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“Ten Things You Should Know About Fluid Therapy”

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The Development of the Fluid Therapy Plan

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Introduction

Fluid therapy is an important component of everyday veterinary medicine. In order for fluid therapy to be effective, we must have a basic understanding of what we are trying to achieve. In veterinary medicine, fluid therapy is a supportive measure. The underlying disease process that has caused a fluid or electrolyte imbalance must be diagnosed and managed appropriately.

The ultimate goal of any fluid therapy is to improve or maintain the delivery of oxygen to tissues. Oxygen delivery (DO₂) is dependant on cardiac output (CO), hemoglobin (Hb) concentration in blood, and the arterial oxygen concentration of arterial blood.

Effectively using fluid therapy involves thinking about fluid therapy as more than just treating the symptoms of circulatory dysfunction. This is important because the presence or absence of symptoms of circulatory dysfunction and shock does not correlate well with oxygen delivery to tissues. How do we overcome this problem, and why is it important? To overcome this problem, and to arrive at a rational approach to the management of tissue oxygen delivery, rather than directing our treatment at the symptoms of circulatory dysfunction and shock, we must understand the determinants of tissue perfusion and tissue oxygen delivery, and then arrive at an approach to fluid therapy that meets the demands of the tissue under any given circumstance.
Tissue Perfusion and Oxygen Delivery

Why are concentrating on tissue perfusion and oxygen delivery?

1. Adequate delivery of oxygen to tissues is essential to ensure adequate cellular energy production. Without adequate cellular energy production, cellular function will decline. Stated another way, oxygen is required in aerobic biochemical pathways to produce biological energy from energy substrates. Inadequate tissue oxygenation results in anaerobic metabolism. Anaerobic metabolism leads to a reduction in cellular energy production (ATP production), and an increase in lactic acid production from pyruvate. A reduction in ATP production leads to a reduction in activity of cell membrane pumps, loss of cell membrane integrity, osmotic stability, and cell lysis. In addition, tissue hypoxia and anaerobic metabolism leads to the development of acidosis, which further blunts normal metabolic pathways within cells and organs.

2. Although cardiac and respiratory functions are directly measurable, tissue perfusion and oxygenation are not quantifiable. However, tissue perfusion and oxygenation are of greater consequence in terms of outcome. Inadequate tissue perfusion leads to tissue hypoxia, which, when extensive in degree or protracted time, produces organ dysfunction, multiple organ failure, and death. When the early manifestations of shock are alleviated by fluid therapy that is insufficient to correct poor tissue oxygenation, the resultant oxygen debt may not be recognized until the appearance of organ dysfunction or failure occurs - often 2-3 days after the initial appearance of shock. Symptoms of the organ dysfunction most commonly include vomiting, diarrhea, and anorexia, but often progress to include ARDS, sepsis, acute cardiac failure, renal failure, hepatic failure, DIC or coma.

In summary, these two points reinforce the statement that the delivery of oxygen to tissues is required to ensure normal organ function, and to prevent mortality. This statement underlies the focus of our treatment and stabilization of the critical patient.
How is Tissue Oxygen Delivery Determined?

Tissue oxygen delivery is dependent on five (5) factors. These factors and their relationships with each other are summarized in the equation below.

\[
\text{Oxygen delivery (DO2)} = \text{CO} \times [\text{Hb}] \times \text{SaO2} \times 1.3 + 0.03 \times \text{PaO2}
\]

\[= \text{Cardiac output} \times \text{hemoglobin saturation} + \text{partial pressure of oxygen in the blood}\]

From this equation, we can see that there is a lot more to treating a patient with intravenous fluids than just giving intravenous fluid therapy with an isotonic crystalloid – there are other things to consider as well – things like PCV or hemoglobin content of the blood, colloid oncotic pressure, heart rhythm to name a few. The aim of this presentation is to review the pathophysiology and treatment of shock, and to then follow-up on the completion of our fluid therapy plan – to ensure tissue oxygen delivery is optimized.

