Canine and feline reproductive and pediatric difficulties are commonly encountered emergencies due to a combination of factors, including small animal anatomy and owner ignorance. Reproductive emergencies are considered all illnesses involving the reproductive system, with those occurring most frequently discussed here. Pediatric emergencies refer to life-threatening illnesses in small animal patients under six months of ages. An important part of understanding how to treat these types of emergencies is understanding why they occur, which is a matter of the varying physiology in pregnant and young animals.

Pregnant animals live in a state of relative anemia, decreased lung capacity, and increased risk of hypotension, impairing their ability to adapt to stress and illness. Not surprisingly, these risks translate to the fetuses as well. Knowing that these differences exist allows proper handling and care to avoid unnecessary stress, anesthetics, and other therapies that could harm mother and offspring alike. Special considerations for pregnant animals are simple to employ and help maximize successful treatment of all involved. Examples include providing preemptive oxygen and ventilatory support, avoiding positioning in dorsal recumbency, and minimizing the use of medications that could lead to hypotension or bradycardia.

Appropriate treatment of reproductive emergencies begins with a thorough understand of normal small animal pregnancy and parturition. A normal gestation length is 63 to 65 days (from the day of first breeding if multiple attempts occurred) in both dogs and cats. However, variation exists with large litters often arriving several days prematurely. Premature animals often lack fur on the dorsal aspects of their paws and carry a higher risk of mortality. If the exact time of breeding is unknown, radiographs should be performed, as a full-term fetus demonstrates ossification of its radius, ulna, tibia, and fibula. Normal labor is initiated by the fetus, and successful parturition requires a live fetus, as secretions from the fetal adrenal gland causes prostaglandin release in the placenta, which then promotes uterine contractions and antagonizes the ovarian progesterone needed to maintain pregnancy. Stage I of labor begins with the onset of uterine contractions and ends with dilation of the cervix. However, as the cervix is not palpable via vaginal exam in dogs and cats, behavioral changes are often used to determine this stage instead. Stage I typically lasts between 6 to 12 hours, although may last up to 24 hours without issue. Typical behavior changes include restlessness, panting, and nesting. Stage II of labor begins with full dilation of the cervix and ends with complete expulsion of a fetus. Stage III begins following expulsion of a fetus and ends with expulsion of the placenta. Stage II and III repeat until all fetuses are delivered.

Dystocia, put simply, means ‘difficult birth’ and refers to the inability to expel the conceptus from the uterus through the birth canal. Recognizing dystocia is critical to appropriate care, as rapid treatment is needed for survival of mother and offspring. A small animal patient meeting any of the following criteria is considered to be experiencing dystocia: (1) greater than 24 hours of Stage I labor, (2) greater than 60 minute of active labor without delivery of conceptus, (3) greater than 4 hours between delivery of conceptus, (4) greater than 36 hours to deliver the entire litter, (5) obstruction of a fetus within the birth canal/obvious radiographic abnormality, (6)
history of previous dystocia, or (7) gestation period greater than 72 days from day of first breeding. Dystocias may result from many different maternal and fetal factors, including uterine inertia, birth canal narrowing, history of previous Caesarean section, malpresentation of the fetus, oversized fetus, or fetal death. Uterine inertia, one of the most common causes of dystocia, is failure of sufficient uterine contractions to expel the conceptus. There are several categories of uterine inertia, including primary inertia due to maternal factors (breed, systemic illness, electrolyte imbalances) versus secondary to fetal obstruction within the birth canal. Uterine inertia can be further divided into complete, where no conceptus is delivered, versus incomplete, meaning a portion of the litter is delivered normally prior to the uterine fatiguing. Treatment of uterine inertia may be medical (oxytocin or calcium gluconate) or surgical (Caesarean section) based on careful consideration of maternal and fetal factors. Medical management is best reserved for patients with normal labwork, radiographs, and fetal heart rates greater than 180 beats per minute. Surgical intervention is warranted when uterine inertia is unresponsive to oxytocin or if radiating identifies evidence of fetal death, oversize, obstruction, or distress (heart rate less than 140 beats per minute). As dystocia is known to recur with all future litters, ovariohysterectomy at the time of first Caesarean section is recommended.

