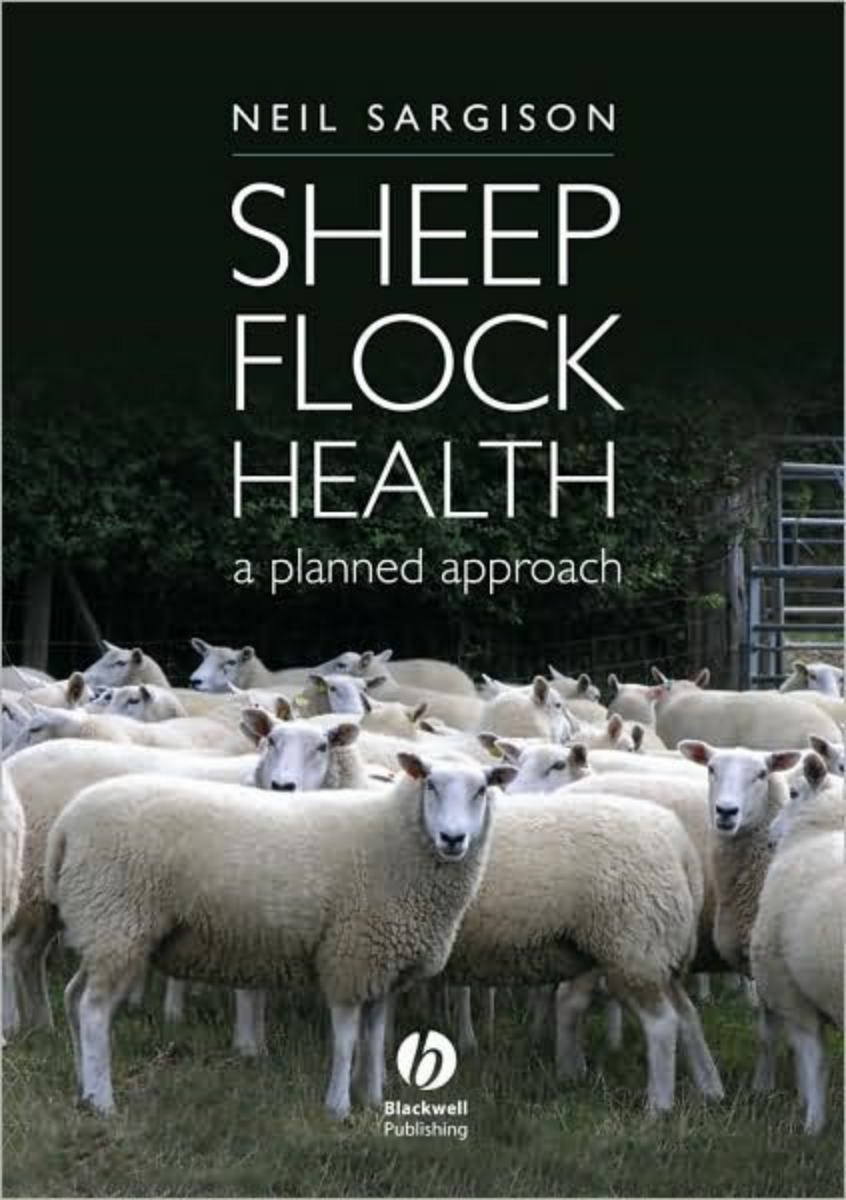
NEIL SARGISON

SHEEP FLOCK HEALTH

a planned approach



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Sheep Flock Health

Sheep Flock Health: A Planned Approach. Neil Sargison
 ISBN: 978-1-4051-6044-5

Sheep Flock Health
A Planned Approach

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Blackwell
Publishing

Blackwell Publishing editorial offices:
Blackwell Publishing Ltd, 9600 Garsington Road, Oxford OX4 2DQ, UK
Tel: +44 (0)1865 776868
Blackwell Publishing Professional, 2121 State Avenue, Ames, Iowa 50014-8300, USA
Tel: +1 515 292 0140
Blackwell Publishing Asia Pty Ltd, 550 Swanston Street, Carlton, Victoria 3053, Australia
Tel: +61 (0)3 8359 1011

First published 2008 by Blackwell Publishing Ltd

ISBN: 978-1-4051-6044-5

Library of Congress Cataloging-in-Publication Data
Sargison, Neil.
Sheep flock health : a planned approach / Neil Sargison.
p. cm.
Includes bibliographical references and index.
ISBN: 978-1-4051-6044-5 (hardback : alk. paper)
1. Sheep–Diseases. 2. Sheep–Health. I. Title.

SF968.S28 2008
636.3′089–dc22
2007039120

A catalogue record for this title is available from the British Library

Set in 10/12pt Times by Graphicraft Limited, Hong Kong
Printed and bound in Singapore by C.O.S. Printers Pte Ltd

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Preface

This book was conceived with the aim of providing farmers and veterinary practitioners with up-to-date information relevant to the diagnosis and management of production-limiting diseases in sheep flocks. Information is provided about a comprehensive range of UK sheep diseases, with particular emphasis placed on those that are common, economically significant, or politically important. Attention is focused on the management of disease rather than just on the treatment of individual animals. This approach is important because the list of licensed pharmaceutical products for the treatment of sheep diseases in the UK is diminishing, and resistance is emerging to a variety of previously useful drugs.

The poor health status of many UK sheep flocks became apparent in the aftermath of the 2001 foot-and-mouth disease outbreak. Furthermore, the spread of foot-and-mouth disease highlighted an inadequacy of national, regional and individual farm biosecurity. In addition to facilitating the spread of exotic disease, poor biosecurity also enables the unchecked and unrecognised spread of important endemic, production-limiting diseases. This book places emphasis both on the identification of existing health problems within sheep flocks and on the flock health management that is required to reduce the risk of introducing new diseases.

The historical system of subsidy support for UK sheep farmers, based first on the variable premium and then on quota-based headage payments, guaranteed a reasonable income but discouraged flexible, progressive flock management. Decoupling subsidy payments from production will progressively reduce subsidy incomes, but will free UK farmers from the complex and restrictive rules surrounding livestock premium payments. In the longer term, public acceptance of continued subsidy support for UK sheep farmers is not guaranteed. Thus many UK sheep farmers must improve the productivity of their flocks if they are to remain profitable. For most, opportunities to produce higher-value products or to reduce fixed costs efficiently are limited, so the most rewarding means of improving economic performance is through improved flock disease management. The aim of this book is to demonstrate the rational approach to the diagnosis of potentially production-limiting diseases and explain management changes to optimise sheep health and productivity.

During the 1980s, New Zealand sheep farmers had to contend with the removal of subsidy support, the collapse of the wool and skins market and a reduction in the European demand for lamb. These pressures created considerable hardship, but those businesses that survived became highly efficient and profitable. This improvement in production efficiency was achieved by adapting to market demands, the active marketing of the New Zealand product, the adoption of new technologies and the development and regular appraisal of a rational approach to sheep disease

control. New Zealand's unique climate and fertile soils favour extremely productive livestock farming on clover and perennial ryegrass pastures, enabling sustainable, unsubsidised agriculture. It is unlikely that unsubsidised sheep-only farming could be sustained throughout the UK. Nevertheless, it is hoped that the author's New Zealand experience emphasising management and based on scientific principles provides a relevant example.

Sheep production cannot be considered in isolation from other forms of land use. For example, on UK hill farms sheep production on its own is often uneconomic. However, sheep can have an important role in maintaining the environment for other activities such as grouse shooting, thus ensuring their profitability. In these situations where maximum sheep productivity is not sought, disease management is nonetheless important.

The layout of this book is organised according to the way in which production-limiting problems are presented, with major sections on the lambing percentage, lamb growth, unexpected disease, ill thrift in adult sheep, neurological diseases, lameness and skin diseases. Some diseases do not fit neatly into any single section, so are described within the context of greatest relevance. Decisions on flock health management can only be made by farmers in conjunction with their own veterinary practitioner, always based on specific individual circumstances. It is hoped that this book will provide a source of information to support the decision-making process.

This book is based on the author's experience as a farm animal veterinary practitioner mostly in Scotland but also in New Zealand. Nevertheless, the diagnosis and management of many sheep diseases is common worldwide and, in those situations where substantial differences exist, it is hoped that this text will at least provide useful information on the principles of diagnosis and control of disease.

The preparation of this book would not have been possible without: the critical and constructive advice of Marian, Andrew, Nicola, Fiona, Catriona and Ian Sargison; the support over the last 23 years of many professional colleagues, in particular Charlie Cameron, Dave West, Phil Scott, Colin Penny, Kath Dun, Gareth Bell, Alastair Macrae, Dave Wilson and Frank Jackson; membership of the Sheep Veterinary Society; and the encouragement, experience and patience of farmer clients of the Seafield Veterinary Group, Keith, Banffshire; Woodside Veterinary Group, Torphins, Aberdeenshire; Institute of Veterinary Animal and Biomedical Sciences at Massey University, New Zealand; and the Royal (Dick) School of Veterinary Studies, Large Animal Practice, Midlothian.

Neil Sargison

Abbreviations

AAD	amino-acetonitrile derivative
AGIDT	agar gel immunodiffusion test
AST	aspartate aminotransferase
ATTM	ammonium tetrathiomolybdate
BDV	border disease virus
BOHB	3 (β) hydroxybutyrate
BSP	bromosulphophthalein
BTV	bluetongue virus
BUN	blood urea nitrogen
BVD	bovine viral diarrhoea
CAE	caprine arthritis and encephalitis
CAP	Common Agricultural Policy
CCN	cerebrocortical necrosis
CFT	complement fixation test
CIE	counter immuno-electrophoresis
CK	creatinine kinase
CLA	caseous lymphadenitis
CODD	contagious ovine digital dermatitis
CSF	cerebrospinal fluid
DEFRA	Department for the Environment, Food and Rural Affairs
EHA	egg hatch assay
EL_4	early fourth-stage larvae
ELISA	enzyme linked immunosorbent assay
ENTV	enzootic nasal tumour virus
ERDP	rumen degradable protein
FSH	follicle stimulating hormone
EU	European Union
FAT	fluorescent antibody test
FECRT	faecal egg count reduction test
FIGLU	formiminoglutamic acid
FWEC	faecal worm (nematode or strongylid) egg count
GABA	gamma amino butyric acid
GGT	gammaglutamyl transferase
GLDH	glutamate dehydrogenase
GNRH	gonadotropin releasing hormone
GSHPX	glutathione peroxidase
HCN	hydrocyanic acid

IFAT	indirect fluorescent antibody test
IGR	insect growth regulator
ITS2	internal transcribed spacer 2
L_1	first-stage larva
L_2	second-stage larva
L_3	third-stage larva
L_4	fourth-stage larva
LAT	latex agglutination test
LDA	larval development assay
LFIA	larval feeding inhibition assay
LH	luteinising hormone
LMIA	larval migration inhibition assay
ME	metabolisable energy
MHC	major histocompatability complex
MJ	megajoules
MMA	methyl malonic acid
NTS	non-transcribed spacer
OIE	Office International des Epizooties
OIKC	ovine infectious keratoconjunctivitis
PCR	polymerase chain reaction
PII	plasma inorganic iodine
PLD	phospholipase D
PMSG	pregnant mare serum gonadotropin
PPR	peste des petits ruminants
SAT	serum agglutination test
SEERAD	Scottish Executive Environment and Rural Affairs Department
SMCO	S-methylcysteine sulfoxide
SOD	superoxide dismutase
T_3	tri-iodothyronine
T_4	thyroxine
VIDA	Veterinary Investigation Diagnosis Analysis

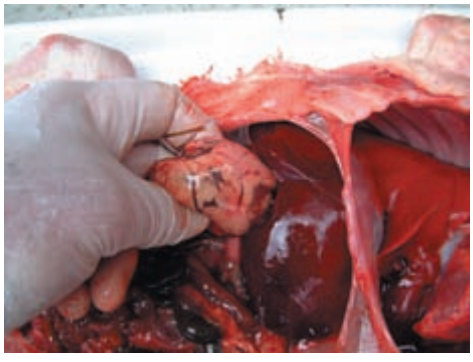

Plate 1.1 Normal brown fat around the right kidney, with an opaque grey-white appearance.

Plate 1.2 Atrophied brown fat around the right kidney, with a translucent, gelatinous, purple-coloured appearance.

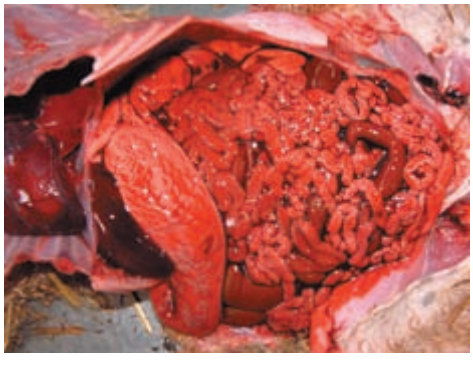

Plate 1.3 The presence of blood in the peritoneal cavity indicates tearing of the liver capsule, associated wither with dystocia or prepartum foetal infection.

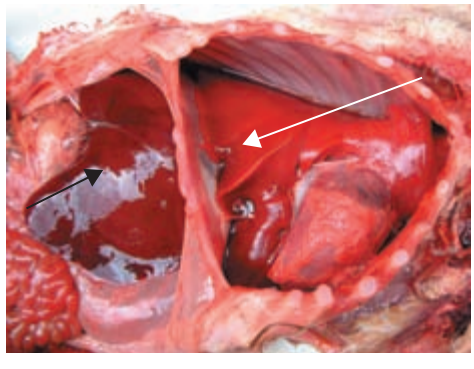

Plate 1.4 Uninflated lungs (*white arrow*) have a similar appearance to liver (*black arrow*).

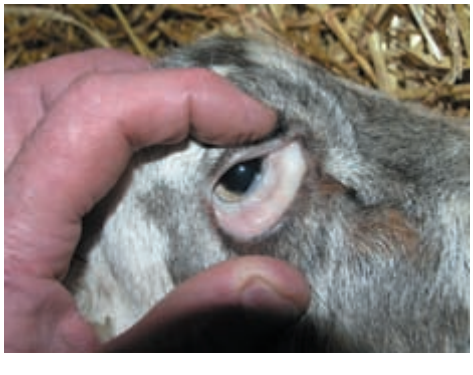

Plate 1.5 Anaemic conjunctival mucous membranes resulting from a uterine tear.

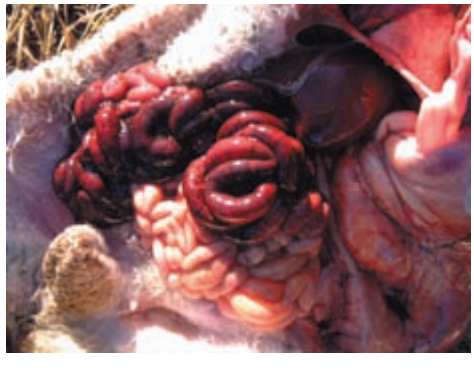

Plate 1.6 Discrete, localised distension and dark red discolouration of the small intestines of a 24-hour-old lamb, due to lamb dysentery.

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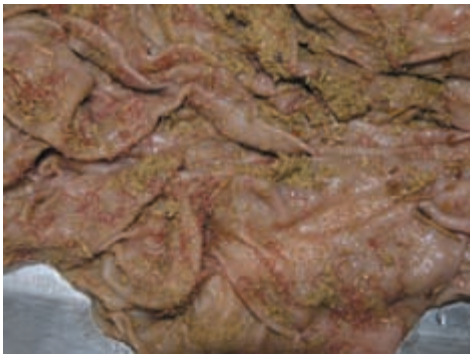


Plate 2.1 Large numbers of *H. contortus* on the mucosa of the abomasum.

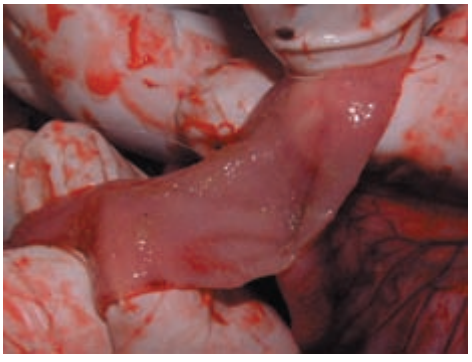


Plate 2.2 Catarrhal enteritis and *N. battus* on the mucosal surface of the small intestine of an 8-week-old lamb.

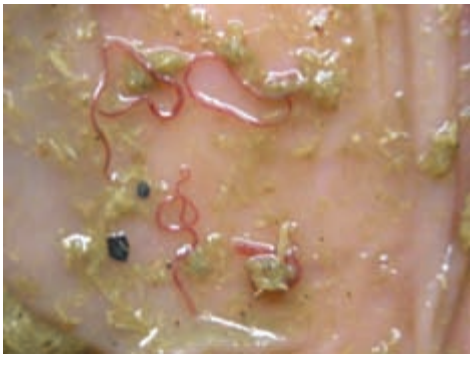


Plate 2.3 *H. contortus* on the mucosal surface of the abomasum, feeding on blood.

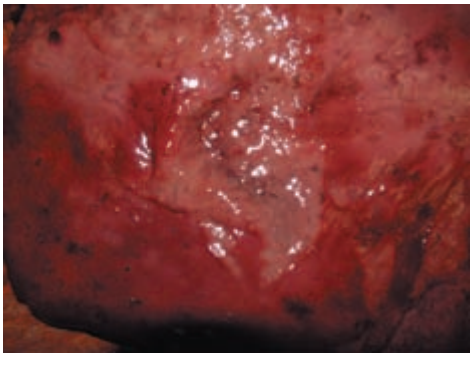


Plate 2.4 Black disease, characterised by a white-coloured zone of hepatic necrosis, demarcated from an otherwise haemorrhagic liver.

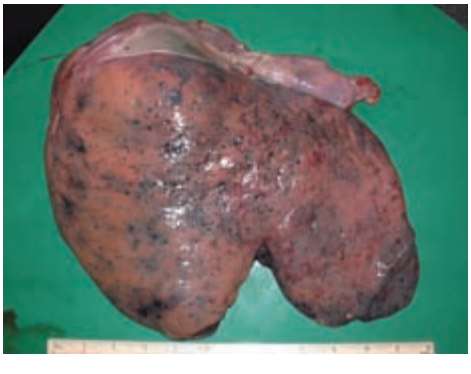


Plate 2.5 A large, swollen, haemorrhagic liver, caused by subacute fascioliasis.

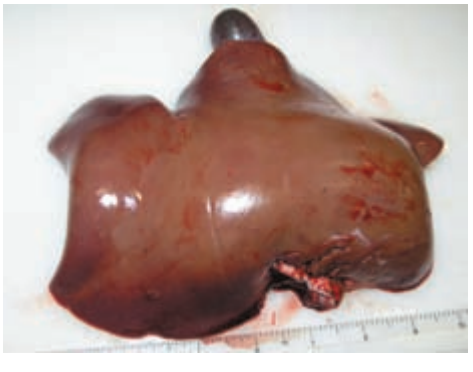


Plate 2.6 A swollen, fatty and friable liver from a 5-month-old cobalt-deficient lamb.

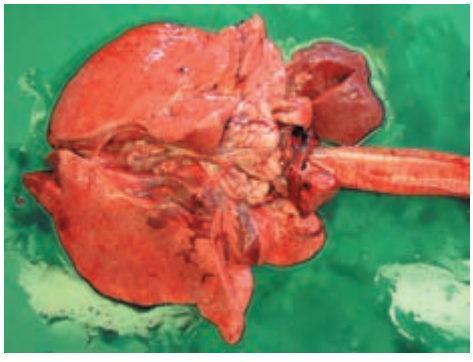


Plate 2.7 Focal areas of lung consolidation and necrosis characterising atypical pneumonia.

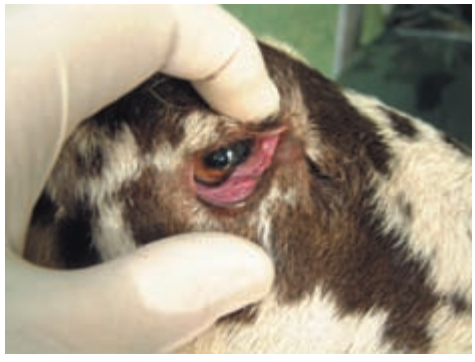


Plate 2.8 Cyanotic mucous membranes indicating severe respiratory disease.

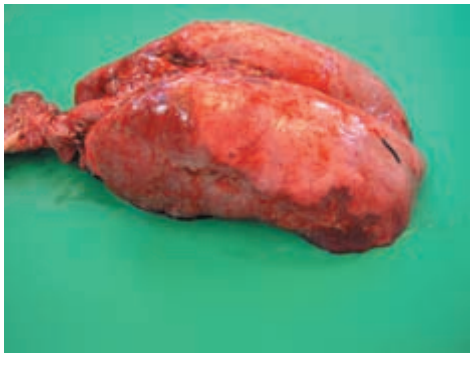


Plate 2.9 Extensive consolidation of the cranial and ventral lung tissue.

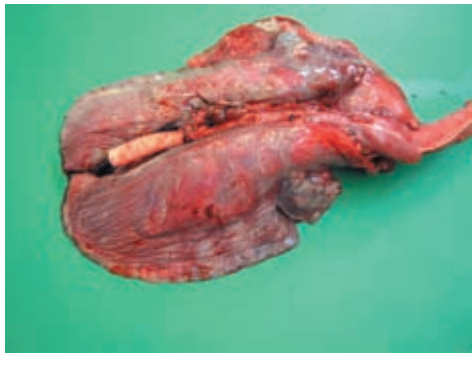


Plate 2.10 Lung consolidation and overlying fibrinous pleurisy.

Plate 2.11 Multiple, raised, grey-coloured lung and subpleural nodules seen as an incidental finding on postmortem examination.

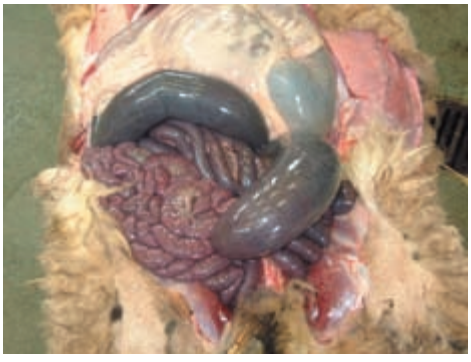


Plate 3.1 Clockwise (when viewed from above) torsion of the small and large intestines, with distension and bright red discolouration of the large intestine and caecum.

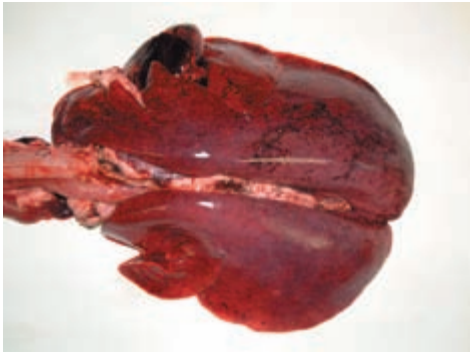


Plate 3.2 Haemorrhages on the surface of the pleura of a lamb which died as a result of systemic pasteurellosis.

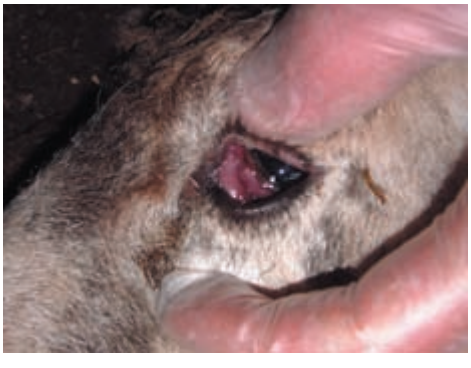


Plate 3.3 Congestion of the ocular mucous membranes of a lamb with systemic pasteurellosis.

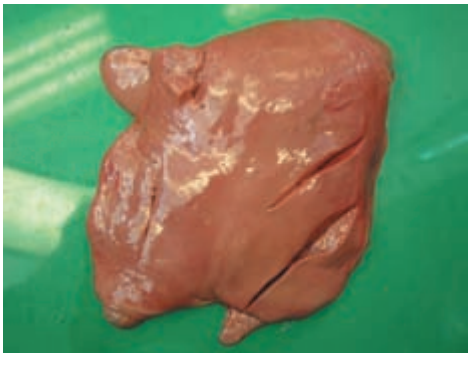


Plate 3.4 Fatty infiltration of the liver is sometimes seen, although this sign also occurs with other diseases such as cobalt deficiency and is normal in late-pregnant ewes.

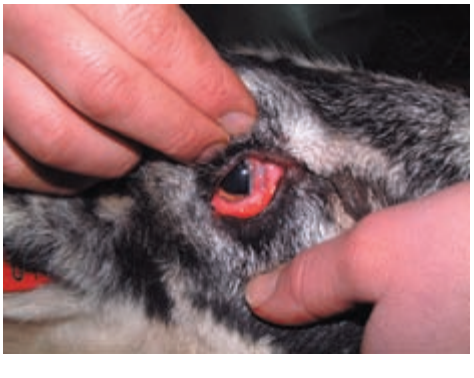


Plate 3.5 Iridescent, bright red conjunctivae characteristic of HCN poisoning.

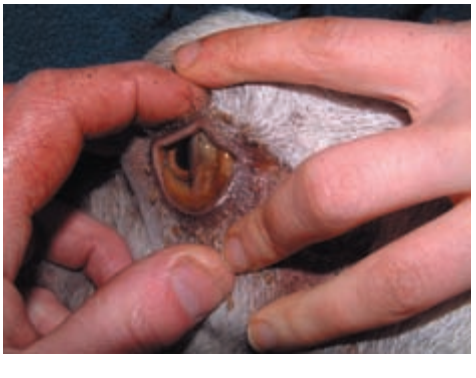


Plate 3.6 Brown-coloured ocular mucous membranes, characteristic of nitrate poisoning.

Plate 3.7 A severely jaundiced, collapsed Bluefaced Leicester shearling ram, affected by chronic copper poisoning.

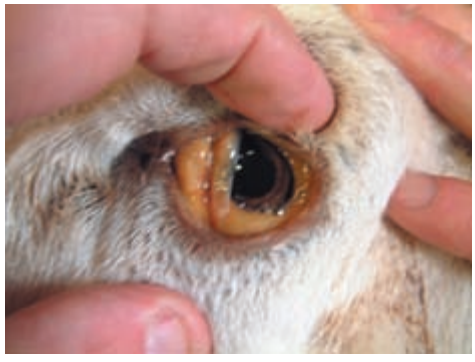


Plate 3.8 Marked jaundice of the conjunctiva of a Bluefaced Leicester shearling ram.

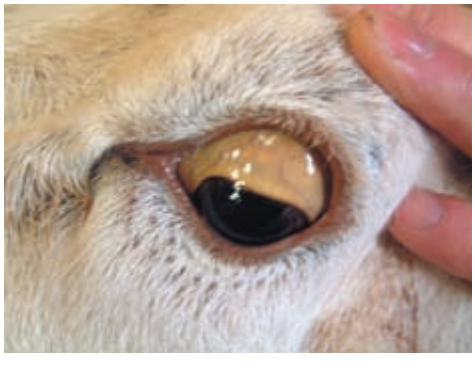


Plate 3.9 Jaundice of the sclera of a Bluefaced Leicester shearling ram.

Plate 3.10 Pallor and jaundice and of the oral mucous membranes.

Plate 3.11 Postmortem appearance of jaundice of the skin and subcutaneous fat of a Bluefaced Leicester shearling ram.

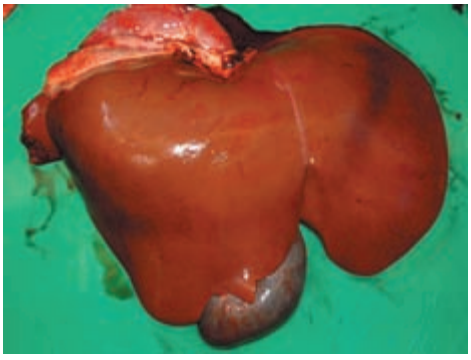



Plate 3.12 (a) Bronze colouration of the liver. (b) Bronze appearance of the cut surface of the liver.

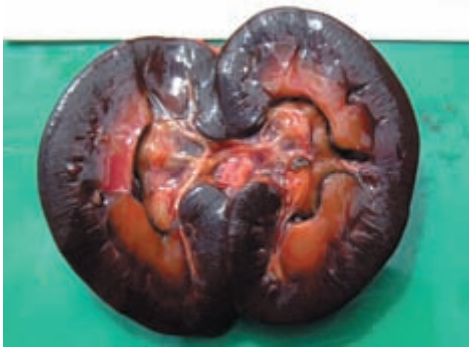



Plate 3.13 (a) Black colouration of the kidneys of a sheep which died of chronic copper poisoning, with a characteristic 'gun metal' sheen. (b) Dark red appearance of the renal cortex and jaundiced medulla.

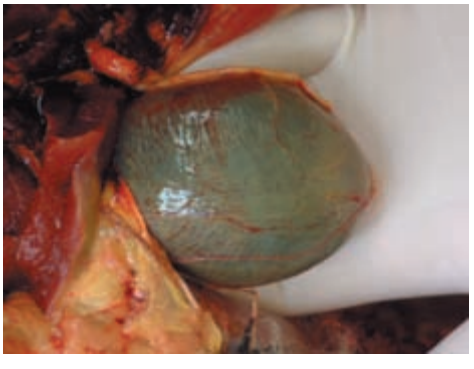


Plate 3.14 The bladder of sheep affected by chronic copper poisoning contains black-coloured urine.

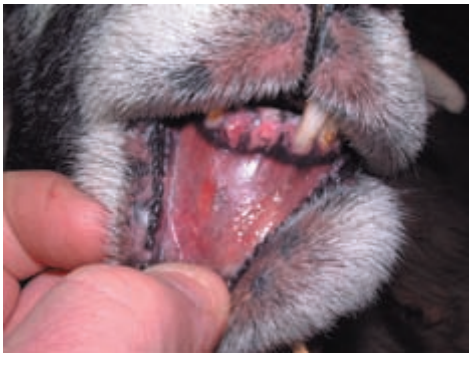


Plate 3.15 Cyanosis of the oral mucous membranes of the Scottish Blackface ewe shown in Fig. 3.56. This ewe subsequently died from struck.

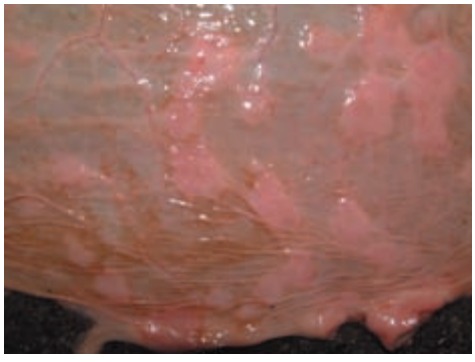


Plate 4.1 Lymphoid nodules on the caecal wall of a sheep with Johne's disease.

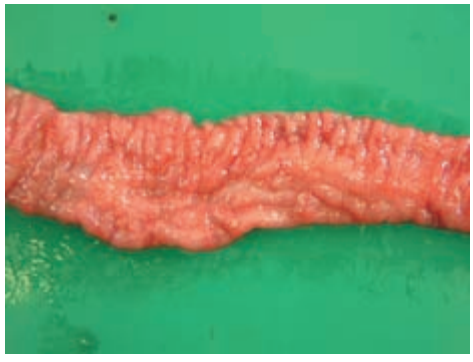


Plate 4.2 Corrugations of the mucosa of the terminal ileum of a sheep with Johne's disease.

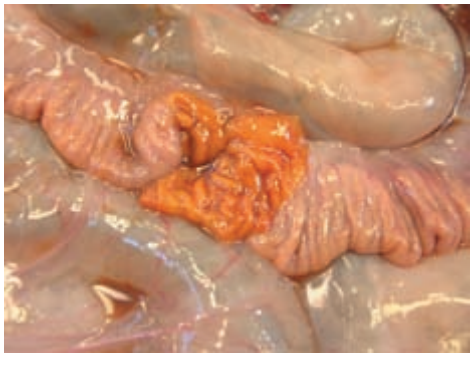


Plate 4.3 Orange pigmentation of the small intestinal mucosa associated with pigmented strains of *M. a. paratuberculosis*.

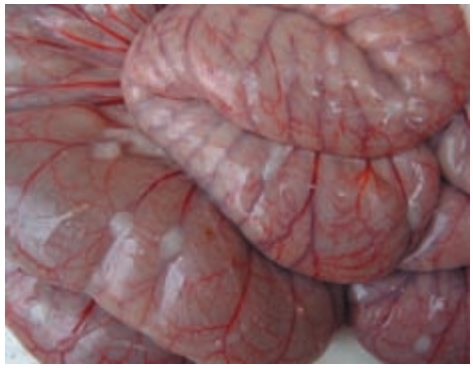


Plate 4.4 Early white, nodular adenocarcinoma lesions on the serosal surface of the small intestine.

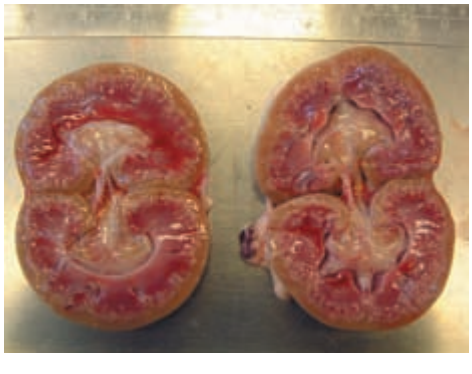


Plate 4.5 Localised lymphosarcoma involving both kidneys of a 1-year-old Greyface ewe lamb.

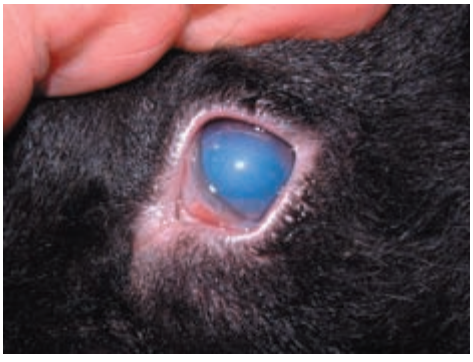


Plate 7.1 Congenital corneal opacity in a week-old Suffolk cross lamb.

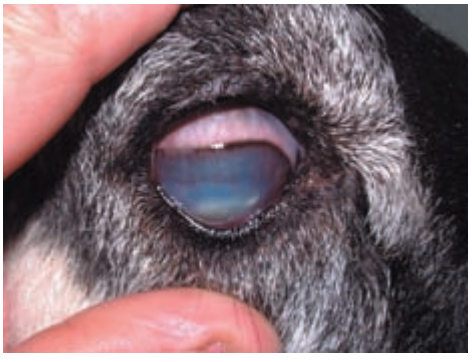


Plate 7.2 Scleral inflammation and keratitis, with peripheral vascularisation and slight opacity of the cornea during the early stages of OIKC.

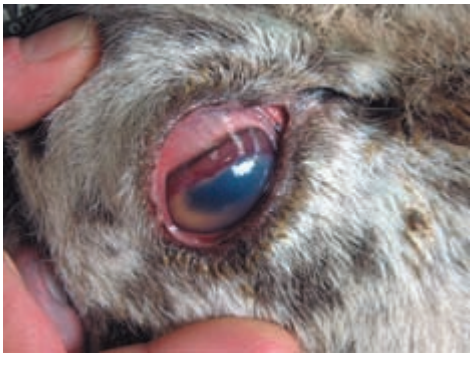


Plate 7.3 Severe scleral inflammation with prominent vascularisation around the periphery of the cornea.

Chapter 1

The Lambing Percentage

The lambing percentage refers to the number of lambs born and surviving until a definite event such as marking or weaning per 100 ewes mated. While a few lowground, upland and hill flocks achieve 220, 180 and 140 percent, average lambing percentages for these flocks are about 170, 150 and 100 respectively. Some variation between flocks, particularly in hill flocks, is associated with differences in climate, altitude, soil type and topography, which cannot be influenced by management, other than to modify their effects. In many hill flocks, the achievement of lambing percentages above 90 is undesirable, because their poor-quality grazing cannot support the metabolic requirements of ewes carrying and rearing twins. In these flocks, improvements in the lambing percentage may only be sustained by a high level of supplementary feeding, which may be both uneconomic and impractical. In lowground flocks, lambing percentages greater than 200 are often undesirable, due to the uneconomic production costs of rearing triplet lambs. Furthermore, triplet-bearing ewes are more prone to pregnancy toxaemia, vaginal prolapse, evisceration through a vaginal tear, and rupture of the prepubic tendon. The mortality rate of low birthweight triplet lambs is typically six times that of twins in the same flock.

Optimisation of the lambing percentage is critical to the profitability of most sheep flocks. While it may not be desirable to improve the lambing percentage in every case, many flocks fail to achieve reasonable targets, offering potential for economic improvement. The lambing percentage is determined by the following events, which are in turn influenced by flock management throughout the year:

- oestrus behaviour
- ovulation rate
- fertilisation
- conception
- foetal development
- foetal survival/abortion
- ewe deaths
- perinatal lamb mortality
- lamb losses from 1 week old to weaning.

Failure to achieve target lambing percentages may be caused by problems associated with any of these events. However, the most important periods which can be influenced by management intervention are mating (oestrus behaviour, ovulation, fertilisation and conception), late pregnancy (abortion) and lambing (ewe losses and perinatal lamb mortality). A basic understanding of each stage is therefore required to ensure a logical approach to the investigation and rectification of poor reproductive performance and lambing percentages.

Sheep Flock Health: A Planned Approach. Neil Sargison
 ISBN: 978-1-4051-6044-5

The mating period

Oestrus behaviour

The oestrous cycle

Seasonal sexual activity in ewes is controlled by the hormone, melatonin, which is secreted by the pineal gland in the brain. Melatonin secretion increases in response to declining day length during the late summer and is reduced with increasing day length in late winter. Melatonin acts on the hypothalamus gland within the brain, stimulating release of gonadotrophin releasing hormone (GnRH), which causes the release of luteinising hormone (LH) and follicle stimulating hormone (FSH) from the pituitary gland. These gonadotrophic hormones regulate the development and maturation of follicles within the ovary. Developing ovarian follicles produce oestradiol, which results in behavioural oestrus (Fig. 1.1).

Following rupture and release of ova, mature ovarian follicles become corpora lutea which secrete the hormone progesterone, necessary for the maintenance of pregnancy. Progesterone also suppresses further gonadotrophic hormone release from the pituitary gland, thus preventing the ewe from coming into oestrus. After 12 to 14 days the non-pregnant uterus produces the hormone, prostaglandin, which destroys the corpora lutea,

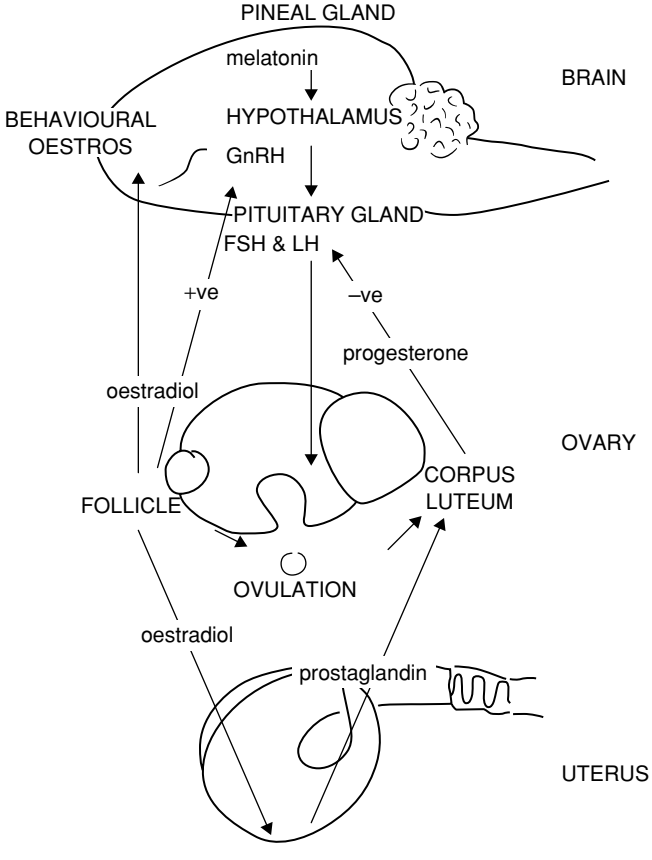


Fig. 1.1 A simplified summary of the hormonal pathways of the ewe's oestrous cycle.

Fig. 1.2 Having been sought out by a ram, an oestrus ewe: repeatedly crouches, while turning her back towards him; nuzzles at his flank as he moves behind her; repeatedly elevates and swishes her tail; stands while turning her head back towards the ram; and follows him.

causing progesterone levels to fall rapidly and enabling the ewe to return to oestrus. If the ewe conceives, the presence of the embryo blocks the release of prostaglandin.

During the breeding season, ewes exhibit oestrus behaviour every 15 to 17 days, unless they become pregnant (Fig. 1.2). Mature ewes show oestrus behaviour for about 24 hours (range 12 to 36 hours), while gimmers show oestrus behaviour for a shorter period of between 30 minutes and 24 hours. Ovulation occurs towards the end of the oestrus period.

The transition period

Growth and regression of ovarian follicles occur throughout the year, but during the non-breeding season these follicles do not ovulate, becoming atresic. Ovulation and behavioural oestrus only occur during the breeding season. During the transition period between the anoestrous non-breeding and breeding seasons, enhanced pituitary activity results in an increase in luteinising hormone pulses, which stimulate an increase in size of ovarian follicles, eventually culminating in ovulation. However, in the absence of progesterone priming, as occurs at the beginning of the breeding season, the first ovulation is not accompanied by behavioural oestrus, but is silent. Corpora lutea arising from this first ovulation often regress prematurely, resulting in a second silent oestrus, again because of inadequate progesterone priming.

Ewe breed

British ewe breeds are all seasonal breeders, only showing oestrus behaviour during a defined period each year. While the length of the breeding season varies with different breeds of sheep, in most cases peak fertility does not occur until late autumn. Scottish Blackface ewes normally exhibit oestrus behaviour from late October to early January, with maximum reproductive activity during late November and early December. Scottish Blackface ewes may therefore only show oestrus behaviour three or four times each year if they do not become pregnant. Dorset Horn ewes may show oestrus behaviour between June and February, with maximum reproductive activity from August to December and 10 to 17 oestrus periods per year. Other ewe breeds show behavioural oestrus between these two extremes. In general, the harsher the environment to which the ewe breed has become adapted, the shorter the breeding season.

Other factors affecting the onset of oestrus behaviour

The onset of the breeding season is influenced, to a lesser extent, by latitude and altitude, being up to 4 weeks earlier in lowground areas in the south than upland areas in

Fig. 1.3 Barbados hair sheep can breed at any time of year, even when kept in the UK.

the north of the UK. In equatorial regions, where daylength varies little, native ewes such as the Barbados hair sheep (Fig. 1.3) can breed at any time of year.

The onset of oestrus activity is earlier in adult ewes than in ewe lambs or gimmers, which cycle later, show behavioural oestrus for a shorter time and are less active in seeking out the ram.

Stressful conditions immediately before or during the mating period may delay the onset of oestrus activity. This effect is most pronounced in ewe lambs and gimmers.

Example

Rams were introduced to flock of 500 Scottish Halfbred ewes (flock 1) in the south-east of Scotland at the beginning of October, with the aim of completing lambing before the start of spring calving in April. Rams were introduced to a similar neighbouring Scottish Halfbred ewe flock (flock 2) in the first week of November. Only 40 percent of the ewes in flock 1 lambed during March, with the majority lambing in April (Fig. 1.4), while in flock 2, 90 percent lambed during the first 17 days of the lambing period in April.

The protracted lambing in flock 1 necessitated a high labour input and failed to achieve the objective of ensuring more time for spring calving. Furthermore, pasture for flushing ewes was not managed to its maximum advantage, the feeding of late pregnant ewes was both inefficient and more expensive and the perinatal lamb mortality rate higher than in flock 2, due to a build-up of environmental pathogen contamination over time. The net results of advancing the joining date to the beginning of the breeding season were a lower lambing percentage and higher production costs.

Fig. 1.4 Scottish Halfbred ewes in the north of the UK do not all show behavioural oestrus when rams are introduced at the beginning of October, resulting in a protracted lambing period and low lambing percentage.

The effect of shearing or plunge dipping is probably of lesser importance than that of prolonged wet weather.

The introduction of rams outwith the peak period of reproductive activity results in a protracted lambing period and high barren rate. The dates of ram introduction and removal, therefore, provide essential information for the investigation of poor

Example

Only 30 (18 percent) of a group of 160 cast-for-age ewes in a flock in the south-east of Scotland were raddle marked after a 5-week mating period. Ten Cheviot ewes were all marked, while 130 of 150 Mule ewes were not marked. Most of the ewes were in reasonable body condition (score 3 to 4 on a scale of 1 to 5), but about 20 Mule ewes were in poorer body condition (score 1.5 to 2 on a scale of 1 to 5) associated with chronic liver fluke. Most of these leaner ewes were raddle marked. Four Texel rams, which had been checked for breeding soundness beforehand, with no abnormalities found, had been introduced on 19 October.

All of the ewes should have been marked by the end of a two-cycle mating period. The possible reasons for ewes not being raddle marked are:

- failure to show oestrus behaviour
- failure of rams to mate oestrus ewes (lack of libido)
- failure of the raddle.

Raddle failure involving all four rams would have been unlikely and did not occur, while poor ram breeding soundness was discounted on the basis of previous examinations. The problem was therefore associated with failure of the ewes to show oestrus behaviour. The time of mating with reference to ewe breed, latitude and altitude, or stressful husbandry could not account for the high proportion of the flock affected. Thus, the most likely reason for failure of the ewes to show oestrus behaviour was pregnancy (Fig. 1.5).

The flock was ultrasound scanned for pregnancy and most of the unmarked ewes were found to be between 6 and 10 weeks pregnant. The 10 Cheviot ewes and leaner Mule ewes were probably raddle marked because they had not shown signs of oestrus behaviour before the introduction of fertile rams, associated with their breed and body condition respectively.

The economic consequences of this problem included the subsequent birth of lambs with poorer genetic merit than planned, inefficient feed management of ewes in late pregnancy associated with uncertainties over their predicted lambing dates, and problems associated with a protracted lambing period.

Fig. 1.5 In this example, the failure of ewes to show oestrus behaviour was associated with the presence of a ram lamb (*circled*). The ram lamb had been kept with the ewes for several months before the mating period, but had somehow been overlooked.

lambing percentages. Consideration should also be given to the altitude and latitude of the farm, stressful husbandry and the proportion and management of ewe lambs or gimmers in the flock. Poor reproductive performance may also be related high ewe culling rates following tooth diseases, jaagsiekte or Johne's disease, which result in an imbalance in age structure of the flock, with a high proportion of ewe lambs or gimmers.

The ram effect

The onset of the breeding season is influenced by the presence of sexually mature rams, referred to as the ram effect. Sexually active rams secrete pheromones from sebaceous and scent glands in their skin, which cause an immediate and rapid increase in the frequency of LH pulses in ewes which are approaching their natural breeding season. Provided that they have been kept away from the sight or smell of rams for at least 3 weeks, most ewes have a silent ovulation, without showing signs of behavioural oestrus, within 3 to 4 days of ram introduction. In 40 to 60 percent of these ewes, the subsequent corpus luteum is maintained for a normal period, followed after 18 to 20 days by normal oestrus activity and ovulation. In the remaining ewes, the corpus luteum regresses after 6 to 8 days followed by silent ovulation. The corpus luteum formed after this silent ovulation is maintained for the normal period and normal oestrus activity occurs after another 18 days. Thus, normal oestrus activity commences with two peaks of activity in the flock between 18 and 26 days after ram introduction.

Once the ewes have commenced normal behavioural oestrus activity, their oestrous cycle is unaffected by the presence of rams. The ram effect therefore only occurs during the transitional period between silent and behavioural oestrus.

Manipulation of the onset of oestrus behaviour

The onset of oestrus activity can be manipulated to advance the breeding season by a few weeks by:

- exploitation of the ram effect
- pregnant mare serum gonadotrophin (PMSG) injections following a period of progesterone priming using intravaginal progestogen sponges
- melatonin implants.

Use of the ram effect

Vasectomised, teaser rams can be used to advance oestrus activity by about one or two weeks and tighten up the lambing period. It is important that the vasectomised rams are sexually mature and have good libido. Ill-thrifty, 'tail-end' ram lambs, or adult rams which have been shown to be unsound for breeding, are inappropriate as teasers. Although vasectomised rams are often left with ewes for a few weeks, satisfactory results can usually be achieved after 48 hours of exposure. Vasectomised rams should be removed and replaced with fertile rams no later than 14 days after their introduction. Two vasectomised rams are usually used per 100 ewes. Fertile rams (Fig. 1.6) can be introduced for 48 hours and used in the same way as teasers, but a small proportion of ewes may become pregnant earlier than required. Exposure to the ram effect synchronises oestrus activity, usually necessitating a ratio of one sound fertile ram per 25 to 30 ewes.

Fig. 1.6 Exposure to entire rams in an adjacent field may be sufficient to induce oestrus behaviour in ewes.

Summary guidelines for the use of vasectomised rams

- Keep ewes away from sight or smell of rams (or bucks) for at least 3 weeks before introducing vasectomised rams.
- Leave vasectomised rams with ewes for 2 to 3 days and definitely no more than 14 days.
- Use about two vasectomised rams per 100 ewes.
- Two peaks of synchronised oestrus activity occur in ewes between 18 and 26 days after introduction of vasectomised rams.
- A high ratio of fertile rams to ewes is needed (one ram per 25 to 30 ewes).

Before considering the use of vasectomised rams, it is important to check that there are sufficient fertile rams to cope with a compact breeding period, and that there is sufficient labour to manage a concentrated lambing period.

Ram vasectomy

Vasectomy surgery is easier to perform on shearlings than on ram lambs or subfertile mature rams. Whenever practical, it is better to vasectomise homebred animals to reduce the risk of introduction of disease to the flock.

Feed should be withheld for about 24 hours before surgery. Vasectomy can be performed under sedation and local anaesthesia; under general anaesthesia; or under spinal analgesia using extradural lignocaine injection at the lumbosacral site. Sedation using intravenous xylazine at dose rates of 0.05 to 0.1 mg/kg and infiltration of 5 to 10 ml of 2 percent lignocaine into the neck of the scrotum is safe and requires less skill than the other methods. However, the technique does not ensure satisfactory relaxation of the cremaster muscles, and often leads to haematoma formation at the site of the surgical incisions, associated with infiltration of local analgesic into the vascular plexus of the spermatic cord. No drugs are licensed for general anaesthesia in sheep in the UK, but pentobarbitone and thiopentone are commonly used for vasectomy surgery. However, recovery can be extended if incremental amounts have been administered during surgery and there are associated risks of gaseous ruminal distension and aspiration pneumonia. Lumbosacral spinal analgesia affords excellent control of pain and cremaster muscle relaxation with no haemorrhage at the operation site. There are associated risks, for example posterior paralysis, leading to urinary retention and eventually bladder rupture has been reported associated with haematoma formation at the injection site, but these are low when compared to the risks associated with general anaesthesia.

Spinal analgesia using extradural lignocaine injection at the lumbosacral site

The sheep is first positioned in sternal recumbency with the hips flexed and the pelvic limbs fully extended alongside the abdomen, thus flexing the spine, resulting in widening of the dorsal lumbosacral space. The lumbosacral space is palpable in all but the fattest of sheep as a depression in the midline 1 to 3 cm caudal to an imaginary line joining the cranial border of the ilium on each side. An area of skin about 2.5 cm^2 is clipped and surgically prepared in the dorsal midline midway between the last palpable lubar dorsal vertebral spine (L6) and the first palpable sacral dorsal vertebral spine (S2). Maintaining a strict aseptic technique a 19 gauge x 1.5 inch needle (19 g x 2 inch in sheep weighing more than 75 kg) is inserted into the lumbosacral space in the midline with the needle point directed at a 10 to 15° angle cranial to the vertical, with the bevel directed cranially, in the plane of the vertebral column (Fig. 1.7). The needle is advanced slowly and carefully through the skin, subcutaneous tissue, supraspinous and interarcuate ligaments, and finally through the ligamentum flavum into the epidural space. A distinct reduction in resistance is felt as the needle penetrates the epidural space. The needle should be advanced no further at this stage to avoid penetration of the leptomeninges. Lignocaine hydrochloride 2 percent, which has been warmed to body temperature, is then injected at a dose rate of 4.5 mg/kg (1 ml per 4.5 kg). A drop of anaesthetic solution can be placed in the needle hub, which will be drawn down the needle by negative pressure when the epidural space is penetrated (commonly referred to as the hanging drop technique) and if the needle point is correctly positioned, the local anaesthetic solution will be injected without any appreciable resistance. Immediately following injection the sheep's head should be held at a level higher than the spinal cord as a precaution against loss of consciousness following accidental subarachnoid injection.

Penetration of the meninges is sometimes unavoidable, resulting in fine muscle tremors and tail swishing, sometimes followed by free flow of CSF from the hub of the needle. In the sheep, the spinal cord extends to the mid sacral region so care should be taken at this stage not to advance the needle any further, avoiding trauma to the spinal cord. If the needle penetrates the subarachnoid space it should be withdrawn until CSF ceases to flow and half the calculated epidural dose administered to avoid the risk of transient loss of consciousness.

Posterior analgesia commences within 5 minutes and lasts for 1 to 2 hours, although it is often 4 hours before motor function returns and the sheep is able to stand.

The technique of lumbosacral epidural analgesia requires a detailed knowledge of anatomical structures, a strict aseptic approach, and the ability to detect subtle changes in resistance to the travel of the needle. It is an excellent technique but can be difficult to perform in large fat adult sheep under field situations. In the UK, epidural analgesia can only be administered by a veterinary surgeon.

Fig. 1.7 A 19 gauge 1.5 inch needle inserted into the lumbosacral space. Spinal analgesia can only be administered by a veterinary surgeon.

Vasectomy surgery

Vasectomy can only be performed by a veterinary surgeon (Figs 1.8 to 1.13). Vasectomised rams should be permanently identified to avoid future confusion. It is recommended that the excised sections of vas deferens are placed in labelled containers of formal saline and submitted for histological examination to confirm that the correct tissue has been removed. Alternatively, any contents of the excised tissue can be milked onto a microscope slide, and examined for the presence of spermatozoa, supporting the identity of vas deferens.

Vasectomised rams are seldom useful as teasers for more than two breeding seasons, because their testes gradually atrophy because of the formation of spermatic granulomas (Fig. 1.14).

Fig. 1.8 The ram is cast and held in a sitting position, restrained by an assistant seated on a seat or small straw bale. The skin over the spermatic cord is shaved, surgically prepared and draped.

Fig. 1.9 A 2- to 3-cm incision is made in the skin antero-laterally over the spermatic cord at a level below the accessory teats.

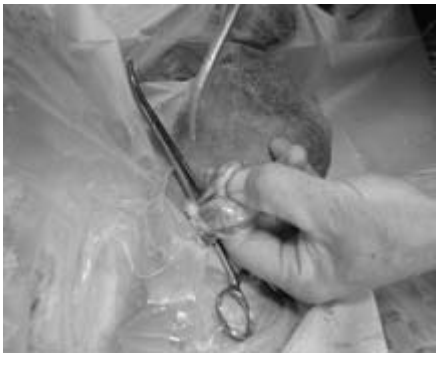


Fig. 1.10 The spermatic cord is exteriorised following blunt dissection and the vas deferens localised as a firm tubular structure medially within the spermatic cord, by rolling the cord between the thumb and index finger.

Fig. 1.11 The tunic is nicked with a scalpel blade point and about 6 cm of vas deferens freed from connective tissue.

Fig. 1.12 Each end of the freed vas deferens is clamped and ligated and the section between the sutures is then removed.

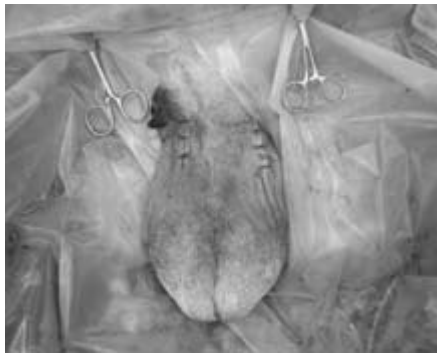


Fig. 1.13 The skin incision is closed with interrupted mattress sutures and the procedure repeated for the other side.

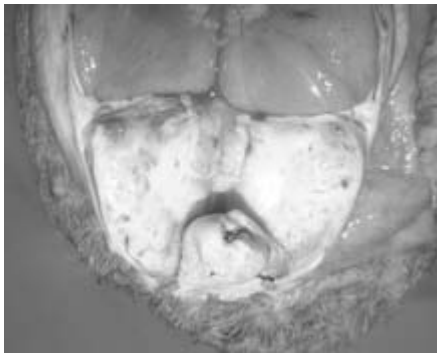


Fig. 1.14 Postmortem appearance of a large spermatic granuloma in the tail of the epididymis of a ram which was vasectomised about one year previously. Granulomas result from the accumulation and leakage of spermatozoa from the epididymal tubules into the surrounding interstitial tissue, where they evoke a 'foreign body' type reaction.

Progestogen sponges and gonadotrophin injections

During the breeding season, intravaginal progestogen (synthetic progesterone) sponges can be used to mimic the effects of natural progesterone from the corpus luteum (Fig. 1.15). Sponges are inserted for 12 to 14 days to provide a period of progesterone priming which outlives any natural progesterone from corpora lutea that may have been present when the sponge was inserted. When the sponges are removed, the sudden fall in progestogen levels precipitates a surge of gonadotrophin hormones that leads to oestrus activity within 36 to 48 hours. Intravaginal progestogen sponges can be used in this way to synchronise oestrus activity in ewes.

Progestogen sponges must be inserted carefully and hygienically to avoid trauma and infection of the ewe's reproductive tract. The procedure should, therefore, only be performed in a clean and dry environment. The applicator should be cleaned between ewes, using only the disinfectant supplied with the sponges.

The presence of distinctive, unpleasant-smelling fluid on sponges when they are removed is normal and does not interfere with the ewe's health or subsequent fertility. However, used sponges are attractive to farm dogs. Sponges can become lodged in the dog's gut, following which the attached strings cut into the intestinal lining, resulting in potentially fatal injury. Individual sponges should, therefore, be carefully disposed of immediately following removal.

Fig. 1.15 Intravaginal progestogen sponges, applicator, antiseptic solution and obstetrical lubricant.

The gonadotrophin surge only occurs in ewes which are already cycling at the time of sponge insertion. Outwith the breeding season, pituitary activity and gonadotrophin release is insufficient to induce oestrus following sponge removal in most ewes. This problem can be overcome by injecting the ewes with PMSG at the time of sponge withdrawal. The combined use of progestogen sponges and PMSG injections can therefore advance the breeding season by 1 to 2 weeks and synchronise oestrus behaviour. Attempting to advance the breeding season by more than about 6 weeks using progestogen sponges and PMSG is generally unsuccessful. When sponges are used out of season in this way, ewes which fail to conceive at the induced oestrus do not usually show oestrus again until the start of the normal breeding season.

PMSG should be stored in a cool and dark place and only reconstituted immediately prior to use. In some animals, PMSG injection increases the ovulation rate, resulting in larger litter sizes, although this effect is variable and unpredictable.

The dose of PMSG must, therefore, be selected carefully to avoid the risk of excessively large litters. Appropriate doses range from 300 to 1000 units per ewe (usually about 500 units). The dose is influenced by:

- Ewe body condition. In general, ewes with body condition score less than 3.5 (on a scale of 1 to 5) require an above-average PMSG dose rate.
- Ewe breed. In general prolific breeds require a lower PMSG dose than less prolific breeds.
- Seasonal oestrus behaviour. Hill sheep with a short oestrus period require an above-average PMSG dose rate.
- The interval from the last lambing. Higher than average doses of PMSG may be required for ewes which lamb more than once per year.

The timing of ram introduction following oestrus synchronisation is crucial. Rams should be introduced 36 to 40 hours after sponge withdrawal and left in for at least 48 hours. If rams are introduced too soon, they may repeatedly serve the first ewes to show behavioural oestrus, depleting their semen reserves for the later oestrus ewes. Ewes should be mated in a relatively confined area with a maximum of 50 ewes per group. Rams should be removed after no less than 48 hours and reintroduced if required 15 days after sponge withdrawal.

Under natural mating systems with a ratio of one ram to 50 ewes, the rams serve between three and five ewes per day during the first few days of the joining period. However, ewes all show behavioural oestrus within a 36- to 48-hour period following

sponge removal, so a ratio of more than one ram to 10 ewes is required. A slightly higher number of rams is required following the use of progestogen sponges outwith the normal breeding season. Alternatively, synchronised ewes can be mated by artificial insemination.

Synchronised ewes generally lamb over a period of about 7 days. However, synchronisation means that the mating date is known, so lambing can be induced using intramuscular soluble corticosteroid (16 mg betamethasone or dexamethasone) injections on day 143 of pregnancy, to lamb within 36 hours. Good records are essential to ensure that ewes are injected no earlier than day 142 to avoid the risk of premature birth and reduced lamb survival. It is therefore particularly important to identify accurately ewes which returned to oestrus and were mated during the second or subsequent behavioural oestrus following sponge removal.

Melatonin implants

The costs involved in early lamb production are high, and it is clearly advantageous to achieve high lambing percentages with these systems. However, many early lambing flocks struggle to achieve lambing percentages above 120, with lambing spread over a 6-week period, less than 50 percent lambing during the first 17 days, and barren rates often exceeding 15 percent (Fig. 1.16). The result may be uneven groups of lambs, many of which fail to reach slaughter weights before prices fall, despite an expensive production system. The problem is associated with the fact that January lambing ewes require to be mated in August, but while some Suffolk ewes may exhibit oestrus behaviour as early as July, the highest lambing percentages are generally achieved when they are mated in late October or November.

Melatonin implants can be injected under the skin of the ear (Fig. 1.17) of early-lambing ewes in June to improve January lambing percentages.

Under natural conditions, melatonin release from the pineal gland increases about 10 minutes after the onset of darkness and returns to basal levels about 10 minutes

Fig. 1.16 Rams are usually introduced to pedigree Suffolk ewes at the beginning of August, with the aim of lambing during the following January. In northern parts of the UK, ram introduction coincides with the transition period or start of the seasonal breeding period, leading to disappointing lambing percentages.

Fig. 1.17 When administrating melatonin implants, care must be taken to maintain hygienic conditions and to avoid unnecessary injury to both ewe (laceration of the ear and face) and operator (deep wounds to the hand or fingers).

after dawn. Increasing darkness triggers seasonal sexual activity. Under UK conditions melatonin implants can override the effect of long daylength to give a modest advance in the breeding season. Ovulation rates can be increased to those of the peak breeding season, although the response depends on the breed of ewe and the depth of anoestrus at the time of implant administration. Implants are most effective when used shortly before the transitional period.

A standard treatment regime is:

- day 1 (30 weeks before lambing starts): remove all rams from sight or smell of the ewes
- day 7: implant ewes and maintain isolation from sight or smell of rams
- day 42: introduce rams.

Peak mating activity occurs about 25 days after ram introduction. Vasectomised rams and progestogen sponges can be introduced to the system during this period to achieve further control of breeding and a more compact mating period.

Ovulation

While the lambing percentage is influenced by several different events, the maximum possible number of lambs born is determined by the ovulation rate. A high ovulation rate is therefore the key to the achievement of a high lambing percentage. In fact, differences in ovulation rate probably account for between 60 and 70 percent of the difference in lambing percentage between flocks.

The ovulation rate can be influenced by:

- stage in the breeding season
- body condition and plane of nutrition
- ewe breed and type
- age of the ewe
- PMSG injections.

Stage in the breeding season

The ovulation rate is generally highest in late autumn during the middle of the breeding season, and lowest at the beginning and end of the breeding season. Provided that the date of ram introduction is unaltered, any management practice which advances the onset of the breeding season, especially in early-lambing ewes, will result in an increased ovulation rate at the time of mating.

Body condition and plane of nutrition

Ewes with body condition scores of 3 to 3.5 on a scale of 1 to 5 generally have higher ovulation rates than leaner animals. The ovulation rate is also influenced by the plane of nutrition before and during the mating period, with improved nutrition during this period referred to as ‘flushing’. The benefits of flushing ewes before mating are well recognised. Ewes which are in moderate body condition (score 2.5 to 3 on a scale of 1 to 5) generally respond best to a rising plane of nutrition for at least 3 weeks before and continuing for about 3 weeks after mating. The response is less defined in

heavy ewes (score more than 4.0), but weight loss should always be avoided during the mating period, because it may result in reduced ovulation rates. Nutritional management of the ewe between weaning and the following mating is therefore critical to ensure optimal body condition at mating. Flushing can often be achieved by moving ewes onto good pasture about one month before and during the mating period. Flushing on good pasture for 4 to 6 weeks can provide for a 0.5 to 1 unit increase of body condition score. Pastures may need to be rested from mid summer for 4 to 6 weeks to ensure adequate growth (8 cm) for flushing in late September/early October, which has implications for the whole farm management in terms of fertiliser requirements and reduced pasture availability for growing lambs. Thus, flushing is sometimes managed by supplementary concentrate feeding.

There is some between-breed variation in response to flushing with Scottish Blackfaces responding well while some types of Cheviots respond poorly. The response is generally least pronounced in naturally prolific breeds.

Excessive body condition can also result in reduced ovulation rates, possibly associated with an effect of circulating fatty acids on reproductive hormone concentrations. Early-lambing ewes can become excessively fat after a prolonged summer dry period, so if possible should be stocked tightly on medium-quality pasture after weaning.

Reduced fertility has been identified in ewe flocks with mean faecal worm egg counts at mating greater than 100 eggs per gram, possibly due to an effect on ovulation rate. This effect depends on the nematode species present and is probably only important when ewes are in poor body condition, but illustrates the need to control worm burdens. However, the decision to dose ewes before mating should be based on assessment of risk including monitoring of faecal worm egg counts rather than on tradition.

Pattern of early growth

Bodyweight is the most important determinant of the onset of oestrus in ewe lambs. On a flock basis, ewe lambs need to exceed 60 percent of their mature bodyweight to achieve reasonable conception rates, sustain pregnancy and ensure optimal future breeding performance. In general, adult ewes reared on a good level of nutrition for their first year have higher ovulation rates than those reared on a poorer level of nutrition, irrespective of their mature weight and body condition.

New Zealand studies have shown that exposure of sexually immature Finn × Texel × Romney ewe lambs weighing about 38 kg to the ram effect for between 8 and 18 days before the introduction of fertile rams can be used to induce oestrus behaviour. Consequent higher ovulation rates at the time of mating and improved lambing percentages have been demonstrated. However, this strategy should only be adopted with caution, because enabling lightweight and immature ewe lambs to become pregnant introduces the risks of problems associated with dystocia and subsequent poor reproductive performance.

Ewe breed

There is considerable between-breed variation in ovulation rates, associated with adaptation to different environments. Thus, the ovulation rates of longwool breeds

such as the Bluefaced Leicester are higher than those of hill breeds. The ovulation rates of hybrid breeds such as the Mule, Greyface and Halfbred are generally intermediate between those of their longwool and hill parent breeds. However, there is also significant within-breed variation in ovulation rates, which in some cases such as the Booroola Merino and Inverdale Romney is regulated by a single gene, which can be selected for. Furthermore, these fertility genes can also be incorporated into other breeds through crossbreeding, potentially to increase their prolificacy.

Age of ewe

The ovulation rates of ewe lambs and gimmers are generally lower than those of adult ewes.

Gonadotrophin injections

Gonadotrophins such as PMSG can be used to increase the ovulation rate of ewes, although the response is variable. Some ewes do not respond, while others overrespond. The main uses of PMSG injections are to induce ovulation outwith the breeding season and to induce superovulation in donor ewes for use in embryo transfer programmes.

Other factors influencing the ovulation rate

Mating ewes on lucerne or red clover with high levels of phyto-oestrogens has been shown to reduce their ovulation rate. This type of effect can be important in Australia, but is of unknown significance in the UK.

In some countries, subclinical liver disease due to the mycotoxin sporidesmin is associated with significant reductions in ovulation rates. Sporidesmin toxicity is unimportant in UK flocks. In New Zealand flocks, the oestragenic mycotoxin, zearalanone, can result in decreased oestrous cycle lengths and increased oestrus duration, with a reduction in ovulation and fertilisation rates. Oestrogenic mycotoxins are probably of little significance in the UK, although their presence in poorly stored grain has been putatively associated with vulval oedema in concentrate-fed, late-pregnant ewes (Fig. 1.18).

Fig. 1.18 Perivulval oedema, colloquially referred to as 'Baboon bum'.

Fertilisation

Under natural mating conditions, achievement of a satisfactory fertilisation rate is dependent on good ram management and breeding soundness. Fertilisation failure is primarily associated with high barren rates, although the barren rate is also influenced by several other factors. The average barren rate for UK flocks following a 35-day mating period is about 5 percent, although many flocks achieve a reasonable target of 2 percent. Barren rates exceeding 5 percent generally merit investigation.

Ram management

The basic requirements of breeding rams are that they can confer the physical characteristics of ease of lambing, rapid growth rate, good conformation, desirable body fat distribution, wool production, resilience or resistance to disease and hardiness to their progeny. The relative importance of these characteristics depends on the production system in which they are used. Rams need to be free from physical defects and production-limiting diseases, immune to those important diseases which are endemic within the flock and sound for breeding purposes.

Nutrition

Rams often lose about 0.5 units of body condition per week during the mating period, so must be in body condition score 3.5 to 4 (on a scale of 1 to 5) at joining. It is also important that they are in good body condition and free of disease for at least 2 months before mating, during which time sperm production occurs. Nutritional preparation for the mating season requires long-term planning and simply turning the rams away to a bare field outwith the mating period is usually inadequate (Fig. 1.19). Care should also be taken to ensure that rams do not become excessively fat (Fig. 1.20). Fat in the neck of the scrotum compromises thermoregulation in the testis, reducing semen quality. Furthermore, rams with body condition scores greater than 4 (on a scale of 1 to 5) have reduced libido. Ram body condition should therefore be monitored regularly throughout the year to ensure that they maintain or can reach target body condition in time for the start of the mating period. In general,

Fig. 1.19 Rams must not be allowed to become too thin outwith the mating period.

Fig. 1.20 Fat and lazy rams have reduced libido and may fail to seek out oestrus ewes.

adult rams can be expected to gain about 0.1 to 0.2 units of body condition score per week when grazed on well-grown, good-quality summer pasture. While it is usually possible to regulate ram body condition through grazing management alone, between 0.5 and 1 kg/day of supplementary concentrate feeding may be required if they fail to reach target body condition or pasture becomes sparse. Any supplementary diet should be formulated to avoid problems of urolithiasis, balanoposthitis or copper poisoning.

Ram to ewe ratio

The ideal ratio of sound rams to ewes during their natural mating period is one to two rams per 100 ewes, depending on:

- enclosure size and topography
- sheep breed
- stage in the breeding season
- age of both the ewes and the rams.

Whenever possible, sheep should be mated in small flat fields to avoid dispersion of the flock and ensure that all oestrus ewes can be found by the rams. When such mating paddocks are unavailable, a higher than average ratio of rams to ewes should be employed (Fig. 1.21). The pasture cover in the mating paddocks should be good, to ensure that ewes can display oestrus rather than foraging behaviour.

In lowground flocks, mated in small flat fields, a ratio of one sound adult ram to up to 80 adult ewes can be used without any reduction in the fertilisation rate, although in most UK flocks a ratio of one ram per 30 to 50 ewes (typically three rams to groups of 100 to 120 ewes) is commonly used. A ratio of one adult ram per 80 to 100 ewes is commonly used in UK hill flocks. Rams are usually rotated after 17 days, or fresh 'chaser' rams introduced, to mitigate against the presence of reproductively unsound animals, and with the aim that ewes which are not pregnant after the first mating will conceive with the new group of rams.

Higher numbers of rams are required for groups of ewe lambs or gimmers. When ram lambs are used, the ratio should not generally exceed one ram per 30 ewes and mating inexperienced ewe lambs or gimmers with ram lambs should be avoided.

Most ram breeds can mate at any time of year, but libido and semen quality deteriorate during the equivalent period to the ewe's non-breeding season. This effect is

Fig. 1.21 Mating on steep slopes is far from ideal. When there is no alternative, then a high ratio of rams to ewes is required.

most pronounced in hill breeds. A higher than average ratio of rams to ewes is therefore generally required during the transition period and at the beginning or end of the breeding season.

General ram management

Sperm production and maturation takes about 2 months, during which period the spermatozoa are susceptible to the effects of stressful handling, excessive heat, or disease. Stressful gathering, shearing, dipping and transport should therefore be avoided during this period and rams should be provided with shade during hot weather (Fig. 1.22).

Some flocks adopt a 50-day joining period, although more than 98 percent of the ewes should be pregnant after two oestrous cycles (34 days). The management of the small number of ewes that conceive over a prolonged period after 34 days is often uneconomical.

Ram harnesses (Fig. 1.23) or keel (Fig. 1.24) can be used to provide reassurance that the rams are working. The crayon or keel is usually changed to progressively darker colours at intervals of about 14 days. Alternatively, harnesses or keel are used from about 16 days after introducing the rams to monitor the number of ewes returning to oestrus. Hard or soft crayons should be used during warm or cold weather respectively. Ram harnesses should be a comfortable fit and frequently checked and adjusted to avoid brisket sores (Fig. 1.25). Infected brisket sores often involve the cartilage of the sternum and respond poorly to antibiotic and supportive treatment. Stressful gathering to adjust harnesses or apply keel should be avoided, for example by training the rams to accept a small amount of concentrate feed from a bucket or bag and continuing feeding throughout the mating period.

Keel or crayon marks can be used to aid future feeding management by enabling pregnant ewes to be separated into different groups on the basis of their predicted lambing dates. The recording of keel or crayon marks should be an important part of the investigation of poor breeding performance, providing information about the mating pattern, submission and return rates.

Management of purchased rams

Replacement rams should be acquired at least 8 weeks before the start of the breeding season, to allow them to adapt to their new environment and diet. Introduced rams should be dosed with a combination of imidazothiazole and milbemycin anthelmintic groups on arrival and housed or placed in an area not intended for subsequent sheep grazing for 48 hours, to reduce the risk of introduction of anthelmintic-resistant nematode parasites. Introduced rams may require further treatment for sheep scab. If rams are brought in from potential fluke areas, they should be treated with triclabendazole to remove adult and immature flukes. Ideally, introduced rams should be separated from the main flock for about 4 weeks, during which period they should be closely monitored for signs of disease. Problems such as lameness and skin diseases should be managed during this period to avoid spread to the main flock. Introduced animals should be vaccinated against clostridial diseases. Where flock vaccination against pasteurellosis and louping ill is required, the introduced rams should also be included in these vaccination programmes.

Fig. 1.22 Rams must be provided with shade during warm and sunny weather.

Fig. 1.23 Raddle marks provide reassurance that rams are working, enable ewes to be batched for late pregnancy management and provide a basis for the investigation of poor reproductive performance.

Fig. 1.24 Provided that rams have been trained to be handled without major disturbance to the ewes, daily application of keel provides a reliable alternative to the use of harnesses, without the associated risks of brisket sores and injury.

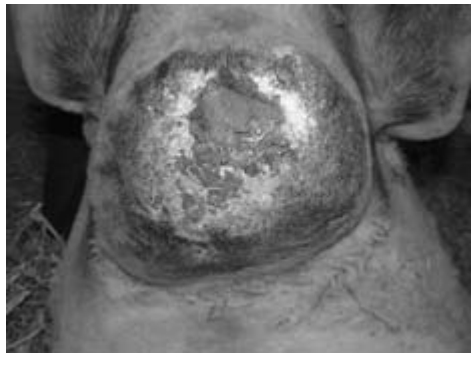


Fig. 1.25 Brisket sores often become infected, with extensive granulation and development of sinus tracts involving the underlying sternal cartilage. Affected rams are unwilling to mate ewes and do not respond to antibiotics and supportive treatment.

To avoid temporary infertility associated with louping ill and tick-borne fever, replacement rams for use in tick-infested areas should be sourced from other flocks within tick areas which experience a similar incidence of these diseases. If this is not possible, homebred ram lambs should be selected and retained, or replacements purchased as ram lambs to allow a period of natural exposure to the tick-borne diseases before use as shearling rams.

Contusive injury to the brain and fractures of the cervical vertebrae of rams due to fighting are common (Fig. 1.26), particularly during the autumn months. Such injuries occur least frequently in established groups of rams, but can prove difficult to avoid following the addition of introduced animals. Penning the ram group in a small area for a period after mixing may help to reduce the incidence of serious injury by reducing the momentum of any impact. Groups of rams should also be tightly

confined after shearing or plunge dipping, until they recognise each other and re-establish their order of dominance.

Ram sales are an integral part of sheep farming life. However, many of the practices which are required to ensure the maximum sale price are not conducive to optimal subsequent breeding soundness. Sale rams are frequently over-fat and struggle to acclimatise to a new harsher environment and feeding regime, while the common practice of feeding of large quantities of oestrogenic brassicas (Fig. 1.27) and heat stress associated with transport, shows and sales (Fig. 1.28) may reduce fertility. Furthermore, differences in feeding regimes and in the skill of preparation of rams for sale make between-flock comparisons difficult. Ram sales also provide an opportunity for the spread of diseases such as sheep scab and caseous lymphadenitis. The alternative strategy of selling rams directly from the farm of origin on the basis of their estimated breeding values for liveweight gain and wool growth overcomes most of these problems and is widely practised in countries such as New Zealand (Fig. 1.29). This

Fig. 1.26 An extreme case of abscessation of the atlanto-occipital joint leading to meningitis in a Texel ram. This lesion probably resulted from haematoma formation associated with contusive fighting injury.

Fig. 1.27 Cabbages are traditionally fed to ram lambs with the aim of making their heads and other features appear 'stronger' in deference to perceived market requirements.

Fig. 1.28 Heat-stressed, fat Beltex rams at a large ram sale. The cyanotic, mouth-breathing ram on the left is unlikely to be reproductively sound.

Fig. 1.29 A ram sale ring within a New Zealand wool shed. Most New Zealand rams are sold directly from their farm of origin on the basis of production criteria rather than fashionable appearance.

strategy has the added benefit of enabling purchasers to assess the environment in which the replacement rams were reared, but may be impractical for smaller UK flocks.

Selenium deficiency has been associated with poor fertilisation rates due to a role of non-glutathione peroxidase selenoenzymes in testis metabolism and semen membrane quality. Where selenium deficiency is recognised within a flock, rams should be supplemented at least 8 weeks before the start of the mating period.

Ram breeding soundness

Overseas, where for several decades breeding soundness has been as important a ram selection index as conformation and growth rate, satisfactory flock reproductive performance is commonly achieved with a ratio of less than one ram to 100 mixed-aged ewes. Ratios of more than one ram to 40 ewes are commonly employed in UK flocks, where it is estimated that between 3.5 and 10 percent of rams are unsound. The use of unsound rams generally reduces the lambing percentage and may result in a protracted lambing period. These problems are compounded when an unsound ram is the dominant animal in a group. Ram breeding soundness is particularly important where groups of pedigree ewes are single-sire mated, or where a high ratio of rams to ewes is required following synchronisation with intravaginal sponges.

Breeding rams require normal genital organs, normal libido, the physical ability to mount and mate ewes and an adequate number of normal spermatozoa in each ejaculate. Rams must therefore be mobile enough to locate and mate ewes in oestrus, hence the importance of good limb conformation, and free from congenital or acquired abnormalities of the reproductive tract, which could lead to altered sexual function and poor fertility.

Ram breeding soundness examination

Rams should be checked for breeding soundness well in advance of the mating season to allow time for the purchase of replacements if necessary. In commercial flocks, rams are usually examined 6 to 8 weeks before the mating season, when they should be sexually active. The thoroughness of the breeding soundness examination depends on the reason why it is performed:

- To ensure good reproductive performance in commercial flocks.
- Where rams are to be used for single-sire mating or as semen donors for artificial insemination.
- To investigate the cause of poor reproductive performance in a group of ewes.
- For insurance or legal reasons.

A useful preliminary assessment of the breeding soundness of otherwise healthy rams can be made by palpation of the scrotal contents. Such examination is straightforward and is usually performed on standing animals in a race or pen. Sound rams during the breeding season have two large, firm, freely mobile testes, which are similar in size. The testes should be resilient to compression and neither soft nor hard on palpation. The tails of the epididymes should be firm and smooth and heads of the epididymes and spermatic cords should be free from nodular defects or hard swellings. Most mature rams should have a scrotal circumference at its widest point

between 30 and 40 cm. The scrotal circumference and testis volume are highly correlated and rams with large symmetrical scrotal contents free of defects are likely to produce large quantities of good-quality semen, while those with small soft testes or other palpable defects are likely to produce poor-quality semen. Rams which are identified as being of questionable breeding soundness may be further investigated by the collection and evaluation of semen samples. A very small number of unsound rams may be missed when this protocol is used, but it remains a practical and efficient method for use on commercial flocks.

When ram soundness examinations are performed because of poor previous reproductive performance, or for legal and insurance purposes, the investigation should include a relevant history, full clinical examination, thorough genital examination and semen examination.

Relevant history:

- Establishment of the ram's permanent identification conclusively.
- Information about the ram's general health, especially during the two months before examination.
- Information about the ram's reproductive performance and previous breeding soundness examinations.
- Information about previous feeding management.
- Vaccination history.

Physical examination:

- Perform a basic clinical examination.
- Record body condition or weight.
- Assess the ram's general conformation.
- Check for signs of lameness.
- Check the teeth and eyes.

Genital examination:

- Palpate the scrotal contents.
- Measure the scrotal circumference.
- Check for evidence of skin flushing in the inguinal area.
- Examine the prepuce and extrude and examine the penis.

Physical examination

Rams must be physically sound if they are to seek out and mate oestrus ewes. Furthermore, it is important to ensure that they will not pass hereditable defects to their offspring. Stress associated with chronic disease or injury, such as lameness, results in production of poor-quality semen. It can take 2 months following a bout of pyrexia before normal semen production is resumed. This period is longer whenever pyrexia persists for more than a few days.

The ram's conformation and gait should be assessed. Sound pelvic limb conformation is important because the pelvic limbs bear most of the weight when serving ewes. The limbs should be angled caudally at the stifles and cranially at the hocks, so that contusive forces during locomotion and mounting are distributed evenly. Excessively straight pelvic limb conformation, as shown by certain heavily muscled European breeds, can result in hip and stifle arthritis and consequent poor breeding

Fig. 1.30 'Post leg' pelvic limb conformation in a Texel ram. The hock and stifle joints are aligned vertically, and unable to cushion contusive forces associated with supporting the animal's bodyweight.

Fig. 1.31 A ram is restrained by sitting on its pelvis facing the operator for examination of the genital tract. This animal has a swelling immediately anterior to the scrotum associated with injury to the penile urethra and is permanently unsound.

soundness (Fig. 1.30). Arthritic problems are compounded in these rams by injury resulting from failure to achieve a suitable posture during mating.

Genital examination

The genital tract can be examined with the animal in a sitting position facing the operator (Fig. 1.31). With the animal in this position, the opportunity should not be missed to check the brisket for evidence of sores and to examine the feet and eyes. The skin of the inguinal area is examined for purple colouration indicative of seasonal sexual maturity. The prepuce is checked for swellings and ulceration and with the ram pushed forwards, the penis is then secured near to its sigmoid flexure, extruded and examined for evidence of damage to the urethral process, inflammation, ulceration, or necrosis. The scrotal skin is examined for evidence of mange, vasectomy scars, infected shearing cuts, or discharging sinuses. The scrotal contents are then palpated and any abnormalities noted (Fig. 1.32).

The scrotal circumference should be measured (Fig. 1.33). Size differences between the testes of ram lambs may be associated with different rates of testicular descent at puberty. These animals should be noted and re-examined a few months later. The age at which ram lambs reach puberty varies between breeds and different management systems. Terminal sire ram lambs may reach puberty at about 5 months of age.

Semen evaluation

Assessment of the gross appearance, motility, ratio of live to dead spermatozoa, morphology of the spermatozoa and presence of inflammatory cells in semen samples provides a useful index of ram breeding soundness.

The method of semen collection depends partly on the reason for the examination. Semen which is to be used for artificial insemination is usually collected from trained rams using an artificial vagina, but in most other cases an electroejaculator is used. When samples are to be examined for bacteriology the ram should be restrained in lateral recumbency, the penis extruded, held with a piece of soft bandage and the

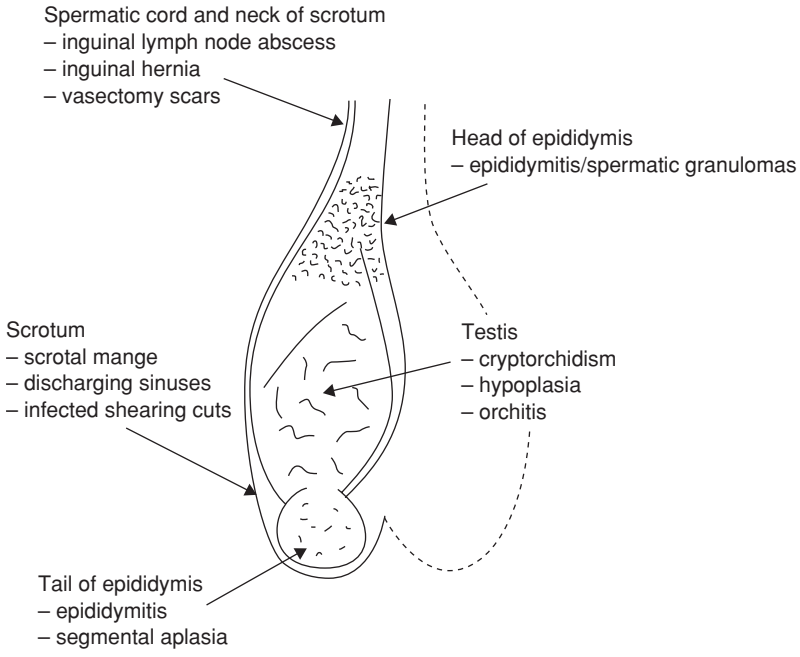


Fig. 1.32 Schematic representation of the scrotal contents, showing sites of common abnormalities.

urethral process directed into a sterile collection jar. In other cases non-sterile samples can be collected into plastic cups from standing animals. The microscope stage, pipettes and stains should be prepared beforehand and semen collection vessels should be warm to prevent seminal damage due to cold shock.

The ram electroejaculator which is most commonly used in practice is based on the New Zealand Ruakura model which has two annular electrodes at the tip of a 2 cm diameter probe (Fig. 1.34). The probe is lubricated and inserted carefully into the

Fig. 1.33 Use of a scrotal tape. Sound rams should have a scrotal circumference between 30 and 40 cm, depending on their breed and age.

Fig. 1.34 A Ruakura-type ram electroejaculator probe.

rectum to a depth of about 15 cm for most mature terminal sire breeds. With the handle of the machine held in the midline against the ram's tail and the probe directed slightly ventrally, a small oscillating electrical current is directed towards the lumbar sympathetic and sacral parasympathetic nerves for about 5 seconds. As the current is turned off the ram may briefly extend its pelvic limbs before thrusting and ejaculating. If no ejaculate is produced, the procedure can be repeated after about 5 seconds' rest. Three or four attempts may be required in some animals, but when no ejaculate is produced after four attempts the procedure should be abandoned and only repeated about 24 hours later if justified.

Muscular spasms of the pelvic limbs associated with electrical stimulation have understandably led to concerns about the ram's welfare during the procedure. This physical reaction may be attributed to the fact that the annular electrodes stimulate nerves other than those required for electroejaculation, in particular those supplying muscles of the hind limbs. The problem of undesirable muscular stimulation may be partly overcome by using probes with longitudinal ventrally placed electrodes (Fig. 1.35), although such machines are neither widely available, nor proven in general practice. The relative aversiveness associated with electroejaculation has been assessed by repeatedly measuring the time taken to move rams through a series of races to a pen where they were either electroejaculated using a Ruakura-type probe; part-shorn; or not handled. While electroejaculation was shown to be more aversive than non-handling, it was not more aversive than part shearing. Shearing is an animal husbandry necessity, which is not generally considered to incur significant welfare concern. While electroejaculation has not been shown to be a serious welfare concern, the procedure should be restricted to rams of questionable breeding soundness and should not be used routinely during flock examinations.

Good-quality ram semen collected by electroejaculation should be thick and creamy and free from pus. A swirling wave motion should be obvious when a drop of semen is examined under low power microscopy. A drop of semen can then be stained with a drop of 5 percent nigrosin and 1 percent eosin, made into a thin smear and examined under an oil immersion objective for the assessment of the live:dead ratio and identification of morphological spermatozoal defects. Normal ram semen may contain up to 20 percent abnormal spermatozoa (Fig. 1.36). Primary abnormalities include detached heads, abnormally shaped heads, double heads or tails and dag defects of the tail. Secondary abnormalities associated with sperm maturation include protoplasmic droplets and coiled tails. Seminal abnormalities should be interpreted in conjunction with the history and clinical signs. When abnormalities are detected in otherwise normal animals, it may be appropriate to resample after a few weeks to rule out the possibility of apparent defects associated with poor sample handling. Semen samples can also be stained with methylene blue or Leishman stain to determine the presence of large numbers of inflammatory cells. Samples collected directly into sterile containers can be submitted for bacterial culture.

Ultrasonography

Trans-scrotal ultrasonography using a B-mode real-time scanner with a 7.5 MHz linear array transducer is a useful non-invasive technique for the identification of scrotal pathology in the ram. The main role of ultrasonography is to aid the interpretation of uncertain palpation findings, but it can also be used to detect early lesions

Fig. 1.35 A ram electroejaculator probe with longitudinal electrodes.

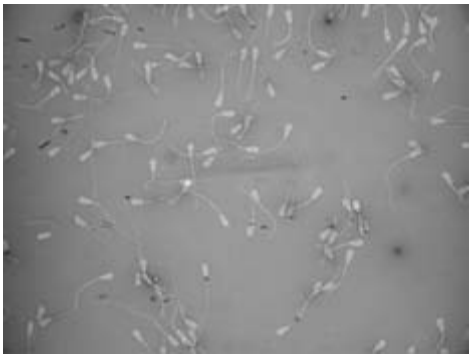


Fig. 1.36 A ram semen smear stained with 5 percent nigrosin and 1 percent eosin.

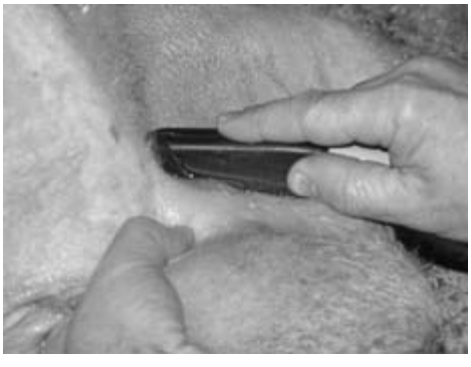


Fig. 1.37 Ultrasound examination of a ram's scrotal contents. The probe is aligned longitudinally along the scrotal contents. The procedure can also be performed on standing rams.

Fig. 1.38 The pampiniform plexus of normal rams is identified in the neck of the scrotum, extending to the proximal margin of the testis, with the appearance of numerous convoluted non-echogenic tubules.

which cannot be identified by palpation. Ultrasonography also offers the facility to monitor changes in lesions.

The hair or wool of the scrotum is first closely clipped and the skin wetted. Coupling gel is liberally applied to the scrotal skin or scanner head to exclude air and ensure a good contact between skin and transducer (Fig. 1.37). Starting at the neck of the scrotum, the transducer is moved along the caudal surface of the scrotum to obtain either transverse images of both scrotal contents, or longitudinal images of one spermatic cord, testis and epididymis (Figs 1.38 to 1.40).

Following breeding soundness examination, rams can be classified as sound at the time of examination, temporarily unsound or permanently unsound. In some cases follow-up investigations may also be appropriate.

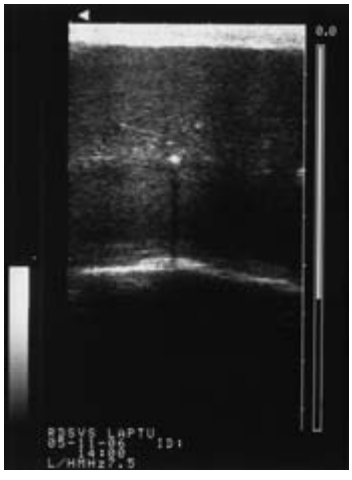


Fig. 1.39 The testicular parenchyma of normal rams has a uniformly moderately echogenic appearance. It is surrounded by a distinct hyperechoic line representing the testicular tunics and skin. The intertesticular septum appears as an hyperechoic line between the two testes. The head of the epididymis is slightly more heterogeneous and less echogenic than the testis, but is masked by the pampiniform plexus and difficult to identify in normal rams.

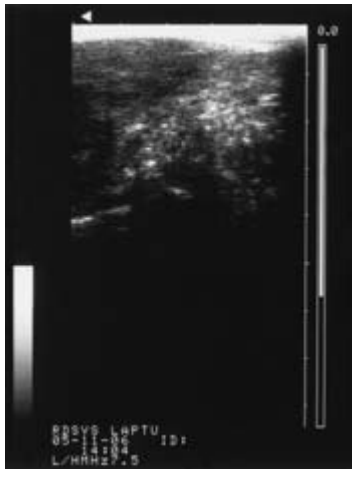


Fig. 1.40 The tail of epididymis has a more heterogeneous and less echogenic appearance than the testis.

Common causes of poor ram breeding soundness

Epididymitis

Epididymitis is a common cause of permanent breeding unsoundness in rams. In the UK most cases are associated with Gram-negative pleomorphic bacterial infections, but overseas the most important infectious cause of epididymitis is *Brucella ovis*.

The term Gram-negative pleomorph refers to a group of bacteria with similar morphologic and biochemical properties, which includes *Actinobacillus seminis*, *Haemophilus somnus* and *Histophilus ovis*. In New Zealand, infection commonly causes epididymitis in 3 percent of young rams, although this high incidence of the disease only became apparent with the advent of routine ram soundness examinations, which was driven by a need to control *B. ovis* infection. Annual wastage of up to 10 percent of sale ram lambs has been reported. The incidence of epididymitis in UK flocks is unknown, because most rams are not routinely checked for breeding soundness.

The severity of presenting clinical signs varies. In the majority of cases no overt clinical signs are seen and epididymitis is only identified when abnormal scrotal contents are palpated during ram breeding soundness examination. Some rams stop grazing, isolate themselves and become recumbent, while others display a unilateral, straddled pelvic limb gait. In severely affected rams the scrotum is hot, swollen and painful during the acute stages of the disease (Fig. 1.41), although in most cases scrotal palpation causes no apparent discomfort. Lesions associated with Gram-negative pleomorphic bacterial infections are usually found in the tail of the epididymis,

Fig. 1.41 Painful swelling of the left scrotal contents associated with epididymitis in a Bluefaced Leicester ram lamb.

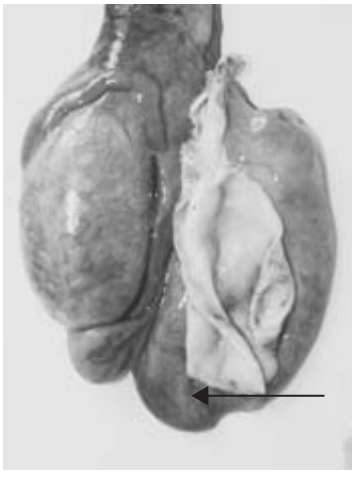


Fig. 1.42 Inflammation of the tail and body of the left epididymis (*arrow*) with fibrous adhesions to the tunica vaginalis.

which is hard and enlarged and often covered by a thickened layer of vaginal tunic and skin (Fig. 1.42). In some cases, the body and head of the epididymis are also affected and contain multiple firm granulomatous lesions. Testicular atrophy usually follows. A discharging periorchitis is infrequently seen, where abscesses in the epididymis rupture through the scrotal wall and discharge thick creamy pus.

Epididymitis lesions are associated with the formation of spermatic granulomas caused by leakage of spermatozoa from the inflamed epididymal tubules. Spermatic granulomas have the ultrasonographic appearance of poorly defined irregular anechoic lesions within the epididymis (Fig. 1.43).

Understanding of the pathogenesis of epididymitis due to Gram-negative pleomorphic bacterial infection is largely based on studies in South Africa, where epididymitis occurred mainly in breeding flocks, with a peak incidence in ram lambs during the autumn, coinciding with the onset of puberty, and often associated with poor environmental hygiene. Gram-negative pleomorphic bacteria are not obligate pathogens of the reproductive tract. They have been associated with suppurative polyarthritis and mastitis in ewes, meningoencephalitis in lambs, abortion in ewes and metritis in ewes. This ability of the organism to cause infection at a number of sites other than the genital tract has led to the hypothesis that epididymitis is the result of an ascending urogenital tract infection rather than bacteraemic spread from another site.

The preputial cavity is in direct contact with infected bedding or soil when the ram lies down and can be invaded by various organisms. Suitable conditions are created for the migration of bacteria in the sheath to the deeper lying organs of the genital tract when, under the influence of systemic hormonal stimulation, the genitalia undergo development. The bacteria can then initiate disease in the seminal vesicles, epididymes and testes. In problem flocks, there appears to be a stepwise progression of infection from the opening of the urethra to deeper tissues. Gram-negative pleomorphic bacteria cause prostatitis with abscessation and seminal vesiculitis in a large proportion of ram lambs. However, while many ram lambs become infected, the majority become free of genital infection by 15 months of age and do not develop clinical signs of epididymitis.

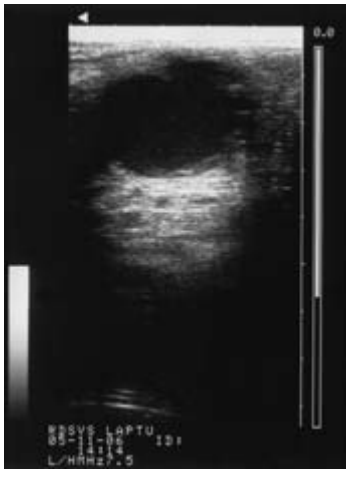


Fig. 1.43 Ultrasound image of epididymitis.

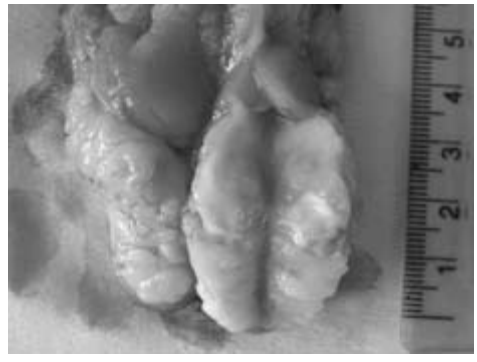


Fig. 1.44 Abscessation of the tail of the epididymis of a 2-month-old ram lamb, associated with non-specific bacteraemia.

The urethra and accessory sex glands can be infected without clinical signs of epididymitis. Thus infection can spread in the semen of palpably normal rams, and the tendency for ram lambs to congregate in small groups in paddocks and mount each other may contribute to the spread of the disease. The incidence of epididymitis is higher where ram lambs are kept under intensive conditions rather than under open range conditions. There is an association with trough feeding, probably because trough-fed ram lambs spend a longer time lying down compared to those grazed extensively. Other factors such as confinement and moist soil conditions may contribute to the spread of the disease.

On the basis of our current understanding of Gram-negative pleomorphic bacterial infection, the best advice for problem flocks is to graze ram lambs in several small groups rather than in large mobs, to change paddocks frequently and to avoid confinement. Regular examination of rams for breeding soundness and early identification and culling of clinical epididymitis cases should further reduce the spread of infection.

Epididymitis is occasionally reported in 1- to 3-month-old ram lambs, associated with non-specific bacteraemias (Fig. 1.44).

Inguinal hernia

Large diffuse swellings of the scrotal sac above the testes are usually associated with unilateral herniation of the intestinal contents through the inguinal canal (Figs 1.45 and 1.46). In many cases the testes are small and difficult to differentiate by scrotal palpation from the inflamed herniated tissue.

Ultrasonography of the scrotal sac often reveals only clusters of hyperechoic lines and occasional reverberational echoes, associated with the presence of omental fat. The presence of air in herniated small intestine is occasionally identified by anechoic areas and complete attenuation of the ultrasonic beam.

Affected rams are permanently unsound for breeding purposes. In some cases the problem may be inherited, although firm evidence does not exist to support this. However, the probability of an hereditary basis for inguinal herniation should be considered before attempting the surgical repair of early uncomplicated cases.

Fig. 1.45 Herniation of the intestinal contents into the right scrotal sac. The right testis is atrophied and displaced to the distal scrotal sac.

Fig. 1.46 Postmortem appearance of an inguinal hernia, with thick, fibrinous adhesions between the herniated viscera and the tunica vaginalis.

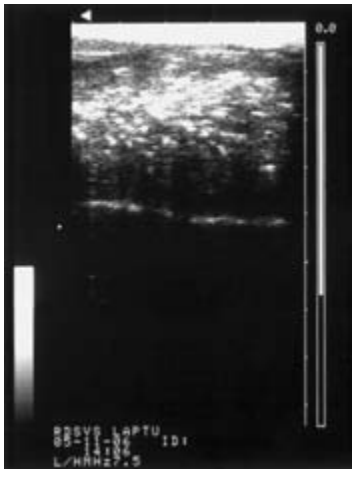


Fig. 1.47 Irregular hyperechoic dots within the testicular parenchyma.

Fig. 1.48 Bilateral testicular degeneration does not necessarily result in permanent breeding unsoundness.

Testicular degeneration and testicular hypoplasia

There are many possible causes of subnormal testicular size in rams, which cannot always be differentiated on the basis of physical ram soundness examination alone. However, irrespective of the cause, rams with small soft testes are likely to produce poor-quality semen. When the case history suggests satisfactory previous fertility, this would indicate acquired degeneration (atrophy) rather than hypoplasia. The diagnosis of testicular degeneration may also be supported by the ultrasonographic appearance of irregular hyperechoic regions within the testicular parenchyma (Fig. 1.47).

There are several possible causes of bilateral testicular degeneration including systemic disease, inflammation of the scrotum or its contents, extreme fatness, transport, physical exertion and high environmental temperatures. The preparation of rams for shows or sales may result in some degree of testicular degeneration, following which it is not unusual for few progeny to result from the first year's mating, but for normal breeding soundness to return in subsequent years. In most cases bilateral testicular degeneration only results in temporary unsoundness (Fig. 1.48). The

spermatogenic cycle in rams takes about 2 months, so if the underlying problem is corrected they may return to breeding soundness within about 3 months. However, unilateral testicular degeneration is commonly associated with inflammation in the contralateral testis and is usually permanent (Fig. 1.49).

Testicular hypoplasia can affect one or both testes and is associated with permanent unsoundness. The condition has been associated with cryptorchidism, and a variety of hereditary diseases such as XXY chromosome disorder (Klinefelter's syndrome). A tendency towards high carriage of the scrotal contents in certain breeds or family lines of rams may predispose to some degree of testicular hypoplasia and subfertility.

Cryptorchidism

Partial or total cryptorchidism is a common problem in rams. Most unilaterally cryptorchid rams remain fertile and cryptorchid animals of particular phenotypic merit are often used in pedigree breeding flocks. However, poor pregnancy rates may follow when cryptorchid rams are introduced to large numbers of ewes. Furthermore, the high incidence of cryptorchidism of 0.5 percent in some ram breeds suggests that there might be a hereditary basis for the problem and that it is unwise to retain affected animals.

Scrotal mange

Chorioptic mange is now widespread in UK ram flocks, causing pruritic, exudative dermatitis over the poll, above the coronary bands, around the accessory digits and over the pasterns of the hind limbs of ewes and rams, and on the scrotum of rams. Skin lesions are associated with a hypersensitivity reaction evoked by *Chorioptes bovis* mites. Scrotal mange is characterised by superficial, exudative, fissured and haemorrhagic lesions with some serous exudation and matting and staining of any overlying wool on the lower third of the scrotum (Fig. 1.50). Handling of the scrotum often initiates a nibble response (Fig. 1.51). Severe mange affecting more than one third of the scrotum can raise the temperature of the testes and is an important cause of seminal degeneration and reduced fertility.

Scrotal mange of sheep occurs in most countries around the world, but was not reported in the UK between 1970 and 2001. The disease was thought to have been eradicated as a result of compulsory dipping for the control of sheep scab. Systemic endectocides, used for the control of sheep scab, do not prevent scrotal mange, associated with the feeding behaviour of chorioptic mites, which feed on epidermal debris without ingesting body fluids, so the re-emergence of the disease was probably associated with the withdrawal of compulsory plunge dipping.

The husbandry system, breed of sheep or nutritional status of the rams appears to have little effect on the severity of scrotal mange lesions. Mite populations in ram lambs increase very slowly over winter and spring, so in untreated animals severe mange may not appear until the following autumn.

Thickening and fissuring of the scrotal skin, without evidence of scab formation or bleeding, is a common finding, particularly when rams have previously been winter housed, and is not necessarily indicative of scrotal mange. In some cases, these lesions might be associated with skin penetration by infective *Strongyloides papillosus* larvae. The diagnosis of scrotal mange can be confirmed by the identification of live mites in superficial skin scrapings from scrotal lesions, although mites are only present in small numbers and are often not found, even in animals with obvious

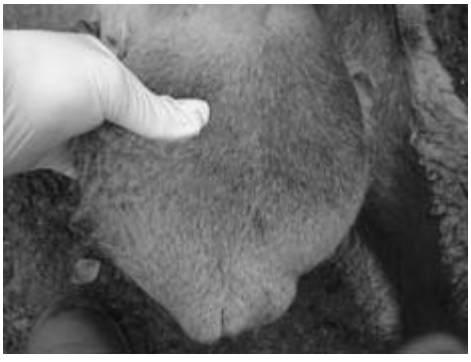


Fig. 1.49 Unilateral atrophy of the right testis (held below the finger and thumb) associated with inflammation of the left epididymis in a Suffolk shearling ram.

Fig. 1.50 Thickening, exudation and fissuring of the scrotal skin caused by chorioptic mange.

Fig. 1.51 A characteristic 'nibble response' to handling of the scrotum of a ram with severe chorioptic mange.

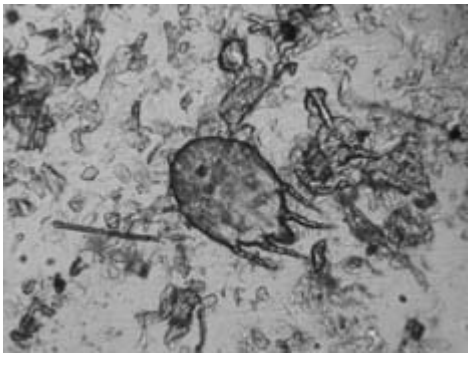


Fig. 1.52 A *Chorioptes bovis* mite on clear adhesive tape applied to the scrotal skin.

mange lesions. *C. bovis* mites are oval-shaped, 0.25 to 0.5 mm long (Fig. 1.52), with rounded mouthparts, short unjointed pedicels and bell-shaped suckers (Fig. 1.53).

Overseas, prevention and treatment of scrotal mange is usually achieved by the topical application of an organophosphate dip. In the UK, prevention currently depends on acaricide plunge dipping.

Traumatic injury to the scrotal contents

Most cases of orchitis in UK rams occur as a sequel to epididymitis, although focal necrotising orchitis has been reported associated with traumatic injury and extensive haematoma formation within the tunica vaginalis surrounding the testis (Fig. 1.54).

Affected rams generally show no signs of systemic illness, but may appear uncomfortable. The scrotal contents may be enlarged on one side, and firm nodular swellings may also be palpated within the testis. The problem is differentiated from inguinal herniation and epididymitis, which are characterised by swelling above the testis and within the epididymis respectively.

The diagnoses of haematoma formation within the tunica vaginalis and orchitis can be supported by the real-time ultrasonographic appearance of a matrix of

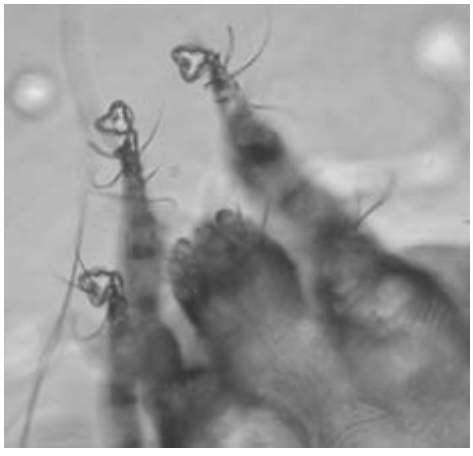


Fig. 1.53 Bell-shaped suckers and rounded mouth parts characterising a *C. bovis* mite.

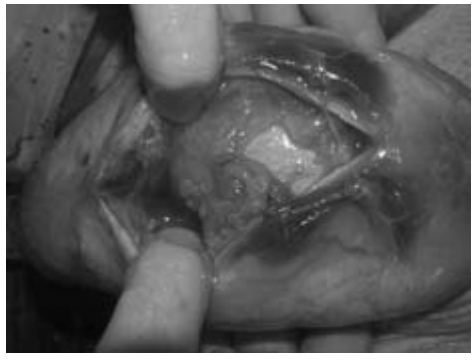


Fig. 1.54 An organising haematoma within the tunica vaginalis surrounding an atrophied testis.

hyperechoic lines and pockets of anechoic fluid, lying between a broad hyperechoic line corresponding to the tunica vaginalis and the testis. Focal hypoechoic necrotic regions may be seen within the testis close to its ventral pole.

On postmortem examination, areas of necrotising coagulative necrosis and fibrovascular reaction within the testis appear as firm, dark-red, focal lesions (Fig. 1.55). The histological appearance of the lesions is consistent with ischaemic damage and subsequent necrosis of seminiferous tubules, release of spermatozoan antigen, inflammation and fibrosis. Overseas, similar lesions are sometimes caused by brucellosis, which is characterised by intracellular acid-fast organisms seen in modified Ziehl-Neelsen stained sections.

Affected rams are generally permanently unsound, because the presence of extensive inflammatory lesions within the scrotal sac results in atrophy of the contralateral testis. However, fertility has been successfully preserved in valuable animals following surgical excision of the scrotal contents on the affected side using an open hemicastration method.

Testicular neoplasia

Sertoli cell tumours and seminomas are rarely diagnosed in rams. Most intratubular seminomas occur secondarily to testicular atrophy.

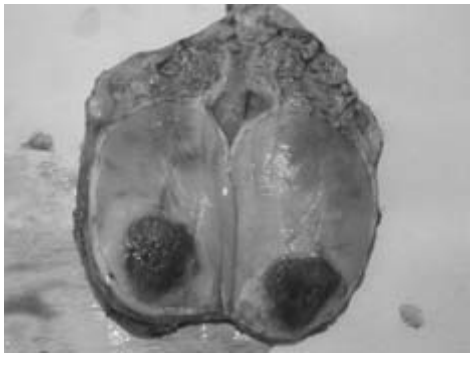


Fig. 1.55 A large focal necrotic lesion within a sectioned testis.

Segmental aplasia of the tail of the epididymis
Hypoplasia or aplasia of the tail of the epididymis has been identified as a very rare congenital abnormality of rams.

Spermatocoele
Obstruction to the epididymal tubules results in accumulation and leakage of spermatozoa into surrounding tissues. Foreign body reaction to the spermatozoa leads to the formulation of granulomas or spermatocoele. The condition has occasionally been reported in ram lambs following puberty, associated with imperfect formation of the epididymal tubules, but usually occurs following inflammation of the epididymis or vasectomy.

Scrotal abscesses
Shearing wounds of the scrotal skin may develop into abscesses under unhygienic or warm and humid conditions. Such abscesses seldom result in permanent unsoundness, but require radical treatment to avoid the possible involvement of deeper structures.

Inguinal lymph node abscessation
Discrete firm swellings high in the spermatic cord may be associated with abscessation of an inguinal lymph node. This is seldom a cause of poor breeding soundness in itself, but is important because it could be confused with epididymitis. The possibility of caseous lymphadenitis should also be considered.

Damage to the urethral process
Damage to the urethral process may result from shearing injury. The effect of such damage on ram breeding soundness is unknown.

The urethral process may also become necrotic and slough as a consequence of urolithiasis. In these cases necrosis often extends to involve the glans penis and results in permanent unsoundness.

Phimosis and other congenital or acquired defects of the penis
Inability to erect the penis fully may be congenital, or can occur as a sequel to balanoposthitis. Preputial prolapse has been reported as a rare cause of poor breeding soundness.

Urolithiasis (gravel)
Individual cases of urolithiasis are sometimes seen in intensively fed pedigree rams, often associated with preparation for shows or sales. Urolithiasis is an important disease of feeding wether lambs, potentially involving large numbers of animals with high mortality rates and subsequent ill thrift in recovered animals.

Most cases of urolithiasis in UK sheep are characterised by blockage of the urethra by a sludge of calcium, magnesium, ammonium and phosphate solutes, precipitated from urine. Most outbreaks are associated with high levels of concentrate feeding. Wether lambs are most susceptible because their urethra is less developed and narrower than in ram lambs, with blockage most commonly at the ischial arch or vermiform appendage of the penis.

The clinical signs in mildly affected animals are separation from the flock, anorexia, discomfort, frequent straining and dribbling of small amounts of urine.

Fig. 1.56 Swelling and the presence of blood-stained urine at the preputial orifice, with struvite crystals on the preputial hairs, indicative of urolithiasis.

Fig. 1.57 Swelling and necrosis of the vermiform appendage.

Fig. 1.58 Hydronephrosis caused by back pressure due to urethral obstruction. The renal pelvis is grossly enlarged and the ureter is distended. A normal, unsectioned kidney from a similar-aged sheep is shown for comparison.

Blood-stained urine and rice grain-like crystals are often visible at the preputial orifice and on the preputial hairs (Fig. 1.56). In more severe cases the lower abdomen and prepuce become swollen due to leakage of urine into subcutaneous tissues, which eventually slough. In some cases the abdomen becomes distended following leakage of urine from a distended bladder. Blockage at the vermiform appendage of the penis can sometimes be seen when the animal is closely examined (Fig. 1.57). Affected animals are azotaemic with greatly elevated urea and creatinine concentrations.

The diagnosis of urolithiasis can be supported by real-time ultrasonography to determine the size of the bladder, examine the right kidney and determine the presence of uroperitoneum. In cases of urolithiasis, the bladder is grossly enlarged and hydronephrosis of the right kidney may be identified by the presence of a grossly enlarged renal pelvis and much-reduced medulla.

Affected animals can be treated surgically in the short term, but their long-term prognosis is poor, associated with the rapid, irreversible development of hydronephrosis (Fig. 1.58). The most important consideration should therefore be the prevention of further cases. The ration should be checked for phosphate and magnesium levels and altered accordingly. Addition of calcium carbonate and increasing the roughage in the diet may help to correct a calcium:phosphate imbalance. Ewe rations frequently contain high levels of magnesium, so should never be fed to male sheep.

Acidification of the urine reduces the likelihood of sludge precipitation. This can be achieved by feed withdrawal for 24 hours and daily dosing with 1 g ammonium chloride in solution. In the longer term, addition of ammonium chloride to the diet can be useful.

Concentrates should be introduced gradually and a source of roughage should be provided to reduce the risk of acidosis. It is also important to ensure that animals have unrestricted access to fresh water at all times and that the mineral content of the ration is correctly formulated to avoid the risk of urolithiasis.

Overseas, flock outbreaks of urolithiasis are associated with siliceous and oxalate calculi, although these occur rarely in the UK. Deposits of desquamated cells and accessory gland secretions referred to as 'clover stones' can cause urethral obstruction in both male and female lambs grazed on subterranean clover. The deposits are probably formed under the influence of oestrogenic substances from the crop.

Balanoposthitis (pizzle rot)

Balanoposthitis is most important as a production-limiting disease of wethers, but can also be an important cause of poor breeding soundness in rams.

Early and mild cases of balanoposthitis are characterised by small ulcers and stained wool around the external orifice of the prepuce (posthitis) (Fig. 1.59). Scabs develop over the ulcers, which become necrotic and slough. The wool surrounding the prepuce becomes stained, smelly and sometimes flystruck. In severe cases the internal preputial membranes (balanitis) are also involved. Foul-smelling exudate accumulates within the prepuce, which may become blocked by the presence of scabs at its opening. Animals may have difficulty urinating and the prepuce and ventral abdomen may become swollen due to the accumulation of urine and exudate. Severely affected animals may die from kidney failure, although most cases in rams involve only external lesions and the severe disease is usually limited to wethers.

The disease is caused by *Corynebacterium renale* which grows in alkaline urine produced by animals on a protein-rich diet. *C. renale* produces ammonia from urinary urea, which burns the prepuce and penis. High levels of urea are present in the urine of animals which are fed on protein-rich diets such as improved pastures or barley and maize gluten concentrate rations. Balanoposthitis is most common in wethers because their penis is less developed than in rams, so they urinate into the prepuce.

Mildly affected animals usually respond to topical antiseptic and parenteral antibiotic treatment within a few days. Affected animals should be isolated to avoid further contamination of the environment by *C. renale*. The wool around the prepuce should be clipped to remove bacteria-contaminated wool and to enable wound irrigation and topical treatment with antiseptic ointment. Where possible the amount of protein in the diet should be restricted and free access to fresh water provided. Oral administration of ammonium chloride (1 g three times daily) with water may help by acidifying the urine.

Severely affected animals may require surgical drainage of the prepuce and the inclusion of ammonium chloride in the drinking water to promote diuresis. The longer-term prognosis is poor in these cases.

Occasional outbreaks of balanitis in rams have been associated with parapox virus (Fig. 1.60). Ulceration of the glans penis of unknown aetiology has also been reported.

Fig. 1.59 Posthitis with ulceration and necrosis of the prepuce.

Fig. 1.60 Scab formation and bleeding of the prepuce of a Charollais ram caused by orf infection.

Artificial insemination

Artificial insemination offers some advantages over natural service, particularly in pedigree flocks:

- Enables sharing of rams.
- Allows sires with desirable genotypes to be used across several flocks.
- Enables introduction of new genotypes to closed flocks without the risk or introduction of disease.
- Overcomes the need for a high ram-to-ewe ratio following synchronisation of oestrus.
- Enables comparison and ranking of genotypes through sire reference programmes.

Sheep have a tortuous cervical canal which prevents the vaginal route of deposition of semen into the uterus, as used in cattle. About 400 to 500 million fresh live spermatozoa must, therefore be deposited at the cervical os into its first fold. The process is usually performed with ewes standing in a narrow race with their pelvic limbs raised over a straw bale or equivalent hurdle. Target conception rates to cervical artificial insemination using fresh semen are about 70 percent. Frozen semen gives unacceptably low conception rates when inseminated cervically. Attempts have been made to develop transcervical artificial insemination techniques in sheep, but results have been poor and associated complications of localised and generalised peritonitis are unacceptable.

Conception rates using fresh semen can be increased by laparoscopic deposition of semen directly into the uterus. Laparoscopic intrauterine insemination also enables the use of much smaller numbers of fresh spermatozoa or the use of frozen semen. Ewes are first sedated, usually by intramuscular injection of acepromazine, and restrained in a purpose-built cradle with their pelvic limbs uppermost. Next, 5 to 10 ml of 2 percent lignocaine is infiltrated in two sites on the ventral abdomen, before surgical preparation and making two small stab incisions. The peritoneal cavity is then insufflated with carbon dioxide, a rigid endoscope inserted into the abdomen to visualise the uterus, and semen deposited into each horn through the second incision using a needle and syringe. One operator can inseminate up to 200 ewes per ram per day using this method. Target conception rates for laparoscopic intrauterine

insemination using fresh and frozen semen in terminal sire breeds are 75 percent and 65 percent respectively.

Sheep are synchronised for artificial insemination using progestogen sponges and injected with PMSG at sponge withdrawal to induce ovulation reliably. Cervical and laparoscopic artificial insemination are usually performed at a fixed time, 56 hours after sponge removal.

Semen is usually collected using a 'teaser' ewe and artificial vagina. An electro-ejaculator can be used, but the quality of the semen collected is lower. Semen quality is assessed on the basis of motility, concentration and spermatozoa morphology. Fresh semen is commonly extended to approximately 1000 million sperm/ml using skimmed milk or egg yolk diluents and is used within 6 hours. Alternatively, the semen can be frozen, enabling:

- insemination of larger numbers of ewes
- international movement of genetic material
- storage of semen from rams of high genetic merit for later use.

Frozen semen cannot be used for cervical artificial insemination due to poor viability and impairment of spermatozoa transport through the cervix. However, intrauterine artificial insemination bypasses the cervix and enables the use of frozen semen.

Multiple ovulation and embryo transfer

Reasons for using embryo transfer include:

- increasing the rate of within-flock genetic improvement
- eradication of diseases such as maedi-visna from a flock, without losing desirable genotypes
- import and export of genetic material
- storage of embryos for future transfer.

Progestogen sponges are first used to control the oestrus cycle. Superovulation can then be induced using follicle stimulating hormone (FSH) injections of between days 11 and 13, towards the end of the subsequent normal oestrous cycle. Donor ewes are laparoscopically inseminated 40 to 48 hours after sponge removal to achieve optimal embryo recovery rates.

Embryos are usually recovered as 24- to 32-cell morulae at day 5, or late morulae/early blastocysts by day 6. Embryos can be recovered laproscopically under general anaesthesia, using a similar technique to that used for artificial insemination, or by laparotomy, involving exteriorisation of the uterus, ovaries and oviducts by a ventral midline incision. The oviducts are cannulated and the uterus flushed with sterile Dulbecco's phosphate-buffered saline to recover the embryos. The embryos are evaluated by microscopy and graded according to appearance, stage of development and abnormalities. Poor-quality embryos are usually not transferred. Recovery rates are usually between 6 and 11 embryos per superovulated ewe. Embryos can be stored frozen in liquid nitrogen.

Synchronisation of oestrus behaviour in donor and recipient ewes to within 12 hours of each other is usually achieved using progestogen sponges. One embryo is usually laparoscopically transferred to each uterine horn of the recipient, with successful transfer rates between 60 and 80 percent.

The success of artificial insemination and embryo transfer programmes is determined by:

- appropriate selection of ewes
- correct nutrition and body condition score
- minimising stress by performing routine management procedures at least 1 month beforehand and by avoiding husbandry and disease stress for 1 month afterwards
- accurate and effective manipulation of the breeding cycle.

Embryonic development

There is evidence from studies using multiple ovulation and embryo transfer technologies that between 20 percent and 40 percent of embryos die at or around the time of implantation (day 19). Rejection of abnormal karyotypes only accounts for 5 to 10 percent of embryos lost. Embryonic loss is highest in ewes with high ovulation rates, indicating that, at least in prolific flocks, it might be possible to reduce the overall lamb mortality between conception and weaning, through attention to stock management during the mating and early pregnancy period. In spontaneously ovulating, less-prolific commercial ewe flocks not employing modern embryo transfer technology, the importance of early embryonic loss cannot be easily quantified.

Total embryo loss at or around implantation results in a delayed return to oestrus following slow regression of the corpus luteum and, in the case of flocks with a restricted mating period, a high barren rate. Where there is partial embryo loss from a multiple litter, surviving embryos are restricted to a smaller number of placentomes than would have been the case had the resultant litter size been established before implantation. Subsequent lamb birthweights are therefore reduced.

Severe heat stress of ewes during early pregnancy can significantly reduce embryo survival. Under normal UK conditions, while environmental heat stress is unlikely, gathering and driving of the flock during the immediate post-mating period may cause occasional, but avoidable, early embryonic deaths. For other stressors, such as persistent rain, transport, shower and plunge dipping, the duration of the stress is important, prolonged exposure having a significant effect on embryo survival.

Inflammatory disease during or shortly after the mating period can give rise to a marked increase in barren rates, in particular subacute liver fluke and sheep scab outbreaks.

Embryo survival is probably lower at each end of the breeding season, although the data are difficult to interpret because of the well-documented differences in ovulation rate at these times. Embryo survival is poorer in ewe lambs and gimmers than in mixed-aged ewes. Pre-mating shearing has been shown to enhance the reproductive performance of ewe lambs, but it is not known if this is due to increased ovulation rate or improved embryo survival. In other circumstances, such as the prolonged stress of subsequent exposure to cold, the effects of pre-mating shearing may even be damaging.

Trace element deficiencies are frequently blamed for poor embryo survival. Although correction of copper, cobalt, selenium, iodine, manganese and iron deficiencies have all been shown to improve litter size, only selenium and iodine have been shown to have any direct effect on embryo survival. Selenium-responsive infertility is a result of embryonic mortality 3 to 4 weeks after conception, while subclinical iodine deficiency appears to have the greatest effect on ewe reproductive performance during the first third of pregnancy.

Example

The results of ultrasound scanning for pregnancy and foetal numbers on 27 January for a lowground Mule ewe flock in the south-east of Scotland are summarised below:

- 225 Greyface gimmers
  - 12 barren (5.3%)
  - 80 singles
  - 131 twins
  - 2 triplets (scanning percentage 155%)
- 481 Greyface ewes
  - 23 barren (4.8%)
  - 69 singles
  - 304 twins
  - 85 triplets (scanning percentage 194%)

The results for the ewes were comparable with previous years. These ewes had been kept on a different part of the farm from the gimmers during the previous summer, but were mated in fields adjacent to those used for the gimmers. Most of the barren ewes had been managed in one subgroup during the mating period, with two rams which were subsequently shown to be unsound.

The barren rate in the gimmers of 5.3 percent was abnormally high and the twinning rate disappointing. Under similar management during the previous years the gimmer flock had achieved a barren rate of about 1.8 percent and an overall scanning percentage of 181 percent. The gimmers had been run for 35 days with six rams, which had been used during previous years and were checked for breeding soundness before introduction. Ten of the 12 barren gimmers were raddle marked twice, while the other two were not marked at all (Fig. 1.61).

The fact that 10 of the 12 barren ewes had been marked twice by sound rams suggested conception failure, or failure to maintain very early pregnancy. The gimmers were all in body condition score 3.5 to 4.0 (on a scale of 1 to 5) at the time of ram introduction, but about 10 percent had lost 1.5 to 2 units of body condition score by the time of scanning.

Blood sampling of seven barren and three pregnant gimmers revealed low albumin concentrations, elevated globulin concentrations and raised concentrations of the liver-specific enzyme glutamate dehydrogenase (GLDH) (Table 1.1).

These findings were consistent with recent subacute liver fluke infection. The diagnosis of subacute liver fluke during the mating period was further supported by the identification of large numbers of fluke eggs in faecal samples, collected from 7 of the 10 gimmers about 12 weeks after the time of ram introduction.

Fig. 1.61 Records of keel mark data are important for the investigation of high barren rates.

Table 1.1 Serum protein and liver enzyme concentrations of 10 gimmers.

Scanning	Albumin (g/L)	Globulin (g/L)	GLDH (iu/L)
Barren	21.7	30.4	67
Barren	28.4	39.7	163
Barren	25.2	31.0	309
Barren	28.8	32.6	12
Barren	36.9	35.2	48
Barren	29.5	35.2	122
Barren	33.4	30.4	32
Single	16.1	61.9	17
Twins	24.9	31.2	129
Twins	27.8	35.6	32
Reference values	(30–36)	(32–38)	(2–10)

Any diagnosis of selenium-responsive poor reproductive performance is problematic. While blood glutathione peroxidase (GSHPx) concentrations generally provide a reliable indication of the animals' longer-term selenium status, they do not reflect recent dietary changes, which may be significant in cases of high barren rates. A diagnosis of selenium-responsive poor reproductive performance is seldom made on the basis of low blood GSHPx concentrations, but is suspected whenever selenium concentrations of less than 500 nmol/L are identified in serum or plasma pooled from five ewes. Serum and plasma selenium concentrations are dependent on the amount of selenium in the diet, so provide an indication of the animals' current daily selenium intake. Unfortunately laboratory determination of blood selenium concentrations is expensive and the reference values for UK sheep are poorly defined.

Some varieties of subterranean and red clovers contain high concentrations of phyto-oestrogen precursors, which are known to affect adversely the lambing percentage. It is unclear if this effect is on ovulation rate, embryo survival, or both. Prolonged feeding of brassica crops such as rape and kale before and during the mating period has also been demonstrated to have detrimental effects on embryo survival, although the mechanism is again unclear. Overfeeding during early pregnancy can influence pregnancy rates by suppression of circulating progesterone concentrations, while severe undernutrition during the mating period has also been shown to cause embryo mortality in gimmers.

Infection with *T. gondii* or border disease virus during early pregnancy may result in embryonic death, although such losses are not easily quantified, compared with the more obvious consequences of infection during later pregnancy.

High barren rates associated with observations of resorbing foetuses on ultrasound scanning are usually attributed to toxoplasmosis. However, these findings sometimes occur in vaccinated ewes, or in animals with low latex agglutination test titres for *T. gondii*. There is growing evidence to suggest that these problems might occasionally be associated with *Neospora caninum* infection. Non-suppurative foci of inflammation, similar to lesions identified in cattle abortions caused by *N. caninum*, have been identified in the placentomes of aborted ewes.

N. caninum is a protozoal parasite closely related to *T. gondii*, which is recognised as an important cause of abortion in UK dairy cattle. The definitive host for the *N. caninum* has been shown to be dogs, which shed oocysts. Cattle and horses are recognised intermediate hosts, within which the parasite has a rapidly multiplying

Example

A protracted lambing period and high barren rate were reported in an upland flock of about 400 Scottish Halfbred ewes. Similar problems had occurred during previous years. The ewes had been mated in fields adjacent to disused lime kilns. About 14 sound Suffolk rams were introduced during mid October and removed during March. Most of the ewes were keel marked during the first 17 days of the mating period. Few of these were marked again during the next 17 days, but several were observed to be mated in late December and January, after moving to turnips (Fig. 1.62).

Ultrasound scanning during May, 5 weeks after the start of lambing, showed that 17 ewes would be late lambers and 18 were barren. Had the rams been removed after 34 days, the barren rate would have been 9 percent.

The fact that most of the ewes were keel marked during the first 17 days of the mating period showed that the ewes had been cycling at the time of ram introduction. The rams had all been checked for breeding soundness before introduction, so the possibility of fertilisation failure was unlikely. Observations of marked ewes showing oestrus after moving onto turnips at the end of December were consistent with failure of pregnancy at the time of implantation, or early foetal death.

The ewes were all vaccinated against toxoplasmosis and no signs of border disease had been seen in the flock. There was no evidence to support a diagnosis of subacute liver fluke. However, the history of grazing on fields adjacent to old lime kilns, possibly with raised soil pH values, during the mating period raised the possibility of selenium deficiency.

Confirmation of a diagnosis can prove to be problematic in situations such as this, where the problem is only identified several months after the event. While the disease history often points towards a particular cause, it is often necessary to implement potential preventive measures and monitor the flock closely over the following mating season.

All of the ewes in the flock were supplemented with selenium before each subsequent mating period and the barren rates have consistently been reduced to 2 percent, supporting the diagnosis of selenium deficiency as the cause of poor reproductive performance.

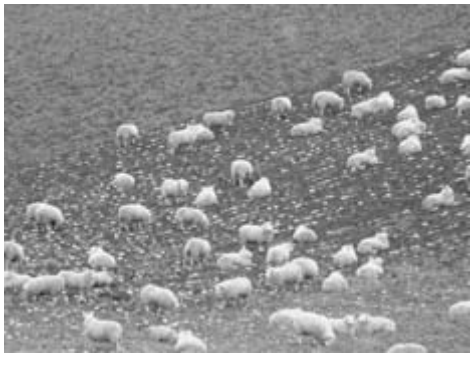


Fig. 1.62 Several ewes showed signs of oestrus behaviour after they were moved onto turnips during January.

(tachyzoite) stage in cells of all tissues, and a slowly multiplying (bradyzoite) stage in tissue cysts in brain, muscle and placental tissue. Tissue cysts can cause significant pathology because they are enclosed in a cyst wall and not recognised by the host's immune system. Cattle become infected either by eating sporulated oocysts, or congenitally from their dams.

While there is little doubt that sheep can be infected with sporulated *N. caninum* oocysts from the environment, nothing is known about the dose required to cause disease. Abortions and the birth of congenitally infected lambs have been demonstrated

Example

The results of ultrasound scanning for pregnancy and foetal numbers on 7 February for an upland flock in the south-east of Scotland are summarised below:

- 148 Mule and Texel cross ewe lambs
  - 50 barren (34%) (breeds equally represented)
  - 76 singles
  - 22 twins
- 547 Mule and Texel cross ewes
  - 22 barren (4.0%)
  - 117 singles
  - 340 twins
  - 68 triplets

The ewe lambs had been run with to two vasectomised rams between 26 October and 1 November (6 days). Three sound adult rams had been introduced to the ewe lambs on 5 November and removed on 1 December (25 days). All of the ewe lambs had exceeded 43 kg at the time of ram introduction. The scanning results for the ewe lambs were disappointing. Most of the barren ewe lambs had only been keel marked once. A total of 16 ewe lambs were identified by the scanner as being pregnant, but with evidence of foetal resorption. A further three ewe lambs were scanned as pregnant but showed signs of blood-tinged vaginal discharge.

The scanning results for the ewes were marginally better than during previous years. Ten rams had been introduced to the ewes on 5 November and removed on 9 December (43 days). The whole flock had been supplemented with selenium and managed to control liver fluke before the mating period.

All of the ewes were vaccinated against toxoplasmosis and chlamydial abortion, but the ewe lambs were only vaccinated against chlamydial abortion, toxoplasmosis vaccine having been unavailable before the mating period. A young cat had been seen hunting on the farm during the mating period.

There were several potential reasons for the high barren rate in the ewe lambs. While most Mule and Texel cross ewe lambs weighing more than 43 kg will show signs of oestrus behaviour at the time of ram introduction in November, they will not achieve comparable pregnancy rates to adult ewes of the same breed.

Correct use of vasectomised rams can advance the breeding season of ewe lambs and thus improve pregnancy rates. However, because oestrus activity is also synchronised following the use of teaser rams, the ratio of one sound ram to 50 ewe lambs that was used may have been insufficient. However, it is unlikely that this was the only cause of the high barren rate, because few of the barren ewe lambs had been raddle marked after 17 days.

Ultrasonographic evidence of foetal resorption in 16 percent of the pregnant ewe lambs indicates early foetal death. The known causes of this are toxoplasmosis, and border disease, although the latter is uncommon, and usually accompanied by other clinical signs within the flock. The fact that the ewe lambs were not vaccinated against toxoplasmosis and the presence of a young cat were also consistent with the diagnosis of toxoplasmosis.

Serum samples were collected from six barren and six pregnant ewe lambs with evidence of resorbing foetuses. The latex agglutination test titres of the barren ewe lambs for *Toxoplasma gondii* were: negative; negative; negative; 1/16; 1/256; and 1/2048. The titres of the pregnant ewe lambs were: negative; negative; negative; 1/16; 1/64; and 1/64. The involvement of ewe lambs and very high titres of some animals strongly support a diagnosis of toxoplasmosis as a cause of foetal resorption and the high barren rate. However, toxoplasma serology is difficult to interpret. Latex agglutination test titres greater than 1/64 provide evidence of infection, but provide little indication of when exposure occurred because toxoplasma antibodies persist for several years and frequently remain high into the following breeding season. Following exposure, it may take several weeks before animals seroconvert, so negative results do not necessarily rule out the diagnosis of toxoplasmosis.

following experimental infection during the early stages of pregnancy, but it is not known if comparable levels of challenge can occur under field conditions.

Careful attention to stock management during the mating period, in particular through the avoidance of prolonged stress due to gathering for shearing or dipping, may limit losses to early embryonic death. However, current understanding of the use of management practices to increase embryo survival is limited, and the net contribution of such practices to the lambing percentage is unknown. In a significant proportion of ewes which are identified as barren at the time of ultrasound scanning or at lambing, the problem is associated with failure to maintain pregnancy rather than infertility. However, these animals should be culled, because many will fail to maintain pregnancy in subsequent years.

Investigation of high barren rates

For most lowground and upland flocks, the following pregnancy rates should be achievable:

- All ewes mated during the first 17 days of the mating period.
- Less than 8 percent of ewes returning to oestrus during the second 17 days of the mating period.
- A barren rate of less than 2 percent after two oestrus cycles.
- Less than 2 per cent abortions.

Some variation from these targets is normal. Some large lowground flocks achieve barren rates of only 1 percent, but investigation is usually only instigated when they exceed 4 percent. Targets for twinning rates vary greatly, depending on the sheep breed, farm location and sheep production system.

