CONTINUING EDUCATION

Pediatric Emergencies

The neonatal stage is a major risk period in dogs, as around 20% of live-born puppies die before they are 21 days old, with 70% of those deaths being in the first week postpartum.[1,2] A similar mortality rate is expected in kittens.[1,2] The Apgar scoring system is used to evaluate newborn humans but is not commonly used for newborn puppies and kittens. Other methods are used in veterinary medicine , including the neonatal resuscitation form used at the University of Pennsylvania Ryan Veterinary Hospital. Parameters measured using this scoring system include respiratory effort, heart rate, muscle tone, response to stimulation, and mucous membrane color **(BOX 1)**.

The terms *neonate* and *pediatric* tend to be used interchangeably, but cats and dogs are normally defined as being neonates for the first 7 to 14 days of life. Neonates have poor neurologic function and are completely dependent on the dam or queen, as their auditory and visual senses, along with spinal reflexes, are continuing to develop. The term *pediatric* is used to describe animals between 2 and 6 weeks of age,[4] although some texts describe pediatric patients as being between 2 weeks and 6 months of age.

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PHYSIOLOGIC CHARACTERISTICS OF PEDIATRIC PATIENTS

Neonatal and pediatric patients differ significantly from their adult counterparts. Veterinary nurses and technicians must understand these unique physiologic differences and how they affect diagnosis and treatment (TABLE 1).

Thermoregulatory Mechanisms

Normal body temperature in neonates is 96°F to 97°F at birth, rising to 100°F by 4 weeks of age. By the time of weaning, rectal temperature approaches that of adults. Thermoregulation in neonates is difficult because they are unable to shiver and show poor peripheral vasoconstriction in response to hypothermia. They also lack fully developed organs (liver) that contribute to cellular metabolism and produce heat. Neonates also have little body fat and poor blood flow to the periphery and lack the ability to pant, additional factors that make them unable to respond properly to hyperthermia.

Cardiorespiratory System

In the fetal circulatory system, blood is shunted past nonfunctioning lungs via the ductus arteriosus, which is located between the left pulmonary artery and ascending aorta. During intrauterine life, fetal respiration is through a blood–gas exchange process across the placenta. In the last days before birth, production of surfactant in the lungs is stimulated. When the umbilical cord is separated at birth, the respiratory and cardiovascular systems undergo numerous changes. Umbilical circulation stops, resulting in severe hypoxia. At the same time, peripheral resistance in the

TABLE 1
Common Pediatric Emergencies

CONDITION	THERAPY
Anorectic	Nutritional supplementation
Hypoglycemia	■ IV glucose or dextrose boluses (0.5–1.5 mL/kg IV of 50% concentration diluted 1:1–1:2, or 2–4 mL/kg of a 10% solution ■ If IV access is not available, oral glucose can be administered ■ Isotonic fluids supplemented with 2.5%–5% glucose as a constant-rate infusion can also be used
Hypoxia	■ Oxygen supplementation ■ Reversal of any agents likely to cause respiratory depression: for opioids, naloxone; for benzodiazepines, flumazenil; consider administration of doxapram
Hypothermia	■ Slow warming over 1–3 hours ■ Check temperature before feeding (hypothermia can cause ileus) and do not feed if temperature is <93.9°F and/or no bowel sounds are heard on abdominal auscultation ■ Consider supplemental oxygen, but avoid neonate becoming cold
Dehydration	Fluid therapy: 120–180 mL/kg/day in neonates; 80–120 mL/kg/day in pediatrics
Hypovolemia	Administer shock rate bolus of crystalloids: 30–45 mL/kg, dogs; 20-30 mL/kg, cats

BOX 1 Viability Scoring Systems

In human pediatric medicine, the Apgar scoring system is used to objectively measure neonatal viability during the first few minutes of life. This is not commonly used in veterinary medicine, but other systems have been developed. Apgar evaluates such parameters as muscle tone, respiratory effort, heart rate, response to stimulation, and mucous membrane color. This system gives clinicians information about the general condition of the neonate, and the scores allocated to an animal correspond with the chance of survival and viability. Studies have demonstrated that a decreased rooting reflex and suckling and swallowing response are related to reduced Apgar scores.[3]

