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Intravenous fluid therapy provides numerous benefits to our sick patients, which include providing for rehydration, correction of hypotension, correction of electrolyte disturbances, a route for administering parenteral nutrition, and a route for the administration of many important medications. Following along the lines that no medication is perfectly safe, this paper will review the types of various complications that can arise from intravenous fluid therapy. Some of these problems are minor while others can be of life threatening proportions to the patient.

**Thrombophlebitis, catheter sepsis, and catheter embolus**

These are three of the most catastrophic complications associated with indwelling intravenous catheters. All catheters support infection and may trigger thrombophlebitis. There are differences between plastics as cannulas made of fluoroethylene propylene (Teflon) are associated with lesser incidence of venous irritation than those made of polyvinyl chloride and tetrafluoroethylene. Technique of catheter insertion and its maintenance also play critical roles on the incidence of catheter irritation and infection. The contributing factors include inadequate preparation of the cannula insertion site, unhygienic maintenance of the patient and the catheter site, the infusion of highly irritating medications, not providing for catheter patency, and leaving the catheter in one site for too long. The infusion of hypertonic and highly acidic or alkaline solutions can set the stage for phlebitis. Strict asepsis must always be used at the time of catheter insertion and its subsequent use throughout hospitalization.

The signs of phlebitis include redness, pain, and warmth at the catheter insertion site. Body temperature is usually normal and there are no signs of systemic involvement unless it serves as a focus of secondary infection and bacteremia (see below). Thrombus formation will change the vein texture from soft and pliable to firm and cord-like. Phlebitis and thrombophlebitis can be detected early through daily inspection of the catheter insertion site. The presence of any signs of phlebitis calls for the immediate removal of the cannula. It is highly recommended that any such catheter be cultured for microbial identification and antimicrobial sensitivity. Catheter related sepsis will cause systemic signs including mental depression, generalized weakness, anorexia, and often fever. Inspection of the cannula insertion site usually shows signs of phlebitis. Action measures call for removing the catheter and culturing it and the patient’s blood. The main problem with detecting catheter related sepsis in the critically ill patient is that its signs can easily be masked by the primary underlying disease process. The clinician caring for such patients must be able to respond to any sudden or subtle signs of deterioration and act accordingly.

Catheter embolus occurs when the catheter or any fragment becomes free and enters the circulation to lodge in the heart, lungs or vena cava. This occurs when the catheter is withdrawn before the needle is removed from the skin or from failure to secure the needle after it is withdrawn from the skin. It also occurs during bandage or catheter removal when the plastic intravenous cannula is inadvertently cut free. If this should occur, a tourniquet should be immediately applied just proximal to the entry site. Most catheters are radiopaque and therefore radiographically visible. Surgical removal should be done as soon as possible if the catheter is deemed retrievable. The main consequence of catheter embolus is large thrombus formation and its attending complications. In fortuitous situations, the thrombus might be of no consequence.

**Subcutaneous extravasation**

This is a frequent and sometimes serious complication associated with intravenous fluid therapy. It can result from faulty cannula insertion technique or excessive movement of the catheter site allowing for its subsequent migration. Excessive injection pressures can also play a causative role. The earliest and least consequential signs include: (1) swelling of the surrounding soft tissues, (2) failure to withdraw blood into the catheter, and (3) a change in the resistance pattern accompanying catheter flow. If caught early, the extravasated fluids will usually be absorbed without consequence. However, the escape of large quantities of fluid or irritating solutions can lead to tissue sloughing which can have remarkable consequences for the patient and clinician-pet owner relations.

**Circulatory overload**

This is one of the most serious complications of intravenous fluid therapy. It is usually iatrogenic through the clinician’s failure to provide fluids according to the patient’s actual pathophysiologic status. Clinical circumstances allowing for this include: (1) rapid
administration of large volumes, (2) cardiac dysfunction, (3) oliguria/anuria, (4) anemia, (5) hypoproteinemia. The most important preventative measure entails a through daily evaluation of the patient. Essential parameters include: attitude, urine output, respiratory rate, skin texture, and body weight. Central venous pressure measurements will act as an informative gauge for intravascular fluid overload. Pressures of 10-12 cm of water are indicative of impending overload. Levels of 16 or more indicate that overload is already taking place. Volume resuscitation can be a frequent cause of circulatory fluid overload even if the administered dose is within acceptable guidelines. The pathophysiologic settings for this include: (1) failure to recognize that normal or low normal hematocrit and total protein readings in a volume-depleted animal might actually mean anemia and hypoproteinemia in the rehydrated patient; (2) the animal in septic shock might have depressed cardiac and vascular tone to accommodate the intravenous fluid load; and (3) the administration of excessive amounts of hypertonic solutions (saline, mannitol, dextrose, colloids) to patients with cardiac dysfunction.

