CHAPTER

41

Pediatrics

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OUTLINE

Most veterinarians have been presented with kittens that have failed to thrive, often called "fading kitten syndrome." These patients are challenging due to their small size, their unfamiliar physiology, and the tendency for their status to deteriorate quickly. Sick neonates should be examined as soon as possible with a systematic approach, including a complete history of the kitten, litter, and queen; examination of the kitten and queen; and diagnostic tests.[21,52]

KITTEN MORBIDITY AND MORTALITY

High-risk time points for kitten morbidity and mortality are at birth, in the first 2 weeks of life, and around the time of weaning. There is no specific disease entity attributable to "fading kittens," but rather a variety of causes have been identified. "Toxic milk syndrome" is often implicated when the cause of neonatal illness or death cannot be determined. There is no evidence that such a syndrome exists in kittens, and attention should be focused on finding the actual cause of illness or death. The most common causes of morbidity and mortality in kittens include[20,39]

- Perinatal events (e.g., dystocia, poor maternal care)
- Low birth weight
- Congenital defects
- Failure of passive transfer of immunity
- Neonatal isoerythrolysis
- Inadequate nutrition
- Environmental factors (e.g., poor ventilation, temperature fluctuations, overcrowding)
- Infectious diseases

In free-roaming populations, kitten mortality may be as high as 75%, with trauma and infectious disease accounting for most deaths.[60] The lowest mortality rates (less than 5%) are found in well-managed, specific–pathogen-free colonies. Pedigreed breeding catteries are a unique opportunity to collect feline reproduction data, including data on neonatal health and illness. In a study in the United Kingdom, data were collected using a convenience-sampling questionnaire on the births of 1056 litters (4814 kittens), representing 14 pedigreed breeds.[75] The breeds included in the study were Persian, Burmese, Siamese, British Shorthair, Oriental Shorthair, Birman, Devon and Cornish Rex (data analyzed together), Asian, Abyssinian, Korat, Somali, Maine Coon, Tonkinese, and Exotic Shorthair. Several parameters were affected by breed, such as mean stillbirth rate (highest in Persians and Exotic Shorthairs, lowest in Maine Coon) and mean kitten birth weight (highest in Maine Coon, lowest in Korat). Selected data from this study are summarized in Table 41-1.

In another study of 1191 litters (4804 kittens), data were collected prospectively using an Internet-based convenience-sampling submission system on 11 breeds

TABLE 41-1 Breed-Specific Reproduction Data Collected Using a Questionnaire-Based Survey in the United Kingdom

Breed	Number of Litters (Total = 1,056)	Mean Litter Size (No. Kittens)	Mean Birth Weight (Grams)*	Mean % Kittens Born Alive	Mean % Alive at 8 Weeks	% Litters with ≥1 Kitten Defects
Persian	212	3.8	92.8	89	75	9
Burmese	150	5.7	86.2	93	84	20
Siamese	138	4.9	92.4	92	82	19
British Shorthair	110	4.6	104.4	94	87	11
Oriental Shorthair	92	4.7	89.8	92	83	24
Birman	88	3.6	101.0	98	94	7
Devon + Cornish Rex	47	4.2	91.4	92	84	13
Asian†	41	6.5	84.7	95	93	19
Abyssinian	40	3.9	100.0	98	94	11
Korat	32	4.6	72.7	97	85	28
Somali	31	3.6	90.7	94	86	10
Maine Coon	27	4.6	116.1	99	90	15
Tonkinese	27	5.3	84.1	93	82	31
Exotic Shorthair	21	4.2	97.2	88	82	5
Overall means		4.6	93.5	93	84	14

*Kittens born alive.

†The Asian breed is similar to the Burmese but with a wider range of colors and patterns (tabbies, smokes, and solids or selfs). It also includes the Burmilla, Bombay, and Tiffanie.

Modified from Sparkes AH, Rogers K, Henley WE et al: A questionnaire-based study of gestation, parturition and neonatal mortality in pedigree breeding cats in the UK, *J Feline Med Surg* 8:145, 2006.

(S. Little, unpublished data, 2001 to 2005). Breeders submitting data were located primarily in the United States and Europe. The breeds included in the study were Bengal, Birman, Egyptian Mau, Havana Brown, Manx, Munchkin, Norwegian Forest Cat, Ocicat, Ragdoll, Devon Rex, and Sphynx. The mean stillbirth rate was highest in the Havana Brown and lowest in the Ocicat and Devon Rex. Mean kitten birth weight was highest in the Norwegian Forest Cat and lowest in the Havana Brown. Selected data from this study are summarized in Tables 41-2 and 41-3.

Data from such studies are very useful to help breeders evaluate their breeding programs and identify areas of poor performance. For example, mean kitten mortality by 4 to 8 weeks of age in these studies is approximately 15%. A study in Sweden involving 694 litters from various breeds calculated mean kitten mortality by 12 weeks of age as 18%.[76] Breeders experiencing greater losses should investigate possible causes. Caution must be exercised, however, because even though breeds may have the same name and similar phenotype in different countries, the genetic constitution may be quite different. A good example is the Burmese breed. The definition of this breed varies by country and registering organization, leading to genetically distinct populations with different disease risks.

EXAMINATION OF THE NEONATAL KITTEN

Neonatal kittens (up to 4 weeks of age) cannot be approached as small adults. Not only is their physiology different, but the basic approach to history and examination is different. Start with a complete medical history for the kitten in question as well as for littermates. It may also be helpful to have a medical history for the queen if available (e.g., illness, nutrition, vaccinations) and information about the labor and delivery, especially for kittens less than 2 weeks of age.

Investigate the kitten's home environment, noting temperature and humidity, sanitation, population size and density, and prevalence of infectious diseases and parasites. A home visit can gather important information, especially when working with breeders. However, if a home visit is not possible, the breeder can be asked to supply a floor plan and photos to help identify management issues. Unfortunately, little information exists on the optimum design of breeding catteries. Recommendations are often adapted from those designed for laboratory animals, or boarding facilities.[66] The Cat Fanciers' Association (http://www.cfa.org) has produced minimum requirements for catteries that address the environment (e.g., temperature, ventilation, lighting),

TABLE 41-2 Breed-Specific Reproduction Data Collected Using an Internet-Based Survey from Breeders Primarily in the United States and Europe, 2001 to 2005

Breed	Number of Litters (Total = 1,191)	Mean Litter Size (No. Kittens)	Mean Birth Weight (Grams)*	Mean % Kittens Born Alive	Mean % Alive at 4 Weeks
Ragdoll	232	4.4	98.8	95	90
Birman	204	3.6	95.3	93	87
Bengal	176	4.0	94.1	94	82
Ocicat	136	4.3	98.2	96	86
Norwegian Forest cat	124	4.5	105.3	95	89
Devon Rex	103	3.7	86.1	96	87
Munchkin	54	4.1	83.5	94	83
Egyptian Mau	52	3.7	104.9	94	89
Sphynx	50	4.0	95.5	95	80
Manx	39	3.1	93.4	92	82
Havana Brown	21	3.9	91.1	89	83
Overall means		4.0	95.1	94	85

*Kittens born alive.

TABLE 41-3 Data on Congenital Defects in Selected Pedigreed Cat Breeds Collected Using an Internet-Based Survey from Breeders Primarily in the United States and Europe, 2001 to 2005

Breed	No. Litters	% Litters with at Least One Congenital Defect	Examples of Congenital Defects
Bengal	181	18	FCK, PE, CP, UH, SYN
Birman	217	12	DER, UH, SYN, CP, GAS
Burmese: traditional	94	15	FCK
Burmese: contemporary	54	80	HD, DER, FCK
European Burmese	66	15	FCK
Devon Rex	204	13	FCK, CP, UH
Egyptian Mau	52	23	UH
Havana Brown	27	11	UH, FCK
Manx	31	13	GAS
Munchkin	54	11	CP, UH, GAS
Norwegian Forest cat	124	5	FCK, CP
Ocicat	128	25	FCK, PE, XIPH
Ragdoll	199	13	CP, COL
Sphynx	104	9	CP, PE, UH

COL, Eyelid coloboma; *CP,* cleft palate; *DER,* dermoids (nasal and ocular); *FCK,* flat chest defect; *GAS,* gastroschisis; *HD,* craniofacial (head) defect; *PE,* pectus excavatum; *SYN,* syndactyly; *UH,* umbilical hernia; *XIPH,* everted xiphoid process.

hygiene, and caging facilities. Although written about 20 years ago, valuable information on cattery design is found in *Feline Husbandry: Diseases and Management in the Multiple-Cat Environment* (http://www.vetmed.ucdavis.edu/ccah/felinehusbandry.cfm).[62]

Neonatal kittens should be examined with the queen and any available litter mates when possible, and in the home or cattery if problems are ongoing. Neonates should be handled gently, on a warm surface, such as a clean towel. Wash your hands and wear gloves. Simple

BOX 41-1

Developmental Milestones for Kittens

Umbilical cord falls off: 3 days of age
Eyelids open: 10 days of age (range 2 to 16 days)
Menace/pupillary light reflexes: 28 days of age or later
Normal vision: 30 days of age
Adult iris color: 4 to 6 weeks of age
Ear canals open: 9 days of age (range 6 to 17 days)
Functional hearing: 4 to 6 weeks of age
Crawling: 7 to 14 days of age
Walking: 14 to 21 days of age
Voluntary elimination: 3 weeks of age
Deciduous incisors/canines erupt: 3 to 4 weeks of age
Deciduous premolars erupt: 5 to 6 weeks of age

equipment will suffice: gram scale, pediatric digital rectal thermometer, otoscope with infant cones, penlight, and stethoscope with an infant bell and diaphragm.

One of the first challenges facing the clinician examining the neonatal kitten is to determine the age and sex. Unless the kitten comes from a breeding cattery, the exact birth date is often unknown. Several developmental milestones and parameters can be used to estimate the age of kittens (Box 41-1), such as dentition and body weight (see below). Sex may be surprisingly difficult to determine in very young kittens, especially without another kitten of opposite sex for comparison, because testicles are not readily visible until more than 6 weeks of age. In male kittens, the distance between the anus and genitals is greater (about 1.25 cm [0.5 inches]) than for female kittens. The genitals appear slitlike in female and appear rounded in the male. Coat color may also be a clue; almost all calico or tortoiseshell kittens are females, and orange kittens are most likely to be male (but not exclusively so).

Before handling the kitten, observe its body condition and response to the environment, including alertness, posture, locomotion, and respiratory rate. Healthy neonates will have strong righting, rooting, and suckling reflexes. Muscle tone should not be too flaccid or too rigid. Flexor muscle tone predominates for the first few days of life so that newborn kittens typically rest in a curled position. Once extensor tone develops, kittens rest on their side or chest with head extended. Normal kittens sleep about 80% of the time and are generally quiet when healthy, warm, and well fed. When stressed by hunger, the absence of the queen, or other reasons, normal kittens will cry and crawl around the nest box, moving the head from side to side in a searching motion. Pain perception is present from birth, but withdrawal reflexes are not well developed until about 1 week of age. Sick kittens have a limited number of clinical signs (e.g., crying, restlessness, failure to gain weight, weakness, respiratory difficulty, diarrhea), and therefore changes from normal should be investigated promptly.