The University of Pennsylvania Ryan Veterinary Hospital has a canine neonatal status and treatment form that features a version of the Apgar score for veterinary patients; it allocates scores based on heart rate, respiratory effort, mucous membrane color, spontaneous activity and muscle tone, suckle reflex, and lumbosacral stimulation. Neonates are allocated a score from 0 to 2 for each parameter, with a potential maximum score of 12. The evaluation also considers interventions that have been carried out, including oxygen therapy, ventilator assistance, acupuncture, chest compressions, use of doxapram, and administration of dextrose and fluids.

peripheral vessels increases. The sense of dyspnea prompts the first chest contraction and the creation of negative pressure within the lungs, which allows air to enter the lungs. The increase in oxygen tension allows the ductus arteriosus to narrow and the pulmonary vessels to dilate. The ductus usually closes 2 to 5 days after birth.

Normal heart rate for neonates is usually around 200 to 220 beats/min in the first week of life.[5] Compared with adults, newborns have decreased stroke volume and peripheral vascular resistance and lower blood pressure. They maintain perfusion by having a much higher heart rate, cardiac output, plasma volume, and central venous pressure.[5] The baroreflex control of their circulation is not fully developed because of incomplete autonomous innervations of heart and vessels; myocardial contractility is also limited.

Heart rhythm is usually a normal sinus, as the vagal reflex develops around 8 weeks of age. It is important to remember that in the first 4 to 5 days of life, neonates respond to hypoxemia with bradycardia and hypotension; thus a heart rate around 150 beats/min in a neonate should suggest a serious underlying disease.

Normal respiratory rate in neonates is approximately 15 to 35 breaths/min; it becomes similar to that of adults at 4 weeks of age. Neonates are susceptible to relative hypoxemia because of their large metabolic oxygen requirement and the immaturity of carotid body chemoreceptors.

Lung expansion in newborns is essential to release both surfactant and prostacyclin, which increases pulmonary blood flow and pulmonary vasodilation. Nitric oxide synthesis is probably induced by fetal oxygenation and may also contribute to pulmonary vasodilation, therefore resulting in less pulmonary vascular resistance at birth and subsequent closure of the ductus arteriosus.[6]

Furthermore, because of a higher compliance of the thoracic wall, neonates must work much harder at breathing to maintain a normal tidal volume compared with adults. This factor is important to remember as any respiratory disorder that shortens inspiratory duration has the potential to negatively affect gas exchange.

Neurologic System

Normal neonate puppies or kittens spend most of their day sleeping; when awake, they should be able to respond to odor, touch, and pain. They should show strong suckle, rooting, and righting reflexes. The withdrawal reflex should be present, although it is often slower than in adults.

The menace reflex normally is not fully developed until 16 weeks of age but can be present as early as 2 weeks in some animals. Pupillary light reaction should be present around 10 to 20 days of age, and vision is normal by 30 days. Pediatric puppies and kittens have a more developed neurologic system, and a neurologic examination can be performed around 6 to 8 weeks of age, when the postural reaction should be present.

Gastrointestinal System

At birth, the gastrointestinal (GI) tract is sterile and characterized by a neutral gastric pH and time-dependent increased permeability of the intestinal mucosa, which decreases dramatically after 10 hours. The motility of the GI tract is affected by the presence of food and especially body temperature; temperature <94°F is associated with GI stasis and paralytic ileus, so checking body temperature in neonates before they feed is useful in the detection and prevention of ileus.

Kidney function and development are incomplete in neonates, with nephrogenesis continuing for at least 2 weeks after birth. Because of this, neonates are unable to concentrate their urine. Glomerular filtration rate is decreased, as is rate of tubular secretion, reaching adult level at 8 weeks of age. Autoregulation of renal blood flow and glomerular filtration rate in neonatal puppies appear to be relatively inefficient in response to rapid changes in systemic arterial blood pressure.[7] In adult dogs, the renin–angiotensin system is an important regulatory mechanism; however, in neonates, renal blood flow is directly correlated with arterial pressure and does not seem to be altered by inhibition of angiotensin until approximately 6 weeks of age. Caution must be exercised when administering renally excreted or metabolized antimicrobials (penicillin, ampicillin, cephalosporins,

fluoroquinolones, and aminoglycosides) to neonates and pediatric patients.

