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Cryptorchidism in Horses. Part I. Anatomy, Causes, and Diagnosis

Auburn University
Dwayne H. Rodgerson, DVM
R. Reid Hanson, DVM

FOCAL POINT

- In horses, failure of one or both testes to descend into the scrotum represents a common developmental defect and a diagnostic challenge.

KEY FACTS

- Abdominal and inguinal retention are most often associated with the left and right testes, respectively, p. 1280.
- Left- and right-sided retention are reported with nearly equal frequency, p. 1280.
- The process of descent of the testes is complex and not completely understood but is believed to be controlled by genetic, hormonal, and mechanical factors, p. 1284.
- Gonadotropin-induced androgen stimulation is believed to influence descent of the testes, p. 1284.
- Diagnosis of cryptorchidism in a horse with an unconfirmed castration history may require a diagnostic workup, p. 1285.

Cryptorchidism, or failure of one or both testes to descend into the scrotum, is a common developmental defect in horses.[1–4] Affected horses are sometimes referred to as rigs or ridglings.[1,5,6] Like normal stallions, horses with a retained testis frequently have undesirable behavioral characteristics.[1–3,6,7] Cryptorchid stallions are generally diagnosed as having a retained testis during inspection of the scrotum at the time of castration or during examination before or after sale.[8] It is a challenge to confirm or diagnose cryptorchidism in a mature horse that is purported to be a gelding and that exhibits stallionlike behavior; an unconfirmed history of castration will complicate the process.

PREVALENCE

In a retrospective study at 16 veterinary teaching hospitals in North America, one of every six 2- to 3-year-old referred colts was cryptorchid.[9] The prevalence of cryptorchidism was highest in Percherons, followed by American saddle horses and Quarter Horses.[9] The overall prevalence in Thoroughbreds and Standardbreds was lower than that in other breeds.

Although both testes may fail to descend into the scrotum, unilateral retention is nine times more prevalent than bilateral retention.[10] Studies indicate that the frequencies of left- and right-sided retention are nearly equal.[5,10–12] The testis was within the abdomen in 75% of horses with left-sided retention but in only 42% of horses with right-sided retention.[10] Inguinal retention of the testis was most often associated with the right testis (58%, versus 25% for the left testis).[10]

The incidence of inguinal retention decreases with age, whereas the incidence of abdominal retention increases with age.[11–13] The overall frequency of bilateral retention of the testes is reportedly 9% to 14% of all cases of cryp-

torchidism.[10,12] Bilaterally retained abdominal testes were nearly 2.5 times more common than were bilaterally retained inguinal testes.[10]

Left-sided unilateral testicular retention was present in 42 of 77 (55%) cryptorchid horses evaluated at the Veterinary Teaching Hospital at Auburn University during the last 5 years. Twenty-five (32%) of these horses had right-sided unilateral retention. Abdominal retention occurred in 32 of 42 (76%) horses with left-sided retention and in 6 of 25 (24%) with right-sided retention. Inguinal retention was documented in 76% (19 of 25) of the horses with right-sided retention and in 24% (10 of 42) of those with left-sided retention. The incidence of bilateral retention was 13% (10 of 77).

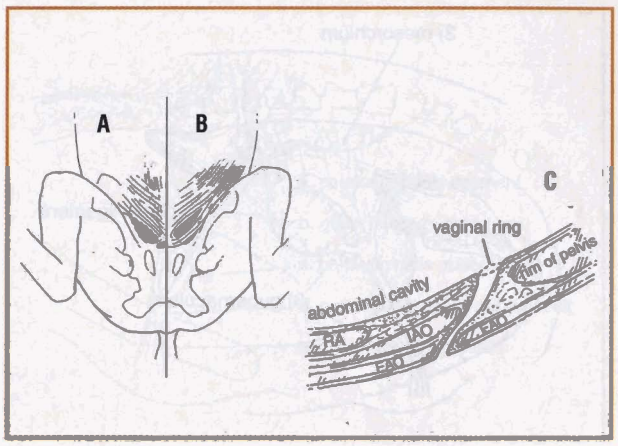

Figure 1—(A) The deep inguinal ring is a triangular space bordered cranially by the caudal edge of the internal abdominal oblique muscle (*IAO*), caudally by the pelvis and inguinal ligament, and ventromedially by the rectus abdominis muscle (*RA*) and prepubic tendon. (B) The superficial inguinal ring is a slit in the aponeurosis of the external abdominal oblique muscle (*EAO*), slightly cranial to the deep inguinal ring. (C) A sagittal section through the inguinal canal demonstrates the relationship between the vaginal ring and the deep and superficial inguinal rings. The vaginal tunic traverses the deep and superficial inguinal rings and surrounds the testis in the scrotum.

ANATOMY AND DEVELOPMENT OF THE TESTES

The testes move from the abdomen into the scrotum by passing through the right or left inguinal canal. The inguinal canal is a slitlike passage formed between the superficial and deep inguinal rings.[14,15] Compared with that of other domesticated species, the inguinal canal of a horse is relatively long (average length, 10 cm).[4,14]

The superficial inguinal ring is a slit in the aponeurosis of the external abdominal oblique muscle[14,15] (Figure 1). The direction of the superficial inguinal ring is parallel to the course of the fibers in the tendon of the external abdominal oblique muscle. The superficial inguinal ring is external to and slightly cranial to the deep inguinal ring,[14] which is a triangular space bordered cranially by the caudal edge of the internal abdominal oblique muscle, caudally by the pelvis and inguinal ligament, and ventromedially by the rectus abdominis muscle and prepubic tendon.[14,15]

The vaginal ring is internal to the deep inguinal ring and is considered to be separate from the inguinal canal[14] (Figure 1C). The vaginal ring is the opening of the vaginal tunic; it demarcates the division between the peritoneal cavity and the cavity of the vaginal tunic.[14,15] The vaginal tunic traverses the deep and superficial inguinal rings and surrounds the testes in the scrotum.

