FOURTH EDITION

DISEASES OF THE GOAT

JOHN MATTHEWS

WILEY Blackwell

Diseases of the goat

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4TH EDITION

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Preface to the fourth edition

It is now 25 years since the first edition of *Diseases of the Goat* was published as *Outline of Clinical Diagnosis of the Goat* and 7 years since the third edition was published. The original concept was to provide a reasonably priced text that would provide useful and practical information for veterinary surgeons, whether they were in farm animal, mixed or small animal practice, and that would also be of use to students and goatkeepers. Despite the plethora of information that is now available on the Internet, I still believe that the book provides a valuable source of information that is readily accessible, whether kept in the car, surgery or on the farm.

The identification of a new disease in ruminants throughout Northern Europe, caused by Schmallenberg virus, which followed the arrival of blue tongue virus earlier in the century, and outbreaks of tuberculosis, long thought by British goatkeepers to be of no importance in goats, which occurred in both commercial and show herds, emphasised the fact that no country is an island, let alone an individual farm, so I have included more information on exotic diseases in this edition. In response to requests from readers of the third edition, I have expanded the chapter on poisonous plants to make it more relevant for readers out-with the United Kingdom and included information on predators, euthanasia, post-mortem techniques and fracture repair. As in the previous editions, I have tried to include new references that are likely to be relevant to the veterinarian in practice and updated the information throughout the book.

I hope that this new edition will continue to provide general practitioners with the support they need when dealing with caprine patients.

Acknowledgements

As with the previous editions, I am extremely grateful to my wife Hilary, who has provided encouragement and support during the compilation of this edition and given valuable advice on goat husbandry.

Tony Andrews, David Harwood, Peter Jackson, Katherine Anzuino and Leigh Sullivan have supplied photographs that are reproduced with their permission and I am pleased to acknowledge their contribution and that of their colleagues involved with the clinical cases to which they relate. Peter Cox supplied photographs for the cover.

I am pleased to acknowledge the contribution of the many members of the Goat Veterinary Society and the American Association of Small Ruminant Practioners, whose tips and advice, which they have willingly shared with other veterinary surgeons and goatkeepers, I have incorporated in this edition.

Author's note

For many medical conditions, there are no drugs available that are specifically licensed for use in goats. Dose rates are quoted in the book for many unlicensed drugs. These drug rates have been obtained from published reports, data held on file by the drug manufacturers and from personal experience. Whenever possible, the clinician should use drugs that carry a full product licence, both for goats and for the condition being treated. In all cases where unlicensed drugs are used, milk should not be used for human consumption for a minimum of 7 days and meat for a minimum of 28 days following the administration of the drug. Not all the drugs mentioned have a current licence for food-producing animals in the United Kingdom. It is the reader's responsibility to ensure that he/she is legally entitled to use any drug mentioned.

CHAPTER 1
Female infertility

The normal female goat

In temperate regions, female goats are seasonally polyoestrus. Most goats are totally anoestrus in the northern hemisphere between March and August, although fertile matings have been recorded in all months of the year. Anglo-Nubian and pygmy goats in particular have extremely long breeding seasons. Recently imported goats from the southern hemisphere may take time to adjust to a new seasonality. The breeding season is initiated largely in response to decreasing day length, but is also dependent on temperature, the environment (particularly nutrition) and the presence of a male. Decreasing day length also stimulates reproductive activity in the buck. Table 1.1 details the reproductive aspects of the goat.

Table 1.1 Reproduction in the goat.

Breeding season	September to March (northern hemisphere)
Puberty	5 months
Age at first service	4 to 6 months (male) 7 to 18 months (female)
Oestrus cycle	19 to 21 days (dairy goats) 18 to 24 days (Pygmy goats)
Duration of oestrus	24 to 96 hours (usually 36 to 40 hours)
Ovulation	24 to 48 hours after start of oestrus
Gestation length	150 days (145 to 156 days)
Weight at first mating	60–70% of predicted adult weight ~30 kg for meat goats 30–40 kg for dairy goats

Investigation of female infertility

> Because of the seasonal pattern of breeding, infertility must be investigated as early as possible in the breeding season.

The investigation of female infertility in the goat presents major difficulties when compared with the cow because of the inability to palpate the ovaries and because of the seasonal pattern of breeding – does are often presented towards the end of the season, limiting the time available for remedial measures. Figure 1.1 lists possible causes of infertility in the doe.

Initial assessment

The preliminary history should consider:

- Individual or herd/flock problem.
- Feeding, including mineral supplementation.
- Management practices – hand-mating, artificial insemination (AI), buck running with does.
- Disease status of herd/flock.

If there is a *herd problem*, investigate:

- Male infertility (Chapter 3).
- Intercurrent disease – parasitism, footrot, etc.
- Nutritional status – energy or protein deficit, mineral deficiency (phosphorus, copper, iodine, manganese).
- Stress – overcrowding, recent grouping of goats.
- Poor heat detection.
- Services at incorrect time.

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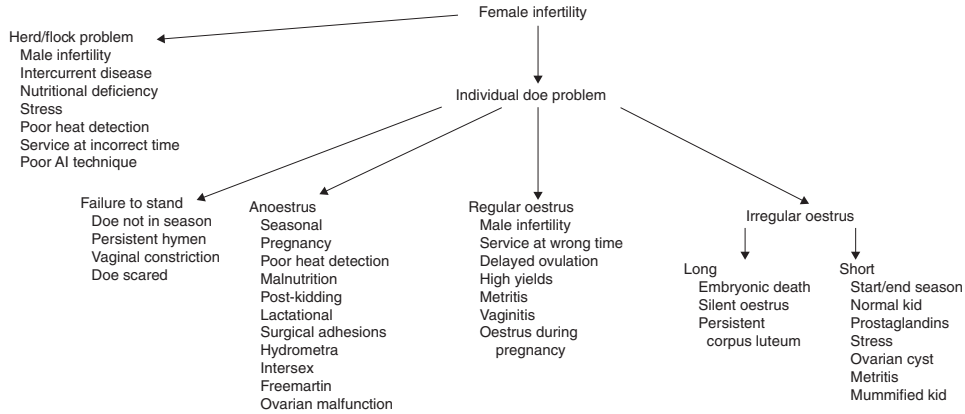

Figure 1.1 Causes of female infertility.

Assessment of individual doe

General assessment

- Conformation.
- Body condition.
- Dentition.
- Clinical examination.

Any obvious clinical signs such as debility, anaemia or lameness should be investigated and corrected where possible before commencing specific therapy aimed at correcting a reproductive disorder.

In the UK overfeeding is probably a greater cause of infertility than poor condition.

Specific examination

- Specific examination of the reproductive and mammary systems. Include, where necessary, examination of the vagina and cervix with a speculum to identify anatomical abnormalities.

Specific history

- Date of last kidding/stage of lactation.
- Daily milk yield.
- Presence or absence of obvious oestrus signs.
- Length of oestrus cycles.
- Date of last service.
- Willingness to stand for male.
- Kidding difficulties last time – malpresentation/manipulation, metritis, retained placenta, abortion, mummified fetus, stillbirths.

Further investigations

- Specific laboratory tests:
- Progesterone assay
- Oestrone sulphate assay
- Bacteriological examination of vaginal or uterine samples
- Feed analysis
- Real-time ultrasound scanning
- Laparoscopy or laparotomy.

Individual infertility problems

Individual infertility problems will generally fall into one of four categories:

1 **Difficulty at service**.
2 **Anoestrus.**
3 **Irregular oestrus cycles**.
4 **Regular oestrus cycles**.

Difficulty at service

- Doe not in season.
- Doe scared – common with maiden animals, particularly if a large buck is used on a small doe.
- Persistent hymen or vaginal constriction.

Anoestrus

> Always consider the possibility of an undetected pregnancy (even if the owner insists that no mating has occurred) before attempting treatment, particularly with prostaglandins.

The causes of anoestrus are listed in Table 1.2 and discussed below.

- *Seasonal*. Most goats are totally anoestrus between March and August.
- *Pregnancy*.
- *Poor heat detection*.

Although some dairy goats show only minor behavioural changes during oestrus, oestrus detection is generally easier than in Angora goats, with most does showing obvious signs of tail wagging, frequent bleating, urination near the buck, swelling of the vulva and a mucous vaginal discharge. The signs are generally accentuated in the presence of a male or even a 'billy rag', that is a cloth that has been rubbed on the head of a buck and stored in a sealed jar.

Oestrus can be determined visually by means of a speculum. At the onset of heat, the cervix changes from

Table 1.2 Causes of anoestrus.

Seasonal
Pregnancy
Poor heat detection
Malnutrition
Post-kidding anoestrus
Lactational anoestrus
Adhesions following surgery
Hydrometra
Intersex
Freemartin
Ovarian malfunction

its normal white colour, becoming hyperaemic, and the cervical secretions are thin and clear. The secretions rapidly thicken, becoming grey/white and collecting on the floor of the vagina. Conception is best when mating occurs at the stage at which the cervical mucus is cloudy and the cervix is relaxed.

Unlike cows, most does will not stand to be ridden by other females even when in oestrus. Riding behaviour is sometimes seen as an expression of dominance in the herd or as part of the nymphomaniac behaviour of goats with cystic ovaries. Many young bucks will mount and serve females that are not in true standing oestrus if the female is restrained, although older bucks are more discriminating. The doe will stand to be mated only when she is in oestrus.

In the milking doe, a rise in milk production may occur 8 to 12 hours before the start of oestrus and milk production may fall below normal during oestrus.

When the buck is running with the flock or herd, sire harnesses with raddles or marker paste will aid oestrus detection. A marked vasectomised ('teaser') buck can be used to detect (and help initiate) the start of oestrus in a group of does.

- *Malnutrition*. An energy or protein deficit due either to poor nutrition or intercurrent disease may cause anoestrus. Deficiencies of minerals such as cobalt, selenium, manganese, zinc, phosphorus, iodine and copper and deficiencies of vitamins B12 and D are all reported to cause infertility.
- *Post-kidding anoestrus*. Many does will not show signs of oestrus for 3 months or more after kidding, even if kidding takes place during the normal breeding season.
- *Lactational anoestrus*. Some high yielding does do not exhibit marked signs of oestrus. These animals may respond to prostaglandin injections with careful observation for oestrus 24 to 48 hours later. Animals that do not respond may need a further injection 11 days later.
- *Adhesions following surgery*. The goat's reproductive tract is sensitive to handling and adhesions will occur unless very high standards of surgery are maintained during embryo transplant or other surgical procedures. Talc from surgical gloves will produce a marked tissue reaction.

False pregnancy (hydrometra, cloudburst)

False pregnancy occurs when aseptic fluid accumulates in the uterus in the absence of pregnancy, but in the presence of a persistent corpus luteum, which continues to secrete progesterone. The incidence of false pregnancies is fairly high, particularly in some strains of dairy goats and incidences of between 3 and 30% have been reported in commercial herds.

Aetiology

- A persistent corpus luteum following an oestrus cycle in which pregnancy did not occur. This may occur in any sexually mature female but is particularly common in goats in their second year of a lactation ('running through') without being mated. Certain families seem prone to develop the condition.
- A persistent corpus luteum following embryonic death with resorption of the embryo.
- Occurrence is increased following use of progestagen sponges and treatment with equine chorionic gonadotrophin (eCG).

Clinical signs

- The doe acts as if pregnant, with enlargement of the abdomen and a degree of udder development if not milking (Plate 1.1). Milking does may show a sharp drop in yield and this may result in a significant economic loss if the condition is not corrected.
- Fetal fluids collect in the abdomen (*hydrometra*) and the doe may become enormously distended, although the amount of fluid varies from 1 to 7 litres or more.
- When the hydrometra occurs following embryonic death, the false pregnancy generally persists for the full gestational length, or longer, before luteolysis occurs, progesterone secretion ceases and the fetal fluids are released (*cloudburst*). Some does milk adequately following a natural cloudburst.
- When the false pregnancy occurs in a doe which has not been mated, the release of fluid often occurs in less than the normal gestation period, the doe may cycle again and a further false pregnancy may occur if she is not mated. Subsequent pregnancies are not generally affected, but the doe is likely to develop the condition again the following year. The expelled fluid is generally clear and mucoid. The vulva and perineum become moist and the tail sticky (Plate

1.2). Some goats that spontaneously cloudburst early, before a large amount of fluid has accumulated, have a bloody discharge. The abdomen decreases to a normal non-pregnant size and bedding appears wet. Some does continue to squirt small amounts of fluid for a couple of days and in fat does this could be confused with cystitis.

