A Handbook for the Sheep Clinician

7th Edition

Agnes C. Winter and Michael J. Clarkson

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About the Authors

Agnes Winter grew up on a small family farm in the Yorkshire Dales. After graduating from Liverpool Veterinary School she worked in mixed practice in north Wales where her interest in farm animal work, in particular with sheep, developed further. With her Welsh farmer husband she kept a flock of sheep, including a small flock of pedigree Wensleydales with which she had considerable success in the show ring. After some years she returned to Liverpool Veterinary School to undertake a PhD supervized by her co-author Michael Clarkson. After completion of this she became a lecturer, and later senior lecturer, in sheep health. For the last four years of her academic career she was head of the Clinical Department and, on retiring, was made an Honorary Professor in the University. She remains an active member of the UK Sheep Veterinary Society, of which she was President from 1987–88. She has written five other books on various aspects of sheep health and disease and has received several prestigious awards for her contributions to the sheep industry.

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Michael Clarkson was born in Yorkshire and graduated from Liverpool Veterinary School. He undertook specialist studies in the Liverpool School of Tropical Medicine where he taught veterinary parasitology and did research on turkey coccidiosis, cattle trypanosomiasis, liver fluke infection in cattle and sheep and a number of zoonotic infections, especially hydatid disease. Michael has worked in several countries including Kenya, Zambia, Peru, Chile and Libya. He was appointed Professor of Farm Animal Medicine by Liverpool Veterinary School and studied the epidemiology of a number of important sheep diseases including parasitic gastroenteritis, chlamydial and toxoplasma abortion and fasciolosis and continued his interest in hydatid disease. He taught sheep and cattle medicine and worked on a number of sheep farms developing sheep health programmes. He was appointed Emeritus Professor on his retirement and still undertakes research work at the Veterinary School. He has been a member of the UK Sheep Veterinary Society for many years, edited its Proceedings for 25 years, and was President of the Society from 1986–87.

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Agnes Winter and **Michael Clarkson** were the first two Royal College of Veterinary Surgeons Diplomates in Sheep Health and Production and the first two RCVS-recognized sheep specialists.

Abbreviations

AADs Amino-acetonitrile derivatives
AGID Agar gel immunodiffusion test
AHVLA Animal Health and Veterinary Laboratories Agency
AI Artificial insemination

BDV Border disease virus
BLUP Best linear unbiased prediction
BOHB Betahydroxybutyrate
BSE Bovine spongiform encephalopathy
BTV Bluetongue virus
BVA British Veterinary Association
BVDV Bovine virus diarrhea virus
BZ Benzimidazole

CAE Caprine arthritis encephalitis
CaMD Calcium borogluconate, magnesium and dextrose
CAP Common Agricultural Policy
CCN Cerebrocortical necrosis
CF Complement-fixing
CK Creatine kinase
CLA Caseous lymphadenitis
CNS Central nervous system
CODD Contagious ovine digital dermatitis
CP Crude protein
CS Condition score
CSF Cerebrospinal fluid

D value Digestibility value
DET Dry ewe therapy
DM Dry matter

EAE Enzootic abortion of ewes
EBLEX English Beef and Lamb Executive
EBV Estimated breeding value
ELISA Enzyme-linked immunosorbent assay

EM Electron microscope/microscopy
EU European Union

FMD Foot and mouth disease
FR Footrot

GGT Gammaglutamyl transferase
GSHPx Glutathione peroxidase

ID Interdigital dermatitis
IM Intramuscular
IV Intravenous

LA Long acting
LM Levamisole and morantel (drugs)

MAP *Mycobacterium avium* subspecies *paratuberculosis*
ME Metabolizable energy
MJ Megajoule
ML Macrocyclic lactones
MLC Meat and Livestock Commission
MOET Multiple ovulation embryo transfer
MRI Magnetic resonance imaging
MV Maedi visna

NADIS National Animal Disease Information Service
NSA National Sheep Association
NSAID Non-steroidal anti-inflammatory drug
NSP National Scrapie Plan

OP organophosphate
OPA Ovine pulmonary adenocarcinoma

PCV Packed cell volume
PGE Parasitic gastroenteritis
PI3 Parainfluenza virus type 3
PME Post-mortem examination
PMSG Pregnant mare's serum gonadotrophin
PrP Prion protein
PT Pregnancy toxaemia

RCVS The Royal College of Veterinary Surgeons

SAC Scottish Agricultural College
SAHPS Animal Health Planning System
SC Subcutaneous
SCOPS Sustainable Control of Parasites of Sheep
SFP Single Farm Payment
SP Synthetic pyrethroid
SPA Sheep pulmonary adenomatosis
SVS Sheep Veterinary Society

TB Tuberculosis
TBF Tick-borne fever
TSE Transmissible spongiform encephalopathy

VLA Veterinary Laboratories Agency (now AHVLA having merged with Animal Health)
VMD Veterinary Medicines Directorate

Preface

The original idea for this book was to produce a concise practical clinical guide to the diagnosis, epidemiology, treatment and control of the common conditions affecting sheep in the UK and an early version appeared in 1983. It was first used for teaching veterinary students at the Liverpool Veterinary School but we were encouraged when a number of veterinary surgeons and sheep farmers indicated that they found it helpful and it became well known as the 'Green Book'. The last edition was published in 1997 by the Liverpool University Press, although the document has been updated internally every couple of years since then.

The present book has been considerably extended both in the information given on the main conditions and in the geographical range covered. Although the emphasis remains on the common conditions affecting sheep in the UK, we have considered their importance in the countries of northern and southern Europe and elsewhere including Australasia and other southern hemisphere countries and in North America. We have not discussed the conditions of sheep maintained for milk production, however, since that is a specialized area mostly outside our experience.

The emphasis has continued to be on the practical clinical aspects of sheep medicine, concentrating on common and important aspects. No attempt has been made to cover more unusual or obscure conditions. Knowledge of the details of pathology and the organisms involved has been assumed or can be sought elsewhere. Where specific drugs are referred to we have used the generic name; availability and legislative aspects of use refer to UK conditions and may vary in other countries.

We hope that this book will prove a useful practical handbook for dealing with the common conditions of sheep, both as individuals and in flocks and will be helpful to veterinary students and practitioners and also to farmers and agricultural students and advisors.

We are grateful to many veterinary surgeons and farmers who have taught us so much and have benefitted enormously from membership of the Sheep Veterinary Society. We especially acknowledge the contributions to the early editions from our colleagues Bill Faull and Alun Davies.

We appreciate the encouragement we have received and the care and attention given to the publication by the staff of CABI and especially by Sarah Hulbert, Commissioning Editor and Alex Lainsbury, Editorial Assistant.

Agnes Winter and Michael Clarkson
July 2011

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Production

Structure of Sheep Industry and Economics

Sheep are found in almost every country in the world and due to breeding and selection are able to live in environmental extremes as wide as dusty deserts and snowy peaks. It has been estimated that there are over one billion sheep and lambs in the world with the greatest number in China with 128 million followed by Australia with 73 million, India 65 million, Iran 54 million, Sudan 52 million and Nigeria 35 million. New Zealand and the UK have a similar number of around 32 million. The USA has six million and Canada less than one million sheep and lambs. Considerable numbers of sheep are found in South America, especially in Argentina, Uruguay, Peru and Chile.

In the countries of the European Union (EU), the UK and Spain have similar numbers of breeding ewes of around 14 million, with Greece, Italy and France having around seven million with a total of around 60 million.

It is important that veterinary surgeons involved in sheep work should have a working knowledge of the structure of the sheep industry in their own country and should have some appreciation of the value of individual sheep and the way that flock profitability can be improved by veterinary advice and involvement. It is often said by veterinary surgeons that farmers are not willing to pay for advice and on the other hand, many farmers complain that their veterinary surgeon is not willing (or possibly not able!) to provide an overall service for sheep. This service should involve more than the occasional visit to investigate an abortion outbreak, for example, or to carry out a caesarean operation on a ewe which has been brought to the surgery.

Probably the best way to become involved and experienced is to offer to help with the production of a health programme for one of your farmer clients who has sheep as a secondary venture on a dairy herd to which you are already carrying out regular visits to advise on herd health. An alternative would be one or more hobby flocks where veterinary costs are often not such an issue as on commercial farms and the owners are usually extremely enthusiastic. The knowledge which can be transmitted between farmer and veterinary surgeon is mutually beneficial and knowledge learnt on one flock can then be offered to other farmer clients. Membership of organizations like the Sheep Veterinary Society (SVS) and the National Sheep Association (NSA) is essential for the successful sheep clinician.

Sheep are kept under a wide variety of systems which need to be understood for

the clinician's own country. For example, in the UK the stratification system of hill or mountain, upland and lowland farms should be understood (see Fig. 1.1) and the physical and financial performance of the three farm systems should form part of the veterinary input into the farm.

Many sheep clinicians have learnt and gained 'street credibility' by keeping some sheep of their own as there is nothing that sheep farmers like talking about more than sheep! This personal involvement, even on a small scale gives experience on the production, marketing and disease problems and the financial constraints which are similar on large commercial flocks.

It is also invaluable to know the main breeds of sheep and particularly those that are likely to be encountered in the area in which you work. It would be an expert who was able to identify the more than 60 breeds in the UK but finding information about examples of the main breeds in the different layers of the stratification system is relatively easy with the aid of the internet and the excellent charts which are available from many sources such as the British Wool Marketing Board, for example. The main hill breeds are Scottish Blackface, Welsh Mountain and Swaledale (Fig. 1.2), all of which are now bred in large numbers and can be found a long distance from their area of origin. These breeds, bred pure on the mountains for three or four lamb crops, then move to upland farms as draft ewes where they are crossed with a ram of a long-wool breed, especially the Bluefaced or Border Leicester (Fig. 1.3), to give the Mule or the Halfbred which forms the breeding ewes of the lowland farms. These are then crossed with a Down type ram, classically the Suffolk but including continental breeds such as Texel and Charollais to produce the prime lamb for slaughter.

Figures for physical and financial performance are obtainable for many sheep systems in many parts of the world by internet searches from flocks involved in

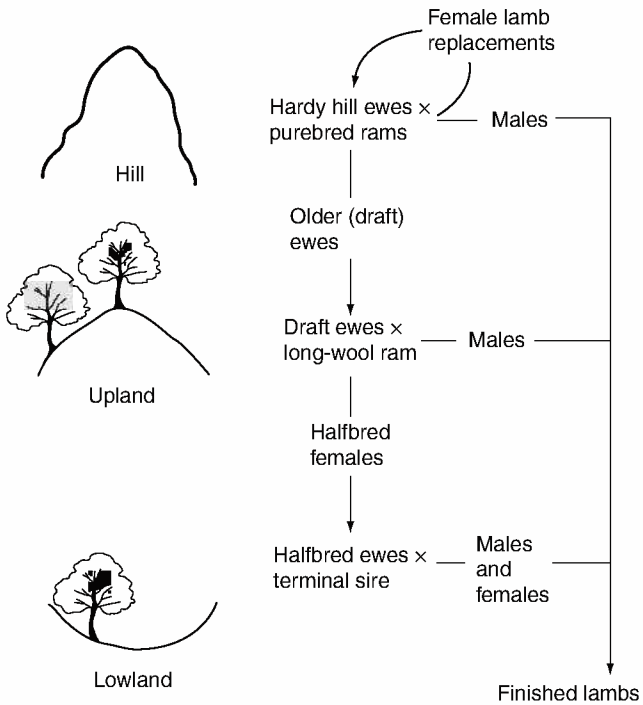

Fig. 1.1. Stratification of sheep in the UK.

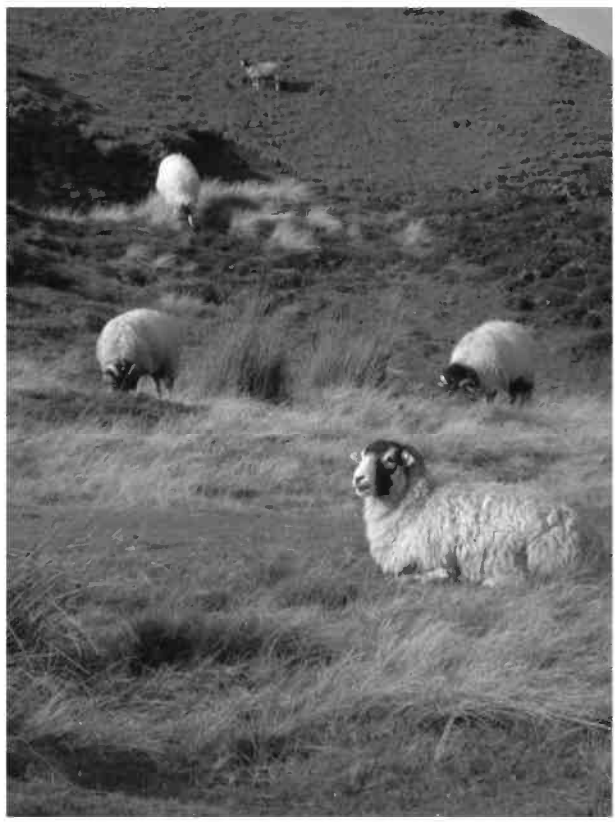

Fig. 1.2. Swaledale sheep in a hill farm environment.

Fig. 1.3. These are Bluefaced Leicesters that are crossed with Swaledales to create the Mule.

recording schemes. These flocks are likely to be above average in their production so may act as targets to improve production by veterinary advice. In England, for example, EBLEX, the English Beef and Lamb Executive produces information from their recorded flocks and divides this into the average of a particular sector and the performance of the top-third farms in that sector. This may usefully form the basis for improving a farm in the average section to one in the top third. This data is available on the web site www.eblex.org.uk/business pointers. The main factors which distinguish the flocks in the top third of the sector are number of lambs reared and stocking density suggesting several areas where veterinary input and the production of farm health programmes can contribute significantly to farm economics. Similar data is available for Scotland and Wales and for many other countries. Production figures are usually expressed as a quantity per 100 ewes which are put to the ram (tupped). Representative figures of lamb performance in the UK are 110 lambs per 100 ewes tupped for mountain farms, 140 for upland farms and 170 for lowland farms, with an average of 10% lamb mortality across all three systems.

While the physical performance figures differ little over recent years, the financial information shows marked differences due to market conditions but up-to-date figures for the cost of cull ewes and prime lambs can be obtained online. The sheep clinician will find this information invaluable in discussions with farmers.

In the EU, there is a complex and massive support mechanism for many agricultural systems including sheep whereas many countries including New Zealand, Australia and the USA removed such support many years ago. The Common Agricultural Policy (CAP) of the EU made payments based on ewe numbers or lamb production but since 2005, this payment, known as the Single Farm Payment (SFP) has been independent of production. In the UK, this has resulted in a marked fall in the numbers of breeding ewes from around 19 million to below 14 million which is doubtless partly responsible for the increase in prices for prime lamb in 2011. Different methods have been used to calculate the SFP for England, Scotland and Wales but producers indicate that without such support, which is likely to reduce, sheep farms will be unable to continue to exist financially. It is important to understand the main features of the schemes in the local area as these may allow or demand a greater input from clinicians into health schemes, which are an essential ingredient for some payments.

There are, however, so many variations that it is not possible to summarize them but the clinician should be aware of the opportunities afforded by the SFP in their own area. In any case, these may change following the review of the CAP due in 2013.

Feeding Management

(This section provides general advice on feeding sheep. See Chapter 4 for a practical exercise on the specific common problem of how to assess a diet which is being fed to sheep for adequacy.)

Grass, either fresh or conserved as hay or silage is the main and cheapest food for sheep in the UK. However, because of seasonality of grass growth coupled with its effect on quality and quantity of the crop and the varying requirements of ewes throughout the production cycle, alternative foods may be used to replace and/or supplement grass, either grazed or conserved. Supplements include simple or complex rations of cereals and protein sources and mineral supplements used when copper, cobalt and selenium concentrations are deficient in grass grown on certain soil types.

Feeding the ram

Many rams are purchased by sheep farmers each year. These, almost invariably, will be in condition score (CS) 3.5–4.5 having been well fed with concentrates in preparation

for sale. It is important that this concentrate feeding is continued up to mating, at no more than 400 g/day. This should minimize the effects of diet change from mainly concentrates to grass only and at the same time prepare the rams for the mating period during which concentrate feeding of the rams only can be difficult.

Rams already on the farm should have a CS of 3.5 (i.e. be fit not fat) 6–8 weeks prior to the beginning of mating. Overfat rams usually have reduced reproductive performance. Most rams will be in correct condition provided reasonable grazing has been available during the summer months.

If there is insufficient grass and/or rams are in poor condition, concentrates and hay will need to be provided during the pre-mating period and possibly during the mating period itself. The amount of concentrates required will vary depending on CS but up to 600 g/day of a concentrate containing 160 g/kg (16%) crude protein (CP) can be provided. Proprietary ewe concentrates should be avoided, especially if large amounts are being fed, because of the high concentrations of magnesium and phosphorus which they contain, predisposing to urinary calculi. Coarse mixes of cereals, sugarbeet pulp and vegetable protein sources with a minimum addition of minerals are recommended.

Feeding the ewe

Condition scoring is an essential guide to feeding management (see Chapter 4).

Pre-mating

The production cycle can be considered to end and start at the time of weaning. At this time, the CS of ewes will depend not only on the amount of food which has been available but also on within-flock factors such as previous suckling load. The period from weaning to mating provides an opportunity to adjust the feeding so that ewes are in optimum condition at mating.

In lowland conditions, where high lambing percentages are expected, the CS at mating should be 3 or 3.5 but in hill flocks, where much lower lambing percentages are required, the optimum score will normally be 2.5.

Increasing the nutrition in the 2–3 weeks pre-mating so that ewes gain 0.5 of a CS (flushing effect) will increase ovulation rate and reproductive performance if ewes are below CS 3. However, if ewes are in good condition, flushing has little advantage over simply maintaining ewes in condition and there is a risk that flushing ewes already in good condition will lead to ewes becoming overfat which may result in reduced reproductive performance.

Grass, provided it is in good supply, should meet the requirements of the ewe for maintenance and some increase in body condition. However, in late lambing flocks in which mating takes place in late November and December, grass will need to be supplemented with concentrates and probably conserved forage also. Feed blocks and liquid molasses diets can be an alternative to concentrates and do not require to be replenished daily. The aim in these circumstances should be to have ewes in good condition 3 weeks pre-mating and then to provide a maintenance ration for them over the mating period. If conditions are very wet, windy and cold, maintenance needs will increase.

Post-mating

MATING TO 42 DAYS During pregnancy 20–25% of embryos/fetuses die and some of these deaths are caused by a combination of incorrect nutrition and handling and climatic stress, the latter especially in late mating flocks. The aim should be to feed ewes at maintenance level or just below. Both overfeeding, with ewes gaining in weight, or significant underfeeding will increase embryo and fetal losses. Above average losses may also be attributable to grazing low selenium content herbage and to grazing pasture containing large amounts of red clover. Alternative forage crops, such as kale and rape, should also be avoided.

Low cobalt-content herbage has also been found to have an important longer term effect by reducing the vigour of the lamb at birth and its resistance to disease.

Grass will meet the needs of the ewes for nutrients in the post-mating period during the early part of the mating season, but in areas where cobalt and selenium are known to be deficient in herbage, providing a small amount of concentrates containing enhanced amounts of these minerals may be beneficial. It should be noted that cobalt and selenium concentrations of herbage are usually at their lowest in late summer and autumn and are most likely to be below requirements in wet seasons.

PERIOD 42–90 DAYS The fetus grows slowly throughout this period but growth of fetal membranes and the placenta is considerable, especially during the later weeks. This results in an extra energy requirement above maintenance of approximately 2 MJ/day at 90 days. However, 1 kg of body weight loss will provide 20 MJ of metabolizable energy (ME), so the extra needs can easily be met by a small loss in ewe condition. Only severe underfeeding will reduce placental growth and hence lamb birthweight. Virtually no placental growth occurs after 90 days.

The aim should be to maintain CS in the 3–3.5 range for lowland ewes. Ewes which have been scanned to have three or more lambs should be carefully managed so that their CS does not decrease below 3–3.5, but in lowland ewes with twins and singles and in hill ewes carrying singles, some loss of condition (up to 0.5 CS) can be tolerated and will be desirable in ewes in CS 4 or above. In contrast, thin ewes and ewe lambs and shearling ewes which are still growing will need an above maintenance ration; the ME required for 1 kg live weight gain = 24 MJ. Grass will often not be abundant throughout the period and will have to be supplemented with conserved forage and, in some cases, concentrates or feed blocks. Medium to good quality hay or silage should easily meet the nutrient requirements of the ewe so, provided these forages are available, concentrate supplementation is unnecessary.

PERIOD 90 DAYS TO LAMBING During this period, fetal growth accelerates rapidly with the fetus doubling its size in the last 4 weeks of pregnancy. Mammary growth also occurs, especially in the final 2 weeks before lambing, when udder weight increases by more than 100%.

The net result of the above is an increasing requirement for nutrients for fetal and mammary tissue growth and development.

The appetite limitations of the ewe during this period impose considerable restrictions on ration formulation. The voluntary food intake is approximately 15 g/kg body weight (dry matter, DM) and is determined by a number of factors.

- In housed ewes, the intake of ewes which have been sheared is approximately 25% greater than those in full fleece.
- During this 8-week period, appetite increases slowly to a maximum at 2 weeks pre-partum. There is an equally slow decline until a few days before parturition when appetite decreases markedly.
- The digestibility of the forage, the amount of concentrates provided and the type of forage all influence intake.

Quality of forage not only indicates feeding value, but also the daily intake. Hay is very variable in quality and a chemical analysis of a representative sample is recommended.

Silage usually has higher ME and CP values than hay but DM intakes are lower (approximately 90%) than those of equivalent ME value of hay. Intakes increase with higher DM concentrations up to 250 g/kg DM. The intake of long material in a big bale will be 85% of precision-chopped material.

Straws may also be used as forage. Spring barley varieties are most suitable but all cereal straws can be used.

Chemical analysis of the whole straw is not a suitable means of assessing feeding quality because in most cases the ewes only eat the 25% of the straw which is most readily consumable and has the highest feeding value. The remainder should be discarded and used as bedding. Fresh straw should be provided daily either in a rack or on the floor. In the former system, uneaten straw should be removed from the rack daily.

Straw feeding value may be enhanced by treatment with a strong alkali or urea.

The body tissue of the ewe may be considered as a reservoir of nutrients which can be built up and depleted to a certain extent without adverse effects provided the depletion occurs slowly. An alternate 'flat-rate' feeding system has been devised which utilizes this fact. It is first necessary to calculate the total concentrate needs of the ewe over the period and then to provide the same daily amount throughout the period. Ewes gain weight at first and then, as lambing approaches, they lose condition. Practically, this is an easy-to-operate system and it also avoids the risk of metabolic acidosis occurring when ewes are fed large amounts of concentrates in late pregnancy. Lamb birthweights are similar to those obtained when ewes are fed on the traditional increasing plane of nutrition, but especially in young ewes, colostrum production may be somewhat reduced and there is increased possibility of hypocalcaemia. A single-step system therefore may be more appropriate with ewes fed at a first flat rate up to approximately 3 weeks pre-partum when the quality of the concentrate can be increased and then fed at a second increased flat rate until the end of pregnancy.

WATER REQUIREMENTS Housed pregnant ewes require a daily intake of 2.5 kg water in mid-pregnancy and up to 5 kg in late pregnancy.

Lactation

Colostrum production and consumption by the lamb is crucial. Sufficient colostrum should be available to meet the passive immunity requirements and also the energy demand in the first day of life. Output to 24 h post-partum will vary from 1.5 to 3.5 kg.

In early lactation, ewes suckling singles are likely to be producing a daily average yield of 2 kg or more, those suckling twins 3 kg or more and those suckling triplets 4 kg or more, provided they are well fed. Maximum daily yields occur at approximately 4 weeks post-partum and will vary from 2 to 5 kg. After 4 weeks, there is a slow, steady decline in yield but ewes will still be producing 1.5–2 kg at 10 weeks after lambing.

Nutrient requirements largely reflect the milk production of the ewe. The production of 1 kg of milk requires 7.1 MJ ME and 72 g of metabolizable protein. Formulation of rations to meet these requirements is modified by a number of factors including: (i) stage of lactation; (ii) body CS of the ewe; and (iii) foods which are available.

In the immediate post-partum period, ewes are hungry and careful rationing of concentrates to avoid gorging and consequent digestive upsets is necessary. Post-lambing DM intake increases steadily up to 5–7 weeks of lactation. Despite this increase in appetite, lactation demands usually outstrip the intake and ewes usually lose condition in the first month of lactation. A loss of one CS is not uncommon and is frequently a reflection of high milk yields. Growth rate(s) of the lamb(s) are a good indicator of milk yield in the early stages of lactation. Yields may be calculated from live-weight gain by assuming the DM content of ewes' milk is 200 g/kg and the conversion ratio of milk solids to live-weight gain is 1:1.

In practice, many ewes will be at grass throughout the lactation period. It is quite likely that grass will be limited at the beginning, which results in a considerable loss in body condition. Limitation is indicated by a sward length below 4 cm and ewes spending more than 9 h/day grazing. When sward length is below 2 cm, supplementation should be given to provide almost all nutrient needs (i.e. forage and concentrates). When the sward length is 2–3 cm, concentrates can provide approximately half of nutrient needs.

Concentrate feeding is also a vehicle by which magnesium can be made available to the ewe. An intake of 6 g/day of calcined magnesite, equivalent to 4 g of magnesium, should prevent hypomagnesaemia, which can be a problem in early spring when young grass high in protein is being grazed. High intakes of non-protein nitrogen and a rapid rate of passage through the gut both reduce magnesium absorption. Feeding of a high-energy concentrate food such as sugarbeet

pulp should be a considered option to minimize the problem. Providing ewes with shelter to reduce environmental stress is also beneficial.

Feeding the lamb

Providing the lamb with a supply of colostrum is essential. Ideally, the lamb will receive this from its dam within a few hours of birth and will continue to suck its dam and receive further amounts in the following 48 h.

Ewes also vary widely in production. In the first 24 h, the mean yield should be 3.0 l but some ewes will produce 500 ml and others more than 4 l.

For details of substitute colostrum preparations, see section on lamb survival (Chapter 7).

Most post-lambing mortality occurs in the first few days of life and the major cause is starvation. Insufficient colostrum or milk may be causative but the ewe not allowing the lamb to suck and lack of sucking drive and udder problems, such as blocked teats, may also be responsible. Starvation after the first few days is usually the result of poor feeding of the ewe in lactation, sore teats, mastitis or mismothering.

The lamb is dependent almost entirely on the ewe's milk until 4 or 5 weeks of age. Solid food consumption will begin at 3–4 weeks of age when the lamb starts to consume measurable amounts of grass and/or, if available, concentrate creep feed. This time is important as it is obviously when lambs commence to ingest significant numbers of parasite infective larvae.

Feeding the naturally reared lamb post-weaning

Most lambs will be weaned between 14 and 22 weeks of age. Some of these, especially single lambs, will be ready for slaughter at the time of weaning and will be finished off on grass alone or grass with a minimum concentrate supplementation in the autumn. The system depends on having available good quality grass such as aftermaths (the fresh grass growth after a crop of hay or silage has been taken off) and cow grazing. If such pastures are not available, performance figures will be disappointing partly because of low intakes. Intakes and performance will also be depressed if the grass is deficient in trace minerals such as cobalt. The post-weaning period is particularly problematical, especially if the grass is lush. In areas of cobalt deficiency, providing concentrates which contain minerals could paradoxically be more beneficial when there is lots of grass following a good rainfall in the late summer and early autumn than in drier conditions when less grass is available.

Many of the lambs, especially wether lambs from hill and uplands, will be managed so that they reach the required slaughter weight between Christmas and Easter. During the winter period, these lambs may graze green forage crops, turnips or sugarbeet tops. However, increasingly, they are being fed on silage with some concentrate supplementation. Growth rates of between <50 g/day and >200 g/day may be obtained, depending on the quantity and quality of the supplement provided. Farmers who rear store lambs have the option of finishing these store lambs early or late.

Grass silage if fed alone, even if of high DM and digestibility (D) values, will be unlikely to give growth rates in excess of 80 g/day. Part of the problem is that of low intakes which become more apparent: (i) if the DM is below 220 g/kg; (ii) if the pH is <4.0; and (iii) if the material is big bale rather than precision chopped. Maize silage has also been fed successfully.

Supplementing silages with 200 g concentrates/day will increase total intake and give growth rates between 120 and 150 g/day and 400 g concentrates will give growth rates of 175–225 g/day.

Shearing lambs in late summer and autumn usually results in higher food intakes and improved growth and carcass weight performance. Shearing may be particularly useful if the final finishing period is indoors because of nutritional and non-nutritional factors such as reduced space allowance and cleaner sheep being sent for market.

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Reproduction

Control of Breeding

Most breeds in the UK are seasonally anoestrus, although the Dorset Horn or polled Dorset and its crosses and some continental breeds will breed throughout most of the year. The Merino, which is a very important breed in countries such as Australia and South Africa, is not a strongly seasonal breeder so can be stimulated to breed at most times of the year. In seasonal breeders, the breeding cycle is controlled by decreasing daylight length and so ewes show peak oestrus activity in the autumn.

There are a number of methods which advance the breeding season or synchronize tupping and lambing.

Early breeding with progestogen sponges

Breeding may be advanced by up to 6 weeks by the use of progestogen sponges with injections of pregnant mare's serum gonadotrophin (PMSG).

Medroxyprogesterone acetate and flugestone acetate are different synthetic progesterone-like compounds that have been used by incorporation into sponges for intravaginal insertion. Currently, only flugestone is available in the UK. Both may be used with the injection of PMSG at sponge removal, which is essential in the seasonally anoestrus ewe. The farmer should be warned that individual ewes react differently to the same dose of PMSG and the results are unpredictable.

Table 2.1 provides an example of a schedule for early lambing with progestogen sponges (for normal conditions in the UK, although a similar schedule can be used at any time during the breeding season by substituting appropriate dates).

Discuss with the farmer when the finished lambs can be sold, and whether it is preferred that lambs are born before Christmas or after.

Roughly 70% of the ewes should lamb to the induced oestrus and most have twins. Results vary widely, according to: (i) breed; (ii) feeding; (iii) time of year; and (iv) weather. Test a batch of ewes and record the results in order to find out whether the procedure is worthwhile on a particular farm. The recommended dose of PMSG ranges from 300 to 750 IU at sponge withdrawal, with a higher dose in July than in August. Ewes and rams should be in good condition (CS 3) and fit. Put the rams near the ewes at sponge withdrawal, and put them with the ewes 48 h after withdrawal, for 48 h. If possible, remove ewes from the ram when they have been marked (raddled) convincingly, to avoid repeated

Table 2.1. Example of devising a schedule for early lambing (15–23 December), working backwards from desired lambing date.

Calculate	Activity	Date
Longest gestation 150 days[a]	Last lambing required	23 December
Shortest gestation 144 days[b]	First lambing anticipated	15 December
Last mating date	Remove rams	26 July
First mating date[c]	Introduce rams	24 July
Sponges in for 14 days	Remove sponges	22 July
	Insert sponges	8 July

[a]Counting 150 days back from 23 December the last mating date can be calculated (26 July).
[b]Counting 144 days back from 15 December the first mating date can be calculated (24 July).
[c]The sponges need to be removed 2 days before the rams are introduced (i.e. 22 July).

serving of favourites. Introducing rams at sponge withdrawal reduces the pregnancy rate drastically (68–40% in one trial). Two weeks after first oestrus, put rams, with a different raddle crayon, with the ewes for 1 week and then remove. The usual advice is to allow one ram for ten ewes, though it is possible with some individual rams to achieve good fertility with more ewes. There are considerable differences between individuals and between breeds in the ability of rams to breed outside the usual season.

Only well-managed farms can benefit from early, concentrated lambings. Housing and labour for lambings must be planned well or welfare is compromised and lamb mortality may be high.

Out-of-season breeding with progestogen sponges

It is generally agreed that in the UK disappointing results are obtained with most commercial breeds and halfbred ewes with tupping earlier than July though a successful attempt was made in 40 barren or aborted Welsh crossbred ewes in North Wales. Sponges were removed and 1000 IU PMSG injected on 25 May. Twenty ewes lambed, producing 30 live and seven dead lambs, and the lambs fetched a high price when sold for slaughter.

Similar results have been obtained with Mule (Bluefaced Leicester × Swaledale) ewes in June, with progestogen sponges and PMSG with 50% of ewes becoming pregnant.

Frequent breeding with ewes of aseasonal breeds

With selected breeds, it is possible to produce three crops of lambs every 2 years. In practice, few people have achieved this although one successful system produced lambs every 8 months from ewes mated in December, April and August with Finnish Landrace × Dorset Horn ewes and Down rams. Lamb sales approached 300% and profitability was three times higher than in annual lambing flocks.

A sophisticated but very practical system of frequent breeding has been described and developed over the past 30 years in the USA by Cornell University, New York known as the Cornell STAR accelerated lambing system. This system has been applied successfully by commercial prime lamb producers in the USA. Dorset and Finn sheep and their crosses are used and batches of ewes lamb every 2 months with individual ewes achieving five lambings in 3 years. No hormones or light regimes are used and the system results in a regular production of similar carcasses throughout the year. The system is well described on the Cornell University web site which includes an instructive and beautiful video and all details needed to implement a successful programme (www.sheep.cornell.edu).

Synchronization of oestrus during the normal breeding season with progestogen sponges

Sponges may be used in order to bring a batch of ewes into oestrus within a few hours

of each other, which may be useful to help produce a short lambing period, especially if used together with induction of lambing.

The sponges are removed after 12–14 days and rams introduced 48 h later. Almost all the ewes will be tupped over a 12-h period and the majority will hold to service, provided adequate numbers of rams are used.

Early breeding with vasectomized rams

A vasectomized ram run with ewes 1–2 months earlier than the usual start of the breeding season will stimulate a large proportion of the flock to come into oestrus about 3–4 weeks after his introduction. The ewes must be away from rams for 4–6 weeks before the teaser is introduced. This is also a cheap method of inducing synchronized oestrus. A vasectomized ram may also be used to withdraw individual ewes, as they are raddled, for service by a fertile ram, or for artificial insemination.

Early breeding with melatonin

Melatonin is secreted by the pineal gland in response to darkness and can be administered artificially by implant simulating short day-length, which stimulates the reproductive system of the ewe.

Melatonin is injected subcutaneously near the base of the ear with a special implanter. It can be used to advance the breeding season by 4–6 weeks so that for example, Suffolk-type ewes may be mated successfully at the end of June by giving an implant about 5 weeks earlier. It is vitally important that the ewes should be in good condition and that they should be isolated from sight, sound and smell of rams (and male goats) for 6 weeks before ram introduction.

Induction of lambing

Predictable lambing dates can be achieved by injecting 16 mg of betamethasone or dexamethasone at 140 days of gestation or later. This system has been used to batch lamb at times when labour is present, thus reducing lamb mortality.

As an example, in a trial at Liverpool University, 33 ewes were selected for injection with 16 mg betamethasone at 9.00 p.m. on day 142 of pregnancy (Saturday). Two ewes lambed before treatment, three within 24 h, three between 24 and 36 h, and 24 between 36 and 48 h (9.00 a.m.–9.00 p.m. Monday). Only one ewe lambed later, 55 h after treatment. It is clearly possible to give better attention to ewes, and to give attendants a rest, particularly avoiding the few ewes at the end of a group with long gestations. Side effects appear to be negligible, but if some ewes have been mated after the recorded service date (e.g. one cycle later) there is a serious risk of abortion.

Ram Examination

This is best done 2 months before tupping time as part of a flock health visit (Figs 2.1 and 2.2). About 10% of rams have poor fertility. Many of these can be detected by palpation of testicles without the need for electroejaculation but there are some conditions which are missed if examination of fresh semen is not carried out.

Examination of a ram lamb before use, or of a freshly-bought ram, is a wise investment. An infertile ram which is not detected can lead to ewes lambing 2 months late. If several rams of the same breed work together, an individual infertile ram may never be detected and may result in a reduction in overall fertility of the flock.

Any history based on good breeding records is valuable. Illness, however brief, can lead to temporary infertility up to and beyond 2 months later. If the ram is examined before entry to the flock, his conformation and any inherited condition should be checked. Teeth should make contact with the dental pad. Orf should be looked for around the lips. The CS should be 3 to 3.5. Feet should be checked; about 50% of rams were found to have severe foot lesions in one survey, which can drastically reduce fertility.

Fig. 2.1. Rams should be checked over 6–8 weeks before putting in with the ewes.

Fig. 2.2. These Texel rams (foreground) with a group of ewes are showing the Flehmen response (curling the upper lip) which helps detect ewes in oestrus.

Before the breeding season (before July in the UK), the size of testicles and numbers of spermatozoa are lower, especially in breeds with a short breeding season.

The testicles should be of similar size, very resilient (turgid) and move freely in the scrotum (compare with others of the same age and similar breed). The scrotal circumference ranges from 30 to 44 cm in mature lowland rams and from 30 to 40 cm in hill breeds. The scrotal circumference of ram lambs at 9 months old should be at least 28 cm in lowland and 26 cm in hill breeds. Testicular hypoplasia is incurable: no spermatozoa are produced, though libido is normal. The head and tail of the epididymis should be palpated for evidence of pain, lumps or adhesions since epididymitis is a common cause of reduced fertility in rams. A variety of organisms may be associated with epididymitis but *Brucella ovis* infection is of great importance in southern

European countries, Australia, New Zealand, South Africa and North and South America. Control schemes have been described where rams are tested and culled and this has been successful in eradicating the infection in the Falkland Islands and in some flocks in New Zealand and elsewhere. This infection is not present in the UK where it is a notifiable disease but other bacteria have been found in epididymitis cases including *Mannheimia* and *Histophilus*.

The penis can be extended with care, with the ram in the sitting position, to check for rare defects, by holding the prepuce and pushing forwards with the other hand at the sigmoid flexure, which is found at the base of the testicles. (Have a bit of practice so you can do it well!)

There are a number of battery-operated electroejaculators which are used to obtain semen samples from rams. The tip of the probe should be on the pubic brim, but there are marks to act as a guide to the depth of insertion in the average ram's rectum. The method has been criticized on welfare grounds and it seems preferable to avoid electroejaculation unless there are strong grounds for suspecting the ram is infertile in the absence of obvious physical abnormalities. In addition, it seems reasonable to give up if a sample is not obtained after three or four attempts and to try again later. The ram can stand or lie. The semen can be collected in a warm beaker or into a small transparent plastic bag. Between 0.5 and 2 ml of dense creamy semen is normally collected from a fertile ram. Semen obtained by electroejaculation is not necessarily typical of a natural ejaculate and some breed societies require examination by artificial vagina, which often needs training (for the ram!) or semen can be recovered from the vagina of a ewe just served by the suspect ram.

Semen examined immediately on a warm slide in a warm room should show good wave motion (as if being vigorously stirred) as seen in bull semen. If a poor sample is obtained, a second sample should normally be taken. After mixing one drop of semen on a warm slide with five drops of nigrosin-eosin (1.67 g eosin, 10.0 g nigrosin, 100 ml water) for 3 min, a smear can be made. Further evidence of the likely fertility is provided by: (i) the total number of spermatozoa; (ii) the number of live (unstained) spermatozoa; and (iii) the number of spermatozoa free of morphological abnormalities. Smears can be stained by Romanowsky methods as used for blood smears to detect the presence of polymorphs, suggesting an inflammatory response.

If the history and examination of the ram and semen show reduced fertility, treatment is not normally possible. A potentially valuable ram may be tested again 2 months later, as infertility is occasionally temporary. A report should always remind the client that the only final evidence of fertility is the production of lambs.

Rams should be permanently marked for identification and reports should be made on the special certificates designed by the British Veterinary Association (BVA) 'Certificate of Veterinary Examination of a Ram Intended for Breeding' which can be downloaded by BVA members or found on the SVS web site (www.sheepvetsoc.org.uk). This certificate also contains guidelines for semen collection by electroejaculation.

Urolithiasis

This is mainly a hazard for the housed 2–4-month-old castrated male lamb fed a lot of concentrate, although it does occasionally occur in adult males, particularly heavily-fed pedigree rams. The fine sand-like calculi, usually consisting of magnesium ammonium phosphate, obstruct the penis either at the vermiform appendage or at the sigmoid flexure.

Clinical signs

- The lambs show discomfort with straining ('hiccups'), kicking at the abdomen, twitching of the tail and general restlessness.
- A precipitate of crystals may be found on the preputial hairs which are often dry and sometimes bloodstained.

- 'Water belly' may occur, with urine leaking subcutaneously from a ruptured urethra, or into the abdominal cavity from a ruptured bladder.

Treatment (not easy, but urgent)

Assess the state of the animal as treatment differs depending on whether salvage or retention of breeding potential is the aim. Check whether the bladder is intact by ultrasound or paracentesis, and consider decompressing the bladder with a long needle through the abdominal wall. For valuable breeding animals, take blood for haematocrit, urea, creatinine and potassium, sodium and chloride concentrations.

Possibilities then are:

- Examine the vermiform appendage. If this is obstructed, cut it off at the base with a pair of clean scissors. If you are lucky and this is the only obstruction, urine will flow, but keep a close check as blockage may recur.
- Give a spasmolytic and analgesic injection.
- If the obstruction is higher and the animal is not required for breeding purposes, do a perineal urethrotomy under sacrococcygeal anaesthesia. If urine still does not flow, you can attempt retrograde flushing into the bladder, but catheterization is difficult because of urethral diverticulum.
- For non-breeding animals again, amputation of the penis can be carried out, exteriorizing the stump through the skin incision below the anus. This is probably the best method where urethral rupture has occurred.
- For breeding animals, either:
  - do a laparotomy, open the bladder and attempt to flush down the urethra. This is also an option if the bladder has already ruptured and repair is to be attempted; or
  - insert a Foley catheter into the bladder via the laparotomy and exteriorize the tube through the incision. The bladder can be flushed daily with Walpole's solution in an attempt to dissolve the stones. Flushing with a solution of soluble oxytetracycline, which is acidic, is worth considering.
- For all cases correct the fluid deficit and electrolyte balance with intravenous (IV) fluids.
- For all cases drench twice daily with 5g ammonium chloride or other urine acidifier, but not until metabolic acidosis has been corrected.

These cases are always a worry and the prognosis is always guarded.

Control

When cases are occurring

- Check the type and amount of concentrate being fed. Make sure it is formulated for feeding to breeding rams or fattening lambs and is not being fed in excessive amounts.
- Ensure plenty of fresh water is available.
- Supply salt (NaCl) as licks, or in the ration or in the drinking water.
- Add 2% ammonium chloride to the ration.
- Contact the food supplier to alert them to a possible problem, particularly if feeding instructions have been correctly followed.

In future

- Ensure no magnesium is added to concentrates; do not exceed 200g MgO/t.
- Ensure calcium:phosphorus ratio is at least 1.2:1 up to 2:1; include 1.5% ground limestone in the diet if necessary.
- Ensure there is a minimum of 1% salt in the concentrate.
- Ensure there is plenty of fresh water available.
- Avoid castration.

Artificial Insemination (AI) and Multiple Ovulation Embryo Transfer (MOET)

There have been considerable advances in the use of these techniques over the past

20 years and although ethical objections have been raised to the use of laparoscopic techniques, the stress to ewes when this technique is used by experienced veterinary surgeons is slight. Superior sires based on objective criteria are now available so the arguments for breed improvement are considerable and offset the minor surgical interference needed. The expansion of Sire Reference Groups (see later) has necessitated the use of AI with frozen diluted semen, which can only be done successfully by laparoscopic techniques. Commercial companies are now actively promoting AI, by cervical and laparoscopic techniques, and MOET is also available commercially and widely used in many countries. Throughout the UK, a wide range of breeding services including AI and MOET are available from Innovis (www.innovis.org.uk) and have been responsible for great advances in flock performance. Similar organizations exist in most sheep-producing countries.

AI with fresh semen

Synchronization of ewes with progestogen sponges and injection of PMSG at withdrawal is used, with AI 56 h after sponge withdrawal. Fresh semen can be placed in the vagina but better results are obtained by placing semen by pipette just into the cervix. One ejaculate will inseminate 20 ewes with a conception rate of around 70%. This procedure has been used for many years in large flocks in Eastern Europe.

AI with frozen semen

Conception rates with frozen ram semen given into the cervix are low (30%) and due to the tortuous nature of the cervical canal in ewes it is extremely difficult and often impossible to place the semen into the uterus, although attempts have been made to develop transcervical AI.

This is the reason for intrauterine insemination by laparoscopy, under local analgesia, with diluted frozen ram semen so that one ejaculate can inseminate 100 ewes with a conception rate of 60–70%.

Breed and Flock Improvement

Breed improvement has always been a feature of sheep farming from its earliest days in the development of different breeds suited to particular areas of the world. The naming of breeds of sheep according to the geographical area of origin such as Suffolk, Clun Forest and Swaledale indicates the selection which has taken place over centuries. The selection of rams and ewes according to breed standards was also an attempt to improve flocks though physical features were not necessarily associated with production criteria. In the past 40 years, recording of data on production, the recognition that much of this data is genetically inherited and the introduction of breeding techniques such as AI and MOET have given sheep breeders objective measurements to guide improvements and have made marked advances in flock performance.

In the UK, the Meat and Livestock Commission (MLC) introduced sheep recording schemes in the 1970s as its Sheepbreeder Services and these schemes provided an enormous stimulus to ram improvement and thus to flock genetic improvement. The traits identified have been chosen in order to effect the performance of sheep under UK conditions for meat production but similar methods have been used in Australia and other southern hemisphere countries where the main sheep product is wool. Twenty years ago a more ambitious ram evaluation scheme was introduced by Signet Breeding Services in the UK which offered best linear unbiased prediction (BLUP) technology to their recorded Sheepbreeder flocks. This system identifies superior rams for a number of criteria and the production of estimated breeding values (EBV) for each criterion and has now been extended to include ewes and lambs in pedigree flocks. The pedigree and performance data are analysed to estimate how much of a sheep's performance is due to its genetic makeup and how much to the flock environment. The standard performance traits

are: (i) litter size; (ii) maternal ability (milk production); (iii) lamb 8-week weight (a measure of milk production); (iv) a number of traits obtained by lamb examination at 21 weeks including weight and muscle and fat depth by ultrasound examination; and (v) mature size by weight at first lambing. More recently, computed tomography has been applied to selected animals to extend the number of traits to include: (i) carcass live weight/fat weight; (ii) carcass shape; and (iii) faecal egg count as a measure of genetic resistance to nematode worms. All these measurements are carried out on lambs at 21 weeks old.

An EBV is measured in the same units as the trait (e.g. 8-week weight in kg) and expressed as a positive or negative value compared with the average for the trait. A ram having a value of +5 kg for 8-week lamb weight, for example, means that on average, its progeny will be 2.5 kg better than those from a ram with a zero value, since only half its genes are transmitted. A recorded ram will have a value for each trait and in addition, different individual EBVs are combined to give breeding indexes to identify superior rams for the different stratifications in the UK sheep industry (see www.signetfbc.co.uk).

Similar schemes are used in other countries including Australia in which one EBV is for wool fibre diameter and in New Zealand, where a total of 94 individual EBVs are measured with around half for traits associated with wool and half for meat. Additional traits are likely to be included in future developments in sheep breeding as the genetics of production and disease factors are studied further.

Many breed societies now use Sire Referencing Schemes and EBV values to increase the rapidity with which genetic improvement can be used across flocks by identifying superior rams from the whole breed. In these schemes, AI is used on all farms in a Group Breeding Scheme with semen from the same rams so that genetic comparisons can be made even though the environment and husbandry of the farms differ. 'Nature' is thus separated from 'Nurture'!

It is also clear that commercial sheep farmers should purchase rams with known EBVs for traits that are needed to improve their own flocks and the financial indications are that the investment of around £600 per ram compared with £500 for an unknown ram will be repaid in extra revenue from lamb sales of around £3.00 per lamb sold.

3

Vaccination

A vaccination schedule should be in place as part of each flock health plan, based upon the particular disease risks within the flock. Always read the data sheet before using vaccines as instructions and protocols sometimes change (Fig. 3.1). At the time of writing, vaccines are available in the UK and many other sheep-producing countries for control of the following:

- clostridial diseases;
- pasteurellosis;
- combined clostridial diseases and pasteurellosis;
- enzootic abortion of ewes (EAE);
- toxoplasmosis;
- footrot (FR);
- erysipelas;
- louping-ill; and
- orf.

Other diseases for which vaccines can be used in special circumstances in the UK are caseous lymphadenitis, botulism and Johne's disease. In some countries, exotic diseases such as foot and mouth disease (FMD), sheep pox and *Brucella melitensis* may be controlled by vaccination.

Bluetongue vaccine was used in the UK while the threat of disease existed (see below for more information). See also under specific diseases in relevant chapters.

Clostridial Diseases

Clostridial diseases (see Table 3.1), other than tetanus and botulism, generally cause rapid collapse and death, therefore diagnosis and control are key issues. The diseases are an ever-present risk in virtually all flocks and should be controlled by vaccination. It is common for mistakes to be made in timing of vaccine administration or accidentally missing individuals or groups. It is also quite common to find farmers who do not routinely use these vaccines either on the grounds of cost (questionable as they are not expensive) or the mistaken belief that their farm is not subject to this group of diseases. Clostridial organisms are commonly found in the intestinal tract, are widespread in soil and form resistant, long-lasting spores, so it is never safe to assume freedom from these diseases.

Vaccination against clostridial diseases

There are several vaccines available ranging from comprehensive '8- or 10-in-1' to single component for blackleg. *Mannheimia* (*Pasteurella*) *haemolytica* and *Pasteurella trehalosi* antigens are also combined with clostridial in one of the commonly used vaccines. By using a multicomponent vaccine

Fig. 3.1. Vaccination should be carried out carefully and cleanly.

Table 3.1. Clostridial infections and associated diseases.

Disease	*Clostridium* sp.	Age affected	Season	Trigger factors
Enterotoxaemias				
Lamb dysentery[a]	*perfringens* type B	<2 weeks	Spring	Flush of milk
Struck[a]	*perfringens* type C	Growing lambs and adults	Spring	Flush of milk or grass
Pulpy kidney[a]	*perfringens* type D	>2 weeks	Any	Flush of milk, grass or concentrates
Braxy[a]	*septicum*	4–8 months	Autumn	Frosted food
Black disease[a]	*novyi* type B	Adults	Winter	Liver fluke
Bacilliary haemoglobinuria[a]	*novyi* type D	Adults	Winter	Liver fluke
Acute abomasitis[b]	*sordellii*	Growing lambs and adults	Spring	Intensive feeding
Gas gangrenes				
Blackleg[a]	*chauvoei*	Any	Any	Injury, wounds
Big head	*novyi* type A	Adults	Any	Fighting, wounds
Neurotropic				
Tetanus[a]	*tetani*	2 weeks–4 months	Spring	Docking, castrating
Botulism[c]	*botulinum*	Adults	Any	Toxin in food

[a]Included in 8-in-1 vaccines.
[b]*C. sordellii* is increasingly being implicated by laboratories as a cause of death; included in 10-in-1 vaccine.
[c]*C. botulinum* is rare in sheep and usually associated with chicken litter but is included for the sake of completeness.

and efficient primary and secondary doses, it is possible to maintain a protective level of immunity throughout the year in both ewes and lambs, against all the common clostridial diseases. Vaccines containing fewer antigens may be cheaper and are sometimes used for growing lambs, but it is arguably better to have a fully comprehensive insurance policy for these killing diseases.

Two doses of vaccine at a 4–6 week interval produce sufficient antibodies to protect a ewe for the first year with sufficient spare, via colostrum, to protect its lamb(s) for up to about 16 weeks, providing

the secondary or booster dose is given approximately 1 month before lambing (Fig. 3.2). In subsequent years, only one pre-lambing booster injection is required to protect both ewe and lamb(s).

Assuming a flock to have no initial protection, one effective vaccination schedule is as follows.

Ewes

- Primary as soon as possible.
- Secondary – 4–6 weeks later.
- Booster – 4–6 weeks before lambing.
- Repeat booster annually.

Lambs (ewe and ram)

Those to be kept over 16 weeks (for slaughter or breeding):

- Primary – at 10–12 weeks.
- Secondary – at 14–16 weeks.
- Booster (for breeding lambs only) with the adult ewes and rams pre-lambing time.

