SECTION 3 MALE THERIOGENOLOGY

3.1 CLINICAL CONDITIONS OF THE MALE

GENERAL INTRODUCTION

Diseases and conditions of the male reproductive system are approached from a locations perspective, beginning at the site of production of spermatozoa and ending with the penis and prepuce. This parallels the problems based approach as diseases tend to involve the scrotum and contents, the accessory genital glands and or the penis and prepuce. Diseases of individual species will be dealt with in the context of anatomical locations. If you are interested in one particular species only, the information for other species is still relevant because a disease in one is usually identical to that in another!

My reproductive pathology website (www.VetReproPath.com) provides additional information.

SCROTAL CONTENTS

INTRODUCTION

The testes are often considered the center of the 'male reproductive universe'. Not every disease of the scrotal contents involves the testes, however. **Orchitis** is a common clinical diagnosis, but it is often a misleading diagnosis because severe inflammation of the testes is a rare event. The inflammation and/or infection are usually of the tunica vaginalis and epididymis. Often times, the reaction is to spermatozoa that have leaked into the tissues. The ability of spermatozoa to stimulate a florid inflammatory reaction is overlooked, but is the most important factor when considering prognosis.

SPERMATIC GRANULOMA

The inflammatory and immunologic response to spermatozoa is of critical importance in many diseases and conditions of the male reproductive tract. Any condition that causes leakage of spermatozoa or spermatozoal antigens into the extratubular compartment is potentially complicated by this reaction. **Spermatozoa incite a granulomatous and pyogranulomatous reaction known as a spermatic granuloma**. Macroscopically, these have an appearance similar to pus. Spermatozoa have a cell wall similar in composition to keratin as they have many sulphur bonds. Spermatozoa produce a foreign body type reaction and the large number of them and their keratin-like composition means that degradation is a slow process. In addition, immune responses are florid and result in the accumulation of many immunoglobulin producing cells as well as CD4- and CD8-expressing lymphocytes. Upregulation of major compatibility complex (MHC) I in epithelial cells also occur. The reaction leads to chronic inflammation and fibrosis, and continued disruption and obstruction of tubes and tubules. **Spermiostasis, spermatocele and/or further spermatic granulomas are the consequence**.

DISEASES OF THE VAGINAL TUNICS

Diseases that directly affect the tunica vaginalis, primarily, are very rare. Secondary involvement after epididymitis, is a much more frequent finding.

Of the domesticated species the most well known primary cause of inflammation of the vaginal

tunics is feline infectious peritonitis (FIP). Scrotal swelling in the cat is a presenting sign that should alert the practitioner to consider feline infectious peritonitis (FIP). Tunica vaginitis is also seen in traumatic events that allow introduction of bacterial pathogens into the vaginal recess. This recess is continuous with the peritoneum; any disease affecting the peritoneum can and will affect this area.

DISEASE OF THE TESTIS AND EPIDIDYMIS

The testes produce spermatozoa, testosterone and other hormones. Regulation of testicular development, descent, and function is a complex procedure that involves many different hormones, growth factors (including insulin like growth factors) and the interaction of systemic and local factors and their receptors. Not all are well understood.

Germ cell, testicular sustentacular (Sertoli) cell and interstitial endocrine cell function is closely interrelated. Control of single cell death (regulated cell death and apoptosis) is now identified as a critical process in testicular physiology and pathophysiology. It is a normal process that limits potential spermatogenesis by about 25 to 75%. Gonadotrophins (FSH, LH) and intratesticular androgens act as survival factors, whereas external events such as elevated temperature increase single cell death of spermatocytes.

Small testes

Small testes are cause for concern. Compensatory hypertrophy of one testis may be an indication that all is not well in the contralateral testis. **Small testes mean reduced production of spermatozoa because testicular weight and volume are correlated with daily sperm output.** Other measurements are also correlated to daily sperm output. Scrotal circumference in the bull is highly correlated with daily sperm output, but there are few good measures used in the other species. Scrotal circumference can be used in the ram and buck, but the lack of accessible testes in the other species means that we must rely on comparing the testes with objects of the same shape and known volume - orchidometers. Testicular size is seldom a selection criterion on non-ruminants.