Normal body temperature for newborns is 97° F to 98° F (36° C to 37° C). The rectal temperature rises slowly, reaching 100° F (38° C) by about 4 weeks of age. For the first few weeks of life, kittens are poikilothermic, relying on external sources of heat. Neonatal kittens are very susceptible to cooling because of their high surface area to body weight ratio, immature metabolism, immature shiver reflex, and poor vasoconstrictive abilities. The ability to shiver begins around 1 week of age. They gradually become homeothermic by 4 weeks of age.

The typical kitten birth weight is 90 to 110 g (range, 80 to 140 g), although there is considerable variation by breed (see Tables 41-1 and 41-2) and by sex (males typically weigh more than females).[75] Low birth weight is a common cause of mortality, with kittens weighing less than 75 g at birth at highest risk. Slight weight loss (less than 10%) can occur in the first 24 hours of life, but the kitten should then gain weight daily. Normal kittens gain 50 to 100 g per week (10 to 15 g/day) and should double their birth weight by 2 weeks of age. Breeders and other caretakers of newborn kittens should be instructed to weigh the kittens twice daily for the first 2 weeks of life, and then daily for at least the next 2 to 4 weeks. Often the earliest sign of illness is failure to gain weight in a 24-hour period.

Poor nutrition or inadequate nursing is a common cause of failure to gain weight. Smaller or weaker kittens may be excluded from nursing by stronger litter mates. Other causes of poor nutritional intake include congenital defects, such as cleft palate, or maternal factors, such as mastitis, systemic illness, or poor body condition.

The first deciduous teeth to appear are the incisors and canines at 3 to 4 weeks of age. The premolars erupt at about 5 to 6 weeks of age. The dental formula for deciduous teeth is 2(I3/3, C1/1, P3/2); there are no deciduous molars.

Inspect the kitten for gross anatomic abnormalities, such as cleft palate or lip, umbilical hernia or infection (omphalophlebitis), open fontanelles, limb deformities, chest wall deformities, and nonpatent urogenital or rectal openings. The normal umbilical cord is dry, with no redness, swelling, or discharge at the umbilicus (Figure 41-1). Umbilical cords will fall off at about 3 or 4 days of age.

Kittens younger than about 3 weeks of age cannot eliminate urine and feces voluntarily. The kitten's micturition and defecation reflexes can be evaluated by using a cotton ball with mineral oil to stimulate the anogenital area. Gastrointestinal function is well developed at birth, and normal flora is acquired within the first few days of life. Diarrhea is present in about 60% of sick neonatal kittens. Normal neonatal urine is dilute and pale in color. Hematuria or pigmenturia may

FIGURE 41-1 The normal umbilical cord is dry, with no redness, swelling, or discharge at the umbilicus and falls off at about 3 to 4 days of age.

be signs of urinary tract infection, trauma, or neonatal isoerythrolysis. Dark yellow urine is typically a sign of dehydration in neonates.

The eyes should be inspected for abnormalities of the globe or eyelids and for neonatal conjunctivitis (ophthalmia neonatorum, see below). The eyelids typically open at about 10 days of age, but occasionally kittens are born with eyes already open, or the eyes open in the first day or two of life. Because tear production will not be normal for several weeks, a topical lubricating ophthalmic ointment should be applied for about the first week of life to prevent corneal damage. A menace reflex does not appear until 28 days of age or later. Pupillary light responses may appear as late as 28 days as well. A divergent strabismus may be present and may be normal until about 8 weeks of age. Evaluation of the fundus is difficult until after 6 weeks of age.

The pinnae should be inspected for evidence of trauma, parasites such as ear mites, and skin disease. The ear canals are not easy to inspect with an otoscope until after 4 weeks of age. The neonate's hair coat should be clean and shiny. Healthy neonatal kittens may have hyperemic mucous membranes until 7 days of age, whereas sick neonates often have pale, gray, or cyanotic mucous membranes.

Neonatal kittens may have lower blood pressure than adults, as well as greater cardiac output, and a faster heart rate in the first 2 weeks of life. Functional murmurs may be present in neonates because of anemia, hypoproteinemia, fever, or sepsis. Innocent murmurs not associated with disease are more common in puppies than kittens; murmurs still present after 4 months of age should be investigated. Congenital heart disease usually produces murmurs that are loud and accompanied by a precordial thrill. The normal neonatal heart rate can be more than 200 beats per minute in the first 2 weeks of life (range 220 to 260). By 4 weeks of age, once vagal tone has been established, the heart rate decreases to the normal adult range. The normal respiratory rate is 15 to 35 breaths per minute. See Box 41-2 for normal physiologic values for neonatal kittens.

A full abdomen is normal in a well-fed kitten, but an enlarged abdomen in an ill kitten may indicate aerophagia. The normal liver and spleen may not palpable; the kidneys are frequently palpable. The stomach may be palpable if it is full. The intestinal tract is palpable as fluid-filled bowel loops that should be freely moveable and nonpainful. The urinary bladder is also palpable, moveable, and should be nonpainful.

BOX 41-2

Normal Physiologic Values for Neonatal Kittens

Birth weight: 90 to 110 g
Rectal temperature (newborn): 97° F to 98° F (36° C to 37° C)
Rectal temperature (1 month): 100ºF (38° C)
Heart rate: 220 to 260 beats/minute for the first 2 weeks of life
Respiratory rate (newborn): 10 to 18 breaths/minute
Respiratory rate (1 week): 15 to 35 breaths/minute
Urine specific gravity: <1.020
Urine output: 25 mL/kg/day
Water requirement: 130 to 220 mL/kg/day
Caloric requirement: 20 kcal ME/100 g/day
Stomach capacity: 4 to 5 mL/100 g

ME, Metabolizable energy.

CONGENITAL DEFECTS

Congenital defects are abnormalities of structure, function, or metabolism that are present at birth. A defect may cause physical impairment, or it may cause the death of the kitten before or after birth. Congenital defects in stillborn kittens often go unrecognized, because few stillborns are submitted for complete necropsy. Many congenital defects are cosmetic or minor, while others may cause serious impairment of health (Box 41-3). Congenital defects may be of various types:

- Obvious at birth (e.g., cleft palate [Figure 41-2] or imperforate anus [Figure 41-3])
- Found only with diagnostic testing or at necropsy (e.g., diaphragmatic hernia)
- Subtle abnormalities found only with sophisticated testing (e.g., lysosomal storage diseases)

BOX 41-3

Selected Common Congenital Defects in Kittens

Eyes and Ears

- Glaucoma
- Colobomas
- Microphthalmia
- Corneal dermoids
- Congenital nystagmus

Neurologic

- Hydrocephalus
- Cerebellar hypoplasia
- Deafness

Skin and Musculature

- Hypotrichosis
- Umbilical hernia
- Gastroschisis/schistosoma (abdominal hernia)

Cardiac

- Patent ductus arteriosus
- Atrioventricular defects

Skeletal

- Radial hemimelia
- Polydactyly
- Syndactyly
- Flat chest defect
- Pectus excavatum (“funnel chest”)

Urogenital

- Renal aplasia/hypoplasia
- Ambiguous genitalia/pseudohermaphroditism
- Cryptorchidism

Endocrine

- Congenital hypothyroidism/dwarfism

Gastrointestinal

- Cleft palate
- Atresia ani/anogenital fistula

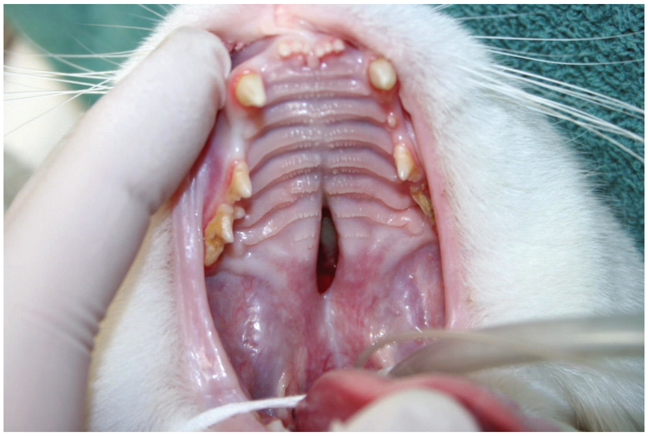

FIGURE 41-2 All kittens should be examined at birth for the presence of cleft palate, a common congenital defect. *(Courtesy Chris Carter.)*

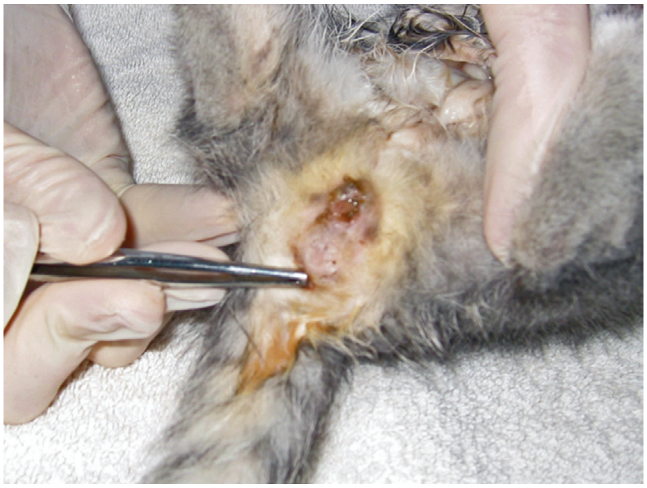

FIGURE 41-3 This female kitten has an imperforate anus and an anovaginal fistula. *(Courtesy Rosalyn MacDonald.)*

Feline fetal development can be divided into three stages: preimplantation (days 0 to 12), embryogenesis (days 12 to 24), and fetal growth (day 24 to term). The “critical period” is the stage during which each developing organ or structure is most sensitive to disruption. For most organs and structures, the critical period occurs during embryogenesis, in the third and fourth weeks of gestation. At the end of embryogenesis, the fetus is about ½ inch long. Developmental errors that occur during the first 2 weeks of gestation are usually lethal. It is also important to note that a defect in the development of one organ system or structure can result in the abnormal development of other organs or structures.

