CRYPTORCHIDISM IN DOG

(Criptorquidismo no cão)

Marcos Renato Franzosi MATTOS*, Lucilene SIMÕES-MATTOS & Sheyla Farhayldes
dOMINGUES

College of Veterinary Science, University of Ceará, Fortaleza, CE, Brazil

ABSTRACT

The present review aims to discuss cryptorchidism in dogs, accentuating the importance, incidence, pathogenesis, pathology, macroscopic and microscopic appearance of cryptorchid testicles, complications and disturbances, diagnosis, treatments and control. The zootechnical and ethical aspects related to this disturbance are also discussed.

KEY WORDS: cryptorchidism, dog, pathology, diagnosis, control, treatment

INTRODUCTION

Cryptorchidism is of major importance among the pathologies that affect reproduction in dogs, as well as other animal species. It is characterized by the failure in the descent of one (unilateral) or both (bilateral) testis from the abdominal cavity to the scrotum (BURKE, 1986; MICKELSEN & MEMON, 1995). Besides the failure in the descent of the testis, BURKE (1986) suggests that cryptorchidism may also be the failure to maintain the normal position of the testis in the scrotum. For PINTO (1962) and FERREIRA (1986), the term “cryptorchidism” is a combination of three Greek words, “kriptós” (hidden, occult), “orchis” (testicle) and “idion” (small, diminutive). The terms monorchidism (from greek mónos = only, alone) and anorchidism (from greek anorchos = without testis) are more correctly applied for the congenital absence of one (monorchidism) or both (anorchidism) testis in the body. These are extremely rare anomalies among domestic mammals (NASCIMENTO & SANTOS, 1997). Unfortunately, the term “monorchidism” is often used erroneously, with reference to the unilateral cryptorchidism (BURKE, 1986, COX, 1986, MIALOT, 1988, DAELS et al., 1991; NELSON & COUTO, 1994; HAFEZ, 1995; SORRIBAS, 1995; NASCIMENTO & SANTOS, 1997). ALLEN (1995) reported a single case of anorchidism in dogs.

In several species, when the testicle is not in its normal position in the scrotum, it can

* Autor para correspondência
e-mail: mattos@latinmail.com

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be present in different locations, including the abdominal cavity, inguinal canal and subcutaneous tissue. When the testis is located in the subcutaneous position, it can be found in the perineal and inguinal regions, in the medial part of the posterior members, around or inserted in the prepuce. Many authors differentiate between abdominal and inguinal positions of subcutaneous location, considering as cryptorchidism only the former two and naming the subcutaneous localization as testicular ectopy (MIALOT, 1988; NIEMAND & SUTER, 1992; NASCIMENTO & SANTOS, 1997). As the etiology, pathogenesis, symptoms, pathology and treatment are similar for all locations, this review will not enter that description and will consider testicular ectopy and cryptorchidism as synonymous.

**Incidence**

According to HAFEZ (1995), cryptorchidism incidence is greater in swine and equines than in other domestic animals. However, according NASCIMENTO & SANTOS (1997), while the most affected species are the equine and canine, it may occur less frequently in other species. MIALOT (1988) reports that the canine species has the highest frequency of this pathology (10% of adult animals) and that the tendency is increasing. MICKELSEN & MEMON (1995) and ACLAND (1998) suggest that cryptorchidism is the most common disorder of the sexual development in dogs, occurring in 13% of the males. NELSON & COUTO (1994) describe a smaller prevalence in dogs and cats that is about 1% to 2%. COX (1986) reports frequencies from 0.8% to 10.9%. There is breed predisposition in clinical practice as in the literature. COX (1986) mentions those breeds that are most predisposed to cryptorchidism are the Yorkshire terrier, Poodle, the smaller variants being most affected (toy > miniature > standard). The author also mentions that the Boxer and the Beagle breeds are of relative risk. NELSON & COUTO (1994) include the breeds Chihuahua, German Spitz, miniature Schnauzer, Pekinese, Maltese, Shetland Shepherd and Cairn terrier among the most affected dogs. These authors also report that cryptorchidism may either happen in normal males or intersex animals (male pseudohermaphrodites). BROWN et al. (1976) describe the occurrence of male pseudohermaphrodites associated with the unilateral or bilateral cryptorchidism and testicular neoplasia (Sertoli cells) in three miniature Schnauzers. These animals presented clinical signs of hyperestrogenism, and a cystic endometrial hyperplasia. In cats, the Persian breed is mentioned with a larger prevalence of cryptorchidism (NELSON & COUTO, 1994).