Another commonly occurring reproductive emergency is pyometra, or uterine infection. Pyometra is a life-threatening condition due to the risks of a patient developing septicemia or uterine rupture as a result of the uterine infection. A typical history for patients with pyometra includes an intact female with a previous heat cycle three weeks prior. Clinical signs may include polyuria/polydipsia, gastrointestinal upset, and lethargy. Vaginal discharge may or may not be present. Definitive diagnosis involves visualization of fluid-distended uterine horns via abdominal radiographs or ultrasound. Treatment of pyometra is emergency ovariohysterectomy and perioperative antibiotics. Medical management is infrequently performed given the high risk of side effects, pain associated with treatment, and risk of recurrence infection following all subsequent heat cycles.

Eclampsia is another life-threatening reproduction-related illness and refers to severe hypocalcemia in a lactating dam. More specifically, eclampsia is defined as a total calcium less than 7 milligrams per deciliter (mg/dL) or ionized calcium less than 0.8 mg/dL. Eclampsia occurs due to calcium loss secondary to milk production. Additional risk factors include large litters, first litters, toy breed dogs, and poor prenatal nutrition. Clinical signs may include restlessness, ataxia, paresis, diffuse muscle tremors, or seizures depending on the severity of hypocalcemia. Treatment involves calcium supplementation, first immediate intravenous administration of calcium gluconate following by maintenance therapy with oral calcium carbonate. Diet change to a puppy or kitten diet is encouraged. Offspring greater than three weeks of age are weaned, whereas, litters less than three weeks old are prevented from nursing for 24 hours and then alternated between bottle feeding and nursing for a ten day period.

In shifting the focus from damn to offspring, discussing pediatric emergencies continues to demand special consideration of the known differences in these patients’ physical exam findings and physiology when compared to adults. Many pediatric ‘normals’ fall outside the adult ranges included on complete blood count and serum chemistry profiles. In addition, urinalysis and radiographic differences are expected and important factors in assessing hydration status of a
pediatric patient. True abnormalities reported in the history of a sick pediatric may include crying, lethargy, weakness, gastrointestinal upset, decreased nursing, seizures, and/or weight loss. Examination of a pediatric patient should include assessment for problems such as congenital abnormalities, oral or skin ulceration (indicative of sepsis), or umbilical and inguinal hernias. TPR of a sick pediatric should always include a blood glucose level, as hypoglycemia is a very common. Pediatric dogs and cats have a very high risk of developing hypoglycemia due to several factors, including their greater glucose requirements versus adults combined with inefficient glucose synthesis and decreased glycogen stores within the liver. Causes of hypoglycemia include gastrointestinal disturbances (vomiting, diarrhea, anorexia), inadequate feeding, overfeeding if diarrhea ensues, and sepsis. Treatment is initiated with intravenous dextrose boluses to achieve normoglycemia and followed by a 2.5% to 5% dextrose constant rate infusion for several hours to avoid rebound hypoglycemia. Frequent feeding and treatment of any underlying gastrointestinal disease is also recommended.

Dehydration occurs rapidly in pediatric patients due to their higher fluid requirements and increased water loss. Diagnosis of dehydration is made throughout a combination of fitting history of gastrointestinal upset and exam findings such as dry mucous membranes. Treatment of dehydration clearly involves rehydration, however, venous access is a challenging and essential aspect of fluid therapy in pediatric patients. An intravenous catheter is preferred, but when small patient size prevents such placement, an intraosseous catheter as a suitable second choice. Intraosseous catheters should be removed as soon as adequate rehydration allows for placement of an IV catheter, ideally within six hours, to reduce the risk of infection. Rehydration is best achieved with crystalloids given at an initial dose of 30 milliliters per kilogram followed by a 90 milliliter per kilogram per day constant rate infusion. Lactated ringers are the fluid of choice for pediatric patients as lactate is the preferred metabolic fuel in a hypoglycemic neonate.