Bought-in ewes (for breeding) and store lambs (for slaughter)

Unless there is reliable vaccination information, it is best to assume that these have not been vaccinated, or at least that their immunity has waned and give them the full course of primary and secondary injections, starting as soon as possible after purchase. A short cut is often taken with the bought-in ewes (with some justification if there are no obvious winter clostridial risks, e.g. black disease) and that is to vaccinate them on arrival but delay the secondary injection until 4–6 weeks before lambing, but this strategy can be risky.

Bought-in rams

If their vaccination status is in doubt, give two doses at a 4–6 week interval, followed by an annual booster.

Lambs out of unvaccinated or inadequately vaccinated ewes

These lambs should either be vaccinated (primary in first week, secondary at 6 weeks) or rely on 200 ml of colostrum (fresh or frozen) taken from a vaccinated ewe and given at birth.

In an outbreak

Control measures in the rest of the flock consist of:

Fig. 3.2. These newborn lambs are born with no antibodies and are dependent on colostrum to acquire them.

- Remove sheep from the predisposing source (where possible), for example take off the lush grass or reduce the concentrates.
- Give an antibiotic injection (e.g. penicillin) for those with local infection, for example 'gas gangrene' and tetanus.
- Give antitoxin, if available, to all at risk, but in the UK this is now only available for tetanus. As the protection provided by antitoxin persists for only approximately 2 weeks, it is appropriate to inject a primary dose of vaccine at the same time (but at a different site), followed by the secondary dose of vaccine 4 weeks later, thus providing both immediate and long-term cover; the potency of the antigen overcomes any significant interference by circulating antibody.

Pasteurellosis

In flocks experiencing pasteurellosis, a combined vaccine containing clostridial and *Pasteurella* antigens is often used on the same schedule as for clostridial vaccination. A specific vaccine for pasteurellosis is available if, for any reason, the combined vaccine is not suitable. Note that passive antibodies to *Pasteurella/Mannheimia* spp. obtained by lambs from colostrum only protect for 2–4 weeks compared with up to 16 weeks for clostridial protection. If pneumonia is a problem in young lambs they can be vaccinated with a single vaccine from 3 weeks of age with a booster after 4 weeks.

Abortion

Effective vaccines are available to protect against EAE (caused by *Chlamydophila abortus*) and toxoplasmosis (*Toxoplasma gondii*).

Although there has been an effective dead vaccine to protect against EAE, this is not available at the time of writing. It had the advantage of being able to be administered to already pregnant ewes. The available vaccines are live and must be given at least 4 weeks before mating. Boosters are recommended a few years after the initial dose, but in practice most sheep are only vaccinated once.

The vaccine against toxoplasmosis is a live vaccine and should only be given to non-pregnant sheep at least 3 weeks before tupping. The EAE vaccine and that for toxoplasmosis can be given at the same time but from different syringes at different sites (e.g. on each side of the neck).

Footrot (FR)

One vaccine is available and should be used as part of a package of control measures when dealing with an infected flock. It is a dead vaccine containing ten strains of *Dichelobacter nodosus.* It is prepared in an oily adjuvant which can lead to lumps developing at the injection site. The vaccine can be curative as well as preventive, with a response usually noticed after a single dose. Boosters are needed, the frequency and timing depending on individual flock circumstances and identification of particular high-risk times for disease spread.

Monovalent vaccines have been used in some places where only a single strain of *D. nodosus* has been implicated, for example in Nepal. Research is ongoing looking at candidate antigens in order to develop a better vaccine for the future.

Erysipelas

In flocks experiencing problems of chronic arthritis of lambs shown to be caused by *Erysipelothrix rhusiopathiae,* or where post-dipping lameness is a recurrent problem, vaccination with an erysipelas vaccine is usually highly effective in preventing future problems. The vaccine is a dead one and requires two injections 3 weeks apart to produce immunity. Young lambs are protected through colostral antibodies so vaccination should be carried out on a similar schedule to clostridial vaccines, with pre-lambing boosters in succeeding years.

Louping-ill

This vaccine is obviously only necessary in tick areas where louping-ill is a known or potential problem. The vaccine is a dead one and requires a single injection repeated every 2 years. Lambs from vaccinated ewes are protected by colostral antibodies for 2–3 months.

Bluetongue

Bluetongue suddenly appeared in northern Europe in 2006 and continued in 2007 causing devastating losses in some sheep flocks. Cases appeared in the east of England in 2007. A vaccine against the specific serotype (8) was developed and was widely used in 2008 and 2009. This has resulted in no further disease in the UK and a huge reduction involving this serotype elsewhere. The disease remains active in countries around the Mediterranean and involves other serotypes which always pose a potential threat. The use of vaccine will now depend on the disease risk. The UK has now been declared free of the disease and vaccination is no longer allowed, however, should the disease recur, it is likely that vaccination will again be used.

Orf

Orf vaccine is different from all of the above, being applied to a scratch on the skin rather than being injected. It consists of live virus and should **not** be used in flocks which have no previous history of the disease. Care needs to be taken in its use. A scab forms at the vaccination site and this contains live virus and may be shed into the environment for up to 7 weeks. It is therefore important that contamination of lambing pens and fields is avoided by not putting recently vaccinated sheep into them. Ewes should not be vaccinated within 7 weeks of lambing. It can be used in lambs but risks transmitting infection to ewes' teats. As with all vaccines, read the data sheet thoroughly before recommending!

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4

Thin Sheep

Thin ewes are interesting and important for the following reasons:

- They are common which should encourage us to do something about them.
- The causes are not infinite; in fact, for most cases, the list is short:
  - lack of food;
  - inadequate teeth – particularly the cheek teeth; and
  - chronic disease, for example chronic lameness (joints rather than feet), chronic fasciolosis, chronic pneumonias, Johne's disease.

This should encourage us to diagnose them accurately.

- If there are many thin ewes, look first at the food and the number of fetuses or lambs; if there are a few thin ewes, also look at the 'fangs' (teeth) and the faeces; if there are only one or two thin ewes, also look at the limbs and the lungs.
- Thin ewes have suffered an insult for some time, which may imply irreversibility (i.e. they may not get better).
- Thin ewes are not 'worth' much, but deserve a lot because they represent a serious welfare problem.
- Thin ewes are generally unprofitable, for the following reasons:
  - If at tupping, their ovulation rates are reduced and therefore they produce fewer lambs.
  - If in late pregnancy, they produce smaller lambs and risk pregnancy toxaemia.
  - If at lambing, they produce less colostrum and show less interest in their lambs, which in turn are more susceptible to disease.
  - If in early lactation, they produce less milk leading to poor growth rate in their lambs.

Condition Scoring

One of the reasons for thin ewes being common is that the fleece masks their condition and it is only when the ewe is shorn or handled that the thinness is disclosed, so that the condition has continued for much longer than would have occurred in other species. Condition scoring (Fig. 4.1) was introduced to overcome this problem and is now an essential tool in good flock management as well as in the clinical examination of individual sick sheep. In some countries (e.g. New Zealand, Australia) body weight is an essential monitoring tool, but cannot be generally applied in the UK because of the

Fig. 4.1. Condition scoring by feeling muscle and fat cover over the loin is a vital technique.

large number of breeds and wide variation in mature body weight whereas a more standard breed type is found in these other countries. Condition scoring does not suffer from breed differences though it will be necessary to use different ideal target scores for different breeds, particularly between mountain and lowland sheep.

The crucial element in the technique is to decide by feeling the mid-lumbar region, whether the ewe is 'too thin' or 'too fat' or just about right, so that action follows, for example, by separating and supplying extra, or occasionally less, food (see Fig. 4.2).

A number score (1 to 5, where 1 is emaciated and 5 is so fat you can't feel anything else) is given and it needs practice across the full range of scores and standardizing with other scorers from time to time. Each time of condition scoring usually requires doing a few 'to get your hand in', but it is a technique that is learnt quickly and needs no kit, only one sensitive hand! Half scores are useful as most people find the distinction between full

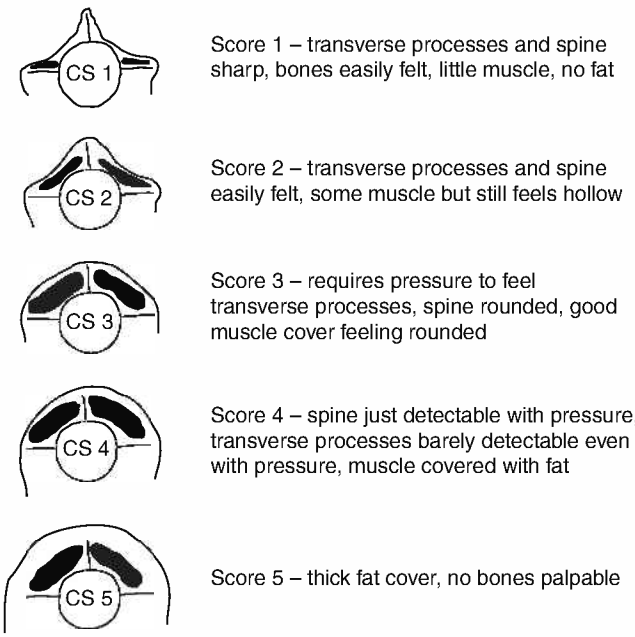

Fig. 4.2. Condition scoring: descriptions of the mid-lumbar region denoting condition score (CS) 1 to 5.

scores too large for decisiveness. It is usually possible to teach the technique rapidly with agreement within half a score.

Two vital times when farmers should use the technique of condition scoring their flock are 6–8 weeks pre-tupping and 6–8 weeks pre-lambing, because then there is time to alter events by, for instance, reallocating resources such as food, housing and shepherding. Repeat scoring in about 4 weeks is advisable because at least it will suggest what the future holds, although it may be too late by then to alter some of the events, particularly late in pregnancy when the demands by the lambs are high. Lowland ewes with a CS of less than 3 (hill 2.5) should be separated and clinically checked. Most will require more, perhaps better food, while some will require treatment or even culling.

Assessing a Feed Ration for Adequacy – a Practical Problem

One of the simple nutritional problems which the practising veterinary surgeon can carry out is to decide if a group of sheep are thin because of inadequate feed or if some specific disease is involved. The following steps illustrate how to investigate the problem by an examination of what is being fed to the thin ewes. It should be emphasized that it is a simplification but will indicate if there is a gross inbalance in the requirements and the intake of the ewes. It has been used by student groups and by us over many years and usually works!

Is thinness in a group of ewes due to feed or is something else responsible?

The most common problem is when a group of pregnant ewes are housed and a significant number of them are in poor condition.

Work on metabolizable energy (ME) only

STEP 1. Assume the following approximate ME requirements of a 70kg ewe, which is around the average weight of a lowland ewe:

Maintenance	10 MJ
At lambing with two/three lambs	20 MJ
Full lactation	30 MJ

These figures are easy to remember and any stage of gestation can be obtained by linear extrapolation between maintenance at 80 days and lambing at 145 days (e.g. day 115 is approximately 15MJ).

If the ewes are significantly different in weight from this, maintenance ME can be obtained by the formula:

$$ME = 0.4(\text{body weight})^{0.75}$$

For example for a 70kg ewe ME = 0.4×24.2 = 9.7MJ (rounded off above to 10MJ).

Does the ration supply these requirements?

STEP 2. Examine the concentrate consumption:
The farmer may know how many kilograms are fed daily or may state, for example, that 'six bucketsful are given to the group'. Weigh a bucketful and calculate how many kilograms are fed to the group. Count the number of ewes and let the quantity fed **per ewe** be 'c'.

1. Check the ME of the concentrate.
This may be: (i) obtained by asking the manufacturer; (ii) calculated from formulae; or (iii) assumed to be about 12.4MJ/kg DM. This assumption is risky since some feeds (usually cheap) may be as low as 11.0MJ/kg DM. Take great care to distinguish between values given by manufacturers for air-dry or oven-dry material. It is assumed that the value obtained is for oven-dry material (i.e. true DM).
2. Remember that the megajoule in (**1**) will be per kilogram DM. Since concentrates have a DM of about 85%, the DM intake will be 0.85c.
3. Calculate the mean intake of each ewe from the concentrate:

ME intake concentrate = 0.85c × MJ
(e.g. 0.85c × 12.4 = 10.5c)

STEP 3. Examine the forage consumption:
The farmer may state that this is given *ad libitum* which should mean that there is always some available for the ewes (i.e. hay in the rack all day or self-feed silage). If this is not

true when you visit the farm, the quantity fed must be measured by weighing a bale and asking how many bales are fed per day. If the forage is truly available *ad libitum*, work on forage being consumed to appetite. (This is a problem in the calculation since so many factors influence food intake but a check is included later – see STEP 5).

(In the example given below it is assumed that the ewes are being given hay, but the calculation for other forages such as silage is identical. It is probably even more important to have the forage analysed as there are greater variations than for hay. The DM content of hay is around 85–90% whereas that for silage is 25–30% so a much greater weight of silage must be eaten to provide the same energy. It should be remembered that care must be taken with silage production to reduce the risk of listeriosis.)

1. Calculate forage consumed.
Assume a 70 kg ewe has an intake of approximately 1.5 kg DM, which is reasonable if they are fed medium or good quality hay. If the hay is poor, with a ME of 8, the intake will be less, around 1.25 kg DM.

DM forage consumed
= 1.5 – concentrate consumed
= 1.5 – 0.85c

2. Calculate the ME of the forage.
This can be obtained by: (i) analysis; (ii) calculation; or (iii) by estimating the quality and assuming that poor, medium and good quality hay will have values of approximately 8, 9 and 10 MJ/kg DM, respectively. The values for silage are higher than for hay and the corresponding values are 9.5, 10.6 and 11.7, respectively.

ME intake forage = (1.5 – 0.85c)
× ME forage
For example with medium quality hay
= (1.5 – 0.85c) × 9

STEP 4. Calculate the total ME intake:

Total ME intake = ME from concentrate
plus ME from forage
= 0.85c × ME concentrate
+ (1.5 – 0.85c)
× ME forage

For example:
Total ME intake = 0.85c × 12.4
+ (1.5 – 0.85c) × 9

STEP 5. Look at modifying factors on the farm:
These calculations have made a number of assumptions, some of which can be checked on the farm.

1. Check that your calculation of forage agrees roughly with what is fed (i.e. weigh and count bales).
2. Allow for wastage of forage if this is not fed *ad libitum*, which will depend on its quality. Look at the bedding and see how much hay can be seen and observe the ewes eating and see how much they reject. As an estimate, wastage of good hay will be about 5% and can be ignored but poor hay may have wastage of 20%. Make an allowance for this in your calculations unless the hay is truly *ad libitum*.
3. Look at the trough space which should be at least 45 cm for each ewe in the group. Watch the ewes when they are given concentrates and see if many are forced to snatch small quantities and keep moving. Each ewe should be able to stand fairly quietly and eat its share. Watch for bullying which may give rise to a small number of thin (bullied) ewes with the rest normal or overfat.
4. Ask if there are any ewes with particular requirements. We have assumed that the ewes are uniform but different lamb numbers, initial condition, etc. may need to be taken into consideration.

A calculated example

A 70 kg Mule ewe at 115 days of gestation given hay *ad libitum* with an analysis of 9.0 MJ/kg DM and 0.25 kg of a concentrate with ME of 12.4 MJ/kg DM. Is this diet adequate to maintain body condition?

Requirement = approximately 15 MJ

Intake = 0.85 × 0.25 × 12.4 + (1.5 – 0.85
× 0.25) × 9 (from STEP 4 above)

Intake = 14.2, which is just less than requirement, so some loss in condition will result and it would be preferable to increase the concentrate fed slightly.

Teeth

It is very important to be able to examine sheep teeth properly, not only for ageing (see Fig. 4.3 and Table 4.1) but also because faulty teeth are one of the most common causes for adult sheep to be thin (Fig. 4.4).

With regards to teeth the following points are worth noting as there is a lot of variation within and between flocks, and some confusing features:

- Eight worn permanent incisors can look like eight temporaries and vice versa, especially in mountain breeds such as Welsh Mountain. Generally, the temporaries are smaller, more triangular and without a table (i.e. a flat surface). However, since this means that a yearling is being compared with an old ewe, it is generally possible to tell the difference by general observation so common sense is needed rather than unthinking application of the data shown in Table 4.1 and Fig. 4.3.
- The permanent corner incisor is usually less obvious than the other permanent incisors (it sometimes never erupts). It can look like a temporary tooth, making the sheep appear like a 3-year-old (six teeth).
- In rapidly growing lowland breeds eruption of incisors can take place

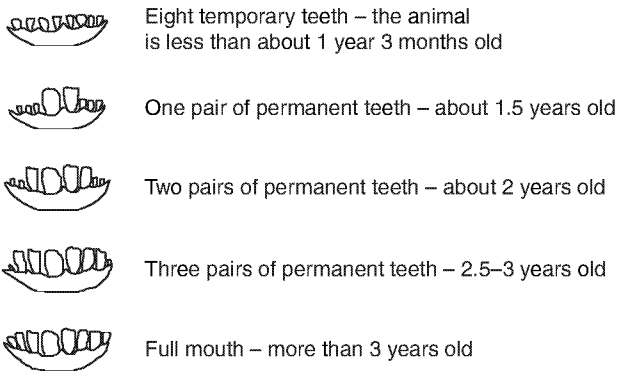

Fig. 4.3. Teeth and ageing.

Table 4.1. Ageing – a rough-and-ready guide (see points worth noting in text).

Age (years)	Incisors	Cheek teeth (one side)
Up to 1 (lamb, hogg)	Four pairs (eight teeth) temporary	Three premolars – temporary, two molars – permanent
1–2 (yearling, shearling)	One pair permanent (two teeth), three pairs temporary	Three premolars – permanent, three molars – permanent
2–3	Two pairs permanent (four teeth), two pairs temporary	As above
3–4	Three pairs permanent (six teeth), one pair temporary	As above
Rising 4 and over (sometimes to a grand old age)	Four pairs permanent (full mouth)	As above
Old sheep	Less than four pairs permanent (broken mouth)	May lose some premolars and/or molars

Fig. 4.4. Examining the incisor teeth gives a good indication of age.

earlier than indicated in Table 4.1. The first set of permanent incisors can be present by 15 months, the second set by 21 months, the third set by 2 years 3 months and a full mouth by less than 3 years.

- Age the sheep in relation to the time of year and the normal time of lambing (e.g. in the autumn in the UK, most sheep will be X years plus or minus 6 months, while in the spring they will be just X years, since most ewes are born in March).
- Periodontal disease causes premature incisor and molar tooth loss and sheep then look older than they really are.
- The first and second molars (fourth and fifth cheek teeth) are present early in the lamb's life and a year or more before the temporary premolars are shed and the permanent premolars erupt. The region between the third premolar and the first molar is, therefore, a very susceptible site for food impaction and progressive disease, starting in the first year or so of life. It is almost certainly the reason why the mid-ramus is the area most prominently affected (lump and sinus) in periodontal disease of the cheek teeth which is the area where careful examination should be carried out.

Teeth problems

Incisors

Very occasionally, faulty eruption occurs and a dentigerous cyst may result which ultimately causes considerable bony deformity and may cause premature culling though it is surprising how efficiently such sheep maintain condition. Malposition in relation to the dental pad (over or undershot) is quite common but it is probably not significant unless extreme. It has low heritability, and the selection for good apposition (bite or occlusion) is unreliable before 3 years old. Premature loss of incisors (broken mouth) is extremely common and is connected with periodontal disease.

To some extent, tooth eruption always causes a local 'itis', but in many sheep there is a serious progressive gingivitis leading to gum recession early in life, and subsequent loosening of teeth following subgingival plaque formation and periodontitis. Osteomyelitis, abscessation and sinus formation are natural consequences of the initial gum pathology. The reason for such serious progression is uncertain; the role of some oral bacteria has been investigated and there is a strong suspicion that gritty food may be involved, which may help to explain why the prevalence of the disease is

much higher on some farms than others, where the soil types and grazing environments differ. Sometimes, there is a family or breed prevalence within a flock, suggesting a genetic component.

Premature loss of incisors in hill and upland sheep reduces the ability of ewes to maintain body condition and to feed their lambs well, and so often results in premature culling. Its effect on lowland sheep is not so serious providing the cheek teeth are not involved, and therein lies the problem. Broken-mouthed sheep should always have their cheek teeth checked, because incisor loss often means molar loss, although occasionally the incisors may appear healthy while the cheek teeth are not (and vice versa). So the customary reliance on incisor examination alone (ageing) can be very misleading and all thin sheep need their cheek teeth examining by external examination of the jaws. Studies have shown a poor correlation between incisor loss and body condition but a good correlation between molar tooth disease and condition.

Cheek teeth

First examine from outside (most problems can be detected by this method, but a more detailed examination can be done if considered really necessary – see below).

- Note CS, dribbling, staining of lips and mouth, mouthing (when trying to eat) and quidding (swelling of cheeks with wads of food and spilt quids and cuds on the ground). Observe sheep chewing the cud and regularity of movement of lower jaw.
- Feel along the outside of the cheek for evidence of pain (flinching on pressure) and for irregularity of teeth (shear mouth and wave mouth).
- Feel with finger and thumb along the two rami for bony swellings and missing teeth.
- Compare the thickness of the rami with each other, but also with a normal young animal – bilateral thickening may be missed if you are unfamiliar with the normal, though it is usual for only one side of the mandible to be involved which allows comparison between them.
- Note whether there are discharging maxillary or mandibular sinuses.
- Note abdominal distension for ruminal 'impaction' and/or pregnancy (likely to develop pregnancy toxaemia).

Now look inside if you really need to, but remember sheep cannot open their jaws widely and it may cause, at the least, discomfort to force them apart. Make sure the animal is facing the light (and have a good torch) and also have a good gag (e.g. block of bevelled hard wood 12 × 2 × 2 cm or a sheep gag). With the gag in place, note any gaps (particularly common in the lower jaw), irregularities, loose teeth, food impaction and spikes. Look particularly in the mid-third region (check teeth three and four): if there are gaps, the corresponding upper (or lower) tooth lengthens through lack of wear, making it even more difficult and painful for the sheep to chew properly.

- Be very careful in inserting a finger since the edges of the teeth are usually very sharp and it is easy to sustain a painful cut.
- We have found increasingly that we can determine the presence of molar tooth problems by external palpation and rarely resort to the use of a gag to examine inside the jaw.

Treatment

- Assess whether it is worth doing anything other than cull. Some action usually must be taken on grounds of welfare as well as economics, but dentistry provides only temporary relief and may only be indicated in late pregnancy in order to allow the ewe to produce valuable lambs which will probably have to be reared apart from the ewe.
- Loose incisors are often best removed rather than left and can usually be removed without anaesthetic. Remove obvious loose molar teeth under sedation or light anaesthesia and inject long-acting (LA) antibiotic twice at

4-day intervals. Ewes can thrive, given a chance, without any incisors ('gummers') providing their cheek teeth are healthy. Molar rasping may have short-term advantage, but is very difficult to do effectively.

- Feed high quality concentrates and roughage that are comfortable to eat and separate from greedy competitors. Proprietary nuts are usually too hard. A useful mix is 40% whole barley, 40% sugarbeet pulp, 17% soya and 3% minerals.

Control

Check feeding and breeding. A fault in Ca/P, vitamin D, copper and fluorine levels in the feed should be considered, as well as generally inadequate feeding which may lead to excessive soil eating (which compounds the problems of mineral deficiencies and gum irritation). The prevalence in a flock may suggest genetic factors, in which case the ram(s) or even the breed needs changing. Feeding blocks and roots may not promote incisor loss, but certainly require healthy teeth.

Fasciolosis

Fasciolosis, most importantly *Fasciola hepatica* (liver fluke) infection, is of immense economic importance in sheep throughout the UK and most sheep-producing countries of the world. Some years ago, it was only a major problem in the wetter west of the UK but it is now much more widespread perhaps due to climate change. No effective immunity develops in sheep, unlike cattle, and thus disease can occur in any age of sheep. A high proportion of the flock is usually affected in epidemic years. *F. hepatica* has a wide host range including humans and wild mammals which makes control difficult where mixed host species occur on farms.

Clinical disease is associated with the ecology of a mud snail of the genus *Lymnaea* (*Galba*) often with different species in different countries though all have similar habitats, so that work on one species can be extrapolated to others and rational control principles suggested. In the UK, the snail is *Lymnaea* (*Galba*) *truncatula.* All these snails are small (a maximum of 10mm though much smaller snails can support the development of the fluke) and are found in muddy areas at the edge of streams and ditches. When searching for the snails, it is often useful to look for dried mud on the snail shell which means that it stands out on the muddy habitat. The development of the stages in the snail is dependent on a minimum temperature of 10°C and moist habitats which allows the important times for the appearance of disease to be forecast from meteorological data, even if the country has never been visited or studied! The epidemiology of the disease is dependent on wet conditions for a cumulative period of 12 weeks during the months when the temperature is above 10°C. This association allowed a forecast to be made dependent on meteorological conditions and resulted in very effective control schemes being devised. In the UK, this invaluable forecast is still produced in the National Animal Disease Information Service (NADIS), available online at www.nadis.org.uk.

In summary, acute disease, associated with migrating immature flukes, is most commonly seen from October to February after a wet summer, and chronic fasciolosis, in which adults are found in the bile ducts, is seen from December to April. A subacute form of the disease has been described as occurring in December and January when a mixture of adult and immature flukes will be present. The best way to decide if a farm has liver fluke is to take faeces from between ten and 20 untreated thin ewes in December to May and look for eggs, though eggs are likely to be present all the year round in untreated ewes.

Acute fasciolosis

Sudden death may occur in sheep of all ages in epidemic years and the mortality can be very high. Affected animals may be seen with extreme weakness, severe anaemia (packed cell volume (PCV) of 10%), abdominal pain and ascites. The liver is enlarged with haemorrhagic tracts. Eggs are not found in the faeces and there are usually over 1000 immature (0.5 cm long) flukes in the

substance of the liver. Acute disease is seen about 6–8 weeks after the ingestion of a large number of metacercariae.

The flock should be treated (triclabendazole is effective against all stages of the parasite from 2 days old onwards unless resistance has developed, which is not common as yet) and moved to a clean pasture, wherever possible. The metacercariae will remain viable for several months, especially over the winter, so do not return sheep to the pasture containing an extensive snail habitat before the end of May.

Chronic fasciolosis

Affected animals show a progressive loss in condition proceeding to emaciation, and signs of anaemia will be present, since the PCV is usually about 15%. Mucous membranes are pale and the nictitating membrane is pale and oedematous ('flukey-eye'). Ascites often occurs but sub-mandibular oedema ('bottle-jaw'), often described as being typical of chronic fasciolosis, is uncommon and does occur in other clinical conditions. Wool yield is reduced even in light infections, as is the milk production of ewes, thus resulting in reduced weight gain in the lambs. The liver is small and cirrhotic, the bile ducts enlarged and thickened. There are usually about 250 adult flukes present and eggs can be found in the faeces. The prepatent period is about 10 weeks but chronic fasciolosis usually occurs some weeks after patency.

Control

This is based upon strategic anthelmintic use and knowledge of the environmental conditions which are needed to allow the development of the fluke from egg to infective metacercaria, on the ground and in the snail, *L.* (*G.*) *truncatula*. This mud snail measures up to 10 mm in length and the operculum is on the right side when held upright. There are, however, a number of rather similar snails which can easily be confused with *L. truncatula*.

In the UK, the most important cycle of infection involves the 'summer' infection of snails which results in the main pasture infection with metacercariae occurring in September and October. The 'winter' infection of snails occurs when snails become infected in October of one year and the life cycle is complete in May and June of the next year. This can give rise to acute fasciolosis in July and August rather than in the autumn. It is a rare occurrence in the UK because May is usually a dry month and the over-wintered snails commence to die off at this time.

Use of anthelmintics

Modern anthelmintics act against immature stages as well as against adults. Some drugs, such as triclabendazole are effective against the very early stages (from 2 days old), others against somewhat older, immature flukes (rafoxanide, nitroxynil, closantel from 4 to 6 weeks old) while some drugs are only effective against adults (oxyclozanide, albendazole). Closantel also prevents egg laying by adult flukes for over 10 weeks. Triclabendazole is the drug of choice in the treatment of acute fasciolosis. Drugs are a very important part of any control programme, the aim of which is to prevent fluke eggs being passed by the sheep (or cattle) so that the infection rate of snails is greatly reduced. Field trials with rafoxanide have shown that if all adult sheep are treated in mid-April, followed by a second treatment 6 weeks later, the summer infection is reduced to a very low level. Further treatment in October and January reduced fluke burdens over a 3-year period to about 10% of pre-trial numbers, and resulted in considerable increase in wool yield and lamb productivity (the extra revenue from fleece sales was sufficient to pay for the anthelmintic treatments). This work was done in an area where fluke was widespread and habitats extensive, and in areas of lighter infection it would probably be sufficient to treat in April, October and January. Good results have been obtained on a high-risk farm by the treatment of all sheep with triclabendazole in January or February and then every 3 months. Although snail numbers increased, the number of infected snails was very low and the prevalence of infection in the sheep reduced by about 75%.

These months are based on UK conditions but it is easy to extrapolate to conditions in other countries including those in the southern hemisphere by consulting local meteorological information.

Although anthelmintic resistance to *F. hepatica* is not as common or widespread as to intestinal nematodes, flukes resistant to triclabendazole have been described in sheep in many parts of the world including the UK and Australia. Field studies on sheep harbouring the resistant adult flukes showed that they were susceptible to closantel, nitroxynil, albendazole and oxyclozanide; younger resistant flukes are probably susceptible to rafoxanide and nitroxynil from 4 to 6 weeks old as above.

Short-term control

The publication, *UK VET Livestock* has a regular section called 'Parasite update and forecast' which includes a liver fluke forecast. This is extremely valuable and can be used to avoid acute fasciolosis in epidemic years by preventing sheep from ingesting large numbers of cercariae from September onwards by:

- removal of sheep to a pasture with no snail habitats; or
- fencing off small habitats.

Sheep should be kept away from habitats until May of the following year since metacercariae are long lived during winter months. If sheep must be grazed on habitats during the winter in epidemic years since no other grazing is available, anthelmintics which are effective against very young flukes given at 4–6 week intervals may help in preventing serious disease. This is not very satisfactory however, and it is preferable to use control measures which prevent such heavy pasture contamination.

Long-term control

This used to be based on improved drainage and even use of molluscicides (it is now many years since use of these was contemplated) but current support for wildlife habitat improvement means that farmers are more likely to be encouraged to keep boggy ground rather than drain it! Improvement of hill grazings with basic slag and lime also no longer takes place. This increased the pH and allowed previously unsuitable land to become very suitable for snail multiplication. Severe outbreaks of fasciolosis were seen on these improved pastures where previously the acid pH of the peat prevented extensive habitats.

Control therefore has to rely on:

- fencing off habitats if feasible; and
- prophylactic use of anthelmintics to reduce pasture contamination with eggs so that the proportion of infected snails is reduced.

On many farms where habitats are few and small, eradication is possible by a logical combination of these principles.

A survey of snail habitats is essential before any of these control schemes can be developed. This may be done by the veterinary surgeon with a large-scale map of the fields and an experienced eye for suitable snail habitats.

Johne's Disease (Paratuberculosis)

Johne's disease is found in older sheep in most sheep-producing countries of the world. However, it is not diagnosed very often but it may be much more common than we realize since diagnosis is difficult and there are many sheep which harbour the causative organism *Mycobacterium avium* subspecies *paratuberculosis* (MAP; synonym *Mycobacterium johnei*). It needs to be on one's list of causes of thinness in middle-aged ewes. However, it can become established in a closed flock, and a 10% incidence has been recorded in some flocks with an even higher prevalence in some abattoir surveys.

Diagnosis

The typical story is of a few dirty-tailed and broken-woolled ewes of about 3 years of age

which are unaccountably thin (feed and teeth okay), often deteriorating soon after lambing. The disease is similar to that in cattle but there are a number of specific features:

- Scouring is not so obvious, often being intermittent with only soft faeces.
- As with most debilitating diseases of sheep, the fleece pulls out easily (wool break).
- As well as the common ovine strain, there is also a yellow pigmented strain which occurs in some restricted geographical areas and can be seen at necropsy, staining the intestine.
- The organisms are not easily found in the faeces though it may be worthwhile staining a faecal film for the acid-fast bacteria by the Ziehl-Neelsen technique but no conclusion should be drawn from a negative result. A necropsy with gut histology is usually required to establish the diagnosis in a flock and thereafter it is often assumed that thin ewes are caused by MAP.
- Individual complement-fixation tests are unreliable and are really only for use as a flock test. Similarly, an enzyme-linked immunosorbent assay (ELISA) is available though cannot be relied on.
- Since the disease is a protein-losing enteropathy, it has been suggested that the very low serum albumin concentration seen in Johne's disease in sheep (mean of 14.1 g/l cf. normal 32.7 g/l), together with a normal globulin concentration, may be useful as a screening diagnostic test.
- Many different strains of MAP are known in a wide range of hosts including cattle, deer and rabbits and the organism is found in Crohn's disease in humans, giving rise to fears that this disease is associated with poor pasteurization of milk. There is still considerable debate as to the relationship between these strains.

Control

- Cull clinical cases and fatten their lambs for slaughter rather than keeping them for breeding. Infection occurs by ingestion of MAP by young lambs with clinical signs developing 2–3 years later.
- Vaccination – live, attenuated vaccines are used in many countries and can be imported specially into the UK with permission from the Veterinary Medicines Directorate (VMD). Experience has shown that injection of lambs at about 3 months of age increases their resistance to the disease, slows shedding of bacteria and reduces clinical disease. It does not prevent infection.
- Ensure good control of other debilitating conditions (e.g. lack of food, worms, fluke and cobalt) which appear to predispose to the development of the clinical disease and the shedding of large numbers of bacteria.
- Think carefully about the use of surplus colostrum in flocks which keep lambs for future breeding. Infection can be spread through this route, therefore in problem flocks use of commercial substitutes may be a wiser choice.

Chronic Respiratory Diseases

The two slow virus diseases affecting the lungs, maedi visna (MV) and ovine pulmonary adenocarcinoma (OPA otherwise known as sheep pulmonary adenomatosis, SPA, or Jaagsiekte), can both lead to an increase in the number of thin older (young adult) sheep, and be the cause of premature culling (see Chapter 13).

Caseous Lymphadenitis (CLA)

This chronic bacterial disease can also sometimes cause wasting in older animals but is more often seen as abscesses in external lymph nodes (see Chapter 12).

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5

The Pregnant Ewe

Ultrasound Scanning – Diagnosis of Pregnancy and Number of Lambs

Ultrasound scanning of pregnant ewes has become a widespread valuable technique in sheep husbandry throughout the world. In many situations, it is sufficient to know if the ewe is pregnant or not but the technique has the additional value in flocks with high lambing percentages, to distinguish the number of fetuses. Ewes can then be marked and separated and fed appropriately for their fetal burden.

Commercial operators and veterinary surgeons have provided ultrasound services in Australia, New Zealand and the UK and in many other countries. Most operators offer a service which includes the determination of fetal numbers.

It is important that the operator scans a large number of ewes regularly to maintain expertise in detecting fetal numbers and accuracy will then be over 98%. An experienced operator can scan 80–120 ewes/h. Several veterinary practices with a large number of sheep clients operate scanning services which help in the introduction of sheep health programmes on to the farms.

The value of the technique is that: (i) barren ewes can be sold when prices are high or fed less; (ii) ewes with single lambs can be fed less, saving feed and avoiding over-large lambs; and (iii) ewes with two or more lambs can be fed more concentrates which should reduce the possibility of loss of condition and pregnancy toxaemia (PT).

The ewes are scanned in the standing position without shearing and purpose-built handling systems allow large numbers of ewes to be scanned in a day. For accurate diagnosis of fetal numbers, the fetuses should not be less than 50 days and not more than 100 days gestation. Allowing for a 28-day period with tups in the flock, this means that the flock should be scanned between 80 and 100 days from the time that the tups were turned out with the flock.

The main alternative is regular condition scoring and transfer of thin ewes to groups fed more concentrates. This avoids the need for pregnancy diagnosis, but requires regular examination by good shepherds during late pregnancy.

Housing

The practice of housing pregnant ewes during the last 2 months of pregnancy or in wet, cold climates for longer periods has become adopted in the UK and many northern European countries and in other countries such as the USA (Figs 5.1 and 5.2).

Fig. 5.1. Heavily pregnant sheep are often housed for lambing.

Fig. 5.2. This shed holds a large flock of pregnant ewes divided into suitable groups.

Advantages

- Housing prevents damage to the pasture by the sheep, which otherwise poach land during the wet winter and nibble young grass shoots in February and March. This is particularly important on wet, heavy land where removing the sheep in winter results in good grass growth in April when ewes are lactating heavily. This strategy allows an increase in stocking rate which is necessary if

the costs are to be covered. Housing usually commences in December/January and sheep are turned out near the end of March, depending on weather.

- Housing improves the working conditions of the shepherd and protects sheep (and newborn lambs) from extreme weather conditions. Ewes are more carefully observed and shepherded during lambing which reduces lambing losses.
- Housing allows ewes to be sorted into different batches according to CS, number of lambs being carried (if ultrasound pregnancy detection is used), state of teeth, lambing dates, etc. The feeding of the ewes is more controllable and regular condition scoring allows appropriate adjustment to feeding to be made.
- Housing reduces the wastage of food, both forage and concentrates, if well-designed racks and troughs are used, whereas considerable wastage occurs in adverse weather if fed outside.
- Housing allows the winter shearing of ewes.

Disadvantages

- Capital costs: these depend on the type of building and whether existing buildings can be modified. The building does not need to be of complex design, the cheapest being the 'polythene tunnel'. The latter is composed of steel hoops covered with polythene and usually with walls of plastic mesh. The polythene covering has to be replaced periodically, some types every 3 or 4 years, but some last 10 years. Most buildings may be used for some other venture (e.g. calves) during the summer and autumn.
- Disease risks: intensive housing may increase the risk of certain diseases such as neonatal scours, coccidiosis, FR and pneumonia but these can be controlled by good construction and management.
- Food and bedding costs: food costs in March will be increased because the young grass is deliberately not made available to the ewes but this will be beneficial in the long run. Straw is an additional cost.

Principles of Design

Site

The house should: (i) be convenient for the shepherd; (ii) be sheltered from the prevailing wind and snow; and (iii) have ready access to water and electricity.

Design

It is usual to have a wide centre passage, which allows access for feeding silage, or may hold hay and concentrate feed, and can be used for individual lambing pens later. Pens to hold 30–60 ewes are sited on either side of the passage. The centre passage should be concreted but hardcore is best for the pens to permit good drainage. The size of the pens varies with breeds and shearing but should allow 1.0–1.3 m^2 (10–14 ft^2) per ewe at least. It is most important that the ewe should have ample trough space (0.45 m/18 inches each). A useful design is to have removable, double-sided pen dividers, each side consisting of a lower food trough and an upper hay rack with a central walkway between the two sides. Ground level 'walk-through' troughs are also often used for hay feeding. Alternatively, silage can be floor-fed in the passage with ewes reaching through a simple barrier, with concentrates also fed similarly, or scattered on the (clean) bedding. Straw bedding should be provided; about two bales of straw are needed for each ewe for a 3-month period. Slats can be used, but ewes should not lamb down on them. The house must be well ventilated by Yorkshire (slatted) boarding, plastic mesh or open sides but be draught proof at sheep level by having solid walls to a height of 1.0–1.2 m. Water may be provided in troughs, self-fill bowls or continuous flow along a rainwater gutter. The water

should be at a height that the sheep can reach but not too low or it will become contaminated with faeces and thought should be given to the increase in bedding height which occurs during housing. Good lighting is essential as are power points for shearing and particularly for infra-red lamps over the individual lambing pens and for the shepherd's kettle!

Shearing Housed Ewes

For many years in the UK, the winter shearing of ewes was done when the ewes were housed since this: (i) reduced heat stress and humidity which predisposes to pneumonia; (ii) enabled more ewes to be kept in the same space; (iii) increased food consumption and lamb birthweight; and (iv) enabled easy observation of lambing ewes without handling. It also provides some control of lice and keds since they breed more rapidly in thick wool.

There were, however, disadvantages associated with the practice. If conditions were extreme, ewes became too cold and huddled together and it was necessary to allow at least 2 months' wool growth before turnout which may coincide with bad weather and thus delay turnout. There were increased feed costs and single lambs may be too large, causing dystocia and 'wool slip', associated with stress, was common. In addition, the thick fleece which followed in the summer caused ewes to be too hot and distressed.

As a result of these disadvantages the practice seems to have died out and it is unusual to find sheared ewes in houses during the winter.

Abortion

The incidence of abortions in 'normal' flocks is usually quite low (1–2%) and is tolerated by the farmer without investigation. However, it must be remembered that many of these abortions are caused by pathogens or nutritional disturbances, which also cause: (i) apparently 'barren' ewes; (ii) fetal mummies; and (iii) very weak nonviable premature lambs. Abortions, therefore, even in small numbers should always be considered as only part of the losses in the production process and farmers should be questioned about these other aspects of reproductive loss before dismissing them as unimportant.

When, however, an infectious abortion agent is introduced into a fully susceptible flock, the incidence of abortions is often alarming and is recognized as a 'storm'.

Causes

All abortions should be regarded as infectious until proved otherwise. Although the prevalence of the different types of infectious abortion varies from year to year and from area to area, overall the four most common causes of infectious abortion throughout the UK are:

- EAE caused by *Chlamydophila abortus*;
- *Toxoplasma*;
- *Campylobacter*; and
- *Salmonella* – various serotypes.

Although these four infections are the most important in the UK, some do not occur in other countries and the relative importance of each will be indicated when dealing with each specific infection. In summary, these are the most common causes in countries in northern Europe. *Brucella melitensis*, which does not occur in the UK, where it is notifiable, is common in southern Europe and is an important zoonosis. *Campylobacter* is more important in North America than in the UK. Australia and New Zealand are fortunately free of EAE.

A number of other agents can cause abortions, but these are generally sporadic occurrences which are relatively insignificant. These include:

- Border disease virus (BDV);
- *Listeria*;
- tick-borne fever (TBF) – *Ehrlichia*;
- Q fever – *Coxiella*;
- *Arcanobacterium pyogenes*;

- *Yersinia pseudotuberculosis*; and
- fungi.

Reports from diagnostic laboratories give useful figures to indicate the relative importance of the infections in different countries of the world.

For the UK, annual reports from Veterinary Laboratories Agency (VLA) laboratories (now AHVLA having merged with Animal Health) give a good indication of abortion trends over at least the previous 8 years (good for other disease information too! – see www.defra.gov.uk/ahvla and in the site A–Z find 'VIDA' in the index page; VIDA is the Veterinary Investigation Surveillance Report). Table 5.1 shows figures for the years 2005–2009 inclusive, 2009 being the latest completed figures. Each figure is a separate incident, not the total number of samples received and may therefore include several individual ewes. The figures show a trend for a reduction in EAE (*Chlamydophila*) and *Toxoplasma* whereas *Campylobacter* and *Salmonella* incidents remain similar for each year, perhaps reflecting the use of the vaccines for EAE and *Toxoplasma*.

It might be thought that since almost half the incidents do not result in a definite diagnosis, laboratory investigation of abortion outbreaks is unrewarding and not worth the costs involved. However, as a general rule, if there is a significant abortion problem and the laboratory receives sufficient useful material, the cause will be identified allowing the appropriate specific control to be instituted and perhaps allowing treatment of the ewes which have not yet aborted. The most likely reasons for laboratories failing to arrive at a diagnosis are that the material submitted is unsuitable and incomplete and may be just the 'odd' abortion from the 'normal' flock. In addition, laboratories may only test for a limited number of infectious agents, though all will look for the four main agents.

Table 5.1. Abortion incidents reported by the VLA 2005–2009 inclusive.

	Number of incidents
Total abortion incidents	10,493
Diagnosis not reached	5,045 (48.1%)
Chlamydophila	2,152 (20.5%)
Toxoplasma	1,484 (14.1%)
Campylobacter	675 (6.4%)
Salmonella spp.	291 (2.8%)
Other diagnoses	846 (8.1%)

In order to allow the laboratory the best possible chance of finding a causal agent, the following materials should be submitted as soon as possible after the abortion incident:

- Fresh fetal membranes with cotyledons (if these are not available then supply swabs from the wet skin of the fetus or of vaginal discharge).
- Fresh fetus(es).
- Information, which will include:
  - size and nature of flock (e.g. is it self-contained? Are the abortions in a particular group such as purchased, yearlings?);
  - feeding of flock;
  - previous abortion history, including any laboratory reports;
  - dates of lambing;
  - facilities for isolation; and
  - possible reasons for the abortion(s) for example handling and dosing, predisposing diseases such as fluke and PT and dogs (though the evidence for the latter is slight!).
- It may also be useful to submit a clotted blood sample (red tube) to test for serum antibodies for *Toxoplasma* or EAE (it is worth tagging aborting ewes so that they can be identified later). However, it is best to leave this for 1–2 weeks and only submit the sample if no diagnosis was reached from the fetal membranes or these were not available.

Remember that some ewes can produce twins, one of which is apparently normal, and the other abnormal and infected. A flock may be infected with more than one pathogen (e.g. *Toxoplasma* and EAE) so that it is worth continuing to submit aborted material to the laboratory even after the first positive diagnosis has been established (perhaps 10% of subsequent abortions).

General advice

- Isolate the aborting ewe and retain the fetus(es) and membranes for laboratory investigation; treat these as infectious (use polythene gloves and bags), not forgetting the public health risks from *Chlamydophila, Salmonella* spp., *Listeria,* Q fever and (less directly dangerous) *Toxoplasma* (since oocysts are the most dangerous stage).
- *Chlamydophila* is particularly dangerous for pregnant women and their unborn children and they should not be involved with the lambing flock, since abortion may arise in a clean flock without notice! Keep aborting ewes in isolation until a positive diagnosis has been made and until obvious vaginal discharge has ceased (up to 3 weeks) and tag so they may be identified later, if necessary, for blood sampling or culling. If the ewe is ill, antibiotic therapy should be given but only after vaginal swabs have been obtained to submit to the laboratory.
- Do not use aborting ewes or ewes producing premature lambs as foster mothers until it is known to be safe to do so.
- It is usually better to retain aborting ewes and not sell them, because in general, they will now be immune to at least one pathogen and not abort again.
- For *Campylobacter*, it may be worth allowing the aborted ewes to mix with the ewes which have already lambed, and so induce general flock immunity (i.e. 'move on' rather than 'move back'). It is essential, however, to be absolutely certain that *Chlamydophila* is not also present before recommending this practice. (Mixing ewes that have aborted with *Chlamydophila* will increase the number of latently infected animals which may then abort the next year!).
- It is no longer sound advice for bought-in sheep to mix with the resident sheep, the general farm environment and on-the-farm food before tupping, for a variety of biosecurity reasons. They should, where possible, lamb separately in their first season on the farm, in case they are infected with *Chlamydophila* and introduce EAE into the whole flock.
- An abortion 'storm' is usually not repeated in subsequent seasons and subsequent fertility is good, unless a different pathogen is introduced.
- At the time of abortions, those ewes yet to lamb should be kept apart from the infected group and spread out, and the possibility of therapy (see specific infections) should be considered. Where possible, they should not lamb in the same area as the infected group, or at least the lambing yard should be re-strawed.

Enzootic Abortion of Ewes (EAE)

This form of abortion is the most commonly diagnosed in the UK and most northern European countries but is notably absent from Australia and New Zealand. EAE is caused by *C. abortus*, a highly specialized bacterium which parasitizes host cells, in which it undergoes a complex life cycle involving the formation of reticulate and elementary bodies.

Although chlamydiae are bacteria and are susceptible to certain antibiotics, they require 'virological' (tissue-culture) techniques for their isolation and propagation and cultural confirmation of diagnosis is not routinely available.

Infection of pregnant women causes death of the fetus and abortion and a very serious life-threatening disease in the mother, though only a small number of cases have been reported. Considering the widespread prevalence of EAE, it is perhaps not very infectious to humans though the best advice is that pregnant women should not be involved with lambing flocks.

Clinical signs

Abortion occurs in the last 2 or 3 weeks of pregnancy with no evidence of illness in the ewes. The pathology of infection is essentially a necrotic placentitis so lambs usually

appear fresh and show little or no gross pathology, although the abdomen is sometimes distended with blood-stained fluid. In addition to aborted fetuses, premature and weak live lambs, and even apparently healthy lambs but with infected membranes, are seen. Fetal membranes are sometimes retained, which may lead to metritis, but usually no clinical signs are seen in the ewes.

Epidemiology

- The most important source of infection is from aborting ewes since the organism is mainly excreted at the time of abortion and in subsequent vaginal discharges, which resolve in a maximum of 3 weeks. Infection is, therefore, mainly spread at lambing time and the rate of infection and the development of flock immunity is usually slow. Few 'storms' occur but the incidence of abortions tends to persist for years unless otherwise controlled. The disease is almost unknown in hill flocks where the management at lambing is less intensive than lowland, whereas it represents a serious and increasing hazard for sheep lambing under intensive, housed conditions.
- Infection is introduced to a 'clean' farm by the purchase of ewes which are latently infected. The chlamydiae are mobilized during pregnancy, giving rise to abortions etc. 1 year after the ewe became infected. **No serological response can be detected during the 'latent' infection and there is no method of deciding whether purchased ewes are infected.** Since only a few purchased ewes may be infected and abort, material may not be submitted for diagnosis and the disease is not recognized until a much greater number of ewes abort at the next lambing season.
- The characteristic picture, therefore, is that the first season of infection shows itself as a few purchased ewes aborting or producing premature lambs; the next one or two seasons, abortions and stillbirths occur in all age groups and the following seasons abortions etc. are mainly confined to yearlings and bought-in sheep, since the older ewes will have acquired immunity, which is dependent on infection of membranes.
- Lambs can be infected at birth from their mother or other ewes and produce infected lambs and membranes at their first lambing (which may not be until they are shearlings) and then become solidly immune.
- Ewes infected for the first time in late pregnancy do not usually abort then but may do at their next pregnancy. It takes about 40–50 days from infection to abortion which means that infection and abortion can occur within the same lambing season if batch lambing is practised. Ewes with lambing dates of more than 6 weeks apart should not be mixed close to lambing.
- Intestinal chlamydiae are common in sheep but the majority of these are now described as a separate species, *Chlamydophila pecorum*. The intestine may also act as a reservoir of the abortion chlamydia, *C. abortus*, but there is little evidence that clean ewes become infected from these chlamydiae.
- Rams can become infected and may show epididymitis, but it is generally accepted that there is little likelihood that infection will be introduced into a flock or transmitted within a flock by rams. However, it seems unwise to purchase rams from flocks which are known to be infected.
- Chlamydiae are not transmitted in the milk of infected ewes.
- Some workers have reported that ewes which have previously aborted may shed chlamydiae at subsequent oestrus periods, but there is no strong evidence that this is an important route of transmission. Most aborted ewes are considered to be solidly immune.
- Recently, *Chlamydophila* of the vaccine strain have been isolated from a few cases of abortion but it is accepted that this is no reason to cease vaccination as a control method.

Diagnosis

There is usually an obvious placentitis with thickening and necrosis, so look for them before sending to the lab. The lesions of abortion caused by *Y. pseudotuberculosis* are very similar and the membranes should be stained for chlamydiae and subjected to bacteriology for this agent. Modified Ziehl-Neelsen staining of smears from chlamydia-infected cotyledons will usually show large numbers of intracellular inclusion bodies. However, infection may not be very obvious in the placenta when organisms are few and tissue culture has revealed organisms where smears have been negative. If no membranes are available, smears can be made from the wet skin/fleece of a recently aborted fetus or from the vagina of the ewe for 24 h or so after abortion. Examination of the sera of an aborting ewe for complement-fixing (CF) antibodies will usually show a titre of at least 64 if EAE was responsible, but it is not usually necessary unless membrane material is not available. Interpretation of CF titres must take account of vaccination history and previous abortions because these may produce persistent titres. Kits are now available which will identify chlamydia antigen in membranes.

Treatment

Aborting ewes rarely require any treatment other than isolation until their discharges cease. It is wrong to mix these sheep with lambed ewes until discharges cease since the lambed ewes may become latently infected and abort next year.

A decision has to be taken immediately after a positive diagnosis has been made as to whether it is worth treating the ewes yet to lamb. This decision will be influenced by the size and value of the flock as well as a guess as to what the subsequent abortion incidence is likely to be! Oxytetracycline is effective against chlamydiae and injection of an LA preparation will maintain pregnancy until nearer the expected lambing date. Injection could be repeated 2 weeks later for ewes which appear not to be close to lambing. Oxytetracycline does not sterilize the infection so membranes are still infective. It is as well to warn the farmer that, despite treatment, some ewes may still abort or produce weak lambs.

