# Reproductive disorders of male camelids

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## Introduction

Unlike the cattle and sheep industries, there are no standards for male breeding soundness examination classifications in camelids. Some guidelines have been cited in the literature as to minimum requirements for testicular size and semen quality; however, many males, outside of South America, are bred to few females, and may produce offspring despite any observed abnormalities in testes size or semen quality. Maximal reproductive performance is often not required of these males. Furthermore, males are often selected as a herd sire based on pedigree, fiber quality, conformation, or show performance, and not on reproductive parameters.

The most common reasons for which male camelids are presented to the veterinarian for reproductive evaluation include: 1) clinically normal males for breeding soundness examination (BSE; including prepubertal and adult males); 2) males with reproductive problems (including infertility and abnormal breeding behavior); and 3) reproductive emergencies (primarily scrotal/testicular or penile injuries). Retrospective analysis of cases at the authors' clinic demonstrated that of 164 males which had been presented for evaluation, infertility was most commonly due to testicular hypoplasia and testicular degeneration (45%) and testicular or epididymal cysts (17%).[1] In proven males, the most commonly diagnosed cause of infertility was testicular degeneration secondary to heat stress.[1]

This paper will discuss the clinical examination of the male camelid for reproductive evaluation, noting specific findings associated to subfertility or infertility. Reproductive emergencies will be covered in a subsequent paper in these proceedings.

## Selection of the herd sire

Selection of a herd sire begins at birth. The male should have two descended testes and no other congenital defects. Any significant cardiovascular, musculoskeletal, or visceral defects should remove the animal from breeding consideration due to the unknown heritability of most defects in these species.[2,3] The male should be examined by the veterinarian starting at six months of age. Testicular measurements as well as ultrasonography should be performed to ascertain the presence of rete testis cysts and record the size and location, if present. Presence of cysts will not remove a male from breeding consideration unless they obliterate the entire testicular parenchyma. The initial examination of a male will serve as a baseline as he grows and matures and can be referred to in the case of any changes in the yearly BSE or fertility.

The male alpaca or llama reaches sexual maturity at 4-5 years of age although many are used for breeding starting at two or three years of age.[4] As a general rule all prospective herd sires should undergo a complete veterinary breeding soundness examination prior to use around two years of age. This examination aims to determine the ability of the male to produce and deliver spermatozoa (normal spermatogenesis, sperm transport, and mating behavior). Some young males may show signs of immaturity (poor sperm quality and quantity). A final decision on the breeding ability of the male is usually achieved at three years of age.

## History

Any male camelid which presents to the veterinarian for reproductive examination should be accompanied by a detailed health and reproductive history, including all previous examinations of testicular size, ultrasonography, and semen. Unfortunately, in many cases, the first examination of a male by a veterinarian is upon identification of a problem, whether physically visible (such as penile or scrotal injury) or observed after review of the breeding records (such as increased number of open females or number of breedings or cycles per pregnancy).

Breeding records are extremely valuable, with the inclusion of the name or identification number of the female which was bred, date and duration of breeding, and outcome (establishment of pregnancy). Increased observation of interrupted breeding should be noted. Breeding management scheme is also important: breeding in-hand, breeding in a small pen, or pasture breeding. Travel history should be included, particularly to shows or other breeding farms where contact with sick animals may have occurred. General health history should include episodes of fever, skin lesions, lameness, colic, or injuries. Nutritional history should include the type of feed (hay, concentrates, pasture access), and any hay or trace mineral analysis, if previously performed.

For specific complaints regarding the reproductive tract, the duration and severity should be noted. For example, acute onset of scrotal enlargement should be differentiated from chronic. For penile injuries due to masturbation, the frequency, duration, and objects bred should be recorded. For decreased libido or difficulty completing the breeding, a time-frame and associated other events (travel, injury) should be noted.

In many cases, males are sold and purchased without a reproductive examination. Conscientious owners may request a BSE as part of a pre-purchase agreement. Examination in these cases may prevent loss of money, time, and labor if a male is unable to perform to expectations.

**Clinical examination**

Physical examination

Clinical examination, after review of the history and breeding records, begins with general inspection of the animal. Any systemic disorders should be investigated (enlarged lymph nodes, heart murmur, etc.). Reproductive examination begins with examination of the scrotum. The testes should be present in the scrotum at birth. The scrotum is non-pendulous but may appear so in older males or animals which have experienced testicular degeneration.[5] The testes are nearly equal in size and may be slightly off-set. The head of the epididymis may be palpated as a small firm structure. The skin of the scrotum should be examined for punctures, lacerations, or dermatitis. The testes are measured for height and length (Table 1),[6] and ultrasonography is used to evaluate the parenchyma for presence of rete testis cysts or hyperechoic areas suggestive of fibrosis.

