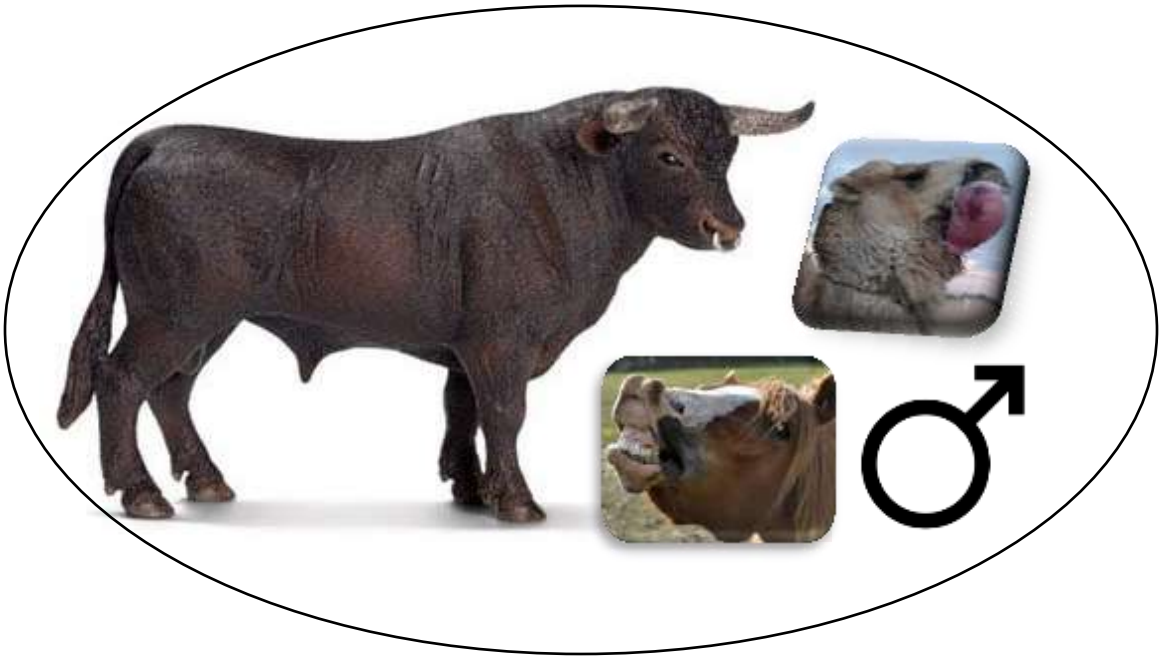
South Valley University
Faculty of Veterinary Medicine
Department of Theriogenology, Obstetrics, and
Artificial Insemination

ANDROLOGY IN FARM ANIMALS



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Introduction:

The male animal is half the herd. And with the rapid adaptation of the technique of artificial insemination, the problem of fertility and infertility in bulls is increasingly felt with. It is most important to investigate and evaluate the breeding animals before they are put into service and discard those which might cause serious losses in the herd. From an economical point of view it is not possible to maintain a sterile animal, which has lost his reproductive capacity or an infertile one, which has disturbances in his spermatogenesis or sexual functions.

Infertility or sterility is not a disease in itself, but it can be the result of various errors in management and large group of diseases mainly of genital and reproductive origins. Most of the case of infertility or sterility is caused by hereditary (genetic) and/or environmental factors.

Ontogenesis: see fig. (1), (2)
(Development of male genitalia)

As discussed in the part of gynecology, fetal reproductive system consists of:

- Two sexually undifferentiated gonads “primary sex cords” from which the ovaries or testes are originate.
- Two pairs of ducts (Mullerian “paramesonephric” and wolfian “mesonephric” ducts) that give rise to the duct system starting from rete testis to the urethra.
- Genital tubercle, the genital tubercle is the origin of the copulatory organ “penis”
- Vestibular folds, from which the scrotum is formed. It originates from the 2 germinal ridges on dorsal side of abdominal cavity

<u>In male animals</u>, testicular androgens and mullerian inhibiting factor stimulates development of wolfian ducts and inhibits the development of mullerian ducts.

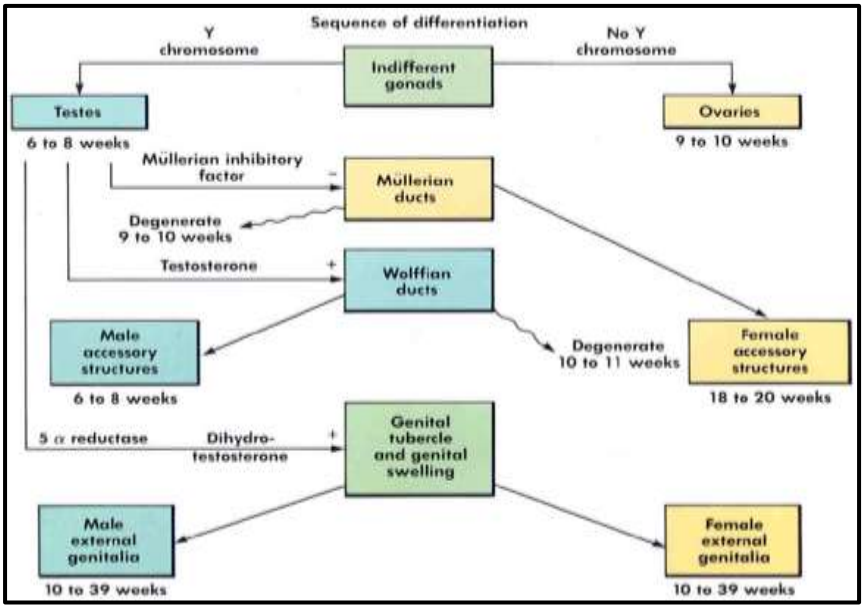


Fig. 1: Scheme showing the sequence of sex and reproductive organs differentiation under the effect of Y chromosome and male hormones/factors.

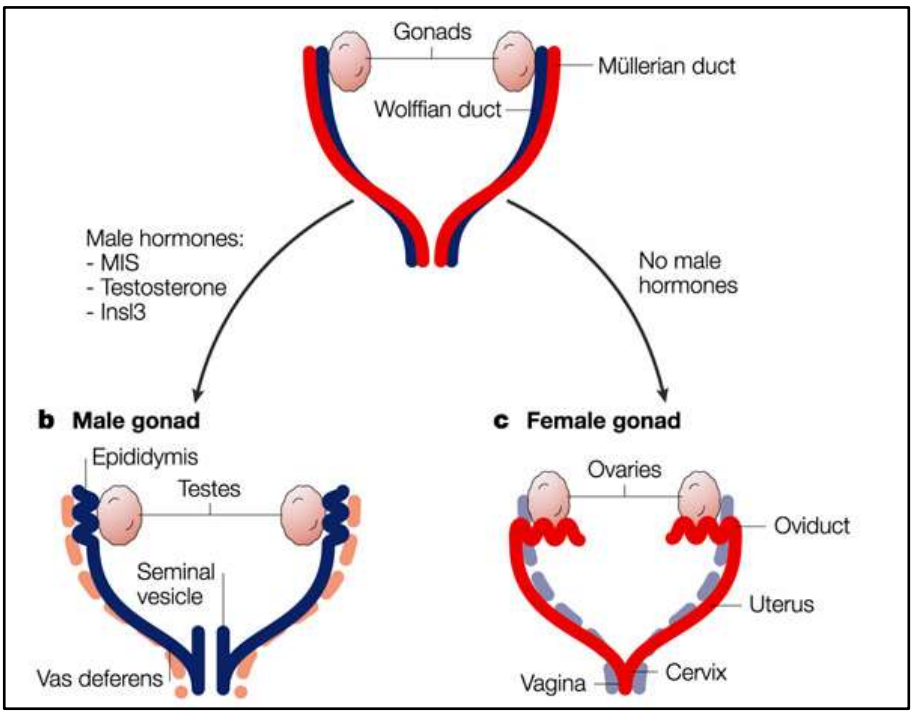


Fig. 2: Diagram showing the embryonic development of male and female genital systems.

Females are considered the “fundamental” sex that is, without much chemical prompting, all fertilized eggs (conceptus) would develop into females. To become a male, an individual must be exposed to the cascade of factors initiated by a single gene on the male Y chromosome. This is called the SRY (Sex-determining Region of the Y chromosome, see Fig. (3). because females do not have a Y chromosome, they do not have the SRY gene. Without a functional SRY gene, an individual will be female see Fig. (1).

In both male and female embryos, the same group of cells has the potential to develop into either the male or female gonads; this tissue is considered bipotential. The SRY gene actively recruits other genes that begin to develop the testes, and suppresses genes that are important in female development. As part of this SRY-prompted cascade, germ cells in the bipotential gonads differentiate into spermatogonia. Without SRY, different genes are expressed, oogonia form, and primordial follicles develop in the primitive ovary.

Soon after the formation of the testis, the Leydig cells begin to secrete testosterone. Testosterone can influence tissues that are bipotential to become male reproductive structures. For example, with exposure to testosterone, cells

that could become either the glans penis or the glans clitoris form the glans penis. Without testosterone, these same cells differentiate into the clitoris.

Not all tissues in the reproductive tract are bipotential. The internal reproductive structures (for example the uterus, uterine tubes, and part of the vagina in females; and the epididymis, ductus deferens, and seminal vesicles in males) form from one of two rudimentary duct systems in the embryo. For proper reproductive function in the adult, one set of these ducts must develop properly, and the other must degrade. In males, secretions from sustentacular cells trigger a degradation of the female duct, called the Müllerian duct. At the same time, testosterone secretion stimulates growth of the male tract, the Wolffian duct. Without such sustentacular cell secretion, the Müllerian duct will develop; without testosterone, the Wolffian duct will degrade. Thus, the developing offspring will be female. Different genitalia of male and female fetuses develop from the same tissues in the embryo.

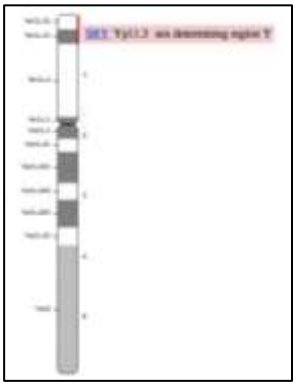


Fig. 3: Diagram showing the chromosome Y indicating the sex determining region Y (SRY)

1. Functional Anatomy of male genital system:

Good reproductive performance of a bull is necessary to obtain a high percent calf crop when natural service is used for breeding. A bull must be fertile, capable and willing to mate a large number of cows during a short breeding season for optimum production. A basic knowledge of the reproductive tract is beneficial for improved management. An understanding of the bull's reproductive system will also help the better understand of the breeding soundness examinations, reproductive disorders/problems and breeding impairments.

The reproductive tract of the bull consists of

1 -Primary male sex organs:

It consists of two testes in an external scrotal sac

2 -Secondary sex organs:
It includes the efferent ducts, epididymis, and ductus deference

3- Accessory sex organs (glands):
The accessory sex organs include the prostate gland, two seminal glands, and two bulbourethral glands.

These organs work in concert for formation, maturation and transport of spermatozoa, which are eventually deposited in the female reproductive tract. The secondary sex organs are the **efferent ducts, epididymis, vas deferens** and **penis**. The three accessory sex glands include the **seminal vesicles, prostate** and **bulbourethral gland** (Cowper's gland). This basic anatomy is illustrated in Fig. (4).

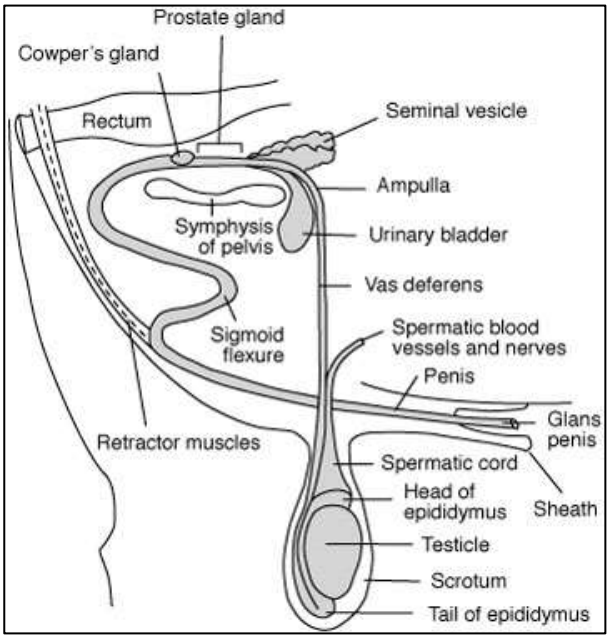


Fig. 4: Drawing of the reproductive tract of the bull (from Nebraska Guide G80-536)

1.1. Testicle:

The testicle is located outside the body cavity in the scrotum and has two vital functions: producing the spermatozoa, and producing the male hormone, testosterone. Location of the testicles exterior to the body cavity is essential for normal sperm formation, which occurs only at 4 degrees to 5 degrees below body temperature. The scrotum provides physical protection to the testicle and helps regulate the temperature for optimum spermatozoa development. This regulation is done by coordination of three structures: a temperature-sensitive layer of muscle (tunica dartos) located in the walls of the scrotum, which relaxes when hot and contracts when cold; the external cremaster muscle within the spermatic cord, which controls the proximity of the testicle to the body by lengthening or shortening depending on environmental temperature; and a counter-current temperature exchange regulated by a blood flow process known as the *pampiniform plexus,* which is a coil of testicular veins that provide an effective mechanism for cooling arterial blood entering the testicle and transferring its heat to the venous blood leaving the testicle.

Anatomical descriptions of testicle in farm animals are:

- Ovoid in shape suspended in scrotal pouch by spermatic cord
- Testicular volume measured as “length*breadth*depth*0.52”
- Two testes are nearly equal size and freely movable within the scrotum
- The upper, posterior, and lower pole occluded with head, body, and tail of epididymis.
- Firm consistency except in boar it has soft consistency
- Testes are covered by firm connective tissue capsule “tunica albuginea”

Position:

Inguinal: in ruminant and equine

Perineal: in camel bull, dog, Tom-cat, and boar .

Direction/orientation: see fig. (5).

Vertical: bull, ram, buck with epididymis head dorsally

Horizontal: stallion, donkey with epididymis head cranially

Oblique: camel, canine

Inverted: boar with epididymis head ventrally

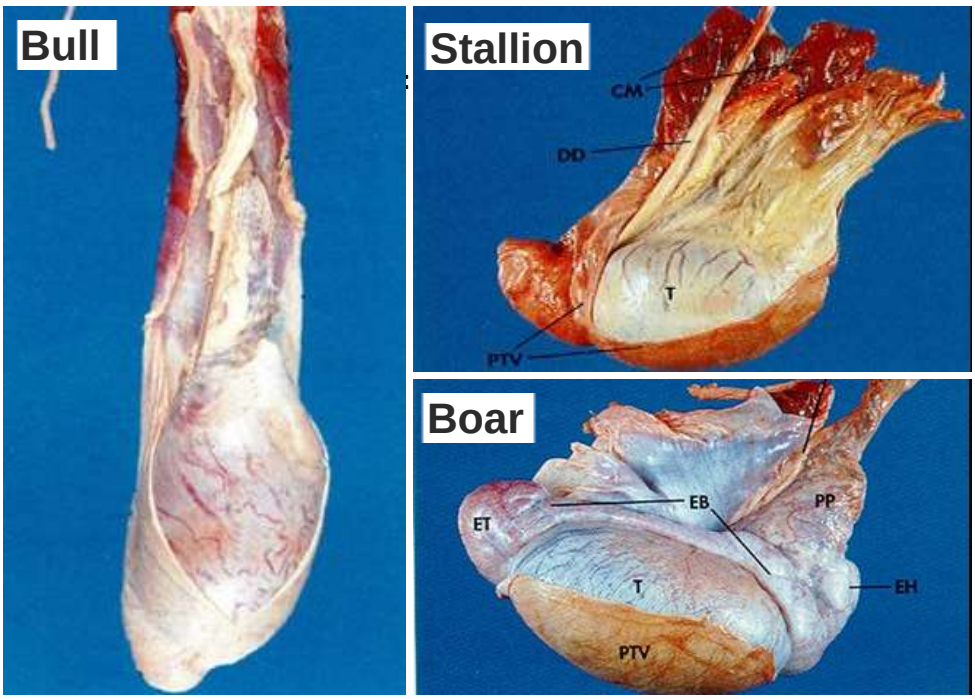


Fig. 5:
Showing the direction of testicles in different animals

Parts of testes:

1. **Testicular capsule:**

It is the outer layer of whitish connective tissue with smooth muscle fibers. It undergo contraction and relaxation to facilitate pumping of spermatozoa into rete testis and efferent ductules

2. **Testicular Parenchyma "Functional layer":** see fig. (6), (7).
   - **Seminiferous tubules:** surrounded by peritubular myoid cells. Contains 2 types "spermatogonia and sertoli cells"
   - **Leydig cells:** in-between seminiferous tubules

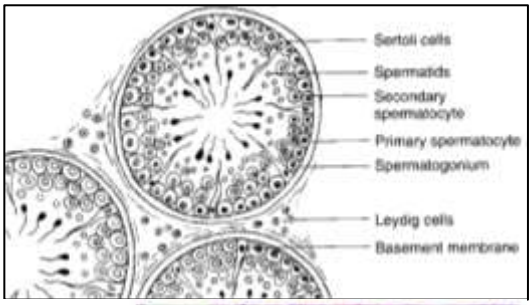


Fig. 6: Cross section in testis showing the seminiferous tubules and interstitial tissue containing leydig cells.

