Manual of Sheep Diseases

Second Edition

J.C. Hindson BVSc, Hon FRCVS
Woodhouse Farm, Hatherleigh, Devon

Agnes C. Winter BVSc, DSHP, PhD, MRCVS, ARAgS
Department of Veterinary Clinical Science and Animal Husbandry,
University of Liverpool

'My flocks feed not,
My ewes breed not,
My rams speed not,
All is amiss.'
Sonnet to Sundry Notes of Music
Shakespeare

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Editorial Offices:
Osney Mead, Oxford OX2 0EL, UK
Tel: +44 (0)1865 206206
Blackwell Science, Inc., 350 Main Street, Malden, MA 02148-5018, USA
Tel: +1 781 388 8250
Iowa State Press, a Blackwell Publishing Company, 2121 State Avenue, Ames, Iowa 50014-8300, USA
Tel: +1 515 292 0140
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Preface

This book, although it has a different title, is an updated and expanded version of our previous book, *Outline of Clinical Diagnosis in Sheep*. It is written at a time when farmers are having to adapt to a very different political and economic environment following noticeable changes that took place during the 1990s. Perhaps the most significant of these changes is the imposition of oppressive but necessary bureaucratic controls. The setting up of the Food Standards Agency and its concerns about spongiform encephalopathies have led to the imposition of identification and marketing rules, and the National Scrapie Plan has been introduced. The 2001 epidemic of foot and mouth disease and European Union regulations have introduced identification and movement recording. These changes are here permanently.

When these regulations are superimposed on the increasing vertical integration of the sheep industry, which gives rise to the movement of millions of sheep annually, the result is a tightly controlled enterprise very different from that which has, perhaps, been one of the attractions of sheep farming in the past.

Looking ahead, the possibility of climate change may influence the pattern of production and disease, although any effects on the sheep industry may be less than on other types of livestock farming. Indeed, the high dependence of sheep farming on grassland may mean that areas of the country where grass production is currently limited by adverse climatic conditions may in future see increased output. This could, however, be offset by the arrival of diseases at present limited to southern Europe, and the spread to our country of insect vectors from southern Europe or even North Africa.

In the last few years, changes to the economics of sheep production have led to a situation where many farm animal practitioners have had neither the opportunity nor the incentive to acquire the knowledge and expertise on this species that their clients have a right to expect. Although a number of texts on sheep diseases are available, ranging from comprehensive coverage of diseases on a global basis, to shorter ones aimed at students and/or farmers, we now feel that there is a need for a text to which the practitioner can refer for information not only on diagnosis, but also on treatment and prevention strategies. This new edition, aimed particularly at the nonspecialist veterinarian, is designed to update and expand the information in the first edition to cover not only diagnosis, but also treatment and prevention of diseases and poor production in sheep. The format is, as in the first edition, based on specific and accurate diagnosis, since without this, treatment and prevention will be a lottery.

We have found it difficult to decide how much detail to include on prevention and, in particular, treatment. Sometimes these are so obvious or simple that no comment is needed, for example treatment of a simple infection with antibiotic. In other cases brief details are included at the most logical point in the text. In the case of major influences on flock productivity, new sections have been written and included as appendices. In particular, new sections have been added on nutrition (which has a significant effect on all aspects of production), health programmes and parasite control. New information is also included on poisons, zoonoses, anaesthesia and some common surgical techniques. Unless there is a particular reason, we have tried to avoid referring to specific commercial products since new products come on the market, old products disappear and drug companies merge, making this particular information soon out of date.

One further difficulty is the reclassification and name changes of some bacteria and parasites that are important in sheep – notably, *Chlamydia psittaci* is now known as *Chlamydophila abortus* and *Pasteurella haemolytica* type A as *Mannheimia haemolytica*. In the case of the latter, though, the disease it causes is still referred to as pasteurellosis. We have tried to use the most recent names, but doubtless further changes will occur.

We hope the expanded text will be of help to all veterinarians, particularly those inexperienced in dealing with sheep. We hope also that it will assist in some small way in safeguarding the health and welfare of the national flock.

Jim Hindson
Agnes Winter

Acknowledgements

Few textbooks are the result of the authors working in isolation. We are very happy to acknowledge the help and support of colleagues in the production of both this new edition and the first edition. In particular Professor Michael Clarkson and other colleagues in the Veterinary Faculty, University of Liverpool, Judith Charnley, Colne and Neil Spedding, Ripon, commented on the content of the first edition, and Charles Stone and Kate Phillips gave advice on the nutrition section of this edition. Without the help of these and other colleagues, friends and families, the production of this book would have been a much more difficult undertaking.

1 Introduction

Successful animal health care, be it for disease, welfare or economic considerations, must rest on the pillars of diagnosis, prognosis, treatment and prevention. Without a high degree of accuracy in diagnosis, prognosis becomes a lottery, treatment may well degenerate into a dependence on 'shotgun' or 'spray' therapy, and prevention may be totally misdirected.

This text has been prepared in an attempt to assist the clinician to answer accurately the questions – 'Why are my sheep too fat? too thin? too dead? – this being the starting point in most investigations. The hope is that, by starting with diagnosis and basing the sections on clinical indications rather than systems, the clinician may more readily arrive at accurate and successful solutions in the treatment and prevention of problems presented by sheep keepers.

No attempt has been made to include every possible condition that could occur at very low incidence – 'the once in a lifetime type' – since this would make the text confusing and the selection of probable causes difficult. Nor have details of underlying pathology been covered; this detail can be found in other texts (see further reading in Appendix 12) and will not normally be of direct relevance during everyday clinical problem solving.

It is impossible to produce a definitive text for any changing science. In particular, laboratory diagnostic techniques are progressing rapidly – note for example the introduction of DNA probes. This text is therefore based on widely accepted and commercially available techniques, commonly used at the time of going to press.

Special difficulties of clinical diagnosis in the sheep

For the sheep when well is often times capricious and when sick does wish to die. Anon

Diagnosis of the cause of a deviation from normal in any species of livestock makes special demands on the combined knowledge and experience of the clinician. The sheep does, however, present its own particular problems. The simple process of isolating the patient from its group frequently induces sufficient stress to mask any behavioural change, which is often the first indicator to the shepherd of an abnormality. Confirmation of the owner's original complaint may not, therefore, be open to the clinician; indeed behavioural change as a primary indicator of disease cannot be relied on. The sheep, in common with some other domesticated species and with the majority of 'wild'

species, exhibits a passive or 'dumb' response when unpleasant external stimuli reach a certain threshold. Beyond this point, no further response will be produced even to painful stimulation and diagnosis must be based on other means. This attitude may also occur during the terminal phases of disease and is the probable origin of some people's conviction that all sheep have a death wish.

In the diagnosis of disease in most animals, variations from normal in respiratory rate, pulse rate and temperature are commonly used early in the diagnostic procedure. None of these is of great value in the sheep, except in the very young lamb where abnormal temperature is significant. Pulse rate may be of little diagnostic value, as catching and handling usually cause a marked increase, except in animals well accustomed to regular handling. Respiration is the primary route of heat loss, so wide variations in respiratory rate occur depending on ambient temperature, length of fleece, pregnancy and other factors. Similarly, body temperature may be elevated above that generally recognised as normal, particularly in hot weather in animals carrying substantial amounts of fleece.

The clinician must be aware of these factors and must carefully evaluate deviations from normal, not placing reliance on body temperature, heart or respiratory rate alone in the absence of other signs. The value of auscultation is also limited by the presence of fleece cover, which masks the details of abnormal respiratory and heart sounds.

Routine for clinical examination

It is important even for the experienced clinician to have a routine procedure for the investigation of any problems. There is a very real temptation to take short cuts based either on first impressions or on previous experience of what may well mistakenly be assumed to be an identical incident.

When carrying out the initial history taking, perhaps the most essential requirement is that we keep an open mind, and it is very important to realise that the complaint the client presents may not in fact be the real one. An animal presented by the sheep keeper as suffering from constipation, for example, is most unlikely to be so affected, and in all probability the problem lies in a different body system.

In the investigation of disease in the sheep the postmortem examination (PME) is likely to play an important role – in major sheep-keeping countries such as Australia and New Zealand, the PM room is considered an essential part of the practice. Thus a routine for this technique in both the adult and the neonate, together with details of sample taking, form important parts of the text.

The following list is a suggested sequence for the examination of an individual or group, and for the building up of the necessary information on which to base a diagnosis and prognosis, treatment and prevention strategy (see also Figure 1.1):

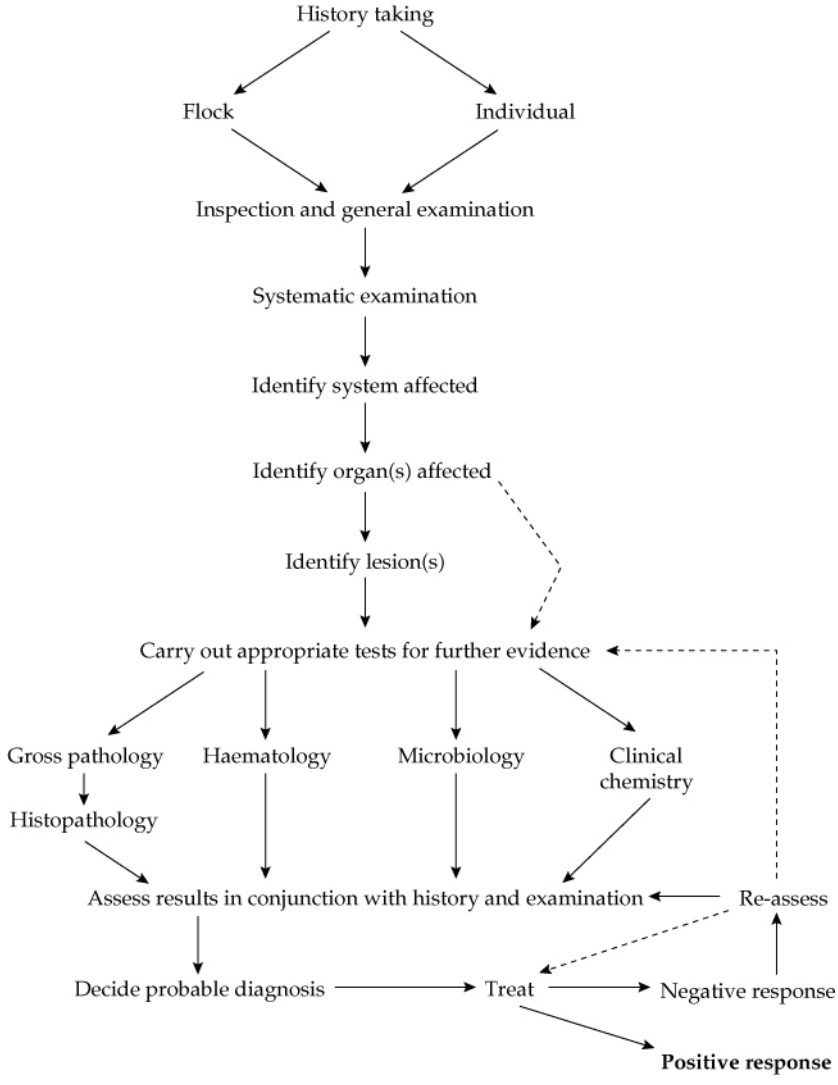


Figure 1.1 Steps in diagnostic procedure.

- The owner's complaint.
- Description of the animal(s) – age and sex.
- Number affected – many animals, a few, or single.
- History of this incident, and in the past.
- Knowledge of the farm, client and area.
- Inspection.
- Systematic examination.
- PME where applicable.
- Specimen taking.
- Laboratory examination and results.

- Diagnosis.
- Prognosis.
- Treatment.
- Prevention.

Examination checklist

The following checklist is a suggested routine for a full clinical examination of a sheep. Obviously not all cases will require such a comprehensive examination, therefore any sections irrelevant to a particular case can be omitted. It is best to carry out as much of the examination as possible with the sheep restrained in a standing position. The animal can then be turned over and examination of the limbs, feet and ventral abdomen completed. Heavily pregnant animals should be treated with care and without turning unless essential.

Owner Date
Animal/s identity
Sex
Age
Inspection
  Appearance
  Behaviour
  Respiration
  Gait
  Appetite
  Faeces
  Fleece
Examination
  Temperature (normal range 39–40°C)
  Pulse (or heart rate)
  Respiratory rate
  Condition score (1–5)
  Fleece and skin
  Head
    Position
    Mouth (lips, incisor teeth, gums, palate, breath)
    Jaws (palpate mandibles and molar tooth arcades)
    Nostrils (movement, discharge, breath)
    Eyes (position, conjunctiva, sclera, cornea, pupil, discharge)
    Ears (position, discharge)
    Lymph nodes (submandibular, parotid)
  Neck
    Prescapular lymph nodes
  Chest
    Auscultate heart and lungs

Palpate over heart
Wheelbarrow test
Abdomen
Size, shape, auscultate, palpate, ballot
Rumen movements (frequency, strength)
Urinary system
Urine
Vulva (urine staining)
Urethra (male)
Prepuce (urine crystals)
External genitalia
Male – scrotum, testicles, prepuce, penis
Female – vulva, mammary glands
Limbs
Lymph nodes (precrural and popliteal)
Joints
Feet
Nervous system
Full neurological examination if necessary

Further aids to diagnosis

Radiography may be helpful in valuable animals, particularly in the case of lameness. Modern diagnostic aids such as endoscopy and ultrasound, now widely available, may be used to investigate, for example, the larynx (endoscope) and chest, kidney, pregnant uterus, bladder, urethra and testes (ultrasound), although experience is needed in interpretation of images. Sampling of blood, urine or other body fluids is suggested where appropriate throughout the book.

Interpretation of clinical signs

In this book, each chapter tackles a clinical problem as it is likely to be presented by the client. In the forefront of the veterinarian's mind when carrying out the initial discussion and examination will be the fact that a number of clinical entities have an obvious age distribution – lamb dysentery limited to the neonate, coccidiosis to the young lamb, and pregnancy toxaemia to the pregnant adult ewe, for example. Similarly, certain conditions are definitely group or flock problems, while others affect individuals or small numbers. For this reason certain sectors have been subdivided to reduce the amount of text the reader must follow.

2 Suboptimal reproductive performance (SORP)

In most flocks, at least 60% of the profitability is a function of litter size and stocking rate, together with lamb price. Since feeding the ewe accounts for some 90% of total feed costs in the case of the production of single lambs, and in excess of 80% in the case of twins, it is entirely understandable and right that the client be concerned by poor flock reproductive performance, and that solutions are urgently demanded from the veterinarian. Suboptimal reproductive performance will, therefore, be one of the most common complaints with which the veterinarian will be presented. In turn, the clinician must accept the responsibility to investigate, but will need great tact in presenting the solutions.

The client will probably assume in the first instance that the cause must be some 'dread' disease, since that absolves him or her from any blame or reflection on the management of the flock. Unfortunately for the clinician, this problem will be presented most often at lambing time when the cause may have existed some 5 months previously and treatment is not possible. Identifying what went wrong will probably be extremely difficult, and no immediate solution can be offered. The final complication is that, with very few exceptions, the cause will not be some 'dread' disease, but will frequently be multifactorial and difficult to identify with total conviction.

> As will become apparent throughout this section, adequate nutrition is absolutely central to many of the reasons for SORP; reference should therefore be made to the separate section on nutrition (Appendix 3) whenever it is suggested that this may be implicated.

Suboptimal reproductive performance can be caused by any of the following:

- Low ovulation rates.
- Low conception rates.
- Early fetal loss/reabsorption.
- Late fetal death.
- Abortion.
- Stillbirth.
- Dystocia and prolapse.
- Poor mothering ability.
- Male infertility.

Table 2.1 highlights common causes of the problem. Abortion, dystocia and prolapse, and male infertility are dealt with in separate chapters (Chapters 3, 4 and 5).

It is essential that we start with definitions of reproductive performance. These must consider:

- Total output.
- Duration of the lambing period.
- Litter size.
- Percentage of ewes in any group failing to breed.
- Percentage of ewes in any group failing to rear lambs which they have carried to term.

Table 2.1 Suboptimal reproductive performance.

Problem	Common causes
Anoestrus	Season Nutrition Lactation Pregnancy
Low ovulation rate	Season Nutrition Breed
Failure to conceive	Male infertility Abnormality of female tract
Low conception rate	Male:female ratio Nutrition Social effects
Embryonic loss	Genetic/chromosomal Nutrition – very high, very low Stress
Early fetal death	Infection – toxoplasmosis, border disease Nutrition – sudden change Stress
Late fetal loss	Infection – all infectious causes of abortion Placental insufficiency (fetal overload)
Intrapartum fetal death	Management Dystocia
Postpartum death	Dystocia Management Maternal factors Environmental factors Infection } Dystocia – mismothering – exposure – starvation – complex

> Irrespective of breed, season or location there can only be one true indicator of reproductive output, the lambing percentage, i.e. the number of lambs reared per 100 ewes put to the ram. This may be a wide and rather ruthless definition and does include losses other than those strictly under consideration under the heading of SORP, but any other definition is only deluding the breeder.

When deciding what constitutes an acceptable flock performance, it is sensible to take into consideration contemporary losses in similar flocks, particularly in adverse seasons or environment, otherwise much time may be wasted looking for other causes which do not exist. There are wide variations in acceptable output, not only between breeds but within any one breed due to genetic factors. There will also be wide variations due to environmental factors. For example, output from the Scottish Blackface in high hill conditions may be as low as 60%, but the same ewes under lowland conditions might produce up to 150%.

The first essential exercise is to establish the true output upon which the client is basing the complaint, and to compare that with some known standard for the breed, season and environment. These standards must be realistic, and not taken from breed propaganda or other biased sources. The Meat and Livestock Commission (MLC) Flockplan records form a firm basis on which to begin comparisons, giving average and top third performances for a wide variety of breeds and crosses in a range of environmental conditions.

Some examples of national figures and targets are:

- Ewe deaths – national range is 4–10%, target is 2%.
- Barren ewes – national range is 4–10%, target is 2%.
- Percentage of ewes mated during first cycle – target is 80%. This figure is required to achieve a maximum duration of lambing of 4 weeks, for optimum output and management efficiency. This may seem to be a peripheral factor, but is regarded rightly by MLC as indicative of good matching of breed, season, environment and nutrition for maximum ovulation and conception rates.

(The higher figures apply to hill flocks which are unlikely to be able to achieve a very low target in adverse environmental conditions.)

The starting point in any flock investigation will be the stage in the breeding cycle at which the complaint originates, and whether the problem involves individual or many ewes, a whole group or all groups. Although male infertility is considered separately, the possibility of ram involvement in any problem must always be kept in mind.

Problems during the mating period

Early in mating period, low mating activity

Check for

- Possible pregnancy (Doppler pregnancy detector can be used from 30 days, scanner from 50 days).
- Ewe and season compatibility (e.g. Dorset Horns will breed any time, Suffolks are early breeders, Mules mid-season, Texels mid–late season, Welsh Mountain late season, Soay limited to three cycles starting in November) – see Figure 2.1.
- Nutrition of ewes (Figure 2.2) – condition score (minimum of 10–15% if large group) – see Table 2.2 for effect of condition score on litter size. Metabolic profile if necessary.
- Weaning to mating interval (minimum 5 weeks).
- Rams – breed, number, reproductive and physical soundness.
- Ram for brisket sores – may cause pain.
- Ewe immaturity (ewe lambs will not begin cycling until several weeks after mature ewes of same breed).
- Ram exhaustion if ewes are synchronised.

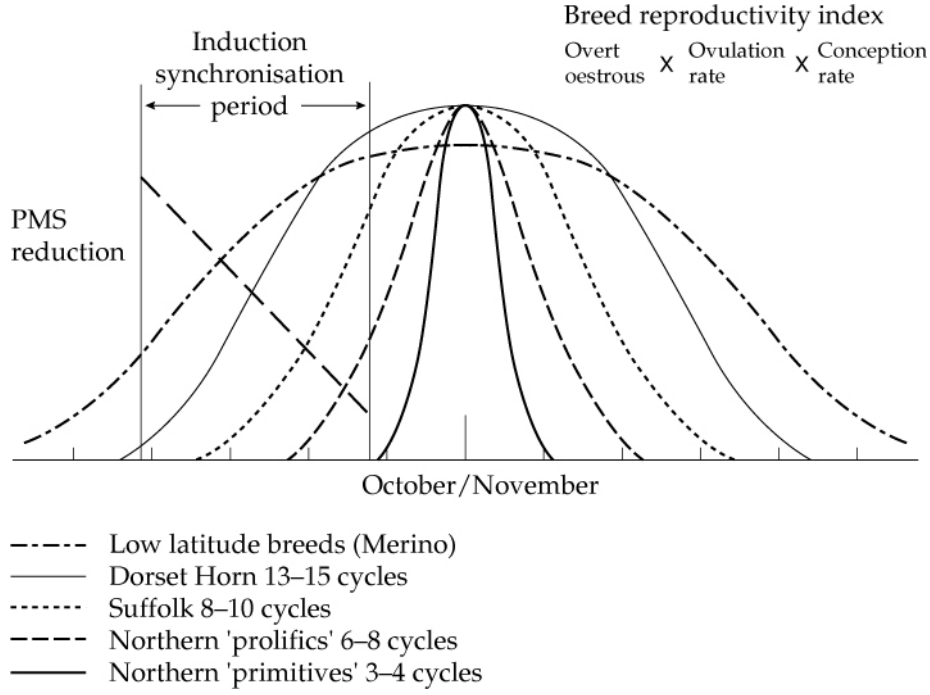


Figure 2.1 Spread of oestrous activity in different breeds.

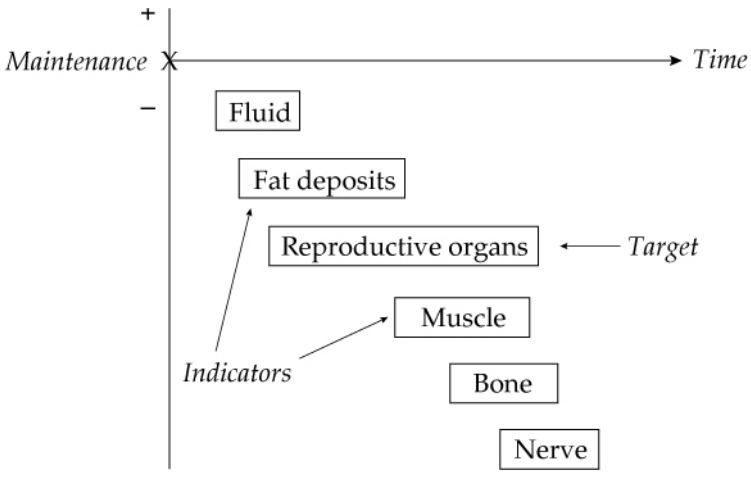


Figure 2.2 Effects of nutritional deprivation on body tissues.

Treatment

If the client is aware early in the breeding season that a problem is developing, there may be opportunity to correct management, based on a correct diagnosis:

- If the mating date does not match the optimum for the breed involved, there will be little financial penalty if a later mating date is used.
- If the ewes are not in adequate body condition, feed inputs can immediately be increased, though it will take 3 weeks to produce a detectable increase in body condition score.
- If the weaning to mating interval was too short, or ewes are immature, delaying mating should improve matters in the same season.

Prevention

This should be straightforward once the reason has been identified.

Full mating activity, high return rate

Check

- Rams' maturity and fertility (see Chapter 5).
- Male:female ratio (1:40 recommended for mature male, 1:25 for ram lamb, 1:10 if synchronised).
- Harness application (if put on incorrectly may interfere physically with mating, or too tightly can cause pain).
- Have sponges been used to synchronise? (see below).

If a problem is detected early there may be an opportunity for correction – removing and replacing immature ram lambs, increasing male:female ratio, removing poorly fitted harness.

High return rate with vaginal discharge

Check for

- Infectious vaginitis or balanoposthitis.
- Hygiene precautions if sponges were used.

Treatment

This may be possible if an infectious agent can be identified and appropriate antibiotic therapy instituted. It is probably wise to withdraw the ram(s) for at least one cycle to allow resolution.

Late in mating period, individuals not mated

Check for

- Pregnancy (lost or worn crayon).
- Nonremoval of sponge if synchronised (may require use of vaginoscope) – presence of characteristic vaginal discharge is a good indicator of retained sponge.
- Congenital abnormality – although it is not usual for freemartins to occur as in cattle, intersex sheep do exist, more commonly from mixed sex high multiple births. The external genitalia may be abnormal, and the teats may be much smaller than in normal females. Vaginal length can be checked by inserting a lubricated vacutainer or blunt probe.

Late in mating period, individuals repeatedly returning to service

Check for

- Excessive fatness in perineal area, or fat tail – may physically interfere with mating. Tail wool may require trimming.
- Trauma from previous lambing – vaginal adhesions (digital examination or vaginoscope).
- Genetic or hormonal abnormality.

Examination of the individual ewe is of limited economic value, although with high priced pedigree or exotic breeds it may be requested and justified in some animals. Use of a vaginoscope may allow examination of the vagina and cervix for anatomical defects or evidence of trauma. With high value animals, use of a laparoscope will allow examination of ovaries and fallopian tubes.

In most cases culling is strongly indicated as the animals will probably have poor fertility in subsequent years, as may any offspring.

Late in mating period, many not mated

Check

- Validity of complaint – harness fitting, crayon loss.
- If already pregnant.
- Age – if ewe lambs, possible immaturity, if shearlings, possible social factors, e.g. breed factors, likes and dislikes of rams.
- True age – may be late lambs therefore late to cycle.
- Body condition score – too fat, too thin.
- Weaning date – lactation effect if very recent.
- Rams – lameness, inexperience, low libido, small ram lambs expected to mate large ewes.

Late in mating period, high rate of returns to service

Check

- Male:female ratio.
- Male – sudden infertility, exhaustion, trauma, infections.
- Possible stress effect, e.g. dipping.

High rate of returns more than 30 days after first mating

Check for

- Stress, e.g. dipping.
- Sudden nutritional stress.
- Toxoplasmosis – rising titres.
- Border disease – rising titres.

If animals are not pregnant, correction is not indicated at this late stage of the breeding season. Very late born lambs are rarely profitable under UK conditions. It may be possible to salvage something by implementing an early breeding system the following breeding season, though nonbreeding ewes tend to become too fat, reducing productivity further. Stressors such as dipping should be avoided for at least the first 30 days after mating until implantation is well established. Likewise, nutrition should not be compromised during the same period. If an infectious agent is implicated, see relevant section in Chapter 3 on abortion.

Manipulation and monitoring of pregnancy

The sheep industry has been well served by applied research in the field of reproduction, with results that have the potential to give significantly increased outputs. These include methods to:

- Extend the breeding season with intravaginal progestagen sponges + PMSG, or by adminstration of melatonin.
- Synchronise ovulation within the normal breeding season with intravaginal sponges (prostaglandins may be used but may have poor results).
- Increase ovulation rates with intravaginal progestagen sponges + PMSG, or by use of breeds with high ovulation rates.
- Monitor litter size by ultrasound scanning.
- Synchronise lambing with injectable steroids (prostaglandins are not effective since maintenance of pregnancy in sheep does not depend on survival of the corpus luteum after the first 50 days).

Unfortunately severe economic restraints within the industry, together with the inherent biological inefficiency of some methods (increased periparturient lamb losses), have led to withdrawal of some products (e.g. Fecundin) and very limited use of others (Boroola and Cambridge genes for increased prolificacy; melatonin).

Some techniques have stood the test of time, particularly synchronisation with progestagen sponges and increased ovulation with PMSG. Other procedures such as laparoscopic AI with frozen semen and ET, although still in use successfully, are limited to high value pedigree units. Poor conception rates with direct vaginal AI (especially with frozen semen) have reduced its use except in high throughput, low cost commercial units using fresh semen. Welfare concerns have made transcervical AI unacceptable.

Since the financial inputs for many of these methods are considerable, requests for explanations of failure of technique or product and avoidance of any repetition are frequently met. If use of a prescription-only medicine (POM) is involved, careful advice should have been given at the time of supply; thus any discussions on unsatisfactory results should rightly be directed at the clinician in the first place.

> When any POM product is supplied, the client must be directed to study the instructions and must be informed that any deviation from the instructions will preclude any complaint to the manufacturer.

Use of ultrasound scanning

In addition to manipulating ovulation rates and synchronising oestrus, monitoring for pregnancy and litter size as a management tool has become more sophisticated, but also should be subject to more rigorous scrutiny on a cost/benefit basis. Scanning can be fully justified under the following conditions:

Hill flocks

- To identify empty ewes which can then be turned back on to the hill, so as not to compete for limited resources.

- To identify any twin-bearing ewes which can then be offered optimum nutritional inputs with maximum cost benefit.

It should however be noted that there may be a problem in correlating a gather with the optimum time for scanning.

Lowland flocks

- To identify expected lamb numbers for each ewe, so that the flock can be split into groups requiring different types of management.
- To reduce lamb losses at parturition by having the necessary labour available.
- To identify single-bearing ewes to avoid overfeeding.
- To identify barren ewes and sell at time of high demand therefore good prices.
- In the case of ewe lambs, to identify and remove nonpregnant animals, and to identify those carrying twins for special support.

Large commercial flocks will be subject to the greatest cost/benefit analysis. As well as the direct cost of the scanning contractor, labour costs will be at least as much again. Flocks with an expected lambing rate in excess of 180% will see no cost benefit in feed saving over feeding all ewes for twins. Below that expected lambing rate, the division of the flock according to feed requirements will justify scanning costs most directly by saving on feed costs to ewes carrying singles, together with reduction in lamb losses at parturition. Scanning must not be applied too rigidly, so that a ewe carrying a single but in poor condition is recognised as requiring a higher feed input than a fat twin-bearing ewe. Common sense therefore needs to be applied with flexible pen grouping to allow for these individual variations.

Special problems associated with manipulation of the breeding season or prolificacy

Many sheep keepers have had disappointing results from first attempts to either synchronise or advance the breeding season, and methods of increasing lambing percentage have also produced disappointing results in some units, with either a poor response or 'litters' of small lambs with poor survival rate resulting.

Forward advancement of breeding season, ewes not mating after sponge removal

Check

- Breed of ewe and realistic advancement achievable. Treatment is only really worthwhile in transition from anoestrum when some ovarian activity is beginning (see Figure 2.1).

- Ram breed, libido and fertility – seasonal breeding patterns occur in some breeds more than others. Dorset, Friesland and Finn are very early, down breeds early, Texel and hill breeds late.

Poor conception rate to synchronised first service in advanced or normal breeding season

Check

- Timing of ram introduction – too early? too late? Recommended time is 48 h after sponge removal. If introduced too early, rams may be exhausted before period of maximum fertility; if too late, will miss fertile period.
- Ewe to ram ratio, 10:1 recommended.
- Fertility of rams.
- Technique and hygiene of sponge application.
- Condition of ewes.
- Weaning to sponging interval.

Within normal breeding season, low mating activity after sponge removal

Check

- Rams – numbers, libido and fertility.
- Timing of ram introduction.
- Lost sponges (alters timing of oestrus).
- Retained sponges (prevents oestrus).

Too high a litter size

Check

- PMSG dosage. Variations in the dose response for PMSG are notorious and are the rule rather than the exception (very high doses may have a negative effect). The dose can vary between 200 and 750 units, but an average dose of 500 units is recommended as a starting point. The nearer to the normal breeding season, the lower the required dose (see Figure 2.1).

Poor response to melatonin (Regulin)

Check

- Breed suitability.
- Seasonal timing.
- Implant to mating interval.

- Body condition and nutrition at implant, mating and throughout pregnancy.
- Sexual activity of males at time of mating – if the drug is used in males they may show a refractory period later in the season.

> It is essential that a full discussion takes place before use of any of these products and that there is a clear understanding of the aims on the part of both client and clinician. Fortunately the initial overenthusiasm for these products has now faded and a much more realistic approach is taken. The most common misunderstandings are that the use of sponges, with or without PMSG, will give normal lambing rates at times of year well outside the normal breeding period, and that melatonin will synchronise oestrus. Neither of these is the case. The risk of litters from the use of sponges and PMSG in prolific breeds is also fully recognised.

Suboptimal lamb numbers

The first step must be to establish whether the complaint is justified by comparing production with similar units, the same breed, season and region. As previously stated, any problems investigated at this time will reflect events which have taken place some months before and the effects of any advice will only emerge some months in the future. It will be difficult, most of all, to establish the true nutritional status of the flock at the time of mating and conception. In addition, the clinician must at all times be aware of the client's wish to escape any 'blame' for the problem. If it is established that the complaint is genuine, then the detailed analysis must establish whether the problem is due to low litter size throughout the flock, normal litter size with a high barren ewe rate, or high perinatal lamb mortality (see Chapter 8).

Low litter size

Individual animals consistently producing low numbers will not be noticed in an otherwise normal flock unless detailed recording is carried out.

Individual pedigree persistent low producers may involve a genetic factor. The tendency to buy well-grown ram lambs for breeding means that, in the absence of records to the contrary, these animals are often singles, which perpetuates low litter size.

Many ewes producing low litter size

Check for

- Age of affected animals – immaturity, old age.

- Familial relationship if known – owner selection for other characteristics may be selecting for low fecundity, e.g. use of single-born ram lambs as above.
- Body score at mating – poor condition at mating gives low ovulation rates, poor conception rates (see Table 2.2).
- Nutrition through pregnancy – low conception rate plus high fetal loss or reabsorption – check placentae for evidence.
- Evidence of toxoplasmosis or border disease (see Chapter 3).
- Stress at critical stages of gestation – may be possible to demonstrate growth retardation lines in long bones as evidence of growth check.

Table 2.2 Effect of body condition score on lambing percentage (from MLC, *Feeding the Ewe*, 1981).

	Lambs born per 100 ewes put to ram						
	Condition score						
Type of ewe	1	1.5	2	2.5	3	3.5	4
Hill ewes							
Scottish Blackface		79			162		
Gritstone (hill)			75	103	119	109	
Welsh Mountain	60	65	105	116	123		
Swaledale		78	133	140	156		
Lowland ewes							
Gritstone (lowland)				132	154	173	
Masham				167	181	215	
Mule			149	166	178	194	192
Greyface			147	163	176	189	184
Welsh Halfbred		126	139	150	164	172	
Scottish Halfbred			148	170	183	217	202

Normal litter size, many barren ewes

Check

- Age of barren ewes – immaturity or old age.
- Breed of barren ewes – late maturing breed, social factors – different breed from ram, breed incompatibility.
- Duration of mating period – too early ram withdrawal for some ewes because of breed or season incompatibility.
- Weaning dates of barren group – lactation effect.
- For evidence of unobserved abortions – border disease and toxoplasmosis.
- For evidence of concurrent disease or stress during pregnancy causing fetal loss.
- If are recent introductions – rejects from other units.

Normal litter size, inadequate birth weights

Check

- Nutrition in early pregnancy (inadequate placentation).
- Body condition score – inadequate feeding in late pregnancy.
- For infectious agent (see Chapter 3).

See also Chapter 8, Perinatal lamb losses, for prevention of inadequate birth weight lambs.

Investigations required before next season irrespective of the cause of this episode

Check

- Ram management, owner knowledge of good ram preparation.
- Owner knowledge of effects of nutrition throughout the breeding cycle.
- Body condition score at weaning and selection of fat or thin groups for appropriate feeding in dry period (see Tables 2.1 and 2.2).
- Nutrition at critical times (condition score, pasture and feed assessment, metabolic profile if necessary), especially 6–8 weeks pretupping when on standard intake.
- For evidence of specific infections – abortion profile of representative group if indicated.
- Parasite control.
- For possible micronutrient deficiency – copper deficiency is not thought to have any effect on conception rates but can cause lambs of low viability; selenium deficiency may have an effect on reproduction although this is not proven.

> On maximum sustainable nutritional inputs, it takes 3–4 weeks to gain $\frac{1}{2}$ condition score.

For the next breeding season provide:

- Record keeping facilities.
- Nutrition guidance.
- Disease control plan.
- Facility for prompt investigation and diagnosis as problems arise.

3 Abortion

Abortion itself clearly does not present any problem of diagnosis. It is in the investigation of the cause or causes of an individual, or more usually, a flock problem that the clinician will be involved. Abortion should be regarded as part of the total picture of suboptimal reproductive performance, even though the sheep keeper will present it as a separate problem.

> It is important to remember that many of the infectious causes of abortion in sheep constitute a zoonosis risk, and that care in handling abortion material is necessary. In particular, enzootic (chlamydial) abortion and *Toxoplasma* abortion are serious risks to pregnant women, and advice to this effect should always be given to the client.

Definitions used in this text

- Abortion is the expulsion before full term of a fetus which is incapable of independent life.
- Premature birth is the expulsion before full term of a fetus which is capable of independent life.
- Stillbirth is the expulsion of a dead full term fetus.
- Gestation length – although the usually accepted length is 147 days, it is important to recognise that there are individual and breed variations, with a range of 140–150 days to be expected.

Although expulsion of a dead fetus before term is the usual evidence of abortion, during any abortion episode some fetuses may be carried to term yet be infected, and some may be carried to term and be fully viable. It is also important to recognise that abortion incidents may be multifactorial, not only in terms of infectious agents but also involving nutritional and management factors. These may be additive; for example, inadequate maternal nutrition may be the critical factor which changes a mild infective placentitis into terminal interference with the life support mechanisms of the fetus.

The clinician must be aware of the possibility of mixed infections arising, therefore establishment of a causal factor early in the investigation of an abortion outbreak is not the end of the matter. In addition, the advice required where mixed infections are diagnosed, for example whether to isolate or mix ewes, whether to use as foster mothers, etc., may be contradictory for different abortion agents.

What comprises an abortion problem?

Premature births occasionally occur in all flocks, but where more than 2% of lambs are born outside the normal gestational variation, it must be presumed that some factor is present which is jeopardising fetal survival. The owner's complaint must be treated as a matter of urgency and a full investigation initiated if an early diagnosis is to be achieved and losses reduced to a minimum.

While abortion storms in sheep never reach the catastrophic proportions of the initial brucellosis storms in cattle, losses of up to 50% have been recorded after the first exposure of a totally 'naïve' flock to a new infectious agent; 20–30% will be a more usual proportion even during the initial storm, while 5–10% may well occur during chronic flock infection in seasons following that in which the infection was first introduced. The degree of loss will not act as a guide to the type of infection, but only to the presence of a chronic or new infection. Sudden change to this incidence will often indicate the addition of a further cause to an existing pattern. The stage during gestation at which abortion takes place will not be a guide to the specific cause, but only in some cases a possible guide to the timing of infection or nutritional stress. Finally, gross pathology will often be unhelpful except in the case of EAE and toxoplasmosis.

Causes of ovine abortion

Infectious causes (Table 3.1)

Some of these infectious agents are primarily placentotrophic, e.g. *Chlamyophila*, *Toxoplasma*, *S. abortus ovis*, *S. montevideo*. Others cause more generalised disease with abortion the result of septicaemia, e.g. *S. typhimurium*, *S. dublin*, tick-borne fever.

Noninfectious causes

The following factors have been implicated, and should be considered if infectious causes are eliminated:

- Inadequate nutrition.
- Pregnancy toxaemia.
- Stress.
- Poor handling.
- Vaccination.
- Transport.
- Dog worry.
- Concurrent disease.
- Pasteurellosis.
- Chronic fluke.

Table 3.1 Infectious causes of abortion.

Protozoon	*Toxoplasma gondii*	Toxoplasmosis
	Neospora caninum	Neosporosis
Bacteria	*Chlamydophila abortus*	
	(Chlamydia psittaci)	Enzootic abortion
	Campylobacter fetus fetus	Campylobacteriosis
	Campylobacter jejuni	(vibriosis)
	Salmonella abortus ovis	
	Salmonella arizonae	
	Salmonella dublin	Salmonellosis
	Salmonella montevideo	
	Salmonella typhimurium	
	Leptospira interrogans	Leptospirosis
	Listeria monocytogenes	Listeriosis
	Miscellaneous bacteria e.g. *Yersinia, Histophilus ovis*	
Rickettsia	*Ehrlichia (Cytoecetes) phagocytophila*	Tick-borne fever
	Coxiella burnetii	Q fever
Virus	Pestivirus	Border disease
Fungae	*Aspergillus fumigatus*	Aspergillosis
	Claviceps purpurea	Ergotism
	Various mycotoxins	

Of the many causes of ovine abortion, by far the most important are *Chlamydophila (Chlamydia)* and *Toxoplasma* which account for over 40% and over 35% respectively of all diagnosed incidents. A positive diagnosis of an infectious cause only results with material from about 50% of incidents submitted to veterinary diagnostic laboratories, but it does not follow that the remaining 50% are not due to infections. Inadequate or unsuitable material may have been submitted, or tests to identify more unusual causal agents may not be carried out, or fragile organisms may not survive the journey to the laboratory.

Investigation of an outbreak (Figure 3.1)

It is essential that the owner retains all material and that it is as fresh as possible, so that the clinician is in a position to choose appropriate samples to maximise the chance of an early accurate diagnosis. Samples should be submitted from a minimum of 10% of affected ewes, preferably more, and if abortions continue over a prolonged lambing time, a check should be made at intervals to make sure that different agents are not operating.

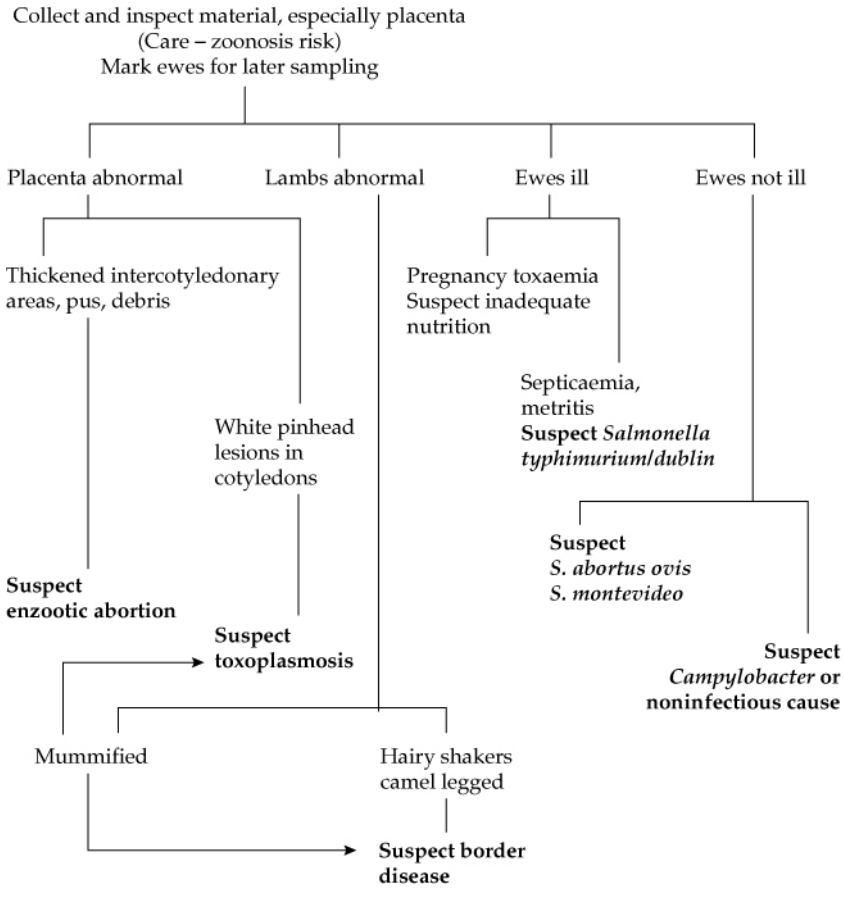


Figure 3.1 Investigation of multiple abortions.

Material required varies for different agents but the following material should be obtained in every investigation:

- Dead fetuses.
- If whole fetus not available, fetal stomach contents and pleural fluid in vacutainers, plus piece of fetal liver.
- Placenta including cotyledons.
- Vaginal swab if placenta not available.

Aborting ewes should be marked for later identification and sampling if necessary and should be isolated from both pregnant and nonpregnant sheep until the cause is established.

Since dystocia and metritis will be common sequelae of any individual case of abortion, septicaemia may occur as a secondary feature.

Guide to diagnosis (Tables 3.2 and 3.3)

The following points may help to give an initial diagnosis, but a final diagnosis must rest upon laboratory confirmation and no possibility should be excluded on macroscopic appearance only. As mentioned previously, beware of mixed or changing causes.

Discolouration and necrosis of the cotyledons, oedema or rough thickening of adjacent intercotyledonary tissue, dirty pink or pus-like exudate on placenta surface

Suspect

- Enzootic abortion caused by *Chlamydophila abortus* (formerly *Chlamydia psittaci*) (see colour Figure 1) – normally infection is acquired in one lambing season, with abortion following in the next season. Latently infected ewes which have not yet aborted cannot be identified by serology. If the lambing season is prolonged, with ewes more than about 6 weeks from term exposed, infection and abortion can occur in the same season.

Diagnosis (Table 3.4)
This depends on demonstration of the characteristic organisms in a smear from the abortion material, or on showing a high titre on serological testing of aborting ewes.

Where a live but infected lamb is born, pathological change may be limited to a small area of placenta.

Q fever (*C. burnetii*) is similar in smears, and can be distinguished only on serology, but is much rarer than EAE.

Treatment
It is essential that an early diagnosis is achieved, since in-contact pregnant ewes can be treated with an immediate injection of long-acting oxytetracycline (10 mg/kg body weight). This should be repeated after 3 weeks for ewes yet to lamb. The results are often dramatic with abortions stopping within a few days. Treatment does not eliminate infection, but merely reduces the number of

Table 3.2 Abortion investigation – diagnostic aids.

Type	Gross pathology		Concurrent maternal disease	Laboratory material required	Tests
	Placenta	Fetus			
Chlamydophila	+++	–	–	Placenta Fetal skin swab Maternal blood	Smear CFT
Toxoplasma	+++	+/– mummification	–	Placenta Fetal pleural fluid	IFAT LAT ELISA PCR Histology
Campylobacter	–	+/– liver lesions	–	Placenta Fetal stomach contents Maternal blood	Smear Culture CFT
Salmonella	–	–	– *S. abortus ovis*, – *S. montevideo* + other spp.	Placenta Fetal stomach contents Maternal blood	Smear Culture SAT ELISA
Border disease	–	++ characteristic lambs or mummification	–	Fetal blood Fetal brain, spleen, lymph nodes	Virus isolation IFAT Histology
Listeria	–	+ sawdust liver	++ neurological disease within flock	Placenta Fetus	Culture IFAT Histology
Leptospira	–	–	+ milk drop	Maternal blood	FAT
Mycotic	++	+/–	–	Placenta Fetal stomach contents	Smear Histology
Q fever	–	–	+/–	Placenta Maternal blood	Smear CFT
Tick-borne fever	–	–	+/–	Maternal blood	Smear IFAT Known tick area

CFT = complement fixation test IFAT = indirect fluorescent antibody test LAT = latex agglutination test
ELISA = enzyme-linked immunosorbent assay PCR = polymerase chain reaction SAT = serum agglutination test FAT = fluorescent antibody test

Table 3.3 Timing of infection in relation to time of abortion.

Organism	Minimum time infection to abortion	Likely source
Chlamydophila	6 weeks Usually infection one lambing season, abortion next season, but infection and abortion possible in same season if lambing prolonged	Infected ewe Placenta Fetal fluids Discharges up to 2 weeks after lambing
Toxoplasma	6 weeks Infection in early gestation gives reabsorption and barrenness	Feed or forage contaminated with cat faeces *No* sheep to sheep transmission
Campylobacter	7 days	Carrier sheep Wild life
S. abortus ovis	2 weeks	Carrier sheep
Other salmonellae	1–2 weeks	Food contamination Water contamination Wildlife
Listeria	5–10 days	Poor quality silage High pH Soil contamination
Border disease	Infection <85 days gestation gives abortion and diseased lambs Infection >85 days gestation gives normal antibody +ve lambs	Carrier sheep (virus +ve antibody −ve)

Table 3.4 Diagnosis of enzootic abortion.

Material	Test	Comments
Placental smear	Modified ZN stain	Red organisms, singly or in groups, on blue background
Vaginal discharge	Modified ZN stain	Take within 24 h of abortion
Fetal fleece smear	Modified ZN stain	Organisms sparse, best site is behind ears
Serology	CFT	>1:32 indicates recent infection, use paired samples or as flock screen EAE vaccine gives positive result

organisms and thus on-going damage to the placenta, making carriage of fetuses to nearer full term more likely.

Prevention

Prevention is more complex, since latently infected ewes may not be protected by vaccination. Currently, live attenuated and inactivated vaccines are available. The relevant data sheet should be carefully read, since instructions as to when to use vary. All types can be given to nonpregnant ewes. The inactivated vaccine may be used in pregnant ewes from 4 weeks after removal of the ram.

It may be possible to adminster *Toxoplasma* vaccine at the same time (not to pregnant animals), but check data sheet carefully before doing this.

> Remember that ewes already infected may not be protected by vaccination, and lambs or ewes introduced into the flock may also be latently infected, therefore it cannot be assumed that a flock is free of infection, so protection must be maintained.

EAE-free animals

The SAC Premium Health Scheme includes monitoring of flocks for EAE. Breeding stock are warrented free of infection, but the animals themselves are not immune, so the status of the resident flock should be established to ensure that the added value of such replacements is justified.

Rams

Rams are not thought to be implicated in the spread of EAE, but to be absolutely safe EAE-free flocks should only use rams from flocks of the same status.

White pinhead-size necrotic foci present in the cotyledons ('frosted strawberry' appearance), intercotyledonary area normal

Suspect

- Toxoplasmosis caused by *Toxoplasma gondii.*

> Toxoplasmosis early in pregnancy may cause resorption of fetuses leading to a large number of barren ewes at lambing time.

Diagnosis (Table 3.5)

Although characteristic lesions may be present in the placenta (see colour Figure 2), diagnosis should be confirmed by submitting abortion material to a laboratory. Positive serology does *not* confirm abortion was caused by *Toxoplasma*, only that the animal has been exposed to infection at some stage in the past.

Table 3.5 Diagnosis of *Toxoplasma* abortion.

Material	Test	Comments
Fetal pleural fluid	Latex agglutination	1:16 is positive. If LAT is negative, use another test
	Indirect fluorescent antibody test (IFAT)	
	Modified agglutination test	
	Dye test	
	Immunoperoxidase methods	Useful with decomposed material
Placenta	FAT	
Fetal brain	Histology	
Fetal liver	PCR	PCR not yet used commercially
Placental cotyledon		

Treatment

The position is not as hopeful as for EAE. Any treatment will only be effective if given before serious damage to cotyledons has occurred. If the affected flock has a long lambing period and diagnosis has been made early, addition of the coccidiostat decoquinate to the feed may reduce losses. Inclusion rate depends on stage of pregnancy – check data sheet. Potentiated sulphonamides have also been shown to be effective, but only if cotyledon damage has not reached a critical state.

Prevention

Prevention is now possible with the arrival of an efficient attenuated live vaccine. Given at least 3 weeks before mating, a good degree of protection is achieved.

Reduction of contamination of the environment with *Toxoplasma* oocysts is a valid aim, but difficult to achieve. Young cats are the source of contamination as they excrete massive numbers of oocysts in their faeces when first infected by eating wild life such as mice. Prevention of contamination of feedstuffs, while retaining good rodent control, is difficult.

> If sheep introduced at the beginning of a breeding season later abort with toxoplasmosis, it is often assumed that they were infected when purchased. This is not the case – they will have picked up infection during pregnancy. Animals infected when empty become immune. A positive titre indicates only that the animal has been exposed to infection at some time in the past and is not diagnostic of *Toxoplasma* abortion.

> Vaccination of a whole flock for the first time can be expensive, particularly if both EAE and toxoplasmosis are involved. A suitable compromise may be to vaccinate only young and replacement ewes, assuming that older ewes will have been exposed and gained immunity. This may not be entirely the case, so some abortions should be expected, but after a few years the majority of the flock will consist of vaccinated animals.

Mummified fetuses produced, with or without normal lambs

Check for

- Toxoplasmosis, as above.
- Border disease, see below.

Thickened leather-like placenta

Check for

- Mycotic abortion (fungal hyphae may be demonstrated in placenta or fetal stomach contents).
- Spoilage of homegrown or stored cereals with *Aspergillus fumigatus* or *Fuserium gramminarium.*

Mycotic abortion can be prevented by careful storage of cereals and by avoiding feeding spoiled forage or concentrates contaminated with obvious mould growth. There is no treatment once the problem has been identified.

Characteristic 'hairy shaker' lambs born

Suspect

- Border disease caused by a pestivirus.

Diagnosis (Table 3.6)
There are no macroscopic distinguishing features in the placenta. The presence of characteristic lambs is a strong indicator of disease, with virus isolation from aborted material providing confirmation.

Treatment
Treatment is not possible as infection will have occurred some time before any problem is apparent.

Control
If a new outbreak occurs, the probable source of infection should be identified and culled – this will be a persistently infected (virus positive, antibody

Table 3.6 Diagnosis of abortion due to border disease.

Material	Test	Comments
Placenta or fetal tissues (fresh)	Immunostaining	
Placenta or fetal tissues (in virus transport medium)	Virus isolation	
Brain tissue	Histology	
Clotted blood from dam and lamb	Serology	Antibody presence is not diagnostic
Heparinised blood from suspected lambs (precolostral)	Virus isolation	Colostral antibody may interfere with isolation

negative) animal. The whole lamb crop from that season should be sold, as there are likely to be persistently infected animals present. Future purchases should be made from unaffected flocks if possible. If infection is widespread or endemic, control should be aimed at exposure of the whole flock before mating, with the build up of natural immunity. Lambs which are obviously affected but survive should not under any circumstances be retained. Epidemiology resembles that of BVDV in cattle.

Prevention

If the flock is free of BD, maintenance of a closed flock gives the best protection. If animals have to be bought in, testing for virus positive, antibody negative status may be worthwhile in a valuable flock. A vaccine is available for BVDV in cattle, but not for BD in sheep.

> Cattle can be a source of infection for sheep, having a much higher percentage of persistently infected animals. Sheep are much less likely to pose a risk to cattle.

Significant incidence of septicaemia, enteritis and mortality in ewes

Suspect

- Salmonellosis. Verotoxigenic *E. coli* has also been reported to cause abortion with septicaemia and scouring, but not usually significant ewe deaths.

Diagnosis (Table 3.7)

Systemic illness in aborting ewes should always alert the clinician to the likelihood of a zoonotic infection such as salmonellosis, although some serotypes cause little or no illness. Diagnosis must therefore depend on laboratory testing.

Table 3.7 Diagnosis of abortion due to *Salmonella* spp.

Material	Test	Comment
Placenta Fetal stomach contents	Smear and culture	Gram-negative bacteria
Faeces, intestinal contents, lymph nodes, internal organs of affected ewe	Smear and culture	In chronic infections use posterior mesenteric lymph nodes
Serum from aborted ewes	Serum agglutination test	May be useful on flock basis within 4 weeks of abortion

S. typhimurium and *S. dublin* usually cause systemic illness in ewes. *S. abortus ovis* and *S. montevideo* often cause abortion only.

Some *Salmonella* spp. are zoonotic so extreme care must be taken in dealing with an infected flock.

Treatment

Infected animals must be isolated. Systemic antibiotics can be given to affected and contact animals and may limit spread and death rate, but mortality can be high (apart from *S. abortus ovis*). After an outbreak, bedding and other debris should be burnt, composted or spread only on arable fields.

Prevention

As this is often an opportunistic infection, outbreaks are sporadic, so little can be done other than minimising contamination of feed or water by wildlife or sewage effluent.

Silage fed flock, possibly with neurological signs in ewes or septicaemia in lambs

Check for

- Listeriosis – abortion may occur in absence of neurological disease within the flock.

Diagnosis

Placental and fetal material may be taken for smears, culture and histological examination.

Treatment
Outbreaks rarely involve a large number of sheep, so it may not be cost-effective to use antibiotic treatment.

Prevention
Infection is by ingestion from contaminated soil or grass, most commonly via poorly made silage. As the organism does not multiply below pH 5, achieving good fermentation during silage making is essential, as well as minimising soil contamination. Sheep should not be fed from the open face of a silage clamp, or from stale block cut silage, or with big bales that cannot be eaten within 2–3 days maximum. Uneaten silage should be cleared before putting out new supplies.

> Where a flock experiences a persistent problem with *Listeria* infection, it is best to avoid grazing sheep in grass fields to be conserved for silage as the organisms are present in normal sheep faeces.

No obvious placental lesions, ewes generally not ill

Check for

- Salmonellosis, particularly *S. abortus ovis* (limited to south-west England) and *S. montevideo* (see above).
- *Campylobacter* infection – this is most often introduced into a flock either by carrier sheep or contamination of feed by birds such as crows and seagulls.

Diagnosis
Take samples of placenta, fetal stomach contents and fetal liver as above. Sometimes characteristic circular lesions, 'smoke rings', are present in the liver.

Treatment and control
Treatment is difficult since infection spreads quickly. Antibiotics are of dubious value. Often the outbreak fizzles out after a time. Aborted ewes should be removed from other pregnant animals but may be introduced to those that have already lambed to try to spread infection and therefore immunity through the flock.

Prevention
Vaccines are not available in the UK, though they are used in some countries. Feed troughs should be kept clean to avoid contamination by birds.

Other infectious causes and toxins

There may be sporadic or isolated cases where the following causes may be implicated:

- *Leptospira hardjo* – uncommon, may be treatable with dihydrostreptomycin.
- *Arcanobacterium (Corynebacterium) pyogenes.*
- *E. coli* – this is occasionally isolated but may be a faecal contaminant in many cases. A verotoxigenic strain has been associated with abortion and illness.
- *Bacillus licheniformis.*
- Tick-borne fever – introduction to tick area of unacclimatised pregnant ewes.
- Ergotism – infected cereals.

Advice should be sought from the laboratory regarding samples required for investigation.

No obvious signs of an infectious agent involved

Check for

- Recent handling, e.g. vaccination, drenching.
- Excessive use of dogs, dog worry.
- Condition of ewes – inadequate feeding, concurrent disease, pregnancy toxaemia.

Metabolic profiles of at least six ewes may be helpful – BHB, plasma protein, urea, glucose. Micronutrient deficiencies are not thought to be implicated in abortion problems, with the possible exception of vitamin A deficiency.

> Fetal overload rarely seems to lead to abortion. In fact, it is surprising how often a pregnancy is maintained in cases of fetal overload or severe undernutrition, putting the life of the ewe at risk, when it would seem that rejection of the fetuses would be the obvious biological solution!

Occasionally, a request for advice is received following an abortion incident when no aborted material is available. It may be possible to obtain paired sera taken at an interval of 2–3 weeks from affected ewes, provided the first samples are taken soon after abortion, or the clinician may choose to sample those ewes which have aborted and compare with a group which have carried to term with normal viable fetuses. Any results obtained will need careful interpretation in conjunction with the history of the outbreak.

There is no substitute for early and thorough investigation at the time of the incident, with as much material as possible collected for examination, and this should be stressed to clients in advance of the lambing season via newsletters, practice meetings, etc.

4 Dystocia and vagino-cervical prolapse

Perinatal ewe and lamb losses are major components of SORP, and a higher than acceptable incidence of both prolapses and dystocia makes an important contribution in these areas. Diagnosis is self-evident but increasing incidence may bring requests to identify causes. Dystocia increases the perinatal lamb death rate by perhaps 60%. Prolapse of the cervix or vagina places fetal survival at risk by stimulating premature straining, causing changes in intrauterine pressures which jeopardise the integrity of placental circulation. The introduction of infection through a traumatised cervix or an oedematous infected vaginal wall in neglected cases (regrettably common) all too often leads to bacteria gaining entry to the uterine contents and loss of the lambs if not also of the ewe.

The clinician must again approach the request for help with a confident knowledge of acceptable proportions of either of these problems for the breed, the management system and the season. It could be said that zero is the only acceptable incidence but the clinician lives in the real world!

Regarding prolapses, surveys of crossbred ewe flocks such as the Mule and Greyface have revealed a worrying incidence, although other flocks have a low incidence. One possibility is that crossbred ewes of that type have a particularly large pelvic inlet, predisposing to the condition. A flock of Scottish Blackface ewes on normal high hill nutrition, with little social stress and carrying single fetuses of low birth weight, will usually experience a very low incidence of both prolapse and dystocia. On the other hand, young Texel ewes on a high plane of nutrition carrying single male lambs have been quoted as requiring manual help for up to 70% of lambings. Superimposed on any 'natural' incidence will be not only the degree of expertise of the shepherd, but also an intangible 'panic' factor which may be the result of 'sympathy', concern for welfare or just plain impatience. The clinician must learn to recognise this when called in to resolve a problem.

As well as dealing with day-to-day prolapses and dystocia cases, it is likely that the clinician will be called in response to an unacceptable change for the worse in incidence of either problem in an established flock. Sometimes the call for help may be from a new entrant who has no base line for comparison or experience in dealing with such cases.

Problems involving prolapse and/or dystocia will tend to be concentrated at the early stages of the lambing season. Abortions and stillbirths will also occur around the same time, so the client may be unduly concerned that a major disaster is about to happen. The clinician must walk a fine line between reassurance and analytical support.

Increase in incidence of dystocia (Figure 4.1)

Normal fetal size, normal litter size

The first check must be for any change in management, primarily intensification, and particularly either a change to a housed system or alterations to that system.

> Ewes, if free to do so, will isolate themselves at the onset of the second stage of labour, and then assume dominance in their chosen site for a limited period. If limitations of space prevent this normal behaviour pattern, the parturition process may not proceed smoothly.

Check

- Overall space per ewe – 1 to 1.5 m^2 recommended depending on ewe size.
- Trough space per ewe – 400–500 mm per ewe for concentrates, 200–225 mm for hay or silage feeding ad libitum.