It is almost impossible to differentiate the cause of small testes in older animals unless the testes have been examined previously. Primary testicular hypoplasia and testicular atrophy cannot be accurately differentiated because hypoplastic testes undergo degeneration. When animals are examined at puberty, ones confidence in diagnosing hypoplasia is much higher. In general, **a hypoplastic testis has a small hypoplastic epididymis attached**. The epididymis is usually disproportionately larger in testicular atrophy.

Hypoplasia

Testicular hypoplasia is a Disorder of Sexual Development (DSD) (and thus could be Chromosomal, XY or XX in type). It occurs in cryptorchidism, in conjunction with abnormal sex chromosomes, and as an ‘uncomplicated’ condition. It can be either unilateral or bilateral. The failure of one or both testes to grow to a normal size is because of a reduction in the amount of the seminiferous lining. An affected testis is grossly similar to normal testis in almost every way – except size.

Hypoplasia is the potential end result of a large number of different abnormalities that may operate at a systemic or local level (perhaps in the case of unilateral disease). This potentially large pool of possibilities means that we are seldom able to identify a cause.

The difference in size is theoretically because of a reduction in either the number of tubules, the length of tubules, the diameter of the tubules, or one or more combinations of these. Germ cells may be absent or present but fail to produce enough spermatozoa. Germ cells may have failed to migrate to the genital ridge *en utero*, failed to migrate in sufficient numbers, failed to survive, have arrested development, undergo excessive single cell death or undergo degeneration at some stage of spermatogenesis. Although we are aware of these possibilities, we often lack the tools and finances to determine which may be operating; the molecular mechanisms are largely unknown.

Hypoplastic testes with a total lack of germ cells are very small and fail to enlarge from their size at birth.

A deficiency of gonadotrophins is associated with hypoplasia (hypogonadotrophic hypogonadism) in humans and mice, but studies in domesticated mammals indicate a normal or raised serum concentration of follicle stimulating hormone and luteinizing hormone. Testosterone concentration was lower than normal in some bulls with hypoplasia or degeneration, while in sheep, testosterone and inhibin concentration may be normal.

At the chromosomal level (Chromosomal DSD), hypoplasia in animals with a Klinefelter like syndrome is recognized. Affected cats, bulls, dogs, pigs, horses and sheep have an XXY genotype or a mosaicism with XXY chromosomes. The most famous of these is the male tortoiseshell or calico cat. In goats of the Saanen breed with the gene for polledness, testicular hypoplasia is seen in XX testicular DSD (previously called female pseudohermaphrodites or XX sex reversal). A variety of other chromosomal abnormalities have been identified as being associated with hypoplasia.

At the genetic level, hypoplasia is known or suspected to be hereditary in the dog, bull, ram, and buck. The exact genetic abnormality is often not established.

Cryptorchidism

The diagnosis of testicular maldescent can be a clinical challenge when there are no scrotal testes or only one scrotal testis but no history of whether castration was performed previously. Monorchia or anorchia is a very rare but possible occurrence. The cryptorchid testis is hypoplastic and will eventually degenerate. Although spermatogenesis may be absent, testosterone production continues for a long time. Tests such as HCG stimulation can be performed in bilateral cryptorchids, but there are other ways. Structures that are testosterone dependent include the prostate of the dog, the barbs on the penis of the cat, and the vesicular glands (seminal vesicles) of the bull and stallion. Castration of a cryptorchid may be straightforward or difficult, depending on the location and size of the retained testis.

Cryptorchidism is very common, and is the most common reproductive abnormality of cats and horses. Juvenile dogs may have an incomplete inguinal ring for about 6 months and it is possible for a testis to move back and forth. In the majority of cases, the testis of dogs is in the scrotum by day 10 after birth.

The cause of cryptorchidism is difficult to establish in a single individual. Cryptorchidism is the potential end result of genetic, hormonal, structural or other abnormalities. A polygenic recessive - hereditary basis has been established or suggested in all species. Chromosomal abnormalities were implicated in rams, but in humans, chromosomal anomalies were detected in 4% of a group of 160. The genomic control of testicular descent is not known.