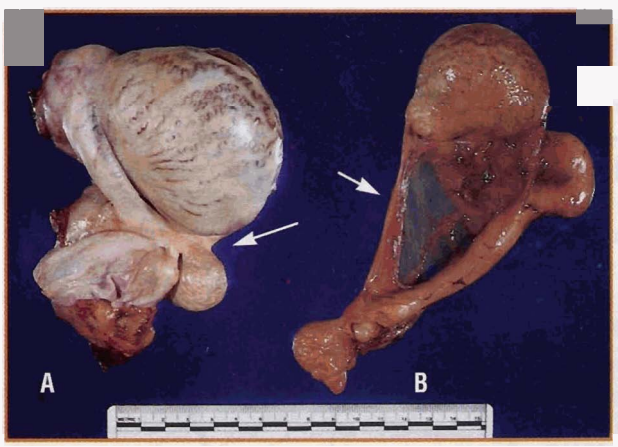

Figure 2—Association between the testis and epididymis. (A) In the normal scrotal testis, the tail of the epididymis is closely attached to the caudal pole of the testis by the proper ligament of the testis (*arrow*). (B) the cryptorchid testis, the proper ligament of the testis is often elongated, giving the testis and epididymis varying degrees of association (*arrow*).

Testicular and Epididymal Attachments

The paired equine testes are normally situated in the scrotum with their long axis in a horizontal direction.[2,16] The head of the epididymis joins the cranial aspect of the testis. The body of the epididymis is situated on the dorsolateral aspect of the testis. The tail of the epididymis is loosely attached to the caudal pole of the testis by the proper ligament of the testis (Figure 2A) and to the vaginal tunic by the ligament of the tail of the epididymis. The vaginal tunic is attached to the scrotum by the scrotal ligament.[2]

In cryptorchid stallions, the retained testis and the tail of the epididymis can have varying degrees of association.[4,17,18] With cryptorchid testes, the proper ligament of the testis, the ligament of the tail of the epididymis, and the scrotal lig-

ament are often elongated (Figure 2B).

The Fetal Testis

The equine gonads originate in the dorsolumbar region of the embryo at approximately day 27 of gestation. Sexual differentiation of the gonads into testes or ovaries does not occur until day 40.[19–22] Gonadal development entails the merging of three separate embryonic tissues: the primitive germ cells, the intermediate mesoderm, and the early embryologic mesonephric kidney.[21] The primitive germ cells line the inner surface of the mesonephric tubules in the cranial portion of mesonephric kidney; the primitive germ cells of the male gonad evolve into the spermatogonia of the testis. The extensive network of mesonephric tubules in this portion of the mesonephric kidney gives rise to the seminiferous tubules of the testis. The mesonephric duct of the associated mesonephric kidney evolves into the epididymis and ductus deferens. The surrounding intermediate mesoderm becomes the stroma or interstitium of the testis.[21]

The equine fetal testis is unique in that it changes dramatically in size and shape throughout gestation.[17,19–22] At day 150 of gestation, the testis is cylindric, weighs approximately 20 g, and is 3 cm in diameter.[19] By day 250, the fetal testis has enlarged to approximately 5 cm in diameter and weighs 50 g. At day 300, it is elliptic, weighs 30 g, and has decreased to approximately 2.5 cm in diameter. These changes are related to the early proliferation and subsequent degeneration of the fetal interstitial cells.[17,19]

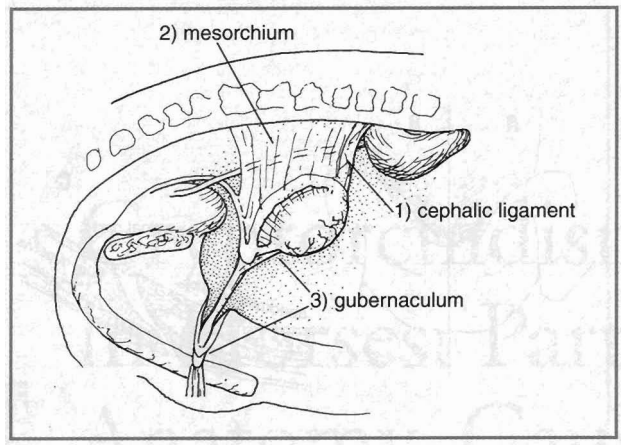

Figure 3—The attachments of the fetal testis—(*1*) cranial, the diaphragmatic or cephalic ligament; (*2*) dorsal, the mesorchium; and (*3*) caudal, the gubernaculum or posterior gonadal ligament.

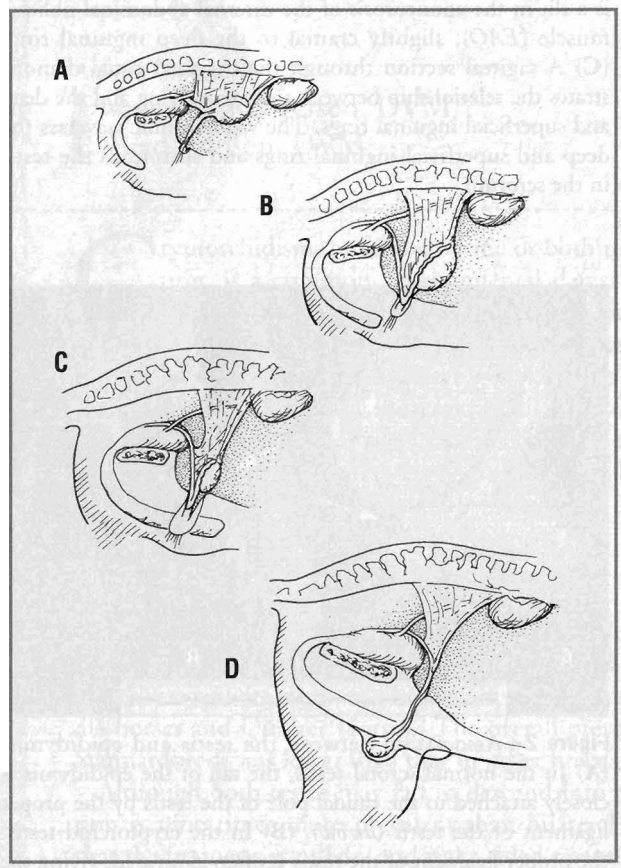

Figure 4—Various stages of descent of the testis. (**A**) The vaginal process is formed at approximately day 45 to 50 of gestation. (**B**) The tail of the epididymis normally reaches or enters the vaginal ring at approximately day 150. (**C**) The testis reaches the vaginal ring at approximately day 275 to 300. (**D**) The testis enters the scrotum between day 315 of gestation and 10 days after birth.

