Kirby's Rule of 20- The Cat is Not a Small Dog in ICU

Rebecca Kirby, DVM, DACVIM, DACVECC
Animal Emergency Center
Glendale, WI

There are a group of diseases that share a common pathophysiology: an inciting stimulus initiates the production and release of circulating mediators which cause systemic inflammatory changes. The response of the body to this cascade of inflammatory mediators is called the systemic inflammatory response syndrome (SIRS). Inciting diseases include sepsis and septic shock, pancreatitis, heat stroke, severe multiple trauma, snake bite, viremia, parasitemia (such as babesiosis), and pansystemic neoplasia.

Once the mediators have entered the circulation, the progression and complications are the same for each disease: peripheral vascular dilation, increased capillary permeability, and depressed cardiac function. Shock ensues with multiple organ dysfunction. The lungs become dysfunctional within 24-72 hours after onset of shock, and the liver and gastrointestinal tract malfunction within 72-96 hours. Acute renal failure follows, with the final stages resulting in heart and brain failure. This progression of organ dysfunction is termed multiple organ dysfunction sydrome (MODS). Sepsis, SIRS and MODS remain tremendous obstacles to the successful treatment of critically ill small animals. Mortality rates in humans with SIRS have been reported as high as 40-60%, and as the patient progresses to MODS, mortality increases to 80-100%.

SIRS

Increase capillarypermeability
- Albumin loss
- coagulopathy

Hypovolemic Shock

Negative inotropes
- Arrhythmias
- Poor cardiac output

Cardiogenic Shock

Vasodilation
- Hypotension
- Increased vascular
- Volume

Distributive shock

Cats are not small dogs in ICU

Cats and dogs in crisis share many of the same disease entities, but may not show the same clinical signs. Pancreatitis in the cat may cause anorexia, severe hypotension and hypothermia, without vomiting or obvious abdominal pain typically seen in the dog. Heart disease in the cat may be evidenced by rear limb paralysis rather than fulminate respiratory distress. Cats have diseases and viruses unique to their species such as hyperthyroidism, feline asthma, feline lower urinary tract disease, and feline leukemia virus and immunodeficiency virus related diseases. Cats are difficult to resuscitate from hypotensive episodes. In the cat, when the baroreceptors have detected inadequate arterial stretch, vagal fibers are stimulated simultaneously with sympathetic fibers (Schwartz, 1973). As a result, the heart rate may be normal or slow, instead of the typical tachycardia demonstrated by other species. In aseries of hypotensive cats (blood pressure by doppler < 80 mmHg systolic) at the Animal Emergency Center, most of the cats were found to have normal or slow heart rates (heartrate < 180 bpm). The heart rates of normotensive cats presented for emergency examination were 180-210 bpm. Because cardiac output is a function of contractility and rate, this compensatory response to shock most likely blunted. The hyperdynamic signs of shock seen in other species are not typically seen in the cat. Shock in the cat is most commonly decompensatory, manifested by normal or slow heart rate, severe hypothermia (< 98 F), weak or non palpable peripheral pulses, and profound mental depression (Figure 1). The mucous membranes are gray or white and capillary refill is not evident. The bradycardia and low cardiac output contribute to hypothermia, and hypothermia accentuates the bradycardia. The hypothermia most likely plays a significant role in the poor compensatory response and to the difficulty in providing adequate fluid resuscitation without causing pulmonary edema. Our theorty is that as the rectal temperatures falls, the adrenergic receptors become refractory to catecholamines. This then leads to the normal or slow heart rate and most likely impaired compensatory vasoconstriction, inspite of the presence of norepinephrine and epinephrine. Part of the resuscitation plan in the cat MUST include rewarming. Once the rectal temperature approaches 100 F, it appears that the adrenergic receptors begin to respond to the catecholamines once again. There are many aspects

of critical care that are unique for the cat. Their physiologic response to shock, the procedures required for resuscitation, and many of the parameters that require careful monitoring present specific challenges. Knowledge of the traits specific to the cat is mandatory to optimize their ability to recover from critical illness. These differences will be highlighted throughout the Rule of 20. It is often necessary to resuscitate, support and stabilize the dog and cat for extended periods of time prior to or throughout the course of definitive therapy.

Rule of 20

The Rule of 20 is a check-off list of 20 critical parameters to evaluate at least daily in the critically ill animal. This check-off list is pasted into the patient record with the daily SOAP to prompt the clinician to assess and intervene as required. Comments are written regarding the status and therapeutic strategy for each. The following is a brief synopsis of these 20 parameters. The order of priority will differ depending upon the clinical situation.

1. Fluid balance:
2. Inappropriate replacement and maintenance of intravascular and

interstitial volume is the number one cause of patient decompensation and death in this author's experience. Animals with systemic inflammatory response syndromes (SIRS) or large third body fluid spaces will have massive loss of fluids from the intravascular compartment into the interstitial and third body fluid spaces. Dehydration and poor perfusion are different problems, requiring different therapeutic strategies. Perfusion deficits are due to a loss of intravascular fluid volume (though heart failure must be ruled out as the cause). Replacement of these deficits should occur rapidly and involves giving enough solution to expand and maintain the intravascular space. Dehydration is an extravascular (primarily interstitial) volume deficit. This must be replaced with crystalloids, such as lactated ringers, Plasmalyte® or Normosol-R ®. If the interstitial loss has been over an extended period of time, then replacement can occur slowly (over 8-12 hours). If the interstitial volume is rapidly lost, then the interstitial fluid deficit should be rapidly replaced (1-4 hours). It is quite possible to have perfusion deficits without significant dehydration and dehydration without significant perfusion deficits.