How Tissue Oxygen Delivery Affects Fluid Therapy

Having considered the determinants of tissue oxygen delivery, a rational approach to fluid therapy can be made with the knowledge that:
1. The patient requires a functional respiratory tract
2. The patient requires adequate cardiac output
3. The patient requires adequate hemoglobin concentrations
4. The patient requires appropriate vascular tone to ensure oxygenated blood is received by the tissues
5. The patient requires adequate blood flow through capillary beds to enable oxygenated blood to be extracted into the tissues

The aforementioned requirements for tissue oxygen delivery as they apply to intravenous fluid therapy are summarized in “The Ten Rules of Fluid Therapy”
The Ten Rules of Fluid Therapy

As stated previously, the requirement of a patient for intravenous fluid is common in veterinary practice. If we understand firstly that the goal of our fluid therapy is to improve tissue oxygen delivery, and secondly, that each patient is an individual, with individual fluid therapy requirements, we must develop a plan or checklist to ensure we do not overlook any component of the fluid therapy plan. This has lead to the development of ‘The Ten Rules of Fluid Therapy’.

Rule No 1: Diagnose and Manage Shock

Shock is a common occurrence in small animal veterinary medicine and surgery. Shock is a condition whereby the blood and oxygen delivery to our tissues becomes disrupted, due to some occurrence either within the body - such as inflammation or infection - or some traumatic or stressful event outside the body, that affects the nervous system and body function, such as trauma, stress, or illness. The diagnosis of shock is a clinical one - meaning it is based on things you can see.

The time-frame of this lecture does not allow us to discuss shock in great detail, but essentially, in order to treat shock, we must know what happens in shock - because the treatment is most effective if we know what we are treating. So let’s have a look at what happens in shock!
1. **Diversion of blood to the Heart, Lungs, Brain, and Voluntary Muscle** - this occurs early in shock, as a result of the “stress response” - the sympathetic nervous system and adrenaline rush that occurs immediately following a stressful, traumatic, or tissue injury or inflammatory event in the body. Blood vessels in the kidneys, gut, liver, and skin become constricted, raise our blood pressure, and divert blood to the “essential” organs for basic life function - the heart, lungs, brain, and muscles. The down-side of this response is that tissues in the kidneys, gut, liver, and skin become deprived of oxygen. Symptoms of early shock therefore include
   a. **Elevated heart rate**
   b. **Strong pulses**
   c. **Elevated respiratory rate**
   d. **Increased level of alertness**
   e. **Good mucous membrane characteristics**

2. **Reduced oxygen delivery to Kidneys, Gut, Liver, and Skin** - vasoconstriction in these tissues (kidneys, skin, gut and liver) early in shock results in reduced oxygen delivery to these organs. This reduced oxygen delivery causes blood vessels to eventually dilate, rather than constrict. Progressive blood vessel dilatation means that blood in the body now has to be shared, between not only the heart, lungs, brain and muscle, but is now also diverted to the kidneys, gut, liver and skin as well! The problem with this is that the body does not have enough blood in it to fill all of these blood vessels at once if they are all dilated - this causes a decrease in blood flow to all of our organs. The symptoms we now get in shock are
   a. **Elevated heart rate**
   b. **Weaker pulses**
   c. **Elevated respiratory rate**
   d. **Decreased level of alertness**
   e. **Poor mucous membrane characteristics**

3. **Development of organ dysfunction and organ failure** - if shock is not treated, the reduced blood flow mentioned above will result in organ dysfunction and failure, activation of the blood clotting system, blockage of our blood vessels, and eventually, death.

Treatment for shock initially involves immediate treatment with intravenous fluid therapy, but also involves assessment and management of the patient airway, respiratory tract, and many of the subsequent steps involved in the rules mentioned above, including management of anemia, low serum protein levels, electrolyte analysis etc. To begin with, following assessment and correction
or the patient airway and breathing, we will reach for intravenous fluid therapy. The most common fluid used is lactated Ringer’s solution, because it has a similar composition to normal extracellular fluid. Doses vary, but recent human experience and animal trials favor the following protocol

### The Small Volume Resuscitation Protocol – Lactated Ringer’s Solution Protocol

The practice of small volume resuscitation involves the administration of the traditional shock rates of fluid therapy (for example Hartmann’s @ 60-90 ml/kg/hr in dogs; 30-50 ml/kg/hr in cats) in small aliquots of between 5-10 ml/kg given over 5-10 minutes, followed by patient reassessment of heart rate, mucus membrane color etc. If the patient is still manifesting clinical signs of shock, a further aliquot of shock rate fluid is given. The process of fluid administration and patient reassessment is repeated until the patient is stable – that is, they have a normal heart rate, respiratory rate, mentation, and mucous membrane characteristics, and are beginning to produce urine. This resuscitation protocol allows titration of shock rate fluid to clinical effect, rather than using large-dose empirical therapy. The advantages of this fluid therapy over traditional empirical volume administration, includes reduced accumulation of interstitial edema post shock fluid therapy, lower risk of hypertension in patients with liver or splenic trauma, reduced incidence of pulmonary edema – especially in cats, and closer patient monitoring during the critical stabilization period. Addition of a synthetic large-molecular weight colloid such as hydroxy-ethyl starch @ 3-5 ml/kg IV over 10 minutes at the start of resuscitation, can reduce the volume of crystalloid used by 40-50%.

