Neonatology

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Neonatal survival

Average reported neonatal puppy and kitten mortality rates (greatest during the first week of life) vary, ranging from 9-26%.[1] Prudent veterinary intervention in the prenatal, parturient and postpartum periods can increase neonatal survival by controlling or eliminating factors contributing to puppy and kitten morbidity and mortality. Poor prepartum condition of the dam, dystocia, congenital malformations, genetic defects, injury, environmental exposure, malnutrition, parasitism and infectious disease all contribute to neonatal morbidity and mortality.

Neonates that fail to survive to weaning are most commonly stillborn or die within the first three days of life. Factors implicated in perinatal deaths include prematurity, in utero infection with viruses such as canine distemper, canine parvovirus, feline herpes, feline infectious peritonitis, panleukopenia, and feline leukemia virus, as well as anatomic birth defects, birth trauma/dystocia, low birth weight, inadequate nutrition, maternal neglect, and environmental stresses. Optimal husbandry impacts neonatal survival favorably by managing labor and delivery to reduce stillbirths, controlling parasitism and reducing infectious disease, preventing injury and environmental exposure, and optimizing nutrition of the dam and neonates. Proper genetic screening for selection of sires and dams minimizes inherited defects.[1-4] The neonatal period here is defined as the first 4-6 weeks of life.

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Neonatal physiology

Cardiovascular system

1. The neonate has a low pressure, low volume, low peripheral resistance circulatory system.
2. Higher heart rate, cardiac output, plasma volume and central venous pressure result.
3. Sympathetic innervation of the heart is incompletely developed, response to anticholinergics minimal.

4. Baroreceptor reflexes are present after 4 days of age, prior to that hypotension results from anoxia.[5]

   Clinical implications: One of the most important considerations of cardiovascular physiology in the neonate is that in the fetus and during the first 4 days of life bradycardia is not vagally mediated and is indicative of hypoxemia. Although during this time the neonate appears able to resist circulatory failure to a greater extent than the adult animal, it is far more appropriate to supplement oxygen than to administer parasympatholytic agents such as atropine; administration of which will only exacerbate cardiac hypoxemia via increasing oxygen demand in the face of hypoxemia. Additionally, due to incomplete maturity of the autonomic nervous system, the neonate is less able to respond to physiological stresses. Care should be given to maintain the neonate environment such that demands on the cardiovascular system are minimal.

Respiratory system

1. Stimulation of the genital or umbilical region of the neonate induces reflex respiration in the first three days after birth and may be clinically used to stimulate respiration in the immediate post partum period.

2. Normal respiratory rate in the neonate is low, ranging from 10 – 18 breaths per minute during the first week, despite a high metabolic oxygen demand.

3. The mechanisms that control respiratory function (carotid body chemoreceptors) in the newborn develop well before birth but require maturation in the post natal period.

4. The amount of work and pressure that is required by a neonate to maintain tidal breathing is increased as compared to that of the adult due to the high compliance of the chest wall.[5]

   Clinical implications. The neonate is very susceptible to the development of hypoxemia and/or jeopardized ventilation and gas exchange due to the immaturity of chemoreceptor responses to hypoxia and chest wall construction. Although there are adaptations present to help compensate for this physiological state, such as an extremely low circulatory failure pressure until four days of age, it is important to recognize that hypoxemia in the neonate may result in life threatening sequelae such as septic shock due to bacterial translocation despite a lack of mucosal lesions. It is vital that the environment be kept free of airway irritants and oxygenation is adequate.

Hematopoietic system

1. At birth the neonate red blood cell exhibits macrocytosis with corpuscle volume decreasing to that of the adult by four weeks of age as fetal red blood cells are replaced by adult red blood cells.

2. The hematocrit of the neonate may be as high as 60 per cent accounting for the red mucous membrane color often noted at birth. By three days of age red blood cell counts have decreased dramatically and continue to decrease for approximately three weeks. Adult levels for red blood cell count, hemoglobin, and hematocrit are generally not detected in most dogs until six months of age.

