



CrossMark

Review: Abnormalities of the bull – occurrence, diagnosis and treatment of abnormalities of the bull, including structural soundness

D. F. Wolfe[†]

Department of Clinical Sciences, J.T. Vaughan Large Animal Teaching Hospital, College of Veterinary Medicine, Auburn University, 1500 Wire Road, Auburn, AL 36849-5522, USA

(Received 17 November 2017; Accepted 26 March 2018; First published online 2 May 2018)

Selecting bulls for reproductive soundness requires that the bull be structurally sound, free of abnormalities that impair his ability to produce adequate numbers of motile, morphologically normal spermatozoa, and be able to successfully complete coitus. This review discusses the diagnosis and etiology of abnormalities of the penis, prepuce as well as common musculoskeletal conditions that prevent normal pasture breeding soundness. A review of testicular and thermoregulation addresses the potential impact of musculoskeletal disorders on normal sperm production.

Keywords: bull, prepuce, penis, scrotum, lameness

Implications

There are very few sterile bulls, but many bulls are subfertile due to a variety of physical conditions that reduce reproductive soundness. Developmental anomalies or injuries of the penis and prepuce are seen during breeding soundness examinations or when bulls fail to achieve a satisfactory pregnancy percentage during natural breeding (Carson and Wenzel, 1995). Numerous congenital abnormalities prevent normal development that allow for breeding soundness. Many musculoskeletal abnormalities or injuries impair a bull's ability to move freely in his environment and complete coitus. Any febrile or inflammatory condition may impair normal scrotal and testicular thermoregulation and normal sperm production. Failure to achieve pregnancy in a limited breeding season results in a significant economic loss for livestock owners.

Introduction

The purpose of this review is to summarize common clinical abnormalities that prevent successful breeding performance or semen collection of bulls. This review will include normal anatomy, physiology and breeding behavior of bulls. Body systems included in the review are the penis, prepuce and sheath as well as common musculoskeletal conditions that impair efficient breeding performance. In addition, a review of normal thermoregulation of the scrotum and testes and implications for common disrupters of normal testes function.

Anatomy of the sheath, penis and prepuce

The sheath of the bull is a protuberance of the skin along the ventral abdomen and ends at the preputial orifice where the skin joins the non-haired epithelium of the prepuce. The prepuce ends at the preputial ring, which is the junction with the free portion of the penis several centimeters proximal to the glans penis. The non-erect penis is held in the preputial cavity by the paired retractor penis muscles (Wolfe *et al.*, 1998; Wolfe, 2015a).

The diameter and length of the prepuce vary considerably among bulls of varying breeds and ages, but the prepuce of an adult bull is 35 to 40 cm long and ~4 cm in diameter. The prepuce of *Bos indicus* bulls averages 5.5 cm longer than bulls of *Bos taurus* breeds and they have a more pendulous sheath and larger preputial orifice, which is 2 to 4 cm in diameter in *B. taurus* breeds and may exceed 10 cm in some *B. indicus* bulls (Bellenger, 1971).

Multiple interdigitating layers of elastic tissue between the preputial epithelium and the tunica albuginea of the penis allow the penis to glide within the sheath from full retraction to full extension. There is wide variation among bulls such that the penis extends 25 to 60 cm beyond the preputial orifice during full erection, therefore, the full excursion of the glans penis may be >1 m (Beckett and Wolfe, 1998).

[†] E-mail: wolfedf@auburn.edu

Penile and preputial conditions of young bulls that may prevent coitus

The most common anomalies of the penis, prepuce, or sheath of young bulls that prevent breeding soundness are penile fibropapillomas, persistent frenulum and incomplete separation of the epithelium of the penis and prepuce. Occasionally hair rings may cause pressure necrosis of penile tissues. Other anomalies include inadequate penile development such that penis is too short to complete coitus (Wolfe and Carson, 1998). Erection failure in young bulls will be discussed later in this manuscript.

Fibropappiloma of the penis

Fibropappiloma of the bull penis is somewhat common among young bulls housed together and is caused by the bovine pappiloma virus. The virus enters the epithelium of the penis through wounds or abrasions and causes focal benign neoplastic growths of fibroblasts on the free portion of the penis and prepuce. Homosexual riding of young bulls housed together may allow penile abrasions that allow virus entry into the epithelium (McEntee, 1950). Frequently, multiple bulls within a pen or pasture develop these growths, which are often pedunculated and often located near the glans penis. The lesion is often first detected when the young bull is presented for breeding soundness evaluation or semen collection. Rarely, large lesions may prevent retraction of the penis into the preputial cavity. The lesions may be removed by sharp excision or with the aid of cryotherapy or CO₂ laser being careful to avoid damage to the distal urethra (Wolfe et al., 1998).

Persistent frenulum

The frenulum, a collagenous band of connective tissue, is present at birth and attaches the glans penis to the penile section of the prepuce. The attachment should begin to break down by 4 weeks of age and separation should be complete by 8 to 11 months of age. Persistence of the frenulum attachment beyond 11 months of age is considered pathologic and the frequency of this condition reportedly varies from 3.64 to 5.2/1000 bulls (Ashdown, 1962). When this thin band of collagenous connective tissue fails to rupture the penis can extend, but the persistent frenulum deviates the tip of the penis ventrally and may prevent intromission. A persistent frenulum is a band of tissue from the median raphe at the posterior of the glans penis to the prepuce. This epithelium-covered band varies in width and thickness and usually contains one or more blood vessels. A persistent frenulum is easily surgically repaired by ligating each end to reduce the possibility of hemorrhage and transecting the tissue (Elmore, 1981). Affected bulls should be used only in commercial herds and not for the production of purebred breeding stock as several reports prove the condition is inherited and strongly suggest a simple autosomal recessive pattern (Elmore et al., 1978; Elmore, 1981).

Delayed or incomplete separation of the penile and preputial epithelium

During puberty, the penis of young bulls grows in length and diameter and a sigmoid flexure develops. The surface epithelium of the free portion of the penis is firmly attached to the epithelium of the prepuce at birth and these interdigitating tissues begin to separate at ~4 weeks of age and proceed caudally until complete separation occurs between 8 and 11 months of age (Ashdown, 1960).

In recent years, our hospital has seen an increased number of bulls over 11 months of age with incomplete separation of the epithelium of the penis and prepuce, which prevents complete penile extension (Wolfe D.F., unpublished results). Manual traction of the free portion of the penis and prepuce may complete the separation but avoid excessive force to prevent tearing or hemorrhage of tightly adhered tissues. This condition may be associated with later maturity and perhaps may have an undesirable heritable component.

