to produce teratogenic defects in livestock.

The presence of a birth defect is vivid and alarming to the breeder. However, of perhaps even greater importance are the effects that chemical agents may have on the reproductive process without obvious outward expression. Teratogens may have a direct effect on ova or spermatozoa and cause infertility. High doses early in gestation may cause fetal death with resorption or undetected abortion. Lethal effects may be the result of maternal ingestion early in gestation, even though fetal death occurs late in gestation or postpartum. Nonlethal effects may either prevent reproduction or allow reproduction of constitutionally unsound individuals that may be highly susceptible to other diseases.

HEREDITARY TRAITS[21,25,65,67,100,123]

Hundreds of anatomic and physiologic traits are passed from parents to offspring by gene pairing.[11,94,99] More than 20,000 gene loci have been mapped on human chromosomes,[16,69] but only nineteen in cattle,[66] sixteen in sheep,[18,99] eleven in horses,[42] and five in pigs.[43,63] Llamas and alpacas have received attention by both South American[30] and North American[30,36,56–58,71–73] investigators, but definitive genetic studies have not been conducted and reported. A detailed discussion of coat color determination is beyond the scope of this book.[117,118]

Body conformation is also an inherited characteristic and is discussed in detail in Chapter 24.[16] Twinning is rare in camelids.[28,83]

Chromosomal Aberration

A number of chromosomal abnormalities have been reported in domestic animals. These defects occur during meiosis and include fusion of chromosomes, translocations of segments of chromosomes, loss of a segment, and other changes.[55,104] The expression of the defect is determined by whether or not the change occurred on an autosomal pair or one of the sex chromosomes. Chromosomal aberrations may affect a single individual or be perpetuated as inherited characteristics. Chromosomal aberrations may be identified by a combination of family pedigree analysis, identification of interspecific somatic cell hybrids, and cytogenetic studies, including karyotyping and various banding staining.

Genetic studies have been developed to the stage of highly technical, submolecular/biochemical complexities that are far beyond the scope of this work. Camelid inheritance is still in the descriptive stage. Although most of the congenital defects reported in camelids are known to be inherited in one or more species of other domestic animals or humans, it is important to recognize that many may also be produced by other etiologic agents, as listed in Tables 22.1 and 22.2. In fact, such defects as arthrogryposis are more likely to be caused by exposure of the dam to a toxic substance at a crucial time during gestation than by genetic damage (Table 22.3).

Nonetheless, prudent llama/alpaca breeders and veterinarians should consider the prevalence of certain defects in particular camelid bloodlines and in multiple environmental situations. A veterinarian should not be dogmatic but should educate and assist.

Detection of Inherited Traits[53,55,77,114]

The detection of an inherited trait depends on the mode of inheritance. If a characteristic is dominant,

Table 22.4. Number of matings required to exclude carrier status of a male SAC for a simple recessive defect.

	Probability of error	
	5%	*1%*
Male mated to females that are		
Homozygous aa	5	7
Heterozygous Aa	11	16
Mixed: 50% Aa, 50% AA	23	35

Source: Modified from Hamori 1983, p. 47.

Table 22.5. Expected expression of a phenotype among the offspring of parents having a recessive trait.

AA × Aa = 0
Aa × Aa = 25%
Aa × aa = 50%
Aa × aa = 100%

Source: Modified from Hamori 1983.
Note: AA = homozygous (free from trait), Aa = heterozygous (carrier), aa = homozygous (affected).

one of the parents will be phenotypically positive, and generally, at least 50% of its offspring will express the phenotype. However, even though a characteristic may be dominant, environmental or genetic factors may affect the degree of expression of a phenotype.

The majority of inherited defects are recessive, and both parents must contribute the gene for the offspring to exhibit the trait. Recessive traits may be simple, in which only one gene is involved, or multifactorial, which complicates expression and detection in a population.

A carrier male and female will produce 75% phenotypically normal offspring, but 50% of their offspring will be carriers. Veterinarians dealing with suspected inherited traits in camelids should discuss this thoroughly with owners.

The diagnosis of an inherited trait is a laborious, costly, time-consuming process. Familial repetition is the most important information necessary, and this requires detailed genealogy of both normal and abnormal offspring. Statistical evaluation of familial data is frequently required.

The ultimate evaluation must be based on breeding trials. No such trials have been reported for camelids, but camelid owners may consider the studies conducted in artificial insemination (AI) establishments for cattle. Table 22.4 provides a list of the number of matings required to exclude carrier status of a male for a simple recessive defect.

Breeding in such numbers is possible in an AI dairy stud, where selected sires can be test mated with cows known to be homozygous or heterozygous carriers, but this is difficult or impossible on the working ranch. Another method is to test breed sires on daughters, because, if heterozygosity is suspected, at least 50% of the male's daughters should also be heterozygous. This method requires twenty-three matings to detect a recessive gene in the male.

It is both expensive and time-consuming to conduct such test breedings in a male, and it is impossible to test breed a female to detect a recessive carrier state. Reversing the sex status of animals in Table 22.4, if the female were mated to (aa) males, five births would be required, and if bred to (Aa) males, eleven births. Such proof is not likely to be forthcoming in camelids.

Detection of the carrier state of a simple recessive trait in camelids by breeding trials is unlikely; therefore, other methods must be used to identify and eliminate carrier animals from a breeding program. This may be possible using information and practices developed in cattle and sheep breeding.

Consider a hypothetical simple recessive trait (Table 22.5). Homozygous AA individuals are completely free of the trait, and homozygous aa individuals express the phenotype. While heterozygous (Aa) individuals do not express the phenotype, they may not be as healthy as an AA individual in performance characteristics such as milk production, fertility, growth, mothering ability, and disease resistance, all factors that may be evaluated in livestock.

Furthermore, in livestock, a number of biochemical defects such as afibrinogenemia may be measured in the laboratory. Aa individuals for this trait show an intermediate level of fibrinogen that can be detected by serum analysis.

The problem in the llama/alpaca industry is that no biochemical defects are known; while basal blood parameters are reported, standards have not been established for milk production, growth, mothering ability, etc. Nonetheless, these standards can be developed. It is important that responsible breeders begin to use the basic principles of animal breeding developed with livestock.

Cytogenetic studies should be carried out and karyotypes and banding staining should be done. Detailed, sophisticated biochemical standards must be established.

BREEDING MANAGEMENT SYSTEMS[63]

It is inappropriate to discuss all of the various breeding management systems in a medical text such as this. Some of the systems have profound influence on the prevalence of congenital defects; therefore, veterinarians and breeders would be well advised to consult contemporary books on the subject.[45] Inbreeding is a mating system in which the progeny produced

by parents are more closely related than the average of the population from which they come. Father-daughter, brother-sister, grandfather-granddaughter, and other close relationship breedings have been carried out with llamas both intentionally and unknowingly.[82] The parentage of some llamas is unknown, and only recently has it become possible to verify parentage in camelids as is routinely done in cattle and horses.[27,86,87] This is discussed later. Linebreeding is a form of inbreeding in which an attempt is made to concentrate the inheritance of one ancestor or ancestral line in a herd.

Inbreeding increases homozygosity and is used in livestock breeding to strengthen a given characteristic. Unfortunately, it can also concentrate undesirable traits. Inbreeding is a technique that should be practiced only by highly skilled and experienced breeders who are willing to cull (not sell) individuals exhibiting undesirable traits.

In general, inbreeding is followed by a decline in traits closely related to physical fitness such as fertility, mothering ability, viability, and growth rate.[94] Detailed records were kept on a herd of inbred dorcas gazelles (*Antidorcas gazellei*) at a zoo. As the inbreeding coefficient increased, so did the neonatal mortality rate. The calves died of inanition, weakness, white muscle disease, and a variety of other infectious and noninfectious diseases. The neonates lost their "coping" ability.[94] Breeders should be fully cognizant of the ramifications of the practice of inbreeding.[33,81]

CONGENITAL CONDITIONS[49]

Table 22.2 lists more than 100 congenital conditions of SACs that have been identified by the author,[24,28] reported in the literature,[1,6,8,17,23,34,37,48,54,63,66,111,121] or reported by personal communications. The inheritability of similar conditions in humans and other animal species is indicated, if known. The reader is again reminded that these conditions may have other than hereditary causes. Some of the conditions are discussed in detail because of their importance to the llama/alpaca industry. Others are simply listed. It may be valuable to attach this table to a clipboard near physical examination forms to be reviewed prior to conducting a health and soundness examination for prepurchase, insurance, or evaluation for breeding soundness.

The list of congenital defects of OWCs is not as extensive as those of SACs. Perhaps this is a matter of under reporting. Defects that have been reported include ventricular septal defects, ocular anomalies, scoliosis, growth retardation, polydactyly, cleft palate, unilateral hernia, atresia ani, and coloboma.[77]

Skeletal Defects of SACs

Angular Limb Deformity

Conformation of the limbs in association with the body is an inherited trait in all animals. There seems to be a high prevalence of crooked legs in llamas and alpacas. Carpal valgus is most prevalent,[16] but carpal varus, metacarpal phalangeal valgus, and femorotibial valgus have also been seen. Similar defects of the long bones of horses and livestock have been reported.

Although there is evidence that some forms of angular limb deformity are familial,[23] all types should not be placed in the same etiologic and diagnostic category. Nutrition is thought to be a factor in some, trauma in others, while in many cases the true cause is unknown. This is also true in the horse. Angular limb deformity is a common sequel to rickets in crias. The cause of the bowing is different (cortical thinning) in rickets, and the radiographic picture is different (Chapter 2).

In carpal valgus, the prominent sign is inward bowing of one or both carpi (Figures 22.1, 22.2A–D). When the defect is allowed to progress, the carpi

Figure 22.1. Carpal valgus.

Figure 22.2A. Carpal valgus in a neonate.

Figure 22.2B. Carpal valgus in a cria.

Figure 22.2C. Severe carpal valgus causing the legs to cross each other

overlap each other when the animal is standing still. Abrasions at the medial aspect of the carpi may be noted from the trauma of the carpi knocking against one another when walking. In one individual, the deviation was so severe that the legs were actually crossed, though when the animal was viewed from the front, the legs appeared to be straight because hair hid the crossed upper forearm and arm. Outward bowing of the carpi is more rare (Figure 22.3). Bowing of the fetlock has been seen (Figure 22.4).

Carpal valgus may be present at birth, but an evaluation should be delayed for a month to allow normal straightening to occur. The degree of deviation may

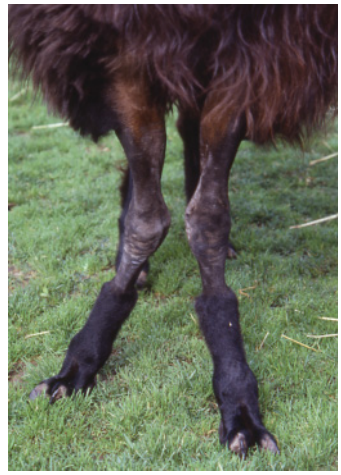

Figure 22.2D. Same animal as 22.2C with legs spread apart.

Figure 22.3. Carpal varus (outward bow).

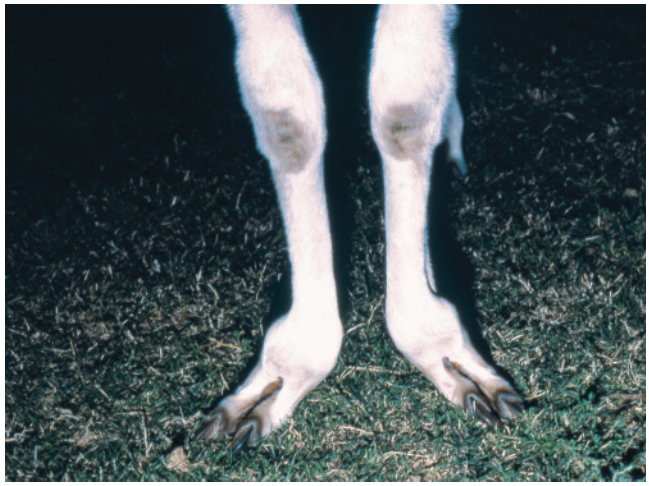

Figure 22.4. Angular deformity of fetlock.

develop at two to six months of age and progressively become more severe, up to fifteen months of age. However, even in cases of development a few months after birth, a spontaneous correction of the deviation may occur as the animal grows.

Radiographs should be taken to evaluate the carpus and contiguous long bones. The ulnar physis is approximately 3 to 5 cm proximal to the radial physis. The ulnar epiphysis extends distally along the lateral radius and attenuates as it becomes a part of the radial epiphysis. Radiographs and anatomic preparations show no physical separation of these two epiphyses in the majority of individuals. However, the author has examined some animals with separated epiphyses.

Variable radiographic changes may be observed in the carpal region. Lesions of metabolic bone disease are rarely observed. Inflammation of the radial physis, characterized by flaring and widening of the physis, may be caused by trauma.

The typical lesion of carpal valgus as seen in radiographs is a wedge-shaped radial epiphysis, with the base of the wedge on the medial aspect of the carpus. The width of the radial physis is variable, but the ulnar physis is flared, doubly cupped in shape, with hyperplasia of the distal ulna (Figure 22.5).

The pathogenesis of carpal valgus appears to be a cessation of growth at the ulnar physis on the lateral aspect of the limb; this allows continued growth of the medial radial physis, producing inward bowing of the limb at the carpus.

The surgical management of angular limb deformity is discussed in Chapter 6. Tubular splints may be applied to the limbs of young animals (less than two months of age) with mild deviation. It is unlikely that any appreciable straightening of the limb will occur after the animal is fifteen months of age, though the physis may not be entirely closed until three years of age.

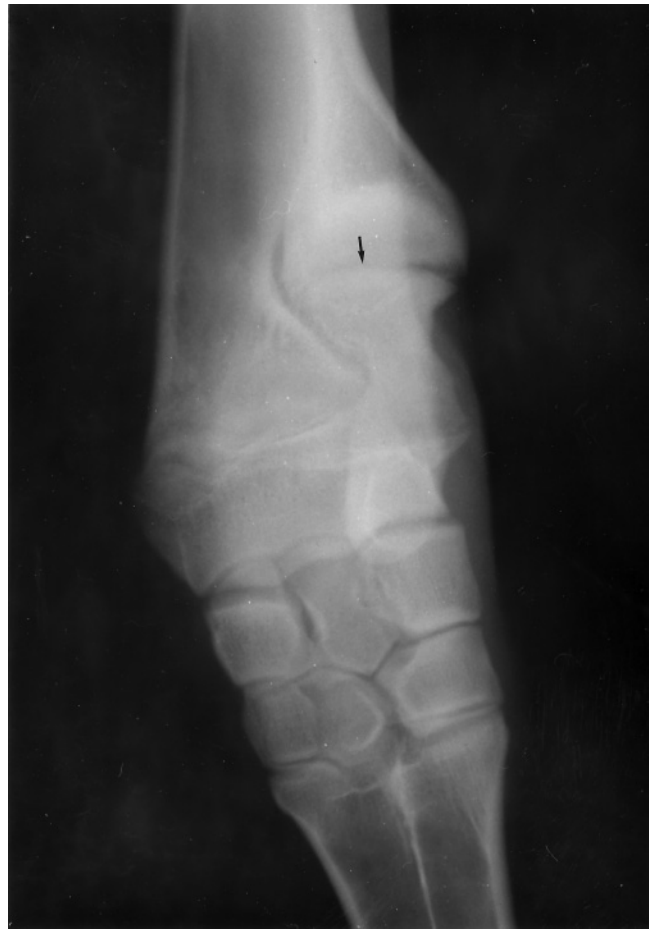

Figure 22.5. Dorsopalmar radiograph of a llama with carpal valgus.

Arthrogryposis

Arthrogryposis is congenital, persistent flexure or contracture of a joint. The bones of the joint are malformed, and their relationship to contiguous bones is distorted to the extent that it is difficult to identify the bones or the joint with radiography. Arthrogryposis may be uni- or bilateral, with single or multiple joints involved. In camelids, the elbow and carpus have been most frequently affected (Figure 22.6), but other joints also have been affected (Figures 22.7, 22.8). Arthrogryposis is frequently present in the neonate with multiple congenital defects (polydactyly, carpal tendon contracture, choanal atresia).

The cause of arthrogryposis in camelids is unknown. Teratogens are frequently involved in livestock (Table 22.3). Affected crias are unable to stand. It is impossible to straighten the limb. The distal segment of the limb may be pointed in any direction or rotated 180 degrees. The joint is usually swollen, but not from the accumulation of synovia. The diagnosis of multiple joint arthrogryposis is obvious, but it should be differentiated from tendon contracture, in which the

bones of the joint are usually properly formed. Trauma should be considered when a single joint is involved.

Surgical correction of arthrogryposis is usually impossible. Euthanasia should be recommended.

Overextension of the Carpus

Overextension of the carpus (calf-knee) may be a serious defect in racing camels (Figure 22.7).

Shortening of Long Bones[112]

Unilateral shortening of the femur and metacarpus has been reported in camelids. The cause is unknown. The animal may compensate and be used as a pet, depending on the degree of shortening.

Luxation of the Patella

The author has dealt with two cases of congenital bilateral medial luxation of the patella in llamas. Others have also observed lateral luxation. Full medial luxation causes the cria to stand in a crouched position (Figure 22.9). The stifle joint is thickened, and the patella is palpated in the medial position rather than

Figure 22.6. Carpal arthrogryposis and polydactyly.

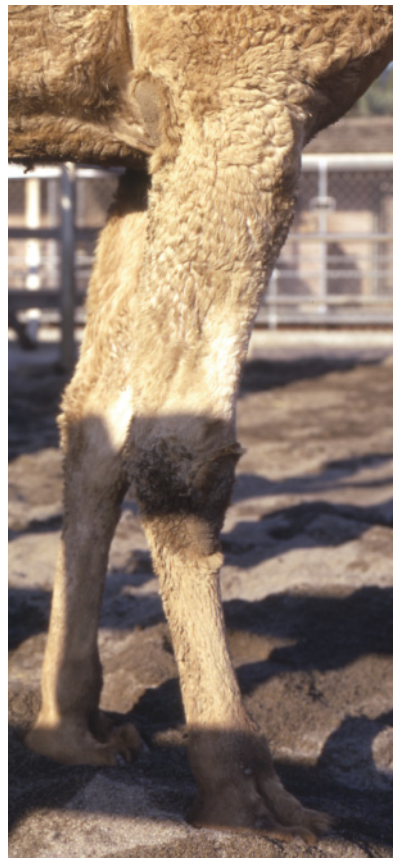

Figure 22.7. Camel with over extension of the carpus (calf knee).

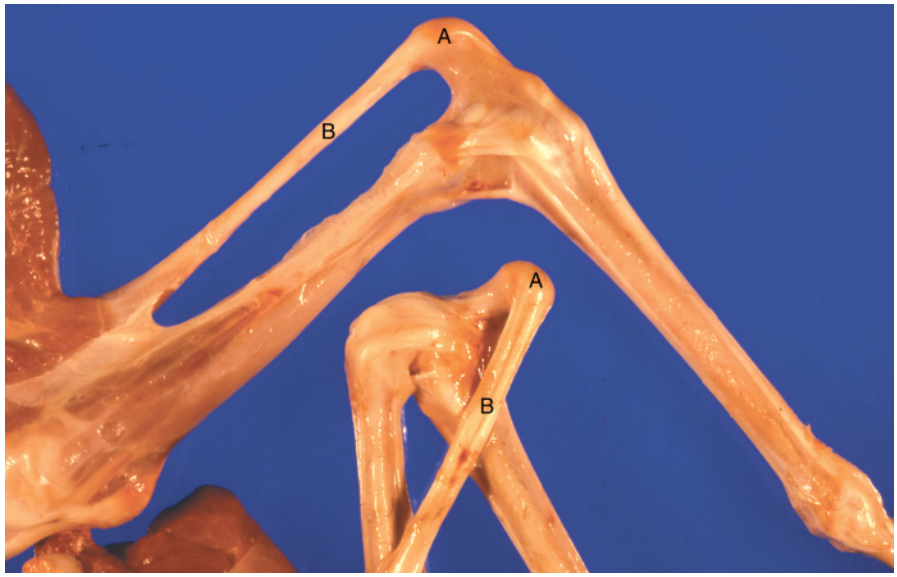

Figure 22.8. Tarsal arthrogryposis. (A) Tuber calcis, (B) tendon of gastrocnemius.

in the dorsal groove of the femur. It is impossible to manipulate the patella to the normal position.

Upward fixation of the patella is an acquired condition in older llamas (Chapter 6). A predisposition may be a congenital conformational weakness (straight rear limbs and laxity of the tendons and ligaments). The mechanisms for upward fixation in a camelid is different than in a horse. The distal patella ligament is a sheet of tendinous tissue rather than one, two, or three discrete ligaments. With laxity of the tibiopatellar and femoropatellar ligaments, the patella may lodge at the dorsal tip of either the medial or lateral ridge of the trochlea.

The prognosis for medial luxation is unfavorable. Surgery was partially effective in one cria, in which the patella was firmly bound medially by shortened collateral patellar ligaments. These attachments were severed, and the crest of the tibia, on which the major mass of the distal patellar ligament was inserted, was relocated more laterally and secured to the tibia with lag screws. The trochlear groove was deepened to provide a better channel for the patella. These are standard procedures, developed for dogs with a similar luxation.

Figure 22.9. Bilateral medial patellar luxation in a llama neonate.

Hemivertebra[119]

Hemivertebra refers to partial agenesis of one or more of the vertebrae. Spina bifida is the ultimate manifestation of this defect. Malformation of the spinal cord is usually associated with spina bifida, which has been reported in a llama.[a] Hemivertebra of the fourth cervical (C-4) vertebra has been diagnosed in association with subluxation of the cervical vertebrae and spinal cord trauma in a juvenile alpaca. Hemivertebrae of the coccygeal vertebrae is one cause of crooked tails.

Clinical signs included ataxia and falling when attempting to avoid capture for restraint. The ataxia was exacerbated by restraint, particularly if the head and neck were manipulated. The diagnosis was confirmed by radiography and myelography. The body of C-7 was only partially formed. There was a marked subluxation between C-6 and C-7 (Figure 22.10). In the dorsoventral projection, the articular facets were missing, allowing even greater instability (Figure 22.11). The stenosis of the neural canal was confirmed

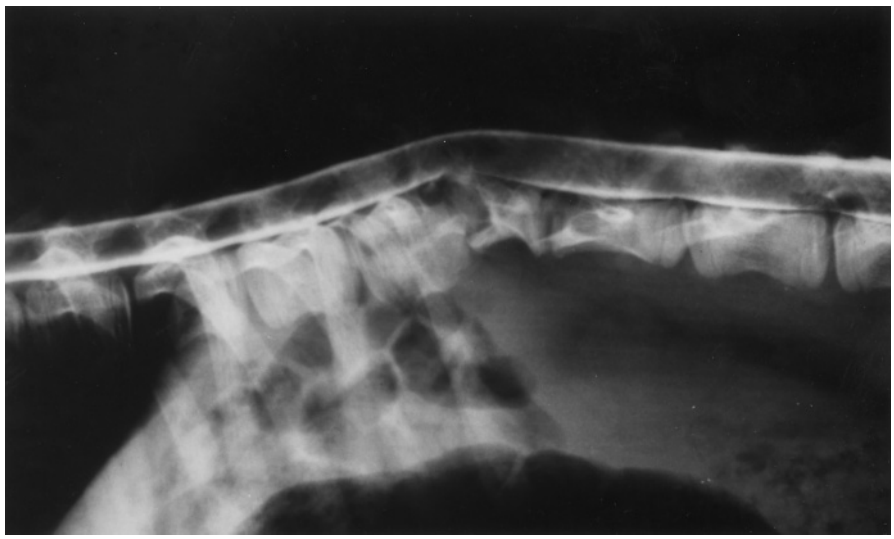

Figure 22.10. Lateral radiograph of hemivertebra of the first lumbar vertebra in a llama neonate. Spinal cord compression is noted by contrast myelography. (Photo courtesy of Dr. Richard Cambre, Denver)

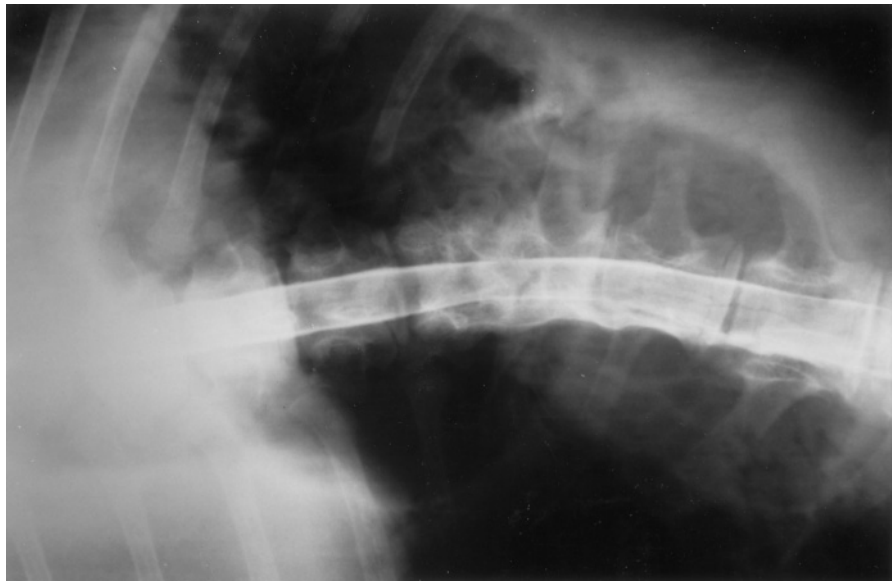

Figure 22.11. Dorsoventral radiograph of hemivertebra of the first lumbar vertebra in a llama neonate. (Photo courtesy of Dr. Richard Cambre, Denver)

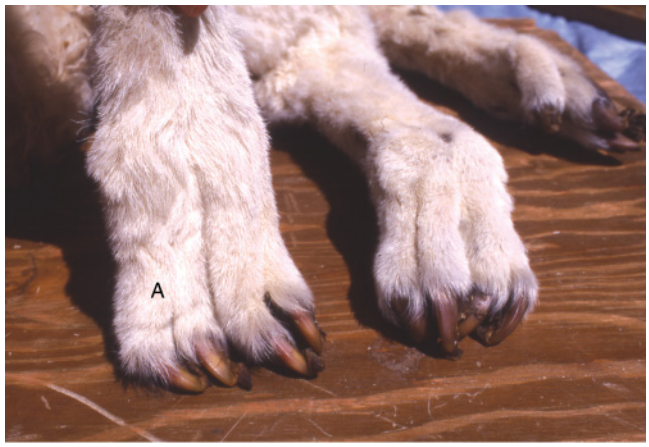

Figure 22.12A. Polydactyly.

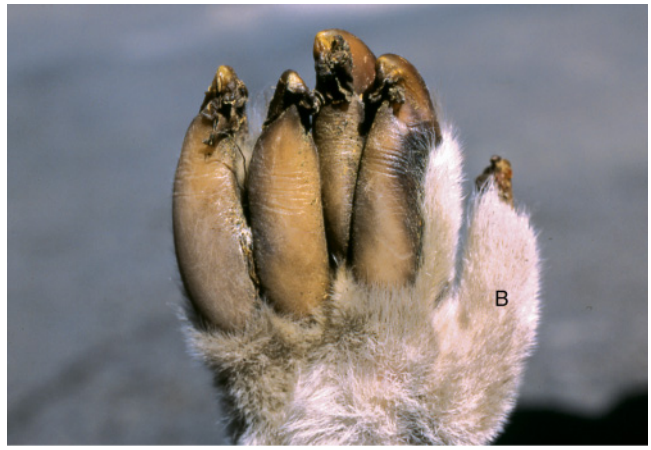

Figure 22.12B. Polydactyly.

by contrast myelography administered via the lumbosacral space.

The anatomic defect was easily identified at necropsy. The spinal cord was flattened at the region of the subluxation, and neuronal degeneration was observed histologically.

Polydactyly/Syndactyly

Polydactylism is a common congenital defect in camelids.[2,115] From one to three accessory digits may occur on one or all four limbs (Figures 22.12A and B, 22.13A and B). Fusion of two normal digits (syndactyly) is less common (Figure 22.14A, 22.14B). Both conditions have been identified as inherited traits in cattle, dogs, and humans. The conditions are evident on clinical examination. The degree of development of the accessory digits varies, but it may be complete, with a full complement of tendons, ligaments, and bones, including metacarpals and metatarsals. Polydactyly is frequently seen in multiple-anomaly situations. The genetics of this trait are not known for camelids, but Figures 22.13A and 22.13B illustrate the trait in a dam and her fetus, suggesting heritability. Some native pastoralists in Peru believe it to be good luck to have a polydactyl animal.

Accessory digits may be removed surgically to allow use of the animal as a pet, but prudent breeding practice should exclude such an animal from further breeding.

Scoliosis[4,50,97,119]

Various forms of curvature of the spinal column occur as isolated incidents (Figures 22.15, 22.16A and B). The etiology is unknown. Wry neck may be trauma

Figure 22.13A. Polydactylae in llama mother.

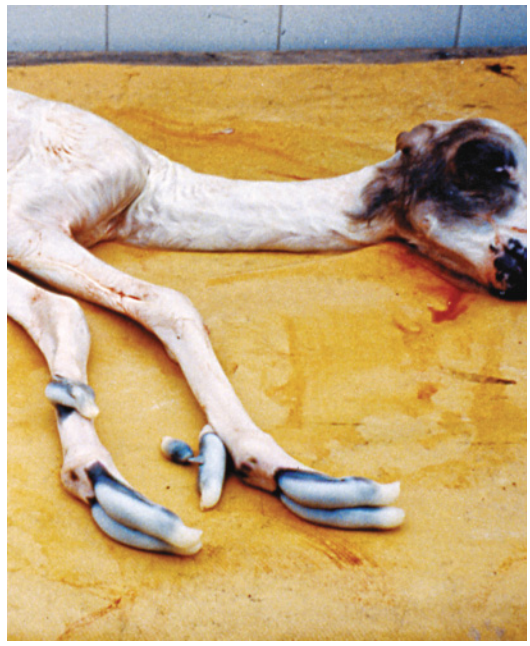

Figure 22.13B. Polydactylae in a fetus from the previous mother.

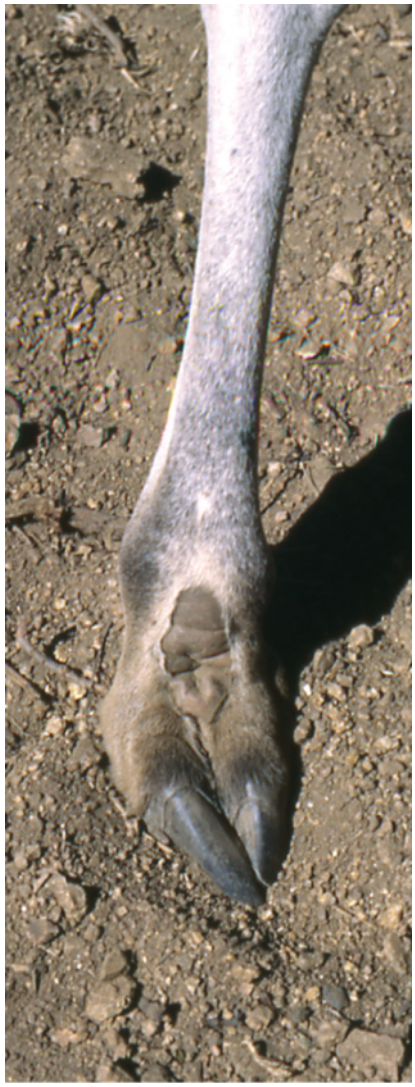

Figure 22.14A. Syndactyly.

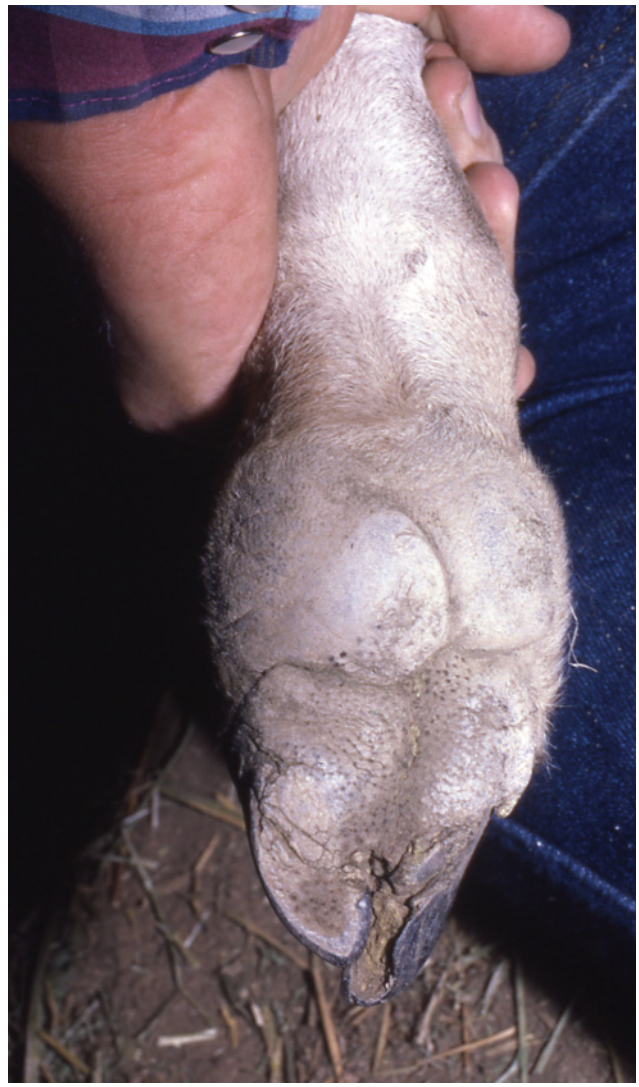

Figure 22.14B. Syndactyly.

