

REVIEW ARTICLE

A Review on Vaginal Prolapse in Dog Breeds

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ABSTRACT

Vaginal prolapse in dogs is a clinically significant reproductive disorder characterized by protrusion of vaginal tissue through the vulvar labia, predominantly occurring during estrus or parturition. This literature review synthesizes current knowledge regarding the etiopathogenesis, breed predispositions, clinical manifestations, diagnostic approaches and treatment modalities of canine vaginal prolapse. Evidence from peer reviewed literature indicates that brachycephalic and large or giant breed dogs demonstrate significantly higher prevalence rates with estrogen-driven edema during proestrus and estrus serving as the primary pathophysiological mechanism. Conservative and surgical management options are reviewed along with prognostic considerations and preventive strategies.

Keywords: Brachycephalic breeds, canine vaginal prolapse, estrogen edema, reproductive disorders, vaginal hyperplasia

INTRODUCTION

Vaginal prolapse (vaginal hyperplasia, vaginal fold prolapse or vaginal edema) is a condition in which vaginal mucosa protrudes beyond the vulvar lips of intact female dogs (Fossum, 2019). The condition is most commonly observed during proestrus and estrus phases of the reproductive cycle when elevated estrogen concentrations stimulate mucosal hypertrophy and edema (Johnston et al., 2001). Less frequently, the condition manifests during parturition or in the immediate postpartum period where physical straining and relaxation of pelvic ligaments predispose the vaginal floor to prolapse (Wykes & Soderberg, 1983).

The clinical importance of vaginal prolapse cannot be overstated. Beyond the immediate welfare implications, including pain, dysuria and susceptibility to

mucosal trauma, the condition carries significant reproductive consequences. Affected dogs may experience dystocia, inability to achieve natural mating, urethral obstruction and secondary bacterial contamination of exposed mucosa (Bigliardi et al., 2004; Root Kustritz, 2006). Furthermore, the condition demonstrates a well-established hereditary component, raising ethical concerns regarding the breeding of affected animals (Ettinger & Feldman, 2017). Despite its recognized clinical significance, vaginal prolapse in dogs remains an area where systematic, large-scale epidemiological data are limited. Much of the existing literature consists of case reports, case series and retrospective analyses from veterinary teaching hospitals. This review aims to consolidate available evidence, identify knowledge gaps and provide a structured reference for clinicians and researchers engaged with canine reproductive medicine.

CLASSIFICATION AND CLINICAL GRADING

The most widely adopted classification system divides vaginal prolapse into three grades (Fossum, 2019; Johnston et al., 2001)

Type I (Mild):

Mild folding of the vaginal mucosa within the vaginal vault. Tissue does not protrude beyond the vulvar lips and often detected only on digital or speculum examination.

Type II (Moderate):

A tongue shaped or donut shaped mass of edematous vaginal mucosa protrudes through the vulvar lips and urethral orifice may be displaced but remains accessible.

Type III (Severe):

Complete circumferential eversion of the vaginal wall and the entire vaginal cylinder is exposed. Urethral catheterization may be difficult and risk of ischemic necrosis is highest.

This grading system has been validated in multiple retrospective studies and is used to guide treatment decisions (Bigliardi et al., 2004; Pretzer, 2008).

EPIDEMIOLOGY AND BREED PREDISPOSITION

Overall prevalence

Vaginal prolapse is considered relatively uncommon in the general canine population, though exact prevalence figures are difficult to establish due to variable reporting and the likelihood of underdiagnosis in subclinical cases (Root Kustritz, 2006). Retrospective data from veterinary teaching hospitals suggest that it accounts for a small but consistent proportion of reproductive emergency presentations, particularly during spring and early summer months that correspond to peak estrous activity in many geographic regions (Schaefer-Somi et al., 2003).

