Chapter 6

Macroscopic Pathology of the Reproductive Tract of Domestic Ruminants

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Detailed examination of the genitalia is often overlooked in routine necropsy procedures; the ovaries or testes normally receive little attention in comparison with "vital" organs such as liver, kidney or brain. This is no doubt because changes in these vital organs are more likely to tell why an animal sickened and died – the usual reason for necropsy being performed. Thus even experienced pathologists may not be certain of what is normal, and may have difficulty in assessing pathological changes in genitalia.

It also needs to be noted that changes associated with aging tend to be more pronounced in the genitalia than in other organs, and in the female, knowledge of morphological changes associated with pregnancy and the ovarian cycle is essential before interpretation of pathological change can be attempted.

NECROPSY TECHNIQUE

Before dissection commences it is of course important to conduct an external examination, which in so far the reproductive organs are involved, might reveal such disorders as hermaphroditism, vulvitis in the female, or cryptorchidism in the male.

Removal of Genitalia

Satisfactory examination of the pelvic organs can be achieved only after removal of a portion of the bony pelvis.

Most necropsy procedures involve placing the animal on its side, disarticulation of the uppermost hip joint, reflection of both legs and the skin on one side, removal of the abdominal and thoracic walls, and examination *in situ*. Then follows removal of the thoracic viscera, and all abdominal viscera other than the genito-urinary organs.

After completion of these steps, removal of part of the bony pelvis can be achieved by cutting (with saw or garden pruning shears) along the symphysis pubis and through the shaft of the ilium on the uppermost side. A knife is then used to cut adjacent fibrous and muscular tissue, and a complete segment of the bony pelvis is removed.

Before cutting along the symphysis pubis in the male, it is necessary to carefully dissect free the fibrous attachments of the root of the penis to the ischium. If care is taken, the penis can then be freed from its ischial attachments and dissected proximally to include the prepuce.

Because of their intimate association, it is frequently desirable to examine and remove the entire genital and urinary systems, including the kidneys, simultaneously – and more or less intact. This is best accomplished by dissecting the kidneys and ureters free by cutting attachments on their *lateral* aspects. In the female, in order to achieve complete removal of the ovaries and related structures, the ovarian ligaments should also be cut at their lateral extremities, as close to the body wall as possible. Likewise in the male, the urogenital organs can be removed by cutting the vas deferens near the internal inguinal rings.

In small ruminants it is possible to hold the kidneys, ovaries or accessory sex glands and attached ductal structures in one hand and use the other hand to dissect free the remaining intra-pelvic structures. Inclusion of a generous amount of contiguous adipose and fibrous tissue with the urogenital organs will ensure complete removal. These organs are lifted through the opening made on the ventro-lateral aspect of the pelvis. Removal is then completed by incising the skin around the anus and vulva or prepuce, in the female or male, respectively. A small portion of rectum is removed with the genitalia, so tying this off to avoid faecal contamination, may sometimes be advisable.

Although veterinarians are encouraged to include genital examination as part of their routine necropsy procedure, for the assessment of reproductive disorders in farm animals, genitalia are frequently collected after slaughter at an abattoir, rather than at necropsy. Important in this regard is realisation that the genital organs *per se* are of little commercial value so can normally be obtained gratis. In abattoirs it is customary for the genitalia to be removed from the carcass along with other viscera. Prior discussion with the slaughterman will result in more suitable specimens; little extra effort is required to achieve removal of the entire genital tract.

When it is decided to collect male genitalia after slaughter in an abattoir, it is convenient to obtain the accessory sex glands, urinary bladder and urethra collectively, by dissecting these organs free from the rectum after the animal has been eviscerated. Care should be taken not to miss the small bulbourethral (Cowper's) glands, which (in the bull) are located posteriorly, associated with the bulbocavernosus muscle; they are more conspicuous in the ram and buck. The remainder of the genitalia can then be readily obtained by taking the root of the penis plus the scrotum and its contents and dissecting these free by cutting forward to the anterior aspect of the prepuce.

Gross Examination of Genitalia

Female

The urinary organs should first be examined, and the occurrence of conditions that might involve both the genital and urinary systems (eg congenital malformations, endometritis-pyelonephritis) should be noted.

The *ovaries* should next be exposed, and particular attention should be paid to whether any cysts that may be present are located in the ovary or in the paraovarian tissues. Adhesions between these tissues or between genital and extra-genital organs should also be noted.

Ovarian size, weight, surface characteristics, colour and internal appearance after sagittal midline sectioning can all be used to ascertain the presence of congenital defects (eg ovarian hypoplasia), ovarian activity or inactivity, inflammation, intra-ovarian cysts and neoplasia.

Patency of the *oviducts* can then be checked by carefully inserting a blunted hypodermic needle (with syringe containing saline attached) in the ovarian extremity of the oviduct. Insertion of the needle into the minute opening (the abdominal ostium) is facilitated by stretching the delicate ovarian and oviduct membranes over one forefinger, with the funnel-shaped dilation of the oviduct lumen (infundibulum) uppermost. Gentle pressure on the syringe will force saline into the oviduct, which becomes visibly dilated as the saline progresses downward. Small incisions into the tips of the uterine horns permit examination of the entry of saline from the oviducts. Size of the needle will depend on the species; an 18 - SWG needle is suitable for the cow. For fixation the oviduct is dissected away from the mesosalpinx, wrapped around a small piece of cardboard and placed in Bouin's fixative (saturated aqueous picric acid 75 ml, formalin 25 ml and acetic acid glacial 5 ml.).

The remaining genitalia should initially be palpated and examined externally for the presence of congenital defects such as segmental aplasia (in which a distinct segment is absent) or incomplete fusion (especially caudally) of the tubular organs. Using blunt pointed Mayo scissors, both uterine horns, the uterine body, cervix, vagina and vulva are then opened on the dorsal aspect. Patency of the cervix should be recorded.