In the first year after an outbreak in a valuable flock, it may be worth injecting LA oxytetracycline 6 and 3 weeks prior to the expected lambing date to reduce abortions in ewes which are latently infected.

Control

If EAE has been diagnosed

The following measures should be employed:

- Reduce transmission to other ewes.
  - Remove and destroy all membranes, whether believed infected or not (on-farm burial is not now legal therefore they should be burnt or disposed of with dead lambs).
  - Attempt to clean up the area in the lambing shed and cover with clean straw.
  - The use of aborted ewes to foster lambs should be discouraged but, if practised, the lambs must be sent for slaughter and females not retained for breeding since it is highly probable that they will have become latently infected.
  - Ewes which abort should be kept since they will be immune and most evidence suggests that they will not continue to shed chlamydiae.
- Protection of ewes by vaccination. There are two *Chlamydophila* vaccines but both consist of the same live heat-sensitive strain. Vaccination not only protects against abortion but also reduces the number of chlamydiae shed at lambing.

Vaccines should be given in the 4 months prior to mating, but not within 4 weeks of the ram being introduced. Boosters are not usually given. Although the vaccine is live, the chlamydiae do not

usually persist in the vaccinated ewe. Vaccinating ewes which have been infected the previous lambing season (i.e. the latent carriers) reduces the abortion rate by about 50% so the farmer should be warned that vaccinated ewes might abort or produce weak lambs. Great care should be taken to prevent self-injection of the vaccine since it contains live chlamydiae and it is wise that pregnant women should not handle the vaccine at all. The wearing of gloves and the use of a guarded needle system is essential.

If EAE is not present on the farm

The following strenuous efforts should be made to prevent its introduction:

- Maintain a self-contained flock. No female breeding stock or lambs for fostering should be purchased except from known clean flocks and although it is generally believed that rams are of little risk, it is preferable to use the same precaution in their purchase. The penalty for introducing EAE is so severe that no sheep is a bargain!
- Purchase replacements from flocks which are monitored free from EAE as part of the Premium Health Scheme of the Scottish Agricultural College (SAC) or on other well-attested evidence.
- Lamb purchased ewes separately from or later than the 'home' flock for the first year and investigate all abortions and apparently barren ewes, carefully.

Toxoplasmosis

Caused by *Toxoplasma gondii*, a coccidian protozoan, in which tissue cysts are found in virtually all mammals including man, and in birds, and in which a sexual cycle is completed only in cats and other Felidae, to produce resistant oocysts. When susceptible sheep ingest sporulated oocysts (*Toxoplasma* resemble *Isospora* oocysts, with two sporocysts each containing four sporozoites), the sporozoites penetrate the epithelial cells of the small intestine and are distributed to many organs, including muscles, brain and, in pregnant ewes, the placenta. Multiplication occurs and cysts develop in these sites, which remain viable for the life of the sheep but are not mobilized. This means that adverse effects are only seen in pregnant ewes soon after the ingestion of oocysts. A solid immunity develops about 4 weeks after infection which lasts for life.

Infection of susceptible pregnant women by oocysts or from tissue cysts in undercooked meat may result in abortion or fetal abnormalities including hydrocephalus.

Clinical signs

These depend on the stage of gestation at which infection takes place.

- If oocysts are ingested in the first 60 days, fetal death and resorption occurs. If the rams have been removed the ewes will then appear to have been barren or, if the rams are still present, the ewes will be served again.
- Infection between day 60 and day 120 results in the typical signs – abortion in late pregnancy, often with mummification of one or more fetuses, or with one or more stillborn lambs, or the production of weak lambs.
- Infection after day 120 results in an infected normal lamb which becomes immune.

The ewes are not obviously ill, though they do have an increased temperature and lose appetite a few days after infection. Abortion usually occurs about 30–50 days after ingestion of the cat oocysts.

Epidemiology

- Sheep become infected by oocysts passed in cat faeces. Infection and abortion can result from infection with as few as 200 oocysts. The most likely sources of infection to sheep are by contamination of stored grain or hay or from pasture, by the spreading of manure containing cat faeces or by cats

defaecating on the pasture. The oocysts are very resistant and remain viable for many months.

- Immunity following infection is strong and ewes never abort more than once. If infection occurs outside pregnancy, the ewe will become immune, without showing any signs.
- Although it is known that rams can excrete the organism in their semen for a short time after infection, this does not seem to be of importance and infection at this time, of course, would not result in abortion.
- Ewes do not become infected by eating infected membranes, that is **direct ewe-to-ewe transmission at lambing does not occur**.
- As mentioned above, vertical transmission occurs if susceptible ewes ingest oocysts after 120 days of gestation. However, although infected ewes have viable *Toxoplasma* cysts in their tissues for life, their mobilization and vertical transmission probably occurs very rarely.
- Young cats first become infected when they commence to hunt, particularly from mice, and shed many millions of oocysts over a few days about a week later.

Diagnosis

- Fresh fetal cotyledons often show small (2 mm) white necrotic or calcified foci – 'white spot placenta' or 'frosted strawberries', often attached to brown, dry mummifying fetuses. The white foci are often easier to see if a microscope slide is placed on the cotyledon, or the cotyledon pressed against the side of a clear polythene bag.
- Direct immunofluorescent antibody techniques on sections or smears of cotyledons show brightly fluorescing tachyzoites or cysts.
- Serology on pleural or peritoneal fluids of stillborn lambs, live lambs before they have taken colostrum or IgM antibody in post-colostral live lambs, as the presence of antibody in the fetus indicates an active infection in the uterus at the dangerous time.
- Histology or smears of placenta and histology or squash preparation of fetal brain may show *Toxoplasma* cysts or a specific cell reaction.
- The detection of specific antibody by various techniques (e.g. indirect haemagglutination, latex agglutination, ELISA) is useful in epidemiological studies but antibody persists for years and serology is of little value in individual aborting sheep. Paired sera taken at abortion and 2 weeks later with a marked increase in titre may be helpful.

Treatment and control

- During an outbreak of toxoplasmosis, aborting ewes are not dangerous to other ewes, nor useful in transferring infection and therefore immunity, so there is no obvious justification in continuing to isolate them once the diagnosis has been established. However, the farmer can easily become confused with this exception to the rule, and one must also always be aware of mixed infections, especially with *C. abortus*. Abortions and fetal membranes should be handled with disposable gloves due to the zoonotic risk.
- During the outbreak there is usually little to be done other than to grin and bear it, although it is prudent to look for the possible ways that sheep could have ingested cat faeces containing oocysts and postpone feeding any contaminated material until after lambing. Keep cats away from cereals, hay and bedding likely to be available to pregnant sheep (pelleted concentrates are likely to be safe since they are subject to high temperatures).
- The organism is, however, susceptible to sulfonamides and injection of a high dose (four times the normal dose) is worth considering (there is no licensed sheep product but cattle products are

available), although some fetuses will be dead and undergoing mummification at the time of treatment. Treatment of experimentally induced toxoplasmosis in ewes with a combination of sulfamezathine and pyrimethamine resulted in more live lambs and less pathology than untreated controls.

- After lambing, retain the aborters and maintain within a closed flock, or introduce bought-in replacements to the farm environment for as long as possible before tupping so that they are exposed to material likely to contain cat oocysts.
- A commercial vaccine was first marketed in New Zealand and has been used widely in the UK and elsewhere. It consists of live attenuated tachyzoites and is injected into non-pregnant ewes at least 3 weeks prior to tupping and can be given at the same time as an EAE vaccine but at a different site. The vaccine is given via the intramuscular (IM) route, multiplies in the sheep for a few days but does not give rise to tissue cysts in the muscles. It is infectious to humans and the same precautions as are necessary for the live vaccines for EAE must be used. It is assumed that immunity is boosted by natural infection so it is usual to only vaccinate once.
- Chemoprophylaxis with decoquinate given in pregnancy (perhaps to purchased ewe-lambs only, during mid-pregnancy) at a concentration designed to produce 2.0mg/kg body weight is effective in reducing abortion and is palatable. The problem is that it is unusual to feed concentrates during the susceptible period (60–120 days gestation) and thus costs are increased and practical problems of feeding ewes at this time are encountered. Under most circumstances, vaccination is the better option but this must be planned before tupping.

Campylobacteriosis (Vibriosis)

The organism causing this form of sheep abortion was originally classified as *Vibrio fetus*, leading to the disease being called vibriosis but it is now agreed that it belongs to the genus *Campylobacter*. Sheep abortion is caused by *Campylobacter fetus fetus* and *Campylobacter jejuni*. It is particularly important in New Zealand and North America.

Clinical signs and epidemiology

- Infection is ingested and is primarily intestinal. It is therefore excreted in the faeces from symptomless carriers, which often introduce infection into a susceptible flock.
- Ewes are rarely ill at the time of abortion.
- Birds (e.g. crows and magpies) and voles may introduce the infection to a flock as well as introduced carrier sheep; the infection is then spread from sheep to sheep via faeces, abortions and personnel.
- Early fetal loss is rare as ewes appear to be resistant in the first 3 months of pregnancy; campylobacter infection is therefore not associated with 'barren ewes'. Bacteraemia follows infection in later pregnancy followed by placental infection which results in abortion 7–25 days after infection.
- Immunity following infection is strong to a particular serotype of which there are three in the UK and many more in other countries like New Zealand, and subsequent fertility is good. Outbreaks may re-occur every few years because of lack of immunity in replacement stock, but usually abortions are few in number after the initial year of introduction.

Diagnosis

There are no visible lesions in the placenta though visible focal necrosis of fetal liver is sometimes present but the organism can be identified in smears or cultured from cotyledons, fetal stomach or liver. *Campylobacter*

is not difficult to culture, but exact typing is complicated. Serology is not helpful.

Treatment and control

- Usually the individual aborting ewe does not require any therapy. Once the diagnosis is confirmed, such ewes should mix with the ewes which have already lambed, but not with the pregnant sheep.
- The remaining pregnant sheep must be kept away from the infection as far as possible and spread out, although the number and spread of abortions may not make this possible. Consideration should be given to antibiotic treatment of groups of pregnant ewes, with, for example, penicillin and streptomycin for 2 days or LA oxytetracycline.
- The farmer should consider keeping a closed flock, although infection can be introduced by birds. Aborting ewes should not be sold. If replacements are bought then they should be mixed with the resident flock for as long as possible before mid-pregnancy, but separated in late pregnancy.
- Effective vaccines are not available in the UK but are used in other countries such as Australia and New Zealand where the disease is more common.

Salmonellosis

One sheep-specific salmonella, *Salmonella* ser. Abortusovis, causes abortion as do a number of other less-specific serotypes including *Salmonella* ser. Typhimurium, *Salmonella* ser. Dublin and *Salmonella* ser. Montevideo. *S.* ser. Abortusovis is now rarely reported in the UK but is found elsewhere. Abortion is the only clinical sign with some serotypes like *S.* ser. Abortusovis and *S.* ser. Montevideo but some serotypes are much more pathogenic for ewes, resulting in severe illness and death in ewes. This is particularly true for infection with *S.* ser. Typhimurium and *S.* ser. Dublin.

Clinical signs and epidemiology

- Abortions occur mainly in the latter half of pregnancy, although apparently barren ewes may reflect earlier fetal loss.
- Ewes may also be ill at or before the time of abortion: some scour, some die, some have an offensive smelling vaginal discharge for a week or more.
- Except for *S.* ser. Montevideo, lambs are often ill at or soon after birth, and develop a fatal septicaemia or pneumonia.
- Symptomless carriers are found in both ewes and lambs though the serotypes which cause the more severe disease in ewes often result in a solid immunity without carrier status in those ewes which do recover.
- A measure of immunity to a particular serotype is apparently induced because it is uncommon for ewes to abort the next season.

Diagnosis

- There are no obvious lesions in the placenta.
- *Salmonella* is usually easily cultured from membranes and fetus, providing faecal over-growth is avoided (a possibility if only vaginal swabs are provided).
- Blood testing and rectal swabs provide little worthwhile information.

Treatment and control

Salmonella is frequently found to be sensitive to a range of antibiotics *in vitro* and theoretically treatment of individual sick animals is indicated. In practice such treatment frequently fails to cure the sick or sterilize the carrier. Consideration must be given in any salmonella outbreak to the risks of infecting humans and to producing drug-resistant organisms.

The principal aim in the control should be to:

- Reduce the weight of infection by the isolation of aborting, scouring and sick sheep.
- Prevent further infection of pregnant sheep, as far as possible, by keeping the aborting group (usually the nearest to term) separate from other groups yet to lamb.
- Upset the flock as little as possible and make sure food (hay or concentrate) is always available, particularly immediately following flock movement.
- Try and ensure that most of the flock becomes immune after lambing by spread from carrier ewes to non-pregnant ewes (i.e. mix recovered aborters with ewes that have lambed).

Avoid re-introduction of the disease from sources such as: (i) 'foreign' slurry; (ii) bought-in sheep and other grazing stock; and (iii) infected feed (including bird faeces). Attempts should be made to prevent birds, especially magpies, entering sheep houses and feeding from the sheep troughs.

Q Fever (*Coxiella burnetii*)

This has recently been a problem in Germany and the Netherlands where there have been many outbreaks of disease in sheep and goats and associated human disease. Control measures have included slaughter of pregnant animals to reduce the risk of zoonotic spread; however a vaccine is now available to aid control.

Box 3.1. Schmallenberg Virus

As this book went to press, an apparently completely new virus disease of cattle, goats and sheep was emerging in countries in northern Europe. Named after the German town near which it was first identified, the disease has now also been diagnosed in the Netherlands, Belgium and on 23 January 2012 in flocks in some eastern counties of England. The disease causes fetal malformations (hydranencephaly, arthrogryposis and scoliosis) and stillbirths, affecting up to 50% of lambs in infected flocks. The causal organism has been identified as an orthobunyavirus transmitted most probably by midges (*Culicoides* spp). Initial infection probably occurred during late summer/early autumn 2010 in ewes in early pregnancy, with the results not evident until lambing time. At the time of writing no decision has been made regarding making the disease notifiable or not, or possible control measures. Latest information is available at www.defra.gov.uk/ahvla

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6

The Periparturient Ewe

Farmer's First-aid Cupboard

This is the time of the production cycle at which most can go wrong and is when most vets have their main contact with sheep. Remember 'animals under your care' guidance for prescribing drugs – the flock should be seen within reasonable time prior to dispensing items such as antibiotics. Here is a list of items that should be available on farm well in advance of lambing time:

- lubricant (petroleum jelly or soapflakes can be used as alternative);
- arm-length disposable gloves;
- disposable paper towels;
- prolapse harnesses;
- lambing cords and snare (Hughes snare or make one from plastic-coated cable);
- lamb stomach tube and syringe or funnel;
- frozen colostrum (200ml lots) – preferably ewe, but goat or cow as an alternative;
- commercial dried colostrum is useful for an emergency;
- antibiotic injection;
- antibiotic aerosol;
- antibiotic oral doser for lambs;
- antibiotic ophthalmic preparation;
- dextrose 40% injection;
- calcium borogluconate injection (400ml bottles of 20% Ca);
- oral twin-lamb disease remedy;
- tincture of iodine (e.g. in a spray);
- cotton wool;
- surgical spirit;
- needles 16G × 1" (i.e. 16 gauge 1 inch), 18G × 1", 19G × 1", 21G × 5/8";
- sharps container;
- syringes 50, 10, 5 and 2ml; and
- thermometer.

Important Periparturient Problems

The major problems to be faced (Fig. 6.1) will be dealing with:

- abortions (see Chapter 5);
- metabolic diseases;
- prolapses; and
- obstetrical problems.

Metabolic Diseases (PT, Hypocalcaemia and Hypomagnesaemia) – a Clinical Approach

This is a particularly difficult set of conditions to cope with, mainly because:

- They are difficult to differentiate clinically.
- They may coexist.

Fig. 6.1. Individual pens allow ewes with health problems to be cared for as well as for separating ewes with newborn lambs.

- The farmer has often already treated the animal and confused the picture with various treatments as well as causing delay in diagnosis.
- A number of cases may occur at the same time.
- The results of treatment are often disappointing.
- Prevention is not simple.

However, if one tries to keep one's feet on the ground, some sort of logic can prevail which should lead to a satisfactory outcome.

Clinical diagnosis

It is always useful to take a pre-treatment blood sample (red or green top for Ca, Mg, betahydroxybutyrate (BOHB); grey top is needed for glucose and P). This is then available if the animal(s) fails to respond to treatment. If it recovers, your diagnosis was correct! Treatment needs to be prompt for all three conditions, otherwise the prognosis is hopeless; hence therapy by the farmer is usual, with the vet being called if usual treatments fail or many animals are affected. It is important to emphasize the necessity to treat early – delay leads to permanent damage (especially with PT) or death with loss and dissatisfaction all round.

If the diagnosis is uncertain

A 'rough-and-ready' approach is:

1. Take pre-treatment blood sample (red or green and grey tubes) for Ca/Mg/glucose/BOHB estimations.
2. Give 20–40ml of a calcium borogluconate, magnesium and dextrose (CaMD) solution and 20–40ml dextrose 20% slowly intravenously.
3. Give 50–100ml CaMD subcutaneously.
4. Give electrolyte and/or propylene glycol orally.
5. If there is no response send bloods for analysis and review your diagnosis, checking particularly for signs of listeriosis.

Pregnancy toxaemia (PT twin-lamb disease)

- This occurs in the last month of pregnancy and before spring-grass growth.
- It affects thin ewes (CS 2 or less), less commonly it may occur in overfat ewes.
- Insufficient food has usually been given, particularly concentrates, although poor quality forage also contributes to the condition. Underfeeding has usually

gone on for some weeks but also may be sudden (e.g. following bad weather or movement).
- Affected ewes are usually old and may have broken mouth and molar problems, but shy young ewes with inadequate access to limited feed may also be affected.
- Affected ewes usually have a large abdomen (two or more lambs).
- The first sign is refusal of feed. Affected ewes become separated from the rest of the flock, are easy to catch, apparently blind and stand motionless, showing fine tremors and may develop convulsions on handling.
- Test the urine (obtained by occluding nostrils for a few moments). A rapid and strong positive result for ketones suggests PT; a negative result makes it unlikely.
- The blood sample shows level of BOHB is >3 mmol/l.
- If not treated, the condition progressively **worsens over days** and the animal dies.

Treatment

- Treat as soon as refusal of feed is noticed – don't 'wait until tomorrow to see what happens', it may be too late!
- Treatment by the farmer on the first day – administer proprietary PT oral treatment and/or 50 ml propylene glycol orally and repeat in a few hours. Offer good hay and a little high energy palatable concentrate (e.g. flaked maize). You hope that the ewe will begin to feed by then. If the owner is prepared to give the time, hand feeding can start an improvement. If housed, turning out for a few hours on to good grass, if available, may help to improve the appetite.
- Treatment by the vet – give 50–100 ml dextrose 40% plus 50 ml Ca 20% intravenously (use the cephalic or jugular vein and inject very slowly to avoid adverse effects on the heart) in addition to the above oral treatment. Dextrose can be repeated after a few hours. If there is no response, review your diagnosis and prognosis.
- The next day – if the ewe is improving, continue with a drench until the ewe is feeding well, then monitor carefully. Avoid repeated propylene glycol for more than 2 or 3 days as it appears to lead to scouring.
- If the ewe is not improving, either slaughter, or if less than 1 week before term, abort the lambs by 8 ml (16 mg) of beta or dexamethasone, or perform a caesarean section. The outlook is poor anyway – it takes 36–48 h for induced lambing to take place, and the lambs will be dead or have low viability. A caesarean is also unlikely to produce viable lambs, but may save the ewe if it is not in irreversible stage of disease (it is definitely not worth doing if the ewe is comatose).
- **Remember, early treatment is essential; delay will lead to loss of ewe and/or lambs.**

Control

- Condition score the sheep 6–8 weeks before lambing is due to start and split according to condition; also separate shy feeders.
- If ewes have been scanned, split them according to fetal numbers but use the CS as well. Put thin ewes scanned for a single lamb with the twins group and thin ones scanned for twins with the triplet group.
- Feed more good quality concentrates (including some whole grain) to all sheep below CS 3, preferably giving two feeds daily. Be careful not to increase concentrates too quickly as this may lead to acidosis. Check the quality of concentrates – contact manufacturer for details of ME value.
- Check the quality of hay or silage – feed the best available to ewes expecting twins or triplets.
- If possible, give access to green grass. If the problem was precipitated by housing, consider turning out again. Give more feed outside and re-house when eating well.

- Consider giving high energy or protected energy self-help blocks.
- If outdoors, try to move the ewes to more sheltered fields.

In future, scan ewes at 50–80 days gestation and mark according to the fetal load. About 2 months before lambing, condition score all the ewes and split them according to fetal load and body condition and feed accordingly. Check the CS again at the time of vaccination. Select the best forage for late pregnancy. The season with the lush autumn (therefore high ovulation rate) and hard winter is the one which can create the worst problems. The loss in condition is masked by fleece growth, hence the need to handle the sheep.

- **Remember, it is dangerous to assume sheep with a heavy fleece are in good condition by just looking at them.**

Hypocalcaemia (trembling, lambing sickness)

- This is most common in late pregnancy rather than early lactation, in contrast to cattle, but can occur just after lambing.
- It can affect ewes in any condition.
- Affected ewes show typical signs of 'milk-fever' – ataxia, leading to recumbency, depression and atony (bloat, no faeces, etc.) and loss of consciousness. In the later stages, saliva or regurgitated rumen contents may trickle down the nose. It is easy to assume this is a nasal discharge and to misdiagnose it as pneumonia.
- Often there is a history of recent movement (e.g. the ewes have been gathered for vaccination or housing) and a delay or sudden change in feeding, therefore a number of ewes may be affected simultaneously.
- The blood sample shows level of Ca is <0.8 mmol/l.
- If not treated, the condition progressively **worsens over hours** and the animal dies.

Hypocalcaemic ewes may go on to develop PT, especially if they have not eaten for several hours.

Treatment

Give 20–40 ml Ca 20% intravenously slowly (the cephalic vein can be used if the jugular is hard to find) and 50–100 ml Ca 20% subcutaneously. You expect the ewe to respond like a cow with milk fever, eructating, defaecating and even 'walking off the needle'.

- If it does not respond, review your diagnosis. In particular, consider the possibility of listeriosis.

Control

- Watch the flock after movement and have a calcium injection handy.
- If the flock is being moved any distance (walking or by wagon) provide hay and concentrates before leaving and on arrival.
- In theory, keeping the calcium content of the diet a little below requirements until the last month of pregnancy then increasing the content in the ration should help in control, but this method is much more difficult to operate than with cattle, because of the spread of lambing.

Hypomagnesaemia (grass staggers)

- This nearly always occurs after lambing, when the ewe is rearing twins and is at peak lactation (i.e. there is a big metabolic drain on the ewe).
- Usually it occurs when the animal is grazing lush grass (low in magnesium, low in fibre), but occasionally when on bare pastures (insufficient food).
- Signs, if seen, come on very rapidly: excitable, tremors leading to convulsions and rapid death. Often the animal is simply 'found dead'.
- The blood sample shows the level of Mg is <0.6 mmol/l, sometimes coexisting with hypocalcaemia (hence the confusion).

- If not treated, the condition progressively **worsens over minutes** and the animal dies.

Treatment

Give 20 ml CaMD intravenously (where possible, but if ewe is twitchy this may be very difficult) and 50 ml $MgSO_4$ 25% subcutaneously. You hope that the ewe will quickly become less excitable and behave normally within an hour, but some equally quickly die.

- **Remember, don't give $MgSO_4$ 25% intravenously!**

Control

- Watch the flock after movement on to lush or bare fields and have a magnesium sulfate injection handy.
- If on lush pasture, move the animals to poorer grass, until a magnesium supplement is added.
- Feed extra magnesium (up to 14 g MgO/day) via:
  - magnesium-enriched cake; or
  - magnesium-enriched feed blocks or molasses licks (less reliable than above); or
  - magnesium-intraruminal bullets given just before turnout or the risk period – they last 3–4 weeks.
- Give extra food to those animals which are under-fed.
- Provide shelter.

The cost of supplementation has to be weighed against probable losses, based on previous flock history or estimated risk.

Prolapses

Epidural anaesthesia

Epidural anaesthesia is a valuable technique which should be more commonly used for dealing with vaginal prolapses and difficult lambings, or any other condition causing excessive straining.

Control of straining can be achieved for 24–36 h by the correct choice of drugs:

- Local anaesthetic – this will give analgesia for 2–4 h; the hind limbs will be affected if more than 2 ml is used.
- Local anaesthetic plus 2% xylazine – the addition of xylazine will prolong the action for up to 36 h and is particularly useful to control straining associated with prolapses. Draw up 1.75 ml (35 mg) of 2% lignocaine and add 0.25 ml (5 mg) xylazine.
- 2% xylazine – given alone this takes up to 1 h to produce an effect, but then lasts for up to 36 h. A dose of 0.25 ml (5 mg) is made up to 2.5 ml with sterile water.

Epidural technique

The best site is the sacrococcygeal space (locate the first intercoccygeal space which is easily identified by manipulating the tail, and then feel forward for a depression indicating the sacrococcygeal joint which has little mobility). The site should be clipped and surgically prepared. For a ewe, a 20G × 1–1.5" needle is inserted in the midline with the bevel facing cranially (a 21G × 5/8" needle for lambs). The needle usually needs to be angled with the hub caudal to the vertical at an angle of 10–45° (very variable). Advance the needle slowly until a loss of resistance indicates entry of the epidural space (the ewe may wag her tail at this point). Inject the contents of the syringe (preferably warmed to body temperature) slowly. Success is indicated by loss of tone in the tail and loss of sensation in the perineal area. The first intercoccygeal space may be used if difficulty is experienced finding the sacrococcygeal space, but results are not as reliable.

Vaginal prolapse (eversion of the vagina/cervix)

This is an important problem, the cause of which is still uncertain. The incidence, although generally low (1%) and very low

in hill sheep, is sufficient to be a worry, and in some flocks over 5% of the ewes are affected over a short period. Minor prolapses may not cause much disturbance, popping in and out as the ewe gets up and lies down, but if left, severe complications often arise which endanger both the ewe and the lambs; the condition must therefore always be taken seriously and treated quickly.

The prolapse usually occurs in the last 3 weeks of pregnancy (although it may occur as early as 6 weeks pre-lambing), particularly during the last week, in ewes carrying more than one lamb. There may be an association with a particular breeding line or breed of ram but this is unproven. Ewes that prolapse almost invariably do it again next year. Cases therefore need to be marked for culling, as well as to spot breed connections. It is possible to keep such ewes (e.g. if valuable pedigree animals) providing that a suitable external support is put on at the first sign of the prolapse reappearing.

Many causes have been suggested including: (i) multiple lambs; (ii) condition of ewe (too fat or too thin!); (iii) gut fill and roughage; (iv) calcium deficiency; (v) hormone imbalance; (vi) short docking; (vii) steep ground; and (viii) coughing. In other words, there is no one obvious predisposing factor.

Treatment

If treated in the early stages, replacement of the prolapse is usually easy. If there is a delay, damage and subsequent swelling provokes further straining and the urethra may become kinked, preventing urination and the vicious cycle of even more straining is initiated.

- Clean the prolapse and examine the cervix to check that no fetal membranes are visible. If they are, lambing has started and the ewe must be carefully observed after replacement of the prolapse and assistance given if progress does not occur.
- Epidural anaesthesia should be used in cases showing severe straining. This will assist in replacement, allow suturing if considered necessary, and can give prolonged relief from straining if xylazine is administered in addition to local anaesthetic.
- It may be helpful to raise the hind end of the ewe before gently replacing the prolapse. Urine will usually be passed as soon as the urethra is straightened.
- Incomplete dilatation of the cervix (ringwomb) is a common sequel so assistance to lamb or a caesarean may be necessary.

Retaining the prolapse

Non-invasive methods are preferred and are very successful if the prolapse is caught early (Fig. 6.2). More serious cases may require suturing of the vulva.

The most common methods are:

- Wool-tying – this is sometimes used by farmers for only the simplest cases, and is not applicable for shorn ewes! It is no good if animal is straining. It is not a method for the vet to use.
- Application of a harness (truss) using:
  - String (there is a danger of cutting into skin).
  - Soft rope – this is a simple and successful method with a reduced risk of chafing.
  - Commercial truss made of webbing – several varieties are available which work well if fitted correctly. This is the method of choice.
- Intravaginal devices ('spoon') – these support the cervix but can cause irritation and may be pushed out.
- Suturing (under epidural anaesthesia) – there are several patterns including mattress sutures and Buhner suture (see Fig. 6.3). These may tear out if straining recommences and need removing at lambing.

As complications are common, such as persistent straining, ringwomb and dead lambs, the ewe requires special observation. To reduce abdominal pressure, the quantity of roughage should be reduced and antibiotic cover provided where there is obvious

Fig. 6.2. This Herdwick sheep has had a cervical prolapse which is being managed by use of a harness.

damage or infection. Non-steroidal anti-inflammatory drugs (NSAIDs) are also beneficial.

- **Treated ewes must be observed carefully for the onset of lambing – if a harness or suturing has been used, these will need to be taken off once lambing is underway.**

Control (difficult when the cause is not clear)

- Assess the ration, in particular the quality and quantity of the roughage in relation to the ewes' CS and fetal load.
- Look at feeding arrangements – too little rack or trough space leads to pushing which may increase intra-abdominal pressure.
- Encourage regular exercise. Housed ewes may become lazy.
- Mark, record and cull cases before the next breeding season.
- Consider these records and note susceptible lines and breeding.

Post-lambing prolapse of the cervix

This can occur several days or even weeks after lambing and usually follows damage at lambing, particularly tearing of the cervix, which becomes swollen and inflamed, triggering straining. They can be difficult to replace because the pelvic ligaments have tightened up, and difficult to retain in place – suturing the vulva may be necessary, with the suture left permanently until culling, which is definitely indicated.

Vaginal rupture/intestinal prolapse

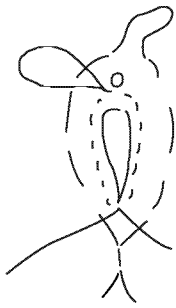
This catastrophe follows a rupture of the vaginal wall near the dorsum of the cervix, with loops of intestine prolapsing through the vulva. Death from shock is rapid. If the ewe is found alive an emergency caesarean, then immediate euthanasia may be performed, but the chance of survival of the lambs is poor. The cause is unknown.

Uterine prolapse

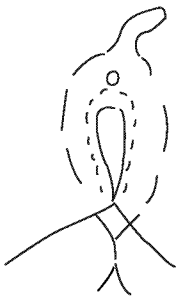
This usually occurs immediately after lambing and, providing the uterus has not been damaged or become chilled, the ewe is not often obviously shocked. The uterus should be cleaned and fetal membranes carefully removed if easily detached, or trimmed if still tightly attached. With the aid of epidural

The suture can be placed, under epidural anaesthesia, using either a Buhner needle or an ordinary large curved suture needle. The Buhner needle has the eye at the point so is inserted then threaded but the same end result can be achieved using either type.

Step 1 – Place the suture subcutaneously to one side of the vulva



Step 2 – Using the same exit points, the suture is similarly placed on the opposite side of the vulva



Step 3 – The suture is tightened leaving sufficient space for the ewe to urinate comfortably

Step 4 – The free ends are tied in a secure bow. Do not cut off the ends – this is so the suture can be undone and retied if necessary

Fig. 6.3. Inserting a Buhner suture.

anaesthesia and with the ewe's hind end raised, the uterus is usually easy to replace. Care needs to be taken not to damage it and check that the horns are fully everted into their correct position. A truss or sutures should be used to prevent a recurrence. Some prolapses occur a few days after lambing, and then shock is often more serious and death is common.

Abdominal Muscle Rupture

The rupture may involve any part of the abdominal wall although most commonly affects the left side, presumably because of the weight of the rumen. It occurs in late pregnancy in lowland ewes carrying twins or triplets and although the ewe is not usually distressed or disturbed at first, complications may arise from trapping of the contents and it may interfere with lambing. Surgical correction is usually impossible. Affected ewes need assistance lambing (rolling on to their back may make reaching the lambs easier) but a caesarean may be needed. Old ewes are most prone, particularly if they are too thin, and perhaps if they are squeezed or knocked during handling and housing.

Obstetrical Problems

It has been calculated that:

- 70% of ewe deaths occur at or near lambing.
- At least 75% of lamb deaths occur at or near lambing.
- 90% of vets' visits occur at or near lambing.

These figures demonstrate that parturition is a high-risk occupation and also that vets are very dependent upon this time for their contact with sheep farmers; but it is rewarding work!

Manipulation of lambs within the uterus is relatively easy in most breeds, particularly for small hands, and limited embryotomy and caesarean operations are simple procedures.

There are particular problems associated with certain breeds and different problems presented in hill and lowland flocks.

The seasonal pattern of lambing presents problems, with: (i) sudden numbers and lack of practice; (ii) a large susceptible population; and (iii) very intensive conditions.

Shepherd interference is common and the lamb(s) are often dead when the vet's assistance is sought, although valuable pedigree ewes may be presented for a caesarean sooner rather than later, particularly if carrying a large single or for a large lamb in posterior presentation. The shepherd's job is usually quite different from the vet's – the shepherd has a problem of numbers, timing (when to interfere), observation, cleanliness, and often simple manipulations or the removal of 'tight' lambs in normal presentation and position, and to ensure the lambs are not smothered and/or mismothered. The vet's role is: (i) to deal with difficult lambings; (ii) to educate the shepherd when it is better to leave well alone and seek help (but remember that many shepherds are extremely good at lambing!); (iii) to diagnose and treat sick ewes and lambs; and (iv) to investigate and advise when flock problems arise. It is also a good time to suggest flock health programmes for the future.

Maternal factors

- Ewes do not withstand prolonged vaginal and uterine interference; a sick ewe often becomes a dead ewe!
- Uterine, cervical and vaginal tears are common following rough handling and inadequate lubrication.
- Clostridial infections are common after vaginal interference if vaccination is inadequate and/or antibiotic not injected.
- Retention of the fetal membranes is rarely a problem.

Fetal factors

- Often there is more than one lamb – a sorting out job – be patient. Remember

how to differentiate front and back legs by feeling the direction of flexion of joints.
- Head and leg deflections are common. Do not pull legs until you have sorted the head out.
- The head is the major cause of obstruction and there is rarely sufficient room in the pelvis for the lamb's head and legs plus your hand, so use a snare over the head to permit traction.
- Tight lambings can lead to fractured ribs, ruptured liver or intra-cranial haemorrhage leading to rapid death, or if lamb survives, it often lies about and is very susceptible to hypothermia. Good aftercare is needed.

Equipment

Ensure that you have the equipment listed below.

- Gel hand disinfectant or soap and water
- Lubricant (proprietary gel or liquid paraffin, petroleum jelly, soapflakes or even lard will do in an emergency) and small hands
- Disposable arm-length polythene gloves (although these can tear or get in the way in difficult lambings)
- Snares (nylon cords with loops or plastic-coated wire – a clean piece of baler twine will do in an emergency)
- Injectable antibiotics
- A sharp knife.

History

Some pertinent questions are:
- Is the ewe at full term?
- How long has the ewe been lambing? The normal range is approximately:
  - change in temperament – 2–4 h;
  - cervical dilatation – 0.5–2 h; and
  - delivery – 0.5–2 h.
- Have any lambs been born or delivered?

Examination

Take special note of the following when examining the animal.

External inspection

- Does the ewe look ill or exhausted?
- Are there signs of shepherd interference? Note bleeding and bruising.
- Presence or absence of lamb or membranes at vulva, strings attached to legs or head.
- Smell – putrefaction implies bad risk, little value and indicates embryotomy rather than caesarean.
- Udder – may indicate prematurity, mastitis, lack of colostrum.

Internal examination

EWE

- Note the size and shape of the pelvis.
- Observe any damage to the vagina and the degree of dryness.
- Consider the extent of softening and dilatation of the cervix. If the cervix is not fully dilated, particularly if unruptured membranes are palpable, re-examine in about half an hour, rather than risk manual dilation. It may just be too early. Be careful if attempting to dilate – tearing and haemorrhage may result.

LAMBS

- Are they dead or alive? (It is not always easy to be certain; assume alive if not sure).
- How many? A jumble of legs and heads implies at least two, probably more.
- Presentation – anterior? posterior? transverse? Breech suggests either large single or triplets (lack of space in the uterus).
- Ankylosed joints may indicate other deformities.

Delivery

- Aim to complete delivery within 15 min or make a quick decision about doing caesarean.

- Consider giving an epidural – this makes things more comfortable for the ewe and easier for you.
- Lubrication is very important – use plenty. Filling the uterus with warm water may help in a difficult dry case.
- Raising the ewe's hind end (but carefully and not for too long) can help to avoid straining and allow repulsion of a lamb.
- Where the uterus is contracted down, consider giving clenbuterol. This is not licensed for sheep, but can make a difficult case easier. (Note: In the UK providing a drug is licensed for a food-producing species it can be used for another species under the so-called 'cascade' system if no suitable drug is licensed.)

Anterior presentation

HEAD ONLY It is common for only a swollen head to be presented outside the vulva, with both front legs retained.

- If the head is not swollen, put a snare on, then carefully replace, find the legs, then deliver normally.
- If the lamb is dead (no suck or blink reflex) then decapitate. If uncertain, treat as if alive.
- If the lamb is alive, lubricate well and insert a small hand beside the head and neck and try to find a limb and extend it. It is often then possible in ewes (but not ewe lambs) to withdraw the lamb with only one limb extended – twisting the lamb while pulling will help to prevent the shoulder becoming locked.
- When the lamb is large or ewe is tight, get one leg up, put a rope on, replace, and repeat with the other leg before using both ropes to pull both legs.

HEAD AND NECK DEFLECTIONS These are common and often made worse by pulling on the legs, expecting the head to follow. The head is the main bulk, so put a loop on the head first, then straighten the head, and then find the correct legs before pulling.

Posterior presentation

Large lambs in posterior presentation are very vulnerable to hypoxia and to physical damage such as broken ribs. Provided both hind limbs and tail are present, withdraw with gentle traction. Be aware of the possibility of stifles locking under the pelvic brim – lift and extend the legs before pulling.

Breech presentations

It takes a good shepherd to spot a breech presentation early; often the only sign is wetness down the back of the udder (indicating rupture of membranes). Gently repel the lamb and search for the hind legs, then bring them up one at a time into the pelvis by flexing/extending the limb joints.

Embryotomy

For ewes with dead smelly lambs, this is a better option than a caesarean as the survival rate is only 50% compared with ewes with live or freshly dead lambs.

For known dead lambs: with a swollen head, decapitate; for other anterior presentations, find a front leg and pull so you can cut around the skin above the knee OUTSIDE the vulva with a sharp knife, then use your fingers to undermine the skin and break down the muscles holding the shoulder blade to the chest. Both forelegs can be removed leaving the head to pull. The more decomposed the lamb, the easier it is. Remove any detached membranes and general debris.

Caesarean operation

Indications for a caesarean are listed below.

- Non-dilatation of the cervix (ringwomb) after one or two examinations at approximately half-hour intervals.
- Fetal oversize.
- Large (valuable) lamb in posterior/breech presentation.

- Irreversible malpresentation.
- Deformities.
- Uterine torsion (very rare – try 'unrolling' first).
- PT – rarely get viable lambs.

The operation is relatively quick and easy, compared with other species, and needs to be considered early in the proceedings rather than too late! The site is the left flank, with anaesthesia by local infiltration, inverted L block or paravertebral injection.

Paravertebral anaesthesia

This is easy and satisfying to perform in lean ewes, but can be difficult in fat animals. The technique is as in cows: (i) prepare the skin over the posterior thoracic and lumbar transverse processes; (ii) identify the first lumbar vertebra; (iii) then inject 1–2 ml 2% local anaesthetic subcutaneously and into the muscle about halfway between the midline and the end of the process; and (iv) then 'walk' the needle off the front edge of the transverse process to catch T13 and inject 3–5 ml just below the level of the process. Repeat off the posterior edge of L1, then the posterior edges of L2 and L3. Another option is a sacrococcygeal epidural injection of 0.25 ml 2% xylazine, but this requires about an hour to take effect.

Caesarean technique

1. Make an incision in the centre of the left flank, being careful not to cut too deep, accidently cutting into the rumen – the abdominal wall can be surprisingly thin.
2. Palpate the uterus, identify the extremity of the lamb and carefully exteriorize, holding it through the uterine wall.
3. Incise over the extremity along the greater curvature.
4. Grasp the lamb and remove.
5. Check the uterus for more lambs – it can sometimes be difficult to reach a lamb in the other uterine horn – feel down towards the cervix before 'turning the corner' and entering the far horn. Remove any remaining lambs carefully.
6. Remove the fetal membranes if they are detached, trim and leave *in situ* if still attached.
7. Repair the uterus with an inverting suture making sure no membranes protrude.
8. Replace the uterus in the normal position.
9. Suture the flank incision in two or three layers.

The survival rate of a ewe undergoing a caesarean with live or freshly dead lambs should be high.

After delivery

Revive the lamb: (i) clear the mouth and nose; and (ii) stimulate breathing (doxapram drops are often used). For an hypoxic lamb, consider giving 10 ml of 8.4% sodium bicarbonate solution intravenously to reverse respiratory acidosis. A stomach tube can be used to inflate the lungs by positioning it in the pharynx and blocking off the oesophagus and nose. Once the lamb is breathing, return it to the dam quickly for licking.

Finally carry out the following:

- Re-examine for:
  - the presence of other lamb(s) both internally and by ballotting (it doesn't do your reputation any good to miss a lamb!);
  - damage – tearing, haemorrhage; and
  - membranes – remove gently if detached.
- Give a LA antibiotic injection.
- Consider giving NSAID, particularly if the ewe is bruised or toxic (this is not licensed for sheep, but nothing suitable is).
- Check the udder – milk off some colostrum and give it to the lamb(s) using a stomach tube; if there is none, give a substitute.
- Check and treat the lamb's navel with tincture of iodine spray or antibiotic aerosol.
- Check for defects such as entropion – correct immediately by everting the affected eyelid(s).

7

Newborn Lambs

Lamb Mortality

Many farmers accept that up to 15% of all lambs are either dead when born or die within the first few days of life (occasionally these losses rise to 50%, e.g. in abortion 'storms' or in unexpected severe weather conditions). This means that in a flock of 1000 lowland ewes anticipating 1800 lambs, over 250 lambs may be lost in the perinatal period (0–10 days).

As a bench mark, losses of:

- 5% or less indicate excellent management.
- 8% indicates above-average management.
- 10% is still better than average achieved, but is open to some improvement.
- 15% is average, with definite room for improvement.
- 20% or more is too high, indicating very poor management or exceptional circumstances.

In an average flock with losses of 15%, perinatal losses can be split roughly into:

- Pre-partum stillbirths (including abortions) – 2%. These could be due to uterine infections such as *Toxoplasma, Salmonella, Campylobacter*, enzootic abortion, Border disease, plus TBF, pregnancy toxaemia and swayback.
- Parturient stillbirths (dystocias) – 5%. If the losses are due mainly to lambing problems, then the quality and quantity of the workforce needs looking at, and also the age, breeding and feeding (e.g. large singles, and ewe lambs that are CS 4 and crossed with a Texel ram!).
- Post-partum mortality – 8%. This includes some that were said to be born dead, but actually died soon after birth and losses from neonatal diseases (see Table 7.1).

It is important to try and split lamb deaths into these three groups by fresh post-mortem examination (PME), in order to apply sensible control measures.

Post-mortem Features in Newly Born Lambs

- Decomposition – indicates pre-partum death.
- Clouding of the cornea indicates pre-partum death.
- Weight – singles should be 4.5 kg, twins 3.5 kg (but allow for breed variations). Excessive weight suggests dystocia, underweight suggests a pre-partum problem (e.g. feeding of ewe or uterine infection, or large litter size).

Table 7.1. Common diseases of neonatal lambs.

Route of infection	Organism	Disease
Oral	*C. perfringens* type B	Lamb dysentery
	Escherichia coli (non-specific strains)	Watery mouth
	E. coli (enterotoxigenic strains)	Coliform diarrhoea
	S. ser. Typhimurium and *S.* ser. Dublin	Salmonellosis
	Cryptosporidium parvum	Cryptosporidiosis
	Rotavirus	Viral diarrhoea
	Parapox virus	Orf
	Streptococcus dysgalactiae	Joint ill
Navel (also probably blood-borne)		
	Strep. dysgalactiae	Joint ill
	E. coli, Arcanobacterium pyogenes	Joint ill, meningitis
	Fusobacterium necrophorum, Staphlococcus aureus	Joint ill, navel ill
	Erysipelothrix rhusiopathiae	Joint ill, but more commonly, insidious development of polyarthritis
Castration and docking wounds		
	C. tetani	Tetanus

- Umbilical vessels – thrombus indicates that death is post-partum. In lambs dying immediately before or during parturition, the end of the artery is sharply pointed (because of contraction of the arterial wall), but if death occurred some time pre-partum, the ruptured end is square. If the end is shrivelled, death is at least some hours post-partum.
- Lung aeration indicates post-partum death (check to see if a piece of the lung floats or sinks in water).
- Presence of food in the stomach indicates post-partum death but does not rule out hypothermia if a stomach tube was used to feed shortly before death. Unclotted milk suggests recent feeding probably by stomach tube.
- Subcutaneous oedema of distal parts of the legs, tail or head indicates dystocia or hypothermia.
- Hepatic rupture, thoracic, abdominal or meningeal haemorrhage (open up cranium and spinal canal to check) indicate dystocia.
- Well-nourished lambs are born with brown fat reserves which support them during the first few days of life. In starvation, as the fat is metabolized, it is replaced by soft, red gelatinous tissue – look particularly around the kidneys and heart.
- Wear on feet indicates the lamb has lived for a few hours.

Checking for the presence or absence of the above features should enable you to decide if deaths are occurring mainly in one category. That is, if the deaths are mainly pre-partum, look for causes of death *in utero*; if at birth, then the shepherd's vigilance and availability may be inadequate; if after birth, then the lambs have probably not sucked adequately and have become cold.

Lamb Survival

As a general rule, lambs survive if they are of average birthweight, are kept warm and receive adequate colostrum, whereas those that are small, cold and fail to obtain adequate colostrum, die (Figs 7.1 and 7.2).

Fig. 7.1. A good feed of colostrum provides energy to protect against hypothermia as well as supplying antibodies.

Fig. 7.2. Triplets need special care to make sure they get sufficient colostrum.

Colostrum Store

Provision of adequate colostrum is crucial to maximizing lamb survival. Lambs need 200–250 ml colostrum/kg in the first 24 h; this is to satisfy energy demands and will provide plenty of IgG if the ewe has been correctly vaccinated.

Ewe colostrum

This is by far and away the best for a colostrum store. It can be stored in a freezer for at least a year, so surplus can be available for the next season. Although it seems a bit of a nuisance to have to milk sheep in the midst of a hectic lambing period, it is well worth taking any surplus from milky ewes. Oxytocin (1 ml intramuscularly) can be given to help letdown. The only drawback with using pooled colostrum is the possibility of disease transmission (e.g. MV, Johne's disease). This is not a problem in commercial flocks producing finished lambs, but needs considering in a breeding flock.

Cow colostrum

Cow colostrum is quite often used as a replacement for ewe colostrum. If good quality material is used (the first milking and before the calf is allowed to suck), it is an adequate substitute food (though less energy dense than ewe colostrum) and most lambs tolerate it. To provide clostridial protection with cow colostrum it is suggested that the cow can be vaccinated with sheep 8-in-1 vaccine (10 ml 3 months before calving and again at 1 month and 2 weeks before calving). If 100 ml of the subsequent colostrum (given by bottle or stomach tube) is given soon after birth, it should provide protection to the lamb for at least 3 months.

Anaemia caused by feeding cow colostrum

It is now known that some cows produce a factor in their colostrum which, as a result of an immunological reaction in some lambs, leads to the rapid removal of the recipient lamb's red blood cells together with the precursors in the bone marrow. This causes lambs of about 10–14 days old to become suddenly weak and to stop sucking; they show extreme pallor (PCV <0.10) and they urgently require a whole-blood transfusion. This is most economically and conveniently supplied by taking blood from a ewe in a large syringe containing a drop of heparin, and giving it intraperitoneally (10 ml/kg) as for a glucose injection. Valuable pedigree lambs may be given an orthodox transfusion. Corticosteroid and antibiotic injections are also indicated both to these lambs and to other lambs which may have received that batch of colostrum. A response is expected within a day, although some die and reveal very watery blood and creamy white bone marrow on PME.

It is important (although seldom practised!) to identify sources of colostrum, so that donating cows can be traced and problem ones avoided in future. Alternatively, mix colostrum from several cows before feeding to lambs to reduce the possibility of this problem occurring.

Goat colostrum

Colostrum from goats is safer than from cows; it is very similar to ewes' in composition and antibody content, if the goats have received clostridial vaccines. The only thing to watch is that it comes from caprine arthritis encephalitis (CAE)-accredited herds, certainly for flocks producing breeding stock, as this is a disease closely related to MV. For finished lamb producers only, this is not so crucial, as lambs will be long gone before there is any possible danger of disease developing.

Commercial colostrum substitutes

These are widely available and popular, but expensive and often relatively poor in both energy and antibody content when compared with ewe colostrum. However, it cannot be denied that they are handy to have available, and certainly better than nothing. They are most effectively used to 'extend' a limited amount of ewe colostrum so that lambs get some of each.

Commercial milk powders

These are fine for lambs of 1 day or older, but are not a substitute for colostrum.

Feeding by Stomach Tube

Lubricate the tube by dipping in colostrum, then introduce it into the lamb's mouth and push gently. In most cases the tube will enter the oesophagus and can then be advanced into the stomach. If inexperienced, check the length against the position of the lamb's stomach first and mark at the appropriate point. It is possible to check that the tube is not in the trachea by carefully watching the neck as the tube is passed and seeing or feeling it as it passes down inside the oesophagus. You can then be confident in giving colostrum or milk (one × 50ml syringe for a small lamb, two × 50ml for a medium lamb and three × 50ml for a large lamb). This is a quick and easy method of ensuring that weak lambs or multiple births get a good start and do not quickly succumb to hypothermia.

Early Post-partum Problems

It is worth remembering that early problems are often an expression of **pre-partum** disturbances, for example: (i) so-called soft lambs may indicate enzootic abortion; (ii) hairy shakers indicate Border disease; and (iii) ataxia/paresis/recumbency may indicate daft lamb disease, swayback or nutritional myopathy.

However, the main culprit is often inadequate feeding of the ewe in late pregnancy, which leads to:

- Short gestations, giving small weak lambs with poor protective fleeces and few brown fat reserves. Such lambs become hypothermic very quickly. They require a bit of luck with the weather, and an observant shepherd, if they are to survive.
- Poor mothering, too little colostrum and milk, leading to a vicious circle ending in hypoglycaemia and hypothermia. Surviving lambs are more susceptible to post-partum infection because of the reduced colostrum intake.
- Specific deficiencies of copper and selenium leading to swayback and nutritional myopathy.

In common with all other species, lambs are exposed to a variety of **post-partum** infections introduced orally, via the navel, or via docking and castration wounds. These infections produce a variety of clinical signs such as:

- hyperexcitability, tremors, opisthotonus, convulsions, nystagmus;
- dull, sleepy, weak, recumbency, coma;
- salivation;
- atony of gut, with abdominal distension and no faeces;
- scour;
- navel ill;
- joint ill;
- ataxia/paresis; and
- scabby lips and proliferative sores on gums and tongues.

However, farmers (and sometimes vets!) are inclined to place too much significance on the pathogens that cause these infections and therefore on therapy with antibiotics and other drugs. Actually, the main problem more often stems from: (i) the presence of susceptible lambs; (ii) lack of colostrum; (iii) a heavy weight of infection because of dirty conditions, instruments and hands; as well as (iv) inadequate observation, isolation and treatment of individual cases. Therefore prevention is paramount with good forward planning, attention to detail and continual close observation.

It is worth repeating that lambs generally survive if they are average birthweight, kept warm and receive adequate colostrum, whereas those that are small, cold and fail to obtain adequate colostrum, die.

Investigation of a Perinatal Problem

It needs time and thought and should include: (i) history; (ii) farm observations; (iii) clinical examinations; and (iv) laboratory examinations.