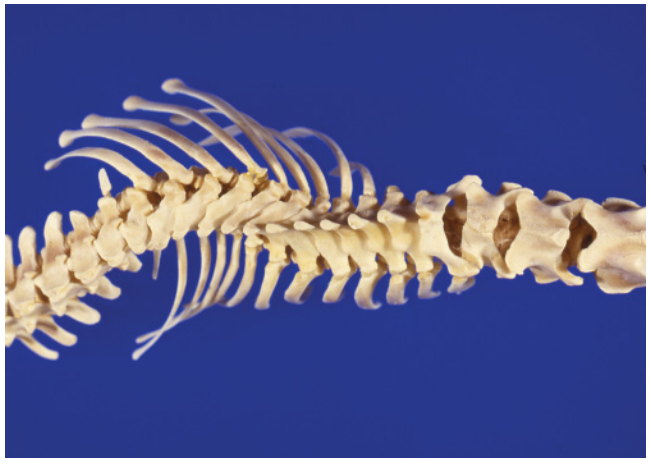

Figure 22.15. Scoliosis.

Figure 22.16A. Scoliosis of thoracic vertebrae in a llama cria.

Figure 22.16B. Scoliosis of the cervical region.

Figure 22.17. Kyphosis in a mature alpaca.

induced from overzealous traction during dystocia. In true congenital scoliosis, changes that indicate permanent distortion of the vertebrae may be seen on radiography. Kyphosis is also seen (Figure 22.17).

Luxation of the Tibiotarsal Bone

Bilateral luxation of the tibiotarsal bone has been diagnosed in a number of SACs, including a seven-month-old female llama. This condition in SACs may be congenital or acquired via trauma.

In an SAC suffering from luxation, the tibiotarsal bone lies in a horizontal rather than a vertical position, and the articular grooves of the tibia articulate with the caudal aspect of the tibiotarsal bone rather than the trochlea (Figure 22.18). Externally, the cranial surface of the tarsus bulges where the tibiotarsal bone projects forward. Movement of the tarsus is restricted, causing a mechanical lameness.

The condition in llamas is similar to congenital convex pes valgus (congenital vertical talus, congenital flat foot, teratologic dislocation of the talonavicular joint) in human infants.[73] The etiology in human infants is unknown, but it is thought to be genetic. Peculiar fetal positioning within the uterus has also been considered. Surgical realignment and pinning of the tibiotarsus may correct the defect in human infants but has not been performed in llamas.

Face and Head Defects

Craniofacial Dysgenesis

A number of congenital defects of the face, nasal cavity, and pharynx (Table 22.2) may be lethal because of the obligate nasal breathing of the cria. The precise relationship between the various defects is unknown. The embryologic development of the nasopharyngeal region is complex, yet it seems logical that these condi-

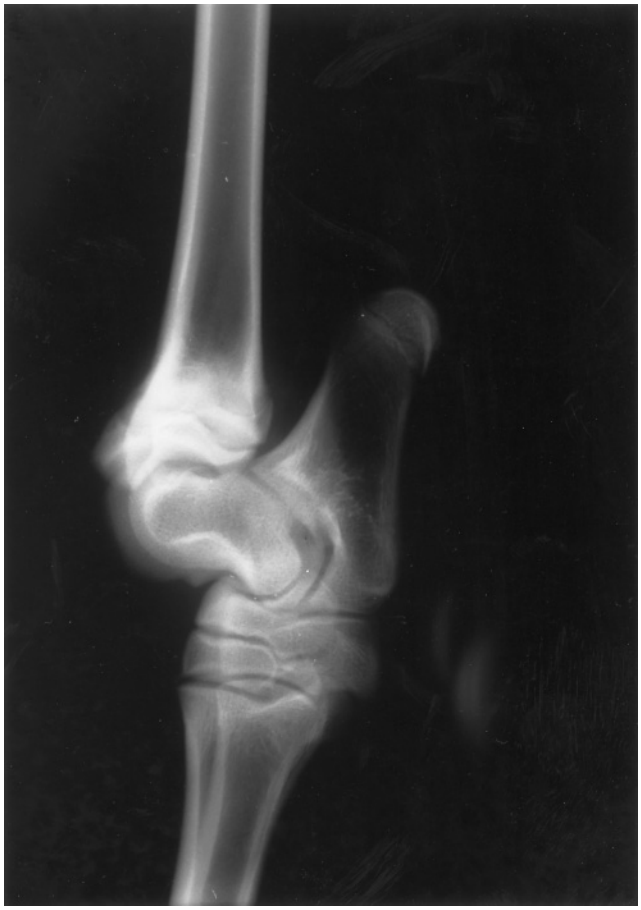

Figure 22.18. Lateral radiograph of tibiotarsal bone luxation. Compare with Figure 11.31.

tions may be the result of interference with development at a particular time.

The least evident defect may be stenosis of the nasal passages. Choanal atresia is common and may consist of a membranous or osseous partition between the nasal and pharyngeal cavities (Figure 22.19).[22,34,55] Agenesis of the facial bones causes variable shortening of the face and muzzle (Figures 22.20A and B to 22.23) and accentuates the doming of the forehead that may be mistaken as a hydrocephalic condition.[1,3,9] In extreme cases, agenesis of the facial bones, along with other tissue dysgenesis, may result in cyclopia. The nares may be totally occluded (Figure 22.20A and B).

The etiology of camelid facial dysgenesis is unknown. Teratogens are known to produce similar congenital defects in sheep. A familial relationship is known in humans with choanal atresia.[20]

Clinical signs vary with the nature of the defect. All produce some impairment of respiration. Complete occlusion of the nasal passageways causes a characteristic breathing pattern in the cria. On inspiration, the mouth is opened slightly and filled with air. Then the lips are slightly closed, while air continues to be sucked into the mouth, ballooning the cheeks. The lips close

Figure 22.19. Flared nostril, open mouth breathing characteristic of choanal atresia.

Figure 22.20A. A Choanal atresia with deformed face.

Figure 22.20B. Nostril agenesis.

Figure 22.21. Craniofacial shortening in a llama cria.

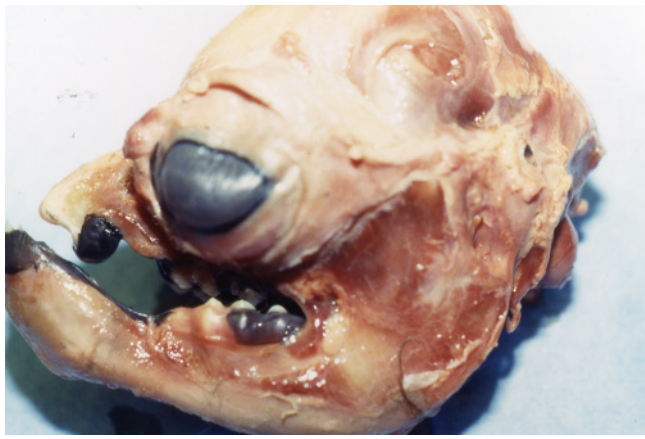

Figure 22.22. Craniofacial shortening in a llama cria.

Figure 22.23. Polydactylae in the cria also having craniofacial shortening.

tightly, and the cheeks compress to force air around the elongated soft palate, which is positioned ventral to the epiglottal cartilage. With expiration, air is forced out of the larynx into the oropharynx, and complete exhalation requires further effort to push the air around the soft palate and out of the mouth.

An affected cria stands with the head extended, because this position restricts air flow the least. Also, from the extended head position it may be possible for the soft palate to flip dorsal to the epiglottal cartilage, allowing free flow of air into the trachea and more normal breathing.

Restricted expiratory airflow entraps excessive air in the pharynx, which may, in turn, be swallowed, causing tympanites. Affected crias not only have difficulty breathing but find it almost impossible to nurse. The time required to obtain sufficient oxygen to sustain life precludes time for swallowing milk. Aspiration pneumonia is a common sequel. Affected crias are known to chew at and ingest fiber from the mother. Numerous hairballs have been observed in compartment 1 of the stomach at necropsy.

Nasopharyngeal obstruction may be partial or complete and uni- or bilateral. Some affected animals are not detected as neonates but have respiratory deficiencies as adults. Such conditions are difficult to differentiate from acquired chronic respiratory diseases.

Definitive diagnosis of these conditions may require radiographs. In a neonate with suspected choanal atresia, a 5-mm catheter should be inserted intranasally. The head should be maintained in an elevated position to prevent back flow of the medium from the nostril; 10 ml of a contrast medium (hypaque) should be deposited into the nasal cavity, followed by immediate exposure of both a lateral and a dorsoventral view (Figures 22.24, 22.25). Both nasal cavities should be evaluated.

The prognosis for the life of a cria with choanal atresia is unfavorable. Surgery has been performed in an attempt to salvage a cria as a pet, but results have been poor (Chapter 6).[59] Euthanasia is a humane alternative.

Jaw Dysgenesis[111]

The most common congenital disorder of llamas/alpacas involves malformation of the mandible or maxilla (Figures 22.26 to 22.31A and B). This is a significant problem within the industry. Overgrowth and protrusion of the incisors are considered by some to be normal, yet the teeth are cut off to improve appearance. Though alpacas have continuously growing incisors, with proper alignment, the teeth are naturally worn off, and overgrowth does not occur. There is little question that the various forms of shortening or elongation of the jaws are hereditary. Genetic transmission is known in other livestock, pets, and humans (Table

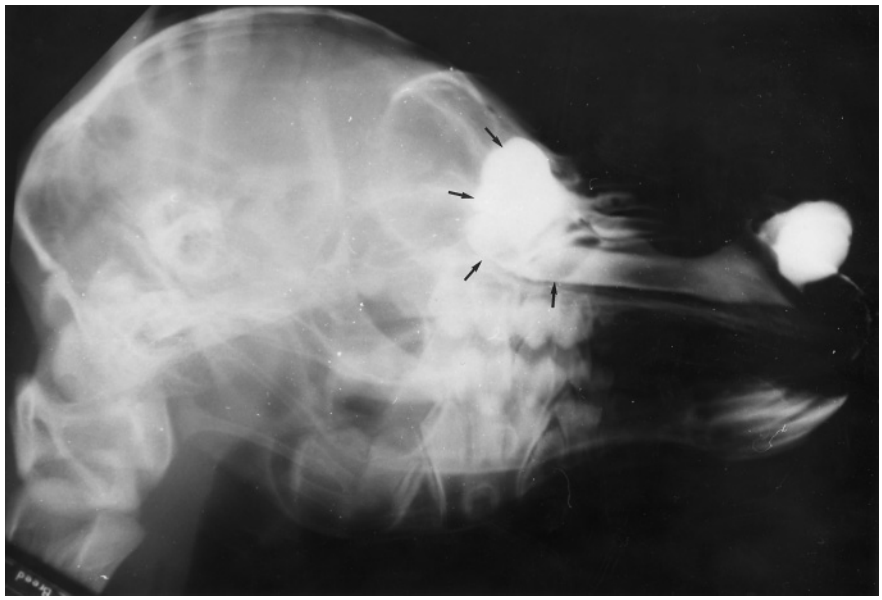

Figure 22.24. Lateral radiograph of choanal atresia, radiopaque liquid instilled into nostril.

Figure 22.25. Dorsoventral radiograph of choanal atresia, radiopaque liquid instilled into nostril.

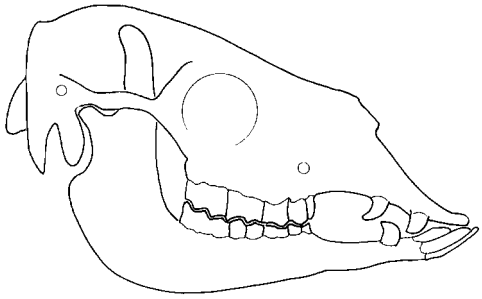

Figure 22.26. Diagram of normal jaw relationships.

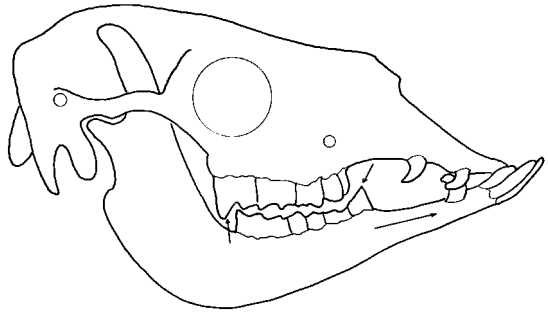

Figure 22.27. Diagram of jaw deformity, elongated mandible.

Figure 22.28. Mandibular hypogenesis in an alpaca.

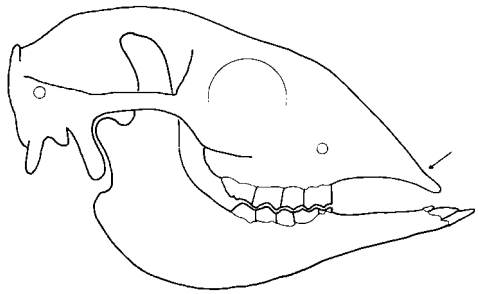

Figure 22.29. Diagram of superior brachygnathism in a neonate, which may prevent nursing.

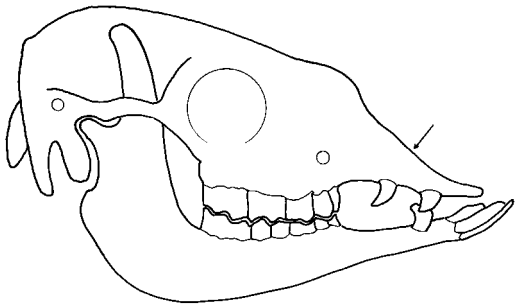

Figure 22.30. Diagram of superior brachygnathism in an adult male llama.

22.2). Peruvian investigators have reported on genetic transmission in llamas and alpacas.[7,70]

Retention of deciduous incisors is common, and a familial tendency is suspected in camelids, but the precise etiology is unknown (Figure 22.32). Other skull defects are illustrated in Figures 22.33 to 22.35.

Dysgenesis of the Palate

Palatal defects of camelids have involved both the hard and soft palates, including clefting or complete absence (Figure 22.36). Cleft palate does not seem to be a part of the choanal atresia/facial deformity syndrome. The etiology is unknown. Clinical signs may

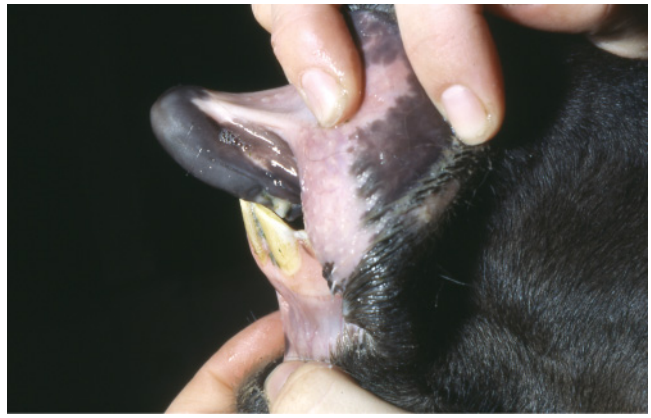

Figure 22.31A. Inferior brachygnathism in an alpaca.

Figure 22.31B. Mandibular prognathism in an alpaca.

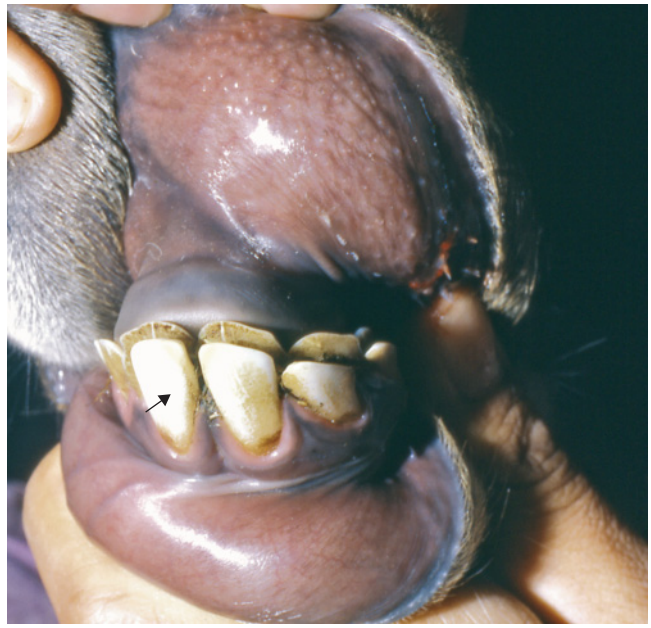

Figure 22.32. Retained deciduous incisors on labial side.

Figure 22.33. Hydrocephalic skull from an alpaca.

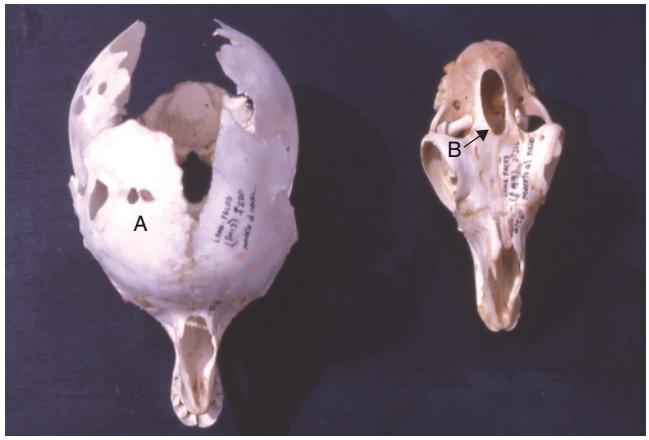

Figure 22.34. Alpaca skull deformities. (A) Hydrocephalus, (B) cerebromeningocele.

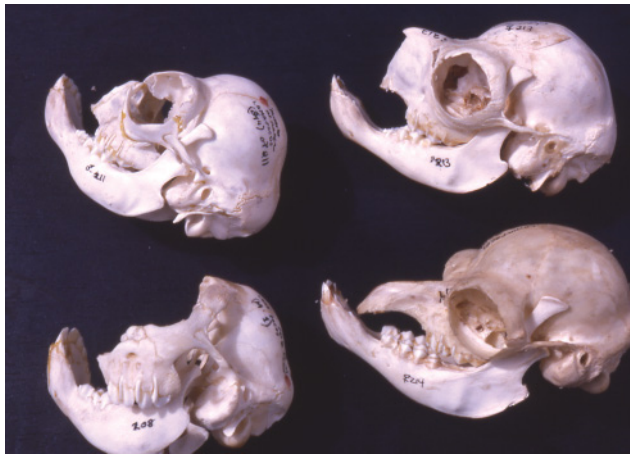

Figure 22.35. Skull deformities in alpacas.

be present at birth, with milk flowing from the nostrils while the cria nurses. With a small cleft, signs may not be evident until solid food ingestion begins and food particles enter the nasal cavity, producing a

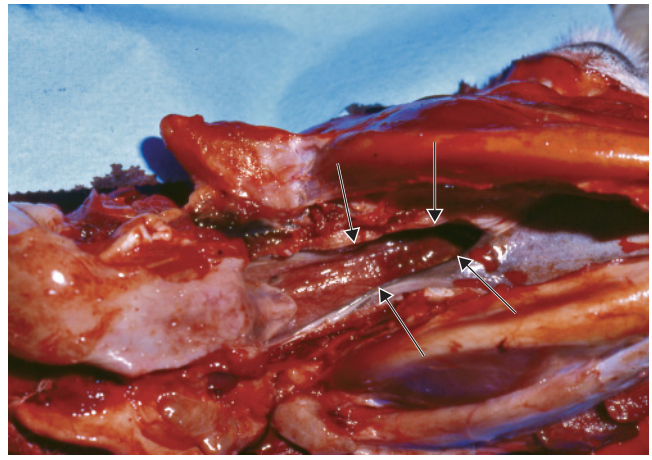

Figure 22.36. Cleft palate in a llama neonate.

Figure 22.37. Nasal exudate containing feed particles, characteristic of cleft palate.

rhinitis (Figure 22.37). A nasal exudate that contains food particles is highly suggestive of a cleft, which may be confirmed by examining the oral cavity. A laryngoscope may aid in visualizing the extent of the defect.

Surgical correction of cleft palate has not been reported. Feeding management is likely to provide only temporary relief, and euthanasia should be recommended.

Reproductive System Defects

Much has been written about congenital/hereditary defects of the reproductive system of llamas and alpacas. Many of the defects seriously impair or prevent reproductive performance. The prevalence of reproductive defects in both North and South America is alarming. In one study in Peru, as many as 10% of the animals had one or more defects.[113] Veterinarians should be aware of the scope of the problem (Table

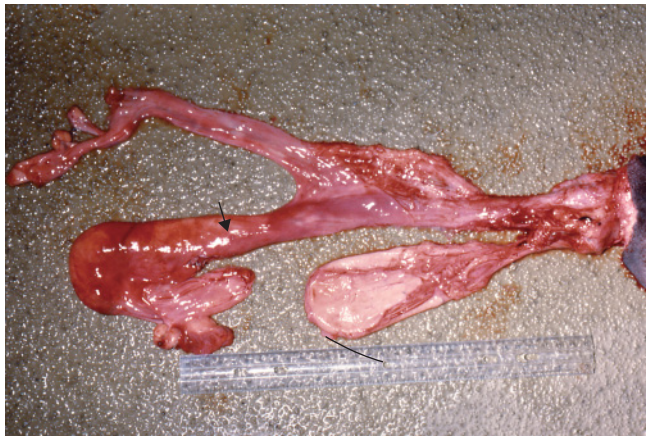

Figure 22.38. Uterus unicornis.

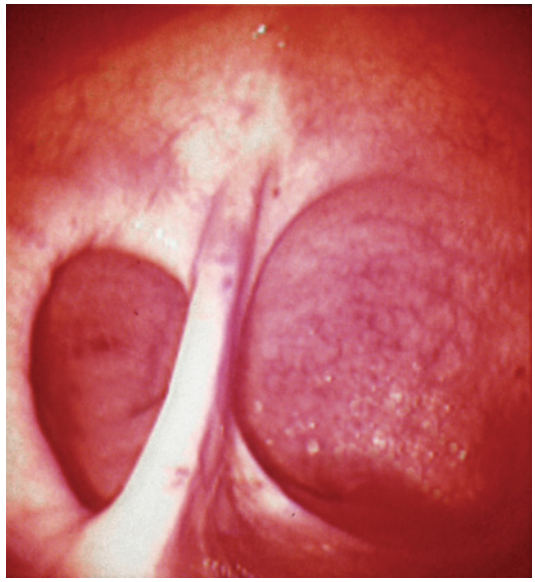

Figure 22.39. Double cervix in a llama.

22.2) and consider these conditions on any soundness or infertility examination.

Hypogenesis/Agenesis of Reproductive Organs

Failure of development, particularly of female reproductive organs, is often seen. The etiology is unknown in camelids, but genetic transmission has been reported in cattle and humans (Table 22.2). The major clinical sign is infertility, although pregnancy is possible in some cases, such as uterus unicornis (Figure 22.38) or double cervix (Figure 22.39).[1,5]

Segmental agenesis of the tubular genital tract of the female is common. Stenosis or occlusion of the tract may occur at any location from the oviduct to the hymen. Unilateral agenesis of an oviduct may never be detected. Agenesis within the uterus or vagina prevents outflow of uterine secretions, resulting in accumulation of fluid (mucometria) and dilatation of the segments of the tract cranial to the agenesis (Figure 22.40).

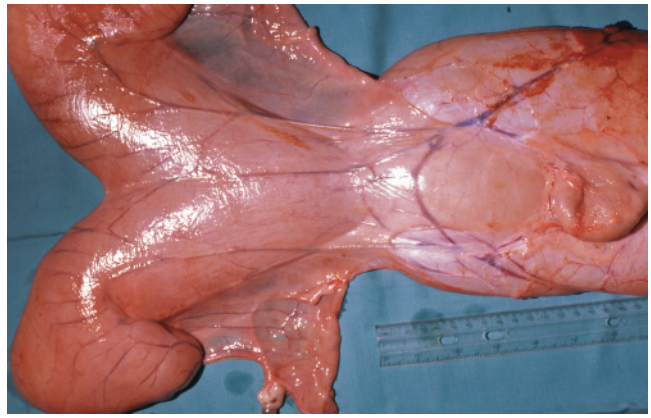

Figure 22.40. Mucometria caused by segmental agenesis of the vagina.

The dilated uterus may be mistaken for pregnancy on rectal palpation. The fluid may be milky to slightly reddish in color and have the consistency of skim milk. The fluid is sterile unless organisms have been introduced by diagnostic manipulations. It is difficult to differentiate mucometria from placental fluids on palpation, but it is easy to differentiate it from pyometra on ultrasonography. Exudates caused by pyometra have a flocculent appearance, while mucometria and placental fluids are homogenously clear.

The diagnosis of segmental agenesis involves a combination of rectal palpation, ultrasonography, examination with a vaginal speculum, aspiration of fluid, and visualization at laparotomy or laparoscopy, depending on the location of the lesion. See Chapter 17 for discussion of the normal anatomy of the reproductive system.

Uncorrected tubular agenesis precludes pregnancy except for uterus unicornis and unilateral oviduct agenesis. The veterinarian may be asked to open the tract, especially if the occlusion is at the level of the hymen or caudal vagina. Such surgery is possible, but the client should be advised of the likelihood that this condition is hereditary and that such an animal should not be used for breeding. Furthermore, depending on the length of time that the mucometria has been present, damage to the uterine mucosa may be irreparable. Ovariohysterectomy may be indicated to allow use of the female for fiber production, as a pet, or as a packer (Chapter 6).

Ovarian hypogenesis and agenesis also occur (Figures 22.41A and B). Persistent embryonic tissue that may be confused with an ovary is illustrated in Figure 22.42.

Intersex[42]

An intersex is any animal in which there is ambiguity in the structure of the gonads, reproductive tract, or external genitalia. Various forms of intersex are known to occur in camelids. Anatomic variations are

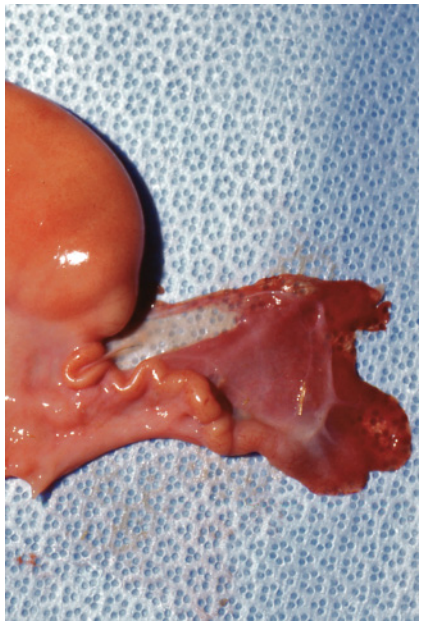

Figure 22.41A. Ovarian dysgenesis, hypogenesis.

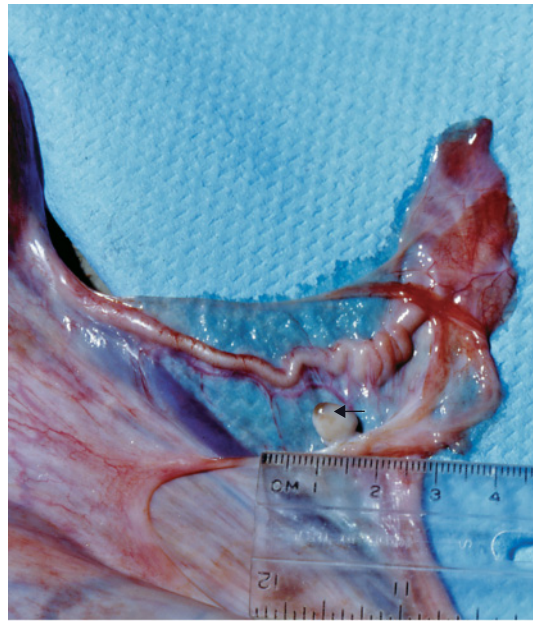

Figure 22.41B. Ovarian agenesis.

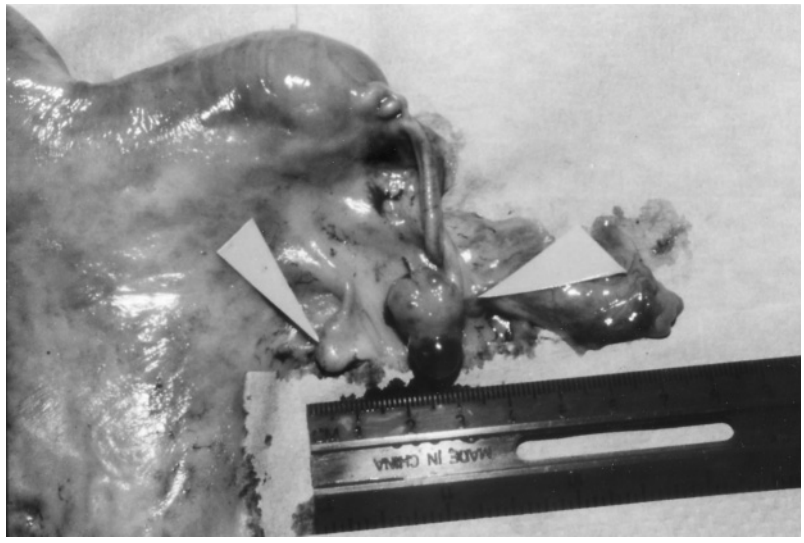

Figure 22.42. Persistent embryonic tissue that may be confused with the ovary on rectal palpation.

numerous, and clinical manifestations vary according to the hormone levels involved.

There are four stages of development of the male and female sexual organs: (1) chromosomal sex, as determined by the X and Y chromosomes at the time of fertilization, (2) gonadal sex, which occurs as the ovary and testis develop from the undifferentiated embryo gonad, (3) somatic sex, as other sexual organs develop, and (4) psychic sex, as the animal develops male or female behavior patterns.

A definition of terms follows.[96] A pseudohermaphrodite has the gonads of one sex but an alteration in one or more of the other criteria of sex identification. A true hermaphrodite has both ovarian and testicular tissue, with intermediate external genitalia. A lateral hermaphrodite has an ovary on one side and a testicle on the other. A unilateral hermaphrodite has an ovotestis on one side and either an ovary or a testicle on the other. A bilateral hermaphrodite has ovotestes on both sides.

The embryologic anatomy of genital organs is complex and confusing, but basic understanding is necessary for a consideration of the topic (Table 22.6).[51] Gonadal development begins with a swelling called the genital ridge, which forms on the Wolffian body in the dorsal abdomen. The testicle and ovary develop from the genital ridge. Initially, the gonad is indifferent, with a medulla and cortex. The medulla

Table 22.6. Homologies of male and female reproductive systems.

Undifferentiate stage	*Male*	*Female*
Internal genitalia		
Gonad	Testis	Ovary
Mesonephric tubules (Wolffian body)	Vas deferens	Epoophoron
	Paradidymus (rudiment)	Paroophoron
Mesonephric duct (Wolffian duct)	Epididymus	Duct part of epoophoron (Gartner's duct)
	Vas deferens	
	Ejaculatory duct	
Mullerian duct	Appendage of testis (rudiment)	Fimbria of oviduct
	Prostatic utricle (uterus masculinus)	Uterus
		Vagina (all or part)
Urogenital sinus	Prostatic, membranous and cavernous urethra	Urethra vestibule, vagina in part
	Bulbourethral glands	Vestibular gland
	Prostate	Paraurethral gland
External genitalia		
Genital tubercle	Glans penis	Glans clitoris
	Corpus penis	Corpus clitoris
Urethral fold	Raphe of scrotum and penis	Labia minor
Labioscrotal swelling	Scrotum	Labia majora

is the potential male portion, and the cortex the potential female portion. The Wolffian body is attached to the cloaca by the Wolffian duct, which will become the vas deferens and the epididymis in the male.

As sexual differentiation takes place in the female, the Mullerian duct develops, which eventually becomes the uterus, vagina, oviduct, and fimbria of the oviduct.[69] The Wolffian duct disappears, but vestigial structures may persist, which are called the epoophoron, paroophoron, and Gartner's duct.

The etiology of intersex in camelids is unknown, but certain forms are known to be genetically transmitted in all livestock species. Intersexes may also result from hormonal defects. Genital development may fail at any of three stages.

First, distributions of sex chromosomes during meiosis or even mitotic division may result in sex chromosome aneuploidy in gametes. Fragments of sex chromosomes or autosomes bearing sex-influencing genes may be abnormally distributed as a result of partial or complete deletion or translocation in chiasma formation. Second, gonadal morphogenesis may be disturbed because of abnormal corticomedullary relationships on the genital ridge. Third, secondary and accessory genital structures may develop abnormally under the influence of an irregular endocrine environment or as a result of teratogenic factors.

The clinical manifestations of intersex are highly variable. Behavioral patterns may be altered, and the external genitalia may appear as intermediates from expected morphology. A female pseudohermaphrodite llama had an enlarged clitoris, with the cartilaginous projection normally seen on the glans penis (Figure 22.43). Her tubular organs were normal, based on rectal palpation and digital vaginal examination. It was not possible to insert a vaginal speculum past the hymen, but a finger could explore as far as the finger could reach. The ovaries were not located. This female mounted other females, attempting to force them into recumbency. This is normal male behavior, but it has also been seen in normal females.

