Proceedings of the
World Small Animal Veterinary Association
Sydney, Australia – 2007

Hosted by:

Australian Small Animal Veterinary Association (ASAVA)

Australian Small Animal Veterinary Association (ASAVA)

Australian Small Animal Veterinary Association (ASAVA)

Next WSAVA Congress

33rd Annual
World Small Animal Veterinary Association
14th FECAVA Congress
DUBLIN, IRELAND
20th - 24th August 2008
Disorders of the prostate are fairly common in the dog with prostatomegaly evident in two-thirds of dogs, over five years of age.

Pathophysiology

As a dog ages, spontaneous enlargement of the prostate gland occurs, which is referred to as benign prostatic hyperplasia. Despite this enlargement, the prostatic secretory capacity decreases as the hyperplasia develops. The prostate gland can undergo both glandular and complex hyperplasia.

Glandular hyperplasia begins at 1-2 years of age and its prevalence peaks at 5-6 years. During this time the prostate develops a proliferation of secretory structures that are uniformly distributed throughout the gland. There is an increase in the number and size of the epithelial cells, accompanied by a smaller but significant proliferation of stromal fibroblasts and smooth muscle.

Complex hyperplasia generally develops later, but the first evidence appears at 2-3 years of age. Cystic, dilated alveoli filled with eosinophilic material are a characteristic feature and can give the surface of the gland an irregular contour. The cysts can be distributed throughout the gland but are more numerous in the peri-urethral areas. Infiltration with lymphocytes and plasma cells is common, appearing as clusters or nodules of cells in the stroma. Frequent epithelial atrophy and occasional epithelial squamous metaplasia are associated with these areas of cell infiltration.

Excessive androgen or oestrogen (iatrogenic, neoplastic lesions, or testicular dysfunction) can cause prostatic enlargement. An androgen imbalance leads to cystic hyperplasia of the acini and ducts together with hypertrophy of the fibro-muscular framework. On the other hand, an oestrogen imbalance causes hyperplasia of the ducts together with squamous metaplasia of the epithelial lining and desquamation into the lumen. The acini may or may not become hyperplastic. In time, prostatic secretions pool in multiple small to large cysts, which dilate the ducts and the gland.

Diagnosis Of Prostatic Disorders

Most dogs with prostatic disorders are middle-aged or older intact animals. Although neutered dogs are at a reduced risk for prostatic disease, prostatic neoplasia is still possible.

Prostatic disease can present with clinical signs attributed to the urinary tract (haematuria, pyuria, dysuria, incontinence), colon (constipation, diarrhoea, tenesmus, dyschezia), locomotory (hindquarter stiffness or lameness), as well as non-specific systemic signs (fever, weight loss, anorexia, PuPd, vomition).

Due to the variety of prostatic disorders that can occur in dogs, each necessitating a different therapeutic approach and prognosis, an accurate diagnosis is of great importance. If the history, clinical examination, and rectal examination palpation are suggestive of
prostatic disease, the following diagnostic procedures can be performed to establish an aetiological diagnosis:

- Survey and contrast radiography.
- Ultrasonography.
- Cytological evaluation and bacterial culture of urine, prostatic fluid wash, or semen.
- Fine needle aspirate cytology of the prostate.
- Prostate biopsy.

Radiological Signs of Prostatic Disease

- Cranio-ventrally displacement of the bladder.
- Ill defined mass between the bladder and pelvic brim.
- Dorsally displaced colon.
- Calcification either within the prostate (indicative of neoplasia) or peripherally, which is usually the wall of paraprostatic cysts.
- A periosteal reaction along the ventral lumbar vertebral bodies or ileum may be indicative of neoplasia.
- Sub-lumbar lymphadenopathy may indicate neoplasia or infection.

Ultrasonographic Signs of Prostatic Disease

- **Cavitating bacterial prostatic disease** – intraparenchymal cavity or cavities greater than 1½ cm diameter and associated with increased background echogenicity, prostamegally and asymmetry. Cavity may have an irregular internal border, is anechoic to hypoechoic, and usually shows some acoustic enhancement.
- **Non-cavitating bacterial prostatic disease** – similar to above but cavities <1½ cm in diameter and often accompanied by focal irregularly marginated hyperechoic areas throughout the glandular parenchyma.
- **Benign prostatic hyperplasia** – symmetrical prostamegally with smooth contours and may be hyperechoic.
- **Cavitating prostatic disease** – intraparenchymal cavity or cavities greater than 1½ cm diameter. Symmetrical prostamegally with smooth contours and may be hyperechoic. Possibilities include haematomas and cystic hyperplasia.
- **Neoplasia** – multifocal poorly defined echogenic areas within the parenchyma; prostamegally with an irregular peripheral outline and occasional intraparenchymal calcification; and sub-lumbar lymphadenopathy.
- **Paraprostatic cyst** – smoothly marginated anechoic structure resembling a urinary bladder extending from the prostate gland. May be septated and may be accompanied by cysts within the prostatic parenchyma and occasional calcification of cyst wall.

Benign prostatic hyperplasia

Benign prostatic hyperplasia (BPH) is the most common prostate abnormality in intact male dogs and is estimated to occur in the majority of old, intact dogs. Androgens are essential for the development and maintenance of BPH. With aging, a decrease in the serum testosterone concentration, combined with no change in the serum oestradiol-17-beta concentration, results in a relative decrease in the serum androgen to oestrogen ratio.
This altered hormonal milieu is believed to contribute to the pathogenesis of BPH by increasing androgen receptor expression.