History

- Numbers involved (size of problem).
- Problems in previous years.
- Purchases and related incidence of disease (is the problem confined to one group? e.g. replacement ewe lambs).
- Breeding and lambing dates.
- Weather conditions throughout pregnancy.
- Feeding of ewes, vaccination and dosings (e.g. copper, fluke).
- Farmer's account of mortality, clinical signs/treatment.

Farm observations

- Cleanliness.
- Drugs and instruments.
- Shelter, isolation and warmth.
- Feed available including colostrum (do your observations agree with the farm story?).
- Shepherding quantity and quality.

Clinical examinations

- Ewes – age, CS, udders.
- Weak lambs – age, size, litter size, clinical signs, castration/docking.

Laboratory examinations

Submit plenty of fresh material (including fetal membranes). It may include paired blood samples from marked ewes for EAE, *Toxoplasma* and BDV, plus blood biochemistry (copper and glutathione peroxidise (GSHPx); gammaglutamyl transferase (GGT) and BOHB from ewes yet to lamb). If possible, submit whole carcasses of lambs rather than 'bits'.

Some Specific Post-partum Problems

These include: (i) hypothermia; (ii) watery mouth; and (iii) neonatal diarrhoea/scours.

Hypothermia

This occurs when heat loss is greater than heat production and is indicated by a body temperature lower than the normal:

- 39–40°C (102–104°F) is normal.
- 37–39°C (99–102°F) is mildly hypothermic.
- Below 37°C (<99°F) is severely hypothermic.

Newborn lambs (<5 h old) which are still wet suffer from exposure (high rate of heat loss); they usually have some brown fat reserves left and will respond to drying, warming, then feeding, by stomach tube if necessary.

Slightly older lambs (>12 h old) suffer from starvation (low rate of heat production). They have no brown fat stores left; if warmed without being given energy, they will develop hypoglycaemic fits and die. Energy can be supplied as follows:

- If the lamb can hold its head up, feed it by stomach tube, then warm, then re-feed.
- If the lamb cannot hold its head up, it is dangerous to feed (will probably regurgitate, inhale and die). Give an intraperitoneal injection of glucose before warming the lamb.

Technique for intraperitoneal glucose injection

- Use 10 ml/kg of warm 20% glucose/dextrose solution (if using 40% solution, use an equal volume of freshly boiled water to dilute and to bring it to body temperature).
- Holding the lamb by the front legs and, using a 19G × 1" needle directed backwards at 45°, inject it into the abdominal cavity just below and to one side of the navel (see Fig. 7.3).

Then put the lamb to warm, preferably in a warming box with a fan heater under wire mesh. An infrared lamp can be used but great care must be taken to avoid overheating the lamb. When the lamb's temperature has returned to almost normal and it is

The correct site is about 1.5 cm to the side and 2.5 cm behind the navel. This avoids the prepuce and penis of a male lamb. Use a 19G × 1" needle directed backwards towards the opposite hip.

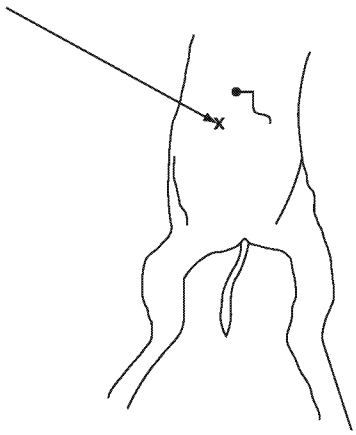


Fig. 7.3. The intraperitoneal injection site.

conscious and able to swallow, feed 150–200 ml colostrum via a bottle or stomach tube and return it to the ewe as soon as it is able to suck the ewe vigorously. If the lamb is one of twins, remove the other from the ewe and return them together when ready. If it cannot be returned to its mother, it will need to be reared artificially or mothered on to another ewe.

Note: Do **not** give an intraperitoneal injection to a lamb with scour or watery mouth.

Watery mouth (slavers, rattle belly)

These are common terms used to describe a condition which affects very young lambs (up to 3 days old). The condition is characterized by the following:

- Lambs rapidly become dull and weak, look miserable and don't get up, and they become unwilling to suck.
- They have cold, wet lips and muzzles from drooling saliva (but note that terminally ill lambs dying of other causes often drool saliva).
- No faeces (meconium) are visible and the tail is dry.
- The temperature is at first normal, but hypothermia follows.
- The abdomen is relaxed at first and looks full, but later there is tympany and tenseness which causes 'rattling' when tapped or shaken, and the distension may cause distressed breathing.
- Without treatment, they usually die within the day, but some live long enough to develop scouring and even joint ill, just to complicate the picture!
- The incidence can be frighteningly high (>20%), and is most common in twins or triplets from ewes with CS <3 and lambed indoors.

Cause

Watery mouth is an endotoxaemia which develops when lambs swallow a lot of bacteria (*E. coli* from a contaminated environment) before they have sucked colostrum. The bacteria are not destroyed in the abomasum (pH is 7 after birth and there are no colostral antibodies there to deal with them), so they pass to the gut, multiply and die, producing endotoxin. This is absorbed into the portal circulation, passes to the liver, which has limited capability to detoxify, so reaches the general circulation resulting in endotoxic shock. One early effect is slowing of gut movements with an accumulation of saliva and gas giving the bloated rattling effect.

Treatment (has to be early)

- Antibiotic injection (broad spectrum), repeated daily.
- Oral antibiotic against *E. coli* (usually a 'pump' dispenser product).
- 50 ml of glucose/electrolyte solution administered by stomach tube, repeated every few hours and increased if the lamb is not sucking the ewe. Do not feed milk until recovering.
- A small dose of NSAID such as flunixin (anti-endotoxin).

- Warm soapy-water enema (10–20 ml administered via a cut-down stomach tube).
- Leave the ewe and lamb(s) together if possible, and keep warm.

Control

Make sure lambs get plenty of colostrum early (within minutes, not hours):

- Feed the ewes well so that CS is at least 3.
- Use a stomach tube to feed colostrum quickly to at-risk lambs (weak lambs, triplets, etc.).
- Ensure good bonding (avoid moving a ewe and her lambs too soon after birth).
- Ensure the lamb(s) suck well (avoid early castration and have good shepherding).

Reduce the weight of infection:

- By keeping things as clean as possible – bedding, pens and udders.
- At birth, oral dose lambs with a suitable antibiotic. This should only be done if other methods fail as there is the possibility it will it lead to an increase in antibiotic-resistant bacteria in the long term.

Check for iodine deficiency in the ewes as there is a suggestion that this may predispose to the problem.

Neonatal diarrhoea/scours

While individual scouring lambs can be dealt with without resort to laboratory diagnosis, it is important in an outbreak or in any flock with a persistent problem with scouring lambs to establish, if possible, if there is one dominating causal organism. To do this requires submitting up to ten faeces samples (not swabs), as well as any terminally ill untreated lambs and/or fresh carcasses. This should provide sufficient information to prescribe appropriate treatment and control measures. Common causes are:

- Lamb dysentery – antiserum is no longer available; vaccinate any ewes remaining to lamb and, for the next season, implement a proper ewe vaccination policy.
- Salmonellosis – there is special advice about the risk of spread to other species, including zoonotic potential.
- *E. coli* – use antibiotic sensitivity testing to establish the appropriate treatment, administer oral rehydration therapy and ensure there is adequate colostrum intake for the remainder of lambs yet to be born. A vaccine used to be available but is no longer sold in the UK.
- Rotavirus – it may be worth considering giving all lambs soon after birth 50 ml colostrum from cows vaccinated with rotavirus vaccine.
- Cryptosporidia – you will have to rely on non-specific measures (clean pens, disinfection, good colostrum intake, etc.).

In general, it is vital to provide isolation, warmth and plenty of fluids/electrolytes and when in doubt about the causal organism, to initiate antibiotic treatment aimed at *E. coli*.

Treatment of lambs with other infections such as navel ill, joint ill, meningitis, spinal abscess

Provide vigorous and prolonged (7–14 days) systemic treatment with high-dose, twice daily broad-spectrum antibiotic. NSAID such as flunixin may help. Use local treatment to clean up navel, joint lavage, etc. where necessary.

- **A decision should always be taken about the likely outcome of sick lamb cases. If the outlook or welfare is poor, the lamb should be humanely destroyed rather than being allowed a lingering death.**

General Measures to Control Lamb Mortality

- Do not try to lamb outdoors at inappropriate times of the year and be prepared

for bad weather even during normal outdoor lambing times.
- In large flocks, plan a break in the lambing sequence (batch lambing) to allow cleaning out of the buildings and lambing pens and recovery time for shepherds.
- Observe a high standard of cleanliness – instruments, hands, bedding and disposal of fetal membranes/carcasses. Change lambing pens/areas (even re-strawing of the area is helpful) and turn out if the weather allows.
- Make sure lambs get adequate colostrum quickly. Ensure there is a good supply of frozen colostrum or a good commercial substitute.
- Provide adequate shelter (even a zig-zag of straw bales in the field is helpful).
- Reduce stocking density in pens or heavily-used lambing fields.
- Spray navels with tincture of iodine.
- Ensure early detection and isolation of sick lambs.
- Cull ewes that are thin and have faulty teeth and/or udders; and feed the others properly.
- Review the future policy for vaccination, feeding and the provision of copper and selenium.

Neurological Problems

In addition to the central nervous system (CNS) diseases of young lambs which may follow any post-partum bacteraemia or septicaemia, there are three specific conditions which originate pre-partum: (i) swayback; (ii) Border disease; and (iii) daft lamb disease. These need differentiation and while at first the clinical signs may appear non-specific or confusing, careful history-taking and clinical examination should give you the confidence to arrive at a provisional diagnosis; this usually needs biochemical and histological support before flock control measures are introduced.

Some questions of history

- Ask the farmer to describe the clinical signs.
- When did the signs first show (at birth, 1 week or older, etc.)?
- How many cases have there been so far this season?
- How many normal lambs are there?
- How many have died?
- Do any get better? With or without treatment?
- Are the affected lambs out of one particular batch of ewes?
- Are they single lambs?
- Has the farmer 'done anything' to them recently (e.g. changed pasture, weaned, dock/castrate, inject/drench)?
- Has the farmer any cases which can be examined today?
- Are the sheep exposed to ticks?
- Ask questions about the ewes – abortions, breeding, copper supplementation.

Swayback (enzootic ataxia)

This is one of the most important of the many CNS diseases of lambs, and is caused by faulty development and degeneration of nervous tissue, particularly myelin sheaths, in the brain and spinal cord of lambs whose mothers had low copper concentrations in the blood in the latter part of pregnancy. Rather than an absolute deficiency, the cause in the UK is usually an induced deficiency caused by an excess of molybdenum, sulfur and iron, although soils deficient in copper can be found in other parts of the world.

It is very important to remember that copper still heads the list of poisons in sheep, so copper supplementation should **never** be given without a clinical reason, backed up with confirmatory tests. Note that it is illegal to add copper to sheep feeds or minerals.

Diagnosis

Diagnosis is based on a combination of the history, clinical signs and laboratory tests.

HISTORY

- Swayback has been diagnosed clinically on the farm before.
- Copper has not been given to the ewes during pregnancy.
- It has been a mild winter. This influences the quality and quantity of the grass, the amount of soil eaten (minerals such as molybdenum, sulfur and iron ingested with soil 'lock up' copper) and the quantity of concentrates consumed by the ewes (although no copper is added there is inevitably a 'background' concentration of copper).
- Housing greatly reduces the risk because ewes are prevented from ingesting soil and are fed concentrates. Forecasts of likelihood of swayback, based on weather, are published in the farming and veterinary press.
- The grazing is lush. Improved, fertilized and reclaimed pastures with single-sward grasses and high molybdenum and sulfur levels are particularly likely to give rise to swayback.

CLINICAL SIGNS Signs may be present at birth, within a few days of birth or not appear until lambs are several weeks old.

- Congenital – lambs at or within a few days of birth show varying degrees of ataxia, paresis and inability to stand. Some may be born dead, or die within a few hours. Some will be able to suck if permitted and most appear alert and aware. The signs are, at best, only suspicious and can easily be confused with the other CNS disturbances of young lambs. It has been shown that lambs in flocks suffering from subclinical copper deficiency (i.e. no obvious cases of swayback) have increased lamb mortality rates. The main differential diagnosis for lambs of a few days to a few weeks is spinal abscess and joint ill.
- Delayed – lambs a few weeks old, which were normal at birth, develop varying degrees of hind-leg weakness, ataxia (swayback) and paresis. The signs are more obvious when the lamb is chased about and turns quickly. It looks like a spinal rather than a brain condition and the lambs can suck and graze normally; some will fatten because they learn how to cope with the disability.
- Acute delayed – rapidly developing signs in lambs a few weeks old which die within a few hours. This type is uncommon.

Incidence

This will depend upon the control measures adopted and the weather but it is common for a swayback-prone farm to have a few years with very few cases, followed by a calamitous 'storm' (50%). Fortunately, the advent of housing and safer copper supplementation has reduced these incidents.

Pathology

Material required for diagnosis should include:

- Formalinized brains and brainstems.
- At least ten heparinised blood samples (green tubes) from pregnant ewes that have not recently had supplementary copper. Concentrations below 9 µmol/l suggest a copper deficiency problem.
- Liver samples if available – copper concentrations below 80 mg/kg DM are suspicious.
- Herbage analysis – the following are suspicious: Cu – below 5 ppm DM, Mo – above 1 ppm DM, S – above 0.20% DM.

Treatment

Because swayback can be a progressive condition, it is worth treating those young lambs which are not too severely affected with a single injection of copper (no more than 5 mg) in an attempt to stop the condition getting worse, although no improvement should be expected. Badly affected lambs should be euthanized.

Control

1. During an outbreak – all the remaining pregnant ewes (unless the breed is susceptible to copper toxicity, e.g. Texel) should

be treated with a suitable copper preparation, together with those healthy lambs already born out of ewes not previously dosed, in order to prevent delayed swayback.

2. Next year – consider housing the ewes during the last half of pregnancy, thus removing them from the source of the problem. If housing is not practicable, dose all ewes in early pregnancy with copper (capsules containing copper oxide needles are the safest) but also then remove other known sources of copper such as licks or minerals that contain copper and proprietary cattle and pig concentrates.

If there is a problem of ill-thrift, poor quality fleece and multiple fractures in lambs due to copper deficiency, then the lambs also require dosing, but not before other causes have been eliminated and copper deficiency established.

COPPER PREPARATIONS

- **Beware of copper toxicity! Some breeds, especially continentals, are very susceptible.**
- **Check very carefully for the recommended dose of particular product to be used.**

Injectable (copper heptonate) and oral preparations (copper oxide) are available. The safest and arguably the most reliable preparations are in the form of capsules containing copper oxide needles, delivered orally by a plastic gun. Ewes are dosed once in mid-pregnancy and lambs as required when they are over 5 weeks old. A combined copper, selenium and cobalt oral soluble glass bolus is also available.

Short-acting drenches are sometimes used, but they require repeat dosing and risk toxicity. Copper sulfate on its own (1.0g in 30ml water) is still used prophylactically by farmers, but it is a risky material to have about on sheep farms.

To avoid **toxicity**, either within hours of injection or weeks after dosing:

- Make sure copper is necessary.
- Reduce the dose in small sheep.
- Remove any other obvious source of copper, particularly if the animals are housed and fed concentrates.
- Treat ewes with care, and follow makers' recommendations.

Individuals and breeds vary in susceptibility to copper deficiency and toxicity. For example: (i) Scottish Blackface and Swaledales are more prone to deficiency; (ii) Texels and other continental breeds are prone to toxicity; and (iii) the North Ronaldsay, whose natural diet is seaweed which is very low in copper, is extremely susceptible to toxicity, often succumbing on diets which would be deficient to other breeds. A change in breed might be considered, or an alteration in the lambing date, or special precautions for selected breeds, or cull those ewes that produce swayback lambs (there is familial variation as well as breed variation). Remember that high sulfate and molybdenum levels in the diet are known to decrease copper 'availability', hence the term 'conditioned' copper deficiency, and hence also the use of ammonium tetrathiomolybdate and sodium sulfate in attempts to control chronic copper toxicity.

Border disease (hairy shakers)

This is a congenital disease, first reported from the English–Welsh border counties but since recognized throughout the world. It is caused by infection with a pestivirus (BDV) serologically related to bovine viral diarrhea virus (BVDV). Antigenic differences can be recognized between BDV and BVDV, and sheep strains will infect cattle and vice versa, though it is unclear how much spread occurs between the two species under natural conditions. Sheep which are immune to one strain are susceptible to the others.

Clinical picture

BDV infection produces a number of clinical signs, though they do not all occur in every flock. One or more of the following pictures may be seen:

- A number of lambs showing a typical long hairy coat, some of which are also ataxic with fine rhythmic tonic/clonic contractions of skeletal muscles ('hairy shakers'). These signs are usually evident at birth, although in the naturally long-woolled breeds, the abnormal birth coat is not obvious. The incidence of Border disease lambs is variable but 'storms' are reported. Gimmers produce the highest percentage of Border disease lambs, indicating progressive immunity.
- Many affected lambs survive for a time, but their growth rate is poor and they appear stunted with domed heads and lag behind the rest of the flock. In addition, persistently infected lambs occur, many of which do not show the early signs, but some show chronic unthriftiness and scouring. Some survive to breeding age, and though their fertility is reduced, they can produce infected lambs in successive pregnancies. Persistently infected rams can transmit virus in semen.
- There is a high prevalence of barren ewes, abortions or even of just weak lambs. This means that the disease may show as an abortion and infertility problem rather than as a disease of young lambs.

Pathogenesis

- Infection of healthy non-pregnant sheep produces no obvious clinical signs and gives rise to the production of neutralizing antibodies and an immune sheep. It is 'antibody-positive, virus-negative'.

Infection of pregnant ewes leads to a similar course of events in the ewe, but the virus crosses the placenta and leads to the various clinical pictures, depending on virus strain and breed of sheep, but most importantly, on the stage of gestation:

- Infection prior to 60 days, at a time when the fetus has no ability to mount an immunological response, will result in fetal death in a high proportion of cases, leading to resorption and barren ewes or mummification and abortion. Virus strains vary in virulence and these early infections may allow some infected fetuses to survive with virus in virtually every organ. Infection of the CNS leads to myelin deficiency and 'shaker' lambs and infection of the hair follicles leads to 'hairy' lambs. Weak or even apparently healthy infected lambs may be produced. All these lambs will be immunologically tolerant to BDV and 'antibody negative, virus positive' at birth, and although they will acquire antibody in the colostrum, this will wane and they will be 'virus positive', probably for life. These lambs are persistent carriers.
- If infection occurs after about 85 days, the fetus mounts an immunological response and destroys the virus. Most will be normal at birth and will be 'antibody positive, virus negative', like the dam.
- If infection occurs between 60 and 85 days, when the fetus is becoming immunologically competent, either of the above responses may occur with either clinical outcome.

Epidemiology

Most flocks become infected by the purchase of persistently infected lambs as breeding replacements. It has been suggested that, rarely, infection may be introduced by pestiviruses from other species such as cattle, goats or deer, or even by a contaminated live vaccine.

Diagnosis

The following indicates the tissues needed for laboratory confirmation of clinical diagnosis:

1. From 'hairy-shaker', weak, scouring lambs or suspect persistently infected sheep – clotted and heparinised blood (red and green tube) from live lambs for virus isolation and serology. In dead animals, virus can be detected by immunofluorescence and virus isolation from fresh tissues put in transport

medium (e.g. kidney, spleen, thyroid; brain and spinal cord in calcium formolsaline for histology).
2. From aborting or barren ewes – clotted and heparinised blood for virology and serology, and placenta for virus isolation.

Treatment and control

There is no treatment and there are dangers in attempting to support weak or hairy shaker lambs which will be excreting virus.

- Ideally, a closed flock should be maintained or replacements obtained from a flock which is serologically negative (this may form part of a sheep health scheme in the future). Maintaining replacements as a separate flock from tupping to lambing will enable monitoring of lambing to be done.
- In a flock which has recently had Border disease for the first time, the sheep suspected of introducing the infection may be identified as 'antibody negative, virus positive' from blood samples. They and their lambs should be culled before the next tupping season.
- In an endemically infected flock, all breeding ewes should be deliberately exposed to infection 2–3 months before mating by housing in close contact with persistently infected sheep for 3–4 weeks.
- A vaccine has been developed for BVD in cattle, so it may eventually be produced for sheep.

Other congenital neurological problems

Several neurological diseases affecting newborn or young lambs with a particular breed association have been recognized.

- Hydrocephalus (Dandy Walker malformation) has been seen in Suffolks – this causes dystocia so needs to be considered if dealing with this breed. Sometimes the lamb's skull is so soft that it can be crushed, aiding delivery; sometimes a caesarean may be necessary. It is fatal.
- Daft lamb disease has been seen in several breeds – lambs are born with cerebellar abnormalities such as head nodding, ataxia and tremors and, as the name suggests, may appear mentally stupid, having difficulty finding the ewe's teat.
- Cerebellar abiotrophy has been seen in Charollais lambs – cerebellar signs appear during the first weeks of life with ongoing deterioration.

Affected lambs should be euthanized and diagnosis may require PME and histological examination of the brain. Breeding records should be carefully examined since these diseases are often familial.

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8

The Lactating Ewe

Ewes rearing twins almost invariably have an energy deficit in early lactation so will lose weight. This is not a problem, providing they were in good condition at lambing and do not become extremely thin, as they will regain body condition after weaning. Ewes rearing triplets usually need supplementary food, even if grass supply is good, and are prone to mastitis problems because of hungry lambs competing for the teats and causing teat lesions (Fig. 8.1). These ewes should be managed separately with lamb creep feed supplied so that the demand on the ewe is reduced as soon as the lambs start eating.

Particular problems of the lactating ewe are:

- mastitis;
- teat lesions; and
- hypomagnesaemia.

Mastitis

Our knowledge of sheep mastitis still lags a long way behind that of the disease in cattle. It mostly remains a sporadic problem, with an incidence of 4–5% in lowland flocks, lower in hill flocks. The sector where it is important is in dairy sheep, in which knowledge about cattle mastitis can be usefully applied. Research into mastitis in dairy sheep in countries such as Greece, where they are an important part of agriculture, is advancing knowledge in this sector.

Mastitis in the non-dairy ewe is commonly seen at three stages in the production cycle and is usually irreversible:

1. Acute, often gangrenous, at peak lactation – may be fatal; if not, requires life-saving treatment, although one could argue about the merits of this, as the animal should be culled when recovered.
2. Chronic, seen after weaning or at the pre-tupping examination; requires culling.
3. Blind teat at lambing – this is same category as (2), but was not detected at that time. Also eventually requires culling but would rear a single lamb if the other half is normal.

Organisms commonly involved are:

- *Staphylococcus aureus* which is found on the skin.
- *Mannheimia* (*Pasteurella*) *haemolytica* which is found in the nasopharynx of healthy lambs.

Singly or together, these bacteria cause 80% of cases (both acute and chronic forms) and invade the udder via the teat canal especially when the skin of the teat is damaged. Other bacteria sometimes found include

Fig. 8.1. Ewes at peak lactation are vulnerable to mastitis.

streptococci, *E. coli*, coagulase-negative staphylococci, with occasionally *A. pyogenes* and *C. perfringens*.

Other causes of mastitis include:

- MV – this causes an indurative mastitis.
- *Leptospira hardjo* (sometimes also called *Leptospira interrogans* serovar *hardjo*) has been associated with sudden milk drop.
- *Mycoplasma agalactiae* is important in the Mediterranean area, causing contagious agalactia, but this does not occur in northern Europe.

Acute mastitis

This occurs most commonly early in lactation, when the ewe is at peak milk production, usually suckling twins. It is difficult to know precisely what triggers an attack, but it may be associated with movement on to lush pasture, or a period of bad weather. Shorn or crutched ewes are particularly vulnerable shortly after turnout, perhaps because the udder is exposed to chilling. Teat lesions caused by orf or staphylococci (staphylococcal dermatitis) also predispose to mastitis.

The clinical picture is dramatic and typical: the ewe is obviously ill with a swollen udder (usually only one half), she walks stiffly and the lambs appear hungry. The teat and a portion of the udder skin often become cyanotic and cold, and oedema extends along the region of the mammary vein. Death may occur within hours so the ewe may be simply found dead. If the ewe survives, the necrotic udder and, commonly, the skin and tissue surrounding the milk vein, slowly slough off with an extensive loss of abdominal skin, leaving tubes of mammary ducts and blood vessels exposed. Healing then takes many weeks and the ewe loses much condition. It may be necessary to tie off and amputate these granulating tubes to speed healing and also to take measures to avoid flystrike.

Treatment

Consider first whether treatment is justified on both welfare and economic grounds. If so, vigorous systemic treatment is indicated immediately, but the local pathological change is often already irreversible. Give a high dose of broad-spectrum antibiotic, repeated twice daily until the ewe shows improvement (a day or so) or dies. Fluids (1–2 l) given orally or intravenously and a NSAID are also indicated. Strip the secretion out of the gland each time the ewe is treated. Drainage may be aided by removal

of the teat if it is gangrenous. Intramammary tubes are an optional extra. Ensure the ewe is comfortable and has tempting food and water nearby. Her lambs will need to be fed with milk replacer unless old enough to manage on creep feed.

Control

Control is difficult as predisposing factors are often not clear. There is evidence that infection does not arise from pre-existing subclinical intramammary infection, but from opportunistic invasion shortly before clinical events; therefore dry ewe therapy (DET) is unlikely to be effective.

The incidence varies between seasons, perhaps because of changing management and climate, but it is usually of the order of 1%; this level usually makes the condition a nuisance to the farmer and does not justify whole-flock control measures.

Some suggestions are:

- If the ewes are too thin (CS <3), consider increasing the feed and so ensure adequate milk for vigorous lambs.
- Attend to teat sores – pustules, orf and sucking wounds; this may mean antibiotic injections and even weaning the lamb(s) if they are old enough.
- Avoid turning out in cold wet weather and/or ensure shelter.
- Ensure there is clean, dry bedding at lambing ('squelch' test!).
- Ensure proper clostridial vaccination. There is no evidence that *Pasteurella* vaccination is protective against mastitis. If orf is a problem, it is worth considering vaccination, but it must be done not less than 7 weeks pre-lambing.

Chronic mastitis

This is usually discovered before tupping at a time when the ewes are selected for breeding or culling (e.g. because ewes are too thin, have problem teeth, are barren, or have a known disease). One half of the udder, less commonly both, is lumpy and distorted with palpable fibrosis or chronic abscesses. The flock prevalence varies between 1% and 15%. If cases are not obvious at this time, they become apparent at lambing and probably account for many of the ewes which lamb-down with only one functional gland, and for the problem of neonatal disease that this leads to. It is uncertain when infection occurs, but it is assumed that it is at weaning time, because LA antibiotic, introduced then, is known to reduce the disease incidence, very similar to the dairy cow story.

Control

This is difficult, but consider the following:

- DET at weaning: this is a two-person job and the teats need to be cleaned before infusion. It is not necessary to insert the nozzle into the teat – just line up as in heifers.
- It seems a reasonable idea to dip or spray teats with a commercial cow teat dip at the time of infusion.
- Place ewes on poor pasture at weaning and keep them well away from the sound and sight of their lambs, to reduce milk production. Do not restrict water intake.
- Mark cases for culling.

Teat Lesions

These commonly predispose to mastitis. Additionally, affected ewes may be reluctant to let their lambs suck, so may be a cause of ill thriven or dead lambs.

The main causes are:

- *Staph. aureus*; and
- orf.

Staphylococcal infection

This produces very painful ulcerated and swollen lesions at the base of the teat, probably triggered by damage from the sharp incisors of lambs when sucking. It is crucial to treat these as soon as possible, otherwise the consequences of mastitis and starving lambs follow. Aggressive treatment with

injectable antibiotic and NSAID, plus local application of antiseptic and local anaesthetic gel helps. While at first sight removal of the lambs might seem a good idea, it is better to allow them to continue sucking (aided by application of anaesthetic gel if available), restraining the ewe if necessary. This may seem rather brutal but lambs are the most effective way of emptying milk from the udder and if recovery is speeded up by the above treatments, it may be possible to have a good outcome for both ewe and lambs.

Orf infection

This is more of a problem, since the viral infection has to run its course. It is often complicated by lambs also having infection on their mouths. Local and parenteral antibiotics help to control secondary infection, but there is no quick cure. If this is a real problem, for the future, vaccination of ewes before the last 7 weeks of pregnancy may be appropriate.

Hypomagnesaemia

As described elsewhere, this is most usually seen in ewes at peak lactation suckling twins (Fig. 8.2). The stress of lactation, combined with grazing low-fibre, rapidly-growing grass, often fertilized with nitrogen, trigger the disease. Most often, ewes are found dead, but may sometimes be seen collapsed and in convulsions. See Chapter 6 for treatment and control.

Fig. 8.2. Ewes suckling twins or triplets are particularly at risk of hypomagnesaemia.

9

Growing Lambs

A common complaint made by farmers is that a significant number of the lambs are not growing as rapidly as expected (Figs 9.1 and 9.2). The growth rate of lambs on lowland farms being raised for slaughter should be 250–300 g/day though single lambs and lambs being raised for sale as rams will manage higher growth rates than these. Although in the UK it is rare for farmers producing prime lambs for slaughter to weigh their lambs regularly and so the growth rates will not be known, these can be estimated by looking at lamb sales data and working out the times taken to achieve slaughter weight. For example, if we assume that the lambs are born in March and slaughtered at around 40 kg live weight in July, the weight gain will be 300 g/day whereas if they are sent for slaughter in September, the weight gain will only be 200 g/day. Many lambs grow well until some specific incident occurs, the timing of which will help in diagnosis. Although many causes are possible, the most common reasons for a significant proportion of the lamb crop being thin are coccidiosis and parasitic gastroenteritis (PGE) and in some areas, mineral deficiencies, particularly of cobalt, which also predisposes to parasitic problems. It is highly likely that the poor weight gain will also be associated with scouring.

Coccidiosis

The incidence of coccidiosis has increased steadily over the last 15 years in the UK, probably due to increased intensity of production, and is recognized as a considerable hazard of lowland sheep production throughout the world. Several species of *Eimeria* have been described which are specific to sheep, and their pathogenicity differs considerably. Many of them produce few if any clinical signs, whereas two species (*Eimeria ovinoidalis* and *Eimeria crandallis*) may cause severe disease, characterized by diarrhoea, weight loss and death. It is a specialist task to identify these species so oocyst counts do not usually differentiate the species involved.

History

The majority of outbreaks, which involve a high proportion of the lambs, occur in animals of 4–8 weeks old which have been housed since birth, or grazed on constantly-used pasture at high stocking rates.

Clinical signs

The first signs are that the farmer may recognize that the lambs have lost 'bloom' and

Fig. 9.1. These Herdwick lambs look to be thriving well.

Fig. 9.2. These lambs at weaning are rather a mixed bunch and show quite a variation in size.

show an 'open' fleece. Some animals may have slight diarrhoea with soiling of the wool below the anus. These signs extend to considerable numbers of the flock and weight gains are reduced, followed by weight loss. Severe untreated cases proceed to extensive scouring, dehydration and death, which in infections with *E. ovinoidalis*, occurs 14–21 days after infection. Treated recovered lambs often continue to make poor weight gains due to slow repair of the intestinal lesions, resulting in villous atrophy, with consequent malabsorption of nutrients.

Epidemiology

The ewes act as a source of oocysts, and sporulation occurs in 2 or 3 days. Oocysts remain viable for up to a year. Infections are built up by multiplication through lambs and it is often later batches of lambs which show clinical disease.

The peak incidence at 4–8 weeks of age was believed to be due to the fact that one cycle had to take place in the lambs, taking 2–3 weeks, in order to build up a heavy environmental contamination with oocysts. Disease was believed to result from the multiplication of this second cycle in the intestine before an effective immunity had developed. It is now known that lambs are not susceptible to coccidial infections (with *E. ovinoidalis*, the most pathogenic species) very early in life and that susceptibility increases up to about 4 weeks of age. If heavy infections are encountered early in life, clinical signs are not seen (due to passive transfer of immunity), active immunity is stimulated and the lambs are less likely to show clinical signs of coccidiosis at 4–8 weeks old. After this age, most lambs have encountered infection and develop a strong immunity though in exceptional circumstances, coccidiosis may occur in older lambs.

Diagnosis

This is based on clinical signs and history. Oocyst counts are not reliable since it is possible to see clinical coccidiosis associated with developing sexual stages, before peak oocyst production occurs, and counts of up to one million oocysts/g in a faecal sample can be seen without clinical signs associated with the non-pathogenic species. It has been stated that it is easier to arrive at a diagnosis of coccidiosis if you don't possess a microscope! Nevertheless, it is often helpful to examine a number of faecal samples (say ten) when counts of over 100,000 oocysts/g in some samples, together with suggestive clinical signs and history, warrant treatment for coccidiosis. In lambs out at grass, *Nematodirus battus* infection should be considered since it occurs at almost the same time. It has been shown that concurrent infection with *E. ovinoidalis, E. crandallis* and *N. battus* causes a much more severe disease than any of these by themselves.

Treatment

There are two products which are effective in the treatment of clinical outbreaks of coccidiosis in lambs.

1. Diclazuril, which has been available for a few years, is formulated as an aqueous suspension given orally by drenching gun. All lambs in the affected group should be given a single dose. The drug is effective against all stages of the two pathogenic species of coccidia and under field conditions allows the development of immunity. It reduces oocyst excretion significantly. It has a zero meat withdrawal period.

2. Toltrazuril, which is a relatively new drug, is formulated as an oral suspension given by drenching gun as a single dose. The drug is effective against all stages of the two pathogenic species and allows the development of immunity. Although the drug can be used to treat clinical coccidiosis, it is recommended that in order to obtain maximum benefit, it should be given before the onset of clinical signs and thus is more suitable for prevention and control of coccidiosis. It markedly reduces oocyst production. The meat withdrawal period is 42 days but this is not usually of importance unless early, light lamb production is planned.

Control

On many farms, it is known that coccidiosis occurs on certain well-used pastures each year and toltrazuril and diclazuril may be used to prevent such outbreaks. All lambs should be treated at the normal dose rate at around 3 weeks of age and in conditions of heavy challenge, the treatment could usefully be repeated 3 weeks later.

Very occasionally, severe scouring has followed diclazuril treatment which has been shown to be associated with freezing of the aqueous suspension and the active ingredient coming out of suspension resulting in under-dosing and a failure to control the coccidial parasites.

Parasitic Gastroenteritis (PGE)

PGE is the most important cause of thin, scouring lambs from as early as 2 months of age up to about 1 year old in all sheep-producing countries of the world. During the first year of life, a strong immunity develops to most worm species (*Haemonchus* is an exception) which not only means that clinical signs are rarely, if ever, seen in older ewes but also means that sheep may ingest infective larvae (L_3, i.e. third-stage larvae) but the life cycle is not completed and egg counts are very low for most of the year. The important exceptions to this rule are the periparturient rise in faecal egg count in ewes and the frequent observation that adult rams may have high egg counts.

The important worms in lambs in the UK and northern Europe are *Ostertagia* species (in sheep, renamed *Teladorsagia* spp.) in the abomasum and *Trichostrongylus* and *Nematodirus* species in the small intestine. Explosive outbreaks of *Haemonchus* infection in the abomasum are seen in July and August following suitable weather conditions in lambs and ewes and a more chronic form of haemonchosis has been described in periparturient ewes.

Haemonchus is the most important worm in tropical and subtropical countries and is of immense importance in the southern hemisphere countries, including Australia, New Zealand, South Africa and the countries of South America. It is the important nematode which above all others rapidly develops resistance to anthelmintic drugs.

Although *Haemonchus* is recognized as the most important worm of warmer climates and was thought to be of occasional importance only in southern England, it is now found widely across the UK including Scotland, perhaps associated with climate change.

Clinical signs

- *N. battus* causes severe scouring, loss of bloom, dehydration, loss in condition and death in lambs, typically in May and June but increasingly in the autumn also. Ewes are not affected and immunity develops rapidly in the lambs.
- *Teladorsagia, Trichostrongylus* and *Nematodirus filicollis* contribute in different proportions to PGE during the rest of the year, usually commencing in July and continuing into the late winter with chronic scouring and unthriftiness. Subclinical parasitism characterized by reduced weight gains without significant scouring is also of great economic importance.
- *Haemonchus*, which differs from the other worms in being an active blood sucker, leads to anaemia, loss in condition and death, without scouring.

Life cycles and epidemiology

Nematodirus eggs, which can be distinguished from those of other worms in that they are much larger and even distinguishable between species, develop slowly with the moults to the infective L_3 stage occurring within the egg in a minimum of 2 months, even under favourable conditions. The typical life history of *N. battus* involves only one generation every 12 months with the egg containing the L_3 overwintering on the pasture.

After being exposed to a low temperature over the winter, the L_3 hatch when the temperature rises to above 10°C, thus resulting in a heavy pasture infestation over a short period in the spring. An annual nematodirosis forecast is published in the UK by UKVET (available at: www.nadis.org.uk). If the mass hatch is early (e.g. February/March), most spring-born lambs will not ingest many L_3, since the larvae will have died off before the lambs are consuming much grass. If, however, there is a late winter and the mass hatch does not occur until May, severe disease is likely since the older lambs will be eating a considerable quantity of grass. The cycle is essentially a lamb-to-lamb annual infection. It should be noted that though this typical life cycle is still important, *N. battus* has developed the ability to complete its life cycle without the fall in temperature associated with winter and is becoming an important cause of PGE in autumn. This appears to be due to the fact that hatching is more complex than was thought and it has been shown that hatching is inhibited at an upper temperature of around 18°C. Selection factors such as climate change may be responsible for the increasing proportion of *N. battus* eggs which are hatching in the autumn. It also appears that different strains have different hatching patterns which may explain differences in nematodirosis on different farms.

With *Teladorsagia* and *Trichostrongylus*, the eggs, which are indistinguishable from each other (or from *Haemonchus* and a number of other trichostrongylid worms of little pathogenicity), develop to a first-stage larva (L_1) which hatches. The L_3 is the infective stage and the time taken for an egg to develop to L_3 varies with the environmental temperature from as little as 2 weeks to as long as 4 months. Eggs are produced for a few weeks by ewes of all ages around lambing time and during lactation by the 'activation' of inhibited fourth-stage larva (L_4) in the mucosa and by completion of the life cycle in these ewes whose immune response has been reduced. In addition, eggs are produced by lambs which ingest overwintered L_3. These factors result in the well-known 'telescoping' of larval development so that eggs passed in March–June all reach the infective stage in July as the early eggs take months and the late eggs weeks to become L_3. Infective larvae overwinter on the pasture quite happily but most die off rapidly during May and June. This larval peak in July onwards is the main challenge to lambs which are nearing market weight at this time and ingesting much grass as the ewe's milk supply dries up.

Haemonchosis usually occurs when there has been a spell of very warm humid weather in July and August so that a high proportion of the enormous numbers of eggs which are passed by *Haemonchus* females develop rapidly to the infective stage, ingestion of which, together with a poorly developed immune response to this worm, results in explosive outbreaks of disease.

Diagnosis

PGE should be suspected from the clinical signs and the history of a high proportion of the lambs being involved, and can usually be confirmed by faecal egg counts on a number of affected lambs. Egg counts are usually over 1000 eggs/g of faeces though, with *N. battus* in particular, severe scouring can occur just before the worms become mature and produce eggs. *Haemonchus* infections produce high egg counts of 5–10,000 eggs/g. Worm counts on dead or killed affected lambs in the abomasum and the first 10 m of the small intestine will usually yield counts of 50,000 *Teladorsagia* or *Trichostrongylus* or more, but many may be immature. *Haemonchus* counts in the abomasum are generally around 1000 or more.

Treatment

Early recognition of clinical signs and particularly the use of faecal egg counts allow the treatment of affected lambs before severe lesions of villous atrophy have developed which can render treatment ineffective. Villous atrophy, resulting in continued clinical signs, can also result if the lambs are

left on a heavily contaminated pasture which may cause the farmer to believe that this is due to anthelmintic resistance.

Anthelmintics for PGE

Although there are an enormous number of proprietary anthelmintic drugs available to farmers, which gives rise to confusion, they fall into five main chemical groups: (i) benzimidazoles (BZ drugs); (ii) imidazothiazoles and tetrahydropyrimidines (levamisole and morantel, so these are known as LM drugs); (iii) avermectins and milbemycins (macrocyclic lactones or ML drugs); (iv) amino-acetonitrile derivatives (AADs); and (v) spiroindoles. The groups are discussed below with examples given of some of the individual drugs in each group for illustration. The first three groups were the only anthelmintics for 25 years, until recently when two new anthelmintics were described, each belonging to a new group. It is probable that more drugs belonging to these two groups will become available in the future. It is important that the data sheets for each drug should be consulted since they have important differences in: (i) the range of activity; (ii) residual action; and (iii) withdrawal periods for meat and milk.

BZ drugs

- These are the oldest and most widely used drugs for the treatment and control of PGE.
- The first member, thiabendazole, no longer available, was introduced in 1961 and is the parent drug from which the large number of benzimidazoles has been derived. They are known as 'white drenches'.
- All benzimidazoles (except triclabendazole – see fluke, Chapter 4) are effective against all stages of the normal life cycle of BZ-susceptible worms which cause PGE (e.g. oxibendazole); some are also effective against lungworms (e.g. mebendazole); and some are effective against inhibited fourth-stage larvae (L_4), involved in Type II ostertagosis (e.g. fenbendazole).
- They are available in a wide range of formulations including a drench suspension, feed pellets, feed block or powders. Oral preparations containing cobalt and selenium also are available.
- Anthelmintic resistance is widespread to benzimidazoles and resistance to one means that the worms will be resistant to all benzimidazoles so changing from one white drench to a different one is not useful, which is not always appreciated by farmers.

LM drugs

- The major anthelmintic is levamisole, the l-isomer of tetramisole. There are dozens of proprietary formulations.
- Levamisole is effective against all stages of the life cycle of worms responsible for PGE, including inhibited larvae and against lungworm infection.
- Levamisole is available as feed granules and as a solution, which may be given orally or by subcutaneous (SC) injection.
- Morantel is a related drug, which is effective against BZ-resistant nematodes but its range of activity is not wide.

ML drugs

- Until 1996, the one anthelmintic in this group was ivermectin. An injectable or oral preparation, it is effective against all stages of worms causing PGE, including inhibited larvae, lungworm infection and against sheep scab mites.
- Moxidectin is available as a drench and an SC injection in sheep and is effective against all stages of worms causing PGE, including inhibited larvae, and against lungworm. The injectable form is active against sheep scab and itch-mites (*Psorergates*). It has a marked persistent effect, the 1% injectable solution possessing residual activity against *Teladorsagia* and *Haemonchus* of 5 weeks. There is also

a 2% LA formulation which is active against *Teladorsagia* and *Haemonchus* for around 100 days but which has a very long withdrawal period exceeding 100 days.
- Doramectin is available as an intramuscular injection in sheep. It has a wide range of activity, including sheep scab and itch-mites and has residual activity of 3–6 weeks.

AADs

- This is a novel class of drugs with the one marketed anthelmintic, monepantel. This drug acts by binding to a specific receptor only found in nematodes and so possesses a high safety margin. It is available as an oral drench with a special gun.
- It kills all stages of all the important worms and is particularly useful on farms where resistance to the three older groups of anthelmintic has developed. It has the advantage of a short (7 day) withdrawal period.

Spiroindoles

- This is the most recently developed group of anthelmintics and at present, the one marketed anthelmintic is derquantel. It has limited availability at present to New Zealand (but will doubtless become more widely available) as a combination oral drench with abermectin, thus combining its unique mode of activity with the wide range of activity of an ML drug.
- It is highly effective against adults and L_4 larvae of all important worms including nematodes which are resistant to other anthelmintic groups. In addition, it is active against lungworm, nasal botfly and itch-mite.

(Some other drugs, which are mainly used against fluke infections, are also effective against the blood sucking nematode, *H. contortus*. Examples are closantel and nitroxynil. They have been used in worm-control programmes against BZ-resistant strains of *H. contortus* in Australasia.)

Control and anthelmintic drug resistance

Many species of nematodes have become resistant to one or more of the major anthelmintic drug groups and surveys indicate that these strains are widespread throughout all sheep-producing countries including the UK which must be taken into account in formulating control programmes. The major nematodes involved are commonly *Haemonchus* and *Teladorsagia* but, at present, rarely in *Nematodirus*, though resistant strains of this worm have recently been recognized. Recent surveys have found widespread BZ resistance, less widespread resistance to levamisole and, on a small number of farms, resistance to all three of the older anthelmintic groups. It is recognized that many of the methods which were used to control PGE selected for resistant worms and a re-appraisal of control strategies has taken place and given rise to recommendations under a programme called SCOPS (Sustainable Control of Parasites of Sheep).

It is believed that all populations of worms contain a small proportion of individuals that are more resistant to anthelmintics than the rest and progressive selection of these worms in preference to the susceptible worms eventually results in a population which is largely resistant. While this may take years, depending on the selection pressure and the genetic basis of drug resistance, once established it is impossible to reverse. The SCOPS programme seeks to reduce the selection pressures so that the population of worms remains mainly susceptible to anthelmintics. In particular, it emphasizes the importance of the parasite population which is not subject to selection pressure which is mainly the stages on the pasture. This population is said to be '*in refugia*'. The principle behind the SCOPS recommendations is that this population should be kept as large as possible so that parasites with resistant genes are regularly diluted with those with susceptible genes.

The traditional control schemes which exert a strong selection pressure are:

- The Weybridge 'dose and move' in July – in this, all lambs are dosed in July

and moved to a clean pasture such as aftermath after hay or silage. Clearly, the worms remaining after the dose are the resistant population which results in the clean pasture being contaminated with a resistant population only.

- The same is true for all 'dose-and-move-to-clean-grazing' programmes, including dosing housed ewes at lambing and putting on a clean pasture, such as might be provided by rotational grazing with cattle.
- Early-season dosing of ewes and lambs within the pre-patent period or with drugs possessing residual activity in order to prevent the July peak of larval development – this method is very effective at exerting a strong selective pressure. It means that any worms which do develop are resistant so, as susceptible overwintered L_3 die off, the (small) July peak consists solely of resistant worms. This is then built up over years to a largely resistant population.

The SCOPS programme seeks to maintain a population of L_3 which are largely susceptible, so that anthelmintics remain effective for when they are really needed. **It should be recognized that this means that worms will not be as well controlled as ideal and subclinical disease may occur. It is also possible that clinical parasitism will result if the system is not monitored carefully which emphasizes the need to carry out periodic egg counts to anticipate this possibility.**

The main features of the SCOPS programme (slightly amended) are as follows:

1. Avoid importing resistant worms with purchased sheep by quarantine procedures. Treat all purchased sheep with full doses of monepantel and moxidectin, consecutively but not mixed, and keep the animals on a concrete yard for 24 h with food and water. Put the treated sheep on to a contaminated pasture to allow a susceptible population to develop. Do not put faeces produced during the holding period on pasture for a year.
2. Test for resistance annually by the use of the Faecal Egg Count Reduction Test. In principle, give anthelmintic to a group and do egg counts 7–14 days later.
3. Give anthelmintics effectively by checking the accuracy of the drenching gun, weigh sheep and dose for the heaviest.
4. Use anthelmintics only when really necessary. For example, do not dose ewes at tupping as most ewes are immune. Since it has been shown that dosing ewes at lambing once and putting on dirty pasture, containing overwintered L_3 is no better than not dosing at all on the overall periparturient egg count, do not dose or dose only a proportion of the ewes, such as those in poor condition.
5. Choose anthelmintic thoughtfully. It is usually best not to use fluke-roundworm combinations as the best times for dosing differ for the two groups of worms: use BZ drugs for nematodirosis in May–June as resistance is rare; use closantel if haemonchosis is the problem.
6. If using 'dose-and-move' strategies, leave 10% of the lambs untreated so that resistant eggs are diluted with susceptible eggs.
7. Never have goats on a sheep farm as they metabolize anthelmintics rapidly, which exposes worms to sublethal doses.
8. Do faecal egg counts on six to ten sheep periodically to check efficiency of the programme and anticipate any breakdown, thus allowing appropriate anthelmintic treatment. Appropriate times might be ewes around lambing, and lambs around June and in September, but this needs modification on each farm. If egg counts are not being made regularly there is a real danger that clinical PGE may occur.

Since this programme is complicated and easy to get wrong, veterinary practitioners should be involved in its formulation and monitoring.

Immunity to worms has a high genetic inheritance and it is possible to select rams which will produce lambs which develop immunity to worms more rapidly. Rams with EBVs for genetic resistance to worms are becoming available.

Although many of the features of this programme are not controversial, there are those who disapprove of the basic recommendation of leaving a large population of

susceptible parasites *in refugia* and its counter-intuitive rationale has been criticized as being difficult to explain and assess. It is clear that the successful dilution of resistant parasites with additional susceptible parasites means that the total parasite burden is increased which may lead to the appearance of clinical PGE. It has been suggested that early-season treatment of ewes with a product such as moxidectin LA or ivermectin capsules, which have a residual action for *Haemonchus* of around 100 days, followed in the first year only by dosing the lambs and moving to clean pasture at weaning, reduces anthelmintic use and resistance is likely to be delayed for many years. The fact that there are now potentially five different groups of anthelmintic is encouraging and reassuring.

One exciting development is the research at the Moredun Research Institute on the isolation of antigens from the gut epithelium of *Haemonchus* and their use to stimulate protective immunity in lambs. This experimental work which has extended over several years, is leading to a field trial of a recombinant vaccine and it is hoped it will lead to a commercial vaccine soon.

Anthelmintics for cestode infections

Adult tape worms of the genus *Moniezia* are extremely common in young lambs and strings of white segments are frequently seen in the faeces by farmers and blamed for any clinical signs which may be found at the same time. There is little evidence that they exert any adverse effect on production. However, many BZ drugs (e.g. albendazole) are very effective against *Moniezia*. Praziquantel is also very effective and is incorporated into a mixed anthelmintic with moxidectin which kills cestodes and nematodes.

It should not be forgotten that sheep act as intermediate hosts for several cestodes of the dog and that treatment of dogs should form part of a sheep health programme.

Cobalt Deficiency

Cobalt deficiency is the cause of 'pine' or 'ill-thrift' in lambs, a condition which is well recognized in all sheep-producing countries of the world. The incidence is highest in areas where soils are derived from acid igneous rocks and where there are coarse, sandy soils. In Scotland, cobalt deficiency is widely distributed but in England, the localities most likely to be deficient are in the limestone areas of the Pennines, the old red sandstone areas of Hereford, Shropshire and Worcester, Dartmoor and the Greensands at the edge of the chalk in south-east England. As well as these areas where cobalt deficiency is endemic, other areas became cobalt deficient as a result of farming practices, such as liming and reseeding which have improved pastures and in so doing, lowered the available cobalt. A constant intake of cobalt is needed to allow rumen organisms to produce vitamin B_{12}, and the disease caused by cobalt deficiency is thus an induced vitamin B_{12} deficiency.

Clinical signs

The typical disease is characterized by:

- Loss of appetite.
- Reduced weight gains proceeding to weight loss and extreme emaciation.
- Lambs have a dry coat and a tight skin.
- Severe anaemia and lachrymation in the terminal stages.

Subclinically, a marginal deficiency of cobalt may be of considerable economic importance since typical signs may not be present but weight gains may be reduced. PGE is often also present and its clinical effects are more serious, and copper deficiency may be a complicating fact in some countries including Australia, New Zealand, South Africa and the USA but rarely in the UK. However, farmers tend to suspect deficiency states with little evidence so care should be taken over diagnosis.

The adult ewe may show signs of cobalt deficiency in late pregnancy due to fatty liver, leading to perinatal mortality.

Diagnosis

- History of farm and geographical area.
- Clinical findings.
- Response to treatment – this is often very marked after cobalt administration, but it should be noted that lambs with reduced appetite due to other causes may also show a response to dosing with cobalt.
- Laboratory confirmation.
  - In the animal, by serum and liver vitamin B_{12} concentration.
  - In the food, by herbage and soil cobalt concentration.

Treatment and prevention

There is no placental transfer of vitamin B_{12} and only low concentrations in milk; lambs require colostrum for their first supply.