According to NASCIMENTO & SANTOS (1997), unilateral cryptorchidism is more frequent than bilateral. According to MIALOT (1988), the right testicle seems to be more frequently affected. He supposes that is because embryologically, there is a longer migration (since they are originally located at the caudal region of the kidneys, and the right kidney is more cranial than the left one). Similarly, CHRISTIANSEN (1986) suggests that the lack of descent, in dogs, is found twice as often on the right side as on the left (with a relationship of 2.3:1, for testis retained in the inguinal region, and 2:1 for abdominal retention). HAFEZ (1995) reports that, in several other animal species, the left unilateral cryptorchidism is more frequent. According to NELSON & COUTO (1994) there is no difference between the prevalence of left or right cryptorchids in dogs and cats.

**Embryology and fetal development**

In most mammals, the testis originate from the caudal portion of the kidneys, in the abdominal cavity, pass across the inguinal canal, and descend to the scrotum during the fetal period (SETCHELL, 1978, apud KAWAKAMI, et al. 1993). The descent of the testis is induced by the contraction of a gelatinous cord, the gubernaculum testis (Fig. 1), which extends from the caudal pole of the testis to the genital tubercle (COX, 1986). The exact function of the gubernaculum in testicular descent is not clearly elucidated. With the degeneration of the mesonephros (transitory kidney), the gubernaculum is attached in the caudal portion of the testis, extends through the inguinal canal and
down to the scrotum (MOORE & PERSAUD, 1994). The distal portion of the gubernaculum is called the gubernacular bulb. It is sometimes thought that the expansion of the gubernacular bulb out of the abdominal cavity (Fig. 2) may contribute to the traction that moves the testis caudally (COX, 1986).

Descent of the testis

The normal period for the descent of the testis to the scrotum is not strictly established in dogs or in cats. According to NASCIMENTO & SANTOS (1997), the testicles are usually present in the scrotum at birth in dogs. NELSON & COUTO (1994), NELIS (1995) and MICKELSEN & MEMON (1995) propose that in this species, the descent usually occurs at about 10 days of age. SORRIBAS (1995) mentions one month after birth, while ALLEN (1995) and MIALOT (1988) observed it within 10-12 weeks of age, and NIEMAND & SUTER (1992), at nine weeks. There is a breed variation (NELSON & COUTO, 1994; NASCIMENTO & SANTOS, 1997). In dogs, the testis are palpable in most of the cases from 4 to 5 weeks and usually within 6 to 8 weeks after birth (CHRISTIANSEN, 1986; MICKELSEN & MEMON, 1995; NASCIMENTO & SANTOS, 1997). However, the diagnosis of cryptorchidism in dogs is not really certain before 6 months of age (CHRISTIANSEN, 1986).

Pathogenesis

The causes of cryptorchidism may be genetic, anatomical or endocrine. However, these causes are still controversial and may be intrinsically related. The genetic origin has been incriminated countless times, since the condition has been frequently observed in inbred dogs (MIALOT, 1988). The most often accepted hypothesis attributes it to an autosomal sex-linked gene (ROMAGNOLI, 1991; MICKELSEN & MEMON, 1995), recessive in all examined species (BURKE, 1986, MIALOT, 1988,
ROMAGNOLI, 1991; NELSON & COUTO, 1994; ALLEN, 1995; HAFEZ, 1995; MICKELSEN & MEMON, 1995), except in equine, in which it is due to a dominant autosomal gene (HAFEZ, 1995). Males and females carry the gene and can transmit the pathology to their offspring (NELSON & COUTO, 1994; MICKELSEN & MEMON, 1995; NASCIMENTO & SANTOS, 1997). So it is important to realize that normal animals can transmit the pathology due to its recessive character (MICKELSEN & MEMON, 1995).