3. Neonatal isoerythrolysis is uncommon in the cat and rare in the dog. In the feline, the phenomenon occurs in association with a type A kitten born to a type B queen that has anti-A alloantibodies (agglutinating and hemolytic). White blood cell parameters in the canine and feline neonate are typically consistent with those of their adult counterparts. Lymphocytosis may also be noted in the normal neonate.[5]

   Clinical implications. During the neonatal period, as fetal red blood cells are replaced polychromasia and elevated reticulocyte counts may be noted. Care must be taken to ensure adequate ectoparasite control as iron demands are high; the presence of microcytosis is suggestive of iron deficiency anemia. Extramedullary hematopoeisis is commonly noted in the neonate liver.

Urinary system

1. In the canine, the neonatal kidney is morphologically and functionally immature; nephrogenesis continues for at least two weeks after birth.

2. The canine neonatal kidney is functionally characterized by a low glomerular filtration rate (GFR.), low renal plasma flow (RPF), low filtration fraction (FF), depressed reabsorption of amino acids and phosphate, exaggerated proximal tubule natriuresis and low concentrating ability.

3. Serum creatinine levels and blood urea nitrogen (BUN) concentrations are lower than in the adult animal; typically 0.4 mg/dl and 8 – 10 mg/dl respectively. Serum phosphorous concentrations are elevated; typically 9 mg/dl, due to skeletal growth.

4. At birth arterial pressure is low (50 – 60 mmHg). During renal maturation increased blood pressure and decreased vascular resistance result in an increase in GFR and RPF. In the

neonate, renal blood flow is directly correlated with arterial pressure and does not appear to be altered by inhibition of angiotensin until approximately 6 weeks of age.[5]

Clinical implications. A urine sample is easy to obtain from the neonate with gentle stimulation. The immature nature of the kidney alters interpretation of urinalysis. Low urine specific gravity (1.006 – 1.0017) is normal as is detection of protein, glucose and various amino acids due to the immaturity of the proximal tubule. By three weeks of age urine protein and glucose concentrations approach that of the adult dog and urine concentration is expected to compare to that of the adult dog by six to eight weeks of age.

As the neonatal kidney is less able to concentrate or dilute urine, renal blood flow parallels blood pressure, and there is altered sodium excretion by the proximal tubule. Fluid therapy should be administered with care to ensure adequate volume maintenance without over hydration or oncotic loading. Recommended daily fluid rates for the canine neonate range from 60–180 ml/kg/day. Caution must be exercised when administering renally excreted or metabolized antimicrobials (penicillin, ampicillin, cephalosporins, fluroquinolones, and aminoglycosides) to neonates. Generally, ß-Lactam antibiotics (penicillins, cephalosporins) are the antimicrobial drugs of choice, as although the half-life may be prolonged there is a large therapeutic margin. Ceftiofur, for example, administered at 2.5mg/kg SQ q 12hrs, maximum five days is an acceptable antimicrobial choice. Due to altered metabolism of nonsteroidal antiinflammatories the potential for renal toxicity from their use in the neonate is far greater than in the adult animal.[5]

Hepatobiliary system

1. During pregnancy, the maternal placenta supports many functions performed by the liver and biliary system in the adult animal. Prior to birth, the ductus venosis shunts blood through the liver, effectively bypassing the neonatal sinusoid. The canine neonatal liver and biliary system is functionally immature at birth.

2. There is a significant reduction in bile flow in the newborn puppy as compared to the adult dog, and a complete failure of secretin and glucagon to stimulate bile flow at 3-28 days of age. Despite a relative functional cholestasis in the neonate, serum bile acids may be used to detect hepatocirculatory abnormalities in puppies and kittens as young as four weeks. Alkaline

phosphatase (ALP) and gamma-glutamyltransferase (GGT) liver enzyme activities are markedly elevated in neonates less than two weeks old and moderately elevated after two weeks of age. Elevations in ALP and GGT enzyme activity have been attributed to placental, colostral, and intestinal activity. Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) are typically comparable to that of the adult. Alkaline phosphatase can be physiologically elevated during skeletal growth.