Age of onset

The condition predominantly affects young, sexually mature, intact females during their first or second estrous cycle (Johnston et al., 2001). Meyers-Wallen et al. (1986) reported a mean age of onset of approximately 2.5 years in a retrospective case series, consistent with subsequent reports. While the condition can recur in successive estrous cycles without ovariohysterectomy, the severity may vary between cycles (Linde-Forsberg, 2005).

Breed predisposition

Breed predisposition is one of the most consistently reported epidemiological features of canine vaginal prolapse. Brachycephalic breeds and large-to-giant breed dogs are disproportionately represented in the literature (Ettinger & Feldman, 2017; Fossum, 2019). The genetic basis is supported by the familial clustering of cases and the recommendation against breeding affected individuals (Root Kustritz, 2006). Similar breed predispositions have been reported in Nepal and India (Koirala & Paudel, 2018; Kumar et al., 2015).

Hormonal mechanisms

The fundamental pathophysiological mechanism underlying vaginal prolapse in dogs is the exaggerated tissue response to estrogen during proestrus and estrus (Johnston et al., 2001; Romagnoli & Concannon, 2003; Sontas et al., 2003). Estrogen stimulates proliferation of vaginal epithelial cells and promotes fluid accumulation within the submucosal connective tissue, resulting in edema and

hypertrophy of the vaginal mucosa (Schaefer-Somi et al., 2003). In genetically predisposed individuals, this normal physiological response is amplified to a degree that causes structural prolapse (Root Kustritz, 2006).

Elevated circulating estrogen concentrations during proestrus trigger increased vascular permeability in the vaginal submucosa (Bigliardi et al., 2004). The floor of the vagina, particularly the region cranial to the urethral tubercle, is most susceptible due to its relatively loose attachment to surrounding structures (Fossum, 2019). As edema accumulates, the mucosal fold enlarges and may eventually protrude beyond the vulvar lips, constituting a clinical prolapse (Linde-Forsberg, 2005).

Table 1. Dog breeds most commonly affected by vaginal prolapse

Rank	Breed	Category	Relative risk	Reported frequency	References
1	English Bull dog	Brachycephalic	Very High	Most frequently cited breed in case series	Fossum (2019); Johnston et al. (2001); Ettinger & Feldman (2017)
2	Boxer	Brachycephalic	Very High	Consistently among top 3 reported breeds	Wykes & Soderberg (1983); Root Kustritz (2006)
3	Mastiff (English & Bull Mastiff)	Large / Giant	High	Frequently reported in large-breed case series	Bigliardi et al. (2004); Pretzer (2008)
4	Saint Bernard	Large / Giant	High	Multiple case reports and retrospective data	Johnston et al. (2001); Linde-Forsberg (2005)
5	Labrador Retriever	Large / Sporting	High	High overall population numbers inflate reporting	Schaefer-Somi et al. (2003); Root Kustritz (2006)
6	German Shepherd	Large / Herding	Moderate–High	Recurrent cases documented	Meyers-Wallen et al. (1986); Fossum (2019)
7	Great Dane	Giant	Moderate	Giant breed predisposition confirmed	Johnston et al. (2001); Fossum (2019)
8.	Doberman Pinscher	Large / Working	Low–Moderate	Occasional case reports	Root Kustritz (2006); Pretzer (2008)
9.	Rottweiler	Large / Working	Low–Moderate	Sporadic reports in literature	Ettinger & Feldman (2017)
10.	Mixed Breeds	Variable	Low	Underrepresented relative to purebreds	Meyers-Wallen et al. (1986)

Note: Relative risk categorizations are based on proportional representation in published case series and retrospective studies relative to population-level breed prevalence. **"Very High"** denotes breeds appearing in the majority of published case series; **"High"** denotes breeds frequently mentioned; **"Moderate"** denotes breeds with consistent but less frequent reporting; **"Low–Moderate"** denotes breeds with sporadic but documented occurrences.