The monogenic theory does not explain the existence of right, left or bilateral cryptorchidism in dogs, and neither does it explain the disagreement between theoretical and clinical frequencies of the anomaly. To remedy these contradictions, a theory was proposed supposing the intervention of two genes, one for each testicle. The dominant allele would allow the migration, while the recessive allele would lead to the retention of the testis. Some studies emphasize the existence of a simple autosomal factor, but do not discard the polygenic hypothesis of double entrance. This hypothesis considers that, even if the cryptorchidism is determined by larger genes, its severity appears to be related to environmental factors, as well as to the action of modifier genes. Following this hypothesis, the genetic determinism is not necessarily constant in many cases (MIALOT, 1988). For NASCIMENTO & SANTOS (1997), three mechanisms can be responsible for the abnormal descent of the testis: 1) lack of development of the gubernaculum; 2) abnormal development of the gubernaculum, which results in an alteration of its normal position; 3) excessive growth of the testicle and absence or retardation in the regression of the gubernaculum. MIALOT (1988) reports an anatomical etiology, which can be linked to the reduction of the gubernaculum (due to as abnormal insertion) and combined with the narrowing or closing of the inguinal canal. Furthermore, NIEMAND & SUTER (1992) propose other factors such as the occurrence of adherences of the spermatic cord, hormonal alterations from the frontal lobe of the pituitary and large testicular size. On the other hand, ACLAND, (1998) mentions testicular hypoplasia as a predisposing factor of cryptorchidism. The same author also reported parturition with the male fetus in posterior presentation as compromising the blood supply to the testis and delay in the closing of the navel, causing retardation in the capacity for increasing the abdominal pressure.

The endocrine origin of cryptorchidism is not well-known (MIALOT, 1988) but seems to be related to a testosterone deficiency (NASCIMENTO & SANTOS, 1997) either due to a malfunction by the testis or abnormal testis. Normal descent is dependent on a normal testis during fetal life and removal of fetal testis results in no gubernaculum development. The testis removal 0-3 days post partum results in failure of the gubernaculum to regress (NIEMAND et al., 1972). It may be a pituitary hormone deficiency. The Müllerian inhibiting substance (MIS) hormone, a non-androgenic factor, secreted by the Sertoli cells of the testis and a growing abdominal pressure at the time of the development of the organs, could play an important part in testicular descent (KAWAKAMI et al., 1993). The follicle stimulating hormone (FSH) and the luteinizing hormone (LH) probably have an important function in the descent of the testis, because they are important in the differentiation process and function of the Leydig and Sertoli cells (NASCIMENTO & SANTOS, 1997). However, it is believed that androgens secreted by the fetal testis are the most decisive factors to testicular descent (KAWAKAMI et al., 1993).

Macroscopic and microscopic appearance
The macroscopic aspect of the cryptorchid testis is normal before puberty; after sexual maturation, the testis becomes progressively smaller and fibrotic (ACLAND, 1998). According to COX (1986), cryptorchid testicles are always small and soft, especially when they are in abdominal position. However, the normal size testis in abdominal bilateral cryptorchid is reported in animals at six years of age (MATTOS et al., 2000). Histologically, ACLAND (1998) reported interstitial deposition
of collagen, hyaline thickening of the basal membranes and atrophy of the germinative epithelium and only some spermatogonia remained together with the Sertoli cells (Figure 3). COX (1986) described only one single layer of spermatogonia, primary spermatocytes and Sertoli cells. The same author mentioned that the Sertoli cells are characterized by increased Golgi’s complex and smooth endoplasmic reticulum (site of steroidal synthesis) and decreased number of lysosomes. ACLAND (1998) reported that the interstitial cells seem relatively more numerous than in the descended testis, and that the differentiation of the epididymides were coordinated by the testicular descent. An epididymidal differentiation could be, consequently, late in some cases of cryptorchidism.