- If the false pregnancy follows fetal death, fetal membranes and possibly a decomposed fetus are present; otherwise no fetal membranes are formed.

Diagnosis

- Realtime ultrasound scanning of the right ventrolateral abdominal wall in early false pregnancy, or of either flank later, shows large fluid-filled hyperechoic compartments with the absence of fetuses or caruncles (Plate 1.3). The uterus is separated into compartments with thin tissue walls, which undulate when balloted. White flecks may be seen in the fluid. Scanning should take place at least 40 days after mating to avoid confusion with early pregnancy and is easier before 70 days. *Pyometras* (rare) also present as fluid filled uteri but are more hyperechoic.
- Elevated milk or plasma progesterone levels are consistent with pregnancy, but with low milk or plasma oestrone sulphate levels at >45 days.
- X-ray at 70–80 + days fails to show fetal skeletons in an anoestrus doe with a distended abdomen.
- Pregnancy specific protein is negative in pseudopregnancy.

Treatment

- As pseudopregnancy is maintained by the presence of a corpus luteum, treatment is by prostaglandin injection:

  Dinaprost, 5–10 mg i.m.or s.c. or Clorprostenol, 62.5–125 μg i.m. or s.c.

  Dinaprost has a direct effect on uterine muscle and may be preferable to clorprostenol.A second injection of prostaglandin 12 days after the first may cause evacuation of further uterine fluid and, it is suggested, may make the condition less likely to recur.
- An oxytocin injection a few days after treatment with prostaglandin stimulates uterine contractions and aids involution:

  Oxytocin, 2–10 units, 0.2–1.0 ml i.m. or s.c..

  Pituitary extract (posterior lobe), 20–50 units, 2-5 ml i.m. or s.c. or 2–10 units, 0.2–1.0 ml i.m. (preferred) or s.c.
- The prognosis for future fertility is good, with 85% of goats becoming pregnant if mated during the same breeding season.

Other conditions causing anoestrus

- *Hydrops uteri*. A false pregnancy may need to be distinguished from hydrops uteri. Hydrops uteri is an unusual condition of pregnant goats caused by an abnormal accumulation of fluid in either the amniotic (hydamnios) or allantoic (hydrallantois) sacs. Distension of the uterus is caused by accumulation of fluid, which may be greater than 10 litres, leading to bilateral, rapidly progressive abdominal distension. Other clinical signs, similar to those of pregnancy toxaemia, are a result of compression of other organs by the fluid – lethargy, inappetence, decreased defaecation, recumbency, tachycardia and dyspnoea.

  Ultrasonography can be used to distinguish between false pregnancy (hydrometra), where the uterus is distended with fluid but no fetuses, membranes or cotyledons are present, and hydrops uteri, where fluid, fetuses, membranes and cotyledons are present. Most fetuses of animals with hydrops uteri have congenital defects and are underdeveloped, but may appear normal although not viable.

  Treatment is by caesarian section or by induction of parturition with prostaglandins, but cardiovascular support with intravenous fluids should be provided because of the danger of hypotension from the sudden loss of large volumes of fluid.
- *Intersex (pseudohermaphrodite)*. An intersex is an animal that shows both male and female characteristics. In goats the dominant gene for absence of horns (polled condition) is associated with a recessive gene for intersex. Thus an intersex is normally polled with two polled parents. Intersex is a recessive sex-linked incompletely penetrant trait resulting from the breeding of two polled goats – intersex goats are homozygous for the polled (hornless) gene and homozygous for the intersex gene.

  A mating between a homozygous (PP) polled male and a heterozygous (Pp) polled female will produce 50% intersexes; a mating between a heterozygous (Pp) polled male and a heterozygous (Pp) polled female will produce

25% intersexes. In theory, mating two homozygous (PP) polled animals should produce 100% intersexes, but the gene has incomplete penetrance.

Affected animals are genetically female with a normal female chromosome complement (60 XX), but phenotypically show great variation from phenotypic male (Plate 1.4) to phenotypic female (Plate 1.5). Some animals are obviously abnormal at birth with a normal vulva but enlarged clitoris or a penile clitoris. The gonads are generally testes or ovotestes, which may be abdominal or scrotal and phenotypic males may have a shortened penis (hypospadias), hypoplastic testes or sperm granuloma in the head of the epididymis. Other animals may reach maturity before being detected and may present as being anoestrus. A phenotypically female animal may have male characteristics due to internal testes.

Intersexes with female appearance are sometimes presented as kids or goatlings with a history of anoestrus. Although the vulva is normal, there is no true vagina or cervix, the clitoris may be enlarged and the anogenital distance may be > 3 cm. The presence or absence of a vagina of proper length should always be investigated in anoestrus kids. The absence of a vagina can be demonstrated by gently inserting a lubricated plastic rod, for example a ballpoint pen, into the vulva (Plates 1.6 and 1.7) or endoscopically. Care should be taken not to mistake a persistent hymen for a shortened vagina.

Intersexes with male appearance may have a penis or penis-like structure just below the anus. These animals may have urine scalding down their hind legs or have dysuria. Urine may accumulate in the perineal area causing dermatitis. In some cases, the urethra does not pass through the vestigial penis/clitoris and surgery may be required to establish an effective urethral opening. Localised hypospadia has been described in some cases.

- *Freemartins (XX/XY chimeras)*. Most female kids born co-twin to males are normal females, because placental fusion is much less common than in cattle. A freemartin is a female rendered sterile in utero when her placenta and that of her twin male fuses in early gestation, allowing vascular anastomosis between the allantoic membranes, exchange of cells and hormones between the two foetuses and XX/XY chimaerism. The developing genital tract of the female is influenced by the male and results in hypoplasia of the female gonads. A freemartin may be polled or horned. There is some evidence that the condition is slightly more common when the female shares the uterus with two or more male fetuses. Externally freemartins appear female but internally show a variable degree of masculinisation:
  - Heavy masculinisation, Gonads resemble testes and may contain tubules and interstitial tissue.
  - Light masculinisation. Oocytes have been found in the gonads.
- *Whole body chimera*. The rarest type of caprine intersex, which arises from the fusion of two embryos, produces a true hermaphrodite with an XX/XY karyotype and gonads of both sexes.
- *Ovarian malfunction*. Ovarian inactivity is poorly understood in the goat, but some anoestrus goats will respond to treatment with gonadotrophin releasing hormone [GnRH]:
  Buserelin, 0.020 mg i.m., s.c. or i.v. or Gonadorellin, 0.5 mg i.m.
  Other goats will respond to treatment with prostaglandins, suggesting a *persistent corpus luteum* or *luteinised cystic ovaries*.
  Ultrasound scanning can be used to examine the ovaries but is not as easy as in cattle, because the reproductive tract cannot be manipulated manually so it is impossible to scan all the surfaces of the ovary. Both transrectal (using a lubricated 5 or 7.5 MHz linear transducer) or transabdominal (using a 5 MHz transducer) scanning can be carried out with the goat in a standing position. The bladder is located as a landmark and the transducer rotated to the left or right until the ovary is visualised. The ovary appears as a tissue-dense, circular to oblong structure cranial to the bladder. Follicles are non-echogenic fluid-filled structures that appear as black circular sacs.
  Increased use of laparoscopic techniques may aid the diagnosis of these conditions.

Irregular oestrus cycles (see Table 1.3)

Long oestrus cycles

- *Embryonic death*. Early embryonic death with loss of the corpus luteum will produce a subsequent return to oestrus following resorption of the embryonic material. Following embryonic death, a percentage of does will not return to oestrus but develop hydrometra.
- *Silent oestrus*. Some does will exhibit oestrus early in the season and then show no further oestrus signs for

Table 1.3 Irregular oestrus cycles.

Long	Short
Embryonic death	Start/end of season
Silent oestrus	Normal kid behaviour
Persistent corpus luteum	Prostaglandins
	Premature regression of the corpus luteum
	Stress
	Ovarian follicular cyst
	Metritis
	Mummified kid
	Ovarian tumour

some months. These goats may be cycling silently and will respond to treatment with prostaglandins.

- *Persistent corpus luteum*. Failure of the corpus luteum to undergo luteolysis at the correct time will delay the return to oestrus. Treat with prostaglandins (see this chapter).

Short oestrus cycles (<18 to 21 days)

- Short anovulatory cycles of about 7 days are common at the *start of the breeding season* and occasionally occur at the end of the breeding season.
- *Kids* commonly show short cycles during their first breeding season.
- Very short oestrus cycles have been recorded following administration of *prostaglandins* to abort does. A normal oestrus pattern returns after 3 to 4 weeks.
- *Premature regression of the corpus luteum* is recognised as a problem in goats undergoing oestrus synchronisation for embryo transplant. In some cases this will be a result of stress (see below). In other cases, the cause is unknown.
- *Stress* will often cause groups of goats to show short cycles of around 7 days, presumably because of premature regression of the corpus luteum. For this reason goats being brought together for a breeding programme, for example for embryo transplant, should be grouped at least 3 months before the start of the programme.
- *Ovarian follicular cysts* produce oestrogens, which result in a shortened oestrus cycle of between 3 and 7 days or continuous heat. Eventually the oestrogenic effects produce relaxed pelvic ligaments and the goat displays male-like mounting behaviour. The diagnosis can be confirmed by laparoscopy or laparotomy.

  Treatment is exceptionally difficult in goats because the relatively short breeding season means that by the time treatment is completed the doe has already entered seasonal anoestrus. Medical treatment is only successful if commenced early:

  Chorionic gonadotrophin 1000 U, i.m. or i.v. or

  Gonadotrophin releasing hormone (GnRH): buserelin, 0.020 mg i.m., s.c. or i.v. or gonadorellin, 0.5 mg i.m.

  Surgical treatment to exteriorise and rupture the thick wall of the cyst should be considered in valuable animals.
- Ovarian tumours are rare in goats, with granulosa theca cell tumours being the most common type. Clinical signs include short cycles, nymphomania and male behaviour. Examination of the ovary laparoscopically or with rectal or transabdominal ultrasound usually shows an enlarged ovary that may be cystic.
- *Endometritis* may cause short cycling or return to oestrus at the normal time.
- *Vaginitis*: see 'Regular oestrus cycles'.
- The presence of fetal bone remaining from a *mummified kid*, which is not expelled at parturition, will act as a constant source of stimulation and result in short oestrus cycles. There may be a history of bones and fetal material being expelled at kidding or subsequently.

Regular oestrus cycles (see Table 1.4)

- *Male infertility* (Chapter 3).
- *Service at the wrong time*.
- *Delayed ovulation/follicular atresia*. There is little scientific evidence describing these conditions in goats, but in practice a 'holding' injection given at the time of

Table 1.4 Regular oestrus cycles.

Male infertility
Service at the wrong time
Delayed ovulation
High yielders
Metritis
Vaginitis
Oestrus during pregnancy

service or AI will aid fertility in some animals by stimulating ovulation on the day of service:
Chorionic gonadotrophin, 500 U i.m. or i.v.
Gonadotrophin releasing hormone (GnRH): buserelin, 0.010 mg i.m., s.c. or i.v.
Gonadorellin, 0.25 mg i.m.

- *High yielding females*. Some high yielding females may have suboptimum fertility, possibly due to a pituitary dysfunction resulting from the heavy lactation. Maturation of follicles, ovulation and formation of the corpus luteum may be promoted by chorionic gonadotrophin, 500 U i.m. or i.v.
- *Metritis*. A low-grade metritis may result in the failure of the embryo to implant and subsequent return to service at the normal time.
- *Vaginitis*. Vaginitis occasionally occurs, particularly after the removal of vaginal sponges, and may result in short oestrus cycles or repeated return to service at a normal cycle length. In New Zealand, Australia and the United States, *caprine herpesvirus 1 (CpHV-1)* causes vulvovaginitis with short oestrus cycles and resulting infertility. Initial clinical signs are oedema and hyperaemia of the vulva with a slight discharge, which becomes more copious over the next few days. Multiple, shallow erosions with yellow to red-brown scabs develop on the vulvar and vaginal mucosa. Lesions heal spontaneously in about two weeks but may recur. Infection may be subclinical.

