

## DISORDERS OF THE PERIPARTURIENT PERIOD

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The periparturient period can be associated with high morbidity and even mortality for the dam and neonates. The periparturient period is defined here as the immediate prepartum period (1-2 weeks before parturition) and the 30-45 day post partum period before weaning. The diagnoses of periparturient problems first require their recognition and differentiation from normal situations; effective treatment depends on both timely diagnoses and intervention.

#### Normal Postpartum Events

Normally, dams stay very close to their offspring during the first 2 weeks postpartum, leaving the whelping/queening box briefly if at all to eat and eliminate. They are alert and content to remain with their offspring. Some protective dams may show aggression to housemate animals or even people with whom they are normally tolerant; such behavior tends to dissipate after 1-2 weeks of lactation. Lactation typically presents the greatest nutritional and caloric demand of the female's life. Weight loss and dehydration may occur and impact lactation if food and water are not made readily available; sometimes this entails leaving both in the nest box with a nervous dam. Partial anorexia can be exhibited during the last weeks of gestation and in the immediate postpartum period, but the appetite should return and increase as lactation progresses. Poor appetite during the last weeks of gestation can be due to displacement of the gastrointestinal tract by the gravid uterus. Partial anorexia early in the postpartum period can occur secondary to digestive upset following the consumption of numerous placentae. Diarrhea can occur secondary to increased rations and rich food (bacterial overgrowth secondary to carbohydrate malassimilation). Marked postpartum effluvium is normal in the bitch, usually occurring at 4-6 weeks after whelping, and sparing only the head. This is usually more marked than that which occurs in conjunction with the typical estrous cycle, and can be interpreted as pathologic by an owner, especially in conjunction with the weight loss typically associated with lactation.

The body temperature of the dam may be mildly elevated (<103.0 degrees F) in the immediate postpartum period, reflecting anticipated normal inflammation associated with parturition, but should return to normal levels within 24-48 hours. If a cesarean section took place, differentiating normal post surgical inflammation from fever associated with pathology may be difficult. The physical examination and a complete hemogram help the clinician differentiate between the two. Normal postpartum lochia is brick red in color, non odorous, and diminishes over several days to weeks (uterine involution and repair occur for up to 16 weeks in the bitch). The mammary glands should not be painful; rather they are symmetric and moderately firm without heat, erythema, or palpable firm masses. If expressed, normal milk is grey to white in color and of watery consistency.

#### Clinical Problems

##### Inappropriate Maternal Behavior

Appropriate maternal behavior is critical to neonatal survival and includes attentiveness, facilitation of nursing, retrieving neonates, grooming and protecting neonates. Although maternal behavior is instinctual, it can be negatively influenced by anesthetic drugs, pain, stress, and excessive human interference. Maternal bonding is a pheromone-mediated event initiated at parturition. Whelping and queening should take place in quiet, familial surroundings, with

minimal human interference, yet adequate supervision. Dams with good maternal instincts exhibit caution when entering or moving about the nest box so as not to traumatize neonates by stepping or lying on them. A guardrail along the inside of the whelping box prevents inadvertent smothering of canine neonates.

The neuroendocrine reflex regulating mammary gland myoepithelial cell contraction and subsequent milk ejection is mediated by oxytocin and activated by neonatal suckling. During stress, epinephrine induces vasoconstriction, blocking the entry of oxytocin into the mammary gland and preventing milk ejection. A nervous, agitated dam will likely have poor milk availability. Dopamine antagonist tranquilizers, with minimal prolactin interference (acepromazine 0.01-0.02 mg/kg) administered at the lowest effective dose to minimize neonatal sedation, can improve maternal behavior and milk ejection in nervous dams. Piling of littermates near their dam facilitates the maintenance of their adequate body temperature (neonates cannot thermoregulate/shiver for up to 4 weeks of age) and makes nursing readily available. Normal maternal behavior includes gentle retrieval of neonates who have become dispersed and isolated across the nest box. Grooming of the neonates immediately following parturition stimulates their cardiovascular and pulmonary function and removes amniotic fluids. Dams demonstrating little interest in resuscitating neonates can have poor maternal behavior throughout the postnatal period. Later, maternal grooming stimulates reflex neonatal urination and defecation and maintains the neonatal coat in a clean, dry state. Occasionally, excessive protective behavior or fear-induced maternal aggression can occur. Mild tranquilization of the dam with an anti-anxiety agent can help, but neonatal drug administration via the milk can be problematic. Benzodiazepines, GABA synergists, are reportedly superior to phenothiazines for fear-induced aggression (diazepam 0.55-2.2 mg/kg). The role of newer anti-anxiety pharmaceuticals in maternal aggression has not been described in a controlled setting.

