

COMMON DISORDERS OF THE SMALL ANIMAL NEONATE

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INTRODUCTION

Mortality of puppies and kittens is high, even in well managed colonies. The rate of stillbirths averages 12.9% with a range of 4.7 - 22.1% in cats.¹⁻⁵ Greatest incidence of mortality occurs within the first week of life⁶ and averages 27.3% in kittens^{3-5,7} and 26.0% in puppies.^{8,9} Overall mortality by weaning at 8-12 weeks of age averages 23.2% in kittens^{1-3,5,7} and 18.7% in puppies.¹⁰

Neonatal mortality in puppies is associated with age of the dam. The number of pups weaned per litter decreases and puppy mortality increases with increasing age of the dam.¹⁰ In cats, increased kitten mortality has been associated with obesity of the queen, increasing parity of the queen, and litter sizes of one or ≥ 7 kittens.^{4,10} Mortality is increased in Persians, Manx, and Himalayans compared to short-haired cat breeds.³

It is recommended that all dead puppies and kittens be submitted for necropsy. If a large number of animals is affected, sacrifice of a failing animal may be indicated.¹¹ Dead animals should be stored in the refrigerator, not frozen, to prevent artifactual changes in tissue. Although necropsy is expensive and is reported to identify cause of death in only about 1/3 of submissions,^{11,12} diagnosis of a specific condition may enhance the veterinarian's ability to treat the remaining littermates or control a colony-wide problem.

Trauma and maternal neglect are common causes of neonatal loss. The rate of puppy deaths from trauma was 37% in one colony described.¹³ Cannibalism is not uncommon in cat colonies; 25 of 107 live-born kittens (23%) were cannibalized within 3 days of life in one colony.⁴ Anecdotal reports suggest that hypocalcemia may cause bitches to savage pups. Other traumatic insults to neonates include dystocia, suckling by littermates, and overzealous cleaning by the dam.^{11,14} Maternal neglect may occur in high-strung dams or if the neonate is sick or chilled.¹¹ If a bitch or queen repeatedly rejects a neonate, even after re-warming and physical examination to rule-out overt abnormalities, it may be best to remove that animal for hand-raising.

Behavior that suggests illness of a pediatric patient includes separation from the dam, indicating either fever or culling by the dam, crying for more than 20 minutes at a time, and decreased activity.¹⁵ On physical examination, decreased muscle tone, pale to cyanotic mucous membranes, lack of normal bowel sounds, panting or labored breathing, rough hair coat, and diarrhea may be evident.^{15,16} For puppies and kittens that present having very recently died or that arrest during examination, the followings steps should be taken:¹⁷

- A = airway - Aspirate secretions from the oral cavity and respiratory tract, intubate if possible.
- B = breathing - Provide 100% oxygen via endotracheal tube or mask, or room air with an Ambu bag.
- C = circulation - Direct chest compression should approximate normal heart rate. If intrathoracic cardiac compression is attempted, it should be instituted within 2 minutes of starting cardiopulmonary resuscitation. Other techniques that may be used include intermittent abdominal compression, epinephrine (0.04 - 0.4 mg/kg intratracheal or intracardiac), or stimulation of the Jen Chung acupuncture site, in which a 25 ga needle is inserted into the nasal philtrum at the base of the nares and twisted once to reach the periosteum.¹⁷

GENERAL PROBLEMS

LOW BIRTH WEIGHT / POOR GROWTH RATE

The puppy or kitten should have been weighed at birth, and should be weighed daily thereafter. Kittens should weigh about 3.5 oz (100 gm) at birth.¹⁷ Puppy birth weight varies by breed; estimates for various breeds are 4.2 oz (120 gm) for Pomeranians, 9 oz (250 gm) for Beagles, 17 oz (490 gm) for Greyhounds, and 22 oz (625 gm) for Great Danes.¹⁴ Low birth weight is correlated with poor survivability in puppies and kittens. Birth weight is not influenced by sex of the neonate, and is more likely an indicator of inadequate intrauterine nutrition or congenital abnormalities than of prematurity.¹¹ In one cat colony, 60% of 192 kittens with low birth weight died by weaning, while only 32% of normal weight kittens died by weaning.⁴ Fifty-four percent of stillborn kittens and 73% of kittens that died by 3 days of

age had low birth weight in that colony.⁴ This suggests that a guarded prognosis should be given to neonates with lower than average weight at birth.

