CPB FMEA #33 Failure to recognize the presence of major aortic to pulmonary collateral arteries (MAPCAs).

Friends-

The history of dealing with MAPCAs (Major Aortic to Pulmonary Collateral Arteries) on CPB goes back to the very beginnings of open heart surgery. It has been a long and difficult learning curve, especially for the patients. It is one of those unfortunate circumstances where we learned by trial-and-error over many years, during which time the patients (or their parents) had complete, but unjustified confidence in the surgeon and cardiac team. Unjustified because surgeons and perfusionists did not know what they did not know. Nowadays, experienced pediatric perfusionists are well versed in using CPB on a MAPCA patient; adult perfusionists not so much. But as congenital patients grow older they will require additional surgery for revision or for acquired disease. The adult perfusionist faced with the MAPCA patient may be unaware of the dreadful ramifications that can ensue. The CPB may appear to work well during these cases, while in reality a devastating outcome may develop. The patient’s bad outcome might be incorrectly attributed to poor patient selection or high risk circumstances. But in reality the perfusion methods used (or not used) may contribute substantially to post-operative morbidity without the perfusionist even being aware. Not all perfusionists are qualified by training or experience to pump MAPCA cases, but they may be pressed into it by circumstance. Maybe this FMEA will provide the institutional memory and guidance for those perfusionists needing it for these rare procedures.

The term “Major… Arteries” is deceiving. It implies that these are large vessels between the aorta and lungs. True, that is often the case. And sometimes these vessels can be mapped at cath. Then severed from the aorta, gathered together by the surgeon and affixed to whatever diminutive main pulmonary artery is present; a so-called unifocalization. But “Major” can also refer to the volume of blood these vessels carry rather than their anatomical size. Often the vessels are very small and individually indistinct, but very numerous. When the aorta is injected with dye at cath, the presence of these collaterals is seen as an indistinct dye cloud moving from the aorta to the lungs. I think it looks like a puff of smoke. Just because the vessels aren’t large and well defined does not mean that the collateral blood flow they are carrying is small and inconsequential. There can also be a mix of large and small vessels. So even if a unifocalization of the large vessels is successful, there may still be a significant collateral flow to the lungs from the smaller vessels not incorporated in the unifocalization. Further adding to the problem is the fact that the collaterals could be composed of either systemic or pulmonary tissue in different patients. This means that they vasoconstrict under different physiologic conditions, further adding to the perfusionist’s difficulty in controlling the collateral blood flow.

I discourage the use of bloodless CPB techniques that tolerate a lower than normal hematocrit in MAPCA patients. I know that there are those who would rigorously disagree with me. It is just my educated opinion that, in these patients, the risk of brain damage due to under perfusion is substantially higher than the ill-defined risk of an uncomplicated blood transfusion. I would asses an uncomplicated blood transfusion on CPB with a Harmfulness RPN of 3/5. But I would asses a Harmfulness score of 5/5 for MAPCA-related under perfusion problems. School age children with MAPCAs who have undergone heart surgery earlier in their childhood have substantially more learning problems than normal children. I have no idea how much of this can be attributed to either brain damage from MAPCA-related under perfusion during CPB or to any uncomplicated blood transfusion they may have received. In adults, MAPCA-related under perfusion during CPB could conceivably accelerate other age-related health problems like chronic kidney failure, dementia or even stroke if the patient survives the surgery. I have no specific proof of this, just a suspicion (Murphy et al. Optimal Perfusion During Cardiopulmonary Bypass: An Evidence-Based Approach Anesth Analg 2009;108:1394 –417). Nonetheless, if your program chooses to go bloodless in a MAPCA patient just modify this FMEA and the RPN to reflect your practice.

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FAILURE: Failure to recognize the presence of major aortic to pulmonary collateral arteries (MAPCAs).

EFFECT:

Many congenital heart patients have collateral blood vessels that can steal perfusion from major organ systems causing:

1. hemodynamic instability

2. acidosis

3. reperfusion injury

4. organ damage

5. death

CAUSE:

MAPCAs are often present in patients with congenital hypoxic cardiac lesions and develop spontaneously to provide additional blood flow to the lungs. This results in a significant systemic to pulmonary shunt that can reduce effective systemic blood flow during CPB. MAPCAs can be anticipated in any hypoxic lesion patient, but are most often present in patients with pulmonary artery atresia, hypoplastic right and left hearts, univentricular anatomy or Tetralogy of Fallot with pulmonary atresia. MAPCAs may or may not be detected by cardiac catheterization. They may be present as large, readily identifiable vessels or as a large plexus of small, individually unidentifiable vessels or a mix of the two.

Patients at increased risk of MAPCAs:

1. Adults with congenital heart disease are at elevated risk of having MAPCAs even if the lesion was palliated or repaired in childhood.

2. Fontan completion patients, particularly those older than two years and those who have failed a previous Fontan completion attempt.

3. Patients with limited pulmonary blood flow but whose room air arterial hemoglobin saturation exceeds 75% AND whose existing R or L pulmonary arteries are smaller than normal for patient size or who have pulmonary artery pressures above 25 mmHg. (Extent of Aortopulmonary Collateral Blood Flow as a Risk Factor for Fontan Operations, Ann Thorac Surg 1995;59:433-7.)