Penile hair ring

Due to the homosexual mounting of young bulls housed together, an aggressor bull may accumulate body hair from the penmates on his penis. Alternatively, a young bull may accumulate hair on his penis from his own sheath during masturbation. In either case, the accumulated hair may form an encircling ring just proximal to the glans penis. The hair ring may become sufficiently tight to cause pressure necrosis of the urethra or in severe cases, avascular necrosis with damage to the dorsal penile nerves or sloughing of the glans penis if the condition is undetected. Treatment consists of removing the hair ring and topical application of an emollient antibacterial ointment. If a urethral fistula with granulation tissue has formed, surgical repair may be necessary to avoid hemorrhage contamination of semen during ejaculation (Wolfe and Carson, 1998).

Congenital short penis

Impotence due to the congenital short penis is an occasional cause of infertility. Observation of serviceability is the cornerstone of diagnosis of the congenital short penis. A presumptive diagnosis is possible if penile protrusion with the aid of a pudendal nerve block or with the bull anesthetized on a table is >25 cm from the preputial orifice to the tip of the penis in an adult bull (Gilbert, 1989).

Acquired conditions of the penis and prepuce

Preputial trauma

Preputial prolapse in bulls may follow breeding injury or herpes viral infections. The prepuce may be traumatized during coitus with the extent of the injury varying from mild abrasion, contusion or superficial hematoma, to bursting of the epithelium and deep laceration of the peripenile elastic tissue. The prepuce may become edematous and inflamed and in the case of preputial laceration, the deep elastic tissues become infected. Bulls that are naturally lacking a

preputial retractor muscle and frequently prolapse a few centimeters of prepuce when they are relaxed. These bulls are more likely to suffer preputial frostbite in severe cold climates (Wolfe *et al.*, 1998).

Preputial lacerations are more common in *Bos indicus* breeds and their crosses than in *Bos taurus* breeds. *Bos indicus* bulls are more likely to have an excessively pendulous sheath with a large preputial orifice and an excessively long prepuce (Bellenger, 1971). Therefore, they are more likely to sustain severe injury than bulls lacking excessive epithelium on the prepuce and sheath. *B. indicus* bulls usually prolapse the injured prepuce and preputial injuries in *B. taurus* usually do not lead to prolapse. In either breed, the injury likely occurs due to the discrepancy of the size of the prepuce and the vulva and vagina of the female. Consequently, the injury occurs more frequently when bulls are breeding heifers (Wolfe *et al.*, 1998).

Perhaps the most critical factors for determining the likelihood of returning to breeding soundness for a bull with preputial laceration are the extent of the damage to the peripenile elastic tissue and the length of preputial epithelium damaged. The goal of treatment for preputial laceration is to reduce edema, inflammation and infection so that the elastic tissue can heal sufficiently to allow the penis to extend and retract freely. Fortunately, most mild preputial lacerations heal spontaneously. Severe lacerations may require surgical repair for the bull to return to breeding soundness. *B. taurus* bulls that do not prolapse the injured prepuce are at greater risk of developing a retropreputial abscess than bulls of *B. indicus* breeds.

Treatment of preputial prolapse with edema consists of applying an emollient antibacterial ointment to the damaged tissue, covering the exposed tissues with a bandage and supporting the edematous tissue to prevent additional desiccation or trauma. A support sling consisting of an open weave such as burlap supported by elastic cords works well for this purpose. Continue treatment until the bull can retract the prepuce back into the sheath (Wolfe *et al.*, 1998). Surgical repair of preputial injury will be discussed with acquired conditions of the prepuce causing phimosis.

Avulsion of the prepuce from the free portion of the penis Occasionally bulls sustain avulsion of the penile epithelium at the fornix of the prepuce, an injury that most commonly occurs during semen collection with an artificial vagina (AV) but may occur during intromission with natural service. The separation of the epithelium of the penis and prepuce typically occurs transverse to the long axis of the penis and should be repaired surgically (Parker *et al.*, 1987; Wolfe, 2015b).

Anatomy and physiology of erection

Following olfactory or visual stimulation, blood flow increases in the deep artery of the penis and into the crus penis and subsequently into the corpus cavernosum penis (CCP) causing erection of the penis. The CCP in the bull is a closed system in that blood flows into the crus penis to cause erection and leaves the same area during detumescence following erection. The same stimulation that caused reflex dilation of the deep artery of the penis also causes relaxation of the paired retractor penis muscles, which causes relaxation of the sigmoid flexure and the mildly engorged penis protrudes from the sheath. With continued sexual stimulation, the ischiocavernosus muscles (ICM) begin rhythmic contraction, which raises blood pressure within the CCP and peak pressure within the CCP may be >14000 mmHg. This rapid increase in blood pressure within the CCP causes complete penile extension and erection. Following ejaculation the ICM relax, detumescence occurs as blood pressure within the CCP decreases and the penis is withdrawn back into the preputial cavity (Watson, 1964; Beckett et al., 1974b). The erection may be induced in the bull with an electroejaculator although the optimal method for evaluating erection is with observed test mating. Normal function of the penile nerves is essential for coitus and is most accurately assessed by observed test mating or by semen collection by AV (Beckett et al., 1978; Wolfe, 2015b).

Penile hematoma

The term hematoma of the penis indicates rupture of the tunica albuginea of the penis with subsequent hemorrhage and hematoma formation at the dorsum of the distal bend of the sigmoid flexure of the penis. The tunica albuginea rupture occurs at the time of peak erection pressure when there is a sudden angulation or bending of the penis due to the bull missing the vulva at the eiaculatory lunge or the bull or female slips or falls during coitus. When the penis bends, the effective volume of the CCP is reduced causing a sudden drastic increase in pressure within the CCP which may reach \geq 75 000 mmHg (Beckett *et al.*, 1974b). The tunica albuginea ruptures on the dorsum of the distal bend of the sigmoid flexure (Beckett *et al.*, 1974a). Peripenile elastic tissues and perhaps dorsal penile nerves may be damaged by jetting of blood under pressure from the CCP at the time of rupture. Light skinned bulls may develop extensive bruising of the sheath near the neck of the scrotum (Wolfe et al., 1998).