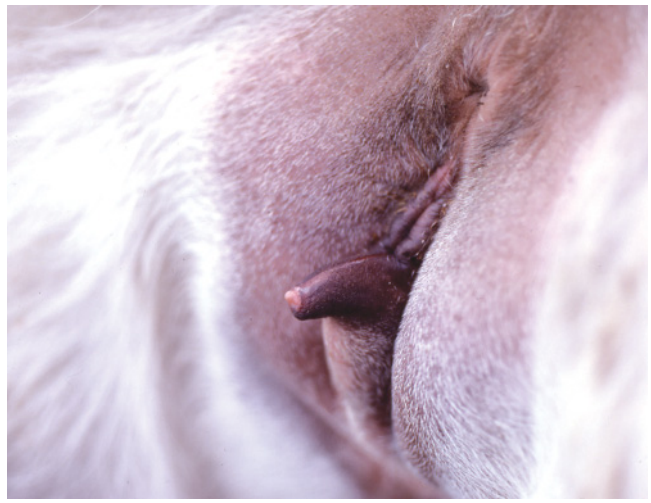

Figure 22.43. Female pseudohermaphrodite.

The definitive diagnosis of intersex is difficult. The ultimate diagnosis is based on gonadal histology, but other clues include the appearance of external genitalia, morphology of the reproductive ducts, altered

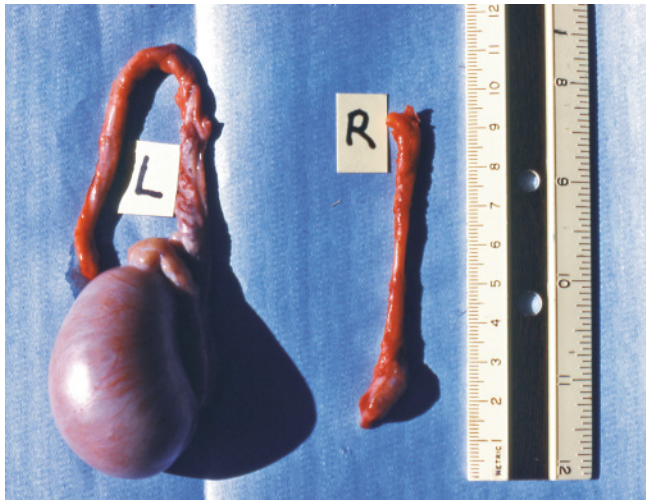

Figure 22.44. Testicular hypogenesis (R), normal (L).

karyotype, and hormone analysis. The female pseudohermaphrodite described above had a normal karyotype but significantly altered testosterone levels. The female's estrogen level was 6.2 pg/ml, progesterone 0.1 ng/ml, and testosterone 325 pg/ml. The basal estrogen level of the domestic female mammal is usually less than 10 pg/ml. If there is any follicular activity, the levels are higher than 10. A level of 6.2 pg/ml estrogen in the female llama indicated no follicular activity. A progesterone level of 0.1 ng/ml is basal. A testosterone level of 325 pg/ml is approximately ten times the normal level for a female. A cow would have 10 to 15 pg/ml and a mare 25 to 40 pg/ml as basal testosterone. The level in this female llama was approximately what would be expected in a cryptorchid stallion (200 to 500 pg/ml).

Newly Reported Defects

The following defects of the female camelid reproductive system have been reported since the second edition: urethrovaginal fistula,[92] transverse vaginal septum,[93] and vulvar deformations.[125]

Male Defects

Any of a long list of hereditary defects of male livestock species warrants exclusion of the individual from breeding.[38] Congenital defects of male camelids include testicular hypoplasia (Figure 22.44), cryptorchidism,[95] testicular cysts, penile hypoplasia, persistence of the penile frenulum, and curvature of the penis. It should be recommended to clients that such defects warrant exclusion of males for breeding.

Digestive Tract Defects

Atresia ani, atresia coli, and atresia ilei[103] are known to occur in camelids (Figure 22.45).[10] The etiology is unknown. In cattle, atresia coli has been associated

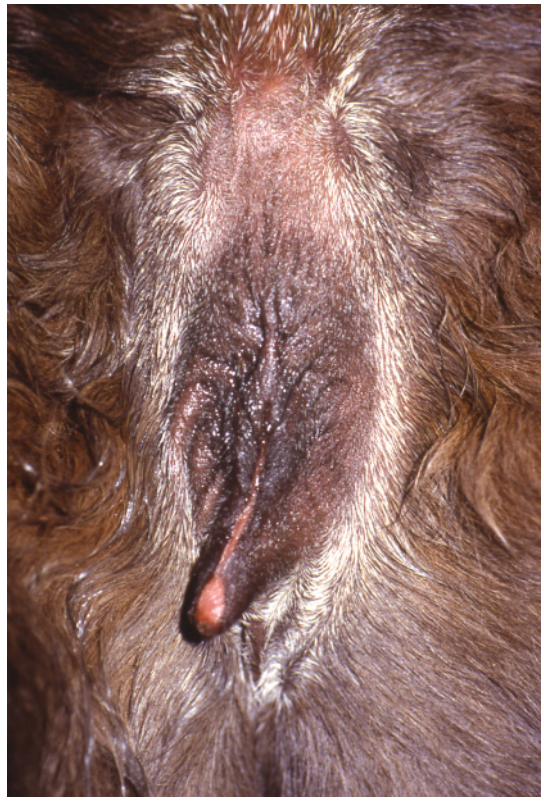

Figure 22.45. Atresia ani in a llama.

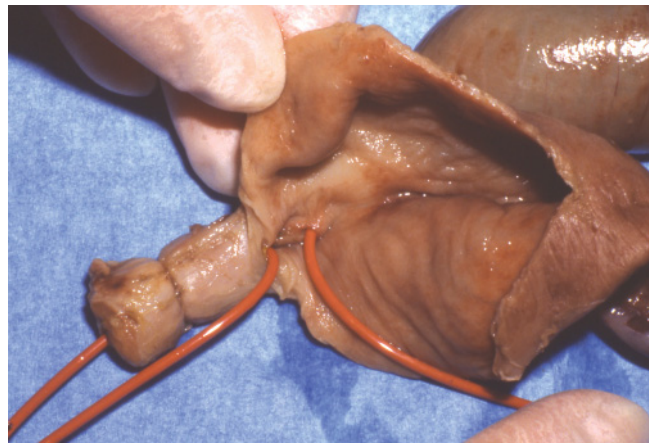

Figure 22.46. Congenital defect with colon emptying into the bladder.

with excessive pressure exerted during rectal palpation. Megaesophagus may be either congenital or acquired (Chapter 13). The etiology is unknown. The colon emptied into the bladder of one llama fetus (Figure 22.46).

Cardiovascular Defects[60,109]

Cardiovascular defects are seen sporadically in most animal species. None is unique to camelids (Table 22.2). Ventricular septal defect (VSD) (Figures 22.47 A

Figure 22.47A. Ventricular septal defect.

Figure 22.47B. Ventricular septal defect.

and B) is relatively common in llamas and may occur singly or be associated with other cardiovascular or congenital defects.[12,77,97]

Clinical signs may be limited to auscultation of a holosystolic murmur or accompanied by cyanosis and exercise intolerance. The author is aware of an adult llama with VSD that lives a normal life. The presence of a murmur is a frequent, perhaps normal, clinical finding in the newborn. The murmur should disappear by one week of age. Transposition of the great vessels is rare (Figures 22.48A and B, 22.49) and would

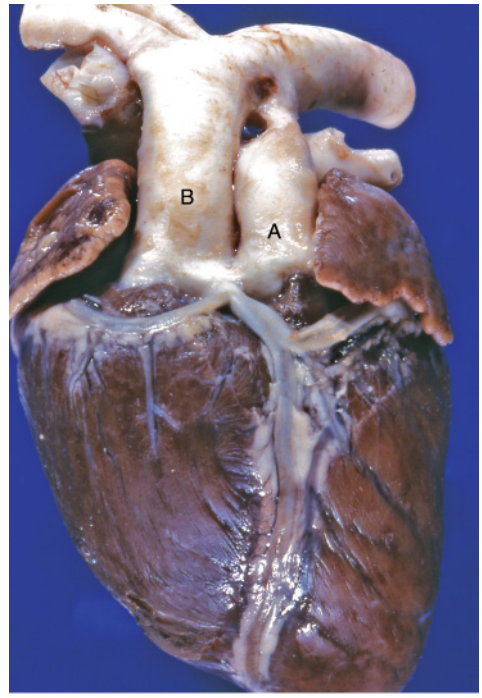

Figure 22.48A. Transposition of the aorta and pulmonary artery, closed. (A) Pulmonary artery, (B) aorta.

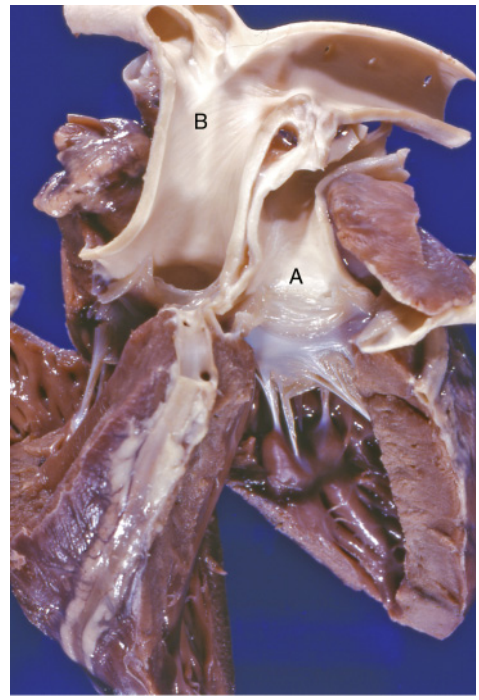

Figure 22.48B. Transposition of the aorta and pulmonary artery, open. (A) Pulmonary artery, (B) aorta.

be lethal shortly after birth unless accompanied by ventricular or atrial septal defect.

The diagnosis of VSD should be suspected with the presence of a murmur but can only be definitively diagnosed with ultrasonography. Other cardiovascular defects may be diagnosed only though radiography, fluoroscopy, or angiography.

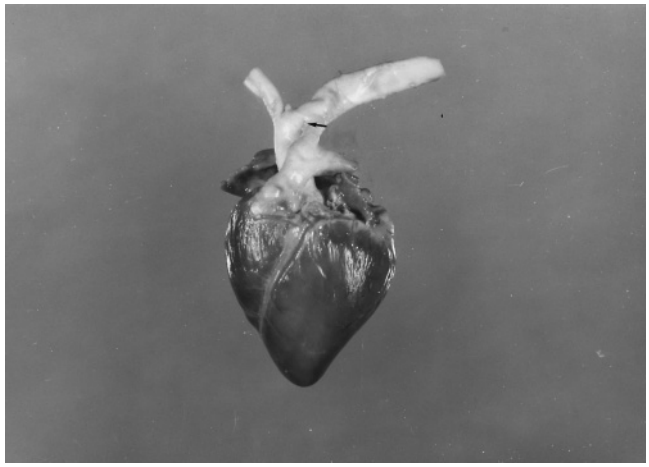

Figure 22.49. Normal crossover of aorta and pulmonary artery in a llama neonate.

Figure 22.50. Curled toenails in a llama.

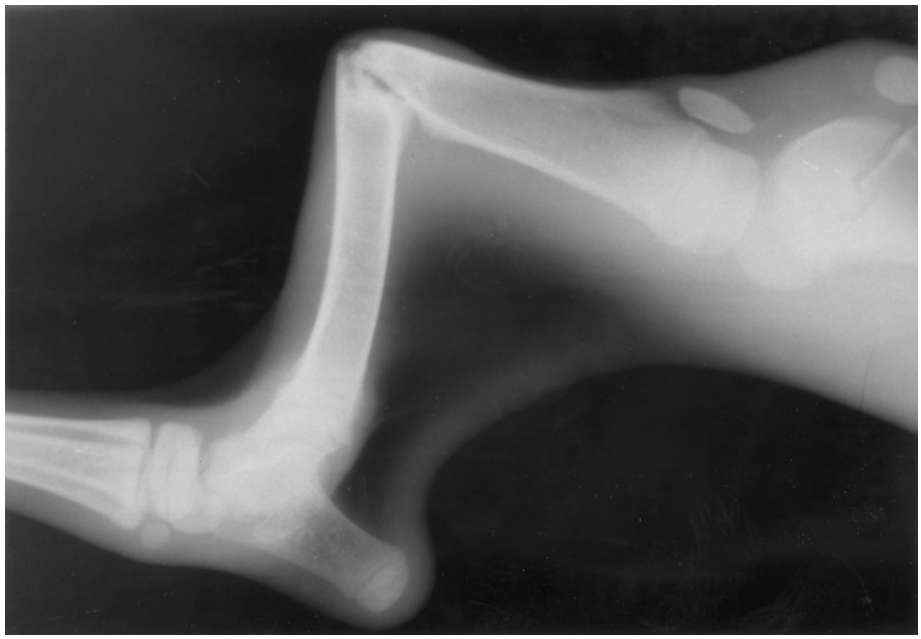

Figure 22.51. Contracted tendon of the gastrocnemius muscle, resulting in tibial fracture in the fetus.

Miscellaneous Defects[26,35,74,76,102]

Many other defects are listed in Table 22.2. Similar defects have been reported in other livestock, pets, and humans.[46] One other defect that is worthy of special mention is crooked toenails (Chapter 10). Overgrown nails and crookedness are common. Some nails grow crooked despite judicious and regular nail trimming. Crooked hoofs and nails are known to be genetically transmitted in cattle, horses, sheep, and pigs (Figure 22.50). Selection of breeding stock should include a consideration of sound feet and legs. An intrauterine defect caused fracture of the tibia in a fetus (Figure 22.51). Super- and subnumerary teats are illustrated in Figure 22.52. Wry face is also thought to be a hereditary defect (Figures 22.53A and B). Figure 22.54 shows a dwarf Bactrian camel.

Nonanatomic Defects

A discussion of congenital/hereditary defects would be incomplete without mention of physiologic characteristics that have a genetic basis. These are not known to be inherited traits in camelids, but they are so important in livestock that mention should be made here. Consider the following: milk production, mothering ability, disease resistance, growth rate, thermoregulatory adaptability, feed utilization, birth weight, semen quality, neonatal mortality, fertility, early embryonic death, and resistance to neoplasia.

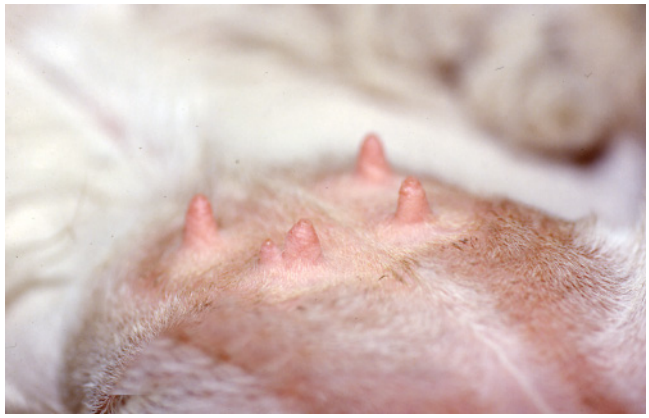

Figure 22.52. Supernumerary teats.

Figure 22.53A. Wry face in a llama.

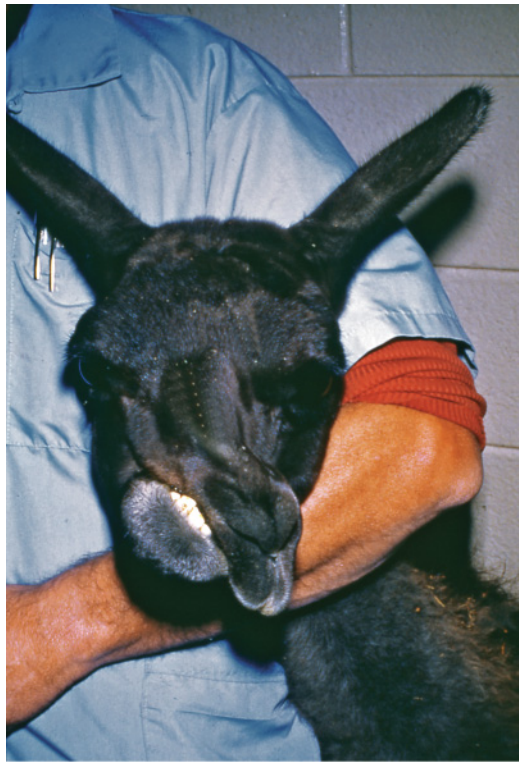

Figure 22.53B. Wry face in a llama

Figure 22.54. Dwarf camel.

NEW CONGENITAL DEFECTS OF SACs

The following congenital defects have been reported since the second edition: glaucoma,[15] wry face,[50] urethro-vaginal fistula, renal agenesis,[40,91] cataracts,[15] astrocytoma,[31] ocular changes,[77] vascular ring anomaly, hepatoblastoma[124] of the liver, Factor VIII deficiency of the blood, labial fusion of the vulva,[38] cystic rete testis,[61] vulvar deformities, cranial cruciate ligament aplasia,[b] and caudal vertebral malformations.

THE CAMELID IMMUNE SYSTEM

Immune competence is one of many characteristics determined by genetics. The immune deficiency syndrome is discussed in Chapter 9. An overview of what is known about the immune system of horses, livestock, and companion animals may be related to, and correlates with, what is known about camelids. An understanding of body defenses against microorganisms has tremendous clinical relevance. The health of the immune system affects how a camelid responds to vaccinations, determines susceptibility to and recovery from infectious and parasitic diseases, and controls whether or not passive transfer of immunoglobulins occurs correctly.

Clinical findings suggestive of immunodeficiency include recurrent illnesses, unexplained neonatal illness, illness from nonpathogenic organisms, failure to respond to immunoprophylaxis, disease resulting from the use of modified-live organism vaccines, recurrent uveitis, and some anemias.

Body Defenses

Barrier Systems

Healthy skin and mucosal surfaces form a barrier against noxious agents entering the body.

Nonspecific Defenses

These systems may be genetically transmitted or may be acquired with maturation and include the phagocytic, plasma lysozyme,[44] and complement systems.

Specific Defenses (Acquired Immunity)

Antigen/antibody and B and T cell systems are specific for given microorganisms. Included are both cellular and humoral activities.

Evaluation of Immune System Components

Phagocytic Cell System

1. A total and differential leukocyte count provides an indication of numbers of neutrophils available.
2. A bactericidal test is done in the laboratory to determine how efficiently phagocytes deal with microorganisms. The test is generic and can be performed in most diagnostic labs.

Complement System

1. Complement titers may be quantitated. Titers are not species specific, and are based on the ability to hemolyze erythrocytes in the presence of an antibody.
2. There is the ability to opsonize.
3. Quantification of individual components requires specific reagents and is not readily available in diagnostic labs.
4. It may be important to know that camelids may have variable titers of anticomplement that must be run in parallel with complement fixation tests to determine significant activity levels.

Plasma Lysozyme System

Plasma lysozymes may be quantitated by a turbometric method.[44]

Humoral Immunity[44]

Evaluation of B cell activity:

1. Quantification of immunoglobulin levels by single radial immunodiffusion (SRID). In a study of the comparison of two commercially available kits for quantitation of immunoglobulin G (IgG), it was found that the IgG Test Kit of Triple J Farms, Richmond, Washington, recorded levels approximately twice that of the Vet-RID Kit of Bethyl Laboratories, Montgomery, Texas. The investigators found that the reference solutions provided with the kits appear to have been calibrated to different standards. This is not a problem as long as a user consistently uses the same product and is aware of the standards stated.[32] Other methods may be used to estimate IgG levels, including determining total protein levels, globulin measurement, refractometer readings, protein electrophoresis, and the Llama S Test (latex agglutination). Refractometer readings are an indirect method but are quick and may be used in following the response to therapy. In another study glutaraldehyde coagulation, zinc sulfate turbidity, and sodium sulfite precipitation tests did not correlate well with RID.
2. Quantification of the antibody response to common vaccines, such as tetanus toxoid, *Clostridium perfringens* type D, and *leptospira bacterins*. Precise data are available on llama response to tetanus toxoid and clostridium.
3. Quantification of B cell numbers by immunofluorescence techniques.

Evaluation of T cell activity:

1. Response of T cells to mitogen stimulation (lymphocyte stimulation test).
2. Quantitation of total T cell and T cell subpopulations. This requires camelid-specific reagents and is not available at present.
3. Lymphocyte count.
4. Lymph node biopsy. Lymph nodes have T cell areas and B cell areas; the pathologist looks for the relative cellularity in these areas.

Necropsy

Lesions suggestive of immunodeficiency include lymphoid atrophy, adrenal hyperplasia, and gastrointestinal ulceration. Lesions may be subtle and masked by secondary infections and/or parasitism. Tissue should be properly collected and stored for future evaluation, even if it seems that expense or other factors may prevent immediate evaluation of the tissue. The lymphoid system is of major importance in evaluating immunodeficiency. Tissues used to evaluate possible immunodeficiency are listed in Table 22.7.

BLOOD-TYPING AND PARENTAGE VERIFICATION

Blood-typing was first applied to solve problems of mismatched blood transfusions in humans and later to solve problems of disputed paternity. In the late 1920s, systematic studies of blood groups in cattle were undertaken, which led to a wide use of blood-typing tests to verify the accuracy of pedigrees in registered cattle. Similar studies have been carried out in other domestic animals, but blood-typing to date has been most successfully applied to cattle and horses. Both cattle and horse breed registries have developed intensive blood-typing programs designed to keep

Table 22.7. A checklist of tissues to collect in suspected immunodeficiency cases.

Neonate

_____ Thymus, _____ bone marrow, _____ liver, _____ lung, _____ skin

Adult

_____ Standard tissues (lung, liver, kidney, GI, heart, and specific lesions)
_____ Adrenal gland
_____ Areas of ulceration of the GI tract

Lymphoid tissue

_____ Spleen, _____ bone marrow, _____ Peyer's patch,
_____ Tonsilar tissue, _____ lung
_____ Skin. [Aggregations of lymph cells are characteristic of the skin of -llamas, as is also bronchiolar-associated lymphoid tissue (BALT).]
Lymph nodes: _____ Mediastinal, _____ mesenteric, _____ prescapular

Figure 22.55. Paco-vicuna (alpaca × vicuna).

pedigree errors to a minimum among registered animals.[27,75,83–88,103]

Basic studies of blood group factors, protein variation, and enzyme patterns have now been conducted in camelids.[27,84,85,98] Sufficient variation among individuals has been found to validate the use of blood-typing tests for parentage verification. Specific blood group antibodies are harvested from immunized llamas and prepared in the laboratory for serologic testing against the blood of animals being tested. To date, five erythrocyte factor systems have been identified: A-B, C-no C(-), D-(-), E-(-), and F-(-). The A-B, C, and D systems have been tested on more than 100 sire, dam, and offspring trios. The E and F systems have been tested on fewer animals.

Parentage verification is accomplished by comparing the blood types of an offspring with those of its alleged sire and dam. If the blood types of the offspring can be accounted for on the basis of the types present in the parents, the parents qualify. All parentage cases are based on the principle of genetic exclusion. This means that the burden of proof rests in showing that a certain animal cannot be a parent of the animal in question. When a parentage is said to qualify, it can only be assumed that such is correct. It may be impossible to eliminate one of two possible sires that are themselves closely related based on blood type. DNA fingerprinting is now being used to adjudicate a conflict.

Parentage verification was carried out by evaluating blood proteins and enzymes from 1988 to 1997. With the development of microsatellite markers, DNA testing became the method of choice for parentage verification. The accuracy is calculated to be 99.5%.

DNA technology has been applied to establish evolutionary lineages of camelids.[63,68,69,78–80] Future genomic studies will aid in the progress of this field.[41,90,101,104–106,110,116,122]

CAMELID HYBRIDS

All SACs will inter-breed and produce fertile offspring (Figure 22.55). Likewise, OWCs interbreed

(Figure 22.56). Hybrids between OWCs and NWCs are rare but have been produced using artificial insemination. A stillborn fetus resulted from an alpaca and camel cross in Israel, (Figure 22.57). Scientists in the United Arab Emirates have produced live offspring from a guanaco-camel cross (Figures 22.58 to Figure 22.59 A and B). It is not yet known whether or not these crosses are fertile.

A concluding remark: Don't make a diagnosis of congenital crooked legs on newborn camelids until thirty days post delivery. Frequently newborn ungulates have ligament and tendon laxity that corrects itself after a few days outside the uterus. Figures 22.60 and 22.61 show a newborn camel that by thirty days had normal camel conformation.

Figure 22.56. Dromedary × Bactrian camel hybrid.

Figure 22.57. Stillborn alpaca × camel, Israel.

Figure 22.58. Mother of Rama, a guanaco × camel, United Arab Emirates.

Figure 22.59A. Rama the Cama, age ten months.

Figure 22.59B. Rama the Cama age six years.

Figure 22.60. Crooked legs on a newborn camel.

Figure 22.61. Crooked legs on a newborn camel.

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[a] Dr. L.W. Johnson, Fort Collins, Colo., personal communication, March 1997.

[b] Dr. Claire Whitehead, personal communication, June 18, 2009.

23

Toxicology

Only a few instances of poisoning have been reported in camelids, yet it is likely that they are susceptible to many of the noxious substances that affect other domestic and wild animals. Numerous texts deal with the effects of poisonous substances in humans and domestic animals.[5,8,10,22,23,26,29,33,36,48,51] The reader is directed to these sources for details of the diagnosis and treatment of specific toxicants.

Although examples of toxicities are given and tables presented to list known toxic substances that might affect camelids, the primary focus is enumeration of basic concepts. Understanding these may make it possible to advise clients on how to avoid poisoning by altering potentially dangerous situations.

Toxicity is often thought to be a wholly artificial phenomenon, which humans have complete power to cause or prevent. This is a naive belief because toxicants are as much a part of any environment as are substances that nourish an animal. Adaptation to the ingestion of poisonous substances is a basic part of evolution. Just as a certain animal population may develop resistance to a microorganism, many animals develop tolerance to a given toxicant. The problem is that virtually nothing is known about the resistance of camelids to most toxicants.

ADAPTATION TO TOXICANTS

Animals cope with toxicants through one or more of the following strategies: avoidance, dilution, degradation, or detoxification.[18] Should these strategies fail, the animal will be adversely affected and may ultimately die.

Avoidance is a crucial skill that must be learned early in life. It would be natural for camelids in their native habitat to avoid unpalatable (potentially toxic) plants unless driven by extreme hunger. Camelids in other areas of the world have been transported to a wide variety of new habitats and have had no opportunity to learn appropriate avoidance skills. Fortunately, camelids are fastidious in food selection, so they may be partially protected.

Few irrigated and managed pastures are totally free of poisonous plants. As many as 40% of the plants growing on native rangelands may contain secondary plant compounds (poisonous substances). Harvested hays and prepared feeds may contain harmful weeds.

An animal with a choice of plants may dilute a toxicant by ingesting only small quantities that fail to reach the threshold level for production of toxicity. Camelids are highly adapted to eating small quantities of a variety of plants, if given a choice.

A toxicant ingested by a camelid may be degraded within the digestive tract. The multicompartmented stomach of camelids and ruminants is well designed to degrade toxic compounds, but the animal must depend on crucial gastrointestinal (GI) microorganisms to assist in rendering the toxicant harmless.

Once the toxic agent is absorbed from the GI tract, the body must either excrete it unchanged, sequester it in a nonactive storage site, detoxify it by molecular rearrangement, or suffer the ill effects caused by the toxicant. All vertebrates have general detoxification pathways that can deal with many different substances. Some have specialized mechanisms, unique to a given species. Specific mechanisms are unknown in camelids. Much detoxification is carried out by hepatic microsomal enzyme activity. Enzyme systems require priming and periodic reactivation by exposure to nonlethal quantities of toxicants. The intensively managed camelid may never be given an opportunity to stimulate these systems and may be at a great risk if suddenly exposed.

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DIAGNOSIS OF POISONING[6,25]

The clinical signs caused by most potential toxicants in camelids are unknown. Rarely will a diagnosis of poisoning be evident on initial examination. No pathognomonic signs of poisoning have been identified, and a limited number of pathognomonic lesions have been reported.

Diagnosis of poisoning depends upon analyzing a detailed history, evaluating clinical signs, using special laboratory diagnostic procedures, and, in some cases, necropsy and chemical analyses of tissue. Other possible disease conditions must be eliminated simultaneously by a thorough examination and medical workup.

Poisoning cases frequently result in litigation. It is imperative that detailed records be kept. It is unwise for the veterinarian to casually intimate, "It looks like a poison." A diagnosis of poisoning is not valid if based solely on the premise that no other disease entity or cause could be determined.

TREATMENT OF POISONING

The basic principles of treating a suspected case of poisoning include removal of the source and removal of the toxicant from the animal. If the toxicant has been applied externally, the animal must be bathed. This is difficult with camelids, but copious amounts of water should be used to attempt to penetrate the coat and rinse the toxicant from the surface of the skin.

Emetics are inappropriate for use in camelids. The volume of ingesta in compartment 1 (C-1) of the stomach precludes effective gavage. Catharsis may be produced by using magnesium oxide (10 to 20 g), magnesium sulfate (0.2 g/kg), or mineral oil (10 ml/kg). Activated charcoal may adsorb toxicants and may be administered via gastric intubation (100 to 200 g in 2 L water, 13 g/kg). Activated charcoal is produced by super heating a source of carbon to aerate the carbon to create more surface area to capture and adsorb toxic substances.

A limited number of general and specific antidotes are available. General antidotes include 20% calcium gluconate, 10% glucose, and 10% to 20% sodium thiosulfate. Specific antidotes include calcium versenate against lead, sodium thiosulfate and sodium nitrite against cyanide, and atropine against anticholinesterase compounds used as insecticides or parasiticides.

Early in the course of most poisonings, symptomatic and supportive therapy should be instituted, which will be of benefit in infectious or metabolic disease as well. Maintaining hydration with fluids, supporting respiration with oxygen and circulation with steroids and cardioactive drugs, and controlling central nervous system (CNS) stimulation with diazepam are indicated.

PREVENTION OF POISONING

Three basic concepts are important in the prevention of poisoning in camelids. The most important is elimination of exposure to the toxicant. Purchased hay should be carefully inspected for quality and the presence of weeds or foreign material. Processed feeds must be of the highest quality. Since it is impossible to inspect certain types of processed feeds for poor or toxic ingredients, the integrity of the processor is of primary importance. Pastures should be walked periodically and unknown plants identified.

The second concept is avoidance of stress that enhances toxic effects. The third concept is provision of sufficient quantity and quality of nutrients in the diet to sustain healthy populations of GI microflora and microfauna.

CLASSES OF POISONS[31,45]

Poisons that may be encountered in camelids include insecticides, rodenticides, disinfectants, cleansing agents, paints, antifreeze, plant toxins, mycotoxins, drugs, and animal venoms. Tables 23.1 and 23.2 provide a short list of potential poisons in camelids. Specific examples follow.

Insecticides

More than 1,800 products are listed as pesticides.[4] Pesticides or insecticides are classified by chemical content, and include organic phosphates (OP), carbamates, and chlorinated hydrocarbons (CHC). A few OPs that are used in the United States include chlorpyrifos, coumaphos (Co-Ral), Ronnel (Korlan), dichlorovos (Vapona strips), and Fenton (Tiguvon). Carbamates include aldicarb and carbaryl. Chlorinated hydrocarbons include DDT, DDF, aldrin, heptacholor, toxaphene, chlordan, lindane, dieldrin, eldrin mirex. Pyrethroids are another class of insecticides that have a natural plant source (pyrethins) or are produced synthetically (Permethrin).

Camelids are probably as susceptible to insecticides as other livestock species.

Organic Phosphate Poisoning

Etiology

Many organic phosphate (OP) insecticides have been produced.

Clinical Signs

The clinical signs in camelids are typical of organic phosphate poisoning in livestock and other animals.[44]

Signs are associated with their effect on the nervous system. Signs of cholinergic action include diarrhea, sweating, micturation, and salivation. Central nervous system signs include muscle fasiculation, muscle

Table 23.1. Miscellaneous toxicants with similar effects on all species.

Toxicant	*Source for llamas*	*Clinical signs*	*Special diagnostic procedures*	*Pathology*	*Management*
Iodine	Therapeutic overdose	Dermal hyperemia and scaling, lacrimation	History and signs	Dermatitis	Removal
Fluoride	Rodenticides "1080", NaF	Hyperirritability, cardiac arrhythmias, trembling, colic, convulsions, death within 15–30 min	History and signs, chemical analysis of stomach, liver, and kidney	Biochemical lesion	Nonspecific, diazepam, anesthesia, calcium gluconate
Insecticides					
Organophosphates	Parasiticides	Salivation, colic, diarrhea, vomiting, dyspnea, miosis, muscle twitching, tetany, depression	History and signs, analysis of cholinesterase activity	No lesions	Removal of substances, atropine (0.4 mg/kg) 2-PAM (20 mg/kg)
Organochlorides	Insecticides	Hypersensitivity, muscle fasciculation, tonic/clonic convulsions, depression	History and signs, chemical analysis of liver, kidney, and stomach contents	No lesions	Removal, symptomatic, anesthesia
Rodenticides					
Strychnine	Rodent bait	Tenseness, tetany, convulsions following stimulation, mydriasis	Chemical analysis of stomach contents	No lesions	Removal, sedation and anesthesia
Anticoagulants	Rodent bait	Hemorrhages at trauma sites	Clincal pathology, prolonged clotting time, bleeding time and clot retraction	Hemorrhages	Blood transfusion (20 ml/kg), vitamin K (20–30 mg)

twitching, weakness, tremors, incoordination, dyspnea, and convulsions.