Anatomical predisposing factors

Anatomical conformation plays a significant contributory role, particularly in brachycephalic breeds (Ettinger & Feldman, 2017). Shorter pelvic dimensions, altered perineal conformation, and reduced structural support of pelvic floor musculature in these breeds are hypothesized to lower the threshold for prolapse development (Fossum, 2019). In large and giant breeds, the increased body mass and gravitational forces on pelvic structures may similarly predispose to prolapse (Johnston et al., 2001).

Genetic factors

A hereditary predisposition is strongly suggested by the breed clustering of cases and the observation of familial recurrence (Meyers-Wallen et al., 1986). Root Kustritz (2006) explicitly recommends that dogs with vaginal prolapse not be used for breeding, both to prevent perpetuation of the hereditary predisposition and because affected females frequently cannot achieve natural mating due to the mechanical obstruction posed by the prolapsed tissue. The specific genetic loci or inheritance patterns have not been fully characterized, representing a gap in current knowledge (Pretzer, 2008).

Parturition associated sectors

A subset of vaginal prolapse cases occurs during or immediately after parturition, where the combination of physical straining, relaxation of pelvic ligaments under the influence of relaxation and progesterone withdrawal, and increased intra-abdominal pressure creates conditions favorable for prolapse (Wykes & Soderberg, 1983). These cases may involve true circumferential vaginal prolapse rather than the more common focal fold prolapse associated with estrus (Bigliardi et al., 2004).

CLINICAL PRESENTATION

History and signalment

Clinicians should maintain a high index of suspicion for vaginal prolapse in any intact female dog of a predisposed breed presenting with vulvar swelling, excessive licking of the perineum or apparent dysuria during proestrus or estrus (Root Kustritz, 2006). The owner may report observation of a pink-to-red tissue

mass protruding from the vulva which may be the first indication of a Type II or Type III prolapse (Fossum, 2019).

Physical findings

On physical examination, the hallmark finding is a pink, moist, edematous tissue mass protruding from the vulvar lips (Johnston et al., 2001). The mass may range from a small, tongue-shaped protrusion in Type II cases to a large, donut-shaped or spherical mass representing complete circumferential eversion in Type III cases (Bigliardi et al., 2004). The surface of the prolapsed tissue may appear smooth and glistening in early cases, but prolonged exposure leads to desiccation, ulceration, hemorrhage, and secondary infection (Schaefer-Somi et al., 2003). Urethral patency must be assessed in all cases, as the urethral orifice may be displaced or compressed by the prolapsed mass (Pretzer, 2008). Inability to pass a urinary catheter constitutes a medical emergency requiring immediate intervention (Fossum, 2019).

Systemic signs

Most cases of vaginal prolapse are not associated with systemic illness and affected dogs are typically alert, normothermic, and otherwise healthy (Johnston et al., 2001). However, secondary complications including urethral obstruction, ascending urinary tract infection and ischemic necrosis of the prolapsed tissue can produce systemic signs including pyrexia, lethargy, anorexia, and signs of sepsis in severe or neglected cases (Bigliardi et al., 2004).

DIAGNOSIS

Clinical diagnosis

In the majority of cases, diagnosis is straightforward and based on clinical presentation, signalment and reproductive history (Root Kustritz, 2006). The presence of a protruding vaginal mass in an intact female dog of reproductive age during proestrus or estrus is essentially pathognomonic (Fossum, 2019).

Vaginal cytology

Vaginal cytology is a valuable adjunct that confirms the stage of the estrous cycle and can support the clinical diagnosis (Johnston et al., 2001). During proestrus and estrus, cytological examination reveals predominantly superficial and

cornified epithelial cells with reduced numbers of neutrophils, consistent with estrogen dominance (Schaefer-Somi et al., 2003).