Slight weight loss may occur in the first 24 hours of life. Puppies that lose greater than 10% of birth weight in the first day of life have a poor prognosis.⁶ After that, the puppy or kitten should gain weight daily, doubling their birth weight by 7-10 days of age.¹⁸⁻²⁰ Kittens should gain 1.8 - 3.5 oz (50-100 gm) weekly.²¹ Puppies should gain 0.05 - 0.1 oz (1-2 gm) per pound (2-4 gm/kg) of anticipated adult weight daily, about a 10% increase per day.^{19,21}

Pediatric animals with poor growth rate should be assessed for adequacy of nursing. Ensure that they are not being excluded from nursing by competition with stronger littermates, that the nipples are not hyperkeratotic, that mastitis is not present, and that there are no congenital abnormalities in the patient precluding effective suckling, such as cleft palate.¹⁴ During and after weaning, the animal should be fed a high quality, energy-dense food. Animals with slow growth and poor body condition may suffer from disorders such as a portosystemic shunt, renal failure, megaesophagus, exocrine pancreatic insufficiency, or cardiac disease. Animals with slow growth and normal body condition may suffer from disorders such as hypothyroidism, diabetes mellitus, or adrenal disease.²²

ANOXIA

Length of the anoxic episode is not associated with outcome in neonates.⁴ Anoxia is associated with bradycardia and hypoventilation in very young animals, and often is accompanied by hypothermia and a subsequent lower oxygen demand.^{6,8}

HYPOTHERMIA

Rectal temperature varies with age and environment. Puppies and kittens may lose as much as 8.0°F (3.8°C) in the first day of life, even in a normal environment.⁸ In the first week of life, brown fat is the main source of thermogenesis; after the first week, the shivering reflex permits some thermoregulation by the pediatric patient itself.²³ Normal rectal temperature is 96.0 +/- 1.5°F (35.6 +/- 0.7°C) in the first week of life, 98.6 - 100.0°F (37.0 - 38.2°C) in the second and third weeks of life, and gradually rises to adult levels by the age of seven weeks.^{5,8,14}

Re-warming of hypothermic puppies and kittens should be gradual, taking 30 minutes to 2 hours.^{9,24} The neonate should be turned and rectal temperature monitored frequently. Do not warm to a rectal temperature greater than 101°F (36.3°C) so as not to cause dehydration.²⁴ Re-warming can be accomplished with careful use of surface heat, such as circulating hot water pads and hot water bottles, warmed inspired air or warmed fluids administered intravenously or intraosseously.^{16,24}

Body temperature of less than 94-95°F (34.5 - 35.0°C) is associated with failure to suckle and ileus, visceral paralysis.^{8,9,12,16} Do not give milk products orally until body temperature is returned to normal since hypothermic animals are incapable of digesting milk and predisposed to aspiration if the stomach is distended.^{9,12} Calories can be provided by parenteral or oral administration of glucose-rich solutions, which do not require normal peristalsis for absorption.

DEHYDRATION / FLUID THERAPY

Dehydration is more difficult to assess in pediatric patients than in adult animals. Skin turgor may be used as a measure of dehydration; dehydrated pediatric animals exhibit more wrinkling and less turgor of the skin, and deepening of the red color of the ventral abdomen and muzzle.¹⁴ Other signs of dehydration include dryness of the eyes and oral mucous membranes, and visible yellow color of the normally dilute urine.²⁵

Pediatric animals require more water relative to body mass compared to adults and suffer relatively greater surface losses, with a fluid requirement of about 200 ml/kg day.^{5,26} Pediatric animals have decreased cardiac capacity and underdeveloped renal function, and so cannot tolerate large volume replacement.²⁶