The penis may be examined under sedation or anesthesia. The glans is inspected for any lesions, including pustules, lacerations, hair-rings, or abrasions. The preputial attachment should be freed by two to three years of age.[4] In a recent study in the authors' laboratory, 85% of males had observation of preputial detachment by 18 months of age.[7] Inability to exteriorize the penis in an adult male should raise suspicion of adhesions or persistent frenulum.

Some males may present dysuria or stranguira. Urolithiasis is a common finding. In pubertal animals the prostate may enlarge under testosterone influence resulting in transient stranguria.

Examination of the internal genitalia may be required, usually as preparation for electroejaculation. The prostate and bulbourethral glands are examined for size and presence of any lesions. The distance from the prostate to the anus is measured to assist in proper placement of the electrodes during semen collection.

A table of reproductive lesions which have been diagnosed in male camelids at Washington State University is provided in Table 2.[6]

Semen collection and analysis

Semen collection in camelids is difficult due to the nature of ejaculation: semen is dribbled continuously throughout the mating period. There is no discrete ejaculate volume as observed in other species.

Semen may be collected by one of several methods in camelids. First, the male may be mated to a receptive female and semen aspirated from the female tract using a sterile insemination pipette. This method of semen collection will allow for observation of the libido of the male, and his physical ability to

mate. However, aspirated semen is typically contaminated with erythrocytes and/or leukocytes from the female tract.

An uncontaminated sample may be obtained by electroejaculation (EE). This technique is most successful if the male does not have a full bladder. The animal is placed under general anesthesia in lateral recumbency. An electroejaculation probe with linear, non-circumferential electrodes is placed over the prostate, at the distance measured ultrasonographically earlier in the examination. The penis is exteriorized and held manually with a collection tube. Electrical stimulation is applied starting at very low voltage. Despite general anesthesia, electrical stimulation will result in hind limb movement and muscle contraction. In a series of studies performed in the authors' laboratory, EE was shown not to incite a higher stress response than anesthesia alone.[8] Furthermore, EE resulted in collection of samples adequate for evaluation, ranging in volume from 0.25 to 1.75 mL.[9]

Some males may be trained to serve an artificial vagina (AV). However, facilities must have a suitable mounting dummy, often with integrated circulating warm water to maintain ejaculate temperature over the mating period, and the AV must be carefully constructed to simulate the intrauterine breeding of these species.[10] This technique is not feasible for semen collection in most males.

Examination of the semen includes motility, volume, color, and viscosity. The motility is variable and dependent on the viscosity of the sample – the authors include an "activity" parameter in viscous samples rather than progressive motility in some cases. Morphology is variable – some studies found greater than 70% normal morphology,[11] however, 50% normal morphology is not unusual in sexually rested males.[6] Cytology should not demonstrate leukocytes or erythrocytes in ejaculates collected by EE or AV.

Advanced diagnostic techniques for infertility or subfertility include testicular biopsy, trace mineral assay, endocrinology, and cytogenetics.[12] Testicular biopsy is useful in cases of azoospermia, testicular hypoplasia, and neoplasia.[13] The technique can help distinguish azoospermia of testicular (spermatogenic arrest) or non-testicular (occlusion of the epididymal ducts, ductus deferens, etc.) origin. Trace mineral assay can help to identify deficiencies in zinc, copper, and selenium, all minerals which impact sperm development and function. Endocrinology testing can determine if poor libido is due to low testosterone or to diagnose cryptorchidism/ectopic testis. Cytogenetics can identify chromosomal abnormalities in males which have poor semen quality, infertility, or pregnancy loss in bred females.

**Evaluation of specific complaints**

Poor libido

Poor libido in male camelids is not well understood as far as the eitiopathogenesis or predisposing factors. In young males, lack of sexual interest may be related to inexperience, shyness, or previous reprimands by handlers.[6] In these cases, initiating of breeding in a familiar environment (i.e., on the farm, and not at the veterinary clinic), or observation of a mature male completing a breeding may increase the libido of the young male. Alternatively, hormone imbalance or systemic disease may affect sexual performance.[6] Serum endocrinology can evaluate testosterone levels, and a complete veterinary examination should be performed. Young males may require some patience to develop or encourage training of normal sexual behavior.

In the adult male, or proven herd sire, any decrease in libido should be immediately investigated. The most common causes of decreased libido are systemic infections, musculoskeletal disorders including arthritis, or poor body condition.[6] Additionally, heat stress or group housing with females may result in decreased sexual interest over time.

