

## CANINE PREGNANCY AND DYSTOCIA: CLINICAL MANAGEMENT

Philip Thomas  
Queensland Veterinary Specialists  
263 Appleby Rd  
Stafford Heights QLD 4053

### Introduction

Canine pregnancy is supported by progesterone from the ovarian corpora lutea which in turn are supported by pituitary LH and prolactin. Therefore, prostaglandins and dopaminergic agents are luteolytic. Several hematologic and biochemical changes are characteristic of pregnancy (Table 1). Definitive indicators of pregnancy are listed in Table 2). Days are listed from the LH peak.

*Table 1: Normal physiologic changes associated with pregnancy in the bitch. Parameters marked \* also change in diestrus.*

Parameter	Pregnancy-Associated Change
PCV	Falls from day 28 to 40% (d35) and <35% (d63)
Body Weight	Increases 20-50% in second half of pregnancy
Haemoglobin	Falls in second half of pregnancy
IgG	Falls to <500mg/dl after d21
Fibrinogen	Peaks at d30 then falls and peaks again at parturition
Prostacyclin	Increases
Acute Phase Proteins	Increase in first trimester
Insulin*	Decreases in response to rising progesterone
Glucose*	Reduced sensitivity to insulin after d 35
Creatinine	Increases
Total Protein	Increases
Calcium	Falls in last trimester
Thyroxin*	Increases
Relaxin	Rises from d20
Progesterone*	Rises from preceding estrus
Estrogens*	Peak in proestrus and late pregnancy
Growth Hormone*	Increases
Prolactin*	Rises from d35

Table 2: Indicators of pregnancy in the bitch and queen

Parameter	Bitch	Queen
Palpation	Vesicles palpable d21 Poor sensitivity and specificity	Vesicles palpable d21
Radiology	Fetal skeletons d45 Good sensitivity and specificity Litter size/fetal size ? Fetal death of >24 hours detectable	Fetal skeletons d38
Sonography	Vesicles detected d20 Fetal heart rates, litter size d25-28 Viability, uterine disease detectable	Vesicles detected d17
Acute Phase Proteins	Fibrinogen, Haptoglobin, C-reactive Protein Peak d20-30 False + common	
Relaxin	Detectable d20 In-clinic assay False + and - possible	

### Early embryonic death and abortion

Definition/overview: Early embryonic death and abortion accounts for all conceptus loss between conception and birth. Reliable data are not available but up to 25% of conceptions may not result in birth. Bacterial infection is overdiagnosed as a cause of conceptus loss when specific infectious causes are limited to *Brucella canis* and herpesvirus canis in the bitch.

Aetiology: Causes of abortion include: fetal defects; maternal environment (cardiac disease, reduced uterine blood flow, uterine disease, progesterone-associated diabetes mellitus, maternal stress in the queen, drugs, Table 3); trauma; infection (*Brucella canis* and other *Brucella* spp., herpesvirus canis, Minute Virus of Canines, Bluetongue virus, *Campylobacter jejuni*, *Salmonella* spp, *Listeria monocytogenes*, *Escherichia coli*, opportunistic bacteria, *Toxoplasma gondii*, *Mycoplasma* spp., and *Ureaplasma* spp.);

Pathophysiology: The pathogenesis of fetal loss in the bitch and queen is poorly documented except in the case of *B. canis* where bacterial infection causes placentitis.

Clinical presentation Signs depend on the stage of gestation at which fetal death occurs. Early loss may go unnoticed whereas loss in the last half of pregnancy may involve a discharge from the vagina. With later fetal loss, owners may report expulsion of fetal parts.

*Table 3: Drugs and chemicals which have been shown to cause pregnancy associated disease or teratogenicity in the bitch, queen, or woman. The time at which drugs should particularly be avoided is organogenesis, which is day 11–34 in the bitch and day 12–36 in the queen.*

Heavy Metals	lead, mercury
Corticosteroids	of any kind, at adequate dosage
Antimicrobials	tetracyclines, streptomycin, nitrofurantoin, chloramphenicol, griseofulvin, ketoconazole
Parasiticides	carbaryl, diazinon, dichlorvos
Steroids	progestins, estrogens, androgens, anabolics
Others	primidone, phenobarbitone, cimetidine, antiprostaglandins, prolactin antagonists

Diagnosis Diagnosis: Difficult and often unrewarding and diagnostic steps should include:

- History taking, including potential teratogens and any changes to the animal's environment such as the introduction of new animals.
- Examination of dam for systemic illness, including hematology, biochemistry, and urinalysis.
- Ultrasonography and radiography allow examination of the uterus.
- Culture of the vaginal discharge and abortus yields a result which is usually difficult to interpret.
- Histology of the abortus is usually more useful.