The structural anomalies associated with the failure of the testis to migrate to the scrotum include splenogonadal fusion, retention of the cranial gonadal suspensory ligament, and abnormalities of the gubernaculum and vaginal process. There are three phases of testicular descent, the abdominal translocation, the transinguinal migration and the inguinoscrotal migration. The first phase involves anchoring of the testis to the inguinal ring by the gubernaculum and relaxation of the cranial suspensory ligament, and this may be controlled by insulin like peptide 3. The second phase involves opening of the inguinal ring by an enlarged gubernaculums and intraabdominal pressure. The third phase takes the testis from the subcutaneous location into the scrotum, and is an interaction of androgen, calcitonin gene related protein and the genitofemoral nerve. The exact mechanisms are yet to be elucidated, but they will no doubt be complex and involve an interrelationship of systemic and local factors. The usual location of the retained testis is adjacent to the inguinal ring, within the inguinal ring and beneath the inguinal ring in a subcutaneous location, representing failure of one of the phases of migration. Diagnosis and therapeutic intervention to modify or control testicular descent, based on correcting the exact abnormality, is currently a theoretical possibility.

Some of the known causes of testicular degeneration in mammals, including rodents.

- Advancing age
- Chlorinated naphthalenes
- Epididymitis
- Chemicals
  - Chemotherapy
  - Halogenated compounds including
    - hexachlorophene
  - Nitrogen containing compounds including
    - Benzimidazoles
    - Nitrofurans
- Heat
- Hormones
  - Dexamethasone
  - Estrogen
  - Testosterone
  - Zeranolone
- Metal compound toxicity
- Neoplasia
  - Pituitary tumors
  - Sustentacular (Sertoli) cell tumors
- Nutritional disorders
  - Negative energy balance
  - Fatty Acid deficiency
  - Hypovitaminosis A
  - Hypervitaminosis A
  - Hypovitaminosis B
  - Hypovitaminosis E
  - Hypovitaminosis C
  - Protein and amino acid deficiency
  - Zn deficiency
- Plants
  - locoweed (Astragalus)
  - Lysine seeds
- Radiation
- Stress/Corticosteroid therapy
- Trauma
- Ultrasound
- Viral infection
  - *Betaarterivirus suid 1* (PRRS virus)
  - *Pestivirus A*, B (BVDV)

Testicular Degeneration

Testicular degeneration is manifested clinically and grossly as atrophy, mineralization, and fibrosis. The germinal epithelium reduces in amount and eventually all spermatogenesis stops. There is often spermiostasis, and mineralization of the

tubular content. Spermatic granulomas may develop.

Testicular degeneration usually occurs because of influences external to the testis (cf. hypoplasia). There is a seemingly endless list of potential causes. The close interrelationship of the testicular sustentacular (Sertoli) cells, interstitial endocrine cells and germ cells means that insults on any one or several of them eventually affect them all. Even though the insult is external to the testis, the manifestation in the testis is not always bilateral or uniform. Each species has a distinct pattern. The testes of bulls frequently degenerate from the ventrum, and rams from the dorsum. Mineralization, a common sign of degeneration, may involve whole or part of one seminiferous tubule, or whole regions. This pattern of change is only significant if the testes are biopsied, as the finding of mineralization may not reflect the state of the entire testis.

The basic mechanism of degeneration of the cells that make up the germinal lining is presumed to be similar to degeneration of other cells and tissues. The more we learn about the mechanisms involved in other tissues, the more will be identified in the testis. The more recent emphasis of the importance of apoptosis is a good example.

Neoplasms

Testicular neoplasms are most commonly found in the dog. There is no satisfactory explanation for this, but intact dogs are allowed to live as long as they are able, and they tend to be watched closely. Male cats tend to be castrated early and fewer of those that are intact live to old age. Neoplasms arise in all species sporadically, and with the exception of the dog, it is possible to predict the histologic type of neoplasm based on species and age. Seminoma is the common tumor of the aged, and teratoma is more common in young horse. Cats, rams and bucks get neoplasms rarely, and both seminoma and testicular sustentacular (Sertoli) cell tumor are reported. Bulls are more likely to have interstitial cell tumors, but testicular sustentacular (Sertoli) cell tumors are also reported.

Three main testicular neoplasms of dogs are the testicular sustentacular (Sertoli) cell tumor, the interstitial cell tumor and the seminoma. Multiple types of neoplasia may be found in one testis. Most primary testicular neoplasms in dogs are benign. Exceptions are extremely rare. Identification of metastasis is the only way to determine that the neoplasm is malignant; there are no good cytological or histologic markers.

Most neoplasms cause enlargement of the testis. In general, seminomas are white, soft and usually bulge on cut section. Testicular sustentacular (Sertoli) cell tumors tend to contain a lot of fibrous tissue so they are white in color and are firm. The interstitial cell tumor is tan or yellow in color, often contains areas of hemorrhage, and is soft.