3. Postnatal hepatic microsomal enzyme activities at four weeks of age are 85% of that seen in an adult dog. Adult dog levels of microsomal enzyme activity are achieved by four and one-half months of age.[5]

Clinical implications. At birth, the neonate experiences functional cholestasis with altered liver enzyme serum biochemical profiles. Due to the absence of fully developed microsomal and P_{450} enzyme activity in the neonate until four to five months of age, caution must be exercised when prescribing medication that requires hepatic metabolism or excretion. The detection of serum increases in GGT and ALP in the newborn may be indicative of colostrum intake and potentially passive transfer.

Gastrointestinal system

1. Dentition eruption in the neonate first occurs at two to three weeks of age. All deciduous teeth should be present by 12-16 weeks of age.

2. At birth, the gastrointestinal tract is sterile and has a neutral gastric pH. It is characterized by a time-dependent increased permeability of the intestinal mucosa which decreases dramatically after ten hours. Normal nursing pup feces are semiformed and tan in color (acholic). GI motility prior to 30-40 days of life is dependent upon pressure gradients rather than electrical intestinal motility.

3. Body temperature is known to have a dramatic effect on gastrointestinal movement in the neonate. At rectal temperatures below 94 °F, ileus develops. As ileus progresses, the willingness to nurse decreases and the necessity for tube feeding puppies increases. Inherent to the tube feeding process is the risk for aspiration and subsequent development of pneumonia.[5]

Clinical implications. Care should be taken to ensure adequate environmental conditions to maintain normal body temperature in neonates to minimize gastrointestinal ileus. Due to altered absorption from increased gastrointestinal permeability and neutral gastric pH in the immediate post natal period, care must be taken if administering oral drug therapy. Diarrhea can result from overeating, and is then complicated by subsequent bacterial overgrowth.

Immune system

1. Five to ten percent of canine neonatal serum antibodies are derived from trans-placental transfer. At birth, the canine neonate is antibody deficient and immunologically incompetent. The acquisition of passive immunity requires adequate ingestion and absorption of colostrum during the first 24 hours of life. Gastrointestinal absorption of colostral antibodies decreases markedly after 12 hours.

2. Providing adequate ingestion of quality colostrum, the puppy is protected by maternally derived immunoglobulins during the neonatal period. Puppies are capable of producing challenge specific antibodies within two weeks and with repeated challenge can produce a secondary immune response at 40 days. However, even by 40 days of age, T cell mitogenesis and differentiation, and phagocytic cell function systems may not be fully mature.[5]

Clinical implications. Incompletely developed immune systems and inadequate thermoregulation during the first days of life make neonates vulnerable to systemic infection (bacterial and viral). Adequate ingestion of colostrum must occur promptly post partum for puppies to acquire passive immunity. The transmission of protective immunity (placental or colostral antibodies) between a bitch and her puppies depends upon the prior existence of adequate serum maternal antibodies. When colostral intake is not possible or is of questionable quality, pooled adult dog serum (20-150 ml/kg SC divided) may be administered to elevate serum immunoglobulin concentrations in the puppy.[5-7]

Neurologic system

1. The neonatal puppy's main activities during the first two weeks of life are sleeping and nursing. The rooting reflex orients the neonate to its source of food, the dam. Vestibular function is present at birth and is important for positioning during nursing. Muscular coordination however is absent. Initial movements are characterized by swimming-like movements of the limbs, while sliding along

on the ventral abdomen and thorax. The ability to raise the head is present at birth in puppies and the head may be used initially for righting reflex. An upright posture in puppies cannot be maintained until ten to 14 days.

2. The EEG of the neonatal puppy initially is similar during periods of sleep and waking.

3. At birth the body posture is primarily one of flexion. If suspended by the head, flexor hypertonicity is present. At four to five days in puppies, the flexor hypertonicity is replaced by extension until three to four weeks of age when the puppies will begin to struggle to escape when held in suspension.