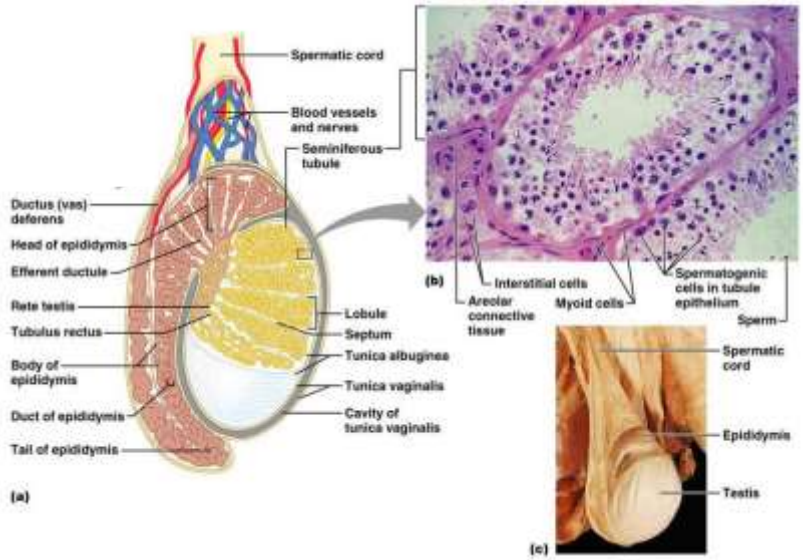


Fig. 7: Diagram showing the testis, epididymis, spermatic cord with histological cross section of the seminiferous tubules and the interstitial tissues.

The testicle contains many long, tiny, coiled tubes known as *seminiferous tubules,* within which the sperm are formed and begin to mature. Scattered throughout the loose connective tissue surrounding the seminiferous tubules are many highly specialized cells, the *interstitial cells of Leydig,* that produce testosterone. There are hundreds of individual seminiferous tubules in the body of the testicle which unite with one another to form a few dozen tubules that exit from the testicle and pass into the *epididymis.*

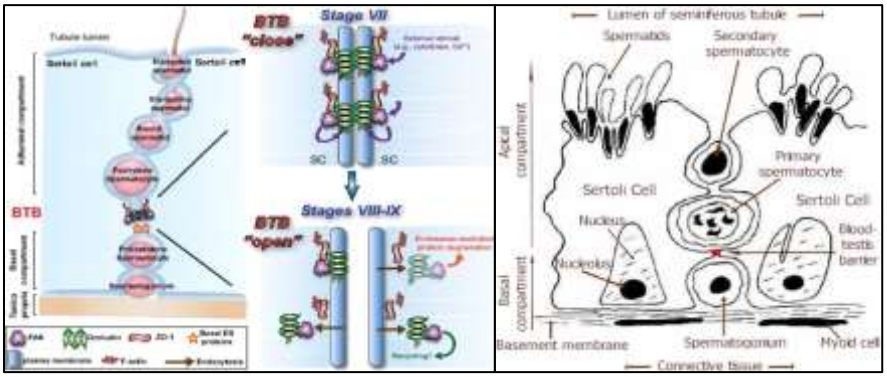


Fig.8: Diagram showing the Sertoli cells and the blood-testis barrier formed by the sertoli cells junctions.

Functions of testes:

1. **Exocrine function:** Spermatogenesis will be discussed in details, later.
2. **Endocrine Function:** Hormonal; peptide “inhibin”
   Steroid “testosterone”

It will be discussed with the hormonal regulation of male reproduction, later.

Testicular descent: see fig. (9).

1 -Intact fetal hypothalamic-pituitary gonadal axis stimulates the secretion of androgens.

2-Androgens stimulate secretion of descendin from testes.

3-Descendin stimulate selective growth of gubernaculum.

4 -Androgens stimulate regression of gubernaculum.

5- Descent of testes into and through inguinal canal towards the scrotal cavity occurs due to intra-abdominal pressure and regression of gubernaculum.

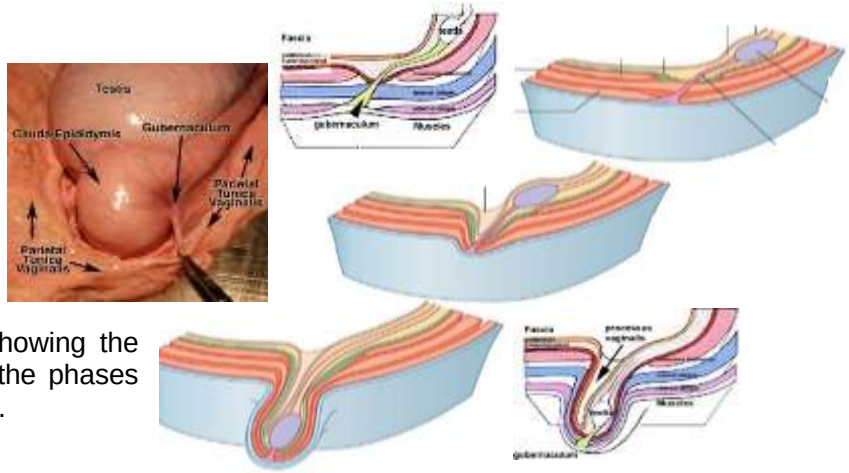


Fig. 9: Diagram showing the gubernaculum and the phases of testicular descent.

The testicle initially forms high in the abdominal cavity near the lower thoracic vertebral level. At approximately 5 weeks gestation, during formation of the genital ridge, the caudal mesonephros and the future gubernaculum are connected near the internal inguinal ring. Upon testis formation, a dorsally located **cranial suspensory ligament (CSL)**, or cranial mesonephric ligament, connects the upper mesonephros to the diaphragm. Together, the cranial and caudal ligaments loosely stabilize the position of the testis. As the fetus elongates and the abdominal cavity grows differentially, the CSL elongates, and its cranial aspect, mesonephros, regresses, and the testis is held near the inguinal ring. As a result, the relative position of the testis becomes more caudal, following a path toward the junction of the mesonephros and gubernaculum. Ultimately, the transabdominal descent of the testis is usually complete by approximately 10–15 weeks gestation, with the testis reaching the inguinal region.

Early in the second trimester, there is a **surge in testosterone and insulin-like factor 3 (INSL3) productions by fetal Leydig cells**. This generally occurs after transabdominal descent and is accompanied by a shortening and swelling or outgrowth of the gubernaculum that is thought to draw the testis closer to the internal ring and create space in the inguinal canal for the testis to pass. **Trans-inguinal descent** of the testis usually occurs between weeks 22 and 27 of gestation. During the inguinoscrotal phase of testis migration, intra-abdominal pressure is thought to provide the main driving force for descent. However, evidence in rodents suggests the genitofemoral nerve (GFN) produces **calcitonin gene-related peptide (CGRP)**, which stimulates contractions in the muscle fibers in and around the gubernaculum, further propagating this stage of testicular descent.

During the **inguino-scrotal phase**, the processus vaginalis extends caudally as an outpouching of the parietal peritoneum, increasing in length as the testis descends to its final location. Upon completion of inguinoscrotal descent, the processus vaginalis closes off from the peritoneum, forming the tunica vaginalis of the testis. During the remainder of the pregnancy, much of the gubernacular tissue involutes as the testis settles in the dependent part of the scrotum between 32 weeks gestation and birth.

There is a contradictory evidence, and there are questions regarding gonadal descent. It is unclear by what exact mechanisms androgens affect intra-abdominal descent. **INSL3** likely primarily promotes transabdominal descent by early effects on the gubernacular development, while androgens contribute to CSL regression.

During inguino-scrotal descent, testosterone is thought to be the dominant factor resulting in masculinization of the GFN with subsequent gubernacular growth and contractions. While MIS was once thought to be the most important factor in transabdominal descent of the testis, now, there is conflicting evidence as to the importance of MIS' role in testicular descent.

One or both testicles occasionally fail to descend into the scrotum during embryological development and are retained in the body cavity. This condition is known as *chryptorchidism.* Hormone production by chryptorchid males is near normal and the male develops and behaves like a normal male, but will generally be sub-fertile. This condition is genetically inherited; therefore such males should not be used for breeding.

Spermatogenesis:

It takes 54 days in bull, 49 days in ram, and 35 days in boar

Transit of sperm from efferent ductules to the ejaculate takes 10-12 days bull and ram, or 15 days stallion, dog and boar

A. Spermatocytogenesis: see fig. (10).

It lasts approximately about 13-32 days

1. <u>Mitotic division:</u>

Spermatogonia undergo four mitotic divisions that result in 16 primary spermatocytes "each spermatocyte has 2 N"

2. <u>Meiotic division:</u>

<u>1st meiosis:</u> Primary spermatocytes "2N" undergo meiotic division and result in two secondary spermatocytes "1N contains sister chromatids" either X or y.

<u>2nd meiosis:</u> Secondary spermatocytes "1N" undergo a 2nd meiotic division "division of chromosomes into two single chromatids" results in two spermatids.

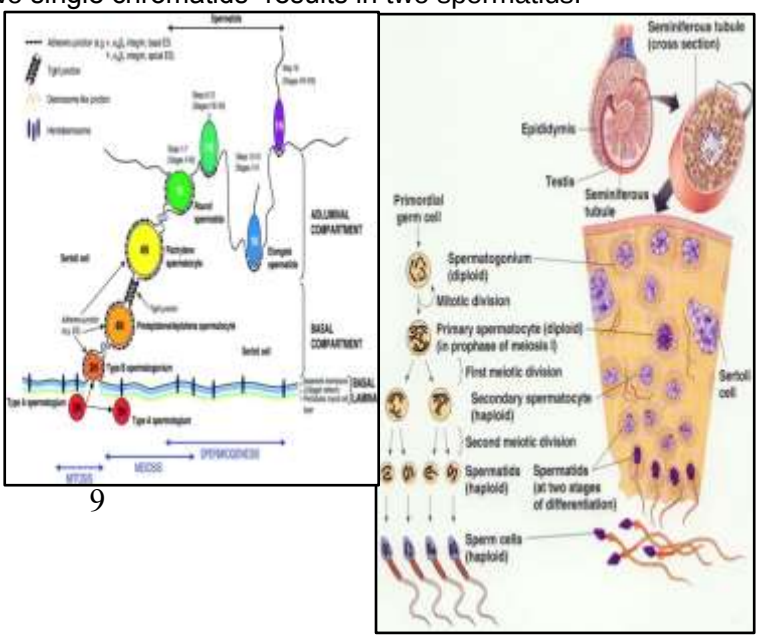


Fig. 10: Diagram showing the process of spermatogenesis. And illustrate the relation between blood-testis barrier, and the different stages of spermatocytogenesis.

B- Spermiogensis: see fig. (11).

Spermiogensis includes some dramatic morphological changes of rounded spermatids to spermatozoa until spermiation occurs.

Spermiogensis includes:

1- Condensation of nucleus.

2- Formation of acrosomal cap.

The acrosomal cap is formed from the Golgi apparatus by its modification.

3- Development of Middle piece.

The development of the middle piece includes migration of centrioles to post-nuclear region to form axon. And the mitochondria concentrated around flagellum forming the middle piece .

4- Development of tail.

The distal centriole acts as a cytoskeleton to form the contractile part of the tail.

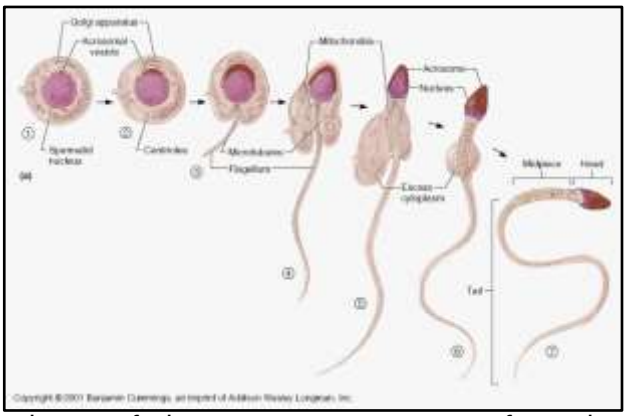


Fig. 11: Diagram showing the process of spermiogensis from rounded spermatids to mature long spermatozoa.

C- Spermiation:

Spermiation means the release of the mature spermatozoa from the surrounding membrane of the sertoli cells towards the lumen of the seminiferous tubules under the effect of the LH hormone.

1.2. Spermatic cord:

It suspends the testes in the scrotum through the inguinal ring “between abdominal and scrotal cavities”

Parts of spermatic cord:

1- Vascular part.

1. Testicular artery: it originates from the abdominal aorta as a highly coiled long artery.

2. Testicular vein: into caudal vena cava

Pampiniform plexus it is a vascular structure that formed of intertwining of the testicular artery and vein which act as countercurrent heat exchange that cool the blood toward the tested. See fig. (12).

3. Testicular lymphatics

4. Testicular nerve plexus.

2- Avascular part.

1. Cremastric muscle.

It is a continuation of internal oblique abdominal muscle.

It supports the testes and aid in control of the testicular temp by - elevation and lowers of testis and facilitates blood flow in pampiniform plexus

2. Ductus deferens.

Functions of spermatic cord:

- Heat exchange action. See fig. (13).
- Provide vascular and nerve supply to testes.
- House of ductus deference and cremastric muscle.

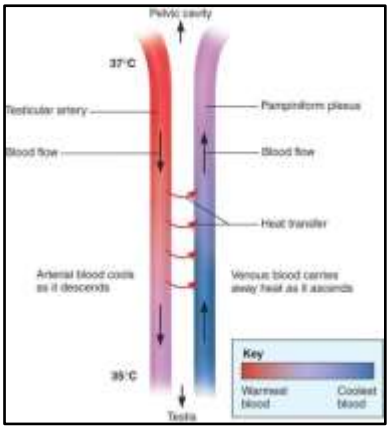


Fig. 12 to the right: Diagram showing the contents of the spermatic cord.

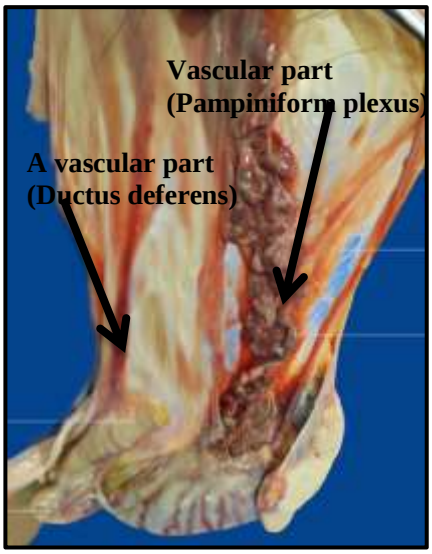


Fig. 13, to the left: Diagram showing the heat-exchange action.

1.3.Scrotum:

It is a pendulous sac of two symmetrical pouches containing the testes.

Inguinal: in bull, ram, buck, and stallion.

Perineal: in camel, boar, dog, and cat.

Layers of Scrotum: see fig. (15).

1. **Skin "outer layer":** it is a fine elastic layer that contains large number of sweat and sebaceous glands.
2. **Tunica dartos:** it is closely lining the skin and divide scrotum into two pouches .
3. **Scrotal fascia:** it consists of 3 elastic layers external, internal spermatic , and internal cremastric fascia
4. **Tunica vaginalis:** it is double layers of T. vaginalis propria that covers the testes and epididymis, and T. vaginalis reflexia which is the lining of the scrotal cavity. see fig. (14).
5. **External cremastric muscle:** it originates from the internal oblique abdominal muscles.

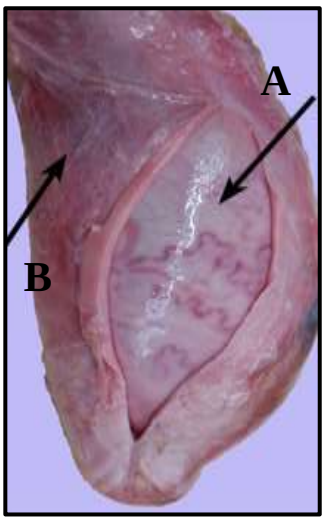


Fig. 14, to the right: Diagram showing visceral layer (A), and parietal layer (B) of T. Vaginalis.

