CPB FMEA # 30 High pressure excursion

Friends-

This week’s FMEA was prompted by several perfusion list server postings on high pressure excursions (HPE). See three examples below:

Example 1: “Recently, within the past month or two, we have had 8 or so cases in which the pre-membrane pressures have been extremely elevated - approximately 500 - 600 mmhg. Post membrane pressures were measured as well and were found to be normal or much lower. There haven’t been any issues with oxygenation and therefore we have yet to make the decision to change any of them out. All the patients have recovered without incident.”

Example 2: “Went on CPB, everything is fine. Within 5 minutes my transmembrane pressures are off the charts and I'm only able to flow about 1.5LPM.”

Example 3: “We have seen a similar increase in high pressure excursion cases. In the past two months we have had 6 suspected cases, three of which have been confirmed through post bypass analysis. …. To date we have not identified any alternations or defect in the manufacturing of these oxygenators.”

I have a theory based on my own experience. I have used plain polypropylene (non-silicone coated) hollow fiber oxygenators in neonatal ECMO. Normally these types of oxygenators are not used for ECMO due to their propensity to leak plasma after 3-5 days. But unusual circumstances made their use necessary. I noticed that when the ECMO infants were administered lipids as part of their nutrition regimen, the plasma leaking began soon after starting the lipids. This is what I believe was happening. Lipids already in the patients' blood were absorbed by the hydrophobic fibers, making them partially hydrophilic after 3-5 days. When they became hydrophilic, the fibers swelled, making the diffusion holes larger and allowing proteins to pass. The initiation of parenteral nutrient lipids speeds the process up, causing the fibers to swell and leak more quickly. We normally changed the units out soon after leaking began. I never saw the affluent pressure increase substantially during this process, but we never pumped blood anywhere near the maximum capacity of the oxygenator (<500 mls/min in an oxygenator rated for 2000 mls/min) during neonatal ECMO. So if some portion of the fibers was swelling enough to obstruct a higher blood flow, I probably wouldn't have noticed.

I think that in some adults, the naturally occurring lipids in their blood may cause the fibers to swell, partially blocking the blood path. How quickly the swelling would occur just depends on the quantity of lipids in their blood. If a lipid based anesthetic is administered, the situation might be greatly accelerated causing a high affluent blood line pressure to occur quickly.

The presence of an electromagnetic field is thought to accelerate the deposition of lipids on the fibers by electro-osmosis. Electro-osmosis is the movement of liquid (in this case lipids) induced by an applied electric potential across a porous material, capillary tube, membrane, microchannel, or any other fluid conduit. Pumps can generate a piezoelectric effect or triboelectric charge in the blood. Sometimes this can be seen as an ECG abnormality during CPB. Even very dry sweep gas might generate a static charge across hollow fibers before the gas becomes humidified upon exiting the oxygenator. The heater/cooler might also be a source of electromagnetic potential. A few years ago an oxygenator was withdrawn from the market because plastic fibers in its heat exchanger were ruptured by an internal electro-static discharge during use.

Other things such as cryoprecipitates, mannitol precipitates, temperature and heparin resistance may also play a significant role. So a HPE large enough to limit blood flow through the oxygenator might be caused by a ‘perfect storm’ combination of more than one of these factors. This ‘perfect storm’ scenario could be one reason why the cause of HPE is so difficult to pin down definitively.

This is a difficult FMEA to write because I don’t really know what causes HPE in CPB. Nobody knows. So I have written this FMEA with the best evidence and advice I can find on the subject. Why write about something that we don’t know anything about? Because HPE really do sometimes occur and may cause the perfusionist to make a dangerous intervention. With this FMEA we are acknowledging that a risk does exist. And rather than ignoring the risk because so little is known about it, I think it is worthy of an FMEA discussion.

AmSECT Safety Committee

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FAILURE MODE AND EFFECTS ANALYSIS: CPB FMEA # 30 High Pressure Excursion

FAILURE: Failure to prevent the development of high pressure excursion (HPE) during CPB.