Diagnosis of penile hematoma should be confirmed by history and physical examination of the sheath and penis. Immediately after the rupture occurs the bull retracts the penis into the sheath, and blood and edema within the elastic tissues cause a symmetrical swelling on the dorsum of the distal bend of the sigmoid flexure, which will be immediately cranial to the scrotum. The size of the swelling is variable depending upon how many breeding attempts the bull makes following the rupture. The prepuce may prolapse due to blood and edema within the peripenile elastic tissue. The prepuce will be swollen, somewhat dry, and have a distinct bluish appearance due to extravasated blood in the subcutaneous areas of the prepuce (Wolfe *et al.*, 1998).

Approximately 50% of bulls with rupture of the tunica albuginea resume breeding with rest and conservative therapy. Approximately 80% of bulls undergoing surgical repair

of the ruptured tunica albuginea return to breeding soundness.

Conservative therapy consists of systemic antibiotics for 7 to 10 days, hydrotherapy on the swollen sheath at least once daily to help reduce swelling, and protective emollient treatment of the prepuce if indicated. The bull should have absolute sexual rest for at least 90 days before attempted return to breeding (Wolfe *et al.*, 1998).

Surgical repair of penile hematoma should be performed as quickly as possible but no later than 7 days following the injury. Place the bull under general anesthesia in right lateral recumbency and following aseptic preparation make a 20 cm incision through the sheath just cranial to the scrotum to exteriorize the dorsum of the distal sigmoid of the penis. Judiciously trim the frayed edges of the transverse rupture of the tunica albuginea and carefully oppose the edges with synthetic absorbable suture in a bootlace pattern. Evacuate the blood clot from the surgical site, oppose the elastic tissues with absorbable suture, and close the skin in routine fashion. Maintain the bull on systemic antibiotics for 5 days postoperatively and ensure a minimum of 60 days sexual rest before conducting breeding soundness examination before return to breeding (Wolfe *et al.*, 1998).

Several sequale to penile hematoma may prevent bulls from returning to breeding soundness. Abscessation with subsequent scar tissue formation may prevent adequate penile extension. Damage to the dorsal nerves of the penis will render the bull incapable of attaining intromission. Development of vascular shunts between the CCP and the peripenile vasculature prevent the CCP from being a closed system and consequently, the bull cannot achieve adequate blood pressure for erection. Injury to the prolapsed prepuce may result in fibrosis, which prevents normal penile extension or coitus (Young *et al.*, 1977; Wolfe, 2015b).

Observed test mating

The normal bull approaches the cow from the side then moves to her hindquarters to smell the vulva and confirm that she is in estrus. The bulbospongiosus muscles begin rhythmic contractions visible as pulsations just ventral to the anus. As the retractor penis muscles relax, the penis begins to protrude from the sheath and the bull prepares to mount. As he mounts, the penis becomes engorged and the free portion should visibly extend from the sheath. When the bull fully mounts the glans penis makes two or three searching motions near the vulva then the penis makes forceful intromission, the bull ejaculates in one thrust then dismounts the cow (Chenoweth, 1983).

Bulls with erectile dysfunction never achieve sufficient erection pressure to complete coitus. Bulls with nerve dysfunction mount the cow but the penile searching motions near the vulva are not evident and the bull fails to make intromission. Usually, the penis is placed along the cow's hip or below the vulva in the escutcheon area above the cow's udder (Young *et al.*, 1977; Wolfe, 2015b).

Erection failure due to corpus cavernosal shunts (*Impotential erigendi*)

Congenital vascular shunts

Occasionally young bulls fail to achieve intromission due to congenital corpus cavernosal vascular shunts. These bulls are usually normal on physical examination but fail to achieve adequate intracorporeal pressure for erection. When observed during test mating or with an erection induced by electroejaculation the free portion of the penis becomes noticeably bluish during attempted erection. The discoloration is due to blood exiting the CCP by subcutaneous capillaries and veins. Typically the shunts are multiple and not considered to be repairable (Young *et al.*, 1977; Wolfe, 2015b).

Acquired vascular shunts

Bulls may develop vascular shunts following trauma that penetrates the tunica albuginea of the penis. In the author's experience, this injury is a very uncommon cause of erection failure. However, as the penis heals following penile hematoma, vascular shunts between the CCP and the peripenile vasculature may develop which prevent the CCP from being a closed system. Consequently, the bull cannot achieve adequate blood pressure for erection. Affected bulls typically display normal libido and penile engorgement but do not develop adequate intracorporeal blood pressure to achieve intromission.

Utilize contrast cavernosography to confirm suspected vascular defects in the penis. With the bull restrained in lateral recumbency on a tilt table manually extend the penis and place a towel clamp under the dorsal apical ligament ~5 cm from the distal end of the penis to aid in manipulation of the penis during the procedure. Apply traction to the towel clamp to extend the penis and insert a sterile 16-gauge \times 3.8 cm needle at a 45° angle proximally through the skin and tunica albuginea and into the CCP. Place the needle on the dorsum of the penis near the towel clamp. Place a radiographic cassette under the penis then rapidly inject 30 ml of water-soluble radiographic contrast medium and expose the film. Slowly inject an additional 15 to 30 ml of medium and take two or three sequential radiographic exposures to include the free portion of the penis and the distal bend of the sigmoid flexure. Ideally all radiographic exposures should be completed within 60 s (Moll et al., 1993).

There are no vascular communications from the CCP to peripenile vasculature in the normal penis, and there should be no contrast media outside the CCP. A vascular shunt is identifiable as contrast media exiting the CCP. (Young *et al.*, 1977; Moll *et al.*, 1993).

Inability to extend the penis (Phimosis)

Several conditions may prevent a bull from being to extend the penis sufficiently to achieve coitus. These conditions may be due to preputial pathology or inadequate penile development.

Acquired preputial conditions causing phimosis

Healed preputial laceration as previously described, or preputial frostbite may lead to preputial stenosis and affected bulls exhibit phimosis, the inability to extend the penis. B. indicus bulls with preputial stenosis may require circumcision, resection and anastomosis of the prepuce, to restore breeding soundness. The circumcision procedure entails two circumferential incisions through the preputial epithelium proximal and distal to the damaged tissues. Deepen the incision judiciously to remove fibrous tissue with the peripenile elastic layers, maintaining as much normal elastic as possible. Achieve hemostasis and anastomose the proximal and distal epithelial edges with absorbable suture. Secure a 2.5-cm Penrose drain over the free portion of the penis to provide urine egress away from the surgical site and return the penis to the preputial cavity. Apply an elastic tape bandage around the sheath with sufficient pressure to prevent eversion of the prepuce while being careful not to occlude the lumen of the preputial cavity and compromise urine drainage. B. taurus bulls usually do not have sufficient preputial length to allow circumcision. In order for bulls to return to breeding soundness following surgery, the length of prepuce must be a minimum of 1.5 times the length of the free portion of the penis (Wolfe *et al.*, 1998). These bulls may require preputial scar revision in order to restore breeding soundness. In each case, the bull's value should determine if surgical intervention is economically justified.

