Cardiopulmonary Bypass (CPB) Safety Program and Failure Mode Effect Analysis (FMEA)
Open circuit, roller and centrifugal pump

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CPB safety is the avoidance of unnecessary incidents that result in adverse patient outcomes. These are mostly associated with:

1. Malfunctioning/defective equipment and supplies
2. Communication failure between healthcare professionals
3. Human error or incorrect execution of procedures
4. Failure to anticipate adverse events

There are eight steps to safety for any complex medical process:

1. Policies, processes and procedures provide authorization and specific instructions to perform specific tasks in the safest, most effective manner.
2. Safety devices include hardware that can prevent injury or accidents.
3. Checklists ensure consistency and completeness of a task and compensate for limits of memory and attention.
4. Documented competency is used to ensure that personnel are fulfilling their duties properly as required by the appropriate authority. Definition: competency is the ability of personnel to apply their skill, knowledge, and experience to correctly perform their duties. Competency assessment is used to ensure that personnel are fulfilling their duties as required by the appropriate authority. For example, only qualified perfusionists are hired and their competency is assessed and documented by annual evaluations, frequent case reviews by their peers and annual re-certification requirements which include continuing education and documentation of cases performed.
5. Support staff that is adequately trained shall be available on site to assist during procedures.
6. Trouble shooting is problem solving for failures as they occur.
7. Root cause analysis (RCA) identifies the cause of a serious failure after it occurs and proposes actions and conditions that could have prevented the failure.
8. Failure Modes and Effects Analysis (FMEA) examines how a system can fail before the failure occurs.

Definition: Trouble shooting deals with an unanticipated failure while it is occurring using the following plan:

1. Identify what the failure is.
2. Devise an immediate plan to solve the failure.
3. Implement the plan.
4. Assess the results.

Definition: A RCA examines why a system failed, after the failure occurs. The system for performing RCA uses the steps listed below. Usually, the RCA recommends the implementation of an FMEA for the process and the incident being investigated as a means to prevent future occurrences.

1. Choose investigators
2. Get the facts
3. Identify the hazards
4. Identify why controls failed
5. Plan for future events
6. Inform all players
7. Follow-up

In 2001 the Joint Commission Leadership Standard LD 5.2: *Support of Patient Safety and Medical/Health Care Error Reduction* was implemented with the goal of reducing sentinel events and significant errors. Under this standard, hospitals are required to prevent adverse events and errors, rather than just react to them, by conducting proactive risk assessments. A sentinel event RCA is reactive and does not meet this standard on its own. Hospitals (and by implication CPB programs) must provide a "failure mode analysis" for proactive process review. Analysis of a process in active use, such as the operation of a CPB pump, with an FMEA can fulfill the Joint Commission accreditation requirement for proactive risk assessment.

Definition: An FMEA is a technique which 1) identifies potential problems in a design or process by itemizing the conceivable failures, 2) describes the effects of a failure, 3) recognizes the cause the failure, 4) lists specific pre-emptive management or management actions that can prevent or mitigate the failure and 5) numerically ranks the risk of each failure.

This CPB FMEA is based on material supplied by an article from Wehrli-Veit M, Riley JB, Austin JW. A Failure Mode Effect Analysis on Extracorporeal Circuits for Cardiopulmonary Bypass. JECT. 2004;36: 351-357. Additional material has been added by the other perfusionists. The table on subsequent pages details the FMEA.

FMEA Column Headings and Definitions:

Column I. Failure Mode: a list of potential failures.
Column II. Potential Effects of Failure: possible consequences of the failure.
Column III. Potential Cause of Failure: the specific action that can result in the failure.
Column IV. Management/Intervention Column: this column lists specific actions taken by the perfusion staff to prevent each failure mode by pre-emptive management or manage each failure if it does occur.
Column V. Risk Priority Number (RPN): Some RPNs are available for common failure modes associated with open and closed circuits and roller and centrifugal pumps used in cardiopulmonary bypass systems (2). However, these published RPNs do not include the Patient Frequency Rating Scale (sub-column D below). For example, only a few patients (those with a history of a cyanotic congenital heart disease) would be at risk for collateral flow runoff, a significant number would be at risk for excessive bleeding (those with redo sternotomy or some other surgery or those with coagulopathy, etc.). But all patients would be at risk for roller pump failure.