4. The nociceptive threshold is much lower than in adults. This may be due to a lack of some of the descending inhibitory mechanisms found in older animals. The coordination of motor responses to noxious stimuli is not well-developed and the animal may have much wider receptive fields to noxious events. Neurotransmitters may not have reached full function.[8]

Clinical implications. Although the nervous system of the neonate is immature there is no doubt that nociceptive pathways are present and that pain is perceived by the neonate subjected to noxious stimuli. Drugs which might be effective in adults may not be as effective in neonates. Procedures carried out on neonates with insufficient pain control produce greater stress responses than those where analgesia has been provided. A local anesthetic (lidocaine, dose extrapolated from humans) can be used and is very effective; The dose requirement is lower because of the immaturity of the nerves but the neonate does not appear to be at any greater risk of toxic side effects with a single dose of lidocaine. Bupivicaine is not advised in the neonate due to the risk of cardiotoxicity with overdosage.

The pharmacokinetics of the opioid analgesics are different in the neonate versus the adult. Lower doses of these drugs are required for analgesia at one day of age compared with 34 days (three to four-fold differences).

Metabolism

1. The normal birth weight of the puppy is breed dependent; generally, 500 gm ± 150 gm for a medium breed dog. Birth weights lower than 300 gm in the medium size dog are associated with an increased risk of neonatal mortality. Increased mortality in low birth weight puppies is most

likely associated with negative effects of chilling (higher body surface area: mass) and the ability to nurse and maintain glucose concentrations. Generally, there is a similar pattern of growth amongst different breeds of dogs; the most rapid weight gain occurring during the first 12 weeks. Puppies should gain on average 10% of their body weight each day for the first few weeks of life.

2. Unlike their homeothermic adult counterparts, neonates are poikilothermic. However, they have well-developed behavioral heat-seeking responses which enable them to maintain a stable rectal temperature providing sources of heat are available. Shivering and vasoconstrictive reflexes are not functional in the newborn. Physiological responses noted during hypothermia include bradycardia, cardiovascular failure, neuronal injury, and ileus. Normal rectal temperatures in the puppy are 95 – 99 °F (week one), 97 – 100 °F (weeks two and three) and by weaning rectal temperatures approach that of the adult.

3. At birth the neonate must transition from placental support to endogenous food stores for glucose production. During the first three to 24 hours after birth, hepatic glycogen stores decline by more than 50% and there is a shift from glycogenolysis to a mixture of glycogenolysis and gluconeogenesis. For maintenance of blood glucose concentrations, regular feeding is required. In addition to regular nursing, the dam's nutritional state must be adequate to provide for the needs of her puppies.[5]

Clinical implications. The neonate is susceptible to a wide variety of toxic, environmental, infectious and congenital insults; however, the ability for them to respond is limited. One of the first signs of illness in both the kitten and puppy is a failure to gain weight. This finding is often noted well before any other clinical signs of disease are present. Twice daily weighing of neonates during the first week(s) of life dramatically facilitates early detection of illness, ensures adequate intervention in a timely manner to prevent poor weight gain and positively impacts neonatal survival.[1]

Neonatal/pediatric conundrums

Fading Puppies

A fading puppy commonly dies following the onset of rapidly progressive, vague signs of illness. Premortem diagnosis is challenging. Immediate necropsy of a neonate dying without obvious cause is

warranted to provide proper veterinary care of the littermates. Clients should be advised to refrigerate (not freeze) deceased neonates and present them promptly for evaluation.

Neonatal bacterial peritonitis with septicemia can cause rapid deterioration of the puppy resulting in death if not recognized and treated promptly. Factors shown to predispose a puppy to septicemia include endometritis in the bitch, a prolonged (often not recognized or reported) delivery/dystocia, feeding of replacement formulas, the use of ampicillin, stress, low birth weight (< 350 gms), and chilling with body temperature <35.5 °C. The umbilicus of neonates should be treated with tincture of iodine immediately after birth to reduce contamination and prevent ascent of environmental bacteria into the peritoneal cavity (omphalitis-peritonitis).