Descent of the Testis

The process of descent of the testis is complex and incompletely understood.[3,10] Normal descent depends on many developmental events occurring properly and in synchrony. After the deterioration of the caudal portion of the mesonephric kidney at approximately day 65 of gestation,[19,22] the testis is suspended from the dorsal body wall by cranial, dorsal, and caudal attachments[14,19,22] (Figure 3).

Intraabdominal pressure in the fetus increases shortly after the umbilical canal closes to produce a closed peritoneal cavity.[19] The pressure from the peritoneal fluid in the abdominal cavity causes an outward extension of parietal peritoneum (the tunica vaginalis parietalis, or vaginal process) at 45 to 50 days of gestation[14,19,22] (Figure 4A). The vaginal process extends through the deep inguinal ring and eventually through the inguinal canal into the scrotum to form the vaginal tunic of the testis. The vaginal process extends around the lateral and ventral borders of the caudal inguinal insertion of the gubernaculum.[19]

The gubernaculum, or posterior gonadal ligament, is a fetal mesenchymal cordlike structure that extends caudally from the caudal pole of the testis to the inguinal region of the peritoneal cavity.[14,19,21,22] Initially, the gubernaculum is

retroperitoneal; during gestation, it becomes suspended from the dorsal body wall by a peritoneal fold that is continuous with the mesorchium of the testis.[14,19,22] Early in gestation, the mesonephric duct from the early embryologic mesonephric kidney divides the gubernaculum into two segments.[22]

With the further development of the vaginal process and deterioration of the caudal portion of the mesonephric kidney, the gubernaculum can be divided into four segments[22] (Figure 5). The segment of the gubernaculum that extends from the caudal pole of the testis to the mesonephric duct is the caudal gonadal ligament (referred to after birth as the proper ligament of the testis). The portion of the gubernaculum that extends caudally from the mesonephric duct to the blind end of the vaginal process is the gubernaculum proprium (referred to after birth as the ligament of the tail of the epididymis). The gubernaculum proprium is partially within the abdominal cavity and partially within the vaginal process until the tail of the epididymis passes through the vaginal ring.

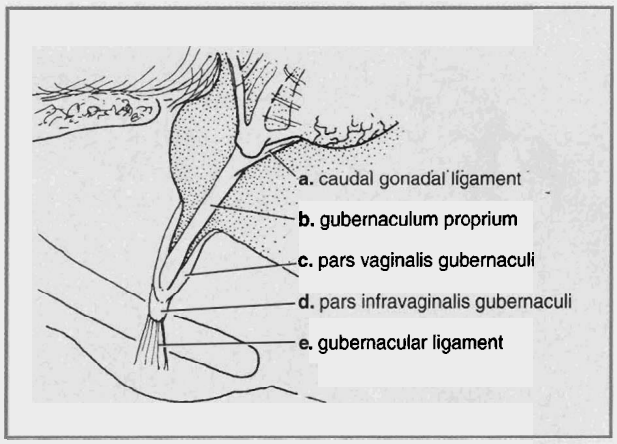

Figure 5—After the deterioration of the caudal portion of the mesonephric kidney and the further development of the vaginal process, the gubernaculum can be divided into four parts: (**a**) the caudal gonadal ligament, (**b**) the gubernaculum proprium, (**c**) the pars vaginalis gubernaculi, and (**d**) the pars infravaginalis gubernaculi. The latter part is continuous with a separately described structure, (**e**) the gubernacular ligament.

The extraabdominal part of the gubernaculum that intricately surrounds the vaginal process is the pars vaginalis gubernaculi. The extraabdominal segment that is not invaded by the vaginal process is the pars infravaginalis gubernaculi, which is continuous with the gubernacular ligament. This ligament, which is described separately from the other four segments of the gubernaculum,[22] is believed to provide some directional guidance for the vaginal process[19] or to create a tunnel of low resistance into which the vaginal process extends.[22] The gubernacular ligament and pars infravaginalis gubernaculi are together referred to as the inguinal extension of the gubernaculum.[23,24] After birth, this embryologic structure becomes the scrotal ligament.[23,24]

The gubernaculum guides the testis caudally toward the vaginal ring and through the inguinal canal.[2] The testis and epididymis shift caudally because of the tension placed on the gubernaculum by the everting vaginal process, the enlargement of the metanephric kidney, and the elongation of the pelvic region.[7,19,20,22,25] The gubernaculum does not actively contract to pull the testis caudally.[20] The tail of the epididymis normally reaches the vaginal ring or is within the inguinal canal at approximately day 150 of gestation[19] (Figure 4B). The testis does not reach or enter the vaginal ring until approximately 270 to 300 days of gestation (Figure 4C).

Pressure from the abdominal viscera is believed to help keep the testis positioned near the vaginal ring.[19,22] The change in size and shape of the testis and the enlargement of the vaginal ring and inguinal canal enable the testis to move through the inguinal canal.[19,22] Expansion of the inguinal canal is related to the enlargement of the caudal three segments of the gubernaculum and the tail of the epididymis.[14,19,22] The enlarged caudal portion of the gubernaculum is often palpable in the scrotum after parturition and may be confused with a scrotal testis.[2,19,22] The testes move into the scrotum at day 315 of gestation to 10 days after birth[19] (Figure 4D).

CLASSIFICATION

Failure of testicular descent can be categorized according to the location of the nondescended testis (inguinal or abdominal).[12,17,19,22] *Inguinal retention* describes a testis that has passed through the vaginal ring but has not reached or entered the scrotum.[10] An inguinal retained testis can be located external to the superficial inguinal ring (Figure 6A) or in the inguinal canal[26] (Figure 6B). Inguinal retained testes have reportedly entered the scrotum in horses up to 2 to 3 years of age.[4,11,13]

Abdominal retention describes a testis that has not passed through the vaginal ring and remains in the abdomen.[10] Such retention has been classified as incomplete or partial abdominal retention (Figure 6C) or complete abdominal retention[10,11,26,27] (Figure 6D). With incomplete or partial abdominal retention, the testis remains within the abdomen but the tail and a portion of the body of the epididymis descend through the vaginal ring and into the inguinal canal.[11,26,27] Complete abdominal retention is failure of both the testis and epididymis to pass through the vaginal ring. In horses with complete abdominal retention, the vaginal ring and the vagi-

nal process are often poorly developed.[17,26]