### Rule No 2: Manage Blood Hemoglobin Concentrations

Hemoglobin is the most important carrier of oxygen in our blood. Hemoglobin binds large amounts of oxygen as blood passes through the pulmonary circulation. Without hemoglobin, we would die of hypoxia, even if we were breathing 100% oxygen. Normal hemoglobin concentration is between about 12 g/L and 18 g/L or about 1/3 of the normal PCV (35-55% for the dog; 28-50 for the cat). Once the hemoglobin level drops below about 7-10 g/L (PCV < 20-24%), body tissues begin to receive less oxygen than they need for normal metabolism. This results in cell and organ dysfunction, and ultimately, organ failure.

As we can see, hemoglobin concentration in most patients is generally directly related to the PCV/TP. Patients with anemia are unable to deliver adequate oxygen to cells. This in turn results in organ dysfunction.
In hospitalized patients requiring intravenous fluid therapy, the packed cell volume AND total protein levels should be evaluated on admission to the clinic for treatment. We routinely monitor PCV/TP every 1-6 hours in acutely ill patients, and thereafter every 12-24 hours once they are stable i.e. the patient shows no evidence of being in shock. The normal PCV is between 38-55% in dogs, and between 28-50% in cats. An elevated PCV may occur in patients immediately following trauma or diarrhea (due to splenic contraction in shock), or in patients with brain disease, cardiac disease, renal tumors, or bone marrow disease. A decreased PCV may occur in patients with hemorrhage, renal disease, immune-mediated disease, or severe illness.

**Treatment for elevated PCV** includes provision of intravenous fluid therapy, and in some cases, phlebotomy. These measures are required to improve blood flow – as patients with an elevated PCV have, understandably, very thick, viscous blood, that does not flow readily through small blood vessels like capillaries. If patients with excessively elevated PCV are left untreated, the thick viscous blood in their blood vessels will begin to clot and clog/block small arterioles and capillary beds. This in tum results in a reduction in tissue oxygen delivery, and subsequent organ damage.

Treatment for a low PCV usually involves replacement of red blood cells with transfusion therapy. The aim of red blood transfusions is to raise the patients’ PCV to a level of approximately 27%. But when should we transfuse? The answer lies in the phrase “look at your patient”

- Patients that are anemic have less oxygen carrying capacity in their blood
- If anemia develops slowly – over a period of days to weeks – as it does in slow chronic blood loss, or chronic renal disease, immune-mediated hemolytic anemia or some bone marrow neoplasia cases, the patient may have time to “adapt” to a slowly decreasing PCV. Chronic hypoxia due to the slow development of anemia results in an adaptive response within red blood cells - an increase in the red blood cell enzyme 2,3 diphosphoglycerate (2,3 DPG), which causes a decrease in the affinity of hemoglobin for oxygen, facilitating oxygen delivery to tissues. This means that potentially, an animal may look reasonably healthy – albeit with quite a low PCV – even as low as 14-15%
- If anemia develops rapidly – over minutes, hours, or over less than a few days – the body does not have the time to “adapt” to reduced oxygen carrying capacity in the blood. Animals with rapidly developing anemia often show immediate or dramatic signs of lack of oxygen.
- Patients that are suffering from oxygen deprivation show clinical signs. These signs include:
  - Increased rate and depth of respiration; occasional panting
  - Weakness or collapse
  - Increased heart rate, possibly strong OR weak pulses, arrhythmias may be present
  - Depression or Obtundedness, lethargy
Pale mucous membranes
Signs of organ dysfunction
- Anorexia or reduced appetite
- Vomiting
- Diarrhea
- Reduced urine output
- Weight loss
- Lethargy and ataxia

Patients showing ANY clinical sign listed above require transfusion therapy with as little delay as possible in order to normalize their clinical signs.

How much blood should we give when transfusing a patient? The short answer is that in MOST cases, transfusion to a MINIMUM PCV of 27-30% is required to alleviate clinical signs of anemia. The longer answer is that in some cases of severe Immune-mediated hemolytic anemia, transfusion to a PCV of 27% provides the immune system with an excess number of red blood cells, which will be prematurely destroyed, leading to increased morbidity, and in some cases mortality. In these select patients, transfusion to a PCV of approximately 20-24% is considered an adequate end-point for transfusions. When administering whole blood, a useful rule of thumb is that 2 ml/kg of whole blood will raise the recipient PCV by 1% - use this as a guide to the volume of blood required to reach a desired end-point PCV in your recipient.

