Federation of Boxer Clubs of Southern Africa
Breed Council

CRYPTORCHIDISM IN THE BOXER

Cryptorchidism is the failure of one (unilateral) or both (bilateral) testes to descend and be maintained in the normal position in the scrotum. The retained testis may be in the abdominal cavity, in the inguinal canal or just outside the scrotum. When only one testis is present in the scrotum, the term monorchid is sometimes used. This is incorrect – in a monorchid only one testis exists in the body, a condition which, like anorchidism (no testes), is extremely rare.

OCCURENCE

Cryptorchidism occurs in many species e.g. pigs, horses, goats, sheep and other mammals, but is most common in the dog. In dogs it is found more often in purebred dogs and small breeds are more at risk than large breeds. In breeds with different sized varieties, such as the poodle, schnauzer and dachshund, it occurs more frequently in the smaller (miniature) variety than its larger relatives. Breeds with short skulls have a higher incidence than normal and the risk for Boxers has been found to be the highest of all large breeds. Generally unilateral cryptorchidism occurs more often than the bilateral form.

WHAT EFFECT DOES AN UNDESCENDED TESTIS HAVE ON A MALE DOG?

REDUCED FERTILITY

Bilateral cryptorchids are sterile as spermatogenesis does not occur due to the raised temperature and the abnormal size and primitive morphology of the retained testes and epididymus (coiled segment of the spermatic ducts that serves to store, mature and transport sperm).
Some breeders argue that unilateral cryptorchids should not be regarded as abnormal, as they are often fertile. However, Badinand et al (1972) found that cryptorchidism results in lowered fertility and a variably lowered ejaculation reflex. 47 cryptorchid dogs were examined. Less than half of the unilateral cryptorchids ejaculated and 27% of these had no sperm in the ejaculate. Only 8% of the unilateral cryptorchids examined produced a normal volume of semen.

INCREASED RISK OF TESTICULAR TUMOURS

It is very well documented that the incidence of tumours (Sertoli cell tumours) is significantly higher (about 13.6x) in retained gonads than in normally descended testes. Further complications of testicular tumours are feminization, hair loss, blood dyscrasias and testicular torsion. Removal of the retained testes is therefore recommended before 4 years of age.

OTHER ASSOCIATED PROBLEMS

Cryptorchidism has been linked to other defects such as penile/preputial defects, umbilical hernia, inguinal hernia, hip dysplasia and particularly subluxation of the patella. Although both cryptorchidism and patellar subluxation are common in small breeds, there was also a strong association in breeds not normally at high risk for patellar subluxation. The association with inguinal hernia could be due to a defect of the gubernaculum leading to an abnormally large inguinal canal, combined with the absence of a spermatic cord to act as a plug for the opening (Cox 1986).

NORMAL DESCENT OF THE TESTES IN THE DOG

In mammals the sex of the individual is determined at fertilisation by the sex chromosome present in the sperm. If a Y chromosome is present, the embryo will develop into a male. An X chromosome will result in a female. However, early development of the genital system is indeterminate and similar in male and female embryos. The differences between the sexes arise when the undifferentiated gonads develop into ovaries in females, which remain in the abdominal cavity, or testes in males, which then have to migrate from the body cavity to their correct position in the scrotum.

About 53 days after conception the developing testes are located at the rear end of the kidneys in the male foetus. Each testis is attached to a cord of connective tissue, (the gubernaculums), which causes the testis to descend into the scrotum. During the first phase of descent, this cord grows through the abdominal wall, with the inguinal canal forming around it, and towards the future scrotal pouch. The gubernaculum contracts, which pulls the testis into the inguinal canal. Several foetal testicular hormones may be involved in the regulation of the first phase of testicular descent. The part of the gubernaculum immediately outside the abdominal wall forms a bulb. This outgrowth and swelling of the gubernaculum has been observed to continue until about five days after birth. The expansion of the bulb outside the abdominal cavity might add traction on the portion inside and help to pull the testes towards the inguinal ring. After the fifth day regression of the gubernaculum starts. Its jelly-like composition gradually becomes opaque and fibrous. The major part of the descent of the testes – passage from the inguinal ring to the scrotum – takes place during this regression phase of the gubernaculum.

The exact timing of the process of testicular descent may vary in dogs, depending on breed, just as the age at which puberty and enlargement of the testes starts, varies. However, in the majority of dogs studied, progress of normal descent of the testis occurred as follows:

- at birth the testis are about halfway between the kidney and the internal inguinal ring;
- it passes through the inguinal ring by about the 3rd or 4th day after birth;
- by 15-17 days after birth it reaches the halfway mark between the inguinal ring and the scrotum;
- by 35-40 days after birth it attains its final position at the lowest point of the scrotal pouch.