Kirby's Rule of 20

1. Fluid balance
2. Colloid osmotic pressure
3. Oxygenation/ventilation
4. Glucose
5. Electrolytes
6. Blood pressure
7. Albumin
8. Mentation
9. Nutrition
10. RBCs and Hg
11. Heart rate, rhythm, contractility
12. GI motility and integrity
13. Renal function
14. Coagulation
15. Immune status, antibiotics
16. Drugs dosage/metabolism
17. Wound care and bandages
18. Pain control
19. Nursing care
20. Tender loving Care

The blood volume in the cat is 40-55 ml/kg in contrast to 90 ml/kg in the dog. Whenintravascular volume deficits result in poor perfusion, it has been recommended in the past that crystalloids be administered fast in volumes equivalent to the animals' blood volume. However, resuscitation with crystalloids alone can result in significant pulmonary and pleural fluid accumulation. The resultant hypoxemia contributes to the shock athophysiology. Resuscitation from hypovolemic shock can be safely accomplished with a combination of crystalloids and colloids and rewarming procedures. In the hypovolemic cat, a rapid infusion of isotonic crystalloids is administered at 10-15 ml/kg. Since rapid intravenous infusion of hetastarch results in vomiting and hypotension in the cat, hetastarch is administered at 5 ml/kg over 5-10 minutes. The blood pressure is checked and once it is above 40 mmHg systolic, then only maintenance crystalloids are given while the cat is aggressively warmed. The warming procedures should be done within the next 30 minutes with warm water bottles and warming the IV fluids. Once the cat's rectal temperature has risen to 100 F, the blood pressure is rechecked. The hetastarch can then be repeated at 5 ml/kg increments over 15 minutes until the systolic blood pressure > 90 mmHg

and the CVP is 6-8 cm of water (with adequate cardiac and renal function). Most commonly, no further resuscitation fluids are required. The rectal temperature must be maintained as needed by hot water bottles and warm fluids. If the blood pressure does not remain increased, another 5 ml/kg of hetastarch may be required, followed by a CRI of hetastarch at 3-5 ml/cat/hr. Both colloids and crystalloids are administered at the minimum amount required to maintain pressure and volume in the cat. The cat must be closely onitored for overhydration. Should volume overload occur, decreasing crystalloid rate of infusion,stopping colloid infusion, and administering furosemide at 2-7 mg/kg IV can help eliminate signs.

Dogs appear to tolerate rapid infusion of large amounts of crystalloids and colloids as required. The safest resuscitation procedure for the hypovolemic dog is to rapidly infuse 10-25 ml/kg of crystalloid, followed by 5 ml/kg of colloids (preferrably hetastarch or oxyglobin) until the desired end-points of resuscitation are reached. Dogs can require as much as 40 ml/kg of large molecular weight colloids before reaching the desired endpoints when there are SIRS diseases or third body fluid spacing present. Maintenance fluid therapy is delivered utilizing a balanced crystalloid solution. When the animal has a SIRS disease, a CRI of large molecular weight colloids may be required, with the crystalloid, to maintain the intravascular colloidal oncotic pressure and reduce interstitial edema.

Oncotic pull

The administration of colloids with crystalloids during resuscitation and maintenance fluid therapy will restore and maintain intravascular oncotic pull and intravascular fluid volume while minimizing interstitial fluid accumulation. Choices of colloids that can be utilized include whole blood, fresh frozen plasma, gelatins, dextran, hydroxyethyl starch (hetastarch) and stroma free hemoglobin. [It should be noted that stroma free hemoglobin is not approved for use in cats. It has been reported to have been used in cats at a slow rate of administration (10 ml/kg over 24hours).] During initial resuscitation, a combination of synthetic colloid and crystalloids will be utilized as presented above. During maintenance therapy, when the albumin is less than 2.0 g/dl, the author will administer fresh frozen plasma as the colloid of choice.

Immediately after the initial resuscitation with hetastarch as a bolus, the author will administer hetastarch, 10-40 ml/kg/day IV CRI as part of the daily maintenance fluids in the dog. In the cat, the maintenance dosage is 1-8 ml/hour per cat. This volume should be weaned down as soon as possible to avoid volume overload. This has been found to be the most effective way to maintain colloidal oncotic pressures above 20 mmHg.

Glucose

The stress response in the cat frequently results in a transient hyperglycemia, requiring recheck to rule out diabetes mellitus. The blood glucose should be maintained between 100-200 mg/dl. Hypotensive animals must be closely monitored for hypoglycemia and glucose replacement can be accomplished by giving 0.25-0.5 gm/kg IV of a 50% glucose solution followed by a 2.5% concentration in the maintenance fluids. Initial volume resuscitation fluids *should not* contain glucose. When glucose is added to the intravenous fluids on a maintenance basis, it is important to recognize that this does not meet the animal's caloric requirements. This is only to provide a readily available substrate for energy production on a continuing basis.