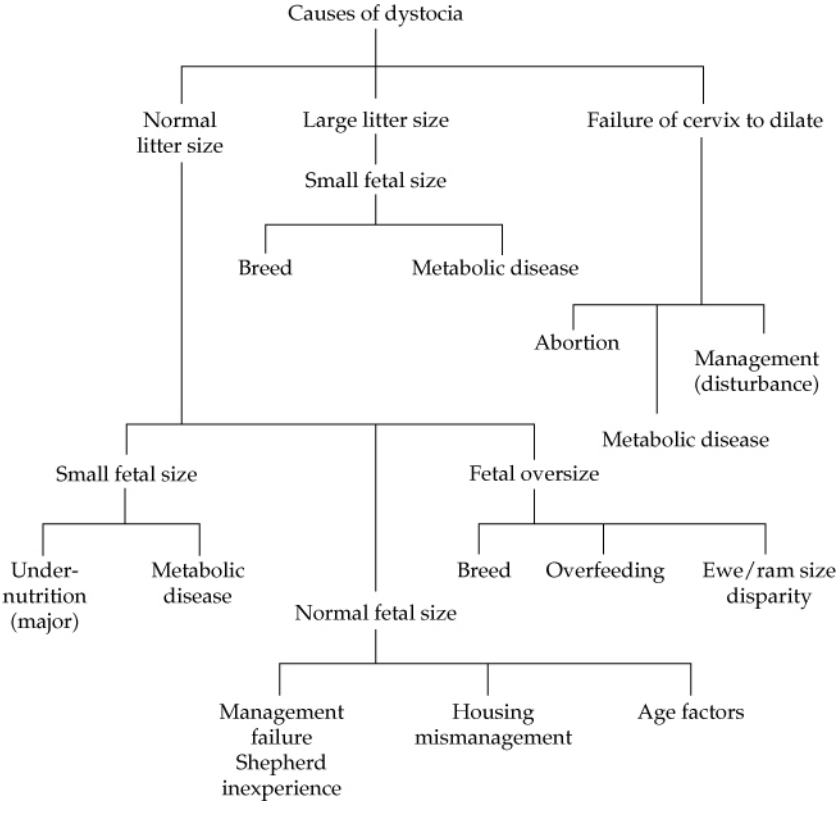


Figure 4.1 Causes of dystocia.

- Age distribution per pen – may be advisable to segregate old and young ewes.
- Age of high incidence group – social factors may interfere with normal progression of labour.
- Group size – social stress. Maximum 50 per pen for lowland breeds, 80 for hill breeds is recommended.
- Client-induced stress – any stress during the early stages of parturition can interfere with the coordinated uterine and abdominal contractions. In housed units, even opening doors and switching on lights may be sufficient in animals unused to the conditions.
- Availability of 'creep' areas – research has shown that parturient ewes will lamb in partially shielded areas if these are provided.
- For rams left in groups of housed ewes – aggressive behaviour may lead to physical damage and presence of rams may cause stress effect.
- Experience of shepherd – premature interference can precipitate dystocia where it would not have developed if the ewe had been allowed more time.

> One of the most difficult areas for the inexperienced to judge is between allowing sufficient time for progress to take place (which can be quite long in primiparous ewes), and not risking the life of the lambs by waiting too long. Recommend attending a practical lambing course offered by your practice!

Treatment and prevention

This may simply be a case of advising minor changes in management, or extra training of farm staff. Detailed information on the management of dystocia is outside the scope of this text.

Fetal oversize, normal litter size

> Relatively minor feto-maternal disproportion, particularly in young or inexperienced ewes, may show as high dystocia rate due to inadequate pelvic inlet area.

Check

- Feeding in relation to lambing percentage.
- If scanning has been carried out. This is of most benefit for flocks lambing at 120–180%, and allows cost-effective feeding of different groups to be carried out.
- Compatibility of sire breed – choosing the wrong breed can lead to a high degree of feto-maternal disproportion.
- Lambing spread – late-lambing ewes will have had prolonged overfeeding, and are usually less prolific.

Treatment
If the problem is presented early in the lambing period, a reduction in feed intake may give sufficient check in fetal growth to reduce the problem. However, this must be applied with regard to fetal demand and the risk of inducing pregnancy toxaemia.

Prevention
A review of late pregnancy nutrition and ultrasound scanning to pick out single-bearing ewes will usually deal with this problem.

Small fetal size, normal litter size

If this problem occurs on a flock basis, the combination must mean that there has been prolonged macro- or micronutrient deprivation, dystocia being either a function of inanition or terminal cachexia. This may be manifest as myometrial inertia associated with calcium deficiency.

Check

- Body condition score of flock.
- Incidence of pregnancy toxaemia.
- Metabolic profile of representative group of animals (BHB, glucose, Ca, P, Mg).

> Selenium deficiency in cattle has been shown to be associated with prolonged labour and increased death rate of calves, but there has been no similar picture confirmed in sheep.

Treatment
It is not possible to influence fetal size at this stage. Improve feeding and treat with calcium if laboratory tests suggest a need.

Prevention
Review late-pregnancy feeding.

Small fetal size, high litter size

A high dystocia rate under these conditions is a function of prolificacy. It must be remembered that prolificacy of itself will increase the incidence of dystocia, partly through the increased risk of malpresentation but also because of a breakdown in the normal dominant/quiescent uterine horn sequence which operates in twinning.

In prolific breeds reproductive output has outstripped the digestive ability to maintain such output. This is partly due to abdominal space limitations, but also to sheer nutrient demand (a ewe with triplets is equal to the demand of a sow with 30+ piglets).

Check for

- Pregnancy toxaemia induced as a result of fetal demands.
- Calcium deficiency.
- Abdominal muscle weakness.

Treatment

Treatment is not possible by the time the problem is apparent.

Prevention

The drive for litters has now been devalued as being both biologically and economically inefficient. A change of ewe breed or dilution of prolificacy by crossing are the best way of dealing with this problem.

Failure of cervix to dilate (ringwomb)

The incidence of failure to dilate varies widely both between flocks and from season to season. Because dystocia inevitably follows, with reduced lamb survival, and because there is always a question mark against future breeding from affected ewes, calls for an explanation will be common.

Check for

- Prematurity – may be the first evidence of an abortion storm.
- Management changes – housing, overcrowding, interference with parturition process.
- Malpresentation – no fetus present to complete dilatation.
- Calcium deficiency.
- Exogenous oestrogens, e.g. home stored corn with fungal contamination (*Fusarium* spp.).

Treatment

Many methods have been tried, but there is no easy answer. Methods include manual dilation, but this often produces unacceptable degrees of trauma; prostaglandins may help but delay may lead to the loss of the lamb; oestradiol injection has been used, but similarly leads to delay and probable loss of the lamb. If it is economically worthwhile a caesarean operation can be performed, but survival rate of the lambs is often disappointing. The other option is humane slaughter.

Prevention

There is no easy answer, as the cause is often obscure and incidence varies from year to year.

Prolapses

There are several separate types of prolapse, diagnosis of all being self-evident. Some of the conditions may be linked but the aetiology is not fully understood. The problem is included in SORP, for although fetal loss is by no means inevitable (except in the case of rupture with prolapse of the intestines where death of ewe and fetuses is extremely rapid), there is a much higher than normal fetal death rate following the occurrence of prelambing prolapses. Even if placental integrity is not damaged to a critical point, and live lambs are delivered, the chance of the ewe repeating the condition in future years is so high that it is normal to cull those affected, so adding to culling rate and reducing the true flock output. Since there is no problem with the diagnosis of this condition, the clinician will be concerned with aetiology and treatment, and advice will be required which will try to reduce losses in future years.

Types of prolapse

- Cervix and/or vagina before lambing.
- Intestines through a rupture of the vagina before lambing.
- Uterus after lambing.
- Cervix after lambing.

In addition, if straining is severe, prolapse of the rectum may also occur.

Prolapse of the cervix and/or vagina

This has long been recognised as being multifactorial, all surveys having picked out several predisposing factors, not all of which will be present in any one incident. It remains a mystery why some flocks should show an incidence of perhaps 15% while others with similar breeds and management may have few cases.

The condition appears to be a result of an increase in intrapelvic pressures late in pregnancy. In the early stages a small part of the cervix protrudes from the vulva when the ewe is lying, but disappears when she stands. The prolapse eventually gets to a size that causes the pressure receptors in the wall of the pelvic canal to be activated, initiating extremes of expulsive efforts on the part of the ewe. It is also likely that kinking of the urethra and therefore inability to empty the bladder plays a role in the onset of severe straining. The cervix and/or vagina are then forced through the vulva. Occasionally the prolapse consists of a ballooned portion of the dorsal vaginal wall. At first the prolapse contains fetal fluids, but in more advanced cases parts of the fetus and/or bladder may be involved. Factors implicated in the occurrence of these prolapses include:

- Breed with large pelvic inlet, e.g. Mule.
- Prolificacy (but this does not always follow – the most prolific breed, the Cambridge, is not particularly prone to prolapse).
- Multiparous ewes – prolapse is less common, but by no means unknown in primiparous ewes. If prolapse does occur in a primiparous ewe, this can be difficult to control because of extreme straining efforts.
- Bulky feed – sometimes associated with change to silage feeding, or roots.
- Feeding only once daily – splitting both roughage and concentrates into at least two feeds is advisable.
- Fat deposits in abdomen or pelvis – overfatness, particularly in young ewes.
- Steep slope in field allowing abdominal contents to 'drift' into pelvis during sleep or cudding (heavily pregnant ewes always stand or lie facing uphill which eases some of the pressure on the thorax).
- Reduced perineal support – short docking may reduce muscular support of posterior vagina and vulva (short docking is illegal).
- Chronic cough (rather unlikely on flock basis).
- Reduced calcium concentrations have been implicated, but whether as a primary cause or secondary to stress is not clear.

The clinician is left, therefore, with a detailed history taking to establish which factor or combination of factors is the cause of any one incident. Usually many can be eliminated, but is what is left the answer? At the end of the investigation we may be left unable to identify the exact cause, but breed and strain do have a big effect, and there is probably a hereditary component. There seems to be little doubt that the incidence is reaching worrying proportions in some flocks.

Treatment

Most sheep farmers will treat simple cases themselves. However, any cases showing significant straining or where the prolapse is swollen or damaged, should receive veterinary treatment.

> The most essential procedure that can be offered by the clinician is provision of analgesia of the perineal region by means of sacrococcygeal epidural anaesthesia. A combination of local anaesthetic and xylazine will facilitate replacement of the prolapse and give 24 hours or more relief from straining. A suitable dose for a 65 kg ewe is 1.75 ml 2% lignocaine and 0.25 ml 2% xylazine. These are mixed in the same syringe and administered into the sacrococcygeal or first intercoccygeal space. See Appendix 8.

After providing analgesia and cleaning the prolapse, it should be replaced. There are a number of methods of retaining the prolapse, the choice being a result of experience and personal preference. Those commonly used are:

- Commercial or home-made harness – this supports the perineal area and, if correctly fitted, should allow the ewe to reach term.
- Intravaginal device – favoured by some, but not always successful and can lead to swelling of the perineal area and vaginitis.
- Suturing the vulva by the Buhner method or with a deep mattress suture.

Whichever method is used, the ewe must be observed carefully for the onset of parturition so that the harness or suture can be removed. Parenteral antibiotics and an NSAID such as flunixin should be given if the prolapse is infected or swollen.

Well managed, an affected ewe can reach term and a favourable outcome with healthy lambs. Badly managed, the outcome can be total loss of ewe and lambs.

As the condition is likely to recur in the next pregnancy, it is best to cull affected ewes, although valuable pedigree ewes can be successfully managed if avoiding action is taken by fitting a harness towards the end of pregnancy or at the first sign of prolapse threatening.

Prevention

If this is more than a sporadic event in a flock, management factors need to be examined, for example body condition, feeding (roughage type, quality), concentrates (amount, how often fed), amount of exercise, etc.

Prolapse of the intestines through vaginal rupture

This sporadic event probably occurs if a part of the intestine, or occasionally the bladder, is trapped within the pelvis, stimulating expulsive efforts which may be so extreme as to cause a rupture in the dorsal vaginal wall forcing abdominal contents through it. Severe shock and death follow rapidly.

Treatment

If found alive the sheep should be euthanased on welfare grounds. It may be worth carrying out an emergency caesarean section to deliver the lambs. This can be done immediately after shooting the ewe, or by anaesthetising the posterior abdomen with an epidural injection into the lumbo-sacral space, then destroying the ewe after delivering the lambs. However, as these are usually premature, survival rate is poor. As this is a sporadic event, no advice can be given on prevention.

Prolapse of uterus

This is also a sporadic event which may follow a difficult lambing, or may follow an unassisted lambing. Lack of uterine muscle tone due to subclinical hypocalcaemia may be involved in some cases.

Treatment

Posterior epidural anaesthesia should be provided using the sacrococcygeal space (see above). The prolapsed uterus should be cleaned and examined for

injuries and the placenta detached if possible, although if this is still firmly attached it should be trimmed and left in situ. The prolapse is then returned into its normal position in the abdomen. Calcium, long-acting antibiotic and an NSAID should be administered. Provided the organ was not damaged, the prognosis is usually good.

Prolapse of cervix after lambing

This usually occurs following a difficult lambing when the cervix has been damaged and become infected. It often occurs several days or even weeks after lambing. As the prolapse is not as obvious in a pre-lambing case, it may be swollen and contaminated with faeces by the time it is seen.

Treatment

Posterior epidural anaesthesia (as above using lignocaine and xylazine for long duration) should be provided. The prolapsed tissues should be thoroughly cleaned before replacing. It is strongly advised to insert a retaining suture in the vulva, as these cases can recur. These animals should be culled as soon as practically possible.

Prevention

Careful manipulation when dealing with dystocia cases to avoid damage to the cervix, particularly with incomplete dilatation of the cervix or oversized lambs, coupled with adequate antibiotic cover, should reduce the incidence.

5 Male infertility

Routine examination of the ram before the breeding season begins is now a recognised procedure in the sheep industry, but even so, only a minority of animals actually are examined. Requests are commonly made for the examination of individual rams of suspect fertility, or for assistance in flocks experiencing apparent difficulties getting ewes in lamb, and it is with these aspects that this text is concerned. Surveys have suggested that some 10% of rams are likely to be infertile, with a further 30% being of suspect fertility.

The UK is currently free of the important genital infection *Brucella ovis*, but sporadic cases of epididymitis and orchitis do occur caused by a variety of organisms including *Histophilus ovis* and *Actinobacillus seminis*. Scrotal mange caused by *Chorioptes bovis* has been reported in the UK and is common in some other countries.

> Sperm maturation takes 6–8 weeks to complete, therefore any illness or stress can be followed by infertility becoming apparent several weeks later.

> There is a widespread belief among farmers that treatment of rams with systemic antibiotics will make them temporarily infertile. This has never been proven, but one must ask the question 'Why was the ram being treated in the first place? Could the infertility be a result of that condition rather than the antibiotic administration?'

Incidents of infertility involving whole groups of rams

Check for

- Infections of the external genitalia – these occasionally occur in both ewes and rams around mating time, but do not generally cause infertility. Mycoplasmas, ureaplasms and *Streptococcus zooepidemicus* have been isolated from some of these cases of balanoposthitis and vulvovaginitis but their significance is not clear.
- Epididymitis and orchitis, possibly caused by *H. ovis*. This problem has been reported in groups of young rams.

- Scrotal mange, if thickening and scabbing of scrotal skin is evident. Take skin scraping. These mites can also be found on the lower limbs.
- *Dermatophilus* infection of scrotal skin – impression smear of underside of scab should help diagnosis.
- Orf virus – take scabs for laboratory confirmation.
- Short tail docking in ewes – fly nuisance may be significant.
- Hygiene and technique if sponges have been used.
- Immaturity of whole group of rams – occasionally an inexperienced client may use only ram lambs.

> In the immediate pre-mating period, the male group often indulges in mounting behaviour. This frequently results in insertion of the penis into the rectum of other rams, leading to contamination with faeces. It is not known whether this can act as a source of infection.

Treatment

Treatment will depend on the diagnosis. Infections of the external genitalia may be amenable to appropriate antibiotic treatment. *Dermatophilus* infection can be treated by application of antiseptic washes to soften and remove the scabs. Chorioptic mange can be difficult to clear. Injection of moxidectin or doramectin may be successful.

Individual male infertility

Detection and identification of an infertile male will be no problem in small flocks or pedigree flocks where a single male is used. In larger flocks, without routine examination of all rams, detection of an infertile male will often be difficult as the rams are usually run in groups with large numbers of ewes. The rams may well be of differing ages and will always be of varying dominance, aggression and libido. In addition, it is normal for the ewe to 'seek out' the ram, and the female group will have an established order of dominance.

A combination of these two patterns can give rise to quite complex social interactions, which may not only mask the true fertility rate but also hinder the search for an infertile animal. As an example, if a particular male is extremely aggressive he may spend the whole period preventing access by other rams in the group to females in oestrus, but may not himself mate, either through exhaustion or by constantly defending the ewe group from other males. Conversely, an older dominant ewe may 'appropriate' the male for such a long period that other ewes remain unmated. It is always wise to use either one ram or three, not two which may spend the whole time establishing dominance rather than seeking out ewes in oestrus.

Although it has been established that trained rams are capable of producing fertile semen throughout the year, in the field there is undoubtedly a strong photoperiodic effect in the male as well as the female. Finn, Friesland and

Milksheep rams will be active very early in the breeding season, and the Suffolk soon after. In contrast, the Texel is notoriously late (fully fertile rams may show little interest until late September).

Just as the early onset of puberty in the female and prolificacy appear to have a strong correlation, so in the male early puberty is linked to the seasonal duration of active sexual drive. In addition, there is a correlation between large testicle size in relation to body weight and the early onset of puberty.

Full fertility is dependent on a scrotal temperature several degrees below normal body temperature. Any condition that prevents this differential will interfere with fertility (equivalent to the 'tight jeans' syndrome in man!). Some breeds have heavy wool cover over the scrotum, sometimes made worse by copious lanolin secretion, which raises scrotal temperature. Scrotal mange and infection with *Dermatophilus* will have the same effect.

Where a flock fertility problem exists, the investigation of the male role in this will be in two stages:

- Establishing the validity and nature of the problem.
- Diagnosis of the cause by observation of behaviour and clinical examination.

> Requests may be made to examine a ram at the end of the lambing season, for example, if rams of different breeds were used and few offspring of one were born. This is not a suitable time to make an accurate assessment, and examination should be delayed until nearer the normal mating season.

Apparent sexual inactivity

Check

- Ewes are not already in lamb. Before any detailed investigations of the ram, establish if the complaint is valid. The most common cause of nonmating is pregnancy. This should be detectable from about 30 days with a Doppler pregnancy detector, and 50 days with a scanner. If vasectomised rams have been used in the flock, check these for time of surgery in relation to exposure to ewes (a minimum of 2 weeks should be allowed), or misidentification of teaser rams.
- Body condition – if ram is too thin, suspect nutrition or concurrent disease (see Chapter 12 on adult weight loss).
- For overfeeding if ram is too fat – newly purchased high value rams may show temporary infertility due to sudden change in environment and nutrition.

If ram is in good body condition (3.5–4.5 body score):

Check

- For breed or season incompatibility.
- For immaturity – look at testicle size.

- If young inexperienced ram is with inexperienced females – may be better with older ewes.
- If young ram was reared in all-male group – may be shy when put with group of females.

If the above factors are eliminated, the clinician must establish whether the ram really has poor libido, or if he is incapable of demonstrating interest because of other disease factors, in particular interference with mobility.

Check

- For footrot, foot abscess, arthritis, lumbar pain.
- For other concurrent disease – parasites, pulmonary or cardiac disease.

Low libido

This may be the transitional phase to or from apparent sexual inactivity (see above).

Check

- As above.
- Possible onset of exhaustion – if libido was normal earlier in breeding season, may be overwork. Ram:ewe ratio recommended is 1:25 for ram lamb and 1:40 for mature ram, except in synchronised flocks (see Chapter 2).
- For small testicle size.

> Serving capacity can be correlated with testicle size. Tests for serving capacity have been established in New Zealand and Australia, but are not in common use here because of possible welfare problems.

> An apparent lack of sexual activity in the daytime does not preclude full activity during darkness. Raddle or harnesses should be used to obtain firm evidence.

Normal libido, little mounting activity

Check for

- Pregnancy.
- Pain focus in those structures most involved in the mounting process – hind limb lameness, poor hind limb conformation, lumbar or pelvic pain.
- Painful lesion of penis or prepuce.

Normal libido, normal mounting, failure to mate

> The clinician must be able to differentiate between mounting and full mounting with intromission and thrusting during ejaculation.

Check for

- Inexperience – age and previous use.
- Ability to extrude penis fully.
- Abnormality of the penis, either anatomical or traumatic, e.g. deviation.

Normal libido, normal mating behaviour, high return rate

Before investigating the ram, the clinician should be satisfied that there are no adverse factors affecting the fertility of the ewes (see Chapter 2).

In particular check

- Ewe:ram ratio.
- That synchronisation problems are not involved.
- That expectations are not too great at either end of normal breeding season.
- For recent illness in ram – pyrexia can induce temporary infertility.

A full clinical examination of the ram should then be carried out. This will be essentially the same examination as that carried out in the pre-breeding check, but will always require the collection and examination of a semen sample unless gross lesions accounting for the fertility problem are detected.

Routine for the clinical examination of a ram

There is a considerable amount of information about testicle size of normal rams. Some useful guidelines for adult rams are:

- Scrotal circumference, 36 cm.
- Testicle length, 10–11 cm.
- Testicle diameter, 6–9 cm.
- Epididymis diameter, 3.5–4 cm.

These figures are those which could be expected in mature rams of medium/large breeds, at the beginning of the breeding season. Ram lambs and adults of small breeds will show slightly smaller dimensions. Primitive breeds tend to have less elongated testes than other breeds. Out of the breeding season these measurements may be considerably reduced, and an opinion on fertility should not be given unless gross abnormalities are present.

General examination (see Table 5.1 and Figure 5.1)

Check the general health and conformation

- Condition score.
- Head, mouth, jaws, eyes.
- Lymph nodes.
- Legs, feet, stance, gait.

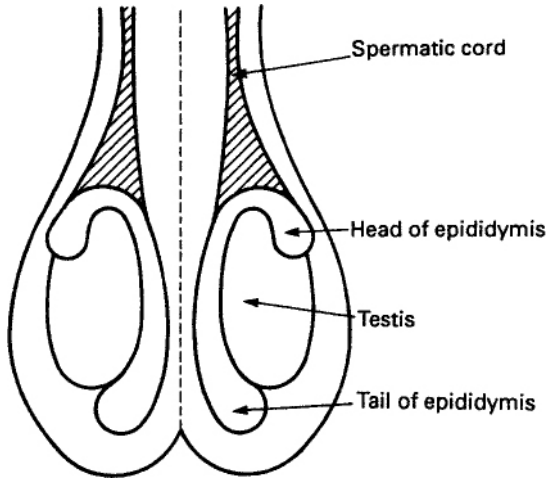


Figure 5.1 Scrotal anatomy.

Check for evidence of gross lesions of the genitalia

With ram in the standing position check:

- Number of testicles.
- Size and symmetry of testicles.
- Shape of testicles – ovoid not cylindrical.
- Presence and size of head and tail of epididymis.
- Mobility of testicles within scrotum.
- Obvious palpable abnormalities.
- Inguinal region for herniation.
- Scrotum for heavy wool cover.

With ram in upright sitting position check:

- Feet and legs.
- Brisket for sores.
- Extent of inguinal 'blush' inside thighs (marked erythema of skin) indicating testosterone surge.
- Scrotal skin for thickening or scarring (including vasectomy scars).
- Testicles for evidence of asymmetry in size or texture.

Table 5.1 Ram examination checklist (A BVA-approved certificate is available).

Owner			
Address			
Date			
Ram identity			
Breed			
Age			
Condition score			
Teeth	Incisors	Molars	
Feet and legs			
Other remarks on general condition			
Scrotum	Circumference Abnormalities		
Testes	Length Diameter Resilience Abnormalities	R R R R	L L L L
Epididymes	Head texture Tail diameter Consistency Abnormalities	R R R R	L L L L
Prepuce			
Penis and appendage			
Semen	Volume Colour Density Motility Live:dead ratio Abnormalities Red cells White cells		
VERDICT			

- Testicular size in relation to breed, maturity, time of year.
- Testicular resilience: 25–30% deformation is normal, 40% is too soft, <20% is too hard – scar tissue.
- Any developmental abnormality, e.g. spermatocele.
- Epididymis for presence and normality of head and tail, or epididymitis.
- Prepuce for evidence of trauma or infection.
- Preputial orifice for trauma or infection.

Extrude penis and check for:

- Full extrusion, normal length is 20–25 cm.
- Injury or anatomical defects.
- Urethral fistula.
- Presence of appendage.

> The function of the appendage is almost certainly to spray semen at increased pressure over the cervix. Total absence without subsequent scar tissue is probably of less significance than a partial absence which may cause 'misdirection' of the ejaculate.

Table 5.2 Hereditary defects in the ram.

Cryptorchid
Testicular hypoplasia
Testicular aplasia
Inguinal hernia
Entropion
Split eyelid
Poor incisor tooth apposition

If no evidence of gross pathology is detected, then the clinician must proceed to a full semen examination. In contrast to the situation when routine pre-service examinations are carried out, when there is some question as to the necessity to ejaculate a ram which appears physically normal, there is an obligation to carry out not only ejaculation but also a full examination, including staining and evaluation of the semen sample (see Table 5.3).

Semen collection

There are three methods of collecting semen:

- With an electro-ejaculator.
- With an artificial vagina (AV).
- From the vagina of a newly served ewe.

Table 5.3 Appearance of semen.

Sperm density (accurate counts can be made with a haemocytometer)	Comment
Watery $< 0.5 \times 10^9$/ml	Probably infertile
Cloudy 0.5–1 $\times 10^9$/ml	Probably infertile
Milky 1–3 $\times 10^9$/ml	Low fertility
Creamy 3–4 $\times 10^9$/ml	Probably fertile
Thick creamy $> 4 \times 10^9$/ml	Probably fertile

Heavily worked rams could show poor quality semen – rest and retest after 1–2 weeks

Motility*	Comment
None	Sperm dead, infertile
Slow wave motion	<50% active, low fertility
Distinct waves with motion	70–80% active, probably fertile
Dark waves with rapid motion	>80% active, ideal

* Beware of cold shock which will affect motility. If semen has cooled warm gently and re-examine. This may revive sperm if cold shock was not severe.

Collection for artificial breeding is done with an AV. Some rams require training before successful use of this method, so most clinicians use the electro-ejaculator method, although welfare concerns have been expressed about its use. Provided it is used with due regard to the welfare of the ram and not repeatedly, it is probably the most practical method of obtaining a sample. The main disadvantage of the method is that accessory gland secretions may be produced rather than a representative semen sample. If this equipment is not available it is possible to allow the ram to serve a ewe in oestrus and collect from the anterior vagina with a long pipette immediately afterwards. This method has the advantage that the ram is observed to see that he is able to mate normally.

A ram should not be condemned on the basis of a single poor sample collected with an electro-ejaculator.

When collecting a semen sample, great care should be taken to avoid cold shock which will give a false impression of the quality of the sample.

Equipment required for semen collection and examination

Ram ejaculator, batteries, lubricant or AV
Microscope with $\times 40$, $\times 100$ and oil immersion lenses
Plate warmer or flat-sided bottle filled with water at body temperature

Clean slides and cover slips
5% nigrosin/eosin stain (freshly prepared if possible)
Leishmann's stain or 'Diff-Quik' slides
Collection tubes and funnel or small polythene bags
Waterproof marker pen
Record sheet

Restraint of ram during collection

Collection may be carried out with the ram either standing or restrained in lateral recumbency, according to personal preference, with sedation provided if indicated. When using the electro-ejaculator, if no sample is obtained after three stimulations of approximately 3 seconds each, the ram should be rested for 30 min before trying again. Semen should be collected into a warmed container and immediately transferred to a warm environment to avoid cold shock.

Check

- Semen quantity.
- Semen density – creamy, watery, milky, marbled, clotted, haemorrhagic.
- Motility (low power) – dense waves, waves forming and dissolving; quivering may indicate urine contamination.

Make stained smear with fresh nigrosin/eosin (1 drop semen to 3–5 drops stain), examine with oil immersion lens.

Check

- Sperm density.
- Live:dead ratio (dead sperm stain pink, live sperm repel stain and are colourless).
- Normal:abnormal ratio.

Make stained smear with Leishmann's or 'Diff-Quik'.

Check for

- Neutrophils. These are not present in normal semen, their presence indicates an infection.

Characteristics of normal semen

Volume – 0.5–2 ml
Density – thick, creamy colour
Motility – distinct or fast swirling wave motion
Dead or abnormal sperm < 20%

> Some coiled or reflected tails are generally thought not to be of significance – probably an artifact due to technique of staining.

It is only at this stage that a verdict on the likely cause of subfertility in the individual ram can be attempted.

> No ram should be considered as infertile on the basis of a single unsatisfactory semen examination, in the absence of gross pathological change. Spermatogenesis and libido may fluctuate. There is also a time delay of some 6–8 weeks between the end of any interference with full sperm production and the appearance of a normal sample. Except in the presence of gross abnormalities, an opinion should not be passed on a ram outside the normal breeding season. In the end, the only true evidence of fertility is pregnancy.

Treatment of infertility

Treatment of infertility is rarely an option within the current mating cycle. Where infection is present and detected early, it is worth trying antibiotic therapy but there is no guarantee that a return to gross normality will be accompanied by normal fertility. Obvious epididymitis and/or orchitis is unlikely to be reversible. If only one testicle is affected it may be worth surgically removing it in the hope that the remaining one will still be functional. In the case of a high value animal which is immature, or has been affected by systemic illness which has responded to treatment, it is worth resting the animal by removing from the ewes for the remainder of the current season and retesting early the following season.

In the commercial situation the most economic course of action is to cull the ram immediately and obtain a new one so that a reasonable lamb crop can still be achieved.

Prevention

On a flock basis prevention depends entirely on routine examination of breeding stock several weeks in advance of the breeding season. New acquisitions should be fully examined, including semen collection; for established rams a full physical examination will suffice, unless there is evidence of need to evaluate a semen sample.

6 Periparturient ewe losses

It is estimated that some 4–6% of ewes die annually, perhaps up to 10% in hill units, and that of these about three-quarters are lost at or near lambing. These losses will rarely be 'sudden deaths', many of the ewes having exhibited signs of illness and probably having received treatment of some kind. Many deaths are undoubtedly related to mismanagement of factors such as prolapses, or obstetrical interferences, but it is likely to be the exceptional shepherd who volunteers responsibility for a death by admitting ineptitude. However, deaths do occur at this time in spite of the best endeavours of conscientious and skilled shepherd care, but a death rate in large units of above 2% (except in difficult environmental conditions) should give rise to concern.

This chapter deals specifically with losses related to pregnancy and parturition, and in particular with the flock which exhibits an unacceptable rise in losses. Further relevant information may be found in Chapter 22 on sudden death and in Appendix 1 on postmortem examination.

In any investigation of a flock with an unacceptably high periparturient ewe loss, the following facts need to be established:

- The history of the incident.
- Incidence of losses in relation to previous seasons.
- Timing of losses in relation to parturition.
- Body condition of affected ewes and rest of group.
- Any signs observed.
- Any treatments given and their efficacy.
- Vaccination history of the flock, particularly clostridial and pasteurella programme.

In particular, the following factors should be checked (see also Table 6.1 and Figure 6.1).

If losses are before lambing period

Check

- How far from lambing.
- Body condition and nutrition.
- For evidence of impending abortion – vaginal discharge (see Chapter 3).
- For scouring – possible salmonellosis.
- For evidence of recumbency – excoriation of lower limbs, trauma to eye area, hypostatic congestion. This may indicate metabolic disease (calcium deficiency or pregnancy toxaemia).

Table 6.1 Aids to diagnosis in periparturient ewe losses.

	Gross pathology	Biochemistry	Microbiology
Pregnancy toxaemia	Fatty liver Fetal overload Poor fat reserves	Urine – ketones Serum – BHB Plasma – glucose	None
Hypocalcaemia	None	Aqueous humour – Ca, Mg CSF – Ca, Mg	None
Septicaemia	Petechiae Fluid in serous cavities		Culture heart blood, liver, lung, spleen
Clostridial disease	Haemorrhages and gas in affected area especially perineum		Tissues for IFAT
Abortion	See Chapter 3		

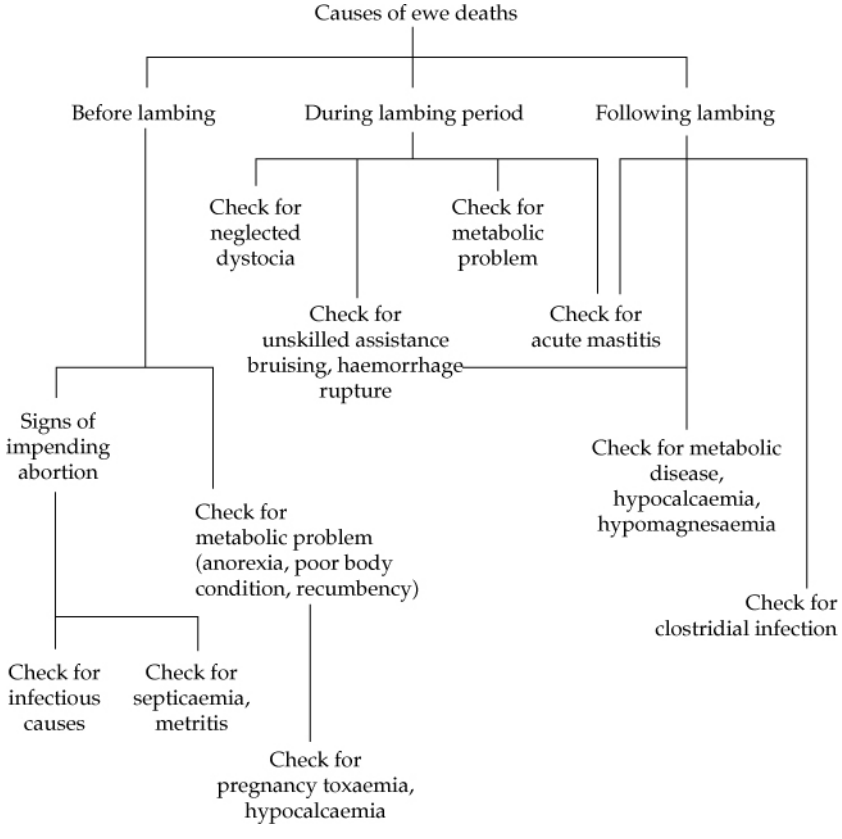


Figure 6.1 Periparturient ewe losses.

> Aqueous humour is a useful sampling fluid in dead animals, as concentrations of minerals and metabolites are stable for up to 48 h.

If losses are during lambing period

Check as above plus

- Whether parturition has commenced – evidence of fetal fluids, fetal parts, or placenta.
- Skill of shepherd, students or other helpers.
- For external evidence of bruising or trauma of vulval area, haemorrhage.
- For concurrent abortion problem.
- For neglected cervical or vaginal prolapse.
- For prolapsed intestines.

If losses occur after lambing

Check for

- Vulval or vaginal bruising following dystocia or manual interference.
- Signs of haemorrhage.
- Severe vulval swelling, gas or discolouration – clostridial infection.
- Metritis – purulent discharge.
- Evidence of acute mastitis – gangrene.

Postmortem examination

It will almost always be necessary to carry out postmortem examinations of a representative selection of affected animals. Because of the degree of supervision at lambing time there is likely to be less delay between death and PME than at other times. For details of postmortem technique see Appendix 1.

At postmortem examination these fundamental questions should be answered:

- Was the death a direct result of pregnancy or parturition?
- Was death due to other disease but triggered by the stress of pregnancy or parturition?
- Was death unrelated to pregnancy or parturition? (see Chapter 22)

Particular points to note during the postmortem examination

Death before commencement of parturition (cervix closed)

Check for

- Poor nutrition – condition, fat deposits.
- Nutrition-related disease – pregnancy toxaemia (fatty liver, urine sample for ketones).
- Metabolic disease – calcium deficiency (sample aqueous humour).
- Evidence of separation of placenta.
- Placentitis – impending abortion.
- Time of death of fetus in relation to maternal death.
- Disease unrelated to pregnancy, e.g. pasteurellosis.

Death during parturition (cervix partly or completely open)

> If the cervix is patent, fetal autolysis is very rapid within the uterus (death, emphysema and disintegration may take as little as 48 h).

Check for

- Dystocia – ringwomb, malpresentation.
- Manual interference – bruising, haemorrhage, trauma.
- Impending abortion, placentitis, mummification.
- Signs of septicaemia.
- Catastrophe – torsion of uterus, rupture.
- Other disease unrelated to parturition.

Death after parturition completed

Check for

- Manual interference – bruising, trauma, haemorrhage, uterine rupture.
- Retained placenta, or metritis – placental separation normally occurs very rapidly, and in contrast to the cow, retention is rare.
- Clostridial infection – rapid decomposition, gas in tissues.
- Choking on fetal membranes.
- Metabolic disease – calcium or magnesium deficiency (aqueous humour sample).
- Acute mastitis.

> Particular care must be taken in the interpretation of any bacteriological results of material taken from the uterus if parturition has started or been completed, since bacteria will have been introduced in any manual interference.

Prevention

The loss of ewes at or near lambing is of particular significance in both welfare and economic terms. Nearly all the variable costs will already have been incurred and output of ewes involved is jeopardised or lost. Extra work involved in fostering or rearing any surviving lambs is unwelcome. Loss of ewes through a poor standard of management of dystocia or lambing-related problems has particular welfare implications.

If poor management is a factor, the priority lies in improving the skills of the shepherd through a course either in-house or at a local agricultural training centre. The first of these is preferable. Setting up such a course demonstrates commitment of the practice to the welfare of stock under its care and also shows an involvement in the economic survival of the client.

If a particular disease has been implicated it may be possible to implement a specific vaccination programme, for example for clostridial disease, pasteurellosis and some forms of abortion.

7 Mastitis, udder and teat lesions

Initially, it would appear that the diagnosis of mastitis in the ewe is so self-evident that it does not require any amplification. However, losses through deaths and premature culling can be significant in some flocks, with estimates made of a 5–10% annual culling rate as a result of mastitis. In a slaughterhouse survey of culled ewes, 50% were found to have udder abnormalities. In addition the advent of sheep milk production as a method of diversification has brought its own special problems.

In flocks other than dairy sheep, cases of mastitis are usually presented to the veterinarian at a much later stage in the disease process than is the case with dairy cattle, because there is no day-to-day inspection of the udder. The clinician will be concerned with changes in incidence, the causes of such change and future prevention rather than the diagnosis of the presence or absence of overt disease. The incidence of mastitis in a flock can vary widely even in the absence of any obvious management change. Frequently the clinician will fail to identify clearly any cause for a sudden increase in incidence.

In some cases, particularly where the owner is inexperienced, a sheep with acute mastitis may be presented as a case of lameness, since often the first sign of acute mastitis is dragging of the hind leg on the affected side. It is essential, therefore, that the clinician checks the udder if the patient is near to lambing, is lactating or is in the immediate post-weaning period. In addition, the presence of udder or teat lesions should always be suspected if lambs appear constantly hungry, or if the ewe does not stand to allow hungry lambs to suck.

Nondairy flocks

Bacteriological tests are always necessary to determine the cause of mastitis cases, since there are no clinically distinguishing features.

These organisms have been implicated most commonly:

- *Mannheimia (Pasteurella) haemolytica.*
- *Staphylococcus aureus.*
- Mixed infections involving *M. haemolytica* or *Staph. aureus* are also common.

Other less common causes include:

- *Streptococcus* spp.
- *E. coli.*
- *Arcanobacterium (Corynebacterium) pyogenes.*

Most of these organisms are normal commensal bacteria. *M. haemolytica* is thought to be transferred from the pharynx of the lamb to the teat of the ewe. Organisms then presumably gain entry into the udder via the teat canal, because of some loss of the defensive integrity of the teat sphincter and canal. Teat and udder lesions are, therefore, closely linked to the incidence of mastitis.

Less likely possibilities:

- Maedi-visna (MV) causes an indurative mastitis.
- *Leptospira interogans var. hardjo* has been implicated in otherwise fit ewes with poor milk production after lambing.
- *Mycoplasma agalactiae* (contagious agalactia) is an important cause of mastitis in the Mediterranean region and Middle East, but is not present in the UK (notifiable).

Common times for occurrence of mastitis

Mastitis is most commonly seen at four stages in the production cycle (see Table 7.1):

Table 7.1 Mastitis.

Stage of lactation	Predisposing factors
At lambing - blind teat	Weaning policy Inadequate culling examination
After lambing - acute	Hygiene, bedding
Full lactation	Supply/demand imbalance Large litters Inadequate nutrition Failure to provide lamb creep Orf infection Teat lesions Udder exposure - winter shearing, short docking, excessive crutching, lack of shelter
Mid/late lactation	Supply/demand imbalance Marketing policy - removal of one of twins
Post-weaning/culling examination	Weaning policy - removal of one of twins only, no reduced inputs to discourage lactation, no use of dry ewe therapy

- At or near lambing - often the results of an earlier chronic infection which only becomes apparent at this time. Acute mastitis at lambing is rare in sheep in contrast to cattle.

- At 4–8 weeks into lactation.
- After weaning.
- At the culling examination.

Udder problems at lambing

Factors such as poor teat conformation, injury, or the inability of the lamb to suck may predispose to mastitis if the udder is not emptied.

If individual ewes have congested udders, or lambs are hungry

Check

- If clinical mastitis is present.
- Teat canal for patency. Wax plug may block teat (this is normal, and is usually removed when the lambs first suck). Occlusion of the canal with a 'fibrous' cord indicates infection during a previous lactation.
- For absence of canal (congenital).
- For inverted teats (congenital).
- For shearing injury (partial or complete removal of teat).
- Teat/udder conformation – 'bottle tits'.
- Lamb viability – can it suck?

Treatment

See below for treatment of mastitis. If the lambs are unable to suck because of weakness, the cause of weakness needs investigating. Poor teat conformation can be overcome by milking out the udder and feeding lambs with a bottle or stomach tube until they become strong enough to cope with the ungainly teats.

Most of the other conditions are untreatable.

If many ewes have insufficient colostrum at lambing or insufficient milk early in lactation

Check

- Ewe condition and feeding in late pregnancy.
- Possibility of *Leptospira interrogans var. hardjo* infection – this has been reported as a cause of lack of milk in groups of otherwise apparently healthy ewes. If suspected, take blood and milk samples for culture at time of milk lack. Positive serology is not necessarily evidence of disease as this is apparently quite widespread.
- Possibility of MV – causes indurative mastitis. Blood sample proportion of affected ewes for serology.

Treatment

If there is evidence of inadequate feeding it may be possible to raise intake over 2 or 3 days based on a high protein (up to 22%) feed. Those ewes still some way

off lambing should have sufficient time to benefit. Beware of introducing this type of food too quickly, causing digestive upsets.

Leptospirosis may be treated with an antibiotic mixture containing streptomycin, but milk yield may not return to normal.

If MV is diagnosed, the extent of infection throughout the flock needs full investigation. Culling positive animals will be necessary (see Chapter 21).

Udder problems at 4–8 weeks into lactation period

These will mainly present as obvious clinical mastitis (sometimes the fatal gangrenous type), sometimes associated with teat lesions or, less obviously, as failure of lambs to thrive.

Check for

- Clinical mastitis.
- Insufficient milk to supply demands of lambs. In the case of prolific ewes suckling multiples, especially triplets, the lambs may make constant demands upon the ewe for milk. This is particularly seen if no special arrangements have been made to manage them separately as far as ewe nutrition and provision of lamb creeps are concerned. Competition for teats often leads to teat damage with the formation of painful thickened plaques on the medial surface of the teats, in the position at which the incisor teeth of the lambs contact the teats (see colour Figure 3). Ewes usually refuse to allow the lambs to suck, and mastitis is a common sequel.
- Staphylococcal infection of teats.
- Orf lesions (see colour Figure 4). These may begin on the lambs' mouths and be transferred to the teats or vice versa. Dry scabs should be submitted for confirmation by electron microscopy.
- Orf vaccination of ewes too close to lambing – transfer of vaccine virus via lambs to teats (minimum recommended time between ewe vaccination and lambing is 8 weeks).
- Use of orf vaccine in lambs without prior ewe vaccination.
- Winter shearing or excessive crutching. There is a suggestion that these practices predispose to mastitis because of chilling of the udder. There is also a suggestion of breed susceptibility, e.g. Mule.
- Oversupply of milk. This period coincides with a change from primarily 'milk nutrition' in the lamb, to primarily 'grass based' nutrition. In the case of ewe breeds with a high lactation potential on high planes of nutrition, supply and demand for milk will rapidly become out of balance. The resultant back pressure will act as a potential cause of udder inflammation and infection as has been established for many years in the dairy cow.

Treatment of mastitis

Clinical mastitis is rarely detected in time to save the affected gland, except in milking sheep when an early diagnosis is more likely to lead to a successful

outcome. If an early case is seen, a similar regimen to that adopted in cattle is appropriate, although there will be the complication of the lambs to deal with. If there is hope of saving the gland, temporary separation of lambs (but leaving visual contact) will be necessary to allow intramammary treatment to have an effect.

In acute cases, particularly where gangrenous change is present, a decision has to be made as to whether treatment is justifiable on both welfare and economic grounds. If so, perhaps in the case of a valuable pedigree ewe, intravenous antibacterial therapy, NSAID administration and nursing support are necessary. Drainage of the gland may be improved by slitting or amputating the teat. Later during the recovery phase, amputation of the necrotic part of the udder may be necessary. The clinician needs to be aware of the likelihood of large blood vessels still being present, so the procedure must be done carefully and vessels adequately ligated. If an obvious pedicle is present a castration ring sometimes provides a good method of haemostasis.

Treatment of teat lesions

These are often very painful, causing the ewe not to allow lambs to suck. They should be treated as a matter of urgency, as mastitis is likely to result if neglected. There is no specific treatment for orf virus, but antibiotic treatment will reduce secondary infection. Staphylococcal lesions will respond to broad-spectrum antibiotics and NSAIDs, aided by local application of antiseptic udder cream. Ewes should be restrained several times daily to allow the lambs to suck. This may seem harsh but lambs sucking are the most effective way of removing milk from the udder and vigorous treatment should bring about a rapid improvement.

Prevention of mastitis and teat lesions during lactation

This is often difficult but a full investigation of possible predisposing factors should be made. Prolific ewes are particularly susceptible if demand for milk by the lambs outstrips supply. Ewes rearing triplets should be managed separately if possible, with extra concentrates provided and creep feed introduced for the lambs. A continuous check on growth rate of lambs should be made and, where growth rates within a litter differ, removal of either the smallest or largest lamb for artificial rearing may be economically beneficial.

High lactation ewes will benefit from provision of shelter and avoidance of excessive crutching, as chilling of the physiologically active udder may predispose to problems.

Udder problems after weaning

Many of these infections are subclinical during the suckling phase and only become apparent after weaning. Sudden removal of the lambs, with a build up of milk in the udder, allows the infection to become clinically apparent. There is the common danger that many such infections will not be identified until the

culling inspection, by which time permanent damage to udder function will have occurred.

Check

- Weaning technique – removal of one of twins (e.g. early prime lamb for slaughter) may result in one side of the udder becoming distended. Unless careful watch can be kept on the udder it may be better management to wean both lambs simultaneously.
- Feeding after weaning – a short period on minimal inputs, e.g. straw, may be necessary to rapidly terminate lactation (but not restriction of water supply).
- For healed teat lesions, over-sucking, orf.
- For use of dry ewe therapy – this should be preventive, but unhygienic application has been known to result in severe flock problems.

Treatment
Most udder problems are untreatable at this stage, since the udder tissue will be irreversibly damaged.

Prevention
Weaning management is *central*. All lambs should be removed simultaneously and ewes put on to a low plane of nutrition to rapidly stem milk production. Where a flock problem is apparent, dry ewe therapy may be valid on welfare and economic grounds. Some bovine products are licensed for use in sheep. Infusion must be carried out in a hygienic manner and the temptation to use one tube between both teats avoided, as this risks transfer of bacteria between the two halves of the udder. Infusion should be carried out by apposing nozzle and teat end – there is no need to insert the nozzle into the teat.

Udder problems at the culling inspection

These infections are those described in the previous paragraph, i.e. that occurred at weaning, but were not noticed at that time, since concurrent systemic disease is rare. The clinician is not likely to be called in to identify the presence of such lesions, but only requested to find reasons for high or changed incidence.

Treatment
Not appropriate.

Prevention
Weaning management is likely to be implicated – see above.

Dairy sheep flocks

It is becoming apparent that mastitis occurs in the dairy sheep flock in very similar patterns to those which exist in the dairy cattle industry. Presumably

the stresses of machine milking, with all the risks of damage to the integrity of the teat and sphincter, are very similar. The changes to the udder are not usually as severe as those in the ewe suckling her lamb, although gangrenous mastitis can occur. This is not primarily due to differing infections, but because the sheep milker has the same frequent opportunities as the cow milker to observe changes in the udder, and therefore to initiate treatment and control. In addition, the practice of preventive therapy is much more justified in this type of enterprise.

When called to the 'milking' flock the clinician will have a large body of experience in dairy cattle to apply to the control of the problem. Diagnosis, treatment and prevention should be carried out as for the dairy cow, for the dairy sheep is effectively 'a cow with wool' rather than a sheep which happens to give a lot of milk.

The investigation will include:

- Assessment of parlour and dairy hygiene.
- Use of udder washes and disposable teat cloths.
- Milking routine.
- Teat dipping.
- Milking machine maintenance.

The present state of knowledge of sheep milking machine technology is still in the developmental stage, so optimum vacuum and pulsation rates are not well established. Reference should be made to the milking machine manufacturer for further advice.

8 Perinatal lamb losses

The success or failure of a flock depends to a large extent on its performance around lambing time. Attention has already been drawn to the importance of periparturient ewe losses, which form a large proportion of the estimated 10% total annual ewe losses. Although the lambing percentage for the UK as a whole is approximately 125%, it is disturbing but true that the net output is less than 100%, and analysis of flock outputs has shown that as many as 5 million lambs that have reached full term fail to reach the point of sale. The figures for ewe and lamb losses taken together give an alarming overall annual death rate of about 17% of the total sheep population. Clearly then, the efficient sheep keeper experiencing significant losses will be concerned with the cause and will require answers from an advisor within a very short timescale, i.e. within this lambing season.

Numerous surveys of lamb losses have indicated that some 90% of these occur perinatally. Losses in individual flocks vary widely depending on husbandry, weather, shepherd skill, etc., but may exceed 25% of those born. Indeed, some flock owners may be unable to give an accurate figure, preferring to remain in ignorant bliss! Analysis of losses has concluded that 'whilst ewe losses are due to disease, lamb losses are due to management'. But the fatalism of some older traditional shepherds that 'you are always going to lose some lambs' may lead to communication difficulties. Institution of some form of record keeping must, therefore, be a priority in any such flock. However, it must be realised that while it is easy for the clinician to insist on full records from the security of a warm surgery, it is not so simple for the shepherd to record in detail the results of a very difficult lambing – a sick ewe, three stinking lambs, and six more sheep 'waiting'!

What then are the normally expected or acceptable losses?

- Zero should be that which is acceptable.
- Less than 5% should be that which is expected.

In reality, losses of more than 10% are 'normal' even in lowland conditions, while on the high hill the figure may reach several times higher. The clinician must be aware of the pattern of loss during the whole perinatal period if valid advice is to be given. Losses occur in two phases:

- Lambs born dead are often quoted as 8–10%.
- Lambs dying in the first 72 h are often quoted as 10–12%.

In fact, these figures contain great untruths. If lambing is carried out under a regime of total shepherding, i.e. 24 h supervision, then the truly 'born dead'

figure is often extremely low (2–3%). This shows that, in all probability, in less supervised flocks many of those classed as 'born dead' were actually alive but failed to live because no one was present to revive them. If the same management pressure is applied within the next 72 h, then the losses during this time are reduced to 1–2%. Thus the difference between these 'unavoidable' deaths and the generally accepted rates quoted above could be classed as 'avoidable' deaths. (Whether it is good policy to adopt the 'save everything' attitude, and perhaps perpetuate inherited characteristics such as poor mothering ability, is another matter.)

So we are left with the fact that lamb losses are essentially a result of failures in management. MLC figures comparing losses in top-third performing flocks with those in average and bottom-third performing flocks confirm that improvements are achievable, with number of lambs reared being responsible for 20% of their superiority. It must be recognised, however, that even in well-managed flocks lambing out of doors, sudden adverse weather conditions can have a marked influence on these figures in spite of the best efforts of the shepherd.

It is against this difficult background that the clinician must operate, and will require top quality communication abilities in addition to good clinical skills, if conclusions are to be accepted and applied by both owner and shepherd. With the contrast between achievable loss rate and actual loss rate being so wide, perhaps the greatest challenge to the clinician will be to avoid inducing a guilt complex for the owner and therefore immediate rejection of recommendations. If the owner can convince himself that losses are inevitable and that management plays no part, he will be content but have no incentive to improve.

Flock investigation (Figure 8.1)

Attempts should be made to categorise the losses into antepartum (e.g. abortion agents), intrapartum (dystocia), or postpartum categories. By far the most important cause in this last category, accounting for some 30–50%, is hypothermia (starvation and/or chilling) which is often the result of the dystocia–mismothering–exposure–starvation sequence.

In obtaining a history and carrying out a general examination of the flock, the following points will be of particular relevance:

- Breed.
- Normal litter size and lamb weights.
- Hardiness.
- Ewe nutrition particularly in last 8 weeks of pregnancy.
- Ewe condition scores.
- Vaccination history.
- Age group of ewes from which losses are occurring, e.g. young, old, thin, fat.
- Any signs of infectious agents operating in ewes, e.g. abortions, or lambs, e.g. enteritis.
- Colostrum production by ewes, i.e. udder development, teat function.

- Lamb management immediately after birth.
- Routine for checking colostrum intake.
- Use of stomach tube.
- Provision of colostrum bank (species used?).
- Method of defrosting and heating colostrum (overheating will denature IgG).
- Hygiene in lambing pens.
- Navel hygiene.
- Timing of castration and docking if done.
- Use of individual lambing pens (bonding).
- Management of multiples especially triplets, quads, etc.
- Provision of heat lamps or warming boxes.
- Outdoor management, availability of shelter.

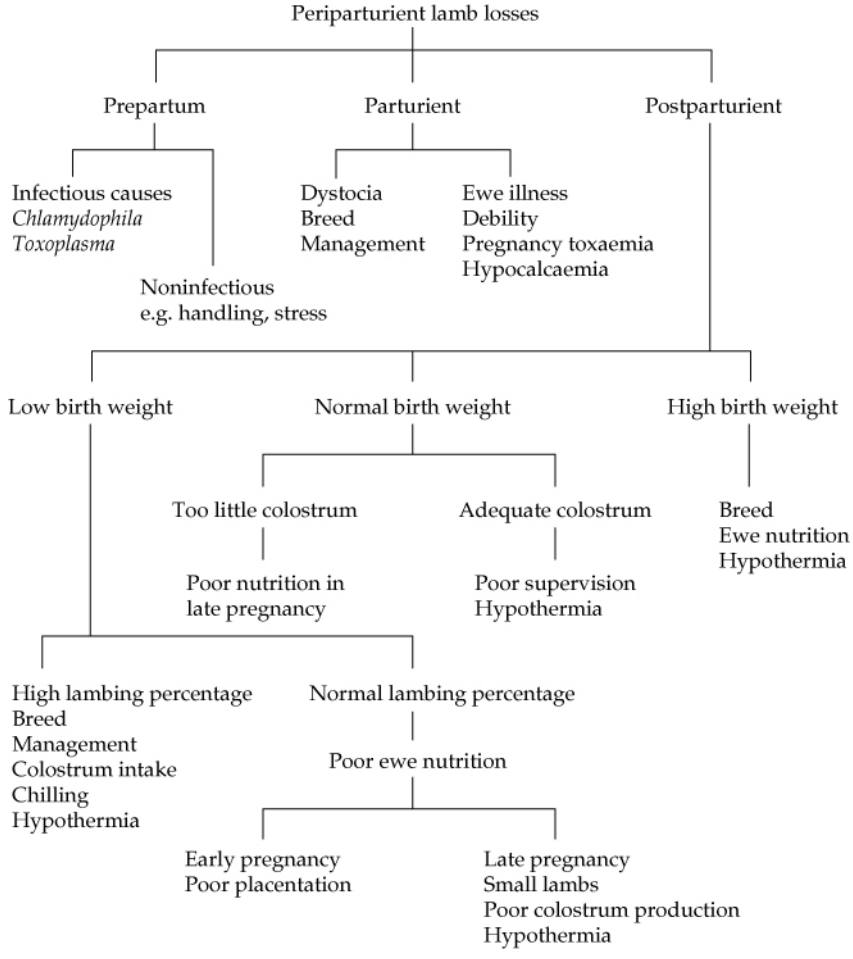


Figure 8.1 Investigation of periparturient lamb losses.

Postmortem examination

In any investigation of unacceptable lamb losses, it is essential that as much postmortem material as possible is examined. Do not rely on the findings in only a small number of carcasses as there may well be a multiplicity of causes of death.

Although the following list may seem alarmingly long, the technique of postmortem examination in young lambs is usually simple and quick to carry out. In combination with laboratory help where necessary, it will usually allow the clinician to identify the main stages and area(s) of loss. Examination should be carried out in a logical manner so that shortcuts are not taken which may lead to inaccurate diagnosis, and a standard recording form is of benefit to guard against this. Where infectious conditions are suspected, e.g. neonatal septicaemia or enteritis, samples should be selected as the postmortem examination proceeds. The most useful samples will be liver, spleen, lung, heart blood; with, in addition, intestinal contents plus mesenteric lymph node for enteritis, joint fluid in polyarthritis, brain for swayback and border disease.

Checklist for PME of young lamb

Single/twin/triplet/quad
Full term/premature
Weight – normal/underweight/excessively large
Fresh/decomposing/mummified
Fleece dry/wet – fetal fluid/meconium staining/rain-soaked
Fleece type – normal/premature/excessively hairy
Predator damage – after/before death (signs of haemorrhage)
Eyes – normal/sunken (dehydration)
Cornea – clear/opaque (indicates time of death)
Mouth – clean/wet/congenital deformity/trauma/orf
Navel – wet/dry/antibiotic spray or iodine applied/thickened/infected
Feet – walked/not walked (foot membranes present)
Anal region – meconium/orange colostral/scour/blood
Subcutaneous oedema – dystocia (part of body?)/hypothermia (extremities, yellow colour)
Muscles – colour/dryness/wasting
Blood – normal/watery
Abdominal cavity – clear fluid (<1 ml is normal)/fibrin/blood
Umbilical arteries – end straight/end tapered/clot
Bladder ligament – normal/oedema/fibrin/pus
Abomasum – milk clot/liquid milk/saliva/gas/wool/empty
Intestines – food present/absent/hyperaemia/gas/torsion (small undigested clots in colon indicate alimentary problem)
Food absorption – good/poor/absent (mesenteric lacteals should be white up to or beyond mesenteric lymph nodes if absorption is good)
Meconium/faeces – present/absent/imperforate anus
Liver – haemorrhage/rupture/abscesses
Kidneys – normal/autolysed/enlarged/pale (nephrosis)
Perirenal fat – normal/metabolised (gelatinous, pink)

Ribs – fractures
Thorax – clear fluid/fibrin/haemorrhage
Lungs – uninflated (sinks in water)/partially inflated/fully inflated (floats in water)/pneumonia
Pleural cavity – fluid (<1 ml normal)/pleurisy
Pericardium – fluid/fibrin/fat present/metabolised
Heart – congenital defects
Epicardial fat – present/metabolised
Joints – normal/enlarged
Joint fluid – absent/clear/cloudy/pus
Bone marrow – red/pale
Brain – haemorrhage/cavitation/congenital deformity

> Brown fat stores are metabolised in a set order. It is thus possible to assess the degree of starvation. Pericardial fat is used first, followed by that in the coronary groove, around the left coronary artery and finally the renal fat. If these last two sites are metabolised (indicated by gelatinous pinky material), starvation is definitely indicated.

As a result of this examination it should be possible to place the death in one of the categories in Tables 8.1 and 8.2.

Guide to cause of death (see also Table 8.3)

Antepartum deaths

Mummification, autolysis, collapse of eyeball, haemoglobin staining, all indicate dead a long time

Check for

- *Toxoplasma* abortion.
- Border disease.

Renal autolysis, but little autolysis of rest of body, some corneal opacity, wool loosening, square end to umbilical artery with no thrombus (or sometimes thrombus whole way along), indicate dead only a few days prepartum

Check for

- Infectious cause, e.g. chlamydial abortion.
- Fetal overload, placental insufficiency.
- Recent handling, dog worry, etc.
- Maternal disease – pregnancy toxaemia, calcium deficiency, vaginal prolapse.

Table 8.1 Time of death in common diseases in lambs.

Prepartum		Maternal disease, e.g. pregnancy toxaemia, hypocalcaemia Placental insufficiency – fetal overload Abortion – infectious agents
Partum		Dystocia Maternal disease, e.g. pregnancy toxaemia, hypocalcaemia
Postpartum	At birth	Parturient trauma Congenital disease, e.g. swayback, border disease, cerebellar hypoplasia, nutritional myopathy
	Hours	Failure to suck Mismothering Exposure → Hypothermia
	Day 1	Watery mouth Hypothermia
	Day 2	Lamb dysentery Enteritis (*E. coli*, rotavirus) Neonatal polyarthritis Navel ill
	Day 3	Liver abscess
	Day 4	Spinal abscess
	Day 7	Orf
	Any time	Accident Trauma Predator

Table 8.2 Postmortem features indicating time of lamb death.

Features	Time of death	Conclusion
Mummification, autolysis, collapse of eyeball	Antepartum	Dead a long time
No thrombus in umbilical artery, square end to artery	Antepartum	Recently dead
No thrombus in umbilical artery, tapered end to artery, renal autolysis	Intrapartum	Early in parturition
No thrombus in umbilical artery, tapered end, no renal autolysis, localised oedema	Intrapartum	Late in parturition
Thrombus in end of umbilical artery, lungs uninflated	Postpartum	In first few minutes
Lungs inflated, hoof membranes present but may be beginning to separate, navel cord wet	Postpartum	In first few hours
Hooves hardened, navel cord shrivelled	Postpartum	Lived a few days

Table 8.3 Predisposing factors in lamb deaths.