Testicular sustentacular (Sertoli) cell tumors, but rarely interstitial cell tumours may produce a hyperestrogenism like syndrome and feminization. This is usually manifested by attractiveness to other male dogs, gynecomastia, and alopecia. Some develop bone marrow aplasia. Affected animals return to normal after removal of the neoplasm. The signs are not always associated with estrogen production and not all dogs will have a raised serum estrogen. In these instances inhibin secretion by the neoplastic testicular sustentacular (Sertoli) cells inhibits the secretion of follicle stimulating hormone and luteinizing hormone by the pituitary, which in turn, inhibits testosterone production. The imbalance between testosterone and oestrogen is

responsible for the relative increase in oestrogen and feminisation. Some animals will develop bone marrow suppression, and a poorly responsive pancytopenia. Feminization is much more common when the neoplasm is larger, and therefore is more common in cryptorchid dogs. It is also in these dogs that an unfortunate sequel of testicular torsion can occur.

Testicular Torsion

With the exception of stallions, torsion of the testis is very rare unless there is incomplete descent. A testicular neoplasm is often also present to provide sufficient weight to maintain the torsion. The usual clinical presentation is acute abdominal pain, and the offending mass is blackened due to venous infarction and is sometimes indistinguishable as testis. Spontaneous torsion of the cryptorchid testis of boars is seen commonly at slaughter.

Orchitis

Apart from bulls in areas endemic for *Brucella abortus*, orchitis is a rare and sporadic disease in domesticated animals. The vast majority of cases diagnosed clinically as orchitis are really epididymitis. Focal accumulations of lymphocytes are occasionally seen in the testes of most species and suggest a subclinical disease.

Orchitis as the primary manifestation occurs sporadically and has been reported in cats with feline infectious peritonitis, rams and bucks with *Corynebacterium pseudotuberculosis*, pigs with *B. suis* or *Burkholderia pseudomallei* and stallions with migrating larvae of *Strongylus spp.* nematodes. Sporadic infection with other bacteria will no doubt occur from time to time. The isolated position of the testis suggests that infection with the various agents is hematogenously derived, or occurs by direct traumatic penetration.

Developmental epididymal disease

There are many different diseases that affect the epididymis, but only two that we will deal with specifically here. They are segmental aplasia and spermatic granuloma of the epididymal head.

The first of these, segmental aplasia occurs when a portion missing. Usually it is the tail of the epididymis. As can be expected, the affected side is sterile. This disease has a hereditary basis in bulls.

The second disease is called spermatic granuloma of the epididymal head. In this condition, not all efferent ductules attach to the epididymal tube -- some are blind ending. In the blind ending ones, spermiostasis develops and subsequently becomes a spermatic granuloma. This disease is recognised as the granulomas only occur in the region of the epididymal head.

Infectious Epididymitis

Bacterial agents cause most infections of the epididymis. Viruses, such as Equine Arteritis Virus and others are reported to induce epididymitis.

Primary infection with *Brucella spp*. in each species results in epididymitis. *B. ovis, B. canis, B. melitensis* and *B. suis* are especially virulent for the epididymis. It is assumed that the infection is systemic and the bacterium localizes in the epididymis.

Direct infection of the epididymis by penetrating injury is a rare event. Secondary infection from periorchitis, or peritonitis is an occasional possibility.

Almost all species develop infection of the epididymis by the ascending route. This has been studied in the ram where *Actinobacillus seminus* and *Histophilus somni (Histophilus ovis)* are common isolates. While the exact mechanism is not known, the work of Jansen (1983) in the 1980's

indicated that preputial organisms migrate to the accessory genital glands and infect the epididymis by retrograde movement. The privileged environment of the lumen of the epididymal duct allows the organisms to infect the organ and incite damage. The formation of spermatic granulomas means that the reproductive potential of the affected side is lost. Complete return to normal is rare. In the dog, the sequel of self-trauma of the scrotum and systemic affects of infection with endotoxin producing bacteria such as *Escherichia coli* further complicates epididymitis by causing systemic illness.