Penile conditions causing phimosis

Bulls that have suffered prior hematoma of the penis may not be able to sufficiently extend the penis to complete coitus due to complications from the previous injury. Abscessation of the hematoma with subsequent scar tissue formation or preputial stenosis secondary to hematoma may prevent adequate penile extension. Bulls with adhesion of the peripenile elastic tissue to the skin will have a distinct wrinkling in the sheath during erection and attempted penile extension. That condition has poor prognosis for surgical correction. Bulls with congenital short penis make lack sufficient penile length to extend the penis adequately.

Inability to achieve intromission due to neuropathy

Several clinical conditions may prevent a bull from achieving intromission and ejaculation. These conditions may be due to the prior injury of the penis or prepuce or from acquired penile deviations.

Inability to achieve intromission due to penile neuropathy

Neuropathy of the dorsal nerves of the penis is not a common cause of inability to breed. However, trauma to the penis such as during preputial laceration or penile hematoma may result in nerve injury. Penile hematoma is defined as rupture of the tunica albuginea on the dorsum of the distal bend of the sigmoid flexure (Beckett *et al.*, 1974a). The jetting of blood under pressure from the CCP at the time of rupture may damage peripenile elastic tissues and perhaps dorsal penile nerve (Ashdown, 1970). The paired dorsal nerves of the penis lie immediately along the tunica albuginea of the penis adjacent to the dorsal arteries and veins (Larson and Kitchel, 1958). Observed test mating remains the method of choice for evaluating dorsal penile nerve function (Beckett *et al.*, 1978). Bulls with loss of penile innervation usually achieve an erection but fail to make searching motions with the glans penis and fail to make intromission. Typically, these bulls place the erect penis beside the tailhead on the cow's hip or below the vulva against the escutcheon or udder (Beckett *et al.*, 1978).

A very reliable method of assessing dorsal penile nerve function is to place a properly prepared bovine AV over the penis of a bull that has been adequately stimulated and allowed to mount a female in estrus. Bulls with normal nerve function will readily ejaculate into the AV. Measurement of nerve conduction velocity of the dorsal penile nerves provides a quantitative test of nerve function (Mysinger *et al.*, 1994). This procedure requires sophisticated instrumentation seldom found outside veterinary teaching hospitals.

Inability to achieve intromission due to penile deviation

Deviation of the normal penile architecture occasionally cause copulation failure in bulls. Penile deviations are classified according to their shape and in descending order of occurrence are either spiral (corkscrew), ventral or S-shaped. Bulls affected with penile deviations are usually between 2.5 and 5 years of age and typically had one or more successful breeding seasons before copulation failure. Usually, there is no known history of previous traumatic penile injury.

Spiral deviation

The distal portion of a bull's penis may spiral during electroejaculation and masturbation and approximately half the bulls serving a transparent AV developed spiral deviation of the penis (Seidel and Foote, 1969). Therefore, spiral deviation should consider pathologic only when consistently observed during natural mating. The deviation may initially occur intermittently and several test matings may be necessary to confirm the diagnosis. Diagnosis of penile deviation may be difficult in *B. indicus* bulls with a pendulous sheath and excessive prepuce if the deviation occurs before complete penile extension and remains hidden by these tissues. Careful observation of the test breeding, including palpation of the penis during attempted breeding may be necessary to differentiate spiral deviation from erection or extension failure.

There are two potential explanations for the development of penile spiral deviation in bulls. The first involves malfunction of the dorsal apical ligament which is a thick collagen band arising from the dorsum of the tunica albuginea about 2.5-cm proximal to the distal end of the prepuce and inserting back into the tunica albuginea near the distal end of the CCP. The apical ligament helps maintain the straight shape of the penis during erection. Historically, spiral deviations were considered to result from a short apical ligament that slipped off to the left side of the penis at peak erection just before intromission (Ashdown and Coombs, 1967).

More recently, based on a better understanding of erectile pressures and ejaculation, spiral deviations have been associated with the high maximum pressures within the CCP that occur during ejaculation. In fact, the penis of many normal bulls likely develops a spiral orientation in a cow's vagina during ejaculation (Seidel and Foote, 1969). Therefore, a possible explanation for spiral deviation is that a bull whose penis spirals before intromission reaches this maximum CCP pressure prematurely (Beckett *et al.*, 1974b). Although the cause of spiral deviations remains uncertain, both apical ligament malfunction and altered penile pressure may contribute to the condition.

Ventral deviation

Ventral penile deviations occur less commonly than spiral deviations, and their etiology is uncertain and perhaps multifactorial. Ventral deviations may occur due to the altered architecture of the tunica albuginea of the penis or apical ligament and altered blood flow through the ventral portion of the CCP, both of which probably result from chronic traumatic injury. Ventral deviations appear as long, gradual curvatures of the erect penis and the curvature frequently originates proximal to the junction of the free portion of the penis and prepuce. The deviation becomes more obvious as the bull's erection increases. The ventral penile deviation may occur during electroejaculation but should be confirmed by observed natural breeding or semen collection with an AV (Wolfe *et al.*, 1998).

S-Shaped deviation

The S-shaped deviation is the least common type of penile deviation and typically occurs in bulls over 4 years of age. The S-shaped curvature results from a normal penis with a short apical ligament or an excessively long penis with a normal apical ligament. A shortened apical ligament may result from trauma with resultant contracture of the ligament. In either case, the apical ligament prevents the penis from fully straightening during erection. No effective therapy exists for this type of penile deviation, but the bull could be used for artificial insemination if the deviation prevents copulation.

Repair of spiral and ventral penile deviations

Surgical repair of spiral deviations is much more successful than repair of ventral deviations. The author recommends attempted repair of ventral deviation only when the deviation is limited to the free portion of the penis. Surgical repair for either condition entails placing a fascia lata or synthetic graft between the apical ligament and the tunica albuginea of the penis. The graft serves as a lattice to strengthen and stabilize the apical ligament on the dorsum of the penis. If the graft procedure is successful, adequate healing usually occurs by 60 days to allow natural breeding (Walker and Young, 1979).

