Nursing for the Post-Operative Cardiac Patient

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While the title of these proceedings can suggest surgical intervention or techniques performed on the actual heart of the patient the intent is the post-operative care for animals with pre-existing cardiac disease. Nonetheless, for those of you that do perform cardiac surgery the following techniques still apply, sourced from the McGill University protocols of critical care management. A holistic approach to the patient and watching trends rather than focusing on specific numbers is still standard. Goal directed monitoring also has its place in intensive care settings, but can lead to missed clinical signs or features.

It is crucial to have trained staff monitor critical patients. It may even be necessary to have one dedicated technician for a particular animal in the ICU setting with little distraction, especially of the patient was a ASA IV or above. Advanced techniques to monitor hemodynamtic parameters can involve placing a central line for central venous pressure measurement or even placing and maintaining an arterial catheter. This sort of monitoring is still a gold standard. As we all know this may be difficult and not practical for the duration of a case or feasible in a certain species. Not to mention financially burdensome to the client or out of the scope of your practice's needs and abilities. This, in turn, can lead to less effective treatment and response in our patients. There is now non-invasive technology which includes Perfusion Index (PI) and Pleth-Variability Index (PVI). These are two newer monitoring parameters that can be very telling when obtained correctly. The perfusion index is the ratio of the pulsatile blood flow to the non-pulsatile or static blood in peripheral tissues. What this means is now we can monitor peripheral tissue perfusion in our patients non-invasively giving better insight into our fluid therapy management, cardiac/renal output, and efficacy of medications. A defined reference variable is not yet established in the canine or feline patient or any other species for that matter, other than humans, which tends to be quite broad. However, the PI parameter, as well as all of the other non-conventional parameters, is great for trending and monitoring. The PI is also a great tool for assessing the efficacy of opioids and epidurals. When full onset of the opioid or epidural occurs, we see a spike in the PI showing via vasodilation.

The Pleth Variability Index (PVI) is a new technology even in human medicine. It is a measurement of the change of perfusion index with a complete respiratory cycle. With this in mind, PVI is most reliable with patients undergoing mechanical ventilation. In a scientific abstract presented at the American College of Veterinary Anesthesiologists conference, one research group found that the PVI had a good correlation in detecting hypovolemia and return to normovolemia in dogs, but could not be used in definitively stating hypervolemia. Several more recent veterinary papers on PVI have come out with positive conclusions as to the reliability of predicting fluid responsiveness using this non-invasive tool.

History- A clear history should be gathered about the patient, from its primary veterinarian, surgeon, anesthetist and any other sources.

- Patient background (age, sex, code status)
- Type of operation and outcome
- Indications for operation and pre-operative diagnosis
- Current inotropes, vasopressors, or anti-hypertensives (if any)
- Need for cardiac pacing when applicable
- Bleeding risks and clotting times
- Other significant co morbidity, with emphasis on those conditions that may alter the post-operative management or course (asthma, diabetes, renal failure, hepatic failure, etc.)
- Pre-operative medications
- Allergies

Physical exam and assessment

- If indeed the animal did endure a cardiac surgery (typical in research or university settings) assure that the endotracheal tube is in proper position and the patient has equal air entry bilaterally. Remember that tube displacement or pneumothoraxes' can occur or become apparent at any moment.
- Verify that the patient's oxygen saturation is adequate. Check the ABG results as soon as they are available. If this is not available an SpO2 and monitoring of the pH, bicarbonate and electrolytes must be evaluated.
- Verify correct ventilator settings.
- Check the initial hemodynamic readings (HR, BP, cardiac output and index, CVP) and determine what vasoactive infusions the patient is on and at what rates.
- Check the patient's heart rhythm. Verify pacemaker settings if the patient is connected to one.
- Examine heart sounds. Listen for murmurs.
- Check all peripheral pulses. Do repeated assessments if there is concern for acute limb ischemia. A Doppler can be placed for on a peripheral limb for continuous evaluation.
- Do a more complete neurologic exam when the patient begins to awaken from GA.

Labs and tests Electrocardiogram

- Note any changes from pre-op ECG
- Rhythm - post-operative bradycardias, blocks, or atrial fibrillation
- ST-T changes - diffuse non-specific changes are not uncommon and may reflect pericardial inflammation or ischemic events
- Chest X-Ray
- Rarely used in the non-research sector, verify correct position of the Swan-Ganz catheter.

Laboratory Results

- Hemoglobin
- Coagulation parameters (PLT, PT, PTT, ACT)
- Potassium, magnesium, calcium - a vigorous diuresis is common in the first few hours after the OR. This can lead to significant hypokalemia and hypomagnesaemia which increases the likelihood of post-operative dysrhythmias. Standing orders are in place to replace these electrolytes.
- Glucose - tight glycemic control post-operatively reduces morbidity in humans.
- Cardiac markers - elevations of CPK, CPK-MB, and troponins are non-specific. They should be assessed as part of the overall clinical picture including the hemodynamic status of the patient and the EKG.

Warming

Effects of hypothermia

- Predisposes to ventricular dysrhythmias and lowers VF threshold
- Increases SVR; increases afterload and myocardial workload
- Patient shivering causes increased peripheral O2 consumption
- Decreases CO2 production; a patient who has a respiratory alkalosis (low PCO2) on initial ABG usually will increase their PCO2 with rewarming
- Coagulopathy; impairs platelet function and the coagulation cascade. Rewarming is an important part of the treatment of a bleeding patient.