A teratogen is anything that disrupts normal fetal development (e.g., a drug or chemical). The timing of exposure of the fetus and the dose are important factors in determining outcome. Embryos are susceptible to teratogens, but this susceptibility tends to decrease as the critical developmental period for each organ system

BOX 41-4

Examples of Nongenetic Causes of Congenital Defects in Kittens

1. Infections in utero
   a. Usually viral, for instance, panleukopenia virus as a cause of cerebellar hypoplasia
2. Drugs
   a. For instance, griseofulvin as a cause of cleft palate
3. Chemicals, environmental toxins
4. Hyperthermia
   a. For instance, fever, high ambient temperatures, resting on heating pads, radiators or hot air vents, and so forth
5. Poor intrauterine environment
   a. For instance, inadequate development of the placenta, cystic endometrial hyperplasia/ pyometra complex
6. Nutritional factors
   a. For instance, taurine deficiency as a cause of neurologic abnormalities[79]

FIGURE 41-4 The flat chest defect is characterized by a dorsoventral flattening of the rib cage. *(Courtesy Barbara Hickmann.)*

passes. This makes the fetus increasingly resistant to the effects of teratogens with age, with the exception of structures that differentiate late in gestation, such as the cerebellum, palate, and urogenital system.

Congenital defects may be heritable, and the inheritance pattern or mutation(s) responsible may or may not be known (see Chapter 44). A few congenital defects are due to chromosomal abnormalities, such as pseudohermaphroditism. Many congenital defects are not heritable but caused by other factors (Box 41-4). In some cases, defects may be caused by interplay of both environmental and genetic factors. When pedigreed breeders encounter a congenital defect where no information is available on heritability, several factors can be evaluated. A defect is more likely to be heritable if there is evidence of a breed or familial predisposition and the problem has a consistent age of onset and clinical course. A defect is less likely to be heritable if more than one abnormality occurs in a kitten or a litter or there is potential exposure to teratogens. An informative fact sheet ("What to do if your cat produces a deformed kitten?") has been produced by the Feline Advisory Bureau to advise breeders on steps to take if a defect is observed (http://www.fabcats.org).

As in many other species, congenital defects are a significant contributor to neonatal mortality in the cat. Excellent reviews of congenital defects have been published,[31] including neurologic,[38] ocular,[25,56] renal,[26] cardiac,[45] and vertebral column defects.[57] There are also studies of congenital defects specifically in pedigreed cat breeds in the literature. One study noted that congenital disease was more common in pedigreed kittens than in the nonpedigreed kittens in a necropsy examination of 274 kittens aged up to 16 weeks.[9] However, the difference was not statistically significant and no individual breed of cat was significantly predisposed to congenital diseases in the data.

In a survey of 3468 pedigreed kittens in the 1970s, 6.8% had malformations.[70] Individual breeds ranged from no defects reported to 17% (Colorpoint Shorthair) and 19% (Manx) of kittens affected. Another study from the same time period reported 4.2% of Burmese kittens from one cattery had congenital defects, as well as 12.7% of Persian kittens from four catteries.[71] The types of abnormalities reported included heart defects, open fontanelles, gastroschisis, eye and eyelid defects, and gastrointestinal tract defects.

In the United Kingdom analysis of reproduction data from 14 breeds,[75] 14.9% of the litters included one or more kittens with congenital defects, ranging from 6% of Devon Rex litters to 31% of Tonkinese litters (see Table 41-1). The survey of cat breeders conducted primarily in North America and Europe (S. Little, unpublished data, 2001 to 2005) found wide variation in prevalence of congenital defects, from 5% (Norwegian Forest Cat) to 80% ("contemporary" American Burmese) of litters demonstrating at least one defect (see Table 41-3). The high prevalence of congenital defects in "contemporary"-style American Burmese is due to a common craniofacial deformity caused by an autosomal recessive genetic mutation.[59] Congenital defects common to many breeds include thoracic wall deformities (e.g., pectus excavatum, flat chest defect), cleft palate, and umbilical hernia. Although certain common congenital defects of kittens are well described in the literature, others are less well documented.

Flat chest defect is one of the common thoracic wall deformities in kittens that are not well described in the literature. Although the defect may be seen in any kitten, it appears to be most common in Orientals, Burmese, and Bengals. The defect is characterized by a dorsoventral flattening of the rib cage and sharp angulation at the costochondral junction (Figure 41-4).[78] Curvature of the cranial thoracic spine may also be present (Figure 41-5).

FIGURE 41-5 **A,** Kittens with flat chest defect may also have spinal curvature. **B,** Sharp angulation at the costochondral junction is evident.

FIGURE 41-6 Owners of kittens with flat chest defect often apply temporary splints made of cardboard to help shape the compliant chest wall. *(Courtesy Barbara Hickmann.)*

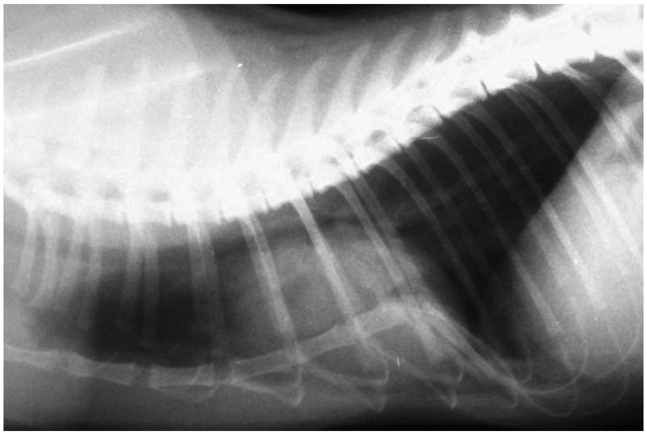

FIGURE 41-7 Pectus excavatum is characterized by dorsal deviation of the caudal part of the sternum. The defect reduces the size of the chest cavity and can lead to dyspnea and failure to grow.

The defect is not present at birth, but the mean age at which it is recorded is 9.5 days.[78] The defect has a variable presentation and is even transient in some cases. Mildly affected individuals may be difficult to detect. Moderate to severely affected kittens show poor weight gain, increased respiratory rate and effort, exercise intolerance, and they may die. In one study of Burmese kittens in the United Kingdom, 8.7% of the kittens with flat chest defect also had pectus excavatum, suggesting a possible association.[78] Although similar thoracic wall deformities have been reported in taurine-deficient kittens, affected Burmese kittens had higher whole blood taurine levels than unaffected kittens.[78] No investigation into the best treatment for kittens with life-threatening chest wall deformity has been published, but breeders and owners often apply temporary splints made of cardboard or plastic to force the compliant thoracic wall and sternum into a more normal confirmation until the ribs and sternum mature (Figure 41-6).

Pectus excavatum ("funnel chest") is characterized by dorsal deviation of the caudal part of the sternum (Figure 41-7). The sternal abnormality is noticed early in life, and the defect is usually progressive. The etiology in kittens and puppies is poorly understood, although reviews of the disease and its management have been published.[5,17] No breed or sex predisposition has been identified. The disease may be classified as mild, moderate, or severe based on radiographic measurements.[17,18] Mildly affected kittens typically have no clinical signs and do not require intervention. Severely affected kittens may be dyspneic, exercise intolerant, and fail to thrive. A heart murmur may be present, either resulting from congenital disease or secondary to abnormal positioning of the heart because of the sternal deformity.[18] The most commonly described surgical correction is percutaneous circumsternal sutures and external splinting (Figure 41-8).[18,19,50,72,85] This technique works well in kittens with a nonossified sternum (less than 4 months of age), because the goal is to pull the sternum outward and maintain the correct position while the sternum matures.

FIGURE 41-8 This Ragdoll kitten has had correction of pectus excavatum with circumsternal sutures and external splinting. *(Courtesy Sue Shorey.)*

Potential complications of the technique include inadvertent lung puncture or laceration and development of pneumothorax. One case of fatal reexpansion pulmonary edema has also been described.[72] Surgical approaches, such as wedge ostectomy,[72] trans-sternebral pinning,[13] and sternal wedge chondrectomy with internal splinting[64] have also been described.

Tarsal hyperextension (also known as "twisted legs," limb contracture, tendon contracture) is a recently described congenital defect in kittens.[7] The defect is obvious at birth and typically affects only one kitten in a litter. There is no breed or sex predisposition. The abnormality is characterized by severe tarsal hyperextension and metatarsal rotation (Figure 41-9). Typically both hind limbs are affected, but the condition can also be unilateral. No bone abnormalities or neurologic deficits are present. The etiology is unknown, but the defect appears similar to clubfoot in human infants. In the author's experience, the deformity completely resolves on its own in the majority of cases as the kitten begins to crawl and bear weight on the affected limbs. In some cases, delayed resolution has prompted the use of either soft molded splints or fiberglass casts for external coaptation.[7] If external coaptation is required, it should be instituted earlier rather than later to take advantage of the greater flexibility of the joints of young kittens. Splints and casts must be changed weekly as the kitten grows, and may be required for 6 weeks or longer. Physical therapy to gently manipulate the tarsal joint into normal configuration may also be helpful.

NEONATAL DIAGNOSTICS

Blood chemistry and hematology values for neonates differ from the adult; most values normalize to adult levels by 3 to 4 months of age (Tables 41-4 and 41-5). For several analytes, reference ranges change rapidly within the first few days and weeks of life so that using age-appropriate reference ranges is important. In one study of kittens up to 8 weeks of age, reference ranges for alkaline phosphatase (ALP), creatine kinase, triglycerides, calcium, and phosphorus were higher than for adults.[41] Reference ranges for aspartate aminotransferase (AST), bilirubin, urea nitrogen, and creatinine were higher in newborns, but similar to or lower than adults by 8 weeks of age. Reference ranges for albumin and total protein were lower than for adults for the entire 8 weeks, and values for calcium and phosphorus were higher.

For venipuncture, the holder positions the kitten in dorsal recumbency with the forelegs drawn back toward the abdomen and the head and neck extended. Blood is drawn from the jugular vein using a 1-mL syringe with a 25- or 26-gauge needle. Slow aspiration of blood is essential to avoid collapsing the vein. A small volume

FIGURE 41-9 A newborn Korat kitten with tarsal hyperextension, showing the characteristic tarsal hyperextension and metatarsal rotation. *(Courtesy Carine Risberg.)*

TABLE 41-4 Hematology Values for Kittens from Birth to 8 Weeks

	2 Weeks	4 Weeks	6 Weeks	8 Weeks
PCV (%)	33.6-37.0	25.7-27.3	26.2-27.9	28.5-31.1
RBC ($\times 10^6/\mu L$)	5.05-5.53	4.57-4.77	5.66-6.12	6.31-6.83
WBC ($\times 10^6/\mu L$)	9.10-10.24	14.10-16.52	16.08-18.82	16.13-20.01
Neutrophils	5.28-6.64	6.15-7.69	7.92-11.22	5.72-7.78
Lymphocytes	3.21-4.25	5.97-7.15	5.64-7.18	8.02-11.16
Monocytes	0.0-0.02	0.0-0.04	0	0.0-0.02
Eosinophils	0.53-1.39	1.24-1.56	1.22-1.72	0.88-1.28

PCV, packed cell volume; *RBC,* red blood cell; *WBC,* white blood cell.
Adapted from Moon P, Massat B, Pascoe P: Neonatal critical care, *Vet Clin North Am Small Anim Pract* 31:343, 2001.