Diagnosis

Clinical signs are subjective, but one must differentiate organochlorine poisoning from other central nervous system disorders. In the laboratory, evaluation of blood cholinesterase activity is used.

Management

Administration of atropine sulfate is the standard therapy for organic phosphate and carbamate insecticide poisoning. Poisoned animals are resistant to the effects of atropine, so the initial dose should be 0.2 to 0.4 mg/kg rather than the usual mammalian dose of 0.04 mg/kg. If possible, half the dose should be given intravenously and the rest subcutaneously. Pupillary dilatation effects may last for days to weeks following atropine therapy, so clients should be advised to keep animals in the shade until pupillary constriction is normal. Pralidoxime (2-PAM) is effective against organic phosphate poisoning, but it is not recommended for carbamate poisoning. The dose is 20 mg/kg. Activated charcoal should be administered orally at 1 to 3 g/kg if the organic phosphate was taken orally. Administer diazepam to control convulsions.

It is necessary to be certain that the airway is open. Oxygen insufflation is appropriate if the animal is breathing, but it may be necessary to intubate and institute positive pressure respiration.

In an experiment, three llamas were given therapeutic doses of a drug used for treating lice infestations—

Table 23.2. Heavy metal poisoning in camelids.

Metal	*Source for llamas*	*Clinical signs*	*Diagnosis*	*Pathology*	*Management*
Lead (Pb)	Contaminated forage, paint	Depression[a], anorexia, stomach atony, colic, diarrhea, ataxia, circling, blindness	Blood, liver, and kidney analysis	Encephalopathy, gastroenteritis	Calcium EDTA (110 mg/kg) for 5 days
Arsenic (As)	Contaminated forage, pesticides, herbicides	Colic[a], weakness, salivation, trembling, diarrhea, depression, dehydration, shock	Chemical analysis of liver, kidney, and stomach contents	Gastroenteritis	Removal, symptomatic, fluids, prevent secondary infection
Selenium (Se)	Plants containing high Se, therapeutic overdose	See Nutrition, Chapter 2	Chemical analysis of liver and kidney	Pansystemic, hepatic necrosis, renal necrosis	Removal, symptomatic
Copper (Cu)	Water contamination, insecticides, food supplements	See Nutrition, Chapter 2	Chemical analysis	Hemolytic anemia, hepatic necrosis	Nothing for acute toxicity, add molybdenum
Molybdenum (Mo)	Plants high in Mo and low in Cu	See Nutrition, Chapter 2. Diarrhea, coat depigmentation, decreased fertility, emaciation, anemia	Response to Cu supplementation	Microcytic, hypochromic anemia, hemosiderosis, emaciation	Cu supplementation, Cu injection

Note: Signs listed are a composite.
[a] Clinical syndrome has not been described in lamoids. Signs are variable in livestock.

chlorpyrifos (25 mg/kg body weight).[44] Plasma levels of pseudocholinesterase were followed. Plasma pseudocholinesterase activity decreased to as low as 38% of baseline by five days and returned to 90% baseline in approximately thirty-six days. None of the three llamas exhibited overt signs of toxicity. Twenty-one healthy llamas were used to establish a baseline level of plasma pseudocholinesterase activity. The mean was 209 ± 29.16 IU/L (range = 150 to 252).[44]

Rodenticides

No cases of rodenticide poisoning in camelids have been reported, but these agents affect a broad host range, and camelids are likely to be susceptible if exposed. Owners should be cautioned about the use of rat and mouse bait in impregnated grain that may be placed where camelids can gain access.

Heavy Metals

Lead, arsenic, mercury, copper, and molybdenum are used in agricultural products and paints. It should be assumed that camelids are susceptible. Clinical signs and lesions are presumed to be similar to those of poisoned cattle and sheep.

Copper[2,9,32]

Absorbed copper is stored in hepatocyte lysosomes. When the storage capacity of the liver is exceeded, copper is released into the cytoplasm of the hepatocyte causing hepatocellular necrosis. Once the hepatocyte cell wall has been destroyed, copper is released into the circulation, causing hemolysis, icterus, and anemia. The excessive amount of free hemoglobin in the circulation may obstruct normal kidney function, resulting in renal failure.

Copper poisoning was diagnosed in four zoo llamas being fed a dairy pellet supplement containing 44.8 mg/kg of copper.[14] The total dietary intake was estimated to be 25 mg/kg of feed (cattle tolerate relatively higher levels of copper in the diet as compared with sheep). Owners should be advised that pellets or mineral mixes intended for cattle are not suitable as supplements for camelids unless the total dietary intake of copper, including the supplement, is <15 mg/kg of feed. It is also necessary to maintain a copper/molybdenum ratio <10:1.

Clinical Signs. In one animal, the first signs noted were lethargy and inappetence. The following day the llama was recumbent, markedly depressed, dyspneic, and hypothermic, and had pale mucous membranes. It died eight hours later. None of the poisoned llamas were icteric, nor did they show evidence of hemoglobinuria. The classical signs of copper poisoning in cattle are anorexia, weakness, hemoglobinuria, hemoglobinemia, and icterus.

Diagnosis. At necropsy, hepatomegaly was the primary gross lesion and hepatocellular necrosis and bile duct proliferation were the histologic lesions. Serum copper levels varied from 4.3 to 5.7 µg/dl, and liver copper levels were 847 to 1,700 mg/kg dry weight. Normal hepatic copper levels in llamas are reported to be 6.7 to 330 mg/kg in neonates and 10 to 36 mg/kg in adults.[2] Serum enzymes associated with hepatic necrosis were elevated: aspartate transaminase (2,525 to 5,137 U/L [normal 179 U/L]), lactic dehydrogenase (2,286 to 8,652 U/L [normal 287 U/L]), and γ-glutamyl transferase (177 to 216 U/L [normal 125 U/L]).

Serum copper levels remain within normal limits until hemolysis and hepatic necrosis occurs.

Plants[7,52]

There are only a few published reports of plant poisoning in OWCs and NWCs. In North America the most common plant poisonings are oleander (*Nerium oleander*)[21] and various species of the family Ericaceae.[19,20,24] No plant poisonings have been reported from South America. Camels and llamas browse a wide variety of plants and may be affected, in certain circumstances, by the plants listed in Tables 23.3 to 23.5 (Figures 23.1 to 23.20B). Alpacas are primarily grazers and hence are less likely to consume shrubbery. Other plants may also affect camelids, but little

Figure 23.1. Corn lily (false hellebore) (*Veratrum californicum*).

Table 23.3. Poisonous plants that may affect llamas on trek.

Common name	*Scientific name*	*Habitat*	*Poisonous principle*	*Signs of poisoning*	*Therapy*
Arrowgrass	*Triglochin maritina*	Meadows at low to moderate elevations	Cyanogenic glycoside	Muscle twitching,-convulsions, dyspnea, bright red blood	Methylene blue, sodium-thiosulfate
False hellebore, corn lily (Fig. 23.1)	*Veratrum californicum*	High mountain meadows	Alkaloids	Vomiting, salivation, convulsions, fast irregular heart beat	S
Death camas, sandcorn (Fig. 23.2)	*Zigadenus* sp.	Hillsides, fields, meadows in spring of year	Alkaloids	Foaming at mouth, convulsions, ataxia, vomiting, fast weak pulse	S
Nightshade (Fig. 23.3)	*Solanum* sp.	Ubiquitous	Alkaloid-glycoside solanine	Vomiting, weakness, groaning	S
Jimsonweed, thornapple (Fig. 23.4)	*Datura metaloides*	Waste places	Alkaloid-atropine	Dry mucous-membranes, dilated pupil, mania	Parasympathomimetics
Chokecherry,wild cherry (Fig. 23.5)	*Prunus virginiana*	Streamsides	Cyanogenic glycoside	Dyspnea, convulsions, rapid death, bright red blood	Sodium nitrite and sodium thiosulfate
Western sneezeweed (Fig. 23.6)	*Helenium hoopesii*	High mountain meadows	Glycoside	Vomiting, depression, frothing at mouth, coughing, weak -irregular pulse	S
Labrador tea (Fig. 23.7)	*Ledum glandulosum*	Around lakes, meadows, streams	Andromedotoxin (diterpene), arbutin	Vomiting, colic, paresis, anorexia, muscle twitches	S
Black laurel, Mt. laurel (Fig. 23.8)	*Leucothoe davisiae*	Around lakes, meadows, streams	Andromedotoxin (diterpene), arbutin	Vomiting, colic, paresis, anorexia, muscle twitches	S
Western azalea (Fig. 23.10)	*Rhododendron -occidentale*	Around lakes, meadows, streams	Andromedotoxin (diterpene), arbutin	Vomiting, colic, paresis, anorexia, muscle twitches	S
Rhododendron (Figs. 23.9, 23.10)	*Rhododendron* sp.	Around lakes, meadows, streams	Andromedotoxin (diterpene), arbutin	Vomiting, colic, paresis, anorexia, muscle twitches	S
Oleander (Fig. 23.19)	*Nerium oleander*	Ornamental	Cardioactive glycoside	Diarrhea, colic, cardiac irregularities, cyanosis	S, gastrotomy
Castorbean (Fig. 23.11)	*Ricinus communis*	Ornamental, may escape	Ricin, water soluble albumitoxin	Anaphylactic shock, diarrhea	Treat for shock, fluids
Tobacco, tree tobacco (Fig. 23.12)	*Nicotiana* sp.	Waste places	Alkaloid, nicotine	Stimulations of CNS, then depression; sweating, muscle twitches, convulsions	S

Note: S = Symptomatic. In most cases of poisoning from ingestion of poisonous plants, there is no specific antidote; rather, it is necessary to treat symptomatically.

Table 23.4. Additional plants that may cause poisoning in SACs.

Scientific name	*Common name*	*Poisonous principle*	*Habitat*	*Signs of poisoning*	*Management*
Robinia pseudoacacia	Black locust	Alkaloids	Ornamental	Diarrhea, collapse, shock	Supportive
Digitalis purpurea	Purple foxglove	Cardioactive glycoside	Ornamental	Same as oleander poisoning, see text	Supportive
Taxus baccatta	Yew	Alkaloid	In gardens and parks as trees, hedges. Found wild in California.	Sudden death, dyspnea, collapse, diarrhea	Supportive
Lantana camara	Lantana	Hepatotoxins	Ornamental ground cover, escapes into waste areas	Hepatic insufficiency, photosensitization, icterus	Remove source, keep out of light, treat liver
Asclepias spp.	Milkweed	Alkaloidal glycosides	Along roadsides, edges of cultivated fields, waste places	Depression, weakness, convulsions, diarrhea	Symptomatic
Senecio spp.	Ragwort, groundsel, fireweed	Pyrrolizidine alkaloids	Weed in hay and waste places	Hepatic insufficiency	None
Dieffenbachia seguine	Dumbcane, dieffenbachia	Oxalate crystals	Ornamental house plant	Head shaking, salivation, excessive drinking	Wash mouth out with water
Nandina domestica	Heavenly bamboo	Cyanogenic glycosides	Ornamental shrub	Same as cyanide in all species, see text	See text
Nitrate accumulator plants	Many species of forage grasses and weeds	Nitrate/nitrite	Harvested crops, waste areas	See text	Methylene blue, see text
Quercus spp.	Oaks	Tannins, tannic acid, pyrogallol	Trees planted for shade around homes and barnyards, also found in woods and forests	Renal failure, high BUN and creatinine	Symptomatic, prognosis poor
Osteospermum echionis	South African daisy	Cyanogenic glycoside	Ornamental, Australia	See text for HCN	See text for HCN
Acer rubrum	Red maple	Oxidant	Eastern USA	Hemolytic anemia	Blood transfusion

Table 23.5. Poisonous plants that may affect camels.

Scientific name	*Common name*	*Country*	*Poisonous principle*	*Signs of poisoning*
Capparis tomentosa	Magico medicinal plant	Africa	Glycoside	Torticollis, tremors, weakness, incoordination, dyspnea, convulsions
Erythrophloeum chlorostachys	Ironwood, camel poison	Australia	Alkaloid	Staggering, heart pounding, blindness, dyspnea, star gazing
Nerium oleander	Oleander	USA, Australia, India, Africa	Oleandrin, cardioactive glycoside	Diarrhea, salivation, tremors, convulsions, cardiac arrhythmia
Gastrolobium grandiflorium	Desert poison bush	Australia	Fluroacetate	Tachycardia, dyspnea, muscle spasms, convulsions
Oxylobium spp.	Box poison	Australia	Fluroacetate	Same as above
Gyrostemon granulosus	Camel poison	Australia	?	Coughing, nasal discharge, foaming, dyspnea
Dubosia hoopwoodii	Emu poison bush	Australia	Alkaloid, nicotine	Trembling, weakness, incoordination
Trema tomentosa	Poison peach	Australia	glycoside	Depression, twitching, staggering, coma
Homeria breyniana	Cape tulip	Australia, Africa	?	Tremors, colic, stiffness, incoordination, diarrhea
Peganum hirmale	African rue	Africa, India	Alkaloid	Paralysis
Datura stramonium	Thorn apple	Africa, USA, United Arab Emirates	Alkaloid, atropine	Bloat, colic, dyspnea, maniacal behavior
Iphiona aucheri	Composite	United Arab Emirates	Diterpine glycosides	Signs of liver failure from liver necrosis
Palicourea grandiflora	Mata	Brazil	Fluroacetate	Sudden death, cardiac failure
Diplotaxa harra		North Africa		Stiffness in the hind quarters
Cassia truncata				
Uvaria denhardtin				
Thasia gargancia	Apiaceae	North Africa	Organic acids	Excessive salivation, colic, diarrhea
Ipomoea carnea	Morning glory			

benefit may be gained by listing every poisonous plant that has ever been suspected of causing poisoning in any species of animal. It is better for the veterinarian and the camelid owner to become knowledgeable about a few key, likely plants than to bother with hundreds of less likely plants. References are listed, if greater detail is desired.[17–22,24,27,40,42,44,49]

Ornamental plants are a special hazard for camelids.[17] Unfortunately, such plants may not appear in poisonous plant lists and booklets of the region, because many ornamentals are not native.

Rhododendron Poisoning[12,19]

A number of genera and species within the plant family Ericaceae contain a resin, andromedotoxin, and a glycoside, arbutin, which produce an identical syndrome in a broad host range. A description of the history and clinical signs of two llamas poisoned with Sierra laurel (*Leucothoe davisiae*) follows.[19]

Two pack llamas were tied to some shrubs near a grassy area at the edge of a small lake during a lunch stop. The first clinical sign was noted one and a half hours later when one llama began to cough and appeared to be choking. Within an hour, he was recumbent and refused to rise. The second llama began foaming (shaving cream consistency) from the mouth. He, too, began coughing and choking. Both llamas ultimately began projectile vomiting between episodes of rolling, retching, and groaning. Depression, anorexia, vomiting, groaning, and signs of colic persisted for two days. On the third day the animals had recovered sufficiently to slowly move out of the mountain to a trailhead.

The signs observed in these llamas are similar to those seen by the author in cases of rhododendron poisoning in both llamas and domestic livestock. The composite of the clinical signs includes anorexia, repeated swallowing, salivation, depression, vomiting, bloat, colic (straining, rolling, groaning, grinding

Figure 23.2. Death camas (*Zigadenus* spp.). (A) Underground corm.

Figure 23.3A. Nightshade (*Solanum sarrachoides*).

Figure 23.3B. *Solanum nigrum*.

Figure 23.4A. Jimsonweed (*Datura stramonium*).

Figure 23.4B. Tolguacha (*D. metaloides*).

of the teeth), weakness, ataxia, prostration, dyspnea (aspiration pneumonia), bradycardia, and hypotension. Hepatopathy may be a sequel.

Treatment of rhododendron poisoning is nonspecific. Administration of a cathartic such as magnesium oxide (10 to 20 g) is appropriate. Activated charcoal (100 to 200 g) is a general antidote and may be administered by stomach tube or as a drench consisting of a slurry of activated charcoal in water. However, such therapy is dangerous in a vomiting animal. Atropine (0.04 mg/kg) may be used to alleviate bradycardia. Other symptomatic treatment is indicated.

Oleander Poisoning

Oleander (*Nerium oleander*) is one of the most toxic shrubs to which a camelid or any other mammal may be exposed. The lethal oral dose of either green or

Figure 23.5A. Chokecherry (*Prunus demissa*).

Figure 23.5B. *P. runus emarginata.*

Figure 23.6. Western sneezeweed (*Helenium hoopesii*).

Figure 23.7. Labrador tea (*Ledum glandulosum*).

Figure 23.8. Black laurel, Mt. laurel (*Leucothoe davisiae*).

Figure 23.9. Azalea (*Rhododendron* spp).

dried leaves is 225 mg/kg. For a 150-kg (330-lb) llama, this would mean less than 35 g of leaves (15 medium-sized leaves or 1 leaf/10 kg body weight). Comparable quantities based on weight would kill camels.

The poisonous principle is a cardioactive glycoside, similar in action to digitalis.

Oleander is a beautiful ornamental shrub, native to the Mediterranean area and grown extensively throughout California and along the southern tier of the United States (Figure 23.19). As a potted shrub that can be moved inside during the winter, it may be found almost anywhere. Camelids may obtain olean-

Figure 23.10A. California rosebay (*Rhododendron macrophyllum*).

Figure 23.10B. Western azalea (*R. occidentale*).

der leaves in a variety of ways. Rarely will they ingest leaves directly from the living shrub. However, if cuttings or lawn clippings containing leaves are placed where llamas can get at them, there is great risk. A sprig of oleander that had been chewed on was found in the manger of an eight-month-old llama that died with signs of oleander toxicity.

In another instance, oleander leaves and branches were included with other tree cuttings and run through a shredder. The mulch was then spread along a path next to a llama pasture. Some of the dried mulch blew into the pasture. Within twenty-four hours, one of three llamas was sick and died four days later. Another llama became ill but recovered.[8]

The clinical signs of oleander poisoning are similar in all species of mammals. Llamas become anorectic and depressed and lie down unless forced to rise. The major observable sign is frequent, projectile, catarrhal to hemorrhagic diarrhea. Colicky signs may accompany the diarrhea. If the ingested dose is high, the

Figure 23.11A. Castor bean (*Ricinus communis*).

Figure 23.11B. Castor bean foliage.

Figure 23.11C. Castor bean seed.

Figure 23.12A. Tree tobacco (*Nicotiana glauca*).

Figure 23.12B. Tree tobacco (*Nicotiana glauca*).

animal may die of cardiac complications before diarrhea develops.

The cardioactive glycoside has a direct effect on the cardiac musculature. Various conduction abnormalities may be heard on auscultation over a period of a few minutes because of the rate and rhythm change. There may be bradycardia, tachycardia, drop beats, and partial and complete blocks. Impaired circulation may result in cyanosis, muscle tremors, patchy perspiration, and dyspnea. Terminally, tachycardia progresses to ventricular fibrillation and agonal struggling.

Figure 23.13. Black locust, false acacia (*Robinia pseudoacacia*).

Figure 23.14. Purple foxglove (*Digitalis purpurea*).

Figure 23.15. Yew (*Taxus* spp.).

Figure 23.16. Lantana, (*Lantana camara*).

Figure 23.17. Water hemlock (*Cicuta douglasii*).

Figure 23.18A. Milkweeds, Mexican whorled milkweed (*Asclepias fasicularis*).

Figure 23.18B. Showy milkweed (*A. speciosa*).

Figure 23.19. Oleander, (*Nerium oleander*).

Clinical signs in ruminants may develop five to twenty-four hours after ingestion. Camelids may develop clinical signs sooner because C-1 of the stomach has an absorptive mucosa (Chapter 13). Death may occur within ten hours or after three to four days. The severity of clinical signs is not directly correlated with possible mortality.

At necropsy, lesions are limited to enteritis plus petechial or ecchymotic hemorrhages of the GI serosa or of the epicardium, endocardium, or pericardium. Stomach contents of all three compartments should be carefully examined for the presence of leaf segments. Oleander leaves have a unique vein pattern, with a single midrib and parallel secondary ribs (Figure

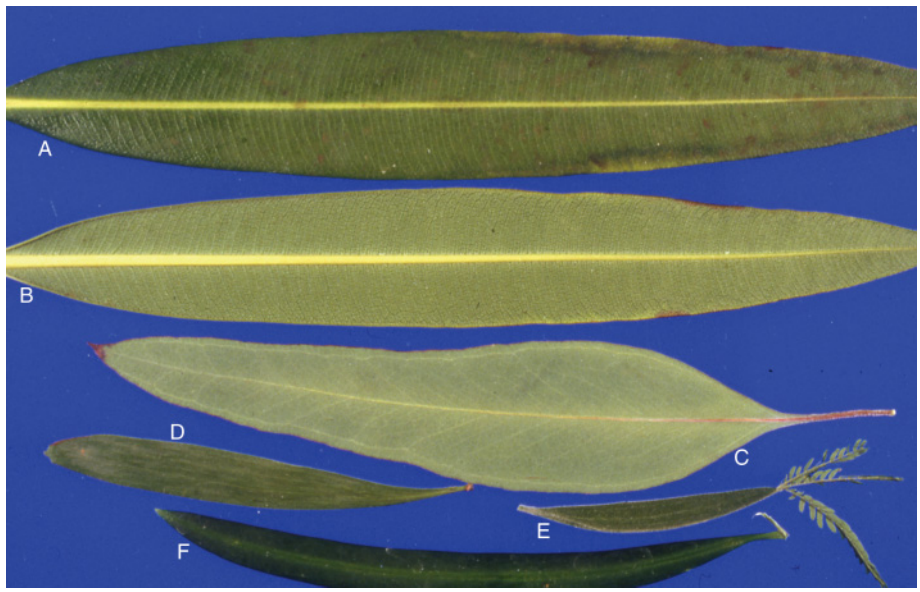

Figure 23.20A. Veination of leaves that may be confused with oleander. (A) Oleander, top view, (B) oleander, bottom view, (C) eucalyptus, (*Eucalyptus* spp.), top view, (D) acacia, (*Acacia* spp.), top view, (E) acacia, bottom view, (F) Oleander fruit.

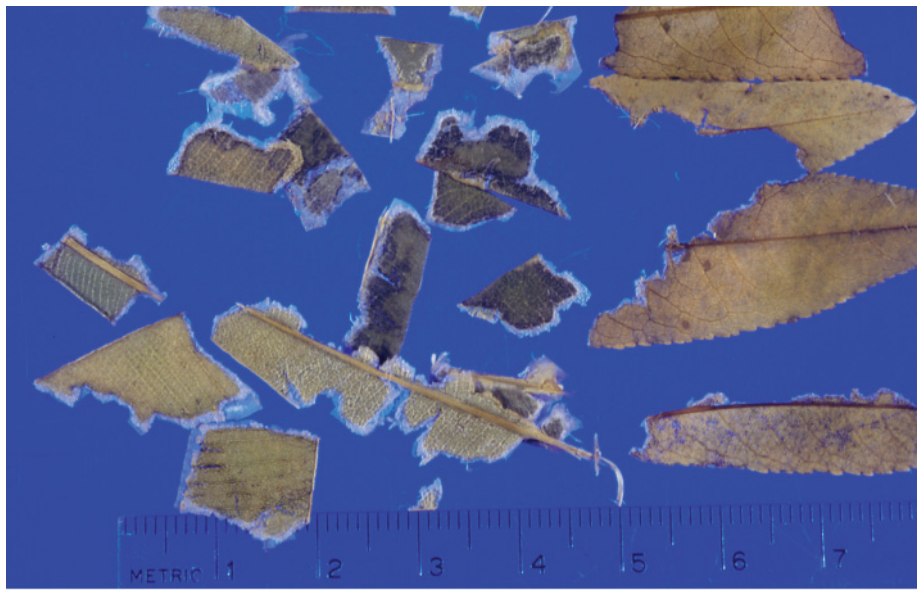

Figure 23.20B. Oleander leaf segments collected from the ingesta at necropsy.

23.20). Finding even a small segment of a leaf in the stomach contents justifies a diagnosis of oleander poisoning. A two-dimensional thin-layer chromatography test has been developed that detects oleandrin in a 0.05-ppm concentration in ingesta.[a]

Treatment of oleander poisoning is difficult. The regimen for treating an overdose of digitalis in humans is not satisfactory for camelids (lowering serum calcium by titration with sodium versenate, administration of potassium glutamate). The use of various drugs to counter arrhythmias has not been worked out. If a definitive diagnosis is made, the logical recourse is to remove the remaining oleander material from the digestive tract. This means that a gastrostomy must be performed (Chapter 6). All of the contents of C-1 must be removed and the lumen carefully washed. Small leaf segments left in the stomach may be lethal. The microorganisms from C-1 must be replaced by a transplant from another camelid or a cow.

Unfortunately, patients with circulatory deficiencies are not good surgical anesthetic risks. Gastrostomy may be performed in the standing camelid under local anesthesia, but it is more difficult.

Animals in a herd are not all equally curious and all may not consume leaves in their enclosure. However, all exposed animals should be examined for cardiac irregularities. A cathartic may be administered to hasten emptying of the GI tract. Oleander is so toxic it is dangerous to wait for signs to appear.

Pyrrolizidine Alkaloid Poisoning[34]

Dozens of pyrrolizidine alkaloids are found in plants broadly distributed throughout the world. These alkaloids vary in toxicity from no effect to pronounced effect on the liver of the host. Many animals have some innate resistance to pyrrolizidine alkaloids. The alkaloid must be metabolically altered to an active metabolite before toxicity occurs, and certain species such as sheep degrade the alkaloid in the digestive tract and liver, avoiding production of the active metabolite.

Of the domestic animals studied, horses are the most susceptible; next are cattle, then swine. Humans seem to be as susceptible as horses. It is not known just where camelids fit on the scale, but in an unreported study conducted on three llamas in Oregon, clinical signs and/or lesions could not be produced. The plant material used was tansy ragwort (*Senecio jacobea*), which is known to be highly toxic. The llamas would not eat the dried plant material, so the material was suspended in a slurry and administered by stomach tube or via a stomach fistula. The llamas were given 50% of their body weight of the dried plant material.[b]

It is a commonly held belief the pyrrolizidine alkaloid poisoning (PAP) occurs in alpacas in Australia from consuming fireweed (*Senecis quadidentatus*) and other species of *Senecio*. While this author has not been able to find any scientific report of PAP in alpacas, clinical veterinarians in Australia have reported that the lesions of PAP occur in alpacas with hepatic insufficiency.[c]

Dieffenbachia Poisoning (Dumbcane)

Some discarded ornamental dieffenbachia (*Dieffenbachia seguine*) was fed to a llama, which immediately began to salivate, spit, cough, shake its head, and rub its mouth on a fence post.[50] Dumbcane produces the same effect on any animal that takes it into the mouth. Both oxalate crystals and a proteinaceous agent may irritate the mucous membrane, but the speed of reaction indicates a toxic effect rather than a mechanical effect. The affected llama appeared to recover within thirty minutes.

More severe signs may occur if laryngeal edema is sufficient to partially obstruct the larynx. Glossal edema may inhibit tongue mobility and swallowing. These effects may last for a number of days. The only treatment is to wash the mouth out with water from a garden hose. The animal should be monitored for signs of severe dyspnea, in which case a tracheostomy is necessary.

Yew Poisoning

Yew (*Taxus* spp.) has been known to be highly toxic to farm animals and humans for centuries. Yews are common ornamentals but should not be planted at a site where animals may gain access. A group of llamas came in contact with some yew shrubs, and although they consumed only a small amount, it was sufficient to kill some animals.

Death is so rapid that signs may not be observed. Taxine, the poisonous agent, affects the central nervous system, causing vomition, tremors, convulsions, and dyspnea. Furthermore, no lesions are produced. Unless a history of ingestion is forthcoming, the only diagnostic aid is to find the characteristic needles in the stomach. There is no treatment for yew poisoning.

False Hellebore Poisoning

Veratrum californicum and other species of Veratrum are known to produce teratogenic effects in sheep, pigs, horses, and cattle fetuses (Figure 23.1). If pregnant sheep ingest *Veratrum* on the fourteenth day of gestation the fetus is affected and the lamb will be a cyclopian (monkey faced). Lakritz has reported four cases affecting camelids in Ohio.[d] Others have reported similar cases in Oregon. The author has pictures of facial malformations of skulls from Peru. Presumably *Veratrum* sp. or other teratogenic plants are also present in South America.

Varying degrees of facial deformities and other congenital conditions are produced in sheep depending upon the precise time during the fourteenth day that the *Veratrum* is ingested. The time of insult to the fetus in camelids is not known.

Veratrum spp. contain several alkaloids, some of which are responsible for the teratogenic effects and others for general systemic affects.

Castor Bean Poisoning

Ricinus communis (castor bean, castor oil plant, palma Christi) is an erect perennial shrub of warm climates but is an annual herb in areas where frost is common, Figures 23.11A to 23.11C. Castor bean is native to the Mediterranean region but has become naturalized in many countries of the world. It is commonly grown as an ornamental throughout the world. The seeds of castor bean contain a high concentration of oil that has be used medicinally since 2000 B.C. The oil is highly purgative and several generations of people will remember having a teaspoonful of castor oil forced upon them for all sorts of maladies. The oil has also been used for fuel for lanterns and has even be used as a substitute for diesel fuel. Castor beans are

grown commercially for oil production; India, China, and Brazil are the major world suppliers.

The hard seed coat contains a water soluble substance (ricin) which is extremely toxic and is feared by disaster agencies as a potential bioterrorism agent. Ricin has been used in assassinations and has been mailed to government officials. While plant poisoning with castor bean is highly unlikely in camelids, veterinarians should recognize its potential as a bioterrorism agent and be prepared to alert appropriate authorities should a suspected case present itself.

Chemically, ricin is a protein and classified as a toxalbumin in the toxicologic literature. It is similar in action to the toxalbumin found in rosary pea (*Abrus precatorius*). As with many proteinaceous substances it is possible for humans to develop an allergic sensitivity to ricin. Ricin powder may be aerosolized and inhaled or ingested. It is readily absorbed by either route. It is not absorbed through the intact skin. Horses and humans are the most susceptible species but essentially all animals may be poisoned.

Ricin is insoluble in oil so is not present in castor oil. The seeds may contain from 0.2% to 1% of ricin. The residue left after squeezing the oil may be rendered nontoxic by steam heating, which coagulates the ricin. If castor bean seeds are ingested without breaking the seed coat, they will pass through the digestive tract without causing harm. If the seed coat is broken, as little as one seed could be fatal to a child. Four seeds could be lethal for a horse. The toxic dose for a camelid us unknown.

Clinical Signs. The classical signs of ricin toxicity are a profuse watery to hemorrhagic diarrhea associated with colic and straining. However, a high dose of ricin may cause collapse and circulatory failure before diarrhea begins. The signs may appear in a few hours following ingestion, or with lower doses it may be a few days.

The syndrome in the horse begins with colic and a pounding heartbeat that may be observed at a distance. The inciting cause of the tumultuous heartbeat is fluid being withdrawn from the circulation to enter into the intestines. The packed cell volume increases up to 60%. The blood essentially becomes a sludge that requires tremendous effort by the heart to circulate. The body temperature begins to rise and there is a leukopenia. The clinical signs to this point would indicate a possible viral infection. Other signs include anorexia, dullness, weakness, profuse sweating, and dehydration. Extravasation of plasma from the circulation leads to hypovolemia, hypotension, and potential shock. Trembling, incoordination, and convulsions may result from the hypoxemia associated with poor circulation, or be incited by ricinin, an alkaloid also contained in the seeds.

In cattle, ruminal tympany and decreased lactation may be seen in a more chronic syndrome. In human ingestion of a biotoxin, mild poisoning causes nausea, vomiting, diarrhea, and colic. In more severe cases the diarrhea may become hemorrhagic. Hypotension and liver and/or renal failure may lead to death. When ricin is inhaled signs may appear in six to eight hours and include coughing, dyspnea, arthralgia, fever, and death, which may occur in four to thirty-six hours.

Diagnosis. A history of exposure to castor bean seeds may be important. A hemogram indicates a leukopenia and elevated PCV associated with a fever. At necropsy there may be evidence of seeds in the ingesta. The intestines are ballooned with fluid with petechial to ecchymotic hemorrhages on the serosa and/or mucosa. It may be possible to analyze for ricin, but this may require contacting the local poison control center to find a laboratory capable of running the test.

Management. The only specific therapy is an antiserum that is unlikely to be available to veterinarians. Fluid therapy may be helpful in correcting the hypovolemia, hypotension, and elevated PCV. In humans, gastric lavage followed by activated charcoal may be beneficial. Oxygen insufflation is indicated.

Cyanide Poisoning

A case of lethal cyanide poisoning has been reported in two alpacas that browsed heavenly bamboo (Sacred bamboo, *Nandina domestica*), an ornamental shrub which is not a member of the grass family as is true bamboo.[e]

The poisonous principle is p-Hydroxynadelonitrile, a cyanogenic glycoside. The glycoside is rapidly hydrolized in the compound stomach, to free CN^-.