Initially the left testis always precedes the right one during the descent; probably due to the fact that the left kidney (and the accompanying developing testis) is situated further back) in the body cavity. It is interesting to note that in cryptorchid dogs the right testis is more frequently retained than the left. (The ratio is about 2:1). This could reflect a generally slower descent on the right side, due to the greater distance that that testis has to travel. In normal dogs the right testicle normally lies in front of the left in the scrotum.

In a week-old puppy the scrotum is filled with subcutaneous fat. This fat should disappear by about 25 days of age to make room for the testes. However, from 3-4 weeks of age onwards, “well-reared” puppies lay down considerable deposits of fat in the scrotal area, which may in some cases hinder the final descent of the testes.

Boxer pups conform to the “typical dog” described above and the testes of normal pups are usually in the scrotum by six weeks of age. If both testes are not fully descended by the time a Boxer pup is eight weeks old, he must be regarded as genetically suspect. Due to a highly effective reflex of the cremaster muscle, puppies up to 10 or 12 weeks of age may retract their testes into the groin area when picked up and examined. In normal pups, the testes can be pushed back into the scrotum easily, with light digital pressure. If a testicle tends to remain at the entrance to the scrotum and requires a good deal of traction to reach the correct position, particularly if it is then retracted again when released, this should be regarded as a minor degree of cryptorchidism.

CAUSES OF CRYPTORCHIDISM

Anything that impedes the initial outgrowth and swelling of the gubernaculum and its subsequent regression, will lead to cryptorchidism.

Anatomic examination of unborn pups has shown numerous abnormalities of the structures involved, eg underdevelopment of the gubernaculum or defects where it passes through the abdominal wall. Such abnormalities may cause some cases of cryptorchidism. However, autopsies on 7 bilateral cryptorchid adult males in a colony of research dogs (miniature schnauzers) revealed that a well-defined cord-like gubernaculum was present on the left side but indistinguishable on the right side in every case, although both testes were undescended (Cox et al, 1978). It has also been suggested that genetically influenced maldescent could be partly related to the rate of growth of the involved structures - the anatomy of the inguinal canal must permit the testis to pass through unimpeded (Hayes et al 1985)

The importance of hypophyseal hormone is reported in a study of mutant dogs, which showed a big variety of abnormalities, which included cryptorchidism. Malfunctioning of the pituitary, thyroid and adrenal glands have been mentioned, as has insufficient production of the interstitial cell stimulating hormone (ISCH). Histo-chemical analysis by Baumans, Dijkstra and Wensing (1981) indicated that active steroid synthesis and secretion might be important for the outgrowth of the gubernaculum.

INHERITANCE

Whatever the mechanisms involved, there is general agreement that the defect is an inherited trait, as indicated by the following evidence (Patterson 1983):

- It occurs more often in some breeds than in others.
- The frequency is higher in certain lines within breeds and in these lines, it increases with inbreeding.
- Studies in other species (pigs, goats) have shown that the incidence of cryptorchidism can be increased by using known carriers as parents and reduced by eliminating them from the breeding population.

In the colony of miniature schnauzers referred to above, deliberate inbreeding was practiced to increase the incidence and severity of another defect. The observation of the high incidence of cryptorchidism in the colony was coincidental and the study of the condition was retrospective. It provided good evidence for the hereditary nature of the condition, and morphological observation suggested a multiple gene defect.

The colony consisted mainly of the offspring of an inbred littermate pair. Over a span of four generations 12 cases of cryptorchidism (5 unilateral and 7 bilateral) were found in the colony. The degree of inbreeding was greater for the bilateral cases than the unilateral cases. The greater than normal incidence of bilateral over unilateral cryptorchids suggests that the severity of the condition increases with inbreeding. Increased severity was based on the following four criteria:

- The high incidence of bilateral cryptorchids.
- The more primitive structure of the epididymus found in the bilateral cryptorchids.
- The location of the testes in the bilateral cryptorchids – all the bilaterally retained testes were near the kidneys, while the unilaterally ectopic testes were lower down next to the bladder and one was in a prescrotal position.
- Abdominal retention rather than an inguinal or prescrotal position of the retained testes.

Regardless of the exact mode of inheritance, it must be accepted that both parents of a cryptorchid male are carriers, and that male and female siblings may also carry the genes responsible for the defect.

A heritability of 40% has been established for cryptorchidism in the Boxer, as well as a polygenic mode of inheritance. This signifies that environmental factors (which have not been identified) may be responsible for the remaining 60%.

HOW CAN THE INCIDENCE OF CRYPTORCHIDISM BE REDUCED?