Possible methods of treatment and prevention include:

- Cobalt boluses – the most effective treatment is to give each lamb, at about 8 weeks of age (not before, because they are too small to dose, and the rumen is not fully developed) a cobalt pellet or bolus. The bolus consists of soluble glass containing cobalt, selenium and copper and is effective for at least a year, but it is expensive, and for fattening lambs it may not be worth it, though it would be for lambs which are to be kept for breeding. Beware giving products containing copper unless there is a proven need for it.
- Oral dosing with cobalt – a drench of cobalt sulfate can be given to affected lambs which will need to be repeated every 3 weeks.
- Cobalt supplementation in mineral mixture – it is unlikely that lambs will be on concentrates but if so, the mineral mixture should contain sufficient cobalt to raise the whole feed to 0.11 mg Co/kg DM.
- Injection of vitamin B_{12} – this will deal rapidly with an immediate problem, but cost and the need for injections every 3 weeks precludes this as a preventive measure.
- Application of cobalt sulfate to grazing land – cobalt sulfate may be applied as a spray or as a granular top dressing at 2.0 kg/ha. Dressing need only be repeated every 3 years (or 6 years if the deficiency is only marginal) and only one-third of the grazing need be treated (at 6 kg/ha), since the sheep graze the treated strip selectively. The pasture should not be treated soon after liming.

10

Sudden Death

Sudden death or, more accurately, 'found dead' is a common problem in sheep keeping. Since the sheep flock is usually not as carefully observed as a dairy herd, the actual death is rarely observed and may have taken place at any time since the last inspection, so making an estimate of how recently death occurred is important (freshness, appearance of eyes, smell, autolysis). It is useful to look at the possible causes in relation to the age of the sheep affected and whether few or many are involved. The rest of the group or flock should be observed carefully to see if any more sheep are showing early clinical signs of disease. PME is an important diagnostic tool but it should be remembered that autolytic changes occur rapidly due to the insulating qualities of the fleece.

Some of the diseases which must be considered in the differential diagnosis of sudden death (Figs 10.1 and 10.2) are described in other parts of this book and will only be briefly mentioned here.

Young Lambs

In young lambs up to about 4 weeks old, a small number of deaths may be caused by:

- accidents;
- starvation/exposure (e.g. where a ewe develops unobserved mastitis or a lamb becomes mismothered);
- watery mouth where colostrum intake is delayed or absent;
- abdominal catastrophe such as gastric torsion; and
- lamb dysentery due to faults in vaccination technique, individual ewes being missed or lack of colostrum intake.

If many lambs are involved, the most likely causes are:

- hypothermia as a result of extreme weather conditions;
- lamb dysentery in a non-vaccinated flock; and
- septicaemia associated with *M. haemolytica*.

Growing Lambs

Up to about 6 months old, a small number of deaths may be due to:

- redgut (torsion of the mesentery) particularly if being artificially reared or on *ad libitum* creep feed; and
- dosing-gun injuries if drenching is carried out by an unskilled person.

Fig. 10.1. This well-grown lamb is ready to wean and will be vulnerable to clostridial disease if not already vaccinated.

Fig. 10.2. These weaned mule lambs should be protected against clostridial diseases and pasteurellosis, both of which cause sudden death.

If many lambs are involved the main possibilities are:

- pulpy kidney disease;
- braxy;
- acute abomasitis (*Clostridium sordellii*);
- acute pneumonia due to *M. haemolytica*;
- septicaemia due to *Pasteurella trehalosi*; and
- massive parasitic infections, particularly of *Nematodirus, Haemonchus*,

coccidiosis or acute fasciolosis but other lambs in the flock will be showing less acute signs.

Adult Sheep

In adult sheep, single or a few deaths may be associated with:

- accidents such as newly introduced rams fighting and fracturing the occipital process of the axis;
- sheep in fat condition becoming cast on their backs (common); and
- periparturient problems in lambing ewes.

If many sheep are involved, the most likely causes are:

- metabolic disorders, particularly hypocalcaemia or hypomagnesaemia (see Chapter 6);
- clostridial diseases, especially black disease, struck, blackleg or acute abomasitis;
- acute pneumonia associated with *M. haemolytica*;
- acute haemonchosis or fasciolosis; and
- toxicity associated with copper, yew or rhododendron.

Clostridial Diseases

Clostridial diseases have already been discussed in Chapter 3 so it is worth referring to this chapter too.

Clostridia are ubiquitous in the environment, especially in soil, so sheep are potentially always exposed to infection. The modern generation of farmers tends to be unaware of the catastrophic losses from these diseases which occurred in the era before vaccines were available. The majority of these diseases (apart from tetanus and botulism) cause rapid death so sheep are usually found dead with few or no clinical signs having been observed. Clinical signs if seen and death are associated with the production of powerful toxins by different species or types of clostridia which multiply in a number of different sites in the body.

Diagnosis

This needs to be speedy if control is to be effective. It is usually dependent upon obtaining from the farmer, accurate and relevant historical information (a trigger factor or other reason for the disease) supported, where possible, by a PME of fresh carcasses. It is important not to rely on PME alone, not least because the material is often too decomposed for useful interpretation. Further laboratory tests to identify the presence of specific toxins may be required.

The information you need includes:

- Did the sheep die 'suddenly' or was it 'found dead'? (Time since last inspection?)
- Has clostridial disease occurred on the farm before?
- What has the farmer done to control clostridial diseases? Have vaccines been used? (If so, closely check what and when and if individuals could have been missed or colostrum not provided to affected lambs.)
- Were there any obvious abnormal signs? (scouring, convulsions, stiffness).
- Is a particular age group affected? (note the age ranges for the different clostridial diseases).
- Has the farmer done anything to precipitate or introduce the disease? (e.g. change to better pasture or more concentrate fed/injected with dirty needles or irritant material/used rubber rings).
- What season is it? (Spring grass predisposes to pulpy kidney and struck, fluke in winter links with black disease, frost in autumn precipitates braxy.)
- Are the affected sheep in good condition? (Clostridial diseases often 'select' the greedy, fatter animals).

Treatment

This is rarely an option and is usually unrewarding even if the animal is seen alive.

Control

Clostridial vaccines are so cheap, so available and so effective (though no vaccine is ever guaranteed 100% effective) that it is not unreasonable to expect very few losses from these diseases. However, laboratory reports continue to show that serious losses still occur and that these are usually due to mistakes in application, for example: (i) choosing the wrong vaccine combination; (ii) injecting at the wrong times; (iii) 'dirty' injections (slick and clean SC injection technique is needed to avoid abscess formation); or (iv) just not vaccinating at all. The vet has a significant part to play in promoting a suitable vaccination schedule as part of a planned flock health programme.

The recipe for control contains three ingredients:

1. Avoid the predisposing factors: in practice, this really means the control of fasciolosis, the use of sterile instruments and working in clean, dry conditions with dry, clean sheep.
2. Antibodies derived via vaccination and adequate colostrum. Antisera for lambs from unvaccinated ewes are no longer available.
3. Antibiotic cover (LA antibiotic) following trauma (e.g. assisted lambing, dog bites).

Main clostridial diseases

These can be grouped into diseases: (i) causing enterotoxaemia affecting the digestive system, liver or kidneys (lamb dysentery, pulpy kidney, braxy, struck, abomasitis, black disease and bacilliary haemoglobinuria); (ii) causing muscle or soft tissue necrosis (blackleg, big head); and (iii) affecting nerve function (tetanus, botulism).

Lamb dysentery

This is seen in young lambs, up to about 3 weeks old, caused by *C. perfringens* type B. A few lambs are found dead, often only a few days old, with older lambs being affected later in the outbreak. A few lambs may be seen with signs of abdominal pain before death, with a hunched-up appearance. Dysentery is not necessarily present but PME usually shows haemorrhages in the intestinal walls and haemorrhagic gut contents. As with many clostridial diseases, the largest, best-fed lambs are often affected. Up to 25% of the lambs in the batch may die.

Pulpy kidney

This is the most common clostridial disease and is caused by *C. perfringens* type D. It is seen especially in the best growing lambs of around 4–10 weeks old and in store lambs being finished in the autumn on good grass or in the winter, often being fed concentrates. Rams may be affected, especially those on supplementary feed prior to mating. Most affected lambs are found dead as it is peracute, but some may show ataxia and convulsions before death. PME shows typical softening of one or both kidneys and yellowish fluid in the pericardium. A quick test is to check the urine for glucose.

Braxy

This is seen in autumn and winter in lambs or shearlings, often after eating frosty grass, and is caused by *C. septicum*. Affected sheep are found dead or may show abdominal pain or recumbency prior to death. PME shows acute abomasitis.

Struck

This is seen most commonly in the UK in adult sheep in spring, caused by *C. perfringens* type C. The disease often follows a change in diet and feeding of concentrates. Signs are the classic 'found dead' with others showing dullness and recumbency. Losses are usually low. PME shows haemorrhagic enteritis and excess fluid in body cavities.

Acute abomasitis

Abomasitis caused by *C. sordellii* is seen in growing lambs of 4–10 weeks old, almost always in housed creep-fed lambs.

These bacteria also cause toxaemia in older lambs and adults.

Black disease

Infectious necrotic hepatitis, or black disease, caused by *C. novyi* type B is seen in adult sheep during the winter and is associated with migrating immature liver flukes. These damage the liver and give rise to the conditions which allow the bacteria to multiply. The classic dark haemorrhagic appearance of the liver gives the disease its name.

Bacillary haemoglobinuria

This disease is caused by *C. haemolyticum* (*novyi* type D) and affects the liver and kidneys. The disease is sporadic and uncommon, occurring in adults in winter, also often associated with migrating liver flukes. Haemorrhage in the kidneys colours the urine dark red which may cause staining of the wool in the perineal area. Affected animals may live long enough for jaundice to develop.

Blackleg and big head

These diseases are caused by *C. chauvoei* and *C. novyi* type A, respectively, (malignant oedema is similar but caused by other clostridia). These diseases are associated with injuries and wounds which allow the multiplication of clostridia in the damaged tissues and are often called gas gangrene because of the typical necrosis and gas formation which quickly develop in the affected area. The sites affected most commonly are the muscles of the hind limbs, shearing wounds, uterus, pelvic and perineal areas in recently lambed ewes and the heads of fighting rams.

Tetanus

Affected animals are usually seen alive and show rigidity of muscles including those of the jaws and limbs. It is usually unrewarding to treat. For more details see Chapter 14.

Botulism

This is occasionally seen in sheep which have come into contact with manure from poultry sheds. The bacteria multiply in decaying carcasses, producing toxins which, when ingested, cause progressive muscle paralysis. So, unlike the other clostridial diseases in which toxins are produced from bacteria within the body, in this case the toxin is preformed. Signs include salivation and increasing flaccidity of muscles. It is usually fatal.

Systemic Pasteurellosis

This disease is a major killer of fattening lambs in autumn, particularly following weaning, transport or movement on to richer pasture and should always be at the top of the list of possible diagnoses, along with pulpy kidney, when losses are experienced in this age group. For further details see Chapter 13.

Toxicity

A variety of poisons, both organic and inorganic, cause severe illness and death. The most common are: (i) copper (usually a result of feeding concentrates or minerals formulated for cattle); and (ii) plants such as yew and rhododendron (often in sudden wintry weather when grazing is inaccessible, or from garden waste thoughtlessly thrown into fields). Obvious indicators may be present – jaundice and dark-coloured urine in the case of copper toxicity, or the presence of plant material in the mouth or rumen in cases of plant poisoning. Laboratory assistance may be necessary to confirm diagnosis.

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11

Lameness

Lameness is so common in most flocks of sheep that many farmers regard it as a 'fact of life' and give it only irregular attention (Fig. 11.1). Apart from the discomfort to the sheep, the loss in production can be considerable (e.g. 0.5 kg/week in the fattening lamb) and inadequate food intake by pregnant and lactating ewes contributes to pregnancy toxaemia and neonatal diseases. It is therefore one of the major welfare concerns in sheep farming in the UK and worldwide. The prevalence of lameness in a flock varies greatly with: (i) climate; (ii) pasture; (iii) age; and (iv) intensification. For example over 50% of lowland lambs can be lame with scald or footrot (FR) if the weather and pasture conditions are suitable, whereas the prevalence in hill flocks is nearly always low; similarly, many lowland sheep may be lame with soil balling, a condition which rarely occurs in hill sheep.

Techniques

Paring feet

In general, too much 'routine' paring is done, which may actually damage feet. The emphasis should be on routine inspection, with feet being left well alone in many cases. However, overgrowth with cracking of horn is common. Paring very overgrown or loose horn is a necessary part of diagnosis and treatment (Fig. 11.2) but should be done carefully with a good pair of sharp foot clippers. Paring should be sufficient only to cut away very overgrown horn or to make a clinical diagnosis. Recent work has shown that foot paring as a part of treating relatively early cases of FR delays healing. Great care must be taken not to cut too deeply into sensitive tissues. In particular, it is very easy to cut too deeply at the toe causing profuse bleeding. This may lead to the formation of a toe granuloma which will never heal unless treated correctly.

- **Remember, hoof paring should never make the feet bleed!**

Anaesthesia of the foot

For amputation of the digit, removal of granulomas or interdigital fibromas, IV regional anaesthesia is the best technique, though a ring block around the cannon bone is also effective.

Intravenous (IV) regional anaesthesia

Clean and disinfect the distal leg and place a tourniquet around the leg above the knee

Fig. 11.1. Lame sheep should be examined and treated as soon as reasonably possible.

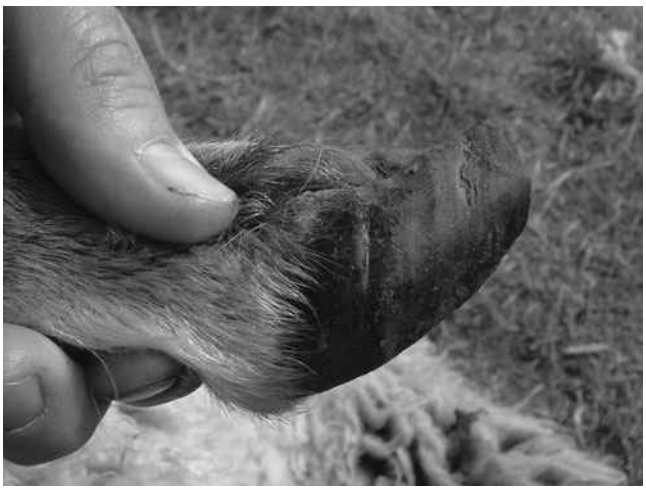

Fig. 11.2. This foot is overgrown and misshapen and requires careful trimming to investigate the cause.

or hock. Identify a suitable vein distal to the tourniquet and inject up to 5 ml of local anaesthetic. Anaesthesia develops in about 10 min. The tourniquet should not be released for 15–20 min to avoid potential toxicity problems.

Ring block

The cannon bone of the affected leg is cleaned and prepared. Then 8–10 ml local anaesthetic is injected at sites around the leg. Following this procedure the leg sometimes swells at the site of injection, so it may be worth putting a firm bandage on for a day or two.

Causes of Lameness

The main causes are:

- interdigital dermatitis (ID, scald);
- FR – classic separation and undermining of the sole and often the wall with a characteristic foul smell; and

- contagious ovine digital dermatitis (CODD) – separation and detachment of the hoof capsule starting at the coronary band.

Other common causes are:

- soil and grass accumulating between the claws (soil balling);
- interdigital hyperplasia (sometimes called a 'fibroma');
- granuloma caused by over-trimming or other injury, or associated with FR;
- white line problems:
  - localized tracks in the white line with pus formation; or
  - generalized white line separation with impaction of soil and debris (shelly hoof); and
- infection and sepsis in the pedal joint.

Although, as in pigs and cattle, the foot is the most common site for lameness, there are a number of important conditions affecting other parts of the limbs, for example:

- injury;
- joint ill – at various ages and with a range of causes;
- post-dipping lameness;
- strawberry footrot; and
- nutritional myopathy.

Common Foot Conditions

See Fig. 11.3 for the main diagnostic features. Recent work on ID and FR has overturned the traditional view of the microbiology of these conditions (see below) and it is now clear that they should be considered part of the same disease process. Often at least two feet are involved at the same time, leading, for example, to walking on both knees with consequent rubbing and loss of hair over the carpal joints. Sheep with FR or ID can run about and are not easy to catch in a field, and it is often difficult to pick out individually affected sheep when a group are penned together, without turning all the sheep up which is very hard work and time consuming, but probably cannot be avoided if a lameness problem is to be adequately tackled.

Interdigitial dermatitis (ID, scald)

Traditionally, this has been considered to be caused by *Fusobacterium necrophorum* alone, but it has now been shown that *D. nodosus* can initiate the lesion following damage to the skin of the interdigital space. If not treated, it is likely to progress to FR if virulent strains are involved. The main features are:

- The lesion is limited to the skin between the claws.
- This looks moist and reddened or greyish white, often with loss of hair.
- It is common in growing lambs.
- It is often associated with long, wet grass in warm weather.
- It can affect a large number of animals and develops quickly (overnight).
- It may affect more than one foot.
- It is likely to progress to FR unless only benign strains of *D. nodosus* are involved.

Most flocks are vulnerable and it is difficult or impossible to eradicate, although some flocks which have eradicated FR also report few cases of ID.

Footrot (FR)

This is a result of the primary action of *D. (Bacteroides) nodosus* with *F. necrophorum* as a complicating factor. These are both anaerobes. *D. nodosus* invades the horn resulting in separation of the sole beginning at the interdigital skin/horn junction near the heel. The degree of horn separation and severity of FR depends upon the strain of *D. nodosus* involved (there are at least 11 strains varying from benign types which cause little underrunning, to virulent very aggressive forms) and may be further aggravated by other secondary organisms such as spirochaetes and corynebacteria. The main features of the disease and the causal organism are:

- Separation and under-running of the horn of the sole, starting from the interdigital space, often spreading across the sole to affect the wall (degree depends on virulence).

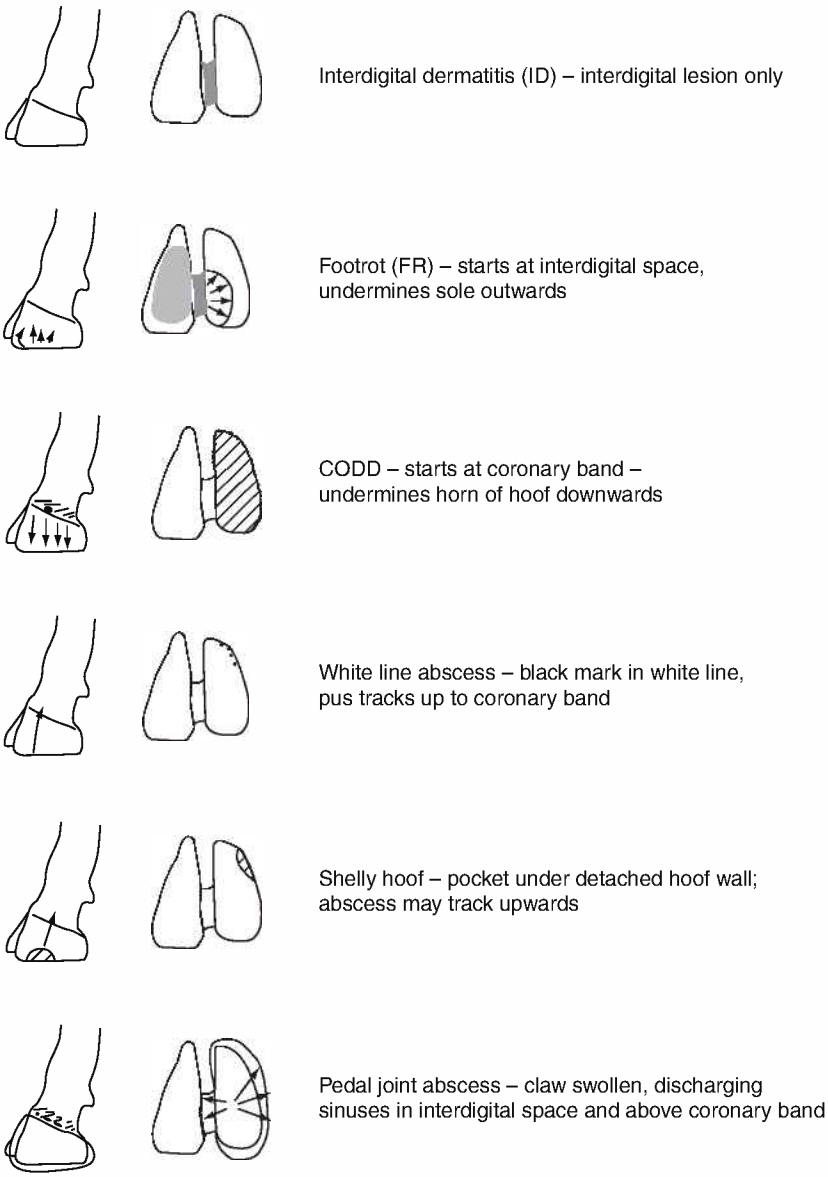

Fig. 11.3. Diagnostic features of foot lesions.

- A characteristic necrotic smell with dirty greyish cheese-like debris under the horn.
- It often affects both claws and may affect more than one foot.
- Most of the horn of affected claws may become detached, often remaining only attached near the toe.
- *D. nodosus* only lives in sheep's feet – survival on pasture is less than 2 weeks.
- It may also be spread by deer and cattle, but these are not thought to be important vectors and infected sheep are the usual source of infection.
- It only spreads in warm, damp weather, therefore spread in hot, dry summers

and in cold winter weather is much reduced.
- It does spread in winter housing, especially if pens are damp and inadequately bedded.
- It is less common in hill sheep because of the low grazing density and the organism does not survive well on peaty soil.

If untreated, self-cure can occur but can take many weeks and it is unacceptable to leave animals untreated in the hope this will occur. There is little naturally induced immunity, re-infection is common and feet may show more than one stage of the disease. As the main causal organism only lives in sheep's feet, it is possible to eradicate the disease and have a FR-free flock. This can be difficult to achieve as carrier animals are common.

Contagious ovine digital dermatitis (CODD)

This disease, which is distinct in appearance from ID and FR, was first reported in 1994 in the UK and called 'new virulent footrot'. It became clear that it was a different clinical entity and subsequent work implicates spirochaetes (*Treponema* spp.), similar to those found in digital dermatitis in cattle, hence the name. So far, for unknown reasons, it appears to be restricted to the UK. It is a severe disease, the main features being:

- The lesion starts at the coronary band, and is often bloody in appearance.
- It spreads down from the coronary band rapidly detaching the horn capsule.
- There is no necrotic smell as with FR.
- There is loss of hair above the coronary band.
- If not treated, the foot may sustain permanent damage.

Nothing is currently known about: (i) survival of the organism in the environment; (ii) whether animals become immune; or (iii) whether carriers exist.

Dealing with Flock Lameness

Diagnosis

It is essential to examine a representative sample of the group or flock to establish which conditions are present. It is common to have cases of ID and FR, and some flocks now have CODD as well. The under-running that results from FR infection and detachment of horn in CODD needs to be distinguished from the very common simple overgrowth of the outer wall and toe, where no sensitive tissues are involved and the sheep are not lame, and from conditions affecting the white line. Trimming should only be sufficient to remove obviously loose horn and confirm diagnosis, but should **not** cause bleeding. Treatment(s) should be based upon a thorough understanding of the diseases present and it is likely that a combination of measures will be required. Picking out and treating only obviously lame individual animals will never get on top of a flock problem.

Possible methods of treatment

A selection of treatments will be required in most flocks, the choice depending on: (i) the type of flock; (ii) management; and (iii) the main problems present. For flocks badly affected with FR and/or CODD, whole-flock treatment with tilmicosin (vet-only administration in the UK), has been described as being effective, but use of this type of antibiotic on such a scale must be seriously questioned and justified.

Vaccination

An effective vaccine is available for FR and may be curative as well as preventive so is worth considering as part of a treatment package, but is not effective against any other foot diseases. Diagnosis therefore needs to be certain. The vaccine is in an oily adjuvant and can produce lumps at the vaccination site. It is dangerous if accidentally self-injected; medical advice should be sought.

An overall improvement is usually seen following the first injection. Timing and frequency of subsequent injections depends on individual flock situation and identification of main risk periods.

Topical sprays

Various brands of antibiotic spray are available which are effective for treating ID and should be used in addition to injectables in treating FR. Treated sheep should not be immediately released into wet grass which will simply wash off the spray!

Parenteral antibiotics

Injectable antibiotics are now the method of choice for treating FR and CODD. These should be administered to all lame sheep and any others with lesions. As mentioned above, whole-flock treatment with macrolides has been questioned on the grounds of the risk of the development of antibiotic resistance. After successful treatment with antibiotics every effort must be made to follow treatment through to prevent resurgence of infection at a future time.

Possibilities are:

- Tilmicosin (which must only be administered by a vet in the UK) is effective for both FR and CODD and has been used successfully (although at considerable cost) as a whole-group or flock blitz treatment but note concerns expressed above.
- Very recently, gamithromycin has been described as being successful in whole-flock treatment for FR but, as this is not licensed for sheep in the UK and other effective antibiotics are licensed, its use has to be questioned.
- LA oxytetracycline is effective against FR and is currently the treatment of choice. There are mixed reports of its efficacy in CODD.
- Penicillin/streptomycin combination at twice the recommended dose has been successfully used for treating FR.
- Amoxycillin in combination with footbathing with chlortetracycline has had reasonably good results in treating CODD.

Following parenteral antibiotic treatment, there is usually a rapid improvement of the foot within a few days. If cases of FR have been treated early, paring the foot has been shown to delay healing, so definitely do not pare. If foot lesions are long standing, with overgrowth and detachment of the horn, some careful paring may be necessary but do not carry this out until some days after antibiotic treatment when healing is well under way. Recent work suggests the foot may regain a reasonable shape eventually without paring at all though there may be some disagreement on this point.

Footbaths

Footbathing is appropriate for treating outbreaks of ID, also as a precaution after gathering for any procedures in handling pens, and before housing. However, facilities must be good and the bathing must be carried out correctly. The trend is to build larger, wider footbaths in which a group of sheep can be held for the required length of time. The volume of chemical can be reduced by placing a specially designed mat in the base. After leaving the bath, keep the sheep in a concrete drying pen for at least 15 min.

- Formalin is cheap but unpleasant to both the shepherd and the sheep, as well as very painful to sheep with severe foot lesions. In some countries its use is not allowed. The strength used should never be more than 5% and 2–3% is adequate and effective for treating ID. 'Walk-through' formalin is effective, but be careful of rushing animals through as the solution can be splashed into eyes. Formalin 'goes off' quite quickly especially when contaminated by organic matter so fresh solutions are needed after a few days. Formalin hardens the horn so should not be used excessively.
- Zinc sulfate (10%) is usually very effective for FR and is the preferred treatment. Stand-in times vary according to the formulation of the product. Penetration may be aided by addition of a detergent (surfactant). For ID treatment, a stand-in

time of 2 min is usually adequate. For FR, up to 15 min or even longer may be necessary, usually repeated weekly, though daily treatment will speed up the cure rate. Zinc sulfate does not deteriorate and is still effective when it appears to be very dirty. It has no adverse effect on horn.

- Copper sulfate solution is also effective, but the potential toxicity of copper for sheep and the environment in general means that it is should be used with care, and disposed of safely. A product containing copper has been widely used in the successful FR eradication campaign in Australia.
- Various proprietary preparations are available (often containing organic acids) through agricultural merchants and these are favoured by some farmers. Check that data is available on efficacy before recommending.
- Antibiotic solutions are being increasingly used, particularly for CODD, which may be unresponsive to other treatments. There is nothing licensed for use in this way, therefore these should only be used if no other treatment is effective. Antibiotic footbaths are effective against scald and FR and farmers will want to use them for treating these diseases. The legal background should be pointed out. Those commonly used are lincomycin/spectinomycin and tylosin, and chlortetracycline has also been used. The dilution rate used is 1 g/l water. For CODD, this has been used as a hand spray to treat individually affected animals and seems to be effective if repeated several days in succession, but obviously does not address a flock problem.

Control

Chronically lame sheep are a serious welfare problem. In the UK in 2011, a Farm Animal Welfare Council report said that the extent of lameness was unacceptable and must be reduced. Most shepherds routinely trim and footbath, but there is often no planned control and sheep and shepherd live with FR. The more energetic the programme, the more effective is the control. For every lame sheep treated, there will be several others with less obvious disease. Frequent use of handling pens may actually increase the spread, therefore these should be cleaned frequently and all sheep footbathed every time they pass through the pens, even if for some other procedure.

- For ID or mild FR – regular (weekly) footbathing in **weak** formalin (now not used in some countries because of possible carcinogenic effect) or zinc sulfate should be effective. In small flocks, spraying affected individuals with antibiotic spray is effective.
- For FR where there is a severe or virulent form present, antibiotic injection is becoming favoured rather than repeated footbathing because this is less time and labour intensive. However, repeated footbathing in zinc sulfate (stand-in time at least 15 min for clinical cases) has been effectively used over many years. Vaccination is also helpful. Separation of infected and uninfected animals, with the infected group treated separately until cured, is likely to lead to a better outcome.
- For CODD only, tilmicosin injection, or amoxicillin injection combined with antibiotic footbathing may be the best options.
- If CODD *and* FR are present, the above treatment for CODD will be effective for FR as well.

Having got lameness under control, it is then crucial to: (i) carefully monitor the situation; (ii) treat newly lame animals as soon as possible; and (iii) re-impose group or flock treatments if the incidence starts to increase again.

Eradication of FR

FR has been eradicated in some flocks in the UK but the main area in which success has been achieved is in Australia. In New South Wales the number of properties with virulent FR has been reduced from 6000 to 15 as a result of the combined efforts of vets,

farmers and other advisors. It has helped that there is much more of a willingness to cull affected animals or even whole flocks than there is in the UK. It also helps that there are predictable weather patterns with long, dry periods when the disease is not transmitted.

FR has only recently been recognized as a problem in Scandinavia and serious attempts to control or eradicate are being made.

Eradication is a difficult and time-consuming exercise and by no means certain. It certainly requires that the farmer be prepared to examine all feet, pare sufficiently to determine presence or absence of infection, separating clean and infected sheep, and to treat everything that is infected. In Australia parenteral injection of antibiotic is now favoured over footbathing during this phase. Persistent cases are culled. Eradication must include safeguards against re-introduction and this means maintaining self-contained flocks (including adequate sheep fencing) or inspection and eradication of infection in purchased sheep. Few flocks in the UK, particularly the large ones, can manage all this. It is best to attempt it when most of the lambs have been marketed and old ewes culled, but before tupping. It is also wise to choose a time when there are fewer cases, brought about perhaps by a previous vaccination programme and in a period of dry weather if possible.

Other Foot Lameness

White line lesions

There are two common types of white line lesion: (i) shelly hoof; and (ii) white line abscess. Shelly hoof occurs when a pocket develops beneath the outer wall of one or more claws. This then becomes impacted with soil and grit. When the loose horn is pared away, a characteristic half-moon appearance of the wall results, with the underlying laminae visible. Lameness does not occur until the impacted soil is pushed into the deeper sensitive laminae when pus forms. The sheep then develops acute lameness which is not relieved until the pus is either released by paring or bursts out after tracking up to the coronary band.

White line abscesses develop in small discrete tracks in the white line which again cause acute lameness and can sometimes be very difficult to locate, although the affected digit should be identifiable by pain response and the presence of heat. Careful paring may release pus.

For both types, if careful paring does not find pus, application of a poultice covered with a polythene bag and bandaged, left on for a couple of days will soften the horn and encourage bursting, or at least make re-examination easier. Treating with injectable antibiotics may suppress but not eliminate infection, so may actually prolong the duration, but it would probably take a brave person to withhold antibiotics from a valuable animal! Once the pus has been cleared, healing occurs leaving a loose piece of horn which can be trimmed.

Foot abscess (pedal joint sepsis)

Infection of the pedal joint is a serious condition which needs to be differentiated from white line lesions. There is severe pain (10/10 lameness) with obvious swelling of the digit and pus bursts out at several sites around the coronary band. One theory is that infection gains access from the interdigital space, but this is not proven and it is possible that infection may be blood-borne. Radiography will help to decide if the joint is involved, as will careful examination with a probe. For early cases, irrigating the joint via a cannula inserted either through a lateral coronary band sinus, or by drilling through the lateral hoof wall into the joint has been described and may lead to a satisfactory result. Either of these forms of treatment should be carried out under regional anaesthesia. If recovery is not rapid after this form of treatment, amputation, which is relatively cheap and easy, should be considered, and certainly immediately for cases where treatment has been very delayed.

Amputation of a digit

This should be carried out under regional anaesthesia as described previously. Clean the lower limb and shave the skin above the coronary band. When anaesthesia has developed, incise through the skin all the way around the affected claw about 0.5 cm above the coronary band. Take great care in the interdigital space that the incision does not damage the opposite claw. Then remove the claw either by sawing through the lower end of P1 using an embryotomy wire, or disarticulate the joint between P1 and P2. Place a non-stick dressing over the wound, pack with cotton wool and bandage tightly. Cover with waterproof tape. Then release the tourniquet. The bandage can be removed 2–3 days later. Opinions vary about re-dressing – some people prefer to leave the wound open, others prefer to re-bandage for a bit longer. In any case, the animal should be kept in clean, dry conditions until healing has occurred. This takes about 3 weeks.

Interdigital hyperplasia ('fibromas')

Fibrous outgrowths in the interdigital space (mainly hind feet) are a feature of some breeds. Lameness occurs only when they become ulcerated and infected. Frequent footbathing maintains some control, but the worst cases require simple surgical removal under local anaesthesia; this is not a job to be done just before tupping time but should be identified and carried out well in advance.

Toe granulomas

Most large flocks have a few sheep with strawberry-like granulomas, usually at the toe, often the result of over-paring, but also following injury or FR. Although loose horn will grow over and partly hide the lesion, healing will never occur without radical treatment. Loose horn should be pared away (made easier if the foot is anaesthetized), the granuloma cut off after application of a tourniquet to the leg, and the base cauterized (a calf disbudding iron is suitable). Alternatively, repeated application of an astringent such as copper sulfate or formalin (5%) may eventually work.

Post-dipping Lameness

This is caused by *Erysipelothrix rhusiopathiae* which can invade broken skin. At the time of dipping, this organism, which is present in soil, can seriously contaminate the dip solution and enter limb wounds sustained in the dipping process. The disease has also been reported in the absence of dipping, after sheep passed through very muddy pens. Within a day or so, many sheep may be very lame with a local cellulitis of the hairy parts of the limbs. Early treatment with penicillin is essential. In future, ensure that dip and/or handling pens are cleaned before use. Contaminated dip should not be used after standing overnight especially in warm weather.

Joint Infections

Young lambs up to 1 month – mainly lowland

Polysynovitis and arthritis, particularly of the stifle (soft, bulging and painful synovia in the patellar region), carpus and hock joints, follow a septicaemia possibly from bacteria gaining entry via the navel, tonsils or gut. The lambs appear miserable and crouched with flexed joints and arched back and are reluctant to move about. The most commonly isolated organisms are:

- *Strept. dysgalactiae* – this is becoming a widespread problem, with source and route of infection not clear as it is found in well-managed flocks with good hygiene standards.
- Other opportunistic bacteria including *E. coli* and *A. pyogenes.*

Control is not always easy as it is not always obvious what is at fault. Navel treatment and lambing pen hygiene should be checked – is the navel dipped or sprayed?

How effectively? Is a contaminated dip being used? Is cow teat dip, which is emollient rather than astringent, being used? Is colostrum intake adequate? Are hygiene procedures such as tailing, docking and ear tagging adequate? If all possible predisposing factors have been dealt with, injection of lambs with a single dose of antibiotic (penicillin should be adequate) as a preventive measure can be considered as a last resort. As the problem may not recur in future lambing times, this should not become a procedure carried out routinely each year.

There is some evidence that *Strep. dysgalactiae* can be found in the posterior reproductive tract of ewes, so lambs may become infected during and immediately after birth. Use of disposable gloves for handling lambing ewes and newborn lambs may help to reduce spread of infection.

Lambs over 2 months – lowland

Stiff lambs/erysipelas arthritis

On some farms, a significant number of fattening lambs at grass show a stiff, stilted, short-striding walk or hopping run, appearing to be lame on more than one leg. Very careful examination and palpation discloses some synovial swelling, general thickening and pain in some limb joints, particularly the stifle, hock and carpus. Although probably infected soon after birth, the condition is insidious and often irreversible by the time it is recognized. *E. rhusiopathiae* is the most common organism causing this chronic fibrinous synovitis and arthritis. Often the organism cannot be isolated from the joints but a blood titre of 1/320 and over indicates this infection. Badly affected animals should be killed on humane grounds, but it is worth treating less severe cases for which penicillin should be effective. Such lame sheep need housing so that more intensive treatment and feeding can be applied and where they do not have to walk far for food or shelter. Repeated penicillin treatment is advisable. Vaccination is effective, but relies on colostral transfer of antibodies, therefore vaccinating ewes in late pregnancy (as for clostridia) is necessary.

In the USA, polyarthritis in lambs has been associated with *Mycoplasma* spp. and, in fattening lambs in feedlots, with *Chlamydophila* infection.

Lambs up to 4 months – hill

Tick-bite pyaemia, following tick bites and staphylococcal septicaemia is the usual cause (see Chapter 16).

Adult sheep

Joint infection as a result of injury may be a problem in individual adult sheep, leading to visible swelling of the affected joint(s). Early treatment with a prolonged course of penicillin is necessary if this is to be successful. Delay leads to irreversible joint damage and welfare issues in affected animals.

A chronic degenerative arthritis of one or more joints, particularly elbows, but sometimes shoulders, hips and stifles is quite often seen in elderly sheep. Elbows should be carefully examined for characteristic thickening and restricted flexion. Such sheep become progressively more lame and thin and need very careful attention. Culling should be advised.

Vitamin E and Selenium Deficiency

Deficiency of either selenium or vitamin E, or both together, lead to problems affecting muscle function variously known as white muscle disease, stiff lamb disease, nutritional myopathy or nutritional muscular dystrophy. Many areas of the world (e.g. parts of northern Europe, Australia and New Zealand) have soils low in selenium content. Vitamin E content is high in fresh grass but falls in stored forage and grain, so deficiencies can be made worse if home-produced diets are fed.

This is essentially a disease of young lambs which are growing fast. Predisposing factors include: (i) feeding only home-grown foods; (ii) adding preservatives to straw and

cereals; and (iii) use of artificial fertilizers with high nitrogen (N) and sulfate (SO_4), promoting rapid grass growth. Selenium-responsive ill-thrift in lambs has been reported in some countries as has early fetal death leading to barren ewes and the increased susceptibility to disease because of reduced phagocytic activity.

Clinical signs

The usual picture is that thriving young lambs (50% occur at 0–30 days old and 25% at 30–60 days old) have just been turned out to fresh spring grass and within a few hours or days of running around, a few are found down and reluctant to get up; one or two may also be found dead due to cardiac failure. Those that are able to stand if forced to get up, take a few tottering steps, showing particular stiffness in the shoulders, and lie down again, looking alert and not uncomfortable. Some may show distressed respiration due to failure of respiratory or cardiac muscles, and pneumonia is a secondary risk. This situation is, at first, easily confused with pulpy kidney, pasteurellosis, swayback, polyarthritis, spinal abscess and injury.

Diagnosis

- Suspicion arises if there has been previous evidence of selenium deficiency on the farm and, in particular, if the flock is receiving little concentrate and no vitamin/mineral supplement and also if a lot of roots and/or poor quality roughage is being fed.
- The sudden onset in several otherwise healthy-looking lambs (often the fastest growing) should point to this condition and the absence of obvious signs of injury and arthritis should increase the suspicion.
- Necropsy should eliminate pulpy kidney disease and pasteurellosis, but note that the muscle lesions are bilateral and therefore no comparisons are possible within the lamb and that if there is cardiac involvement, the lungs may look congested.
- If analysis of plasma and whole blood (green tubes) from affected lambs show plasma creatine kinase (CK) levels of over 1000 IU/l this indicates severe muscle damage, but the blood must be taken very early, as concentrations drop in a few days; and whole blood GSHPx is often <1 unit/ml.
- If analysis of whole blood (green tubes) from ten apparently healthy contacts show suspiciously low levels of GSHPx (i.e. <20 units/ml).
- Inject with vitamin E and selenium and see whether there is a response (it may take a few days).

Treatment

- Inject with vitamin E and selenium, and repeat the next day if there is not much improvement. Usually there is a good (diagnostic) response if caught early.
- Rest the affected lambs – bring inside.
- Consider giving an NSAID injection to affected lambs – this can make them more comfortable and speed recovery.

Prevention

Be careful if recommending products containing several trace elements, particularly those containing copper as accidental copper poisoning may occur in copper-sensitive breeds.

- Ensure there are adequate levels of selenium and vitamin E in the diet of ewes in late pregnancy: avoid low selenium foods (e.g. turnips), and feeds which reduce vitamin E concentrations (e.g. treated grain, spoilt hay, oil seeds). Ensure a dietary concentration of 0.1 mg/kg DM for selenium and 30–50 mg/kg DM for vitamin E.
- Inject ewes in the third month of pregnancy with a selenium/vitamin E preparation.

- Short-term control in lambs can be achieved by injecting them at strategic times, such as shortly before turnout or folding on roots.
- Some anthelmintics contain added cobalt and selenium. These might be sufficient to help lambs with marginal deficiencies in the short term.
- Long-term control can be provided by supplementing with selenium by various methods including:
    - An annual injection to lambs at weaning and to pregnant ewes, of a LA barium-complex of selenium.
    - Dosing at strategic times (tupping, lambing, weaning) with a proprietary trace element product containing selenium.
    - Dosing weaned lambs and ewes with LA selenium pellets – these give 3 years protection.
    - If multiple deficiencies of copper, cobalt and selenium, or zinc, cobalt and selenium occur (particularly on upland farms), it is worth considering use of soluble glass boluses containing these minerals. These give about 6 months protection.

12

Skin and Wool

Although a number of specific conditions affect the skin and wool of sheep (see Table 12.1), it must be remembered that because the production of wool is an active process, many systemic diseases will result in local or generalized loss of wool (Figs 12.1 and 12.2). In addition, primitive sheep shed the fleece in early summer, a feature which is still seen to a limited degree in some breeds and crosses.

Disorders Associated with Pruritus

Correct diagnosis is very important. Most often, scab or lice is the cause but infection with both parasites is possible. Treatment should be appropriate for the diagnosis – incorrect treatment based upon farmer diagnosis or assumptions without confirmation of presence of the parasite, has resulted in the development of resistance to some of the available (ever-reducing) treatments. **Always check for the latest information on drug use as availability, indications for use and withdrawal periods may change.**

The flock should be viewed from a distance to obtain some indication of the proportion of ewes showing signs of pruritus: (i) restlessness; (ii) stamping of feet; (iii) rubbing against posts; or (iv) nibbling or biting at areas of the body. Individual affected sheep should be examined to:

- determine the areas affected;
- establish the nature of the lesions including involvement of the skin;
- discover the location of visible parasites (use a hand lens); and
- take specimens from active lesions for further examination.

Parasitic conditions

Sheep scab (psoroptic mange) and other mites

Sheep scab occurs in most sheep-keeping countries, although North America and Australasia are free of this disease. It was a notifiable disease in the UK for many years and was eradicated from the country by compulsory dipping in 1952. Unfortunately, after years of freedom, sheep imported from Ireland in 1973 reintroduced the disease and despite compulsory dipping regulations which exerted some control, outbreaks continued to occur every year. In spite of a rising number of outbreaks, control was deregulated in 1992 although it is once again notifiable in Scotland. Control is the responsibility of farmers. In Scotland, treatment or slaughter of infected sheep is compulsory.

Table 12.1. Summary of conditions affecting skin and wool.

Condition	Cause	Pruritus	Area affected
Non-infectious			
Natural shedding	Genetic	No	Neck, back, belly
Wool break	Illness, debility	No	Body
Wool slip	Winter shearing	No	Flanks
Sunburn	Lack of pigment	No	Ears
Photosensitization	Photodynamic plant, liver damage	Yes when healing	Face, ears
Parasitic			
Sheep scab	*Psoroptes ovis*	Intense	Body
Ear mites	*Psoroptes cuniculi*	Head shaking	Ears
Foot and scrotal mange	*Chorioptes bovis*	Medium	Lower legs, scrotum
Lice	*Bovicola ovis*	Medium	Body
	Linognathus ovillus	Slight	Head
	Linognathus pedalis	Medium-severe	Legs
Keds	*Melophagus ovinus*	Little	Body
Blowfly strike	*Lucilia sericata*	Intense	Breech, shoulder
	Phormia terrae-novae		
Headfly	*Hydrotaea irritans*	Intense	Head, base of horns
Infections			
Mycotic dermatitis	*Dermatophilus congolensis*	Little	Back, ears, nose
Staphylococcal dermatitis	*Staphylococcus aureus*	Little	Periorbital, nose, legs, teats
Caseous lymphadenitis	*Corynebacterium pseudotuberculosis*	No	Abscesses in lymph nodes esp. parotid
Actinobacillosis	*Actinobacillus lignieresi*	No	Head and neck abscesses
Fleece rot	*Pseudomonas aeruginosa*	No	Body
Orf	Parapox virus	No	Lips
Strawberry footrot	*D. congolensis* and orf	No	Lower leg
Ringworm	*Trichophyton verrucosum*	No	Head
Ulcerative balanoposthitis/vulvitis	*Streptococcus zooepidemicus?*	No	Prepuce, vulva
Scrapie	Spongiform encephalopathy	Usually intense	Tail, flanks, head

In other parts of the UK, owners of infected sheep can be prosecuted under various welfare regulations if the sheep are judged to be suffering unnecessary pain or distress. Regulations introduced in 1977 allow local authorities to take action where farmers do not, particularly on common grazing. It is also illegal to sell infected animals other than direct for slaughter.

EPIDEMIOLOGY The disease is caused by a non-burrowing mite, *Psoroptes ovis*, which is specific for sheep. It is highly contagious, and is usually spread by direct contact, often being introduced by the purchase of infected sheep, or spreads among sheep on common grazing. The mite can also live for about 16 days in pieces of scab or wool, on fences, etc. and can be spread by contaminated shears or clothing.

The mites 'graze' on the skin surface, feeding on skin secretions. Rapidly extending moist lesions develop from the pustules at the feeding sites, the skin reaction being due to hypersensitivity to the mite excreta, rather than to the mites themselves. Sheep show signs of extreme irritation with head-tossing, occasionally even developing epileptiform fits, and rub vigorously and nibble at the affected areas which are particularly over the shoulder and flanks. The fleece becomes stained and matted with loose tags,

progressing to areas of wool loss and bleeding often extending, in neglected cases, to most of the body. Severe infections result in marked loss in condition and death.

Fig. 12.1. Wool growth is an active process, some breeds such as this Wensleydale producing heavy, long fleeces.

The mites are capable of enormous multiplication over a relatively short time with maximum numbers occurring 6–12 weeks after infection. It is now known that there are different strains, which vary in virulence. The disease is most active during the winter, though latent infections with small numbers of mites are common in the summer, with a flare-up to typical disease in autumn and winter.

DIAGNOSIS In typical outbreaks of sheep scab, when a high proportion of the flock is affected and lesions are severe, diagnosis is straightforward since the white mites can just be seen with the naked eye. When mites are not so numerous, superficial skin scrapings should be taken from the edge of the lesion for microscopic examination (add 10% KOH to clear skin debris). *Psoroptes* has well-developed legs which protrude beyond the body, and the first two pairs of legs, in both male and female mites, end in bell-shaped suckers which have long, segmented (the diagnostic feature) pedicels.

A number of other mites are found on sheep, including *Chorioptes bovis*, usually on the legs, scrotum or brisket (see later) and tyroglyphid or forage mites which can be found all over the body and which,

Fig. 12.2. This Texel ewe has wool break following a difficult lambing.

though they are non-parasitic, occasionally cause pruritus.

TREATMENT AND CONTROL Always check the latest information as availability and instructions for use may change. The range of available products has decreased as synthetic pyrethroid (SP) dips are no longer allowed to be used in the UK because of their severe effects on the environment (they are much more toxic to wildlife, especially in watercourses, than are organophosphates (OPs)). This leaves only:

- Diazinon dip (an OP) – this will treat and prevent reinfection for about 4 weeks (the withdrawal period is 70 days). For treatment, sheep must be in the dip for 1 min and must be completely submerged at least once.
- Injectable avermectin or milbemycin – there are four possible products. All have long withdrawal periods (6–15 weeks). **Remember these are also anthelmintics so how they integrate into worm control must be taken into account.**
    - Ivermectin – this has no residual action therefore two treatments are needed 7 days apart to make sure all the mites are killed. It has no protective action.
    - Moxidectin 1% – this can be used for protection (28 days) and treatment (two injections at a 10-day interval).
    - Moxidectin 2% LA – this is administered as a single injection for treatment and protection for up to 60 days.
    - Doramectin – this is administered as a single IM injection for treatment and control.

Pour-ons are **not** suitable for treating scab as some mites are exposed to a sublethal dose of chemical. Resistance to SPs has already been reported in the UK; this almost certainly developed as a result of repeated application of SP pour-ons.

Concern has been expressed over the toxicity of OP compounds for those dipping the sheep and this has brought about licensing of those who use them and, overall, a reduction in routine dipping. Alternatives being used include showers, sprays and jets to apply chemicals, but OP dips are not licensed for use by any of these methods and only dipping is effective for scab treatment. There are strict rules about safe disposal of dips.

Psoroptes otitis

A sub-strain of *Psoroptes* (probably *cuniculi*) appears to have become adapted to living in the ears of sheep. Affected sheep show head shaking, often with a purulent discharge from the ears, and a number develop aural haematomata. This infection can be present in flocks with no classic scab, and does not appear to spread from the ears to the body. Diagnosis can be confirmed by examining swabs or washings from the ears for mites. Treatment is by two doses of ivermectin or moxidectin 1% injected 7–10 days apart, or a single injection of doramectin or moxidectin 2%.

Chorioptic mange (C. bovis)

These mites colonize mainly the scrotum of rams or the udder of ewes and the lower legs, and are becoming common in some breeds. Affected animals show itching of the legs (e.g. rubbing against troughs or on fence wire). Affected rams show thickening of the skin of the scrotum which may decrease fertility. Mites live in small colonies and are most easily picked up by using a tape strip, rather than a skin scraping. The mites are surface feeders of skin debris, therefore treatment by injectables is not effective. Dipping with diazinon is the only current treatment option.

Sarcoptic mange (Sarcoptes scabei)

This is not present in the UK or northern Europe but may be a problem in countries in the Middle East, Asia and Africa.

Lice and keds

There are two types of lice: (i) chewing, *Bovicola* (*Damalinia*) *ovis*; and (ii) sucking, *Linognathus ovillus* and *Linognathus pedalis*. The sheep ked, *Melophagus ovinus*, is a

wingless fly and has made something of a comeback since the reduction in dipping. The life cycle of these highly host-specific insects is similar, with all stages on the sheep. Adults live for only a few days off the sheep.

Infestation with lice has become a significant problem in the UK since dipping has become less common. Lice are a major problem in Australia where they adversely affect wool production (the most important sheep product) and in New Zealand where they cause downgrading of skins (an important by-product of the meat lamb industry) with an abnormality called 'cockle' (also seen in the UK).

Clinical signs are similar to sheep scab, hence much confusion, with heavy infections leading to irritation involving a high proportion of the flock with resulting rubbing, nibbling and damage to fleece. Populations are highest in the winter months. Keds are large brown insects and are easily seen but lice may be difficult to see. A few staples of wool should be clipped from the edge of a lesion and examined in a bright light with a magnifying glass for adults and eggs attached to the wool.

Lice and keds are mostly susceptible to insecticidal drugs including diazinon (by dipping) and SPs (pour-on), although resistance to SPs is developing. Sucking lice and keds (but not chewing lice) are susceptible to the systemic endectocides (ivermectin, etc). Treatment is usually carried out in the autumn or winter. If infested sheep have a heavy fleece, it is better to treat after shearing.

Resistance to SPs is particularly a problem in Australia. This develops if the chemical is not applied in sufficient amounts to thoroughly saturate the fleece, which occurs commonly when automatic jetting races are used rather than showers or dips. Insect growth regulators have been used as an alternative but resistance has also developed to these products.