**Rule No 3: Normalize Colloid Oncotic Pressure**

What IS colloid oncotic pressure? Colloid oncotic pressure is the pressure - kind of like a mini-gravity force (although, technically this is not entirely correct, but it helps us imagine what’s going on) - that large molecules - most notably albumin in the blood - exert on fluid. Although cells and other proteins within the blood all contribute to the colloid oncotic pressure, albumin exerts the majority of the oncotic pull in most animals. Let’s try to imagine what would happen if there was no protein in the blood vessels - most of the fluid within the blood vessels would ‘leak out’ of the blood vessels, and equilibrate with all of the fluid in the tissues. In actual fact, if this happened, we would have a MASSIVE increase in the amount of interstitial fluid, and a MASSIVE decrease in blood volume. We would all look like big, giant, fluid filled Michelin men (persons) – AND, because our blood volume would shrink, we would all be in shock.
This is the reason why, in disease states associated with inflammation or tissue trauma, such as dog attacks, GDV’s, pancreatitis, pyometra, surgery etc., colloids such as dextran 70 (discontinued), Pentaspan (NZ) HES (Voluven) and fresh frozen plasma are necessary in order to maintain effective circulation throughout the body.

So, what does a patient with low colloid oncotic pressure look like? Outward physical symptoms of low colloid oncotic pressure are often not seen until significant reductions in colloid oncotic pressure have already occurred, and include soft swelling of the feet and limbs, neck, and gravity-dependant skin. However, early indications of a decrease in colloid oncotic pressure are frequently manifested as changes in the patient cardiovascular status – including developing symptoms of shock - a decrease in urine production, increasing lung sounds and breathing difficulty, often with falling pulse oximetry readings, associated with fluid leakage out of blood vessels, into the lungs. Note that these are things we can measure in our practices!

Laboratory indications of colloid oncotic pressure can include measurement of serum albumin level, and serum total protein (although serum albumin is considered better, since most colloid oncotic pressure is provided by albumin).

Extensive human evidence in peer-reviewed literature suggests that EARLY therapy with colloids can PREVENT a loss of colloid oncotic pressure and that this is associated with improved patient survival and reduced hospital stays. It is therefore recommended to BEGIN colloid therapy within 6-12 hours of hospitalization in patients with the following diseases:

1. The “P” diseases – pyothorax, peritonitis, pancreatitis, prostatitis, pyometra, parvovirus etc.
2. Diseases associated with tissue injury or inflammation – such as the “P” disease, intestinal diseases such as hemorrhagic enteritis, GDV, intestinal rupture; traumatic conditions such as road traffic trauma associated with extensive soft tissue damage, dog bite wounds, pig-hunting injuries and septic disease processes.

Once colloid oncotic pressure has fallen, associated with either clinical signs of a reduced COP, or a low albumin level or both, correction of low colloid oncotic pressure and serum albumin concentrations is usually best achieved through the following fluid therapy options:

- Transfusion of Fresh Frozen Plasma – given at doses of between 10-20 ml/kg/day, depending on the underlying disease process.
- Infusion of synthetic colloids such as Dextran 70, HES, or pentaspan – given at 10-20 ml/kg/day (up to 25 ml/kg/day with HES). In patients showing symptoms of shock due to poor perfusion caused by diseases that lead to a loss of serum proteins (such as hemorrhagic diarrhea, pancreatitis, pyothorax, peritonitis etc) part of the fluid therapy plan in the treatment of shock should include bolus therapy with a synthetic colloid such as...
as HES, given at 3-5 ml/kg IV over 10-15 minutes, followed by a maintenance infusion rate of 10-20 ml/kg/day

**Rule No 4: Correction of Electrolyte Imbalances**

Electrolytes are essential for normal cell and organ health and function. Electrolytes such as calcium, potassium, sodium, chloride, and acid base balance should all be measured daily, and corrected where appropriate. Why? Here’s a sample of what these electrolytes do, and what happens when abnormal concentrations of electrolytes are found in the body.