TABLE 41-5 Serum Chemistry Values for Kittens from Birth to 8 Weeks

	2 Days	1 Week	2 Weeks	4 Weeks	8 Weeks
Albumin (g/dL)	1.6-2.6	2.0-2.5	2.1-2.6	2.4-2.9	2.4-3.0
ALP (U/L)	275-2021	126-363	116-306	97-274	60-161
ALT (U/L)	12-84	11-76	10-21	14-55	12-56
Bilirubin (mg/dL)	0-0.7	0-0.6	0-0.2	0-0.3	0-0.1
Calcium (mg/dL)	8.6-12.7	10.0-13.7	9.9-13.0	10.0-12.2	9.8-11.7
Cholesterol (mg/dL)	80-175	119-213	137-223	173-253	124-221
Creatinine (mg/dL)	0.5-1.1	0.3-0.7	0.4-0.6	0.4-0.7	0.6-1.2
GGT (U/L)	0-5	0-5	0-4	0-1	0-2
Glucose (mg/dL)	75-154	105-145	107-158	117-152	94-143
Phosphorus (mg/dL)	4.1-10.5	6.7-11.0	7.2-11.1	6.7-9.0	7.6-11.7
Total protein (g/dL)	3.9-5.8	3.5-4.8	3.7-5.0	4.5-5.6	4.8-6.5
Urea (mg/dL)	24-71	16-36	11-30	10-22	16-33

ALP, alkaline phosphatase; *ALT,* alanine aminotransferase; *GGT,* gamma-glutamyltransferase.
Adapted from Levy J, Crawford P, Werner L: Effect of age on reference intervals of serum biochemical values in kittens, *J Am Vet Med Assoc* 228:1033, 2006.

(0.5 mL) of blood can be used for the most critical tests (Box 41-5). Use of 0.5-mL microsample blood collection tubes is recommended and has been validated for evaluation of biochemistry and hematology samples.[82,83] Repeated sampling should be done cautiously, because the circulating blood volume of kittens is small (approximately 70 to 95 mL/kg). Daily blood sampling should not exceed 10% of the kitten's estimated total blood volume.

Urine is collected for chemistries, microscopic examination of sediment, and specific gravity by stimulating the perineum; cystocentesis should be performed with great care or avoided in the very young, because the bladder wall is easily lacerated. Urine specific gravity is 1.020 or less in the first few weeks of life; adult values are reached by about 8 weeks of age.[46] A fecal sample should be examined for common intestinal parasites, such as *Giardia* spp., *Isospora* spp., and roundworms, using both zinc sulfate centrifugation and a direct saline smear.

Radiography is a useful diagnostic tool, but is more difficult to perform than in adults because of the small size and often uncooperative nature of kittens. If required, the author prefers mask induction and maintenance with isoflurane as the safest method for immobilization. It is also difficult to interpret radiographic images of kittens, because contrast is poor from lack of body fat and mineralization of the skeleton is incomplete. The quality of images can be improved by setting the kilovoltage to about half of that used for an adult and using high-detail film or screens.

Quality thoracic images may be difficult to obtain because of the high respiratory rate of kittens and greater chest wall motion. Absence of motion and good positioning are critical. Common indications for thoracic radiographs are evaluation of heart murmurs and diagnosis of pneumonia. Thoracic radiographs of kittens show a generalized increase in pulmonary interstitial opacity because of the increased water content of the lung parenchyma. The thymus may appear as a sail sign in the cranial left hemithorax, and the cranial mediastinum will appear wider than in the adult. The heart appears proportionately larger in the thoracic cavity because of decreased alveolar volume.

Interpretation of skeletal radiographs presents difficulties because of decreased mineralization, open physes, and secondary centers of ossification. Trauma and infection are the most common lesions found, and are often associated with soft tissue swelling. It can be very helpful to radiograph the unaffected limb at the same time for comparison to aid in interpretation.

BOX 41-5
Minimum Database for Sick Neonatal Kittens

- PCV and total solids using microhematocrit tubes and refractometer
- Complete blood count: white blood cell count from one drop whole blood directly into Unopette, differential from blood smear
- Blood urea nitrogen with whole blood on reagent strip
- Blood glucose determined with drop of whole blood using glucometer (note these machines tend to underestimate blood glucose)

The usefulness of abdominal radiography in kittens is hampered by poor abdominal detail because of lack of intraabdominal fat, a small amount of normal peritoneal fluid, and a higher proportion of total body water. The liver appears comparatively larger than in the adult. The most common diagnoses are radiopaque foreign bodies and intestinal obstruction.

Ultrasonography is an effective modality for pediatric patients, especially for imaging the abdomen. Machines with a curvilinear variable frequency scan head (6.0 to 8.0 MHz) have been recommended.[3] Sedation is rarely required for the procedure, and the kitten is best positioned in dorsal recumbency in a padded trough. Techniques for examining the abdomen and the normal appearance of structures have been described.[3] Common indications for abdominal ultrasonography include gastrointestinal foreign bodies, intussusceptions, congenital hernias, congenital renal disease, and urolithiasis, among others.

Normal electrocardiographic values for kittens in the first 30 days of life have been described.[44] Changes in normal findings occur during the first month of life, such as shift of the electrical axis from right to left, a progressive increase in R wave amplitude, and a progressive decrease in S wave amplitude. Measurements for P wave, PR intervals, duration of QRS complexes, and the duration of the QT interval are similar to adult cats. Neonatal kittens have a sinus heart rhythm.

Necropsy is underutilized as a diagnostic tool for multicat environments, such as shelters or catteries. It is not uncommon for kittens to die or be euthanized before a definitive diagnosis can be established. Necropsy results may provide information necessary to save remaining littermates or a future litter, especially for infectious diseases. For the best results, the whole body should be submitted (refrigerated, not frozen) to a qualified pathologist. If necessary, freezing is preferable to autolysis, because some information can still be obtained.

BASIC THERAPEUTICS

Rapid identification of illness and prompt intervention are the keys to success when treating ill neonatal kittens. Intensive care of the sickest kittens can be very successful although intimidating. Often the exact cause of a kitten's illness is not apparent at the time of presentation, and therapy must be focused on supportive care. Initial therapy typically includes treatment of the "3 H" problems (hypothermia, hypoglycemia, and hydration) with supplemental warmth, hydration, glucose, and nutrition.[22,52] Awareness of physiologic differences between neonatal and adult cats is important, and recently published information should be reviewed.[29]

For newborn kittens, a fourth "H" can be added to the list—hypoxia. The main risk factor for hypoxia is dystocia, leading to prolonged time in the birth canal with placental separation and therefore decreased oxygen. Resuscitation of these neonates must be prompt and aggressive, involving rubbing to dry and stimulate breathing, and aspiration of airway secretions (see Chapter 40). Other factors that have a negative impact on the survival of kittens born during dystocia include the effect of drugs given to the queen for surgery, brain trauma from "swinging" to resuscitate (no longer recommended),[30] and hypothermia induced by the cool temperatures of most operating rooms. These kittens may survive the dystocia, but are often weak and unable to nurse effectively. The effects of hypoxia and hypothermia may cause death within a few days.

Rectal temperatures less than 34.4° C (94° F) are associated with depressed respiration, impaired function of the immune system, bradycardia, and ileus. Never attempt to feed a hypothermic kitten, because aspiration pneumonia is a significant risk. Hypothermic kittens should be re-warmed slowly, during 2 to 3 hours to a maximum rectal temperature of 38.3° C (101° F). Warming too rapidly may cause increased metabolic demand, resulting in dehydration, hypoxia, and loss of cardiovascular integrity. An incubator or oxygen cage is a good way to accomplish re-warming and has the benefit of providing humidified air. However, hot-water bottles, circulating warm-water blankets, and heating lamps can also be used with very careful monitoring. The kitten should be able to move away from the heat source. The environmental temperature for neonatal kittens should be about 27° C to 32° C (80° F to 90° F).

For severely hypothermic kittens, fluids warmed to 35° C to 37° C (95° F to 98.6° F) may be administered by the intravenous (IV) or intraosseous (IO) route. Bags of fluid can be warmed (but will cool quickly) or an in-line IV infusion warmer may be used. Another option is to place the portion of intravenous tubing nearest the kitten under a warming device, such as a heating pad or a circulating warm-water blanket. Monitor closely for recurrence of hypothermia after re-warming.

Clinical hypoglycemia occurs when the blood glucose is less than 3 mmol/L (50 mg/dL) and is a common problem for sick neonates because of immature liver function and rapid depletion of glycogen stores. Hypoglycemia may be caused by vomiting, diarrhea, sepsis, hypothermia, or inadequate nutritional intake. Kittens with hypoglycemia will be weak and lethargic, and may be anorexic. Prolonged hypoglycemia may cause permanent brain damage, because the neonatal brain depends on glucose and carbohydrates as energy sources. If the kitten is not hypothermic or dehydrated, administer 5% to 10% dextrose orally at 0.25 to 0.50 mL/100 g body weight periodically by gastric tube until the kitten is stronger and normoglycemic. Then begin feedings of kitten milk replacer. Critically ill neonates may require a bolus infusion of 12.5% dextrose IV or IO (0.1 to 0.2 mL/100 g body weight, or more) followed by a constant rate infusion of 1.25% to 5% dextrose in a balanced electrolyte solution to prevent rebound hypoglycemia.[51,54] Hypertonic dextrose solutions should not be administered subcutaneously, because tissue sloughing may occur.

Dehydration occurs easily in neonatal kittens with diarrhea, vomiting, or reduced fluid intake. Neonates have poor compensatory mechanisms and immature renal function so that both dehydration and overhydration during treatment are concerns. Daily urine output in 1-month-old kittens is 25 mL/kg compared with 10 to 20 mL/kg in the adult cat.[54] Neonates also have higher fluid requirements than adults because of a higher total body water content (about 80% of body weight, compared with 60% in adults), a greater surface area-to-body-weight ratio, a higher metabolic rate, and decreased body fat. Hydration status may be difficult to assess in the youngest patients. Skin turgor is not a reliable test of hydration for kittens less than 4 weeks of age, because their skin has decreased fat and increased water content compared with adults. The kitten's mucous membranes should be moist and either hyperemic or pink. Pale mucous membranes and a decreased capillary refill time indicate at least 10% dehydration. Neonatal urine is normally colorless and clear; in dehydrated kittens, the urine is dark with a specific gravity greater than 1.020.