The bacterial organisms most frequently associated with septicemia are *E. coli*, *Streptococci*, *Staphylococci*, and *Klebsiella* spp. Commonly, a decrease in weight gain, failure to suckle, hematuria, persistent diarrhea, unusual vocalization, abdominal distention and pain, and sloughing of the extremities indicate septicemia may be present.

Prompt therapy with broad spectrum, bactericidal antibiotics, optimal nutrition via supported nursing, tube feeding or bottle-feeding, maintenance of body temperature, and appropriate fluid replacement are indicated. The third generation antibiotic, ceftiofur sodium, is an appropriate choice for neonatal septicemia as it alters normal intestinal flora minimally and is usually effective against the causative organisms. The prognosis for septicemic neonates is poor. Failure to respond to antibiotic therapy should prompt consideration of canine herpes virus infection.

Canine herpesvirus (CHV) is a widely recognized and commonly blamed cause of fading puppy syndrome. Premortem and postmortem diagnosis of CHV infection in neonates can be challenging. Typical necropsy findings include multifocal petechial renal hemorrhages. Confoundingly, these can be also be present with bacterial septicemia. Intranuclear inclusion bodies can be difficult to find. Diagnosis by virus isolation or CHV-specific PCR is confirmatory. Treatment has been reported to be unrewarding and recovery is rare. Recovery has been reported to result in residual cardiac and neurologic damage. Treatment with immune serum from affected dams is reported to be ineffective in infected puppies. One case report of successful treatment with the antiviral drug, acyclovir exists. Successful vaccine

development has been hampered by the poor immunogenicity of other herpesviral vaccines developed for other species, as with feline and bovine rhinotracheitis. Neonates of a naïve bitch exposed to CHV during the last two to three weeks of gestation or the first three weeks postpartum are at risk.[9,10]

Acyclovir is an antiviral agent with activity against a variety of viruses including herpes simplex. Acylcovir is preferentially taken up by susceptible viruses and converted into the active triphosphate form, which inhibits viral DNA replication. Acyclovir is poorly absorbed after oral administration and is primarily metabolized by the liver. Acyclovir can increase the toxicity of nephrotoxic drugs. The half-life in humans is approximately three hours. Its use in veterinary medicine is not well established and it should be used with caution and only in situations where indicated. The safety and effectiveness in humans less than two weeks of age is not established. The dose is extrapolated from that for humans.[10]

Juvenile cellulitis

Juvenile cellulitis (puppy strangles) is a progressive, granulomatous, pustular disorder of puppies, most commonly occurring in dogs younger than four months of age, but it is occasionally reported in dogs up to four years of age. The eyelids, pinnae, lips, chin, muzzle, paws, abdomen, thorax, vulva, prepuce and anus can be affected with lesions that fistulate, drain and crust. Lymphadenomegaly, most commonly mandibular and superficial cervical, can be distant from the affected skin sites and is often painful. Pustules and lymph nodes are usually sterile when cultured. Superficial cutaneous flora can be cultured from open, draining lesions. Pyrexia, anorexia, sterile suppurative painful arthritis and an inflammatory hemogram can occur. The diagnosis is confirmed by histopathologic evaluation but is commonly made on the basis of clinical appearance. The predominant inflammatory cell in juvenile cellulitis, characterized by light and electron microscopy and immunohistochemical staining, is an epithelioid macrophage Juvenile cellulitis requires aggressive immunosuppressive therapy early in the course of the disease for resolution and to avoid the sequellae of cicatricial lesions. Traditionally, puppies have been placed on immunosuppressive doses of prednisone (2.2 mg/kg/day), causing concerns with immunization efforts. Griseofulvin therapy offers an apparently effective treatment without the side effects associated with corticosteroid administration, enabling discontinuation of corticosteroids sooner in the course of the

disease. It has been reported to be effective as sole immunomodulatory therapy (14.2 to 34 mg/kg PO Q 12 h). Griseofulvin is postulated to induce down regulatory signals within the lesions. The use of griseofulvin as sole therapy could be attempted in early cases. Vaccination of puppies undergoing immunosuppressive therapy is not advised and they must be strictly isolated from sources of infectious disease.[11,12]