1. **Potassium**
   a. Decreases in serum potassium cause the following
      i. Metabolic acidosis
      ii. Muscle weakness
      iii. Gut stasis contributing to intestinal ileus and gastroparesis - vomiting!
      iv. Respiratory muscle paralysis
      v. Ventricular arrhythmias
      vi. Carbohydrate intolerance, impaired insulin release, weight loss
   b. Hyperkalemia causes the following
      i. Muscle tremors, followed by muscle weakness
      ii. Bradycardia, ventricular fibrillation, and death

2. **Sodium**
   a. Hyponatremia causes
      i. Cell swelling and cell death
      ii. Most symptoms seen involve the central nervous system, and include seizures, depression, mild lethargy, nausea, and slight increases in bodyweight
   b. Hypematremia causes
      v. Cellular dehydration
      vi. Central nervous system depression
      vii. Lethargy, depression, coma, seizures
      viii. Vomiting
      ix. Death
      x. Tachycardia, dehydration, weak pulses

Obviously, these lists are not exhaustive. However, the conditions outlined above underline the importance of needing to think about electrolyte balance. In addition, nearly all patients on
intravenous fluid therapy for longer than 24 hours require the addition of potassium to their intravenous fluids. Patients on intravenous fluid therapy for longer than 24 hours also require the addition of free water to their fluid therapy regime. This is accomplished by using 0.45% NaCl + 2.5% glucose. Therefore, if you see a patient in hospital on IV fluid therapy with Hartman’s for greater than 24 hours - ask the question - “is this the right fluid therapy for this patient?”

**Rule No 5: Correct Acid-Base Imbalances**

The topic of acid-base balance is HUGE, and at first, it can be a little confusing. So let’s take a little while to look at the importance of acid-base balance, and at how we can manage abnormalities with our fluid therapy.

Acid-base balance refers to the maintenance of hydrogen ion concentration in extracellular fluid (ECF) at or around 40 nEq/l, (pH = 7.4) a level necessary for the normal function of many body enzyme systems. The hydrogen ion is very reactive, and will bind or dissociate with dissociable groups, changing the structure, charge and configuration of molecules involved in the body’s enzyme systems. For this reason, the concentration of hydrogen ions in the body fluids must be kept constant, so that detrimental changes in enzyme function and cellular structure do not occur.

When acid-base is out of balance and the pH is low, the central nervous system becomes depressed. If the pH becomes as low as 7, the central nervous system becomes so depressed that the patient can (but not always) become comatose. At the other end of the scale, a pH above 7.45, hyper-excitability of the nervous system develops, and convulsions followed by death may result.

**Respiratory acidosis** is a result of increased carbon dioxide in the blood, brought about by inadequate ventilation. Diseases that cause inadequate ventilation are numerous, and include the following:

- Diseases of neuro-muscular junction
  - Snake bite
  - Tick paralysis
  - Botulism
  - Tetrodotoxin
  - Polyradiculoenepathy
- Diseases of the central nervous system
  - Head trauma
  - Seizures (including status epilepticus)
  - Coma or stupor
- Diseases of the respiratory tract
  - Airway disease - e.g. brachycephalic airway disease, laryngeal paralysis, bronchitis etc.
  - Pulmonary parenchymal disease - e.g. pulmonary edema, pneumonia, neoplasia etc.
  - Diseases of the pleural space - pneumothorax, pyothorax, Chylothorax etc.
  - Diseases of the chest wall - e.g. blunt-force trauma, flail chest, rib fractures, obesity etc.

The kidneys will eventually help maintain acid-base balance within 2-3 days by increasing bicarbonate absorption and excreting more hydrogen ions (metabolic compensation) - however as we can see, these conditions all represent conditions we must treat early, in order to prevent patient deterioration and death.

Hyperventilation causes a decrease in blood carbon dioxide concentration in the blood, and respiratory alkalosis is the result. This may occur in patients with cerebrovascular injury. Metabolic compensation by the kidneys results in a decrease in excretion of hydrogen ions, and increased excretion of bicarbonate ions.

Metabolic acidosis is results from a decrease in bicarbonate concentration in the blood. Diarrhea or renal dysfunction will cause loss of bicarbonate ions. Accumulation of another acid such as in the diabetic keto-acidotic patient, or in patients with poor circulation, resulting in production of lactic acid results in bicarbonate ions being “used up” as they buffer, or try to neutralize acid in the body fluids. Failure of the kidneys to excrete H+ ions in the urine will also cause acidosis.