PATHOLOGY

Nondescended testes are generally smaller and softer than are scrotal testes.[7,17,28] Nondescended testes are sterile because spermatogenesis is inhibited by the elevated environmental temperature around the retained testis.[4,29] In unilateral cryptorchid stallions, the descended testes are capable of producing sperm.[7,12,19,30] If the scrotal testis of a unilateral cryptorchid stallion is removed, the retained testis may hypertrophy to a size comparable to that of a normal scrotal testis.[13]

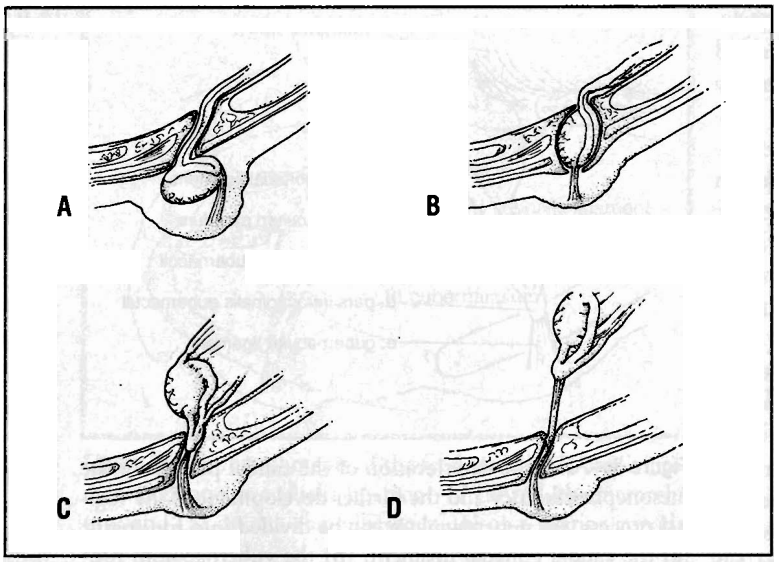

Figure 6—(**A**) Inguinal retention with the testis situated external to the superficial inguinal ring. (**B**) Inguinal retention with the testis situated within the inguinal canal. (**C**) Incomplete or partial abdominal retention; the testis fails to pass through the vaginal ring, but the tail and part of the body of the epididymis descend through the vaginal ring and into the inguinal canal. (**D**) Complete abdominal retention; the testis and epididymis fail to pass through the vaginal ring.

The histologic appearance of abdominal retained testes differs from that of inguinal retained testes.[12] Spermatogenesis in abdominal retained testes does not proceed beyond the A- or B-spermatogonia (Figure 7) and ceases after production of the primary spermatocytes in inguinal retained testes.[12] Retention of the testis also impedes the development of the seminiferous tubules. The extent of development of the tubules in an abdominal retained testis is similar to that of a 3- to 4-month-old foal; the development of the tubules in an inguinal retained testis is comparable to that of a 9- to 12-month-old foal.[17] Generalized hyperplasia of the interstitial cells may be present in the retained testis.[31,32]

Neoplastic or cystic enlargement of the retained testis has been reported.[31,33–36] Various tumors (e.g., teratomas,[31,34–36] interstitial cell tumors,[37] and seminomas[38,39]) have been associated with cryptorchid testes. Such congenital defects as splenic–gonadal fusion,[40] fused abdominal testes,[41] and elongation of the suspensory ligament of a testis[42] have been associated with retained abdominal testes. Torsion of the spermatic cord of abdominal retained testes affected by a seminoma has been reported in two cryptorchid stallions.[38] Congenital monorchism is rare in horses,[18,43,44] and we can find no reports of anorchism or polyorchidism in horses.

CAUSES

A genetic basis for equine cryptorchidism has been proposed.[2,7,30,45] The decreasing incidence of cryptorchidism in certain lines of horses suggests that prudent selection of breeding stock may have some effect on the condition.[7,30] Autosomal dominant[7,46] and autosomal recessive[7] modes of inheritance have been proposed. Some researchers suggest that cryptorchidism is linked to the sex chromosomes.[4,7,47]

Cryptorchidism has been associated with intersexuality and with abnormal karyotypes,[48–50] suggesting that normal descent of the testis is controlled by multiple genes.[4] In one study, only a small percentage (9 of 5018 horses) of cryptorchid stallions were actually intersexes.[9] Genetic factors that cause cryptorchidism may be expressed as abnormalities associated with hormone secretion or the improper development of structures that are important in the normal descent of the testis. Because of the hereditary possibilities of cryptorchidism, the use of unilateral cryptorchid stallions in breeding programs should be discouraged.[3,30]

Hormones play a role in the normal descent of the testes.[22,25] Descent is believed to be associated with gonadotropin-induced androgen stimulation,[19] and the changes in size and shape of the gubernaculum are believed to be controlled by hormones originating from within the fetal testis.[19,20,22] In young boys, hormonal imbalance as a cause of cryptorchidism is suggested by the fact that testicular descent may occur after human chorionic gonadotropin (hCG) is administered.[3]

Mechanical causes of cryptorchidism are often difficult to establish. Faults in the development of the gubernaculum, vaginal process, vaginal ring, inguinal canal, or testis may have a genetic basis and may in turn impede the descent of the testes. The most obvious of the hypothesized mechanical causes of cryptorchidism are undue stretching of the gubernaculum; inadequate ab-

dominal pressure for proper evagination of the vaginal process; inadequate expansion of the gubernaculum and tail of the epididymis to enlarge the vaginal ring and inguinal canal; and displacement of the testis within the abdomen, where the testis is held by organ pressure (which prevents it from reaching or passing through the inguinal canal).[19]

In horses, the left testis has been observed to be slightly larger and to lag slightly behind the right testis during descent.[13,19,25] Either factor could prevent the left testis from passing through the vaginal ring or reaching the vaginal ring before it begins to constrict (shortly after parturition). These factors may explain the higher frequency of abdominal retention associated with the left testis.[25]