#### Uterine Disorders

Complete or partial prolapse of the uterus is an uncommon postpartum condition in the bitch, occurring rarely in the queen. The diagnosis is based on palpation of a firm, tubular mass protruding from the vulva postpartum, and inability to identify the uterus with abdominal ultrasonography. Vaginal hyperplasia and prolapse, secondary to a hypersensitivity of focal (periurethral) vaginal mucosa to estrogen, can recur near parturition and should be ruled out by physical examination, vaginoscopy, or contrast radiography. The prolapsed uterine tissues are at risk for maceration and infection from exposure and contamination. The size of most bitches and queens precludes manual replacement; laparotomy and ovariohysterectomy are usually indicated.

Rupture of the uterus occurs most commonly with very large litters causing marked stretching and thinning of the uterine wall, especially in multiparous dams with dystocia. Immediate laparotomy for retrieval of fetuses and repair or removal of the uterus, as well as culture and lavage of the abdominal cavity, is indicated. The uterus should be carefully examined at any cesarean section for any areas with or prone to rupture. Peritonitis can result from an undetected uterine tear. A unilateral hysterectomy can be considered if the damaged area is limited and the dam valuable to a breeding program.

The persistence of serosanguinous to hemorrhagic vaginal discharge beyond 16 weeks post partum can indicate subinvolution of the placental sites of attachment (SIPS) in the bitch.

Histologically, fetal trophoblastic cells have persisted in the myometrium instead of degenerating, endometrial vessel thrombosis is lacking, and normal involution of the uterus is prevented. Normal interplacental regions exist. Eosinophilic masses of collagen and dilated endometrial glands protrude into the uterine lumen, oozing blood. The cause is unknown, blood loss is usually minimal, intrauterine infection not present, and fertility is unaffected. Treatment is generally not necessary, as recovery is spontaneous and symptoms mild. In the uncommon situation where vaginal bleeding from SIPS is copious enough to cause serious anemia, coagulopathies (likely defects in the intrinsic pathway or (thrombocytopenia / thrombocytopathies), trauma, neoplasia of the genitourinary tract, metritis and proestrus should be ruled out. Vaginal cytology, vaginoscopy, coagulation testing and abdominal ultrasound assist in the diagnosis. Treatment in these cases can be attempted with ergonovine (0.2 mg/15kg IM) administered once or twice. The benefit of therapeutic prostaglandins and/or oxytocin is questionable and not proven in any controlled study. The preventative value of oxytocin given in the immediate postpartum period is also unproven. Laparotomy and ovariohysterectomy are curative. Histologic examination of the uterus is indicated to confirm the diagnosis.

Acute infection of the postpartum endometrium should be suspected if lethargy, anorexia, decreased lactation and poor mothering occur accompanied by fever and malodorous vaginal discharge. Metritis is serious and sometimes preceded by dystocia, contaminated obstetrical manipulations, or retained fetuses and/or placentae. Hematologic and biochemical changes often suggest septicemia, systemic inflammation reaction and endotoxemia. Vaginal cytology shows a hemorrhagic to purulent septic discharge. Ultrasound of the abdomen allows evaluation of intrauterine contents and the uterine wall. Retained fetuses and placentae can also be identified with ultrasound. A guarded cranial vaginal culture is likely representative of intrauterine flora and should be submitted for both aerobic and anaerobic culture and sensitivities, and permits retrospective assessment of empirically selected antibiotic therapy. Bacterial ascension from the lower genitourinary tract is more common than hematogenous spread, and *Escherichia coli* the most common causative organism in both bitches and queens. Therapy consists of intravenous fluid and electrolyte support, appropriate bactericidal antibiotic administration and pharmacologic uterine evacuation, usually with prostaglandin f 2 alpha (in the United States) at a dose of 0.10-0.20 mg/kg q 12-24h for 3-5 days. An ovariohysterectomy may be indicated if the bitch's condition permits, and she is poorly responsive to medical management. Ergonovine (0.2 mg/15 kg given once IM) is also an effective ecbolic agent, but may cause rupture of a friable uterine wall. Synthetic prostaglandins offer more uterine specific therapy where available. Oxytocin is unlikely to promote effective uterine evacuation when administered >24-48 hours postpartum. Nurslings should be hand reared if the dam is seriously sick. Metritis can become chronic and cause infertility.