  The virus is transmitted venereally and in the male produces penile hyperaemia and erosions of the preputial and penile epithelium. There is prolonged shedding of the virus by the preputial route.

  CpHV-1 is also responsible for lethal systemic infections in one to two week old kids and for subclinical infections of the respiratory tract in adults.
- *Oestrus during pregnancy*. A few goats exhibit regular oestrus signs during pregnancy although this is less common than in cattle. Ovulation does not occur and the signs of oestrus are usually rather weak. Accurate pregnancy diagnosis is important before attempting treatment, particularly with prostaglandins.

Pregnancy diagnosis

> Non-return to service is not a reliable method of pregnancy diagnosis. Many does do not outwardly cycle throughout the breeding season and the non-return may be due to seasonal anoestrus or false pregnancy. Neither is mammary development in primiparous goats a reliable method of pregnancy diagnosis as maiden milkers are common. Nor is abdominal distension.

Although animals may have behavioural changes during late pregnancy (for example, a 'dog sitting' position is normal for some pregnant goats (Plate 1.8), these are very variable. Accurate pregnancy diagnosis is essential to distinguish between pregnant goats, those with false pregnancies and those that are not cycling.

A vasectomised and harnessed teaser male running with the does will detect return to service, that is non-pregnancy, but should not be relied upon as some males will mount females that are not cycling. *Always* undertake an accurate pregnancy diagnosis before using prostaglandins to induce oestrus. Table 1.5 lists the methods available.

Table 1.5 Techniques available for pregnancy diagnosis in the doe.

	Days	Fetal numbers	Accuracy (%)	Usefulness
Vasectomised male	>20	No	65–90	Moderate
Abdominal palpation	60–115	No	60–90	Moderate
Progesterone assay	18–22	No	90–95	Moderate
Oestrone sulphate assay	>50	No	>95	High
Pregnancy specific protein B	>26	No	>95	High
Realtime ultrasound	28–100	Yes	95–100	High
Doppler ultrasound	60–90	No	85–90	Moderate
Radiography	>70	Yes	>90	Low

Oestrone sulphate assay

Oestrone sulphate concentrations in milk and plasma increase steadily during pregnancy and can be used to diagnose pregnancy 50 days post-service. This test will distinguish between true pregnancy and hydrometra, but occasional false negatives do occur, particularly if the sampling is close to 50 days, and repeat sampling may be indicated before the induction of oestrus with prostaglandins to avoid the possibility of aborting a pregnant doe.

Ultrasonographic scanning

Realtime ultrasonographic scanning has the added advantage of giving some indication of the number of kids being carried, thus enabling a better estimate of the nutritional requirements of the doe during pregnancy. The technique is virtually 100% accurate in determining pregnancy and 96 to 97% accurate in determining twins and triplets. Good operators can distinguish hydrometra, resorbed fetuses and other abnormalities as well as live kids (Table 1.6). Goats can be scanned transabdominally or transrectally. Sector scanners are best for transabdominal scanning but linear scanners can be used and are better for transrectal scanning. Transrectal ultrasound techniques are preferred for very early pregnancies and permit diagnosis 4 to 5 days earlier than transabdominal techniques.

Transabdominal scanning is usually carried out with the goat standing. A 3.5 or 5 MHz transducer is suitable for most of the pregnancy, but may not penetrate as far as the foetus in late gestation, although caruncles will be visible. Before about 90 days a 5 MHz transducer gives the best results; in later pregnancy, a 3.5 MHz probe is preferable. Scanning can be used from 28 days post-service when a fluid-filled uterus can be identified, but is best used between 50 and 100 days of pregnancy. Cotyledons can be distinguished from about 40 days and individual foetuses by 45 to 50 days. By 100 days individual fetuses more than fill the entire screen, making accurate determination of numbers difficult (Table 1.7). The most common error is to underestimate the number of fetuses.

The transducer is placed on the right side of the restrained standing doe in the relatively hairless area

Table 1.7 Transabdominal ultrasound scanning for pregnancy diagnosis.

Day of gestation	Ultrasound findings
28	Fluid-filled uterus
30–35	Fetal heart beats detectable
40	Cotyledons visible (doughnuts or c-shaped structures)
45–50	Individual fetuses first identifiable
45–90	Accurate determination of multiple kids Gestational age corresponds to crown rump length, biparietal diameter and chest diameter
>100	Identification of number of fetuses becomes difficult because individual kids fill the screen; fluid and fetuses shift cranially

Table 1.6 Abnormal finding on ultrasonographic examination of the uterus.

Abnormality	Ultrasound findings
Recent abortion	Margins of the enlarged uterus observable, with caruncles often visible but with no fetus or fluid
Hydrometra	Anechoic or hypoechoic fluid-filled uterus, often with membranous strands visualized in the lumen of the uterus or apparent septae within the lumen of the uterine horn
Pyometra	Fluid-filled uterus; fluid more hyperechoic than hydrometra, often has a swirling appearance
Retained mummified foetus	Hyperechoic bone shadows in the absence of fluid contrast Lack of fluid contrast, dense bony shadows, or cranium or ribs in an organized foetal mass Usually smaller than expected foetal mass No sign of viability of fetus
Macerated foetus	Hyperechoic bone shadows in the absence of fluid contrast Overriding bony densities, usually linear or curvilinear images, with no sign of normal fetal architecture

just cranial and dorsal to the udder, with the transducer beam aimed towards the opposite brim of the pelvis (towards the pelvic inlet) and the abdomen scanned by slowly sweeping cranially. The uterus is normally dorsal or cranial to the bladder. Early in pregnancy (30 to 45 days), the uterus lies towards the pelvis inlet, but later is usually against the right abdominal wall. Clipping the area helps in fibre or long-coated goats. The area should be as clean as possible and large amounts of ultrasound gel used.

Transrectal scanning can be carried out from 25 days. Faeces are removed from the rectum and the lubricated 5 or 7.5 MHz linear transducer is advanced gently until it is adjacent to the reproductive tract. Initially the animal should be examined in a standing position, which is generally less stressful for the animal, but if the uterus is not identifiable the doe can be placed in dorsal recumbency.

Foetal viability can be evaluated during ultrasonographic examination, the presence of fetal movement or heartbeat indicating a live fetus. The fetal heart beat can be detected 35 days into the pregnancy by transabdominal ultrasonography (earlier by transrectal ultrasonography). Lack of echogenicity of amniotic fluid, the proper amount of fluid for the gestational stage and normal foetal posture and movement are signs of a healthy fetus. Fetal size incompatible with the expected gestational age may indicate earlier fetal death, as may increased fluid echogenicity, 'floating' membranes, collapsed fetal posture and failure to detect a heartbeat or fetal movement. Hyperechogenicity of the cotyledons is a common finding in a non-viable pregnancy.

Age determination is most accurately carried out early in gestation. Gestational age can be subjectively assessed based on size of the fetus and cotyledons, or the size of the amniotic vesicle in early gestation. Between 40 and 100 days, the length of the fetuses and the fetal head width or biparietal diameter (BPD) correlate closely with gestational age (see Table 1.8 and Figure 1.2). Later in pregnancy, the variation in size of fetuses is too great to permit accurate age determination.

Determination of fetal gender is by visualisation of the male/female genital tubercle or male scrotum. Best results are obtained between 55 and 75 days. Accuracy is decreased when multiple kids are present, because the spontaneous movement and repositioning of the foetuses during the examination makes visualising individual foetuses difficult.

Table 1.8 Correlation of fetal length with age of fetus.

Gestation (days)	Fetal length (mm)
45	40
60	100
90	250

Pre-breeding examination is important, especially in herds with out-of-season breeding programmes, for routine examination of does before assignment to breeding groups will allow detection of animals that would not respond to synchronisation, treatment of abnormal does and identification of does for potential culling. Abnormal findings by ultrasound can be followed by a vaginal speculum exam or other diagnostic procedures.

Doppler ultrasound techniques

Doppler ultrasound techniques can detect the fetal pulse after about 2 months' gestation, using either an intrapelvic probe or an external probe placed on a clipped site immediately in from the right udder or lateral to the left udder using ultrasound gel or vegetable oil to improve contact. Between 60 and 120 days' gestation the accuracy in detecting non-pregnancy is more than 90%, but the method is unreliable in detecting multiple fetuses.

Pregnancy specific protein B

Pregnancy specific protein B (PSPB) is produced by the placenta and is identifiable in plasma or serum, using an ELISA test, from 26 days after mating and then throughout pregnancy, dropping rapidly after parturition, but still detectable for several weeks. It has an accuracy rate of >95% – false positives are likely to be caused by loss of the embryo, rather than inaccuracy of the test. A positive result therefore means that the animal is pregnant or has recently been pregnant (or aborted or resorbed). If it is suspected that a doe has or may have resorbed or aborted, a second blood sample several weeks later would distinguish between pregnancy (continuing high protein level) or non-pregnancy (precipitous drop in protein level). Goats carrying multiple fetuses have higher PSPB concentrations than those carrying singles but there is sufficient overlap to prevent accurate identification of

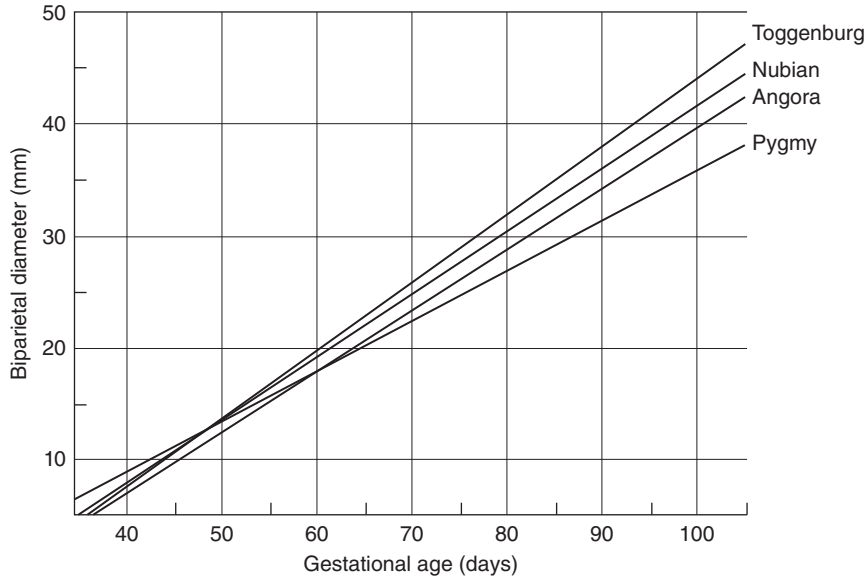

Figure 1.2 Biparietal diameter and gestational age of goats (from Haibel *et al.*, 1989).

single from multiple fetuses. The test is marketed in the United States by BioTracking, Moscow, ID.

Progesterone assay

Progesterone secreted by the corpus luteum of a pregnant goat can be detected by radioimmunoassay or by ELISA methods in milk or in plasma. Progesterone levels remain high throughout pregnancy.

Random sampling will not lead to accurate pregnancy diagnosis because the corpus luteum of the normal oestrus cycle and that of hydrometra also produce progesterone. A sample taken 24 days after mating will give nearly 100% accuracy in determining non-pregnancy but only about 85 to 90% accuracy in determining pregnancy because of factors such as early embryonic death and hydrometra. *A low progesterone level always indicates non-pregnancy*.

Radiography

Fetal skeletons are detectable by radiography between 70 and 80 days, although the technique is more useful after 90 days. An enlarged uterus may be detected at 38 days and over.

Rectoabdominal palpation

In the non-pregnant goat a plastic rod inserted in the rectum can be palpated at the body wall. Between 70 and 100 days post-service, the pregnant uterus prevents palpation of the rod. However, the technique produces unacceptably high levels of fetal mortality and risk of rectal perforation.

Ballotment

Ballotment of the right flank or ventrally is a time-honoured goatkeepers' technique for pregnancy diagnosis, but in the author's experience it is extremely unreliable. Fetal movements can often be observed in the right flank of the doe during the last 30 days of gestation.

Use of prostaglandins

Unlike other ruminants where placenta-derived progesterone becomes significant, the goat depends on corpus-luteum-derived progesterone throughout pregnancy, and is thus susceptible to luteolytic agents, including prostaglandins, throughout the whole of the pregnancy. Prostaglandins can be used for:

- Timing of oestrus.
- Synchronization of oestrus.
- Misalliance.
- Abortion.
- Timing and synchronization of parturition.

