CPB FMEA # 40 Calcium Regulation

Friends-

On May 25, 2005 a pediatric patient received a lethal overdose of calcium that was probably administered by a perfusionist. The patient died two days later. To make a long story short, this incident prompted the first and (so far) only national, objective and authoritative examination of the perfusion profession. The Gritten Report was released in October of 2007. This report caused a major and immediate shakeup of perfusion practice in Great Briton. Perfusionists in the USA were slow to recognize the importance of the Gritten Report. I never saw it on an AmSECT meeting program and, except for me, never heard it even casually discussed by others at any meetings I attended; national, state or local. In 2014 AmSECT began to focus seriously on perfusion safety as defined by the Gritten Report. Only within the last few years have individual perfusion programs begun to take a closer look at the safety of their practice. These FMEAs I am posting are an attempt to help programs organize a valid safety program that will meet or exceed the standards of any other paramedical occupation.

This CPB FMEA discusses the clinical use of calcium during CPB. There are many variations on how and when calcium is monitored and administered during CPB. This FMEA does not pretend to be the best method, it is just the best method that I know of. The regulation of calcium during CPB is very complicated with the potential to injure or even kill a patient, as described in the Gritten Report. All too often, calcium regulation is too cavalier. When this FMEA has a table top scenario discussion at your program I hope that it sparks a critical examination of your current practice.

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CPB FMEA # 40 Calcium Regulation

FAILURE: Failure to regulate blood ionized calcium (iCa+2) as necessary.

EFFECT:

1. Hemodynamic instability

2. Cardiac distention.

3. Risk of overdose.

4. Brain damage.

5. Death.

CAUSE: CAUSE:

1. Serum and extracellular iCa+2 can be a major mediator of reperfusion injury.

2. The extracellular concentration of iCa+2 is 10,000 times higher than intracellular concentration.

3. During a period of ischemia, such as occurs when the heart is cross clamped or the patient undergoes deep hypothermic circulatory arrest, the cell membrane may be weakened allowing excess calcium to leak into the acidotic cardiomyocyte or neuron from the extracellular fluid.

4. Excessive calcium within the heart or brain cells can damage the mitochondria by forming a mitochondrial permeability transition pore (MPTP).

5. This irreversible damage to the mitochondria is a major component of the phenomenon known as reperfusion injury.

6. The cells most susceptible to this type of damage are 'excitable' cells such as neurons and cardiac myocytes.

7. Excitable cells can generate an action potential at their membrane in response to depolarization and may transmit an impulse along the membrane.

8. Calcium is vital to promote the contractility of the heart. So, any reduction in the blood iCa+2 can result in the distention of the heart muscle, possibly causing irreparable damage.

9. On the other hand, since calcium is a major mediator of reperfusion injury, even normal calcium blood levels can cause damage in suddenly reperfused ischemic tissues.

10. To this end, when the heart is exposed to ischemia as occurs during aortic cross clamping, the blood iCa+2 is often kept intentionally low to mitigate reperfusion injury upon reperfusion of the coronary arteries.

PRE-EMPTIVE MANAGEMENT:

1. Safe Practice Recommendation: The Institute for Safe Medication Practices recommends the use of either calcium gluconate (CaGluc) or calcium chloride (CaCl2) in an institution, but not both. Hospitals often store both CaGluc and CaCl2. <https://www.ismp.org/newsletters/acutecare/articles/19970507.asp>

2. There is a three-fold difference in the primary cation between the two drugs.

3. An ampule of 10 ml/10% CaGluc contains 8.9 mg/mL (4.65 mEq/gm) of elemental calcium.

4. An ampule of 10ml/10% CaCl2 contains 27.2 mg/mL (13.6 mEq/gm) of elemental calcium.

5. CaCl2 is more caustic and may cause intravascular tissue damage or tissue necrosis with extravasation.

6. CaGluc must be metabolized in the liver before it becomes bioavailable.

7. Calcium can temporarily counter act the myocardial effects of high potassium by restoring cardiomyocyte resting membrane potential, but it does not lower the serum potassium level.

INTERACTIONS:

1. If serum phosphate is elevated during calcium administration, precipitation of calcium phosphate may occur in the vasculature with potential end organ injury such as interstitial pneumonitis.

2. When serum phosphorus is low, larger quantities of calcium may be needed for replacement.

3. Rapid injection of calcium may cause bradyarrhythmias, especially in patients on digoxin.

4. Calcium may antagonize calcium channel blockers causing increased systemic vascular resistance.

MANAGEMENT:

1. A low blood calcium strategy is used by some surgeons anytime ischemia is to be intentionally induced with certain exceptions (see EXCEPTIONS below).Such induced ischemia includes aortic cross clamping with cardioplegia or total body deep hypothermic circulatory arrest (Chen 1996).

2. If a pump is primed with calcium free crystalloid solution or if a blood prime is used, there is no re-calcification. This will reduce the iCa+2 in children and many adults below the normal level (1.1 – 1.4 mmoles/L) after CBP is initiated.

3. If low calcium strategy is used or if the iCa+2 is already low upon the initiation of CPB, drain the heart completely to prevent the risk of over distention.

4. After cross clamp removal or reperfusion after circulatory arrest, the blood can be re-calcified to normalize the iCa+2 using slow, non-bolus, injection into the venous reservoir.

EXCEPTIONS

1. If no induced ischemia is expected, the pump prime calcium levels can be normalized prior to initiating CPB. CaGluc should be added to the prime prior to the initiation of CPB, but only after heparin is added to the prime and re-circulated. CaCl2 may precipitate any bicarbonate that is in the prime solution.

2. Since a low blood iCa+2 can impair the heart's contractility, patients with aortic insufficiency are at risk of ventricular distention and damage when CPB is initiated. This can be particularly dangerous when initiating CPB peripherally (fem-fem or neck), which prevents the surgeon from installing a left ventricular vent in a timely fashion. In these cases, re-calcification of the pump prime takes precedent over low calcium strategy. CaGluc should be added to the prime prior to the initiation of CPB.

a. Maintain left ventricular ejection after the initiation of CPB. If ejection stops (no pulse pressure wave on the arterial pressure monitoring line), the heart may be distending.

b. Should distention occur, the pump flow should be lowered to an arterial pressure of no more than 20 mmHg, followed by immediate calcium supplementation and assessment of contractility.

c. Should ventricular fibrillation occur, a similar strategy as above should be employed until defibrillation can be performed.

\*The Detectability RPN of 2 is based on the premise that point of care testing for iCa+2 is immediately available in the OR. If not, the Detectability RPN should be increased to 4, resulting in a total RPN of 5\*2\*4\*3 = 120.

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 failure a Critical RPN, 5.)

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

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

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 2 on the premise that point of care testing is immediately available in the OR. If not, then the Detectability RPN would be 4.)

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. (All patients would be at risk. So the Frequency RPN would be 3.)

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

The lowest risk would be 1\*1\*1\*1\* = 1. 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 = 5\*2\*2\*3 = 60. If there is no point of care testing for iCa+2 in the OR, the total RPN would be 5\*2\*4\*3 = 120.)