Problem	Result
Abortion	Premature low birth weight lambs
Poor ewe body condition	Low birth weight lambs Poor colostrum supplies
Ewe age	Young - inexperienced mothers Old - poor colostrum supply
Ewe disease	Poor maternal ability Poor colostrum supply
Litter size	Inadequate colostrum for all lambs
Birth weight	High - dystocia Low - hypothermia
Dystocia	Subcranial haemorrhage Hypoxia Poor temperature regulation
Housing	Reduces hypothermia risk Increases infection risk
Weather	Hypothermia risk

Intrapartum deaths

Pointed end to umbilical artery, no thrombus, renal autolysis, little localised oedema, indicate death early in parturition

Check for

- Ewe debility or illness.
- Weak lambs resulting from abortion agents.

Localised oedema, no kidney autolysis, no thrombus, pointed end to artery, indicate death late in parturition, i.e. dystocia

Check

- Degree of supervision.
- Shepherd knowledge and skill.
- Breed of ewe and ram - disproportion.

Meconium staining of coat may indicate fetal distress during parturition, or prolonged labour.

Postpartum deaths

Lambs with severe congenital abnormalities, e.g. hydrocephalus, severe swayback, may die soon after birth or may require euthanasia.

Lungs uninflated or poorly inflated, thrombus at end of umbilical artery, indicate immediate postpartum death

Check for

- Injury during birth – fractured ribs, liver haemorrhage, brain haemorrhage.
- Prolonged parturition – anoxia, placental separation.
- Lack of supervision at birth – asphyxia in membranes.
- Severe congenital abnormality.

Lungs inflated, foot membranes still present, indicate lamb has breathed but not walked

Check for

- Dystocia causing brain damage – especially in large single. These rapidly succumb to hypothermia because they are unable to control body temperature.
- Severe swayback – cavitation of brain may be visible macroscopically.
- Border disease – fleece changes, tremors in live lamb.
- Other congenital abnormality, e.g. cerebellar hypoplasia – abnormally high head carriage or opisthotonus in live lamb.
- Severe nutritional myopathy – white streaks in muscles, blood sample ewes for glutathione peroxidase (GSH-Px).

Lamb has walked (foot membranes absent), empty gastrointestinal tract, meconium may not be expelled, brown fat partially or completely metabolised, indicate failure to feed

Check

- Degree of postpartum supervision.
- If one of multiples especially triplets.
- Ewe udder – mastitis.
- For mismothering, rejection, inexperienced mother.
- For extreme chilling – lack of shelter, severe weather.
- For congenital abnormality – e.g. cleft palate.

Lambs dying at more than 1 day of age (fetal fluids dried, navel partly or completely dry)

The most important cause is hypothermia/hypoglycaemia (exposure and/or starvation) – see below for more details. The now common use of a stomach tube for feeding may give a false impression of the lamb not having starved. Recent feeding by stomach tube may be indicated by presence of liquid milk rather than clot, no absorption into lacteals, and metabolism of brown fat reserves.

Check for

- Starvation – empty gastrointestinal tract (unless stomach tubed as above), brown fat stores metabolised. This is most likely to arise from lack of colostrum due to mismothering, undernutrition of ewe, large litter, udder abnormality.
- Exposure – in addition to empty gastrointestinal tract and brown fat metabolism, there is usually some yellow-coloured oedema of the extremities. Bad weather and lack of shelter are the common causes.
- Subclinical copper deficiency – lambs may be of low viability, succumbing to hypothermia, and yet not show swayback.

> Remember that FMD causes sudden death in lambs because of the effect of the virus on heart muscle.

Muzzle is wet, abomasum filled with saliva and gas, and often meconium not expelled, indicates watery mouth

Check for

- Supervision of colostrum intake – amount and delay after birth. This condition is now thought to be a result of the action of bacterial endotoxins produced in lambs with a delayed or inadequate colostrum intake. Research indicates that lambs which have received colostrum within 30 min of birth do not succumb to watery mouth. It is probable that 150–200 ml of good quality colostrum, i.e. from the first milking, are adequate to prevent this condition (but not enough to prevent hypothermia).

If enteritis or dehydration is present

Check for

- Digestive scour.
- *E. coli* scour.
- Lamb dysentery.
- *Salmonella.*

- Rotavirus.
- Cryptosporidia.

Laboratory tests are likely to be required for confirmation of diagnosis. For full details, see Chapter 10.

If signs of local or generalised infection (neonatal septicaemia) are present, e.g. navel infection, joint infection, liver abscess, spinal abscess

Check

- Hygiene at lambing.
- Lambing pens – cleaning out, bedding, etc.
- Technique of routine navel disinfection (a quick squirt with a purple spray may not be adequate in the face of pathogenic bacteria, or iodine dip may be poured back into stock bottle after use).
- Colostrum intake – volume and timing. Check serum samples from several lambs for IgG content. Values <20 mg/ml indicate inadequate colostrum supply or delayed intake, <30 mg/ml indicate moderate supply, >40 mg/ml good supply. Zinc sulphate turbidity test is an alternative – <20 units indicates poor or delayed uptake.
- Possibility of subclinical copper deficiency – such lambs may be of low viability, yet not showing swayback, and may succumb more easily to infections.
- For tick pyaemia if in tick area.

Laboratory tests are likely to be required if the pathogen is to be identified. While common organisms such as *E. coli*, *Staph. aureus* and *F. necrophorum* are likely to be found in many cases, other organisms such as *Listeria*, *Chlamydophila*, mycoplasmas, *Erysipelothrix* and streptococci may be responsible, and their isolation will affect treatment.

Other common causes of neonatal deaths

- Predation – care should be taken to distinguish whether this occurred before death and was a contributing factor, or whether it occurred after death. If before death, there should be signs of bruising and haemorrhage at the damaged sites. Lambs weak for other reasons are most likely to succumb to predators, e.g. foxes, or crows which peck out eyes. Predation may also take place during parturition.
- Inhalation pneumonia – this may occur where weak lambs are fed with a bottle, where a stomach tube is used incorrectly, i.e. placed in the trachea or too high up the oesophagus, or where a moribund lamb is fed by stomach tube and regurgitation takes place (such lambs have no swallowing reflex).
- Severe gingival orf – starvation is the usual cause of death in such cases.
- Nutritional myopathy (selenium/vitamin E deficiency) – in some affected

flocks, lambs may be weak or unable to stand at birth. White streaks or patches should be visible in skeletal or cardiac muscle, and diagnosis can be confirmed by histology and GSH-Px estimations in the same group.

- Anaemia caused by feeding cow colostrum – this condition should be suspected if the carcass is pale with very watery blood. Deaths occur at 1–3 weeks of age. See Chapter 17.
- Trauma – this is a common cause of death, particularly fractured ribs caused by treading by housed ewes.
- Overlying – again in housed animals. Lambs are simply found dead, but if careful attention is paid to the head, this often appears flattened where it was trapped by the ewe sitting on it.
- Ruptured liver – deaths have been reported in lambs up to 3 weeks of age, associated with low vitamin E but normal selenium status. Check blood samples for vitamin E as well as GSH-Px and liver samples for vitamin E.

Dealing with higher than acceptable perinatal lamb losses

As already indicated, making a decision as to the main cause of lamb loss is the first priority. In many cases this will be multifactorial, therefore the whole management of the pregnant ewe and lambing time will need to be examined.

If antepartum deaths are the main feature and an abortion agent is identified, the appropriate action as discussed in Chapter 3 should be taken. If no infectious agent is identified after repeated sampling, management and handling of ewes in late pregnancy should be carefully examined.

If intrapartum deaths are the main feature, supervision of the lambing ewe must be the major cause for concern, with retraining of staff dealing with the animals being a priority. If dystocia is caused by oversized lambs it may not be possible to take avoiding action this season, unless ewes are still some way off lambing, other than to identify ewes in difficulty early in the parturition process and consider caesarean section.

If postpartum deaths are the main feature, there may be some scope for identifying and treating affected lambs before they actually die, but the main priority must be to identify any management failures, for the majority of losses at this stage are the result of these.

The most important factors to consider are:

- Colostrum intake.
- Hygiene of shepherds and in lambing area.
- Bonding of ewes and lambs and avoidance of mismothering.
- Provision of shelter.

These will now be considered in turn.

Colostrum intake

An early (within 30 min of birth) and adequate intake of colostrum will protect against hypothermia, watery mouth and many of the infectious causes of ill-

ness and death in young lambs. Provision of a colostrum store, preferably by milking out excess ewe colostrum, augmented by other sources such as goat or cow colostrum or commercial substitutes, is an essential management activity. All staff should be trained in the use of a stomach tube for easy administration of colostrum. Suspect lambs such as triplets, small lambs, very large lambs or those from sick or inexperienced ewes should be monitored and tube-fed if there is any doubt about colostrum supply or ability to suck.

The amount of colostrum required to prevent hypothermia is estimated to be 180–210 ml/kg body weight in 24 h, depending on weather conditions, housing, etc. An initial feed within 30 min of birth of 100–200 ml depending on size of lamb will usually ensure that lambs get on their feet, unless there is an underlying problem such as birth injury or congenital defect. If insufficient ewe colostrum is available, goat or cow colostrum may be substituted. Care should be taken that colostrum from only CAE tested goats is used for maedi-accredited flocks. If cow colostrum is used, the possibility of anaemia developing in some lambs should be noted. See Chapter 17.

Vaccination schedules for pregnant ewes to provide colostral antibodies for lambs need careful advance planning to ensure that individual farm disease situations are addressed. The following vaccines are available:

- Clostridial – should be used in every flock. Protects lambs for 12–16 weeks.
- *Pasteurella (Mannheimia)* – useful in flocks where young lambs contract pneumonia, but colostral antibodies only protect for 2–4 weeks.
- *E. coli* – protects young lambs against *E. coli* scour.
- Erysipelas – protects growing lambs against erysipelas polyarthritis.
- Colostrum from cows vaccinated against rotavirus has been used to protect lambs where this virus has been implicated in outbreaks of scour.

Hygiene of shepherds and in lambing area

Poor hygiene in lambing sheds and individual lambing pens is primarily responsible for outbreaks of infectious disease such as navel ill, joint ill and infectious scours, and will contribute to watery mouth problems. Although bedding can be expensive, it is false economy to allow dirty, wet conditions to

develop where ewes are lambing. Individual lambing pens should be cleaned out regularly. Disinfection of lambs' navels soon after birth should be routine.

Bonding of ewes and lambs and avoidance of mismothering

This is crucial to the survival of lambs, particularly where ewes have two or more lambs. Problems often begin in overcrowded lambing pens, so correct stocking density and close supervision, with removal of family groups to individual pens, is important. Identifying family groups by numbering or other marking scheme will help in sorting out problems which occur after ewes and lambs are grouped in larger pens or turned out.

Provision of shelter

Shelter is crucial for outdoor lambing flocks and may consist of nothing more complicated than good hedges or walls around lambing fields. In the absence of these, shelter for lambs can be provided by strategic placing of straw bales, plastic mesh or other windbreaks. In indoor lambing units, care needs to be taken that individual lambing pens are not placed in draughty areas.

> Mass administration of antibiotics at lambing time – in some flocks administration of antibiotics is seen as routine to prevent problems such as watery mouth and joint ill. It cannot be a good principle to use antibiotics on a flock basis, unless as a last resort when all contributory management factors have been corrected. Particular attention should be paid to early colostrum intake and lambing area hygiene.

Treatment of sick lambs

Treatment of sick lambs is time consuming and often unrewarding at a time when staff are under intense pressure. Steps must be taken to diagnose the problem and institute preventive measures as soon as possible. Where an infectious cause exists, selection of an appropriate antibiotic may depend on the result of laboratory tests.

Treatment of hypothermia (see Figure 8.2)

Well recognised guidelines exist, and training should be given to shepherds on farms where lambs are at risk of hypothermia, especially those where lambing is outdoors.

Treatment of watery mouth

Only cases observed early will respond to treatment. Once the lamb has collapsed it is highly likely to die as a result of endotoxaemia. Where treatment is attempted, oral and parenteral antibiotics and a small dose of NSAID are

indicated. Adminstration, little and often, by stomach tube of an oral rehydration product (a calf product with glucose added at the rate of 10 g/100 ml) is advisable. Constipation may be relieved by administering an enema of warm soapy water (15–20 ml via an old stomach tube). Lambs should be kept warm and milk can be introduced once abdominal distension is reduced and a suck reflex is present.

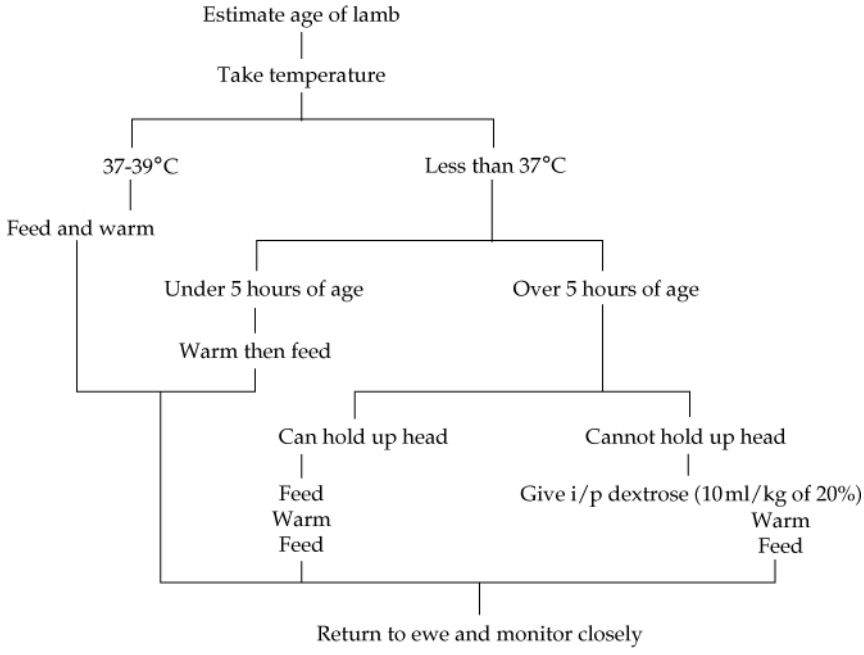


Figure 8.2 Treatment of hypothermia.

9 Inadequate growth rate

Inadequate growth rate is an extremely common complaint by sheep keepers and yet is the most difficult for which to give a quick and easy solution. Rarely, if ever, is the cause simple or indeed single. Nothing other than optimum growth rates will satisfy the owner, but by the time it is apparent that the flock is falling short of this aim, pathological change, if present, may have progressed to a point which will require time to correct. The end result may be conflict and dissatisfaction to all, including the bank manager!

The most commonly implicated factors in poor growth rate are faulty nutrition, poor parasite control and micronutrient deficiency. These are so important (not only in this area) that information is given separately in Appendices 3 and 4.

But what is the optimum growth rate? The clinician must be able to arrive at an independent decision as to the presence or absence of depressed growth rates. To do this, one must be very familiar with normal growth rates for breed, age, management system, season and nutritional inputs, as well as the probable effects on growth of any disease that may be involved.

Estimation of optimum growth rate

The following factors will be relevant.

Birth weights

- Are these adequate for the breed or cross? (Can be estimated as the mean for the parent breeds plus any heterosis effect.)
- Presence of disease affecting birth weight and early growth, e.g. copper deficiency, border disease or other forms of abortion.
- Placental competition in prolific ewes.

Food conversion rates

- 1:1 (on dry matter basis) may be achieved during early milk nutrition.
- With the change to grass-based nutrition, it then falls rapidly and may be as low as 10:1 on very poor quality forages.
- With the newer 'complete' feeds for lamb finishing it may be as good as 3:1.

Target weights

- Prime finished lamb – 18 kg carcass at 20 weeks. (Estimated dead carcass weight is usually 1–2 kg less than half live weight, but early fast growing lambs can kill out at 50% or even higher.) To achieve this with a birth weight of 4–5 kg, a minimum growth rate of 1.5 kg per week is required. Fast growing single lambs can achieve double this.
- Breed replacements should achieve 60% of mature weight at 36 weeks if they are to be mated as ewe lambs. If the mature weight is 75 kg, this requires an average weight gain of 1.4 kg per week.

Projected grass output

This will give the clinician great difficulty. Veterinarians in the UK are not trained in grassland management, but neither are many clients! In contrast to the position in New Zealand, where 'forage budgeting' is an accepted part of livestock production, the vital significance of grass of high nutritive value, correct grass length and quantity is often not appreciated in the UK. If in doubt, the clinician should not hesitate to ask for assistance from outside expertise. If the client is able to offer the necessary information, it may be possible to assess output against a true target as follows:

Total live weight gain/hectare = (total forage output × DM × utilisation × digestibility × conversion rate × stocking rate)

Growth rate is a function of the availability, absorption and utilisation of inputs over and above those required for basal metabolic rate under prevailing environmental conditions. It follows that suboptimal growth rates are a result of one or more of the following:

- Inadequate inputs (quantity and/or quality).
- Reduced appetite (parasites).
- Accelerated throughput (scouring).
- Protein loss into intestine or urine (disease).
- Excessive demands (raised basal metabolic rate in adverse weather conditions).

Frequently there will be a combination of several of these factors and it is this potentially complex equation which makes the diagnosis and treatment of the causes of inadequate growth rate or ill thrift so difficult.

In the development of the growing lamb, there are two distinct divisions in the nature of food inputs, and therefore two phases of potential reductions in growth rate. These are the 'milk dominated' and 'grass dominated' phases. In addition, modern husbandry methods include complete feeding of housed lambs. Table 9.1 lists the causes of inadequate growth rates in lambs and the tests possible.

Table 9.1 Inadequate growth rates.

Problem	Possible cause	Laboratory and other tests
Poor birth weight	Maternal under-nutrition	Condition score ewes Blood (serum) for metabolic profile/BHB
	Abortion agents	See Chapter 3
Poor early growth	Maternal under-nutrition – poor milk supply	Condition score ewes Check feed quality
	Udder and teat lesions	Clinical examination Electron microscopy for orf virus
	Neonatal disease	Postmortem material See Chapter 8
	Coccidiosis	Faeces samples – oocyst counts Postmortem material – intestinal smears
	Parasitic gastroenteritis	Faeces samples – worm egg counts
	Border disease	Blood (whole and serum) for virus isolation and serology
Growing lambs at grass (ill thrift)	Nutrition, grazing management	Herbage height Herbage quality
	Parasites	Faeces samples for oocysts and nematode eggs Blood (serum) for plasma pepsinogen
	Micronutrient deficiency	Blood (serum) for cobalt/B_{12} Blood (plasma) for copper Liver samples for copper and cobalt
	Other chronic diseases	Postmortem material Blood (serum) for border disease and erysipelas Blood (whole) for BD virus isolation Blood (serum) for kidney function (nephrosis)
Growing lambs housed	Nutrition	Food analysis
	Chronic disease	Postmortem material

Milk dominated feed period

Inadequate birth weights

This subject is discussed under suboptimal reproductive performance (Chapter 2) and perinatal mortality (Chapter 8), but is also of significance in this present area of consideration.

Low birth weight lambs may arise as a result of:

- Prematurity due to noninfective conditions.
- Prematurity as a result of infectious agents such as *Toxoplasma*, *Chlamydophila*, etc. causing subcritical placentitis.
- Restricted placental development in large litters.
- Early fetal loss.
- Inadequate maternal nutrition.

Whatever the cause, low birth weight lambs often have a poor sucking drive, or are unable to compete with stronger lambs for available milk. They are thus disadvantaged both from total milk intake and reduced immunoglobulin intake. It is a common observation that these lambs take a noticeably longer time to reach adequate weights than normal contemporaries.

Individual or sporadic low birth weight lambs

Check

- Possibility of early fetal loss. This results in poor placentation for the surviving embryos, since the caruncles which would have been utilised by the dead embryos do not become available to the survivors. This is commonly the explanation for the birth of lambs of widely differing birth weights to the same ewe. Examination of the placenta may show few cotyledons (the normal number is about 40 per horn) or abnormally small cotyledons.
- Health and condition of ewe – underfeeding or debility give poor lamb weights.

Many low birth weight lambs

Check

- If flock is highly prolific – limited placentation available for each fetus.
- For inadequate ewe nutrition in early pregnancy causing poor placentation (difficult to establish at this stage, but size of cotyledons may give guide).
- For inadequate ewe nutrition in late pregnancy – overestimate of winter grass quality, failure to feed concentrates, poor concentrates.
- For abortion agent operating in flock – see Chapter 3.

Inadequate growth during milk dominated nutrition

At this stage, poor growth is usually a reflection of inadequate milk supply, although the presence of neonatal disease within the flock will obviously also have an effect.

Individual lambs showing poor growth

Check for

- Milk supply – mastitis, teat lesions, competition if one of multiple.
- Mismothering or rejection (may follow rounding up for dosing, etc.).
- Ability to suck – mouth lesions (severe orf affecting the gums may not be noticed unless inside of mouth is examined).
- Disease – neonatal septicaemia, joint ill.
- Nephrosis – blood sample for urea and creatinine estimation. Lambs are dull, anorexic and afebrile with increased thirst for water. The cause is not known. PME usually shows enlarged pale kidneys, although histology may be required to confirm some cases.
- Possibility of 'wool' or 'milk ball' in abomasum – may be detectable on palpation.

> Poor growth plus green 'grass' type liquid faeces in the first 4 weeks of life indicate an inadequate milk supply. It may be difficult for the client to accept this.

Many lambs showing poor growth rates

During the early part of the 'milk' nutrition phase, this is almost invariably due to poor milk supply following inadequate nutrition of the ewe flock during both late pregnancy and early lactation. If optimal peak lactation is not reached very soon after parturition, it will never be attained. Since optimum growth rate is initiated at this critical time (with very high conversion rates if the supply is adequate), this must be the first area of investigation. Often client resistance will be evident, since there is inevitably some reflection on management. The other common cause of poor performance in the 2–8 week stage is coccidiosis. Although scouring would normally be expected to be a major feature of heavy infections, some lambs may simply show ill thrift with poor open wool.

Where a significant number of lambs are affected, it is always worth carrying out PMEs on a small number of typical cases to assist in reaching an accurate diagnosis.

Check for

- Ewe condition and nutrition – before and after lambing.
- Litter size of affected lambs. If prolific flock, may be poor management of triplets.
- Prolonged wet or cold weather with high chill factor.
- Coccidiosis – oocyst count is not reliable (see Chapter 10).
- Copper deficiency – subclinical deficiency may exist without evidence of 'swayback' lambs. May lead to poor viability of lambs, with poor sucking

instincts and higher susceptibility to disease. Take blood samples (minimum six animals) and liver samples for copper estimation.
- Border disease – characteristic hairy shakers are diagnostic, but may not always be present. Take blood samples for virus isolation.
- Orf infection of lambs' lips and ewes' teats.
- Evidence of other disease restricting mobility or after-effects of neonatal disease.
- Nephrosis as above.

Transitional milk to grass period

Frequently this period (from 4–5 weeks of age) will coincide with imposed limitations of forage supply, much of the available area being closed for forage conservation. Several factors follow from this.

The owner may underestimate the demand by the lamb crop for grass, planning stocking rates for adults only. By the time it is apparent that forage is short, the stock are several days overdue for a move. This both delays the onset of full grazing by the lamb crop and produces a degree of malnutrition, hence reduced growth.

The same limitation of grazing will require the lambs to take in grass contaminated with both coccidial oocysts and, unless clean grazing is available, infective nematode larvae. These last two factors are considered in Chapter 10 as important causes of scouring, but also have major effects on growth rates.

Even where adequate grass appears to be available, large mobs of sheep may cause poaching or spoilage, particularly in wet weather, drastically reducing palatability and utilisation which can drop to below 50% in bad conditions.

Check for

- Stocking rate and grass length (4–6 cm is optimal).

> Where set stocking is used, grass availability must be carefully monitored, with buffer areas available for grazing or conservation according to growing conditions.

- Provision of creep feed if grazing is inadequate.
- Parasitic disease – faeces samples for oocysts and nematode eggs (Chapter 10).

> Where mixed infections of Coccidia and *Nematodirus* occur, losses can be significant with a major effect on growth rate of survivors.

Grass nutrition period to weaning

In contrast to milk, which is a more or less standard product as far as nutrient content is concerned, the nutrient value of grazing varies widely. This ranges from the short dense rapidly growing specific 'sheep' mixture of the intensive lowland unit, to the low nutrient value of the high hill. It is important that the practitioner recognises this variability in quality as well as quantity, and the effect on both intake and growth rates. The effects of parasitism, particularly parasitic gastroenteritis and nematodirosis, are again major factors if clean or safe grazing is not available. In addition the effects of any micronutrient deficiency begin to be seen, although they assume a greater significance after weaning.

Check

- Quality and quantity of grazing before searching for more exotic causes.
- Dry matter content. If low, this may directly limit nutrient availability for optimum growth and produce nonparasitic scour.

If no clean or safe pastures have been available for the ewe and lamb post lambing:

Check

- Faeces samples for coccidial oocyst and nematode egg count. See remarks above re mixed coccidial and *Nematodirus* infection. The usual upper age limits for these infections are about 8 weeks for coccidiosis and 12 weeks for *Nematodirus*.

Post-weaning growth retardation (ill thrift)

Although the occasional individual case will be due to chronic infection contracted at an earlier stage, the vast majority of incidents will affect significant numbers of lambs within a group or flock. These cases pose perhaps some of the most difficult diagnostic problems in sheep clinical work, since the causes are almost invariably multifactorial. This is often combined with client resistance to the more obvious solutions. The cry 'But I have been worming them every three weeks' often echoes through the sheep clinician's dreams! The only solution for a successful diagnosis is that the clinician works methodically through the possibilities.

The main causes of poor performance at this stage of the production cycle are:

- Nutritional.
- Acute gastrointestinal parasite damage.
- Chronic after effects of parasitism.
- Micronutrient deficiency.
- Chronic disease, e.g. erysipelas, border disease, pasteurellosis.

Some of these will be accompanied by other signs which help to narrow the field, e.g. scouring (see Chapter 10), lameness (Chapter 14).

Check for nutritional causes

There may be an inadequate supply of forage if the season has been difficult for conservation. Check the amount and type of pasture available and grazing history since weaning. In late autumn this age group of lambs is often grazed on cattle pasture, which may be of poor palatability. The value of autumn and winter grazing is often overestimated. Check also for slurry or manure spreading which greatly reduces palatability.

> Pasture spread with pig slurry is dangerous because of the high copper content and may induce copper poisoning.

Check for parasitic causes

In this age group, chronic gastrointestinal parasitism is the most common cause of failure to thrive. Although anthelmintics may be administered regularly, re-infection occurs immediately if contaminated pasture is being grazed, and repeated damage is caused to the intestinal mucosa by the developing larvae.

> Negative results for nematode eggs in faeces samples from such animals do not exclude the possibility of chronic worm damage.

- If samples are negative, check for recent worming (prepatent period is about 3 weeks, so animals wormed within that time will show negative counts). Blood samples for pepsinogen and albumin content may also assist in indicating chronic gastric damage.

> Poor growth, linked to chronic parasitism, has been shown to be due to depression of appetite, with reduced food intake as well as increased nitrogen loss into intestine and urine.

- If poor growth rates are experienced with clean or safe grazing systems, check faeces samples and past grazing history for evidence of inadvertent contamination.

> Mixed grazing or alternate grazing with cattle and sheep is now known to allow the possibility of breakdowns, especially with *Nematodirus*, as calves are able to act as hosts.

Check for micronutrient deficiencies (see also Appendix 3)

Deficiencies of cobalt (pine) and copper are both implicated as causes of ill thrift in this age group. Local knowledge of geology and disease patterns should assist the clinician in deciding the possible involvement of these trace elements, but even so the diagnosis is by no means straightforward.

Apparently healthy, thriving animals may be found to have marginal or low blood values, and unthrifty animals may have apparently normal values. In the end, the clinician may have to rely on response to treatment as the final diagnostic feature (if the temptation to treat with everything 'in case' is resisted).

Cobalt

Cobalt deficiency produces poor growth, weakness and emaciation. Lambs are anaemic, become anorexic and often show a watery discharge from the eyes. Cobalt is an essential component of vitamin B_{12} which is manufactured by the rumen microflora.

Check

- Serum vitamin B_{12} (clotted blood sample) – <0.15 pmol/ml indicates deficiency.
- Liver B_{12} if samples available – <0.09 μmol/kg wet weight indicates deficiency.
- Urine sample for formiminoglutamic acid (FIGLU) and methylmalonic acid (MMA) – these are abnormal metabolites of propionic acid which accumulate in Co deficiency.
- Herbage and soil cobalt content – <1.9 μmol/kg in diet and <5 μmol/kg soil indicate deficiency.

Test dose a group of affected animals and monitor weight gain against untreated group.

Copper

Copper deficiency produces effects on the wool (loss of crimp – steely wool), depressed growth and anaemia. It is usually only seen where hill grazing has been improved by draining and liming. In this situation the copper deficiency is induced by increased availability of minerals in the pasture – molybdenum, iron and sulphur, which are ingested in soil taken in with the herbage.

Check

- Plasma copper concentration (heparinised sample) – <9.5 μmol/l indicates deficiency, but is less accurate than liver concentration.
- Liver copper concentration – this gives a better indication of true copper status than plasma – <160 μmol/kg DM indicates deficiency.

- Blood sample for superoxide dismutase (SOD) concentration – low value indicates prolonged deficiency (normal value in lambs is 0.4–0.5 i.u./mg Hb).
- Herbage and soil analyses for copper, molybdenum and sulphur content – although these can be difficult to interpret if pasture improvement (which increases Mo and S availability and decreases Cu availability) has taken place. Specialist help may be required in interpretation.

Selenium

Selenium deficiency causes ill thrift in lambs in New Zealand and Australia, but has not yet been implicated in the UK.

Other chronic diseases

Check for

- Border disease (blood sample for virus isolation). This may affect flocks without the appearance of characteristic hairy shaker lambs. Affected lambs are stunted and often show diarrhoea (see colour Figure 5).
- Nephrosis – as previously stated, the cause is not established. In some cases it may be associated with *Nematodirus* infection, and dehydration after severe scouring, but this is not always the case (blood sample for urea and creatinine concentrations).
- Chronic erysipelas (serology).
- Chronic pasteurellosis.
- Subclinical liver fluke if lambs are retained late in the season.
- Heavy infection of sarcocysts – these coccidia are spread by dogs and have occasionally been identified as a cause of ill thrift.

Treatment of inadequate growth rate

This will depend on identifying the most important cause(s). It will rarely be possible to initiate change quickly enough during the current season to have great influence in the short term, and affected lambs will probably never reach their full potential. The combination of shortage of grazing and chronic gut damage with possible micronutrient deficiency cannot be easily overcome.

Prevention of problems during the grazing period

The greatest challenge will be to convince the client that, after weaning, the lamb crop must have priority over any other sheep. This must be planned for by both the production of sufficient grass at a period when there will be the competing demands of forage conservation, and an integrated parasite control programme.

For further information on nutrition, parasite control and micronutrient deficiencies see Appendices 3 and 4.

Finishing lambs on 'complete' diets

This now common method of finishing lambs is highly cost effective and, under good management, gives the opportunity to monitor accurately growth and food conversion rates. Suboptimal growth rates should therefore be apparent at a much earlier stage than under less carefully controlled grass-based systems.

Check for

- Analysis of diet and cost. Low cost almost certainly means poor quality.
- Fibre and ash content – high values mean poorer quality (note, though, that diets including processed straw will be higher in fibre but still acceptable). If in doubt check with manufacturer for ingredients and expected growth rates.
- Feeding routine – freshness of supply. Soiled or stale food will not be eaten.
- Change in batch – different palatability.
- Trough space allowed.
- Uniformity of age groups, size of animals.
- Ventilation in building – heat stress or excessive humidity.
- Bedding quantity and dryness – possible ammonia build-up.
- Water supply – sheep are particularly susceptible to rejection of water supply if soiled.
- Too rapid introduction of feed, chronic acidosis.
- Concurrent disease especially atypical pneumonia.

All losses should be checked by postmortem examination for signs of chronic lung disease, or other feed-associated problems such as urolithiasis.

10 Diarrhoea

Normal faeces in the sheep can vary from hard pellets to a paste-like consistency, mainly as a result of variations in the water content of the diet, but there are also some individual and breed variations. Liquid faeces are a reflection of increased fluid passage from the lower intestine and are not in themselves proof of pathological change or overt disease. Since the sheep requires some 2.5% of body weight in dry matter intake daily for maintenance, fluid intake on a low dry matter diet may be beyond the capacity of the gastrointestinal tract to reabsorb.

Diarrhoea, therefore, may be the end result of:

- Increased fluid intake (either because of increased thirst or low dry matter intake).
- Reduced reabsorption.
- Fluid loss into the intestine.

Increased thirst may result from normal physiological processes such as pregnancy or lactation. It also results from the attempt to maintain tissue fluid normality after electrolyte loss, or may follow increased electrolyte intake, e.g. in feed blocks. Low dry matter intake is very common, especially in the autumn grass flush, but can also result from feeding poor quality wet silage or excessive amounts of roots.

A combination of reduced reabsorption from the lumen of the intestine, and increased fluid loss through damaged mucosa into the intestine, is found in many forms of both gastroenteritis and inflammatory change in the lower intestine. These range from bacterial infections such as *E. coli* and *Salmonella* to parasitic diseases such as coccidiosis and parasitic gastroenteritis.

Except in the newborn, scouring itself does not comprise a threat to life if fluid and electrolyte loss are compensated for by increased intake, and provided that there is no major damage to intestinal mucosa. It is necessarily much more serious in the neonate, with its poor thermoregulatory abilities and lack of large fluid reserves in tissues and rumen. The adult, on the other hand, having the advantage in all these factors, can and does compensate, frequently suffering only marginal reductions in production.

The many causes of scouring are commonly both age and diet related (see Table 10.1); thus in the investigation of any particular incident, many conditions can be excluded on these grounds alone. Aids to the diagnosis of scouring are given in Table 10.2.

Table 10.1 Scouring – common causes.

Age group	Type	Cause	Clinical features
Neonatal	Digestive	Feed management	Generally not ill
	Infective	*E. coli* *Salmonella* Rotavirus Cryptosporidia	Generally ill, rapid dehydration
		Clostridia	Severe illness or sudden death
Growing lambs	Dietary	Milk supply failure	Premature grass faeces Failure to thrive
		Low dry matter grass	Persistant scour
		High nitrogen in grass	Less effect on growth rate
		Overeating	Characteristic faeces, ill, rumen stasis
	Infective	*Salmonella*	Ill, associated with abortion in ewes
		Border disease	Not ill but ill thrift, persistent scour
	Parasitic	Coccidiosis Nematodirosis	Severe haemorrhagic scour, deaths, ill thrift in survivors
		Parasitic gastroenteritis	Ill thrift, deaths if neglected
	Toxic	Nitrates	Methaemoglobinaemia
Adults	Dietary	Overeating	Characteristic faeces, ill, rumen stasis
		Low dry matter intake	Not ill, little effect on body weight
		Forage spoilage	Reluctance to eat
	Parasitic	*Teladorsagia* type II *Trichostrongylus*	Young ewes or rams, loss of weight, anaemia
	Genetic	Breed association	Suffolks
	Infective	*Salmonella*	Ill, abortions in flock
		Johne's	Sporadic cases, chronic weight loss

Lambs from birth to 4 weeks

Scouring in the neonate can be subdivided into infectious and digestive causes for the purpose of diagnosis. The two groups may be distinguished by the severity of the general signs shown, rather than the type or appearance of the faeces. In addition, digestive scour may affect individual animals or a small group, whereas an infectious agent will usually affect many or all lambs. Infectious forms are more commonly encountered towards the end of the

Table 10.2 Scouring – aids to diagnosis.

	Gross pathology	Microbiology	Parasitology	Haematology/ biochemistry
Neonatal enteritis	Enteritis, septicaemia	*E. coli* *Salmonella* Clostridia Rotavirus	Cryptosporidia	IgG status $ZnSO_4$ turbidity
Coccidiosis	Lower small intestine and colon – dysentery		Faecal oocysts Intestinal smear Species differentiation	PCV
Parasitic gastroenteritis	Abomasum *Teladorsagia*, *Haemonchus*, *Trichostrongylus* Small intestine *Trichostrongylus*, *Nematodirus* (dysentery)		Faecal egg counts Worm counts Mucosal scrape	PCV Plasma pepsinogen
Nitrate poisoning	Brown blood			Methaemoglobinaemia
Acidosis	Rumen contents, pH			
Johne's disease	Thickened lower small intestine, yellow pigment sometimes	Faecal smear (repeated sampling) Intestinal smear Histology		AGID/CFT

lambing period, particularly in housed flocks, as a result of a build-up of infection.

The organisms in Table 10.3 are those most commonly responsible for enteritis in the neonatal lamb.

Differentiation of the specific causal agent in any individual outbreak will usually be difficult on clinical grounds alone. Although the field can be narrowed to some degree, the clinician must be prepared to obtain laboratory confirmation of the presence of any particular agent. Some outbreaks will involve significant lamb mortality, and full use should be made of any available postmortem material. See Chapter 8 for further details.

The following points will be a guide to the possible cause of any particular incident, in association with laboratory findings. The possibility of mixed infections should also be borne in mind, and if laboratory tests or response to therapy indicate a complex aetiology, appropriate action should be taken.

Table 10.3 Infectious causes of enteritis in young lambs.

Type of organism	Species	Specific disease
Bacteria	*E. coli*	Colibacillosis
	Salmonella spp.	Salmonellosis
	Cl. perfringens type B	Lamb dysentery
	Campylobacter	
Viruses	Rotavirus	
	(Enterovirus, reovirus, adenovirus, astrovirus)	Significance not known
Protozoa	*Cryptosporidium parvum*	Cryptosporidiosis

Lambs not ill, appetite maintained, dehydration not severe

Check for

- Digestive scour – the faeces often have a pasty white appearance and the problem is usually associated with artificial feeding or supplementation of lambs from ewes with an inadequate milk supply. Lambs do not exhibit the rapid and severe depression which usually accompanies infectious forms of scour, and provided the lamb continues to suck, the end result may not be serious.
- Excess milk intake, e.g. loss of a twin.
- Management of artificially reared lambs – type of milk substitute, correct mixing, feeding temperature, amount per feed, frequency of feeding.
- Hygiene and cleaning of feeders.

> Lambs can be reared very satisfactorily (although expensively), on ad libitum artificial feeding systems. These may be based on either warm or cold milk. Fluctuations in the temperature of the milk should be avoided since this may cause digestive problems. Female lambs on ad libitum feeding occasionally exhibit urine scalding, which superficially may give the appearance of scouring.

Lambs show profuse bloody scour, abdominal pain and rapid death

Check

- Vaccination history of ewes.
- For lamb dysentery. This often affects the strongest lambs. The postmortem picture is pathognomonic – severe enteritis affecting the ileum with necrotic mucosa, serous or bloodstained fluid in peritoneum. For confirmation submit intestinal contents (30 ml) to laboratory for direct smear, or ELISA.

Lambs show severe scour (often greenish colour), septicaemia and death; illness and abortions in ewes

Check for

- Salmonellosis – bacteriological examination of faeces, rectal swabs and carcasses (posterior mesenteric lymph nodes are particularly useful).

Lambs show profuse scour, weakness and dehydration

It is this group which gives the most difficulty in diagnosis. The lambs are very susceptible to dehydration and a specific diagnosis is essential for the selection of appropriate treatment; yet laboratory diagnosis must inevitably take some time. The nature of the faeces and the severity of the depression are of little assistance.

Check for

- Enterotoxigenic *E. coli* (ETEC) – indirect fluorescent antibody test (IFAT) on fresh sample of small intestine, culture, bacterial counts ($>10^8$/g of intestinal contents), identification of K99 antigen. Although cases of watery mouth may occur in a flock affected by ETEC, scouring is not a common feature of that condition.
- Salmonellosis as above.
- Rotavirus – faeces sample for electron microscopy, ELISA, IFAT.
- *Campylobacter* – not usually thought to be a significant problem in lambs.

Lambs ill from 4 days approximately, afebrile, anorectic

Check for

- *Cryptosporidium* – faecal smears stained with Giemsa for detection of oocysts (these are much smaller than coccidial oocysts), histology of small and large intestines. This disease is self-limiting, but requires time for damaged villi to regenerate. There is no specific treatment.

> Although *Cryptosporidium* is a protozoon, the short life cycle (2–4 days) gives the appearance of a bacterial infection. The parasite is not host specific, therefore it can be contracted from other species, commonly calves. It is also a zoonosis.

Treatment of scour in young lambs

This will in part depend on diagnosis, although fluid therapy should form the first line of treatment, particularly if the lambs are dehydrated or distressed.

Calf rehydration products can be used but require extra glucose (10 g/100 ml) to supply the energy needs. There is continuing debate in both human and animal health on the true place of antibiotic therapy in the treatment of diarrhoea. Clearly, where there is little evidence of an infectious cause, use is contraindicated on grounds of both cost and possible future development of antibiotic resistance. Where a bacterial cause is confirmed, drug sensitivity should be determined as this can often vary from farm to farm, or season to season. Pressure from a client for a particular product because 'it worked last time' or 'it works for a neighbour' should be resisted. Where the cause is viral or protozoal, antibiotics will have no effect and fluid therapy and supportive nursing are most important.

Prevention

Prevention is first of all based on husbandry – clean bedding in lambing pens, isolation of suspect lambs and safe disposal of contaminated debris, thorough disinfection of hospital pens and disinfection of protective clothing between handling suspect and healthy lambs. The ewe vaccination programme should be reviewed if clostridial, *E.coli* or rotavirus infection have been diagnosed.

Growing lambs

With increasing age, the lamb is less susceptible to the acute dehydration of scouring, except in the case of severe damage such as that produced by acute nematodirosis and coccidiosis. When the full transfer to grass-based nutrition takes place, the fluid reserves of the rumen and large intestine become available to buffer the effects of scouring. However, in addition to the chronic intestinal damage persisting after neonatal infections, the lamb frequently has to adapt to very low dry matter forage intake, as well as the challenge of parasitic diseases such as coccidiosis and helminthosis. Chronic intestinal damage caused by parasites with erosion of villi leads to long-term malabsorption, protein leakage and chronic anaemia, giving severe diagnostic problems over the long term. Conflict with the client concerning aetiology and perceived failure to respond to treatment is common.

The following causes of scouring should be considered:

- Dietary
  Milk supply failure and premature dependence on grass
  Nitrogenous or other fertiliser ingestion
  Overfeeding/acidosis
- Bacterial
  Salmonella spp.
  Campylobacter
  Listeria
- Viral
  Border disease

- Parasitic
  Coccidiosis
  Nematodirosis
  Parasitic gastroenteritis
- Toxins (worth considering if other causes eliminated)
  Molybdenum
  Copper
  Lead
  Ragwort

At this stage, the incidence, i.e. individuals or whole groups affected, and any history of prolonged housing or heavy stocking on lambing paddocks, will be of significance.

Individuals or small numbers affected

Check

- Ewe for milk supply failure – teat lesions or mastitis. Such lambs have severely reduced growth rates, a 'potbellied' appearance, and a 'grass scour' before the age at which they should be totally grass dependent.
- For fibre-filled abomasum (palpate) as result of above.
- For weaning too early, if artificially reared, before rumen adequately developed.

Many lambs or whole groups affected

Although dietary factors may be implicated, parasitic disease is the most common cause.

Check for

- Coccidiosis.
- *Nematodirus battus* infection.
- Parasitic gastroenteritis.

These are discussed here in turn.

Coccidiosis

The diagnosis of coccidiosis is not a straightforward matter. Perfectly healthy lambs may show very high faecal oocyst counts (1×10^6/g), while severe pathological lesions may be present in lambs showing negligible or low counts.

This anomaly can be explained by two factors:

(1) Relatively nonpathogenic species of coccidia, e.g. *Eimeria bakuensis* may be involved. These infect the cells of the small intestine causing little damage to the crypt stem cells, and minor amounts of damage can be compensated for within the large intestine.

(2) Pathogenic species, *E. crandallis* and particularly *E. ovinoidalis*, which infect the lower small intestine, caecum and colon, may cause severe damage to crypt cells early in the life cycle, before there is any significant production of oocysts. In addition, the period of maximum oocyst production is short, so that samples taken late in an infection may also show low counts.

Confirmation of diagnosis rests upon demonstration of a combination of the following factors:

- Age of affected lambs 4–7 weeks.
- Lambs housed for extended period after birth, *or*
- Lambs and ewes kept tightly stocked after lambing.
- Severe diarrhoea often with blood.
- Some lambs show tenesmus.
- Some lambs with oocyst counts $>1 \times 10^5$/g.
- Species present are *E. crandallis* and/or *E. ovinoidalis* (differentiation of oocysts is a specialist laboratory task).
- Postmortem examination shows lesions in lower small intestine, caecum and colon – raised white spots, mucosa thickened and inflamed.
- Smears of intestinal scrapings show presence of developing stages of coccidia.

> Response to treatment with sulphonamides is not diagnostic, since these have a wide range of action against other infectious agents.

Treatment of coccidiosis

The greatest problem is whether the whole group or only those significantly affected should be treated. In a flock of mixed singles and multiples, the larger single lambs may be little affected clinically but the general approach should be to treat the whole group. Depending on the product, further treatments may be necessary for those severely affected. The main products are:

- Diclazuril – a single oral dose (rarely, severe scouring has followed the use of this product; should this occur fluid therapy is necessary for affected lambs – read data sheet).
- Sulphamethoxypyridazine – injected daily for 3 days.
- Decoquinate is commonly included in creep feed, but lambs may not eat sufficient, particularly if they are already ill.

Prevention

From a management point of view there is a finely balanced equation between lambs acquiring sufficient exposure to allow build-up of immunity and acquiring a pathogenic infection. Lambs kept indoors for several weeks are at greatest risk, particularly mixed age groups and later batches kept in uncleaned pens where earlier batches have been, because of a great build-up of oocysts in

the environment. The same can occur at grass where ewes and lambs are kept tightly stocked on contaminated pasture.

The strategies available to prevent disease are:

- Aiming for limited trickle exposure and build-up of natural immunity by avoiding grossly contaminated indoor or outdoor environments.
- Offering lambs creep feed medicated with decoquinate.
- Strategic drenching with diclazuril or injecting with sulphonamide before clinical disease occurs, usually 4–6 weeks of age, repeated 3 weeks later if necessary.
- Avoidance of overgrazing pasture.

> Feeding pregnant ewes with decoquinate to suppress oocyst output is contraindicated, as this reduces colostral immunity and may lead to increased susceptibility of lambs.

Nematodirus battus infection

This is caused by sudden mass hatching of infective larvae on pasture contaminated by young lambs grazing the pasture the previous year. Hatching follows a rise in temperature to above 10°C following a period of cold sensitisation. Forecasts of disease are based on weather conditions together with timing. Disease will occur if the mass hatching coincides with the presence of susceptible lambs on the pasture. If hatching occurs early in the season before the lambs are eating significant quantities of grass, or if hatching is very late when lambs are past the most susceptible age, disease is not likely. Disease due to *N. battus* is rare in lambs over 3 months of age, but *N. filicollis* can cause scouring and weight loss in hoggs in autumn.

Diagnosis is based on the following:

- Sudden onset of severe scouring often with dysentery.
- Grazing history – land grazed by lambs in previous year.
- Seasonal forecast of disease.
- Lambs are dull, dehydrated, sometimes show abdominal pain.
- Faeces sample may show characteristic *Nematodirus* eggs, but lambs may be ill before many are present as developing immature worms do severe damage; some deaths will occur.
- PME shows acute enteritis with worms present in the intestine (look like cottonwool on mucosa).

> Concurrent infection with coccidiosis may increase the effect of *Nematodirus* infestation

For treatment and prevention of nematodirosis see Appendix 4.

Parasitic gastroenteritis

This is still the major source of loss in growing lambs. The main species of helminths involved are *Teladorsagia* (*Ostertagia*) spp. and *Trichostrongylus* spp. *Cooperia, Strongyloides* and *Bunostomum* may be found in the small intestine but rarely in sufficient numbers to be pathogenic. Parasites of the large intestine (*Oesophagostomum, Chabertia* and *Trichuris*) are rarely present in sufficient numbers to be pathogenic.

> *Haemonchus* infection usually causes severe anaemia, oedema and weakness. It is not usually associated with scouring.

> All the above worm species cannot be distinguished in a routine worm egg count, since the eggs are virtually identical (although computer-aided identification may be a technique available in the future). Differentiation requires larval culture, or examination of eggs by a very experienced parasitologist.

Diagnosis is based on the following criteria:

- Scouring and weight loss from July onwards.
- Grazing history – contaminated pasture.
- Anthelmintic treatment programme – inadequate or infrequent dosing early in the season when on contaminated grazing, allows a build-up of infective larvae by early summer.
- Egg counts of undosed lambs (may be >1000 eggs/g).
- Raised plasma pepsinogen concentration (>1 i.u./litre).
- *Teladorsagia* causes characteristic abomasal lesions (raised nodules in thickened mucosa).
- *Trichostrongylus* causes enteritis, hypertrophy of mucosa with flattened areas in long-standing cases.
- Presence of large numbers of worms at PME – 10 000–15 000 *Teladorsagia*, 20 000–30 000 *Trichostrongylus* (*Teladorsagia* are easily visible in abomasum, but hair-like *Trichostrongylus* in small intestine can easily be overlooked.)

For treatment and prevention of PGE see Appendix 4.

> All the above conditions are involved in the ill thrift problem – see Chapter 9.

Problems in diagnosis

Lambs may continue to scour after anthelmintic or anticoccidial treatment because of chronic damage to the intestine. The absence of parasites does not

necessarily mean that they were not the original cause of the damage. Histological examination of the intestine usually shows erosion of villi, leading to malabsorption and protein leakage. It is often difficult to convince the client that parasitism has been involved when they say 'but I have wormed them regularly' and faeces examinations are negative (often taken after a recent treatment with anthelmintic, anyway).

Resistance to anthelmintics has now been clearly identified in the UK and is a significant factor in lack of response to drenching. It is largely a factor with the benzimidazole group of drugs but has also been identified with levamisole and ivermectin. In view of the catastrophic results on the sheep industry in South Africa and Australasia, it is vital that resistance is identified and eliminated at an early stage. Laboratory tests are available based on egg reduction or larval hatching rates in faeces samples taken 7–10 days after dosing with the drug under suspicion. Multiple resistance to all three groups has recently been reported.

Breakdowns with 'clean' or 'safe' grazing systems (see also Appendix 4)

The introduction of safe grazing systems has been a major advance in parasite control where the farming system allows, but breakdowns frequently occur. Successful operation depends on a clear knowledge of worm life cycles, effect of age, sex and pregnancy upon worm burdens, care with timing and accuracy of drenching, and careful recording of grazing management.

Check for

- Transmission of *Nematodirus* via susceptible calves.
- Correct timing of anthelmintic treatments, i.e. immediately before turning on to clean pasture (holding in pens for 12–24 hours after dosing).
- Correct dosage with anthelmintic (gun calibration).
- Area grazed by lambs in previous autumn.
- Transmission by unwormed groups, e.g. rams. These may carry a significant worm burden throughout the year, unlike adult ewes.

> Although segments of the tapeworm *Monezia expansa* are commonly seen in the faeces of growing lambs, this parasite is thought not to have any pathogenic effects other than very rarely causing physical obstruction of the intestine if present in large numbers.

If these parasitic conditions are eliminated as a cause of scouring the following conditions should be considered:

Check for

- Low dry matter forage intake, lush grass growth, excessive rainfall.
- Fertiliser application without rainfall.

- Feed change, irregular water supply, excessive intake of concentrate feed (acidosis) if housed.
- Border disease – congenital infection can lead to poor thriving lambs showing chronic scour. See ill thrift in Chapter 9.
- Chronic inflammatory bowel disease – this has been reported as a cause of failure to thrive and pasty faeces. The cause is obscure but may be immune-mediated. Histological examination of intestines is required for confirmation.

A nephrosis syndrome has been identified, with some affected lambs also showing black scour, rapid weight loss, fits, followed by death. Although the cause(s) are not yet known, one suggestion is a trickle-infection with *Nematodirus*.

Scouring in store lambs or young adults in late autumn/winter

Parasitic disease may again be a problem in this age group.

Check for

- *Teladorsagia* type II.
- Chronic *Trichostrongylus* infection.
- *N. filicollis* infection.
- Liver fluke infection

Adult sheep

The same underlying factors apply to the control of the consistency of faecal material in the adult, as apply to younger animals, i.e. both physiological and pathological change will produce variation, which may or may not be of significance. In addition to the physiological variation of dry matter content of forage, the ewe will have the extra demands of pregnancy and lactation. Pregnancy will require extra nutrient intake to cope with the 'production' demand of fetal growth. If this is combined with forage of varying dry matter, especially silage, and access to feed blocks with a high electrolyte (salt) content producing increased fluid intake, the result may well be continuously fluid faeces. Finally, the adult will be subject to greater risk of 'slow onset' conditions such as Johne's disease.

Sudden onset of scouring with systemic illness

Check for

- Salmonellosis.
- Concentrate overfeeding/acidosis.

Scouring with little significant weight loss

This may be 'normal' in some animals.

Check for

- Breed disposition – individuals of several breeds, notably the Suffolk and Wensleydale, may scour or have soft faeces for no apparent reason.
- Low dry matter intake – lush autumn grass, wet silage, roots, etc.
- Availability of thirst-inducing mineral or feed blocks.
- Interrupted water supply or frozen water supply.

Scouring with weight loss

This may not be easily apparent if the starting point is from low body weight at the end of the lactation period.

Many cases or whole groups affected

Check for

- *Trichostrongylus* infection.
- *Teladorsagia* type II.
- *Chabertia* may occasionally be implicated in scouring.
- High nitrogen fertilizer application.
- If fodder is available, conserved forage spoilage – mycotoxins.
- Signs of listeriosis – scouring may precede onset of abortions or nervous signs.

If individuals or small numbers affected

Check for

- Johne's disease – diarrhoea is not a constant feature of this disease in sheep, but soft faeces may occur sporadically. It is more commonly implicated as a cause of adult weight loss – see Chapter 12.
- In autumn, check for access to acorns – excess intake causes black tarry scour followed by constipation. This is often fatal.
- Chronic inflammatory bowel disease has been reported in animals up to 3 years old. See above.
- Other occasional causes such as tumours will be diagnosed at PME only.

Treatment and prevention

For further information on nutrition and parasite control see Appendices 3 and 4.

11 Tenesmus

This is a common sign in sheep (straining, striving, forcing), causing the sheep keeper to request assistance from the veterinarian. Unless the problem is obviously linked to parturition, the request is often in the form 'I have a sheep which is constipated'; this, almost invariably, is not the case. Thus, extra vigilance is needed, since to arrive at a considered diagnosis with which the owner will always disagree places the clinician in particular hazard.

The condition will usually be acute and will have welfare implications as the animal will be suffering discomfort or pain. It will call for a rapid solution since, if not already present, continual tenesmus will often lead to prolapse of the rectum or vagina. On the other hand, except in outbreaks of urolithiasis or coccidiosis, cases will usually be limited to a single animal or small numbers, so the economic significance may be limited.

The diagnostic procedure must always begin with the identification of the system involved (see Figure 11.1):

- Alimentary.
- Urinary.
- Genital.

This will frequently not be obvious, except in the case of prolapse or a vaginal discharge. If the animal is a female of reproductive age and generally, but not

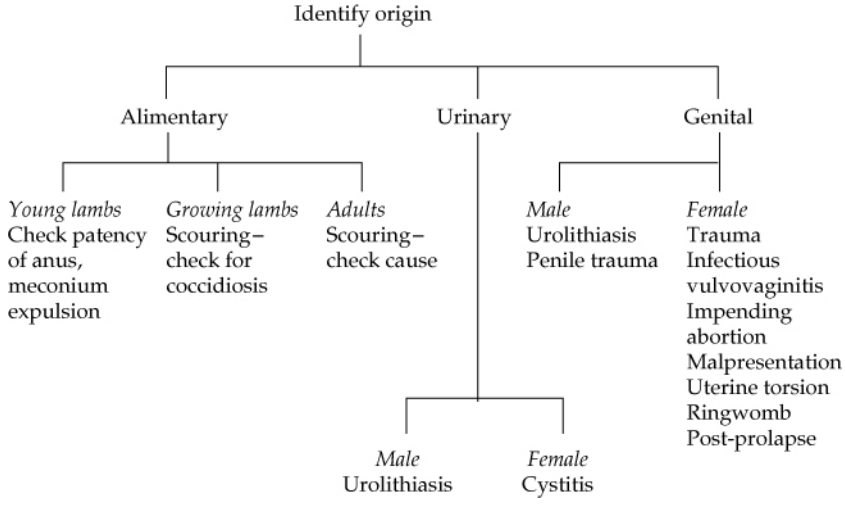


Figure 11.1 Tenesmus.

inevitably, at some active stage of the reproductive cycle, the identification of the tract involved can usually be established by digital examination of the vagina, use of a speculum, or insertion of a swab into the vagina. If no positive evidence emerges then a similar examination can be applied to the rectum.

As with much else in the diagnosis of disease in the sheep, the area for consideration can be narrowed by first establishing the sex and age group of the patient. This will eliminate many conditions.

Neonate

> Some lambs show tenesmus and vocalise with apparent pain when defecating normally during the first few days of life.

Check for

- Failure to expel meconium. This is more commonly a cause of loss of appetite, or a part of the 'watery mouth' syndrome.
- Tail adhesion due to dried meconium leading to a total seal of the anal ring.
- Congenital rectovaginal fistula. Usually only the vulva is patent.
- Imperforate anus. This usually causes abdominal distension rather than tenesmus.
- Placement of rubber ring if recently castrated.

Treatment

Treatment depends on the cause of the condition. Cleaning the perineal area of dried meconium is obvious, but should be done with due regard to minimising discomfort. An enema of warm, soapy water will relieve constipation. Imperforate anus may be operable if the blind end of the rectum is just below the skin. A bleb of local anaesthetic should be placed in the skin where the anus should be, then, after incision of the skin, careful dissection carried out to try to identify the closed rectum. After opening and evacuation of faecal contents, the rectal mucosa should be sutured at intervals to the skin. If the rectum cannot be identified, the lamb should be euthanased. (See also Appendix 8.)

Growing lamb (prepubertal)

Generally the cause of straining will be alimentary, and secondary to increased throughput of faecal material. This commonly results in irritation, inflammation and oedema of the rectal mucosa and anal sphincter, with persistent attempts to defaecate even with an empty rectum.

Check for

- Evidence of scouring, including faecal sample. Cause of scouring then to be investigated (Chapter 10).

- Coccidiosis. This is perhaps the most common cause, and will be one of the rare episodes when many animals will be affected.

If young male or castrated male and no evidence of scouring, particularly if on concentrate feed, then examination of urinary tract is essential

Check

- If recently castrated with bloodless castrator – incorrect technique may damage urethra.
- For urolithiasis. Examine prepuce for crystals adhering to preputial hairs or orifice. In young animals the penis cannot be extruded for examination. Palpate bladder for distension.
- For pain or discomfort when pressure is applied to pelvic inlet area.
- Patency of urethra by careful pressure on bladder.
- By ultrasound for bladder size and integrity.

> A check on ability to urinate can be made by placing the animal in a clean dry pen for a couple of hours, if this cannot be established by other methods. The extremely small diameter of the urethra makes catheterisation difficult or impossible.

See below for treatment and prevention of urethral calculi.

Adult female

Although cystitis can occur at any time of the year and will cause tenesmus, most episodes of straining will be associated with active stages of the reproductive cycle.

During the mating period

Check for

- Traumatic vaginitis due to reactivation of adhesions resulting from damage at previous lambing.
- Infectious vulvovaginitis (examine rest of group and rams).

During mid-pregnancy

Check for

- Impacted mummified fetus.
- Early abortion.

During pre-lambing and lambing period

Prolapse of the vagina and/or cervix will be an obvious cause of tenesmus and is dealt with in Chapter 4.

Check for

- Impending abortion.
- Malpresentation.
- Incomplete dilatation of cervix.
- Torsion of uterus.
- Peri-anal herniation.

After lambing

Check for

- Retained lamb.
- Bruising or vaginal tears after traumatic lambing, or shepherd interference.

> If no abnormality of the reproductive tract is detected, and the animal is not scouring, cystitis is a possibility, and a urine sample should be obtained for examination. This can be done by occluding the nostrils for a few seconds which stimulates urination, by catheterisation, or if all else fails, by introducing a strange ram to the ewe which will usually react by urinating.

Treatment

Treatment will depend on diagnosis and is usually self-evident. The technique of caudal epidural anaesthesia (using xylazine) will control straining and assist any manual or surgical intervention (see Appendix 8).

Adult male

Check for

- Urolithiasis (extrude penis and examine urethral appendage particularly).
- Penile trauma (urethral obstruction). Groups of rams may indulge in reproductive activity before joining, with mounting and insertion of the penis into the rectum of other members of the group. This can cause penile trauma and the possibility of rectal trauma.
- Posthitis.
- Traumatic occlusion of the prepuce.

Treatment of urolithiasis

See also Appendix 8 for further details. The most common site for obstruction is

the urethral appendage (see colour Figure 6). Attempts should be made to exteriorise the penis to examine the appendage, although this can be difficult in a young animal or if there is a large amount of swelling in the ventral abdomen because of urethral rupture and leakage of urine. If the appendage is blocked it should be removed by cutting with a clean pair of scissors. If this is not effective, a spasmolytic such as butylscopolamine (Buscopan) can be given intravenously. If still unsuccessful options are:

- Euthanasia if the animal is severely uraemic, if there is a lot of urine leakage into the ventral abdomen, or if treatment is not economically viable.
- Subischial urethrostomy or amputation of the penis and exteriorisation through the skin of the perineum. Both these techniques can be done under caudal epidural anaesthesia. They are salvage treatments only and of no use in valuable breeding rams.
- Percutaneous placement of a Foley catheter into the bladder to allow drainage of urine. This can be done using ultrasound guidance, or via laparotomy. Walpole's solution should be introduced into the bladder via the catheter twice daily and the catheter occluded for an hour or so to aid dissolution of calculi. If this technique is going to be successful, urination through the penis should be evident within 10–14 days. This is the only technique to offer hope of salvaging valuable breeding rams.