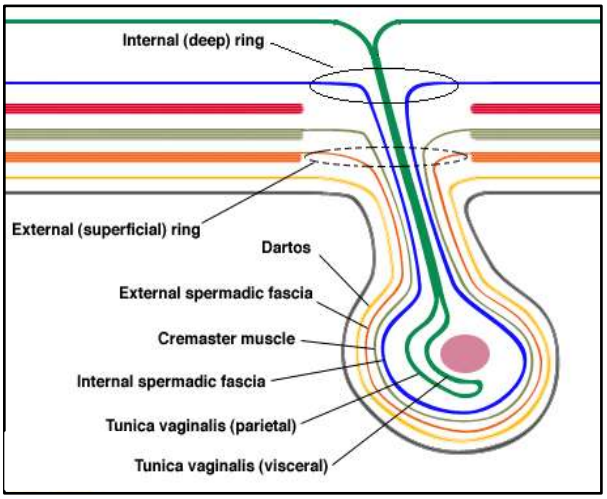


Fig. 15: Diagram showing the different layers of the scrotum

Functions of Scrotum:

1. Support and protect testes.
2. Thermoregulation to maintain spermatogenesis.
3. This mechanism is maintained at the time of puberty.
4. In response to environmental temperature T. dartos, cremastric muscle, and T. vaginalis.
5. In cold weather: it becomes short, corrugated “wrinkled” and close to abdomen.
6. In hot weather: it becomes elongated, flaccid and away from abdomen.
7. Increase the secretion of sweat and sebaceous glands and evaporation of sweating that allows cooling of scrotum.
8. Expansion of scrotum in hot weather increases the surface area of the scrotum that allows more heat exchange to lower the temperature inside the scrotal cavity.

- **Secondary organs of reproduction (tubular part):**

1.4. Epididymis:

The epididymis is a compact, flat, elongated structure closely attached to one side of the testicle. It is divided into three regions, the head, body and tail. The many tubules entered the head of the epididymis from the testicle unite to form a single tubule of 33 to 55 meters in length, and 13-40 gm in its weight. This tubule is convoluted and packed into the 6- to 8-inch epididymis. Four major functions occur in the epididymis, including the transport of the developing sperm cells from the testicle to the *vas deferens*; the concentration of the sperm by absorption of surplus fluids; the maturation of the developing spermatozoa; and the storage of viable sperm cells in the epididymis tail. If sexual activity is slowed, resorption of sperm cells from the epididymis tail occurs.

The epididymis serves as an outlet for all the sperm produced in the testicle and any blockage of this tube will cause sterility. Temporary blockage due to swelling following an injury or infection (epididymitis) will result in short-term infertility. If the swelling or infection results in formation of scar tissue in the tubule, it may permanently block the passage of sperm. If blockage occurs in both epididymides, the bull will no longer be useful as a breeder. Surgical removal of the tail of the epididymis (epididectomy) is frequently used as a means of sterilization for teaser bulls for estrus detection. Epididectomized bulls will still service cows in the usual manner, but will not deposit sperm in the female reproductive tract.

In conclusion, it consists of 3 parts: see fig. (16).

1. Head “Caput” It begins by 12-15 efferent ductules at its posterior part about 1/3 of the head .
2. Body “Corpus” It extends along posterior part of testis
3. Tail “Cauda” It terminates in vas deferens. In stallion, it has loose attachment or even separated from testis.

Head and tail can be palpated. Head is more firm than testes while tail varies from soft to firm

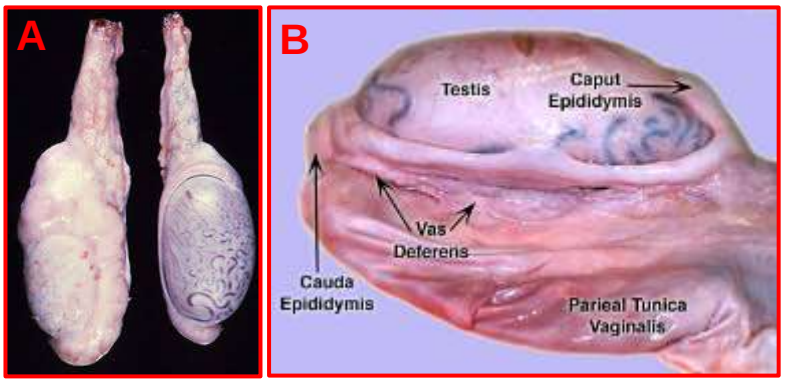


Fig. 17: Diagram showing the epididymis in relation to the testes and spermatic cord (A), and the parts of the epididymis (B)

Functions of epididymis:

1. Transportation of spermatozoa.

It is maintained by peristaltic movement and movement of beating cilia. Cilia are higher in number in head and body. In case of epididymitis, there is increase in the number of detached cilia “medusa”

2. Storage of spermatozoa.

- 30-35 % in the head and body of the epididymis.
- 50-55 % in the tail of the epididymis
- 10-15 % is stored in ampulla ductus deference.

3. Maturation of spermatozoa.

Epididymis secrets glycerol phosphoryl choline and lipoprotein that aids in sperm maturation

4. Absorption of dead spermatozoa.

1.5. Vas/ductus deferens:

The vas deferens, also known as ductus deferens, emerges from the tail of the epididymis till reach the colliculus seminalis as a cord-like straight tubule and passes as part of the spermatic cord through the inguinal ring into the body cavity. Spermatozoa are transported further along the reproductive tract to the pelvic region through the vas deferens by contraction of the smooth muscle tissue surrounding this tubule during ejaculation. Bulls may also be sterilized by a vasectomy in which a section of the vas deferens is removed so that sperm cannot pass to the outside of the body.

Ampulla ductus deferens:

It is a fusiform enlargement before the end of the vas deferens. It secrets fructose and citric acid.It passes dorsal to the urinary bladder, penetrate the body of the prostate gland and open in to the urethra at the colliculus seminalis through the ejaculatory duct.

1.6. Urethra:

The two vas deferens eventually unite into a single tube, the urethra, which is the channel passing through the penis. The urethra in the male serves as a common passageway for semen from the reproductive tract and urine from the urinary tract.

1.7. Penis and prepuce: see fig. (17).

The *penis* is the copulatory organ of insemination. Spongy-type material within the penis is filled with blood during sexual arousal, resulting in erection of the organ. The end of the penis is the glans penis and is richly supplied nerves, which are stimulated during copulation to induce ejaculation. Impairments of the glans penis may exist and should be detected during a fertility exam.

The *sigmoid flexure* is an anatomical structure that provides a means by which the penis is held inside the sheath except during time of service. Strong retractor muscles hold the penis in the "S" shaped configuration. Occasionally these muscles are too weak to function properly and a portion of the penis and

sheath lining protrude at all times. This exposes the male to the danger of injury and this characteristic should be avoided when selecting a herd bull.

Type of penis:

- **Musclocavernous penis:** as in horse, donkey**,** these animals show slow erection and ejaculation.
- **Fibroelastic penis:** as bull, buffalo bull, camel bull, ram, buck, and boar, these animals shows rapid erection and ejaculation.

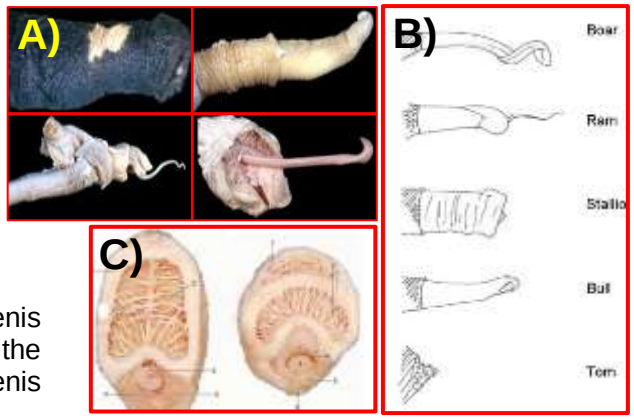


Fig. 17: Diagram showing the glans penis in different animals A), and B), and the cross section of the musclocavernous penis (right) and fibroelastic one (left).

1.8. Accessory genital glands: see fig. (18).

- **Prostate gland.**
- **Bulbourethral "Cowper's" glands**
- **Seminal vesicles.**

Two of the accessory glands are found in the general region where the vas deferenses unite to become the urethra. Secretions from these glands make up most of the liquid portion of the semen. In addition, the secretions activate the sperm to become motile. The seminal vesicles consist of two lobes about 4 to 5 inches long, each connected to the urethra by a duct. The *prostate gland* is located at the neck of the urinary bladder where it empties into the urethra. The prostate is relatively small in the bull, as compared to other species, and does not produce a very large volume secretion.

The third accessory gland, the *Cowper's gland, is* small, firm glands located on either side of the urethra. The clear secretion that often drips from the penis during sexual excitement prior to service is largely produced by these glands and serves to flush and cleanse the urethra of any urine residue that may be harmful to spermatozoa.

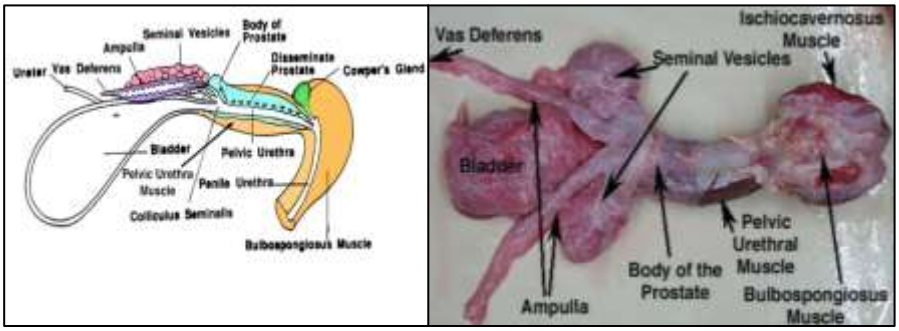


Fig. 18: showing the male pelvic genitalia including accessory genital glands.

1.8.1. Seminal Vesicles: see fig. (19)

- They are two compact lobulated glands which are located dorsolateral to the ampullae on the pelvic floor.
- Seminal vesicles measure 10-15 cm Length, 3-4 cm Breadth, and 1-2 cm Thickness.
- They can be palpated rectally with a tense firm consistency.
- Each has a main excretory duct with tree-like branching interiorly and excretory duct posteriorly at the colliculus seminalis beside the ejaculatory duct.

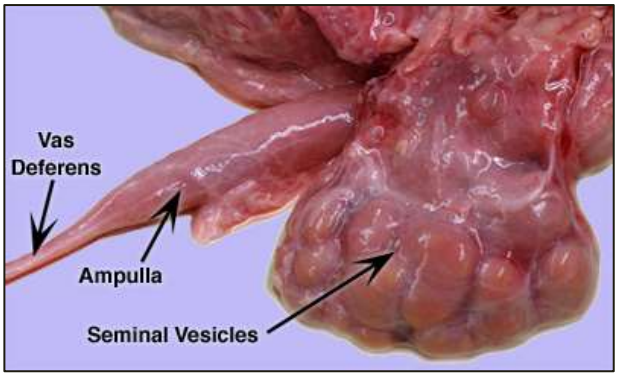


Fig. 19: showing the seminal vesicle with its grapes-like lobulations, in bull.

Function of seminal glands:

1. Secrete the main volume of seminal plasma (>50%) which acts as a vehicle to sperm activity and motility.
2. Secrete fructose, as the energy source for sperm.
3. Secrete citric acid as buffer to semen.
4. Secrete potassium and sodium ions to control the equilibrium of the osmotic pressure in semen.
5. Secrete flavin pigment which gives yellow coloration of cow bulls' semen in some breeds.

1.8.2. Prostate gland: see fig. (20).

It consists of:

Pars Externa (body, palpated rectally).

Pars Interna (Disseminate, encircled completely with the urethral muscle)

It is the major part, extends along the pelvic urethra and opens with many ducts into the urethra.

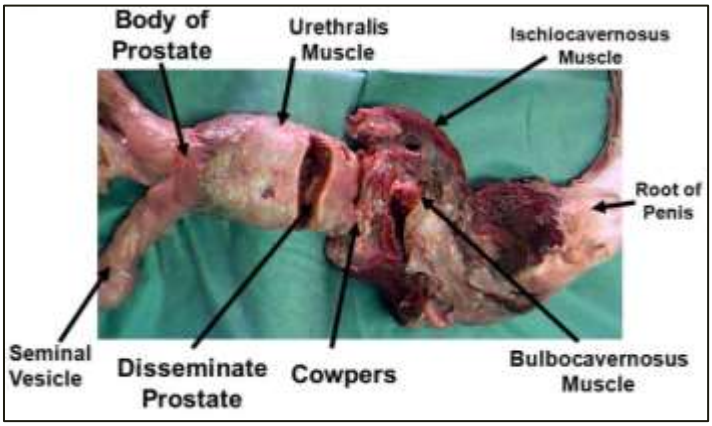


Fig. 20: showing the body and disseminate parts of the prostate gland, in bull.

Functions of the prostate gland:

- Secretes large amount of minerals that regulates the buffering system of seminal plasma .
- Its secretions are alkaline to neutralize the acidity of spermatozoa.
- Secretes amino acids for sperm nutrition.
- Participates as a vehicle media for sperm with the seminal glands.

1.8.3. Bulbourethral glands:

- The two Cowper's glands are located on the pelvic urethra close to ischial arch and covered completely with bulbocavernosus muscle
- They are oval in shape, and can't be palpated clinically; it is 2-3 cm length and 1-2 cm thick.
- Each gland has a single excretory duct at the posterior end of the pelvic urethra
- Its viscous secretions clean and neutralize the extra pelvic urethra before ejaculation.

One of the accessory glands may occasionally become infected, resulting in semen samples that are yellow and cloudy and which contain puss cells. It is not uncommon in bulls for the seminal vesicles to be so affected (*seminal vesiculitis*). Antibiotic treatment is sometimes necessary, but time will generally correct the problem.

2. Regulation of male hormones: see fig. (21).

The normal functions of male reproduction are largely controlled by hormones that are secreted from the endocrine glands. The testicle functions as an endocrine gland because of its production of the male hormone, **testosterone,** by the interstitial cells. Testosterone has several major functions:

- It is largely responsible for development and maintenance of the male reproductive tract
- It causes the development and maintenance of the secondary sex characteristics associated with masculinity, such as the crest and heavily muscled shoulders of a bull
- It is a major factor in the normal sex drive and behavior of the male
- It increases muscular and skeletal growth
- It is essential for normal sperm formation.

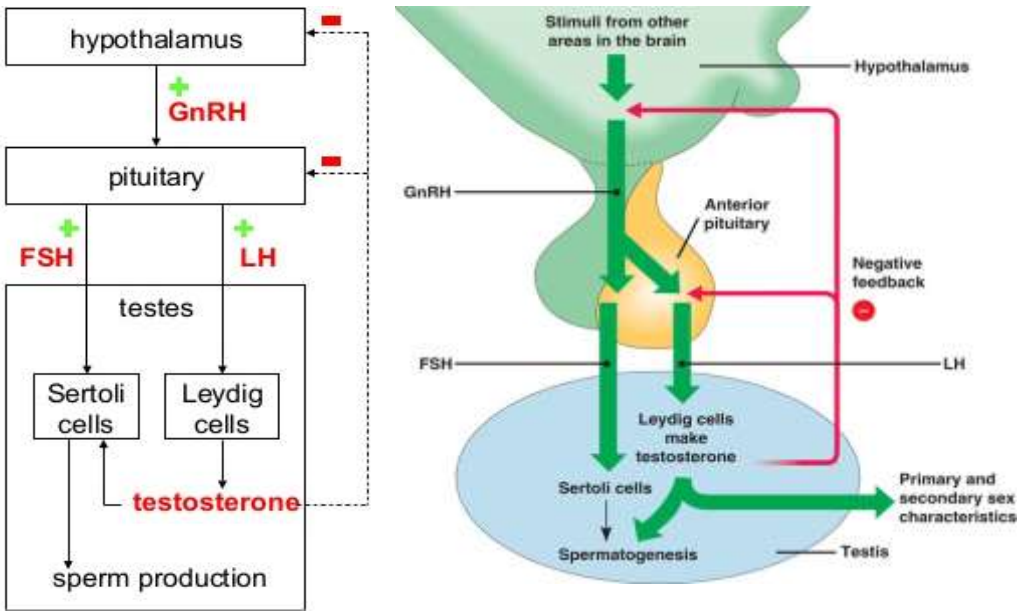


Fig. 21: showing the hormonal regulation of reproduction in male animals.

