CPB FMEA #48 Failure to prevent osmotic demyelination syndrome during CPB in patients with chronic severe hyponatremia

Friends-

This FMEA is inspired by Susan Canaday’s article on the risks of osmotic demyelination syndrome (ODS) during CPB (Canaday S, Rompala J, Rowles J, Fisher J, Holt D, Chronic Severe Hyponatremia and Cardiopulmonary Bypass: Avoiding Osmotic Demyelination Syndrome, JECT. 2015;47:228–230). I asked Susan to comment on this narrative and FMEA. Her comments are indicated in quotes below.

ODS can occur during CPB in patients with chronic severe hyponatremia.  A demyelinating disease causes damage to the myelin sheath of neurons in the central nervous system. This damage impairs the conduction of signals in the affected nerves. If the damage is due to rapid iatrogenic correction of hyponatremia the disease is called central pontine myelinolysis (CPM).

CPM was initially described by Adams in alcoholics and malnourished patients in 1958 (Adams RD, Victor M, Mancall EL. Central pontine myelinosis: a hitherto undescribed disease occurring in alcoholic and malnourished patients. Arch Neurol Psychiatry 1959;81: 154-6.) Back then the disease was not associated with cardiac surgery patients.  So I wonder if some of the patients I saw back in the 60’s with unexplained brain damage after CPB could have had this problem.

The normal serum sodium is 136-145 mmol/L.  Mild hyponatremia is 130-135, moderate is 125-129, severe is 116-124 and lethal is <116. If chronic hyponatremia is present in any patient it is important to correct this slowly, at a rate of less than 8 mmol/l/day or 0.3 mmol/l/hr to minimize the risk of developing ODS.  With such a long time span for correction, it is usually not within the perfusionist’s power to safely correct the situation during CPB.  Indeed, there is a significant danger that the influx of crystalloid prime with a normal sodium and osmolarity from the pump could precipitate demyelination. The additions of sodium bicarbonate and/or mannitol to the prime could make the risk even worse.

Susan Canaday and colleagues were confronted with an emergent CPB case in a patient with severe chronic hyponatremia of 122 mmol/L serum sodium. By remixing they were able to reduce their normal prime from 153 mmol/L sodium to 120 mmol/L. (S. Canaday: “I had to reread the article to notice that our pump prime as tested by the lab was 145 even though we calculated it to be 153mmol/L. I am not even sure this matters except that as you say later, calculating the [Na+] and testing it often yield different results.”) This avoided the major trigger for ODS in this patient and greatly reduced the risk. As Susan states in her article, “In the case of chronic severe hyponatremia and the emergent open-heart procedure, it is essential to note the catastrophic consequence of initiating CPB with a prime of normal sodium concentration.” The article and this FMEA can both be teaching and learning tools to help mitigate the risk of this rare but devastating condition.

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CPB FMEA #47 Failure to prevent Osmotic Demyelination Syndrome during CPB .

FAILURE:  Failure to prevent Osmotic Demyelination Syndrome (ODS) during CPB in patients with chronic severe hyponatremia

EFFECT:

1. The effect is severe damage of the myelin sheath of nerve cells resulting in paralysis, irreversible brain damage, or death.   If the damage occurs in the central pons it is called central pontine myelinolysis (CPM) which is the iatrogenic form of ODS.

2. ODS and CPM are associated with many different CNS dysfunctions;  dysarthria, dysphagia, dystonia, mutism, ataxia, parkinsonism, quadraparesis, seizures, lethargy, coma and death.

CAUSE:

1. A demyelinating disease causes damage to the myelin sheath of neurons and impairs the conduction of signals in the affected nerves.

2. CPM is a severe damage of the myelin sheath of nerve cells in the area of the pons brain stem which is primarily iatrogenic due to the incorrect treatment of hyponatremia.

3.Chronic hyponatremia allows extracellular water into the cerebrospinal fluid with migration of electrolytes and other osmolytes from brain cells. This compensation mechanism prevents shrinkage of the central nervous system (CNS) including the brain and minimizes CNS edema.

4.The normal serum sodium is 136-145 mmol/L.  Mild hyponatremia is 130-135, moderate is 125-129, severe is 116-124 and lethal is <116. Normal osmolarity varies from 270-300 mosmoles/L depending on patient age.

5.ODS and CPM develop in the setting of chronic hyponatremia with the rapid correction of serum sodium concentration without sufficient time for osmolytes to re-accumulate within the brain cells which may cause demyelination.

7.Chronic hyponatremia is multifactorial and is most often associated with alcoholics, the aged and infants; <<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1246086/>>.

8.Chronic malnutrition, prolonged diuretic use, liver failure, organ transplant, and extensive burns are also associated with ODS and CPM.

9.The influx of a CPB pump prime containing normal sodium and osmolarity levels may be enough to trigger demyelination in a patient with chronic hyponatremia.  The addition of sodium bicarbonate and/or mannitol to the prime increases the osmolarity and magnifies the danger to the patient.

10. Smaller patients’ sodium concentration will be affected more by prime volume as they have a smaller plasma volume.

11.Patients with a high preoperative hematocrit will be affected more because they have a lower plasma volume percentage.

12. Over tIme the impact of even minor changes to the pump prime components can result in major electrolyte and osmolarity changes that go unnoticed by the perfusionist. (Darling E, Harrimeis-Holloway S, Kern FH, Ungerleider R, Jaggers J, Lawson S, Shearer I. The impact of modifying priming components and fluid administration using miniaturized circuitry in neonatal cardiopulmonary bypass. Perfusion. 2000 Jan;15(1):3-12.)