Transfusion

The principle objective when giving PRBC's is the improvement of inadequate oxygen delivery and the minimization of adverse outcomes as a result of this. In a patient who is actively bleeding and thus who's hemoglobin mass is not in a steady state, one must be more liberal in transfusing PRBC's to avoid severe impairments in peripheral oxygen delivery. However, with a patient who is not bleeding rapidly, one can take a more deliberate approach to transfusion. Transfusions are often over prescribed. If the patient appears to

have a normal neurologic status and respiratory pattern/rate, a transfusion may not be warranted. There are several potential risks associated with the transfusion of red blood cells.

Hemodynamic management

Hypotension and low cardiac output

1. *BP = CO x SVR*
2. *CO = HR x SV (stroke volume)*
3. *Stroke volume is determined by preload, contractility, and afterload*
4. *Bradycardias or tachydysrhythmias that decrease ventricular filling can decrease CO.*

There are numerous causes for hypotension post-operatively. Proper management of the hypotensive patient in the ICU requires that the precise etiology for the hypotension is determined and therapy is directed towards reversal of this specific problem. Equation 1 demonstrates that hypotension can be caused by a "pump problem" (low cardiac output) or a low SVR (arterial "circuit" problem). The following is an approach to managing the hypotensive patient:

1. Look at the recent hemodynamic parameters.
2. Assess the cardiac output/index. Is this a "pump" problem? Or is it due to low SVR?
3. Look at the cardiac rhythm.
4. Look at the CVP to assess preload.
5. Is the afterload high?
6. Is contractility decreased?

- Is this tamponade? Look at the recent hemodynamic parameters obtained from the Swan-Ganz catheter or evaluate via echo.
- Assess the cardiac output/index.
- If the cardiac index is in the normal range or high, then the patient does not have a significant "pump" problem and the cause of the hypotension is secondary to diminished peripheral arterial tone (low SVR). A vasopressor agent should be considered. The differential diagnosis of low SVR includes:
    - SIRS - a proportion of patients post CPB will have significant cytokine increases
    - Sepsis
    - Anaphylactic or anaphylactoid reactions
    - Drug-induced, toxicological - nitrates, antihypertensives, narcotics and sedatives, etc
    - Adrenal insufficiency (Was the patient steroid dependent pre-operatively?)
    - Hyperthyroidism, hypothyroidism
    - Neurogenic (spinal) shock
- If the cardiac index (CI) is low then the cause of the hypotension is inadequate flow or a "pump" problem.
- Look at the cardiac rhythm. Absolute or relative bradycardias or tachycardias can lead to decreased CO and should be corrected.
- Look at the CVP to assess preload. A patient with a low CI and a CVP that is "relatively" low should be given a fluid challenge. Remember, what you really are interested in is a volume measurement (preload= right or left end-diastolic volume), but what you are measuring are pressures (CVP = Right or left ventricular end-diastolic pressures).
- High afterload. Secondary to vasoconstriction and hypertension.
- Decreased contractility. This should be managed with inotropic agents while simultaneously looking for the cause.
- Tamponade
- Acute valvular regurgitation. Check for a new regurgitant murmur.

Inotropes and vasopressors

Inotropes

1. Adrenergic (catecholamine)

- Dobutamine - beta-agonist (ß1 >ß2). Increases contractility and HR. ß2 effect can sometimes decrease SVR and BP. ß1 effect can cause dysrhythmias. Start at 2.5 mcg/kg/min. Titrate upward by 2.5 mcg/kg/min until adequate cardiac index.
- Epinephrine -alpha and beta agonist (ß > alpha). Increases HR, CO, and SVR. Generally a second-line inotrope. A subset of patients who do not respond to dobutamine will respond to epinephrine. Potential detrimental effects include significant increases in myocardial oxygen consumption, increased lactic acidosis, arrhythmias. Start at 0.5 to 1.0 mcg/min and increase by these amounts until adequate cardiac index.
- Dopamine - stimulates dopaminergic, beta, and alpha receptors in dose-dependent fashion. May be less effective in cats. Inotropic effect (beta-effect) predominates in the 5 to 10 mcg/kg/min range. In humans, low doses (2 - 4 mcg/kg/min) it has been purported to have beneficial renal protective effects ("renal-dose dopamine"). While it can increase urine output by several mechanisms, there is little evidence that it improves creatinine clearance or decreases the incidence of acute renal failure.

Vasopressors

1. *Adrenergic (catecholamine)*
    - Norepinephrine -Strong alpha agonist with beta activity as well. Causes vasoconstriction and thus increases SVR and BP. Theoretically, since it has inotropic activity as well, it is less likely to cause a decrease in cardiac output due to increased afterload compared to a pure alpha agonist such as phenylephrine. Negative effects include myocardial and mesenteric ischemia, dysrhythmias, and decreased cardiac output due to afterload increases.
    - Phenylephrine (Neosynephrine) - Pure alpha agonist. Can be used as a continuous infusion but more commonly used as bolus for sudden severe hypotension not responding to volume infusion.
2. *Peptides*
    - Vasopressin - used for hypotension with a normal or high cardiac output and low SVR state that is refractory to norepinephrine. Has a significant side effect profile including myocardial and mesenteric ischemia.