4. Proximal aortic cannulation may cause poor perfusion below the diaphragm. Whereas, femoral arterial cannulation may cause poor perfusion above the diaphragm.

PRE-EMPTIVE MANAGEMENT - If MAPCAs are suspected:

1. Select circuit size that will easily accommodate blood flow equal to 1.5 times the calculated flow based on a

cardiac index of 2.5 L/min. If QPQS exceeds 2:1 consider step-up to larger oxygenator or circuit.

2. Select a ventricular vent cannula and tubing size that will aspirate at least 50% of the calculated blood flow.

3. Prime circuit with PRBCs if a REDO sternotomy to maintain a relatively high hematocrit should systemic flow falter.

4. Control sweep gas variables to maintain systemic blood flow. The physiologic composition of MAPCAs may vary from patient to patient, with some patients’ MAPCAs being more pulmonary in nature and some patients’ MAPCAs being more systemic in nature. As a result, the effect of varying the paO2 and paCO2 on MAPCA vasoconstriction may also vary from patient to patient with the paCO2 being the more powerful of the two.

5. In the cyanotic infant, begin using an FiO2 of 21% and adjust as necessary to maintain an SVO2 > 65% at normothermia.

6. In larger individuals and adults, use a higher FiO2 initially.

7. Use either a higher or lower paCO2 or pvCO2; whichever method best maintains systemic blood flow.

8. Note changes in mean arterial blood pressure as adjustments are made to the sweep gas composition. Higher mean arterial pressures are probably indicative of better systemic blood flow and less pulmonary blood flow, but this is not always the case.

9. The use of vasopressors to improve systemic perfusion pressure may actually increase the L>R shunt through the MAPCAs.

10. Be aware that if the patient has a residual ASD, VSD or some other residual L>R shunt to the right heart, fully oxygenated blood from the MAPCAs may cross to the right atrium and mix with the systemic venous return giving false SVO2, pvO2 and pvCO2 values.

11. If the true systemic venous return has a low SVO2, it may be masked by the mixing with the fully oxygenated MAPCA blood.

12. The best non-invasive monitor to assess for inadequate systemic perfusion below the diaphragm is a NIRS flank probe over the kidney. The best non-invasive monitor to assess for inadequate systemic perfusion above the diaphragm is a NIRS cerebral probe. (\*Without NIRS monitoring the Detectability score should be increased to 5. This would make the total RPN 5\*3\*5\*1 = 75.)

13. Control the pump blood flow to maintain mean arterial pressures in an acceptable range. Expect blood flow to greatly exceed the calculated blood flow as MAPCAs shunt systemic circulation back to the heart, particularly if a ventricular vent is in operation.

14. If appropriate, maintain high/normal ionized calcium and potassium electrolyte values to keep the heart beating rigorously and prevent over distention. If the aorta is cross clamped, ensure that the ventricular vent is adequately removing blood from the ventricle. Anticipate that vent flow may equal as much as 50% of the calculated flow.

15. Monitor base deficit frequently. If base deficit develops and persists, alter blood flow and/or sweep gas composition as needed to stop the increase in base deficit.

16. If unable to control base deficit accumulation, correct with NaHCO3 and inform the surgeon of the situation.

17. If hypothermia is initiated, utilize pH stat control initially and reassess for base deficit changes during cooling. The higher CO2 utilized for pH stat may vasoconstrict the MAPCAs if they are made primarily of pulmonary-type arterial tissue. If that doesn't work, change to alpha stat where the low CO2 may constrict the MAPCAs if they are made primarily of systemic-type arterial tissue. Watch the NIRS monitor for an appropriate response.

18. These patients are poor candidates for bloodless CPB techniques or some other blood conservation measures due to the need for redo sternotomy and the high potential for under perfusion during CPB. However they may be good candidates for pre-donation because they often have elevated hematocrits.

MANAGEMENT - If MAPCAs are present but were not anticipated:

Maximize blood flow estimating 1.5 times the normal calculated blood flow.

Maximize ventricular vent flow (if vent is used) and calculate MAPCA flow to the lungs by measuring vent flow.

Maximize sweep gas FiO2.

Maximize hematocrit till base deficit subsides or until hematocrit reaches 35%.

Consider hypothermia to mitigate under perfusion.

POST-CPB:

1. IABP support may be limited or ineffective due to MAPCA runoff.

2. Under perfusion below the diaphragm may be due to MAPCAs when using aortic cannulation during CPB. Poor urine output and/or lower body mottling may indicate severe under perfusion below the diaphragm.

3. Under perfusion above the diaphragm may occur if femoral arterial cannulation is used. Low radial blood pressure and low cerebral NIRS level may indicate severe under perfusion above the diaphragm.

4. Patient may regain consciousness normally after surgery. However activated WBCs in the under perfused areas may migrate to the kidneys, lungs and/or brain causing ARF, ARDS, cerebral edema or brain stem herniation as late as 12 hours post-CPB.

5. Consider early traumatic brain protection strategy to mitigate reperfusion injury potential if patients show signs of worsening cognitive dysfunction.

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 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 4\* with NIRS monitoring. Without NIRS monitoring the Detectability score should be increased to 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 would have this congenital defect. 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 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\*3\*4\*1 = 60 if a NIRS monitor is used. Without NIRS the total RPN 5\*3\*5\*1 = 75.)