- Treatment of hydrometra.
- Treatment of persistent corpus luteum.

> Prostaglandins can be used to terminate pregnancy throughout the whole gestation period.

Suggested doses of prostaglandins in dairy goats are:

Dinaprost, 5–10 mg i.m. or s.c. or Clorprostenol, 62.5–125 μg i.m. or s.c.

Smaller doses will produce luteolysis in Angora goats.

The effect of prostaglandin administration is seen between 24 and 48 hours (generally around 36 hours) post-injection, provided the animal being injected has an active corpus luteum, that is between days 4 and 17 of the normal oestrus cycle or during pregnancy. For induction of parturition where live kids are required, prostaglandins should not be used alone before day 144 of gestation, because prostaglandins bypass the steps involved in producing fetal lung surfactant. Before day 144, dexamethasone should be used and will produce parturition in about 48 to 96 hours (Figure 1.3).

Where rapid termination is required and the viability of the kids is not critical, for example when the doe is collapsed, prostaglandins can be used at any stage of gestation. There is generally no problem with retained fetal membranes following induction with prostaglandins or dexamethasone.

Table 1.9 Methods for controlling oestrus cycles.

Transitory Period	Breeding season	Out of breeding season
Buck effect	Prostaglandin injection(s)	Lighting regimes
Progestagen sponge or CIDR + PMSG	Progestagen sponge or CIDR + PMSG	Progestagen sponge or CIDR + PMSG Lighting regime + Melatonin

Control of the breeding season

Out-of-season breeding is being increasingly used to enable milk producers to maintain regular supplies of fresh milk and to produce three kid crops in 2 years from fibre goats. Best results are obtained when the techniques are used to extend the breeding season, that is by early or late season breeding, rather than in deep anoestrus. Table 1.9 shows methods available for controlling oestrus cycles.

Introduction of a buck or teaser male (buck effect)

Introduction of a buck or teaser male produces oestrus before the start of the breeding season, with loose synchronisation of oestrus. The introduction of a teaser or entire male into a group of does, which have been deprived of the sound, sight and smell of a male for at least 4 to 6 weeks during the transitional period before

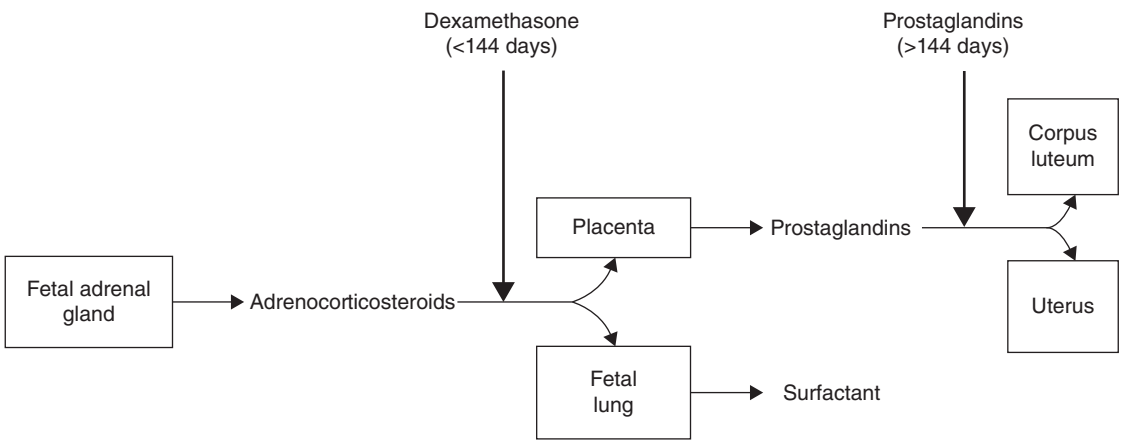

Figure 1.3 Induction of parturition.

the start of the normal breeding season, will produce oestrus cycles within 3 to 10 days, but the first one or two oestruses may be silent, without any sign of behavioural oestrus. A silent oestrus may be followed by a fertile oestrus 21 days later or the first silent oestrus may be followed by a short cycle and a second silent oestrus or fertile oestrus after about 5 days, following premature regression of the corpus luteum.

The fertility of the females after exposure is variable – the closer to the breeding season, the higher the fertility.

Prostaglandin injections

During the normal breeding season, the luteolytic effect of prostaglandins can be used to induce oestrus in animals with a corpus luteum, that is between days 4 and 17 of a normal oestrus cycle. Return to oestrus occurs between 24 to 48 hours post-injection, generally about 36 hours; 60–70% of the herd should respond to a single injection.

Dinaprost, 5–10 mg i.m. or s.c. or Clorprostenol, 62.5–125 µg i.m. or s.c.

For synchronisation of oestrus, two injections should be given 9 to 11 days apart.

Lighting regimes

Lighting extends the breeding season into the spring, with synchronisation of oestrus. Does respond to a shortening daylength by ovulation and oestrus. Keeping goats under an artificially long daylight regime during the winter months, followed by a sudden change to normal daylength in the spring, enables out-of-season breeding to be achieved from April to June during the normal anoestrus period.

From 1 January, 20 hours of artificial light are given daily for 60 days. After 60 days, the goats are returned to normal lighting. Oestrus occurs 7 to 10 weeks later. The oestrus period may be shorter than normal (often only 8 to 10 hours compared to the normal 24 to 96 hours) and the signs of oestrus are not very obvious, so best results are obtained if the males are run with the females. Sudden introduction of the male to the females after their return to normal daylength increases the percentage of successful matings.

The lights must be of sufficient number and intensity to simulate daylight and suppress melatonin – 200 lux is provided by one 36 watt fluorescent light per 18 m^2 of floor space with lights at a height of about 2.75 m. The males should undergo light treatment at the same time as the females. With this regime, kidding rates of 50 to 60% can be expected, although up to 90% has been obtained.

Theoretically, it is not necessary to provide continuous lighting for the extended long day period. In sheep, a long day effect can be obtained by providing one extra hour of light during the night, provided it is given 7 to 8 hours before dawn. However, equivocal results have been obtained with goats, so the practical application of short light pulses remains to be established. One regime is to provide additional light between 6 a.m. and 9 a.m. (fixed dawn) and between 10 p.m. and midnight.

Progestagen-impregnated intravaginal sponges

Progestagen-impregnated intravaginal sponges enable:

- Oestrus synchronisation within the breeding season.
- Extension of the breeding season.
- Out-of-season breeding.

Two types of impregnated sponge are currently available in the UK:

Medroxyprogesterone, 60 mg (Veramix Sheep Sponge, Zoetis) and Flugestone acetate, 20 mg (Chronogest, MSD)

Applicators are available for insertion of the sponges, but because the goat's vagina is more delicate than that of sheep, it is generally better to insert the sponges manually with a gloved hand using a small amount of antiseptic cream. If the animals are handled gently, the incidence of vaginitis or vaginal adhesions is low, but oxytetracycline powder may be used at insertion.

The principal of progestagen synchronisation is that:

- Gonadotrophin releasing hormone (GnRH) and gonadotrophin release is inhibited because of the negative feedback of the progestagen on the hypothalamus/pituitary.
- When the progestagen is suddenly withdrawn, there is a rebound effect with the sudden release of GnRH, and thus gonadotrophin, provided that there is no residual luteal function, which is itself secreting progesterone.
- Thus the progestagen must be administered for at least 12 days, but preferably longer (up to 20 days),

or used in conjunction with prostaglandins given 48 hours before sponge removal.

Improved pregnancy rates are generally obtained with shorter periods of progestagen treatment, particularly in large goats, as the hormonal levels in the sponges are designed for sheep. Progestagens are used for 9–12 days with prostaglandin treatment 48 hours before removal of the sponges (see Table 1.10). Removal of the sponges promotes oestrus in 95 to 100% of animals in the transitionary and normal breeding periods, but in only about 70% of animals outside these periods.

Two days before sponge removal, or at sponge removal if during the breeding season, a dose of follicle stimulating hormone, that is serum gonadotrophin (PMSG), is administered s.c. or i.m. to ensure optimum ovulation. The dose depends on the size of the goat, its yield and the season when the sponges are being used (Table 1.11). If high doses of serum gonadotrophin are used, superovulation may occur, with resultant multiple births.

There are usually very strong signs of oestrus following sponging. The presence of a male improves the response. Oestrus occurs 24 to 72 hours after sponge removal (generally 30–36 hours). This is dependent upon several factors, which include:

- Social cues.
- Day length and ambient temperature.
- Whether the goat has been routinely cycling and producing regular pulsatile amounts of LH.
- Age of animal.
- Amount and type of chorionic gonadotropin or follicle stimulating hormone given before or at sponge or CIDR removal.

Table 1.10 Regimes for sponging goats.

Breeding season			
Day 0 Sponge inserted	**Day 9** Prostaglandin injection	**Day 11** Sponge removed + serum gonadotrophin injection	**Day 12–13** Onset of oestrus
Out of breeding season			
Day 0 Sponge inserted	**Day 9** Serum gonadotrophin injection	**Day 11** Sponge removed	**Day 12–13** Onset of oestrus
Transitory period			
Day 0 Sponge inserted	**Day 9** Prostaglandin injection + serum gonadotrophin injection	**Day 11** Sponge removed	**Day 12–13** Onset of oestrus

Table 1.11 Serum gonadotrophin treatments for sponged goats.

	Time of injection of serum gonadotrophin	**Dose of serum gonadotrophin (i.u.) (PMSG)**	
		Production of milk/day <3.5 kg > 3.5 kg	
Out of season (March–June)	48 hours before sponge removal	600	700
Transitory period	48 hours before sponge removal	500	600
Breeding season (September–February)	At sponge removal	400	500

Table 1.12 Optimum time for artificial insemination following sponge removal.

Fixed time (once)	Between 43 and 46 hours after sponge removal
Fixed time (twice)	30 and 50 hours after sponge removal
Fixed time (laparoscopically)	48 to 52 hours after sponge removal
After oestrus detection	12 to 24 hours after the onset of oestrus

- Whether the doe can smell a buck or is being physically teased with a vasectomized or epididymectomised buck.

Although there is some breed variation, the doe typically ovulates between 19 and 23 hours after going out of heat. The optimum times for artificial insemination using frozen semen are shown in Table 1.12. Fixed time AI results in pregnancies but the rate is reduced when compared to insemination based on oestrus detection. Sufficient males should be available if natural mating is used in a synchronised herd, for example 1 male to 10 females during the breeding season and more for out-of-season breeding.

> Conception rates during the transitory period or out-of-season will be lower than for mating within the breeding season. Fertility will improve closer to the start or end of the breeding season. Many factors can influence fertility including breed, age and weight of the doe, month of treatment, buck selection and management.

Because the sponges are not licensed for goats, there is no defined milk withhold period but synthetic progesterones like medroxyprogesterone are known to be excreted into the milk in large quantities whilst the sponge is in place and for at least 3 days afterwards.

Controlled internal drug release

Suitable progesterone-impregnated intravaginal controlled internal drug-releasing devices (CIDRs) are available in many countries, although at present not the UK, for oestrus synchronisation in goats. CIDRs, silicon rubber elasomers moulded over a nylon spine, are inserted into the vagina like a sponge and similarly release a controlled amount of progesterone into the bloodstream, placing the treated does in the luteal phase of the oestrous cycle. The level of progesterone in milk from healthy goats after CIDR insertion for 19 days is similar to or less than those endogenously produced during dioestrus or pregnancy.

CIDRs available in the United States, Australia and New Zealand (Eazi-Breed CIDR, Zoetis) contain 0.3 g of a natural progesterone and similar timing and hormone regimes to sponges are used.

During the breeding season they are inserted and left in place for 14 to 18 days. The majority of does will be in oestrus approximately 48 hours after device removal. For AI using frozen semen, it is recommended that pregnant mare serum gonadotrophin (PMSG) 200 to 400 IU is given 48 hours before device removal. The use of vasectomised bucks is recommended prior to insemination. Insemination using a laparoscopic or cervical technique should be performed within 48 hours after device removal.

During the transitional periods, at the start and end of the breeding season, PMSG injections should be given at CIDR removal to help promote ovulation.