Attention should first be directed toward the uterine contents, particularly any foetal or embryonic remnants that may be present. Such remnants may easily be overlooked if present in a large volume of exudate.

The colour, viscosity, volume and odour of any intra-uterine exudate should be noted. The underlying endometrium is then examined for the presence of inflammation and/or hyperplastic and cystic changes.

In the cervix, vagina and vulva, the colour, viscosity and volume of mucus should again be noted and a search made to detect vesicles,

pustules, erosions or ulcerations of epithelium, and neoplasms. Other abnormalities that may be observed after opening the genitalia in this location include cystic Gartner's ducts (cattle), imperforate hymen, fusion defects as previously indicated and incomplete separation of the vagina and rectum, resulting in a form of cloaca.

Male

Dissection of the male genitalia involves three areas: the external genitalia, the testes and epididymides, and the accessory sex glands. Examination of the *external genitalia* should commence with the scrotal skin. Lesions here that may impair fertility include dermatitis and varicose dilation of scrotal veins.

The skin of the prepuce, especially the preputial orifice, should be examined, and the penis should then be extruded to permit examination. Conditions to be noted in this location include preputial eversion or prolapse, persistent penile frenulum, congenital short penis, duplicated penis (diphallia), hypospadias, phimosis or paraphimosis, inflammation, traumatic lesions, and neoplasms such as papillomatosis.

In the bull the sigmoid flexure should be palpated to detect the presence of haematomas, and the retractor penis muscle should be examined in its mid-portion for evidence of degeneration and calcification.

When indicated, the penis should be opened by cutting along the urethra; predilection sites for urinary obstruction are the sigmoid flexure, and the vermiform penile appendage in the ram.

On removal of the *testes* and *epididymides* from the scrotum, the attached spermatic cords should be examined, and the presence and type of any tunic adhesions (fibrinous or fibrous) should be noted. After checking the completeness and continuity of the epididymides and attached vas deferens (to detect segmental aplasia), the epididymides are then separated from the testes; it is advisable to weigh and measure these organs.

The testes are next dissected by a longitudinal (mid-sagittal) cut to expose the mediastinum, and the testicular parenchyma then examined to detect the presence of calcified foci (occasional small foci are of no significance), fibrosis, orchitis and neoplasms.

Testicular "halves" are then cut transversely with a sharp knife, making slices no more than 1 cm in thickness.

The epididymides are examined for the presence of cysts, and the size, location and content of these should be recorded. Sperm granulomas and larger foci of epididymitis will be detected at this time, especially in the ram. Occlusion and dilation of more proximal epidydimal ducts result from these conditions. On gross examination it is to be remembered that

accumulated sperm resemble purulent exudate and that microscopic examination of a smear of the exudate will quickly clarify its composition.

The *accessory sex glands* (asg) comprise the seminal vesicles, prostate, bulbourethral (Cowper's) glands and the ampullae (dilations of the proximal extremities of the vas deferens). Among the domestic species there is considerable variability in the morphology and pathological involvement of these glands. In domestic ruminants the seminal vesicles are most often affected.

After examination of the urinary bladder, a cut in the neck of the bladder on its dorsal aspect is extended along the urethra to the root of the penis.

The asg should be examined for the presence of congenital defects and should be palpated to detect cysts, foci of inflammation, fibrosis, hyperplasia or neoplasia. Any adhesions to adjacent structures should be noted.

After collection of specimens for microbiological examination, all glands are opened by multiple incisions. The type of exudate present in each gland should be recorded.

It is often convenient to collect samples of spermatozoa at this time. In the bull, spermatozoa for morphological examination can usually be obtained by incising the ampullae with a sharp scalpel and aspirating the contents into a Pasteur pipette. This sample can be washed into saline or formal saline or used to prepare fresh smears. As compared with the clinical collection of semen, spermatozoa obtained at necropsy in this way can be identified with either the right or the left gonad; such identification may be helpful in evaluating other findings in the testis or epididymis on a particular side.

Specimen collection and processing

Female

Samples obtained at necropsy for microbiological study are best acquired before actual dissection of the genitalia is commenced.

The surface of the uterus is seared with a heated spatula, and a sterile Pasteur pipette or disposable sterile needle and syringe used to aspirate any contained exudate. A drop of the aspirate may be placed on a glass slide, coverslipped and examined directly (eg for trichomoniasis in cattle), or smears may be prepared for fixation, Gram-staining and bacteriological identification.

Exercising care to avoid or minimise contamination, the remaining aspirate should then be transferred to a sterile screw-topped container, and dispatched for culture and other laboratory examinations (eg dark-ground study).

Specimens for histopathological study should be placed in fixative as soon as possible. Bouin's fluid [is] preferable to formalin for fixation of genital tissues. Tissues to be fixed should be cut to a thickness of less than 1 cm, and the ratio of tissue to fixative should be about 1 to 20. These tissues must be removed from the fixative after 24 to 48 hours and stored in 10 per cent formalin.

Male

For microbiological sampling of the preputial cavity, the penis is held with rat-tooth forceps and drawn from the prepuce. The moist preputial and penile epithelia are then gently scraped with a sterile scalpel blade. Preputial contents so obtained may be placed directly into tissue culture medium for virological studies (eg to detect bovine herpesvirus 1 infection (IBR) in cattle), incubated in special media for the detection of trichomoniasis (in cattle) or used for bacterial (eg *Campylobacter* sp.) isolation. Samples for microbiological examination from lesions in the remaining genitalia are collected using sterile procedures as previously described for the uterus.

For satisfactory histopathological evaluation of the testis and epididymis, fixation within a short time after collection is necessary. As for the female genitalia, Bouin's fluid is again the preferred fixative. It is best to fix thin transverse slices of testicular tissue (previously discussed) taken from three locations - the dorsal pole in the area of attachment of the epididymal head, the central region and near the ventral pole. If a sharp knife is used, it is possible to cut slices less than 1 cm that include both the tunica albuginea laterally and the mediastinum centrally. After the epididymis has been grossly examined and cut by multiple sections, it is best fixed *in toto*. Representative portions of the remaining genitalia and tissue from any lesions that maybe present are fixed as indicated.