If the kitten is minimally dehydrated and normothermic with no gastrointestinal dysfunction, warmed electrolyte solution may be given orally or warmed subcutaneous (SC) fluids can be administered. However, subcutaneous absorption of fluids is slower in hypovolemic kittens because of peripheral vasoconstriction. If the kitten is moderately to severely dehydrated, IV fluid administration is the most effective. A mini-set (60 drops/mL) is used with a fluid or syringe pump or a burette. The cephalic or jugular vein can be catheterized with a 24-gauge ¾-inch or 22-gauge 1-inch catheter (Figure 41-10). Small-gauge catheters develop burrs easily during placement. Making a small hole in the skin with a 20-gauge needle first may facilitate placement. Balanced crystalloid solutions are the preferred fluid therapy choices. Lactated Ringer's solution is ideal for rehydration, because lactate can be used as an energy source; 1.25% to 5% dextrose can be added if necessary.

Warmed fluids may be given as a slow IV bolus of 1 mL/30 g body weight (30 to 45 mL/kg body weight), followed by a maintenance infusion of 80 to 120 mL/kg/day (8 to 12 mL/100 g) plus any ongoing losses.[46,51] It is important to monitor fluid therapy closely, because it is easy to overhydrate young kittens. Hydration status can be monitored by several methods, but weighing the kitten every 6 to 8 hours on an accurate gram scale is useful and easily accomplished. Other methods include serial packed cell volume (PCV)/total protein measurements, central venous pressure measurement, and urine output measurement with placement of a 3.5-Fr red rubber urinary catheter. Electrolyte and glucose status should also be monitored.

FIGURE 41-10 The jugular vein can be catheterized with a 24-gauge ¾-inch or 22-gauge 1-inch catheter. Lactated Ringer's solution is ideal for rehydration of neonatal kittens. *(Courtesy Jennifer Waldrop.)*

If it is difficult to achieve intravenous access, an alternate route for administration of fluids must be employed. The intraperitoneal route should not be used in neonatal kittens because of the risk of inducing peritonitis. Intraosseous access using the trochanteric fossa of the proximal femur is the best alternative to IV access; blood, fluids, and some medications can be administered in this way (Figure 41-11).[46] Contraindications to IO access are few, such as fracture or infection at the site. A 20- to 22-gauge 1-inch spinal needle or 18- to 25-gauge hypodermic needle may be used as a catheter.

The hair over the hip is clipped, and the skin is surgically prepared before the needle is inserted. The limb is positioned toward the ventral midline to rotate the trochanteric fossa laterally. The kitten's stifle can be supported in one hand while the needle is pushed and twisted into place. Resistance is felt initially as the needle penetrates bone. The spinal needle is firmly seated in the shaft of the femur, and the stylet is removed. If a hypodermic needle is used, the shaft may become clogged with bone debris. Placing a piece of surgical wire in the shaft of the hypodermic needle during placement will help avoid clogging.[49] If the needle still becomes clogged, it can be removed and another needle can be placed in the same location. A correctly placed catheter will move with the limb. Ideally, correct placement should be confirmed with a radiograph.

A T-port is inserted into the needle hub and the catheter is flushed with nonheparinized saline. If resistance

FIGURE 41-11 Placement of an intraosseous (IO) catheter in the femur is a quick way to administer fluids, whole blood, and some drugs. The IO catheter should be replaced with an intravenous catheter as soon as possible if ongoing fluid therapy is required. *(Courtesy Jennifer Waldrop.)*

to saline infusion is felt, rotate the needle 90 to 180 degrees to free the bevel from the wall of the femur. Fluid rates and amounts by the IO route are the same as for intravenous access. Administration can be with a syringe or a standard IV set. Use of cold fluids, rapid infusion, or hypertonic or alkaline solutions will cause pain. The IO catheter can be sutured in place with a stay suture through the skin for temporary use (typically less than 24 hours).[49] Bandaging IO catheters in small kittens is difficult and may not be necessary if excellent hygiene is maintained. IV access should be established as soon as possible. Once the kitten becomes more active, the catheter is easily dislodged. Complications of IO catheters include infection, extravasation of fluids, and bone and soft tissue trauma.

Blood transfusions may be necessary in some sick neonatal kittens, particularly those with anemia resulting from flea infestations or intestinal parasites. Indications for blood transfusion are weakness, tachycardia, pale mucous membranes, and a hematocrit less than 15%. Blood from a compatible typed donor is diluted 9:1 with a citrate anticoagulant and given using a Millipore blood filter by the IV or IO route at a rate of 20 mL/kg during a minimum of 2 hours.[46]

IMMUNITY

Queens have a colostral phase of lactation in which immunoglobulin concentrations are high, often exceeding their serum levels. The predominant immunoglobulin in both colostrum and milk is IgG. IgG concentrations are highest in the first 7 days of lactation and then steadily decrease, while IgA remains at a constant low level throughout lactation.[10] Kittens receive almost all their passive immunity during the first 12 to 18 hours of life (before gut closure) with the ingestion of colostrum; there is little or no transplacental transfer of immunoglobulins in the cat.[8] The amount of passively acquired immunity is determined by the amount of colostrum ingested, the time of ingestion, and the concentration of immunoglobulins in the colostrum. The kitten's serum IgG nadir is reached between 3 and 5 weeks of age because of catabolism of maternal IgG and correlates with a period of vulnerability to infection.[10,40] IgG levels then steadily increase as the kitten's own adaptive immunity develops.

Failure of passive transfer of immunity (FPT) is a well-documented concern in large animals and is diagnosed when neonates have serum IgG concentrations less than 400 mg/dL.[10] Several treatments, such as colostrum replacements, are commercially available for these species. Extrapolation from large animals would suggest that kittens with FPT may be at increased risk for infection. Although no studies on a correlation between FPT and susceptibility to infection in kittens have been published, neonatal sepsis is one of the most common causes of kitten mortality in the first few weeks of life. Kittens with uncorrected FPT start to produce IgG at about 4 weeks of age; they are therefore most vulnerable to infection from birth to at least 4 weeks of age. Adequate intake of colostrum during the first 18 hours of life may therefore be critical.

Many factors can influence whether a kitten ingests sufficient amounts of immunoglobulins, such as

- Birth order and litter size: In a large litter, kittens born later face increased competition to nurse.
- Suckling success: Weak kittens (e.g., due to low birth weight, effects of dystocia, congenital defects, disease) may not suckle effectively in the first day of life.
- Delayed ingestion: Queens recovering from cesarian section may not be well enough to nurse kittens effectively in the first day of life.
- Sick queens: An ill queen may not produce sufficient colostrum for the kittens, or it may be of poor quality with low IgG concentrations.
- Orphans: If the queen died during parturition or shortly afterward, orphan kittens may not have had an opportunity to ingest colostrum.
- Deliberate prevention of colostrum ingestion for prevention of neonatal isoerythrolysis: See below.

If a full history is not available for a kitten or litter, it may be difficult to know if FPT has occurred. No rapid assay for detection of FPT in cats is available. Definite diagnosis is by measurement of serum IgG concentration, a test only performed at some reference laboratories. One study evaluating surrogate markers for FPT in kittens found that adequacy of passive transfer correlated only with serum alkaline phosphatase activity, and then only for the first 2 days of life.[12]

A common recommendation for orphan kittens at risk of FPT is to foster the kitten on another lactating queen. However, colostrum-deprived kittens fostered on surrogate queens in the milk phase of lactation acquire insufficient levels of immunoglobulins.[10] Colostrum-deprived kittens fed milk replacer also fail to acquire sufficient levels of immunoglobulins.[10] Commercial milk replacers for kittens may be advertised as containing colostrum, but there is no evidence that the ingredient provides any immunity.

Correction of FPT can be accomplished by SC injection of adult cat serum from a cat with compatible blood type that has been screened for infectious diseases (15 mL/100 g body weight, divided into three doses during 24 hours).[40] This protocol requires collection of large volumes of blood and may be impractical, especially for more than one kitten at a time. Whether smaller amounts of adult cat serum may be effective in correcting FPT is not known. A commercially available equine IgG product licensed for treatment of FPT in foals was evaluated in kittens with FPT in an effort to find a more readily available solution.[11] Although treated kittens achieved adequate serum concentrations of the equine IgG, the equine antibodies failed to promote bacterial phagocytosis by feline neutrophils in vitro. Therefore equine IgG cannot be recommended for treatment of FPT in kittens.

BOX 41-6

Common Pathogens of Kittens

Upper and Lower Respiratory Tract Disease

Feline herpesvirus-1
Feline calicivirus
Bordetella bronchiseptica
Mycoplasma spp.
Chlamydophila felis

Gastrointestinal Tract

Parvovirus
Coliform bacteria
Tritrichomonas foetus
Giardia spp.
Isospora spp.
Ancylostoma spp.
Toxocara spp.

Systemic Disease

Feline leukemia virus
Feline immunodeficiency virus
Feline infectious peritonitis
Toxoplasma gondii
Gram-positive bacteria (e.g., *Streptococcus* spp., *Staphylococcus* spp.)
Gram-negative bacteria (e.g., *Escherichia coli, Salmonella* spp.)

INFECTIOUS DISEASES

The highest kitten mortality rates from infectious diseases are in the first 2 weeks of life and in the postweaning period. Common pathogens include those causing upper and lower respiratory tract disease, diarrhea, and systemic disease (Box 41-6).

Respiratory Tract Disease

Respiratory tract disease can cause significant morbidity and mortality in young kittens. Clinical signs in acute upper respiratory tract infections include fever, sneezing, depression, anorexia, and bilateral nasal and ocular discharge (mucoid, serous, or purulent) (Figure 41-12). Severe disease is characterized by dehydration, debilitation, and even death. Most infections involve feline herpesvirus (FHV-1), feline calicivirus, *Mycoplasma* spp., and/or *Chlamydophila felis*. Pathogens implicated in lower respiratory tract disease in kittens include *Bordetella bronchiseptica* and *Streptococcus canis* (see below). Treatment of respiratory tract disease in kittens is largely symptomatic and supportive, and aggressive nursing care is often required. The diagnosis and management of respiratory pathogens is found in Chapter 30.

FIGURE 41-12 Conjunctivitis resulting from feline herpesvirus is common in young kittens. Clinical signs include fever, sneezing, and bilateral nasal and ocular discharges.

Ophthalmia neonatorum is conjunctivitis under the closed eyelids of neonatal kittens (Figure 41-13). Commonly implicated pathogens include FHV-1 and *Chlamydophila felis*.[47] Other possible pathogens include *Staphylococcus* spp., *Mycoplasma* spp., and *Bordetella*

FIGURE 41-13 Ophthalmia neonatorum is conjunctivitis under the closed eyelids of neonatal kittens. Commonly implicated pathogens include feline herpesvirus and *Chlamydophila felis*. *(Courtesy Sandra Brau.)*

bronchiseptica. The eyelids are swollen from the accumulation of purulent material underneath, some of which may be discharging at the medial canthus. Drainage must be established by opening the eyelids using warm compresses and gentle traction. If this approach fails, the closed tip of a small mosquito forceps may be inserted at the medial canthus and gently opened to separate the eyelids. The forceps should never be returned to the closed position while inserted between the eyelids to avoid damage to ocular structures. Sharp instruments should never be used to separate the eyelids. The eyes should then be flushed with saline and the cornea should be stained with fluorescein for evidence of ulceration. A broad-spectrum topical antibiotic ointment should be applied 2 to 4 times daily for 1 week. When FHV-1 is suspected, topical antiviral therapy may be useful (see Chapter 29). Symblepharon (conjunctival adhesions) may be a sequel to severe or untreated cases.