Anatomy the scrotum

The scrotum of the bull is protuberance of the ventral abdominal wall and contains the paired testes and their adnexa. The testes are enclosed by the tunica vaginalis visceralis and the tunica albuginea, a tough connective tissue layer, immediately surrounds the testicular parenchyma. Testicular thermoregulation is a complex process involving numerous local and systemic components such as countercurrent heat exchange in the pampiniform plexus, the function of the cremaster and tunica dartos muscles to regulate the surface of the scrotum, temperature sensors and systemic events (Waites, 1970). The testes are maintained at 4°C to 6° C cooler than core body temperature to maintain normal spermatogenesis (Waites, 1970). Thermographic patterns of the scrotum of normal bulls consist of a symmetrical and consistent pattern with a temperature gradient of 4°C to 6°C from the base to the apex which maintains testicular temperature 2°C to 6°C below core body temperature (Purohit et al., 1985).

There are numerous potential causes of elevated testicular temperature such as increased or decreased ambient temperature, fever, inflammation of the scrotum or testes, prolonged recumbency due to lameness or impaired neurologic control of the testicular thermoregulatory mechanisms (Kastelic *et al.*, 1995; Brito *et al.*, 2004).

Numerous sperm head and midpiece abnormalities increased in bulls following brief scrotal insulation for 4 days or administration of 20 mg dexamethasone IM for 7 days. These experiments were designed to investigate the effects of short-term elevations in testicular temperature or stress on sperm production (Barth and Bowman, 1994).

The remainder of this review will summarize disease or injury that may lead to impaired testicular thermoregulation and subsequent decrease in bull fertility.

Musculoskeletal disease or injury that impairs breeding soundness

Lameness

Cattle are relatively stoic animals and often do not show lameness until significant pathology is present. One report suggests that subfertility in bulls may due to subclinical lameness (Persson *et al.*, 2007). An auction market study in the Western USA found lameness in 15% of beef bulls and 26% of dairy bulls presented for sale (Ahola *et al.*, 2011). Lameness may occur from a broad array of genetic, nutritional, environmental, structural and infectious causes as well as injury. This discussion will explore anatomic and manage relationships with common causes of lameness in cattle.

Anatomy and conformation

Any discussion of lameness must begin with a review of normal anatomy. Conformation refers to the dimensions and shape of an animal and posture refers to the manner in which an animal stands (Greenough, 1972). Conformation varies

widely among cattle breeds and undesirable conformation traits may lead to unsoundness as animals grow due to abnormal stressors on bones, tendons, ligaments and joints.

When viewed from the rear cattle should stand with feet approximately as far apart as their hips. Cattle that are base wide that is their feet are farther apart than the width of their hips, are not common. However, cattle that are base narrow, their feet are closer together than the width of their hips are guite common, especially among beef breeds. Base-narrow conditions of the rear limbs frequently lead to the formation of screw claw as the animal grows. Animals with screw claw frequently develop bruises on the sole of the lateral claw as the lateral (abaxial) hoof wall grows under the hoof displacing the sole dorsomedially. These bruised areas frequently lead to sole ulcers or subsolar abscesses. Likewise, due to abnormal lines of stress on the hoof during weight bearing, vertical cracks frequently develop in the axial hoof wall. In addition, degenerative arthritis frequently develops prematurely in the coffin, pastern and fetlock joints of affected animals. This condition is common in continental breeds of beef cattle and their crosses. Although the classic twisted or corkscrew claw frequently does not develop until the animal is 2 or more years of age, the conformation leading to this condition is evident at weaning. Animals with this conformation are usually heavily muscled and base narrow when viewed from the rear.

Front limb conformation is also related to physical soundness. When viewed from the front the distance between the feet should be slightly less than the width of the shoulders and the hooves should point straight forward. Cattle with toes that point out (laterally) are more likely to develop abnormal hoof growth similar to screw claw in the rear hooves. The adjacent toes on one hoof should be equal length and the distance from the coronary band to the sole at the heel should be equal.

Laminitis

Clinical lameness in cattle with acute laminitis is generally less severe than horses with the similar condition. However, chronic laminitis is quite common, especially due to high concentrate feeding for high yearling weights in beef cattle. Animals with laminitis develop overgrown 'slipper' hooves and frequently suffer vertical or horizontal fissures in the hoof wall due to loss of flexibility of the hoof (Hoblet and Weiss, 2001). Subsolar hemorrhage, bruising and ulceration are frequent sequelae of chronic laminitis. In addition, as the hoof grows long toes, the angles of the coffin, pastern and fetlock joints change leading to abnormal stresses on these joints and their supporting soft tissue structures. Premature degenerative joint disease is a common occurrence in severely affected cattle.

Sole ulcer – pododermatitis circumscripta

This a specific lesion located at the junction of the sole and bulb of the heel in cattle. This lesion is most commonly found on the lateral claw of the hind limb of larger framed or more heavily muscled cattle. Bilateral lesions are quite common (Greenough, 1987). These lesions often lead to necrosis and sepsis of deeper soft tissue structures of the hoof as well as the coffin joint, deep digital flexor tendons flexor tendon sheath and navicular bone or more. One recent study questions the long-held the opinion that these lesions develop as sequelae to clinical or subclinical laminitis. This study found no gross or histologic evidence of laminitis in affected hooves compared with normal hooves and suggests the lesions develop due to the displacement of the third phalanx caused by excessive elasticity of the suspensory apparatus of the hoof (Lischer *et al.*, 2002).

Interdigital fibroma

Interdigital fibroma, (interdigital hyperplasia, corn) are proliferative growths of the skin of the interdigital space caused by chronic irritation or dermatitis (Berry, 2001). The condition is more common in bulls than females and more common in heavy than lighter weight animals. *B. indicus* crossbred cattle appear to have a higher incidence of interdigital fibroma than *B. taurus cattle*. Cattle that are extremely wide or that are extremely narrow in the interdigital space appear more at risk for development of this condition than cattle with normal interdigital conformation. Rarely does the problem develop in cattle <2 years of age and most animals presented for treatment are 4 to 7 years of age. These lesions may become ulcerated and perhaps infected leading to lameness. Treatment involves surgical excision of the hyperplastic tissue and bandaging the surgical site for a few days.

Infectious diseases of the digits

It is beyond the scope of this discussion to review all infectious diseases of the bovine foot. Numerous pathogens such as *Dichelobacter* (*Bacteriodes*) *nodosus*, *Fusobacterium necrophorum*, *Treponema* spp. are associated with diseases of the foot. In addition, interdigital fibroma may become infected as well as the bones, joints, tendons and ligaments of the foot secondary to sole ulcers or penetrating wounds. The reader is encouraged to seek additional references addressing these disease conditions.