Management: Supportive therapy and symptomatic treatment of the dam which might include intravenous colloids and antibiotics if systemic disease is present. Rarely, pregnancy can continue if viable fetuses are present. If viable fetuses are not present, uterine contents may be expelled by the dam or may require expulsion with ecbolic agents or by ovariohysterectomy.

### **Pseudopregnancy**

Definition/overview: In the bitch and the queen, pseudopregnancy is a manifestation of normal physiology and not a disease. All bitches are pseudopregnant following estrus; some show signs (overt), others do not (covert). Queens have a period of pseudopregnancy following any breeding which stimulates ovulation but does not result in pregnancy.

Aetiology: Signs of overt pseudopregnancy in the bitch occur at the end of diestrus when progesterone levels fall and prolactin rises.

Clinical presentation: Females might show no clinical signs or show mammary gland development, abdominal enlargement, weight gain, and changes in behavior.

Differential diagnosis: Pregnancy.

Diagnosis: Physical findings and history provide the diagnosis. Pregnancy can be excluded by abdominal ultrasonography or radiography.

Management: Most bitches and all queens require no treatment. Reduce food and water intake with care. Avoid phenothiazines (e.g. acetylpromazine) which inhibit dopamine and promote prolactin secretion. Bromocriptine (20–30 µg/kg p/o q24h) will inhibit prolactin in the bitch Treatment with progestogens will lead to a resolution of clinical signs; however, when progestogens are discontinued, clinical signs will recur and the problem may worsen.

### **Uterine torsion**

Definition/overview: Torsion of the uterus is an uncommon condition in bitches. Typically it occurs in a near-term pregnancy and it is a life-threatening condition.

Pathophysiology: The gravid uterus rotates about its long axis from the focus of the uterine body (both horns) or base of one uterine horn (single horn) in the bitch

Clinical presentation: Affected animals might show abdominal pain, tenesmus, serosanguineous vaginal discharge, and dystocia usually at term, although the disease can occur in the last trimester.

Differential diagnosis Any systemic disease of late pregnancy.

Diagnosis Diagnosis is made on radiologic, sonographic, and clinical findings.

Management Surgical correction of the torsion and delivery of the neonates is indicated. Ovariohysterectomy may be necessary if ischemia has caused uterine necrosis.

### **Uterine rupture**

Definition/overview: Uterine rupture is a rare and usually occurs peri-partum.

Aetiology: In the bitch and queen, uterine rupture is usually associated with abdominal trauma in the last trimester or with obstructive dystocia. It can occur following administration of oxytocin or PG for dystocia, metritis, or pyometra or following untreated luminal disease.

Clinical presentation: Presentation is usually that of an abdominal crisis in a peri-parturient bitch. Free-floating mummified fetuses have been reported as incidental findings in otherwise healthy bitches and queens.

Diagnosis: Exploratory laparotomy will confirm the diagnosis.

Management: Exploratory laparotomy also allows removal of the conceptus, ovariohysterectomy, and local and systemic treatment for peritonitis.

### **Acromegaly and diabetes mellitus**

Definition/overview: In the bitch, progesterone is an agonist of growth hormone (GH) and an antagonist of insulin. Therefore, acromegaly and diabetes mellitus may be apparent during both diestrus and pregnancy.

**Aetiology:** Acromegaly is caused by an excess secretion of GH leading to overgrowth of soft and bony tissue. In the bitch it is caused by either exogenous progestogens used to prevent estrus or endogenous progesterone during diestrus. In the queen a functional adenoma of the pituitary pars distalis may be responsible. Excess GH also results in hyperglycemia, carbohydrate intolerance, and, eventually, insulin-resistant diabetes mellitus.

**Pathophysiology:** Increased progesterone concentrations during diestrus or pregnancy stimulate excess secretion of GH, which may inhibit postreceptor pathways or cause downregulation of insulin receptors in some animals leading to diabetes mellitus.

**Clinical presentation Acromegaly:** Interdental spaces are increased and there is usually excessive skin and wrinkling in combination with edema, particularly of the limbs, throat, and head. Animals with advanced disease may present with a hoarse voice as a result of the pharyngeal tissue hyperplasia.