The testicle is, in turn, under the influence of hormones produced by other glands in the body. The same gonadotropic hormones that regulate ovarian functions in the cow also regulate testicular functions in the bull. Luteinizing hormone (LH) and follicle stimulating hormone (FSH) appear to be misnamed as pertaining to male reproduction, yet carry out several important male functions.

Luteinizing hormone and FSH are released from the pituitary gland and cause the testicle to secrete testosterone, which then acts on the germ cell lining of the seminiferous tubules to stimulate formation of primordial sperm cells. The maturation of spermatids into fully developed sperm cells requires the presence of FSH. Normal functioning of the male accessory glands requires testosterone.

Not only the hormone production of the testicle is regulated by hormones released by the anterior pituitary, but the reverse is also true. The level of testosterone in the blood regulates the secretion of gonadotropic hormones from the anterior pituitary via a feedback system. A proper balance of all hormones is vital to successful reproductive functions.

3. Sexual behaviour in male animals: see fig. (22).

- **Pre-copulatory "preparatory or courtship"**

The pre-copulatory behavior includes:

- Vision, Sniffing, smelling and licking "the female may urinate at the same time"
- Flehmen posture. see fig. (23), boar show no flehmen
- Chinning "chin resting"
- Sexual arousal. In which the female animal show receptivity by lordosis.
- Erection and penile protrusion

- **Copulatory stage**

It lasts for 1-3 seconds in bull, 20-60 seconds in stallion, and 3-20 minutes in camel-bull.

- Mounting behaviour. The male animal firmly holds the hind quarters of the female by its forelimbs.
- Intromission. Started by seeking the female vulva by the glans penis and entrance of penis.
- Ejaculation. The ejaculation type depends on the anatomical type of the penis either thrust ejaculation in bull or frictional ejaculation in stallion.

Special movements and position that end by semen deposition in female genital tract

- **Post-copulatory stage**

"Period of refraction"

- Dismounting
- Vocal emission

Fig. 22: showing the sexual behaviour in cow-bull including sniffing, flehmen, chinrest, and mounting.

Measurement of sexual desire:

- **Reaction time:**

It is the time between the introductions of male to female until the first copulation.

Normally it takes about 30 seconds.

- **Number of mounts:**

It can be calculated as the number of mounts during certain unit of time limited to 10-40 minutes.

It depends on the type of ejaculation, either Thrust ejaculation or Frictional ejaculation.

It includes special movements and position that end by semen deposition in female genital tract.

- **Number of copulations:** Until sexual exhaustion.
- **Recovery "refractory" time:** Time required until the male regain sexual interest.

Degrees of sex drive in male:

(0) No interest ---------- Abnormal
(1) Little interest ---------- Abnormal
(2) Some interest with hesitation ---------- Questionable
(3) Good interest with comparatively quick mounting without good holding and seeking ---------- Good
(4) Good interest with quick mounting ---------- Good
(5) Eager with very good holding ---------- Very good
(6) Uncontrolled "extremely eager"---------- Abnormal

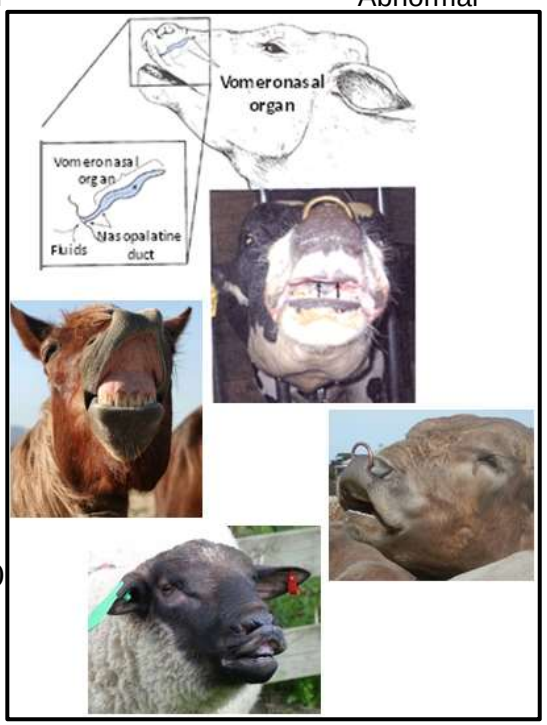


Fig. 23: the diagram showing the Vomeronasal organ with the nasopalatine duct. And the other images are showing flehmen posture in cow-bull, buffalo-bull, stallion, and ram. They look like laughing animals.

Mechanism of erection:

- Sexual excitement through sight, smell, touch, hearing, and licking.
- Stimulation of nervi eregentis (Vasodilatation to blood spaces or cavernous tissue).
- Contraction of erector muscles at root of penis (the two ischiocavernosus and one bulbocavernosus muscles).
- Relaxation of retractor penis muscle with relaxation of the sigmoid flexure and protrusion of penis.
- Continuous inflow of blood with decreased out flow lead to full erection and hardness.
- Erection and copulation controlled with parasympathetic nerve fibers
- About 2-3 minutes are required for erection and copulation in bulls

Mechanism of Ejaculation:

- When the glans penis get s in contact with the vulva and vestibulum ,during copulation, a reflex ejaculatory thrust occurs with semen deposits in the dorsal fornix of the anterior vagina
- Impulses arise at the sensory nerves of galea glandis transmitted by the internal pudic nerve to the bulbo-sacral plexus of the spinal cord at the lumbo-sacral section

4. Forms of infertility in the male:

These may be divided according to **Lagarlof** into three forms:

1) Diminished to lack sexual desire (sex drive or libido) known as **Impotentia erregendi.**
2) Inability to copulate (**Impotentia quandi or coeundi**).
3) Reduced ability to fertile (**Impotentia generandi**).

These conditions are present in all species with various degrees among species, breeds and individuals. These varied from mild to severe, and demonstrate a wide range of reproductive ability. In male animals, semen collection is usually possible so that direct measurement of sperm quality and other tests may be applied. Also for examination of males for infertility or sterility accurate breeding and health records of the male and herd are essential and also mating behavior of the bull will be of good benefits.

4.1. Reduced or lack of sex desire (Impotentia erregendii):

- **Causes**:

It may be due to either hereditary or environmental origin.

A- Hereditary (congenital - genetic)

It is largely genetic in nature, it is subjected to great modification by many environmental or physical factors, i.e. males of strong sex drive require more severe and prolonged environmental and/or physical factors to significantly affect their mating behavior than males with weak sex drive.

B· Environmental:

1) <u>Nutritional:</u>

Both under and overfeeding will result in lack of sexual desire.

<u>a- Under feeding:</u> Thin, emaciated, semi starved or those suffering from deficiencies of total digestive nutrient (TON), vitamin A, protein and/or certain minerals (phosphorus, iodine and cobalt), affect the gonads and subsequently reduce sexual drive.

<u>b- Over feeding:</u>

I- Males tend to become obese (fatty) and lazy, and suffer from joint and foot troubles.

II- Excessive roughage may cause great enlargement of the rumen and abdomen interfering with normal easy copulation.

<u>2) Systemic diseases:</u>

Any acute (pneumonia or enteritis) or chronic debilitating (tuberculosis)

diseases resulting in rapid or prolonged loss of weight, depression and weakness will cause varying degrees of loss of sex desire.

3) Age:

Young and old males' exhibit reduced to complete lack of sexual desire.

a- In young age, they are significantly affected by the nutritive level. High feeding levels hasten the onset of sexual maturity and sexual desire, whereas, low or subnormal feeding levels greatly retard the time at which the young males show sexual desire. They have also less experience and they do not know the task demanded from them and not yet well trained. The inexperience in young males may be confused with a lack of libido.

b- In old age, this may be due to decline in testosterone level, sterility, loss of condition, over use or arthritis.

4) Management:

Potency varies within animals (both young and old) depending upon the way they are trained, handled and managed.

- Young males kept apart from other stock are frequently frightened by the clothes presence of other animals, large number of people or color of the.
- If a male associates mount with pain or punishment he may refuse to mount.
- Heavy painful nose ring.
- Rough handling of the attendants.
- Improper restraint of the mount animal.
- Improper footing.
- Improper preparation of the male for mounting.
- Difference between the size of both male and mount animals.
- Use of artificial vagina that is too cold or too hot.
- Excessive use of the male (frequency of co11ection).

All the previous mentioned factors might reduce the sexual desire.

5) Lack of experience:

Sexual desire may be reduced particularly if the male is restricted in a poorly lighted, damp and cold stable.

6) Psychic factors:

It may interfere with normal coitus. Usually, there is no obvious clinical reason for this failure. If good history is available, it often reveals traumatic painful experience at the last attempt of copulation. This includes slipping, kicking or beating by the teaser and penile injuries especially in males with high degrees of sexual desire. Those injuries are remembered by the male and remain as a cause of lack of sex drive.

7) Hormonal:

Reduced to complete lack of sex drive -may occur in some cases of hypothyroidism, hypogonadism and hypopituitarism. This might show no clinical symptoms other than reduced sexual desire.

8) Other factors, such as structural lesions of the limbs and diseases of the penis and sheath may cause a reduction in libido in some males, especially if accompanied by pain. But _mostly these interfere with or prevent normal copulation, that is to say impotantia quandi (coeundi).

- **Symptoms:**

Symptoms vary from complete lack of any interest in the female, even in estrus, to a slight slowness or delay in copulation when compared with a vigorous active male.

Complete lack of sexual desire is uncommon, while moderate to slight degrees of reduced sexual desire are frequently observed. With proper teasing and handling many of these slow breeders will complete coitus.

Numerical grade	Descriptive grade
0	No interest or lack of sexual desire.
1	Little interest in mounting, sniffing at the back of the cow and lead to mounting attempts, which may sporadically make service.
2	Mounting the cow after obvious and repeated hesitation, feeble holding of the cow and feeble seeking.
3	Obvious Comparatively quick mounting without showing satisfactory holding and seeking
4	Quick mounting bull attention concentrated only on the cow, holding and seeking well.
5	Eager mounting, holding and seeking very well.
6	Extremely hard and Uncontrolled extremely eager mounting, holding and seeking intensive.

- **Prognosis:**

It depends upon the cause and degree of impotency. In case of genetic cause there is little that can be done. The environmental factors causing or promoting the lack of libido can often be overcome or even moderated. Early and proper diagnosis and treatment of all diseases of male animals is indicated to prevent

loss of reproductive ability.

- **Treatment:**

For the treatment of this condition, the breeding history of the animal must be known exactly, beside investigation of the animal for its nutritional state, sexual behavior under deferent environmental conditions must be undertaken to reach a proper diagnosis and direct the treatment for this cause.

1- *In hereditary* nothing could be done and the male should be discarded from breeding. This condition will be reflected not only on the male off-springs but also on the female ones.

2- *In environmental conditions*, the cause must be searched off and corrected:

1- Correct the feeding in under or over feeding. In fatty animals, the ration should be reduced accompanied by exercise. In under fed animals, the ration should be of good quality containing sufficient amount of vitamin A, protein and minerals. If the male is thin and emaciated it should be determined whether this is due to a lack of nutrients or to disease. Treat the animal against internal parasite if it is the cause.
2- Proper housing of the bull in a well ventilated box. The animal should have exercise and sunlight especially on pasture. Ring exercise (mechanical exercise) is sometimes used in AI Centers.
3- If bulls are overused, sexual rest is given for a period of about two weeks or more followed by a proper regular collection.
4- Young bulls must be handled carefully and trained for the purpose of use. Males needed for AI centers must be trained on a male teaser or a non-estrous female, while those used for detection of females in estrus must be trained on an estrous female. Both young and old bulls must be used on a lower frequency than middle aged one.
5- Any systemic disease or pathological cause of reduced sex drive must be treated.
6- *In reduced sexual desire due to the management*, it is advice to:

a- Rough and maltreatment of males should be avoided, before, during and after the act of coitus.
b- Changing of the stimulus or mount animal (teaser).
c- Changing of the breeding site.
d- Presence of other male near the mount animal or in sight of the breeding site.
e- Provide further stimuli.
f- Mounting the male to several different sites for teasing.

7- *In reduced sexual desire due to psychic factors* it is reported to:

a- Give a prolonged period of sexual rest (two months)
b- Change the site of copulation, teaser and/or attendant
c- Prepare the male carefully before breeding
d- Handle the male properly and avoid rough handling
e- Control the vigorous males

8- *In reduced sexual desire due to hormonal deficiency*, treatment should only be started after careful study of the male's breeding history, physical examination and after careful repeated observations of the male during coitus.

a- Testosterone is administered intra muscular in oily suspension with a dose of 100 - 500 mg for bull and stall ion, 50 - 100 mg for boar and ram and 10 - 50 mg for dog. The dose is repeated every 5 to 10 days for several injections. Avoid high level of androgen, because of the possibility of producing testicular degeneration and atrophy caused by the suppression of gonadotropic hormones.

b- Chorionic gonadotrophin is administered in a dose of 5000 - 10000 i.u for bull and stallion and 100 - 500 i.u for boar, ram and dog. Four injections are applied with an interval of 4 - 7 days for a period of 2 - 3 weeks (2 - 3 doses). This helps in stimulating testosterone production by the Leydig cells.

c- In hypothyroidism, as seen in some obese and lazy bulls, feeding iodinated casein with 3% thyroxin at a rate of 1 gm / l00 Ib/day is beneficial. This helps in increase the metabolic rate, hastens the loss of weight and occasionally improves the libido. Thyroid tablets 50 - 100 mg/day are also beneficial.

4.2. Impotentia quandi (coeundi):

The male usually has a normal or only slightly reduced sexual desire (libido), but unable to copulate due to severe physical or physiological reasons. The degree of inability varies and all phases of mating action may be affected. Thus the impotency may be manifested in one of the following forms:

1- Inability to mount. 2- Incomplete erection.

3- Incomplete penile protrusion. 4- Failure to thrust or ejaculation.

- **Causes:**

Bulls with high level of sexual desire usually try to overcome the causative agent, while those with low level of libido are more liable to be affected. The main causes are:

A- Affections (injuries) of the locomotor system:

Joints, muscles, nerves, bones, tendons, ligaments or blood vessels injuries, inflammation or lesions may cause inability to copulate due to pain.

1- Inflammation, lesions or dislocation of the hip or coxofemoral joints may cause inability to copulate or may cause sufficient pain so that copulation is not attempted. These lesions are not common in the bulls but more common in dogs and boars.

2-Intlammation of the ilio-sacral joint is relatively a common cause particularly in young bulls. It may results from very strong fodder and rapid growth, which produces a relatively weak skeleton. The joint under such condition could not stand the strain to which it is subjected when the young bulls are left free and ride one another or when put in hard service. The lesions affecting the iliosacral joint are characterized by peri-articular ossification and complete synostosis.

3- Inflammation of the stifle joint (gonitis) is the commonest of the joint injuries that prevent copulation in bulls. It is usually results when a large male mounts a cow on a slippery surface and falling at that time. The condition develops when small young males are bred to large females and fall down.

5- Tuberculosis in the vertebrae or sternum may cause inability to copulate. The animal shows good libido and erection but pain is manifested at the time of mounting or resting the sternum over the teaser back. In the early stages few ejaculates may be obtained, but with the progress of the disease the animal is unable to complete the service and may loose its sex desire.

6- Injuries of the extremities may be also a cause inability to copulate.

a- Foot diseases, as in foot and mouth disease, may be painful enough to prevent copulation.

b- Long overgrowth claws or hoof that cause extensive strain on the tendons of the hind limb during service.

c- Fracture or injury in the bone of the limbs, lumber vertebrae or the pelvis.

d- Tendonitis affecting the rear limbs may occasionally cause inability to copulate.

e- Nerve affection especially spinal pathology usually results in paresis or paraplasia.

7- Muscle affections may be also a cause inability to copulate.

a- Rupture or strain of the gastrochnimius muscle.

b- Spasms in the muscles of the hind limb and back or rheumatic myositis usually result in difficult service or even prevent mounting.

- **General symptoms:**

1- Lameness.
2- The animal could not bear weight on the hind limbs and dismounts with signs of pain.
3- In case of affections of sternum or vertebral column, pain is manifested at the time of mounting or resting the sternum over the teaser's back.
4- In early stages and mild cases few ejaculates may be obtained, but with the progress of the affection the animal is unable to complete service.
5- Sexual desire is gradually lost.

- **Prognosis:**

It depends largely upon the condition, species, age and value of the animal for breeding as well as upon nature of the lesions and injury.

- **Treatment:**

1- Sexual rest.
2- Restriction of activity.
3- Good keeping and proper feeding.
4- Proper treatment according the case.
5- In valuable animals semen could be collected by massage of the ampulla or electro-ejaculator.