Out of season, if the does are not cycling, only a short period of progesterone is required to initiate follicle production. CIDRs are inserted for 7–9 days, followed by PMSG when the CIDR is removed. Breeding behaviour will likely occur without the PMSG, but the does do not ovulate and therefore the conception rate is very poor.

Outside the normal breeding season, buck fertility is usually much reduced. When using sponges and CIDRs, it is important to recognise this limitation and to use an increased male to female ratio, possibly with hand mating, or use AI. Reduced numbers of pregnancies and decreased numbers of feti/doe are common at this time. Does generally only cycle once, so failure to exhibit oestrus should not be used as an indicator of pregnancy.

Good record keeping will help improve conception rates using natural service and AI:

- Season of year/doe already cycling or not.
- Amount and type of chorionic gonadotropin given.
- Time of sponge or CIDR removal.
- Time to first signs of oestrus (hours).
- Time to end of oestrus; doe will no longer stand for buck (hours).
- Natural service or AI; fresh or frozen semen.
- Time AI carried out after sponge or CIDR removal (hours).

- Number of does confirmed pregnant.
- Number of progeny produced.

Melatonin

Animals measure day length using melatonin secreted during the hours of darkness by the pineal gland. In sheep, treatment with melatonin provides a short day/long night signal that will advance the breeding season. Goats appear to need exposure to long days, provided by artificial light, before they will respond to melatonin. Twenty hours of artificial light from 1 January for 60 days followed by a return to natural light, combined with melatonin treatment by subcutaneous implant, will advance the breeding season by 2 to 3 months. Males should be light-treated under the same regime as the females and their fertility may be further increased by melatonin treatment. The males should be removed from the herd at the start of the melatonin treatment and kept apart (out of sight, sound and smell) until they are reintroduced 35 to 40 days later. Fertile oestrus will occur from 2 to 6 weeks after the introduction of the males (i.e. from late April to June), with peak mating activity occurring 3 to 4 weeks after introduction (during May) and peak kidding during November.

If light treatment is not used, it is recommended that does should not be implanted before mid-May.

Melatonin can also be used in cashmere goats to delay the shedding of fleece, so obviating the need for winter shearing when weather conditions require goats to be housed and there is increased risk of post-shearing deaths.

Melatonin, 18 mg implant, s.c. behind ear

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CHAPTER 2
Abortion

The most common cause of abortion is 'unknown'.

The cause of most cases of abortion is never determined, but it is estimated that only 50% of abortions are infective in origin. Determining the cause of a small ruminant abortion can be a somewhat subjective decision. The identification of an infectious agent, using a highly sensitive test such as the polymerase chain reaction (PCR), does not always indicate disease causation for organisms that may be normal resident microflora. Examination of the products of abortion, acid-fast stains, bacterial and viral culture and other diagnostic tests may give a better picture of the likely cause of the abortion. A list of the possible causes of abortion is provided in Table 2.1.

'Abortion outbreaks' are generally diagnosed more easily than abortion in an individual animal because diagnostic material is more readily available and because individual abortions are more often due to non-infective causes. Even when infection is responsible for the abortion, fetal infection and/or death may have occurred weeks, or even months, before the actual abortion occurs and routine diagnostic procedures may be unable to identify the cause.

Initial advice to owners

- Instruct the owner to save all the products of abortion for further examination – fetus/fetuses, fetal membranes. The diagnosis will probably depend on laboratory investigation of aborted material.
- Advise isolation of the aborted doe until a diagnosis is reached and/or uterine discharges have ceased.
- Warn of possible zoonoses, particularly during pregnancy. Care should be taken when handling aborted products – wear gloves. Any aborted material not required for laboratory examination should be burned or buried. Dogs and cats should be kept away from the aborted material.

Initial assessment

The preliminary history should consider:
- Individual or flock/herd problem.
- Possible exposure to infected animals – mixing with bought-in stock, etc.
- Homebred or bought-in does.
- Feeding, for example silage.
- Vaccination.
- Known disease status.
- Whether abortions have occurred in previous years.

Specific enquiries should cover:
- The length of gestation/timing of abortion (see Table 2.2).
- The general breeding history – return to service, etc.
- The incidence of abortion, still births, weak kids.
- The age of does that are aborting.
- Signs of illness in the aborted does.
- Drugs used on herd, for example prostaglandins.
- Access to possible poisons.
- The possibility of stress on the doe – handling, transport, etc.

Clinical examination

The doe should be fully examined for signs of disease. Aborting does may be ill and the clinical signs may aid diagnosis, but because abortion may occur some

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Table 2.1 Causes of abortion.

Luteolysis
- Trauma
- Stress

Iatrogenic
- Prostaglandins
- Corticosteroids

Poisoning
- Plant
- Wormers

Nutrition
- Starvation
- Vitamin A deficiency
- Manganese deficiency

Infection
- Enzootic abortion
- Toxoplasmosis
- Listeriosis
- Campylobacter
- Q-fever
- Leptospirosis
- Salmonellosis
- Tickborne lever
- Border disease
- Brucellosis
- Neosporosis/sarcocystosis
- Caprine herpesvirus 1
- Any bacteria responsible for bacteraemia
- Foot and mouth disease

Fetal developmental abnormalities

Table 2.2 Timing of abortion.

Abortion throughout gestation	Abortion in late gestation
Toxoplasmosis	Listeriosis
Chlamydia	Campylobacter
Leptospirosis	Q-fever
Prostaglandins	Salmonellosis
Stress	Border disease
Tickborne fever	Corticosteroids
Foot and mouth disease	Multiple fetuses
Bluetongue	Energy deficit
	Mineral deficiency

time after infection, many aborting does will show few additional clinical signs. Abortion may follow acute septicaemia and pyrexia caused by conditions not normally associated with abortion, for example enterotoxaemia. A blood sample should be taken for laboratory investigation.

The aborted fetuses and placentae should be examined grossly before submitting to the laboratory. If a number of does have aborted, samples from several animals should be submitted because of the possibility of more than one infectious agent being involved.

Laboratory investigation

Ideally, material from every abortion should be submitted for laboratory analysis but this may not be economically feasible. Clusters of abortions should always be investigated as should sporadic abortions affecting >2% of the herd.

The diagnostic laboratory should have as complete a case history as possible, including:
- Number of infected animals.
- Age of infected animals, stage of gestation at which abortion occurred.
- A description of placental and fetal lesions.
- Clinical signs in the aborting does.
- Knowledge of any intercurrent diseases on the farm.

Submission of correct specimens is essential for accurate diagnosis:
- *Placenta* including several cotyledons – fresh or fixed in formol saline.
- *Fresh fetuses* (refrigerated but not frozen).

The preferred samples are fetus *and* placenta collected into an intact polythene bag (one per doe). Mummified fetuses are of little diagnostic value. If unable to submit whole fetuses:
- Fetal lung and liver – fresh and fixed
- Fetal abomasal contents – fresh; collect aseptically using a vacutainer tube and needle
- Fetal heart blood or thoracic fluid – fresh; collect into a plain vacutainer tube using syringe/pipette
- Fetal brain – fixed.

The gross appearance of placentae and fetuses may be suggestive of a particular aetiology, but are rarely pathognomonic, and adequate samples permit the demonstration of pathogens by microscopy, direct culture or virus isolation.
- *Serum from aborted animals*. At least 10 animals or 10% of the herd in 'abortion storms'. Maternal serology is suitable for enzootic abortion and *Toxoplasma*, both of which are better diagnosed by submission of fetal and placental material as above. Paired serum samples, with the second sample 14 days after the first, are often of more use than single samples.
- *Fetal serum*. The presence of antibodies in fetal serum allows a definitive diagnosis as the uterus should remain sterile during gestation.
- *Vaginal swabs* may be of value if placentae or fetuses are not available.

Samples should be double-bagged for transport with fetuses and placenta from individual does kept separate. If being sent by post, samples should be packed according to postal regulations, preferably in rigid containers with screw-top lids and sealed in leakproof inner packaging

such as a plastic bag with sufficient absorbent material to soak up the entire contents if leakage occurs.

Infectious causes of abortion

There are important differences between sheep and goats in the behaviour of some infectious organisms that cause abortion. Because the epidemiology of these conditions is different in the two species, the control measures taken to limit the spread of disease in sheep will not necessarily be applicable to goats. More than one infective agent may be involved in an 'abortion storm'

Enzootic abortion (chlamydial abortion)

Abortion occurs at any stage of pregnancy (unlike sheep where abortion is restricted to the last 2 to 4 weeks) because of the luteolytic effect from endometrial inflammation. The incubation period is as short as 2 weeks, so infection and abortion may occur within one pregnancy, even if infection occurs late in pregnancy (see Table 2.3).

Aetiology

- *Clamydophila abortus* (formerly *Chlamydia psittaci*), an intracellular organism containing both RNA and DNA.

Transmission

- Ingestion of the organism shed in faeces but more generally from aborted material as *Chlamydophila* are not very resistant in the environment. Shedding in vaginal secretions may begin as early as 9 days before abortion and last as long as 21 days after abortion.

Table 2.3 Chlamydial abortion in goats compared to sheep.

Abortion may occur	*at any stage of pregnancy*, rather than in the 3rd trimester as in sheep
Colonization of the placenta can occur	*at any stage of pregnancy*
The	*time from infection to abortion can be as little as two weeks*; in sheep it may be many months later, mostly during the next pregnancy
The	*doe may become ill* with septicaemia and pneumonia following abortion

- Carrier does or males in endemic herds and bought-in does continue the spread of infection. Kids born live will also carry the infection, although they remain negative serologically, and may shed organisms when they kid themselves.

Clinical signs

- Often abortion is the only clinical sign and the doe rapidly recovers, but severe illness with metritis, keratoconjunctivitis and pneumonia has been reported.

Post-mortem findings

- Placenta – intercotyledonary placentitis often with a covering of yellow purulent material, giving a leathery appearance. Advanced autolysis occurs.
- Fetus – no specific gross lesions occur; the fetus may be autolysed or fresh.

Diagnosis

- Examination of smears from the placenta, the mouth or nostrils of the foetus or the vagina of the doe using modified Ziehl–Neelsen stain demonstrates red-staining elementary bodies.
- The standard serological test is the complement fixation test. Does have significant antibody titres to chlamydial antigens after abortion. Titres of at least 4/32 are considered positive and indicative of infection in the current season. Paired serum samples taken 2 to 3 weeks apart can aid diagnosis. Antibodies can also be detected in fetal serum. Vaccination titres are usually low.
- The Western blot (WB) test can be used to confirm the diagnosis but is not suitable for screening large numbers of samples.
- Isolation of the organism from placenta or fetal tissue in tissue culture or in embryonic eggs – an extremely labour-intensive technique.
- Other tests used in some laboratories include ELISA, PCR and immunofluorescence with monoclonal antibodies.

Treatment and control

- Segregate aborting animals for 2 weeks until the excretion of chlamydia has ceased.
- Dispose of aborted material and disinfect the area.
- Cull any live kids born to infected does.

- Treat all pregnant goats in the herd with tetracyclines for 10 days and move them to uncontaminated pasture halfway through treatment:

  Dairy goats: 20 mg long-acting oxytetracycline/kg i.m. every 3 days
  Fibre goats: as dairy goats, or 400–450 mg oxytetracycline/head/day orally

- Consider a vaccination programme, but only where infection is already present in the herd. Attenuated live and inactivated vaccines are licensed in the United Kingdom for use in sheep but not goats. Live vaccines must only be used in non-pregnant animals; the inactivated vaccine can be used during pregnancy. Vaccination of healthy females at least 4 weeks before should prevent infection but may not prevent abortion in goats already infected. However, there is some evidence in sheep that use of the inactivated vaccine during an outbreak may reduce the number of abortions. In sheep, vaccination will provide protection for up to three lambings post-vaccination, depending on the vaccine. Vaccination should continue indefinitely as vaccination does not eliminate infection. Inactivated vaccines do not pose a zoonotic risk and can be safely handled by women of child-bearing age.
- Fertility is usually normal in pregnancies subsequent to the abortion, but immunity may wane after about 3 years.

Public health considerations

- The organism is excreted in body fluids including milk. Pregnant women are particularly at risk from contact with aborted material and from drinking unpasteurised milk.

Toxoplasmosis

Aetiology

- *Toxoplasma gondii*, an obligate protozoan parasite of endothelial cells.