Fractures of the coffin bone

Fractures of the coffin bone are not common and develop as two distinct clinical entities. The first, less common scenario is a traumatic fracture with acute lameness with little or no soft tissue swelling of the affected limb. When the foot is examined, there is no overt sign of trauma or disease. However, the animal is moderately to severely sensitive to pressure when hoof testers are applied. Traumatic fracture of the coffin bone is readily identified with radiography of the claw. These patients respond very well to the application of a hoof block to the sole of the adjacent claw. The block elevates the injured claw, which provides immediate relief from pain, and removal of weight from the injured claw allows bone healing to occur. Most animals return to soundness in 6 to 8 weeks following the injury.

Unfortunately, the more common scenario for coffin bone fracture is sequale to chronic septic conditions of the foot

such as foot rot or sole ulcer, which progress to osteomyelitis of the coffin bone. The resultant septic fracture of the coffin bone generally has guarded prognosis for recovery. Several complex surgical options are salvage of the affected bone and restoration of breeding soundness. Due to the extent of tissue damage, animal welfare concerns and economic impact, humane slaughter or claw amputation is frequently the treatment of choice for affected animals (Desrochers *et al.*, 2008).

Influence of osteoarthritis on bull infertility

Osteoarthritis can be a significant cause of infertility in bulls. Postmortem examination of hind limb joints of 34 non-lame beef bulls revealed that 12 with severe osteoarthritis had unsatisfactory semen quality. The joints of remaining bulls had mild or moderate lesions. No lesions were found in 11 control beef bulls with good fertility results (Persson et al., 2007). The authors propose that a negative effect of pain caused by moderate to severe osteoarthritis may lead to deteriorated sperm quality. The most prevalent location of lesions were lateral trochlear ridge of the stifle followed by the tibial plateau. Lesions in other locations of the stifle or tarsal joints were less frequent. Almost all lesions were characterized as being secondary to osteochondrosis dessicans. In all, 23 of 48 young growing bulls on intensive feeding had radiographic evidence of osteochondrosis at several locations (Reiland et al., 1978). A total of 13 of 25 bulls of lower intensity feeding had radiographic lesions of this disease process. In all, 25 dairy bulls sent to slaughter for non-medical reasons were evaluated and found to have osteochondrosis lesions. Fourteen of the bulls had degenerative disease predominantly involving the femoral trochlear ridge and 21 bulls had vertebral osteophytosis. Although these lesions were common in the middle-aged bulls, even when severe, were rarely associated with clinical lameness (Weisbrode et al., 1982).

Stifle disease and injury

When viewed from the side there should be obvious but not excessive angulation of the joints of the rear limb. The pastern, hock and stifle should align within an angle of 140° to 145°. Animals with excessively straight rear legs (post legs) are more prone to develop joint, tendon and hoof diseases. In the author's experience, large breed beef cattle with this conformation are more likely to develop cartilage disease in the stifle than more moderate conformation cattle.

Stifle injury or disease is a significant cause of proximal hind limb lameness in bulls and may be highly correlated with hind limb conformation (Pentecost and Niehaus, 2014). Bulls with little angulation in the tibio-tarsal and femoraltibial joints are more likely to sustain injury and osteoarthritis in those joints. Straightness in the tarsocrural joint predisposes straightness of the stifle, which leads to meniscal damage, joint instability with subsequent degenerative osteoarthritis and potentially leading to fraying or rupture of the cranial cruciate ligament (Bartels, 1975). The meniscus provides crucial joint surface congruency between the femur and tibial plateau contributing to friction reduction within the joint (Frithian *et al.*, 1990). Bulls most commonly develop medial meniscal damage associated with collateral ligament rupture (Nelson *et al.*, 1990). These injuries may induce the bull to spend more time in sternal recumbency thereby leading disrupting normal testicular thermoregulation. In addition, affected bulls may be reluctant to mount for breeding or semen collection.

Stifle injuries are common in cattle and one or more structures may be involved (Ducharme, 1996). Of the common injuries, rupture of the collateral ligament produces the least degree of lameness. Cattle with this condition are slightly lame and the injury may be easily diagnosed by watching them walk away from you. There will be medial-to-lateral instability and the stifle will deviate either medially or laterally, toward the affected side when the animal is full weight bearing. When the animal adequately restrained, place fingers of one hand on the medial aspect of the stifle joint while abducting the lower limb. If the medial collateral ligament is torn, there will be excessive joint space while the leg is abducted. With the fingers of one hand placed on the lateral aspect of the stifle the lower limb is adducted, there will be excessive motion if the lateral collateral ligament is torn.

Meniscal tears cause the next most severe lameness in cattle. The most common injury is similar to other species in that the posterior horn of the medial meniscus is injured more commonly than the lateral meniscus. With acute injury, there will be noticeable lameness and there may be evidence of joint effusion. The injury appears to occur more commonly in heavily muscled beef bulls than in other cattle. There may be an audible or palpable 'click' during the weight-bearing portion of the stride. The mass of the animal usually precludes palpation of the classical anterior drawer sign as may be detected in dogs. However, many beef cattle will tolerate flexion of the affected limb whereby the veterinarian may be able to detect excessive motion in the stifle joint and perhaps grating of bony surfaces due to loss of articular cartilage.

The third common and most severe stifle injury is rupture of the anterior crucial ligament. This injury causes marked lameness and usually obvious joint effusion. The animal is very reluctant to bear weight on the affected limb. These injuries are discussed together as they all appreciably shorten the productive life of bulls. Animals with only collateral ligament tears develop degenerative joint disease due to joint instability and abnormal wear of joint surfaces. Animals with meniscal tears do likewise with the added risk of suffering cruciate ligament tears due to the atrophy of leg muscle that frequently rapidly accompanies this injury and more severe loss of stability of the stifle joint. Animals with cruciate ligament tears suffer severe joint instability, rapid muscle atrophy and frequently quickly suffer meniscal tearing and loss of articular cartilage.

Shoulder injuries in cattle

Shoulder injuries are relatively uncommon in breeding bulls. Fractures or bruises of the shoulder are occasionally

encountered in lightweight cattle while being worked in a chute. These injuries occur due to excessively wild or excited cattle and/or inadequate footing, maintenance or design of the working chute. Fractures of the scapula or humerus occasionally result from bulls fighting. These injuries are readily diagnosed by the degree of lameness and swelling accompanying the injury. In very light muscled cattle, such as dairy breeds, the spine of the scapula; humerus and shoulder joint are readily palpable which is rarely the case with heavily muscled beef cattle, especially beef bulls (Desrochers, 2017). In addition, due to the size and conformation of these animals, these bones and joints are quite difficult to obtain diagnostic quality radiographs (Wolfe, personal observation).