Prevention of urolithiasis

In all cases the feeding regimen should be examined. If urolithiasis occurs within a group, check suitability of feed. Concentrates with a high phosphorus content are now thought to be the cause, although until recently high magnesium content was blamed. Calcium:phosphorus ratio should lie within 2:1 to 1:2 boundaries. Some brands of ewe nuts specifically state that they should not be fed to lambs or rams.

In the case of pedigree rams, these are often show animals that are fed large amounts of concentrates for a prolonged period. Texels are particularly susceptible as they are efficient at absorbing various minerals (also susceptible to copper poisoning for the same reason). Concentrate diets should be carefully formulated, with sugarbeet pulp avoided. Owners should be advised to allow regular periods at grass with reduction or removal of concentrates if at all possible.

For all classes of stock, attention needs to be paid to provision of adequate clean water. Administration of urinary acidifiers such as ammonium chloride (5–10 g/day) orally will help to protect.

12 Adult weight loss

Adult weight loss or poor body condition has always been a major problem in sheep keeping. However, the pattern and causes have changed over the years. In earlier periods when the industry was characterised by low input, low output systems, concurrent disease was likely to be the major cause. More recently, increased demand on inadequate inputs has become recognised as the most frequent source of problems. Thus nutrition is a key feature of investigation of weight loss, and so a new section on the subject is included in this book (Appendix 3).

Imbalance between nutritional demand and the supply of nutrients may be brought about by:

- Overt undersupply.
- Inability to ingest an adequate supply.
- Disease processes causing malabsorption.
- Interference with metabolic processes.

The masking effect of the fleece remains a major factor, hindering detection of early weight loss in adult sheep. Frequently, by the time a problem is recognised, weight loss may have progressed to such a degree that correction is difficult. This occurs particularly and commonly during late pregnancy, when increasing nutritional demands and limited time available to deal with this are irreconcilable. The practice of winter shearing greatly facilitates control of body condition, where the management system allows this to be carried out.

A weight loss of 5% is recognised as indicating a problem in the human, if the loss takes place over a short time on normal inputs. This degree of monitoring is seldom available to the sheep keeper in the UK, as the variety of breeds and crosses have widely varying normal weight ranges. In countries where a more uniform sheep type predominates, regular weighing forms a vital management tool. This can be done by the use of individual 'indicator' groups.

The handling of individual sheep, and the application of the system of body condition scoring (see Appendix 3), allow a similar control of body condition to be maintained. It can be applied irrespective of breed, size or weight. It is important that the operator recognises that its value lies in repeatability of results for the individual rather than between operators. The value can be much enhanced if a correlation is established between body condition scores and body weight for an individual breed or type, thus allowing a value to be placed on any change, i.e. a loss of one body score means the ewe has lost X kg. In the case of common types of crossbred ewe such as the Mule or Welsh Halfbred, one unit of condition score is equivalent to approximately 5 kg.

In the investigation of any weight loss problem, the following aspects of the history are particularly relevant (see also Figure 12.1):

- Number affected – individual, many, whole group, all groups.
- Validity and extent of weight loss.
- Timescale of loss.
- Stage of cycle of nutritional demand.
- Recent management of nutrition.
- Any evidence of recent management changes, e.g. housing, change from hay to silage, etc.

Weight loss with apparent appetite increase

This is apparent by continual hunger, continual bleating, or escape attempts, usually with whole group or dominant members affected. This is almost invariably caused by feeding mismanagement.

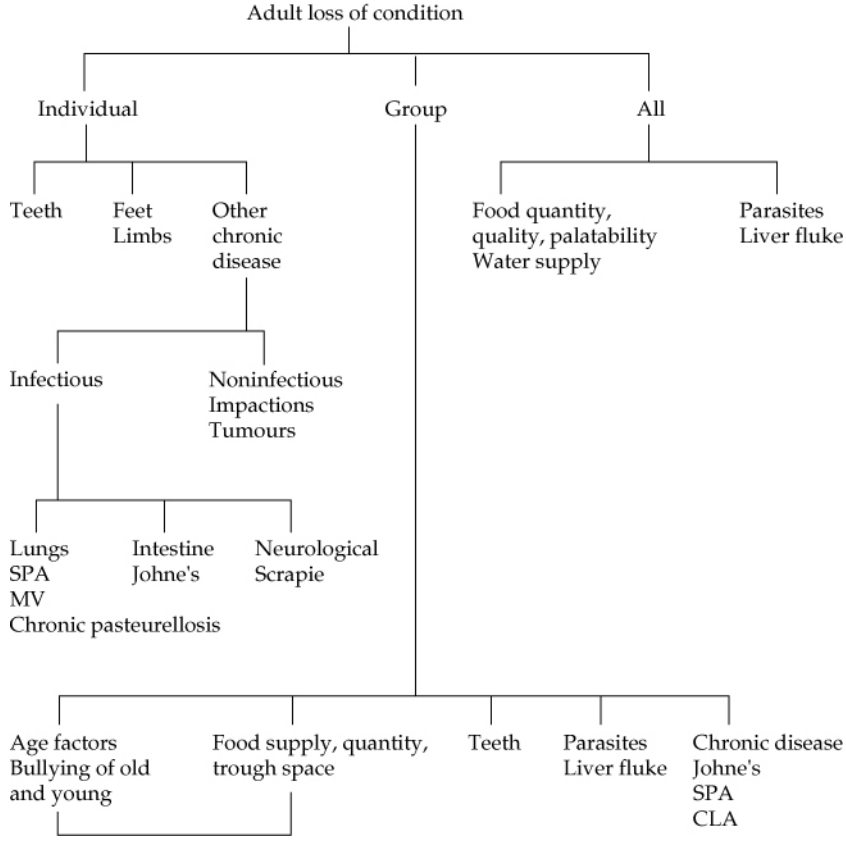


Figure 12.1 Adult loss of condition.

Check

- Quantity of forage. If at pasture, check grass length (4–6 cm is considered optimal). If at pasture in winter, check fodder supply (empty racks may mean quantity insufficient, full racks may mean palatability poor). If housed, check supply and actual consumption.
- Quality of forage. Late autumn grass or undergrazed pasture is poor quality; unimproved hill with rushes, bracken or heather will only support low stocking rate. If hay or silage is being fed, check quality, analysis if available, stage of maturity, how well conserved.

If in last 8 weeks of pregnancy check

- Concentrate feed – contact manufacturer for detailed analysis.
- Price if feed bought-in (cheap = nasty), ash content (>10% is poor).
- Formulation if feed home mixed, probable energy and protein content.
- Amount fed.
- Frequency of feeding.
- Trough space available.

> In the last 8 weeks of pregnancy, ewes which have been allowed to lose too much condition should be managed separately to allow extra feeding. This will produce better lamb birth weights, and possibly adequate colostrum, but it is impossible to put condition back on the ewes themselves.

Energy requirements of a 70 kg ewe rearing twins

In dry period	9.0 MJ/day
Flushing	15.0 MJ/day
Early pregnancy	15.0 MJ/day
Mid pregnancy	12.0 MJ/day
Late pregnancy	20.0 MJ/day
Early lactation	25.0–30.0 MJ/day
Mid lactation	15.0 MJ/day
Weaning	9.0 MJ/day

> If winter shorn, extra feed allowance is necessary – appetite will increase by at least 10% especially in cold weather.

Weight loss, appetite apparently normal, intake reduced

The most common cause of this pattern of weight loss is tooth problems. Incisor loss (broken mouth) will cause weight loss if grazing is short, but otherwise such sheep can remain in good condition, particularly if extra attention is given

at times of nutritional stress. Molar problems are a very common and often unrecognised cause of weight loss (see also Chapter 13 for more details). Shedding of temporary premolars is occasionally implicated, but most often premature loosening of the lower, first molar is the cause, and can occur as young as 2 years of age.

Check

- Incisor teeth.
- Molar teeth (palpate through cheeks, and check mandibles for swellings).
- For lameness – painful foot lesion, e.g. footrot or foot abscess can lead to rapid weight loss, particularly in late pregnancy.

Weight loss with normal appetite and normal intake, individual animals affected

If the remainder of the group is in good bodily condition, the most likely cause is concurrent, usually chronic, disease.

Check

- Molar teeth as above.
- For Johne's disease – examine faeces sample smear stained by Ziehl Neelsen (may need multiple samplings, and a negative result does not rule out the possibility of this disease.) May require PME and smear of ileal mucosa or mesenteric lymph nodes, or histology of intestine to confirm diagnosis. The pigmented form is easily recognisable, with bright yellow colouration of the mucosa of the ileum.
- Chronic inflammatory bowel disease (see Chapter 10).
- For pulmonary adenomatosis (wheelbarrow test for excess pulmonary fluid). No other diagnostic test apart from PME. See Chapter 21.
- Lungs for chronic pneumonia. Maedi-visna (MV) is an important cause of chronic lung lesions in other countries and clinical cases have now been seen in the UK. See Chapter 21 for details.
- For cud spilling – long-term sufferers often are poor thrivers (see Chapter 13).
- For early signs of scrapie – other neurological signs should be detectable, or an 'itch' reflex. Scrapie is notifiable.

Other individual causes of weight loss such as tumours, liver abscess may only be diagnosed at PME. A drop in albumin:globulin ratio may indicate a septic focus or Johne's disease.

Treatment

For most of these chronic diseases, treatment is not appropriate. Affected animals should be slaughtered as soon as possible, particularly if an infectious disease is suspected. Remember that these animals are worth submitting to PME to keep a check on common chronic diseases within a flock.

Prevention

See Chapter 21 for respiratory diseases. In the case of Johne's disease, this may be more widespread than realised. As diagnosis is difficult in the live animal, there is no simple advice about prevention. Animals should be culled as soon as disease is suspected. In pedigree flocks offspring should also be culled as they may be infected as young lambs. The cattle vaccine may be used in sheep flocks (with permission from the Divisional Veterinary Manager (DVM)) if the situation warrants it. Wildlife such as rabbits and deer may act as reservoirs of infection.

Weight loss with normal appetite and normal intake, many animals affected

Here the cause is likely to be either nutritional or concurrent disease, particularly parasitic.

If in mid/late pregnancy or lactation

With ewes carrying multiple fetuses, it is almost impossible to prevent loss of condition in the last 3 weeks of gestation, when demand for energy is twice that for maintenance. The uterus and contents occupy up to 60% of available space so that appetite is much reduced. If the ewes entered mid pregnancy in good condition, the loss of condition can be accommodated. In early lactation the appetite increases dramatically, but with energy demand now being three times that of maintenance, weight loss is inevitable. A ewe maintaining triplets faces energy demands equivalent to the demands on a sow with 30 piglets.

> Rations which contain significant amounts of undegradable protein (UDP) actively encourage the metabolism of body fat to produce milk. Such diets should not be used for ewes of already poor bodily condition.

Check for

- Quantity and quality of food, as above.
- Age distribution – social pressures on young, old or less dominant members.
- Trough space allowed.
- Chronic liver fluke – faeces sample. Number of eggs present is probably an indication of severity of infection, but even one is significant. Blood sample for liver enzymes – gamma glutamyl transferase (GGT) is indicator of chronic damage especially to bile ducts.
- *Haemonchus* infection – anaemia and oedema associated with wasting. Chronic infection resembles malnutrition.

Weight loss, whole flock affected

Where a whole group or flock is involved, nutritional factors are again the most likely cause, although parasitic problems should also be considered.

Check

- Whole feeding policy – roughage and concentrate quantity and quality.
- For owner inexperience.
- For owner with cash flow problem – unable to purchase sufficient feed.
- For overstocking – unreasonable expectation of capacity of land available.
- For mismanagement of winter shearing – failure to provide increased feed supply.
- Cobalt and copper status. Although these trace elements are involved in failure to thrive in young animals, it would be uncommon for deficiency of these to cause weight loss in the adult – see Chapter 9. Occasionally where copper availability is reduced because of land improvement, weight loss in adult sheep may result.

> Where all feed inputs are home grown, the potential for accumulating deficiencies is aggravated. In this situation blood biochemistry is likely to be helpful, with samples taken from a minimum of six affected animals.

Samples required – heparinised, oxalate-fluoride and clotted blood. Check: glucose, AST, GLDH, SDH, BHB, albumin, urea, Ca, Mg, inorganic P, copper, cobalt (vitamin B_{12}).

Weight loss, reduced appetite, individual animals affected

Here a reduction in appetite is usually a result of concurrent disease affecting either the ability of the animal to obtain or ingest food, or the result of the disease process itself, particularly in the terminal stages.

Check for

- Lameness reducing mobility especially footrot, foot abscess, arthritis.
- Mouth lesions – see Chapter 13.
- Gastrointestinal problem, e.g. rumen impaction, abomasal impaction, terminal Johne's disease.
- Sight problems – inability to see food.

If in late pregnancy

Check for

- Pregnancy toxaemia – typical signs of blindness, dullness and depression with evidence of large fetal load. Check blood sample for glucose, BHB, urea, albumin, calcium, magnesium.

- For imminent parturition – animals often have a reduced appetite immediately prepartum.

Weight loss, reduced appetite, many animals affected

Here the cause is likely to be major interference with feed intake, or gross neglect of chronic disease such as liver fluke.

Check

- For major outbreak of parasitic disease – liver fluke, haemonchosis – faeces samples for egg counts (see above).
- Water supply – animals on a wet diet such as silage or roots need little extra water except when lactating. Animals on dry diets may drink several litres daily and considerably more when lactating. In such a case, deprivation for more than 24 h, e.g. frozen pipes, will have an effect on appetite.
- Water palatability – contamination with chemicals, e.g. bitumen, creosote.
- Food palatability – fungal spoilage of concentrates or forage, especially big bale silage, accidental contamination with chemicals, mistakes in mixing feed additives.

See Appendices 3 and 4 for information on nutrition and parasite control.

13 Mouth disorders

Mouth lesions in sheep have assumed great importance since the 2001 foot and mouth disease epidemic. Although erosive lesions, often relatively minor, are seen on the dental pad of FMD-affected animals, it has become apparent that many normal sheep have unexplained deep ulcerations present on the gums below the incisor teeth (subsequently referred to as OMAGOD lesions) and this has complicated the clinical diagnosis of FMD. In the first edition of this book FMD was included as a possible cause of salivation and lameness for the sake of completeness, rather than in expectation of seeing the disease. This position has clearly changed and every clinician will need to be constantly aware of the possibility of FMD in sheep and the difficulty of diagnosis.

Aside from FMD, the presence of mouth lesions that cause difficulty in eating may often not become obvious until the animal loses flesh, or obvious signs of debility are present. This is an inevitable consequence of the inability of the shepherd in many commercial flocks to closely observe the grazing and ruminating behaviour of each individual animal. Where closer supervision is possible, for example in a small pedigree flock, mouth problems may be noted earlier, as a result of noticeable discomfort during eating or ruminating or by signs of quidding.

Before examination of the inside of the mouth of adult sheep, it is extremely helpful to palpate the mandibular rami where the presence of swellings will immediately indicate molar tooth problems. Additionally, palpation through the cheeks along the length of the molar teeth allows identification of overgrown, displaced and missing teeth. Resentment to application of pressure will also indicate likely problems.

A sheep mouth gag and torch are essential pieces of equipment where any mouth examination is to be carried out. It is impossible to perform this examination adequately, except in the case of the young lamb, without these pieces of equipment because of the anatomy and temperament of the sheep. The distance to which the jaws can be opened is very limited, and it is impossible to examine the deeper recesses without an external light source. In addition, the clinician risks injury or loss of fingers if these are inserted into the mouth without the protection of a gag.

Prolonged examination of the molar teeth causes considerable stress in the adult sheep, and the use of sedation or even general anaesthesia should be considered, particularly in the case of potentially valuable pedigree animals, where remedial dental work is likely to be required following the examination.

Although tooth problems in the adult animal dominate this subject, mouth problems of various types can affect all age groups.

Neonatal lambs

Mouth problems should always be suspected in the young lamb which appears unable or unwilling to suck, although bright and alert in all other respects. There will rarely be external evidence that the mouth is the source of the problem.

Individual lamb affected

Check for

- Parturient trauma – separation of mandibular symphysis (poor lambing technique), swollen tongue.
- Cleft palate.
- Predator trauma – tongue tip missing.

Many young lambs affected

Check for

- Periodontal orf lesions.

> Severe orf lesions can be present in the mouth, with little external evidence on the lips, within a few days of birth. This is particularly common in groups of artificially reared lambs.

Treatment

There is no specific treatment for orf (although homeopathic or other oral remedies have been used). Lesions will eventually heal if the lamb can continue to feed, but antibiotic cover may help to avoid secondary infection.

Prevention

Prevention is also difficult. Some advocate vaccination of ewes at least 8 weeks before lambing, but vaccination scabs must not be allowed to contaminate lambing sheds, otherwise the situation can be made worse. Having vaccinated the ewes, it is then also necessary to vaccinate the lambs soon after birth. There is no transfer of protective antibodies via the colostrum. In the case of orphan lambs, attention should be paid to frequent disinfection of feeding bottles and teats.

> Orf is a zoonosis. The vaccine is live, therefore should be handled with care.

Growing lambs

Individual lambs affected

Check for

- Trauma.

Many lambs affected

Check for

- Drenching gun injuries. These will often be unilateral and similar in position in all affected animals, usually starting in the lateral buccal pouch and continuing towards the throat. Frequently there will be a characteristic putrid smell of necrotic tissue. If the injury is of several days' duration, the throat will be swollen, but in the early stages there will be no indication that the inability to eat is buccal in origin. Drenching should always be carried out in a careful and considerate manner to prevent injury.
- Necrotic stomatitis. Commissures of lips show multiple necrotic lesions; may involve tongue, hard palate and cheeks. This condition is usually secondary to orf, or results from grazing abrasive weeds such as thistles or docks and brambles, and may be aggravated by trimming such weeds. If in any doubt, check with Divisional Veterinary Manager (DVM) about FMD.

Adult sheep

Mouth disorders in older sheep fall generally into two categories. They may affect individual animals, producing signs which directly indicate that the mouth is the source of the problem. Alternatively, mouth lesions will only be identified during investigation into a wider flock problem, usually weight loss, involving a number of animals. Figure 13.1 shows the common sites of tooth and jaw disorders. The possibility of FMD should also be borne in mind.

Presence of lesions on gums or dental pad

Check for

- FMD. Sheep may not be particularly ill or very lame, since this phase appears to pass quickly in this species, if seen at all. Lesions consist of blisters which burst, leaving shallow erosions on the dental pad particularly. Check for pyrexia and closely examine feet for blisters around coronary band or in interdigital space. If in any doubt, call the DVM and stay on the farm.
- OMAGOD (ovine mouth and gum obscure disease). This consists of deep, well-demarcated ulcers, often on the gum below the incisor teeth. The ulcers

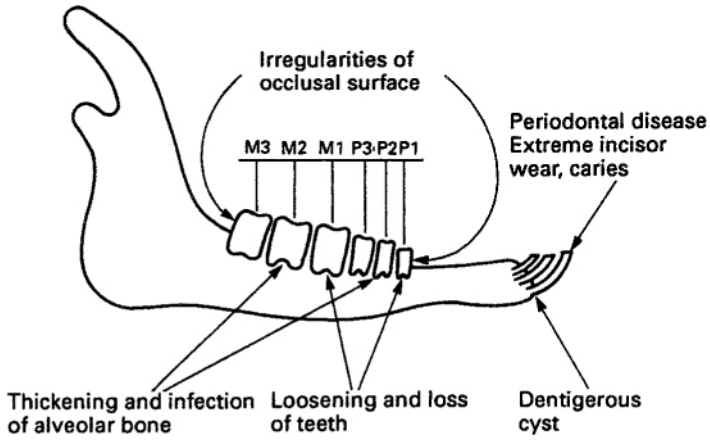


Figure 13.1 Common sites of tooth and jaw disorders in adult sheep.

are thought to be traumatic in origin, resulting from poor, rough grazing, newly introduced feed or salt blocks, or recent application of fertiliser to pasture. If in any doubt, treat as FMD suspect.

Individual mouth problems

Excess salivation

Check for

- Trigeminal/facial paralysis – unilateral paralysis of ear, eyelid and facial muscles suggests listeriosis or middle/inner ear infection.
- Access to irritant plant or chemical poison.
- Throat lesions – inability to swallow saliva, drenching gun injury.

Cud spilling

This is an annoying and puzzling condition particularly in young animals, which may also affect a group of contemporaries.

Check for

- Molar tooth problem – eruption, impaction, looseness, loss.
- Ulceration or fibrosis of tongue – actinobacillosis.
- Possible actinobacillosis of oesophageal groove.
- Possible partial obstruction of oesophagus by enlarged lymph node (CLA).
- Possible oesophageal dilatation.
- Breed – may be genetic predisposition – seen in longwools, but other breeds are also affected. There seems to be a functional disturbance of rumination with failure to swallow the liquid part of regurgitated food boluses. The green liquid dribbles from the mouth and stains the neck and front legs.

Treatment

Sheep with severe tooth problems should be culled. In the case of valuable old pedigree sheep, it may be possible to buy time by removing loose teeth and filing off sharp edges.

Cud spilling in young animals often stops if affected animals are housed on hay, but may restart when returned to grass. It is worth trying a course of parenteral antibiotics if actinobacillosis is suspected.

Swelling of mandible or submandibular region

In this category, molar tooth problems predominate. Almost invariably the first molar tooth is the first to become involved in any disease process (see colour Figure 7). This is because this is the first permanent cheek tooth to erupt (at 3 months of age), whereas the adjoining permanent third premolar does not erupt until nearly 2 years of age.

In some cases, radiography of the jaws may be helpful to assess the extent of damage.

Check for

- Molar tooth problem.
- Osteomyelitis of jaw.
- Actinobacillosis of cheek.
- Dentigerous cyst – incisor root area.
- Submandibular oedema – chronic fluke, haemonchosis.

Mouth disorders usually identified during investigation of a flock problem

Apart from FMD, there are two common conditions which usually affect significant numbers of animals or even whole groups. The client will consult not as to the presence, which is self-evident on examination, but because of a high incidence or changing incidence, and the possible reasons for such changes. These conditions are incisor loss and extreme incisor wear. Molar disease may also affect groups as well as more sporadic individual cases as described above, but this may not be recognised by the farmer.

There may not be a good correlation between body condition score and incisor tooth loss, except perhaps during winter feeding or in adverse environmental conditions, i.e. high hill farms. The relationship between body condition and molar tooth condition is more significant. Although farmers readily recognise incisor loss and cull for it (sometimes unnecessarily), molar disease which has much more serious consequences may go largely unrecognised, as previously mentioned.

Extreme incisor wear

Check

- Nutrition in early life – during tooth development.
- For evidence of fluorosis (bone samples required).
- Geology – high sand content causing tooth wear.
- Winter feeding – root feeding in sandy or frozen ground.

True dental caries, with pitting of the incisors, does sometimes occur and may be associated with high carbohydrate diets.

Periodontal disease, incisor tooth loss ('broken mouth')

Check

- Breed – incisor/dental pad apposition.
- Feeding – big bale silage, self-feed silage.
- For use of feed blocks.

In spite of these suggestions, there is still no clear evidence as to the cause of this widespread condition which forces premature culling of many hill ewes. Trials involving moving sheep from unaffected flocks on to affected farms have shown that there is a definite individual farm incidence.

Treatment of tooth disease

Shortening the crown length of incisors (tooth grinding) in an attempt to reduce premature loss is not legal in UK. Apart from removal of loose teeth or attempting to rasp sharp edges on molar teeth, treatment of tooth disease is not appropriate.

Prevention of tooth disease

There is a strong breed and farm factor in periodontal disease, premature incisor loss and molar tooth disease, although no breed is excluded. Mule and Border Leicester sheep are particular sufferers, with jaw conformation playing a part as differential rates of upper and lower jaw growth are involved. Whatever the breed, more attention should be paid to careful selection of breeding stock for soundness of incisor and molar teeth.

14 Lameness

The individual lame sheep has long been assumed by the sheep keeper to be one of the inevitable burdens of the trade – like the poor, always with us. However, it is now realised that lame sheep often present welfare problems out of all proportion to the severity of the visible lesions. Quite apart from the concern of allowing unnecessary suffering, the progressive sheep keeper accepts that any lameness, whatever the cause, will give a significant loss of production. The clinician may therefore be called on to diagnose and treat individual (particularly valuable pedigree) lame animals more frequently, and may no longer be limited to giving assistance only in 'outbreaks' of lameness. Sadly, however, many flocks are still seen containing an unacceptable number of sheep on their knees.

The first decision the clinician must make is – does this clinical picture represent true lameness, i.e. the result of some pathological process in the foot, or in muscle, bone or joint? Or is it rather a case of peripheral nerve injury or central nervous system lesions? Inevitably there will be some overlap with conditions discussed in Chapter 15, which covers neurological disease.

In some cases the differentiation may be obvious, for example a flock affected with chronic footrot, or cases of swayback, in a known area or farm subject to copper deficiency. Generally however, the veterinarian must rely on detailed examination of the history of each episode, combined with a knowledge of farm or area, and then an examination of the patient.

In young lambs, joint problems are a more important cause of lameness than foot lesions. In contrast, the vast majority of cases of lameness in adult sheep originate in the foot. An initial rapid examination may be made of the upper limb to eliminate obvious gross lesions such as fractures, but the main examination should concentrate on the foot, returning to higher structures if the site of lameness is not found.

The possibility of FMD must always receive serious consideration, and should never be relegated to the 'unlikely' category. Experience during the 2001 FMD epidemic showed that the degree of lameness seen in infected flocks can be very variable. Some sheep may be unwilling to move, while others show only fleeting signs of lameness.

Examination of the foot

In some cases of acute foot lameness, the site of the lesion may be very difficult to locate. That the foot is indeed the site is simple enough to determine when the classic signs of heat, pain and swelling are present. However, in the case of

some penetrating foreign bodies or the early stages of white line abscessation, careful searching is necessary to locate first the affected claw and then the actual site of the lesion. A good sharp knife, used carefully, together with trained eyesight are the most important diagnostic tools.

> Foot paring should always be done with great care. At the toe, the digital artery is easily cut if too much horn is removed, and this can lead to profuse haemorrhage immediately and chronic granuloma formation in the long term.

Examination of the upper limb

- Atrophy of muscles – indicates lameness of some duration.
- External injuries – may indicate possibility of fractures.
- Joint swellings – indicate septicaemic arthritis in the lamb, osteoarthritis in the aged animal.
- Other swellings – may be abscess, haematoma, trauma.
- Stance – if abnormal, this may be to ease pain in foot or joint, or may indicate nerve or tendon damage.

In the diagnosis of the cause of lameness, the age of the affected animal(s), the number affected, and the findings of the clinical examination will all narrow the field of possibilities.

Neonatal lambs

True lameness immediately after birth is not common, most cases resulting from trauma. In contrast, incoordination is common in young lambs, associated with faulty development of the nervous system, e.g. swayback, border disease, cerebellar hypoplasia. For more details of these conditions see Chapter 15.

Check for

- Obstetric trauma (excessive traction) – fractures, dislocations, radial paralysis, bruised tendons.
- Fractures resulting from treading by dam or other ewes in confines of small pens.
- 'Short' (contracted) tendons, usually bilateral affecting forelegs. Severe cases may be unable to stand, or walk on dorsal aspect of fetlocks. If not too severe, these may be corrected by splinting.

Young lambs over 2 days of age

With the exception of trauma, incidents of lameness in young lambs will usually involve significant numbers of animals, either because the causes are man-

agement related or infectious in origin in a group which is immunologically naive. Joint lesions are likely to be more common than foot lesions in this group.

Individual lamb, sudden lameness

Check for

- Trauma – fractured leg (trodden on by adult sheep).
- Foreign body – thorns easily penetrate the soft horn.
- Abscessation in claw – abscesses occasionally develop within a few days of birth and usually affect one digit only. These are seen quite commonly, although the cause is obscure. Even though they look alarming, drainage of pus and treatment with antibiotic usually brings about complete resolution.
- 'Redfoot' – this probably hereditary condition is seen occasionally, particularly in Scottish Blackface lambs, although rarely other breeds may be affected. It is a defect of the skin epithelium which leads to horn loss, infection and progressive lameness. Mouth and eye lesions may also be present. There is no treatment. If related to the introduction of a new ram, he should be culled.

Many lambs affected

By far the most common cause of lameness in groups of lambs of this age is joint infection (neonatal septicaemia/polyarthritis). Nutritional myopathy due to selenium/vitamin E deficiency is also common, causing stiffness and inability to walk far. Scald and footrot are not common in very young lambs unless there is a high incidence in the flock and poor management conditions such as dirty bedding facilitating spread.

Check for

- Neonatal polyarthritis.
- Tick pyaemia.
- Scald or early footrot lesions.
- Nutritional myopathy.

These are considered in detail in the following paragraphs.

Neonatal polyarthritis

This usually affects several joints in more than one limb, but is rarely bilaterally symmetrical. The knee and stifle are most commonly affected. In some cases joint distension may be obvious, but in other cases severe lameness may exist with minimal detectable joint abnormality. Concurrent navel infection is common. A variety of bacteria is found in these cases – *Streptococcus dysgalactiae* is probably the most common, but others include Staphylococci, coliforms, *Arcanobacterium (Corynebacterium) pyogenes*, *Fusobacterium necrophorum*,

Erysipelothrix rhusiopathiae. These cannot be differentiated on clinical grounds. If lambs do not respond to treatment, samples of joint fluid from untreated cases should be submitted for bacteriological examination. See also Chapter 8, Perinatal lamb losses.

Treatment of neonatal polyarthritis

Treatment can be unrewarding unless cases are identified early, since joint damage may be irreversible. Adminstration of broad-spectrum antibiotic by injection, based on the result of bacteriological tests, is labour intensive but necessary. Steroids may also help resolution. Severe cases may require drainage and flushing of affected joints.

Prevention of neonatal polyarthritis

This condition is usually associated with poor hygiene at lambing, or application of dirty castrating or tailing rings. Provision of adequate clean dry bedding both in pens where ewes lamb and in individual pens is crucial. Routine for navel dressing should be checked – tincture of iodine is probably best. The whole navel, particularly the broken end, should be covered by dipping; nonspill teat-dipping cups are ideal for this. Spraying can easily leave parts untreated unless great care is taken. Containers of navel dip should be changed regularly to prevent contamination. Lambs' navels should be checked 24 h after birth and retreated if they are still damp. In the face of an outbreak, treatment of every newborn lamb with injectable antibiotic may be necessary until hygiene is improved, but blanket treatment with antibiotics to cover management failures is not acceptable.

Tick pyaemia

Lambs are lame from about 2 weeks onwards with swollen joints, and are on rough or tick-infested pasture. This results from *Staph. aureus* infection gaining access to joints in lambs suffering from concurrent tick-borne fever which lowers resistance to infection. Abscesses may be present in other parts of the body, e.g. spinal cord, liver. If bacteriological investigation shows bacteria other than *Staph. aureus* present, the case is likely to be one of neonatal polyarthritis rather than tick pyaemia.

Prevention of tick pyaemia

As treatment is usually not successful, prevention is essential. Ticks can be controlled by application of a synthetic pyrethroid 'spot-on' or 'pour-on' to both ewes and lambs before turning on to tick-infested grazing. If this is not sufficient to control the disease, lambs may be given an injection of long-acting antibiotic prior to exposure to ticks.

Scald or early footrot lesions

Typical moist lesions in cleft, nonprogressive if scald, progressive to separation of sole from heel if footrot present. Footrot is less common in young

lambs than in older ones but can occur if housed in poor conditions. See later for more details.

Nutritional myopathy (white muscle disease, vitamin E/selenium deficiency)

Lambs are stiff, unable to move, or collapse on being driven. This disease picture can be confused with neonatal polyarthritis – careful examination should be made of the joints. The condition often affects fast growing lambs, and stiffness is characteristically seen in the shoulder muscles which may atrophy if successful treatment is not given quickly. The condition is often very painful to the affected lamb, and it may be unable to rise from lateral recumbency. Check creatine kinase (CK) concentration (serum or plasma sample required). Should be taken early in course of disease as concentration falls after a few days – >1000 i.u./ml is significant. Check rest of group (six minimum) for GSH-Px – heparinised samples required.

Treatment of nutritional myopathy

Affected lambs and contacts should be treated with an injectable vitamin E/selenium product. Administration of small doses of an NSAID may help very stiff lambs to get on their feet, and nursing is important to ensure they do not starve.

Prevention of nutritional myopathy

This can be done by dosing pregnant ewes orally with a pellet or a soluble glass bolus containing selenium. Alternatively, ewes may be injected with a slow-release selenium product or a combined selenium/vitamin E preparation about 2 months before lambing is due to start. Diets based on poor quality hay or grain treated with propionic acid are likely to be particularly low in vitamin E, therefore require supplementation.

> Monensin poisoning used to occur occasionally as the result of miscalculation in inclusion rate when added to creep feed, and resembled nutritional myopathy. *It is now illegal to feed monensin to sheep.*

Growing lambs (Figures 14.1 and 14.2)

Some of the conditions affecting very young lambs may still be apparent at this intermediate growing stage. Lesions acquired in early life in a mild undetected form, and nutritional myopathies and osteopathies, may also become apparent. In addition, growing lambs become susceptible to the important infectious causes of foot lameness.

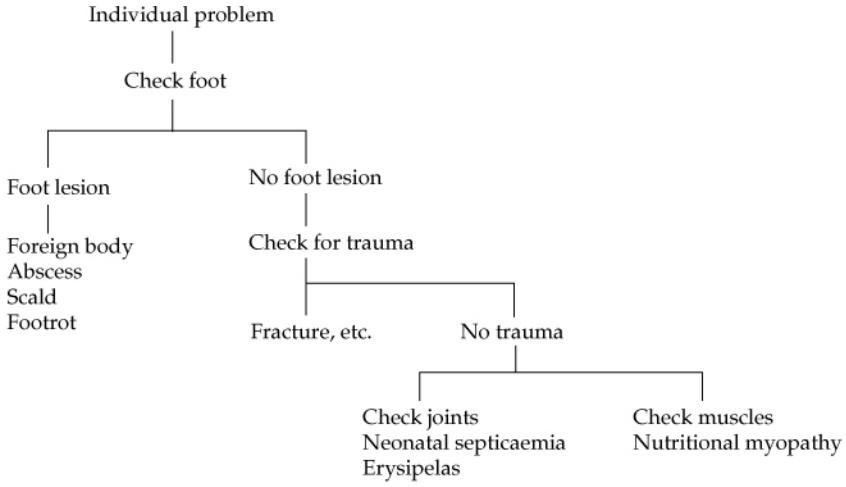


Figure 14.1 Lameness in growing lambs – individual problem.

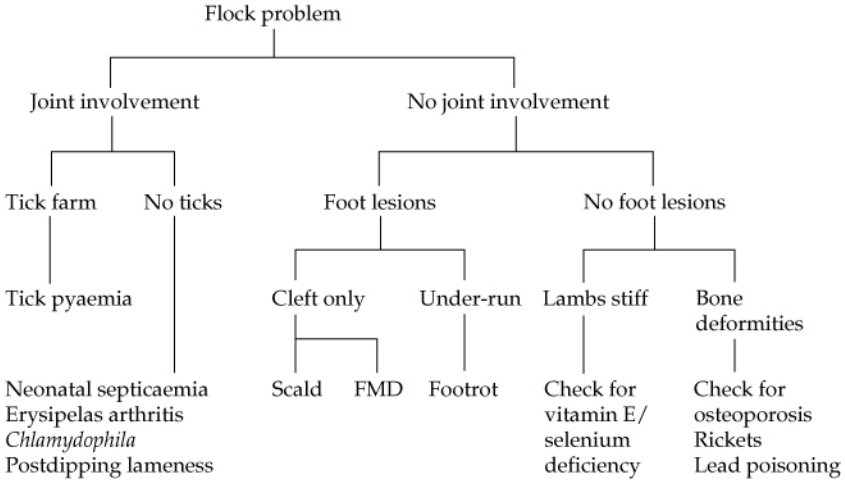


Figure 14.2 Lameness in growing lambs – flock problem.

Lameness due to foot problems

Check for

- Scald – the lesion is limited to the cleft of the foot, with moist raw areas on the axial surfaces of the claws which cause considerable pain, although the sheep is able to run when necessary. Scald is extremely common in growing lambs, particularly where they are grazing long wet grass, or where muddy areas are present around feed troughs, creep feeders or in gateways. See below.

- Soreness of interdigital space which may result from fertiliser application while lambs remain on field.
- Footrot – this is characterised by separation and under-running of the horn, starting from the heel. See below.
- Ovine digital dermatitis – this relatively new and serious cause of lameness begins around the coronary band, with detachment of the whole horn capsule. See below.
- Foreign body especially thorns.

Lameness due to joint problems

Check for

- Polyarthritis caused by *Erysipelothrix rhusiopathiae (insidiosa)* or *Chlamydophila abortus*. Lambs are 'poor thrivers' with marked stiffness or lameness and muscle wastage, especially in upper hind limbs. These two conditions cannot be distinguished clinically. Both cause insidious joint damage without much obvious joint swelling and are the result of infection acquired in the first week of life. Careful palpation is required to detect affected joints although these become more obvious as the disease progresses, because of muscle wastage (see colour Figure 8). Collect sample of joint fluid for stained smear and bacteriological examination (may be no growth if antibiotic treatment has been given). Blood sample for serology. This is very helpful in diagnosis of erysipelas, where a high titre is significant. Check for possible contact with pigs, or use of old pig pens for young lambs. Chlamydial polyarthritis occurs in some countries, but as far as is known, is not a problem in the UK. The chronic effects of neonatal polyarthritis (pus present in joints which may burst) may also still be evident at this stage.
- Postdipping lameness (*E. rhusiopathiae*) – see below.

Treatment of chronic polyarthritis

Since major joint damage has already been done, treatment is unlikely to be successful. Mildly affected animals may be housed and fattened indoors. Severely infected animals should be culled.

Prevention of chronic polyarthritis

Polyarthritis due to erysipelas infection may be prevented by vaccination of pregnant ewes to supply colostral antibodies to lambs. Two injections are necessary in previously unvaccinated stock; thereafter a single injection is given 2–3 weeks before lambing. This regimen is usually highly successful.

Other causes of lameness

- Nutritional myopathy as above.
- Rickets/osteoporosis (bent leg, double scalp) – lambs have leg deformities and may have stunted growth. Seems to be uncommon now, but may occur

in fast growing lambs reared indoors away from sunlight, or associated with chronic lead poisoning. Osteoporosis usually occurs on poor hill pasture but may also occur on lush grass. Lead poisoning may occur in lambs or ewes grazing land with old lead workings. Radiography may be helpful, together with analysis for lead if appropriate (blood, soil), or analysis of the diet for calcium, phosphorus and vitamin D content. Vitamin D can be administered if this is thought to be deficient.

Adult sheep

Although infectious diseases such as footrot, scald and ovine digital dermatitis are the major causes of flock lameness, there are many other causes, particularly affecting individuals or small numbers. The ageing process plus the demands of reproduction lead to other disease processes such as osteoarthritis and postparturient sacroiliac arthritis, together with the long-term effects of any poor conformation of joints or digits, e.g. corkscrew toe and straight hocks. Another important cause of hind limb lameness in the lactating ewe is acute mastitis. The udder should always be examined in cases of sudden lameness in this category of animal.

> Be alert to the possibility of foot and mouth disease where sudden flock lameness is accompanied by systemic illness, but remember that some sheep show few clinical signs.

Individual lame sheep (Figure 14.3)

All the previous conditions may occur at a low incidence within a flock. In addition, there are a variety of important causes of lameness which may affect individual sheep. The most common are: foot abscess, white line disease, foreign body, trauma, granuloma, interdigital fibroma, impacted interdigital sebaceous gland, laminitis, poor conformation, e.g. corkscrew claws, osteoarthritis and mastitis.

As stated previously, a very careful examination of the foot should be carried out. While some lesions will be obvious, others may not.

Acute lameness

Check for

- White line abscess.
- Pedal joint abscess.
- Foreign body.
- Acute mastitis in a lactating ewe.
- Laminitis

These are considered in turn here.

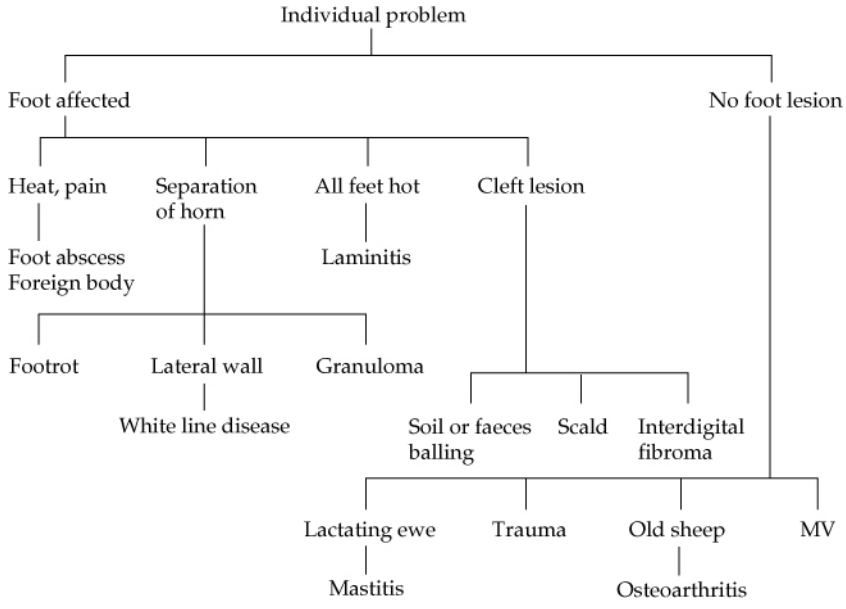


Figure 14.3 Lameness in adult sheep – individual problem.

White line abscess (see colour Figure 9)

Heat and pain will be present in the affected claw, but there may be little or no swelling. Careful paring along the white line will reveal either points of black discolouration or more extensive separation of the wall from the underlying laminae ('shelly hoof'). Pus formation remains superficial, running up under the lateral wall of the claw, eventually bursting at the coronet if not relieved before. After drainage of pus, the lateral wall separates and can eventually be pared away leaving no permanent damage.

Treatment of white line disease

The affected claw should be carefully pared and loose wall horn trimmed. Any dark tracks should be carefully followed, particularly those running up the wall of the hoof. Great care should be taken to avoid causing haemorrhage which may lead to long-term granuloma problems. If pus is not found or the horn is so hard that paring is impossible, poulticing for 24 h softens the horn, making paring much easier and encouraging pus to burst out. Once pus has been released, the severity of lameness will soon decrease, exposed laminae will keratinise and a strip of hoof will loosen. This can later be pared away. Some sheep have repeated attacks at intervals of a few months and may be better culled.

Prevention of white line disease

It is difficult to advise on this. Since this condition is not related to footrot, foot bathing and vaccination will not help. Regular paring to remove overgrown

horn may help to prevent acute episodes but the main problem is poor horn quality. It may be worth considering addition of biotin to the diet, based on experience in cattle. Selection of breeding stock should also take into account foot health.

Pedal joint abscess (see colour Figure 10)

In this case there is obvious swelling of the affected digit, and sinuses discharging pus appear at several points around the coronary band including axially within the cleft. A septic arthritis with disintegration of the pedal bone quickly becomes established. Heavy rams seem particularly prone to this type. Use of local or regional anaesthesia should be considered in the investigation of these cases. Radiography may be helpful in assessing the degree of joint damage.

Treatment of pedal joint abscess

Usually by the time these cases are seen, damage to the joint and pedal bone is such that amputation of the affected digit is the only course open. If seen early, drilling into the pedal joint and placing a catheter to allow frequent flushing of the joint may promote healing and ankylosis.

Prevention of pedal joint abscess

Regular foot trimming will keep the feet in shape, but there is no specific method of preventing this condition. Selection of breeding rams should take into account foot health and conformation.

Foreign body

This may affect the cleft or the claw itself. A foreign body within the cleft should be obvious. One penetrating the claw may be much more difficult to find, e.g. sharp thorns may become completely embedded and great care will be required to detect the head.

Acute mastitis in a lactating ewe

Lameness or dragging the hind leg on the affected side is an early sign, and early diagnosis may lead to successful treatment.

Laminitis

Characteristic stance, all four limbs bunched under body, arched back, heat in feet, increased digital pulse. Check feed quantity and analysis, especially protein content, sudden feed changes, or concurrent disease, e.g. metritis. Laminitis may occur in heavy ewes confined to fostering pens, either due to overfeeding or failure to lie down.

Chronic lameness

Check for

- White line disease – see above.
- Interdigital 'fibroma'.
- Impaction of the interdigital sebaceous gland.
- Chronic granuloma formation.
- Osteoarthritis.
- Abnormalities of conformation.
- Visna.

More details are given here.

Interdigital 'fibroma'

This is an outgrowth of skin originating from the skin/horn junction on the axial surface of the claw, and may arise from one or both claws, occupying the space between the claws. Excoriation and infection of the fibroma is common, leading to 'pinching' and lameness. It is particularly common in heavy Suffolks and may have a genetic predisposition. Removal under local anasthesia may be necessary.

Impaction of the interdigital sebaceous gland

This is not usually a cause of serious lameness but occasionally the swelling forces the claws apart, allowing excoriation of the skin of the cleft.

Chronic granuloma formation (see colour Figure 11)

This usually results from over-paring of the foot, particularly at the toe, but can also be the result of footrot, foot abscess or foreign body. A strawberry-like lump of granulation tissue develops, which bleeds easily when touched. Often the horn grows to hide the granuloma, but careful paring will re-expose it. If the granuloma is cut, profuse haemorrhage results.

Treatment of toe granuloma

If the granuloma is simply cut off, the foot will bleed and the granuloma will regrow. It may be treated with success, after anaesthetising the foot with intravenous local anaesthetic, by carefully paring back loose horn, cutting it off at the base and cauterising with heat (use a calf disbudding iron). A more time consuming alternative is to expose the granuloma and use repeated applications of 5% formalin or copper sulphate crystals to promote keratinisation. The granuloma can be gradually pared away as it becomes keratinised.

Prevention of toe granuloma

Avoidance of over-paring, particularly cutting the toe too short, and control of chronic footrot will prevent most cases.

Osteoarthritis

This is quite common in old sheep. It may affect one or more limbs, front or hind. In the forelimb, the usual site is the elbow. Palpation will reveal bony enlargement of the joint with restricted flexion. It is usually, although not always, bilateral. Severe lesions lead to a characteristic 'restricted' movement of the front legs when walking, and to 'paddling' movements resting alternate limbs when standing. In the hind limbs, the sacroiliac joints and hip joints may be affected, causing incoordination rather than actual lameness. Affected animals should be culled.

Abnormalities of conformation, e.g. corkscrew claws or overgrowth of horn

These may predispose to lameness by allowing impaction with mud, or placing abnormal stresses on joints. Similarly, abnormalities such as excessively straight hocks or extreme sloping pasterns may induce stresses leading to eventual lameness. A hereditary component may be involved in all these conditions, so culling may be indicated.

Visna

This may be indicated if one hind limb is dragged, causing wearing of the dorsal aspect of the claws.

Lame sheep, flock problem (Figure 14.4 and Table 14.1)

Remember the possibility of foot and mouth disease. Check feet for vesicles and palate for erosions. Call DVM if unsure.

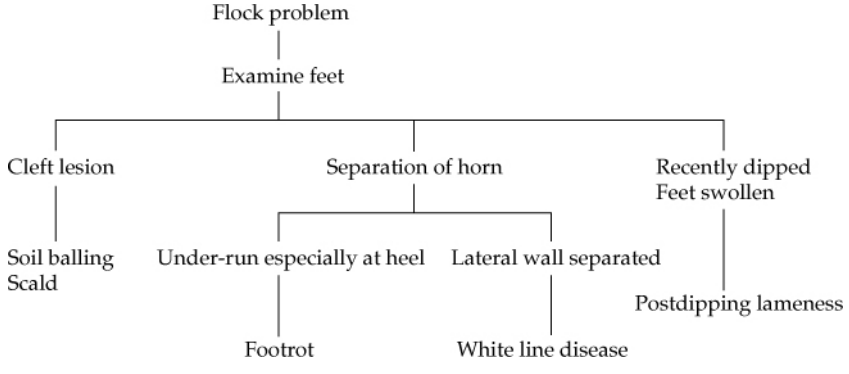


Figure 14.4 Lameness in adult sheep – flock problem.

Table 14.1 Main features of important causes of adult lameness.

Disease	Lesion	Cause
Scald	Interdigital dermatitis	*Fusobacterium necrophorum*
Footrot – benign	Interdigital dermatitis and separation and under-running of horn at heel	*Fusobacterium necrophorum* and *Dichelobacter nodosus* (benign strains)
Footrot – virulent	Separation and under-running of horn of sole and wall, necrosis and foul smell	*Fusobacterium necrophorum* and *Dichelobacter nodosus* (virulent strains)
Ovine digital dermatitis	Separation of horn capsule beginning at coronary band. Whole hoof may be shed	?spirochaete (related to digital dermatitis in cattle?)
Foot and mouth disease	Vesicles around coronary band and in interdigital space. Erosions on dental pad	Aphthovirus
White line separation (shelly hoof)	Separation of hoof wall from laminae, resulting pocket impacted with mud	None specific
White line abscess	Black marks in white line leading to abscess under wall horn. Claw hot and painful. Bursts at coronary band	Mixed opportunistic infection
Pedal joint abscess	Very painful swollen claw. Discharging sinuses around coronary band, disruption of pedal joint and pedal bone	Mixed infection especially *Fusobacterium necrophorum* and *A. pyogenes*
Postdipping lameness	Feet and lower limbs hot and painful. Cellulitis affecting skin of lower limb and laminae of foot	*Erysipelothrix rhusiopathiae*

Check for

- Scald.
- Footrot.
- Contagious ovine digital dermatitis.
- White line separation and/or abscess.
- Soil balling/bedding 'shoe'
- Postdipping lameness.

These are discussed here in detail.

Scald (see colour Figure 12)

This infection, which is caused by *Fusobacterium necrophorum* (or noninvasive *Dichelobacter nodosus*), never progresses beyond the foot cleft. The primary

lesion is interdigital dermatitis, with reddening or whitening, swelling and hair loss of the skin between the heels. Although the lesion looks relatively insignificant, affected animals are very lame. Facilitates the invasion of *D. nodosus* if present in the environment, and the development of footrot. Predisposing factors include dirty or damp bedding if housed, dirty or muddy areas around feeding or drinking troughs and long wet grass if outdoors.

Treatment of scald

If small numbers are affected, treatment with an oxytetracycline spray will be effective. If larger numbers are involved, footbathing in weak formalin (2–3%) is the most practical solution as the animals can be walked slowly through and do not need to stand in the bath. However, formalin is unpleasant and often it is difficult to get lambs to enter the footbath. Zinc sulphate (10%) can be used if preferred, but animals should stand in this for a few minutes. Treated sheep should be allowed to stand on a hard surface for a short time, particularly if the grass to which they are to return is wet. Formalin is acceptable in organic units.

Prevention of scald

Prevention is difficult as *F. necrophorum* cannot be eradicated from the environment. Keeping the grass sward grazed short should help. Feed or water troughs should be sited on well-drained ground, or hard core used to prevent muddy areas developing, with gateways similarly treated. Spreading builders lime in gateways may also help by raising pH. Regular footbathing in 2% formalin or 10% zinc sulphate may be necessary to keep control if there is a persistent problem.

Footrot (see colour Figure 13)

This is still the most common flock problem, since repeated infection does not induce a useful immune response. It is caused by the synergistic action of *Fusobacterium necrophorum* and *Dichelobacter nodosus.* There are a number of strains of *D. nodosus* which vary in invasiveness. Early lesions are similar to scald but progress to separation of the sole, usually beginning at the heels, with necrosis of the laminae and development of the characteristic foul smell. Invasive strains cause separation right across the sole and up the wall of the hoof. More than one foot may be affected at the same time. Carrier sheep are very common, and detailed examination of all feet is necessary to detect all such animals, although in many cases an obvious deformity of the hoof or horn is present. Isolation of *D. nodosus* and typing of strain can be done but is rarely required. Special transport medium is needed.

Treatment, control and prevention of footrot

It must be remembered that this is an infectious disease, so treating only obvious clinical cases will never result in a satisfactory flock situation. For most flocks, good control is the most practical aim, but the disease can be eradicated from closed flocks since *D. nodosus* can only survive on pasture for about 16 days maximum. Control must be based on a combination of procedures:

- Regular examination of all feet.

> Loose and overgrown horn should be carefully trimmed, but avoid cutting too deeply causing bleeding.

- Regular footbathing, preferably in 10% zinc sulphate solution – zinc sulphate (10%) is very effective as a footbath but animals must stand in it to allow penetration of the horn (detergent may be added to speed this). Instructions for a particular product should be followed, but in general, sheep with few foot problems should stand in it for 5 minutes and those with infected feet for at least 15 minutes.

> Formalin is often used, but is irritant and causes too much hardening of the horn if used repeatedly. It should never be used at more than 5% strength.

- Injection of antibiotic to badly affected animals – treatment with long-acting oxytetracycline (normal dose) or penicillin/streptomycin mixture (double the recommended dose), will speedily improve badly infected feet. Trimming should be left until a few days after giving the injection.
- Possible use of vaccine – vaccination can be curative as well as preventive, but immunity is fairly short-lived, so vaccination will need to be repeated just before particular risk periods such as housing. It should not be used within 8 weeks of lambing. The vaccine has an oily adjuvant which can leave vaccination reactions. It is also dangerous if accidentally self-injected, so care needs to be taken in handling. Read the data sheet carefully before use and make sure the client is aware of correct handling and vaccination schedule.

> Animals given footrot vaccine should not be treated with injectable moxidectin.

Eradication of footrot

Eradication is possible in closed flocks but the client must be aware of the risks of reintroduction by straying sheep, showing sheep, buying rams, etc. The principle employed in eradicating the disease is to examine every foot of every sheep and to divide them into infected and uninfected groups. Uninfected sheep are footbathed then put on clean ground (no sheep in the previous 3 weeks). Regular checks should be made of the uninfected group until it is clear that no inapparent infection has been missed. Infected sheep are treated and examined at weekly intervals. If cured, they can be added to the uninfected group. *Any persistently infected animals should be culled if repeated treatments are not effective.*

Contagious ovine digital dermatitis (CODD) (see colour Figure 14)

The cause of this apparently new disease and its differentiation from classic

footrot is still open to debate. There are suspicions of the involvement of spirochaetes, as in digital dermatitis in cattle. The main distinguishing feature is that the initial lesion is at the coronary band with rapid separation and detachment of horn proceeding downwards and loss of hair above the coronet. In many cases there is no response to orthodox treatments such as parenteral oxytetracycline or footbathing in zinc sulphate. Good response has been obtained footbathing with tylosin, lincomycin or lincomycin/spectinomycin, as used for digital dermatitis in cattle. Administration of tilmycosin by injection is also effective, but expensive.

White line separation and/or abscess

This is a common cause of lameness in individual sheep but may also be sufficiently widespread to be considered a flock problem. Separation of the lateral hoof wall occurs at the position of the white line. The pocket thus formed becomes impacted with mud and grit, and may lead to the development of a foot abscess. The cause is unknown, although there is a suggestion that biotin deficiency may be implicated. See above.

Soil balling (packing of cleft with a hard mixture of grass and mud)

This is extremely common in lowland flocks on lush grass, particularly in late autumn. The skin of the cleft becomes sore as a result of the hard grass/mud mixture which becomes trapped between the claws. Little can be done to prevent this, except for keeping sheep on well-grazed pasture at this time. A similar condition can develop in housed sheep where a 'shoe' of matted bedding and faeces can cause similar discomfort.

Postdipping lameness

This is usually associated with dipping in dirty or contaminated dips but has also been seen after handling sheep through muddy pens without dipping having taken place. A few days after dipping or handling, the feet are hot and swollen and the sheep may be pyrexic with septicaemia and polyarthritis. History and clinical signs should be diagnostic. Serology is not helpful. It may be possible to isolate the causal organism (*E. rhusiopathiae*) from blood or exudate from lesions.

Treatment of postdipping lameness

The organism is sensitive to penicillin and affected animals should be treated as soon as possible to prevent the development of chronic joint infection.

Prevention of postdipping lameness

Handling pens should be cleaned regularly and not allowed to become muddy. Gross contamination of dips should be avoided and dip should be discarded at the end of the day or a suitable bacteriostat, recommended by the dip manufacturer, should be added to prevent bacterial multiplication overnight. Badly maintained dip liners will predispose to problems, either through loss of integrity or by causing injuries to animals' legs.

15 Changed behaviour and neurological dysfunction

Over and above the natural variation in behaviour between breeds and in varying habitats, there exists a wide range of conditions resulting from dysfunction somewhere within the nervous system. These either have an effect on the behaviour of the animal or cause alterations in the ability to stand or walk normally. Depending on the site and nature of the problem, the animal may be mentally normal, depressed or hyperexcitable, and may exhibit other signs ranging from slight incoordination to total collapse. In some cases there is an obvious macroscopic lesion centrally or peripherally (for example an abscess in the brain or traumatic injury to a nerve), or microscopic lesions may be present (for example in listeriosis or scrapie). In other cases the problem results from alterations in blood chemistry (for example hypocalcaemia, hypomagnesaemia) which affect neurological function without the presence of any macroscopic or microscopic lesion.

While major changes will be immediately apparent to the sheep keeper, minor changes may only become noticeable during normal shepherding activities such as rounding up with dogs, driving, etc. A knowledge of individual behaviour within the flock (leadership, dominance, awkwardness, etc.) will need to be combined with observations of response to external stimuli in order to reach an overall assessment of behaviour as being normal or abnormal. To the competent shepherd it is often minor changes in behaviour or response to stimuli which indicate the early onset of many diseases.

The clinician must determine whether any behavioural change observed is in fact nonphysiological. Change in behaviour can be profound but can still be a normal physiological response. For example, some normal animals will adopt a recumbent or freezing posture merely in response to overstimulation. A neonatal lamb may not show 'flight' behaviour simply because it has failed to bond with a ewe. A parturient ewe may not show flight because a lambing site has been selected, and perhaps most obviously, the ewe with a newborn lamb will show fight and not flight behaviour to possible predators. It is also important to recognise an inability to react because of some gross physical disability such as severe lameness or arthritis.

Common types of neurological disease

A multiplicity of diseases, not surprisingly, have effects on the normal function of the central and peripheral nervous system, but for common conditions of

sheep these can be broken down into a small number of groups according to the type of disease process:

- Developmental, e.g. congenital malformations.
- Infections
  - viruses
  - bacteria
  - parasites.
- Metabolic and nutritional.
- Toxins.
- Injuries.
- Peripheral nerve or muscle abnormalities.

In many cases diseases are limited to, or are most common in, a particular age group. Speed of onset and progression of the signs are also important indicators in diagnosis (Table 15.1). Where the cause of a particular problem is not immediately clear, a neurological examination should be carried out in a logical progression, which should enable the clinician to answer the questions – where is the lesion (brain, spinal cord, peripheral nerves or muscles?) and what is likely to be causing it? The following outline summarises the most useful neurological tests which can be applied to sheep. Notes should be made of the results of all observations, since it is likely that a diagnosis can only be made by an overall assessment. Table 15.2 summarises the postmortem diagnosis of some neurological conditions.

Table 15.1 Timescale of onset of signs as guide to diagnosis in neurological disease.

Time taken for signs to develop	Disease	Diagnostic aids
Immediate	Cervical injury	Group of rams fighting
Minutes	Hypomagnesaemia	Response to treatment
Minutes to hours	Hypocalcaemia	Response to treatment
Hours	CCN	Opisthotonus, blindness, strabismus Response to treatment
Hours to day(s)	Pregnancy toxaemia	Urine (ketones) Blood (BHB, glucose)
	Louping ill	Tick area Time of tick activity
	Listeriosis	Silage feeding, scour, possible abortions
Days	Spinal abscess	Progressive, raised total WBC, crossover
Weeks to months	Scrapie	Usually >2 years old Pruritus, ataxia
	Gid	Usually <2 years old Gradual onset, progressive

Table 15.2 Postmortem diagnosis of some neurological diseases.

Disease	Gross pathology	Microbiology	Biochemistry	Parasitology	Histopathology
Swayback	+/– cavitation	–	Low Cu (liver)	–	+
Border disease	+/– characteristic lambs	+ virus isolation	–	–	+
Cerebellar hypoplasia	+/–	–	–	–	+/–
Hypothermia/ hypoglycaemia	+/– oedema of extremities	–	Low glucose	–	–
Encephalitis/ meningitis	+	+	–	–	+
Abscess	+	+	–	–	+
CCN	+ yellowing of cerebrum	–	Raised TK	–	+
Louping ill	–	+ virus isolation	–	–	+
Gid	+	–	–	+	No inflammatory reaction
Hypocalcaemia	–	–	Low Ca (Mg)	–	–
Hypomagnesaemia	–	–	Low Mg	–	–
Pregnancy toxaemia	+ fatty liver	–	Ketones in urine, low glucose, raised BHB	–	+ (liver)
Listeriosis	–	+	–	–	+
Scrapie	–	–	–	–	+

Routine for neurological examination

- Assess state of consciousness:
  Hyperexcited – increased responsiveness, nervousness, fits
  Alert – normal demeanour
  Depressed – reduced responsiveness
  Stupor – difficulty arousing
  Comatose – cannot be aroused
- Assess for any abnormal behaviour:
  Circling
  Head pressing
  Aimless wandering
  Turning in one direction
  Becoming stuck in corners
- Assess stance
  Normal or wide based
- Assess gait and posture
  Dysmetria (hypometria/hypermetria)
  Ataxia
  Paresis
  Paraplegia
  Quadriplegia
- Test postural reactions – these help to identify minor deficits or differences between the two sides of the body. Blindfolding accentuates abnormalities
  Head position and posture
  Wheelbarrow test
  Hemistanding
  Hemiwalking
  Hopping
  Proprioceptive positioning (foot position)
- Assess limbs
  Muscle tone (normal, flaccid, spastic)
  Reflexes (triceps, patellar, pedal)
- Assess panniculus reflex
- Examine head, assess cranial nerves
  Menace test
  Blindfold each eye in turn and assess vision
  Pupil size and symmetry
  Pupillary light response
  Eye position
  Eye movement
  Palpebral reflex
  Facial sensation
  Facial symmetry
  Jaw tone
  Balance
  Prehension and swallowing

- Take blood samples if necessary – neutrophils may show increase, or may be 'crossover' (more polymorphs than lymphocytes) if there is a septic focus, but not necessarily if the lesion is well circumscribed.
- Take CSF sample if necessary (under general anaesthesia from the atlanto-occipital space, with head flexed – site is in midline slightly proximal to a line joining anterior borders of wings of atlas, or under local anaesthesia from the lumbosacral space).