Abnormal erection

Male camelids breed intrauterine within the female reproductive tract. Therefore, to complete a breeding, the penis must free of its preputial attachments, of sufficient length, of normal shape to penetrate the cervix, and free of lesions. As mentioned previously, preputial detachment of the penis should occur in all males by two to three years of age.[4] Inability to exteriorize the penis may indicate

persistent frenulum or development of preputial-penile adhesions. Congenital short penis has been observed in some cases of infertility. In these cases, the male is unable to complete the intrauterine breeding, which can result in infertility both by failure of intrauterine deposition of semen and failure to induce ovulation. Penile lesions may include lacerations, hair rings, ulcerations, warts, or other lesions which may inhibit mating ability due to pain. Last, one of the authors (AT) has seen cases of erection failure due to neurologic disease caused by meningeal worm infection.

Azoospermia and oligozoospermia

Many cases of infertility in camelids are due to azoospermia or oligozoospermia, the presence of no or very few spermatozoa in the ejaculate, respectively. Azoospermia may be of congenital or acquired pathogenesis. Congenital azoospermia is most often due to inherent defects in spermatogenesis or outflow obstruction via segmental aplasia along the epididymal ducts or ductus deferens. Testicular hypoplasia affects up to 10% of male camelids.[14] The authors have observed a male in which spermatogenesis did not progress through meiosis. In animals with outflow obstruction, spermatogenesis may progress normally (as verified by testicular biopsy). However, ejaculation results only in emission of accessory sex gland secretions, with no testicular component.

Acquired azoospermia is usually of testicular origin. The most common inciting cause is testicular degeneration, typically due to heat stress, but may be reflective of other systemic processes.[9] Azoospermia may be suspected based on infertility, failure to aspirate spermatozoa from a mated female, or failure to identify spermatozoa in a collection via EE. Confirmation is achieved by testicular biopsy, which may demonstrate varying degrees of testicular degeneration and/or fibrosis.[5,13] As the spermatogenic cycle in camelids is thought to be approximately 60 days, sexual rest for at least two months may demonstrate resumption of spermatogenesis if the insult was transient; however, in many cases the effects are permanent. The authors have treated several males with experimental hormone therapy of follicle stimulating hormone (FSH) and luteinizing hormone (LH) to try and increase spermatogenesis, which demonstrated variable efficacy and a high rate of development of savage behavior in treated males.[12] Azoospermia may also be observed in males with large rete testis cysts which obliterate the entire normal testicular parenchyma.[15]

The authors have recently shown that measurement of seminal plasma alkaline phosphatase is not a marker of ejaculation in alpacas, compared to in dogs and horses.[9] Therefore, testicular biopsy remains the gold standard for diagnostic evaluation of azoospermia.[13]

Teratozoospermia

High proportion of morphologically abnormal spermatozoa in the ejaculate is classified as teratozoospermia and may reflect inherent defects in spermatogenesis or epididymal maturation.[16] In other species, a high percentage of morphologically normal spermatozoa are required for a male to be classified as a satisfactory potential breeder (i.e., 70% in bulls). However, male camelids may demonstrate high percentages of abnormal morphology. Studies have demonstrated successful herd sires which only demonstrated at times 50% morphologically normal spermatozoa.[16,17] Many other non-breeding males which were submitted to the authors' practice had less than 30% normal morphology.[9] Several other studies in both alpacas and llamas have demonstrated highly variable normal morphology.[10,11,17,18] As the BSE is not standardized for camelid species, and because the proportion of males submitted for BSE is low (compared to the number of breeding males in use), much research is needed to determine the parameters of an ejaculate which are required for maximal fertility. Males outside of South America are rarely taxed to their reproductive capabilities, and so reduced numbers of normal sperm may not result in reduced numbers of pregnancies.

Evaluation of teratozoospermia may require use of several staining methodologies.[19] The traditional eosin-nigrosin stain for morphology may help to identify the specific morphologic abnormalities which are present. Specialized stains such as Spermac® are used to identify acrosome integrity. In one case, the authors utilized electron microscopy to diagnose abnormalities of the spermatozoa ultrastructure at the level of the mitochondrial sheath.[19]

For diagnosed cases of teratozoospermia, the prognosis is guarded. A thorough evaluation of the nutrition, trace minerals, and general health status of the animal is warranted.[12] Testicular ultrasonography and biopsy may provide diagnostic information. In some cases, despite at least 60 days sexual rest and correction of all identified management issues, the etiopathogenesis of the condition remains undiagnosed.