Electrolytes (calcium, sodium, chloride, potassium, magnesium) and acid-base Balance

Alterations in potassium concentrations are to be expected in critical animals, especially cats. Though ventroflexion of the neck and generalized weakness can occur with hyokalemia in the cat, these signs are rare. This requires that serum potassium levels be monitored and maintenance intravenous fluids supplemented (5-20 mEq/250ml of fluids). Cats with chronic renal disease can have profound potassium wasting, and require oral long term supplementation. One of the key physical signs of hyperkalemia in the dog is bradycardia. Heart rate is not a reliable predictor of hyperkalemia in the cat. Normal and rapid heart rates have been seen in male cats with urinary outflow obstruction and serum potassium concentrations greater than 10 mEq/L. When a cat has bradycardia (< 120 bpm) due to hyperkalemia, there may be only minutes to respond before circulatory collapse and death. Careful volume resuscitation and the administration of regular insulin (0.2-0.4 units/kg IV followed by 2 gms glucose per gram insulin) or calcium gluconate (0.2 - 1.5 ml/kg of 10% solution IV slowly) can be life-saving. Hyophosphatemia can lead to red blood cell hemolysis and energy depletion. It is most commonly seen in the anorexic animal that is beginning to receive nutritional supplementation. Careful monitoring is required and replacement therapy given as required (potassium or sodium phosphate 0.01-0.06 mmol/kg/hr IV). Sodium concentrations are typically a reflection of intravascular solute free water. Hypernatremia is an indication that there is a free water deficit – likely associated with improper fluid therapy, renal dysfunction, diabetes insipidous, or other CNS or endocrine disorders. The intravascular volume must be replaced with a sodium containing fluid. One the deficits have been corrected, free water can be administered in quantities sufficient to replace the deficits: (new Na+ - 140)/140 x kg body wt x 0.6 = free water deficit in liters This volume is replaced by 5% dextrose in water over 12-24 hours. Maintenance fluid requirements are provided utilizing a balanced electrolyte solution and potassium supplementation as needed. Hyponatremia less than 115 mEq/L in a patient with severe neurologic alterations may require sodium supplementation. Fluids deficits will be corrected using normal saline with potassium supplementation as indicated. Hypertonic saline (3% or 5%) can be used during maintenance therapy to replace additional sodium requirements. The

sodium deficit is calculated using the following formula: (140-new Na) x kg body wt x 0.3 = mE/L Na deficit. This should be replenished slowly over 12-24 hours. Rapid replacement has resulted in central pontine myelinolysis.

Oxygenation and ventilation

Arterial blood gases should be evaluated to show any evidence of hypoxemia, hypercarbia, or hyperventilation. This is important for early detection of pulmonary edema or acute respiratory distress syndrome (ARDS) common to animals with SIRS diseases. Oxygen supplementation is needed if there are perfusion or breathing problems. Oxygen is supplied best by nasal cannula or hood. Observation of the breathing pattern of the cat can determine the location of the problem and allow intervention without stressful diagnostics. Administration of a mild sedative (butorphanol 0.2-0.4mg/kg IV) may be required while giving oxygen support. Animals with significant work of breathing must have their airway and breathing controlled with positive pressure ventilation on 100% oxygen early in the disease process. Do not wait until they are agonal! Early intervention is the key to success. Cats with SIRS commonly develop pulmonary edema and pleural fissure lines. There are no obvious signs until the edema is advanced. Initially, the cat will have an increase in respiratory rate and poor mucous membrane color. Auscultation finds louder than normal lung sounds and occasionally a pleural friction rub. When moist crackles are ausculted, pulmonary edema is severe. During ventilator therapy, it is important to frequently monitor arterial blood gases to assess the efficacy of the therapy as well as adjust the respiratory parameters.

Parameters that can be adjusted on e ventilator to improve PaO2 and PaCO2 include: breaths per minute, inspired oxygen concentration (FiO2), inspiratory pressure, positive end-expiratory pressure, tidal volume, inspiratory:expiratory time ratios, and mode of ventilation (assist, mandatory, etc.).

Level of consciousness and mentation

A decline in the level of consciousness or mentation of the animal warrants immediate investigation for hypotension, hypoglycemia and hyperammonemia. Hypoxia, hypocarbia, hypercarbia, hypernatremia, hyponatremia, hyperglycemia, hypoglycemia, hepatic encephalopathy, hyperosmolality, severe fever, severe dehydration, shock, overwhelming sepsis, hypokalemia, hyperkalemia, tachyarrhythmias, bradyarrhythmias, and thiamin deficiency must be considered in the list of etiologies for changing consciousness. Depressed mentation or level of consciousness requires that precautions be taken to protect the airway from aspiration of gastric or esophageal contents and the animal monitored for vasovagal reflex. A nasogastric tube might be useful to assure that the stomach is empty and decompressed. The cause is aggressively pursued and therapy instituted to treat the underlying cause. Specific therapeutics may be required to reduce the intracranial pressure while the cause is being determined. The patients osmolality should e monitored, especially if the animal is being given parenteral nutrition. Glucose levels must be maintained, and appropriate nursing procedures employed (turn every 4 hours, lubricate eyes, elevate head, etc.)

Blood pressure

Systolic pressure must be maintained above 90 mmHg, and more importantly, the mean arterial pressure is maintained above 60 mmHg. A comparison of indirect blood pressure measurement techniques in the cat has found the doppler to provide the most accurate information(McLeish, 1977). Poor perfusion that is nonresponsive to adequate intravascular volume resuscitation necessitates a search for ongoing fluid loss, yoglycemia, hypoxemia, cardiac dysfunction, prolonged hypothermia and bradycardia, arrhythmias, electrolyte imbalances, cardiac tamponade, brain stem pathology, and hypertension. Persistent hypotension, not attributable to these complications, requires assessment of central volume, oxygen supplementation, pain control, evaluation of cardiac function with treatment as indicated, and evaluation for vasopressor therapy. Serial echocardiograms done by the author on dogs with non-responsive hypotension and SIRS have shown dilation of the left ventricle and decreased contractility during their severe hypotensive stages of the syndrome. Though this improves as the disease resolves, positive inotropic support (dobutamine 1.0 - 5.0 ug/kg/min) can be required in the interim. It is ideal to monitor these patients by serial echocardiogram to evaluate contractility before, during and after therapy. Though this can be seen in the cat, it is a rare complication. Blindly placing a hypotensive cat on dobutamine may be a fatal mistake if the cat has an underlying hypertrophic cardiomyopathy.