B- Inner diseases:

1- Painful lesions of the peritoneum, such as necrobacillus of the liver,

traumatic gastritis or pericarditis are recorded as a cause of impotentia quandi. Such bulls should not be used during the acute stage and as long as they feel or manifest pain during service.

2- Osteomalacia, here the abdomen is enlarged, the animal is heavy and could not raise his weight to mount or reach the cow.

3- Hernia, both umbilical and ventral hernia (if large enough) may hinder the act of coitus. The condition may be hereditary, and so such bull should not be used in breeding.

4- Urinary calculi, this is usually accompanied by refusal to serve or show pain at the time of mounting.

5- Abscess in the liver or kidney, here pain is exerted at the time of coitus and leads to incompletion of service. The animal usually groans at the time of service.

6- Infection and painful inflammation of the testis, ampulla or seminal vesicles are sometimes associated with refusal to copulate.

- **Prognosis:**

In all these cases the prognosis depends on the severity of the case. The animal should not be put into service before complete cure.

C- Affections of the penis and prepuce:

These affections are the common causes for inability to copulate. These include inability to protrude the penis, inflammation of the penis and prepuce (balanopothitis), persistent frenulum, inability of the penis to coming out from the prepuce (phimosis), inability of the penis to withdraw in the prepuce (paraphimosis), rupture of the penis, deviation of the penis, tumors of the penis and prepuce or prolepses of the prepuce.

4.2.1. Inability to protrude the penis:

It may be due to congenital or psychic causes and observed especially in young bulls. This condition may not noticed until the bull reaches adult size or it may be occurred later in life after the bull have mated normally.

- **Symptoms:**

Varying from complete inability to protrude the penis out of the sheath, to protrusion of only 4 - 6 cm, which is not far enough to breed a cow. In these bulls, sexual desire is good or slightly reduced and there is usually no obvious physical defect or adhesion with the prepuce.

- **Causes**:

1- Congenital underdevelopment of the sigmoid flexure and/or the length of the penis causing them to be shorter than normal.
2- Insufficient relaxation of the retractor penis muscle preventing the extension of the penis and preventing the straightening of the segmoid flexure.
3- In other bulls the condition may be due to psychic impotency. This is usually met with in young bulls when the bull had been injured previously.

- **Diagnosis:**

Diagnosis is based on pudendal nerve block' or injection of a tranquilizer in a dose of 1.5 to 2 mg/kg to produce relaxation of the retractor muscle and the penis is withdrawn out of the prepuce.

- **Treatment:**

1- In hereditary causes cannot be treated and the bull must be discarded from breeding.
2- In psychic condition, where the bull has previously bred normally, sexual rest for about two months must be advised and the environmental conditions must be changed. Most bulls overcome this temporarily psychic inability and refusal to protrude his penis by proper teasing and good handling.
3- Sectioning of the retractor penis muscle has been tried, but it is contraindicated as it may results in paraphimosis.

4.2.2. Persistent frenulum: see fig. (24).

Persistent penile frenulum is the presence of a persistent band of fibrous tissue from the median raphi of the glans penis to the prepuce. This band causes the penis to be directed backwards in a semicircular manner during the act of coitus. The anomaly hinders natural service or semen collection.

- **Treatment:**

The case is surgically corrected by cutting through the persistent frenulum after anesthesia of the penis. If there is blood vessels transverse the frenulum they must be ligatured by catgut no. 2. And Oily antibiotic is applied after operation.

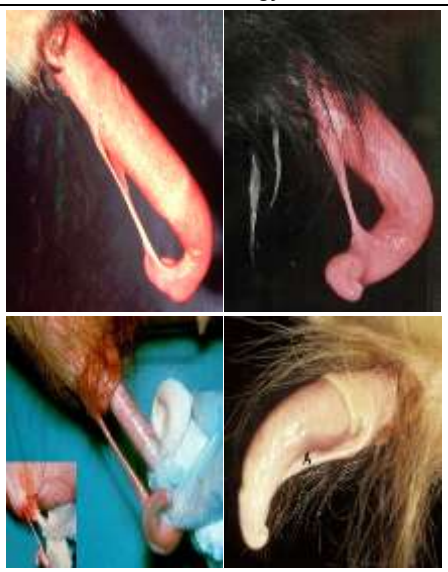


Fig. 24: these images showing different degrees of persistent penile frenulum in cow-bulls; this affection leads to phimosis.

4.2.3. Inflammation, injuries of the penis and prepuce (balanopothitis):

Balanitis is the inflammation of the glans penis. Posthitis is the inflammation of the prepuce. In a case of acute balanopothitis, the pain and irritation may be severe enough to prevent copulation. In chronic form adhesion may develop between the penis and prepuce resulting in phimosis.

- **Causes:**

1- It may be due to infection with bacterial or viral organisms. In the preputeal cavity of the bull, one may found a wide variety of organisms, e.g., trichomoniasis, vibriosis, coliform, molds, actinomyces, etc. Lesions of granular venereal diseases are found in the prepuce and on the penis of bulls, which are usually mild and chronic, are usually seen in natural service.

2- Lymphoid accumulation on the penis and prepuce may be due to mechanical injury or chemical irritant during the preputial sheath wash, the pain and irritation may be severe enough to prevent copulation. These may result in swelling of the prepuce and pain and the bull refuses to copulate.

- **Treatment:** see fig (25), (26)

1- Douching of the sheath with 20 - 250 ml of mild warm aqueous or oily antiseptic as chloramines 0.5 % or acriflavine 0.1 %.
2- Sulfanilamide and antibiotics in oily preparation or water suspension.
3- Treatment may be repeated every 2 - 3 days intervals
4- Sexual rest and housing of the bull apart from cows.

- **Prognosis:**

It is unfavorable in case of adhesion.

Fig. 25: these images showing acute (A), (B), and chronic (C) balanopothitis in cow-bull

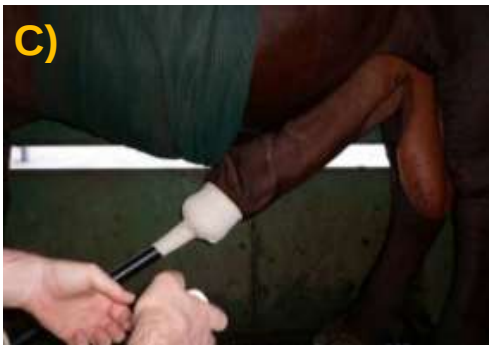


Fig. 26: these images showing the treatment of bull suffer from balanopothitis with douches of the preputeal sheath.

4.2.4. Ruptured, fractured or broken penis:

It is usually accompanied by a secondary haematoma. The condition is common in the active bull with strong sex drive, serving cow on posture. The injury occurs at coitus due to sudden bending of the erected penis against the stanchion or the hindquarters at the time the bull thrusts. In the stallion, it may occur when a mare kicks the erected penis during mounts.

- **Symptoms:** see fig. (27)

1- Shortening of the gait as well as stiffness in walking.
2- A swelling rapidly develops just cranial to the scrotum; its size depends upon the amount of hemorrhage from the ruptured penis.
3- The swelling is first soft and fluctuating, later it becomes firm and hard as the haematoma clots and organizes.
4- Pain is usually evident.
5- The bull may show prolepses of the preputial epithelium as a result of pressure.
6- The bull shows inability to copulate.
7- If treatment is not undertaken, the clot organizes and adhesions occur between the prepuce and penis and abdominal wall usually rendering the bull useless for future service.
8- Infection of the haematoma may develop an abscess, which may burst to the exterior.

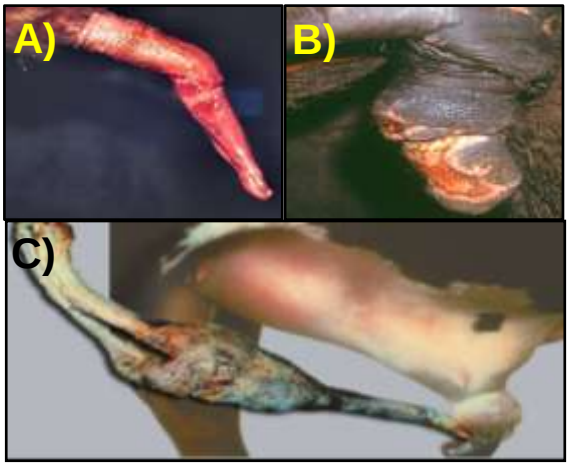


Fig. 27: these images showing the clinical picture of the broken/ruptured penis in bull (A), (C) or in stallion (B).

- **Differential diagnosis:**

It must be differentiated from abscesses, tumors, and rupture of the urethra.

- **Prognosis:**

About 50 % of the cases respond satisfactory to treatment even when an operation is performed. Haematoma that becomes infected seldom responds to treatment and surgery having poor prognosis.

- **Treatment:**

1- Removal of the clotted blood 3 - 8 days after the accident (after coagulation and before organization).
2- The operation is performed under general anesthesia and the bull restrained in lateral recompensing with the upper hind leg drawn backward to expose the pre-scrotal region.
3- After shaving and disinfecting of the operation site, an incision of about 15 - 25 cm long is made through the skin over the swelling and parallel to the penis.
4- The clotted blood in the haematoma is removed by gentile dissection.
5- The fibrin is removed and the break in the tunica is sutured by chromic catgut no. 2.
6- Oily suspension of antibiotic or ointment is applied to the area and the skin sutured with silk or nylon.
7- Sexual rest for 2 - 3 weeks although some workers advice slightly sexual stimulation to prevent adhesions.

4.2.5. Deviated or curved penis:

It usually occurs in the distal third of the preputial portion of the penis or glans penis and usually prevents natural service.

- **Causes:**

This condition may be hereditary in nature. It may also be a sequel to unilateral penile injury.

- **Symptoms:** see fig. (28).

1- In most cases, the penis is bent downwards, upwards or less commonly spiral or lateral deviation may occurs causing difficulty in extending the penis.
2- Semen is deposited away from its normal position (cranio-dorsal).
3- Examination of the penis could be done carefully by letting the bull to mount a teaser cow to determine the extension and severity of the condition.

- **Treatment:**

1- Surgical operation may be undertaken, yet, prognosis is usually unfavorable.
2- A double V shaped or elliptical incision at the convex side of the penis is made and the cut ends are sutured with catgut no. 2.
3- Oily suspension of antibiotic is applied.
3- Sexual rest, the animal is given sexual rest although mild sexual stimulation may be preferred to prevent adhesions.

Fig. 28: This image shows that the bull fail to copulate in spite of his very good sexual desire and ability to mount due to deviated/curved penis.

4.2.6. Tumors: see fig. (29).

Tumors of the glans penis and prepuce may be a cause for inability to copulate because of their large size or due to the pain accompanying service.

In bulls, fibropapilloma is the type of tumors found on the prepuce and penis. It is probably infectious in nature and the cause is considered to be a virus. They are similar in nature to those observed on the vulva of the heifers.

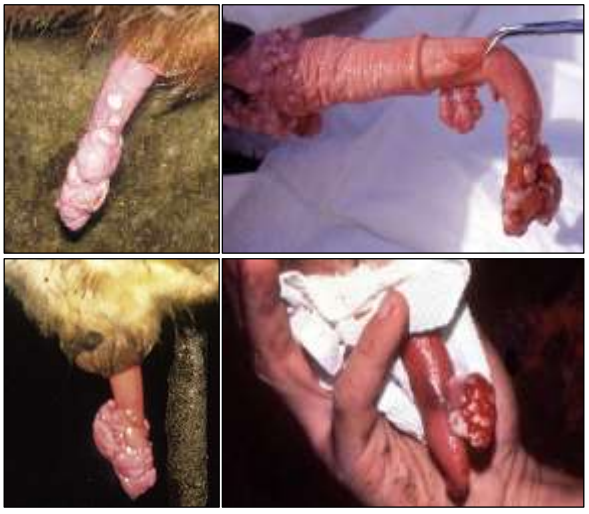


Fig. 29: These images show number of fibropapilloma of different shape and size in affected bulls.

- **Treatment:**

Fibropapilloma on the penis of a bull, if pendulant could be removed by grasping and pulling them off as the bull mounts the cow. Anesthesia of the penis is applied and the tumors are removed surgically by scissors or by a cautary and the mucous membrane is sutured by catgut. The prognosis is good, whenever treatment is used.

4.2.7. Phimosis:

It can be defined as stenosis of the preputeal orifice prevents the penis from coming out/protrude from the prepuce more than 10-15 cm. It is usually acquired and may be congenital. It usually occurs secondary to posthitis; prolapse of the prepuce or after amputation of the prolapsed prepuce. It may also results from large sized tumors or haematoma of the penis rendering it relatively larger in size, persistent frenulum, or underdeveloped sigmoid flexure.

4.2.8. Paraphimosis:

It is the inability to withdraw (retract) the protruded penis in the prepuce after protrusion through the preputeal orifice.

- **Causes:**

1- The condition is usually due to strangulation of the penis by a small preputeal opening. In rare cases, it may occur secondary to fibropapilloma of the penis.
2- It may be due to trauma and abscess formation in the preputeal opening.
3- Rabies results in paralysis of the penis in bulls.
4- Sectioning of the retractor penis muscle results in prolepses of the penis.
5- In stallion, generalized weakness, debility or neuromuscular paralysis may be the cause.

- **Treatment:**

1- Clean the penis and remove the necrotic tissue.
2- Apply antibiotic ointment, and with gauze, to protect the penis, try to reduce the penis inside the prepuce.
3- The antibiotic ointment should be packed into the sheath to prevent the adhesions after placing the penis into the sheath.
4- If the penis is much swollen, cold applications are used to reduce its volume.
5- In Stenosis of the preputeal opening, surgical interference is indicated.

- **Prognosis:**

It depends on promptness of treatment and the degree of necrosis present. A bull with a gangrenous glans penis never breeds due to loss of sensory nerve endings in glans penis.

4.3. Impotentia Generandi:

This condition in males is usually characterized by normal sexual desire and the good ability to copulates, but a complete or abnormally high percentage of failure of fertilization or conception in the females is occurred.

This incapacity or reduced capacity to fertilize may be accompanied by either apparently normal semen characteristics, or the semen is abnormal in morphology, concentration, motility or other qualities. When the semen is normal, it is difficult to explain the cause of infertility.

1- Impotentia generandi associated with apparently normal semen picture: Repeat breeders or failure of conception were reported in females served by bulls infected with brucellosis, vibriosis or trichomoniasis. Fai1.ure in fertilization "in repeat breeders" was in the past due to unknown factors. It is possible that some sterile bulls with apparently normal semen may have sperm cells with acrosomal defects which does not affect the concentration or motility of spermatozoa but usually impaired fertility and cannot be detected except by differential stains. This condition is usually inherited in nature and results in complete sterility, since the condition affects about 90 % of the spermatozoa.

It seems logical that other hereditary or congenital sperm cell defects may occur in the semen and be undetected by our present diagnostic tests. Higher magnification by the electron microscope or other types of stains may 'reveal more of these defects and causes failure of the spermatozoa to fertilize.
The condition may also occur if the chromosomes carry a congenital lethal factor, which causes early embryonic death.

2- Impotentia generandi associated with apparently abnormal semen picture:
It is usually met with in cases of testicular pathology or pathology of the accessory glands and duct system. The most of the cases of impotentia generandi are due to some pathological changes in the testes, epididymis, deferent ducts and the accessory genital glands.
1- Testicular pathology: The pathological changes of the testes, which give, rise to disturbances in the spermatogenesis as classified follows:
1- Testicular aplasia
2- Cryptorchidism.
3- Testicular hypoplasia.
4- Degenerative changes in the seminal epithelium.
5- Testicular fibrosis.
6- Inflammatory testicular changes.

4.3.1. Testicular aplasia:

Testicular aplasia is the complete absence of the testis. The condition may be unilateral or bilateral.

- **The Symptoms:**

The scrotal sac is small and empty. In bilateral aplasia the accessory glands and the secondary sexual characteristics are not developed, in spite of reaching the age of puberty.

- **Diagnosis:**

It is based on the clinical signs and rectal examination.

- **Treatment:**

The affected bull should be discard from breeding even if the condition is unilateral.

4.3.2. Cryptorchidism:

It is a congenital form of hypogonadism in which one or both testis fail to descend in the scrotum. The anomaly occurs more often in horses than cattle. The left testis is more affected than the right one.

The case may be complete (the testis retained in the abdominal cavity) or incomplete (the testis if found in the inguinal canal at the external inguinal ring).

- **Symptoms:** see fig. (30)

1. In the unilateral' complete cases, asymmetry of the scrotum is noticed.
2. In bilateral complete cases, the scrotum is small and shrunken. Sexual desire and behavior are normal and both the accessory glands and secondary sexual characteristics develop normally.
3. In unilateral cryptorchidism, the sperm cell concentration is reduced and the pH is alkaline, while in bilateral cryptorchidism azospermic semen (azospermia or aspermia) can be collected.