Although probably not contributing to circulatory overload, the administration of large fluid loads to patients with either pulmonary hemorrhage or acute respiratory distress can cause serious worsening of respiratory function and perhaps the patient’s eventual demise. The same applies to animals suffering from acute brain trauma where the disturbance in circulatory autoregulation might allow for cerebral edema formation. These are best avoided by allowing for a slower rate of parenteral fluid administration at just below maintenance levels once the patient’s blood pressure is restored to safer levels. Providing fluid therapy for the oliguric/anuric patient first entails replacing dehydration volumes followed by precise maintenance amounts as determined by insensible losses (10-20 ml/kg per day), measured urine output, and extra losses through vomiting and diarrhea. Anuric patients will eventually require dialysis in order to maintain adequate fluid balance. Anemic patients are predisposed to circulatory overload because the heart is already strained in its attempt to oxygenate the body’s tissues. This is shown with the tachycardia that commonly occurs in anemic patients. An expanded plasma component within the intravascular space also occurs in these patients. Adding large volumes of crystalloid to such patients can often cause pulmonary edema and/or pleural effusion. Therefore, it is prudent to administer intravenous fluids cautiously to animals with chronic anemia.

Hypoproteinemic patients are deficient in plasma oncotic pressure thereby favoring Starling’s forces toward the interstitial space. The administration of excessive crystalloids can quickly worsen edema and effusion accumulations. These patients will benefit greatly from colloid therapy.

**Acute hypotonicity**

Plasma hypotonicity occurs in conditions that cause either the accumulation of excess water within or a loss of sodium from the intravascular space. The rapid administration of 5% dextrose solution can cause significant declines in serum osmolality (normal, 290-310 mosm/L). Most of 5% dextrose solution will exit the plasma space within minutes after its infusion and enter the interstitial and intracellular spaces. The brain is the organ most likely to suffer because the cerebral edema that occurs is poorly accommodated by the bony calvarium. An altered state of consciousness leading to coma with or without seizures are the catastrophic consequences of hypotonic encephalopathy. Treatment in acute situations (where the hypotonicity occurred within a period of and not exceeding 24 hours) such as this require the administration of sodium chloride solution in conjunction with diuretic therapy in order to promote both sodium gain and water loss. The goal of acute therapy is to increase the serum sodium level at a rate of 2.5 mmol/L per hour but not to exceed 20 mmol/L over the 24 hour period. Hyponatremia occurring over more than 24 hours should be corrected slowly with the serum sodium concentration increasing at no more than 10-12 mmol/L per 24 hours.

**Acute hypertonicity**

Increases in plasma tonicity occur in those conditions causing either sodium gain or excessive water loss. Acute hypernatremia with ECF (extracellular fluid space) volume expansion is usually seen in patients receiving excess amounts of hypertonic saline or sodium bicarbonate solutions. Although intravascular overload and its consequences can eventually occur in situations of sodium gain, the most immediate adverse effects will involve the brain where the hypertonic plasma space will draw water from the brain parenchyma resulting in neuronal dehydration and widespread brain hemorrhages. The latter lesion occurs with the tearing of the small pial blood vessels as the dehydrated brain shrinks away from the calvarium. The clinical signs are similar to those accompanying hypotonic encephalopathy. Acute hypertonic brain dysfunction calls for removal of the excess sodium with diuretics or dialysis followed by replacement of fluid losses with 5% dextrose in water or 0.45% sodium chloride solutions. The correction of acute hypertonicity can occur over a period of hours so long as the condition occurred over a period of 24 hours or less. More chronic conditions (occurring over more than
24 hours) should be corrected slowly over a 48-72 hour period at a rate of change in serum sodium not to exceed 10-12 mmol/L in 24 hours.

Hyperkalemia and hypokalemia

Hyperkalemia most commonly occurs in patients with oliguric acute renal failure and adrenocortical insufficiency. It can also occur with certain forms of metabolic acidosis through potassium translocation from the ICF (intracellular) to the ECF space. Iatrogenic hyperkalemia occurs when potassium chloride solution is infused too rapidly or when it is inadequately mixed in the vehicle solution. Most texts list the maximal rate of infusion as 0.5 mmol/kg/hr, but in extreme hypokalemic conditions the amount infused can be increased to 1.0 mmol/kg/hr. Slower rates should be used if the patient is oliguric. Infusion problems can occur when the infusion rate is set when there is a kink in the catheter which subsequently happens to straighten out thus allowing for a too rapid infusion.

Hypokalemia is one of the most common serum electrolyte disturbances in small animal patients. Potassium losses can occur through the gut or kidneys or result from translocation of extracellular potassium into the intracellular space as seen with metabolic alkalosis and the administration of insulin. Iatrogenic hypokalemia accompanies the administration of parenteral fluids that are devoid of adequate amounts of potassium chloride. The 4 mmol/L found in lactated Ringer’s solution are grossly inadequate. Normokalemia can usually be maintained with the infusion of maintenance solutions containing 7-8 mmol KCl/250 ml. It is essential that all containers of intravenous fluids be clearly labelled for any additives added to the solution.