When the intravascular volume and cardiac contractility are adequate, and hypotension persists, dopamine at 5-15 ug/kg/min is infused for its vasopressor effects. The dose is initially 5 ug/kg/min and can be increased by 1-2 ug/kg/min intervals (up to 20 ug/kg/min) until the desired effect is seen. When blood pressures appear to be stable for 2-4 hours, the pressor drugs are gradually weaned to prevent volume overload and ischemic renal damage. Hypertension is suspected in the cat when systolic/diastolic blood pressures are >160/100 mmHg.(Kobayashi, 1990) Pulse quality is not a reliable indicator. Significant hypertension can lead to poor peripheral perfusion, retinal hemorrhage and detachment, renal damage, and myocardial wall thickening. The underlying cause is treated and therapy chosen based on the cause; chronic renal disease: amlodipine at 0.625mg SID; hyperthyroidism: propranolol at 0.2-1.0mg/kg PO TID or atenolol at 0.2-0.5mg/kg SID; hypertrophic cardiomyopathy: diltiazem at 1-2mg/kg PO TID, or 10mg/kg PO SID of the CD formulation; and enalopril at 1/2 mg/kg sid.

Heart rate, rhythm and contractility

Careful auscultation of the heart is required to detect murmurs and gallop rhythms, suggestive of underlying cardiac disease. Murmurs are usually heard in one location in the cat, just left of the sternum between the 4th and 7th ribs. The gallop or murmur may be intermittent, requiring patience and concentration during auscultation. Underlying cardiomyopathy and mitral insufficiency can interfere with cardiovascular stabilization, if not presenting as a primary problem. The author has a "Rule of 4" for cardiomyopathy in the cat. When any 1 of these 4 clinical signs are seen in the cat, cardiomyopathy must be ruled out: 1) murmur or gallop; 2) unexplained hypothermia; 3) unexplained bradycardia; and 4) louder than normal or moist lung sounds. It is not infrequent for a gallop or murmur to become noticeable after fluid resuscitation has occurred. Cardiomyopathy in the cat can be in the dilated, hypertrophic or intermediate form. The cardiac dynamics and therapeutics are different for each, requiring careful diagnostics prior to definitive therapy. In addition, the authors recommend that ketamine not be used in cats with a murmur or gallop since it can increase blood pressure, causing decompensation of pre clinical cardiomyopathy.

Arrhythmias in cats most often have a definable and treatable underlying cause, such as hyperkalemia, hypokalemia, hypoxemia, hypercarbia, hypercalcemia, hypocalcemia, acidosis, hypomagnesemia, cardiomyopathy, or endogenous toxins from organ failure such as liver or kidney. It is always best to treat the underlying problem rather than give antiarrhythmics. Dogs require antiarrhythmic therapy more frequently than cats. Criteria for initiating antiarrhythmic drug therapy include: sustained significant tachycardia associated with poor cardiac filling; sustained significant bradycardia assoicated with poor cardiac output; multiform ventricular contractions or torsade de pointe; poor perfusion attributable to the arrhythmia; or procedure required for the animal that will potentially further compromise the heart (eg. Anesthesia, decompression of gastric torsion). It is always best to treat the underlying problem rather than give antiarrhythmics. Oxygen supplementation and antiarrhythmic agents can be required with careful attention given to drug dosage.

Albumin

Serum albumin concentration should be maintained above 2.0 g/dl in the acutely ill animal. Hypoalbuminemia can result from increased membrane permeability, glomerular or intestinal loss, liver failure, cytokine suppression of albumin production in SIRS, and supplementation of maintenance fluids with 5% glucose without amino acids(Kaminski, Haase, 1992; Gray, Kaminski, 1985). As serum albumin falls, the body compensates by mobilizing interstitial albumin through the lymphatics into the vasculature. Therefore, low serum albumin concentrations are a reflection of a true total body albumin deficit. As albumin is replaced intravascularly, it is mobilized back into the interstitium to restore that "albumin pool". Serum albumin is necessary to maintain adequate intravascular oncotic pressure and for transport of cations and hormones. It is well recognized that critical human patients with acute persistent hypoalbuminemia have a higher mortality rate. This mortality rate was significantly decreased in those patients that were supplemented to maintain their serum albumin above 2.0 g/dl. This has appeared to be true in the dog and cat. Therefore, when the serum albumin concentration is below 2.0 g/dl, albumin is administered through fresh frozen plasma or whole blood transfusion.

Coagulation

Disseminated intravascular coagulation (DIC) is a hypercoagulable condition that is to be anticipated in any animal that has: capillary abnormalities or stasis, severe hypotension, massive tissue damage, red blood cell hemolysis, or pansystemicdisease. During the acute decompensatory phase of the underlying disease process, DIC can be present without outward signs. The goal is to detect DIC as early as possible so that bleeding and microvascular occlusion from microthrombi and multiple organ dysfunction are prevented. Typically, the order of change of monitored laboratory parameters in DIC are: 1) decrease anti-thrombin III (ATIII), 2)decrease platelet number (usually requires several assessments to detect a declining trend in numbers) 3) shortened coagulation times (PT, PTT, ACT), 4) decrease fibrinogen , 5) prolongation of coagulation times (PT, PTT, ACT) and 6) elevated fibrin degradation products. Because DIC is a dynamic process, evaluation of these parameters is initiated early and repeated frequently so that a trend of change can be established and success of therapy determined.