- **Treatment:**

The presence of hereditary factor makes the animal unfit for breeding.

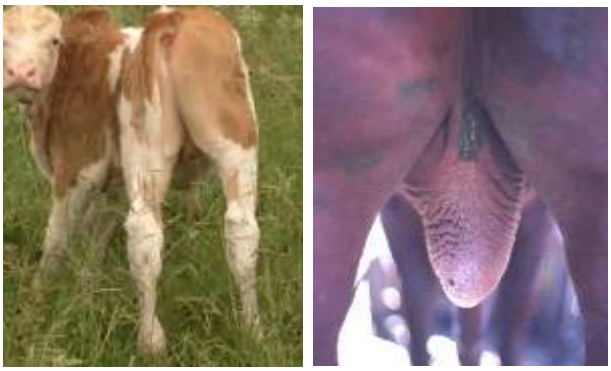


Fig. 30: These images show young cow-bull with cryptorchidism.

4.3.3. Testicular hypoplasia:

It is the underdevelopment or incomplete development of the testis. Incidence: It occurs in all species of domestic animals.

- **Causes:**

The condition is inherited in nature, caused by a recessive autosomal gene. The condition affects the development of the semineferous tubules leaving the interstitial cells intact. It may unilateral or bilateral.

- **Gross anatomy:**

Testicles are less than the normal weight and volume. Commonly, they are one-third to half the normal size. The epididymis is small unexpended and contain few spermatozoa. The seminal vesicles have been described as reduced in size but usually there is no alteration from the normal size or consistency.

- **Histopathology:**

According to the severity, it can be classified into 3 or 4 forms. The mildest form usually unnoticed and only 3 forms of hypo plastic changes can be diagnosed by the histological changes, between each of which there is a gradual transition.

1- In the severe form, all the semineferous tubules are affected and formed of one cell layer of inactive cells lying on the basement membrane with abundant interstitial tissue between them.
2- In the less severe (middle) form, about Ii - 2/3 of the tubules are affected. The remaining showed varying degree of activity with formation of spermatozoa.
3- In the mild form, only few tubules consist of a single layer of cells, but in the majority, spermatogenesis precedes though less activity than normal.

- **Symptoms:**

The testicular hypoplasia occurs only as an established condition at and after puberty. There is marked reduced fertility from puberty onwards. Affected animals are either sterile or have an extremely low conception rate about 5 %. The scrotum is reduced in size; the testicles are smaller than normal. The degree of reduction in size depends upon the severity of the condition. Consistency is normal, more soft or firmer on palpation. They can be moved freely within the tunica vaginal is. The epididymis is small and unexpended and its tail is small and hard. Sexual desire and Service behavior are almost normal although some authors stated that they might be increased. The condition can occurs where general physical condition and health is normal.

- **Seminal changes:**

The volume of the semen may not be affected. The semen is relatively clear and more fluid than normal. Concentration of semen depends on the degree of hypoplasia; however, concentration is always subnormal in mild form. In severe bilateral cases, no spermatozoa are detectable in the semen (azospermia). The concentration varies from zero to 200 X 106 per ml. Motility and metabolic activities are always subnormal. Giant cells are met with and the majority of the sperms are abnormal.

- **Diagnosis:**

The condition is diagnosed by the finding of low fertility in an animal at puberty together with the reduction in size of the testicles. It is confirmed by examination of the semen.

- **Differential diagnosis:**

It must be differentiated from chronic orchitis by the absence of adhesion and from degeneration by the breeding history, age and physical examination of the genitalia.

- **Treatment:**

No successful method of treatment is known, as far as the condition in congenital. The animal must be discarded from breeding.

4.3.4. Testicular degeneration:

- **Incidence:**

Degenerative processes in the testicles are the most common cause of reduced fertility in male animals especially in bulls and rams.

- **Etiology:**

It is not the result of specific disease of the testicles, but is the result of other side effect of more generalized disease as: Thermal degeneration usually results from increased temperature of the testicle as a consequence of cryptorchidism, shortening of the external cremaster muscles, excess of scrotal fat, inguinal or scrotal hernia, edema of the scrotum and inflammatory changes involving the tunica vaginalis. Degeneration may occur as a result of febrile diseases and toxemic conditions.

- **Gross anatomy:** see fig. (31)

Changes in the degenerated testicle are rarely visible to the naked eye. Slight reduction in size, where the condition has been long established. If fibrosis is extensive, the testicles are well below normal size and weight.

- **Histopathology:**

The histological changes vary with the degree and extent of the disease. In the

mildest form, there is necrosis of the spermatids and spermatozoa in some tubules but others appear normal. In advanced cases, extensive changes of this sort are found throughout the testicles and many tubules are completely destroyed, except for basement membrane. Giant cell formation occurs in some tubules. This stage is followed by increase of fibrous tissue, which occupies the spaces left by the degenerated tubules.

- **Symptoms:**

The first symptoms are reduction in fertility to a varying degree. In most cases of degeneration, no visible changes in the scrotum and testicles.

On palpation, the testicles move freely within the scrotum. In the bull there is usually little alteration in size. The consistency of the testicles is usually not noticeably changed but both hardening and softening of the tissues had been described. In advanced cases the testicles become smaller in size. The epididymis is easily palpable and in mild or early cases, it is of normal size and consistency. Later it tends to become smaller and harder.

Service desire and behavior are always normal and general health and physical conditions good.

- **Seminal changes:**

1. The characters of the semen vary widely with the stage and type of degeneration. Semen volume remains normal.
2. At the beginning the only alterations are usually morphological ones in the spermatozoa. In the bull, the proportion of morphologically abnormal spermatozoa of all types is greater than normal. The number of immature spermatozoa, those with protoplasmic droplets on the neck or at the end of the middle piece is also greater than the normal proportion of2 - 3 %. Giant cells are seen with other large cells, the volume of semen is usually within the normal range for the species.
3. The volume, in the early stage, is also normal. As the degenerative process advances, both the concentration and activity are reduced in parallel with the testicular changes.
4. When testicular degeneration is permanent, progressive and followed by fibrosis, the stage may be reached where no spermatozoa are, or these few present are dead. During the recovery phase, the number of active spermatozoa increased and tends to reach the normal level. .

- **Diagnosis:**

It is based on the reduction of fertility in an animal previously fertile and without well-marked changes in the testicle or other parts of the reproductive tract. Changes in the semen confirm the diagnosis.

- **Differential diagnosis:**

It is differentiated from testicular hypoplasia by the history of earlier fertility and the size and development of the tests from orchitis, by the absence of gross adhesions of the testis to the tunica vaginalis and marked atrophic changes in

the testicles.

- **Prognosis:**

It depends on the diagnosis of the primary cause of the degenerative process. In febrile conditions, the primary cause can be successfully treated and a favorable prognosis with regard to fertility given. Where the degenerative process is so far advanced as to have produce extreme changes in the semen, the prognosis is always less favorable where there is testicular fibrosis and atrophy recovery rarely occurs.

Fig. 31: This figure is showing the testis with testicular degeneration (right one) in relation to the normal one (left side).

- **Treatment:**

Testicular degeneration cannot be treated itself without treatment of the primary cause.

Any treatment should be applied to the primary cause, which is responsible for the degenerative process.

1. Endocrine treatment by means of gonadotrophins in various forms has been tried with no great success, as the condition is not due to hypogonadism.
2. When the primary disease diagnosed and successfully treated, the period of regeneration may be shortened; otherwise the animal can be kept only under optimum conditions of housing and nutrition, rested completely from service and reexamined after a period of three months.
3. It is advised to injection of glucose or calcium borogluconate to act against the toxins.

4.3.5. Testicular fibrosis:

Testicular fibrosis is the last result of the degenerative and inflammatory changes of the testicles.

In this case the semineferous tubules are destroyed and replaced by the interstitial connective tissue and the bull is completely sterile.

4.3.6. Orchitis:

- **Incidence:**

Inflammation of the testicles and the changes, which follow it, are amongst the commonest causes of reduction in spermatogenesis.

- **Etiology:**

1. Most cases of orchitis are due to bacterial infection. There is general agreement that Brucella abortus is the most common organism in the bull. Other organisms including Mycobacterium tuberculosis, Corynebacterium pyogenes, actinomyces bovis and streptococci have been incriminated. In porcine, orchitis, the main infecting organism is Br. Suis. In ram, ovine orchitis is due to Br. Melitensis, C. pyogenes, Cl. purifaciens, C. ovis and Pasteurella pseudotuberculosis.

2. In stallion, orchitis is mainly due to Salmonella abortus equi and influenza viruses. Infection may sometimes arise by extension from wounds penetrating the scrotal sac.

3. Traumatic orchitis is, however, rare and the majority of cases of infectious orchitis are probably haematogenous, particularly where Brucella abortus is concerned.

- **Pathology:**

In the acute stage the affected testicles are enlarged and hyperemic and there is a profuse serous exudation between the testicles and the tunica vaginalis. This inflammation is often followed by ischemic necrosis and sometimes by abscess formation or this may be followed by fibrosis and atrophy.

One or both glands may be affected. This may be followed by fibrosis and atrophy, or by chronic orchitis with abscess formation and fibrosis.

Both in chronic orchitis and post orchitic atrophy there are almost invariability in extensive adhesions between the testicles and the tunica vaginal is. The histological changes do not differ, in either the acute or chronic form those, which are typical of other inflammatory processes.

In chronic orchitis, the spermatogenic tubules are destroyed and replaced by fibrous tissue or by purulent foci.

- **Symptoms:** see fig. (32)

1. Acute orchitis is generally associated with high fever and other symptoms of

general health disturbances.
2. If both testicles are affected, the scrotum is swollen, hot and painful on palpation.
3. When the condition is unilateral, the scrotal swelling and pain is confined to the affected side.
4. The acute phase lasts for 7 - 14 days, after which the scrotal enlargement gradually subsides. Later, adhesions can be felt which completely fix the testicle within the scrotum.
5. As the lesions progress and fibrosis develops, the testicle become reduced in size, often irregular in shape and very hard in consistency. It may remain enlarged with fluctuating areas due to gross abscess formation.
6. There is an increased firmness in consistency in the non-affected unilateral testicle owing to the production of degenerative changes as the result of the increased scrotal temperature.
7. Sexual desire and behavior-are normal in affected bulls in the chronic stage.
8. During the acute phase, fertility is markedly subnormal. This is also in the chronic phase where both testicles are affected, but if only one gland is affected and the condition resolves and is followed by fibrosis, the neighboring testicle which usually degenerates following the acute attack, may regenerate. Therefore, after a period of low fertility, an improvement may occur, though not to the normal level.

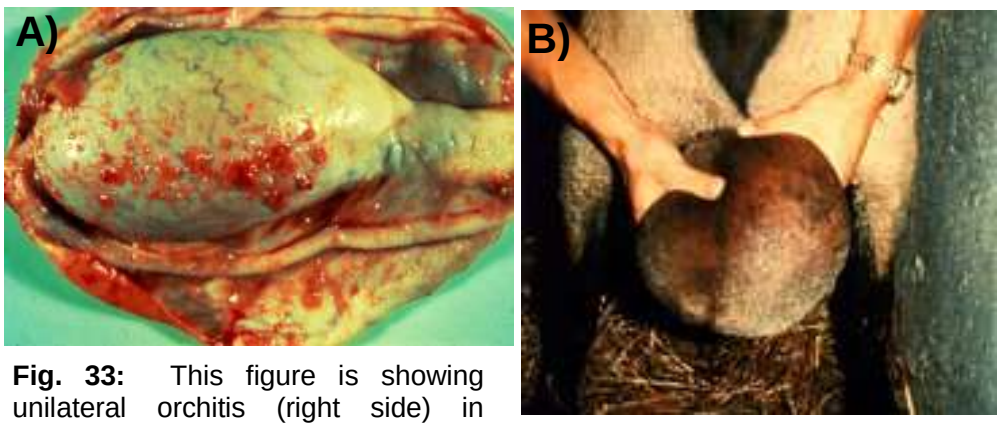


Fig. 33: This figure is showing unilateral orchitis (right side) in affected ram (B) and the gross lesion of the affected testis after its surgical removal (A).

- **Seminal changes:**

Changes in the semen take place within one or two days of an acute attack of orchitis and remain during the chronic phase.
The activity of spermatozoa is below normal. The number of spermatozoa becomes reduced and within a short time is very markedly subnormal, so that the semen is watery and clear.

A high proportion of morphologically abnormal spermatozoa are present. Giant cells, blood and pus cells are in the ejaculate. The volume of semen is normal.
When one testicle is only affected and the other has undergone degenerative changes because of high scrotal temperature, the semen remains abnormal if regeneration does not occur.
In some case spontaneous resolution of the degenerated testicular tissue takes place after a period of about three months. During this time the semen remains abnormal then gradually improves in all respects in parallel with the regeneration, but sperm concentration rarely reaches the normal level.

- **Diagnosis:**

It is based on the clinical findings in the testicle in both acute and chronic orchitis and on the alteration in the semen picture. Bacteriological examination of the blood, semen or from fluids taken by scrotal puncture in brucella orchitis is of diagnostic value.

- **Treatment:**

1. Where the causal organism can be isolated, treatment is directed against it. Antibiotics are of no value.
2. Brucella orchitis can be treated by using chloromycin and Aureomycin, otherwise only symptomatic treatment with complete rest can be used.
3. In acute unilateral orchitis, it has recommended that the affected testicle should be immediately removed surgically so that degenerative changes in the unaffected gland may be prevented.

4.3.7. Diseases of the epididymis:

4.3.7.1. Aplasia of the epididymis:

Complete absence of the epididymis is known as aplasia. Its cause had been suggested to be an inherited defect as a result of congenital absence of the walffian duct, from which the epididymis and vas deferens develop. Both ducts may be affected; right-sided aplasia is more common.
Complete absences of the body and tail as well as the vas deferens are often occurs.
The head of the epididymis may become swollen due to the accumulation of sperms from the testis, which have no egress to outside.
Degenerative atrophy of the testicle will then occur.
The absence of the tail of the epididymis is easily detected clinically. In cases of developmental defects of the epididymis a rectal exploration should be made, this may reveal an absence or abnormal shape or structure of the ampulla of the vas deferens or of the seminal vesicles.

In bilateral cases, aspermia is present. Animal's affected one side only showed normal semen and are fertile. Congenital or acquired obstruction of the epididymis may be followed by pyogenic infection with purulent semen.

- **Symptoms:** see fig. (33)

Unilateral absence of the epididymis and vas deferens does not produce sterility, although it lowers fertility because of the reduction in the number of spermatozoa through the functional absence of the testicle.
On examination of the scrotum no epididymis can be palpated, the testicle of the same side is normal as are the testicle and epididymis is of the other side.
Sperm numbers are below normal in varying degree depending on the level of spermatogenic activity of the testicle of the normal side.

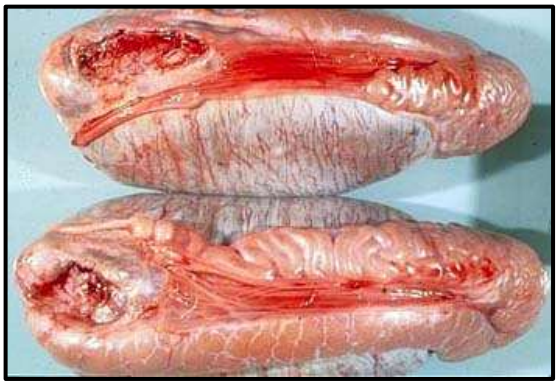


Fig. 33: This figure is showing segmental aplasia of the epididymis (upper one) in compare to the normal epididymis (lower).

- **Diagnosis:**

It depends on the clinical examination.

- **Prognosis:**

The fertility is below normal if normal service is employed. It is probably unwise to breed from affected bulls, because it is possible that the condition may be hereditary.

4.3.7.2. Epididymitis:

Inflammation of the epididymis occurs almost in association with orchitis.

The inflammatory lesions, which are caused, and the fibrosis and atrophy, which commonly follow, are not different from these in the testicle. The symptoms and treatment are those, which occur and used in orchitis.

4.3.7.3. Spermatocele:

In spermatocoele, an obstruction of the epididymis followed by enlargement and Spermatocele formation is common in rams and bucks.

- **Etiology:**

The condition follows epididymitis, but it can arise apart from inflammatory changes, and may appear at puberty.

- **Gross anatomy:** see fig. (34)

1. In rams the tail of the epididymis is most commonly affected although the head or the whole organ may be involved.
2. In advanced cases the affected portion is enlarged, nodular and indurate.
3. On cross section, cavities up to one cm in diameter containing thick suspensions of spermatozoa and cellular debris are found. The condition is unilateral or bilateral.
4. The testicle associated with an affected epididymis in advanced cases often shows white patches of fibrous tissue and in some cases small areas of calcification.