Clinical signs are typical of signs of cyanide poisoning in livestock species, including weakness, incoordination, dyspnea, recumbency, paddling the feet, and tetanic seizures. Death may ensue within fifteen minutes of the first signs. Venous blood is bright red in color. Evaluate this sign carefully because normal venous blood in SACs is brighter than in livestock species.

Alpacas exhibited depression, ataxia, head bobbing, dyspnea, bradycardia, and subnormal rectal temperatures (35.9°C, 96.6°F). Both alpacas in the cases cited above salivated profusely.

Management is difficult because of the peracute nature of the syndrome. However, cyanide poisoning is one of the few plant poisonings for which there is a specific antidote. Sodium nitrite at 10 mg/kg is administered IV to convert some of the hemoglobin to methemoglobin, which in turn combines with CN^- to form a less toxic cyanomethemoglobin. The sodium nitrite is followed by the IV administration of sodium thiosulfate at 0.25 to 0.5 g/kg to form

sodium thiocyanate from the slow release of CN^- from cyanmethemoglobin.

Nitrate (Nitrite) Poisoning[37]

Excessive nitrate may be found in water and fertilizers and from plants that are capable of accumulating nitrates. Nitrogen in various forms is frequently applied to forage crops as a fertilizer. All forms of nitrogen are converted to nitrates before being absorbed and utilized by plants. Nitrate poisoning is rare in camelids but has been reported as occurring in alpacas fed orchard grass hay (*Dactylis glomeratus*) containing high levels of nitrates.[f] In Australia, four alpacas died of nitrate poisoning after eating oat hay (*Avena sativa*) containing 3.2% nitrate.[38]

The poisonous principle is nitrate, which is reduced in the compound stomach of foregut fermenters (cattle, sheep, goats, camelids) to nitrites. Nitrites are readily absorbed from the gastrointestinal tract and disrupt hemoglobin metabolism by converting hemoglobin (Fe^{++}) to methemoglobin (Fe^{+++}). Oxygen is unable to bind to methemoglobin, thus preventing transport of oxygen to tissues. Signs of toxicity may be seen when methemoglobin levels reach 30% to 40%.

Clinical signs of acute poisoning usually appear within four hours of ingestion of a toxic dose of plants containing excessive nitrates. Signs include dyspnea, cyanotic mucous membranes, and a rapid, weak pulse. Venous blood is dark brown (chocolate) in color. Other signs of anoxia include muscle tremors, weakness, ataxia, and exercise intolerance. Terminally there may be agonal struggling or convulsions.

Management. The key to management is an accurate diagnosis. Differential diagnosis should include other hypoxic conditions such as pneumonia or cyanide poisoning. Cyanide poisoning should be eliminated as a possibility because therapy for cyanide poisoning exacerbates nitrite poisoning. Brown colored (chocolate) blood is highly suggestive; however, poisoning with sodium chlorate (herbicide, defoliant) produces the same effect on blood.

Methylene blue is a specific antidote that reduces methemoglobin back to hemoglobin. The IV dose for SACs is 6 to 10 mg/kg or 1 to 5 ml of a 1% solution, extrapolating from the cattle dose. Be cautious because an overdose of methylene blue may actually produce more methemoglobin.

Mycotoxins

Several mycotoxins produce disease in camelids.

Sporodesmiomycosis (Facial Eczyma)

Sporidesmin is a mycotoxin produced by the fungus *Pithomyces chartarum*, and is a common contaminant on perennial ryegrass (*Lolium perenne*).[11] The fungus is found worldwide. Sporidesmin causes hepatic necrosis resulting in photosensitization (Facial eczema). The disease is reported in camels in the UAE[51] and alpacas in New Zealand and Australia. Sporodesmins have been isolated from perennial ryegrass in the United States, but facial eczema has not been reported in livestock or camelids.

Diagnosis. Diagnosis is based on clinical signs and history of possible exposure to plants harboring *P. chartarum*. At necropsy the hepatic lesions are characterized by bile duct proliferation, fibrosis, and venoocclusive lesions. Take note that this is a similar finding found in pyrrolizidine alkaloid poisoning (PAP), except that in PAP there is generalized hepatocytomegaly, which is not present in facial eczema. It is also the opinion of Australian veterinarians that both hepatic necrosis from sporodesmins and PAP may be present in the same animal.

Management. There is no treatment for the hepatic necrosis. If the necrosis is mild the animal may live with diminished hepatic function. The animal should be kept in an environment devoid of exposure to the sun, and the skin lesions treated with protectants.

Aflatoxicosis[43,46,47]

Aflatoxicosis is caused by the mycotoxin aflatoxin which is produced by the fungi *Aspergillus flavus* and *Aspergillus parasiticus*. These fungi are ubiquitous and saprophytic, growing on a variety of grains and seeds including peanuts, corn, rice, sorghum, cottonseeds, and sunflower seeds. Fungal growth and subsequent aflatoxin production is enhanced by warm, moist, humid conditions and trauma to the seeds when harvesting. Humans are also affected by aflatoxins. Aflatoxicosis has been reported in camels.

Clinical Signs. Clinical signs are consistent with hepatic insufficiency. Hepatic tumors are also associated with aflatoxicosis. Immunocompromise caused by aflatoxins may foster secondary infection. Other signs include reduced appetite, weight loss, and variable icterus.

Diagnosis. At necropsy the acute lesions consist of hepatic necrosis, bile duct proliferation, and fatty infiltration of the hepatocytes. In chronic cases the lesions are similar to pyrrolizidine alkaloid poisoning, including fibrosis and hepatocytomegaly.

Ryegrass

Perennial ryegrass is the second most popular pasture grass (second to tall fescue) in the middle and northern United States. Perennial ryegrass grows best in a cool, moist climate. It is a popular pasture grass for cattle and sheep in the northern coast regions of California, particularly those within the fog belt. It is also grown in drier climates where irrigation is available. Ryegrass may be involved with three syndromes produced by separate mycotoxins.

1. Gangrene of the extremities: Ryegrass is host to ergot (*Claviceps purpurea*). Consumption of ergotized seed heads may result in gangrene of the extremities (feet, tips of ears, and tip of tail). Ergot and other fungi require specialized environmental conditions for maximum production. Ergot infects the shoots as they germinate and ultimately replaces the seed head. Ergot-caused gangrene develops in the autumn following a wet spring that fosters growth of the fungus. Such gangrene has not yet been reported in alpacas or llamas, but there is no reason to assume they would not be susceptible if exposure occurs.
2. Ryegrass staggers: The clinical disease has been diagnosed in cattle, sheep, horses, alpacas,[30,39,40,41,42] and llamas in New Zealand, Australia (Victoria, Tasmania, South Australia, and West Australia), and the United States. For many years the toxic agent(s) causing ryegrass staggers remained unknown. In California, it is now believed that mycotoxins (lolitrem-B, etc.) produced by the endophyte fungus *Neotyphodium (Acremonium) lolii* is responsible for the clinical signs. *N. lolii* is found concentrated in the stems, in a 2-cm (1-in.) section just above the root crown.

   Affected animals may appear to be normal until excited or agitated, when the prominent sign, shaking of the head and neck, appears. Moderate signs include stiffness when walking or trying to run, swaying, incoordination, falling, and a high-stepping gait when forced to run. Severely affected animals may stand in a sawhorse stance and sway back and forth. Some animals may be unable to rise, may paddle, have marked head shaking, and have their heads pulled backward. Affected animals being driven may fall and roll down a hill or into a ravine and be unable to extricate themselves from a predicament. Drowning is a particular hazard as animals may fall into a body of water.

   Most animals recover with no residual effects if removed from pastures containing the source of the mycotoxin. Even animals exhibiting severe neuromuscular signs usually recover if left unmolested. Clinical signs usually disappear within one to three weeks after no access to the source of the fungus.

   The climatic conditions that foster ergot growth also foster endophyte proliferation, in which case the seeds become infected with the fungus. As the new plant grows, the infection appears in the lower stem. Ryegrass staggers is a late summer and fall disorder.

   A history of grazing on ryegrass is paramount. Clinical signs may develop within seven to fourteen days of continuous ingestion of endophyte-infected ryegrass. Laboratory tests may be performed.

   No treatment is available for affected animals, except for removal of the animals from the source of the mycotoxin. Affected animals should be kept calm; moving or handling should be minimized or avoided once the condition is recognized in a herd.
3. Facial eczema is a secondary photosensitization caused by the hepatotoxic mycotoxin sporidesmin, produced by *Pithomyces chartarum*, as already described.

Phalaris Staggers

Phalaris grasses have been imported to the United States as pasture grasses and have become naturalized. Hardinggrass (*Phalaris aquatica, P. tuberosa*), reed canarygrass (*P. arundinacea*), and annual canarygrass (*P. minor*) grow in the United States.

Poisoning is usually seen when there is new growth of harding- or canarygrass, especially when new green shoots appear around standing dry grass. Animals seek out the tender green shoots.

In sheep, phalaris staggers is a progressive, usually irreversible, fatal brain disorder that develops two to three weeks after sheep graze on pasture, and it may develop days after removal from the pasture. In the early stages of this syndrome the clinical manifestations are similar to those seen in ryegrass staggers, including tremors, head nodding, staggering, a high-stepping gait, and falling when excited. The differences are that an uneven heartbeat is prominent in phalaris staggers, the tremors don't abate, and the severity progresses until the animal dies. Affected sheep often walk stiff-legged and may drag their hindquarters (unable to flex the hocks). Some may walk on their knees. Animals that collapse exhibit convulsions, paddling, muscle tremors, nodding of the head, and salivation.

Cattle also may be affected, but the signs are usually restricted to stiffness of the hocks and dragging the hind toes. Only severely affected animals show intense nervous signs similar to those seen in sheep.

Phalaris poisoning has been diagnosed in alpacas in Australia in regions where sheep losses also occur from poisoning. Signs are similar to those seen in sheep. High mortality in affected animals is characteristic. The lesions in the brain are also characteristic.

No successful treatment has been found once the animal is poisoned. Many years ago a report was published indicating that cobalt (Co) prevented the development of phalaris staggers in sheep. Although the use of Co as a preventive has been long practiced in

Australia, recent investigations have cast doubt on the effectiveness of such supplementation for sheep and cattle in the United States. Nothing is known about the effectiveness of Co as a preventive in alpacas or llamas.

Fluroacetate Poisoning

Camels may be exposed to fluroacetate compounds by ingesting poisonous plants containing the compounds. Camels have been poisoned in Australia. Any camelid may come in contact with the rodenticide sodium monofluroacetate (Compound 1080). Monofluroacetic acid occurs naturally in several species of plants including *Dichapetalum cymosum* (gifblaar) in South Africa, *Gastrolobium grandiflorum* (desert poison bush) and *Oxylobium parviflorum* (box poison) in Australia, and *Palicourea margravii* (mata) in South America. No fluroacetate plants occur in North America.

Sodium monofluroacetate (Compound 1080) and Fluroacetamide were developed as rodenticides, which are extremely toxic and are banned in many countries. They may be used by professional exterminators for specific purposes in the United States. The only legal use is for collars on sheep and goats for the control of coyotes (*Canis latrans*). Unfortunately, unused packets of 1080 may persist in farm and ranch medical storerooms, or even be used illegally.

Mechanism of Action. In the normal Kreb's (Tricarboxylic acid) cycle, acetate combines with CoA to form acetyl-CoA, which in turn is acted upon by enzymes that convert acetyl-CoA to citrate, then to isocitrate, and through systematic decaroxylation to carbon dioxide, water, oxaloacetic acid, and ATP. Fluroacetate, which itself is a relatively harmless compound, is metabolized to fluroacetyl-CoA and ultimately to the highly toxic (2R-3R)-1-flurocitrate which disrupts the cycle and causes a catastrophic loss of cellular respiration in all organ systems.

Signs of Plant Ingestion. A latent period of four to twenty-four hours allows for plant digestion in the stomach, absorption into the blood stream, and subsequent disruption of the tricarboxylic acid cycle. The lethal dose varies with species. The western grey kangaroo (*Macropus fuliginous*) is immune to toxic effects from ingestion of fluroacetate-containing plants, while its cousin, the eastern grey kangaroo (*Macropus robustus*), a native to eastern Australia where the plants don't grow, is highly susceptible. Several species of wild antelope in South Africa are able to consume gifblaar with impunity.

Signs of 1080 Exposure. There is considerable difference in the lethal dose in various species. The latent period in 1080 poisoning is much shorter (one-half to two hours). The major effects may be either on the central nervous system or the heart. Signs of nervous system involvement predominate in carnivores (dogs and cats), whereas cardiac effects are more common in cattle and sheep. Signs in cattle and sheep include marked cardiac arrhythmias, tachycardia, weak pulse, and ventricular fibrillation. Animals are often found dead. The clinical syndrome associated with exposure to 1080 in camelids has not been reported.

Diagnosis. The action is biochemical and usually there is insufficient time for lesions to develop. Chemical analysis of ingesta may yield fluroacetates.

Management. The rapid death usually precludes an opportunity to treat.

Fescue Poisoning

Tall fescue (*Festuca arundinacea*) is an important pasture grass in the United States, but under certain situations it may be responsible for mycotoxicosis from the fungus *Neotyphodium (Epichloe) typhina (Acremonium coenophialum*). The syndromes produced are as follows:

1. Fescue foot: Cattle and sheep grazing endophyte-infected pastures in the late summer and fall may develop gangrene of the extremities, similar to that caused by ergotism. Clinical signs include lameness, arched back, and diarrhea that develop within a few days to months after grazing of infected pastures. More severe signs appear as the disease progresses, including loss of body weight; emaciation; and gangrene of the tail tip, hooves, and ears.
2. Summer slump: Cattle and, possibly, llamas grazing tall fescue pastures in the summer may experience weight loss, a dull rough hair coat with failure of winter coat shedding, anorexia, and elevated body temperature (this is critical for llamas that normally may have a higher body temperature in general—the added effect of the mycotoxin could trigger hyperthermia).
3. Reproductive problems in mares: Prolonged gestation (thirteen to fourteen months), abortion, difficult birth, weak neonates, high foal mortality, thickened placenta (foal may not be able to escape the amnion), retained placenta, and decreased milk production (agalactia) have all been observed in mares grazing endophyte-infected tall fescue pastures during late gestation. Foals from affected mares may have failure of passive transfer or starve from lack of milk. Agalactia has been observed in llamas in Kentucky.

Forty percent or more of the tall fescue pastures in the United States are thought to be infected with *A. coenophialum*. Pastures may be plowed up, fallowed, and replanted with endophyte-free tall fescue seed. Several endophyte-free strains of tall fescue have been developed by researchers in agronomy departments of

agricultural universities. Pregnant females should be removed from endophyte-infected tall fescue thirty to ninety days prior to parturition. Mammary gland development should be monitored. Parturition should be attended and the cria monitored for colostrum intake and adequate nourishment.

Miscellaneous Plant Poisonings[3,13,16,35]

See Tables 23.3 and 23.4.

Acorn poisoning (tannin toxicity) has been diagnosed in our clinic. Predisposing factors include heavy acorn production and individual addiction. Clinical signs include constipation followed by a black, tarry diarrhea. The ultimate syndrome is that of renal failure. Other signs include anorexia, depression, colic, gastrointestinal atony, anuria, and perirenal edema. On clinical pathology, the BUN is elevated (136 mg/dl) as is creatinine (11.5 mg/dl)

Mechanically Injurious Plants

Many plants and grasses have fruits or seeds that contain barbs or hooks, which aid in their distribution within the environment. Grass awns, clover burs, and other plant parts catch on the fiber coat and work their way deeply into the coat, much to the chagrin of camelid groomers. Of greater concern are grass awns or plant foreign bodies that lodge in the conjunctival sac, ear, nostril, or mouth.

Foxtails (*Hordeum* spp.) (Figures 23.21A to C) are a special problem in the western United States. These grasses are widespread weeds of pastures and hayfields. If weedy hay is fed to llamas, the awns may lodge alongside the tongue and in the buccal cavity. The tongue of the llama is not highly mobile and rarely protrudes from the mouth. The immobility of the tongue may inhibit the clearing of foxtails from the mouth.

Foxtails have also been extracted from the conjunctival sac and the external ear canal, producing signs of head shaking, head tilt, exudation from the ear canal, lacrimation, photophobia, blepharospasm, and depression. In one case, the foxtail had penetrated the tympanic membrane, entered the middle ear, worked its way through the inner ear and the temporal bone, and finally became lodged in the contiguous brain stem, producing an abscess and subsequent death of the llama. See Chapter 19 for a discussion of ear disorders.

Another mechanically injurious plant is yellow bristle grass (*Setaria lutescens*) (Figure 23.22). In the

Figure 23.21A. Foxtail (*Hordeum jubatum*).

Figure 23.21B. Foxtail.

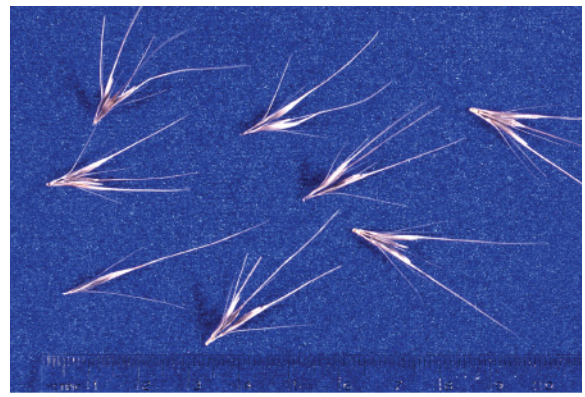

Figure 23.21C. Foxtail.

Figure 23.22. Yellow bristle grass (*Setaria lutescens*).

eastern United States *Setaria* spp. is given the name of foxtail. The grass causes no problem in a pasture; in fact, it is probably good forage. The problem arises with dried grass in hay. The dried brittle awns of the seed head become tiny needles. Yellow bristle grass is a common weed of irrigated alfalfa hay. If the grass constitutes more than 25% of the hay, there is a risk of mechanical injury to animals that consume the hay.

The awns penetrate the mucous membranes of the tongue, lips, gingiva, and buccal cavity. Ulcers varying in size from 1 to 3 cm in diameter may be characterized by the protrusion of dozens of awn segments. The lips may be swollen. Salivation is common, and prehension and mastication are inhibited.

In a large herd of alpacas in Washington, fifty or sixty animals were affected.[8] The first sign noted in the alpacas was a major weight loss as a result of anorexia. Upon examination, the practitioner noticed ulcers and vesicles and alerted federal authorities. Fluid was withdrawn from some of the larger vesicles and submitted to an appropriate laboratory for evaluation for foreign vesicular diseases. All tests were negative for vesicular diseases.

Once the vesicle ruptured, a raw ulcer formed. Deep scrapings from these ulcers yielded awns of the grass embedded in the tissue, but awns did not protrude from the surface of the ulcer as the author has seen in horses and cattle. The awns of yellow bristle grass are normally straight, but in the moist environment of the ulcer they became spiraled like a coil spring.

Ideally, the awn segments should be curetted from the surface of each ulcer. The application of disinfectants or other medication to the ulcers is superfluous. The weedy hay must be eliminated from the diet.

In the Washington case, the contaminated hay was eliminated and the herd treated with broad-spectrum antibiotics.

Tarweed (*Hemazonia* spp.) exudes a dark resin onto the leaves and stems that may stick to the fiber and skin of grazing animals (Figures 23.23A, B).

Figure 23.23A. Plant resins on face, neck, and limbs from grazing in tarweed (*Hemazonia kelloggii*).

Figure 23.23B. Plant resins on face, neck and limbs from grazing in tarweed.

Snake Envenomation

Venomous snakes are found throughout the world and are likely to be as hazardous to camelids as to livestock species, horses, and humans. The prevalence of snakebite in camelids depends on the curiosity of the individual, the environment in which the camelid is found, and the species of snakes found in the area. Most venomous snakes are quick to move away from an encounter unless startled or in a situation wherein they are unable to retreat.

Four types of venomous snakes are found in the United States: rattlesnakes (*Crotalus* spp.), copperhead (*Agkistrodon contortrix*), water moccasin (cottonmouth) (*Agkistrodon piscivorus*), and coral snake, (*Micrurus* spp. and *Micruroides euryxanthus*).[23]

Copperheads are found in eastern North America, but their venom has low toxicity, and serious envenomation is unlikely to occur in camelids. Water moccasins are found in the swampy, marshy areas of southeastern North America, and camelids are unlikely to encounter them. Coral snakes are small, inoffensive, secretive snakes of Florida, Texas, and Arizona and pose little threat to camelids. The other snakes are likely to be a hazard to camelids.

Camelids, especially juveniles, are curious and may be bitten on the nose while investigating a strange animal. Nose bites are especially hazardous to SACs because local swelling may occlude the nostrils. Because SACs are primarily obligate nasal breathers, dyspnea and suffocation may ensue.

Figure 23.24. Rattlesnake bite on muzzle four hours after bite.

Figure 23.25. Rattlesnake bite twenty-four hours after bite.

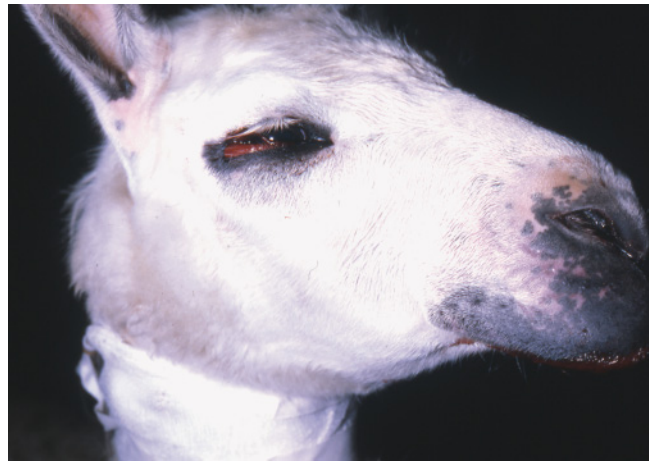

Figure 23.26. Rattlesnake bite forty-eight hours after bite.

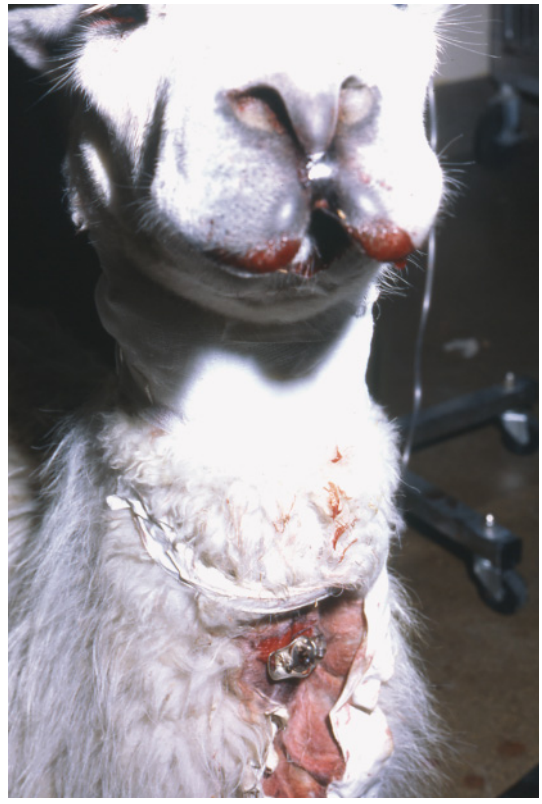

Figure 23.27. Rattlesnake bite after forty-eight hours. Tracheostomy tube in place.

Clinical signs of rattlesnake bite include local tissue swelling at the bite site that spreads proximally and may involve any tissue or organ. Figure 23.24 shows a llama four hours after a bite; Figure 23.25, twenty-four hours after the bite; Figures 23.26 and 23.27, forty-eight hours after the bite; and Figure 23.28, seventy-two hours after the bite. Sloughing of the skin may be seen in serious envenomation (Figures 23.29A and B). Bites on the limb show unilateral edema of the limb proximal to the bite.

Systemic manifestations of rattlesnake envenomation are generally absent or minimal in large animals such as camelids. However, the bite from a large eastern diamondback rattlesnake (*C. adamanteus*) could produce effects on the kidney and cardiovascular system (hypotension) as well as the local necrotizing effects.

The diagnosis may not always be clear if the bite was not observed, because trauma may produce similar signs. Supportive and symptomatic therapy

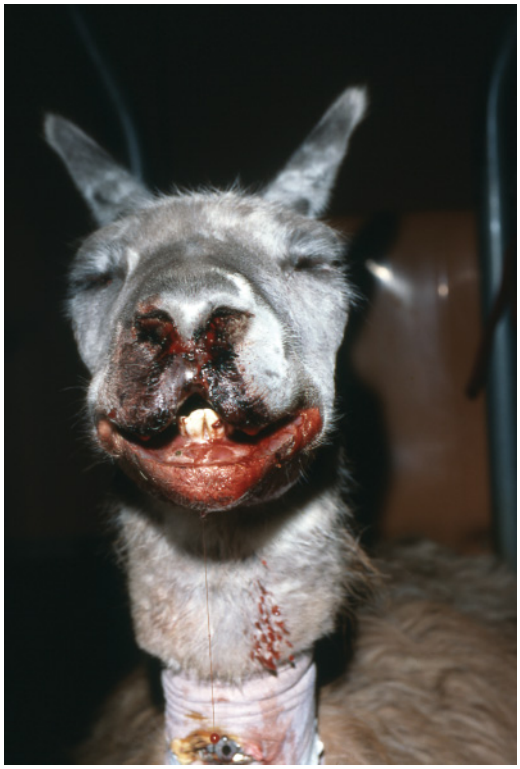

Figure 23.28. Rattlesnake bite seventy-two hours after bite.

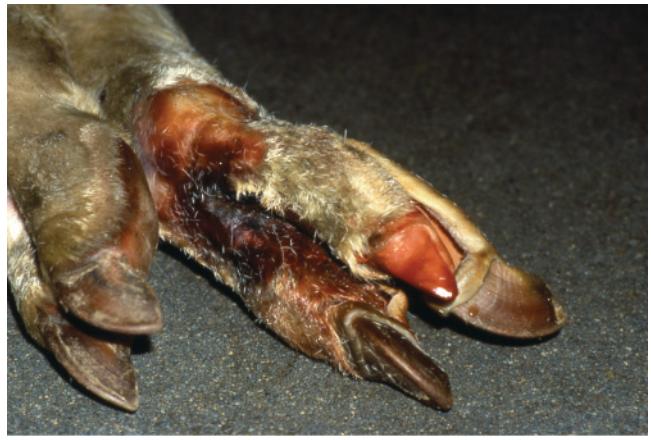

Figure 23.29A. Sloughing of the skin on the foot following rattlesnake bite.

Figure 23.29B. Sloughing of the skin on the foot following rattlesnake bite.

may be instituted while monitoring the progress of the swelling. In some regions of the United States, malignant edema (*Clostridium hemolyticum*) may be a sequel to rattlesnake bite.

The only specific treatment for crotalid envenomation is the administration of polyvalent antivenin.[23] The amount of antivenin to be used depends on the amount of venom injected by the snake, not the size of the victim, and because that is not possible to determine, usually, multiple vials (two to four) should be administered intravenously. Antivenin is prepared from horse serum and may sensitize the camelid to future use of any equine-prepared serum product. Immediate medical treatment may require that a tracheostomy tube be placed and maintained until the animal can breathe properly with the orifice of the tube occluded; this usually occurs within two to three days. Most bites are on the muzzle, so a first aid procedure that may be recommended is to place an 11-cm (4-in.) long, 1-cm (1/2-in.) wide tube in the nasal passageway. The swelling occurs around the tube, maintaining air flow.[23] Alternatively, the inside roller of a hair curler may be used.

Early in the course of a bite, cold water packs or spraying with water from a garden hose tends to restrict spread of the edematous process. After twenty-four hours, the use of warm water packs and spray is indicated to enhance circulation and drainage of accumulated fluid and tissue breakdown products. Corticosteroids have been administered but are of questionable value. Antihistamines are contraindicated because of their potential hypotensive action, which may be additive to the hypotensive action of the venom. For more details on snakebite and therapy, consult the reference cited.[23]

Snakebite in camels and SACs may occur in any country where the camelids and venomous snakes coexist. Camelids do not naturally inhabit the tropics or densely forested areas of the world so their being at risk from some venomous species is remote. Nonetheless, camelids may be maintained in zoological gardens in such habitats and veterinarians working in those areas should be aware of snakes in their area.

Australia, Africa, Asia, and the Middle East each have their own species of venomous snakes to deal with. The reader is referred to publications covering local snakebite or Fowler[23]. Following are lists of

selected venomous species according to geographical areas. No attempt is made to be all inclusive.

Mexico and Central America

Neo-tropical rattlesnake (*Crotalus durissus terrificus*)
Western diamondback rattlesnake (*Crotalus atrox*)
Fer-de-lance (*Bothrops asper*)
Cantil (*Agkistrdon bilineatus*)
Bushmaster (*Lachesis muta*)

South America

Neo-tropical rattlesnake (*Crotalus durissus terrificus*)
Fer-de-lance (*Bothrops asper*)
Several species (*Bothrops* spp.)
Bushmaster (*Lachesis muta*)

Australia

Tiger snake (*Notechis scutatus*)
Taipan (*Oxyuranus scutallatus*)
Common Brown snake (*Pseudonaja textilis*)
King brown snake (*Pseudechis australis*)

The venomous snakes of Australia are in the Elapidae (cobra) family. Their venom is neurotoxic but varies in the syndrome produced. The only specific treatment is an appropriate antivenin.

Africa: Has both Elapidae and Viperidae venomous species.

Egyptian cobra (*Naja haje*)
Black mamba (*Dendroaspia plylepis*)
African puff adder (*Bitis arietans*)
Gaboon viper (*Bitis gabonica*)
Rhinoceros viper (*Bitis nasicornis*)

Miscellaneous Bites and Stings[23]

Occasionally, all animals are stung or bitten by insects or arachnids (wasps, bees, ants, mosquitoes, and spiders).[10] Reactions vary but are unlikely to produce serious illness. The local inflammatory response produced by venom injection cannot easily be differentiated from contusions or foreign body penetration (slivers). Multiple bee or wasp stings may produce systemic manifestations, and horses and cattle have died from the envenomation when subjected to a swarm attack from a disturbed hive of bees or wasps. Africanized honeybees (*Apis mellifera adamsonii*) are now in the United States. They are no more toxic than European honeybees (*A. mellifera*) but are much more aggressive. Fire ants (*Solenopsis invicta*) found in the southeastern United States, have produced serious injury and even death to livestock tied near an anthill and unable to flee. Neonates and moribund animals are especially at risk. Camelids are at the same risk.

There have been no reports of scorpion envenomation in camels, but it is likely that stings have occurred or will occur if camelids are exposed to these arachnids. Likewise, envenomation from the black widow spider (*Latrodectus mactans*) or the brown recluse spider (violin spider, *Loxosceles reclusa*) has not been reported. Both of these spiders are poisonous to humans but are unlikely to inflict other than transiently painful bites to animals. The bite of the brown recluse spider (*Loxocoles* sp.) causes a slow-healing ulcerous wound in humans, while the black widow spider causes muscle spasms and CNS depression.

Drugs[15]

No drugs have been cleared for use in camelids by the Federal Drug Administration in the United States, and none have been tested for efficacy or safety in camelids. Extra-label usage of medications must be carried out according to the rules promulgated by the FDA. Nonetheless, it is necessary to use these drugs to practice good medicine and surgery. That some risk may be involved in such use must be recognized. On balance, thousands of experiences indicate that camelids tolerate the use of drugs as well as other livestock species. Empirical dosages are given, usually based on size and consideration of doses for cattle and sheep. No unique drug idiosyncrasies have been reported in camelids, but drug toxicities have been seen.

Suspected toxicity of chlorpyrifos was described previously. Lidocaine toxicity is described in Chapter 5. Aminoglycoside antibiotics may be nephrotoxic. Gentamicin has produced renal tubular degeneration in a llama. The signs of gentamicin poisoning are similar to acorn poisoning, including weight loss, dehydration, and loose stool, along with elevated BUN (200 mg/kg) and creatinine (21.5 mg/kg)

Ionophore Poisoning[1,28]

Ionophores are naturally occurring antimicrobial drugs that are widely used to control coccidial parasites of poultry, cattle, and swine. They also enhance feed utilization and thus are considered growth promoters.[1] Ionophores are produced by bacteria in the Actinomycetales order. Three common ones and most likely to be involved with camelid poisoning are monensin, salinomycin, and lasalosid.

Epidemiology. Poisoning has been reported in camelids as a result of feed mixing errors, wherein a mill miscalculated the dose in a feed or from feed that is not supposed to contain the drug but has been mixed in improperly cleaned mixers that were used for other batches of feed.

Clinical Signs. Ionophores inhibit cellular metabolism and may affect any organ system of the body. The syndrome exhibited may differ according to the species affected. In camels, signs include weakness, hind limb incoordination and death.[51]

In llamas and alpacas the following signs may be seen: anorexia, weakness, trembling, apparent depression, incoordination, decreased anal and tail tone, difficult urination, dyspnea, tachypnea, exercise intolerance, and death.

Diagnosis. Clinical signs and history of exposure are helpful. Feed analysis for ionophores is more definitive than tissue analysis of a suspected victim. Peracute death may occur prior to lesions being produced. Subacute lesions may include myocardial necrosis, epicardial and endocardial hemorrhage, and hepatic and renal necrosis. Rhabdomyositis may also be present.

Clinical pathology is characterized by an increased level of liver and kidney enzymes, elevation of the PCV, and low levels of calcium and potassium.