Vaccination and local immunity to prevent epididymitis is not likely to be achieved very soon. The epididymis has no natural local immune system of antigen receptors, recirculation of immunocytes and no local plasma cell population. Some aggregates of lymphocytes are occasionally seen in otherwise normal animals. After infection, the epididymis must develop a local immune system, but alas, the damage is usually so extensive, and the sequelae so severe that immunity is too late. Even so, the epithelial cells have the ability to express MHC I and II and lymphocytes and plasma cells can be recruited after challenge.

SPERMATIC CORD

The spermatic cord is composed of the deferent duct (ductus deferens), pampiniform plexus, muscle and nerve. There are many diseases that affect this area. The principal ones we will deal with are varicocele and inguinal hernia.

When palpating the region of the spermatic cord and the inguinal ring, all of the structures of this area can usually be identified. The deferent duct may have indications of a previous vasectomy, the lymph node may be enlarged with lymphoma, and a large inguinsl hernia will be palpated as a sack like structure that may contain intestines. It is particularly important in the horse to palpate the scrotum of animals that have colic.

Varicocele is a disease that particularly affects old rams. It can be a cause of subfertility because of a lack of thermoregulation. It is a very common disease in humans where up to 20 percent of individuals may have varicocele of the left side. Varicocele is the best recognised when there is thrombosis.

ACCESSORY GENITAL GLANDS

The accessory genital glands (AGG) include the ampullae, vesicular glands (seminal vesicles), prostate, and bulbourethral glands. They are frequently overlooked during necropsy examination, unless there are specific indicators of disease. For routine necropsy examination of these glands, the pelvic bones must be cut and a block of bone removed. I find it easier to remove the pubis by cutting the pubic and ischiatic arches from the obturator foramina.

Diseases of the AGG are sporadic in most of the species and tend to be incidental findings. Disease of the prostate of the dog, and vesicular glands (seminal vesicles) of bulls, is common and important.

Prostatic disease in the Dog

The prostate is the only genital gland of the dog and it is prone to diseases that can be difficult to differentiate. The gland is relatively inaccessible and signs of prostatic disease are not always specific to that organ.

Clinical signs

The major diseases that affect the prostate are prostatic hyperplasia, prostatic cysts, paraprostatic cysts, bacterial prostatitis and prostatic neoplasia.

Enlargement of the prostate due to hyperplasia or some combination of hyperplasia and cyst formation is common. In its pure form, excessive prostatic enlargement may be either completely incidental or found on rectal palpation, or may cause lethargy, straining to defecate and anorexia. Bacterial prostatitis, in its acute form, causes hematuria, lethargy, and signs of systemic illness. Infertility will occasionally be the only indication. Prostatic neoplasia tends to be insidious and it is only in advanced disease that the signs of tenesmus, stranguria, dysuria, or the cachexia of malignancy are seen. When there is evidence of tenesmus, urethral discharge including hemorrhage, and hematuria, one can focus specifically on the prostate as the origin.

Diagnosis of prostatic disease.

Apart from clinical signs that can be quite vague, rectal palpation is often useful in diagnosis.

Hyperplasia tends to produce a prostate that is uniform and smooth surfaced. Acute prostatitis will usually result in pain on palpation and a “doughy” feeling to the prostate with sponginess and a lack of definition due to periprostatic edema.

In prostatic neoplasia palpable changes will vary from no significant findings, to a total inability to palpate the prostate because of extensive periprostatic fibrosis and prostatic infiltrate.

Ancillary diagnostic aids that greatly assist in the diagnosis include ultrasonographical imaging, cytological examination of urine, prostatic washes, semen evaluation, penile discharge, urethral brush biopsy and fine needle aspiration. Punch biopsy, direct incisional prostatic biopsy, or excision biopsy are also possible.

Prostatic hyperplasia.

The prostate gland has been the focus of much research, especially in the area of prostatic hyperplasia. Dogs and men both develop prostatic hyperplasia as they get older. The hyperplasia in dogs does not usually cause a urinary obstruction as it does in men. The enlargement occurs in an eccentric fashion so that it may act as a ball valve in the pelvic inlet and restrict the passage of feces along the colon. Enlargement of the prostate with age is progressive and dependent on the presence of testes.

There is little useful information available about the rate of prostatic enlargement for species other than the Beagle. Scottish Terriers are reported to have a normal prostate that is much bigger than in other species.

Castration of juveniles reduces the number of prostatic diseases to one - carcinoma. (see below).

Prostatitis.