Fig. 34: Spermatocoele (right one)

- **Symptoms:**

1. Before charges in the epididymis are palpable, semen quality is said to deteriorate with increasing proportion of abnormal sperms.
2. In well-developed cases the lesions can be palpated easily on the epididymis as producing nodules. The whole of the affected part can be felt to be hardened and enlarged.
3. In advanced cases, with extensive bilateral lesions, the semen is usually a clear fluid containing only isolated, morphologically abnormal, feebly motile or dead spermatozoa together with other cellular debris.

- **Diagnosis:**

1. Well marked characteristic nodular lesions in the epididymis.
2. The seminal changes.
3. The absence of other abnormalities.

- **Differential diagnosis**:

Congenital cysts of the epididymis are occasionally found and may cause slight palpable protrusions. These cysts are not to be confused with Spermatocele because they do not give rise to any indurations.

- **Prognosis:**

This condition is Incurable.

- **Treatment:**

No effective treatment is known.

4.3.8. Diseases of the accessory glands:

4.3.8.1. Aplasia of the seminal vesicles:

The seminal vesicle of one side may be absent in the bull. This occurs usually in association with absence of the ductus deference of the same side and sometimes also complete or partial aplasia of the corresponding epididymis.

- **Diagnosis:**

It can be diagnosed by rectal examination, the absence of one seminal vesicle being easily detected.

4.3.8.2. Seminal vesiculitis:

- **Definition:**

It is inflammation of the seminal 'vesicle is relatively frequent in the bull and is probably always bacterial in origin, the most common pathogen Brucella abortus and after it Coryne pyogenes and streptococci. Tuberculous inflammation also occurs.

- **Gross anatomy:**

One or both vesicles are involved. The organs are enlarged and fibrosed. Abscess formation is common.

In some cases the inflammation may spread and involves the adjacent peritoneum and sometimes the rectum.

The mixture of the abscess formation may occur through the rectal wall and cause fistula formation.

In old established cases, extensive fibrosis and adhesions of the seminal vesicles together with the ampullae, ductus deferens and sometimes the adjacent peritoneum with the rectum.

- **Symptoms:** see fig. (35).

1. General health disturbance usually does not occur except for two days of acute infection with febrile symptoms.
2. The condition soon enters the chronic phase and is associated only with a reduction of fertility if the affected gland or glands still communicate with the

urethra as they usually do.

3. The changes in the seminal vesicle can be readily palpated per rectum. There is asymmetry; the size of the affected gland is bigger, irregular in shape and well-marked indurations. When adhesions are extensive, the whole areas are felt as hardened mass attached sometimes to the rectal wall. Fistula formation can be felt per rectum. Service behavior' remains' normal.

4. The concentration of spermatozoa is within the normal range, but motility us reduced probably from the adverse effect of normal accessory secretion and purulent floccules, isolated pus cells and cellular debris and sometimes blood may be found in semen.

5. A varying transient vaginitis and cervicitis is recorded as following a few days after service by bulls affected with streptococcal or Corynebacterial seminal vesiculitis.

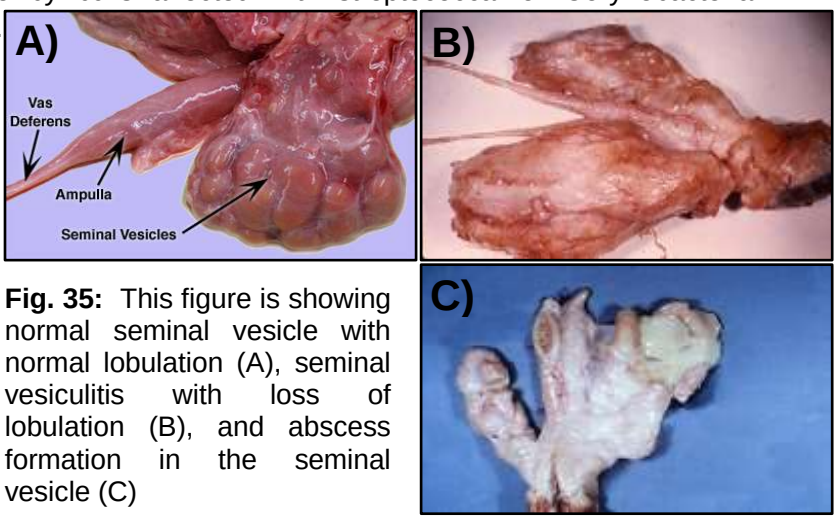


Fig. 35: This figure is showing normal seminal vesicle with normal lobulation (A), seminal vesiculitis with loss of lobulation (B), and abscess formation in the seminal vesicle (C)

- **Diagnosis:**

The condition is diagnosed by the reduction in fertility and the clinical examination. The causal organism may be isolated bacteriologically from semen samples.

- **Treatment:**

1. Using antibiotics in streptococcal seminal vesiculitis, penicillin treatment is of value, while in brucella infection Chloromycetin and Aureomycin could be used.
2. The affected animals should not be used in service, but isolated because of fear of infection being transmitted generally.

- **Prognosis:**

It varies with the extent of the lesions. If the disease is bilateral and the testicles and epididymis are involved, the chance of recovery is very little unless effective treatment is started early.

When there is unilateral infection of the seminal vesicles and the treatment is applied early, the affected glands may be wholly or partially destroyed, but the

animal still remain fertile.

4.3.8.3. Diseases of the prostate gland:

It is uncommon in the large animals. A retention cyst of the body of the prostate in the bull has been reported.

4.3.8.4. Diseases of the bulbo-urethral glands:

These glands are rarely the seats of disease. Abscesses may occur in pyaemic conditions.

The next table (table 1) shows the changes in the semen characters in relation to various affections of the reproductive organs of the bull, which can be valuable aid in diagnosis. (a; normal or not changed ,while x ; pathological or changed)

Semen characters	Orchitis	Testicular degeneration	Epididymitis	Dysfunction epididymis	Seminal vesiculitis
Motility	x	x	x	x	x
Sperm cell concentration	x	x	a/x	a/x	a
Primary abnormalities	x	x	a	a	a
Secondary abnormalities	x	x	x	x	a/x
Leucocytes	x	a	x	a	x
Germinal epithelial cells	x	x	a	a	a

1. a; normal or not changed,
2. x; pathological or changed.

5. Breeding soundness examination:

There are 5 general factors that can affect bull fertility:

- Structural soundness
- Capability of the reproductive organs
- Quality of semen
- Level of libido
- Plane of nutrition.

All are important and a deficiency in any category will negatively affect fertility.

5.1. Structural Soundness

General health and structural soundness are important aspects of fertility. Poor health can affect libido, mating ability, and semen production and quality. Structural soundness, including functional feet, legs and associated joints, is critical for the bull to effectively travel the breeding pasture and service females in heat. Any disease that impairs the mobility of the bull hinders reproductive performance.

A conformational problem commonly associated with poor breeding performance is extreme straightness of the rear leg (post-legged). A moderate angle in the hock joints is necessary for the bull to thrust properly after mounting. Proper angulation in the leg joints also helps to absorb the shocks produced during each step and increases the productive life-span of the bull.

The bull's fertility is the most important of his traits. You want him to be able to sire many calves, and sire them early each joining season. To do this, a bull must be sound in his structure so that he lasts many years, serving many cows in a short period of time without suffering injury.

Structural soundness is hence an integral part of this fertility. The bull's ability to remain fertile is dependent on his structural soundness.

Elements of a bull's conformation are heritable. If a bull shows straight legs and badly grown claws, for example, any sons or daughters kept in the herd may also have these problems.

When structural soundness in a bull is mentioned, most producers think in terms of leg and shoulder structure and the wear and tear on feet and joints. This is very important, but structural soundness includes all visual aspects of the structure of the beast.

To understand all aspects of the bull's structure and be able to compare one animal to the next, it is wise to adhere to a routine examination. One pattern to use may be this:

Start with head, neck, brisket, shoulders and front legs and feet. Next, view along the underside to the sheath, then, testicles and the back legs and feet. Follow your

way up to the pin bones and hips, then the topline and back to the shoulders and neck.

The bull should be viewed from the side, from the front, and from behind. He should be allowed to walk out, and again be viewed from the side, from the front and from behind to confirm any suspicions of poor leg structure.
Let's look at each of these areas individually.

The head
The head should show reasonable length and width yet not too large in proportion to the body. A head that is too big could potentially increase calving problems.

The eyes
Some breeds are very susceptible to eye cancer. Eye cancer is a serious condition leading to wastage in cattle and possible condemnation of the carcass. Susceptibility to it is a heritable trait, and while several factors contribute to its onset, exposure of the eye to sunlight plays a major part.

The eyes should be well set into the head to reduce this exposure. There should be a strong forehead over the eye, providing protection from sunlight (hooding). Eyes that bulge out from the head should be avoided. Pigment around the eye will also assist in reducing eyelid cancer in the white-faced breeds.

The muzzle
The muzzle should be wide for efficient grazing. The teeth on the lower jaw should meet squarely with the upper pad. Bulls with overshot jaws (lower jaw protruding) and undershot jaws may have difficulty grazing on pasture, especially when conditions get tough.

The neck
The neck should be of good length and held high. A bull which holds his head and neck low may in fact be straight in the shoulder. This affects the bull's gait and mobility. A straight-shouldered bull is also likely to be straight in the hind legs, a very serious fault, leading to early breakdown.

The brisket
The brisket is one area in which fat will be deposited. The bull should be trim in the brisket, as he should be throughout his body. Overfat bulls may in fact be light in their muscle, producing progeny with lower yielding carcass.
Bulls that appear fat at sale may have been overfed in their preparation. Overfeeding, especially on high grain rations, may affect the fitness and longevity of the bull, as he carries more weight than his structure is designed for. Overfat

bulls may also show temporary reduced fertility, if fat is laid down in the neck of the scrotum.

The shoulders see fig. (36).
The shoulders and front leg structure of the bull are shown in Figure 1 below. The shoulders are naturally sloping. A slope of 45–60 degrees is considered acceptable. A beast whose shoulder blade is tipped forward (straight-shouldered) has less angle at the shoulder joint and elbow joint and this reduces the shock-absorbing ability of these front joints.

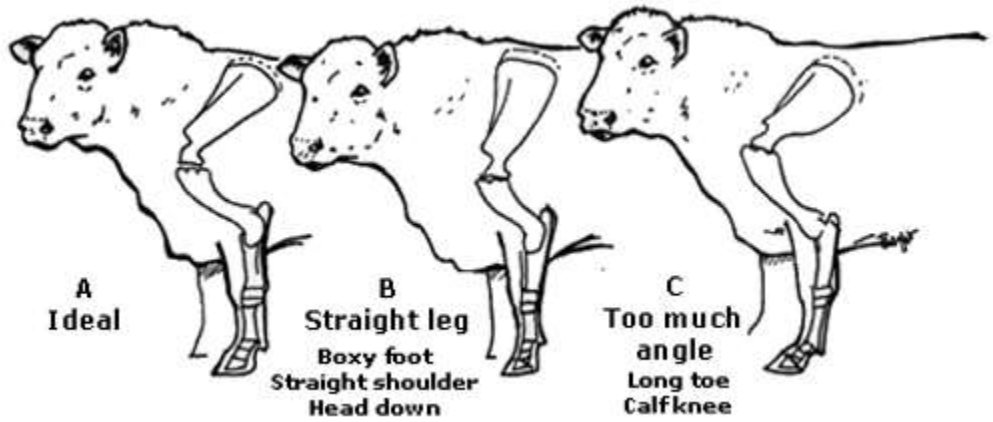


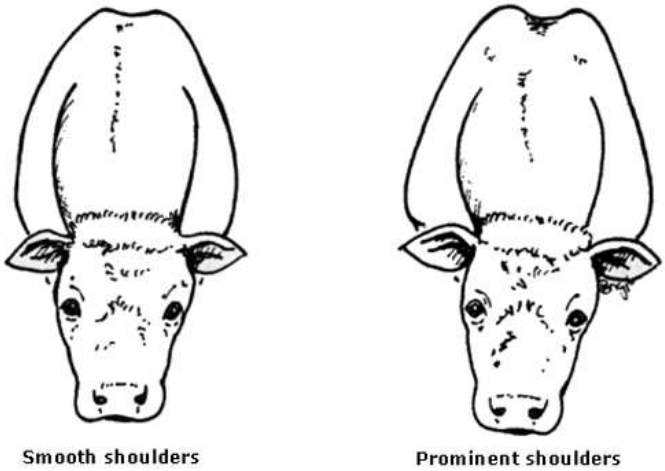
Fig. 36: This figure is showing structural soundness of the shoulder in breeding bull. The ideal shoulder with ideal elevation of head and neck (A), Straight shoulder with the head down (B), and too much abnormal angle of the shoulder (C)

The straight-shouldered bull tends to walk with a short choppy gait. He will carry his head low and may have difficulty raising his head much above his backline. Quite often the tip of the shoulder blade is prominent above his backline.
Usually, a bull that is straight in the shoulder will also be straight in the hind leg.

These bulls are particularly prone to early breakdown through the wearing of the leg joints, and the onset of arthritis. While many straight-shouldered bulls will break down in the hind leg, they are also more susceptible to arthritis in the pasterns and knees of the front leg. Straight-shouldered bulls may also be straight in the pasterns, causing rapid wearing of the front of the hooves.

The shoulder should be smooth against the rib cage. Bulls whose shoulders are wide at the point of the shoulder (the base of the neck) or wide between the shoulder blades (when observed from above) may throw heavily shouldered calves, increasing the chance of calving problems. See Fig. (37).

Fig. 37: This figure is showing anatomical difference between the smooth shoulder and the prominent shoulder in bull (front view).



Bulls with straight shoulders may also affect the ease of calving. Any deviation away from the normal angles of the calf may produce an abnormal calf shape, causing calving difficulty.

It should be remembered that many things affect calving difficulty, and that calf size (weight) in relation to dam pelvic size will have the greatest effect on ease of calving.

Front legs and feet

The front legs of the bull should be straight when viewed from in front. On a structurally sound animal, a vertical line may be drawn from the point of the shoulder to the middle of the claw. This line should intersect the knee (see Fig. 38).

As the knee joints carry more than half the bull's body weight, deviations from this line may cause excessive wearing of these joints.

A 'knock-kneed' bull may have turned-out front feet (up to 10 degrees is considered normal). A bull is considered 'knock-kneed' when the knee joints lie inside this line, which may eventually lead to overgrown outside claws.

A bull that is wide at the knees (bow-legged) presents a more serious problem. These animals are often narrow in their stance and may roll their feet as they walk. They can also be wide in their shoulders.

Fig. 38: This figure is showing the vertical line from the shoulder to the middle of the claw. Normal bull front view (Left), Knocked-kneed abnormal bull (middle), and bowed-legged abnormal bull (Right).

joint forward of this line (buck-kneed) can be associated with steep shoulders and pasterns, and may be a serious fault.

A knee joint set back of this line (calf-kneed) may be associated with sloping shoulders and has little impact on function.

The way the claws of the feet grow often indicates structural problems higher up the legs. Long or excessively short even claws may indicate too much or not enough pastern angle, causing both claws of the hoof to grow or wear excessively. Overgrown claws affect the mobility and performance of the animal. Fig. (4) Indicates the correct angle of the pastern joint.

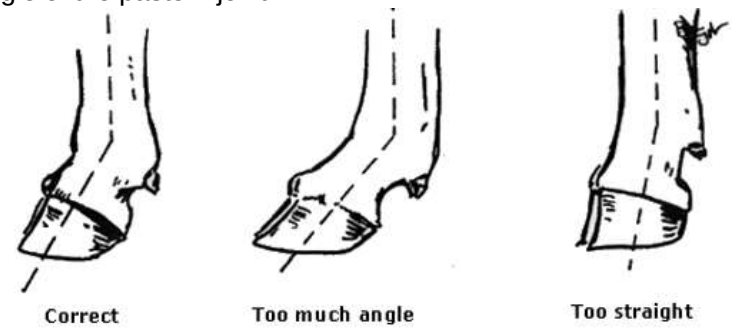


Fig. 39: This figure is indicating the correct angle of the pastern joint in the breeding bull (lateral view). The correct angle of the pastern joint (left), too much angle (middle), and too straight (left).

Uneven wearing of the two claws, where one grows longer than the other, is often due to a problem in the leg structure. It is caused by an uneven distribution of weight through the foot.

If the claws curl across each other without growing long, a serious genetic fault ('scissorclaw') may be apparent. These cattle wear the back of the hoof, causing lameness and reducing mobility.

Where excessive claw growth is caused by things other than structure (soft soil, heavy grain feeding, lack of exercise), extra pressure is placed on the leg joints, eventually causing lameness.