GENITAL LESIONS IN CATTLE

Essentially because genital pathology has been most studied and is best documented in cattle, lesions in cattle are considered first and in most detail; sheep and goats are then considered on a comparative basis.

Female genitalia

Ovary

Rare anomalies of the ovary include agenesis, and vascular hamartomas – which increase in size with time, and resemble neoplasms.

Ovarian hypoplasia is an important condition in cattle and may occur in a herd at the same time as testicular hypoplasia in bulls. In one breed (Swedish Highland cattle), ovarian hypoplasia was shown to be a genetic

defect conditioned by an autosomal recessive gene. In other populations prevalence rates are in the order of 1-2% although prevalences of 7 to 19% were recorded in particular beef herds in northern Queensland. The condition is usually bilateral but varies in severity and symmetry. Differentiation of less severe "partial" hypoplastic ovaries from ovaries in anoestric cattle is important. Whereas hypoplastic ovaries have characteristic grooves running longitudinally on the surface, anoestric ovaries are smooth, rounder and larger. Also in ovarian hypoplasia there is accompanying hypoplasia of remaining genitalia. Severe generalised hypoplasia of the genitalia is a feature of freemartins (genetic females born co-twins to males), many of which have seminal vesicles.

Haemorrhage in the ovary may be intra-follicular and of unknown cause in calves or in atretic follicles in cows. Extensive haemorrhage, of up to several litres and even progressing to death, may be the result of manual enucleation of corpora lutea – especially in cows that are pregnant or have pyometra.

Cysts in and around the bovine ovary are important but are of varied origin and significance.

Paraovarian cysts are located adjacent to the ovary and arise from either mesonephric or paramesonephric tubules or ducts. They may be up to several centimetres in diameter. Histological examination is sometimes necessary to distinguish these from true ovarian cysts.

Cystic rete tubules arise from the rete ovarii, a structure of mesonephric origin that is composed of groups of anastomosing tubules in the medulla of the ovary.

True ovarian cysts may be defined clinically, as follicular structures of at least 2.5 cm in diameter that persist for at least 10 days in the absence of a corpus luteum. The incidence on ovarian cysts in dairy cattle is reported to be from 6 to 19%. Therefore cystic ovarian disease is a serious cause of reproductive failure even though 40 to 80 % of cows with ovarian cysts re-establish ovarian cycles following treatment with products high in luteinising hormone (LH) activity. Ovarian cysts may be classified as follicular cysts or luteal cysts. Probably the pathogenesis of both follicular and luteal cysts is similar – ie failure of hypophyseal release of adequate LH.

Follicular cysts (cystic Graafian follicles) are more common than luteal cysts. They may be single or multiple on one or both ovaries. They are larger and have thicker walls than normal follicles and are under more tension. Luteal cysts are usually solitary on one ovary and are generally thicker-walled than follicular cysts. Their yellow-orange luteal mass is smooth and rounded and a layer of fibrous tissue lines their central cavity.

Note that both the follicular and luteal cysts lack an ovulatory papilla and this latter feature can be used to distinguish luteal cysts from cystic corpora lutea in which the ovulation papilla distorts the outline of the cyst. Cystic corpora lutea can only be considered pathogenic if they do not produce adequate progesterone for normal cyclic events.

If ovarian cysts persist, changes develop in the remaining genitalia. These include cystic endometrial hyperplasia (see below), the accumulation of mucus (mucometra), cystic distension of Gartner's ducts (vestigial remnants of male ducts in the vaginal wall) and Bartholin's glands, and oedema of the vagina and vulva.

Neoplasms of the ovary may arise from surface coelomic epithelium, gonadal stroma, or the germ cells. Review of 104 bovine ovarian tumours revealed that 66 were granuloma cell or other stromal tumours. Therefore most ovarian tumours in cattle are of gonadal stromal type; they are unilateral and there is a tendency for the tumour to occur in the daughters of affected dams. Also, removal of the affected ovary may result in development of a tumour in the remaining ovary. The most common associated clinical sign is nymphomania, but anoestrus or normal cycling may occur.

Gonadal stromal tumours may be quite large (eg 15 – 20 cm in diameter), are smooth externally, and on section are composed of cystic and solid areas, which are white to yellow depending their lipid content. These tumours are rarely malignant.

Epithelial tumours constituted 17 of the 104 tumours in the above study and included cystadenomas, which when small, might be confused with cystic ovaries. Germ cell tumours including dysgerminoma and teratoma are rare in cattle but are recorded.

Ovaritis (oophoritis), with lesions of sufficient severity to be recognised grossly are rare in cattle but abscessation may be a consequence of enucleation of the corpus luteum in cows with pyometra. Necrotising ovaritis has been observed in heifers infected 11 – 15 days previously with the virus of infectious bovine rhinotracheitis (IBR); the occurrence of such lesions in spontaneous infection does not appear to be reported.

Uterine (Fallopian) tubes

Hydrosalpinx (distension of the uterine tube by fluid) is not infrequent in cattle, and (depending on case history) caution may be needed in ascertaining whether or not such lesions resulted from speying, "webbing" (oviduct removal), or other manipulation of the ovary or oviduct. Hydrosalpinx may also be secondary to salpingitis. In hydrosalpinx the uterine tube is distended up to a diameter of 1.5 cm with clear watery fluid – which can be seen through the thin stretched wall.

Macroscopic evidence of inflammation of the uterine tubes (**salpingitis**), or their distension with pus (**pyosalpinx**) is rare in cattle, the latter being most likely to accompany similar changes in the uterus.