Unfortunately the only way to entirely eradicate the problem would be not to use for breeding the cryptorchid dog, its parents, their parents and any of its siblings. Regrettably this would probably also eradicate the Boxer as a breed.

The main problem that breeders face in respect of genetic defects is the fact that the genetic base of the entire Boxer breed as it exists worldwide is quite limited. There is probably not a Boxer alive today which does not trace back to the four great German stud dogs – Sigurd von Dom and his three grandsons Utz von Dom, Dorian von Marienhof and Lustig von Dom. These dogs were widely used in Europe and thereafter exported to the USA. Unfortunately all four had cryptorchid progeny. The tight linebreeding to these dogs both in Europe and North America is probably why the incidence of cryptorchidism is such a problem in the breed today.

While there is evidence that inbreeding can increase the incidence of cryptorchidism, (Cox et al, 1978), with a defect as widespread as this is in the Boxer, Padgett (1998) has postulated that the risk is as high with outcrossed as with inbred pedigrees.

Unilateral cryptorchid males have been excluded entirely from breeding in Germany from as early as 1942. This did not reduce the incidence at all. In fact there was an increase in the incidence from 6.39% in 1941 to 10.21% in 1981 in East Germany. In West Germany it increased from 7.4% in 1959 to 14.2% in 1985. Breeders were encouraged to try and increase the heterozygosity of the breed by not only using the popular, top-winning stud dogs, but by breeding to lesser known males and to males in neighbouring countries. The results were disappointing. Some of the widely used stud dogs had percentages of above 20% and as high as 30%. After the reunification of Germany, the incidence increased further to 17.0% in 1995. In 1996 various quite severe measures to address the problem were instituted in Germany. A bitch was excluded completely from breeding if she produced cryptorchids in two litters, and the further use of males with more than 15% cryptorchids produced in a total of more than 20 male offspring, was restricted. This resulted in the prevalence of cryptorchidism coming down to 11.1% in 1998. This however had to be weighed up against the alarming fact that these and the numerous other existing breeding restrictions had further limited the genetic base of the Boxer in Germany to the extent that only 16% of the population could still be used for breeding. The initial reduction in cryptorchidism also did not last and slowly started rising again. In addition breeding activities lessened dramatically with the number of matings recorded falling from 597 in 1998 to 483 in 1999.

As a result all previous restrictions in respect of cryptorchidism and certain other inherited traits were revoked in Germany after the year 2000. The method already in operation for curtailing HD (Breed Value Assessment) was extended to include cryptorchidism. Breeding partners are selected on the basis of a system where the risk of producing affected offspring is calculated for every dog and bitch to be used for breeding, based on all available information for that animal, its parents, siblings, halfsibs, as well as its progeny. The combined figure for a proposed breeding pair may not exceed a specified threshold value (105 for cryptorchidism).

Breeding prohibition was replaced by breeding recommendations. In this way they hoped to gradually improve the incidence of the inherited defects without further restricting the genetic base of the breed. By 2002 breeding activities had shown a slight increase and the incidence of cryptorchidism had gone down to 10%. Unfortunately, the gene pool remains very limited and the degree of inbreeding very high.

Just to complicate matters further, a study carried out in the Netherlands (Janneke et al 2009) using data of 11 230 litters in 12 purebred dog breeds, found that litters produced by two parents who were both cryptorchidism carriers had an increased number of males (all breeds), a reduced number of females (8 breeds) and an increased litter size (11 breeds) when compared to litters from non-carrier or mixed parents. They concluded that a mechanism exists in the dog species which causes both cryptorchidism as well as increased male/female ratios and increased litter sizes. A consequence would be that selection for bigger litters frustrates selective efforts to eliminate cryptorchidism.

CONCLUSIONS

In South Africa with its small Boxer population, it would obviously be very dangerous and foolish to base the selection of breeding partners entirely on one or two aspects. A Boxer does not consist of testes or hips or a fawn coat only. Most knowledgeable breeders would list correct type, sound conformation and a steady disposition as their main concerns. The absence of serious health problems, such as heart defects and hip dysplasia, are also regarded as important, with some breeders placing a premium on working ability. Genetic defects, while a problem, can however never be the only selection criterion for our Boxers.

In respect of cryptorchidism, perhaps in our circumstances we should heed the advice of Fred Lanting (2001):

"At any rate, the unilateral condition at least is so widespread in many families and breeds that an all-out effort to combat it would take our minds and efforts away from more serious disorders, which would consequently increase. It's just a minor thing we have to put up with. Simply remove the affected ones from the gene pool and shift your preference away from breeding any (including females) that come from litters in which the defect occurred.

Since cryptorchidism is sex-limited (only affected males, not carrier females, show it) it is likely to persist at about the same prevalence in the breed for a long, long time."

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