Although less common than other pediatric emergencies, sepsis is the most life-threatening and often occurs simultaneously with hypoglycemia and dehydration. Sepsis is systemic inflammation in response to an infection. In pediatric patients, the infection most often results from wounds such as tail docking or umbilical ligation, gastrointestinal upset, pneumonia, or failure of passive transfer. Clinical signs include crying, anorexia, oral or skin ulceration, and as in adults, hypoglycemia and hypotension. Treatment involves fluid resuscitation, dextrose supplementation, and broad-spectrum antibiotics. Unlike other causes of pediatric emergency room visits, sepsis carries a guarded prognosis even with rapid and aggressive care.
Blood products are an essential and life-saving aspect of small animal emergency medicine. The keys to successful treatment of an anemic or bleeding patient include knowing when a blood transfusion is indicated, which blood product is required, and how to safely obtain and administer these blood products. In small animal medicine, there are several types of commercially available blood products. In-hospital blood donors can be used for collection of whole blood. Canine blood donors must be young, healthy, and weight at least 25 kilograms. Complete blood count, serum chemistry panel, thyroid hormone levels, and urinalysis must all be normal. They should be blood types and negative for fecal parasites and blood borne infections, including heartworm, tickborne infections, hemotropic Mycoplasma, Brucella, and Leishmania. In suitable donors, a total of 450 milliliters of whole blood is aseptically collected from the jugular vein using a closed collection system. Feline donors should also be young, healthy, and weigh at least 5 kilograms. In addition to normal baseline bloodwork and blood typing, they must be negative for infections including hemotropic Mycoplasma, feline leukemia virus, feline immunodeficiency virus, and Toxoplasma. Heavy sedation is recommended to allow safe and aseptic collection of 50 milliliters of whole blood from a jugular vein.

Blood typing is a classification based on the presence or absence of antigenic substances on the surface of red blood cells. Identifying a patient’s blood type is recommended prior to every blood transfusion, although this testing displays limited accuracy in patients whose blood is auto-agglutinating. Unlike dogs, cats possess naturally occurring antibodies against other blood types and should never receive a transfusion without appropriate typing, as life-threatening reactions can occur. There are six dog erythrocyte antigens (DEAs), but as DEA 1.1 is the most immunogenic, it is also the most clinically important blood type. In simplest terms, canine blood typing divides patients into two groups: DEA 1.1 positive or negative. A Dal blood type is also present in some Dalmatians, however, due to limited availability of cage-side Dal typing tests, transfusion of exclusively Dalmatian blood to other Dalmatians is recommended. There are three feline blood types: A, B, and AB. Type A is the most common in domestic cats. Type B is the predominant blood type among Abyssinians, Persians, and Devon Rex breeds. Type AB is extremely rare with limited AB blood products available for clinic use.

Crossmatch testing simulates the in vitro response of the recipient’s immune system to the donor’s blood components. Major crossmatching tests donor red blood cells against recipient plasma. Minor crossmatching tests donor plasma against recipient red blood cells. Crossmatching is recommended in patients that have received a blood transfusion greater than five days earlier, as there is sufficient time for anti-erythrocyte antibodies to be produced. It is also recommended in patients with auto-agglutination, as accurate blood typing cannot be performed. Despite these advantages, crossmatch testing cannot predict the risk of an immediate hypersensitivity reaction to the donor white blood cells or platelets present in whole blood.

A blood transfusion is generally recommended in an acutely anemic patient with a packed cell volume (PCV) less than 20% or a chronically anemic animal with a PCV less than 10%. Ultimately, the need to transfuse should be guided by a patient’s clinical signs, including lethargy, tachycardia, tachypnea, and anorexia. Blood administration ideally occurs 3 to 4 hours
via a blood administration set and gravity delivery. Excessively rapid or prolonged transfusions increase a patient’s risk of developing a transfusion reaction. Delivery via a volumetric or syringe pump has been shown to result in significant lysis of canine red blood cells. The same has not been demonstrated with feline red blood cell transfusions. Blood products are typically warmed to room temperature prior to administration to avoid recipient hypothermia, although this is not essential when the need for transfusion is emergent. Appropriate testing prior to and close monitoring during a blood transfusion minimize the risk of a transfusion reaction, which occur in only 2% of small animal patients. It is the result of destruction of donor red blood cells by the recipient’s immune system. Common signs of a transfusion reaction may include pyrexia, tachycardia, gastrointestinal upset, and urticaria. Immediate transfusion reactions result from intravascular hemolysis, causing hemoglobinemia and hemoglobinuria. Delayed reactions are the result of extravascular hemolysis of donor red blood cells and cause hyperbilirubinemia hours to days following a transfusion. Treatment of a transfusion reaction involves immediate discontinuation of the transfusion and initiation of medications to stop hypersensitivity (such as antihistamines, corticosteroids, epinephrine, and others based on the patient’s need).