Bacterial overgrowth syndrome-associated diarrhea

Pediatric dogs and cats are often presented to the veterinarian for signs referable to the abdominal cavity. Dietary indiscretions, parasitism and infectious disease (primarily viral, less commonly bacterial) account for most of these presentations. Congenital and developmental disorders should also be considered.

Symbiotic colonic bacteria assist digestion. The upper GI tract was once believed to be sterile, but normal colonization of the duodenum, jejunum, and ileum is now appreciated. Bacterial overgrowth syndrome (BOS) occurs when the normally low bacterial colonization in the upper GI tract significantly increases. Neonates are particularly at risk for developing BOS. Mucosal injury resulting from a minor viral or bacterial gastroenteritis can induce BOS in these individuals if a proper post infectious dietary regimen is not followed.

A particular bacterial pathogen has never been implicated; instead, abnormally large numbers of normal or pathological flora appear to cause BOS. Under normal conditions, gram-positive bacteria and fungi colonize the duodenum and jejunum in quantities less than 1×10^5 organisms per milliliter of fluid. Aerobic and anaerobic bacteria colonize the ileum in quantities less than 1×10^8 organisms per milliliter of fluid. This is in sharp contrast to the 1×10^{11} organisms per milliliter of fluid that colonize the colon. Studies of duodenal aspirates have not identified any particular bacteria as a cause of BOS; however, 1×10^5 organisms per milliliter of aspirate fluid is diagnostic for BOS. Usually, abnormally large numbers of anaerobic bacteria and normal florae grow from cultured fluid of patients with BOS.

The following are protective factors that stabilize the number and type of bacteria that colonize the upper GI tract. Abnormalities in these mechanisms put a patient at risk for bacterial overgrowth.

1. Two coordinated motor phenomena produce the continuous propulsive peristaltic action of the upper GI tract. Both the migrating motor complex and the migrating action potential complex clear the upper intestine of unwanted bacteria and undigested substances. Desynchronization of these complexes results in diarrhea and weight loss in animal models. Neonates lack propulsive peristaltic action. Gut motility in neonates results from aboral pressure.

2. Gastric acid normally reduces the proximal small intestine bacteria populations, particularly anaerobic bacteria. The bowel mucosa integrity and mucin layer protect the gut from bacteria. Neonates have reduced gastric acidity.

3. Malabsorption of bile acids, fats, carbohydrates, proteins, and vitamins causes many of the symptoms of diarrhea and weight loss associated with BOS. Anaerobes and *Bacteroides fragilis* actively deconjugate bile acids, thereby preventing proper bile acid function and enterohepatic circulation. Fatty acid absorption is reduced because deconjugated bile acids cannot help micelle formation. Deconjugated bile acids directly inhibit carbohydrate transporters. These unabsorbed sugars ferment into organic acids because of the intestinal flora, which reduces the intraluminal pH and produces osmotic diarrhea. The unconjugated bile acids also damage intestinal enterocytes and induce water secretion by the colonic mucosa.

4. Fat, protein, carbohydrate, and vitamin malabsorption result from poor enterocyte function and bacterial transformation of nutrients into nonabsorbable and toxic metabolites. Toxic metabolites damage the intestinal mucosa. Malabsorption and enterocyte dysfunction further degrade the health of the gut by reducing local and systemic nutrition delivery.