**Management:** The conditions are transient and usually resolve once the source has been controlled.

**Normal pregnancy length**

Detecting full-term normal parturition must be based on several parameters (Tables 4 and 5).

*Table 4: Length of Pregnancy in the Bitch.*

58 to 71 days	from breeding date
63 to 70 days	from the LH peak or progesterone rise
54 to 60 days	from the first day of cytologic diestrus

*Table 5: Indicators of Impending Parturition in the Bitch.*

**Premonitory Signs**

Behavioural changes	0 to 7 days pre-partum
Nesting behavior intensifies	12–24 hours pre-partum
Mammary secretion	0 to 14 days pre-partum
Rectal temperature falls up to 1C	10–24 hours pre-partum
Progesterone falls	
<8-10nmol/L	0-12 hours pre-partum
Foetal Heart Rates on Sonography	
<180bpm	0-12 hours pre-partum

**Normal parturition in the bitch**

•**Stage I** labor (6–12 hours):

begins with uterine contractions of reducing interval  
ends with cervical dilatation: bitch restless, nervous,  
pant, paces, vomits, shivers.

•**Stage II** (fetal delivery) and **III** (placental delivery) labor (0–18 hours):

- first pup within one hour
- pups born at least every two hours
- placenta delivered usually 5–15 minutes after pup
- placenta delivery may be after all pups
- bitch may rest for 2 (or possibly 3) hours between pups
- delivery alternates between uterine horns
- posterior presentation is normal and occurs in 40% of pups.

## **Dystocia**

Definition/overview: Dystocia is an inability of the bitch to give birth.

Aetiology: Dystocia is either maternal or fetal in origin.

### Maternal origin

- Abdominal: age, abdominal hernia, diaphragmatic damage
- Pelvis: immaturity, fracture, neoplasia, degeneration, developmental anomaly, breed, diet.
- Uterus: trauma, rupture, torsion, developmental anomaly.
- Uterine “inertia”:
  - hormonal: abnormalities of estrogen, progesterone, relaxin, prolactin, PGs
  - metabolic: abnormalities of calcium, glucose
  - myometrial: single pup, age, toxic degeneration, overstretching.
- Cervix, vagina, vulva: immaturity, insufficient dilation, softening, fibrosis, disease, neoplasia, developmental anomaly.
- Psychologic.

### Fetal origin

- Fetal oversize: breed (Persian, Himalayan), small litter size, developmental anomaly (e.g. anasarca).
- Fetal position: ventral (dorsopubic), lateral (dorsolateral).
- Fetal posture:
  - posterior: hock flexion, hip flexion
  - anterior: head flexion, shoulder/elbow flexion.
- Fetal presentation: transverse/bicornual, simultaneous.
- Fetal abnormality: breed associated (Burmese, Scottish Fold, Manx), random.

Categories of bitches at high risk of dystocia: Brachycephalic breeds; toy breeds; bitches with single fetus; primiparous bitches >6 years old; history of abortion or dystocia; very large litters.

Pathophysiology: Since the physiology of parturition in the bitch and queen is poorly understood, the pathogenesis of dystocia is largely unknown but is dependent on the etiology.

Diagnosis: Diagnostic work up for dystocia might include:

- Physical examination.
- Reproductive tract examination: digital rectal, digital vaginal, abdominal palpation.
- Vaginal cytology.
- Hematology and biochemistry: total protein, PCV, BUN (urea), creatinine, glucose, calcium, electrolytes.
- Abdominal radiographs
- Abdominal ultrasonography.

Diagnostic indicators for intervention include:

- Gestation length:
  - >72 days from breeding
  - >70 days from the LH peak
  - >60 days from the first day of diestrus.
- Pelvic obstruction on vaginal examination.
- Abdominal contractions for 2 hours without birth.
- Failure to begin labor within 24 hours of temperature drop or progesterone baseline.
- Bitch/queen in pain, toxemic, or weak.
- No neonate within two hours of amniotic fluid delivery.
- More than 2 hours between pups
- Fetal heart rates indicative of fetal stress on sonography.
- Undelivered neonate in birth canal.
- Green, black or sanguinous vaginal discharge in absence of labor.

Management: Any pup in the vaginal vault should be delivered first. Fluid, electrolyte, calcium, and glucose imbalances must be addressed. Oxytocin is widely overused and often misused. Oxytocin excess is contraindicated and will cause tetanic and unproductive contractions of the uterus. Oxytocin treatment is contraindicated in obstructive dystocia.