Influence of spinal pathology on bull fertility

Spinal osteophytosis or spinal injury may impair normal testicular thermoregulation and thereby normal sperm production. In one study the superficial perineal nerve and the caudal scrotal nerve of bulls was surgically transected and thermographic evaluations and breeding soundness evaluations according to the standards of the Society for Theriogenology were performed for 8 months following the nerve disruption (Chenoweth et al., 1992). Unilateral neurectomy caused loss of the normal thermal gradient, thermal patterns and thermal symmetry of the scrotum on both neurectomized and the non-neurectomized sides. All bulls had unsatisfactory semen quality for >3 months after the surgery. Readjustment of thermal patterns began as early as 6 weeks after surgery and all bulls returned to normal thermal patterns and testicular function 5 to 8 months postoperatively. This study confirmed that loss of sensorysympathetic tone causes thermal changes with loss of sympathetic tone causing excessive vasodilation and increased heat in the affected areas. The increased heat of the scrotum in neurectomized bulls led to testicular degeneration. The authors postulate that spinal injuries, rear leg lameness and scrotal injuries may cause extensive impairment of neurogenic thermoregulation, thus inducing testicular degeneration. This study also confirmed that nerve regeneration might occur leading to reversal of the neurogenic injury and return to breeding soundness (Purohit et al., 2007).

Other spinal conditions that impair normal fertility include spastic syndrome, also called stretches, krampfigkeit and crampy. This is a progressive neurologic disease of the spinal cord affecting spinal musculature and often progressing to involve the hind legs, hips and perhaps an extension of the head and neck during spastic episodes. The condition affects both dairy and beef breeds and is believed to be inherited by mode of an autosomal dominant with incomplete penetrance (Gentile and Testoni, 2006). These diseases, which causes episodic spasm of muscles of the hind limbs, hips and back, may or may not be associated with the impaired testicular function. Often affected bulls may spend more time lying down which may disrupt normal testicular thermoregulation. Even if the testicular function is not disrupted, due to the progressive nature of the disease, affected bulls may lose mobility and the ability to mount for breeding or semen collection.

Summary

Numerous physical conditions may cause subfertility in bulls. The reader is advised to seek additional resources for discussion of ocular disease, scrotal size and pathology, as well as abnormalities of the accessory sex gland that may impair fertility. Breeding bulls should be maintained with housing and nutrition that ensures optimal health and body condition. Numerous injuries and developmental or skeletal disorders may hinder the bull's ability to display libido and maintain normal scrotal thermoregulation to ensure the production of adequate numbers of motile, normal spermatozoa and to achieve erection, intromission and ejaculation. A thorough history and physical examination are crucial to determining causes of infertility.

Acknowledgments

The author wishes to thank the numerous faculty and residents in the Food Animal Services in the Department of Clinical Services at the Auburn University College of Veterinary Medicine who have assisted with case management over the past 40 years.

Declaration of interest

There are no conflict of interest with this manuscript.

Ethics statement

There are no compliance or ethics issues to declare for this manuscript.

Software and data repository resources

Data for this manuscript are only maintained on the author's personal computer.

References

Ahola JK, Foster HA, VanOverbeke DL, Jensen KS, Wilson RL, Glaze JB, Fife TE, Gray CW, Nash SA, Panting RR and Rimbey NR 2011. Survey of quality defects in market beef and dairy cows and bulls sold through livestock auction markets in the Western United States: I. Incidence rates1. Journal of Animal Science 89, 1474–1483.

Ashdown RR 1960. The adherence between the free end of the bovine penis and its sheath. Journal of Anatomy 94, 198–204.

Ashdown RR 1962. Persistence of the frenulum in young bulls. Veterinary Record 74, 1464–1468.

Ashdown RR 1970. Angioarchitecture of the sigmoid flexure of the bovine corpus cavenosum penis and its significance in erection. Journal of Anatomy 106, 403–404.

Ashdown RR and Coombs MA 1967. Spiral deviation of the bovine penis. Veterinary Record 80, 737–738.

Bartels JE 1975. Femoral-tibial osteoarthrosis in the bull: I. Clinical survey and radiological interpretation. Journal of American Veterinary Radiological Society 16, 159–173.

Barth AD and Bowman PA 1994. The sequential appearance of sperm abnormalities after scrotal insulation or dexamethasone treatment in bulls. The Canadian Veterinary Journal 35, 93–102.

Beckett SD and Wolfe DF 1998. Anatomy of the penis, prepuce, and sheath. In Large animal urogenital surgery (ed. DF Wolfe and HD Moll), pp. 201–209. Williams and Wilkins, Baltimore, MD, USA.

Beckett SD, Hudson RS, Walker DF and Purohit RC 1978. Effect of local anesthesia of the penis and dorsal penile neurectomy on the mating ability of bulls. American Journal of Veterinary Research 173, 838–839.

Beckett SD, Reynolds TM, Walker DF, Hudson RS and Purohit RC 1974a. Experimentally induced rupture of corpus cavernosum penis of the bull. American Journal of Veterinary Research 35, 765–767.

Beckett SD, Walker DF, Hudson RS, Reynolds TM and Vachon RI 1974b. Corpus cavernosum penis pressure and penile muscle activity in the bull during coitus. American Journal of Veterinary Research 35, 761–764.

Bellenger CR 1971. A comparison of certain parameters of the penis and prepuce in various breeds of beef cattle. Research in Veterinary Science 12, 299–304.

Berry SL 2001. Diseases of the Digital Soft Tissues. Veterinary Clinics of North America: Food Animal Practice 17, 129–142.

Brito LFC, Silva AEDF, Barbosa RT and Kastelic JP 2004. Testicular thermoregulation in Bos indicus, crossbred and Bos taurus bulls: relationship with scrotal, testicular vascular cone and testicular morphology, and effects on semen quality and sperm production. Theriogenology 61, 511–528.

Carson RL and Wenzel JGW 1995. Over a thousand BSE's using the new form. In Society for Theriogenology, San Antonio, TX, USA, pp. 65–72.

Chenoweth PJ 1983. Sexual behavior of the bull: a review. Journal of Dairy Science 66, 173–179.