Fluids can be administered via intraperitoneal (IP), subcutaneous (SQ), intravenous (IV), or intraosseous (IO) routes. Absorption of fluids from the IP space is slow, especially in hypovolemic animals, and fluids must be administered with strict aseptic technique.²³ Similarly, SQ administration of fluids is made difficult by the limited amount of fluid that can be provided in the SQ space, and sporadic absorption, even in only moderately dehydrated animals.^{5,27} Isotonic fluids without glucose should be given SQ so as to prevent sloughing of skin over the injection site.²⁷

Intravenous catheters for bolus or continuous infusion of fluids are best placed in the external jugular vein.²⁷ Twenty-three to 25 ga catheters can be placed in the cephalic vein but the small size of the veins and short legs of pediatric animals make cephalic placement and maintenance of fluid flow difficult.²³ One recommended protocol for IV rehydration in pediatric patients is 4-10 ml/kg/hr of 0.45% saline with or without 5% dextrose.²⁷

Intraosseous administration of fluids is an alternative to IV therapy in animals in which placement of an IV catheter is impossible or would take an excessive amount of time. An 18-22 ga spinal needle is passed through the soft cortical bone at the trochanteric fossa of the femur or greater tubercle of the humerus. The needle is inserted parallel to the long axis of the bone into the intramedullary canal. Fluid flow rates of up to 11 ml/minute can be achieved with gravity, and the fluid is readily absorbed.²⁷ Catheter maintenance is as for IV placement.

INFECTIOUS DISEASES

Severity of clinical signs with infectious disease is dependent on nutrition, thermoregulation, concurrent parasitism and developmental or hereditary defects of the immune system, and acquisition of passive immunity.

PUPPIES

HERPESVIRUS

Herpesvirus is an opportunistic virus that most readily infects bitches in late gestation, causing stillbirths and abortion, or puppies during passage through the birth canal or in the first 3 weeks of life, causing acute neonatal viremia.^{15,29,30} Puppies exhibit abdominal pain and constant crying and die within 24-48 hours of onset of signs.³⁰ Diagnosis usually is made at necropsy, during which hemorrhagic necrotizing changes are seen as petechiation of the kidneys, liver, and intestinal mucosa.^{15,29} Excessive pleural and abdominal fluid may be present, and inclusion bodies may be identified in hepatocytes.²⁹ Treatment generally is unrewarding. The optimal temperature for incubation of the virus in tissue culture is 95.0 - 98.6°F (35.0 - 37.0°C); replication may be inhibited in infected puppies by maintaining body temperature above 101.0 - 102.2°F (36.3 - 37.0°C).^{12,15}

INFECTIOUS CANINE HEPATITIS

Puppies may be infected with this adenovirus *in utero* or during passage through the birth canal. This disease usually is diagnosed at necropsy; characteristic intranuclear inclusion bodies are present in hepatocytes. Infectious canine hepatitis is rare due to vaccination.¹⁵

CANINE DISTEMPER

This disease also is uncommon due to vaccination. It usually is diagnosed at necropsy in puppies; thymic atrophy, bronchopneumonia, and characteristic intranuclear inclusion bodies are seen.¹⁵

KITTENS

UPPER RESPIRATORY INFECTION

A complex of respiratory diseases occurs in cats caused by viruses, such as rhinotracheitis and calicivirus, bacteria including Bordetella bronchiseptica and Chlamydia psittaci and, rarely, fungal organisms.^{5,24,31} Queens often are asymptomatic until stressed by queening, at which time organisms are shed and the less immunocompetent kittens infected.^{5,31} Clinical signs vary from mild conjunctivitis and serous oculonasal discharge to sneezing, tenacious oculonasal discharge, self-trauma due to pawing at the face, and respiratory distress.^{5,31} The condition usually is self-limiting and resolves in 10-14 days. Antibiotic therapy may hasten resolution of clinical signs. Appropriate vaccination within the colony is important for control. Calicivirus infection may be associated with mononuclear cell infiltration of joints, causing a lameness that usually is self-resolving; this is sometimes termed "limping kitten syndrome".²⁴

PANLEUKOPENIA

Kittens infected with feline distemper parvovirus *in utero* develop cerebellar hypoplasia. Those infected as neonates exhibit acute onset of vomiting and diarrhea, fever, rapid dehydration, leukopenia and death. The disease is uncommon due to vaccination.⁵