Examination of CSF

This may be helpful where a diagnosis is in doubt. Normal CSF is clear, colourless and contains no clots. CSF can also be used for calcium and magnesium estimation, and for bacterial culture.

Some abnormalities

- Yellow colour indicates haemorrhage (old) or jaundice.
- Turbidity indicates increased cell content – infection.
- Blood indicates faulty technique, haemorrhage (recent) or trauma.
- Clots show increased fibrinogen or protein.
- Increased protein or lowered glucose content may indicate infection.

Identification of site of lesion from assessment of neurological examination

In attempting to localise CNS lesions, all the abnormalities and deficits detected during a neurological examination need to be considered to see if a pattern emerges indicating a possible site. The most common patterns are as follows.

Indicating involvement of cerebrum (see colour Figure 15)

- Altered mental state (particularly depression, stupor).
- Behavioural change (e.g. circling).
- Visual deficit (if unilateral lesion, opposite eye is affected; if bilateral, both eyes affected).
- Strabismus if cerebral oedema present.
- Altered head position (aversion/turn).
- Proprioceptive deficits (affecting opposite side).

The commonest problems affecting the cerebral hemispheres are gid, abscess, CCN, meningitis/encephalitis, pregnancy toxaemia.

Indicating involvement of cerebellum (see colour Figures 16, 17 and 20)

- Wide-based stance.
- Ataxia.
- Dysmetria.
- Tremors.

- Head tilt.
- Poor head coordination.
- Opisthotonus and pelvic limb flexion.

The commonest problems affecting the cerebellum are gid, border disease, daft lamb disease.

Indicating vestibular involvement

- Head tilt (to affected side).
- Nystagmus.
- Facial nerve involvement (because this runs close to the middle and inner ear).

The commonest cause of vestibular disease is middle ear infection.

Indicating pontomedullary involvement

- Multiple cranial nerve deficits (facial and trigeminal paralysis commonly).
- Propulsive circling.

The commonest cause of pontomedullary signs is listeriosis.

Indicating spinal cord involvement

- Paresis, paralysis.
- Proximal to T2 – recumbent, unable to sit up.
- Distal to T2 – recumbent, uses forelimbs only.
- T1 to S1 – panniculus reflex affected.

The commonest cause of spinal cord damage is spinal abscess.

This is by no means an exhaustive list. Some signs are more reliable than others, and transmitted pressure effects of a large lesion may cause anomalous signs. Generalised CNS lesions will cause confusing signs.

Common diseases in which neurological abnormalities are seen

In all age groups, trauma to either the head, back or extremities is an obvious cause of neurological problems. In addition, three common procedures, if carried out incorrectly or carelessly, can cause damage:

(1) Injection close to a major nerve, e.g. the sciatic, can cause muscle paralysis.
(2) Injection with contaminated needle or drug can lead to abscess formation which can involve adjacent nerves.
(3) Drenching gun injuries in the pharynx cause sepsis which can easily spread to the high cervical spinal cord.

Other common neurological problems can be categorised according to the age group most often affected, and often by the speed of onset and progression of signs.

Neonatal lambs

In the newborn lamb, neurological abnormalities fall into two main categories – congenital abnormalities and those due to dystocia (see Figure 15.1). The commonest are a result of:

- Trauma during birth (anoxia, subcranial haemorrhage, peripheral nerve damage).
- Hypoglycaemia as a result of failure to suck. Lambs which have suffered dystocia, particularly oversized lambs, are very susceptible.
- Congenital swayback.
- Border disease infection in early pregnancy.
- Cerebellar hypoplasia.

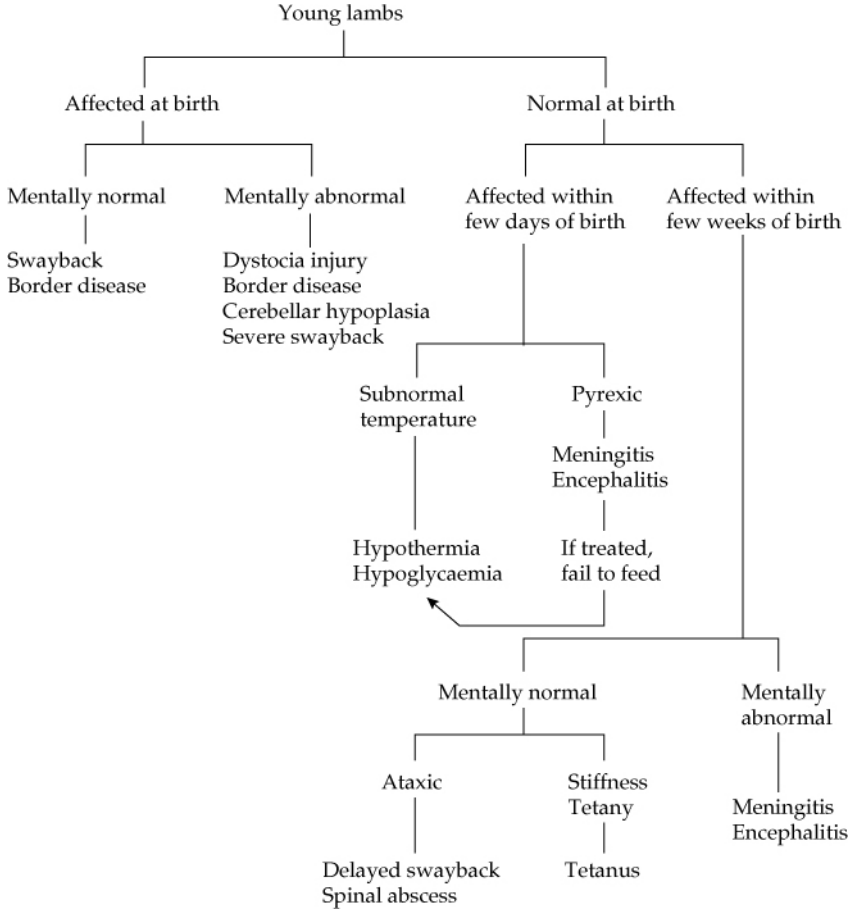


Figure 15.1 Diagnosis of neurological disease in young lambs.

Lambs unable to stand, or ataxic at birth

Check for

- History of dystocia, especially if large lamb.
- Congenital swayback.
- Border disease.
- Cerebellar hypoplasia.
- 'Dandy-Walker' syndrome.

These are discussed in turn in the following paragraphs.

History of dystocia, especially if large lamb

Hypoxia or subcranial haemorrhage may affect ability of lamb to stand and suck. These lambs are particularly susceptible to hypothermia/hypoglycaemia. Prolonged dystocia may also cause peripheral nerve damage, e.g. radial paralysis.

Congenital swayback

Severity of signs varies from mild hind limb ataxia to severe brain damage and death soon after birth. PME may show cavitation of brain in severe cases, or diagnosis requires histopathology and analysis of blood and liver for copper content, to confirm. Treatment is not possible because damage is irreversible. Adminstration of a copper product may prevent further deterioration, but care must be taken with dose rate as copper is very toxic. Badly affected lambs should be euthanased.

Prevention of swayback (see also Appendix 3)
Although usually thought of as a copper deficiency, the situation is more complex and is particularly related to the amount of molybdenum, sulphur and iron in soil, herbage and other foodstuffs ingested by the pregnant ewe. As there is a narrow margin between deficiency and toxicity, and considerable breed variation in absorption of copper from the diet, copper supplementation should *not* be given without a firm diagnosis and consideration of husbandry and nutritional factors. Some areas of the country are known to be 'swayback' areas. Ewes wintered outdoors take in considerable amounts of soil while grazing, so are at risk, particularly if the winter is mild and supplementary feeding is minimal. Housed ewes are removed from soil intake, therefore do not require supplementation normally. If supplementation is required, injectable and oral products are available. These should be administered to ewes about 10 weeks before lambing.

Border disease

Lambs show tremors, characteristic abnormalities of conformation (camel legs, domed skull, jaw defects), excessive hairiness and abnormal pigmentation (hairy shakers). Confirmation of diagnosis is by histopathology of CNS, virus isolation from spleen, lymph node or blood clot, and serology of flock. Treatment is not appropriate and badly affected lambs should be euthanased.

Control of border disease

Persistently infected, virus positive, antibody negative animals are responsible for spread of infection. In the first year of introduction into a flock, the client should be advised to sell the whole of that year's lamb crop together with the animals suspected of having introduced the disease. If the flock has a persistent problem, the best way forward is to try to expose all breeding stock to infection before they become pregnant, since infection outside pregnancy stimulates immunity. This can be done by close herding of the flock, preferably indoors, for at least 3 weeks. Replacement animals can be blood tested to ensure freedom from the virus and this is worthwhile when buying rams for closed or uninfected flocks. The disease is closely related to BVD in cattle, with a similar epidemiology. Infected cattle are more likely to pose a risk to sheep than vice versa. Vaccination of sheep may be a possibility at some time in the future.

Cerebellar hypoplasia (daft lamb disease)

Affected lambs show opisthotonus ('star gazing'), inability to maintain balance, and head swaying. Diagnosis is based on clinical signs, as histological examination may be unhelpful. This abnormality is thought to be genetic in origin and if it can be linked to the introduction of a new ram he should be culled.

'Dandy-Walker' syndrome (hydrocephalus)

This inherited defect is seen in Suffolk sheep. Work is in progress to see whether a marker can be found to identify a genetic predisposition. In the absence of a specific test, culling animals which produce affected offspring would seem to be prudent.

Young lambs (milk dependent)

In the first few weeks of life, lambs are exposed to a multiplicity of pathogenic bacteria which may affect the nervous system (see Figure 15.1). Congenital abnormalities may become more noticeable, and hypothermia with hypoglycaemic fits is a major problem in the first week or two (see Chapter 8).

If lamb is depressed, comatose or in fits, and has a subnormal temperature

Check for

- Hypothermia. Should respond to intraperitoneal or intravenous glucose administration followed by warming. If it does not recover, PME shows brown fat stores depleted (see Chapter 8).

If lamb is mentally normal, with incoordination or paresis of hind legs

Check for

- Spinal abscess – common site is C7–T2. Infection may also spread forwards from docking wounds or from infected navel. Signs are progressive, beginning with slight ataxia which usually worsens over a few days to complete hind limb paralysis. May be response to antibiotics if treated early enough.
- Tick pyaemia if in tick area – multiple abscesses from which *Staph. aureus* is isolated (see Chapter 14).
- Delayed swayback – may also be progressive, but not usually to total paralysis of hind legs; swaying at hocks when made to run is most characteristic. There is no response to antibiotic therapy. Diagnosis requires blood or liver for copper estimation, and may require histological examination for confirmation. This is not treatable, but adminstration of copper may prevent further deterioration. See above.

If lamb is mentally abnormal, pyrexic, stiff neck, progressing to opisthotonus

Check for

- Meningitis/encephalitis – this is often associated with other conditions resulting from poor hygiene at lambing – navel infections, septicaemia, enteritis, polyarthritis. *L. monocytogenes* – may occasionally cause neurological signs in young lambs, although septicaemia is more commonly associated with this organism in this age group. Early treatment with antibiotic or sulphonamide that will penetrate the blood/brain barrier may be successful.

If lamb shows generalised stiffness, hyperaesthesia and spasms.

Suspect

- Tetanus – check vaccination history, recent wounds, e.g. tailing, castration. Although treatment with antibiotics and antiserum can be attempted, this

may not be economic and the prognosis is poor. Vaccination policy should be reviewed.

- If Swaledale, check possibility of Swaledale encephalopathy – this is a congenital condition and may be inherited although little is known about the condition.

Growing lambs (Figure 15.2)

In this age group, the speed of onset of signs may be helpful in reaching a diagnosis.

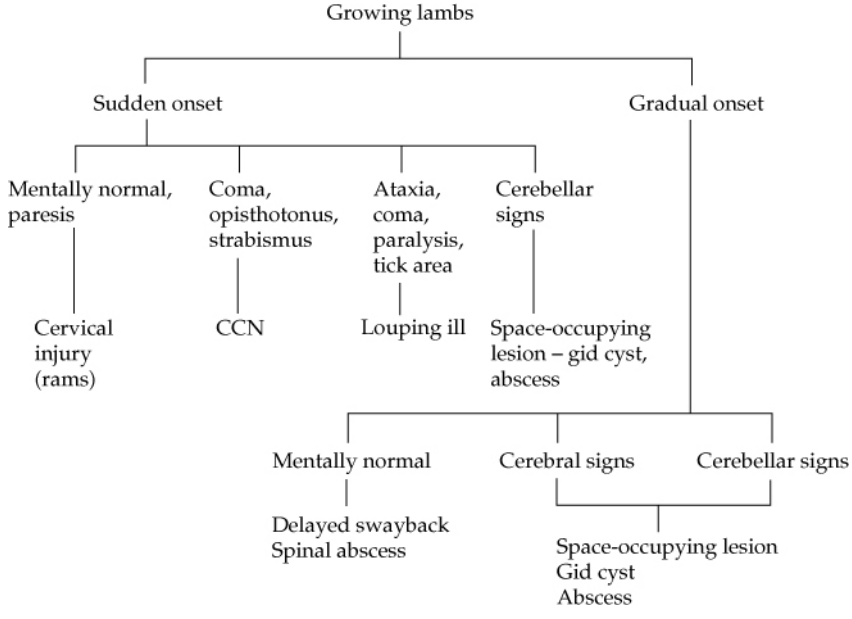


Figure 15.2 Diagnosis of neurological disease in growing lambs.

Sudden onset of signs

If animal is recumbent, blind, showing strabismus and opisthotonus (see colour Figure 18)

Suspect

- Cerebrocortical necrosis (CCN) – response to therapy (thiamine i/v) is the best pointer to correct diagnosis in an early case. Blood sample (heparinised) can be taken for transketolase estimation (this is a specific test for CCN), and faecal sample for thiaminase estimation. In a dead animal rumen contents can be checked for thiaminase, and examination of the brain (yellow discolouration of the cerebral hemispheres and fluorescence under UV light) will confirm.

Treatment of CCN

If cases are identified early, spectacular success can be achieved with intravenous injection of thiamine (vitamin B_1). The injection (2.5–5 ml of 15 mg/ml solution) should be given slowly and can be repeated at 3 h intervals if necessary up to five times. Alternatively, follow-up treatment can be given intramuscularly. If treatment is delayed, permanent damage to the CNS may result, with varying deficits including blindness persisting.

Prevention of CCN

Prevention is not possible due to the sporadic nature of the condition and lack of information on predisposing factors.

If animal shows cerebellar signs (tremor, dysmetria and nystagmus), often with rapid deterioration

Suspect

- Cerebellar gid cyst or possibly abscess (see below).

If in tick area

Check for

- Louping ill – incoordination progressing to paralysis, coma and death in 1–2 days. Samples required from live animal are heparinised blood and CSF; for dead animal remove brain, put small sample of brain stem into 50% glycerol saline for virus isolation and the rest into formal saline for histology. As this is a viral disease, treatment is not possible. The disease can be controlled by vaccination of replacement stock, reduction of the tick habitats and treatment of sheep with a synthetic pyrethroid pour-on preparation before turnout to tick infested ground.

If lamb has been losing condition, drinking excess and develops fits

Suspect

- Nephrosis – take blood sample for creatinine and urea estimation to confirm. The cause of this condition is unknown. It is possible it may be linked to intestinal parasitic diseases and associated dehydration, so attention should be paid to parasite control; however, other cases are apparently unrelated to these factors. (Where lambs with pulpy kidney are seen alive, these may exhibit fits before death occurs.)

If one of a group of young rams shows sudden onset of ataxia but is mentally alert

Suspect

- Damage to cervical vertebrae (cervical subluxation) as result of fighting. Radiography may be helpful in the case of a valuable animal. Similar signs may also result from a rapidly developing cervical spinal abscess. Check white blood cells (WBC) and differential count, which may help to indicate if an abscess is present. Prognosis is poor, but less severely affected cases may recover with confinement and steroid administration if injury is the cause. If an injury is suspected, soluble steroid should be given intravenously at a high dose as soon as possible after the injury has occurred.

Gradual onset of signs

If hindlimb ataxia is present

Check for

- Delayed swayback or spinal abscess as above.
- Chronic lead poisoning if in area of high lead content. Animals show hind limb ataxia, and there is often an associated osteoporosis with bone fractures. Diagnosis is best done by sending a piece of kidney from an affected animal for lead assay.

If animal shows visual deficits, postural deficits, circling or head aversion

Check for

- Space-occupying lesion in cerebral cortex (on opposite side from that showing deficits). The commonest causes are brain abscesses and *Taenia multiceps* (*Coenurus cerebralis*) cysts (coenurosis, 'gid', 'sturdy'). Rarely, a brain tumour may be the cause. Occasionally, vague neurological signs may be associated with a heavy infection of migrating *C. cerebralis* larvae soon after infection.

  Differentiation between these may be difficult unless there is a history of gid on the farm. Blood sampling may be helpful (a raised WBC count or crossover may indicate an abscess), and there may be response to vigorous antibiotic therapy in the case of an abscess. In advanced cases of gid there is often detectable skull softening present (but not necessarily on the side of the cyst).

Treatment of coenurosis (see also Appendix 8)
Neurological examination should identify the most likely site of the lesion, although if multiple cysts are present (uncommon but not unknown) the signs

will be confusing. Removal of a cyst from the cerebrum, under general anaesthesia, is worth attempting if it is economically worthwhile. The most reliable sign is visual deficit, which is on the opposite side from the cyst. The site for operation is just behind the horn or the depression where the horn would be (horns complicate access). The skin is incised and a trephine used to remove a piece of bone 1–1.5 cm in diameter. Great care needs to be taken, particularly in young animals as the bone may be very thin. Bulging of the underlying brain indicates raised intracranial pressure. The meninges are incised if they are still intact after trephining. Sometimes the cyst is immediately visible. If so, the thin translucent wall should be grasped with small forceps and then some of the fluid removed with a catheter and syringe before carefully removing the remainder of the cyst. If the cyst is not visible, a 14–16G catheter can be used to gently probe to locate the cyst. Care must be taken not to angle the catheter across the central division between the cerebral hemispheres as serious bleeding will result. Once the cyst has been located by clear fluid dripping from the catheter, negative pressure applied with a syringe attached to the catheter should enable the cyst lining to be trapped and grabbed with forceps, then removed as above. The wound is closed by suturing the skin. Supportive treatment should consist of intravenous steroid to reduce brain swelling, and antibiotic to guard against infection, both given preoperatively.

Prevention of coenurosis

Since the life cycle involves the dog as the definitive host, regular worming with praziquantel is necessary (as often as every 6 weeks) of sheep dogs and other dogs with access to the countryside. Dogs should not be allowed to scavenge dead sheep. Foxes may be implicated in transmission but are much less effective hosts for the tapeworm.

Adults (Figure 15.3)

Sudden onset, individual animals affected

If an adult ram in group of rams shows ataxia or paresis

Suspect

- Cervical trauma or concussion from fighting. See above.

If animal shows signs of space-occupying lesion in cerebrum or cerebellum

Suspect

- 'Gid' (rarely affects those over 2 years old) or abscess. See above.

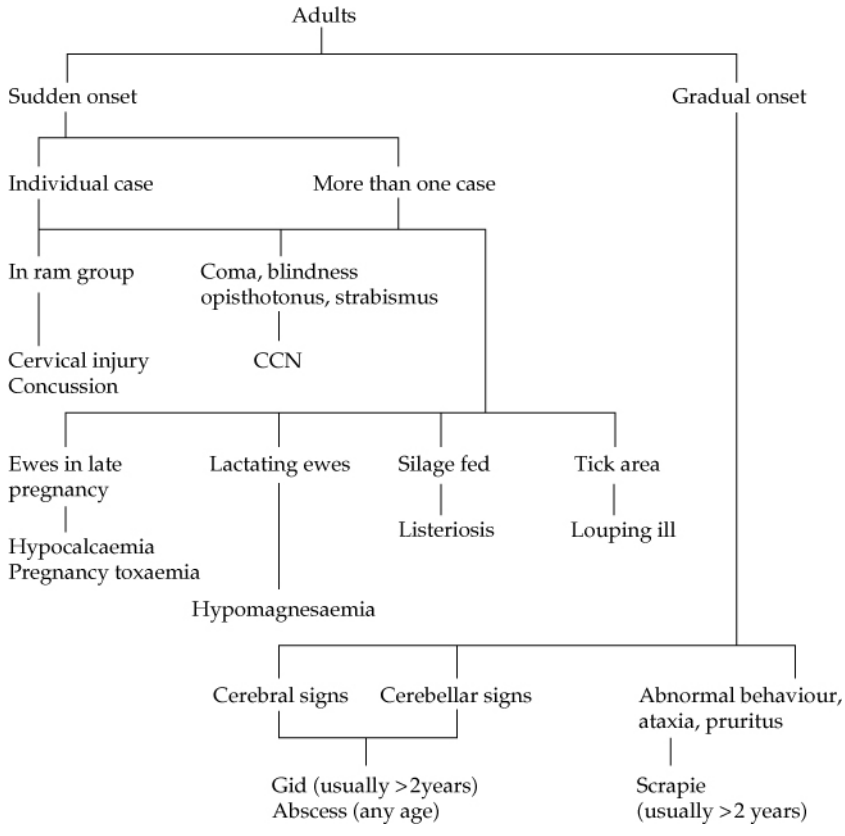


Figure 15.3 Diagnosis of neurological disease in adult sheep.

If animal shows collapse, blindness, strabismus and opisthotonus

Suspect

- CCN. See above.

Sudden onset, many animals affected

If ewes are in late pregnancy

Suspect

- Hypocalcaemia – animals show incoordination followed by recumbency and death within a few hours. Response to treatment is the best confirmation of diagnosis, but take clotted blood sample pretreatment in case of no response. Hypocalcaemia often follows stress such as gathering, housing, vaccination, bad weather. It may co-exist with pregnancy toxaemia.

- Listeriosis – if recumbent animal does not respond to treatment with calcium, reassess to see if facial paralysis is present. See below.
- Pregnancy toxaemia – see below.

Treatment of hypocalcaemia

Straightforward cases respond quickly to intravenous calcium administration (within a few minutes). About 25 ml calcium 20% should be given slowly intravenously and a further 60–100 ml given subcutaneously. If there is no response to treatment reassess diagnosis and send pretreatment blood sample for analysis.

Prevention of hypocalcaemia

As this condition is often triggered by stress of movement, handling or food deprivation in late pregnancy, animals should be handled carefully for routine procedures such as vaccination. Carrying out too many procedures at the same handling should be avoided, e.g vaccination, worming, fluking, coppering. Vaccination against clostridial diseases is now recommended at 4–6 weeks before lambing because of potential stress problems. Management by keeping calcium intake low to promote mobilisation from bone, as done in cattle, is rarely practical in sheep. See also Appendix 3.

If ewes are in poor condition in late pregnancy

Suspect

- Pregnancy toxaemia – depression, loss of appetite and apparent blindness are followed by recumbency within a few days. Clinical signs, body condition and probable high fetal load all point to the diagnosis which may be confirmed by showing ketones in urine, high BHB and low glucose concentrations in blood samples (take oxalate fluoride (OxF) and clotted samples). Liver enzymes are also raised. If the animal dies, PME shows a pale fatty liver.

Treatment of pregnancy toxaemia

Unless carried out early, treatment is unrewarding as permanent brain damage occurs. Farmers should be given instructions to give an oral drench of propylene glycol (50–60 ml twice daily) or electrolyte/dextrose mixture as soon as a sheep is noticed to be off its food. Calcium 20% should also be given subcutaneously in case hypocalcaemia is also present. Good quality hay, green food and palatable concentrates should be offered. If the appetite has not returned within 12 h, it is worth trying dextrose intravenously (this needs repeating every few hours). If there is still no response, induction of parturition can be tried by injecting 16 mg of betamethasone or dexamethasone, but this takes a minimum of 36 hours to work. The lambs can also be removed by caesarean operation. However, many cases end in failure with at minimum loss of lambs, often loss of ewe and lambs.

Prevention of pregnancy toxaemia
Correct nutrition in the last 2 months of pregnancy, together with scanning for fetal numbers, regular condition scoring, and separating into groups according to body condition and nutritional requirements, are all necessary features. See Appendix 3.

If ewe is lactating at grass, shows sudden collapse and fits

Suspect

- Hypomagnesaemia – treat as for hypocalcaemia with 40–50 ml calcium with magnesium mixture intravenously and 100 ml 25% magnesium sulphate subcutaneously. Consider supplementation of remainder of group with magnesium. See Chapter 22 and Appendix 3.

If animal shows visual deficits, postural deficits, circling or head aversion

Check for

- Space-occupying lesion in cerebral cortex (on opposite side from that showing deficits). The commonest causes are brain abscesses and *Taenia multiceps* (*Coenurus cerebralis*) cysts ('gid', 'sturdy'). Occasionally, vague neurological signs may be associated with a heavy infection of migrating *C. cerebralis* larvae soon after infection. See above.

If animals are silage fed, show profound depression, circling, recumbency, and facial paralysis with protrusion of tongue and drooling of saliva

Suspect

- Listeriosis – many affected animals die, in spite of treatment, within a few days. Diagnosis is by characteristic histology of brain, plus isolation of organism (this may take time as it is slow to grow). The organism is thought to gain access to the brain via wounds in the mouth, or at teething, and travels along the trigeminal nerve to the brain stem. Various other cranial nerve roots can become involved, causing a variety of deficits in addition to the characteristic facial nerve signs.

> Facial paralysis without depression indicates middle ear infection rather than listeriosis (see colour Figure 19).

Treatment of listeriosis
Treatment is often unrewarding. High doses of semi-synthetic penicillin or potentiated sulphonamide product plus an NSAID are worth trying, but success rate is low, particularly if the animal is already recumbent.

Prevention of listeriosis

The organism is widespread in the environment and in faeces. The disease is most commonly associated with silage feeding. Poor quality silage, soil contamination and poor fermentation (pH of good silage should be below 5) all allow survival of the organisms. Big bale silage is particularly implicated as it is difficult to achieve a low pH and bales are often punctured allowing secondary fermentation. Uneaten silage should be cleared daily to prevent spoilage. If there is a particular problem, check if sheep grazed silage fields before making – faecal excretion can contaminate grass.

- Although rare, the possibility of botulism should be borne in mind (badly made big bale silage containing decaying carcasses of birds or rodents may be responsible). Signs are muscular weakness progressing to flaccid paralysis.
- If in tick area, check for louping ill – see above.

Gradual onset of signs, slow progression, individuals affected

If animal shows visual deficits, postural deficits, circling or head aversion, or cerebellar signs

Check for

- 'Gid' – see above. Gid is rarely seen in animals more than 2 years old.

If animal shows incoordination, abnormal behaviour, loss of weight, excitement, excessive scratching, or any combination of these signs

Suspect

- Scrapie – this transmissible spongiform encephalopathy is notifiable. Cases deteriorate over a period of weeks or months. There is currently no commercial diagnostic test in the live animal, although the abnormal PrP can be detected in lymphoid tissue, and tests are likely to become available at some time as the result of related work on BSE (possibly by taking a biopsy of third eyelid). Positive diagnosis requires brain histology or detection of abnormal PrP in the brain by immunological methods. Scrapie is rare in animals less than 2 years old. If scrapie is suspected the DVM must be notified. The animal will be euthanased and samples taken for confirmation of diagnosis.

Control of scrapie

Susceptibility to scrapie and length of incubation period are genetically controlled and depend on the amino acids at certain crucial points (codons 136, 154 and 171) on the PrP gene which produces PrP protein. There are up to 15 different genotypes in some breeds, and breeds vary as to which are the most susceptible. The National Scrapie Plan (NSP) has been introduced to encourage

genotyping of rams and slaughter of the most susceptible types. Resistant rams (Type I) are homozygous ARR types. Type II rams are those carrying one ARR gene, provided the other gene is not VRQ which is the most susceptible type. Participating farms must agree to slaughter or castrate rams of undesirable genotypes. It is anticipated that participation in the NSP will become compulsory.

> It is important to impress on farmers that the scrapie testing scheme is testing for resistance or susceptibility to infection with scrapie, *not* for the presence or absence of the disease.

If animal shows dullness and twitching of muscles

Consider

- Uraemia – blood sample for urea and creatinine estimation.
- Sarcocyst infection – heavy infection occasionally causes vague neurological signs including above plus paresis of hind legs.

In a lactating ewe rearing twins or triplets, if function of both forelegs is lost with muscle atrophy

Suspect

'Kangaroo gait' - the cause of this peculiar condition is unknown but it is reversible, animals returning to normal after weaning.

If animal drags one hind leg

Suspect

- Visna (see maedi-visna, Chapter 21).

Gradual onset of signs, slow progression, many animals affected

If animals are grazing ryegrass pasture

Consider

- Mycotoxicosis ('migram', ryegrass staggers) – animals show tremors, knuckling of extremities and collapse, but recover after rest,. This results from a toxin produced in fungi which parasitise the seeds of ryegrass. Animals mostly recover if removed to safer grazing.

The recumbent ewe

This is perhaps one of the most common problems referred to the clinician over the telephone and one for which 'diagnosis at a distance' is almost invariably required on a basis of cost-effectiveness. Whereas the 'downer cow' will merit a visit and full diagnosis, an individual ewe will not. If a number of ewes are affected, then direct examination may be possible, but with the exception of hypocalcaemia, this is rarely the case. The clinician must, therefore, have a list of questions in mind which should help to lead to a correct diagnosis and treatment. Sadly, many farmers tend to administer some form of treatment to these cases and only consult when their own ministrations have failed; in many cases it may then be too late for the expected miracle cure by the vet to occur. The issue of 'animals under one's care' must always be kept in mind in dealing with these cases. Animals suffering prolonged recumbency often turn into welfare cases unless nursing is of a high standard. The clinician should, therefore, encourage early telephone consultation, when a satisfactory outcome for all parties is more likely. Rams too may, of course, become recumbent, but as their value is greater than individual ewes, requests for a visit are more likely. Since many problems resulting in recumbency are neurological in nature, the clinician is also referred to other sections of the chapter, in particular the routine for a full neurological examination.

History and appearance

The following list includes a number of important aspects of the case which may help the clinician to form a reasonable opinion about a possible diagnosis and treatment:

- Age.
- Breed.
- Number affected.
- Speed of onset, progression and duration.
- Time of year.
- Pregnant, recently lambed, lactating or nonpregnant.
- If pregnant, when due and how many lambs expected.
- If lambed, how long ago, any assistance.
- If lactating, when lambed and how many lambs rearing.
- Housed or outside.
- Body condition (has the farmer actually felt?).
- Is it eating, can it eat, is it drooling.
- What is being fed, feed availability, quality and feeding routine.
- Group size.
- Any recent handling or treatments.
- Can animal stand at all.
- Appearance – alert, dull, unresponsive, excitable.
- Sitting up or flat out.

- Head position.
- Can it see, can it blink.
- Are the legs stiffly held out or easy to move.
- Any peculiar behaviours seen.
- Any treatment already given.

Based on the answers to these questions and the information in Figure 15.4 it should, in the majority of cases, be possible to form a reasonable idea of a likely diagnosis and to advise on treatment or other appropriate action.

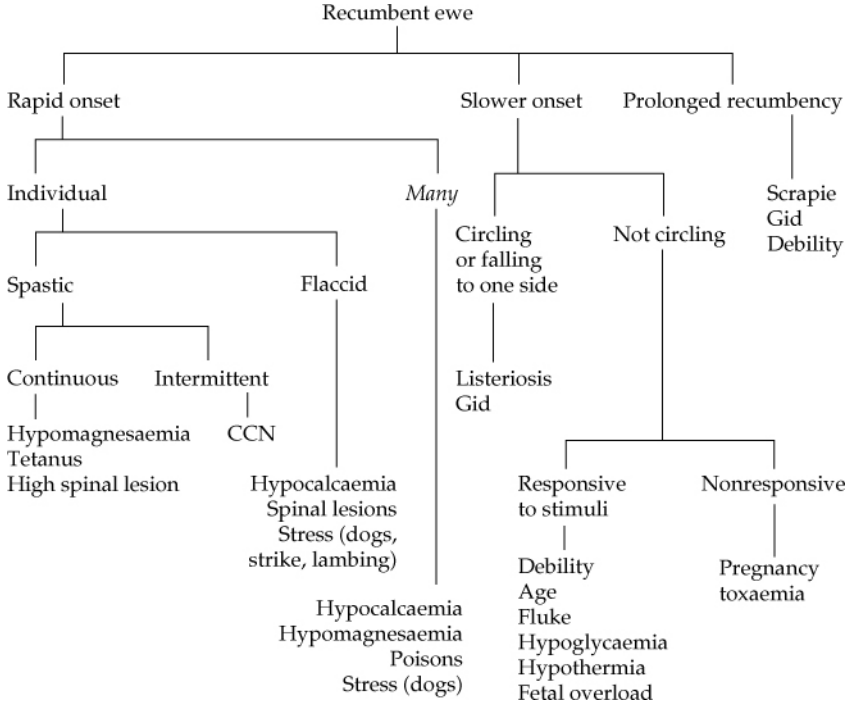


Figure 15.4 Diagnosis of recumbency in ewes.

16 Eye disorders/visual defects

Defects in vision can arise from a wide variety of conditions, ranging from corneal disease at one extreme to a space-occupying lesion in the visual cortex at the other. Diagnosis of the cause of impaired vision must start with the establishment of the site and nature of such impairment. In the case of corneal involvement, this may be obvious. In other cases, however, where the lesion is not obvious, it is essential that a full history is obtained and clinical examination is carried out.

Age, number affected, stage of reproductive cycle, general behaviour patterns, response to stimuli, and body score in comparison with contemporaries, may all give vital indications of the presence of a disease which has its origins centrally, rather than in the orbit, but manifests itself primarily as a defect in vision.

The clinical examination should begin with an examination of the eyelids and superficial structures of the eye (Figure 16.1). Use of an ophthalmoscope will assist in the diagnosis of bright blindness (retinal atrophy), but oedema of the optic disc does not seem to be a feature of increased intracranial pressure in the sheep.

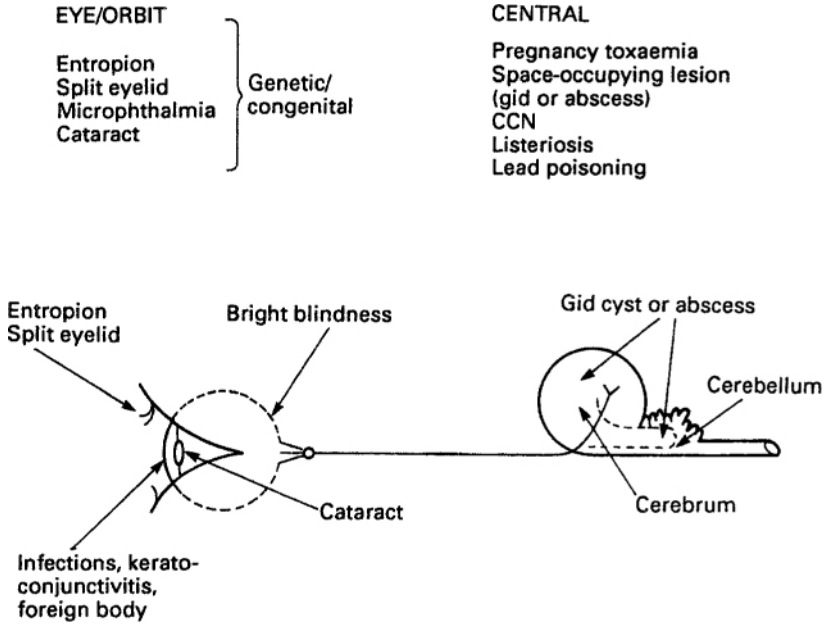


Figure 16.1 Site of lesion in diseases affecting the eye or vision.

The integrity of the visual pathways can be tested with the menace (blink) test. Where lesions causing visual defects are present in the brain, other neurological signs or deficits will usually be present. See Chapter 15 for more details.

The age of the patient(s), whether one eye or both affected, and number of animals involved, will be the first guide to a possible diagnosis.

Neonatal lambs

Check for genetic or congenital factors

- Entropion – this leads to a secondary keratitis if treatment is delayed. The condition now affects many breeds and crosses.

> Many cases of young lambs which are presented by the farmer as suffering from 'New Forest disease' will in fact be corneal infection or ulceration as a result of undetected entropion. This disease has important welfare aspects if neglected.

- Microphthalmia – vitamin A deficiency or genetic defect in Texels.
- Upper eyelid defect ('split eyelid') associated with four horn gene – seen in some rare breed units (Hebridean, Manx Loghtan and Jacob breeds).

Treatment of entropion

If detected early and of mild degree, manual correction together with topical application of ophthalmic ointment may be sufficient. If detection is delayed or manual correction is not effective, the simplest option, after rolling the eyelid out, is to inject 0.5–1 ml of fluid into the lower eyelid. An antibiotic suspension, such as penicillin or liquid paraffin, is commonly used. Alternatives involve making a 'tuck' of excess skin in the lower lid by inserting a small vertical mattress suture of dissolvable material, or using a Michel clip. Application of local anaesthetic to the cornea will prevent retraction of the eyeball and assist in resolution. See also Appendix 8.

> In flocks affected with entropion an 'eyelid' check of newborn lambs will allow early detection and successful manual correction in most cases.

Prevention of entropion

This is, at least in part, a genetic condition. If a problem appears in a previously unaffected flock, a change of ram is indicated. However, the condition is so widespread in some breeds that freedom from the defect may not be guaranteed even if new blood is introduced.

Check for infectious or traumatic causes

- Parturient trauma – head swollen with oedema of eyelids, use of fingers or eye hooks for delivery.
- Border disease if cataract is present.
- Infectious disease, e.g. neonatal septicaemia, joint ill, scouring, which can be followed by uveitis.
- Conjunctivitis – if entropion has been excluded, this is the other possible cause of excess lachrymation. Bacteria involved are probably the same as those affecting adults.

Growing lambs and adults

Large numbers or whole group affected, primary lesion is keratoconjunctivitis

Check for

- Infectious keratoconjunctivitis (pink eye). This is usually caused by *Mycoplasma conjunctivae* or less frequently, *Chlamydophila abortus.* Animals suffering from listeriosis often have injected scleral vessels, sometimes with ulceration of the cornea resulting from exposure because of lack of palpebral reflex. Other organisms have been isolated from the eyes of sheep, but their pathogenicity is not known. These include: *M. arginini, M. ovipneumoniae, Acholeplasma oculi, Branhamella ovis, Moraxella bovis.*

Identification of the causal organism in any particular outbreak is often difficult for several reasons:

(1) Both *Mycoplasma* and *Chlamydophila* require the use of special transport media – consult the laboratory for advice.
(2) Mycoplasmas can often be isolated from sheep with healthy eyes.
(3) Isolation of *Chlamydophila* requires a deep scraping of conjunctiva with a swab in order to obtain infected cells.
(4) Sampling from eyes which are apparently failing to respond to treatment is usually of little value because of residual effects of antibiotic.

M. conjunctivae is the organism that has been most frequently isolated in outbreaks in the UK, in contrast to some countries where *C. abortus* assumes a greater importance. The true incidence of the two organisms has been difficult to determine because of the problems with isolation and culture. However, diagnosis should include an attempt to identify the cause, as both the course of disease and response to treatment may vary. *C. abortus* as an eye infection is not a threat as a source of abortion, but may give a serological response.

Clinical disease often follows the introduction of new animals into a flock, but can exist as persistent infection within a flock due to the presence of carrier animals and variations in immune response. Young animals are generally less severely affected than older ones.

Clinically, an impression may be gained that there are two distinct patterns of disease:

- *C. abortus*
  bilateral involvement
  lymphoid follicles in conjunctival sac
  swelling of conjunctiva
  severe corneal opacity
  poor response to topical treatment
  relapses common even after parenteral antibiotic treatment.
- *M. conjunctivae*
  may be unilateral
  later development of lymphoid follicles
  corneal opacity less severe
  good response to topical treatment.

It must be emphasised, however, that similar lesions can be produced by both organisms experimentally, and none of the above signs are pathognomonic.

Stages in progression of keratoconjunctivitis

Grade 1 – scleral congestion, excess lachrymation, blepharospasm.
Grade 2 – corneal inflammation, vascularisation, opacity (pannus) at dorsal aspect.
Grade 3 – corneal ulceration.

Treatment of infectious keratoconjunctivitis

Mildly affected cases may self-cure without treatment. Where there is significant corneal involvement, individual treatment is necessary. Topical application of chlortetracycline ophthalmic ointment or powder 2–3 times daily, or subconjunctival injection with oxytetracycline, should be successful. If a large number of animals are affected, intramuscular injection of all affected animals with long-acting oxytetracycline may be the most practical option.

> *Mycoplasma* spp. are not susceptible to penicillins, so some products produced for treating ophthalmic disease (New Forest disease) in cattle will not be effective in sheep.

Neglected cases may develop corneal ulceration with eventual rupture of the eyeball. Protection of the cornea by suturing across the third eyelid may save the eye.

Prevention of infectious keratoconjunctivitis

Since the condition is sporadic and carriers occur, it is difficult to give any practical advice regarding prevention. Treatment of new introductions to the flock with antibiotics before mixing with resident animals has been suggested but is unlikely to be successful as it is not possible to totally sterilise the eye.

Individual animal affected, excess lachrymation, photophobia, often with corneal involvement

Check for

- Recent introduction of infectious keratoconjunctivitis, as above. Will spread rapidly to others.
- Foreign body in eye, particularly if housed. Foreign bodies may be difficult to identify as they rapidly become incorporated into the surface of the cornea by the inflammatory reaction.
- Height of hay racks – these may act as a source of hay seeds or dust.
- Self-mutilation, e.g. following photosensitisation or facial eczema.
- Secondary eye damage as a result of prolonged recumbency.
- Early stages of an outbreak of listeriosis – excess lachrymation and conjunctivitis may be seen. See Chapter 15 for more details.

Individual or small numbers affected, blindness with no eye lesions, other neurological signs present

Check for

- Gid or other space-occupying lesion in brain.
- CCN.
- Pregnancy toxaemia if in late pregnancy.

See Chapter 15 for further details.

Individual or small numbers affected, gradual onset of bilateral blindness, no corneal involvement, sheep have access to bracken

Check for

- Bright blindness (progressive retinal atrophy). This bilateral condition, caused by prolonged intake of toxic substances within bracken, rarely affects sheep under 2 years of age. Affected sheep show a high head carriage and high stepping gait. The eyes shine in poor light due to excessive reflection from the retina, and the pupils react poorly to light. Examination with an ophthalmoscope shows narrowing of the blood vessels on the retina, with no inflammatory reaction or opacity in the eye. There is no treatment for this condition.

Part of flock shows excess lachrymation, no eye lesions, ill thrift in ewes and lambs

Check for

- Cobalt deficiency – see Chapter 9.

17 Anaemia

Anaemia in sheep is often a sign of an underlying disease process rather than a primary condition, although there are exceptions to this general rule. The detection of anaemia during an examination should always alert the clinician to search for other signs of disease. Sudden onset of severe anaemia, for example the result of haemorrhage, will be easily recognised by pallor of the conjunctivae, third eyelid, mouth and vaginal mucous membranes and general weakness. If anaemia develops slowly, a fall to below 50% of normal blood parameters can occur without noticeable effect on the animal unless it is exerted. Changes in exercise tolerance, with increased heart and respiratory rates, may then become apparent (although these are difficult to interpret anyway as mentioned in the introductory chapter). Mild anaemia is often not noticed unless the animal is examined closely, and it may be necessary to compare with other animals or to wait for results from a blood sample to be certain if anaemia exists.

Confirmation of anaemia must be by haematological examination, with the finding of lower than normal values for red blood cells, haemoglobin concentration and packed cell volume.

There are three distinct mechanisms by which anaemia may arise: haemorrhagic, haemolytic and depression/hypoproliferative.

Haemorrhagic

Acute

Obstetric trauma (to ewe)
Obstetric trauma (to lamb)
Other trauma
Umbilical bleeding

Acute/chronic

Acute fluke
Coccidiosis
Haemonchosis

Chronic

External parasites
Chronic fluke

Haemolytic

(This may be accompanied by haematuria and followed by jaundice, see Chapter 18.)

Copper poisoning
Rape/kale poisoning
Nitrate poisoning
Bacillary haemoglobinuria (*Cl. novyi* type D)
Cl. perfringens type A
Leptospirosis

Depression/hypoproliferative

Cobalt deficiency
Copper deficiency
Chronic liver fluke
Chronic parasitic gastroenteritis
Cow colostrum induced anaemia (this is a combination of extravascular haemolysis and bone marrow depression)

> Iron deficiency does not occur in adult sheep, and although young lambs housed on a milk diet have been shown to have lower red cell parameters than lambs supplemented with iron, there was no significant effect on growth rates.

Classification of anaemia on morphological appearance of erythrocytes (Table 17.1)

Although this is done routinely in the investigation of anaemia in man and small animals, it is rarely applied to sheep, but may be of help where a diagnosis is not obvious.

Table 17.1 Classification of types of anaemia.

Shape	Hb content	Comment
Normocytic	Normochromic	Normal appearance, chronic PGE, cobalt deficiency
Normocytic	Hypochromic	Chronic blood loss, e.g. haemonchosis
Macrocytic	Normochromic	Cobalt deficiency
Macrocytic	Hypochromic	Recovery after blood loss, subacute/chronic fluke
Microcytic	Normochromic	
Microcytic	Hypochromic	Chronic blood loss, copper deficiency

Table 17.2 Investigation of anaemia.

Age group	Haematology (EDTA sample)	Biochemistry (serum or heparinised)	Faeces	PME samples
Lambs <3 weeks	PCV (Coombs test)			Pale carcass, watery blood, pale bone marrow indicates cow colostrum anaemia
Lambs <8 weeks	PCV		Coccidial oocysts	Intestinal smear for coccidia Liver lesions with *T. hydatigena*
Growing lambs	PCV	Co (vitamin B_{12}) Cu		Liver for Co, Cu
			Nematode eggs	Worm counts especially *Haemonchus* (abomasum and SI)
Adults	PCV, examine for haemolysis	GGT (chronic liver damage)	Fluke eggs	Liver (fluke)
			Nematode eggs	Worm counts especially *Haemonchus* (abomasum)
		AST, GLDH, SDH (acute liver damage)		Liver for Cu

Investigation of anaemia (Table 17.2)

Unless the source of blood loss is obvious, a search must be made for the underlying disease process. Except with trauma, most cases will affect a significant number of animals and may come to light as a result of investigation into poor growth rates or adult weight loss. It may be helpful to sacrifice an affected animal and carry out a full postmortem examination if the cause is not obvious. Aspects of the history which will be relevant include:

- Age and number of affected animals.
- Type of pasture.
- Recent pasture improvements.
- Concentrate feeding (especially cattle or pig food).
- Access to cattle minerals.
- Parasite control measures.

Sample taking

Where a group of animals is involved, at least six samples should be taken for any haematological, biochemical or parasitological examination.

For haematological examination, EDTA is the anticoagulant of choice. Heparin and OxF can be used for red cell counts but are not suitable for WBC counts.

Cobalt (B_{12}) and copper estimation require a serum or heparinised blood sample.

Normal range of red cell values

RBC	9–15 × 10^{12}/l
Hb	9–15g/dl
PCV	0.27–0.45
Reticulocytes	0%
MCV	28–40 fl
MCHC	31–34%

The commonest causes of anaemia according to age group are given below.

Young lambs (<4 weeks)

Check for

- Haemorrhage – external, e.g. from umbilical vessel, internal, e.g. liver rupture or other obstetric trauma, predation.
- Cow colostrum-induced anaemia. A history of feeding cow colostrum within the first 24 h, with anaemia developing at 6–14 days is virtually diagnostic. Packed cell volume (PCV) is useful and rapid to screen others exposed. PCV <0.20 is significant, although clinical illness may not be seen until <0.12. Can be confirmed by direct Coombs test which shows presence of bovine IgG on erythrocytes in most cases. PME shows pale carcass (see colour Figure 21) with small amount of watery blood, pale creamy bone marrow. Thrombocytopaenia has also resulted from feeding cow colostrum.
- Migrating *Taenia hydatigena* larvae in liver may cause acute haemorrhage. Occasionally seen in orphan lambs reared in close proximity to farm dogs.
- Iron deficiency has been reported in housed growing lambs.

Treatment of cow colostrum induced anaemia

If individual valuable lambs are involved it is worth considering giving a blood transfusion. If several commercial lambs are affected it is worth giving blood by intraperitoneal injection, since red cells will reach the circulation via lymphatics. About 10 ml/kg of blood (with anticoagulant added) taken preferably from the dam can be given into the peritoneal cavity by injecting through the abdominal wall just behind and to one side of the navel (as for giving dextrose

to hypothermic lambs). This should be done in a sterile manner. In addition lambs should be given long-acting steroids (e.g. 0.5 ml/10 kg dexamethasone phenylpropionate/sodium phosphate), iron dextran and antibiotic. Some affected lambs will die in spite of treatment.

Prevention of cow colostrum induced anaemia
In the absence of commercial tests to identify cows producing suspect colostrum, the most practical method is to mix colostrum from at least four cows in the hope that any anti-sheep antibodies will be diluted. Alternatively, careful records should be kept to identify cows whose colostrum has been safely fed to lambs in previous years.

Growing lambs

Check for

- Cobalt deficiency (blood biochemistry).
- Copper deficiency (blood biochemistry).
- *Haemonchus* infection (no scouring, bottle jaw) – faeces sample for nematode eggs, but species cannot be differentiated.
- PGE (concurrent scouring) – faeces sample as above.
- Coccidiosis (blood in faeces, may occur before oocyst production) – may require PME to confirm.
- Acute fluke – recent grazing history, serum or plasma sample for liver enzymes. Aspartate aminotransferase (AST), glutamate dehydrogenase (GLDH) and sorbitol dehydrogenase (SDH) indicate acute liver damage. PME to confirm.

Treatment and prevention
These will depend on diagnosis. See relevant chapters for details.

Adults

Check for

- Acute/chronic fluke.
- *Haemonchus* infection.
- Acute copper poisoning which occurs within a few days of administration of copper injection. Jaundice may not be a feature. See Chapter 18 for treatment.
- Rape/kale poisoning (less susceptible than cattle). Sheep should be introduced gradually to any brassica crop. It is possible to analyse samples for content of the factor responsible for causing haemolytic anaemia.
- Lice infestation
- Nitrate poisoning – nitrate is nontoxic but is converted to nitrite in rumen.

This is absorbed and produces methaemoglobinaemia (chocolate brown blood), with methaemoglobinuria. This can be treated by intravenous injection of 10 ml/kg of 4% methylene blue solution.

Treatment and prevention

These will depend on diagnosis. See relevant chapters for other information.

18 Jaundice

As with anaemia, jaundice is a sign of a disease process and not a primary condition itself. It will be vital evidence of a pathological condition such as a haemolytic crisis or liver dysfunction, and will be identified during clinical or postmortem examination.

Jaundice arises from an increase in the bilirubin content of the blood, giving rise clinically to yellow pigmentation of the mucous membranes and sclera (see Figure 18.1). At PME there is yellowing of all the body tissues, particularly the liver.

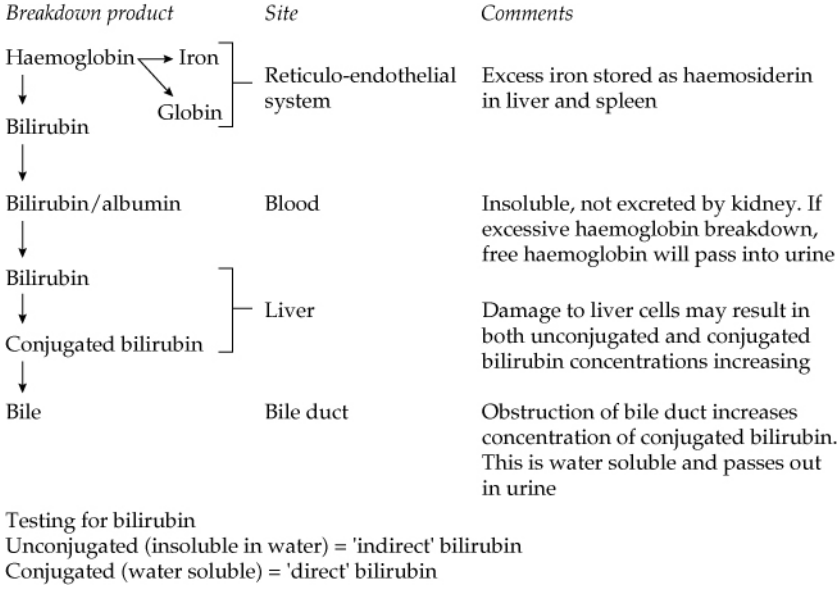


Figure 18.1 Breakdown of haemoglobin.

> Some sheep possess a natural yellow pigmentation of the body fat, and this should not be mistaken for jaundice – check the mucous membranes to differentiate.

Jaundice usually is caused by one of three mechanisms: excess breakdown of erythrocytes by haemolysis, liver dysfunction or bile duct obstruction.

Excess breakdown of erythrocytes by haemolysis (prehepatic jaundice)

There is no impairment to bile flow, and it is the unconjugated (indirect) bilirubin content of the blood which is raised. Possible causes are:

- Copper poisoning.
- Kale poisoning.
- Onion poisoning.
- *Cl. perfringens* type A.
- *Cl. novyi* type D.
- *Leptospira pomona*.

See also Chapter 17 on haemolytic anaemia.

Liver dysfunction

In this type, the conjugated (direct) and unconjugated (indirect) bilirubin content of the blood are raised, because of inability of damaged liver cells to conjugate, and interference with bile excretion. Possible causes are:

- Photosensitisation.
- (Chronic fluke).
- Infections of the liver.

Bile duct obstruction (posthepatic jaundice)

In this type the conjugated (direct) bilirubin content of the blood is raised because of back-pressure from blocked bile duct. Possible causes are:

- (Chronic fluke).
- Tumours.
- Other obstructive conditions.

Investigation of jaundice

Important aspects of history taking include the following:

- Incidence (single case or many affected).
- Age.
- Feeding/grazing.
- Concentrate feeding.
- Access to minerals.
- Parasite status.
- Local knowledge of poisonous plants.

> Sheep with copper poisoning may be found dead, and it is important that all contacts should be thoroughly examined. Severe anaemia may be present in the early stage of the disease before the onset of jaundice.

Blood sampling

- EDTA sample for haematology and bilirubin concentrations. Normal values for bilirubin:
  - Total <10.0 mol/l
  - Direct <1.7 mol/l
- Serum or plasma (heparinised) sample is very helpful. The most useful tests are:
  - for acute liver disease – AST, GLDH, SDH (this is an accurate indicator of acute liver damage, but must be assayed within 4 h of sampling)
  - for chronic liver disease – gamma glutamyl transferase (GGT), total protein and albumin content, phylloerythrin content (in acute phase of photosensitisation only).
- Heparinised blood or serum for copper estimation.

Postmortem material

- Liver and kidney (100 g) for copper content.

The most common causes of jaundice can be linked to particular age groups.

Young lambs

Check for

- Cow colostrum feeding (see Chapter 17). Although there is excessive red cell destruction, jaundice is not usually a feature of this condition.
- Possibility of leptospirosis.

Growing and adult sheep

Check for

- Chronic copper poisoning which is the result of prolonged efficient uptake of copper from the diet (see also Appendices 3 and 6). Check for source of excess copper – cattle concentrates, cattle minerals, grass spread with pig slurry. PME if deaths occur, blood Cu in contacts, liver enzymes AST, GLDH, SDH.

There is considerable breed and strain variation in sensitivity to copper in the diet. Diets which cause toxicity in some breeds may lead to swayback lambs in others, e.g. the Texel is very susceptible to copper poisoning, the Scottish Blackface may produce swayback lambs on a similar diet. An extreme example of susceptibility to copper poisoning is the North Ronaldsay breed, which has developed a highly efficient system for obtaining copper from its normal diet of seaweed, and is extremely difficult to keep alive on normal concentrates.

- Rape/kale feeding.
- Severe fluke. Jaundice is rarely a feature of chronic fluke even in the presence of high fluke numbers and severe liver damage.
- Recent drug administration.
- Photosensitisation – oedema and irritation of head and ears, followed by drying and crusting of skin. See Chapter 20.
- Other rare causes of jaundice such as tumours are likely to be found at PME only.

Treatment of copper poisoning

This may be unrewarding for severely affected animals. Even those apparently less severely affected may never regain their former productivity. Injection of ammonium tetrathiomolybdate (3.4 mg/kg every second day on three occasions) may help. Oral treatment with 100 mg ammonium molybdate daily is worth considering for contacts. All sources of concentrates should be removed.

Prevention of copper poisoning

Supplementation with copper should only be carried out where a known problem exists, or after sampling a representative number of animals. The most important influence on copper uptake is the amount of molybdenum in the diet. In winter, sheep grazing bare fields ingest considerable amounts of soil, which increases molybdenum intake. If there is prolonged snow cover, the soil intake is reduced, therefore more copper is absorbed. In cases where flocks previously outwintered are housed, particular care needs to be taken over copper supplementation, since copper absorption from the diet increases in two ways – because soil ingestion does not take place and from the natural copper content of concentrate feeds.

Pedigree show animals may be particularly at risk because of prolonged feeding of concentrates, even though no copper is added, so the diet needs to be carefully assessed and regular periods given at grass with little or no supplementation if at all possible.

Most feed manufacturers add molybdenum to sheep diets to try to guard against copper toxicity. See also Appendices 3 and 6.

19 Abdominal distension and abdominal pain

Although abdominal distension and abdominal pain can, and frequently do, exist as separate clinical entities, they may be linked in aetiology. Factors causing distension may also cause pain, or pain may result from the distension.

There is one important cause of abdominal distension which the client may not recognise as abnormal: extreme fetal overload, which may nevertheless place maternal survival in jeopardy. The manipulation of nutrition to maximise reproductive potential, or the use of artificial means to increase litter size, in conjunction with unchanged dependence on a ruminant digestive system, have produced a situation in some animals where late pregnancy is a disease. Additionally, old highly prolific ewes commonly have enlarged abdomens as a result of loss of muscle tone.

Diagnosis will be made easier by consideration of the sex, age and reproductive status of the patient. In addition, the speed of onset in the case of abdominal distension will be of great significance.

Abdominal distension (Figure 19.1)

At birth

If the lamb is delivered with a distended abdomen check

- Incidence, breed, cross – possible genetic origin.
- For other abnormalities – achondroplasia, kidney abnormality.
- For other evidence of abortion or stillbirths – premature lambs often give the appearance of a distended or 'watery' belly.

Treatment of these lambs, if born alive, will not be appropriate. They should be euthanased on welfare grounds.

Neonatal lamb

If rapid onset check for

- High alimentary obstruction – pyloric stenosis, pyloric obstruction (milk curd), gastric torsion.
- Watery mouth – excess salivation, depression, loss of appetite (see Chapter 8).
- Castration technique – incorrect ring placement causing urethral occlusion.

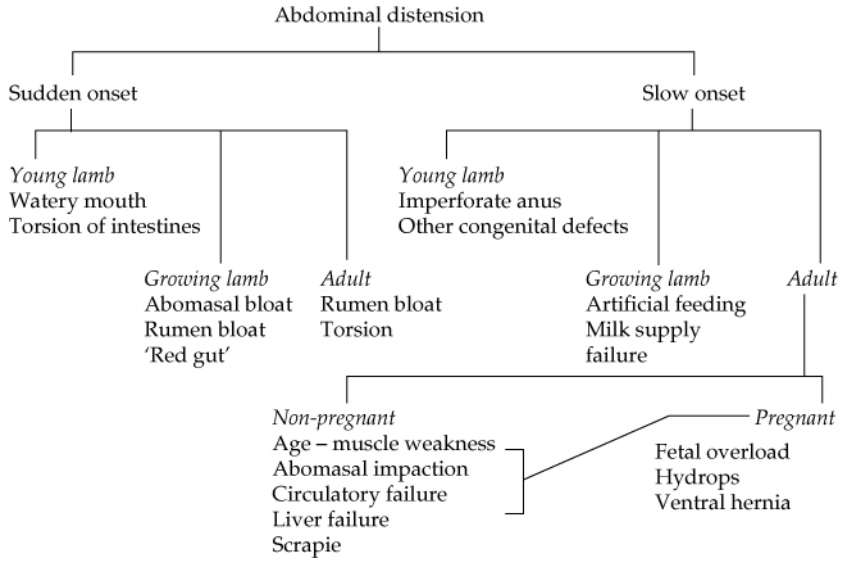


Figure 19.1 Abdominal distension.

If slow onset check for

- Low alimentary obstruction, imperforate anus, or other congenital alimentary defect.
- Ascites (paracentesis) – congenital heart defect or kidney defect.

Treatment

Treatment, if appropriate, will depend on diagnosis.