Acute prostatitis is a disease that arises from ascending infection and often is caused by *E. coli*. The signs are as described above and are often typical of what one finds in any infectious disease. Chronic prostatitis is frequently a subclinical disease. It may cause a urethral discharge, infertility, recurrent cystitis and hematuria.

Diagnosis of prostatitis is made difficult by its proximity to the bladder and urethra, so that semen can be contaminated with urethral material and urine. A urethral brush and prostatic massage is probably the most accurate method for sampling prostatic secretion if there is no facility for direct

needle biopsy that is guided by ultrasonographical imaging.

Paraprostatic cysts

There are a large number of cystic structures that can occur around the prostate. Many of the cysts probably arise as a cystic change in hyperplasia. Cysts that are small and multiple are often clinically silent until they are complicated by infection. Others may become extremely large and have a space-occupying effect. The prognosis for single large cysts is usually very poor because they can seldom be completely removed.

Prostatic abscess

Prostatic abscesses probably arise from bacterial infection of a prostatic cyst. They may be single or multiple. Signs are usually noticed when the dog becomes clinically ill, and fever, anorexia, pyrexia, and urethral discharge are presenting complaints. Gram-negative bacteria especially *E. coli*, *Mycoplasma*, *Staphylococcus*, and *Streptococcus* are possible agents.

Prostatic neoplasia

There is considerable confusion regarding the terminology of carcinomas of the prostate and the literature is difficult to interpret because different terminologies are used. The list of subtypes continues to expand! The phrase ‘carcinoma of the prostate’ will be used as a general term. There is no known prognostic benefit to subtyping as yet.

Previously neutered dogs develop carcinoma at about the same prevalence as entire males.

Signs if carcinoma of the prostate include obstruction of the urethra, erosion of blood vessels and resultant hematuria, or evidence of extensive metastasis.

Carcinoma of the prostate can produce widely metastatic disease while maintaining a very small primary lesion. Alternatively, large prostatic carcinomas may have no metastasis. Instead they can be a locally infiltrative disease and result in extensive periprostatic and intrapelvic spread. The extensive fibrosis and space occupying effect can result in stricture of the rectum and colon, resulting in tenesmus and other signs of constipation. Hind leg lameness and weakness, and emaciation are frequently seen. Carcinoma of the prostate inconsistently produce prostatic specific antigen (PSA), so this is not a good marker for the disease. Recommended classification is currently prostatic adenocarcinoma, prostatic urothelial carcinoma and mixed prostatic adenocarcinoma and urothelial carcinoma. There are also rare subgroups.

Vesicular adenitis (Seminal vesiculitis) in bulls

Vesicular adenitis occurs commonly as a subclinical disease in ruminant species. In bulls it is recognized as an important clinical disease and a cause of infertility and poor freezibility of semen. It is a disease of young bulls predominantly – those under 2 years. As with epididymitis, it is believed that most cases not associated with *Brucella* infection are the result of ascending infection.

Two forms of vesicular adenitis in the bull are recognized, an acute fibrinopurulent form where there are the typical signs of acute inflammation – swelling, pain on palpation and neutrophils in the semen. Some bulls may have systemic signs. A chronic interstitial form is the second type and there is a considerable increase in size, excessive fibrosis, firm consistency and loss of lobulation.

PENIS AND PREPUCE

Diseases of the penis and prepuce are common in all species. Examination of the penis and prepuce

can be a challenge in many species – and in some conditions, examination during mating is required. Many of the diseases that are congenital or developmental are associated with disorders of sexual development. Congenitally short penis, lack of sigmoid flexure in ruminants and other obvious and less obvious anomalies may be seen from time to time. Congenital deviations of the penis occur and may require observation of mating to diagnose.

The epithelium of the penis and prepuce are fused until puberty, when there is separation of the epithelium. Failure of complete separation will result in a persistent membrane (called a persistent preputial band) or frenulum that may cause deviation of the penis when it is erect.

Eversion of the preputial mucosa is common in bulls of the *Bos indicus* and in polled breeds of the *Bos taurus* species. The eversion is permitted by inadequate muscular arrangements in the prepuce, and its importance lies in the injuries acquired to the everted epithelium. Trauma and desiccation lead to edema, inflammation, and preputial prolapse.

Forced deviation of the penis (hematoma of the penis, broken penis) is a disease of bulls mostly. Deviation of the penis during coitus causes the extremely high pressure generated by the coital thrust to result in rupture of the penis at the level of the insertion of the retractor penis muscle. A hematoma develops and it may be large enough to cause hypovolemic shock. Most are not so immediately life threatening and will heal with scarring and phimosis.