Clotting times (ACT, PT, APTT) in the cat are normally shorter than in the dog, requiring their own set of normal coagulation values. Animals can become hypercoagulable with low flow states and SIRS diseases, requiring careful monitoring for declining platelet numbers and declining antithrombin levels. Profound hyothermia, as well as particular drugs, can interfere with platelet function. The most common clinical evidence of excessive bleeding from disseminated intravascular coagulation is failure of clot formation in a clot tube, and occasionally subcutaneous hemorrhage. Therapeutics for DIC has five components. First, it is vital that oxygenation and perfusion be improved and capillary stasis be eliminated. Second, the underlying disease must be treated. Third, the target organs of the underlying disease and the target organs of DIC must be supported. The lungs, kidney, heart, brain, and intestines are vulnerable to microthrombi and ischemia. Fourth, if there is consumption of ATIII, the ATIII is replaced through a transfusion of fresh frozen plasma, fresh whole blood or cryoprecipitate. Fifth, the interaction of ATIII with thrombin is greatly accelerated when heparin is available as a cofactor. When there is ample ATIII, heparin can be administered at low dose (50-100 units/kg) subcutaneously every 8 hours. Once the patient is systemically heparinized, heparinization of the blood components is not deemed necessary. Cats with cardiomyopathy are prone to thromboemboli formation in their left atrium or in the peripheral vessels. Evidence

of turbulent blood flow in the left atrium or presence of a clot is indication for anticoagulant therapy. Heparin is used initially at (200 IU/kg IV) and warfarin at (0.5mg/cat PO SID). The PT should be prolonged by 1.5 times normal. Thrombolytic therapy has had mixed results.

Red blood cell/hemoglobin concentration

Frequent blood sampling of critical cats and small dogs can cause anemia severe enough to require blood transfusion by day 3 or 4 of hospitalization. The use of blood tubes and blood culture tubes designed for neonatal humans will minimize the quantities of blood withdrawn. Microhematocrit tubes can be used to harvest small aliquots of serum for in-house biochemical testing. Cats have 3 major blood types: A, B, and AB. Cats with type B erythrocytes have strong, naturally occurring anti-A antibody. Less than 30% of type-A cats have anti-B antibodies, and type AB cats have no preformed antibodies to blood types. Because of the strong possibility of a significant transfusion reaction in type B cats receiving type A blood, a cross-match is recommended prior to blood transfusion. There are inherent differences in feline red blood cells (RBC). They have a life span of 72 days. Rouleaux formation is common and can be confused with red cell agglutination macroscopically. Normal feline RBC have Heinz bodies due to oxidative stress. There are 2 types of reticulocytes: punctate and aggregate. Aggregate reticulocytes mature into punctate reticulocytes with a longer life-span. This makes the corrected reticulocyte percentage a more accurate reflection of bone marrow response than the reticulocyte index in the cat.(Tvedten, 1989) Macrocytic-normochromic RBCs are most common in FeLV-related myeloproliferative disorders, especially without reticulocytosis. Hemobartonella felis can be evident in cats stressed by illness and cause a hemolytic anemia. A normocytic-normochromic anemia commonly develops in critically ill cats. When a significant anemia is due to chronic renal failure, treatment with human recombinant erythropoietin is indicated. Hemoglobin is the most significant factor responsible for oxygen concentration of the blood. When the red blood concentration is too low, hemoglobin is low and oxygen delivery is compromised. However, when the red blood concentration is too high, the viscosity of the blood is increased, compromising blood flow and tissue oxygen delivery. The PCV should be maintained above 20%, and ideally between 30-45%. When red blood cell and hemoglobin are deficient, whole blood or packed red cell transfusion or oxyglobin infusion may be indicated. Infusion of erythrocytes may be beneficial not only in terms of oxygen delivery, but also because these cells contain high concentrations of endogenous enzyme antioxidants, particularly catalase and glutathione (from Zimmerman, 1994). Should the red blood cell concentration be too high (> 55% - exceptions are cats that live at high elevations), fluids are administered to lower the concentration below 50%. When an absolute polycythemia is present, the animal should be bled and the volume of the blood removed is replaced as crystalloid or synthetic colloid. The blood should be saved for transfusion should it be required at a later time.

Renal function

Shock, severe dehydration, hypotension, hypoxia, and nephrotoxic drugs can each contribute to renal dysfunction or failure. Baseline BUN, creatinine, and urinalysis are obtained prior to fluid resuscitation when possible. Creatinine and/or blood urea nitrogen will elevate as glomerular filtration is reduced. Urine output is assessed on an ongoing bases as a reflection of renal function, blood pressure and fluid balance. Glycosuria without hyperglycemia reflects proximal tubular cell damage, a complication of nephrotoxic drugs or renal hypoxia. Urine sediment is evaluated daily in animals on nephrotoxic drugs (such as gentamicin) for the appearance of renal tubular or granular casts. These will appear before there are significant elevations in BUN and creatinine. Should these be seen, the drug is stopped and a decision is made whether to treat the kidneys with mannitol. In addition, the sediment is monitored for signs of infection. It is important to determine whether it was present initially or has occurred in-hospital. Hospital acquired infections suggest nosocomial bacteria which are often resistant to first line antibiotics (see Immune status, antibiotic dosage and selection, WBC count). Impaired renal function is managed in the following order: assure intravascular volume is adequate; assure that MAP is greater than 60 mmHg; mannitol at 0.1 g/kg IV if animal is not volume overloaded and renal insufficiency is caught early; and furosemide 1mg/kg/hr for 4 hours combined with dopamine at 1-3 ug/kg/min as constant rate of infusion for as long as required.Geriatric cats have a high incidence of chronic renal insufficiency complicating thepresenting disease. The inability to concentrate urine leads to volume depletion and potassium wasting. Hyperphosphatemia can cause hyperparathyroidism and hypocalcemia. Anorexia is significant and chronic anemia common. Renal hypertension can complicate resuscitation procedures.