Metabolic alkalosis is results from an increase in bicarbonate ion concentration in the blood. Excessive vomiting may cause alkalosis, as hydrogen ions are lost from the stomach; this is the most common cause of metabolic alkalosis. Treatment of metabolic alkalosis includes replacement of potassium and chloride via fluid therapy and eliminating the cause of the metabolic alkalosis.
As we can see, there are many different causes of acid-base problems. In general, however, three things are required in the treatment of acid-base disorders. The first is to maintain normal respiratory function, through provision of supplemental oxygen, and ventilation assistance when required. The second involves administering appropriate intravenous fluid therapy – involving the “Ten Rules of Fluid Therapy” to improve tissue perfusion and oxygen delivery to all body tissues. The third involves appropriate management of the underlying disease process.

**Rule No 6: Correct Hydration Deficits**

**Assess and correct patient hydration status** – using physical parameters. The presence of dry or tacky mucous membranes, prolapsed third eyelids, skin tenting, depression, lethargy and elevations of PCV or TP all may indicate dehydration. In addition, the presence of reduced oral intake of fluids and nutrition prior to hospital presentation may provide an early indication of potential dehydration.

Hydration deficits should be corrected prior to any anesthesia if possible. Dehydration should be corrected using a combination of isotonic crystalloid fluids such as lactated Ringer’s solution, in combination with a synthetic colloid such as hetastarch (if dehydration is compounded by low plasma protein or albumin, or if the patient is losing significant amount of fluid, such as in hemorrhagic gastroenteritis, dog bite wounds etc. Fluid volumes for rehydration should be based on the following equation:

$$\text{Fluid to be administered (ml) } = \text{Bodyweight (kg)} \times \% \text{dehydration}$$

Generally, hydration deficits should be replaced over several hours, to allow proper rehydration of the patient. A general rule of thumb is to rehydrate patients within 4-6 hours if dehydration has developed acutely e.g. due to acute gastroenteritis - and to rehydrate patients over 12-24 hours if hydration deficits have developed over a longer period of time i.e. over a few days. Frequent weighing of the patient may allow early detection changes in hydration status within the hospital setting, particularly in patients that are losing large amounts of fluid through vomiting, diarrhea, fever, and skin wounds.
Rule No 7: Provide Fluid Therapy for Ongoing Losses

Patients that are unwell may have ongoing fluid requirements of between 2 and 7 times normal maintenance fluid requirements, depending on their illness – for example, a patient with chronic renal disease may require 2-3 times maintenance fluid rate to maintain hydration, whereas a patient with severe hemmorhagic diahrea or severe untreated diabetes mellitus may require up to 4-6 times maintenance fluid rates, depending on the amount of fluid the body is losing. The use of synthetic colloids such as hydroxy-ethyl starch or natural colloids such as plasma will reduce fluid losses into the gut or body cavities, and are usually combined with crystalloid fluids such as lactated Ringer’s solution to improve effectiveness of ongoing fluid therapy. Fresh frozen plasma is useful in assisting maintenance of effective circulating blood volume, tissue oxygen delivery, and the maintenance of coagulation factors in patients with seve re illness. We will review ongoing fluid requirements of patients with specific diseases next week in a lot more detail – but here’s some food for thought

Fluid Therapy to Prevent Renal Failure: Patients that have experienced hypotension during anesthesia or secondary to their underlying disease process are at risk of developing acute renal failure. Failure to adequately treat shock following trauma, and failure to monitor blood pressure also increase the risk of acute renal failure. Fluid requirements in most critically ill patients exceed maintenance requirements by a factor of 2-4 - that is, these patients need to receive at least 5 ml/kg/hr of fluids such as Hartman’s in order to prevent them from becoming hypotensive, dehydrated and at risk of developing renal failure. Monitoring urine output is essential in critical patients. Urination should be recorded on inpatient charts, and, if necessary, the patient should be catheterized to obtain a more accurate idea of urine production. In most cases, we would aim for a urine output of between 2-4 ml/kg/hr in our critical patients. Lack of urination should prompt immediate action. Management of suspected acute renal failure includes fluid therapy, administration of diuretic therapy, and correction of acid/base and electrolyte disturbances.