**Table 2. Causative and predisposing factors for vaginal prolapse in dogs
 (most common to least common)**

Rank	Causative	Category	Frequency	Mechanism	References
1.	Estrogen-driven mucosal edema during proestrus/estrus	Hormonal	Most Common	Elevated estrogen → submucosal edema → mucosal hypertrophy and protrusion	Johnston et al. (2001); Fossum (2019); Root Kustritz (2006)
2.	Genetic / Hereditary predisposition	Genetic	Very Common	Familial clustering; breed-specific amplified tissue response to estrogen	Meyers-Wallen et al. (1986); Root Kustritz (2006); Ettinger & Feldman (2017)
3	Brachycephalic conformation	Anatomical	Common	Altered pelvic geometry; reduced structural support; exaggerated estrogen response	Fossum (2019); Ettinger & Feldman (2017); Johnston et al. (2001)
4	Large / Giant breed body type	Anatomical	Common	Increased gravitational force on pelvic structures; greater intra-abdominal pressure	Johnston et al. (2001); Bigliardi et al. (2004)
5	Straining during parturition (dystocia)	Obstetric	Moderately Common	Increased intra-abdominal pressure + pelvic ligament relaxation	Wykes & Soderberg (1983); Bigliardi et al. (2004)
6	Relaxation of pelvic ligaments (relaxin effect)	Hormonal–Obstetric	Moderately Common	Relaxin and progesterone withdrawal reduce structural support at parturition	Schaefer-Somi et al. (2003); Linde-Forsberg (2005)
7	Chronic tenesmus/straining (non-obstetric)	Mechanical	Less Common	Rectal or urinary straining increases intra-abdominal pressure chronically	Pretzer (2008); Fossum (2019)
8	Exogenous estrogen administration	Iatrogenic	Less Common	Pharmacological estrogen → same mucosal edema pathway as endogenous hormone	Root Kustritz (2006); Johnston et al. (2001)
9	Ovarian cystic disease (follicular cysts)	Pathological–Hormonal	Less Common	Persistent follicular cysts produce sustained elevated estrogen	Bigliardi et al. (2004); Schaefer-Somi et al. (2003)
10	Poor perineal muscular support / conformation	Anatomical	Less Common	Reduced structural integrity of perineum and pelvic floor	Fossum (2019); Linde-Forsberg (2005)
11	Previous vaginal trauma or scarring	Pathological	Uncommon	Disruption of normal tissue architecture reduces resistance to prolapse	Pretzer (2008); Ettinger & Feldman (2017)
12	Obesity	Systemic / Metabolic	Uncommon	Increased intra-abdominal fat increases chronic pressure on pelvic structures	Root Kustritz (2006); Fossum (2019)
13	Recurrent estrous cycles without pregnancy	Reproductive	Uncommon	Repeated estrogen stimulation without the protective effects of gestation	Meyers-Wallen et al. (1986); Johnston et al. (2001)
14	Secondary bacterial infection (perpetuating factor)	Infectious	Uncommon (secondary)	Infection causes mucosal swelling, edema, and prevents spontaneous reduction	Bigliardi et al. (2004); Schaefer-Somi et al. (2003)
15	Nutritional deficiencies	Nutritional	Rare	Poor collagen synthesis reduces structural support of vaginal wall	Pretzer (2008); Linde-Forsberg (2005)

	(connective tissue integrity)				
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Note: Frequency designations are based on relative reporting rates across published case series and reviews. "Most Common" to "Rare" reflect the weight of evidence in the available literature rather than precise epidemiological quantification.

Cytology also helps differentiate vaginal prolapse from vaginal neoplasia, where exfoliated neoplastic cells may be identified (Pretzer, 2008).

Vaginoscopy

Vaginoscopy allows direct visualization of the vaginal lumen and characterization of the prolapsed tissue, facilitating classification by grade and identification of the urethral orifice (Linde-Forsberg, 2005). It also enables differentiation between true circumferential prolapse and focal fold prolapse which has therapeutic implications (Fossum, 2019).