Cesarian section is indicated if: fetal stress; failure to progress; fetal oversize (absolute or relative); fetal death *in utero*; obstructive dystocia, bitch unresponsive to oxytocin.

### **Postpartum metritis**

Definition/overview: Metritis is a postpartum disease involving the full thickness of the uterine wall, which occasionally becomes systemic. Postpartum bitches or queens, especially those who have had dystocia, retained placentas, or intervention in parturition, are prone to accumulation of fluid in the uterus.

Aetiology: Metritis may occur following lack of hygiene during parturition; following dystocia, retained placental membranes or fetuses; or following obstetric manipulation. Rarely, metritis may occur after mating, insemination, abortion, or normal parturition. Bacteria (coliforms, *Staphylococcus* spp., *Streptococcus* spp.) are implicated.

Pathophysiology: Infection of the uterine wall by bacteria which may be ascending or hematogenous. The disease is characterized by resultant endotoxemia.

Clinical presentation: Signs of endotoxemia; poor lactation and mothering; fetid red-brown, thin, seropurulent vaginal discharge in the postpartum female; uterine distension with placental tags and luminal fluid can be confirmed sonographically.

Diagnosis: Typical clinical presentation; transabdominal ultrasonography confirms uterine luminal fluid; vaginal cytology: red blood cells, degenerate neutrophils, bacteria, and debris; hematology/biochemistry consistent with endotoxemia.

Management: Medical management involves systemic treatment for endotoxemia including intravenous crystalloids and broad-spectrum antibiotics chosen with the aid of culture and sensitivity. Enrofloxacin is an appropriate choice in the absence of culture.

Uterine lavage with saline is indicated if the uterine lumen can be catheterized. Ecbolic agents such as oxytocin (0.5 IU/kg s/c) or dinoprost trimethamine (25–100 µg/kg s/c) can be used but with extreme caution to avoid uterine rupture and may not be indicated. The postpartum uterus shows reduced response to oxytocin. Hysterectomy is an option in females not intended for breeding.

### **Postpartum Mismatching (Inappropriate Maternal Behaviour)**

Definition/overview: Normal maternal behaviour post-partum is poorly understood and is probably driven by physical interactions at parturition and by pheromones. Failure of normal behaviour can endanger the health of neonates. Normal female behaviour includes attentiveness, lactation and nursing, protecting and cleaning neonates.

Aetiology and Pathophysiology: Shock, pain, nervousness, maternal inexperience, human interference and anaesthetic agents may precipitate abnormal maternal behaviour.

Clinical presentation: Signs include maternal nervousness, lack of feeding, cleaning, over-grooming or not grooming, and aggression towards neonates.

Diagnosis: typical clinical presentation

Management: Avoid Caesarian section especially in maiden bitches; sedation (Acetylpromazine at very low doses eg 0.01mg/kg); encourage nursing; reduce human interference.

### **Subinvolution of placental sites in the bitch**

Definition/overview: Prolonged serosanguineous vaginal discharge in the postpartum period in an otherwise healthy bitch suggests failure of complete involution of the uterus.

Aetiology: The cause of subinvolution of placental sites (SIPS) is unknown.

Pathophysiology: The postpartum uterus of the bitch has placental sites containing eosinophilic collagenous masses which contain trophoblast-like cells. These masses fail to be shed in SIPS and the syncytial trophoblast cells invade the deeper layers of the endometrium and myometrium causing bleeding and secondary luminal infections.

Clinical presentation: Up to 20% of bitches are affected, especially first litter bitches. The bitch is systemically normal and the pups unaffected. Bitches show a chronic nonresponsive sanguineous and occasionally purulent vaginal discharge up to 16 weeks postpartum.

Differential diagnosis: Normal lochia, coagulopathy, oestrus.

Diagnosis: Cytology of the discharge may show syncytial cells. The haemogram is normal, although slight anaemia may develop. Diagnosis is based on clinical findings, although endometrial biopsy is definitive.

Management: Many treatments have been proposed which have not been shown to be successful. These include progestins, estradiol, prostaglandins, androgens, uterine ecbolics, vitamin A, oxytocin, and surgical curettage. All cases will resolve spontaneously, although antibiotics may be necessary for superficial infection and care should be taken with bitches with coagulopathies. The prognosis is excellent for life and fertility. Affected bitches are no more susceptible to SIPS at subsequent litters.