Infertility of male dogs has been less described in the literature than its female counterpart. Infertility can be classified as congenital, if it appears early in sexual life, or acquired, if it appears after the animal has been fertile. The presence or absence of normal libido also helps the clinician to categorize the infertility and to reduce the number of possible causes. Three common complaints concerning male dog infertility are observed: failure to mate, absence of libido, and failure to produce litters after normal matings. In practice, the third complaint—acquired infertility with normal libido—is most often seen: a dog that has been siring litters stops doing so despite a normal libido. These males may also show reduced libido if they are not treated (primary testicular failure). A detailed evaluation of the animal can lead to an etiologic diagnosis and appropriate treatment. Therapy should be initiated only after a definitive diagnosis has been achieved. Indiscriminate use of fertility drugs is usually ineffective, potentially dangerous, and frequently confusing for the veterinarian. This article aims to serve as a guide, via a systematic approach, to diagnosis and treatment of this reproductive problem of the stud dog.

**SIGNALMENT**

Evaluation of complete signalment of the affected dog ensures a correct interpretation of the complaint on the basis of the stud dog’s breed and age. Certain breeds, such as beagles, are prone to autoimmune orchitis, which may be associated with autoimmune thyroiditis. A normal total sperm count should also be interpreted in view of the body weight for each particular breed because a positive correlation between these two parameters has been noted. Also, advanced age has a negative influence on fertility.
HISTORY

Clinicians should question the owner about any change in the dog’s management, housing, medications and supplements, potential environmental toxins, concurrent training or competition, travel, and diet. Information pertaining to the general health history (past episodes of illness, hyperthermia, or fever) and previous laboratory tests should be recorded. The relationship between the timing of the management changes or health disorders and the appearance of infertility should be analyzed with care. Testicular infertility problems are not expected to appear simultaneously with insults but rather after at least one spermatic cycle (approximately 62 days5), and they may take 6 months or longer to improve. In contrast, epididymal insults may be evident in the spermogram in less than 14 days.5

Functional Evaluation

Functional evaluation of the ejaculation process should usually precede a stressful physical examination and blood and urine sampling; it should be repeated on another day if results are abnormal. Semen may be collected either on a table or on the floor, with good footing being essential. The male must be comfortable and secure to maintain good libido. Use of a teaser bitch allows collection of a better quality sample. The most frequently used method is manual stimulation: The prepuce is gently massaged with one hand, and as the penis becomes partially erect the sheath is pulled behind the bulbus glandis. With a gloved hand, the bulb is encircled with gentle, constant pressure. Functional abnormalities of ejaculation include aspermia, retrograde ejaculation, and incomplete ejaculation.

Aspermia (failure to ejaculate) may be due to sexual immaturity, drug therapy, psychologic factors, idiopathic causes, or secondary neuropathy. This result should be confirmed by several attempts at semen collection, with careful attention to providing a favorable environment. The use of a teaser bitch, frozen vaginal discharge from an estrual bitch, or synthetic pheromones (e.g., methyl p-hydroxybenzoate, Aldrich Chemical) will help rule out inadequate sexual stimulation.

Retrograde ejaculation of semen into the urinary bladder is a rare condition, and its etiology is not completely understood. Diagnosis requires collection of urine before and after mating or ejaculation; more spermatozoa will be present in the second sample. Retrograde ejaculation is clinically manifested as aspermia, oligozoospermia, or azoospermia (the specific type depending on the grade).
Acquired Infertility in Male Dogs with Normal Libido

Apprehensive dogs may ejaculate incompletely (only the first fraction is ejaculated) if they are in uncomfortable surroundings or are not stimulated by an estrous bitch. Diagnosis of incomplete ejaculation can be made by measuring levels of carnitine or alkaline phosphatase, which are produced by the epididymides and are used as markers. Carnitine testing, however, is not usually available to veterinarians. When a semen sample is collected from a male with poor libido or under inappropriate conditions, incomplete ejaculation should be considered as a diagnosis. This disorder can be distinguished from true testicular azoospermia by an alkaline phosphatase value of less than 5,000 IU/L (Figure 1).

ETIOLOGIC AGENTS

Acquired infertility may be caused by various agents that can be classified as pretesticular, testicular, or posttesticular (see boxes on this page).