FELINE LEUKEMIA

Feline leukemia virus can be transmitted to susceptible kittens while nursing or by any other close contact with infected animals. Infected kittens undergo lymphoid depletion with thymic atrophy. Clinically, they exhibit failure to thrive and secondary sepsis.⁵

FELINE INFECTIOUS PERITONITIS

Feline infectious peritonitis (FIP) is rarely described as a cause of mortality in very young kittens. Diagnosis via serology is difficult due to cross-reaction with the relatively non-pathogenic enteric coronavirus (FECV). Animals infected with FECV, including kittens, shed virus before becoming viremic, lessening the value of serologic testing as a means to decrease introduction of infected animals into the cattery.³²

BARTONELLA

Bartonella henselae is the causative organism in cat scratch disease, a zoonotic disease most commonly passed to humans from cats less than one year of age. Infected kittens may be asymptomatic or may show lymphadenopathy, fever, lethargy and anorexia.³³

SEPTICEMIA

Septicemia, also called neonatal sepsis, is system-wide infection with one or more bacterial organisms.¹¹ Entry occurs most commonly via the umbilicus.³⁴ Other possible points of entry include the gastrointestinal tract, peritoneal cavity, respiratory tract, skin lacerations, and urinary tract.¹¹ Causative organisms usually can be cultured from the dam's vaginal secretions.^{35,36} The animal may be predisposed to septicemia by inadequate ingestion of colostrum or concurrent disease of the neonate or dam.¹⁶ Gram-negative organisms are most commonly involved, with E. coli the most prevalent isolate.^{11,14} Other possible causative organisms include species of Staphylococcus, Streptococcus, Klebsiella, Pseudomonas, Pasteurella, Enterobacter, Enterococcus, Clostridium, Bacteroides, Fusobacterium, Brucella, and Salmonella.^{11,14-16,19,34}

Clinical signs vary with the organ(s) affected. Reported syndromes include gastroenteritis with foamy vomitus, liquid diarrhea, reddening of the anus, rapid dehydration and death, pyelonephritis with abdominal pain, fever, dehydration and hematuria, omphalitis, conjunctivitis, pneumonia with respiratory distress and cyanosis, and non-specific weakness, vocalization and dehydration.^{12,14,19,34} Acute respiratory distress syndrome, characterized by life-threatening non-cardiogenic pulmonary edema, may occur secondary to septicemia, as may sloughing of the extremities, perhaps due to concurrent disseminated intravascular coagulation (DIC), tissue hypoxemia or vasculitis.^{12,31}

Putative diagnosis is based on clinical signs, presence of normocytic normochromic anemia, thrombocytopenia, and mild to moderate neutrophilia with a left shift on complete blood count, and hypoglycemia on serum chemistry profile.¹¹ Hypoglycemia may develop due to impaired glycogenolysis and gluconeogenesis, decreased liver perfusion due to congestion of major organs, and increased use of glucose by bacteria and leukocytes.^{34,37} Definitive diagnosis requires blood culture. Blood cultures can be performed by diluting 1 ml of whole blood with 5-10 ml enrichment broth, and examining the broth culture 6-18 hours later.^{16,24} Urine culture may be positive in some septicemic animals.²⁸ Septicemia is commonly diagnosed at necropsy.⁴

Treatment of septicemia requires fluid therapy to counter dehydration and hypoglycemia (balanced electrolyte solution with 5% dextrose and KCl supplement if serum potassium concentrations are less than 2.5 mEq/L), oxygen therapy for management of tissue hypoxemia, and appropriate antibiotic therapy.^{12,15,16} Penicillins and cephalosporins are appropriate empirical choices pending culture and sensitivity results.¹⁴

CONGENITAL DEFECTS

Congenital defects, those present at birth, may be hereditary, developmental, or due to exposure of the dam to teratogenic substances.^{11,38} The fetuses are most susceptible to noxious influences in the first third of pregnancy, during organogenesis.^{6,38} The congenital defect most commonly reported in a survey of 51 kittens was cleft palate.⁴ A survey of 1679 pups aged 8-16 weeks identified cryptorchidism, patellar luxation, cardiac abnormalities with murmurs, cleft palate, and umbilical and inguinal hernias as the most common congenital defects.³⁹