Transmission

- Infective oocysts are passed in the faeces of cats, which act as the definitive host, multiplying and disseminating infection and contaminating stored foodstuffs or pastures. An asexual phase occurs in intermediate hosts, which include mammals and birds and a reservoir of infection for susceptible cats exists in birds and wild rodents, which may pass the infection vertically between generations. Cats then amplify and spread the infection.
- Goats act as intermediate hosts, becoming infected by ingesting foodstuffs contaminated with cat faeces containing oocysts and the resulting asexual developmental stage, the tachyzoite, actively enters host cells and multiplies. An immune response is detectable after about 12 days post-infection and the infection is either cleared or bradyzoites (tissue cysts) are formed. When a pregnant goat is infected, the tachyzoites travel to the placenta, causing a placentitis.
- Direct contact with the products of abortion – does eat placentae containing bradyzoites and tachyzoites.
- Transplacental infection of kids. Very low levels of infection may result in abortion.

Clinical signs

- Pyrexia and lethargy may occur in the doe about 2 weeks after infection, but the doe will be clinically normal at the time of abortion. Occasional fatalities occur in adult goats – post-mortem findings include nephritis, cystitis, encephalitis, hepatitis, enteritis and abomasitis.
- Goats exposed to infection prior to mating will not abort.
- Resorption, fetal death and mummification occur if infection is during the first third of pregnancy.
- Abortions, stillbirths and weak kids are produced if infection occurs later in pregnancy.
- Normal but infected kids may be produced to does affected in late pregnancy. Up to 80% of females may be infected and abort.

Post-mortem findings

- Placenta – yellow or white focal lesions, 1 to 3 mm in diameter on the cotyledons, with the intercotyledonary areas not affected.
- Fetus – there are usually no specific gross lesions on the fetus.

Diagnosis

- Diagnosis is aided by the clinical picture, the presence of the characteristic small white necrotic foci in placental cotyledons, the possible presence of a mummified fetus and on fetal serology and histopathology.

- Indirect fluorescent antibody (IFA) test on fluid collected from the fetus thorax or abdomen.
- ELISA and latex agglutination tests are used to detect antibodies in fetal and maternal serum. Polymerase chain reaction (PCR) tests specific for *T. gondii* are available in some laboratories.
- Serological examination of dam (complement fixation test) may be confusing due to high serological levels in the normal population. Antibody titres can persist for years and vaccination produces similar titres. Rising titres between paired samples is indicative of infection and a low titre means that the abortion was not due to *Toxoplasma*.

  Where dams are seropositive from exposure but have not aborted because of recent *Toxoplasma* infection, tissues will be PCR negative.
- Serological examination of live kids before suckling will demonstrate high specific antibodies.
- Histological examination of fetal tissues – brain, lung, liver, heart, kidney and spleen – shows necrotic foci surrounded by inflammatory cells and a non-suppurative mengioencephalitis typical of protozoan infection.
- Immunohistochemistry (IHC) can be used on formalin-fixed tissues to demonstrate antigen in bradyzoites and tachyzoites.

Treatment and control

- Animals remain infected for life.
- Sheep that have experienced abortion do not generally abort the following year, but it has been suggested that infected goats *may* sometimes abort in subsequent pregnancies, so it *may* be advisable to cull infected does.
- Chemoprophylaxis is likely to be of limited value in goats because of the risk of abortion during subsequent pregnancies, but decoquinate, 2 mg/kg bodyweight daily, can be added as a premix to commercial rations from mid-gestation to reduce perinatal losses. Monensin sodium, 15 mg/kg bodyweight daily, is also effective but banned from use in many countries, including the United Kingdom.
- Infection can be prevented by stopping the access of cats to grain stores, feeding troughs and hay barns.
- Products of abortion should be destroyed as soon as possible.
- Oocysts may persist on pasture or in soil for over a year and are very resistant to most disinfectants.
- Cats acquire immunity to reinfection and do not subsequently present a threat to livestock unless they become immunosuppressed. Preventing cats breeding on the premises will prevent the spread of the disease via the intermediate host – younger cats generally pose the greatest threat.
- Rodent and bird control will reduce the reservoir of infection that exists for susceptible cats.
- Vaccination. A vaccine containing living tachyzoites of *Toxoplasma gondii* is licensed in the UK for use in sheep but not goats; a single injection 3 to 4 weeks prior to mating gives a minimum of two seasons' protection. Vaccination will not introduce infection into the herd.

  The vaccine against toxoplasmosis can be given at the same time as the live vaccine against *Chlamydophila abortus* produced by the same manufacturer or at the same time as the inactivated vaccine against *C. abortus*. The vaccines should be given on opposite sides of the neck using different equipment.

Public health considerations

- Toxoplasma tachyzoites are passed in the milk of infected docs, so there is a risk to children and pregnant women from drinking infected milk as well as from handling aborted material. Aborted material should never be handled with bare hands.

Listeriosis

See Chapter 11.

Although two species *Listeria monocytogenes* and *Listeria ivanovii* are associated with abortion in sheep, only *L. monocytogenes* causes abortion in goats.

- Abortion results from infection early in gestation; abortion occurs from the 12th week but generally in late pregnancy.
- Infection later in gestation results in still births; some kids are born alive but die soon afterwards.
- Retained placentae and metritis are common after abortion.

Post-mortem findings

- Placenta – placentitis with cotyledons and intercotyledonary areas affected.

- Fetus – necrotic grey-yellow foci 1 to 2 mm in diameter in the liver and sometimes the lung.

Diagnosis

- The organism is easily grown and identified in the laboratory as a Gram-positive beta-haemolytic bacillus and can be isolated from the fetal stomach, uterine discharges, milk and the placenta.
- Fluorescent antibody examination of aborted material.

Treatment and control

- Clinically ill animals can be treated with ampicillin or potentiated sulphonamide.
- Aborted does are considered immune and should be retained in the herd.

Campylobacter (vibriosis)

Aetiology

- Comma-shaped Gram-negative bacteria. *Campylobacter* fetus (formerly *Vibrio* fetus). *Campylobacter jejuni* is a significant cause of abortion in the United States.

Transmission

- Ingestion of organisms from aborted material.
- Some does remain permanent carriers and are thought to be the main source of novel infection for herds.
- Wildlife can act as vectors.

Clinical signs

- Abortion in the last 4 to 6 weeks of gestation; some infected goats do not abort but produce weak kids at full term. These kids usually die within a few days.
- Does may be pyrexic and lethargic with diarrhoea for a few days either side of the abortion, but often the goat appears clinically normal.
- A post-abortion mucopurulent vaginal discharge is usual.
- Males are infected but do not show clinical signs.

Post-mortem findings

- Placenta – changes minimal.
- Fetus – may have doughnut-shaped foci of 10 to 20 mm diameter in the liver; the fetus is usually autolysed.

Diagnosis

- Direct Gram smears of fetal abomasal contents and placenta. The organism appears as curved, Gram-negative rods.
- Culture of fetal abomasal contents under microaerophilic conditions on selective media.
- Immunohistochemistry (IHC) can be used on formalin-fixed tissues to demonstrate *C. fetus* antigens.

Treatment and control

- Segregate aborting animals immediately and then cull them because of the possibility of a carrier state.
- Cull any live kids born.
- Dispose of abortion material and contaminated material by burning or burying.
- Treat the remainder of the herd with penicillin/dihydrostreptomycin or long-acting tetracycline 20 mg/kg every 48 hours during the outbreak or tetracycline 200–300 mg /head/day in feed before and during the kidding season. Tetracycline resistance appears quite common in *Campylobacter jejuni* isolated from sheep in parts of the United States.
- Vaccination. Killed, adjuvanted vaccines are available in North America and New Zealand, but are not available in the United Kingdom.

Public health considerations

- *Campylobacter* causes acute gastroenteritis in humans, usually by faecal contamination from diarrhoeic or apparently healthy goats. Faecal contamination of raw goats milk produced human disease in an outbreak in the United Kingdom.

Q-fever

Q-fever occurs in domesticated and wild animals throughout the world and is an important zoonosis. Clinical signs of disease are very uncommon, but abortions can occur in cattle, sheep and goats. The disease is considered endemic in livestock in Australia and, although it is almost never diagnosed as a clinical problem in animals, people working closely with animals are routinely vaccinated. An outbreak in the Netherlands in 2007–2009 was linked to abortions in dairy goat herds and thousands of goats were culled as a public health measure.

Aetiology

- Very small intracellular parasite *Coxiella burnetii*, a member of the Rickettsia family.

Transmission

- *C. burnetii* is highly infectious. Only one organism is required to produce infection under experimental conditions.
- By ingestion after direct contamination with abortion material or the urine, faeces and milk of an infected animal. Cattle, sheep, goats and wildlife may be infected and infection can be spread between species.
- By inhalation or through injured skin after contact with the organism.
- By infected ticks (unconfirmed in the United Kingdom).
- Healthy carrier goats spread the infection. Goats and cows shed the organism in milk and vaginal secretions for months or years, whereas sheep are more likely to shed in faeces. The organism is persistent in the environment and can infect any type of animal – cats, dogs, rabbits, insects – so a farm will stay contaminated.
- The organism can survive for weeks in the environment, surviving best in dry and dusty conditions, where it can attach to dust particles. It is extremely stable and highly contagious in the dried state, remaining dormant for 50+ years under the right conditions. One organism is said to be able to infect a human by the aerosol route.

Clinical signs

- Most infected goats are healthy carriers, but some aborting animals will be clinically ill with a retained placenta.
- Abortion occurs in the last month of pregnancy and stillborn or weak infected kids may be born at term.
- Further abortions have been shown to occur at a further two subsequent pregnancies.

Post-mortem findings

- Placenta – placentitis with clay-coloured cotyledons and intercotyledonary thickening; indistinguishable from that seen with *Chlamydophila abortus* abortion.
- Fetus – autolysed with no specific lesions.

Diagnosis

- Smears from the placenta or fetal stomach contents stained with a modified Ziehl–Neelsen stain show acid-fast pleomorphic coccobacilli.
- Immunohistochemistry on formalin-fixed sections of the placenta can be used to demonstrate *C. burnetii* antigens. Conventional histopathology and IHC are considered to have good specificity but poor sensitivity.
- A complement fixation test on the dam's serum shows the antibody level rising about a week after infections and persisting for a month. Titres of >1/10 are considered positive.
- ELISA and indirect fluorescent antibody (IgG phase 1 and 2 antigens) tests are available in some laboratories; both the specificity and sensitivity of serology are considered inferior to IFA.
- Realtime polymerase chain reaction (PCR) tests specific for *C. burnetii* are useful if tissues are available. PCR can utilise a variety of tissues – placenta, fetal lung, meconium, liver and abomasal content and maternal uterus, vaginal mucosa (tissue or swab) and whole blood.
- Culture is not routinely used for diagnosis due to the zoonotic risk.

Treatment and control

- Tetracyclines will probably control an outbreak and reduce abortions, but do not prevent shedding or reduce the zoonotic risk.

  Oxytetracycline, 20 mg/kg, 2 injections, the first at around 105 days' pregnancy and the second 2 weeks later

- A inactivated vaccine is available in Europe (Coxevac, CEVA) and could be used in the United Kingdom in the event of a disease outbreak. The effectiveness of the vaccine in goats has been determined in two field studies with pregnant goats exposed to *Coxiella burnettii*. The studies conducted in cattle and goats showed that Coxevac reduces bacteria shedding (which is a major factor in spreading the disease) in vaginal discharge and milk, whilst in goats Coxevac reduced bacteria shedding in faeces and the placenta as well. The studies in goats also showed a lower pro-

portion of abortions in the goats that were vaccinated compared with unvaccinated goats. The duration of protection was established to be 280 days in cattle and one year in goats. In goats it is very common to see a palpable reaction of 3 to 4 cm diameter at the injection site, which may last for 6 days. The reaction reduces and disappears without need for treatment. In goats, it is also very common to observe a slight increase of rectal temperature for 4 days post-vaccination without other general signs. The withdrawal period for Coxevac for meat and milk is zero days.