DIAGNOSTIC TESTS

Many tests are used to arrive at a correct diagnosis of acquired infertility with normal libido (see box on p. 21). Selection of the appropriate methods depends on presenting signs. Invasive, risky, or expensive methods are usually proposed only after cheaper, safer ones have proved unsatisfactory.

Spermogram

The first evaluation usually performed in all cases is the spermogram, which permits identification of patients as those with normal semen and those with abnormal semen (Figure 2). Semen evaluation is necessary to determine the severity of the problem and to set priorities for further diagnostic testing. Diagnosis of dogs with normal semen may be benefited by the use of spermatozoa functional tests and electron microscopic examination of spermatozoa, which may reveal morphologic abnormalities that were not apparent during routine semen evaluation.

Several semen samples obtained on different days are necessary to determine the average semen quality for a particular dog, and multiple semen evaluations should be done for 90 to 120 days to permit regeneration of sperm in case a transient insult was present. Semen abnormalities and their most common causes are summarized in the box on p. 24.

Endocrine Testing

Dogs with normal libido usually have a normal gonadotropin-releasing hormone (GnRH)–luteinizing hormone (LH)–Leydig cell–testosterone axis, but the GnRH–follicle-stimulating hormone (FSH)–germ cell/Sertoli cell/inhibin axis can be abnormal. Increased

ETIOLOGIC AGENTS

Pretesticular

Incorrect breeding management, functional alterations (e.g., incomplete ejaculation), systemic illness

Testicular

Toxins, medications, elevated scrotal temperature, trauma, aging, irradiation, infectious agents (Brucella canis; Escherichia coli; Proteus vulgaris; Streptococcus, Staphylococcus, and Mycoplasma spp; canine distemper virus), immunologic and neoplastic disorders

Posttesticular

Bilateral duct obstruction caused by granuloma, spermatocele, or neoplasia; retrograde ejaculation

Some Agents Reported to Alter Reproductive Function

- Methyltestosterone
- Estrogens
- Betamethasone, prednisolone
- GnRH agonists and antagonists
- Ketoconazole
- Cimetidine
- Antineoplastic agents
- Glucocorticoids, anabolic steroids

Figure 1. Interpretation of the alkaline phosphatase value in azoospermic cases.
Acquired Infertility in Male Dogs with Normal Libido

Because FSH and LH are secreted episodically into peripheral blood, repetitive sampling (three determinations separated by 20 minutes) or challenge testing with GnRH is recommended for their evaluation. Unfortunately, commercial laboratory measurement of serum concentrations of canine gonadotropins or inhibin is not yet available. Testosterone is also secreted in a pulsatile manner, so a single determination is not accurate.

Alternatively, stimulation tests can be performed to evaluate the hypothalamic–pituitary–gonadal axis. In a blood sample drawn 1 hour after administration of GnRH (2 µg/kg IM) or 4 hours after administration of human chorionic gonadotropin (hCG; 40 IU/kg IM), the testosterone concentration should increase by 50% or more. If the testosterone response is abnormal, serum LH concentrations should be measured.16,17

**Testicular Biopsy**

In the case of persistent oligozoospermia or azoospermia in a dog with normal physical and clinicopathologic findings, the clinician can offer testicular biopsy as a diagnostic and prognostic tool, although less invasive procedures such as ultrasonography should always be used first. Because the procedure must be done aseptically, a sample of the tissue obtained can be cultured for the presence of microbial agents.

Testicular biopsy can also be used to determine whether the spermatogenic apparatus is intact and spermatogenesis is adequate and complete. Neoplastic and inflammatory lesions may be found by this method.18 In autoimmune orchitis, testicular biopsy performed early in the course of disease can identify lymphoplasmacytic inflammation.2

Fine-needle aspiration is the simplest procedure, with the sample obtained from the caudae epididymides or testis. Because leakage of semen outside of excurrent ducts may result in formation of sperm granuloma and antisperm antibodies, epididymal aspirates should be obtained only if the potential benefits outweigh the risks. Diagnosis of testicular problems can benefit from fine-needle aspiration, with the finding of mature spermatozoa indicating active spermatogenesis.