Excellent reviews of congenital and hereditary defects in puppies and kittens exist.⁴⁰⁻⁴⁴ Examples of hereditary defects are cryptorchidism and patellar luxation. If a given defect occurs in more than one litter from a given dam or sire, or frequency of the defect increases with inbreeding, a hereditary basis should be suspected and the animals involved removed from the breeding program.^{10,28} Gross anatomic abnormalities are visible at necropsy. Microanatomic and biochemical abnormalities may be identified by special testing available at the University of Pennsylvania.^{11,28}

Developmental abnormalities may occur in offspring from dams with metabolic disease. For example, women with diabetes mellitus are three times more likely to have babies with defects of the heart and neural tube than women from the general population.¹⁰ Chromosomal aberrations have varying effects on development; autosomal abnormalities generally are incompatible with life, while sex chromosome anomalies more commonly produce offspring with abnormalities of the reproductive tract and genitalia.⁴⁵ Developmental defects may or may not cause secondary disease.^{8,46}

Effects of drugs during pregnancy in dogs and cats often are extrapolated from work in other species (Table 1). Teratogens present during the first 26 days after conception often cause cephalic, ocular, otic, and/or cardiac abnormalities while those present in the transition period immediately following day 26 are more likely to cause palate, cerebellar, and/or urogenital defects.³⁸ Defects of the central nervous system, cardiovascular system, and respiratory tract, depending on degree, often are incompatible with life.¹¹ Queens infected with or, presumably, vaccinated with modified live virus vaccine for panleukopenia may give birth to kittens with cerebellar hypoplasia.⁵

Table 1. Teratogenic drugs^{10,38}

Type	Representative drug(s) and effect
Anti-convulsants	Primidone (cardiac defects, cleft palate, skeletal abnormalities)
Anti-infectives	Griseofulvin (microphthalmos [kittens], cleft palate [puppies]) Ketoconazole Tetracycline and aminoglycoside antibiotics Metronidazole
Anti-inflammatories	Aspirin Dimethylsulfoxide (DMSO) Glucocorticoids (anasarca in brachycephalic breeds)
Anti-neoplastic agents	
Hormones	Diethylstilbestrol (DES) and estradiol cypionate (ECP) (feminization of males) Testosterone and mibolerone (masculinization of females) Progesterone (masculinization of females)
Sedatives	Diazepam and midazolam
Vitamin excess	Vitamin A (cleft palate, kinked tails, cardiac defects [kittens]) Vitamin D (tissue calcinosis, enamel hypoplasia, cardiac defects)

PROBLEMS OF SPECIFIC SYSTEMS

EYES / EARS

Ophthalmia neonatorum is conjunctivitis and infection behind closed eyelids in kittens and puppies less than 10-14 days of age.^{5,12} It is common in catteries with endemic herpesvirus (rhinotracheitis) infection.⁵ Treatment is gentle separation of the eyelids and application of topical ophthalmic ointment.

Cataracts may develop in puppies and kittens fed either commercial or home-made milk replacers.^{5,12} These small focal cataracts usually resolve spontaneously after weaning.

Upper respiratory infections also may cause ocular disease. Chlamydia psittaci often causes only mild ocular disease in kittens in the United States. Recommended therapy is daily treatment with tetracycline ophthalmic ointment for 2 weeks.²⁴ The kittens should be monitored carefully for adverse reactions. If a negative reaction occurs, the eyes should be flushed with saline, chloramphenicol ophthalmic ointment substituted, and white blood cell number monitored for decline.²⁴

SKIN

Fleas are a common external parasite of young animals. Severe flea infestation can cause anemia, with clinical signs including pale mucous membranes, lethargy, tachycardia, and collapse and death.⁵ Fleas also can transmit tapeworms. The preferred treatment for fleas in animals less than 2 months of age is thorough bathing and grooming with a flea comb.²⁴ Dipping and/or systemic treatment is not recommended for animals less than 3 months of age.²⁴

Dermatophytosis is relatively common, especially in catteries. Microsporum caninum is the most common isolate.⁵ The kittens exhibit progressive crusty alopecia and may or may not be pruritic. The organism lives in dead skin and in the hair shafts of infected animals. It is difficult to eradicate from the environment and is zoonotic.⁵ If griseofulvin is used for treatment, kittens may exhibit side-effects of anorexia, vomiting, diarrhea, anemia and leukopenia, and ataxia. Griseofulvin is slowly metabolized in kittens and may be hepatotoxic.