Imaging

Radiography and ultrasonography are not routinely required for diagnosis of uncomplicated vaginal prolapse but may be indicated when complications are suspected (Ettinger & Feldman, 2017). Abdominal ultrasonography can identify concurrent uterine pathology, ovarian cysts or urinary tract abnormalities (Bigliardi et al., 2004). In cases of suspected neoplasia, computed tomography may be employed to assess for local invasion or metastatic disease (Pretzer, 2008).

Histopathology

Biopsy of the prolapsed tissue is rarely required for diagnosis of uncomplicated vaginal prolapse but is indicated when neoplasia cannot be excluded on clinical grounds (Root Kustritz, 2006). Histopathological examination reveals marked edema, vascular congestion and epithelial hyperplasia of the vaginal mucosa, without evidence of cellular atypia in benign cases (Schaefer-Somi et al., 2003).

Differential diagnosis

Several conditions must be differentiated from vaginal prolapse including (Fossum, 2019; Johnston et al., 2001):

- Vaginal neoplasia: Transmissible venereal tumor (TVT), leiomyoma, fibroma and adenocarcinoma may produce vaginal masses. TVT is

particularly important in endemic regions and is distinguished by cytological and histopathological examination.

- Clitoral hypertrophy: Enlargement of the clitoris may be confused with vaginal prolapse. Digital examination and vaginoscopy clarify the anatomical origin.
- Uterine prolapse: True uterine prolapse is rare in dogs and involves protrusion of the uterine body or horn and it is most commonly postpartum.
- Urethral prolapse: Primarily reported in male dogs but documented in females. It involves protrusion of the urethral mucosa at the external urethral orifice.
- Perineal hernia: Disruption of the pelvic diaphragm may produce perineal swelling that can be confused with genital prolapse.

MANAGEMENT

Conservative management

For Type I and mild Type II cases, conservative management may be appropriate, particularly if the owner declines surgery or the dog is intended for breeding (Root Kustritz, 2006). Conservative measures include manual reduction of the prolapsed tissue, application of hypertonic agents such as dextrose solution to reduce edema, lubrication with water soluble gels, application of protective bandaging and placement of a temporary vulvar retention suture to prevent re-prolapse (Fossum, 2019; Johnston et al., 2001).

Medical management with progestins or GnRH agonists to suppress estrus has been employed as an adjunct to conservative treatment (Pretzer, 2008). The goal is to hasten the transition from estrus to diestrus, reducing circulating estrogen and thereby facilitating spontaneous resolution of the edema (Linde-Forsberg, 2005). Importantly, vaginal prolapse associated with estrus will typically resolve spontaneously when the dog transitions into diestrus, making conservative management viable in cases without complications (Root Kustritz, 2006).

Surgical management

Surgical intervention is indicated for Type III prolapse, cases with urethral obstruction, ischemic or necrotic tissue, failure of conservative management or owner preference for definitive treatment (Fossum, 2019; Sathiamoorthy et al., 2014; Patil & Pawshe, 2012).

Episiotomy and Manual Reduction: For severely prolapsed tissue, episiotomy may be required to facilitate manual reduction before definitive surgical repair (Wykes & Soderberg, 1983). Vaginoplasty (Resection of Prolapsed Tissue): Surgical resection of the hyperplastic vaginal fold is the definitive treatment for recurrent or severe cases (Bigliardi et al., 2004). The procedure involves circumferential resection of the edematous mucosal fold with primary mucosal apposition. Careful identification and protection of the urethral orifice is essential (Fossum, 2019).

Ovariohysterectomy (OHE): Ovariohysterectomy is the definitive preventive measure and eliminates the hormonal stimulus for recurrence (Johnston et al., 2001). It is strongly recommended for dogs not intended for breeding and may be performed concurrently with vaginoplasty or as the sole treatment in cases where spontaneous resolution is anticipated following hormone withdrawal (Root Kustritz, 2006).