Protein Status - many critically ill patients have alterations in capillary permeability and/or decreased levels of albumin, which can cause a loss of fluid out of the vascular space, which contributes to ongoing symptoms of poor tissue perfusion and oxygen delivery. Albumin levels should not persist below a value of 20 g/l, and generally require protein transfusion in the form of fresh frozen plasma. Albumin should preferably be maintained above 23 g/l. Administration of synthetic large molecular weight colloids such as HES is advised when it is anticipated extravasation of serum proteins will occur. This intervention before an increase in vascular permeability occurs can prevent peripheral and pulmonary edema, and retain intravascular
volume. Colloids administered include Pentaspan, HES, dextran 70 (discontinued), plasma and plasma bi-products. Doses of synthetic colloids are 10-20 ml/kg IV q 12-24 hours; for plasma – give to effect, to achieve albumin >23g/l, 10 –20 ml/kg IV q 12 - 24 hours. Note that when giving blood products, premedication with chlorpheniramine is advised to reduce the incidence of nonspecific transfusion reactions.

**General Rules:** in general, in order to provide fluid for ongoing losses, you must be able to quantify them - that is measure the quantity of fluid being lost from the patient in addition to normal everyday losses. The best way to do this is to

1. Measure and record Urine Output every 1-2 hours via urinary catheter and closed collection system
2. Weigh bedding (and record weight gain in bedding) that has been soiled with urine or feces - the change in weight will closely reflect fluid loss from the patient
3. Record the volume of fluid obtained from body cavities such as chest drains
4. Weigh bandaging following changes (and record weight gain in the bandaging)
5. Weigh the patient at least 3 times daily

Much of the weight changes seen in bedding and bandages will be due to the presence of fluid. So, to correct our fluid plan for ongoing losses, we should add together all potential losses from the patient

- Diarrhea, vomitus – 1 gram weight change in bedding = 1 ml of fluid
- Wound exudate – 1 gram change in bandage weigh = 1 ml of fluid
- Pleural or abdominal fluid removal – 1 ml removed = 1 ml of fluid
- Urine losses – 1 ml or urine = 1 ml of fluid

Add these values together to determine the volume of fluid that will need to be added to maintenance fluid rates in order to maintain adequate patient hydration.

**What type of fluid is required to replace ongoing losses?** Well, to answer this question, we need to look at the TYPE of fluid being lost from our patient – and to replace the fluid lost with a fluid of similar composition in terms of electrolytes, protein, and red blood cells. For example, a patient losing hemorrhagic diarrhea should have blood volume restored with a colloid +/- blood (red cells if required), plus a balanced solution of electrolytes - resulting in replacement with a combination of a solution like Lactated Ringer’s solution with potassium chloride, and HES or fresh frozen plasma, +/- packed red blood cells. Likewise, a cat with a pyothorax, having pus drained from the chest, will likely need fluid therapy for ongoing losses that includes a colloid such as HES.
or plasma, to replace the protein lost in the pus drained from the chest. Patients with exudative wounds likewise are likely to require colloid fluid therapy to replace proteins lost through wounds. The procedure for calculating and replacing fluids for ongoing losses is:

- Measure fluid loss from the patient every 4-12 hours
- Calculate the volume of fluid lost from the patient in the previous 4-12 hours
- Replace this volume to the patient over the next 4-12 hours i.e. fluid replacement for ongoing losses may lag behind losses by 4-12 hours
- Replace fluid from ongoing losses with a fluid similar in composition with respect to protein, red blood cells, and electrolytes

To help reduce the “lag” using this method of fluid replacement, many clinicians prefer to begin fluid therapy with supra-normal rates of IV fluids in anticipation of fluid loss e.g. at 2 times maintenance rates. This method also works very well in assisting maintenance of both circulating blood volume and in hydration of the patient.

**Rule No 8: Maintain Fluid Balance**

Fluid balance refers to the maintenance of a balance between fluid loss or elimination from the body, and the rate and quantity of fluid administration to the patient, in order to maintain some kind of balance – so that the patient both remains hydrated and normovolemic, but not overhydrated. Fluid balance also refers to the maintenance of normal electrolyte and acid-base balance as well. Let’s take a look at some numbers:

1. Normal daily fluid requirements for animals are approximately 40-60 ml/kg/day
2. Normal urinary losses from the body are 30-40 ml/kg/day
3. Normal gut losses of fluid are 10-15 ml/kg/day
4. Normal losses of fluid from respiratory tract and body surfaces (called insensible losses) are approximately 5-10 ml/kg/day

These losses are for NORMAL patients that are not on intravenous fluid therapy. Hospitalized patients on intravenous fluids may be expected to have higher fluid losses and fluid requirements, depending on their underlying disease process. So how do we know how much fluid our patients need? There are three easy methods we can use to determine if our patient is receiving sufficient fluid therapy - or whether they are becoming deficient in fluid.
1. **Bodyweight** – acute changes in bodyweight are generally indicative of body fluid loss or gain (weight loss or gain respectively). Bodyweight changes due to nutritional and body mass changes occur at a relatively slow rate (frequently less than 10-50 g/kg/day). Therefore, frequent weight assessment of patients on intravenous fluids may give an indication of relative fluid deficiency or excess (or rehydration early during fluid therapy), and can allow the clinicians to adjust fluid rates to account for fluid deficits indicated by changing bodyweight.