 PRE-EMPTIVE:

1. The pump prime should be routinely tested periodically as a quality control measure to confirm the electrolyte and osmolarity composition and as a baseline for stoichiometry modification if needed *(S. Canaday: “The only other professional to contact me about the article asked if it was CLIA approved to test a non-blood product via the lab.  I asked our lab and they said it was not. However our director agreed w/ me that it was imperative to patient safety and was fine by her if we continued this practice.  We were also able to add it to our ABG parameters by buying a different cartridge, I believe, as part of the testing – we have a GEM 3000 that anesthesia techs use to help us calibrate our CDI.  Also not sure this matters because safety and our unusual circumstance w/ hitting the patient w/ 1+L within seconds I would hope could overrule CLIA regulations and allow us this as a secondary check to our calculation  – just wanted to mention it in case you found it interesting.”)*.

2. A procedure should be developed and written ahead of time by the perfusionists and pharmacy to compound a priming solution that closely matches the sodium and osmolarity of the chronically hyponatremic patient. *(S. Canaday: “This is an awesome point and we have done exactly this anytime we get another chronic severely or moderately severe hyponatremic patient.  If we take a newly calculated prime w/ Na+ concentration of 127 for example, we write these exact components [amount Plasmalyte, Albumin, heparin, TXA/Amicar, etc.] and the actual Na+ concentration per our lab on a running sheet of paper in our protocol notebook.  This allows us to re-create this prime again without remembering or calculating.  Then in an emergent situation all we have to do is hand a sample of prime to anesthesia when they draw an ABG post intubation/pre-CPB and we get a Na+ concentration check of that prime.”)*

3. In the absence of such a pre-planned procedure, stoichiometry should be used to calculate the correct component amounts to achieve the desired sodium content. *(S. Canaday: “Also of note, in a “crash” on pump situation, you can always use 0.45 NS [Na+ concentration of 77mmol/L] and add 43 mEq or 43mL of sodium bicarbonate and have 1L bags of crystalloid that has a Na+ concentration of 120mmol/L.  Obviously, you can create any Na+ concentration by adding the appropriate amount of 1mEq/mL of sodium bicarbonate to these bags.  I then use a label across the bag w/ huge red marker saying [Na+] = xx and if you need quick prime later you can also drop quickly on pump, it is all ready and isotonic to patient.  This is a trick I’ve never had to use, but I keep in my back pocket just in case such a situation arises and I don’t have time to calculate or check via lab the prime’s Na+ concentration with the many components//Albumin, etc., added.  The colloidal pressure, etc., would be unhelpful, but you could slowly add Albumin etc after on pump and prevent an unknown Na+ concentration prime from hitting the patient all at once.”).*

4.  Autologous priming should be considered to reduce the impact of prime sodium levels on the inherent sodium of the patient.

5. Sodium and osmotic testing should be performed to compare the patient and prime values if time allows.

6. Mannitol should be used sparingly in the prime or during CPB, if at all, due to its hyperosmotic property.

7. Sodium bicarbonate should be used cautiously during CPB to correct acidosis or hyperkalemia.

8.Hyperglycemia can correlate to hyponatremia, so glucose monitoring before and during CPB should be considered.  *(S. Canaday: “Insulin can also help prevent hyperkalemia.”)*

9. If sodium correction is attempted during CPB, the serum sodium concentration should be raised no more than 0.3 mmol/L every hour.

RISK PRIORITY NUMBER (RPN):

A. Severity (Harmfulness) Rating Scale: how detrimental can the failure be:

1) Slight, 2) Low, 3) Moderate, 4) High, 5) Critical (I would give this Harmfulness failure an RPN of 2 if hyponatremia is mild, a 3 if moderate, a 4 if severe and a 5 if the level is lethal.)

B. Occurrence Rating Scale: how frequently does the failure occur:

1) Remote, 2) Low, 3) Moderate, 4) Frequent, 5) Very High. (This failure occurs very infrequently. So the Occurrence is Remote. The RPN would be a 1.)

C. Detection Rating Scale: how easily the potential failure can be detected before it occurs:

1) Very High, 2) High, 3) Moderate, 4) Low, 5) Uncertain. (The Detectability RPN equals 1 if either a serum sodium or whole blood test is performed on the patient prior to priming and CPB. Although highly unusual, if blood sodium testing is not performed or the results overlooked prior to CPB, the RPN would be a 5.)

D. Patient Frequency Scale: 1) Only a small number of patients would be susceptible to this failure, 2) Many patients but not all would be susceptible to this failure, 3) All patients would be susceptible to this failure. (Only a few patients are at risk. So the Frequency RPN would be 1.)

Multiply A\*B\*C\*D = RPN. The higher the RPN the more dangerous the Failure Mode.

The lowest risk for any failure would be 1\*1\*1\*1\* = 1 and the highest risk would be 5\*5\*5\*3 = 375. RPNs allow the perfusionist to prioritize the risk. Resources should be used to reduce the RPNs of higher risk failures first, if possible. (The total RPN for this failure is very low if the pre-CPB chronic sodium level is mildly hyponatremic, the perfusion procedure includes a plan to remix the crystalloid pump prime to be compatible with the patient’s sodium and osmolarity level and the hyponatremia is monitored pre-CPB: 2\*1\*1\*1 = 2. On the other hand, if the chronic hyponatremia is severe, there is no plan to modify the crystalloid prime and the sodium is not monitored pre-CPB, the RPN would be 5\*1\*5\*1 = 25.)