Wound Care and Postoperative Management

Regardless of treatment approach, meticulous wound care is essential (Schaefer-Somi et al., 2003). The exposed vaginal mucosa should be kept clean and moist with regular lavage using sterile saline or dilute chlorhexidine solution. Topical antibiotics and lubricants prevent desiccation and secondary infection (Pretzer, 2008). An Elizabethan collar prevents self-trauma (Fossum, 2019).

Antibiotic therapy

Systemic antibiotic therapy is indicated when secondary bacterial infection is confirmed or when necrotic tissue is present (Bigliardi et al., 2004). Antibiotic selection should ideally be guided by culture and sensitivity results from swabs of the prolapsed tissue surface (Schaefer-Somi et al., 2003).

PROGNOSIS

The prognosis for vaginal prolapse in dogs is generally favorable when appropriate treatment is instituted promptly (Root Kustritz, 2006). Cases managed conservatively during estrus typically resolve without permanent sequelae as the dog enters diestrus (Johnston et al., 2001). However, recurrence in subsequent

estrous cycles is highly probable without ovariohysterectomy, occurring in the majority of affected dogs (Meyers-Wallen et al., 1986). Surgical resection combined with ovariohysterectomy provides the best long-term prognosis, with low recurrence rates (Fossum, 2019). Cases complicated by ischemic necrosis, urethral obstruction or sepsis carry a more guarded prognosis and require intensive management (Bigliardi et al., 2004). Reproductive prognosis is compromised in affected dogs. Natural mating is often impossible due to mechanical obstruction by the prolapsed tissue, and artificial insemination may be required (Linde-Forsberg, 2005). Whelping complications including dystocia are more frequent in affected dogs (Wykes & Soderberg, 1983). Given the hereditary predisposition, breeding of affected individuals is ethically questionable and professionally discouraged (Root Kustritz, 2006).

PREVENTION AND CONTROL

Prevention centers on elimination of the hormonal stimulus through early ovariohysterectomy in predisposed breeds (Johnston et al., 2001). Breeders of predisposed breeds should be counseled regarding the hereditary nature of the condition and the ethical implications of breeding affected individuals (Root Kustritz, 2006). Avoidance of exogenous estrogen administration in intact females of predisposed breeds is also advisable (Ettinger & Feldman, 2017).

CONCLUSION

Vaginal prolapse in dogs is a well-characterized reproductive disorder with a clear hormonal etiopathogenesis, strong breed predispositions, and a spectrum of clinical severity ranging from incidental subclinical findings to life-threatening emergencies. The English Bulldog, Boxer, Mastiff, Saint Bernard, Labrador Retriever and German Shepherd are among the breeds most frequently reported in the literature. Estrogen-driven mucosal edema during proestrus and estrus is the predominant causative mechanism, with genetic predisposition, brachycephalic conformation, large body type and parturition-related factors serving as significant contributing elements.

Diagnosis is primarily clinical, supported by vaginal cytology and vaginoscopy. Treatment ranges from conservative management with lubrication and hypertonic agents to surgical vaginoplasty with concurrent ovariohysterectomy for definitive prevention of recurrence. The prognosis is generally favorable with appropriate and timely intervention.

Future research priorities should include large scale epidemiological studies to establish true prevalence and breed specific incidence rates, genetic studies to identify specific hereditary markers and prospective clinical trials comparing conservative versus surgical management outcomes across different severity grades. In Nepal, published epidemiological data on canine vaginal prolapse are limited and existing knowledge is primarily based on hospital case records and clinical experience. Veterinary teaching and referral institutions such as Himalayan College of Agricultural Sciences and Technology report that the condition is encountered periodically in small animal practice, particularly in urban centers such as Kathmandu. However, no structured breed wise or population based epidemiological studies are currently available in Nepal and therefore no definitive conclusions regarding breed specific risk can be made for the country. This highlights a significant gap in veterinary reproductive epidemiology in Nepal and underscores the need for further systematic studies to better understand disease distribution and risk factors in the canine population.