Differential diagnosis must consider clostridial myositis, selenium toxicity or deficiency, severe muscle trauma, and exertional myopathy.

Management. There is no specific treatment. On a herd basis, if ionophore toxicity is suspected, administer activated charcoal and a cathartic. Symptomatic treatment is appropriate, but the prognosis is unfavorable.

Prostaglandin Toxicity

Prostaglandins are used extensively in veterinary medical practice. Numerous commercial products result in different pharmacologic effects, depending on the form of prostaglandin employed. One such, $PGF_{2\alpha}$, is prescribed as a smooth muscle activator or for its luteolytic action. The effects of $PGF_{2\alpha}$ are vasoconstriction, bronchiolar constriction, intestinal muscle contraction, uterine muscle contraction, and luteolysis if a corpus luteum is present.

Tromethamine is an aqueous solution, with benzyl alcohol as a preservative. Generally, $PGF_{2\alpha}$ is considered to be nontoxic. However, there are species differences as to effective doses. The toxic dose for camelids is unknown, but three llama females died shortly after $PGF_{2\alpha}$ was used to produce luteolysis. In all cases the llamas were determined to have retained corpora lutea.

Case one was a sixteen-year-old female llama with a cria at side. Dinoprost tromethamine (15 mg total dose) was administered intramuscularly at 3 p.m. The llama was found dead the next morning. Excessive fluid was found in the lungs at necropsy.

Case two was a seven-year-old female. Dinoprost tromethamine (20 mg total dose) was administered intramuscularly at 7 p.m. She was found dead one hour later. Pulmonary edema and intestinal hyperemia were noted at necropsy.

Case three was a mature female. Miscommunication resulted in an overdose (50 mg) of dinoprost tromethamine administered intramuscularly. She was returned to the owner's ranch but died within an hour from what was described as anaphylactic shock. Although the response was immediate, it is unlikely that this was an anaphylactic response but was rather a direct toxic effect of $PGF_{2\alpha}$.

Three other females have exhibited dramatic, but temporary, clinical responses to therapeutic doses of tromethamine.

The three llamas that died were not observed prior to death. The presence of pulmonary edema would indicate that they were dyspneic. The three females that developed transient signs reacted within five minutes. Two females rolled on the ground and foamed from the mouth. The client described periodic abdominal spasms, somewhat like hiccups.

The pharmacologic effect of producing constriction of the intestinal musculature is likely to produce spasmodic colic and the signs of colic as described. One llama was observed to become extremely dyspneic, with open mouth breathing and marked salivation within five minutes of a therapeutic dose (Figures 23.30A and B). The three dead animals had pulmonary edema and, if they had been observed, would have exhibited dyspnea and terminal signs of suffocation. None of the animals was examined by a veterinarian, so it is not known whether the heart rate was altered or blood pressure was elevated. Prostaglandins are often used to produce abortion or initiate parturition of a term fetus. This action requires forty-eight to seventy-two hours.

Based on limited observations and evaluations, it appears that colic is the primary and immediate clinical sign to be observed. Bronchiolar constriction and pulmonary edema produce dyspnea and are probable causes of death.

Therapy. Unfortunately, no antiprostaglandins are available that can be given intravenously to counter the toxic effects of $PGF_{2\alpha}$. Signs develop rapidly and culminate within an hour by recovery or death. Unless the veterinarian remained for an hour after administration of the drug, the animal would be dead before it could be reached.

Oxygen insufflation, via a face mask, may aid the llama with mild pulmonary edema or bronchiolar constriction. If pulmonary edema is severe, the only effective treatment is administration of oxygen under positive pressure. This requires anesthesia and tracheal intubation and is not likely to be available in the field.

Prostaglandins are valuable therapeutic agents for dealing with infertility, but their use is not without risk. The animal should be kept under observation for at least an hour following administration of $PGF_{2\alpha}$. An apparatus for administration of oxygen should be available in the event of a reaction.

Figure 23.30A. Prostaglandin $F_{2\alpha}$ toxicity.

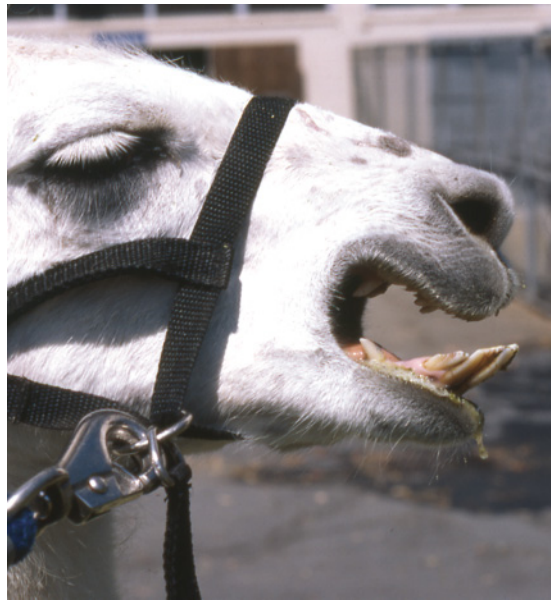

Figure 23.30B. Prostaglandin $F_{2\alpha}$ toxicity.

Cobalt Salts

Cobalt toxicity associated with treatment for phalaris staggers is described in Chapter 2.

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[c] Dr. Andrew Dart, University of Sydney, personal communication, May 2009.

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24

Conformation and Gaits

Conformation is the proportionate shape or contour of an animal resulting from the appropriate arrangement of all body parts (balance).[2,3,5] Each species of animal has a unique conformation that fits it for a specific function. Horses are built for running. Cattle produce milk and meat. Llama conformation suits them well for carrying loads in inhospitable places. Alpacas have produced fiber for untold generations of humans. Camels have been a beast of burden, used in warfare and sport, and used for food (milk and meat) and fiber for centuries. Camelid owners should seriously consider the importance of conformation in any breeding program, and veterinarians should be in a position to evaluate conformation and advise clients.

Conformation is inherited but not in a typical Mendelian fashion with dominant and recessive genes. Rather, it results from the cumulative effect of a large series of alleles that influence conformation positively or negatively. Furthermore, environment and nutrition may modify the expression of genetic predisposition to a given trait.

In regard to changing a trait with a breeding program, conformation falls into the midrange of difficulty. Using 0 to 1 to represent the ultimate ease of changing a trait, fiber, ear shape, size, and color would lie between 0.4 and 0.7. Conformation would be 0.25 to 0.39, and general fertility and health factors would be 0.1 to 0.24.[a]

No breed standards have been established for camelid conformation. However, the general principles of form, function, and balance apply to all animals. The terminology is that customarily used in describing horses and livestock. Many conformation faults become visible when a llama or alpaca moves at a walk or pace. Seeing a camelid in motion is crucial to optimal evaluation. Observation is the key element in evaluating conformation.

Veterinarians should begin an evaluation with the premise that no individual camelid has perfect conformation; thus a priority of conformation faults should be established. A conformation trait may be inconsequential to the health and well-being of an animal but have significant esthetic appeal (color, size, and shape of the ears). Some traits are acquired and may be classified as blemishes (scars from trauma, frostbite of ears). Many traits are classified as undesirable faults and most animals have one or more of these, exhibited in various degrees. Inherited defects are the most serious and should be avoided if at all possible. None of the congenital defects of llamas and alpacas have been proven to be inherited, but sufficient information is available from studies of other species to be reasonably sure that certain traits are inherited. In the show ring, traits that should constitute grounds for disbarring animals from showing (unacceptable faults) include cryptorchidism (only one testicle visible), evidence of surgery to correct a fault, umbilical hernia, and significant malalignment of the dental pad and incisor teeth.

Many conformational faults may be seen in the neonate. Owners are frequently alarmed to see a calf-kneed cria walking on its fetlocks. However, judgment should be reserved until the cria is a few weeks old to give tendons and ligaments an opportunity to strengthen, after which the fault may disappear.

CONFORMATION CHARACTERISTICS

An overview of the llama form is illustrated in Figure 24.1. The skeleton of an alpaca is illustrated in Figure 24.2. A Bactrian camel skeleton is illustrated in Figure 24.3.

Medicine and Surgery of Camelids, Third Edition by Murray E. Fowler

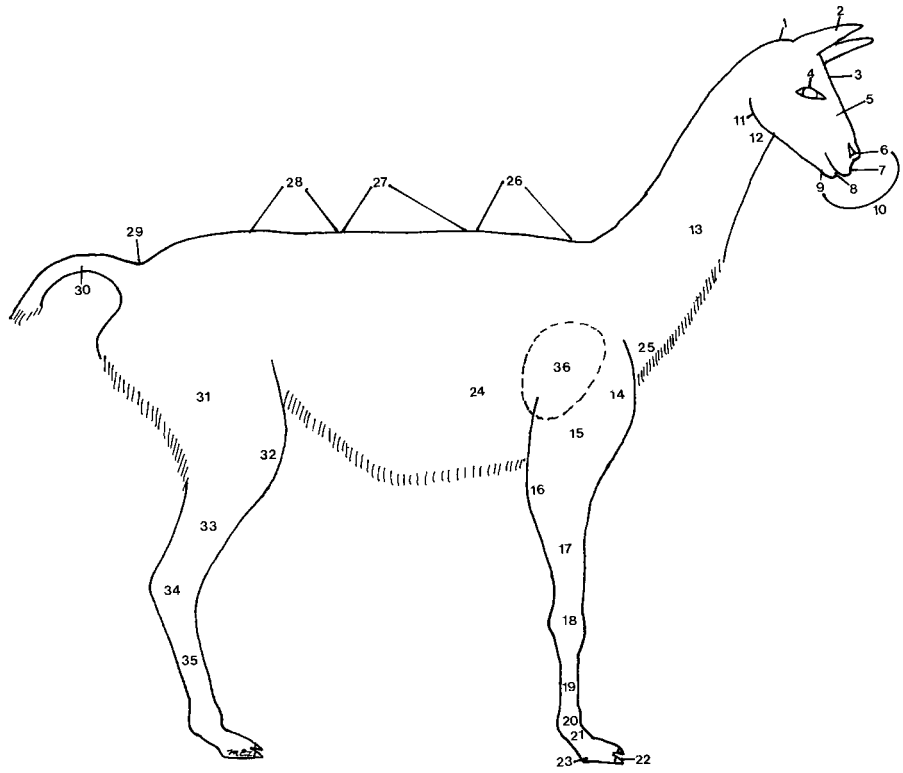

Figure 24.1. Surface body areas of a llama. (1) Poll, (2) pinna, (3) forehead, (4) eye, (5) face, (6) nostril, (7) upper lip, (8) mouth, (9) lower lip, (10) muzzle, (11) angle of the jaw, (12) throat latch, (13) neck, (14) shoulder, (15) arm, (16) elbow, (17) forearm, (18) knee, (19) cannon bone, (20) fetlock, (21) pastern, (22) sole, (23) toenail, (24) ribs, (25) chest, (26) withers, (27) thoracic vertebrae, (28) lumbar vertebrae, (29) tailhead, (30) tail, (31) upper leg, (32) stifle, (33) gaskin, (34) hock, (35) hind cannon, (36) cardiac silhouette.

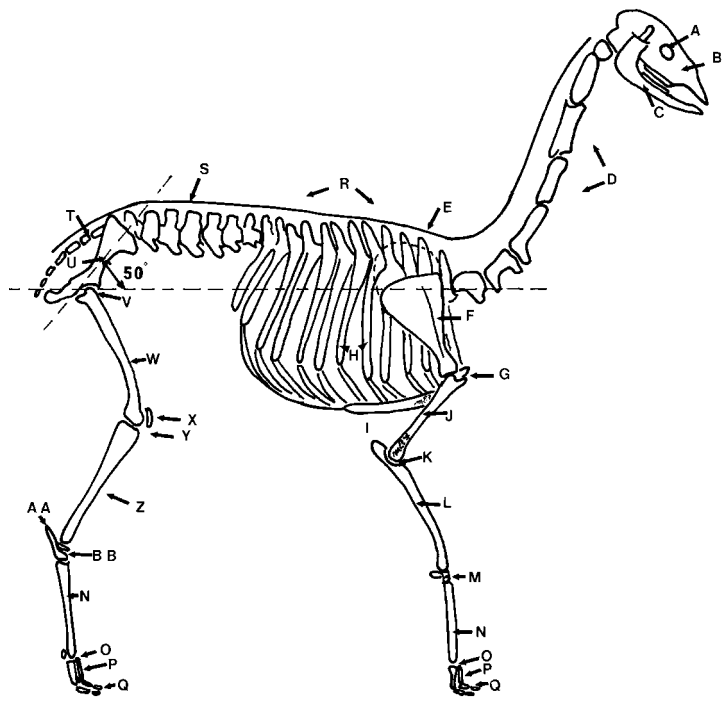

Figure 24.2. Skeleton of an alpaca. (A) Orbit, (B) maxilla, (C) mandible, (D) cervical vertebrae, (E) withers, (F) scapula, (G) shoulder joint, (H) ribs, (I) sternum, (J) humerus, (K) elbow, (L) radius, (M) carpus, (N) metacarpus, (O) fetlock, (P) pastern bones, (Q) P-3, (R) thoracic vertebrae, (S) lumbar vertebrae, (T) coccygeal vertebrae, (U) pelvis, (V) hip joint, (W) femur, (X) patella, (Y) stifle, (Z) tibia, (AA) tuber calcis, (BB) tarsus.

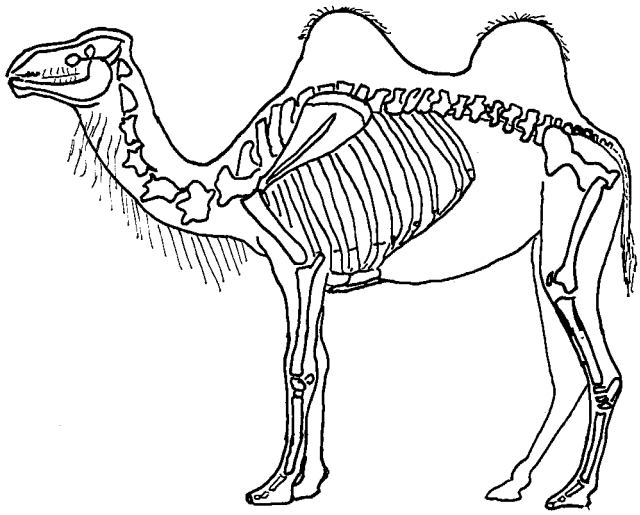

Figure 24.3. Skeleton of a Bactrian camel.

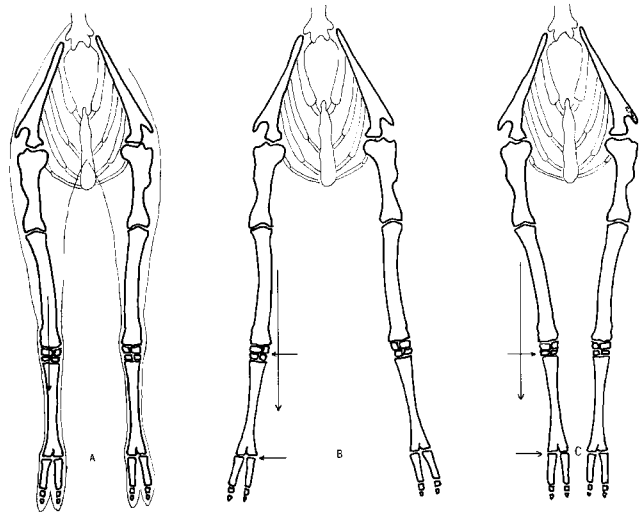

Figure 24.5. Forelimb, front view. (A) Normal, (B) base wide, (C) base narrow.

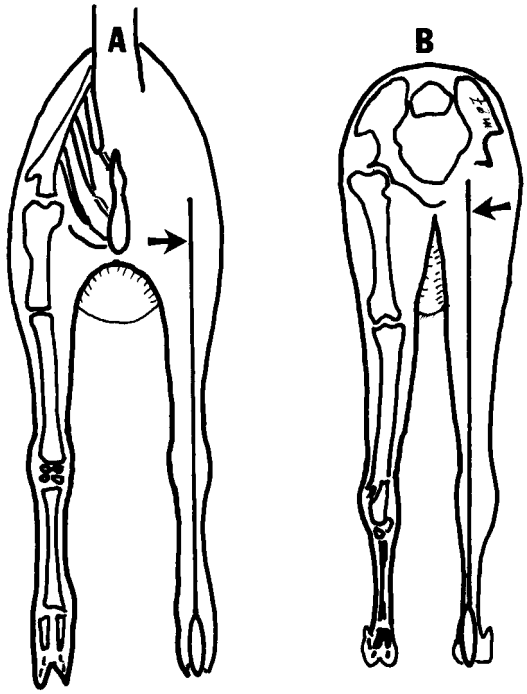

Figure 24.4. Proper stance of the legs of a camelid. (A) Front view of forelimbs, plumb line to bisect the leg; (B) rear view of the rear limbs, plumb line to bisect the leg.

Figure 24.6. Base-narrow stance.

Front and Rear Views

Fore Limb, Front View

The front and rear views are the same for both OWCs and NWCs (Figure 24.4). A plumb-line view helps to give a perspective to straightness of the limbs (Figure 24.4). The limbs of camelids are positioned closer to the midline than they are in most other domestic animals. Faults are illustrated in Figures 24.5 to 24.9.

Hind Limb, Rear View

The camelid may stand base wide or base narrow. Cow-hocks is a common fault. Long bone angular limb deformities of the hind limbs are less common than in the front (Figures 24.10, 24.11).

Side View, Llamas[2,3,6,8]

Besides obvious differences in size, camelids don't differ in conformation when viewed from the side (Figures 24.12 to 24.17)

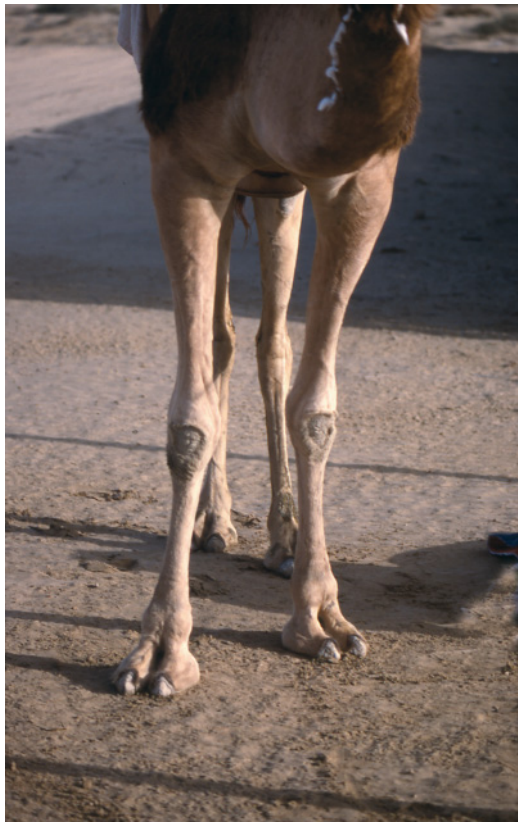

Figure 24.7. Normal front view of a camel.

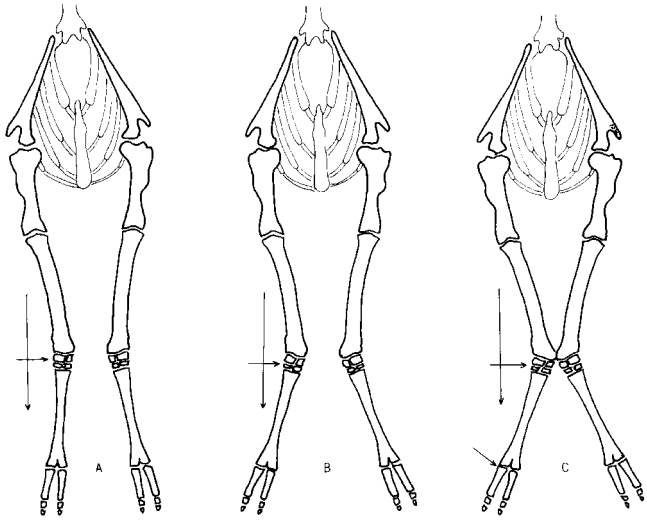

Figure 24.8. Angular limb deformity. (A to C) Degrees of carpal valgus.

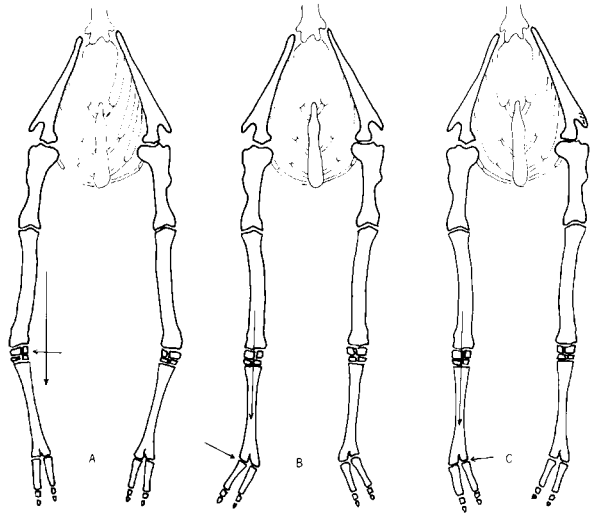

Figure 24.9. Forelimb, front view. (A) Carpal varus, (B) angular deformity of fetlock (splayed), (C) angular deformity of fetlock (pigeon-toes).

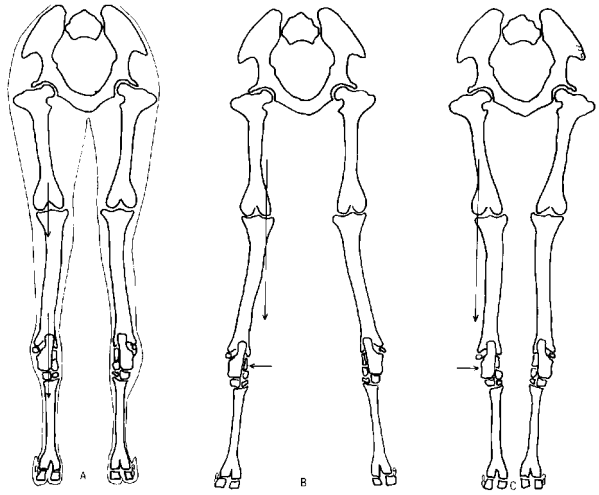

Figure 24.10. Hind limb, rear view. (A) Normal, (B) base wide, (C) base narrow.

Fore Limb

Normal stance is illustrated in Figure 24.12. Other defects are seen in figures 24.13 to 24.16. The angulation of the pastern and the fibroelastic digital cushion of the foot provide the most important cushions of the limbs (Figure 24.16). Figure 24.17 illustrates a condition called "down in the fetlock," "weak pastern," or hyperextension of fetlock. Normally, phalanx 1 (P-1) is semivertical, with an approximately 45 to 50 degree angle with the ground. P-2 and P-3 lie horizontally within the foot. Dropped fetlock may be a specific congenital defect, or it may be acquired from trauma or deterioration of the ligamentous support because of prolonged weight bearing if the opposite limb has been incapacitated for a long time. Females that have lived a long, productive life may be down in the fetlock. Congenital weakness would be suspected with bilateral or quadrilateral defects in young animals.

Camels have a slightly different arrangement of the phalanges. P-I is essentially vertical, P-2 at 45 degrees, and P-3 horizontal.

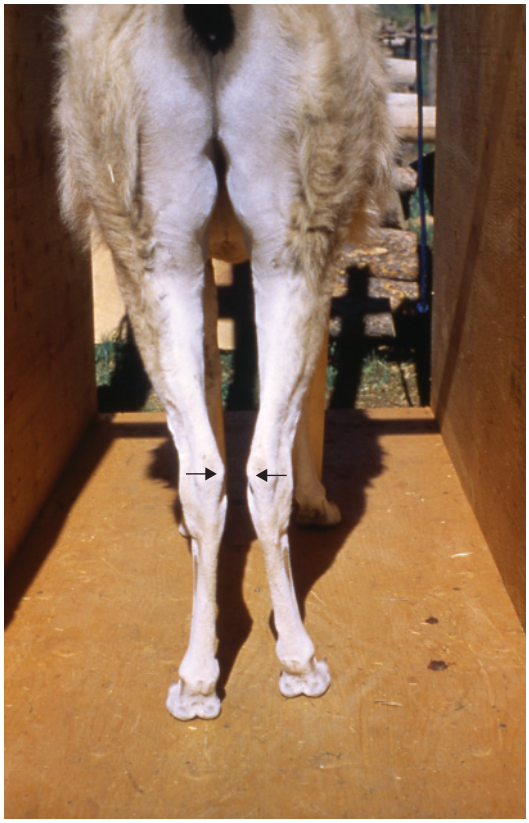

Figure 24.11. Cow-hocks.

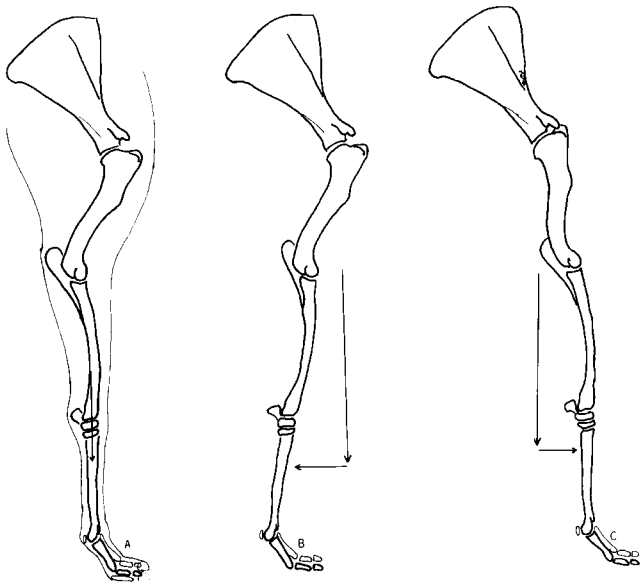

Figure 24.12. Forelimb, lateral view. (A) Normal, (B) camped behind, (C) camped forward.

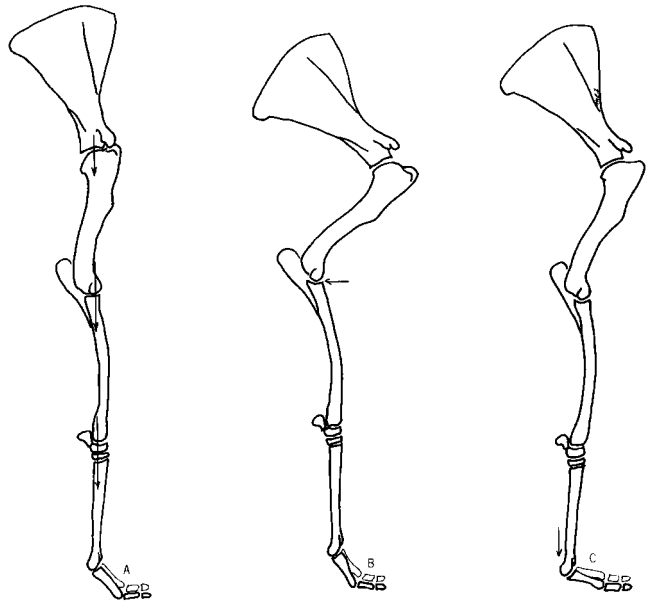

Figure 24.13. Forelimb, lateral view. (A) Straight or post leg, (B) excessive angulation of shoulder, (C) hyperextension of the fetlock.

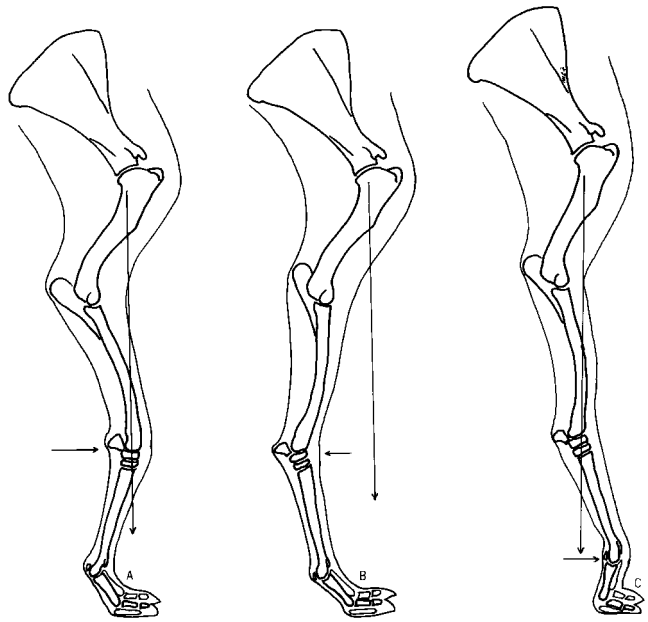

Figure 24.14. Forelimb, lateral view. (A) Buck-knee, (B) calf-knee, (C) contracted flexor tendons of fetlock.

Hind Limb

Normal, camped behind, and camped forward are illustrated in Figure 24.18. Many domestic camelids have a tendency to stand slightly sickle-hock (Figures 24.19, 24.20); however, the overall stance of the rear limb is straighter than that of the horse or cow. The lateral conformation faults are illustrated in Figures 24.19 to 24.33.

Miscellaneous Side View Faults

Camelids may have an excessively narrow chest, barrel chest, sway back (lordosis), camel back (kyphosis), variable set of the neck and tail, small head, or

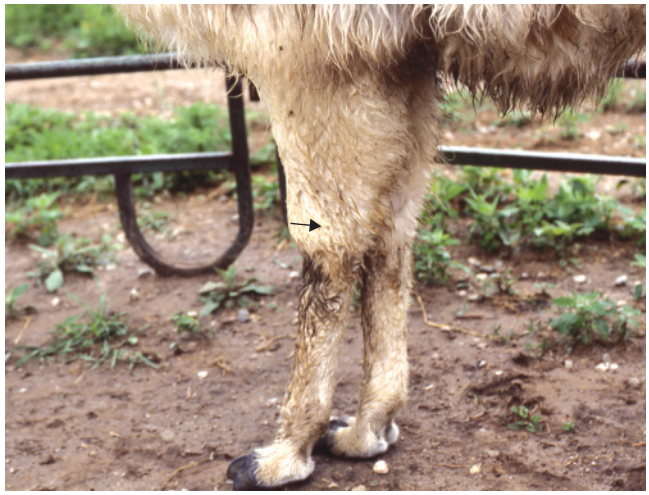

Figure 24.15. Calf-knee of a llama.

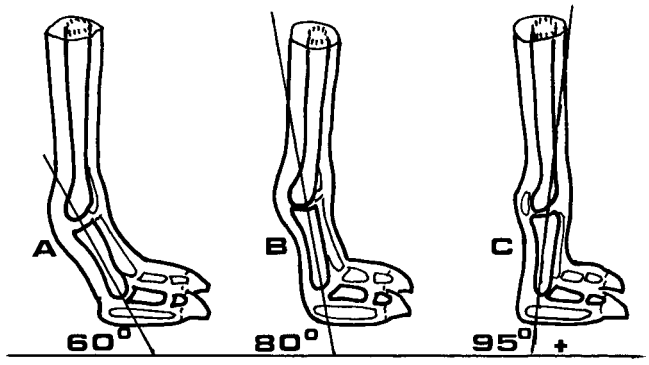

Figure 24.16. Diagrams of pastern angulation. (A) Appropriate angulation for a llama, (B) appropriate angulation for an alpaca, (C) over at the fetlock (hyperflexion of the fetlock).

Figure 24.17. Hyperextension of fetlock.

large head. Conformational faults and defects of the head were discussed elsewhere. Other congenital conditions are illustrated in Figures 24.24 to 24.33. Figure 24.33 illustrates leg length compared with body length.

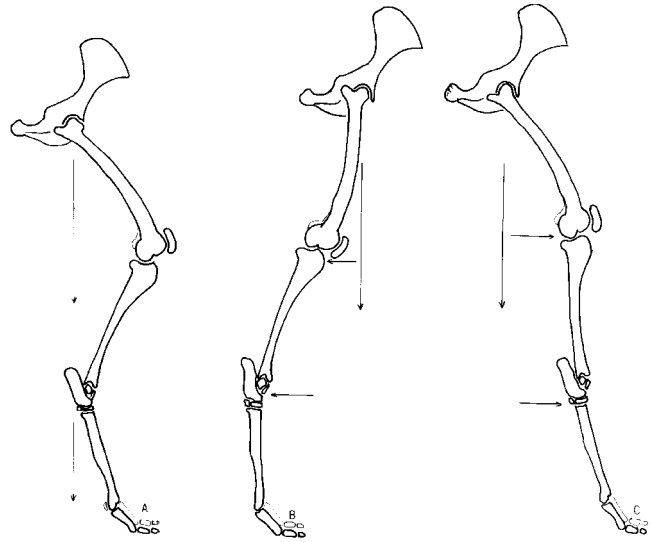

Figure 24.18. Hind limb, lateral view. (A) Normal, (B) camped behind, (C) camped forward.