Uterus, Cervix, Vagina and Vulva

Excluding intersexuality (see freemartins, above)-, in which enormous variation in the extent of genital development may be seen, there are two important congenital defects of the so-called tubular genitalia

(including uterine tubes) of the cow; these are segmental aplasia, and anomalies of fusion.

In **segmental aplasia of the paramesonephric (Mullerian) ducts** a distinct segment is absent so there is an accumulation and inspissation of genital secretion from above this point. On rectal examination this secretion, forming a mass of 10 or more cm in diameter, may be interpreted as a neoplasm or perhaps a retained foetus. Segmental aplasia may take various forms ranging from rudimentary development of the cranial vagina, cervix or uterus to degrees of hypoplasia or aplasia of a uterine horn. Where only one horn is involved the cow can conceive in the opposite horn. Imperforate hymen is one of the simplest forms of aplasia. In general, the more serious defects of the paramesonephric duct differentiation involve its anterior part, and the less serious are caudal.

Necropsy examination in these cases will reveal the firm doughy intra-luminal accumulations of brown mucoid material and the absence of pus and foetal remnants.

Segmental aplasia is especially common in white Shorthorn cattle ('white heifer disease') but probably occurs occasionally in all breeds.

Anomalies of fusion usually affect the cervix where, once again, a number of structural defects can occur. These include such variations as double uterine body and cervices (uterus didelphys) or double cervices in a single body. Such abnormalities may cause little trouble with fertility as such but may give rise to problems at parturition.

Endometrial hyperplasia with or without cysts is a sign of excessive stimulation by endogenous (cystic ovaries – see above) or exogenous (plant) oestrogens. Macroscopically, cysts may reach 5 cm in diameter and be associated with an excessive amount of mucoid fluid in the uterus. Cases complicated by endometritis become more purulent.

Although not as important as in ewes, endometrial hyperplasia has been observed in cows grazing oestrogenic (subterranean) clover and has been related to infertility.

Most uterine inflammations are confined to the mucosa or endometrium (**endometritis**) and it is important to note that there are no gross lesions in mild forms of endometritis such as those caused by post coital infection with pyogenic organisms of low pathogenicity, *Campylobacter fetus* or *Tritrichomas foetus.* Low-grade catarrhal endometritis with minor epithelial damage has been associated with a variety of bacteria such as pyogenic cocci, *Escherichia coli* and some *Corynebacterium* spp.

Severe endometritis is most likely to follow calving and veterinarians in bovine practice will readily anticipate the macroscopic changes. The uterus is flabby and enlarged by much chocolate-coloured lochia that is slightly tenacious. The content progressively becomes a dirty greyish

yellow because of a mixing of inflammatory exudate and placental detritus. Removal of this exudate reveals that the underlying endometrium is congested, swollen and mottled as a result of foetal haemorrhage and tattered intercotyledonary areas. In cases where foetal maceration has occurred, fragments of bone may be present in the exudate. Suppurative, ulcerative endometritis associated with Bovine Hepesvirus-4 in post-parturient dairy cows has recently been described as a herd problem. In these cases ulcerated endometrial epithelium was replaced by fibrino-necrotic suppurative "mats" and there was pyometra from which *Arcanobacterium pyogenes, Escherichia coli,* and rarely *Clostridium perfringens* and *Streptococcus* sp., were isolated.

Although uncommon, spread of inflammation from the endometrium to deeper layers (**metritis, uterine abscess**) may occur and be seen as "paintbrush" haemorrhages beneath the serosa of the un-dissected uterus. Partial resolution of such lesions may result in the formation of well-encapsulated abscesses 1 to 3 cm in diameter. Larger abscesses (eg up to 15 cm in diameter), especially in the dorsal uterine wall, are more likely to be post surgical.

Pyometra (uterus filled with pus) may result from acute suppurative nephritis or persistent low-grade infections with organisms such as *T. foetus*. Other organisms involved are haemolytic streptococci, coliforms, *Arcanobacterium pyogenes*, and *Pseudomonas aeruginosa.*

In the cow pyometra is also associated with corpus luteal activity in the ovary, but in contrast to the bitch it is the uterine disease that causes the corpus luteum to persist.

In bovine pyometra the amount of pus varies from a few ml to several litres. The cervix has no seal so a small amount of pus escapes into the vagina. Pyosalpinx and perimetritis may co-exist.

Inflammation of the vagina (**vaginitis**) and vulva (**vulvitis**) are not uncommon in cattle. Numerous pale small papular eruptions on the vulva (but not vagina) are seen in non-pregnant animals and the condition is referred to as granular venereal disease. Microscopically the papules are seen to be lymphoid accumulations . This change, which also occurs on the penis and prepuce of the bull, is in response to mild irritation and is not specific for any particular infectious agent.

Larger and more discrete ulcers (2 – 3 mm diameter) of both vulval and vaginal epithelium are characteristic of infectious pustular vulvo-vaginitis; these lesions are transient and again resemble those seen in the bull (see below) in response to infection with the same herpesvirus. This herpesvirus (or a subtype of it) also causes infectious bovine rhinotracheitis (IBR) but as a rule nasal and vaginal infections behave epidemiologically as distinct diseases.

With the exception of vulval fibropapilloma, **neoplasms of the tubular genitalia** of cattle are quite rare. Smooth muscle tumours (leiomyomas)

are seen occasionally as discrete and very firm round swellings up to 10 or more cm in diameter. They are pale, and bulge on cutting.

Endometrial carcinomas are sometimes seen in old (usually dairy) cows and differ from leiomyomas in that tumour growth is quite infiltrative and sclerosing – causing irregular nodular thickening of the uterine wall. Metastases in the regional (medial iliac) lymph nodes are likely.

The tubular genitalia of cattle are probably a more common site for lymphosarcoma than is the case in other domestic species; both diffuse and nodular infiltration occurs.