Neonates have immature liver function and limited glycogen stores, and gluconeogenesis impaired. Hepatic glucose stores will be depleted after 24 hours and hypoglycemia will ensue. In addition, neonates have poorly developed microsomal and P450 enzyme activity until 4 to 5 months of age, so caution must be exercised when using medications that require hepatic metabolism or excretion.[7]

Immunologic Immunity

The ingestion of colostrum is essential during the first 12 to 24 hours of life, as only 5% of maternal antibodies are acquired transplacentally. Pediatric patients are unlikely to have fully developed immune systems until around 3 to 4 months of age.[8]

Puppy survival within the early weeks is highly dependent on colostrum, a specific secretion of the mammary gland produced during the first 2 days postpartum. Colostrum is the first mammary secretion produced after delivery (and is occasionally present before parturition), with the transition to milk occurring between day 2 and 3 of lactation; it is both a source of nutrients, including high amounts of protein and lipid, and a source of immunoglobulins (IgG), as puppies are almost agammaglobulinemic at birth.[8] This means the risk of neonatal mortality depends on two factors: the quality of the transfer of passive immunity (evaluated by circulating IgG levels at 2 days of age) and the growth of the puppy during its first 2 days.[9] Despite this essential requirement of colostrum for immunity and calorie energy, there are no guarantees that all puppies or kittens in a litter will consume sufficient amounts of colostrum.

Colostrum also contains a number of cells, including macrophages, neutrophils, and lymphocytes, that must be consumed by the puppy before the intestinal barrier closes; these cells also play an essential role in cellular, humoral, and local digestive immunity.[10,11] For passive immunity to be acquired, puppies must receive colostrum within the first 8 hours of life. This timeframe is critical for two reasons: colostral IgG decreases rapidly in the first few hours postpartum, and the intestinal barrier closes rapidly (within 24 hours in puppies and 16 hours in kittens), meaning that macromolecules (including IgG) can no longer cross the intestinal wall to enter the bloodstream. Thus, while puppies absorb around 40% of ingested colostral IgG at birth, only 20% is absorbed 4 hours after delivery and 9% at 12 hours.[8]

Nutritional Requirements

Because of their limited glycogen stores, it is essential that neonates suckle every 1 to 2 hours, spending the remainder of their time sleeping. Provided the dam is in good health, her milk will be sufficient to maintain a litter's health for the first 3 to 4 weeks. In situations in which milk production is nonexistent (e.g., death of the dam or queen, agalactia [lack of milk]) or insufficient (e.g., mastitis, an exceptionally large litter), milk substitutes will be required; they can also be used if neonates have low body weight at birth (e.g., 25% less than the expected average for the breed), lose >10% of their initial weight in the first 24 hours of life, or do not double their birth weight in the first 2 weeks of life[8] **(FIGURE 1)**.

Milk produced by dams and queens has a high lipid content because neonates use fat, not lactose, as an energy source. Thus any milk substitute needs to replicate this. Cow's milk, which is rich in lactose but low in fat and protein, is completely unsuitable.

Neonates have a daily energy requirement of

FIGURE 1. Neonates should be weighed regularly.

around 20 to 26 kcal/100 g body weight, but most commercial milk replacements generally have only 1 kcal/100 g. Most neonates have a stomach capacity of about 4 mL/kg; therefore, it is possible to estimate an individual's nutritional requirements and the frequency of feeding needed to meet them.[8]

When feeding neonates, a bottle, syringe (FIGURE 2), or orogastric tube can be used as appropriate, and a suckling reflex should be present before feeding is attempted. A feeding bottle, or sometimes a sponge, is ideal, as this initiates the suckling reflex, therefore reducing the risk of aspiration. During feeding, a neonate should be held in a normal feeding position (horizontally without an overly stretched neck; FIGURE 3).

Ideally, body temperature should be assessed before feeding. If it is low, gut motility is reduced, and ileus can occur, the abdomen will become distended, and regurgitation may occur, potentially resulting in aspiration pneumonia. Body temperature should be at least 86°F and/or intestinal sounds auscultated before commencing supplemental feeding. If intestinal sounds are present at a lower body temperature, feeding can be initiated, as this suggests sufficient GI motility.

FIGURE 2. Puppy feeding from a syringe with a teat added.

TECHPOINT

Pediatric patients have glucose requirements 2 to 4 times those of adults.