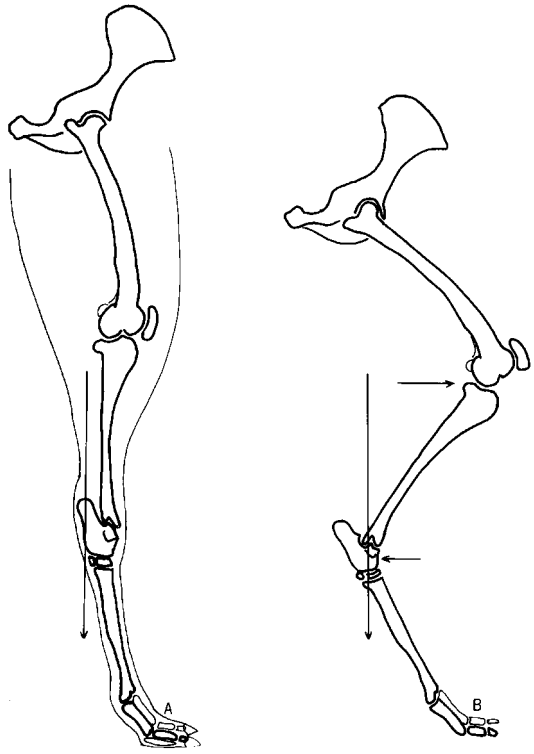

Figure 24.19. Hind limb, lateral view. (A) Straight leg, (B) excessive angulation (sickle-hock).

Side View, Alpacas[5,7,9]

The side view of an alpaca differs markedly from the side view of a llama.[5,7,9] Figure 24.34 illustrates the topline and rounded tailhead of a shorn alpaca and Figure 24.35 a fully fibered alpaca. Conformation is not easy to assess in a heavily fibered animal. Figures 24.34 to 24.45 depict conformation in shorn alpacas. Con-

Figure 24.20. Sickle-hock.

Figure 24.22. Hyperextension of the pastern.

Figure 24.21. Contracted flexor tendon, over-flexinon of the fetlock.

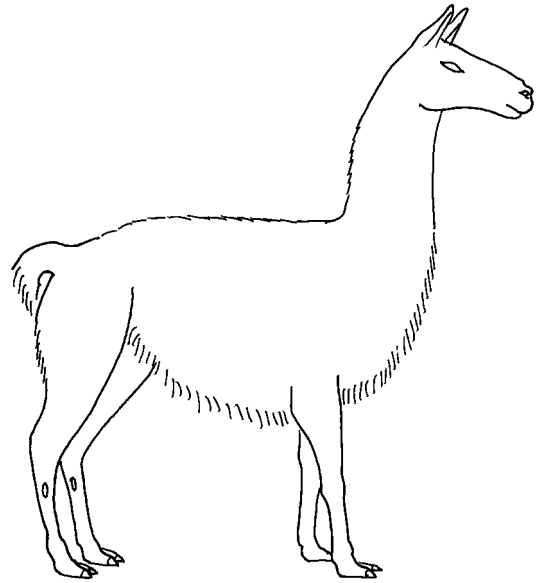

Figure 24.23. Llama conformation, sloped rump.

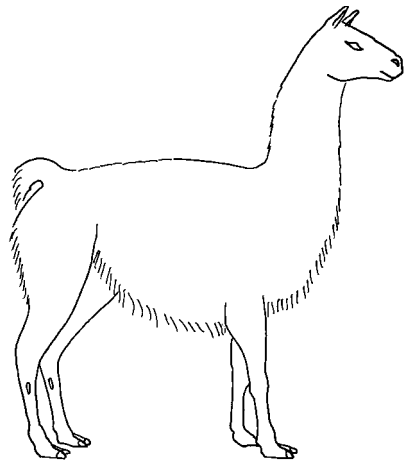

Figure 24.24. Llama conformation, humpback.

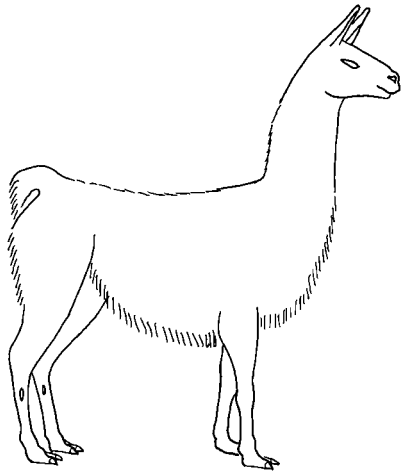

Figure 24.25. Llama conformation, swayback.

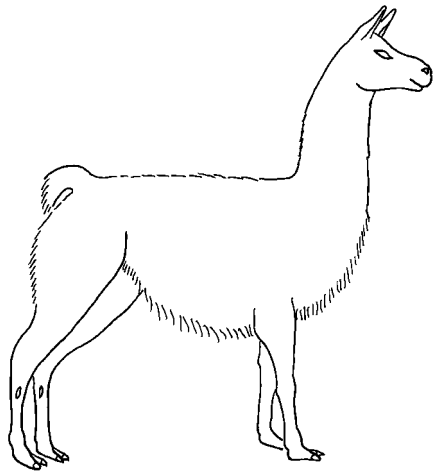

Figure 24.26. Llama conformation, camped rearward behind. Dished face.

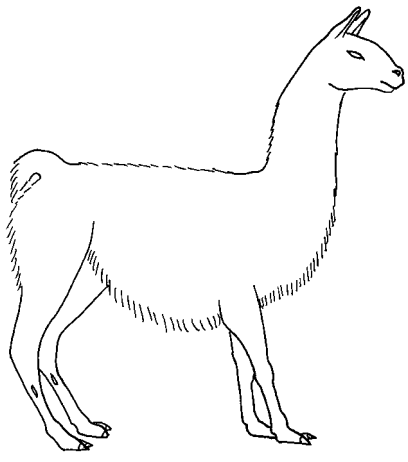

Figure 24.27. Llama conformation, camped forward behind. Roman nose.

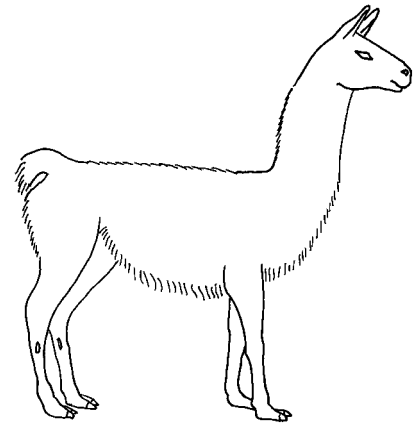

Figure 24.28. Llama conformation, shallow body.

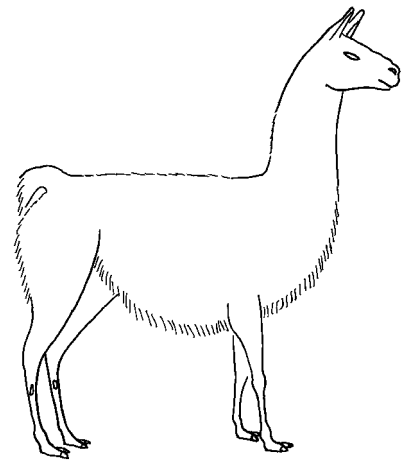

Figure 24.29. Llama conformation, small leg bones.

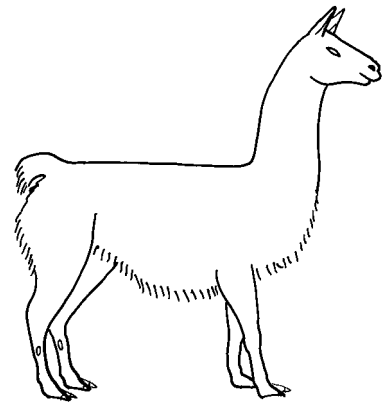

Figure 24.30. Llama conformation, short legs.

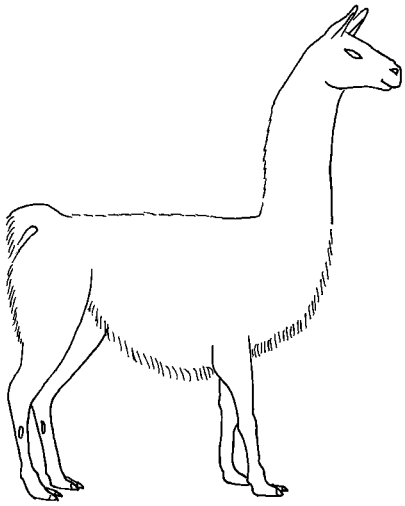

Figure 24.31. Llama conformation, long neck.

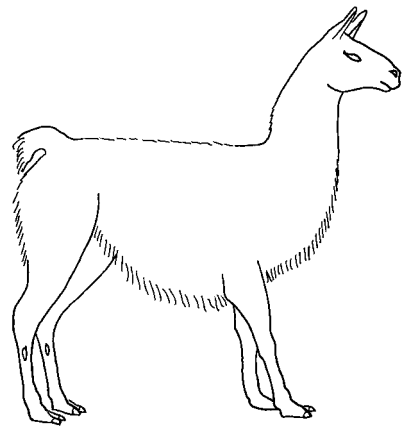

Figure 24.32. Llama conformation, short neck and camped forward in front.

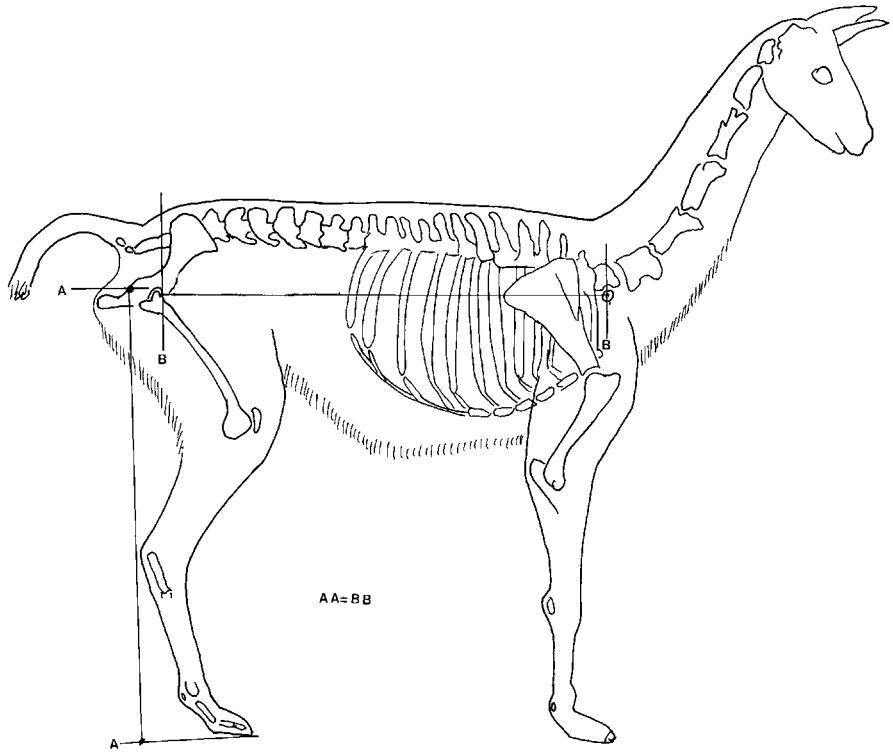

Figure 24.33. Llama conformation, showing relationship of leg length to body length.

sider the ideal conformation of an alpaca side view as three rectangles (Figure 24.34). Body depth, leg length, and neck length should be equal. Figures 24.36 to 24.47 illustrate side view conformation of alpacas.

Alpacas have a more vertical pastern (P-1), approximately 70 degrees, while the llama has more slope to the pastern. Thus, alpacas may be more inclined to develop hyperflexion of the fetlock (cocked ankle) (Figure 24.47). When standing relaxed, alpacas appear to be slightly base wide in front. The appearance is enhanced by the fiber on the limbs and may even give the impression that an animal is knock-kneed. Be particularly observant because such an alpaca walks to make certain that the legs are tracking straight.

Other Traits

Alpacas have a relatively short, triangular face, while the llama's face is longer. Ear shape is variable

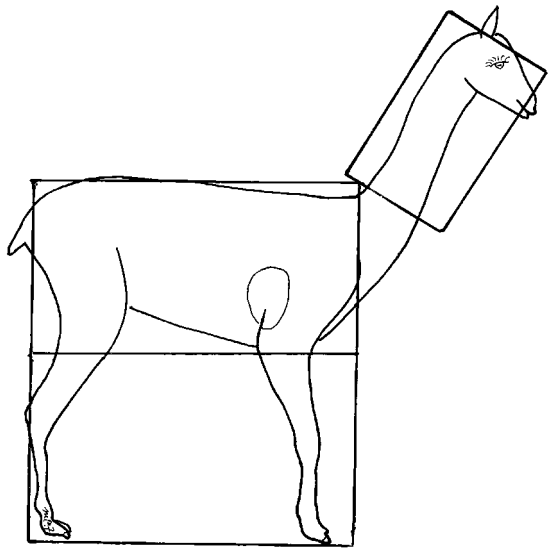

Figure 24.34. Normal alpaca conformation. Rectangles show proportional depth of body, length of legs, and length of neck. All should be the same distance.

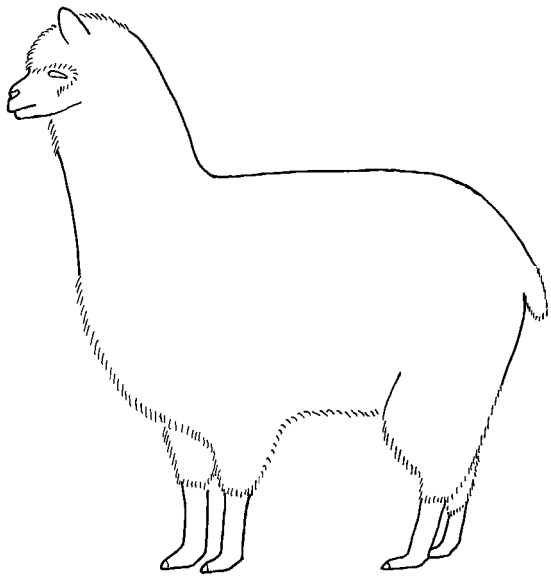

Figure 24.35. Alpaca conformation in a heavily fibered animal.

but distinctly different in llamas and alpacas (Chapter 19). Proper occlusion of the teeth is important in both llamas and alpacas. A full discussion and illustrations of the digestive tract are found in Chapter 13. Some owners may attempt to justify elongated teeth by saying that the animal has not eaten the same kind of harsh grasses that grow in South America and worn down the teeth. The fact is that malocclusion is a conformational fault and is familial in most cases. Alpaca incisors continue to grow well into adulthood, but if the bite is correct, the teeth wear off appropriately. The

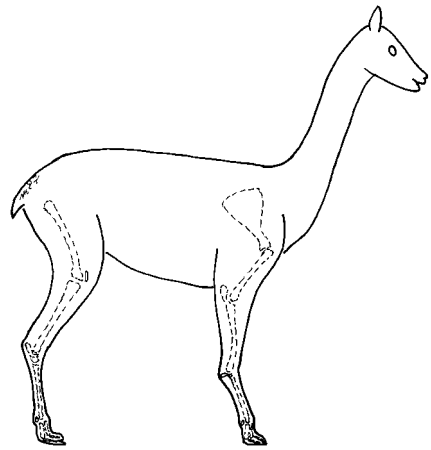

Figure 24.36. Alpaca conformation, calf-knee.

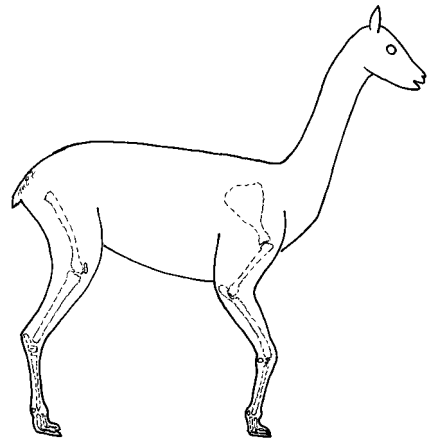

Figure 24.37. Alpaca conformation, buck-knee.

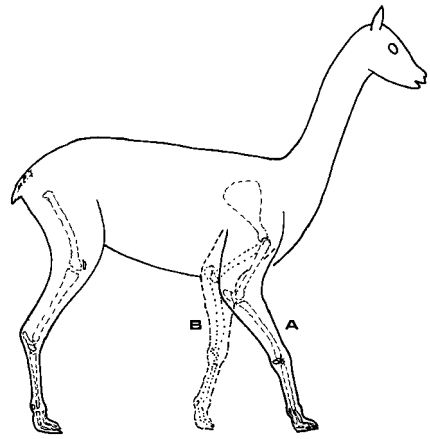

Figure 24.38. Alpaca conformation, camped forward (A) and rearward (B) in front legs.

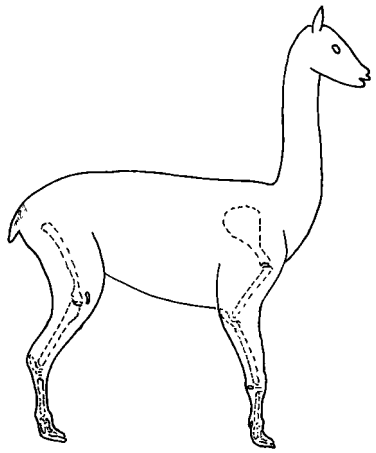

Figure 24.39. Alpaca conformation, short legs.

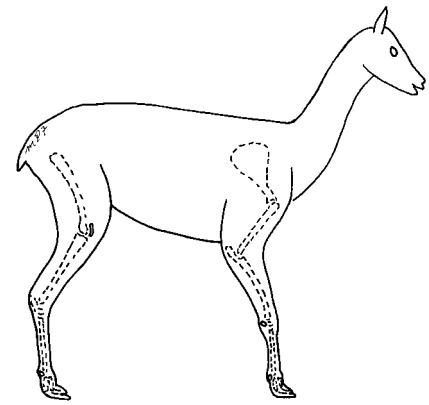

Figure 24.42. Alpaca conformation, short neck.

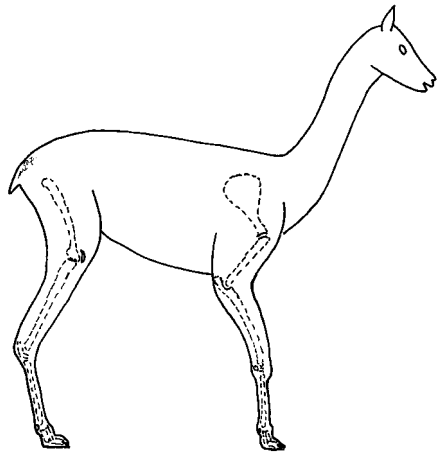

Figure 24.40. Alpaca conformation, long legs.

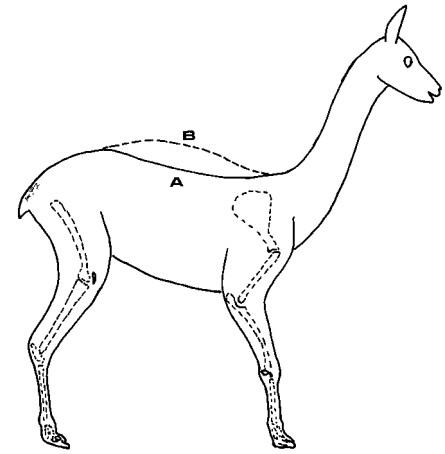

Figure 24.43. Alpaca conformation, lordosis (A) and kyphosis (B).

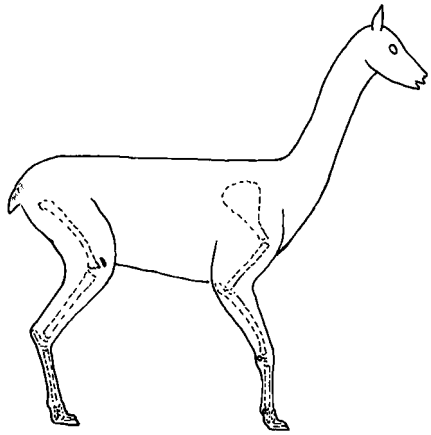

Figure 24.41. Alpaca conformation, crouched.

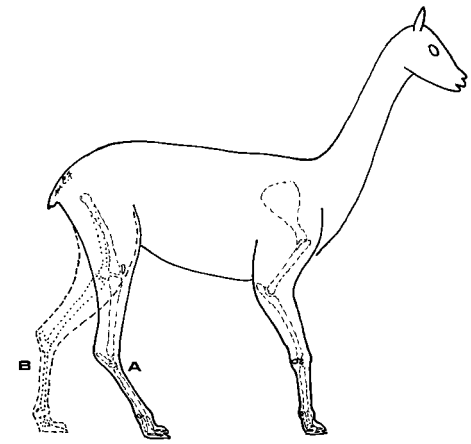

Figure 24.44. Alpaca conformation, camped forward (A) and rearward (B) in hind legs.

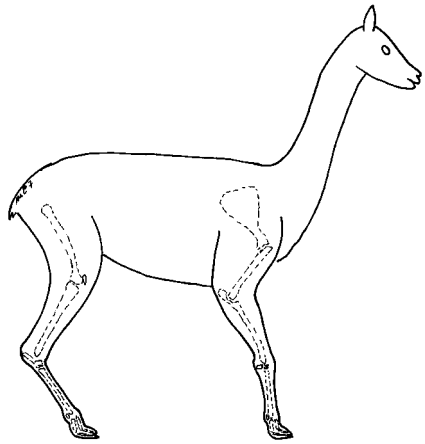

Figure 24.45. Alpaca conformation, sickle-hock.

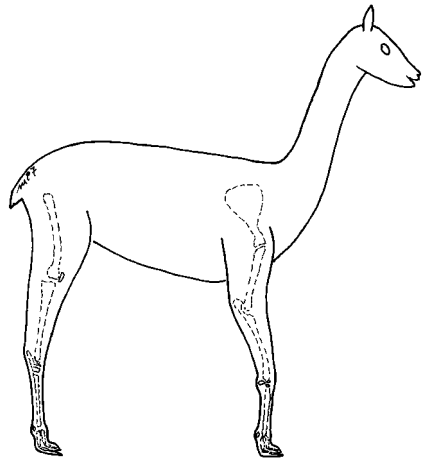

Figure 24.46. Alpaca conformation, standing straight in front and post legged behind.

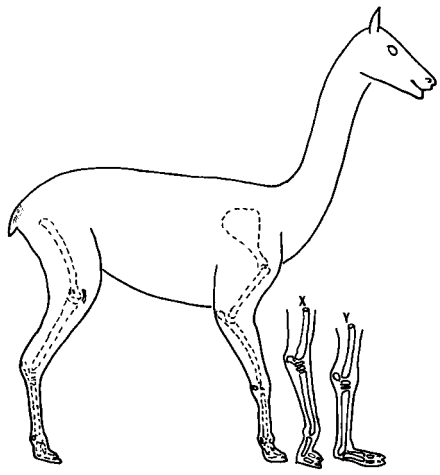

Figure 24.47. Alpaca conformation. (X) Cocked ankle and (Y) down in fetlock. Long head.

most common fault is inferior prognathism (undershot lower jaw), but superior prognathism (parrot mouth or overshot upper jaw) is also seen. Miscellaneous alpaca faults include short-legged alpaca, long-legged alpaca, and sway-backed llama, Figures 24.48 to 24.50.

A dromedary camel skeleton is illustrated in figure 24.51.

Figure 24.48. Short-legged alpaca.

Figure 24.49. Long-legged alpaca.

Figure 24.50. Sway-backed llama.

GAITS[1,4,10–12]

All camelids use the same basic gaits: walk, pace, and gallop. SACs may also trot, especially alpacas, but

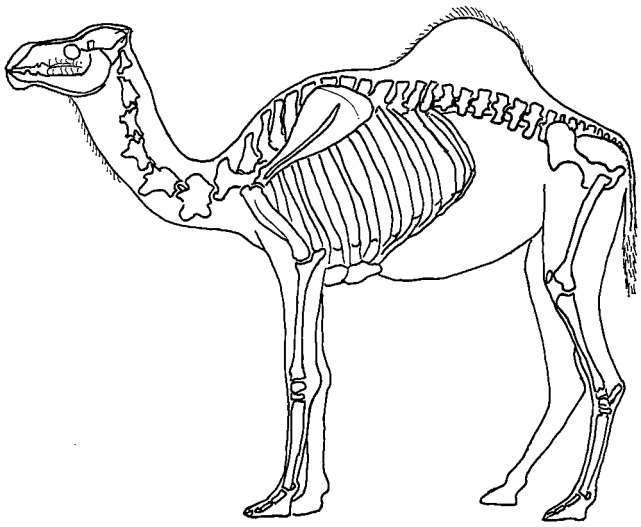

Figure 24.51. Dromedary camel skeleton.

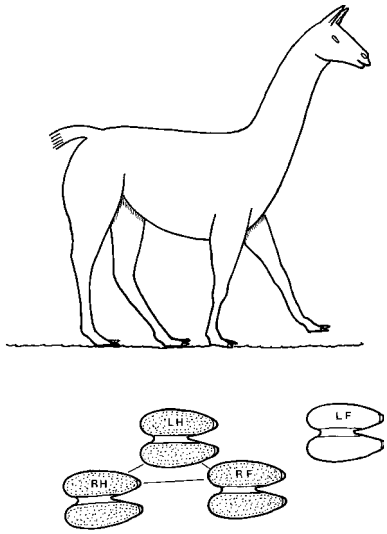

Figure 24.52. Gaits, walk.

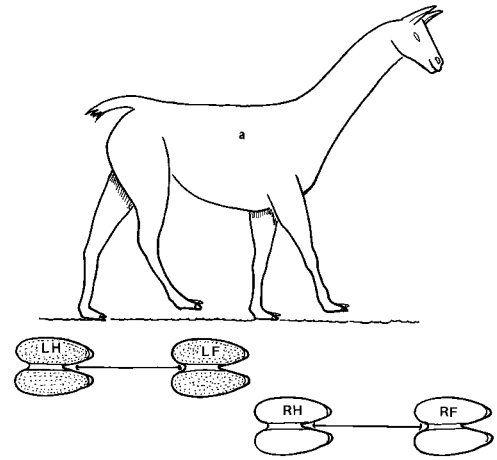

Figure 24.53. Gaits, pace.

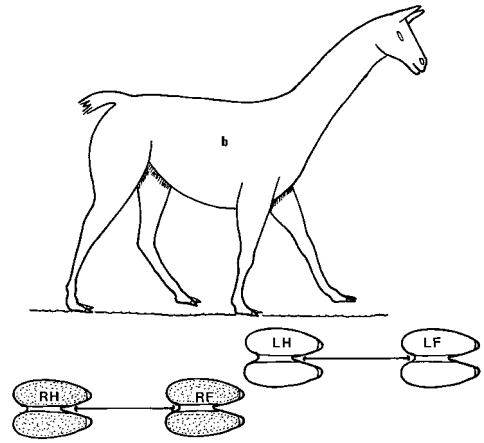

Figure 24.54. Gaits, pace.

camels rarely trot. Nature mandates conformation for function. The conformation of camelids with a narrow body, long legs, and no flank fold to impede movement of the hind limbs makes for energy efficiency of movement. Domestication often interferes with the natural inclinations of the animals, and modern humans may interfere through inappropriate breeding programs, disregarding basic conformation, to the detriment of the species.

A fifth gait or playing gait of SACs is called the pronk or stott.

The walk is an evenly spaced four-beat gait in which each foot strikes the ground separately and in sequence. Three feet are always on the ground (Figure 24.52). It is the slowest gait and can be sustained for long periods. It is also the most stable gait, providing the greatest base of support.

The pace is a medium speed, two-beat gait in which the fore and hind limbs on the same side of the body move in unison (Figures 24.53, 24.54). Although the pace is the least stable of all animal gaits, the llama is uniquely conformed to use it. Natural pacers have relatively long legs, with each limb being longer than the trunk, allowing the animal to develop a long stride. The forward part of the thorax is narrow, permitting the upper fore limb to freely move forward and backward. Also, in camelids, the attachment of the hind limb to the pelvis is narrow, and the abdomen is less rounded than in other livestock species, allowing the hind limb freer motion. Camelid limbs are also set more closely to the midline than in other species, eliminating some of the side-to-side rolling that occurs when the center of body gravity is changed with each stride.

Basically, the pace is an unstable gait because lateral stability is decreased. The pace is designed to permit an animal to move swiftly over open country. The unique foot of the camelid may be an adaptation to

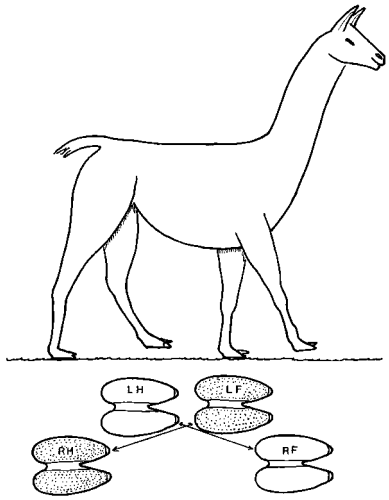

Figure 24.55. Gaits, trot.

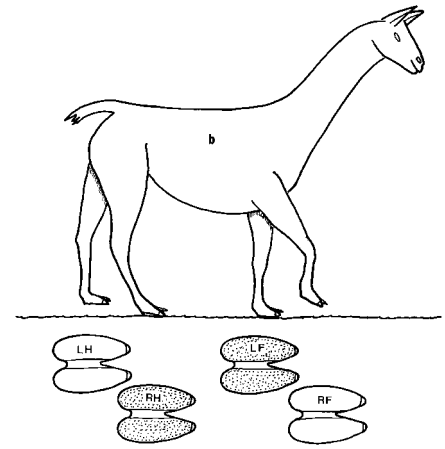

Figure 24.57. Gaits, gallop.

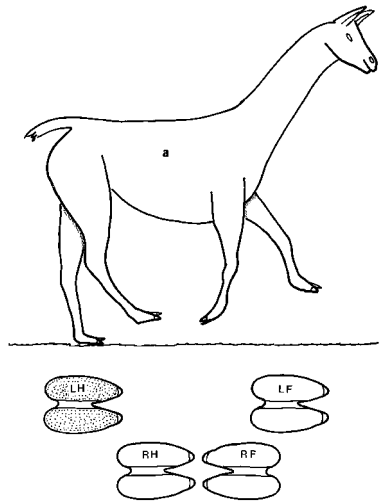

Figure 24.56. Gaits, gallop.

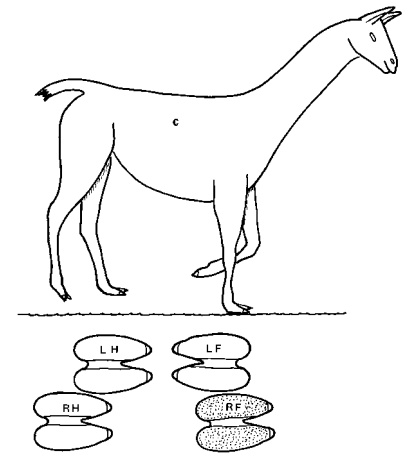

Figure 24.58. Gaits, gallop.

increase the stability of the animal for the pacing gait. All other two-toed ungulates have ligaments that tie the digits together. Camelids have a splay-toed foot (not to be confused with splay footed) that spreads and provides a strong base of support. This, combined with the padded foot, makes the camelid one of the more surefooted ungulates.

Some llamas and alpacas routinely trot instead of pace (Figure 24.55). The trot is also a two-beat gait, in which the diagonal fore and hind limbs move in unison. The fastest gait is the gallop, or run (Figures 24.56 to 24.58). It is the most fatiguing and can only be sustained for a few moments because, unlike horses, llamas lack heavy propulsion muscles in the rear limbs.

Dromedary camels alternate between a pace and a gallop when racing, Figure 24.59.

The speed of various gaits in camelids and other species are listed in Table 24.1.

Pronking (stotting) is a form of locomotion in which the SAC thrusts the body upward and forward, landing on all four feet at the same time in a stiff-legged stance (Figures 24.60 to 24.62). This is a playing gait and is more commonly seen in young animals, especially in the evening or when new animals are introduced into the herd.

Although alpacas and llamas use the same basic gaits, conformational differences are reflected in gait modification. The angle of attachment of the pelvis to the spinal column is approximately 50 degrees in the alpaca, whereas the angle is approximately 40 degrees in the llama. Thus, there is a slight difference in the stance and movement of the rear limbs. Alpacas have a shorter stride than llamas and they tend to gallop more than pace when excited or running away from pursuers. Numerous conformation faults detract from balance and inhibit the pacing gait.

Camels differ from horses and man in exercise physiology. This has been determined by basic research on racing camels as described by Saltin and Rose.[10]

Table 24.1. Speed of camels compared with other animals.

Gait	*Kilometers per hour (kph)*	*Miles per hour (mph)*	*Meters per second (mps)*
Camel, walk	7.2	4.5	2
Camel, slow pace	14.4	8.9	4
Camel, fast pace	21.6	13.4	6
Camel, racing gait, average	34.2	21.25	9.5
Camel, racing gait, peak	40.32	25.05	11.2
Camel, 18-hour endurance	16	9.94	5.1
Horse, 1.5 mile, Secretariat	52.39	32.55	16.7
Horse, Quarter, at finish	52.74	38.99	20
Horse, avg. walk	8	5	2.6
Human, fastest, 100 meters	37.27	23.16	10.35
Human, avg. walk	4.8–6.4	3–4	1.5–2
Human, brisk walk	8	5	2.6
Dog, running	29–50	18–31	9.24–15.9
Dog, greyhound	68	Up to 42	21.7
Oxen	2	1.2	0.64

Figure 24.59. Camel racing in the United Arab Emirates.