The list of possible causes of high barren rates includes:

- ewes not cycling at the time of ram introduction due to:
  - inappropriate breed of sheep
  - a high proportion of ewe lambs or gimmers in the flock

  - the latitude and altitude of the farm
  - prolonged stress associated with unskilled handling or persistent rain
- fertilisation failure due to:
  - unsound rams
  - an inappropriate ram:ewe ratio
  - unsuitable mating paddocks
- failure to conceive or maintain early pregnancy due to:
  - severe undernutrition
  - low protein status associated with diseases such as sheep scab and subacute liver fluke
  - persistent stress
  - selenium deficiency
  - iodine deficiency
  - toxoplasmosis
  - border disease.

The investigation of these potential causes therefore includes: consideration of the sheep breed, dates of ram introduction and farming system; examination of rams for breeding soundness and consideration of ram management before and during the mating period; and monitoring of the ewes' protein, selenium and iodine status and investigation of the possibilities of toxoplasmosis or border disease.

Analysis of keel or raddle mark data can be useful, to determine the proportion of the flock displaying oestrus behaviour during the first 17 days of the mating period, the proportion of the flock not mated at the end of a 34-day mating period and the proportion of the flock mated twice during this period (Fig. 1.63). For example, ewes may be marked twice if the problem is associated with the use of unsound rams or with conception failure, while diseases such as selenium and iodine deficiency, which result in pregnancy failure at the stage of implantation, or toxoplasmosis and border disease, which are associated with early foetal death and abortion are characterised by a high proportion of barren ewes being marked only once.

It is not always possible to determine the reasons for high barren rates when they are first identified at the time of ultrasound scanning for pregnancy or after lambing. In these cases it is necessary to investigate potential problems during the following autumn, before and during the mating period. Such investigation may include the examination of rams for breeding soundness, investigation of the ewes' selenium status and the use of ram harnesses or keel to enable careful recording of the mating

Fig. 1.63 Analysis of keel marks is an important part of the investigation of poor reproductive performance.

pattern. In large flocks, detailed information collected from a small group of sheep is often adequate.

Misalliance

It is sometimes desirable to terminate pregnancy following inappropriate matings. In most cases, pregnancy can be prevented or terminated by intramuscular injection of 250 µg cloprostenol, either between days 5 and 12 after mating or between days 21 and 45 after mating.

Mid to late pregnancy (abortion)

The normal gestation length of ewes is about 147 days (range 142 to 150 days). Following implantation, the embryo or foetus is reasonably resilient to the effects of nutritional, management or environmental stress.

During early pregnancy, the body condition of ewes which were at or below target scores for mating of 3 to 3.5 (on a scale of 1 to 5) should be maintained. Ewes with body condition scores greater than 4.0 (on a scale of 1 to 5) can afford to lose up to 9 percent of their bodyweight. Between days 30 and 90 of pregnancy, the placenta develops rapidly. During this period, ewes which were mated in target body condition can afford to lose up to 9 percent of their bodyweight (0.75 units of body condition score). Overfeeding during this period may have an adverse effect on placental development as well as contributing to excessive body condition at parturition with subsequent dystocia or possibly vaginal prolapse problems. Underfeeding results in ewes entering the final 6 weeks of pregnancy, and ultimately lambing, in poor body condition.

Nutritional stress during mid pregnancy only affects the lambing percentage when it is severe and of at least 10 days duration. These conditions may result in retarded placental development and the subsequent birth of twin lambs with disproportionate weights (for example 5.5 kg and 3.5 kg). This situation may partly be explained by the fact that while three embryos may have been implanted, followed by competition within the uterus for caruncles and early foetal development; one may have subsequently died and been resorbed, leaving a surviving twin foetus in the ipsilateral uterine horn with the placentomal attachment and placental nutrition of a triplet foetus. While the placetomes can increase in size and blood flow, these compensatory mechanisms often fail to overcome their reduced number.

Generally, losses from day 30 to parturition are low when compared with losses during early pregnancy or deaths occurring soon after birth. The principal causes of foetal death during the mid to late pregnancy period are placental and foetal exposure to infectious agents (abortion).

In closely monitored lowland flocks, late abortions are apparent, and it is estimated that the average incidence of abortion in such flocks in the UK is between 2 and 3 percent. However, abortion storms causing up to 70 percent losses occasionally occur in individual naïve flocks. In more extensively farmed hill flocks, abortions may be less apparent and are difficult to quantify, although whenever ewes in these flocks are extensively set stocked during mid to late pregnancy, abortion is unlikely to be a serious problem. The two most important annual causes of abortion in UK flocks are

chlamydial abortion and toxoplasmosis accounting for about 40 and 35 percent respectively of all diagnosed problems. Sporadic abortion outbreaks associated with other pathogens, in particular *Campylobacter fetus* subspecies *fetus* and *Salmonella* spp., or physical stressors are sometimes encountered. In some cases, mixed infections are present in the same flock.

General management of abortion in ewes

Appropriate flock preventive measures must always be taken before the actual cause of abortion is confirmed. These should include:

- isolation and marking of aborted ewes
- disposal of any products of abortion
- implementation of strict biosecurity to avoid further spread of infection
- strict hygienic precautions to minimise the zoonotic risk
- whenever possible a reduction in the stocking density of pregnant lambing ewes.

Chlamydial abortion

Chlamydial abortion (formerly referred to as enzootic abortion of ewes) costs the UK sheep industry an estimated £15 million per year. Chlamydial abortion is present in many other countries worldwide, but does not occur in Australia or New Zealand. Chlamydial abortion is caused by the bacterium *Chlamydophila abortus* (formerly known as ruminant abortion subtypes of *Chlamydia psittaci*).

Clinical signs

Chlamydial abortions typically occur during the final 3 weeks of pregnancy. Ewes appear healthy until about 3 days before aborting, when they may stop eating and a red vaginal discharge is sometimes noted. Aborted lambs may be born freshly dead or alive, often with abdominal distension caused by accumulation of fluid within body cavities. Live-born lambs are often weak and unable to suckle, so are susceptible to starvation, hypothermia and neonatal diseases. Apparently normal live lambs, weak lambs and dead lambs may be born in the same litter. Varying amounts of discoloured uterine discharge are present for several days after abortion (Fig. 1.64), but the ewes are otherwise healthy, only rarely developing retained placentae and consequent metritis.

Pathogenesis

C. abortus has an unusual life cycle, which involves alternation between an extracellular infectious stage (elementary bodies) and an obligatory intracellular replicative phase, which results in disease, but is not infectious. Infected ewes shed vast numbers of chlamydiae in diseased placentae and vaginal discharges. Other ewes become infected by ingestion or inhalation of *C. abortus* elementary bodies from an environment mostly contaminated by infected placentae or vaginal discharges. Following infection, *C. abortus* cannot be detected in the ewe until about the 90th day of the next pregnancy, when the organism invades the placenta. The outcome is then dependent on the ewe's immune status:

Fig. 1.64 Hollow flanks and blood-tinged uterine discharge following chlamydial abortion. This ewe should have been removed from other lambing ewes immediately to reduce spread of infection.

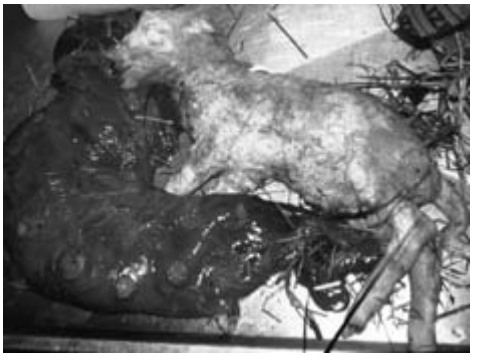


Fig. 1.65 Inflammation of the intercotyledonary placenta. (Healthy intercotyledonary placentae are translucent.)

- Ewes which have previously aborted due to chlamydial abortion are immune to further chlamydial abortions, but may continue to shed *C. abortus* in vaginal discharges following subsequent lambings.
- Infection of susceptible ewes during the first half of pregnancy can result in abortion during the final 3 weeks of the same pregnancy. In practice this situation is uncommon, other than in flocks with both early and late lambing ewes.
- Infection of susceptible ewes during late pregnancy, or when not pregnant, usually results in abortion during the final 3 weeks of the subsequent pregnancy.
- Surviving ewe lambs born to aborted ewes, and some ewe lambs born to ewes which aborted during a previous season, become infected through licking or inhaling placental fluids/vaginal discharges on their coats and abort during their first pregnancy.

Rams and wethers are probably unimportant in the spread of infection between flocks. However abortion and placentitis have been shown experimentally in naïve ewes infected with *C. abortus* before mating. A suggestion has also been made that infected ewes may shed the organism during oestrus, raising the possibility that transmission of infection might occur during the breeding season, through natural mating or artificial insemination.

Diagnosis

Severe placentitis is seen as variable discolouration and necrosis of cotyledons with oedema and rough thickening of adjacent intercotyledonary tissue (Fig. 1.65). The placenta is sometimes covered by a dirty yellow-pink exudate. The foetal liver is frequently congested and ruptured during parturition. Aborted foetuses may have inflammatory foci in the liver, lungs and brain.

The clinical and gross pathological signs may be indicative of chlamydial abortion, but are not specific. The diagnosis is confirmed by demonstration of large numbers of chlamydial elementary bodies in Ziehl-Neelsen stained smears of placental tissue. In the absence of aborted material, paired serology can sometimes provide useful support for a diagnosis.

Disease control

Chlamydial abortion is usually introduced with purchased sheep. Orphan lambs and female breeding replacements are the most common sources. Occasionally foxes, seagulls and other vectors are blamed for carrying aborted placentae or infected fomites onto neighbouring farms. Thus, the options for control of chlamydial abortion in flocks which are free from the disease are:

- maintain a closed ewe flock
- never buy in orphaned lambs
- only purchase replacement ewe hoggs and gimmers from accredited chlamydial abortion-free sources
- isolate all aborted ewes
- investigate abortions if they occur in more than 1 percent of the flock or multiple cases occur over a short period of time.

The main sources of infection are aborted foetuses and placentae. Ewes may have an infective vaginal discharge for about 3 weeks following abortion and aborted live-born lambs harbour infection on their coats for several days. Other sheep become infected when they lick or inhale any of the above. Thus, control of chlamydial abortion in infected flocks involves the following actions:

- immediately isolate all aborted ewes and their surviving lambs
- remove products of abortion and contaminated bedding
- whole-flock antibiotic treatment
- vaccination.

Before the introduction of effective vaccines, 20 mg/kg of long-acting oxytetracycline was commonly administered to pregnant ewes 3 to 6 weeks before lambing, with the aim of increasing the proportion of viable lambs born. There is little strong evidence to support this strategy.

The current chlamydial abortion vaccines are based on either an attenuated ovine abortion strain of *C. abortus*, or on an inactivated egg-grown preparation. In the field these vaccines have been shown to be very effective for the prevention of chlamydial abortion in previously unexposed sheep. Experimental data also indicates that the inactivated vaccine may reduce the risk of abortion in infected ewes in the face of an outbreak. Ideally all female breeding sheep are vaccinated once in the first year and in subsequent years breeding replacement ewe lambs or gimmers are vaccinated once at any time up to 4 weeks before mating. The inactivated vaccine can also be given from 4 weeks after ram removal if necessary.

Elimination of chlamydial abortion from an infected flock is very difficult; however, the level of infection can be significantly reduced using a combination of purchasing accredited-free replacement ewes, running replacements as a separate flock at lambing, hygiene at lambing and introducing a vaccination programme.

The Premium Health Scheme

The Premium Health Scheme is a national, voluntary, farmer-led programme with the aims of preventing chlamydial abortion in specific flocks and of supplying buyers with breeding replacement ewes free of chlamydial abortion. The scheme has about

2000 members and represents about 7 percent of the UK breeding flock. All tests, including abortion examinations, are paid for by an annual membership fee which, for most flocks, is significantly less than the cost of vaccination. Flocks are accredited free from chlamydial abortion on the basis of:

- submission for 2 years or more of all aborted material found and failure to identify *C. abortus* in stained placental smears
- blood sampling and negative serology of all known aborted ewes
- blood sampling and negative serology of barren ewes (up to a maximum of 5 percent of hill flocks)
- blood sampling within 3 months of the end of lambing and negative serology of a significant proportion of lactating ewes.

Disease control by monitoring works best when a self-contained area is free of disease. Designated chlamydial abortion-free areas and gimmering flocks are included on the basis of a commonsense approach to the epidemiology of chlamydial abortion.

A reliable diagnostic test is a prerequisite for any scheme which provides accreditation of freedom from disease. However, no serological test accurately detects exposure to or freedom from disease with 100 percent reliability. Interpretation of serology therefore requires a clear understanding of the disease epidemiology, diagnostic tests and the concepts of sensitivity, specificity and predictive values of the tests used. Interpretation errors can be costly, for example a single positive complement fixation test (CFT) result in a flock with no history of abortion could indicate the first case of disease, or could be a false positive result.

Premium Health Scheme blood samples are examined by a CFT, whereby the test sera are added to mixtures of chlamydial antigen, complement and sensitised red blood cells. In the absence of serum antibodies, complement binds to the sensitised red blood cells and causes haemolysis. If antibody is present in the serum, complement binds to the antibody–antigen complex, rather than sensitised red blood cells (Fig. 1.66).

Sera are tested at different dilutions of antigen (4, 8, 16, 32, 64, 128, etc. fold). The reaction at each dilution is read on a scale of 0 (no reaction) to 4 (complete reaction). Thus a titre of 2/32 means a 50 percent reaction in the 4th dilution, while 4/128 means total reaction at the 6th dilution. The interpretation depends on the specific test.

Sensitivity refers to the ability of a test to detect infected or previously exposed animals, while specificity refers to the ability of a test not to react if the animal is not infected or previously exposed. In general, the more specific a test, the less sensitive it becomes, and vice versa. Thus, tests are used to achieve the best compromise between sensitivity and specificity.

In the first year of joining the Premium Health Scheme sufficient lactating ewes are sampled to ensure a 99 percent probability of detecting seropositive animals if the prevalence of the disease is 5 percent. In subsequent years, the sample size ensures a 90 percent probability of detecting seropositive animals at the same disease prevalence. The CFT for chlamydial abortion at the cut-off titre of 1/32 is relatively insensitive, and may only detect 80 percent of infected animals. It would therefore be possible that infected ewes might not be identified in flocks with a low disease prevalence. This potential problem is overcome by ensuring that foetuses and placentae

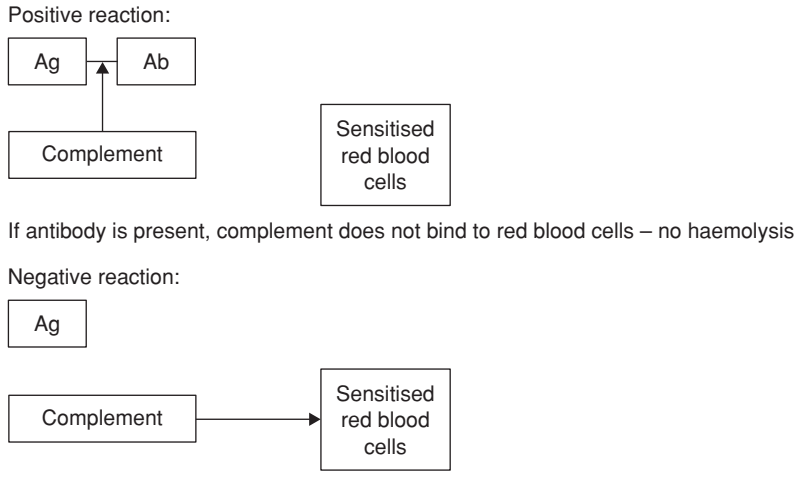


Fig. 1.66 Schematic representation of the complement fixation test.

from all aborted ewes are submitted for laboratory investigation and by additional blood sampling of aborted and barren ewes.

The specificity of the CFT for chlamydial abortion is high, but not 100 percent. Positive CFT results must therefore be interpreted in conjunction with the flock and individual animal history. Individual animals with suspect serum CFT titres

Example

There are 10 000 animals in the population and the prevalence of the disease is 10%. For a test with a sensitivity of 97% and specificity of 99%:

Test result	Infected	Not infected
+	970	90
–	30	8190
Total	1000	9000

The positive predictive value of the test is 970/(970 + 90) = 91%. Thus positive test results are correct 91% of the time.

But, if the prevalence is only 1%:

Test result	Infected	Not infected
+	97	99
–	3	8190
Total	100	9900

The positive predictive value is 97/(99 + 196) = 50%. Thus positive test results are only correct 50% of the time.

of ≥ 4/32 are followed up by the more specific, but not necessarily sensitive, Western blot test. (The Western blot test involves separation of serum proteins into distinct bands by electrophoresis and blotting onto a membrane. The presence of antigen on the membrane is revealed by bathing the membrane in a solution of labelled antibody.)

Zoonotic risk

C. abortus can infect the human placenta giving rise to abortion, stillbirth and serious maternal illness. Pregnant women should therefore never work with infected lambing flocks and precautions must always be taken to avoid exposure to *C. abortus* through contaminated clothing or equipment. Indeed the risks to pregnant women

Example

Six of 350 Greyface ewes aborted during the final three weeks of pregnancy (Fig. 1.67). All of the ewes had originally been sourced from flocks which were accredited free from chlamydial abortion and abortion problems had not previously been diagnosed in the flock. Groups of 80 to 90 ewes had been housed in four large pens. The products of four abortions were submitted for laboratory investigation and chlamydial abortion was diagnosed in each case. The risk of a serious abortion storm during the following year was high, because the flock was naïve and the large lambing groups would have enabled spread of infection throughout the flock.

About three weeks before the following mating period, half of the ewes were vaccinated using the inactivated vaccine. The remainder of the flock, comprising mostly older ewes, was not vaccinated. During the following lambing season, 8.6 and 12.4 percent of the lambs born to vaccinated and unvaccinated ewes respectively were stillborn or aborted.

This treatment-control study showed that vaccination with the inactivated vaccine reduced the incidence of abortion in infected ewes. However, despite vaccination of half of the flock, serious losses occurred.

The source of the chlamydial abortion problem was not identified. The flock has since continued to include purchased Greyface ewe lambs from accredited chlamydial abortion-free sources and chlamydial abortion has been effectively prevented by vaccination of these replacement animals before their first mating as gimmers.

Fig. 1.67 Housing ewes in large groups increases the risk of spread of infectious abortion.

posed by contact with all lambing flocks, and not only known-infected flocks, should be considered. *C. abortus* can also cause respiratory disease and flu-like illness in some people working with infected sheep.

Toxoplasmosis

Toxoplasmosis is an important worldwide production-limiting sheep disease. Losses can be difficult to quantify, because in addition to identifiable late abortions, foetal loss can also occur during early pregnancy, and there are difficulties associated with retrospective diagnosis.

Life cycle

Toxoplasma gondii is a protozoan parasite, which requires more than one host species to complete its life cycle. The organism is primarily an intestinal parasite of domestic cats, with a wide range of intermediate hosts including sheep and mice. Sexual reproduction is usually initiated when a non-immune cat ingests food, usually mice, contaminated by tissue cysts. Bradyzoites freed from the tissue cysts by proteolytic enzymes in the cat's gastrointestinal tract pass through several stages before gametogeny begins in small intestinal epithelial cells. Resultant oocysts are then discharged into the intestinal lumen and passed with the cat's faeces. Sporulation occurs within 1 to 5 days and each sporulated oocyst contains four sporozoites. (Simultaneously with this sexual life cycle, some ingested bradyzoites penetrate the lamina propria of the cat's intestine and reach other organs, especially mesenteric lymph nodes, brain and muscle, where tissue cysts develop.) Shedding of oocysts only persists for about 8 days before the cat becomes immune. Young cats starting to hunt for the first time are, therefore, the most important animals in the epidemiology of toxoplasmosis. However, recrudescence of infection can occur when older cats are stressed or suffering from immunosuppressive disease (Fig. 1.68).

Oocysts are infective to any mammal or bird. Following ingestion of oocysts, tachyzoites are released. Tachyzoites actively penetrate host cells and multiply asexually until the cell ruptures, releasing the organisms which then parasitise further host cells. When the host develops immunity, extracellular parasites are eliminated and multiplication of intracellular tachyzoites slows as they form bradyzoites in tissue cysts. Tissue cysts are usually found in brain and muscle.

Fig. 1.68 While healthy adult cats are not a major source of *T. gondii* oocyst contamination, the status of individual animals is unknown, so they should be kept away from lambing accommodation.

The most important source of infection to cats is tissue cysts in small rodents. Infection in rodents can spread vertically from generation to generation. Furthermore, mice or birds with brain tissue cysts may be more prone to being caught by young cats. A non-immune cat can shed over one million oocysts per gram of faeces. There is, therefore, potential for the contents of entire hay sheds to become contaminated following drying and airborne spread. Likewise concentrate feed or straw bedding may become infected. Pastures may be infected following spreading of oocyst-contaminated manure. Only 200 oocysts may be sufficient to cause disease in a pregnant ewe, so one infected cat can provide sufficient environmental contamination to infect a large number of sheep. Oocysts can survive in a suitable cool and moist environment for up to 2 years.

While cat faeces are considered the main source of toxoplasma infection for susceptible sheep, the organism has been isolated from ram semen and rams have been implicated in the transmission of disease. Other modes of transmission to sheep, such as vertical transmission when a ewe infected during late pregnancy gives birth to live infected lambs, are not considered to be significant.

Clinical signs

Infection of non-pregnant ewes results in strong immunity with parasite survival only in intracellular tissue cysts as already described. In pregnant animals, the placenta and subsequently the foetus are infected, although the severity of the disease depends on the stage of gestation when infection occurred and the level of challenge.

Infection of ewes during early pregnancy may result in embryonic death and resorption which may present as a barren ewe problem. Infection during mid pregnancy may result in either foetal death followed by mummification, or foetal retardation due to compromised placental nutrition and foetal infection. Late pregnancy infection may result in the abortion of freshly dead lambs or the birth of weak lambs with high consequent neonatal mortality rates. Litter mates may be affected to different degrees. In practice, a combination of all of these outcomes occurs within a flock.

Aborted ewes remain clinically normal and some may not be diagnosed until lambing, when they are identified as being barren. The classic picture of toxoplasmosis is of a late aborted lamb alongside a brown-coloured mummified foetus and placenta (Fig. 1.69). In practice, late aborted lambs are born in various stages of decomposition, often with serosanguinous subcutaneous oedema and varying amounts of pleural, pericardial and peritoneal fibrinous exudate. Placentae are often autolytic, with

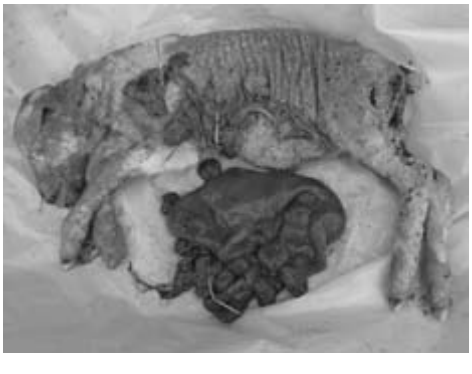


Fig. 1.69 A late aborted lamb alongside a mummified foetus and placenta.

hyperaemic cotyledons having grossly visible, small white focal areas of necrosis and calcification. In endemically infected flocks, without control measures, the abortion or barren rate is highest in ewe lambs and gimmers, and lowest in older ewes. However, in naïve flocks the disease prevalence is similar in all age groups.

Diagnosis

Gross postmortem signs of cotyledonary inflammation and focal necrosis in late aborted foetuses are suggestive of toxoplasmosis. The diagnosis can be confirmed by histological examination of placental cotyledons, demonstrating the presence of small numbers of intra- and extracellular, ovoid and 1 to 4 μm long tachyzoites, usually in close proximity to necrotic areas. Intercotyledonary membranes are usually normal in appearance. Small focal areas of necrosis surrounded by inflammation are often identified in the foetal brain, heart, liver, lung and spleen, representing primary damage caused by *T. gondii*. Other brain lesions such as focal leucomalacia are non-specific and represent anoxic changes due to placental insufficiency.

The diagnosis is supported by positive serology results from foetal pleural or peritoneal fluids. Antibodies are only present in immunocompetent foetuses which have been exposed to infection. Several serological tests are available, including a latex agglutination test (LAT), an indirect fluorescent antibody test (IFAT) and an enzyme linked immunosorbent assay (ELISA). Most UK diagnostic laboratories use the IFAT on foetal fluids and LAT on maternal serum.

Serological tests can also be applied to ewes, although the interpretation of maternal serology is problematic. High titres are usually consistent with recent infection and failure to demonstrate antibodies provides good evidence that toxoplasma abortion has not occurred. However, positive LAT titres below 1/1024 merely indicate infection at some stage in the past. Antibody levels frequently remain high into the following breeding season and can persist for several years. Serology on paired sera collected 3 to 4 weeks apart is also difficult to interpret, due to persistent and varying titres.

Evidence of an increase in lambing percentage and reduction in the portion of barren ewes following vaccination may retrospectively support a diagnosis of toxoplasmosis.

Control

Injectable or oral sulphonomide antibiotics have been used to reduce losses in the face of late abortion storms. Sulphadiazine or sulphamezathine/pyrimethamine injections given on or about days 100, 115 and 130 of pregnancy have successfully mitigated late pregnancy losses. Unfortunately few of these drugs are still licensed for use in food-producing animals in the UK.

The level of oocyst contamination may be reduced by rodent control and keeping feed stores secure from cats (Fig. 1.70). Hay or straw bales from the top of stacks are most likely to be contaminated by cat faeces, so should be used for cattle. Measures should be taken to limit cat breeding, and to maintain a healthy adult cat population. However, depopulating cats is seldom successful as a control method for toxoplasmosis, because oocyst shedding is generally highest in young cats hunting for the first time and removing an established cat population allows new young cats to move onto the farm.

The coccidiostat drug decoquinate can be fed to pregnant ewes as an aid to prevention of abortions due to toxoplasmosis. Unfortunately toxoplasmosis can cause

Fig. 1.70 Feed stores should be covered to prevent them being used by cats for defaecation.

abortion or foetal loss at any stage, so to be fully effective decoquinate should be fed throughout pregnancy. This is prohibitively expensive and impractical for most commercial flocks, partly due to the cost of concentrate feed required as a carrier. In practice, decoquinate is included only during the final 6 to 8 weeks of pregnancy, during which period concentrates are regularly fed.

Ewes which are exposed to an oocyst-contaminated environment before their first pregnancy develop a life-long immunity and do not abort. This situation is difficult to reproduce naturally, because it generally takes about 4 years for 90 percent of ewes on endemically-infected farms to develop *T. gondii* antibodies. Vaccination is, therefore, the most obvious method of control. A live vaccine was developed in New Zealand based on a strain of *T. gondii* (strain 48) that had lost the ability to form bradyzoites in tissue cysts following vertical transmission through several generations of mice. This vaccine is used in many UK flocks.

Replacement ewe lambs for breeding or gimmers are vaccinated once, no less than 3 weeks before mating. The vaccine data sheet claims protection for 2 years, but in practice booster doses are not required. Ideally sheep should not be vaccinated with a live enzootic abortion vaccine within 4 weeks of toxoplasmosis vaccination, although in practice it is acceptable to administer both products on different sides of the neck at the same time. The live toxoplasmosis vaccine is delicate and has a very short shelf life, so must be handled strictly according to the manufacturer's instructions. The S48 strain of *T. gondii* has lost the ability to complete its life cycle, so is considered safe if accidentally injected into human operators. Nevertheless, protective gloves and goggles should always be worn when handling live vaccines. Live vaccines should never be handled by pregnant women.

Zoonotic risk

Humans can become infected following ingestion of bradyzoites in uncooked meat and following ingestion of sporulated oocysts from an environment contaminated by cat faeces. It is also possible that tachyzoites in aborted placentae may be a source of infection for humans. In most cases, infection is asymptomatic, perhaps only resulting in formation of a few muscle tissue cysts. A strong immunity develops following first exposure. In pregnant women infected for the first time, the foetus and placenta can become infected, and while disease is seldom as severe as in sheep, abortion can result from infection during early pregnancy. Human infection during

mid pregnancy can result in tissue cyst formation in the foetal brain and cause intellectual and development problems in the baby. Women who are or may be pregnant should therefore wear gloves when gardening, not eat undercooked sheep meat and stay away from lambing ewes. Cat litter trays are safe if cleaned daily, because there is insufficient time for oocyst sporulation.

Campylobacteriosis

Campylobacteriosis occurs sporadically in the UK and accounts for about 3 percent of diagnosed abortions. Abortions usually occur during the last 3 to 6 weeks of pregnancy involving between 1 and 20 percent of affected flocks, depending on their immune status and the level of exposure, with the most severe outbreaks being seen where sheep are intensively housed under suboptimal nutritional and hygiene conditions.

Campylobacteriosis has been reported in most sheep-rearing countries worldwide, but has a particularly high prevalence in New Zealand where high winter stocking densities predispose to high levels of environmental contamination.

Clinical signs

Typically, one or two abortions occur about 6 weeks before the start of lambing, followed 2 to 3 weeks later by more abortions, which continue into the lambing period as stillbirths. Late aborted foetuses may be born alive, but are weak and subsequently die from starvation, hypothermia or other diseases.

Affected ewes show no premonitory signs. Aborted ewes are identified by hollow flanks and blood-stained, or sometimes purulent vaginal discharges (Fig. 1.71), persisting for up to 3 weeks. Retained foetal membranes are sometimes observed and occasionally severe metritis develops, characterised by pyrexia and anorexia of a few days' duration.

Aborted foetuses are usually fresh and externally normal in appearance. The placenta is inflamed and oedematous, and may be covered by large quantities of thick, white-yellow exudate. On postmortem examination, foetuses may contain large quantities of serosanguinous peritoneal, pleural and pericardial exudate. In New Zealand, about 25 percent of aborted foetuses have characteristic 1 to 5 mm diameter, cream coloured 'doughnut-shaped' necrotic areas in swollen and haemorrhagic livers, although these lesions are seldom seen in the UK.

Fig. 1.71 Purulent vaginal discharge following campylobacter abortion.

Cause

Abortion outbreaks are most severe when animals are closely confined in unhygienic conditions which enable the build-up of a substantial level of infection in the environment. Thus, abortion storms occur most commonly in silage-fed ewes, housed in large, overcrowded groups. The principal cause is *C. fetus* subspecies *fetus*. The source of infection is usually carrier ewes and transmission is either by direct contact with aborted material and vaginal discharges, or from a contaminated environment. Ewes may remain carriers and continue to shed the organism in vaginal discharges for up to 6 weeks following abortion. The organism can survive for 3 weeks on feed or in soil. Enteric infection of carrion-eating birds, such as seagulls, magpies and crows, following ingestion of aborted placentae provides another important route for spread of infection between flocks. Birds have been shown to excrete *C. fetus* subspecies *fetus* for up to 213 days after experimental infection. Trough feeding is sometimes associated with high levels of infection because it attracts scavenging birds. Unlike *Campylobacter fetus* subspecies *veneralis* infections in cattle, venereal infection does not appear to occur in sheep.

Following ingestion of *C. fetus* subspecies *fetus* by late-pregnant ewes, a bacteraemia occurs, with localisation in the placenta after about 14 days, which results in an acute, necrotic inflammatory response. The organism also crosses the placenta, and in the foetus causes multiple necrotic foci in all organ systems, notably in the liver and lungs. Foetal death is followed by abortion between 7 and 25 days after the initial infection. Some aborted ewes become intestinal carriers for a long unknown period of up to 18 months, and may be responsible for maintenance of the infection within a flock from year to year.

Diagnosis

The gross lesions in the foetus and placenta are not specific. A quick positive diagnosis can be made by direct microscopic examination of Ziehl-Neelsen or Gram-stained smears from stomach contents of freshly aborted foetuses. *C. fetus* subspecies *fetus* appears as small, paired, comma-shaped bacteria, with the appearance of 'seagulls'. The diagnosis is confirmed by bacterial culture of foetal stomach contents. The probability of reaching a positive diagnosis is increased when several samples are submitted and the presence of other infectious abortion agents should never be overlooked.

Treatment and control

Aborted or aborting ewes should be immediately and effectively isolated from other late-pregnant ewes and treated with broad-spectrum antibiotics. The products of abortion should be disposed of to avoid further contamination of the environment. Affected lambs born alive should also be considered as a source of infection to late-pregnant ewes and isolated. However, the principal source of infection is a contaminated environment. Moving the flock to fresh fields, regularly turning and moving troughs (Fig. 1.72) and reducing the stocking density of housed ewes can be helpful, although these recommendations do not always fit in well with lambing and

Fig. 1.72 Turning feed troughs between use may reduce contamination with bacterial causes of abortion by bird faeces.

pasture management. Hygienic precautions should be taken to prevent spread of infection on contaminated boots, clothing, or equipment. Whole-flock treatment with high doses of parenteral antibiotics is sometimes practised in the face of abortion storms, although there are no controlled study data to indicate that this strategy is effective.

Aborted ewes are solidly immune, as are ewes infected when not pregnant. In closed flocks infection is, therefore, usually self-limiting and significant losses seldom recur within 5 or more years of an abortion storm. When such losses do occur, the probability of other causes such as chlamydial abortion or toxoplasmosis should be considered. In other flocks, replacement gimmers are at greatest risk of acquiring infection from carrier ewes. In New Zealand, an adjuvant prepared killed vaccine is commonly used, which provides good protection against the most common strains of ovine *C. fetus* subspecies *fetus*. Initially, two injections are required 4 to 6 weeks apart, followed in subsequent years by a single injection before mating or at ram withdrawal. The New Zealand vaccine is not effective against all strains of *C. fetus* subspecies *fetus* and little is known about its possible efficacy in the UK.

C. fetus subspecies *fetus* can cause gastroenteritis in humans, so as with all cases of ovine abortion, strict and sensible hygienic precautions should be taken.

Campylobacter jejuni

C. jejuni is found in the intestinal tract of all domestic mammals and birds and occasionally causes abortion in individual animals. *C. jejuni* is more commonly associated with diarrhoea in weaned lambs and is a potentially important zoonosis.

Salmonellosis

Various serotypes of *Salmonella enterica* sporadically cause abortion in UK flocks. Overall, *S. enterica* only accounts for a small number of abortion outbreaks annually, but the incidence can reach 25 percent in infected flocks. Most abortion outbreaks due to salmonellosis in the UK are caused by *Salmonella enterica* serotype Montevideo, *Salmonella enterica* serotype typhimurium and *Salmonella enterica* serotype Dublin, although other serotypes are occasionally implicated. Overseas, *Salmonella enterica* serotype abortus ovis and *Salmonella enterica* serotype Brandenburg are

Fig. 1.73 A fresh aborted foetus and inflamed placenta caused by *S. montevideo* infection during mid-gestation.

Fig. 1.74 An autolysed and emphysematous foetus caused by late-gestation *S. montevideo* abortion.

important causes of ovine abortion. Strain variation exists within individual serotypes of *S. enterica*, but only certain strains cause abortion in sheep. However, new pathogenic strains emerge periodically. The emergence as important pathogens in UK flocks of currently uncommon serotypes of *Salmonella enterica*, such as *S. brandenburg*, should be anticipated. Therefore, the cause of abortion should always be investigated whenever more than 2 percent of the flock is affected.

Salmonella enterica *serotype Montevideo*

Most reports of *S. montevideo* abortion have been from flocks in the south-east of Scotland. In some cases, abortions have occurred in cattle grazing alongside sheep. In sheep, abortions occur throughout the second half of pregnancy. In many cases the aborted foetuses are not found, but the first sign of the problem is the presence of a red vaginal discharge and hollow flanks in ewes. Some of these ewes become sick and require antibiotic treatment, sometimes associated with retention of the placenta. When abortions occur during mid pregnancy, the foetuses are often fresh (Fig. 1.73), but the placentae appear inflamed. When abortions occur during later pregnancy, aborted foetuses are often autolysed (Fig. 1.74).

Abortions can occur in up to 30 percent of the flock, despite efforts to confine the spread of infection and repeated metaphylactic treatment with long-acting oxytetracycline injections. Gimmers may be more severely affected than older ewes, with abortion storms occurring at roughly 5-year intervals, associated with cycles of previous exposure and immunity.

Salmonella enterica *serotype Dublin*

In parts of the UK, such as the south-west of Scotland, *S. dublin* infection is an important cause of abortion, systemic illness and diarrhoea in ewes (Fig. 1.75). Pregnant ewes sometimes die as a result of systemic disease before aborting. Infection is probably introduced and maintained with carrier cattle.

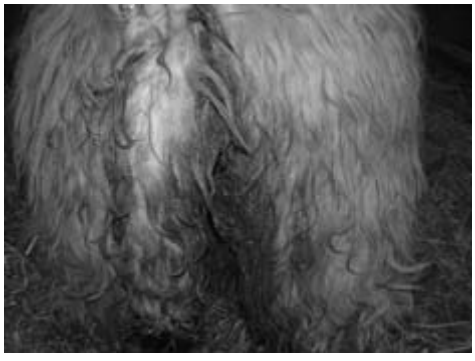


Fig. 1.75 A blood-stained, purulent vaginal discharge and diarrhoea following *S. dublin* abortion.

Salmonella enterica *serotype Brandenburg*

In New Zealand, *S. brandenburg* was first isolated from a Merino ewe flock and diagnosed as the causative organism in an outbreak of abortion and ewe deaths in 1996. Prior to this, *S. brandenburg* was identified as an infrequent human pathogen and occasionally isolated from cattle faeces. In 1997, *S. brandenburg* was diagnosed as the cause of serious abortion outbreaks in 18 flocks in the South Island of New Zealand. Subsequently, *S. brandenburg* has emerged as a major cause of abortion and ewe death throughout New Zealand. The pattern of the disease has been consistent with that of the spread of a new pathogen in a susceptible population.

Disease outbreaks have been characterised by acute necrotising metritis and late abortion, frequently of decomposed lambs. Average lamb losses of 17 percent have occurred and up to 30 percent of aborted ewes have died. Large numbers of *S. brandenburg* bacteria are shed from aborted foetuses and placenta resulting in spread to other ewes and into waterways. Carrier ewes and carrion-eating gulls are implicated in the spread of infection and risk factors include multiple litters and stressful husbandry. There has been an increase in human salmonellosis cases associated with abortion outbreaks in sheep.

Strain typing has shown that the New Zealand *S. brandenburg* epidemic originated from a single unidentified source. The possibility of a similar problem emerging in UK flocks should not be dismissed.

Diagnosis of salmonellosis

As with all ovine abortion problems, it is important to establish the cause promptly so that effective management practices can be employed. Salmonellosis should be suspected when abortion of fresh foetuses occurs in several mid to late pregnant ewes. The likelihood of other causes of abortion may be reduced on the basis of vaccination history, health scheme membership and the presence of systemic disease in some aborted ewes.

Whenever possible, aborted foetuses and placentae should be submitted for veterinary laboratory investigation, but if these samples are not available, vaginal swabs can be useful for the diagnosis of salmonellosis. The diagnosis is confirmed by the bacterial culture of *Salmonella* sp. from the placenta or foetal stomach contents.

Management of salmonella abortion outbreaks

Infection occurs several weeks before ewes abort, so by the time that salmonellosis has been identified, it will probably have spread throughout the group. Nevertheless, precautions should be taken to limit the level of infection and prevent spread to other groups of animals.

Most biotypes of *S. montevideo* are sensitive to a wide range of antibiotics and whole-flock treatment with a long-acting injection of oxytetracycline is often recommended. In practice, treatment is often instigated before the diagnosis is confirmed, although there are few data to support the efficacy of such treatment. Infection is usually established before abortions are seen and treatment instigated.

Zoonotic risk

In common with other causes of ovine abortion, *Salmonella* sp. can cause disease in humans, so sensible hygienic precautions should be observed when working with pregnant and aborted animals.

Prevention of salmonellosis

Salmonellosis seldom becomes endemic in UK flocks and salmonella abortion storms seldom occur in the same flock in subsequent years. There are several possible sources of infection, including contaminated feed, carrier animals, wild birds, contaminated watercourses and humans. Little can be done to avoid some of these possible sources, but moving and turning over of troughs between feeds may reduce contamination by birds.

Border disease

Border disease is a rare cause of ovine abortion in the UK, which can result in serious economic loss in infected flocks. The disease is characterised by barren ewes, abortions, the birth of weak and abnormal lambs, and ill thrift in lamb. Border disease is caused by a pestivirus, very similar to bovine viral diarrhoea (BVD) and swine fever viruses. The differentiation between BVD virus and border disease virus (BDV) is not clear cut, and while most strains can be differentiated, it is possible that cattle pestiviruses may infect and cause sporadic disease in sheep and vice versa. Border disease was first identified in the English-Welsh border area, but has subsequently been reported from sheep producing areas worldwide.

Pathology and clinical signs

There are many similarities between border disease in sheep and BVD in cattle. Healthy sheep exposed to most strains of border disease virus for the first time do not normally show signs of disease. Clinical signs are usually only seen following infection of pregnant ewes.

When a pregnant ewe is first challenged with BDV, viraemia is followed by necrotising placentitis at about 10 days post-infection. In some cases this is sufficient to cause foetal death and abortion, otherwise the virus can cross to the foetus. The out-

Fig. 1.76 A fine tremor and wide-based stance in a stunted lamb.

Fig. 1.77 A hairy birth coat.

come then depends on the immunocompetence of the foetus. The ovine foetus first mounts an immune response at between 60 and 85 days, hence infection before day 60 usually results in uncontrolled virus multiplication and foetal death. Survivors are persistently infected and may have a variety of central nervous system and skin abnormalities due to poor myelination of nerve cells and poor differentiation of primary to secondary wool fibres respectively.

Infection before day 60 of pregnancy

Infection of naïve ewes during the first 2 months of pregnancy generally results in foetal death and resorption, which may pass unnoticed until barren ewes are identified. Sometimes, depending on the virus isolate, breed of ewe and level of infection, lambs survive but are born with combinations of the following clinical signs:

- low birthweights and general weakness
- fine bones and immobile limb joints
- excessively long limbs
- narrow heads, with short mandibles and domed skulls
- nervous signs including a fine tremor at rest, ataxia and hypermetria (Fig. 1.76); many lambs adapt to their nervous disorders, but show noticeable signs following stress or a change of environment
- hairy and abnormally brown pigmented fleeces (Fig. 1.77).

Surviving lambs, which were infected with border disease virus before they were immunocompetent, are generally persistently infected throughout their lives. These lambs often become ill thrifty from about 3 months old, when protective maternal colostral immunity wanes. Some persistently infected lambs die at this stage, while others survive for several years, acting as a source of infection to other animals in the flock.

Infection between days 60 and 85

Foetal infection between days 60 and 85 of pregnancy, during the period when the foetus first develops an immune response, can produce a variety of outcomes. Violent necrotising encephalitis can lead to extensive destruction of areas of the brain, with cerebral hypoplasia and hydrancephaly. Skeletal abnormalities and arthrogryposis may also follow.

Infection after day 85

Foetal infection after 85 days of pregnancy is met by an immune response, and while some abortions follow, surviving lambs are born normal.

This summary greatly oversimplifies the pathogenesis of border disease, which is complex and not fully understood. Overseas, isolates of border disease virus have occasionally caused severe disease in lambs over 3 months old, characterised by produced profound leucopenia, haemorrhagic enteritis, fibrinous pneumonia and death of up to 50 percent of the flock.

Diagnosis

Confirmation of a diagnosis of border disease is not straightforward and is based on clinical signs, histopathology of central nervous tissue and detection of the pestivirus antigen in foetal or lamb blood, kidney, spleen or lymph nodes. However, colostral antibodies can mask the presence of the virus in live lambs. Identification of border disease virus antibodies in ewe blood samples provides evidence of exposure, but is sometimes insufficient to confirm the cause of abortion. Blood sampling single animals is seldom helpful.

Control

BDV is usually brought into a flock with introduced persistently infected ewe lambs or gimmers. Any intensification of husbandry increases the speed of transmission. Other vectors such as cattle or wild ruminants persistently infected with BVD virus or pestivirus-contaminated live vaccines are theoretically possible sources of infection.

Control is very difficult. Lambs that are obviously affected should be culled soon after birth, as they are likely to be persistently infected. Ewe lambs or gimmers should be mixed with the main flock at least 6 weeks before mating and any ewes which produce diseased lambs for two years in succession should be culled. It may be helpful to purchase breeding replacements from unaffected flocks rather than retaining homebred animals, although this introduces a risk of introduction of other diseases. While these strategies will lead to reduction in the prevalence of border disease within a flock, they will not eliminate the infection.

Other infectious causes of abortion

The list of other sporadic infectious causes of ovine abortion includes *Escherichia coli*, listeriosis, leptospirosis, coxiellosis, yersiniosis, *Fusobacterium necrophorum*, *Arcanobacterium pyogenes*, *Mannheimia haemolytica*, *Bacillus* spp. and mycotic infections. These are seldom associated with significant economic loss in UK flocks. Sometimes more than one abortion agent is present in a flock, so the possibility of common infectious causes of abortion should not be dismissed following the diagnosis of these uncommon causes.

Escherichia coli

Shiga toxin-producing *E. coli*, such as serotype O15: krvc383, have been diagnosed as an annual cause of abortion involving between 3 and 10 percent of lowground ewes.

Abortions are characterised by the birth of well-developed foetuses during the final 2 weeks of pregnancy. Some aborted lambs are born alive, but die within 24 hours despite intensive supportive treatment. Some lambs are stillborn and freshly dead, while others are in different states of decomposition. Some abortions are characterised by the birth of live lambs alongside freshly dead or autolysed foetuses. Most aborted ewes are pyrexic and anorexic, with diarrhoea and a dark red vaginal discharge. Many die despite broad-spectrum antibiotic and intensive supportive therapy.

Disease surveillance laboratories occasionally report the isolation of *E. coli* from ovine abortion material. However, *E. coli* are faecal commensal bacteria and common contaminants of abortion material, so their identification does not necessarily confirm the cause of abortion. The diagnosis of *E. coli* abortion should be based on clinical findings of maternal bacteraemia, moderate suppurative inflammation of the placental membranes, and on the demonstration of heavy, pure growths of *E. coli* on bacterial culture of stomach contents of fresh aborted foetuses. The serotype of the *E. coli* and the expression of shiga-like toxins can be determined by specialised laboratories.

Shiga toxin-producing *E. coli* have caused high losses, despite exceptionally high standards of farm hygiene and animal husbandry. Abortions have continued despite whole-flock antibiotic treatment. The persistence of the disease from year to year suggests a potential role of carrier ewes, although this has not been proven.

Shiga toxin-producing *E. coli* can cause serious disease in humans, so sensible precautions should be taken to minimise the risk of human infection.

Listeriosis

Listeriosis is usually associated with encephalitis or septicaemia in silage-fed sheep. However, abortions due to *Listeria monocytogenes* or *Listeria ivanovii* are occasionally reported.

Listeria abortions can occur at any stage of pregnancy. Abortions occur about one week after introduction to contaminated silage and there are generally no distinguishing characteristics or complications to the abortions. The route of infection is believed to be through the gastrointestinal tract with subsequent bacteraemic spread.

Aborted material often shows few lesions because of intrauterine autolysis, but occasionally white miliary necrotic foci are seen throughout the foetal liver (Fig. 1.78). Vasculitis of the allanto-amnion is a consistent feature of listeria abortion.

Fig. 1.78 Multiple abscessation of a foetal liver associated with *Listeria* spp. abortion.

The diagnosis of listeria abortion is confirmed by bacterial culture of *Listeria* spp. from foetal stomach contents.

Leptospirosis

Leptospira interrogans serovar *hardjo* infection is rarely confirmed as a cause of late abortions and stillbirths in UK flocks. The source of infection is pasture, feed or water contaminated by leptospires in cattle or sheep urine.

Coxiellosis

Coxiellosis in ewes is a generally benign disease, which is occasionally associated with abortion (rickettsial abortion).

Coxiella burnetti is spread either directly by tick bites or indirectly by contact with tick faeces. There are also tick-free cycles of infection, with aerosol spread in uterine fluids at normal parturition or abortion, or in milk, faeces or urine. Humans can be infected by any of these routes.

The diagnosis of rickettsial abortion is based on the demonstration of the organism in Ziehl-Neelsen stained placental smears and foetal stomach contents. Positive maternal serology using a complement fixation test indicates exposure, but does not confirm the cause of abortion.

Coxiellosis is an important zoonosis. Human infection is usually asymptomatic, but can result in influenza-like illness referred to as Q fever, which is sometimes followed by pneumonia, hepatitis or endocarditis.

Physical causes of abortion

In practice, non-infectious causes of abortion such as dog worry, rough handling, or stress associated with vaccination are unusual, and unlikely to result in significant losses. Abortions due to severe maternal undernutrition are unusual, and ketotic ewes seldom abort. Overseas, exposure of ewes during early pregnancy to high levels of nitrates in feeds such as brassica crops has occasionally subsequently been associated with early abortions.

Investigation of abortion problems

Abortion outbreaks result in obvious economic loss and welfare concern in sheep flocks. In many cases, abortions occur several weeks or months after infection, so specific management in the face of an outbreak is unhelpful. However, control of the problem in subsequent years relies on an accurate diagnosis, which can usually only be achieved at the time of abortion.

The cause of 30 to 50 percent of abortions is not determined following submission of material for laboratory investigation. This poor positive diagnosis rate is often attributed to submission of inappropriate samples, but may also be associated with failure to look routinely for less common pathogens such as pestivirus, *C. burnetti* or shiga toxin-producing *E. coli*.

The investigation of ovine abortion is no different from that of any other disease problem and includes a relevant disease history, assessment of the environment,

assessment of clinical signs and submission of appropriate samples for laboratory investigation.

Placentitis with necrosis of cotyledons and oedema of adjacent intercotyledonary tissue and the presence of inflammatory foci in the liver, lungs and brain may suggest chlamydial abortion. Gross signs of cotyledonary inflammation and focal necrosis, with normal intercotyledonary membranes in late aborted foetuses may be suggestive of toxoplasmosis. However, these signs are not diagnostic and, in most cases, specific gross lesions in the foetus and placenta are not seen.

Whenever possible, whole aborted foetuses and placentae should be submitted for laboratory investigation. Swabs of vaginal discharges are less helpful. Maternal serum samples are of limited value, although paired sera may occasionally be useful. Samples need to be submitted as quickly as possible and, in general, the more samples submitted, the better the chance of reaching a diagnosis.

When whole foetuses and placentae cannot be submitted, the following samples should be collected:

- a piece of placenta, including a cotyledon and some intercotyledonary tissue
- foetal stomach contents, collected aseptically into a plain vacutainer
- foetal thoracic or peritoneal fluids
- fresh spleen
- various tissues including cotyledon, foetal liver and foetal brain in buffered formalin.

The common causes of abortion can be diagnosed from the placenta, foetal stomach contents and foetal fluids, so preliminary investigation is usually focused on these samples. Ziehl-Neelsen stained placental smears are examined for the diagnosis of chlamydial and *Coxiella* abortions. Amniotic fluid and foetal stomach contents of healthy ewes and foetuses are sterile, so the identification of bacteria in smears or culture of the stomach contents of recently aborted foetuses indicates placental infection. Foetal fluids are used for the serological diagnosis of toxoplasmosis and border disease. The cause of abortion can be further investigated by examining fresh spleen for detection of border disease virus antigen or by histological examination of foetal tissues.

Perinatal lamb survival

About 80 to 90 percent of lamb losses from mid pregnancy to weaning occur between the last week of gestation and the first week of life, or are a consequence of events during this perinatal period. Figures quoted for the incidence of perinatal lamb mortality in UK flocks range between 3 and 30 percent. National statistics, however, need to be interpreted with caution, because of considerable variation within and between flocks, districts, seasons, sheep breeds, ewe age groups, farm management systems and record keeping. Practical experience suggests that the true average perinatal lamb mortality rate in lowland and upland flocks is between 10 and 20 percent. There are few reliable survey data to substantiate these figures, but if they are correct, they represent an important source of economic loss to many individual sheep farmers.

On lowland farms, checked several times per day at lambing, reasonably accurate figures for perinatal lamb losses are available, based on the numbers of dead lambs

collected. In contrast it is impossible to count dead lambs in many hill flocks, where production targets for perinatal lamb mortality are difficult to determine. Traditionally, the only reliable data available for hill flocks were the number of ewes put to the ram and the number of lambs marked, from which a lambing percentage was calculated. Perinatal lamb mortality rates were estimated from the number of lambed ewes not sucking lambs at marking as a percentage of the number of ewes mated, although such estimations may be grossly inaccurate.

Ultrasound scanning to determine pregnancy and foetal numbers is now commonplace, and the accuracy of experienced operators is generally greater than 98 percent. There is now considerable anecdotal evidence that on many UK farms, the reduction in the difference between the number of foetuses present at scanning and the number of lambs docked is between 25 percent and 30 percent. In a few flocks abortion losses may account for a high proportion of this loss, although abortion is not generally a major problem in extensively managed hill flocks. The true perinatal lamb mortality rate in some hill flocks may, therefore, be as high as 30 percent. This represents a significant wastage, and it provides an opportunity for management changes to improve the lambing percentage.

Principles underlying the investigation of perinatal lamb mortality

Systematic postmortem examination of dead lambs can provide information about the time of death in relation to parturition or sucking and provide a pathological diagnosis. From a number of lamb postmortem examinations, the pattern of lamb mortalities emerges. There have been several large-scale postmortem surveys of perinatal lamb mortality in a range of flock types and management systems, where dystocia and starvation-mismothering-exposure have been most often diagnosed, associated with high and low birthweights respectively.

Unfortunately, there are several possible reasons for the various pathological diagnoses. For example, dystocia may be a consequence of sire breed, the dam's pelvic conformation, maternal overfeeding, or prolonged parturition in multiple litters. Furthermore, dystocia injury alone may not result in lamb death, which may only occur when the lamb is subsequently subject to cold stress or undernutrition. Likewise, the catch-all diagnosis of starvation-mismothering-hypothermia has several causes, including dystocia.

The reality is that many postpartum deaths are a consequence of a combination of events occurring pre-partum, during parturition and postpartum. For example, many deaths pathologically categorised as being due to infectious diseases are a consequence of dystocia and/or starvation, and many deaths categorised as starvation-mismothering-hypothermia are a consequence of dystocia or events during late pregnancy such as maternal underutrition or sub-lethal infection with abortive pathogens. Therefore, pathological categorisation of perinatal lamb mortality inevitably oversimplifies the problem, and does not alone provide a practical solution to a flock perinatal lamb mortality problem.

A more useful approach is to investigate the cause of the problem, based on the principle that to ensure periparturient survival, lambs must be born mature, with adequate energy reserves and free from birth stress, and then receive adequate postpartum nutrition. Severe pre- or postpartum malnutrition or dystocia alone may

be lethal, although many neonatal lamb deaths are a consequence of different combinations of moderate pre- and postpartum undernutrition and dystocia.

Pre-partum (foetal) nutrition

Severe maternal undernutrition during mid pregnancy results in inhibited placental development, which causes poor oxygen, nutrient and electrolyte transfer and ultimately results in poor lamb birthweights. Long-term undernutrition of the pregnant ewe inhibits the newborn lamb's capacity for thermoregulation, thereby increasing its susceptibility to hypothermia. Severe undernutrition during the final 6 weeks of pregnancy results in the birth of hypoglycaemic lambs with low liver glycogen and brown fat reserves, and in poor udder development and colostrum production.

Overfeeding of single-bearing ewes during late pregnancy can influence the perinatal lamb mortality rate through dystocia losses of oversized lambs. Regardless of other factors, terminal sire-cross lambs with birthweights below 3.5 kg or greater than 5.5 kg suffer the highest rates of perinatal mortality. Optimum lamb birthweights depend on breed. For example, for Suffolk × Greyface lambs, optimum weights are about 5.5 kg, 4.5 kg and 3.5 kg for singles, twins and triplets respectively. Average birthweights more than 1 kg lighter than these generally indicate maternal underfeeding during late pregnancy.

Birth stress (dystocia)

The important causes of dystocia are poor maternal pelvic conformation, foetal oversize, malpresented lambs and unskilled shepherding. Other causes include partial uterine inertia in polytocous ewes, vaginal prolapse, ringwomb, uterine torsion and ectopic pregnancy.

Anoxia of vital centres of the central nervous system, or the compounding effect of parturition hypoxaemia on pre-existing foetal hypoxia due to placental insufficiency, usually results in parturient foetal death. However, birth-stressed lambs do not always die during parturition. Protracted labour, compression of the umbilical cord, or mild trauma to the foetal central nervous system during parturition result in short-term, usually reversible, foetal hypoxaemia. Maintenance of body temperature, teat searching and sucking behaviour are inhibited in surviving lambs. Furthermore, soft tissue trauma occurring during parturition and subsequent infection may compromise maternal behaviour. Therefore, many neonatal lamb deaths pathologically categorised as starvation-mismothering-hypothermia are in fact a consequence of birth stress.

Postpartum nutrition

An average 5 kg outdoor-born lamb requires about 1 litre of colostrum during its first 24 hours. Failure of the neonatal lamb to feed, or failure of the newly lambed ewe to provide adequate colostrum, results in starvation and poor passive immunity to disease.

Healthy lambs are born with limited energy reserves of plasma glucose and fructose, liver glycogen and brown fat. In physiologically compromised lambs these reserves are depleted or absent. Starved lambs rapidly become hypoglycaemic, and are weak, lethargic and unable to maintain body temperature.

Maternal factors responsible for lamb starvation include:

- genotype (some individuals and certain ewe breeds demonstrate poor mothering behaviour)
- inexperience (ewe lambs and gimmers refusing to suckle their lambs)
- undernutrition (resulting in poor colostrum accumulation)
- dystocia
- generalised infection
- mastitis
- multiple births.

Lamb factors leading to starvation include:

- genotype (some terminal sire bred lambs are slower to suck than pure hill breed lambs)
- multiple litters (three lambs to share two teats)
- birth stress and/or prenatal malnutrition
- hypothermia (hypothermic lambs don't feed)
- infectious disease.

External factors responsible for lamb starvation include:

- high stocking density of lambing ewes (resulting in mismothering)
- disturbance of lambing or newly lambed ewes
- human interference
- poor pasture availability near to the lambing site
- exposure.

Other causes of perinatal lamb mortality

Occasionally severe cold, wet and windy weather results in primary hypothermia, where the rate of body cooling in small lambs is so rapid that death intervenes before brown fat can be catabolised.

Common diseases such as non-specific *E. coli* bacteraemias and watery mouth are a consequence of poor colostrum intake during the first few hours of life and poor hygiene of the lambing environment. In housed flocks, environmental contamination may become overwhelming and infectious diseases, in particular non-specific bacteraemias, can become important and result in significant deaths of lambs between 1 and 3 weeks old. In other individual flocks, *F. necrophorum* navel infections, *Streptococcus dysgalactiae* or *Erysipelas rhuisiopathiae* joint ill, lamb dysentery, *M. haemolytica* septicaemias or enterotoxigenic *E. coli* diarrhoea can become important but, again, there are usually other primary predisposing factors. Inherited abnormalities, such as chondrodysplasia (spider syndrome), Dandy Walker malformation, or arthrogryposis in the Suffolk breed, have the potential to cause large losses, although most have been successfully controlled or eliminated. Iodine deficiency (goitre), copper deficiency (congenital swayback) and selenium deficiency (congenital white

muscle disease) have been associated with high perinatal lamb mortality rates. On some farms predation by foxes can be significant, although most of the casualties are probably already compromised.

Investigation of perinatal lamb mortality problems

Disease history

Concerns about high perinatal lamb mortality rates are usually first raised after lambing, at which time investigation of the problem is limited to the determination of the relevant history.

Information can be gathered about:

- ewe feeding throughout pregnancy
- ewe body condition scores at mating and lambing
- ram breed and selection
- ewe breed and parity
- the lambing percentage
- weather conditions and the provision of shelter during lambing
- the management of ewes at lambing
- the size, shelter and topography of lambing paddocks
- the provision of skilled labour at lambing
- the stage and estimated numbers of lamb losses
- ultrasound scanning data to determine whether losses are in single or multiple lambs
- ewe vaccination programmes
- history of trace element deficiencies and supplementation regimens
- history of abortions
- preventive treatments for other known disease problems.

Monitoring

While this disease history may indicate possible problem areas, it does not provide a diagnosis. It is therefore necessary to outline a monitoring programme for the following breeding and lambing seasons. This may include condition scoring of ewes at mating, blood sampling for 3-hydroxybutyrate, albumin and urea concentrations during late pregnancy, precise recording of lamb losses from a specific sub-flock and the collection of dead lambs for postmortem examination.

Postmortem examination of lambs

The systematic postmortem examination of lambs can provide information on the time of death and provide a pathological diagnosis, such as: dystocia; starvation-mismothering-hypothermia; stillbirths/abortions; and others (including trace element deficiencies, predation, infectious diseases or developmental abnormalities). From a number of necropsies (a minimum of 10 lambs, but ideally more than 20), the pattern of lamb mortality emerges.

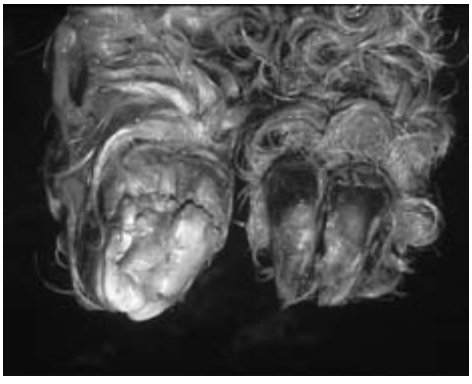


Fig. 1.79 The foot on the left is still covered by a gelatinous cap, indicating that the lamb had not walked, while the foot on the right is hard and belongs to a lamb which had walked.

The basic approach to the postmortem examination of newborn lambs is as follows:

- Lambs should first be weighed.
- The feet should be examined to determine whether or not the lamb had walked (Fig. 1.79).
- The umbilical cord and blood vessels should be examined for signs of blood clotting and desiccation.
- The carcase should be checked for signs of meconium staining, trauma, swellings and other physical abnormalities.
- The carcase is then placed on its back and opened along the midline to expose the peritoneal viscera. The ribcage is incised through the costo-chondral joints and reflected to expose the thoracic viscera and the incision continued along the neck to expose the thyroid glands, larynx and submandibular tissue.
- The brown fat around the kidneys and pericardium should be examined for signs of atrophy. Normal brown fat has a grey-white appearance (Plate 1.1), while atrophied fat is purple coloured and gelatinous in appearance (Plate 1.2).
- The abomasum should be opened to determine the presence of clotted milk.
- The presence of blood in the peritoneum and any rupture of the liver capsule should be noted (Plate 1.3).
- The lungs should be examined to determine if they are inflated or not. Inflated lung tissue is pink coloured and spongy, while uninflated lung tissue has the appearance and consistency of liver tissue (Plate 1.4).
- The submandibular tissue should be examined for the presence of oedema (Fig. 1.80). Skin incisions can also be made over the tail head and hind limbs to determine the presence of subcutaneous oedema.
- The thyroid glands should be examined for evidence of enlargement. In some cases careful dissection, removal and weighing of the thyroid glands to determine the ratio of thyroid to bodyweight may be useful.
- The carcase should be examined for other evidence of sepsis or inflammation.

This simple approach to the postmortem examination is quick and practical. Interpretation of the postmortem findings along with a detailed flock history and examination of ewes and surviving lambs usually provides an indication of the cause of the problem and the basis for constructive advice. For example, postmortem findings of average bodyweights, atrophied brown fat and absence of clotted milk

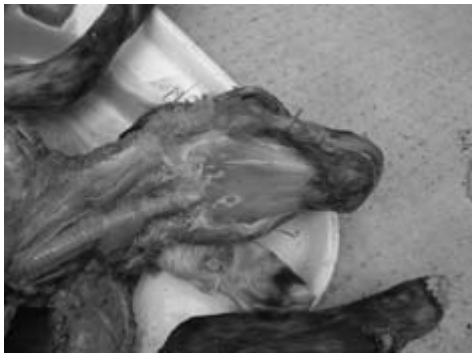


Fig. 1.80 Submandibular oedema following dystocia due to a head-only ('hung lamb') presentation.

in the abomasum indicate deaths from hypothermia, which on the basis of the history might be attributed to poor ewe nutrition, poor mothering ability of primiparous ewes, or disturbance of lambing ewes. Findings of heavy bodyweights, submandibular oedema and/or rupture of the liver capsule indicate deaths from dystocia, which might be attributed on the basis of the history to poor ewe pelvic conformation, poor sire conformation, or unskilled supervision of lambing ewes.

Different factors may be important in different flocks and in different seasons, all with the same end result.

Management practices to minimise perinatal mortality

With the advent of ultrasound scanning for pregnancy, individual sheep farmers are becoming more aware of the magnitude of their perinatal lamb mortality losses. The annual cost of 15 percent perinatal mortality in a 250-ewe lowground flock with a lambing percentage of 180 is approximately 67 lambs × £25 ≅ £1700. Reducing perinatal mortality to 7.5 percent increases the gross margin by about £850, sufficient to illustrate the significance of the problem and justify the cost of preventive management.

Any management practices which ensure correct nutrition of the pregnant ewe, avoidance of dystocia, provision of energy and protective antibodies through colostrum, and a strong maternal bond will enhance the perinatal lamb survival rate. However, the relative importance and practicality of such practices differ between farms, hence general advice may be inappropriate. Recommendations depend on the type of farm, for example, recommending increased supervision of lambing on extensive hill farms would be counterproductive.

Preparation for lambing

The past decade has seen a substantial reduction in labour inputs to UK sheep flocks. Despite this constraint, careful preparation for lambing is paramount to protect the welfare of pregnant and lambing ewes and their newborn lambs.

Lambing should be seen as the critical time when the benefits of preparation throughout the year are realised. The incidence and severity of many lambing problems can be avoided by planned nutritional management of the pregnant ewe.

Careful shepherding and selection of lambing paddocks is required to minimise disturbance of lambing ewes thus enabling the establishment of a good ewe–lamb bond and enhancing the survival of newborn lambs. Whenever possible, steep and exposed fields should be avoided. Lambing fields should not be too large and water sources not too far apart. When unsuitable lambing paddocks must be used, they should be reserved for single-bearing adult ewes.

Despite long-term planning, the need for careful skilled assistance of some lambing ewes is inevitable. A clear plan is required to avoid suffering in ewes which cannot be lambed. This should include guidelines about when to seek assistance, or provision for the immediate humane destruction of distressed animals.

Lambing equipment as follows should be prepared in advance:

- arm-length disposable gloves
- obstetric lubricant
- a head rope, noose or lambing aid
- antiseptic solution
- antibiotics, clean needles and syringes.

Provision should be made for the management of those diseases which occur annually in most flocks around lambing. Clear guidelines should be established about when to seek assistance and to ensure prompt and humane destruction to prevent further suffering when treatment is unsuccessful or uneconomic.

The list of essential supplies includes:

- calcium borogluconate injection for the treatment of hypocalcaemia
- antibiotics for the treatment of mastitis and metritis
- plastic retainers or harnesses, local anaesthetic, clean obstetric tape and needles for the management of vaginal prolapse
- energy supplements for the treatment of pregnancy toxaemia.

Preparation for lambing should also be aimed at prevention and management of disease in newborn lambs. Despite careful preventive management, the occurrence of disease in newborn lambs is inevitable. Provision should be made for the treatment of the common problems and specific diseases which occur in the flock:

- stomach tubes, colostrum, a warming box, glucose injection, syringes and needles for the treatment of starvation and hypothermia
- clips or small syringes and needles for subconjunctival injections to correct entropion
- oral antibiotics for watery mouth prevention
- injectable antibiotics for the treatment of neonatal bacteraemias.

Ensuring adequate maternal nutrition

Ewes lambing in good body condition and well fed during the final 6 weeks of pregnancy suffer the lowest rates of perinatal lamb mortality. Skilful planning is essential to ensure that ewes are mated in good body condition, and correctly fed throughout pregnancy.

Fig. 1.81 In most flocks, triplet litters are undesirable. For example, perinatal lamb mortality associated with underfeeding during late pregnancy is highest in triplet litters.

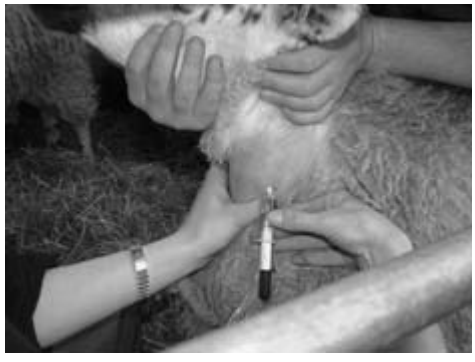


Fig. 1.82 Blood sampling ewes during late pregnancy to determine the adequacy of their protein and energy nutrition is an extremely useful, but under-used management tool.

The maximum period of foetal growth is during the final 6 weeks of gestation. Nutrition during this period has a large effect on lamb birthweights, their subsequent viability and growth rates. The mean birthweights of lambs born to adequately nourished and severely undernourished ewes are significantly different, with higher lamb mortality rates in triplet than twin lambs born to underfed ewes, singles being largely unaffected (Fig. 1.81).

The use of ram harnesses during the mating period, weighing or body condition scoring during mid pregnancy and ultrasound scanning for single or multiple pregnancies, are valuable management tools which can ensure that early lambing, thin or multiple-bearing ewes receive the best nutrition available.

Monitoring changes in liveweight or body condition scores during late pregnancy only provides retrospective information, so is inappropriate as an index of nutritional adequacy. A more immediate assessment of the adequacy of dietary energy supply relative to metabolic demands of late pregnancy can be reliably afforded by measuring ewes' serum (or plasma) 3-hydroxybutyrate (BOHB) concentrations (Fig. 1.82). Increased concentrations reflect the failure of dietary propionate or glucogenic amino acid supply to meet the high glucose requirements of the developing foetuses. Experimental studies have determined the relationship between serum BOHB concentrations and nutritional metabolisable energy intake of single, twin and triplet-bearing ewes of different liveweights during different weeks of late pregnancy. While it is normal for the dietary metabolisable energy intake of late pregnant ewes to fall short of the requirements of twin and triplet litters, maintenance of serum BOHB concentrations below 1.1 mmol/L generally ensures sufficient accumulation of colostrum in the udder and the birth of healthy lambs of normal birthweight. Furthermore, whenever serum BOHB concentrations of late pregnant ewes exceed 1.1 mmol/L, their precise nutritional metabolisable deficit can be determined.

In intensive lowland flocks, blood sampling a representative group of 10 to 12 ewes for BOHB concentrations 3 to 4 weeks before the start of lambing, and adjusting the energy concentration of their ration accordingly is practised to prevent perinatal lamb losses. A range of BOHB concentrations is often encountered in a flock test, which is largely associated with foetal numbers in individual ewes. In those flocks

where foetal numbers have not been determined, a target BOHB value of 0.8 mmol/L (less than the value of 1.1 mmol/L for ewes with known foetal numbers) is used. In those flocks which have been ultrasound scanned to determine foetal numbers, there is little benefit in collecting samples from ewes carrying single lambs, so equal numbers of twin and triplet-bearing ewes are sampled and a target BOHB value of 1.0 mmol/L used. Sick ewes, thin ewes, and those not representative of the flock need to be investigated separately.

The practice of blood sampling ewes to determine the adequacy of their nutritional management is less applicable to extensive hill sheep which appear to have a greater ability to adapt to poor energy nutrition with less reliance on catabolism of body energy reserves, and more regulation of foetal growth. Concurrent health problems during the final 6 weeks of pregnancy such as vaginal prolapse, chronic

Example

Blood samples were collected from a March-lambing flock of 450 Greyface ewes, to determine the adequacy of their energy nutrition. The ewes were grouped according to their predicted lambing dates based on keel marks, but had not been ultrasound scanned for foetal numbers, so were not separated into groups of twin and single-bearing ewes. The ewes had been fed good-quality clamp silage since housing in January, with 0.25 kg/head/day of a good-quality supplementary compound ration (ME = 12.5 MJ/kg DM, crude protein 180 g/kg DM) introduced at the beginning of February, about 6 weeks before the start of lambing. The serum BOHB concentrations and body condition scores of 10 ewes, selected at random from a group which was due to lamb in 3 weeks, are shown in Table 1.2.

The average BOHB concentration of the 10 ewes was 1.2 mmol/L, 0.4 mmol/L more than the target value of 0.8 mmol/L. The additional feeding required to bring the BOHB concentration back to 0.8 mmol/L was calculated by referring to published reference curves for 70 kg ewes (Fig. 1.82). This showed that an increase of 3.0 MJ metabolisable energy per ewe per day was required, which could be met by feeding an additional 280 g/ewe/day of the compound feed with a metabolisable energy concentration of 12.5 MJ/kg DM and estimated dry matter content of 86 percent.

Table 1.2 Serum BOHB concentrations and body condition scores of 10 ewes.

BOHB (mmol/L)	Body condition score (1–5)
0.98	2.0
0.53	2.5
0.93	2.5
1.62	1.5
1.02	1.5
1.04	2.0
0.68	2.5
1.46	2.0
2.63	1.5
1.26	1.5
Mean 1.21	
Target <0.8 (mean)	Target 2.5–3.0

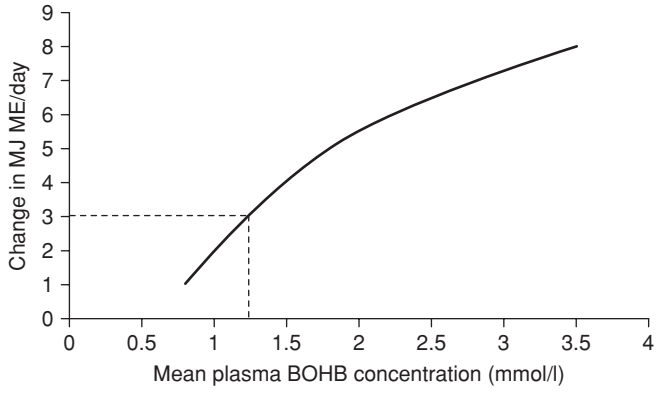


Fig. 1.83 Additional daily energy requirements for a 70-kg ewe to achieve a serum or plasma BOHB concentration of 0.8 mmol/L. (Based on Russel, A. Nutrition of the pregnant ewe. *In Practice* 7, 23–28, 1985.)

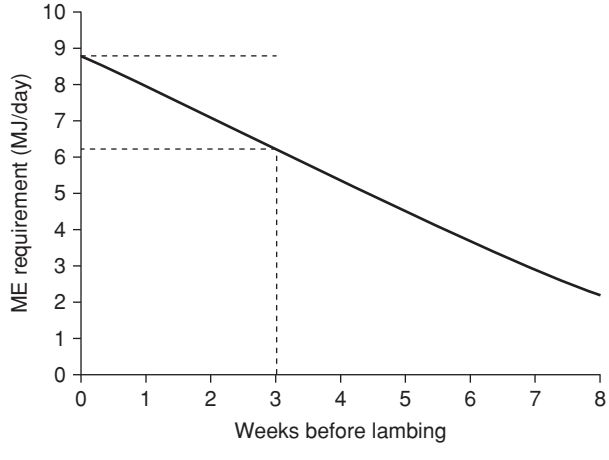


Fig. 1.84 Average daily ME requirements for foetal growth of 70-kg twin-bearing ewes. (Based on Russel, A. Nutrition of the pregnant ewe. *In Practice* 7, 23–28, 1985.)

In addition, provision had to be made to match the increasing demands of the growing foetuses and products of conception during the remaining weeks of pregnancy. Published reference curves for twin-bearing ewes (Fig. 1.84) showed that the metabolisable energy content of their ration would need to be gradually increased over the remaining 3 weeks of pregnancy up to a further 2.6 MJ metabolisable energy per ewe per day, for example by introduction of additional compound feed up to a level of 240 g/ewe/day at lambing.

Additional concentrate feed must be introduced gradually, to avoid poor energy utilisation due to ruminal acidosis. It is often better to substitute better-quality forage rather than to rely solely on supplementary concentrate feeding.

Repeat blood samples were collected 2 weeks later to monitor the response to energy supplementation. The BOHB concentrations of all 10 ewes were less than 0.8 mmol/L, indicating adequate dietary energy nutrition. Routine monitoring of BOHB concentrations in this case enabled timely corrective management to avoid potentially serious pregnancy toxaemia and perinatal lamb mortality problems.

lameness, tooth problems, Johne's disease or jaagsiekte may adversely affect the ewe's feed intake, or protein retention, and result in poor lamb birthweights.

There is some evidence to suggest that shearing ewes during mid pregnancy, using a cover comb, can lead to increased lamb birthweights and weaning weights associated with more efficient energy utilisation during late pregnancy and with higher lamb survival weights.

Avoidance of birth stress

An estimated 5 percent of ewes in UK lowground flocks die around lambing time, with about 70 percent of these deaths caused by dystocia. About 10 percent of lowground ewes require assistance at lambing (Fig. 1.85).

The normal ewe gestation period is 143 to 147 days. Impending parturition is signalled by udder development, accumulation of colostrum, slackening of the sacroiliac ligaments and dropping of the abdominal contents. The birth process is divided into three stages:

- First-stage labour is represented by cervical dilation which takes 2 to 6 hours, being fastest in multiparous ewes.
- Second-stage labour is represented by expulsion of the foetus(es) and typically takes about 1 hour. Rupture of allanto-chorion is indicated by a rush of fluid, following which the amnion and foetus are engaged in the pelvic inlet. The amniotic sac appears at the vulva and frequently ruptures at this stage (Fig. 1.86). Powerful reflex and voluntary contractions of abdominal muscles (straining) serve to expel the foetus (Fig. 1.87). In multigravid ewes, the interval between the birth of lambs ranges from 10 to 60 minutes.
- Third-stage labour is completed by expulsion of foetal membranes which usually occurs within 2 to 3 hours of the end of second-stage labour.

Intervention with lambing should only be considered when failure to do so might compromise the health of the ewe or unborn lamb. Distant assessment of the ewe usually gives an indication of prolonged birth stress. Lateral recumbency with

Fig. 1.85 A 7 kg, birth-stressed lamb with a yellow, meconium-stained coat, and recumbent, injured ewe caused by dystocia.

Fig. 1.86 The amniotic sac has ruptured and the foetal nose is just visible at the vulva.

Fig. 1.87 Expulsion of a foetus in normal anterior presentation.

Fig. 1.88 A foetal head, but no forelimbs ('hung lamb') presentation requires immediate intervention.

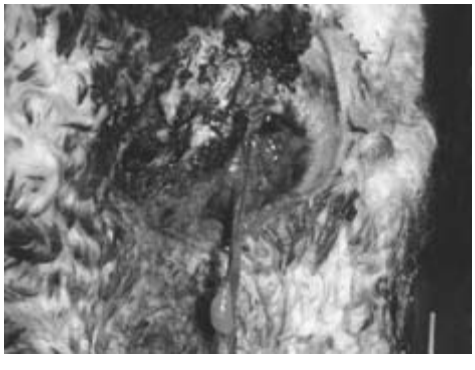


Fig. 1.89 Foetal membranes and the presence of a foetal tail head at the vulva.

Fig. 1.90 The cause of dystocia is obvious in this case.

frequent abdominal straining and vocalisation may indicate engagement of the lamb within the pelvis. Tooth grinding (bruxism) and an elevated respiratory rate with abdominal component (panting) may indicate more serious concerns. The need to intervene is intuitive to experienced shepherds, but as a rough guideline, lambing should be assisted when:

- The interval between rupture of the allanto-chorion and expulsion of a foetus exceeds 1 hour.
- No further progress has been made 20 minutes after parts of the foetus have been seen at the vulva.
- A foetal head, but no forelimbs (Fig. 1.88); a tail (Fig. 1.89); or obviously stuck foetus (Fig. 1.90) is seen at the vulva.
- 30 minutes have elapsed after the birth of the first lamb, but twins or triplets are expected.
- A foetid brown vulval discharge is noted, indicating the presence of an autolytic foetus.
- Other problems such as vaginal prolapse have been identified.

Fig. 1.91 Strict hygiene, in this case involving wearing of disposable arm-length gloves, is essential.

Unskilled intervention often results in oedema and bruising of vulva, with evidence of vaginal bleeding on the tail and perineum. When strict hygienic precautions are not observed, this often leads to metritis (Fig. 1.91). Ewe discomfort can be reduced and manipulations facilitated by blocking reflex abdominal contractions using sacrococcygeal (caudal) epidural analgesia.

Sacrococcygeal epidural analgesia

Sacrococcygeal epidural analgesia is best achieved with the ewe standing or in sternal recumbency. The sacrococcygeal site (Fig. 1.92) is identified as a depression one joint cranial to the first intercoccygeal joint, which is identified by obvious movement on manipulation of the tail. The site is surgically prepared, before a 20 gauge x 1 inch needle is inserted into the space in the midline, with the point directed at a 10° to 20° angle cranial to the vertical and in the plane of the vertebral column. The needle is advanced slowly into the epidural space, which is identified by a change in resistance to its passage. Lignocaine is then injected at a dose rate of 0.25 to 0.5 mg/kg (1 to 2 ml of 2 percent lignocaine hydrochloride). Injection into the epidural space requires little pressure, indicating that the needle is correctly placed (Fig. 1.93).

Fig. 1.92 The sacrococcygeal site prepared for epidural injection.

Fig. 1.93 Correct placement of the needle and effortless caudal epidural injection of lignocaine.

Fig. 1.94 Adequate provision of shelter is essential.

Ensuring adequate early lamb nutrition

Any management practice aimed at ensuring adequate nutrition of the pregnant ewe and the prevention of dystocia will also influence early lamb nutrition, helping to ensure the birth of vigorous lambs, adequate colostrum accumulation, the prevention of pregnancy toxaemia and mastitis, and non-disturbance of the newly lambed ewe. Additional skilled labour employed for the supervision of lambing should also be available to ensure that lambs are correctly mothered, to supplement the nutrition of small or weak lambs, and to treat hypothermic or diseased lambs. These tasks should be seen as the primary reason for supervision. Lamb stomach tubes, a supply of colostrum, glucose solution for intraperitoneal injection, a lamb warming box and a bottle of penicillin are essential lambing kit.

The selection of appropriate lambing paddocks is important. For example, steep slopes should not be used if at all possible, to avoid the risk of the newborn lamb rolling away from the ewe, becoming isolated and dying from starvation-mismothering-hypothermia.

When provided with adequate neonatal care, even lambs suffering from mild birth stress or pre-partum undernutrition can survive. The provision of adequate shelter in lambing paddocks is an essential component of such care on all farms. The positioning of supplementary wind breaks such as straw bales or tin sheets should be carefully planned around the ewes' preferred lambing sites. On extensively managed hill properties, provision of shelter is paramount (Fig. 1.94), because it may be the only possible method of improving conditions.

Specific causes of dystocia

Foetal dystocia

Foetuses should normally present anteriorly in the birth canal, with both forelimbs extended underneath the head. Dystocia can result from alteration in the relation between the long axis of the foetus and the maternal birth canal, alterations in the relation between the foetal vertebral column and the maternal birth canal, or alterations in the position of the foetal limbs and head. These problems are defined as presentational, positional and postural abnormalities respectively. Dystocia can also result from relative foetal oversize where the foetal dimensions are normal but the

ewe's pelvis is too small, and absolute foetal oversize where the maternal pelvis is normal but the foetus abnormally large.

Presentation and postural abnormalities are very common but are generally simple to correct provided that such dystocias are identified early during second-stage labour. The presence of a foetus in posterior presentation is usually characterised by normal first-stage labour, but reduced straining during second-stage labour, because the lamb does not become fully engaged within the ewe's pelvis to stimulate reflex abdominal contractions. Excessive traction of lambs in posterior presentation can cause trauma to the ribcage at the costochondral junctions and/or rupture of the liver, resulting in impaired respiratory function or death.

Posterior presentation with both hips extended and the pelvic limbs drawn under the body, aligning the tail to the pelvic inlet, is referred to as breech presentation. Most breech presentations occur in twin and triplet lambs. Correction of a breech presentation involves gentle repulsion of the foetus into the body of the uterus, before careful flexion of the stifle, hock and fetlock joints and manipulation of the pelvic limbs in turn into posterior presentation. Unskilled manipulation can result in rupture of the uterus and subsequent death of the ewe.

Relative foetal oversize is commonly seen in ewe lambs because of their small pelvic diameter and relatively large foetal size. As a general rule, ewe lambs should not be mated unless they have reached about 70 percent of their mature adult weight and careful consideration should be given to the choice of terminal sire. The incidence of relative foetal oversize is highest in 'stocky' breeds of ewe such as the Texel, Beltex and North Country Cheviot, whose pelvises tend to be angled forward at the base, reducing the effective area of the pelvic inlet.

Incomplete cervical dilation (ringwomb)

Ringwomb is typically characterised by failure of the cervical os to dilate beyond 3 to 5 cm diameter within 2 hours of the onset of second-stage labour. Such incomplete cervical dilation allows passage of only two or three fingers, even after the gentle application of digital pressure for 5 to 10 minutes. The cervix often feels thickened with obvious corrugations. While ringwomb is commonly reported, it is uncertain if it is a primary disease, or if it occurs secondary to other problems such as disturbance during early first-stage labour; uterine torsion; incomplete cervical dilation associated with a foetus in posterior presentation; or cervical and vaginal scarring as a result of previous dystocia. Furthermore, the incidence of ringwomb is difficult to determine because in situations such as overcrowded, housed flocks, or extensive pasture-managed systems, the timing of onset of first-stage labour is uncertain.

Some cases, where the cervix feels thin with no obvious corrugations, respond to gentle digital pressure applied for 5 to 15 minutes. However digital manipulation is seldom successful in cases where the cervix feels thickened and corrugated. Furthermore, forceful digital pressure encourages bacterial invasion of the posterior reproductive tract and causes cervical tearing and bleeding, which are invariably fatal.

Various smooth muscle relaxant and hormone injections have been advocated for the management of ringwomb, but there is no convincing evidence that any are effective. Cases of ringwomb where the cervix feels thickened and corrugated should therefore be managed by immediate Caesarean section (or euthanasia when the value of the ewe and potential value of the lambs do not justify the cost).

Uterine torsion

Uterine torsion is an unusual condition, characterised by straining, but failure to identify a cervical opening or passage through the vagina into the uterus. Thin spiral folds are sometimes palpable in the vaginal wall. The incidence is highest in thin ewes carrying a large single foetus.

Some cases can be corrected by casting the ewe and rolling her in the perceived direction of the torsion. However, most require delivery of the lamb by Caesarean section.

Prepubic tendon rupture

Rupture of the prepubic tendon occurs in older multigravid ewes during the final 2 to 3 weeks of pregnancy. The problem appears to be particularly common in large Halfbred ewes and is characterised by distinct swelling of the lower left abdomen, immediately cranial to the pubis. In most cases this leads to rupture of the left ventral body wall, with extensive ventral oedema and ventral displacement of the gravid uterus (Fig. 1.95). In some cases the skin touches the ground and becomes excoriated.

Ewes with rupture of the prepubic tendon often have difficulty moving and feeding, leading to pregnancy toxaemia. Affected ewes require close supervision and in most cases lambing must be assisted due to the altered position of the uterus in relation to the pelvic inlet. Euthanasia is required for welfare reasons, whenever the skin becomes seriously excoriated, or the ewe develops pregnancy toxaemia or, sometimes, following delivery of the lambs.

Rupture of the uterus

Excessive manual interference can cause rupture of the uterus with subsequent shock, acute peritonitis and death of the ewe. Ewes are often anaemic (Plate 1.5) and present with fast, shallow abdominal breathing, a rapid pulse and abdominal straining. Bright red arterial blood is often seen at the vulva and over the wool of the hindquarters (Fig. 1.96).

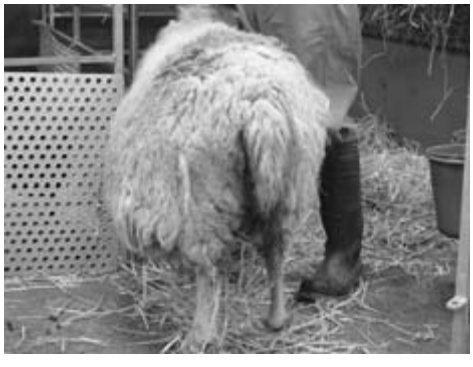


Fig. 1.95 Prepubic tendon rupture in a Merino x Scottish Blackface ewe.

Fig. 1.96 The presence of large clots of bright red, arterial blood at the vulva indicates a uterine or vaginal tear.

Preparturient prolapse

Vaginal prolapse

Preparturient vaginal prolapse occurs annually in most sheep flocks. An incidence of about 1 percent is commonplace, but in some flocks more than 2 percent of ewes of all ages may be affected.

Prolapse of the vagina generally occurs during the last 4 weeks of pregnancy. The extent of prolapse varies from eversion of the vaginal wall, visible as an approximately 8 cm diameter red swelling when the ewe is lying down (Fig. 1.97), to total eversion of the vagina and cervix, up to 20 cm in diameter (Fig. 1.98). The prolapse may contain the urinary bladder, the body of the uterus or both of these structures. The prolapsed tissue rapidly becomes inflamed, swollen, traumatised and secondarily infected. The bladder usually becomes obstructed due to folding of the urethra, leading to urinary retention and uraemia. If uncorrected, infection and necrosis of the prolapsed organ eventually lead to death. Additional problems resulting from persistent straining include rectal prolapse or tearing of the vaginal wall, with herniation of the intestines through the vulva. Vaginal prolapse also leads to dystocia.

Ewes with vaginal prolapse may show many behavioural signs consistent with first-stage labour including isolation from the remainder of the flock, failure to come forward for concentrate feeding, periods spent in lateral recumbency with repeated, short duration, forceful abdominal contractions and associated vocalisation. The duration of prolapse affects the degree of contamination with faeces, bedding material and soil, which compromises the integrity of the vaginal mucosa.

The causes of vaginal prolapse are far from understood, so it is difficult to offer sound advice about prevention of the problem. Most cases occur in ewes with multiple foetuses. There are anecdotal reports to suggest that bulky feed, high-fibre diets, lack of exercise, housing, excessive body condition, subclinical hypocalcaemia, or allowing animals to rest on steep slopes may induce vaginal prolapse, but there are no sound data to support any of these theories. Short-docked tails have been implicated in vaginal prolapse but the condition also occurs in hill breeds with undocked tails.

Fig. 1.97 Eversion of the vaginal wall.

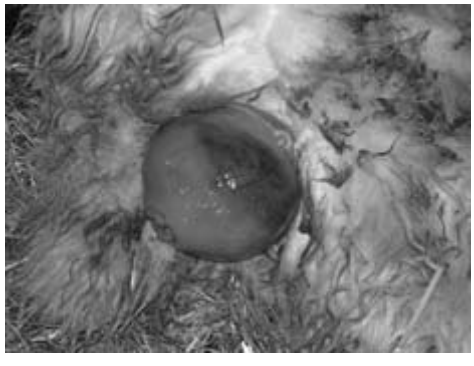


Fig. 1.98 Total eversion of the vagina and cervix, with oedema and bruising.

Ewes with vaginal prolapse require immediate attention to reduce the risks of persistent straining associated with trauma or inflammation, rectal prolapse, intestinal herniation or dystocia. Uncomplicated cases where there is only slight eversion of the vaginal wall can often be managed using plastic retention devices, or preferably harnesses.

Plastic retention devices are shaped such that the central loop is placed within the vagina which is then held within the pelvic canal by the two side arms tightly tied to the fleece of the flanks. These devices can cause considerable discomfort with irritation and secondary infection of the vaginal mucosa resulting in frequent tenesmus, and even re-prolapse. The effectiveness of harnesses depends partly on the skill of the shepherd. Harnesses or trusses are often difficult to fit and can give rise to pressure sores if too tight or not inspected regularly. Faecal staining of the perineum and detection of first-stage labour can become problematic. Most vaginal prolapses respond best to surgical correction involving suturing of the vulva.

Surgical treatment of vaginal prolapse

- **Provide satisfactory pain relief**: Correction of vaginal prolapses and retention using sutures around the vulva is painful and should be performed with effective analgesia. Sacrococcygeal epidural analgesia using lignocaine at a dose rate of 0.25 to 0.5 mg/kg provides good analgesia for the correction of vaginal prolapses and prevention of vigorous straining, but is only effective for 2 to 4 hours. Sacrococcygeal epidural xylazine at a dose rate of 0.07 mg/kg provides caudal analgesia in sheep without hindlimb paralysis for about 36 hours. Unfortunately the analgesic effect of xylazine is slow in onset, taking about 20 minutes. Thus, best results are achieved by injection into the sacrococcygeal space of a combination of 1.8 ml of 2 percent lignocaine and 0.2 ml of 2 percent xylazine (for a 60-kg ewe). There are data to show enhanced ewe survival following this procedure. The administration of epidural injections is an act of veterinary surgery. As an alternative, infiltration of local anaesthetic around the vulva may provide adequate analgesia to enable stitches to be placed, but does not prevent straining.
- **Clean the prolapsed tissue**: It is important not to use strong disinfectants which may cause irritation and lead to further straining. In many cases, dirt and faecal material may be ingrained in the vaginal mucosa and a compromise must be reached between adequate cleaning and causing further trauma.
- **Replace the prolapse carefully and completely**: Gentle consistent pressure is applied with the palm of the hand or clenched fist, first around the perimeter of the prolapse and then at the cervix. This is not always easy, especially if there is involvement of the bladder or urethra. Correction can be performed with the ewe in a standing position, provided that pain relief and prevention of excessive straining is effective. Placing the ewe on her back with the pelvic limbs raised can facilitate vaginal prolapse replacement, but this method should only be adopted when correction in the standing ewe has failed. Once the prolapse is replaced, the ewe should be allowed to urinate.
- **Suture the vulval lips to prevent recurrence**: Suture material should be clean and soaked in dilute antiseptic solution before use. Obstetrical tape is superior to catgut or braided nylon, as it is softer and less likely to act like a 'cheese-wire'. Baler twine and safety pins are not recommended. Simple interrupted and horizontal mattress sutures are straightforward. However, penetration of the vaginal mucosa by

these sutures causes urine scalding and secondary bacterial infection with large diphtheritic areas which results in considerable discomfort and tenesmus. Furthermore, single interrupted and mattress sutures must be removed to permit digital examination of the posterior reproductive tract during periods of suspected first-stage labour, and cannot easily be retied. A purse-string of 5 mm umbilical tape under the skin around the vulva, about 1 cm from the labia and tightened to allow an opening of 1.5 cm diameter, is more satisfactory and causes minimal tissue reaction. The purse-string suture has the additional advantage that it can be tied with a bow, which can easily be untied to allow examination of the posterior reproductive tract for signs of first-stage labour.

- **Control inflammation**: Antibiotic and anti-inflammatory injections are required in cases where inflammation or trauma are obvious.
- **Removal of the sutures**: It is important that ewes are clearly marked following the correction of vaginal prolapse, so that the sutures can be removed before lambing and assistance given if required. Failure to supervise ewes closely can result in the lamb(s) being forced through the vaginal wall before the suture has been slackened, causing perineal laceration.

Abortion sometimes occurs 24 to 48 hours after replacement of the vaginal prolapse, so ewes must be confined and carefully supervised for signs of impending abortion. Unless affected animals are marked and culled at weaning, about 40 percent of vaginal prolapses recur during subsequent pregnancies.

Vaginal prolapse occurring during first-stage labour is often accompanied by incomplete cervical dilation, associated with trauma, superficial infection and oedema of the vaginal wall (Fig. 1.99). Typically, the foetal membranes are presented through the external cervical os and vulva (Fig. 1.100). These cases require management by Caesarean section.

Rectal prolapse

Prolapse and marked oedema of about 5 cm of the rectum (Fig. 1.101) is sometimes seen in ewes with severe tenesmus caused by vaginal prolapse (Fig. 1.102). Most of

Fig. 1.99 Appearance of the amniotic sac at the cervical os. This case required Caesaean section.

Fig. 1.100 Foetal membranes protruding through the cervical os.

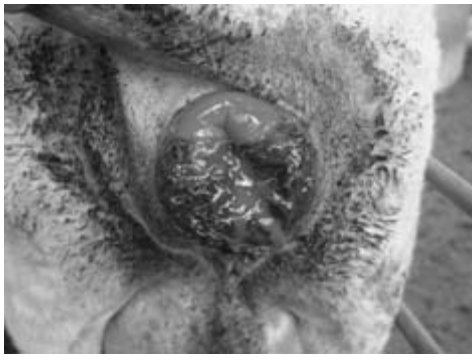


Fig. 1.101 Typical oedema, bruising and haemorrhage of a rectal prolapse.

Fig. 1.102 Rectal prolapse caused by tenesmus due to vaginal prolapse.

these cases can be managed simply by correction of the vaginal prolapse and provision of effective, prolonged caudal analgesia using a combination of lignocaine and xylazine. A single injection of 5 mg of dexamethasone may help to reduce the oedema, with only a small risk of causing premature birth.

It occasionally proves necessary to place a purse-string suture of monofilament nylon subcutaneously around the anus. In these cases, the suture needs to be tightened to reduce the internal diameter to approximately 1.5 cm. However, a balance between ease of defaecation and retention of the rectal prolapse is difficult to achieve, and the long-term recovery rate is poor. Correction of the rectal prolapse by surgical amputation can be attempted, although the procedure is complex and messy, the success rate is limited and the cost may not be justified in commercial ewes.

Evisceration through vaginal tear

Spontaneous evisceration of intestines, caecum and omentum through a tear in the dorsal vaginal wall sometimes occurs in heavily pregnant ewes during the last month of gestation. There is usually no history of previous vaginal prolapse or tenesmus. In some housed flocks the incidence occasionally reaches 2 percent. Excessive body condition, triplet pregnancy and high-fibre diets are thought to be risk factors but the precise mechanism is not known and the problem often occurs spontaneously and for no obvious reason in flocks where management factors have not changed from previous years.

The prognosis following evisceration of the intestines is hopeless, so affected animals should be quickly and humanely destroyed. It is occasionally possible to salvage the lambs immediately after the death of the ewe, although this procedure is rarely

Fig. 1.103 Postparturient vaginal prolapse.

Fig. 1.104 Uterine prolapse about 24 hours after lambing.

successful. Furthermore, when evisceration occurs during late gestation in flocks lambing over a concentrated period there are no ewes to accept such weakly foster lambs.

Postparturient vaginal prolapse

Vaginal prolapse is sometimes seen in ewes during the first 4 weeks after lambing (Fig. 1.103). These cases are usually, but not always, associated with previous preparturient prolapse or trauma of the birth canal at parturition. The management of these cases is the same as for preparturient vaginal prolapses.

Uterine prolapse

Uterine prolapse (Fig. 1.104) results from prolonged and powerful abdominal straining, mostly associated with large single lambs and unskilfully assisted lambings. Uterine prolapse may occur almost immediately after lambing, or after an interval of 12 to 48 hours. Those occurring after an interval of 12 to 48 hours generally result from tenesmus caused by pain arising from infection and swelling of the posterior reproductive tract.

Cases of uterine prolapse require urgent attention to prevent subsequent problems associated with shock, trauma or toxaemia. The principles of replacement are similar to those for the management of vaginal prolapse (Fig. 1.105). Unless the uterus is replaced correctly and fully inverted to its normal position within the abdomen, the ewe will continue to strain, causing considerable distress and suffering, and re-prolapse.

Antibiotics should be administered for 3 to 5 consecutive days after replacement of the uterine prolapse to limit bacterial infection of the traumatised tissues. The ewe's milk yield will be reduced for a number of days after replacement of the uterine prolapse and her lambs will require supplementary feeding. Unlike vaginal prolapse, it is unusual for a ewe to prolapse the uterus the following year, thus there is no necessity to cull surviving ewes prematurely.

Metritis

Opportunistic infection of the uterus by contaminant environmental bacteria commonly results in metritis. Disease is seen in ewes after unskilled or unhygienic

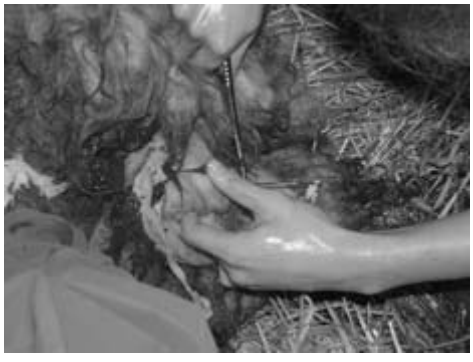


Fig. 1.105 Inserting a Buhner type suture to retain a uterine prolapse.

Fig 1.106 Red/brown, purulent vaginal discharge.

interference at lambing, after delivery of dead lambs, and following some infectious causes of abortion. Metritis is also common following replacement of uterine prolapse.

The diagnosis of metritis is based on the history of dystocia and the exclusion of other diseases of the postpartum ewe. Affected ewes are depressed and inappetant, spend long periods in sternal recumbency and show little interest in their lambs. The mucous membranes are congested and rectal temperature sometimes marginally elevated. The vulva is usually swollen and oedematous with a red/brown, sometimes purulent, discharge (Fig. 1.106). Milk production is poor and lambs appear gaunt and hungry, attempting to suck whenever the ewe stands.

The response to parenteral antibiotic and soluble corticosteroid treatment is generally good. Failure to respond to treatment indicates the presence of a retained foetus or concurrent disease such as mastitis or peritonitis associated with uterine rupture.

Clostridium chauvoei infection (blackleg) is often associated with difficult and unhygienic lambings. The incubation period is 1 to 3 days and the disease is characterised by marked swelling of the vulval and perineal areas, oozing of blood-tinged droplets from the vulval wall and adjacent skin, and dark red areas of necrosis extending to adjacent muscles (Fig. 1.107).

The risk of metritis can be greatly reduced if unnecessary interference with lambing ewes is avoided, great care is taken not to injure the reproductive tract of lambing ewes and simple hygienic measures are adopted for all assisted lambings. These measures should include washing hands and lambing aids in antiseptic solution, the use of clean disposable arm-length plastic gloves, use of obstetrical lubricant and judicious use of long-acting antibiotics following assisted lambings. In multigravid litters, delivering only the lamb in malpresentation or malposture, rather than all of the lambs in quick succession, greatly reduces the likelihood of introducing infection deep into the uterus.

Caesarean section

Unfortunately, the current economics of sheep production mean that veterinary involvement with dystocia is not routinely requested. However, Caesarean section remains a cost-effective and welfare-friendly option when compared to the alternative

Fig. 1.107 Postparturient blackleg, with marked swelling of the vulva and perineum, oozing of blood-tinged droplets and dark red areas of necrosis.

Fig. 1.108 Successful Caesarean section.

of killing the lambs and in all probability the dam as well (Fig. 1.108). Caesarean section can only be performed in the UK by a veterinary surgeon. Pregnancy rates following Caesarean section are comparable to those following normal parturition. The most common conditions which necessitate Caesarean section are relative foetal oversize, large pure-bred lambs in posterior presentation, incomplete cervical dilation and vaginal prolapse.

Anaesthesia for Caesarean section

In practice, ewe Caesarean sections are seldom pre-planned, so withdrawal of feed for 12 hours beforehand is impractical and general anaesthesia is not without risk. Caesarean section is, therefore, usually performed under local analgesia. The options are:

- Infiltration of the left flank incision site with about 50 ml of 2 percent lignocaine solution. This technique is straightforward, but not always effective and may interfere with subsequent wound healing.
- An inverted L block of the left flank incision site using about 60 ml of 2 percent lignocaine solution. This method is straightforward, most commonly practised, and generally effective, but does not prevent abdominal straining.
- A distal paravertebral block of T_{13}, L_1, L_2 and L_3, using about 15 ml of 2 percent lignocaine solution at each site. This method is very effective, but requires skill and experience to perform.
- Lumbosacral extradural injection of 4 mg/kg of 2 per cent lignocaine solution. This method is very effective, providing good visceral relaxation and preventing straining, but it is often 4 hours before motor function returns and the ewe is able to stand. Extradural anaesthesia is particularly useful when there is either considerable trauma to the posterior reproductive tract, a large or deformed foetus *in utero* or a concurrent traumatised vaginal prolapse resulting in tenesmus.

Caesarean surgery

The use of different techniques is a matter of personal preference and the method described is that used by the author. The ewe is placed on two small square straw bales or equivalent and restrained in right lateral recumbency by gently holding down the head. Placing a jacket or towel over the ewe's head usually prevents struggling. The left flank is shaved or clipped (plucking wool from the skin is unacceptable), local anaesthetic injected as appropriate for the situation, and the skin scrubbed for surgery. A fenestrated drape is then applied and held in position with towel clips.

A 12 to 15 cm scalpel blade incision is made in the skin and underlying subcutaneous fat of the left flank, approximately midway between the last rib and the wing of the ilium and starting about 10 cm below the level of the transverse processes of the lumbar vertebrae. The external and internal abdominal oblique muscles are split using scissors, and the transversus muscle and closely adherent peritoneum grasped and raised with forceps. A small nick is made in the peritoneum using scissors, before the incision is carefully extended, taking care to avoid puncturing underlying viscera.

The closest gravid uterine horn is then located and a foetal limb identified. In the case of an anterior presented foetus, the left hock is grasped through the uterine wall and drawn to the incision site, taking care not to puncture or tear the uterus. Using scissors, a 10 to 12 cm incision is made in the body of the uterus over the exteriorised foetal limb. (Such manipulation and exteriorisation of part of the uterine horn can prove difficult when it is oedematous and friable due to the presence of an emphysematous foetus.) The lamb is then delivered through the surgical site (Fig. 1.109). Those in anterior presentation are delivered pelvic limbs first, while those in posterior presentation are delivered head and thoracic limbs first. Further foetus(es) are then delivered as they present through the same incision in the left uterine horn. Those in the right uterine horn are grasped by a hand placed through the uterine incision and carefully directed through the body of the uterus into the right horn.

The uterine incision is closed using 6.0 metric chromic catgut or equivalent absorbable suture material and an inverting Connell suture method. The integrity of the suture line is checked and the uterus gently cleaned using sterile swabs. Continuous sutures of the same material are then used first to repair the peritoneum and transverse abdominal muscles, followed by the internal and the external abdominal oblique muscle layers. The skin incision is closed with a forward interlocking suture of 6.0 metric monofilament nylon or similar.

About 20 ml of procaine penicillin is infused into the peritoneal cavity before closure, although this practice is contentious. Intramuscular injections of 44 000 iu/kg of procaine penicillin are given before surgery commences, and for three consecutive days afterwards. Intravenous injection before surgery of 2.2 mg/kg flunixin helps to control inflammation and pain.

Fig. 1.109 Caesarean section.

Mastitis

Most cases of acute mastitis are seen within 3 weeks of lambing. Most flocks experience an annual incidence of about 2 percent, but in many the incidence is considerably higher. Affected ewes do not feed and often appear lame in one hind limb. The udder is initially swollen, hard, warm and painful (Fig. 1.110), but may become cold and purple-coloured within a few hours (Fig. 1.111). Many ewes die as a result of generalised toxaemia. Those that survive are generally ill thrifty and the affected side of the udder eventually sloughs (Fig. 1.112) to reveal finger-like protrusions of deeper glandular tissue (Fig. 1.113), which become secondarily infected and flystruck.

Mastitis causes substantial economic losses and welfare concerns associated with ewe deaths, reduced lamb growth rates, perinatal lamb mortality (Fig. 1.114) and premature culling of chronically affected animals.

In less severe cases, the udder does not become cold and necrotic, but deep abscesses form within the udder as the acute infection subsides. Lamb growth rates and weaning weights are poor. Chronic mastitis may go unrecognised during the initial stages of the disease, but is later identified by the presence of hard swellings

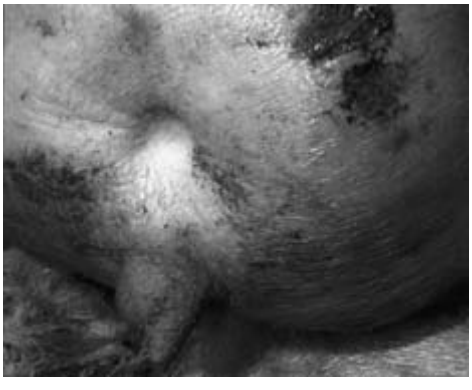


Fig. 1.110 Pitting oedema (remaining impression of a finger pressed against the skin of the udder) in an early case of acute mastitis.

Fig. 1.111 A cold, purple-coloured udder, within a few hours of the onset of acute mastitis.

Fig. 1.112 Necrosis of the skin of the udder, which is about to slough.

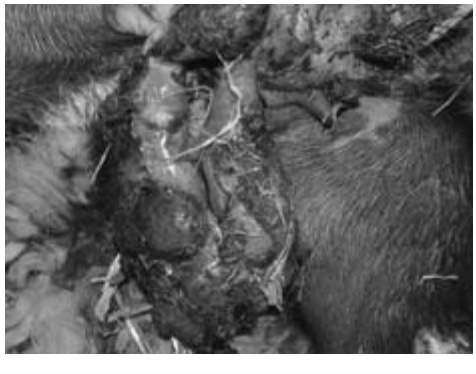


Fig. 1.113 Protrusions of glandular tissue following sloughing of the skin of the udder. These lesions often become flystruck and secondarily infected.

Fig. 1.114 Acute mastitis causes death or ill thrift and compromises the welfare of both ewes and their lambs.

within the udder. These cases are often not detected at the time of weaning when ewes are still lactating, but are found after the subsequent lambing when milk production is poor. In problem flocks, ewes' udders should, therefore, be re-checked before mating.

Mastitis in lactating ewes usually results from bacterial infection of the udder by the bacteria *Staphylococcus aureus* or *Mannheimia haemolytica*. *S. aureus* is present on the teats of all ewes and *M. haemolytica* is found in the mouths of most young lambs. Mastitis occurs when these bacteria have the opportunity to enter into the normally protective teat canal. This can occur when the ewes' milk supply is insufficient, resulting in excessive suckling by lambs and teat injury. Lesions on the teats, or exposure of the teats to cold winds, can also predispose to mastitis. Occasionally other opportunistic pathogens such as *E. coli* are involved, associated with a wet, dirty environment, or with dirty hands checking newly lambed ewes for milk flow.

Early cases respond well to a single subcutaneous injection of 10 mg/kg of tilmicosin antibiotic. However, the treatment response is poor where the udder is already cold, purple-coloured and gangrenous, in which case ewes should be euthanased to avoid further suffering and ill thrift associated with chronic suppurative infection. Those that survive are usually emaciated by the time of weaning.

Prevention of acute mastitis depends on identifying the predisposing factors. In most cases the underlying cause is poor milk production, which can be addressed in subsequent years by attention to the body condition and protein nutrition of ewes during the second half of pregnancy. There is some evidence to support the use of dry cow mastitis tubes or tilmicosin injections in problem flocks at weaning, but substantial data are not available to determine the cost-effectiveness of these strategies. Furthermore, unless intra-mammary tubes are infused under strict hygienic conditions, their use may prove counterproductive.

Metabolic diseases of ewes

Skilful nutritional management throughout the second half of pregnancy is crucial to ensure that good scanning results give rise to high lambing percentages. Undernutrition often results in poor lamb survival associated with low birthweights and poor milk production, while overnutrition is wasteful and can result in dystocia (birth stress) problems.

Nutritional requirements for mammary development

Protein and energy requirements for foetal growth are relatively small during the middle third of pregnancy, when most of the placental development occurs. Indeed ewes with adequate body condition (score 2.5 to 3.5 on a scale of 1 to 5) can afford to lose between 0.5 and 0.75 units of body condition score during this period without affecting lamb birthweights. Undernutrition needs to be severe for at least 7 days before placental development is seriously impaired. However, while most mammary development occurs during the final third of pregnancy, there is some evidence to indicate that sustained protein deprivation from 8 to 10 weeks before lambing can result in poor colostrum accumulation and milk production, with subsequent poor lamb survival, poor lamb growth rates and ewe losses from acute mastitis. Substantial improvements in lamb survival and weaning weights have been demonstrated following supplementation of a hay diet with a source of rumen-degradable protein during the period from housing in early January to the start of concentrate feeding in mid February.

Determination of the adequacy of protein nutrition

The rumen degradable protein content of hay and straw is variable. The adequacy of protein nutrition can be reliably determined by blood sampling about 10 ewes and measuring serum or plasma concentrations of urea nitrogen (BUN) and albumin, which reflect short- and longer-term protein intake respectively. Care must be exercised with the interpretation of these parameters because recent concentrate feeding can influence BUN concentrations. Ewes should be sampled either before feeding, or at least 4 hours after feeding to avoid post-prandial increases in BUN. Low BUN concentrations usually indicate a shortage of rumen degradable protein (ERDP).

Serum albumin concentrations fall during the last month of gestation due to immunoglobulin production and accumulation in the udder. Thus serum albumin concentrations of 26 to 30 g/L are normal during the last month of gestation. (Plasma protein concentrations are often 10 to 20 per cent higher than corresponding serum protein concentrations.)

Example

A high perinatal lamb mortality rate (about 20 percent) associated with poor milk production after lambing was reported in a flock of 800 Greyface ewes (Fig. 1.115). Many of the ewes had firm, poorly developed udders, with 4 percent developing acute mastitis. The lamb birthweights were good, but the lambs did not attain their target weights at weaning. No other signs of disease were seen in ewes or lambs. Similar losses had occurred during previous years.

The ewes had been mated on good-quality dairy cattle aftermath pastures and then housed during mid January. After housing they were fed ad-lib poor-quality hay (ME = 8 MJ/kg DM, crude protein 65 g/kg DM). A small amount of a good-quality concentrate feed (ME = 12.5 MJ/kg DM, crude protein 180 g/kg DM) was introduced during mid February, which was increased rapidly to a high level of about 1.1 kg/head/day for twin and triplet-bearing ewes by early March.

Fig. 1.115 A high lamb perinatal mortality rate and high incidence of acute mastitis was reported in this flock which employed standard nutritional management, suggesting that similar problems are unrecognised or not reported in other UK flocks.

Table 1.3 Results of blood (serum) sampling on 8 February, about 2 weeks after housing and 6 to 8 weeks before the start of lambing.

	BUN (mmol/L)	Albumin (g/L)	BOHB (mmol/L)	Body condition score (1–5)
Ewes scanned for triplets	2.09	32.8	0.23	3.5
	1.58	36	0.49	3.5
	1.5	31.7	0.33	3.5
	1.29	33.7	0.34	3.5
	1.36	31.7	0.35	3.5
	Mean 1.54	Mean 33.1	Mean 0.34	
Ewes scanned for twins	1.83	36.3	0.21	3.5
	1.93	32.8	0.37	3.5
	1.8	33	0.34	3.5
	3.1	33.3	0.29	3.5
	2.61	28.4	0.34	3.0
	Mean 2.2	Mean 32.7	Mean 0.3	
Gimmers scanned for twins	2.14	32.1	0.29	2.5
	1.58	30.3	0.4	2.5
	1.28	33.9	0.34	3.0
	2.1	30.6	0.36	3.0
	2.37	30.5	0.37	2.5
	Mean 1.85	Mean 31.5	Mean 0.35	
Gimmers scanned for singles	1.37	28.1	0.25	2.5
	1.69	28.4	0.26	3.0
	1.33	29.9	0.28	3.0
	2.04	28.9	0.26	3.0
	2.45	33.5	0.25	3.0
	Mean 1.73	Mean 29.7	Mean 0.26	
Target	>2.0	>30.0	<1.0 (mean)	2.5–3.0

The problem was first reported at the end of the lambing period during April, so could not be fully investigated until the following winter, when the ewes were body condition scored and blood sampled for BOHB, urea and albumin (Tables 1.3 and 1.4).

The low BUN values on 8 February indicated a lack of ERDP in the diet which was being fed at the time of sampling. The normal albumin concentrations and good body

Table 1.4 Results of blood (serum) sampling on 22 February, 2 weeks after the first sampling and 4 to 6 weeks before the start of lambing.

	BUN (mmol/L)	Albumin (g/L)	BOHB (mmol/L)
Ewes scanned for triplets	5.58	29.6	0.44
	3.08	32	0.48
	2.7	29.1	0.33
	1.65	27.9	0.46
	3.17	28.6	1.55
	Mean 3.0	Mean 29.4	Mean 0.55
Ewes scanned for twins	2.52	34.3	0.27
	3.02	30.9	0.54
	2.28	29.5	0.36
	3.29	30.5	0.34
	2.73	26.6	0.38
	Mean 2.74	Mean 30.3	Mean 0.37
Gimmers scanned for twins	1.8	27.5	0.36
	1.65	26.3	0.73
	1.88	26.4	0.51
	3.06	30.3	0.58
	2.65	25.4	0.92
	Mean 2.14	Mean 27.1	Mean 0.59
Gimmers scanned for singles	1.88	27.5	0.31
	1.45	26.3	0.22
	1.19	26.4	0.27
	1.49	30.3	0.29
	1.2	25.4	0.26
	Mean 1.42	Mean 27.1	Mean 0.27
Target	>2.0	>26.0	<1.0 (mean)

condition scores provided evidence of adequate longer-term protein nutrition prior to housing. In response to the blood sample results, the ration was supplemented with 100 g/head of soya (ME = 13.3 MJ/kg DM, crude protein 497 g/kg DM) in an attempt to correct this dietary ERDP imbalance. The same 20 sheep were then re-sampled 2 weeks later on 22 February.

Normal BUN concentrations on 22 February in the ewes and gimmers scanned for twins suggested that their dietary ERDP levels were adequate. The albumin values were normal, but lower than at the previous sampling because levels tend to fall in late pregnancy due to colostrum production. The mean BOHB concentrations for each group were below the target of 1.0 mmol/L, indicating adequate dietary energy supply. However, the low BUN concentrations of the gimmers scanned for singles presented a dilemma, which was solved when it became clear they had been overlooked and had not received ERDP supplementation. Soya supplementation was immediately introduced, and its adequacy confirmed by normal BUN concentrations 2 weeks later.

The subsequent perinatal lamb mortality rate was less than 10 percent, the incidence of acute mastitis was less than 1 percent and for the first time in several years lambs achieved their target weights by weaning. This case illustrates the importance of feeding sufficient ERDP during mid to late pregnancy and demonstrates the value of metabolic testing and monitoring of pregnant ewes.

Rumen-degradable protein supplementation

Adequate ERDP supplementation can be achieved by the inclusion of distillers' dark grains (beware of the copper content) or soya, although the protein content of these feeds can also be variable, so further blood sampling about a week after the introduction of these supplements is usually recommended. Urea can provide a cheap and effective means of ERDP supplementation, although great care should be taken to avoid excessive intake by individual animals, which can lead to toxicity problems.

Protein and energy requirements during the final 6 weeks of pregnancy

About 75 percent of foetal growth occurs during the last 6 weeks of pregnancy. Energy and protein undernutrition during this period have a direct effect on lamb birthweights and colostrum accumulation, particularly in twin and triplet litters. The adequacy of dietary energy supply relative to metabolic demands can be determined in most flocks by blood sampling about 10 ewes 4 weeks before lambing and measuring serum or plasma concentrations of BOHB. Furthermore, when the mean 3-hydroxybutyrate concentration is raised the dietary energy deficit can be estimated and feeding adjusted accordingly.

Separation of ewes into different feeding groups on the basis of ultrasound scanning results, ram harness marks and body condition score can also serve to ensure adequate nutrition during late pregnancy and avoid wasteful overfeeding of late-lambing or single-bearing animals.

Ultrasound scanning

Many ewes are ultrasound scanned between about days 80 and 100 of pregnancy to determine the number of foetuses and identify any barren animals (Fig. 1.116). It is important that the information gained is used:

- to remove barren animals from the lambing group
- to allocate ewe feeding according to foetal numbers
- to ensure that those animals likely to require assistance at lambing (usually twin- and triplet-bearing ewes) are separately managed, enabling less time to be devoted to the lower-risk groups

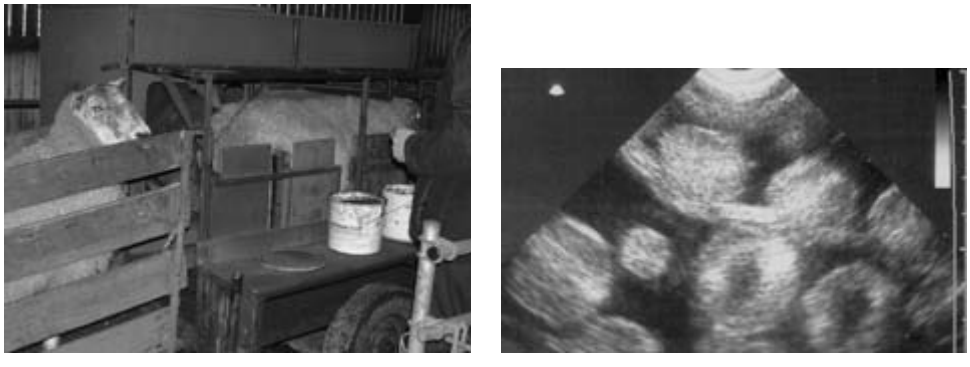


Fig. 1.116 Ultrasound scanning is a useful management tool, but is pointless if the information gained is not used.

- to put an accurate figure on lamb losses before docking/marking, so that unexpectedly high perinatal lamb mortality rates can be investigated and addressed in subsequent years.

Scanning time also provides an opportunity to body condition score ewes and separate ill-thrifty animals for preferential feed management. When ill thrift is seen across the whole flock, inadequate nutrition or diseases such as chronic liver fluke, lameness or sheep scab should be considered. When only a small proportion of the animals are affected, and obvious signs of infectious disease are absent, diseases such as Johne's disease, jaagsiekte or maedi-visna should be considered.

Pregnancy toxaemia

Pregnancy toxaemia is a common disease of undernourished, stressed ewes carrying multiple foetuses, associated with a failure to adapt to the increasing metabolic demands of foetal growth during late pregnancy. All sheep breeds and management systems are affected, but the incidence is highest in lowground flocks of crossbred ewes, typically involving 1 to 2 percent of the ewes in well-managed flocks and up to 10 percent in undernourished flocks. The mortality rate is high and treatment is expensive and generally unsuccessful unless the disease is recognised promptly.

Affected ewes are mostly in low body condition, although the disease is occasionally seen in excessively fat ewes, associated with stressful husbandry. The early clinical signs include separation from the group, reluctance to move, apparent blindness, salivation and fine tremors of the face and ears (Fig. 1.117). As the disease progresses, ewes become sternally recumbent, stuporous and anorexic (Fig. 1.118). Some ewes die within a few days of the observation of clinical signs, but most survive for up to 10 days, becoming soaked in their own urine and developing extensive decubital sores. The death of foetuses in utero sometimes causes a temporary improvement in the condition of the ewe, but subsequent autolysis results in rapid deterioration. Ewes may start to lamb, but the cervix seldom fully dilates and only decomposing foetal membranes are presented. Similar clinical signs may be seen in cases of listeriosis, hypocalcaemia and polioencephalomalacia. The diagnosis of

Fig. 1.117 Early clinical signs of pregnancy toxaemia include separation from the group, reluctance to move and apparent blindness.

Fig. 1.118 Later signs of pregnancy toxaemia include sternal recumbency, stupor and anorexia, with apparent blindness and fine tremors of the face and ears.

Fig. 1.119 A pale fatty liver in a ewe that died as a result of pregnancy toxaemia.

pregnancy toxaemia is differentiated from these by demonstration of blood BOHB concentrations greater than 3.0 mmol/L.

At postmortem examination, carcases are usually emaciated, with multiple foetuses in various states of decomposition in utero and swollen, friable, pale livers (Fig. 1.119). The adrenal glands may be hypertrophied and pale, or hyperaemic. Histopathological lesions consistent with hypoglycaemic encephalopathy have been described in the brain. However none of these postmortem findings are specific for pregnancy toxaemia.

Treatment must be instigated early in the course of the disease, otherwise it is invariably unsuccessful. Successful therapy depends on the ability to raise cerebrospinal fluid glucose concentrations, before irreversible changes occur in the brain. Ewes should be individually penned and offered fresh concentrates, good-quality hay and clean water. Various treatments including oral glycerol, glucose, propylene glycol and concentrated rehydration solutions, intravenous glucose and B vitamins are advocated. However, it may take several days for a sustained rise in plasma glucose concentration to occur following daily intravenous glucose injections or oral dosing with glucose precursors such as propylene glycol or glycerol. The response to oral administration three or four times daily of 160 ml of a concentrated dextrose and electrolyte rehydration solutions is also poor, possibly because these products are mostly deposited into the ruminoreticulum, where the glucose is rapidly metabolised by the microflora.

An alternative approach to the treatment of pregnancy toxaemia involves removing the foetal glucose demands. Parturition can be induced in healthy ewes from about day 135 of pregnancy by injection of 16 mg of dexamethasone or betamethasone, with lambing about 36 hours later. Unfortunately, this method is less reliable in ketotic ewes, whose high endogenous cortisol levels may desensitise them to any drug-induced rise in corticosteroid concentrations. In particularly valuable ewes, Caesarean operation in the early stages of the disease may be an effective method of treatment.

In many cases, treatment is uneconomic and humane destruction should be considered to prevent further suffering whenever the prognosis is poor. In a study of 53 lowground ewes with pregnancy toxaemia, only 33 percent survived, despite intensive therapy, with only 12 percent of their potential lamb crop born alive.

The treatment response in hill ewes, which are often severely undernourished throughout pregnancy, is generally better than in lowground ewes. This is possibly because prolonged undernutrition results in compensatory intrauterine growth retardation, decreased maternal thyroid activity and increased tissue insulin sensitivity.

The occurrence of pregnancy toxaemia usually indicates an urgent need to increase the energy nutrition of the flock. In the short term this can be achieved by introducing ad-lib treacle. In the longer term it may be necessary to increase the amount and/or quality of the concentrate feed, and/or change to higher-quality hay or silage.

Example

Between 2 and 5 December, 8 of 250 housed Dorset cross ewes, which were due to start lambing on 27 December, showed signs of isolation from the main group, depression, apparent blindness and fine muscle tremors of the face and ears (Fig. 1.120). The serum BOHB; BUN; and albumin concentrations of two affected ewes were: 6.28 and 5.2 mmol/L; 3.1 and 4.7 mmol/L; and 30.0 and 29.0 g/L respectively, supporting the clinical diagnosis of pregnancy toxaemia, while indicating adequate protein nutrition.

The ewes had been housed about 1 week beforehand, having been offered hay ad-lib and up to 0.8 kg/head/day of a barley, sugar beet and 34 percent protein balancer mix for the previous 5 weeks (Fig. 1.121). All of the ewes were in fat body condition. The ewes had all been ultrasound scanned for foetal numbers, but were managed as a single group. Oestrus had been synchronised using progestogen sponges and PMSG injections. The BOHB, BUN and albumin concentrations of 10 randomly selected twin-bearing ewes, which were due to lamb 3 weeks later, are shown in Table 1.5.

While two ewes had marginally elevated serum BOHB concentrations, the mean concentration for the group showed that their dietary energy supply was adequate. Likewise, serum BUN and albumin concentrations showed adequate dietary protein nutrition.

All of the ewes which developed pregnancy toxaemia had been scanned for triplets and all failed to respond to treatment. The pregnancy toxaemia problem was associated with low dry matter intakes and impaired gluconeogenesis in individual, excessively fat, triplet-bearing ewes, and was probably initiated by transient stress and competition for space at ring feeders when they were housed. No further cases occurred once the ewes had become adapted to their housed environment. This case report illustrates some of the problems associated with excessively fat body condition and large litter sizes.

Fig. 1.120 Eight Dorset cross ewes showed clinical signs suggestive of pregnancy toxaemia.

Fig. 1.121 Many of the fat ewes refused to eat the average-quality hay that was provided.

Table 1.5 Serum BOHB, BUN and albumin concentrations and body condition scores of 10 ewes.

	BOHB (mmol/L)	BUN (mmol/L)	Albumin (g/L)	Body condition score (1–5)
	0.41	5.5	31.0	4.5
	0.54	2.9	29.0	4.5
	1.34	3.0	30.0	5.0
	0.59	4.1	27.0	4.5
	1.02	2.8	30.0	5.0
	0.77	3.4	24.0	5.0
	0.62	2.4	26.0	4.0
	0.62	2.5	26.0	4.5
	0.97	3.3	31.0	5.0
	Mean 0.76	Mean 3.3	Mean 28.2	
Target	<1.0 (mean)	>2.0	>26.0	3.0–3.5

The poor treatment response of ovine pregnancy toxaemia cases and the observation that the highest mortality rate occurs in ewes with the highest rates of tissue catabolism prior to the onset of disease support the need for flock preventive management. Correct feeding of the dam throughout pregnancy is essential. To achieve this, it is essential that ewes are mated in good body condition and correctly fed throughout pregnancy.

The traditional strategy of flushing ewes before and during the mating period must be adopted with caution. Under many management systems, the potential benefit of having more lambs born is outweighed by the cost of the nutritional management required to maintain multiple pregnancies, twin-bearing ewes in hill flocks and triplet-bearing lowground ewes being prone to pregnancy toxaemia.

Hypocalcaemia

Hypocalcaemia is a common metabolic disease of pregnant ewes which is usually seen from about 6 weeks before lambing. The disease is only rarely seen after lambing. It is important that the condition is accurately recognised, because while untreated clinical cases die, treatment with injections of calcium borogluconate is usually successful. The disease occurs both in housed and outdoor-managed flocks (Fig. 1.122). Under some management conditions, large numbers of animals can be affected.

The initial clinical signs associated with hypocalcaemia are weakness and excitement, but affected ewes are usually found recumbent, often lying with their chins resting on the ground (Fig. 1.123). The pupils are dilated and the ewes appear to be blind. Affected ewes are constipated and sometimes appear slightly bloated. Untreated animals become comatose and die after about 24 hours. In practice, the diagnosis is supported by a history of stressful management and a rapid response to treatment is evident.

Ewes are generally unable to absorb sufficient calcium from their diet for the metabolic requirements of pregnancy and lactation, so depend on the mobilisation of skeletal calcium reserves. Clinical hypocalcaemia occurs when the rates of dietary

Fig. 1.122 Hypocalcaemia in an outdoor-managed pregnant Scottish Blackface ewe the morning after a day of persistent rain.

Fig. 1.123 Typical presentation of hypocalcaemia in a housed Dorset ewe.

calcium absorption and resorption of skeletal calcium reserves are insufficient to meet the requirements for pregnancy and lactation.

Clinical hypocalcaemia is usually associated with a combination of severe dietary calcium deficiency and stressful husbandry. Older ewes are most susceptible, due to depletion and subsequent incomplete repletion of skeletal calcium reserves during and after previous pregnancies. Rapidly growing lush pasture and cereal-based diets contain particularly low levels of calcium, while stress associated with transport, change in feed, hunger due to snow-covered pasture, or temporary water deprivation can precipitate the clinical disease.

Treatment by injection under the skin of about 1 ml/kg of warm 40 percent calcium borogluconate solution is usually successful. Ewes stagger to their feet within 15 to 30 minutes of treatment, urinate, defaecate, show generalised muscle tremors and compulsively eat. However, it is sometimes useful to administer about 20 ml of the drug very slowly intravenously so that any response can be quickly noted. Failure to respond to treatment or relapse after a few hours may indicate the presence of another disease problem, for example, ewes with pregnancy toxaemia are often also hypocalcaemic and may respond briefly to calcium borogluconate treatment.

Prevention of hypocalcaemia in ewes depends on the avoidance of stressful conditions during late pregnancy, in particular unnecessary gathering or transport and sudden changes in feed. Feeding of high levels of calcium to pregnant ewes is ineffective and probably counterproductive, because it could inhibit the rapid mobilisation of skeletal calcium reserves.

Hypomagnesaemia

Naturally occurring hypomagnesaemic tetany is extremely uncommon in sheep. Clinical disease occurs when older lactating ewes, with low magnesium levels and rearing twins or triplets, are stressed due to inadequate feed or bad weather. Affected ewes are often found dead.

The early signs of hypomagnesaemia include separation from the flock, hyperaesthesia to auditory and tactile stimuli, repeated bouts of fine muscle fasciculations of

Fig. 1.124 Lateral recumbency, arching of the head and neck and violent paddling, consistent with hypomagnesaemia in a ewe grazing on lush pasture.

Fig. 1.125 Clinical signs suggestive of hypomagnesaemia, but caused by polioencephalomalacia in a recently lambed Greyface ewe.

the face and periodic urinary tenesmus. The stride is shortened and the ewes display a stiff-legged gait. Their lambs appear to be hungry. If disturbed, ewes develop generalised muscle tremors and become excited. As the disease progresses, they collapse into lateral recumbency, arch their head and neck backwards and paddle violently with all four limbs (Fig. 1.124).

Other diseases which may present with similar clinical signs (Fig. 1.125), include listeriosis, louping ill, polioencephalomalacia and enterotoxaemia although these can often be eliminated on the basis of the flock management history and response to treatment. The diagnosis of hypomagnesaemia is supported by the demonstration of serum magnesium concentrations below 0.6 mmol/L (normal 0.7 to 1.3 mmol/L). When animals are found dead, it is important to rule out clostridial diseases. A history of failure to vaccinate and a postmortem examination may provide a positive diagnosis of clostridial disease, but there are no specific postmortem signs associated with hypomagnesaemia. The postmortem diagnosis of hypomagnesaemia may be supported by identification of low magnesium concentrations in the aqueous humour of the eye.

Treatment by subcutaneous injection of 60 to 80 ml of warm 25 percent magnesium sulphate must be administered promptly because the prognosis is poorer in longer-standing cases.

Magnesium reserves in the body are not readily available, so ewes are dependent on continued dietary intake to meet their very high metabolic requirements for lactation. Hypomagnesaemia results when magnesium concentrations in the extracellular fluid fall due to the combined effects of high metabolic requirements, secretion into milk and poor absorption from the forestomach. Absorption of magnesium is dependent on its concentration in ruminal fluid, and permeability of forestomach wall, which is influenced by oral potassium intake as well as other factors. High rumen potassium concentrations interfere with magnesium absorption. Grazing lush pasture causes reduced chewing and salivary secretion, which increases the ratio of potassium to sodium in the rumen. The problem may be compounded by the early application of potash fertiliser or slurry.

Outbreaks of primary hypomagnesaemia can be managed by drenching surviving ewes with magnesium salts or administration of ruminal magnesium boluses, although this is usually unnecessary. The disease can usually be prevented by adding hay to the ration or moving animals to rougher pasture, which encourages salivation, thus reducing the rumen potassium to sodium ratio. Ewes should only be gradually re-introduced to lush, potassium-rich pasture. Stressful handling should be avoided and shelter provided from cold winds. The spring use of potassium fertiliser should be avoided unless absolutely necessary.

Sodium deficiency

A negative correlation has been observed between the sodium concentration of pasture and incidence of hypomagnesaemic tetany. Sodium is required for active transport of magnesium. Furthermore, sodium deficiency stimulates secretion of the hormone aldosterone, which causes a decrease in sodium and increase in potassium concentrations in saliva and then in the forestomach and circulation. Thus, the consequence of pasture sodium deficiency is the same as that of high potassium intake with respect to reduced magnesium absorption.

Sodium deficiency is probably caused by soil depletion over several years of cereal and silage cropping, compounded by location away from prevailing coastal winds and by leaching from free-draining soil. Herbage sodium concentration is also reduced by application of potassium and nitrogen fertilisers.

Sodium-deficient pasture is generally unpalatable, reinforcing the importance of reaching an accurate diagnosis; because suboptimal production is the consequence of reduced dry matter intake caused by the decreased palatability of pasture, and also of reduced daily magnesium intake. While hypomagnesaemia is traditionally managed by oral supplementation during the spring and summer with magnesium salts, this may be unnecessary if the predisposing causes are identified. In the case of sodium deficiency, the problem can be practically and economically addressed by moving sheep onto permanent pasture, or top-dressing the sodium-deficient pasture with rock salt.

Kangaroo gait

Kangaroo gait is probably a metabolic disease which is usually seen in inadequately fed ewes suckling twin lambs, but the cause is unknown. The condition has been reported occasionally in New Zealand and the UK but has no economic or flock health implications. Affected sheep remain bright and alert, but show difficulty in placing their forelimbs, sometimes knuckling at both front fetlocks, and move with shortened strides with their pelvic limbs drawn well forward under the body, propelling them forward with a characteristic bounding gait (Fig. 1.126). The condition is associated specifically with a bilateral peripheral neuropathy of the radial nerves, characterised histologically by Wallerian degeneration. Most affected animals recover spontaneously after their lambs are weaned.

It is probable that the condition is greatly over-diagnosed due to incomplete clinical examination. Important differential diagnoses include vertebral abscessation (Fig. 1.127), elbow arthritis and any painful condition affecting both forefeet.

Fig. 1.126 Kangaroo gait, with the pelvic limbs drawn forward under the body in a Greyface ewe with large, 1-week-old twin lambs.

Fig. 1.127 Similar clinical signs to kangaroo gait in a Lleyn ewe, caused by a lower thoracic spinal abscess.

Selection for ease of lambing

'Easy care' lambing management

'Easy care' is a misnomer, used to refer to a system of lambing with a minimum amount of human interference. Easy-care systems were developed in New Zealand in response to severe economic pressures following the total removal of subsidy support, the collapse of the wool and skins market and a reduction in the European market for lamb. The primary aim of easy-care systems is the optimisation of lambing percentages through enhanced neonatal lamb survival, but they also confer the advantage of reducing labour at lambing time.