#### Metabolic Conditions

Gestational diabetes occurs infrequently in the bitch and queen, and is attributed to the anti-insulin effect of progesterone (mediated by increased levels of growth hormone) during the luteal phase. Polydipsia, polyuria and polyphagia with weight loss occur. Higher protein / lower carbohydrate diets may be helpful in the queen, while high fiber diets promote euglycemia in the bitch. Insulin may be indicated. Oversized fetuses can result from their increased production of insulin in response to maternal hyperglycemia, and may cause dystocia due to fetal-maternal mismatch.

Pregnancy toxemia in the bitch occurs as a result of altered carbohydrate metabolism in late gestation resulting in ketonuria without glycosuria or hyperglycemia. The most common cause is poor nutrition or anorexia during the last half of gestation. Hepatic lipidosis can occur. An improved plane of nutrition can resolve the condition in most cases, but termination of the pregnancy may be indicated in severe cases.

Puerperal tetany or eclampsia occurs most commonly during the first 4 weeks postpartum, but can occur in the last few weeks of gestation. The condition occurs in bitches more frequently than queens. Puerperal tetany can be life-threatening, caused by a depletion of ionized calcium in the extracellular compartment. Predisposing factors include improper perinatal nutrition, inappropriate calcium supplementation and heavy lactational demands. Small dams with large litters are at increased risk. Excessive prenatal calcium supplementation can lead to the development of puerperal tetany by promoting parathyroid gland atrophy and inhibiting parathyroid hormone release, thus interfering with the normal physiologic mechanisms to mobilize adequate calcium stores and utilize dietary calcium sources. Thyrocalcitonin secretion is stimulated. The use of a balanced growth (puppy/kitten) formula commercial feed without additional vitamin or mineral supplementation is optimal during the second half of gestation and throughout lactation. Supplementation with cottage cheese should also be avoided as it disrupts normal calcium-phosphorus-magnesium balance in the diet.

Metabolic conditions favoring protein binding of serum calcium can promote or exacerbate hypocalcemia, such as alkalosis resulting from prolonged hyperpnea during labor or dystocia. Hypoglycemia and hyperthermia can occur concurrently. Therapeutic intervention should be initiated immediately upon recognition of the clinical signs of tetany, without waiting for biochemical confirmation. The signs preceding the development of tonic clonic muscle contractions (progressing to seizures) include behavioral changes, salivation, facial pruritus, stiffness/limb pain, ataxia, hyperthermia and tachycardia. Immediate therapeutic intervention should be instituted with a slow intravenous infusion of 10% calcium gluconate (1-20 ml) given to effect. Cardiac monitoring for bradycardia and arrhythmias should accompany administration; their occurrence warrants temporary discontinuation of the infusion and a slower subsequent rate. Because cerebral edema can occur from uncontrolled seizures, diazepam (1-5 mg intravenously) or barbiturates can be used to control persistent seizures once eucalcemia is attained. Mannitol may be indicated for cerebral inflammation and swelling. Corticosteroids are undesirable because they promote calciuria, decrease intestinal calcium absorption and impair osteoclasia. Hypoglycemia should be corrected if present, and exogenous treatment for hyperthermia given if necessary. Once the immediate neurologic signs are controlled, a subcutaneous infusion of an equal volume of calcium gluconate, diluted 50% with saline, is given, repeated q 6-8 h until the dam is stable and able to take oral supplementation. Calcium gluconate or carbonate (10-30 mg/kg q 8 h) should be instituted. Each 500 mg calcium carbonate tablet (TUMS) supplies 200 mg calcium. Efforts to diminish lactational demands on the dam and improve her plane of nutrition are indicated. If response to therapy has been prompt, nursing can be gradually reinstated until the neonates can be safely weaned, usually at a slightly early age (3 weeks) and concurrent supplementation with commercial bitch/queen milk replacement is encouraged. The administration of calcium throughout lactation, but not

gestation, may be attempted in dams with a history of recurrent eclampsia (calcium carbonate 500-4000 mg/dam/day divided).