Chenoweth PJ, Spitzer JS and Hopkins FM 1992. A new bull breeding soundness evaluation form. In Annual Conference Society for Theriogenology, San Antonio, TX, USA, pp. 63–70.

Desrochers A 2017. Diagnosis and prognosis of common disorders involving the proximal limb. Veterinary Clinics of North America: Food Animal Practice 33, 251–270.

Desrochers A, Anderson DE and St. Jean G 2008. Surgical diseases and techniques of the digit. In Veterinary clinics of North America: food animal practice. (ed. DE Anderson and MD Meisner), pp. 535–550. Elsevier, Philadelphia, PA, USA.

Ducharme NG 1996. Stifle injuries in cattle. Veterinary Clinics of North America: Food Animal Practice 12, 59–84.

Elmore RG 1981. Surgical repair of bovine persistent penile frenulum. Veterinary Medicine Small Animal Clinician 76, 701–704.

Elmore RG, Brever J and Youngquist RJ 1978. Breeding soundness evaluations in 18 closely related inbred Angus bulls. Theriogenology 10, 355–363.

Frithian DC, Kelly MA and Mow VC 1990. Material properties and structurefunction relationships in the menisci. Clinical Orthopaedics and Related Research 252, 19–31.

Gentile A and Testoni S 2006. Inherited diseases of cattle: a selected review. Slovenian Veterinary Research 43, 17–29.

Gilbert RO 1989. The diagnosis of short penis as a cause of impotentia coeundi in bulls. Theriogenology 32, 805–815.

Greenough P 1987. Pododermatitis circumscripta (ulceration of the sole) in cattle. Agri-Practice 6, 17–22.

Greenough PR 1972. Conformation, growth, and heritable factors. In Lameness in cattle (ed. PR Greenough), pp. 71–86. W.B. Saunders, Philadelphia, PA, USA.

Hoblet KH and Weiss W 2001. Metabolic hoof horn disease claw horn disruption. Veterinary Clinics of North America: Food Animal Practice 17, 111–127.

Kastelic JP, Coulter GH and Cook RB 1995. Scrotal surface, subcutaneous, intratesticular, and intraepididymal temperatures in bulls. Theriogenology 44, 147–152.

Larson LL and Kitchel RI 1958. Neural mechanisms in sexual behavior. II. Gross neuroanatomical and correlative neurophysiological studies of the external genitalia of the bull and ram. American Journal of Veterinary Research 19, 853–865.

Lischer C, Ossent P, Raben M and Geyer H 2002. Suspensory structures and supporting structures of the third phalanx of cows and their relevance to development of typical sole ulcers (Rusterholz ulcers). Veterinary Record 151, 694–698.

McEntee K 1950. Fibropapillomas of the external genitalia of cattle. Cornell Veterinarian 40, 304–312.

Moll HD, Wolfe DF and Hathcock JT 1993. Caversonography for diagnosis of erection failure in bulls. Compendium of Continuing Education Practicing Veterinarian 161 15, 1160–1164.

Mysinger PD, Wolfe DF and Redding R 1994. Sensory nerve conduction velocity of the dorsal penile nerves of bulls. American Journal of Veterinary Research 55, 898–900.

Nelson DR, Huhn JC and Knelle SK 1990. Peripheral detachment of the medial meniscus with injury to the medial collateral ligament in 50 cattle. Veterinary Record 127, 59–66.

Parker WG, Braun R, Bean B, Hillman R, Larson L and Wilcox CJ 1987. Avulsion of the bovine prepuce from its attachment to the penile integument during collection with an artificial vagina. Theriogenology 28, 237–256.

Pentecost R and Niehaus A 2014. Cranial cruciate ligament, meniscus, upward fixation of the patella. Veterinary Clinics of North America Food Animal 30, 265–281.

Persson Y, Söderquist L and Ekman S 2007. Joint disorder; a contributory cause to reproductive failure in beef bulls? Acta Veterinaria Scandinavica 49, 31.

Purohit RC, Hudson RS, Riddell MG, Carson RL, Wolfe DF and Walker DF 1985. Thermography of the bovine scrotum. American Journal of Veterinary Research 46, 2388–2392.

Purohit RC, Carson RL, Riddell MG, Brendemeuhl J, Wolfe DF and Pablo LS 2007. Peripheral neurogenic thermoregulation of the bovine scrotum. Thermology International 17, 137–139.

Reiland S, Stromberg B, Olsson SE, Dreimanis I and Olsson IG 1978. Osteochondrosis in growing bulls. Pathology, frequency and severity on different feedings. Acta Radiologica Supplement 358, 179–196.

Seidel GE and Foote RF 1969. Motion picture analysis of ejaculation in the bull. Journal of Reproduction and Fertility 20, 313–317.

Waites GMH 1970. Temperature regulation and the testis. In The testis (ed. GW Johnson AD and Vandemark NL), pp. 241–279. Academic Press, New York, NY, USA.

Walker DF and Young SL 1979. The fascia late implant technique for correcting bovine penile deviations. In Society for Theriogenology, Mobile, AL, USA, pp. 99–102.

Watson JW 1964. Mechanism of erection and ejaculation in the bull and ram. Nature 204, 95–96.

Weisbrode SE, Monke DR, Dodaro ST and Hull BL 1982. Osteochondrosis, degenerative joint disease, and vertebral osteophytosis in middle-aged bulls. Journal of the American Veterinary Medical Association 181, 700–705.

Wolfe DF 2015a. Restorative surgery of the prepuce. In Bovine reproduction (ed. RM Hopper), pp. 142–154. John Wiley and Sons, Ames, IA, USA.

Wolfe DF 2015b. Restorative Surgery of the Penis. In Bovine reproduction (ed. RM Hopper), pp. 154–171. John Wiley and Sons, Ames, IA, USA.

Wolfe DF and Carson RL 1998. Juvenile anomalies of the penis and prepuce: bulls. In Large animal urogenital surgery (ed. DF Wolfe and HD Moll), pp. 233–235. Williams and Wilkins, Baltimore, MD, USA.

Wolfe DF, Beckett SD, Carson RL, Powe TA and Pugh DG 1998. Acquired qconditions of the penis and prepuce. In Large animal urogenital surgery (ed. DF Wolfe and HD Moll), pp. 237–272. Williams and Wilkins, Baltimore, MD, USA.

Young S, Hudson R and Walker DF 1977. Impotence in bulls due to vascular shuns from the corpus cavernosum penis. Journal of American Veterinary Medical Association 171, 643–648.