2. **Urine output** – normal urine output in the dog and cat is approximately 25 (cat) – 40 (dog) ml/kg/day – or 1-2 ml/kg/hr. Insertion of a urinary catheter can provide information about excessive urinary losses, or inadequate urine production. Animals on intravenous fluid therapy should generally have a urine output of greater than 2 ml/kg/hr. Urine output of less than 2 ml/kg/hr should alert you to the possibility of:
   a. Urinary catheter blockage or displacement
   b. Inadequate intravenous fluid rate
   c. Development of urinary obstruction or acute renal failure

As we can see, any of these three things can be very serious, and even life-threatening, and therefore should receive immediate attention by the veterinary and nursing team to rectify the problem.

Animals with greater-than-normal urine output of 2-3 ml/kg/hr or above require this urinated volume to be replaced by an equivalent volume of intravenous fluids, in order to prevent the patient becoming dehydrated or hypovolemic. We discuss protocols for weaning of intravenous fluids from animals with high urine output next week – but in general, once a patient is recovering from illness, intravenous fluids should be gradually removed over 12-24 hours (up to 2-3 days for cases of renal insufficiency) by sequential 25% reductions in intravenous fluid rates every 8-12 hours, followed by bodyweight assessments to ensure the patient is not becoming dehydrated.

3. **PCV/TP assessment and physical examination** – these methods are more crude, and a little less accurate than either urine output or bodyweight change, however, the presence of clinical signs of dehydration or a rising TP level may indicate the patient is not receiving sufficient fluid therapy to keep up with losses caused by disease or illness. Likewise, clinical signs of over-hydration such as patient discomfort and serous nasal discharge may indicate over-hydration.
In addition, the monitoring of electrolyte levels will assist us in determining the level of supplementation required in our intravenous fluids in order to maintain fluid balance.

**Rule No 9: Provide Appropriate Electrolyte for Maintenance**

Electrolytes - calcium, potassium, sodium, chloride, and acid base balance should all be measured daily, and corrected where appropriate. Let’s recap.
Decreases in serum potassium cause the following
- Metabolic acidosis
- Muscle weakness
- Gut stasis contributing to intestinal ileus and gastroparesis - vomiting!
- Respiratory muscle paralysis
- Ventricular arrhythmias
- Carbohydrate intolerance, impaired insulin release, weight loss

Hyperkalemia causes the following
- Muscle tremors, followed by muscle weakness
- Bradycardia, ventricular fibrillation, and death

Hyponatremia causes
- Cell swelling and cell death
- Most symptoms seen involve the central nervous system, and include seizures, depression, mild lethargy, nausea, and slight increases in bodyweight

Hypematremia causes
- Cellular dehydration
- Central nervous system depression
- Lethargy, depression, coma, seizures
- Vomiting
- Death
- Tachycardia, dehydration, weak pulses

Nearly all patients on intravenous fluid therapy for longer than 24 hours require the addition of potassium to their intravenous fluids. Patients on intravenous fluid therapy for longer than 24 hours also require the addition of free water to their fluid therapy regime. This is accomplished by using 0.45% NaCl + 2.5% glucose. Therefore, if you see a patient in hospital on IV fluid therapy with Hartman’s for greater than 24 hours - ask the question - “is this the right fluid therapy for this patient?” and raise it with the veterinarian on duty.
Rule No 10: Correct the Underlying Disorder

Obviously without correction of the underlying disorder, our fluid therapy will provide only temporary improvement in patient condition. However, many disease processes will be unable to be adequately managed without fluid therapy and the assistance of provision of adequate oxygen delivery to injured or diseased tissues and organs – making fluid therapy an essential component of the management of many disease processes.

Conclusion

Effective Fluid Therapy involves paying attention to several different patient parameters, or collections of parameters, assessing how to best correct abnormal parameters, and then combining fluid requirements needed to correct each parameter in order to provide the patient with the fluid that will ensure optimum tissue oxygen delivery and organ function. Failure to systematically examine the patient in a manner similar to that described will invariably lead to a decrease in the effectiveness of the fluid therapy, and an increase in patient morbidity and/or mortality.