### Mammary Disorders

Agalactia is defined as a failure to provide milk to neonates. Primary agalactia, a lack of mammary development during gestation, results from a failure of milk production and is uncommon. A defect in the pituitary-ovarian-mammary gland axis is suspected. The use of progesterone compounds late in gestation can interfere with lactation. Secondary agalactia, a lack of milk availability due to a failure of ejection, is more common. Mammary development is marked, but milk cannot be readily expressed through the teat sphincter. The normal production of colostrum in the immediate post partum period should not be confused with agalactia. Agalactia can occur secondary to premature parturition, severe stress, malnutrition, debility, metritis, or mastitis. Treatment includes providing supplementation to the neonates while encouraging suckling to promote milk ejection, providing optimal levels of nutrition and adequate water to the dam, and resolution of any underlying disease. If detected early, milk let down can often be induced pharmacologically. Mini dose oxytocin, 0.25-1.0 units per injection, is given subcutaneously every 2 hours. Neonates are removed for 30 minutes post injection, and then encouraged to suckle, or gentle stripping of the glands performed. Metoclopramide, 0.1-0.2 mg/kg sc is given q 12h (dopamine antagonist) to promote milk production. Therapy is usually rewarding within 24 hours. Some authors advise a much higher dose of metoclopramide, but neurologic side effects become possible.

Galactostasis can cause engorgement and edema of the mammary gland with associated discomfort making further nursing unlikely, and becoming self perpetuating. Galactostasis occurs secondary to inverted or imperforate teats, failure to rotate nurslings, litter loss, an unusually small litter, or rarely with pseudocystitis.

Mastitis, septic inflammation of the mammary gland, can be acute and fulminate, or chronic and low grade, involving a single or multiple mammary glands. Coliforms, staphylococci, and streptococci are most commonly isolated in both bitches and queens. The source of bacteria is cutaneous, exogenous or hematogenous. Mild mammary discomfort and heat, galactostasis, cutaneous inflammation, and the presence of an intramammary mass are the earliest signs. Milk is commonly discolored red or brown due to the presence of red and white blood cells. Moderate cases exhibit pain, reluctance to nurse or lie down, anorexia and lethargy. Fever can be marked and may precede other clinical signs. Advanced cases can present in septic shock, with abscessed or necrotic glands. The diagnosis is based upon physical examination. Milk cell counts in bitches are not predictive of mastitis. Culture and sensitivity of milk collected aseptically from affected glands allows retrospective evaluation of antibiotic selection. Therapy should begin immediately, consisting of broad spectrum, bactericidal antimicrobials and gentle physical therapy. Analgesics may be indicated; neonates tolerate opioid analgesia in the dam. First generation cephalosporins (cephalexin 10-20 mg/kg q8-12 h) and beta lactamase resistant penicillins (Clavamox 14 mg/kg q 12 h) are advised and safe for the neonates. Antibiotic therapy may be warranted until weaning, and can preclude further nursing if sensitivities force the choice of a drug potentially toxic to neonates. Warm compresses or whirlpool therapy of the affected gland with gentle stripping of milk can potentially avert abscessation and rupture of the gland. Severe necrosis warrants mastectomy when the dam is stabilized, and aggressive wound

management. Antiprolactin therapy (cabergoline 1.5-5.0 ug/kg/day divided bid) may be indicated in severe cases, to reduce lactation. There is no evidence that nursing from affected glands is problematic for neonates, but they tend to avoid glands which are difficult to obtain milk from. The affected gland should be protected from trauma from nest box edges and neonatal claws. Mastitis can recur in subsequent lactations regardless of preventative measures taken. Early detection and treatment is optimal, rather than prophylactic antibiotics, which tend to favor resistant organisms.

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