Diarrhea

Although diarrhea in kittens is a common and frustrating condition facing clinicians, there is very little published research on the specific causes and treatments in this age group. Acute diarrhea is often self-limiting and may be managed with symptomatic and supportive therapy. For chronic diarrhea, a specific diagnosis should be made and treatment should be targeted (see Chapter 23). Inappropriate use of antibiotics to treat diarrhea should be discouraged. Indiscriminate use of antibiotics in young kittens may alter the commensal intestinal microflora, worsen the diarrhea, and induce antibiotic resistance. The most common causes of diarrhea in kittens are infectious agents, primarily parasites and viruses (see Box 41-6). Recently, enteroadherent *Enterococcus* spp. infection has been identified as a cause of diarrhea and failure to thrive in kittens.[58]

A comprehensive fecal examination is an important first step in diagnosis (see Chapter 23). Fecal examination should include a direct fecal smear (wet mount), a stained smear, and fecal flotation (zinc sulfate centrifugation with fresh feces). The next level of diagnostics would include immunoassays or polymerase chain reaction (PCR) testing for *Giardia* spp. and *Tritrichomonas* spp. The indications for performing fecal enteric panels on kittens with diarrhea are not well defined. Bacterial enteropathogens and toxins are commonly found in asymptomatic kittens as well as those with diarrhea, making interpretation difficult.[25a] Fecal cultures and toxin analysis are probably best reserved for specific situations, such as kittens with bloody diarrhea and evidence of sepsis. In general, for kittens with chronic diarrhea where no diagnosis has yet been made and response to therapy is poor, repeating previously negative diagnostic tests is more rewarding than intestinal biopsy (either by laparotomy or endoscopy).[48] Finding an inflammatory response on intestinal biopsy in kittens does not ensure a diagnosis of inflammatory bowel disease.

It is acceptable to administer a broad-spectrum anthelminthic to kittens with diarrhea even in the face of negative fecal examinations. Pyrantel pamoate (5 to 10 mg/kg, PO) can be started as young as 2 weeks of age. Most anthelminthics for kittens are labeled for use from 8 weeks of age, although a few are labeled from 6 weeks of age (e.g., Interceptor; Novartis Animal Health, Greensboro, NC). Another common empiric treatment is metronidazole (7 to 10 mg/kg, PO, every 12 hours for 5 days). This drug may resolve clinical signs by several mechanisms, such as altering intestinal microflora, moderation of cell mediated immunity, or treatment of a specific pathogen. The use of prebiotics and probiotics is receiving attention, although no studies on efficacy in kittens with diarrhea are yet available. In addition, there is little regulation or mandatory quality control for most commercial products, and label descriptions do not always match the contents.[81] Finally, some kittens with chronic diarrhea may develop vitamin B_{12} (cobalamin) deficiency. Empiric treatment can be safely instituted at 100 μg/kitten, SC, once weekly for 4 to 6 weeks.[48]

External Parasites

Fleas are the most common and most important external parasite affecting kittens (see Chapter 22). Severe flea infestations can lead to anemia and even death. Many effective flea control products are available, but most are labeled for use in kittens from 8 weeks of age. Exceptions include lufenuron (Program, Novartis Animal Health), which is labeled for use from 6 weeks of age, and nitenpyram (Capstar, Novartis Animal Health), which is labeled for use in kittens that are 4 weeks of age and weigh at least 0.9 kg (2 lb). Fipronil spray is labeled for use on kittens from 2 days of age in some countries, such as the United Kingdom. When treating very small kittens, it is safer to apply the spray to a cloth or cotton balls and wipe the kitten, avoiding the eyes and mucous membranes. Very young kittens should be flea-combed daily. Most pyrethrin flea shampoos are not labeled for use in kittens less than 12 weeks of age, and their efficacy is questionable. Anecdotally, hand dishwashing detergents are often used, although care must be taken to avoid hypothermia when bathing neonates. Many flea treatment products are safe for nursing queens (see Table 22-1); so, the queen and the environment should also be treated.

Neonatal Sepsis

Neonatal sepsis is a significant cause of death in kittens, and may be caused by a variety of pathogens. Risk factors may include failure of passive transfer of immunity, mastitis or metritis in the queen, prolonged delivery or dystocia, low birth weight, chilling because of low environmental temperature, and inappropriate use of antibiotics.

Pathogens that may be associated with neonatal sepsis include *E. coli*, *Staphylococcus* spp., *Pasteurella* spp., *Enterobacter* spp., *Clostridium* spp., *Salmonella* spp., and *Streptococcus* spp. These organisms typically enter through the umbilicus, but other points of entry include the gastrointestinal tract, the respiratory tract, and the urinary tract. Clinical signs will vary with the organ system involved, although death can occur quickly with few signs. Common syndromes include gastroenteritis (vomiting, diarrhea, dehydration), pyelonephritis (fever, dehydration, hematuria), and pneumonia (respiratory distress, cyanosis). Noncardiogenic pulmonary edema secondary to sepsis can lead to acute respiratory distress. Generic clinical signs associated with sepsis include failure to thrive, crying, restlessness, and hypothermia.

A diagnosis of neonatal sepsis is suspected based on clinical signs. Laboratory diagnostics may show anemia, neutrophilia, and hypoglycemia. A definitive diagnosis in the living animal requires blood or urine culture, but is rarely of use to the affected kitten because of the length of time required before culture results are received. However, culture results may help treat litter mates that also become ill. Therefore empiric treatment with a broad-spectrum antibiotic, such as a cephalosporin, is justified pending culture results. Unfortunately, a definitive diagnosis of neonatal sepsis is often made at necropsy. In addition to appropriate antibiotic therapy, treatment is mainly aggressive supportive care (Box 41-7). Antibiotics recommended for use in young kittens include the quinolones and beta-lactams (Table 41-6). More information on drug therapy for neonates in found in Chapter 4.

BOX 41-7

Summary of Therapeutics for Neonatal Septicemia in Kittens

Treat Dehydration

Fluid therapy through intravenous, intraosseous routes
Warmed balanced electrolyte solution (BES) with 1.25% to 5% dextrose
Slow bolus: 1 mL/30 g body weight
Maintenance rate: 80 to 120 mL/kg/day plus ongoing losses

Treat Hypoglycemia

PO: 0.25 to 0.50 mL/kg, 5% to 10% dextrose
IV bolus: 0.1 to 0.2 mL/100 g, 12.5% dextrose
IV CRI: 1.25% to 5% dextrose in BES

Treat Hypothermia

Rewarm during 30 minutes to 2 hours to maximum rectal temp of 101° F (36.3° C)
Environmental temperature for neonates should be 27° C to 32° C (80° F to 90° F)

Treat Infection

Start broad-spectrum empiric antibiotic therapy pending culture results

Monitor Treatment

Hydration: weigh every 6 to 8 hours, serial PCV/TP, central venous pressure, urine output, respiratory rate/effort, auscult chest
Serial electrolyte, glucose measurements
Rectal temperature every hour

CRI, Constant rate infusion; *PCV,* packed cell volume; *TP,* total protein.

Streptococci have been associated with severe infections in kittens, including sepsis.[27] They are typically beta-hemolytic infections, caused by the Lancefield group G organism, *Streptococcus canis*. Streptococci are commensal microflora of the feline skin, pharynx, upper respiratory tract, and genital tract. Neonatal kittens acquire infection from the vagina of the queen. The bacteria gain entrance through the umbilical vein, causing omphalophlebitis, peritonitis, and sepsis. A toxic shock–like syndrome has been reported in 8-week-old kittens,[80] and cervical lymphadenitis may be seen in kittens from 3 to 7 months of age. Untreated or undetected cases of lymphadenitis may progress to myocarditis or pneumonia (Figure 41-14). *S. canis* has also been associated with high-mortality disease outbreaks in shelters where

TABLE 41-6 Antibiotic Choices for Neonatal Kittens

Drug	Dose
Amoxicillin	6 to 20 mg/kg, q12h, PO
Amoxicillin + clavulanic acid	12.5 to 25 mg/kg, q12h, PO
Ampicillin	25 mg/kg, q8h, IV/IO/IM
Ceftiofur	2.5 mg/kg, q12h, SC; maximum 5 days
Cephalexin, cefazolin	10 to 30 mg/kg, q12h, PO
Enrofloxacin	2.5 to 5 mg/kg, once daily, SC or IV
Ticarcillin + clavulanic acid	15 mg/kg, q12h, IV or IM
Trimethoprim sulfa	30 mg/kg, once daily, PO

IM, Intramuscular; *IO,* intraosseous; *IV,* intravenous; *PO,* by mouth; *SC,* subcutaneous.

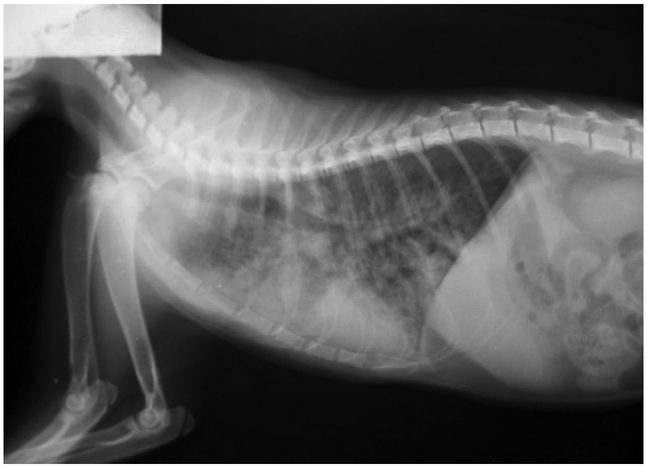

FIGURE 41-14 *Streptococcus canis* may be a cause of severe pneumonia in kittens.

affected cats may have skin ulceration, necrotizing sinusitis and meningitis, necrotizing fasciitis, toxic shock–like syndrome, and sepsis.[63]

Kittens born to queens less than 2 years of age are most at risk of streptococcal infections. Young queens maintain a carrier state with high numbers of bacteria in the vagina throughout pregnancy. Often, more than one kitten in a litter is affected, and mortality rates in the first 2 weeks of life may be high, especially when the organism is first introduced into a naïve population, such as a breeding cattery. Affected kittens fail to gain weight and may have an infected umbilicus. They may be found dead with minimal ante mortem clinical signs. Diagnosis may be made by culture of umbilical exudates or at necropsy, when the organism is most often isolated from liver, lung, umbilicus, and peritoneal cavity.