Core or incisional biopsy is a better technique for evaluation of spermatogenesis and testicular lesions. Concern about complications related to the procedure (e.g., inducing immune intolerance by disruption of the blood–testis barrier) should be weighed against the value of data obtained but is usually overridden by a pre-
Types of Semen Abnormalities and Their Most Common Causes

**Teratozoospermia:** >30% of morphologically abnormal spermatozoa in ejaculate

Acquired teratozoospermia can be due to inflammatory, neoplastic, or infectious diseases of the reproductive tract; increased temperature; and sexual abstinence. The presence of >10% primary abnormalities and 20% secondary abnormalities is cause for concern, particularly if the sperm count is at the lower limit. A high percentage of primary abnormalities indicates testicular disorders, whereas secondary abnormalities indicate epididymal problems or artifacts of semen collection or preparation. Fortunately, data linking specific morphologic abnormalities to infertility are rare.

**Asthenozoospermia:** <70% of progressive forward motility

Asthenozoospermia is usually associated with teratozoospermia (primary abnormalities of sperm: midpiece or tail defects) and shares most of its causes. Asthenozoospermia could be the first sign of infection with, for example, *Brucella*, or of prostatitis. Contaminated equipment (detergent, water, urine, lubricants) or use of sperm samples that have cooled to room temperature can cause this abnormality.

Spermatozoa motility is normally acquired during epididymal transit, so alterations in the epididymides can cause this abnormality without altering other semen characteristics. Incomplete ejaculation can cause asthenozoospermia, as only aged sperm cells from vas deferens are obtained. Lack of motility does not necessarily mean that spermatozoa are not viable.

**Necrozoospermia:** >50% of spermatozoa dead, as seen by viability staining, without other pathologic characteristics being detected

Necrozoospermia can be found in inflammatory or infectious diseases.

**Oligozoospermia:** <200 million sperm per ejaculate (≥4.5 kg body weight)

Small breeds may not have enough testicular mass to produce more than 20 million sperm/kg of body weight. Oligozoospermia can be caused by pretesticular, testicular, or posttesticular factors and is usually associated with subfertility.

**Azoospermia:** Ejaculation of seminal fluid without spermatozoa

Any pretesticular, testicular, or posttesticular process that alters spermatogenesis can cause oligozoospermia, which may progress to azoospermia. Epididymal markers can aid in differentiating secretory (testicular) from obstructive azoospermia (Figure 2). In bilateral outflow obstruction, the alkaline phosphatase value in seminal fluid is >5,000 IU/L, whereas dogs with true testicular azoospermia have alkaline phosphatase concentrations of >5,000 IU/L. Care should be taken in interpreting values for epididymal markers as they are likely to be influenced by androgenic control, and their concentrations may thus be altered in dogs with abnormal androgen production.

**Hemospermia:** Presence of blood in seminal fluid

Hemospermia usually occurs secondary to benign prostatic hyperplasia, prostatitis, penile trauma, or neoplasia of the genital tract. Hemospermia can decrease sperm cell longevity and is associated with infertility.

**Sperm agglutination:** Massing or clumping of sperm

Dogs chronically infected with organisms such as *B. canis* show agglutination of sperm caused by formation of antisperm antibodies. Testicular trauma, rupture of the blood–testis barrier, and immune-mediated diseases are also possible causes. In human semen, agglutination has been related to production of antisperm antibodies. Standard techniques for identifying antibody-coated sperm are not readily available in veterinary practice.

**Inflammatory ejaculate:** >10,000 aerobic bacteria/ml or >100,000 CFU/ml and >2,000 white blood cells/µl (leukospermia)

Inflammatory characteristics suggest infection. These results should be correlated with culture and ultrasound findings to localize the anatomic origin of infection (prostatitis, orchitis, epididymitis, or urinary tract infection).
Acquired Infertility in Male Dogs with Normal Libido

because artifacts produced by formalin make the specimen less valuable.

Unfortunately, by the time subfertility or infertility is noticed by the owner, the first insult is often not found; instead, diminished spermatogenesis and testicular atrophy are usually evident.

**TREATMENT**

Treatment should be based on the specific cause of the infertility (Table 2). If the seminiferous tubules are affected, improvement is not likely to be noted for at least 62 days after treatment, and a minimum of three spermatogenic cycles (6 months) is probably necessary before results of treatment can be fully assessed.