Blowfly myiasis (flystrike)

Strike is a common condition seen throughout the summer months in the UK (and found in many other parts of the world) in which larvae of *Lucilia sericata* (green bottle fly), *Phormia terrae-novae* (black blowfly) and *Calliphora erythrocephala* (bluebottle) invade the skin under soiled fleece, resulting in extreme discomfort and rapidly leading to death. The most common areas which are affected are: (i) around the breech associated with soiling due to scouring; (ii) on the side of the body where fleece is contaminated from FR lesions when the sheep lie down; and (iii) in various sites associated with wounds. Affected animals are restless, show vigorous wagging of the tail when the breech is affected or biting at affected areas and are often separated from the flock. The larvae of the flies, which develop within a few days from the eggs laid in the moist contaminated area, burrow into the skin and cause a foul-smelling wound. This extends rapidly as a result of secondary strike. With early lesions a careful hunt has to be made to find the maggots. It is important that flocks at risk should be examined at least once a day to detect early cases as affected animals die within a few days of the initial attack.

Affected areas should be clipped and the maggots removed physically. An SP pour-on will kill the maggots and protect the area against further strike. Since secondary bacterial infection is common, injections of antibiotic are valuable and an NSAID injection will also help.

CONTROL This is by application of a suitable product in the early summer. Choices are:

- Diazinon dip (30 seconds in dip) will control blowflies for about 8 weeks.
- SP (cypermethrin) pour-on will treat and protect for 8–10 weeks.
- Insect growth regulator pour-on – either cyromazine which protects for 10 weeks or dicyclanil which protects for about 8 or 16 weeks depending on the particular product. **Note that these products do not treat, therefore they must be applied before the eggs are laid.**

Control of the factors predisposing to strike, especially faecal soiling, is also critically important.

Screw worms

Fortunately northern Europe and Australia and New Zealand are free of these flies which

lay eggs in a similar manner to blowflies, but whose larvae (screw worms) are very invasive and difficult to control. Australia is potentially at risk because of their presence in New Guinea. They are a great problem in parts of South America but an eradication programme using irradiated, sterile males has cleared them from the USA, Mexico and Central America.

Headfly

This disease is caused by a non-biting muscid fly, *Hydrotaea irritans*, which swarm around the heads of sheep, causing rubbing which leads to self-trauma. The flies then feed on the exudate of blood and lymph, which leads to further attacks. The area where horns and skin join is often the site of small breaks in the skin which probably explains why the condition is particularly important in horned breeds, but any open wound is liable to attack. The disease is most common in the North of England and south of Scotland but the fly occurs throughout the UK. The wounds do not heal while the flies are active and extensive lesions involving much of the skin of the head are produced. Loss in condition and disfigurement of the skin occurs.

The flies are active from late June until the end of September on still, warm humid days and there is only a single generation of flies each year. Non-feeding females rest in trees and can fly 0.5 km/day. Each female produces one or two batches of about 30 eggs which hatch to larvae in pasture soil (not dung) from September to May especially near woods. Pupae are found near the soil surface from May to June. The flies can feed on a variety of animals; meal analysis shows that about 80% feed on cattle.

Many methods of control have been tried; none is entirely successful, but the most effective method is the application to the top of the head and base of the horns of an SP pour-on (cypermethrin). In areas of high challenge, the treatment will have to be repeated at least every 6 weeks while flies are active.

Ticks

See Chapter 16.

Midge hypersensitivity

This has been seen affecting individuals or significant numbers in several breeds, particularly those with black wool (Black Welsh Mountain) or black belly wool (Torddu). It seems to resemble sweet itch in horses and is seen from early May to autumn, while midges are active. Affected animals show severe itching and discomfort with thickened, scabby, often bleeding lesions on the hairy parts of the body, especially face, ears, ventral abdomen, udder and perineum. Control is by improving the habitat to make it less attractive to midges (cut overgrown grass) and application of an SP pour-on before midges become active.

Non-parasitic conditions

Scrapie

Scrapie should be suspected when an individual adult sheep begins to rub, nibble or suck its fleece for no obvious reason, leading to bilateral semi-bald patches on its flanks, hind legs, bridge of the nose and top of the head. The skin and fleece appear normal except for self-inflicted healing scabs. The irritation is increased by firm finger rubbing of the back and itchy areas. Other neurological signs are often present (and sometimes on their own) and there is a worsening over days/weeks. Scrapie is notifiable. For details see Chapter 14.

Disorders with Usually Little or No Pruritus

Infectious

Orf (contagious pustular dermatitis, scabby mouth)

This disease, which occurs worldwide, is a constant worry to sheep farmers as shown by the priority that they give it for further research. If it occurs around lambing, it threatens to develop into an outbreak and spread to the teats of ewes, which in turn

can lead to difficulty for lambs to suck and mastitis developing in the ewe. Later in the year, it can disrupt sales of fat or breeding animals. Occasional cases also inexplicably develop extensive and persistent lesions. **It is a zoonosis so there is a risk of spread** to shepherds, particularly those caring for orphan lambs; lesions on the fingers, face or neck are, at best, irritating and painful, and at worst there is a very marked reaction with local lymphatic involvement. Doctors are often unfamiliar with this infection – sometimes the vet diagnoses it first!

Control measures are difficult and the epidemiology of the condition remains unclear. However, while the appearance and site of the lesions in and around the mouth suggest a lot of discomfort and interference with sucking and grazing, many lambs survive without problems, most cases self-cure in a few weeks and the incidence in most flocks remains low.

Orf is caused by a parapox virus which survives in dry scabs, from year to year indoors, but for shorter periods outside; small scabby lesions on the hairy areas of the face and limbs are also persistent sources of the virus; it is susceptible to iodophor disinfectants. It has a distinct shape and size and is easily recognized by electron microscopy (EM), although it is morphologically indistinguishable from the paravaccinia (pseudo-cow pox) virus found in teat lesions of cattle. It has an affinity for hairy areas particularly at the weak junctions between skin and mucosae (e.g. commissure of lips) but it requires some surface damage to allow invasion (e.g. splitting of lips, eruption of incisors, rough grazing and teat sores following vigorous sucking and incisor injury by hungry lambs).

The lesions are characteristic and diagnostic, although when in doubt, unpreserved scabs should be sent to the lab for EM examination. They start as raised red papules which coalesce and in a few days proceed through vesicles and pustules to thick scab formations which are firmly attached to what looks like exuberant granulation, and removal causes haemorrhage. Secondary mixed bacterial infection is common and exaggerates the local response. The lesions are often confined to the outside of the lips and are sometimes barely noticeable, but in young lambs, granulomatous lesions may occur within the mouth, involving the gums and sometimes the tongue. Despite their appearance, the lesions do not appear to cause much distress unless particularly exuberant and extensive, and while there may be some lambs who fail to thrive, most will self-cure within 3–6 weeks. The situation becomes much more serious, however, if and when the lesions secondarily arise on the teats of ewes (see Chapter 8).

TREATMENT Individual severe cases should be separated to ensure proper nursing with daily application of antibiotic to tackle secondary bacterial infection. Ewes should be inspected for teat lesions and these should be treated with antibiotic. Ointments containing local anaesthetic may allow lambs to carry on sucking until healing occurs; in extreme cases it may be necessary to wean and hand rear their lambs (easier said than done as lambs will often refuse a bottle, plus the risk of frequent handling) and to dry off the ewe following infusion of LA antibiotic. Where necessary, sheep should also be removed from pastures that appear to be causing face or limb abrasions (e.g. thistles). Where possible, contaminated pens should be cleaned out and disinfected before re-use, and again at the end of housing.

PREVENTION Immunity following primary infection is incomplete and short-lived, perhaps only a few months, but subsequent infections are likely to produce milder lesions which heal more rapidly. Immunity is largely cell mediated and although humoral antibodies are produced, they are not protective, and therefore neither is colostrum.

A vaccine is available which consists of live virus. It is applied by skin scarification, usually on the hairless area of the inner thigh. Its efficacy and timing is debatable but it probably shortens the recovery time if not preventing infection. It is commonly applied to ewes in early or mid-pregnancy, especially to those which will be housed at

lambing, in an attempt to reduce the weight of infection in carriers, and to protect the teats. It is sometimes applied to lambs in the summer a few weeks before sale, in an attempt to avoid lip lesions and subsequent refusal at markets. It is also sometimes applied to young lambs in contact with infections, or where there has been a previous history of disease, and even to those with lesions, in an attempt to prevent or control an outbreak; the consensus seems to be that this should be a last rather than a first resort. It certainly should not be used on ewes within 7 weeks of lambing nor in flocks that have no history of the disease, as the induced scabs are a source of further infection. The vaccine, like the natural infection, needs to be handled with care. It should never be used in a flock without a previous history of the disease.

Strawberry footrot

Occasionally lambs at pasture, often in wet, muddy and abrasive conditions, develop orf-like granulomatous lesions on the skin of the lower limbs particularly around the coronary bands, making the lambs obviously lame. The condition does not involve the foot and has no resemblance to FR, but the granulation tissue is likened to a strawberry. Orf virus particles can usually be isolated from the scabs, but so also can *Dermatophilus congolensis*, the organism more commonly associated with lumpy wool; it is uncertain which gets there first! Treatment should be for both, which means local dressing and systemic antibiotic. The lambs need to be removed to dry pasture or housed. Some cases are particularly persistent and extensive, like other forms of orf, and these require euthanasia.

Ulcerative balanoposthitis (pizzle rot) and ulcerative vulvitis

It is quite common to find a scabby ulcerative lesion at the junction of the skin and mucosa of the prepuce in rams and vulva in ewes. Raw, bleeding tissue is exposed if the scabs are removed, rather like orf, but the lesion is ulcerative rather than proliferative and it is unusual to find orf virus particles. In some cases in rams, the ulceration is confined to the glans penis and therefore goes unnoticed. The incidence can be quite high and is sometimes associated with tupping time, suggesting venereal transmission.

Most cases self-cure or remain as minor lesions and there is no interference with breeding or fertility. However, occasionally the lesions are extensive with swelling, superficial sepsis and necrosis; in rams this may mean the involvement of both the glans penis and the mucosa of the sheath which becomes noticeably pendulous; subsequent scar tissue may interfere with breeding.

The cause is still uncertain, although ureaplasms or *Streptococcus zooepidemicus* have been found in some outbreaks. Antibiotic aerosol spray is adequate for mild cases, but the more severe ones require systemic antibiotic and frequent local dressing and irrigation.

Staphylococcal folliculitis

Small discrete pustules are often seen around the lips, muzzles and under the tails of very young lambs, and on the udders of ewes (mammary impetigo). The surrounding skin is hyperaemic but not proliferative as is orf and a crater is left when the pustules rupture, which heals quickly leaving a white patch if the skin is pigmented. Treatment is rarely necessary although local dressing and antibiotics directed at the causal organism, haemolytic coagulase positive *Staph. aureus*, may occasionally be justified.

Staphylococcal dermatitis (facial or periorbital dermatitis or exzema)

This is another variant of the skin lesions associated with *Staph. aureus*. Extensive, suppurating scabby lesions, which bleed easily and have a deep ulcerated centre surrounded by a zone of hair loss, are seen over bony prominences such as the orbit, nose and lower legs. Sheep feeding at crowded sites are usually affected, typically late-pregnant ewes being fed concentrates with insufficient trough space, where they both

injure themselves and each other, and transmit the infection to one another. Removal from the source of the problems and treatment with local dressing and antibiotics usually leads to rapid improvement, although serious and disfiguring complications can arise from eye involvement and from scar tissue. In future, adequate trough space must be provided (at least 45 cm per ewe) and the food spread evenly and quickly to minimize aggression or adopt floor feeding if this is practical.

Mycotic dermatitis (lumpy wool)

This condition occurs in many countries and in many if not most flocks, but generally as a low-grade self-limiting infection, and is of little consequence in the UK other than some downgrading of the fleece. However, occasional outbreaks occur where the infection becomes more active and extensive, and secondary problems arise such as flystrike; treatment is then necessary.

The disease picture is an exudative dermatitis passing rapidly through the stages of hyperaemia, exudate and scab. It is caused by an actinomycete *D. congolensis* which also infects other species (e.g. horses, cattle and occasionally humans) and can remain viable in dry scabs for many months. It induces only weak local immunity, all of which means that successive 'attacks' are common and eradication is not an option.

The condition is often only noticed when a sheep is handled (e.g. being condition scored) and on parting the fleece, bundles of wool fibres are seen matted together by rough scabs. These scabs originate from the skin and are carried up in the growing fleece, eventually working to the tips and breaking off. The back and top sides of sheep are most commonly affected as these are the sites which get soaked following heavy rain or dipping; fat, flat-backed, close-woolled sheep are the most susceptible, where the fleece acts as a sponge. Other areas do get infected, appearing as scabs on the ears, head, limbs, udder of ewes and scrotum of rams, and these are constant sources of re-infection elsewhere.

The distribution of the scabs and the general lack of pruritus are usually sufficiently diagnostic but if there is doubt, impression smears should be made of skin exudate or scabs taken, preferably those close to an active lesion, for laboratory examination.

Significant outbreaks usually follow wet weather or dipping, but it can also follow shearing when skin wounds may admit infection.

Sheep that need treatment should be housed and given either a 5-day course of a conventional dose or a single high dose (70 mg/kg) of penicillin and streptomycin; topical dressing with aluminium or zinc sulfate may be justified to prevent further surface spread. Combined insecticidal and antimycotic dips are no longer available.

Other fleece problems

- Fleece rot is caused by *Pseudomonas aeruginosa* leading to discoloration (green, brown or blue) of the wool.
- *Pseudomonas* infection has also been associated with severe fatal necrotic dermatitis, probably induced as a result of bad dipping practice.
- Canary stain is a permanent yellow coloration of the wool.

Ringworm

This is not particularly common in sheep, perhaps because of lack of contact with other hosts such as cattle or because of the protective fleece, but housed, shorn ewes occasionally develop the condition, particularly if housed in buildings also used by cattle. It has also been seen on the heads of rams. It is caused by the fungus *Trichophyton verrucosum* and on the hairy areas of head and face, it looks just like the condition in cattle, but the infection in the woolly areas often produces more obvious circular raised plaques which eventually lift off, leaving the more typical crusty skin beneath.

The condition usually has to run its course, waiting for immunity and turnout; topical iodine sprays may be useful but **handle with care as the disease is a zoonosis!**

Caseous lymphadenitis (CLA, cheesy gland)

This disease, seen in many sheep-keeping countries including Australasia, was first identified in the UK in 1990 in imported goats. It spread to sheep and is present in some pedigree flocks producing terminal sires and is being seen increasingly in commercial flocks, usually introduced by infected rams. There is also a real possibility of spread via contaminated shears and shearing equipment if these are not adequately disinfected between flocks.

The disease is caused by *Corynebacterium pseudotuberculosis* and is seen as chronic abscesses in superficial lymph nodes. These often burst producing greenish cheesy or putty-like pus. Abscesses repeatedly wall off, producing a characteristic 'onion-ring' appearance. Abscesses may also form in internal organs especially the lung and mediastinum, causing chronic wasting, or signs resulting from pressure on vital structures from large internal abscesses. Confirmation of diagnosis is by identification of the organism from samples of pus. An important differential diagnosis is bovine tuberculosis (TB) which is widespread in parts of the UK. Fortunately sheep seem to be relatively resistant to this although a small number of flock outbreaks have been reported.

EPIDEMIOLOGY In the UK, the mode of spread and the lymph nodes most commonly affected are rather different from those in Australia where the disease is common. In both countries, discharging lymph nodes in the lung form a common source of infection.

- In Australia, the disease spreads at major gathering times such as shearing and jetting or dipping. Entry of the disease is via skin abrasions; hence body lymph nodes such as the prefemoral and popliteal are commonly affected.
- In the UK, spread appears to take place more commonly via the oral route – trough feeding facilitates the spread from discharging lung abscesses, thus head lymph nodes, especially the parotids, are commonly infected. These are situated just below the ears (see Fig. 12.3) and should always be carefully examined during any ram examination, as well as the main superficial nodes (submandibular, prescapular, prefemoral, popliteal, supramammary). Infection also spreads directly from sheep to sheep from discharging abscesses.

TREATMENT Although the organism is susceptible to antibiotics, the nature of the abscesses means successful treatment is unlikely. Treatment of superficial abscesses

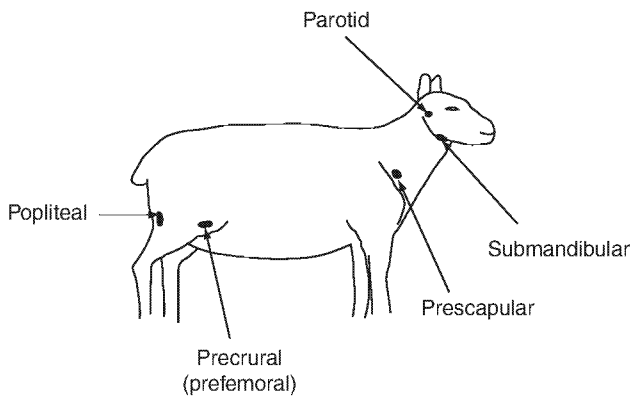

Fig. 12.3. Important superficial lymph nodes.

is of little use since inevitably other lymph nodes become infected and infected internal nodes cannot be detected.

CONTROL Vaccination is widely used in Australia and other countries (usually in combination with clostridial vaccine) and seems to be quite effective. The vaccine is prepared from the bacterial toxins. This vaccine is not generally available in the UK, but in certain circumstances can be imported under special licence from VMD. Autogenous vaccines made from killed bacteria have been used in the UK and recent information is that they may provide more protection than the Australian vaccine. It is possible that a new vaccine may become available in the UK in the reasonably near future.

It is best to try to avoid introducing infection into a flock by careful selection of purchased animals and quarantine. An ELISA test is available and has led to the introduction of the CLA Monitoring Scheme offered by SAC. Monitored groups have to be isolated for at least 12 weeks, are subject to two clinical examinations and two blood tests. If clear, these animals can be sold as 'CLA monitored status' giving buyers confidence that they are unlikely to be infected.

If infection gets into a flock, measures to reduce spread include: (i) floor feeding rather than trough feeding; (ii) weaning of lambs as early as possible ('snatching' lambs at birth and artificial rearing in extreme cases); and (iii) keeping age groups separate. Ram breeders may sell ram lambs rather than keeping them to shearlings and risk infection. Blood testing using the ELISA test with culling of positive animals has had some success in eradicating the disease from some flocks but is expensive.

Actinobacillosis

The main differential diagnosis for CLA is sporadic cases of actinobacillosis ('cruels') where abscesses occur along the lymphatics in the head and neck region. These are responsive to treatment (penicillin/streptomycin by injection for 5 days should be adequate).

Non-infectious

Wool break

A wool break usually follows any severe illness or prolonged debilitating condition including inadequate nutrition. It is most marked during pregnancy and lactation, particularly following pregnancy toxaemia, difficult lambing or acute mastitis. It is common to see ewes, often in poor condition, in late winter or spring patchily shedding their complete fleece and in the process looking very 'moth-eaten'. Underneath, the skin is normal and providing the precipitating factors are reversed, normal wool regrows.

Wool slip

This is an allied but specific condition frequently seen in housed, winter-shorn ewes. Here, there is patchy loss of wool mainly confined to the hind quarters and back, developing within a few weeks of shearing. It is thought to be related to the stress of housing, shearing and cold, resulting in raised blood cortisol concentrations. The ewes are not obviously adversely affected but it can lead to delay in turnout.

Sunburn

Occasional cases of sunburn follow exposure to bright sunlight, particularly in recently shorn white-faced breeds. Marked skin erythema and oedema develop in exposed surfaces such as the ears, back and udder and necrosis with extensive scabs may develop. Affected sheep need housing and if secondary infection is suspected (e.g. *Dermatophilus*) antibiotic therapy is indicated. Shade will be required in future.

Photosensitization (yellowses)

Bright sunlight can also induce photosensitization if the white sheep have previously been eating a photodynamic agent as found, for example, in St John's wort (*Hypericum perforatum*), or they have raised circulating concentrations of the photodynamic agent phytoporphyrin (a breakdown product of chlorophyll), which arises when liver

function is impaired. This impaired liver function can be caused by a variety of factors including copper toxicity, toxic plants and fungi, and some drugs, so the precise factor is rarely discovered, particularly if only one or two sheep in a flock are involved. In northern Europe, the plant bog asphodel (*Narthecium ossifragum*), found growing on natural hill or mountain pasture, is widely associated with the problem. In New Zealand, the condition, known as facial eczema, is caused by ingestion of sporidesmin fungal spores.

The most noticeable feature is the marked oedema of the head and face including the eyelids and ears, which makes the sheep look very 'droopy' and miserable; other non-pigmented areas may also be affected and if severe enough, there is necrosis and eventual sloughing, particularly of the tips of the ears which then look 'cropped'.

Cases should be housed and carefully nursed. Blood samples for liver enzyme estimations may help to decide the prognosis. If cases continue in a flock, look at copper blood concentrations, check for toxic plants and investigate recent treatments.

13

Respiratory Diseases

Several of these diseases (see Table 13.1) are involved in two important areas of loss: (i) sudden death; and (ii) thin adult sheep. In both cases PME is going to be an important part of the diagnostic process in addition to clinical examination of any live animals. Recently, ultrasound examination of the chest has been developed as an aid to diagnosis of lung lesions, although some experience is required in obtaining and interpreting images.

Pasteurellosis

Microbiologists seem to like reclassifying bacteria at intervals, seemingly to confuse students and clinicians! This is particularly the case with bacteria commonly referred to as '*Pasteurella* spp.'. The bacterium *Pasteurella haemolytica*, which used to be divided into two biotypes, was then split into two separate species, *Mannheimia haemolytica* and *Pasteurella trehalosi*. The latter has more recently been reclassified as *Bibersteinia trehalosi*. In this book, to avoid confusion, we will continue to use *P.* rather than *B. trehalosi* and to refer to the diseases by the term 'pasteurellosis'. This group of bacteria is a common and important cause of pneumonia and rapid or sudden death in sheep.

As most sheep carry *M. haemolytica* in the naso-pharynx or tonsils, the mere isolation of this organism in nasal swabs or at PME, does not establish the diagnosis; it is generally held that it requires some stress factor or intercurrent disease to precipate pasteurellosis.

There are three different manifestations of disease:

1. Septicaemia and rapid death in young lambs caused by *M. haemolytica* (a few days to 3 months old) – affected lambs are usually simply found dead or profoundly depressed with a high temperature and a PME is required to confirm diagnosis.
2. Pneumonia in older lambs and adults, also caused by *M. haemolytica*, most commonly in late spring and early summer (May–July) but also in store and fattening lambs in the autumn and winter. Affected animals are febrile and depressed with nasal discharge, coughing and laboured respiration. Some may survive but go on to develop chronic disease with weight loss.
3. Septicaemia and rapid death in the fattening and store lamb (6–9 months old) in the autumn and winter caused by *P. trehalosi* (systemic pasteurellosis). The most common first sign is one or two sheep found dead. A few others may appear very ill and separated from the rest and unwilling to be driven; they are usually febrile (over 40°C) and show

Table 13.1. Summary of main respiratory diseases.

Organism	Disease	Main age group affected
Bacteria		
Mannheimia haemolytica	Septicaemia	Young lambs
	Pneumonia (acute and chronic)	Growing lambs, adults
Pasteurella trehalosi	Systemic pasteurellosis	Growing lambs in autumn
Mycoplasma ovipneumoniae	Atypical pneumonia	Growing lambs
Arcanobacterium pyogenes	Laryngeal chondritis	Adults, especially heavily muscled terminal sire breeds
Internal parasites		
Dictyocaulus filaria	Parasitic bronchitis, husk	Growing lambs
Viruses		
Retrovirus	Ovine pulmonary adenocarcinoma	Adults over 2 years (occasionally in growing lambs)
Lentivirus	Maedi	Adults over 2 years

increased respiratory rates. Some may have crusted eyelids and nostrils and usually there is some coughing in the group. About 10% may become affected in an outbreak.

Diagnosis

- Necropsy of fresh carcasses if available. In the septicaemic and systemic forms, widespread petechiae and ecchymoses are usually present with varying degrees of pleurisy and lung pathology present in all forms. *Mannheimia/Pasteurella* need to be isolated in large numbers to confirm diagnosis and ask the laboratory for serotyping to check against the vaccine serotypes.
- Try to find a predisposing reason for the deaths or illness; in particular, with systemic pasteurellosis enquire if the group has been moved or handled in the last day or so, or if the weather has been noticeably different, for example wet and windy (rain/wind-chill factor) or warm and still. Other intercurrent infections such as parainfluenza virus type 3 (PI3), Jaagsiekte and tick-borne fever may also predispose to clinical outbreaks of the pneumonic form.
- Consider clostridial diseases as the main differential, in particular pulpy kidney and braxy, and check on the vaccination history and dates.

Treatment and control

- For young lambs, control is by vaccination of ewes (for colostral antibodies) and early vaccination of lambs – remember, colostral protection is short, only 2–4 weeks. Also improve the ventilation of lambing sheds. Administer antibiotics such as oxytetracycline or tilmycosin (not if under 15 kg) if animals are seen alive.
- For older lambs and adults, control is by vaccination before the risk period, and improve ventilation if poor housing is a trigger (Figs 13.1 and 13.2). Administer antibiotics as above in acute cases. Chronic cases may not respond unless the course is very prolonged and this may not be economic.
- Systemic pasteurellosis can be difficult to deal with and control:
  - Sometimes the group is large and inaccessible, which means the incidence is unsure, PME is delayed and there is a very real difficulty in deciding when to do something, that is whether to gather (which may make matters worse) and treat (all?). Often a number of deaths occur over a period of days or weeks and then unaccountably cease.
  - Rescue and isolate affected sheep – treat these and as many contacts as seem worthwhile. Tilmycosin is

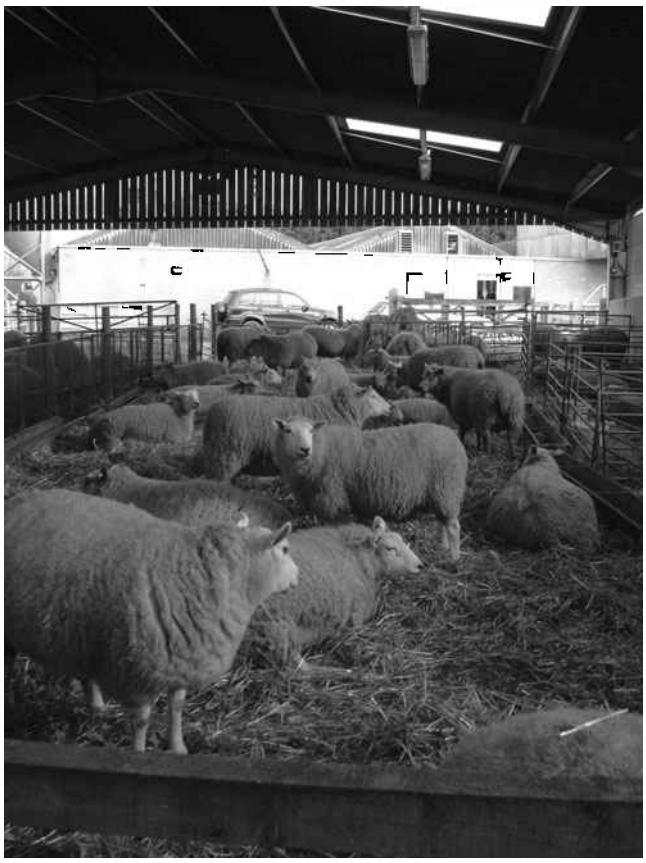

Fig. 13.1. This lambing shed has good ventilation reducing the risk of pneumonia.

very good for pneumonia but is now vet-only administration, so if you are going to treat sick animals yourself this might be the drug of choice. If the farmer has to treat, other drugs will have to be used, for example LA oxytetracycline given twice at a 4-day interval.

- Recommend vaccination – a dead vaccine is available, containing most of the serotypes known to cause the disease, and requires two doses at approximately a 4-week interval to provide useful protection. If given to ewes in late pregnancy it not only affords protection to the ewes that spring, when the incidence of *Mannheimia* pneumonia is highest, but also to the lambs via colostrum for the first 3–4 weeks of life and so helps to cover the first major risk period for this type of septicaemia (contrast with passive clostridial protection which lasts 12–16 weeks). To cover adequately the next few weeks and months, lambs need to be given two doses of vaccine at a 4-week interval, starting 3 weeks after birth; there is evidence that any colostrum-derived antibody does not interfere with the response to this early vaccination.
- Look at the environmental conditions (e.g. wet, exposed, overcrowded, lush food) and consider providing plenty of shelter or moving the group, although the moving could make matters worse.
- Consider giving LA oxytetracycline immediately before the period of

Fig. 13.2. These fattening lambs are vulnerable to pneumonia if the ventilation of the housing is poor.

risk (e.g. on purchasing or moving store lambs in the autumn).

- In the long term, avoid, if possible, very exposed wet and windy sites which induce stress and encourage the flock to crowd into small sheltered sites which, in turn, intensify the aerial spread of pathogens. Also, avoid putting store lambs on lush grass in the autumn without considering vaccination first.

The position regarding vaccination is complicated by combining this vaccine with a clostridial vaccine to form a very popular product which is considerably more expensive than clostridial vaccines alone. While such a combination is ideal for the vaccination of ewes, it matches the needs of lambs less well because of the disparate length of protection by colostral antibodies for the two categories of disease. In practice, if this combination is to be used in flocks where there is a significant incidence of septicaemia and sudden deaths in young lambs which is diagnosed as pasteurellosis, the normal lamb clostridial vaccination schedule needs to be brought forward to 1 and 2 months rather than the usual 3 and 4 months; alternatively *Pasteurella* and clostridial vaccines can be given separately at appropriate times.

The pasteurellosis vaccine was improved a few years ago and is now more effective in the control of disease in the later store and fattening lamb. Antigenicity of some serotypes involved in this form of the disease was improved by growing the bacteria in a very iron-depleted medium which increased the expression of some important antigens on the surface of the bacteria.

Parasitic Pneumonia (Parasitic Bronchitis, Husk)

Although sheep are commonly infected with a number of nematodes which are

found in various parts of the respiratory system, the only species which is associated with clinical disease is *Dictyocaulus filaria*. It is found in the trachea and bronchi. The heaviest pasture infections with infective larvae occur from September to November. These larvae can overwinter and act as a source of infection to the next season's lambs.

The disease is not as important clinically as 'husk' in cattle. This is probably because of the more frequent dosing of lambs for PGE, which, with most modern wormers, incidentally reduces the number of worms in the lungs. (Note that morantel is not effective against lungworms.) However, the worm is common and can cause coughing and loss in condition in lambs in August–October. The lambs develop a strong immunity and few worms are found in older sheep, although they can be found in old or debilitated animals. Many lambs are sent to slaughter before the peak of infection.

Diagnosis

Diagnosis is based on clinical signs and seasonal incidence and can be confirmed by faecal examination for first-stage larvae by the Baermann apparatus. Faeces should be fresh and *D. filaria* can be distinguished from the larvae of other non-pathogenic worms in that they: (i) are long (500 µm); (ii) have a blunt tail; (iii) have a cephalic knob; and (iv) contain refractile granules.

Treatment and control

All the modern benzimidazoles, levamisole and avermectins are effective against *D. filaria* and can be used to treat clinically affected lambs. Parasitic bronchitis is unlikely to occur where these drugs are used earlier in the year to control PGE but morantel, although effective against stomach and intestinal worms, is not effective against *D. filaria* at the usual dose.

Atypical Pneumonia

This is an unsatisfactory name given to the type of pneumonia which closely resembles that so commonly found in the fattening pig and housed calf. It is principally a disease problem in the housed or densely stocked fattening or store lamb (3–12 months old) and associated with a variety of agents such as *Mycoplasma ovipneumoniae*, PI3 and adenovirus. It is particularly common following the mixing of lambs introduced from the market in autumn and winter.

Clinical signs

The disease is characterized by chronic coughing, ocular and nasal discharges, and is usually afebrile, although the occasional acute pneumonic lamb arises which is febrile, off-food, listless, and with obvious respiratory distress; these cases are associated with secondary *M. haemolytica* infection. Affected lambs take several weeks longer to reach slaughter weight and consume more food to do so.

Diagnosis

This is based on the clinical signs, environmental conditions and the pathology of lungs, which show collapsed areas in the lower parts of the lung lobes at PME or slaughter.

Control

As for pigs and calves:

- Supply more fresh air.
- Reduce numbers under one roof, and in any one group.
- Split groups according to age, size and origin.
- Inject with LA oxytetracycline if animals show signs of clinical illness.
- Consider pasteurellosis vaccination to protect against additional disease caused by these pathogens.

Slow Viral Pneumonias

Ovine pulmonary adenocarcinoma (OPA, sheep pulmonary adenomatosis, SPA, driving sickness, Jaagsiekte)

Although this condition has caused worrying losses in some flocks, particularly of Scottish origin where the incidence can be up to 10%, it usually only causes sporadic deaths, but its potential danger is illustrated by the losses that occurred in Iceland in housed sheep. It is a contagious adenomatous tumour of the lungs of sheep and possibly goats, and thought to be caused by a B-type or D-type retrovirus, perhaps associated with a herpes virus, with an incubation period of up to 3 years.

Clinical signs

The signs are of loss of condition and severe respiratory distress, but without much coughing, in an individual adult sheep when driven. This respiratory embarrassment, which sounds like 'bubbly porridge' on auscultation, increases over several weeks. Characteristically, if the ewe is held with its head lowered (the 'wheelbarrow' test), relatively large volumes (30–300 ml) of clear mucous exudate flow from the nostrils; this fluid contains virus so is infectious. There is no fever and the animal feeds up to the terminal stages when there is often fulminating pasteurellosis.

Diagnosis

There is no serological test and diagnosis is based on clinical signs (including the 'wheelbarrow' test) and PME; the lungs may weigh up to 4 kg (1.5 kg is normal) and show areas of grey tumour tissue with frothy white fluid in the trachea and bronchi. Although the virus can be detected in laboratory tests, no commercial test is yet available. Pasteurellosis may mask the presence of OPA, so where significant losses of older sheep occur from pasteurellosis, lungs should be checked histologically for OPA.

Control

Take care in introducing new stock to uninfected flocks. There should be prompt slaughter of the obviously affected animals. Once a serological test becomes available, then monitoring and pre-clinical culling becomes a possibility. The disease has been eliminated from a few flocks by removing lambs at birth and rearing them artificially in the same way as used to produce maedi-free animals from infected flocks (see below).

Maedi-visna (MV, ovine progressive pneumonia)

Maedi means 'air hunger', visna means 'wasting'. Maedi is seen as a chronic progressive pneumonia affecting sheep over 3 years old and visna is the nervous form of the disease, again affecting older sheep. Both diseases are caused by a lentivirus, similar but not identical to that causing caprine arthritis encephalitis in goats. The virus causes lesions in the lungs, CNS, joints and udder. Maedi was first recognized in the UK in 1978, having probably been introduced from the continent, where it was common, with imported stock. It does not occur in Australia and New Zealand.

A number of flocks, mainly in East Anglia and southern Scotland, have had clinical cases, but because of the insidious nature of the disease which takes 3–4 years for clinical signs to develop, it is likely that many more flocks have subclinical infection. In 1993, a random check on blood samples showed a seroprevalence of 0.4%, which extrapolated to at least 70,000 seropositive sheep then, more now. Economic losses occur from premature culling of infected ewes and from reduced lamb growth rates. Unless sheep farmers take the threat from this disease seriously, it could have a major impact on the health of the national flock in the foreseeable future.

Unlike OPA, diagnostic serological tests can be used to identify MV-free flocks and this is applied in the SAC Premium Sheep and Goat Health Scheme. This has particular application for the pedigree breeder,

especially those wishing to export sheep, but should be taken up more widely by commercial breeders. A monitoring scheme aimed at commercial flocks has been introduced with less rigorous rules than the accreditation scheme.

The disease is spread in two ways:

1. By coughing or sneezing infective droplets, which is important in housed sheep.
2. Most importantly, by colostrum and milk from infected ewes, which contains the virus.

Clinical signs

Maedi is seen as a chronic progressive pneumonia affecting sheep over 3 years old and is invariably fatal. Affected sheep lose condition and lag behind when driven, with characteristic difficulty in breathing which becomes progressively worse over a period of months.

Visna is the nervous form of the disease, again affecting older sheep, in which weight loss and vague, slowly progressive neurological signs occur. One recognizable feature may be the development of a unilateral conscious proprioceptive deficit of one hind limb, with dragging and scuffing of the toe progressing to paralysis of the hind legs.

The virus also affects the udder causing an indurative lymphocytic mastitis which is not always recognized because milk remains normal in appearance, but lambs from affected ewes have been shown to weigh 3 kg less at weaning than those from healthy ewes. Arthritis and vasculitis are other possible components of the clinical picture.

Diagnosis

In the individual, this is based on clinical signs and PME. Serological tests such as the agar gel immunodiffusion test (AGID) and ELISA are useful at the flock level, but false negative results, due to delay in antibody production or immunosuppression, do occur, which makes them less useful in the individual animal.

Control

- Slaughter affected animals.
- Do not breed from the offspring of affected ewes.
- Be careful about the source of ewe colostrum fed to lambs.
- Keep a young flock and only keep replacements from the young ewes.
- Purchase from accredited flocks only.
- Join the SAC Premium Sheep and Goat Health Scheme and eradicate by test and slaughter.

Because the infection is not passed to the lamb before birth, it is possible (though demanding) to obtain uninfected lambs from an infected flock by supervising lambing and immediately removing lambs, certainly before sucking the ewe. Lambs are then given colostrum substitute and reared artificially.

Laryngeal Chondritis

This acute obstructive upper respiratory condition, seen most commonly in Texel sheep (and Belgian Blue cattle), is characterized by severe dyspnoea with laryngeal stridor and is often fatal if not treated quickly. The laryngeal occlusion results from chronic suppurative lesions (usually associated with *Arcanobacterium pyogenes*) within the arytenoid cartilages, the etiology of which is unclear but is probably connected to the characteristic exaggerated anatomy of the breed with its short, broad neck. Rams, ewes and growing lambs are all known to have been affected. No investigations have been made into any genetic predisposition, as some breeders are reluctant to admit to having had cases.

Treatment

These cases are often presented as emergencies.

- Treat with a large dose of steroid, intravenously if possible, and broad-spectrum antibiotic. Carry on

with reduced doses for 5–7 days. This regimen alone may be adequate for less severe cases.

- If dyspnoea is severe, an emergency tracheostomy is necessary under local infiltration anaesthesia. This usually gives immediate relief, but good aftercare is needed with the tube changed twice daily initially, reducing to once daily if the tube remains clear of discharges. After the tracheostomy tube has been in for 2–3 weeks, infection may have subsided, so try leaving the tube out. If dyspnoea is still present, the only remaining option is surgery to try to remove the necrotic tissue from the larynx.

The prognosis should always be guarded as recovered cases may subsequently relapse, sometimes just being found dead. Think about the advisability of using recovered animals for future breeding even if they are said to be valuable.

14

Neurological Diseases

Neurological diseases of sheep are common throughout the world, often affecting individuals but some can become a flock problem. They are a notoriously difficult area in which to make a firm diagnosis on which to base a specific treatment. Indicators such as age, number involved and speed of onset will help. In addition to a clinical examination, a full neurological examination should be carried out (for the routine to follow see Appendix 2 'Neurological Examination of Sheep'), but this is not easy in a collapsed sheep! The aim should be to localize signs to a specific part of the CNS, or to decide that signs are generalized.

The main CNS 'syndromes' which should be identifiable are:

- Cerebral – depression, blindness, circling, proprioceptive deficits, head turn (e.g. cerebrocortical necrosis (CCN), PT and meningitis are diffuse diseases; gid, abscess and tumour are more likely to be unilateral).
- Cerebellar – wide-based stance, high head carriage, ataxia, dysmetria, intention tremors, nystagmus (e.g. gid, Border disease, daft lamb).
- Vestibular – head tilt, circling, falling or rolling, nystagmus, sometimes facial paralysis (e.g. inner and middle ear infection).
- Ponto-medullary (brainstem)–depression, multiple cranial nerve deficits especially V, VII and VIII (e.g. listeriosis).
- Spinal cord – varying degrees of ataxia, paresis or spasticity depending on location of lesion (e.g. fighting injury, Texel 'wobblers', spinal abscess, swayback).

Other adjuncts to diagnosis include: (i) collection of cerebrospinal fluid (CSF, usually the lumbosacral site will suffice); (ii) white cell counts (total and differential); (iii) blood biochemistry; and (iv) enzyme measurements.

Metabolic Diseases of Ewes (PT, Hypocalcaemia, Hypomagnesaemia)

See Chapter 6.

Scrapie

Scrapie occurs in most sheep-keeping countries except Australia and New Zealand. This ancient disease of sheep (a transmissible spongiform encephalopathy, TSE), with no proven risk to human health, has received much attention since the bovine spongiform encephalopathy (BSE) epidemic in cattle.

Originally there was a theory that BSE had got into cattle by feeding meat and bone meal contaminated by the scrapie agent, but this has been disproved; additionally BSE has never been diagnosed in sheep.

Scrapie is notifiable and is being tackled on an EU-wide basis with the aim of eventual eradication. There is a lot of information online – here in this book there is a summary of the main features. See the web site www.defra.gov.uk where the information is likely to be more up-to-date than this book can be.

In the last few years the number of diagnosed cases in the UK has fallen to a very low level, probably due, at least in part, to the genotyping programme which has been in place since 1991.

Classical scrapie

The disease

Scrapie is a chronic progressive degenerative TSE of adult sheep (and goats). The true incidence is difficult to assess as there is probably under-reporting and some cases may not be recognized by the farmer. Usually just one or two cases have occurred from year to year in a flock and so the disease has been of little importance to the average commercial sheep farmer producing prime lambs, but for the pedigree breeder where older sheep may be retained and where there has been a susceptible strain, the incidence could have been significant. The causal agent is thought to be a prion which causes an abnormal form of prion protein to accumulate in various tissues, especially the brain. It is very resistant to heat, formalin and UV light. Specified offals, including brain, spinal cord and spleen of sheep over 6 months of age are now disposed of by incineration to prevent possible recycling of infection and feeding ruminant-derived protein to sheep (and other animals) is now banned. The disease is probably transmitted prenatally as well as laterally in the lambing pens and fields (via fetal membranes), although the exact mechanism is not clear.

Genetic background and National Scrapie Plan (NSP)

It has long been recognized that there is a genetic susceptibility and much work has been done on this in recent years. The NSP was introduced in the UK to promote the breeding of resistant sheep by genotyping. This has been done by taking a blood sample, but some countries use small skin samples instead. The sheep is given an electronic identification by means of a rumen bolus at sampling.

Sheep have two copies of the PrP gene, which encodes for the production of PrP (prion) protein in the brain, each consisting of 256 amino acids. It is the production of abnormal PrP protein that leads to the development of the disease. There are three sites (codons) on the gene which are significant in determining whether an animal is susceptible or resistant if it meets the infection. These are 136, 154 and 171. There are five different alleles (see Table 14.1) and 15 different genotypes depending on which alleles are inherited from each parent.

The genotype is written as ARR/ARR, ARR/AHQ, ARR/VRQ, etc. and sheep are classed in five different categories (type 1, type 2, etc.) according to susceptibility. ARR/ARR is the most resistant type, VRQ/VRQ is highly susceptible. There is a breed variation in which genotypes predominate; for example Suffolks, where resistant rams have been selected for a number of years, are now mainly ARR/ARR. The Texel breed has a more complicated mixture of genotypes with a smaller percentage of resistant types. In contrast, primitive breeds may

Table 14.1. Alleles[a] involved in resistance or susceptibility to classical scrapie.

Codon		
136	154	171
A	R	R
A	H	Q
A	R	H
A	R	Q
V	R	Q

[a]A, Alanine; R, arginine; H, histidine; Q, glutamine; V, valine.

have few if any resistant genotypes (the 'wild' sheep genotype seems to be ARQ/ARQ).

The NSP has had a number of schemes within it but these have been closed except for a monitoring scheme for flocks wishing to export breeding sheep. There is random testing of deadstock and carcasses and a compulsory scheme for flocks where cases of classical scrapie are confirmed.

Clinical signs

Clinical signs vary and are usually seen in animals over 2 years of age. The most common are shown below and may be present singly or in any combination:

- A change in temperament or behaviour, sometimes the sheep appearing apprehensive, wild and excitable. Some other cases just look dazed and depressed and don't appear to focus properly.
- The most common and obvious sign is irritation, with scratching, rubbing, sucking and nibbling the fleece in an adult sheep. It is often seen at first as restlessness with the sheep darting about from place to place as if it has been bitten, and turning to nibble anywhere it can get at, as well as rubbing on posts and walls, etc. (watching a case one could imagine it feels like 'prickly heat'). The rubbing leads to a loss of fleece or hair over any area which the sheep can get at, including the head and both sides of the flank and hind legs. A fine rough stubble is left, often with scabs. If one rubs the sheep, it responds by standing still and nibbling its lips in apparent pleasure and relief. (Note that this test is applicable for all conditions causing pruritus and is not diagnostic of scrapie. If several sheep are affected, remember the possibility of external parasites!)
- Change in posture and gait – these usually show incoordination, so that the sheep becomes awkward to catch and to examine, and it won't relax and allow its head to be held comfortably. Rams can be quite dangerous and the difficulty in raising the head can suggest a high cervical lesion (e.g. from fighting).

If allowed to live, all these signs worsen over the weeks and sometimes months, and although there is sometimes a temporary stasis or even remission; all animals become progressively thinner and die sooner or later.

Diagnosis

The clinical signs are eventually diagnostic but obviously primary skin conditions (e.g. sheep scab!) have to be ruled out. If in doubt notify the local Animal Health Office which will arrange examination of the suspect animal, with slaughter and laboratory testing if the disease is suspected. Diagnosis is by immunohistochemistry or rapid immunochemistry, although definitive diagnosis is by brain histology looking for characteristic lesions (these differ between different TSE strains, e.g. the lesion 'profiles' of BSE and classical scrapie are different and distinctive). There is no diagnostic test currently in use in the live animal, although promising results have been achieved by taking tiny samples of rectal mucosa immediately adjacent to the anus, which contains lymphoid follicles. These can then be subjected to immunohistochemistry.

If scrapie is confirmed there is a compulsory scheme consisting of genotyping the flock and culling of susceptible genotypes. In the case of atypical scrapie, the flock is just monitored for 2 years.

Atypical scrapie – a complication

Fairly recently, a small number of so-called atypical scrapie cases have been identified as a result of surveillance in several countries including the UK. Here, out of over 100,000 samples tested, only about 80 were positive. These differ from classical scrapie and BSE in current diagnostic tests and may affect only one or a small number of animals (usually over 5 years of age) within a flock. Most notably, some cases have had the resistant ARR genotype. The significance of

atypical scrapie is unknown at present but restrictions are not currently placed on flocks with just the odd case.

Listeriosis

This is a sporadic disease of sheep (and less commonly, cattle) seen in many countries and in a number of forms. As encephalitis is by far the most common presentation, the disease as a whole will be dealt with in this chapter. The causal organism is commonly excreted in faeces and milk by apparently normal animals and is found widely in soil.

Types of disease seen are:

- encephalitis – by far the most common and significant form;
- abortion;
- diarrhoea and septicaemia;
- a few reports of kerato-conjunctivitis and also mastitis; and
- septicaemia and death in young lambs.

Encephalitic listeriosis

There has been an increase in reported incidents of listeriosis over the past 20 years in the UK, and it is now one of the most common causes of sporadic disease in individual adult sheep. It is seen particularly in the late winter months when silage is being fed. Poor quality, badly fermented (i.e. high pH) silage, particularly if it is contaminated with soil, and stored where air can get at it, can contain large numbers of *Listeria monocytogenes*. It is thought that the organism gains entry via any mouth lesions (e.g. changing teeth) and travels up the V cranial nerve to involve the nuclei of the V, VII and VIII cranial nerves in the brainstem (pons and medulla). The central nervous form appears to have a longer incubation period than other forms, cases usually occurring about 6 weeks after silage feeding starts. This is probably because the bacteria take some time to travel up the nerves to the brain. Thus, it is mainly a winter disease with a peak incidence in February/March, when many ewes are pregnant.

- Do not dismiss the possibility of the disease if silage is not being fed as the organisms are found in soil so could occur, for example, when sheep are being fed concentrates on the ground if grass cover is poor.

Clinical signs

The signs are variable but classically the animal is very depressed and there is unilateral V, VII and sometimes VIII cranial nerve paralysis (Fig. 14.1). This shows as: (i) drooping ear, eyelid and lip (facial paralysis); (ii) loss of facial sensation; and (iii) paralysis of cheek muscles with consequent dribbling and difficulty in eating and drinking. If the VIII nerve is involved, there is aversion of the head, propulsive circling and falling over on to one side. The animal is unable to respond to the menace test (also palpebral and corneal reflexes may be absent) which

Fig. 14.1. Encephalitic listeriosis causes facial paralysis so examine the head carefully – this sheep is normal.

may mislead one into thinking that the ewe is blind in that eye, and so confuse the condition with, for example, gid. Deterioration usually occurs over just a few days, leading to recumbency and death.

Diagnosis

The CNS form requires differentiating clinically particularly from:

- Hypocalcaemia (always check a recumbent ewe for facial paralysis before assuming it is hypocalcaemia).
- Middle and inner ear infection – confusion may arise because infection spreading from the ear locally to involve the facial nerve will produce classical signs of facial paralysis but without the extreme depression of listeriosis. These cases usually respond well to antibiotics and probably account for some supposedly 'cured' listeria cases!
- Other differentials are PT, gid, CCN and brain abscess.

A CSF sample obtained from the lumbosacral space may help to differentiate. Brain histology is necessary to confirm listeriosis; serology is not helpful.

If, as expected, silage is being fed, or was fed some time during the last 6 weeks, examine it for signs of: (i) poor fermentation; (ii) spoilage (mould); and (iii) soil contamination. Most silage contains some *Listeria*, although very rotten silage may be sterile. Be very careful when handling suspect material, including silage, particularly if you are pregnant or unwell.

Treatment

Therapy for the CNS form can be disappointing. The organism is sensitive to a wide range of antibiotics *in vitro*, but in spite of intensive treatment, many cases fail to respond. Success is more likely if treatment is early and vigorous; try crystalline penicillin, at least 10 mg/kg (one-quarter of the reconstituted vial) plus a high dose (1 mg/kg) of steroid given intravenously. Follow with procaine penicillin for 5 days. The common unilateral eye complications of corneal ulceration and uveitis also require protection from further damage and intensive local therapy.

Control

Vaccines have been tried particularly in Eastern Europe with equivocal results. The main control is in reducing contamination of silage.

SILAGE MAKING AND FEEDING Although a few incidents do occur at pasture, particularly when soil is exposed by heavy grazing, most follow silage feeding. It is almost impossible to avoid contamination by *Listeria*, so one must seek to avoid the conditions in which it multiplies.

- Make high quality silage with a pH <5 and a D value of >65%, with additives where necessary. Avoid gross soil contamination (e.g. mole hills) by rolling and not cutting grass too short, so that it has an ash content of <100 g/kg DM.
- Compact and completely seal the silage as soon as possible (the same day) and so avoid air getting into the sides and tops of clamps.
- With big bales make sure they are securely wrapped and do not get punctured when moved or stored.
- If there are persistent problems, try to avoid grazing sheep on fields from which silage will be made later in the season – grass may be contaminated if sheep are passing *Listeria* in faeces.
- Avoid feeding poor quality silage, which can be seen and smelt on the top or sides, as a layer or even in lumps in the middle, or left over in the troughs.
- Do not give more silage than can be cleared in 48 h maximum. Clear away uneaten silage before giving new.
- It is less risky to feed suspect silage to cattle.
- Live attenuated vaccines are available on the continent, but there is argument over their efficacy and they are not used in the UK. Some argue they can make matters worse rather than better, since

the disease may actually be an allergic response in a previously sensitized animal.

Other forms of listeriosis

The alimentary form often produces diarrhoea and even brief general illness, and if the ewe is pregnant, abortion may follow a week or so later, sometimes with retention of fetal membranes and subsequent systemic illness, but usually not encephalitis. However, others in the flock may show characteristic neurological signs of encephalitic listeriosis. Lambs may show septicaemia. For diagnosis submit faeces, vaginal discharges, fetuses and milk (if any) for culture and paired blood samples for serology.