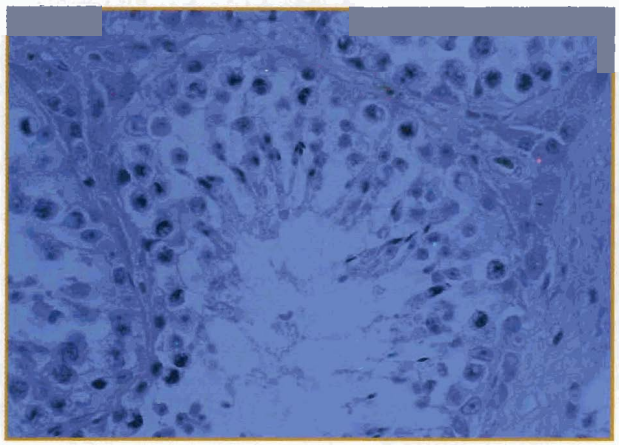

Figure 7A

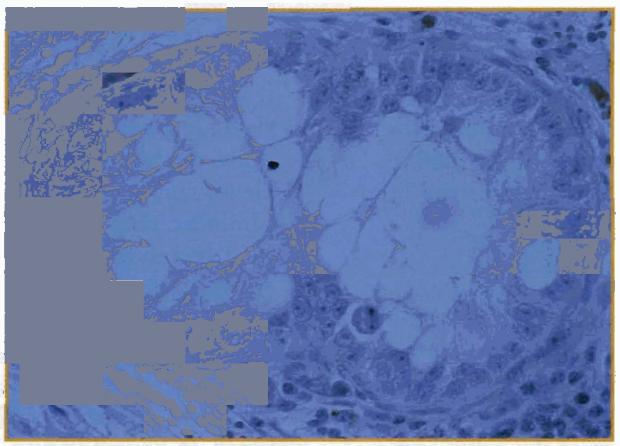

Figure 7B

Figure 7—(A) Histologic cross-section of a seminiferous tubule from a scrotal testis; normal spermatogenesis is evident. **(B)** Histologic cross-section of a seminiferous tubule from an abdominal retained testis; alterations in spermatogenesis are evident.

DIAGNOSIS

Identifying a retained testis can be as simple as observing the horse's behavior, inspecting the scrotum, and obtaining a castration history. A recently purchased gelding with an unknown castration history that exhibits stallionlike behavior may be a bilateral cryptorchid or a unilateral cryptorchid that has had its descended testis removed (i.e., a hemicastrate). The veterinarian must determine whether the horse is truly a cryptorchid and, if so, which testis is retained (or whether both are retained).[4]

The masculine behavior displayed by cryptorchid stallions is often enough to suggest that a horse has a retained testis. Cryptorchid stallions generally have the physical characteristics of an entire stallion (i.e., one with both testes located in the scrotum) because the androgen-producing interstitial cells (Leydig's cells) of the testis remain functional.[51] Like entire stallions, cryptorchid stallions can be nervous and irritable, attempt breed nearby mares, and have dispositions that are hazardous to owners and trainers.[5,7]

Visual Examination and Palpation

To determine whether a horse has a retained testis, the scrotum is initially examined visually. The scrotum and superficial inguinal ring should be palpated externally.[2,4] The scrotum and inguinal region should be examined for the presence of an incisional scar, which indicates that castration was attempted. In unilateral cryptorchids, digital palpation of the descended testis and associated spermatic cord can indicate whether the left or right testis is retained.

Palpation of a testis that lies in the inguinal canal is often difficult. Tranquilization or sedation may make an inguinal testis more accessible by relaxing the cremaster muscle. Intravenous sedation with either xylazine (0.5 mg/kg) or detomidine (0.025 mg/kg), alone or in combination with butorphanol (0.02 mg/kg), relaxes the cremaster muscle.

Palpation per rectum can facilitate identification of a retained testis or evaluation of the extent of testicular descent.[2,10,52–55] Because of the risk of rectal tears, caution is necessary when palpating stallions per rectum.[2,25] They are usually unaccustomed to the procedure; young stallions may be more predisposed to rectal tears because of their smaller stature.[2,4]

Palpation of the vaginal ring and the structures that enter it may help in determining whether the testis has entered the inguinal canal.[10,53] During palpation per rectum, the examiner should use the left hand to determine whether the right testis is retained and the right hand to determine whether the left testis is retained.[54] The vaginal ring is located 8 to 12 cm from the midline, just cranial to the brim of the pelvis[2,4,53] (Figure 8). The easily palpated ductus deferens enters the caudo-

medial aspect of the vaginal ring and can be used as an aid in locating the ring. The testicular vessels that enter the vaginal ring can be difficult to palpate and identify per rectum.[3]

Palpation of the ductus deferens at the vaginal ring does not help in distinguishing between complete and incomplete abdominal retention. An inability to identify the vaginal ring and the ductus deferens may indicate that the testis and epididymis are retained in the abdomen.[4] An abdominal testis is often difficult to palpate per rectum because it is smaller and more flaccid than a normal scrotal testis. Because of its mobility, an abdominal testis is difficult to identify in the caudal abdomen.[7,10,53]

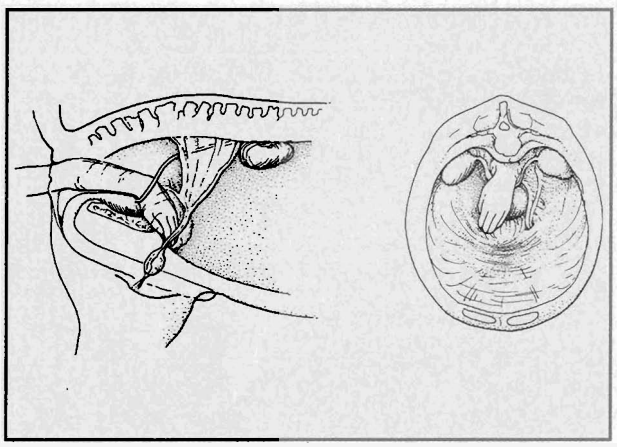

Figure 8—Lateral and craniocaudal views of palpation per rectum of the vaginal ring.

Ultrasonographic Examination

Ultrasonographic examination of the caudal portion of the abdomen per rectum via a 5-MHz probe may be useful in identifying an abdominal testis.[56] Abdominal testes are less echogenic than scrotal testes.[56] Scans per rectum should begin at the brim of the pelvis and continue cranially in a sweeping pattern between the midline and the lateral abdominal wall.