Damage to only the spermatogonia (seminiferous tubule failure) results in infertility with maintenance of normal libido. Tubular cell degeneration is progressive, and loss of plasma inhibin results in elevated FSH levels (total seminiferous tubule failure; Table 1). There is no effective treatment when the plasma FSH value has already increased. Some oligospermic dogs may have normal plasma FSH concentrations, probably representing an early stage (partial seminiferous tubule failure) of primary failure of spermatogenesis; these dogs may respond to treatment\(^\text{20}\) (Table 1).

Often, no reason for oligozoospermia or azoospermia can be identified, and the disorder is then classified as idiopathic. Several drug regimens have been proposed for the idiopathic disorder, although they are based largely on results in humans.\(^\text{20}\) Medical treatment of idiopathic oligozoospermia or azoospermia includes use of GnRH (1 µg/kg SC) with or without hCG (1,600 IU IM).\(^\text{21}\) A synthetic androgen, mesterolone, which stimulates testicular function without inhibiting gonadotropin release, was shown to improve semen morphology in an oligoasthenoteratospermic dog.\(^\text{22}\)

Antiestrogens such as clomiphene and tamoxifen are commonly used to treat idiopathic oligozoospermia or azoospermia in men. These drugs are synthetic, nonsteroidal estrogen analogues that stimulate pituitary gonadotropin secretion by blocking estradiol from specific receptor sites and, therefore, stimulating GnRH secretion.\(^\text{23,24}\) Contradictory data exist in the literature regarding antiestrogen-induced improvement of semen quality in humans.\(^\text{25,26}\) Similar studies of dogs are lacking, and duration of treatment and dose have not been established. Care should be taken with these compounds because they have a dose-dependent effect on germinal epithelium, and

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**Table 1. Diagnostic Findings for Selected Causes of Acquired Testicular Failure**

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Libido</th>
<th>Sperm Defect</th>
<th>Alkaline Phosphatase Level</th>
<th>FSH Level</th>
<th>LH Level</th>
<th>T2 Level</th>
<th>T2 Challenge Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary testicular failure</td>
<td>N or D</td>
<td>Oligozoospermia, azoospermia, or teratozoospermia (primary defects)</td>
<td>N</td>
<td>E</td>
<td>E</td>
<td>N or D</td>
<td>D</td>
</tr>
<tr>
<td>Partial seminiferous tubule failure</td>
<td>N</td>
<td>Oligozoospermia or teratozoospermia (primary defects)</td>
<td>N</td>
<td>N or E</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Total seminiferous tubule failure</td>
<td>N</td>
<td>Azoospermia or teratozoospermia (primary defects)</td>
<td>N</td>
<td>E</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Retrograde ejaculation</td>
<td>N</td>
<td>Oligozoospermia or azoospermia(^b)</td>
<td>N or D</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Duct obstruction</td>
<td>N</td>
<td>Oligozoospermia or azoospermia(^b)</td>
<td>A or D</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Incomplete ejaculation</td>
<td>D</td>
<td>Oligozoospermia or azoospermia(^b)</td>
<td>A or D</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
</tbody>
</table>


\(^b\)Depends on degree.

A = absent; D = decreased; E = elevated; N = normal; T2 = testosterone.
high doses may suppress gonadal function.\textsuperscript{27}

Alternative management of male dogs with oligozoospermia includes semen collection and intrauterine insemination of a proven fertile bitch on day 5 after an LH peak. Implications related to potential genetic problems should be considered before use of these procedures. Methods for intrauterine insemination include transcervical catheterization and surgical or laparoscopic deposition of semen.

**PROGNOSIS**

The prognosis for a dog that has spermatozoa in the ejaculate depends on the site and extent of the insult and ultimately on the etiologic diagnosis and appropriate therapy. Thus, blockage of excurrent ducts, although relatively easy to diagnose, requires surgical expertise for correction. Prostatitis can usually be managed medically. Dogs presenting with testicular causes of azoospermia generally have a poor prognosis for return to fertility, whereas those with oligozoospermia have a prognosis that is guarded to poor. With elevated FSH concentrations, treatments are unlikely to be of benefit because reserve spermatogonia are either absent or incapable of responding to additional FSH. Prognosis is guarded if no improvement in semen quality is seen after 3 months, poor if no improvement is seen after 6 months, and grave if no change is seen after 1 year.