Another common misconception surrounds the way in which easy-care systems evolved in New Zealand. Criticism of the welfare of sheep kept under such systems is mostly unfounded, and fails to appreciate the longer-term welfare gains associated with reducing perinatal lamb mortality. Prior to the development of easy-care systems in New Zealand, there had been several postmortem surveys examining the pattern of perinatal lamb mortality in a range of flock types and management systems. The most common pathological diagnoses were dystocia and 'starvation-mismothering-hypothermia', the latter diagnosis in many cases being related to previous dystocia injury. Early New Zealand investigations of the cause of dystocia involved dissection and comparison of pelvic dimensions of easy lambing and persistent problem lambing ewes. Other studies compared pelvic radiographs of ewes from unshepherded flocks with groups of ewes which had been assisted at lambing. Ease of lambing was positively correlated to the functional area of the pelvic canal, with incompatibility in size between the maternal pelvis and the lamb at birth identified as the main cause for repeated assistance at lambing. Considerable variation was identified within the national Romney flock, with only a proportion possessing the desirable characteristics required to minimise dystocia losses. While these early studies concentrated on maternal pelvic conformation, lamb birthweight, the size and shape of the head and shoulders and limb bone thickness were also identified as important risk factors.

Example

During the early 1960s, a general dissatisfaction with a continuing rise in lamb losses and the ever increasing need for intensive shepherding at lambing led one pioneering New Zealand farmer to run a group of 200 Romney ewes on steep hills and leave them completely unattended for 8 years. Ewes which were identified at docking as having lost their lambs (wet-dry) were culled, and sires were selected from dams which consistently reared live lambs. During the first 4 years 100% were docked; with 15.4% wet-dry and 4% dead or missing ewes. During the next 4 years, 118% were docked; with 10.2% wet-dry and 2.8% dead or missing ewes. The type of ewe most successful for easy care was observed to have long legs and sharp shoulders, associated with pelvic diameter and angle, and the incidence of dystocia was greatest in 'blocky' types. This important farm study showed that ability to rear lambs could be selected for.

Although easy-care systems were aimed at avoidance of dystocia, early observations showed a disproportional high incidence of social stress and mismothering at high stocking densities. Ewes needed to be spread out about a month before lambing commenced so that they had time to settle down and find the warm, sheltered and dry areas. There was a need for careful pasture management to ensure pasture availability for set stocking through to the end of lambing. So long as there was sufficient pasture, ewes tended to remain at the lambing site until the lambs were strong enough to move further afield, but if pasture availability was limited ewes tended to abandon the lambing site to look for food, resulting in lamb losses. Human intervention of any kind invariably resulted in ewes being displaced from their lambing site and subsequent lamb losses.

Thus, prerequisites for New Zealand easy-care systems are:

- a purebred flock enabling selection for ease of lambing and mothering ability in both ewes and rams, or the availability of similarly-selected sheep from other flocks
- a very high level of flock management to enable minimal disturbance of lambing ewes.

Health concerns associated with 'easy-care' systems

In many respects, selection for 'easy care' provides an ideal opportunity to reduce flock health problems. Large pure-bred flocks afford the opportunity to select for resistance or resilience to nematode parasites, which may become crucially important with the emergence of multiple anthelmintic resistance in UK flocks. Maintenance of a more or less closed flock is a practical option for easy-care systems, which affords numerous benefits in terms of avoiding many of the new and emerging flock health problems. However, the prevalence of some slow-onset diseases, notably Johne's disease, jaagasiekte, scrapie and maedi-visna, is commonly higher within closed flocks than within the national flock as a whole. Maedi-visna and scrapie are subject to national control programmes.

Non-disturbance of lambing ewes is only possible if they are healthy and provided with adequate nutrition. Easy-care systems therefore necessitate skilled pasture and

animal management. The adoption of easy-care systems usually necessitates fundamental flock management changes, such as a delaying the start of lambing to coincide with optimal pasture growth.

Technologies such as the use of ultrasound scanning data, recording of raddle marks, and blood sampling pregnant ewes to determine the adequacy of protein and energy nutrition can be used to enable more efficient pasture management at lambing. Careful selection of lambing fields is important, and a high standard of flock health care is required to remove the need to disturb the whole lambing flock while treating individual sick or lame animals.

Easy-care systems essentially involve a move towards pastoral farming, with the potential risk of associated flock health problems. While natural grazing of pasture is generally considered to be healthy for animals, dependence on pasture diets can predispose to certain animal health problems. Trace element deficiencies and gastrointestinal parasitism can become important when the diet is not supplemented by imported forage or concentrate feeds. Agronomic selection of pasture cultivars for resistance to disease may predispose to mycotoxicoses and iodine deficiency.

There is a risk that selection for one advantageous trait may inadvertently enable the emergence of other less desirable traits. For example, selection for good mothering ability and avoidance of interference with ewes at lambing may predispose to stress-related diseases. Salmonellosis characterised by sudden deaths, severe systemic illness and scour in adult sheep, and enzootic pneumonia characterised by sudden death of growing lambs and downgrading of carcases at slaughter, are more common in sheep managed in easy-care systems in New Zealand than in conventionally managed UK flocks.

Alternative management for ease of lambing

The reduction in subsidy support, which will inevitably occur over the next few years, will force UK farmers to consider, try or adopt new technologies such as easy-care systems. Easy-care lambing systems have been an important innovation in New Zealand sheep farming, without which current large-scale, efficient sheep production would be unsustainable. Differences between the UK and New Zealand in the seasonal pattern of pasture growth mean that the New Zealand system of easy care cannot be applied directly to all UK flocks (Fig. 1.128). Furthermore, any attempt at achieving the goal of easy care without consideration for the high level of management

Fig. 1.128 UK winter weather conditions mean that most early and spring lambing flocks require supplementary feeding during the lambing period, which inevitably results in disturbance of lambing ewes.

required could be disastrous. However, there are several measures which can be taken to reduce the level of dystocia and interference with lambing ewes on many UK farms. For example:

- The selection of replacement ewes on the basis of external pelvic conformation in preference to face colour or head size.
- The permanent marking and culling of ewes which require assistance or show signs of poor mothering ability.
- The selection of terminal sires for ease of lambing.
- The use of raddle or keel marks to ensure that early lambing, thin or multiple-bearing ewes receive the best nutrition and lambing management available, while labour inputs to other ewes can be reduced.
- Careful shepherding to minimise disturbance of lambing ewes or employment of sufficient skilled labour to observe and assist with difficult lambings. Some lambing management systems such as drift lambing or housing ewes at night probably result in more disturbance of lambing ewes than others (Fig. 1.129).

Lamb fostering

Most ewes are unable economically to rear triplets, but most can support twins. The benefits of fostering lambs are therefore obvious. Several fostering methods are described. Generally, the most successful method is to smear the orphan lamb with the foster ewe's lambing fluids. It is important that the orphan lamb and the ewe's own lamb are well matched for size and that the orphan lamb is not too old. Use of ultrasound scanning results can facilitate immediate fostering of orphan lambs onto single-bearing ewes.

The use of lamb adopter crates or confinement of the ewe and lamb in a pen with the ewe tied so that she cannot injure the lambs require skilful management to protect the welfare of the ewe and lambs (Fig. 1.130). Attaching the skin of the foster ewe's own dead lamb onto the orphan lamb is sometimes successful (Fig. 1.131). Alternatively, stockingette tubing cut to fit around the ewe's own lamb to absorb its smell and then transferred to the fostered lamb is sometimes successful.

Fig. 1.129 While disturbance of lambing ewes cannot be avoided in housed flocks, its effects can be minimised by careful design of lambing pens and skilful shepherding.

Fig. 1.130 Placing ewes in lamb adopter crates is not always successful, and the practice raises welfare concerns if ewes are ineffectively restrained for too long.

Fig. 1.131 Attaching the skin of the ewe's own dead lamb to the foster lamb.

Fig. 1.132 Orphan lambs require feeding four times per day during their first week of life.

Careful monitoring is required after releasing the foster ewe and lambs. Orphan lambs often appear to follow the foster ewe at first, but are later found starving and hypothermic or dead.

Artificial rearing

Artificial rearing of orphan lambs is often uneconomic and frequently unsuccessful. A high standard of skilful management is required to avoid compromising the welfare of the lambs.

Newborn lambs should be fed 50 ml/kg of ewe colostrum or a colostrum substitute within the first 4 hours of life. During their first week they should be fed about 300 ml of warm milk replacer 3 to 4 times daily (Fig. 1.132). During the following weeks lambs can be fed about 1 to 1.25 litres of cold milk replacer twice daily. It is preferable to introduce lambs to cold milk replacer as soon as possible. Cold milk does not sour as quickly as warm milk and lambs feed less greedily. Bottles, buckets, teats and valves should all be kept clean.

Lamb milk replacers have a higher fat content than calf milk replacers or cow's milk, which are generally unsuitable. Good-quality hay and fresh creep feed should be made available from about 1 week old to promote early rumen development. Orphan lambs can usually be weaned at 4 to 6 weeks old, or when they reach 15 kg liveweight.

Pens should be kept clean, at least 0.4 m^2 of space provided for each lamb, and stocked at no more than 15 lambs per pen (Fig. 1.133).

Fig. 1.133 Pet lamb pens should be kept clean and lambs provided with at least 0.4 m^2 of space to reduce the risk of spread of infectious diseases.

Abomasal bloat

Milk replacer-fed lambs are sometimes found dead, with grossly distended abdomens, about 1 hour after feeding. The problem is associated with overfilling of the abomasum and rapid proliferation of gas-producing organisms. The incidence of abomasal bloat is highest when lambs are fed infrequently, using warm milk replacer, encouraging greedy feeding behaviour. The problem is prevented by feeding lambs regularly with measured amounts of cold milk replacer.

Specific causes of perinatal lamb mortality

Hypothermia

Approximately one million neonatal lamb deaths are attributed to hypothermia each year in the UK. Losses are highest in lambs born outdoors into a wet, cold and windy environment, but can also occur in heavy single lambs born indoors, for example as a consequence of failure to feed due to dystocia injury and chilling draughts. Hypothermic lambs most commonly die during the first 72 hours of life. Small lambs with wet birth coats are most susceptible to heat loss from exposure, while older lambs become hypothermic due to a combination of heat loss and starvation, sometimes secondary to infectious disease.

In the absence of infectious abortion, lambs born to well-fed ewes generally have adequate birthweights, brown fat around the heart and kidneys, and carbohydrate reserves in muscle and liver tissue, which enable them to maintain body temperature for several hours after birth. The period of maintenance of body temperature is dependent on the lamb's colostrum intake; the ewe's mothering ability and the rate of chilling from the environment. Once these energy reserves have been depleted, lambs rapidly become hypothermic, unless they receive sufficient colostrum and shelter to ensure that their rate of heat production exceeds their rate of heat loss.

The normal rectal temperature of newborn lambs exhibiting normal sucking behaviour is between 39 and 40°C. Moderately hypothermic lambs with rectal temperatures between 37 and 39°C are weak, but still capable of following their dam and feeding. Severely hypothermic lambs, with rectal temperatures below 37°C, stand with an arched back, hollow flanks and lowered head, sometimes sheltering close to the ewe's udder giving an impression of feeding, but are unable to suck. In the absence of human intervention these lambs become recumbent, leading to coma and death.

The management of hypothermic lambs depends both on their rectal temperature and on their age.

- Moderate hypothermia (rectal temperature: 37–39°C):
  1 Dry thoroughly.
  2 Ensure a colostrum or milk feed.
  3 Return to ewe.
  4 Supervise closely.
- Severe hypothermia (rectal temperature <37°C) and under 5 hours old:
  1 Dry thoroughly.
  2 Warm to >37°C.
  3 Give a colostrum feed at a rate of 50 ml/kg.
  4 Warm to 39°C.
  5 Return to the ewe.

Fig. 1.134 A Moredun-type lamb warming box provides regulated, all round heat, and is preferable to a heat lamp or hot water bottles.

6 Monitor closely and check dam for milk supply, disease or poor maternal behaviour.

- Severe hypothermia (rectal temperature <37°C) and over 5 hours old:
  1 Inject intraperitoneal 20 percent glucose at a rate of 10 ml/kg.
  2 Dry thoroughly.
  3 Warm to >37°C.
  4 Give a colostrum feed at a rate of 50 ml/kg.
  5 Warm to 39°C.
  6 Return to the ewe.
  7 Monitor closely and check dam for milk supply, disease or poor maternal behaviour.

Lambing buildings should be draught-free and all-round shelter should always be available in outdoor lambing fields. Variations of the 'Moredun-type' lamb heater, which provide a thermostatically regulated all-round heat source, are preferable to heat lamps for warming lambs (Fig. 1.134). Lambs which are unable to suck from a teat should be fed using a stomach tube.

Severely hypothermic lambs over 5 hours old are hypoglycaemic. Warming results in increased cerebral metabolism, which rapidly leads to convulsions, coma and death if the hypoglycaemia is not first corrected by intraperitoneal administration of glucose. Oral administration of fluids to hypothermic lambs causes regurgitation and inhalation asphyxia or pneumonia.

Prevention of hypothermia relies on ensuring that pregnant ewes are adequately fed, avoiding dystocia problems, providing sufficient skilled labour to ensure that newborn lambs feed within the first few hours of life, prevention of neonatal infectious diseases and provision of adequate shelter.

Watery mouth

Watery mouth is a colloquial term used to describe the clinical signs of a disease of 1- to 3-day-old lambs characterised by lethargy, profuse salivation and abdominal distension (Fig. 1.136). In many flocks, watery mouth is an important problem due its high morbidity rate and the poor treatment response in advanced cases. The disease

Intraperitoneal glucose injection

The lamb is held by its pelvic limbs, facing away from the operator (Fig. 1.135). A 2 cm^2 area is cleaned and swabbed with surgical spirit, about 2 cm to the side of and 2 cm below the navel. A 19 gauge, 1 inch needle is then inserted into the peritoneal cavity, directed towards the tail head. Using this method, the peritoneal viscera fall away from the point of the needle and are not punctured. Warm 20 percent glucose solution is prepared by diluting cold 40 percent solution with an equal volume of boiled tap water.

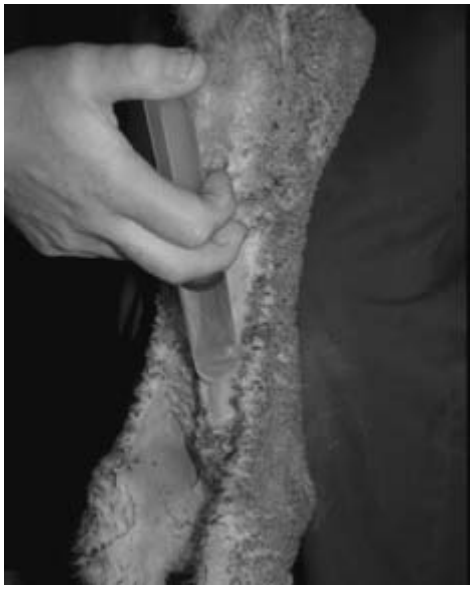


Fig. 1.135 Intraperitoneal glucose injection.

is seen under all management systems, but the morbidity rates are highest in intensive indoor lambing flocks of prolific ewes, affecting up to 24 percent of their lambs. Losses are highest during the second and subsequent weeks of lambing and triplets are three times more likely to be affected than single and twin lambs (Fig. 1.137).

Watery mouth follows reduced or delayed colostrum intake and is essentially a generalised endotoxaemia. The abomasal pH of lambs during their first 24 hours is neutral, a physiological mechanism which enables colostral antibodies to pass undamaged to their site of absorption in the small intestine. The neutral pH also favours the enteric passage and rapid multiplication of non-enterotoxigenic strains of *E. coli* acquired soon after birth, during teat searching, from the ewe's contaminated fleece and udder. Endotoxin is released from the cell walls of the Gram-negative bacteria following their lysis in the intestinal lumen, the metabolites of which are responsible for the clinical signs of watery mouth. Early colostrum intake prevents watery mouth, but the mechanism of protection is not fully understood. It may be associated with promotion of gut motility, neutralisation of endotoxin in the gut lumen, an effect of colostral antibodies in the gut lumen, or simply the fact that

Fig. 1.136 Collapse, saturation of the skin around the mouth and abdominal distension in a 2-day-old Romney lamb due to watery mouth.

Fig. 1.137 Triplet lambs are three times more likely to succumb to watery mouth than twin or single lambs.

colostrum-satisfied lambs cease teat searching and lie down. Bacteraemia has been identified by blood culture from 38 percent of affected lambs early during the course of the disease and 90 percent of lambs at a later stage, but the bacteraemia is not believed to be the primary cause of the disease.

The diagnosis of watery mouth is based solely on the clinical signs and only supported by non-specific postmortem findings. Thorough examination is required to identify all of the clinical signs and to exclude other diseases such as neonatal enteritis which may also present with a wet chin in their terminal stages. The clinical signs of watery mouth begin with depression, anorexia and frequently hypothermia, and rapidly progress to recumbency and collapse. The mouth is cold and the angles of the lips and the lower jaw are wet due to drooling of saliva (Fig. 1.138). Lambs are frequently dehydrated, with abdominal distension. The rectal temperature may be normal, but the extremities are often cold. The ocular mucous membranes are congested and scleral blood vessels dilated. Abdominal palpation is resented and there are usually insufficient faeces in the distal rectum to stain a rectal thermometer.

Non-specific pathological evidence of abomasal distension with clear and mucoid fluid contents, gas-filled, pale small intestines and congestion of various viscera support the clinical diagnosis. Multifocal fibrinous hepatitis, focal suppurative pneumonia and diffuse meningoencephalitis are present in some cases.

The prognosis in advanced cases of watery mouth is guarded and treatment is seldom justified economically. However, lambs are frequently presented for veterinary treatment, which may involve:

- Flunixin meglumine administered intravenously at a dose rate of 2.2 mg/kg, which acts by inhibiting the cyclo-oxygenase pathway in the cytokine cascade, preventing many of the actions of endotoxin.
- Oral dextrose-electrolyte solutions administered at a rate of 50 ml/kg every 6 hours, which help to expand the circulating fluid volume and improve tissue perfusion. Intravenous or intraosseous isotonic or hypertonic saline solutions are preferable to oral fluid therapy and can be administered to valuable lambs.
- Enemas may promote intestinal motility.
- Broad-spectrum systemic antibiotics to treat bacteraemia.

Fig. 1.138 Watery mouth: the chin and angle of the lips are wet due to drooling of saliva.

Fig. 1.139 Oral administration of aminoglycoside antibiotics within 15 minutes of birth for the management of watery mouth.

Emphasis should always be placed on the prevention of further cases; including ensuring correct nutrition of the pregnant ewe; management of abortion and dystocia; adequate supervision to ensure all lambs feed or receive 50 ml/kg of colostrum or colostrum substitutes by stomach tube within their first hour; and maintenance of a clean lambing environment. On many farms, the prophylactic use of oral aminoglycoside antibiotics (apramycin, neomycin and spectinomycin) or amoxycillin administered to all lambs within 15 minutes of birth is practised (Fig. 1.139). Alternatively, oral antibiotic prophylaxis is reserved for the second half of the lambing period, when maintenance of a hygienic environment becomes difficult. Good results are attributed to suppression of *E. coli* multiplication in the intestinal lumen.

Lamb dysentery

Lamb dysentery is a peracute and fatal disease of young lambs caused by the beta and epsilon toxins of *Clostridium perfringens* type B. Over the past two decades the disease had become rare due to the widespread use of clostridial vaccines. However, lamb dysentery is becoming a common problem again, following the withdrawal of ewe vaccination from many flocks. Affected lambs are usually less than 2 weeks old and most commonly 1 to 3 days old. Most cases are characterised by the sporadic sudden deaths of stronger single lambs, which had been consuming the largest quantities of milk. Outbreaks of lamb dysentery can occur during cold and wet springs when lambing ewes are confined to small sheltered areas and conditions become unhygienic. In extreme cases, losses of between 20 and 30 percent have been reported.

Outbreaks of lamb dysentery are initially characterised by the sudden death of young lambs, but as they progress, slower-onset disease, including acute abdominal pain and non-specific nervous signs, is sometimes seen in older, 2- to 3-week-old lambs. Faeces may be semi-fluid and blood-stained, but in most cases they are normal, due to the rapid course of the disease.

The diagnosis of lamb dysentery depends on postmortem findings (Plate 1.6). At postmortem examination large, localised areas of the intestines appear dark red and distended, with ulceration of the mucosa and serous, blood-stained peritoneal fluid. The liver may be pale and friable and the kidneys enlarged. Numerous Gram-positive rods are present in smears from intestinal scrapings. Almost pure anaerobic cultures

Fig. 1.140 Enterotoxigenic *E. coli* can cause severe diarrhoea with high morbidity rates in affected flocks.

of *C. perfringens* from intestinal contents, and positive beta and epsilon toxin ELISA results from intestinal contents or peritoneal fluid support, but do not confirm the diagnosis.

Effective prevention of lamb dysentery is achieved through vaccination using a multi-component vaccine containing toxoids of *C. perfringens* in an adjuvant. Vaccination of ewes protects their newborn lambs through colostral transfer of passive immunity.

Enterotoxigenic E. coli

Disease associated with enterotoxigenic *E. coli* is uncommon, but morbidity and mortality rates are generally very high in affected flocks (Fig. 1.140). Enterotoxigenic *E. coli* possess adherence pili (K99 ± F41) which enable attachment to the intestinal mucosa. Lambs are particularly susceptible to enterotoxigenic infection and colonisation of the small intestinal villi during the first 24 hours of life, caused by a lack of protective hydrochloric acid secretion within the abomasum, slow gastrointestinal motility and an absence of competing microflora in the intestine. It has been suggested that cells containing receptors for adherence pili in neonatal lambs are rapidly replaced by cells without such receptors. Consequently, enterotoxaemia only affects lambs less than 48 hours old. A stable toxin is produced which causes severe watery, brown-coloured diarrhoea. Most lambs die unless prompt fluid therapy is administered.

Control and prevention of enterotoxigenic *E. coli* depend on strict hygiene, ensuring adequate early colostrum intake and immediate isolation of sick lambs. Natural exposure of ewes does not result in significant protection of their lambs, so control of the disease in subsequent years relies on immunisation of ewes using a vaccine (no longer marketed in the UK) prepared from killed *E. coli* containing K99 and F41 antigens administered at the same time as clostridial vaccination. Anti-adherence pili colostral antibodies are believed to act within the small intestine of the newborn lamb to coat binding sites and prevent colonisation of the villi and production of enterotoxin.

Cryptosporidiosis

The protozoan parasite *Cryptosporidium parvum* is not species specific and hence potentially zoonotic. *C. parvum* infection causes villous atrophy of the distal small intestine, leading to malabsorption, secondary fermentation and diarrhoea. *C. parvum* alone seldom causes severe disease in lambs; however, if environmental oocyst

contamination is high or if the lambs are otherwise compromised or stressed, the organism may cause acute onset, pale green-coloured, watery and occasionally blood-stained diarrhoea in lambs between 2 and 20 days old. Morbidity rates are high and, if not promptly treated, mortalities can occur.

Anti-coccidial agents are ineffective for the control or treatment of cryptosporidiosis, so treatment relies on supportive oral fluid therapy. Oocysts can survive for long periods in the environment and are resistant to most disinfectants, hence prevention depends on hygiene and the regular movement of susceptible lambs to a cleaner environment.

Neonatal salmonellosis

Outbreaks of salmonellosis in lambs are rare and usually follow the purchase of infected carrier sheep or calves. Most cases are caused by *S. typhimurium* or *S. dublin*, although outbreaks associated with exotic salmonellae have been reported.

Salmonella spp. cause severe intestinal inflammation, destroying the absorptive capacity and stimulating secretion. *Salmonella* spp. are also invasive leading to bacteraemia and infection of other organ systems. Endotoxins are released into the systemic circulation on bacterial death, causing depression, circulatory failure and cardiovascular collapse. The clinical signs are weakness and profuse green/brown-coloured, blood-stained, foetid-smelling diarrhoea, with variable pyrexia, dehydration and dyspnoea, rapidly progressing to recumbency and death.

The diagnosis of salmonellosis is confirmed by bacterial culture on selective media from faeces, or tissues collected during postmortem examination. Treatment success with intensive fluid therapy, broad-spectrum systemic antibiotics and flunixin meglumine is variable and lambs which recover are ill thrifty and may be carriers.

Enteric viruses

Rotavirus and coronavirus infections do not appear to cause primary disease in lambs, although lambs may be infected during the first week of life. They invade small intestinal villous epithelial cells causing villous atrophy and compensatory crypt cell proliferation, resulting in decreased absorption and increased secretion. The main pathogenic role of enteric viruses is to enable the establishment of other enteric infections.

Hepatic necrobacillosis

Navel infection with *F. necrophorum* can result in the formation of characteristic 'white spot', 2 to 10 mm diameter abscesses in the liver (Fig. 1.141) and secondary spread to joints and lungs (Fig. 1.142). Necrobacillosis is usually associated with poor environmental hygiene and poor passive immunity.

Clinical signs are typically seen in 10- to 20-day-old lambs and are characterised by ill thrift and a hunched-back stance. The response to antibiotic and anti-inflammatory treatment is poor.

Neonatal bacteraemias

Most cases of joint ill (polyarthritis), navel ill (omphalophlebitis), endocarditis or meningitis occur in colostrum-deprived lambs following tonsillar or enteroinvasion

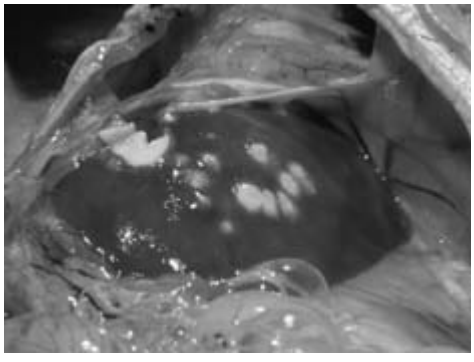


Fig. 1.141 Characteristic hepatic necrobacillosis lesions.

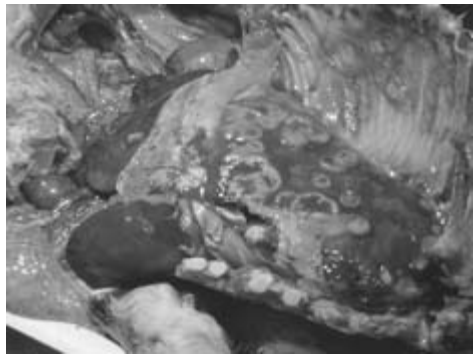


Fig. 1.142 Spread of necrobacillosis lesions to the lungs via the hepato-portal blood vessels.

by opportunistic pathogenic bacteria acquired from a heavily contaminated environment. Bacterial culture from infected tissues usually yields *E. coli*, *Mannheimia haemolytica*, *Pasteurella multocida*, *Arcanobacterium pyogenes*, *Staphylococcus aureus*, or *Streptococcus dysgalactiae*.

Joint ill

Polyarthritis associated with neonatal bacteraemia is sometimes seen in lambs as young as 5 days old, although clinical signs are not usually seen until lambs are 2 to 3 weeks old. Joint ill is characterised by sudden-onset lameness with pain, heat and swelling of multiple limb joints (Fig. 1.143), reduced sucking behaviour and ill thrift. The response to antibiotic and anti-inflammatory drug treatment is generally poor, so the emphasis should be placed on preventive management. On postmortem examination, incised joints contain pus, synovial membranes are thickened and congested and articular surfaces eroded (Fig. 1.144).

S. dysgalactiae is an important emerging cause of polyarthritis in lambs less than 4 weeks old. In the acute stages of the disease, lambs are stiff or weak, but affected

Fig. 1.143 In this case, swelling of the right carpal joint is obvious when compared with the corresponding left joint, so painful manipulation is unnecessary for the diagnosis of joint ill.

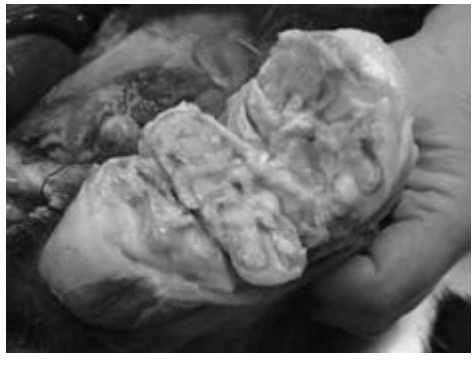


Fig. 1.144 The prognosis for chronic or neglected cases of joint ill is poor, due to thickening of the joint capsule, pus in the joint and erosion of the articular surfaces.

joints are not generally distended. Joint enlargement due to the accumulation of pus only appears as a chronic feature. Involvement of the atlanto-occipital joint is common. The pathogenesis of *S. dysgalactiae* polyarthritis is not fully understood, but it is thought that lambs acquire infection in their first 2 weeks of life, perhaps from the teats or milk of a carrier ewe. *S. dysgalactiae* survives well on dry straw and wool, so an alternative source of infection may be from a heavily contaminated environment. Morbidity rates can be high, despite good hygiene and colostrum management. The disease is often seen in both lambs from a pair of twins and in many flocks recurs from year to year. Treatment with high doses of penicillin and corticosteroids is generally successful when cases are recognised early, but treatment of advanced cases is seldom successful and euthanasia is frequently indicated.

Spinal abscessation

Spinal abscesses are diagnosed sporadically in 4- to 12-week-old lambs following haematogenous bacterial spread during the neonatal period from another focus of infection. Most cases involve *A. pyogenes* or *S. aureus*. Spinal abscesses may also occasionally arise following trauma or lymphatic spread of infection from docking wounds.

Abscesses in the vertebral column either form in the intervertebral foramina, leading to perforation of the dorsal spinal ligament, or in the vertebral body. The clinical signs depend on the location of the abscess and the degree of spinal cord compression. Signs are often sudden in onset in otherwise healthy lambs. Lesions involving the cervical vertebral column are characterised by ataxia and weakness involving all four limbs, with exaggerated thoracic and pelvic limb reflexes and recumbency in severely affected lambs. Lesions between the 6th cervical (C6) and 2nd thoracic (T2) vertebrae are characterised by flaccid paralysis with loss of reflexes of the thoracic limbs, and spastic paralysis with increased reflexes of the pelvic limbs. Most spinal abscesses in young lambs involve the region between T2 and the third lumbar (L3) vertebra (Fig. 1.145). Affected lambs are often found in a 'dog-sitting' position or dragging themselves along by their thoracic limbs (Fig. 1.146). The thoracic limbs have normal function, but there is spastic paralysis of the pelvic limbs.

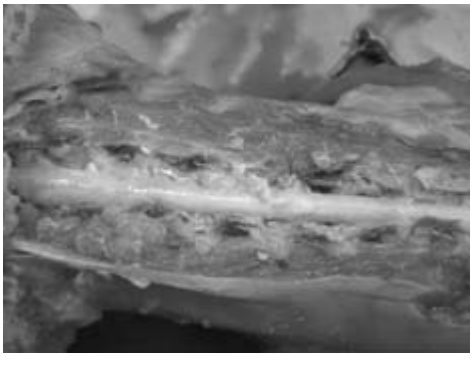


Fig. 1.145 Lumbar vertebral body abscess found on postmortem examination of the lamb pictured in **Fig. 1.146**.

Fig. 1.146 A lamb adopting a dog sitting position and dragging itself along by its thoracic limbs due to a lumbar vertebral body abscess.

Fig. 1.147 Hollow flanks and a 'hunched-back' stance in a lamb with navel ill.

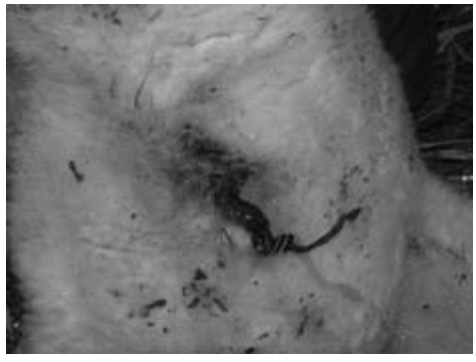


Fig. 1.148 A moist, swollen and painful navel.

The treatment response to antibiotics and corticosteroids is poor because there is extensive damage to the vertebral body before the onset of clinical signs. Most cases require euthanasia to prevent further suffering.

Navel ill

Infection of the umbilical vessels and urachus occurs as a sequel to neonatal bacteraemia, or following direct infection from a contaminated environment. Affected lambs adopt a hunched-back stance, show poor sucking behaviour, develop a hollow-flanked appearance and lose weight (Fig. 1.147). Affected navels are moist, swollen and painful and sometimes exude purulent material (Fig. 1.148). Abdominal palpation sometimes demonstrates painful internal swelling extending along the round ligament of the bladder or along the falciform ligament to the liver. The response to antibiotic and anti-inflammatory drug treatment is dependent on the extent and duration of infection.

Meningitis

Meningoencephalitis occurs sporadically in 4- to 6-week-old lambs. The early clinical signs include a lack of a sucking reflex, with episcleral congestion, dorsal strabismus, a reduced menace response, weakness and an altered gait. As the disease progresses, lambs develop signs of depression leading to stupor and become hyperaesthetic to auditory and tactile stimuli. Lambs are often found in sternal recumbency (Fig. 1.149), with their pelvic limbs extended and their neck flexed backwards. Lateral recumbency, seizures and opisthotonus are observed during the terminal stages of the disease.

Meningoencephalitis is predisposed to by failure of passive antibody transfer and usually arises following bacteraemic spread of infection from the upper respiratory or intestinal tracts. Trauma and local extension from a lesion in adjacent tissues such as the middle ear may occasionally be involved. Initial localisation of the infection within the meninges is followed by an acute inflammatory reaction, with the cerebrospinal fluid providing a vehicle for dissemination of infection over the surface of the brain.

Fig. 1.149 Typical appearance of a Bluefaced Leicester lamb with meningitis. The pelvic limbs are extended, while the head and neck are periodically arched backwards.

Fig. 1.150 Access to these pens involves climbing over gates and lighting is non-existent, hindering the efficient care of newborn lambs.

The diagnosis of bacterial meningoencephalitis is based on the clinical signs and can be differentiated from other neurological diseases of young lambs by the identification of a high protein concentration (0.5 to 8.0 g/L) and neutrophilic pleocytosis in a lumbosacral cerebrospinal fluid (CSF) sample.

The treatment response to corticosteroids and high doses of antibiotics is poor. If attempted, cephalosporin antibiotic therapy should be continued for 4 to 6 weeks, which is cost prohibitive other than for potentially exceptionally valuable lambs. Control depends on ensuring adequate early passive antibody transfer and maintaining a hygienic environment.

Management practices to prevent neonatal bacteraemias

Correct maternal nutrition, control of abortion and the prevention of dystocia are essential to ensure optimal physiological adaptation to extrauterine life. Management practices to further minimise the prevalence of neonatal bacteraemias include:

- Employment of sufficient skilled assistants to monitor all neonatal lambs, to detect and investigate disease and instigate early treatment.
- Installation of good access to lambing pens and good lighting (Fig. 1.150).
- A compact lambing period to maximise the use of labour and minimise the potential for build-up of disease in the lambing environment.
- Maintenance of strict hygiene in lambing accommodation. In the case of housed ewes, it is important that both the lambing courts and individual pens are kept clean, to minimise contamination of ewes' fleeces and udders. Buildings should be well ventilated and drained. Individual pens should be well-lit, easily accessible and ideally cleaned between occupants. Ewe lambs and long-tailed ewes should be dagged prior to entering lambing accommodation. Daily application of paraformaldehyde granules to the bedding can prove to be helpful.
- The stocking rate of housed lambing accommodation should not exceed one ewe per 1.1 m^2 and the area of individual pens should be at least 3.0 m^2. Provision of one individual pen per eight housed ewes is recommended.
- Dipping all lambs' navels in strong iodine solution at birth (and again 4 hours later). Dipping is preferable to spraying with oxytetracycline (Fig. 1.151).

Fig. 1.151 Navel dipping in strong iodine solution.

Fig. 1.152 Stomach tubing with colostrum.

- Ensuring that all lambs receive adequate colostrum within the first 4 hours of life. If in doubt, administer 50 ml/kg of colostrum or colostrum substitute by stomach tube (Fig. 1.152).
- Ensuring that hot water and a full clean lambing kit is easily available.
- Checking all penned lambs regularly for signs of brightness and full stomachs. Navels should be brittle within 36 hours of birth.

Alternatives to fresh colostrum

Ewes frequently have more colostrum than is immediately required by their own lambs. Excess colostrum can be milked, batched and stored or frozen in suitable small quantities. Care is needed not to overheat frozen colostrum during the thawing process.

Cow colostrum, procured in advance of the lambing season and frozen in small containers, is a useful alternative to ewe colostrum. Cow colostrum contains approximately 20 percent less energy per ml than ewes' colostrum, hence correspondingly larger volumes are required. It contains some useful antibodies, but may not protect against specific pathogens found in individual flocks. Clostridial antigens can be boosted by prior immunisation of the cow with an ovine clostridial vaccine. Rarely, cow colostrum contains antibodies against antigens on the lamb's red blood cells, causing severe and usually fatal anaemia when the lamb is between 10 and 20 days old. Laboratory tests are available to screen for anaemia-producing antibodies in cow colostrum. Alternatively, pooling of colostrum from several cows will dilute the effect of any anti-sheep red blood cell antibodies present.

Most proprietary powdered colostrum substitutes are derived from cow colostrum, tested for anaemia-producing antibodies. Some are derived from ewe colostrum and therefore superior. Powdered colostrum is convenient, although cost precludes its widespread use.

Trace element and vitamin deficiencies affecting neonatal lambs

Iodine deficiency

Iodine deficiency is an occasional cause of high perinatal lamb mortality rates in UK flocks, associated with grazing late-pregnant ewes on pastures or crops containing high levels of thiocyanate goitrogens. Thiocyanates act as goitrogens by blocking the uptake of inorganic iodine by the thyroid gland. Their goitrogenic effect is largely overcome by iodine supplementation. In New Zealand, the prevalence of severe iodine deficiency is especially high in Merino flocks and frequently associated with the feeding of goitrogenic brassica crops during late pregnancy. The prevalence of iodine deficiency in the UK is highest in western regions, associated with early pasture growth. Selection of modern pasture cultivars with high concentrations of thiocyanate goitrogen precursors has probably improved their resistance to slug and insect predation, but may have inadvertently resulted in an emerging problem of iodine deficiency in sheep flocks. The most obvious clinical sign of iodine deficiency is goitre in newborn lambs (Fig. 1.153). Lambs with congenital goitre may be pot-bellied in appearance, and have scant wool which lacks crimp (Fig. 1.154). Lambs from the same litter can be affected to different extremes. Animals with severe goitre usually die soon after birth.

Iodine deficiency sometimes results in high perinatal lamb mortality rates in flocks where clinical goitre is not diagnosed. Typically high lamb losses occur during adverse weather conditions due to starvation and hypothermia, or stillbirths where the foetal membranes still cover the lamb's nose. This is due to the role of iodine in thyroid hormones in foetal maturation and thermoregulation.

The diagnosis of iodine deficiency is not straightforward. Severe goitre can often by diagnosed by palpation of the thyroid gland of newborn lambs, and is easily confirmed at gross postmortem examination and by histological examination of the thyroid gland. Production responses, however, have been recorded in flocks with no clinical evidence of goitre. Data collected from several studies around New Zealand indicate that a production response to iodine supplementation is likely when the ratio of thyroid:bodyweights of newborn lambs exceeds 0.4 g thyroid:1 kg body-weight. Careful dissection and accurate weighing of the lamb and its thyroid glands

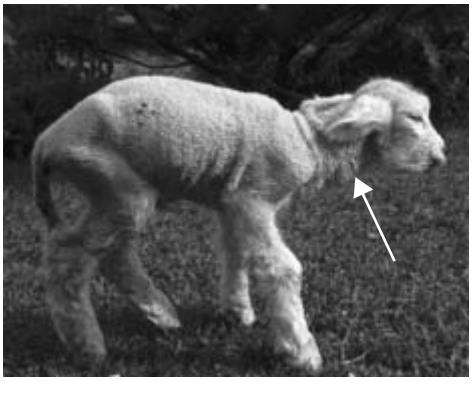


Fig. 1.153 A hypothermic, 2-day-old Romney lamb with palpable goitre (*arrow*).

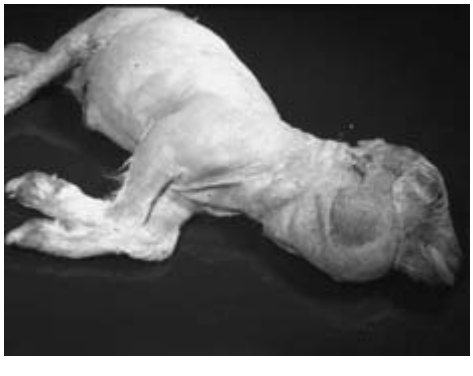


Fig. 1.154 Congenital goitre.

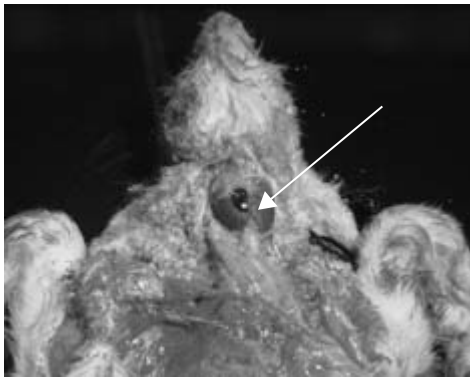


Fig. 1.155 The thyroid glands (*arrow*) of this lamb were not palpably enlarged, but weighed 1.0 g per kg of body-weight, indicating subclinical iodine deficiency.

are required in order to determine such thyroid:bodyweight ratios. Postmortem examination of about 15 lambs usually provides a good indication of the flock iodine status (Fig. 1.155). Unfortunately this information is retrospective, and not necessarily applicable to subsequent years, when environmental conditions and pasture management may not result in iodine deficiency.

Controlled supplementation trials provide accurate information about flock iodine status, but are not always a practical diagnostic method. Serum tri-iodothyronine (T_3) and thyroxine (T_4) concentrations of ewes pre-mating or during pregnancy appear to be unreliable indices of iodine deficiency; reference ranges being based on values in clinically healthy animals grazing farms with no history of goitre. Plasma inorganic iodine (PII) provides a measure of contemporary iodine intake in excess of the animal's requirements. However, reliable reference values have not been established through controlled supplementation trials or growth response trials. PII probably provides a good estimate of iodine sufficiency, but should be used with care for the diagnosis of deficiency. Herbage iodine concentrations also appear to be unhelpful, due in part to the role of goitrogens in inducing deficiency.

Iodine deficiency can be prevented by treating ewes before mating with an intramuscular injection of iodised oil, although such supplementation is expensive and laborious, due to the high viscosity of the product. Supplementation can also be achieved by oral dosing of ewes with 280 mg potassium iodide solution about 8 and 4 weeks before lambing. Oral potassium iodide treatment has also been successfully used for the treatment of clinical goitre. Sustained-release ruminal boluses containing iodine, selenium and cobalt are available. While they provide effective iodine supplementation, in many cases they are wasteful, providing unnecessary or irrational supplementation of cobalt and selenium.

Copper deficiency and swayback

Swayback in young lambs occurs as a consequence of severe copper deficiency in mid to late-pregnant ewes. Most outbreaks of swayback have been seen following mild winters when little supplementary feeding was provided during mid pregnancy. In recent years there have been few confirmed reports of swayback in the UK, partly due to widespread copper supplementation of pregnant ewes and partly due the high level of concentrate feeding in intensively managed flocks.

Example

Poor lambing percentages associated with low twinning rates and high perinatal lamb mortality rates had occurred over a period of several years in a flock of about 1000 New Zealand Romney ewes. Goitre had been diagnosed in the flock about 20 years previously, but serum T_4 concentrations of ewes and lambs had been inconclusive.

A controlled supplementation trial was run over a period of 2 years to investigate the potential role of iodine deficiency. Three weeks before the start of mating, ewes were randomly allocated to groups of 500 supplemented and 500 control animals (Fig. 1.156). Supplementation was by the injection of iodised oil. Supplemented and control ewes were mixed until set stocking, 2 weeks before the start of lambing. The possible confounding factor of different lambing paddocks on perinatal lamb mortality was removed by the reversal of the paddocks used by the supplemented and control ewes during the second year. Ewes were counted into each paddock and ewes and lambs were counted at docking. The lambing paddocks were checked twice daily and all dead lambs were collected and recorded.

Despite different weather and pasture growth patterns, iodine supplementation had a significant positive effect on ewe fertility during both breeding seasons, with a greater proportion of twin-bearing ewes identified in the supplemented groups at ultrasound scanning, 100 days after the introduction of rams.

Scanning results:

	Year 1			Year 2		
	Singles	Twins	Barren	Singles	Twins	Barren
Supplemented	46%	51%	3%	35%	64%	1%
Control	53%	45%	2%	43%	56%	1%

This effect on ewe fertility was confirmed at lambing, by counting of the number of lambs born in each group.

Twinning rates in supplemented and control mixed-aged ewes:

	Year 1	Year 2
Supplemented	65.5%	71.1%
Control	36.8%	56.5%

The greater effect on fertility was recorded following the first breeding season, during which warmer and wetter weather would have influenced pasture growth, composition and goitrogen content. Mild winter conditions favour the growth of white clover which contains high concentrations of thiocyanate precursors, while wet winter weather can result in substantial iodine intake by pregnant ewes along with soil-contaminated pasture.

Fig. 1.156 Large-scale controlled supplementation trials can be useful for the diagnosis of trace element deficiencies in sheep.

During both years of the trial, survival of newborn lambs was enhanced in the supplemented group when compared with the control group. Most lamb deaths were associated with starvation.

Perinatal lamb mortality rates:

	Year 1	Year 2
Supplemented	9.9%	6.7%
Control	23.5%	8.9%

This effect was greatest in the first year when the weather conditions during the lambing period were appalling. In the second year, the perinatal lamb mortality rate was low in both control and supplemented groups, reflecting favourable lambing conditions. Iodine-deficient lambs have a low metabolic rate and impaired suckling behaviour. During cold and wet seasons, many of these lambs die from starvation and hypothermia, but when conditions are favourable and shepherding is good, many survive.

This case illustrates some of the complexities associated with the prediction of trace element deficiencies. Nevertheless, the annual cost associated with iodine deficiency in this flock was conservatively estimated at £2.00 per ewe, considerably more than the annual cost of iodine supplementation.

Congenital swayback is characterised by stillbirths and the birth of small and weak lambs, which may show fine tremors of the head. Less severely affected lambs are bright, but incoordinated (Fig. 1.157) with characteristic weakness of the pelvic limbs (Fig. 1.158), which results in a swaying or stumbling gait. These lambs are often fine boned and dull coated. A delayed form of the disease with slow, progressive weakness and muscle atrophy of the pelvic limbs is occasionally seen in older lambs, sometimes initiated by gathering or handling.

Copper is an essential component of several biologically important metalloenzymes (Table 1.6). It is difficult to define which role is first to become rate limiting in copper deficient animals, but the most obvious sign of copper deficiency is usually swayback in young lambs. The other clinical signs associated with copper deficiency in sheep, which may be present in swayback-affected flocks, are:

Fig. 1.157 A wide-based stance and low head carriage, associated with incoordination due to congenital swayback.

Fig. 1.158 It can prove difficult to clinically differentiate swayback from thoracolumbar spinal cord lesions, for example due to abscessation or *Sarcocystis* spp. infestation.

Table 1.6 Functions of copper enzymes and disease syndromes associated with deficiency.

Copper enzyme	Function	Disease associated with deficiency
Superoxide dismutase	Free radical scavenging, phagocytosis, neutrophil chemotaxis	Enzootic ataxia, subclinical disease
Ceruloplasmin (ferroxidase)	Iron metabolism	Anaemia
Cytochrome oxidase	Phospholipid synthesis	Demyelination/enzootic ataxia
Lysyl oxidase	Collagen cross-linkages	Skeletal abnormalities
Dopamine-b-hydroxylase	Neurotransmission	Enzootic ataxia
Polyphenyloxidase	Melanin production	Wool/hair pigmentation

Localised depletion of copper in the intestinal mucosa may influence digestion, gut motility and immunity to gastrointestinal parasitism.

- osteoporosis and bone fractures in young lambs
- defects in connective tissue which may lead to tendon abnormalities, resulting in stiffness
- depigmentation of coloured wool
- loss of wool crimp, distortion of wool fibres and reduction in wool quality, described as 'steely wool' (Fig. 1.159)
- anaemia, associated with prolonged deficiency.

Copper-deficient lambs may be more susceptible to neonatal disease than copper-sufficient lambs, but ill thrift is not a consistent feature of copper deficiency in sheep.

The diagnosis of swayback is based on identification of clinical signs and flock history of copper deficiency. However some of the clinical signs associated with spinal abscessation or border disease are similar, so liver copper assay and brain or spinal cord histopathology are often needed to support the diagnosis.

Most cases of copper deficiency in sheep result from high dietary levels of interfering factors such as molybdenum and sulphur, which form complexes with copper in the forestomach and prevent its absorption. Copper deficiency is sometimes seen following pasture improvement, because raising the pH of soil can make molybdenum more available. Forage crops such as kale have a high sulphur content, and may be implicated in cases of copper deficiency. The requirement for copper is also dependent on the time of year. Pasture copper levels vary little throughout the year,

Fig. 1.159 White wool discolouration and loss of crimp in a copper-deficient Scottish Blackface ewe.

but the levels of interfering factors do. On peat and heavy clay soils, the level of molybdenum often increases throughout the winter and is highest during spring, associated with the seasonal rise in the water table.

Copper absorption and retention are also influenced by the breed of sheep and nature of the feed. Texel and Suffolk sheep retain copper efficiently, while Scottish Blackfaces retain copper relatively inefficiently and are more susceptible to deficiency disease. Copper is well absorbed from feeds which are low in fibre, such as grains and brassicas, but poorly absorbed from pasture. High iron intake associated with pasture soil contamination during wet winter conditions has been shown to deplete liver copper reserves, due to an effect on small intestinal copper absorption. During wet winters, soil consumption can account for up to 10 percent of the total dry matter intake in sheep, with a marked effect on copper absorption. Copper availability is, therefore, frequently lowest in late winter and spring, at the time of year when the animal requirements are highest (late pregnancy and during early lamb growth).

Lambs are not usually born with higher copper reserves than their dams, but their copper absorption is extremely efficient when compared to that of their dams.

The need to supplement copper during mid pregnancy for the prevention of swayback is clear-cut in many hill Scottish Blackface flocks, based on a history of copper deficiency problems. However, supplementation is often unnecessary in lowland flocks and is potentially dangerous in breeds such as the Suffolk and Texel. In these cases, the diagnosis of copper sufficiency relies on appropriate sample collection.

Soil and pasture copper concentrations alone are poor indices of deficiency, because most copper deficiency is induced by interfering factors. The interpretation of blood and liver copper concentrations is based on the following principles:

- Copper is absorbed from the small intestine. Absorbed copper becomes albumin/amino acid bound in the blood and is then transported to the liver. In the liver, it is incorporated into copper-containing proteins such as ferroxidase or stored with other proteins such as copper metallothionines. Copper readily crosses the placenta, but only very small amounts are secreted in milk.
- Absorbed copper in excess of requirements is efficiently stored in the liver, which contains up to 70 percent of the total body copper reserves.
- Copper is transported from the liver to the tissues, where it is required by a variety of different enzymes.
- Blood copper concentrations, mostly in the form of ferroxidase, are maintained for as long as the liver stores are adequate. Animals are described as replete when the liver copper reserves are good.
- When net copper absorption is insufficient to meet metabolic requirements, liver stores are mobilised to meet the shortfall.
- The blood copper concentration remains constant until the liver becomes depleted.
- Blood copper concentrations fall only when the liver reserves are already depleted and metabolic requirements continue to exceed net absorption.
- The concentrations of copper enzymes at the essential sites remain constant for as long as blood levels are maintained.
- Clinical deficiency only occurs when the levels of copper at the essential sites fall.

The choice of diagnostic samples, therefore, depends on the reason for sampling. For the diagnosis of disease, both blood (serum or plasma) and liver are appropriate. Serum levels are 10 to 20 percent lower than plasma levels. However, liver is the only

useful sample when the reason for sampling is to determine whether reserves are adequate to last throughout the winter.

Laboratory reference ranges for liver and serum copper concentrations are based on the results of growth response trials from around the world, examining the performance of animals at different dietary copper levels. These reference ranges are relevant to the diagnosis of clinical disease and to determine whether the animals are likely to respond to immediate copper supplementation or not. However, they provide no indication of the sufficiency of liver copper stores to prevent deficiency occurring, or to monitor the effectiveness of a supplementation programme. There are no definite guidelines to answer these questions, so to a large extent, such judgement relies on personal experience. When sampling is undertaken to determine if copper deficiency is likely to occur, the results need to be compared with values higher than the reference values. When samples are collected to monitor a supplementation regimen, it is necessary to compare pre- and post-supplementation values, taking into account what is expected of the particular supplement. Ideally the same animals should be followed, because individual animal variation in copper concentration is large, especially when the values are in the adequate range.

The correct number of samples is essential. There is a large variation in copper concentrations between individual animals. Seven blood or three liver samples are required, because the reference values are based on these numbers. Some animals with good liver copper stores consistently have low serum copper levels. Caution is therefore necessary in interpreting a group of serum values where just one is low. The interpretation must be based on the mean value, and not on individual values, for the same reason.

Liver samples can be obtained during postmortem examination of casualty animals or from slaughterhouse material. While slaughterhouse samples can be useful for monitoring purposes, they are of limited value for the diagnosis of clinical deficiency, because copper-depleted animals are unlikely to reach slaughter weights. In New Zealand, where flock sizes are large and even small production losses due to trace element deficiencies are economically important, liver samples are routinely collected by biopsy as the basis of a rational scientific approach to determine any need for supplementation. However, sheep liver biopsies are not routinely performed in the UK.

Superoxide dismutase (SOD) is a copper-containing enzyme whose function declines with lower copper states. In theory, analysis of red blood cell or liver SOD should provide a useful diagnosis of deficiency and dysfunction states. However, cost and problems with analysis have hindered its use.

Copper supplementation

While the indiscriminate use of expensive ad-lib free-access minerals, mineralised drenches and other forms of supplementation may provide some insurance against production losses from copper deficiency, it is sometimes wasteful and can be associated with toxicity problems. There are no recipes for trace element supplementation, so a detailed evaluation of each individual situation is required to determine the most appropriate programme. The most effective supplementation method depends on soil type, stocking rates, handling facilities and the availability of labour.

Oral drenching with copper salts

The amount of copper that can be administered orally as copper salts is limited by the potential for toxicity, therefore, a single oral drench with a safe amount of copper

salts only provides a short-term boost to body copper stores. Swayback can be prevented in some flocks by oral dosing of ewes with copper sulphate solutions 8 and 4 weeks before lambing. While copper salts are frequently added to anthelmintic drenches, the copper content and availability from these products is low, so they are not necessarily useful for the prevention or treatment of dietary-induced copper deficiency.

It has been suggested that copper chelates have a much greater rate of absorption from the gut than copper sulphate. Their use has been advocated on this basis. However, there is little evidence to support this claim of greater absorption and they are considerably more expensive than copper sulphate.

Free access minerals

Dietary mineral supplements can be useful where there is an opportunity for precise formulation of total fed requirements, but their ad hoc usage is potentially problematic, due to variable intakes. Because of the risk of toxicity associated with variable intake, the amounts of copper which can be included in free access minerals is small and the majority of animals can remain inadequately supplemented.

Copper injections

The parenteral injection of chelated copper supplies relatively small amounts of copper and is a convenient method of supplementation for a period of 2 to 4 months. In many flocks, where the period of deficiency is only for a few months, copper injection of ewes during mid pregnancy provides an efficient method for the prevention of swayback.

Copper capsules

On most farms during most seasons, the oral administration of capsules, which contain particles of copper oxide wire, provides an effective long-term supplementation option. The capsules quickly dissolve in the rumen and release the copper oxide particles, which gradually move to the abomasum, where they are retained. Ionised copper is released within the low pH environment of the abomasum and subsequently absorbed from the small intestine. Release of ionised copper occurs over a 3- to 4-week period, which enables efficient storage in the liver, without the risk of acute toxicity. Elevations in liver copper levels can persist for a period of almost 300 days. Copper oxide capsules are often given to weaned ewe lambs on severely deficient hill farms.

Unlike oral copper sulphate supplements, copper oxide particles have the advantage that they are not exposed to the interfering effects of molybdenum and sulphur in the rumen. However, high iron intakes, through the ingestion of soil during wet and muddy seasons, can interfere with absorption and probably account for the occasional failure of copper capsules to elevate liver copper stores. Severe gastrointestinal parasitism may also reduce the efficacy of copper oxide particles due to accelerated flow of digesta through the abomasum.

Pasture top-dressing

Effective copper supplementation can be achieved on some farms by regular top-dressing of pasture, but this method has lost favour during recent years due to fluctuations in the cost of copper sulphate. Careful stock management is required. It is important to keep stock off top-dressed pasture for 3 weeks, or until after heavy rain,

so that any copper which is adherent to pasture is cleared and the risk of toxicity avoided. Unweaned lambs should not be grazed on recently top-dressed pasture because of their higher absorption efficiency and the risk of toxicity.

Sustained-release ruminal boluses

Sustained-release ruminal boluses, which contain copper, cobalt, selenium and iodine along with other minerals and vitamins, can provide a useful supplementation method in some flocks. These boluses provide a sustained and constant copper supplementation for about 240 days, but on some farms the daily release of copper may be insufficient for times of peak requirements. The boluses are relatively expensive, and supplementation with the other trace elements included may be unnecessary.

Excessive copper supplementation is potentially toxic to all breeds of sheep. As a general rule, sheep should never be provided with more than one source of supplementary copper. Lambs should not be supplemented before weaning, because of their efficient copper absorption. Furthermore, animals should not be supplemented shortly before they are housed and provided with an improved dietary supply of available copper with concentrate feed.

Congenital white muscle disease

Historically, nutritional muscular dystrophy was an important, common selenium-responsive cause of perinatal lamb mortality in sheep flocks. The disease is now uncommon, due to awareness of selenium deficiency and widespread supplementation.

In lambs, the disease is congenital, or delayed in onset. Congenital disease results in stillbirths, or the birth of weak lambs, which fail to feed and die from starvation. The delayed-onset disease has also been reported in lambs after docking. Clinical signs appear suddenly and are usually precipitated by exercise and stress, such as docking, bad weather or transport. Affected animals show sudden-onset semi-flaccid paralysis, but appear stiff or unable to stand. Lambs show difficulty in sucking due to pharyngeal paralysis, so may die from starvation/hypothermia. Some lambs are found dead as a result of respiratory failure. These clinical signs are a result of severe non-inflammatory degeneration and necrosis of skeletal and cardiac muscle. Surviving animals are ill thrifty.

Congenital white muscle disease is prevented by selenium and vitamin E supplementation of ewes at least 6 weeks before lambing.

Congenital malformations of lambs

A very low incidence of congenital lamb deformities such as limb malformations (Fig. 1.160), defects of the urinary tract (Fig. 1.161), genital abnormalities, brachygnathia inferior (undershot mandible/lengthening of the maxilla), prognathia (overshot mandible), persistent urachus, umbilical herniation (Fig. 1.162), atresia ani (Fig. 1.163), eye abnormalities, micrencephaly, schistosoma reflexa (Fig. 1.164) and spina bifida (Fig. 1.165) is seen in most UK flocks, caused by random genetic mutations and developmental anomalies. Lambs with potentially fatal malformations are sometimes born alive and require euthanasia immediately after birth (Fig. 1.166). Some malformations such as undershot jaw and genital abnormalities are not fatal, but result in ill thrift in affected animals.

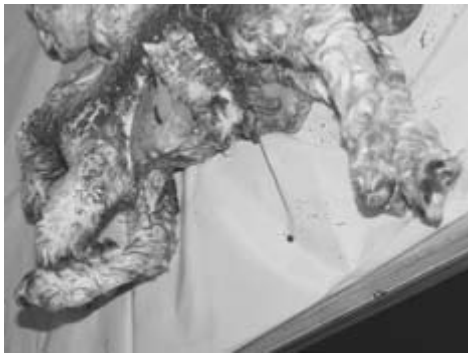


Fig. 1.160 Limb deformities such as arthrogryposis are seen sporadically in many flocks, but occasionally arise as outbreaks, suggesting a genetic, toxic or infectious aetiology.

Fig. 1.161 Incomplete development of the perineal and penile urethra, splitting the scrotum of a male lamb and giving rise to a condition referred to as pseudohermaphroditism, or more correctly hypospadia. Affected lambs often grow normally, but are predisposed to flystrike because their perineal and ventral fleece becomes saturated with urine that dribbles from the open urethra.

Fig. 1.162 Herniation of the small intestines through the umbilicus. Mild cases such as this often respond well to surgical correction under general anaesthesia.

Fig. 1.163 The massively distended abdomen of a 10-day-old lamb with atresia ani.

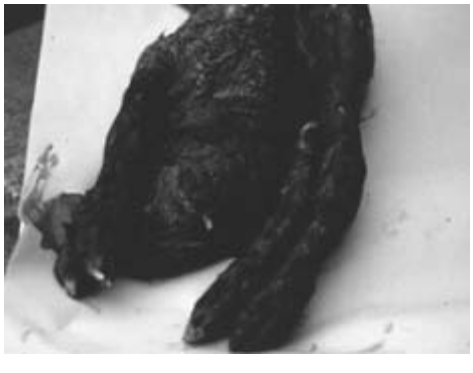


Fig. 1.164 Foetal abnormalities, whereby the head and all four limbs project forwards from an undifferentiated plate of bone and viscera, are referred to as schistosoma reflexa, and occur sporadically.

Fig. 1.165 Spina bifida is a congenital neural tube deficit. Typical signs include pelvic limb paralysis, with a clearly defined swelling and wool-free area of skin in the lumbosacral region of the dorsal midline. The middle of the tail is characteristically deviated dorsally.

Fig. 1.166 Aprosopia, a severe state of agnathia or facelessness, in a New Zealand Romney lamb. This lamb was born alive and required immediate euthanasia.

Surgical correction of atresia ani

Cases of atresia ani where the position of the anus is apparent, because of pressure in the rectum beneath, respond well to surgical correction. Surgery involves making a simple cross-shaped skin incision over the bulging rectum, where the anus should have been. In some cases, the distal rectum is also absent and corrective surgery is not possible.

Economically important outbreaks of congenital malformations associated with foetal exposure to viruses, such as border disease virus, or toxins, or hereditary genetic diseases are extremely rare in UK flocks. In Asia and parts of Australia infection of ewes during late pregnancy with Akabane virus, transmitted by mosquitoes or midges, results in the birth of lambs with hydrancephaly, arthrogryposis and spinal cord defects. The mosquito-transmitted Cache Valley virus sporadically causes similar problems in North America. Numerous plant species including lupins, hemlock and tree tobacco, which are found overseas in range grazing systems, are potentially teratogenic. In North America, false hellebore (*Veratrum californicum*) is a common cause of congenital skeletal abnormalities including arthrogryposis, scoliosis, lordosis, torticollis and ribcage deformities.

Congential malformations of lambs may be associated with genetic disease resulting from unfavourable recombinations and expression of abnormal genes. Most genetic diseases are recessive, or show incomplete penetrance in their expression and are affected by other genetic, phenotypic and environmental influences. Genetic disease is insidious and may be present in large numbers of animals, and therefore difficult to control, before clinical signs are first recognised. While congenital genetic diseases of sheep are relatively uncommon in the UK, they are potentially important, especially in pedigree flocks where they can downgrade previously valuable stock lines. Some congenital genetic diseases of sheep both are listed in Table 1.7. Many of these have not been reported in the UK, but could nevertheless be present.

Some of these diseases are immediately fatal, while in others the onset of clinical signs is delayed. For example, in cases of ceroid lipofuscinosis, the clinical signs of retinal degeneration may not appear until 6 months of age.

It can prove difficult to conclusively establish a hereditary genetic cause of congenital malformations. Carrier rams can sometimes be identified by test mating to

Table 1.7 Reported, proven genetic diseases of sheep.

Genetic disease	Sheep breed affected
Collagen dysplasia	New Zealand Romney, Perendale, Finnish Landrace and Dala[1]
Epidermolysis bullosa	New Zealand Romney,[2] Dorset Down, Scottish Blackface and Welsh Mountain
Osteogenesis imperfecta	New Zealand Romney
Goitre	New Zealand Romney
Gangliosidosis	Corriedale
Mesangiocapillary glomerulonephritis	Finnish Landrace
Multicystic kidneys	Bluefaced Leicester
Arthrogryposis	Suffolk
Hereditary chondrodysplasia (spider syndrome)	Suffolk and Hampshire
Inherited chondrodysplasia (dwarfism)	Texel
Lethal biochemical disorders in albinoid grey Karakul sheep	Karakul and Mongolian
Dandy Walker malformation[3]	Suffolk
Agenesis of the corpus callosum and hippocampus[3]	Southdown
Cerebellar cortical atrophy	Drysdale
Ceroid lipofuscinosis	Hampshire and Rambouillet
Distal axonopathy	Dorset Down
Neuroaxonal dystrophy	Coopworth and Perendale
Spina bifida	New Zealand Romney and Icelandic
Bilateral cataract	New Zealand Romney
Congenital microphthalmia	Texel
Spongiform leucoencephalomyelopathy	New Zealand Romney and Polled Dorset
Holoprosencephaly	Border Leicester

[1] Different biochemical pathways may be involved in different breeds.
[2] The disproportionate inclusion of the New Zealand Romney in this list reflects a culture of investigation, rather than hiding of sheep disease problems in a major sheep-producing country, and not an inherent problem with the breed.
[3] Possibly the same condition.

sufficient numbers of their daughters, although this approach is seldom practical. Alternatively, biochemical or molecular markers can be developed for some of these conditions, enabling control using screening tests.

Mesangiocapillary glomerulonephritis of Finnish Landrace sheep

Mesangiocapillary glomerulitis is a very rare disease which occurs primarily in pure-bred Finnish Landrace lambs less than 4 months old. The disease is associated with deposition of immunoglobulins and complement on the glomerular capillary walls. While lesions develop in utero, lambs with mesangiocapillary glomerulitis are clinically normal at birth, but stop sucking after a few weeks. Affected lambs are anorexic, dull and afebrile, and appear blind, with fine muscle tremors due to secondary encephalopathy, and show signs of crouching and tail swishing, indicative of abdominal pain. Kidneys are enlarged and tender on palpation of the abdomen. Most affected lambs die within a few weeks, although some survive to become adults.

The diagnosis of mesangiocapillary glomerulitis is supported by ultrasonographic identification of enlarged kidneys and determination of azotaemia and hypoalbuminaemia. On postmortem examination the kidneys are enlarged to six times their normal size with red or yellow spots in the cortex, indicative of haemorrhagic glomeruli.

Inheritance of mesangiocapillary glomerulitis is complex and is probably polygenic and dominant, with incomplete penetrance. Defects may be latent in females and only expressed when carrier ewes are mated with a carrier ram. Both affected and unaffected lambs may be born together in a litter, while carrier ewes do not necessarily give birth to affected lambs after consecutive matings with a transmitting sire. Control is straightforward, involving culling the sires of the affected lambs and culling all ewes giving birth to affected lambs.

Epidermolysis bullosa

Epidermolysis bullosa (redfoot) is a rare inherited skin disease which has been reported in Suffolk, Dorset Down, Welsh Mountain and Scottish Blackface sheep. The disease is characterised by ulceration and shedding of the mucous membranes of the gums, dorsal surface of the tongue and lips, resulting in weight loss associated with failure to feed. In some cases, ulcerative skin lesions occur on the pinnae of the ears, the external nares and over the distal limbs. The hoof horn of one or more digits may be partially separated, or completely shed, resulting in severe lameness. Affected lambs often appear normal, with oral lesions first appearing at 1 to 2 days old, and separation of the hoof horn by 3 weeks of age. Surviving lambs are usually emaciated at this stage.

The disease results from sub-epidermal bulla formation and a reduction in the number of hair follicles and sebaceous glands. The stratified squamous epithelium is poorly attached to the underlying corium and therefore liable to be shed.

Osteogenesis imperfecta

Osteogenesis imperfecta is a similar inherited genetic disease to epidermolysis bullosa, which has been reported in New Zealand Romney sheep. Osteogenesis imperfecta, which has probably been eradicated, was inherited as an autosomal dominant trait and was considered to be a new mutation in the germ cells of one ram. Affected lambs were born with multiple bone fractures. Long bones were soft, having thickened diaphyses and an almost complete absence of a medullary cavity. Affected lambs consistently showed moderate brachygnathia inferior, joint laxity, dark blue coloured sclera and small pink teeth.

The disease, which is also sporadically reported in cattle, was caused by a defect in collagen production by fibroblasts.

Dandy Walker malformation

Dandy Walker malformation occurs in several mammalian species including cattle and humans. The disease was confirmed in pedigree Suffolk lambs in Scotland in 1977, and is now thought to be widespread within the breed in the UK, although few cases are reported. The disease is characterised by doming of the skull due to severe hydrocephalus, absence of the septum pellucidum, cystic enlargement of the roof of the posterior aspect of the fourth ventricle and agenesis or hypoplasia of the cerebellar vermis.

Affected lambs have markedly domed and fragile skulls. Most are stillborn or die soon after birth, and most cases require assistance for dystocia. One or more lambs are affected in multiple litters.

Cases are usually first seen following the introduction of a new ram, sometimes involving up to a quarter of his progeny. The problem is managed but not eradicated by the identification and culling of carrier rams.

Hereditary chondrodysplasia

Hereditary chondrodysplasia (spider syndrome) is an autosomal recessive genetic disease of Suffolk and Hampshire sheep which has been reported in North America, Australia and New Zealand. The disease was introduced to Australia and New Zealand following the importation of North American bloodlines. The presence of the disease in the Hampshire breed may be associated with previous crossing with Suffolk sheep. Similar syndromes have been seen in North Country Cheviot and Border Leicester sheep in the UK, although their genetic basis has not been investigated.

Hereditary chondrodysplasia is characterised by birth of lambs with elongated, fine long bones, minimal muscling of the limbs and varying degrees of skeletal deformity. The most obvious deformity is pronounced medial deviation of carpus and hock joints associated with irregular thickening of the distal growth plate cartilages of the radius and ulna, giving the appearance of outward bending of the thoracic limbs from the carpi. Deformities in the pelvic limbs are generally less pronounced. Deformities are also present in the skull, producing a 'Roman nose' appearance and narrowed elongation of the occipital condyles; in the vertebrae causing kyphosis (dorsal curvature of the spine); in the sternebrae, leading to a flattening of the sternum; and in the ribs, causing a pronounced angle at the costo-chondral junctions.

In most cases, lethal abnormalities are present at birth, but clinical signs in some lambs are not apparent until 4 to 6 weeks of age, leading to debilitation and inability to support their own weight. A few affected sheep survive until adulthood, but ram lambs have testicular hypoplasia, while ewe lambs are mostly infertile.

The diagnosis of spider syndrome is based on the clinical signs and supported by identification of radiographical abnormalities such as multiple, irregular centres of ossification in the anconeal process of the ulna, vertebrae, sternebrae and pelvic bones. Postmortem examination reveals excess cartilage in the skull, sternum, vertebrae, elbow, and physes of the long bones.

A molecular test has been developed to identify sheep carrying the spider syndrome gene, which has been used in New Zealand to control the disease.

Arthrogryposis of Suffolk lambs

Arthrogryposis, characterised by bilateral flexion rigidity of the metacarpophalangeal and carpal joints, occurs following exposure to various teratogens. The condition has also been demonstrated as an autosomal recessive genetic disease of UK Suffolk sheep.

Cerebellar abiotrophy

Widespread degeneration of Purkinje cells in the cerebellum, referred to as cerebellar abiotrophy, is a familial syndrome which has been described in UK Charollais sheep. The mode of inheritance has not been established but is likely to be an autosomal recessive condition.

Fig. 1.167 Inability to stand due to an inherited lower motor neurone disease in a New Zealand Romney lamb.

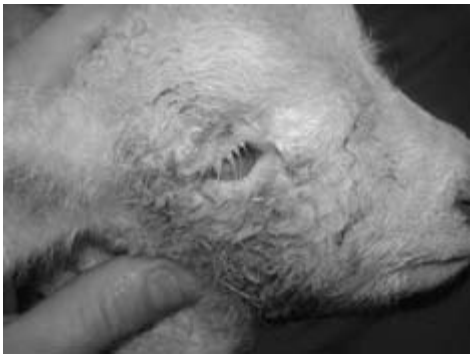


Fig. 1.168 Tear staining of the side of the face associated with severe corneal irritation due to inturning of the lower eyelid.

Clinical signs including a wide-based stance with lowered head carriage, intention tremors with jerky movements of the head and ataxia may be present from birth, but more commonly appear from 4 to 8 weeks old. The disease is slowly progressive.

Lower motor neurone disease in New Zealand Romney lambs

Degeneration and loss of neurones in the ventral horns of the spinal cord and brainstem, Wallerian degeneration of motor nerves and denervation atrophy of skeletal muscle fires has been reported as a genetic disease in New Zealand Romney sheep. Affected lambs typically appear normal at birth and for the first week of life, but then develop clinical signs of weakness and ataxia, which progress until they are unable to stand, at about 3 weeks old (Fig. 1.167).

Entropion

Entropion is a common congenital disorder, which is characterised by turning in of one or both lower eyelids. The condition is seen in most breeds of UK sheep and is probably inherited, but the nature of the inheritance is unknown. Inturned hairs of the lower eyelid rub on the cornea and cause severe irritation (Fig. 1.168). The condition is painful and affected eyes appear half-closed and watery. Some cases spontaneously recover, but in most lambs, unless treated, the cornea becomes cloudy and ulcerated, leading to permanent blindness.

Mildly affected cases often respond to manual eversion of the lower eyelid (Fig. 1.169). Lambs should be carefully monitored afterwards and the procedure may need to be repeated. More severely affected eyes are usually treated by injection of about 1 ml of penicillin under the eyelid or insertion of metal (Michel) clips below the eyelid, to draw the eyelid outwards. Severe or persistent cases sometimes require local analgesia and surgical removal of a strip of skin from under the eyelid (Fig. 1.170).

Lamb losses between 1 week old and weaning

The majority of lambs which die between 1 week old and weaning do so as a result of disease acquired during the perinatal period. However, significant losses sometimes

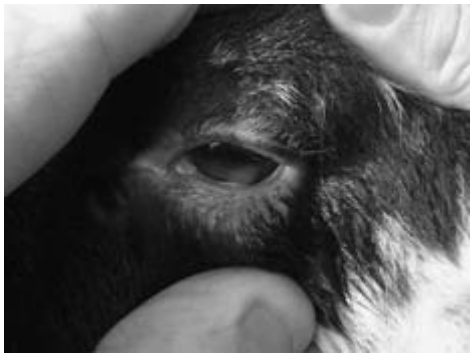


Fig. 1.169 Some cases of entropion respond to manual eversion of the lower eyelid.

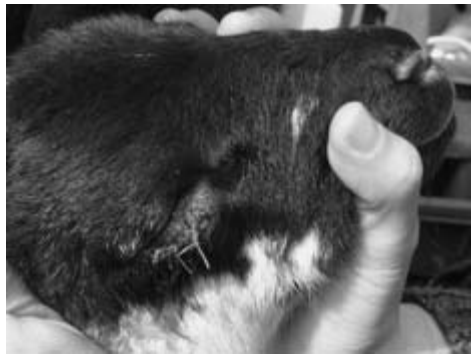


Fig. 1.170 Surgical removal of a strip of skin under the eyelid, in a persistent case of entropion which had not responded to injection of penicillin under the eyelid.

occur due to specific diseases such pasteurellosis, *Erysipelothrix rhusiopathiae*, nephrosis, coccidiosis and nematodirosis which are acquired after 1 week of age.

Significant losses occasionally occur due to exposure. Healthy lambs can withstand severe weather, although blizzard conditions can overcome this resilience. Losses to exposure are particularly distressing, as in many cases there is little that can be done to prevent them. Provision of shelter and use of safe areas can help, but requires considerable skill to provide: many sheep have been lost while seeking shelter in areas subsequently filled by snow drifts.

Erysipelothrix rhusiopathiae polyarthritis

Erysipelothrix rhusiopathiae causes fibrinous polyarthritis in lambs between 2 weeks and about 6 months of age. While disease outbreaks are uncommon, the morbidity rate sometimes reaches 40 percent of lambs in affected groups. Affected lambs initially appear stiff and are pyrexic, leading to lameness in one or more limbs. Affected joints are not initially swollen, but are painful on gentle palpation. Joints become markedly swollen in chronic cases and affected lambs are ill thrifty.

E. rhusiopathiae is an occasional cause of disease in a wide range of mammalian and avian hosts. Outbreaks of disease in sheep are usually associated with exposure of abrasions on the limbs, in the mouth or on the navels of young lambs to contaminated organic material in the soil. The incidence of the disease is generally highest in outdoor lambing flocks following wet springs. In older lambs, castration and docking wounds have been reported as routes of infection. The bacterium survives for long periods in the soil and the main source of contamination is probably other sheep, although pigs and birds have been implicated in some cases.

Bacteraemia follows infection, enabling the bacteria to settle in the joints, causing polyarthritis within 2 to 4 days. Bacteria occasionally lodge in the valves of the heart causing endocarditis. In the acute stages of the disease, joint fluid is turbid, associated with a predominantly fibrinous inflammatory response. The articular surfaces become covered by sheets of fibrin, but articular erosions are notably absent (Fig. 1.171). In chronic cases, there is marked thickening of the joint capsule due to fibrosis and oedema.

The diagnosis of *E. rhusiopathiae* polyarthritis is based on the history of exposure to a potentially contaminated environment, clinical signs and high morbidity rate. Bacterial culture following aspiration of joint fluid is usually unrewarding, but the diagnosis can be supported by use of a slide agglutination test to detect antibodies in joint fluid.

While some lambs recover spontaneously, prompt treatment is necessary to prevent suffering and ill thrift associated with chronic cases. In the acute stages of the disease, treatment with a 3-day course of penicillin injections is usually effective.

Disease control depends on management to prevent direct or indirect soil contamination of wounds. In flocks with a recognised perennial problem, vaccination of ewes can be used to provide passive protection to their lambs. Two doses of a formalin killed vaccine, 4 to 6 weeks apart in the first year, and a single booster dose 3 to 4 weeks before lambing in subsequent years, can be given at the same time as clostridial disease vaccines.

Septicaemic pasteurellosis

Septicaemic disease is a rare cause of respiratory disease and sudden death in young, housed lambs, between about 6 and 12 weeks of age. Morbidity rates during disease outbreaks sometimes reach 75 percent. Affected lambs appear restless, some remaining bright, but having serous nasal and ocular discharges, tachypnoea (>60 breaths per minute) and tucked-up abdomens, while other lambs are dull, pyrexic (rectal temperatures >40°C), lethargic and easily caught, with laboured respiration, injected and cyanotic mucous membranes, and severe dyspnoea. On auscultation of the chest, adventitious lung sounds are audible throughout the entire lung fields of some lambs, while no adventitious sounds are heard in others. Group mean lamb daily liveweight gains may fall from 0.5 kg/day to zero. Mortality rates in affected flocks commonly reach 5 percent.

On postmortem examination, there may be severe, extensive, fibrinous pericarditis, pleurisy and peritonitis (Fig. 1.172) with variable degrees of purple-coloured consolidation and necrosis of the cranial and ventral lung areas. Carcase lymph nodes are enlarged and haemorrhagic and petechial haemorrhages are present in the

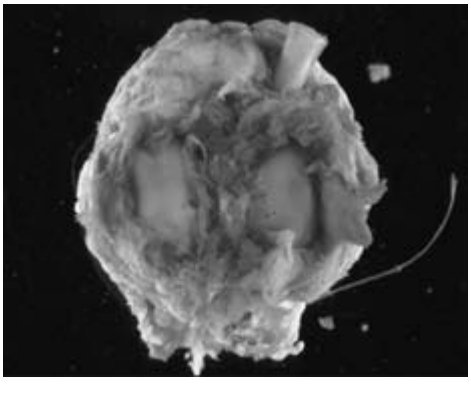


Fig. 1.171 Thickening of the joint capsule and fibrinous arthritis due to *E. rhusiopathiae* infection.

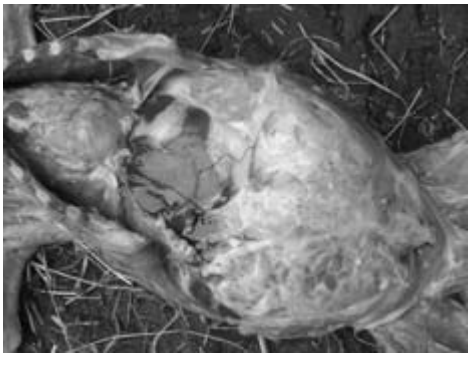


Fig. 1.172 Fibrinous peritonitis, pericarditis and pleurisy due to septicaemic pasteurellosis in a 3-week-old lamb.

myocardium and kidneys. Abomasitis, characterised by thickening and hyperaemia of the abomasal wall and small shallow areas of abomasal ulceration, is sometimes seen, and livers sometimes appear pale and fatty.

Septicaemic pasteurellosis is caused by various serotypes of *M. haemolytica*, which are common commensal bacteria in the nasopharynx of healthy lambs. Serotype 2 accounts for about 34 percent of confirmed septicaemic pasteurellosis cases in the UK. Secondary infection with other respiratory tract pathogens such as Parainfluenza 3 virus, *Mycoplasma* spp. and *Chlamydia* spp. may also be involved. Untreated, surviving cases may become subacute or chronic, leading to subsequent pneumonic pasteurellosis.

A provisional diagnosis of septicaemic pasteurellosis is based on the disease history, clinical signs and postmortem findings, although these vary in severity. Confirmation of the diagnosis depends on the histological identification of pulmonary lesions containing oat cells, and the bacteriological culture of more than 10^6 *M. haemolytica* colony-forming units per gram of lung tissue, although this is not always feasible in field investigations. Most disease outbreaks occur in housed lambs, associated with stressful conditions such as prolonged cold and humid weather.

Vaccination of ewes, even with modern vaccines based on protein antigens that are involved in iron uptake, sometimes fails to prevent disease in their lambs, because passive antibody transfer does not provide lasting satisfactory protection against all serotypes of *M. haemolytica* beyond 1 month old. In the face of an outbreak, treatment of the whole group of lambs with intramuscular long-acting oxytetracycline at a dose rate of 20 mg/kg has been shown to be successful in preventing further cases, improving creep feed intake and restoring daily liveweight gains to previous levels.

Nephrosis

Nephrosis is a sporadic cause of death in 2- to 12-week-old lambs, associated with non-inflammatory degeneration of the renal tubules. In some parts of northern England, annual losses of up to 3 per cent have been reported. The initial clinical signs are dullness, diarrhoea and a staggering gait. Affected lambs do not suck and lose weight (Fig. 1.173), invariably leading to death after a period of 2 to 10 days. Affected lambs are azotaemic, with increased total proteins and a decreased albumin:globulin ratio. Nephrotic lambs develop metabolic acidosis with exchange of intracellular potassium into the circulation. Death is often due to heart failure associated with hyperkalaemia.

On postmortem examination, the kidneys are enlarged, soft and pale, with expanded cortices (Fig. 1.174). On histological examination, groups of cortical tubules are distended with casts of serum protein-like or fibrin-like material, and lined with undifferentiated, low cuboidal cells. Ultrastructural studies have shown evidence of severe toxic effects in proximal convoluted tubules and, to a lesser extent, in some glomeruli.

No putative nephrotoxic factor has been identified and the cause and risk factors for nephrosis are unknown. The disease invariably occurs in lambs at pasture, but no breed or sex predisposition, or association with management practices such as feeding or access to mineral supplements, has been identified. Concurrent cryptosporidiosis, nematodirosis or coccidiosis is often diagnosed, but nephrosis is also seen in flocks

Fig. 1.173 A dull, emaciated nephrotic lamb.

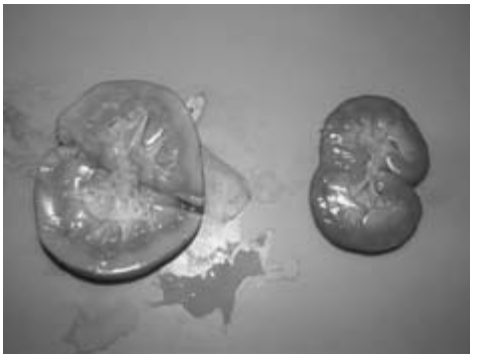


Fig. 1.174 A nephrotic kidney (*left*) and normal kidney (*right*) from a similar aged lamb for comparison.

with no evidence of these diseases. The response to supportive therapy in affected lambs is poor and, until the identity of the nephrotoxic factors is known, it is inappropriate to recommend any preventive strategy.

Coccidiosis

Coccidiosis in lambs is caused by two species of sheep-specific protozoan parasites, *Eimeria crandallis* and *Eimeria ovinoidalis*. Following the ingestion of oocysts from a contaminated environment, the parasite invades, multiplies in, and disrupts cells of the intestinal wall. After a period of 2 to 3 weeks oocysts are shed in the faeces, further contaminating the environment. Under suitable damp conditions many oocysts survive over winter in buildings and on pasture. Oocyst shedding by healthy ewes also contributes to the environmental contamination. Coccidiosis is essentially a disease of intensive husbandry and the severity of disease is proportional to the level of environmental oocyst contamination. Early born lambs may not ingest sufficient oocysts to become clinically affected, but contribute significantly to the contamination of the environment and disease in later born lambs.

In general, coccidiosis only occurs as a primary problem when stressed animals are exposed to large numbers of coccidian oocysts in a heavily contaminated environment. Most outbreaks of coccidiosis occur in 3- to 8-week-old intensively managed housed lambs. It is unusual to encounter coccidiosis in thriving lambs under normal summer pasture grazing management, although the disease is occasionally seen in naïve older lambs after weaning onto heavily contaminated small paddocks. Coccidiosis is reported to be an important disease in growing lambs in parts of Denmark, brought on by sodium deficiency, induced by the pasture application of potassium-rich pig slurry. Whether the coccidioisis is a result of oocyst-rich soil ingestion by sodium-deficient lambs, or by compromised immunity, is unknown. The disease is characterised by acute onset diarrhoea, dullness, anorexia, dehydration and weight loss affecting a high proportion of the lamb flock (Fig. 1.175).

The diagnosis of coccidiosis is based on the history of intensive lamb management and clinical signs. Confirmation of a diagnosis of coccidiosis is problematic. Faecal oocyst counts may support a diagnosis of coccidiosis, but alone are of limited diag-

Fig. 1.175 Diarrhoea, anorexia, weight loss and dehydration in 7-week-old Bluefaced Leicester lambs, caused by coccidiosis.

Fig. 1.176 Intravenous fluid therapy for a severely dehydrated 7-week-old Bluefaced Leicester ram with coccidiosis. In this case treatment was successful.

nostic value because several species of *Eimeria* may be present without causing disease. Oocysts belonging to the different species of *Eimeria* cannot be routinely differentiated when examined in faecal smears using a standard practice microscope. Specialist laboratories can differentiate between the pathogenic and non-pathogenic sporulated *Eimeria* oocysts on the basis of their size and morphology. Confirmation of a diagnosis of coccidiosis usually depends on microscopic examination of gut sections collected during the postmortem examination of freshly dead animals.

Outbreaks of coccidiosis in lambs are usually managed by whole-flock treatment with sulpha drugs and avoidance of intensive grazing. Supportive fluid therapy may also be indicated (Fig. 1.176). It can be useful to keep later born lambs on different areas to early born lambs. In flocks where a high risk of coccidiosis has been identified, the coccidiostat drug decoquinate can be included in lamb creep feed for the prevention of the disease, or fed to ewes to reduce their contribution to environmental oocyst contamination. However, this strategy does not enable good development of immunity and there is a danger of disease when the medication is stopped. Diclazuril can be administered orally to lambs as a single preventive treatment in anticipation of a problem.

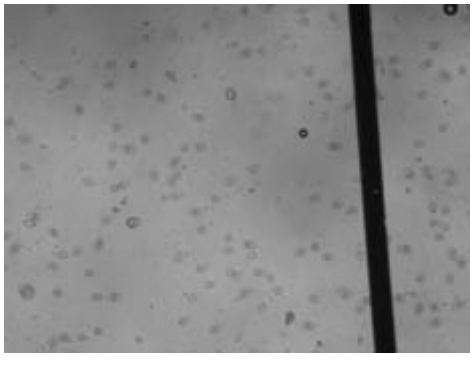


Fig. 1.177 Large numbers of *Eimeria* oocysts are routinely identified in faecal samples submitted from healthy lambs for worm egg counting. (MacMaster slide preparation x50)

Coccidiosis is frequently misdiagnosed as the cause of diarrhoea in growing lambs on the basis of high faecal coccidian oocyst counts alone (Fig. 1.177). Thus, it is important to determine the presence of other underlying and possibly more serious diseases, such as nematodirosis, teladorsagiosis or cobalt deficiency whenever coccidiosis is suspected.

Chapter 2

Lamb Growth

Before the implementation of the Single Farm Payment in January 2005, the principal reason for keeping sheep on many UK farms was to qualify for headage subsidy payments. This situation no longer pertains, and while sheep production affords environmental and social benefits that justify continued subsidy support, the principal reason for keeping sheep on most farms must now be to convert primary forage or herbage crops into a marketable product. Thus, the profitability of sheep farming is now strongly influenced by the efficiency of feed conversion to meat. In some situations, the feed conversion efficiency of ill-thrifty lambs is half that of lambs that achieve maximal growth rates. This point may be illustrated by New Zealand data comparing the feed conversion efficiency of healthy and ill-thrifty terminal sire cross, weaned lambs (Table 2.1).

While daily weight gains of 300 g/day may not be achievable by most UK flocks, and the cost of UK forage production varies throughout the lamb finishing period, the attainment of maximal lamb growth rates is nonetheless a prerequisite for efficient production. This situation arises because lambs have a daily feed requirement for maintenance which must be met before growth can occur, irrespective of the time taken to reach slaughter weight. Breeding replacement ewe lambs must attain satisfactory growth rates during their first year in order to ensure optimal lifelong reproductive performance. Furthermore, lambs which are slow to finish are more susceptible to other production-limiting diseases, such as parasitic gastroenteritis, respiratory disease and trace element deficiencies (Fig. 2.1) than rapidly growing animals which leave the farm before the main risk period for these problems.

Over the past two decades in the UK, the income per individual farm animal has fallen, farms have increased in size and farm labour has been dramatically reduced. Under these conditions, even small losses due to avoidable production-limiting

Table 2.1 New Zealand data showing the feed conversion efficiency of rapidly growing and ill-thrifty weaned lambs.

	Gain 300 g/day	Gain 100 g/day
Weaning weight	20 kg	20 kg
Slaughter weight	38 kg	38 kg
Days to slaughter	60	180
Feed required	1.9 kg DM/day	1.2 kg DM/day
Total feed, weaning to slaughter	114 kg DM	216 kg DM
Feed conversion	14 kg DM/kg carcase	27 kg DM/kg carcase

Sheep Flock Health: A Planned Approach. Neil Sargison
 ISBN: 978-1-4051-6044-5

Fig. 2.1 These Scottish Blackface lambs were housed and introduced to an ad-lib concentrate ration during December because they had not reached finished weights. Despite housing and concentrate feeding they subsequently failed to thrive, due to the combined effects of selenium deficiency and ectoparasite infestation. Had their earlier growth rates been higher, the effects of selenium deficiency and ectoparasite infestation would not have been manifest.

Fig. 2.2 The use of free-access mineral supplements, which often contain unnecessary trace elements, iron and salts to enhance their appearance and palatability, can be wasteful and is uneconomic when alternative, specific, targeted supplementation regimes are available.

disease are economically important. Conversely, wasteful, unnecessary treatments are also uneconomic (Fig. 2.2). These losses can only be avoided by the adoption of a rational scientific approach to the diagnosis and management of production-limiting disease.

The efficiency of lamb growth cannot be assessed without first determining realistic targets. Achievable targets vary according to farm type and production systems. Different sheep breeds and farms are suited to production of finished lamb at different times of year (Table 2.2). Most UK farmers aim to produce lamb from grass, with peak output between August and November and smaller numbers of lambs finished between March and June. Market prices generally reflect this seasonal supply and inevitably confound the economic argument in favour of maximal lamb growth rates.

Investigation of ill thrift in weaned lambs

The diagnosis of the cause of ill thrift requires a rational scientific approach, involving a targeted disease history, clinical examination of the flock and appropriate sample collection (Fig. 2.3). Frequently, several potential causes of ill thrift are identified. In these situations, it is important to determine which are primary causes and which are consequential (Fig. 2.4).

Disease history

As in all other clinical investigations, determination of the causes of ill thrift in lambs begins with a relevant history, focused on the important common problems.

Table 2.2 A simplified overview of UK lamb production systems.

Early lambing flocks 	The objectives are to achieve a high level of output per ewe, to produce a quality product at the lowest possible cost per kg and to achieve a margin per ewe comparable with any grass-based system. These objectives are typically achieved using terminal sire-cross ewes lambing over a 35-day period from the beginning of January, with targeted lambing percentages of 180 percent, average lamb birthweights of 4.7 kg and lamb mortality less than 7 percent. Lambs are generally creep fed from about 3 weeks old and weaned at 8 to 10 weeks old onto a high-quality concentrate ration. Feed, labour and other costs involved with out-of-season breeding are high. The achievement of maximum lamb growth rates is essential to ensure that lambs are finished in time for the high-priced Easter market. The following production targets are typical: • daily liveweight gain from weaning to slaughter – 350 to 400 g/day • feed consumption per lamb from weaning to slaughter – 65 kg • average daily feed intake from weaning to slaughter – 1.7 kg • concentrate feed conversion ratio from weaning to slaughter – 4.5:1 • average age of lambs at slaughter – 95 days • all lambs finished within a period of 7 weeks at about 18.5 kg carcase weight (about 40 kg liveweight), and 3L or 3H fat class and E, U, or R conformation classes[1]
Lamb production from grass 	The objectives similar to those of early lambing systems, with all of the lambs finished as quickly as possible off grass. Spring born lambs are usually weaned at 14 to 16 weeks old and finished from about 14 to 26 weeks old, depending on litter size and pasture quality. Weight gain targets vary between flocks: • singles to weaning – 300 to 350 g/day • twins to weaning – 280 to 330 g/day • weaned lambs – 175 to 270 g/day It is important to ensure that finishing lambs are given grazing priority over other livestock
Store lamb finishing systems  	Hill and upland store lambs are sold in the autumn for finishing on lowland farms. Three basic systems are used: • short keep – lambs are finished by December on grass or forage crops such as stubble turnips • medium keep – lambs are grazed on stubbles or forage aftermaths and then finished between January and March on concentrates • long keep – lambs are grazed on poorer pasture before finishing on brassica crops and/or concentrates in the spring Housing and finishing grass-weaned lambs on a complete diet can be cost effective if well managed. However, ill thrift sometimes occurs associated with management problems such as: • poor-quality, unpalatable or stale feed • inadequate trough space • poor building design causing heat or humidity stress • competition between mixed age groups of lambs • concurrent chronic disease

[1] European lambs are currently graded on the basis of fat class and conformation, which is in turn partially determined by body condition, rather than on lean carcase yield.

Fig. 2.3 This picture of 500 April-born Suffolk and Texel cross lambs from a flock of 400 Greyface ewes was taken during September. While the lambs appear healthy and well grown, most ought to have been finished at least one month previously. The post-weaning growth rates of these lambs would have only been about 140 kg/day.

Fig. 2.4 These lambs are scouring and have high parasitic nematode burdens, but the underlying cause of their ill thrift is poor milk production by their dam, associated with inadequate pasture nutrition.

The common causes of ill thrift in weaned lambs are:

- poor nutrition
- previous perinatal disease
- parasitic gastroenteritis
- cobalt deficiency
- selenium deficiency
- fascioliasis
- other specific infectious and management problems such as:
  - respiratory disease
  - lameness
  - sheep scab
  - coccidiosis
  - border disease.

The basic history should, therefore, include information about:

- presenting clinical signs
- time of year
- farming system and feed management throughout the year
- dates of lambing and weaning
- lambing percentage and spread
- numbers, species and classes of animals which are affected
- stocking density
- stock movements
- worming regime and anthelmintics used
- previous trace element problems and supplements used
- history of pasture improvement/fertiliser application
- observations of scouring, coughing, lameness or skin disease
- weather conditions.

Clinical examination

As with all clinical investigations, establishing the history is followed by assessment of the animals' environment and their clinical examination. In the case of the investigation of ill thrift in lambs it is important that the clinical examination is focused on the whole flock, rather than solely on a few particularly ill-thrifty individuals, which may not be representative of the overall problem. Some diseases, such as chronic liver fluke, may be ruled out on the basis of the time of year, or the age of the lambs.

Examination of the flock should include careful observations of:

- forage and feed availability over the whole farm
- variation in size and weight within the group
- body condition scores across the group
- overt signs of lameness, coughing, scouring, ocular disease and pruritus.

In general, when ill thrift has arisen as a consequence of management before weaning (for example, associated with poor nutrition of pregnant ewes or perinatal lamb problems) the group appears uneven, with some well-grown animals and other individual animals in poor body condition (Fig. 2.5). When the problem has arisen after weaning, the group is typically uniformly affected (Fig. 2.6).

Having appraised the group as a whole, it may be appropriate to perform a detailed clinical examination on one or two representative animals. However, it is important to ensure that any findings are interpreted within the context of the overall problem.

This basic process will identify the presence of specific problems such as respiratory disease, footrot and sheep scab. However the clinical signs associated with trace element deficiencies, parasitic gastroenteritis and liver fluke are seldom specific and further diagnostic tests are usually required to differentiate between these causes.

Further diagnostic tests

Faecal samples can be collected for nematode egg counts (FWECs), coccidian oocyst counts and/or identification of fluke eggs. Serum and blood samples can be collected

Fig. 2.5 The lambs in this group are the same age, but some are well grown and some are ill thrifty. The ill thrift problem is probably related to events before weaning, such as perinatal disease.

Fig. 2.6 The lambs in this group are uniformly ill thrifty, associated with a recent, flock-wide problem such as trace element deficiency or parasitic gastroenteritis.

for vitamin B_{12} and glutathione peroxidase (GSHPx) assays. Before collecting samples, it is important to ensure that:

- the most appropriate animals are sampled
- animal management before sampling is accounted for
- the correct number of samples is submitted
- the most appropriate samples are collected in each individual case
- samples are correctly handled.

Postmortem examination

Ill-thrifty lambs may be of low financial value, so the humane destruction and postmortem examination of one or two of the worst affected animals is sometimes appropriate. While postmortem examination provides a useful opportunity for appropriate sample collection, it is important to concentrate on those signs that may be related to a flock ill-thrift problem, rather than on any incidental or consequential findings.

Postmortem examination of ill-thrifty lambs

Sheep can be placed on their left side and opened to reveal the viscera on their right (Fig. 2.7). The liver should be examined for evidence of fascioliasis and sections of fresh liver can be collected into suitable containers for trace element assay. The abomasum is pinched or ligated close to the pylorus and removed by cutting through the omasum and jejunum. When the abomasum of a freshly dead sheep is opened without allowing escape of digesta, parasite burdens can be assessed by the presence of swirling movement on the surface of the digesta. The mucosal surface should be examined for the presence of *Haemonchus contortus* and *Teladorsagia circumcincta* worms and the abomasal contents can be saved in a leakproof plastic bag for a total worm count, if preferred. The small intestine can be ligated at its proximal end and removed for a total worm count by cutting down through the pancreas and across the rectum. Other postmortem signs such as respiratory pathology, thickening of the abomasal mucosa, abnormal intestinal contents, pallor, or ascites should be noted.

The postmortem examination should be focused on the common causes of ill thrift and, unlike the situation where the cause of death of an individual animal must be ascertained, does not necessarily require a detailed examination of all organ systems. Incidental findings must be kept in context.

Fig. 2.7 On-farm postmortem examinations have the additional advantage of clearly demonstrating the effects of specific diseases. This lamb died from subacute fascioliasis.

Parasitic gastroenteritis

The major gastrointestinal nematode parasites of sheep are *Teladorsagia circumcincta* (formerly *Ostertagia circumcincta*), *Trichostrongylus vitrinus*, *Haemonchus contortus* and *Nematodirus battus* (Table 2.3). These parasites limit the productivity

Table 2.3 Pathogenesis of the major UK sheep nematode parasites.

Teladorsagia circumcincta (brown stomach worm)  1 cm	The presence in the abomasum of 5000 or more *T. circumcincta* third (L_3) and fourth (L_4) stage larvae, or adult nematodes generally results in clinical disease, although individual sheep can tolerate different parasite burdens, depending on factors such as their resilience, plane of nutrition and immunity. Disease is primarily associated with the host's immune response to incoming L_3, resulting in hyperplastic inflammation of the abomasal mucosa. With time, gastric secretory cells are replaced by unfunctional cells, leading to an increase in the pH of the abomasal contents and impaired abomasal function. Affected sheep become hypoproteinaemic, due to protein leakage across the damaged abomasal mucosa. The highest incidence of clinical teladorsagiosis is seen during the summer and autumn months, characterised by scour, loss of appetite and weight loss.
Trichostrongylus vitrinus (black scour worm)  1 cm	The presence of 5000 or more *T. vitrinus* (and/or *T. colubriformis* in southern parts of the UK) in the proximal small intestine generally results in clinical disease. The parasites cause mucosal hypertrophy and villous atrophy, resulting in protein leakage, electrolyte and mineral imbalances. Trichostrongylosis is usually seen during late autumn and winter months, characterised by anorexia, dark-coloured scour, poor skeletal growth, reduced wool quality and chronic ill thrift.
Haemonchus contortus (barber's pole worm)  1 cm	*H. contortus* in the abomasum feed on blood and cause anaemia and hypoproteinaemia. *H. contortus* are extremely pathogenic and the presence of as few as 500 parasites can result in severe clinical disease. Furthermore, *H. contortus* has enormous biotic potential and abomasal burdens of 10 000 parasites are sometimes encountered. In endemically infected flocks, disease is seen predominantly in young lambs during late summer, but in naïve flocks disease is seen in sheep of all ages between spring and late autumn. Clinical haemonchosis is characterised by anaemia, submandibular oedema, ascites, ill thrift and death. Scour is seldom present when *H. contortus* is the predominant nematode parasite species.
Nematodirus battus (no common name)  1 cm	*N. battus* infection occurs mostly in lambs between 6 and 12 weeks old between May and July, causing catarrhal inflammation of the small intestine. Clinical nematodirosis is generally associated with the presence of more than 2000 parasites in the small intestine and is characterised by acute onset, profuse, watery diarrhoea, lethargy and abdominal pain, with rapid weight loss, dehydration and death.

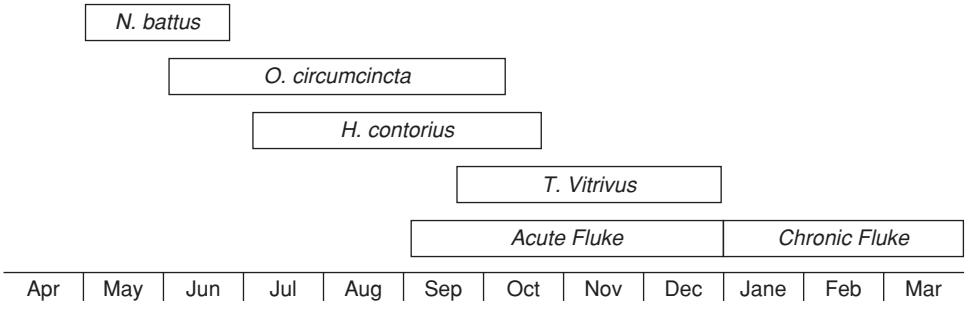


Fig. 2.8 The established seasonal pattern of nematode parasitism in UK sheep.

of susceptible animals because of their various feeding activities: evoking an immune response in their host, damaging the absorptive lining of the abomasum and intestine, removing nutrients from the ingesta or, in the case of *H. contortus*, feeding on blood. The net effects of these activities are inefficient feed utilisation, fluid and electrolyte imbalances and anaemia.

Parasitic gastroenteritis is the most important production-limiting disease of UK sheep. Obvious losses are associated with:

- lamb deaths
- poor lamb growth rates
- reduced carcase values associated with delayed finishing
- treatment and management costs.

Haemonchosis is an important cause of ill thrift and death in some sheep flocks and nematodirosis commonly causes high production losses in young lambs. While there is a general seasonal occurrence of parasitism by different nematodes (Fig. 2.8), several species are usually present at the same time, so it is sometimes more useful to consider parasitic gastroenteritis as a whole, rather than as specific diseases.

Other gastrointestinal nematode parasites are seldom present in sufficient numbers to be considered pathogenic, but may become relevant under specific circumstances:

- *Trichostrongylus axei* (abomasum)
- *Trichostrongylus colubriformis* (small intestine)
- *Nematodirus filicollis* (small intestine)
- *Cooperia curticei* (small intestine)
- *Strongyloides papillosus* (small intestine)
- *Bunostomum trigonocephalum* (small intestine)
- *Oesophagostomum venulosum* (large intestine/caecum)
- *Trichuris ovis* (large intestine/caecum)
- *Chabertia ovina* (large intestine/caecum).

The following groups of sheep are susceptible to the effects of gastrointestinal nematode parasitism and should routinely be included in any control programme:

- Lambs during their first grazing season.
- Naïve adult animals being moved from extensive 'worm-free' grazing to contaminated lowground pasture.
- Rams.
- Sheep with compromised immunity, for example due to poor body condition, undernutrition, concurrent disease and lactation.

Fig. 2.9 Once pastures have become heavily infested with infective nematode larvae, lambs often continue to scour, despite effective, conventional anthelmintic treatment.

Clinical gastrointestinal parasitism of lambs is common, characterised by scour, poor body condition, open fleeces and a pot-bellied appearance. However, overall the greatest economic importance of nematode parasites is subclinical production loss arising from continuous low-level exposure to infective larvae. Daily exposure for 90 days to about 4000 *T. circumcincta* or 2500 *T. vitrinus* infective L_3 can reduce liveweight gains of ad-lib fed sheep by up to 50 percent due to reductions in appetite and feed conversion efficiency. Continuous low-level infection with *T. vitrinus* or *N. battus* can also result in impaired calcium and phosphorus retention and poor skeletal mineralisation. The presence in the gastrointestinal tract of susceptible animals of both adult nematode parasites and larvae is usually production limiting.

Once pastures have become contaminated with infective nematode larvae, it is difficult to achieve satisfactory weight gains in lambs that graze them. Conventional anthelmintic treatment often improves performance for a few days, after which lambs again fail to reach their potential daily weight gains (Fig. 2.9). The reason for this was demonstrated clearly more than 20 years ago by work from the Moredun Research Institute. Weight gains were compared between lambs reared on ad-lib feed in nematode parasite-free conditions, lambs exposed daily to 5000 *T. circumcincta* larvae and lambs exposed to infective parasite larvae and dosed with a conventional anthelmintic every 3 weeks. Daily exposure to parasite larvae reduced growth rates to half those of control lambs, but frequent anthelmintic drenching only restored 20 percent of the loss in growth rate (Fig. 2.10).

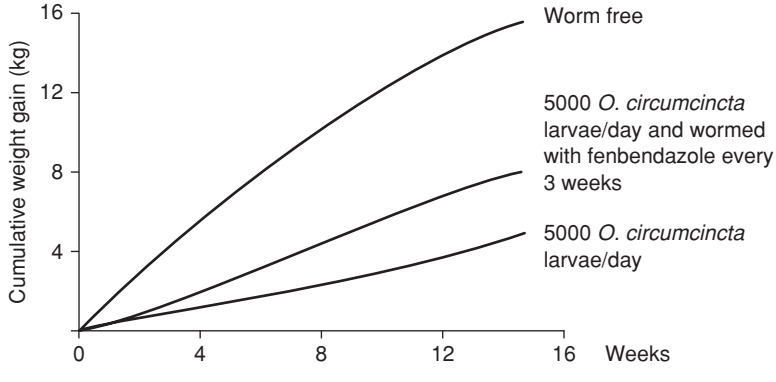


Fig. 2.10 The effect of incoming infective larvae on lamb liveweight gain. (Modified from Coop RL, Sykes AR and Angus KW. *Journal of Agricultural Science, Cambridge*, 1982, 98, 247–255.)

Thus, the aim of effective nematode parasite control is to limit exposure of susceptible lambs to significant burdens of infective larvae on pasture. Sustainable control programmes in individual flocks are based on the commonsense application of knowledge of the farming system and of the relationship between pasture contamination, the availability of infective larvae on pasture and the build-up of infection in sheep.

The epidemiology of parasitic gastroenteritis, with reference to control programmes

Effective control of parasitic gastroenteritis relies on a sound understanding of the life cycle and epidemiology of the important parasites. With the exception of *N. battus*, the important sheep nematode parasites have similar life cycles.

The epidemiology of parasitic gastroenteritis depends on the relationship between pasture contamination, the availability of infective larvae on pasture, the build-up of infection in sheep and the regulatory effects of acquired immunity. Adult female nematodes within the abomasum or small intestine of their sheep host lay eggs, which are voided with faeces. Eggs hatch within the faeces, giving rise to first-(L_1), then second-stage larvae (L_2), which feed on faecal bacteria. L_2 then moult to infective third-stage larvae (L_3). The L_3 are retained within the sheath of the L_2, providing protection from desiccation, but preventing them from feeding. L_3 find their way onto herbage, from where they are ingested by their sheep host. Ingested L_3 exsheath within the ruminoreticulum before passing into the abomasum or intestine and completing their development through fourth-stage larvae (L_4) to adults. For most sheep abomasal and small intestinal nematode parasites, the pre-patent period between ingesting L_3 and significant egg shedding is between 17 and 18 days.

The environmental conditions that favour egg hatching and larval development differ slightly for the different parasitic nematode species, but are generally optimal at high humidity and temperatures above 22°C. Given such conditions, egg hatching may occur within 24 hours of faecal shedding and larval development to infective L_3 may occur within as few as 7 days. The success and rate of egg hatching and larval development to L_3 is reduced at low temperatures, being minimal at temperatures below 10°C. Eggs, L_1 and L_2 are easily killed by desiccation, but given adequate moisture, infective L_3 mostly survive on pasture for 10 to 12 weeks. L_3 of some species can survive for more than 12 months and are not necessarily killed by frosts. Thus, general environmental moisture and temperature have a major influence the epidemiology of parasitic gastroenteritis through their effects on free-living nematode populations on pasture. However, the temperature and moisture within microclimates such as sheep faeces can result in unpredictable larval survival during situations when general environmental conditions are unfavourable. For example in southern parts of the UK, clinical outbreaks of teladorsagiosis and haemonchosis are sometimes reported in autumn, when rainfall at the end of a period of drought enables the release of large numbers of larvae onto pasture from faeces, parts of which remained moist having been protected by a dessicated crust throughout the drought period.

Pasture larval contamination in spring arises both from overwintered infective L_3 on pasture and from nematode eggs shed by recently lambed ewes. The egg output of lactating ewes, referred to as the periparturient rise, derives from nematodes which overwintered within the ewes, and from completion of the life cycle of overwintered

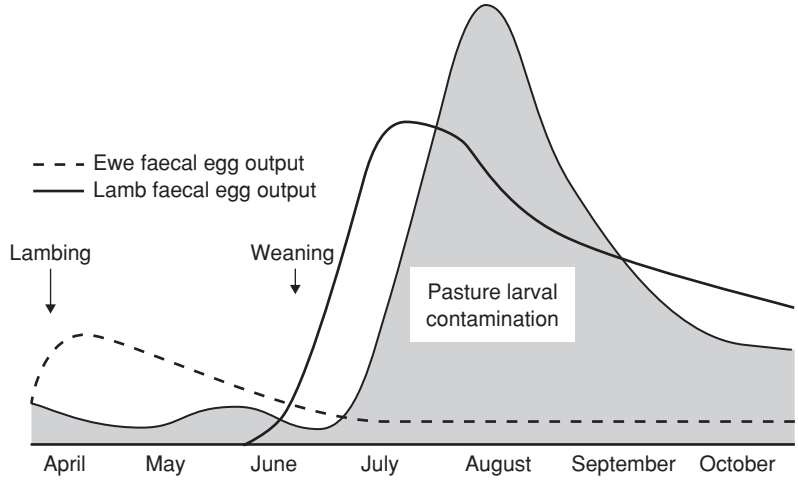


Fig. 2.11 Generalised pattern of pasture larval contamination and lamb faecal egg output in the absence of control measures (except *N. battus*). This summary oversimplifies the situation, which is in practice altered by the use of anthelmintics and safe grazing, by exceptional weather or stocking conditions, by the effects of microclimates, or by the presence of different nematode species.

L_3 ingested with pasture after lambing. The relative importance of these sources of pasture larval contamination differs from year to year with different winter weather conditions and sheep grazing management, and between different regions of the UK. When ingested by naïve lambs, these infective larvae give rise to adult nematodes, which accumulate over the summer months and contribute to subsequent pasture larval contamination, leading to disease (Fig. 2.11).

The epidemiology of N. battus

The life cycle and transmission of nematodirosis differs from that of the other important sheep nematode parasites. Infective larvae develop within the *N. battus* egg and can survive freezing and very low temperatures on pasture for up to 2 years. Hatching and release of infective larvae mostly occurs after a period of cold exposure followed by a period of daily maximum temperatures above 10°C. Infective *N. battus* larvae generally do not survive for long on pasture when weather conditions are warm and dry, but can survive for several months during cool and damp weather. Disease outbreaks are seen when the presence of large numbers of larvae on pasture coincides with grazing by susceptible lambs. Most nematodirosis outbreaks are therefore seen in May and June, the precise timing being influenced by local weather conditions and sheep grazing management.

The classical life cycle of *N. battus* essentially involves transmission from one season's lamb crop to the next and the accumulation of infection on pasture takes place over a period of grazing by susceptible lambs (and occasionally young calves). Typically, lambs which are grazed on pastures used for young lambs during previous years are at greatest risk. Very young lambs are at low risk because their herbage intake is limited, while older lambs develop some degree of protective immunity, which is partly dependent on previous exposure.

Fig. 2.12 High faecal *N. battus* egg counts are sometimes seen in older lambs during the summer and autumn months, potentially complicating the conventional epidemiology of nematodirosis.

During recent years, unpredicted high *N. battus* egg counts and clinical nematodirosis has been seen in older lambs during late summer and autumn (Fig. 2.12). The reason for this is unclear and may involve an evolutionary change in the parasite's dependence on specific weather conditions for egg hatching, or may simply be an effect of climate change on larval survival. Consequently, some pastures may become heavily contaminated as a result of grazing by older lambs during the previous summer, possibly increasing the risk of nematodirosis in young lambs during the following spring.

The diagnosis of parasitic gastroenteritis

As with all disease investigations, the diagnosis of parasitic gastroenteritis begins with a relevant disease history and the interpretation of any clinical signs. Knowledge of anthelmintic treatments and pasture management for the control of gastrointestinal nematodes is essential. The assessment of nematode parasite burdens may be supported by FWECs, coproculture and genus identification, gross postmortem examination, total nematode counts and pasture larval counts.

Faecal nematode egg counts

FWECs provide a reasonable estimate of the sheep host's nematode burden, enabling prompt diagnosis and treatment of parasitic gastroenteritis and providing a basis for effective and sustainable nematode control strategies for individual flocks. FWECs need to be based on freshly voided samples from at least 10 animals, to allow for the variation that occurs within groups of sheep. Bulked samples from 10 or more animals can provide useful information, but the results are harder to interpret than those from individual samples. There are limitations to FWECs, which should be taken into account when interpreting results. Faecal nematode egg excretion can be altered for a number of reasons, such as variation in egg production between genera; sheep breed, age and reproductive status; the length of time that the sheep have been off pasture; and the dry matter content of the faeces. FWECs only indicate the presence of adult worms, but loss of production can occur when only larvae are present. Collection of faecal samples within 2 hours of removal of sheep from pasture can overcome some of the variation in FWECs associated with faecal concentration due to the effect of yarding on faecal dry matter. Faecal larval cultures can mitigate against the effect of variation in egg production between nematode genera.

Various methods can be used for faecal nematode egg counting, all involving the same basic principle, that the faecal mass is first broken up, and the eggs in a known

Table 2.4 Methods commonly used for faecal nematode egg counting.

Cuvette method 	• Collect faeces into a plastic bag. • Weigh the sample within the bag. • Add 10 ml of water per g of faeces. • Thoroughly disperse and mix the faecal sample within the bag. • Withdraw 10 ml of dispersed faeces and pass through a coarse sieve to remove larger particles. • Transfer the filtrate to a flexible centrifuge tube, then spin at 1000 rpm for 2 minutes. • Remove the supernatant, then re-suspend the sediment in saturated sodium chloride solution and centrifuge at 1000 rpm for 2 minutes. • Clamp the top of the flexible centrifuge tube and transfer the meniscus to a counting cuvette. Fill the cuvette with saturated sodium chloride solution. • Count the eggs in the cuvette. Each egg counted using this method represents one egg per gram (epg).
Modified McMaster method 	• Place a small bowl and small sieve on a balance. • Tare the balance and weigh 2 g of faeces into the sieve. • Add 28 ml of saturated sodium chloride solution. • Mix well with a plastic spoon to break up the faecal matter. • Discard the coarse and fibrous material which remains in the sieve. • While gently mixing, load a pipette with a sample of the remaining faecal suspension. • Fill one chamber of a McMaster counting slide. • Re-load the pipette and fill the other chamber of the McMaster counting slide. • Leave the filled slide to stand for a few minutes. • Count eggs in both chambers of the slide. Using this protocol, each egg seen within the counting grids on the McMaster slide represents 50 eggs per gram.

quantity of faeces are then floated onto the underside of a counting chamber using saturated saline solution. Some research and diagnostic laboratories use a cuvette counting method, which is sensitive and accurate, but requires specialist equipment and is generally impractical for use in veterinary practice laboratories or on farms. Most veterinary practices use a variation of the modified McMaster counting method, which is a simple procedure providing useful diagnostic information, but is relatively insensitive for research purposes (Table 2.4). The sensitivity of some methods that were developed for use in countries where *H. contortus* is the predominant nematode parasite, for example involving extraction of eggs in a known mass of faeces through a series of nylon sieves, may be inadequate when applied to the management of less fecund nematodes such as *T. circumcincta*, which predominate in the UK.

The eggs of several different gastrointestinal parasites can be identified using these saturated saline floatation methods, so it is important to differentiate those which have the potential to cause disease from those which are likely to be clinically irrelevant (Table 2.5). Other material such as mite eggs, fungal spores and pollen grains must also be differentiated from gastrointestinal parasite eggs.

FWECs should only be interpreted in conjunction with knowledge of general farm management and parasite control practices, knowledge of seasonal weather

Table 2.5 Eggs seen using standard salt floatation methods, which require differentiation.

Trichostrongyle eggs

100 µm

N. battus

100 µm

- Trichostrongyle eggs belonging to *Teladorsagia*, *Trichostrongylus, Haemonchus* and *Cooperia* genera are thin-walled, oval shaped and between 70 and 100 µm long. The eggs may contain a morula of blastomeres or a pre-hatch larva, depending on the age of the faecal sample and their stage of development. While there are subtle differences in shape and size of some of the trichostrongyle eggs, for example those of *H. contortus* are smaller, while those of *T. colubriformis* are slightly flattened, giving the curvature of their walls an asymmetrical appearance, these differences cannot be reliably differentiated on gross microscopic examination. Trichostrongyle eggs of different genera can be stained with fluorescent agglutinins, which specifically bind to lectins on their surface. For example, *Haemonchus* spp. eggs can be identified using a fluorescein-labelled peanut agglutinin. However, this method is not inexpensive and requires access to a specialist fluorescent microscope.
- *Nematodirus* spp. eggs are larger than the other trichostrongyle eggs, being 150 to 200 µm long, with a small number of discrete, large, round blastomere cells. *N. battus* has a dark, parallel-sided shell, while *N. filicollis* and *N. spathiger,* which do not have the same critical hatching requirements as *N. battus* and are of lesser clinical significance, have clearer shells with curved sides. The absence of *N. battus* eggs in faecal samples does not exclude a diagnosis of nematodirosis, because disease can occur during the pre-patent period of infection.

Strongyloid eggs

100 µm

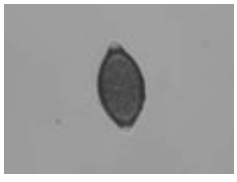
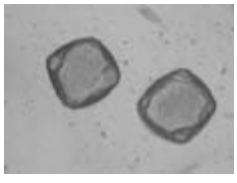
- The eggs of *Chabertia* and *Oesophagostomum* (large intestinal strongyles) appear similar to those of the trichostrongyle nematodes, but are slightly larger and rounder. These strongyloid nematodes are seldom clinically significant in UK sheep, and most pathology occurs during their 6 to 8 weeks pre-patent period. At certain times of year, strongyloid eggs may contribute significantly to the FWECs of some classes of sheep without associated disease or ill thrift (for example, coproculture of ewe faeces during the autumn and winter months often yields predominantly *Chabertia* or *Oesophagostomum* spp. L_3). The presence of strongyloid eggs should be considered when interpreting FWECs of groups of sheep which have not recently been treated with an anthelmintic.
- *Bunostomum* (hookworm) eggs are about 150 µm long with rounded ends and sticky shells to which faecal debris adheres. Hookworms were once common, but are now seldom identified in the UK.

Rhabditoid eggs

100 µm

- *Strongyloides* spp. eggs are small, 40 to 60 µm long, thin walled and almost rectangular shaped, containing a coiled L_1. The identification of these eggs is usually clinically insignificant, even when they are present in large numbers. *Strongyloides* spp. nematodes can have both parasitic and free-living life cycles. Parasitic female L_3 penetrate the skin of their host then migrate through various organs to the small intestine, where unfertilised larvated eggs are produced by parthenogenesis. Free-living life cycles, with production of fertilised eggs, occur on the ground, with females sometimes laying eggs directly onto faeces. Thus, high *Strongyloides* spp. egg counts are sometimes identified in faecal samples collected off the ground. *S. papillosus* has a short pre-patent period, so eggs are often present in faecal samples collected between 10 and 14 days after anthelmintic treatment, and must be differentiated from trichostrongylid eggs to avoid the incorrect diagnosis of anthelmintic resistance.

Table 2.5 (*cont'd*)

Trichuroid eggs  100 µm	• *Trichuris ovis* (whipworms) are sometimes identified as incidental postmortem findings in sheep which have not been treated with an anthelmintic during the previous 7 to 10 weeks, as 4 to 6 cm long nematodes with long filamentous heads embedded in the mucosa of the caecum and large intestine. They are of negligible clinical significance. *Trichuris* spp. eggs are about 60 µm long, brown-coloured, thick-walled and extended oval shaped, with a clear plug at both poles.
Anoplocephalid (tapeworm) eggs  100 µm	• *Monezia expansa* eggs are irregularly shaped and about 60 µm across.
Coccidian oocysts  100 µm	• Most *Eimeria* spp. oocysts are about 15 to 25 µm diameter (those of some species are slightly larger) and ovoid or spherical shaped, with refractive shells surrounding a mass of protoplasm. The oocysts of some species have a small pore at one end. Speciation of these oocysts is a specialist procedure, requiring leaving the faeces to stand for a few days to enable sporulation and accurate measurement.

conditions and the time of year, and consideration of which nematode species are likely to be represented. While mean FWECs of less than 250, between 250 and 800, and more than 800 epg are sometimes arbitrarily used to indicate low, moderate and high levels of gastrointestinal parasitism respectively, these values are largely meaningless when applied without consideration of individual circumstances.

FWECs can be useful to monitor nematode parasite burdens to determine the need for anthelmintic treatment and in the diagnosis of anthelmintic resistance. However their main benefit is in elucidating the pattern of nematode parasitism on an individual farm over time, enabling the development of targeted nematode control strategies.

Various molecular methods have been developed to confirm the species identity of most trichostrongyle nematodes. Polymerase chain reactions (PCRs) can be used to target genus- or species-specific markers such as the internal transcribed spacer 2 (ITS2) or the non-transcribed intergenic spacer (NTS) regions of the nematode genome (Fig. 2.13). The costs involved with these methods currently restrict their application to scientific research.

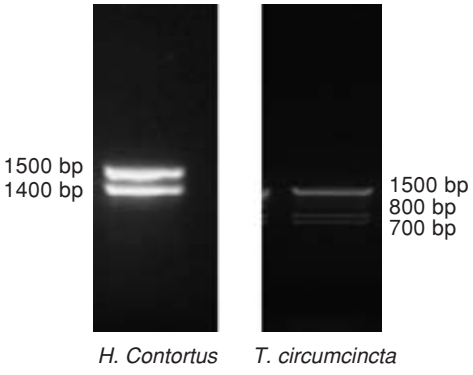


Fig. 2.13 Nucleic acid-stained bands on a 1.5 percent agarose electrophoretic gel, specifically indicating different lengths of DNA PCR products in the NTS regions of *Teladorsagia* and *Haemonchus* spp.

Fig. 2.14 Swirling movements on the surface of the abomasal fluid contents indicate a high nematode parasite burden.

On-farm postmortem examination

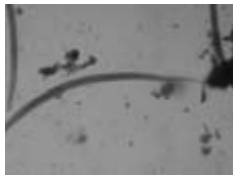
This is a valuable exercise to demonstrate how numerous and damaging gastrointestinal parasites can be. When the abomasum of a freshly dead carcase is opened without allowing escape of digesta, heavy parasite burdens can be assessed from the presence of swirling movement of the surface of the abomasal fluid (Fig. 2.14).

Faecal larval culture

The trichostrongyle and strongyle parasites can be identified to genus level by examining L_3 cultured from faeces (coproculture). Fresh faeces are spread onto a tray lined with a polythene bag and placed within another polythene bag perforated with air holes. Pelleted faeces are left intact and spread to a depth of about 3 cm, while wet faeces are broken up and mixed with vermiculite. The faecal cultures are then incubated at 22°C for 10 days or at room temperature for about 14 days. L_3 are recovered by first flooding the faecal cultures for about 2 hours with tepid water before pouring through a coarse sieve, while taking care not to break up the faecal mass. The filtrate is poured through a filter paper to trap any L_3 present. The filter paper is then submerged at the top of a jar full of tepid water, enabling the L_3 to move through it and to sediment to the bottom of the jar. After about 2 hours, the Baermannised L_3 can be withdrawn from the base of the jar, and either examined immediately, or stored for several months at 4°C. A drop of water containing larvae can be pipetted onto a microscope slide with a drop of helminthological iodine to kill and stain the sheathed L_3 and examined under a cover slip at 100x magnification. A further sample of larvae should be exsheathed by adding a drop of sodium hypochlorite solution and leaving for a few minutes before addition of the iodine and examination.

Parasitic L_3 are generally enclosed in a protective sheath that extends to a point beyond their tail. Larvae that lack this sheath are either free-living nematodes, which appear darkly stained with a double bulb-shaped oesophagus, or *Strongyloides* spp. larvae, which are slender, with a simple oesophagus extending to about half of their length and a blunt tail that appears as if the tip has been broken off. Sheathed larvae are first classified by the length of their sheath tail (Table 2.6).

Table 2.6 Morphological identification of parasitic nematode L_3.

Short sheath tail 	• *Teladorsagia* or *Trichostrongylus* spp. • *Teladorsagia* and *Trichostrongylus* spp. are then differentiated by their morphology on the exsheathed sample. The exsheathed tails of *Teladorsagia* spp. are smoothly rounded and asymmetrical, while those of *Trichostrongylus* spp. have one or two terminal tuberosities.
Medium sheath tail 	• *Cooperia* spp. with square heads with two refractive bodies, or *Haemonchus* spp. with bullet-shaped heads.
Long filamentous sheath tail 	• *Oesophagostomum* or *Chabertia* spp. have about 32 gut cells, but cannot be differentiated by microscopy. *Bunostomum* spp. are about two-thirds of the length of *Oesophagostomum* or *Chabertia* spp. and have 16 gut cells. • *Nematodirus* spp. also have long filamentous tails, with about 16 gut cells, but do not hatch in standard coprocultures.

More than 100 sheathed L_3 must be examined and consideration given to the fact that larval yield may differ between genera, depending on the culture conditions. For example, egg hatching in *Haemonchus*, *Cooperia* and *Chabertia/Oesophagostomum* spp. is reduced if the faecal sample is chilled before incubation, while larval development in *Haemonchus* spp. is reduced at lower incubation temperatures. Desiccation or saturation of the coprocultures may have differential effects on different genera.

An alternative L_3 culture method involving egg extraction from faeces, egg hatching and inoculation of L_1 into a broth of live *Escherichia coli* on filter paper before incubating at 24°C for 5 days may overcome some of the problems associated with differential larval development, but is complex and better suited to experimental studies.

Adult *H. contortus* on the mucosal surface of the abomasum are easily visible as 2- to 3-cm long nematodes with a characteristic 'barber's pole' appearance to the females (Plate 2.1). *T. circumcincta* are also visible on the abomasal mucosa as up to 1-cm long, hair-like and brown-coloured nematodes. The small intestine can be ligated at its proximal end and removed by cutting down through the pancreas. *T. vitrinus* are small and hair-like, less than 7 mm long and only just visible to the naked eye. *N. battus* can be seen as slender 2-cm long white worms in the lumen of the intestine (Plate 2.2). *N. battus* are sometimes seen in tangled knots with a characteristic 'cottonwool ball' appearance (Fig. 2.15) but, in animals which have died as a result of acute nematodirosis, many of the worms may have been expelled and few are found in the small intestine.

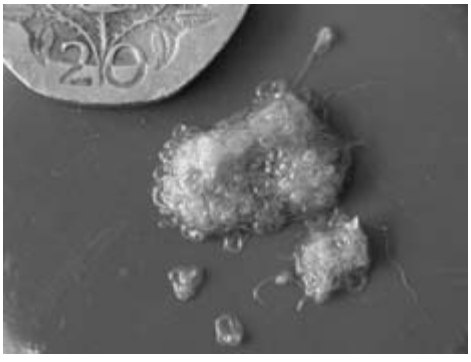


Fig. 2.15 Tangled knot of *N. battus* removed from the small intestine of an 8-week-old lamb.

Fig. 2.16 Thickening of the abomasal mucosa caused by teladorsagiosis.

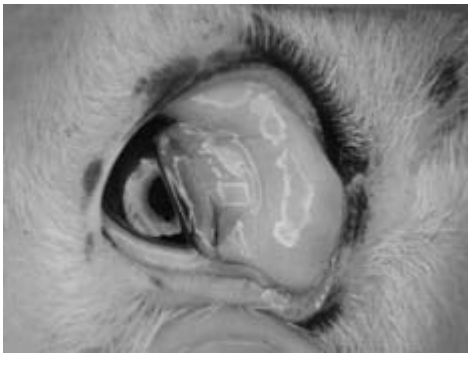


Fig. 2.17 Anaemic ocular mucous membrane caused by haemonchosis.

Other gross postmortem signs such as thickening of the abomasal mucosa and glandular hypertrophy (Fig. 2.16), abnormal intestinal contents, catarrhal enteritis, pallor (Fig. 2.17), or ascites may further support a diagnosis of gastrointestinal parasitism.

Histological examination of stained sections of abomasal or intestinal wall can provide diagnostic information, but is seldom necessary.

Plasma pepsinogen concentrations, which are sometimes useful in cattle, are of limited diagnostic value in sheep.

Total nematode counts

These involve collection of the abomasal or intestinal contents and mucosal washings, rinsing the digesta through a series of sieves to remove coarse fibrous material, and counting the nematodes within a known proportion of the total volume. Total nematode counts are tedious and seldom performed in the UK for diagnostic purposes, although they can provide valuable information. Worm counts for *Nematodirus*, *Teladorsagia*, *Trichostrongylus* and *Haemonchus* genera of 2000, 5000, 5000 and 500 respectively are clinically significant.

Control of parasitic gastroenteritis

Heavily contaminated pastures may harbour more than 1000 infective L_3/kg herbage, which can result in daily intakes of 5000 L_3 per susceptible lamb. Under these con-

Fig. 2.18 Lambs often stop scouring and become tighter fleeced for a few days after anthelmintic treatment, but scour again and appear ill thrifty within about one week. This is not necessarily an indication of the presence of anthelmintic resistance, but merely an indication of the level of pasture infective L_3 contamination.

ditions, while frequent treatment with a conventional, short-acting anthelmintic may improve animal performance for a few days, production losses are inevitable (Fig. 2.18). While lambs that are exposed to a high level of infective larval challenge can achieve satisfactory growth rates subsequent to the onset of acquired immunity, their cumulative weight gains never match those of lambs which are only exposed to a small challenge. The underlying principle of nematode control in finishing lambs is to limit their exposure to infective L_3 on pasture. Very low levels of infective larval challenge have negligible effects on productivity, while enabling the development of immunity, which may be important in store lambs or future replacement breeding stock. There are no simple recipes, but sustainable control programmes can be developed for most individual farms based on the commonsense application of knowledge of the farming system, life cycle and epidemiology of the parasites.

In practice exposure to infective L_3 on pasture can be minimised by:

- Finishing lambs quickly before pasture L_3 burdens become production limiting. This strategy has positive knock-on effects, with lower overwinter larval survival and challenge during the subsequent spring.
- Grazing susceptible sheep only on 'safe' pasture.
- Use of anthelmintics to suppress pasture larval contamination.
- A combination of the above, sometimes referred to as integrated control.

Most cattle nematode parasites do not survive in sheep and vice versa, so pastures which have not been grazed by sheep or goats during the previous 12 months harbour few sheep nematode parasites. Safe pasture can, therefore, be generated through the annual rotation of grazing by the two species, also using cattle to remove sheep nematode larvae from the pasture and open up the sward to expose infective L_3 to the effects of desiccation. The inclusion of cereal crops, brassica crops and silage in the rotation can also be useful. While the practice of mixed grazing of lambs with cattle does not generate safe grazing, it can achieve a dilution effect as cattle remove a proportion of sheep nematode larvae from the pasture. In some circumstances resistant adult sheep may have a similar role, which can be useful for some farm management systems such as organic production.

Grazing management needs to be rigorous and has implications for the whole farm, and few commercial sheep farms are able to provide sufficient safe pasture for the purpose of nematode parasite control in all susceptible sheep, without compromising the efficiency of crop or cattle production. Most nematode control regimes, therefore, rely to some extent on the use of anthelmintics.

Anthelmintics

There are currently more than 70 broad-spectrum anthelmintic products marketed in the UK for the control of nematode parasites in sheep. However these all belong to one of only three groups based on their mechanisms of action. The modern broad-spectrum anthelmintics are grouped as:

- Tubulin binding drugs (benzimidazoles and pro-benzimidazoles; white drenches). These compounds interfere with the nematode parasite's ability to transport and absorb glucose, thus susceptible nematodes die of starvation. Benzimidazole drugs also inhibit nematode egg hatching.
- Ganglion blocking agents (imidazothiazoles and tetrahydropyrimidines; levamisole and morantel). These drugs cause rapid-onset sustained muscle contraction and reversible spastic paralysis. Paralysed nematodes are dislodged into the intestinal lumen. These drugs are not ovicidal.
- Macrocyclic lactones (avermectins and milbemycin). The mechanisms of action of these drugs are not fully understood. Their known effects on nematode parasites include reduced pharyngeal pumping, paralysis of body muscles and effects on the uterus, leading to failure to feed, move or lay eggs respectively. The main drug targets are considered to be glutamate- and gamma-aminobutyric acid (GABA)-gated chloride channels, while non-specific targets such as the P-glycoproteins involved with trans-membrane drug efflux pumps may also prove to be important.

These modern anthelmintic drugs, when given at the recommended dose rates, have a wide safety margin to both sheep and operator and are highly effective at removing susceptible nematode parasites from their host, but when used intensively, can impose strong selection pressure for the development of resistance.

The choice of anthelmintic is based on:

- its efficacy against the nematode parasites present at the time of dosing
- its persistence of action
- the preferred method of administration
- the meat withdrawal period
- cost effectiveness.

In the absence of anthelmintic resistance, all of the modern broad-spectrum anthelmintics are highly effective against most stages of *T. circumcincta*, *T. vitrinus* and *H. contortus*. Some drugs, in particular mebendazole, fenbendazole and levamisole, are not always effective against arrested early fourth-stage larvae (EL_4) of the abomasal nematode parasites *T. circumcincta* and *H. contortus*. This may occasionally be of practical significance when ewes or lambs are treated during late autumn or winter.

For most conventional anthelmintics, effective drug concentrations at their site of action are only maintained for 24 to 36 hours. Moxidectin is the only anthelmintic available in the UK which achieves activity against nematode species for a significant period of time after the administration of the product. The persistence of moxidectin depends on the parasite species present (Table 2.7). Overseas, controlled-release albendazole and ivermectin capsules also fulfil a role as persistent anthelmintics.