Ultrasonography can be helpful in distinguishing between inguinal fat, vessels, the ductus deferens, and other confusing structures that may be palpated per rectum. Ultrasonographic examination of the inguinal canal and surrounding tissue can be performed externally to identify an inguinal retained testis.[30] Transinguinal and transabdominal ultrasonography have been used successfully at the Veterinary Teaching Hospital at Auburn University to identify retained testes within or closely associated with the inguinal canal.

Hormonal Assays

Hormonal assays for concentrations of testosterone and estrogen can be useful in distinguishing castrates from cryptorchid stallions with no scrotal testes.[12,29,32,52,57–65] Serum concentrations of testosterone are reportedly 86% to 95% accurate in identifying the presence of testicular tissue.[29,58] In one study, serum concentrations of testosterone over 100 pg/ml were associated with the presence of testicular tissue; concentrations less than 40 pg/ml indicated the absence of such tissue.[58] In the same study, stallions younger than 18 months of age usually had serum concentrations of testosterone less than 100 pg/ml. In another study, the presence of testicular tissue was identified when serum concentrations of testosterone were greater than 44 pg/ml; the absence of testicular tissue was identified when concentrations were less than 24 pg/ml.[29]

The hCG stimulation test is used to identify cryptorchidism when measurement of basal concentrations of testosterone alone yields inconclusive information.[29,32,58,60,62,63,65–67] In one study, the hCG stimulation test was 94.6% accurate in identifying cryptorchidism in horses.[58] The intravenous administration of 6000 to 12,000 units of hCG stimulates the production of testosterone from the interstitial cells of the testis.[29,45] Serum is collected before hCG is administered, and a second sample is usually collected 30 to 120 minutes after hCG is given.[29,45,58,65] There are greater increases in serum concentrations of testosterone in response to hCG if the second sample is collected 24 hours (or even 72 hours) after the initial sam-

In one study, serum concentrations of testosterone in cryptorchid stallions were greater than 100 pg/ml and increased after hCG was administered.[58] Castrates have a low basal concentration of testosterone (less than 40 pg/ml) and exhibit no response to the administration of hCG.

Hormonal assays that measure serum concentrations of conjugated estrogens have been used to identify cryptorchid horses.[29,32,58–60,63,64,67] Assays are routinely performed for estrone sulfate. Measurement of serum concentrations of estrogen was 95% to 96% accurate in differentiating cryptorchid stallions from true castrates if the horses were older than 3 years of age.[58] The serum concentrations of estrogen in geldings were less than 50 pg/ml, but the concentrations in horses with testicular tissue exceeded 400 pg/ml. The serum concentration of conjugated estrogens in donkeys of all ages and in horses younger than 3 years of age was much less than that in horses older than 3 years age.[58]

A recent study evaluated the ability to analyze the concentrations of unconjugated estrogens in the feces as a method for determining the presence of testicular

tissue.[68] Cryptorchid and entire stallions had increased concentrations of fecal unconjugated estrogens; in castrated horses, concentrations of unconjugated estrogens were much lower. Following are advantages of using fecal samples: The samples are collected by noninvasive means, estrogens are stable in feces for at least 1 week, and it is not necessary to perform a stimulation test.[68]

For hormonal assays, it is crucial to use a laboratory that has established an accurate reference range for serum concentrations of equine testosterone and estrogen.[2,3,30] Normal serum concentration ranges of these enzymes in other species may be spurious when applied to horses and should be interpreted cautiously.[30]

Laparoscopic Examination

Laparoscopic techniques can be used to diagnose and identify an abdominal testis.[69] Such a testis is usually visualized near the vaginal ring; identifying the structures that enter the ring can help in distinguishing abdominal and inguinal retention. Laparoscopic techniques are relatively noninvasive, are associated with few complications, and can be performed in a standing horse.[69]

CONCLUSION

Cryptorchidism is a common developmental defect encountered by equine practitioners. Faults in the descent of the testis are believed to result from genetic, hormonal, or mechanical factors or from a combination of these factors. A thorough physical examination and an understanding of the available diagnostic procedures are important in identifying a retained testis or a horse with a retained testis.

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About the Authors

Drs. Rodgerson and Hanson are affiliated with the Department of Large Animal Surgery and Medicine, College of Veterinary Medicine, Auburn University, Auburn, Alabama. Dr. Hanson is a Diplomate of the American College of Veterinary Surgeons.