Immune status, antibiotic dosage and selection, wbc count

The ability of the body to fight infection is assessed through white blood cell count and differential, fever response, and globulin levels. Immunocompromise can be the result of he underlying disease, FeLV or FIV viral infection, or the therapy. Animals that are on immunosuppressive drugs or have neutropenia require isolation and strict aseptic procedures and minimal invasive monitoring and therapeutic techniques. Viral (feline leukemia virus, feline immunodeficiency virus, feline panleukopenia, canine distemper, canine parvovirus), protozoal (toxoplasmosis), and overwhelming bacterial (gram positive and negative) infections are common causes of immunosuppression. Feline leukemia virus can cause an immunodeficiency affecting lymphocyte function before myeloproliferative T-cell changes are manifest.(Olsen, 1984) Feline immunodeficiency virus generally requires the presence of intercurrent bacterial or other infectious agents toinduce immunosuppressive syndromes. Feline anleukopenia produces an acute cellmediated

immunosuppression resulting in leukopenia. Testing for these viruses should occur when there are opportunistic infections, or persistent or reoccurring diseases in the cat. When these viruses are discovered in the critically ill cat, treatment of the illness can be difficult and prolonged. Chronic illness associated with fevers and neutropenia may also suggest toxoplasmosis infection. Serum IgM titers may suggest active infection. A response is generally seen with administration of clindamycin HCl (Antirobe, UpJohn) at 25mg/kg PO q 12 hr for 2-3 weeks. For bacterial infections, antibiotic selection is confirmed by microbiological culture and antibiotic sensitivity results. The capability of the critically ill cat to metabolize and eliminate the antibiotic, as well as potential untoward side-effects of the drug, are considered in the antibiotic selection process, as well. Most gram positive cocci and gram negative rods are susceptible to first generation cephalosporins. Cephazolin (loading dose: 40 mg/kg IV; then, 20 mg/kg IV qid) has few toxic side effects and can be given slowly intravenously. When a more aggressive approach is required, gentamicin (3-5 mg/kg IV sid) is given with the cephalosporin after hydration and renal function are determined adequate. Urine dipstick and sediment are monitored for proteins, glucose and casts daily as early signs of nephrotoxicity. The dose and milliliter amount administered should be double checked at each administration. Suspected anaerobic pathogens are treated with metronidizole (20-30 mg/kg/day divided into 3 doses) by intravenous slow infusion (reduce dose by 50% if cat has liver disease). In seemingly resistant bacterial infections, the considerations of mycobacterium, mycoplasma, and Lform bacteria should be investigated. Bacterial L-forms and mycoplasma are cell wall-deficient forms of bacteria. Penetrating bite wounds or surgical incisions are common locations of L-form infection, often manifesting with dermal abscesses, cellulitis and polyarthritis. Mycoplasma is commonly associated with the development of secondary respiratory infections. The diagnosis of infections with L-forms and mycoplasma organisms is difficult because of the difficulty of isolating or identifying these bacterial by microbiological culture techniques and light microscopy. The treatment of these infections consists of administration of oral doxycycline (Lyphomed) at 5mg/kg PO q 12 hr until the discharges or respiratory signs have resolved for a week. Atypical mycobacteriosis can occur and can manifest as multiple fistulous draining tracts in the skin and subcutaneous tissue following traumatic wounds. Microbiological confirmation is difficult with multiple sample submissions and acid-fast staining are often required to isolate the type. Treatment with high dose quinolones (enrofloxacin [Baytril,] 5mg/kg PO q 12 hr) can be tried for 6 week course.Geriatric animals require added consideration of concomitant chronic diseases that may contribute to immunosuppression and longer recovery periods in the ICU. Other than chronic infectious diseases, hyperthyroidism, inflammatory bowel disease, renal insufficiency, diabetes mellitus, and dental disease need to be considered.

GI motility and mucosal integrity

Critical illness is frequently complicated by gastricparesis, ileus and gastric ulceration. Stress gastric ulcerations may be subclinical but predispose the patient to bacterial translocation, gastric paresis and blood and fluid loss. Any patient that is post-anesthetic, post-operative (especially abdominal surgery – and particularly gastrointestinal), hypokalemic, suffering from gastrointestinal, reticuloendothelial, or neuromuscular diseases, or on narcotic analgesics has a probability of having gastroinestinal paresis. Ileus predisposes the patient to bacterial and endotoxin translocation, poor intestinal nutrient digestion and absorption, gastrointestinal ulceration and vomition. The patient should be ausculted at least three times daily for bowel sounds. Ileus is best treated by nasogastric tube suctioning and relief of fluid and gas accumulation. This reduces vomiting and subsequent aspiration pneumonia. In non-pancreatic patients, metoclopromide (1mg/kg/day IV by CRI) is utilized to promote gastric and duodenal motility. The dosage of metoclopromide should be reduced by 50% if there is liver disease present in the cat. A small amount (0.6 - 2 ml/kg q1-2h)of oral glucose solution (e.g. Resorb ®)helps protect against gastric microulceration. The use of H2 antagonists is controversial, with literature supporting the idea that altering the gastric pH predisposes to gastric bacterial translocation and more severe aspiration pneumonia. Antiemetics are needed in the patient with protracted vomiting, predisposition to vasovagalreflex, altered level of consciousness, abnormal or depressed gag reflex, or abnormal breathing. particularly if the animal is critical, recumbent, or has a depressed gas reflex. Antiemetic selection should be based on the suspected mechanism of vomiting. If the vomiting is due to gastric paresis and stimulation of peripheral reception by gastric distension, metoclopromide is the agent of choice. Metoclopromide also blocks the chemoreceptor trigger zone. When drugs or toxins are stimulating the chemoreceptor trigger zone, trimethobenzamide (Tigan®) may be the agent of choice. Protracted vomiting from any etiology can be slowed or stopped by chlorpromazine or perchlorperazine (cats: 0.01 mg/kg IV q4-8 h). The cat must be volume and pressure replaced prior to administration of promazine derivatives. Hypotension and severe depression are side-effects.