In general, oral dosing of lambs is preferable to injection, because of the risk of injection site reactions and differences in meat withdrawal intervals. However, injectable anthelmintics are convenient for use in ewes. Injectable macrocyclic lactone drugs have the added potential benefit of sheep scab control.

Table 2.7 Minimum persistent activity of macrocyclic lactone anthelmintics in sheep.

Parasite species	Moxidectin		Doramectin	Ivermectin	
	oral	injection	injection	oral	injection
T. circumcincta	21 days	35 days	Limited	Nil	Nil
H. contortus	21 days	35 days	Limited	Nil	Nil
T. colubriformis	Limited	14 days	Limited	Nil	Nil

Fig. 2.19 The periparturient rise in FWEC of ewes reinforces pasture infective L_3 contamination and must be minimised in order to prevent parasitic gastroenteritis in lambs.

Nematode control using anthelmintics

One major source of summer pasture larval contamination derives from the periparturient rise in faecal egg output of the ewes. This normally persists for about 6 weeks after lambing, associated with peak lactation, although ewes probably contribute to pasture larval contamination until weaning. During this period significant pasture larval contamination can occur, arising from maturation of arrested EL_4 in the ewe and, more importantly where ewes are turned onto contaminated pasture after lambing, the ingestion and development to adults of overwintered nematode larvae from pasture. The role of ewes in contamination of pasture should not be underestimated, because they produce a large mass of faeces when compared to lambs (Fig. 2.19). Thus, even apparently low FWECs of 100 per gram can result in significant pasture contamination, even in circumstances where development of egg to L_3 is not optimal.

When safe grazing is available and ewes were not grazed on potentially heavily contaminated pastures during the previous winter, anthelmintic treatment after lambing may be unnecessary. In situations where there is a risk that ewes may have acquired significant nematode parasite burdens during the previous autumn and winter, a single benzimidazole or ivermectin anthelmintic treatment around lambing is generally sufficient to remove arrested EL_4 and adult nematodes, thus limiting the subsequent contamination of the pasture.

In the majority of cases, clean pasture is not available for newly lambed ewes and their lambs. Under these conditions, a single ewe dose of a conventional anthelmintic at lambing generally affords unsatisfactory reduction of subsequent pasture contamination during the ewes' periparturient period. Repeated dosing of periparturient ewes with a conventional anthelmintic at 3- to 4-weekly intervals is more effective in suppressing pasture contamination, but is usually impractical due to the difficulties

associated with gathering and handling of ewes with young lambs at foot. Under these circumstances, treatment at lambing with the persistent-acting anthelmintic, moxidectin, can provide satisfactory control of the periparturient rise in nematode egg output and enable the ewes to remove infective L_3 from the pasture without further contributing to pasture contamination.

The periparturient relaxation in immunity to nematode parasites is associated with the protein drain due to lactation. This effect is greatest in ewes suckling twin lambs, so in some situations, anthelmintic treatment of ewes that are in good body condition and suckling single lambs may be unnecessary.

Having controlled the periparturient rise in ewes, it is not usually necessary to dose lambs until weaning, except for the control of *N. battus*. The choice of anthelmintic and subsequent control programme depends on the availability of safe grazing. When safe grazing is available, the traditional advice was to treat the lambs once with an effective anthelmintic before moving. While this practice affords excellent control of nematode parasites, it may also select for anthelmintic resistance, so should be avoided. The timing of lamb anthelmintic treatments should be based on risk assessment and monitoring of FWECs.

When clean grazing is unavailable, anthelmintic treatments may be required throughout the summer months with the primary aim of suppressing pasture contamination. The frequency of dosing depends on the anthelmintic used, and predicted level of pasture infective L_3 contamination, but for conventional anthelmintics and situations where lambs are intensively stocked on warm and damp pastures, a dosing interval of about 4 weeks is generally required. The objective of suppressive anthelmintic treatments is not to totally remove infective larval challenge, rather to permit sufficient low-level challenge to enable the acquisition of immunity.

During the summer months, the predominant parasite species is usually *T. circumcincta*, so parasitic gastroenteritis can be controlled in the short term using a suppressive oral moxidectin dosing interval of 7 weeks. However, in the longer term this strategy affords a survival advantage to *T. vitrinus* or *T. colubriformis*, against which oral moxidectin achieves no significant persistence, enabling the establishment of production-limiting nematode burdens (Fig. 2.20). Furthermore, such reliance on moxidectin may select for anthelmintic resistance during the 'tail' period when drug concentrations fall below levels that are lethal to the nematode parasites.

Control of nematodirosis

The risk of nematodirosis exists whenever susceptible young lambs are turned onto pasture which was used for young lambs during the previous 2 years (Fig. 2.21). Lambing throughout the UK extends from January to May, so it is impossible to provide specific advice about the best timing of preventive management. The risk of a mass hatch of *N. battus* larvae coinciding with the presence of grazing lambs can be predicted on the basis of weather data and local knowledge of the current disease status. Where a risk of nematodirosis exists, susceptible lambs born during late January and February may require treatment by mid-April and again in mid-May, while late-March and April born lambs should be treated in mid-May and may need a second treatment in June.

Most anthelmintics are effective against *N. battus*, although the parasite is the dose limiting species for some of the earlier benzimidazole drugs such as mebendazole

Fig. 2.20 Trichostrongylosis is usually seen during the autumn and winter months. Routine use of moxidectin, which has no persistent activity against *Trichostrongylus* spp., may favour the establishment of high levels of *Trichostrongylus* spp. L_3 on pastures, predisposing to disease outbreaks.

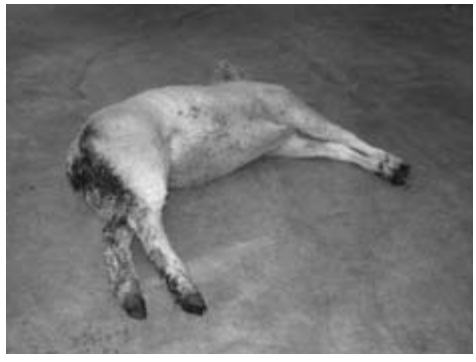


Fig. 2.21 Nematodirosis is an important cause of scour, ill thrift and death in lambs during the spring and early summer.

and for most of the macrocyclic lactone anthelmintics. In the case of doramectin, this problem is overcome by adopting a higher recommended dose rate for the control of nematodirosis. Oral moxidectin has no significant persistent activity against *N. battus*, while the injectable formulation has poor efficacy against the adult nematodes.

Anthelmintic resistance by *N. battus* has not been confirmed in the UK, so drug groups may still be useful for *N. battus* control in flocks where resistance has been identified in *T. circumcincta*.

Alternative methods of nematode parasite control

Good nutrition and general disease management are prerequisites for effective nematode parasite control. Control of parasitic gastroenteritis in intensively managed sheep flocks will always rely on the strategic use of anthelmintics. However, currently unproven alternative strategies, such as breeding sheep for resistance to nematode parasites, the development of antiparasitic vaccines, manipulation of protein nutrition, grazing of condensed tannin-rich herbage species, or the use of nematophagous fungi, may prove to be useful and important adjuncts to control of gastrointestinal parasitism in the face of emerging anthelmintic resistance problems, or in organic production systems.

Genetic selection for both host resistance and reduced susceptibility to nematode parasites could potentially reduce reliance on anthelmintic prophylaxis. Lines of Romney, Perendale and Coopworth sheep have been selected in New Zealand since the late 1970s. However, earlier results showed unfavourable side effects in the selected low FWEC lines when they were grazed alongside unselected sheep, including slower growth, poorer wool production and increased dags. These findings are consistent with observations that experimentally immune-suppressed sheep develop greater nematode burdens than healthy sheep exposed to the same infective nematode larval challenge, but achieve higher growth rates; explained by the protein requirements of immunity and the fact that much of the pathology caused by nematode parasites

arises due to the host's immune response. These problems may be surmountable and genetic selection using simple phenotypic markers such as individual FWECs may prove to be a responsible strategy for some large UK sheep flocks. However, in the absence of reliable genetic markers that can be measured using a simple one-off blood test, selection for resistance to nematode parasites while minimising adverse consequences will be very slow. The application of selection for host resistance to nematode parasites relies on naturally acquired immunity, which is abrogated by nutritional and reproductive stress. Furthermore, the strategy may prove to be of little benefit where lambs are finished quickly, before their onset of acquired immunity.

The development of antiparasitic vaccines affords an obvious potential nematode control strategy. Various experimental methods have been tested, such as the oral administration of crude nematode homogenates, infection by abnormal routes and the use of irradiated attenuated larvae. The latter strategy affords effective protection against lungworm in cattle, but to date has been unsuccessful against gastrointestinal nematode parasites. Current research is focused on the understanding and potential manipulation of the host's immune mechanism and the identification of target antigens on or secreted by the nematode. Some experimental success in terms of raised circulating antibody levels, reduced nematode egg output and reduced worm numbers has been achieved using crude vaccines against *H. contortus*, whose blood feeding activity means that molecules on the surface of its gut are appropriate vaccine targets. However, the development of recombinant subunit vaccines remains elusive. Furthermore, while single nematode species vaccines may have a role in some countries, they are unlikely to be useful in the UK. Vaccination is, therefore, unlikely to provide a useful method of gastriontestinal nematode parasite control in the foreseeable future.

Biological control aimed at free-living sheep gastrointestinal nematode larval stages has been attempted using predatory nematophagous fungi, such as *Duddingtonia flagrans*. This strategy involves feeding fungal spores to sheep, faecal excretion of fungal spores alongside nematode eggs, and subsequent fungal development in the faeces, where their hyphae trap, kill and digest nematode larvae. Efficient nematode trapping has been demonstrated under laboratory conditions, but the success rate in the field has been poor, associated with effects of temperature, diet, season, faecal consistency and larval density. Fungal spores survive passage through the sheep gut, but not long-term residence there, and an effective method of continuously delivering fungal spores to grazing sheep has not been found. Furthermore, any successful system would probably not be licensable in the UK. It is therefore unlikely that nematophagous fungi will provide an alternative method of gastrointestinal nematode parasite control for use in UK sheep flocks.

Forage crops such as sulla, chicory, sainfoin and lotus have some bioactivity against gastrointestinal nematode parasites. These effects may be directly mediated by plant secondary metabolites such as condensed tannins, flavanols, or cysteine proteases, or indirectly mediated through enhanced host immunity resulting from optimised rumen-degradable protein nutrition. While these bioactive forages may prove to be useful adjuncts to nematode parasite control in some countries, they do not withstand heavy UK grazing pressure. Furthermore, their bioactivity is not specific, potentially leading to toxic and anti-nutritional effects on grazing sheep as well as their nematode parasites.

Problems with anthelmintic control of nematode parasites

In the past, anthelmintic control of parasitic gastroenteritis was relatively straightforward for most sheep farmers. However scour, ill thrift and lamb deaths due to parasitic gastroenteritis have become common in recent years, often despite adherence to the basic nematode control principles outlined above. Several reasons have been identified for these problems including:

- Exceptional overwinter survival of infective nematode larvae on pasture associated with the effect of warmer autumn and winter weather on pasture growth, with grazing by lambs with high parasite burdens and enhanced larval development.
- Failure to suppress the post-lambing rise in worm egg output of lactating ewes grazing heavily contaminated pastures.
- Changes in the seasonal pattern of nematodirosis, trichostrongylosis and haemonchosis.
- Anthelmintic resistance.

These problems are compounded by unsound advice about nematode control, constraints associated with reduced farm manpower and increasing flock sizes and inadequate handling facilities.

Timely management to avoid production loss due to these problems depends on knowledge of the situation on each individual farm, based on the accurate diagnosis of previous disease outbreaks, strategic monitoring of FWECs to establish the pattern of nematode parasitism and assessment of the risks.

Haemonchosis

H. contortus differs from the other gastrointestinal parasitic nematodes in that warmer conditions are required for egg hatching and larval development on pasture. Furthermore, *H. contortus* eggs and larvae seldom survive over winter on pasture in

Case report

Between April and June, a group of 192 one-crop lowground Greyface ewes and their 350 Suffolk and Texel cross lambs were grazed on two fields which had been used for about 120 ill-thrifty 6- to 7-month-old weaned lambs until late October. Their ill-thrift problem had been due to severe parasitic gastroenteritis, associated with a failure to recognise a benzimidazole resistance problem. All ewes on the farm were housed for about 12 weeks before lambing, which commenced in the last week of March. The ewes were drenched with moxidectin at turnout, between 1 and 3 days after lambing, and the lambs were treated with levamisole at the beginning of May for the control of nematodirosis.

Ill thrift was reported in the 7- to 10-week-old lambs during the second week of June. The lambs were open-fleeced and had faecal staining of the tail and perineum (Fig. 2.22). During the following week, ten lambs died or were euthanased after being found in extremis. Postmortem examinations performed on three of these lambs revealed very high abomasal *Teladorsagia* species burdens (Fig. 2.23) and large numbers of *Nematodirus* species in the small intestines.

Fig. 2.22 Open fleeces and faecal staining of the perineum of 7- to 10-week-old lambs.

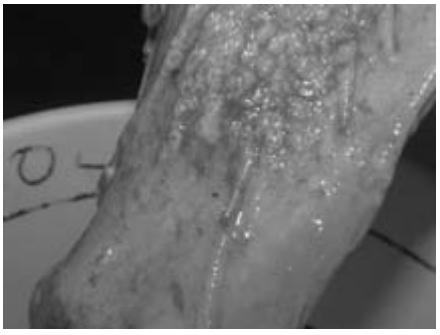


Fig. 2.23 Lymphoid hyperplasia and large numbers of *T. circumcincta* on the surface of the abomasal mucosa.

The FWECs were:

trichostrongyle	*N. battus*
1400 epg	0 epg
2900 epg	4100 epg
4400 epg	1600 epg

Several ewes were scouring and had high FWECs (500–1100 epg) between 7 and 13 weeks after oral moxidectin treatment.

The surviving lambs were treated orally with ivermectin. Clean pasture was unavailable, so the lambs were treated 2 weeks later with oral moxidectin. The lambs appeared more thrifty within 2 days of the first treatment and no more deaths occurred.

Ill thrift, scour and death in the 7- to 10-week-old lambs were caused by concurrent nematodirosis and teladorsagiosis. The diagnosis of teladorsagiosis in the young lambs indicated exceptional overwinter survival of nematode larvae on pasture, associated with previous autumn grazing by lambs with high parasite burdens. Dosing the ewes with moxidectin at lambing should have enabled them to remove a high proportion of overwintered infective larvae from the pasture, without themselves contributing to further pasture larval contamination. However, the appearance of teladorsagiosis in their young lambs illustrates the fact that, under individual farm circumstances, prescriptive nematode control programmes are not always effective.

The rise in ewe faecal egg outputs 7 to 12 weeks after lambing may have occurred because the persistent activity of moxidectin effectively removed exposure of the ewes to *T. circumcincta*, necessary for their redevelopment of immunity. Survival of infective larvae on the pasture following heavy contamination during the previous autumn could have increased the likelihood of large numbers of infective larvae still being present 3 weeks after moxidectin treatment of the ewes. Ewes produce a large mass of faeces when compared to lambs, so these high faecal egg outputs would contribute substantially to pasture larval contamination. Furthermore, the rate of hatching and development to infective larvae would be greater for eggs deposited on pasture during warmer and moister May and June months, than for eggs deposited in March and April. Once pasture has become heavily contaminated, it is difficult to prevent production losses in lambs which graze on that pasture, so in exceptional cases, ewes may require a second anthelmintic treatment to prevent parasitic gastroenteritis in their lambs. Monitoring ewe FWECs from about 6 weeks after oral moxidectin treatment would limit anthelmintic treatment to when it is most appropriate, while minimising the selection pressure for the development of anthelmintic resistance.

Fig. 2.24 Submandibular oedema in a Texel cross gimmer during February, caused by protein loss due to haemonchosis.

northern parts of the UK. The biotic of *H. contortus* is extremely high (one adult female can lay up to 4000 eggs per day) and, given ideal conditions of warmth and moisture, parasite numbers can increase extremely rapidly resulting in disease outbreaks in naïve or immune-suppressed sheep.

H. contortus L_4 and adults in the abomasum feed on blood (Plate 2.3), causing anaemia and protein loss (Fig. 2.24), but in the absence of other gastrointestinal parasites, affected sheep do not scour. Haemonchosis is mostly confined to warmer southern districts of the UK, although reports of the disease in northern Scotland are increasing.

The geographical change in the incidence of haemonchosis is putatively associated with adaptive evolution of the parasites' ability to undergo developmental arrest as EL_4 in the abomasum of its sheep or goat host, completing development to adults during the spring. Developmental arrest (hypobiosis) probably arises in response to changing environmental conditions acting on developmental stages of the parasite, or on its sheep host, although the precise stimuli have not been consistently demonstrated. In temperate regions, maturation of hypobiotic EL_4 occurs in lactating ewes in spring, associated with temporary suppression of immunity, while in non-lactating sheep, most EL_4 are removed by their host's immune response before they can mature. Hypobiosis also occurs in warmer regions, but is not absolute, with wide variations in the proportion of EL_4 involved, while in hot climates, hypobiosis is either not observed, or occurs as a parasite survival strategy during seasonal drought periods. *T. circumcincta* also possesses the ability to evade unfavourable environmental conditions for eggs and developing larvae by arrested EL_4 development, but in cold or temperate regions this is not as predictable or absolute as in *H. contortus*.

Management of outbreaks of clinical parasitic gastroenteritis

Provided that anthelmintic resistance is not present in the flock, any of the anthelmintics that are licensed for use in sheep can be used for the treatment of clinical parasitic gastroenteritis. However, lambs often fail to achieve satisfactory weight gains when they are returned to heavily contaminated pastures after treatment due to the pathogenic effects of subsequently ingested infective L_3. This problem can be overcome by moving lambs to clean pasture after dosing with an effective conventional anthelmintic, although this strategy risks rapid selection for anthelmintic resistance. Alternatively, if clean pasture is unavailable the persistent anthelmintic,

Case report

During the second week of May, the death of six ewes from a group of 40 on a low-ground farm in the south-east of Scotland was investigated. Most of the surviving ewes were in poor body condition, and their lambs had become noticeably ill thrifty during the previous week.

About one-third of the ewe flock had been purchased during the previous autumn, the remaining two-thirds being homebred. Purchased sheep were not routinely dosed with an anthelmintic or combination of anthelmintic drugs on arrival. All of the ewes were dosed with levamisole as they were turned out of the lambing shed, within a few days after lambing at the beginning of March. The ewes were turned onto a 7 ha field of grass and clover, which had been sown during the previous autumn and would have harboured insignificant numbers of parasitic sheep nematodes, thus providing 'clean' grazing.

Postmortem examination of two emaciated, moribund ewes confirmed a diagnosis of haemonchosis (Fig. 2.25). Their FWECs were 16 000 epg and 900 epg. A subsequent FECRT provided no evidence of resistance to the levamisole drench that had been used to treat the ewes after lambing.

Despite metaphylactic anthelmintic treatments, clinical haemonchosis was apparent in the lambs from the beginning of August, resulting in significant production loss for the remainder of the grazing period. Few lambs were finished off the farm and most were sold as stores at the beginning of September.

This case provides further evidence of failure of a prescriptive nematode control programme. Parasitic gastroenteritis could not have been predicted in the group of sheep which were set stocked on clean grazing following anthelmintic treatment of the ewes. The problem probably arose because the levamisole treatment was ineffective against hypobiotic *H. contortus*. EL_4 are generally dose limiting for anthelmintics, either because they evade exposure to the drug, or because their low metabolic rate enables them to tolerate a short period of exposure to drugs.

The lamb ill thrift and clinical haemonchosis which were apparent from the beginning of August were an inevitable consequence of pasture contamination by their dams. This case reinforces the importance of effective quarantine anthelmintic treatment on arrival. The primary aim of such treatment is to prevent introduction of multiple anthelmintic-resistant nematodes, but the concurrent objective of avoiding introduction of new pathogenic nematode parasites, such as *H. contortus*, is also important.

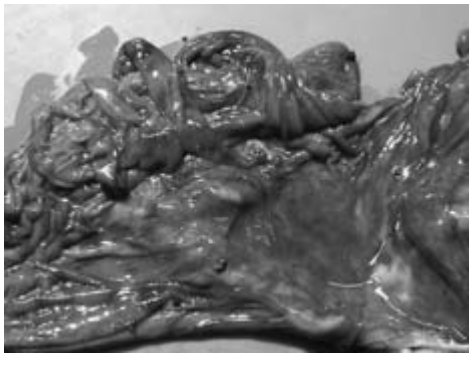


Fig. 2.25 Severe haemorrhage and inflammation of the abomasal mucosa, putatively associated with the mass emergence of arrested *H. contortus* EL_4.

moxidectin, can be responsibly used so that ingested L_3 are killed before they can cause production losses. Consideration must be given to the meat withdrawal intervals, which are 20 and 70 days respectively for the oral and injectable formulations.

Anthelmintic resistance

Most sheep farmers rely on the use of effective anthelmintic drugs for the control of gastrointestinal parasitism. However, on some farms the usefulness of these drugs is reduced because of the presence of anthelmintic-resistant nematodes. The potential emergence of significant levels of multiple anthelmintic resistance in UK sheep flocks presents the single most important sheep disease threat to economic and nationally sustainable lamb production.

The first indications of the presence of anthelmintic resistance are often the failure of lambs to reach finished weights by late autumn, scouring and deaths due to parasitic gastroenteritis, despite preventive anthelmintic treatments (Fig. 2.26). However, anthelmintic resistance can result in clinically unapparent, suboptimal growth rates for some time before overt disease is seen.

The diagnosis of anthelmintic resistance

The useful application of the various tests for anthelmintic resistance is determined by their accuracy, sensitivity, cost and practicality under field conditions.

Drench check

An indication of the effectiveness of anthelmintic treatment can be provided by FWECs performed on fresh faecal samples collected from about 10 sheep, 7 to 14 days after anthelmintic treatment. Individual FWECs are generally most informative, although useful information can be obtained from pooled samples. The identification of positive post-treatment FWECs within the pre-patent period for the parasitic nematodes provides evidence that the drench was not 100 percent effective.

While the drench check is useful, it has limitations. The test cannot differentiate between inefficacy due to poor drenching practice and anthelmintic resistance. Thus, the identification of positive post-treatment FWECs usually requires follow-up investigation using a FECRT or in-vitro assay. Negative post-treatment FWECs do not provide evidence of drench efficacy, unless an assumption is made that adult egg-laying parasites were present before treatment.

Fig. 2.26 These March-born lambs have failed to reach target finishing weights by mid October as a consequence of ineffective control of parasitic gastroenteritis, due to anthelmintic resistance.

Case report

In October 2001, the cause of ill thrift was investigated in a lowground flock of Suffolk lambs (Fig. 2.27). The lambs were scouring and open-fleeced, but the problem was only recognised when they failed to reach slaughter weights as expected. The lambs had been grazed on pasture which had been used for lambs for several years. Pasture cover had been adequate throughout the grazing season, but the lambs had only gained about 120 g/day. The lambs had been dosed orally with ivermectin at 4-week intervals since May. Ivermectin had been used for the previous 2 years, before which drench groups had been rotated annually. Benzimidazole resistance had been identified in the flock in 1998.

The history and clinical signs were consistent with parasitic gastroenteritis, despite the regular macrocyclic lactone anthelmintic dosing. The mean FWEC of four lambs, 2 weeks after ivermectin treatment, was 775 eggs per gram (epg), supporting the diagnosis of parasitic gastroenteritis and indicating that the anthelmintic had not been effective.

A FECRT was subsequently performed to determine the presence of anthelmintic resistance. A total of 33 lambs were weighed, marked and their tags recorded. Faecal samples were collected from each lamb before they were randomly allocated to three groups and dosed orally with a benzimidazole, levamisole or ivermectin anthelmintic. Further faecal samples were collected from the marked lambs 7 and 12 days after treatment. FWECs were performed using the modified McMaster method. Pooled faecal material taken on the day of treatment and from each of the treated groups on days 7 and 12 post-treatment was cultured for 7 days at 23°C to provide infective larvae for identification.

Fig. 2.27 Scour and ill thrift due to parasitic gastroenteritis, despite suppressive ivermectin treatments throughout the spring and summer months.

Table 2.8 Mean FWECs of groups of eleven lambs before and after dosing orally with a benzimidazole, levamisole or ivermectin anthelmintic.

	Benzimidazole		Levamisole		Ivermectin	
	Mean FEC (epg)	Percentage reduction	Mean FEC (epg)	Percentage reduction	Mean FEC (epg)	Percentage reduction
Pre-treatment	255		223		514	
7 days post-treatment	150	41	164	26	564	Nil
12 days post-treatment	255	Nil	235	Nil	720	Nil

Fig. 2.28 This case illustrates the needs to establish effective flock nematode control programmes to ensure satisfactory animal production while preserving the efficacy of the current remaining anthelmintic groups and to provide cohesive and effective recommendations to manage nematode parasites once resistance has occurred.

Resistance to all three anthelmintic groups was identified. The mean pre- and post-treatment FWECs are shown in Table 2.8. Pre- and post-treatment larval cultures yielded 100 percent *T. circumcincta*.

Subsequent nematode control proved to be very difficult in this flock, and sheep farming became uneconomic (Fig. 2.28).

Faecal egg count reduction test

The faecal egg count reduction test (FECRT) provides a practical, reliable, on-farm diagnostic test for anthelmintic resistance, while controlling other causes of poor anthelmintic efficacy.

- Pre-treatment FWECs are performed on between 10 and 12 sheep.
- The sheep are weighed and accurately drenched with the anthelmintics under investigation at their recommended dose rate.
- The sheep are marked to identify which anthelmintic group they were treated with.
- The sheep are re-sampled and post-treatment FWECs are performed between 7 and 14 days later.
- The reduction in FWEC is calculated from the arithmetic mean pre- and post-treatment counts. A reduction of less than 95 percent supports a diagnosis of anthelmintic resistance.

It is important that the anthelmintics are purchased from a reputable source, correctly stored according to the manufacturer's recommendation, and used within their expiry date. Dosing guns must be checked beforehand by delivering aliquots of a preset volume into a calibrated container such as a syringe. Alternatively the sheep can be dosed using a syringe.

The timing of post-treatment sampling is partly determined by the anthelmintic under investigation. For imidazothiazole or tetrahydropyrimidine (levamisole or morantel) anthelmintics, samples should be collected between 7 and 10 days post-treatment, because the efficacy of these drugs against EL_4 is poor: trichostrongyle eggs seen in faeces after 10 days may have arisen from the maturation of female EL_4 which evaded exposure to a lethal dose of anthelmintic. For benzimidazole and avermectin anthelmintics, samples should be collected between 12 and 14 days after treatment. FWECs can remain negative for up to 10 days after treatment with these anthelmintics, despite the presence of anthelmintic resistance, due to temporary suppression of egg laying by surviving resistant female nematodes. Trichostrongyle eggs

may be identified in faeces from about 15 days after effective anthelmintic treatment associated with the minimum pre-patent periods of the trichostrongyle nematodes. Interpretation of FECRT results is potentially complicated for the persistent anthelmintic drug, moxidectin.

The grazing management of the animals between dosing and post-treatment sampling is unimportant. It is usually necessary to collect most of the faecal samples per rectum. Provided that sufficient animals were sampled on the first day, it is not essential that every animal is re-sampled post-treatment. However, the probability of obtaining a sample from each animal is increased if the sheep are gathered shortly before sampling, rather than stood in yards for a prolonged period. Pre-treatment counts of more than 350 epg are usually required to identify the presence of a low frequency of resistant nematodes.

The inclusion of a control group of untreated animals in addition to, or instead of, performing pre-treatment FWECs is sometimes recommended. While this practice can improve the sensitivity of the FECRT under situations where FWECs are declining rapidly, for example during the autumn associated with the onset of host immunity to nematode parasites, or the possible influence of the age of the adult nematode population, it is usually unnecessary and impractical. While the accuracy of the FECRT is dependent on performing FWECs on individual faecal samples, anthelmintic efficacy can sometimes be usefully estimated from the reduction in pre- and post-treatment bulk FWECs.

The sensitivity of the FECRT can be improved by performing separate coprocultures on pooled pre- and post-treatment samples corresponding to each anthelmintic under test. The percentage of each nematode genus in the samples can be determined, enabling FWEC reductions for each genus to be calculated, thus defining the resistant populations more accurately.

In-vitro *bioassays*

Various laboratory bioassays are available for the diagnosis of anthelmintic resistance (Table 2.9). Some, such as the egg hatch assay (EHA) which is used for the diagnosis of benzimidazole resistance, are straightforward and consistently accurate, while others are less reliable, such as the larval development assay (LDA) which is used for the diagnosis of benzimidazole and levamisole resistance. While tests such as the larval feeding inhibition assay (LFIA) and larval migration inhibition assay (LMIA) have been developed as research tools for the study of ivermectin resistance, they are not yet suitable for routine diagnostic use.

Critical efficacy studies

The 'gold standard' confirmation of anthelmintic resistance depends on artificial infection of nematode-free sheep, anthelmintic treatment, humane slaughter and postmortem total nematode counts. This method is only used for research purposes.

The importance of anthelmintic resistance

There has been a marked increase in the prevalence of anthelmintic resistance in UK sheep flocks over the past 20 years. Most reports have involved populations of *T. circumcincta*, although other resistant nematode species have been identified. A survey of sheep farmers in the south-east of Scotland conducted in 2000 indicated

Table 2.9 In vitro bioassays for the diagnosis of anthelmintic resistance.

Egg hatch assay 	Faeces are washed through a series of sieves before retaining nematode eggs on a 38 μm mesh. The eggs are then washed before incubating in a standard range of concentrations of thiabendazole or water-only controls for 48 hours at 22°C (24°C for *H. contortus*). Unhatched eggs and larvae (L_1) are then killed and stained with helminthological iodine, before counting to determine the percentage hatched in the different concentrations of thiabendazole. Benzimidazole resistance is deemed to be present if more than 50 percent of the eggs hatch in 0.1 μg/ml thiabendazole.
Larval development assay 	Nematode eggs are extracted and incubated for 24 hours in water at 22°C (24°C for *H. contortus*) to yield L_1. Aliquots of Baermannised L_1 are then transferred into wells of a 96-well plate, containing different concentrations of thiabendazole or levamisole and water-only controls in a 2 percent agar gel. The setup is then incubated at 22°C for 10 days before staining with helminthological iodine and counting the numbers of L_1, L_2 and L_3 in each well. Resistance is deemed to be present if more than 50 percent of the larvae develop to L_3 at a given drug concentration. Unfortunately, the setup is prone to desiccation or fungal infections, sometimes causing inconsistent results.
Larval feeding inhibition assay 	Nematode eggs are extracted from fresh faecal samples and incubated to yield L_1. About 100 Baermannised L_1 are then incubated for 2 hours in different concentrations of ivermectin, or in water-only control conditions, before fluorescein isothiocyanate labelled, lyophilised *E. coli* are added and the L_1 are incubated for a further 15 to 20 hours at 22°C (24°C for *H. contortus*). Larval feeding is determined by the observation of fluorescence in the pharynx and gut of the L_1 under an inverted fluorescent microscope, enabling the production of dose-response curves and calculation of discriminatory doses that indicate the presence of anthelmintic resistance. Unlike the EHA and LDA, the difference in discriminatory doses between anthelmintic-sensitive and -resistant nematode populations is only 2- to 3-fold, potentially resulting in inaccuracy.
Larval migration inhibition assay 	The LMIA involves determination of the numbers of exsheathed L_3 that migrate or fail to migrate through a 25 μm mesh during a 12-hour incubation period in different concentrations of ivermectin at 37°C. Unfortunately the concentrations of ivermectin involved are much higher than those involved in the sheep host, so the results may not represent the true anthelmintic resistance status of the nematode population.

that benzimidazole resistance may now be present in as many as 81 percent of lowground flocks, 56 percent of upland flocks and 45 percent of hill flocks. Populations of *T. circumcincta* resistant to all three anthelmintic groups (benzimidazoles, imidazothiazoles and avermectins) were identified in a goat flock in the south of Scotland during the 1980s and have subsequently been identified in several sheep flocks, including a terminal sire flock. The nematode control practices adopted by this particular flock, which might select for anthelmintic resistance, were similar to those used in most UK terminal sire flocks, from which sheep have been disseminated to

Fig. 2.29 The identification of triple anthelmintic resistance in terminal sire flocks is cause for concern. Sheep from these flocks are dispersed annually throughout the UK.

commercial flocks throughout the country (Fig. 2.29). *T. circumcincta* carrying genes conferring multiple anthelmintic resistance are therefore probably present in commercial sheep flocks throughout the UK, but remain unrecognised because they are present at a low frequency that cannot be detected using current insensitive diagnostic tests, and because few UK sheep farmers routinely check the efficacy of the anthelmintic which they are using.

The emergence of anthelmintic resistance is an inevitable consequence of good nematode control, and not a result of bad farming practice. Anthelmintic-resistant nematodes are no more pathogenic than non-resistant nematodes, so resistance itself is not production limiting. However, effective nematode parasite control in the face of multiple anthelmintic resistance is complicated, usually involving fundamental and sometimes expensive changes to sheep production systems. Nevertheless, in most cases, gastrointestinal nematodes can still be adequately controlled and it is important to keep the existence of multiple anthelmintic resistance in individual flocks in perspective.

The genetic basis of anthelmintic resistance

Significant populations of anthelmintic-resistant nematodes may be introduced to sheep flocks with sheep or goats, or selected from individual resistant nematodes which are probably already present at an extremely low frequency within all flocks. In the absence of exposure to anthelmintics, individual nematodes carrying genes expressing resistance to a particular anthelmintic or combination of anthelmintics probably have no survival advantage compared with the vast majority of genetically anthelmintic-susceptible nematodes. However, whenever anthelmintics are used to kill susceptible nematodes in their sheep or goat host, faeces produced during the subsequent pre-patent period contain mostly eggs of the surviving resistant nematodes, which consequently contribute to a greater proportion of succeeding generations. The evolution of resistance is determined by the extent to which nematodes which survive anthelmintic treatment contribute their genes to future generations. The risk of development of resistance depends on its genetic basis (whether resistance is controlled by a single major gene or by several genes, and whether these genes are dominant, partially dominant or recessive).

The genetic basis of resistance by different nematode parasites to different anthelmintic groups is not fully understood. In the case of benzimidazole resistance, single

nucleotide polymorphisms have been identified resulting in a single amino acid substitution at position 200 of the polypeptide encoded by the isotype 1 β-tubulin gene from tyrosine to phenylalanine. However, while polymorphism in the p200 codon of *T. circumcincta* is associated with survival of homozygous resistant genotypes, some heterozygous and homozygous susceptible genotypes also survive, implicating the involvement of other resistance mechanisms (for example involving a tyrosine to phenylalanine mutation at the p167 position of the β-tubulin isotype 1 gene), or of non-specific genetic pathways involving P-glycoproteins or cytochrome P450, involved with drug metabolism and excretion.

For ivermectin resistance it is likely that resistance will be associated with multiple functional polymorphisms in genes coding for glutamate-gated chloride channels, γ-aminobutyric acid receptors and the P-glycoproteins, but relevant polymorphisms have not been consistently identified and the potential role of other neutral markers involving drug regulatory mechanisms cannot be discounted.

Strategies which may reduce the rate of development of anthelmintic resistance

Once resistance to an anthelmintic group has emerged within an individual sheep flock, parasitic gastroenteritis can no longer be controlled using any of the drugs belonging to that anthelmintic group. Reversion to susceptibility does not occur within flocks, even after a period of 20 years or more of not using the anthelmintic group to which resistance is present. While lowground farmers can change from uneconomic sheep production to cereal cropping in response to unsustainable nematode control, the options for many hill and upland farmers are limited. There is therefore a need for all UK sheep farmers to establish effective nematode control programmes which ensure satisfactory animal production, while preserving the efficacy of the remaining anthelmintic groups, in particular the macrocyclic lactones.

Management practices to delay the onset of anthelmintic resistance in UK flocks are not clearly defined. Most recommendations are based on theoretical principles, or on practical experience of anthelmintic resistance in Australia, New Zealand and South Africa, where sheep management, and the epidemiology of parasitic gastroenteritis, may not reflect the situation in the UK. Unfortunately, the sensitivity of current diagnostic tests for anthelmintic resistance is inadequate for the detection of subtle changes in the frequency of resistant genotypes associated with different management practices. Furthermore, the emergence of anthelmintic resistance may prove to be an inevitable consequence of nematode control using anthelmintics. Nevertheless, action must be taken now based on the best information currently available.

The probability should be considered that anthelmintic resistance is already present in most UK flocks, albeit at an extremely low and clinically insignificant level. It is therefore important to ensure that these resistant nematodes are not afforded any survival advantage as a result of flock nematode control practices.

The selection pressure for anthelmintic resistance is influenced by:

- the frequency and timing of anthelmintic treatment
- the anthelmintic dose rate
- drug efficacy
- the life expectancy and fecundity of the adult nematodes

Fig. 2.30 This picture of a pen of ram lambs repeatedly nibbling at their accessory digits and the plantar aspects of their fetlocks was taken at a major ram sale. These animals were almost certainly infested with chorioptic mange mites, which are an unnecessary nuisance and potential cause of reduced ram breeding soundness. Chorioptic mange is clinically apparent, but its presence in a group of sale lambs raises a question about what other, clinically unapparent, sheep disease pathogens could be introduced to a sheep flock with purchased animals. For example, are they carrying multiple anthelmintic-resistant nematode parasites?

- the proportion of the susceptible population exposed to the anthelmintic compared with that on pasture
- the parasite generation time.

Anthelmintic treatment of introduced sheep and goats

It is important to focus attention both on slowing the rate of development of anthelmintic resistance within a flock, and avoiding bringing in significant numbers of resistant nematodes with introduced sheep or goats. In the absence of any sensitive, rapid and accurate diagnostic test for anthelmintic-resistant nematodes in individual sheep, all introduced sheep and goats should be assumed to be sources of multiple anthelmintic resistance (Fig. 2.30). All introduced sheep and goats should, therefore, be treated with an effective anthelmintic on arrival and yarded for 48 hours to ensure that any viable nematode parasite eggs have been voided before they are turned onto pastures which might be grazed by sheep within the next 6 months. Ideally, introduced sheep should then be turned onto likely contaminated pasture, so that any resistant nematode parasites that survive anthelmintic treatment only make up a very small proportion of the otherwise susceptible population in refugia. Entry of stray sheep or goats should be prevented and basic biosecurity should be imposed to ensure that sheep or goat faeces are not brought onto the farm.

The choice of anthelmintic for quarantine treatment is not straightforward. Resistance to the benzimidazole anthelmintics is already widespread, so for most flocks the main reason for quarantine anthelmintic treatment is to prevent the introduction of macrocyclic resistant nematodes, as this group may become the only remaining useful class of anthelmintic. The current options are to use a combination of anthelmintic drugs with different mechanisms of action, or to use moxidectin.

Anthelmintic drug combinations

The use as quarantine drenches of full-dose combinations of injectable macrocyclic lactones, which would also afford some control of sheep scab, and another anthelmintic with a different mechanism of action is widely promoted for quarantine treatment of introduced sheep although there are no UK field data to support this strategy.

Case report

Triple anthelmintic resistance was identified in a terminal sire flock during 2004 (Fig. 2.31). Provision of safe grazing was impractical, so during 2005 nematode parasite control was based on treatment of lambs with a sequentially dosed combination of ivermectin and levamisole anthelmintics in response to the identification of high FWECs. A FECRT was performed in October using a group of ewe lambs to monitor the efficacy of the anthelmintic combinations. Pre- and post-treatment coprocultures were performed on pooled faecal material to identify the nematode genera present and improve the efficacy of the undifferentiated FECRT. The results are shown in Table 2.10.

While the post-treatment efficacies for the anthelmintic combinations in the undifferentiated FECRT were sufficient to provide adequate nematode parasite control, the post-treatment efficacies for ivermectin, ivermectin/benzimidazole and ivermectin/levamisole combinations against *Teladorsagia* spp. were 0, 76 and 70 percent respectively. These results confirm the presence of ivermectin resistance, and provide evidence of resistance to the two anthelmintic drug combinations. Under these situations, further selection for drug combination resistance is inevitable. Furthermore, these results indicate that full-dose combinations of ivermectin and benzimidazole or levamisole anthelmintics may be ineffective in removing ivermectin-resistant *T. circumcincta* from introduced sheep.

Fig. 2.31 Triple anthelmintic resistance has been identified in flocks similar to this.

Table 2.10 FECRT results.

Treatment group	Mean pre-treatment FWEC (± SD) (epg)	Mean post-treatment FWEC (± SD) (epg)	Day 12 post-treatment efficacy
Ivermectin (n = 8)	443 (± 343)	150 (± 287)	66%
Ivermectin & Benzimidazole (n = 8)	600 (± 423)	50 (± 107)	92%
Ivermectin & Levamisole (n = 8)	469 (± 251)	50 (± 80)	89%
Coproculture results	35% *Teladorsagia*, 35% *Trichostrongylus*, 30% *Chabertia*/*Oesophagostomum*	100% *Teladorsagia*	

Moxidectin

The macrocyclic lactone group of anthelmintics includes the avermectins (ivermectin and doramectin) and milbemycin (moxidectin). These anthelmintic drugs all have structural similarities and share some mechanisms of action, but differ in their potency. Moxidectin is much more liphophilic than the other macrocyclic lactone drugs, becoming concentrated in the sheep's body fat soon after administration, creating a reservoir from which it is then slowly released. The high potency of moxidectin compared with that of ivermectin means that a much lower concentration of moxidectin is required to kill macrocyclic lactone-susceptible nematode parasites. Oral formulations of moxidectin and ivermectin are both administered at a dose rate of 200 μg/kg. However, the concentration of the less potent ivermectin that is subsequently released from the sheep's fat is too low to kill nematode parasites, while that of the more potent moxidectin is sufficient, affording persistence for about 4 weeks against *T. circumcincta* and *H. contortus*. This persistence is reduced in thin sheep with low body fat reserves.

The presence of side resistance between the avermectins and milbemycin is illustrated by experimental efficacy studies, which have shown that 31 times the dose of moxidectin may be required to kill a population of ivermectin-resistant *T. circumcincta* than to kill an ivermectin-susceptible population. However, the dose of moxidectin required to kill the ivermectin-resistant *T. circumcincta* remained lower than the standard dose rate of 200 μg/kg, indicating that in the short term moxidectin could be used as a quarantine drench to control ivermectin-resistant nematodes. Furthermore, experimental critical efficacy studies have shown that when moxidectin is administered orally at the manufacturer's recommended dose rate of 200 μg/kg, it is more than 98 percent effective against a UK ivermectin-resistant population of *T. circumcincta*.

The preservation of efficacy of moxidectin against ivermectin-resistant nematode populations within the host while its persistent activity is reduced is related to the fact that the drug is effective at the manufacturer's recommended dose, but as blood concentrations fall, as occurs soon after dosing when the drug is redistributed in the body fat reserves, they become too low to kill incoming resistant nematodes. The apparent effectiveness of oral moxidectin at a dose rate of 200 μg/kg in removing ivermectin-resistant *T. circumcincta* from their host might indicate that it would be effective as a quarantine anthelmintic drench. However, the reduced persistence against *T. circumcincta* indicates the early expression of resistance, against which background the routine use of moxidectin alone as a quarantine drench will rapidly further select for resistance and is imprudent.

The need for quarantine anthelmintic treatment applies equally to all introduced sheep and goats, including animals returning from grazings away from home and purchased animals. Replacement rams may pose a particularly high risk because they may have been treated frequently with anthelmintics to gain a production advantage, and underestimation of their body weights is common. In the absence of any new anthelmintic group, the choice of anthelmintic treatment regime to reduce the risk of introduction of macrocyclic lactone-resistant nematode parasites presents a dilemma. The efficacy of moxidectin as a quarantine treatment for triple-resistant nematodes will prove to be unsustainable, while full-dose combinations of ivermectin and levamisole may be ineffective. The best current advice may be to treat

Case report

At the beginning of April 2002, 90 ewes and 130 lambs of 1 to 38 days old were turned onto pasture from which multiple anthelmintic-resistant *Teladorsagia* had been identified during the previous year. The ewes were all orally dosed with moxidectin 10 days after turnout and again when they were removed from the pasture following weaning. All of the lambs were dosed orally with moxidectin at weaning and at 6-week intervals thereafter.

The FWECs of 20 ewes, 14 days after moxidectin treatment, were all zero, while their mean FWECs 28 and 35 days after treatment were 65 epg (range 0–550 epg) and 230 epg (range 0–950 epg) respectively. All of the larvae recovered following coproculture belonged to the genus *Teladorsagia*. The FWECs of all 90 ewes about 21 days after moxidectin treatment at weaning were all zero.

Oral moxidectin treatment of the lambs failed to suppress their FWECs beyond 21 days post-treatment. By August, the mean FWECs of 30 lambs, 28 and 35 days after moxidectin treatment, were 203 (range 0–2250) and 730 (range 0–5750) epg respectively. Coprocultures of pooled faecal material confirmed the predominance of *Teladorsagia*. Scour and poor performance consistent with clinical parasitic gastroenteritis were seen in some lambs within 28 days of anthelmintic treatment (Fig. 2.32).

Thus, the treatment regime including oral dosing of the ewes with moxidectin to control their periparturient rise in FWECs, and suppressive oral dosing of lambs with moxidectin at 6-week intervals throughout the summer, which might have been expected to control ivermectin-susceptible *T. circumcincta*, failed to prevent the establishment of significant numbers of infective larvae on the pasture and achieve the primary objective of parasitic nematode control.

While oral dosing with moxidectin was apparently effective in removing adult female burdens of ivermectin-resistant *T. circumcincta* from their host, positive FWECs from 21 days post-treatment and the coproculture results provide evidence that the drug achieved no persistence against the UK ivermectin-resistant population of *T. circumcincta*. (In the absence of anthelmintic resistance, oral moxidectin at a dose rate of 200 μg/kg would be expected to achieve about 28 days' persistence against incoming *T. circumcincta* larvae. The minimum pre-patent period for *T. circumcincta* is about 16 days, so faecal *Teladorsagia* egg counts would be expected to be zero for at least 44 days after oral moxidectin treatment.)

Fig. 2.32 Ill thrift due to parasitic gastroenteritis, despite suppressive moxidectin treatment and good nutrition.

introduced sheep sequentially with a full-dose combination of moxidectin and levamisole, or with a combination of all three anthelmintic groups, as is routinely practised in Australia and New Zealand. Given this uncertainty surrounding quarantine anthelmintic treatments, it would be prudent to routinely monitor their efficacy, so as to enable the timely implementation of alternative nematode parasite control strategies, should triple-resistant nematodes be introduced.

Monitoring anthelmintic efficacy

Strategies such as the use of moxidectin, sequential dosing with different classes of anthelmintics, and targeted anthelmintic treatments in response to FWECs have generally proven ineffective for the control of gastrointestinal nematode parasites in flocks where triple resistance was first identified as a cause of serious lamb ill thrift. The first UK flock in which triple resistance was identified in 2001 has now been sold (Fig. 2.33), while other affected farms have had to adapt to suboptimal productivity or are now considering fundamental, expensive changes to their flock management, such as adoption of safe grazing, or earlier lambing and lamb creep feeding to ensure that most lambs are finished before pathogenic nematode burdens develop on pasture. Unfortunately, the practical and economic sustainability of these strategies is by no means guaranteed.

In those flocks where triple anthelmintic resistance was only identified following routine monitoring, economic lamb production has mostly been sustained. Thus, the need to identify the anthelmintic resistance status of nematode parasites in individual sheep flocks is clear.

Recommendations aimed at ensuring that nematodes are exposed to an effective anthelmintic concentration

Exposure of nematodes to sub-therapeutic drug concentrations increases the selection pressure for benzimidazole and imidazothiazole resistance, where the genetic basis is incompletely recessive or partially dominant. In these situations, homozygous susceptible (SS) and heterozygous (RS) nematodes are killed at the full therapeutic dose, while homozygous resistant (RR) nematodes survive. RR nematodes are extremely rare in unselected populations, but sub-therapeutic drug concentrations enable RS nematodes, which are initially much more common, to survive, thus increasing the frequency of resistant alleles in subsequent populations.

Fig. 2.33 Multiple anthelmintic resistance was identified in sheep grazed in this field during 2001. While lowground farmers can change from uneconomic sheep production to cereal cropping in response to unsustainable nematode control, the options for many hill and upland farmers are limited. Maintenance of anthelmintic efficacy in UK flocks is therefore essential.

Fig. 2.34 Failure to store anthelmintic drugs correctly may result in reduced efficacy.

Under-dosing

Under-dosing due to inaccurate judgement of sheep bodyweights and faulty dosing guns is commonplace. While this may have little immediate economic effect on animal production, it inevitably selects for anthelmintic resistance. Poor drenching technique, miscalculation of the correct dose volume and use of inaccurate weigh scales compounds the problem. Most benzimidazole and macrocyclic lactone drenches have a wide safety margin so, if necessary, it is preferable to overestimate the required dose volume.

Under-dosing may also arise following incorrect storage of anthelmintic drugs (Fig. 2.34), use of expired product, mixing incompatible drugs or chemicals before dosing, or use of products of dubious origin.

Drug bioavailability

The efficacy of drugs against nematode parasites can be altered by the effects of disease or management on the physiology of their host. Anthelmintic bioavailability may be reduced due to rapid flow of digesta through the intestines of scouring lambs, or due to the effects of liver disease, for example associated with subacute fascioliasis or ovine white liver disease, on drug metabolism.

The efficacy of benzimidazole and oral macrocyclic lactone anthelmintics is dependent on the duration of nematode exposure to a therapeutic drug concentration, and can be enhanced by prolongation of the drug's plasma concentration profile. (The efficacy of levamisole is determined by the initial concentrations achieved.)

Within 2 hours of oral administration, benzimidazole and macrocyclic lactone anthelmintics become largely associated with particulate digesta in the ruminoreticulum. The residence time of the drug-digesta complex in the ruminoreticulum and the subsequent flow rate of the digesta through the gastrointestinal tract contribute significantly to the rate of drug absorption and recycling and to the duration of anthelmintic availability. The duration of anthelmintic availability is, therefore, potentially shortened if the oral dose bypasses the ruminoreticulum due to closure of the reticular groove (Fig. 2.35). The duration of the particulate digesta-associated anthelmintic reservoir is determined by the rate of flow of digesta from the ruminoreticulum, which is influenced by feed type, being shorter for fresh green feed compared with conserved rations, but also responds quickly to a reduction in feed intake, and is about halved within 24 hours of halving the feed intake. Thus a significant interaction exists between feed intake and anthelmintic efficacy.

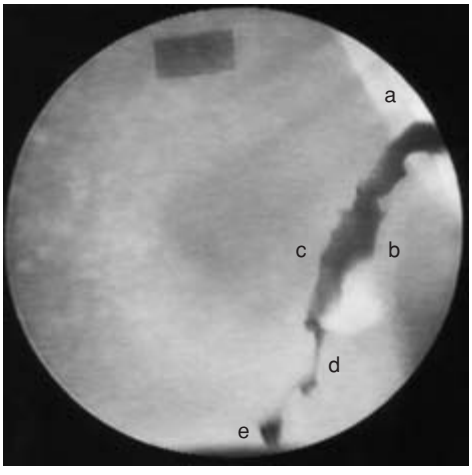


Fig. 2.35a Fluoroscopic image of contrast medium-labelled drench (*dark*) reaching the distal oesophagus (a), bypassing the ruminoreticulum (b) via the reticular groove (c) and omasal canal (d) and being deposited directly into the abomasum (e) of a lamb that had been yarded for 24 hours before drenching.

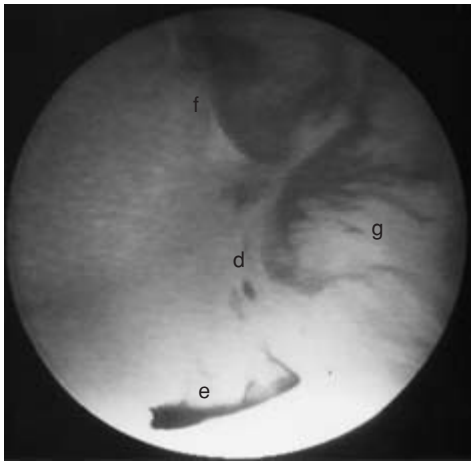


Fig. 2.35b Fluoroscopic image for comparison of contrast medium-labelled drench in the cranial sac of the rumen (f), the reticulum (g), the omasum (d) and abomasum (e) following partial forestomach bypass in a lamb that had been yarded for 24 hours before drenching.

In some circumstances, reducing feed intake by yarding for 24 hours before and 8 hours after drenching extends the plasma profile of benzimidazole and macrocyclic lactone drenches (Figs 2.36 and 2.37). In the presence of resistant nematode parasites, this may restore the anthelmintic efficacy to a practically useful level. This practice may also reduce the risk of emergence of anthelmintic resistance. However, under certain grazing conditions, for example where sheep are grazed on lush green pasture, their ruminoreticulum contents become fluid after yarding for a period of 24 hours. This has been shown to result in closure of the reticular groove following oral dosing in about 35 percent of weaned lambs, diverting the dose into the abomasum rather than the ruminoreticulum and potentially reducing the efficacy of benzimidazole or macrocyclic lactone anthelmintics (Fig. 2.38). However, the importance of reticular groove closure with regard to anthelmintic efficacy in UK sheep is uncertain.

Fig. 2.36 The practice of yarding sheep for 24 hours before anthelmintic drenching, with restricted access to feed but free access to water, has been promoted to enhance the efficacy of the drench.

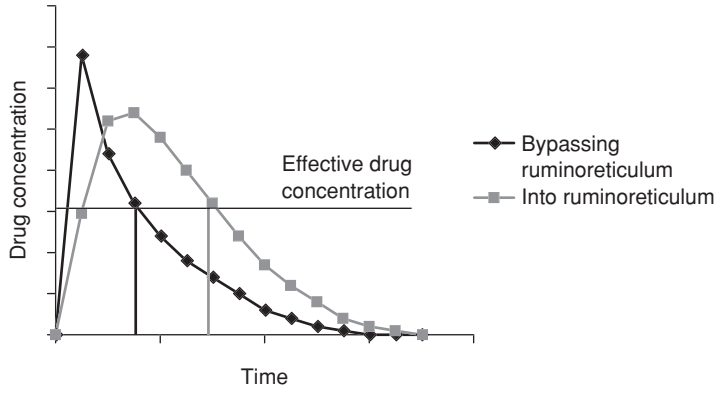


Fig. 2.37 The effect of reduced feed intake before dosing.

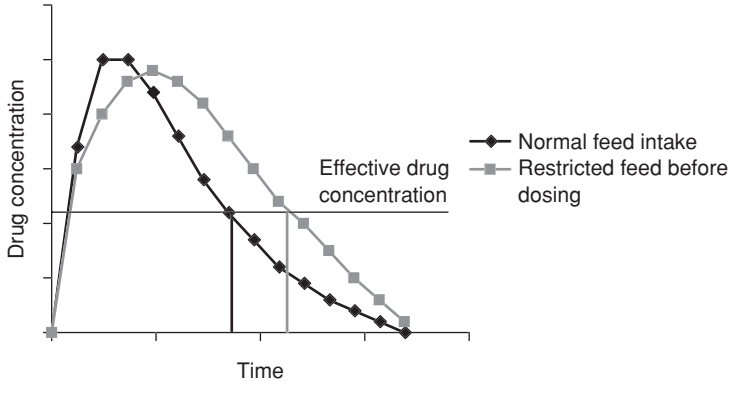


Fig. 2.38 The effect of ruminoreticulum bypass on anthelmintic efficacy.

Recommendations aimed at the timing and frequency of anthelmintic treatment and ensuring that only a small proportion of the nematode population is exposed to the anthelmintic

The rate of selection for anthelmintic resistance by a nematode parasite species is influenced by the proportion of its total population which is exposed to the drug. The greater the proportion of the nematode population exposed to the drug in its sheep host compared to that on pasture at the time of anthelmintic treatment, the faster the selection for resistance. Thus, the rate of selection for anthelmintic resistance is inversely proportional to the percentage of the total parasite population that is on pasture as eggs and developing larvae (i.e. in refugia) at the time of treatment. If the proportion of free-living nematode parasites at the time of anthelmintic treatment is large, then the offspring of resistant nematodes are diluted, but if the nematode population in refugia is small, then the offspring of resistant nematodes will constitute a larger proportion of the next generation. This relationship between the size of the nematode population in refugia and that in its host is further influenced by factors which affect the survival and rate of development of the free-living nematode stages.

The objective of nematode control is to limit exposure of susceptible sheep to infective larvae on pasture. Thus, the rate of selection for anthelmintic resistance is highest on those farms which achieve the most effective nematode control. Some guidelines aimed at slowing the emergence of anthelmintic resistance, therefore, inevitably involve a compromise between achieving adequate nematode parasite control and reducing the rate of selection for anthelmintic resistance.

Extend the interval between anthelmintic treatments

Suppressive control of nematode parasites aimed at preventing pasture contamination with eggs and developing larvae frequently involves conventional anthelmintic treatment of susceptible sheep at intervals close to the parasite's pre-patent period. This high-frequency treatment strategy has been an important factor in the emergence of anthelmintic resistance. Anthelmintic usage and selection for resistance could be reduced by extending the suppressive conventional anthelmintic treatment interval, although this strategy may not prevent production losses due to parasitic gastroenteritis. This dilemma might be addressed by basing the timing of anthelmintic treatments on monitoring of FWEC data. However the success of this strategy depends on the level of parasite challenge, the stocking density, geography and weather conditions on individual farms. During spring and early summer on heavily stocked farms, lamb FWECs can increase rapidly, so substantial pasture contamination may already have occurred by the time that intervention targets are identified. FWEC monitoring can be useful to determine the optimal timing of suppressive anthelmintic treatments on some farms where the level of pasture larval challenge is likely to be low, as a result of summer drought conditions or low stocking densities, but intervention targets must be based on previous knowledge of the epidemiology of parasitic gastroenteritis in the individual flock. Thus, the principal reason for monitoring FWECs is to establish the pattern of pasture larval contamination on individual farms, providing a basis for future nematode control strategies.

Avoid unnecessary anthelmintic treatments

Reducing the reliance on anthelmintics by removing unnecessary treatments, while maintaining good control of parasitic gastroenteritis, is intuitive. In many flocks, ewes are treated with an anthelmintic before mating in the belief that the practice might improve lambing percentages. The benefits of this strategy are that if the ewes are in poor body condition, anthelmintic treatment before mating can lead to improved lambing percentages, provided that they are subsequently well fed. Anthelmintic treatment before mating can also provide some insurance against the effects of the possible introduction of new nematode parasite species (Fig. 2.39). However, the disadvantages associated with anthelmintic treatment of ewes before mating are that it does not lead to improved lambing percentages where ewes are already in good body condition, incurring unnecessary expense and effort. Furthermore the practice possibly selects for anthelmintic resistance by exposing parasites to anthelmintics at a time when a significant proportion are in a hypobiotic, hard-to-kill state, and by affording surviving resistant nematodes with a prolonged period during which they dominate egg production in immune sheep. It is therefore impossible to make general recommendations about anthelmintic treatment of ewes, apart from during their periparturient period. Risks must be assessed for individual flocks. The decision can be aided by monitoring of ewe FWECs, although these are difficult to interpret

Fig. 2.39 It is important to weigh up the risks for and against anthelmintic treatment of ewes before mating. Routine anthelmintic treatments before mating can provide some insurance against the unknown presence of *H. contortus*, which has the potential to cause clinical disease later during the year. These gimmers are ill thrifty associated with a winter outbreak of haemonchosis. They had been grazed away from home during the previous summer and were not treated with anthelmintic on their return before mating.

without previous knowledge of the flock. For example, in some cases low counts can become significant when the total daily faecal output of ewes is taken into account. In other cases, high counts may be insignificant due to the predominance of less pathogenic large intestinal nematodes, or the fact that larvae hatched from eggs shed by immune ewes may be less fit, and therefore less likely to reach the L_3 stage, than those hatched from eggs shed by naïve sheep. In some cases, the best strategy may be only to treat lean ewes before mating. This strategy avoids unnecessary anthelmintic usage and permits limited pasture contamination by eggs of susceptible nematodes shed by the untreated ewes.

Treatment of lambs may be unnecessary when they are grazed on safe pasture, or later in the year when they have acquired some immunity. In these situations, the decision to treat lambs or not can be supported by monitoring of FWECs.

Integrated nematode parasite control

Reliance on anthelmintics may be reduced by the use of safe grazing. On many farms, the opportunity exists to introduce safe grazing for a proportion of the sheep flock by alternating annually between arable cropping or cattle grazing and sheep grazing. However, while the use of safe grazing can provide excellent control of parasitic gastroenteritis in the short term, the practice selects for anthelmintic resistance when it is integrated with anthelmintic treatments, such as in a traditional dose-and-move system. This system results in a situation whereby the new pasture is contaminated only with nematodes which survive anthelmintic treatment and are then afforded a prolonged period of reproductive advantage over susceptible nematodes. While the subsequent level of pasture larval contamination remains low, providing excellent short-term nematode control, the nematode population in refugia becomes predominantly anthelmintic resistant. Over a period of time, these anthelmintic-resistant nematodes are multiplied in susceptible sheep that graze on the pasture, resulting in potentially hard-to-manage parasitic gastroenteritis (Fig. 2.40).

All of the recommendations concerned with anthelmintic treatments before a move onto safe pasture involve a compromise between nematode control and selection for anthelmintic resistance. This risk of selection for anthelmintic resistance may be reduced by leaving a proportion of the flock untreated. It may be possible to target anthelmintic treatments to scouring or lighter-weight lambs, leaving faster-growing animals untreated. Alternatively, the move onto safe grazing can be delayed for about 5 days after conventional anthelmintic treatment. When anthelmintic treatment

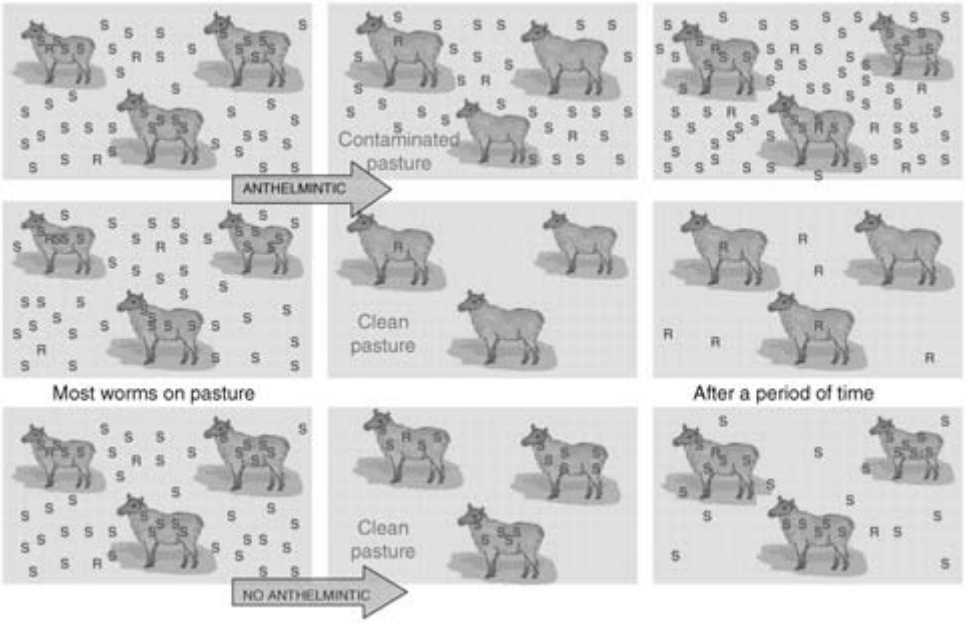


Fig. 2.40 When sheep are returned to contaminated pasture after anthelmintic treatment, only a small increase occurs in the proportion of resistant (R) genotypes in refugia. However, this strategy does not satisfactorily control parasitic gastroenteritis, because susceptible sheep are exposed to large numbers of infective larvae after treatment. Movement to safe pasture after anthelmintic treatment affords excellent control of parasitic gastroenteritis, but selects for anthelmintic resistance, because the proportion of resistant genotypes in refugia is increased. Not treating sheep with an anthelmintic before movement onto clean pasture does not select for anthelmintic resistance and can control parasitic gastroenteritis in the short term, because susceptible sheep are not exposed to infective larvae, but enables rapid contamination of the previously safe pasture, albeit with predominantly susceptible (S) genotypes.

is considered essential before a move to safe grazing, sheep grazing of the pasture should be avoided during the following season.

Targeted selective treatments

Individual sheep differ in their tolerance to the same level of infective larval challenge, with some animals suffering reduced production and developing high FWECs, while others develop low FWECs. In South Africa, where natural selection for tolerance to haemonchosis has occurred over a period of several decades, less than 30 percent of the animals in some flocks may develop clinical signs of disease and contribute significantly to pasture contamination. An effective system referred to as FAMACHA© has been tested in South Africa whereby anthelmintic treatments are targeted to members of the flock that are least tolerant to haemonchosis, based on the monitoring of conjunctival colour for signs of anaemia. Using this system the number of anthelmintic treatments can be substantially reduced, while maintaining a reasonable level of flock productivity. Studies in France have shown that the highest milk-producing goats have highest FWECs and that milk production can be used as an indicator for targeted selective anthelmintic treatment. When applied to flocks or herds with a high proportion of nematode-tolerant animals, these approaches ensure a reasonable level of parasite control, substantially reduce anthelmintic treatment

costs, and importantly ensure that a population of susceptible nematodes is maintained in refugia.

When UK sheep are exposed to *T. circumcincta* infective larvae, individual animals show different liveweight gain responses, with the least tolerant subsequently developing the highest FWECs. However, changes in liveweight gain can be detected before FWECs increase, providing a potential indicator for targeted selective treatments. Field studies have been performed comparing targeted selective treatments based on fortnightly weighing to determine changes in liveweight gain with a 4-weekly neo-suppressive anthelmintic treatment regime. Preliminary results suggest that while targeted selective treatments may result in slightly poorer short-term productivity than neo-suppressive treatments, they select more slowly for anthelmintic resistance.

Targeted anthelmintic treatment regimes may prove to be essential in order to preserve anthelmintic efficacy in UK sheep flocks. However such approaches based on maintaining a susceptible nematode population in refugia are more complex than conventional whole-flock treatment regimes, and are only likely to be adopted if their theoretical basis is understood.

Anthelmintic treatment of periparturient ewes

Successful control of gastrointestinal nematode parasitism in UK lambs generally starts with anthelmintic treatment of ewes at lambing to limit the periparturient rise in FWECs. A single oral treatment at lambing with moxidectin removes nematode parasites from the ewes and enables them to remove overwintered infective larvae from the pasture without themselves further contributing to subsequent pasture *T. circumcincta* contamination for a period of about 6 weeks. Under certain circumstances, this practice may select strongly for macrocyclic lactone resistance. UK pasture burdens of overwintered infective larvae vary from year to year, associated with environmental temperature and humidity, the nematode burden accumulated over the previous grazing season, and the period for which the pasture was rested from *T. circumcincta*-parasitised sheep. While the population of overwintered infective larvae in refugia can be very high, it may also sometimes be extremely low, incurring a high selection pressure for resistance when periparturient ewes are unnecessarily dosed with moxidectin before moving onto pasture. Selection for resistance by moxidectin at the time of dosing would have been unlikely, due to its high efficacy against resident adult nematodes, but its poorer efficacy against larvae with ivermectin-resistant genotypes may enable selection for resistance during the protection period after drug administration.

Annual drench rotation

The use of a different broad-spectrum anthelmintic group each year is widely promoted. However, epidemiological evidence and field data to positively support this strategy are not currently apparent. Mathematical modelling simulating control of *T. colubriformis* under Australian conditions by two anthelmintic drug groups with independent resistance genes indicated that, in the long term, there was no difference in the rate of evolution of resistance between strategies involving rotation at each drench, annual drench rotation, 5-, or 10-year rotation. In the absence of reversion to anthelmintic susceptibility, the presence of resistance was merely hidden when the alternative anthelmintic was being used.

While annual drench rotation is not contraindicated and may indeed be shown to be beneficial under UK conditions, the perceived need to adhere to an annual anthelmintic group rotation may interfere with good nematode parasite control practice, for example involving the choice of anthelmintic treatment for quarantine treatment of introduced sheep or periparturient ewes. Rather than adhering strictly to the annual rotation of anthelmintic groups, consideration should be given to ensuring that the most appropriate drug is used for each anthelmintic treatment.

Avoid keeping sheep and goats on the same farm

Goats metabolise anthelmintic drugs more rapidly than sheep and may also show a higher incidence of ruminoreticulum bypass, reducing the effective drug concentration. Furthermore, adult goats do not become fully immune to nematodes and may harbour significant numbers of abomasal parasite EL_4. These differences in the pharmacokinetics of anthelmintics and epidemiology of nematode parasites between sheep and goats mean that some drugs which are effective in sheep struggle to kill nematode parasites in goats, selecting for resistance. Sheep and goats are host to many of the same nematode parasite species, so selection for anthelmintic resistance in goats presents a significant risk to sheep production.

Management of parasitic gastroenteritis in the presence of anthelmintic resistance

There are no straightforward solutions for the management of parasitic gastroenteritis in the face of high levels of triple anthelmintic resistance. There are differences between farms in terms of environment, nutrition, management and nematode population size, composition and resistance. Nematode parasite control programmes therefore need to be developed for individual flocks, based on an understanding of their epidemiology, control methods and of the principles involved with anthelmintic resistance.

In most flocks, production losses to parasitic gastroenteritis cannot be prevented without some reliance on anthelmintic drenches, although sustainable control of nematode parasites depends on the development of methods which do not rely totally on anthelmintics. Practical solutions for UK flocks might include:

- Creep feeding or provision of alternative forage crops to finish lambs early, before pasture infective larval burdens become production limiting.
- Development of strategies involving preparation of pastures for susceptible sheep using cereal crops or prior grazing with cattle or older ewes.
- Manipulation of strategies to increase the proportion of anthelmintic-susceptible nematodes in refugia.
- Development of molecular or serological tests which might help to select for host resistance or resilience to nematode parasites.
- Manipulation of nutrition and body condition scores to enhance natural immunity.

Full-dose anthelmintic combination drenches can extend the application of the drugs involved when compared to their use independently and have proven useful overseas for the management of parasitic gastroenteritis on farms with benzimidazole resistance.

Benzimidazole-resistant nematodes possess a variant form of tubulin which only binds weakly to the benzimidazole, and once the concentration of the drug falls, their benzimidazole-tubulin complex dissociates. In the short term, benzimidazole

anthelmintics may become more effective against resistant nematodes when the parasite is exposed to an effective concentration of the drug for a longer period. Similar principles apply to macrocyclic lactone anthelmintics, but not levamisole or morantel. Two to three full anthelmintic doses, repeated 12 hours apart for benzimidazoles or 36 to 48 hours apart for ivermectin, have substantially better efficacy against resistant nematodes than a single treatment at double or treble the recommended dose rate. However, this practice is unlikely to prove to be practical or sustainable.

Controlled-release albendazole and ivermectin capsules are not available in the UK but are widely used overseas. The controlled-release capsules prolong the equilibrium between anthelmintic and tubulin. The anthelmintic efficacy against adult resistant nematodes is slightly improved, but the main advantage of the controlled-release capsules is against more sensitive incoming larvae. When benzimidazole resistance is present, parasitic gastroenteritis can be controlled in lambs by treatment with an alternative effective anthelmintic at the same time as bolus administration. The effective anthelmintic removes the adult benzimidazole-resistant parasite population, while the controlled-release albendazole is effective against subsequent pasture-acquired larvae. In ewes, controlled-release capsules are most effective if given pre-lambing when the parasite burdens are low.

The prospect of a new class of anthelmintic drug

During the last 25 years, no new class of anthelmintic drug for use in production animals has reached the market. Advice on sheep management to minimise the risk of selection for anthelmintic resistance is mostly based on the principle that this situation will continue. However, pharmaceutical drug discovery programmes have identified various potentially useful anthelmintic compounds, such as the paraherquamides, cyclodepsipeptides and amino-acetonitrile derivatives (AADs), and there is now a realistic probability that the development of one or more of these compounds will result in a marketable product within the foreseeable future. Any new drug will inevitably be proven to be effective against nematode parasites that are resistant to the existing anthelmintic groups, and could therefore provide a solution for the control of multiple-resistant nematodes. However, it is possible that certain mechanisms conferring resistance to a new drug may already be present in nematode populations at a higher level than they were before the introduction of the existing drugs, and that the emergence of resistance to the new drug will be rapid. It is therefore important that any new anthelmintic drug is used responsibly – as a quarantine treatment and strategically in an attempt to preserve the efficacy of the existing anthelmintic groups, rather than as a panacea for nematode control.

Cestode parasites

The common cestode parasites of UK sheep are *Monezia expansa*, *Taenia hydatigena* and *Taenia multiceps*. These parasites seldom cause economically significant disease in sheep flocks. *Taenia ovis* is a potentially important cause of carcase condemnation at meat inspection and hydatid disease due to *Echinococcus granulosus* infection is an important zoonosis. The definitive host for *M. expansa* is sheep and the definitive hosts for the other ovine cestode parasites are canids.

Cestode parasites (M. expansa) *with sheep as their definitive host*

M. expansa segments are often seen in lamb faeces (Fig. 2.41). Tapeworm segments (proglottids) are large (about 10 mm × 2 mm × 2 mm), white and obvious. Tapeworms do not actively feed from their host; instead they absorb nutrients through their cuticle from the lumen of the intestine. Tapeworms clearly grow body mass at the expense of their host, so it is understandable that questions are raised about their possible harmful effects whenever large numbers of tapeworm segments are seen. Tapeworms have been blamed for a variety of problems, including digestive disorders, scour and dags, and poor liveweight gains (Fig. 2.42). However, there are no substantial trial results to support claims of any harmful effects associated with *M. expansa* infection. In fact, overseas experiments have shown no adverse effects following experimental infection with large numbers of tapeworms, or controlled studies where half of the flock were dosed with a drug specifically to remove tapeworms.

M. expansa eggs are released from segments before or after they have been voided with faeces. Further development only occurs when the eggs containing the first-stage larvae (onchospheres) are eaten by oribatid mites on the pasture. Eggs develop to second-stage larvae (cysticercoids) in the body cavity of the mite over a period of 15 to 30 weeks. Most of the tapeworm population overwinters in the forage mites and seasonal fluctuations in the incidence of sheep tapeworm infection are associated with the activities of the mites during the summer months. Sheep become infected when they ingest mites along with forage, following which the tapeworm grows to maturity over a period of about 6 weeks. Adult tapeworms are short-lived and patent infections only persist for about 3 months.

The benzimidazole anthelmintics – fenbendazole, albendazole and oxfendazole – are usually effective against *M. expansa*. However, levamisole, morantel, ivermectin, doramectin and moxidectin do not remove tapeworms. A combination product including levamisole and praziquantel is marketed in the UK for the treatment of the adult stage of *M. expansa* in sheep.

Fig. 2.41 Tapeworm segments in the faeces of a hill lamb.

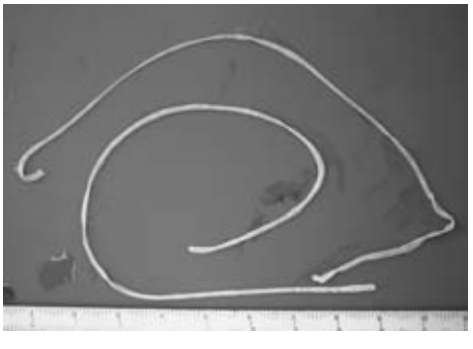


Fig. 2.42 Although adult *M. expansa* tapeworms in the small intestine are large and obvious, there is little evidence that they have any effect on production.

Cestode parasites with sheep as their intermediate host

Sheep can serve as intermediate hosts for a number of dog and/or wild canid intestinal tapeworms. The most important are *T. ovis*, *T. multiceps*, *T. hydatidigena* and *E. granulosus*. These tapeworms can cause clinical disease in sheep, result in downgrading or condemnation of carcases at meat inspection, and can present serious human health risks.

Sheep and other intermediate hosts are usually infected when they ingest tapeworm eggs containing first-stage larvae (oncospheres) with pasture contaminated by infected canid faeces. Oncospheres then penetrate the intermediate host's gut wall and travel via the blood to different target organs, where they develop into second-stage (cysticercus, coenurus or hydatid) larval cysts, which occupy normal tissue and may result in clinical disease. Dogs are in turn infected by eating cysts in tissues of infected intermediate host carcases. Dogs do not show signs of clinical disease, but adult tapeworms in their intestinal tracts shed thousands of eggs daily. In addition, intact tapeworm segments are periodically voided, each containing up to 250 000 eggs.

Coenurosis (gid)

Gid remains a common cause of neurological disease in sheep in parts of Wales, western England and Ireland, although the prevalence appears to be lower than during the 1980s, putatively associated with increased awareness, routine tapeworm treatment of farm dogs, not feeding uncooked sheep meat to farm dogs, and prompt disposal of fallen stock.

Ingested *T. multiceps* oncospheres are carried in the blood to the brain or spinal cord, where they develop into coenurus (gid) cysts (Fig. 2.43). Mature cysts take up to 8 months to develop, measuring up to 5 cm in diameter, with clusters of scolices on their internal wall. The onset of clinical signs ranges from 2 to 6 months following oncosphere ingestion.

Most cases of gid are seen in sheep between 6 and 24 months old. The onset of clinical signs is insidious and the disease is slowly progressive. The clinical signs are determined by the site of the cysts within the central nervous system. Survey data have shown that about 80 percent of cysts occur in one cerebral hemisphere (coenurus cerebralis), 10 percent in the cerebellum and 8 percent at multiple locations. Individual coenurus cysts have been reported within the spinal cord.

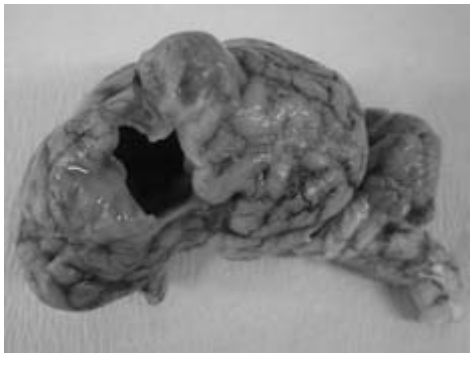


Fig. 2.43 A large coenurus cyst involving the left cerebral hemisphere of a lamb.

Coenurus cysts within the cerebrum are characterised by blindness in the contralateral eye and proprioceptive defects involving the contralateral limbs. Affected sheep often circle compulsively, narrow circles indicating a deep lesion and wide circles indicating a superficial lesion. Animals generally circle towards the side of superficial cysts and away from deeper cysts. Coenurus cysts in the frontal part of the cerebrum can also cause depression and head pressing. In some cases, the presence of cerebral cysts is accompanied by localised softening of the frontal bone of the skull, although this does not provide a precise guide to the location of the cyst.

Coenurus cysts within the cerebellum usually result in bilateral proprioceptive defects. Affected sheep appear dysmetric and ataxic, with a wide-based stance, but preservation of limb strength. The clinical signs associated with cerebellar cysts usually progress more rapidly than those associated with cerebral cysts.

The precise site of most coenurus cysts can be identified on the basis of a thorough neurological examination. The important differential diagnoses for coenurosis are brain abscessation, listeriosis, louping ill, sarcocystosis, polioencephalomalacia, hypomagnesaemia and pregnancy toxaemia. With the exception of some cases of brain abscessation, these can mostly be discounted on the basis of history and detailed neurological examination. The diagnosis of gid is confirmed on the basis of either postmortem findings or a successful response to surgical treatment.

In most cases, a decision is made to humanely kill affected animals. However, a good response is reported to surgical removal of superficial coenurus cerebralis cysts, following accurate localisation. Surgery is performed under general anaesthesia and involves boring a 1 cm hole through skull to expose the brain. Following incision of the dura, the cyst can be visualised and drained before removal of the cyst wall using forceps. Postoperative anti-inflammatory and antibiotic treatment is required.

Acute coenurosis has been seen in sheep about 10 days after introduction to pastures heavily contaminated by dog faeces, associated with migration of large numbers of oncospheres through the brain. The clinical signs include nervousness and head aversion, leading to convulsions and death within 3 to 5 days in severe cases.

Abdominal cysticercosis (Cysticercus tenuicollis)

Abdominal cysticercosis is caused by the second larval stage of *T. hydatigena*, a common intestinal tapeworm of dogs in the UK. Oncospheres ingested by the intermediate sheep host are carried in the blood to the liver, where they migrate for about 4 weeks before attaching to the omentum, mesentery or peritoneal surface of the abdominal organs. Larval cysts up to 8 cm across (Fig. 2.44) develop either singly or in small groups, but while infection is common, clinical disease is rarely seen. Most *Cysticercus tenuicollis* cysts are seen only as incidental findings at postmortem examination or meat inspection.

The migration of large numbers of developing cysticercae through the liver of young sheep occasionally causes abdominal pain and anaemia, which rapidly progresses to death. The syndrome is referred to as hepatitis cysticercosa and has many clinical and pathological similarities to subacute liver fluke (Fig. 2.45).

Occasionally greenish subcapsular 1 cm diameter nodules are identified in livers as incidental postmortem findings. These lesions are associated with the death of developing cysticerci during their migratory phase.

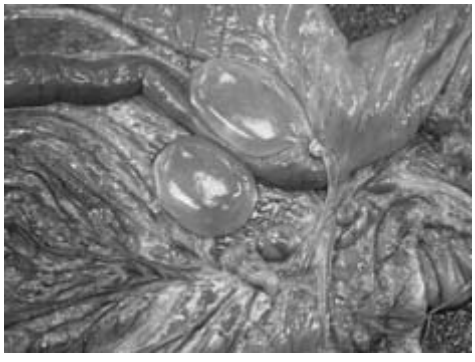


Fig. 2.44 Two *Cysticercus tenuicollis* cysts attached to the mesentery of a 6-month-old lamb.

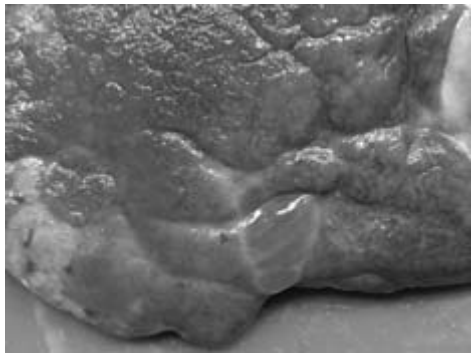


Fig. 2.45 Haemorrhage and inflammation of the liver, with formation of a subcapsular cyst, caused by large numbers of migrating cysticercae.

Cysticercus ovis

Cysticercus ovis is caused by the second larval stage of the dog tapeworm *T. ovis*. Infected dogs can void up to 750 000 *T. ovis* eggs daily. Dogs do not develop significant immunity to the tapeworms, which often survive in their intestines for 6 months. A single infected dog can, therefore, spread infection over a large area. *T. ovis* eggs from dog faeces can also be spread by flies. Oncospheres ingested by the intermediate sheep host are carried in the blood to muscles, in particular the heart and diaphragm, where they develop to form numerous 1- to 2-mm diameter cysts. Many *Cysticercus ovis* cysts die as the sheep mount an immune response, forming firm, white nodules.

The prevalence of *Cysticercus ovis* is high in New Zealand, where the disease, referred to as sheep measles, is considered to be important because of aesthetic objections to the appearance of cysts in sheep meat and economic losses associated with rejections from export consignments. *Cysticercus ovis* is managed in New Zealand by bimonthly treatment of farm dogs with cestocides, a regime which was encouraged by an industry-financed education programme.

Hydatid disease

The current incidence of hydatid disease in UK sheep is extremely low and it is crucially important that this status is maintained due to its serious zoonotic implications. Elsewhere in southern and eastern Europe, South America and Africa, hydatid disease is common in sheep, goats and deer.

Hydatid disease is caused by larval stages of the dog and wild canid tapeworm *E. granulosus*. *E. granulosus* oncospheres can survive in the environment for up to 2 years. Oncospheres ingested by intermediate hosts penetrate the gut wall and are carried in the portal blood to the liver or in the lymphatics to the lungs (Fig. 2.46). Occasionally oncospheres enter the general circulation and are carried to other organs. About 70 percent of sheep hydatid cysts are found in the lungs, 25 percent in the liver and the remainder in other organs.

Multiple hydatid cysts in the liver and lungs grow up to 20 cm in diameter over a period of 6 to 12 months, while those in the peritoneal cavity can become enormous.

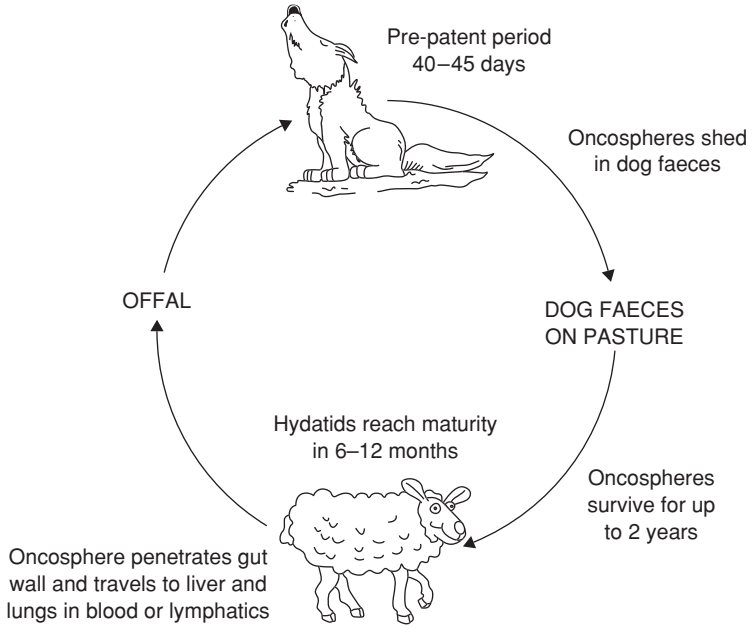


Fig. 2.46 The life cycle of *Echinococcus granulosus*.

Brood capsules containing numerous budded scolices are seen in the cyst walls, and the cyst fluid has a grainy appearance due to the presence numerous free scolices.

The clinical signs of hydatid disease are determined by the organs involved. Hydatid cysts are often present in the lungs or liver without causing clinical signs, only being found at meat inspection. However, cysts at other sites can cause severe dysfunction. Sudden death sometimes occurs due to an anaphylactic reaction to ruptured cysts.

When humans are involved as an intermediate host, hydatids can result in life-threatening pulmonary or hepatic disease. Most human infections are acquired by eating vegetables contaminated by dog faeces, or due to failure to wash hands after petting infected dogs. However, infection may also arise from accidental ingestion or inhalation of cyst contents during postmortem examination of infected sheep carcases.

Control of cestode infections

Gid, abdominal cysticercosis, sheep measles and hydatid disease are controlled by preventing access to sheep carcases by dogs. While foxes are sometimes implicated in transmission, they are less efficient than dogs as definitive hosts.

Farm dogs should be treated with a cestocide every 6 to 8 weeks. Praziquantel is effective against all of the common dog tapeworms, including *E. granulosus*, while nitroscanate is effective against *T. ovis* and *T. hydatigena*, but not against *E. granulosus*. Dogs should be confined for 48 hours after treatment and any infected faeces collected and carefully disposed of.

Recent outbreaks of gid have been seen, despite the diligent application of preventive measures on the farms involved. These cases were probably associated with stray

dogs or public access, but highlight the need for a concerted approach to tapeworm control on a district level.

Liver fluke

Liver fluke caused by *Fasciola hepatica* is a major parasitic disease of sheep in many countries worldwide. In western parts of the UK, the disease is a traditionally important annual cause of ill thrift and deaths of ewes and lambs during autumn and early winter. Recently, following prolonged wet weather, the disease has been reported earlier in the year in western areas and has also become important in eastern regions where it may be unrecognised and cause significant losses. In parts of western Scotland and Ireland, a different fluke, *Dicrocoelium dentriticum*, also assumes importance.

Life cycle of F. hepatica

F. hepatica is a trematode parasite with a two-host life cycle involving mud snails of the genus *Lymnea*, mostly *Lymnea truncatula*, and a variety of mammalian species including sheep, cattle, goats, deer and rabbits (Fig. 2.47).

Sheep become infected when they ingest metacercariae which are encysted on pasture. The cysts hatch in the small intestine before immature flukes eat through the intestinal wall and migrate through the peritoneal cavity to the liver. This migration

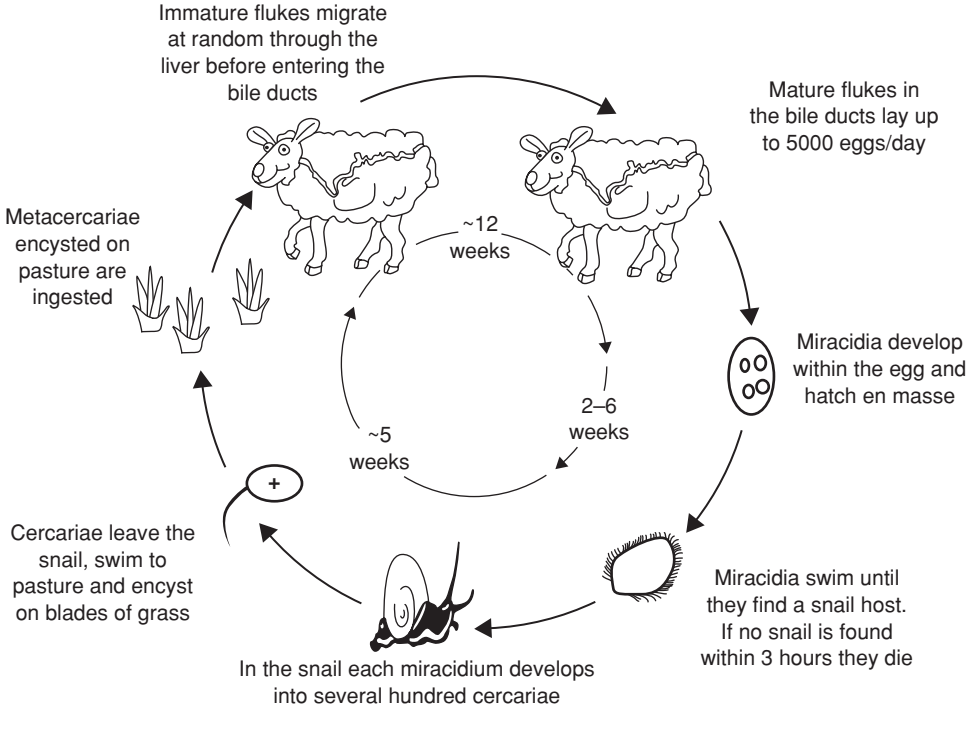


Fig. 2.47 The life cycle of *F. hepatica*.

to the liver takes about 24 hours. Immature flukes then migrate at random through the liver for 4 to 6 weeks before reaching the bile ducts and maturing to adults. Adult liver flukes in the bile ducts lay eggs from about 12 weeks post-infection. An adult female liver fluke can lay up to 5000 eggs/day from about 12 weeks post-infection.

When temperatures are above 10°C miracidia develop within the egg. At temperatures of 15°C miracidia hatch about 6 weeks after eggs are shed, but at temperatures above 22°C hatching can occur after only 10 days. Thus, given suitable conditions of temperature and moisture, hatching can occur en masse. Free-living miracidia swim until they find a snail host, but if a host is not found within 3 hours they die. Each miracidia develops in the snail's intestinal tract and liver through sporocysts and rediae to give rise to several hundred cercariae. Under optimum conditions of temperature and humidity the development in the snail from miracidia to cercariae takes about 5 weeks. Cercariae leave the dying snail, swim to pasture and encyst on blades of grass as metacercariae. Metacercariae can survive for a year, but most are killed by desiccation, displacement or prolonged freezing.

L. truncatula is a small (5 to 10 mm long) brown snail which inhabits ponds and slightly acidic marshy areas. Areas trampled by cattle provide ideal snail habitats, but they do not survive in fast running water. Snail populations drop to low numbers during the winter, but rapidly multiply in spring and summer when temperatures are between 15°C and 26°C. *L. truncatula* can survive dry periods by burrowing into mud and aestivating, re-emerging when moist conditions return. Cercariae are sometimes released from re-emerging snails en masse.

Thus, the seasonal appearance of fascioliasis is determined by the effects of moisture and temperature on the snail populations and free-living stages of *F. hepatica*. At temperatures below 10°C fluke eggs do not develop and hatch and snail development is halted. Under typical UK conditions, the only time when temperatures and moisture are adequate for both snails and fluke is between April and November. Typically, snails are infected in late spring and early summer by miracidia deposited during the previous autumn and winter, or in the same spring. Cercariae from this infection encyst as metacercariae in late summer and autumn. Sometimes snails are infected in winter from fluke eggs shed in late summer and early autumn and produce cercariae during the following spring.

Adult *F. hepatica* can survive for as long as their sheep host and, unlike the situation in cattle, sheep are susceptible to re-infection throughout their lives.

Clinical signs

Acute disease is caused when grazing sheep ingest very large numbers of metacercariae, with invasion of the peritoneum and liver parenchyma by thousands of migrating immature flukes. The first evidence of a problem may be the sudden death of previously healthy sheep, while other animals are seen with reduced grazing activity and signs of lethargy, pallor, abdominal pain and dyspnoea (Figs 2.48 and 2.49). Acute fascioliasis typically occurs from September to December, but occasionally, when sheep are not introduced to dangerous pasture until later in the year, the disease is seen as late as February. Sudden deaths, as a result of haemorrhage and liver damage, may occur when animals are handled.

Subacute disease results from ingestion of metacercariae over a period of several weeks or months. The disease typically occurs from December onwards but may be

Fig. 2.48 Severe abdominal pain in a Texel ewe lamb due to acute fascioliasis.

Fig. 2.49 A moribund Soay lamb with acute fascioliasis.

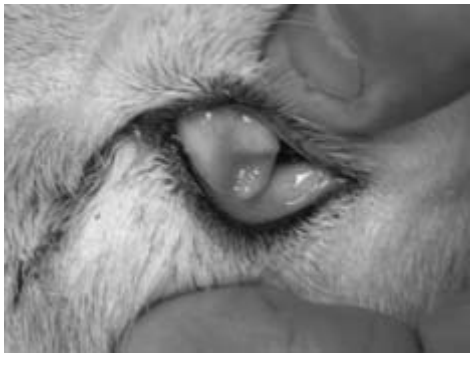


Fig. 2.50 Anaemic ocular mucous membranes due to subacute fascioliasis.

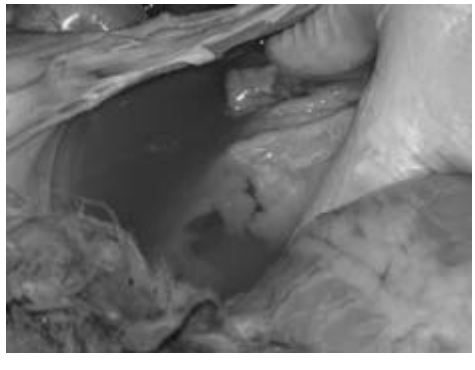


Fig. 2.51 Postmortem appearance of ascites associated with subacute fascioliasis.

seen much earlier with severe challenge. The major presenting clinical finding is poor body condition score. Affected sheep show marked anaemia most noticeably affecting the conjunctivae (Fig. 2.50). Liver enlargement is reported but is not easy to appreciate during clinical examination. Ascites may be present (Fig. 2.51), but cannot usually be appreciated without recourse to ultrasonographic examination, except in extreme cases. Sudden deaths from black disease (Plate 2.4) or bacilliary haemoglobinuria (*Clostridium novyi* types B and D respectively) may occur in unvaccinated sheep.

Chronic disease is caused by the presence of adult flukes in the bile ducts of the liver (Fig. 2.52). The chronic disease is usually seen from February to March onwards (new cases occasionally occur in the summer associated with winter infection of snails) and is characterised by poor body condition (Fig. 2.53), poor fleece quality and death. Anaemia, ascites and submandibular oedema (Fig. 2.54) are present in advanced cases. Affected sheep may die in an emaciated state, especially when infestation is compounded by the metabolic demands of advanced pregnancy.

In many cases where infection has occurred over a prolonged period, liver fluke disease may be due to the presence of both migrating larvae and adult flukes, characterised by ill thrift, lethargy and dyspnoea in ewes and lambs between December and March.

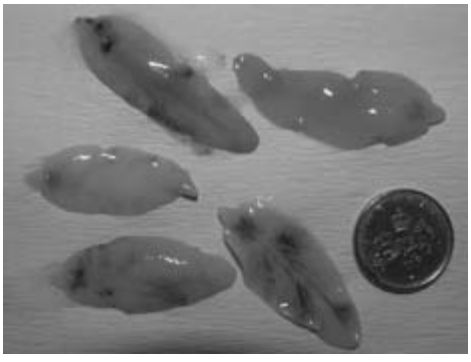


Fig. 2.52 Adult *F. hepatica* from the bile ducts.

Fig. 2.53 Poor body condition in ewes due to chronic fascioliasis.

Fig. 2.54 Submandibular oedema due to hypoalbuminaemia resulting from chronic fascioliasis.

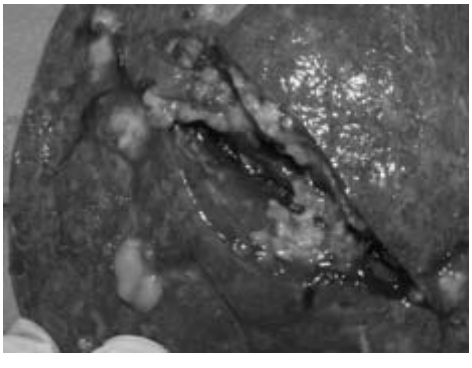


Fig. 2.55 Haemorrhagic tracts and abscessation caused by migrating *F. hepatica* larvae.

Pathology

Acute or subacute fascioliasis is caused by migrating immature flukes. On postmortem examination the liver is friable and has a mottled appearance. Haemorrhagic tracts are present throughout the parenchyma, which may appear autolysed, even in fresh carcases (Plate 2.5 and Fig. 2.55). Blood-stained fluid is present in the peritoneal cavity and occasionally there may be extensive, severe, fibrinous peritonitis. The carcases of animals with severe liver damage are usually anaemic due to haemorrhagic blood loss (Figs 2.56 and 2.57).

Chronic fascioliasis results from impaired liver function and anaemia caused by the presence and blood-feeding activity of adult flukes in the bile ducts. On postmortem examination, varying degrees of hepatic fibrosis are seen. Raised areas of fibrosis around the bile ducts may be seen on the liver surface (Fig. 2.58) and the liver may be irregularly shaped due to compensatory hypertrophy of less affected areas. Mature, 1- to 3-cm long, leaf-shaped, adult flukes are found in the bile ducts and gallbladder (Fig. 2.59).

Diagnosis

The diagnosis of acute or subacute liver fluke is based on the history of sudden deaths or ill thrift in recognised fluke areas at the relevant times of year. The diagnosis is

Fig. 2.56 An anaemic carcase with evidence of blood-stained fluid in the peritoneal cavity, fibrinous peritonitis and severe hepatitis.

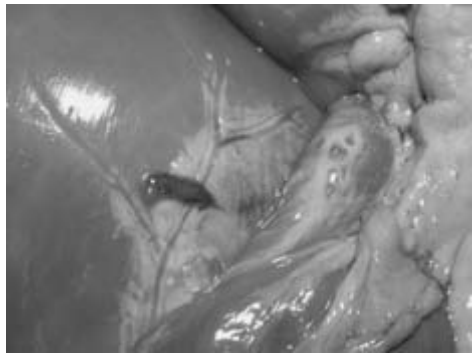


Fig. 2.57 Large numbers of immature flukes are sometimes seen on the surface of the peritoneal viscera.

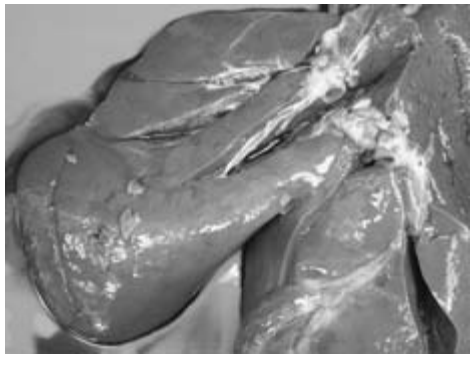


Fig. 2.58 Raised areas of fibrosis surrounding the bile ducts. Adult *F. hepatica* can be seen oozing from the cut surface of a hyperplastic bile duct.

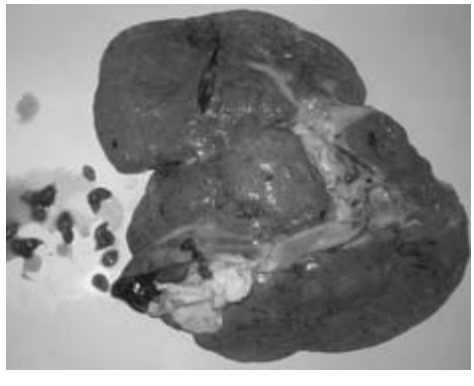


Fig. 2.59 An irregular-shaped liver, with both parenchymal haemorrhage and bile duct hyperplasia, caused by concurrent subacute and chronic fascioliasis.

usually confirmed by the characteristic postmortem findings. The 3- to 10-mm long migrating flukes may be identified under a dissecting microscope after squeezing a section of liver into water (Fig. 2.60).

Subacute fascioliasis usually results in profound hypoalbuminaemia (serum concentrations of 10 to 20 g/L; normal range 28 to 34 g/L) and hyperglobulinaemia

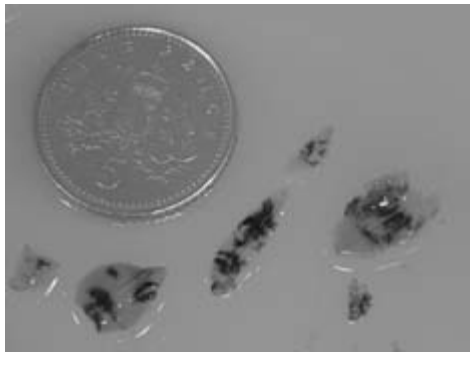


Fig. 2.60 Different sized immature liver flukes squeezed from a cut section of liver.

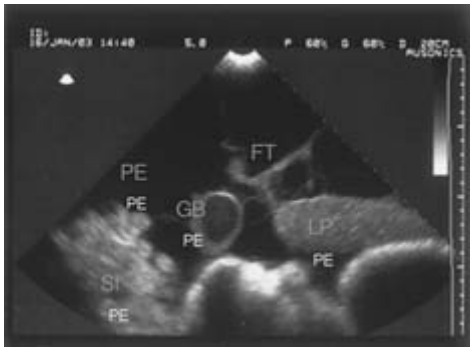


Fig. 2.61 Ultrasonographic image using a 5 MHz sector transducer connected to a real-time, B-mode ultrasound machine. The liver parenchyma (LP) has a granular appearance with multiple hyperechoic dots. The hepatic vessels and bile ducts are poorly defined. The small intestines (SI) are freely moveable and their shape and outline continuously change with the passage of digesta, associated with their being surrounded by peritoneal exudate (PE). Fibrin tags (FT) and adhesions are seen as broad hyperechoic lines.

(serum concentrations of 65 to 80 g/L; normal range 38 to 42 g/L). Serum concentrations of aspartate aminotransferase (AST), gammaglutamyl transferase (GGT) and glutamate dehydrogenase (GLDH) are raised. GLDH concentrations are typically up to 30 times their normal value (150 to 300 iu/L; reference range 2 to 10 iu/L), while alterations in AST and GGT concentrations are less dramatic.

While serum concentrations of albumin, globulin and AST return to normal values within 10 days of effective treatment, GLDH concentrations remain elevated for more than 4 weeks. Blood sampling to determine GLDH concentrations is therefore useful when it is necessary to investigate poor performance after treatment for subacute fascioliasis.

Subacute fascioliasis is associated mostly with the presence in the liver parenchyma and peritoneum of thousands of migrating early and later immature flukes. However, a few young adult flukes are sometimes found in the bile ducts and gallbladder. Thus, the diagnosis can occasionally be supported by the identification of small numbers of fluke eggs in faecal samples.

The diagnosis of subacute fascioliasis can be supported by real-time ultrasonographic examination, with the probe applied immediately caudal to the costal arch and approximately two-thirds of the way down the right side (Fig. 2.61). Ultrasonography of subacute fascioliasis cases shows accumulation of peritoneal exudate and extensive fibrinous adhesions between the liver and small intestine or body wall. The liver is enlarged and its capsule has a hyperechoic appearance due to fibrin deposition. The liver parenchyma has an abnormal diffuse granular appearance, observed as multiple hyperechoic dots throughout its substance, while the liver of normal sheep has a uniform hypoechoic appearance with anechoic areas sharply demarcated by narrow hyperechoic lines representing hepatic vessels and bile ducts.

Chronic fascioliasis can be confirmed in the live animal by the identification of fluke eggs in faeces (Fig. 2.64). However, unlike the situation with parasitic gastroenteritis, the severity of disease cannot be related to the fluke egg count and failure to identify fluke eggs does not rule out a diagnosis of chronic fascioliasis. Various quantitative and non-quantitative, sedimentation and filtration methods can be used for the identification of fluke eggs in faeces.

The presence and blood-feeding activity of adult flukes in the bile ducts results in impaired liver function and anaemia, with packed cell volume values as low as 0.06 L/L. Hypoalbuminaemia and hypergammaglobulinaemia are seen in advanced cases. GGT levels are typically elevated to 2 to 5 times normal levels.

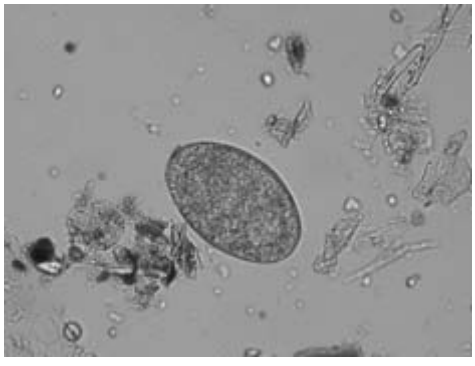


Fig. 2.62 *F. hepatica* eggs seen following water sedimentation are oval shaped, about 100 μm long and brown-coloured (due to faecal staining), with a distinct operculum at one pole.

A sedimentation method for the identification of liver fluke eggs in faeces

The sedimentation method described below is straightforward and can be performed in any farm animal practice (Fig. 2.63).

- Place 2 to 5 g of faeces in a sieve over a bowl.
- Add about 100 ml of water and 2 drops of washing-up liquid and stir to break down faecal mass.
- Discard fibrous material which remains in the sieve.
- Transfer the remaining faecal suspension to a measuring cylinder.
- Leave to stand and sediment overnight.
- Discard the water while retaining the sediment.
- Add 100 ml of water and stir.
- Leave to sediment for a few hours.
- Discard the water.
- Examine the sediment under 100x microscopy for the presence of fluke eggs.

Fig. 2.63 Faecal sedimentation in water in a practice laboratory, using a discarded syringe case.

Table 2.11 Examples of sheep flukicides included in the National Office of Animal Health *Compendium of Animal Medicines* (2007) or non-participating manufacturer's lists.

Product	Active chemical	Dose rate and administration route	Spectrum of activity
Fasinex 5% or Combinex[1]	Triclabendazole	10 mg/kg oral drench	2 week immature to adults
Trodax 34%	Nitroxynil	10 mg/kg s/c injection	6 week immature to adult
Flukiver or Supaverm[1]	Closantel	10 mg/kg oral drench	6 week immature to adult
Levafas Diamond Fluke and Worm Drench[1]	Oxyclozanide	15 mg/kg oral drench	adults
Various benzimidazole anthelmintics	Albendazole	7.5 mg/kg oral drench	adults
Various benzimidazole anthelmintics	Netobimin	20 mg/kg oral drench	adults

[1] Combined with levamisole.

Antibodies against components of liver fluke can be detected using an ELISA test, although the test provides evidence of previous exposure rather than current infection and is not commonly used in sheep.

Treatment

While all of the flukicides licensed for use in sheep in the UK are effective against adult flukes, the drugs differ in their efficacy against immature *F. hepatica* (Table 2.11).

For the treatment of subacute fascioliasis it is necessary to use triclabendazole which has good efficacy against early immature flukes. When 'safe' dry pasture is available, a single treatment is given before moving the sheep. However, when such pasture is not available, repeated treatments at 3-week intervals are required throughout the high-risk period of autumn and early winter. Alternative flukicides, nitroxynil and closantel, only kill adult and immature flukes from 6 weeks, while oxyclozanide, albendazole and netobimin only kill adult flukes. These drugs are not useful for the treatment of subacute fascioliasis.

While some deaths may occur shortly after treatment, associated with stressful handling, the response to oral triclabendazole treatment is usually rapid. Provided that sheep do not become re-infected, reasonable subsequent growth rates can be expected.

Concern has been expressed in recent years that, in some cases, poor responses to triclabendazole treatment may be due to the emergence of flukicide resistance. Triclabendazole resistance has been confirmed in western areas of Ireland and Scotland, associated with the need in these wet areas for repeated treatments at 3-week intervals throughout the autumn and winter months.

The efficacy of triclabendazole is reduced in sheep with severe liver disease. Thus, treatment failure in severe outbreaks of subacute fascioliasis is not necessarily due to flukicide resistance. Confirmation of triclabendazole resistance during the treatment of subacute fascioliasis is problematic. The absence of adult flukes and fluke eggs in faeces precludes the use of FECRTs. Specialist tests are being developed to determine the presence of resistance by examining miracidial hatching and motility in the presence of triclabendazole, but are not generally available.

Fig. 2.64 Control measures aimed at avoiding snail and fluke habitats are often impractical, while in some cases these habitats are poorly defined.

Where triclabendazole resistance is suspected, the treatment of subacute fascioliasis relies on the use of flukicides such as closantel or nitroxynil, which are ineffective at removing early immature flukes and may not cure the disease. In these flocks, future control of liver fluke depends on the spring treatment of all grazing livestock with a flukicide other than triclabendazole, to remove adult fluke and thereby reduce miracidial infection of snails.

Control

The risk of fascioliasis can be predicted using epidemiological knowledge of life cycle and weather conditions and local knowledge of the disease. The standard recommendation for fluke control in known fluke areas is that: all sheep and cattle are strategically treated in late spring to remove adult flukes, in October/November to remove immature flukes and in January/February to remove immature and adult flukes; in years when a high risk of fascioliasis is forecast, extra strategic treatments are given 6 weeks after these standard treatments. However, in some high-risk areas sheep are treated at 3- to 4-week intervals from October to February.

Where possible sheep should be grazed away from wet areas during high-risk periods. Drainage and fencing off wet and boggy areas may help, but is often impractical (Fig. 2.64).

While it may be possible to eradicate liver fluke from a farm by strategic drenching, the disease is likely to be re-introduced with purchased animals or wildlife harbouring flukes, or with infected snails washed along watercourses from neighbouring ground.

Following a recent series of wet summers, subacute fascioliasis is now common in eastern parts of the UK, where the disease was not previously seen. Subacute fascioliasis is sometimes particularly serious in these areas because it is unexpected and preventive treatments are not routinely given. Once liver fluke has become established on farms, the risk of disease may persist despite dry summer weather, because infected snail populations may be maintained in microhabitats such as ditches and ponds. The need for future preventive treatments should therefore be based on assessment of the disease risk.

Dicrocoelium dendriticum

D. dendriticum is a small lancelate liver fluke (1.2 cm × 0.25 cm) which causes disease in sheep in a few islands off the west coast of Scotland and in parts of Ireland. This parasite is of interest because its life cycle differs from that of *F. hepatica*.

The miracidium is already present in the fluke egg when voided and can survive for several months on pasture. Miracidia are ingested by land snails, which don't require wet and boggy conditions. In the snail the parasite develops to cercariae, which are shed by the snails in slime balls. These are then eaten by ants, where they develop to metacercariae. Metacercariae in the ants' brain modify their behaviour, so that they climb onto herbage and are eaten by sheep. The metacercaria then hatch in the intestinal tract of the sheep and migrate along the bile ducts to the liver.

Clinical signs are related only to liver damage because the parasite does not migrate through the peritoneum. Anaemia and ill thrift are seen with severe infections.

Control of *D. dendriticum* is difficult because the eggs can survive for long periods on pasture. Control depends on regular routine flukicide treatments.

Ill thrift due to trace element deficiencies

The diagnosis of trace element deficiencies

Trace element deficiencies are a common cause of ill thrift in growing lambs. On many farms, some insurance against production loss is provided by indiscriminate use of expensive ad-lib free-access minerals, mineralised drenches and other forms of supplementation, although this can be wasteful, or even lead to toxicity and meat residue problems. Furthermore, as UK flock sizes become larger and returns per sheep lower, the luxury of such indiscriminate supplementation is likely to become unaffordable.

The potential economic benefit of trace element supplementation can seldom be determined by ad-hoc sampling of a few animals which are not thriving, or interpretation of individual biochemical results, but can be determined with confidence, through an understanding of appropriate sample collection and of the derivation of laboratory reference ranges.

Few of the clinical signs associated with trace element deficiency are specific (Table 2.12); for example, there are several causes of ill thrift or poor reproductive performance, including multiple trace element deficiencies. It is therefore essential

Table 2.12 Clinical signs associated with trace element deficiencies in farmed ruminants.

Copper	Cobalt	Selenium	Iodine
Ill thrift (C, D)	Ill thrift (S, G, D, C)	Ill thrift (S, C)	Poor reproductive performance (S)
Coat changes (C, S)	Watery ocular discharge (S, C)	White muscle disease (G, S, C)	Perinatal mortality (G, S, C)
Diarrhoea (C)	Anaemia (S)	Poor milk production (C)	Goitre (G, S, C)
Skeletal abnormalities (C, D, S)	Poor wool growth (S)	Poor reproductive performance (S)	
Poor milk production (C)	Reduced immune response (S, C)	Reduced immune response (S, C)	
Postparturient haemoglobinuria (C)	Poor milk production (C, S)		
Poor reproductive performance (C, D)	Perinatal mortality (S)		
Poor antler production (D)	Poor reproductive performance (S)		
Reduced immune response (D)			
Perinatal mortality (D)			
Enzootic ataxia (D, S)			
Anaemia (C)			

C; cattle: D; deer: G; goats: S; sheep, in order of prevalence.

Fig. 2.65 These ill-thrifty, scouring lambs have concurrent intestinal parasitism and cobalt deficiency. Determining whether the intestinal parasitism was exacerbated by cobalt deficiency, or the lambs became cobalt deficient due to poor mineral absorption as a consequence of parasitic gastroenteritis, presents a diagnostic challenge.

to determine the contributions of poor feed availability and quality, or gastrointestinal parasitism, or other diseases such as pneumonia and lameness. Furthermore, malnourished sheep and animals with enteric disease are more susceptible to the effects of trace element deficiencies than healthy sheep (Fig. 2.65). In these cases, the trace element deficiency is frequently resolved when the primary problem is corrected. This confusion is avoided through the adoption of a rational and scientific approach to determine the trace element status of animals.

As with all disease investigations, a relevant disease history, including information about previous diagnoses and trace element supplementation, is essential. Before collecting samples, consideration must be given to the following:

- Which trace elements are likely to be deficient?
- Is there a best time of year to sample?
- How many samples to collect?
- What samples to collect?
- Which species, age and production group of animals to sample?
- How to interpret the reference ranges?

In many situations, the number and types of samples required depends on the reason for the investigation, for example:

- routine monitoring
- investigation of poor performance problems
- determination of the adequacy of reserves for the next high-risk period
- determination of the need to spend money on supplementation.

Cobalt deficiency

Cobalt deficiency is a common cause of ill thrift in weaned, growing lambs. Deficiency in pregnant ewes has been associated with poor milk production and high perinatal lamb mortality rates. Weight gains following supplementation have been reported in goats and deer, although these species appear to be less susceptible to cobalt deficiency than sheep. Signs of cobalt deficiency occur infrequently in cattle.

Clinical signs

The principal sign of cobalt deficiency in lambs is ill thrift. Affected lambs are frequently open-fleeced and poorly fleshed, with hollow sublumbar fossae and distension

Fig. 2.66 Ill-thrifty cobalt-deficient lambs.

Fig. 2.67 Low-grade conjunctivitis in a cobalt-deficient lamb.

Fig. 2.68 Clinical signs including a lack of menace response, sudden-onset depression progressing to stupor, fine muscle fasciculations over the head, neck and ears, ataxia, and head pressing associated with ovine white liver disease.

of the ventral abdomen (Fig. 2.66). Low-grade conjunctivitis and a watery ocular discharge are often present (Fig. 2.67). Severely affected animals appear anaemic, although in these cases the differential diagnoses of haemonchosis and subacute fascioliasis should be investigated. Fatty infiltration of the liver (ovine white liver disease) with clinical signs of hepatic encephalopathy (Fig. 2.68) is sometimes seen in severely deficient weaned lambs.

Animal requirements

Dietary cobalt is converted to vitamin B_{12} by the microflora of the ruminoreticulum, released in the abomasum and absorbed in the small intestine. Vitamin B_{12} is required in the liver for the utilisation of propionic acid in energy production. Poor utilisation of propionic acid results in reduced appetite, so the principal clinical sign of cobalt deficiency is ill thrift due to inadequate feed intake. Vitamin B_{12} is also required for the metabolism of certain sulphur-containing amino acids which are necessary for optimum wool growth. This may partly account for the higher requirement for cobalt by sheep than cattle and deer, and for the fact that where sheep and cattle are grazed on similar pastures, deficiency may be diagnosed in the sheep flock but not in the cattle. Growing animals have a higher requirement for vitamin B_{12} than adults, but the requirements of pre-ruminant animals are low. Vitamin B_{12} readily crosses the placenta and is stored in the foetal liver. Concentrations in colostrum are also high.

Serum vitamin B_{12} concentrations in newborn lambs rise rapidly following ingestion of colostrum, but then gradually fall because milk contains less vitamin B_{12} than colostrum. Serum vitamin B_{12} concentrations remain low until the rumen develops. The requirements of pre-ruminant lambs are less than those of ruminants, because the pre-ruminant animals rely mainly on glucose as an energy source, while most energy in ruminant animals is derived by vitamin B_{12}-dependent gluconeogenesis from propionic acid.

Diagnosis of cobalt deficiency or sufficiency

Cobalt deficiency appears to be a bigger problem in some years than in others. Unfortunately, the causes are multifactorial, and it is difficult to predict a bad season in advance. Different plant species, poor clover growth or low soil cobalt can rapidly result in the onset of clinical disease.

Soil cobalt concentrations

Knowledge of local soil types can provide a general guide to the likelihood of cobalt deficiency occurring, but this alone is of limited value for the diagnosis of deficiency. Soils derived from acid igneous rocks such as granite are low in cobalt. Other elements such as manganese, iron and nickel can interfere with cobalt uptake by plants. Soil pH above the optimum range of 5.8 to 6.3 can affect cobalt availability and soil compaction may reduce pasture uptake of cobalt.

Pasture cobalt concentrations

Pasture cobalt concentrations are more useful than those of soil, but in common with most pasture mineral analyses are confused by soil contamination. Pasture cobalt concentrations are useful for monitoring the success of top-dressing programmes, as an adjunct to the interpretation of animal results, or to identify deficient areas of the farm that might be grazed by less susceptible groups of animals during periods of high risk of deficiency. Pasture cobalt concentrations can explain the cause of animal deficiency, but cannot be used alone to diagnose deficiency in animals. Pasture cobalt uptake is lowest when pasture growth is rapid and in mature pasture, corresponding with the spring and summer periods. Pasture grown on waterlogged soil has a higher cobalt concentration than pasture grown on well-drained soils (Figs 2.69 and 2.70). Plant species differ in their ability to accumulate cobalt, although when soil cobalt is low the concentrations in all plants are low. In general, the cobalt content of clover is greater than that of other pasture species.

Postmortem findings

The postmortem identification of fatty liver changes associated with ill thrift in growing lambs may support a diagnosis of cobalt deficiency, but these signs are not specific (Plate 2.6).

Dose-response trials

Arguably the most accurate method to diagnose cobalt deficiency is to perform a dose-response trial, comparing growth rates of supplemented animals with unsupplemented controls. The two groups need to be grazed and managed under the same conditions. However, such an approach is not always practical or appropriate.

Fig. 2.69 The hill grazing to the left of the fence has been improved, while that to the right has not. The risk of trace element deficiency is generally greater on the improved hill, associated with higher soil pH and drainage.

Fig. 2.70 The three smaller Scottish Blackface lambs in this picture were hefted on an improved hill, while the similarly managed larger Scottish Blackface and Crossbred lambs had strayed from an adjacent heft on unimproved hill. The smaller lambs are cobalt deficient, while the larger lambs are not.

Blood and liver vitamin B_{12} concentrations

Serum vitamin B_{12} concentrations reflect the immediate dietary cobalt intake, while liver vitamin B_{12} concentrations provide a guide to the limited continuous body storage. Serum vitamin B_{12} levels respond within a few days to an improvement in dietary cobalt, therefore values must be interpreted with caution. Yarding for a period of more than about 4 hours before sampling, liver damage and poor handling of samples can result in markedly elevated serum vitamin B_{12} concentrations. Furthermore, unless animals are severely deficient, their individual variation in serum vitamin B_{12} concentrations is high. It is therefore recommended that a minimum of ten serum samples be collected. There is less variation in liver vitamin B_{12} concentrations, and three samples are adequate.

Blood and urinary methylmalonic acid

Methylmalonic acid (MMA) accumulates in the plasma of cobalt-deficient sheep as a result of reduced vitamin B_{12} coenzyme activity. Raised concentrations of MMA have been used to support a diagnosis of cobalt deficiency. However reference ranges are not well defined, so MMA concentrations cannot be used to predict the probability of a response to cobalt supplementation.

The choice of diagnostic sample: serum vs. liver vitamin B_{12}

The most appropriate choice of animal samples depends on the reasons for sampling. In sheep, serum vitamin B_{12} concentrations provide information about the animal's current status and are useful for the diagnosis of clinical deficiency. Despite the poor hepatic storage of vitamin B_{12}, practical experience indicates that liver concentrations provide information about the animals' longer-term status and are useful to determine either the need for supplementation, or to monitor a supplementation programme. Liver samples collected from lambs at weaning or ewes pre-lambing provide a good indication of reserves to maintain the animals through the major risk

periods. Material collected from slaughterhouse animals probably provides useful information about the flock cobalt status, although animals which remain on the farm may have a lower cobalt status for a variety of reasons, such as the association between cobalt status and gastrointestinal parasite burdens. Overseas, liver samples are often collected from weaned lambs by biopsy, but the technique is not generally accepted in the UK and most laboratories are unable to analyse the small samples obtained.

Interpretation of reference ranges

The laboratory reference ranges for vitamin B_{12} are derived from the relationship of both liver and serum concentrations to weight gain responses following numerous controlled supplementation trials. The reference curves produced give an indication of the probability of a weight gain response to supplementation at a particular concentration of serum or liver vitamin B_{12}.

The interpretation of the results depends on the reasons for sampling. For the diagnosis of clinical deficiency, the laboratory reference values are used (normal ≥395 pmol/L). When samples are collected to determine what reserves are available before the likely period of deficiency, the values can be interpreted in the low end of the normal reference range, taking into account the effects of season and stage of the production cycle. Different age groups should have different reference ranges, due to the different importance in the role of vitamin B_{12}. Samples collected from pre-ruminant lambs at docking are difficult to interpret.

Weaned lambs are more sensitive to cobalt deficiency than cattle; therefore, where the two species are grazed on the same farm, and the cobalt status of the lambs is adequate, that of the cattle will also be adequate.

Cobalt/vitamin B_{12} supplementation

There are no recipes for trace element supplementation, therefore a detailed evaluation of each situation is required to determine the most appropriate programme. The most effective supplementation method varies from farm to farm, depending on soil type, topography, stocking rates, handling facilities and the availability of labour. The degree and duration of trace element deficiencies varies from farm to farm and between different years, therefore supplements which have been effective on one farm during one season may not work on another. Even in deficient animals, supplements only provide a small portion of the trace element requirements, most of which are absorbed from the diet. In many cases, the monitoring of supplementation programmes over a 2- to 3-year period is required before the most cost-efficient regimen can be determined for a flock. The cost of supplementation needs to be balanced against the expected increase in production.

Short-term supplements

Oral administration of cobalt salts

Oral drenching of weaned lambs with about 35 mg cobalt sulphate (for example, using 70 g of cobalt sulphate dissolved in 5 litres of water, then dosing lambs at 1 ml per 10 kg) raises their serum vitamin B_{12} levels for about 7 days, providing an effective supplement for up to 4 weeks. Slightly higher amounts can be given safely, but have

no added benefit. Mineralised anthelmintic drenches mostly provide insufficient cobalt to be worthwhile.

Foliar spraying with cobalt salts

Liquid application of cobalt sulphate at a rate of 175 to 350 g per hectare raises pasture cobalt levels for about 6 weeks (about three times greater elevation in pasture cobalt, depending on soil type, than following solid top-dressing with the same quantities). Selected paddocks can be sprayed before lambs and adult sheep graze. Silage paddocks can also be sprayed at the early growth stage to raise the cobalt content of the stored feed.

Soluble vitamin B_{12} injections

On many farms, vitamin B_{12} injections are considered to be the most practical and cost effective method of short-term cobalt supplementation for fattening lambs. An injection of 1 to 2 mg of soluble vitamin B_{12} raises lambs' serum and liver vitamin B_{12} concentrations for 1 to 4 weeks, depending on their liver vitamin B_{12} concentration, cobalt intake and vitamin B_{12} requirements (which are related to pasture digestibility). Where repeated vitamin B_{12} injections are required, supplementation becomes expensive in terms of labour and the cost of the drug.

Long-term supplements

Intraruminal cobalt bullets

On most farms, cobalt bullets raise the cobalt levels of sheep for over one year, although there are reports of poorer efficacy in some circumstances. The main role of the cobalt bullets is at weaning for the supplementation of long-keep replacement ewe lambs. Intraruminal bullets must be administered with care to avoid injury to the back of the pharynx (Fig. 2.71).

Pasture top-dressing with cobalt salts

On most UK farms, pasture top-dressing is not cost effective due to the price of cobalt supplements. The practice is most useful where stocking rates are high. Overseas data suggest that where pasture top-dressing at a rate of 350 g cobalt sulphate

Fig. 2.71 Extensive pharyngeal trauma, leading to death following rough, inexperienced administration of a cobalt bullet.

per hectare has been applied annually for 7 to 10 years, adequate pasture cobalt levels are maintained for a period of 3 to 9 months after top-dressing, depending on soil type. On these farms, application rates have been effectively halved to 175 g per hectare. Where cobalt sulphate has not been applied annually for several years, pasture concentrations may only be raised for a few weeks after top-dressing. Cobalt sulphate is best applied in late spring, which enables the lambs to graze the recently top-dressed pasture when they are most at risk, and to carry the reserves over the summer/autumn period. On some farms, factors such as high soil pH may limit the efficacy of pasture top-dressing.

Combined trace element ruminal boluses

Soluble glass boluses containing cobalt along with copper and selenium can effectively provide cobalt supplementation for up to 8 months. However, on farms where the copper and selenium status of animals is adequate, multiple trace element boluses may not be a cost-effective method of cobalt supplementation.

Dietary mineral supplements

While the indiscriminate use of expensive ad-lib free-access minerals may provide some insurance against production losses from trace element deficiencies, it is wasteful and not necessarily the most cost-effective option. The accurate inclusion of mineral mixes to concentrate rations can provide effective cobalt supplementation to intensively fed sheep.

Selenium deficiency

Large areas of the UK, with soils derived from granite and volcanic rocks, are considered to be selenium deficient and various selenium-responsive conditions have been described. Clinically the most important role of selenium is in the protection of cell membranes from damage by reactive oxygen metabolites in a complementary role to vitamin E. Failure of this protection leads to membrane damage and then tissue necrosis. The greatest concentrations of reactive oxygen metabolites are found in skeletal, cardiac and respiratory muscles and white blood cells, hence the role of selenium in the prevention of nutritional muscular dystrophy and immune system disorders. A number of other biological roles of selenoproteins, such as thyroid hormone production, have also been identified.

The occurrence of clinical disease caused by selenium deficiency is related to:

- Dietary selenium status.
- Supply of other antioxidants, in particular, vitamin E.
- Amounts of dietary oxidants. For example spring grass has a high polyunsaturated fatty acid content, which compounds the effects of dietary antioxidant deficiencies. Root crops are particularly low in both selenium and vitamin E, but contain large amounts of sulfoxides, which cause oxidative stress.
- Generation of oxidants, which is increased with exercise, infection or toxaemia.

Clinical signs

The important selenium-responsive conditions in sheep are ill thrift and poor reproductive performance. Historically, nutritional muscular dystrophy (white muscle

disease) was an important common selenium-responsive condition, but this disease is now uncommon, due to awareness of selenium deficiency and widespread supplementation of ewe concentrate rations.

Ill thrift

Ill thrift is the most economically important selenium-responsive condition in growing lambs up to the age of 15 months (Fig. 2.72). Growth responses of 5 to 10 percent can be achieved by supplementation on typical selenium-deficient pastures. Selenium-responsive ill thrift and poor wool production also occur in adult sheep. Selenium-responsive ill thrift is usually seen during the autumn.

Poor reproductive performance

In ewes, selenium-responsive poor reproductive performance results from embryonic mortality 3 to 4 weeks after conception. The problem also occurs in cattle but, on the same deficient pasture, sheep are the more susceptible. Selenium is also necessary for spermatozoa viability and motility, so on deficient farms rams should be supplemented several weeks before the start of breeding season.

Nutritional muscular dystrophy

Nutritional muscular dystrophy (white muscle disease) in lambs is seen as either a congenital or a delayed-onset disease. Congenital disease results in stillbirths, or the birth of weak lambs which fail to suckle and usually die from starvation within a few days. The delayed-onset disease is also seen in lambs between 1 and 3 months old. Clinical signs appear suddenly and are usually precipitated by exercise, such as gathering, or stress, such as docking, bad weather or transport. Clinical signs result from severe non-inflammatory muscle degeneration and necrosis, and vary according to which muscles are affected. Involvement of skeletal muscles results in stiffness and discomfort, which may be confused with joint ill, and a reluctance to stand or move (Fig. 2.73). Involvement of respiratory muscles results in respiratory distress, which

Fig. 2.72 An ill thrift problem was first identified in these 18-week-old lambs when they failed to reach target weights. Prompt investigation led to a diagnosis of selenium deficiency, enabling timely implementation of a supplementation regime and a return to satisfactory growth rates.

Fig. 2.73 White muscle disease is now rare, and must be differentiated from joint ill, in particular due to *Streptococcus dysgalactiae* and involving the cervical vertebrae.

is often confused with pneumonia. Involvement of heart muscle sometimes results in sudden death. Surviving animals are ill thrifty.

Impaired immune function

Antioxidant deficiency has been shown to lead to increased susceptibility to infection and has been implicated as a cause of perinatal lamb mortality.

Diagnosis of selenium deficiency

Selenium-responsive ill thrift is usually diagnosed through a combination of the exclusion of other important causes, the farm history, previous experience and knowledge of other flocks in the district, and laboratory tests. Controlled supplementation trials are generally unnecessary, because of the reliability of animal sample results.

Soil and pasture

Unlike the other important trace elements, there is a good relationship between soil, plant and animal for selenium. Areas with low soil selenium concentrations have been mapped, and a high incidence of selenium-responsive ill thrift in lambs and calves corresponds with areas of low soil selenium. However, over-vigorous extrapolation of soil map data to individual farms should be avoided.

Pasture selenium concentrations are lowest in spring, associated with rapid grass growth, but the seasonal fluctuation is relatively minor. However, this slight seasonal variation is compounded by increased metabolic demands of low dry matter intake and of pregnancy and peak lactation. Fertiliser application can lower the pasture selenium concentration, mainly through a dilution effect of increased growth.

Animal samples

Despite the good relationship between soil, plant and animal, animal samples give the best indication of selenium that is absorbed. Absorbed selenium is transported to the liver as selenoprotein P, where it is metabolised and either bound to globulin in the blood or incorporated into selenoproteins such as GSHPx.

Serum and liver selenium

Selenium analyses are expensive so rarely performed. Selenium is not stored in the body, so the selenium concentration in liver, serum or plasma is dependent on selenium in the diet. Changes in selenium intake are rapidly reflected in liver, plasma or serum selenium concentrations. Interpretation of selenium levels is also imprecise, depending on other factors such as dietary vitamin E levels and the existence of other diseases.

Blood glutathione peroxidase

Red blood cell concentrations of the seleno-enzyme GSHPx are reasonably correlated to selenium concentrations in blood, so are a reliable index of selenium status and the diagnosis of deficiency. The erythrocyte GSHPx depends on the selenium availability during erythropoeisis. Red blood cells can remain in circulation for 2 to 4 months, therefore blood GSHPx concentrations provide information about the animals' selenium status during this period, but do not reflect dietary changes.

All of these samples provide useful diagnostic information. Liver samples collected from finished lambs or culled ewes at slaughterhouses can be used to determine the need to supplement, although in practice the collection of material is difficult to organise. There is little between-animal variation in blood, serum or liver selenium, and blood GSHPx concentrations, therefore, as few as four samples may provide useful information.

Reference ranges

In New Zealand, where the vitamin E status of livestock is generally adequate due to pasture nutrition, laboratory 'normal' values are based on numerous published and unpublished controlled supplementation and growth response studies. In the UK, it can be difficult to separate the antioxidant effects of selenium and vitamin E, and some conditions attributed to selenium deficiency may be due to vitamin E deficiency and vice versa. It is therefore difficult to provide firm guidelines about low, adequate and marginal reference ranges for UK livestock (Table 2.13).

The interpretation of results requires careful consideration of the class of livestock, time of year, degree and period of deficiency, and depends on the question to be answered. Marginal values indicate that the animals are at risk of disease, but only afford a guide to a response to supplementation. Sometimes a decision is made to supplement animals, even though the likelihood of a response is uncertain.

The same reference range appears to be applicable to lambs as to adult sheep.

Nutritional muscular dystrophy

The diagnosis of nutritional muscular dystrophy is usually based on clinical and postmortem findings. The clinical diagnosis must be differentiated from joint ill, spinal abscessation and pneumonia. Serum concentrations of the enzyme, creatinine kinase (CK), which is released soon after the onset of muscle damage, can be used to support the diagnosis of white muscle disease. However, CK levels decline rapidly after a few days. Levels over 5000 iu/ml (normal range <200 iu/ml) indicate severe muscle damage, but smaller rises to 500 to 1500 iu/ml are difficult to interpret and cannot be differentiated from levels due to catching and handling.

On postmortem examination, white necrotic lesions streaked with calcified deposits can sometimes be seen in the myocardium and skeletal muscles, typically in the pelvic limbs, shoulders and diaphragm. Histology is often required to confirm the diagnosis.

Table 2.13 UK reference ranges for selenium and GSHPx.

	Abnormal/Low	Marginal	Normal/Optimal
Whole blood GSHPx[1] (units/ml cells)	<20	20 to 40	>40
Blood selenium (μmol/l)	<0.5	0.5 to 0.9	>0.9

[1] GSHPx concentrations are determined by the measurement of an enzyme rate reaction, and the values depend on the analytical method used. Reference ranges for GSHPx may, therefore, differ between laboratories.

Selenium supplementation

The choice of supplementation method is determined by the severity of the deficiency, likely duration of the deficiency, production class of sheep, production system and stocking density involved and cost.

Short-term supplements

Oral and injectable administration of selenium salts

When used strategically, oral or injectable selenium salts provide a useful method of short-term supplementation, although such regimens require monitoring and modification accordingly. Oral dosing with sodium selenate (for example using 28 g of hydrated sodium selenate dissolved in 5 litres of water, then dosing lambs at 1 ml per 10 kg^{1}), or subcutaneous injection of sodium or potassium selenate or selenite (costing about three times more than oral dosing) can provide adequate supplementation for 1 to 3 months. Sodium selenate is commonly incorporated with anthelmintic drenches. Both salts are potentially toxic so overdose should be avoided.

Long-term supplements

Controlled-release injections

Controlled-release subcutaneous injections of barium selenate paste provide adequate selenium supplementation for 9 to 12 months, and the risk of toxicity is low. Where stocking rates are low, they provide a cost-efficient method of supplementation. Selenium readily crosses the placenta and is secreted in colostrum and milk, therefore supplementing pregnant animals with selenium improves the selenium status of the foetus and sucking lamb.

Intraruminal selenium bullets

Intraruminal elemental selenium pellets provide adequate supplementation for 9 to 12 months and have a similar role to the controlled-release injections. Selenium is included with copper, cobalt and iodine in some trace element boluses, although these are generally expensive and supplementation with all of the elements included is unnecessary.