Complications

According to MIALOT (1988), cryptorchidism can cause various complications in relation to the individual’s age and the type of testicular ectopy, such as: sterility, behaviour disturbances, neoplasia, local pain and skin diseases. He also mentions that abnormalities have been seen in the eutopic testis, as well as gland atrophy and hyposexualism (reduction of size of the prepuce and penis). The normal position of the testis, outside the abdominal cavity and inside the scrotum, allows maintenance at a temperature lower than body temperature (JUNQUEIRA & CARNEIRO, 1990). NELSON & COUTO (1994) explained that the spermatogenesis of a cryptorchid testis, especially when abdominal, is totally absent, because the temperature is too high for this function, but, if the testicle is inside the scrotum, the spermatogenesis is generally normal, in line with other authors (BURKE, 1986; CHRISTIANSEN, 1986; COX, 1986; MIALOT 1988; JUNQUEIRA & CARNEIRO, 1990; ROMAGNOLI, 1991; NELSON & COUTO, 1994; NIEMAND & SUTER, 1992; ALLEN, 1995; HAFEZ, 1995; SORRIBAS, 1995; ACLAND, 1998). PINART et al. (1997) report that unilateral abdominal cryptorchidism in male goats can cause disturbances at the end of spermatazoa maturation during spermiogenesis of the normal testis, but not in the epididymal maturation process. In experimental unilateral cryptorchidism, no spermatogenesis was observed in the cryptorchid testis and the number of germ cells in the contralateral testis had decreased 52 weeks later (KAWAKAMI et al., 1999). They suggest that a large quantity of estradiol-17-beta secreted by the cryptorchid testis inhibits the endocrine and spermatogenic

Figure 03. Histological appearance of normal (left side) and cryptorchid (right side) testis in dog. Observe the germinative epithelium atrophy in cryptorchid testis (arrow).
functions of the contralateral testis in the dog. BADINAND et al. (1972) observe that less than 50% of unilaterally cryptorchid dogs were able to ejaculate, and that 69% of these animals which can ejaculate, do not have spermatozoa in their semen. The same authors mention that the fructose and citric acid concentrations of the seminal plasma are increased, while the lactic acid concentration is reduced. The acid phosphatase level is higher than in the normal animals.

Despite the finding of azoospermia and decrease in the ejaculate volume in the unilateral cryptorchid dogs, we feel that on the contrary, the unilateral cryptorchid animal is generally able to mate and produce offspring, despite their lower sperm concentration. Regarding the reduction of the secondary sexual characteristics and libido, a high number of animals it is commonly observed that, although being unilaterally or bilaterally cryptorchid, normal sexual behaviour and libido are mentioned. According to JUNQUEIRA & CARNEIRO (1990), NELSON & COUTO (1994), NIEMAND & SUTER (1992) and ALLEN (1994), the interstitial cells continue to produce testosterone, thus inducing the secondary sexual characteristics and normal libido which can even be increased, according to NELSON & COUTO (1994), due to a disarray in the negative feedback. MATTOS et al. (2000) report a natural case of a bilaterally cryptorchid dog that presented high libido. They observed the absence of libido after six months of cryptorchidectomy.

In humans where the testicle is surgically moved and placed in the scrotum at a young age, the spermatogenesis returns to normal, because the spermatogonia are still normal (JUNQUEIRA & CARNEIRO, 1990). However, in animals, this procedure is not recommended.