Drug dosages and metabolism

Many drug dosages in the cat have been extrapolated from studies done in the dog. Cats have a greater body surface area per unit of body weight when compared to the dog and dosage extrapolation between these species can be inaccurate. The liver plays a key role in the unique metabolism of many drugs in the cat. Lipid soluble drugs (e.g. morphine, chloramphenicol, aspirin, primidone, acetaminophen, phenols, barbiturates, benzodiazepines, propofol) must be converted to water soluble by-products before excretion. Cats lack many of the hepatic glucuronyl transferases that normally enable conjugation and excretion of these drugs. Toxic levels of these drugs or metabolites can accumulate. Enterohepatic recycling can also occur in cats and this can affect cumulative plasma concentration of certain drugs such as digoxin. Hepatic acetylation is well developed in the cat, causing faster clearance of drugs such

as hydralazine and diltiazem. Procainamide requires acetylation prior to elimination, making its activity more predictable in the cat when compared to the dog. Many of the published dosage recommendations by manufacturers are based on drug blood levels and clinical signs of toxicity. Unfortunately, there is scant information on the metabolic responses required for drug conversion and elimination by cats. The dosage recommendations for cats in the veterinary medical literature are often based on clinical experience and anecdotal information. When administering any drug to the cat, the proper dosage, route of administration, and dosing interval should be confirmed. In addition, based on the route of elimination, dosage and dosing intervals must be altered in cats with renal or hepatic disease. It is always wise to review current drug dosages in critical animals that have polypharmacy. Errors in calculation can lead to devastating complications. A list of drugs that lead to catastrophic consequences with overdose should be posted in the ICU, and dose calculations and milliliter amounts administered should be reviews at each dosing. The following drugs should begin this list: insulin, digoxin, aminoglycosides, dopamine, dobutamine, nitroprusside, promazine drugs, metronidazole, and phenobarbitol.

Nutrition

Being carnivores by nature, cats require no carbohydrates but need high levels of meat-based protein. The cat's protein requirement is 50% higher for growth and over 100% for maintenance as compared to the dog. This is due to the presence of comparatively persistent, increased activity of hepatic proteolytic enzymes (transaminases and deaminases).(Rogers,1977) Cats require dietary sources of arginine. Arginine is required for normal protein synthesis and ammonia detoxification. Domestic cats lack intestinal pyrroline-5-carboxylate synthase, which is required for the production of an arginine precursor, ornithine. A urea cycle intermediate, arginine converts ammonia to urea. Cats can develop severe hyperammonemia from anorexia or ingestion of an arginine free meal.(Morris, 1978). Arginine has other important roles that include: increasing endocrine secretagogue activity, improving nitrogen retention, acting as a substrate for nitric oxide production, reducing nitrogen loss in post-operative patients, enhancing collagen deposition in wounds, enhancing T-cell function, and the growth of lymphocytes.(Babul, 1994) Cats also require a dietary source of taurine. Cats cannot synthesize enough from dietary precursors to meet obligate intestinal loss. The cat uses only taurine for bile salt synthesis (in comparison to dogs that can substitute glycine), causing an ongoing obligate loss of taurine with excreted bile salts . Taurine deficiency has been proven to cause dilated cardiomyopathy and retinal degeneration. Cats do not have the ability to convert beta-carotene to active vitamin A (retinol). Cats lack dioxygenase enzymes in the intestinal mucosa that splits the beta-carotene molecule to vitamin A aldehyde (retinal). Preformed vitamin A must be ingested or administered as neither dietary nor intravenous beta-carotene can prevent the development of vitamin A deficiency and its consequences of blindness. Since the cat cannot convert tryptophan to niacin, the niacin requirement in the cat is about four times that of the dog. Animal tissue is high in niacin and this requirement is normally met by ingestion of a high meat diet. Arachidonic acid is needed in the feline diet, since cats cannot synthesize it from linoleic acid in comparison to the dog. Arachidonic acid is an essential fatty acid required for maintenance of cell wall integrity and can be found in diets containing animal source fats. Essential fatty acids should constitute 1% of the diet dry matter. Fatty acid deficiency results n poor hair coat quality and poor tissue integrity. The nutritional requirements of the critically ill animal should be addressed early to minimize tissue catabolism and development of hepatic lipidosis. Meat-based diets should be selected that provide a good quality protein, vitamin A, thiamin and niacin. The diet should be adequately supplemented with taurine and arginine. When the food is warmed prior to feeding or highly aromatic foods are fed, the palatability of the food offered to stressed and ill cats is often increased. Initial support, after rehydration, can be done with an intravenous 3.5% amino acid solution with glucose in a balanced electrolyte solution (Freeamine® - requires the addition of glucose; Procalamine® - has 3% glycerin). This can be given as a portion of the maintenance fluids. The patient is weaned onto enteral nutrient by giving small amounts of glucose and electrolyte solution (e.g. Resorb ®). Small amounts are given frequently to test how well the animal tolerates the fluid in the stomach. When vomiting does not occur, the animal is weaned onto enteral nutrition. If the patient is unwilling to eat, then feeding must occur either by force feeding or by tube feeding. Force feeding can prove to be very stressful to critical cats and is not to be attempted if there is depressed mentation, difficulty swallowing, or potential of stress induced complications. Tube feeding is done by orogastric intubation, nasoesophageal intubation, gastrostomy, jejunostomy, or esophagostomy intubation. The author prefers esophagostomy intubation, finding it to be easily accomplished and well tolerated by the patient. Most of these animals will have nasal oxygen tubes, making occlusion of the other nostril by nasoesophageal tube a profound discomfort. Whichever tube method is selected, nutritional support is first provided by a dilute solution of low volume. If the animal experiences vomiting with this procedure, the clinician should first consider decreasing the concentration or the volume being administered prior to administering antiemetics. The cat can then be weaned onto bolus administration of the solution prior to discharge if tube feeding is still required. In the patient with pancreatitis or gastroduodenal pathology, it is best that nutrients do not pass from the stomach into the duodenum, since this can stimulate pancreatic secretions. Total parenteral nutrition has been the method of choice in the past, however enteral feeding is recommended as soon as possible. Should this cat be explored for any reason surgically, a jejunostomy tube should be place so that enteral nutrition that by-passes these structures can be utilized. Feeding through the J-tube is accomplished as described above for the other tubes.

