CPB FMEA #34 Failure to wean from CPB due to acute pulmonary hypertension (APH) crisis.

When I first started doing kids back in the early 80s, acute pulmonary hypertension (APH) was a huge problem. We didn’t see it so much in the run-of-the-mill ASD, VSD or ToF. But in AV canals it was often critical. When patients awoke after surgery they often went into APH and died; “wake up and die” we called it.

When procedures for univentricular patients came along, our deficiencies in treating APH were magnified, especially when low pressure shunts were used. All too many times, patients would fail to wean, not respond to resuscitation and eventually die. Often we tried to keep them alive just long enough to make it to the PICU so their parents could be there when they actually died. I hated that stuff! Finally, ECMO came along. So at last we gave them a better chance to survive.

If possible, elective second or third stage univentricular procedures were put off until flu season was over, knowing that an RSV infection acquired in the hospital could cause APH after surgery and would likely mean the need for ECMO if not outright death. New medications and inhaled NO have greatly reduced the mortality from APH. But I don’t see that they have reduced the Occurrence of APH. Some surgeons still leave a ‘pop off’ between the shunt and atrial chambers so that if APH should occur cardiac output can be maintained even if blood flow through the lungs is obstructed. As univentricular and other congenital patients enter adulthood and require surgery, APH will become much more of an issue to adult perfusionists.

In the early 1990’s, a very effective tool to treat pulmonary hypertension was developed (Naik et al, 1991). Arteriovenous modified ultrafiltration (A-V MUF) prevents or reduces APH caused by interstitial pulmonary edema. The A-V MUF blood flow targets the pulmonary capillary bed, sending hyper-oncotic MUF blood through the pulmonary circulation and pulling excess fluid from the pulmonary interstitial tissues. The effect can sometimes dramatically improve the patient’s hemodynamics in a short period of time (a few minutes) without inotropes or other medications. The effect is most pronounced in babies and children. Post-CPB APH is not nearly so prevalent in adults. But it does occur. The beneficial pulmonary effect of MUF can be enhanced in adults by mixing either 50 mEq of NaHCO3 or 2 grams of mannitol into each liter of residual pump volume before beginning A-V MUF. A detailed explanation of this can be found at < <http://perfusiontheory.com/how-ultrafiltration-and-muf-during-cardiopulmonary-bypass-really-work/> >.

Gary Grist RN CCP, contributor

AmSECT Safety Committee

<garygrist@comcast.net>

CPB FMEA #34 Failure to wean from CPB due to acute pulmonary hypertension (APH) crisis.

FAILURE: Failure to wean from CPB due to acute pulmonary hypertension (APH) crisis.

EFFECT:

1. Fall in arterial O2 sat

2. Fall in systemic BP

3. Fall in end tidal CO2

4. Increase in CVP

5. Increase in airway pressures

6. ECG: S-T segment changes

7. ECHO:

a. RV systolic pressure more than one half systemic systolic pressure

b. abnormal MAP/MPAP ratio

c. worsening tricuspid valve regurgitation

d. RV dilatation or dysfunction

e. systolic septal flattening

8. Failure to wean from CPB:

a. vasoactive support for more than 24 hours

b. ECLS

9. Cardiac arrest

10. Death

CAUSE:

The incidence of post-CPB APH associated refractory RV failure is about 0.1% in routine adult surgery and in 20-30% of patients receiving an LVAD. (\* These VAD patients would have an Occurrence RPN = 4., but a Frequency = 1.) The presence of RV failure after CPB has been associated with a mortality of 44% to 86% in adults. The presence of APH in pediatric heart surgery is at least 3%.

1. Pulmonary vessel vasoconstriction or obstruction

a. Pre-capillary in pulmonary arteries

b. Post-capillary due to LV failure

2. Secondary to left heart disease

3. Secondary to lung disease, pulmonary hyperinflation, high PEEP, hemothorax and pneumothorax

4. Secondary to inflammatory response, pulmonary reperfusion injury, hypoxemia, hypoxia, hypercapnea or blood transfusion.

5.Secondary to thrombotic and/or embolic disease

5.Iatrogenic or idiopathic causes;

a. aortic prosthesis-patient mismatch (PPM)

b. mitral PPM

c. protamine reaction (1.8% of patients)

d. pulmonary edema

e. indeterminate cause.

PRE-EMPTIVE:

1. Check for sea food or antibiotic (AB) allergy history before surgery. If present, consider small test dose of protamine or alternative AB prior to CPB. Consider slow infusion of protamine for heparin neutralization after CPB.
2. Check for history of cor pulmonale, lung disease or chronic pulmonary hypertension prior to surgery. If present and before weaning is attempted:
3. have inhaled nitric oxide (iNO) readily available
4. Consider steroid administration to attenuate inflammatory response.
5. have ECLS readily available
6. If APH is suspected, prior to weaning:
7. Hyperventilate with sweep gas and ventilator.
8. Consider additional alkalization with NaHCO3 prior to weaning.
9. Administer 100% oxygen by ventilator and sweep gas.
10. Consider high frequency ventilation.
11. Check venous blood gas for elevated pvCO2 before and during weaning.
12. For COPD patient, consider return to base line blood gas even if abnormal.
13. Provide additional preload fluid.
14. Perform arteriovenous modified ultrafiltration to target the pulmonary capillary bed and remove pulmonary edema.
15. Consider NIRS monitoring, especially if patient has carotid artery disease.
16. Maintain NSR and AV synchrony

\*\* The potential for post-CPB APH may not be detectable prior to surgery.

MANAGEMENT:

If weaning fails:

1. Attenuate noxious stimuli:

a. deepen anesthesia/sedation

b. administer narcotic

1. Repair PPM if present.
2. Consider pulmonary vasodilators:
3. iNO
4. Milrinone
5. Nitroglycerine
6. Nitroprusside
7. Prostaglandin
8. Prostacyclin
9. For COPD patient, consider return to base line blood gas even if abnormal.
10. Utilize inotropes sparingly to prevent excessive systemic vasoconstriction that could limit blood flow if ECLS is required:
11. dopamine
12. dobutamine
13. epinephrine

5. Consider ECLS:

a. intra-aortic balloon pump if LV failure is causing APH.

b. RVAD or LVAD as indicated

c. ECMO

6. Leave chest open to reduce intrathoracic pressure on pulmonary vessels and air way.

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 Remote with the exception of VAD patients. So the RPN would be a 1\* unless it is a VAD patient or a child. Then the Occurrence RPN would be 4.)

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 3\*\*. The potential for post-CPB APH may not be detectable prior to surgery.)

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 potentially 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\*1\*3\*3 = 45. If this were a VAD patient or a child, the total RPN would be 5\*4\*3\*1 = 60.)