NERVOUS SYSTEM

Neurological disorders may be congenital, such as hydrocephalus and cerebellar ataxia, or acquired, such as traumatic injury to the spinal column, lead toxicity, or parasitism.^{5,47,48} Bilirubin encephalopathy (kernicterus) is a rare cause of brain damage in kittens, due to high concentrations of unconjugated bilirubin in serum.⁴⁹

GASTROINTESTINAL TRACT

“Toxic milk syndrome” is a term often used to describe increased vocalization and abdominal distension in 3-14 day old puppies or kittens.¹² This is more likely to be due to hypothermia with secondary ileus or to overfeeding than to abnormalities of the dam’s milk.^{12,24}

Diarrhea often occurs secondary to overfeeding of neonates or disruption of the normal gastrointestinal environment with changes in diet or antimicrobial therapy.⁸ Primary bacterial diarrhea also has been reported.^{50,51} Diarrhea may be difficult to diagnose in young animals with zealous dams and may be first observed as lack of weight gain and dehydration. Treatment is supportive care with fluid therapy and assessment of the feeding schedule.

Intestinal parasites are common in young animals, especially those born in warm climates or in closely managed facilities. In puppies, roundworms (Toxocara canis) and hookworms (Ancylostoma caninum) are very common. Roundworms can pass transplacentally in late gestation.^{12,15} Within infected puppies, the larvae may migrate to the lungs and liver, causing a non-productive cough and poor weight gain.¹² Hookworms are passed via the dam’s milk and can cause significant blood loss in infected pups within 8 days of infection although oocysts are not shed in the feces until about 14 days after infection.^{12,15} Treatment of puppies 2 weeks of age or older is with pyrantel pamoate (5-10 mg/kg per os once daily for 2-3 weeks).^{16,24} Prevention involves treatment of the bitch with fenbendazole (50 mg/kg per os once daily) from the fortieth day of gestation to 14 days post-partum.^{12,15} Kittens also can be infected with roundworms; treatment is with pyrantel pamoate, as described previously. Use of piperazine is not recommended in kittens.²³

Coccidiosis can occur in either puppies or kittens. Infection usually is asymptomatic and self-limiting.⁵ If diarrhea occurs, treatment may be instituted with sulfadimethoxine (30 mg/kg once daily or 15 mg/kg twice daily in puppies, 30 mg/kg once daily in kittens weighing at least 1 kg) until signs regress.²³ Similarly, infection with Giardia sp. usually is asymptomatic and self-limiting.⁵ If necessary, puppies and kittens can be treated with either metronidazole (30 mg/kg per os once daily for 7-10 days or 25 mg/kg per os twice daily for 5 days, then 10 mg/kg twice daily) or fenbendazole (50 mg/kg per os once daily for 3-7 days).^{16,23} Pentatrichomonas hominis is a trichomonad parasite, reported to cause diarrhea in seom affected kittens.⁵²

Hemobartonella felis may be an incidental finding on complete blood counts in kittens, or may be a cause of red blood cell destruction, anemia, and icterus. It can be treated with doxycycline (10 mg/kg once daily) and decreasing doses of prednisone.²³

Toxoplasma gondii is an uncommon cause of a syndrome in kittens characterized by neurological signs, fever, respiratory disease, anemia, lymphadenopathy, and death within 3-12 days.^{5,53} Mode of transmission is undefined, but congenital infection seems likely since the disease has been definitively diagnosed in kittens as young as 2 weeks of age.⁵³ Treatment is with triple sulfonamides, pyrimethamine, and folic acid; efficacy is questionable. Infected kittens, their feces and their bedding should be isolated and all contaminated materials destroyed. Toxoplasma oocysts are resistant to commonly used disinfectants. A zoonotic potential exists; pregnant women should not handle infected cats or contaminated materials.