REFERENCES

- Arlt, S. P., & Haimerl, P. (2016). Cystic ovaries and ovarian neoplasia in the female dog—A systematic review. *Reproduction in Domestic Animals*, 51(Suppl. 1), 3–11. <https://doi.org/10.1111/rda.12785>
- Bigliardi, E., Parmigiani, E., Cavirani, S., Luppi, A., Bonati, L., & Corradi, A. (2004). Ultrasonography and cystic hyperplasia pyometra complex in the bitch. *Reproduction in Domestic Animals*, 39(3), 136–140. <https://doi.org/10.1111/j.1439-0531.2004.00489.x>
- Blendinger, K., Bostedt, H., & Hoffmann, B. (1997). Hormonal state and the use of progestagens to induce a dioestrus-like state in bitches with hyperplasia of the vaginal mucous membrane. *Journal of Reproduction and Fertility Supplement*, 51, 271–276.
- Boothe, H. W. (2003). Surgical management of specific reproductive disorders. In D. Slatter (Ed.), *Textbook of small animal surgery* (3rd ed., pp. 1487–1496). Saunders.
- Brearley, M. J., & Brearley, J. C. (1988). Vaginal tumors in the bitch: A review of 40 cases. *Journal of Small Animal Practice*, 29(5), 255–266. <https://doi.org/10.1111/j.1748-5827.1988.tb02127.x>
- Concannon, P. W. (2011). Reproductive cycles of the domestic bitch. *Animal Reproduction Science*, 124(3–4), 200–210. <https://doi.org/10.1016/j.anireprosci.2010.08.028>
- Concannon, P. W., Hansel, W., & Visek, W. J. (1975). The ovarian cycle of the bitch: Plasma estrogen, LH and progesterone. *Biology of Reproduction*, 13(1), 112–121.
- Davidson, A. P. (2014). Reproductive disorders of the female dog and cat. In C. G. Couto & R. W. Nelson (Eds.), *Small animal internal medicine* (5th ed., pp. 924–951). Elsevier Mosby.
- Ettinger, S. J., & Feldman, E. C. (Eds.). (2017). *Textbook of veterinary internal medicine: Diseases of the dog and the cat* (8th ed., pp. 1655–1667). Elsevier.
- Feldman, E. C., & Nelson, R. W. (2004). *Canine and feline endocrinology and reproduction* (3rd ed., pp. 852–860). Saunders.
- Fossum, T. W. (Ed.). (2019). *Small animal surgery* (5th ed., pp. 834–838). Elsevier Mosby.
- Groppetti, D., Pecile, A., Arrighi, S., Di Giancamillo, A., & Cremonesi, F. (2010). Vaginal bacterial flora and cytology in proestrus bitches: Role on fertility. *Theriogenology*, 73(8), 1053–1060.
- Holt, P. E., & Sayle, B. (1981). Congenital vestibula-vaginal stenosis in the bitch. *Journal of Small Animal Practice*, 22(2), 67–75.