Pasture top-dressing with sodium selenate prills

The use of top-dressing to supplement animals' selenium has proven highly successful in New Zealand, and is the preferred supplementation method on about 30 percent of selenium-deficient properties. Pasture top-dressing with sodium selenate prills is an efficient supplementation method where stocking rates are high. Top-dressing of pasture with 10 g selenium per hectare is effective for approximately 12 months, and a slow-release prill containing barium and sodium selenate provides pasture with adequate selenium for 20 to 24 months. Pasture top-dressing is not currently considered to be cost-effective in the UK, where stocking rates on typical selenium-deficient farms are lower.

Inclusion of selenium salts in compound feeds

Selenium and vitamin E are added to most ewe concentrate rations.

Fig. 2.74 Relatively low vitamin E concentrations in root crops may compound the risk of selenium-responsive ill thrift in store lambs, when combined with increased antioxidant requirements associated with cold weather, high levels of dietary oxidants and low soil selenium levels.

Free-access minerals

These are provided as blocks or licks, usually containing salt to improve palatability and encourage excessive intake. Intakes are variable and they are generally prove to be an expensive supplementation option.

Vitamin E deficiency

Vitamin E has a complementary role to selenium as an antioxidant. Vitamin E (tocopherols) is synthesised by plants, levels being high in green pastures, but relatively low in root crops (Fig. 2.74). Pasture vitamin E levels decline during dry periods or with long-term storage of forages. Vitamin E is also destroyed by various treatment methods such as propionic acid application to grain or forages. Thus, vitamin E deficiency states tend to occur in housed livestock, especially animals fed poor-quality forages and home-mixed concentrate rations.

Plasma α-tocopherol levels below 1 μmol/L indicate a significant risk of nutritional muscular dystrophy, even if selenium status is adequate, while levels below 2 μmol/L may give rise to signs of disease if selenium levels are marginal.

Vitamin E can be supplemented by injection in combination with selenium salts, or orally using tablets or vitamin/mineral supplements. Treatment often needs to be repeated at weekly intervals, although this incurs the risk of selenium toxicity when injectable combinations are used.

Vitamin E is concentrated in colostrum, so supplementation of the dam's diet with vitamin E in late pregnancy will help to ensure adequate supply to the newborn lamb.

Copper deficiency

While copper deficiency is commonly seen throughout the UK, it is rarely identified as the primary cause of ill thrift in growing lambs. Lambs which are ill thrifty due to poor nutrition or parasitic gastroenteritis are often shown to be copper deficient, associated with poor dietary availability or impairment of mineral absorption respectively. In these cases, the copper deficiency is usually corrected when the primary problem is resolved. Copper and cobalt deficiencies frequently occur concurrently, with the cobalt deficiency being the primary cause of ill thrift.

The main disease associated with copper deficiency in UK sheep is swayback.

Liver biopsy

The limitations of serum copper and vitamin B_{12} concentrations for the diagnosis of copper and cobalt deficiencies have been discussed. A number of alternative tests have been described, such as the determination of the ratio between serum ferroxidase and copper concentrations, or measurement of red blood cell or serum superoxide dismutase (SOD) as indices of copper deficiency. Serum concentrations of methylmalonic acid (MMA) and urine concentrations of formiminoglutamic acid (FIGLU) have been proposed as indices of cobalt deficiency. Unfortunately reference ranges have not been determined for these alternative indices. Furthermore, because they are not routinely performed, they tend to be expensive.

Liver samples can be obtained from on-farm postmortem examination of dead or sacrificed animals, from slaughterhouse material, or from live-animal biopsy. However, animals presented for on-farm postmortem examination may not be representative of the whole flock and, while slaughterhouse material provides valuable diagnostic information, collection of samples is usually impracticable in the UK. In addition, severely copper-, cobalt- or selenium-depleted animals may not reach slaughter weight.

Liver biopsy has been demonstrated to be a valuable technique for collection of samples to assess the copper and cobalt status of live sheep, cattle and deer. Liver biopsy is also useful where serial samples are required to monitor the efficacy of supplementation regimens, or to assess the risk of copper toxicity. The technique of liver biopsy has proven to be quick, safe and reliable, and therefore applicable to field conditions. Serial biopsy samples have been collected from large numbers of sheep and cattle on several occasions, using an intercostal approach, with no adverse effects on growth or milk production.

The sheep are restrained on a raised board, in left lateral recumbency with their dorsum nearest to the operator. A 200 cm^2 area is clipped and surgically prepared, centred over the 11th intercostal space (second-to-last intercostal space), at approximately one-quarter of the distance between the thoracic vertebrae and the sternum. Lignocaine 2 percent (3 to 4 ml) is injected subcutaneously and into the intercostal space at the biopsy site. A 4 mm x 23 cm stainless steel trochar and cannula is introduced through a stab incision in the skin and advanced, perpendicular to the body wall, through subcutaneous connective tissue, intercostal muscles, pleural cavity, diaphragm and into the liver (Fig. 2.75). The position of the trochar and cannula is determined by the depth and the resistance to passage. Once through the diaphragm, at a depth of 5 to 7 cm, the trochar is removed and the cannula twisted as it is further advanced through the rougher-textured liver by approximately 2 cm. A 5 ml syringe is attached to the cannula and the plunger withdrawn by 1 to 2 ml to provide sufficient negative

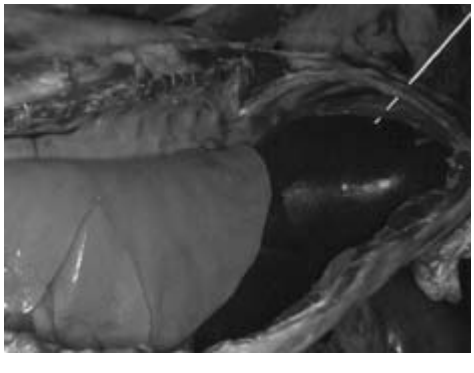


Fig. 2.75 The position of the canula relative to the thoracic wall, intercostal muscles, pleural cavity, diaphragm and liver is shown on a cadaver.

Fig. 2.76 Liver biopsy being performed in a New Zealand wool shed to monitor the adequacy of cobalt and selenium. Unlike the situation in New Zealand, where liver biopsy is routinely performed, most UK diagnostic laboratories are currently unable to assay the small samples that are obtained.

pressure to retain a core of liver in the cannula (Fig. 2.76), which is then quickly withdrawn. It is not necessary to suture the skin incision, although in most cases a temporary Michel clip is inserted. The trochar and cannula are rinsed with deionized water and immersed in aqueous hibitane solution, before use on the next animal. Useful samples of 20 to 80 mg (mean 30 mg) of liver tissue are obtained.

Ill thrift associated with chronic disease

Non-specific chronic suppurative infections of growing lambs, for example following tick-bite pyaemia, dosing gun injury, or subacute fluke, cause poor food intake, pain and pyrexia, resulting in ill thrift (Fig. 2.77). Affected animals usually have elevated globulin concentrations above 50 g/L, but routine haematology is normal.

Lameness causes ill thrift as a consequence of reduced feed intake, pain and chronic inflammation, while skin diseases such as sheep scab, blowfly strike and orf lead to poor growth rates due to reduced feed intake, exudative albumin loss (sheep scab) and increased energy requirements of infection. Ill thrift also occurs with specific diseases, such as border disease, nephrosis and coccidiosis.

Respiratory disease

Respiratory disease is important in some flocks, causing ewe and lamb deaths, poor lamb growth rates and downgrading of carcases at slaughter. In some flocks respiratory

Fig. 2.77 Chronic abscessation resulting from a dog bite.

disease causes an annual lamb mortality rate of 1 to 2 percent. In other flocks, few deaths occur, but growth rates are reduced and lambs cough when disturbed.

Respiratory viruses such as parainfluenza type 3 (PI_3) virus, respiratory syncytial virus (RSV) and adenoviruses, *Mycoplasma* spp., and other bacteria such as *Mannheimia haemolytica* and *Pasteurella multocida* individually can cause acute pneumonia in sheep. However, in practice, respiratory disease results when virus or *Mycoplasma* spp. infection of stressed animals enables the establishment of bacteria, in particular *M. haemolytica*.

Acute viral pneumonia

RSV, reoviruses and herpesviruses have been isolated from the lungs of sheep with acute respiratory disease, but their significance is questionable. Mild respiratory disease outbreaks have been associated with PI_3 and adenovirus infections, characterised by a dry, non-productive cough affecting most animals within a group of weaned lambs. Evidence of patchy dull red areas of apical lung lobe consolidation is seen on postmortem examination of recently affected animals.

One serotype of ovine PI_3, which is antigenically related to, but distinct from, bovine and human strains of PI_3 virus, is present in about 80 percent of sheep populations worldwide. In infected flocks, lambs are protected by colostral antibodies until about 6 weeks of age, following which most become infected, probably through contact with persistently infected adult sheep.

Respiratory virus infections suppress the lungs' physical defence mechanisms against further infection and provide an ideal environment of dead cells and inflammatory exudate within the lungs to enable the establishment of pathogenic bacteria, in particular *M. haemolytica*.

Mycoplasma *spp. pneumonia*

Mycoplasma spp. can be found in the upper respiratory tract of many but not all healthy sheep, and can cause mild respiratory disease, sometimes referred to as atypical, non-progressive, or proliferative pneumonia. The disease is usually seen in heavily stocked, housed, fattening or store lambs up to 1 year old, usually following the mixing of groups of lambs bought in from different sources, with rapid spread of the organism and establishment of infection in naïve animals (Fig. 2.78). The disease

Fig. 2.78 The prevalence and severity of atypical pneumonia is highest in intensively housed lambs. In this case the problem was exacerbated by concurrent cobalt and selenium deficiencies.

is characterised by a chronic persistent dry cough and slight mucopurulent nasal discharge, involving most of the group. Ill thrift has been reported, but not conclusively linked with *Mycoplasma* spp. infection. *Mycoplasma* spp. infection may persist in the lungs for several months.

On postmortem examination, clearly demarcated areas of red-brown or grey consolidation are seen in the margins of the apical and cardiac lobes (Plate 2.7). An overlying pleurisy is sometimes seen associated with secondary bacterial infection. Histopathology reveals a characteristic lymphocytic cuffing pneumonia.

Reasonable responses are reported following whole-flock oxytetracycline or tilmicosin antibiotic therapy. More importantly, prevention and control depend on improving ventilation of housing, reducing stocking densities, avoidance of mixing of sheep from different sources and different age groups, and quarantine isolation of purchased groups of animals.

Pneumonic pasteurellosis (M. haemolytica)

M. haemolytica is normally present in the nasopharynx of healthy sheep. *M. haemolytica* can cause severe septicaemic disease in young lambs, probably becoming established following the failure of passive, colostral immunity to protect against respiratory virus infections. In diseased or otherwise severely stressed older animals, pneumonic pasteurellosis results from secondary infection following viral and/or *Mycoplasma* spp. pneumonias. The prevalence of pneumonic pasteurellosis is reported to be highest during the summer and autumn.

There are several serotypes of *M. haemolytica* (A1, A2, A5, A6, A7, A8, A9, A12, A13, A14, A16 and A17) all of which can cause severe acute disease. About 50 percent of pneumonic pasteurellosis outbreaks in the UK are associated with serotype A2. All of these serotypes produce a leucotoxin which kills pulmonary macrophages involved in the lung defence mechanism to bacterial infection. Other components of the *M. haemolytica* bacterial cell wall enable epithelial attachment of the bacteria and colonisation of the lower respiratory tract. Bacterial cell death releases endotoxins, responsible for severe lung damage.

Sheep with chronic respiratory diseases such as jaagsiekte or maedi frequently become secondarily infected with *M. haemolytica*.

Chronic atypical pneumonia

Combined infection with respiratory viruses and/or *Mycoplasma* spp. and *M. haemolytica* is referred to as chronic atypical pneumonia. These pathogens are ubiquitous, thus the appearance of chronic atypical pneumonia is essentially determined by predisposing management factors. The proportion of the flock affected and the severity of the disease can vary, depending on stock management practices.

The principal stressors associated with outbreaks of chronic atypical pneumonia in the UK are difficult to quantify, but include persistent rain, changes to warm or cold weather, weaning, and animal handling for anthelmintic treatment or plunge and shower dipping. Overseas, the most important predisposing management factors are hurried mustering and yarding in dry and dusty conditions. It is presumed that panting and mouth-breathing associated with these conditions aids the establishment and proliferation of pathogens. Stress associated with poor nutrition or concurrent diseases such as acute liver fluke, gastrointestinal parasitism and trace element deficiencies

probably compromise the animals' immunity and enable the establishment of respiratory pathogens.

Outbreaks of chronic atypical pneumonia are sporadic and more severe and common in some years than in others. The most severe outbreaks are generally seen in lambs in hill and upland flocks after weaning and in housed sheep during the winter. Severe outbreaks are characterised by sudden-onset anorexia, dullness, high rectal temperatures (>40°C) and rapid laboured breathing. Affected animals frequently mouth-breathe, have a serous nasal discharge (Fig. 2.79) and drool frothy fluid from the mouth. The oral and ocular mucous membranes sometimes appear cyanotic (Plate 2.8). In some cases, animals are found dead. In less severe cases clinical signs may not be noted, but a high proportion of the flock is ill thrifty.

The clinical diagnosis of chronic atypical pneumonia in individual sheep can be problematic. The main clinical signs are coughing and a nasal discharge, while elevated rectal temperature and respiratory rate are inconsistent and affected by stress of handling. The diagnostic value of thoracic auscultation is limited by the confounding effect of air trapped in the fleece. Sheep which are moribund for reasons other than pneumonia often show a scant nasal discharge of ruminal contents, which is commonly mistaken for a sign of pneumonia.

At postmortem examination, subcutaneous ecchymotic and petechial haemorrhages are seen throughout the body. There are pleural and pericardial exudates. Large amounts of pink froth are usually present in the airways. Depending on the severity of the disease, the lung changes range from swelling and cyanosis with purple-coloured solid areas to consolidation, hepatisation (Plate 2.9) and pleurisy involving the cranial and ventral lobes. A thick grey-green exudate is often present over the affected lung tissue and pericardium (Plate 2.10). Abscesses may develop within the lung tissue (Fig. 2.80) and the bronchial lymph nodes are enlarged.

The diagnosis of chronic atypical pneumonia can be supported by histological examination of lung tissue from the periphery of affected areas and the isolation of *M. haemolytica* on bacterial culture, although this is generally unnecessary.

Treating the whole flock with a long-acting antibiotic can be useful in the face of a chronic atypical pneumonia outbreak. *M. haemolytica* is sensitive to oxytetracycline and tilmicosin, but not necessarily to penicillin.

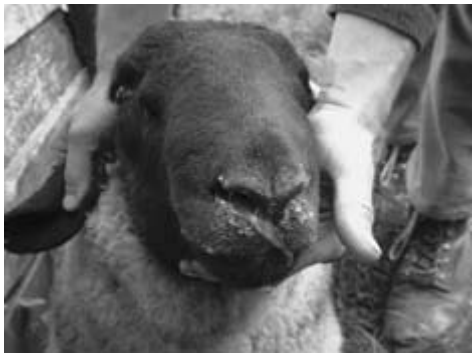


Fig. 2.79 Serous nasal discharge in a Suffolk lamb, also showing signs of pyrexia, rapid breathing and cyanosis of the ocular mucous membranes.

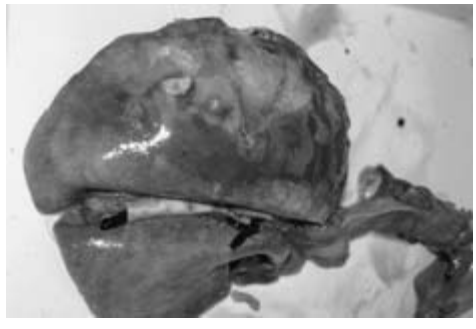


Fig. 2.80 Lung consolidation, pleurisy and pulmonary abscessation.

Management of chronic atypical pneumonia depends on avoiding the important predisposing causes. Obviously, nothing can be done to alter the weather, but careful gathering of lambs with the minimum use of dogs may reduce the severity of outbreaks. Buildings used for winter housing should be well ventilated, well drained to ensure dry bedding, and not stocked too densely. Ideally, introduced animals should be housed separately to avoid introduction and spread of new viruses or bacterial biotypes.

Intranasal vaccination of lambs at housing with a cattle PI_3 virus vaccine was widely practised during the 1980s. Anecdotal evidence suggested that the practice reduced the severity of chronic atypical pneumonia outbreaks, but this was not proven.

Early *Pasteurella haemolytica* (*M. haemolytica* and *Pasteurella trehalosi*) vaccines were not useful, particularly against *P. haemolytica* biotype A2. Modern vaccines based on iron-regulated protein antigens are more effective and offer good cross-protection against different *M. haemolytica* serotypes. Introduced animals are usually vaccinated twice, 3 to 4 weeks apart, and a booster dose given to pregnant ewes 4 to 6 weeks before lambing, to ensure colostral protection of their lambs to 5 weeks old. Vaccination is usually combined with clostridial disease protection. Maternal colostral antibodies do not interfere with active immunisation, so lambs can be vaccinated from 10 days old, using two injections 3 to 4 weeks apart. Active immunity is only short lived, but may be boosted by a continued low level of sub-lethal challenge. However, vaccination courses should ideally be timed so that the second injection coincides with the highest risk period. Some apparent failures of the modern vaccines may be associated with an effect of concurrent *Mycoplasma* spp. infection on the immune response.

Pasteurella vaccines also provide some protection against septicaemic disease in young lambs caused by *M. haemolytica* and some degree of protection against systemic pasteurellosis in store lambs caused by *P. trehalosi*.

The recommended dose of vaccine should be administered under the skin over the neck. Clean needles should be used and regularly changed. Vaccines should be correctly stored in a dark place at about 5°C, but protected from freezing. Vaccines should be used before their expiry date and, as a general rule, partially used packs should be discarded at the end of the day.

Chronic suppurative pneumonia

Suppuration occurs sporadically in individual lambs as a sequel to enzootic pneumonia, when their immune system fails to clear the bacterial pathogens, leaving pockets of bacteria which develop into chronic abscesses. The abscesses may be located in the lung itself, or in the pleural space where they are walled off by fibrous pleural adhesions. Typical bacteria involved include *A. pyogenes*, *M. haemolytica*, *P. multocida* and *E. coli*.

The clinical signs are predominantly those of weight loss and ill thrift. Pyrexia and respiratory signs are usually absent, but dyspnoea and a purulent nasal discharge may be observed following rupture of an abscess into the airways. When large pleural abscesses are present, auscultation of the thorax may reveal muffling of lung or heart sounds on the affected side. Serum globulin levels are elevated due to chronic immune stimulation.

Parasitic pneumonia (lungworm)

Lungworm infection is a frequent incidental postmortem finding, although it is not generally considered to be clinically significant in sheep. However, under certain poorly defined circumstances lungworm can cause severe disease, probably associated with primary infection of previously unexposed sheep, or re-infection of previously exposed sheep following heavy challenge. The most important lungworm of sheep is *Dictyocaulus filaria*, but *Muellerius capillaris* and *Protostrongylus rufescens* are commonly identified.

Adult *D. filaria* in the trachea and bronchi lay eggs which contain fully developed L_1. L_1 hatch quickly and are coughed up, swallowed and passed in faeces. Development on pasture to infective L_3 can take as little as 5 days. L_3 migrate to the tips of the grass, are ingested and penetrate the intestinal mucosa. The development to L_3 in the faeces is fastest during warm weather and escape of L_3 to herbage is partially moisture dependent. Moulting to L_4 occurs in the mesenteric lymph nodes, followed by migration to the ventral parts of the caudal lobes of the lungs via the lymphatics and blood. L_4 enter the alveoli where they moult to adults. The pre-patent period is 21 to 28 days.

D. filaria larvae in the alveoli cause an eosinophilic exudate, blocking small airways, resulting in gradual-onset coughing. Severely affected sheep display signs of persistent harsh coughing, and dyspnoea. Rectal prolapse has been reported in some animals following particularly severe coughing paroxysms. Most acute lungworm outbreaks occur in lambs between July and September. Deaths are uncommon and tend to be due to secondary pasteurellosis.

The epidemiology of lungworm is more complex and infection less predictable than parasitic gastroenteritis. Not all aspects of larval survival and transmission are known. Some L_3 can overwinter on pasture and some parasites survive in carrier animals as adult worms, with excretion of L_1 by adult animals, particularly ewes, in the spring. Because adult female *D. filaria* produce eggs containing fully developed larvae, the time from hatching to infective larvae is short, and in optimum warm and wet conditions the numbers of infective L_3 on pasture can increase rapidly. Lambs develop a strong immunity. Thus, the severity of the disease depends on the number of L_3 ingested and the animals' immune status.

The clinical diagnosis of lungworm is problematic. A presumptive diagnosis is based on the clinical signs of a mild to moderate pneumonia with coughing in lambs grazing likely contaminated pastures. Identification of larvae in faeces can be useful, but faecal larvae are only present in patent infections (Fig. 2.81).

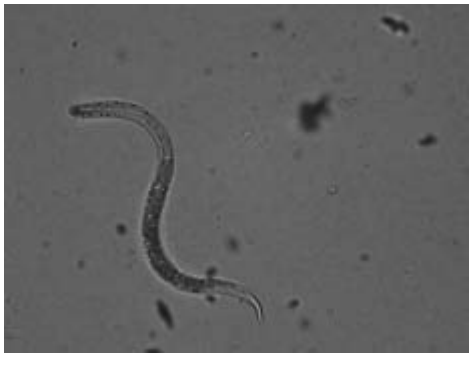


Fig. 2.81 *D. filaria* L_1 on a microscope slide following Baermannisation.

A Baermann technique for the identification of lungworm larvae

- Break up 5 to 10 g of faeces and wrap it in gauze.
- Close over the bottom of a funnel and fill it with lukewarm water.
- Immerse the faeces/gauze in the water in the funnel.
- Leave the Baermann set-up overnight.
- Larvae migrate to the surface of the faecal mass and drop to the point of the funnel.
- Withdraw a few drops from the funnel.
- Examine under low-power microscopy for the presence of larvae.
- Confirm the identity of the larvae.

Fig. 2.82 Severe bronchopneumonia secondary to lungworm infection in a Greyface ewe lamb.

Fig. 2.83 Adult *D. filaria* in the trachea and bronchi.

Postmortem findings include parasitic pneumonia, involving ventral areas of the caudal lung lobes (Fig. 2.82), severe bronchiolitis and bronchitis. Adult worms 30 to 100 mm long can be seen in the trachea (Fig. 2.83) and bronchi (Fig. 2.84).

Severely affected sheep require anthelmintic and long-acting antibiotic treatment. Anthelmintic treatment of severe cases can exacerbate clinical signs due to allergic reaction to dead parasites in the lungs. Ganglion blocking agents (levamisole or morantel), which first paralyse the worms, are arguably safer than benzimidazoles and macrocyclic lactones which kill the worms immediately.

In most farm situations the routine control of gastrointestinal parasitism will control lungworm at the same time. As yet, there is no commercially available vaccine for *D. filaria* along the lines of the *Dictyocaulus viviparus* irradiated larval vaccine for cattle.

M. capillaris and *P. rufescens* lungworms are frequently found in UK sheep as incidental postmortem findings, but are seldom associated with clinical disease. *M. capillaris* (Fig. 2.85) is found in the alveoli, associated with multifocal, 2-mm diameter, grey subpleural nodules, mostly in the caudal lung lobes (Plate 2.11). *P. rufescens* are seen in small bronchioles as slender, 1.5- to 3.5-cm long, red adult worms. On postmortem examination lesions are seen as small yellow/grey granulomas in the caudal lung lobes.

M. capillaris and *P. rufescens* both require land snails or slugs as an intermediate host to complete their life cycle and develop to the infective stage. Because of this, control of these parasites is difficult, but seldom necessary.

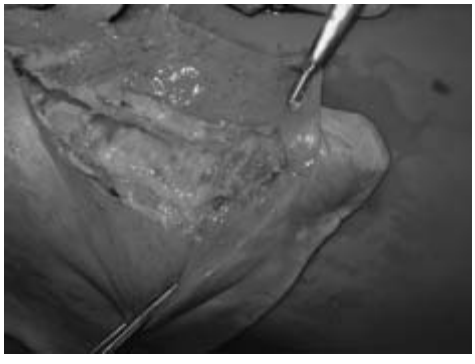


Fig. 2.84 *D. filaria* L_4 and early adults in the lower airways.

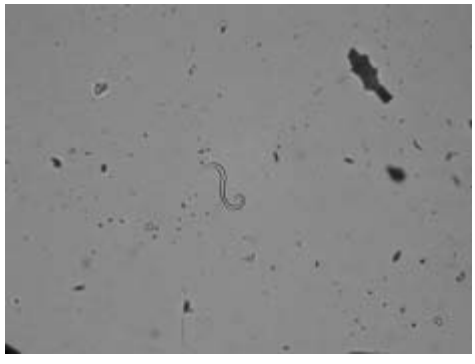


Fig. 2.85 *M. capillaris* L_1 on a microscope slide following Baermannisation.

Other causes of respiratory disease

Carbolic dip poisoning occurred occasionally following the use of 'bloom' dips to enhance the appearance of sheep prior to showing. It was characterised by severe dyspnoea 1 to 3 days after dipping, with a high mortality rate. Postmortem examination showed severe red/purple consolidation of both lungs, but histopathology of the lung was required for definitive diagnosis. Treatment was largely symptomatic, with washing of the fleece to remove as much dip as possible.

Bordetella parapertussis (similar to *Bordetella pertussis*, the cause of whooping cough in humans) has been isolated from the lungs of sheep, and experimental evidence suggests that it may play a role in enzootic pneumonia by predisposing lambs to secondary pneumonic pasteurellosis.

Note

1 This recipe was used successfully for many years to supplement trace element deficient store lambs in the north-east of Scotland. It will not be suitable in all situations and great caution must be taken in every case to avoid the risk of toxicity.

Chapter 3

Unexpected Disease or Death

Different practices are required for the prevention and management of different causes of unexpected deaths in growing lambs and adult sheep, so it is important that the cause of unusually high losses is investigated. The diagnosis is usually based on the flock management history and postmortem findings. Most of the common causes of sudden death are associated with specific postmortem signs in fresh carcases, although in many cases appropriate samples must be submitted for further laboratory tests. Sometimes losses occur as a result of more than one cause, so as many carcases as possible should be examined.

Sudden death in store lambs

Sudden deaths often occur following the movement of lambs during the late summer and autumn onto stubbles, forage crops or lush aftermath pastures (Fig. 3.1). The common causes of sudden death associated with stressful husbandry, feed changes or toxic substances in forage crops are:

- clostridial diseases
- redgut
- systemic pasteurellosis
- grain overload
- subacute liver fluke
- nitrate poisoning
- brassica poisoning.

Fig. 3.1 Sudden deaths occur commonly in growing lambs turned onto forage crops.

Sheep Flock Health: A Planned Approach. Neil Sargison
 ISBN: 978-1-4051-6044-5

This list of causes of sudden death in growing lambs is by no means comprehensive. For example, louping ill should be considered when losses occur in naïve animals introduced to tick areas.

Redgut

Redgut is a colloquial term, which is used to describe intestinal displacement and torsion in sheep. The condition is characterised by sudden death and occurs most commonly when weaned lambs have been fed lush forage for a period of 3 weeks or more. Sporadic losses of 1 to 2 percent can occur.

The most plausible hypothesis for the cause of redgut is that the feeding of a highly digestible feed promotes a rapid rate of passage of digesta through the forestomach. After a period of about 3 weeks on such diets, the volume of the ruminoreticulum is reduced. Furthermore, significant amounts of fermentable carbohydrates remain in the digesta which leaves the forestomach. Fermentation of carbohydrate in the large intestine and the production of volatile fatty acids result in intestinal distension and reduced motility. The net effect is a reduction in ruminoreticulum volume (Fig. 3.2), accompanied by an increase in large intestinal volume. It has been speculated that these unstable conditions enable displacement of the abdominal contents and, in extreme cases, cause intestinal torsion.

The postmortem examination of freshly dead lambs is required to differentiate redgut from other causes of sudden death such as clostridial diseases. In cases of redgut, the displaced intestine is seen immediately on opening the abdominal cavity. The displaced intestine is bright red and distended from the duodenum to the terminal colon. In most cases a clockwise torsion of the intestinal mass can be determined (Plate 3.1).

Where there is a commercial requirement to feed lambs on fast-growing leguminous crops, roughage supplementation in the form of ad-lib hay can reduce the incidence of redgut. Anecdotal evidence suggests that a 5-day lush pasture and 2-day rough pasture feeding cycle effectively prevents deaths from redgut.

Systemic pasteurellosis

Systemic pasteurellosis is the most commonly diagnosed cause of sudden death in 6- to 10-month-old UK lambs between October and December. Mortality rates of 20 percent have been reported, but losses of about 2 percent are more commonplace.

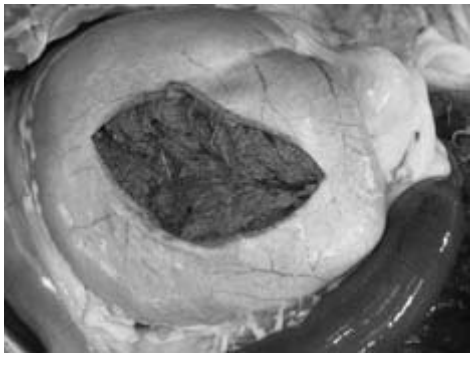


Fig. 3.2 A relatively small forestomach containing lush green forage in a case of redgut.

Fig. 3.3 Outbreaks of systemic pasteurellosis often follow the movement of lambs onto brassica crops, particularly when this coincides with wet weather.

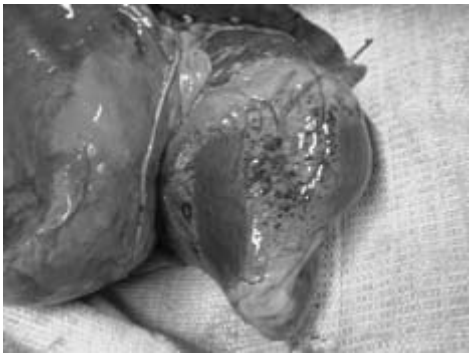


Fig. 3.4 Superficial haemorrhages on the surface of the epicardium. These signs are seen on postmortem examination of lambs which have died suddenly, irrespective of the cause.

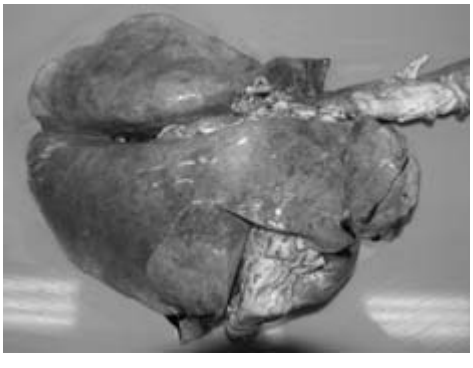


Fig. 3.5 Swelling and slight purple discolouration of the lungs. *P. trehalosi* was cultured from the lung tissue.

Fig. 3.6 Froth and blood in the trachea and bronchi. The cut surface of the lung appears swollen and haemorrhagic, and oozes froth.

Systemic pasteurellosis is caused by serotypes T3, T4, T10 and T15 of the bacterium *Pasteurella trehalosi*, but the reasons for disease outbreaks are not fully understood. Outbreaks frequently, but not always, follow movement of lambs onto rape, turnips or improved pastures (Fig. 3.3). Wet and cold weather has also been implicated. *P. trehalosi* is found in the tonsils and upper gastrointestinal tract of healthy sheep and it has been suggested that, under certain stressful conditions, the bacteria multiply and spread rapidly to the lungs and other organs. Endotoxin is released from the cell walls of dead bacteria. Concurrent diseases such as cobalt deficiency or tick-borne fever may also predispose to outbreaks of the disease.

Postmortem examination sometimes reveals subcutaneous haemorrhages over the neck and thorax, with superficial haemorrhages on the pleura (Plate 3.2), diaphragm and epicardium (Fig. 3.4). The lungs are swollen with widespread haemorrhages (Fig. 3.5), and bloodstained froth in the airways (Fig. 3.6), but no evidence of consolidation. Necrotic erosions are sometimes seen in the pharynx around the tonsils (Fig. 3.7), nasal mucosa, oesophagus (Fig. 3.8) and abomasum (Fig. 3.9). The tonsils and retropharyngeal lymph nodes are enlarged. The abdominal viscera are

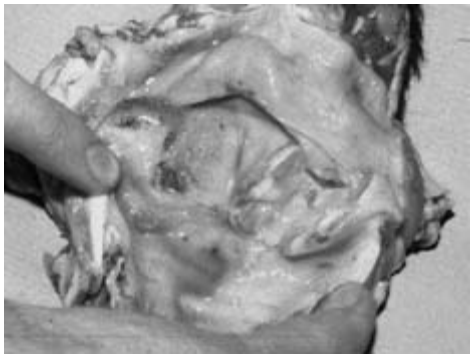


Fig. 3.7 Necrotic erosions in the pharynx and proximal oesophagus are sometimes but not consistently seen in lambs which have died as a result of systemic pasteurellosis.

Fig. 3.8 Erosions along the length of the oesophagus, covered by a layer of necrotic exudate. These signs are rarely seen in lambs which have died as a result of systemic pasteurellosis. In this case *P. trehalosi* was cultured from the lesions, but the differential diagnoses of viral diseases such as border disease and foot-and-mouth should always be considered.

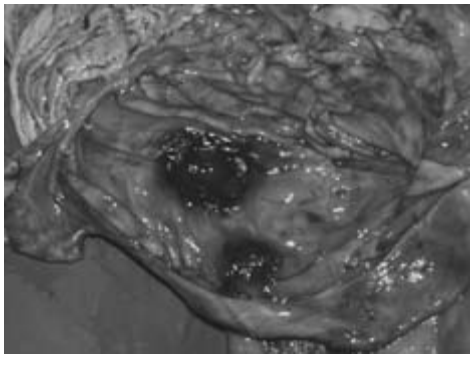


Fig. 3.9 Necrotic erosions in the abomasum.

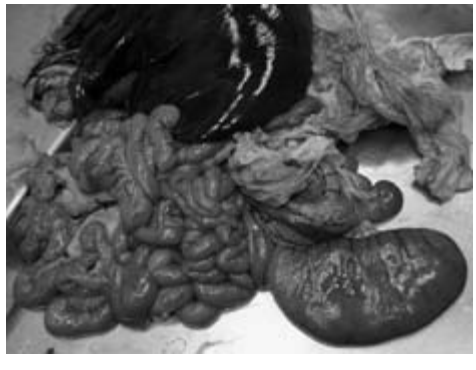


Fig. 3.10 Congestion of the liver and intestinal viscera. These signs are typical of endotoxaemia, which occurs with systemic pasteurellosis.

often congested (Fig. 3.10) and, in some cases, necrotic infarcts may also be present in the liver, spleen and kidney, visible as small grey foci.

Cases of systemic pasteurellosis are seldom seen alive. Affected lambs are usually moribund, with rapid shallow respiration and congested mucous membranes (Plate 3.3). Clinical signs of sudden-onset dullness, recumbency, respiratory distress and copious dribbling of saliva from the mouth are mostly associated with endotoxaemia, and usually progress to death within a few hours.

In the face of an outbreak of systemic pasteurellosis, whole-flock treatment with a single injection of long-acting oxytetracycline or tilmicosin may help to prevent further losses, although the stress of gathering and injecting the lambs may itself precipitate some deaths. It is, therefore, impossible to predict the effectiveness of this strategy and the decision to treat or not is based on individual flock circumstances.

Prevention of systemic pasteurellosis is also problematic. Avoidance of nutritional stress and wet weather conditions, which predispose to the disease, is difficult; based on the high incidence of the disease in pasteurella-vaccinated flocks, vaccination only affords variable short-lasting protection.

Grain overload (rumen acidosis)

Grain overload is associated with failure to adapt to sudden increases in the level of fermentable carbohydrate feeding. The problem is common in late-pregnant ewes and intensively grain-fed finishing lambs, and is sometimes seen in sheep grazing stubbles where piles of grain have been left in the field (Fig. 3.11).

Fermentation rather than digestion of grain in the ruminoreticulum results in production of large amounts of lactic acid and other toxins, which when absorbed into the circulation result in acidosis and endotoxic shock. Large quantities of lactic acid decrease the pH of the rumen and kill the normal flora of bacteria and protozoa. Water is drawn from the circulation into the rumen, resulting in dehydration.

The severity of the disease depends on the amount of grain eaten, the physical state of the grain, for example bruised grain ferments more rapidly in the rumen than whole grain, and previous adaptation to grain feeding. In the early stages of the disease, animals may show signs of mild colic and tooth grinding, progressing in severely affected animals to a staggering gait, apparent blindness and stupor (Fig. 3.12). Severely affected animals may become recumbent and die within 24 hours of grain engorgement.

Recovery in many cases is slow because the rumen becomes inflamed with secondary fungal and bacterial infections. Lameness is often seen in recovering animals due to laminitis.

Fig. 3.11 Grain overload is sometimes seen in lambs grazing on stubbles shortly after harvest, associated with grain spilt from the combine.

Fig. 3.12 Severe depression and early signs of scour in lambs recovering from grain overload. This particular disease outbreak was associated with feeding housed lambs on a home-mixed ration of beet pulp and whole wheat. The beet pulp should have provided sufficient fibre to reduce the risk of grain overload. However, the beet pulp had been badly stored and was stale. Disease resulted from selective feeding on the grain component of the ration.

Fig. 3.13 Large amounts of partially digested grain from the forestomach of a lamb which died as a result of grain overload.

The diagnosis of grain overload is based on the feeding history and clinical signs. Postmortem examination of dead lambs is often unrewarding, although the presence of large amounts of partially digested grain in the rumen (Fig. 3.13) of a dehydrated and congested carcase, and fatty infiltration of the liver (Plate 3.4) may support the diagnosis. Identification of a low ruminal fluid pH can support the diagnosis in freshly dead animals.

When grain overload is suspected, animals should be removed from the source of grain and provided with good-quality roughage. Affected animals can be treated orally with 30 ml of milk of magnesia (magnesium hydroxide) and given multivitamin injections, but the prognosis is poor in advanced cases. In potentially valuable animals, supportive intravenous fluid therapy may be justified.

Subacute liver fluke

In wet areas of western UK, subacute liver fluke is an important annual cause of sudden death of sheep of all ages during autumn and early winter. The disease is caused by massive liver and peritoneal damage due to the presence of migrating immature flukes and is characterised by lethargy, pallor, laboured breathing and death. Sudden deaths may occur when animals are handled, as a result of liver rupture. Sudden deaths from black disease or redwater may occur in unvaccinated sheep.

Poisonous plants

Many plants are potentially poisonous, but poor performance, unexpected clinical disease and death due to plant poisoning in well-fed sheep are uncommon. Cases of plant poisoning are often associated with:

- Poor pasture availability – drought conditions or heavy snow often force animals to graze hedges or toxic pasture weeds (Fig. 3.14).
- Overgrazing/poor pasture management – enable the establishment of toxic weeds.
- Forage conservation – most growing toxic plants are avoided because they are unpalatable. However, these plants may become palatable, without losing their toxicity, when they are cut or conserved with forage (Fig. 3.15).
- Accessibility of poisonous plants – many isolated poisoning cases are associated with dumping of hedge cuttings and garden waste in fields. Fallen and overhanging

Fig. 3.14 Heavy snowfall sometimes forces animals to graze toxic plants in hedges, which they would otherwise avoid.

Fig. 3.15 Some toxic pasture weeds which are normally avoided become palatable without losing their toxicity when preserved as hay or silage.

Fig. 3.16 Poisoning cases are sometimes associated with the careless dumping of toxic garden plants (in this case yew and rhododendron prunings) in fields.

branches following storms and heavy snowfall may become accessible to inquisitive animals (Fig. 3.16).

- Grazing experience – young animals, animals moved from another area, or animals reared on their own often lack the protective behaviour to avoid poisonous plants.
- Transport – tired and hungry animals may graze indiscriminately.
- Environmental factors – drought, shade, high temperatures or injudicious use of herbicides may increase the toxicity of some plants.
- Animal species – sheep have different biochemical pathways from other farm animal species that make them more or less susceptible to certain phytotoxins.

Some plants contain a single toxic substance, while others contain several toxins with widely different chemical properties. Thus, the clinical signs associated with plant poisonings are varied and often non-specific. The major toxic principles are:

- alkaloids
- glycosides
- nitrates/nitrites
- oxalates
- photosensitising agents
- proteins/peptides/amino acids
- tannins.

Alkaloids

Alkaloid-containing plants generally taste bitter and are avoided, although individual sheep can become addicted to them. Climate, soil and other environmental factors can modify the alkaloid content. Poisoning is usually fatal and, in animals that survive, recovery is often incomplete.

Some alkaloids are structurally similar to acetylcholine, dopamine and serotonin, thus induce toxic effects by mimicking or blocking the action of neurotransmitters (for example, yew and laburnum poisoning). The signs of acute alkaloid poisoning therefore include excess salivation, dilation or constriction of the pupil, vomiting, abdominal pain, diarrhoea, incoordination, convulsions and coma.

Pyrrolizidine alkaloids are transformed in the liver to reactive pyrrols, which exert their toxic effects in cells, causing necrosis, inhibition of mitosis or vascular damage (for example, ragwort poisoning). Pyrrolizidine alkaloid poisoning usually results in chronic liver disease. Some alkaloids are teratogenic (for example, hemlock and lupin poisoning).

Glycosides

Glycosides are widely distributed in plants and are mostly non-toxic. The important toxic principles are:

- cyanogenic glycosides
- goitrogenic glycosides
- cardiac glycosides
- saponins
- unknown, for example ptaquiloside which is present in bracken.

Cyanogenic plants (for example, linseed and cherry laurel) contain an enzyme system capable of converting glycosides to hydrocyanic acid (HCN). Within the plant the enzymes and glycosides are separated, but disruption of plant cells during decomposition or ruminal digestion enables the production of HCN.

In general, the highest concentrations of cyanogenic glycosides are found in the leaves. Drying of the plant material does not reduce the potential toxicity, but toxic forage loses much of its cyanide content when made into silage. Linseed loses its toxicity when boiled.

HCN inactivates the cytochrome oxidase system in the mitochondria, starving cells of oxygen. Thus tissues with the highest oxygen requirement (brain and myocardium) are first affected. As oxygen transfer to tissues is blocked, venous blood becomes oxygenated with a bright red appearance. HCN is absorbed rapidly and clinical signs sometimes follow within a few minutes, including dyspnoea, convulsions, muscle tremors and death. Animals' tolerance to HCN appears to increase with experience, although prolonged exposure of pregnant ewes to small amounts of cyanogenic glycosides, present in some modern clover and pasture grass species, can have a goitrogenic effect on their lambs.

The diagnosis of cyanide poisoning is based on the feeding history, clinical signs, and non-specific postmortem findings of bright red mucosae (Plate 3.5) and a 'smell of almonds' in the rumen. The diagnosis can be supported by analysis of the HCN content of liver or muscle tissue.

Treatment must be initiated rapidly after exposure to HCN if it is to be successful. Treatment regimes are based on the fact that cyanide radicals form complexes with a

number of chemicals within tissues. Large doses of vitamin B_{12} may enable cobalt to combine with cyanide in the circulation. A regime consisting of intravenous injection of sodium thiosulphate (500 mg/kg) and sodium nitrite (20 mg/kg) has been described, but is seldom practical. Repeat treatments are usually required.

Goitrogenic glycosides (glucosinolates) (for example, present in brassica crops and white clover) can cause goitre, reduced growth rates and/or diarrhoea, depending on their composition. Sudden-onset blindness (rape blindness) and digestive disturbances in animals grazing on rape are also thought to be associated with glucosinolate poisoning. The highest concentration of these glycosides is present in the seeds of mature plants.

Brassica crops contain variable amounts of both glucosinolate and thiocyanate goitrogens. Glucosinolates interfere with thyroid hormone synthesis, while thiocyanates impair uptake of iodine by the thyroid gland. Glucosinolate-induced goitre cannot be managed by iodine supplementation, while thiocyanate-induced goitre can.

Cardiac glycosides (for example, digitoxin and digitalin present in foxgloves) have a specific action on the myocardium, increasing contractility and slowing the heart rate (Fig. 3.17). Moderate intoxication results in bradycardia, depression, regurgitation and diarrhoea, while larger amounts of toxin cause various cardiac irregularities, including bradycardia, then tachycardia and dysrhythmia. Signs usually develop within 4 to 12 hours of ingestion of the plant may persist for 2 to 3 days. Most deaths occur within 12 to 24 hours. Plants containing cardiac glycosides do not lose their toxicity when dried or boiled.

A treatment regime consisting of supportive fluid therapy, atropine (0.5 mg/kg) and propranolol (5 mg dosed to effect) is described. Oral administration of charcoal (5 g/kg) and rumenotomy have been reported to be effective.

Saponins (for example, present in ivy) are naturally occurring glycosides with the physical properties of soaps. Most saponins found in plants are absorbed very slowly, but large quantities can cause gastroenteritis (Fig. 3.18).

Fig. 3.17 Foxgloves contain high concentrations of cardiac glycosides, which can result in death within 12 hours of ingestion.

Fig. 3.18 Individual animals very occasionally develop a taste for ivy, resulting in severe diarrhoea.

Fig. 3.19 Under certain conditions, the nitrate levels in brassica crops can be extremely high, resulting in sudden-onset signs of toxicity in lambs.

Nitrates and nitrites

Plants absorb nitrates from the soil and generally convert them rapidly into other nitrogenous compounds. Nitrates accumulate in the soil during periods of drought and are taken up by plants in large amounts when the drought ends. Overcast conditions favour plant storage of nitrates, while in bright sunlight nitrates are converted to amino acids and proteins. The use of nitrogenous fertilisers can cause high nitrate concentrations in plants. Some plants (for example, clovers and brassicas) can accumulate particularly high concentrations of nitrates (Fig. 3.19). Roots and stems usually contain more nitrate than leaves.

While ingestion of large amounts of nitrates can cause gastroenteritis, the main importance of nitrates is as a source of nitrites, which are formed in the rumen after ingestion of nitrates.

Nitrite poisoning is most common in cattle, but is sometimes seen as a cause of sudden-onset disease leading to death in store lambs, often resulting in high losses. The animal's capacity to convert nitrate to nitrite is enhanced by continued feeding of nitrate-rich plants, due to the adaptation of the ruminal microflora. Poorly fed animals are more susceptible to nitrite poisoning than those on diets with adequate carbohydrate levels, in which case much of the nitrate is converted to ammonia.

Absorbed nitrites combine with haemoglobin in the blood to form methaemoglobin, which is incapable of transporting oxygen. The clinical signs associated with nitrite poisoning include dyspnoea, characterised by gasping and rapid respiration, tachycardia, muscle tremors and weakness. In severe cases the oral, conjunctival and vulval mucosae appear dark brown-coloured (Plate 3.6), and eventually cyanotic in appearance due to the high blood methaemoglobin content. Death from anoxia can occur within a few hours of eating nitrate-rich plants, although it is more usual for a few days to elapse before signs appear. Abortion is sometimes seen as a sequel to severe nitrate poisoning.

The diagnosis of nitrate poisoning is usually based on the grazing history, clinical signs and postmortem findings. Blood samples from affected animals are chocolate-coloured and opaque. Postmortem findings include brown discolouration of blood and tissues, pulmonary congestion and petechial haemorrhages in the heart muscle and trachea. The postmortem diagnosis can be supported by identification of high nitrite or nitrate concentrations in aqueous humour or cerebrospinal fluid. The percentage of methaemoglobin in the blood can be determined, but this is usually unnecessary.

The clinical signs associated with nitrate poisoning may resemble the signs of HCN poisoning, but in cases of nitrite poisoning the blood is dark brown in colour in contrast to the bright red colour of the blood in HCN poisoning.

Animals should be removed slowly from the source of nitrates and provided with carbohydrate-rich supplementary feed. If necessary they should only be reintroduced gradually to the high-nitrate feed, by grazing for no more than one hour per day. Particular caution should be taken during overcast, warm weather.

Treatment using intravenous infusion of methylene blue (4.4 mg/kg as a 2 percent solution) can be effective, but is seldom practical. Methylene blue causes rapid reconversion of methaemoglobin to haemoglobin. The half-life of methylene blue in tissues is about 2 hours, so repeat treatments at 6-hourly intervals may be necessary. Unfortunately, methylene blue is seldom immediately available in sufficient quantities. (Methylene blue is used as a microbiological stain and is available from laboratory suppliers.)

Oxalates

Intake of large amounts of oxalate-rich plants (for example, beet, rhubarb and sorrel) can result in the ruminal absorption of free oxalate and precipitation of calcium oxalate crystals in submucosal arteries. Free circulating oxalate damages arterioles and capillaries in the lungs and brain, causing pulmonary and cerebral oedema, while continuous ingestion of small amounts of soluble oxalates causes nephrosis due to precipitation of calcium oxalate crystals in the lumen of the renal tubules. Affected sheep sometimes become hypocalcaemic.

Sheep are more susceptible to oxalate toxicity than other ruminant species, lambs being more susceptible than adults because most ingested oxalates are metabolised in the ruminoreticulum to innocuous carbonates and formates.

The clinical signs associated with oxalate poisoning are usually vague. The diagnosis is based on the history of access to oxalate-rich plants, or postmortem histological findings of calcium oxalate crystals in the renal tubules and rumen wall.

In the short term animals may respond to calcium borogluconate therapy, but most relapse or die from acute renal failure.

Proteins, peptides and amino acids

The toxic mushroom, *Amanita phalloides*, contains aminotoxins. Toxicity symptoms include severe abdominal pain, diarrhoea and eventually coma. Mistletoe contains polypeptide viscotoxins which have irritant and necrotising effects.

Blue-green algae (*Microcystis* spp.) that form blooms on still water contain peptides that are potent hepatotoxins. The toxins are chemically stable and not inactivated by the usual treatments used for drinking water. Blue-green algae are widespread in inland waters in UK and poisoning has been described in dogs and cattle, but not in sheep.

The amino acid S-methylcysteine sulfoxide (SMCO) is converted in the rumen during normal fermentation to dimethyl disulfide. This metabolite causes poor growth rates and haemolytic anaemia. Various amounts of SMCO are found in different brassica crops.

Fig. 3.20 Oak twigs incorporated throughout a silage bale. Tannin poisoning occurred in a group of ewes fed on baled silage, which had been made shortly after a storm had brought down oak twigs and unripe acorns.

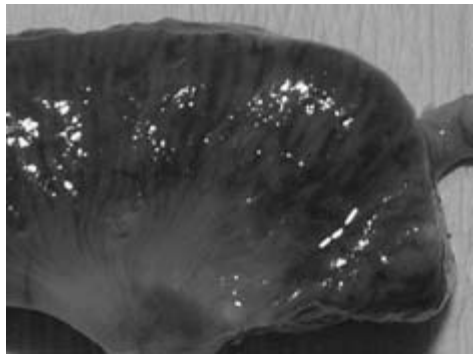


Fig. 3.21 Postmortem appearance of toxic nephropathy in a section kidney from a ewe which died as a result of acorn poisoning.

Tannins

Tannins bind to protein and produce an astringent reaction in the mouth. Oak leaves and acorns contain hydrolysable tannins, which are broken down to toxic metabolites in the digestive tract, before causing necrosis, primarily of the proximal renal tubules. Individual sheep may develop cravings for oak leaves and acorns, and unripe acorns brought down after storms are particularly toxic (Fig. 3.20). Some individual sheep appear to be able to tolerate large amounts of acorns, while others cannot.

Signs of tannin poisoning may not appear for some days after ingestion. Initially there is dullness, anorexia, constipation and cessation of urination. Later there is persistent diarrhoea and dysentery, with dark urine and serous ocular, nasal and oral discharges. Death sometimes occurs before diarrhoea becomes apparent.

Postmortem findings include a distinctively uraemic-smelling carcase, acorns or oak leaves in the forestomach, haemorrhagic abomasitis, subcutaneous haemorrhages and oedema, especially of the abomasum and perineum. The kidneys are swollen and nephrotic (Fig. 3.21).

Treatment consists of the administration of liquid paraffin with milk, mucilage and appetite stimulants. Saline purgatives are contraindicated because of kidney damage. The prognosis is aided by measurement of blood urea concentrations, those sheep with levels exceeding 50 mmol/L (normal 2 to 6 mmol/L) usually dying, while in surviving animals, levels usually remain elevated between 10 and 20 mmol/L for several weeks.

Bracken poisoning

Bracken (*Pteridium* spp.) covers over 1600 km^2 of mostly rough and hill grazings in the UK. In many parts of western Scotland (Fig. 3.22) and northern England, the area covered by bracken is increasing by about 2 percent each year. The whole plant including the rhizome is poisonous. Younger plants are the most toxic and drying does not remove the toxicity. Poisoning usually occurs when animals are forced to eat a diet of bracken, often in dry years, in late summer and autumn. Large amounts

Fig. 3.22 Sheep generally only eat bracken when pasture growth is limited, for example during drought conditions.

Fig. 3.23 Swelling over the lower jaw of a Scottish Blackface ewe, associated with a mandibular tumour.

of bracken need to be ingested over a period of several weeks before poisoning is seen.

The major toxic principles present in bracken are:

- A cyanogenic glycoside – usually present in harmless quantities.
- Thiaminase – only responsible for poisoning in monogastric animals, which don't synthesise their own thiamine.
- Ptaquiloside – a carcinogenic glycoside, present in varying amounts, depending on season and location of the bracken.
- An aplastic anaemia factor – an unknown toxic factor responsible for bone marrow depression.
- Prunasin – a cyanogenic glycoside present in most bracken varieties, which appears to act as a deterrent to grazing.

Sheep are more selective in their grazing habits than cattle, and generally do not eat bracken rhizomes. Consequently, bracken poisoning is less common in sheep than in cattle, and some well-documented clinical manifestations of the disease in cattle, such as laryngeal disease in young calves, peracute haemorrhagic disease and haemangiomas in the bladder wall, have not been reported in sheep. Prolonged ingestion of bracken by sheep can result in ill thrift, depression, exercise intolerance, anorexia and anaemia. Sometimes the development of clinical signs may be delayed for up to 8 weeks after access to bracken has been stopped.

In some areas of northern England, prolonged exposure of sheep to bracken is associated with progressive retinal degeneration, referred to as 'bright blindness'. The disease is seen in 3- to 4-year-old animals during autumn and is characterised by separation from the flock, high head carriage and a high-stepping gait. The eyes shine abnormally brightly in semi-darkness, giving the disease its name.

A high prevalence of neoplasms, especially tumours of the jaw (Figs 3.23 and 3.24), alimentary squamous cell carcinomas (Fig. 3.25) and papillomas, has been recorded in sheep grazing bracken. It is thought that bracken toxicity somehow interferes with the animals' normal immune response to tumour-inducing viruses.

The diagnosis of bracken poisoning is based on the grazing history, clinical signs and postmortem findings, including multiple internal haemorrhages, ulceration of

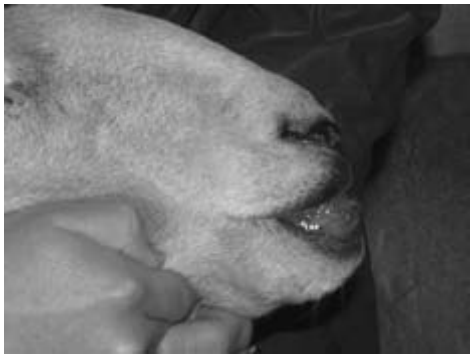


Fig. 3.24 The prevalance of jaw tumours is particularly high in sheep grazing on bracken, although tumours such as this involving the front of the lower jaw of a Scottish Halfbred ewe also occur in sheep with no access to bracken.

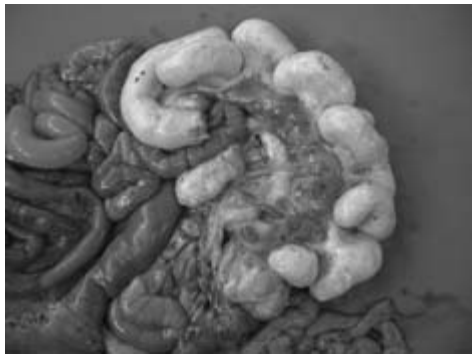


Fig. 3.25 An alimentary carcinoma in a Scottish Blackface ewe. While these tumours are common in bracken areas, they are also a sporadic cause of ill thrift in adult sheep elsewhere.

the intestinal mucosa over the Peyer's patches and free blood in the intestinal contents. The diagnosis is supported by haematological findings of thrombocytopenia, leucopenia and anaemia.

Prevention of bracken poisoning is difficult. The plant is difficult and expensive to control, partly because of its extensive rhizome system. Hay should not be made from fields infested by large amounts of bracken.

In recent years, concerns have been raised about the carcinogenic effects in man of long-term exposure to bracken spores, although spore production is only significant during warm and dry summers.

Rhododendron poisoning

Rhododendrons (*Rhododendron ponticum*) (Fig. 3.26) and azaleas (*Rhododendron* spp.) contain andromedotoxin and related compounds which give rise to hypotension and respiratory depression and also depress the central nervous system after an initial, transient period of excitation. Rhododendron poisoning is most commonly seen in sheep following periods of heavy snowfall when they are forced to graze on hedges.

Fig. 3.26 *Rhododendron* spp.

Fig. 3.27 Typical presenting signs of rhododendron poisoning in a Cheviot ewe.

Fig. 3.28 *Senecio jacobaea*.

Clinical signs commence a few hours after the plant is eaten. The first clinical signs are excessive salivation, the presence of greenish froth around the mouth and nose, attempts to retch, severe abdominal pain and a staggering gait, followed by recumbency, convulsions with opisthotonus, dyspnoea and sometimes death after 3 to 7 days (Fig. 3.27). The main differential diagnosis in pregnant ewes is hypocalcaemia. (In goats, projectile vomiting is sometimes seen, caused by an action of the toxin on the vagal nerve endings in the stomach.) Cases of rhododendron poisoning in humans have been associated with eating honey made from rhododendron flowers.

There are no characteristic postmortem findings, but chewed rhododendron leaves may be recognisable in the rumen.

Several treatment regimes have been recommended, including use of purgatives and stimulants (morphine, coffee and other caffeine-rich drinks). There is little evidence to show that any of these are successful. When provided with supportive therapy, many cases recover within 7 to 14 days. Administration of fluids by stomach tube evokes a painful nervous response, so should be avoided.

Ragwort poisoning

Ragwort (*Senecio jacobaea*) is widely distributed throughout the UK in poorer, sandy pastures, roadsides and waste ground (Fig. 3.28). Growing ragwort plants are generally unpalatable and are avoided, provided that adequate grazing is available. However, ragwort becomes palatable when it is cut, killed by weedkillers or incorporated into hay or silage. Mature flowering plants are most toxic. Ragwort contains several pyrrolizidine alkaloids which are metabolised to hepatotoxic toxins. Cattle and horses are most susceptible to pyrrolizidine alkaloid poisoning. Sheep are seldom affected and are sometimes even utilised to make ragwort-infested pastures safer for grazing cattle.

Unlike cattle, sheep partially enzymatically detoxify ragwort toxins in the rumen, so are less susceptible to poisoning. Nevertheless, ingestion of large amounts of ragwort in young growing sheep can result in chronic ill thrift, associated with liver damage. Some animals die from chronic copper poisoning due to the premature release of copper from damaged hepatocytes.

Gross and histological postmortem findings in cases of ragwort poisoning include diffuse subendocardial haemorrhages, inflammation of the digestive tract, cirrhosis of the liver and spongy degeneration of the brain and spinal cord.

The diagnosis of ragwort poisoning is based on the history of ragwort availability and clinical signs. The diagnosis can be supported by elevated serum AST and GGT concentrations, increased bromosulphophthalein (BSP) dye clearance times or liver biopsy.

Brassica poisoning

Forage rape (*Brassica napus*) and kale (*Brassica oleracea*) provide a balanced source of carbohydrate, protein, fibre and minerals and are traditionally used as a green winter feeds for sheep. However, when fed exclusively, lambs seldom achieve growth rates which would be predicted on the basis of the feed analysis. This is because brassica crops also contain several important toxic substances which cause depressed appetite.

The list of toxic substances includes:

- nitrates
- photosensitive substances
- haemolytic anaemia factors
- oxalates
- glucosinolates – goitrogen precursors, which can induce iodine deficiency
- sulphur and molybdenum – these can induce copper deficiency.

One particularly important toxic substance is a haemolytic anaemia factor, S-methylcysteine sulfoxide (SMCO). SMCO is converted by bacterial fermentation in the rumen to dimethyl disulfide, which causes haemolysis. Dimethyl disulfide precipitates haemoglobin molecules in the erythrocytes to form the Heinz bodies which are characteristic of the disease. These are extruded from the red blood cells and removed from the circulation by the spleen. Severity of the disease is proportional to the SMCO content of the crop. When present in small amounts, the toxin results in poor growth rates. However, when SMCO is present in high concentrations, lambs become anaemic with haemoglobinuria, progressing rapidly to death.

SMCO concentrations in plants can be analysed and brassica crops categorised as low or high potential risk. However, SMCO increases with the age of the crop, so even low-risk varieties can become potentially hazardous as they reach maturity or if they are fed to excess (Fig. 3.29). To avoid these risks, long-keep store lambs should not be grazed on brassica crops for prolonged periods and animals should be provided with a pasture run-off or supplementary feed.

Fig. 3.29 The SMCO content of brassica crops increases with maturity.

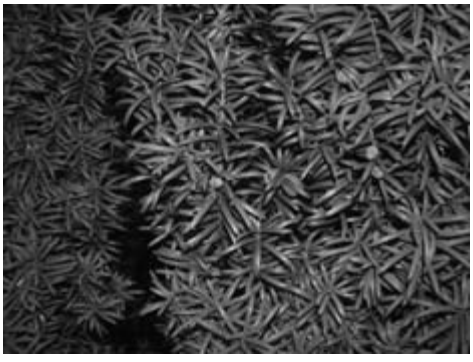


Fig. 3.30 *Taxus baccata.*

The diagnosis of haemolytic anaemia is based on the history of grazing potentially high-risk brassica crops, clinical signs in affected animals and postmortem findings from freshly dead animals of jaundice, haemoglobinuria and anaemia, with congestion of the internal organs. The kidneys are dark and swollen and in peracute cases there is swelling of the spleen. There may be histopathological evidence of moderate hepatic necrosis and glomerulonephritis. Blood samples from sick animals are dark red and watery.

When the disease is suspected, animals should be removed from the crop and carefully introduced to supplementary feed.

Yew poisoning

European yew (*Taxus baccata*) is one of the most poisonous trees in the UK (Fig. 3.30). Yew trees are commonly found in old churchyards, gardens and estates. Animals, inquisitive cattle in particular and occasionally sheep, are periodically poisoned due to careless disposal of pruned branches or fallen branches following storms. All parts of the tree are poisonous and all species of animals are susceptible. The needles do not lose their toxicity after drying. The toxic principle of yew is taxine, a complex mixture of alkaloids that has a strong depressive effect on the heart. Taxine probably also affects the respiratory centre in the brain.

Yew poisoning is usually seen as sudden death. If clinical signs are seen, they include trembling, bradycardia, dyspnoea and collapse.

On postmortem examination, the abomasum is usually distended with gas. If death has been delayed for a few hours, intense abomasal inflammation, congestion of the spleen, hyperaemia of the lungs and liver, and cardiac dilation are sometimes seen. Yew twigs or leaves may be seen within the ruminal contents.

In ruminants known to have eaten yew, rumenotomy, removal of the contents and supportive therapy is the only treatment option.

Laburnum poisoning

Laburnum (*Laburnum anagyroides*) trees are commonly found in parks and gardens (Fig. 3.31). All parts of the tree are toxic, especially the bark and seeds. All species (especially horses) are susceptible to laburnum poisoning. The main toxic principle of laburnum is the alkaloid, cytisine, which stimulates the respiratory centre of the

Fig. 3.31 *Laburnum anagyroides.*

Fig. 3.32 Hemlock can be recognised by its smooth, spotted stem and by the distinct smell of mice emitted by the plant when crushed or bruised.

brain, causes excitation of skeletal muscle and paralyses peripheral sympathetic nerve ganglia.

Poisoned animals show abdominal pain, accompanied by muscular spasms, salivation, incoordination, regurgitation and diarrhoea.

There are no specific postmortem findings, except laburnum leaves may be seen in the ruminal contents. No specific treatment has been described.

Hemlock poisoning

Hemlock (*Conium maculatum*) is common on the banks of streams, in hedgerows and along the borders of fields (Fig. 3.32). Toxicity is due to a group of alkaloids, including coniine. All parts of the plant are toxic. Matured hay containing hemlock is unlikely to be harmful, but fresh leaves are particularly toxic. Coniine has a nicotine-like action, first stimulating and then depressing autonomic ganglia, and a curare-like effect in paralysing the motor nerve endings to the skeletal muscles.

Clinical signs are seen within a few hours of hemlock ingestion, including frequent regurgitation, excess salivation, bradycardia, which changes to tachycardia, tachypnoea followed by slow, irregular and laboured respiration, dilation of the pupils, ruminal atony, weakness and staggering gait. Exhaled air has a peculiar mousy smell. Hemlock poisoning in animals is usually fatal, but treatment may be attempted by administration of tannic acid to neutralise the ingested alkaloid.

Common mineral and inorganic poisonings

With the exception of copper and lead poisoning, heavy metal and other inorganic poisonings such as arsenic, mercury and ionophore toxicities are now extremely uncommon in UK sheep.

Arsenic compounds disrupt cellular enzyme systems, particularly affecting small blood vessels of the gastrointestinal tract, which lose the ability to contract and become dilated. Arsenic poisoning was once common, associated with its inclusion in sheep dips and insecticide or herbicide sprays, but these have now been banned or

superseded by more selective and less dangerous organic chemicals. Other sources of arsenic once included tannery waste, medicinal arsenicals used in the poultry industry, and timber preservatives. Sheep were at risk when they fed from new troughs made from treated timber.

Inorganic mercury salts cause direct tissue necrosis and renal tubular necrosis, while organic mercurials interfere with metabolic activity leading to cell breakdown and necrosis, particularly affecting the brain. Mercury poisoning is now very uncommon due to the discontinuation of mercurial purgative medications and fungicidal seed dressings. Disease outbreaks have also been linked to toxic levels of mercury in the food chain, for example in environmentally contaminated fishmeal.

Until recently, ionophore antibiotics (monensin, lasalocid and salinomycin) were widely used in the UK as growth promoters, coccidiostats and for the prevention of toxoplasmosis. Ionophores are safe at their recommended therapeutic dose, but the safe inclusion rate in compound rations is very low, so poisoning resulting from accidental overdosing was common. Ionophores interfere with potassium transport, damaging mitochondria and reducing cellular energy production. The clinical signs of acute poisoning are depression, weakness, ataxia and diarrhoea. Some animals appear to recover, but subsequently die from myocardial failure, often when moved or heat stressed. Young lambs are particularly susceptible, and toxicity is compounded by exposure to high levels of nitrates or to hepatotoxic plants. Inclusion of ionophores in ruminant rations is no longer permitted and poisoning is now rare.

Copper poisoning

Copper poisoning is very common in sheep flocks, often resulting in significant losses. Chronic poisoning is seen as an acute event following the ingestion of relatively small amounts of copper over a period of time. Acute poisoning is occasionally seen following the administration or ingestion of toxic amounts of copper.

Chronic copper poisoning

Over a period of time, dietary and parenterally administered copper accumulates within lysosomal cells in the liver. Chronic copper poisoning occurs when the lysosomal capacity for copper storage is exceeded, resulting in sudden release of copper into the peripheral circulation, causing intravascular haemolysis and jaundice.

Lysosomal rupture and release of large amounts of copper into the circulation occurs when hepatic copper concentrations exceed about 15 000 μmol/kg DM. However, subclinical liver damage, which may be associated with ill thrift, starts to occur at much lower copper concentrations. Stressful events (for example, ram sales), concurrent liver disease (for example, subclinical ragwort poisoning, subacute liver fluke or cobalt deficiency), or metabolic disturbances caused by other concurrent diseases may result in lysosomal rupture at lower hepatic copper concentrations. Circulatory copper causes intravascular haemolysis through a series of biochemical interactions with red blood cell membrane components. During the haemolytic crisis blood copper concentrations may be 38 to 100 μmol/L (normal range 9 to 23 μmol/L). Debris from haemolysed red blood cells may block renal tubules and cause renal failure with accumulation of metabolites which are toxic to other tissues including the brain.

Fig. 3.33 Sheep on North Ronaldsay (not Cheviots as shown in the picture) are kept on the foreshore for most of their lives on a diet consisting solely of seaweed. The dietary availability of copper is extremely low. As a result North Ronaldsay sheep have evolved to absorb copper extremely efficiently, predisposing them to copper toxicity when exposed to normal dietary copper levels.

Fig. 3.34 Head-pressing behaviour in a Suffolk lamb, caused by high levels of ammonia in the circulation following liver cell rupture due to copper toxicity.

There is variation in breed susceptibility to chronic copper toxicity related to the ability to absorb dietary copper. North Ronaldsay sheep have become adapted to extremely low dietary copper levels, so absorb copper efficiently and are highly susceptible to copper toxicity (Fig. 3.33). Suffolk, Texel, Bluefaced Leicester and various other terminal sire and longwool breeds are relatively susceptible, compared to most types of Scottish Blackface.

Clinical signs

The clinical signs associated with chronic copper poisoning are sudden in onset. Affected animals become progressively ataxic, sometimes with periods of aimless wandering or head pressing (Fig. 3.34), progressing to stupor (Fig. 3.35). As the disease progresses, jaundice and haemoglobinuria develop, with rapid shallow respiration due to anaemia and production of methaemoglobin. Most animals die following a short period of recumbency (Plate 3.7).

Fig. 3.35 Recumbency and stupor in a North Ronaldsay ram due to copper toxicity, associated with grazing terrestrial herbage.

Diagnosis

A provisional diagnosis is based on the feeding and copper supplementation history, clinical signs and postmortem findings.

Serum copper concentrations cannot be used to confirm a diagnosis of chronic copper poisoning, because values are frequently raised above the normal range of 9 to 20 µmol/L associated with other inflammatory conditions of the liver.

On postmortem examination, carcases of sheep that have died as a result of chronic copper poisoning are dehydrated and pale or jaundiced (Plates 3.8, 3.9, 3.10 and 3.11). Livers are pale tan to bronze-coloured (Plate 3.12a and b) and the kidneys have a dark red or black 'gun metal' appearance (Plate 3.13a and b). Urine is dark red or black-coloured (Plate 3.14). Kidney copper concentrations greater than 314 µmol/kg DM are used to confirm a diagnosis of copper poisoning. Large amounts of copper are released from the liver following lysosomal rupture, hence estimations of liver copper concentrations are not always useful.

Management of disease outbreaks

Whenever clinical signs of copper poisoning are identified in individual sheep, it is likely that other animals in the same group will have subclinical liver damage or hepatic copper concentrations that will lead to lysosomal rupture in the near future. Serum copper concentrations are of limited value for the identification of these animals, because values usually remain within the normal range until the hepatic storage capacity is exceeded. However, elevated AST concentrations above 105 iu/L can provide a useful, non-specific, early indicator of subclinical hepatic damage, enabling the identification and timely treatment of individual at-risk sheep.

In the past, the standard treatment regime for chronic copper poisoning involved subcutaneous injection of 3.4 mg/kg of ammonium tetrathiomolybdate (ATTM) on three alternate days (Fig. 3.36). The success rate was poor in animals which were showing advanced clinical signs, but the regime was effective in preventing haemolytic crises in at-risk sheep. Unfortunately, the use of ATTM in food-producing animals is now prohibited under European Union law, because minimum residue levels have not been established.

Treatment with 70 mg/kg sodium calcium edetate, a chelating agent commonly used for the treatment of lead poisoning in ruminants, diluted to a 5 percent solution in normal saline solution and injected intravenously on two consecutive days has been suggested, but there are few data to support its efficacy. Supportive therapy

Fig. 3.36 Subcutaneous injection of ATTM once afforded an effective treatment protocol for individual valuable sheep, but is no longer permissible in the UK.

with anti-inflammatory and antibiotic preparations and intravenous fluid therapy are also indicated for the treatment of secondary lung infection and dehydration.

Copper antagonists such as molybdenum or sulphur can be added to the ration to lower the availability of dietary copper and prevent further hepatic accumulation, either directly, in mineral supplements, via drinking water or by pasture application of approximately 0.25 kg/ha sodium molybdate. Subsequent monitoring is required to avoid deficiency syndromes.

Prevention

Chronic copper poisoning is associated with the efficiency of absorption of dietary copper and with the presence of dietary copper antagonists such as iron, sulphur and molybdenum, rather than with dietary and parenteral copper concentrations alone.

In the highly susceptible North Ronaldsay breed of sheep, or in intensively managed housed sheep, poisoning can result from prolonged feeding of diets with relatively low copper concentrations. The concentrations of copper antagonists (molybdenum, sulphur and iron) are often low in preserved rations fed to housed animals.

Housed sheep should, therefore, not receive copper supplementation before or during housing.

The availability of dietary copper varies between different feeds. Feeds with high concentrations of available copper include:

- Pasture, silage and root crops grown on ground to which large quantities of pig or poultry manure has been applied. (Pig and poultry feeds often contain high quantities of copper.)
- Distillery by-product feeds such as dark grains produced from copper stills.
- Concentrate feeds containing palm oil or molassed sugarbeet pulp. (Wholegrain cereals are relatively poor sources of copper.)
- Milk provides a highly available source of copper and copper absorption is very efficient in young animals, hence lambs suckling dams fed on copper-rich diets are at risk of copper poisoning.

Other potential sources of copper include: access to cattle minerals; copper sulphate foot baths; and fungicide-treated timber.

Acute copper poisoning

Acute copper poisoning occurs sporadically, usually within 24 hours of overdosage with injectable or oral copper preparations used for prevention of deficiency syndromes. Disease occurs when high circulatory copper concentrations overwhelm the hepatic capacity for removal and storage, which is influenced by concurrent disease or administration of other therapeutic agents. Exposure of tissues to toxic copper concentrations results in generalised oedema and gastrointestinal inflammation.

Clinical signs are sudden in onset and non-specific, and include depression, anorexia, rapid heart and respiratory rates, subnormal rectal temperature and mucoid diarrhoea. Collapse and death usually follow within 24 hours, but if the animal survives for a longer period dysentery and jaundice become apparent.

The diagnosis of acute copper poisoning is based on clinical signs and recent history of parenteral copper administration, supported by postmortem findings of generalised oedema, petechiation and inflammation, erosion and ulceration of the

abomasal and intestinal mucosa. Liver copper concentrations are normal and kidney concentrations high.

Acute copper poisoning is prevented by ensuring accurate dosage of copper preparations. As a general rule, injectable copper preparations should not be given to pre-ruminant, stressed or diseased animals.

Lead poisoning

Lead poisoning was once a common, fatal disease of domestic animals. While cattle are most often affected, due to their lack of discriminatory feeding behaviour, disease is also periodically seen in UK sheep in areas where there are high concentrations of lead in soil.

Sources of lead

Traditionally, lead and lead salts have been employed in a variety of ways, although many of these potential sources of poisoning have been superseded by more modern alternatives.

- Lead-based paints – early paints, particularly primers and undercoats, contained extremely high levels of lead. Poisoning cases have been associated with licking paint on gates and shed walls, introducing disused painted objects to the environment, access to flaking paint from adjacent buildings and access to old, burnt or buried paint tins (Fig. 3.37).
- Soil contamination – due to naturally high lead levels, or general urban pollution.
- Industrial sources – soil and atmospheric lead associated with lead mining and smelting.
- Lead in petrol – causing atmospheric pollution, high herbage lead levels close to busy roads, or access to lead in sump oil.
- Metallic lead – batteries, shotgun pellets, fishing weights, solder, asphalt and lead piping (Fig. 3.38).
- Other sources – linoleum, putty and other building materials, or sprays used for fruit trees. Often carried onto pastures by flooding.

Fig. 3.37 (a) Decrepit railway carriages that are used on many hill sheep farms as winter hay stores are a notorious cause of lead poisoning, associated with flaking paint and perished lead-based roof coverings. (b) Flaking lead-based paint on an old railway carriage.

Fig. 3.38 Severe outbreaks of lead poisoning have been associated with the careless disposal of old car batteries.

Drinking water from lead pipes is sometimes implicated as a possible source of lead poisoning, but livestock do not appear to be highly susceptible to poisoning by this route.

Lead toxicity

Most orally ingested lead forms insoluble complexes within the alimentary tract, which are excreted in the faeces. Consequently, only a small proportion is actually absorbed, most of which is subsequently excreted in the bile, milk and urine. The solubility and availability of inorganic lead is associated with acidity of water or soil. Absorption is also enhanced when diets are deficient in protein, calcium, or zinc.

Absorbed lead is initially deposited in the liver and kidneys, but following longer-term exposure it is redistributed into bone, particularly the growth plates. Within the bones, lead results in osteoporosis, although the mechanism is unclear. Bone acts as a reservoir for lead until it becomes saturated, leading to release of large amounts of lead into the circulation and sometimes resulting in acute-onset disease. Pregnancy, poor nutrition, illness and severe exertion may trigger premature release of lead from bone.

Circulating lead becomes strongly bound to red blood cells, increasing their fragility and suppressing haematopoiesis. Mild anaemia and the presence of immature red blood cells in the circulation are, therefore, seen in both acute and chronic lead poisoning. Lead binds to the endothelial cells of the brain capillaries, increasing their permeability and causing brain oedema. Young animals are most severely affected.

Lead readily crosses the placenta, causing foetal damage, teratogenesis, or abortion. Lead can also cause general muscle weakness and immunosuppression.

Clinical signs

Lead poisoning is conveniently described as acute where the signs develop rapidly and chronic where the signs develop over a period of time. The type of disease seen is primarily dependent on the amount of lead ingested, but is also influenced by the source of the lead.

Unlike the situation in cattle, which appear to be extremely susceptible to the effects of lead, acute lead poisoning is uncommon in sheep. The initial signs are apparent blindness, muscle tremors, colic and a staggery gait. The palpebral eye preservation reflex is absent or markedly diminished. This characteristic can be used

to differentiate lead poisoning from polioencephalomalacia, in which this reflex is usually normal. The disease progresses rapidly to a state of recumbency and then death.

Lead poisoning in sheep is usually chronic. Disease is seldom seen during the long cumulative phase and the onset of clinical signs is insidious resulting in periodic fits, anorexia, constipation, emaciation, recumbency and death. Long-term ingestion of metallic lead by pregnant sheep can cause abortion and poor reproductive performance. Stiffness and posterior paresis has been seen in 3- to 12-week-old lambs in old lead mining areas. Osteoporosis leading to brittle bones and bone malformations have also been reported in these areas.

Diagnosis

Circulatory lead is associated with red blood cells, so blood samples must be collected into lithium heparin (green top) tubes. Blood lead levels are usually elevated above 1.2 μmol/L in ill animals, although concentrations fluctuate and a negative diagnosis based a single sample may be equivocal. Blood lead concentrations may also be raised in apparently healthy animals which have been exposed to the same source of lead as ill animals. Apparently healthy animals can therefore be used to show the extent of a problem. Blood lead concentrations decline rapidly once the source of lead is removed, so cannot be used to predict the level of lead accumulation in soft tissues or bone. Determination of lead levels in wool can be used to support a diagnosis of chronic poisoning. Liver samples can be collected by biopsy, which in some circumstances may provide useful diagnostic information.

Haematological examination can be useful in cases of chronic lead poisoning, showing normocytic, normochromic anaemia.

There are no specific postmortem signs, although the kidneys often appear swollen and nephrotic. Histology of the kidneys may show renal tubular necrosis, consistent with heavy metal toxicity. The diagnosis of lead poisoning is supported by demonstration of raised lead concentrations (>~15 mg/kg) in fresh kidney cortical tissue.

Management

Treatment is seldom worthwhile because the disease has usually progressed too far before a diagnosis can be made. Treatment should not be instigated without consideration of the fact that it is inevitably complicated, expensive and protracted. Furthermore, treatment is ineffective at increasing the rate of elimination of lead from animal tissues, so recovered animals may remain indefinitely unfit for human consumption.

Administration of calcium versenate (calcium disodium ethylenediamine tetraacetate) has been successful in cattle and might be considered for high-value sheep. Blindness may persist for several days after general recovery or may continue indefinitely.

Many cases of lead poisoning can be avoided by good waste management on the farm. Prevention is more difficult whenever soil contamination rather than rubbish is the cause of the problem.

Fluorine poisoning

Fluorine is present in all animal tissues, with highest concentrations in bone and teeth. The main sources of fluorine are: spring water arising from fluoride-rich granite

rocks; industrial pollution, in particular arising from aluminium smelters, steel processing plants, coal-fired power stations and phosphate rock processing plants; natural contamination of phosphate- and superphosphate-based fertilisers or mineral supplements; and overseas, volcanic ash.

In the UK, fluorine poisoning is usually chronic. Fluorine displaces hydroxyl groups from bone and teeth, leading to permanent fluoride accumulation. The presence of high concentrations of fluoride in bone has a toxic effect on osteocytes, osteoblasts and odontoblasts, leading to osteodystrophy, particularly in areas of high metabolic activity and remodelling, such as the jaw. The toxic effect on ameloblasts leads to faulty enamel formation and severe dental defects.

Acute fluorine poisoning was seen in New Zealand during 1997, following the eruption of Mount Ruapehu. The clinical signs included ruminal stasis with constipation or diarrhoea, muscle weakness, collapse and death.

Selenium poisoning

Acute selenium toxicosis usually results from excessive selenium supplementation. The difference between normal requirements and toxic amounts of selenium is low and problems often arise due to:

- failure to calculate and administer the correct dose, especially in young animals
- inadequate mixing of multiple trace element drenches or selenium-fortified anthelmintics
- errors in compounding rations
- accidental use of the wrong selenium salt (sodium selenite contains double the available selenium of sodium selenate)
- over-supplementation of pedigree animals in the misguided hope of achieving an added growth response
- simultaneous use of two or more methods of selenium supplementation.

Oral selenium salts and injections of sodium selenate or selenite are potentially dangerous, while slow-release injections of barium selenate paste and oral selenium pellets do not present a high risk.

Acute selenium toxicity is associated with toxic damage to the cardiovascular, respiratory and urinary systems and damage to lymphoid tissue in several organs. Various non-specific clinical signs have been described, including a staggering gait, dyspnoea, tympany, colic, diarrhoea, recumbency, cyanosis and death due to respiratory failure within as short a period as 5 hours after selenium supplementation.

Postmortem findings include subcutaneous haemorrhages, straw-coloured fluid in the pericardium, severe pulmonary oedema, abomasitis, intestinal and hepatic congestion, destruction of renal cortices and haemorrhages of the brainstem. Histopathological findings of marked kidney tubular degeneration and cast formation have been reported. The diagnosis of acute selenium poisoning is supported by elevated selenium concentrations (>1 mg/kg DM) in the liver, heart and kidney.

A successful treatment regime has not been described. Great caution is therefore required when administering selenium supplements.

Chronic selenium poisoning is associated with ingestion over long periods of time of herbage grown on seleniferous soils. The problem, referred to as alkali disease, is well recognised in parts of North America, Israel, Ireland and Australia. In these

regions, the presence of certain seleniferous plants, such as milk vetch, indicates a potential danger of selenium poisoning. Chronic selenium poisoning is also seen in areas with low soil selenium concentrations, associated with the presence of selenium accumulator plants, which concentrate selenium in their leaves.

The clinical signs associated with chronic selenium poisoning are non-specific, and include dullness, ill thrift and anaemia, associated with atrophy of myocardium and toxic liver damage. Brittle hooves with cracks and raised rings below the coronary band have been associated with interference of blood circulation to the extremities. In extreme cases, sloughing of the hoof has been reported.

Mycotoxicoses

Mycotoxins are toxic metabolites produced by fungi. Standing cereal crops, poorly stored feed and mature or dry grass can become contaminated by fungi. Many of the fungi involved require warm and moist conditions for growth, although these conditions are not always optimum for toxin formation.

Many sheep diseases worldwide are associated with fungal metabolites (Table 3.1), although they are only rare causes of economically important disease in the UK. The diagnosis of mycotoxicoses usually depends on the grazing/feeding history and clinical signs. Unfortunately there are few tests available to determine the presence of most mycotoxins in feed or in the animal.

Facial eczema

Facial eczema is caused by ingestion of the mycotoxin, sporidesmin, and results in ill thrift and hepatogenous photosensitisation. The disease occurs in cattle, sheep, goats and deer, but is of greatest importance in sheep. While facial eczema commonly occurs throughout the southern hemisphere, in the USA and in southern Europe, it is a particularly important production-limiting disease in coastal areas of New

Table 3.1 Some of the more important mycotoxicoses. This list is by no means comprehensive. There are many other known mycotoxicoses of farm animals, poultry, fish and companion animals. Furthermore, the role of mycotoxins in many non-specific disease syndromes is unknown.

Disease	Species affected	Fungus genus	Substrate	Toxin
Facial eczema	Sheep Cattle	*Pithomyces*	Pasture litter	Sporidesmin
Ryegrass staggers	All	*Acremonium*	Ryegrass	Lolitrem B
Fescue foot	Cattle	*Acremonium*	Tall fescue	Ergot alkaloids
Paspalum staggers	Sheep Cattle	*Claviceps*	Paspalum	Paspalanine
Ergotism	All	*Claviceps*	Grass and grain	Ergot alkaloids
Fusarium infertility	Pigs Sheep	*Fusarium*	Ryegrass	Zearalenone
Fescue toxicity	Cattle Sheep	*Acremonium*	Fescue	Ergot alkaloids
Slobbers	Cattle	*Rhizoctonia*	Clover	Slaframine
Fusarium toxicosis	All	*Fusarium*	Grain crops	Trichothecenes
Lupinosis	Sheep	*Phomopsis*	Lupins	Phomopsin A
Aflatoxicosis	Pigs Cattle	*Aspergillus*	Grain crops	Aflatoxin
Citrinin toxicosis	Pigs Cattle	*Penicillium*	Grain crops	Citrinin
Ocratoxin	Pigs	*Aspergillus*	Grain crops	Ocratoxin

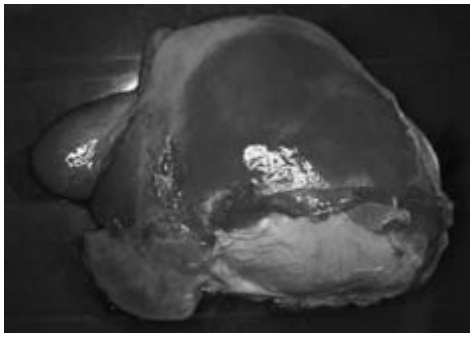


Fig. 3.39 Loss of the normal lobar appearance in the cirrhotic liver of an ill-thrifty ewe, resulting from sporidesmin toxicity.

Fig. 3.40 Early photosensitive dermatitis leading to rubbing of the face of a New Zealand Romney ram.

Zealand. Disease outbreaks usually occur during late summer and autumn. In severe outbreaks up to 3 percent of the flock can show clinical signs of disease. However, the most important economic losses are associated with the high prevalence of subclinical ill thrift, which might affect up to 50 percent of the flock. The severity of the disease can be controlled but the management and treatment methods required are tedious and may not fit well with normal farm practices.

Facial eczema is only sporadically reported in sheep in the UK, although this situation could change if the current trend towards warmer weather continues. The following description of facial eczema in New Zealand sheep flocks also provides an example of some of the general farm management principles involved in the control of many sheep diseases.

Sporidesmin is present in the spores of the saprophytic fungus *Pithomyces chartarum*, which multiplies in the mat of leaf litter beneath shaded pasture, when weather conditions become humid (close to 100 percent humidity) following a prolonged warm period (>20°C, with a minimum night temperature of 14°C). Young spores are more toxic than aged spores. Under ideal conditions, a significant increase in spore numbers can occur rapidly, although several such rises are usually required before signs of the disease are seen.

After ingestion of the fungal spores, sporidesmin is rapidly absorbed and becomes concentrated in the liver where it causes epithelial necrosis of the bile ducts with periductal oedema and inflammation, leading to cirrhosis (Fig. 3.39). Serum GGT concentrations can be used to diagnose subclinical disease. Hepatic damage results in ill thrift at all stages of the production cycle: in lambs this can result in failure to reach marketable weights, while in breeding ewes, in addition to the direct consequences of ill thrift, sporidesmin toxicity can decrease ovulation rates. Ram fertility may also be compromised. Severely affected animals become photosensitised (Figs 3.40 and 3.41) due to the derangement of biliary excretion of phylloerythrin, jaundiced, emaciated and recumbent.

Outbreaks of facial eczema are both distressing and expensive, therefore intensive monitoring is performed in high-risk areas to assess the likelihood of disease and expedite preventive measures (Fig. 3.42). Monitoring consists of recording of weather conditions and measurement of pasture spore counts. Those greater than

Fig. 3.41 Severe photosensitive dermatitis on the face of a New Zealand Romney ram caused by sporidesmin toxicity.

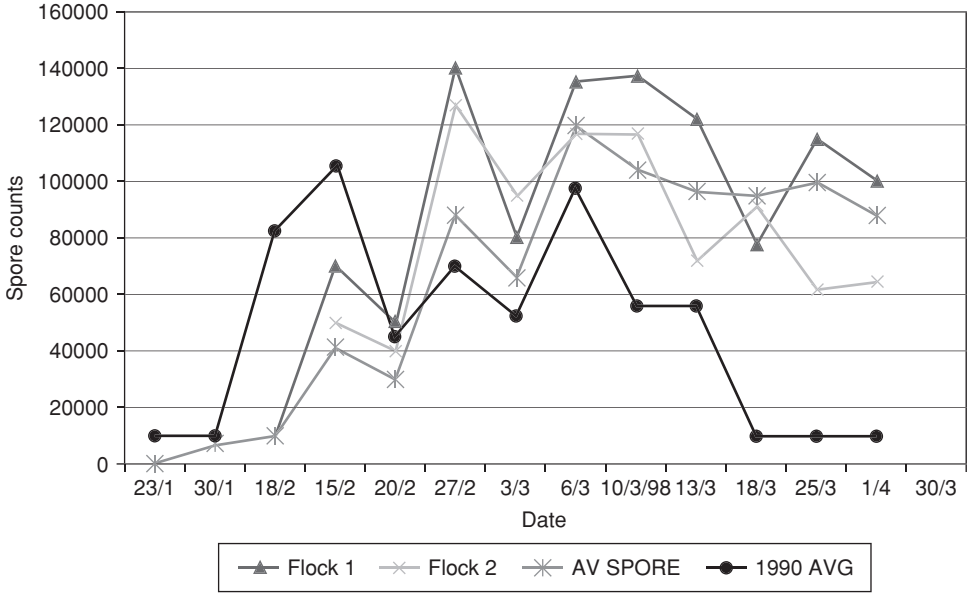


Fig. 3.42 *P. chartarum* spore count monitoring data from a New Zealand sheep farm. Outbreaks of disease occur whenever counts exceed about 100 000 per gram of grass, which usually follows a series of lower peak counts.

100 000 per gram of grass are considered to be dangerous, although the threshold count depends on the age of the spores, pasture length and density, time spent grazing the risk pasture and previous exposure to sporidesmin. During the late summer and autumn risk periods, regional spore counts are published regularly in the farming press. Many New Zealand farmers also perform their own spore counts using spore trapping or pasture washing methods, thereby identifying safe and dangerous paddocks. Rising spore counts in high-risk, shaded areas usually provide sufficient warning for instigation of preventive measures before the disease appears. When counts rise rapidly, or reach a threshold value, the options are to move stock to safer pasture, to instigate protective treatment with zinc salts, to apply fungicides to the pasture, or a combination of these practices.

The toxicity of high-risk pastures can be reduced substantially by spraying with a benzimidazole fungicide. It is normal practice to spray enough pasture for 7 days

grazing, 5 days before stock are introduced; provided the pasture does not receive heavy rainfall within 3 days of spraying, it remains safe for 6 weeks. The practice is widely adopted, but can be expensive, especially when it has to be repeated due to rainfall, or a prolonged risk period.

Oral administration of large doses of zinc salts before exposure to toxic levels of sporidesmin effectively reduces the severity of liver damage, although the mechanism is unclear. However, zinc salts are of little therapeutic value when administered after exposure to sporidesmin. Weekly oral dosing with a zinc oxide suspension provides effective control of facial eczema and is widely practised despite the impracticality of regular gathering of large flocks of sheep and considerable difficulties of keeping the zinc oxide in suspension for its administration through a dosing gun. Sheep are dosed weekly with 0.33 g zinc oxide per kg liveweight. Wax-coated slow-release zinc oxide intraruminal boluses slowly dissolve in the rumen to provide about 6 weeks of protection against facial eczema. In an average year, a single bolus administered in advance of the high-risk period will provide adequate protection, but during some seasons, a second bolus may be required. The boluses are useful where other management practices are impractical, for example in the face of an outbreak of ryegrass staggers. In dairy cattle zinc sulphate can also be administered through drinking troughs or by pasture spraying, but these methods are ineffective for the prevention of facial eczema in sheep.

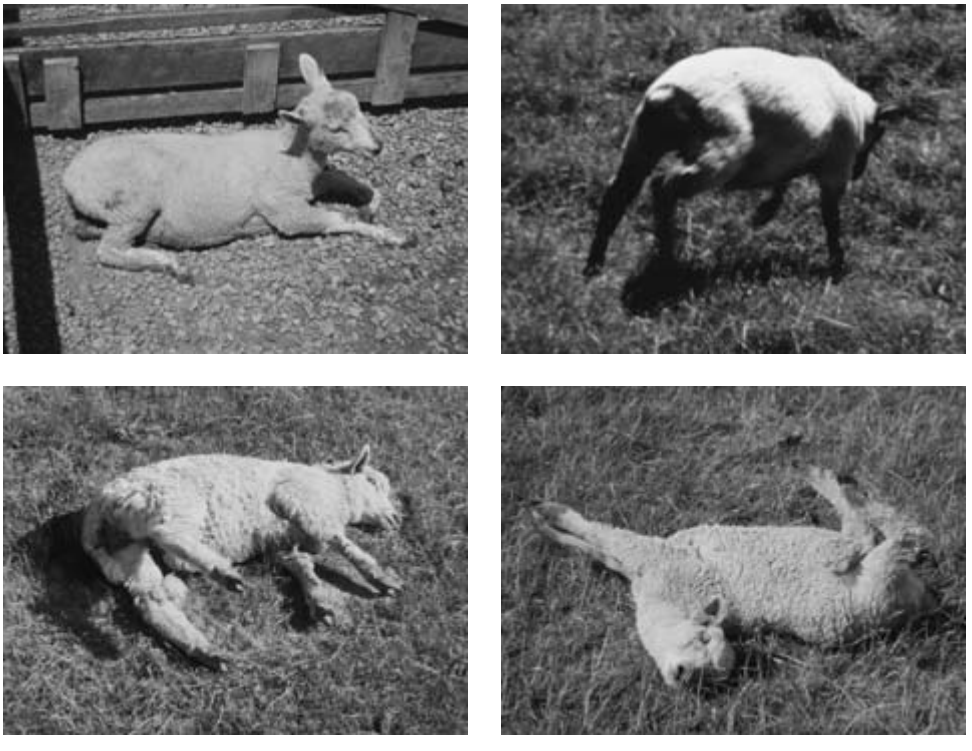
Outbreaks of facial eczema can be avoided by judicious farm management practices, such as the use of brassica crops during high-risk periods, feeding of hay, avoidance of close grazing, and removal of stock from high-risk areas. On farms where the risk of facial eczema is severe, ewes can be mated early to terminal sires, to ensure that lambs are finished and off the farm before the risk period. Cast-for-age ewes, purchased from hill areas with no history of facial eczema, are less susceptible to the cumulative effects of liver damage than homebred lowland ewes.

The within-breed heritability of susceptibility to facial eczema of 0.42 is sufficiently high to enable selection for the trait. Selection involves challenging ram lambs with small doses of sporidesmin, and assessing subsequent liver damage by the measurement of serum GGT concentrations, with the smallest rise in serum GGT concentration indicating the most resistant rams. Selection for resistance to facial eczema has been highly successful, based on farmers' observations and the amount of sporidesmin required to produce a rise in GGT.

Ryegrass staggers

Ryegrass staggers is a neurological disease characterised by tremors and incoordination caused by the mycotoxin lolitrem B. The disease affects sheep, cattle, deer, goats and horses. Severe outbreaks of ryegrass staggers are common in Australia and New Zealand, under close grazing conditions during summer and autumn in pasture-fed livestock. The disease occurs periodically in many other countries, including the UK. The disease is disturbing and disruptive to normal farm practices, but its economic importance has been difficult to determine. Stock losses ranging from 2 to 10 percent have been reported.

Lolitrem is produced by the perennial ryegrass endophyte *Acremonium loliae*. The prevalence of the endophyte in perennial ryegrass is greatest during summer and autumn, in particular in the plants' lower sheaths, old leaves and seeds. Close grazing



Figs 3.43, 3.44, 3.45 and 3.46 Nervous signs associated with ryegrass staggers in New Zealand lambs.

and pasture predominance of perennial ryegrass increase the risk of disease. *A. loliae*-infected ryegrass is largely resistant to attack by the Argentine stem weevil (*Listronotus bonariensis*), an important agronomic pest. Therefore, many modern ryegrass cultivars have been selected for high levels of the *A. loliae* endophyte. Such selection has inadvertently resulted in an increased incidence of ryegrass staggers. High-endophyte ryegrasses produce a number of other toxins, which can reduce their palatability and depress growth rates in lambs.

Sheep develop clinical signs within 1 to 2 weeks after introduction to toxic pasture. The morbidity and severity of ryegrass staggers vary considerably between flocks and between seasons, and the susceptibility of individual animals to the disease varies widely. At rest, the only visible signs may be fine tremors of the head and neck (Fig. 3.43), but when animals are disturbed, clinical signs immediately become apparent with development of head nodding and jerky movements of the neck and limbs. Moderately affected animals have a wide-based, swaying stance and a high-stepping gait (Fig. 3.44). Severely affected animals show a stiff-legged gait and usually collapse when moved. It is not uncommon for sheep to collapse head-first before rolling into lateral recumbency (Fig. 3.45), with their necks arched back and limbs extended (Fig. 3.46). Such rigid tetanic spasms persist for several minutes, usually followed by sudden apparent relaxation and rapid recovery. In sheep, clinical signs persist for about 1 week after removal from toxic pasture, but apparently recovered animals are predisposed to further severe episodes of the disease when reintroduced to high-endophyte ryegrass pastures.

Figs 3.47 and 3.48 Ryegrass staggers interferes with the ability to perform routine procedures such as anthelmintic treatment.

While the disease is distressing to observe, most losses are associated with misadventure, such as drowning in water troughs and streams. A few sheep become permanently recumbent and require humane destruction. Movement of animals can become impossible, interfering with normal management practices (Figs 3.47 and 3.48), such as the transport and sale of finished lambs, grazing rotation, parasite control and zinc dosing for the prevention of facial eczema. Affected rams may have difficulty mating ewes. Furthermore, the inability to manage pasture effectively for ewe nutrition before mating can result in reduced lambing percentages during the following spring.

The mode of action of the tremogens has not been determined, and there is no specific treatment for the disease. The avoidance of close grazing of high-endophyte, ryegrass-dominant pastures is not always possible, especially during dry summer conditions. The disease can be managed by feeding supplementary hay, or the judicious use of brassica crops, but these strategies are not always practical due to the prolonged risk period. Reseeding pastures with endophyte-free perennial ryegrass cultivars is expensive, and does not solve the problem of control of the Argentine stem weevil. Consequently the problem of ryegrass staggers remains complicated and unresolved.

Fusarium *toxicosis*

Unlike the symbiotic relationship which exists between perennial ryegrass and the *Acremonium loliae* endophyte, *Fusarium* spp. are active plant pathogens, causing discolouration (often pink, but the colour varies depending on the fungus species) of grain crops (Fig. 3.49). Mould growth is greatest during cold and wet weather. *Fusarium* spp. produce several toxins, including various trichothecenes and zearalenone.

Trichothecene toxins, also produced by several other cereal and forage crop storage moulds (Fig. 3.50), are cytotoxic to lymphocytes and can cause generalised microvascular haemorrhages. Ingestion of large amounts of toxin can result in a variety of non-specific hepatic and nervous signs and haemorrhagic disorders associated with necrotic lesions of the dermis, oral cavity, gastrointestinal tract, liver and kidneys. Immunosuppressive effects of trichothecene toxins have been incriminated in

Fig. 3.49 Various mycotoxicoses are associated with fungal growth on poorly stored grain.

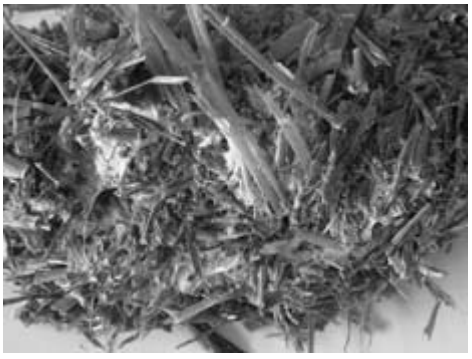


Fig. 3.50 Moulds on stored forage crops can produce a variety of mycotoxins.

decreased host resistance to a variety of common diseases, although it usually proves impossible to define precisely the role of mycotoxicosis in these cases.

Zearalenone toxin has potent oestrogen-like characteristics. Zearalenone poisoning associated with *Fusarium* spp. growth on mouldy stored feeds is most common in pigs, where in addition to poor fertility and stillbirths, vulvovaginitis with extreme swelling of the vulva is sometimes seen. A putative link has been implicated between zearalenone and similar clinical signs in concentrate-fed sheep. Signs appear within 3 to 6 days after feeding mouldy grain and abate soon after the feeding stops, although in the case of male animals it may take several months before normal fertility returns.

In some parts of the North Island of New Zealand poor reproductive performance in ewes has been associated with high pasture concentrations of zearalenone associated with saprophytic *Fusarium* spp. fungi. The pasture and environmental conditions which favour the growth of *Fusarium* spp. are similar to those for *Pithomyces chartarum*. When the daily zearalenone intake of cycling ewes exceeds 3 mg/day their oestrous cycle length is decreased and duration of oestrus increased, resulting in reduced fertility. The severity is dependent on the period of exposure and daily dose rate. The diagnosis of *Fusarium* infertility is supported by the measurement of zearalenone concentrations in pasture or urine. The principles and frustrations of management control are similar to those for facial eczema and ryegrass staggers.

Ergotism

Ergots appear when compacted masses of some species of fungus (sclerotium) replace grass seeds and project from the ear of the grass as a dark horn-like structure. Ergots are a resting stage of the fungus from which, after overwintering in the soil, spores develop and infect new plants. Diseases associated with ergot poisoning are rare in the UK.

The most important ergot disease in the UK is caused by *Claviceps purpurea*, which is a parasitic fungus of rye and other small grain crops. *C. purpurea* produces several ergot alkaloids (including ergotamine, ergometrine and ergotoxine) which have stimulatory, then depressive effects on the central nervous system and cause arteriolar, intestinal and uterine smooth muscle constriction, leading to capillary damage, arteriolar thrombosis, ischaemia and gangrene. Ergotism is seen in all

species, associated with consumption of large amounts of seed heads containing ergots over a period of 2 to 6 days.

In the early stages of the disease the extremities of affected animals are painful and inflamed, leading to coldness, numbness and the development of dry gangrenous lesions in the lower legs, ears and tail. Affected animals become ill thrifty. Irritation of the digestive tract can occur following ingestion of large amounts of ergot, accompanied by abdominal pain and vomiting. Abortion can occur, but is not a common feature of ergotism.

Paspalum staggers (convulsive ergotism) is a similar disease to ryegrass staggers, caused by tremorgens present in *Claviceps paspali* ergots of paspalum grasses. The clinical signs, management and control are essentially similar to those for ryegrass staggers.

Fescue toxicity

Fescue toxicity is seen during the autumn and winter in Australia, New Zealand and North America in cattle and sheep grazing tall fescue. The disease is caused by ergot alkaloids produced by the fescue endophyte *Acremonium coenophialum*. There is a similar symbiotic relationship between tall fescue and *A. coenophialum* to that between ryegrass and *A. loliae*, where *A. coenophialum* offers the plant protection against insect attack and tall fescue offers the fungus a substrate, protection and means of dispersal.

Ergot alkaloids are vasoconstrictor compounds which affect the extremities of cattle and sheep during cooler weather. Consequent disease is characterised by severe pelvic limb lameness 10 to 14 days after mycotoxin exposure. New cases are seen for up to one week after removal from the high-risk pasture. Affected animals rapidly lose weight. The first sign of the disease is dry skin at or near to the coronary band, progressing to hair loss and moist gangrene over the lower extremities. The distal limb and tips of the ears may slough off due to complete cessation of blood circulation.

Lupinosis

Lupinosis is caused by the fungus *Phomopsis leptostromiformis*, which grows on the stems of dead, wild lupin plants. The disease occurs worldwide, but is only of major importance in western Australia and parts of North America where conserved lupins are fed to livestock. Most disease outbreaks are seen in sheep.

Optimal fungal growth occurs during humid and overcast weather when temperatures exceed 25°C. *P. leptostromiformis* produces hepatotoxins, which cause inappetence, weight loss, lethargy and jaundice. An acute disease syndrome resulting from a large intake of toxin over a short period of time is characterised by high morbidity and mortality rates, while chronic lupinosis, associated with repeated intakes of small quantities of toxin, is characterised by ill thrift affecting a variable proportion of the flock. Postmortem findings are similar to those associated with facial eczema.

Aflatoxicosis

Aflatoxins are metabolites produced by *Aspergillus flavus* and possibly other *Aspergillus* spp. and *Penicillium* spp. fungi growing on stored feeds. *A. flavus* is ubiquitous

Fig. 3.51 Most clostridial diseases result in sudden death.

in stored food, but outbreaks of aflatoxicosis in the UK are mostly associated with feeding grain imported from subtropical regions.

Aflatoxins cause liver centrilobular necrosis, bile duct hyperplasia, cirrhosis and fatty infiltration of the liver. Aflatoxins are also immunosuppressive and carcinogenic.

Various clinical signs have been associated with aflatoxicosis, including apparent blindness, epistaxis, photosensitive dermatitis, circling, ataxia, haemorrhagic diarrhoea, tenesmus and abortion. Affected animals generally die within 48 hours of the onset of clinical signs. On postmortem examination, a range of liver lesions, sometimes including tumours, and serosal and subcutaneous haemorrhages are seen. The diagnosis of aflatoxicosis can be supported by raised serum concentrations of liver enzymes and the identification of aflatoxin in blood.

Known contaminated grain should be diluted with clean feed or disposed of. Aflatoxin formation in stored grains can be prevented by ensuring that stores are cleaned out before use, grain moisture content is below 13 percent and the store is adequately ventilated to prevent damp spots forming.

Slobbers

Slobbers is caused by a parasympathomimetic toxin, slafremine, which is produced by the *Rhizoctonia leguminicola* endophyte of red clover and other legumes. The disease is characterised by salivary episodes which begin within 30 minutes of ingestion of contaminated clover hay and continue for several days. Slobbers occurs worldwide and is problematic in parts of North America and Japan. The disease is most important in cattle, but sheep can also be affected.

Clostridial diseases

The various clostridial bacteria associated with disease in sheep are anaerobic, spore-forming, toxin-producing organisms, which are normally present in soil, faeces or intestinal contents of healthy animals (Table 3.2). With the exception of botulism, clostridial diseases result from toxin production following the opportunistic, rapid multiplication of bacteria in the animal. A variety of management, disease and other factors enable such rapid clostridial multiplication. Most of the clostridial diseases are characterised by peracute fatal illness (Fig. 3.51). The clostridial bacteria are

Table 3.2 The major clostridial diseases of UK sheep.

Disease name	*Clostridium* spp. involved
Lamb dysentery	*C. perfringens* type B
Pulpy kidney	*C. perfringens* type D
Struck	*C. perfringens* type C
Tetanus	*C. tetani*
Blackleg	*C. chauvoei*
Malignant oedema	*C. septicum*, *C. novyi* type A and probably others
Black disease	*C. novyi* type B
Braxy	*C. septicum*
Abomasitis	*C. sordellii*
Botulism	*C. botulinum* types C and D

Fig. 3.52 Outbreaks of enterotoxaemias such as pulpy kidney often follow a change in diet, such as moving store lambs onto brassica crops.

ubiquitous, so eradication of clostridial diseases is impossible, but good control can be achieved by vaccination. Clostridial diseases of sheep are, therefore, endemic throughout the UK.

Sheep farming had become untenable in many parts of the UK before the development in the 1950s of the first multi-component clostridial vaccines. While modern vaccines provide excellent protection against the important clostridial diseases of sheep, many flocks are improperly vaccinated or not vaccinated at all, sometimes in an ill-advised attempt to reduce production costs and sometimes associated with adherence to certain irrational rules governing organic production.

Enterotoxaemias due to Clostridium perfringens

Clostridium perfringens bacteria are normally present in the intestinal contents of sheep. In healthy animals a balance exists between multiplication and passage into faeces, maintaining a low level of infection. Enterotoxaemias occur when management conditions result in this balance being upset. *C. perfringens* is saccharolytic and can multiply rapidly when the anaerobic conditions in the abomasum and small intestine are combined with the presence of large quantities of fermentable carbohydrate. These conditions occur when sudden changes in diet enable the overflow of undigested feed to the small intestine (Fig. 3.52). Conditions which result in gut stasis, such as insufficient dietary fibre or severe gastrointestinal parasitism, may also contribute to the build-up of toxins in the intestine.

C. perfringens bacteria produce non-toxic protoxins which are converted to toxins by the action of digestive enzymes. The three major lethal necrotising toxins are the alpha, beta and epsilon toxins and the pathology of the enterotoxaemia is determined by the combination and amounts of these toxins.

The most important enterotoxaemias are lamb dysentery and pulpy kidney. Cases of struck are increasingly reported. *C. perfringens* type A has been implicated in a fatal haemolytic syndrome of young lambs, but is rare.

Pulpy kidney

Pulpy kidney is a common, peracute and usually fatal disease of sheep of all ages caused by the epsilon toxin of *C. perfringens* type D. High levels of epsilon toxin in the gut cause mucoid diarrhoea and vascular permeability, thus enabling its own absorption. Once absorbed, the epsilon toxin causes oedema and haemorrhage in several tissues including the brain, lungs and myocardium. The disease occurs most frequently in well-grown lambs between 4 and 10 weeks old, or fattening lambs between 6 months and 1 year old, associated with a change in diet such as movement onto silage aftermath or brassica crops. Usually the best lambs are affected first. Pulpy kidney is also common in ram lambs and shearlings as they are prepared for sale.

Following a feed change, partially digested food containing carbohydrate can escape to the small intestine, where is provides a substrate for *C. perfringens* until the ruminal microflora adapt to the improved diet. Most cases of pulpy kidney present as sudden deaths, but animals are occasionally seen alive with hyperaesthesia and ataxia, which progresses rapidly to recumbency, opistotonus, convulsions and death. Non-specific neurological signs associated with focal symmetrical encephalomalacia, and diarrhoea are seen in lambs which survive longer. In extreme cases, losses of between 10 and 15 percent have been reported.

The initial diagnosis of pulpy kidney is made on the basis of a history of sudden deaths in well-grown, unvaccinated lambs fed on a carbohydrate-rich diet. However, confirmation of this diagnosis depends on postmortem findings. On gross postmortem examination of fresh carcases there is often an excessive amount of pale yellow, fibrinous fluid in the pericardium (Fig. 3.53) and haemorrhages on the epicardium and endocardium. The lungs are oedematous and congested (Fig. 3.54) and the liver is swollen and friable. The brain may be swollen with cerebellar coning. In fresh cases, the kidneys are swollen and pale, but they autolyse rapidly so that the parenchyma is washed away when they are held in running water (Fig. 3.55). The intestines are gas filled and decompose rapidly. Carcases of sheep which died from pulpy kidney characteristically decompose rapidly, limiting their diagnostic value.

The diagnosis of pulpy kidney can be supported by demonstration of the presence of glucose in the urine, although in animals which have been dead for several hours this may be absent due to bacterial proliferation. Positive ELISA test results for identification of epsilon toxin in intestinal contents or peritoneal fluid support, but do not confirm, the diagnosis because immune animals may have high concentrations of epsilon toxin, but not suffer from its effects. The diagnosis can be confirmed by brain histopathology, which may show symmetrical foci of perivascular oedema, vascular haemorrhages and focal malacia involving the meninges, cerebral cortex, thalamus, mid brain and cerebellum. These changes are characteristic of focal symmetrical encephalomalacia.

Fig. 3.53 Fibrinous fluid in the pericardium of a lamb that died as a result of pulpy kidney.

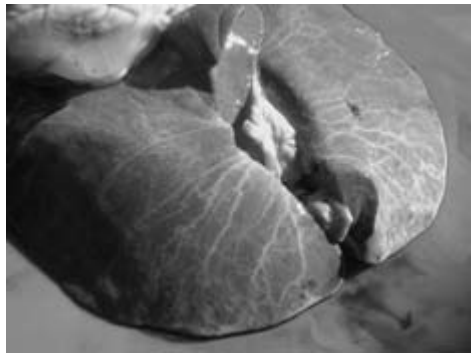


Fig. 3.54 Pronounced interstitial tissue in oedematous lungs of a lamb which died as a result of pulpy kidney.

Fig. 3.55 The parenchyma of this kidney from a freshly dead lamb was washed away when it was held under running water, suggesting but not confirming a diagnosis of pulpy kidney.

Fig. 3.56 A sternally recumbent Scottish Blackface ewe which subsequently died from struck.

Struck

Struck is a rare peracute disease of adult sheep caused by the beta toxin of *Clostridium perfringens* type C. The disease was first reported in the Romney Marsh area of Kent and occurs most commonly in England, but has also been seen in south-east Scotland. Elaboration of toxins results in enteritis, peritonitis and sudden death. Live animals may be seen collapsed in sternal recumbency (Fig. 3.56), with cyanotic mucous membranes (Plate 3.15), rapid shallow respiration and dark coloured, mucoid scour.

The diagnosis of struck depends on the history of dietary change in unvaccinated sheep and postmortem findings. An excessive amount of pale pink fluid is usually present in the pericardium, thoracic and abdominal cavities. Haemorrhages may be seen in the lungs. Haemorrhagic enteritis of the abomasum (Fig. 3.57) and proximal intestinal tract may be seen in fresh carcases and the caecum may be distended with digested blood-tinged fluid. The diagnosis is supported but not confirmed by the anaerobic culture of *C. perfringens* from intestinal contents, and positive beta toxin ELISA results from intestinal contents or peritoneal fluid.

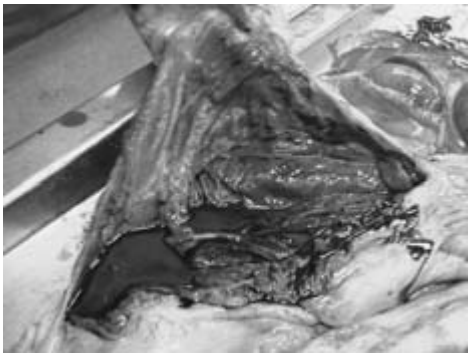


Fig. 3.57 Severe abomasitis and haemorrhagic abomasal contents seen on postmortem examination of the ewe shown in Fig. 3.56.

Prevention of enterotoxaemia

Effective prevention of enterotoxaemia is achieved through vaccination using a multi-component vaccine containing toxoids of *C. perfringens* in an adjuvant (usually aluminium hydroxide, which primes the immune response). Vaccination of ewes protects their newborn lambs against lamb dysentery through colostral transfer of passive immunity. Previously unvaccinated ewes should be given an initial course of two vaccine injections 4 to 6 weeks apart when they enter the breeding flock, followed by an annual booster about 6 weeks before lambing. This pre-lambing booster with a multi-component vaccine also ensures transfer to lambs of passive protection against pulpy kidney, which lasts up to 16 weeks of age. Lambs born to vaccinated dams should receive a first sensitiser dose at about 8 to 12 weeks old, followed by a second booster at least 4 weeks later. All of the multi-component clostridial vaccines available in the UK protect against pulpy kidney, but not all protect against lamb dysentery (Table 3.3). The use of the product which does not protect against lamb dysentery should be reserved for finishing lambs.

Vaccination should be combined with good stock husbandry, including good hygiene at lambing, the insurance of adequate early colostrum intake and careful introduction to improved planes of nutrition.

Management of C. perfringens *enterotoxaemia outbreaks*

Many UK sheep are inadequately vaccinated against clostridial diseases and severe outbreaks of pulpy kidney occur sporadically. In these cases it is important to instigate a vaccination programme immediately. Where a sensitiser, but no booster dose of vaccine has been previously used, a single injection of vaccine in the face of an outbreak usually provides immediate protection.

Even when no vaccine has been used, a single injection of vaccine offers significant protection within 48 hours, probably because most animals are naturally sensitised by the presence of small amounts of epsilon toxin in their intestines. Yarding of animals for vaccination and restricted feeding usually halts the progress of the disease until the vaccine is effective.

Injections of purified beta and epsilon antitoxins of *C. perfringens* can be useful in the face of an outbreak of lamb dysentery. These *C. perfringens* antisera injections are expensive so their use for the management of pulpy kidney is limited to valuable animals.

Table 3.3 Clostridial vaccines and antisera listed in the National Office of Animal Health *Compendium of Animal Medicines* (2007).

Vaccine (manufacturer) and antisera	Against
Covexin 8 (Schering-Plough Animal Health)	
C. perfringens B	Lamb dysentery
C. perfringens D	Pulpy kidney
C. tetani	Tetanus
C. septicum	Braxy and malignant oedema
C. chauvoei	Blackleg and malignant oedema
C. novyi B	Black disease and malignant oedema
C. perfringens C	Struck
C. novyi D	Bacillary haemoglobinuria
Covexin 10 (Schering-Plough Animal Health)[1]	
C. perfringens A	
C. perfringens B & C	Lamb dysentery
C. perfringens D	Pulpy kidney
C. tetani	Tetanus
C. septicum	Braxy and malignant oedema
C. chauvoei	Blackleg and malignant oedema
C. novyi B	Black disease and malignant oedema
C. perfringens C	Struck
C. haemolyticum	Bacillary haemoglobinuria
C. sordellii	Abomasitis
Heptavac and Heptavac P plus (Intervet UK Limited)	
C. perfringens B	Lamb dysentery
C. perfringens D	Pulpy kidney
C. tetani	Tetanus
C. septicum	Braxy and malignant oedema
C. chauvoei	Blackleg and malignant oedema
C. novyi B	Black disease and malignant oedema
C. perfringens C	Struck
Ovivac and Ovivac P plus (Intervet UK Limited)	
C. perfringens D	Pulpy kidney
C. tetani	Tetanus
C. septicum	Braxy and malignant oedema
C. chauvoei	Blackleg and malignant oedema
Lambivac (Intervet UK Limited)	
C. perfringens B	Lamb dysentery
C. perfringens D	Pulpy kidney
C. perfringens C	Struck
C. tetani	Tetanus
Blackleg vaccine (Schering-Plough Animal Health; Intervet UK Limited)	
C. chauvoei	Blackleg and malignant oedema
Tetanus toxoid concentrated (Intervet UK Limited)	
C. tetani	Tetanus
Tetanus antitoxin Behring (Intervet UK Limited)	
	Tetanus

[1] The duration of passive immunity depends on the antigen, but may be as little as 2 weeks for *C. septicum*, *C. chauvoei* and *C. sordellii*. Lambs can be vaccinated with a 1 ml dose from 2 weeks old.

Other enterotoxaemias

Abomasitis due to Clostridium sordellii

An association has recently been described between acute abomasitis in sheep of all ages and *Clostridium sordellii*. *C. sordellii* is widely distributed in the environment, but the reasons are unclear for its establishment and rapid multiplication in the abomasum, with the production of potent lethal and haemolytic toxins. Usually the strongest and most rapidly growing lambs are first affected, with correspondence to a high level of carbohydrate feeding.

Disease is characterised by sudden death, most commonly in intensively reared, creep-fed lambs, aged 3 to 10 weeks. Lambs which are seen alive appear bloated and profoundly depressed. The disease is usually sporadic in incidence, but losses of up to 8 percent of housed, creep-fed young lambs have been reported.

The gross postmortem findings include intestinal distension, pale to congested mucous membranes, enlargement and haemorrhage of the mesenteric lymph nodes (Fig. 3.58), subcutaneous oedema and congestion of blood vessels. In young lambs, the abomasum is consistently partially distended and displaced so that it lies across the body cavity. The abomasal wall is thickened, associated with subserosal emphysema and oedema. Necrotic areas of intense congestion and erosion are present on the mucosal surface of the abomasal folds and small intestine. Carcases of lambs which died from *C. sordellii* enterotoxaemia generally decompose rapidly. Postmortem findings in older animals are less pronounced.

The diagnosis of *C. sordellii* abomasitis is based on gross pathological findings and supported by the identification of Gram-positive rods in Gram-stained smears from necrotic lesions in the abomasal folds. Anaerobic culture of *C. sordellii* from necrotic lesions of fresh carcases and positive indirect fluorescent antibody test results on air-dried smears provide further support for the diagnosis.

Vaccination of ewes and lambs from 2 weeks old with multi-component clostridial vaccine including *C. sordellii* may prove useful for the prevention of sudden deaths in creep-fed lambs. It is uncertain what factors are responsible for initiating rapid bacterial multiplication and subsequent toxin production, but it has been suggested that the risk factors may be similar to those for *C. perfringens* enterotoxaemias. The avoidance of rapid changes in dietary fermentable carbohydrate may, therefore, help to prevent the disease.

Fig. 3.58 Intestinal distension and an enlarged haemorrhagic mesenteric lymph node in a 4-week-old Suffolk lamb.

Fig. 3.59 Outbreaks of braxy are commonly associated with folding on frosted turnips.

Braxy

Braxy is a rare peracute disease of older store and ewe lambs caused by a variety of toxins produced by *Clostridium septicum*, which occurs primarily during late autumn when the first severe frosts occur. *C. septicum* is ubiquitous in soil and ruminant faeces and ingestion of frosted food such as frozen turnips (Fig. 3.59) is believed to provide a suitable environment in the abomasum for multiplication of the organism, invasion of tissues and production of toxin. *C. septicum* produces lethal, necrotising, haemolytic and hyaluronidase toxins and causes severe abomasitis and generalised signs of toxaemia. Affected sheep are seldom seen alive.

The diagnosis of braxy is based on the history of unvaccinated lambs grazing frosted food during late autumn and on the postmortem findings in fresh carcases. As with the other clostridial diseases, carcases autolyse rapidly, however if postmortem examinations are performed on freshly dead animals, the abomasum is oedematous with mucosal and submucosal haemorrhages and sometimes ulceration. Oedema may spread to adjacent tissues and there is usually an excess of serous pericardial and peritoneal fluid. The diagnosis is supported by the identification of large numbers of Gram-positive pleomorphic rods in Gram-stained smears from abomasal lesions. Groups of bacteria can appear filamentous due to formation of chains of rods. Positive fluorescent antibody test results on air-dried smears from the affected areas support but do not confirm the diagnosis. *C. septicum* normally proliferates rapidly in decomposing sheep carcases, overgrowing other more fastidious bacteria, so its identification is not necessarily consistent with the diagnosis of disease.

Control of braxy is by vaccination along with other clostridial diseases using the formol toxoid of *C. septicum.*

Blackleg

Blackleg occurs commonly in a variety of species including sheep, cattle and pigs. Blackleg is caused by toxins produced by *Clostridium chauvoei* which are spore-forming, Gram-positive rods with peritrichous flagellae. Spores of *C. chauvoei* survive in soil for many years, so high levels of contamination can build up on some farms. Viable spores are sometimes brought to the surface by earthworks, such as drainage, having originated from cases of blackleg several decades previously.

C. chauvoei multiplies in an anaerobic environment and produces a number of toxins which cause cellulitis, necrotising myositis, toxaemia and gas formation. Outbreaks

of the disease in sheep usually require some predisposing factor such as docking, castration, shearing under dirty conditions, using dirty needles for routine vaccination, or wintering on root crops. Blackleg in ewes is often associated with poor lambing hygiene and dystocia. The incubation period following infection is 1 to 3 days.

The clinical signs depend on the site of infection. When limbs or back muscles are involved, sheep become pyrexic, stiff and unable to move. There is subcutaneous oedema and gas production, but these signs can be difficult to determine because of the presence of the fleece. Cases of blackleg associated with parturition injury are characterised by erosion of the vulval mucosa, and vulval and perineal oedema with dark red and gassy necrosis extending to adjacent muscles. Bloodstained droplets may ooze from skin (Fig. 1.107). Blackquarter metritis occasionally occurs before lambing, in which case the entire uterus may be oedematous and the foetus dead and anasarcous. When the head is affected, as sometimes occurs in rams during the autumn following fighting, the whole face may swell. Such cases may bleed from the nose and are referred to as malignant oedema or 'bighead'. In all cases, affected animals are dull and the disease rapidly progresses to recumbency, coma and death. *C. chauvoei* infection of lambs' navels is occasionally seen, leading to rapid death.

In common with sheep which have died from other clostridial diseases, the carcase swells and bloats rapidly after death. On postmortem examination affected muscle is oedematous, dark red, crepitous and sometimes rancid smelling when incised. Overlying fascia also oozes oedema fluid. Other signs associated with toxaemia such as pericarditis, lung congestion and a pale swollen liver may also be seen.

The gross postmortem signs of blackleg in freshly dead animals are characteristic, but when animals have been dead for a few hours the signs may become difficult to interpret due to postmortem autolysis. Identification of Gram-positive rods in smears taken from oedema fluid or the margins of the muscle lesions shortly after death may aid diagnosis. Confirmation of the diagnosis can be gained from histopathology of formalin-fixed sections from the edge of lesions, or from positive fluorescent antibody tests for *C. chauvoei* on smears of unfixed tissue from the periphery of lesions. However, postmortem invasion by *C. septicum* can swamp the growth of *C. chauvoei* in animals which have been dead for a few hours. Smears of bone marrow from a rib are useful for fluorescent antibody testing of autolysed carcases, because this is one of the last sites to be invaded by *C. septicum* after death.

Treatment of early cases can be attempted but is seldom effective. Treatment involves cleaning and surgical debridement of any obvious wound, high doses of penicillin, supportive fluid therapy and high doses of corticosteroids.

Blackleg prevention is achieved by vaccination with formalin-killed bacteria and toxoid. These are usually included in a multi-component vaccine along with other clostridial toxoids. Additional hygienic precautions should be taken when lambs are castrated and docked and when assisting ewes with dystocia. Care should be taken to avoid the disposal of lambs' tails or animal carcases close to handling pens or lambing areas. On high-risk farms the routine use of long-acting penicillin at docking and lambing may also be useful.

Black disease

Black disease is a fatal peracute infection of sheep of all ages caused by alpha and beta toxins of *Clostridium novyi* type B. *C. novyi* form large Gram-positive rods with

peritrichous flagellae, which are part of the normal flora of soils and sheep intestinal contents. Under anaerobic conditions the bacteria become motile and it is believed that some pass through the intestinal wall and become lodged as spores in the livers of healthy animals. Liver fluke larvae migrating through the liver parenchyma leave tracts of necrotic debris and inflammatory exudate, which provide suitable conditions for germination and multiplication of *C. novyi* spores and toxin production. Other forms of liver damage may also trigger black disease.

Black disease is characterised by sudden deaths following a short incubation period. Losses can occur throughout the high-risk period of liver fluke larval migration, which sometimes amount to 5 percent. Black disease also occasionally affects cattle.

The diagnosis of black disease is based on the history and knowledge of migrating liver fluke larvae and postmortem findings. In common with the other clostridial diseases, carcases autolyse very rapidly, limiting the value of postmortem diagnosis. In freshly dead animals, subcutaneous blood vessels are engorged, giving the carcase a characteristic black colour. The liver is dark and contains distinct paler areas of necrosis up to 3 cm in diameter, distributed throughout the parenchyma. There is usually evidence of recent migration of liver fluke larvae. Blood-tinged serous fluid is usually present in the pericardium and peritoneum and there may be myocardial petechial haemorrhages. The diagnosis can be supported by the identification of large Gram-positive rods in smears of cut liver tissue and positive fluorescent antibody test results from fresh air-dried smears of liver tissue. However, *C. novyi* is found in the livers of healthy sheep, so these results must be interpreted alongside the history and gross postmortem findings. Postmortem invasion by *C. septicum* often overgrows *C. novyi*, further confusing the diagnosis of black disease.

Control is by vaccination, usually combined with the other clostridial toxoids. Strategic treatments for the control of liver fluke will reduce the incidence of black disease, but not eliminate the disease because the fluke treatments will not remove all migrating larvae.

Bacillary haemoglobinuria (Redwater)

Redwater is primarily a disease of cattle caused by *Clostridium novyi* type D (*Clostridium haemolyticum*), which is occasionally encountered in adult sheep. Spores of *C. novyi* type D are found in soil and in the livers of healthy sheep. The toxin produced by *C. novyi* type D causes intravascular haemolysis. In sheep the disease usually occurs in summer or autumn in animals grazed on wet pasture. Migrating liver fluke larvae may precipitate the disease in some cases. The disease is sudden in onset and clinical signs include fever, ruminal stasis and abdominal pain. Affected animals are anaemic and jaundiced with rapid shallow breathing and dark red urine. Death follows over a period of 2 to 3 days.

The diagnosis of redwater is based on history, clinical signs and postmortem findings, which include jaundice, generalised subcutaneous gelatinous oedema, myocardial haemorrhages, and blood-tinged pleural, pericardial and peritoneal fluid. Pale necrotic foci up to 5 cm in diameter are often present within the parenchyma of the liver. The diagnosis is supported by the demonstration of large Gram-positive rods in Gram-stained smears of liver tissue and positive fluorescent antibody test results on fresh air-dried smears.

Fig. 3.60 Malignant oedema causing swelling of the head of a Scottish Blackface ram lamb.

Some cases may be seen early enough to attempt treatment with high doses of penicillin. Control can be achieved by vaccination.

Malignant oedema

Malignant oedema (bighead) is a non-specific disease of sheep of all ages, which is characterised by massive facial swelling with pitting oedema and gas production (Fig. 3.60). The disease is most commonly seen in ram lambs during the autumn associated with contamination of fighting wounds of the head. The disease is primarily a cellulitis, with infection spreading between fascial planes. The skin overlying the lesions may be gangrenous and there is gelatinous subcutaneous oedema and crepitus.

Cases of malignant oedema are associated with wound infections involving *C. septicum*, *C. chauvoei*, *C. sordellii* and *C. novyi*, alone or in combination, although it is seldom possible to attach specific pathological significance to any individual bacteria.

Cases which are recognised early sometimes respond well to parenteral penicillin antibiotic treatment. Control is partly achieved through vaccination.

Tetanus

Tetanus is a fatal paralysing disease of all species caused by a neurotoxin, which is produced following multiplication and bacterial death of *Clostridium tetani*. *C. tetani* is ubiquitous in soil and causes disease when spores from the environment enter deep wounds with devitalised tissue. In sheep the disease is commonly associated with docking wounds, especially following the use of rubber rings in unhygienic conditions.

Following an incubation period of 1 to 3 weeks, being longer in adults than in lambs, the neurotoxin reaches the brain via peripheral nerves and the spinal cord and causes a state of sustained spasm and rigidity of voluntary muscles. Affected animals are unable to swallow or eructate. As the disease progresses animals become laterally recumbent with a characteristic ‘saw horse’ appearance due to extension of the neck, thoracic and pelvic limbs. The disease is extremely painful and humane destruction of advanced cases is recommended, before animals eventually die from asphyxiation due to paralysis of respiratory muscles.

There are no specific gross postmortem signs, so the diagnosis of tetanus depends largely on the interpretation of history and clinical signs.

Early cases can be successfully treated with tetanus antitoxin, antibiotics and anti-inflammatory drugs, although the prognosis is always guarded and treatment expensive. Outbreaks of tetanus in lambs are usually triggered by a specific event such as docking, with an incubation period of 1 to 3 weeks. Tetanus antitoxin can provide immediate protection of lambs for about 3 weeks, so in some cases in potentially valuable lambs administration of tetanus antitoxin in the face of an outbreak to known at-risk animals may be useful.

Prevention of tetanus is achieved by vaccination. Because most cases in sheep are associated with docking, it is important that ewes are vaccinated to ensure passive protection of their lambs.

Botulism

Botulism is a rare disease of all species, caused by ingestion of *Clostridium botulinum* toxin and characterised by flaccid paralysis and incoordination. Botulism in sheep has been reported in Australia following periods of drought when feed is short and sheep resort to eating vegetation soiled by rotting carrion. Fortunately these conditions do not occur in the UK. However, cases of botulism due to *C. botulinum* types C and D have been suspected in UK sheep grazing pastures to which poultry manure has been applied (Fig. 3.61).

C. botulinum is a spore-forming obligate anaerobe, which is saprophytic on animal and vegetable material and commonly found in soil. The organism multiplies rapidly in decomposing carcases and spores survive in soil for long periods of time. Soil, vegetation or carcases contaminated by spores of *C. botulinum* type B may become incorporated into conserved feeds such as big bale silage (Fig. 3.62) and, given suitable conditions, may use grass protein as a substrate for multiplication, followed by bacterial death and the release of large amounts of toxin.

Ingestion of the botulinum toxin by sheep causes stiffness, incoordination and excitability, which usually progresses to recumbency and death within 24 hours. Drooling of saliva due to flaccidity of the tongue and difficulty swallowing is sometimes noted. Generalised flaccid paralysis which characterises the disease in cattle is reported to be less evident in sheep.

Fig. 3.61 Poultry litter often contains carcases and a substrate for *C. botulinum* types C and D multiplication and toxin production.

Fig. 3.62 The risk of clostridial multiplication is highest in unstable baled silage with a dry matter content less than 15 percent.

There are no specific postmortem signs and diagnosis depends on the interpretation of history and non-specific clinical signs.

No vaccine against botulism is available in the UK. Prevention therefore depends on avoiding the main risk factors such as feeding of poor-quality big bale silage or grazing pastures to which poultry manure containing carcases has recently been spread.

Administration of clostridial vaccines

The recommended dose of clostridial vaccine should be administered subcutaneously over the neck. Clean needles should be used and regularly changed to reduce the risk of injection site infections (Fig. 3.63). Vaccines should be correctly stored in a dark place at about 5°C but protected from freezing. Vaccines should be used before their expiry date and, as a general rule, partially used packs should be discarded at the end of the day.

In recent years controversy has arisen surrounding the prevention of clostridial diseases on organic farms. The *Soil Association Standards for Organic Food and Farming* (September 1999) state that 'single, two in one or four in one vaccines are preferred to more complex vaccines unless such cover is specifically required' and 'vaccination is restricted to cases where there is a known disease risk on a farm or neighbouring land which cannot be controlled by any other means'. It should be strongly argued that there is a risk on every farm of lamb dysentery, pulpy kidney, blackleg, tetanus and malignant oedema, which can only be controlled using a multi-component vaccine. Withdrawal of such vaccines will inevitably result in unacceptable sheep losses.

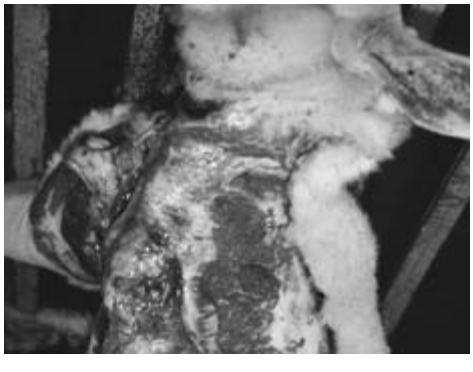


Fig. 3.63 An injection site reaction at an inappropriate site above and extending below the shoulder.

Chapter 4

Ill Thrift in Adult Sheep

In most UK sheep flocks, ill thrift and weight loss in adult sheep are important welfare and economic concerns. Poor body condition is often inapparent because of the fleece cover, and only identified when consequent problems, such as ewe deaths or low lambing percentages, are identified. Thin hill ewes often fail to survive in harsh conditions over winter. The potential consequences of ill thrift in ewes at mating time are low ovulation rates and high barren rates, resulting in low lambing percentages. Thin ewes at lambing time give birth to light-weight lambs, have poor colostrum production and have a high incidence of metabolic disease, resulting in high perinatal lamb mortality rates and management costs. Excessive weight loss after lambing results in poor milk production and a high incidence of ewe disease, leading to poor lamb growth rates. Management costs associated with culling or replacement of dead ewes and achieving adequate target body condition are high.

The body condition of full-fleeced ewes cannot be reliably judged from their visual appearance alone. In New Zealand, where most flocks comprise purebred Romney, or Romney crossbreed (Perendale and Coopworth) sheep, with clearly defined breed production targets, weighing of ewes at specific times of year and comparison of the average weights with target data is routinely performed. Ewes in commercial UK flocks are seldom weighed, because between-flock targets have not been established due to the confounding effects of variation between different breeds and their crosses, variation associated with age and stage in the production cycle, individual animal variation and the effect of rumen fill. However, these complications need not preclude the establishment of reference weights for ewes in individual flocks at different times of year.