Pain control

Pain can manifest in animals by mental depression, tachycardia (in the dog and rarely in the cat), restlessness, and/or and irritable attitude. It is vital to the maintenance of cardiovascular function and the mental well being of the cat to provide pain control. In the critically ill cat, it is best to titrate analgesics and sedatives to effect, as responses are variable and can be affected by underlying renal and hepatic dysfunction. For mild to moderate pain control, butorphanol 0.2-0.8mg/kg IV q 2-6 hrs is given initially. For control of severe pain, the combination of injectable opioids oxymorphone (DuPont) 0.05-0.1mg/kg IV or morphine (Steris Labs) 0.1mg/kg IM with diazepam (Steris Labs) 0.2mg/kg IV is effective and reversible. In the hemodynamically stable feline, acepromazine (Fermenta Animal Health) 0.02 - 0.1 mg/kg IV can be combined with the narcotic. Fentanyl patches (Duragesic, Janssen), 25mcg/hr patch per cat, provide therapeutic blood levels after approximately 12 hours lasting up to 72 hours. The use of regional anesthesia (nerve blocks or infusion in body cavities), epidural analgesia, and/or pre-emptive analgesia with administration of injectable opioids given prior to surgical intervention are alternative approaches to consider. Lidocaine administered subcutaneously or intravenously can cause methemoglobinemia and Heinz body anemia in the cat and careful dosing is required.

Nursing care and patient mobilization

The veterinarian is only as good as the nursing staff. A complete description of all nursing concerns in the critical animal is worthy of a textbook. Time should be taken to teach the nurses how and what to monitor and how to perform therapeutic procedures. The nursing staff should speak kindly and softly when working with these animals, using minimal restraint to accomplish any task. The animal must be removed from the cage and thoroughly examined at least twice daily. Changes in their physical condition, even pulmonary function, may be very subtle and occur rapidly.

Catheter sites must be checked daily, and each catheter labeled appropriately to avoid confusion of lines and misuse of the tubes. When catheters are removed, the tips should be saved for culture and sensitivity. The paw distal to the peripheral catheter must be checked multiple times during the day for evidence of paw edema, requiring re bandaging of the catheter. Elizabethan collars are often necessary for catheter security as well as an aid in handling aggressive animals. Hypothermia is a component of most critical cat diseases. The cat is initially warmed passively. Warm fluids are administered and the cat can be wrapped in towels and blankets. Once intravascular volume has been replaced, warm water circulating blankets can be used, making sure the cat can move off of the heating blanket if it desires.

The recumbent patient must be turned every 4 hours or maintained sternal when possible. Physical therapy will assist in maintaining muscle tone and blood flow to the limbs. Urine scalding and fecal soiling is prevented by providing absorbent bedding and cleaning the animal immediately.. Results of monitoring procedures, treatments and observations should be carefully recorded. The nurse must feel confident and comfortable about reporting observed changes to the veterinarian. The staff must anticipate complications and be prepared for immediate intervention. Animals in severe respiratory distress should have an endotracheal tube and laryngeoscope available by the cage. Diazepam or pentobarbitol doses should be predetermined and available for the patient with cluster seizure activity.

Wound care/bandage change

When the patient's underlying disease requires wound debridement or surgical correction, the incision site or wound should be examined daily to insure that appropriate healing is occurring. Anytime a bandage is moist, it must be changed. Any subcutaneous distal limb edema associated with fasciitis is controlled and reduced by compression bandages. These wraps are removed and the subcutaneous sites examined daily.

Tender loving care

The mental health of the patient is often as important as the physical health. It must be remembered that these animals are pampered pets. Visits by the owners are to be encouraged when it benefits the pet and having familiar items in their cage will make the owner feel better, if not the animal It is important for house cats to have fresh litter and a place for their food away from their litter box. Blankets or bedding makes them more comfortable. Providing a box for the cat to hide in or using other techniques of obstructing their view of strange animals, when their condition allows, reduces their level of fear and stress. The animal's biorhythms become disturbed when the ICU lights are on 24 hours per day. When possible, it is good to turn down the lights to stimulate night and promote sleep.

References provided upon request.