The diagnosis of BPH is made indirectly by elimination of other types of prostatic disease. A definitive diagnosis of BPH requires histopathology confirmation. It is difficult, however, to justify a prostatic biopsy when the results of less invasive methods strongly support the diagnosis of BPH and the absence of more sinister prostatic diseases. A presumptive diagnosis of BPH is based on the history, clinical signs, prostatic imaging, prostatic cytology, and culture. Clinical signs associated with BPH include haemorrhagic urethral discharge, haematuria, haemospermia, and tenesmus. Many dogs with BPH show no clinical signs and often only becomes a clinical problem once the enlarged prostate gland results in pressure on the colon or if it encroaches on the muscles and nerves in the pelvic canal.

For non-breeding males, the recommended treatment for BPH is castration with prostatic size significantly decreasing within 7-10 days after castration. Anti-androgenic drugs (progesterone, flutamide, finasteride, chloradinone acetate, osaterone acetate, delmadinone acetate, flutamide, and megestrol acetate) are effective at reducing prostatic size but can affect gonadal function. Oestrogens have also been used to treat BPH; however, because of the risk of myelosuppression and prostatic abscess formation, they are no longer recommended. The liposterolic extract of saw palmetto plant berries or the American dwarf palm tree (Serenoa repens) reportedly improves urinary flow rates in humans with BPH but have been shown to have little effect (positive or negative) in dogs.

Acute prostatitis

This is an acute inflammatory condition, which may occur with or without hyperplasia of the prostate. Bacteria most frequently isolated are E. coli, Proteus, Pseudomonas, Staphylococcus and Streptococcus. Prostatic infection can either be ascending from the lower urinary tract, descending from the kidney or bladder, or of haematogenous origin.

Analgesics and catheterisation may be indicated for urethral spasm, resulting in urine retention. Antibiotic selection is of paramount importance, with effective antibiotics being penicillins, cephalosporins, potentiated sulphonamides, and quinolones. The antibiotic choice should ideally be based on a culture and given for 4-6 weeks. Urine culture will frequently isolate the infective organism and should be repeated at the completion of therapy. As castration is effective in decreasing gland size, it should be considered with recurrent infections. Alternatively anti-androgenic drugs can be used.

Chronic prostatitis

This occurs less frequently than acute prostatitis, however, owing to the relative lack of clinical signs it is most likely under-diagnosed. It is often considered as a sequel to acute prostatitis.

Therapy is aimed at intensive antibiotic therapy, based on culture and antibiogram. Treatment is continued until the causative organism is eliminated, which can be determined by intermittent urine or prostatic fluid culture. Castration should be considered and prostatectomy only done in cases completely refractory to medical treatment.

Abscession
This is defined as a localized accumulation of pus within the parenchyma of the prostate. Abscesses may be small or large, focal or diffuse. Smaller abscess usually coalesce to form one or more larger abscess. Prostatic abscession has been associated with BPH, oestrogen therapy, and as an extension of acute prostatitis.

Conservative antibiotic therapy is often unrewarding. Castration is advocated in all cases to reduce gland size. The abscess can be treated by marsupialisation or by percutaneous drainage under ultrasound guidance.

**Paraprostatic cysts**

Paraprostatic cysts originate near the prostate and are remnants of the mullerian ducts. The cyst is not in direct communication with the prostate and can be completely removed via blunt dissection. Clinical signs of prostatic cysts are usually absent until the cyst becomes large enough to impinge on the bladder and/or rectum, leading to dysuria and constipation, respectively.

**Neoplasia**

Prostatic neoplasia is an uncommon condition with no breed predilection. The aetiology is unknown; however, the aging process and the associated hormonal imbalances appear to be of prime importance in prostatic carcinogenesis. In comparison to man squamous metaplasia has been shown not to be a pre-neoplastic change. Metastasis is common, usually developing in the sub-lumbar lymph nodes, lumbar vertebral bodies, and lungs.

The diagnosis of prostatic carcinoma is made based on the history, clinical signs, and results of prostatic imaging, fluid analysis, and histopathology. In intact dogs, prostatic carcinoma is not always associated with prostamegally. However, in castrated dogs, prostamegally is highly associated with prostatic carcinoma. On palpation, the prostate may be normal in size but may feel firm and asymmetric and adhered to the pelvic canal. Tumour markers used in human medicine, such as PSA, are not useful in the diagnosis of prostatic neoplasia in the dog.

The prognosis for dogs with prostatic neoplasia is poor. Treatment is primarily palliative and includes complete or partial resection of the prostate, castration, and intraoperative orthovoltage radiotherapy. Piroxicam, a specific cyclo-oxygenase-1 (COX-1) inhibitor, administered at a dosage of 0.3 mg/kg given orally once a day, has been used successfully to reduce the size of several canine carcinomas. Combining cisplatin with piroxicam appears to have a synergistic effect therapy.

**Prostatic calculi**

Calculi are seen infrequently as an incidental finding on routine abdominal radiographs and can be exogenous or endogenous.

Exogenous calculi originate in the bladder, lodge in the prostatic urethra and "burrow" into the parenchyma. They have no clinical importance. Endogenous calculi originate in the prostate, usually within a cyst, and is a very rare condition. If they are numerous they may lead to clinical prostatic disease, requiring surgical excision of the calculi via a prostotomy.