Some of the problems associated with weighing ewes are overcome by body condition scoring, involving the palpation of fat over the lumbar vertebrae. While body condition scoring is useful for within-flock management, it is subjective and between-flock comparisons are not always accurate. Body condition is scored as follows:

1 Emaciated – no fat or muscle palpated between the skin and transverse processes.
2 Transverse processes are prominent and clearly defined.
3 The ends of the transverse processes can only be defined with firm pressure. Spinous processes are prominent and clearly defined.
4 The ends of the transverse processes cannot be felt. The spinous processes can only be defined using firm pressure.
5 Obese – neither transverse nor spinous processes can be palpated.

Feed requirements to compensate for below-target body condition scores have been determined experimentally. For example, 0.5 units of body condition score (on

Sheep Flock Health: A Planned Approach. Neil Sargison
 ISBN: 978-1-4051-6044-5

Fig. 4.1 The feed management costs associated with correcting the body condition scores of these thin ewes at mating time may exceed their benefits.

Table 4.1 Target body condition scores for lowground crossbred ewes (these scores should be met by every ewe in the flock).

	Lowland ewes	Hill ewes
Weaning	Ewes should be condition scored and managed to ensure that all reach target scores by mating	
Mating	3.0–3.5	2.5–3.0
Mid pregnancy	2.5–3.0	2.0–2.5
Lambing	2.5–3.0	2.0–2.5

a scale of 1 to 5 units) is equal to 5 to 6 percent of liveweight (4 to 5 kg for a 70 kg ewe), and in order to gain 0.5 units of body condition score a month, a 70 kg ewe requires about 20 MJ of metabolisable energy (ME) per day. Thus, while it is possible for ewes to gain 0.5 units of body condition score per month, for example to achieve adequate body condition for mating and achievement of an optimal lambing percentage, the feed management costs are potentially high (Fig. 4.1), demonstrating the economic benefits of adhering to target body condition scores throughout the year (Table 4.1). In order to ensure that these target body weight or condition scores for mating can be achieved, ewes should be checked about 6 weeks beforehand.

Investigation of ill thrift in adult sheep

Whenever thin adult sheep are identified in a flock, the options may be to cull them, or to manage the problem correctively, either through improved nutritional management or specific disease control. Accurate diagnosis of the underlying cause is therefore important.

As with all disease investigations, the diagnosis of the cause of ill thrift begins with a relevant disease history, determining the timescale of the problem, the number of animals affected, the stage of the production cycle when the problem is identified, flock management, disease control and previous diagnoses. It is important to determine whether or not the problem is significant within the context of the production system; for example, poor body condition may be normal, albeit undesirable, in ewes kept on harsh hill ground. It is then useful to examine the whole group, determining the cohorts and numbers of sheep affected. Whenever a large proportion of the flock

Fig. 4.2 Ill thrift involving a high proportion of older Scottish Blackface ewes, associated with various problems, in particular dental disease and chronic parasitism.

is affected, the common diagnoses of poor nutrition, general flock management problems and flock diseases such as lameness and sheep scab should be considered. Whenever specific cohorts are affected, then management problems or specific diseases such as dental diseases, parasitic diseases or maedi-visna should be considered (Fig. 4.2). Involvement only of small numbers of individual sheep might indicate problems such as suppurative mastitis, internal abscessation, endocarditis, Johne's disease, scrapie, jaagsiekte, or other tumours such as intestinal adenocarcinoma.

Having first conducted a full clinical examination on representative affected animals, it is usually helpful to collect appropriate samples, focused on the most likely causes of the problem. For example:

- Serum proteins – most causes of ill thrift
- Serum metabolites – malnutrition in pregnant ewes
- Faecal fluke eggs – chronic fascioliasis
- Faecal worm egg counts – parasitic gastroenteritis
- Serum liver enzymes – fascioliasis
- Skin scrape material – sheep scab
- Serum AGIDT – maedi-visna
- Serum AGIDT – clinical Johne's disease
- Ziehl-Neelsen stained faecal smears – clinical Johne's disease
- Anaerobic bacterial culture – footrot.

Serum albumin concentrations reflect the balance between hepatic protein synthesis, endogenous demands and losses through the intestinal tract, kidneys, blood or skin, while serum globulin concentrations are raised in response to chronic bacterial infections. (Serum concentrations of other proteins, such as haptoglobin and fibrinogen, are raised with acute disease, but are of lesser value in the investigation of ill thrift.) Thus, measurement of serum albumin and globulin concentrations provides a quick and inexpensive tool to reduce the differential diagnosis list for causes of ill thrift. For example, low serum albumin concentrations and raised serum globulin concentrations indicate chronic suppurative disease, while low serum albumin concentrations and normal serum globulin concentrations, in the absence of specific clinical signs, indicate malnutrition, non-suppurative hepatic disease or enteric disease.

In some situations it may be appropriate to humanely kill severely affected animals before conducting a postmortem examination, focused on the liver (fascioliasis), small intestine (Johne's disease and adenocarcinoma) and lungs (jaagsiekte and

maedi), and collecting appropriate carcase samples to be submitted for laboratory confirmation of the diagnosis. Postmortem examination is often justified due to the low economic value of thin ewes. However, the significance of incidental or unusual postmortem findings must be kept in perspective, and findings in these individual severely affected sheep may not represent the primary flock problem. Furthermore, ill thrift in adult sheep may be a consequence of several concurrent diseases.

Respiratory disease

Respiratory disease seldom presents as a flock problem in adult sheep, although individual cases are common. Affected animals present with clinical signs including tachypnoea, dyspnoea associated with exertion, nasal discharge, chronic weight loss and coughing, depending on the cause.

The common causes of respiratory disease in adult sheep are:

- chronic suppurative pneumonia
- pleuropneumonia/pleural abscessation
- jaagsiekte
- laryngeal chondritis.

The list of other causes which should be considered includes:

- lungworm
- pneumonic pasteurellosis
- maedi
- pulmonary or mediastinal abscessation associated with caseous lymphadenitis
- enzootic nasal tumour
- tuberculosis
- inhalation pneumonia associated with dipping, drenching, or choke
- *Oestrus ovis* infection.

In many cases, effective flock management depends on the accurate diagnosis of respiratory disease in individual sheep. This is based on the disease history, clinical signs, use of ancillary diagnostic tests such as biochemical or serological tests, real-time ultrasonography and postmortem findings. Auscultation of sheep lungs alone is often unrewarding. Tachypnoea may also be seen in the absence of specific respiratory disease, for example in cases of chronic copper poisoning.

Chronic suppurative pneumonia (lung abscessation)

Lung abscessation can result from: bacterial infection of already-compromised lung tissue; inhalation of bacteria from the oropharynx, typically associated with *Fusobacterium necrophorum* infection in young lambs; as a sequel to pneumonic pasteurellosis; or haematogenous spread from a septic focus elsewhere in the body, such as the udder or uterus. In lambs, tick pyaemia is a common cause of lung abscessation. Lung abscesses are common in adult sheep but are difficult to diagnose by clinical examination alone. *Arcanobacterium pyogenes* is the most common bacterium isolated from lung abscesses.

A wide spectrum of clinical signs is seen, depending on the number and size of the lesions. Sheep with significant chronic suppurative pneumonia lesions present with a history of weight loss. Affected sheep are often dull and depressed although appetite may appear normal. The rectal temperature is often within the normal range or slightly elevated. At rest, affected sheep are tachypnoeic compared to normal sheep in the group, and cough occasionally. There may be an occasional scant mucopurulent nasal discharge.

In many cases it can prove difficult to identify the lung as the major site of bacterial infection. Therefore the differential diagnosis list should include the common causes of weight loss in individual sheep. Clinical examination, haematology, serology and biochemistry are rarely helpful in the diagnosis of chronic suppurative pneumonia. Depending upon the site and extent of the lesions auscultation may reveal a localised absence of normal lung sounds and muffled heart sounds. Radiographic examination can prove useful, but is restricted by cost considerations, availability of equipment, and problems associated with the sheep's fleece.

Ultrasound examination provides a cheap and portable alternative to radiographic examination of the chest and readily identifies pleural lesions such as abscesses. Unlike the case with enzootic pneumonia, where lesions commonly involve the cranio-ventral lung field, abscesses associated with bacteraemic spread tend to be distributed to the caudo-dorsal lung field. When abscesses are well encapsulated, the capsule can be readily identified (Fig. 4.3). In some cases abscesses may extend to occupy the whole side of the chest.

The diagnosis of chronic suppurative pneumonia is usually confirmed by post-mortem examination. Abscesses may be identified in the lung itself (Fig. 4.4), or in the pleural space where they are walled off by fibrous pleural adhesions (Fig. 4.5).

Clinical recovery has been achieved in valuable breeding stock following ultrasound-guided abscess drainage and treatment with an antibiotic for 30 consecutive days. Penicillin is the antibiotic of choice because of the frequent isolation of *A. pyogenes*. However, in most cases this strategy is neither practical nor economic, so sheep with significant lung pathology should be humanely killed for welfare reasons.

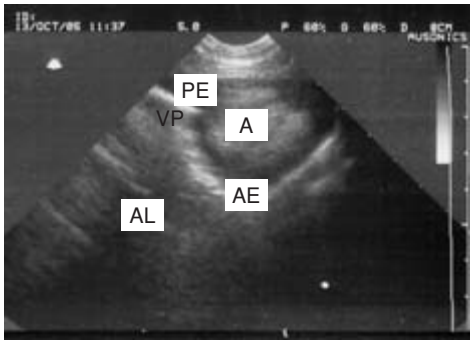


Fig. 4.3 Ultrasound image of a pleural abscess, using a 5 MHz sector scanner. PE pulmonary effusion; VP visceral pleura; AL normally aerated lung; A abscess; AE acoustic enhancement.

Fig. 4.4 Multiple encapsulated abscesses within the dorsal lung tissue of a thin Scottish Blackface ewe.

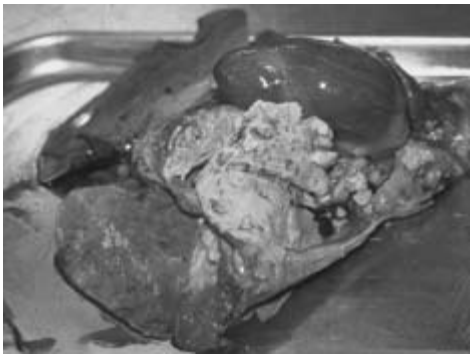


Fig. 4.5 Extensive fibrinous adhesions associated with a large pleural abscess.

Prevention is aimed at prompt treatment of bacterial infections such as mastitis, metritis and cellulitis, before significant bacteraemic spread to the lungs occurs. *Fusobacterium necrophorum* infection of the lungs is not uncommon in orphan lambs reared in unhygienic conditions.

Jaagsiekte (ovine pulmonary adenocarcinoma)

Jaagsiekte is a contagious lung tumour, associated with slow retroviral (jaagsiekte retrovirus) infection. The disease is particularly prevalent in Greyface, Scottish Halfbred and Mule ewe flocks in the Scottish Borders, but has been reported throughout the UK in most breeds. In some flocks, the disease may be responsible for 50 percent of adult sheep losses, although the main economic significance results from the early culling of ill-thrifty sheep.

Clinical signs

The early signs of jaagsiekte are loss of body condition and exercise intolerance when gathered. The incubation in naturally infected sheep is long, with clinical disease usually seen in 3- to 4-year-old animals, although the disease is exceptionally seen in older sheep, or in 8- to 12-month-old lambs, which are generally the progeny of infected dams. Affected sheep are afebrile and maintain normal appetite throughout the course of the disease, unless there is significant secondary bacterial pneumonia. A soft cough is often audible.

As the disease progresses, sheep become increasingly tachypnoeic, especially during hot weather. Respiration is exaggerated by contraction of the abdominal muscles and bouts of mouth breathing. Fluid gathers in the airways, first appearing as a scant serous nasal discharge which may dribble from the nostrils when the head is lowered. Affected sheep sometimes have an anxious expression and stand with their neck extended and head held lowered. Affected lung tissue usually becomes secondarily infected with *Mannheimia haemolytica*, exacerbating the clinical signs and often resulting in death.

The history and non-specific clinical signs of weight loss, respiratory distress and fluid dribbling from the nostrils when the head is lowered, which characterise advanced cases, are indicative of jaagsiekte (Fig. 4.6). However, the clinical signs in the earlier stages of the disease are not specific and there are currently no serological tests to support a diagnosis in the live animal.

Fig. 4.6 Serous nasal discharge running from both nostrils of a Greyface ewe, characterising advanced signs of jaagsiekte.

Increased crackles are heard over a wide area of the chest during auscultation, especially involving the ventral portions of the apical and cardiac lobes. Raising of the sheep's hindquarters while lowering the head ('wheelbarrow' test) often exaggerates the flow of up to 50 ml of clear frothy fluid from the nostrils and can be a useful ancillary diagnostic test. This test causes affected sheep considerable distress and accelerates the subsequent progress of the disease. Euthanasia should, therefore, be undertaken once the positive diagnostic result is obtained.

The ultrasonographic appearance of advanced cases of jaagsiekte is distinctive: the normal aerated lung surface (visceral pleura) reflects sound waves and appears as a hyperechoic (bright white) line; by contrast, areas of cellular proliferation within the lung parenchyma transmit sound waves and are represented by a hypoechoic (darker) area with bright dots where the sound waves hit air within bronchioles and smaller airways. Typically, ultrasonography of jaagsiekte cases reveals extensive, ventral areas of cellular proliferation which are sharply demarcated from adjacent normal lung tissue.

Diagnosis

The diagnosis of jaagsiekte is confirmed by postmortem findings. Tumours are usually confined to the lungs and associated lymph nodes, which are enlarged and heavy (>1 kg; normal 0.4 to 0.6 kg). Large tumours have a characteristic solid and grey appearance and are well demarcated from less affected lung tissue (Fig. 4.7). These lesions may contain abscesses or necrotic centres (Fig. 4.8). The airways contain copious frothy fluid. There is often evidence of secondary bacterial infection, such as abscesses or pleurisy, which is sometimes misdiagnosed as the primary cause of disease. The presence of tumour cells can be confirmed by microscopic examination of stained sections of affected lung tissue.

Management

Antibiotic treatment sometimes results in temporary improvement of clinical signs, associated with remission of secondary bacterial infection, but the disease is invariably fatal. Affected sheep should, therefore, be culled as soon as the disease is diagnosed, to prevent further suffering and reduce the spread of infection within the flock.

Fig. 4.7 A clearly demarcated large tumour involving the ventral lung tissue on both sides.

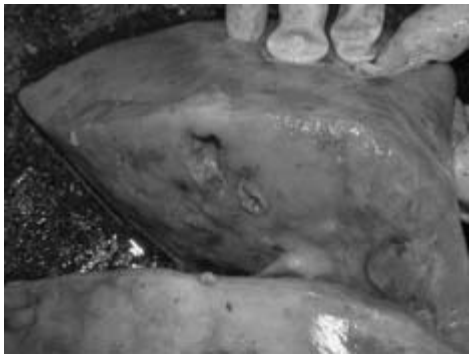


Fig. 4.8 Abscess formation at the centre of a jaagsiekte tumour lesion and oozing of frothy fluid from the airways.

Jaagsiekte is introduced into flocks with purchased sheep, which probably shed the jaagsiekte retrovirus before tumours develop. The main route of infection is likely to be respiratory, with the close confinement of housing or trough feeding increasing the rate of spread of infection. The prevalence of jaagsiekte is highest in those countries where sheep are housed for long periods during the winter months.

There is no recognised effective control method, but regular inspection and prompt culling of lean or dyspnoeic sheep may slow the spread of infection. Keeping mostly naïve younger sheep separate from older infected animals can also help to reduce the prevalence of the disease. The offspring of affected sheep frequently develop jaagsiekte, so should not be kept as replacement breeding stock.

A synergistic effect involving maedi-visna virus and jaagsiekte has been noted, but not proven, so control measures should involve excluding entry of both virus conditions from the flock. De-stocking is the only practical solution when both infections exist in a flock.

Maedi (ovine progressive pneumonia)

Maedi describes a chronic respiratory disease caused by a small ruminant lentivirus infection, which also causes neurological disease (visna), indurative mastitis and arthritis. The main route of transmission of the maedi-visna virus is from mother to offspring in colostrum and milk, although it is likely that infection is also acquired by other routes. Caprine arthritis and encephalitis (CAE) is caused by a similar virus, and cross-infection is possible, when colostrum from goats is fed to sheep and vice versa. The virus somehow escapes the host's immune response and establishes in the lungs, udder, nervous tissue and haemopoietic organs.

Maedi-visna virus infection is recognised in many countries worldwide, with the exception of Australia and New Zealand. The disease once had a particularly high prevalence in Iceland, associated with a prolonged winter housing period, but was successfully eradicated in 1965, following widespread de-stocking and repopulation from disease-free flocks.

In most countries, the main economic effect of maedi is poor production as a result of indurative mastitis and poor body condition, poor reproductive efficiency, high

perinatal mortality and poor lamb growth rates. Maedi-visna was first recognised in the UK in 1979, when seropositive animals were detected in a group of rams intended for export. When the remainder of the flock was tested, 60 percent were found to be seropositive, despite no evidence of clinical disease in the flock. Maedi lesions were found in the lungs of one ewe (0.5 percent of the flock) at postmortem. However the flock had a history of a high culling rate, wastage being attributed to pasteurellosis and jaagsiekte. Maedi has subsequently been diagnosed in several other flocks with no history of contact with imported sheep, and with a similar disease pattern.

During the 1980s it was predicted that the incidence and economic importance of maedi would increase dramatically in UK flocks, associated with failure to identify the presence of the disease in infected flocks until more than 60 percent are seropositive and movements of clinically normal infected sheep. Fortunately this situation has not yet arisen in commercial flocks, although the disease has become problematic for pedigree sheep, possibly related to more intensive husbandry and a longer productive lifespan of ewes in these flocks.

Clinical signs

Despite infection as lambs, clinical disease is seldom seen sheep under 3 years old. Most infected sheep outlive their productive lifespan before overt clinical disease develops. The earliest sign of maedi is exercise intolerance, which is first seen when sheep are gathered. Affected sheep have an extended neck, increased respiratory rate, flared nostrils and an abdominal component to the breathing effort. As the disease progresses, wasting occurs and dyspnoea becomes obvious even at rest (Fig. 4.9). Any exertion results in mouth breathing and tachypnoea, and if severely stressed, cyanosis and collapse may ensue. Sheep remain bright and continue to eat, despite severe dyspnoea and weight loss. Signs of coughing, naso-ocular discharge, lymphadenitis and pyrexia are absent and auscultation is generally unhelpful. Although not a major presenting sign, affected sheep frequently also have an indurative mastitis, identified as a flabby udder with diffuse hardening. Milk production is significantly decreased, although the milk appears normal. Associated lymph nodes are enlarged.

In the USA, maedi-visna virus infection causes arthritis, manifested by a stiff, straight-legged gait, with swelling most commonly of the radial carpal joints. Histologically, there is lymphocytic proliferation over the synovial membranes.

Fig. 4.9 Severe respiratory effort at rest, with an extended neck and obvious abdominal component.

Maedi-visna arthritis has not been confirmed in UK sheep, but is the predominant feature of the equivalent disease, caprine arthritis-encephalitis, in goats.

Diagnosis

The diagnosis of maedi in live animals relies on the detection of antibodies to the maedi-visna virus. Unfortunately seroconversion can take several months and some animals never develop antibodies. The standard test in the UK is an agar gel immunodiffusion test (AGIDT), which is employed as the basis of the Sheep and Goat Health Scheme for maedi-visna. However, this test is gradually being replaced by more sensitive ELISA methods.

On postmortem examination, the lungs are firm and rubbery, and markedly increased in weight (>2 kg). The lungs do not collapse when the chest is opened and impressions of the ribs are sometimes seen on the pleural surface. The lungs may exhibit mottled or grey areas. Histologically, there is smooth muscle hyperplasia, with diffuse interstitial pneumonia and lymphoid infiltration and proliferation of the alveolar septae. Lesions are distributed evenly throughout the lung tissue. The caudal mediastinal lymph node is usually greatly enlarged, with marked cortical hyperplasia, diffuse lymphoid infiltration and fibrosis.

The postmortem diagnosis of maedi is often complicated by concurrent pneumonic pasteurellosis or jaagsiekte. Histological diagnosis may be no more reliable, because smooth muscle hypertrophy may occur with lungworm infection and lymphoid infiltration with mycoplasma and chlamydial infections.

Management

There is no effective treatment for maedi. Vaccination is unlikely ever to be a control option due to the virus's ability to escape the host's immune response. Prevention of infection through adequate biosecurity and the purchase of accredited maedi-visna-free stock is the best option at present in the UK.

A strict culling policy and increased replacement rate can aid control in endemically infected flocks. Control can be attempted in an infected closed flock by regular (3 to 6 monthly) serological testing, culling of seropositive sheep and removal of their offspring, but this is costly and protracted. Alternatively, control can be attempted by the removal of lambs from their dams immediately after birth before they ingest infected colostrum, thus breaking the lactogenic route of transmission. This method is not practical on most commercial farms, due to the cost and husbandry required to rear lambs on colostrum substitutes and milk replacers.

In the UK, accreditation of freedom from maedi-visna can be gained through membership of the Sheep and Goat Health Scheme. Flocks are accredited on the basis of negative AGIDT results in two qualifying flock blood tests between 6 and 12 months apart, involving all sheep (and goats) over 12 months old. A declaration of adherence to strict flock biosecurity is required, involving secure farm boundaries, avoidance of contact with non-accredited animals, and introduction of sheep, semen or embryos only from fully accredited flocks. All sheep are required to be permanently identified and accurate records of all sheep movements must be kept. Subsequent blood sampling, with negative AGIDT results, is required at intervals not exceeding 2 years. These periodic tests involve all rams, added animals and a

proportion of ewes over 18 months old, to provide 95 percent confidence of detecting the disease if it is present at a seroprevalence of more than 2 percent.

Pneumonic pasteurellosis

The main role of *Mannheimia haemolytica* in adult sheep is secondary to other respiratory diseases. Pneumonic pasteurellosis is primarily a disease of lambs, although sporadic cases are reported in adult sheep, characterised by dullness, anorexia, pyrexia and dyspnoea. Serous oculo-nasal discharge may be present, with terminal frothy salivation due to respiratory distress.

On postmortem examination, there may be extensive ecchymotic haemorrhages in the throat and ribs. There are pleural and pericardial exudates, and the lungs are swollen and cyanotic, with dark red/purple consolidated patches. The airways contain pink froth. Less acute cases show grey/red raised consolidation of the anteroventral lung lobes, with green gelatinous pleural exudate. This consolidation may resemble liver in appearance and consistency.

Pneumonic pasteurellosis in ewes is controlled by vaccination and avoidance of stressful husbandry.

Laryngeal chondritis

It is not uncommon for sheep to be seen with signs of upper respiratory disease, characterised by loud open-mouthed breathing (Fig. 4.10). The disease is caused by abscessation of the aretenoid cartilages within the larynx and is referred to as laryngeal chondritis.

Laryngeal chondritis is seen in most breeds of sheep, although a particularly high prevalence is seen in Texel, Beltex and Southdown breeds. The incidence of the disease is greater in rams than in ewes. Affected sheep have progressive, severe inspiratory dyspnoea, loud open-mouthed breathing with the head and neck extended and cyanosis of mucous membranes of the mouth and eyes. External palpation of the larynx is resented and can exacerbate the clinical signs. Unlike the situation with acute pasteurellosis, rectal temperatures are usually normal. In many cases, affected animals die within a few days of the onset of clinical signs. Recovered animals often have chronic roaring breathing, which flares up when they are stressed. Death from diaphragmatic rupture, intrathoracic haemorrhage and lung collapse has been reported, caused by increased respiratory effort due to laryngeal chondritis.

Fig. 4.10 An extended neck, mouth breathing and cyanosis, characterising laryngeal chondritis in a stressed Beltex shearling sale ram.

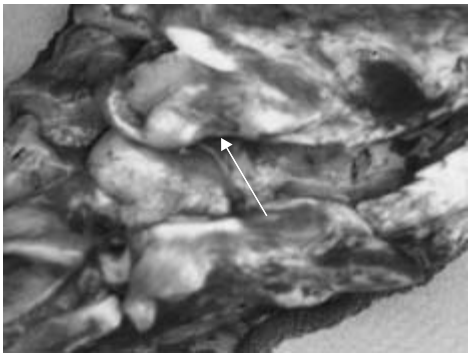


Fig. 4.11 Abscessation of an aretenoid cartillage (*arrow*).

Laryngeal chondritis is associated with the presence of encapsulated 0.5 to 5 mm diameter abscesses deep within the aretenoid cartillages of the larynx (Fig. 4.11), sometimes with sinus tracts and granulation of the laryngeal mucosa. Bacterial culture of abscesses usually yields non-specific environmental pathogens, such as *Escherichia coli*, *F. necrophorum*, and *A. pyogenes*.

The predisposing causes of laryngeal chondritis have not been determined. Grass awns, dusty feed, trauma associated with rough handling or faulty drenching technique, and a hereditary or congenital predisposition have all been suggested, but not proven. The high incidence of the laryngeal chondritis in Texel and Southdown sheep is probably related to the anatomy of their short necks.

A reasonable response to corticosteroid and high-dose broad-spectrum antibiotic therapy is seen when cases are recognised and treated promptly. However the treatment response in established cases is poor. Tracheotomy intubation has been advocated for individual valuable animals, although the long-term benefits are doubtful.

Tuberculosis

While sheep are susceptible to *Mycobacterium bovis* infection, causing encapsulated, yellow-grey, caseated, tuberculous nodules in the lungs, retropharyngeal and mediastinal lymph nodes, tuberculosis is seldom confirmed, even when they are co-grazed with infected cattle herds. This may simply reflect the facts that sheep are less inquisitive than cattle and tend not to graze at night, so are less likely to come into close contact with possible reservoir hosts.

Outbreaks of tuberculosis have rarely been reported in New Zealand flocks, having first been identified during meat inspection. In one case involving a flock of 600 ewes, an intradermal tuberculin test gave 108 reactors, 43 of which were subsequently shown to have tuberculous lesions.

Sheep are also susceptible to *Mycobacterium avium*, rarely causing non-progressive, clinically irrelevant, fibrotic lesions in lymph nodes.

Enzootic nasal tumour (infectious nasal adenocarcinoma)

Enzootic nasal tumour is a contagious upper respiratory disease caused by a retrovirus referred to as enzootic nasal tumour virus (ENTV). Enzootic nasal tumour is an important sporadic cause of ill thrift in 2- to 4-year-old sheep in infected flocks in

southern and eastern Europe, but while there are occasional anecdotal reports of the disease in individual UK sheep, these diagnoses have not been confirmed.

Affected sheep are ill thrifty, with clinical signs including stertorous breathing, coughing and dyspnoea. Continuous flow of a seromucous secretion occurs from one or both nostrils, depending on the site of the tumour. In advanced cases, the skull overlying the tumour may be deviated or deformed.

Tumours are low-grade, cauliflower-like adenocarcinomas, which arise unilaterally or bilaterally from the mucosal secretory glands of the ethmoid turbinates. The tumours do not commonly metastasise. The epidemiology of the disease and principles involved with its management are similar to those for jaagsiekte.

In endemically affected areas, the diagnosis of enzootic nasal tumour is based on the clinical signs, and confirmed by histopathology. A polymerase chain reaction (PCR) test has been developed to detect the presence of ENTV in blood, which could prove useful in the future for disease management.

Alimentary disease

The early clinical diagnosis of alimentary tract disorders in individual sheep is not straightforward and most conditions tend to be chronic and/or severe before they are identified. The important alimentary tract diseases which can result in ill thrift in adult sheep are:

- dental diseases and abnormalities
- Johne's disease
- parasitic gastroenteritis
- fascioliasis.

Occasionally, other diseases such as pharyngeal trauma, choke, abomasal impaction, traumatic reticulitis and ruminal or abomasal bloat may result in ill thrift in individual animals.

Dental diseases and abnormalities

Dental problems are the main cause of premature culling of sheep. For example, UK hill ewes are traditionally drafted off the hill and moved to lowground pastures after only three or four crops of lambs largely because of dental disease. Dental disease is universally acknowledged as an important source of economic loss to the UK sheep industry, due to high flock replacement costs and because it necessitates supplementary feeding. Ewes with dental disease command lower prices than similar ewes with full mouths. Dental disease precludes the feeding of root crops to lambs or pregnant ewes.

There are numerous documented causes of poor dentition, including incisor tooth loss, excessive incisor wear, mandibular deformities, caries, fluorosis and malocclusion. However, those of greatest economic importance to the sheep industry as a whole are periodontal disease and excessive incisor tooth wear. Several causes have been proposed for both syndromes. In fact it may be misleading to separate the syndromes totally, as several dental problems are frequently observed in the same flock. There have been few objective studies to determine the risk factors involved

for these major causes of tooth disease in the UK. Little practical advice can, therefore, be offered for the management or prevention of sheep dental diseases.

Examination of the incisor teeth and gums is straightforward by retracting the lower lip. The medial to lateral temporary incisor teeth erupt at weekly intervals from birth, while the three temporary premolar teeth erupt within 2 to 6 weeks. Adult sheep have 32 permanent teeth, comprising four lower incisors (strictly three incisors and one canine), three lower premolars and three lower molars, and three upper premolars and three upper molars on each side. The medial to lateral permanent incisor teeth erupt at about 14, 20, 26 and 33 months old. The lower permanent molars erupt at about 3, 9 and 18 months old, while the upper permanent molars erupt at about 5, 12 and 24 months old.

Broken mouth

The term 'broken mouth' is used loosely to describe a number of syndromes which result in tooth crown loss, but is more correctly applied to periodontal disease.

Periodontal disease is characterised by inflammation of the gum margins of the teeth, formation of pus-filled pockets, and food impaction around the teeth. Localised gingivitis and plaque formation starts at an early age and progresses as teeth erupt, resulting in recession of the gingival margin (Fig. 4.12) and weakening of the periodontal ligament. A generalised periodontitis then develops, with the breakdown of collagen from within the periodontal ligament (Fig. 4.13). The end result is a lack of mechanical support to the incisor teeth due to a combination of long crowns and short root supports (Fig. 4.14). Premature incisor loss occurs from as young as 3 years old, resulting in inefficient feed utilisation (Fig. 4.15). When pasture is close grazed, ill thrift occurs. Abattoir surveys have confirmed that broken mouth is an important cause of premature culling, but provide few clues to the cause of the disease. Some flocks appear to be badly affected, while others remain relatively unaffected. No breed differences have been reported. There is anecdotal evidence that the problem is worse following pasture improvement by liming and re-seeding.

The possible causes of periodontal disease include:

- The initial localised gingivitis and plaque formation would suggest the involvement of pathogenic bacteria.

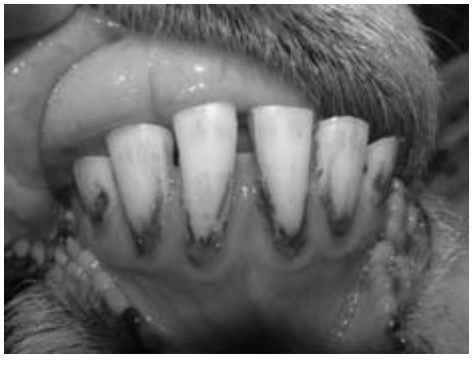


Fig. 4.12 Plaque formation and recession of the gingival tooth margins.

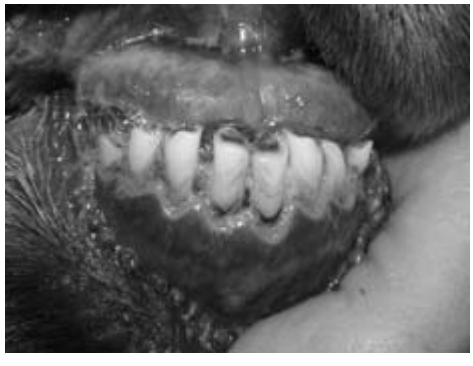


Fig. 4.13 Severe generalised periodontitis.

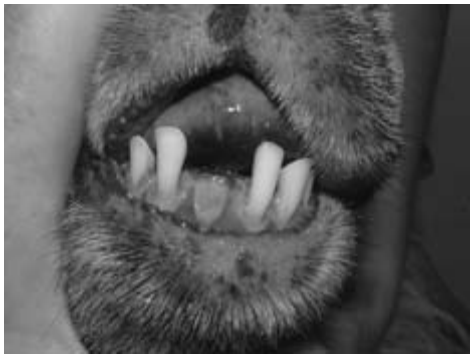


Fig. 4.14 Long crowns and short root supports leading to lack of mechanical support and premature loss of the incisor teeth.

Fig. 4.15 Premature loss of most of the incisor teeth, leading to inefficient grazing behaviour and ill thrift.

- Feeding of root crops may enhance the rate of development of periodontal disease through abrasion and their high carbohydrate content.
- Faulty occlusion of the incisor teeth with the dental pad may have a mechanical destabilising effect on the periodontal ligament.
- Poor protein nutrition may compromise the immune response to periodontal disease.

Extensive studies have failed to establish any causative link with calcium and phosphorus imbalance or trace element deficiency. Unfortunately the cause of periodontal disease remains unclear, which has resulted in the application of unproved, inappropriate and ineffective control methods, such as the use of tooth splints or tooth grinding using an angle grinder on overgrown incisor teeth, which were practised during the 1980s, but are now illegal in the UK.

Incisor tooth wear

Excessive wear of both temporary and permanent incisor teeth to gum level is a common cause of dental disease, sometimes associated with abnormal eruption of the permanent incisors (Fig. 4.16). Reports of the problem in the UK are widespread, although its incidence may have been underestimated because of confusion with periodontal disease.

Fig. 4.16 Concurrent excessive incisor tooth wear, periodontal disease and abnormal tooth eruption.

The precise cause of excessive tooth wear is complex and has defied clarification. The appearance of dental disease has been loosely associated with pasture improvement through fertiliser application, the introduction of modern pasture species and close intensive grazing. However, various New Zealand and Australian studies have identified the following risk factors:

- Soil ingestion results in abrasion during prehension and is generally accepted as the final insult required to cause tooth wear, although other factors probably predispose to such wear occurring. In one New Zealand study, approximately 70 percent of the wear occurred during those months when the soil content of faeces exceeded 40 percent; the provision of supplementary feed, which reduced soil intake, also reduced incisor tooth wear. Factors such as soil type, stocking rate, grazing management and pasture composition all influence the amount of soil ingestion.
- The eruption of permanent incisors with enamel defects can result from various factors, such as extreme nutritional (calcium or copper) imbalance, parasitism and trauma. Parasitism interferes with the absorption of protein and minerals, in particular calcium and phosphorus. However, excessive tooth wear also occurs in apparently healthy teeth.
- Teeth do not simply shorten from the top (Fig. 4.17) but are also dissolved from the sides (Fig. 4.18), so that the incisors are eventually reduced to 'pebbles'. Chemical attack by pasture juices or soil acids may dissolve apatite in teeth.
- There is anecdotal evidence of a breed effect on tooth wear. However, this may be related to feeding behaviour and grazing management rather than to genetic differences in tooth structure.
- Heavy ewes with more lambs eat more than light, poor-producing animals, which may influence the rate of tooth wear.

While it is not possible to provide specific advice about the prevention of excessive incisor tooth wear, providing adequate supplementary feed to reduce soil ingestion during wet weather conditions when pasture is short, and ensuring good gastrointestinal parasite control, may be helpful.

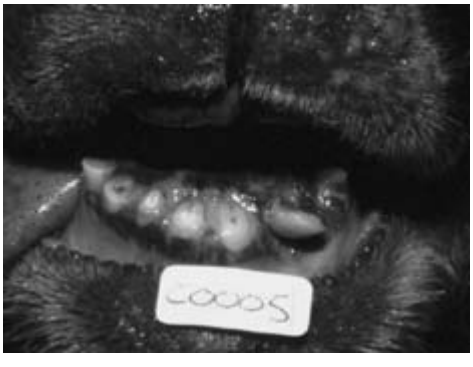


Fig. 4.17 Obvious incisor tooth wear, characterised by shortening of the teeth from their occlusal edge.

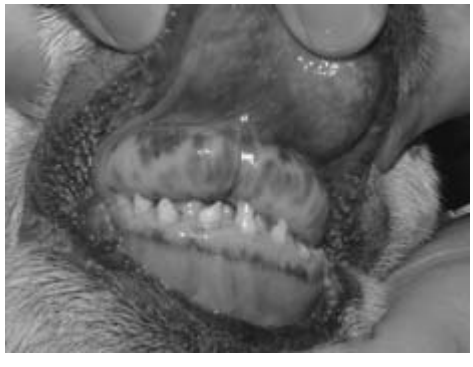


Fig. 4.18 Dissolving of incisor teeth, leading to formation of notches, which subsequently break, leaving only rounded stumps.

Caries and tooth wear in lambs fed on root crops

There is a well-documented association between the feeding of diets high in soluble carbohydrates and the formation of deep caries in the enamel at the base of the temporary incisor teeth of hoggs. Crowns weakened by caries commonly snap at gum level, leaving a rough-edged stump which can be confused with excessive tooth wear. However, this doesn't seem to affect the subsequent health of the permanent teeth. There are few references to caries in adult sheep.

Malocclusion of the incisor teeth

Normal, healthy incisor teeth are aligned with the dental pad. The alignment of the incisor teeth changes at different stages of lamb growth and development. These changes are probably exaggerated in concentrate-fed lambs, so the clinical relevance of slight malocclusion in terminal sire ram lambs is probably overemphasised by some UK sheep breeders.

Severe malocclusion is associated with abnormalities in the proportions of the mandible, maxilla and premaxilla. Projection of the incisor teeth well in front of the dental pad is referred to as overshot jaw or prognathia (Fig. 4.19), while alignment behind the dental pad is referred to as undershot jaw or brachygnathia. Impaired feeding and consequent ill thrift are only likely when discrepancies between the incisor teeth and the dental pad are more than about 5 mm. Severe brachygnathia is believed to be an inherited condition, while high prevalences of prognathia have been reported in intensively grain-fed lambs, putatively associated with calcium deficiency.

Dentigerous cysts

Obvious, smooth, 5 to 6 cm diameter, hard swellings are sporadically seen on the anterior part of the horizontal ramus of the mandible of individual 1- to 3-year-old sheep (Fig. 4.20). Some incisor teeth may be missing, while those that are present are often aligned horizontally, or at unusual angles to each other (Fig. 4.21). The

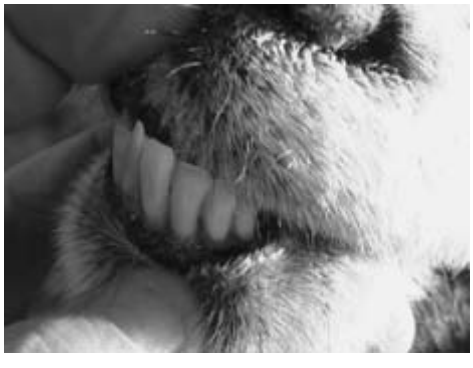


Fig. 4.19 Projection of the incisor teeth in front of the dental pad.

Fig. 4.20 Firm swelling involving both sides of the anterior mandible of a Scottish Halfbred gimmer, indicating the presence of dentigerous cysts.

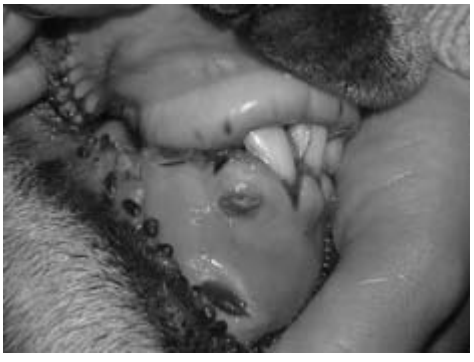


Fig. 4.21 Horizontal eruption of a lateral permanent incisor through the gum, incorrectly aligned central permanent incisor teeth and retention of a temporary incisor tooth.

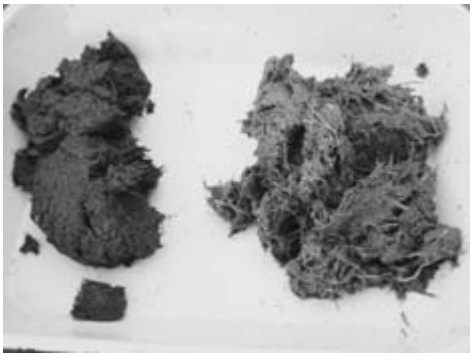


Fig. 4.22 Forestomach contents of a healthy adult sheep, showing normal short fibre-length digesta (*left*) and of a sheep with molar tooth problems, showing poorly masticated long fibre-length digesta (*right*).

fluid-filled, bony swellings, referred to as dentigerous cysts, are closely associated with the supports of unerupted permanent incisor teeth. They are not painful and neither discharging sinuses, nor submandibular lymph node enlargement, which might indicate an infectious cause, are seen. Affected sheep are ill thrifty whenever herbage is short.

Dentigerous cysts arise from odontogenic epithelial cells associated with tooth formation. Their cause is unknown, but most cases have been reported in flocks where root crops are routinely fed and excessive tooth wear has been identified.

The diagnosis of dentigerous cysts is straightforward, based on clinical examination. The diagnosis can be confirmed by identification of characteristic radiological changes, but this is usually unnecessary. While body condition usually improves with supplementary feeding, there is no effective treatment and affected sheep should be culled.

Cheek tooth problems

While incisor tooth problems are a major cause of premature culling in many sheep flocks, premolar and molar tooth problems are of greater clinical significance as a cause of severe weight loss in individual, older sheep. Affected sheep have difficulty in prehending and masticating feed (Fig. 4.22), using jerky jaw movements, with their lower jaw drooping slightly. Attempts to swallow are often accompanied by extension of the neck to assist movement of food over the ridge of the tongue. Food becomes impacted between the teeth and the cheeks, resulting in obvious swelling (Fig. 4.23), and wads of partially masticated, fibrous material are periodically dropped from the mouth. When affected sheep are fed hay, chunks of feed are often seen protruding from the commissures of the lips.

Thorough clinical examination of the molar teeth is problematic. Premolar and molar teeth have sharp enamel ridges and jaw movements are very powerful, so fingers must not be placed near to, or between the cheek teeth. Major abnormalities, such as lost or overgrown teeth, can be appreciated by palpation through the cheeks. A wooden gag can be wedged between the cheek teeth on one side to facilitate examination of the other side using a torch, although this procedure is resented and

Fig. 4.23 Swelling over the cheek and inability to chew long fibre associated with severe cheek tooth disease.

Fig. 4.24 Postmortem appearance of molar tooth disease, showing uneven eruption and impaction of fibrous feed material into pockets around the gingival margins.

not always rewarding due to the presence of impacted fibrous material between the teeth and gums.

Most problems are a consequence of periodontal disease and loss of tooth support structure, resulting in loss of shallow-rooted first or second premolar teeth. Following the loss of a premolar tooth, the opposing tooth erupts beyond the level of its neighbours, resulting in an uneven and inefficient grinding surface (Fig. 4.24). Halitosis and submandibular lymph node enlargement indicating deeper infection of the mandible are seldom seen. Malocclusion of the molar teeth associated with jaw abnormalities is also seen in some breeds.

Supplementary feeding to improve body condition is seldom economic and there is no treatment. Affected sheep should therefore be promptly culled.

Other causes of discrete mandibular swellings

Fibrosarcomas occur rarely in individual sheep, resulting in smooth, firm, 3 to 10 cm diameter mandibular swellings. Tooth root abscesses with associated osteomyelitis and sinus formation are uncommon. Healed fractures, and cellulitis associated with bite wounds, are diagnosed occasionally in individual animals.

Pharyngeal trauma

Pharyngeal trauma is sometimes seen following careless use of drenching guns or bolus application. While usually confined to individual growing lambs, significant flock problems have been reported in sheep of all ages following the use of poorly maintained drenching guns, or following unskilled, hurried administration of sharp-edged trace element boluses. Pharyngeal trauma is usually only identified several days after dosing or bolus application, when affected sheep may be found dead, or showing signs of hypoxia and stertorous breathing due to compression of the larynx resulting from extensive cellulitis and abscessation (Fig. 4.25). Infection occasionally extends into the cervical vertebral canal resulting in tetraparesis. Treatment is generally ineffective and euthanasia should be considered to avoid further suffering.

Fig. 4.25 Neck extension, dysphagia and obvious discomfort associated with pharyngeal trauma.

Pharyngeal trauma occasionally occurs following the administration of intraruminal electronic identification devices to comply with the UK National Scrapie Plan. In these cases, where the sheep are closely observed after bolusing and the progress of the bolus can be monitored using a handheld electronic microchip reader, the problem is usually identified promptly. Boluses can become lodged in the pharynx, or in the oesophagus at the thoracic inlet or as it passes over the heart. In younger lambs, boluses may be forced through the dorsal pharynx and lodged in the retropharyngeal region. Attempts to dislodge the bolus by forceful use of a stomach tube are seldom successful and often compound the problem. The correct positioning of the sheep for bolus application is important. Successful treatment has been reported involving general anaesthesia, radiographic or endoscopic examination and endoscopic or surgical recovery of the bolus from the pharynx or neck respectively.

Choke

Impaction of the proximal oesophagus occurs commonly in individual ewes while greedily feeding on dried sugar beet pulp. Affected sheep usually step backwards from the trough, while repeatedly retching and head tossing. Most cases relieve themselves after about 5 minutes, but occasionally signs of distress persist. In these cases, distension of the cervical oesophagus can be detected by palpation of the neck. Any temptation to relieve the choke by forceful use of a stomach tube should be avoided, because of the likelihood of perforating the oesophagus. Most cases eventually resolve if left alone, although the administration of spasmolytic or smooth muscle relaxant drugs may be helpful.

Diseases affecting the forestomach

Substantial differences occur in the extent of rumen fill and in the pattern of ruminoreticulum contractions between sheep kept under different management systems. Sheep fed on fibrous diets generally have distended forestomachs and one to two ruminoreticulum contractions per minute, while sheep fed on concentrate rations have small forestomachs and infrequent ruminoreticulum contractions. Inadequate mastication due to cheek tooth problems can also lead to ruminal distension. Poor rumen fill and irregular or absent ruminoreticulum contractions also occur in association with generalised disease.

Faecal consistency gives some indication of forestomach, abomasal and intestinal disease. Sheep normally produce pelleted faeces, or if they are fed lush herbage, soft faeces. Diarrhoea, accompanied by faecal staining of the tail and perineum (dags), indicates abnormal digestive function.

Ruminoreticulum function can be gauged by examination of ruminal liquor collected by aspiration through a stomach tube. (The stomach tube is passed through a cylindrical gag, placed between the incisor teeth and dental pad, made from a smooth-edged piece of firm plastic tube such as a section of 20 ml syringe.) Normal rumen fluid is green with a pH between 6.5 and 7.5 and microscopic examination at 100× magnification reveals numerous, variable-sized motile protozoa.

Primary disease of the ruminoreticulum is unusual in sheep. It may occur with oxalate poisoning, with bluetongue, or with grain overload. Gaseous ruminal distension also occurs in ewes with hypocalcaemia and with some clostridial diseases. Ruminal impaction associated with long-term water deprivation, and traumatic reticulitis associated with ingestion of sharp objects, have occasionally been reported in individual ewes.

Frothy bloat, causing marked distension of the left sublumbar fossa, which is sometimes masked by the presence of the fleece, occurs occasionally when sheep are moved onto very lush leguminous pastures. Affected sheep can be treated by dosing with froth-reducing agents such as poloxalene, simethicone or vegetable oil. The disease is prevented by introducing sheep slowly to lush pastures, for example for only 1 or 2 hours per day until they become accustomised.

Abomasal emptying defect

Abomasal emptying defect refers to a syndrome that is occasionally reported in individual adult Suffolk sheep, characterised by gradual weight loss and enlargement of the lower right side of the abdomen, corresponding to distension of the abomasum. Affected sheep are dull and have a poor appetite. The disease progresses over a period of several months, until affected sheep become emaciated. Faeces are dry and mucus-coated, indicating an increased transport time through the gastrointestinal tract. The abdomen is distended and firm, with no fluid thrill, distinguishing the syndrome clinically from small intestinal adenocarcinoma and peritonitis, which are usually associated with ascites.

The involvement of the abomasum can be confirmed by demonstration of rumen liquor chloride concentrations in excess of 30 mmol/L (normal values <15 mmol/L). Rumen chloride concentrations provide an indication of reflux of chloride-rich secretions from the abomasum into the rumen. There is no treatment and affected sheep should be humanely killed to avoid further suffering once the diagnosis is confirmed.

The cause of abomasal emptying defect is unknown. The significance of diagnoses predominantly in the Suffolk breed is unknown and an hereditary basis has not been proven. Similar signs have been reported in sheep with scrapie, occurring after a period of ill thrift, rather than being the primary cause of weight loss.

Johne's disease

Johne's disease is an important cause of slow progressive weight loss in adult sheep (Fig. 4.26). The condition is probably under-diagnosed in UK flocks because ill thrift

Fig. 4.26 Emaciation in a Shetland ram resulting from Johne's disease.

in adult sheep is not routinely investigated and because the only consistent clinical sign is weight loss. Unlike cases of Johne's disease in cattle, scour is not a consistent feature in sheep.

The prevalence of ovine Johne's disease in the UK and economic importance in infected flocks is unknown. The onset of clinical disease is insidious and infection is probably already widespread once clinical disease is apparent. In one closely monitored Scottish Blackface hill flock, annual losses to Johne's disease of 1 to 2 percent of adult ewes have been confirmed, while in a small, intensively managed crossbred flock, annual losses of about 5 percent have occurred. Precise losses to the disease are difficult to quantify, because some animals which are ill thrifty due to Johne's disease are inevitably culled before they die on the farm.

Ovine Johne's disease has been reported from most sheep-producing countries, although the national prevalence and economic importance in infected flocks varies, associated with differences in climate, soil types, local breed susceptibility and production systems. In New Zealand, where economic sheep farming depends on the control of production-limiting disease and ill-thrift problems are routinely investigated, Johne's disease is suspected in about 5 percent of flocks. The incidence of the disease has increased substantially over the past decade and this trend is predicted to continue. The average annual mortality rate due to Johne's disease in infected New Zealand flocks is 1 percent, although losses of 5 percent have been reported.

Johne's disease is caused by the slow-growing, acid-fast bacterium, *Mycobacterium avium* subsp. *paratuberculosis*. Several strains have been identified from sheep, cattle, deer and many other mammal species. In general, most sheep strains do not establish in cattle, and vice versa, and where spread has occurred from sheep to cattle it is likely that the sheep strain is of low virulence for cattle. A sheep-specific pigmented strain is commonly identified in the south-east of Scotland, but is absent from many overseas countries including New Zealand.

Infected sheep shed small numbers of *M. a. paratuberculosis* bacteria in their faeces before they show signs of clinical disease. However, during the later stages of the disease when ill thrift is seen, large numbers of bacteria are shed. *M. a. paratuberculosis* can survive in the environment in faeces, soil or water for several months.

The main route of infection is oral, through faecal-contaminated food or water, although venereal infection and intrauterine infection following advanced disease in the dam have also been demonstrated. Following a long incubation period, *M. a. paratuberculosis* infection causes cellular infiltration and thickening of the mucous lining of the terminal small intestine, caecum and proximal large intestine, resulting in malabsorption and protein loss.

Clinical signs

The clinical outcome depends on the initial bacterial challenge and animal's immune response rather than on any subsequent re-infection. While sheep, unlike cattle, can first become infected at any stage in life, clinical disease is most likely to follow infection as lambs from faecal contamination of the dam's udder. The rate of progression of infection varies, associated with the strain of *M. a. paratuberculosis* involved and the individual animal's immune response. Weight loss and emaciation are usually seen in 2- to 5-year-old sheep, although cases have been confirmed in ewe lambs as young as 10 months old. Some animals die within a few weeks of the onset of ill thrift, while others survive for several months. Many cases are first seen during winter and in late-pregnant ewes, associated with the additional effects of nutritional stress and disease. Faeces may become soft, but not necessarily diarrhoeic, and intermittent oedematous swelling under the jaw associated with hypoproteinaemia is sometimes seen in the latter stages of the disease. Affected animals often have high gastrointestinal parasite burdens, associated with a compromised immune response. Animals maintain normal appetite throughout the clinical stage of the disease.

Sheep with clinical Johne's disease are profoundly hypoalbuminaemic due to albumin loss across the diseased intestine, with serum concentrations between 15 and 8 g/L. Confirmation of a diagnosis of ovine Johne's disease in the live animal is difficult. Serological tests or identification of acid-fast bacteria in Ziehl-Neelsen-stained faecal smears have proved to be unreliable, other than in the terminal stages of the disease, while bacterial culture is generally impractical due to the slow and fastidious growth of *M. a. paratuberculosis*. Negative results do not confirm absence of infection.

The AGIDT and faecal smear examination have high positive predictive values but low sensitivity for the diagnosis of subclinical infection and the specificity of ELISAs in sheep are poor. In the future, PCR may provide a reliable and rapid test.

Diagnosis

The diagnosis of ovine Johne's disease is usually confirmed by postmortem findings, including gelatinous atrophy of fat depots and obvious convoluted lymph vessels on the serosal surface of the affected intestines due to absence of fat cover (Fig. 4.27). Small 1 to 2 mm white lymphatic nodules are sometimes seen on the mucosal surface of the intestines (Plate 4.1), and mesenteric lymph nodes are large and pale. Thickening and corrugation of the mucosa of the terminal ileum are sometimes seen (Plate 4.2), but are not consistent features of ovine Johne's disease. The intestinal mucosa sometimes appears orange coloured (Plate 4.3), associated with the presence of massive numbers of pigmented *M. a. paratuberculosis*.

Fig. 4.27 Obvious lymph vessels on the serosal surface of the small intestines indicate, but do not confirm, Johne's disease.

Gross postmortem signs are not always obvious, so the diagnosis often depends on histological examination of sections of terminal ileum and mesenteric lymph nodes, or identification of large numbers of acid-fast bacteria in Ziehl-Neelsen-stained mucosal smears.

A range of postmortem findings is seen. Ovine Johne's disease is sometimes described as lepromatous, characterised by obvious postmortem lesions and numerous bacteria, or paucibacillary, characterised by less distinct granulomatous lesions rather than intestinal thickening and few bacteria. These different presentations probably reflect the dynamics of the host's ability to deal with infection, rather than the strain of *M. a. paratuberculosis* involved.

Management

There is no treatment for clinical Johne's disease. Scientific evidence about the spread and establishment of Johne's disease in sheep flocks is sparse, so advice about control of the disease is largely pragmatic. Differences in the incidence of Johne's disease between flocks are possibly associated with differences in sheep breed susceptibility and the presence of different strains of *M. a. paratuberculosis*, while differences in stocking rates, climatic conditions, winter housing and feed management probably influence environmental contamination and the rate of spread of infection. Furthermore, nutritional stress and the presence of other diseases such as parasitic gastroenteritis or tick-borne fever probably influence individual animals' susceptibility to clinical disease.

High levels of *M. a. paratuberculosis* infection have been identified in rabbits, wild deer and other wildlife. In a Scottish study, *M. a. paratuberculosis* was found in nearly 70 percent of wild rabbits. However, it is unknown whether wildlife acts as a reservoir or has a role in disseminating infection.

Sheep are most likely to develop clinical Johne's disease if initial infection occurs during the first few months of life. Therefore, high stocking densities or housing at lambing probably influence the spread of disease and its incidence in flocks which retain their own replacement breeding ewes.

The spread of Johne's disease within a flock is insidious, so infection is well established and has probably been present for some time before the presence of disease is

diagnosed. Only a small proportion of infected animals show clinical signs, so in the absence of a reliable test for preclinical infection, ovine Johne's disease cannot presently be controlled using a test-and-cull programme.

Aggressive culling of thin 3- to 5-year-old ewes can help to reduce environmental *M. a. paratuberculosis* contamination. Culling all thin ewes is impractical, so thin ewes which might have Johne's disease are usually initially wormed and provided with supplementary good-quality feed. Some respond because they do not have Johne's disease, while some affected animals also show a temporary improvement, which enables them to be slaughtered rather than die on the farm. Those ewes which do not respond to preferential management should be culled before lambing time.

The incidence of Johne's disease is highest in flocks breeding their own replacements and the risk of clinical Johne's disease is high in ewe lambs born to infected dams, due to infection from faecal contamination of their udders. Careful record keeping can enable culling of female progeny of ewes which subsequently show clinical Johne's disease. Rearing replacement ewe lambs away from heavily contaminated areas may also reduce the risk of clinical disease. Faecal contamination of feeding areas should be avoided by regular moving of troughs, or using cobs spread over different areas each day. Stress such as underfeeding or other concurrent diseases should be minimised.

Ovine Johne's disease is controlled in many overseas countries using live attenuated vaccines. Replacement breeding sheep are selected early and injected once before weaning. Annual boosters are not required. In many endemically infected flocks, vaccination has eliminated clinical cases altogether. In others, vaccination appears to shift infection towards a more manageable tuberculoid, paucibacillary form of the disease.

Once started, vaccination programmes need to be continued indefinitely, because while vaccination prevents clinical disease, it does not eliminate infection. Severe tissue reaction is often seen at the site of injection. Vaccinated animals may show positive reaction to tuberculosis tests, which is largely irrelevant in most commercial flocks, but precludes the use of Johne's vaccines in flocks where animals may require certification for export.

The effect of vaccination on adult animals that may already be infected is uncertain. However, limited evidence suggests that vaccination may modify the natural course of the disease and enable infected animals to maintain productivity throughout a normal lifespan.

A non-commercial live attenuated vaccine was available in the UK until 2005, supplied through veterinary practitioners by the Central Veterinary Laboratory. While this product is no longer available, it is now possible to import overseas vaccine for use in UK flocks. The effectiveness of overseas vaccines against pigmented strains of *M. a. paratuberculosis* is unknown.

Prevention of Johne's disease is difficult. The economic effect of Johne's disease is probably highest in flocks breeding their own female replacements. The only animals entering these flocks are breeding rams, so it is important to ensure that these are sourced from flocks with no history of clinical Johne's disease. However, in the absence of a reliable test for *M. a. paratuberculosis* in live animals, purchased stock cannot be assured free from Johne's disease. Pooled faecal culture may provide an

Reasons for concern about Johne's disease in UK flocks

Annual losses of 1 percent in commercial flocks of mixed-age ewes may not be particularly significant, although the disease has the potential to cause higher losses. Should this become the case, failure to have attempted to control the disease earlier would be regretted. In stud flocks the loss of 1 percent of adult sheep is economically significant. Furthermore there is an ethical dilemma associated with sale of known infected breeding rams, cast ewes, or ewe lambs.

A speculative link has been suggested, but not proven, between *M. a. paratuberculosis* and human Crohn's disease. Crohn's disease causes unpleasant chronic granulomatous enteritis in man, with similar pathological features to ovine Johne's disease. *M. a. paratuberculosis* has rarely, but not consistently, been cultured from Crohn's disease patients and identification of *M. a. paratuberculosis* DNA in pasteurised milk has given rise to the view that Crohn's disease arises as a result of consumption of such contaminated milk. While any link between Johne's disease and Crohn's disease is purely speculative, should further evidence arise, even if tenuous, ovine Johne's disease could become a food standards issue. Should this occur, the sheep industry's case would be strengthened by having adopted a serious approach to the control of ovine Johne's disease.

Johne's disease was first diagnosed in Australia in 1980 and has subsequently spread by sheep movement to some, but not all states. Annual losses of 2.5 to 15 percent of adult sheep have been reported in infected Merino flocks, depending on stocking rate and management practices with replacement sheep.

The Australian sheep industry has not been prepared to accept the economic loss and legal liability of Johne's disease-affected farms, or the potential trade and public health concerns. As a result control programmes have been instigated to prevent the continued spread of the disease from known foci of infection, including eradication of the disease by slaughter followed by depopulation for two consecutive summers and restriction of movement of sheep between zones. Compensation is financed by industry levies. Sheep from known infected flocks may not be sold other than for direct slaughter, under legal constraint.

Johne's disease is probably more widespread in the UK than in Australia, and British breeds are probably less susceptible than Merinos. However, the Australian experience illustrates the need to take the diagnosis, control and prevention of Johne's disease seriously in UK flocks.

opportunity to screen these groups for disease. The risk of introduction of ovine Johne's disease from infected wildlife is unknown.

Bacterial enteritis

Diarrhoea is sporadically reported in weaned lambs and adult ewes during the winter months, putatively associated with severe nutritional stress. Various causative bacteria have been implicated, including *Campylobacter coli, C. jejuni, Yersinia pseudotuberculosis and Y. enterocolitica*. Morbidity rates are usually high and affected sheep lose weight. While metaphylactic antibiotic treatment, using long-acting oxytetracycline injections, has been reported to control outbreaks, most cases recover spontaneously after about 2 weeks.

Fig. 4.28 Ill thrift and ascites in a Suffolk ewe due to intestinal adenocarcinoma. (The cause of the fetlock joint effusions was not determined.)

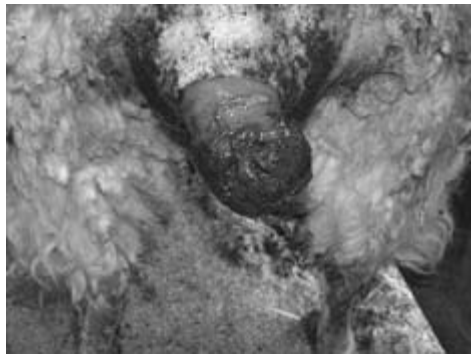


Fig. 4.29 Rectal prolapse is an obvious cause of discomfort and ill thrift in individual sheep.

Small intestinal adenocarcinoma

Small intestinal adenocarcinoma is a sporadic cause of progressive ill thrift, emaciation and death in adult sheep (Fig. 4.28). Affected sheep show a progressive loss of appetite and gradually increasing abdominal distension due to ascites. Cases have been reported in yearling animals, but the incidence increases with age. The disease occurs sporadically in the UK, but in New Zealand, annual prevalences of 3 percent have been reported in some flocks.

On postmortem examination large volumes of ascitic fluid are often present in the abdominal cavity. While the primary tumour occurs in the duodenum (Plate 4.4), the postmortem appearance is usually that of a dense white ring of fibrous tissue circling the serosal surface of large sections of small intestine (Fig. 3.25). Fibrous adhesions are present between adjacent loops of affected intestine. The mucosal and muscle layers of the intestinal wall are thickened, causing constriction of the lumen, with intestinal dilation proximal to the affected tissue. Fibrous deposits of tumour tissue are sometimes seen on the serosal surfaces of other abdominal viscera, on the diaphragm and on the peritoneal wall. Evidence of metastasis to the mesenteric lymph nodes is sometimes seen.

A relationship between small intestinal adenocarcinoma and grazing on bracken has been proposed, but tumours are also commonly seen in flocks with no access to bracken.

Rectal prolapse

Rectal prolapse (Fig. 4.29) occasionally occurs in weaned lambs and adult sheep as a sequel to tenesmus linked to a variety of causes such as diarrhoea, coughing, cystitis and abdominal distension. It has also been reported in Australia and North America in sheep grazing oestrogen-rich pastures, such as subterranean clover.

Other tumours

With the exception of jaagsiekte and intestinal adenocarcinomas, tumours are unusual causes of ill thrift, because most sheep are culled before reaching the age at

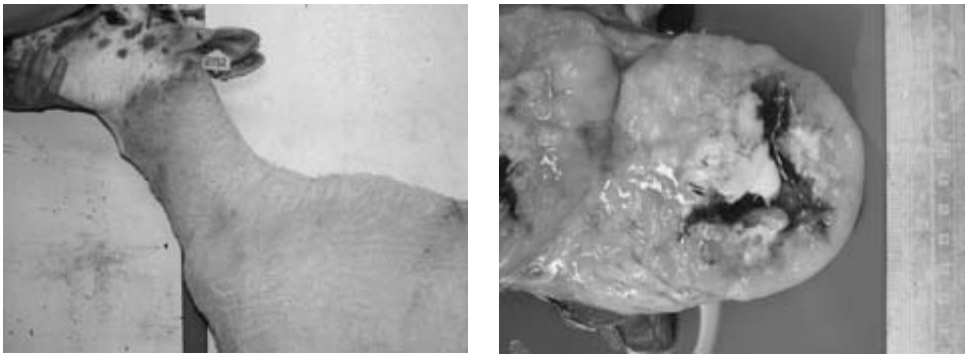


Fig. 4.30 (a) Bilateral enlargement of the superficial lymph nodes of a Greyface ewe due to generalised lymphosarcoma. (b) Postmortem appearance of an enlarged parotid lymph node with a caseous centre from the same ewe.

Fig. 4.31 A massive generalised lymphosarcoma tumour on the flank of a New Zealand Romney ewe.

which they would occur. Multicentric lymphosarcomas occur sporadically in both young lambs and adult sheep, characterised by palpably enlarged superficial lymph nodes (Fig. 4.30) and generalised lymphadenopathy, sometimes with diffuse or nodular tumour cell infiltraton of the thymus and other non-lymphoid organs (Fig. 4.31). Localised lymphosarcomas, involving diffuse tumour infiltration of the kidneys (Plate 4.5), are occasionally diagnosed at postmortem examination of ill-thrifty adult sheep.

Chapter 5

Lameness

Lameness is a major production-limiting disease of sheep of all ages, in addition to being an important animal welfare concern (Fig. 5.1). In some UK flocks more than 15 percent of sheep may be lame at some stage during the year, while in others the incidence remains low as a consequence of effective management. However, no flocks are able to remain completely free from lame sheep.

Diseases of the foot

The most common causes of foot lameness are interdigital dermatitis and footrot, but in individual flocks foot abscess, toe abscess, ovine digital dermatitis, white line separation, interdigital growths and toe granulomas can present serious problems.

Many of these diseases have a common primary cause, the bacterium *Fusobacterium necrophorum*, which is normally present in ruminant faeces and is always present on grazed pasture. *F. necrophorum* produces a number of toxins, which cause necrosis of the superficial layer of the interdigital skin (Fig. 5.2) and enable the establishment of other bacteria, including *Dichelobacter nodosus*, which causes footrot. The occurrence and persistence of foot problems are mostly related to those predisposing factors which enable *F. necrophorum* infection of the interdigital skin or white line (Fig. 5.3). The most important risk factors are maceration of the

Fig. 5.1 Images such as this of a very lame Scottish Blackface ewe, seen from the side of a public road, are not publicly acceptable and threaten the tenability of sheep farming.

Fig. 5.2 *F. necrophorum* produces toxins which cause inflammation of the interdigital skin and lower its defences against more invasive bacteria.

Sheep Flock Health: A Planned Approach. Neil Sargison
 ISBN: 978-1-4051-6044-5

Fig. 5.3 About 50 percent of these Greyface ewe lambs had advanced footrot lesions. The predisposing management factors in this case included housing on wet bedding in an inappropriate open-fronted, concrete-floored shed.

Fig. 5.4 Keeping sheep in muddy conditions results in maceration of the interdigital space and provides an opportunity for bacterial infection.

Fig. 5.5 Grazing on coarse stubbles traumatises the interdigital space, enabling infection with *F. necrophorum*, *A. pyogenes* or *D. nodosus*.

interdigital skin due to constant exposure to moisture (Fig. 5.4), and mechanical trauma, for example caused by grazing on long, mature ryegrass pasture or cereal stubbles (Fig. 5.5).

Before instigating any control programme, it is first important to establish an accurate diagnosis. The diagnosis of foot abscess, toe abscess, white line separation, interdigital growths, thorn injuries and toe granulomas in individual animals is relatively straightforward, but when a large proportion of the flock is affected, the cause is usually either scald or footrot. Interdigital dermatitis and footrot both involve the interdigital skin, but while advanced and severe cases of footrot can be diagnosed on the basis of under-running of the hoof horn and a characteristic smell, early and benign footrot lesions cannot be differentiated from scald on the basis of clinical signs alone. Digital dermatitis is characterised by primary involvement of the coronary band, but the diagnosis is usually complicated by the coexistence of scald or footrot.

Interdigital dermatitis

Ovine interdigital dermatitis (scald) is the most common cause of lameness in sheep, which occurs on all farms, whenever conditions underfoot are wet. The disease is

Fig. 5.6 Interdigital dermatitis causing severe lameness in 8- to 12-week-old lambs grazing on rank grass.

Fig. 5.7 Interdigital dermatitis, characterised by red, swollen interdigital skin, covered by a layer of white exudate.

usually caused by *F. necrophorum* infection alone, and does not require the presence of *D. nodosus*. However, interdigital dermatitis is clinically indistinguishable from benign footrot and early virulent footrot, so *D. nodosus* may also be present in many cases with the clinical appearance of scald.

Typically many animals in the flock are lame in one or more feet. In extreme cases, 90 percent of the flock can be affected. Rubbing caused by long grass can exacerbate the problem and lameness can persist for several months if untreated (Fig. 5.6). At grass, the incidence is generally greater in lambs than in ewes, but scald is commonly problematic in housed ewes, where straw bedding becomes wet and warm.

Clinical signs

The diagnosis of scald is usually based on clinical examination of lame animals. In mild cases the interdigital skin is red and swollen and covered by a thin layer of white necrotic material (Fig. 5.7). In more severe cases, the interdigital skin is eroded to expose deeper, sensitive subcutaneous tissue. Unlike cases of footrot, there is no under-running of the hoof wall or sole and no characteristically unpleasant smell.

Management and control

Uncomplicated cases of scald, which may include interdigital dermatitis and benign footrot, often recover spontaneously when sheep are moved to dry pasture, although this is seldom a practical management strategy. Pasture around feed troughs and gateways can become trampled, muddy and heavily contaminated with faeces, so regular movement of troughs and avoidance of these areas can significantly reduce the incidence of foot diseases (Fig. 5.8).

Individual cases of scald can be treated topically using oxytetracycline aerosol sprays. When several animals are affected, walking of sheep through a 10 percent zinc sulphate solution or 3 percent formalin usually provides effective treatment, although the protection afforded only lasts for a few days (Fig. 5.9) (3 percent formalin is prepared by adding 300 ml of formalin solution (40 percent formaldehyde) per 10 litres of footbath solution). Walk-through footbaths need to be about 5 m long

Fig. 5.8 Simple practices such as regularly moving feed troughs to avoid excessively muddy conditions can help to reduce the incidence of interdigital dermatitis.

Fig. 5.9 Interdigital dermatitis can be both treated and controlled by walking sheep through a 10 percent zinc sulphate solution or 3 percent formalin.

Fig. 5.10 Foot bathing for lameness management is futile if sheep are forced to stand in or walk through muddy gateways soon afterwards.

Fig. 5.11 The practice of periodically replenishing the footbath with an unknown amount of formalin must be avoided to reduce the risk of foot bathing in excessively concentrated solutions.

and 5 to 8 cm deep. Repeat treatments are required at 5- to 14-day intervals throughout the risk period, depending on environmental conditions. Afterwards sheep should be allowed to stand in a dry area so that the formalin or zinc sulphate can dry on the feet (Fig. 5.10). The critical issue is the length of time that the lesions are in contact with the solution before sheep are returned to pastures which remove the chemicals from the feet, so the total time in the bath plus the dry-standing should be about one hour. At concentrations greater than 5 percent, formalin can cause severe irritation of the interdigital space. The practice of regularly replenishing footbaths with a few splashes of concentrated solution should, therefore, be avoided (Fig. 5.11). Failure of this strategy to control the problem often indicates the presence of footrot, which requires more radical control. Interdigital dermatitis cannot be controlled by the use of footrot vaccines.

Interdigital dermatitis is also important because it is the first stage in the pathogenesis of footrot and suppurative infection of the distal interphalangeal joint (foot abscess).

Fig. 5.12 This Cheviot ram is grazing while supported by its knees because of severe foot pain due to footrot.

Footrot

Footrot is an extremely painful production-limiting disease of sheep of all ages (Fig. 5.12). Affected animals are ill thrifty, show reduced wool quality and yield and poor reproductive performance. Furthermore, the cost of treatment and control can be considerable in terms of both labour and veterinary medicines. Unfortunately, finding the time and impetus required for effective preventive management has become difficult due to the economic necessity for lower labour inputs per stock unit and a burgeoning administrative burden associated with sheep farming in the UK. However, in the longer term, the cost of managing an existing problem greatly exceeds that of effective disease prevention.

Footrot occurs as a sequel to interdigital dermatitis under specific circumstances when the bacterium *D. nodosus* is present. The severity of the disease depends partly on the strain of *D. nodosus*. Mild strains result only in separation at the heels and back of the sole (benign footrot), while virulent strains can result in complete separation of the horn of the hoof wall and sole. However, in the early stages of the disease, when infection is likely to be transmitted between animals, it is not possible to differentiate clinically between benign and virulent footrot.

Clinical signs

The first sign of virulent footrot is swelling and moistening of the interdigital skin (Fig. 5.13). A break occurs at the skin horn junction (Fig. 5.14) from where infection spreads under the horn tissue so that the wall of the hoof becomes separated (Fig. 5.15) and the sole under-run (Fig. 5.16). There is a characteristic unpleasant-smelling discharge. In longstanding cases (Fig. 5.17), the hoof walls and toes become overgrown and misshapen, trapping dirt and inflammatory exudate between the inflamed, granulating soft tissues of the sole and overgrown horn (Fig. 5.18). Animals with advanced footrot are extremely lame, remain recumbent for long periods and may carry the affected leg. When both forelimbs are affected, animals may walk on their knees. Severely affected feet often become flystruck (Fig. 5.19). Furthermore, affected sheep are prone to flystrike on their flanks, where the wool is soiled by exudate from their feet when they lie down.

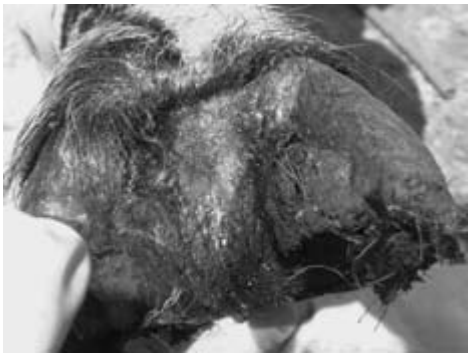


Fig. 5.13 Swelling and moistening of the interdigital skin.

Fig. 5.14 A break at the skin horn junction.

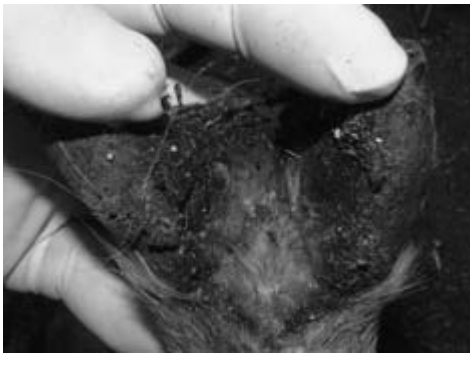


Fig. 5.15 Under-running of the axial hoof wall.

Fig. 5.16 Under-running of the sole.

Fig. 5.17 Under-running and severe inflammation of the sole.

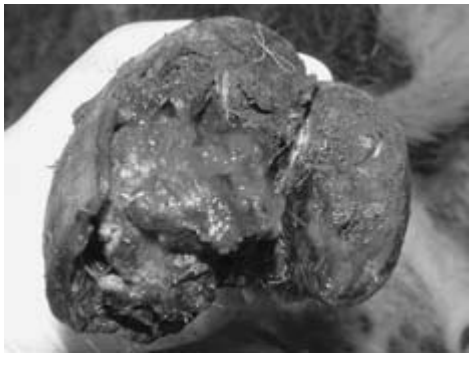


Fig. 5.18 Advanced footrot with a poor prognosis for successful treatment.

Fig. 5.19 Flystrike in an advanced case of footrot.

Footrot control

Various methods are employed for the control of footrot:

- foot bathing
- foot trimming
- antibiotic injections
- vaccination
- selection for resistance
- eradication.

In practice, footrot control is based on a combination of the above. However, it is essential that the application of each of these methods is based on a clear understanding of the pathogenesis of the disease. It is important to distinguish between those methods which are useful for treating severely affected sheep, such as trimming and parenteral antibiotics, and those which will help prevent disease when used correctly, such as foot bathing and vaccination.

The objective of footrot control is to prevent the development of painful and debilitating under-running lesions, using the least arduous and most cost-effective method. This is achieved in infected flocks by reducing the incidence of new infections to maintain the prevalence of disease at a low level. This approach utilises whole-flock control strategies such as foot bathing and vaccination before and from the start of the high-risk periods for disease transmission, rather than individual handling of each affected animal in order to treat advanced cases. If this objective is met, very few sheep in the flock will develop severe under-running infections and require individual treatment.

Foot bathing

Application of antibacterial solutions in a footbath is most effective for the control of footrot when practised during the early stages of the disease, when infection in previously unaffected sheep is limited to the interdigital skin and does not involve the hoof wall. Foot bathing usually needs to be repeated at fortnightly intervals during warm and wet weather when the risk of transmission is high, but when weather conditions are dry, single treatments can be extremely effective. Foot bathing alone is not particularly effective for the treatment of advanced footrot lesions.

Sheep should be walked through a footbath of 3 percent formalin or stood for up to one hour in 10 percent zinc sulphate solution (Fig. 5.20). The standing time in zinc sulphate footbaths can be reduced by the addition of a penetrating agent, such as sodium lauryl sulphate or a squirt of washing-up liquid. Sheep should be allowed to stand in a dry area for at least an hour after foot bathing to allow the chemical to dry on the feet (Fig. 5.21).

Before foot bathing, sheep should be run over coarse stones or slats to remove as much mud and faeces as possible (Fig. 5.22). Alternatively, they can first be walked through a footbath containing only water.

Excessive formalin foot bathing in concentrations exceeding 5 percent can result in foot damage and should be avoided. Furthermore, zinc sulphate is less effective if formalin has been used within the previous 2 months. Straw placed in the footbath to prevent splashing reduces the efficacy of formalin, but has little effect on zinc sulphate. Foam rubber mats, commonly used for cattle, may also be effective for small groups of sheep.

Fig. 5.20 Large footbathing pens can be useful in small flocks to ensure that groups of animals can be stood in the footbath for up to one hour.

Fig. 5.21 Even if a dry standing area is not available, it may be possible in small flocks to improvise to enable sheep to stand for one hour after foot bathing, before returning to fields.

Fig. 5.22 For larger flocks, good handling pen design is essential, to ensure that sheep feet are clean before foot bathing and that a sufficiently large dry standing area is available to accommodate the whole group afterwards.

Fig. 5.23 Foot paring is important for the treatment of footrot, but irrelevant in its prevention.

The failure of regular foot bathing to prevent the development of severe footrot is often caused by a combination of poor handling facilities and an ill-defined transmission period due to persistent wet weather. Effective foot bathing requires:

- walking sheep through water or running over stones or slats beforehand
- using the correct concentration of antibacterial solution
- standing on dry concrete or stones afterwards
- choosing a dry day.

Foot trimming

Traditionally routine annual foot trimming has been recommended for the control of footrot (Fig. 5.23). However, foot trimming has no role in preventing infection and should not be considered as a preventive method when planning control programmes. Furthermore, it has been suggested that unnecessary foot trimming may even facilitate the spread of *D. nodosus* between feet. However, hoof trimming is

Fig. 5.24 Excessive foot paring to the extent that blood is unnecessarily drawn must be avoided, because it predisposes to irreversible, painful granuloma formation.

useful to limit the effect of the disease and assist in the resolution of lesions after the infection has under-run the sole and hoof wall in uncontrolled cases of virulent footrot. If hooves become severely overgrown despite effective footrot control, then the suitability of the sheep breed for the environment and management system should be reconsidered. Over-zealous foot trimming must always be avoided because it often leads to permanent severe lameness due to granuloma formation (Fig. 5.24).

Antibiotic injections

The effect of parenteral antibiotics on interdigital lesions is minimal and they have no role in the prevention of footrot. However, injection of high doses of penicillin (30 mg/kg of procaine benzylpenicillin, compared to the standard therapeutic dose rate of 15 mg/kg), followed by dry standing for 24 hours can be useful for the treatment of advanced under-running lesions, provided that sheep are kept in a dry environment for at least 24 hours after treatment.

Vaccination

Subcutaneous injection of 1 ml of a formalin-inactivated vaccine containing most of the UK strains of *D. nodosus* in an oil adjuvant can be a useful adjunct for both control and treatment of footrot. An initial course of two injections 4 to 6 weeks apart is usually recommended, followed by booster doses in advance of high-risk periods in spring and autumn. Vaccination provides protection against infection for about 4 to 6 months and there is some evidence that it may also enable already affected feet to heal more quickly. In some cases a single dose of vaccine administered in the face of an outbreak can be used to reduce the severity of the disease. The net effect of vaccination can be to reduce both the prevalence and severity of footrot in the flock. However, whole-flock vaccination alone does not eradicate footrot and can prove expensive.

Unfortunately, in many parts of the UK, the high-risk period occurs throughout the year, so vaccination cannot economically provide the length of protection required. In many flocks, vaccination is targeted at specific high-risk groups of animals during known disease transmission periods, such as rams before mating.

Some local tissue reaction can occur at the footrot vaccination site, although lesions regress over time and do not rupture. Furthermore, footrot-vaccinated sheep cannot be subsequently treated with injectable moxidectin because both products employ the same carrier solution and there is a risk of fatal allergic reaction.

Fig. 5.25 Resistance to footrot appears to be moderately hereditable and genetic selection is theoretically possible.

Selection for resistance

There appears to be significant variation in the prevalence of lameness due to footrot between the progeny of different sires (Fig. 5.25). Host resistance to footrot, therefore, appears to be moderately hereditable and breeding of resistant sheep by selective culling could be considered as a potential control strategy. However, genetic progress using this strategy is likely to be slow. Genetic markers for resistance to footrot are currently being sought, aimed at candidate genes within regions of the sheep genome's major histocompatability complex (MHC), products of which are thought to have a role in presenting antigens to T lymphocytes involved in the immune response to *D. nodosus*. Polymorphisms have been found in New Zealand Merino sheep and their crosses and are currently being sought in British breeds. However, it seems likely that the genetic basis of resistance to footrot will be complex and breed specific, perhaps involving multiple polymorphisms as has been shown for resistance to scrapie. There is also a risk that selection for resistance to footrot based on a single genetic marker could have a negative impact on other production traits. Furthermore, while breeding of resistant sheep is a genuine possibility, the value of this strategy is limited by the stratified system of sheep production in the UK. To be most successful, selection should be practised at the level of the hill or longwool ram producer.

Footrot eradication

Unlike *F. necrophorum*, which is always present in the environment, *D. nodosus* only multiplies in diseased feet. Warm and moist pasture is required for the transmission of *D. nodosus* between animals, but the bacteria only survive in the soil for a maximum of about 4 days. It is, therefore, possible to eradicate footrot from affected flocks.

Eradication of footrot is possible through a combination of regular examination and separation of affected from non-affected animals, foot bathing and strict culling of persistently affected sheep. There are several pitfalls and attempts to eradicate the disease are frequently unsuccessful. Eradication should only be attempted during dry weather conditions when transmission of *D. nodosus* is slow and sheep are unlikely to be in the early stages of the infection, which could be missed or confused with scald. Unfortunately, identification of such slow transmission periods is difficult in many parts of the UK.

Replacement animals should be quarantined for at least one month, during which time they should be inspected. However, despite treatment of any infected animals, it may be impossible to prevent the re-introduction of the disease. Stray sheep from neighbouring flocks are another common source of re-infection. *D. nodosus* can also be carried in the feet of cattle and goats, although cattle strains are usually benign for sheep. However some goat strains of *D. nodosus* are particularly virulent for sheep, so goats should be included where eradication of the disease is attempted.

For many flocks, the cost of eradication can be considerable when compared to the cost of management of endemic footrot in the flock. However, in Australia, eradication is the preferred form of footrot management, involving:

- control of the disease during the transmission phase, using foot bathing or vaccination
- inspection and culling during the non-transmission phase to remove all clinical cases
- surveillance during the next 12 months to quickly identify and remove any suspect cases which may have been missed, before they are responsible for transmission to other sheep.

General advice

In the past, the importance of some purported control activities has been overstated. For example, the disinfection of hoof trimming equipment between feet and between sheep to stop transmission is not important when the sheep have been co-grazing the same pasture for weeks beforehand. In addition, advice that pastures should be rested for 2 weeks to avoid re-infection after clinically diseased sheep have been removed from a flock overstates the importance of the risk of re-infection from pastures, compared to the high probability that subclinically infected sheep will be left in the flock and will serve as a source of re-infection.

Foot abscess

The distal interphalangeal joint capsule lies extremely close the interdigital skin, so is vulnerable to trauma and infection. Interdigital dermatitis predisposes to foot abscessation by enabling deeper invasion by other pyogenic bacteria. Foot abscesses involve infection of the distal interphalangeal joint (Fig. 5.26) and are characterised by heat, swelling and discharging sinuses at the interdigital space (Fig. 5.27) and coronary band (Fig. 5.28), usually involving only one digit of the foot. The affected digit often appears to be splayed outwards from its normal axis (Figs 5.29 and 5.30).

Foot abscesses are important because they are extremely painful and result in rapid weight loss. In pregnant ewes, the disease predisposes to pregnancy toxaemia, while in rams it can result in poor reproductive performance. Annual incidences of 2 percent are commonplace, although outbreaks involving up to 10 percent of flocks have been reported, associated with failure to manage interdigital dermatitis, or with excessive use of formalin footbaths at inappropriate high concentrations.

Antibiotic treatment and flushing of the affected joint is generally unsuccessful. Untreated cases remain lame for several months. Overseas reports would suggest that most cases eventually heal if left alone, although this does not appear to be the case in UK flocks. The options of humane destruction or surgical removal of the affected digit should, therefore, be considered to alleviate further suffering.

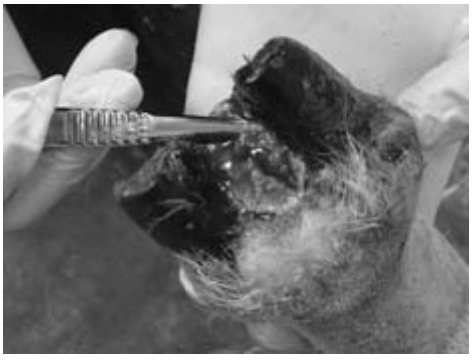


Fig. 5.26 A probe is inserted through a discharging sinus in the interdigital space into the distal interphalangeal joint of a ewe with a foot abscess. (Effective analgesia was administered before carrying out this demonstration procedure.)

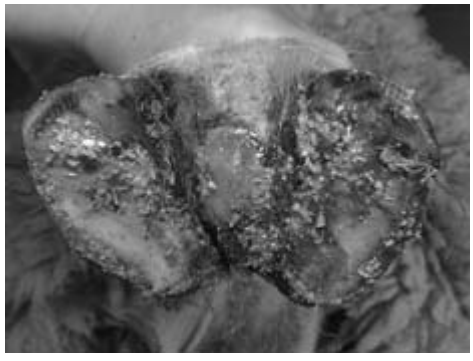


Fig. 5.27 Swelling and a discharging sinus within the interdigital space.

Fig. 5.28 Swelling and pus discharging from a sinus tract above the coronary band.

Fig. 5.29 Discharging sinus tracts in the interdigital space and above the lateral coronary band, severe swelling around distal interphalangeal joint on one side and outward displacement of the distal digit.

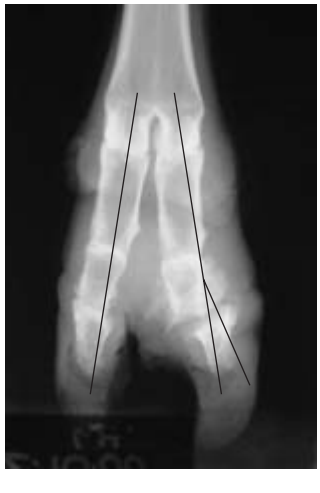


Fig. 5.30 A radiograph showing destruction and dislocation of the distal interphalyngeal joint and lateral displacement of the distal digit from its normal axis.

Digit amputation

Surgical amputation of the digit under intravenous regional local analgesia is a relatively straightforward and cheap procedure, following which most sheep walk normally within a few weeks.

An elasticated bandage is tightly wrapped around the affected limb to form a tourniquet, immediately below the elbow or hock (Fig. 5.31). The hair over the cephalic or recurrent tarsal vein is clipped and the area surgically prepared. The cephalic vein runs diagonally over the antero-medial aspect of the fetlock of the thoracic limb, while the recurrent tarsal vein runs diagonally antero-laterally above the fetlock joint of the pelvic limb. 5 ml of 2 percent lignocaine (addition of adrenalin to the local anaesthetic should not present any problems) is injected into the raised vein using a 20 gauge, 1 inch needle and pressure applied to the vein as the needle is withdrawn (Fig. 5.32). Analgesia distal to the tourniquet is achieved during the time taken to prepare the surgical site.

The distal limb is clipped above the coronary band of the affected digit and the interdigital space and clipped area are thoroughly washed. Embryotomy wire is placed in the interdigital space and the affected digit removed by cutting at an angle of about 15° to the horizontal. Cutting at a steeper angle can result in instability of the healthy digit. A small non-adhesive dressing is applied to the wound, which is then padded and firmly bandaged. The bandage is normally changed twice at 3- to 4-day intervals and removed about 5 days after the second change, at which stage granulation tissue covers the bone (Fig. 5.33). A postoperative course of broad-spectrum antibiotic injections is required.

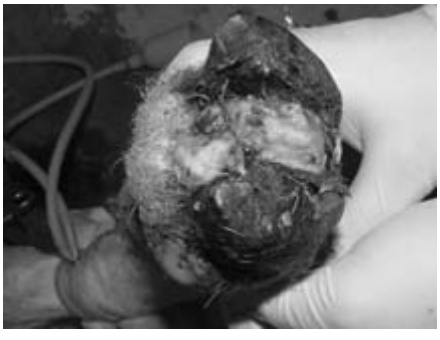


Fig. 5.31 Application of a tourniquet, made from rubber tubing or a length of elasticated bandage.

Fig. 5.32 Injection of local anaesthetic into a recurrent tarsal vein.

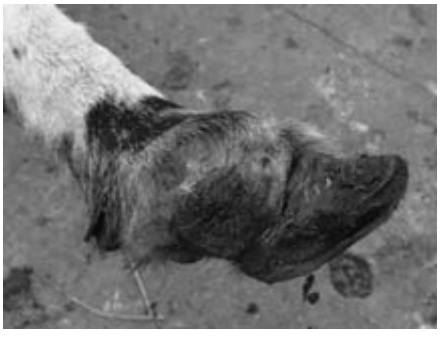


Fig. 5.33 Healing of the skin over the site of digital amputation within about 3 weeks after surgery.

Fig. 5.34 Abscessation of the axial white line.

Fig. 5.35 Abscessation of the abaxial white line.

Prevention of foot abscessation depends on the control of scald and appropriate treatment of interdigital disease. Foot bathing in excessive concentrations of formalin must be avoided.

Toe abscess

Toe abscess (white line abscess; lamellar suppuration) is a sporadic cause of acute-onset, severe lameness, usually involving only one digit. Animals are usually non-weight-bearing on the affected limb. Abscesses are usually present a few millimetres from the point of the toe at the white line on either wall of the hoof (Figs 5.34 and 5.35). The affected digit is usually hot and the coronary band is sometimes swollen at the level of the abscess. Sinus tracts discharging pus are occasionally present at the coronary band. The abscesses can be difficult to find, but animals usually flinch when the affected area is squeezed.

Outbreaks of toe abscesses are sometimes seen in autumn, associated with wet and muddy conditions. Culture of pus from the abscesses usually yields environmental bacteria including *F. necrophorum*. These bacteria gain access to the sensitive laminae through small fissures associated with wet and abrasive conditions underfoot. Shelly toe (lamellar separation) and overgrowth of the hooves associated with other foot diseases probably also predispose to toe abscess.

Most cases respond rapidly to careful paring to reveal and release the trapped pus. Pus is sometimes released under high pressure. It is then usually necessary to pare away under-run horn to open out the abscess. Excessive paring (Fig. 5.36) must be avoided, because it predisposes to granuloma formation (Fig. 5.37). It is common practice to administer parenteral antibiotics.

Ovine digital dermatitis

Ovine digital dermatitis (CODD) describes a rapidly spreading foot disease affecting sheep of all ages, not dissimilar to digital dermatitis in cattle. The disease was first seen in the UK in 1999 and became widespread over the following few years, before more or less disappearing from most areas. Ovine digital dermatitis is characterised by a high morbidity rate (up to 70 percent) and severe lameness in affected animals. Unlike scald and footrot, which involve inflammation of the interdigital space, the

Fig. 5.36 Excessive paring to identify the seat of a toe abscess should be avoided because it predisposes to granuloma formation.

Fig. 5.37 A painful, irreversible granuloma following excessive paring of a toe abscess.

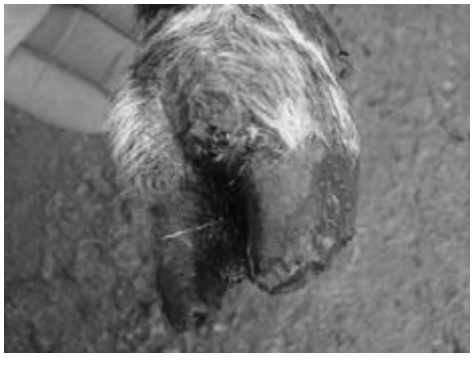


Fig. 5.38 Ulceration of the coronary band at the front of the foot.

Fig. 5.39 Ulceration of the lateral coronary band.

initial lesion appears to be full-thickness skin ulceration at the coronary band (Figs 5.38 and 5.39). In some cases the interdigital space is also involved or the hoof wall is under-run and shed to expose the sensitive laminae (Fig. 5.40). It is not uncommon for the entire hoof wall to be shed, exposing sensitive tissue which bleeds profusely when traumatised (Fig. 5.41). Concurrent 1 cm diameter circular areas of hair loss and ulceration are sometimes seen on the skin of the lower limbs (Figs 5.42, 5.43, 5.44 and 5.45), which appear similar to orf, or so-called ovine viral ulcerative dermatosis lesions.

Cause

In some cases proliferative lesions at the coronary band appear similar to orf. However, orf virus has not consistently been identified in scab material from the lesions. An association has been made between cases of ovine digital dermatitis and the presence of motile spirochaetes. However, motile spirochaetes may also be present on the lower limbs of healthy sheep and their contribution to foot diseases is unclear. While *F. necrophorum* and *D. nodosus* are not consistently involved in the pathogenesis of ovine digital dermatitis, their presence may result in increased severity of

Fig. 5.40 Under-running of the hoof wall starting at its junction with the coronary band.

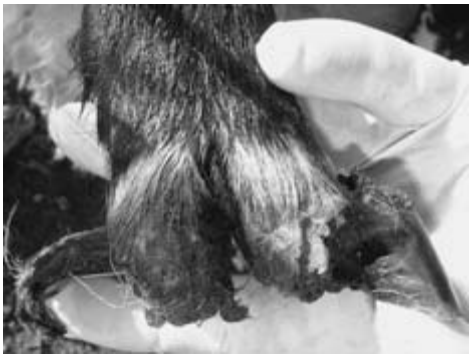


Fig. 5.41 Shedding of the hoof wall of both digits of one foot.

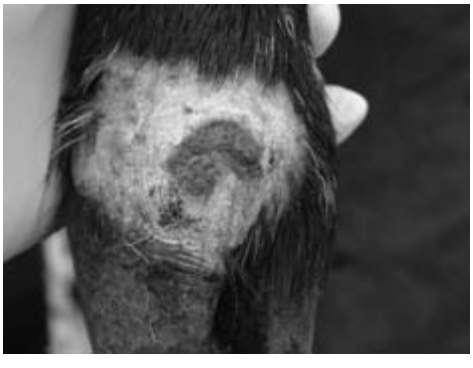


Fig. 5.42 An ulcerative skin lesion below the pastern joint.

Fig. 5.43 A bleeding ulcerative skin lesion above the coronary band.

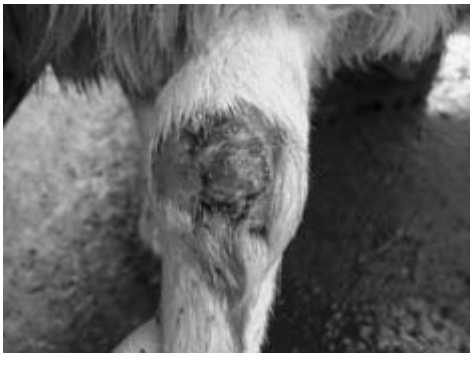


Fig. 5.44 A large bleeding ulcerative lesion over the fetlock joint.

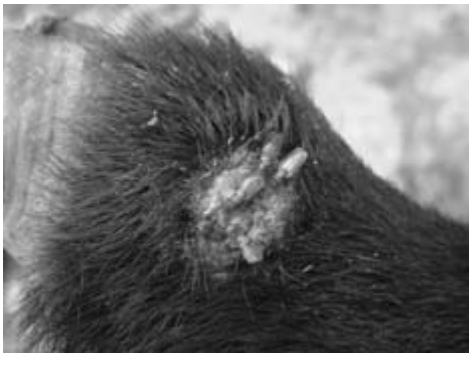


Fig. 5.45 Nuisance flies around an ulcerative digital dermatitis skin lesion on a Suffolk lamb, causing considerable annoyance.

the foot lesions. It is frequently reported that the severity of the disease is exacerbated after walking sheep through formalin footbaths, and it has been suggested that formalin may have a contributory role in the disease. The cause of ovine digital dermatitis, therefore, remains unproven.

Control

The response to conventional footrot control measures such as foot bathing in formalin or zinc sulphate solutions is poor. Lesions heal quickly after topical treatment with oxytetracycline aerosol sprays. However, such treatment is impractical in large flocks and other limbs often become involved after the initial lesion has healed. There is evidence to suggest that regular topical antibiotic treatment or foot bathing for about 20 minutes in tylosin or lincomycin antibiotic solutions is effective for prevention and treatment of ovine digital dermatitis (for example, 1 g tylosin per litre of water). Alternatively, antibiotic solution can be applied to the limbs of small numbers of sheep in a sheep pen using a pressurised garden weedkiller sprayer. Sheep should be allowed to stand in a dry area afterwards so that the medication can dry onto the feet. Individual cases may also benefit from systemic antibiotic treatment.

Overgrowth and separation of the hoof wall

Overgrowth and separation of the superficial hoof wall close to the white line at the toe (sometimes referred to as shelly toe) is commonly seen in sheep grazing on lush pasture. The condition is only associated with lameness when soil or faecal material are forced into the crack, placing pressure on the sensitive laminae (Fig. 5.46). Hoof wall separation may predispose to toe abscessation. Treatment involves paring away the separated hoof wall to alleviate the pressure on the underlying sensitive laminae.

Laminitis

Laminitis is a common problem in concentrate-fed sheep. Affected animals typically spend long periods in recumbency and stand with all four limbs drawn under their body, while shifting their weight from limb to limb (Fig. 5.47). Most cases recover spontaneously within a few days, but the problem may lead to toxaemia in late-pregnant ewes.

Fig. 5.46 Separation of the hoof from the white line resulting in lameness due to subsequent impaction of grit.

Fig. 5.47 All four limbs drawn under the body of a Mule ewe lamb due to pain affecting all four feet.

Fig. 5.48 Granulomas associated with toe abscessation and sinus formation draining to the coronary band.

Fig. 5.49 Granulomas associated with a chronic footrot lesion.

Laminitis is also seen with systemic viral diseases, such as foot-and-mouth and bluetongue.

Granulomas

Granulomas appear as smooth or strawberry-like growths, up to 3 cm across, on the sole or axial hoof wall close to the toe. Granulomas are often associated with injudicious, over-enthusiastic foot paring, but can also occur following toe abscessation (Fig. 5.48) or severe longstanding footrot (Fig. 5.49).

Affected sheep are extremely lame and lesions bleed when handled. Toe granulomas do not recover without treatment. The granulomatous tissue needs to be cut back under local anaesthesia and adjacent loose horn pared. Wounds should be bandaged to prevent bleeding. Cauterisation of the base of the granuloma or repeated application of copper sulphate as an astringent are sometimes advocated, but are unproven management methods.

Interdigital fibromas

Fibrous growths are commonly seen in the interdigital space of one or more feet (Fig. 5.50). They may be associated with a reaction to previous interdigital infections,

Fig. 5.50 Overgrowth of fibrous tissue within the interdigital space of a Suffolk ram.

Fig. 5.51 Thorn injury to the sole of a 3-week-old lamb (*arrow*).

Fig. 5.52 An interdigital wart-like lesion in a 5-month-old Texel cross lamb, possibly associated with previous interdigital dermatitis.

although the cause has not been proven. The prevalence of interdigital fibromas appears to be particularly high in certain terminal sire breeds. While uncomplicated growths seldom cause lameness, affected feet are prone to interdigital infections in the clefts between the growths and interdigital skin. These can be troublesome in rams at tupping time.

Removal of interdigital fibromas under local anaesthesia is sometimes advocated, but many subsequently re-grow. Most secondary infected cases respond to topical antibiotic treatment.

Other causes of foot lameness

The list of other sporadic causes of foot lameness in sheep includes:

- soil and grass balling between the digits
- interdigital foreign bodies
- thorn injury to the sole of young lambs (Fig. 5.51)
- wart-like lesions in the interdigital space (Fig. 5.52)
- frost irritation of the interdigital space
- systemic viral diseases.

Lameness not caused by foot problems

Sheep lameness is occasionally caused by skin diseases of the lower limbs, muscle disease, joint disease and bone diseases. Furthermore, sheep with neurological disorders such as peripheral nerve damage and spinal cord lesions may at first appear to be lame.

Post-dipping lameness

Post-dipping lameness is caused by *Erysipelothrix rhusiopathiae* and is seen in sheep of all ages. Typically, a high proportion of the flock is affected between 2 and 7 days after plunge or shower dipping. The disease is initially characterised by pyrexia and severe lameness, with heat and swelling associated with severe cellulitis involving the coronary band and interdigital area of one or more distal limb.

Most cases resolve after a few days, but prompt parenteral penicillin therapy is usually recommended, because in some individual animals bacteraemic spread results in swelling and painful non-suppurative arthritis in one or more joints about 2 to 3 weeks after dipping (Fig. 5.53). The response to treatment in these cases is poor.

The source of infection is faeces-contaminated dip, in which *E. rhusiopathiae* can multiply rapidly and can reach infectious levels within 24 hours. Sheep should be dagged if necessary, yarded overnight, and run over slats or stones to remove excess soil and faecal material from the feet before dipping. The dipper sump should be drained and refilled the following day rather than being left full overnight. Alternatively, if this is impractical, a dip-compatible bacteriostat can be added to reduce the risk of post-dipping lameness.

Strawberry footrot

Strawberry footrot is a rare cause of low-grade lameness associated with *Dermatophilus congolensis* infection. Orf virus may occasionally be involved in the pathogenesis of the disease. The disease only occurs under exceptionally wet conditions and is characterised by proliferative scab formation above the coronary band at the back of the limbs (Fig. 5.54). Fissures form to expose raw bleeding tissue and result in the characteristic 'strawberry' appearance (Fig. 5.55).

Fig. 5.53 Non-suppurative arthritis affecting the carpal joints of a group of Scottish Halfbred gimmers, about 6 weeks after plunge dipping for blowfly control.

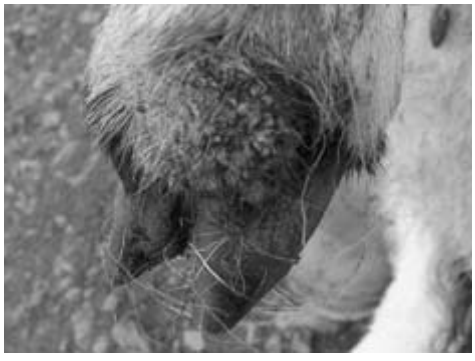


Fig. 5.54 A proliferative lesion above the coronary band, characteristic of strawberry footrot.

Fig. 5.55 A characteristic 'strawberry' lesion.

Most cases respond to movement to a drier environment, so treatment is not always warranted. However, topical antibiotic treatment with aureomycin aerosol sprays and foot bathing in zinc sulphate solution could be considered in severe outbreaks.

Muscle diseases

Stiffness or lameness may be due to muscle disease, which may, for example, be caused by selenium or vitamin E deficiencies. Bruising or abscessation in the muscles may be due to injection site reaction, for example following the use of dirty needles, or caused by trauma or dog bites. Lameness results from pain, limitation of the range of movement, or nerve and muscle damage.

Arthritis

Infectious arthritis

Most cases of bacterial polyarthritis occur as a sequel to neonatal bacteraemia, associated with inadequate passive colostral antibody transfer. Bacteria localise in the highly vascular synovial membranes of the joints, following which infection either resolves or persists. Persistent infection occurs mostly in larger, complex limb joints, causing severe septic arthritis (Fig. 5.56) with destruction of articular cartilage.

Fig. 5.56 The postmortem appearance of septic arthritis of the stifle joint of a 6-month-old Scottish Blackface ram.

Concurrent osteomyelitis sometimes occurs in the epiphyses or metaphyses of adjacent bones, vertebral bodies or intervertebral discs.

The major infectious causes of polyarthritis in young growing lambs are *Streptococcus dysgalactiae* and *E. rhusiopathiae*. Other causes include tick-bite pyaemia and puncture wounds. In the acute stages of infectious arthritis, the joint space becomes distended with inflammatory fluid, which can be imaged using ultrasonography. Radiography is generally unhelpful as there is little change in the bone, except for widening of the joint space. A sample of joint fluid may be obtained using strict asepsis, and will usually be yellow and turbid with an elevated white blood cell count ($>1 \times 10^{10}$/L, mostly neutrophils) and elevated protein levels (over 40 g/L; normally less than 18 g/L).

Chronic cases are sometimes seen in older animals, where the joints are sterile. In contrast to the acute stages, excess joint fluid is not present. However, a chronic synovitis develops leading to marked thickening of the synovial membrane and joint capsule. There is usually marked bony remodelling of the joint, progressing to ankylosis and thus radiography can be helpful in demonstrating the chronicity of the disease.

In some countries, chronic non-suppurative arthritis in older sheep may be caused by lentivirus infection.

Degenerative joint disease

Ostechondrosis has been described in the elbow joints of rapidly growing, male Suffolk lambs, although the condition appears to be much less common in sheep than in other species. The disease is characterised by progressive lameness, although too few cases have been reported in sheep to give an accurate description of the clinical signs. As in other species genetics, age, physical activity, rapid growth rates and high planes of nutrition have all been suggested as predisposing factors.

Chronic degenerative osteoarthritis of one or both elbow joints is commonly seen in adult sheep, resulting in severe lameness and ill thrift (Fig. 5.57). The incidence and predisposing factors are unknown, but may include genetic factors, such as breed, anatomy and conformation, and environmental factors, such as rapid growth rates, nutritional imbalances and repeated trauma. The condition could possibly occur as a sequel to osteochondrosis. Affected joints are grossly thickened, with a limited range of movement and pain on palpation. Radiography may be helpful for definitive diagnosis, revealing extensive new bone formation at the attachment of the joint capsule (osteophytes) and joint ankylosis. Postmortem examination reveals

Fig. 5.57 Severe shoulder arthritis in a Beltex ram. The pelvic limbs are drawn under the body to relieve some of the weight from the affected thoracic limb.

Fig. 5.58 A boiled-out specimen, showing extensive new bone formation around the elbow joint.

Fig. 5.59 Traumatic injury close to the hock joint. Consideration must be given to the fact that lesions are extremely painful when clinically examining affected animals.

erosions of the articular cartilage and extensive new bone formation around the joint, which may make the joint very difficult to open (Fig. 5.58). Effective treatment is impractical and affected sheep should be culled.

Spondyloarthrosis

Progressive lameness and periodic caudal extension of the pelvic limbs of large rams has been associated with the presence of osteophytes on the margins of the lumbosacral vertebral bodies, adjacent to intervertebral spaces.

Bone diseases

Fractures

Traumatic limb injuries commonly occur when individual sheep attempt to escape from handling pens (Fig. 5.59), following over-exuberant use of dogs, or as a result of fighting between rams. It must be borne in mind when examining potentially affected animals that these lesions are extremely painful. The diagnosis may be supported by radiography of the affected limb.

Fractures distal to the carpus or tarsus generally respond well to treatment involving careful alignment of the displaced bones, application of padding to prevent formation of pressure sores, and use of a lightweight external cast or splint. It is much more difficult to provide rigid immobilisation of proximal limb fractures without resorting to the use of internal fixation, which is seldom economically justifiable in commercial sheep.

Metabolic bone diseases

The so-called metabolic bone diseases are associated with imbalances or deficiencies in vitamin D, calcium and/or phosphorus. Sheep can tolerate prolonged mineral and vitamin D deficiencies, so clinical disease outbreaks are rare. Calcium levels in roughages tend to be high, and deficiency problems are most commonly encountered on grain-based diets, with no mineral supplementation. Conversely, cereals and

concentrate feeds have relatively high levels of phosphorous, while forage levels, depending on the phosphorus content of the soil, are low. Vitamin D is either eaten with forage or produced by ultraviolet irradiation of the skin. Forage-based diets may have variable vitamin D levels, and production in the skin by ultraviolet irradiation may be low in housed animals or outside during the short day length of winter.

The clinical signs associated with diseases of bone mineralisation include lameness, stiffness of gait, a tendency to spend prolonged periods lying down with consequent reduced feed intake, and unusually high incidences of fractures of long bones.

Osteoporosis

Osteoporosis describes a reduction in bone density due to an imbalance between formation and resorption, in favour of increased resorption. The remaining bone is chemically normal, but becomes porous and fragile. Osteoporosis is usually seen in grazing lambs, associated with severe gastrointestinal parasitism or under-nutrition, but occasionally occurs in adult sheep as a result of prolonged calcium mobilisation (lactation) and specific deficiencies in calcium, phosphorus or copper. Osteoporosis is usually undetected, because the shape of the bones is unaltered and affected sheep are not lame unless the bones fracture. Osteoporosis also occurs in cases of chronic lead or fluoride poisoning.

Rickets

Rickets is a disease of growing animals, caused by a deficiency in either vitamin D or phosphorus, resulting in defective mineralisation of physeal cartilage or newly formed osteoid. Rickets is characterised by gross lesions at sites of rapid bone growth, such as the metaphyseal and epiphyseal regions of long bones and the costochondral junctions, leading to abnormal curvature of long bones.

Osteomalacia

This refers to the equivalent disease to rickets in older animals, where the growth plates have closed, resulting in gradual bone weakness due to impaired mineralisation of osteoid during the continuous process of bone remodelling.

Copper deficiency

This results in increased bone fragility because the copper enzyme, lysyl oxidase, is involved in cross linkage of bone collagen molecules.

The diagnosis of metabolic bone disease is based on feeding history and clinical signs. Radiography and biochemistry tend to be unhelpful. Postmortem examination is useful to rule out other problems, and to provide bone samples for analysis of density, as measured by their ash content, and the mineral content. Dietary analysis of calcium, phosphorus and vitamin D levels can be important.

Sheep with gross skeletal abnormalities should be humanely killed, while less severely affected cases may respond slowly to correction of dietary deficiencies or imbalances.

Bone tumours

Bone tumours are very rare in sheep. Large chondromas and chondrosarcomas are occasionally seen on the sternocostal complex of aged ewes.

Chapter 6

Neurological Diseases

Sheep can be affected by various specific neurological diseases. Outbreaks of neurological diseases such as louping ill cause obvious economic loss, while the cumulative effect of individual cases of listeriosis, sarcocystosis, gid and polioencephalomalacia can become significant over time. Other neurological diseases, such as brain abscessation, only occur sporadically. However, unless the cause of the first single case of neurological disease within a flock is known, it is uncertain whether or not action is required to expedite prevention of subsequent cases. Established cases of neurological disease generally respond poorly to treatment. The prompt and accurate diagnosis of individual cases of neurological disease is therefore important.

Neurological examination of sheep

Sheep breed, age and feeding management predispose to certain neurological diseases, so clinical diagnosis always starts with a relevant disease history. Neurological examination should only be performed as a part of a full clinical examination, in order to differentiate between specific neurological diseases, and generalised diseases, such as pregnancy toxaemia, with associated central nervous signs. However, in some cases abnormalities in other organ systems may arise as a consequence of central nervous system disease, for example digestive or respiratory signs may be due to acid/base disturbances, or pyrexia may be due to exaggerated muscle activity caused by primary neurological disease.

For most central nervous system diseases, lesions can be accurately localised within the brain or spinal cord, providing a useful, definitive diagnosis. Most sheep brain diseases arise due to lesions in the cerebrum, cerebellum, brainstem or vestibular region. Lesions in these areas of the brain result in specific syndromes; however, in some cases the situation is complicated by common nervous pathways within the brain, causing overlap between syndromes.

Diseases causing cerebral lesions

Cerebral disease results in: an altered mental state, with clinical signs including severe depression, dementia, hyperaesthesia, opistotonus, or head pressing; abnormal behaviour, with clinical signs such as compulsive walking, circling and constant jaw movements; and visual impairment. Cerebral disease may be generalised, as occurs in cases of polioencephalomalacia, bacterial meningitis and pregnancy toxaemia, or

Sheep Flock Health: A Planned Approach. Neil Sargison
 ISBN: 978-1-4051-6044-5

Cerebrospinal fluid analysis

Collection and analysis of cerebrospinal fluid (CSF) can be useful to support the diagnosis of inflammatory disease involving the leptomeninges, such as meningoencephalitis. CSF can be collected from the lumbosacral site of live, conscious sheep. The approach is the same as that used for lumbosacral spinal analgesia, except that the needle point is slowly and carefully advanced to puncture the subarachnoid space, so that CSF wells from the hub of the needle. Ideally, no more than 1 ml of CSF should be collected by free flow. Alternatively, the CSF can be collected into a 2 ml syringe, using minimal negative pressure so as to avoid blood contamination of the sample (if blood contamination occurs, the sample should be left to stand, to separate the CSF from the blood).

CSF can be analysed for protein concentration and white blood cells. The normal range for CSF protein concentration is <0.4 g/L, while normal CSF contains less than 0.012 x 10^9/L white cells, which are predominantly lymphocytes. Raised protein concentrations and white cell counts occur with inflammatory disease. Normal CSF is clear and does not form a stable froth when shaken. A useful indication of raised white cell counts and elevated protein concentration can be provided in the field, by observation of cloudiness and formation of a stable froth on the surface of the shaken sample respectively (Fig. 6.1). CSF can also be analysed for magnesium or glucose concentrations if required.

CSF can be collected from the lumbosacral or cerebellomedullary cistern during the postmortem examination of recently dead sheep. Cisternal samples can be collected using a 2 inch needle, inserted through the foramen magnum via the midpoint of a triangle defined by palpation of the point of the nuchal crest and wings of the atlas, while the head is flexed. In the absence of a focal compressive lesion in the spinal cord there is no difference between the protein and cellular composition of cisternal and lumbosacral CSF samples.

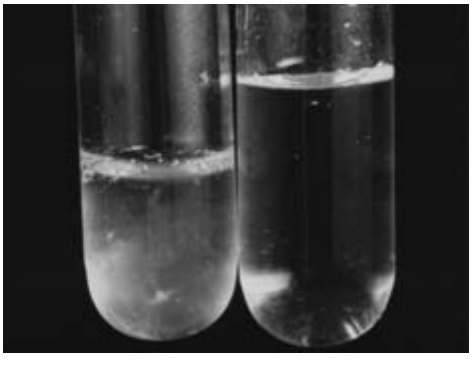


Fig. 6.1 A cloudy CSF sample with a stable froth on the surface (*left*) and apparently normal CSF sample (*right*) for comparison.

localised, as in cases of abscessation or coenurosis. Focal lesions result in unilateral blindness, but with preservation of the pupillary light reflex, compulsive circling and head deviation, generally towards the side of superficial lesions and away from deeper lesions, and so can be localised to one side of the cerebrum. About 90 percent of the nerve fibres from the eye to the cerebrum cross over in the midline at the optic chiasma. Therefore, most sheep that are blind in the right eye have a lesion in the left cerebral hemisphere, and vice versa. Blindness can be assessed using the menace reflex, or by blindfolding one eye at a time.

Polioencephalomalacia

Polioencephalomalacia (cerebrocortical necrosis; CCN) is a common neurological disease of both adult sheep and weaned lambs, but is uncommon in lambs less than 12 weeks old. (Polio- refers to the grey matter of the brain which is comprised of the nerve cell bodies, as opposed to leuco- which refers to the white matter, comprised of the nerve cell processes. Malacia refers to tissue necrosis.) Most cases occur about 2 weeks after dietary change, such as movement to improved grazing, overnight yarding before anthelmintic drenching, or following periods when animals do not feed due to persistent heavy rain and low temperatures.

Clinical signs

The clinical signs of polioencephalomalacia include isolation from the flock, a high head carriage, a high stepping gait due to blindness, aimless wandering and in some cases bouts of head pressing; progressing over a period of 12 to 24 hours to sternal recumbency and depression, with periodic bouts of hyperexcitation and backwards flexion of the neck (Fig. 6.2). Upward and inward rotation of the eyes is observed in some cases. Untreated cases progress to a state of lateral recumbency, with periodic convulsions (Fig. 6.3) leading to death within a few days.

Diagnosis

The diagnosis of polioencephalomalacia is reliably based on the clinical signs and response to appropriate treatment, but can be confirmed by postmortem examination. Affected areas of the cerebral cortex fluoresce when cut sections are viewed under ultraviolet light. The postmortem diagnosis is confirmed by histopathology, showing vacuolation and cavitation, with associated neuronal necrosis within the cerebral cortex.

Treatment and management

The clinical signs of polioencephalomalacia are caused by damage to the superficial tissue of the brain. This brain damage is due to altered glucose metabolism, which usually results from an induced deficiency of vitamin B_1 (thiamine). Vitamin B_1 has

Fig. 6.2 Recumbency, pelvic limb extension and backwards flexion of the neck in a 4-month-old lamb with cerebrocortical necrosis. The treatment response at this stage of the disease is reasonable.

Fig. 6.3 Lateral recumbency, depression and periodic convulsions in an advanced case of cerebrocortical necrosis. The treatment response at this stage of the disease is generally poor.

Fig. 6.4 Typical signs of cerebrocortical necrosis in a newly-lambed Greyface ewe.

Fig. 6.5 The same ewe as in Fig. 6.4, 7 hours after treatment.

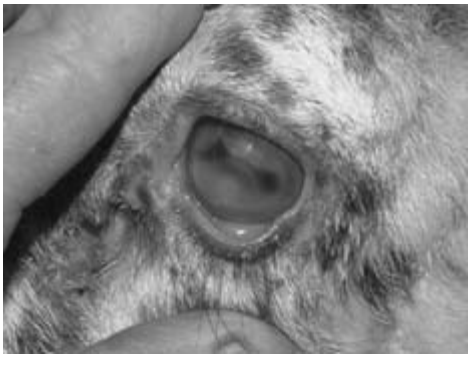


Fig. 6.6 Exposure keratitis associated with absence of eye protection reflexes in the ewe shown in Fig. 6.4.

an essential role in glucose metabolism. Vitamin B_1 is normally synthesised in the rumen, but overgrowth of thiaminase-producing bacteria in the rumen, which can occur following alterations to the ruminal pH and microflora associated with changes in diet or feeding behaviour, destroys the vitamin B_1.

The treatment response to intravenous injections of 10 mg/kg vitamin B_1 (5 ml of the 100 mg/ml product for a 50 kg sheep) and soluble corticosteroids (1 g/kg dexamethasone) is good, provided that affected animals are identified promptly (Figs 6.4, 6.5 and 6.6). Further twice-daily intramuscular injections of vitamin B_1 for 3 days are usually required. Vitamin B_1 on its own is not always available, in which case injections of multivitamins which include vitamin B_1 are required. The dose rate for these products must be adjusted to ensure administration of sufficient vitamin B_1.

Affected sheep should be housed in a quiet and comfortable pen and propped in sternal recumbency if necessary. In many cases sheep are able to stand and eat within 24 hours of commencing treatment, but they may remain blind for a few weeks. It is important that they can find water during this period and that they are kept in a safe area, for example to avoid entanglement in electric fences or falling into streams. Sheep which do not respond to treatment within 2 days should be humanely killed.

Sulphur toxicity

A disease with clinical similarities to polioencephalomalacia has been reported, but without signs of hyperaesthesia, nystagmus and backwards flexion of the neck, and

Fig. 6.7 Head-pressing behaviour in a cobalt-deficient 7-month-old Mule ewe lamb.

with no treatment response to vitamin B_1 injections. The disease has been seen in housed, intensively managed lambs fed on a concentrate ration containing ammonium sulphate as a urinary acidifier, and has been putatively associated with a direct toxic effect of sulphur or sulphur metabolites on the brain. The pathology of sulphur toxicity differs from that of polioencephalomalacia in that there are widespread, clearly defined areas of malacia involving the cerebrum, thalamus and mid-brain. There is no recognised treatment for sulphur toxicity and prevention relies on ensuring that concentrate diets are carefully formulated.

Hepatic encephalopathy

Clinical signs including a lack of menace response, sudden onset depression progressing to stupor, fine muscle fasciculations over the head, neck and ears, ataxia, and head pressing (Fig. 6.7) are reported periodically in the UK, Scandinavia and Australia, affecting up to 5 percent of ill-thrifty cobalt-deficient lambs. The clinical signs are first seen within 24 hours of an improvement in diet, for example following introduction of hill lambs to carbohydrate-rich concentrate feeding. Similar clinical signs have also been seen in individual cases of copper toxicity. Some of these clinical signs are consistent with a diagnosis of polioencephalomalacia, although spontaneous nystagmus, dorso-medial strabismus and hyperaesthesia are absent and the affected animals do not respond to treatment with intravenous injections of vitamin B_1 and dexamethasone. Most affected lambs become recumbent and die, or require euthanasia for welfare reasons, while a few recover slowly over a period of several weeks.

Hepatocytic degeneration and necrosis, referred to as ovine white liver disease, is a common finding in cobalt-deficient lambs. The neurological clinical signs are those of an hepatic encephalopathy, which can be explained by loss of hepatic urea cycle enzymes and secondary hyperammonaemia, although a direct influence of cobalt or vitamin B_{12} deficiency on brain energy metabolism in the development of lesions cannot be excluded.

The postmortem diagnosis of hepatic encephalopathy is supported by the histopathological findings of large droplet fatty change and hepatocellular degeneration in the liver and symmetrical vacuolation and status spongiosus of the neuropil in the brain.

Brain abscessation

Brain abscessation occurs sporadically in 2- to 6-month-old lambs (Figs 6.8, 6.9 and 6.10), probably having arisen following neonatal bacteraemia. Despite the fact that

Fig. 6.8 Recumbency and depression with the head turned towards the flanks in a 4-month-old lamb with a deep cerebral abscess.

Fig. 6.9 Having circled towards its left, this 4-month-old lamb with a superficial left cerebral abscess is standing, leaning against a barrier for support.

Fig. 6.10 Abnormal sitting posture with bilateral blindness and backwards flexion of the neck in a 4-month-old lamb with a large, deep cerebral abscess.

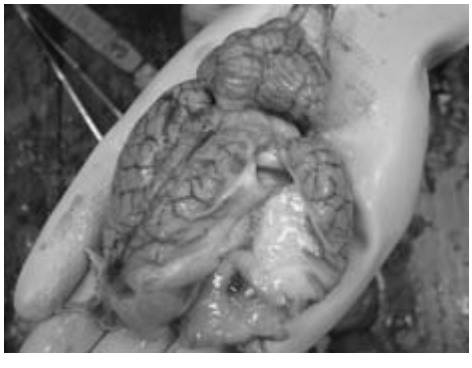


Fig. 6.11 A large, superficial abscess in the left cerebral hemisphere of a 4-month-old lamb.

abscesses increase progressively in size, clinical signs are often sudden in onset. The clinical signs result from the focal, space-occupying nature of the lesion rather than from the inflammatory response. CSF analysis usually shows an increased white cell count and a slight increase in protein concentration. Most abscesses occur in one cerebral hemisphere (Fig. 6.11). The response to antibiotic and inflammatory treatment is poor, so affected lambs should be humanely killed once the clinical diagnosis has been made, to prevent further suffering.

Abscessation of the pituitary body (basillar empyema), with clinical signs including depression, ataxia, head pressing, dysphagia and blindness, has been reported in adult sheep (Fig. 6.12). Some of these signs occur because the trigeminal nerves are closely associated with the pituitary gland.

Diseases causing cerebellar lesions

The cerebellum coordinates voluntary movement, so disease results in rigid (spastic) and jerky movement. Affected sheep often have a wide-based stance (Fig. 6.13),

Fig. 6.12 Blindness, loss of pupillary light reflexes, depression and dysphagia in a Texel ewe with a pituitary abscess pressing on both trigeminal nerves.

Fig. 6.13 An abnormal wide-based stance associated with a cerebellar lesion in a Bluefaced Leicester ram.

abnormal high-stepping gait (hypermetria), incoordination (ataxia) with preservation of normal muscle strength, and jerky head movements (intention tremors).

Scrapie

Scrapie is a politically important disease, due to: national stigma associated with the presence of disease; concern arising from its similarities to bovine spongiform encephalopathy; the fact that large sums of money are spent on scrapie research; and the effects of the disease on the international trade in live sheep. The following brief summary reflects the fact that scrapie does not rank highly as an economically important production-limiting disease. The author's experience of scrapie does not extend beyond the investigation and management of naturally occurring clinical cases, and numerous, better-informed scientific references to the subject are widely available.

Scrapie is a primary transmissible spongiform encephalopathy involving the cerebellum and cerebrum of sheep and goats, which has been recognised in the UK since the mid-eighteenth century. In other species, scrapie-contaminated material has been suggested as a cause of neurological disease such as transmissible mink encephalopathy and possibly bovine spongiform encephalopathy. Human Creutzfeldt-Jakob disease and kuru, and a chronic wasting disease of deer and elk in the Rocky Mountains of the USA, are similar but unrelated diseases.

Prior to the disease becoming notifiable in January 1993 and the subsequent implementation of the National Scrapie Plan, scrapie was known to be widespread in the UK. In a few endemically infected and genetically susceptible flocks it was an important cause of economic loss. About 250 cases of scrapie are now diagnosed annually in the UK, but the disease may be under-reported due to associated stigma and fear of future market restrictions.

Scrapie probably originated in Spanish Merino flocks and then spread to Europe and North America. Although the disease has on occasions been introduced to Australia and New Zealand, prompt identification and slaughter of imported sheep has kept these countries scrapie-free.

Clinical signs

The clinical signs of scrapie in individual sheep are non-specific and variable. Clinical signs are seen rarely in sheep between 1 and 3 years old, most commonly between

3 and 5 years old and with decreasing frequency with increasing age thereafter. While the onset of clinical signs is insidious, the disease is always progressive; the time from appearance of clinical signs to recumbency and death varying from 1 week to several months.

Affected sheep are ill thrifty. Early non-specific behavioural changes such as depression, hyperaesthesia and refusal to be gathered may only be recognised by those with previous experience of the disease. As the disease progresses, signs of pruritus, incoordination and weight loss may develop, either alone or in combination. Vision and pupillary light reflexes are normal.

Pruritic sheep may rub repeatedly against fences or bite at their shoulders and flanks. Patchy wool loss develops over the flanks and tail head and the underlying skin and wool regrowth may become black pigmented. There may also be evidence of self-excoriation to the skin of the face and tail head. Sheep may demonstrate a nibble reflex if scratched or rubbed over the tail head or rump, with the forelimbs extended, pelvic limbs flexed and neck extended dorsally with the ears drawn caudally and the upper lip retracted, while manically nibbling. However, this response is not pathognomonic, also occurring with parasitic mite infestations.

During the early stage of clinical scrapie, sheep may collapse into a narcoleptic state whenever they are stressed, for example during gathering. Affected sheep become sternally recumbent with their neck extended and head held against the ground and cannot be forced to stand. This state is maintained for several minutes after which sheep spontaneously regain their feet.

Sheep with scrapie demonstrate variable degrees of cerebellar dysfunction, with signs of incoordination with forelimb hypermetria and pelvic limb ataxia (Fig. 6.14), and a wide-based stance at rest (Fig. 6.15). When chased, they may stumble and collapse. In the terminal stages of the disease, sheep are unable to stand. These gait abnormalities are most obvious when affected sheep are made to trot downhill or turn acute angles, when hopping with both pelvic limbs is frequently observed.

Signs of head pressing, head tremors, tooth grinding, ruminal stasis and abomasal impaction are sometimes reported. During the terminal stages of the disease, sheep show poor appetite, become emaciated and spend increasing periods in sternal

Fig. 6.14 Advanced signs of scrapie in a Suffolk ewe, including poor body condition, an ataxic gait with dragging of the toe of the left pelvic limb, and patchy areas of wool loss over the hindquarters.

Fig. 6.15 Emaciation and a wide-based stance in a Cheviot ewe showing clinical signs of scrapie.

recumbency. Cases of scrapie appear to be over-represented in the Cheviot, Suffolk, Shetland and Swaledale breeds.

There is no treatment for scrapie, so affected sheep should be humanely killed to prevent further suffering as soon as the diagnosis is suspected.

Diagnosis

The clinical signs associated with scrapie are often vague or non-specific. There is no confirmatory ante-mortem laboratory diagnostic test and gross postmortem findings are normal. The diagnosis is supported by histopathological identification of neuronal vacuolation, glial reaction and astrocytosis in the medulla, pons, midbrain and spinal cord of freshly dead sheep. Unfortunately, the distribution and intensity of the lesions are variable and the histopathological diagnosis must also be differentiated from spontaneous neuronal vacuolation of old age and postmortem autolysis. Confirmation of scrapie relies on electron-microscopic identification of scrapie-associated fibrils in centrifuged extracts of brainstem or use of molecular methods to detect abnormal prion protein.

Putative cause and transmission

Scrapie is caused by an infectious agent, the precise nature of which remains unknown. The infectious agent is smaller than conventional viruses, does not appear to elicit a specific host antibody response and cannot be identified serologically. It appears to be capable of strain variation and mutation and therefore to have a genome independent of the host. It is capable of survival in the environment for up to 2 years and survives conventional sterilisation methods, involving boiling, autoclaving, ultraviolet light, ionising radiation, formalin treatment, freezing, drying and organic solvents.

Various hypotheses have been proposed on the nature of the scrapie agent. It is currently believed to be an abnormal, distorted, glycosylated protein molecule, referred to as an infective prion, that has no associated nucleic acid, and is therefore not live. Prions are found in normal healthy brain tissue, those of different sheep species having different amino acid sequences. Infective prions differ from normal prion proteins by having a distorted three-dimensional shape. Whenever these infective prions find their way to the brain of a susceptible host, they act as a template for the formation of further distorted prions, resulting in clinical disease. The slow rate of this process reflects the slow onset of clinical disease. Ultrastructural examination of brain tissue from scrapie-affected sheep has shown abnormal clusters of material referred to as scrapie-associated fibrils, which are believed to be composed substantially of distorted prion proteins.

In naturally occurring scrapie, both lateral spread of infection from a contaminated environment and horizontal spread of infection from pregnant ewe to foetus are believed to be important.

While pre-partum and periparturient maternal transmission of scrapie infection via the foetal membranes is important, the probability of clinical disease in offspring decreases when lambs are removed from their dams at an early age, suggesting lateral transmission by additional routes. The placenta appears to be the main source of infection, causing contamination of the lambing environment. Infective prions are

resistant to breakdown in the digestive tract. Following ingestion, infection first establishes in the tonsils, retropharyngeal and mesenteric lymph nodes, before moving to the brain. In most cases the incubation period is 3 to 5 years, hence the peak incidence in 3- to 5-year-old sheep. Cases in older sheep or goats may be due to infection as adults or associated with genetic factors.

The question of whether or not scrapie can infect the blastocyst is of paramount importance when considering the merits of embryo transfer techniques for the export of scrapie-free genetic material. Large-scale studies conducted in the USA in 2001 suggested that scrapie was not transmitted to offspring via the embryo, although earlier UK studies were less convincing, possibly because of scrapie contamination following embryo transfer. The question about the absolute safety of washed embryos with regard to freedom from scrapie is therefore unresolved.

Genetic aspects

The incubation period of scrapie is influenced by the infective dose, the strain of the scrapie agent and the genotype of the sheep host. Genetic selection studies in Cheviot, Herdwick and Swaledale sheep have been used to create lines of sheep which are susceptible or resistant to experimental scrapie infection. These studies suggest that scrapie is controlled by a single major gene, referred to as Sip (short incubation period), with two alleles (sA and pA). Sip sA homozygotes have the shortest incubation period, Sip sApA heterozygotes have a longer incubation period and Sip pA homozygotes have extremely long incubation periods and are relatively resistant to clinical scrapie. Further studies in Suffolk sheep indicate that the genetic control of natural scrapie infection is similar.

Studies in sheep and mice suggest that, following infection, the scrapie agent develops and persists in extraneural tissues including the spleen and lymph nodes and clinical scrapie only develops following spread to the central nervous system. In mice, the Sinc (scrapie incubation period) gene controls multiplication of scrapie in the central nervous system, but not in the lymphoreticular system. There is evidence that the Sip gene in sheep acts in the same way, therefore, while clinical disease may not develop in Sip pA homozygotes, these animals may be carriers of the disease in extraneural tissues and a source of environmental contamination or transplacental infection.

Normal prion protein is coded for by a single PrP gene, which is present in all mammalian species. Specific polymorphisms in the human PrP gene are linked with increased susceptibility to familial Creutzfeldt-Jakob disease. There is strong evidence that the supposed Sip gene is the same as, or closely linked to, the PrP gene and that polymorphism in the sheep PrP gene has a fundamental role in the pathogenesis of scrapie. The incidence of natural scrapie in sheep is believed to be associated with polymorphisms of the PrP gene, particularly at codons 136 encoding the amino acids alanine (A) or valine (V), 154 encoding arginine (R) or histidine (H), and 171 encoding glutamine (Q) or histidine (H). Genotyping of these three alleles theoretically enables 36 permutations, although only 15 occur in sheep. Only 10 permutations occur in Cheviots and 7 in Suffolks. The PrP allele encoding V at codon 136 confers an extremely high risk of scrapie, but this allele is rare in Suffolk sheep.

The susceptibility of sheep with different genotypes (annotated as 136, 154, 171/136, 154, 171) to transmissible spongiform encephalopathies can be ranked as types 1 to 5:

1 Genetically most resistant: ARR/ARR
2 Genetically resistant: ARR/ARQ, ARR/ARH, ARR/AHQ
3 Little genetic resistance: ARQ/ARQ, AHQ/ARH, ARH/ARH, AHQ/AHQ, ARQ/AHQ, ARQ/ARH
4 Genetically susceptible: ARR/VRQ
5 Highly susceptible: VRQ/VRQ, ARQ/VRQ, ARH/VRQ, AHQ/VRQ.

While there are subtle differences between breeds, and individual cases of scrapie may periodically occur in resistant genotypes, sheep which are homozygous for Q at codon 171 are generally susceptible and sheep carrying one allele encoding for R at codon 171 are mostly resistant to scrapie. Thus selection of parents that are homozygous for R at codon 171 confers a low risk of scrapie in their progeny, while the use of parents with the $V^{136}R^{154}Q^{171}$ genotype confers a high risk. Other genotypes confer an intermediate risk. These principles underpin the National Scrapie Plan, which was introduced in 2001 to reduce the risk of transmissible encephalopathies in UK sheep. The scheme is primarily aimed at genotyping breeding rams, although the selection process has been accelerated in some flocks by ewe genotyping. The rational scientific approach to sheep disease control that has been used in the National Scrapie Plan appears to have been effective, although caution must be exercised to ensure that other positive attributes are not lost when removing transmissible spongiform encephalopathy susceptible genotypes from the sheep population.

In addition to genetic selection for resistance to scrapie, control in infected flocks relies on the culling of clinical cases and of their maternal relatives. This policy requires reliable identification and detailed records for several generations. The use of different lambing fields in subsequent years may reduce pasture contamination.

Eradication by slaughter has been unsuccessfully attempted in Iceland and the USA. In the absence of a serological diagnostic test in the live animal, a whole-flock eradication policy is required. Unfortunately, persistent environmental contamination and inability to guarantee scrapie-free status in replacement stock makes freedom from scrapie following re-population impractical in most circumstances.

Diseases causing vestibular lesions

The vestibular region of the brain is concerned with environmental orientation, involving the position of the eyes, head, body and limbs with respect to movement (Fig. 6.16). Vestibular disease causes a head tilt (Fig. 6.17) on the affected side (ipsilateral), abnormally jerky eye movements (nystagmus) when the head is moved quickly horizontally or vertically, and sometimes circling. Peripheral vestibular lesions are sometimes associated with facial nerve paralysis (Horner's syndrome), because the facial nerve passes close to the middle ear.