**CONCLUSION**

Infertility of male dogs is one of the most puzzling disorder of small animal reproduction and represents a diagnostic challenge for practitioners. Evaluation of libido, spermogram, and hormone determinations usually permits an initial diagnosis\textsuperscript{25} (Table 1). With treatment, some disorders have a good prognosis for return to fertility. Other disorders, even when the site of insult and cause are determined, may be difficult to reverse and require development of new therapies.

Finally, advanced reproductive technology such as intrauterine insemination and in vitro fertilization can enable clinicians to assist breeders in obtaining a few more litters, but use of these tools should always be weighed against the possibility of propagating a disorder with genetic implications.

### REFERENCES


### Table 2. Selected Treatments of Acquired Infertility in Male Dogs

<table>
<thead>
<tr>
<th>Primary Cause</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blockage of excurrent duct</td>
<td>Microsurgery</td>
</tr>
<tr>
<td><em>B. canis</em> infection</td>
<td>Castration and removal from breeding</td>
</tr>
<tr>
<td>Prostatitis, orchitis, epididymitis</td>
<td>Specific antibiotics</td>
</tr>
<tr>
<td>Immune-mediated orchitis</td>
<td>Prednisone\textsuperscript{11}: 2–3 mg/kg/day</td>
</tr>
<tr>
<td>Retrograde ejaculation</td>
<td>Sympathomimetetics\textsuperscript{13}: phenylpropanolamine (3 mg/kg PO bid) or pseudoephedrine (4–5 mg/kg PO 1–3 hr before mating)</td>
</tr>
<tr>
<td>Benign prostatic hyperplasia</td>
<td>Antiandrogens\textsuperscript{19}: finasteride (0.1–0.5 mg/kg PO)</td>
</tr>
<tr>
<td>Testicular neoplasia</td>
<td>Orchietomy</td>
</tr>
</tbody>
</table>


3. **Canine oligozoospermia is defined as**
   a. more than 200 million sperm/ejaculate.
   b. more than 200 million sperm/kg of body weight.
   c. less than 200 million sperm/ejaculate.
   d. less than 200 million sperm/kg of body weight.

4. **Teratozoospermia in an ejaculate is defined as**
   a. more than 30% of morphologically abnormal spermatozoa.
   b. less than 70% progressive forward motility.
   c. more than 50% dead spermatozoa.
   d. more than 70% of morphologically abnormal spermatozoa.

5. **A semen alkaline phosphatase value higher than 5,000 IU/L in an azoospermic ejaculate suggests**
   a. bilateral duct obstruction.
   b. a testicular origin of the azoospermia.
   c. incomplete ejaculation.
   d. unilateral duct obstruction.

6. **Dogs with normal libido are supposed to have**
   c. increased testosterone production.
   d. a normal GnRH–LH–testosterone axis.
<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>7. Testicular biopsy</td>
<td>a. always ensures an etiologic diagnosis.</td>
</tr>
<tr>
<td></td>
<td>b. never has reproductive complications.</td>
</tr>
<tr>
<td></td>
<td>c. may result in reproductive complications.</td>
</tr>
<tr>
<td></td>
<td>d. is routinely carried out in clinical practice.</td>
</tr>
<tr>
<td>8. Benign prostatic hyperplasia can be treated with</td>
<td>a. finasteride.</td>
</tr>
<tr>
<td></td>
<td>b. hCG.</td>
</tr>
<tr>
<td></td>
<td>c. phenylpropanolamine.</td>
</tr>
<tr>
<td></td>
<td>d. prednisolone.</td>
</tr>
<tr>
<td>9. Mesterolone is</td>
<td>a. a synthetic androgen.</td>
</tr>
<tr>
<td></td>
<td>b. an antiestrogen.</td>
</tr>
<tr>
<td></td>
<td>c. an antiandrogen.</td>
</tr>
<tr>
<td></td>
<td>d. an estrogen.</td>
</tr>
<tr>
<td>10. An elevated serum FSH value in a dog with acquired infertility</td>
<td>a. portends a good prognosis.</td>
</tr>
<tr>
<td></td>
<td>b. portends a poor prognosis.</td>
</tr>
<tr>
<td></td>
<td>c. has no relationship to prognosis.</td>
</tr>
<tr>
<td></td>
<td>d. suggests an obstructive cause.</td>
</tr>
</tbody>
</table>