Growing lambs

If rapid onset

Check for

- Abomasal bloat – almost invariably in artificially milk-fed lambs on infrequent large feeds. These will either be 'pets' or in multiple units fed on milk substitutes with concentrates available. Access to grass appears to be an aggravating factor. It may be possible to prevent further cases by the addition of 5 ml/litre formalin to milk replacer.
- 'Red gut' (torsion of intestines) – an abdominal catastrophe and the lamb is likely to be found dead. If seen alive, there is severe abdominal disten-

sion and pain with rapid deterioration and death within a short time. This condition is thought to be caused by rapid throughput of highly digestible food, with excess gas production in the intestines. Instability develops, followed by torsion of the intestines and occlusion of the anterior mesenteric artery. Changing to a diet with more fibre may control the problem.

- True rumen bloat (pass stomach tube) – may be due to sudden change of diet, low fibre intake, high clover content. Increase fibre content of diet to control.

If slow onset

Check for

- Weaning too early and provision of inadequate or inappropriate food – lambs have 'potbellied' appearance.
- Evidence of wool/hair ball, or excess fibre in abomasum, by palpation – may cause intermittent obstruction and may be aggravated by feeding with a stomach tube.
- 'Grass scours' – premature rumen dependence and development, due to milk failure or mismothering after gathering.
- Concurrent anaemia and debility – parasitism.

Treatment

Lambs affected with any of these conditions may never recover full productivity. Worm and put on clean pasture, with access to good quality creep feed (introduce gradually).

Prevention

Correct management of ewes and lambs in the first two months after lambing, with provision of creep feed if grass supply is limited, and adequate parasite control should avoid significant numbers of these poorly thriving lambs. See Chapters 9 and 10.

Adult (Figure 19.2)

If rapid onset

Check for

- Rumen bloat caused by diet change, low fibre intake, clover intake. Site of distension is high in left flank.
- Abdominal catastrophe, e.g. intestinal torsion. Whole abdomen is distended and tight, animal has rapid pulse, shows rapid deterioration and death.

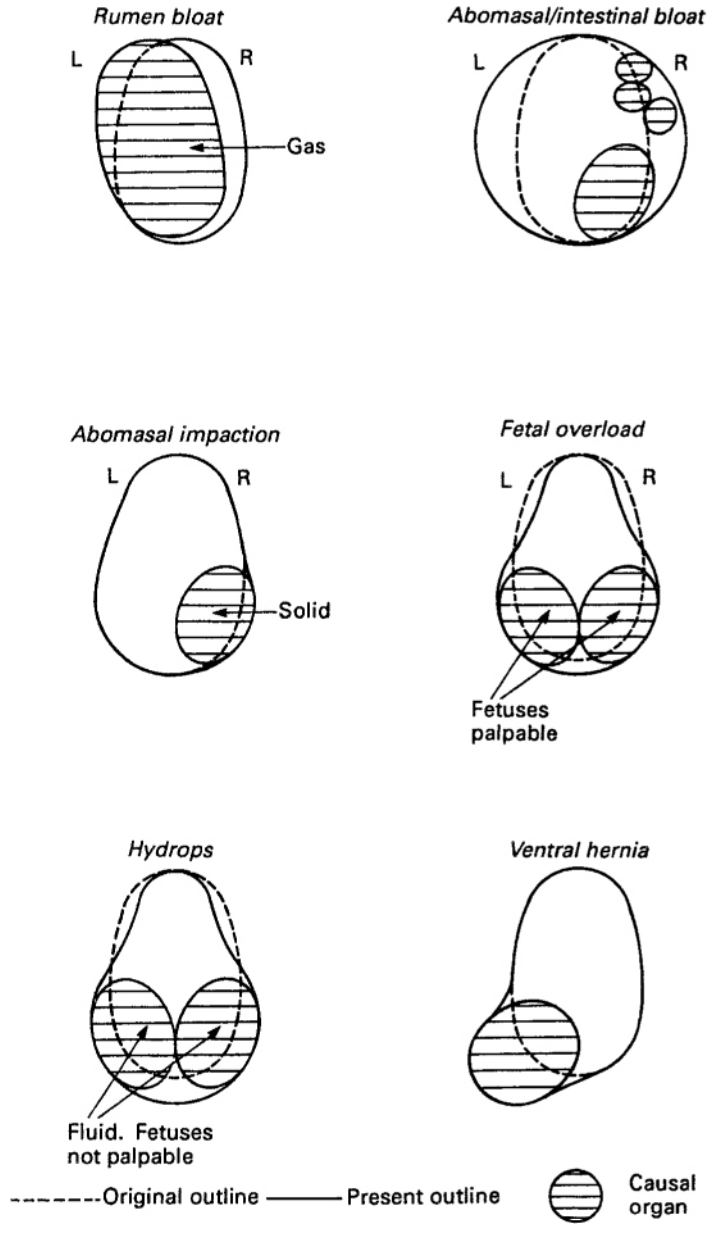


Figure 19.2 Shape of abdomen in cross-section as aid to diagnosis.

If slow onset

Check

- Stage of pregnancy – fetal overload (see colour Figure 22), poor body condition, fetuses palpable.
- Age and reproductive history – muscle stretch after multiple pregnancies.
- For hydrops amnii/allantois – bilateral distension, fetuses not palpable, may be fluid wave detectable. Scanning may be helpful.
- For abdominal wall trauma – ventral hernia.
- Food quality – low digestibility of fibre may cause excess rumen fill.
- For abomasal impaction (see colour Figure 23) – palpable in low right abdomen. Results from inadequate rumination (molar tooth disease), or part of 'vagal indigestion' syndrome. Has also been seen in scrapie cases.
- Heart – circulatory failure may give ascites (do paracentesis, avoiding rumen area).
- For dependent oedema – chronic fluke, mastitis, debility.
- Other causes such as neoplasms are unlikely to be diagnosed until PME.

Treatment and prevention

If appropriate, these will depend on diagnosis.

Abdominal pain

This will accompany some of the conditions indicated in the previous section but may occur without the presence of distension.

It is essential that the clinician recognises evidence of pain in sheep of all ages. This is not easy since its manifestation will vary from vocalisation and convulsions in the very young with acute abdominal pain, to restlessness and frequent alternate lying and standing, or intermittent teeth grinding shown, for example, with urolithiasis. In the latter stages of most abdominal catastrophes signs of acute pain will disappear to be followed by recumbency and death.

Neonatal lambs

> Some young lambs show apparent pain on defaecation even when no abnormality exists. This usually ceases after a few days.

Check for

- Intermittent pyloric obstruction by palpation of abdomen. If obstruction is intermittent, the abomasum will empty between feeds and no distension will be evident.
- Torsion of intestines (rapid deterioration and death).

With imperforate anus, defaecation attempts and pain are usually absent.

Growing lamb

Check for

- Coccidiosis – discomfort or pain may be noticeable before shedding of oocysts. See Chapter 10.
- Intestinal obstruction, e.g. intussusception (palpate). Pain may be severe (colic), followed by temporary respite indicating onset of intestinal necrosis.
- Urolithiasis if male (especially castrated). See Chapter 11.

If recently castrated male

Check for

- Poor technique or urethral obstruction.

Adult female

Check for

- Evidence of abortion or parturition.
- Torsion of uterus if parturition appears imminent but no progress (vaginal examination may show characteristic twists, but torsion anterior to cervix may not be detectable).
- Postparturient infection/trauma.

See Chapters 3 and 4.

Adult male

Check for

- Urinary obstruction – see Chapter 11.
- Inguinal herniation – not treatable and may be a hereditary component.
- Traumatic balanitis.

20 Wool loss and skin lesions

The clinician must keep in mind that hair and wool are products of actively growing cells, even though the visible external structures are 'biologically' dead. Any nutritional deprivation, systemic disease or stress affecting the function of the body as a whole is likely to have a direct effect on these structures, particularly on wool production, though there may be a time lag in the appearance of such changes. Severe illness or debility will soon lead to weakening of the wool fibres or to shedding of the fleece, which is hastened by using the wool to catch or restrain the animal. In animals suffering more chronic illness or under-nutrition, the effects may appear as thinning of the fibres, which can be seen macroscopically or can be detected by applying tension to the staple which will break at any weak point. This weakness is referred to in the wool trade as 'tenderness'. Normal healthy wool has a very high tensile strength.

In addition to this loss of wool as a result of acute or chronic illness, it should be recognised that seasonal partial or complete loss occurs in some breeds. For example, primitive sheep such as the Soay shed or moult the fleece, and some longwool breeds and crosses, particularly older animals, may lose wool along the spine in the late spring. Wool can also be mechanically removed by young lambs which often jump on to the backs of the ewes when playing.

These types of wool loss or poor growth are additional to and separate from the specific skin diseases which may themselves cause wool loss by affecting the follicles, or as a result of self-inflicted damage because of pruritus.

The vast majority of cases of skin lesions or wool loss will be seen in grazing lambs or adult sheep and will be secondary to nutritional, infectious or parasitic disease, but there are a small number of conditions which will be apparent at or soon after birth.

Neonatal and very young lambs

Poor or underdeveloped fleece

This is a sign of prematurity.

Check for

- Infectious causes of abortion if several lambs are affected (see Chapter 3).

Abnormally hairy and/or pigmented fleece

Check for

- Border disease – affected lambs show abnormal number of 'halo hairs' (long kempy fibres), often in conjunction with neurological signs ('hairy shakers'). Such lambs are virus positive, and were exposed to infection *in utero* before about day 85 of gestation. Abnormal brown or black pigmentation in normally unpigmented breeds may also indicate the presence of border disease.

> Normal lambs from Welsh ewes or crosses often show some pigmentation which fades with increasing age. The birth coat may also show marked variations, with patches of hairy and nonhairy coat on the same animal. Border disease should not be diagnosed in such animals without further supporting evidence, e.g. neurological signs, positive serology, virus isolation.

Proliferative lesions on lips and/or inside mouth

Check for

- Orf (see colour Figure 28) – this is particularly common in groups of artificially reared lambs and can appear within a few days of birth. Check teat hygiene, although if lambs are being fed with a multiteat unit it is almost impossible to prevent spread through the whole group. If lambs are sucking the ewe, check for teat lesions – see Chapter 7.

> Take care in handling – orf is a zoonosis.

Loss of hair and horn, recumbency

Check for

- 'Redfoot' – this hereditary condition affects Scottish Blackface sheep and their crosses, and is seen within the first few weeks of life. It is progressive, with ulceration of oral mucous membranes and cornea, with death resulting from infection or starvation. See Chapter 14.

Loss of wool, no systemic illness

This may occasionally result from an allergy to artificial milk, or possibly, rarely, to the ewe's milk.

Grazing lambs and adults

Although the client is likely to call for help on the basis of 'wool loss' or 'skin lesions', this will usually be accompanied by complaints of 'scratching' or 'itching', or comment that these features are absent. The number of animals affected will also be significant. The delineation into these groups may not always be clear cut, however, and no diagnosis should be excluded on the presence or absence of pruritus alone (see Table 20.1 and Figure 20.1).

Table 20.1 Site and degree of pruritus as aid to diagnosis.

		Head	Legs	Body
No pruritus	Individuals affected	*Actinobacillus* Clostridial infection		Season Natural shedding Debility (wool break) Fleece rot
	Many affected	CLA Orf Ringworm	Orf	Season Natural shedding Breed Woolslip Ringworm CLA
Some pruritus	Individuals or many affected	*Dermatophilus* Staphylococcal dermatitis Photosensitisation	*Dermatophilus* Strawberry footrot	*Dermatophilus* Keds Ticks Forage mites
Severe pruritus	Individuals affected Many affected (transient)	Scrapie Head fly Midge worry	 Midge worry	Scrapie Blowfly strike
	Many affected	Head fly Ear mites – (*Psoroptes*) Lice (sucking) Midge allergy	*Chorioptes* Lice (sucking) Midge allergy	Sheep scab Lice (chewing) Blowfly strike Midge allergy (belly only)

Individual animals, nonwoolled areas affected, lesions generally nonpruritic

If the client is observant, the first case of an infectious condition may be noticed. Alternatively, some conditions of low pathogenicity may be confined to a single case.

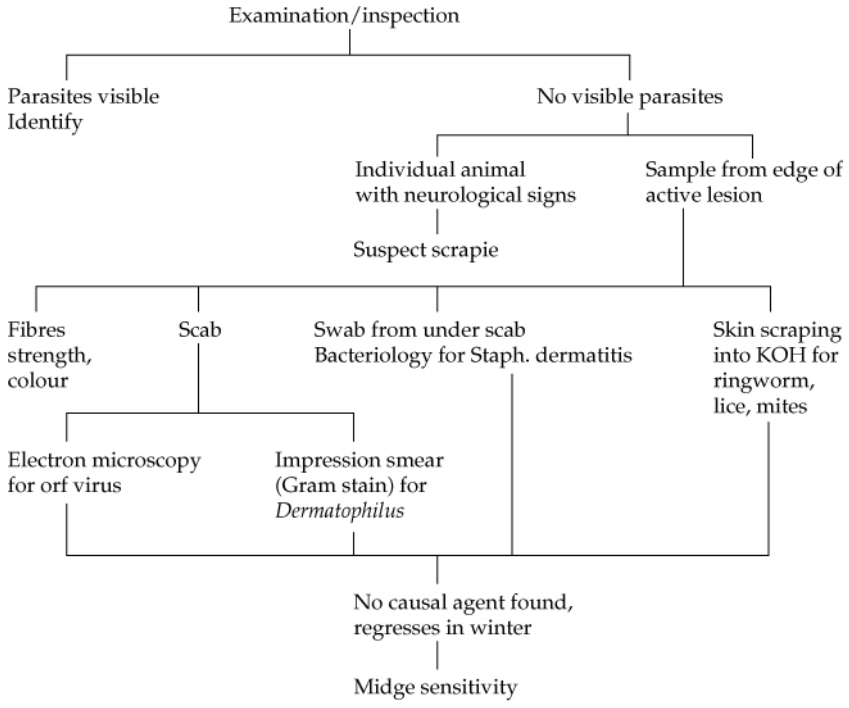


Figure 20.1 Stages in diagnosis of pruritic skin lesions and wool loss.

Abscesses on face and/or neck

Check for

- Caseous lymphadenitis – this infection may be seen in individual animals, e.g. bought-in rams, but is increasingly likely to occur as a flock problem – see below.
- Actinobacillosis – multiple fibrosed nodules, some of which may discharge pus, are present in the subcutaneous tissue, often forming a chain along the course of the lymphatics of the head and neck. This disease occurs sporadically and needs to be differentiated from caseous lymphadenitis since it will respond to treatment with a suitable antibiotic, such as a penicillin/streptomycin mixture.

Pustules in hair follicles on lips, muzzle or perineum of lambs, or on the udder of lactating ewes

Check for

- Staphylococcal folliculitis – scabs may form, but these are insignificant compared with lesions of orf or staphylococcal dermatitis. The conditions

Table 20.2 Diagnostic aids for wool loss/skin lesions.

	Gross pathology	Site	Microbiology	Parasitology
Staphylococcal dermatitis	Deep ulcerations, black scabs	Face, legs	Haemolytic staphylococci	
Caseous lymphadenitis	Abscesses in lymph nodes	Head, body	*Corynebacterium pseudotuberculosis*	
Ringworm	Dry, crusted scabs	Head, body if shorn		Fungal hyphae and spores
Mycotic dermatitis (*Dermatophilus*)	Exudation, matted wool, raised scabs, superficial raw areas under scabs	Body, face, ears	Gram +ve filamentous cocci	
Wool rot	Confined to wool fibres – colouration	Body, especially back	*Pseudomonas aeruginosa*	
Orf	Vesicles, scabs, proliferative lesions	Mouth, lips, legs, teats, prepuce	Virus (with electron microscope)	
Sheep scab	Exudation, yellow crusts, intense pruritus, marked wool loss	Body		*Psoroptes*
Ear mites	Ear discharge, haematomas	Ears		*Psoroptes*
Head fly	Ulcerations on head especially around horn base	Head		Flies visible on lesions
Midge sensitivity	Gross thickening and ulceration of skin	Head, ears, legs, belly, udder, perineum		Midges present
Other external parasites	Parasites visible, varying skin lesions	Depends on species involved		Lice, keds, ticks, blowfly larvae
Scrapie	Self-inflicted lesions, neurological signs	Body, tailhead, face		
Photosensitisation	Exudation, crusting, skin loss	Head, ears (legs, back)		
Debility (wool break)	No skin lesions, weak wool fibres	Body, random		
Woolslip	No skin lesions, recently shorn	Body, back, flanks		

can be differentiated on the grounds of severity, but dry scabs and swabs of lesions can be submitted for viral and bacteriological investigation if necessary. If treatment is necessary, a broad-spectrum antibiotic should be effective.

Severe dermatitis and scabbing of legs

Check for

- Mud fever – due to excessively muddy conditions. Will generally clear when animals are moved to drier area, but may need antibiotic treatment if badly infected.

Strawberry-like lesions on face or legs (see colour Figure 29)

Check for

- Orf virus – these usually affect only individual animals within a group, and may indicate an inability to respond effectively to the virus. See below.

Persistent lesions of hyperkeratosis on poll (especially of rams) or ears

Check for

- Persistent orf infection – see below.

Swelling and oedema of head, fever and rapid death

Check for

- Clostridial infection of wounds, e.g. rams fighting. Check vaccination history and implement suitable vaccination programme.

Individual animals, nonwoolled areas affected, lesions generally pruritic

Head lesions on poll or around base of horns

Check for

- Head flies (*Hydrotea irritans*) – this is usually a flock problem affecting young horned sheep, but individual sheep (polled as well as horned) may be affected if they have head wounds at the time these flies are active. See below.
- Allergy to midges – this can affect individual animals, but may also affect a significant proportion of a flock. See below.

Aural haematoma

Check for

- Fighting injury.
- Recent application of ear tag.
- Ear mites – usually several animals are affected. See below.

Swelling of the head and ears with oedema of the conjunctiva

Check for

- Photosensitisation – face swelling (see colour Figure 25) followed by exudation and crusting of unpigmented skin, especially head, ears, legs and perineal area. Some of the affected skin may slough off, especially the ear tips. Jaundice occurs in some cases. Although it may sometimes be possible to identify the cause, sporadic cases are seen for which no cause can be found.

Photosensitisation occurs due to presence within the body of photodynamic substances which may arise in three ways:

(1) Ingestion of plants containing a photodynamic substance, e.g. St John's Wort.
(2) Aberrant pigment synthesis (not recognised in sheep in the UK).
(3) Hepatogenous, i.e. the result of liver damage, caused by plant toxins, e.g. rape, fungal toxins or certain drugs. This interferes with excretion of phylloerythrin, a product of ruminant digestion normally metabolised by the liver, which thus escapes into the circulation where it is photodynamically active. Bog asphodel has been associated with photosensitisation in the UK ('plochteach') and Norway ('alveld'). The exact mechanism is still debated but may be due to saponins in the plants or fungal toxins on the plants. Facial eczema, which is an important problem particularly in New Zealand, is another form of photosensitisation; it is caused by sporidesmin, a product of the fungus *Pithomyces chartarum* which parasitises ryegrass.

Treatment of photosensitisation

Affected animals should be housed to remove them from direct sunlight and to prevent them eating the causal agent, if identified. Treatment with steroids will usually reduce swelling of the face and ears. In severe cases affected areas of the skin may crack and slough (particularly the ear tips). Antibiotic treatment may be necessary if secondary infection is a problem.

Many animals, nonwoolled areas affected, lesions generally nonpruritic

The conditions described above may appear in more than one animal, but where a number of animals are affected, the cause is likely to be infectious.

Diagnosis will be assisted by sampling typical lesions by taking scab material, swabs from beneath scabs, and scrapings from active areas of lesions as necessary.

Abscesses on face, neck and/or in body lymph nodes

Check for

- Caseous lymphadenitis (*Corynebacterium pseudotuberculosis*) – this infectious disease is found particularly in pedigree terminal sire breeds but is likely to become common in many flocks. The most commonly affected lymph nodes are the parotid (just below the ears) (see colour Figure 26), submandibular and prescapular (see colour Figure 27). In some countries other body lymph nodes such as the popliteal and precrural may be more commonly affected. Remember that internal lymph nodes, especially those in the lung may be also involved and these are important in transmission of the disease. If in doubt about the diagnosis, take swabs of pus and submit to the laboratory for confirmation. Blood testing using an ELISA is being developed to assist in diagnosis on a flock, rather than on an individual basis.

Treatment

Treatment is not usually successful, since antibiotics cannot penetrate the well-encapsulated abscesses. Even if visible abscesses are drained and vigorously treated, it is highly likely that other abscesses will be present elsewhere in the body. Affected animals should be culled if at all possible, particularly if the infection has been recently introduced into the flock.

Control and prevention

Once introduced into a flock, elimination is extremely difficult, therefore every effort should be taken to avoid buying infected animals. Rams of terminal sire breeds should be examined particularly carefully and none with evidence of abscesses should be purchased. Other rams from the same source, even though appearing normal, should also be avoided. It may be worth treating newly purchased rams with long-acting antibiotic in an attempt to eliminate undetected lesions; however, this is not a proven method of avoiding introduction of the disease. If infected animals are introduced into a flock it is worth removing them if discovered quickly, but infection is likely to have spread if they have been housed or trough fed with others, since spread is facilitated by close contact.

Once the infection is established in a flock the strategy adopted will depend on the type and value of the animals. In commercial flocks removal of clinical cases as soon as detected may reduce the spread of infection. Floor feeding rather than trough feeding will also reduce spread. In pedigree flocks it is possible to establish an uninfected nucleus group by removing lambs at birth and rearing them artificially away from the main flock. If such extreme measures are not possible the next best alternative is to separate

clinically uninfected animals and to wean their lambs as soon as possible, then rear them away from contact with the rest of the flock. Keeping different age groups separate, rather than mixing young and old animals, will also reduce the risk of infection spreading. Attention must also be paid to housing, handling pens and feeding troughs since the bacteria can live on these for several months at least. Vaccination against the disease is practised commonly in some countries but the most widely used and successful vaccine (which is directed against the toxin produced by the bacteria) is not available in the UK. A vaccine consisting of killed bacteria can be used but its efficacy is questionable.

Scabs on mouth and lips (in very early stages papules and vesicles present)

Check for

- Orf (contagious pustular dermatitis) – this common infection caused by a parapox virus may also be found on the teats of lactating ewes, inside the mouth of young lambs and on the genitalia of rams. In some individual animals, the virus may persist causing proliferative lesions on the head and legs (see above). See Chapter 13.

> Care should be taken in handling material since orf is a zoonosis.

Raised crusts which separate or can be lifted off leaving raw areas beneath

Check for

- Mycotic dermatitis (caused by the bacterium *Dermatophilus congolensis*) – this may affect the face and ears particularly in lambs, and the scrotum of rams. Make impression smears of the underside of freshly removed scabs and stain with Gram's stain. This condition will show Gram-positive branching filaments breaking into multiple rows of Gram-positive cocci. If further confirmation is required, submit samples to laboratory for FAT (fluorescent antibody test) or isolation. The same organism infects woolled areas of older sheep causing 'lumpy wool', and in combination with orf virus, causes strawberry footrot in lambs, see below. Mycotic dermatitis can be treated by administration of a course of antibiotics such as penicillin/streptomycin mixture or long-acting oxytetracycline. Application of a solution of zinc sulphate (100 g in 10 litres of water) to affected areas is an alternative treatment. In the case of a flock problem, zinc sulphate can also be added to dip baths, but check with the dip manufacturer to make sure the chemicals are compatible.

Proliferative scabby lesions which bleed when scabs are removed affecting coronary and lower leg areas

Check for

- Strawberry footrot – this is generally considered to be caused by a combination of orf and *Dermatophilus* infection. Antibiotics may speed resolution, but remember that these are active only against the bacteria involved, not the orf virus.

Multiple dry crusted areas on head and/or legs

Check for

- Ringworm (usually *Trichophyton verrucosum*) – take skin scrapings and hairs from edge of lesion, mix with potassium hydroxide and examine for spores. There will usually be contact with calves or buildings in which calves have been reared. If treatment is necessary, natamycin can be applied topically as in cattle.

> Ringworm is a zoonosis.

Lesions affecting the prepuce of rams or the vulva of ewes

Check for

- Bacterial infection – *Streptococcus zooepidemicus* has been associated with flock outbreaks, and mycoplasmas and ureaplasms have been isolated but their significance is not known. There appears to be no effect on fertility.

Lesions affecting interdigital space, with lameness

Check for

- Scald (see Chapter 14).
- Remember the possibility of foot and mouth disease.

Many animals, nonwoolled areas affected, lesions generally pruritic (Figure 20.1)

Some of the conditions described above may become pruritic, particularly if secondary infection takes place, or during the healing process, therefore should not be excluded on presence or absence of pruritus alone. In addition there are two important causes of pruritic lesions affecting usually the heads of many animals.

Wounds around base of horns in summer

Suspect

- Head flies (*Hydrotea irritans*) – these affect mainly young horned sheep but may also affect other sheep with skin damage on the head, e.g. rams fighting. These flies are active during the summer and cause intense irritation. In still, warm weather, affected animals can be seen shaking their heads and trying to avoid the swarms of insects. Sheep with wool cover on the head are less likely to be affected. Affected sheep rub their heads, making the wounds bleed and become more extensive. Application of a 'spot-on' preparation containing a synthetic pyrethroid before the flies become active will control the condition. Treatment may need to be repeated at intervals during the fly season.

Aural haematoma

Check for

- Ear mites – *Psoroptes ovis* – take swab or rinsing from ear canal and examine under microscope for mites and thin-walled eggs. If detected, the most effective treatment is ivermectin by injection (two doses at an interval of 7–10 days) or a single injection of doramectin or moxidectin.

Severe ulcerated lesions are present around the eyes, nose, and on legs

Suspect

- Staphylococcal dermatitis (see colour Figure 24) – this is often found in trough-fed groups. Ulcers bleed easily, become covered with black scabs and hair is lost from the surrounding area. The characteristic clinical picture should suggest a diagnosis, but this can be confirmed by isolation of haemolytic *Staph. aureus* from the lesions. Treatment with parenteral broad-spectrum antibiotic may be necessary. Attention should also be paid to management of the affected animals, since the condition is often associated with a shortage of trough or rack space causing sheep to bang their heads and legs when competing for feed. Extra space should be provided, or floor-feeding of concentrates adopted.

Severe pruritic lesions affecting head, legs and belly in summer months

Suspect

- Allergy to midges (similar to sweet itch in horses). This has been reported to affect a high percentage of a Black Welsh Mountain flock and has also been

seen in other, mainly dark-woolled, animals. Lesions start to appear, as midges become active in late spring. Affected animals rub areas of skin not protected by wool – the head, legs, belly and perineal areas. Skin thickening with excoriated and scabby areas develops as a result of self-mutilation. Lesions subside in autumn as midge activity ceases. Control can be attempted by application of an SP pour-on before the midge season starts and by removing animals from low-lying, damp or overgrown habitats which are heavily infested with midges.

Individual or small numbers of animals, woolled areas affected, no pruritus present, no skin lesions

Wool loss is zonal around neck or along spine

Check for

- Natural shedding or delayed shearing. Masham and Mule sheep often lose wool prematurely from these areas. As previously mentioned, some primitive sheep shed the fleece naturally in early summer.

Wool loss is random

Check for

- Acute or chronic illness, undernutrition, debility or other stress (wool break) (see colour Figure 30).

Wool loss follows winter shearing

Suspect

- 'Wool slip' (see colour Figure 31) – new wool growth is lost particularly from the spine and flank areas, giving sheep a 'moth eaten' appearance. This condition is thought to be caused by excess cortisol production as a result of a combination of stress factors associated with housing and shearing, but occasionally may be caused by feed mites, in which case some pruritus would be expected. The condition is of cosmetic importance only, as wool soon regrows. Attempts have been made at prevention by increasing feed intake before shearing, but this is not always successful.

Individual or small numbers of animals, woolled areas affected, pruritus present (see Figure 20.1)

In summer months

Check for

- Blowfly strike – affected animals are restless, show excessive tail twitching and foot stamping, frequently turning the head to attempt to nibble the

lesion. The most commonly affected area is soiled wool around the tail, but other parts of the body and even the feet may be involved. Lesions, which are intensely pruritic, may be difficult to locate in the early stages, simply appearing as areas of damp wool, but a characteristic smell soon develops and maggots should be found on careful searching.

Treatment of blowfly strike

Wool should be clipped from the affected area and all maggots removed. If many animals are affected, dipping in either organophosphate (OP) or synthetic pyrethroid (SP) (high cis cypermethrin) dip is probably most practical, as this will treat the infestation and prevent further problems for at least 8 weeks. Alternatively an SP pour-on can be applied to affected areas.

> Insect growth regulators such as cyromazine will not treat established strike.

Prevention of blowfly strike

Good worm control to reduce soiling of the breech area, and footrot control to reduce body strike, will both contribute to a reduction in incidence of blowfly strike. However, it is sensible to protect animals by chemical means unless they are to be kept under close observation throughout the risk period. This can be done by:

(1) Application of an insect growth regulator such as cyromazine as a pour-on before the flies become active. Treated animals are protected for up to 10 weeks. Dicyclanil will give 16 weeks' protection.
(2) Application of an SP pour-on. This will give protection for 6–8 weeks.
(3) Application of OP or SP by dipping, jetting or showering. This will protect for 8–10 weeks.

> Care must be taken to wear adequate protective clothing when handling OP or SP products. OPs have been associated with ill-heath in some people. Both types of product can damage the environment, and regulations governing their safe disposal must be followed.

If 'nibbling' reflex is present in absence of ectoparasites

Check for

- Scrapie – can be seen in sheep as young as 2 years, although most cases are in older animals. Self-inflicted skin lesions may be present, as well as neurological signs such as incoordination, hyperexcitability (see Chapter 15).

Many animals, woolled areas affected, no pruritus, no skin lesions

Those conditions listed as affecting individual animals (see above) may also affect more than one of a group, particularly:

- Natural shedding – breed.
- Under-nutrition on flock basis, particularly in late pregnancy.
- Wool slip if winter shorn.

In addition:

If wool growth generally is poor in quality and/or quantity

Check for

- Micronutrient deficiency – copper and cobalt deficiency both have effects on fleece growth as well as leading to ill thrift.
- Other chronic disease such as coccidiosis, parasitic gastroenteritis. See Chapter 9.

Many animals, woolled areas affected, skin lesions or abnormal wool present, little pruritus

Check for

- Mycotic dermatitis (lumpy wool) – *D. congolensis* commonly affects fleeced areas as well as hairy areas as described above. Lesions most commonly appear on the back, and consist of an exudative dermatitis, the exudate drying and causing the wool to mat with scab formation. In early cases this may only be seen when the fleece is parted, but becomes more obvious as the disease progresses. Diagnosis is confirmed by staining impression smears of scabs. See above. In some cases pruritus is present.
- Ringworm – this is rare on woolled areas but may occur in sheep exposed to infection soon after shearing, e.g. in winter housing with calf contact. Confirm with examination of hairs and skin scrapings from active part of lesion.

If matting of the wool with abnormal pigmentation, usually yellow or greenish-blue is present

Suspect

- Fleece rot (canary stain). This is thought to be caused by pigment-producing bacteria such as *Pseudomonas aeruginosa*, particularly in prolonged wet weather. Affected fleeces are downgraded. *P. aeruginosa* infection has also been associated with severe dermatitis and death following rough handling while dipping.

Yellow pigmentation of grease which easily washes out of the fleece is commonly found in some breeds, particularly longwools.

Many animals, woolled areas affected, pruritus present, wool loss (see Figure 20.1)

Most conditions with pruritus will involve skin lesions; if not a part of the primary cause, they will become apparent as a result of self-mutilation. Some skin lesions which are not initially pruritic may become so during the healing process or as a result of secondary infection.

There are a few transitory conditions where pruritus occurs without the development of skin lesions but may give an inexperienced owner cause for concern:

- In hot weather immediately pre-shearing, sheep in heavy fleeces may rub against fences, hedges, etc.
- In warm humid weather, small flies and midges cause irritation, with sheep showing sudden head shaking, foot stamping, and alternate lying and standing. This behaviour can be seen in housed sheep as well as in those at grass.
- Store lambs on ad libitum complete diet may show sporadic attacks of pruritus, possibly due to transient food allergy.

As mentioned above, some cases of mycotic dermatitis and ringworm may also show significant pruritus. However, the majority of incidents involving many animals with severe pruritus will be parasitic in origin.

Check for external parasites

- Sheep scab (*Psoroptes ovis*) (see colour Figure 32) – affected sheep show intense irritation, with exudate drying to form yellow crusts in the fleece which becomes moist and matted. Areas of wool are soon lost because of continuous rubbing. The intense irritation is the result of hypersensitivity to the mite faeces rather than to the mites themselves. This disease is now widespread but is no longer notifiable.
- Lice – *Bovicola (Damalinia) ovis* is a chewing louse and is found mostly around the neck and back areas. It has a rounded head. This causes irritation, matting of the fleece and wool loss. *Linognathus ovillus* is found mainly around the head. *Linognathus pedalis* is found on the legs and scrotum. Both are sucking lice and are blue in colour with conical heads. They also cause irritation and rubbing of affected parts of the body. Lice have become much more common in recent years since compulsory dipping for scab ended.

Diagnosis of scab and lice infestations

When many sheep are itching it is important that an accurate diagnosis of the cause is made. Many farmers assume a diagnosis of either scab or lice without

veterinary involvement. If the diagnosis is incorrect, inappropriate treatment is given. This obviously does not work, leading to further treatments, often with exposure of mites or lice to sub-lethal doses of chemicals, thus facilitating the development of drug resistance. Occasionally sheep are infested with both parasites. The clinician's role therefore is to encourage clients to call for help in the diagnostic process so that correct treatment is given. Examination of several characteristically affected animals, together with examination of skin scrapings, should produce a definitive diagnosis. Lice are visible to the naked eye and should be readily seen if the fleece is parted in several places. Scab mites are just visible (like a grain of sugar), but use of a hand lens and examination at the edge of lesions, or where scratching produces an intense itch response, will usually enable mites to be identified, if present. Skin scrapings should be taken from the edge of lesions. These can be added to a solution of potassium hydroxide then heated to dissolve skin debris. If present, mites can then be seen when the remaining sediment is examined under the low power of a microscope.

Treatment of external parasites (see Table 20.3)

> Pour-on SP products should never be used to treat scab cases – sub-lethal concentrations lead to the development of resistance.

Table 20.3 Activity of chemicals against external parasites.

Chemical	Method	Scab	Lice
OP	Dipping	Yes	Yes
	Showering/jetting	Inadequate	Probably
SP flumethrin	Dip	Yes (but resistant strains)	No
SP high cis cypermethrin	Dip	Yes (but resistant strains)	Yes
	Pour-on	No	Yes
Ivermectin	Injectable	Yes (2 doses)	No
Doramectin	Injectable	Yes	No
Moxidectin	Injectable	Yes	No

The only certain treatment that will cover both types of parasite is plunge dipping in an OP product. All other dips vary in their efficacy; resistance of scab mites to flumethrin and high cis cypermethrin has been reported in the UK and elsewhere. Development of resistance in scab mites has been hastened by incorrect application of SP pour-ons designed for treating lice. Products designed to be used in dips should not be used for treating scab by showering or jetting, since adequate coverage of the whole body is unlikely and sub-lethal doses of chemicals are likely to hasten development of resistance.

Scab can also be successfully treated by systemic endectocides. Ivermectin

has no residual action, therefore two doses are required at an interval of 7 days to ensure that all mites are killed. Doramectin and moxidectin by injection have residual action. Mites can survive off the host for up to 17 days so it is important that sheep are protected for longer if they are to be put back into the infected environment. Both moxidectin and doramectin are able to provide sufficient protection in these circumstances. These products are not effective against chewing lice, and, although they may have some efficacy against sucking lice, are not designed for their treatment.

> Injectable moxidectin should not be given to animals that have received footrot vaccine.

> All chemicals used for controlling external parasites are potentially toxic to people and the environment. Instructions for their use and safe disposal must be followed carefully.

Control and prevention of scab and lice infestations

The only way to keep a flock free of infection is to ensure that it is managed as a closed flock. This requires adequate fencing of boundaries to prevent access of straying sheep. Any purchased animals should be isolated for at least 3 weeks and treated with an appropriate product unless freedom from either parasite can be guaranteed. Where sheep are kept on common grazing, cooperation between all owners to gather and treat all animals simultaneously is the only way of controlling these parasites.

Other external parasites

- Fly strike – this can affect more than one animal if weather conditions are favourable for fly activity, particularly if dagging has been neglected. See above.
- Keds (*Melophagus ovinus*) – these are wingless flies and, being much larger than lice, are readily visible. They are susceptible to all products used for scab and lice treatment.
- *Tyroglyphus* (forage mite) occasionally causes problems in housed sheep and requires skin brushings to identify.
- *Ixodes ricinus* (castor bean ticks) are found mainly on the hairy areas of the head, neck, axilla and groin in spring and autumn. If these are found, consider the possibility of tick-borne diseases as well as tick worry. Ticks are best controlled with an SP pour-on.
- *Haemaphysalis punctata* (festooned tick) is occasionally found in southern Britain.

21 Respiratory disease

The standard criteria used in the antemortem diagnosis of respiratory disease in other species are very limited in application to the sheep, yet the two statements 'pneumonia is the single greatest cause of death in the sheep' and 'pasteurellosis is the most over-diagnosed disease of sheep' are both true, and not contradictory as might appear at first glance. In addition, there is a deep-seated belief among farmers that 'all sheep cough' which will often delay the point at which the client seeks help.

The respiratory rate of normal sheep shows wide variations according to factors such as weather conditions, fleece cover and fatness. It can be very rapid (>100/min), sometimes with open-mouth breathing, in the full-fleeced ewe on a hot day in humid conditions, even at rest. If the sheep is fat and is driven, then the rate may become dangerously high, still in a normal healthy animal. A shorn ewe at rest on a cool day may well have a very low respiratory rate (<20/min).

In advanced pregnancy, when reproductive products may occupy in excess of 60% of abdominal volume, the respiratory rate may also be raised to one which, to the inexperienced observer, would suggest pathological change. Thus, respiratory rate alone, without taking into account other factors, is a very unreliable guide to the presence or absence of disease.

Auscultation of the chest may give some limited information, particularly in the young lamb or newly shorn ewe. It is necessary to appreciate that restraint for examination may exaggerate the sounds heard and that, even in normal animals, respiratory sounds are louder than in cattle. It should also be borne in mind that rumen sounds can normally be heard over a large area of the left side of the chest, posterior to approximately the sixth rib. Examination of the chest with ultrasound can give added information about the presence of abscesses or other lesions in the lower parts of the lungs, or fluid in the chest, but requires experience to interpret images.

Coughing, nasal discharge and/or fluid escape from the nares are significant. The type of respiration may also be of significance, although it should be remembered that in the normal sheep it is mainly abdominal rather than thoracic in nature. Body temperature is also of limited significance in the sheep, being subject to the same wide variations as respiratory rate.

To complete the limitations of clinical diagnosis, the sheep seems to have a very low innate resistance to respiratory infection. This characteristic, combined with the usual infrequency of examination by the owner, means that the animal is often found dead (except in case of chronic or slow viral diseases)

before any clinical examination can be carried out. It follows that the vast majority of respiratory disease is diagnosed at PME, and it is in the interpretation of postmortem findings that the clinician will be able to apply expertise, although laboratory assistance will often be required in making a final diagnosis.

In addition to physical causes such as trauma and inhalation pneumonia, the following organisms are frequently involved in the pathology of respiratory disorders of sheep. Frequently, there will be coexistence of two or more infective agents, particularly *Mannheimia* with pulmonary adenomatosis, or with mycoplasmas in atypical pneumonia.

Bacteria

- *Mannheimia haemolytica* (formerly *Pasteurella haemolytica* biotype A) – this organism is a normal inhabitant of the upper respiratory tract and has a number of serotypes. It causes both systemic and pneumonic disease. Even though the name of the organism has changed, the disease is still commonly referred to as pasteurellosis.
- *Pasteurella trehalosi* (formerly *P. haemolytica* biotype T) causes systemic disease mainly in young sheep and is often associated with sudden death (see Chapter 22).
- *Pasteurella multocida* – this is not thought to be associated with disease in the UK but can be implicated in disease in some countries.
- *Arcanobacterium* (formerly *Actinomyces* and *Corynebacterium*) *pyogenes* can cause abscesses in the lungs, often as the result of haematogenous spread.
- Staphylococci and streptococci may be found in lung lesions in very young lambs.

Mycoplasmas

- *Mycoplasma ovipneumoniae* – together with *M. haemolytica* causes atypical pneumonia.
- *Mycoplasma arginini* and *M. capricolum* have been isolated from sheep lungs but their role in disease is uncertain.

Viruses

- A retrovirus (JSRV) causes pulmonary adenomatosis; a herpes virus is often found in association with the retrovirus in diseased animals, but is not thought to have a primary role in the disease.
- A retrovirus of the sub-family lentivirus causes maedi-visna.
- Parainfluenzae 3 (PI3) virus – sometimes causes a mild primary disease but its main importance is as a factor in outbreaks of pasteurellosis (this may also be a normal pathogen of the upper respiratory tract).
- Adenoviruses, reoviruses and respiratory syncytial virus have been found but their role in disease is unclear.

Parasites

- *Dictyocaulus filaria* causes parasitic bronchitis.
- *Muellerius capillaris* is commonly found but thought to be of little clinical significance.
- *Protostrongylus rufescens* and other lungworms have been reported but are of little significance apart from possibly in debilitated animals.

From this formidable list of organisms, a definitive diagnosis can only be made with confidence on a combination of history taking, age of the patient (Table 21.1), speed of onset (Table 21.2), postmortem lesions found, and bacteriological, histological, parasitological and serological tests where appropriate.

Table 21.1 Age incidence of common causes of respiratory problems.

	Individual	Multiple
Neonate	Trauma (ribs) Neonatal respiratory distress Post-dystocia oedema Haemorrhage Milk inhalation	Septicaemic pasteurellosis Nutritional myopathy (intercostal muscles) Cow colostrum anaemia
Growing lambs	Inhalation pneumonia (drench, dip)	Inhalation pneumonia (drench, dip) Atypical pneumonia Acute pasteurellosis Chronic pasteurellosis Parasitic bronchitis Nutritional myopathy
Adults	As above plus Laryngeal chondritis	As above plus Pulmonary adenomatosis (SPA) Maedi (MV)

Antemortem diagnosis of respiratory disease

The following points may assist in diagnosis in the live animal, but it must be emphasised that confirmation may require postmortem examination of representative animals.

Neonatal lambs

Changes in respiratory rate in neonatal lambs are more often of significance than in older sheep but may not necessarily indicate lesions in the lungs or upper respiratory tract. A raised body temperature is also more significant than in older animals, but it should be remembered that hypothermia soon intervenes if the young lamb ceases to feed for any length of time.

Table 21.2 Speed of onset of respiratory disease.

	Acute	Chronic
Neonate	Trauma Neonatal respiratory distress Post-dystocia oedema Nutritional myopathy Haemorrhage Inhalation	
Growing lambs	Acute pasteurellosis Inhalation Nutritional myopathy	Chronic pasteurellosis Atypical pneumonia Parasitic bronchitis
Adults	Laryngeal chondritis Acute pasteurellosis	Maedi (MV) Pulmonary adenomatosis (SPA) Chronic pasteurellosis Chronic nasal obstruction

In the neonate, the clinical pattern of respiratory infections is septicaemic rather than pneumonic. Depression and death are likely to occur before thoracic changes progress to the point at which dyspnoea is apparent.

Dyspnoea/tachypnoea

Check for

- Trauma of the rib cage – parturient trauma, or treading injury. Lambing management should be assessed but sporadic injuries can occur in even the best managed flocks.
- Pharyngeal/laryngeal oedema as a result of head-only presentation – provided the lamb can get sufficient oxygen, the swelling will usually resolve within a few hours. Keeping the head raised will help and steroid injection is worth considering for a valuable lamb.
- Failure of lung expansion – neonatal acute respiratory distress syndrome – prematurity or surfactant failure. These lambs often die within a short time of birth. Administration of a respiratory stimulant, e.g doxapram, as drops under the tongue may help in some cases.
- Congenital defects, e.g. ventricular septal defect in the heart. The lamb is unlikely to thrive and euthanasia should be considered.
- Inhalation pneumonia – often associated with artificial or force feeding – rattling respiration, milky discharge down nostrils. Feeding technique should be checked. Correct use of a stomach tube will avoid this problem.
- Evidence of anaemia – umbilical bleeding, or cow colostrum induced (see Chapter 17).

- Septicaemic pasteurellosis – usually seen as sudden death, although sick in-contact lambs may be noticed with close, frequent inspection. A broad-spectrum antibiotic such as oxytetracycline may be effective if treatment is given early.

Prevention of pasteurellosis in very young lambs

Vaccination of ewes to provide passive immunity to lambs via the colostrum can be effective in controlling this problem. However, it is important that the farmer is aware that immunity only lasts for about 4 weeks. As vaccination of young lambs is advised only from 3 weeks of age onwards, a 'protection gap' is difficult to avoid.

Growing lambs

As noted above, the clinical picture of diseases caused by lung pathogens changes from being primarily septicaemic in the very young, to primarily pulmonary in the growing animal.

Dyspnoea

Check for

- Infectious pneumonia – examination by auscultation may reveal evidence of areas of lung consolidation. This may indicate atypical pneumonia, viral infection or pasteurellosis, or a combination of organisms. With atypical pneumonia and simple viral infections, animals may show only mild signs of illness but there is always the risk of superimposed pasteurellosis. In this case outbreaks often begin with some sudden deaths. PME is required for differentiation of the exact cause. Paired sera may assist with retrospective diagnosis of viral involvement but are not very helpful in pasteurellosis outbreaks because of the number of serotypes involved.

Treatment of infectious pneumonia

Where animals are ill or dying, antibiotic treatment will be necessary. As the whole group is likely to become infected by droplet spread, and isolation of individual affected animals is rarely practicable or helpful, treatment of all contact animals is usually indicated. Long-acting oxytetracycline has been the most commonly used preparation, but more recently tilmycosin has become available and is usually very effective in cases of pasteurellosis, although is expensive. Dosage based on accurate body weight is essential to avoid accidental overdosage and it should not be used for lambs under 15 kg body weight.

> Tilmycosin is dangerous if accidentally self-adminstered. Medical help should be sought urgently.

Prevention of infectious pneumonia

> It is essential to recognise that many of the respiratory pathogens are normal inhabitants of the upper respiratory tract. It is often only when some form of stress or poor husbandry (e.g. inadequate ventilation of buildings, overcrowding) is imposed that clinical disease occurs. Thus avoidance of stress and good standards of housing, with good ventilation and adequate good quality bedding are the starting point.

The vaccination regimen should be examined, remembering that passive immunity with pasteurella vaccine lasts for only 4–5 weeks of life. Lambs can themselves be vaccinated from 3 weeks of age (earlier if the problem is severe in young lambs), but it is difficult to avoid a gap in protection. Two doses of vaccine are required 4–6 weeks apart. If this schedule is already in place, it may be worth introducing extra doses before particular times of risk if these can be identified. The pattern of disease may vary from farm to farm so it is difficult to lay down a standard format. Administration of intranasal PI3 vaccine has been shown to be of value in some cases where pasteurella vaccine alone has failed to provide sufficient protection.

Check also for

- Inhalation pneumonia – there may be evidence of drenched material at nostrils.
- Nutritional myopathy affecting respiratory muscles (blood sample for CK and GSH-Px estimations). Affected lambs may respond to an injection of a selenium/vitamin E preparation. The same product can be administered to ewes in late pregnancy or to at-risk lambs, and should be effective in preventing the condition.

Chronic cough

Check for

- Atypical pneumonia – postmortem confirmation may be necessary. This disease alone rarely causes death but may lead to decreased growth rates. Improving ventilation and general standards of housing will usually control the problem.
- Parasitic bronchitis – faeces samples for larval identification may be necessary, although lungworm infections rarely assume the importance that they do in cattle. Most modern anthelmintics are effective against lungworms, but note that morantel is not effective.
- Chronic pleurisy, pleural adhesions or effusion may follow previous respiratory infection.

Nasal discharge

Check for

- Viral infection and/or pasteurellosis as above – isolation of *M. haemolytica* from nasal swabs does not confirm diagnosis since it is present in most normal sheep. Paired sera with rising titres for viruses such as PI3 may help to indicate viral involvement.
- Transient upper respiratory infection due to stress may occur, but this is usually nonfebrile with little or no systemic involvement.
- Excessively dusty conditions which may cause inflammation of the upper respiratory tract without systemic illness.
- *Oestrus ovis* infection – this leads to thick, often unilateral discharge, sometimes with blood. There is no easy method of diagnosis unless parasites are sneezed out. This parasite is found in animals in the south of the UK only.

Any of above plus reduced weight gain or emaciation

Check by auscultation for

- Chronic lung damage, possible chronic pasteurellosis or neonatal septicaemia with multiple abscesses in lung tissue plus pleurisy. If the degree of chronic lung damage is significant, euthanasia may be the best option on economic grounds.

Adult sheep

The clinical picture of a recent infection with pasteurellosis and/or viral infection will not differ from that shown in growing lambs. When these infections have become long-standing, or where chronic viral infections are involved, chronic weight loss becomes an additional diagnostic feature.

Chronic cough with weight loss

Slow viral infections most commonly affect adults from 2 to 4 years in the case of pulmonary adenomatosis, and from 3 to 5 years in the case of maedi.

All older sheep which are 'thin for teeth', i.e. they have body scores below that of their contemporaries on adequate nutritional inputs, and without evidence of tooth disease, can be presumed to have a high probability of chronic respiratory disease. Culling should be considered with PME if a significant number are affected.

Check for

- Pleurisy and areas of consolidation – usually associated with chronic pasteurellosis.
- Excessive fluid in airways (wheelbarrow test) (see colour Figure 33) – pathognomonic for pulmonary adenomatosis (SPA, Jaagsiekte). This condition is caused by a retrovirus and results in growth of adenomatous tumours in the dependent parts of the lungs (see colour Figure 34). These vary in size from smaller than one centimetre to sufficiently large to occupy most of the ventral portion of the lung. The most characteristic feature is fluid production from the tumour cells, resulting in massive accumulation of fluid in the airways. This may be apparent as mucoid nasal discharge but is most easily seen if the hind quarters of the sheep are raised, when frothy fluid pours from the nose. This fluid contains virus and is highly contagious to other sheep.

Control of SPA

There is no treatment, so infected sheep should be culled as soon as they are identified. This can only be done on the basis of clinical signs as there is at present no serological or other test. There is no method of prevention other than removing infected animals as soon as possible as above, or maintaining a closed flock or buying from a flock with no evidence of infection. With heavily infected flocks, it may be best to cull the whole flock and restock.

- Maedi (MV) – this is caused by a slow (lentivirus) virus which causes progressive lymphoid infiltration and smooth muscle hyperplasia in the lungs, leading to a progressive, fatal pneumonia. Development of clinical signs takes years, with gradual onset of laboured breathing and weight loss. Spread of infection is vertically via colostrum and milk which contains virus, and horizontally via droplet spread. It can also be spread by instruments or needles contaminated with blood of infected animals. Subclinical infection is now present in a number of flocks in the UK, with some clinical cases becoming apparent – it is estimated that over 100 000 sheep are infected. There is no treatment so infected animals should be culled as soon as possible. These can be detected by serology.

Control and prevention of maedi-visna

Since there is no treatment and an infected flock suffers financial losses through premature culling and reduced milk yield, therefore increased lamb mortality and poor lamb growth rates, the primary aim must be to avoid introduction of the agent into the flock. Obtaining replacement stock carrying certified status as being free of infection is highly cost-effective. The Sheep and Goat Health Schemes run by Scottish Agricultural Colleges (SAC) include a maedi accreditation scheme and a maedi monitoring scheme. The accreditation scheme is aimed at pedigree flocks and closed flocks and requires two clear blood tests. Accredited sheep must be kept separate from nonaccredited animals and retesting is carried out every 2 years. Monitored flocks are subject to less

stringent rules and are accepted after one clear test with retesting every second year.

Where a flock has become infected, a test and cull programme will minimise the level of disease but is unlikely to eliminate the problem since there are always animals in the early stage of the disease that will not be detected serologically. For the commercial farmer, lowering the age span of the flock is likely to be beneficial, since the older animals are those most likely to be clinically affected. In the case of heavily infected flocks, the only option may be to completely cull and restock with tested animals. In the case of a pedigree flock, it is worth considering whether to try to retain the genetic lines by removing lambs at birth ('snatching') and rearing artificially completely away from the adults. If 'snatching' of lambs at birth is to be tried, it is important that the client is advised of the hard work involved in rearing these lambs, since adequate alternative supplies of colostrum have to be supplied. In addition, any contamination of lambs with maternal blood, licking of lambs by ewes or suckling of ewes by lambs, risks jeopardising the whole project. Lambing must therefore be totally supervised round the clock. Embryo transfer into maedi-free ewes may be worth considering in very valuable flocks.

With all respiratory diseases, spread throughout the flock can be minimised by reducing stocking density and, if housing is part of the production system, ventilation must be above reproach. Indeed the 'walls' need only consist of screens which prevent rain or snow access but have minimal effect on air flow.

Acute dyspnoea (upper respiratory tract)

Check for

- Laryngeal chondritis – this is particularly common in Texels but is also seen in other short necked breeds. Diagnosis is obvious, based on the characteristic inspiratory dyspnoea and stridor – this can often be heard before the patient is in sight. It seems likely that by breeding animals with a short, wide neck, the laryngeal opening has become reduced in size. Any infection or swelling of the arytenoid cartilages soon leads to life-threatening occlusion of the larynx. Treatment should be given as a matter of urgency – a broad-spectrum antibiotic to deal with infection and steroid to reduce swelling. Relapse is common. In severe cases, an emergency tracheostomy may be necessary to save the animal's life. There is no method of preventing the condition, but breeders should consider whether it is advisable to breed from recovered animals, particularly rams, as there may be a genetic predisposition.

Chronic dyspnoea (upper respiratory 'snoring')

This may result from *Oestrus ovis* infection, chronic infection of nasal passages, or tumours, but antemortem diagnosis is likely to prove difficult. Enzootic

nasal tumour, caused by a retrovirus, is common in many countries but not in the UK, New Zealand or Australia.

Postmortem diagnosis of respiratory disease

The vast majority of sheep suffering overwhelming respiratory disorders also suffer 'sudden death' or more accurately 'found dead'. It follows that accurate diagnosis depends on competence in the technique of carrying out a post-mortem examination, and in the selection of the correct samples, together with their preservation and transit (see Appendices 1 and 2).

It must be borne in mind that with the possible exception of SPA, MV, and perhaps the septicaemic form of pasteurellosis, postmortem lesions are not pathognomonic (see Figure 21.1). Accurate diagnosis depends on observation plus microbiological and histological examinations.

Gross pathology in respiratory disease

It has been stated that by the time a sheep has died it is too late for a post-mortem examination. Although this is an overstatement, the effect of the fleece in delaying heat loss after death, together with possible extremes of pyrexia in the final stages of infective conditions, often gives accelerated autolysis with severe limitations on the value of single necropsies. Some specific signs to check for during PME are as follows (see Figure 21.1).

Neonate (see also Chapter 8)

Check state of lungs

- Unexpanded, sinks in water, indicates stillbirth.
- Apparent consolidation but neutral gravity, indicates neonatal acute respiratory distress syndrome.
- Apparent lobular consolidation indicates incomplete expansion.
- Floats in water indicates full expansion therefore lamb has breathed.
- Pleural haemorrhage indicates parturient trauma.
- Miliary abscess formation indicates neonatal septicaemia.
- Anterior lobe consolidation and fluid in bronchioles indicates inhalation pneumonia.
- Congestion indicates possible acute pasteurellosis/viral infection. (If only one lung affected, may be hypostatic congestion.)

> At this age, pasteurellosis is usually septicaemic. Check for generalised septicaemia lesions – petechiation of epicardium, spleen, liver, kidneys and hyperaemia of abdominal and thoracic lymph nodes. As lambs get older the disease becomes pneumonic in character.

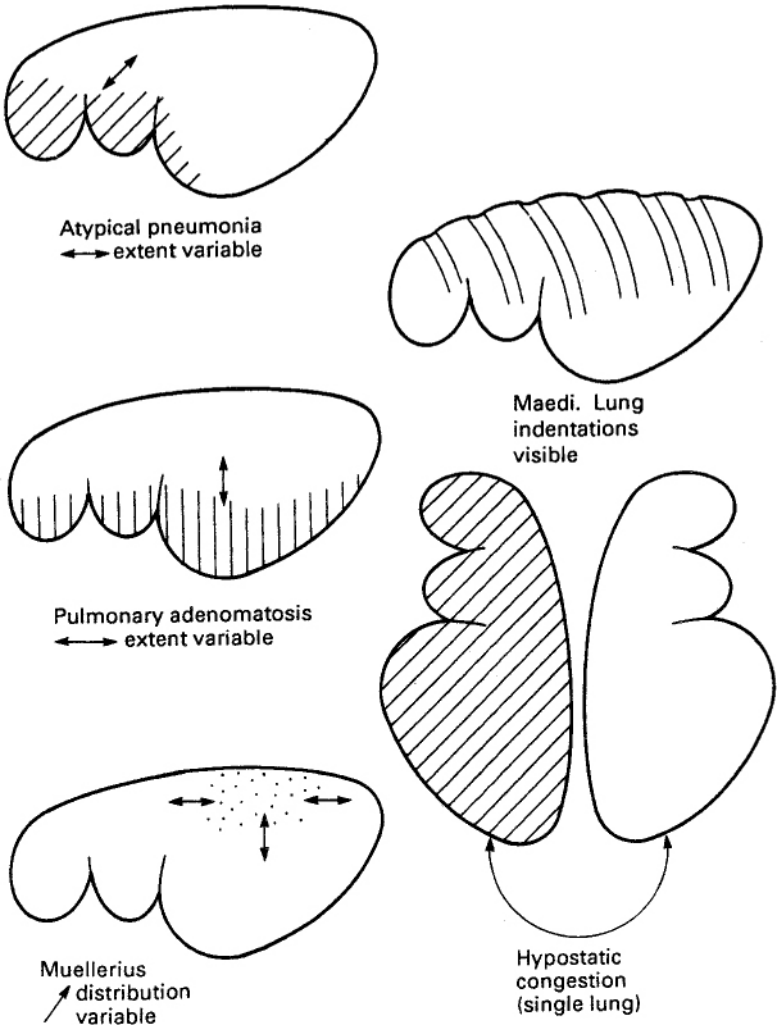


Figure 21.1 Distribution of lesions in respiratory disease.

Growing lambs

If there has been a delay between death and PME, the gross appearance of any lung tissue will frequently show a degree of reddening due to early autolysis; this is easily confused with the changes shown in early pasteurellosis (see colour Figure 35). In the case of the latter, there is more likely to be some fluid in the airways, and possibly evidence of generalised infection in other parts of the carcass.

Uniform congestion with darkened colour

Check for

- Peracute pasteurellosis (isolation and quantification of organisms from piece of lung tissue, plus histology).
- Delay between death and examination – autolysis.
- Viral pneumonia – possibility of PI3 infection.

Lobular consolidation

Check for

- Atypical pneumonia – grey to red raised areas, apical and cardiac lobes are particularly affected.
- Chronic pasteurellosis – edges discrete, possible necrosis, surface sunken.
- Inhalation pneumonia (history).
- After-effects of neonatal septicaemia – abscesses and chronic change at the interface with normal tissue.
- Parasitic pneumonia – emphysema and oedema, presence of worms in bronchi.

Subpleural nodules in caudal lobes

Check for

- *M. capillaris* (incidental finding only).

Single lung congestion

Check for

Hypostatic congestion, recumbency before death (may be other signs such as excoriation of limb or eye region).

Petechiation

Check for

- Peracute pasteurellosis (*P. trehalosi*) – upper respiratory tract will have similar lesions. Usually appears as death of a number of animals in autumn.

Pleurisy

This lesion is likely to accompany any infectious and febrile pneumonic condition, and the extent and degree of pleurisy will be an indication of the duration of the infection.

- Pleural cavity full of straw-coloured fluid with fibrin clots suggests peracute pasteurellosis.
- Thick green gelatinous exudate suggests subacute pasteurellosis.
- Total or near total occlusion of pleural cavity with widespread adhesions suggests chronic pasteurellosis.
- Fibrinous tags associated with clearly demarcated areas of consolidation suggests atypical pneumonia.

Adults

Many of the comments in the previous section will apply to this age group, but the important slow viral conditions will also feature prominently.

> Where lesions of acute pasteurellosis are found, a careful examination should be made for lesions of pulmonary adenomatosis, since these can easily be missed.

Lungs larger than normal

Check for

- Pulmonary adenomatosis tumour (vary in size from 0.5 cm to involvement of up to half diaphragmatic lobe) – solid, grey or purple in colour, clearly demarcated from normal lung unless masked by generalised pneumonia. Excess fluid in airways.

Lungs do not collapse, rib impressions visible

Check for

- Maedi (histology, serology).

Petechiae in upper respiratory tract

Check for

- Septicaemic form of pasteurellosis.

Inflammation and swelling of pharyngeal and laryngeal area

Check for

- Laryngeal chondritis, common in Texels.
- Drenching injury – check for tracks from pharynx.

Bronchial froth and fluid

This will accompany many conditions, even postmortem change. If excessive:

Check for

- Parasitic bronchitis.
- Inhalation pneumonia.
- Pulmonary adenomatosis.

As previously stated, with respiratory disorders confirmation of any causal organism or parasites will depend on laboratory tests such as microbiology, histology and parasitology. It must be again noted that *M. haemolytica* is a normal inhabitant of the upper respiratory tract, and that to establish this as a cause of pneumonia lesions it is necessary to isolate in large numbers from the lesion itself. PI3 may also be isolated from the upper respiratory tract but this does not necessarily indicate a direct involvement in any disease process.

22 Found dead and sudden death

Because of the nature of stock supervision in the sheep industry – at best twice daily in the lowland unit and infrequently in the high hill unit – what appears to be sudden death is usually simply death since the last inspection. It should, therefore, be more accurately described as 'found dead'. Apart from true sudden death caused by accidents, trauma or lightning strike, and some overwhelming bacterial infections, few deaths will be asymptomatic as presented by the owner, who may not wish to admit that he has not examined the stock as often or as thoroughly as he should.