Blood product choice is guided by an individual patient’s needs, hydration status, and source of blood loss. However, in veterinary medicine, blood product availability also weighs heavily into this decision. Whole blood remains a commonly used product amongst general practitioners as it can be obtained on an as needed basis from in-house donors. Whole blood contains all components of blood, including red blood cells, plasma, platelets, and white blood cells, and it requires no special processing following collection. It is best used in patients with acute blood loss or concurrent anemia and hypovolemia, as the dose is twice that of packed red blood cells, which results in significant blood volume expansion. Centrifugation of whole blood results in separation into two components: packed red blood cells and plasma. Packed red blood cells are the treatment of choice for anemic, normovolemic patients. Plasma contains all coagulation factors, albumin, and immunoglobulins. It is available in fresh and frozen preparations and is used to treat patients with coagulopathies, severe inflammatory conditions (pancreatitis, vasculitis, disseminated intravascular coagulation), and severe hypoalbuminemia. Cryoprecipitate is made via specific thawing and centrifugation of fresh frozen plasma. It contains von Willebrand factor, coagulation factors V, VIII, XIII, and fibrinogen. It is often reserved for patients with known Hemophilia or von Willebrand Disease prior to elective surgical procedures. Platelet concentrates are a specialized blood product that is collected from donor animals via plateletpheresis. It is used to treat patients with thrombocytopenia or thrombocytopenia, but is of limited use due to its very short shelf life (days) and even shorter half-life within the recipient (hours). Concentrated albumin is a human albumin product with limited uses in hypoalbuminemic canine patients. Repeat administration is not recommended due to rapid development of anti-albumin antibodies. Use in cats has not been studied.

Lastly, autologous blood transfusions refer to a whole blood transfusion where the donor and recipient is the same patient. In veterinary medicine, autologous blood transfusions are most commonly perioperative red blood cell salvage procedures in a hemorrhaging patient, i.e. hemoabdomen or hemothorax. Although this eliminates the risk of immune reactions against foreign blood components, its safe use is limited by the need for specialized equipment to anticoagulate and filter the blood. In addition, as autologous transfusions are contraindicated in
patients with possible blood borne neoplasia or infection, there remains a very limited veterinary patient population for which this blood product is clinically useful.
Anaphylaxis is defined in human and veterinary medicine as a severe, life-threatening, systemic hypersensitivity reaction. More specifically, anaphylaxis is traditionally a Type I, or Immunoglobulin E (IgE)-dependent, reaction. IgE are antibodies synthesized by the immune system in response to allergen exposure. IgE bind to receptors on the surfaces of mast cells and basophils, which leads to release of mediators such as histamine, heparin, proteases, leukotrienes, platelet activating factor, and many others. Anaphylaxis affects multiple organ systems, but especially targets the dermal, gastrointestinal, cardiovascular, respiratory, and neurologic systems where concentrations of mast cells are highest.

While these definitions are helpful in describing what anaphylaxis is, the challenge remains determining when it is occurring. In human medicine, anaphylaxis is highly probable when one of the following three criteria is met: (1) acute onset of illness with skin involvement and either respiratory compromise or hypotension (allergen exposure unknown); or (2) acute onset of at least two of the following: skin signs, respiratory distress, hypotension, or persistent gastrointestinal signs following exposure to a likely allergen; or (3) acute hypotension following exposure to a known allergen. In veterinary medicine, allergen exposure is typically unknown; therefore, diagnosis of anaphylaxis is based upon clinical signs and exclusion of other differentials. True prevalence of anaphylaxis is unknown in both human and veterinary medicine, but is estimated to be between 0.05-2% lifetime prevalence in people. This combination of vague clinical signs and infrequent occurrence often leads to delayed or misdiagnosis. Ultimately, delayed recognition carries life-threatening consequences, as anaphylaxis can be fatal within one hour without appropriate treatment.

