CPB FMEA #38 Bicarbonate imbalance

Friends-

This week's FMEA for review deals primarily with the administration of sodium bicarbonate (NaHCO3). NaHCO3 is probably the drug most often given during

CPB by perfusionists. But I think this is a failure that hurts patients more than most perfusionists realize. Giving a lot of NaHCO3 for a variety of reasons just to restore the bicarbonate level and keep the pH in the normal range during CPB can increase the osmolarity to a very high level. This is particularly true when mannitol is also used in the prime or given during CPB. It is easy to estimate the blood osmolarity changes caused by NaHCO3 from POC testing of whole blood electrolytes. But if a lot of mannitol is given, its effect cannot be estimated and the blood must be sent to the lab for osmolarity measurement.

To paraphrase two of the volunteer FMEA reviewers, they are not a big fans of NaHCO3, mainly because it is over used, administered too rapidly and seems to have fallen into a “catch all category” as a panacea for poor perfusion. They further describe students who go to various externship sites and sometimes receive instructions to maintain fastidiously low flows (CI <2.2) even during rewarming with the expectation that NaHCO3 will routinely be given before weaning from CPB.

IMHO, the best solution is not to routinely to give NaHCO3 during bypass. My solution to correct acidosis during CPB relied on increasing the blood flow if practical, increasing FiO2 and changing CO2 level or changing CO2 strategy (alpha stat to pH stat or vice versa) if hypothermia is being used. These three actions seemed to improve oxygenation at the cellular level and stopped or reversed the development of a base deficit for me. I only gave enough NaHCO3 during CPB to compensate for any bicarbonate free crystalloid irrigation sucked into the pump. In kids, I prepared a bicarbonate balanced (normal Na) prime for both CPB and ECMO.

Previous American Heart Association advanced cardiac life support (ACLS) guidelines recommended routine bicarbonate administration as part of the ACLS algorithm. But recent guidelines no longer recommend its use because there is little evidence that it is beneficial in a cardiac arrest situation. In fact, NaHCO3 is thought to contribute to a variety of problems: 1) NaHCO3 may reduce systemic vascular resistance. 2) It causes extracellular alkalosis that unfavorably shifts the oxyhemoglobin saturation curve. 3) It causes hypernatremia and hyperosmolarity. 4) It produces excess CO2, which diffuses into myocardial and cerebral cells and paradoxically contributes to intracellular acidosis. 5) It can exacerbate central venous acidosis and may inactivate simultaneously administered catecholamines. I don’t know the cumulative effects these problems may have during CPB, but caution is the better part of valor when alternatives are available.

If you have any additional thoughts on this subject please share them on Perflist or contact me directly.

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CPB FMEA #38 Bicarbonate imbalance

FAILURE

Low bicarbonate (HCO3) level during CPB.

EFFECT:

1. Acidosis.

2. Disrupted metabolism.

3. Potential for hypernatremia w/ renal or brain damage if treated w/ sodium bicarbonate (NaHCO3).

CAUSE:

HCO3is a chemical buffer that helps to keep the pH of blood from becoming too acidic or too basic as long as CO2 is adequately ventilated from the blood. The normal HCO3 level is 25 ± 4 mEq/L. Low levels of HCO3 may indicate acidosis. High levels of HCO3 may indicate a respiratory compensated acidosis.

1. The development of true metabolic acidosis during CPB is relatively rare. Inadequate CPB oxygenation may result in metabolic acidosis and HCO3 consumption.
2. If the HCO3level is iatrogenically reduced, an acidosis may develop which is not the result of metabolic production of acid. This may occur on CPB in two ways:

a. The infusion of HCO3-free crystalloid or the entrainment of HCO3-free crystalloid irrigation into the pump will dilute the circulating HCO3 level. This will induce a dilutional acidosis which is not related to the adequacy of perfusion. Infants and small children are particularly susceptible to this phenomenon. Some crystalloids contain gluconate, acetate or lactate that are converted to HCO3 in about 6 minutes upon passing through the liver in adults. In children, the efficiency of this conversion is much slower (in the 6 hour range in infants). Consequently, waiting for this conversion is not practical in children.

b. Blood bank red blood cells (RBCs), even when washed through autotransfusion equipment, carry a heavy lactic acid load. When RBCs are infused into the CPB circuit, this acid will consume some HCO3 and cause an acidosis to develop.