EFFECT:

1. Interruption of CPB due to elevated back pressure from the oxygenator on the roller or centrifugal pump.
2. Change out of the oxygenator due to uncontrollable back pressure.

2. Embolization, hypoperfusion, blood loss, or contamination during oxygenator change out.

CAUSE:

1. Platelet and fibrin deposition on fiber bundle due to patient heparin resistance, particularly in oxygenators with plastic fiber heat exchangers, may partially block blood flow.
2. Cryofibrinogen can occur in up to 7% of patients and may precipitate on cool heat exchanger (HE) surfaces partially blocking blood flow.
3. Mannitol crystals may precipitate on cool HE surfaces partially blocking blood flow.
4. Hyperlipidemia; swelling of hydrophobic fibers to obstruct the fiber bundle blood pathway may be due to fiber lipid adsorption and subsequent hydrophilic fiber response and absorption of water. (Montoya JP 1992).
5. HPE occurs in 1.14% cases with the most common occurrence in males (87.1%) with CAD (96.8%) and use of the lipid soluble IV anesthetic propofol (74.2%). (Meyers GJ 2003).
6. HPE are not oxygenator-make specific or exclusive to hypothermic temperatures or HE.
7. Time to fiber swelling, obstruction and leakage is dependent on phospholipid concentration that is possibly catalyzed by the presence of a weak electromagnetic field caused by a piezoelectric effect, triboelectric charge or a static charge (Montoya JP 1992, Shchipunov YA 1991, Alberts MS 2009, Snijders J 1999, Cohen J 1971).

PRE-EMPTIVE MANAGEMENT:

1. Monitor oxygenator blood inflow pressure.
2. Have the ability to monitor oxygenator blood outflow pressure should a pressure drop measurement across the unit be needed.
3. Add albumin to the prime to pre-coat fibers and HE surface to prevent platelet/fibrinogen and lipid adsorption. Albumin coating will not prevent precipitation of cryofibrinogen.
4. Test patient for heparin resistance and take appropriate precautions to prevent under coagulation.
5. Increase heparin dose as indicated to prevent platelet/fibrinogen deposition on fiber bundle or HE.

MANAGEMENT:

1. Stop cooling to prevent cryofibrinogen or mannitol precipitation on HE surfaces.
2. Presence of ECG abnormality during blood pump operation may suggest the presence of a piezoelectric effect or triboelectric charge. (Sakiewicz PG 2000, Cheng R 2014).
3. Attempt to isolate the electromagnetic charge generation by varying the speeds of different pumps.
4. If electromagnetic charge generation is originating in the arterial roller or centrifugal pump head, attempt to ground out the charge passing thru the affluent oxygenator blood line.
5. An electromagnetic charge may be more prominent at cooler temperatures when tubing is stiffer.
6. A static electricity charge in the oxygenator generated by the dry sweep gas or heater/cooler water flow may not be detectable under normal operating conditions.

7. If flow obstruction becomes severe, splice in new oxygenator in parallel using a PRONTO line (Parallel Replacement of the Oxygenator that is Not Transferring Oxygen). Flow obstruction may eliminate the ability to cool the patient prior to oxygenator change out. A PRONTO line allows for change out without coming off CPB.

\* Without a PRONTO line, perform series change out of the oxygenator. (Increase Harmfulness score to 4; RPN = 4\*1\*5\*3 = 60).

8. Post-traumatic stress disorder therapy should be available if needed for the perfusionist or other surgery team members, particularly if the patient experiences an adverse outcome.

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, 3. However if a PRONTO line is not in standard use to assist in replacing a defective oxygenator, increase the Harmfulness RPN to 4.)

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 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 5 in this example since there are no set of circumstances that can predict this problem.)

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 = 3\*1\*5\*3 = 45. However if a PRONTO line is not in standard use to assist in replacing an obstructed oxygenator, the total RPN would be = 4\*1\*5\*3 = 60.)