Other causes of poor fertility

*Rete testis cysts.* Observations of rete testis cysts within the testes of male alpacas have been well documented but the etiopathogenesis and prevalence of these lesions remains poorly defined. Ultrasonographic examination was performed on 173 male alpacas presented for castration.[20] Rete testis cysts were identified in 18.5% of animals; 40.6% of cases were bilateral. Cysts ranged in size from 4-45 mm (mean ± SEM; 13.3 ± 1.3) in length and 2-28 mm (6.5±0.8) in width. After castration, cysts were aspirated; 44.4% of cysts contained immature spermatozoa. All cystic testes had evidence of spermatogenesis; however, disruption was observed in testes with large cysts. Examination of the epididymis of affected testes demonstrated that 20% were completely devoid of spermatozoa. Rete testis cysts can be a cause of subfertility or infertility and ultrasonographic examination of the testes should be included in the male breeding soundness examination as they are not palpable externally.[21]

*Testicular degeneration.* One of the most common causes of acquired subfertility or infertility in male camelids is testicular degeneration. Testicular degeneration is hallmarked by disrupted spermatogenesis, decreased sperm output (oligozoospermia), increased abnormal morphology, or azoospermia. Histologically, seminiferous tubules may become smaller in diameter, with vacuolization and sloughing of the germinal epithelium. Inciting causes may include heat stress, high fever, trauma or inflammation of the scrotum or testes directly, chronic systemic illness, toxic insult, nutritional or hormone imbalance, or advanced age.[12] On clinical examination, the testes are smaller than expected for the age of the male, and excess scrotal skin may be apparent, with an increased appearance of pendulous testes. Ultrasonographically, the testes may have small hyperechoic areas which suggest fibrosis.[22] In many cases, once the inciting damage has occurred, it is irreversible. However, in cases where azoospermia has not yet developed, it may be possible to mate males on a restricted schedule to fertile females and achieve a pregnancy.

**Prognosis**

The prognosis for the life and breeding potential of an animal is highly dependent on the diagnosis. Severe penile injuries may require euthanasia, especially if preputial adhesions have developed. Testicular injury, degeneration, or neoplasia may require hemilateral or bilateral castration. Azoospermia due to spermatogenic arrest may improve with time and/or with experimental hormonal treatments; however, in many animals re-initiation of spermatogenesis does not occur. In some cases, as long as the male is producing some viable and morphologically normal spermatozoa, restricted breeding schedules may result in establishment of pregnancy in healthy, normal females.

**Conclusion**

Loss of breeding function of a herd sire can have severe effects on a herd, not only in terms of crias produced but also time, labor, and finances. Owners of male camelids would be well-served to present the animal at least one or two times annually to the veterinarian for reproductive examination. Furthermore, any lesions or abnormalities should be immediately addressed upon identification. Males which are known to fight with other males or breed the ground or objects should be inspected daily for injuries and placed in suitable housing (away from females/other males and with appropriate fencing). Performance of a reproductive examination prior to purchase of a herd sire is recommended to screen for reproductive abnormalities.

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**Table 1:** Recommended testicular size at specific ages in alpacas and llamas

| | Alpacas | | Llamas | |
|---|---|---|---|---|
| **Age (months)** | **Length (cm)** | **Width (cm)** | **Length (cm** | **Width (cm)** |
| 6 | 1 | 0.4 | 2.4 | 1.4 |
| 12 (1 yr) | 2.3 | 1.5 | 3.4 | 2.3 |
| 18 | 2.8 | 1.9 | 3.5 | 2.6 |
| 24 (2 yr) | 3.3 | 2.2 | 3.9 | 2.3 |
| 30 | 3.6 | 2.4 | 4.4 | 2.5 |
| 36 (3 yr) | 3.6 | 2.4 | 4.5 | 2.7 |
| Sires | 3.7 | 2.5 | 5.4 | 3.3 |

**Table 2:** Documented diseases of the reproductive organs in male camelids at the WSU Theriogenology service

| Prepuce | Penis | Testis and epididymis | Accessory sex glands |
|---|---|---|---|
| Preputial edema (Heat stress)<br>Obstruction<br>Laceration<br>Prolapse<br>Necrosis<br>Inflammation (Posthitis)<br>Warts<br>Phimosis | Prolapse<br>Paraphimosis<br>Inflammation<br>Ulcerations<br>Abrasions<br>Hair ring<br>Penile warts<br>Urethral rupture<br>Urethritis<br>Urolithiasis | Cryptorchidism<br>Ectopic testicles<br>Hydrocele<br>Testicular degeneration<br>Testicular hypoplasia<br>Testicular cyst<br>Orchitis<br>Epididymitis<br>Epididymal segmental aplasia | Prostate hypertrophy<br>Prostate abscess |