3. Excess NaHCO3 administration during CPB may result in hyperosmolarity (> 300 mOsmols/L) which may cause renal (>320) and brain (>360) damage.

PRE-EMPTIVE MANAGEMENT:

1. A balanced crystalloid containing acetate, gluconate or lactate can be used in adult primes and as supplemental fluids as these will convert to HCO3 within six minutes of CPB.

2. A crystalloid prime with 25 mEq/L of HCO3 will prevent dilutional acidosis, particularly in children. Pediatric prime should have a Na <145 mEq/L and an osmolarity <320 mOsmols/L.

3. In children, adding 25 mEq/L of NaHCO3 to supplemental crystalloid fluid prior to its infusion into the circuit during CPB will prevent the dilution of HCO3.

4. Osmolarity (in the absence of mannitol) can be estimated from POC testing with this formula: (Na mEq/L X 2) + (glucose mg% /18 ) + 15 = calculated osmolarity.

MANAGEMENT:

1. Maintaining fastidiously low flow (CI <2.2) with acidosis development should trigger a blood flow increase rather than NaHCO3 administration.
2. For metabolic acidosis, administer 1 mEq/L (combined pump and patient circulation volume) for each -1 mEq/L base deficit. Repeat as needed.
3. Alternate formula: ( Desired HCO3 - Actual HCO3) x KG x 0.3 = NaHCO3
4. Each unit of banked RBC will require 5-10 mEq of NaHCO3 to neutralize the effect of the acid load in the RBCs.
5. NaHCO3 should not be given until the base deficit is -4 or greater.
6. The entrainment of excessive irrigation into the CPB circuit will cause a dilutional acidosis requiring the administration of NaHCO3. The excessive irrigation will need to be removed by diuresis or ultrafiltration (UF). The amount of ultrafiltrate from irrigation can be estimated by the amount of NaHCO3 administered. For example, assuming a normal HCO3 level of 25 mEq/L, 400 mls of irrigation ultrafiltrate would require the administration of 10 mEq of NaHCO3 to maintain a normal HCO3 level and pH. On the other hand, the removal of fluid by UF which does not require the need for NaHCO3 supplementation to maintain normal HCO3 levels indicates that the fluid was removed from the patient's own extracellular compartment.
7. Care should be taken to prevent the Na from increasing beyond 145 mEq/L due to NaHCO3 dosing. Small amounts of 0.45% NS w/ 50 mEq/L of NaHCO3 added (127 mEq/L Na w/ 254 mOsmols/L) may be administered to prevent hypernatremia followed by diuresis or UF to remove excess fluid volume.
8. NaHCO3 administered too rapidly can form CO2 gas emboli and trigger a bubble alarm.
9. NaHCO3 given concurrently with Ca chloride or Ca gluconate may form Ca carbonate (chalk) and lower K+.
10. If Na is elevated too quickly with NaHCO3, central poutine myelinolysis (aka osmotic demyelination syndrome) or other brain damage can occur, particularly in infants and patients with severe hyponatremia. This might be confused with post pump chorea.
11. NaHCO3 given immediately prior to weaning may result in systemic vasodilation, decreased cerebral blood flow and decreased cardiac function resulting in lower MAP, lower NIRS values and low cardiac output after weaning.
12. Consider THAM (aka tris or tromethamine, 3.6 gm/100 mL = 30 mEq or 0.3 mol/L w/ 389 mOsmol/L.) for patients with elevated Na levels, chronic acidosis or patients with severe hyponatremia .
13. THAM acetate dosage: ml of 0.3 mol/L = KG x Base Deficit (mEq/L) x 1.1
14. Max THAM dose = 500mg/KG.

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 Low RPN, 2.)

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

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

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.)

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 for this failure so the Patient 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 = 2\*3\*2\*3 = 36.)