REFERENCES

1. Genetzky RM, Shira MJ, Schneider EJ, et al: Equine cryptorchidism: Pathogenesis, diagnosis, and treatment. *Compend Contin Educ Prac Vet* 6(10):S577–S582, 1984.
2. Schumacher J: Surgical disorders of the testicle and associated structures, in Auer JA (ed): *Equine Surgery.* Philadelphia, WB Saunders Co, 1992, pp 674–703.
3. Trotter GW: Normal and cryptorchid castration. *Vet Clin North Am Equine Pract* 4:493–513, 1988.
4. Varner DD, Schumacher J, Blanchard TL, et al: Diseases of the testes, in Varner DD, Schumacher J, Blanchard TL, Johnson L (eds): *Diseases and Management of Breeding Stallions.* Goleta, CA, American Veterinary Publications, 1991, pp 193–232.
5. Hobday FTG: The castration of cryptorchid horses, in Hobday FTG (ed): *The Castration of Cryptorchid Horses and the Ovariectomy of Troublesome Mares.* New York, William R. Jenkins, 1903, pp 17–64.
6. O'Connor JJ: Operations—Castration of cryptorchids, in O'Connor JJ (ed): *Dollar's Veterinary Surgery,* ed 2. London, Bailliere, Tindall & Cox, 1930, pp 322–336.
7. Leipold HW, DeBowes RM, Bennett S, et al: Cryptorchidism in the horse: Genetic implications. *Proc AAEP* 31:579–589, 1986.
8. Teigland MB: Medical examination of horses at auction sales. *Vet Clin North Am Equine Pract* 8:413–420. 1992.
9. Hayes HM: Epidemiological features of 5009 cases of equine cryptorchidism. *Equine Vet J* 18:467–471, 1986.
10. Stickle RL, Fessler JF: Retrospective study of 350 cases of equine cryptorchidism. *JAVMA* 172:343–346, 1978.
11. Cox JE, Edwards GB, Neal PA: An analysis of 500 cases of equine cryptorchidism. *Equine Vet J* 11:113–116, 1979.
12. Coryn M, DeMoor A, Bouters R, et al: Clinical, morphological and endocrinological aspects of cryptorchidism in the horse. *Theriogenology* 16:489–496, 1981.
13. Cox JE: Factors affecting testis weight in normal and cryptorchid horses. *J Reprod Fertil [Suppl]* 32:129–134, 1982.
14. Ashdown RR: The anatomy of the inguinal canal in the domesticated mammals. *Vet Rec* 75:1345–1351, 1973.
15. Sack WO, Habel RE: Pelvis—Pelvic inlet, in Sack WO, Habel RE (eds): *Guide to the Dissection of the Horse.* Ithaca, NY, Veterinary Textbooks, 1988, pp 63–73.
16. Varner DD, Schumacher J, Blanchard TL, et al: Reproductive anatomy and physiology, in Varner DD, Schumacher J, Blanchard TL, Johnson L (eds): *Diseases and Management of Breeding Stallions.* Goleta, CA, American Veterinary Publications, 1991, pp 1–60.
17. Bishop MWH, David JSE, Messervy A: Some observations on cryptorchidism in the horse. *Vet Rec* 76:1041–1048, 1964.
18. Trotter GW, Aanes WA: A complication of cryptorchid castration in three horses. *JAVMA* 178:246–248, 1981.
19. Bergin WC, Gier HT, Marion GB, et al: A developmental concept of equine cryptorchidism. *Biol Reprod* 3:82–92, 1970.
20. Noten DM, De Lahunta A: Derivatives of the intermediate mesoderm: Reproductive organs, in Noten DM, De Lahunta A (eds): *The Embryology of Domestic Animals.* Baltimore, Williams & Wilkins, 1985, pp 322–342.
21. Noten DM, De Lahunta A: Cytogenetics and sex determination, in Noten DM, De Lahunta A (eds): *The Embryology of Domestic Animals.* Baltimore, Williams & Wilkins, 1985, pp 343–356.
22. Smith JA: The development and descent of the testis in the horse. *Vet Annu* 15:156–161, 1975.
23. Arighi M, Horney JD, Bosu WTK: Noninvasive inguinal approach for cryptorchidectomy in thirty-eight stallions. *Can Vet J* 29:346–349, 1988.
24. Valdez H, Taylor TS, McLaughlin SA, et al: Abdominal

cryptorchidectomy in the horse, using inguinal extension of the gubernaculum testis. *JAVMA* 174:1110–1112, 1979.
25. Arthur GH: The surgery of the equine cryptorchid. *Vet Rec* 73:385–389, 1961.
26. Wright JG: The surgery of the inguinal canal in animals. *Vet Rec* 75:1352–1363, 1963.
27. Cox JE: Cryptorchidism, in Robinson NE (ed): *Current Therapy in Equine Medicine*, ed 2. Philadelphia, WB Saunders Co, 1990, pp 571–573.
28. O'Connor JJ: Affections of the male genital organs, in O'Connor JJ (ed): *Dollar's Veterinary Surgery*, ed 2. London, Bailliere, Tindall & Cox, 1930, pp 670–678.
29. Arighi M, Bosu WTK: Comparison of hormonal methods for diagnosis of cryptorchidism in horses. *J Equine Vet Sci* 9:20–26, 1989.
30. Cox JE: Cryptorchid castration, in McKinnon AO, Voss JL (eds): *Equine Reproduction.* Philadelphia, Lea & Febiger, 1993, pp 915–920.
31. Willis RA, Rudduck HB: Testicular teratomas in horses. *J Pathol Bacteriol* 55:165–171, 1943.
32. Arighi M, Bosu WTK, Raeside JI: Hormonal diagnosis of equine cryptorchidism and histology of the retained testes. *Proc AAEP* 31:591–602, 1986.
33. Schumacher J, Lenz SD, Walker W: Cystic rete testis associated with cryptorchidism in a horse. *Vet Pathol* 31:115–117, 1994.
34. Stick JA: Teratoma and cyst formation of the equine cryptorchid testicle. *JAVMA* 176:211–214, 1980.
35. Smyth GB: Testicular teratoma in an equine cryptorchid. *Equine Vet J* 11:21–23, 1979.
36. Deen T: Testicular teratoma in a bilateral cryptorchid horse. *Vet Med* 82:1257–1259, 1987.
37. Gelberg HB, McEntee K: Equine testicular interstitial cell tumors. *Vet Pathol* 24:231–234, 1987.
38. Hunt RJ, Hay W, Collatos C, et al: Testicular seminoma associated with torsion of the spermatic cord in two cryptorchid stallions. *JAVMA* 197:1484–1486, 1990.
39. Smith BL, Morton LD, Watkins JP, et al: Malignant seminoma in a cryptorchid stallion. *JAVMA* 195:775–776, 1989.
40. Noakes DE, White RAS: Splenic–gonadal fusion in the horse. *Vet Rec* 98:382–383, 1976.
41. Sherman K, Myhre G, Wells R: Abdominal fused testicles in a miniature horse: A case report. *J Equine Vet Sci* 9: 191–192, 1989.
42. Wilson DG, Nixon AJ: Case of equine cryptorchidism resulting from persistence of the suspensory ligament of the gonad. *Equine Vet J* 18:412–413, 1986.
43. Parks AH, Scott EA, Cox JE, et al: Monorchidism in the horse. *Equine Vet J* 21:215–217, 1989.
44. Santschi EM, Juzwiak JS, Slone DE: Monorchidism in three colts. *JAVMA* 194:256–266, 1989.
45. Vaughan JT: Surgery of the male equine reproductive system, in Jennings PB (ed): *The Practice of Large Animal Surgery.* Philadelphia, WB Saunders Co, 1984, pp 1083–1108.
46. Technical Development Committee: Cryptorchidism with special reference to the condition in the dog. *Vet Rec* 66: 482–483, 1954.
47. Blanchard TL, Schumacher J, Taylor TS, et al: Detecting unilateral and bilateral cryptorchidism in large animals. *Vet Med* 85:395–403, 1990.
48. Constant SB, Larsen RE, Ashbury AC, et al: XX male syndrome in a cryptorchid stallion. *JAVMA* 205:83–85, 1994.
49. Dunn HO, Vaughan JT, McEntee K: Bilaterally cryptorchid stallion with female karyotype. *Cornell Vet* 64:265–275. 1974.
50. McIlwraith CW, Owen RAR: An equine cryptorchid with testicular and ovarian tissue. *Equine Vet J* 8:156–160, 1976.
51. Ganjam VK, Kenney RM, Gledhill BL: Increased concentration of androgens in cryptorchid stallion testes. *J Steroid Biochem* 5:709–710, 1974.
52. Moore JN, Johnson JH, Tritscher LC, et al: Equine cryptorchidism: Pre-surgical considerations and surgical management. *Vet Surg* 7:43–47, 1978.
53. Adams OR: An improved method of diagnosis and castration of the cryptorchid horse. *JAVMA* 145:439–446, 1964.
54. O'Connor JP: Rectal examination of the cryptorchid horse. *Ir Vet J* 25:129–131, 1971.
55. Vaughan JT: Surgery of the testes, in Walker DF, Vaughan JT (eds): *Bovine and Equine Urogenital Surgery.* Philadelphia, Lea & Febiger, 1980, pp 156–163.
56. Jann HW, Rains JR: Diagnostic ultrasonography for evaluation of cryptorchidism in horses. *JAVMA* 196:297–300, 1990.
57. Cox JE: Testosterone concentrations in normal and cryptorchid horses: Response to human chorionic gonadotrophin. *Anim Reprod Sci* 18:43–50, 1989.
58. Cox JE, Redhead PH, Dawson FE: Comparison of the measurement of plasma testosterone and plasma oestrogens for the diagnosis of cryptorchidism in the horse. *Equine Vet J* 18:179–182, 1986.
59. Cox JE: Testosterone or oestrone sulphate assay for the diagnosis of cryptorchidism? *J Reprod Fertil [Suppl]* 32:625, 1982.
60. Cox JE: Experiences with a diagnostic test for equine cryptorchidism. *Equine Vet J* 7:179–183. 1975.
61. Cox JE, Williams JH: Some aspects of the reproductive endocrinology of the stallion and cryptorchid. *J Reprod Fertil [Suppl]* 25:75–79, 1975.
62. Cox JE, Williams JH, Rowe PH, et al: Testosterone in normal, cryptorchid and castrated male horses. *Equine Vet J* 5:85–90, 1973.
63. Ganjam VK: An inexpensive, yet precise, laboratory diagnostic method to confirm cryptorchidism in the horse. *Proc AAEP* 23:245–248, 1978.
64. Ganjam VK, Kenney RM: Androgens and oestrogens in normal and cryptorchid stallions. *J Reprod Fertil [Suppl]* 25: 67–73, 1975.
65. Stelmasiak T: The nonsurgical diagnosis of cryptorchidism in horses (testosterone and LH response to HCG injection). *NZ Vet J* 27:50, 1979.
66. Cox JE: Diagnostic test for equine cryptorchidism. *Vet Rec* 98:204, 1976.
67. Silberzahn P, Pouret EJM, Zwain I: Androgen and oestrogen response to a single injection of hCG in cryptorchid horses. *Equine Vet J* 21:126–129, 1989.
68. Palme R, Holzmann A, Mitterer T: Measuring fecal estrogens for the diagnosis of cryptorchidism in horses. *Theriogenology* 42:1381–1387, 1994.
69. Wilson DG: Laparoscopy as an aid in the surgical management of the equine hemicastrate. *Proc AAEP* 35:347–353, 1990.