When cryptorchidism is bilateral, the animal is sterile (BURKE, 1986; MIALOT, 1988; DAELS et al., 1991; ROMAGNOLI, 1991; NELSON & COUTO, 1994; ALLEN, 1995; HAFEZ, 1995; MICKELSEN & MEMON, 1995; NASCIMENTO & SANTOS, 1997). There is a certain tendency to obesity, sudden and transitory aggressiveness, nervousness or apathy in these animals (MIALOT, 1988). Neoplasia in the ectopic testis are more frequent than in the eutopic, being Sertoli cell tumors and seminomas mainly, and they generally appear in animals of six to ten years of age (ROMAGNOLI, 1991; NIEMAND & SUTER, 1992). NASCIMENTO & SANTOS (1997) mentioned a risk ten times greater for a retained testicle to develop neoplasia compared to a normal one. This is similar to 9.2 to 13.6 reported by COX (1986). ACLAND (1998) added that there is a tendency for an increase in tumor incidence with the severity of the retention. The same author reports that in dogs, Sertoli cell tumors occur with a greater probability in testis retained in the abdomen, while inguinal testis tend to develop seminomas. REIF et al. (1979) reported the double incidence of testicular neoplasia in inguinal retention compared to the abdominal situation. According to HAYES & PENDERGRASS (1976), seminomas and Sertoli cell tumors occur at the same frequency in cryptorchid as normal dogs, but for REIF & BRODEY (1969), the apparition of these neoplasias is more precocious in the retained testis. The Sertoli cells tumors are more common in Poodles, German Spitz, Yorkshire Terriers, Pekineses, English Bulldogs, miniature Schnauzers, and Old English Sheepdogs (MICKELSEN & MEMON, 1995). Some of the testicular neoplasias are hormonally active, causing alopecia and feminization syndrome in the males. This occurs in 40 % of the cases of Sertoli cells tumors in dogs (COX, 1986). According to the author, this fact may be associated with an increased smooth endoplasmatic reticulum of the Sertoli cells in the retained testis, which are the site of steroidal synthesis, and more precisely for oestradiol production. The skin disorders, according to MIALOT (1988), are caused by hormonal imbalance due to the degeneration of the testicle. KAWAKAMI et al. (1999) report low levels of testosterone and high levels of estradiol-17-beta in experimentally unilaterally cryptorchid testis. MATTOS et al. (2000) report high levels of estradiol in the serum of a natural case of a bilaterally cryptorchid dog. This hormonal imbalance may be the reason of skin and prostatic disorders verified in old cryptorchid dogs.
According to MIALOT (1988), local pain symptoms are rare, but can occur, taking the form of urination difficulties or limping due to a torsion of the spermatic cord, generally associated with a testicular neoplasia. NIEMAND & SUTER (1992) and ROMAGNOLI (1991) reported that it is relatively frequent when there is a torsion of the cord and strangulation of the intestinal loop. COX (1986) also mentioned a blood dyscrasia as a complication of the cryptorchid condition.

**Diagnosis**

Clinical diagnosis is generally easy, by inspection and palpation. By inspection, the absence of one or two testis in the scrotum is diagnostic. By palpation, the testicle can be felt between the scrotum and the inguinal canal, in the subcutaneous tissue or can be non-palpable. By this procedure, the small size of the ectopic testicle may be assessed (MIALOT, 1988). However, especially in the first months, the testis can have a normal size. According to MIALOT (1988), palpation is sometimes difficult; a pressure exercised on the abdomen may allow the testicle to appear, if it is in the inguinal canal. He also cites as characteristic of a retained testis, the mobility, the presence of the epididymides, the smooth surface and firm consistency. In obese animals, palpation and even the ultrasound examination are difficult and do not allow the localization of the retained testicle. The diagnosis can only be accomplished after ten weeks of age (MIALOT, 1988) or even six months (COX, 1986).

CASTILHO (1990) reported that in man the non-palpable testis represents 20% of cases of cryptorchidism. He mentioned that several methods are used for diagnosis of the cryptorchid testicle including ultrasonography, hormonal evaluation, pneumoperitoneography, herniography, venography, arteriography, computerized tomography, magnetic resonance imaging, laparotomy and laparoscopy, indicating that the ideal method of diagnosis and localization does not really exist.

MADRAZO, et al. (1979) cited by MALONE & GUINEY (1985) mentioned that in humans, the ultrasound is a difficult method of diagnosis for abdominal testis because of the great amount of gas in the intestines associated to the small size of the retained testicle. MALONE & GUINEY (1985) recommended diagnosis by ultrasound for non-palpable testis, only when they are in the inguinal canal, thus avoiding invasive procedures such as laparoscopy. WEISS & SEASHORE (1987) cite that in man, a careful physical exam has been more efficient than ultrasonography. Laparoscopy in humans has been recommended as a simple and more efficient technique for localizing non-palpable testis (MALONE & GUINEY, 1985; WEISS & SEASHORE, 1987).

**Treatment**

There are two forms of treatment for cryptorchidism in dogs: hormonal and surgical.

**Hormonal**

The hormonal treatment of cryptorchidism in dogs (table 01) is based on

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<th>Hormone</th>
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<tr>
<td>HCG</td>
<td>35 IU/Kg in six applications, with a two days interval*</td>
</tr>
<tr>
<td>GnRH</td>
<td>50 to 100mg/animal for four days and after continue with a series of six applications of 25 to 50mg/animal with a two days interval*</td>
</tr>
<tr>
<td>ECG</td>
<td>30IU/Kg once a week for two months**</td>
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human treatment presenting various results. These treatments are recommended in the two or three first months of life of the animal. Their goal is to aid the descent of the testis, especially when they are in the inguinal or inguinal-scrotal position.