Studies in the USA have revealed that **retrovaginal constriction** in Jersey Cattle, a condition, which would more accurately be referred to as anovestibular stenosis, is a congenital defect regulated by an autosomal recessive gene. The condition does not directly involve Mullerian duct structures. Non-elastic fibrous bands of tissue at the ano-rectal junction and within the vestibular muscularis are seen in affected cows, which are prone to dystocia.

Male genitalia

Pathological examination of culled bulls is a particularly valuable and cost-effective method of investigating infertility problems in extensively run beef herds, where effective examination of live bulls is extremely difficult, if not impossible.

External genitalia:

The external genitalia comprise the penis, prepuce and scrotum. These organs can suffer pathological conditions that may be congenital (present at birth) or acquired.

Malformations:

Normally epithelial separation and rupture of the frenulum occurs at puberty. Failure to rupture results in temporary or permanent adherence. When present, **persistent penile frenulum** is composed of loose collagenous tissue, some elastic fibres, small blood vessels and epithelium.

Although persistent frenulum is a "trifling anatomical abnormality" – readily corrected by surgery, it has been suggested that the defect is heritable. **Frenular remnants**, observed as flaps and tags of tissue along the raphes of adult bulls, represent lesser degrees of the same defect but are of no pathological significance.

Other malformations which may occur, include: **short penis**, possibly associated with shortening of the retractor penis muscle; **abnormal insertion of the retractor penis muscle**, with stretching of the skin anterior to the testis during erection; **duplication of the penis** (total or partial); **hypospadias**, where the urethra opens under the penis or on the perineum; and so-called "**blood fistula**" in which haemorrhage occurs during erection, from a congenital vascular defect at the penile

tip. Deviation of the penis, an important condition considered to have a congenital or hereditary basis is essentially a clinical entity, and is difficult to study at necropsy.

Preputial eversion and prolapse:

The term **eversion** is used to describe a condition in which the preputial epithelium protrudes temporarily from the preputial orifice. It has been demonstrated that polled but not horned bulls evert their prepuces, and that this may be due to the well developed retractor muscles of the prepuce in horned bulls, as opposed to polled bulls.

While eversion per se is not associated with an increased incidence of preputial abscess, it is suggested that exposure of the preputial lining during eversion may predispose to damage by trauma, myiasis, frostbite and irreversible preputial prolapse.

It is generally agreed that **preputial prolapse** occurs as a result of eversion with subsequent injury, inflammation (posthitis) and oedema which prevents retraction of the prepuce. Bulls with a long and pendulous sheath are especially affected but polled *Bos taurus* beef bulls are also susceptible. Prolapse may be associated with fracture of the penis. Other associated lesions are fibrosis, gangrenous inflammation of the sheath and infection of the prolapsed tissue with staphylococcus, streptococcus, *A. pyogenes* and *E. coli.*

Phimosis and paraphimosis:

Inability to protrude the penis (**phimosis**) may be congenital but is mostly acquired. It may follow severe inflammation of the penis and the prepuce, haematoma, neoplasia or bruising, necrosis and fibrosis of the prepuce with prolapse.

Paraphimosis (inability to withdraw the penis into the prepuce) results in strangulation of the protruded portion of the penis; neoplasms, penile haematomas, and withdrawal of preputial hairs into the prepuce have been incriminated in the cause.

Penile haematoma:

Rupture of the tough fibrous wall of the penis with resultant haemorrhage and haematoma formation, usually occurs as a longitudinal tear on the dorsal surface of the penis at a point just anterior to the scrotum and adjacent to the attachment of the retractor penis muscle. Rupture at this point is not related to the thickness of the wall of the penis but rather to the fact that greatest tension occurs in this location. In one study, however, it was found that rupture in the penile wall was not necessarily involved; haematomas in 9 of 15 bulls examined apparently resulted from injury to blood vessels outside the wall. Abscesses may form in old haematomas but in uncomplicated cases natural healing occurs with varying degrees of residual scarring restricting movement of the penis.

Balanitis and posthitis:

These inflammations of the penile and preputial epithelium usually occur simultaneously, so-called balanoposthitis.

A muco-purulent preputial discharge is the characteristic clinical picture of balanoposthitis due to infectious pustular vulvovaginitis (IPV). Because lesions of IPV are widespread and involve more of the penis than the tip or "glans", the term "penoposthitis" has been suggested as a better description than balanoposthitis. In the acute stage 2 – 3 days post-infection (PI) numerous 2 mm grey-white opaque pustules are present; these pustules may form confluent and flat lesions , on the borders of which, separate pustules are to be seen.

The pustules, which exist for 1 –2 days only, then become indistinct, their surface sloughs and ulcers or erosions remain – especially in the area of the glans. Healing commences 5 – 6 days PI following sloughing. The basic lesion of IPV in bulls is focal necrosis of the lining of the penis and prepuce.

In addition to this typical form, there occurs in repeated IPV infections, a diffuse balanoposthitis with little suppuration and without the formation of typical pustules; pustular lesions are fact rarely seen in chronically infected bulls.

Infections of the penis and prepuce with *C. fetus* and *T. foetus* produce non-specific gross alterations. In the absence of any characteristic lesions the diagnosis of both vibriosis and trichomoniasis must depend on laboratory examination of preputial washings (or scrapings) collected from the live animal or from the genitalia after slaughter.

Tuberculous balanoposthitis is characterised by the presence of typical tubercles. Lesions are seen (1 – 2 mm), brown red nodules in the epithelium and subcutis, forming rounded projections on the surface of the glans.

Other organisms incriminated in the cause of balanoposthitis include mycoplasmas and various fungi, but there are no specific gross lesions.

An ulcerative posthitis of unknown aetiology has been observed in range bulls in South America and Australia; lesions were mostly at the preputial orifice and up to 2.5 cm in diameter. In the South American outbreaks, the lesions were sometimes more extensive, and *Corynebacterium renale* was often isolated. Lesions seem to occur especially in animals on a high plane of nutrition, and pathogenesis may be similar to the condition in sheep – in which however, wethers rather than rams are mostly affected.