Feet

Avoid overgrown, scissor or curved claws (see Fig. 40). Mild curling is normal. It is exaggerated by heavy feeding and soft soils. Overgrown, uneven claws are usually indications of poor limb structure or early signs of hip arthritis. Avoid extremely short feet, which are often associated with over straight legs.

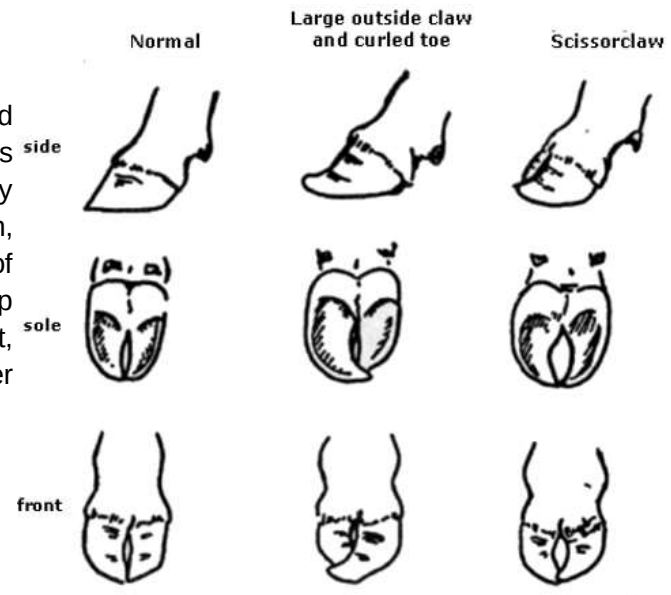


Fig. 40: This figure is indicating the normal and abnormal claws in side, sole, front views in breeding bulls.

Walk

Look for a free-moving gait, with the hind feet stepping into the footprints of the front feet (see Fig. 41). Overstepping or under-stepping are indications of structural problems, as are uneven footprints from the claws.

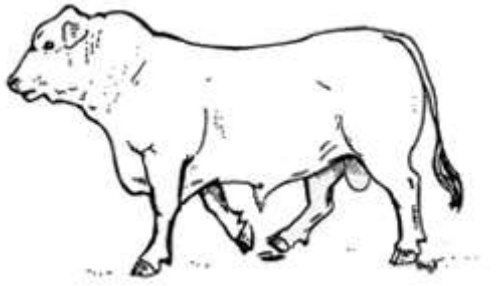


Fig. 41: This figure is indicating the normal walking pattern in breeding bulls.

Hind legs and feet

The structure of the hind legs is similar to that of the front legs. Again there are well-defined angles in the joints at the hip, stifle, hock and pastern joints. The angles are critical, particularly during serving when large amounts of stress are placed on these joints. Deviations from the correct angles (see Fig. 42) will cause excessive wear and tear on the joints, leading to early breakdown. More bulls seem to break down from problems associated with the hind leg than from any other reason.

Too much angle in the leg joints (straight-legged) is a serious structural fault. These cattle don't have the flexing and shock-absorbing effect of the structurally sound animal, and they are prone to severe wearing of the hip joint, leading to arthritis. Bulls showing arthritic problems are reluctant to serve many cows, as the condition can be quite painful.

When a bull mounts a cow, he straightens up the joints in his hind leg. When he thrusts, he further straightens the leg, placing enormous stress on all joints, but particularly the hock. If these joints don't have enough angulations, they become swollen and painful, leading eventually to their breakdown.

Straightness in the hind leg can be seen in the hock and pastern joints, and this indicates straightness in the stifle and hip. These cattle will wear the front of the claws, resulting in short upright hooves.

Straight-legged bulls are also much less athletic than the sound bull and appear to suffer a higher incidence of broken or damaged penises during serving.

A structurally correct bull, when walking, will place his hind foot in exactly the mark left by his front foot. If he is lame, or not moving freely, or if he is straight in his leg structure, he will short-step and not reach his mark. Likewise, if he is suffering arthritis in one leg he may tend to short-step or drag his leg on that side. Problems such as these will affect the serving ability of the bull.

If the degree of the angle in the leg joints is less than ideal, 'sickle hocked' condition may exist. This is less of a problem than straight legs, but in extreme cases may cause strained ligaments (pastern and hocks) and long claw growth,

increasing the chance of injury, and affecting serving ability. Sickle-hocked bulls may overstep the mark of the front feet as they walk out.
Sickle-hocked bulls appear to have less balance than do sound bulls. They may lose their balance during serving, therefore running a higher risk of injury.
Viewed from behind, the tibia and metatarsus (hock joint) should be in a straight line. A bull is 'cow hocked' when the hocks are rotated inwards and the hooves rotated outwards. This may cause problems but usually only in extreme cases where uneven pressure on the claws may cause the outside claw to grow long.
A more serious problem occurs where the legs are wide at the hocks ('bow-legged'), but the feet are turned in. Extra strain is placed on the ligaments of the hock joints causing lameness and even permanent damage.

Leg angulation
Post legs (straight hocks)
See Fig. (42)
These cattle generally walk short and have worn 'boxy' toes. They are prone to arthritis in the hips and other joints, and damage to the ligaments. Penis damage can occur due to serving accidents, as these bulls have poorer balance than do structurally correct bulls. Libido will naturally decline.

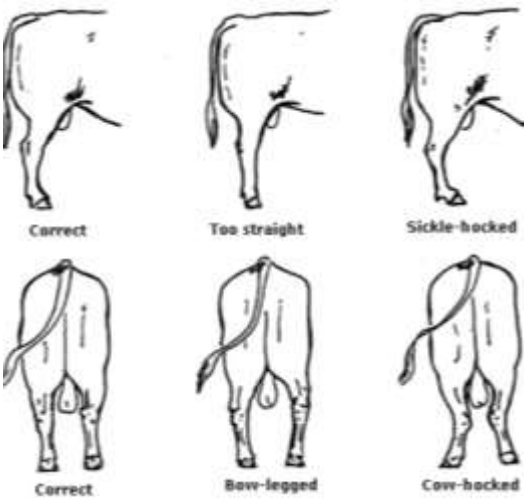


Fig. 42: This figure is indicating the leg angulation. Lateral and back view

Sickle hocks (the opposite to straight hocks)
See Fig. (43)

The joints are over-angled, leading to overstepping and long overgrown claws and lameness. Moderate cases are not a concern, but bad sickle hock strains ligaments, damages the dew claw and affects serving ability.

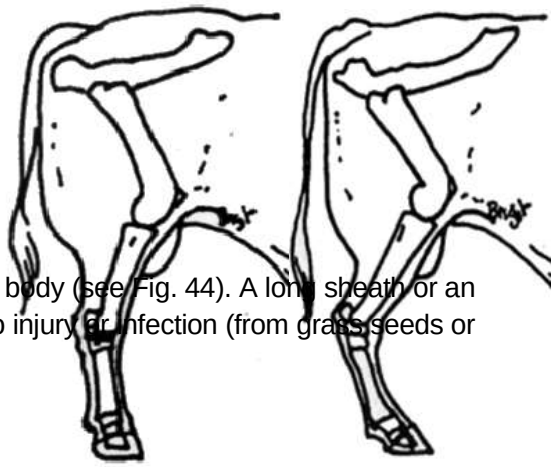


Fig. 43: This figure is indicating the sickle hocks (lateral view)

The sheath
The sheath should be trim and close to the body (see Fig. 44). A long sheath or an excessively angled sheath is more prone to injury or infection (from grass seeds or

other foreign objects) and should be avoided. Some breeds are more susceptible to these problems and buyers of these cattle must be critical in their selection.
A slack prepuce (the fold of skin covering the penis) should also be avoided. A bull who lets his prepuce hang out for long periods of time should be regarded as having a serious structural fault.

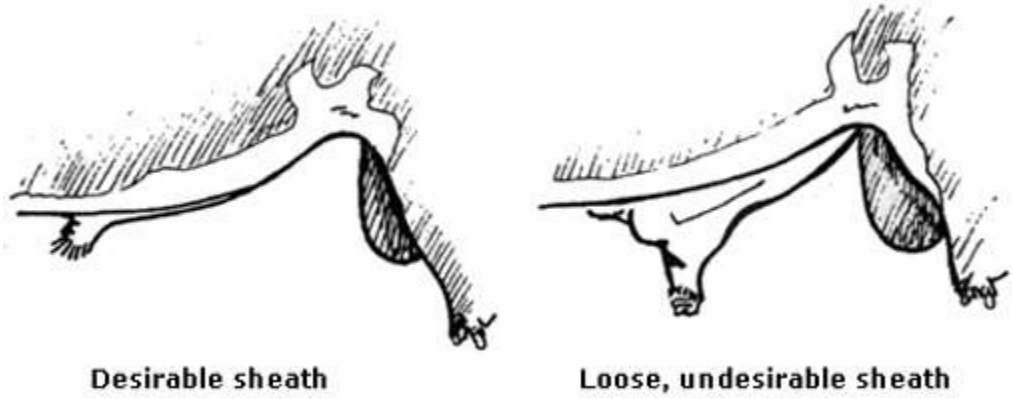


Fig. 44: This figure is showing the desirable perpetual sheath (left) and the undesirable loose preputeal sheath (right).

5.2. Capability of the reproductive Organs

Sperm is produced continuously by the testes and stored in the epididymis. The prostate gland, seminal vesicles and cowper's glands secrete the fluid component of the semen. During mating, the penis is extruded from the sheath by the straightening of the S-shaped sigmoid flexure; sperm are transported up the vas deferens to the urethra and exit via the penis.

Deep body temperatures are too warm for proper sperm production. This is why testes are located outside the body core. As the environmental temperature changes, the testes are raised and lowered in the scrotum to maintain proper temperature for sperm production.

Abnormalities of the Reproductive Organs

Various conditions can affect the function of the reproductive tract. If the testicles cannot move because of fat pads, scar tissue or a small scrotum, proper temperature cannot be maintained and semen quality may suffer. Soft testicles indicate degeneration of tissue and poor semen quality. Very small testicles indicate unsatisfactory development of sperm-producing tissue. Severe frost-bite scabs, tumors or abscesses also indicate potential problems. Infection and inflammation can occur in any of the reproductive organs. If the testicles become inflamed, the semen quality may be impaired long after the

original condition has passed, since it takes approximately 60 days for new sperm to be produced and mature.

Common penile problems include:

- Spiral penile deviation
- Persistent frenulum
- Penile hair rings.

Penile hair rings are most often seen on young bulls. A band of hair encircles the penis. If the condition remains untreated infection and scarring may result. Other conditions that can affect the penis include fractures, warts and scarring from previous injuries.

Scrotal Circumference

Measuring the scrotal circumference of young bulls is an accurate, repeatable method to assess current and future sperm-producing ability (see fig. 45). The measurement gives an estimate of the weight of the testes, which is directly related to the level of sperm production. Scrotal measurement is also positively correlated with semen volume and quality. Table 2 contains minimum recommended scrotal measurements by breed and age. Bulls with adequate scrotal development for their age have a higher probability of becoming satisfactory breeders than bulls with smaller scrotal circumferences.

Scrotal circumference is of medium to high heritability. Fertility of the male offspring can be increased by selection for this trait. The scrotal circumference of a bull is also positively related to the fertility of his daughters. Heifers from sires with larger than average scrotal circumference tend to reach puberty earlier than those from bulls with smaller scrotal circumferences. Increased scrotal circumference in sires is also favorably correlated to their daughter's age at first breeding, pregnancy rate and days to rebreeding after calving. Due to low heritability, direct selection for female fertility traits has not been successful. The strong genetic relationship between scrotal circumference and female reproductive traits provides an alternative selection method.

Scrotal circumference gives an indication of a bull's ability to produce sperm and is related to younger age at puberty. Breeds differ somewhat as to scrotal circumference, but 32 centimeters is generally accepted as the minimum size for yearling bulls to be sound breeds.

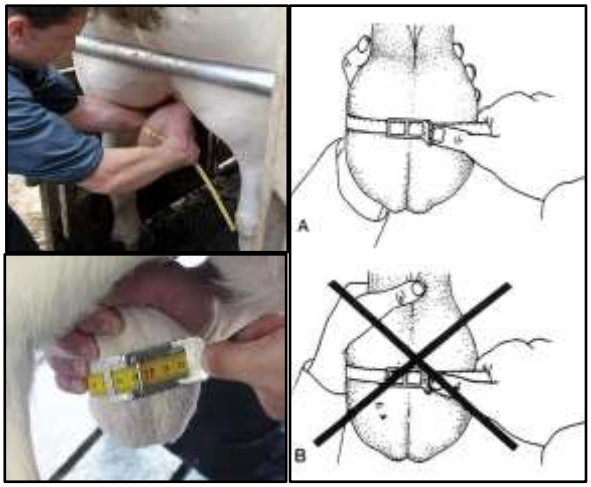


Fig. 45. Showing measurement of the scrotal circumference in bull and ram.

5.3.Semen Quality:

The criteria commonly used to evaluate semen quality include sperm morphology (structure) and motility (rate and percent of progressive forward movement). Semen volume and concentration can also be used.

Consider the following points when assessing the results of a semen test.

- In the field, correlations between semen quality traits and fertility have been low to moderate.
- The repeatability of semen evaluations of the same bull over time has been low.

Four criteria are used to evaluate semen, i.e., volume, concentration, motility and morphology. Proper training is required to accurately evaluate semen. A scoring system for predicting potential breeding soundness of bulls as prepared by the Society for Theriogenology, which incorporates scrotal circumference, semen motility, and semen morphology, is shown in Table 3.

Breeding soundness examinations presently do not include an evaluation of a bull's sexual drive or libido. Libido testing is time consuming and requires use of females in estrus and therefore is difficult to conduct on a large scale. Procedures are being investigated to better evaluate the willingness and desire of bulls to mate.

While a bull must produce some viable sperm in order to be fertile, semen quality is only one aspect of total fertility and must be evaluated in conjunction with all the other factors.

5.4. Libido

Libido (sex drive) is a critical component of fertility. It is independent of scrotal circumference, semen quality, body weight, growth rate or masculinity. One method of measuring libido is to measure serving capacity. This is done by exposing the bull to a group of restrained females and recording the number of mounts and services completed in a given time period.

When 2 or more bulls are used at the same time, in the same pasture, social interactions affect breeding performance. Social rank is related to age and seniority in the herd. The most dominant bulls tend to complete the highest number of services. In this situation, the number of cows serviced may be related more to social dominance than libido as measured by a serving capacity test.

5.5. Nutrition

Proper nutrition is necessary for good reproductive performance. Balanced amounts of protein and energy are required for sperm production and the physical activity associated with breeding. Adequate amounts of vitamins and minerals are also important in reproduction.

During the breeding season, bulls tend to eat less feed than is required to maintain their body weight. At this time they use body fat for energy and may lose up to 68 kg (150 lb). Supplying a grain mix to bulls on pasture is not always effective. Proper pre-breeding nutrition is essential to ensure the bull has adequate reserves for a successful breeding season.

Excessive fat deposits in the scrotum may interfere with temperature regulation. The degree of body fat required to adversely affect sperm production has not been well defined. Extreme fatness has been associated with low serving capacity. On the other hand, large breed yearling bulls starting the breeding season with minimal levels of backfat may have poorer semen quality than similar bulls carrying a moderate level of backfat. The nutrient requirements needed to optimize reproductive performance in breeding bulls needs more research.

Table 2:

Classification	Scrotal circumference in centimeter			
	12 to 14 m	15 to 20 m	21 to 30 m	30+ months old
Very Good	More than 35	More than 37	More than 39	More than 40
Good	30 to 35	31 to 37	32 to 39	33 to 40
Poor	Less than 30	Less than 31	Less than 32	Less than 33

[1]Examination as recommended by the Society for Theriogenology, revised, September 1976.[2]Key to morphology section — spheroids:

Less than 5/HP field = occasional = + 5 percent primary abnormality
5 to 15/HP field = few = + 15 percent primary abnormality
15 to 25/HP field = many = + 25 percent primary abnormality
More than 25/HP = multitudes = + 35 percent primary abnormality

[3]Scrotal circumference data based on data from Angus, Charolais, Hereford and Simmental breeds.
[4]Scoring system — satisfactory potential breeder has between 60 and 100 points; questionable potential breeder has between 30 and 59 points; unsatisfactory potential breeder has 29 points or less

Table 3:

Male reproduction evaluation for breeding soundness[1] (from *Guidelines for Uniform Beef Improvement Programs,* fifth edition, April 1986, BIF). (Table divided into 3 sections.)

Classification	Motility Score	Morphology	
		Primary abnormalities	Total abnormalities
	Number	Percent	
Very Good	More than 30	Less than 10	Less than 25
Good	20 to 30	10 to 19	26 to 29
Fair	12 to 20	20 to 29	40 to 59
Poor	Less than 12	More than 29	More than 60

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