Testicular hypertrophy may be observed with the use of GnRH, followed by a definitive migration. According to NIEMAND & SUTER (1992), some authors still mention the use of testosterone, however they do not recommend androgen due to an excessive response of the negative feedback for gonadotrophin liberation, and due to the premature closing of the growth cartilage in young animals. The same authors also reported a good prognosis for this therapy in the case of inguinal cryptorchidism, but a more uncertain prognosis in abdominal cases.

The indication for hormonal treatment is controversial, and some authors reported an efficacy, which tends to be small or nil (BURKE, 1986; ROMAGNOLI, 1991). It is maybe because the hormonal treatment only speeds up the descent of the testis, which was already descending to the scrotum any way, and, thus only accelerates a late descent. Moreover, many authors do not recommend the hormonal treatment because of the risk of hereditary transmission (COX, 1986; MIALOT, 1988; ROMAGNOLI, 1991; NELSON & COUTO, 1994; ALLEN, 1995 and MICKELSEN & MEMON, 1995). The use of hormonal treatment for the descent of inguinal, inguinal-scrotal or abdominal cryptorchid testicles can be recommended with great restrictions and only if necessary. This procedure can facilitate a posterior orchiectomy, to avoid much more complex surgical procedures, such as laparotomy mainly in animals, in which surgery presents a risk.

It should be clear that this hormonal treatment may be carried out only by veterinarians who understand the seriousness of the case, and the customers’ honesty, avoiding the dissemination of this anomaly.

**Surgical**

Surgical treatment should not be undertaken in dogs under six months old because of the possibility of a late descent (COX, 1986). In the surgical treatment, the traction on the testicle through the scrotum and its fixation (orchiopexy) is not recommended for zootechnical reasons (NIEMAND & SUTER, 1992). BURKE (1986) commented that orchiopexy is a contravention of veterinary ethics. In addition this procedure may harm the blood supply to the tissues by stretching of vessels (MIALOT, 1988). The recommended surgical treatment consists of cryptorchectomy (COX, 1986; MIALOT, 1988; NELSON & COUTO, 1992; ALLEN, 1995; SORRIBAS, 1995). Two reports were found on the use of laparoscopic for cryptorchectomy in dogs (GIMBO et al., 1993 and PENA et al., 1998). These authors affirmed that laparoscopy allows the orchiectomy in a less traumatic manner for the animal than a classic laparotomy. The retreat of the testicle in a normal position (eutopic) is recommended to prevent the crossing and dissemination of the undesirable character (ALLEN, 1995). It is realized by ablation of the normal testicle or by the section of the vas deferens canal (vasectomy). Vasectomy is reserved for young animals, and the intervention can be made by inguinal or abdominal ways, adapted to the position of the ectopic testicle (MIALOT, 1988). The vasectomy of the normal testicle associated with the orchiectomy of the ectopic testis, is valuable or choice alternative for preventing the dissemination of the characteristic without displeasing the animal owners, since there is great opposition by owners to a radical orchiectomy. Complete castration presents some benefits to the animal’s health that the vasectomy does not, such as prevention of prostatic and skin disorders, anal dysplasia and carcinomas, because the source of the sex steroid hormone was removed.

It is important to notice that the testis, which suffered orchiopexy and/or hormonal treatment, may present a greater risk for
developing a neoplasia.

Control

In agreement with what has been previously discussed, it becomes clear that the best control of cryptorchidism is surgical. The complete castration of these animals is the best choice because of the health benefits quoted previously. The veterinarians have to know about the importance of this anomaly, motivating the retreat of the reproduction of unilateral and bilateral cryptorchidic animals, due to the possibility of hereditary transmission of this illness. MICKELSEN & MEMON (1995) firmly recommend not to use in breeding, relatives of the cryptorchid animal, which may transmit this character, even if it is with decreased frequency. ROMAGNOLI (1991) affirmed that the control of animals used in reproduction is highly efficient to decrease the incidence and to control this pathology.

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