Kittens with lymphadenitis or omphalophlebitis should be treated with antibiotics and drainage of abscesses. Kittens with sepsis are treated with antibiotics and aggressive supportive care (see Box 41-7). *S. canis* is very sensitive to penicillins. Litter mates of affected kittens may be treated with oral amoxicillin. Prevention of infection in newborn kittens may be accomplished by disinfection of the umbilical cord in 2% tincture of iodine or 0.5% chlorhexidine solution at birth. In an endemic situation, routine antibiotic treatment of queen and kittens may reduce morbidity and mortality.[4] At the time of delivery, kittens are given a single injection of a combination benzathine penicillin and procaine penicillin product (both 150,000 IU/mL). The penicillin is diluted 1:6 with sterile 0.9% saline and administered at 0.25 mL/kitten, SC. The queen is given a single injection of the same undiluted product (1.0 mL/cat, SC).

NEONATAL ISOERYTHROLYSIS

Blood type is determined by antigenic, species-specific markers on the surface of red blood cells. In cats, blood type is characterized by an AB system.[2] It is believed that one gene with three alleles produces blood types A, B, and the rare AB. Blood type A is caused by the dominant allele and is found in 95% to 98% of nonpedigreed cats. Cats with blood type A have low titers of naturally occurring anti-B antibodies. Blood type B is recessive, and prevalence varies with geographic location and breed (see Tables 25-1 and 25-2).[23,24] Certain cat breeds have a high prevalence of blood type B individuals, such as the British Shorthair, Cornish and Devon Rex, and Birman. Cats with blood type B have high, *naturally* occurring titers of anti-A antibodies. No previous pregnancy or transfusion is necessary to induce antibody formation.

Neonatal isoerythrolysis (NI) may occur in kittens with blood type A or AB born to a queen with blood type B.[6] The queen's colostrum contains anti-A antibodies that are transferred when the kittens ingest colostrum. Kittens are typically born healthy, but stop nursing within a few days with suggestive clinical signs, such as failure to thrive, pigmenturia, icterus, and anemia. Affected kittens may be transfused with washed type B red cells, preferably from the queen, because she will not have antibodies against her own red blood cells.[46] The red cells are suspended in saline and administered at 5 to 10 mL/kitten, IV or IO, during a period of several hours. Despite aggressive intervention, the mortality rate is high.

Prevention of NI in breeding programs necessitates knowledge of the blood types of breeding cats in those breeds with a significant percentage of type B individuals. Breeders may choose to breed queens with blood type B only to males with blood type B to avoid producing kittens at risk of NI. In some cases, queens with blood type B must be bred with a male of blood type A or AB, and steps should be taken to prevent NI. Breeders can remove all the kittens from the queen immediately after birth for the first 18 hours of life to prevent ingestion of colostrum. The kittens are hand-fed milk replacer and cared for by the breeder until they are returned to the queen. Alternately, if the breeder can be present at the time of birth, patient-side blood typing cards (RapidVet-H, DMS Laboratories, Flemington, NJ) may be used to type umbilical cord blood from the kittens before they have a chance to nurse. Care must be taken to avoid cross-contamination with blood from the queen in birth fluids. Only kittens at risk of NI (blood type A or AB) are then removed from the queen for 18 hours, and kittens with blood type B are allowed to remain with the queen.

ORPHAN KITTENS

Veterinarians may be asked to evaluate and treat young kittens that have been orphaned or abandoned. Successful treatment can be accomplished with knowledge of the particular needs of these often fragile pediatric patients. If a foster queen is not available, the caregiver must replace physiologic needs normally provided by the queen, such as warmth, nutrition, elimination, sanitation, and social stimulation. The ideal housing for orphan kittens is an incubator, but any warm and safe enclosure will suffice, such as a pet carrier or cardboard box. Bedding should be absorbent, soft, warm, and either readily cleaned or disposable. The environmental temperature for kittens in the first week of life should be 30° C to 32° C (86° F to 90° F). The temperature can be gradually lowered to 24° C (75° F) during the next few weeks. If a heat source is used in a box or carrier, it should be placed so that a temperature gradient is created, allowing the kittens to move away from the warmest areas when needed. Humidity should be maintained at 55% to 60% to prevent dehydration and maintain the health of mucous membranes.

If a litter of kittens is orphaned, they will often try to nurse on each other. Skin trauma or genital trauma, especially of the penis and prepuce, may occur if the kittens are not separated. Social stimulation should be provided by regular but brief periods of handling. Protection against infectious diseases is important for orphaned kittens, especially if failure of passive transfer of immunity is possible (see above). Although it is tempting to expose young orphans to older kittens or adults for social interaction, this should be avoided until the kitten is immunized. All bedding and equipment should be kept clean and caregivers should wash hands before handling neonates.

Certain procedures should be followed for feeding orphans.[43] Orphan kittens should be fed a commercial milk replacer specifically designed for kittens to approximate the composition of the queen's milk. Homemade formulas are best reserved for short-term or emergency use. The manufacturer's directions should be followed for mixing, storage, and feeding quantities. Strict hygiene is necessary, and if milk replacer must be reconstituted, no more than a 48-hour supply should be prepared at a time. The reconstituted milk replacer can be divided into individual feedings and refrigerated until use. The energy requirement for kittens in the first few weeks of life is approximately 20 kcal metabolizable energy (ME)/100 g body weight/day.[14] The maintenance water requirement is about 180 mL/kg per day (range, 130 to 220 mL/kg/day).[35,46] Initially, only 50% of the recommended amount of milk replacer should be fed to avoid inducing diarrhea. Extra water or an oral electrolyte solution can be added to make up volume and provide fluid needs. Over several feedings, the concentration of the milk replacer can be increased to that recommended by the manufacturer.

Milk replacer should be warmed to 35° C to 38° C (95° F to 100° F) by immersing the container in a warm-water bath. Never microwave milk replacer, because overheating or uneven heating may result. Vigorous orphans with a good suck reflex may be bottle-fed or syringe-fed (Figures 41-15 and 41-16) while in sternal recumbency with the head elevated, simulating a normal nursing position. The hole or slit in the nipple should be made large enough to allow a drop of milk to form when the bottle is held upside down. A drop of milk is expressed from the bottle on the kitten's tongue to help initiate feeding. Milk should never be forced out of the bottle while it is in the kitten's mouth to avoid aspiration. Care should be taken to ensure no air is ingested while bottle-feeding.

Weaker kittens are best fed with a gastric tube (Figure 41-17).[46] Tube feeding is also efficient if more than one kitten must be hand-raised. Feeding tubes should be selected according to the size of the kitten: 5 Fr for kittens weighing less than 300 g, 8 Fr for kittens greater than 300 g. Measure from the tip of the kitten's nose to just before the last rib, and mark this position on the feeding

FIGURE 41-15 Orphan kittens can be fed milk replacer using a syringe.

FIGURE 41-16 Orphan kittens that are otherwise healthy with a good suck reflex can be fed milk replacer from a pet nurser bottle. *(Courtesy Richard Young.)*

FIGURE 41-17 Weaker orphan kittens, or litters of orphan kittens, can be easily fed using a gastric tube. A size 5-Fr soft red rubber feeding tube is used for the smallest kittens.

tube. The tube will have to be remeasured and marked weekly as the kitten grows. With the kitten in a sternal position, the lubricated tube should pass easily down the left side of the mouth into the esophagus and is advanced to the mark. If resistance is felt or coughing occurs, the tube should be removed and repositioned. Kittens do not have a gag reflex until about 10 days of age. Proper placement of the tube can be confirmed by instilling a small volume of saline first and assessing the response. The milk replacer is drawn up in a 3- or 10-mL syringe that is then attached to the feeding tube. The tube is filled with milk replacer warmed to about 38° C (100° F). The milk replacer is slowly infused for several minutes. Before withdrawing, the tube should be kinked to prevent aspiration of formula. Avoid overfeeding; the maximum stomach capacity for kittens is about 4 to 5 mL/100 g body weight.[46] All tube feeding equipment should be cleaned thoroughly after use.

Kittens should be fed every 2 to 4 hours during the first week of life, and then every 4 to 6 hours until weaning. Diarrhea is the most common problem seen in kittens fed milk replacer. It can be treated by temporarily reducing the amount fed and by diluting the formula by 50% with water or oral electrolyte solution for a few feedings. Orphan kittens should be weighed every 12 hours in the first 2 weeks of life and at least daily thereafter to ensure nutrition is adequate to support growth. Daily records should be kept of weight, feedings, elimination, and general behavior. Identification of individuals in a litter is important for monitoring weight and health, and if kittens appear similar, different colors of nail polish can be applied to the claws for differentiation. Orphans less than 3 weeks of age must have the anogenital area stimulated after every feeding to induce defecation and micturition. At least twice a week, orphan kittens should be cleaned gently with a soft moistened wash cloth.

At 3 to 4 weeks of age, kittens can be taught to drink milk replacer from a shallow saucer. Then solid food can be introduced by mixing a small amount of canned kitten food with milk replacer and offering it for 30 minutes at a time, several times per day. Once the kitten has learned to eat from a saucer, the amount of formula fed can be slowly decreased until only solid food is being ingested. By 5 to 6 weeks of age, kittens are able to chew dry food. Weaning is usually completed by 6 to 8 weeks of age.

PEDIATRIC SPAY AND NEUTER

Although elective gonadectomy is one of the most common veterinary surgeries performed in North America, little data exist to suggest the optimal age.[37] Prepuberal gonadectomy refers to spay or neuter before the onset of puberty, which may occur in female cats between 4 and 21 months of age and in male cats between 8 and 10 months of age. Early spay and neuter (ESN) refers to gonadectomy between 6 and 16 weeks of age and is now more commonly practiced as veterinarians gain experience with pediatric anesthesia and surgery. ESN is one useful approach for control of pet overpopulation, because it enables shelters and pedigreed breeders to perform pre-adoption/pre-sale gonadectomy and avoids the risk of owner noncompliance with spay/neuter contracts. Increasingly, it is recognized there are also health benefits to ESN. Several veterinary organizations support ESN, such as the American Veterinary Medical Association, the Canadian Veterinary Medical Association, the International Society of Feline Medicine, and the Winn Feline Foundation.

Veterinarians have varying attitudes about when to spay and neuter cats. In one survey, small animal veterinarians in New York state were asked about their beliefs and practices regarding gonadectomy and their attitudes to surgery performed before 4 months of age.[73] The majority (70%) recommended gonadectomy for all owned animals, and 90% supported routine gonadectomy of shelter animals before adoption. Most respondents believed shelter animals should be gonadectomized at an earlier age than owned animals. The minimum age reported for gonadectomy ranged from 1 to 12 months. Just more than one third of respondents believed the earliest age for gonadectomy of owned animals should be at or more than 6 months of age. Opinions were affected by variables that included the veterinarian's age, sex, and date of graduation.