Neoplasms:

Fibropapillomas (or papillomas) occur singly or multiply on the glans penis. They are most common in young bulls (1 – 2 years) and may not be noticed until after service, when haemorrhage occurs. Mostly they are up to 3 or 4 in number and up to several cm in diameter. Tumours are pink or grey-white, and on section they are composed primarily of fibrous tissue with an epithelial covering of variable thickness.

The malignant epithelial tumour of the penis and prepuce is the squamous cell carcinoma. Though common in other species (stallions) this tumour is extremely rare in the genitalia of male ruminants.

Miscellaneous conditions:

These include strangulation of the penis due to the drawing in of preputial hairs, extensive necrosis and calcification of the retractor penis muscle in old bulls, fracture of the penis, epidermoid cysts of the prepuce, pediculosis (lice infestation) of the prepuce and scrotum, varicose dilation of scrotal veins and frostbite.

Testis and Epididymis

Post mortem alterations can occur relatively quickly in the testis and epididymis; they are apparent after a few hours at room temperature and after 7 – 8 hours in the refrigerator. After 21 hours at room temperature or after 30 hours refrigeration, material is no longer suitable for assessment, especially in regard to differentiation of mild testicular hypoplasia from degeneration.

Testicular hypoplasia:

Testicular hypoplasia was recognised as a genetically determined defect in Swedish Highland cattle (see ovarian hypoplasia - above). In other countries it occurs sporadically at a low incidence. A high occurrence of "small testes", has however been observed in *Bos indicus* bulls but caution is needed in such cases to differentiate true hypoplasia from delayed testicular maturation in these bulls. Hypoplasia, by definition, means "defective or incomplete development". In the bull, one or both testes may be abnormally small, and in the absence of satisfactory history (eg previous clinical examination) it is difficult, even histologically, to distinguish between a hypoplastic testis and one which is small as a result of degeneration. Testes in cryptorchidism, especially those retained in the abdomen, are severely hypoplastic.

Hypoplasia is usually unilateral, generally affecting the left testis. The affected testis may be as small as ½ - 1/3 normal size and it is freely movable in the scrotum. Consistency of the hypoplastic testis varies; probably it usually approaches the normal more than with degeneration where a small hard testis ultimately results from progressive fibrosis. Hypoplastic testis do bulge on cutting.

Other Malformations:

With the exception of congenital cysts, other malformations are rare and include aplasia of the epididymis and or vas deferens (see segmental aplasia – accessory sex glands) and supernumerary testes.

Congenital retention cysts of the epididymis are not uncommon. They are regarded as blind remnants of mesonephric tubules and although they usually cause no harm, sperm accumulation within cysts (spermatocele) with subsequent extravasation and tuberculoid reaction, may occur however, and compromise fertility.

Testicular degeneration (atrophy):

As with most tissues, the testis can respond in only a limited number of ways to a wide variety of insults. It has been shown that the primary spermatocyte is the seminiferous cell most susceptible to insult of varying

type, while the B-spermatogonium and the Sertoli cells are the most resistant.

Degeneration may occur locally or involve the whole testis. Although the damaged testis may at first swell as a result of oedema, it soon becomes smaller than normal. While the organ is initially soft and flabby (does not bulge on cutting), there is subsequent hardening due to fibrosis, often associated with calcification. The relative size of the epididymis is sometimes useful in differentiating testicular hypoplasia from degeneration. This is because atrophy of the testis exceeds that of the epididymis. Therefore a high epididymal–testis ratio is likely to indicate atrophy rather than hypoplasia.

Causes of testicular degeneration are many and varied; they include localised or systemic diseases, after lameness with decubitis, trauma, after mange, poisoning with molybdenum, arsenic or chlornaphthalene, malnutrition or avitaminosis A, hormonal disturbances, high atmospheric temperatures, neoplasia, vascular lesions, obstructive lesions of the epididymis, inguinal hernia, auto-immunity, obesity and intensive overfeeding. In one recent report, testicular degeneration in young bulls was due to impaired thermoregulation of the testes associated with scrotal oedema that occurred as a result of infection with *Eperythrozoon* sp. infection.

Degenerative changes, fortunately, are usually reversible, and regeneration is possible so long as the spermatogonia and Sertoli cells remain.

Orchitis:

Orchitis (inflammation of the testis) is an uncommon lesion in the bull. Mostly it arises by spread via the bloodstream but extension of inflammation from neighbouring organs (eg testicular tunics), or via the genito-urinary tract is possible. The picture and progress of orchitis obviously varies from case to case; a useful classification however is that of intra-tubular, interstitial, and necrotising orchitis.

Macroscopically in intra-tubular orchitis numerous sub-miliary to miliary, eventually confluent, white to yellow foci are seen on cutting the testis.

In interstitial orchitis, the macroscopic appearance is predominantly one of increased scar tissue and the affected testis is harder and smaller than normal.

Necrotising orchitis is characteristic of brucellosis but may result from other infections. The affected testis undergoes complete or partial necrosis. On sectioning, necrotic areas are dry, yellow, often laminated and only slightly calcified.

In association with the above forms of orchitis we may find abscessation, periorchitis (inflammation of the adjacent tissues), and fistulation through the scrotum.

It is difficult and frequently impossible to relate observed pathological changes to specific causal agents or even to the type of agent. This is especially so in chronic orchitis. To complicate the picture still further the so-called "tuberculoid reaction" to spermatozoa which escape from the

duct system (see below) confounds even the diagnosis of specific infections.

Viral orchitis in the bull appears to be uncommon although further work is needed to clarify. IPV virus has been isolated from the testis but its appearance there is probably transitory.

Bacterial orchitis due to Diplo-, Strepto- or Staphylococcus, *A. pyogenes, Mycobacterium tuberculosis* and *Actinomyces bovis* as well as *Brucella abortus* have been described. Other infectious causes of orchitis are *Nocardia farcinica*, Mycoplasmas and Chlamydia.