Vestibular disease

Unilateral infection of the peripheral vestibular system occurs sporadically in sheep of all ages. Most cases in growing lambs are associated with middle ear infection (otitis media) and arise following ascending infection of the eustachian tube. Vestibular disease may also arise following haematogenous spread from other foci of infection,

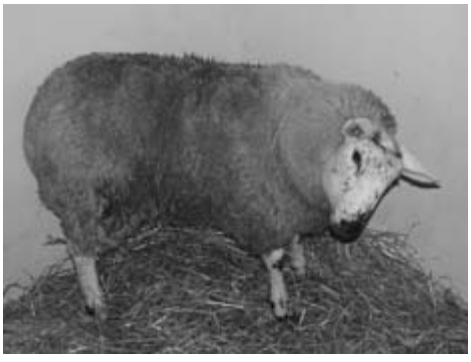


Fig. 6.16 Abnormal limb position and a head tilt in a Cheviot cross ewe with vestibular disease.

Fig. 6.17 Head tilt associated with a vestibular abscess in a 9-month-old Scottish Halfbred wether lamb.

for example associated with fighting injuries in rams. Non-specific bacteria, including *Pasteurella* spp., *Streptococcus* spp. and *Arcanobacterium pyogenes* have been isolated from vestibular lesions.

Provided that vestibular disease is recognised and diagnosed promptly, the treatment response to a 5-day course of injections of procaine penicillin 44 000 iu/kg is good.

Diseases causing brainstem lesions

The brainstem contains many of the cranial nerve nuclei, so disease results in multiple cranial nerve deficits, for example:

- Difficulty in prehension and mastication with cud retention. A drooped lower jaw or asymmetric jaw closure, tongue protrusion, facial analgesia and drooling of saliva (trigeminal nerve nucleus – V).
- Nystagmus and head deviation towards the side of the lesion and circling or falling in the same direction (vestibulo-cochlear nerve nucleus – VIII).
- Drooping of the upper eyelid (ptosis) and loss of menace response, ear drooping and decreased lip tone (trigeminal nerve nucleus – V and facial nerve nucleus – VII).
- Medial squint (strabismus) on the ipsilateral side to the lesion (abducens nerve nucleus – VI).

Listeriosis

Listeriosis is a common neurological disease of 18- to 24-month-old sheep, which also occurs sporadically in younger lambs, older sheep, cattle and horses. Outbreaks of listeriosis in sheep typically involve 2 percent of sheep in silage-fed flocks over a period of a few weeks, although, prevalences of 10 percent have been reported. Cases of listeriosis occur occasionally in young lambs which have had no access to silage.

Fig. 6.18 Poor-quality big bale silage presents a particularly high risk for listeriosis.

Fig. 6.19 Aerobic deterioration and subsequent proliferation of *L. monocytogenes* is minimal in a well-managed silage pit, where the crop is removed with a sharp block cutter and the cover is withdrawn by no more than is necessary to gain access.

Cause

The bacterial cause of listeriosis, *Listeria monocytogenes*, proliferates in soil, faeces and rotting vegetation and can replicate at low environmental temperatures. *L. monocytogenes* can also tolerate the high temperatures which are sometimes generated during aerobic fermentation of poor silage. In anaerobic conditions, *L. monocytogenes* cannot survive below pH 5.6 but in poorly consolidated silage with some oxygen present it can survive at pH levels as low as 3.8.

Poor-quality big bale silage presents a particular risk for listeriosis (Fig. 6.18) because it is usually made later in the season than conventional clamp silage, from grass with lower sugar and higher dry matter contents. The combined effects of a high pH, due to inefficient lactic acid fermentation, inadequate consolidation and failure to totally exclude air from such silage, enable the proliferation of *L. monocytogenes*. These principles also apply to poorer-quality clamp silages. These conditions also favour the growth of certain moulds, so mouldy silage generally presents a high risk for listeriosis.

Entry of air into baled silage during storage permits the slow growth of aerobic bacteria and a slow rise in pH allowing *L. monocytogenes*, already present from soil contamination, to multiply. In tightly packed, hard-centred bales, or well-consolidated clamp silage (Fig. 6.19), aerobic deterioration tends to be superficial, being confined to the site of entry of air, but in soft-centred bales, or poorly consolidated clamp silage, such as whole-crop silages, the aerobic deterioration and subsequent multiplication of *L. monocytogenes* may occur throughout the bale or clamp.

Secondary fermentation of previously stable silage, associated with poor management of the clamp face (Fig. 6.20) and failure to regularly remove stale feed from bunkers or feed rings (Fig. 6.21), also enables proliferation of *L. monocytogenes*.

The feeding of listeria-contaminated silage to sheep can result in several clinical diseases. The most commonly encountered is meningoencephalitis, but anterior uveitis (silage eye), abortion and neonatal septicaemia are sometimes seen. It is unusual for different forms of listeriosis to occur simultaneously in a flock.

Fig. 6.20 Substantial anaerobic deterioration and proliferation of *L. monocytogenes* can occur at a poorly managed silage face, where use of the crop is not rapid.

Fig. 6.21 Secondary fermentation of previously stable silage can occur in bunkers or ring feeders, where stale feed can accumulate.

Fig. 6.22 Compulsive attempts to circle towards the left in an 8-month-old lamb with listeriosis.

Fig. 6.23 Continuous drooling of saliva associated with paralysis of the trigeminal nerve in a 2-year-old Scottish Blackface ewe.

Clinical signs

Listerial meningoencephalitis is caused by formation of microabscesses in the brainstem. The clinical signs vary in severity depending on the degree and precise location of the abscesses, and include fever, anorexia, profound depression and various specific neurological signs. When the condition was first described in sheep it was referred to as circling disease (Fig. 6.22) because of involvement of the vestibulocochlear nucleus.

Affected animals frequently continuously drool saliva (Fig. 6.23), have unilateral impaction of food between the gums and cheek teeth (Fig. 6.24) and have unilateral drooping of the eyelid, lip and ear due to trigeminal nerve paralysis. Some sheep show deviation of the head (Fig. 6.25) and walk compulsively in circles. There is unilateral lack of blink response, often with associated exposure keratitis. Severely affected animals may become frenzied or comatose and die in 10 to 14 days.

Fig. 6.24 Drooping of both ears and impaction of food between the cheeks and teeth in a 6-month-old lamb with listeriosis.

Fig. 6.25 Recumbency, head deviation and unilateral drooping of the ear and upper lip in a 7-month-old lamb with listeriosis.

Diagnosis

Listerial meningoencephalitis can be differentiated from scrapie and pregnancy toxaemia by CSF examination, showing elevated protein concentrations of 0.8 to 4.0 g/L and raised counts of large mononuclear white cells. The diagnosis of listerial meningoencephalitis can be confirmed by postmortem histological identification of microabscesses in the brainstem and bacterial culture of *L. monocytogenes.*

The route of infection is thought to be via abrasions in the mouth and ascending spread to the brain along the trigeminal nerves, thus the incidence of infection is highest in 18- to 24-month-old animals as permanent cheek teeth are erupting. Most cases occur 4 to 6 weeks after feeding of *L. monocytogenes*-contaminated silage.

Management

When the clinical signs are recognised early, treatment with intramuscular injections of 100 000 iu/ml of procaine penicillin, twice daily for 5 days, is effective in about 30 percent of cases. The response to successful antibiotic treatment is slow and sheep require careful husbandry during the protracted recovery period, including hand feeding of soft food and water. Control is aimed at ensuring that only good-quality silage is fed to sheep (Fig. 6.26).

Fig. 6.26 The incidence of listeriosis is generally low when well-stored, good-quality silage is fed and removed after 24 hours if not eaten.

Diseases causing spinal cord lesions

Spinal cord lesions are commonly seen in rams during the autumn, caused by traumatic injury to the cervical vertebrae resulting from head butting between unfamiliar sheep during the mating period. Infective lesions of the vertebral column resulting in spinal cord compression are common in young lambs, but also occur in older sheep. Spinal cord lesions are also seen with specific diseases, such as, sarcocystosis, visna and tumours (meningiomas and lymphosarcomas).

Lesions involving the spinal cord result in a corresponding lack of muscle contraction in response to pinching the skin between the digits (withdrawal reflex), percussion of tendons (tendon jerk reflexes) or stimulation of the skin of the back (panniculus reflex). The nature of the abnormal spinal reflex depends on the site of the lesion. Lesions close to the spinal reflex arc being tested interfere with the nerve supply to the effector muscle and result in flaccid paralysis, referred to as lower motor neuron disease. Lesions cranial to the reflex arc being tested interfere with normal inhibitory signals from upper motor neuron pathways and result spastic (stiff) paralysis, with an exaggerated response to stimulation. Thus, spinal lesions can be accurately localised by evaluation of the responses to stimulation of the thoracic and pelvic limb reflexes:

- Lesions involving the cervical spinal cord (C1–C6)
  - ataxia and weakness involving all four limbs
  - thoracic and pelvic limb reflexes are exaggerated
- Lesions involving the brachial region (C6–T2)
  - more severe ataxia and weakness in the thoracic than the pelvic limbs
  - abnormal wear on the toes of the thoracic limbs
- Lesions involving the spinal cord between the second thoracic and third lumbar vertebrae (T2–L3) (Figs 6.27 and 6.28)
  - normal thoracic limb function
  - ataxia and weakness in the pelvic limbs
  - pelvic limb reflexes are exaggerated
  - absence of panniculus reflex caudal to the lesion

Fig. 6.27 Normal thoracic limb function and pelvic limb weakness in an 8-month-old Texel lamb with a large spinal abscess between T4 and T5 (Fig. 6.28).

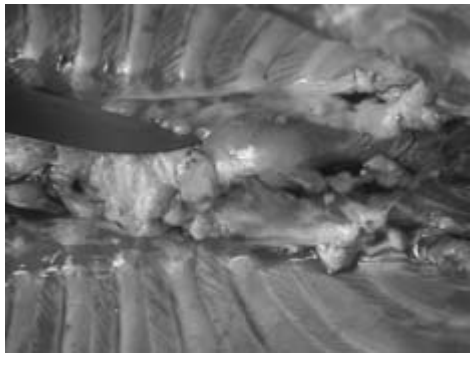


Fig. 6.28 Spinal swelling associated with a large spinal abscess between T4 and T5.

- Lesions involving the spinal column between the fourth lumbar and second sacral vertebrae (L4–S2)
  - flaccid paralysis of the pelvic limbs
- Lesions involving the sacral spinal column (S1–S3)
  - bladder distension and loss of tone to the rectum.

Lesions causing spinal cord compression result in elevations in lumbosacral CSF protein concentrations.

Sarcocystosis

Sarcocystis spp. are obligate two-host sporozoan parasites which can have a variety of carnivore species as their final host, but generally have a specific ruminant intermediate host. Sheep can be infected with four species of *Sarcocystis*, namely *S. tenella* (definitive host: canids), *S. arieticanis* (definitive host: dog), *S. gigantea* (definitive host: domestic cat) and *S. medusiformis* (definitive host: cat). Clinical disease has only been associated with the first two species. *S gigantea* causes thick cysts in the tongue and oesophagus, while *S. medusiformis* forms slender cysts in abdominal muscles, which are sometimes seen as incidental findings on postmortem examination. Sheep become infected by ingesting sporocysts from feed or pasture contaminated by carnivore faeces. Following the release of sporozoites from the intestinal tract, meronts multiply in the endothelium of arterioles and capillaries, before encystment in muscles and nerve cells. Eventually bradyzoites are formed within the tissue cysts, which are infectious to carnivores when ingested with uncooked sheep carcases.

Sarcocysts are found in heart (Fig. 6.29) and skeletal muscles of most UK sheep, but outbreaks of clinical disease only occur when the environmental challenge is high. On some farms, sarcocystis infection of multiple organs (Fig. 6.30) results in the sudden death of young, recently lambed ewes each year. On other farms, sarcocysts within the brain and spinal cord are a common cause of vague neurological disease including generalised tremors, compulsive nibbling and recumbency, and thoracic or

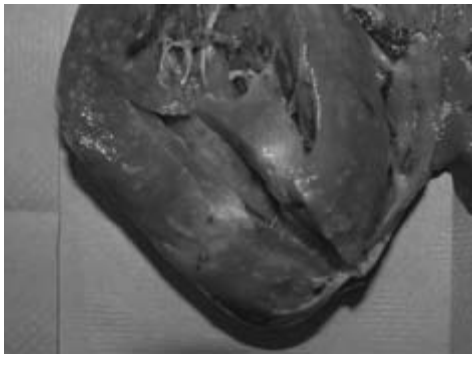


Fig. 6.29 Numerous white sarcocysts in the heart muscle.

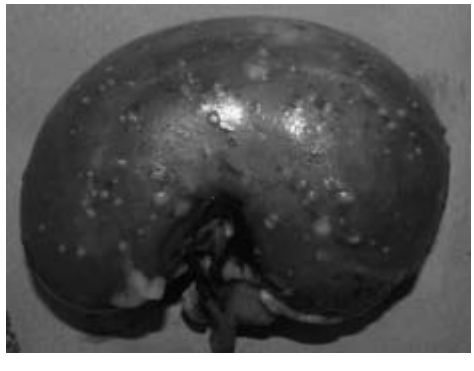


Fig. 6.30 Sarcocysts on the surface (and throughout the parenchyma) of a kidney of a recently lambed Mule gimmer that was found dead with postmortem evidence of generalised sarcocystosis.

Fig. 6.31 Vague neurological signs including paresis, recumbency and muscle tremors associated with *S. tenella* myeloencephalitis.

pelvic limb paralysis in 6- to 12-month-old lambs (Fig. 6.31). The presence of numerous sarcocysts in the muscles controlling the bladder has been associated with clinical signs similar to those seen with urolithiasis.

Definitive clinical diagnosis of sarcocystosis is difficult, because the signs of encephalomyelitis and spinal cord disease are not specific. Since most animals are exposed to *Sarcocystis* spp., almost all are seropositive and serological diagnosis cannot differentiate clinical disease from asymptomatic infection. Confirmation of the diagnosis of sarcocystosis depends on the histopathological findings including multifocal non-suppurative encephalitis associated with focal mononuclear meningitis and identification of meronts in the brain and spinal cord with the ultrastructural appearance of *Sarcocystis* spp. The sporozoan species involved can be confirmed by PCR analysis of tissue cyst material.

Anti-inflammatory and antibiotic treatment of clinical sarcocystosis is ineffective. Sarcocystosis is prevented by not allowing dogs access to uncooked sheep meat or carcases and preventing dog faecal contamination of pasture or bedding. Puppy faeces presents a particularly high risk to sheep. On many farms, any attempts to disrupt the sheep-to-dog life cycle of *Sarcocystis* spp. are hindered by public access and dog-walking activity.

Wobbler syndrome in Texel and Beltex sheep

Clinical signs varying from minor errors in placing the pelvic limbs and a wide-based stance to swaying on the pelvic limbs and severe difficulty when walking, consistent with low cervical spinal cord lesions, have been reported in UK Texel and Beltex sheep. The clinical signs have been associated with the presence of discrete, smooth, nodular, polypoid projections of adipose tissue, prolapsing from the intervertebral space between C6 and C7 and causing localised spinal cord compression. The cause of these lesions is unknown, but they are possibly associated with the short neck conformation of the affected breeds.

Visna

In the UK, visna is a very uncommon manifestation of maedi-visna virus infection, often appearing some years after the diagnosis of maedi in the flock. Visna essentially affects either the lateral ventricles of the brain, or the thoracolumbar spinal cord. The clinical signs are insidious and progressive.

The brain form of visna is manifest by insidious onset signs of a 5° to 10° head tilt and circling towards the affected side, while the initial clinical signs in the spinal cord form of the disease are a unilateral pelvic limb proprioceptive deficit with reduced flexion of the distal limb joints, progressing to pelvic limb paralysis, with characteristic knuckling of the fetlock joint and dragging of the point of the hoof along the ground.

The diagnosis of visna is supported by positive AGIDT serology and histopathological findings of lymphocytic infiltration and demyelination in affected areas of the brain or spinal cord.

Diseases of the peripheral nerves

Peripheral nerves are occasionally affected by trauma, or pressure caused by adjacent inflammation, as in the case of spondyloarthrosis or space-occupying lesions such as abscesses or tumours, resulting in paralysis of the innervated muscle. Bilateral peripheral neuropathy of the radial nerves occurs in cases of kangaroo gait.

Spastic paresis

A motor nerve disorder characterised by reflex hyperextension of the pelvic limbs, fine tremors and abnormal motor reflexes has been described in a Bluefaced Leicester shearling ram (Fig. 6.32) and probably occurs sporadically in large-framed ram breeds.

No abnormalities were detected when the animal was recumbent and at rest, but when disturbed slight postural trembling was seen. When standing, sudden auditory stimulation caused violent trembling, with abduction and caudal extension of both pelvic limbs. Palpation of the scrotum on both sides stimulated a marked extensor muscle reflex in the ipsilateral limb and whenever either pelvic limb was flexed, the ipsilateral cremaster muscle contracted, pulling the testis upwards towards the abdomen. Periodically the ram spontaneously passed small amounts of urine or ejaculated.

While the pathological cause of the disease and reasons for the unusual reflexes are unknown, the condition is similar to barn cramps which occur periodically in large-framed, mature bulls.

Fig. 6.32 Spastic paresis in a Bluefaced Leicester shearling ram.

Chapter 7

Skin and Eye Diseases

Sheep commonly suffer from a variety of skin diseases, many of which are serious welfare concerns and important causes of production loss. The presence of sheep skin disease is usually obvious, but it is nonetheless important to reach an accurate diagnosis to ensure that appropriate management is implemented. The investigation of skin disease should include establishment of a relevant disease history, assessment of the environment, clinical examination of individual animals and collection of appropriate samples.

The disease history should include information about:

- the proportion of the flock affected
- the production group and age of animals affected
- observation of pruritus or the presence of wool on fences
- ectoparasite control measures
- stock movements and introduction of sheep to the flock
- evidence of ill thrift or other diseases
- feeding and grazing management.

Assessment of the environment involves awareness of:

- recent feed availability and management
- recent weather conditions
- the time of year.

Clinical examination of individual animals should include:

- appraisal of rectal temperature, heart rate and mucous membranes to provide information about systemic disease or involvement of other organ systems
- observation for signs of pruritus, nibble reflex or paroxysms
- determination of the distribution of lesions over the body
- close examination for evidence of wool or hair loss
- determination of type of skin lesions present (for example: nodules, swellings, papules, tumours, pustules, vesicles, abscesses, ulceration, erosions, scaling, crusting, scabs, proliferation and bleeding)
- observation of wool changes such as: pigmentation, discolouration, loss of crimp, breaks and presence of exudate
- examination for grossly visible ectoparasites.

Further tests are often necessary to support a provisional diagnosis, for example:

- examination of lesions with a magnifying glass
- skin scrapes

Sheep Flock Health: A Planned Approach. Neil Sargison
 ISBN: 978-1-4051-6044-5

- impression smears
- bacterial culture
- potassium hydroxide preparations
- dermatophyte culture
- collection of blood samples for serology, haematology or biochemistry
- biopsy and histopathology.

Sufficient samples need to be examined to achieve an accurate diagnosis, while keeping laboratory costs in perspective.

Ectoparasitic skin diseases

The incidence of ectoparasitic skin diseases has risen sharply since the deregulation in 1992 of the previous sheep scab control measures. Failure to prevent, recognise or effectively manage ectoparasitic skin diseases frequently results in substantial production losses and poor animal welfare. The common ectoparasitic skin diseases of UK sheep are:

- sheep scab
- blowfly strike
- chewing lice
- headfly
- chorioptic mange.

However, it is important to recognise the presence of other uncommon ectoparasitic infestations, because these are important differential diagnoses for sheep scab or blowfly strike, and have the potential to become important in UK flocks.

Expensive, inappropriate treatment or management of ectoparasitic diseases is often associated with failure to identify accurately which ectoparasite is present. Furthermore, sheep often harbour more than one ectoparasite genus at the same time, for example, concurrent scab and chewing louse infestations, in which case inappropriate treatment is not only ineffective and costly, but may also select for acaricide or insecticide resistance in the unrecognised genus.

While there are differences in the clinical signs associated with different ectoparasitic infestations, confirmation of the diagnosis requires specific parasite identification. Larger parasites such as lice, ticks, keds and blowfly larvae can be seen with the naked eye, but mites require microscopic examination of material from the edges of any visible lesions.

Control of ectoparasitic diseases is not straightforward and attempts to manage different infestations with a single treatment are usually unsuccessful. Effective control strategies require knowledge of the ectoparasite life cycle, in particular whether it spends its entire life cycle on its sheep host, or has at least one free-living stage, and an understanding of the principles involved with the use of insecticides or acaricides.

Sheep scab

Sheep scab is a painful, highly contagious and rapidly progressive disease that affects all ages and production groups of sheep, caused by infestation with the superficial,

Fig. 7.1 This ram is clearly showing signs of discomfort associated with sheep scab.

non-burrowing mite *Psoroptes ovis* (Fig. 7.1). The disease occurs in most sheep-producing regions, with the notable exceptions of North America, Australia and New Zealand. Sheep scab usually occurs as a flock outbreak with up to 100 percent morbidity, although there are circumstances where clinical signs are seen only in individual animals, for example when untreated sheep are mixed with sheep which were recently treated with a residual acaricide, or following re-infestation of previously recovered animals. It is particularly important to establish an early diagnosis, because these cases are potential sources of infestation to the remainder of the group and to neighbouring flocks.

Sheep scab was believed to have been eradicated from Great Britain in 1952, but was re-introduced during the 1970s, allegedly through importations of sheep from Ireland. Subsequent national control measures included compulsory summer and autumn plunge dipping for one minute of all sheep in propetamphos, diazinon or flumethrin solutions; licensing of markets; and the statutory restriction of movement of sheep out of infected areas. These procedures effectively maintained the annual incidence of sheep scab at less than 100 outbreaks per year and the disease would probably have been eradicated from the UK if draconian measures had been enforced in this small number of problem flocks. Sheep scab control (Sheep Scab [National Dip] Order 1990) was deregulated in July 1992 and by autumn 1993 the disease was diagnosed in flocks throughout the UK, highlighting its highly contagious nature and the importance of sheep movements in its spread.

Unfortunately plunge dipping involves considerable animal handling stress, is expensive and raises concerns about operator safety and environmental contamination. Not surprisingly, therefore, plunge dippers are no longer used on many UK farms. Some sheep farmers rely on systemic endectocides for sheep scab control, but many no longer actively prevent the disease in their flocks. Consequently, there has been an appreciable rise in the incidence of sheep scab in the UK over recent years. In 2002, outbreaks of sheep scab occurred in about 50 percent of the flocks served by one veterinary practice in the south-east of Scotland, with new cases diagnosed during every month between November 2002 and May 2004. The prevalence of sheep scab was probably higher in the south-east of Scotland than elsewhere in the UK, associated with a high density of sheep flocks in the area, but if this pattern of disease was present throughout the UK, then more than 12 million sheep could have been infested with an ectoparasite capable of causing serious production loss and suffering.

Fig. 7.2 Tufts of wool on a fence and hedge demarcating the boundary between neighbouring farms. The potential role of fomites in the spread of sheep scab is obvious.

Fig. 7.3 Bacterial and fungal multiplication in serous exudate from the skin results in yellow staining of the proximal wool.

Fig. 7.4 Serous exudation over the lower neck and shoulders and patchy areas of wool loss over the shoulders and flanks.

Fig. 7.5 The fleece overlying the lower neck and shoulders becomes saturated with exudate, and discoloured as it accumulates dirt.

Clinical signs

Sheep scab is characterised by intense pruritus. Up to 90 percent of the affected group may be seen repeatedly rubbing their shoulders and flanks along the ground or against fences, foot stamping, clawing at their flanks, or biting their shoulders. Tufts of wool are often noticed stuck to fences and hedges (Fig. 7.2).

Early cases of sheep scab are characterised by serous exudation and occasional macular skin lesions. Circular, 1 to 5 mm diameter red sores are seen dorsally over the shoulders, lower cervical and upper thoracic regions, with staining of the proximal 1 mm of the overlying wool fibres (Fig. 7.3) and displacement of small tufts of wool.

As the disease progresses, the serous exudation increases in severity and extent, extending over the shoulders to the chest wall, back and flanks. Larger, 5 to 40 cm diameter areas of wool loss develop, predominantly over the shoulders and dorsum (Figs 7.4, 7.5 and 7.6). Skin abrasions are often seen over the alopecic lesions, indicating self-inflicted trauma. The skin of the alopecic lesions becomes thickened and paved with flake-like scabs (Fig. 7.7). Thick-walled, 5 to 20 mm diameter abscesses

Fig. 7.6 Severe pruritus and wool loss associated with advanced sheep scab in Scottish Blackface wether lambs.

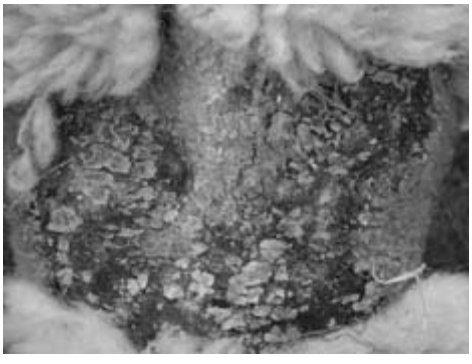


Fig. 7.7 Skin pigmentation and thickening, with a covering of flake-like scabs. While these lesions gave rise to the term sheep scab, their presence indicates an unacceptable delay in the identification and management of the disease.

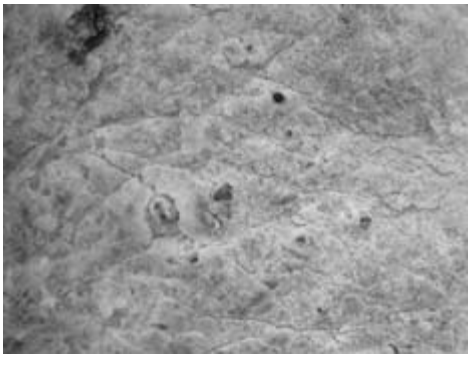


Fig. 7.8 Raised, thick-walled, thick pus-filled epidermal abscesses in a longstanding case of sheep scab.

Fig. 7.9 Debilitation associated with serous exudative protein loss, with saturation of the entire fleece.

develop within the skin (Fig. 7.8). Eventually the skin over the entire cervical, thoracic and lumbar dorsum and flanks is affected, with any remaining wool becoming saturated, matted and discoloured (Fig. 7.9). Handling of scab lesions usually stimulates retraction of the upper lip, protrusion of the tongue and compulsive nibbling (Fig. 7.10).

Some severely affected sheep show signs of head tossing and frenetic biting at their flanks, demonstrating a nibble reflex in the absence of any external stimulation. Individual animals infected with extremely allergenic strains of *P. ovis* exhibit epileptiform seizures when handled, with loss of voluntary control, collapse, opisthotonus, horizontal nystagmus, frenzied champing of the jaws and foaming at the mouth, lasting from 5 to 10 minutes before slowly recovering to normality. Death and severe traumatic injury have been reported associated with such fitting behaviour.

Large subcutaneous fluid swellings (seromas), of between 1 and 10 litres in volume, are occasionally seen over the shoulders and dorsum of sheep with severe exudative lesions (Fig. 7.11). Some severely affected sheep become sternally recumbent,

Fig. 7.10 Handling of scab-affected sheep may stimulate dorsal flexion of the neck and extension of the forelimbs, while sinking at the hocks, accompanied by retraction of the upper lip, protrusion of the tongue and compulsive nibbling.

Fig. 7.11 Large seromas over the shoulders associated with osmotic fluid movement following bruising of a profoundly hypoalbuminaemic ewe. These lesions may become infected, rapidly leading to debilitation.

Fig. 7.12 A high prevalence of aural haematomas is sometimes seen in previously *P. ovis* infested flocks.

associated with an eosinophilic myositis. Some untreated sheep become severely debilitated or die, while others eventually recover. Organising aural haematomas are sometimes seen in recovered cases (Fig. 7.12).

Animals are often seen at different stages of the disease within affected flocks. As the disease regresses and lesions heal, new wool growth occurs, lifting scabs away from the skin.

The severity of the disease is influenced by the stage of infection, the virulence of the strain of *P. ovis* involved, the time of year, fleece length, sheep age, sheep breed and the individual host's immune response. Clinical signs are generally more severe in lambs and yearling sheep than in ewes. Rapid growth of mite populations usually occurs when fleeces are long. Down and crossbreed breeds with a high density of wool follicles per cm^2 show more severe signs than hill breeds with open fleeces, associated with the microclimate maintained at the skin surface. The severity of clinical signs and number of mites present around lesions is highest in Dorset Down sheep, intermediate in Mules and low in Swaledales. Sheep scab is therefore generally obvious in Dorset flocks, but may be unnoticed in Swaledale flocks, which may act as reservoirs of infestation. Skin temperatures and humidity are higher in close-wool, compared with coarse-woolled breeds. Some of the between-breed and

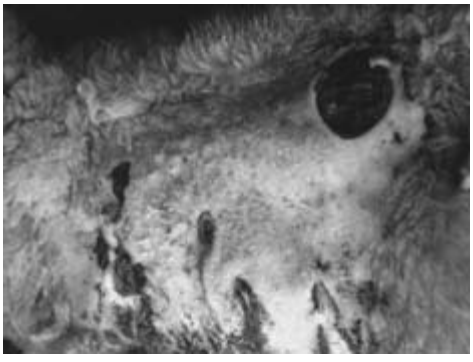


Fig. 7.13 The appearance of deep necrotic skin lesions associated with fat necrosis in an emaciated carcase.

between-individual animal variation in lesion severity is associated with differences in their immunocompetence, rather than the number of mites present.

Sheep with established chewing louse infections do not readily accept subsequent challenge with *P. ovis* mites, possibly because changes initiated by lice render the skin unfavourable for mite colonisation. However, sheep with active scab are readily colonised by lice and the two infestations commonly occur together.

The progression of clinical signs after re-infection follows a different pattern to primary infections. Mite numbers increase more slowly and the lesion area is generally smaller than that associated with primary challenge. Carrier animals with subclinical disease resulting from re-infection may be important in the overall epidemiology of sheep scab.

Postmortem findings

In addition to obvious skin lesions, common gross postmortem findings include enlargement, congestion and oedema of the prescapular lymph nodes. Other occasional findings include cellulitis and severe widespread fat necrosis underlying the shoulders and back (Fig. 7.13), and bilaterally symmetrical, green-coloured, eosinophilic myositis lesions. Septicaemia and septic arthritis have been reported, associated with the spread of infection from areas of subcutaneous fat necrosis.

The main skin histological findings are a neutrophil-rich proteinaceous surface exudate and a mixed inflammatory infiltrate in the superficial dermis, typical of an allergic dermatitis. Marked parakeratotic hyperkeratosis, large areas of subepidermal abscessation and adjacent areas of necrotic adipose tissue are sometimes seen, reflecting the chronicity of the lesions. Gram stains demonstrate the presence of Gram-positive bacteria within the dermal inflammatory lesions. Histopathological findings in prescapular lymph nodes are consistent with non-specific reactive hyperplasia and evidence of chronic eosinophilic enteritis is seen in some cases. The cause of eosinophilic gastroenteritis is unknown, although a chronic hypersensitivity reaction to ingested parasitic antigens is implicated. Eosinophilic myositis lesions are related to release of a heat-stable eosinophil chemotactic factor into traumatised muscle tissue.

Diagnosis

The diagnosis of sheep scab is based on the clinical signs, and to a lesser extent knowledge of the seasonal occurrence of the disease. The diagnosis is confirmed by

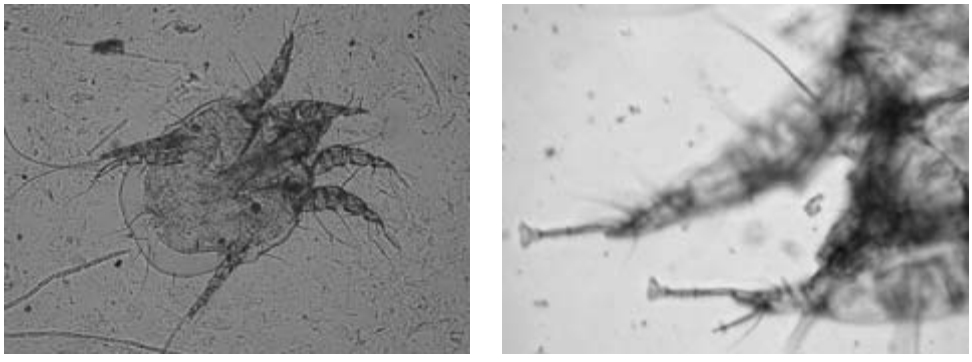


Fig. 7.14 (a) *P. ovis* mite viewed at 100x magnification on the underside of clear adhesive tape stuck to a microscope slide. (b) Funnel-shaped suckers at the end of three-segmented pedicels on the anterior pairs of mite legs.

the identification of *P. ovis* mites in superficial scrapings obtained using a scalpel blade drawn at right angles to the skin, from the periphery of exudative lesions. It can be helpful to apply a few drops of liquid paraffin to the skin, before scraping material directly onto a microscope slide. Alternatively, the presence of superficial mites can often be demonstrated using a piece of clear adhesive tape first applied to the skin, then stuck to a microscope slide and examined at 50× or 100× magnification. Adult *P. ovis* mites are oval bodied, 0.5 to 0.75 mm long and all of their legs project beyond the body margin. The mites are identified by pointed mouthparts and the presence of a three-segmented pedicel and funnel-shaped sucker on the first and second pair of legs (Fig. 7.14). Eggs, larvae and nymphal stages are also seen in scrape material or on adhesive tape (Fig. 7.15).

Whenever microscopic examination is not possible on site, it is necessary to collect samples from several sites, otherwise mites may be missed. It can be useful to view the edge of the lesions with a hand-held magnifying glass to identify the mites prior to taking scrapings.

Identification of mites from the periphery of older visible lesions is usually straightforward, but location of mites in subclinical lesions in early infestations, or in sheep infested for a second time, can be difficult. An ELISA test, based on a homogenised rabbit *Psoroptes cuniculi* antigen and detecting anti *P. ovis* IgG as been developed in Switzerland, which might prove to be useful in these situations where the disease is

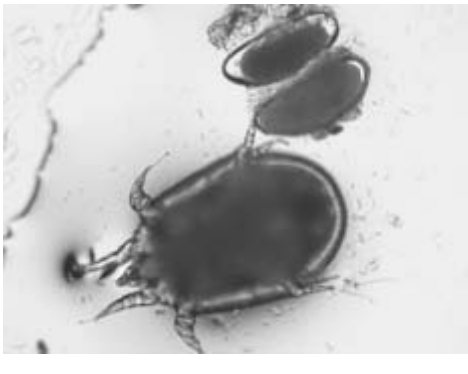


Fig. 7.15 *P. ovis* nymph and eggs on the underside of clear adhesive tape.

latent. Seroconversion occurs within 3 to 5 weeks after infection and antibody levels drop to zero by about 120 days after treatment. This Swiss ELISA test has been used as basis of a sheep scab control programme in that country, avoiding unnecessary treatment of seronegative flocks, thereby lessening the use of acaricides, reducing drug residues and environmental pollution, and removing the risk of selection for drug resistance. Unfortunately the active fraction of the homogenate is unknown and the specificity of the test is poor, with false positives associated with concurrent chorioptic mange or forage mite infestation. These problems could be overcome by development of an ELISA based on recombinant antigens, which could potentially be used to test sheep while in quarantine.

The development of ELISA tests for mite antigens in wool is feasible and would have potential value in the control of sheep scab.

Economic importance

Sheep scab is primarily an animal welfare concern. Economic loss is mostly associated with the cost of prevention or treatment to avoid animal suffering. However, failure to recognise and treat sheep scab promptly, or improper treatment, can incur economic loss due to increased feed costs to compensate for loss of body condition. In young and growing lambs, *P. ovis* infestation can result in rapid weight loss, debilitation and death (Fig. 7.16).

There is a significant inverse correlation between the severity of the scab lesions and the host's serum albumin concentration, associated with a combination of exudative albumin loss through the skin lesions and albumin leakage into the intestinal tract. Low birthweights and high perinatal mortality rates in lambs born to ewes with severe sheep scab during pregnancy probably result from a combination of hypoalbuminaemia and excessive loss of body condition in the ewes.

Slaughterhouse condemnation of carcases on the grounds of generalised lymphadenitis, practical difficulties of removing the pelts from infested sheep, downgrading of leather, and the unwillingness of severely affected rams to work further add to the economic importance of *P. ovis* infestation.

Life cycle

Most sheep scab outbreaks occur during winter and spring months, when fleece length and environmental humidity combine to provide optimal conditions for the

Fig. 7.16 Sheep scab can be debilitating in young lambs. Furthermore the involvement of young lambs compromises effective flock treatment and disease control.

Example

In mid October, sheep scab was diagnosed in a March lambing Poll Dorset and Suffolk cross cast ewe flock (Fig. 7.17). The ewes had been purchased from sources throughout Scotland during the previous 2 months, and were run mostly on arable stubble fields. Sheep scab lesions were present in 90 percent of the flock, but there was considerable individual variation in the severity of clinical signs, with evidence of regression and healing in some animals.

When first examined, the body condition scores of the ewes were normally distributed around a mean of 3.2 (range 1–5). Despite effective treatment using systemic endectocide injections, severely affected ewes lost 0.8 units of body condition score more than less severely affected animals over the following 7 weeks. Subsequent loss of body condition until lambing in March did not differ between those ewes which had shown severe scab lesions and those which had not, but the median birthweights of twin and triplet lambs born to ewes which had severe scab were 10 percent lower than those born to ewes which had less severe lesions.

Fig. 7.17 Some of the Dorset ewes in this group show obvious clinical signs of sheep scab, while others appear to be clinically unaffected. However, all are infested with *P. ovis* mites, illustrating the biosecurity risk associated with introduction of apparently healthy sheep.

multiplication and development of *P. ovis* mites. During the summer months, *P. ovis* populations are generally small, most mites residing in a latent phase in body folds, the axillae, infra-orbital, inguinal and interdigital fossae. While most outbreaks of sheep scab correspond with the natural increase in the activity of *P. ovis*, spread of the infestation can occur during the seasonal latent phase.

The incubation period of clinical sheep scab varies from 2 to 8 weeks, depending on the time of year and strain of *P. ovis* involved. Given ideal conditions for the mite, which usually arise during autumn and winter, the life cycle of the mite through egg, larval and nymphal stages to the point of oviposition takes about 15 days. Eggs are laid on the edges of the lesions and hatch in 1 to 3 days. Adult ovigerous mites can live for a period of up to 40 days, laying one or two eggs daily. Thus, as few as 25 mites may give rise to over one million within 12 weeks.

Following initial infection, there is a lag phase while the mites sensitise their sheep hosts with allergens, with low mite numbers and small undetectable lesions. This phase usually lasts 20 to 25 days, but can be shorter or longer depending on the time of year, sheep breed and strain of mite involved. This is followed by a rapid growth phase, involving an increase in mite numbers, lesion spread and an increase in the hosts' level of circulating IgG antibody. Mites are forced to aggregate at the periphery of lesions, because they cannot feed on hardened scabs, so lesions spread outwards as

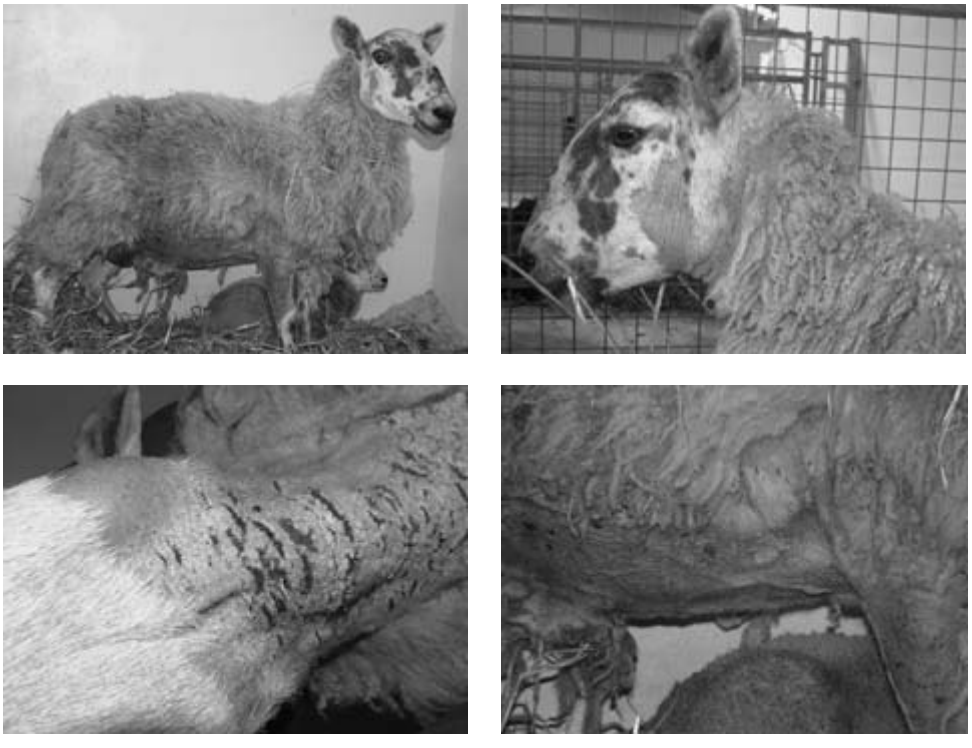


Fig. 7.18 (a) Alopecia, inflammation and skin thickening associated with severe sheep scab. In this case, only *P. ovis* mites were identified at all sites, but the involvement of hair-covered parts of the body should raise concern about the possibility of sarcoptic mange, which has never been reported in UK sheep. (b) Involvement of the face. (c) Involvement of the chin and neck. (d) Involvement of the ventral abdomen and upper limbs.

mite populations increase. Peak transmission occurs between about 45 and 60 days after start of this phase. A plateau phase follows with stabilisation of mite numbers, but a continued increase in lesion area. Mite numbers then decline rapidly due to a lack of feeding sites and their hosts' immune response. In severe cases, the lack of feeding sites may cause mites to disperse randomly over the whole body (Fig. 7.18), but generally lesion growth stops and edges of the lesions become indistinct and scaly. Aural haematomas and secondary bacterial infections commonly occur during this decline phase. During the subsequent regressive phase mite populations may die out completely and animals may make a full clinical recovery. However, some sheep still harbour small numbers of mites under dry scabs or in cryptic sites. In practice, exceptions from this classic description of the *P. ovis* mite population dynamics and manifestation of clinical disease are commonplace.

The entire life cycle of *P. ovis* occurs on sheep skin. However, mites have a fingerprint-like pattern on their cuticle which increases their surface area for oxygen transfer and enables their survival in the environment away from their host, while retaining the ability to re-infect sheep, for up to 17 days. Environmental survival is optimal when weather conditions are humid and cool, ovigerous female mites surviving longest.

Feeding behaviour of P. ovis

The feeding habits of *P. ovis* on sheep are not fully understood. Scanning electron microscope studies have shown that the mites possess sucking mouthparts, but neither embedded mite mouthparts, localised tissue damage, nor cellular infiltrations which might suggest that *P. ovis* penetrates deeper than the outermost loose stratum corneum, have been identified. The mites do not possess salivary ducts or glands, indicating that they feed mostly on serous exudate. Histological examinations of mite body cavities suggest that *P. ovis* mites on sheep ingest lipid from the superficial loose stratum corneum of the skin, although lipid may not be an important constituent of the mite's diet, since enzyme assays have failed to demonstrate any mite lipase activity. Further evidence suggests that the mites prefer to graze the outer epidermal strata of the moist skin around the edges of scab lesions, ingesting lipid, sloughed skin cells and exudate and extracting nutritive material such as live or dead bacteria.

Pathogenesis of P. ovis*-induced skin lesions*

In common with some other sheep parasites, *P. ovis* mites exploit their hosts' immune response, the heat and humidity of inflammation providing an ideal microclimate for the mite, and the serous inflammatory exudate providing nutrients. Sheep with sheep scab show an increase in total white blood cell counts with eosinophilia, neutropenia and lymphopenia and a breed variance has been observed in the level of eosinophilia that correlates with differences in clinical response and parasite numbers.

The clinical signs of sheep scab result from host hypersensitivity to mite excretory products, in particular guanine, packaged in the peritrophic membrane (a part of the mite's intestinal lining). Trials assessing an ELISA technique for the sero-diagnosis of sheep scab have demonstrated a positive relationship between host immunocompetence and the extent of clinical disease whereby animals that produce high antibody titres develop the most extensive lesions.

There are several field strains of *P. ovis* which have different antigenicities. Some strains produce a slow, chronic form of the disease, while others result in minimal disease in older sheep, but severe clinical signs in 6-month-old lambs.

Removal of lipid from the stratum corneum associated with the host's immune response and feeding activities of the *P. ovis* mites results in water loss and exposes the superficial skin layers to bacterial infection and the effects of other noxious agents.

Psoroptic otoacariasis

All field strains of *P. ovis* readily infest cryptic sites including the external auditory meatus. Mites can migrate to the external auditory meatus from lesions as far away as the mid back and in chronically sheep scab infected flocks, the incidence of head shaking, aural haematomas, abscesses of the pinnae, cauliflower ears and wounds to the base of the ear can be high. *P. ovis* mites isolated from the external auditory meatus of sheep which have recovered from clinical sheep scab have been shown to produce only weak or no clinical disease on transfer to the body of clean sheep, possibly due to a dynamic selection process which occurs as mites adapt to changes in environment and host defences.

Lesions associated with the presence of psoroptic mites in the external auditory meatus are also seen in flocks with no history of classical sheep scab. The prevalence of such psoroptic otoacariasis appears to be highest in pedigree flocks, but the relationship between psoroptic mites in the ear and on scab lesions is not understood. Plunge dipping has no effect on psoroptic mite populations in the external auditory meatus, but systemic endectocides appear to be effective.

Principles of sheep scab control

The Animal Health Act 1981 [Sheep Scab Order 1997] prohibits movement of sheep which are visibly affected with sheep scab, requires treatment of visibly affected sheep, and gives local authorities powers to deal with sheep scab on common land.

Sheep scab can be introduced to a flock by carrier sheep, including purchased animals, sheep returning from grazing and strays (Fig. 7.19), or by exposure to mites in the environment, including contaminated fields, handling facilities, shared equipment, sheep transport and fomites on clothing. Recently infected sheep may not show clinical signs of sheep scab, so apparently healthy introduced animals should always be considered as a potential source of infection.

A flock infestation of sheep scab can potentially be instigated by only one egg-laying female mite. It is therefore essential that all sheep are gathered and correctly treated during an appropriate period of time, which takes into account the length of residual protection afforded and the fact that scab mites can survive off sheep for up to 17 days. Handling pens and fields should, therefore be considered as a source of re-infection for at least 17 days after removal of untreated sheep and a residual acaricide must be used whenever it is necessary to return sheep to potentially contaminated fields or pens after treatment. Whenever possible, acaricide treatment should be delayed until all replacement sheep have been introduced, but ideally administered before the mating period. In some areas it may be beneficial to delay treatment until later in the winter when feral sheep in areas of forest emerge to feed and can be caught (Fig. 7.20). Any additional introduced animals should be treated

Fig. 7.19 Stray sheep can prove to be important in the transmission of sheep scab. Helpful passers-by sometimes chase these animals into the nearest field, which may contain sheep from a different, hitherto sheep scab-free flock.

Fig. 7.20 Feral sheep, especially in forested areas, can create problems for sheep scab control.

Table 7.1 Products for sheep scab control listed in the National Office of Animal Health *Compendium of Animal Medicines* (2007).

Product	Chemical	Application method	Protection	Meat withdrawal
Cooper's Ectoforce Sheep Dip	Diazinon[1]	Plunge dip	>28 days	35 days
Osmond's Gold Fleece Sheep Dip	Diazinon[1]	Plunge dip	>28 days	35 days
Paracide Plus	Diazinon[1]	Plunge dip	>28 days	35 days
Robust	High-cis cypermethrin[2]	Plunge dip	<17 days	18 days
Auriplak Fly and Scab Dip	High-cis cypermethrin[2]	Plunge dip	>17 days	12 days
Ecofleece Sheep Dip	High-cis cypermethrin[2]	Plunge dip	>17 days	12 days
Panomec Injection for cattle, sheep and pigs	Ivermectin	2 x s.c. injection 1 ml/50 kg	Non-persistent	42 days
Dectomax Injectable Solution for cattle and sheep	Doramectin	i.m. injection 1 ml/33 kg[3]	~17 days	70 days
Cydectin 1% Injectable Solution for sheep	Moxidectin[4]	s.c. injection 1 ml/50 kg	28 days	70 days

[1] Organophosphate dips and ganglion-blocking anthelmintics should not be used within 14 days of each other.
[2] Marketing authorisation temporarily suspended.
[3] The sheep scab dose rate for DECTOMAX is greater than its anthelmintic dose rate.
[4] Moxidectin injection should never be used in animals which have been vaccinated against footrot.

and quarantined for sufficient time to ensure that scab mites are killed before they are allowed to mix with the previously treated flock.

While there is evidence to suggest that sheep strains of *P. ovis* mites from sheep can temporarily survive on cattle, goats or deer, infestations do not appear to become established and disease is not seen. To-date, re-infection of sheep from cattle and goats temporarily carrying sheep *P. ovis* mites has not been reported, although the potential role as maintenance hosts of these species is a slight cause for concern.

The options for sheep scab control are to maintain closed flocks with effective biosecurity, or timely acaricide treatment of all sheep which are likely to come into contact with each other. Few flocks are able to remain totally closed, so in most cases a minimum of one autumn acaricide treatment is required to mitigate the risk of sheep scab introduction. The choice of acaricide is governed by the persistence of protection against re-infection, the time taken to kill any scab mites present, the reproductive status of the sheep, the meat withdrawal period (Table 7.1), animal handling facilities, labour and cost.

Plunge dipping

The organophosphate acaricides are cholinesterase inhibitors, interfering with mite neuromuscular transmission. Diazinon was the first organophosphate to be approved in 1981 for sheep scab control in the UK, having been used for blowfly and chewing louse control since the early 1970s. Propetamphos was approved for sheep scab control in 1982 and was used throughout the compulsory control period, but then withdrawn for economic reasons. Sebacil is used for sheep scab control in mainland Europe, but not in UK.

Organophosphates were first developed as by-products of nerve gases, so it is not surprising that dip formulations have been incriminated in human toxicity, referred to as 'dipping flu'. Repeat human exposure appears to lead to delayed toxicity. About 5 percent of individuals appear to be predisposed to dipping flu, associated with putative genetic differences in the expression of a target enzyme, such as acetylcholinesterase, or in activation and detoxification enzymes. Consequently continued availability, or further development of organophosphate plunge dips, seems unlikely.

The synthetic pyrethroid acaricides are carboxylic acid esters which act on mite sodium channels, interfering with nerve transmission, but have relatively low mammalian toxicity. Flumethrin was the first synthetic pyrethroid to be licensed in the UK in 1987, followed by the introduction of high-cis cypermethrin. Sale of flumethrin was subsequently discontinued, and in February 2006 the UK marketing authorisation for high-cis cypermethrin plunge dips was temporarily withdrawn due to concerns about environmental pollution and a lack of adequate instructions on how to reduce the risks. The future availability of synthetic pyrethroid plunge dips is therefore uncertain. Pour-on (high-cis cypermethrin and alphacypermethrin) and spot-on (deltamethrin) formulations of synthetic pyrethroids are licensed for control of other ectoparasites, but are ineffective for the control of sheep scab.

Organochloride (γBHC/lindane)- and organophosphate (diazinon)-resistant *P. ovis* mites were first identified in Argentina in 1962 and 1970 respectively. Organophosphate-resistant *P. ovis* mites are now widespread in South America. Propetamphos-tolerant mites were identified in Caithness during 1995, characterised by animals continuing to exhibit pruritus, and recovery of live mites after repeated plunge dipping. These mites were susceptible to diazinon, but the risk of emergence of organophosphate resistance in *P. ovis* mites in the UK is nonetheless significant. Flumethrin-resistant *P. ovis* mites were identified in Somerset and in Caithness during 1994 and in Cumbria during the following year. Cross resistance with high-cis cypermethrin was demonstrated. If the marketing authorisation for pyrethroid dips is re-instated, then pyrethroid resistance in *P. ovis* mites could become a significant problem, associated with plunge dipping in sub-lethal acaricide concentrations and with the irresponsible use of long-wool pyrethroid pour-ons for the treatment of chewing louse infestations in sheep which could have concurrent sheep scab.

Dips are hazardous substances which can be absorbed through the skin, orally or by inhalation of vapour or aerosols. A certificate of competence from the National Proficiency Test Council is required for the purchase of both organophosphate and pyrethroid dips, and the wearing of personal protective equipment when dipping sheep must always be taken seriously, however impractical it may seem.

Plunge dips have a direct effect on *P. ovis* mites, but have no systemic activity if absorbed. Approved dips therefore kill scab mites within 24 hours. Provided that sheep are correctly plunge dipped, diazinon affords residual protection for several weeks. This persistence is partly related to fleece length, requiring at least 1 cm of wool growth and being longer with longer fleeces. The formulation of the high-cis cypermethrin plunge dips differs, with one product not forming a micro-emulsion on dilution and affording little residual protection, while others form micro-emulsions and persist for more than 17 days. Diazinon and high-cis cypermethrin plunge dips can also be used for the control of blowfly, headfly, chewing lice, keds and ticks,

although the appropriate timing for these treatments seldom coincides with that for sheep scab control.

Effective plunge dipping is a complex and precise procedure, requiring careful planning and attention to detail (Fig. 7.21). Failure to control ectoparasitic infestations is commonly associated with poor dipping practice rather than inefficacy of the acaricide used. Dippers need to be cleaned out beforehand and precautions taken to ensure that equipment is in working order, with no sharp edges or obstructions. The volume of the dipper should be worked out from its dimensions and the correct dilution rate for the concentrated dip must be accurately calculated to ensure the correct initial charge. Diazinon dips are now packaged in water-soluble bags of pre-measured concentrate, or require the use of specialist closed transfer dispensing equipment to ensure that human contact with the concentrated chemical does not occur. Depleted dip needs to be replenished by adding water and chemical concentrate whenever the volume falls below a predetermined useable level or after a certain number of sheep have been dipped. When sheep are dipped in organophosphate solutions, they do not simply remove large volumes of dip solution from the bath, but they also actively absorb suspended or emulsified fat-soluble dip particles into their wool grease. This is referred to as stripping. Thus, the chemical concentration in the dipwash falls as dipping progresses. It is therefore necessary to replenish the dipwash according to the manufacturer's instructions, using a higher chemical concentration than that used for the initial charge. Stripping does not occur with pyrethroid plunge dips, so replenishment is at the same concentration as the initial charge and there is no need to wait for a specific drop in volume or specified number of sheep to be dipped, provided that sufficient dipwash remains to immerse the sheep.

For the control of sheep scab, it is important that sheep are immersed in the dipwash for one minute, with their heads submerged twice during this period (Fig. 7.22). While one minute may seem to be an excessively long time, it is necessary because more than double the amount of acaricide is taken up by the fleece during this period compared to immersion for only 30 seconds. Immersion times are less crucial for the control of other ectoparasites. Sheep must be kept moving while in the dipbath to displace air from the fleece and to aid dip penetration.

Fig. 7.21 Effective plunge dipping requires careful planning and attention to detail.

Fig. 7.22 Every sheep must be immersed in the dipwash for at least one minute. Movement in the dipbath helps to saturate the fleece.

Fig. 7.23 Faecal and soil contamination can result in brown discolouration of the dipwash. This contamination progressively reduces the effective acaricide concentration.

Fig. 7.24 Running sheep over coarse stones and gathering into clean yards before dipping can substantially reduce faecal and soil contamination of the dipwash.

Sheep dips often become brown coloured, indicating contamination with up to 5 percent faeces and dirt (Fig. 7.23). This organic matter binds to the dip chemical, reducing its active concentration and efficacy. Ideally, therefore, faecal contamination should be limited by yarding sheep overnight and walking over slats or coarse stones before dipping (Fig. 7.24). Dippers should be emptied and cleaned once substantial faecal contamination becomes obvious.

Dipping very wet sheep, or during heavy rain, or the practice of allowing water to run into the dipbath all the time should be avoided, because these conditions result in dilution of the dipwash without a reduction in its volume, making accurate replenishment difficult to achieve.

To reduce the risk of post-dipping lameness, caseous lymphadenitis, *Pseudomonas aeruginosa* infection, or blackleg associated with dipping sheep in contaminated dipwash, dippers should be emptied and cleaned at the end of each day's session. Alternatively, and only if emptying the dipper is impractical, a compatible bacteriostat can be added, according to the manufacturer's instructions. This should be done at the end of the day, rather than at the start of the next session.

Dipping sheep when replete, hot or thirsty can result in misadventure, inhalation or ingestion of dip solution, so should be avoided. Ideally sheep should be dipped in the morning, so that fleeces can dry before temperatures fall. Debilitated sheep should be humanely killed, rather than subjected to plunge dipping. Dipping of young lambs should be avoided unless absolutely necessary, in which case great care should be taken to avoid misadventure.

Various types of plunge dipper are employed. Short pot dippers are best suited to small flocks, but their use is generally labour intensive, particularly with older apparatus where sheep require to be manhandled into the bath. It can also prove physically difficult and time consuming to ensure that all sheep remain in the dip bath for one minute (Fig. 7.25). Long swim-through dippers and circular dippers with a central operator's platform (Fig. 7.26) can accommodate more sheep at any time, and offer more control over the dipping process, but being larger require more dip concentrate. Sheep are forced to swim in these baths, rather than simply diving in and out, aiding better saturation by displacing air in their fleeces. All types of dippers

Fig. 7.25 Effective plunge dipping is impossible using historic apparatus such as this.

Fig. 7.26 A circular plunge dipper with a central operator platform.

Fig. 7.27 Sheep should not be released from drip pens until dip has ceased to run from their fleeces.

Fig. 7.28 Historic dippers such as this were often built close to watercourses, to facilitate filling. The dippers can no longer be used due to the high risk of groundwater contamination.

should have adequate screens and splashboards to ensure easy operation and minimise the risk of operator exposure to dip solution. After dipping, sheep should be stood in a drainage pen for about 10 minutes until dip solution ceases to run from their fleeces (Fig. 7.27), and then turned onto shaded pasture, away from watercourses. Sheep should never be penned in a closed building after dipping.

For flocks of more than about 200 sheep, the cost of dip concentrate and labour amounts to between £0.40 and £0.60 per head, depending on the size of the dipper and number of sheep to be dipped.

Serious consideration must be given to the disposal of spent dip solution left in the bottom of the dipper at the end of a session and to contamination of groundwater. Pyrethroids are particularly toxic to aquatic invertebrates which support fish stocks. The importance of this cannot be understated, because in many areas game fishing contributes more than sheep farming to the local economy. Most pollution incidents are associated with positioning of older dippers close to watercourses (Fig. 7.28), or with failure to empty dippers immediately after use, allowing their overflow into drains following subsequent rainfall.

Fig. 7.29 Consideration should be given to the fact that newly dipped sheep may need to cross burns when they are returned to the hill, with the associated risk of groundwater contamination.

Fig. 7.30 Commonsense must be used when spreading spent dipwash onto pasture, to ensure that it cannot directly enter any watercourse.

Another important potential cause of pollution arises from recently dipped sheep dripping into streams or drains. Sheep must not be released from the drainage pens for at least 10 minutes after dipping, to ensure that run-off is contained. Where there is a risk of hill sheep crossing streams after dipping, it is advisable to turn them first onto an enclosed field for 24 hours (Fig. 7.29). These considerations must be taken into account for when deciding on the positioning of mobile dippers. Yarding on concrete should be avoided, because run-off from the concrete could find its way into groundwater through drains.

European Union groundwater directives stipulate that a licence from the relevant local environmental agency is required before spent dip solution can be spread on pasture and common sense must be applied to ensure that it cannot enter a watercourse (Fig. 7.30). It has been suggested that addition of agricultural caustic soda or soaked lime and hypochlorite in accordance with the dip manufacturer's recommendations may aid the degradation of spent pyrethroid dips and organophosphate dip solutions respectively. However, these detoxification processes render the spent dip difficult to spread onto the disposal site, so are not widely practised. Alternatively the dip must be removed by a waste disposal contractor, which can prove expensive and difficult to arrange, due to liability insurance and adherence to special waste regulations. Failure to observe strict precautions to avoid groundwater contamination could result in severe penalty through withdrawal of subsidy payment.

Consideration must also be given to the potentially harmful effects to people and the environment of pyrethroid dip residues in wool (Fig. 7.31). In Australia and New Zealand, the risks associated with chemical residues in wool are addressed by voluntary 60- or 180-day withholding periods after treatment for crossbred and fine wool respectively. In the UK, only good husbandry practice is currently recommended, despite a suggestion that a significant proportion of UK wool contains unacceptable residue levels.

Plunge dip solutions are ineffective for the control of sheep scab when used in shower dippers (Fig. 7.32) or jetting races. These methods fail to ensure saturation of the entire fleece and body to skin level.

Fig. 7.31 The harmful effects of dip residues in wool must not be overlooked. Groundwater could be contaminated by effluent from scouring plants, both in the UK and overseas, during the processing of fleeces from UK ewes which were dipped shortly before shearing.

Fig. 7.32 Shower dippers are ineffective for the control of sheep scab and must not be used for this purpose.

Systemic endectocide injections

Many farmers are now uncomfortable about the use of plunge dips because of fears about operator safety, problems associated with the disposal of spent dip solution and potential toxicity of wool residues to aquatic environments. Not surprisingly, therefore, on many farms systemic endectocide injections are now considered to be more appropriate methods of sheep scab control.

Macrocyclic lactones are fermentation products of soil micro-organisms (*Streptomyces avermitilis*), which have been chemically modified to produce avermectins and milbemycins. The macrocyclic lactone endectocides inhibit mite neural transmission, resulting in a state of paralysis. Macrocyclic lactones given by injection are also effective against sucking lice and keds. Two subcutaneous injections, 7 days apart, of 200 µg/kg ivermectin, or a single intramuscular injection of doramectin at a dose rate of 300 µg/kg, provide effective control of sheep scab. The rationale for double ivermectin injection is based on the fact that a single injection does not consistently kill all of the mites present, its efficacy being related to the virulence of the mite population. Ivermectin injections achieve little persistence in sheep, thus failing to kill mite larvae which do not feed for periods of 12 to 36 hours during moulting, or eggs deposited away from the skin which have a delayed incubation. The potential for moulting (pharate) mites to evade systemic endectocides is greatest when mite populations are high at the time of treatment. Long-acting ivermectin formulations are available in South Africa for management of sheep scab and keds as a single injection. While a single injection of doramectin achieves some persistence, this may occasionally be insufficient to provide protection against re-infection for the whole 17-day period during which the scab mite can survive off the sheep. When ivermectin or doramectin are used, sheep should, therefore, be moved to an area which has been free of untreated sheep for the previous 17 days, in order to avoid re-infection from the environment. A single subcutaneous injection of moxidectin at a dose rate of 200 µg/kg provides residual protection against sheep scab for at least 28 days, although the UK data sheet recommends two injections 10 days apart for the treatment of disease outbreaks. The cost of treating a 60 kg ewe with a systemic endectocide for

Fig. 7.33 Accurate injection with endectocides is a skilled and time-consuming task.

the control of sheep scab ranges from about £0.60 to £1.20, depending on the product and supplier used.

Sheep sometimes continue to show signs of pruritus for up to 30 days after systemic endectocide treatment. This is because it may take up to 7 days before all mites are killed. Furthermore, dead mites and mite excretory products remain on the skin long after mites have died, retaining the ability to evoke an allergic dermatitis. The alternative of plunge dipping physically removes most of these allergens, leading to rapid cessation of pruritis.

A sample of sheep should be weighed before systemic endectocide treatment, syringes calibrated and care taken to ensure that all sheep actually receive an injection of the correct drug dose. For subcutaneous injections, the wool must be carefully parted to expose the skin and avoid injection into the fleece (Fig. 7.33). Particular care is required when injecting lambs, and endectocides are unsuitable for lambs weighing less than 10 kg.

While macrocyclic lactones given as oral drenches can reduce *P. ovis* populations by up to 50 percent within 24 hours of treatment, they are of no value for the control of sheep scab. Intraruminal controlled-release capsules releasing a minimum of 20 μg/kg/day of ivermectin, which are used in some southern hemisphere countries for the control of parasitic gastroenteritis, have reasonable efficacy against sheep scab and chorioptic mange, which can aid in the prevention of production-limiting disease, but does not reduce populations by 100 percent. Suppression of subclinical disease in the field by the use of oral macrocyclic lactones could contribute to the spread of sheep scab with clinically unaffected carrier sheep and select for endectocide resistance.

Systemic endectocide injections only have a narrow range of efficacy against other ectoparasites, such as blowflies and chewing lice, but can be useful for the control of parasitic gastroenteritis. The systemic endectocides are particularly useful, when used in sequential combination with levamisole, for quarantine treatment of purchased animals to reduce the risk of introduction of both sheep scab and of anthelmintic-resistant nematodes. However, reliance on systemic endectocides for sheep scab control may impose selection pressure for macrocyclic lactone anthelmintic resistance and create increasing problems due to other ectoparasitic diseases.

There is some evidence that macrocyclic lactones, whether administered orally or by injection, can have an effect on the arthropod fauna associated with faecal decomposition when voided in faeces onto pasture.

Common problems associated with sheep scab control

Annoyingly, sheep scab outbreaks commonly occur in flocks which have been dipped or injected with a systemic endectocide within the previous 6 months. The reasons for the appearance of the disease in these flocks include:

- ineffective preventive management associated with:
  - an inappropriate choice of dip or systemic endectocide
  - poor dipping technique
  - underdosing of a systemic endectocide
  - failure to gather and treat the whole flock
- subsequent introduction of infection with:
  - stray or feral sheep
  - sheep returning from away grazings
  - purchased replacement lambs, gimmers, ewes or rams
  - infected neighbouring sheep rubbing on common fences
  - fomites (tags of wool from neighbouring infected sheep)
  - shared handling facilities or transport
  - human contact with infected sheep.

Example

Every sheep in an upland flock of about 1600 mixed-age Greyface ewes, replacement ewe lambs and rams was carefully plunge dipped in diazinon during October, with the aim of preventing sheep scab. During the following March, sheep scab was diagnosed in the flock, probably having been introduced by across-the-fence contact with an infected neighbouring flock. Treatment by plunge dipping was considered inappropriate due to the advanced stage of pregnancy of the affected ewes, with the only remaining treatment option being systemic endectocide injections. Almost all of the sheep were treated by intramuscular injection of 300 µg/kg of doramectin. However, insufficient doramectin was supplied to treat the whole flock, so the last 20 ewe lambs in the pens were misguidedly treated with a single subcutaneous injection of 200 µg/kg of ivermectin. On 12 May, having already spent £2500 on sheep scab control, the disease was diagnosed in two ewe lambs (Fig. 7.34).

Effective treatment of sheep scab in large flocks of recently lambed ewes and young lambs is impractical. Lambs weighing between 5 and 20 kg cannot be safely plunge dipped and accurate injection of 0.02 to 0.03 ml/kg of systemic endectocide is impractical.

Fig. 7.34 Severe sheep scab lesions in two ewe lambs.

Example

On 11 February, a cast Scottish Blackface ewe with clinical signs consistent with severe sheep scab (Fig. 7.35) was observed in a field adjacent to a flock of 520 Scottish Halfbred ewes. The Scottish Halfbred ewes, which were due to start lambing on 1 March, had all been carefully injected subcutaneously during the previous September with 200 µg/kg of moxidectin for the control of sheep scab. When informed of the potential threat of sheep scab from the neighbouring sheep, the owners of the Scottish Halfbred ewes reported that they had been concerned about pruritus in their own flock, but had been reassured that the obvious wool loss in the neighbouring sheep was a result of chewing louse infestation. Closer inspection of the Scottish Halfbred ewes revealed areas of serous exudation and stained wool over the lower neck and shoulders (Fig. 7.36). *P. ovis* mites were identified by microscopic examination of clear adhesive tape which had been applied to the skin underlying these lesions.

While none of the Scottish Halfbred ewes was severely affected, immediate treatment was required. Plunge dipping of heavily pregnant ewes was not practical, leaving only the option of systemic endectocide injections. However, it takes several days before all sheep scab mites are killed following treatment using systemic endectocides, and the mites can survive off the host for up to 17 days. It was necessary, in this case, to return the ewes after lambing to the previously occupied fields. Thus, the ewes had to be treated immediately to ensure that their lambs would not be infected through contact with fomites in their environment, subsequently acting as a source of re-infection to their dams.

Fig. 7.35 Obvious signs of sheep scab in a cast Scottish Blackface ewe in a neighbour's field.

Fig. 7.36 Early signs of sheep scab in late-pregnant Cheviot and Scottish Halfbred ewes.

Sheep scab is often diagnosed and treated at different times in neighbouring flocks, some requiring treatment three times between autumn and spring. The boxed examples illustrate the futility of sheep scab preventive measures on individual farms in areas with large sheep populations, and demonstrate the need for a co-ordinated regional or national approach to the control of sheep scab.

Regional control of sheep scab

The success of regional control programmes depends on cooperative implementation of control measures within a defined time period, involving effective use of

acaricides and biosecurity, based on an understanding of the epidemiology of the disease.

The control options for sheep scab are unavoidably complicated and regional sheep scab control requires careful planning to overcome potential pitfalls.

- Emphasis must be placed on the recommendation that whenever it is necessary to return sheep to fields, handling pens or buildings used by untreated animals during the previous 17 days, only acaricides affording more than 17 days persistence should be used, and non-persistent acaricides should only be used when it is possible to avoid fields, handling pens or buildings used by untreated animals for the subsequent 17 days.
- It is important that stray or feral sheep, in particular sheep occupying unfarmed areas of hill or forest, are dealt with.
- Organic farmers who are not permitted to plunge dip their sheep may be reluctant to use systemic endectocides in finishing lambs due to the impracticality of observing double the standard meat withdrawal period (2 × 70 days for doramectin and ivermectin), to comply with the rules imposed by the regulatory body. This problem can be overcome by not treating finishing lambs on organic farms, but isolating them from treated sheep, close monitoring and the imposition of strict biosecurity.
- Sheep scab control presents potential problems to store lamb-finishing systems, where several groups of sheep are purchased throughout the winter months. All animals introduced after regional control measures have been taken must be treated on arrival. If these animals are treated with a systemic endectocide, they must not be mixed with the main flock, or placed in areas used by the main flock for at least 7 days after treatment. Unfortunately these restrictions are seldom conducive to efficient farm management. These principles also apply to sheep which are certified as treated before sale. These animals might not have been correctly dipped or injected, or non-persistent acaricides might have been used which would not protect against infection acquired during the sale.
- Farmers with early lambing flocks may be reluctant to plunge dip their sheep when the regional control period coincides with or follows their mating period. While there are few published data to suggest that this would be a problem, the issue can be overcome by use of a systemic endectocide.
- Equipment such as shearing trailers or scanning races should not be allowed near to sheep flocks after the implementation of regional sheep scab control programmes, unless they have obviously been scrupulously cleaned beforehand to reduce the risk of introduction of disease with fomites.
- An effort must be made to define the periphery of the control areas with geographical obstacles to the spread of sheep scab, such as trunk roads, city boundaries and areas of forest. Maintenance of effective biosecurity on farms at the periphery of the control region is a particular concern.
- Any suspicious cases of wool loss or pruritus seen after the implementation of regional sheep scab control should be investigated.
- Potential problems such as farmer cooperation, meat withdrawal periods, feral sheep and dip disposal are all surmountable, and cooperative regional sheep scab control programmes can be effective. The effectiveness of regional control programmes in the south-east of Scotland, Angus Glens and Cheviot Hills shows that eradication of sheep scab from the UK is possible.

In the long term, the collaborative effort required to maintain freedom from sheep scab on a regional basis may not be sustainable due to difficulties identifying every sheep farmer in the region involved, biosecurity on peripheral farms, inherent difficulties associated with the use of systemic endectocide injections, potential widespread emergence of pyrethroid- and organophosphate-resistant *P. ovis* mites, and the foreseen withdrawal of diazinon and pyrethroid plunge dips. Alternative control options are not currently available, and are unlikely to replace the need for effective treatment protocols. There is, therefore, a clear and pressing need for the re-introduction of a national sheep scab control programme, backed up by statutory powers to deal with the disease in problem flocks.

Encouragement should be gained from the fact that sheep scab was eradicated from New Zealand at the end of the nineteenth century, following a farmer-led programme relying on the use of crude nicotine plunge dips derived from tobacco dust and a policy of shooting any sheep which could not be dipped, which was an attractive option due to the high price of tallow at the time.

Future options for sheep scab control

Various potential control measures have been proposed including:

- vaccination
- biological control using entomopathogenic fungi
- manipulation of the microclimate on the sheep skin
- development of mite behaviour modifiers, such as repellents, attractants, confusers or detachers
- development of a mite insect growth regulator.

Sheep acquire strong protective humoral immunity following infection and protection to challenge can be induced using certain crude mite extracts. Preliminary studies have shown that vaccination with these extracts reduces severity of lesions and mite counts, but does not remove mites. These observations support the feasibility of developing a vaccine, based eventually on defined recombinant antigens. Unfortunately, the identification of a pure antigen, which might form the basis for a recombinant vaccine, is proving elusive and a commercial vaccine is unlikely to be available for some time.

Isolates of an entomopathogenic fungus, *Metarhizium anisopliae*, have been identified. It is proposed that these fungi might feed on *P. ovis* mites on the skin surface. However, there are problems associated with the establishment of the fungi on the skin, optimal fungal growth occurring at temperatures slightly lower than those on the skin surface of long-fleeced sheep. While such biological control is unlikely to prove useful in the short term, its longer-term potential should not be overlooked.

Chorioptic mange

Chorioptic mange of sheep occurs in most countries around the world, but was not reported in the UK between the late 1960s and 2001. The disease was thought to have been eradicated as a result of compulsory dipping for the control of sheep scab, but re-emerged following deregulation of sheep scab control.

Cause

Chorioptic mange is caused by the sheep-adapted mite, *Chorioptes bovis*, which feeds on epidermal debris causing exudative dermatitis. The skin lesions are believed to be associated with a hypersensitivity reaction.

Sheep breed and nutritional status appear to have little effect on the severity of chorioptic mange lesions, although winter housing may contribute to the incidence of the disease by providing a suitable environment for the parasite and facilitating transmission between animals. The build-up in mite numbers over winter and spring is believed to be very slow, so in flocks which are not plunge dipped for ectoparasite control, the incidence of chorioptic mange tends to be higher in adult animals than in lambs.

Clinical signs

Wool-free areas of the plantar aspects of the pasterns of the hind limbs (Fig. 7.37), the skin between the coronary band and accessory digits (Fig. 7.38) and the poll (Fig. 7.39) of both sexes of sheep and of the lower part of the scrotum of rams (Fig. 7.40) are

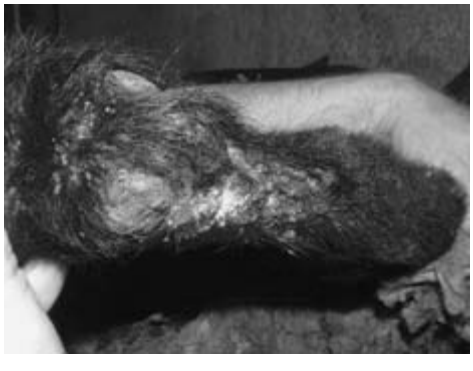


Fig. 7.37 Dry, crusting scabs cemented to the feathered hair on the plantar aspect of the lower pelvic limb of a Suffolk shearling ram.

Fig. 7.38 Moist scabs between the coronary band and accessory digits of a Scottish Blackface ewe.

Fig. 7.39 Chorioptic mange on the poll of a Shetland ram. The lesions are pruritic, so the scabs have been dislodged and the skin is bleeding due to rubbing.

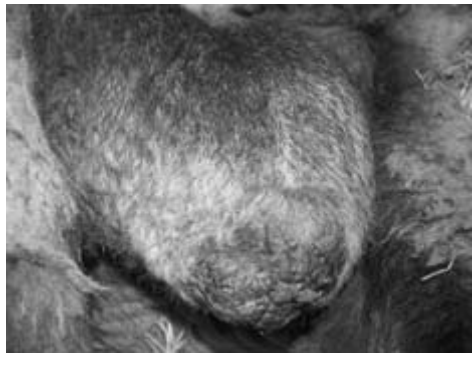


Fig. 7.40 Chorioptic mange on the scrotum of a Texel ram.

Fig. 7.41 A Greyface ewe repeatedly nibbling at the lower limbs due to chorioptic mange.

Fig. 7.42 Protrusion of the tongue and compulsive nibbling stimulated by handling of chorioptic mange lesions.

typically affected. Within a flock, individual animals are often affected in different areas to varying extents.

Lesions on the poll and limbs are typically characterised by dry, flaking scabs, covering 1 to 2 mm diameter, erythematous areas. Scabs become cemented to the hairs over the plantar aspects of the lower limbs and lifted from the skin as the hair grows. Short hairs above the coronary band are often lost. Affected sheep are often observed nibbling at their feet and fetlocks (Fig. 7.41). The skin of the lower part of the scrotum becomes thickened and exudative, developing haemorrhagic fissures. In many cases, handling of lesions initiates a nibble response (Fig. 7.42).

Diagnosis

The diagnosis of chorioptic mange is confirmed by the identification of 0.25 to 0.5 mm, oval-shaped mites in superficial skin scrapings, or on clear adhesive tape (Fig. 7.43). The mites are identified as *Chorioptes* spp. by their rounded mouthparts, short unjointed pedicels and bell-shaped suckers. However, *C. bovis* mites are seldom present in large numbers, so failure to find mites in skin scrapes from individual animals with obvious mange lesions does not rule out a diagnosis of chorioptic mange. In many cases, the diagnosis is based on the flock ectoparasite control history and clinical signs.

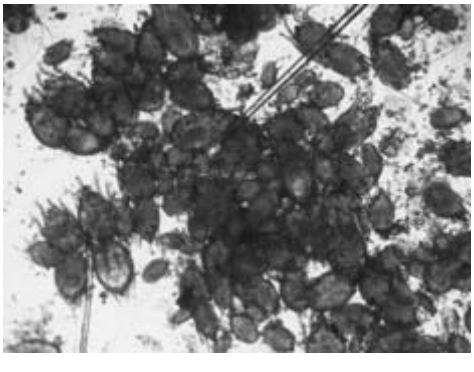


Fig. 7.43 It is unusual to identify *C. bovis* mites in such large numbers as this.

Control

None of the ectoparasiticides currently available in the UK are licensed for the treatment or prevention of chorioptic mange in sheep. Overseas, treatment and control of scrotal mange is usually achieved by topical application of an organophosphate or pyrethroid dip, for example using only the lower nozzles of a shower dipper.

In UK flocks, autumn treatments with systemic endectocide injections for the control of sheep scab appear to be ineffective against chorioptic mange. This may be explained by the fact that chorioptic mites have chewing mouthparts and feed on dry skin exudate, without piercing the skin and ingesting body fluids, thus escaping exposure to systemic ectoparasiticides. Cases of chorioptic mange are seldom identified in sheep which have been recently plunge dipped in diazinon or high-cis cypermethrin for the control of sheep scab.

Demodectic mange

Demodex ovis are small burrowing mites which selectively parasitise follicles and sebaceous glands of the feet, face, eyelids and back. Infested follicles become distended with mites, eggs, mite excretory products and inflammatory cells, forming nodules which develop into pustules following secondary bacterial infection. Demodectic mange is usually clinically insignificant, although severely affected sheep are pruritic, with thickened, scaly, nodular and pustular skin lesions.

The diagnosis of demodectic mange is confirmed by the identification of mites in deep skin scrapings, taken with the scalpel blade held at right angles and the skin scraped until it oozes blood, or in exudate from follicular lesions. *D. ovis* mites are 0.2 to 0.3 mm long, with short legs and elongated, cigar-shaped bodies.

Psorobic mange

Psorobia ovis are non-burrowing mites which occur within the superficial layers of the epidermis of the sides, flanks and thighs of fine-wool merino sheep. Psorobic mange is seen in the southern hemisphere and America, but has never been identified in the UK. The mites have piercing mouthparts and cause mild pruritus with displacement of tufts of wool and self-excoriation due to rubbing. Infected skin is dry, scaly and hyperkeratotic and the overlying fleece becomes yellow coloured and bound together by scurf.

Psorobic mites are difficult to detect in superficial skin scrapings, so the fleece overlying lesions needs to be clipped and the skin scraped until blood starts to ooze. The mites are small, 0.1 to 0.2 mm long and round bodied.

Sarcoptic mange

Sarcoptic mange (face mange) of sheep is endemic in North Africa and southern and eastern Europe, but has never been reported in UK sheep. However, with fewer UK sheep farmers plunge dipping for sheep scab control and greater international animal movements, the possibility of emergence of the disease as a significant production-limiting and animal welfare problem should be considered.

Sarcoptic mange mites burrow into the epidermis causing serous exudation and intense pruritus, with thick crust formation, hyperkeratosis and alopecia. *Sarcoptes*

scabiei var *ovis* typically infests the skin of the head, around the eyes and ears, the axillae and the groin, although the disease may spread to the rest of the body. Sarcoptic mange is usually seen during late winter.

The diagnosis of sarcoptic manage is confirmed by the identification of mites in deep skin scrapings. Sarcoptic mites are 0.2 to 0.6 mm long, oval bodied, with short limbs which only just protrude beyond the margins of the body. Ridges of triangular scales can be seen along their dorsum and they possess suckers on long unjointed pedicels on the two cranial pairs of limbs.

Harvest and forage mites

Harvest mites (*Thrombicula* spp.) occasionally cause low-grade pruritus of the face and lower limbs of grazing sheep during late summer (Fig. 7.44).

Forage mites are only partially parasitic, most stages being free living in the environment. The mites occur in pasture, but hay and straw may also be heavily infested. Larval stages sometimes crawl onto and attach to the skin of grazing mammalian hosts as hypobi (Fig. 7.45), providing a method of dissemination. The presence of forage mites on the limbs, face or flanks causes irritation. Some forage mite excretory allergens are the same as those of psoroptic mites.

Forage mites are a common cause of hair loss over the nose (Fig. 7.46) or around the base of the horns (Fig. 7.47) of housed sheep fed on contaminated hay. Large areas of wool break are sometimes seen over the flanks of housed sheep (Fig. 7.48) bedded on heavily contaminated straw. Unlike the situation with sheep scab where wool is lost from its follicles, wool loss associated with forage mites is caused by sheep nibbling the wool down to about 1 cm from the skin.

Control of forage mites is impossible because they only spend short periods on the sheep host, during which time they do not feed.

Fig. 7.44 Lesions on the face and ears of a pet ewe during late summer, putatively associated with hypersensitivity to harvest mites. While several sheep in the group were affected to a lesser degree, the problem was reported to have occurred in this individual at the same time every year.

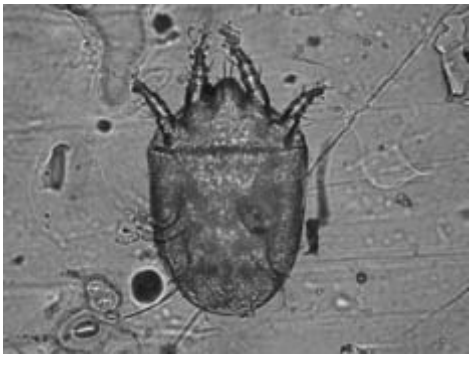


Fig. 7.45 A forage mite on the underside of clear adhesive tape that had been applied to the skin at the base of the horn of a Scottish Blackface ewe. The mite is about 0.3 mm long and does not have feeding mouthparts.

Fig. 7.46 Hair loss around the muzzle and over the bridge of the nose of a Scottish Blackface lamb, resulting from rubbing due to low-grade pruritus caused by forage mite allergens.

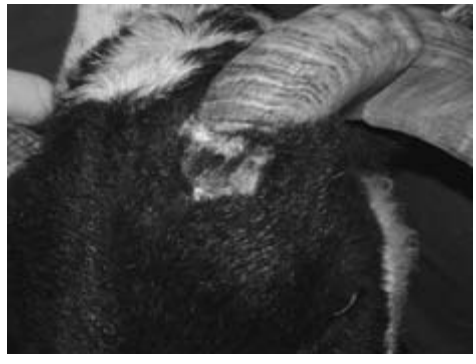


Fig. 7.47 Hair loss and skin excoriation around the base of the horn resulting from pruritus caused by forage mites.

Fig. 7.48 Wool break over the neck, chest wall and flanks associated with an allergic response to forage mite allergens. The problem was particularly severe in this group of cobalt- and selenium-deficient Scottish Blackface lambs.

Fig. 7.49 *Calliphora erythrocephala* (*left*) and *Lucilia sericata* (*right*) blowflies.

Blowfly strike

The terms 'blow' and 'strike' refer respectively to laying eggs by flies and damage caused by maggots. Blowfly strike in the UK results in the opportunistic invasion of living tissues by the larvae of *Lucilia sericata* (greenbottles), *Phormia terrae-novae* (blackbottles) and *Calliphora erythrocephala* (bluebottles). These species are not obligate parasites, and are involved with other environmentally useful tasks such as carcase decomposition. The flies are up to 1 cm long and have a metallic sheen to their bodies (Fig. 7.49). Maggots are smooth, segmented and 2 to 16 mm long, depending on their stage. Eggs are cream coloured and are seen in clusters of about 100 on the wool or skin (Fig. 7.50).

Life cycle

Unlike the situation for sheep scab and lice, most of the blowfly life cycle occurs off the sheep, and adult flies can travel large distances without recognising farm

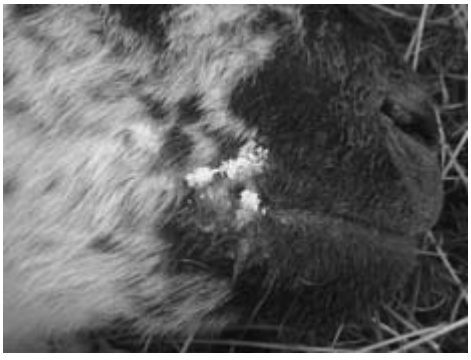


Fig. 7.50 Clusters of about 100 blowfly eggs around the lips of a freshly dead lamb.

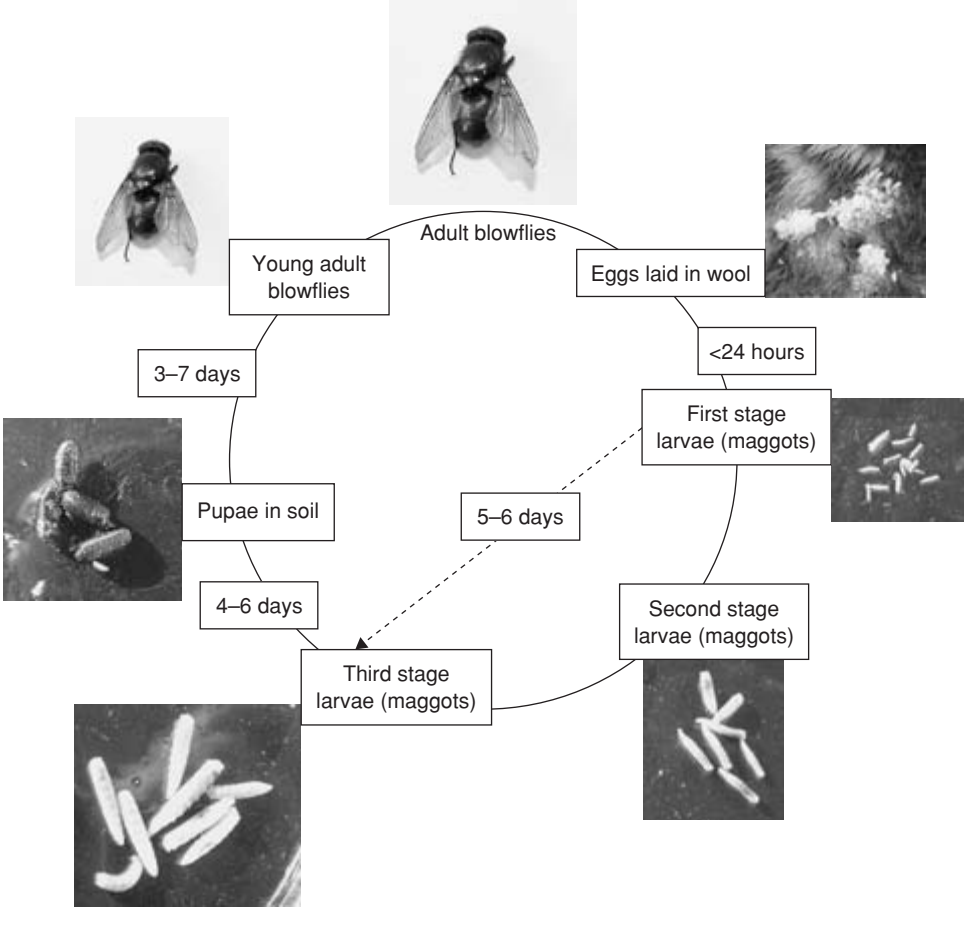


Fig. 7.51 The life cycle of sheep blowflies.

boundaries, creating challenges for disease control (Fig. 7.51). Adult female flies live for about 30 days during which they can lay up to 3000 eggs. Eggs are deposited on dead animals or soiled fleeces, and hatch into first-stage larvae within about 12 hours. These larvae feed on tissue, grow and moult twice, becoming mature maggots in 3 to

10 days, depending on environmental temperature and humidity. Third-stage maggots then drop to the ground and pupate, mature flies emerging between May and September after 3 to 7 days. Blowflies do not mate until after they have had a protein meal from a carcase or dung. Flies overwinter in the soil as pupae, and emerge as soil temperatures rise during the spring.

Blowfly populations are greatest during the summer months when given favourable conditions of humidity and warmth, the entire life cycle from egg to adult can occur in less than 10 days.

Pathology

Primary flies including *L. sericata* and *P. terrae-novae* can initiate strike on living sheep, while secondary flies including *C. erythrocephala* only attack areas which are already struck or damaged.

First-stage larvae penetrate the skin using their hook-like mouthparts and secreting enzymes which liquefy and digest the tissue. Larvae are active and voracious, causing further skin and muscle liquefaction as they develop, with secondary bacterial infection and attraction of secondary blowflies. Toxins released by decomposing tissues and ammonia secreted by the maggots are absorbed through the lesions into the blood, causing systemic illness which often leads to death.

Clinical signs

Affected sheep are usually restless and may bite or kick at the struck area. About 80 percent of flystrikes in UK lambs occur on the breech, possibly associated with faecal or urinary soiling (Fig. 7.52). Flystrike at the base of the neck (Fig. 7.53) or over the body (Fig. 7.54) is usually associated with exudative skin disease. The incidences of flystrike on the breech and body are similar in ewes. On close examination, the wool overlying struck areas is discoloured, moist and distinctively unpleasant-smelling. During the early stages, the maggots, which are approximately 1.5 cm long, are only visible, end-on, when the wool is parted (Fig. 7.55), but as the disease progresses, the wool falls out to reveal the underlying affected tissue (Fig. 7.56).

Fig. 7.52 Typical appearance of blowfly strike adjacent to the tail head of a Suffolk cross lamb.

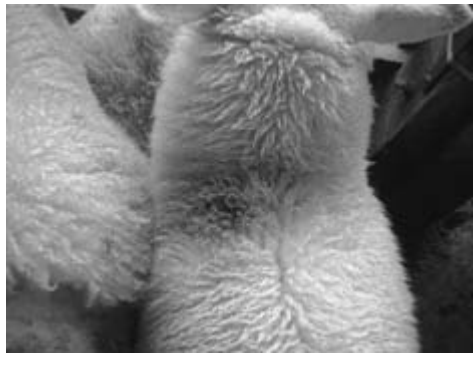


Fig. 7.53 Moist, discoloured wool overlying a flystruck area at the base of the neck of a Texel cross lamb.

Fig. 7.54 Flystrike over the flank of a Texel cross lamb.

Fig. 7.55 In the early stages of flystrike, maggots can be seen only when the wool overlying the lesion is parted.

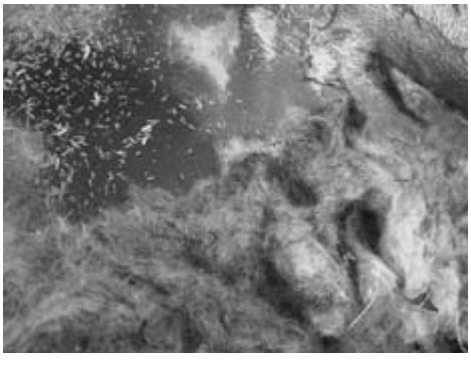


Fig. 7.56 Wool loss and skin necrosis in an advanced and neglected case of flystrike in a Jacob ewe. All stages of maggots and an adult secondary blowfly can be seen.

Fig. 7.57 The tissues overlying the cheeks of this Scottish Blackface ram were flystruck, associated with skin inflammation caused by ingrowth of its horn.

Feet affected by footrot and septic wounds such as shearing cuts, dog bites, or trauma caused by ingrowing horns (Fig. 7.57) commonly become flystruck.

The genus of maggots can be determined by allowing them to pupate in dry sawdust within a polythene bag (Fig. 7.58), then waiting for about 7 days before adult flies hatch and can be identified.

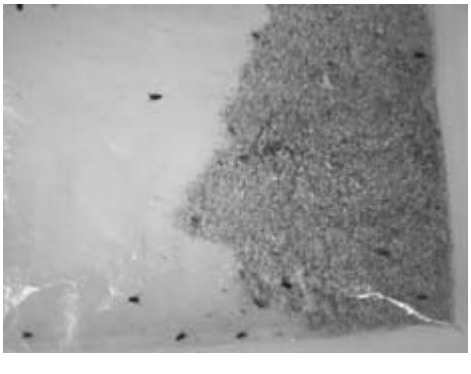


Fig. 7.58 Adult blowflies for identification developed from maggots placed in sawdust in a polythene bag.

Economic importance

Blowfly strike occurs annually in most UK sheep flocks, the incidence being highest in warm and humid areas of south-west England. It is estimated that about 12 000 sheep die each year in the UK as a result of flystrike. Flystrike is a major welfare concern and an important cause of ill thrift in individual affected animals. Furthermore, the disease results in economically significant fleece and hide damage. Prevention of blowfly strike is expensive and complex.

Control

Despite a good understanding of blowfly biology, the effective prevention of flystrike remains problematic. Blowflies can travel for several miles so, unlike lice and scab mites, they cannot be eradicated from a farm. Furthermore, while modern insecticides are extremely effective, in practice correct application of these drugs to achieve satisfactory residual activity is difficult (Table 7.2).

Farm management

The effective control of flystrike is seldom achieved by the use of insecticides alone. Freshly dead animals, faecal material and rotting vegetation provide protein for blowflies, so the prompt disposal of carcases in accordance with statutory regulations and attention to general farm hygiene can aid in their control (Fig. 7.59). Blowflies prefer a warm, moist and sheltered environment, so the risk of strike can be reduced by moving sheep to more exposed pastures (Fig. 7.60). The smell of wool grease and the presence of footrot, urine-soaked wool, skin diseases, scour, or infected cuts attract blowflies to sheep. Excessive chewing of the fleece to relieve irritation by ticks or lice can increase fleece moisture and may attract blowflies. Established strike lesions attract even more blowflies. Recently shorn sheep are seldom struck and effective control of gastrointestinal parasites and footrot, general animal health care, crutching and trimming around the pizzle can further aid in the control of flystrike.

Fig. 7.59 Prompt disposal of carcases is essential. The situation shown in this image would attract blowflies onto the farm, bringing them into close contact with live sheep.

Fig. 7.60 Moving sheep onto exposed areas during high-risk periods can reduce the incidence of blowflystrike. However, sheltered valleys and gullies can provide environments for blowflies on otherwise exposed hills.

Merino sheep are frequently struck around the crutch area, where skin folds provide a moist and attractive environment for blowflies. In some parts of Western Australia, flystrike is controlled by the crude surgical removal of these skin folds in young lambs at marking, resulting in tightening of the skin as the open wounds heal, thus reducing the attractiveness to blowflies. The processes referred to as 'mulesing' and 'tailstripping' are effective, and in some areas of Australia economic sheep production is not possible without them. However, the mulesing procedure raises serious welfare concerns, and is rightly illegal in the UK. Short tail docking is also practised overseas, although there is little evidence to show any positive benefits for flystrike control. Docking the tail above the level of the vulva of female or rectum of male lambs is illegal in the UK.

Overseas, large-scale trapping of blowflies, using insecticide-free trapping systems, has been evaluated for possible control of the Australian blowfly, *Lucilia cuprina*. For example, these systems may involve placing offal on mesh suspended above diesel within several open 45 gallon drums, distributed over a large area. Blowflies are attracted by the smell of the offal, onto which they lay their eggs. Maggots then drop out of the offal into the diesel below and are killed. The results have been encouraging, but it is unlikely that this method will prove useful in the UK, where different species of blowfly predominate. Trapping systems involving sticky paper on scented boards have been tried in the south of England, with some success in reducing the incidence of blowfly strike in sheep.

Plunge dipping

Plunge dipping in diazinon or high-cis cypermethrin (if available) can provide protection from blowfly strike for 3 to 8 weeks, depending on the product used. For optimum control sheep should have at least 3 weeks of fleece growth at the time of dipping. Care should be taken to avoid excessive faecal contamination of the dip, which can bind some of the active chemical and even enhance the attraction of dipped sheep to blowflies. To achieve best results the same principles outlined for sheep scab control apply; dags should be removed, sheep yarded overnight and feet

Table 7.2 Products for blowfly control listed in the National Office of Animal Health *Compendium of Animal Medicines* (2007).

Product	Chemical	Application method	Approximate protection	Meat withdrawal
Cooper's Ectoforce Sheep Dip	Diazinon[1]	Plunge dip	3–6 weeks	35 days
Osmond's Gold Fleece Sheep Dip	Diazinon[1]	Plunge dip	3–6 weeks	35 days
Paracide Plus	Diazinon[1]	Plunge dip	3–6 weeks	35 days
Robust	High-cis cypermethrin[2]	Plunge dip	8–10 weeks	18 days
Auriplak Fly and Scab Dip	High-cis cypermethrin[2]	Plunge dip	Up to 8 weeks	12 days
Ecofleece Sheep Dip	High-cis cypermethrin[2]	Plunge dip	Up to 8 weeks	12 days
Crovect	High-cis cypermethrin	Pour-on	6–8 weeks	3 days
Dysect Sheep Pour-on	Alphacypermethrin	Pour-on	8–10 weeks	28 days
Vetrazin Pour-on	Cyromazine (IGR)	Pour-on	10 weeks	3 days
Clik Pour-on	Dicyclanil (IGR)	Pour-on	16 weeks	20 days

[1] Organophosphate dips and ganglion-blocking anthelmintics should not be used within 14 days of each other.
[2] Marketing authorisation temporarily suspended.

Example

During mid August, 460 Scottish Blackface ewes and their 600 lambs and then 150 Texel cross Scottish Blackface ewes and their 200 Texel cross lambs were plunge dipped in diazinon to control blowfly strike. An historic, 1250 litre capacity pot dipper was used, which was sited at the bottom of a steep slope below a series of earth-based yards and a 10 m^2 concrete-based forcing pen. The initial concentration of the diazinon in the sump was calculated to the manufacturer's recommendation; thereafter the dip concentrate replenishment rate and subsequent concentrations in the sump were unknown.

Seven days after plunge dipping, about 10 percent of the Texel cross ewes and 5 percent of their lambs were flystruck with most strikes being between the shoulders or over the lumbar spine (Fig. 7.61). The Scottish Blackface ewes and their lambs which were dipped first were not flystruck. All of the Texel cross ewes and their lambs required expensive re-treatment with a pyrethroid pour-on to eliminate existing maggots and prevent further strikes.

Diazinon normally persists in the wool for about 6 weeks after correct plunge dipping, so the high incidence of blowfly strike lesions 7 days after dipping shows that there were faults in the dipping procedure. The problem was associated with excessive amounts of organic matter contaminating the dip solution, reducing its efficacy and making sheep more attractive to blowflies after dipping than they were before. The problem was compounded by the design and position of the handling pens, and failure to drain completely and refill the sump of the small pot dipper once it had become excessively contaminated with organic matter. Failure to account for stripping of diazinon when replenishing the dip solution may also have given rise to sub-lethal concentrations of chemical in the sump.

The production loss, expense and poor welfare demonstrated by this case were avoidable. Plunge dipping is a technical and skilled procedure which requires well-designed and maintained facilities. When these are unavailable, alternative methods of blowfly control such as the use of pyrethroid or insect growth regulator pour-ons should be used.

Fig. 7.61 Blowfly strike over the lumbar spine of a Texel cross lamb.

cleaned by running over slats before dipping. High concentrations of high-cis cypermethrin are required for blowfly control. High-cis cypermethrin plunge dips have relatively short meat withdrawal periods, so are useful for groups of finishing lambs.

Flumethrin plunge dips, which were licensed for the control of sheep scab, were ineffective for the control of blowfly strike.

Fig. 7.62 It is essential that shower dippers are maintained in good working order, without blocked or missing nozzles. This neglected machine is of no practical value.

Shower dipping

Many contractors now offer shower dipping services. Shower dipping can be used effectively for the control of flystrike, although it is not effective for the control of sheep scab. When using a shower dipper, it is essential that the machinery is checked beforehand (Fig. 7.62) and that sheep are dipped for long enough to ensure that the chemical reaches skin level. As a rough guide, sheep should be showered for one minute per week off shears. Saturation dipping requires at least 2 to 4 weeks of wool growth for the insecticide to bind. Shower dippers re-circulate dipwash through a sump, so the same precautions must be taken as for plunge dipping with regard to dip stripping and faecal contamination of the dipwash. None of the plunge dip solutions available in the UK are licensed for use in shower dippers and there is no available information concerning their efficacy or operator safety when applied other than by plunge dipping. Contractors using these products off-licence in shower dippers or jetting races, therefore, have no legal back-up in cases of lack of efficacy or adverse effects.

Hand jetting and automatic jetting races

Overseas, the use of hand jetting has gained popularity for the control of flystrike. Dip solution is applied using a high-pressure pump and specially designed handpiece to the breech and rump of lambs and also to the back and flanks of adult sheep. Jetting does not require expensive equipment, uses less dip solution than plunge or shower dipping, does not recycle dip so avoids stripping and faecal contamination problems and does not incur the problem of disposal of used dipwash. It is important to ensure that apparatus is working correctly and that the dip chemical is carefully applied. Alternatively, automatic jetting races can prove useful for blowfly control, provided that the apparatus is correctly maintained and sheep are kept in the race for sufficient time to ensure saturation.

Pour-ons

When applied correctly to potential areas of strike over breech and rump, pyrethroid or insect growth regulator pour-ons can provide effective control of blowfly strike (Fig. 7.63). High-cis cypermethrin pour-ons provide protection for about 6 weeks and alphacypermethrin for 8 to 10 weeks (Table 7.2).

The insect growth regulators (IGRs) are benzoylphenyl-urea (diflubenzuron, fluazuron and triflumuron), triazine (cyromazine) and pyrimidinamine (dicyclanil) derivatives, high concentrations of which mimic insect moulting hormones, disturbing

Fig. 7.63 Use of a pour on for the control of blowfly strike. Note that the operator is wearing protective gloves.

cuticle formation and leading to lethal effects during the next moult. The IGRs have low mammalian toxicity, because their target, chitin, is not found in vertebrates. Only cyromazine and dicyclanil are licensed in the UK, providing blowfly protection for 10 and 16 weeks respectively. Cyromazine causes epidermal cells of blowfly third instar larvae to invade the cuticle and produce necrotic lesions, while diflubenzuron inhibits cuticle synthesis.

The active chemicals in the pour-ons diffuse from the site of application in the emulsion layer that coats the wool fibres and then to the stratum corneum of the epidermis. Some dermal infiltration occurs, mostly at the site of application. The majority of the insecticide therefore remains close to the site of application and concentrations decrease with distance from the site of application. (Pour-ons used in cattle, such as the macrocyclic lactone pour-ons, differ from sheep pour-ons, in that they are absorbed, albeit inefficiently, and act systemically.)

Pour-ons are best applied to short-woolled sheep. No expensive equipment is required and reliance on pour-ons removes potential problems associated with licensing and spent dip disposal. Pour-on chemicals, dissolved in the wool grease, are removed when animals are shorn. In the case of pyrethroid pour-ons, this may lead to wool residue problems. Furthermore, the use of pour-ons in adult animals before shearing may be wasteful.

The timing of chemical application

Baited fly traps made from plastic drinks bottles can be used to monitor crudely the activity of blowflies, so that chemicals can be applied before problems arise (Fig. 7.64). The choice of dip chemical is partly governed by the length of protection required, which varies from as little as 2 weeks for some organophosphate plunge dips up to 16 weeks for the insect growth regulator pour-on, dicyclanil. The choice of application method depends on the product used, facilities available and time interval from shearing.

Insecticide resistance has not been demonstrated in UK blowflies. However, in Australia, New Zealand and South Africa, strains of *L. cuprina* have demonstrated an ability to develop resistance to organochloride and organophosphate insecticides. Organochloride resistance was reported in *L. sericata* in Ireland in 1968. Resistance has been reported in *L. cuprina* in Australia to the insect growth regulator, diflubenzuron. *L. cuprina* is a particularly unpleasant obligate parasite, which unlike the

Fig. 7.64 Baited flytraps can help to determine the risk of blowfly strike and optimal timing of control measures. Baited flytraps can be made from one brown and one clear plastic drink bottle. Four 2 cm^2 holes are made by cross-shaped cuts, with their flaps pushed inwards, about 4 cm from the base of the brown bottle and the upper part of the clear bottle placed over the brown bottle. A 1 cm hole is made in the cap of the brown bottle. Chopped liver or other offal is placed in the base of the brown bottle. Flies which enter the brown bottle are attracted to the clear bottle and trapped. By the time that blowflies are seen in the traps, sufficient numbers are present to initiate strike.

common UK blowfly species, breeds mostly on the bodies of living sheep attracted to the smell of moist fleeces. This life cycle may increase the risk of development of insecticide resistance.

Future options for blowfly control

Various potential control measures have been proposed including:

- genetic selection by culling animals that are persistently struck
- development from fungi of behaviour-modifying chemicals
- topical use of plant oils such as neem (*Azidirachta indica*) which have insecticidal properties
- biological control:
  - odour-baited traps
  - introduction of sterile male flies
  - infection of blowflies with *Bacillus thuringiensis*
  - entomopathogenic fungi (*Beauveria* and *Metarhizium* spp.).

Treatment of flystrike

During high-risk periods, sheep require daily inspection for evidence of flystrike. Flystruck sheep need to be treated immediately. Struck areas are sensitive to sunburn, so should not be clipped other than to gain access to the wound. A good soaking with an insecticidal organophosphate or high-cis cypermethrin dressing will then kill the maggots and protect the surrounding skin from secondary strike. It may be necessary to massage the dressing into the damaged tissue using a gloved hand. If plunge dip solution is used, it should be diluted to normal dip strength. Weak and debilitated sheep with extensive flystrike wounds require humane killing. Application of a topical aureomycin or oxytetracycline spray may aid healing by treating secondary bacterial infection.

Insect growth regulators are ineffective for the treatment of established flystrike.

Headfly

Headflies (*Hydrotea irritans*) are small, green-coloured, non-biting flies which gather around the heads of sheep in large swarms, causing considerable annoyance. While

Fig. 7.65 *Hydrotea irritans* flies feeding on exudate around a wound on the back of a Suffolk ram.

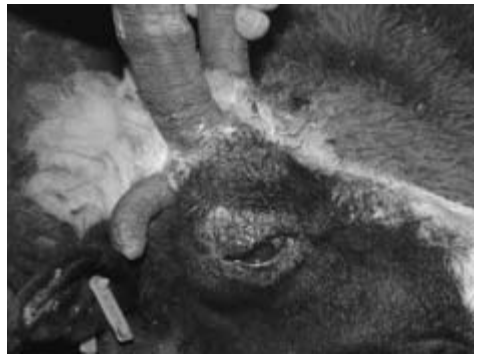


Fig. 7.66 Typical appearance of headfly damage around the eye and base of the horn of a Jacob lamb.

they are not obligate parasites, the flies feed on wounds (Fig. 7.65), for example around the eyes and base of horns (Fig. 7.66) or on the poll of rams. *H. irritans* possess rasping mouthparts, which cause extension of wounds, further serous exudation and attraction of more flies.

Headflies are active from June to September, especially given still, moist and warm weather conditions and nearby conifer woodland. Adult females lay their eggs in faeces or decaying vegetation. Larvae feed on other insect larvae and develop to maturity by late autumn. Headfly populations overwinter as resting mature larvae, which resume development in the spring, giving rise to a new generation of adults in early summer.

Headfly control is problematic. Management strategies such as moving sheep away from wooded areas or fitting protective caps to the heads of horned sheep are often impractical. Topical creams or Stockholm tar prevent skin contact and do not remove annoyance. Pyrethroid and organophosphate plunge dips afford little residual protection against headflies (Table 7.3). Cypermethrin-impregnated polymer matrix ear tags and head caps have been investigated for the control of headfly, but are not currently available.

Prevention is best achieved using a pyrethroid pour-on or spot-on treatment applied to the back of the head. Deltamethrin, high-cis cypermethrin and alpha-cypermethrin provide residual protection for about 2, 3 and 6 weeks respectively. Severely affected sheep may need antibiotic treatment in conjunction with protection of the wounds with Stockholm tar or insecticidal cream.

Table 7.3 Products for headfly control listed in the National Office of Animal Health *Compendium of Animal Medicines* (2007).

Product	Chemical	Application method	Protection	Meat withdrawal
Crovect	High-cis cypermethrin	Pour-on 5 ml to head	~3 weeks	3 days
Dysect	Alphacypermethrin	Pour-on 5 ml to head	6 weeks	28 days

Fig. 7.67 Adult sheep keds, *Melophagus ovinus*, and pupae.

Keds

Keds (*Melophagus ovinus*) are flattened, red/brown, hairy, 5 to 7 mm long, wingless flies with piercing mouthparts (Fig. 7.67), which live permanently on their sheep host, transmission between sheep requiring direct contact. Adult females produce mature larvae, which adhere to the wool and pupate immediately. Development is fastest in the summer, when adults emerge from pupae in about 3 weeks. Ked populations build up slowly, becoming heaviest in autumn and winter.

Moderate ked infestations cause annoyance, while heavy infestations can result in anaemia. Keds are common in Shetland (which was exempt from compulsory dipping for sheep scab control) and now appear to be spreading onto mainland UK. The effects of ked infestation on wool quality are minimal, but keds can cause cockle, resulting in downgrading of pelts. Measures to prevent flystrike also control keds. Keds are also killed by oral and injectable macrocyclic lactones.

Warbles

The major economic importance of warbles arises from damage to cattle hides by the third-stage larvae of the cattle warble fly *Hypoderma bovis*. While *H. bovis* has been eradicated from the UK, deer warble flies, *Hypoderma diana*, are common. *H. diana* rarely appears to be able to infect sheep (but not cattle), although its life cycle is not completed.

Warble flies resemble bees and are active in summer, attaching their eggs to hairs of the legs. First-stage larvae hatch, penetrate the skin and migrate through the host's body, overwintering in the epidural fat of the spinal canal. During the following spring, larvae moult and migrate to tissues under the skin. In their natural host, the larvae moult again and perforate the skin to create a breathing hole. At this stage, the third-stage larvae surrounded by a thick wall of granulation tissue are palpable as warbles. After about 6 weeks, larvae emerge, fall to the ground and pupate, adults emerging a few weeks later.

Deer warble flies do not complete their life cycle in sheep, simply forming nodular lesions without perforating the skin.

Warbles are susceptible to macrocyclic lactone injections. Killing warbles while in the epidural fat may very occasionally cause hind limb paralysis, associated with a severe inflammatory response to the dead larvae within the spinal canal.

Fig. 7.68 A severe, bilateral, purulent, haemorrhagic nasal discharge in a Texel shearling ram, caused by *Oestrus ovis* larvae in the nasal passages.

Oestrus ovis

Myiasis caused by larvae of the nasal botfly, *Oestrus ovis*, does not result in skin disease, but is an important disease of sheep in southern Europe and in some localised regions in southern England. Disease is occasionally seen in individual sheep as far north as the south-east of Scotland (Fig. 7.68).

Adult flies are about 1 cm long, with grey bodies which are covered by short dark hairs. The head and dorsal abdomen are distinctively marked by multiple, small black spots. Adult *O. ovis* have no mouthparts and do not feed, so significant growth and development all occurs as larvae. Adult female flies are viviparous, giving birth to first-stage larvae (L_1), which they squirt during flight into the nostrils of sheep, delivering up to 25 larvae at a time. The L_1 migrate through the sheep's nasal passages, moulting to L_2 before entering the frontal sinuses and moulting to L_3. L_3 complete their development within the frontal sinuses, growing to about 3 cm long, before migrating back through the nasal passages, dropping to the ground, pupating and completing their life cycle to adult flies. Adult female flies live for about 2 weeks, during which time they may give birth to up to 500 L_1. *O. ovis* flies overwinter in their sheep host as L_1 or L_2, which emerge as L_3 during the following spring.

Larvae migrating through the nasal passages stimulate mucus production, resulting in a nasal discharge, which is sometimes unilateral. Affected sheep may be observed to sneeze or rub their noses along the ground. Death of L_3 within the frontal sinuses may give rise to secondary infection and more severe clinical signs, occasionally resulting in death. Circling and development of a head tilt have been reported associated with severe purulent sinusitis. Sheep become anxious when active *O. ovis* flies are present, sometimes flocking together, repeatedly stamping their feet, or pressing their nostrils against the ground.

Nasal bots are killed by oral macrocyclic lactone and closantel anthelmintics, although the risks associated with dead L_3 in situ in the frontal sinuses should be considered whenever treating clinical cases. Active control of nasal bots is seldom necessary.

Humans occasionally become struck by *O. ovis*, not in the nose, but on the surface of the eye, leading in some cases to severe conjunctivitis. However, larvae on the eye do not develop beyond the L_1 stage.

Chewing lice

Infestations by the chewing louse, *Bovicola ovis*, have increased in UK sheep flocks, following the withdrawal of compulsory plunge dipping for the control of sheep scab

Fig. 7.69 Heavy louse burdens are most commonly seen in UK flocks in housed ewes during the late winter.

and greater use of systemic endectocide injections, which are ineffective against chewing lice that do not feed on systemic body fluids. Chewing lice may be present in up to 50 percent of UK flocks.

Life cycle and pathogenesis

The entire life cycle of chewing lice is spent on its sheep host. Adult lice live for about 30 days, during which time females lay two or three eggs every 5 days. White coloured, operculate eggs (nits) are firmly cemented along wool fibres. Given ideal conditions, which occur close to the skin of long-fleeced sheep, development through three larval stages to mature adult parasites takes about 30 days. A high proportion of eggs do not hatch and many nymphal lice fail to grow. Thus, the reproductive rate of chewing lice is slow when compared with mite parasites and it takes several months for heavy infestations to develop.

While the whole life cycle of *B. ovis* takes place on sheep, its bionomics are greatly influenced by climate. The winter pattern of chewing louse infestation that is seen in UK flocks is associated predominantly with fleece length and housing (Fig. 7.69). Optimum conditions for development and oviposition of *B. ovis* occur in long-fleeced sheep during autumn and winter months, but severe infestations are seldom seen until late winter. Chewing lice are intolerant of both high moisture levels and high temperatures, so populations in outdoor sheep fall dramatically following saturation of fleeces during heavy or persistent rainfall and during hot weather, influencing populations for the subsequent 6 months or more. Heavy chewing louse burdens are seldom seen in high rainfall areas.

The spread of infection between infested and clean sheep is slow. Transfer of lice between animals requires close contact, such as yarding, transport or housing, especially in cool cloudy weather. Lice do not readily drop off their hosts and do not survive for long when they do, so significant spread by indirect contact with fence posts or fomites is unlikely. *B. ovis* populations do not establish on other host species, so cattle and goats are not involved in the spread of infection.

Clinical signs

Chewing lice feeding on the skin surface bite their host, causing irritation. Affected animals may stamp their feet, rub against fences or trees (Fig. 7.70), and bite their

Fig. 7.70 Chewing louse infested sheep queuing up to rub against fence posts.

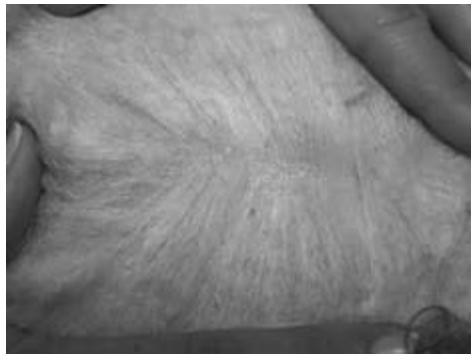


Fig. 7.71 The diagnosis of pediculosis is based on the enumeration of lice in wool partings.

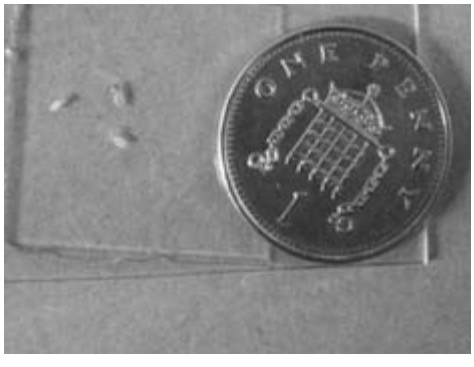


Fig. 7.72 White bodied, nymphal stages of *B. ovis* can be seen with the naked eye.

Fig. 7.73 Adult chewing lice have a broad head and brown coloured body.

shoulders, flanks and backs, breaking and displacing small tufts of wool in those areas. Eventually, the wool becomes matted and discoloured.

Diagnosis

Confirmation of a diagnosis of pediculosis is based on the identification of the lice. Chewing lice are found in fleece-covered areas of the dorsum and flanks of sheep of all ages and can be seen with the naked eye in wool partings, both on the skin surface and moving over wool fibres up to 5 cm away from the skin (Fig. 7.71). Adult *B. ovis* are about 2 mm long with a pale brown abdomen, while nymphal stages are smaller with a whitish coloured body (Fig. 7.72). Species confirmation requires microscopic examination, chewing lice having a broad head and light brown coloured body (Fig. 7.73).

Louse burdens are assessed by parting wool to expose a 10 cm band of skin at about six sites over the back and sides of the sheep. Adult lice tend to move away from light as the fleece is parted, so need to be counted quickly. As a rough guide, less than one louse per parting represents a light infestation of less than 5000 per sheep, while more than five lice per parting represents a heavy infestation of more than 250 000 per sheep.

Lice can also be detected in the vegetable matter residue following testing of wool, although there is currently no mechanism for transfer of this information in the UK. In Australia, an ELISA is used to test for *B. ovis* antigen in wool samples, and the information is relayed to the farm of origin to aid with flock disease control. The adoption of this basic monitoring process for a range of sheep diseases is overdue in the UK.

Economic importance

Chewing louse infestations vary between individuals, but the heaviest burdens are generally associated with young or old animals in poor health and low body condition. There is, therefore, a general perception that chewing louse infestations cause ill thrift and constitute a welfare problem, although it is not certain if chewing lice lead to a reduction in body condition or if they exploit sheep that are ill-thrifty for other reasons. Sheep with clinical Johne's disease frequently have very high chewing louse burdens.

There have been few controlled studies to investigate the economic effects of chewing lice in UK sheep, but based on overseas studies, it seems unlikely that moderate burdens would have any effect on bodyweight or growth rates. Heavy infestations can reduce the manufacturing quality of wool, although this is only likely to be economically significant in fine-woolled (less than 24 microns) breeds. An immune response, even to small numbers of *B. ovis*, can cause hide damage referred to as cockle which is seen in pelts after they have been tanned. Similar damage occurs with other ectoparasitic infestations such as keds and sheep scab. The presence of cockle is seldom reported to individual UK producers, and the substantial economic loss which it incurs is borne by all. Furthermore, potential hide damage will usually have already occurred by the time that the problem is diagnosed. However, should hides become traceable to the farm of origin, then chewing louse infestation would become economically important in UK sheep flocks.

Louse infestation is an important differential diagnosis for sheep scab and few producers are prepared to accept the presence of patchy wool loss and obvious discomfort in their flocks.

Louse control

Management of established louse infestations is problematic and the decision to treat chewing louse-infested sheep is not straightforward, because treatment is expensive and may result in residue problems, while it is not certain that infestations limit animal production or constitute a welfare problem. In some cases, the option of leaving animals untreated should be considered, although the rationale for, and risks associated with, this strategy must be carefully balanced.

Shearing significantly reduces chewing louse burdens by removing a large proportion of the lice and exposing those remaining to the lethal effects of desiccation. However, shearing is usually impractical during winter months when most infestations are seen. Organophosphate, pyrethroid and amide plunge dips can provide effective louse control (Table 7.4), but winter dipping and disposal of dipwash solution is difficult on many farms. Along with several other previously useful sheep pharmaceuticals, amide dips are no longer available in the UK. Shower dips are only

Case report

Ninety-nine Greyface ewe lambs were purchased during the autumn and subsequently managed as one group at pasture. Concern was raised during early February because the presence of tags of wool on fences had been noted (Fig. 7.74). Louse infestation was diagnosed on the basis of the identification of chewing lice in fleece partings along the back and flanks and an absence of *P. ovis* mites in superficial skin scrapings.

All of the yearling lambs were examined and chewing lice counted in six separate 5 cm wool partings over the thorax and pelvis and hips on each side. A mean of approximately two chewing lice per parting was identified on 94 percent of the lambs, representing about 6000 lice on each sheep. The lambs were weighed and a high-cis cypermethrin pour-on was applied along the dorsal midline of half of the flock (Fig. 7.75), while the other half were left as untreated controls. Both groups of lambs were then returned to the same pasture. Louse counting and weighing were repeated 6 and 10 weeks after treatment in mid March and April.

While treatment with a high-cis cypermethrin pour-on resulted in a significant reduction in louse counts compared to untreated control animals, no difference in weight gains of the treatment and control groups was observed. Furthermore, both groups continued to show signs of pruritus for several weeks. Similar studies conducted in New Zealand and Australia have also failed to demonstrate any significant effect of medium to heavy louse infestations on liveweight gain. The routine use of pyrethroid pour-ons in long-woolled sheep, without consideration of its potential welfare and production benefits, is irresponsible, because it selects for resistance and leads to potential drug residue problems.

Fig. 7.74 Pruritus in Greyface ewe lambs caused by heavy chewing louse burdens.

Fig. 7.75 The blue line along the backs of these ewe lambs shows a marker dye in the high-cis cypermethrin pour-on. Accurate application along the midline is difficult in long-fleeced sheep.

effective in short-woolled sheep and thorough saturation is essential. Despite common perceptions, systemic endectocides are ineffective for the control of chewing lice.

Few UK sheep farmers aim to prevent chewing lice infestations in their flocks. Most rely on the use of pyrethroid pour-ons to treat established infestations in long-woolled sheep during the winter and spring. Pyrethroid pour-ons, applied along the midline of the back, translocate over the body in wool grease. The concentration of

Table 7.4 Products for louse control listed in the National Office of Animal Health *Compendium of Animal Medicines* (2007).

Product	Chemical	Application method	Meat withdrawal
Cooper's Ectoforce Sheep Dip	Diazinon[1]	Plunge dip	35 days
Osmond's Gold Fleece Sheep Dip	Diazinon[1]	Plunge dip	35 days
Paracide Plus	Diazinon[1]	Plunge dip	35 days
Robust	High cis cypermethrin[2]	Plunge dip	18 days
Auriplak Fly and Scab Dip	High cis cypermethrin[2]	Plunge dip	12 days
Ecofleece Sheep Dip	High cis cypermethrin[2]	Plunge dip	12 days
Crovect	High-cis cypermethrin	Pour-on	3 days
Dysect Sheep Pour-on	Alphacypermethrin	Pour-on	28 days

[1] Organophosphate dips and ganglion-blocking anthelmintics should not be used within 14 days of each other.
[2] Marketing authorisation temporarily suspended.

the active chemical is influenced by: the predetermined concentration that is applied; the distance on the sheep's body from the site of application; the time interval since treatment; and the total surface area of the wool grease, which increases exponentially with wool length. Thus, a concentration gradient occurs, with smaller amounts of active chemical at sites further from the site of application, the drug taking several weeks to reach distant sites, and proportionately lower drug concentrations with increasing wool length. Pyrethroid pour-ons are expensive and only temporarily reduce louse numbers when used for the treatment of established infestations in long-woolled sheep. Animals often continue to show signs of rubbing, nibbling and scratching for up to 6 weeks after treatment, associated with the time taken to translocate over the body. Furthermore, the use of long-wool pour-ons selects strongly for insecticide resistance, as a consequence of exposure of a proportion of the louse population to sub-lethal doses of the active chemical. The use of long-wool pour-ons also presents potentially serious problems of pyrethroid residues in wool, associated with the high drug concentrations used and short interval between application and shearing.

The viscosity of the wool grease is altered during the 24-hour period immediately after shearing (off shears). When pyrethroid pour-ons are applied off shears, lethal drug concentrations translocate over the whole body within 24 hours. The shorter period of protection that is afforded when compared to application 3 weeks after shearing is irrelevant, because louse populations only increase slowly and spread of infection is slow. Thus, provided that the whole flock is correctly treated, that sheep do not become debilitated for other reasons and that significant infestations of chewing lice are not subsequently introduced with added sheep, off shears use of pyrethroid pour-ons provides a practical and responsible method for louse control for some flocks.

In Australia and New Zealand, failure of pyrethroid pour-ons and plunge dips to control chewing lice was first reported during the late 1980s and resistance is now a serious problem. In Australia, the consequential use of organophosphates to control chewing lice led rapidly to the emergence of diazinon resistance in *B. ovis*. The insect growth regulator, diflubenzuron, is used for chewing louse control in Australia and New Zealand, but is not currently marketed in the UK. Wide variations occur in drug

concentrations at different sites over the body, selecting for resistance, or perhaps augmenting existing pyrethroid resistance within a chewing louse population.

In the UK, pyrethroid-resistant *B. ovis* were identified soon after their re-emergence following deregulation of sheep scab control. The same genes appear to be involved with both DDT and pyrethroid resistance, so the intensive use of lindane dip formulations for sheep scab control between 1945 and 1984 may have pre-selected for pyrethroid resistance in chewing lice. Continued use of pyrethroids as long-wool pour-ons will rapidly increase the rate of selection for pyrethroid resistance in UK flocks.

Chewing louse numbers fall dramatically during periods of hot or wet weather, which can influence populations for the subsequent 6 months or more. In some circumstances, the prospect of such weather conditions may, therefore, remove the need to treat out-wintered long-fleeced sheep.

Sucking lice

Sucking lice (*Linognathus* spp.) are usually confined to the hair-covered parts of the face and limbs. Fleeced areas are only affected when louse populations expand rapidly. While heavy burdens can result in mild anaemia and debilitation, infestation seldom causes intense pruritus. Rams appear to be more severely affected than ewes. Sucking lice are rare (if present) in UK sheep.

Sucking lice feed on blood. Engorged adults are about 2.5 mm long with a short, pointed head and red/blue blood-filled body (Fig. 7.76). The head becomes buried in the skin during feeding.

Linognathus pedalis is generally found on the lower parts of the hindlimbs, extending to the groin, scrotum and belly in heavy infestations. Breeds without wool on their legs are most susceptible. Most infestations appear asymptomatic, although foot stamping and nibbling at affected areas may be seen in heavy infestations. Unlike the situation with chewing lice, *L. pedalis* can survive away from their host for up to 20 days, so transmission does not require direct sheep-to-sheep contact.

Linognathus ovillus occurs on the face and ears, occasionally spreading to the cheeks and neck. Face lice are occasionally seen on the tail and on the scrotum of rams. *L. ovillus* does not survive off the host for more than 2 days, so spread occurs only by close contact of sheep. Face lice are susceptible to cold, so it is unlikely that they will become a significant problem in UK flocks.

Fig. 7.76 A dark bodied sucking louse with a pointed head.

Sucking lice can be controlled by organophosphate or pyrethroid plunge dipping, or by use of macrocyclic lactone drenches or injections. Pyrethroid pour-ons are ineffective because they translocate around the body in wool grease and are not absorbed into the skin.

Ticks

Twelve species of ticks are present in the UK, with a wide variety of hosts including small ruminants, rodents, game birds and rabbits. Ticks are more important as vectors of viral, rickettsial, bacterial and protozoal diseases than they are as primary ectoparasites. Feeding activity for most ticks is optimal at humidity levels above 85 percent and mean daily average temperatures above 7°C. Thus, tick-borne diseases are mostly restricted to upland areas in western parts of the UK, where it is estimated that 300 000 lambs are affected annually, often resulting in ill thrift or death. High tick populations involved in the spread of zoonotic infections are also found in forested areas in southern parts of the UK. Heavy tick burdens on sheep occasionally cause irritation and anaemia, but the primary aim of tick control is to prevent tick-borne diseases. Tick numbers appear to be increasing in the UK, associated with climate change, reduction in hill sheep numbers as a result of economic pressures, and environmental policies encouraging sheep depopulation and regeneration of vegetation. Increasing public access to hill and moorland introduces concerns about potential human health risks associated with tick-transmitted diseases. Furthermore, there is an increasing risk of introduction of exotic ticks and tick-transmitted diseases associated with greater freedom of international movement of pet animals.

Ixodes ricinus is the most important UK sheep tick, although *Dermacentor reticulatus* and *Haemaphysalis punctata* may be significant in certain localised areas. Other tick species such as *Rhipicephalus sanguinis*, *Ixodes acuminatus* and *Ixodes trianguliceps*, which feed primarily on dogs, rabbits and small mammals respectively, may also occasionally be involved in the transmission of sheep and human diseases.

Engorged adult female *I. ricinus* ticks are about 1 cm long, grey-coloured and bean-shaped with four pairs of legs (Fig. 7.77). Male ticks are much smaller. Nymphs are less than 2 mm long with four pairs of legs and larvae are yellowish in colour and have only three pairs of legs.

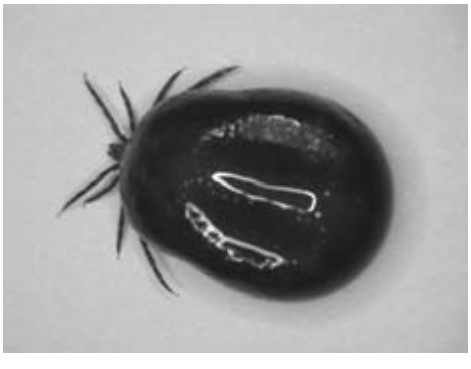


Fig. 7.77 An engorged, adult female *Ixodes ricinus* tick.

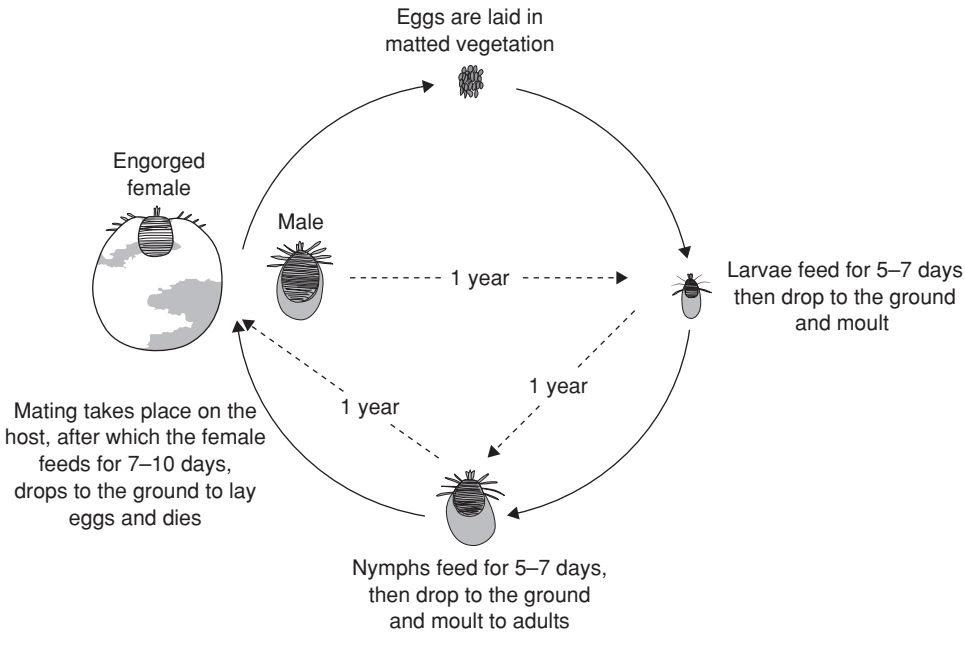


Fig. 7.78 The lifecycle of the three-host tick, *Ixodes ricinus*.

Life cycle of I. ricinus

The life cycle of *I. ricinus* from egg hatching, larva and nymph stages to adults takes 3 years (Fig. 7.78). Nymphal stages feed indiscriminately on small mammals, birds and large mammals including humans, larval stages generally feed on larger animals, while adults feed mostly on large mammals, associated with the size of their mouth parts. *I. ricinus* is a three-host tick, requiring one blood meal during each of its larval, nymph and adult stages. Each stage attaches itself to a host for long enough to feed from blood and tissue fluids, before dropping off into matted vegetation where further development or, in the case of ovigerous females, egg laying occurs. During their lifetime, ticks feed on three different hosts, thus being able to transmit diseases. Ticks only feed for about 26 days in their 3-year lifetime, most time being spent in a resting state in matted vegetation.

Survival of the unfed stages requires high humidity. In the UK, suitable tick habitats are usually found on unimproved upland or hill areas where there is a dense mat of vegetation. However, headlands and hedges around arable fields can also provide suitable habitats on individual farms outwith the traditional tick areas.

When mean daily temperatures reach 7°C, ticks climb out of the vegetation mat and quest for a blood meal. Historically reference was made to periods of UK tick activity during the spring/early summer and during the late summer/autumn. However, this seasonal nature of activity is no longer recognised, perhaps associated with climate change. Questing ticks that fail to feed within 4 to 8 weeks die. The interval between feeds is usually one year, although it can be longer. Ticks are active for longest in the milder western parts of the country due to the requirement of questing ticks for warmth and moisture.

Fig. 7.79 Control strategies involving avoidance of tick habitats are impractical for hill and upland farms.

The predilection sites for tick attachment are the head, neck, axillae and groin. Frequently one sheep will have many hundred ticks, all stages being present at the same time.

H. punctata and *D. reticulatus* are mostly restricted to parts of southern England and Wales respectively. These species are involved in the transmission of babesiosis and theileriosis, which are of little clinical significance in the UK.

Tick control

Effective tick control is not straightforward. Improvement and drainage of hill pastures is not currently an environmentally acceptable or economic option (Fig. 7.79). Grazing of sheep on improved pasture where ticks are less likely to survive during periods of peak tick activity followed by treatment before return to the hill results in a progressive reduction in tick numbers and disappearance of the tick-associated diseases. This only works, however, if there are few alternative hosts for the ticks such as deer and hares.

Control is usually aimed at preventing tick infestation of sheep (Table 7.5). Organophosphate and high-cis cypermethrin (not currently available in the UK) plunge dips afford 3 to 8 weeks' protection against ticks, while deltamethrin, high-cis cypermethrin and alphacypermethrin pour-ons afford up to 6, 8 and 12 weeks' protection respectively. However, quoted protection periods apply to fleeced parts of the body and may be shorter for hair-covered areas, such as the limbs, axilla and groin, where ticks commonly attach and feed. Amide plunge dips (amitraz), which were useful for tick control by providing a break from organophosphate dips, are no longer available in the UK. Ewes are usually treated before lambing and lambs are treated shortly after birth or before movement to high-risk areas. Treatments often have to be repeated, depending on the duration of protection that is afforded. Traditionally, many young lambs were plunge dipped at docking, or before being turned onto the hill. However, pour-on treatments have the advantage of fewer mismothering problems, fewer operator safety problems and greater ease of treating small groups of sheep.

Plunge-dipping or pour-on treatments can be valuable in preventing ticks from spreading onto previously uninfested areas, for example when sheep from infested areas are moved during the tick season onto previously tick-free land.

Pyrethroid resistance is suspected in *I. ricinus* ticks in the UK, but difficult to prove.

Table 7.5 Products for tick control listed in the National Office of Animal Health *Compendium of Animal Medicines* (2007).