ARTICLE #4 REVIEW QUESTIONS

The article you have read qualifies for ½ hour of Continuing Education Credit from the Auburn University College of Veterinary Medicine. *Choose only the one best answer* to each of the following questions; then mark your answers on the test form inserted in *The Compendium.*

1. Abdominal retention of the testis
   a. is most common on the right side.
   b. is most common on the left side.
   c. is equally common on both sides.
   d. does not occur.

2. The testis usually enters the scrotum
   a. at day 100 to 150 of gestation.
   b. at day 275 of gestation to 10 days before birth.
   c. at day 315 of gestation to 10 days after birth.
   d. 3 weeks after birth.

3. The fetal mesenchymal attachment from the caudal aspect of the fetal testis to the scrotum is the
   a. inguinal canal.
   b. gubernaculum.
   c. epididymis.
   d. vaginal process.

4. Cryptorchidism is caused by
   a. mechanical factors.
   b. hormonal factors.
   c. genetic factors.
   d. mechanisms that are not precisely known.

5. The superficial inguinal ring is
   a. slit in the aponeurosis of the external abdominal oblique muscle.
   b. slit in the aponeurosis of the internal abdominal oblique muscle.
   c. bound cranially and caudally by the rectus abdominis muscle.
   d. none of the above

6. Geldings generally have a serum testosterone concentration of how many pg/ml?
   a. 200 to 300
   b. 100 to 200
   c. greater than 75
   d. less than 40

7. The hCG stimulation test stimulates testosterone production by which cells of the testis?
   a. Leydig's
   b. cortical
   c. Sertoli's
   d. none of the above

8. Histologic section of a retained testis will demonstrate
   a. spermatogenesis.
   b. signs of hyperplastic seminiferous tubules.
   c. inhibition of spermatogenesis.
   d. enlarged Sertoli's cells.

9. Comparison of left- and right-sided testicular retention indicates
   a. a greater frequency on the left.
   b. a greater frequency on the right.
   c. a greater frequency on the left in horses up to 3 years of age.
   d. nearly equal frequency on both sides.

10. The cranial, caudal, and ventromedial borders of the deep inguinal ring are the
    a. caudal edge of the external abdominal oblique muscle, the pelvis and inguinal ligament, and the rectus abdominis muscle and prepubic tendon.
    b. caudal edge of the internal abdominal oblique muscle, the pelvis and inguinal ligament, and the external abdominal oblique muscle and prepubic tendon.
    c. caudal edge of the internal abdominal oblique muscle, the pelvis and inguinal ligament, and the rectus abdominis muscle and prepubic tendon.
    d. caudal edge of the rectus abdominis muscle, the pelvis and inguinal ligament, and the rectus abdominis muscle and prepubic tendon.