Neonates should be observed for signs of overfeeding while being fed. These signs include milk at the nostrils, regurgitation, abdominal distention or discomfort, and diarrhea.[8]

COMMON EMERGENCIES IN PEDIATRIC PATIENTS

Hypoglycemia

Pediatric patients have glucose requirements 2 to 4 times those of adults. Hypoglycemia may be a sequela of vomiting, diarrhea, anorexia, dehydration, and/or infection, or it may be a result of decreased hepatic glycogen stores, inefficient hepatic gluconeogenesis, or loss of glucose in the urine. Urinary glucose reabsorption normalizes at approximately 3 weeks of age in puppies. Liver glycogen stores are rapidly depleted in neonatal patients, providing glucose for only a limited time in fasting neonates. The neonatal myocardium uses glucose for energy, whereas adults rely on long-chain fatty acids as a substrate to the myocardium.[4]

FIGURE 3. Feeding should be performed with the neonate in a normal feeding position.

The neonatal brain requires glucose and carbohydrates as its main energy sources, and prolonged hypoglycemia in pediatric patients may result in permanent brain damage. In adults, glucagon, cortisol, epinephrine, and growth hormone are released in response to hypoglycemia to help facilitate euglycemia by increasing gluconeogenesis and antagonizing insulin. These hormones are not released in neonates as these patients have inefficient counterregulatory hormone release during a hypoglycemic event.[3]

Pediatric patients with hypoglycemia may present with many clinical signs, including hypothermia, weakness, seizures, lethargy, and anorexia; they should be treated immediately. Hypoglycemia is considered significant when blood glucose is <40 mg/dL. Intravenous (IV) dextrose boluses (1 mL/kg of 12.5% dextrose [dilute 50% dextrose 1:4 with sterile water]) should be administered. To decrease the risk of rebound hypoglycemia, the bolus should be followed by an infusion of isotonic fluids supplemented with 2.5% to 5% dextrose. Hypoglycemia can become refractory, and patients may require hourly dextrose boluses in addition to a dextrose-containing infusion. Blood glucose should be monitored regularly until hypoglycemia is stabilized. Care must be taken to prevent oversupplementation, as prolonged hyperglycemia may lead to osmotic diuresis, thereby worsening dehydration.[6]

Hypoxia

Any neonate or pediatric patient that demonstrates clinical signs of hypoxemia, including dyspnea, cyanosis, orthopnea, tachypnea, and abnormal lung sounds on auscultation, requires immediate oxygen supplementation. Bradycardia and hypotension are also found in hypoxic neonates.[5] Because of the lower packed cell volume in neonates, cyanosis can be much more difficult to observe, as visual detection of cyanosis is dependent on hemoglobin concentration.[6] Clinical signs of hypoxia are not common in newly born puppies and kittens as neonates

TECHPOINT

Veterinary technicians must be aware that normal methods for assessing hydration may be unreliable in sick neonates.

tend not to hyperventilate until they are several days old, and most newborn animals, including those born via cesarean section, tend to recover within 45 minutes.[5]

Numerous factors can result in respiratory distress, including decreased surfactant in the lungs, aspiration of meconium, pneumonia, and congenital defects that can result in hypertension. Respiratory distress may also be caused by drugs used during anesthesia of the dam or queen for cesarean section (e.g., sedatives, anesthetic agents); if this is a consideration, reversal agents (e.g., naloxone, flumazenil) should be administered. If hypoxia is detected, it should be treated appropriately as it may lead to complications including respiratory depression, bacterial translocation, and chilling, which in turn can reduce resistance against bacterial infections.[5]

Therapy should comprise oxygen supplementation via an appropriate route, which may include flow-by **(FIGURE 4)**, incubator, or endotracheal tube. When choosing an oxygen supplementation technique, it should be remembered that the fraction of inspired oxygen (FiO_2) should not exceed 40% to 60%. The FiO_2 is the concentration of oxygen a patient is inhaling. For example, a patient breathing room air will have a FiO_2 of 21%.

Oxygen toxicosis can manifest as acute respiratory distress syndrome or retrolental fibroplasia (which can result in blindness) as a result of prolonged exposure to a high FiO_2. Care also needs to be taken to prevent high concentrations of oxygen from coming into direct contact with the eyes, as this can result in retinal detachment.[12] If patients do require high levels of oxygen to relieve the signs of respiratory distress, the use of positive-pressure ventilation should be considered; in reality, however, this can be difficult to provide in these small patients.[6]