Non-infectious causes of orchitis include trauma, and allergic responses to spermatozoa (so-called immune orchitis) and possibly other allergens.

Epididymitis:

Epididymitis occurs more frequently than orchitis but may be associated with it or with inflammation of the accessory sex glands. In contrast to orchitis, epididymitis is considered to arise chiefly by spread of infection in the genito-urinary passages.

Macroscopic findings in epididymitis vary; in acute cases there is enlargement with oedema. Abscessation may follow. One has to be careful not to confuse accumulated spermatozoa with pus, since they resemble each other in consistency and colour. Fibrosis, periorchitis with extensive adhesions, and inflammation of the spermatic cord are common sequels to epididymitis. In the bull the causal organisms of epididymitis are those listed for orchitis; additionally Pseudomonas or *E. coli* may be involved.

Tuberculoid reaction (sperm granuloma):

Fatty substances similar to those found in the tubercle bacillus, are contained in spermatozoa. The sperm head especially is resistant to disintegration and its presence in extra-ductal locations in the testis or epididymis elicits a pronounced chronic inflammatory or "granulomatous" reaction. Such granulomas may be indistinguishable from tuberculous lesions.

Neoplasms of the testis:

Primary testicular neoplasms of the bovine testis are rare and are not associated with obvious hormonal changes. Interstitial (Leydig) cell tumours are regarded as the most frequent but we have observed more Sertoli cell tumours. Teratoma (mixed congenital tumour) in the testis of a calf, has been described.

Miscellaneous conditions of the testis and epididymis:

These include partial black pigmentation (melanosis) of the epididymis, spontaneous torsion of the testis in the process of normal descent, thrombosis of testicular arteries, and paradidymis – a structure in the anterior part of the spermatic cord composed of isolated tubules which did not develop normally.

Accessory Sex Glands (asg)

Segmental aplasia (partial absence) of the mesonephric duct: The mesonephric (Wolffian) duct in the male foetus is responsible for the ultimate development of the epididymis, vas deferens, ampulla and seminal vesicle. These structures secrete fluids that are necessary for the storage and transport of sperm. Aplasia or hypoplasia of the mesonephric duct may affect all or any of the structures derived from it. The defect mostly involves the right side, and is rarely bilateral.

Varying degrees of fusion of mesonephric duct derivatives may be associated with segmental aplasia, but in mature bulls caution is needed in distinguishing congenital fusion from old inflammatory lesions., Also, cysts may result from fusion of the mesonephric ducts; in these cases soft fluctuating masses up to ~7 cm in diameter may be found just anterior to the body of the prostate gland.

When segmental aplasia is unilateral, or when only the seminal vesicles are involved, the bull is probably fertile. However, as there is strong evidence that the condition is heritable, affected animals should not be used for breeding purposes.

Other malformations:

These include cysts, aplasia, hypoplasia and fusion of the bulbourethral glands, prostatic "appendage", and melanosis in various locations.

Cysts and concretions:

The lumen of the seminal vesicles may be altered to form cystic dilations of varying size. Cysts may be congenital or acquired and may be generalised or in individual lobules. Similar cysts are observed in the ampullae, bulbourethral glands and prostate.

Concretions in the seminal vesicles may fill the described cysts. They are usually friable, rough externally and may have a laminated appearance on sectioning. They are composed of organic components, spermatozoa, phosphates and carbonates, and are attributed to precipitation of retained secretions or to chronic inflammation.

Seminal vesiculitis:

Of the asg, the seminal vesicles appear to be most commonly involved. Probably they are more often infected than even the testis or epididymis.

Seminal vesiculitis may be classified as being chronic interstitial or predominantly degenerative. In the former type there is a marked increase in size of the gland, excess fibrosis and loss of lobulation; cystic cavities lined by a whitish smooth membrane are frequent. In the predominantly degenerative type, increase in gland size is slight or absent, and increase in consistency is minimal. Complications of seminal vesiculitis include pronounced abscessation with involvement of surrounding tissues, adhesions, and fistula formation into the rectum or urinary bladder.

Infectious agents incriminated in the cause of seminal vesiculitis include IPV virus, Chlamydia, *Mycoplasma bovigenitalium, A. pyogenes, C. renale*, Streptococcus, Staphylococcus, *E. coli*, Proteus, *Pseudomonas*

aeruginosa, Brucella. abortus, M. tuberculosis, M. paratuberculosis, Actinobacillus actinoides, Nocardia-like organisms, and *Aspergillus fumigatus.* There is a report of isolation *of T. foetus* from the (inflamed) seminal vesicles.

In chronic seminal vesiculitis, bacteriological investigations are almost always negative. Autoimmune phenomena against spermatozoa may be implicated.

As with orchitis it is difficult to relate the type of lesion to a specific aetiological agent and the above classification reflects the duration of inflammation rather than the cause. Nevertheless described lesions in seminal vesiculitis attributed to viruses, *A. actinoides* and *M. bovigenitalium* were of the predominantly degenerative type.

Suppurative lesions suggest the presence of bacteria (especially *A. pyogenes*) and in specific inflammations such as tuberculosis, the usual characteristic changes will be observed. Possibly bacterial involvement is secondary in many cases.

Ampullitis, prostatitis and bulbourethral adenitis:

These may occur with seminal vesiculitis and the causal agents are similar. The bulbourethral glands are the least often affected.

Ampullitis usually causes a slight increase in diameter of the gland, with increased turgidity due to oedema. Induration and excess connective tissue around the gland may occur. Purulent ampullitis seems not to have been described. Studies on allergic epididymo-orchitis indicated the ampulla as a prediction site for such inflammation.

GENITAL LESIONS IN SHEEP AND GOATS

Female genitalia

Ovine

In general, pathological conditions of the reproductive tract of the ewe parallel those of the cow. Other important conditions in the ewe, however, include cystic endometrial hyperplasia in response to ingested plant oestrogens (clover disease), and vulvitis – associated with posthitis in wethers and occasionally rams.