Public health considerations

- The disease is most important as a human infection. Infection usually results from inhaling the resistant spore form on dust particles contaminated with fetal, placental and uterine fluids, faeces or urine, but also by drinking infected milk or eating unpasteurised dairy products (the organism migrates into the cream, survives freezing and likes acidity). The organism is killed by pasteurisation. Animal hides, wool and fur are potential sources of infection for abattoir workers. Infection can also occur via tick bites or skin abrasions.
- Outbreaks can occur in urban areas when there is windborne spread from nearby livestock premises.
- Disease often occurs in people without signs of disease in infected animals. Herd immunity tends to be excellent after an outbreak so abortions stop but shedding still continues.
- When working with an infected herd, good personal hygiene is essential, particularly during kidding. Hands should be washed frequently and disposable overalls should be worn or clothing changed and washed before leaving the premises. Personal protective equipment, including facemasks and goggles should be worn for high risk activities such as handling abortion materials, pressure washing or working in very dusty livestock areas.
- Products of kidding. Placentas, aborted fetuses, contaminated bedding, etc., should be removed immediately and disposed of by incineration, burial or autoclaving. Contaminated bedding should be wetted down to prevent the generation of aerosols.
- Manure should be composted well and not spread as it is more contagious in the dry state.
- It is very difficult to eliminate organisms from the environment, even with the removal of organic material and disinfection with quaternary ammonium products.
- Infected animals are safe to eat if thoroughly cooked but the slaughter of positive animals may pose a risk of infection to abattoir workers.
- Vaccination in routinely carried out in some countries for groups of workers potentially at risk from infection. A Q-fever vaccine is available in Australia and is 83 to 100% effective in preventing the disease. However, the vaccine can only be given to individuals 15 years of age and over. Prior to immunisation, a blood and a skin test is recommended to see if the individual has previously been exposed to Q-fever – either naturally or by previous vaccination. Vaccinating those already exposed to Q-fever can result in severe reactions. Vaccination will not prevent disease in someone who has already been infected but is in the incubation period of the disease.

Leptospirosis

See Chapter 17.

Leptospirosis is a rare cause of abortion in goats. Abortions are reported to occur only following septicaemia in acute infections.

Diagnosis

- Serology: microscopic agglutination test (MAT); ELISA.
- Isolation and identification of *Leptospira* spp. in the doe's urine, placenta, or fetal fluids and kidney tissues.

Salmonellosis

Aetiology

- Numerous *Salmonella* serotypes have been reported to cause abortion in sheep and goats, including *S. abortus ovis, S. typhimurium, S. dublin* and *S. montevideo*. Sources of infection include birds, cattle and

human sewage, but the interval between infection and abortion means that identifying the source of infection may be difficult.

Clinical signs

- Some serotypes, including *S. typhimurium* and *S. dublin*, cause systemic illness with diarrhoea in addition to abortion, stillbirths or very weak live kids, which die shortly afterwards. The fetuses are autolytic and oedematous with lesions characteristic of septicaemia.
- *S. abortus ovis* causes sporadic abortions in middle to late gestation. Abortion is also the main presenting sign of *S. montevideo* infection.
- Abortion material is heavily contaminated with salmonellae and acts as a source of infection for other animals.
- Metritis and retained fetal membranes are common following abortion and does may die from septicaemia.

Diagnosis

- Diagnosis is established by bacterial isolation from the abomasum and other fetal organs or from vaginal swabs. Serotyping is carried out at a *Salmonella* reference laboratory.

Treatment and control

- Antibiotic treatment of sick and in-contact pregnant does should be based on the antibiotic sensitivity of bacterial isolates.
- An inactivated *S. dublin* and *S. typhimurium* vaccine is licensed for cattle in the United Kingdom and could be used off-licence for goats in the face of an outbreak of salmonellosis.

Tickborne fever

Anaplasma phagocytophilum (formerly *Ehrlichia phagocytophilia*) causes tickborne fever in sheep, cattle and goats in the United Kingdom and can cause severe economic losses in some locations due to reduced weight gain and milk yields, abortions and secondary infections. *Anaplasma phagocytophilum* is also responsible for human granulocytic anaplasmosis, a severe febrile illness increasing in incidence in the United States and Europe.

Aetiology

- *Anaplasma phagocytophilum* is an intracellular parasite of the Rickettsia family, transmitted by the tick vector *Ixodes ricinus*.
- *A. phagocytophilum* in feral goats in the United Kingdom has been reported from Scotland and Northern Ireland, where it may be a significant wildlife reservoir.
- Very little work has been done regarding the prevalence of *Anaplasma* infections or their clinical significance in sheep and goats in North America. *A. phagocytophilum* is present in the northeast of the United States, together with an appropriate vector, *Ixodes scapularis*. In the west, *Ixodes pacificus,* the western black-legged tick, is a suitable vector. It is not known whether or not clinical illness, similar to tickborne fever in the United Kingdom, is caused by North American strains of *Anaplasma*.

Epidemiology

- In endemic areas most nymphs and adult ticks are infected and thus virtually all ruminants and deer exposed to ticks also become infected, generally very early in life, and so do not encounter infection for the first time when pregnant. Exposure of pregnant does to infection results in abortion. Feral goats may potentially be a significant wildlife reservoir of the disease.

Clinical signs

- Pyrexia, lethargy, weight loss (often not recognised).
- Abortion and metritis.
- Temporary infertility in male goats.

In addition, infection may increase the pathogenicity of other diseases such as tick pyaemia, louping ill, listeriosis and pasteurellosis.

Anglo-Nubian goats react more severely than other breeds of British goats.

Diagnosis

- Detection of the organism in polymorphonuclear leucocytes in Giemsa-stained blood smears.
- Examination of the brains of aborted fetuses reveals eosinophilic multifocal leucomalacia and glial nuclear loss.
- Demonstration of rising antibody titres in paired serum samples using ELISA.

- PCR to detect the parasite in heparinised blood samples from infected does.

Control

- Susceptible pregnant animals should not be exposed to tick-infected pastures.
- *A. phagocytophilum* is sensitive to tetracyclines.

Border disease (hypomyelinogenesis congenita, hairy shaker disease)

Border disease virus (BDV) causes abortion, stillbirth and infertility in small ruminants and probably has a worldwide distribution.

Aetiology

- Pestivirus, serologically related to bovine viral diarrhoea virus (BVDV-1 and BVDV-2).
- BDV has been segregated into several phylogenetic groups, BDV-1 to BDV-7, and further genotypes may be characterised in the future.
- Pestiviruses have been isolated from only a few naturally infected goats and field cases are rarely reported.

Epidemiology

- Persistently infected cattle and sheep are believed to be the main reservoirs of pestiviruses (Border disease and BVD) that infect goats. Bovine virus diarrhoea virus (BVDV) is easily spread between cattle and goats during cohabitation and the virus can spread transplacentally to the caprine fetus.
- Direct animal contact by ingestion or aerosol is the main source of infection; sheep, goats, deer and possibly pigs are potential sources of infection.
- Surviving infected kids will remain carriers.
- Although the oronasal route is probably the most common means of natural dissemination, spread is possible via the use of common needles or ear-tagging or castrating equipment previously used on a persistently infected animal, even of another species.

Clinical signs

- Most clinical cases involve abortion and poor viability of newborn kids.
- Infection during early pregnancy can result in fetal death and resorption with return to service after an extended period.
- Abortion, stillbirths, barren does, fetal death, maceration and mummification result from infection after about the 70th day of gestation.
- Small weak kids occur with varying degrees of tremor and skeletal abnormalities, although hairy coat abnormalities as seen in lambs rarely, if ever, occur. Kids with cerebellar hypoplasia may be unable to stand or present with seizures or opisthotonus.
- When goats are experimentally or naturally infected, persistently infected offspring occur less frequently than in sheep. Immunological competence in goat fetuses seems to occur 20 days later than it does in sheep, corresponding to about 80 to 100 days of gestation. Because infection of goats before the time of immune competence usually kills the fetus, the prevalence of persistent infection in goats is very low.
- Infection in non-pregnant goats is often subclinical.
- One Austrian goat that was naturally infected with BVDV-1 showed poor body condition, stiff gait and difficulty in rising, and subsequently died of fibrinopurulent pneumonia.
- BDV in goat herds with diarrhoea has been reported from eastern China.

Post-mortem findings

- Kids – affected kids show hydrocephalus and cerebellar hypoplasia.
- Placenta – severe necrotising caruncular placentitis.

Diagnosis

- Live animal. Virus isolation and serology from kid or doe blood by ELISA and PCR. Experimental infections of mature goats produce neutralizing antibodies that are detectable for at least 4 years.
- Post-mortem. Antigen can be detected in fresh samples of thyroid, kidney, brain, spleen, intestine, lymph nodes or placenta; virus can be isolated from post-mortem material sent in virus transport medium; heart blood can be used for serology; histopathology of brain and spinal cord shows hypomyelinogenesis and immunohistochemical labelling can be used.

Control

- Infections with pestiviruses in goats do not appear to be a problem and do not usually require any control, but the small potential to be a reservoir of infection for cattle and sheep needs to be considered when planning eradication programmes.

Brucellosis

Brucella abortus

Goats can be infected with *Brucella abortus* and hence are included in dairy cow testing programmes, but the incidence of infection is minute in all parts of the world. In infected goats abortion is rare and mastitis common. Serological testing of goats for brucellosis is reported to have severe limitations.

Brucella melitensis

Goats and sheep are most susceptible to *Brucella melitensis*, which occurs mainly in southern Europe and Middle Eastern countries. It is absent from the United Kingdom, but sporadic outbreaks have been reported in the United States. The disease is important as a major zoonosis producing Malta fever in humans who drink infected milk. It is contagious to cattle and less commonly affects pigs and horses.

Brucella melitensis is a notifiable disease in the United Kingdom. Suspect *Brucella melitensis* when there are late abortions (2 months), stillbirths, weak neonates, mastitis, orchitis, lameness and hygroma.

The disease is most likely to be introduced by the importation of infected animals, semen or embryos. The European Union requires sheep and goats imported on to an officially free holding to originate from a free holding and be subject to testing in isolation before the movement. Contaminated milk and milk products are a zoonotic risk. Pasteurization kills the bacteria.

Epidemiology

- Goats excrete the organism in milk, urine and faeces and in vaginal discharges for 2 to 3 months.
- Kids carried to term become carriers and can shed the organism.
- Infection enters the adult animal through the nasopharynx, conjunctivae or upper alimentary tract by direct or aerosol contamination following abortion, when very large numbers of organisms are excreted. Venereal infection occasionally occurs.
- Brucellosis is usually introduced into the herd by the purchase of an infected animal or by sharing common grazing.

Clinical signs

- Abortion is the main clinical sign and usually occurs during the last months of gestation. The organism is excreted in uterine and vaginal secretions, urine, faeces and milk after parturition or abortion.
- Abortion occurs particularly in the first pregnancy and subsequent pregnancies may proceed to term with the birth of live but weak kids.
- Systemic disease with pyrexia, lethargy, weight loss, diarrhoea, mastitis, metritis, fall in milk yield and lameness may occur.
- Males may develop orchitis.

Post-mortem findings

- The placenta is normal on gross examination.

Diagnosis

- Isolation of the organism from a culture of the placenta or fetal stomach.
- Serological tests include ELISA, SAT, CFT and Rose Bengal tests, but cross-reactions with other bacteria are common, leading to false positives.

Vaccination

- A live vaccine (Rev 1) is considered to be highly effective in small ruminants and is used globally where the disease is endemic. It is generally used in kids 3–6 months of age, although it can be used in adults. Vaccination of pregnant animals results in abortion. The recommended strategy is the mass vaccination of all animals in the first year, followed by the annual vaccination of all newborn animals.
- Vaccination programmes are being used to control brucellosis in areas where the disease is endemic and have been shown to be cost-effective, but complete eradication of the disease is difficult and, without adequate disease surveillance, the disease can rapidly spread from small residual foci.
- It is not possible to distinguish between bacterial titres due to natural infection and vaccination. Rev 1 titres persist for a long time following vaccination.

Public health implications

- Milk and milk products, such as unpasteurised cheese, are potential sources of human infection.

Neosporosis

Neospora is a major cause of fetal loss in cattle that parallels that of *T. gondii* infection in sheep and goats.

While neosporosis is likely to be an uncommon cause of abortion in goats, experimental infection is readily induced in them. Furthermore, clinical signs and pathological lesions in sheep and goats are similar to those induced in them by *T. gondii*, although there are subtle histopathological differences. The incidence of the organism in goats in the United Kingdom is unknown.