Hypothermia

Neonates are unable to thermoregulate and depend on environmental heat sources to maintain their body temperature (poikilothermic) up to 4 weeks of age. They have well-developed behavioral heat-seeking responses that enable them to maintain body temperature if heat sources are available. Neonates are prone to hypothermia because of their greater surface area:body weight ratio, immature metabolism, and impaired shivering and vasoconstrictive mechanisms.[7,13]

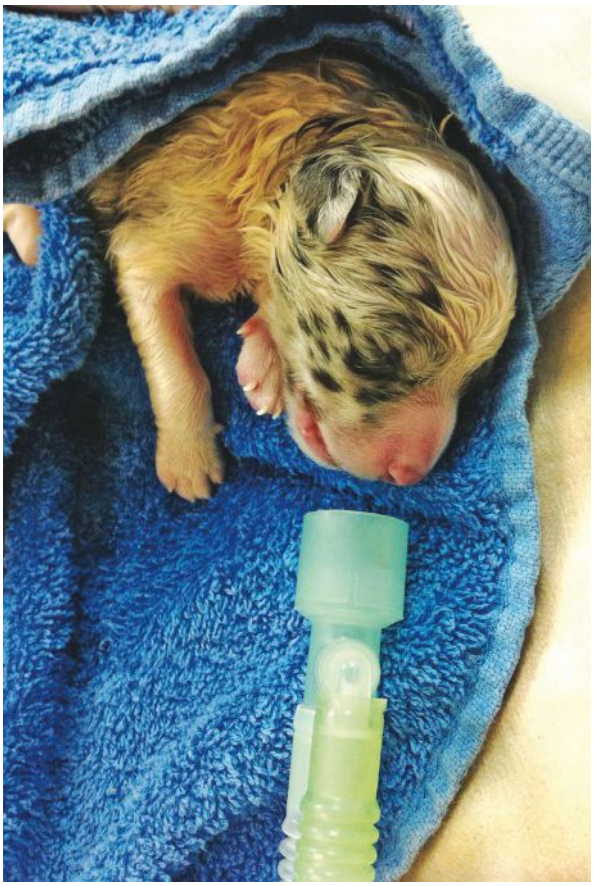

FIGURE 4. Flow-by oxygen being administered to a newly born neonate.

Knowledge of the average body temperatures of pediatric patients is vitally important when nursing these patients. In the first week after birth, normal body temperatures in puppies should be between 96°F and 97°F, increasing to 100°F by 4 weeks of age. At birth, body temperature in kittens should be 98°F, increasing to 100°F by 4 weeks of age.

Physiologic responses to hypothermia (<86°F) may include cardiopulmonary depression and bradycardia, which in time may lead to hypoxia. Normal heart rate is 200 to 220 beats/min during the first 2 weeks. Vagal tone is achieved in neonatal patients at approximately 2 weeks of age, after which time the heart rate should decrease to a normal range of 100 to 140 beats/min.[3]

Hypothermic patients should be warmed slowly before being fed, as hypothermia may result in GI ileus and inability to absorb orally consumed nutrients.

Neonates should be warmed slowly over 1 to 3 hours to prevent overheating. Rapid warming or overheating may cause peripheral vasodilation, which can result in core body temperature shock due to decreased circulating volume to the core.[6]

Many heat sources can be valuable in warming hypothermic neonates, including heat mats, heat lamps, hot water bottles, and warm towels/blankets. To prevent overheating, neonates should be given space to crawl away from any heat source. Human neonatal incubators are a good option for these patients as the temperature and humidity can be controlled and oxygen supplementation can also be added if necessary.

Dehydration

Pediatric patients (particularly neonates) have higher fluid requirements than adults because of their increased extracellular fluid requirements. Decreased body fat, higher metabolic rate, decreased renal concentrating ability, greater surface area:body weight ratio, and increased respiratory rate lead to greater insensible fluid losses. As a result of these factors, dehydration can occur much more acutely and rapidly in pediatric patients. Signs a neonate is dehydrated may include pale mucous membranes, prolonged capillary refill time, cold extremities, lethargy, decreased urine output, and reluctance to suckle. Dehydration and hypovolemia most commonly occur as a result of diarrhea, vomiting, or decreased fluid intake.[14]