Anaphylaxis is a potentially fatal illness due to the rapidity and volume with which vasoactive mediators are released and the widespread location of their receptors throughout most major organ systems. Clinical signs of anaphylaxis are species-specific and dependent upon distribution of mast cells throughout the body. In any species, however, the most severe signs result from histamine and leukotriene release, which causes severe vasodilation, increased vascular permeability, decreased cardiac contractility, and decreased venous return, or in a simpler sense, complete cardiovascular collapse. Portal hypertension is also common, leading to hypoxic liver damage. As histamine receptors are also present within the gastrointestinal tract, skin, airways, and myocardium, a wide range of signs can result, including vomiting, diarrhea/hematochezia, ileus, pruritis, rhinitis, bronchoconstriction, laryngeal edema, and coronary vasoconstriction, among others. Other complications include coagulopathies such as disseminated intravascular coagulation (DIC), hemolysis, rhabdomyolysis, and acute kidney injury. In dogs, the largest mast cell populations reside within the gastrointestinal tract and liver. As a result, canine cases of anaphylaxis typically involve severe GI signs (vomiting, diarrhea, and prolonged anorexia) and liver damage secondary to portal hypertension. In cats, respiratory distress is the most common sign. In humans experiencing anaphylaxis, respiratory and cardiac signs predominate. In some cases, biphasic reactions occur with recurrence of signs 72 hours after initial recovery.
There are many reported causes of anaphylaxis in small animals, the most common including medications (β-lactam antibiotics, non-steroidal anti-inflammatory drugs, and chemotherapeutics), insects (especially Hymenoptera stings), and foods. Non-immune mediated triggers such as cold exposure and exercise have also been reported. When no trigger is identified, idiopathic anaphylaxis is diagnosed. In humans, biochemical markers such as serum histamine, tryptase, and platelet activating factor levels aid in diagnosing anaphylaxis. Limited test availability and lack of veterinary-specific research renders these markers of minimal value in small animal medicine. One recent canine study describes a correlation between alanine transaminase (ALT) elevation and gallbladder wall edema with anaphylaxis in dogs. The ALT elevation results from liver hypoxia. Gallbladder wall edema is visualized via a scanning abdominal ultrasound and is the result of portal hypertension. The study suggests that dogs experiencing acute onset of illness and concurrent ALT elevation and gallbladder wall edema have a high likelihood that their signs are a result of anaphylaxis, but this is as close to a diagnostic tool as we currently have in veterinary medicine.

The mainstay of anaphylaxis treatment is epinephrine. Epinephrine acts to increased system blood pressure through vasoconstriction. It also increases heart rate and cardiac contractility and promotes bronchodilation, thereby counteracting the effects of histamine. Other therapies are based upon clinical signs but typically include intravenous fluid resuscitation, antihistamines, antacids such as H1 blockers, and other gastroprotectants. Broad-spectrum antibiotics are used in cases of severe gastrointestinal upset to avoid bacterial translocation. Albuterol is indicated to treat bronchoconstriction, and corticosteroid use is reserved only for cases of severe laryngeal edema. Plasma transfusion is recommended in coagulopathic patients. Additional vasopressors such as dopamine or norepinephrine are warranted in patients with hypotension refractory to epinephrine and fluid therapy.

Most animals experiencing anaphylaxis present to the hospital in a life-threatening state of hypotension and hypovolemic shock, and despite stabilization of clinical signs within hours, often require days to a week of supportive care prior to discharge. This care is intensive and typically involves frequent blood pressure, clotting time, renal value, packed cell volume, serum protein and platelet level monitoring in addition to routine evaluation of vital parameters. Prognosis is unpredictable and depends on the severity of signs, which again varies between species and route of allergen exposure. Animals with parenteral exposure to the inciting trigger display the most significant illness and therefore the most guarded prognosis. Ultimately, anaphylaxis is such a challenging condition to diagnose and treat due to its sudden onset of life-threatening signs and highly variable clinical manifestation. Recognition of anaphylaxis begins with its remaining a top differential for any patient presenting with acute onset of critical illness.