Aetiology

- *Neospora caninum*, a protozoan parasite with a life cycle similar to *Toxoplasma gondii*. Dogs and wild canids act as definitive hosts, where sexual reproduction occurs.
- Cattle are the major intermediate hosts, where asexual reproduction occurs; sheep, goats, horses, deer, dogs and foxes are also occasionally affected.

Transmission

- Sporocysts are passed in dog faeces and are later ingested by the goat.
- Infection need not occur during pregnancy to cause abortion; vertical (transplacental) transmission from the dam to the fetus occurs and can occur repeatedly in the same animal.

Clinical signs

- Abortion, stillbirth and weak kids. Dams generally show no other clinical signs.

Diagnosis

- Serological examination by immunofluorescent antibody test (IFAT) and ELISA.
- Histological examination of brain and heart – characteristic non-suppurative Encephalitis.
- Immunohistochemistry on formalin-fixed tissues to demonstrate antigen.
- Parasites found in tissues by specific staining (immunoperoxidase (IPX) test).

Treatment

- No drugs have been shown to prevent transplacental transmission.

Control

- There are currently no effective control measures against the parasite. However, as it is now known that dogs can shed *Neospora* oocysts, it is important that dogs be kept away from feed and water and should be prevented from eating aborted foetuses, placental tissue or dead young, which may harbour infection.

Sarcocystosis

Aetiology

- *Sarcocystis capracanis*, a protozoan parasite using the goat as an intermediate host, with the dog as the definitive host.
- Other species-specific prey–predator life cycles have been demonstrated for goat–dog (*S. hircicanis*) and goat–cat (*S. moulei*). Goats are the intermediate host for *S. orientalis*, but are unaffected (dogs are probably the final host).
- *Sarcocystis* relies on the predator–prey relationship of animals. Oocysts are passed through the faeces of an infected animal and undergo sporogony. Lysis of the oocysts releases sporocysts into the environment, where they are ingested by an intermediate host such as a goat. Sporozoites are then released in the new host and migrate to muscle tissue where they undergo asexual reproduction. Once the intermediate host is eaten by the definitive host, such as a dog, the parasite undergoes sexual reproduction to create macrogamonts and microgamonts. They create a zygote, which develops into an oocyst that is passed through the faeces, completing the life cycle.
- The incidence of the organism in the United Kingdom is unknown. It is likely that sarcocystiosis is underdiagnosed as a problem and that better diagnostic methods are needed to show the true extent of the losses caused.

Clinical signs

- *Sarcocystis* spp. infections are quite prevalent in farm animals, following ingestion of food or water contaminated with sporocysts; however, clinical sarcocystiosis is much less commonly diagnosed than toxoplasmosis and neither is it normally associated with fetal infection or abortion in either sheep or goats. Most animals are asymptomatic, and the parasite is only discovered at slaughter.
- Clinical signs depend on the number of sporocysts ingested. They are non-specific and include pyrexia, anorexia, lethargy, haemolytic anaemia, jaundice,

nervous signs and abortion due to maternal failure – the placenta and fetus are not infected; weak kids may be born alive.

Diagnosis

- Diagnosis is difficult as infection is so common and clinical signs are absent, mild or non-specific. Serology may be useful in some situations and histopathology/immunohistochemistry is valuable for confirming the cause of death.

Post-mortem findings

- Non-specific findings; petechial haemorrhages in many organs.
- Haemorrhages in heart and skeletal muscle.
- Immunofluorescent and immunoperoxidase techniques are used to identify meronts in endothelial cells including the maternal caruncle.

Control

- Livestock become infected by sporocysts from the faeces of carnivores. Because most adult cattle, sheep and many pigs harbour cysts in their muscles, dogs and other carnivores should not be allowed to eat raw meat, offal or dead animals. Supplies of grain and feed should be kept covered; dogs and cats should not be allowed in buildings used to store feed or house animals.

Public health considerations

- Human infection is rare but can happen when undercooked meat is ingested.

Other organisms

Any bacterial infection that results in bacteraemia can occasionally cause abortion, for example *Mannheimia haemolytica* (formerly *Pasteurella haemolytica*) and *Trueperella pyogenes* (formerly *Corynebacterium* pyogenes).

Other organisms that have been implicated in abortion are as follows.

Bacillus spp.

Bacillus species occur ubiquitously and are normal components of cut grass. They proliferate in poor quality silage on exposure to oxygen. Abortion outbreaks in cattle fed on big bale silage have been attributed to *B. licheniformis* and *B. licheniformis* is occasionally implicated in caprine abortions.

Yersinia pseudotuberculosis

Yersinia pseudotuberculosis infection in goats has been associated with:

- Enteritis and/or typhlocolitis.
- Systemic abscessation.
- Conjunctivitis.
- Hepatitis.
- Mastitis.
- Placentitis, abortion, perinatal mortality, endometritis, infertility.

Late- and full-term abortions and early neonatal deaths have been reported following enteric infection and subsequent bacteraemia. Reported lesions were necrosuppurative placentitis with intralesional bacterial colonies, and bacterial embolisation with necrosuppurative fetal pneumonia, hepatitis and, less frequently, splenitis and nephritis.

Mycoplasma spp.

M. mycoides and *M. agalactiae* are reported to cause vulvovagintis and abortion, with abortion occurring during the first third of pregnancy. Aborting females shed the organism in milk and amniotic fluid and the organism can be found in the placenta and the liver and spleen of the fetus.

Caprine herpes virus

Caprine herpesvirus 1 (CpHV-1) is responsible for lethal systemic infections in one to two week old kids and for subclinical infections of the respiratory and genital tract in adults. CpHV-1 may also cause infertility, abortions, returns to service and genital lesions (see Chapter 1).

The virus has been linked to sporadic abortion storms following introduction of the virus to herds by latent carriers. Virus isolation, fluorescein-conjugated antibody staining and histological examination of fetuses can be used to identify herpes virus abortion, but serology is of limited use because positive results indicate exposure, which may not be recent. Serology may help

identify goats that could become latent carriers of the virus.

Kids born alive may remain unaffected by the virus and are thus at low risk of being infected with the virus, although kids may have maternal antibodies for several months after birth.

Bluetongue virus

Goats are generally less severely affected by the bluetongue virus than sheep (Chapter 10) and the virus is an unlikely cause of abortion in goats. Infection of sheep during pregnancy can cause fetal death and resorption, abortion or live 'dummy' lambs with developmental defects including hydranencephaly (absence of cerebral hemispheres).

Orthobunyaviruses

See Chapter 8.

Orthobunyaviruses, including Akabane virus (Australia, Japan, Israel, South Africa, the Middle East), Cache Valley virus (United States, Canada, Mexico) and Schmallenberg virus (Northern Europe, including the United Kingdom), can cause an increased incidence of abortions and congenital malformations classified as arthrogryposis hydranencephaly syndrome (AHS) in fetuses and neonatal kids. These include stillbirth, premature birth, mummified fetuses, arthrogryposis, joint malformations, scoliosis, torticollis, kyphosis, lordosis, hydrocephalus, hydranencephaly, microcephaly and cerebellar dysplasia, behavioural abnormalities and blindness. Adult goats are otherwise generally asymptomatic so pregnant animals may remain healthy but abort or deliver mummified fetuses and stillborn or deformed kids. An apparently normal kid can be born twin to a mummified kid or one with severe deformities. Pre-colostral titres in the kid's serum or fetal fluids confirm the diagnosis.

Foot and mouth disease

Abortion is less likely than in sheep but can occur as a result of pyrexia (Chapter 6).

Rift Valley fever (RVF)

RVF is a vector-borne disease of sheep and goats characterised by high rates of abortion and neonatal mortality. Cattle and wild mammals are also susceptible and it is a major zoonosis. Susceptibility of different breeds to RVF varies considerably. Morbidity and mortality can approach 100% in naïve adults and young animals.

Suspect RVF when pregnant goats abort and kids under 1 week die suddenly. Other animals may be jaundiced with haemorrhagic diarrhoea.

The disease could be imported with infected mosquitos, via wild life introduced as exotic pets, illegal imports or bush meat, although the disease is unlikely to become established in the United Kingdom under present climatic conditions. Imports of live sheep and goats into the European Union from affected countries are banned.

Present distribution

Endemic in sub-Saharan Africa with occasional incursions into northern Africa and the Middle East, including Saudi Arabia, and recently South Africa.

Aetiology

- Single-stranded RNA virus of the family Bunyaviridae within the genus *Phlebovirus*, spread by various mosquitos. Transovarian transmission is an important epidemiological feature and infected eggs can survive in soil for years before being released during rainy seasons. In endemic areas RVF virus regularly circulates between wild ruminants and haematophagous mosquitoes; disease is usually inapparent.

Clinical signs

Very short incubation period, as short as 12 hours in kids and 2 days in adults. In an endemic area, infections in non-pregnant animals may be subclinical.

Neonatal kids:

- Weakness, lethargy, anorexia
- Abdominal pain
- Rapid, abdominal respiration
- Death in 24–36 hours

Older kids and adults:

- Sudden death
- Pyrexia, inappetance, lethargy
- Jaundice in some animals
- Abortion storms with rates approaching 100%
- Agalactia
- Vomiting
- Mucopurulent nasal discharge
- Dysentery
- Haematuria
- Death

Post-mortem findings

- Jaundice
- Focal hepatic necrosis
- Multiple haemorrhages
- Intrathoracic and intraabdominal oedema

Diagnosis

- Virus isolation or demonstration of the viral genome, PCR.
- Serology – virus neutralisation, ELISA, haemagglutination inhibition.
- Histopathology – characteristic cytopathology of the liver of affected animals and immunostaining allows the specific identification of the RVF viral antigen in infected cells.

Wesselsbron disease

Wesselsbron disease and Rift Valley fever share many clinical and pathological features. However, Wesselsbron disease is usually milder, producing much lower mortality, fewer abortions and less destructive liver lesions. It is seen mainly in sheep, but goats, cattle, pigs and horses can also be infected. Newborn lambs and goat kids are most susceptible. In lambs and kids, the clinical signs include pyrexia, anorexia, weakness and an increased respiratory rate and mortality may occur in 30% of affected lambs. Infections in adult goats are usually inapparent and limited to pyrexia, but pregnant goats may abort.

Wesselsbron disease is caused by the Wesselsbron virus, an arthropod-borne virus in the genus *Flavivirus* of the family Flaviviridae. It has been isolated from vertebrates and arthropods from many sub-Saharan African countries, and serologic surveys provide evidence of its occurrence in other countries.The infection rate appears to be high in ruminants in endemic regions of Africa. In cattle, sheep and goats, the seroprevalence may be as high as 50%. Animals of all ages seem to be susceptible to infection.

Non-infectious causes

Medication

Prostaglandin administration will produce abortion at any stage of the gestation. *Phenothiazine* used as a worm drench is reported to cause abortion in late pregnancy as will *corticosteroids* given in late pregnancy. *Levamisole*, administered in the last two months of pregnancy, has been implicated in late-term abortions.

Trauma and stress

> As the doe is dependent on corpus-luteum-derived progesterone throughout pregnancy, anything that causes luteolysis will produce abortion.

This means that goats may be more susceptible than other species to abortion from trauma or stress. Any condition that causes release of prostaglandins, and thus luteolysis, will cause abortion.

Heat stress can cause fetal hypoxia, hypotension and endogenous corticosteroid release with subsequent abortion.

Developmental abnormalities

Very few deformed fetuses are produced, but a fetus with developmental abnormalities may be aborted. An hereditary defect of Angora does in South Africa led to chronic hyperadrenocorticism, death of the fetus and then its expulsion.

Multiple fetuses

Abortions or early parturition appear more common in Anglo-Nubian goats, which commonly carry three, four or more fetuses. In these cases, placental insufficiency probably leads to fetal expulsion.

Poisons

Plant poisonings are occasionally implicated in abortion, for example *Astragalus* spp. and *Lathyrus* spp., but are unlikely to be of significance in the United Kingdom. Toxic plants can also produce fetal abnormalities (see Chapter 21).

Poisonings from non-plant sources also occasionally occur, generally causing abortion by producing a systemic illness.

Malnutrition

Energy deficit. Frank starvation will cause abortion, particularly if the energy input is insufficient during the last third of pregnancy.

Mineral deficiencies. Mineral deficiencies, for example, selenium, copper and iodine, generally cause the