Veterinary technicians must be aware that normal methods for assessing hydration may be unreliable in sick neonates. Skin turgor, commonly used in adult cats and dogs, is less reliable in neonates owing to their increased water content and decreased subcutaneous fat. Tachycardia and concentrated urine, responses seen to dehydration in adult patients, do not occur in neonates because their heart rate is already rapid and they are unable to concentrate urine. In neonatal patients, mucous membranes often remain moist until dehydration is severe. Newborns up to 1 week in age have hyperemic mucous membranes. After this time, mucous membrane color and capillary refill time can be used as an indicator of dehydration and shock. Clinical pathology may also be difficult to interpret as neonates have lower packed cell volume, albumin, and total solids values. Neonates that are unable to suckle for the first 24 hours are at high risk for developing infections (because of the deprivation of colostrum), and care must be taken, as with all patients, to adhere to strict asepsis when administering fluid therapy.[14]

Routes of administration for fluid therapy include subcutaneous (SC), intraosseous **(IO; FIGURE 5)**, IV, and intraperitoneal (IP). IV or IO fluid therapy is indicated in severely dehydrated patients or those with perfusion deficits. These routes are best for aggressive fluid resuscitation. IV fluid administration is ideal

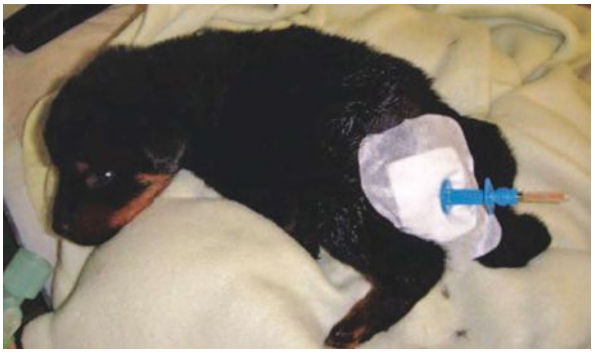

FIGURE 5. Intraosseous catheter in situ.

but sometimes may not be possible in severely dehydrated or small patients. If IV access is not possible, IO administration is the preferred route for fluid therapy. This can be achieved by using an 18- to 22-gauge spinal or hypodermic needle placed in the proximal femur, proximal humerus, head of the tibial crest, or wing of the ileum. As with IV catheterization, strict asepsis must be followed during IO catheterization. IO catheters can be problematic to secure; once vascular volume has been restored, it may be of benefit to place an IV catheter.

Because of the small size of these patients, the jugular vein is commonly used for IV catheterization. Cephalic catheterization may also be achievable using an appropriate-size catheter.

In severely dehydrated or hypovolemic patients, fluid rates administered via the IO or IV route should include an initial shock dose of a balanced crystalloid (20–40 mL/kg in puppies; 20–30 mL/kg in kittens). After stabilization, maintenance rates should be administered depending on the age of the patient (80–180 mL/kg/day). As with all fluid therapy, ongoing losses should be taken into account when instigating a fluid plan. Blood glucose should be monitored frequently and supplementation implemented promptly in hypoglycemic patients **(see HYPOGLYCEMIA)**.

If IV or IO access is not achievable, fluids may be given into the intraperitoneal space. Fluids than can be administered via the IP route include colostrum, whole blood, and crystalloid solutions. Hypertonic dextrose solutions should be avoided: they will pull fluid from the intravascular space and interstitium into the abdominal cavity. The absorption of blood administered IP is slow (48–72 hours); therefore, this route of blood administration is not appropriate for treating patients with severe anemia.[14]

SC fluids may be administered to neonates with mild to moderate dehydration. Maintenance fluid rates for pediatric patients (120–180 mL/kg/day) are significantly higher than for adults.[15] Once calculated, the appropriate amount of fluid can be administered as several boluses or as an infusion. The volume of fluid is calculated at maintenance plus the dehydration deficit (% dehydration x body weight in kg). The best fluid to correct mild to moderate dehydration is a balanced electrolyte solution such as Normosol-R or lactated Ringer's solution.

Ongoing monitoring of dehydrated patients should include weighing the patient every 8 hours as well as measuring electrolyte and glucose status. When urine specific gravity (USG) reaches 1.020, dehydration is likely; USG can also be regularly monitored as an indicator of rehydration.[16] In patients younger than 8 weeks, normal USG is 1.006 to 1.017. All fluids administered should be warmed to body temperature before being administered to aid in the prevention of hypothermia.

CONCLUSION

Understanding the unique differences between pediatric and adult patients will assist veterinary nurses and technicians in the care of these patients. Recognizing how these differences can affect diagnosis and treatment can be challenging and intimidating but also extremely rewarding and educational. ■

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