Postmortem examination (the technique of which is described in Appendix 1) will obviously play a central role in any 'found dead' investigation. However, the rapid onset of autolysis, hastened by the presence of the fleece which delays cooling of the carcass, poses limitations on the value of this examination unless the time lapse is reasonably short. It has been said that the only valuable PME in the sheep is one carried out before death!

A record of findings should be kept but if there is any possibility of insurance claims or legal action, it is essential that careful and comprehensive details are kept. It is essential to obtain a full history, and if there is any suggestion of a flock problem, to examine thoroughly contact animals for any less advanced cases.

Important questions during history taking include:

- Age and sex of the animal – these will exclude some conditions.
- Grazing history.
- Feeding, roughage and concentrates, especially recent changes.
- Vaccination history, any recent vaccination.
- Recent drenching, with what? how much?
- Other recent stress – dipping, shearing, weaning?
- Recent close confinement?

Although suggestions are made of the most likely causes of 'sudden death' or 'found dead' in various age categories, this does not imply that other causes do not exist or that a thorough PME need not be carried out. Most of the following conditions are addressed elsewhere in the text where information is given on preventive measures, where possible.

Sudden death at any age

Obviously accidents, lightning strike, predation and trauma are no respecters of age.

Accidents

Accidents should be capable of diagnosis even if not witnessed, but it is necessary to beware of the 'contrived' accident where no external or internal evidence can be found, particularly where an insurance claim is involved.

Lightning strike

Lightning strike is also a popular candidate for insurance claims, often with considerable client pressure exerted. If lightning is suspected (by the clinician not just by the owner), then the circumstances of death must be investigated, with the body(ies) in the position where found.

Check for

- Weather at time of incident – with local meteorological office if necessary.
- Adjacent trees/buildings/metal fences.
- Scorching of grass.
- Evidence of external singeing or burns.
- Subcutaneous haemorrhages, sometimes in a branching pattern.

Other PM signs are not diagnostic, therefore a final diagnosis must rest on a combination of circumstances and signs.

Trauma

Trauma, particularly by predators such as dogs, is another common occurrence, again possibly involving insurance or legal aspects.

- Check carefully that injury was inflicted before death – there should be signs of bruising or haemorrhage into tissues adjacent to injury.
- Check for puncture wounds and other tooth marks, again with signs of bruising. The gap between tooth marks may help to indicate whether a fox or large dog was involved.

Predation is common once an animal has died if the body is not removed immediately, and it is not unknown for an unscrupulous owner to attempt to simulate predation as the cause of death on an already dead animal.

Common causes of sudden death or found dead

Neonatal lambs

For full details see Chapter 8. Most lamb deaths in the first few days after birth will be 'found dead' rather than true sudden deaths. Because of their susceptibility to hypothermia, however, the period during which symptoms of illness may be seen is likely to be short. Common causes include trauma, overlying/suffocation, exposure, starvation, lamb dysentery or neonatal septicaemia.

Young lambs (milk dependent)

The most common cause of death in this age group is starvation which, by definition, is not sudden death although the owner may think so.

Check

- Ewe milk supply.
- Body fat reserves in lamb, particularly brown fat.
- Abomasum for presence of milk.
- For disease predisposing to starvation, e.g. orf, neonatal polyarthritis, neonatal septicaemia.
- For nephrosis – there should be some warning signs but where very young lambs are affected death can occur quickly.

Other causes of death

Individual lambs dead

There are a small number of abdominal conditions in this age group which can cause rapid death.

Check for

- Vitamin E deficiency if lamb has a ruptured liver (selenium may be normal).
- 'Red gut', i.e. torsion of the mesentery involving the intestines except the first part of the duodenum. If seen alive, lambs are bloated and deteriorate over a very short time (30 min). PME shows very distended dark red intestines, and the site of the twist can be identified. This may be seen in lambs at grass but is probably more common in artificially fed or creep-fed lambs. It is thought to be due to rapid passage of rich gas-producing food into the intestine, producing instability of the intestines which then twist irreversibly, occluding the anterior mesenteric artery.
- Gastric torsion – this occasionally occurs in lambs before rumen development has begun.
- Bloat – this may involve the abomasum, or when developed, the rumen, usually in artificially fed lambs. Unless the lamb is seen alive with bloat, it is difficult to be sure whether bloating occurred before or after death.

Many lambs dead

Check for

- Clostridial disease – see below.
- Peracute pasteurellosis (*M. haemolytica*). This is probably the most common cause of well-nourished lambs being 'found dead' and often occurs at 3–4 weeks of age as maternal antibody wanes. The most common findings are signs of septicaemia – petechiation of heart, liver, spleen and kidneys, and

swelling and hyperaemia of cervical and thoracic lymph nodes. There is often no pneumonia or pleurisy in this young age group. Diagnosis can be confirmed by submitting a selection of tissues for bacteriological examination.
- Acute abomasitis - *M. haemolytica* has been isolated from the mucosa but not from other organs in these cases.

The beginning of outbreaks of severe coccidiosis and/or nematodiriasis may be signalled by lambs 'found dead', but careful examination of the remainder of the group should reveal others showing clinical signs of infection (see Chapter 10).

Check for

- Lesions of coccidiosis in lower small intestine, caecum and colon – raised white spots, mucosa thickened and inflamed. Smears of intestinal scrapings show developing stages of coccidia.
- Immature *Nematodirus* in small intestine – slender worms about 2 cm long which when coiled together can look like cottonwool.

Growing and store lambs

In this group, sporadic deaths can occur from a variety of causes, but the most important conditions are those which cause a series of deaths if not diagnosed initially and preventive measures taken.

Individual lambs dead

Check for

- Abdominal catastrophe – red gut or other acute obstruction.
- In males unobserved urolithiasis and ruptured bladder.
- Nephrosis.
- Dosing gun injury if recently drenched.
- Inhalation pneumonia if recently dipped.
- Heart muscle for white streaks indicating nutritional myopathy.

Many lambs dead

Changes in feeding often trigger flock outbreaks of disease in this age group. This is particularly true of pulpy kidney, which usually follows change to lush pasture or sudden grass growth after rain. There is also a suggestion that pasteurellosis follows feed changes. Remember FMD can cause sudden death in lambs. See Table 22.1 for details on clostridial diseases.

Check for

- Systemic pasteurellosis (*P. trehalosi*) – characteristic necrotic lesions of the tongue, pharynx, oesophagus and sometimes the abomasum and intestine are usually present, together with subcutaneous and subpleural haemor-

rhages, enlargement of tonsils and retropharyngeal lymph nodes, and necrotic foci in liver and spleen. Take samples of lesions and swabs of ulcerations for bacteriological examination. Diagnosis is confirmed by isolation of large numbers of organisms and serotyping.

- Pulpy kidney (enterotoxaemia caused by *Clostridium perfringens* type D, Table 22.1) – in fresh carcass, kidneys are pale and swollen but autolysis is rapid, giving the disease its name. Other findings are excess pericardial fluid, and haemorrhages in the heart. Smears can be made of small intestine and kidney for FAT. Check also urine sample for presence of glucose (there is usually sufficient to moisten a test stick even if the bladder appears empty). Check vaccination history. Maternal antibody wanes after 12–16 weeks. Many lambs are unprotected after this age or receive a single vaccination which is inadequate to ensure full protection.
- Braxy (*Cl. septicum*) (Table 22.1) – occurs particularly in frosty weather. PME shows acute inflammation of the abomasum, excess fluid in the abdomen, and rapid autolysis. Confirm diagnosis by FAT on smear from abomasal lesion.
- Acidosis if fed concentrates, or access to concentrates or grain – examine rumen contents for undigested foodstuffs, and check pH (normal is 5.5, less than 4.5 indicates cereal overeating).
- Acute liver fluke especially in wet season if grazing poorly drained land. Acute disease occurs about 6 weeks after ingestion of large numbers of metacercariae. PME shows enlarged liver with haemorrhagic tracts, often covered with fibrinous exudate, bloodstained fluid in abdomen.
- Black disease (Table 22.1) (*Cl. oedematiens* type B) – rapid putrefaction, oedema of abdominal wall, dark engorged liver with areas of necrosis, associated with migrating immature fluke. Check remainder of group for clinical cases – anaemia, enlarged painful abdomen.
- Other acute parasitic infection – *Nematodirus, Haemonchus, Teladorsagia*. In each of these cases, the observant shepherd should note warning signs before deaths occur.

Adults

Although a large proportion of ewe deaths occur in the periparturient period (see Chapter 6), there is still a wide variety of conditions which may kill ewes or rams at any time of year. It is important again to emphasise the difference between 'sudden death' and 'found dead', and an assessment of the body condition of the animal will usually give a good guide to the presence of disease of some duration. Other vital pieces of evidence are:

- Position of carcass when found.
- Access to poisonous plants, mouth contents, rumen contents.
- Vaccination history.
- Recent feeding or management changes.
- Recent handling, e.g. shearing, dipping, drenching.

Although anthrax rarely appears to be identified as a cause of sudden death in sheep, the possibility should not be totally ignored, especially if a very enlarged spleen is found during PME.

Table 22.1 Clostridial diseases causing rapid death.

Disease	Organism	Age group usually affected	Predisposing factors
Lamb dysentery	*Cl. perfringens* type B	Neonatal lambs <2 weeks	Unvaccinated ewes
Pulpy kidney	*Cl. perfringens* type D	Growing lambs	Waning maternal immunity Fast growing lambs Feed change Lush grass
Braxy	*Cl. septicum*	Store lambs	Unvaccinated Frozen feed
Black disease	*Cl. novyi* type B	Store lambs Adults	Unvaccinated Migrating immature fluke
Struck	*Cl. perfringens* type C	Adults	Unvaccinated Lush grass
Blackleg	*Cl. chauvoei*	Growing lambs Adults	Unvaccinated Wounds Injuries
Abomasitis	*Cl. sordellii*	Growing lambs Adults	Feed change

Individual deaths

Probably the most common cause is 'cast on back', i.e. the sheep is in such a position that it cannot sit or stand up. This most often affects fat sheep in heavy fleece, and bloat is usually the actual cause of death. There is always a pile of faeces behind the animal, indicating the length of time for which it was lying in that position. It is essential to see the carcass *in situ*, in order to make a valid judgement as to whether this was likely to be the cause of death.

If recently handled for lambing, dipping, shearing, drenching, vaccination, etc.

Check for

- Sudden catastrophe, e.g. ruptured major blood vessel, heart lesion.
- Precipitation of metabolic disease (hypocalcaemia, hypomagnesaemia – see below).

If lactating ewe

Check for

- Acute (gangrenous) mastitis.
- Hypomagnesaemia – see below.

If one of group of rams

Check for

- Broken neck caused by fighting. This is particularly common at the beginning of the breeding season or when new rams are introduced. It may also occur in an established group after shearing or dipping.
- Clostridial infection of wounds from fighting, i.e. head wounds.

Many deaths

Where deaths are occurring in sufficient numbers to constitute a flock problem, the causes fall most commonly into four categories:

- Bacterial (*Clostridia, Mannheimia, Pasteurella*).
- Metabolic (hypocalcaemia, hypomagnesaemia).
- Parasitic (acute fluke, *Haemonchus*).
- Toxic (copper, plants).

Ewe deaths have also been reported after prelambing vaccination.

If deaths follow vaccination

Check for

- Poor vaccination technique, sterility of equipment, use of new needles.
- Use of previously opened vaccine packs.
- Vaccinating wet or dirty animals.
- Concurrent metabolic disease, e.g. hypocalcaemia, pregnancy toxaemia – these may have been precipitated by temporary removal from food source, or handling.

- Concurrent administration of anthelmintic, flukicide. It may be advisable not to administer more than one drug at a time.
- True hypersensitivity reaction – pulmonary oedema and pleural effusion may suggest this. Consult manufacturer of suspect drug.

If no obvious lesions found at PME

Check for metabolic disease

- Hypocalcaemia is most commonly seen in late pregnancy. It can occur occasionally at times other than late pregnancy, and in nonpregnant animals, when it is precipitated by severe stress, e.g. long transport, driving long distances.
- Hypomagnesaemia occurs usually in early lactation, especially in ewes on lush pasture rearing twins.

> Aqueous humour is useful for sampling and should be stable for up to 48 h. Cerebrospinal fluid may also be used and is stable for up to 12 h after death.

If lung lesions found

Check for

- Acute pasteurellosis (see Chapter 21). Care must be taken to distinguish between hypostatic congestion and pneumonia but in acute pasteurellosis, areas of necrosis are often found in consolidated areas of lung, and pleurisy and pericarditis are usually present. Confirmation requires isolation of large numbers of organisms from lung tissue, and histological examination.
- Masking of lesions of pulmonary adenomatosis especially if animal is in poor condition.

If liver lesions are found

Check for

- Acute liver fluke (see above).
- Black disease in conjunction with acute fluke (see above).

If jaundice is present

Check for

- Copper poisoning (see Chapter 18).

If carcass is pale and oedematous

Check

- Abomasum for *Haemonchus contortus*. Worms are easily visible (2–3 cm), plus haemorrhagic gastritis is present.

If autolysis is very rapid

Check for

- Clostridial disease (vaccination history?). Struck (*Cl. perfringens* type C) shows excess abdominal fluid, hyperaemia and ulceration of small intestine. Confirm by smear for FAT or toxin neutralisation test with intestinal contents. Blackleg (*Cl. chauvoei*) features acute necrotising myositis. Often the site of the main lesion can be identified by crepitus from gas in the tissues which are dark red and haemorrhagic with a characteristic rancid smell. Diagnosis is confirmed by FAT on smears or frozen sections of affected tissue.

If plant poisoning is suspected

Check

- Mouth contents – yew causes true 'sudden death' by the action of the alkaloid taxine, which acts directly on the heart.
- Rumen contents – remains of plants such as rhododendron or acorns may be visible. Neither of these kill instantaneously so signs of illness should be seen in others exposed to same environment.

> Rhododendron poisoning is the most likely cause of 'vomiting' in sheep. (Excess intake of linseed meal, and acidosis, have also been reported as causes of vomiting.)

Other toxins which may cause death include

- Brassicas which may cause haemolytic anaemia.
- High nitrate content in kale may cause nitrite poisoning (nitrate is converted to nitrite in the rumen). The distinguishing feature is methaemoglobinaemia.
- Acute lead poisoning – death is usually preceded by fits. PME shows grey musculature, liver degeneration, and gastroenteritis. Submit liver and kidney for analysis.
- Phenols present in some dips, disinfectants, creosote, etc. are toxic and can often be detected in a carcass by smell. Signs of an irritant poison are present

if ingestion has occurred, or severe pneumonia if incorrect dipping has taken place. Samples for analysis should include stomach contents, lung, liver, kidney, urine and blood. If immediate transport to a laboratory cannot be arranged, samples can be frozen.

See Appendix 6 for information on poisoning.

Appendix 1
Postmortem examination

It is either an indictment of the sheep industry, or a reflection of the 'death wish' of sheep, that a considerable amount of disease diagnosis in this species depends for a final verdict on material obtained at PME. However, this is often an undervalued technique in practice, carried out in a cursory 'slash and glance' manner, stopping when one gross lesion is found. It *should* be carried out in a careful, scientific and logical manner, so that all organs are examined in order to gain maximum information and to select samples that give the greatest opportunity to obtain a correct diagnosis. It must be stressed that any PME should be performed as soon after death as possible, if autolytic change is not to complicate or mask any pathological change.

Stages in a postmortem examination

(1) External examination
Identity
Age
Sex
Weight
Time since death
Body condition
Fleece state
Mouth, nose, eyes, mucous membrane
Udder, vulva/scrotum, prepuce
Anus

Discolouration of the ventral abdomen is not an indication of infection, simply of autolysis, which may be evident within 24 h of death, or earlier in hot weather.

(2) Place the animal on its back, or on its side (this is easier with a thin animal). Cut through and reflect the skin from mandible to pubis (see colour Figure 36). Cut into axilla and groin on both sides if on back, or on upper side if on side, to free the close attachments of the legs.

(3) Examine subcutis, superficial lymph nodes, udder or penis and testicles.

(4) Cut through abdominal wall, taking care to avoid puncturing the rumen, and expose abdominal organs (see colour Figure 37). Examine *in situ*. Note amount of fat in mesentery.

(5) Cut through ribs along each side with saw or shears (see colour Figure 38) to expose thoracic organs (see colour Figure 39). Examine *in situ*.
(6) Remove alimentary tract by cutting through rectum and separating from roof of abdominal cavity forward until oesophagus can be cut as it passes through diaphragm. Place organs to one side.
(7) Remove liver and spleen and place to one side.
(8) Cut inside mandibles to free tongue, reflect with oesophagus and trachea, and remove with lungs and heart. Put to one side.
(9) Examine pleural cavity.
(10) Cut through cheeks and disarticulate jaw, examine incisor and molar teeth.
(11) Examine and remove uterus if present.
(12) Examine and remove kidneys. Note amount of perirenal fat. Examine bladder, collect urine if necessary.
(13) Examine and open several joints.
(14) Return to thoracic organs. Open oesophagus, trachea and bronchi. Examine mediastinal lymph nodes. Palpate and cut into lung substance. Open pericardium. Open both sides of heart and great vessels.
(15) Return to abdominal organs. Separate rumen, reticulum and omasum from abomasum. Open, examine contents and save if necessary. Wash and examine mucosa.
(16) Separate abomasum from duodenum. Open, examine contents, save if necessary. Wash mucosa and examine.
(17) Examine small intestine, caecum and colon. Open, save contents if necessary, examine mucosa.
(18) Examine mesenteric lymph nodes.
(19) Examine and incise liver, spleen and kidneys.
(20) Remove head by disarticulating atlanto-occipital joint. Reflect skin. Make cuts with saw as indicated in colour Figure 40. Lever off top of skull. Reflect dura and free tentorium cerebelli. Invert, and carefully remove whole brain. Examine surface but do not cut into substance until fixed.

Agonal and postmortem change

Agonal changes take place immediately before death and are due to circulatory failure. The most common change of which to beware in this category is hypostatic congestion of the lower lung, which may be confused with pneumonia. If there is any doubt, a sample of lung tissue should be taken for bacteriology and histology (including quantification of *Mannheimia* (*Pasteurella*) – see Chapter 21).

If barbiturates are used for euthanasia, parts of the small intestine are often found to be dilated and congested.

The most rapid postmortem change in sheep is bloat, which develops rapidly, making a diagnosis of death from bloat extremely difficult.

Decomposition is rapid in warm weather or if the animal has a heavy fleece. In cold weather it will be much slower, making PME useful for a longer period

after death. If clostridial infection is involved, decomposition will be rapid anyway.

Checklist of organs

External examination, wool, skin.
Subcutaneous tissue.
Alimentary tract – teeth, tongue, oesophagus, rumen, reticulum, omasum, abomasum, small intestine, caecum, large intestine, anus.
Respiratory system – larynx, trachea, bronchi, lungs, pleura.
Cardiovascular system – pericardium, heart.
Urinary system – kidneys, bladder, urethra.
Genital system – uterus, ovaries, fetuses and placenta if present, vagina, vulva, udder/scrotum, testicles, penis, prepuce.
Musculoskeletal system – muscles, joints, feet.
Lymphoid system – spleen, lymph nodes, bone marrow.
Nervous system – meninges, brain, spinal cord, eyes.

Appendix 2
Sample taking and sample sending

Submission of incorrect samples is a waste of time and money for the client and the clinician, and may result in the loss of potentially valuable material from the point of view of diagnosis. Most laboratories now supply kits of sample containers, transport media, etc., and will be only too pleased to give advice on sample submission if asked. If any unusual samples or requests are to be submitted, prior consultation is essential.

Packing and posting samples

Written details with practice name, identity of the animal, owner, history, and specific requests for tests required should be enclosed in a polythene bag for protection. Post Office regulations state that pathological specimens must be enclosed in a sealed receptacle which itself must be securely enclosed in a strong outer container so that it cannot move about. If necessary, the receptacle should be enclosed in a polythene bag with a sufficient quantity of absorbent material to prevent possible leakage. The packet should be marked 'Fragile with care' and 'Pathological specimen', and must be sent by first class post, not parcel post. Any packet not packed and marked as above, or found in parcel post, will be destroyed. Anyone not complying with these regulations is liable to prosecution.

Samples for haematology

EDTA is the anticoagulant of choice. Heparin and OxF are not suitable for a full haematological examination. Care should be taken to use the correct size of container for the volume of blood to be collected, i.e. do not put a small volume of blood in a large vacutainer, as the excess of anticoagulant can distort the results.

Samples for enzymology

Serum or plasma (with heparin as anticoagulant) are suitable for most tests, except where red cell enzymes (GSH-Px, SOD, TK) are to be measured, when a heparinised sample is essential. If a clotted blood sample is obtained, it is preferable to separate the serum before submission, as haemolysis may interfere with the tests. Samples should be submitted as quickly as possible (cer-

tainly in less than 36 h), since some enzymes have a short life. SDH must be assayed within 4 h of collection.

Enzymes which may be helpful in ovine clinical diagnosis

- Creatine kinase (CK) – this is raised in skeletal muscle damage, e.g. in nutritional myopathy (muscular dystrophy, white muscle disease). However, this enzyme has a short half-life within the body, therefore sampling should be done early in the course of a disease.
- Aspartate aminotransferase (AST) – raised in muscle damage, and acute liver damage, but needs other tests to support.
- Glutamate dehydrogenase (GLDH) – raised in hepatitis; in conjunction with raised AST indicates acute liver damage.
- Gamma glutamyl transferase (GGT) – raised in chronic hepatitis, particularly with bile duct damage, e.g. chronic liver fluke. May also be raised in cases of pregnancy toxaemia.
- Sorbitol dehydrogenase (SDH) – raised in acute hepatitis and is liver specific. *Must* be assayed within 4 h of sampling. Requires heparinised blood.
- Glutathione peroxidase (GSH-Px) – is used as an indicator of selenium (vitamin E) status.
- Superoxide dismutase (SOD) – is an indicator of prolonged copper deficiency.
- Transketolase (TK) – is a specific indicator for CCN.

Samples for biochemistry

These are most likely to be helpful on a flock rather than on an individual basis. A minimum of six samples from representative animals should be collected. In the case of individual animals suffering from a suspected metabolic disease, it may be wise to collect pretreatment blood samples which will then be available in the event of failure to respond to treatment. Aqueous humour or CSF may be useful in dead animals, especially for calcium and magnesium estimation.

Tests requiring serum or plasma (heparinised)

Calcium, magnesium, beta-hydroxybutyrate, urea, creatinine, total protein, albumin, globulin, copper, vitamin B_{12}.

Tests requiring anticoagulant (potassium oxalate/sodium fluoride)

Inorganic phosphorus, glucose.

Samples for bacteriology

Fresh samples should be taken and submitted as quickly as possible:

- Pieces of tissue, e.g. liver, spleen, lung, lymph node, etc. should be placed in sterile containers.
- If swabs are to be used, these should be of cottonwool with, if possible, transport medium (without antibiotics). Unprotected swabs dry out and are useless – these should be placed in a protective container. *Mycoplasma*, *Campylobacter*, *Leptospira* and *Chlamydophila* (unless in placenta) have particular requirements – contact laboratory for advice before sending.
- If anaerobes are suspected, air should be excluded from samples by wrapping with cling film. If *Clostridia* are suspected, dried smears of lesions may be useful for FAT.

Samples for virology

Take advice from laboratory except for:

- Orf – dried scab is required.
- Border disease – requires blood clot (keep cool and make rapid submission), nasal swab in virus transport medium, serum from affected and contact animals.
- Rotavirus – faeces sample is required (rectal swab is not adequate).

Samples for serology

Single samples are useless, except with EAE where a high post-abortion titre is significant. In other cases paired sera are required, but the first sample must be taken early in the disease process, otherwise seroconversion may have already taken place.

Samples for abortion enquiry

A representative number of samples from an affected flock should be submitted, i.e. at least 10% of abortions.

- Whole placenta or piece of placenta with cotyledons from an area showing pathological change, if present.
- Fetus(es) or vacutainer of stomach contents plus vacutainer of pleural fluid plus piece of fetal liver.
- Vaginal swab if placenta not available.
- Serum sample may be helpful in some cases. Aborting ewes should be marked so that a blood sample can be obtained later if required.

Samples for histology

Take small pieces of fresh tissue less than 1 cm thick (except in the case of brain which should be left whole) and place in a wide-mouthed container (if a narrow container is used it may be impossible to remove the fixed sample without breaking either the container or the sample). Use about 20 parts fixative to 1 part of tissue to ensure rapid penetration.

A suitable fixative is 10% neutral buffered formalin:

100 ml formaldehyde 40% (formalin)
900 ml distilled water
6.5 g disodium hydrogen phosphate (anhydrous)
4 g sodium dihydrogen phosphate monohydrate

or formol saline:

100 ml formaldehyde 40% (formalin)
9 g sodium chloride
900 ml tap water

Samples for toxicology

Consult laboratory if any unusual toxin is suspected. Take particular care if the case is of a nature where litigation may be involved. In this case, duplicate samples should be taken and placed in containers which are sealed in the presence of a witness, if possible.

Helpful samples may include:

- Blood (oxalated) and faeces from live animals.
- Stomach contents, liver, kidney, brain, fat, muscle from PME.
- Food material if suspected (1 kg if available).
- Any suspected poison source.
- For copper poisoning take liver and kidney (100 g).
- For fluorine take urine and bone (tail or rib).
- For lead take liver and kidney (100 g).
- For molybdenum take liver (100 g).
- For organophosphorus/chlorine compounds take brain, fat, stomach contents, liver and kidney.
- For monensin, take food sample plus contents label.

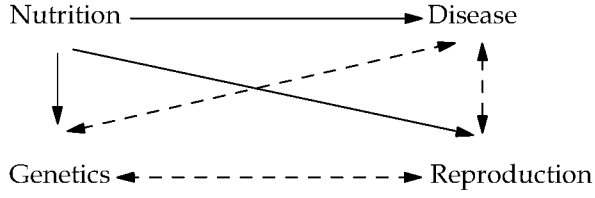
Samples for parasitology

- For worm egg counts, fluke egg counts and coccidial oocyst counts, 10 g of faeces in a plastic container are required for each test. Counts may be carried out on smaller amounts, but these may be less accurate. However, it is realised that there are practical difficulties in obtaining large amounts from small scouring lambs.
- For worm counts, it may be easiest to deliver the whole of the alimentary tract (suitably ligated at each end). It is possible to empty out separately the contents of the abomasum and small intestine, but these require washing through to make sure that all worms have been harvested. A small amount of formol saline should be added if the samples are not to be delivered to the laboratory on the day of collection.
- For cryptosporidia, 5 g of faeces in a plastic container is required.
- For abomasal parasitism, pepsinogen can be measured with 7 ml clotted blood.

- For skin conditions, macroscopically visible parasites should be submitted in a clean dry bottle. A skin scraping from the edge of an active lesion should also be submitted in a clean dry bottle.
- For ringworm and *Dermatophilus* infections, scabs, skin scrapings, and fibres from the edge of an active lesion should be submitted in a clean dry bottle.

Appendix 3
Nutrition

There are four main areas influencing production from a flock – nutrition, genetics, reproduction and disease. Of these, nutrition is the most significant, having not only direct effects on output, but also significant influence on the three other factors.



For example:

- The disease itself may be directly of nutritional origin, for example pregnancy toxaemia.
- The full expression of genetic potential is essential for maximum profitability and is highly dependent on adequate nutritional inputs.
- Ovulation rate, fetal growth and lactation potential are all highly nutrition dependent and can be seriously compromised by inadequate inputs (see SORP, Chapter 2).
- Both the immediate response to a disease episode and degree of resistance to it, for example to parasitic challenge or an infectious agent, often depend on nutritional status at the time of challenge.

However, managing the nutrition of sheep presents great problems to both the producer and the clinician. Production methods vary from the semi-feral of small island populations, through the 'high hill' flocks where ewes are in negative nutritional balance for much of the production cycle except for the period between weaning and tupping, to the very intensive high input systems of terminal sire-producing flocks. Complicating matters still further, in contrast to cattle, pigs and poultry, there are large variations in body weight between commercially viable breeds. Variations in fleece cover affect metabolic rates, and variations in carcass conformation with wide discrepancies in fat cover add to the difficulty of arriving at any standard formulae.

The prime aims in management of nutritional inputs should be:

- Maintenance of body condition score of adult sheep between 2.5 and 4 throughout as much of the production cycle as possible.
- Avoidance of any nutritional constraint on growth rate of lambs through both milk and grass dependence.

To achieve this it is essential that both client and clinician have an understanding of the inputs required in order to maintain these aims, and, in particular, an understanding of the constraints imposed by the physiology of the ewe in late pregnancy. Clinicians cannot expect to have the requirements for all the systems and breed types at their finger tips, therefore some general guidance on commonly used feeds is included at the end of this section.

Effect of nutrition throughout the production cycle

If the effect of nutrition is examined throughout the production cycle, it is apparent that few problems are divorced from the problem of suboptimal nutrient intake. From before mating right through into lactation, inadequate nutrition has a major impact on production.

Mating time and early pregnancy

Ovulation rate (and therefore lambing percentage) is directly influenced by condition score. For lowland flocks the target condition score will be 3.5–4, for hill flocks 2.5–3. The upper limit of ovulations will be the genetic potential of the breed or cross and this must not be compromised by restraint on nutrient intake. (Although in certain special circumstances, for example in the case of the Cambridge breed which can produce up to 10 ova at a single oestrus, large 'litters' may not be welcomed by the average shepherd.) Optimum intake must be maintained not only throughout the mating period, but until implantation is fully completed by 5–6 weeks post-mating. While early fetal loss rates as high as 25% are quoted as the result of stress, the simple and significant effect of suboptimal body score are illustrated below:

	Scottish Blackface		*North Country Cheviot*	
Body score	1.5	3	2	3
Embryo loss	50%	15%	40%	18%

(Figures from Department of Agriculture for Northern Ireland)

Mid-pregnancy

After implantation, the fetus itself is at little risk from nutritional disadvantage although placental growth may be affected, with over-nutrition adversely affecting placental size and therefore lamb birth weight. A very mild reduction in nutritional status can maximise placental size during this period, the placenta being fully developed by 3 months gestation. If there is either a supply or economic constraint on availability of nutrients, this is the only period in which

these can be allowed to influence the feeding regimen. It must be noted that any such constraint cannot be allowed to significantly affect body score (certainly not more than a loss of half a condition score), since any loss will be almost impossible to correct during the period of maximum fetal growth during the last 6–8 weeks of pregnancy.

Late pregnancy

The last third of pregnancy requires careful monitoring if adequate intake is to be maintained. Some 90% of fetal growth takes place during this phase, with 60% occurring in the last month. During the last 6–8 weeks of pregnancy the rapid increase in the volume of the uterus and its contents creates significant competition for abdominal space. The reproductive tract comes to occupy 60% of total available space, reducing the space available for the rumen. In late pregnancy, this reduction in space for the rumen leads to reduced DM intake from 3% of body weight to below 2% in extreme cases.

If M (MJ) is the basic maintenance requirement for energy, a ewe carrying twins requires 2 M by full term. The crude protein content of the diet should be at least 16%, with 30% of this in digestible undegradable form (DUP). For a 70 kg ewe, maintenance requirement is approximately 10 MJ/day; by 4 weeks from lambing this is at least 16 MJ/day and by lambing the requirement is about 20 MJ/day. These intakes are essential if optimum lamb birth weights are to be achieved, maximising lamb survival, and also to allow optimum mammary tissue development in order to achieve full lactation potential. Providing and maintaining this requires the availability of sufficient high quality roughage and concentrates.

It is essential therefore that the feed provided must have the following characteristics:

- Roughage high in dry matter (>30%).
- Total diet highly digestible (>60% digestibility).
- Concentrates high in energy density (13 MJ/kg DM).
- High in crude protein (>16%).

While good hay or silage should supply these requirements, inferior grades will not. The perennial problem for the sheep keeper is that the ewe and lamb crop will consume the 'first cut equivalent' after turn out. Hay and silage made later in the season will have both lower digestibility and lower protein content. Intake will also be adversely affected if roughage is too coarse and fibre length long.

Lactation

The high nutritional demand phase continues into lactation. Energy demands actually rise to 3 M for a ewe with twins or triplets. A 5 kg lamb will have a milk intake of 1 litre/day, so that a prolific hybrid ewe will, relative to body weight, have a lactation demand equal to that of the modern dairy cow. In addition, milk production appears to be highly correlated with high protein intake in

both late pregnancy and lactation itself. A diet of 18% CP may be required for prolific ewes. It is false economy to limit expenditure at this stage. Many low priced concentrate feeds are available, but both very low price and an ash content of 10% or over should be danger signs.

If food availability and quality are not limiting factors, milk quality is likely to be adequate since variations in total solids do not have the same significance as in dairy production. Food conversion rates in lambs of 1:1 in terms of dry matter intake can be achieved during early milk dependence, so nutrition of the ewe must not be allowed to limit the full expression of genetic potential of the lamb. Indeed, growth rate from birth to 8 weeks is a standard indicator of both genetic potential and management efficiency.

Grass dependence

By 6 weeks of age the lamb is converting rapidly to grass dependence and by 8 weeks is effectively a herbivore. Reduced growth rates (ill thrift) at this stage are very common and are likely to be multifactorial in origin. Complex physiological interactions may be involved and the clinician is presented with the problem of making a correct diagnosis and perhaps the greater problem of effective treatment and prevention. See Chapter 7.

In most commercial enterprises this phase is timed to coincide with maximum grass growth when digestibility, protein and energy content are also high. Unfortunately, dry matter content may be low, leading to scouring unassociated with intestinal parasitism. In addition, wet spring weather can cause spoilage of grass, resulting in utilisation of less than 50% of the sward. A combination of these factors can give true intake levels much below that which would appear to be the case. Thus reduced growth rates at this stage may be unrelated to the presence of disease.

This is also the period during which micronutrient shortfall in herbage will start to affect lamb growth. The status of the ewe flock should be known from metabolic profiles and should thus have been corrected, but where both grazing and stored forage derive from the same pastures, the effect of any deficiencies will be aggravated and cumulative.

Weaning to mating

Nutritional control is central to effective 'cut off' of lactation at weaning. Since this is the point at which many cases of mastitis originate (but are often unrecognised), prevention of continued milk production when demand from the lamb has ceased is essential. A short 3–5 day period of low nutritional value feed intake is very effective and can be achieved by housing and feeding straw only, or putting ewes on very bare pasture if housing is not possible. At this stage the lamb crop must take priority for access to quality grazing in order to achieve maximum possible growth rates.

After weaning is completed, the flock should be divided according to body condition score in order once again to achieve the desired score at mating.

Those ewes that are too lean may require supplementation to achieve the desired body score (a period of 3–4 weeks on good quality inputs is required to add half a condition score). Those in adequate condition can simply 'tick over' while a few may require continuing restriction on intake if they are too fat (a body score of over 4 will reduce fertility).

Notes on standard feedstuffs

Silage

- Dry matter content can be estimated by the 'squeeze test':
  - liquid drips without pressure = less than 20% dry matter
  - liquid drips with mild pressure = less than 25% dry matter
  - liquid drips with significant pressure = approximately 30% dry matter
  - no liquid obtained with firm manual pressure = over 35% dry matter
- D value (digestibility) – good silage should have a D value of 65–70, but silage made from very mature grass will be below the 60 D value needed in late pregnancy.
- Crude protein – this can vary from as low as 8% to as high as 20%. It is linked to D value – the more mature the crop (i.e. the lower the D value), the lower the crude protein content.
- Energy – good silage may be as high as 11.5 MJ/kg DM, but poor samples may be as low as 8 MJ/kg DM. This largely depends on the stage of growth at which the silage was made, with energy content falling rapidly as the crop matures.
- Fibre length – intake may be reduced if silage is made from grass of long fibre length. This is a particular problem with big bale silage, in contrast to precision chop silage block cut from a clamp.
- Spoilage – poorly fermented silage or that contaminated with fungal growth, as well as being unpalatable is dangerous to feed to sheep because of the risk of listeriosis. This can be a particular problem with big bales, especially if the wrapping is not air tight, and also may affect the edges of clamps. Fresh silage should be provided frequently, with uneaten parts cleared away (this is usually safe to feed to beef cattle). No more than 2 days' supply should be given otherwise secondary fermentation and spoilage will occur.

Hay

- Dry matter – good hay will be over 85% dry matter. Below this there will be spoilage with mould growth and risk of mycotoxin production.
- D value – this will be about 60 in good samples, but falls with maturity of the grass and the effect of weathering during making.
- Crude protein – this can be up to 14% in good samples but as low as 8% in a poorly made crop.
- Energy – good hay can contain as high as 10 MJ/kg DM, with poor samples as low as 7 MJ/kg DM.

Straw

Straw can be used as roughage, even in late pregnancy, although concentrate allowance will need to be increased by 30% over that needed if hay or silage is fed. Ewes appear to find well-made barley, oat and wheat straw palatable, but it must be provided 'ad lib' so that the sheep can select the most palatable bits, discarding the poorest parts as bedding. There may be a need for increased phosphorus with straw-based diets. Straw is widely used as a base for pelleted 'complete' diets for finishing lambs.

Concentrates

Here the issue of variability should not arise as a list of raw materials and the analysis should be freely available from the compounder. There is serious competition based on price. The purchaser should examine the analysis to ensure that low price is not synonymous with low quality. Products with ME below 12 MJ/kg DM, crude fibre over 10% and ash over 10% should be avoided whatever the price.

Home mix

It is unlikely that clinicians will be involved in the formulation of home mixed feeds unless they have demonstrated expertise in this field. They may, however, be required to comment if a feed-related problem arises. Most home mixes are based on home grown cereals with a protein/mineral balancer added. If the instructions on inclusion rate of balancer are followed, there should be little problem. Other protein sources include soya (over 40% CP) and maize gluten (approximately 20% CP). Occasionally there are problems with palatability of maize gluten.

Cereals should be left whole or rolled only. If they are ground, this can lead to acidosis in greedy feeders. Normally whole grain is simplest to feed, although some may pass through undigested in the faeces. This loss must be balanced against the cost of rolling. If high quality silage is being fed, rolled grain is preferable because of a more rapid transit time of highly digestible feed through the gut.

Body condition scoring (see Table A3.1 and Figure A3.1)

This well-recognised technique is central to estimating the adequacy of current nutritional inputs and is very important because, as already stated, nutrition has a huge influence on level of production in any sheep unit. It is therefore crucial that the clinician has some understanding of the technique. Experienced sheep keepers will be able to quickly and accurately give a measurement to 0.5 of a condition score. This is beyond the requirements of the abilities of the clinician, but it is important that each clinician:

- Recognises what s/he means by each score.
- Can reliably repeat estimations.

- Recognises differences in conformation, musculature and fat cover between extreme breed types

Body scoring is carried out by feeling over the loin and assessing how easily the lumbar vertebrae can be felt, together with the amount of muscle and fat overlying the transverse processes. Assessment of fat cover over the ribs and tail head is not used in body scoring – this is reserved for selecting finished lambs for slaughter.

Macro-elements and micronutrients

The problems of identifying, treating and preventing both macro-element and micronutrient deficiency are very complex and, complicating matters still further, the same element can cause deficiency and toxicity, for example, copper and selenium.

The major factors complicating diagnosis include:

- The often added difficulty of interactions between factors to take into consideration, for example calcium, phosphorus and magnesium, selenium and vitamin E, copper and molybdenum.
- Intake levels will rarely be known since there may be differential uptake by different plant species or even strains of plants, for example magnesium content of grass on which animals are grazing, so soil analysis may not give a definitive answer.

Table A3.1 Definitions of body condition scores.

Score	Description	Spinous and lumbar processes	Lumbar processes, ventral surfaces	Lumbar muscle mass	Lumbar fat
1	Emaciated	Individually identifiable, sharply defined	Easily palpable	Little or no muscle present	No fat present
2	Lean	Individually identifiable, sharply defined	Palpable	Muscle present but surface concave	Little fat present
3	Optimal	Less prominent, smooth and rounded	Palpable with mild pressure	Normal muscle mass present, muscle surface flat to convex	Some fat cover detectable
4	Fat	Moulded but identifiable with pressure	Ends palpable only with significant pressure	Muscle mass fully developed, surface convex	Significant fat cover
5	Grossly fat	Not identifiable	Not palpable	Masked by deep fat	Deep fat cover

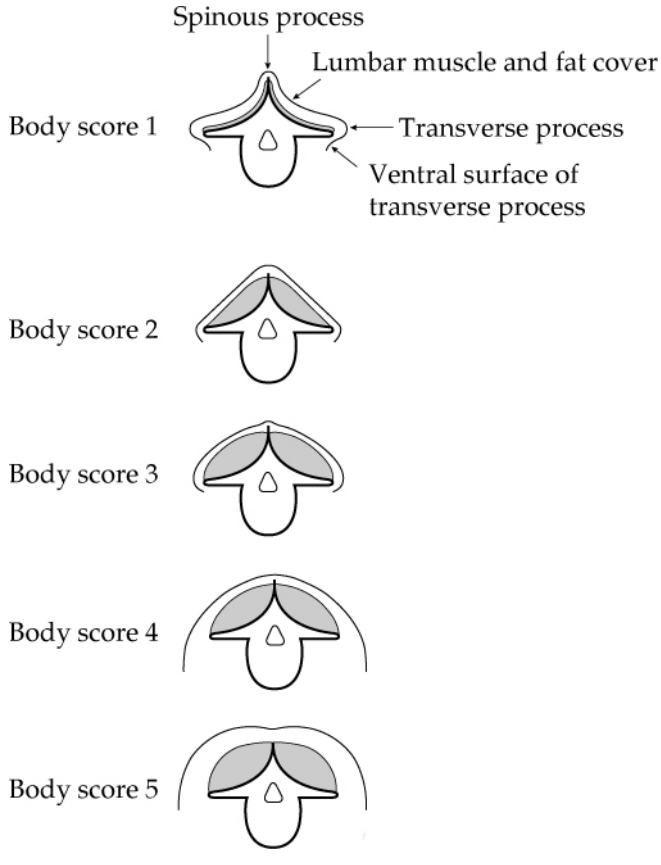


Figure A3.1 Body condition scoring.

- Different breeds, or even strains within a breed, vary in how well they absorb various minerals, for example copper.
- The amount of soil ingested with grass may influence mineral absorption from the digestive tract.
- When concentrates are fed, a manufacturer's statement or detailed analysis is required in order to make any meaningful calculations.
- Concentration of various elements in raw materials may vary widely.
- A micronutrient deficiency may be only one factor in a complex picture, for example, in ill thrift in growing lambs, when chronic gut damage may lead to malabsorption.

Because of all these variables, the diagnosis of a true deficiency of a particular mineral and therefore the appropriate corrective action is fraught with difficulty:

- Does deviation from 'normal' (reference range) blood values indicate a deficiency?

- Does a diagnosis of deficiency require deviation from 'normal' values plus a deviation from normal physiology?
- Is the only true indicator a response to treatment with the substance in question?

The clinician will have to make a decision as to the correct way forward taking into account history, complete clinical examination, laboratory tests and post-mortem examination if appropriate. But in the end, response to treatment may be the final deciding factor.

Notes on some macro- and micro-elements

Diseases caused by an imbalance of the important macro-elements calcium, phosphorus and magnesium, along with pregnancy toxaemia, often pose a diagnostic challenge in the heavily pregnant or recently lambed ewe. Pretreatment bloods are worth taking, particularly if dealing with a potential flock problem. Additionally, the mineral and energy content of the diet may require close scrutiny.

Calcium

The mature sheep has very large reserves of calcium and phosphorus in skeletal tissue. Clinical disease is, therefore, not the result of a true deficiency but of short-term difficulty with mobilisation. Hypocalcaemia is largely triggered by stress (movement, handling, adverse weather conditions, temporary deprivation of food). Unlike hypocalcaemia in the cow, the ewe is most commonly affected in the last few weeks of pregnancy, although disease can also occur in early lactation. Signs are similar to those in a cow with milk fever – unsteadiness followed by recumbency and loss of consciousness over a period of a few hours. Although treatment is usually straightforward (about 40 ml calcium borogluconate 20% intravenously slowly, plus 100 ml subcutaneously) and highly effective, prevention is more difficult. Clearly, prevention of excess stress is the primary aim, but this may not be possible, particularly if bad weather is the trigger.

Attempts to manipulate calcium content of the diet are impractical or unsuccessful. Early attempts by increasing the calcium and phosphorus content of the diet were counter-productive, as this simply reduced mobilisation. Reduction in intake in late pregnancy to enhance mobilisation is extremely difficult as the process of reducing total calcium content in the diet is complex and expensive. In addition, the usual spread of lambing over several weeks makes this method totally impractical.

Phosphorus

True primary hypophosphataemia does not occur in ewes in the UK, nor does 'pica' as reported in other countries. Reserves of phosphorus in bone are high and, although low phosphorus values can be seen in ewes with hypocalcaemia, this is usually a result rather than a cause of the problem and values return to normal after treatment for the primary problem. Having said this, it is common to give combined calcium-magnesium-phosphorus-glucose preparations which cover all eventualities without delay, since demands for a cure far outweigh the demands for a specific diagnosis.

Magnesium

Here a true primary deficiency is just as great a problem as in the cattle industry. The lack of any body reserves and the high day-on-day requirement, together with a drop in circulating levels throughout the winter period, reach a climax around the time of parturition and early lactation. This makes episodes of clinical disease very common. Often the first indication of a problem is finding a dead sheep, usually one in early lactation rearing twins. If the animal is seen alive, it shows tetanic spasms and fits, particularly when stimulated. In such cases treatment is usually straightforward, with 40–50 ml of a 20% calcium borogluconate solution also containing magnesium given by *slow* intravenous injection. This is usually effective in reducing the tetany. Magnesium sulphate (100 ml of 25% solution) should then be given subcutaneously.

Prevention methods have been well researched, with magnesium added to the diet via palatable concentrates being a highly efficient and cost effective method if survival of ewes and lambs is taken into account. It is essential that whatever method is used supplementary intake is maintained for several weeks after lambing. If concentrates are reduced in the face of rapid grass growth, feed blocks or liquid containing magnesium must continue to be offered until the risk period has passed. Dusting the pasture with magnesium oxide (calcined magnesite) at the rate of 7 g/ewe/day is also possible, but this is unpalatable and may reduce grass intake at a crucial time.

Cobalt

Cobalt forms a part of the vitamin B_{12} molecule which is synthesised in the rumen. Adequate uptake is therefore essential. Inadequate intake leads to progressive debility, particularly in growing lambs which show poor growth rate, poor wool quality and growth, inanition and anaemia ('pine'). Adults are less often considered to be affected but there may be an effect on fertility and lamb survival. Confirmation of a tentative clinical diagnosis can be by either liver or plasma assay.

Treatment with vitamin B_{12} by injection will give a very temporary response, as will oral drenching with a cobalt preparation (some anthelmintics contain added cobalt which may be sufficient if deficiency is not severe and lambs are

being wormed every month). A better and longer lasting strategy is administration of cobalt boluses or glass boluses containing copper and selenium as well as cobalt, if these trace elements are also required. If the problem is severe or soil analysis indicates intake is likely to be low, continuous access mineral blocks with a high cobalt content or application of cobalt to pasture may be necessary.

Copper

Copper provides by far the most complex problem in the micronutrient field, as both deficiency and toxicity are severe problems. There are very significant differences in the efficiency of copper utilisation between breeds and even between strains within breeds. Additionally, molybdenum in the diet is a highly efficient 'blocker' of copper uptake (sulphur and iron also have this effect). If grass is contaminated with soil which contains these elements, much of the copper in the diet can be 'locked up' in the rumen and signs of deficiency can occur. Deficiency can also be induced by improving hill pastures by liming. This alters the pH of the soil, releasing molybdenum into an absorbable form taken up by herbage, thus increasing the molybdenum content of the diet.

If copper intake exceeds demand, it is stored in the liver until a critical level is reached, precipitating a haemolytic crisis. During the build-up phase there is little or no clinical indication, and once the crisis occurs, death is rapid. Conversely, if the diet is deficient in copper, clinical disease may not appear until liver stores are very low. Measurement of blood copper levels is of limited value, since the sheep have great ability to maintain circulating copper homeostasis, therefore diagnosis is much more accurately based on concentrations in liver.

In general, the 'continental' breeds have a much better efficiency of copper absorption than native British breeds. The exception is the North Ronaldsay breed which has adapted to a diet so poor in copper that it can suffer toxicity on pastures that would produce deficiency in other breeds. The Texel can suffer toxicity at dietary levels of over 10 ppm, whereas native breeds may have no problems at levels of 15 ppm and over. Feed manufacturers do not add copper to sheep diets but the content in raw materials varies widely, so keeping copper content of diets low can be problematical. Molybdenum-containing antagonists are often added to try to reduce available copper.

Signs of copper deficiency are of two main types. The first is in the form of poor wool growth and structure, the so-called 'steely wool' syndrome, which can be seen in both growing lambs and adults. Growing lambs can also show growth retardation. The second and more dramatic form is that known as swayback, when affected lambs show hind limb weakness in varying degrees from slight (only obvious when driven) to an inability to stand at all, or in extreme cases, brain damage so severe that lambs are not viable. Reduced lamb survival without obvious swayback has also been reported. Swayback can occur in either the congenital form or the delayed form where the lamb may be several weeks old before signs appear. Neither form of swayback is treatable, as

the damage to the nervous system is permanent. As demyelination begins about the end of the fourth month of pregnancy, avoiding action is usually taken by dosing ewes with copper in mid-pregnancy.

Because of the risk of toxicity, supplementation with copper should *never* be contemplated without confirmation of a deficiency. Possible ways of supplementation include oral drenching with copper sulphate, injection of copper heptonate, administration of capsules containing copper oxide 'needles', and soluble glass boluses containing copper, cobalt and selenium. Changes in management such as winter housing previously out-wintered sheep should prompt re-evaluation of the need for copper supplementation, since removal from a source of soil in the diet increases availability of copper. Prolonged feeding of high amounts of concentrates, even with a minimal copper content, such as show animals often receive, can lead to instances of toxicity.

Selenium/vitamin E

It is impossible to consider the effects of these two factors independently since both have similar metabolic functions as antioxidants, and deficiency of either appears to result in similar pathology. Low selenium concentrations in herbage seem to be associated with granite and volcanic soils, so that areas of the UK with this type of geology are known to be at risk. Vitamin E is present in high concentrations in lush grass, but becomes reduced during dry periods. A shortage of one or other of these two chemicals will have the same result, although the actual underlying reason may differ.

The signs associated with deficiency are known as 'stiff lamb disease' (nutritional myopathy). This may occur prenatally, at birth or several weeks later. Pathological changes due to the action of excessive free radicals occur in muscle, both striated and nonstriated, and appear as white striations or plaques. Newborn lambs may be weak or unable to stand ('floppy lambs'). In lambs of a few days or weeks old, the resulting muscle damage can cause sudden death through heart failure. Alternatively, compromise of the respiratory muscles leads to difficulty breathing (which can be misdiagnosed as pneumonia), or stiffness, particularly affecting the shoulders, leading to difficulty standing or walking (this can be confused with polyarthritis). Vitamin E deficiency has also been associated with rupture of the liver and sudden death in young lambs. A deficiency of both factors has been incriminated in ill thrift and reduced growth rate in older animals in some countries, although this has not been shown conclusively in the UK.

Diagnosis may require postmortem examination as well as measurement of selenium and glutathione peroxidase (GSH-Px) in blood. Raised creatine kinase (CK) concentration is also a useful indicator, but samples need to be taken soon after the onset of clinical signs to be helpful.

There are a number of preparations that can be used for treatment and prevention. For growing animals selenium can be supplied by means of oral drenches or by using worm drenches containing added selenium. Several commercial injectable preparations containing both factors are available and

can be given to ewes in late pregnancy if problems are experienced in very young lambs, or to at-risk lambs if a problem has been diagnosed. Slow release boluses containing selenium and soluble glass boluses containing copper, cobalt and selenium can be used in older animals.

Much debate has centred on vitamin E as a metabolic stimulant capable of improving production. The results of controlled experiments have not proved conclusive; however the vitamin is now included in sheep feed at increased levels of up to 100 units/tonne.

Iodine

Inland areas away from the sea and some specific soils are known to be areas of risk for deficiency of iodine (e.g. Derbyshire). Although specific signs of deficiency are unlikely in mature ewes, this is strongly linked with infertility, stillborn lambs and increased neonatal lamb mortality. Postmortem examination of lambs will show enlarged thyroid glands if iodine deficiency is the cause. Iodine is commonly included in commercial mineral preparations, so this is the usual route for prevention. Long-acting injectable preparations are also available.

Iron

It is improbable that true iron deficiency anaemia occurs in sheep, although it has been reported in lambs reared indoors. There are, of course, a number of conditions in which anaemia features (see Chapter 17) and which require investigation as to the cause. If iron is required there are commercial preparations readily available.

Manganese

Although manganese was formerly thought to be implicated in infertility, this is now no longer held to be true. The sheep is tolerant of manganese depletion and, in any case, the mineral is usually included in commercial mineral preparations.

Zinc

Much has been written on the role of zinc in the integrity of hoof horn growth. However, it seems unlikely that a primary deficiency exists and, again, this mineral is included in most commercial mineral preparations. In New Zealand, high oral doses of zinc help to protect against facial eczema, although the mechanism is unknown.

Appendix 4
Internal parasite control

The main diseases requiring control are:

Caused by nematodes

- Parasitic gastroenteritis – caused mainly by *Teladorsagia* (*Ostertagia*) and *Trichostrongylus*.
- Haemonchosis – caused by *H. contortus*, occurs in the warmer southern parts of UK.
- Nematodirosis – caused by *N. battus*, affects lambs only.

Caused by trematodes

- Fasciolosis – caused by *F. hepatica*.

Caused by cestodes

- Infection with *Monezia* is common, but not usually considered pathogenic, although occasionally so many tapeworms are present that physical blockage of the intestine occurs. Prevention of infection is not possible since free-living orabatid mites are the intermediate host. Drugs of the 1-BZ group (see below) are effective if treatment is necessary.

Anthelmintic resistance

Drug resistance in some nematodes (mainly *Teladorsagia*) is undoubtedly present in the UK, although to nowhere near the extent experienced in some countries in the southern hemisphere. Recently, however, multiple resistance to all three anthelmintic groups has been reported in the UK. If there is any doubt as to the efficacy of a particular drug, this should be checked by means of a faecal egg count reduction test. This is performed by carrying out worm egg counts on a sample of the animals to be dosed (6–10), then sampling again 7–10 days after accurate dosing. If eggs are found, resistance is probably present and a laboratory should be consulted about carrying out further tests. Resistance to triclabendazole has been reported in liver fluke.

Avoidance of drug resistance

This is best done by:

- Keeping the number of treatments to a minimum by grazing management where possible.
- Use of strategic spring suppression of larvae.
- Changing the drug group used annually.
- Checking the accuracy of calibration of the dosing gun.
- Checking weight of the animals and avoiding underdosing by dosing to the heaviest in the group.

Control of diseases caused by endoparasites

This must be based on:

- Correct diagnosis.
- Correct treatment.
- Appropriate preventive measures.

Prevention should always be the preferred route, particularly so in the case of endoparasites, as any pathological change affecting respiratory, hepatic or enteric function is likely to be long-lasting or even permanent.

Anthelmintics

> It must be impressed on clients that the choice of anthelmintic should not be governed by freebies on offer.

At first sight there appears to be a bewildering range of anthelmintics targeting internal parasites; however, the true range is limited, since all those which target gastrointestinal and respiratory parasites fall into three chemical groups. These are:

- 1-BZ – benzimidazoles ('white drenches'). Most of these are effective against tapeworms as well as roundworms.
- 2-LM – levamisole (yellow drenches or injection). Morantel (pink drench), which is a different chemical but has a similar action, is included in this group; however it is not active against lungworms.
- 3-AV – avermectins and milbemycins (clear drenches or injectables). Some of these have prolonged action against some roundworms but not against *Nematodirus*.

In the case of liver fluke, there are also several different chemicals available, but the choice is limited by the age range of fluke against which a particular product is effective:

- Albendazole is effective against adult fluke only.
- Closantel is effective against immature fluke from 4–5 weeks and adults.
- Netobimin is effective against adult fluke.
- Nitroxynil is effective against immature and mature fluke.
- Triclabendazole is effective against immature forms from 2 days old.

Gastrointestinal parasites (nematodes)

Treatment

This is a fairly straightforward matter, as all groups of chemicals are effective *provided resistant worms are not involved.* There are, however, several important points to note:

- Correct diagnosis – many clients use anthelmintics without advice, assuming any scouring lamb must have worms. This is clearly not the case. At some point in lamb growth scouring could equally be due to coccidiosis, low dry matter intake or breed tendency to produce fluid faeces, e.g. Suffolks.
- Correct use of equipment – drenching guns are frequently just rinsed out then stored for long periods. It is essential that the calibration is checked before use, i.e. that 5 ml on the barrel actually delivers 5 ml to the sheep. In addition, leaking valves allow air bubbles into the line, preventing the delivery of a full dose.
- Correct technique – is the dose being delivered over the tongue or just in the general direction of the mouth, allowing some to be spat out? (But beware of dosing gun injury to the back of the throat by use of undue force). A period of food withdrawal (24 h) before drenching will accentuate the effect of the drug. Similarly, housing for 24 h after dosing will prevent viable eggs in the lower gut from being deposited on new grazing.
- Extent of damage already present in gastrointestinal tract. Permanent damage before treatment takes place means that animals may not thrive even after an effective anthelmintic has been administered.

Control and prevention of PGE

This is a complex subject and should be approached by bearing in mind key features in the life cycle of the parasite. These include the following features:

- The life cycle has developed to allow optimal take-up by the host.
- The parasite can only persist year-on-year via surviving infective larvae on pasture or via stages in the gut of older animals.
- Infestations of naïve lambs produce large numbers of eggs, contributing to massive pasture contamination.
- Lambs develop little resistance before 6–8 months.
- Adult ewes have a good immunity except during the approach to lambing and in the first 6–8 weeks of lactation.
- Young, old and diseased ewes cannot be assumed to have good immunity.

- Some rams do not appear to develop immunity, but there may be the possibility in the future of selecting those which do have naturally low worm egg counts.
- For each family group from lambing to weaning, the proportion of total intake of grass consumed by the ewe is 90% in the case of single lambs and 80% in the case of twins. Hence the same proportion of infective larvae on pasture will also be consumed by the ewe.

Much has been written about control of internal parasites by nonchemical methods – clean pasture, mixed grazing and rotational grazing. Much of this is no longer relevant to modern intensive agricultural production methods, although it is attempted in organic systems. For most enterprises, control is now based on the key points of the life cycle, combined with use of safe pasture.

Clean grazing i.e. with no egg or larval contamination, is rarely possible except in arable systems with long rotations between grazing seasons. Larvae can survive for up to two years in pasture or even in soil, so integration of sheep into an arable or dairy unit is no guarantee of being able to operate such a system. Similarly, mixed or rotational grazing is unlikely to be considered purely for parasite control.

Safe grazing i.e. pasture considered to be low risk as a source of significant numbers of infective larvae, is achievable on many farms. High hill grazing with very low stocking rates, pasture from which sheep have been withheld since summer of the previous season, and aftermath following the taking of a hay or silage crop from a field not grazed by sheep that spring, are all examples.

Spring suppression of worms

This is a programme which offers true control with minimal risk of gut damage and combines strategic dosing with use of safe pasture. It utilises the fact that 80–90% of grass consumed during the lambing to weaning period is by the ewe, who thus consumes the same proportion of infective larvae on the grass. At the same time, the periparturient rise in output of worm eggs from the ewes on to the pasture is prevented by dosing with anthelmintic. The strategy is as follows:

- Dose ewes after lambing shortly before turn-out with a drug that has a persistent effect (moxidectin or doramectin). This removes virtually all adult roundworms plus overwintered hypobiotic larvae, and because of the persistent action, kills any larvae that are eaten by the ewe from the population which has survived over winter on the pasture. After about 6 weeks, the natural immunity of the ewes quickly re-establishes so further infective larvae ingested do not develop in any significant numbers. As the ewes are therefore prevented from producing significant numbers of worm eggs in the suckling period, the lamb crop should then not be challenged by significant numbers of larvae provided they are weaned by mid-summer.
- At weaning, dose lambs also with a persistent drug, hold indoors or on concrete overnight, then turn on to 'safe' pasture. If the pasture truly is 'safe', no further dosing should be required; indeed if carefully followed,

the programme has proved so effective that after several years the weaning dose has not been required.

This policy is true prevention, removing the possibility of significant gut damage, at the same time minimising the number of doses of anthelmintic required.

There are other variations on the theme, but none so simple:

- If ewes are dosed at housing they do not need to be dosed at turn-out, but obviously there will be no residual anthelmintic activity present. Unless there are pressing reasons to worm at housing, delay the dose to turn-out as above.
- The same effect can be achieved with 1-BZ or 2-LM drugs, but as none of these has residual activity the ewes require dosing at 3, 6 and possibly 9 weeks after lambing, and lambs at 6, 9 and 12 weeks of age, thus negating the advantages of minimal handling described above. However, if drug groups are to be rotated as described above, this more complicated method will have to be used in some years. Note also that ivermectin has no residual action in sheep so would need to be used in the same way.
- It must be emphasised that circumstances on each farm are different and worm control programmes often need to be tailor-made. Careful checking of the grazing history of pasture is required and worm egg counts should be done at intervals to check that mistakes have not been made.

Prevention of nematodirosis

Nematodirus battus has a different life cycle from other gut nematodes, therefore requires a different preventive strategy. The main features of the epidemiology of the parasite are:

- Lambs over 6 months of age and adult sheep are highly resistant to the parasite.
- Infection is passed from one lamb crop to the next.
- Infective larvae, protected within the egg, have great resistance to adverse conditions such as freezing and desiccation and can survive for up to 2 years on pasture.
- Hatching of larvae requires a period of exposure to cold followed by a temperature rise to above 10°C.
- Disease only occurs if mass hatching of larvae coincides with susceptible lambs eating sufficient grass.
- Infestation and disease can be explosive, with debility and deaths over a very short timescale if diagnosis is not rapid and accurate.
- If mass hatching occurs very early before lambs are grazing, or very late when lambs are beginning to acquire resistance to infection, disease will not occur.
- Calves can also be infected, thus contributing to pasture contamination and carry-over of infection even if lambs are not grazing the pasture every year.