MUSCULOSKELETAL - "SWIMMERS"

Swimmer puppies and kittens have dorsoventral compression and lateral widening of the thoracic cavity.¹² Cause is unknown; hereditary and environmental factors (such as slippery flooring) may be involved. Treatment is taping of the limbs in an adducted position and/or provision of a non-slip surface or an uneven surface, such as egg carton foam.⁵⁴ Nandrolone laurate (10 mg) may be administered twice with a 2 week interval, presumably to promote growth of muscle and joint-associated connective tissue.⁵⁵

FADING PUPPIES AND KITTENS

Causes of fading in puppies and kittens depends on definition of the term. Taken at its most broad, fading puppies and kittens are those that are either born weak and fail to thrive, or are vigorous and weaken, dying by about one week of age. The causes are many and include (1) septicemia, (2) inadequate environment, (3) low birth weight, (4) congenital abnormalities, (5) maternal neglect or trauma, (6) lack of ingestion of colostrum or inadequate milk, and (7) neonatal isoerythrolysis (described in cats only).^{7,11,28,38,56-58} In most cases, more than one of these factors probably is causative, with primary factors such as hypothermia, deficient colostrum ingestion and immunodeficiency predisposing the animal to secondary infection with subsequent hypoglycemia and dehydration leading to cardiopulmonary failure.⁵⁶ Hypothesized causes of fading in puppies and kittens include poor thymic development with abnormal development or maturation of T cells,⁸ undefined hereditary factors causing increased neonatal mortality in inbred animals,⁵⁷ thyroid dysfunction which may respond to treatment with 3-5 µg L-thyroxine per os once daily,¹⁹ and abnormalities of surfactant causing inability of the neonate to breathe and suckle normally.³⁸

Treatment of fading puppy and kitten syndrome is dependent on identification of the underlying cause. There is a report of successful treatment of puppies with a non-specific inimmunostimulant.⁵⁹

NEONATAL ISOERYTHROLYSIS^{7,28}

Neonatal isoerythrolysis is an acute disease of kittens in the first days of life. Cats have naturally occurring antibodies blood types they are lacking; development does not require previous pregnancy or transfusion. There are two main feline blood types, A and B. Homologous A/A or heterozygous A/B cats are type A. Homozygous B/B cats are type B. The rare AB blood type occurs with presence of a third allele that is codominant with B.⁶⁰ Type A cats have weak anti-B antibodies while type B cats have strong anti-A antibodies. Kittens with blood type A born to type B queens become acutely ill after ingestion of colostrum and absorption of her anti-A antibodies.

Clinical signs in the kittens include anemia, icterus, tail tip necrosis due to hemagglutination in peripheral capillaries and localized thrombus formation, weakness, tachypnea, tachycardia, hemoglobinuria, and sudden death. Severity of signs may vary within the litter, presumably due to variability in antibody uptake.

The mortality rate is high in affected kittens, even with prompt intervention. The kittens should be removed from the dam and transfused if necessary. The dam is a good blood donor since she has no antibodies to her own red blood cells.

Prevention involves avoiding incompatible matings (type B queens to type A toms). Estimation of proportion of incompatible matings by breed is 0.25% for domestic short-haired cats, and as high as 14-25% for Persians and Abyssinians. The type B blood type is most prevalent in the Devon and Cornish Rex and British Shorthair breeds (Table 2). Blood-typing can be performed in-house (DMS Laboratories, Flemington NJ, 1-800-567-4367) or by commercial laboratories.

Table 2. Frequency of type A and B blood in cats in the United States²⁸

Breed	Type A (%)	Type B (%)
Abyssinian	81	19
Birman	82	18
British Shorthair	41	59
Devon Rex	57	43
Himalayan	80	20
Persian	76	24
Scottish Fold	85	15
Somali	78	22
Domestic Shorthair (DSH)	99	1

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