Coenurosis (Gid, Sturdy, Bendro)

Good anthelmintic drugs for dogs and more strictly enforced legislation on carcass disposal has reduced the number of cases of this CNS disease in the UK. It does occur in many other countries but not in Australia and New Zealand. The causative agent is the metacestode (larval) stage of the life cycle of *Taenia multiceps* (*Coenurus cerebralis*), a tapeworm occurring in the dog (Fig. 14.2). Dogs usually acquire the infection either through scavenging dead sheep, or through being fed sheep heads obtained from abattoirs (now illegal). Surveys in the past have shown that about 5–10% of farm dogs in the UK were affected with the adult tapeworm and that they carried a mean worm burden of about ten adults. The adult cestode also occurs in the fox but the worms seldom reach maturity and become gravid, so foxes should not be blamed until every possible dog source of infection has been considered. The metacestode is also recorded in cattle, goats, horses and deer, but at a much lower incidence than in sheep, and, very rarely, in humans.

Once a sheep or lamb (pet lambs are often most vulnerable as they may be kept in close contact with dogs) has ingested the tapeworm eggs the larvae hatch and migrate in the bloodstream throughout the body but can only continue their development within the CNS. Over a period of 2–8 months, the *Coenurus* grows into a fluid-filled cyst containing around 100 scoleces. Multiple cysts have been thought to only occur occasionally in natural infections, possibly because an established cyst inhibits the development of further cysts, but recent magnetic resonance imaging (MRI) investigation has shown some animals do have more than one cyst.

Fig. 14.2. Dogs can transmit a number of diseases to sheep including the tapeworm responsible for causing coenurosis.

Clinical signs

Acute coenurosis usually goes unnoticed. Some 2–4 weeks after a sheep has ingested a large number of the tapeworm eggs, nervous signs such as ataxia, blindness, muscle tremors, nystagmus, excitability and collapse may be seen. Diagnosis is extremely difficult and is usually at PME where haemorrhagic tracts and migrating parasites are found in the brain.

The chronic form of the disease is much more frequently seen. The onset of clinical signs is usually 3–8 months after infection. The affected animal shows a variety of neurological deficits usually correlating closely with the location of the cyst within the CNS. About 80% of cysts are located in the cerebrum, 10% in the cerebellum, 5% are multiple cysts in several locations and the remainder is in the brainstem and spinal cord. Differentials are other space-occupying lesions such as abscess or tumour.

Affected sheep are usually identified initially by the farmer because of abnormal behaviour such as standing apart or failing to respond to the dog. Neurological examination should then reveal specific localizing signs (see Table 14.2), although it is important to consider combinations of signs rather than relying too heavily on any one deficit and if a farmer has become very good at recognizing early behavioural abnormalities, other neurological signs may not be very obvious. As the cyst develops, the clinical signs gradually increase in extent and severity, progressing to recumbency and death (over days or weeks) if the cyst is not removed surgically. Locating the cyst and attempting surgical removal is an interesting clinical challenge, though rarely financially viable. Use of MRI to locate will rarely be justified.

Note:

- Head tilt is the head rotated but the nose pointing forward. The cyst is usually on the side of the lower ear.
- Head aversion is the head rotated and turned to the side and usually down. The direction is not a good indicator to the side affected.
- Circling may be indicated by a plait of straw wound round the leg on the inside of the circle. The diameter of the circle may indicate whether it is in the right or left side of the brain and depth of the cyst.
- Unilateral menace deficit indicates that the cyst is in the opposite cerebrum because approximately 90% optic axons cross over; residual vision could be from the 10% that are in the nasal field which do not cross over.
- Spontaneous nystagmus indicates vestibular involvement. Cerebellar nystagmus is induced by eye movement and is an intention tremor of the eye muscles.

Table 14.2. Locating the cyst.

Indicator sign		Possible location
Behaviour	Depression	Cerebrum (rostral)
	Excitability	Cerebrum (temporal)
Head position	Tilt	Cerebellum or vestibular
	Aversion (turn)	Cerebrum
	Raised	Cerebellum
	Head tremor	Cerebellum
Movement	Circling – wide	Cerebrum (ipsilateral, superficial)
	Circling – tight	Cerebrum (contralateral, lateral ventricle or basal nucleus)
	Dysmetria (hypermetria)	Cerebellum
Posture	Wide-based stance	Cerebellum
Wheelbarrow and hemiwalking tests	Unilateral deficit	Cerebrum (contralateral)
	Bilateral deficit	Cerebellum
Vision	Unilateral blindness	Cerebrum (contralateral caudal)
	Bilateral blindness	Cerebellum
Eye movement	Nystagmus	Cerebellum or vestibular nuclei
Eye position	Strabismus	Brainstem nuclei of CNs III, IV and VI

Differential diagnosis

Unless there is a flock history, usually the furthest a diagnosis can go is of a space-occupying lesion. The disease usually occurs sporadically, although occasionally outbreaks are seen. It is characterized by the slowly progressive development of CNS signs in sheep over 4 months old (usually 1–2 years old and very rarely over 3). The chronic nature of the disease and the age of affected animals, together with a known farm incidence, will aid diagnosis. In addition, the presence of skull softening at or near the horn bud site, due to the intracranial pressure, is diagnostic but is not a reliable guide to the localization of the cyst.

Key distinguishing features of other neurological problems to be considered are:

- Listeriosis is an acute disease (2–3 days), with severe depression and facial paralysis, occurring as mini-outbreaks mainly in winter, and usually associated with silage feeding.
- CCN is also an acute disease (1–3 days), resulting in staggering gait, abnormal head position (star gazing), blindness, strabismus, tremors and fits, but responds to early therapy with thiamine.
- Louping-ill is a febrile illness characterized by 'leaping' movements and usually occurring as outbreaks among yearlings in tick areas and in the tick season (spring/autumn).
- Other conditions to be considered include CNS abscess, tumour, sway-back, intracranial injury, scrapie, PT, hypocalcaemia and hypomagnesaemia.

A differential white cell count and an analysis of CSF may aid diagnosis. Usually, the white cell picture is not altered by coenurosis so that a neutrophilia or a 'crossover' will generally indicate another cause of the CNS signs (e.g. listeriosis or abscess) but these changes do not occur invariably. CSF sampling should be carried out with care in cases where a raised intracranial pressure is suspected (e.g. coenurosis); only 0.5 ml should be taken from the lumbar cistern to avoid herniation of the cerebellum. In coenurosis, the CSF does not show any diagnostic changes; surprisingly, there is no eosinophilia.

Treatment

Lambs near killing weight should be sent for slaughter. Surgical removal of the cyst can be attempted in breeding animals or those too small for slaughter. With practice, the surgical success rate is as high as 80%. The operation should be carried out under general anaesthesia. We have routinely used pentobarbitone, but this is no longer available except for euthanasia, so induction followed by intubation and maintenance with a gaseous anaesthetic is the usual procedure. Anaesthesia can be induced with a mixture of ketamine (300 mg/3 ml) plus diazepam (30 mg/6 ml) in the same syringe, given to effect. Prior to surgery, crystalline penicillin (1.5 g) and dexamethasone (6 mg) should be given intravenously, although it may be wise to omit the steroid when dealing with a pregnant ewe. A C-shaped skin incision is made just caudal to the horn bud on the appropriate side (left or right cerebral cortex), or in the midline just rostral to the nuchal line (cerebellum). The skull is trephined (0.5–1.5 cm diameter) and a circular plate of bone removed.

Sometimes, the skull is so soft that scissors may be used. However, it is not safe to assume that the site of skull softening indicates the location of the cyst. The bone at the cerebellar site is at least 0.5 cm thick and may bleed. In trephining at the cerebellar site, it is important to avoid the transverse suture line about 1.5 cm rostral to the nuchal line, since this marks the position of the tentorium cerebelli and the transverse sinus and serious haemorrhage could follow. The meninges are cut and reflected to expose the cerebrum, which will bulge due to increased intracranial pressure. If the cyst is lying superficially, the translucent pale grey wall may be visible and should be immediately grasped with artery forceps. If the cyst is not visible, a 16 gauge (16G) needle with cannula (e.g. horse IV catheter) is inserted. When the cyst is

punctured, clear cyst fluid wells up the needle. The needle is removed and a 20–50ml syringe is attached to the cannula and some of the fluid is withdrawn. Cerebral cysts average 35ml but may contain over 100ml; cerebellar cysts contain 10–25ml. Suction is used to trap the pale grey cyst wall in the end of the cannula which is then elevated to allow the pedicle of the cyst wall to be grasped with artery forceps. Gentle tension is then applied until the wall is removed, draining more fluid from the cyst if required. The depth of the trephine hole at the cerebellar site may reduce the angulation of the cannula, making it difficult to grasp a cyst that is laterally situated in the cerebellum. Only the skin incision is sutured, and antibiotic cover is continued for 2 more days; steroids may also be necessary unless cyst removal was straightforward. The sutures are removed in 10 days and the skull heals in about 1 month.

The results of surgery can be very rewarding; uncomplicated cases usually make a good recovery within about a week, but excessive probing, haemorrhage and multiple cysts lead to a poor prognosis (better to euthanize rather than allow to recover from anaesthesia).

Control

- Worm dogs with a drug which is effective against cestodes, for example praziquantel at least as frequently as every 3 months.
- Do not feed uncooked sheep heads to dogs (now illegal).
- Dispose of sheep carcasses properly – burying is no longer legal in the UK.
- Avoid sheep grazing possibly heavily infected pasture, for example following sheepdog trials or if used by hounds.

Other tapeworms with larval stages in sheep

Dogs act as definitive hosts for several other tapeworms whose metacestode stage occurs in sheep.

- *Taenia hydatigena* is very common in sheepdogs and the larval stage, *Cysticercus tenuicollis*, occurs on the serosal surface of the liver and in the omentum in about 50% of lambs. Heavy infections are seen occasionally causing sudden death in young, usually pet, lambs. The liver is full of haemorrhagic tracts. Cysts cause some local condemnation of livers.
- *Taenia ovis* has a larval stage in sheep muscles and heavy infection may result in carcass condemnation.
- *Echinococcus granulosus*, a minute adult worm, gives rise to large hydatid cysts in livers and lungs, resulting in condemnation of offal. Clinical signs are not seen in sheep but hydatid disease is an important zoonosis in sheep-rearing areas in central Wales, occasionally elsewhere in the UK and in many other sheep-keeping countries. Successful eradication schemes have been carried out in many countries including Tasmania, New Zealand, Uruguay and Cyprus and one has been started in Wales.

Regular treatment of sheepdogs should be a component of sheep health programmes.

Cerebrocortical Necrosis (CCN, Polioencephalomalacia, 'Brain Rot')

This is an acute central nervous disease, said to mainly affect lambs 2–6 months old (not under 2 months, as it requires a functional rumen), but it also occurs sometimes in adults. It usually occurs sporadically and 'out of the blue' but there are occasional outbreaks affecting a small number of sheep, and lasting a few weeks following a history of change in food or worm drenching. It is associated with a deficiency of thiamine, probably induced by the microbial (by e.g. *Clostridium sporogenes* and *Bacillus thiaminolyticus*) production of thiaminase in the rumen, causing an initial generalized cerebral oedema followed by pressure necrosis.

Clinical signs

After a short period (a few hours) of aloofness, dullness and wandering, often 'star gazing' (even circling), the sheep becomes increasingly excitable (over another few hours, even up to 2 days) developing tremors, staggering, recumbency, opisthotonus and galloping movements. Opisthotonus, bilateral blindness and bilateral strabismus all point towards a high possibility of this disease. Animals may just be found dead; yet another cause of 'sudden death'. The above clinical signs should help to differentiate it from such conditions as pulpy kidney, listeriosis, gid, PT and hypomagnesaemia, but it requires some confidence to avoid a 'mass medication' approach at the first examination.

Diagnosis

- Clinical signs, especially opisthotonus, blindness and strabismus.
- Response to thiamine treatment.
- If the animal dies, PME shows bilateral discoloration of cortical gyri and bright white fluorescence under Wood's lamp of the sliced cortex.
- Histology.
- Rumen contents for estimation of thiaminase activity.
- Faeces for increased thiaminase activity, and blood samples for increased pyruvate and decreased transketolase activity can give supportive information, but they need special sampling procedures so contact the laboratory first.

Treatment

Give 200–500 mg thiamine (vitamin B_1) IV (slowly) and IM, repeat IM daily for a few days. If the case has been 'caught early' (i.e. before much irreversible necrosis) improvement can be expected within hours (not minutes), that is the sheep will be quieter and have fewer convulsions, and often there is complete recovery within days, although vision often takes longer to return.

Control

It is reasonable to look at the diet and perhaps suggest changing it, for example remove from that field or take off that concentrate or lick (e.g. molasses), but it is only making a shaky guess. There is some evidence that the contacts may appear not to be thriving and show signs of scouring, so have a look at them too. They might be injected with 500 mg thiamine, but the incidence does not usually justify such intervention.

Tetanus

This disease, most commonly seen in young or growing lambs, should not occur if clostridial vaccination has been correctly carried out, since the vaccine is highly effective. It does, however, rely on passive transfer of antibodies via the colostrum, so the disease can occur in vaccinated flocks where colostrum management has been poor. It also needs to be remembered that passive protection only lasts 12–16 weeks, so lambs require active protection after this stage. Most lambs are at risk, since the causal organism, *Clostridium tetani*, is widespread in the environment in the form of very resistant spores. Routine procedures such as tailing and castration provide perfect sites for the bacteria to multiply and to release the toxins which cause the characteristic clinical signs. Adults are also at risk through injuries or associated with bad lambings.

Clinical signs

These should be easily recognized: (i) rigidity of muscles, with the affected animal in lateral recumbency with limbs stiffly extended, jaw tightly closed, tail and ears held stiffly; (ii) spasm of the third eyelid; and (iii) in older animals, bloating of the rumen since they are unable to eructate gas.

Treatment

This is usually unrewarding – consideration should be given to euthanasia on diagnosis. Penicillin is effective against the bacteria and antitoxin can be given to try to 'mop up' free toxins; sedation to help relax muscles and good nursing may help, but the prognosis is always poor.

Prevention

Implementation of a comprehensive clostridial vaccination policy for the flock including vaccination of rams, ewes before lambing and lambs from 10–12 weeks. Remember that two injections 4–6 weeks apart are necessary to provide active immunity. The 7-, 8- and 10-in-1 clostridial vaccines all provide tetanus coverage.

Other Neurological Disorders

- Focal symmetrical encephalomalacia is associated with *C. perfringens* type D enterotoxaemia, causing vague neurological signs, recumbency and death mainly in growing lambs.
- Rams commonly sustain fighting injuries, the results ranging from instant death, to concussion, to neck injuries.
- Wobbler syndrome in Texels – this is a recently-recognized problem, affecting both males and females, which shows up in young adults and has the appearance of 'wobbler' horses and dogs. Affected animals develop a progressive ataxia and swaying of the hind quarters. Radiographic investigations using contrast medium and PME have shown the problem is usually between C6 and C7 vertebrae, where a fat pad protrudes into the vertebral canal from above, pressing on the spinal cord. There may be a familial tendency.
- 'Kangaroo gait' affects ewes, usually during lactation and is a neuropathy of the nerves supplying the forelimbs, especially the radial nerve. Affected animals have difficulty using their forelimbs and, as the name suggests, have a strange gait resembling that of a kangaroo. Surprisingly, most cases recover after a few weeks. Weaning may hasten this. Until they recover they need general nursing care to make sure they are able to get sufficient to eat.
- Ryegrass staggers is occasionally seen in the UK and more commonly in Australia and New Zealand where it can be a serious flock problem. This is a mycotoxicosis (a toxin produced by an endophyte which is found in ryegrass), causing staggering and tremors. Affected animals may fall into ditches or streams and drown, or are attacked by predators. Recovery usually occurs if removed from the affected pasture.

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15

Eyes, Ears and Nose

Eye Diseases

Contagious ophthalmia (ovine infectious keratoconjunctivitis, pink eye, heather blindness)

This is a very common disease of sheep's eyes worldwide. Usually, there is only superficial irritation recognizable by: (i) conjunctivitis; (ii) scleral congestion; and (iii) excessive lachrymation, blinking and some blepharospasm. Some cases, however, particularly adult sheep, show corneal inflammation with blood vessels and pannus spreading from the corneo-scleral margin, and progressing to shallow corneal ulceration and temporary blindness. Both eyes are usually affected but not always simultaneously or to the same extent. Badly affected sheep can find it difficult to find food and are accident prone.

Outbreaks often arise after movement, probably because *Mycoplasma conjunctivae*, the organism most commonly associated with the disease in the UK, is spread by close sheep-to-sheep contact rather than by vector or airborne transmission. It may also be introduced by 'carrier' animals, as many normal animals carry the organism. Bacteria such as *Branhamella ovis* and *Staph. aureus* may play a role in increasing the ocular reaction. *Chlamydophila* spp. have been isolated from some outbreaks in the UK and, commonly, in New Zealand.

Without treatment, the duration of the disease is very variable; if only the sclera is involved, complete resolution may occur in a day or so, but if the cornea is involved, inflammation may persist for 3 or 4 weeks. However, many carriers remain and relapses and new infections are frequent, with the result that the disease persists in the flock.

The disease causes discomfort and individual cases certainly merit treatment when there is obvious blepharospasm, which usually indicates corneal involvement. A single injection of suitable antibiotic (e.g. oxytetracycline) is the most practical treatment, particularly if large numbers of animals are affected. Topical application of an ophthalmic antibiotic preparation may be appropriate for individual cases but needs to be carried out several times daily. Remember that *Mycoplasma* spp. are not susceptible to penicillin-based products. Subconjunctival injection of antibiotic may be necessary for severe cases or even suturing the third eyelid. Separation from the group may be necessary in order to ensure proper care and treatment.

In an outbreak, it is worth considering injecting all the immediate contacts with oxytetracycline: this routine can also be applied to all newly purchased sheep, for

example rams, in order to reduce the risk of importing fresh strains of *M. conjunctivae* into the flock, but the effectiveness of this cannot be guaranteed.

The frequent movement and mixing of sheep are obvious hazards and should be kept to a minimum.

It is worth noting that this condition is very different in its etiology, pathogenesis and severity from so-called 'New Forest' disease in cattle, and that some ocular preparations, for example the penicillins for use in cattle, are generally inappropriate for sheep as they are not effective against *Mycoplasma* spp., although they may have some effect on secondary invaders.

Silage eye

Listeria monocytogenes may occasionally be implicated in eye problems as in 'silage eye' in cattle, although this is uveitis/iritis rather than keratoconjunctivitis so should be distinguishable clinically. Infection probably gains entry to the eye as a result of the sheep burrowing their heads into silage when feeding so it is usually associated with big bales but may also occur if any silage is not cleared away regularly (uncleared silage allows multiplication of the bacteria). Examination of the eye may show folding of the iris with inflammatory changes progressing from the edges of the pupil accompanied by clumps of fibrin underneath the cornea. Treatment is by subconjunctival injection of antibiotic (oxytetracycline) together with a small amount of steroid.

Prevention can be attempted by: (i) avoiding contamination of silage with soil; (ii) preventing damage to the wrapping of big bales; and (iii) providing only sufficient silage so that it is eaten within 2 days.

Bright blindness (clear blind, glass-eyed)

This is an irreversible blindness in adult sheep, mainly reported in flocks in the north of England, but occurring in hill flocks in other areas. The condition affects both eyes equally and simultaneously. The retina shows progressive degeneration with atrophy of the rods and cones; the tapetal arteries and veins are narrowed and there is a marked green reflection from the tapetum. Ophthalmoscopic examination of the retina is easy because the pupils are dilated and the cornea and lens are clear.

It is caused by a toxic factor in bracken, and may require several seasons of bracken consumption before signs become apparent; it is therefore only seen in adult sheep. The prevalence may be over 5% and usually becomes apparent in the autumn following the bracken growth season.

As the condition is slowly progressive, the sheep have time to adapt and the blindness may only become apparent when the sheep are driven or placed in strange surroundings. They are not unwell, appearing perhaps more alert than usual and are not easy to catch. They are, however, more prone to accident and to get lost. They require culling.

Entropion

Entropion (Fig. 15.1) is common in newborn lambs in the UK, involving one or both lower eyelids. The incidence varies from flock to flock but cases occur in many flocks and some receive inadequate attention resulting in unnecessary discomfort and

Fig. 15.1. This lamb has entropion which urgently needs correcting.

corneal damage. Eyes of any lambs showing ocular discharge should be carefully examined for entropion, before assuming it is an eye infection. Some flocks have a distressing number of severe cases and there is sometimes good evidence to suggest a breed factor, with a recessive gene involvement. Entropion can also occur in very thin adult sheep as a result of loss of the fat pad behind the eye.

Treatment

- In mild entropion the eyelid can be easily everted by finger pressure, particularly in newborn lambs. In flocks with an entropion problem, an 'eyelid' check should be routine with correction done as necessary as soon as possible after birth.
- More severe cases, where the lower eyelid is turned in a lot causing blepharospasm, keratitis and corneal ulceration, require more vigorous attention, such as:
  - SC injection of a liquid below the margin of the lid, after manual correction, sufficient to produce a bleb which everts the lid. Liquid paraffin is effective, but an antibiotic preparation (e.g. penicillin) is more sensible as this will also treat any infection that may have resulted.
  - Placing a tuck in the skin below the lid with 14 or 16mm Michel's clips.
  - Surgical correction as in dogs; as the above methods usually work well, this is rarely necessary.
- Most cases that have not been corrected immediately after birth merit antibiotic eye ointment at the time, and if possible for the next day or so to deal with any infection which has become established.

Control

- Consider the breeding lines. Has a new ram been used?
- Mark and record each case and don't breed from them. This is easier said than done with bought-in animals, as cases corrected at birth are undetectable.

Other genetic defects

Occasionally lambs, particularly Texels, are born with very small eyes (microphthalmia) or no eyes (anophthalmia). In some breeds with a gene for four horns (e.g. Jacob or Hebridean) sheep have a defect in the upper eyelid which, if severe, can predispose to eye infections and blindness. Both conditions are likely to be inherited; therefore breeding lines should be investigated.

Ear Problems

The ear may be involved in a number of different diseases (Fig. 15.2), but allowing the ears to droop is usually a general sign that a sheep is unwell. (However, note that normal ear position is very variable between breeds – contrast for example, Suffolks with Border Leicesters for extremes of normal appearance.)

Specific problems include:

- Paralysis of (usually one) ear – see listeriosis and middle ear infection (see Chapter 14).
- Rubbing and shaking, with haematoma formation – see *Psoroptes ovis* (*cuniculi*) infection (see Chapter 12).
- Scabbiness – sunburn, dermatophilus, orf, ringworm (see Chapter 12).
- Swelling of the ears and head – see photosensitization (see Chapter 12) and bluetongue (see Chapter 17).

Fig. 15.2. This Zwarbles ewe has some damage to both ears.

Nose Problems

Nasal discharge

This is commonly seen in various types of pneumonia such as pasteurellosis, atypical (*Mycoplasma ovipneumoniae*) pneumonia, in OPA and as a result of nasal bots. Regurgitation of saliva or rumen contents may be mistaken for that associated with pneumonia. Look carefully at the type and quantity of discharge:

- Copious clear mucoid discharge, which increases in amount when the sheep's rear end is lifted (the 'wheelbarrow test'), accompanied by weight loss indicates OPA.
- Dirty, possibly bloodstained, discharge accompanied by sneezing may indicate nasal bots.
- Serous discharge accompanied by raised temperature may indicate pasteurellosis.
- Serous discharge with coughing but with little or no raised temperature may indicate atypical pneumonia.
- Clear mucoid discharge (saliva) in a collapsed heavily pregnant or recently lambed ewe may indicate hypocalcaemia.
- Green-coloured discharge with typical smell indicates regurgitated rumen contents.

Skin lesions around the nose

The most common causes are infection with orf virus and staphylococcal infection of the skin. See Chapter 12.

Nasal bots

The fly *Oestrus ovis*, which lays larvae inside the nostrils of sheep, is found worldwide but is rarely a serious problem in the UK. The larvae move into the sinuses where they develop over about 6 weeks, or much longer in winter, before the sheep sneeze them out on to the ground where they pupate and complete their life cycle. The adult flies cause a nuisance to the sheep and the larvae cause nasal discharge. Anthelmintics containing ivermectin, doramectin or moxidectin are effective so flocks using these products for worm control will also have nasal bots controlled.

Enzootic nasal tumour

This is not a problem in the UK but may be seen in some other countries, for example in southern Europe. It is caused by a retrovirus similar to that causing OPA.

16

Ticks and Tick-borne Diseases

Sheep act as host for many ticks throughout the world, especially in tropical countries and a number of species of tick are responsible for the transmission of many diseases which will not be discussed here.

The Sheep Tick (*Ixodes ricinus*)

Hard ticks belonging to the genus *Ixodes* are the most important in sheep and the species which is found most commonly is *I. ricinus*, the so-called sheep tick, though it is not host specific and will feed on many mammalian and avian species. The tick is of importance in sheep, cattle, humans and grouse but it is also found on hosts such as deer and other wildlife.

I. ricinus has a wide geographical distribution from the Atlas Mountains in North Africa in the south to Iceland, Sweden and Russia in the north and it extends eastward to Iran and westward to Ireland. It is found extensively throughout central Europe including Austria, Switzerland, Poland and Slovenia. It is the common tick found on farm animals including sheep in the UK. Other species of tick belonging to the genus *Ixodes* occur in North America.

It has a three-host life cycle in which less than 3 weeks of the 3-year cycle is spent on the host. Larvae and nymphs can feed successfully on birds and small wild animals, but the adult female needs a large animal host (sheep, cattle, deer) in order to produce eggs. All stages of this tick will become attached to humans and may transmit Lyme disease and louping-ill. Since 98% of the life cycle is spent off a host, the success of the tick is very dependent on the external environment.

Geographical distribution in the UK

The ticks are dependent on suitable habitats for the stages off the host, which require 100% humidity. They are found, therefore, mainly on rough hill grazing in Scotland, the Pennines, the Lake District, Wales, Devon and Cornwall and a few other small areas. However, small populations of *I. ricinus* are successful in finding suitable but small habitats in other less typical regions such as Richmond Park in London where they may become attached to domestic pets and humans as well as their usual deer host. The areas where ticks are found in large numbers have an annual rainfall of over 100 cm (40 inches). The vegetation is bracken, heather, rushes and coarse grasses and the number of ticks increase with the thickness of the vegetation mat (Figs 16.1 and 16.2). Pasture improvement, involving

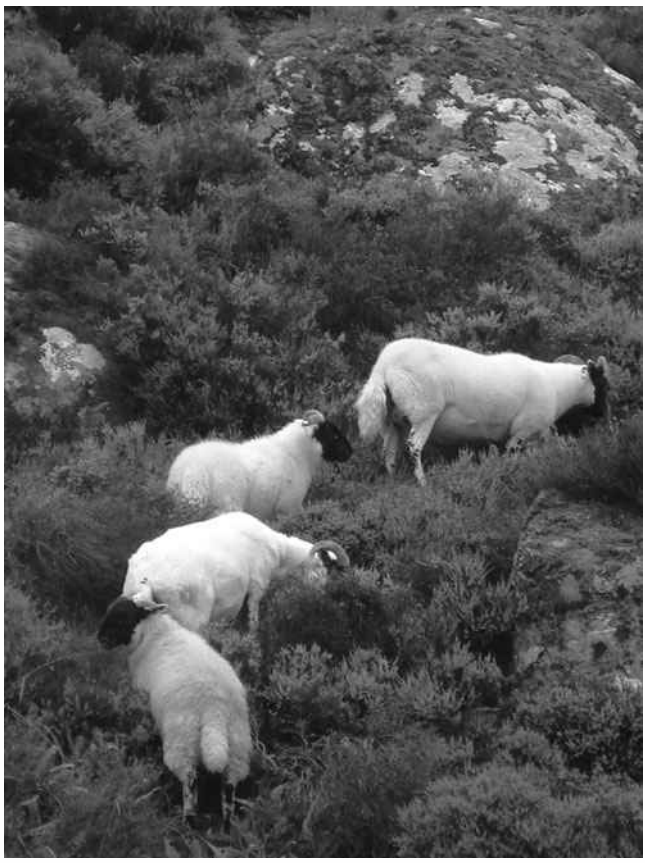

Fig. 16.1. Moorland with heather is a typical tick habitat.

Fig. 16.2. This type of vegetation is also likely to be a tick habitat.

drainage and re-seeding, renders conditions unsuitable for ticks.

Seasonal distribution

In most areas, there is a tick rise (i.e. the ticks climb up the vegetation and thus become available to passing hosts) in spring and another in autumn, but the precise timing varies from year to year, particularly the time of appearance of the major population in spring. This is always close to the main lambing time, but may occur when lambs are plentiful or sparse, which will influence disease occurrence.

Small numbers of *I. ricinus* can be found on sheep throughout the year and in a few areas (Northumberland and south-east Scotland) there is no autumn rise. Spring rise and autumn rise ticks are different populations.

Flock distribution

The number of ticks found throughout the season on sheep increases with the age (or size) of the sheep. In one count on a heavily infected farm, ewes had 400 adult females, yearling hoggs 300 and lambs 50, together with about five times as many nymphs as adults, and 1000 times more larvae than adults.

Sheep distribution

Ticks attach initially to the ears, nose, lips and feet and they migrate to become attached to the hairy parts with only small numbers in the woolly areas. Adults migrate further than nymphs and larvae hardly migrate at all.

Importance

Ticks are responsible for irritation and a certain amount of blood loss, particularly in young lambs (tick worry) and for the transmission of: (i) tick-borne fever (TBF); (ii) tick-bite pyaemia; (iii) louping-ill; and (iv) Lyme disease. The tick-transmitted diseases are more important than tick worry because heavy infestations are uncommon and the blood loss is over a period of some weeks, which allows for compensation, except in lambs. Grouse are especially susceptible to louping-ill and vaccination of sheep and treatment for ticks has been shown to reduce the infection and mortality in grouse on moors where they are living together.

Control

Tick control is often affected by improvement in grazing associated with increases in stocking rate. It can be done by pasture improvement and by chemical treatment of sheep.

Pasture improvement

Many hill farmers drained and re-seeded the relatively low pastures bordering on the high hills in order to improve lambing percentage and live-weight gains in the lambs. This had a dramatic effect on tick populations. However, if some grazing is improved and is used for ewes late in pregnancy and early in lactation, a waning of immunity to tick-borne disease (especially louping-ill) may result, followed by disease when the sheep are later turned on to the tick-infested hill grazings. A similar problem may occur with sheep which are away-wintered on lowland pastures. Heather burning every 5–7 years reduces but does not eliminate tick populations.

Chemical treatment of sheep

Sheep can be treated for ticks by 'plunge-dips', 'pour-ons' or 'spot-ons'.

Plunge-dipping in a bath can only be done with diazinon, an OP compound. There are strict regulations for OP compounds to reduce the likelihood of toxicity to the operators who must also be licensed after taking an appropriate course. Dipping lambs can lead to mismothering so is used less commonly since the advent of the other products.

'Pour-ons' and 'spot-ons' use cypermethrin or deltamethrin (SP compounds) and are applied with a special gun along the midline from the shoulders back but avoiding the tail of lambs in order to prevent mismothering. The drug spreads over the skin surface in the sebum and will control ticks for up to 12 weeks in adults and 4–6 weeks in lambs. These 'pour-ons' are particularly useful for lambs as mismothering often occurs after dipping. Operators should wear protective clothing with SP compounds.

Ewes are dipped to reduce the pasture infestation in successive years and lambs are protected to try and reduce the number of ticks which they pick up. It is recommended that all ewes should be treated either before lambing at the end of March or beginning of April or preferably after lambing before going to the fell. Lambs may be treated from 4 days old.

Sheep should be re-treated in autumn.

Diseases Transmitted by Ticks

Tick-borne fever (TBF)

This is caused by a rickettsia, now called *Anaplasma phagocytophilum*, formerly *Ehrlichia* (*Cytoecetes*) *phagocytophila*. A similar organism of close antigenic identity is found in cattle. The organism is transmitted by the nymph and adult of *I. ricinus* and most ticks are infected throughout the world including the USA. It causes disease in a number of hosts including humans where it is responsible for human granulocytic ehrlichiosis.

The rickettsiae invade the circulating neutrophils and monocytes and results in their destruction, thus causing a neutropenia. It is this feature, combined with pyrexia, which gives rise to clinical signs and predisposes the sheep to tick pyaemia and increases the severity of louping-ill. In experimental studies, it has been shown to reduce the immune response to many antigens and infections such as pneumonia so it is possible that it predisposes to other infections in the field.

Clinical picture

Since most ticks are infected, lambs in a tick area become infected early in life. The clinical signs are mild and are usually not noticed – the lambs may be listless, fail to suck and have a temperature rise (up to 41°C) for a few days. There is some evidence that some strains are more pathogenic than others. Organisms can be found by Giemsa stain of a thin blood film. Immunity then develops and is probably short-lived, but is reinforced throughout life, producing 'acclimatized' sheep. There are no antibodies to TBF in the colostrum, so lambs become infected as soon as they encounter ticks. More severe signs are seen in adult sheep brought from a tick-free area. The high temperature which follows exposure to ticks may result in abortion in pregnant ewes (unusual because the time of year is usually wrong) and temporary infertility (for about 3 months) in rams. This is of considerable importance since rams are usually purchased at September/October sales and become infertile just at the time that they are turned out with the ewes.

Control

The organism is susceptible to sulfonamides and broad-spectrum antibiotics, but it is more important to obtain satisfactory immunity in lambs and to produce acclimatized sheep. Do not move non-immune rams or pregnant ewes into a tick area, at least during the breeding season.

Tick-bite pyaemia

This is a generalized infection with *Staph. aureus*, a normal skin commensal, of lambs of 2–16 weeks old associated with tick bites and usually predisposed by TBF. Neutropenic lambs have been shown to be highly susceptible to staphylococcal septicaemia. The disease is either a septicaemia resulting in death or the bacteria localize in many organs, producing abscesses in joints, CNS, liver, lungs, etc. Lambs are rarely infected before 2 weeks of age, probably because the neutropenia from TBF is not

evident before this time and also because titres of staphylococcal antibodies derived from the colostrum wane at about 2 weeks.

Clinical signs

These vary with the site of the abscesses and include: (i) lameness with painful enlarged joints; (ii) paralysis of the hind legs due to an abscess in the spinal cord; (iii) signs of meningitis due to abscesses in the brain and meninges; or (iv) unthriftiness due to abscesses in liver, lungs, kidneys, etc.

The septicaemic form shows in sudden death. The disease occurs wherever *I. ricinus* is found and is of great economic importance. It has been shown that half the lamb losses and virtually all the lameness in lambs on a tick farm is associated with tick-bite pyaemia.

Treatment

Antibiotics (e.g. penicillin) are effective against the organism and if lambs are treated early, the clinical signs may regress and the lambs recover. Lambs with spinal abscesses or with multiple abscesses in the liver and lungs are unlikely to recover despite treatment and it has been suggested that penicillin only hastens the recovery of lambs which were likely to recover spontaneously.

Control

- Tick control and control of TBF. Synthetic pyrethroids applied to 7-day-old lambs before putting on tick habitats will delay infection until they are 4 weeks old, and disease is then much less severe.
- LA penicillin or oxytetracycline have been employed prophylactically just prior to exposure to ticks, when the lambs are sent with the ewes to the fell, but these only last for a few days and are not likely to influence disease significantly.

Louping-ill ('trembling')

This is a virus disease (flavivirus) which affects sheep and many other hosts, including cattle, deer, man, dogs and red grouse. Typically, there is an initial viraemia about 2–6 days after the tick bite, during which there is a febrile reaction (42°C). The majority of acclimatized sheep show few clinical signs and even susceptible sheep will usually recover rapidly. In some sheep, however, the virus invades the CNS and severe clinical signs and death follow. There is evidence that a primary TBF infection at the same time increases the likelihood of invasion. The virus is transmitted stage to stage by *I. ricinus* and only about one in 1000 ticks is infected. Some tick areas have no louping-ill virus present at all.

Clinical signs

Disease is seen in all ages of sheep from as early as 2 days old (suggesting prenatal infection) to aged ewes. Lambs are dependent on colostral immunity for protection, which is strongest when immune ewes are exposed to re-infection a few weeks before lambing. Changes in grazing practice may prevent this natural boost to the immunity of the ewes and many lambs may then become clinically affected. Lambs may be found dead, but the typical signs are those associated with CNS involvement and are variable in nature. They include: (i) resting the head on the ground; (ii) head pressing; and (iii) abnormal jerky gait ('leaping'), staggering, circling and paralysis. Fine tremors of the facial muscles and ears, nystagmus and twitching or nibbling of the lips may be seen. The disease progresses within 1–4 days to coma and death, often assisted by predators.

Usually only a small proportion of the flock is affected, but outbreaks may be explosive with a high mortality. Animals which have recovered from the viraemia have a solid life-long immunity and specific antibody can be detected in their blood.

Diagnosis in a severe outbreak is usually made on clinical grounds but CCN, listeriosis, swayback, gid or abscesses must be considered. There is a typical histology of the brain and virus can be recovered at the laboratory by mouse inoculation. There is a serological test for antibody. The disease is particularly important in red grouse where about 80% of infected birds die.

Treatment and control

Treatment is not effective but careful nursing indoors may save some cases.

The aim of control is to ensure that ewes are immune and that lambs are protected passively. There is an inactivated vaccine available which is a white oily emulsion. Warming of the vaccine to body temperature facilitates injection. The recommended procedure for ewes is that 1 ml should be injected subcutaneously on the side of the neck between tupping and mid-pregnancy and not later than 1 month before lambing with a boost every other year before lambing. It may be necessary to protect ewe-lamb replacements before the autumn tick rise.

Lyme disease

This zoonotic infection is caused by the spirochaete *Borrelia burgdorferi* and is transmitted in the UK by *I. ricinus*. Serological studies show that in Scottish sheep as many as 40% of hoggs showed antibody, with only small numbers of lambs and older ewes showing significant antibody titres. No clinical signs were seen associated with the positive serology though severe lameness has been described in 6-month-old lambs in Norway associated with high titres of antibody and heavy tick infections.

It is possible that sheep act as a reservoir of infection of *Borrelia* from which ticks become infected and thus transmit the infection to more susceptible hosts like dogs and humans. It is an increasingly recognized disease of hill walkers as ticks easily become attached to humans often without it being realized.

Summary of Control Methods for Ticks and Tick-borne Diseases

Possible control methods for ticks and the diseases they transmit are:

- Dip ewes around lambing and in the autumn (and possibly in the summer but this needs to be related to treatment for blowfly strike) to reduce tick populations.
- Apply 'pour-on' or 'spot-on' preparations to lambs before returning them to tick areas.
- Administer LA penicillin or oxytetracycline to lambs before returning to tick areas.
- Take special care over the introduction of new rams and ewes.
- Vaccinate ewes against louping-ill every other year and lambs before the autumn rise.
- Carry out heather burning of areas on the fells on a 7-year rotation.

17

Notifiable Diseases and Diseases Exotic to the UK and Northern Europe

Notifiable Diseases

All sheep-rearing countries take measures to avoid the introduction of serious diseases and to control them in the event that they do become introduced (Figs 17.1 and 17.2). These measures include the legal requirement for owners of animals to report sheep showing clinical signs associated with a list of notifiable diseases to government veterinary authorities. In addition, many countries require the reporting of other named diseases which do occur in the country but for which special measures are being taken to control or eradicate the disease or which are of particular importance, notably of zoonotic potential.

Australia has an extensive list of 93 notifiable diseases for all species of farm animals and horses for the whole country and in addition, individual states are permitted to include other diseases. This national list contains around 20 diseases of sheep including exotic diseases which do not occur in the country including bluetongue, *Brucella melitensis* infection, contagious agalactia, EAE, FMD, MV, peste des petits ruminants, Rift Valley fever, sheep pox, sheep scab and scrapie. It also contains diseases like ovine pulmonary adenomatosis and paratuberculosis (Johne's disease) which do occur in some states and which form part of an official control scheme.

In the UK, the list of notifiable diseases includes many of the exotic diseases in the Australian list and like Australia, many of them have never been seen in the UK and one, sheep pox, was last seen in 1866. There is one endemic notifiable disease of sheep in the UK, namely scrapie, which is discussed in Chapter 14. There are two notifiable diseases of sheep which are not endemic but have been seen in recent years and which are of considerable significance – these are bluetongue and FMD which are subject not only to UK national legislation but also to EU measures. These two diseases will be discussed here and some of the more important notifiable diseases which occur in some parts of the world but not in the UK or Australia will also be included in this chapter.

Foot and mouth disease (FMD)

The massive outbreak in the UK in 2001 resulted in over six million animals being slaughtered, around five million of which were sheep (this number does not include young animals at foot, especially lambs which were not recorded. An estimate of their number resulted in the often quoted

Fig. 17.1. These sheep kept in the Mediterranean region may suffer from diseases that could potentially spread to other areas of Europe.

Fig. 17.2. Sheep specialists should have some knowledge of exotic diseases such as those that may affect these sheep in South Africa.

figure of ten million animals being slaughtered and valued at around £4 billion).

The primary outbreak was in pigs but large numbers of sheep farms were affected early in the outbreak, associated with the movement of sheep via a market in the affected area to many other areas of the UK, since there was a considerable delay in reporting disease on the pig farm.

Clinical signs

The disease is severe in young lambs with high mortality and in pregnant ewes, resulting in abortion but many sheep do not show obvious clinical signs. Where clinical signs occur, sudden, severe lameness, usually in more than one leg, is the most obvious feature but since lameness due to other causes

is so common in sheep, it is easy to miss the significance of the lameness. Sheep lie down and are often unwilling to rise or stand in a crouching position.

Early lesions are blanching and separation of the interdigital skin at the coronary band occurs; after a day or two, the lesions become ulcerative and granulomatous and are easily confused with early FR.

Mouth lesions are less obvious and the sheep may eat normally with no excessive salivation as occurs in cattle. Blanching and separation of the mucous membrane occurs, particularly on the dental pad and less often, on the tongue and ulceration occurs within 1–2 days of the initial blister. The careful examination of the mouths of many sheep during the outbreak led to the recognition of mouth lesions that looked similar to FMD but were due to other causes, probably associated with mechanical damage. Initially called 'OMAGOD' (ovine mouth and gum obscure disease) or idiopathic oral ulceration, they made diagnosis difficult. A recent retrospective study on stored samples has shown that 23% of premises suspected of having FMD did not actually have the disease. Since the aim was to kill the animals on a suspect farm within 24 h, this was often before viral confirmation was possible which has led to the search for 'pen-side' tests for on-farm use which is showing some success.

If there is any doubt, ask the boss or the Divisional Veterinary Manager to examine the sheep and stay on the farm until they have.

Control

FMD virus has entered the UK in the past by: (i) imported carcasses from South America; (ii) wind from the continent of Europe; and (iii) escape from a laboratory which worked on the disease. It is also possible that scraps of infected meat from ships or illegal imports may bring this disease into the UK. Primary cases were often seen in swill-fed pigs and this practice has now been banned. Careful surveillance of the FMD status of countries from which meat is imported helps to reduce the risk associated with the meat industry. All countries in the EU fall into the highest international FMD status of FMD-free and no country is allowed to use prophylactic vaccination. In addition, the movement of animals in the UK is now much more carefully regulated which would reduce the risk of rapid spread which occurred in 2001.

The control strategy relies on the rapid reporting of suspect animals by farmers and veterinary surgeons followed by diagnosis and typing of any FMD virus isolated. All susceptible animals on the infected premises and on farms which are considered to be dangerous contacts are slaughtered. The EU directive allows emergency vaccination along with the implementation of the slaughter policy but a decision to vaccinate can only be made by the Government Secretary of State for Agriculture after a positive diagnosis has been made. It is believed that this vaccination policy, perhaps of a ring nature of susceptible animals on farms surrounding the infected premises, will limit the spread of the disease. Banks of vaccine and antigens from a range of FMD strains are held in EU countries.

Bluetongue

Bluetongue is a viral infection caused by bluetongue virus (BTV) of ruminant animals which occurs widely in tropical and subtropical countries throughout the world. There are more than 20 serotypes of BTV, each of which is immunologically distinct.

In recent years, there have been many outbreaks in several Mediterranean countries and it has been suggested that global warming has encouraged the northern and western spread of the insect vector, the midge *Culicoides imicola*. It is believed that the virus spread into Europe from Turkey and North Africa and strains of serotypes 2, 4, and 9 have been identified in south-east European countries.

In summer 2006, outbreaks of disease associated with a sub-Saharan strain, BT8 occurred in the Netherlands and Belgium and spread to Germany and France. The origin of this strain has not been proved. The inevitable spread to the UK occurred in September 2007, when cases were seen in East Anglia, believed to be explained by

infected midges being blown across the North Sea. The disease spread to other parts of south and south-east England.

Bluetongue does not affect or infect humans and so has no public health significance.

Epidemiology

The virus is transmitted by species of biting midges of the genus *Culicoides*, some of which are widespread in the UK. Direct animal-to-animal transmission does not occur. In temperate climates, the adult fly is not active during the winter and clinical disease is seen only from July to December so it was believed that the outbreaks on the continent would die out during the winter. However, this did not prove to be the case and outbreaks in northern Europe were more numerous and more virulent in summer 2007 than in 2006, and in 2008 there were over 30,000 outbreaks in the EU. It has been shown that bluetongue overwinters in northern Europe more effectively than had been expected probably due to a combination of: (i) animals maintaining a viraemia for a long time; (ii) the survival of some midge species; and (iii) the transplacental transmission of the virus.

Clinical signs

Usually, cattle remain as carriers with few signs and the disease is more virulent in sheep but in the 2007 Netherlands cases, cattle were also seriously affected. Mortality in sheep can be as high as 70% but the severity is dependent on the breed of sheep.

In sheep, the disease is characterized by fever up to 42°C that may last for several days. Increased respiration and hyperaemia and swelling of the lips, mucous linings of the mouth and nose and eyelids are seen, accompanied by salivation and frothing at the mouth. Nasal discharges are common. There is sometimes oedema of the head and neck. The tongue may be cyanotic, giving rise to the name bluetongue. Laminitis and inflammation of the coronary band may result in lameness and reluctance to stand. Animals lose condition rapidly, including muscle degeneration. Infection during pregnancy may result in abortions and congenital abnormalities.

PME shows typical haemorrhages in the pulmonary artery.

Control

As the result of a successful vaccination campaign, the UK was granted bluetongue-free status in July 2011 which removed the restrictions on the export of cattle and sheep introduced in 2007. BTV cases were only identified in home-bred ruminants in England though a case in an imported cow from Germany was found in Scotland. No cases were seen in Wales.

Control was based on vaccination with new dead vaccines which became available from May 2008, a remarkably rapid development by several drug companies. The use of vaccine was voluntary in England, with the farmer bearing the full cost but the Scottish Parliament decided that vaccination would be compulsory with the farmer only paying half the cost. The vaccination campaign was so successful that a total of only 149 farms had infected animals and no cases have been seen since 2008. EU directives state that prophylactic vaccination cannot be used in a bluetongue-free country but veterinary authorities and farming organizations are seeking permission to allow stock to be vaccinated if farmers wish.

A fall in the number of infected premises has been seen in other EU countries though small numbers of cases due to serotypes other than BT8 are still being seen on the continent of Europe so constant vigil is needed.

Control in countries where the disease is endemic is by vaccination but the large number of serotypes makes this difficult.

Diseases Exotic to the UK and Northern Europe

This book is based primarily on sheep diseases experienced in the UK, much of which also applies to sheep kept throughout northern Europe. Where appropriate, we have tried to indicate which of those diseases are also important in other major

sheep-keeping countries such as Australia, New Zealand and South Africa, and, to a lesser extent, in North and South America. There are a number of serious diseases that do not occur in northern Europe that affect sheep in large areas such as the Mediterranean region, Africa, the Middle East and the Far East, of which it is important that the complete sheep vet has an appreciation. Some of these require vectors not present in temperate countries, but others could potentially occur almost anywhere as a result of uncontrolled or illegal animal or animal product movements. This section briefly brings together some information on the most important of these. More information is available from web sites such as:

- www.oie.int – web site of the OIE (World Organisation for Animal Health);
- www.fao.org/ag/againfo/themes/en/animal_health.html – web site of the FAO (Food and Agriculture Organization of the United Nations); and
- www.ec.europa.eu/food/animal/diseases/adns/adns_en.htm – web site of the European Commission.

One extremely important disease, known for thousands of years, that has in the past affected sheep as well as cattle, was rinderpest (cattle plague, murrain) which occurred as epidemics in Great Britain in the 18th and 19th centuries. The last epidemic in 1865–1867 led to the setting up of the State Veterinary Service. In 2011, while writing this edition, it has been confirmed that rinderpest has been the first animal disease to be globally eradicated.

Peste des petits ruminants

This is caused by a member of the genus *Morbillivirus*, closely related to that causing rinderpest. The disease is found in Africa, the Middle East and eastwards to Afghanistan, Pakistan and India. Clinical signs include fever, catarrhal head discharges, necrotic stomatitis and diarrhoea, often with a high mortality rate. Control is by vaccination.

Rift Valley fever

This disease is caused by a member of the genus *Bunyavirus*, which is transmitted by mosquitoes. It occurs in sub-Saharan Africa and more recently has spread into the Arabian peninsula. Infection in young lambs leads to a high mortality rate which can also occur in adults, though some may show a milder illness. Signs are mostly non-specific but often include haemhorrhagic discharges from body orifices. Control is by vaccination. This disease is a zoonosis.

Nairobi sheep disease

This is also caused by a member of the genus *Bunyavirus*, transmitted by *Rhipicephalus* ticks in East and Central Africa. It causes similar signs to those seen in peste des petits ruminants, with high mortality rates. Control is by the use of acaricides to control the tick vector.

Akabane disease

This is caused by yet another member of the genus *Bunyavirus*, transmitted mainly by *Culicoides* spp. midges. Disease has occurred mainly in Japan and Australia where fetal infection has been the most common presentation, leading to congenital birth defects such as arthrogryposis and hydranencephaly. Diagnosis is by serology and vaccines have been developed but are not widely used.

Sheep pox

This disease occurs in sheep and goats and is caused by a member of the genus *Capripoxvirus*. It occurs in Africa north of the equator, the Middle East and parts of Asia and has spread on several occasions into southern Europe from Turkey. Spread is by direct contact and aerosol. Clinical signs vary according to the breed of sheep and to the strain of virus and the mortality

rate can be high. Affected animals develop fever with swollen lymph nodes and macules and ulcerating papules develop on the skin and mucous membranes. Internally there are lung haemorrhages. Control in endemic areas is by the use of live, attenuated vaccine.

Rabies

This fatal disease, caused by a member of the genus *Lyssavirus*, occurs in areas such as North Africa and the Near East where stray dogs are a problem.

Anthrax

This bacterial disease, caused by the spore-forming *Bacillus anthracis*, is found worldwide. In some countries such as parts of Africa it is endemic. In others, such as the UK, it occurs very occasionally, mostly in cattle, though sheep can be potentially infected. It causes sudden death, the most characteristic signs being dark blood oozing from body orifices and an enlarged spleen. In endemic areas it is controlled by vaccination.

Brucella ovis

This causes epididymitis in rams. It does not occur in the UK but is common in many other countries. See Chapter 2.

Brucella melitensis

This is an important cause of abortion and human disease (Malta fever) in the Mediterranean region, Middle East, India and parts of Africa.

18

Health Schemes

There are two major types of sheep health schemes: (i) national (or regional); and (ii) individual farm schemes. The national schemes aim to produce a pool of sheep flocks of recognized health status with regard to certain specified diseases. Individual farm schemes are provided by practising veterinary surgeons for their clients and are 'tailor-made' for a particular flock.

National and Regional Health Schemes

As described in Chapter 17, most sheep-producing countries have a list of exotic notifiable diseases, designed to prevent their introduction into the country. Other notifiable diseases endemic to the country may be subject to special control measures if they are widespread and of considerable economic importance. In addition, certain other non-notifiable diseases may be of such significance that organized voluntary schemes are designed to improve their control. All these diseases may be included in national or regional health schemes.

In Australia, for example, official national schemes are available to improve the control of the notifiable endemic diseases, paratuberculosis (Johne's disease) and pulmonary adenocarcinoma (Jaagsiekte) and in addition, the State of New South Wales made virulent FR notifiable and subject to eradication schemes some 50 years ago.

In the UK, a national health scheme is available for the control of scrapie, a notifiable endemic disease whereas other endemic diseases, including EAE and CLA, are included in the national health scheme but are not notifiable.

In Scotland, but not in the rest of the UK, statutory measures have been taken to help with the control of sheep scab which requires farmers to notify the veterinary authorities of any known or suspect cases of sheep scab. It also allows inspectors to require farmers to pay for veterinary help in diagnosis and to treat any infected sheep flocks. In addition it makes provision for the control of infection which arises on common grazing or in markets.