- Johnston, S. D. (1991). Disorders of the canine vagina and uterus. *Veterinary Clinics of North America: Small Animal Practice*, 21(3), 501–513.
- Johnston, S. D., Root Kustritz, M. V., & Olson, P. N. S. (2001). *Canine and feline theriogenology* (pp. 312–318). Saunders.
- Koirala, A., & Paudel, S. (2018). Prevalence of canine reproductive disorders in Kathmandu valley. *Journal of Agriculture and Forestry University*, 2, 101–107.
- Kumar, A., Singh, J., & Sharma, R. (2015). Clinical management of vaginal hyperplasia in dogs. *Indian Journal of Animal Reproduction*, 36(2), 70–72.
- Kydd, D. M., & Burnie, A. G. (1986). Vaginal neoplasia in the bitch: A review of forty clinical cases. *Journal of Small Animal Practice*, 27(5), 255–263.
- Linde-Forsberg, C. (2005). Abnormalities in pregnancy, parturition, and the periparturient period. In S. J. Ettinger & E. C. Feldman (Eds.), *Textbook of veterinary internal medicine* (6th ed., pp. 1655–1667). Elsevier Saunders.
- McEntee, K. (1990). *Reproductive pathology of domestic mammals* (pp. 210–215). Academic Press.
- Meyers-Wallen, V. N., Goldschmidt, M. H., & Flickinger, G. L. (1986). Prostaglandin F2 α treatment of canine pyometra. *Journal of the American Veterinary Medical Association*, 189(12), 1557–1561.
- Moxon, R., Whiteside, H., & England, G. C. W. (2016). Prevalence of ultrasound-determined cystic reproductive tract abnormalities. *Theriogenology*, 85(3), 450–454.
- Nelson, R. W., & Couto, C. G. (2019). *Small animal internal medicine* (6th ed., pp. 924–951). Elsevier.
- Nickel, R. F. (1998). Surgical treatment of vaginal prolapse and vaginal hyperplasia in dogs. In J. Bojrab (Ed.), *Current techniques in small animal surgery* (4th ed., pp. 541–546). Williams & Wilkins.
- Noakes, D. E., Parkinson, T. J., & England, G. C. W. (2009). *Veterinary reproduction and obstetrics* (9th ed., pp. 180–185). Saunders Elsevier.
- Nöthling, J. O., & Volkmann, D. H. (1993). Effect of addition of autologous prostatic fluid. *Journal of Reproduction and Fertility Supplement*, 47, 329–333.
- Patil, A. D., & Pawshe, C. H. (2012). Surgical correction of vaginal prolapse in a bitch. *Indian Journal of Veterinary Surgery*, 33(1), 56–57.
- Pretzer, S. D. (2008). Clinical presentation of canine pyometra and mucometra. *Theriogenology*, 70(3), 359–363.
- Romagnoli, S., & Concannon, P. W. (2003). Clinical use of progestins in bitches. *Theriogenology*, 59(1), 59–91.
- Root Kustritz, M. V. (2006). *Clinical canine and feline reproduction: Evidence-based answers* (pp. 420–425). Wiley-Blackwell.
- Sathiamoorthy, T., Raja, S., & Prasad, R. (2014). Vaginal prolapse in a bitch and its surgical management. *Indian Veterinary Journal*, 91(10), 78–79.
- Schaefer-Somi, S., Bär, S., & Aurich, C. (2003). Bacteriological status of canine semen. *Theriogenology*, 60(4), 687–694.
- Sontas, H. B., Ekici, H., & Kibar, M. (2003). Vaginal hyperplasia and prolapse in the bitch: A retrospective study of 15 cases. *Turkish Journal of Veterinary and Animal Sciences*, 27, 1143–1147.
- Sontas, H. B., Milani, C., Mollo, A., Romagnoli, S., & Stelletta, C. (2011). Vaginal fold prolapse in a bitch. *Reproduction in Domestic Animals*, 46(4), 740–743.
- Sontas, H. B., Stelletta, C., Milani, C., Mollo, A., & Romagnoli, S. (2011). Full-thickness resection of vaginal fold prolapse. *Journal of Small Animal Practice*, 52(2), 117–120.
- Theisen, S. K., Bhatt, M. V., & Riedesel, D. H. (1996). Recurrent vaginal prolapse in a dog. *Veterinary Medicine*, 91(2), 154–158.
- Watts, J. R., Wright, P. J., & Whithear, K. C. (1996). Uterine, cervical, and vaginal microflora. *Journal of Small Animal Practice*, 37(2), 54–60.
- Wykes, P. M., & Soderberg, S. F. (1983). Congenital abnormalities of the canine vagina and vulva. *Journal of the American Animal Hospital Association*, 19(7), 995–1000.