Clover disease:

In ewes grazing subterranean clover (*Trifolium subterraneum*), lambing percentages may be decreased by as much as 20%. In affected flocks the disease is characterised by a high occurrence of difficult births, cystic hyperplasia of both cervix and endometrium, inactive ovaries, and hydrometra. Endometritis also occurs, presumably from functional damage to the cervix allowing passage of bacteria into the uterus. Although oestrogens are the cause of disease, prolonged exposure of ewes to these pastures results in masculinisation of the external

genitalia. There is partial fusion of the lips of the vulva, and hypertrophy of the clitoris.

Vulvitis:

In ewes, ulcerative lesions similar to the external ulcers of wethers appear ("scabby ulcers"), and sometimes ulceration extends into the vagina. These ulcers do not seem to interfere with conception or parturition but they are a factor in maintaining the disease in a flock and do predispose to fly-strike. As in wethers, a high protein diet and an organism resembling *Corynebacterium renale* are causally involved.

Spontaneous, partial or total *vaginal rupture in pregnant ewes*, has recently been described. The lesion always consisted of a tear in the dorso-lateral aspect of the vagina with a partial or total perforation of the wall close to the cervix. Most cases occurred one week before expected lambing. Circulatory disturbance resulting from uterine torsion was considered to be the underlying cause.

Caprine

Although genital disease in the doe is, in general, comparable to the ewe, several conditions warrant special mention.

Intersexuality:

This condition is an important cause of infertility in the doe and buck. In both cases the intersex state is related to hornlessness. If either parent is horned the offspring will almost never be an intersex. Affected animals are genetically female but abnormalities present include ovotestes, hypoplastic abdominal testes, and enlarged clitoris (visible externally) or obvious penis - sometimes with hypospadias.

Hydrometra and pseudopregnancy:

Hydrometra is also a significant cause of infertility and abdominal distension in the doe. The uterus is thin-walled and contains up to several litres of clear serous fluid. The cause is apparently unknown.

Pseudopregnancy occurs rather frequently in the doe. Although the cause is unclear, hormonal imbalances and early foetal resorbtion maybe involved – uterine fluid continuing to accumulate thereafter. Pseudopregnancy terminates in the doe voiding much cloudy fluid ('cloudburst'), which sometimes may represent spontaneous correction of hydrometra.

Herpesvirus vulvitis:

As in the buck (see below) the morbidity of this condition may be quite high. Characteristic are initial discrete erosions, pustules then ulcers up to several mm in diameter on the vulval and vaginal epithelia , and an associated erythema with a yellow to grey vaginal discharge. These lesions heal spontaneously, usually within two weeks.

Male genitalia

Ovine

Ulcerative posthitis occurs in wethers particularly, probably because of their tendency to urinate within their sheath. Urine rich in urea facilitates the growth of *Corynebacterium renale* in animals on a high protein diet. Hormonal factors may also be involved.

In early lesions there is focal necrosis on bare epithelium near the preputial orifice. This expands to involve the whole orifice, which may become stenosed, and infection will extend into the preputial cavity.

Fully developed lesions are those of extensive internal ulceration of the prepuce, hence the common name "sheath rot", with much accumulation of urine and pus and ulceration of the penis.

Bacterial epididymitis is of particular importance in sheep as compared with other domestic species. The cause is *Brucella ovis* or other so-called gram-negative pleomorphic organisms (GNPO) such as *Actinobacillus seminis* and *Histophilus ovis*.

There are important differences in pathogenesis depending on age of rams, and the causal organism. Whereas infection with *Br. ovis* mostly infects the epididymis of mature rams via a haematogenous route, infection with the GNPO ascends from the prepuce - especially in young rams, in which hormonal changes associated with puberty are important.

Macroscopic lesions may not always develop, but if present these are similar, irrespective of which of the above organisms is involved. Lesions, which may be uni- or bilateral and overwhelmingly involve the tail of the epididymis, are those of marked enlargement; there is no associated orchitis. The affected epididymis is firm to hard and often irregularly nodular as a consequence of spermatic granuloma formation and fibrosis. Associated tunic adhesions are common.

Varicoceles occur in 1-2% of rams and present clinically as nodular swellings above the testis.

The cause of varicocele is unknown. Early events are likewise unclear as most varicoceles in rams are thrombosed when seen. Once established, varicoceles seem to quickly increase in size - possibly due to a compounding effect of inadequacy of valves and the dependent position of the testis. Some degree of testicular atrophy is associated with large varicoceles.

Most varicoceles are located high in the pampiniform plexus near the inguinal ring and some distance from the testis. Dimensions of 10 - 40 cm are common and such varicoceles are composed mostly of laminated thrombi. Associated changes are testicular mineralisation, oedema of the epididymal head, and sometimes thrombosis of testicular vessels.

Bulbourethral gland hyperplasia in wethers (but not rams) results from their grazing certain clovers (*Trifolium* spp.) which have estrogenic activity. The development of cysts in these lesions may cause enormous

enlargement that gives swelling in the perineal region and sometimes urethral obstruction and death.

Caprine

Herpesvirus balanitis, which as in the bull (but not the ram) causes discrete ulcers, may lead in the buck to extensive necrosis of both penis and prepuce. Lesions vary from discrete 2-4 mm diameter ulcers on preputial epithelium to severe necrosis and suppuration and secondary bacterial infection of prepuce and penis, and phimosis. Stress associated with herding and transport of bucks, is a likely explanation for the severity of lesions.

A further interesting species difference occurs in relation to brucellosis. Whereas *Brucella ovis* infection in the ram typically causes a slowly progressive epididymitis, *Br. abortus* and *Brucella melitensis*, in the bull and buck, respectively, causes are more rapidly progressive inflammatory process that targets the testes.

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