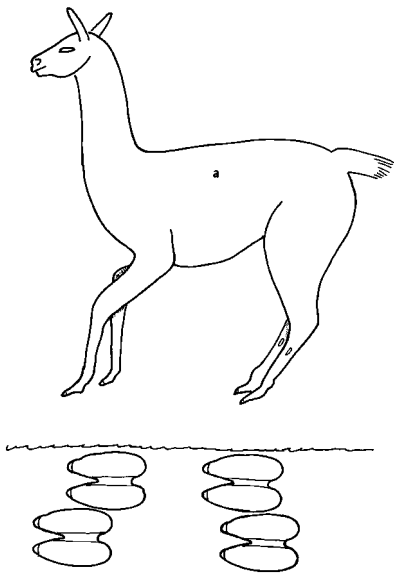

Figure 24.60. Gaits, stott (pronk), in the air.

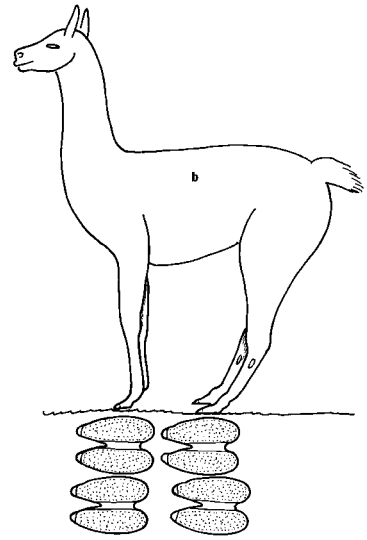

Figure 24.61. Gaits, stott (pronk), on the ground.

Figure 24.62. Pronking alpaca. Photo courtesy of Sue Rosche, Pax River Alpacas, Maryland.

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[a] Dr. Julie Koenig, Corvallis, Ore., personal communication, April. 1995.

25

Disaster and Emergency Management

A disaster is an unusual event that causes much suffering or loss.[1] A heavy snowstorm in Colorado, Utah, or Montana is expected, and reasonable precautions are taken. Excessive heat and humidity are normal for the southeastern states. On the other hand, a hurricane is always a disaster if on land. An emergency is a sudden or unexpected situation that calls for immediate action.

This chapter discusses the disasters or emergencies that camelid owners/managers may encounter and outlines steps to prevent or deal with the situations should they arise. Veterinarians should be able to advise clients on how to deal with disaster situations. In Figure 25.1 a linked chain illustrates the fundamentals for dealing with disasters and emergencies. Attitude is the key to human survival in disaster situations. That same attitude is no less important when dealing with animals. If a disaster strikes and no thought has been given to coping, panic is the usual result. If, however, one can say, "Begin plan A or plan B," energy may be directed positively. The best insurance of a positive attitude is advance planning and preparation.

Basic to this discussion is an understanding that the evacuation and protection of people take precedence over the needs of animals. Evacuation of animals may not be permitted by disaster personnel. Owners may not be allowed to rescue their animals or care for them until officials declare it safe to do so. The experience of llama owners in some disaster situations is that, in the final analysis, each facility must deal with disasters alone. However, networks for help are to be encouraged: there have been numerous examples of instances when they have been lifesaving, but there are times when owners can rely only on themselves and the resources available.

DISASTER AND EMERGENCY SITUATIONS

A partial listing of potential disasters faced by llama/alpaca managers is found in Table 25.1. Some will be discussed further. The results of disasters are listed in Table 25.2.

Extreme Temperatures

Bad weather is a threat in all regions of the world. Normal climatic fluctuations are taken in stride, but the bizarre or unusual may portend disaster. In the summer, ambient daytime temperatures may reach 38°C to 43°C (100°F to 110°F) in the central valley of California, but they usually cool down at night, and the humidity is less than 50%. However, a fifteen-day spell of 40°C (105°F) temperatures with minimal overnight cooling could be lethal for long-fibered llamas or alpacas that lack water pools for cooling.

In contrast to their cousin, the dromedary camel, llamas and alpacas are not adapted to hot environments. They may be raised and maintained in areas with a hot, humid climate, but only with appropriate planning and facilities. Llamas and alpacas are adapted to cool to cold climates but not the subzero temperatures that occurred over much of the northern and eastern United States and Canada in 1994. Although nighttime temperatures drop below freezing 300 days of the year in the Andean Altiplano (above 4,000 m [13,100 ft]), subzero temperatures are rare.

When subzero temperatures are accompanied by wind, the chill factor drops the effective temperature even lower. If a storm prevents access to feed and shelter, this combination of factors could result in a disaster. Many barns are impossible to heat because of high ceilings and lack of adequate insulation. A technique often used by livestock managers to overcome

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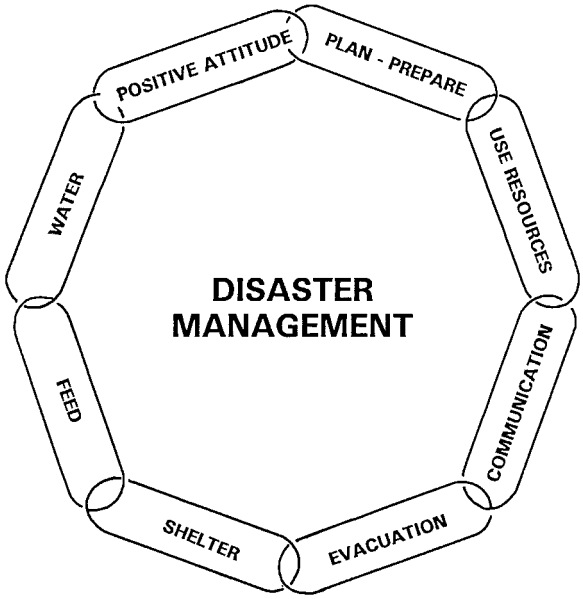

Figure 25.1. Factors for successful management in a disaster situation.

Table 25.1. Potential disasters or emergencies involving Camelids.

Prolonged heat wave
Extreme cold
Heavy snowstorm
Ice storm
Fire (dwelling, barn, grass, brush, forest)
Excessive wind (hurricane, tornado, typhoon)
Drought
Flooding (dam bursting, prolonged and heavy rains, flash flood)
Earthquake
Volcanic activity
Toxic chemical spill
Lightning
Truck or trailer accident
Lost animals
Dog or wild animal attack
Nuclear power plant accident
Terrorist attack
War
Riot

this problem is to construct part of a barn with low ceilings and insulated walls. Lacking such preplanned construction, canvas tarps and sacking may be used to artificially lower the ceiling to inhibit the heat generated by the animals from rising and dissipating. Bales of straw or hay may be used to add insulation to walls. Windows should be boarded over, and cracks or openings stuffed or covered. The use of portable heaters may be contraindicated because of the fire hazard.

Table 25.2. Results of disasters or emergencies involving Camelids.

Loss of power (water pump, lighting, heating, air-conditioning)
Loss of water (burst water mains, pumps nonfunctional)
Loss of communication (telephone lines down, loss of power)
Contaminated feed and water (infectious agents, parasites, chemicals)
Electrocution from downed power lines or torn lines on the premises
Injuries sustained during or following the event
Starvation
Dehydration from lack of water
Thermal disorders (heat, cold, frostbite)
Highways closed (bridges out, flooding, landslides, disruption of roadbed by earthquakes)
Animals wandering away because of damaged fences
Smoke inhalation, eye injuries
Drowning

Special consideration should be given to feeding camelids during cold weather. Adults should be provided with a high-fiber diet because the heat of fermentation (digestion) of the fiber may be used by the animal for warmth. If weanlings and yearlings are restricted to a high-fiber diet, they may be deficient in energy and not grow properly. Although the feeding of concentrates (such as grain) may not produce heat like fiber does, the energy provided may be necessary for youngsters, and furthermore, the concentrates may also increase the efficiency of the microorganisms that carry out the fermentative process.

Neonates are particularly vulnerable to cold.[3] Owners in cold climates frequently adjust the birthing season to avoid the riskier months. Crias to be born during unusually cold snaps should be protected by housing the dam in a heavily bedded stall. The newborn must be dried thoroughly and the ears massaged to increase circulation and warm them. Some owners manufacture special sweaters to encase the chest, shoulders, and neck. Other materials may be used in an emergency. Strips from a wool blanket or artificial fleece may be used to wrap the cria and duct tape used to secure the strips in place. Avoid completely encircling the chest with tape; this could inhibit respiration. Do not cover the prepuce of the male cria.

It has been suggested that a space blanket may be spread out beneath the bedding in an area where a cria is likely to lie down. This could prevent permeation of cold through the floor. Make certain that drafts are eliminated.

Frostbite of the ears is a significant risk during extremely cold spells, particularly in crias. A wool cap or helmet may be appropriate for a newborn. Crias

were lost to hypothermia during the 1994 extreme cold in the north central and northeastern United States. Management is the key to preventing hypothermia, but if it does occur, rapid steps must be taken to prevent death. If a cria is found to have a rectal temperature of less than 33°C (92°F)—the lowest measure on a clinical thermometer—thermoregulation is inhibited, and it is unlikely that the cria can rewarm itself. Temperatures below 30°C (86°F) have been recorded with a more sophisticated thermometer; these animals are usually completely comatose. Intensive care must be provided to warm the cria. Hot water bottles, plastic milk containers, or heavy plastic bags wrapped in towels; hair blow dryers; heat lamps; portable heaters; and warm water enemas are all useful techniques to warm a cria.

The optimal rewarming technique is total immersion (except for the head) of the cria in warm water at a temperature of no greater than 45.5°C (114°F). This is approximately the temperature used for human infant formula or bath water (tested by the elbow, or inside of the wrist, the water should feel slightly warm). The rectal temperature should be measured every few minutes because it is possible to go from too cold to too hot. Be sure to dry the cria completely after removal from the water to avoid rapid heat loss following rewarming. A hand-held blow dryer set on warm may be useful for drying the cria. See Chapter 9 for more details on thermal conditions.

Ice Storms

An ice storm is rain that freezes immediately upon landing on the ground, foliage, or electrical transmission lines. The unique climatic circumstances that produce this phenomenon may last only a few minutes to hours, but the consequences may be great. People and animals may have extreme difficulty walking and vehicular traffic may be snarled, power lines destroyed, and tree branches broken off. Ice storms paralyzed rural central and western Kentucky for days in 1994. If ice storms are a frequent occurrence, it may be well to have a pile of small-sized gravel to spread where walking must be done.

Fire

Fire is an ever-present risk. There are an average of 6,400 barn or stable fires annually in the United States.[6] The estimated annual cost is More than $75 million (U.S. dollars). One llama ranch lost thirty-eight llamas, a barn, and stacked hay. Other llama owners have also experienced barn fires.[6] Llama and alpaca owners frequently move to rural areas because of the lifestyle, less prohibitive zoning ordinances, or sufficient, less expensive land. The hinterland existence may place owners and their animals in areas of dense vegetation (grass, brush, forests) some distance from fire protection. Disastrous fires occurred in wide areas of California during the past two years. Camelid owners were not spared the heartache and economic loss of homes, barns, and vehicles. Fortunately, no llamas or alpacas lost their lives to these fires. Contingency plans for evacuation should be made because owners may be unable to prevent fires fanned by heavy winds. Those with large numbers of animals may find it impossible to find sufficient vehicles to evacuate all of them. Decisions may have to be made, prioritizing which animals are more important for breeding purposes or emotional reasons. This should be done prior to the stress of a disaster.

Sufficient halters and lead ropes must be readily available. Small catch pens should be regularly used in large pastures to allow expeditious catching and moving. The animals should be trained to move in and out of these catch pens with ease. Larger numbers of animals may be moved, even with an automobile if all animals are taught to lead: halter or tie one or more animals to the vehicle; then, using a bowline knot, loosely tie the next animal's lead rope around the neck of the lead animal. This technique is frequently used with pack strings of horses and mules by tying each animal either to the pack saddle or the tail of the animal ahead. The process may be repeated until all animals are in a caravan; then the vehicle is moved off at a slow pace. Obviously, this can be done only with compatible animals. It is out of the question with males.

The key to an evacuation is to move out before the disaster strikes. Don't try to outguess those who issue warnings. Do not turn llamas and alpacas loose in a barn fire situation. Too often they will disregard danger and even return to enter burning buildings.

Wind is an important factor in brush and forest fires. As a general precaution, the area surrounding buildings in wooded areas should be cleared of all flammable material for at least 15 m (50 ft), but this will not avail in high winds, which may cause flames to leap hundreds of feet. Crown fires (flames racing through the tops of trees in a forest) are particularly troublesome.

The California State Department of Forestry and Fire Protection further recommends the following to improve the chances that homes and barns will survive a fire. Clear pine needles, leaves, and other debris from the roof and gutter. The roof should be constructed of fire-resistant materials. Prune the bottom 2 m (6 ft) of trees taller than 5.5 m (18 ft) to keep ground fires from spreading into tree crowns. Keep firewood stacks and butane or propane tanks at least 9 m (30 ft) from any structure and clear a 3-m (10-ft) area around them. Cover chimney outlets with screen to keep sparks from entering the house. If there is no fire hydrant nearby,

keep an emergency water supply of at least 2,500 gallons on your property and make sure it is clearly marked for the fire fighters who may have to defend the house. Plan which valuables to take in case of evacuation and if possible store these belongings together.

Although property loss has been high in some of the fires in southern California, animals seemed to fare quite well. In some instances the llamas were herded into a central, bare ground area. In other situations the llamas were simply turned loose to fend for themselves. Owners remarked that the animals adjusted quite well, without panic. When owners could return to their animals, they found them hungry and thirsty but without evidence of smoke inhalation or eye damage. Llama/alpaca fiber does not burn readily, but it does smolder, and in some animals burn spots were noted in the fleece. At one ranch the only serious problem was burns on the footpads, presumably caused by animals stepping on hot coals. No one was able to observe how the animals avoided the flames, but both adults and young animals did so. No abortions or premature births occurred as a result of the stress.

A northern California owner had to evacuate without taking llamas along when a fire, some three miles distant, caused so much smoke and ash that it became unbearable for humans. The fire never reached the property, and the llamas did not suffer from smoke inhalation.

Sandy Mubarak offered some sage advice following her experience with a brush fire.[7] Have fitted and labeled halters (nylon halters may catch cinders and melt, burning the skin) and lead ropes for every animal, stored in a conspicuous place in the event that persons other than yourself must do the moving. Conduct fire drills to train groups of animals to move where you need them to go. Have some means of water storage and a pump(s) to fight a fire yourself. Pumps should be diesel or gasoline powered because electricity is frequently turned off centrally in an area experiencing a fire.

Get acquainted with local fire officials and disaster management for the county. If in doubt about fire safety for a barn, ask that a fire person inspect your facilities and recommend remedial action. Volunteer to work on the local disaster committee. Be a contributor.

Wind

Coastal residents of the southeastern and southern United States can expect periodic winds of hurricane force. Approximately ten hurricanes form each year in the Caribbean area, and two of these storms usually make landfall and wreak destruction. Hurricanes are accompanied by rain, high tides, flooding, and destruction of buildings, trees, fences, stored feed, and power lines.

Hurricane Andrew swept across southern Florida in 1993. The Miami metro zoo sustained a loss of 60% of the structures in the zoo. Animal enclosures were destroyed, and animals were killed or escaped into surrounding areas. Hurricane Andrew continued on across the Gulf of Mexico and hit land again south of New Orleans. The destructive, but below-hurricane-force, winds uprooted trees on a large llama farm north of New Orleans. In anticipation of the storm, animals had been moved to the sturdiest structures available on high ground. Feed bunks were tipped over and secured if possible. The fabric coverings of the shade frames were removed, and all loose items secured. Except for a few trees, damage was minimal, and no animals were injured.

Tornados are common in the Midwest, from Texas to Minnesota. A year ago I would have said Californians needn't fear tornados, but I've recently seen two form, though they never touched down. One twister trashed a trailer park in northern California in 1993.

The key to protection against high winds caused by hurricanes, typhoons, or tornados is to construct barns and sheds according to the engineering standards of the area.

Flooding

The vast majority of flood damage results because buildings are constructed too near streams or in river floodplains. People rely on the level of the usual high-water or flood stage of rivers, but the horrendously costly and destructive flooding of the Mississippi valley in 1993 and the 1997 California floods illustrate that nature doesn't always follow the rules.

When floods are predicted, animals must be moved to high ground and arrangements made for feed and potable water. Sometimes that must be done by boat. A llama owner in Idaho watched as a wall of water filled a gorge over 30 m (100 ft) deep and nearly engulfed his home, barns, llamas, and farm from a burst dam a few miles upstream. Others were not so fortunate as a swath of total destruction a mile wide swept homes, barns, vehicles, and livestock to oblivion when the gorge opened up near a rural town.

Earthquakes

Earthquakes are a common phenomenon in California but may occur in many places in the United States.[4,5,8] Of the ten largest earthquakes in the United States since recorded history, only one was in California, two were in Missouri, and seven were in Alaska.[4] In the past several years significant earthquakes have hit Maryland, Arizona, Utah, Oregon, and California. Rarely can an earthquake be predicted with any accuracy, so only general plans and precautions can be

carried out. Contingency plans must be made for auxiliary power, water, feed, shelter, and even care.

Wooden structures seem to be the most resilient, but some wooden llama facilities were destroyed during recent California earthquakes. Water pipes may burst and flood a barn. Fires are a common sequel when gas lines rupture. One couple who owned llamas was severely injured when their house was destroyed in a quake. Neighbors pitched in to care for the llamas until the owners regained their health.

Recommendations for human safety include taking shelter beneath a strong table or desk or standing inside the frame of a doorway if a tremor is felt. Turn off gas, water, and power as soon as possible. If you must go outside, use caution because there is a possibility of power lines falling.[2,4,5,8]

Drought

Drought is not a sudden, intense disaster such as a fire or flood, but the consequences may be manifold. Water tables may drop so low that pumps become nonfunctional. Normally continuously flowing streams may dry up. Reservoirs, depended upon to supply culinary and irrigation water, may be emptied. Arrangements for alternate sources for feed and water are the keys to survival.

Volcanoes

Volcano cones form the backdrop to some of the more picturesque llama and alpaca ranches in North America. More than three decade ago, in Washington, Mount St. Helens unleashed a fury that wreaked such havoc that the tourist visiting the area today is struck with awe at the devastation. Camelid ranches may be located on the margins of volcanoes thought to be dormant but that may only be sleeping.

Lightning

Lightning may occur anywhere, but some regions experience frequent thunder and lightning storms. Unfortunately, the erect head and neck posture of the camelid makes it a target and convenient conduit to the ground. A llama was struck and killed while lying in a pasture in Louisiana. The green grass was singed where her knees and hocks touched the ground. In problem areas, buildings should be protected with lightning rods. If possible, llamas and alpacas should be moved inside barns or sheds if lightning is predicted. Do not touch an animal that appears to have been electrocuted by downed power lines.

Toxic Chemical Spills

A railroad tank car derailed in a canyon in northern California and spewed toxic chemicals into a river, causing loss of fish and other animal life for many miles downstream. Llama ranches were nearby but managed to avoid contamination. Farms and ranches near highways must always be aware that tanker trucks may have accidents and spill chemicals into pastures, streams, and borrow pits. Don't neglect to properly store toxic chemicals, pesticides, herbicides, and rodenticides at the farm. In the event of fire or flood, improperly stored materials may contaminate enclosures and pastures.

Nuclear Power Plant Accidents

Llama owners may be situated near nuclear power reactors. Accidents releasing radioactive material into the environment have occurred and may occur in the future. The operators of such plants are fully aware of the importance of wind patterns and may establish zones and prioritize risks. At one plant in northern California, the operators visited with nearby residents and discussed evacuation plans for people. Alarms are situated along the roads in the area to warn residents should an accident occur.

Llama owners in the area arranged to teach neighbors how to catch and halter their animals and how to operate the vehicles and trailers necessary to evacuate them. If evacuation is impossible, try to contain llamas or alpacas in barns to avoid radioactive fallout. Feed may also be contaminated by fallout. Officials may have to establish the safety of feed, water, and surroundings before humans may return to the area.

Miscellaneous Disasters/Emergencies

The list is almost endless, including such mishaps as truck and trailer accidents, malfunctioning pumps, animals lost in the wilderness, dog and wild animal attacks, terrorism, and war.

PLANNING AND PREPARATION

The procedures in the following paragraphs are to be accomplished by the owner or manager, but veterinarians should be prepared to advise.

An evaluation should be made of potential disasters that may occur in an area. Determine if there is a special risk on the ranch or farm. An assessment of the degree of all risks should be made. Record the resources that are available on the farm and determine what equipment and supplies need to be immediately available. Make and periodically update an inventory of resources, equipment, and supplies (Table 25.3). Allocate funds to protect the safety of animals. Produce a written plan containing prioritization of animals in the event of an evacuation. Make certain that all farm or ranch personnel are fully aware of the plan and know their responsibilities in carrying out the plan.

Ensure that all animals are identified in the event they must be evacuated or forced to flee and become lost. Keep vaccinations current to add protection if feed and water become contaminated with infectious

Table 25.3. Supplies for owners to have on hand.

Tools for repairing structures, fences
Plastic netting for temporary repair of fencing
Portable radio and extra batteries (instructions from disaster coordinator)
Flashlights
Candles and waterproofed matches
Portable generator
Fuel (gasoline, heating oil, propane, diesel, wood)
Stored feed
Halters and ropes
Gravel, salt
Emergency water (swimming pools, ponds, cisterns, tanks, plastic bottles [5–10 gal, sterilized with household bleach])
Medical supplies (standard first aid kit, bandaging material, splintage, duct tape)

agents. Be prepared to administer first aid to injured animals (stop bleeding, immobilize fractures, dress wounds). You may be the only source of help to the animals. The veterinarian should help procure a first aid kit that can allow him or her to deal with more than first aid (flush conjunctiva to remove ashes, instill ointment into the conjunctiva, treat burns). Don't neglect other animals (dogs, cats, horses, cattle) that may be on the farm or ranch.

In an earthquake in northern California, an owner's house collapsed. No one could go back into the house to retrieve keys to automobiles, wallets, checkbooks, or valuable papers. Consider how you would function without such items.

Insurance

Make certain that insurance policies cover the disaster most likely to occur in your area. Duplicates of insurance policies, contracts, wills, and other important papers should be kept in a safe deposit box or with an attorney or accountant.

Evacuation

Much has been said about this already. Additionally, make arrangements beforehand as to where to take the animals, and make a practice run. Realize that roads and bridges may be impassable. Plan for alternate routes and train animals to enter trucks and trailers.

Feed and Water

Feed is not a critical factor. Like humans, llamas and alpacas may go for days to weeks without feed and survive, unless the weather is cold. If, however, animals must be left behind, figure that a 160-kg (350-lb) llama normally eats 1.6 kg (3.5 lb) of feed per day. A 27-kg (60-lb) bale of hay should sustain a large llama for two weeks or five animals for five days.

Water is a more crucial requirement. A 160-kg (350-lb) llama requires approximately 2.7 L (3 qt) of water daily. Camelids do not break through ice or eat snow to obtain needed moisture. Waterer heaters are usually electrically powered. If auxiliary power is used, only one waterer heater may be accommodated.

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5. Harwell, G. 1993. Dealing with natural disasters in a zoo. In Fowler, M.E., ed. Zoo and Wild Animal Medicine, 3rd ed. Philadelphia: W.B. Saunders, pp. 15–18.
6. Miller, P. 1993. Preventing barn fires, Llamas 7(8):69–72.
7. Mubarak, S. 1993. Are you prepared for fire? LANA News 46(Dec.):24.
8. Ponte, L. 1994. Earthquakes—closer than you think. Reader's Digest, Apr., pp. 57–64.

Appendix 1

Abbreviations Used in this Book

U.S. customary

ac = acre(s)
apoth = apothocaries' weight (pharmaceutical)
Av = avoirdupois
Bid = twice a day
BW. = body weight
d = day(s)
ft = foot, feet
gal = gallon(s)
hr = hour(s)
IA = intra-arterial
IM = intramuscular
in = inch(es)
IP = intraperitoneal
IV = intravenous
lb = pound(s)
mi = mile(s)
Min. = minute(s)
oz = ounce(s)
PO = by mouth or orally
q = every
QRS = quantity sufficient
sp = species
spp = species (plural)
sq = square
SQ = subcutaneous
Tid = three times a day
ton = 2000 pounds
wt = weight
yd = yard(s)
yr = year(s)

Metric

cc = cubic centimeter = ml
cm = centimeter
cu = cubic
g = gram(s)
ha = hectare(s)
J= Joule(s)
kcal = Kilocalorie(s)
kg = kilogram(s)
L = liter(s)
m = meter(s)
mEq = milliequivalent(s)
ml = milliliter(s) = cc
mg = milligram(s)
mm = millimeter(s)
Mum. = mucous membrane
t = metric ton(s) = 1000 kg
µl = microliter
dl = deciliter = 100 ml

Appendix 2

Conversion Tables

Linear

1 millimeter = 0.039 inch
1 meter = 3.281 feet
1 meter = 1.094 yards
1 kilometer = 0.621 mile

1 inch = 25.4 millimeters
1 foot = 0.305 meters
1 yard = 0.914 meter
1 mile = 1.609 kilometers

Volume

1 liter = 33.815 fluid ounces
1 liter = 1.057 quarts
1 liter = 0.264 gallons US

1 fluid ounce = 29.573 milliliters
1 fluid ounce = 0.03 liters
1 pint = 0.473 liters
1 quart = 0.946 liters
1 US gallon = 0.83 .Br. Imperial gal
= 3.785 liters
1 British Imperial gal = 4.545 liters = 1.2 US gallons

Area

1 hectare = 0.004 square miles
1 hectare = 107,639.1 square feet
1 hectare = (10,000 sq meters = 2.47 acres

1 acre = (43,560 sq ft) = 0.405 hectare
1 acre = 4046.86 sq meters

Mass

1 milligram = 1/60 grain (apoth)
1 gram = 0.035 ounce
1 gram = 15.432 grains (apoth)
1 kilogram = 2.2 pounds
1 metric ton (1000 kg) = 1.102 tons
1 mg/kg = 0.454 mg/lb

1 grain (apoth) = 60 milligrams
1 ounce (av) = 28.35 grams
1 pound = 0.454 kilogram (454 grams)
1 ton (2000 lb) = 0.907 metric ton (1000 kg)
1 mg/lb = 2.2 mg/kg

Temperature (degrees Celsius to degrees Fahrenheit)

C	F	C	F	C	F
25	77.1	37.0	98.6	39.2	102.6
26	78.8	37.1	98.8	39.4	102.9
27	80.6	37.2	99.0	39.6	103.3
28	82.4	37.3	99.1	39.8	103.6
29	84.2	37.4	99.3	40.0	104.0
30	86.0	37.5	99.5	40.2	104.4
31	87.8	37.6	99.7	40.4	104.7
32	90.6	37.7	99.9	40.6	105.1
33	91.4	37.8	100.0	40.8	105.4
34	93.2	37.9	100.2	41.0	105.8
35	95.0	38.0	100.4	41.5	106.7
36	96.8	38.1	100.6	42.0	107.6
36.1	96.9	38.2	100.8	42.5	108.5
36.2	97.2	38.3	100.9	43.0	109.4
36.3	97.3	38.4	101.2	44.0	111.2
36.4	97.5	38.5	101.3	46	114.8
36.5	97.7	38.6	101.5	47.0	116.7
36.6	97.9	38.7	101.7	48	118.4
36.7	98.1	38.8	101.8	49	120.2
36.8	98.2	38.9	102.1	50.0	
36.9	98.4	39.0	102.2		

Appendix 3

Sources for Drugs Mentioned in the Text

1. Abbott Laboratories, 100 Abbott Park Rd, Abbott Park, Illinois 60064, USA, (312) 688 5109.
2. Anaquest, Inc. (Omeda Health Care), 110 Allen Rd., P.O. Box 804, Liberty Corner, New Jersey, 0938-0804, (201) 831 2000.
3. Bristol Myers Squibb Co. (Including Mead Johnson), P.O. Box 4500, Princeton, New Jersey 08543-4500, (609) 897 2000.
4. Butler Animal Health Co (Burns), 5600 Blazer Parkway, Dublin, Ohio 43107-7546 (614) 761 9095.
5. DuPont Pharmaceuticals, Wilmington, Delaware, (see Elanco).
6. Elanco, (Pitman-Moore), Division of Eli Lily, 2001 W. Main St. N. Greenfield, Indiana 27408.
7. Fisher Scientific Co., 711 Forbes Ave., Pittsburgh, Pennsylvania, 15215.
8. Fort Dodge Animal Health, Division of American Home Products, P.O. Box 518, Fort Dodge, Iowa 50501, (515)955 4600.
9. Glaxo Smith Kline, 5 Moore Dr., Research Triangle Park, North Carolina 27709 (888)825 825 5249.
10. Lloyd Laboratories, 604 West Thomas Ave, PO Box 86, Shenandoah, Iowa 51601, USA (800) 831 0004.
11. Merck Sharp and Dohme, Division of Merch Chemical Co.,Whitehouse Station, New Jersey, 07065, (201) 574 4000.
12. Miles Inc. Agricultural Division, Animal Health Products, 12707 W. 63rd St., Shaswnee, Kansas, 66201 (800)255 6517.
13. Novartis Animal Health, 3200 Northline Ave. Suite 300, Greensboro, North Carolina 27408, (800)637 0281.
14. Nutritional Biochemicals Corp., 26201 Miles Rd., Cleveland, Ohio, 44128 (800)321 9322.
15. Ortho McNeil Pharmaceuticals, Division of Johnson and Johnson, Camp Hill Rd., Fort Washington, Pennsylvania, 19047,(215) 233 7000.
16. Parke Davis & Co., (merged with Pfizer), 201 Tabor Rd., Morris Plains, New Jersey 07950, (201) 07950.
17. Pfizer Animal Health, 235 East 42nd St, New York, New York 10017, USA.
18. Pitman-Moore Co. (merged with Elanco), P.O. Box 344, Washington Crossing, New Jersey, 08560, (609)737 3700.
19. A.H. Robins Co., Inc. (with Wyeth), 1407 Cummings Dr., Richmond, Virginia, 23220.
20. Roche Laboratories, Division of Hoffman-LaRoche, Inc. 340 Kingsland St., Nutley, New Jersey 07110-0602, USA, (923) 562 9536.
21. Schering-Plough Animal Health, (merged with Merck), 10488 South 136th St., Omaha, Nebraska, 68138.
22. UpJohn Co, 7000 Portage Rd. Kalamazoo, Michigan, 49001, (269)23 4000.
23. United States Department of Agriculture, APHIS-VSL, Ames, Iowa, 50010, (515) 239 8200.
24. Various pharmaceutical and chemical supply companies.
25. Vedco, Inc., Rt. 6 Box 35A, St. Joseph, Missouri, 64507, (816) 238 8840.
26. Wildlife Pharmaceuticals, P.O. Box 2023, Fort Collins, Colorado 80522-2026, USA, (866)823 9314.
27. Winthrop Laboratories 90 Park Ave., New Your, New York 10016, (212) 907 2000.
28. Wyeth-Ayerst Pharmaceuticals, (merged with Pfizer), 555 Lancaster Ave., Madison, New Jersey.
29. ZooPharm, Division of Wildlife Pharmaceuticals, P.O. Box 2023, Fort Collins, Colorado 80522, (866) 823 9314.

Appendix 4

Generic Names, Common or Trade Names and Sources of Drugs Mentioned in the Text

GENERIC	*COMMON OR TRADE*	*SOURCE*
Acepromazine maleate	Promace, ACE, anavet	7,14
Antivenin	Crotalid antivenin	8,28
Atipamezole HCl	Antisedan	26
Atricurium besylate	Tricurium	6
Atropine sulfate	Atropine	24
Azaperone	Stresnil	26
Butorphanol tartrate	Torbugesic	8,26
Calcium borogluconate	Calcium borogluconate	24
Calcium gluconate	Calcium gluconate	24
Carfentanil citrate	Wildnil	36
Chloral hydrate	Chloral hydrate	7,9
Detomidine HCl	Domosedan	11
Dexamethasone	Azium	21
Diazepam	Vallium, Tranimal	20
Diprenorphine HCl	M50-50	36
Droperidol	Inapsine	18
Droperidol+fentanyl	Inovar Vet	18
Doxapram	Dopram	18
Epinephrine	Adrenaline	24
Etorphine HCl	M99, Immobilon	26,29
Flumazenil	Mazicon	20
Guaifenesin	Glycerol guiacolate	10
Haloperidol	Haldol	29
Hyaluronidase	Hyaluronidase	26,29
Hydrocortisone	Solu-cortef	28
Isoflurane	Aerane, Isoflurane	1
Ketamine HCl	Vetalar, ketalar	3,9,16
Lactated Ringers Sol	Lactated Ringers	24
Medetomidine	Domitor	26,29
Midazolam	Versed	26,29
Nalorphine HCL	Nalline	11
Naloxone HCl	Narcan	18,26,29
Naltrexone	Trexan	5,10,26
Phenobarbital sodium	Nembutal	1,4
Phenylbutazone	Butazolidin	4
Prednisolone	Solu-Delta-Cortef	22
Sevoflurane	Utane, sevoflo	1
Sodium bicarbonate	Sodium bicarbonate	24
Sodium hypochlorite	Bleach, household	24
Thiamyl sodium	Surital	16
Tiletamine/zolazepam	Telazol, zolitil	8,26,29
Tolazoline	Tolazine	26,29
Tuberculin	Tuberculin	23
Xylazine HCl	Rompun	10,25,26
Yohimbine	Antagolnil, Yobine	10,26

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