Product	Chemical	Application method	Approximate protection	Meat withdrawal
Cooper's Ectoforce Sheep Dip	Diazinon[1]	Plunge dip	3–6 weeks	35 days
Osmond's Gold Fleece Sheep Dip	Diazinon[1]	Plunge dip	3–6 weeks	35 days
Paracide Plus	Diazinon[1]	Plunge dip	3–6 weeks	35 days
Robust	High-cis cypermethrin[2]	Plunge dip	8–10 weeks	18 days
Auriplak Fly and Scab Dip	High-cis cypermethrin[2]	Plunge dip	Up to 8 weeks	12 days
Ecofleece Sheep Dip	High-cis cypermethrin[2]	Plunge dip	Up to 8 weeks	12 days
Crovect	High-cis cypermethrin	Pour-on	6–8 weeks	3 days
Dysect Sheep Pour-on	Alphacypermethrin	Pour-on	8–12 weeks	28 days

[1] Organophosphate dips and ganglion-blocking anthelmintics should not be used within 14 days of each other.
[2] Marketing authorisation temporarily suspended.

Tick-transmitted diseases

The major importance of ticks in the UK is the transmission of tick-borne fever and louping ill and their role in the pathogenesis of tick pyaemia. Tick infestation of sheep is also important because of its potential role in the epidemiology of human diseases such as Lyme disease and Q fever.

Tick-borne fever

Tick-borne fever is caused by infection of white blood cells of susceptible sheep by the ixodid tick-borne rickettsial (similar to protozoan parasites) organism, *Anaplasma phagocytophilia* (previously referred to as *Cytocoetes* or *Ehrlichia phagocytophilia*). On affected farms, the entire nymph and adult tick population may be infected with *A. phagocytophilia*, having previously fed on persistently infected hosts. *A. phagocytophilia* establishes in the tick gut epithelium and infection is transmitted by regurgitation of gut contents during feeding. In endemically infected areas and in the absence of control measures, young lambs are infected within the first 2 weeks of life. The greatest risk of disease therefore occurs in newborn lambs and introduced, naïve pregnant ewes.

Within 24 hours of infection with *A. phagocytophilia*, sheep develop a high fever and pronounced neutropenia, lymphopenia and thrombocytopenia. The fever and leucopenia may persist for up to 3 weeks. White cell numbers return to normal within 2 to 3 days of fever having regressed. Most infected animals become carriers of infection and periodically suffer spontaneous recurrences of parasitaemia. Cattle, deer and feral goats can also be infected by *A. phagocytophilia*, potentially acting as reservoirs of infection.

In most cases, primary disease due to *A. phagocytophilia* manifests as no more than a brief period of malaise, with pyrexia and reduced appetite. However, infection is important due to its profound effect on the immune system, occasionally leading to severe haemorrhagic diarrhoea, weakness and anorexia. Naïve pregnant ewes may abort and prolonged fever may inhibit spermatogenesis in rams. Tick-borne fever is

also important because it predisposes to other infectious diseases, in particular, louping ill, polyarthritis and respiratory infections.

The severity of haemorrhagic enteritis, involving systemic mycotic infection, in 3- to 6-week-old lambs is increased when tick-borne fever and louping ill occur concurrently, resulting in high mortality rates. Severe disease seldom occurs in recognised upland tick areas where most lambs have maternally derived immunity against louping ill, but is seen when lambs lack passive immunity against louping ill, for example where their naïve dams are moved onto tick areas, or where new tick habitats are established.

The diagnosis of tick-borne fever is based on knowledge of tick activity, haematological findings of leucopenia and thrombocytopenia, and demonstration of *A. phagocytophilia* inclusion bodies in cytoplasmic vacuoles of monocytes and neutrophils in Giemsa-stained blood smears. Blood smears from about five animals should be examined, as inclusion bodies will not be detected in every case at a single sampling. Exposure to *A. phagocytophilia* can be demonstrated from about 2 weeks after infection using a counter immuno-electrophoresis (CIE) test. The diagnosis is supported by postmortem findings of splenic enlargement and identification of ticks or sites of tick bites in the axilla and groin of affected lambs.

Avoidance of tick-infested areas is seldom practical, so in most cases prevention of tick-borne fever relies on the application of a pyrethroid pour-on to young lambs as they are turned onto tick-infested areas. Hill ewes in endemically infected areas are routinely brought off the hill for lambing, to ensure that their newborn lambs can be treated before they are exposed to *A. phagocytophilia*-infected ticks.

A. phagocytophilia is sensitive to oxytetracycline and, on some farms, tick treatment is combined with a single metaphylactic injection of long-acting oxytetracycline. This strategy is only effective when antibiotic treatment is given at the time of, or 5 days before, infection and may be difficult to justify economically. Exposure of naïve pregnant ewes to tick-borne fever can have disastrous consequences with up to 90 percent abortions and high mortality rates. Pregnant animals should, therefore, never be moved from tick-free to tick-infested areas.

Louping ill

Louping ill is a diffuse, non-suppurative meningoencephalomyelitis, caused by an ixodid tick-transmitted flavivirus. The disease is confined to certain tick-infested areas and has a predominantly seasonal occurrence reflecting tick feeding patterns with most, but not all, cases seen during the spring or autumn. Louping ill virus infection of sheep and in red grouse is a potentially economically important problem on many Scottish and northern English hill farms.

Naïve sheep of all ages are susceptible to louping ill, but following exposure, develop protective immunity. In permanently stocked, endemically infected areas, young lambs are protected by colostral antibodies, so most cases of louping ill occur in weaned lambs and are associated with autumn tick activity, or in yearling sheep associated with spring tick activity. Young lambs with insufficient colostral antibody protection often die. When naïve sheep are introduced to an infected area, or when ticks in a previously louping ill-free area become infected following movement of infected hosts, disease is seen in all age groups (Fig. 7.80).

Following initial infection, viral replication occurs in the lymph node draining the site of the tick bite. Thereafter, infected animals become viraemic for a period of

Fig. 7.80 Louping ill infection is an important cause of poor ram breeding soundness when naïve animals are introduced to louping ill infected tick-infested areas before the breeding season.

about 3 days, before the louping ill virus enters the Purkinje cells of the central nervous system and clinical signs may develop. Serum antibodies are then produced and the virus eliminated. Larval and nymph stage ticks become infected when they ingest a blood meal from a viraemic host, and transmit disease the following year when they feed as nymph and adult stage ticks respectively.

The clinical signs associated with louping ill are variable and include ataxia, seizure activity and opisthotonus. Some cases present as sudden deaths. When sheep are concurrently affected by tick-borne fever, the disease is much more severe, with very high mortality rates in affected sheep. Residual torticollis or posterior paralysis of several months' duration has been reported in recovered sheep. Flock morbidity rates in infected areas range from 5 to 60 percent.

The diagnosis of louping ill is based on clinical signs and knowledge of infected tick activity. The diagnosis is supported by histopathological findings of non-suppurative meningoencephalomyelitis and can be confirmed using a plaque assay for virus isolation from brain tissue, real-time PCR, or by brain histopathology using virus-specific stains. Exposure to louping ill virus can be demonstrated serologically using an ELISA or haemagglutination inhibition test. A wide range of animal species can be infected, without showing signs of disease or being involved in the transmission of infection. Thus, serology from hares provides a good indicator of the presence of louping ill virus.

There is no effective treatment for louping ill, so the primary focus must be on control or prevention. Tick control involving the frequent use of pour-ons or plunge dipping, is important. However, the application of these methods often has to coincide with gathering for other management reasons, so prevention of exposure to ticks throughout the risk period for louping ill is impractical. An effective vaccine is available, based on a killed louping ill virus in an oil adjuvant. On farms where the disease is endemic, all ewe and ram lambs to be retained for breeding are given a single subcutaneous injection of the vaccine in the autumn or during the following spring before the ticks become active. In addition, any introduced sheep are vaccinated once, at least 28 days before exposure to the tick-infested pasture. If the disease occurs for the first time on a farm, it may be necessary to vaccinate the entire flock. The vaccine is expensive, costing about £3.00 per 1 ml dose, but this cost is readily justified when compared to the cost of the disease.

Total eradication of louping ill through the sustained and concerted use of pour-ons, plunge dips and vaccination is seldom successful as a long-term strategy, perhaps associated with reservoirs of infection in feral sheep and goats.

Ixodes ricinus will feed on any vertebrate species, transmitting louping ill virus in the process. Clinical louping ill is most important in sheep and sometimes seen in domestic cattle, goats and llamas, but is very rare in wild mammals and birds apart from the red grouse, willow grouse and ptarmigan. Only sheep and red grouse develop a level of viraemia above the threshold necessary for transmission of infection to ticks. Thus, removal of sheep reduces, but does not necessarily eliminate, the natural reservoir of louping ill virus infectivity.

Larval and nymph stages of *I. ricinus* feed on a variety of smaller animals, including red grouse. Louping ill virus infection of red grouse is generally fatal, resulting in significant losses in some areas. However, the grouse are not important as reservoirs of infection, because they die during the viraemic stage of the disease. Thus, control of louping ill in red grouse depends on control of the infection in sheep. The importance of red grouse to the economy of many hill areas underpins the need to control louping ill in sheep.

The louping ill virus is closely related to the tick-borne encephalitis virus which is an important cause of human disease in mainland Europe. However, human infection with louping ill virus is very rare and mostly reported in laboratory workers. The fact that extremely serious human disease due to louping ill virus infection is rare may be related to an effect of cool environmental conditions on the virus within the tick, rather than the inherent pathogenicity of the virus, raising concern about a possible consequence of climate change.

Human infection can result from contact between skin cuts and blood from viraemic animals, so precautions must be taken when handling animals which might be infected with louping ill virus, including the wearing of protective latex or nitrile gloves. Postmortem examination of potential cases of louping ill should only be performed in a veterinary laboratory, where brain tissue should only be removed in an extraction cabinet to reduce the risk of aerosol exposure to louping ill virus. Louping ill virus infection can also occur orally, through ingestion of uncooked meat from viraemic carcases or of infected sheep milk.

Tick pyaemia

The feeding activity of ixodid ticks may result in intradermal inoculation of staphylococcal bacteria, either derived from the skin surface or from the tick mouthparts and saliva. When aided by the immunosuppressive effects of tick-borne fever in young lambs, this can lead to dermal abscessation and bacteraemia, with subsequent sepsis in joints, the vertebral column and other sites, referred to as tick pyaemia.

The main clinical signs associated with tick pyaemia are severe lameness, usually involving one or two limbs, and pelvic limb paralysis, associated with septic arthritis and vertebral body abscessation respectively. Affected joints become markedly distended with pus, but seldom form discharging sinuses. Ill thrift and death associated with abscessation of internal organs is sometimes seen. Clinical cases are seen from about 2 weeks after exposure to ticks, with up to 5 percent of lambs affected, although the true incidence of the disease is unknown, because severely affected lambs may be predated before they can be identified.

Treatment of cases of tick pyaemia is unrewarding and merely prolongs suffering. Control depends on reducing exposure to ticks using pyrethroid pour-ons. Metaphylactic long-acting oxytetracycline treatment of 3-week-old lambs, in conjunction

with control of tick-borne fever, may reduce the incidence of clinical cases, but the economic benefits of treatment are difficult to define.

Babesiosis

Babesia spp. are tick-transmitted protozoan parasites of red blood cells. Babesiosis in sheep caused by *Babesia ovis* and *Babesia motasi* occurs mostly in southern Europe and subtropical areas, where it is generally subclinical in indigenous sheep, but can cause severe disease in introduced animals. UK sheep can be infected with *B. motasi* and *Babesia capreoli* (a red deer parasite), transmitted by *H. punctata* and *I. ricinus* respectively. Unlike the situation with *Babesia divergens* infection of UK cattle, referred to as redwater, babesiosis in UK sheep seldom results in significant disease.

Ticks can become infected when they ingest a blood meal form a parasitaemic host. Within the tick, the *Babesia* spp. probably reproduces sexually, and is transmitted both between larval, nymph and adult stages, and transovarially, by infection of eggs by a vermicule stage of the parasite. *Babesia* spp. enter the salivary glands of the ticks and are transmitted to their mammalian host by the feeding activities of larvae, nymphs and adults.

Rapid, asexual division of the *Babesia* spp. parasite within its mammalian host's red blood cells leads to haemolysis. The severity of subsequent disease depends on the pathogenicity of the *Babesia* spp., host immunity and the level of challenge. Up to 50 percent of the red blood cells may be destroyed, leading to serious disease. The clinical signs include pyrexia, tachypnoea and tachycardia, with anaemic or jaundiced mucous membranes and haemoglobinuria. Affected animals may abort.

The diagnosis of babesiosis is based on knowledge of *Babesia* spp.-infected tick activity and can be confirmed by the identification of the parasite in Giemsa-stained red blood smears.

Babesiosis can be effectively treated using diminazene aceturate and imidocarb dipropionate, but treatment is seldom necessary in UK sheep.

Q fever

Infection with the rickettsial organism, *Coxiella burnetti*, is referred to as Q (Queensland) fever. Q fever is an occasional cause of abortion in sheep, goats and cattle, but is most important as a zoonotic disease. Human Q fever is an occupational disease of sheep farmers, abattoir workers and farm animal vets. Infection with *C. burnetti* is usually asymptomatic but occasionally results in serious influenza-like illness, sometimes followed by pneumonia, hepatitis or endocarditis.

C. burnetti is distributed worldwide and can survive for many years in the environment. Humans are mainly infected by aerosols arising from foetal fluids of lambing or aborting ewes or ingestion of contaminated milk or faeces. Ticks are probably unimportant in the transmission of Q fever to humans, but play a role in the maintenance of infection in sheep either directly during feeding or indirectly by contact with tick faeces.

The diagnosis of Q fever abortion in sheep is based on the identification of small red intracellular and extracellular coccoid bodies in modified Ziehl-Neelsen stained smears of foetal stomach contents or placenta. Colonies of the organism appear

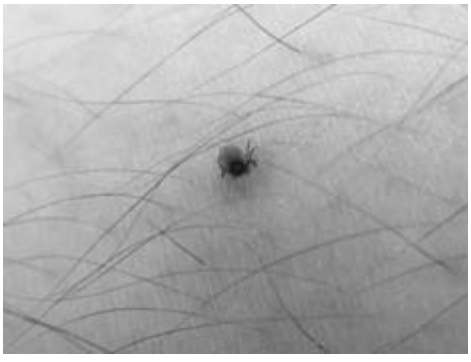


Fig. 7.81 A feeding *Ixodes ricinus* nymph on the ankle of a hill walker. The erythematous ring around the tick is due to the injection of anticoagulants. The infectious dose of *B. burgdorferi* is related to the time for which the infected tick feeds. It is therefore important to remove any ticks as soon as they are identified, using tweezers firmly placed as close as possible to the skin to draw the tick away at right angles to the skin, so as not to leave detached mouthparts behind.

sparser than those of *C. psittaci*, which is seldom present in foetal stomach contents. Serology using a complement fixation test can be used to identify previous exposure.

Lyme disease

Lyme disease is caused by the spirocheate *Borrelia burgdorferi* which is transmitted by ixodid ticks (Fig. 7.81). All mammals are infected and disease is occasionally reported in dogs, cats and hares. The role of sheep and deer as reservoirs for Lyme disease is controversial. The primary importance of Lyme disease is as a zoonosis, with about 600 human cases reported every year in the UK. The incidence of human disease could increase, because of increased access to hills, the spread of tick habitats and growing tick populations.

While *B. burgdorferi* is widespread in UK tick populations and human infection is common, in most cases seroconversion occurs without overt disease. When Lyme disease occurs, the principal clinical sign is the development of a red, inflamed ring, referred to as 'erythema migrans', spreading from the site of a tick bite. In rare cases, serious systemic disease may follow, so it is important that erythema migrans is recognised promptly and effective penicillin or oxytetracycline antibiotic treatment is instigated.

Non-parasitic skin diseases

Non-parasitic skin diseases are common in UK flocks. While they do not all result in significant economic loss, most are important as differential diagnoses for the important ectoparasitic infestations. Skin diseases are often a result of mixed infections, complicating their diagnosis and treatment. Nervous diseases such as scrapie should also be considered as differential diagnoses for skin disease because the clinical signs may include intense pruritus and wool derangement.

The common non-parasitic skin diseases of UK sheep are:

- orf
- dermatophilosis
- photosensitisation
- pyodermas.

However, other less common skin diseases sometimes limit production in individual flocks.

Orf

Orf (also referred to as scabby mouth, contagious pustular dermatitis and contagious ecthyma) is a common, highly contagious, production-limiting disease of sheep, goats and deer caused by a parapox virus. The disease is characterised by proliferation of the epidermis and scab formation.

The parapox virus can survive for several months in a cool (<7°C), dry environment, but is destroyed by high and very low temperatures, ultraviolet light and wetting. The virus may, therefore, be carried over from year to year in buildings. Sheep become infected by contact with a scab-contaminated environment or by direct contact with other infected animals. Breaks in the skin, for example associated with tooth eruption in young lambs, or traumatic handling during plunge dipping in older sheep, facilitate entry of the virus. Proliferative lesions then result from multiplication of the virus in epidermal cells, cell death and repair. The disease is self-limiting and most cases resolve within 4 to 6 weeks. Protective immunity in sheep following infection appears to be short-lived and the disease can recur, but with reduced severity. In most cases, persistently infected carrier animals are probably responsible for the annual reappearance of orf within a flock. Lambs are not protected by colostral antibodies from previously exposed dams.

Clinical signs

The first sign is erythema and a slight swelling of the skin (Fig. 7.82). Blisters then form (Figs 7.83 and 7.84), which develop into pustules (Fig. 7.85) which subsequently rupture to form thick scabs. After about 3 weeks, scabs are shed, leaving a layer of raw skin which heals quickly. A subsequent cycle of proliferation and repair results in the development of obvious proliferative, scab-covered lesions. Secondary bacterial infection of the lesions often occurs and scabs bleed if damaged, progressing to wart-like outgrowths in some animals (Fig. 7.86).

Characteristic lesions are most commonly seen on the muzzle (Fig. 7.87), around the margins of the lips (Fig. 7.88) and around the tooth margin of the gums (Fig. 7.89) of 2- to 6-week-old lambs, or recently weaned lambs and hoggs. In severe cases lesions extend inside the mouth and may involve the tongue and dental pad. Severe secondary infection of orf lesions by *Staphylococcus aureus* and *Dermatophilus*

Fig. 7.82 Erythema and swelling of the skin over the nose of a 5-day-old lamb.

Fig. 7.83 A blister on the lower gum of a week-old lamb.

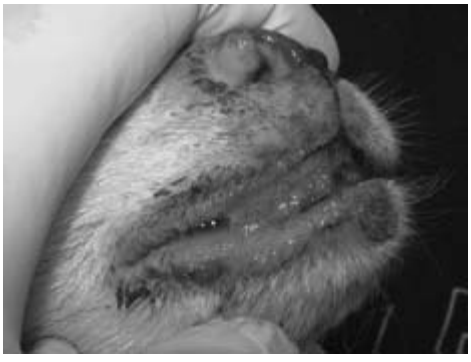


Fig. 7.84 Blisters, covered by frothy saliva in a week-old lamb.

Fig. 7.85 Pustules on the upper lip of a week-old lamb.

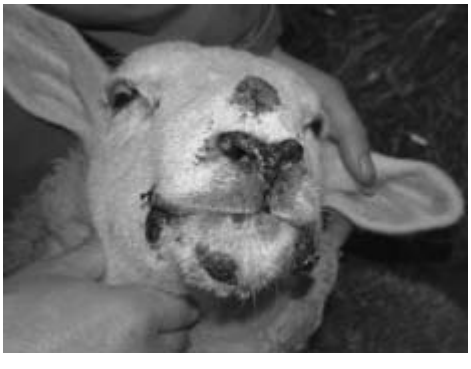


Fig. 7.86 Wart-like growths on the lips, chin and nose of a 3-week-old lamb.

Fig. 7.87 Characteristic orf lesions on the muzzle and around the margin of the lips.

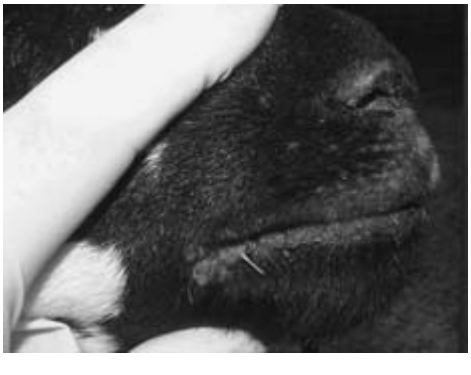


Fig. 7.88 Characteristic orf lesions at the angle of the lips.

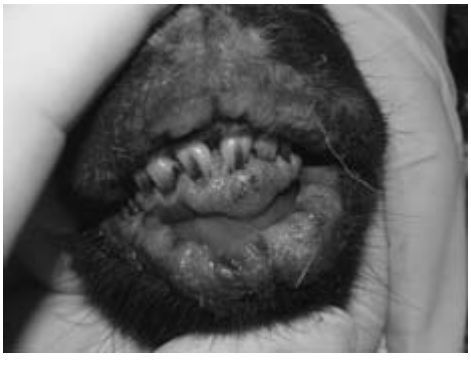


Fig. 7.89 Advanced orf lesions involving the lips and tooth margins of the gums of a 6-month-old Scottish Blackface ram.

Fig. 7.90 Secondary infection of orf lesions following grazing long grass during persistent wet autumn weather.

Fig. 7.91 Orf-like lesions around the base of the teats of a recently lambed ewe. Orf virus can sometimes, but not consistently, be identified in scabs from these lesions.

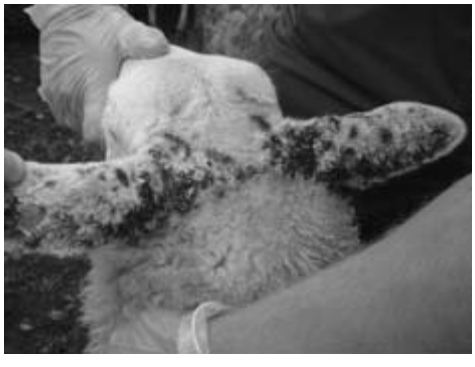


Fig. 7.92 Orf lesions over the pinnae of the ears and poll of a 5-month-old Texel cross lamb.

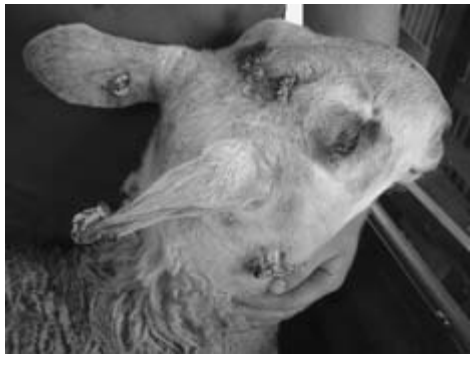


Fig. 7.93 Wart-like orf lesions on the face and ears of a shearling Bluefaced Leicester ram. The lesions first appeared around the site of an ear tag, which has been removed.

congolensis sometimes occurs, associated with close confinement, trough feeding, long grass and persistent wet weather (Fig. 7.90).

Orf lesions are sometimes seen around the base of the teats of ewes (Fig. 7.91). While the disease is usually self limiting, economic losses result from failure of severely infected young lambs to suck, poor weight gains in hoggs and secondary mastitis in ewes.

Orf lesions are occasionally seen at other sites such as the ears (Fig. 7.92) sometimes associated with tagging (Fig. 7.93); between the digits, around the coronary band (Fig. 7.94) and over the fetlock joints (Fig. 7.95), clinically indistinguishable from ovine digital dermatitis; on the plantar aspect of the lower limbs (Fig. 7.96) sometimes associated with persistent wet conditions and *Dermatophilus* infection; around the vulva of ewes; and on the penis of rams. Secondary *Fusobacterium necrophorum* infection of lesions on the penis (Fig. 7.97) and vulva results in the formation of necrotic ulcers.

Fig. 7.94 An orf lesion between the digits and above the coronary band of a shearling Cheviot ram.

Fig. 7.95 A typical orf lesion over the fetlock of a 5-month-old Texel cross lamb.

Fig. 7.96 An orf lesion on the plantar aspect of the lower limb of a 6-month-old Mule ewe lamb.

Fig. 7.97 A secondarily infected orf lesion on the penis of a Suffolk shearling ram.

The incidence of orf within a group of sheep may exceed 50 percent. High levels of infection are associated with the housing of January/February-born lambs, intensive stocking of recently weaned lambs, or contamination of equipment used for feeding orphan lambs. Young lambs can become infected from lesions on their dam's teats. Scratches around the mouth caused by whins or thistles can facilitate spread of infection in hill and upland flocks (Fig. 7.98). Severe outbreaks of orf involving the skin over the thorax and flanks (Fig. 7.99) or hindquarters (Fig. 7.100) have been reported following trauma and overcrowding after plunge dipping in unhygienic conditions. The spread of infection in pet lambs can be particularly rapid, outbreaks lasting 6 to 8 weeks.

Orf is an important zoonosis. Painful lesions in humans are usually seen on the fingers (Fig. 7.101), but can occur at any site. Blisters progress to weeping sores, which persist for several weeks. The disease is often complicated by potentially serious lymphadenitis and flu-like symptoms. Human infection should be avoided by good hygiene and wearing of latex or nitrile gloves when handling infected animals. The same precautions should be taken when handling the live orf vaccine.

Fig. 7.98 Grazing among thistles increases the risk of transmission of orf.

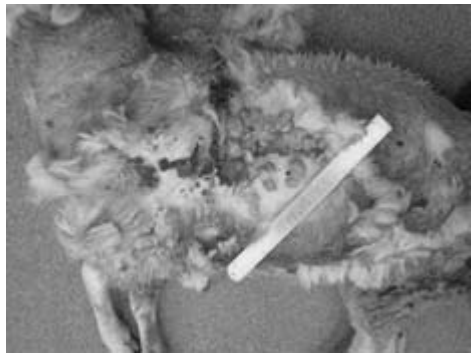


Fig. 7.99 Atypical orf lesions over the thorax and flanks of a 5-month-old Texel cross lamb.

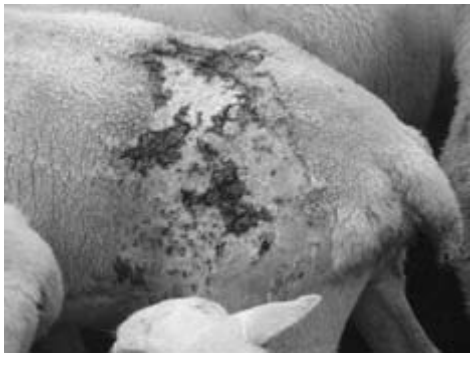


Fig. 7.100 Atypical orf lesions over the hindquarters of a Texel cross Scottish Blackface ewe.

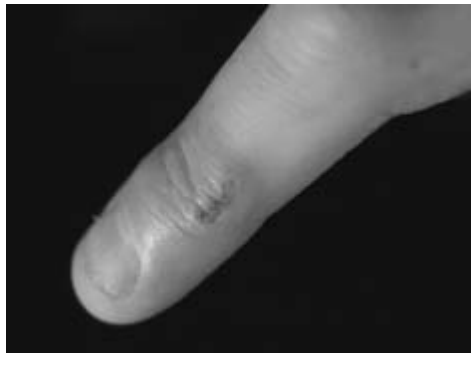


Fig. 7.101 An early orf lesion on the index finger. Gloves should always be worn when handling potentially orf-infected sheep.

Diagnosis

The diagnosis of orf is based on the clinical signs. Early lesions may resemble those of important notifiable exotic viral diseases, such as foot-and-mouth, bluetongue and sheep pox. The diagnosis can be confirmed by recovery of virus in cell culture or electron microscopy on moist or proliferative scab material. Dry scabs are of limited diagnostic value.

Management

There is no proven treatment for orf. The severity of orf outbreaks can be reduced by avoiding overcrowding, or contact with whins or thistles, although these are seldom practical options. Oxytetracycline aerosol sprays can be applied to control secondary infection, which may reduce the severity of the lesions. Severely infected animals may also benefit from parenteral antibiotic injections. Provision of palatable, soft, supplementary feed is important in cases where severe mouth lesions are present. There is no reliable evidence to support the use of sulphur-based oral capsules that are marketed for the treatment of orf. Furthermore, administration of these unproven remedies incurs a high zoonotic risk.

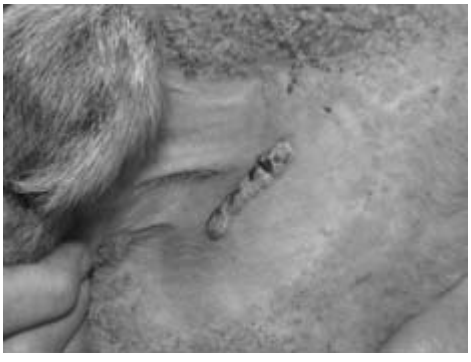


Fig. 7.102 Orf vaccination site in the axilla.

Fig. 7.103 Atypical orf lesions inside the groin of a 2-month-old Bluefaced Leicester lamb.

Prevention

Orf can be managed by vaccination. In most flocks, the preferred methods are to vaccinate housed early-born lambs as they are turned out of lambing pens and older lambs at weaning. Alternatively, ewes can be vaccinated at least 8 weeks before housing or lambing, with the aim of reducing the level of infection at lambing from bedding or teat lesions respectively. The vaccine is live, so it is important that sufficient time elapses for scabs to heal, between vaccination of the ewes and lambing. Vaccination can also be used to protect in-contact sheep in flocks where the disease has appeared, being very useful when signs are seen in less than 10 percent, but of little value once 50 percent are affected. Vaccination immunity only lasts for about 6 to 8 months and vaccination of ewes affords no colostral protection to lambs. The vaccine strain of the virus can produce disease, so its use should not be considered in flocks with no history of orf.

Application is by scratching the skin twice with a scarifying needle dipped in vaccine. Young lambs can be vaccinated on the skin between the top of the foreleg and the chest wall, where they are unable to lick. Older lambs are usually vaccinated inside the thigh and ewes on the skin under the tail. The prongs of the applicator should be wiped regularly to remove any build-up of grease. The vaccine is live, so should not come into contact with disinfectants and animals should not be scratched so vigorously as to make them bleed. The scratch sites of a few animals should be inspected after about 1 week for evidence of rows of small pustules, which indicate that vaccination has been effective (Fig. 7.102). Vaccines should be kept out of direct sunlight, refrigerated between 2 and 8°C, and used before the expiry date.

Unusual orf lesions have been reported in the groin of Bluefaced Leicester lambs within a few weeks after orf vaccination in the axilla (Fig. 7.103).

Dermatophilosis

Dermatophilosis (also referred to as mycotic dermatitis and lumpy wool) is present in all sheep flocks, but the severity of the disease varies depending on wool type and weather conditions. Short-woolled breeds are most susceptible and severe disease outbreaks usually follow prolonged periods of wet weather.

Fig. 7.104 Scab-covered papules on the pinna of the ear.

Fig. 7.105 Scab-covered papules over the bridge of the nose.

Fig. 7.106 Moderate dermatophilosis spreading from the lumbar dorsum over the flanks of a 5-month-old Suffolk cross lamb.

Fig. 7.107 Crusting scabs cemented to the wool fibres.

Clinical signs

The most common clinical presentation of mycotic dermatitis in sheep is papule formation on the pinnae of the ears (Fig. 7.104) and nose (Fig. 7.105). Accumulation of dried exudate gives the lesions a crusting appearance. Following periods of wet weather, coccal forms of the causal bacterium *D. congolensis*, present in lesions on the ears, become flagellate and motile, and invade the warm moist epidermis of the back, resulting in low-grade, moderate or severe infection.

Low-grade infection is characterised by diffuse scaly dermatitis along the dorsum, particularly in the lumbar region and extending downwards over the flanks. In the moderate form of the disease, serum exudation and the formation of moist grey/yellow scabs adherent to the proximal 1 to 5 mm of wool are observed when the fleece of the lumbar dorsum is parted (Fig. 7.106). As the scabs dry, wool fibres are cemented together, giving rise to a 'lumpy wool' appearance. The scabs are carried away from the skin surface as the wool grows (Fig. 7.107). In the severe form of the disease, which usually follows prolonged periods of wet weather, serous exudation is pronounced, and the lesions extend downwards over the flanks

Fig. 7.108 Severe dermatophilosis, with scab formation leading to wool loss over most of the body of a 6-month-old Suffolk cross lamb.

Fig. 7.109 Severe dermatophilosis around the muzzle of a 6-month-old Suffolk cross lamb, associated with persistent heavy rain.

Fig. 7.110 Low-grade dermatophilosis along the site of application of a cyromazine pour on 8-month-old Suffolk ram lambs.

(Fig. 7.108). Severely affected sheep may be pruritic, leading to self-inflicted excoriation and wool loss. Alopecic areas are covered by thick scabs formed from dried serous exudate.

D. congolensis often secondarily infects other skin lesions, such as those seen around the muzzle and over the nose of lambs grazed on long, wet pasture, exacerbating the clinical disease (Fig. 7.109). Dermatophilosis lesions are sometimes seen along the site of application of insect growth regulator pour-ons (Fig. 7.110).

Diagnosis

Dermatophilosis is differentiated from economically more important ectoparasitic diseases by the distribution of the lesions, the degree of crust formation and the low-grade pruritus. The diagnosis of dermatophilosis is supported by microscopic examination of impression smears from the underside of moist scabs, air dried, fixed in methylated spirit and stained with Giemsa or Gram stain to demonstrate characteristic coccoid or parallel rows of branching mycelium forms of *D. congolensis*. Bacterial culture can be used as a confirmatory test.

Fig. 7.111 Healing dermatophilosis lesions around the muzzle of a 6-month-old Texel cross lamb.

Fig. 7.112 A swollen head in a Greyface ewe, 3 days after turnout after lambing onto lush green pasture.

Control

Treatment of established cases of mycotic dermatitis, where crusting scabs are cemented to the wool fibres, can be problematic. Long-acting antibiotic injections can be useful in severely affected animals. Clinical disease outbreaks are often associated with plunge dipping and the addition of 10 kg zinc sulphate per 1000 litres of dip (check first for compatibility) may help to prevent the problem. Newly dipped sheep should be let out of yards as soon as possible to allow the fleece to dry.

D. congolensis-infected lesions around the muzzle and over the nose of lambs grazed on long, wet pasture (Fig. 7.109) respond within a few days to antibiotic treatment using daily penicillin or long-acting oxytetracycline injections (Fig. 7.111).

Photosensitisation

Photosensitisation (also referred to as plochteach, alveld and yellowses) results from the accumulation of chlorophyll metabolites including phylloerythrin under the skin, and their reaction with sunlight, to cause necrotic skin damage. Cases of photosensitisation occur either as a primary condition or secondary to hepatotoxic damage.

Most cases of photosensitisation in the UK follow the movement of sheep from poor to lush green pasture such as silage aftermaths, or occur following turnout of ewes after winter housing and lambing (Fig. 7.112). These cases are associated with failure of the liver to adapt to increased amounts of dietary chlorophyll.

Primary photosensitisation sometimes follows ingestion of photodynamic agents that are absorbed and carried systemically to the skin. St John's wort (*Hypericum perforatum*), which in the UK is occasionally found in hedgerows and scrub ground, contains hypericin, and buckwheat (*Fagopyrum esculentum*), in other countries, contains fagopyrin. Clinical disease occurs within 2 to 3 days of exposure to these agents.

Hepatogenous photosensitisation occurs when the biliary excretion of phylloerythrin, a degradation product of chlorophyll that is normally excreted in bile, is obstructed due to liver disease. Hepatogenous photosensitisation is associated with the ingestion of a number of plant or fungal toxins, or may be due to an inherited defect in Southdown and Corriedale sheep. In many Scottish hill areas, annual outbreaks with up to 10 percent morbidity occur in lambs during early summer,

Fig. 7.113 Bog asphodel growing on coastal grazing in Wester Ross.

Fig. 7.114 Swelling of the face and ears of a 4-month-old Cheviot lamb.

Fig. 7.115 Necrosis of the skin of the lower limbs of a Shetland ewe.

Fig. 7.116 Plochteach lesions over the lumbar dorsum of a 4-month-old Scottish Blackface lamb. The skin underlying the saddle-shaped region over the back is necrotic and feels hard.

probably associated with the ingestion of bog asphodel (*Narthecium ossifragum*) (Fig. 7.113).

In most cases, clinical signs are confined to wool-free unpigmented areas of the head (Fig. 7.114) and limbs (Fig. 7.115); however, in cases of plochteach in Scottish Blackface sheep, the skin of the midline of the back at the parting of the fleece is also affected (Figs 7.116 and 7.117). Affected lambs are anorexic, seek shade (Fig. 1.18), and rapidly lose body condition. Oedema of unpigmented areas of the face and of the dorsal midline is followed by seepage of serous fluid which dries to form yellow crusts. Skin necrosis, sloughing and regeneration follow over a period of several months (Figs 7.119 and 7.120).

The diagnosis of photosensitisation is usually based on the clinical signs. Differentiation from sheep scab is based on the involvement of unpigmented areas of the head and limbs and the distribution of lesions in the dorsal midline. Increased serum concentrations of liver-specific enzymes may aid in the detection of hepatocellular damage, but are not a consistent feature of the disease.

Affected animals should be immediately housed in darkness for up to 3 weeks to avoid further reaction with sunlight until the lesions heal.

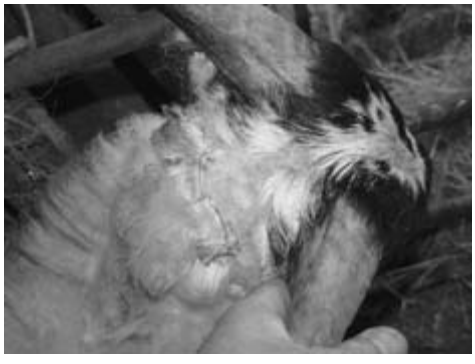


Fig. 7.117 Necrosis and sloughing of the skin behind the poll of a 4-month-old Scottish Blackface lamb with plochteach.

Fig. 7.118 An ill-thrifty, photosensitive Scottish Blackface lamb with swollen, drooping ears. The lamb is separated from the rest of the flock and hidden in a shaded spot.

Fig. 7.119 Necrosis of the skin of the unpigmented part of the pinna of a 9-month-old Scottish Blackface lamb, several months after acute photosensitisation. The affected part of the pinna feels stiff.

Fig. 7.120 Loss of the pinna of the ear of a Shetland ewe following severe photosensitive dermatitis.

Pyodermas

Pyodermas caused by toxin-producing strains of *S. aureus* are common on the head and face in housed sheep. The disease is characterised by deep necrotic ulceration and abscessation of skin. Ulcerative lesions up to 5 cm across are seen on the ears, at the angle between the ears and the face (Fig. 7.121), over the cheeks, over the nose (Fig. 7.122), around the external nares (Fig. 7.123) and around the eyes (Fig. 7.124). The submandibular or parotid lymph nodes are often enlarged (Fig. 7.125). Lesions involving the skin and subcutaneous tissues around the eyes are referred to as periorbital eczema. Lesions also occur on the inside of the hocks. Pyoderma lesions are sometimes seen at other sites such as the teats of lactating ewes (Fig. 7.126) and prepuce of rams (Fig. 7.127).

High morbidity rates of up to 60 percent and rapid spread of the disease are usually linked to winter housing and trough feeding of groups of rams. Pyodermas are occasionally seen at other times of the year, associated with trauma, bites or headfly. In

Fig. 7.121 Pyoderma lesions are commonly seen at the angle between the ears and cheeks, and in the skin overlying the vertical ramus of the mandible. The parotid lymph node is characteristically enlarged.

Fig. 7.122 Deep pyoderma lesion over the bridge of the nose of a Bluefaced Leicester shearling ram.

Fig. 7.123 Pyoderma lesion around and extending inside the external nares.

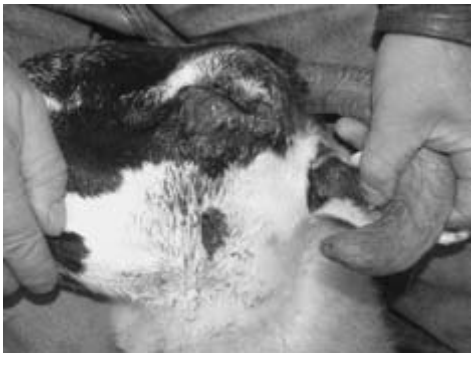


Fig. 7.124 Pyoderma lesions around the eyes are sometimes referred to as periorbital eczema.

Fig. 7.125 Enlargement of a parotid lymph node associated with a pyoderma lesion on the nasal frenulum.

Fig. 7.126 Staphylococcal lesions around the base of the teat, associated with inadequate milk supply and trauma caused by sucking lambs.

Fig. 7.127 Staphylococcal dermatitis involving the prepuce of a Suffolk ram.

Fig. 7.128 Deep necrotic lesions over the back of a Wensleydale ewe, discharging greenish coloured pus and staining the overlying wool.

stud ram flocks pyoderma lesions can prove sufficiently serious to reduce the sale value of the animals.

The diagnosis of staphylococcal pyoderma is based on the clinical signs, but may be supported by the identification of coagulase-positive haemolytic *S. aureus* on bacterial culture.

Staphylococcal pyoderma lesions generally heal completely within 2 to 4 weeks following a course of penicillin antibiotic injections.

Necrotic dermatitis

Outbreaks of severe necrotic dermatitis, associated with *Pseudomonas aeruginosa* infection, are sometimes seen in sheep of all ages associated with soaking of the skin.

The initial clinical signs are displaced tufts of wool along the back, over the flanks, or over the chest, and occasionally on the head or legs. These early signs can be easily overlooked. Closer examination reveals extensive ulcers (10 to 200 mm in diameter), covered by thick, moist, closely attached scabs (Fig. 7.128). Beneath the scabs, caseous, foul-smelling, greenish purulent material extends to the full depth of the dermis. Limb lesions can show extensive invasion and necrosis of deeper tissues including bone. Local lymph nodes are enlarged. Mild lesions may resolve over a period of 2 months, but severely affected animals waste and die. On postmortem examination, greenish abscesses up to 20 mm in diameter may be seen in several organs.

Most outbreaks occur within 6 weeks of shearing and are associated with persistent wet weather, or dipping 3 to 4 weeks beforehand. It has been speculated that shearing, physical trauma or other skin diseases disrupt the skin microflora, enabling colonisation by *P. aeruginosa*. Persistent wetting of the skin favours the multiplication of *P. aeruginosa*, and dipping may further disrupt the microflora and spread the infection. Necrotic *P. aeruginosa* infections of the nasal passages and middle ear have been reported, associated with shower dipping.

P. aeruginosa contamination of plunge-dip solutions can also lead to downgrading of wool due to staining of the fleece. *P. aeruginosa* produces proteolytic enzymes which cause superficial moist eczema with accumulation of sero-purulent exudate. *P. aeruginosa* produces a yellow/orange pigment, but secondary infection with yeasts,

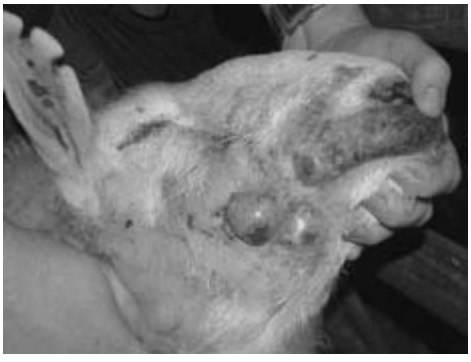


Fig. 7.129 Characteristic nodular lesions over the cheeks and lips of a Scottish Halfbred ewe.

Fig. 7.130 Discharge of thick yellow coloured pus from a small nodule under the lip.

Fig. 7.131 Actinobacillosis abscessation of the tongue of a Greyface ewe, similar to wooden tongue in cattle.

Fig. 7.132 Enlargement of the submandibular lymph node of a Greyface ewe with actinobacillosis of the tongue.

moulds and other bacteria may cause grey/blue or brown discolouration of the proximal 1 to 5 mm of the fleece, which are carried away from the skin surface as the wool grows.

The diagnosis of necrotic dermatitis is based on the clinical signs and confirmed by bacterial culture. Bacterial culture requires incubation at 37°C in 10 percent carbon dioxide for 30 minutes and inoculation onto 5 percent sheep blood agar with 10 000 units/ml polymyxin B.

Actinobacillosis

Actinobacillosis (also referred to as King's evil) is a common, sporadic cause of skin thickening, hair loss and multiple 2 to 5 mm, firm nodules in the subcutaneous tissues of the cheeks and muzzle (Fig. 7.129). The nodules discharge thick yellow pus when incised (Fig. 7.130). The disease is usually seen in young adult animals and is sometimes associated with tooth eruption or abrasions caused by grazing among thistles or whins. The disease is of little economic significance, but is an important differential diagnosis for orf and caseous lymphadenitis. Tongue lesions due to *Actinobacillus lignieresi*, which are common in cattle, are only seen occasionally in sheep (Figs 7.131 and 7.132), resulting in cud spilling and weight loss.

The diagnosis of actinobacillosis is confirmed by the identification of characteristic bacterial colonies on stained smears from discharging abscesses. Most cases respond well to parenteral antibiotic therapy.

Caseous lymphadenitis

Caseous lymphadenitis (CLA) is caused by chronic suppurative infection of lymphatic tissue by the bacterium, *Corynebacterium pseudotuberculosis*. In 1991 the first report of caseous lymphadenitis was confirmed in UK sheep. The disease was believed to have been introduced to the country 4 years earlier with imported goats. The incidence of the disease in sheep remained very low for the next 5 years. However, between 1998 and 2001, UK veterinary disease surveillance laboratories identified caseous lymphadenitis in about 150 new sheep flocks, mostly of terminal sire breeds, and in several goat herds. It was claimed that these figures probably underestimated the true prevalence of the disease, but suggested that caseous lymphadenitis was an emerging and a potentially important problem. A seroprevalence study in 2001 using a Dutch ELISA test suggested that as many as 18 percent of sheep in the terminal sire sector may have been affected and enabled mapping of where the problem flocks were based. Infection rates of more than 25 percent were seen within some flocks. The incidence of the disease has continued to increase, although not at the high rate predicted during 2001 (Fig. 7.133).

Clinical signs

To date caseous lymphadenitis in the UK has been confirmed mostly in pedigree flocks of terminal sire, longwool and hill breeds, and within these flocks the disease has been seen first in rams. Caseous lymphadenitis in UK rams has been characterised by thick-walled abscesses mostly within the lymph nodes of the head and neck. Various lesions have been described, including: inflammation of the skin over the poll; single or multiple discrete superficial 1 to 2 cm diameter firm subcutaneous abscesses on the side of the face, with loss of overlying hair; and firm palpable swellings up to 5 cm in diameter involving the submandibular or parotid lymph nodes, under or behind the angle of the jaw (Fig. 7.134). Abscesses have also been

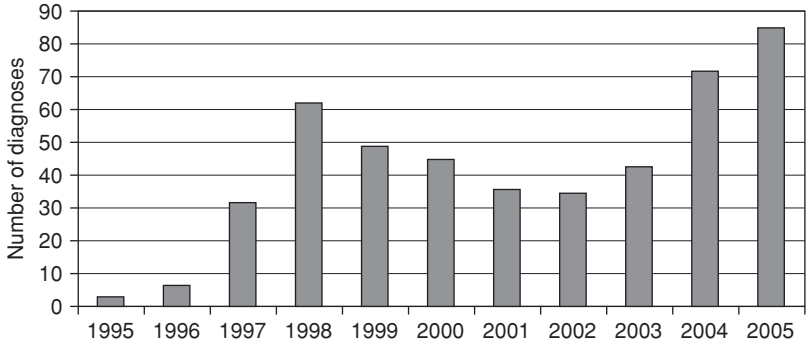


Fig. 7.133 Veterinary Investigation Diagnosis Analysis (VIDA) diagnoses of caseous lymphadenitis in UK sheep flocks.

Fig. 7.134 Abscessation over a parotid lymph node (not caused by caseous lymphadenitis).

Fig. 7.135 The majority of cases of parotid lymph node abscessation are associated with non-specific bacterial infections, rather than caseous lymphadenitis.

identified in front of the shoulders in the prescapular lymph nodes. Lesions may rupture if accidentally lanced, resulting in a discharging wound. No association has been conclusively demonstrated between the presence of lesions and ill thrift or poor reproductive performance.

Studies aimed at the development of an ELISA test have shown that about 25 percent of infected sheep have internal lesions, commonly in the lungs, without the presence of lesions in the skin or superficial lymph nodes. Studies involving artificial infections have shown that these lesions can be substantial, comprising numerous 2 to 5 cm diameter, caseated, thick-walled abscesses. Large, thick-walled abscesses, with an 'onion ring' appearance when sectioned, have been identified in the mediastinal lymph nodes. Abscessation of the liver, mammary glands and epididymes has also been recorded.

The initial lesion in lymphoid tissue is a diffuse inflammatory response to a bacterial exotoxin. Microscopic abscesses then form, which coalesce to form an area of caseation. The abscess then becomes encapsulated, but further necrosis and re-encapsulation continues. The 'onion ring' appearance of some internal abscesses is associated with cycles of pus production and granuloma formation.

A visceral form of the disease is recognised in Merino-type sheep in Australia and America associated with chronic wasting. The visceral disease is characterised by the presence of multiple abscesses in the lungs, bronchi and associated lymph nodes, which sometimes spread to the liver, kidneys and spleen. It is likely that the systemic disease is transmitted by the respiratory route in infected aerosols.

Diagnosis

The diagnosis of caseous lymphadenitis as a cause of superficial abscessation is based on the clinical signs, but similar lesions can result from a variety of causes (Figs 7.135 and 7.136), so confirmation relies on bacterial culture from infected lymph nodes. Bacterial culture takes 3 to 4 days, but is relatively straightforward. However, interference with the lymph nodes for sampling purposes should be avoided, because subsequent discharging fistulae may contribute to the environmental contamination

Fig. 7.136 Caseous lymphadenitis must be differentiated from subcutaneous abscessation of the neck, arising from cellulitis following a penetrating injury.

and spread of infection. An ELISA test is used overseas for the control of caseous lymphadenitis in milking goat flocks, and may prove useful for UK sheep flocks.

Postmortem examination of clinically affected animals and of clinically unaffected seropositive animals has also demonstrated the presence of firm palpable abscesses within the lung tissue and mediastinal lymph nodes.

Economic importance

It has proved difficult to predict the potential economic risks associated with caseous lymphadenitis in UK flocks, and the economic consequences of the disease in the national flock remain uncertain. Substantial research investment involving the development of diagnostic tests and control methods may reflect the importance of the disease with regards to the high-value pedigree sheep trade, rather than any potential economic consequences of infection in commercial UK flocks. Caseous lymphadenitis is widespread in many overseas countries, but the clinical manifestations of the disease differ, so direct comparison of potential economic losses is not possible.

In Australia and New Zealand caseous lymphadenitis causes considerable economic loss to the sheep meat industry, but not necessarily to individual producers, because of the slaughterhouse cost of trimming abscessed lymph nodes from carcases. Meat inspection losses associated with caseous lymphadenitis in Australia are estimated at about $A30 million per year. However, the economic cost to individual producers of carcase abscessation could be greater in the UK because of different meat inspection rules, which could result in total carcase condemnation when more than one abscess is identified.

Caseous lymphadenitis in Merino sheep in Australia has been linked to poor wool production. Any effect on wool production is unlikely to be significant in British sheep breeds.

Infection of supramammary lymph nodes in milk breeds of sheep and goats presents a potential public health concern and is therefore economically important. This has prompted costly eradication programmes in milking goat herds in the Netherlands.

A thin ewe syndrome is recognised in Merino-type sheep in the USA, associated with the presence of abscesses in the lungs and abdominal viscera, but in Australia enormous abscesses are sometimes found in the mediastinal lymph nodes of otherwise healthy sheep. The thin ewe syndrome that is seen in the USA may be confounded by the involvement of maedi-visna or other diseases resulting from the

common management practice of retaining elderly ewes. So far, no association has been made between the presence of pulmonary abscesses and ill health in UK sheep.

Caseous lymphadenitis is of greatest economic importance in UK pedigree flocks, because clinically affected animals may be unmarketable and the presence of the disease could result in loss of export trade, in particular with Ireland and Northern Ireland (despite the fact that the disease has been identified in Northern Ireland).

C. pseudotuberculosis occasionally causes potentially serious swelling of the axillary lymph nodes in humans, although the disease risk is low if sensible hygienic precautions are taken when handling infected sheep.

Disease spread

In experimental studies, the incubation period from infection with *C. pseudotuberculosis* to the appearance of abscesses is about 42 days. The severity of disease outbreaks is associated with the level of environmental contamination. Discharging abscesses are an important source of contamination and *C. pseudotuberculosis* can survive for up to 5 months in soil. The bacterium can also survive on faeces and survives for at least 24 hours in faeces-contaminated sheep dips. Dip antiseptics only kill *C. pseudotuberculosis* at very high concentrations.

The method of spread of caseous lymphadenitis appears to be dependent on the sheep husbandry system, so overseas experience may not be directly relevant to the UK disease. In New Zealand it is believed that the disease is transmitted by infection of superficial skin wounds and subsequent spread to regional lymph nodes. The prescapular and prefemoral nodes are most frequently affected, associated with the common sites of shearing wounds. In some animals, lymph node infection is followed by spread to internal organs. Abscessation of internal organs appears to be more severe in Merino than in British sheep breeds. Transmission occurs at shearing either following contamination of equipment by discharging and accidentally ruptured abscesses, or following infection of wounds from a contaminated environment. Close contact between animals after shearing also contributes to the rate of spread of the disease. *C. pseudotuberculosis* can also penetrate freshly shorn, wet, intact skin and shower dipping within 2 weeks after shearing has been shown to spread the disease.

In Australian Merino sheep, aerosol spread from abscesses in lung tissue onto shearing cuts of unaffected sheep is believed to be important. The disease is only spread by the small proportion of affected animals which have well-established and substantial lung infection.

The clinical signs of caseous lymphadenitis in UK differ from those seen overseas, because the primary site of infection appears to be the skin and lymph nodes of the head. The mode of transmission is still unclear, but the distribution of lesions indicates that infection enters through wounds on the head, or through abrasions in the mouth, possibly associated with tooth eruption. The dissemination of abscesses throughout the lung tissue, which has been seen on postmortem examination, is indicative of subsequent haematogenous spread. The involvement of lung and mediastinal lymph node abscesses as a latent source of infection is apparent, and *C. pseudotuberculosis* bacteria can readily be recovered from the airways of affected sheep. Housing, close confinement for prolonged periods in a contaminated environment, trough feeding and fighting among ram lambs are likely to be important risk factors. Shearing has been implicated in some UK flocks. Practices associated with the

preparation of rams for sale and their handling at markets may also contribute to the spread of caseous lymphadenitis in pedigree sheep in the UK.

Disease management

C. pseudotuberculosis is sensitive to a wide range of antibiotics. However, once thick-walled abscesses are formed, treatment with antibiotics is ineffective. Surgical drainage and flushing may initially reduce the size of the lesions, but will not eliminate the infection. This practice only results in further environmental contamination and should be avoided.

The options for the management of caseous lymphadenitis are:

- radical changes to those farm management practices involved with pedigree sheep production, which probably predispose to spread of the disease
- vaccination
- blood test and cull seropositive animals.

Sheep should be inspected regularly and animals with discharging lesions should be isolated or culled. Attention should be paid to environmental hygiene at shearing and dipping and any clinically affected sheep should be shorn or dipped last. Improvements in general farm hygiene, prevention of unnecessary wounds, reduction in stocking rates at housing and provision of adequate trough space are likely to reduce the spread of infection, but changes may not fit well with traditional pedigree sheep management. Ram lambs should be kept apart from older animals. Sheep should not be handled in heavily faeces-contaminated, dusty yards and should be released from the pens as quickly as possible.

Overseas, caseous lymphadenitis can be controlled effectively using formalin-inactivated toxoid vaccines. Unfortunately, no licensed caseous lymphadenitis vaccines are available in the UK. Imported vaccines are unlicensed and their use in the UK is illegal, unless they are imported under a specific import scheme authorisation from the Veterinary Medicines Directorate. There is no evidence to support the efficacy of overseas vaccines with regard to UK isolates of *C. pseudotuberculosis*, and no commercial justification to make them marketable in the UK.

Crude autogenous formalin-killed vaccines based on whole bacteria can be produced for individual farms under an emergency licence, but are expensive, costing £1200 to £1500 per litre of vaccine, which is only useful for a single holding. Different pulsotypes (strains) of *C. pseudotuberculosis* may be present within a flock and there is no experimental evidence to support the efficacy of these autogenous vaccines.

Studies on the development of a UK vaccine for caseous lymphadenitis have been encouraging, with the best response in terms of prevention of clinical disease and spread of infection achieved by a combined recombinant toxoid (phospholipase D) and killed whole-cell product. Unfortunately, the commercial development of this experimental vaccine is unlikely, associated with prohibitive registration and licensing costs, relative to the cost of the disease.

While culling clinically affected animals can reduce the prevalence of disease within a flock, eradication of caseous lymphadenitis from endemically infected flocks using this method is not possible, because affected sheep do not always show overt clinical signs. This problem could be overcome by the development of a serological test for caseous lymphadenitis.

Development of an enzyme-linked immunosorbent assay (ELISA) for caseous lymphadenitis

ELISA tests detect antibodies specific to the infecting organism. *C. pseudotuberculosis* has numerous different potential surface antigens, but they are not all present in all isolates. The toxin, phospholipase D (PLD), is conserved between isolates and can be produced using molecular methods in an extremely pure form and in adequate quantities to enable it to be useful.

PLD antigen is added to wells of a 96-well plate, in a special solution which enables it to stick to the plate. Excess is then washed off, leaving PLD stuck to the walls of the wells. The sheep blood sample is then added to the wells, left for a while and then washed out, leaving any specific antibody for PLD bound to the PLD on the wall of the wells. A secondary antibody to the sheep antibodies is then added, left and washed out. Finally an enzyme and substrate is added to the wells, which reacts with the conjugate to catalyse a colour change reaction if specific antibodies for PLD were present in the blood sample. The optical density of the solution can be measured to determine the presence or not of anti PLD antibody in the sheep blood sample.

Based on samples from 500 sheep, using positive culture of *C. pseudotuberculosis* as the 'gold standard', the sensitivity of the UK ELISA test is claimed to be 87 percent, while its specificity is 99 percent.

Failure of the test to detect some diseased sheep may be related to the nature of the disease itself, whereby immunity may wane and then recover during the cyclic process of pus production and granuloma formation. The accuracy of the test to detect disease on a flock basis can be improved by increasing the number of sheep sampled, testing the same animals on two separate occasions, and ensuring that any animals showing clinical signs are tested.

Potential problems associated with false positive results can be addressed by using a separate serological test on the samples that test positive. Unfortunately, the only other available serological test for caseous lymphadenitis is the Western blot, which uses the same PLD antigen and secondary sheep antibodies, so is not sufficiently independent from the ELISA to identify primary false positives.

The ELISA test cannot distinguish between titres associated with vaccination and natural infection, so cannot be used to support freedom from disease in previously vaccinated sheep.

All purchased sheep should be carefully examined for the presence of head abscesses and suspect animals should be rejected, along with all the others in the same group. The Scottish Agricultural Colleges have launched a caseous lymphadenitis monitoring scheme, based on the use of the ELISA to screen sheep before sale. The scheme does not guarantee flock freedom from the disease, but provides purchasers with some assurance that the risk of introduction of caseous lymphadenitis to their flocks with monitored sheep is low. This assurance is based on:

- investigation of any suspicious cases with negative results
- isolation of tested animals from other groups of sheep for at least 12 weeks
- avoidance of nose-to-nose contact with other sheep at shows or sales
- two blood samples 6 weeks apart and negative ELISA results
- a signed declaration of adherence to the rules.

Ringworm

Ringworm was traditionally considered to be uncommon in UK flocks. Cases were generally thought to result from contact with fomites contaminated with fungal spores from other species. *Trichophyton verrucosum* was associated with housing in buildings previously occupied by cattle and *Microsporum canis* with contact with dogs. The morbidity rate was generally low and the disease self-resolving within 4 to 6 weeks, associated with the fact that sheep were not the natural hosts for the fungi.

Ringworm due to a putative sheep-adapted strain of T. verrucosum

In recent years there have been several reports of ringworm due to skin infection by a putative sheep-adapted strain of *T. verrucosum*. Disease outbreaks have occurred in flocks with no history of contact with cattle or buildings formerly occupied by cattle. Most reports have been from pedigree flocks of ram lambs, although the disease has also been seen in commercial flocks. In severe outbreaks up to 70 percent of the flock have been affected and lesions have taken many months to heal.

Clinical signs

Ringworm in sheep is characterised by clearly demarcated, 1 to 10 cm^2 areas of hair loss, covered by dry wart-like crusts on the ears (Fig. 7.137), face (Fig. 7.138) and wool-free parts of the neck (Fig. 7.139). Affected animals do not excessively rub or scrape at the lesions. Where the disease has been seen in shearling animals, it has been characterised by raised 4 to 6 cm diameter scabs over the wool-covered parts of the body (Fig. 7.140). When the matted wool covering the lesions is removed, the underlying skin is inflamed, foul smelling and bleeds readily (Fig. 7.141). Lesions in shearling rams have appeared about 6 weeks after shearing and in some cases the disease has persisted for 6 months (Fig. 7.142). The presence of ringworm lesions can prove to be economically important, because it precludes sale of breeding stock. Generalised infection involving the skin of most of the body and fungal infection of internal organs have occasionally been reported in individual animals, presumably associated with concurrent immunosuppressive disease (Figs 7.143 and 7.144).

Fig. 7.137 Wart-like ringworm lesions on the ear of a 5-month-old Suffolk lamb.

Fig. 7.138 Ringworm lesions around the eye of a 6-month-old Suffolk lamb.

Fig. 7.139 Healed ringworm lesions on the neck of a shearling Bluefaced Leicester ram. The healed skin is characteristically dark-pigmented.

Fig. 7.140 Ringworm lesions along the ventral neck of a Bluefaced Leicester shearling ram.

Fig. 7.141 Matted wool and exudation and bleeding of the skin associated with a plaque-like ringworm lesion over the tail head of a Cheviot shearling ram. (The handler ought to have been wearing protective gloves.)

Fig. 7.142 Plaque-like ringworm lesions covering the body of a shearling Suffolk ram, potentially reducing its sale value.

Fig. 7.143 Extensive ringworm lesions, covering the head of a shearling Bluefaced Leicester ram.

Fig. 7.144 The same animal as in Fig. 7.153, 4 months later in a cachectic state due to generalised and systemic ringworm infection.

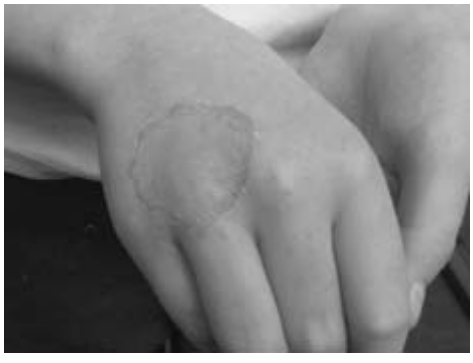


Fig. 7.145 A sheep-associated ringworm lesion on the author's daughter's hand.

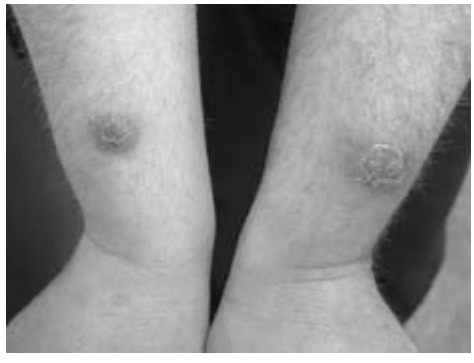


Fig. 7.146 Sheep-associated ringworm lesions on the author's colleague's arms. These lesions took 4 months to heal.

T. verrucosum infection is an important, potentially serious zoonotic disease, human skin lesions associated with contact with infected sheep persisting for several months (Figs 7.145 and 7.146).

Diagnosis

Scab material does not usually fluoresce under ultraviolet light. Ectothrix spores can be seen on microscopic examination of fleece samples and scab material after softening and clearing the samples in 10 percent aqueous potassium hydroxide. Fungal culture on standard selective Sabourad's agar fortified with additional antibiotics can be used to identify the specific dermatophyte.

Management

Topical natamycin, which is used in other species for treatment of ringworm, appears to be ineffective for the treatment of lesions over the body, possibly because the exudate-matted fleece overlying the lesions prevents contact between drug and dermatophyte. Daily inclusion of 7.5 mg/kg griseofulvin in feed for 7 days has proved useful for the treatment of ringworm in other species. Unfortunately griseofulvin does not have a minimum residue level under European Community legislation, so can no longer be used in food-producing animals in the UK. The management of ringworm in sheep, therefore, depends on prompt recognition of the clinical signs, isolation of affected animals and disinfection of troughs and barriers. Precautions are necessary at shearing to prevent the spread of infection from the face to the body of sheep.

Wool break and wool slip

Arrest in wool growth and consequent fibre weakness and wool loss may follow prolonged pyrexia, metabolic disease or physiological stress. Large, often bilaterally symmetrical areas of wool loss may occur anywhere on the body, sometimes eventually affecting the entire fleece (Fig. 7.147).

Fig. 7.147 Wool loss due to a break in the fibres caused by systemic disease in a Scottish Blackface ewe.

Fig. 7.148 Wool loss from its follicles, putatively associated with excessive production of endogenous corticosteroids in a 9-month-old Wensleydale lamb.

A similar condition is seen in winter-shorn housed sheep, with wool falling out from its follicles (Fig. 7.148), possibly associated with cold stress and excessive production of endogenous corticosteroids.

These conditions are easily differentiated from infectious and parasitic skin diseases, since there is no pruritus or involvement of the underlying skin.

Other skin diseases

Burns

The diagnosis of thermal burns is usually obvious from the history and the presence of charred wool. Chemical burns may follow incorrect plunge dipping in strong phenolic solutions and cause superficial necrotising dermatitis, sometimes with necrosis of underlying adipose tissue and severe secondary bacterial and fungal infection.

The skin of sheep infested with *P. ovis* is more vulnerable to the effects of noxious agents than that of healthy sheep due to removal of the skin's protective lipid barrier by the feeding activities of the mite. The wool overlying recovered sheep scab lesions is often pigmented (Fig. 7.149).

Fig. 7.149 Pigmented skin and wool overlying previous sheep scab lesions.

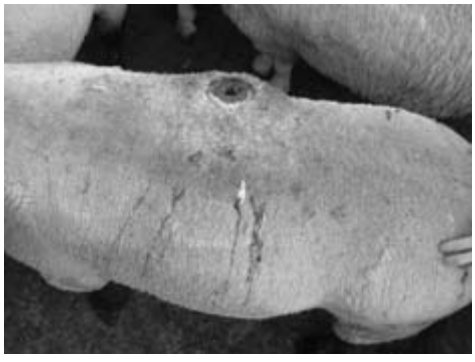


Fig. 7.150 A deep, infected wound over the lumbar spine of a Suffolk ram. Magpies were seen targeting this individual animal, and evidence of their prolonged presence can be seen on the wool adjacent to the wound.

Fig. 7.151 A piece of sackcloth has been glued to the wool adjacent to a site of magpie attack to prevent further injury.

Fig. 7.152 Folliculitis under the tail and around the vulva of a 6-week-old Texel cross lamb.

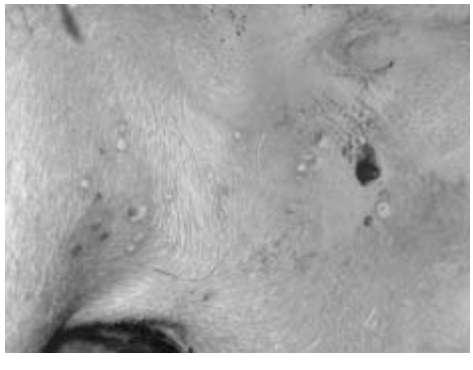


Fig. 7.153 Folliculitis lesions in the groin of a shearling Suffolk ram, seen as an incidental finding while investigating reproductive performance.

Magpie attack

Magpies sometimes inflict deep wounds over the lumbar spine of rams (Fig. 7.150). Individual animals appear to tolerate such attack. These wounds only heal when the affected rams are housed, or the lesions covered (Fig. 7.151), before administration of a course of systemic antibiotic treatment. However, the same individuals appear to be re-affected during subsequent years.

Staphylococcal folliculitis

Staphylococcal folliculitis (plooks) is a common, economically unimportant skin disease of young lambs (Fig. 7.152). Folliculitis lesions are sometimes seen in the groin and axilla of adult sheep (Fig. 7.153).

Fig. 7.154 Abscessation of the skin over the brisket of a 3-week-old Merino cross Scottish Blackface lamb, associated with *F. necrophorum* infection.

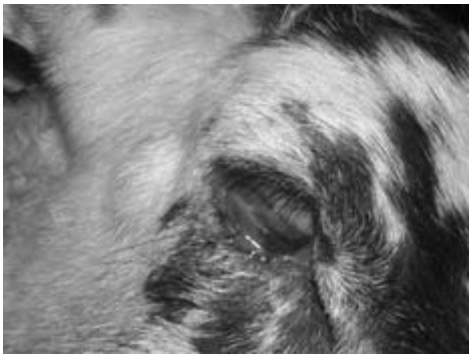


Fig. 7.155 Traumatic injury to the eye of a Mule ewe, associated with a piece of chaff trapped within the conjunctival sac.

Non-specific abscessation

Abscesses involving non-specific bacteria such as *Arcanobacterium pyogenes*, *Streptococcus* spp. and *F. necrophorum* occur in sheep of all ages at various sites, usually associated with trauma, dog bites, or penetrating wounds (Fig. 7.154). Infection often tracks along fascial planes and frequently requires drainage, flushing, debridement and prolonged antibiotic therapy.

Eye diseases

Eye disease due to ovine infectious keratoconjunctivitis (OIKC) is a common problem in UK sheep flocks, while other diseases such as *Listeria* iritis, bracken poisoning (bright blindness), glucosinolate poisoning (rape blindness), vitamin A deficiency and the inadvertent overdose of closantel for the control of fascioliasis periodically cause blindness in UK sheep flocks. Individual sheep can suffer from a range of eye conditions such as traumatic injury (Fig. 7.155), neoplasia (Figs 7.156 and 7.157) and congenital abnormalities (Plate 7.1).

Infectious keratoconjunctivitis

Ovine infectious keratoconjunctivitis (OIKC) is seen in sheep of all ages, but tends to be more severe in ewes than in lambs. Most cases occur during the winter months, often following high winds and driving snow (Fig. 7.158), when up to 70 percent of the sheep in some flocks may be affected. Outbreaks affecting up to 25 percent of the flock are commonplace. OIKC can become particularly important when pregnant ewes are involved.

The first sign of OIKC is conjunctivitis, with closing of the eyelids and tear staining extending from the corner of one or both eyes (Fig. 7.159). In some cases the clinical signs regress at this stage, while in others the cornea becomes cloudy due to inflammation (keratitis), with prominent blood vessels around the periphery (Plates 7.2 and 7.3). This is accompanied by tear production (lacrimation) and blinking

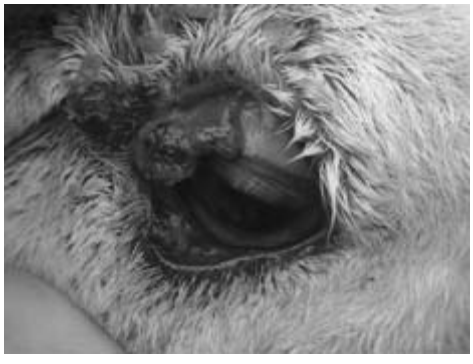


Fig. 7.156 A tumour of the third eyelid of a Scottish Halfbred ewe.

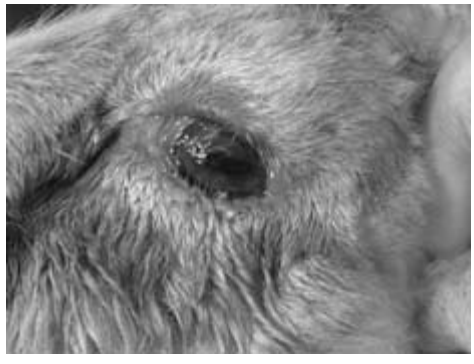


Fig. 7.157 Protrusion of the eye leading to keratitis in a crossbred ewe, associated with the presence of a retrobulbar tumour.

Fig. 7.158 Driving snow probably increases the risk of outbreaks of ovine infectious keratoconjunctivitis.

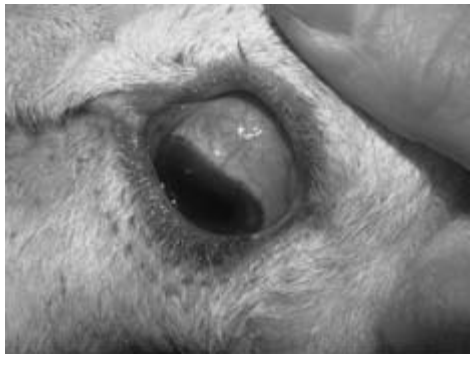


Fig. 7.159 Lacrimation, conjunctivitis and scleral inflammation during the early stages of OIKC.

(blepharospasm), the ocular discharge becoming thicker and purulent (Fig. 7.160). The bulbar conjunctiva and third eyelid develop a thickened, granular appearance due to lymphoid hyperplasia. When both eyes are severely affected sheep become temporarily blind, resulting in handling difficulties and losses due to misadventure. In most cases, lesions start to heal after about 5 days, with recovery over a period of 2 to 3 weeks. However in severe cases, the anterior chamber of the eye may ulcerate. Once the ulcerated area has become vascularised, healing by granulation occurs over a period of several weeks (Fig. 7.161), eventually leaving only a faint corneal scar. A few sheep may be left permanently blind following rupture of corneal ulcers and secondary infection of the anterior chamber of the eye (Fig. 7.162). Acquired immunity following infection is poor and many cases relapse.

The primary causative organisms of OIKC are believed to be *Chlamydia psittaci* and *Mycoplasma conjunctivae*, either alone or in combination. *M. conjunctivae* is the more frequently isolated organism, but several other bacteria may also be involved in a secondary role. Close contact of sheep when trough feeding enables rapid spread of infection and partly accounts for the high incidence of the disease during winter

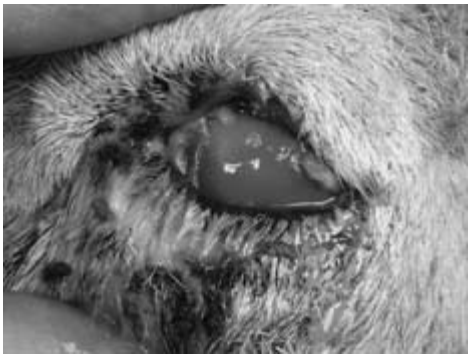


Fig. 7.160 Conjunctivitis, corneal opacity and a purulent ocular discharge in a severe case of OIKC.

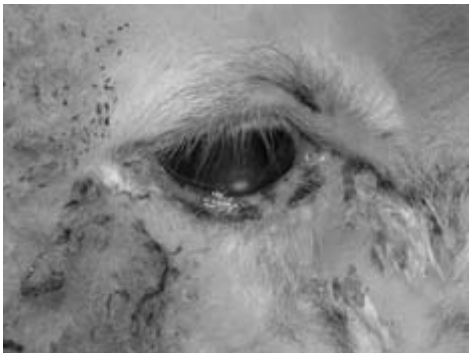


Fig. 7.161 A healing ulcer in the centre of the cornea.

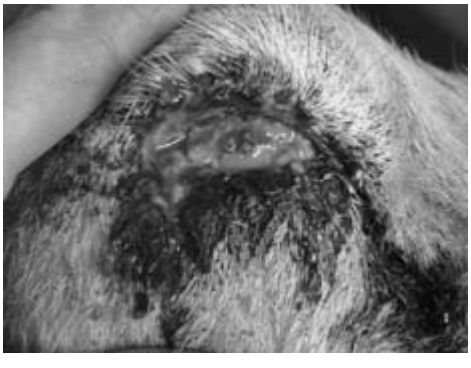


Fig. 7.162 Rupture and secondary infection of the eye, leading to permanent blindness.

Fig. 7.163 Treatment using an aureomycin ophthalmic puffer. The powder tends to adhere to the moist conjunctivae, where ophthalmic ointments tend to slip off.

months. Clinical cases are often first seen following snowfall, perhaps because bright light causes conjunctival irritation which predisposes to the disease. Outbreaks in recently weaned lambs may be associated with high stocking rates, dust, long grass and flies which have been contaminated by tear secretions of infected animals. Handling of the face and head of lambs when drenching may also provide a means of spread. Clinically recovered animals may harbour the causative organisms for several months and may be a source of re-infection of other animals in subsequent years when their immunity has waned.

The disease is painful and prompt treatment of affected ewes is recommended to mitigate against the risk of permanent blindness. Treatment is tedious and usually involves the topical application of aureomycin using a puffer (Fig. 7.163) or ointment. There is some evidence to support the use of intramuscular injections of long-acting oxytetracycline or subcutaneous injection of tilmicosin, which may be a practical method for the treatment of OIKC in some flocks.

Separation of clinically affected animals is sometimes recommended to limit spread of infection, but in most cases the disease is already widespread before the first clinical signs are seen.

Listeria iritis

Inflammation of the anterior chamber of the eye (anterior uveitis, or silage eye) periodically occurs in sheep (and cattle) of all ages associated with the feeding of big bale silage. The disease is associated with silage feeding and is thought to be caused by *Listeria monocytogenes* infection. Up to 25 percent of the flock can be affected in severe outbreaks.

The initial presenting signs are epiphora (excessive lacrimation), blepharospasm, avoidance of bright light (photophobia), pupillary constriction (miosis) and swelling of the iris (iridocyclitis), either unilaterally or bilaterally. The iris may be thrown into a series of radial folds extending from the ciliary border to the pupillary edge. Within 2 to 3 days, more severe inflammatory changes develop with blueish white corneal opacity starting at the limbic border and spreading centripetally. Focal aggregations of fibrin accumulate in the anterior chamber, attached to the inner surface of the cornea, and are seen as accumulations of white material beneath the cornea. Pus in the anterior chamber of the eye (hypopyon) and mild conjunctivitis may also develop at this stage. In more advanced cases, widespread corneal opacity and vascularisation may be observed. If untreated, complete healing of this painful condition may take 1 to 3 weeks.

Anterior uveitis appears to be a primary condition and there are no reports of animals developing symptoms of meningoencephalitis following primary anterior uveal listerial infection.

Subconjunctival injection of a combination of oxytetracycline and dexamethasone usually halts the progression of clinical signs when administered in the acute stages of the disease and the eyes usually return to normal within 2 to 3 days.

Vitamin A deficiency

Vitamin A is required for the regeneration of the retinal pigment, rhodopsin. Deficiency results in night blindness. Vitamin A is also required for maintenance of the integrity of the dura surrounding the brain and spinal cord and bone, deficiency resulting in an increase in intracranial pressure and constriction of the optic canal with impingement of bone on the optic nerve. These changes cause blindness, with a deficient menace response and absent pupillary light reflexes, weight loss, depression, ataxia, hindlimb weakness and convulsions.

Changes in the retina and optic disc are sometimes seen on ophthalmoscopic examination. The upper border of the optic disc is indistinct and casts a shadow on the adjacent retina. Blood vessels are tortuous as they course across the swollen retina. Thickening and whitening of the cornea and serous ocular discharge are seen in advanced cases.

Vitamin A deficiency is rare in UK sheep. Green feed usually provides sufficient beta-carotene vitamin A precursors, so most problems are seen in housed animals, fed diets such as straw, cereals or beet pulp. Vitamin A is not stable, and is sometimes destroyed when feed becomes rancid. Concurrent disease or dietary energy deficiency may inhibit the metabolism or absorption of vitamin A and the requirements of rapidly growing weaned lambs are greater than those of adult sheep.

The diagnosis of vitamin A deficiency is based on the clinical signs and determination of plasma vitamin A levels. The early signs of night blindness are reversible following vitamin A supplementation, but those of blindness due to bony changes and increased cerebrospinal fluid pressure are not. Prevention depends on the provision of adequate vitamin A in the diet.

Appendix A: Exotic and notifiable diseases

During the past three decades, caseous lymphadenitis, maedi-visna, chorioptic mange and foot-and-mouth disease have all been introduced to or reappeared in UK sheep. Experiences with these diseases highlight the need for prompt recognition of exotic diseases and for an understanding of the risks that they pose. This section is focused on the recognition, diagnosis and potential consequences of notifiable diseases, rather than on their management, which is generally statutory, based both on science and political expediency. Specific information about the disease status of UK sheep will inevitably be out of date by the time of publication, or reading, of this appendix. Excellent, detailed and up-to-date descriptions of national and international responses to exotic diseases are provided on the websites of the Department for Environment, Food and Rural Affairs (DEFRA) (www.defra.gov.uk) and Office International des Epizooties (OIE) (www.oie.int). Images of the different diseases are also available on these websites.

The responsibility for biosecurity to minimise the risk of introduction of exotic diseases rests with many different individuals and organisations. For example:

- on individual farms – farmers and veterinary practitioners
- within cooperative groups – breed societies
- within defined regions – advisors and local government
- within devolved administrations – e.g. Scottish Executive Environment and Rural Affairs Department (SEERAD)
- nationally – DEFRA (national government)
- internationally – OIE.

Not all serious exotic diseases are notifiable, for example the tick-transmitted disease, heartwater, is important in parts of Africa but is not notifiable in the UK, and not all notifiable diseases are exotic, but the list of notifiable diseases (Table A.1) highlights those of serious concern.

Any person having in their possession or under their charge an animal affected or suspected of having one of these notifiable diseases must, with all practicable speed, notify that fact to a police constable (or local Animal Health Office).

Foot-and-mouth disease

Foot-and-mouth disease is endemic in parts of Asia, Africa and South America, but periodically occurs elsewhere, as in the case of the 2001 outbreak in the UK. Foot-and-mouth is a highly contagious, rapidly spreading disease of cloven-hoofed animals, causing substantial economic loss not only as a direct consequence of clinical disease, but also due to the cost of control measures and restrictions on international

Sheep Flock Health: A Planned Approach. Neil Sargison
 ISBN: 978-1-4051-6044-5

Table A.1 Notifiable diseases under section 88 of the Animal Health Act 1981, or an Order made under that Act. Source: www.defra.gov.uk/, January 2008.

Disease	Last reported in the UK	Other species affected
Anthrax	Present	Cattle and other mammals
Aujeszky's disease	1989	Pigs and other mammals
Bluetongue	2007	Goats
Tuberculosis	Present	Cattle
Brucella melitensis	1956	Goats
Contagious agalactia	Never	Goats
Brucella ovis	Never	Goats
Foot-and-mouth	2007	Cloven-hoofed mammals
Peste des petits ruminants	Never	Goats
Rabies	2006	Dogs and other mammals
Rift Valley fever	Never	Cattle and goats
Scrapie	Present	Goats
Sheep pox	1866	Goats

trade of livestock and animal products. The 2001 outbreak of foot-and-mouth disease in the UK clearly exemplifies the importance of awareness, biosecurity, notification, rational control measures and disease surveillance.

Cause

Foot-and-mouth disease is caused by any one of seven different serotypes of a picornavirus (O, A, C, Asia 1, SAT 1, SAT 2 and SAT 3), with more than 60 different subtypes. There is little cross-protection between different serotypes; different serotypes may show different host preferences, resulting in different patterns of disease; and determination of the serotype and subtype involved can enable the source of any disease outbreak to be identified.

The foot-and-mouth virus is killed by the low pH which occurs in meat, but not in offal or bone marrow, leading to its persistence in these sources. The 2001 UK outbreak was caused by the serotype O pan Asian strain, with the first case occurring on a swill-feeding pig farm. This index case was only discovered after pigs from the farm had been moved several hundred miles to slaughter. In the meantime, sheep and cattle on nearby farms had become infected by airborne virus arising from infection in pigs. Infected sheep were then moved through markets and cattle became infected as a result of contact with sheep. Consequently, about 4 million UK farm animals had to be culled. It took nearly one year to eradicate the disease, which cost the country in excess of £2.7 billion. Furthermore, some infected sheep were exported for slaughter, leading to cases in Germany, the Netherlands, Belgium, France and Spain, necessitating the culling of a further 60 000 animals. The severity of the disease outbreak was influenced by the delayed reporting of the index case, and the extensive movement of sheep through markets before the disease was diagnosed.

Clinical signs

The clinical signs associated with foot-and-mouth disease in sheep are transient and, if present, they may be easily misdiagnosed. Conversely, other common sheep diseases may present with similar clinical signs. The clinical manifestation of

foot-and-mouth disease in sheep ranges from mild disease involving only a few animals to severe disease involving a high proportion of the flock. This variation is associated with: the serotype of virus involved; the age, breed, nutrition, health and physiological state of the sheep; and the effects of climate, stocking density, housing, stressful management, or co-grazing with more susceptible species such as cattle.

The principal clinical sign that is seen in sheep is blister formation around the coronary band, on the bulb of the heels and between the digits, causing sudden-onset lameness involving one or more limbs. The blisters may not be immediately apparent and it is necessary to examine lame animals carefully by turning them up and parting the hair over the coronary band. If the blisters have ruptured then hair may be damp and bacterial infection may be apparent. Affected feet may feel hot and are painful to touch. Sometimes, when blisters involve the whole of the coronary band, the hoof wall is shed. Affected sheep spend prolonged periods in recumbency and may be reluctant to rise. When all four feet are involved, they are reluctant to move and tend to stand with their hind limbs drawn under the abdomen.

Affected sheep are transiently pyrexic, anorexic and listless, although these signs ore often unnoticed in grazing animals. Lesions in the mouth, which characterise foot-and-mouth disease in cattle, are often absent. When present, the usual lesion that is observed is a small healing erosion or ulcer, with surrounding remnants of epithelium on the dental pad or caudal tongue. Examination of the mouth of sheep is not easy and is best performed using some sort of gag such as a piece of wood jammed between the molar teeth.

When pregnant ewes are involved, a high incidence of metabolic disease may be seen, while in some outbreaks, the only clinical manifestation may be a very high abortion rate. A high incidence of sudden death due to myocarditis may occur in neonatal lambs.

Diagnosis

Successful control of any foot-and-mouth outbreak depends on the prompt identification of infected animals. The clinical signs may provide an indication of foot-and-mouth disease in sheep, especially when seen during a national disease outbreak, when obvious signs are present in cattle on the same farm, or when they are associated with a history of contact with imported animals. However, the clinical signs of foot-and-mouth disease in sheep are variable and there are numerous common differential diagnoses. Other causes of lameness, such as scald, ovine digital dermatitis, post-dipping lameness and laminitis, can present with similar clinical signs, emphasising the need to investigate any unusual lameness outbreaks. It must be borne in mind that foot-and-mouth may be seen in sheep which are also lame for other reasons. The presence of mouth lesions may be indicated by excessive salivation, although this is also seen in cases of listeriosis, orf, dental disease and choke. Similar oral lesions are seen in cases of orf, epidermolysis bullosa and idiopathic oral ulceration (Fig. A.1), which is possibly caused by advanced dental disease or by chewing hard feed blocks or mineral licks. There are several causes of high neonatal lamb mortality and of high abortion rates, highlighting the need to fully investigate these problems.

Confirmation of the diagnosis of foot-and-mouth depends on serology and virus isolation. Serum antibodies can be detected using an ELISA from about 5 days after

Fig. A.1 Superficial erosions of unknown origin involving the gums of sheep are commonplace, and may easily be mistaken for foot-and-mouth lesions.

infection, providing evidence of exposure. Samples of epithelium from fresh lesions in the mouth or feet can be tested using an ELISA for the presence of viral antigen. Blood samples, saliva and samples from older lesions can also be tested but contain smaller amounts of virus. In these cases, the virus can be amplified in cell culture to improve the diagnostic sensitivity of the ELISA, although this process may take 4 to 5 days.

Confirmation of the diagnosis using these current methods inevitably incurs a delay between suspicion of disease and implementation of control measures in response to a positive result. Massive amounts of virus may be shed during this period, leading to substantial further spread of infection. There is therefore a need for the development of a validated, rapid, sensitive and specific and 'pen-side' test.

Disease control

The foot-and-mouth virus is killed by heat, sunlight and disinfectants, but survives for up to 6 months in cool and moist environments. The virus is spread: by direct contact with infected animals; through infected animal carcases or products, such as milk, skins, embryos and semen; indirectly by movement of fomites on the wheels of vehicles, manure or clothing; with carrier sheep, cattle or pigs; airborne for several miles; or rarely with wildlife.

Very small amounts of virus are required to establish infection. Sheep are usually infected by the respiratory route or through breaks in the skin, following which they become viraemic. The virus is then excreted in saliva, milk and faeces, before the onset of clinical signs. Large quantities of virus are present in blisters. Antibodies are then produced, leading to a rapid decline in viral excretion.

Species differences in susceptibility are important in the spread of foot-and-mouth virus. The disease in sheep is generally, but not always, mild, leading to the silent dissemination and maintenance of infection in carrier animals. Cattle are highly susceptible, acting as sentinel species, while respiratory infection of pigs results in the production of vast amounts of virus, leading to airborne spread of plumes of virus over large distances.

Prevention of introduction of foot-and-mouth to the UK involves stopping entry of live animals or animal products from countries and/or areas where foot-and-mouth is present. Importation of some animal products such as de-boned meat or heat-treated products may be permitted. The risk of inadvertent spread of

unrecognised foot-and-mouth disease is reduced by preventing the feeding of waste food to animals, and biosecurity.

Contingency plans for eradicating foot-and-mouth from the UK in the case of any future outbreak are periodically reviewed and can be found on the DEFRA website. These are likely to include:

- Early detection and diagnosis of disease.
- Culling of sheep, cattle, pigs and goats on infected premises and of known dangerous contacts within 24 hours of identification of disease.
- Pre-emptive slaughter to prevent the spread of disease, with or without emergency vaccination.
- Strict controls of animal, people and equipment movements.
- Disposal of infected carcases by burial, burning or rendering.
- Prompt tracing and slaughter of all livestock exposed to disease.
- Cleansing and disinfection of infected premises.
- Restocking with sentinel animals to ensure absence of infection.

Vaccination

Inactivated vaccines can be developed against foot-and-mouth disease, but these are both serotype and subtype specific, so must be made in response to the virus strain involved with or predicted for a specific outbreak. The UK has access to several different strains of foot-and-mouth antigen, which could be used to produce vaccines in the face of any future disease outbreak.

Vaccination does not prevent disease and may promote carrier status when used in animals that are already infected. Whenever foot-and-mouth outbreaks are controlled by culling, the OIE will designate a country free from the disease 3 months after the last reported case, enabling re-establishment of international trade of livestock and animal products. However, if vaccination is practised, this period is currently 6 months. Historically, a major limitation of vaccination has been the inability of serological tests to differentiate between antibodies due to natural infection and antibodies due to vaccination. The development of marker vaccines based on purified antigen may potentially overcome this problem.

Prophylactic vaccination is practised in some countries where foot-and-mouth disease is endemic. Two doses of vaccine are given 2 to 4 weeks apart. Cows and pigs are vaccinated to prevent clinical disease and virus production respectively. In these situations, vaccination of sheep is generally unnecessary due to the milder nature of the disease and lower risk of transmission. Routine prophylactic vaccination is prohibited in the EU.

Strategic vaccination in the face of a disease epidemic involves the use of high doses of potent, multivalent vaccines, which can provide immunity within 4 to 5 days. The objectives of strategic vaccination are either to ensure freedom from clinical disease, for example in the face of challenge from airborne virus, or to reduce the spread of disease by suppressing the production of virus from infected animals. The latter strategy is useful when there is an inevitable delay between detection of disease and slaughter, and involves vaccination of all susceptible animals within a ring around infected premises, or as a buffer zone to stop transmission.

Bluetongue

In many sheep-producing regions, including South Africa and North America, bluetongue is an economically important, endemic disease of sheep production. When the disease occurs outwith endemically infected areas, then its effect on animal production is potentially even more serious, reinforcing the need for its statutory control.

Cause

Bluetongue is caused by an orbivirus. There are at least 24 different serotypes of the virus (BTV), not all of which are necessarily pathogenic in sheep. The sole means of transmission is via biting insects. BTV multiplies in a number of arthropods and vertebrates, but *Culicoides* midges are the most important vectors. Following multiplication in *Culicoides* midges, one bite can be sufficient to cause clinical disease in a sheep.

In most endemically affected areas, the midges preferentially feed on cattle. Cattle infection with most strains of the BTV does not result in clinical signs of severe disease. However, cattle become a temporary reservoir of infectivity for up to 100 days following infection, linked to the lifespan of their cells to which the virus adheres. Severe clinical disease is generally only seen in sheep, north European breeds being most susceptible. Unimproved sheep are usually resistant to the clinical effects of infection. Thus, the typical transmission cycle is between *Culicoides* midges and cattle, with sheep being infected only when few cattle are present or the midge population is high. Sheep are not the preferred host for most serotypes of BTV and the virus seldom persists beyond 30 days in their blood, hence they are not required to maintain infectivity.

Endemic bluetongue is typically restricted to the warm regions due to the environmental conditions required for *Culicoides* survival. A large landmass also appears to be important for retreat of the midges during winter.

Clinical signs

In endemically infected countries, the appearance of clinical signs of bluetongue coincides with peak activity of the insect vectors. Bluetongue is, therefore, a seasonal disease, the incidence being highest during wet and warm summer and autumn months. This in turn is influenced by the availability of alternative hosts for the insect vectors, cattle being preferred to sheep. Where cattle are also present, bluetongue does not appear in sheep until the *Culicoides* midge population is sufficiently high, generally in late summer. The severity of the disease also depends on the strain of virus, breed of sheep, time of shearing and other predisposing stressors, such as hot sunlight and gastrointestinal parasitism.

High fever (up to 42°C) lasting for several days is followed by respiratory distress, with salivation, nasal discharge and oedema of the lips, tongue and submandibular space. Ulcers may develop on the lips, dental pad, gums, palate and tongue. The name ‘bluetongue’ comes from the swollen and cyanotic tongue seen in severe cases. There may also be lameness, with hyperaemia of the coronary band. Animals can

lose condition rapidly and rams become infertile associated with prolonged pyrexia. Pregnant ewes abort or give birth to congenitally deformed lambs. Haemorrhagic diarrhoea is a poor prognostic sign.

Morbidity in susceptible sheep is 100 percent, although severity varies from appearance as sudden death to mild fever with a few ulcers on the tongue. Following the acute disease, surviving animals have a protracted convalescence, or may die from secondary bacterial infections.

Following recovery, sheep have lifelong immunity only to that particular serotype of the bluetongue virus.

Diagnosis

The postmortem appearance of sheep that have died from bluetongue is seldom spectacular, and there are no pathognomonic lesions. Signs of congestion or ulceration of mucous membranes, oedema of the face, ears, eyelids, lips and tongue may be apparent. Other postmortem signs include oedema of the lungs with froth in the trachea, intermuscular oedema, and blood-stained fluid in the peritoneum and thorax. Lymph nodes are swollen and there are widespread haemorrhages. Secondary bronchopneumonia is frequently associated with aspiration of forestomach contents. In pregnant animals there is a haemorrhagic necrotic placentitis.

The diagnosis of bluetongue is confirmed by virus isolation (on whole blood), PCR on various tissues, or serology for group specific antibody (ELISA). In the naïve UK sheep flock, demonstration of antibody alone would be sufficient to confirm the presence of the disease. Serum samples from several animals are required for gel precipitin and ELISA tests. BTV can be isolated from a number of tissues from dead animals.

The situation in Australia

In Australia, bluetongue-infected *Culicoides* midges were first identified in 1977, although disease in livestock is rare. The serotypes present are pathogenic, and do cause disease when experimentally inoculated into susceptible sheep. However, the field conditions seldom allow expression of the disease for several reasons:

- There is a relatively large cattle population in northern Australia for the midges to bite first. (Many of these cattle are seropositive for bluetongue virus, in areas where sheep are not.)
- The early timing of shearing offers the sheep some protection, as the midges prefer freshly shorn sheep.
- The breed of sheep in the area. (Merinos are less susceptible to bluetongue than northern European breeds.)

Control in infected countries

In all countries so far endemically affected by bluetongue virus, authorities have been obliged to accept the presence of the disease, without actively eliminating it.

Newborn lambs receive passive colostral antibody protection for about 70 days, but exposure to each serotype would be required to ensure comprehensive immun-

ity. The immunogenicities of the freeze-dried, live attenuated virus vaccines that are available are hit or miss. Live vaccines are specific for a BTV serotype, sometimes necessitating the costly use of different pentavalent combinations given on successive months, with the aim of providing protection against about half of the BTV serotypes. Vaccination of pregnant ewes may lead to foetal death or resorption. The attenuated virus used in live vaccines can also be transmitted by midge vectors and may show re-assortment of epitopes to produce a more virulent virus than the infecting strain.

Other potential control methods include:

- Early shearing so that there is a protective wool regrowth by the time of peak *Culicoides* activity.
- Avoidance of high-risk areas, insect breeding sites such as warm, wet gullies.
- Housing animals at night. Midges do not usually enter buildings.
- Insecticidal or repellent dips or sprays.
- Grazing sheep alongside cattle.
- Application of larvicides to insect breeding sites.
- Development of a killed vaccine.

Bluetongue in northern Europe

Since 1999 bluetongue has sporadically been reported in Greece, Italy, Corsica, the Balearic Islands and several Balkan countries, associated with incursion of infected *Culicoides imicola* vectors from Turkey and northern Africa. In 2001 it was first suggested that the bluetongue virus might have overwintered in *Culicoides* midges southern Europe. This progressive northerly spread of bluetongue is cause for concern because it is probably related to the effects of climate change both on the survival and spread of the midge and on the capacity of the midge to transmit various vector borne viral diseases.

In 2006, Bluetongue was reported in sheep in the Netherlands, resulting in about 25% morbidity and 15% mortality in infected sheep flocks, and necessitating the implementation of expensive statutory control measures and trade restrictions. Cases were subsequently identified in neighbouring regions of Belgium, France and Germany. In 2007 bluetongue resulted in up to 70% morbidity and 50% mortality in sheep flocks in infected regions of north-west Europe as well as substantial consequential management problems, including poor reproductive performance. The disease in 2007 was first seen in flocks that had been infected in 2006. Many sheep died as a result of an acute haemorrhagic disease and cattle, which were previously generally considered to be symptomless carriers of bluetongue virus were also severely affected, with a 50% morbidity rate in some herds. While the mortality rate in cattle was not high, erosive lesions involving the nose, mouth and teats and severe coronitis led to welfare problems, which frequently necessitated their humane destruction. Unusually, clinical signs of bluetongue were also seen in goats.

Bluetongue was first reported in eastern England in September 2007, having been introduced by airborne spread of infected midges from northern mainland Europe. The virus was subsequently shown to have circulated between local animal and midge populations. Further cases were identified in December 2007, in cattle imported from within bluetongue-affected regions of the Netherlands, although

there was no evidence of virus spread from these cases. There is now serious concern about the potential recrudescence, possibly from recovered, carrier sheep, or re-introduction and spread of bluetongue within the UK.

The 2006 outbreak of bluetongue which was first identified in the Netherlands was caused by the BTV serotype 8. Previous European outbreaks involved serotypes 1, 2, 4, 9 and 16, with serotype 8 only having been seen in sub-Saharan Africa. The most likely sources of infection were therefore considered to be either wild animals illegally imported from this region, or the introduction of domestic ruminants from eastern Europe which had inadvertently been infected with the serotype 8 BTV, perhaps through the illegal use of live vaccines.

European bluetongue outbreaks indicate that *Culicoides pulicaris* and *Culicoides obsoletus* (a complex of four species), which are present in the UK, are important vectors of BTV. Furthermore, 8 of the 35 species of midge present in the UK, including the common highland midge, *Culicoides impunctatus*, are experimentally competent to transmit the BTV.

Disease prevention in the UK

Besides the obvious effects of bluetongue on animal production and management, the presence of the BTV in the UK could have serious consequences for trade. Export certificates for dairy products, wool, skins and hides all require freedom from bluetongue, despite the low probability of their being a source of infection. To prevent bluetongue from being introduced to the UK, animals are serologically tested prior to importation. No animals are imported from high risk areas and quarantine restrictions are enforced. However, nothing can be done to prevent the wind-borne spread of infected vectors from northern mainland Europe.

Midge control involving drainage of breeding sites or the environmental use of insecticides is generally impractical. Repellents are generally ineffective, and avoidance by housing may be futile, because some of the *Culicoides* vectors can enter buildings.

Measures to control the spread of Bluetongue in the UK include restrictions within protection and surveillance zones around the infected holding. Legislation provides for movement restrictions on suspicion of the bluetongue virus circulating in an area. It also provides for eradication of the disease by control of the vector, and if appropriate slaughter and/or vaccination. However, should BTV start to spread within the UK, vector control and slaughter are likely to be inadequate and the preferred option for the control of bluetongue, would involve the use of a dead BTV serotype 8 specific vaccine. The successful production of such a vaccine was announced in January 2008. The situation with regards to bluetongue in the UK is evolving rapidly. More accurate, detailed, up-to-date information is available on the DEFRA website.

Contagious epididymitis (Brucella ovis)

Brucella ovis is an obligatory pathogen of sheep and deer. *B. ovis* epididymitis has not been diagnosed in the UK, but is an important cause of poor ram reproductive performance in Australia, New Zealand, the USA, South Africa and parts of Europe, necessitating expensive disease control programmes. The prevalence of *B. ovis* is thought to be high in southern European countries.

Disease transmission

Infected rams excrete *B. ovis* in their semen. Rams can be infected through any mucous membrane, but the most likely route of infection is venereal, with spread amongst groups of young rams at puberty. Ewes also have a role in the spread of infection, as temporary physical carriers of infection after mating with an infected ram. While there are reports of *B. ovis* abortion in ewes, infection in the ewe is transient and ewes are not responsible for the maintenance of infection in a flock from one breeding season to the next. Deer stags can become infected by licking at infected ram semen and it is possible that rams may be infected from stags in a similar way.

Pathogenesis

Following infection, *B. ovis* remains localised for some time in the mucous membrane and regional lymph node. Infection may then progress through a bacteraemic phase before localisation in the epididymis and accessory sex glands, and occasionally in other organs. Pathological lesions are generally confined to the epididymis and accessory sex glands. Most lesions are found in the tail of the epididymis, although lesions in the head and body of the epididymis are sometimes palpable. The initial lesions are inflammation of the tubular interstitium, with oedema and lymphocyte infiltration. Later the tubular epithelium becomes inflamed and hyperplastic, leading to narrowing of the tubule, sperm stasis and leakage of sperm into surrounding tissue to form a 1 to 3 cm diameter, palpable granuloma. Sperm granulomas become progressively caseated and fibrotic. Infected rams produce semen of variable quality, depending on the stage and severity of infection. Where more rams are used than are required, flock productivity may be little affected, but where single-sire mating is practised, or the ratio of ewes to rams is high, *B. ovis* infection can result in reduced lambing percentages.

Diagnosis

The diagnosis of *B. ovis* epididymitis is based on the palpation of spermatic granulomas and on serology. Not all infected rams have palpable lesions. Various tests are available, but the CFT offers the best combination of sensitivity and specificity. ELISA and gel diffusion tests are used to support CFT results in cases where improved sensitivity or specificity is desired. However, not all infected rams are seropositive and some rams may be seropositive but no longer infected, the organism not having localised in the epididymis. Confirmation can be provided by positive bacterial culture from semen, although failure to culture *B. ovis* provides little diagnostic information.

Control in endemically infected countries

In New Zealand, a voluntary national *B. ovis* control scheme was launched in 1986, with the primary aim of controlling the disease in ram breeding flocks, thus preventing the spread of infection by purchased rams. Similar schemes have been applied in North America. The national accreditation scheme was initiated by members of the New Zealand Veterinary Association's Sheep and Beef Cattle Society, and supported by sheep breed societies and the Central Animal Health Laboratory. Before

the introduction of the control scheme, it was estimated that *B. ovis* was present in about 14 percent of all New Zealand ram breeding flocks and 15 percent of commercial flocks, with a flock prevalence of infection ranging from 10 to 70 percent. In 1997, 90 percent of all stud ram flocks were accredited *B. ovis* free and less than 1 percent of stud rams reacted to the CFT. This reduced incidence of *B. ovis* infection in stud flocks has been accompanied by a substantial reduction in the incidence of the disease in commercial flocks, indicating that the voluntary control scheme has been effective.

The control of *B. ovis* is aimed at the eradication of the disease from flocks by a test-and-cull procedure, and the maintenance of disease-free flocks through regular screening and the purchase only of accredited replacement rams. Palpation and serology using the CFT are used to detect infected rams, although in some circumstances the use of other supportive tests is indicated. When *B. ovis* infection is identified in a flock, the options are:

- immediate eradication by total replacement
- individual animal test-and-slaughter
- gradual eradication by managing a two-flock system.

The choice of methods is determined by a number of management factors such as the number of rams infected, the flock size, and security of fencing within the farm.

Vaccines are available, but they only confer partial flock immunity, so cannot be used to eliminate infection from a flock. Furthermore, vaccine serological titres interfere with disease eradication involving testing and culling.

Prevention in the UK

The European Union requires rams which are traded between member states to be blood tested for *B. ovis* before export, and to originate from flocks in which no cases of contagious epididymitis have been diagnosed in the previous 12 months.

Brucella melitensis

Brucella melitensis is an important cause of abortion in Mediterranean countries, eastern Europe and parts of Africa, Russia and China. The disease does not occur in northern Europe, the USA, Australia or New Zealand.

Pathogenesis

The pathogenesis of *B. melitensis* is similar to that of *Brucella abortus* in cattle. Following infection through the nasopharynx or across abraded skin, *B. melitensis* spreads via the lymphatics to lymph nodes. In susceptible animals, the organism survives and replicates inside phagocytic cells, while in immune or resistant animals it is killed by macrophages. Following repeated cycles of replication, disruption and re-infection of macrophages, susceptible sheep become bacteraemic, *B. melitensis* being found in blood for 30 to 45 days after infection. In pregnant animals, the organism then enters the uterus and reproduces in placental and foetal tissues. Some ewes abort at this stage, following which vaginal excretion of large numbers of *B. meliten-*

sis bacteria persists for several months. Non-pregnant animals become chronically infected. The mammary gland in often colonised, interfering with milk production, and possibly acting as a reservoir of infection until the ewe next becomes pregnant, when abortion may occur.

Disease transmission

The main source of infection arises from products of abortion, which can give rise to environmental contamination for about 1 month afterwards. While infected sheep are the most likely cause of spread into previously clean areas, localised spread of infection between flocks may be due to dogs or birds carrying infected placentae. Cattle can become infected from sheep, but play little role in the transmission of the disease. While *B. melitensis* can cause orchitis, rams are not believed to be important for the transmission of disease, other than perhaps as mechanical vectors at mating. As with *B. abortus* in cattle, viable offspring from infected females may also be infected but seronegative, and may shed infection following their first parturition or abortion. Thus, these animals pose a significant risk when imported into an uninfected flock, and it is essential that animals are added only from flocks of known disease-free status.

Clinical signs

Abortion storms occur following the introduction of *B. melitensis* into naïve flocks, accompanied by fever, depression, mastitis, arthritis, synovitis, orchitis or nervous signs. In chronically infected flocks, fewer abortions occur and clinical signs are seldom seen. *B. melitensis* cannot be distinguished from other causes of abortion on the basis of clinical signs and gross pathological lesions alone.

Diagnosis

Brucellae can be identified in specially stained smears of foetal stomach contents or vaginal discharges. Stained *B. melitensis* antibody methods can also be used. The organism can be cultured using selective media from various tissues and products of abortion. However, while bacterial culture provides unequivocal evidence of infection, negative results do not indicate its absence.

Several serological tests are available. The serum agglutination test (SAT) is useful for screening, but its sensitivity and specificity are poor in chronic cases. The specificity of the Rose Bengal test is poor when it is used with an antigen concentration sufficient to ensure good sensitivity. The CFT offers a generally reliable combination of sensitivity and specificity. Other serological tests such as an ELISA may be useful to determine whether individual positive results are true or false. Some serological cross reactions occur, for example with some salmonella serotypes and *Yersinia enterocolitica*.

Disease control in infected countries

Treatment of infected sheep is ineffective. The control options for infected flocks are:

- identification and culling infected sheep
- vaccination and culling infected sheep
- vaccination.

Attenuated (Rev-1) and inactivated (H-38) vaccines are available. Vaccination is an adjunct to disease control, but does not provide complete protection. The attenuated vaccine can cause abortions when administered to pregnant sheep.

In disease-free flocks the risk of *B. melitensis* infection is reduced by:

- only introducing sheep from known non-infected flocks
- isolating introduced animals
- periodic clinical examination and serological testing of rams
- submission of all abortion material for laboratory diagnosis.

Disease control in the UK

The UK is recognised as officially free from *B. melitensis*. Imported sheep or goats must originate from an officially *B. melitensis*-free holding and be subject to testing in isolation before the movement.

Material submitted for veterinary laboratory abortion investigations is routinely screened for *B. melitensis* to demonstrate with 95 percent confidence that fewer than 0.2 percent of holdings in the UK are infected.

Zoonosis

In those countries where infection widespread, most animals become immune. Thus disease does not cause serious production losses in sheep. However, *B. melitensis* is an important zoonosis, causing fever, sweating and weakness in humans, sometimes referred to as Malta fever. Human infection usually results from drinking raw milk or eating dairy produce, but can also occur following contact with secretions from infected sheep.

Peste des petits ruminants

Peste des petits ruminants (PPR) is a rinderpest-like disease of small ruminants. PPR is enzootic in East and Central Africa and the Middle East, where losses can be high and the disease is seen as a major constraint on the availability of animal protein for human consumption.

Spread of the disease is only by contact between infected and susceptible live animals. The disease has never occurred in the UK, so the small ruminant population is totally naïve. Therefore, if PPR were to be introduced, mortality rates would be high. Furthermore, the effect on sheep and goat exports would be substantial.

Cause and transmission

The disease is caused by a morbillivirus, similar to canine distemper and measles viruses. The PPR virus is distinct from, but very closely related to, the rinderpest virus of cattle and is highly adapted to sheep, goats and other small ruminants such as deer. Transmission is by close contact, the virus being present in all secretions and

excretions. The virus does not survive in fomites, but insects may be involved as mechanical vectors, for example flies via conjunctival contact.

Clinical signs

In susceptible animals, the morbidity is high (100 percent) and mortality can be up to 90 percent. The incubation period is short (2 to 6 days), with rapid-onset fever, naso-ocular discharge, initially serous, but becoming mucopurulent, followed by diarrhoea, and, in susceptible animals, death within 1 week of onset of clinical signs. In the later stages of the disease, there is crusting around the mouth, associated with mucopurulent discharge and stomatitis, similar in appearance to orf. Surviving animals are strongly immune.

Diagnosis

Postmortem findings include extensive erosive stomatitis and pharyngitis, epithelial erosions and ulceration in the upper respiratory tract and pneumonia, frequently with secondary pasteurella infection. Enteric lesions with a 'zebra-striped' appearance are frequently identified. Confirmation of the diagnosis depends on virus isolation from lymph nodes or tonsils of freshly dead animals. Antibody detection is only useful in naïve populations.

Control

In endemically infected countries, annual vaccination with a live attenuated virus strain is practised.

Prevention in the UK

Suspicion of PPR is compulsorily notifiable. Should the disease be introduced to the UK, affected animals would have to be slaughtered, and a 3 km protection zone and 10 km surveillance zone set up around the infected premises. After cleansing and disinfection the restrictions would remain in force for at least 21 days (the incubation period for the disease).

Capripox

Sheep and goat pox are caused by a single virus, and are an important cause of economic loss in many parts of the world due to mortality and production losses. The diseases are endemic in Asia and Africa, and occasionally reported in southern Europe and North America. A combination of dry environment and traditional nomadic pastoral systems of agriculture make the disease difficult to eradicate from these areas. Sheep pox was present in the UK between 1847 and 1866, having been introduced with a flock of imported sheep. The UK has subsequently remained free from the disease.

Cause

Sheep and goat pox are caused by a capripox virus, which is capable of survival in scab material in the environment for periods as long as 3 months. Transmission is through close contact and inhalation or from an infected environment. Dry and dusty conditions favour both transmission and survival of the pox virus in the environment. Flies may also mechanically transmit the disease.

Clinical signs

A wide range of clinical signs reflects differences in virulence of virus strains and differences in the host response. Sheep of all ages are affected, although the disease is most severe in young animals. Signs of peracute disease, as occur in a naïve population, include high fever, paralysis, a generalised haemorrhagic syndrome, widespread cutaneous ulceration and death, within a few days. This form of the disease is seen mostly in young lambs, and the morbidity rate can be 75 percent and mortality rate 50 percent. In acute infections, signs start with high fever, naso-ocular discharges and salivation. Papules appear within 24 hours on external mucous membranes of the eyes and nose and intradermally, being most obvious on the wool-free parts of the skin. Lesions are initially circumscribed, but become confluent. After the fever regresses, the papules become encrusted and form scabs, which become secondarily infected, and are irritant.

A more chronic, nodular form of the disease is sometimes reported in older sheep. Affected animals have high temperatures and suppressed appetite. Eruptions appear on the mucous membranes of the nose and mouth and on wool-free parts of the skin. Palpable lesions also appear in parts of the body covered by wool. The lesions often develop to thick nodules, oozing a yellow discharge and forming crusts. There is evidence of respiratory disease and pregnant ewes often abort.

Diagnosis

Vesicular and nodular lesions, especially in parts of the body not covered by wool, are characteristic. At postmortem examination, lesions are also found in the respiratory and alimentary tracts. The larynx is inflamed and sometimes ulcerated. Lesions in the lungs consist of depressed grey areas up to 3 cm in diameter and secondary bronchopneumonia. Nodular lesions are frequently seen in the lining of the abomasum. The important differential diagnosis for the resolving acute and chronic forms of capripox infections is orf. To confirm the diagnosis, capripox virus can be identified using a direct fluorescent antibody test.

Control

Live attenuated vaccines are available for use in endemically infected areas.

Prevention in the UK

Should capripox be introduced to the UK, affected animals would be slaughtered and a 3 km protection zone and a 10 km surveillance zone imposed around premises

where disease is confirmed. Restrictions would remain in force for a minimum of 21 days, the incubation period of the capripox virus.

Contagious agalactia

Contagious agalactia is a septicaemic disease of sheep and goats, caused by *Mycoplasma agalactiae* and other mycoplasmas. The prevalence of the disease is high in southern Europe and eastern Asia and parts of North Africa, but it has never been diagnosed in the UK.

Transmission occurs by direct contact with infected sheep. Lambs become infected through their dams' milk. Fomites may play a role in the localised spread of infection.

Clinical signs

Following an incubation period of up to 60 days, sheep become bacteraemic, with signs of general malaise, inappetence and pyrexia (up to 42°C). Pregnant ewes may abort during this period. In surviving lactating animals the disease progresses to a severe, bilateral mastitis, the milk appearing yellow, clotted and granular, eventually resulting in atrophy and fibrosis of the udder. The prevalence of mastitis within an infected flock may be high and lambs are ill thrifty due to a lack of milk. Acute mastitis is often accompanied by arthritis, usually involving the tarsal and carpal joints. Affected sheep are lame and their joints are swollen and painful, but do not contain pus. Concurrent unilateral or bilateral keratitis is sometimes seen, resulting in blindness. Younger sheep may develop respiratory disease, leading rapidly to death, without development of other signs.

The relevant importance of mastitis, abortion, lameness and keratitis differs between regions, possibly associated with local management practices and the species of mycoplasma involved.

Diagnosis

The diagnosis of contagious agalactia is based on the clinical signs and postmortem findings and confirmed by bacteriology and serology.

The postmortem findings vary with the stage of infection. Sheep which died during the acute stages of the disease may show generalised peritonitis. Affected udders are atrophied and fibrosed and affected joint capsules are oedematous, with clumps of fibrin within the joint and erosion of articular surfaces.

M. agalactiae can be cultured from milk and joint fluid. Bacterial culture is useful in the acute stages of the disease, but often fails to identify infection in chronically infected sheep. PCR tests can be used to improve sensitivity and produce quicker results. Serology using a CFT is used for flock screening, but has poor sensitivity, while current ELISA tests lack specificity.

Disease control

Antibiotic treatment is seldom totally effective. Live attenuated vaccines have been used to some effect, but their use interferes with longer-term disease control.

Rift Valley fever

Rift Valley fever is a peracute, mosquito-transmitted, viral disease of sheep, goats and cattle. Clinical disease is seen most frequently in sheep, characterised by high mortality rates in young lambs and abortion storms in pregnant ewes. The disease is also a potentially fatal zoonosis. The disease was first identified in the Rift Valley of Kenya, and is mostly seen in sub Saharan Africa. However, in recent years it has also occurred in Egypt, Saudi Arabia and the Yemen, where it has caused serious production loss and human disease.

Cause

Rift Valley fever is caused by a bunyavirus, similar to the Akabane disease virus. Disease is naturally transmitted by several species of mosquito. Following a bite from an infected insect, the incubation period in sheep is between 2 and 4 days. Transovarian transmission can occur within the mosquito population, serving as a reservoir of infection. Disease outbreaks are related to rainfall and subsequent mosquito activity.

Clinical signs

Young lambs become lethargic, weak and inappetent, with pyrexia (up to 41°C) and develop dysentery, progressing to death within 24 hours. Some lambs die suddenly without showing clinical signs. Mortality rates in lambs under 1 week old often reach 95 percent.

Infection in adult sheep is often inapparent. When clinical signs are seen, they include fever, blood-tinged or foetid diarrhoea, laboured breathing, rhinitis and jaundice. Haemorrhages, bleeding and congestion are sometimes seen in the mucous membranes of the eyes, mouth, nose, rectum and vulva. Regurgitation has been reported. Febrile animals are severely leucopenic. Many adult sheep recover over a period of about 1 week, although mortality rates can reach 30 percent. Abortion storms approaching 100 percent are seen when pregnant ewes are involved.

Diagnosis

The diagnosis of Rift Valley fever is based on the clinical signs, haematology, biochemistry and postmortem findings and confirmed by virus isolation from blood or tissues collected during postmortem examination.

The gross postmortem findings include jaundice of the carcase, 1 mm white necrotic foci on the surface of the liver associated with subcapsular haemorrhages, and haemorrhages in the intestine and spleen and on the surface of the kidneys. The lesions have a characteristic histological appearance.

CFT, ELISA, immunodiffusion and immunofluorescence tests are generally sensitive and specific.

Disease control

Protection from mosquitoes is generally impractical so, in endemically infected areas, control depends on vaccination. The use of a live vaccine in pregnant ewes can result in abortion and foetal abnormalities.

Disease prevention in the UK

It is highly unlikely that Rift Valley fever virus will be introduced to the UK. However, the disease is notifiable and provisions exist to confirm the diagnosis if it is suspected.

Zoonosis

Rift Valley fever is a potentially serious zoonotic disease, causing sudden-onset, severe flu-like symptoms within 2 to 6 days after infection. Occasionally, the disease is more serious, resulting in widespread haemorrhages, encephalitis, ocular disease, severe liver damage, kidney failure and even death.

Most human infections result from direct contact with infected animals, although infection via mosquito bites can occur.

Anthrax

Anthrax in farmed animals is caused by the ingestion of spores of the Gram-positive, rod-shaped bacteria, *Bacillus anthracis*. Anthrax spores can survive in a contaminated environment for several decades, providing an enduring source of subsequent infection. Spore germination and rapid multiplication of the bacteria results in an endotoxin-mediated, apoplectic and generally fatal disease. Most UK cases are diagnosed in cattle, although other species, including sheep and humans, can also be affected.

The clinical signs seen in cases of anthrax in ruminants include separation from the group, anorexia, mucosal congestion, high pyrexia (up to 42°C), muscle tremors and dyspnoea, with terminal convulsions, collapse and death. Illness sometimes persists for a few days before death, but most affected animals are found dead. Unclotted blood is characteristically seen oozing from the nostrils, mouth, anus and vulva, but these signs are commonly present following other causes of death, and their absence does not rule out the diagnosis of anthrax.

Any suspicious sudden death must be reported to a veterinary practitioner, who must then inform the local Animal Health Office. Bacterial sporulation occurs when infected body fluids come into contact with air, so carcases must not be moved or opened until a diagnosis of anthrax has been ruled out. In these cases, the diagnosis of anthrax is confirmed by the identification of bacilli, with distinctive pink capsules, in polychromatic methylene blue-stained blood smears, made from a small nick in an ear vein. Blood smears must be made as soon as possible after the animal's death, before the confounding overgrowth of putrefactant bacteria occurs.

If carcases of animals which have died from anthrax are inadvertently opened, non-specific postmortem signs of extensive congestion and oedema, involving the lungs and lymph nodes in particular, and pulpy swelling of the spleen are sometimes seen.

Human contact with *B. anthracis* spores is now rare. Infection used to occur in tannery workers, resulting in the formation of boil-like skin lesions, which develop a black centre. These cases generally respond to antibiotic treatment. The term anthrax literally means 'coal-like', describing the appearance of human skin lesions. Inhalation of spores, which occasionally occurred in wool sorters, results in severe and generally fatal respiratory disease.

Rabies

Rabies is a fatal disease of the nervous system, caused by a rhabdovirus, which can affect all mammals including humans. Rabies was eradicated from the UK in 1922 and freedom from the disease is now protected by the Pet Travel Scheme and quarantine regulations. Occasional incursions of rabies are associated with European bats carrying rabies-related lyssaviruses.

The rabies virus is usually spread by saliva from the bite of an affected animal. In endemically infected countries, rabies in sheep usually results from exposure to sylvatic reservoir (infected wildlife) hosts.

The clinical signs of rabies in sheep involve subtle changes in behaviour, progressing to muscle weakness, drooping eyelids, profuse salivation and difficulty swallowing due to paralysis of pharyngeal muscles. The disease is invariably fatal.

Aujeszky's disease (pseudorabies)

Pigs are the only natural hosts for the Aujeszky's virus, but sheep and other domestic mammals can be infected through direct or indirect contact with infected pigs, causing fatal central nervous system and respiratory disease. The clinical signs include shivering, incoordination and weakness. The diagnosis of Aujeszky's disease is confirmed by virus isolation, or fluorescent antibody test serology.

Identification of UK sheep

The ability to trace sheep movements is fundamental to the control of any future exotic disease outbreak. The Sheep and Goats Identification and Movement (Interim Measures) Order 2003 states that all sheep and goats born after 1 February 2003 (or still on the holding on which they were born and not yet marked) must be individually identified with a tag or tattoo before they move, or within one year of birth, whichever is earliest.

Sheep and goats must be tagged or tattooed with either a holding of birth (UK) mark or an S tag showing the current holding. The holding of birth flock mark or the S mark must continue to be recorded for all movements in the on-farm record book and on movement documents. Only three official marks per animal are permitted. Up-to-date information on sheep identification is available on the DEFRA website.

Appendix B: Overview of the structure and economics of the UK sheep industry, highlighting the increasing need for efficient flock health management

Sheep are farmed throughout the world, with most production constrained by temperature and rainfall to islands, coastal regions and the fringes of continental deserts. In some regions sheep are used to exploit pastures which are unsuitable for other agricultural purposes, while elsewhere sheep production is integrated into other agricultural systems to enable cost-effective and efficient grassland management or crop rotation. Thus, worldwide sheep management differs greatly to cater for different systems of input and types of output.

The sheep industry is an international business so, to a certain extent, the economics of sheep production are beyond the control of individual UK sheep producers. The largest sheep populations are in China and Russia, but the most important contributors to world trade are the European Union (EU), Australia and New Zealand.

The international sheep industry produces four major products: wool, sheep meat (mutton and lamb), milk and skins. Although the use of wool in textiles has faced major competition from synthetic fibres, the world production of wool is relatively stable at just over 2 million tonnes. The major wool producers are Australia, Russia, New Zealand, Argentina, South Africa and Uruguay and the size of these countries' national flocks is determined by the wool trade. Apart from Russia, which also imports a large amount of wool, these countries are major exporters of wool to the EU, Japan and China. The world supply of sheep meat is dominated by Australia and New Zealand, exporting mainly to Japan and the EU. New Zealand is the world's largest trader of sheep meat. There is also a fluctuating market for live exports from Australia to the Middle East. Meat produced by Asia, Africa and Russia is mainly for home consumption. Sheep are kept for milk in Africa, Asia and the Mediterranean countries, much of which is used for manufacturing cheese. Sheep skins are often considered as a by-product, but are a significant source of income for UK abattoirs, at the time of writing valued at between £5 and £7 per skin.

The European Union sheep industry

In 2000, the sheep population of the EU numbered 96 million, the majority of which were based in the UK (31 percent), Spain (25 percent), Italy (11 percent), France (10 percent), Greece (9 percent) and Ireland (6 percent). The EU produced over

Sheep Flock Health: A Planned Approach. Neil Sargison
 ISBN: 978-1-4051-6044-5

1.1 million tonnes of sheep meat and imported 277 880 tonnes, mostly from New Zealand and Australia. The EU was 80 percent self-sufficient in sheep meat.

Sheep production in southern Europe is based on triple-purpose breeds, and is relatively unimportant in terms of trade. About 80 percent of sheep meat imported by the EU is from New Zealand, the remainder being from Australia and eastern Europe.

Sheep meat is the only sheep product regulated under the EU Common Agricultural Policy (CAP). Wool is considered to be an industrial product, sheep skins are regarded as a slaughterhouse by-product and the sheep milk market is relatively small, being confined to Mediterranean regions.

The UK sheep industry

The UK sheep population steadily increased to a peak of about 18 million ewes in the early 1990s. Since then, the national sheep flock has contracted due to low economic margins, changes in subsidy rules and lack of confidence in the industry. The UK breeding flock currently numbers about 16 million ewes.

The UK outbreak of foot-and-mouth disease in 2001 had a substantial impact on the sheep industry. It is estimated that over 5.5 million sheep were slaughtered, leading to a loss of 2.4 million sheep (about 12 percent) from the breeding flock.

Stratification of the UK sheep industry

The UK sheep flock is unique in that it is characterised by a stratified three-tier breeding structure, employing systematic crossbreeding. This stratified system of sheep production enables sheep farming in a wide range of climates, environments and management systems and is ideally suited to traditional sheep breeding management. Stratification enables hill breeds to be kept in inhospitable environments, producing replacement hill ewe and ram lambs, lightweight store or finishing lambs and cast ewes. On upland country, regular aged or cast hill ewes are crossed with longwool rams to produce hybrid ewe lambs and store or finishing wether lambs. Prolific, hybrid ewes such as Greyfaces, Mules and Halfbreds are crossed with terminal sire rams to produce the majority of UK finished lambs. Purebred down breeds are kept on some lowground farms, providing finished lambs and replacement terminal sire rams. There are several variants of this system, such as the practice of mating draft hill ewes directly with a terminal sire breed for slaughter lamb production.

The main advantages of stratification are: that it exploits the characteristics of different breeds, so that Greyface or Mule ewes combine longwool traits of growth rate, litter size and wool production with hill traits of hardiness and manageable size; it enables the use of terminal sires on crossbred ewes, thus producing lamb carcases tailored to the needs of the market; and it matches the attributes of the different breeds to different areas of land use.

The main disadvantages of stratification are: that it necessitates extensive animal movements, providing an opportunity for spread of disease and raising concerns about the welfare of animals during transport; that the majority of finished lamb producers must rely on the purchase of replacement crossbred ewe lambs or gimmers, hindering selection for production characteristics such as ease of lambing, longevity, milk production, back fat depth, birthweight, growth rates and resistance to disease;

Fig. B.1 A major aim of the Common Agricultural Policy was to ensure an adequate standard of living for sheep farmers in remote areas, thus maintaining the character of those areas.

and that failure to meet the demand for crossbred ewes can lead to short-term supply problems that result in unrealistic prices for breeding ewes.

The Common Agricultural Policy

The original objectives of the Common Agricultural Policy, set out in the Treaty of Rome, 1958 were:

- To increase agricultural productivity by developing technical progress and the rational development and the optimum utilisation of the factors of production.
- To ensure a fair standard of living for the agricultural population, particularly individual earnings (Fig. B.1).
- To stabilise markets.
- To guarantee regular supplies.
- To ensure reasonable prices to consumers.

Since the 1960s UK sheep farmers received direct support for the price that they received for finished lambs. Prior to 1980 this support was in the form of a deficiency payment scheme. When the UK joined the EEC (now EU) in 1973, a new enterprise, namely sheep meat production, was effectively introduced, which led to the introduction in 1980 of the sheep meat regime. Support under the sheep meat regime was designed to guarantee income to the sheep sector to the level of a basic price for sheep carcases. This basic price was based on what was considered to be a reasonable return to the industry taking into account: sheep meat marketed during the current year; prospects for production and consumption of sheep meat; sheep production costs; the market situation for other meats; and past experience. The basic price was set annually for the beginning of each marketing year, but was subject to seasonal adjustments, based on prices collected for specified categories of animals and carcases from selected markets throughout the EU, to produce a weekly representative market price. There was provision for this price structure to be supported by private storage aid (paying abattoirs for surplus stock to be taken off the market and kept in frozen storage for a period of 3 to 7 months), intervention buying and a variable premium scheme. Most countries adopted a direct intervention scheme, buying lamb to take it off the market when prices fell below a certain level. However, the UK initially adopted a variable premium support system, due to its previous experience of a deficiency payment scheme. A variable premium, corresponding to the difference

between the weekly average UK market price and the guide price, which was 85 percent of the basic price, was paid on lambs under 1 year old that met certification standards. In order to prevent exports from the UK resulting in intervention buying in other member states, a clawback charge was imposed on exports of lamb to EU countries. In addition, ewe annual compensatory premium payments, based on the shortfall between representative price for the year in question and the basic price, were normally paid as a ewe headage payment. This support system guaranteed sheep farmers a reasonable income and meant that they knew in advance more or less what their finished lambs would be worth. The variable premium support was abolished in Northern Ireland in 1984 because of cross-border difficulties with animal movements.

The sheep meat regime was reviewed in 1990 because the cost of supporting it had risen following an increase in the total number of sheep and goats caused by the accession of new EU states. As a result of this review, the variable premium support system was abolished, bringing the UK into line with the rest of the EU. Changes were introduced in 1992, whereby the amount of support available was slightly reduced to take into account changes in EU sheep numbers, but still based on the basic price. The major source of support for the sheep industry was transferred to the annual ewe premium, a headage payment on eligible ewes which was designed to cover the income loss between the average market price and the basic price for lamb. Individual producer limits were imposed on the number of ewes eligible for support, creating sheep quotas. An upper producer limit of 1000 ewes was introduced on which the full annual ewe premium could be claimed and eligible ewes had to be maintained within a defined retention period. Alongside the ewe annual premium, producers in less favoured areas were paid hill livestock compensatory allowances, at two different rates determined by the quality of the land and suitability of the ewe breed. These were only 25 percent funded by the EU.

In 1993, the average subsidy income from hill, upland and spring-lambing lowland ewes was £30.80, £27.00 and £17.70 respectively, compared with gross margins of £43.90, £51.70 and £44.60 respectively. Thus, subsidy support accounted for 70 percent, 52 percent and 39 percent of the gross margins from hill, upland and spring-lambing lowland ewes respectively. While this system provided a guaranteed level of income for sheep farmers, the efficiency of sheep production was determined more by the ability to manipulate subsidy income rather than on animal production, thus favouring non-progressive sheep farming, while not encouraging improved productivity and flock health management. Furthermore, quotas, retention periods and producer limits did little to encourage flexible farm management and constrained the ability of efficient sheep farmers to expand.

The unsustainable level and method of EU agricultural support during the 1990s led to a reform of the CAP, referred to as the mid-term review of Agenda 2000. From 1 January 2005 subsidy support was decoupled from production and paid as a single payment per farm, combining all existing arable, beef and sheep aids. At the time of writing payment is linked to audited checks of compliance to environmental animal welfare, food safety and good farming practice. However, the basis of the payments and management of claims is devolved and therefore differs between Scotland, Northern Ireland, England and Wales.

The introduction of the single farm payment should free UK sheep farmers from many of the complicated rules governing livestock premium payments, enabling

more flexible farm management, improved production efficiency and more effective flock disease management. However, EU subsidy capping and gradual transfer of direct subsidy payments to environmental development expenditure or early retirement schemes, referred to as modulation, will mean that the total amount of subsidy payable to most individual farmers will be less than the amount previously earned from arable aids and livestock premium payments. Thus, many sheep producers will need to improve the economic productivity of their flocks if they are to remain in business. Cross compliance rules place an artificial cap on the maximum output per hectare as a primary economic determinant. Most commentators suggest a healthy future for intensive lowground sheep production, based on the use of home-grown feed and bedding, with a knock-on effect on other sectors providing breeding and finishing sheep. Disease management is crucially important in these systems, so the identification of production-limiting problems and improvements in sheep health management will become worthwhile.

The financial support for sheep farmers from the EU will undoubtedly continue to change over the next decade, creating further incentive to optimise sheep health management.

The UK sheep meat industry

Until the eighteenth century, before southern hemisphere countries became major wool exporters, the primary role of sheep in the UK was wool production. The UK had a major wool manufacturing industry based on home-produced and imported (initially from Spain) wool. A rapid expansion of the UK population in the eighteenth and nineteenth centuries brought a demand for meat, which is now the most important UK sheep product.

Due to the varied nature of sheep production in the UK, the supply of finished lamb is seasonal. Most lambs finished between January and March are derived from the previous season's lamb crop, referred to as the store lamb trade. This is supplemented during late March and April by new season lamb from early lambing flocks. Finished lamb production increases during June and July, as lowland flocks sell lambs from grass, and peaks in September and October when the hill, upland and remainder of the lowland flocks all market their lambs. Finished lamb production then declines to the end of the year when finished store lambs come onto the market.

Finished lambs are either sold directly off farms to slaughterhouses (~60 percent) or indirectly via auction markets (~40 percent). As a general rule, auction markets are preferred in the north and west of the UK, where the majority of hill and upland sheep are found. While the auction system imposes a need for additional animal movements, it plays an important role in ensuring a fair price structure linked to market demand. The price of finished lamb generally reflects variation in supply, with peak prices paid for Easter lamb in early spring, and the lowest prices paid in late autumn.

Carcase classification

Sheep carcases are classified according to conformation (using the EUROP system; with E being the best and P being the worst and fatness (1, 2, 3L, 3H, 4L, 4H and 5;

with 1 being thin and 5 being fat). Only 50 percent of the carcases produced in the UK reach the target classification classes of E, U and R conformation with fat class 1, 2 and 3L.

In 2000, exports accounted for 27 percent of UK lamb production (99 000 tonnes). Of this, 70 percent was to France, with the rest to other EU countries. Export to southern European countries represents an important outlet for light (8 to 14 kg carcase weight) lambs.

Lambs usually kill out at about 48 percent when slaughtered at half their potential normal adult liveweight. Most buyers specify an acceptable weight range and fatness class range, and will not buy lambs outside this range. Fatness is a problem due to human health concerns, consequent reduction in saleable meat yield and consumer preference. Buyers generally accept poorer conformation if there is a lack of supply in the market. Buyers may refuse to purchase or penalise carcases with obvious damage, such as: bruising related to lifting or pulling by the fleece; abscesses associated with faulty injection technique; visual contamination associated with presentation of excessively dirty lambs for slaughter; evidence of chemical residues; or taints and odours.

Store lambs are sold almost exclusively by live auction. This system is vitally important for the production of finished lambs from hill and upland farms. Success in store lamb finishing depends on purchasing store lambs at a low price, keeping feed costs down, disease management and achieving a good selling price.

There is also a strong domestic market for mutton, with over 70 percent of the mutton produced in the UK going to the Halal market. The remainder is predominantly exported to France. For most of the year, the cull ewe price tends to reflect the lamb price, rather than supply and demand.

Live sheep exports are a contentious issue. At their peak in 1999, the UK exported over 1.1 million sheep but the foot-and-mouth disease outbreak in 2001 stopped all live animal exports. The volume of this trade also depends on the sterling to euro exchange rate and lamb prices.

Wool

The British Wool Marketing Board operates a central marketing system for UK wool, and sells the wool onto the world markets. While the annual wool clip in the UK was 37 million kg in 2002, wool is essentially perceived as a by-product. The UK wool cheque is paid in two instalments and at the time of writing is around £0.50 to £0.70/kg, but can vary considerably depending on grade. As the price for contract shearing of ewes is £0.55 to £0.80/ewe, in many cases the wool cheque barely covers the cost of production.

The price that producers receive for their wool is dependent on a number of factors including: grading; excessive kemp, especially in hill breeds; irregular quality; the presence of grey or black fibres; and contamination including tints, stains, excessive marking, presence of twine, daggy or undocked fleeces, unrolled fleeces, scraps, damaged fleeces and the presence of vegetable matter.

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 ISBN: 978-1-4051-6044-5