In a recent U.K. study, a questionnaire designed to obtain data on the age at which gonadectomy was recommended for cats was sent to over 4,000 veterinarians.[55] Information was also collected on the perceived advantages and disadvantages of gonadectomy in cats between 8 and 20 weeks of age. The mean age recommended for gonadectomy among respondents was 22.6 weeks, despite the fact that the British Small Animal Veterinary Association recommends the surgery be performed at approximately 16 weeks. Just more than one half of veterinarians believed gonadectomy should not be performed before 6 months of age. Only 28% of veterinarians believed it was appropriate to gonadectomize cats between 12 and 16 weeks of age. Beliefs were influenced by several factors, such as time since graduation, perceptions about cat overpopulation, and perceptions about surgical and anesthetic risks.

In addition to avoiding unwanted litters of kittens, the benefits of ESN include safe anesthetic and surgical techniques, shorter surgical and recovery times, and avoidance of the stresses and costs associated with spaying female cats while in estrus, pregnant, or with pyometra.[28] In addition, the risk of mammary adenocarcinoma is reduced by 91% when female kittens are spayed before 6 months of age.[61] Objections to ESN include concerns about effects on growth, increased fracture risk, obesity, behavioral changes, increased disease risk, and safety of anesthesia and surgery in pediatric patients. Several long-term studies have been performed to assess health risks of ESN. These studies confirm that ESN is not associated with any increased risk of disease, but rather is associated with a lower risk of some diseases, such as asthma, gingivitis, and mammary carcinoma.[34,61,74]

The most persistent health concerns associated with ESN are potential effects on urinary tract health. Lower urinary tract disease in cats is a diverse collection of conditions caused by a wide variety of factors, such as diet, water intake, and stressors. The diameter of the male urethra is no smaller in ESN cats than in intact cats.[67] Age at castration does not influence risk of urinary tract disease; in fact, one study showed a decreased risk of lower urinary tract disease in ESN male cats.[74]

Testosterone and estrogen assist maturation of the physes in long bones. Growth stops when physeal closure occurs. Intact cats have distal radial physeal closure at 1 year of age or older. Cats altered at 7 weeks and 7 months of age had distal radial physeal closure about 8 weeks later than intact cats.[69,77] The effect of this delay in physeal closure is unknown, but adult size in cats is not significantly affected by age at altering. It has been suggested that delayed physeal closure may predispose cats to Salter fractures of the femoral capital physes. The femoral capital physis normally closes between 7.5 and 10 months of age. Other risk factors for this type of fracture include obesity and sex/reproductive status (neutered males are at highest risk).[53] The risk of fracture would be the same for cats altered at any age that results in delayed physeal closure, not just cats spayed or neutered at an early age. These fractures appear to be rare in the general cat population. One large study failed to find any association between ESN and physeal fracture risk in cats.[74] Given the development of unwanted sexual behaviors in both male and female cats after puberty, it is not feasible for most owners to delay surgery until after physeal closure (e.g., after 10 months of age), and risk of other diseases such as mammary carcinoma would be increased.

Obesity is a multifactorial problem involving diet, exercise, age, and other factors. Gonadectomized cats have a lower metabolic rate than sexually intact cats regardless of the age at surgery. Castrated male cats require 28% less calories than intact male cats, and spayed female cats require 33% fewer calories than intact female cats.[68] However, one study found a significant increase in food intake in male and female cats after gonadectomy.[16] Therefore when cats are allowed to eat the same amount of food after surgery, or food intake increases because of surgical sterilization, weight gain will inevitably occur. Clients should be counseled on dietary and exercise needs for altered cats before surgical sterilization to avoid obesity.

Compared with gonadectomized cats, sexually intact cats show less affection to humans and more aggression to other cats. An early study showed that ESN male cats are less aggressive to veterinarians and exhibit fewer problems with urine spraying.[77] Other long-term studies have shown there is no difference in the prevalence of serious behavior problems based on age at surgery, although hiding and shyness (at a level not considered serious enough by owners to relinquish the pet) may be more common in cats gonadectomized at less than 5.5 months of age.[74,84] In fact, behavior problems are common in most kittens in the first month after adoption, emphasizing the importance of early owner counseling.[84]

Pediatric patients have unique perioperative, anesthetic, and surgical issues.[36] Kittens should have a complete physical examination as well as at least the first vaccination and treatment for parasites if in a shelter or rescue situation. Client-owned cats can be scheduled for surgery following the last vaccination at approximately 16 weeks. The minimum laboratory data that should be collected before surgery includes packed cell volume and total solids, blood glucose and blood urea nitrogen, and urine specific gravity. Postpone surgery if any illness or abnormality is found. Anesthesia and surgery do not affect response to vaccination so that kittens can be vaccinated at the time of surgery if required.[42]

With the use of safe and effective techniques, it has been shown that ESN does not increase morbidity or mortality associated with anesthesia and surgery.[1] In fact, in one study, kittens gonadectomized at less than 12 weeks had lower postoperative complication rates than those that had surgery at more than 23 weeks of age.[32] Surgical benefits of ESN include less bleeding, improved visualization of organs, shorter surgery times, and more rapid recoveries. Many veterinarians are comfortable with a 2-lb (1-kg) rule as a minimum weight for pediatric spay/neuter patients.

Pediatric patients distribute and metabolize drugs differently; so, the clinician must be careful with drug selection and doses. Weigh each kitten to the nearest 100 grams and calculate drug doses carefully. Renal and hepatic function does not reach mature levels until about 3 to 4 months of age. Certain anesthetic concerns must be addressed:

- Maintenance of breath rate is important because pediatric patients have limited ability to respond to elevated carbon dioxide in blood or tissues and have little oxygen reserves in the lungs; yet they have high oxygen needs (2 to 3 times that of adult cats). Use anesthetic equipment with minimal dead space and resistance.
- Cardiac output is believed to be dependent on heart rate, and there is poor tolerance for both volume overload and hypotension.
- Kittens are less able to compensate for blood loss than adults; therefore care should be taken when handling tissues and blood vessels. Even small volumes of blood loss may result in clinical anemia in kittens less than 8 weeks of age.

Hypothermia occurs easily because of the greater surface area to volume ratio of the neonate, less subcutaneous fat, and reduced ability to shiver. Hypothermia can cause bradycardia and prolonged recovery from anesthesia. The preparation, surgery, and recovery areas should be kept warm. Ensure kittens are never placed on cold metal surfaces. Use warmed towels, circulating water blankets, or other methods to maintain body temperature. Plastic "bubble pack" wrapping is also useful for maintaining warmth. Warm the surgical preparation solutions, and replace alcohol with sterile saline. Minimize the amount of hair coat that is clipped, and the surgical incision should be appropriate for the size of the patient to reduce the risk of hypothermia. Rectal temperature should be monitored during surgery and postoperatively.

Hypoglycemia occurs easily in neonates because of small hepatic glycogen reserves; so, the youngest patients should not be fasted for more than 1 to 2 hours before anesthesia. Water should never be withheld. Within 1 hour of recovery, patients should be offered a small meal. Kittens unwilling to eat after 1 hour or more can be given oral dextrose to prevent hypoglycemia.

Equipment suitable for pediatric gonadectomy is simple and includes

- Cuffed/noncuffed endotracheal tubes, 2 to 3.5 mm
- Clear, snug face mask
- Kit with small surgical instruments
- Stainless steel hemostatic clips for ligation if desired
- Absorbable suture material, such as 4-0 or 5-0 Vicryl (Johnson & Johnson; New Brunswick, NJ) or chromic gut; nylon may be used for closure of linea alba

Stress and anxiety may lead to unpredictable results of premedication and anesthesia. Decrease stress by keeping litter mates together before surgery in a warm quiet environment, minimize patient handling, avoid IV injections, and reunite litters of kittens as soon as possible after recovery. Certain anesthetic drugs should be avoided in pediatric patients. Xylazine may cause bradycardia and decreased cardiac output. Thiobarbiturates require IV administration, which is more stressful than other routes of administration. These drugs are also protein-bound, and neonates are easily saturated, because they have lower plasma protein levels than adult cats.

Several drugs and drug combinations using inhalant anesthesia are safe and effective for pediatric anesthesia.[15,32,36] Brief mask induction may be required with some protocols, then maintenance on isoflurane or sevoflurane (intubate females, use a snug-fitting mask for males). Examples of suitable protocols include

- Midazolam (0.22 mg/kg IM), ketamine (11 mg/kg IM), butorphanol (0.2 to 0.4 mg/kg IM), ±glycopyrrolate (0.011 mg/kg IM)
- Ketamine/diazepam (0.1 mg/kg IM of 1:1 mixture), butorphanol (0.2 to 0.4 mg/kg IM), ±glycopyrrolate (0.011 mg/kg IM)
- Tiletamine/zolazepam (11 mg/kg IM) for castration of male kittens
- Acepromazine (0.055 mg/kg IM), butorphanol (0.22 mg/kg IM), glycopyrrolate (0.011 mg/kg IM)
- Injectable only protocol for both spays and neuters: medetomidine (40 μg/kg), ketamine (20 mg/kg)

and buprenorphine (20 μg/kg), combined and given subcutaneously[65]; atipamezole (0.5 mg IM) is given at the end of surgery

Anesthetic monitoring is necessary and should include assessment of level of anesthesia, color of mucous membranes, Doppler monitoring of heart rate and blood pressure, pulse oximetry, and breath rate. Prolonged recovery is most often caused by hypothermia, but can also be caused by residual effects of drugs or hypoglycemia. Corrective measures for prolonged recoveries include warming, using reversal agents if available, and providing 50% dextrose for hypoglycemia.

Certain surgical concerns must be addressed.[33] Meticulous hemostasis is necessary because tissues are more friable than in adults; handle tissues gently and avoid the use of spay (Snook) hooks in young female kittens. For males, perform closed castration through a single or double scrotal incision, ligate the spermatic cord with absorbable suture or use hemostatic clips. Leave the scrotal incision open. For females, the ovariohysterectomy or ovariectomy technique is the same as for a mature cat. Close the skin with subcuticular sutures or tissue adhesive and avoid skin sutures. A small amount of serous fluid in the abdomen of kittens is normal.

Postoperative analgesia for spays can be addressed with splash blocks. One suggested protocol mixes two parts 0.5% bupivacaine, one part 2% lidocaine, and one part 0.9% NaCl.[28] Splash 0.22 mL/kg onto the incision after closure of the linea alba but before closure of the subcutaneous tissue and skin. A single postoperative dose of meloxicam (0.1 to 0.2 mg/kg, SQ) may be used for kittens more than 6 weeks of age; butorphanol or buprenorphine are also good analgesic choices.

ESN surgeries are safe and easily performed. Providing routine surgical sterilization for kittens less than 6 months of age also allows clinicians to become confident and proficient with anesthesia, surgery, and supportive care that can be applied to other surgical situations in this age group.

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