Prevention is therefore best based on grazing control, with lambs not using the same paddocks for early grazing in successive seasons. If this is not possible,

extreme vigilance is required, with drenching required at the earliest signs of scouring. If there is a recurring problem, strategic dosing with anthelmintic at 4 and 7 weeks may be necessary. It must be noted that the drugs moxidectin and doramectin have no residual action against *Nematodirus*.

> Control of nematodes will become increasingly difficult if anthelmintic resistance becomes widespread. It is most important that planned control programmes combining safe grazing with minimal drug use are implemented in as many flocks as possible. Regular monitoring by faecal egg counts will allow strategic dosing and eliminate unnecessary dosing.

Liver fluke

In the case of this parasite, prevention of disease is extremely important, since:

- Chronic infestation produces severe and cumulative liver damage.
- There is no protective immune response, therefore adult sheep continue to be susceptible and, if untreated, constitute a continuous source of infection.
- Liver damage can be so severe in acute infections that either the disease or treatment can be fatal, or leave irreparable damage.
- The damage to the liver can act as the trigger for black disease.

Most sheep keepers in the wetter western half of the UK will be well aware of the high risk and the severe effects of infestation. They will also be aware of the main risk period for their own particular area. The pattern of disease is largely due to the effect of temperature and moisture on the snail *Lymnaea truncatula* which is the intermediate host. This requires a temperature range of about 15–25°C in order to reproduce in large numbers; in dry or cold conditions it withdraws into the deeper layers of mud, therefore at such times the life cycle of the developing fluke cannot be completed.

Acute fluke

This is caused by large numbers of migrating immature flukes and can occur from autumn through to mid-winter in the mild south-west of England, whereas the risk period is much shorter in the colder north of Scotland. The usual picture is sudden death of several animals, with others showing lethargy and pale mucous membranes. Remember that because the disease occurs in the prepatent period, fluke eggs will not be present in faeces samples. Diagnosis will depend on PME of dead animals and presence of anaemia and raised liver enzymes in blood samples from survivors. If it is possible to move sheep to uninfected pasture, a single dose of triclabendazole before moving will suffice in controlling the disease. Farmers in risk areas where uninfected pasture is not available will aim to control acute

infections by drenching at 3–4 week intervals during the summer and autumn. If a drug with good activity against immature fluke is used (preferably triclabendazole), this will give a good degree of control but will not totally avoid liver damage and is not an effective method for long-term reduction of infestation of the flock.

Chronic fluke

In the case of chronic disease, this occurs usually through winter into early spring or, less commonly, in summer following a wet spring. The typical signs are loss of weight, anaemia and development of oedematous third eyelid and sometimes bottle jaw. These signs, together with identification of fluke eggs in faeces and raised liver enzymes, are diagnostic. Control depends on strategic dosing with flukicides, the timing and frequency depending on likelihood of disease from disease forecasts based on weather. At the simplest, treatments in April, October and January should give control, but the exact schedule will depend on individual farm circumstances.

Dependence on flukicides can be reduced by improvement to pasture by drainage in order to reduce snail habitats, or fencing off particularly dangerous areas if at all possible, or removal of sheep from dangerous grazing between September and May.

Long-term control of fluke

There is a policy which can go some way towards achieving long-term reduction in weight of infection on land; it resembles the policy of spring suppression of nematodes. This needs to take account of the fact that the parasite can survive the winter in three forms:

- As mature flukes in the liver (these can survive as long as the host, since no immunity develops in sheep).
- As infested snails.
- As encysted metacercariae on the herbage

If triclabendazole is used, which kills immature and mature stages and therefore suppresses egg output for about 12 weeks, the effect can be cumulative, resulting in markedly decreased numbers of infected snails after several seasons. Drenching the ewe after lambing or at turn-out will remove fluke from the liver, ensuring that no new snail infestation takes place. Thereafter, infested snails and metacercariae remain, so for several seasons it will be necessary to drench a further three times at intervals of about 12 weeks to prevent any immature forms completing their life cycle. Total clearance of fluke can eventually be achieved provided no new infected animals are introduced. No new infestation means that snail numbers become of no significance and become merely added protein and minerals in the diet of the ewe. Other drugs are less expensive but do not remove immature flukes below 4–6 weeks of age, therefore will not be effective in such a schedule.

If a flock is effectively cleared of the major endoparasites by the above methods, there remains the problem of possible future re-infestation. It is therefore essential to monitor the parasite status of all replacements, or to confine to yards and dose all replacements before allowing on to pasture.

Appendix 5
Health plans and vaccination programmes

National plans

There are national or regional plans covering maedi-visna and enzootic abortion, run as the Sheep and Goat Health Scheme by the Scottish Agricultural Colleges (SAC). In addition the National Scrapie Plan, designed to increase the proportion of genetically resistant animals in the national flock, was introduced in 2001.

Individual flock plans

A general plan is given in Table A5.1 of the essential features which must be considered by the clinician when developing health plans, although it must be recognised that each flock is different. There are likely to be variations, subtle or more major, depending on individual flock circumstances, economic factors and the degree of risk a flock keeper wishes to take. The type of flock in which most impact can be made by planned veterinary input is one which has a reasonable level of performance, but which is capable of significant improvement and has an interested owner/shepherd. The best managed and highest performing flocks often provide a challenge in terms of identifying areas for significant improvement, but the keen clinician can benefit greatly from such involvement and indeed learn a great deal through such a link. The poorest flocks which, at first sight, should easily be capable of significant improvement are often unrewarding because of lack of interest or failing finances. These latter flocks are those in which the most obvious welfare problems are likely to exist. Current suggestions of licensing farmers might, hopefully, lead to pressure on such farmers to improve or get out of the business, as these are the minority who give farming a bad name.

Table A5.1 Example of health plan for lowland flock housed for lambing (optional vaccinations and other treatments in brackets).

Stage of production	Group	Health and disease monitoring	Vaccinations/treatments	Nutrition
Weaning to mating	Ewes	Cull for teeth, feet, udder, fertility		Condition score and group according to nutritional requirements to achieve mating target 3–4
		Establish abortion status	Give appropriate vaccines (EAE, *Toxoplasma*)	
		Metabolic profile for micronutrients	Correct where indicated	
		Examine feet	Trim and footbath (footrot vaccine)	
		Check fluke status	Drench if necessary	
	Weaned lambs		Worm and hold overnight Clostridial vaccination (+ *Pasteurella*) if not done before – two doses 4 weeks apart	Transfer to safe grazing
	Rams	Check fertility, teeth, feet (6–8 weeks before mating)	Booster clostridial vaccine Worm Trim feet and footbath	Adjust diet for condition score 4 by mating time
Mating to implantation	Ewes	Monitor mating activity with raddle marks	Minimal interference to reduce embryonic loss	Maintain body condition
	Rams	Check mating activity	Treat any lame	Supplement feed if necessary
Mid-pregnancy	Ewes	Scan for fetal numbers		Adjust groups for body score and fetal numbers
		Check feet	Treat where necessary, footbath (footrot vaccine) (Orf vaccine) (Copper supplement)	
	Rams	Check feet	Treat where necessary, footbath	Adjust condition if excessive weight loss, maintain in adequate condition
6–8 weeks pre-lambing	Ewes	Full metabolic profile		Condition score, increase energy if deficient (BHB>1.2)
4–6 weeks pre-lambing	Ewes		Clostridial (+ *Pasteurella*) booster (Erysipelas, *E. coli* vaccine boosters)	Continue to monitor body condition

			Worm at housing unless using persistent wormer Check fluke status, treat if necessary	
Lambing	Ewes	Monitor for mastitis, metritis, hypocalcaemia, check blood mineral levels if necessary	Treat as necessary	Increase dietary allowance gradually to 3× maintenance
		Check feet before turn-out Check bonding with lambs	Treat as necessary	
	Lambs	Check colostral intake	(Tail and castrate)	
Turn-out	Ewes		If using persistent wormer, drench at turn-out	Maintain nutritional intake at 3×M
		Monitor for mastitis, metritis Monitor Ca and Mg status	Treat as necessary	
	Lambs	Monitor for hypothermia, enteritis, septicaemia, watery mouth	Treat as necessary	
Lactation	Ewes	Monitor for mastitis, hypomagnesaemia	Supplement with Mg if necessary	
	Lambs	Monitor growth rate Check for coccidiosis	Strategic treatment if necessary	
Lamb growth period – milk to grass nutrition transition	Ewes	Faeces samples to confirm spring suppression		Reduce feed inputs
	Lambs	Monitor faecal egg counts	Worm at 6–7 weeks with persistent wormer (worm every 3 weeks until June if not persistent) (Clostridial vaccination if not sold by 16 weeks – two doses at 4 week interval)	Check dry matter intake
At weaning	Ewes			Put on bare pasture to discourage milk production
	Weaned lambs		Worm and hold overnight	Transfer to safe grazing

Appendix 6
Poisons

Poisoning episodes in sheep are less common than in most other domesticated species, for a number of reasons:

- The sheep, being a pure herbivore, is less at risk than pigs and poultry.
- Feed additives are used much less commonly than for intensively kept species.
- Intake of concentrates is less than in cattle.
- Sheep graze much more selectively than cattle, therefore are at less risk of plant poisoning.
- Sheep are by behaviour less inquisitive than cattle and goats.
- Because of husbandry methods sheep have less access to toxic chemicals.

The greatest risks come from access to mining sites and disused industrial areas on open grazing, and from overdosing or mistaken concentrations of therapeutic products such as selenium, footbath chemicals, or feedstuffs containing copper. Garden plants also pose a risk, either when discarded or when sheep gain accidental access, or when grazing is exhausted or made inaccessible because of heavy snowfall.

Diagnosis will usually depend on a combination of:

- History taking, since affected animals may well not be found near the source of the toxin.
- Inspection of areas where animals have had recent access. Expert assistance in identification may be needed if unusual plants are involved.
- PME (which may not be helpful since the toxic cause may not be apparent in the gut because of rapid absorption or digestion).
- Laboratory tests (which may be expensive).

Plant poisons

Acorns

The risk of poisoning is high after autumn storms bring down large numbers of acorns which, particularly when green, are attractive to sheep. It is not clear why some sheep can apparently eat many with impunity while other animals will become ill. The acorns contain highly astringent tannins which cause gastroenteritis and kidney failure. Affected sheep become anorectic, suffer very

rapid weight loss and become recumbent. Bloody diarrhoea is followed by constipation. Kidney failure leads to uraemia and death. PME shows enteritis, but recognisable acorns are often not seen as they will have been broken down before death. Urea concentration in serum is raised by a factor of at least 10. In animals less severely affected, fluid therapy and appetite stimulants may be effective. Prevention is by removing access until the acorns have rotted or, if this is not possible, spreading the area with slurry will reduce palatability.

Bog asphodel

This is a cause of photosensitisation. See below.

Bracken

Bracken is perhaps the most successful plant on earth. It is rapidly colonising huge areas of upland Britain since any form of control such as repeated cutting and spraying has become uneconomic. Although all parts of the plant, both fresh and dried, are poisonous, sheep do appear to tolerate limited access to the very young fronds. The plant has also been used in its dead form as bedding in hill areas from time immemorial, though more for cattle than for sheep. The toxic factors are complex and produce different clinical syndromes in different species. A thiaminase produces thiamine deficiency in horses and pigs but not in ruminants. Carcinogenic factors are implicated in bladder cancer in cattle, and increased incidence of bowel and bladder tumours may be present in sheep that have grazed bracken for prolonged periods, although these are rarely seen, as few exposed animals are subjected to PME.

Bracken spores may pose a risk to humans. Acute bracken poisoning (haemorrhagic fever) is common in cattle but less so in sheep. The only well-documented problem in sheep is a progressive, irreversible retinal atrophy (bright blindness). This results from prolonged exposure, affecting only sheep over two years of age. Diagnosis is based on presence of blindness with dilatation of the pupil and lack of pupillary response to light. Examination of the eye with an ophthalmoscope will show retinal blood vessels to be narrowed and reduced in number. Affected animals can survive in familiar surroundings provided there is sufficient food, but will not recover their sight. Prevention is only by removal from the bracken source, but this may not be possible in hill areas.

Brassicas

Several types of brassicas are fed to sheep, including rape, stubble turnips, kale, mangolds and other beets. Fattening lambs are also commonly fed on waste left after vegetable crops have been harvested. These form highly palatable feeds during winter and early spring but have the potential to cause various toxicity problems, particularly if introduced too quickly or forming too high a proportion of the diet. The most commonly encountered problems arise from the

disulphide, nitrate and oxalate content, which varies from crop to crop and, in the case of roots, with length of storage. Mangolds and beets should be stored for several months before feeding. Perhaps the greatest risk comes with the use of stubble turnips in intensive systems, reinforcing the need already apparent for welfare reasons for a run-back on to grass.

Clinical problems arising from feeding brassicas include:

- Haemolytic anaemia (from disulfides) – this is usually low grade haemolysis, giving rise to suboptimal growth rates but not usually fatal. Occasionally disease may be more severe with haemolysis, haematuria and jaundice. Blood samples will help in distinguishing between haemolytic anaemia and nitrite poisoning (below). In this case the blood is dark red and watery. Animals should be removed from the crop on to alternative feed sources.
- Nitrite poisoning (from nitrates accumulated in roots in particular) – nitrates are converted into nitrites in the rumen, then absorbed into the blood where they combine with haemoglobin to form brown-coloured methaemoglobin. Affected animals become weak, recumbent and die. Blood samples will show the characteristic brown, cloudy appearance. Treatment can be attempted with 10 mg/kg methylene blue intravenously. Animals should be removed to alternative feed sources.
- Hypocalcaemia (from oxalates in leaves and roots) – if large amounts of oxalates are consumed, insoluble calcium oxalate forms in the blood giving rise to typical signs of hypocalcaemia. Longer-term ingestion leads to chronic kidney damage.
- Bloat – severe episodes have been reported associated with feeding kale. These seem to be related to specific strains of the crop grown on specific fields.
- Photosensitisation – this is seen occasionally – see below.
- Oestrogenic effects – some crops have been reported as being oestrogenic; certainly ewes before tupping and during pregnancy should be limited to below 30% of the diet in the form of brassicas.

Hemlock water dropwort (Dead men's fingers)

The roots of this plant are toxic, and poisoning is almost always the result of drainage projects which expose them, leading sheep to eat them if given access. The toxic factor, oenanthetoxin, causes convulsions and death. Prevention is based on great care when drainage projects are being undertaken, with removal and safe disposal of the roots.

Ragwort

Fatalities due to ingestion of ragwort are less common than in cattle, which are 30–40 times more sensitive, but do occur occasionally in sheep. The plant contains pyrrolizidone alkaloids which, when metabolised, become highly

toxic to the liver. Affected animals lose weight, become emaciated and die of liver failure. PME shows obvious and typical cirrhosis of the liver. There is no treatment. Prevention is by avoidance of pastures with a high density of the weed, eradication of the weed (preferably by pulling and burning) and vigilance to avoid inclusion in hay.

Rhododendron

Catastrophic episodes of poisoning can occur from ingestion of the leaves of this shrub which contain diterpenoids. Incidents which can affect whole flocks often occur due to accidental access to the plant at times of reduced feed availability such as heavy snowfall. Affected animals attempt to vomit (this is the main cause of vomiting in sheep), become bloated and drool saliva, show severe abdominal pain, become recumbent and die. Treatment is symptomatic only and less severely affected animals often recover. Prevention depends on vigilance at times of risk, together with removal of overhanging branches and care with disposal of garden waste.

St John's Wort

This plant causes photosensitivity - see below.

Yew

Yew is highly poisonous to stock as it contains alkaloids, including taxine which is directly toxic to the heart. The leaves are more toxic than the berries and death is very rapid, animals being found dead still in the vicinity of the tree, with foliage found in the mouth or upper gut. There is no treatment since death is inevitable and rapid. Animals should be removed from the source immediately.

Plants causing photosensitisation

(See also Chapter 20). This is a common result of the ingestion of a number of plants. It takes two forms - primary, in which case the photodynamic agent, having been ingested in the plant, is carried direct to the skin in the blood; or secondary, where liver damage leads to incomplete metabolism of chlorophyll, allowing the photodynamic metabolite phylloerythrin into the general circulation. The end result is the same, with areas not protected by wool affected, particularly the head, conjunctivae and ears, which are grossly swollen and often exude serum. If the animal recovers, the ear tips may be lost.

Perhaps the most commonly implicated plant is bog asphodel, which contains steroidal saponins, and is found on high ground throughout northern Europe. Each area often has its own name for the syndrome, often very descriptive (yellowses, plochter, alveld, hard lug). There is also the possibility of the involvement of mycotoxins in this syndrome. Other plants implicated are

St John's Wort, buckwheat and rape. Treatment is with steroids to reduce swelling, and housing to protect from sunlight. An increasing risk is giant hogweed which is spreading rapidly and is difficult to control. In this case the response is the result of direct skin contact with the plant rather than ingestion.

Miscellaneous

A number of plants all containing toxic (often cyanogenic) glycosides have been identified as poisonous to sheep and are recorded as being responsible for random poisoning incidents. These include cherry laurel, privet, elder, hydrangea, some indigenous clover species and bird's foot trefoil, although some of these are normally unpalatable. Signs usually include cyanosis, breathing difficulty, muscular spasms and death. As death is usually rapid, plant fragments are often found in the gut. Sodium thiosulphate is recorded as a treatment. Dog's mercury has also been recorded as a cause of deaths.

Apart from mature plants, fungi have been recorded as a source of poisoning in sheep. *Fusarium* spp. have been recorded as oestrogenic. Ryegrass staggers results from fungal infestation of ryegrass. Aflatoxins produced by moulds in stored feeds are a rare problem in sheep. Blue-green algal blooms are regarded as dangerous if water is in short supply.

Chemical poisons

Chemical poisons can be divided into two groups – poisoning from accidental access to or overdose of therapeutic substances, and poisoning by accidental access to products which play no part in metabolism or therapy.

Chemicals used in feeds or as therapeutic agents

Copper

As outlined earlier (see Chapters 15 and 18), the metabolism of copper is very complex in the sheep. Although it is an essential trace element, the efficiency of absorption varies widely between breeds, and the margin between deficiency and excess is narrow. The copper content of feedstuffs varies (without any supplementation) as do levels in various geological structures and, therefore, in soil and herbage. Added to this are complex interactions between copper and other minerals, especially molybdenum, sulphur and iron, altering availability to the animal. There are therefore risks in precipitating poisoning by administering copper products, even in areas of known deficiency. Acute poisoning from access to footbaths containing copper sulphate is now less likely as zinc sulphate has become the treatment of choice.

Chronic poisoning can be a side effect of high concentrate intakes in terminal sire units, simply as a result of the inability of feed manufacturers to control natural copper levels in raw materials. Native breeds can tolerate intakes of

15 ppm but for continental breeds, perhaps Suffolks, and in high concentrate intake units, concentration should be limited to 10 ppm in feed. Copper is stored in the liver and poisoning becomes apparent when a critical level is reached, precipitating a haemolytic crisis and kidney damage. Death can be sudden or after a short period of debility. Diagnosis of chronic poisoning is usually straightforward as the carcass is jaundiced and copper content of the liver is high. Diagnosis of acute poisoning is less obvious, since jaundice may not occur and copper content of liver may not be particularly high. When poisoning occurs, other sheep in the group should be examined carefully and if they are at risk 'copper blockers' can be administered. Sick animals should be given ammonium tetrathiomolybdate by injection at a rate of 3.4 mg/kg on three occasions on alternate days. Other at-risk animals may be dosed by mouth or in feed with 100 mg ammonium molybdate daily and the diet assessed as a matter of urgency.

Prevention can also be complex. Accurate concentrations in feed are difficult to assess and copper blockers are now routinely included in concentrate feeds for terminal sire breeds. Mineral supplements intended for species other than sheep should be excluded as these may contain copper. In addition cattle and pig foods are completely unsuitable for sheep. Copper should *never* be administered until a deficiency has been identified.

Levamisole

Accidental overdosing of levamisole has been reported, the effect on the sheep being a reflection of the effect on the parasite – neuromuscular paralysis. Death is rapid from respiratory failure following brief signs including excess salivation, tremors, staggering, defaecation, urination and collapse. There is no record of effective treatment, and prevention is by attention to detail in dosing.

Nitrates

As well as plants being a source of nitrates, nitrogenous fertilisers can be toxic if ingested in large quantities. Signs are as described under poisoning by brassicas, above.

Organophosphates

Although the risks to man in handling organophosphates have been widely publicised and the side effects of contact with the concentrated product are well known, there is little evidence of problems from the immersion of large numbers of sheep annually in dips containing these products. This is in spite of the fact that if dipping is carried out correctly total saturation of the skin should occur and also many sheep do undoubtedly ingest significant quantities of the diluted chemical. In any case of accidental poisoning the effects are those of an anticholinesterase – excessive salivation, tremors, incoordination and collapse.

Confirmation of diagnosis is difficult but response to administration of atropine is highly suggestive.

Selenium

Although deficiencies of selenium are widely identified, there is some debate as to the true clinical effect and at what level of intake these become apparent. The mineral is commonly included in both mineral supplements and worm remedies, so if there is in fact no deficiency, toxicity can occur. It can occur if such remedies are inadequately mixed and can also follow administration of selenium by subcutaneous injection. Death can occur within hours of administration. Confirmation is best carried out by assay of selenium in liver or kidney. Prevention should be based on accurate diagnosis of, and the severity of, deficiency. If no deficiency exists, products containing selenium should not be administered.

Other toxins

Other chemicals used in farming which have been responsible for poisoning include ionophores, superphosphates, urea and zinc.

Nontherapeutic agents

Lead

As outlined at the beginning of this appendix, sheep are less inquisitive than cattle, hence acute lead poisoning in sheep is not common, though when it occurs the sources (industrial spillage or dumped rubbish such as old paint and car batteries) are the same as for cattle. Signs are also the same – neurological with staggering, fits, blindness, collapse, intermittent convulsions and death. If a single animal is involved and no obvious source of lead is seen, there will be an immediate confusion with magnesium deficiency. PME shows gastroenteritis, liver and kidney lesions and a characteristic greyish appearance of muscles. A chronic form has also been identified where animals have access to old lead mine workings. In this case signs are of stiffness and general locomotor difficulties, with osteoporosis and occasional fractures and deformities of bones. Diagnosis should include lead assay of kidney samples (values above 50 μmol/kg are indicative of poisoning). Treatment is by slow intravenous injection of sodium calcium edetate at rates of 75 mg/kg daily, when the response can be quite rapid.

Phenolic compounds

There are a number of sources of phenols to which sheep may become exposed. These include phenolic navel dressings, farm disinfectants, wood preservatives and anti-fox predation products. This group is poisonous through skin

absorption as well as by ingestion. Death is often rapid – failure to correctly mix a fox-oil product led to a 70% death rate in a group of lambs immediately after application. Death may be preceded by convulsions, diarrhoea and depression. If the product has been ingested, PME shows inflammatory changes in the upper gut, but in all cases there is usually a strong smell of phenol from the tissues. Treatment of survivors is largely symptomatic, including demulcents and fluids. Prevention is as for most poisons – care and prevention of access. If wood surfaces of sheep housing are to be treated, this should be done well before any use is to be made of the building.

Other toxic chemicals

Other chemicals which have been the cause of sheep deaths include arsenic, fluorine, mercury and warfarin.

Appendix 7
Zoonoses

Attention has been drawn throughout this text to diseases of sheep which pose threats to man, particularly people working in close contact with them such as shepherds and lambing assistants. Less obviously, some diseases may be spread on contaminated clothing and others may exist in areas frequented by sheep, although not directly transmitted by them. Finally, nonfarming visitors, especially children, may be especially at risk. In particular, markets and farm parks are high risk locations. It is no longer safe to allow children to bottle-feed pretty fluffy lambs without thinking of the risks and providing suitable washing facilities. The clinician should always be aware of any potential zoonotic risks and advise clients accordingly.

The following list includes diseases that may pose risks in the UK.

Campylobacter

This causes abortion in sheep and gastroenteritis in people. Care should be taken in handling sheep and lambs in infected flocks.

Caseous lymphadenitis

This causes multiple abscesses in sheep and has occasionally been reported as infecting man. It is sensible to wear rubber gloves when handling infected animals.

Cryptosporidia

This causes scouring in young lambs and is an important cause of gastroenteritis in children. They usually become infected by handling or feeding young lambs, particularly if hands are not washed afterwards. People can also become infected through the water supply if this is contaminated with sheep faeces and is not adequately treated before drinking.

E. coli O157

The source of contamination by this particular strain of bacteria is not always clear but has been linked to sheep at least once. Young and old people are particularly susceptible to the toxin, which can cause serious kidney damage.

Enzootic abortion (chlamydial abortion caused by Chlamydophila abortus*)*

This is an important zoonosis and particularly dangerous to pregnant women, resulting in loss of the baby and occasionally death of the mother. Pregnant women should therefore definitely avoid working with lambing sheep. It can be transmitted on clothing so protective clothing should not be brought into the house without prior disinfection. Premature and sick lambs should also not be brought into the house if a pregnant woman is present. In other people, particularly immunosuppressed or elderly, symptoms are flu-like. Great care should also be taken in handling the live vaccine.

Erysipelas

This can occasionally cause illness in people, so care should be taken to wash hands well after handling infected sheep.

Hydatid disease

Although sheep can be infected with hydatid cysts, they do not form the source of infection for people. The source is the tapeworm *Echinococcus granulosus*, for which the definitive host is the dog. These should be wormed regularly with praziquantel, which is also effective against other tapeworms such as *Taenia multiceps* and *Taenia hydatigena*, both of which infect sheep as the intermediate host. In fact most hydatid disease in man seen in the UK is contracted overseas.

Johne's disease

Controversy still exists over whether Johne's disease is linked with Crohn's disease in man. The two diseases are similar in their pathology but mycobacteria have only rarely been found in the tissues of human patients.

Leptospirosis

Leptospirosis is rare in sheep but is potentially zoonotic via the products of abortion, urine and milk.

Listeriosis

Infection with *Listeria monocytogenes* causes meningitis or encephalitis in man, with pregnant women suffering abortion or stillbirth. The most common routes of infection are through cheese made of unpasteurised milk, or contaminated uncooked vegetable matter such as coleslaw.

Liver fluke

This may be contracted by eating wild watercress carrying encysted metacercariae in the same way as sheep become infected.

Louping ill

This is rare in man, but as approximately one sheep tick in 1000 is infected with virus, care should be taken to avoid being bitten by ticks when walking through tick habitats.

Lyme disease

This disease, caused by *Borrelia burgdorferi*, is also transmitted by the sheep tick and is more common in man than is louping ill.

Orf

This is an important and common disease caught from infected sheep. It is seen most commonly on the hands but can infect any area of broken skin. The most common presentation is a painful, swollen, red area with a blood-filled blister at the centre. Occasionally spread of lesions with severe systemic illness occurs. Doctors are often unaware of the condition. As well as taking great care in handling infected sheep and lambs, the live vaccine also requires care in handling. Rubber gloves should be worn and hands thoroughly washed afterwards (but not scrubbed as this could inoculate the virus).

Q fever

This causes abortion in sheep and can cause a nasty flu-like illness in man, particularly in immunosuppressed people.

Ringworm

This is less common in sheep than in cattle but is equally capable of infecting people, causing circular red, flaky skin lesions.

Salmonellosis

Several species of *Salmonellae* infect sheep, some causing severe illness, others causing abortion without illness. Those most likely to cause human disease are *S. typhimurium*, *S. dublin* and *S. montevideo*. Suitable advice on hygiene precautions should always be given if these are diagnosed in a flock.

Toxoplasmosis

Toxoplasmosis is an important disease in humans, particularly in pregnant women, the elderly and immunosuppressed. It is also a common cause of abortion in sheep. However, aborting sheep are not particularly dangerous as the most infective stages of the parasite are not found in the placenta of the sheep. Like sheep, the most common source of infection for man is oocysts

produced in cat faeces. Another source is undercooked sheep meat. Normal hygienic precautions should, however, be taken if toxoplasmosis has been diagnosed in a flock as it is always possible for more than one abortion agent to be present. Great care should also be taken in handling the live vaccine.

Appendix 8
Anaesthesia and common surgical procedures

Anaesthesia and analgesia are problematic in sheep because of the lack of pharmaceuticals licensed for use in the species and the diminishing list of products licensed for use in food species. The use of most products will therefore be based on the application of the 'cascade' system, and standard withdrawal periods will need to be applied. Even though licensed products may not be available, there are several anaesthetic and analgesic agents that have been widely used and established to be safe, subject to certain precautions being taken. The methods described below are some that have been reliably used under practice conditions; however, for detailed information a text on anaesthesia should be consulted.

Sedation

The most commonly used sedative is probably 2% xylazine, which is licensed for use in cattle. This has sedative and analgesic properties and its effects are dose-dependent, although more variable than in cattle.

- The dose ranges generally used are 0.05–0.2 mg/kg (0.12–0.50 ml/50 kg) by intramuscular injection.
- It should not be given to heavily pregnant animals unless they are actually lambing.
- The animal should be left undisturbed for at least 15 minutes for the drug to take full effect.
- If the animal is disturbed soon after administration, less sedation than anticipated may be achieved.
- With an animal that has been starved longer than usual or is debilitated, the sedative effect can be more than anticipated so care needs to be taken in dealing with such cases.
- If sedation is worryingly deep, the effect can be rapidly reversed by administering atipamezole (Antisedan). The same volume of atipamezole as was originally given of xylazine should be administered by intramuscular injection.

Analgesia

Sheep are stoical creatures and often show few overt signs of pain (tooth grinding is a common sign). However, this should not be taken to mean that they do not feel pain, so administration of analgesics should be considered in conditions where pain is likely to feature. The nonsteroidal anti-inflammatory drug (NSAID) flunixin meglumine is probably most widely used in sheep. The dose rate is 1.1–2.2 mg/kg (up to 2 ml/45 kg) given intravenously or intramuscularly. Ketoprofen, meloxicam and carprophen have also been used in sheep. All these drugs can precipitate abomasal ulceration, so should not be used for more than 3 days.

Acetylsalicylic acid (aspirin) is a very cheap and effective analgesic for sheep. Two 300 mg soluble aspirin tablets dissolved in a little water and given orally are a simple way of providing pain relief, but this old-fashioned drug is not licensed for food-producing animals.

Local and regional anaesthesia

Many of the surgical procedures carried out in sheep can be done under local or regional anaesthesia, with or without sedation, depending on the procedure and the temperament of the animal. 2% lignocaine with adrenaline is the most widely used local anaesthetic, although at the time of writing it is not licensed for use in food animals as maximum residue limits have not been set. 5% procaine is licensed but is an inferior anaesthetic. For epidural anaesthesia, it is safer if a product without adrenaline is used but these are becoming unavailable. In all cases correct cleansing and disinfection of the site of injection is assumed.

Anaesthesia for surgery of the flank (most commonly caesarean section)

Options are:

- Local infiltration along the incision line or as an 'inverted L' above the incision site. For adult sheep the maximum volume of 2% lignocaine used should not exceed 3 ml/kg; in practice it is rarely necessary to use anything like such a large amount.
- Paravertebral anaesthesia – this is a very satisfactory and satisfying method of anaesthetising the flank, uses a minimum volume of anaesthetic, produces good relaxation of the site and avoids the problems of haematoma formation and patchy anaesthesia associated with local infiltration. However, it may be difficult to carry out in very fat animals in which it is difficult to identify the landmarks for injection. In addition, the thickness of the lumbar muscles and overlying fat mean the narrow lumbar transverse processes can be difficult to locate.

The technique to block spinal nerves T13, L1, L2 and L3 requires identification of the first, second and third lumbar transverse processes. In thin sheep it is easy to palpate these; in fatter animals, locate the fifth lumbar transverse process which lies just in front of the external angle of the ilium and count forward to find the first process which lies just behind the last rib. Inject a small amount (0.5–1 ml) of anaesthetic under the skin over the midpoint of each process. After a few minutes, using a long needle (19G × 6 cm is usually adequate), block T13 by inserting the needle 4–5 cm from the midline over the centre of the first lumbar transverse process until bone is felt. This process is narrow so it may need more than one attempt to find it. Once the needle has contacted bone, withdraw slightly and redirect the point of the needle forward, 'walking' it off the cranial surface of the bone. Inject 2–3 ml just below the level of the bone, then another 2–3 ml as the needle is withdrawn. The same procedure, except that the needle is directed off the caudal edge of the process, will block L1. L2 and L3 are blocked in a similar manner by injecting off the caudal edges of the second and third lumbar transverse processes. Anaesthesia of the flank will develop in about 10 minutes.

- Caudal epidural anaesthesia – it is possible to produce flank anaesthesia by injection of xylazine (with or without lignocaine) into the sacrococcygeal space (see below). However it has the disadvantages that anaesthesia is slow to develop (about 45 min) and that the animal is off its hind legs for up to 24 h – this is not helpful if there are newborn lambs to consider.

Anaesthesia of the perineal area

In the female, this is necessary to relieve straining and ease replacement of prolapses of the vagina and cervix, and to facilitate replacement of prolapsed uterus. In the male it can be used in cases of urethral obstruction where a urethrostomy or amputation of the penis is to be performed. It is also suitable if amputation of the tail is necessary for therapeutic reasons. The site of injection is preferably the sacrococcygeal space. This joint has relatively little mobility and lies just in front of the much more mobile first intercoccygeal space which is easily identified and can be used if necessary, although the more anterior site gives more reliable anaesthesia. The tail is grasped at its base with one hand and moved up and down, while the exact site is located with a finger of the other hand. A 2.5–3 cm × 19 or 20G needle is inserted at about 45° to the spine, with the point directed cranially and advanced until it is felt to pass between the bones and 'pop' through the interarcuate ligament into the epidural space. If only short-duration anaesthesia is required, 2 ml (for a 60 kg ewe) of 2% lignocaine is injected (preferably plain, but if only a product containing adrenaline is available this does not usually cause problems in sheep). There should be no resistance to injection. If a longer duration of analgesia is required to control straining, a suitable combination is 0.25 ml 2% xylazine added to 1.75 ml 2% lignocaine in a 2 ml syringe for a 60 kg ewe. This will give at least 24 h duration of effect.

Lumbosacral anaesthesia can also be carried out in sheep but is more tricky to perform (it is easy to penetrate into the subarachnoid space in spite of taking care) and the animal always loses the use of the hind limbs for the duration of anaesthesia.

Anaesthesia of the foot

This is necessary for amputation of a digit or removal of a toe granuloma. The most satisfactory method is by use of a tourniquet and intravenous injection of local anaesthetic. It is essential that a good tourniquet that effectively stops blood flow into the leg is used. For a foreleg, apply immediately below the elbow, for a hind leg, as far above the hock as possible. Locate a suitable vein (usually cephalic in the foreleg and recurrent tarsal or saphenous in the hind limb). With a 21G needle inject 5–7 ml 2% lignocaine (with adrenaline if plain unavailable) intravenously. After removing the needle, apply pressure to the injection site to try to prevent haematoma formation. Anaesthesia takes up to 15 min to develop. The tourniquet should not be released in less than 10 min from injection to prevent possible effects on the heart of lignocaine being released into the circulation.

Anaesthesia for castration

It is a legal requirement that sheep over 3 months old undergoing castration should be given an anaesthetic. The most commonly used methods are:

- About 2 ml of local anaesthetic is injected under the skin of the scrotum and a further 3–5 ml is injected into each spermatic cord.
- 3–5 ml of local anaesthetic is injected into each testicle, with a little injected under the skin as the needle is withdrawn.

Anaesthesia develops in about 10 min.

General anaesthesia

General anaesthesia can be employed safely in practice, provided the following precautions are taken before and during anaesthesia and during the recovery phase:

- Starve well, at least overnight, preferably for 24 h.
- Remove water about 6 h before anaesthesia.
- If at all possible intubate with a cuffed endotracheal tube after induction to prevent inhalation of saliva or regurgitated rumen contents (the risk of regurgitation is reduced by starving as above).
- Intubation is facilitated if the head and neck are extended in a straight line, a gag is placed in the mouth, a laryngoscope is used to illuminate the larynx and the endotracheal tube is stiffened by insertion of a wire (a straightened wire coat hanger will do).

- If intubation is not possible, keep the head well extended and lower than the body, pull the tongue well out and ensure that saliva drains freely from the mouth. Keep a very close watch throughout the whole of the procedure that saliva flow is unobstructed.
- If rumen bloat develops, pass a stomach tube to relieve – bloat is less likely if food has been withheld as above.
- At the end of the procedure, place in sternal recumbency as soon as possible and keep under observation until the swallowing reflex is regained.
- If an endotracheal tube has been used remember to remove as soon as safe to prevent accidental chewing.

General anaesthetic agents

Anaesthesia can be induced with 4% halothane, although this is not a desirable method because saliva may be inhaled during induction. The animal should then be intubated and maintained on halothane at 1.5–2%. Alternatively induction can be by injection using one of the following methods:

- Ketamine and diazepam combination – these agents can be administered mixed in the same syringe. If 300 mg (3 ml) ketamine is added to 30 mg (6 ml) diazepam this gives a total volume of 9 ml. This is given intravenously to effect – approximately 6 ml of this mixture will induce anaesthesia in a 50 kg ewe. The remainder can be used for topping up if necessary, but anaesthesia is of short duration only and it is best to intubate and maintain on halothane as above.
- Pentobarbitone sodium 6% – this old-fashioned, cheap anaesthetic is no longer licensed but has been satisfactorily used by the authors for many years. The dose in healthy unsedated animals is approximately 1 ml/2.2 kg body weight. It is rapidly metabolised, giving about 15 min duration of anaesthesia. It can be used for induction followed by intubation and gaseous anaesthesia, or can be used alone and topped up as necessary, although this will increase recovery time. If it is to be used by topping up, it is much easier if a catheter has been inserted into a vein – the cephalic vein is very useful. Induction and recovery are generally smooth. As well as observing the general precautions listed above, it is important to note the following points:
    - inject about one third of the computed dose, then inject slowly to effect allowing plenty of time between increments. If given too fast it is easy to overdose
    - in the case of sedated or debilitated animals or those with neurological problems, the dose required is considerably less than for healthy animals – give no more than a quarter of the computed dose and top up slowly to effect as above
    - intravascular haemolysis has been reported (but not experienced by the authors), although an apparent adverse reaction (marked excitement) has been seen by one of the authors in a single sheep (also to diazepam in the same animal).

General anaesthesia of very young lambs

Very young lambs can also be safely anaesthetised, but remember to:

- Keep under anaesthesia for the minimum possible time.
- Guard against excessive heat loss.
- Feed as soon as conscious (with a stomach tube if necessary) to prevent hypothermia developing.

The safest methods are with inhalation anaesthetics, or by using the (unlicensed) steroid mixture alphaxalone/alphadolone (Saffan) by intravenous injection. This product gives safe anaesthesia for approximately 10 min and has a smooth rapid recovery. The dose is about 2–2.5 ml for a lamb (or goat kid – it is a very good choice for disbudding kids) of under a week or so of age. Give half the dose into the cephalic vein, top up to the desired effect and do the job quickly. This product can also be used for older animals but is generally too expensive.

Common surgical procedures

Again, it is assumed that the operation site will be correctly prepared and disinfected before any surgical procedure is undertaken. Appropriate antibiotic and analgesic cover is also assumed.

Lambs

Atresia ani

This can usually be corrected provided the blind end of the rectum is present within the pelvis. It is easier to locate in a lamb of a few days old when intestinal contents have accumulated and produce obvious distension of the rectum. The anal area should be anaesthetised by injecting a small amount of anaesthetic. A cruciate incision is made through the skin where the anus would be. The end of the rectum is usually visible as a dark sac which becomes more obvious if a little pressure is applied to the abdomen. If this is not easily found, the lamb should be euthanased as the defect is unlikely to be reparable. Having identified the closed sac, it should be opened with a scalpel blade. A large quantity of meconium will be voided. When this has ceased to be passed, clean the area and identify the rectal mucosa (it looks like dark red velvet). Attach the wall of the rectum to the skin using a suitable number of simple interrupted sutures of absorbable suture material. Most do well, although some cases have other developmental defects. The anal area should be kept clean and if necessary a thermometer can be gently used to make sure that patency is maintained.

Entropion

These rarely need surgery if identified and treated early. In severe cases which cannot be corrected by simpler methods, surgery to remove excess skin can be

carried out as in dogs – this should, however, rarely be necessary. In newborn lambs, simply pulling the lower lid out into its correct position may be all that is required. If this is not effective, or in slightly older lambs that have not been noticed at birth, the eyelid should be corrected then maintained in position by injecting liquid paraffin or an antibiotic such as procaine penicillin into the lower lid in such a way that the conjunctiva is caused to 'balloon out'. To do this, insert a 20 or 21G needle through the skin of the lower lid, starting about 0.5 cm below one corner of the eye and parallel to the eyelid. The needle should lie between the skin and conjunctiva. Inject 1–1.5 ml of material to ensure that the lid does not roll inwards again.

The owner of affected lambs should be reminded that this is a genetic defect and that treated animals, particularly rams, should not be kept for breeding. Unfortunately the simple correction methods described above leave no evidence so many treated animals are kept for breeding, thus perpetuating the problem.

Fractured limbs

Fractures are common in young lambs. If below the carpus or hock, these almost always heal well. Fractures higher up the limbs are difficult to immobilise and euthanasia should be considered unless the lamb is valuable, in which case the same techniques as used in small animal orthopaedics can be applied. For simple fractures of the lower limbs, splinting is preferred rather than application of a cast. Young lambs grow so fast that casts are liable to become too tight very quickly. A splint can be made of plastic pipe of suitable diameter and length split in half. Sharp edges should be smoothed and adequate padding applied to prevent pressure sores developing. The advantages of splinting are that splints are light in weight and can easily be removed to check the progress of healing, then replaced as necessary.

Prolapsed intestines

This catastrophe occurs at birth or soon after, either as the result of a defect in the abdominal wall at the umbilicus, or as a result of excessive licking and pulling of the umbilical cord by an attentive ewe. If the intestines are damaged or very contaminated (barley awns are almost impossible to remove), the lamb should be euthanased. If the intestines are viable and not badly contaminated, the lamb should be anaesthetised as described above. Warm saline is used to clean and warm the prolapsed intestines. It is almost always necessary to enlarge the umbilical opening in order to replace them – this should be done carefully to avoid nicking. The remnants of the umbilical cord will also need to be removed. Having successfully replaced the intestines, the abdominal incision and defect should be securely closed with adequate sutures.

Growing sheep and adults

Amputation of digit

This is required in cases of severe infections of the pedal joint. After application of a tourniquet and anaesthetising the foot, an incision is made above the coronary band of the affected digit, as close as possible, but above any discharging sinuses. Great care must be taken with the incision in the foot cleft as it is possible to damage the pedal joint capsule of the sound claw if the incision strays too far towards that side. The incision is then deepened until the bone (P2) is exposed. The claw is then removed either by disarticulation at the proximal interphalangeal joint, or with embryotomy wire by sawing through the lower part of P1. Any necrotic tissue should be trimmed away, then a nonstick dressing applied to the wound. The foot is then padded with cottonwool and a firm pressure bandage applied, and finally a waterproof dressing. It is essential that this first bandage is firmly applied and does not slip off, otherwise the animal may bleed to death. Redressing should take place after 3–4 days, then at intervals of 5–7 days. Healing takes place in 4–6 weeks.

Caesarean operation

Caesarean operation has a high success rate provided the ewe has not been damaged during excessive attempts to lamb it, and the lambs are alive or freshly dead. Survival rates are lower if the posterior tract has been damaged or if the fetus(es) is/are autolysed and smelly. In such cases euthanasia should be considered. The site for surgery is the left flank but the exact site will be the personal preference of the surgeon. As long as the incision is kept away from the landmarks of last rib, external angle of the ilium and mammary vein, there should be no problems in repairing it. An incision higher in the flank is less likely to break down as there is more muscle present. However, a lower incision makes access to and exteriorisation of the uterus easier, which is important if the uterine contents are infected.

The abdominal musculature is thin, especially in the lower flank, and it is necessary to take care to avoid accidental incision into the rumen during the making of the flank incision. A uterine horn containing an extremity of a lamb should be exteriorised, then a small incision made with a scalpel over the lamb (taking care not to accidentally cut it), then the incision enlarged with scissors by cutting along the greater curvature of the uterus, avoiding cutting too close to the ovary. The lamb can then be removed. Still with the uterine horn held outside the abdomen, the other horn should be checked for the presence of more lambs. These can usually be removed through the same incision, although the fetal membranes sometimes make it difficult.

If the hand is first directed towards the cervix it is usually possible to find the lambs in the other uterine horn. Before repairing the uterus, a check must be made that all lambs have been removed – it is inexcusable (but known) for lambs to be left inside. If the fetal membranes are detached, they should be removed,

otherwise trim off any long pieces and leave the remainder *in situ*. The uterine incision is closed with a continuous inverting suture pattern. Most surgeons will prefer to insert a second suture layer unless very satisfied with the closure produced by the first. The uterus is then replaced in its correct position inside the abdomen. If desired, antibiotic can be placed into the abdominal cavity.

The flank incision is then closed, usually with two or three layers of sutures, the exact pattern being according to individual preference of the surgeon. If the lambs are alive, attention must be given to resuscitation, then they should be placed in a box to prevent them becoming chilled. Administration of colostrum from the ewe or other source helps to ensure survival.

Castration

Many lambs are castrated with rubber rings within the first week of life, or with a bloodless castrator at under 3 months of age, when an anaesthetic is not a legal requirement. It may be necessary to castrate older hill lambs when they are brought off the fells, or lowland lambs that have been delayed in fattening for reasons of shortage of food or disease. Most are castrated under local anaesthesia only. Open methods include cutting off the lower part of the scrotum, exposing the testicles and pulling each, with or without twisting the spermatic cord, until it breaks. In older animals with large testicles, it is advisable to use emasculators to guard against haemorrhage. Newly castrated animals should either go back out to clean grass or be placed in a well-bedded shed. They should have completed a full course of vaccination against clostridia as tetanus is a distinct risk in unvaccinated animals.

Gid cyst removal

This is a technically rewarding procedure, though rarely financially rewarding. The likely site of the cyst must be determined by a full neurological examination as described in Chapter 15. The procedure should be carried out under general anaesthesia, although in long-standing cases with a cerebral cyst, the bone overlying it may be completely eroded away and it may be possible to use local anaesthesia. There are two operation sites. Generally a suitable trephine is needed, unless the skull is very softened.

Cerebral cyst removal

The site is just behind the horn or the depression where the horn would be, on the side opposite the eye with the visual defect. A semicircular incision is made over the site and any tissue overlying the bone reflected. The periosteum should also be reflected from the bone with a suitable instrument. A trephine of 1–1.5 cm is then used to remove a circular piece of bone. This must be done with care to avoid suddenly entering and damaging the brain below.

Having removed the bone, the dura mater, if undamaged by the trephine, will be seen slightly bulging into the hole. This should be incised. The under-

lying brain tissue will then bulge from the trephine hole indicating increased intracranial pressure. If this does not occur, the diagnosis is likely to be incorrect. Sometimes a superficial cyst will be immediately visible with its pale grey translucent wall and white scolices visible through it. If so, grasp the cyst wall with a small pair of forceps. The wall is quite fragile so should be handled carefully. Having grasped it, fluid can be withdrawn with a syringe and needle until it is possible to lift the cyst out. If the cyst is not immediately visible, a 16 g cannula with needle in place should be inserted into the brain tissue to locate it. When the needle is removed and clear fluid flows from the cannula, a 20 ml syringe is firmly attached and negative pressure applied to try to draw the cyst lining into the tip of the cannula. This is carefully withdrawn, the cyst lining grasped and the above procedure followed. Any cyst fluid spilt during removal can also be sucked out with the cannula, although it does not seem to matter if some remains. The skin incision only is then sutured.

As well as antibiotic cover, recovery is more likely if soluble steroid is administered intravenously to reduce postoperative swelling (if the animal is pregnant the risk of abortion must be weighed against the risk of brain swelling). Recovery can be dramatic provided the brain tissue is not damaged by excessive probing. In particular, the blood vessels in the central division between the two cerebral hemispheres must be avoided as any significant haemorrhage will greatly reduce the success rate.

Cerebellar cyst removal

This site is much more technically difficult, since access is poor. The site is between the nuchal ridge and the transverse bone suture line about 1.5 cm in front of it. A trephine of 0.5 cm is necessary and the hole is made in the midline. The bone is much thicker than in the cerebral site. The same procedure of using a catheter and syringe to extract the cyst is followed but is made difficult because of the thicker bone and restricted access.

Toe granuloma removal

These usually result from overparing at the toe and although loose horn will grow over and partially or fully conceal them, surgery is usually necessary to permanently remove them. With the foot anaesthetised by intravenous local anaesthesia, the overlying horn is carefully removed to expose fully the granuloma. This is then cut off at the base, and heat (a calf disbudding iron is usually most readily to hand) is used to cauterise the base. It is important to thoroughly cauterise so that no bleeding points remain. In most cases this will result in a satisfactory cure and regrowth of normal horn.

Urolithiasis

See also Chapter 11. The options, if amputation of the urethral appendage does not resolve the problem, depend on the type and value of the affected animal. These are:

- Euthanasia.
- Perineal urethrostomy – this is carried out under caudal epidural anaesthesia. A vertical incision is made in the midline 6–8 cm below the anus. The penis is exposed by blunt dissection, and the urethra is identified on the surface of the penis and is opened. If urine still does not flow, a dog catheter can be introduced and passed towards the bladder. This may enter the urethral diverticulum rather than entering the bladder. Retrograde flushing with water through the catheter may remove the obstruction. If successful the incision is left open and will gradually close without further intervention.
- Amputation of the penis – this can be performed if urine has leaked into the tissues of the abdomen through a rupture of the distal urethra. The penis is exposed as above, but is freed from surrounding tissues. The penis is then transected at a point which will leave a stump of about 2.5 cm protruding from the incision. The dorsal artery of the penis (which will lie on the ventral aspect of the stump) should be identified and ligated. Blood will ooze from the corpus cavernosum penis but this should not lead to significant blood loss. The skin incision is then closed, leaving the stump of the penis protruding from the incision.
- Placement of a Foley catheter percutaneously into the bladder – this is worth attempting in breeding rams in an attempt to salvage them for breeding purposes. This can be done either by means of a paramedian abdominal incision, in which case the catheter is placed into the bladder by direct vision and exits through the abdominal wound, or can be performed with the aid of ultrasound. In either case, once the catheter tip is in the bladder the balloon is inflated to retain it in position and urine is allowed to drain through it. Walpole's solution should be introduced into the bladder twice daily through the catheter, which should then be closed by means of a clamp for about an hour. This helps to dissolve calculi in the bladder and urethra. If the procedure is successful the ram will begin to pass urine through the penis. This usually takes 10–14 days to achieve. Once urine flow is satisfactory (test by clamping the catheter for longer periods), this can be removed.

Vasectomy

This is a commonly requested procedure but is one that can have legal repercussions if not correctly performed. In choosing suitable animals to be vasectomised the following guidelines should be observed:

- Choose a young animal, not an old ram.
- Choose a different type from the stock rams so that no mistakes are made about identity, e.g. different face colour, horns, or leave a long tail. If this is not possible for some reason, e.g. a closed flock, make sure that the vasectomised animals have some form of easily visible permanent identification. In any case, use of an eartag specific to the practice is recommended.

- Choose reasonably lean animals so that there is not too much fat around the operation site.

There are three different forms of anaesthesia that can be used:

- Sedation and local infiltration of the operation site – this has the disadvantage that haematomas may form where anaesthetic is injected, and also that relaxation of the spermatic cord is not good.
- Caudal epidural anaesthesia using xylazine – this gives good anaesthesia and relaxation of the spermatic cord but has the disadvantage that anaesthesia takes at least 45 min to develop, and also that the animal will be off its hind legs for 24 h.
- General anaesthesia – this gives good relaxation but carries the usual risks of general anaesthesia.

It is essential for a successful vasectomy that as long a piece as possible of each vas deferens is removed – at least 5 cm. If this is done, recanalisation is extremely unlikely. This is most difficult to achieve if local anaesthetic is used. The animal can either be placed in a shepherd's chair (not if under general anaesthetic), or on a table in dorsal recumbency. After preparation of the site, which is just below the level of the teats, one spermatic cord is grasped and tensed, then a longitudinal incision is made through the skin overlying it. The spermatic cord is freed by blunt dissection and lifted from the incision by inserting a closed pair of curved forceps under it. The vas (which looks like a bootlace) is identified through the tunica vaginalis – usually the spermatic cord has to be rolled somewhat to expose the medial aspect where the vas lies.

A small incision is made through the tunica vaginalis over the vas which usually bulges out of the incision. Alternatively it can be extracted with an instrument such as a spay hook. A pair of artery forceps is applied as far down as possible and the distal part of the vas is then ligated with nonabsorbable suture. A second pair of forceps is applied close to the first and the vas is divided by twisting off (this is an insurance against bleeding should the ligature slip). As long a piece as possible of the vas is then exposed by traction and the same procedure followed proximally to remove it. Extra safeguards against recanalisation are to turn the end of the vas back over itself and incorporate in the ligature, and to anchor the proximal end in the fascia outside the vaginal sac. The skin incision is then closed and the procedure repeated on the other side.

The lengths of vas removed should be retained for confirmation that the correct tissue has been removed. One method is to squeeze the contents onto a slide and examine under the microscope – sperm should be present. Another alternative is to submit the pieces to a laboratory for histological examination. A third option for experienced surgeons is to place in formalin (a plain vacutainer is ideal), label and store in case of future problems arising.

Appendix 9
Possible new production and disease patterns

Global disease patterns are not, and perhaps never have been, static. However, a number of factors make the situation less stable than previously, although the sheep industry is less vulnerable than some species because of a less intense interface between man and animals. The factors which may bring about changes and make it imperative to maintain vigilance for the occurrence of new diseases include the following.

Climate change

- The probable northward extension of continental weather patterns will influence both production and disease patterns. Production changes will be very gradual and of limited significance during present lifetimes. The high production capabilities of temperate grassland in southern areas may well become lower during the critical high demand period of lamb growth, but this should be compensated for by higher output in the more northern areas in which the greater concentration of sheep occurs.
- While there will be no change of photoperiodic effect on reproduction, higher temperatures could have an effect on male fertility. Any such change could also make the introduction of finer woolled breeds (with their fertility patterns) more economically viable.
- As far as disease patterns are concerned, the greatest risk is extension of vector-spread conditions. The insect vectors of southern Europe will not recognise the English Channel as a barrier. Blue tongue and even diseases of North Africa may sooner or later appear within the spectrum of our disease pattern.

Changes through increased mobility of stock

The greatest risks will come through an increase in international trade in live animals. More sophisticated livestock transporters which enable longer distances to be travelled increase the risk. The extension of the EU to include countries of eastern and southern Europe also increase the risk of the arrival of exotic diseases. The best efforts of those of high integrity have failed to ensure the safety of international boundaries – for example BSE and FMD have

travelled from the UK to mainland Europe while brucellosis and warble fly have travelled in the opposite direction.

Exotic organisms and diseases that may enter the UK through livestock movements include:

Brucella ovis (orchitis and epidydimitis)
Brucella melitensis (abortion, mastitis)
Mycoplasma agalactiae (contagious agalactia)
Mycoplasma mycoides (pleuropneumonia)
Capripoxvirus (sheep and goat pox)

Human influence

- Recreational use of farmland – while it is now accepted that the general public has a 'right to roam', there may be an adverse effect by the leaving of litter. Also, if people come from an area where a significant disease is endemic, there may be direct spread of disease, for example FMD.
- Illegal importation of infected meat products – as seen with FMD and classical swine fever.
- Environmental concerns – while it is right that the general public has a say in the methods of production of food, and on the environment food animals are kept in, there can be some negative consequences. On some occasions the public concept of reality can be more significant than reality itself. Thus the perception that a landscape is 'ancient' and therefore must be preserved can be a mistaken one – resistance to clearance of bracken is a classic example.
- Welfare concerns – the public concept of welfare is open to manipulation. While this has led to welcome resistance to and legislation on intensive methods of production, it has also led to resistance on control of vectors of disease, e.g. the badger and tuberculosis debate.

Intensification of production

Intensive production is less of an issue in the sheep industry than in any other livestock area and we jeopardise our 'green' image at our peril. However, there are real welfare concerns in relation to high hill production. If public perceptions lead to more extensification, there is a substantial risk of the known penalties of intensification being exchanged for the less obvious one of climate challenge.

Appendix 10
Standard reference values

The following values are for guidance only. 'Normal' values may vary slightly according to the test method used (especially in the case of enzymes). The guidance of the particular laboratory performing the test should always be followed.

Haematology

RBC	9–15 $\times$ 10^{12}/l
Hb	9–15 g/dl
PCV	0.27–0.45
MCV	28–40 fl
MCHC	31–34 g/dl
Reticulocytes	0%
WBC total	4–12 $\times$ 10^{9}/l
Lymphocytes	2–9 $\times$ 10^{9}/l (40–75%)
Neutrophils	0.7–6 $\times$ 10^{9}/l (10–50%)
Monocytes	0–0.7 $\times$ 10^{9}/l (0–6%)
Eosinophils	0–1 $\times$ 10^{9}/l (0–10%)
Basophils	0–0.3 $\times$ 10^{9}/l (0–3%)

Enzymology

AST	<60 iu/l
CK	<50 iu/l
GGT	<30 iu/l
GLDH	<2 iu/l
GSH-Px	>40 iu/ml RBCs (Se equivalent> 1.3 µmol/l) is adequate <13 iu/ml RBCs (Se equivalent <0.6 µmol/l) is deficient
Pepsinogen	<1 iu/l
SDH	<5 iu/l
SOD	>0.4 iu/mg Hb in lambs, >0.3 iu/mg Hb in ewes
TK	>90 iu/ml RBCs indicates CCN. <12 indicates not CCN.

Biochemistry

Albumin	25–35 g/l
BHB	<1 mmol/l

Bilirubin	<10 μmol/l
Ca	2–3 mmol/l
Creatinine	<150 μmol/l
Cu	10–20 μmol/l
Fibrinogen	1–5 g/l
Glucose	2–3 mmol/l
IgG	25–40 g/l
Inorganic P	1–2.5 mmol/l
Mg	0.7–1.5 mmol/l
Protein (total)	60–80 g/l
Se	>1.3 μmol/l is adequate, <0.6 μmol/l is deficient
Urea	3–8 mmol/l
Vitamin B_{12} (Co)	>0.15 pmol/l
Vitamin E	>2.3 μmol/l (>25 μg/g in liver)

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Appendix 11
Abbreviations

AGID	Agar gel immunodiffusion test
AI	Artificial insemination
AST	Aspartate aminotransferase
AV	Artificial vagina
BD	Border disease
BHB	Beta hydroxybutyrate
BVDV	Bovine viral diarrhoea virus
Ca	Calcium
CAE	Caprine arthritis-encephalitis
CCN	Cerebrocortical necrosis
CFT	Complement fixation test
CK	Creatine kinase
CLA	Caseous lymphadenitis
CNS	Central nervous system
Co	Cobalt
CP	Crude protein
CSF	Cerebrospinal fluid
Cu	Copper
DM	Dry matter
DUP	Digestible undegradable protein
DVM	Divisional Veterinary Manager
EAE	Enzootic abortion of ewes
EDTA	Ethylene diamine tetra-acetic acid
ELISA	Enzyme-linked immunosorbent assay
EM	Electron microscope
ET	Embryo transfer
ETEC	Enterotoxigenic *E. coli*
FAT	Fluorescent antibody test
FIGLU	Formiminoglutamic acid
FMD	Foot and mouth disease
GGT	Gamma glutamyl transferase
GLDH	Glutamate dehydrogenase
GSH-Px	Glutathione peroxidase
HAT	Haemagglutination test
Hb	Haemoglobin
IFAT	Indirect fluorescent antibody test

IgG	Immunoglobulin G
IHA	Indirect haemagglutination
LAT	Latex agglutination
M	Maintenance requirement
MCHC	Mean corpuscular haemoglobin concentration
MCV	Mean corpuscular volume
Mg	Magnesium
MJ	Megajoules
MLC	Meat and Livestock Commission
MMA	Methylmalonic acid
Mo	Molybdenum
MV	Maedi-visna
NSAID	Nonsteroidal anti-inflammatory drug
NSP	National Scrapie Plan
OMAGOD	Ovine mouth and gum obscure disease
OP	Organophosphate
OxF	Oxalate/fluoride
P	Phosphorus
PCR	Polymerase chain reaction
PCV	Packed cell volume
PGE	Parasitic gastroenteritis
PI3	Parainfluenzae 3 virus
PME	Postmortem examination
PMSG	Pregnant mares' serum gonadotrophin
POM	Prescription-only medicine
RBC	Red blood cells
S	Sulphur
SAT	Serum agglutination test
SDH	Sorbitol dehydrogenase
SOD	Superoxide dismutase
SORP	Suboptimal reproductive performance
SP	Synthetic pyrethroid
SPA	Sheep pulmonary adenomatosis
TK	Transketolase
WBC	White blood cells
ZN	Ziehl Neelson

Appendix 12
Further reading

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Journals

Proceedings of the Sheep Veterinary Society (annually). Contact Sheep Veterinary Society at SVS Secretariat, Moredun Research Institute, Pentlands Science Park, Bush Loan, Penicuik, Midlothian EH26 0PZ. Tel. 0131 445 5111.

Sheep Farmer (bimonthly) Contact The National Sheep Association, The Sheep Centre, Malvern WR13 6PH. Tel. 01684 892661.

In Practice (monthly) from British Veterinary Association, 7 Mansfield Street, London W1G 9NQ. Tel. 020 7636 6541.

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