Premium Sheep and Goat Health Schemes

The Premium Sheep and Goat Health Schemes are administered by SAC and are the official UK sheep health schemes. They are all voluntary and aim at improving the health and welfare of sheep throughout the UK. The diseases covered are:

- MV.
- CLA.
- EAE.
- Scrapie.

Full details are available on the web site www.sac.ac.uk/consulting/services or contact as follows: Sheep and Goat Health Schemes, PO Box 5557, Inverness, IV2 4YT.

The schemes offered are briefly outlined here.

Maedi visna/caprine arthritis encephalitis (MV/CAE) accreditation

This certifies flocks free of MV (and the similar disease CAE in goats) after passing two qualifying blood tests, providing that stringent biosecurity rules are observed, preventing contact with non-scheme sheep. A proportion of the flock is tested every 2 years to make sure that animals remain negative.

Caseous lymphadenitis (CLA) testing

This aims to identify infected animals at an early stage before significant transmission has occurred. An ELISA test developed at the Moredun Research Institute which detects antibody to *C. pseudotuberculosis* is used on all animals every 3 or 4 months. Positive animals are then removed from the flock by culling. This is obviously an expensive method but has been shown to be effective in producing a clean flock. Another use is to screen animals before purchase to prevent introduction of the disease into a clean flock. Animals screened by a combination of blood testing and clinical examination are available at a limited number of ram sales.

Enzootic abortion of ewes (EAE) accreditation

This aims to establish a register of flocks with a high health status based on freedom from EAE. It is of particular value in providing EAE-free halfbred replacements for lowland farms from hill flocks which are members of the scheme. EAE monitoring is by examination of all abortions at the local SAC/VLA Centre, by serology on all aborting and barren ewes and on a statistical sample of the healthy ewes within 3 months of lambing. Testing is carried out annually and after 1 clean year, the flock is designated as being 'EAE supervised' and, after 2 years, as 'EAE accredited' status and included on a register. Every consignment of breeding stock from a certified flock is given a certificate, the flock may be advertised as EAE accredited and a premium can be expected from purchasers for this status. Vaccination of ewes cannot be used in scheme flocks due to interference with the serological test.

Scrapie monitoring and genotyping

A monitoring scheme is available for breeders wishing to export sheep throughout the EU. This involves annual inspection of the flock and screening of brains from some dead or cull animals. A genotyping scheme is available for selecting breeding sheep of a resistant genetic makeup (see Chapter 14).

Individual Farm Health Schemes

Veterinary surgeons with their special knowledge of the epidemiology, control and the economic importance of disease have made a considerable contribution to the improved welfare and success of dairy, pig and poultry flock health and production throughout the world. There are numerous studies on the adverse economic effects of conditions in dairy cows like lameness, mastitis and infertility which show that the distinctive knowledge and expertise of the veterinary profession can be applied to improve the finances of a farm as well as the health and welfare of the cows. Planned health schemes and regular visits are a feature of the work of dairy and pig specialist veterinary surgeons but although many attempts have been made over a number of years, in the UK, there seems to be reluctance on the part of some sheep farmers to use their veterinary surgeons for this type of scheme. In Australia and New Zealand and a few other countries, sheep veterinary specialists are able to work and make a living solely on sheep but this is rarely true in the UK.

There are many reasons for this lack of take up. Long-term studies on the effect of health programmes developed by and run with veterinary supervision are needed to demonstrate whether the benefits are significantly greater than the costs. Some studies on the costs of specific disease like lameness, fasciolosis and other parasitic diseases are becoming available and show that veterinary advice does give a good benefit-to-cost return.

A great opportunity exists for UK veterinary surgeons to be involved in the formulation of health programmes with farmers (Figs 18.1 and 18.2) as these are required as part of the SFP scheme for the CAP of the EU. The Animal Health Planning System (SAHPS) is a web-based scheme

Fig. 18.1. Health programmes are aimed at maximizing production and minimizing losses from disease in flocks.

Fig. 18.2. Owners of small hobby flocks such as this are often very keen to follow health plans and economics are usually not very important.

developed by the SAC but available to vets throughout the UK (www.sahps.co.uk) and is now being used in nearly 800 flocks so shows signs of optimism on the part of both vets and some enlightened sheep farmers. This web-based system has the advantage of being updated continuously as news becomes available or new technical advances are made. It produces a calendar of farm events, analyses production and financial data and allows comparison with data obtained from other farmers which is helpful for arriving at targets. Data is easy to update each year. It can be accessed anywhere there is internet access, allows communication between farmer and veterinary surgeon and contains links to useful information such as descriptions of specific diseases and drug withdrawal times. Another web-based scheme is produced by Welsh Lamb and Beef Promotions Ltd at www.wlbp.co.uk and is provided for registered members of its lamb, beef and dairy schemes and their veterinary surgeons. Other schemes may be produced by groups or individual vets and others for more localized use.

Successful on-farm schemes often start in a modest way with reaction to a particular disease event. If the control plan has a positive outcome it may then lead on to a more detailed discussion with the farmer and the production of a more comprehensive health plan. However, experience has shown that it is usually best to concentrate on a limited number of diseases or issues at first, rather than going for a hugely complicated plan consisting of many pages of information and instructions that may well lie in the office drawer on the farm until the next visit.

All farms have unique features and so an individual health plan is needed for each. The following is an account of schemes which we and others have used on UK commercial sheep farms.

Objective of farm health scheme

To produce, monitor and maintain a unique health scheme for a particular farm, in the belief that this will both decrease disease and increase production and contribute to the welfare of sheep in the flock.

The scheme

The scheme has three components:

1. A written health programme containing recommendations for the control of expected diseases and to improve production and welfare.
2. Planned visits (three or four annually) to monitor health and production.
3. Reactions to events and advice on new advances during the year, to keep the programme up to date.

Its success hinges on the degree of cooperation between the farmer and the practising veterinary surgeon and other advisors, and requires honest pooling of information.

Health programme

Prior to producing a health programme, information about some or all of the following aspects of the flock will need to be obtained:

- objectives of the flock;
- breeds, numbers, age structure;
- pasture and grazing management (including possible fluke habitats);
- feeding plans (types, quality and quantity);
- housing availability and management;
- lambing management including labour;
- clinical diseases experienced in the past;
- laboratory findings (including PME) if available; and
- production and financial data as available.

As a result of the first planned visit during which a full and open discussion should take place, a programme, in the form of a diary of important management events through the year, together with brief information on disease areas highlighted as being particularly important, is tailor-made to suit the needs of the farm. It is based around the fixed dates of the sheep breeding cycle – the proposed dates for tupping, housing, lambing and weaning. It should address some or all of the following major areas, depending on the commitment

and interest of the farmer involved and on them having identified the areas of most concern in their flock. The document may range from its most basic form of a simple calendar of important events to a sophisticated computerized web-based system.

- Rams – examination and preparation for tupping time; use of teaser rams or other control of breeding.
- Ewes – condition scoring and culling if not done previously.
- Breeding schedule – date rams go in with ewes and date removed (to produce a compact lambing period).
- Vaccination schedule – essential and optional disease cover.
- Lameness – identification of major cause(s) and control.
- Ectoparasites – status and control.
- Roundworms – control including anthelmintic use and awareness of resistance.
- Liver fluke – control if applicable.
- Trace elements – known deficiencies including tests if necessary.
- Lamb mortality – main causes and control.
- Feeding plans.
- Health of farm dogs including worming.

Planned visits

Several visits will be needed in order to assess the application and usefulness of the health programme. In the first year, three or four visits are recommended though in subsequent years, fewer visits and samples will be required.

Reactions to events

It will be necessary to modify the original health programme from time to time during the year, as a result of unexpected events or following information obtained during and in between the planned visits. The programme will need amendment at the end of each year to incorporate new research, drugs and vaccines in the light of the experience of the past year and any alteration in the aims of the farmer.

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Appendix 1

Clinical Examination

Systematic Examination of a Sick Sheep

Carry out the examination in the order shown, examining in more detail where necessary. If signs indicate a neurological problem, a full neurological examination should be carried out. Use of an ultrasound scanner may be helpful particularly for the thorax and abdomen.

From a distance

Parameter	Some possible findings
Grade of illness	Mild, severe, dying
Duration of illness	Acute, chronic
General appearance	Normal, excited, depressed
Mobility	Recumbent, unwilling to move, lame, ataxic, circling
Feeding	Poor appetite, off food, difficulty eating, quidding, ruminating, regularity of jaw movements if eating or cudding
Condition	Thin, fat (beware of fleece masking)
Fleece	Broken, patchy, rubbing
Faeces	Loose, scour, absent
Respirations (count)	Distressed, very fast, deep, coughing, nasal discharge
Head	High, low, turn, tilt, ear(s) drooping, dribbling
Eyes	Blinking, blepharospasm, discharge

On handling standing

Parameter	Some possible findings
Condition score (CS)	1–5 (emaciated, thin, fit, fat)
Temperature	Normal is 39–40°C
Pulse (femoral) and/or heart rate	Strong, weak, rapid (may be fear, recheck later)
Fleece and skin	Wool break, itchy (rub test), ectoparasites, scabs, sores
Age and teeth	
Incisors	Number of temporary/permanent, broken mouth, periodontal disease, apposition
Mandibles and molars (feel through cheeks)	Thickening of mandibles, lumps, discharging sinuses, pain, sharp edges, irregularities, food impaction
Lips and gums	Sores, scabs, smell breath, gum colour (may be pigmented), ulcers, dribbling, capillary refill time
Cheeks	Impacted food, paralysis
Nostrils	Movement, discharge
Eyes	Discharge
Menace and palpebral reflexes	Presence/absence
Eyelids	Entropion
Conjunctiva	Anaemia, toxaemia
Sclera	Vessels injected, jaundice
Cornea and anterior chamber	Keratitis, uveitis
Pupils and lens	Size, symmetry, cataract
Ears	Position, paralysis, discharge, haematoma, mites
Lymph nodes	Enlargement, abscesses
Larynx	Obstruction causing stridor
Chest	
Auscultate heart	Rate and sounds
Auscultate lungs	Abnormal sounds, absence of sounds
Wheelbarrow test	Excess fluid from lungs
Ultrasound chest	Abscesses, tumours
Abdomen (size, consistency, ballot, ultrasound). Right kidney may be palpable behind last rib	Distended, tucked up, gas, fluid, solid
Rumen (palpate, feel contractions, listen)	Irregular or absent contractions, abnormal resonance, gas, abnormal consistency
Abomasum and intestines (listen, ballot)	Abnormal sounds, no sounds, gas, solid
Faeces	Pellets, soft, scour, blood, smell
Vulva and vagina (F)	Discharge, fetal membranes, smell, injury, bruising, haemorrhage
Prepuce, penis, testes (M)	Urethral obstruction/rupture, orchitis, epididymitis
Legs and joints	Stiff, pain, swelling, abrasion, muscle wasting

Tipped up, sitting comfortably with back against legs of examiner

Parameter	Some possible findings
Abdomen (palpate, ballot)	Fluid, solid, fetus
Udder (palpate udder and teat canal)	Swelling, mastitis, fibrosis, abscess, gangrene, milk, colostrum, serum, blind teat, sores, scabs
Feet (locate painful digit by manipulation and pressure if not obvious)	Overgrown/loose horn, interdigital lesions/growths/foreign body, smell, under-running (wall or sole), swelling, cracks, coronary band lesions
Joints (knees, elbows, hips, stifles, hocks)	Pain, stiffness, swelling (fluid/firm)

Samples

Blood	From jugular vein (tube type depends on tests needed)
Urine	By occluding nostrils ('smothering') for a few moments or use catheter. Use test stick, check colour, turbidity, protein, ketones, blood
Faeces	Usually for internal parasites

Appendix 2

Neurological Examination of Sheep

By working through the following sequence, noting down any abnormalities, it should be possible to localize any lesion/s to assist in making a diagnosis.

Indicator	Some possible abnormalities
Behaviour	Wandering, circling, head pressing, fits
Mental state	Hyperexcited, depressed, stuporous, comatose
Head position	Turn, tilt, high/low carriage, star gazing
Head coordination	Jerkiness, intention tremors
Cranial nerves	
Menace test	Visual deficit
Pupil size	Dilated/constricted
Pupil symmetry	Asymmetry
Pupillary light response	Absent
Eye position	Strabismus
Eye movement	Nystagmus
Palbebral reflex	Absent
Facial sensation	Absent
Jaw tone	Slack
Facial symmetry	Facial paralysis
Balance	Loss of balance, rolling
Prehension	Difficulty
Swallowing	Drooling saliva
Stance	Wide based
Gait	Ataxia, hypo/hypermetria
Neck sensation and movement	Stiffness, opisthotonus
Forelimbs	
Skin sensation	Absent (where?)
Muscle tone	Flaccid, spastic
Proprioception	Placing reflex absent
Wheelbarrow test	Difficulty right/left/both
Hemiwalking test	Difficulty right/left/both
Triceps reflex	Reduced/increased/absent

Indicator	Some possible abnormalities
Pedal reflex	Reduced/increased/absent
Deep pain sensation	Absent
Trunk	
Panniculus reflex	Absent (side? level?)
Hind limbs	
Skin sensation	Absent (where?)
Muscle tone	Flaccid, spastic
Proprioception	Placing reflex absent
Hemiwalking test	Difficulty right/left/both
Sway response	Can push over (side?)
Patellar reflex	Reduced/increased/absent
Pedal reflex	Reduced/increased/absent
Deep pain sensation	Absent
Tail/anus	
Anal reflex	Absent, rectum distended
Tail tone	Flaccid
Bladder control	Incontinence

Blindfold (each eye in turn, then both) then reasses balance and gait, repeat wheelbarrow and hemiwalking tests.

Appendix 3

Checklist for Examination of a Sick Lamb

Indicator	Select applicable description
Age	Minutes/hours/days
Birth	Normal/assisted/difficult/caesarean
Litter size	1/2/3/4/5
Lamb size	Normal/small/large (? kg)
Ewe condition score	1/2/3/4/5
Ewe age	? years
Colostrum intake	Sucked unaided/sucked with help/not fed/not known/ tube fed (volume ? ml)
Temperature	Normal/hypothermic/pyrexic (?°C)
Respiration	Normal/slow/rapid/laboured (?/min?)
Heart rate	Normal/slow/rapid/murmur (?/min?)
Demeanour	Normal/depressed/comatose/fits
Posture and gait	Normal/standing but weak/sitting/lateral recumbency/ ataxic/shaking/lame
Birthcoat	Normal/poor/hairy/pigmented
Head	
Jaw	Normal/abnormal
Salivation	Excessive
Eyes	Normal/entropion/discharge/other abnormality
Chest	Fractured ribs
Abdomen	Normal/empty/distended (gas/fluid?)
Navel	Wet/dry shrivelled/swollen/pus
Anal area	Atresia/meconium/scour/blood
Legs	Normal/swollen joints/fracture/contracted tendons
Castration	No/yes (When? How?)
Tailing	No/yes (When? How?)

Appendix 4

Professional Development

Specialist Societies

Most countries with a significant sheep population have an organization with a specialist interest in sheep, small ruminants or perhaps one combined with beef cattle, for which details will be available from country or state veterinary organizations.

Sheep Veterinary Society (SVS)

This is a specialist division of the British Veterinary Association (BVA) and membership is open to all members of the veterinary profession (whether BVA members or not) and others interested in sheep. The Society holds two meetings a year (normally 2 or 3 days) in spring and autumn which include farm visits as well as lectures and discussion in a friendly atmosphere. Membership is a 'must' for all who wish to keep up with developments in sheep veterinary work. Every fourth year (alternating with the international meeting, see below) the Society holds a meeting in another European country (for example meetings have been held in Denmark, Holland, Germany and Spain). Contact the Secretariat at:

Sheep Veterinary Society (www.sheepvetsoc.org.uk)
Moredun Research Institute
Pentlands Science Park
Bush Loan
Penicuik
Midlothian
EH26 OPZ
UK

International Sheep Veterinary Congresses and the International Sheep Veterinary Association

International meetings for veterinarians interested in sheep have been held every fourth year for over 25 years, initiated by the SVS in the UK. Meetings have been held in the UK, New Zealand, Australia, South Africa, Greece and Norway. The International Sheep Veterinary Association has been set up to foster relationships between sheep veterinarians worldwide and to oversee decisions regarding the sites for future international congresses (www.intsheepvetassoc.org).

Specialist Qualifications for Sheep Veterinarians

The Royal College of Veterinary Surgeons (RCVS)

The RCVS offers certificate and diploma level qualifications. The certificate (Cert AVP) is modular and can include farm animal and sheep modules. It is designed for vets in general practice who have more than an average interest in sheep and are willing to study a little more widely. The diploma (DSHP) is the highest standard of examination and requires determination and a real commitment to achieve. Details of syllabuses, regulations and fees are available from:

RCVS (www.rcvs.org.uk)
62–64 Horseferry Road
London
SW1P 2AF
UK

European College of Small Ruminant Health Management

This college is a member of the European Board of Veterinary Specialities and is responsible for approving residents and training establishments and for awarding the DipECSRHM. Applicants with at least 7 years relevant experience, publications and presentations can apply for *de facto* diplomate status until June 2013. After that date the diploma will only be achievable by examination. Although this is a European College, several notable *de facto* diplomates are from countries outside Europe, for example New Zealand, South Africa and Canada. Details are on the web site www.ecsrhm.eu.

Many other countries have schemes for achieving specialist sheep veterinary qualifications and information about these will be available from country or state veterinary authorities or other local veterinary organizations.

Appendix 5

Revision Problems and Questions

These are intended to help test your understanding of the sheep industry and the common problems and diseases associated with it. Please note that these are based on sheep farming in the UK and are not comprehensive. The suggested answers (given in Appendix 6) may not be complete or correct in every situation but are designed to make readers think about what concerns sheep farmers, problem solving and the most likely possibilities to consider first. Problems and suggested solutions may differ in sheep farming systems in other countries.

Question 1

You meet one of your sheep clients at the local agricultural show. He mentions he is in the market for some new Charollais rams and there is a forthcoming sale including rams with EBVs. He asks whether you think it is worth spending extra money on these or whether just to buy the ones he likes the look of as usual.

i. What does EBV mean?
ii. What main traits make up the EBV of terminal sires such as Charollais rams?
iii. What are the potential advantages to your client of using rams with above-average EBVs?

Related questions

a. What are the main characteristics of the Charollais breed and what does it look like?
b. How far in advance of the breeding season should rams be checked and what are the most important checks?
c. How long do sperm take to mature?
d. Are the reproductive capabilities of rams affected by season in the same way as ewes are?

Question 2

You are asked in late July to do a flock health plan for a new client who is keen to improve the management of the flock of 250 Lleyn ewes. One objective is to tighten up the spread of lambing which currently starts in the second week of February and lasts to the end of March. The owner would like to get most of the lambing completed in February.

i. What factors do you have to take into account in planning for the coming breeding season?
ii. What possibilities are available?
iii. What are the advantages and disadvantages of each in this situation?

Related questions

a. What are the particular attributes of the Lleyn breed?
b. What is the length of the oestrous cycle in sheep?
c. What is the length of the oestrous cycle in goats?
d. Which British breed of sheep will breed naturally throughout most of the year?
e. Which type of British breed has the shortest breeding season?

Question 3

The college-educated son has just taken over from his traditional father on one of your practice farms. It is an upland farm with a flock of about 500 Swaledale ewes, mostly purebred, with a few rams sold for breeding but most females kept as replacements and castrated male lambs sold as stores. Draft ewes are sold after four breeding seasons. The son wants to move to breeding Mules which will be kept and crossed with Texels to produce prime slaughter lambs. Some redundant cattle sheds are available for housing and lambing. Father didn't believe in wasting money on vaccines but the son wants to implement a proper health plan including vaccination if justifiable.

i. What aspects of flock management would you take into account in determining your recommendations?
ii. Would you recommend the use of any vaccines and why?
iii. If so, draw up a vaccination schedule for the first year, starting in September or justify your advice not to use vaccines.

Related questions

a. What type of sheep is a Swaledale and what does it look like?
b. What is a draft ewe?
c. How is a Mule produced and what are its main attributes?
d. What type of sheep is a Texel and what does it look like?
e. At what age and weight would you expect Texel cross Mule lambs to be ready for slaughter?

Question 4

You are carrying out a fertility visit to one of your dairy farm clients who also has a lowland flock of about 300 Mule ewes. At the conclusion of the visit, he says that he is worried because he seems to have a lot of thin ewes which are due to lamb in 8 weeks' time.

i. What would you do and what are the most likely causes?
ii. What dangers are associated with this situation?

Related questions

a. What adverse consequences are likely for a flock of ewes with a low condition score (CS) at tupping?
b. What is the most likely age for a lowland ewe which has one pair of broad (permanent) incisors in September?
c. Do sheep develop protective immunity to liver fluke infection?
d. Name two pneumonic conditions which lead to thinness in old sheep.
e. Name three diseases which might be associated with liver flukes.

Question 5

One of your sheep clients phones in a panic as he has had eight abortions last week and six abortions today.

i. What immediate actions would you take?
ii. Are there any dangers for himself and particularly his wife, who is expecting their first child in 5 months?

Related questions

a. What effects other than abortion may you see in a flock with enzootic abortion?
b. Can you detect ewes latently infected with *Chlamydophila abortus* by serological tests?
c. What happens if a susceptible ewe becomes infected with *Toxoplasma* in the first 60 days of gestation?
d. How is *Campylobacter* introduced into a clean flock?
e. Name two causes of abortion in which mummified fetuses are a feature.

Question 6

One of your clients contacts you about his flock of 250 Welsh Halfbred ewes which he housed 5 days ago in four pens. They are due to start lambing in about 6 weeks and have not been scanned. While out at grass they were supplied with molasses buckets and with some hay of which they did not eat much, which the farmer was not worried about as he thought the sheep looked well. He housed them in a bit of a rush as bad weather was forecast. Their diet now consists of big bales of silage and 450 g purchased concentrate nuts per head fed once daily in troughs. Now two or three sheep in each pen have become recumbent and others look dull and are off their food.

i. What are your initial thoughts about the most likely disease problems that might be involved?
ii. Which factors in the management described may be responsible?
iii. How would you proceed to investigate the problem?
iv. What actions would you take to start to tackle the problem?

Related questions

a. How is a Welsh Halfbred produced?
b. What is their main role?
c. When are crucial times to carry out condition scoring?
d. When is the best time to scan pregnant sheep?
e. What is the normal gestation length for sheep?

Question 7

One of your clients in northern England is proposing to change from indoor lambing a flock of 400 crossbred ewes (producing 1.6 lambs reared per ewe) in February to outdoor lambing in March as there have been problems with coccidiosis and joint ill in lambs born indoors.

i. What problems can be expected with outdoor lambing?
ii. What precautions can be taken to minimize these?

Related questions

a. How is lambing percentage defined?
b. How much colostrum do lambs require in the first 24 h of life and does this vary between lambs born indoors and outdoors?
c. For how long is the intestine permeable to large protein molecules (immunoglobulins)?
d. For how long, approximately, do colostral clostridial antibodies protect lambs?
e. What is the most common bacterial cause of joint ill?

Question 8

The owner of a flock of approximately 50 pedigree Suffolk ewes contacts you during lambing time. Five of the mature ewes have lambed with only one teat functioning. As these all have twins it means that extra lambs have to be artificially reared. Your client realizes that nothing can be done with these ewes but wants to try to prevent the same thing happening next year.

i. What is the most likely time that these sheep are becoming infected?
ii. What do you suggest?

iii. Would your advice be any different for a flock of 400 commercial ewes with the same proportion of ewes affected?

Related questions

a. Name the two most common bacteria associated with mastitis in ewes.
b. What are the most likely sources of infection?
c. What are some possible predisposing factors?

Question 9

One of your clients has a limited number of pens for housing lambing ewes so ewes and lambs are normally turned out within 24 h of birth. This year, due to adverse weather conditions towards the end of lambing, ewes and lambs had to be held indoors for 7–10 days, in some cases. Now, about 3 weeks after turnout, some of these lambs, especially the twins, are scouring and losing condition.

i. What are the possible causes and how would you go about making a diagnosis?
ii. For each of the conditions you identify what is the possible sequence of events?
iii. Suggest how you would proceed in each case.

Related questions

a. What are the main anthelmintic groups?
b. Which anthelmintic group is most likely to have worm resistance to it?

Question 10

You are reviewing a flock health programme for one of your clients who has a flock of about 300 Swaledales, most of which are crossed with Bluefaced Leicester rams to produce Mule lambs. The plan was originally drawn up by the client himself. Lambing time is March into early April. The best female Mule lambs are sold as breeding animals in the autumn sales and the males (castrated) are sold for meat throughout the late summer and autumn. After weaning in August, the lambs are put on to silage aftermath in order to get them into good condition for sale. The ewes are vaccinated with clostridial vaccine but the lambs are not vaccinated. Each year there are a few deaths in the lambs in the weeks following weaning.

i. What are the main health issues that need to be considered for these lambs following weaning?
ii. What would you suggest is included in the health plan specifically aimed at this age group?

Related questions

a. What does a Bluefaced Leicester look like?
b. What are the breed's main attributes?
c. What is meant by 'aftermath'?
d. What are the advantages and disadvantages of castration of lambs?

Question 11

One of your clients, who has a flock of about 50 pedigree Suffolk ewes, asks you about tackling an ongoing problem with footrot (FR). The flock sells mainly yearling rams as well as surplus females for breeding. Attempts to get rid of the problem in the past have been based on vaccination and footbathing, but the owner would prefer not to use vaccination as it has resulted in a few lumps developing at the vaccination site.

i. What do you suggest should be the aim in this flock – control or eradication?
ii. What management factors might influence your decision?
iii. What measures would your action plan be based around?

Related questions

a. What bacteria are involved in causing FR?
b. What are the main products used for footbathing and what are the advantages and disadvantages of each?
c. Clinically, how does FR differ from contagious ovine digital dermatitis (CODD)?
d. What bacteria are associated with CODD?

Question 12

One of your clients who does not use you much for sheep work contacts you a week before lambing is due to start. Since housing his sheep 6 weeks previously many have been scratching and losing some wool. He is now concerned that the lambs will become affected after birth.

i. What would you do first?
ii. What are the two most likely diagnoses?
iii. For each diagnosis what are the treatment options?
iv. What are the particularly difficult issues presented by this problem at this time?

Related questions

a. What is a common cause of wool loss without pruritus in late pregnancy/early lactation?
b. What would be the approximate weight of a fleece from an adult Mule ewe?
c. What is the name of the chalky white hairs found in the fleeces of some sheep and why are they a problem when wool is being processed?

Question 13

Your client has been building up a small flock of pedigree Beltex sheep for the past 6 years. He asks you to examine a ewe which was purchased about 2 years previously and which he has treated for pneumonia twice in the last few weeks. She is still breathing rather fast and has lost some weight.

i. What particular techniques can you use to examine sheep with respiratory disease?
ii. What are the main possibilities in this case?
iii. For each main diagnosis, how can you confirm, what are the implications for the rest of the flock and what measures can be taken?

Related questions

a. What are the particular characteristics of Beltex sheep?
b. What upper respiratory disease is sometimes seen in short-necked, highly muscled breeds?
c. What is the cause of atypical pneumonia and in what age group is it most commonly seen?

Question 14

One of your clients has been feeding hay as the source of forage for his flock of crossbred ewes which are housed for 6–8 weeks before lambing in February to early March. Hay is becoming difficult to make and expensive to buy so he proposes to change to making and feeding big-bale silage. He is aware of certain problems related to silage feeding and asks your advice to minimize any risks.

i. What disease is particularly associated with silage feeding?
ii. In which part of the brain are lesions found?
iii. What are the key factors in making and storing silage to minimize the disease risk?
iv. What nutritional problems can be associated with silage feeding?

Related questions

a. How do the organisms you have identified in (i) gain access to the body to cause neurological disease?

b. What other disease pictures can they cause?
c. What are the most common types of space-occupying lesion found in sheep?
d. If a sheep is showing neurological signs including depression, circling and blindness in the right eye, where is the lesion most likely to be sited?

Question 15

A client calls in your surgery asking for eye ointment to treat his lambs (1–3 weeks old) as a number have runny eyes. He has just had an outbreak of New Forest disease in his beef cattle housed in adjacent buildings and suspects the lambs have caught it from the cattle.

i. Do you agree with his diagnosis? What would you want to do?
ii. What possible causes of eye disease are there in this age group of lambs?
iii. If your suspicions are confirmed what treatment and advice do you give?

Related questions

a. What is the cause of New Forest disease in cattle?
b. What is the commonest bacterial cause of eye disease in adult sheep?
c. Do sheep develop a good immunity to this?

Question 16

You have new clients who have just moved in summer from a lowland smallholding to a North Wales upland farm with more land, some of which is rough grazing with heather and bracken. They have taken over the small existing flock of crossbred sheep but have brought their own small flock of rare breed Greyfaced Dartmoor sheep with them. The boundary is well fenced and there are several small fields of improved pasture near to the farm buildings.

i. Apart from the usual disease issues when mixing sheep from different sources, what is a major disease risk for the newly moved sheep in this particular environment?
ii. How do you suggest this is managed?

Related questions

a. Is this environment suitable for Greyfaced Dartmoors?
b. Suggest some other minority breeds which might be suitable for keeping in this environment in this area, rather than the resident crossbreds.
c. What disease of humans might occur in this type of environment?
d. What problems are associated with animals eating bracken?

Question 17

On a training course on notifiable diseases you are asked to name diseases causing lesions on and around the mouths and feet of sheep which might be confused with foot and mouth disease (FMD).

i. Name the most common and give their main diagnostic features.
ii. What signs might make you suspect that you had a possible outbreak of FMD?

Related questions

a. How is the bluetongue virus transmitted?
b. Name two diseases of sheep, neither occurring in the UK, caused by *Brucella* spp.

Appendix 6

Suggested Answers

Please remember these are suggestions! They indicate the most common factors you might consider but may not be the only solution – all cases are different.

Question 1

i. Estimated breeding value.
ii. Weight at scanning (21 weeks), lumbar muscle depth, lumbar fat depth.
iii. To produce faster growing lambs which should be ready while finished lamb prices are still high. To produce better, more valuable lamb carcasses with more muscle in the expensive joints such as leg and loin and less wasteful fat. It is estimated that lambs from high index rams can earn an extra £2–5 each. For a flock of 400 crossbred ewes producing 1.7 lambs per ewe weaned, this could increase income by over £3000.

Answers to related questions

a. A medium-sized, hornless sheep with a pinkish face, with or without light-coloured hair. It has a short, dense fleece. The head and bone are finer than Texels or Suffolks making lambing easier. The main disadvantage is that newborn lambs do not have much wool so may be more prone to hypothermia in cold weather.
b. About 8 weeks before putting with the ewes – check particularly teeth, toes and testicles.
c. Six to seven weeks so if condition is not correct at pre-tupping examination fertility may still not be good by tupping time.
d. Rams are affected by season – testicle size is reduced during spring and early summer but they do not become infertile and are usually willing to mate at any time of year.

Question 2

i. To start lambing in early February, rams need to be in with the ewes about 7 September so there is only 5–6 weeks left. Are ewes and rams in good condition? – look at them, condition score and advise accordingly. Lleyn ewes should be capable of lambing in February. Are rams near ewes – if so, move well away.
ii. Vasectomized rams, progestagen sponges, melatonin implants (but not really enough time to organize this).
iii. Melatonin – barely time this year. Vasectomized rams – if not on farm already will need to acquire and prepare some. Just about time providing suitable animals can

be found (may not be easy at this time of year). Will cost a bit of money if need to buy (probably three for this size of flock), biosecurity risks, cost of surgery. Plenty of time to use sponges but also involves costs and question as to whether pregnant mare serum gonadotrophin (PMSG) needed (probably not in late August). One possibility this year is to put rams in field adjacent to ewes (after keeping them well away till then) for 2 weeks before letting in with ewes, but this needs really good fences. Suggest keeping some of next year's ram lambs to vasectomize for next season.

Answers to related questions

a. Similar production to Mules – prolific, milky. Advantage is being able to breed replacements and keep closed flock.
b. 16–17 days.
c. 21 days.
d. Dorset (horn or polled).
e. Primitives such as Soay (only has two to three cycles usually).

Question 3

i. Increased intensification, increased value of lambs, grow faster than purebreds so may be more susceptible to clostridial disease, housing may make pneumonia more likely.
ii. Recommend use of 8-in-1 clostridial vaccine, possibly plus pasteurella vaccine, depending on quality of housing and farmer's attitude to risk.
iii. Start in September by giving first dose of clostridial or combined clostridial/pasteurella vaccine to all sheep that are remaining on the farm. Include rams. Give second dose 4–6 weeks later. Give third dose to pregnant ewes 4–6 weeks before lambing due. Could risk leaving second dose to prelambing one in view of no history of disease (is this correct? Enquire about history) and providing farmer understands risk. Give first dose to all female lambs to be retained by 10–12 weeks of age and give second dose 4 weeks later. Fast-growing lambs are likely to be ready for slaughter by about 16 weeks so could take chance of not being vaccinated (colostral antibodies should just about cover for clostridial disease) but this is a risk. Slower fattening lambs should be fully vaccinated (two doses) but could use 4-in-1 vaccine designed for this age group.

Answers to related questions

a. A blackfaced mountain sheep with a white nose, horned.
b. A hill ewe that has bred successfully for several years and is sold to a lower, less harsh environment where she could breed successfully for another couple of years.
c. Sire is Bluefaced Leicester, dam is a Swaledale or Dalesbred (North of England Mule) or Welsh Mountain or Specklefaced ewe (Welsh Mule). Mule ewes are prolific, milky and good mothers.
d. White faced, hornless, very meaty sheep. Short neck, wide shoulders and large muscular legs.
e. Singles ready from about 12 weeks, twins from about 16 weeks (depending on milk supply of ewes and quality of grass) but sales can go on throughout summer and autumn. Finished weight can be estimated from: weight of adult Mule ewe (65–70 kg approx.) + weight of adult Texel ram (85–95 kg approx.) divided by 4, that is approximately 36–38 kg, although some may be sold a bit lighter or heavier depending on trade and time of year.

Question 4

i. It is probable that the ewes are still out at grass so arrange to visit the farm when the ewes have been penned. Condition score at least 50 ewes and estimate the extent of the problem. If there are over five ewes which have a condition score (CS) of 2 or below, it is likely to be caused by the food or liver fluke infection though it is possible that molar teeth are involved, especially if the ewes are draft hill ewes and are thus over

4 years old. If the sheep are on grass, examine the pasture for quantity as the quality of grass in January is likely to be low (if quality is low, more quantity should be available). Carry out a clinical examination of two thin ewes and take faeces samples for fluke eggs from five.

ii. There are several possible serious consequences and it is important that the food should be improved immediately by giving 0.25 kg of concentrate feed per sheep daily. The lambs are likely to be small, increasing lamb mortality and decreasing lamb growth, both of which will be exacerbated by the effect that that the low condition has on colostrum and milk quality and quantity. In addition, the ewes which have two or more fetuses are in danger of ketosis (twin-lamb disease).

Answers to related questions

a. The number of lambs born is likely to be low as ovulation rate is influenced by condition and can be improved by ensuring that ewes are in increasing body condition during the tupping period.

b. The ewe is likely to be around 18 months of age as the first pair of permanent teeth erupt at just over 1 year old and most lowland lambs are born in March.

c. No, which means that in bad fluke years, ewes of all ages may die from acute fasciolosis.

d. Maedi visna (MV) and ovine pulmonary adenocarcinoma (OPA).

e. Liver flukes may cause acute and chronic fasciolosis alone or predispose to black disease and bacillary haemoglobinuria.

Question 5

i. In order to ensure the best chance of establishing a rapid diagnosis, visit the farm immediately, after telling the farmer to keep fetal membranes and any aborted fetuses for you to examine. Examine any fetal membranes for signs of placentitis including inter-cotyledonary thickening (enzootic abortion of ewes, EAE) or necrotic foci on the cotyledons (toxoplasmosis). Send membranes and aborted fetuses to diagnostic laboratory. Keep good notes as litigation may follow later. Indicate the steps which he might take to reduce transmission to other ewes by isolating aborted ewes, collecting and disposing of all fetal membranes and spreading clean straw in sheds. Discuss possible treatment of pregnant ewes when a positive diagnosis has been reached. Arrange to visit later to discuss long-term control methods.

ii. Advise on danger of zoonosis, especially of EAE and stress that his wife should not be involved in any way with lambing or rearing lambs and that he should change clothes when he comes home to avoid introducing infectious agents. Treat as an infectious disease and use disposable gloves and polythene bags and use good biosecurity before leaving the farm and calling elsewhere.

Answers to related questions

a. You are likely to see weak lambs and ewes with little milk over the course of an outbreak.

b. No, there is no serological response to the organisms in the latent sites as this only occurs after invasion of the placenta.

c. If the rams have been removed, there may be a high barren rate due to resorption of early fetuses or the ewes may return to the ram if present.

d. This is usually by purchased carrier ewes but birds feeding around troughs in fields may be responsible.

e. Toxoplasmosis and Border disease.

Question 6

i. The main possibilities are hypocalcaemia, pregnancy toxaemia, rumen acidosis and listeriosis.

ii. The main issues are:

- Probably were too thin for this stage of pregnancy – no CS checks done,

hay may have been poor quality so not palatable (check), molasses buckets may have been insufficient.
- Rushing to house them – may have precipitated hypocalcaemia.
- Sudden change of diet so some may not adapt quickly so intake reduced also leading to possibility of pregnancy toxaemia (PT).
- Groups too large so may not all get equal share of food leading to PT.
- Too much concentrate introduced too quickly and stronger ewes may have eaten more than their share leading to acidosis.
- No scanning so probably some ewes carrying multiples are too thin leading to PT.
- Unlikely to have condition scored at housing so thin and fat ewes together.
- Big-bale silage may be poor contributing to reduced food intake and also possibility of listeriosis (but possibly a bit early for this).

iii. Visit to look at sheep, housing conditions and food supplied. Clinically examine affected sheep looking for specific signs such as scouring (rumen acidosis) or facial paralysis (listeriosis). Try test dose of Ca if hypocalcaemia a possibility – they should respond in minutes if this is the only problem. Take bloods for Ca, Mg and betahydroxybutyrate (BOHB) from affected and normal animals. Condition score other animals. Look at silage bales for quality and signs of spoilage.

iv. The action taken will depend on your diagnosis or diagnoses but, apart from treating affected animals, is likely to include splitting into smaller groups based on CS, possibly changing back to hay if good quality is available, splitting concentrate feed into at least two meals, floor feed if possible. Animals should be stressed as little as possible during handling procedures.

Answers to related questions

a. A Border Leicester ram on Welsh Mountain ewes.

b. Prime lamb mothers, similar to Mules but perhaps slightly less prolific.

c. At least 1 month before tupping and 8–10 weeks before lambing is due to prevent too much loss of weight. But handle whenever possible at more frequent intervals.

d. Between 50 and 80 days of gestation. Earlier than 50 days means early pregnancies are missed, later than 80 days makes accurately counting fetal numbers difficult.

e. 145–147 days.

Question 7

i. Outdoor lambing can be very successful but creates more work for the shepherd. It is very weather dependent and bad weather can create a lot of problems for lambs, swapping existing problems for hypothermia.

ii. Are there some sheltered fields near the farm buildings available for lambing? If not, provide extra shelter (straw bales, hay racks, plastic netting, etc. are possibilities). Have some hospital pens available in buildings. Make sure ewes are in good condition so have plenty of milk and colostrum. Check ewes regularly between first light and dark. Number the lambs and ewes so lost lambs can be identified. Keep a look out for at-risk lambs (small, triplets, slow to stand) and make sure they get colostrum (tube feed for speed). Don't castrate or tail too early. If weather is bad, especially wet, could still get joint ill problems.

Answers to related questions

a. Number of lambs reared divided by number of ewes put to ram multiplied by 100. Can also be expressed as for example 1.6 lambs per ewe.

b. 150–250 ml/kg. Outdoor born lambs require most, especially if weather is bad.

c. 24 h maximum, but ability to pass through intestine declines from about 12 h.

d. 12–16 weeks.

e. *Streptococcus dysgalactiae.*

Question 8

i. During lactation and/or immediately after weaning.
ii. Look at the management of weaning – are lambs all removed at the same time? Is the nutrition plane of the ewes reduced to rapidly cut milk production? Make a very careful examination of udders at and just after weaning. Consider the use of intramammary antibiotics as in drying off cows – this is probably cost effective in this type of pedigree flock. Disinfect the end of the teat and use one tube per teat (don't split them between both teats as this can transfer infection). No need to insert nozzle – just appose with teat opening as in heifers.
iii. It is uneconomic to suggest dry ewe therapy for a commercial flock so concentrate on weaning management.

Answers to related questions

a. *Staphylococcus aureus* and *Mannheimia haemolytica.*
b. *Staph. aureus* lives on skin so is present on the teats; *M. haemolytica* is found in the pharynx of lambs and can be transferred to the udder while lambs are sucking.
c. Presence of teat lesions – these often occur as a result of trauma from lambs' sharp incisor teeth. They are particularly common where the milk supply of the ewe is insufficient for lambs (thin ewe with twins, ewe rearing triplets) as lambs compete for milk.

Question 9

i. Lack of milk in ewes; coccidiosis (very likely); nematodirosis. Examine ewes and lambs. If any dead or dying lambs have post-mortem examination (PME) done. Faeces samples may not be very helpful as oocyst numbers can be misleading if non-pathogenic strains are present and may be no *Nematodirus* eggs present as disease can occur before prepatent period.
ii. Ewes may not have had sufficient food and water during extended housing period so milk supply reduced. In the case of coccidiosis the earlier lambs would multiply oocysts massively without becoming diseased; later born lambs which were housed for longer than usual could then pick up large numbers of oocysts from bedding developing clinical disease about 3 weeks later. Nematodirosis is a possibility if improvement in weather has stimulated mass hatching of larvae just as later lambs were turned out.
iii. If it seems to be a milk supply problem, separate ewes with twins if possible, increase concentrates for ewes and supply creep feed. Coccidiosis may be involved in any case – use specific anticoccidial such as diclazuril or toltrazuril drench. If nematodirosis is diagnosed most anthelmintics are effective.

Answers to related questions

a. 1 – benzimidazole (BZ) drugs; 2 – levamisole and morantel (LM) drugs; 3 – macrocyclic lactones (ML) drugs; 4 – amino-acetonitrile derivatives (AADs).
b. Anthelmintic resistance is most common to the BZ group although it has also been detected to LM and ML group products. In rare cases, resistance has been found to all three groups in the same flock. AADs is a new drug group.

Question 10

i. They are not protected by vaccination (passive protection will have completely waned). Losses could be due to pulpy kidney and/or systemic pasteurellosis. Also consider the worm situation.
ii. Need vaccination programme to be started so first and second injections can be completed before weaning. Definitely need clostridial protection. Pasteurella protection is desirable as history suggests this could be implicated, so could use a combined vaccine. Correctly vaccinated ewe

lambs will be more attractive to buyers (some sales specify they must be vaccinated anyway). As silage aftermath may be 'safe' grazing, need to think of worm control strategy. Worming immediately before putting on new grass would have been done in the past, but you need to think of the possibility of increasing the resistant worm population. Action depends on the future use of the grass field – if it is to be used for cattle or arable you can worm and move. If it is to be grazed by sheep you may need to consider developing a susceptible '*refugia*' population.

Answers to related questions

a. A tall, long-bodied sheep with a short, curly, fine, lustrous fleece and a pronounced roman nose and upright ears. Skin colour of head is blueish, hence the name, but often covered with short white (or sometimes brownish) hairs.
b. It is very prolific and passes this trait and milkiness on to its female offspring (Mules).
c. This is the fresh grass growth after a crop of hay or silage has been taken off it. Depending on management earlier in the spring it may be classed as 'safe' grazing.
d. Advantages are that it removes the difficulty of managing male lambs after sexual maturity (this is at about 5 months). Slaughter lambs reach the correct stage of finish at an earlier age than entires. Advantages of not castrating are improved welfare for animals themselves, lambs grow to heavier weight and carcasses are leaner at slaughter. Disadvantages of not castrating are management of older males, may be slow to fatten later in year, possibility of unwanted pregnancies and welfare issues associated with these.

Question 11

i. It should be possible to eradicate footrot (FR) from this flock.
ii. Risks will be buying in rams (could they use AI?) and if they show sheep. Complications will include whether they are prepared to sell chronically infected or non-responsive animals.
iii. Best tackled after weaning ewes. Examination of the feet of all sheep to see the extent of the problem. Separate infected animals so they can be closely checked. Consider giving the whole flock antibiotic treatment (tilmicosin is probably best option but has to be vet administered; gamithromycin has been used very recently under cascade) – this is in case any slightly infected feet have been missed. Move on to pasture not grazed for at least 3 weeks to be on the safe side. Alternatively inject infected animals only (but in a probably valuable flock such as this it would be worth doing all). Re-examine all animals 5–7 days after treatment. Consider what is to happen to those which are still infected – preferably cull but there may be resistance to this if they are seen as valuable. Remember that susceptibility to FR is heritable. Footbathing could also be incorporated as a 'belt-and-braces' approach. Must keep close check and examine any lame sheep at first sign in case this is a resurgence of FR.

Answers to related questions

a. *Dichelobacter nodosus* initiates the disease. *Fusobacterium necrophorum* is a secondary invader.
b. Zinc sulfate (10%) is the product of choice – as it is non-irritant, doesn't harden feet and doesn't degrade. Disadvantage is stand-in time. Formalin is unpleasant, potentially carcinogenic, hardens feet if used too frequently. Advantages are is cheap and walk-through. Copper-containing products are effective but bear in mind risks of having this potentially toxic chemical around.
c. FR lesion starts in the interdigital space and extends across the sole towards the wall. Contagious ovine digital dermatitis (CODD) lesion starts at the coronary band and extends down the wall.
d. Treponemes.

Question 12

i. Visit and examine a representative sample of the ewes. Assess how pruritic the sheep are. Carefully examine the skin for lesions and parasites using a hand lens if possible. Take skin scrapings from the edge of any lesions.
ii. Lice and/or sheep scab.
iii. Only plunge-dipping in an organophosphate (OP) dip will treat both parasites. Lice can be treated with synthetic pyrethroid (SP) pour-on. Scab can be treated with an injectable such as doramectin or moxidectin.
iv. These are heavily pregnant ewes so it is not appropriate to dip now. If the cause is lice, applying an SP pour-on to heavily fleeced sheep is a problem because of wool residues and difficulty of getting sufficient concentration at skin surface. Probably best to leave treatment until after shearing which should be done at first opportunity then treat all, including lambs. If the cause is scab, these can survive off the sheep for 16 days so will be in the environment to infect the newborn lambs. Probably best to treat with a long-acting (LA) moxidectin preparation immediately (be aware of anthelmintic effects as well). Then treat whole flock after shearing either with injectable or by dipping. Do not use SP pour-on if scab is implicated.

Answers to related questions

a. Wool break, caused by weakening or breaking of wool fibres either as a result of poor nutrition or illness.
b. 1.5–2 kg.
c. This is called kemp and causes problems in manufacturing because the fibres break easily and do not take dye.

Question 13

i. Listen with stethoscope, although this does not tell you a great deal. Do a 'wheelbarrow' test to check for excessive fluid in the airways. Use ultrasound to examine the chest (but requires some experience to carry out). Serology, depending on your possible diagnosis.
ii. Chronic pneumonia, possibly with a lung abscess; OPA; maedi.
iii. May be possible to pick up a lung abscess with ultrasound. May respond to long treatment with antibiotic (weeks not days). No real implications for rest of flock but might consider vaccination against pasteurellosis. OPA can be diagnosed if excess fluid is present in the airways as demonstrated by the wheelbarrow test. Ultrasound may be helpful if you are experienced in interpreting image. No serological test is available. PME is the ultimate diagnostic test. Serious implications for rest of flock – may be others infected but not yet showing signs. Expensive and time consuming but can remove lambs at birth and rear artificially away from rest of the flock to create an uninfected nucleus. Maedi can be confirmed by serology. This also has implications for the rest of the flock. Can use serology to identify and remove other infected animals to eventually create an uninfected flock or remove lambs at birth as above.

Answers to related questions

a. Beltex are an extreme form of the Texel, with a small head, short neck and highly muscled body, particularly the hind quarters which look more like those of pigs. They are used for crossing to produce meat lambs with top quality carcass conformation.
b. Laryngeal chondritis, obstruction of the larynx resulting in difficulty breathing and, quite often, death.
c. *Mycoplasma ovipneumoniae*, seen in housed fattening lambs.

Question 14

i. Listeriosis.
ii. In the brainstem.
iii. Preparation of the field – elimination of moles to reduce soil contamination; not

cutting grass too low, again to reduce soil contamination; thoroughly wrap bales to eliminate air; make sure bales are not damaged during transport; make sure wrapping is not damaged during storage.

iv. If silage is too wet or too mature, sheep will be physically unable to eat sufficient to supply metabolizable energy (ME) requirement.

Answers to related questions

a. Thought to gain access through mouth injury or tooth socket (young sheep changing temporary teeth for permanents). Then travel up trigeminal nerve to brainstem where they lead to micro-abscess formation.
b. Abortion, diarrhoea, septicaemia.
c. Abscess, tumour, coenurosis.
d. Left cerebral hemisphere affecting visual cortex. About 90% of neurons cross over at the optic chiasm so blindness occurs in the opposite eye from the lesion.

Question 15

i. Unlikely to be the same as the cattle. You would want to see some affected lambs to check.

ii. Most likely cause is entropion. Hairs rubbing on the eye surface cause lachrymation and keratitis if not treated quickly. Occasionally can get eye infections in young lambs without entropion but always check.

iii. If it is entropion, show client how eyelids should appear and advise checking all lambs after birth. Usually it is fairly easy to correct at this stage just by pulling down the affected lid (always lower ones). Those with runny eyes and keratitis will need treating, probably by injecting antibiotic (procaine penicillin) into the lower lid to stiffen it after correcting the position. Alternative is insertion of Michel clips to take 'tuck' in lower lid. Ask about whether a new ram has been used which might have introduced the problem, since there is a genetic component.

Answers to related questions

a. *Moraxella bovis.*
b. *Mycoplasma conjunctivae*, sometimes *Chlamydophila.*
c. No, immunity is slow to develop so outbreaks can be prolonged and carrier sheep occur.

Question 16

i. It is likely that there will be ticks in the rough grazing areas. The newly introduced sheep will not be immune to tick-borne diseases.

ii. If possible, keep the Greyfaced Dartmoors on the improved pasture through the first autumn and winter until after lambing next spring. If they are introduced to tick-infested land around tupping time, fertility will be badly affected if the sheep get tick-borne fever (TBF). If the ewes are introduced in late pregnancy and get infected they may abort. If louping-ill occurs in the area, the vaccine can be used. Applying an SP pour-on to ewes and lambs on turnout will reduce the tick burden. Keep a close eye on lambs in case they develop tick pyaemia – perhaps consider prophylactic antibiotic injections for lambs on turnout, although this will not protect for very long. After the first year, lambs will become infected with TBF early in life and the flock will gradually become acclimatized.

Answers to related questions

a. Yes, the breed is quite hardy and has a thick fleece so should be able to adapt.
b. There are several interesting Welsh minority breeds that are attractive, interesting to keep and relatively easy to manage. These are the Black Welsh Mountain, the Torddu and Torwen (Badger Faced) and the Balwen (black with a white blaze on the face, white on the lower legs and a white tail tip).
c. Lyme disease.
d. Bracken can be poisonous (especially to cattle), can cause retinal atrophy (bright

blindness) and may be linked to tumour development (e.g. intestinal and bladder).

Question 17

i. Mouth lesions include traumatic ulcers (often on the central lower gum) which have a well-defined raised border and orf which causes vesicles and scabs on the lips. Foot lesions include interdigital dermatitis (skin looks damp and greyish but no vesicles), FR can be confused with foot and mouth disease (FMD) lesions several days old, CODD (haemorrhagic lesion at the coronary band spreading downwards under the wall of the claw, rarely affects all feet) and bluetongue (inflammation around the coronary band of all feet).

ii. Sudden severe lameness of many sheep, some ill particularly pregnant ewes, deaths in young lambs. Erosions on dental pad, blanching and separation of interdigital skin. Ask for help immediately from the Animal Health Office if you are suspicious of the possibility.

Answers to related questions

a. *Culicoides* spp. (midges).

b. *Brucella ovis* causes epididymitis; *Brucella melitensis* causes abortion and mastitis in sheep and is a zoonosis causing Malta fever in man.

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