Treatment of BOS is aimed at reducing the damage caused by malabsorption and restoring nutritional health and normal gut flora. Prompt recognition and treatment can prevent the development of

severe malnutrition. The antimicrobials of choice for therapy of BOS-associated diarrhea are ampicillin or amoxicillin in the pediatric patient (due to the neurotoxicity associated with metronidazole overdosage).[14]

Anasarca

Anasarca, a lethal congenital edema, can occur with or without concurrent cardiovascular abnormalities. Generalized subcutaneous edema, with intrathoracic and intraperitoneal fluid accumulation is present. Congenital hereditary lymphedema causes edema of the extremities and sometimes head, and is associated with morphologic lymphatic abnormalities. Prepartum ultrasonographic evaluation of the fetuses can be used to screen for this disorder. Dystocia can result due to fetal oversize. Anasarca is a problem common in Bulldogs, but recognized in other breeds as well (Labrador retriever). It is suspected to have a heritable component. Its exact pathophysiology in the dog is not understood. The genetics are not known; anasarca is thought to be inherited as an autosomal dominant trait. There are multiple anecdotal remedies, none proven or reported in the scientific literature. It is debated and discussed on theriogenology list serves and on the layman's internet exhaustively. As well as causing dystocia, anasarca usually results in stillborn puppies or puppies needing to be euthanized. Some veterinarians promote various therapies, usually doomed to failure. Diuretic therapy of affected neonates can sometimes cause slight normalization, but euthanasia is usually indicated if the neonate is not stillborn. Environmental, dietary, and pharmacologic contributory factors are not scientifically defined. Anasarca has been recognized for many years, yet its incidence remains unchanged. An attempt to recognize the presence of anasarca prepartum with ultrasonography should be made in bitches with a history of affected puppies or in breeds with high incidence, due to the higher incidence of dystocia associated with the syndrome.

"Swimmer" puppies

Swimmer puppies fail to develop normal ambulation at ten to 14 days of life, moving instead by paddling their limbs laterally and caudally. Compression and deformation of the sternum and thorax occur concurrently. Obese puppies from small litters, commonly raised on relatively slippery surfaces are predisposed. Treatment should be instituted immediately upon diagnosis, consisting of caloric restriction, physical therapy, and improved traction in the nest box. If diagnosed early (three to five weeks of age) the condition is reversible and does not require binding of puppies.

Puppy vaginitis

Puppy vaginitis is characterized by an apparently healthy female puppy presented with mucoid vulvar discharge that is usually white to yellow, and sometimes copious. The discharge can be accompanied by mild perivulvar dermatitis. The puppy is not typically attentive to the discharge, and there is not any associated change in urinary behavior (dysuria or polakiura). Clients often have a difficult time deciding if a puppy has normal urinary behavior or not. The age of onset ranges from six weeks to puberty, the duration is from days to months, and the disorder is often intermittent.

Cytologic examination of the discharge finds suppurative inflammation. Vaginal cultures (aerobic) generally fail to grow anything but normal flora in small numbers, similar to unaffected littermates. A urinalysis, acquired by cystocentesis, is characteristically normal (a decreased urine specific gravity is typical for young dogs lacking adult concentrating abilities), and the urine culture negative. The clinician needs to perform enough diagnostics to rule out more significant causes of vulvar discharge and feel comfortable with the diagnosis of benign puppy vaginitis.

The specific etiology of puppy vaginitis is unknown. An imbalance of juvenile vaginal glandular epithelium is postulated. The condition is reported in the literature to resolve both with puberty and with ovariohysterectomy, two very different events endocrinologically, therefore neither likely to truly cause resolution. Puppy vaginitis diminishes with maturity. The term "puppy vaginitis" is a misnomer, as it is asymptomatic and not indicative of inflammation. Important rule-outs (some of which are associated with inflammation) include urinary tract infection, urinary incontinence with associated mucosal scalding, the onset of the initial estrous cycle, vaginal foreign bodies (i.e. foxtails) and urogenital anatomic anomalies (ectopia, disorders of sexual differentiation, significant strictures). Cleansing the perivulvar area with a gentle solution (non-alcoholic otic preparations or "baby wipes"), benign neglect and tincture of time are advised.[14,15]

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