Paraphimosis is a complication of tranquilizing horses, especially if phenothiazines are used. Affected horses are unable to retract the penis, and engorgement and trauma result. Dogs develop paraphimosis as a 'spontaneous' lesion.

In dogs, trauma from car accidents, mating injuries or foreign material within the prepuce presents little diagnostic challenge if the area is carefully examined. Where there is extensive trauma to the penis, fracture of the os penis may occur.

Most species develop urolithiasis - but especially in cats and ruminants - that result in stones lodging in the penile urethra. Penile necrosis, and 'water belly' with bladder and or urethral rupture, occurs.

Inflammation of the penis and prepuce

Inflammation and or infection of the prepuce (posthitis) mostly occur as a nonspecific event. There are several terms used to describe this region of the male anatomy. Balanitis is inflammation of the head of the penis. Inflammation of the head of the penis and prepuce is called balanoposthitis but in all species except the dog, should more accurately be called phalloposthitis. In dogs, the head of the penis is the portion of the penis from the bulb to the tip, so balanoposthitis is the appropriate term.

As with any external site, the prepuce has a normal flora that contains potential pathogens. Sexually transmitted organisms are usually found in the prepuce and may not be a cause of disease in males. Organisms of importance include the herpesviruses, mycoplasmas and ureaplasmas of most species. Specific agents include *Tritrichomonas foetus*, *Campylobacter fetus*, *Corynebacterium renale* and *Ureaplasma diversum* in bulls, and *Eubacterium suis* (basonym of *Actinobaculum suis*)in pigs

A preputial discharge is seen frequently in male dogs. Most cases are due to a non-specific balanoposthitis. Geldings tend to extrude their penis less often than stallions, and they urinate in

their sheath. The buildup of smegma and or the effect of urine allow secondary organisms to flourish.

Outbreaks of posthitis are recorded in wethers, and rarely in rams. There is a combination of factors operative in this disease. The animals are usually on a high protein diet thus producing abundant urea. The causative agent is *Corynebacterium renale*, a urease-producing organism that breaks down urea to ammonia. It is believed that the ammonia causes ulceration adjacent to the preputial orifice. Further damage and infection results in a severe posthitis that can result in preputial obstruction, scarring and eventually death.

Preputial prolapse in bulls with resultant severe inflammation and swelling has already been discussed.

Boars develop a unique disease that involves the preputial diverticulum. Preputial diverticulitis with ulcers and necrotic debris is seen.

Intrapreputial foreign bodies will cause inflammation also. Sand in dogs and 'hair ring' in ruminants are occasionally seen.

Neoplasms of the penis and prepuce

Canine transmissible venereal tumour in endemic areas is relatively common, and diagnosis of masses around the prepuce can usually be made on either fine needle aspiration or incision or excisional biopsy. Most transmissible venereal tumours are exquisitely sensitive to treatment with vincristine.

Squamous cell carcinoma of the penis of the horse is a relatively common condition of older animals. It was once thought that smegma may be carcinogenic, but that theory has fallen into disrepute. *Equus caballus papillomavirus*-2 (EcPV-2) is now considered an important factor.

Young bulls commonly develop **fibropapillomas**. The papilloma virus, bovine papilloma virus type 2, causes these exophytic lesions. They often occur during the first mating season, and they are fleshy lesions that become ulcerated and cause hemorrhage and pain.

DIFFERENTIAL DIAGNOSES

Testicular masses

- neoplasms
- spermatic granuloma
- ectopic adrenal

Epididymal masses

- spermatic granuloma/epididymitis
- cystic structures
- parasitic cysts
- ectopic adrenal

Spermatic cord

- varicocele
- lymphadenitis
- spermatic granulomas
- abscesses
- cystic structures (Hydatid of Morgagni, stallion)
- parasitic migration tracts - strongyles of horses

Tunica vaginalis

- peritonitis/periorchitis
- cystic structures
- neoplasia - mesothelioma
- scrotal hernia
- parasitic cysts/migration tracts
- scrotal hernia

Penis

- Nonspecific posthitis

Herpesvirus
Trauma
Transmissible venereal tumor
Fibropapilloma
Squamous cell carcinoma
Habronemias