RPNs can be determined subjectively by experienced perfusionists based on the categories listed below. The RPN is calculated by multiplying the four numerical values of sub-columns A, B, C and D; the lowest risk being 1*1*1*1 = 1 and the highest risk being 5*5*5*3 = 375. The RPN prioritizes the risk so that essential or limited resources can first be applied to the failure modes with the highest risk affecting the most patients.

Sub-column A. Harmfulness Rating Scale: how harmful the failure can be?	Sub-column B. Occurrence Rating Scale: how commonly does the failure occur?	Sub-column C. Detection Rating Scale: how easily can the failure be detected before it occurs?	Sub-column D. Patient Frequency Rating Scale: how often does the failure occur in the total patient population?
1. Slightly harmful 2. Low level harm 3. Moderately harmful 4. Seriously harmful 5. Critically harmful	1. Rarely occurs 2. Infrequently occurs 3. Moderate occurrence 4. Frequently occurs 5. Commonly occurs	1. Very easily detected 2. Easily detected 3. Moderately difficult to detect 4. Difficult to detect 5. No means of detection	1. Few patients are at risk 2. A significant number of patients are at risk 3. All patients are at risk
Sub-column E. Calculated RPN: A*B*C*D = E			

Measuring Absolute Risk Reduction (ARR) or Increase (ARI): The FMEA can also be used to rank reductions or increases in risk. See the examples for 2012, 2013 and 2014 at the top of the FMEA table below. The average overall risk at a hospital CPB program for 2012 was (37.5/375)*100 = 10.0%. This predicts that on average one out of every ten cases will have some form of failure. Most of the failures are minor, such as having an 'on pump' hematocrit slightly below the target value or a slightly low pH. Much less frequent are the major failures such an oxygenator change out or aortic dissection. Subjective numerical predictions such as this come very close to the actual frequency of incidents. Further suppose that for 2013 the overall average risk at a hospital was (36.8/375)*100 = 9.8%. This is an ARR reduction from 2012 of 0.2%, (10.0% - 9.8% = 0.2%).

Changes in risk are the result of reductions or increases in sub-column risks. For example, this could be a result of a reduction in the average Occurrence risk (from 1.9 to 1.8) and a reduction in the average Frequency risk (from 2.7 to 2.5). These could be the result of new safety devices, new safety procedures or a change in patient demographics. Or there could be an increase in the Detectability risk (from 2.3 to 2.4.) which could have been the result of new personnel or the addition of high risk procedures not previously used. Calculations of this type can confirm to both inside and outside risk managers and safety assessors that perfusionists are proactively analyzing a process in active use which fulfills the Joint Commission accreditation requirement for proactive risk assessment.

Changes in risk are also the result of previously unidentified risks being incorporated as new FMEAs. For example the RPN increased to 10.9% for 2014 due to the addition of many previously unidentified risks added as new FMEAs. This caused a 2014 ARI over 2013 of 1.1%, (9.8% - 10.9% = -1.1%).

Measuring Relative Risk Reduction (RRR) or Increase (RRI): A risk reduction from 10.0% to 9.8% does not seem to be a significate improvement. However by looking at the RRR, the number is larger. To determine the RRR or RRI divide the ARR for 2013 by the risk for 2012, (0.2% / 10.0% = 2.0% RRR). Calculations of this type can confirm to both inside and outside risk managers and safety assessors that perfusionists are actively improving the safety of CPB from year to year. However the relative risk for 2014 increased over 2013 due to the addition of many new FMEAs for previously unrecognized risks. But even though the risk increased for 2014, safety practice improved because previously unrecognized risks were evaluated by the new FMEAs which included preventative management actions not considered previously.

FAILURE MODE / TROUBLE SHOOTING CATEGORIES	
A) EQUIPMENT FAILURE	G) OXYGENATION/VENTILATION FAILURE
B) DISPOSABLE COMPOMENT FAILURE	H) PROCEDUREAL FAILURE
C) BLOOD LEAKS	I) SPECIAL & EMERGENT SITUATIONS
D) INADEQUATE VENOUS RETURN	J) HYPOTENSION DURING CPB
E) AIR IN THE CIRCUIT	K) HEMORRHAGE
F) WATER HEATER/COOLER FAILURE	L) ELECTROLYTE CONTROL

I. Failure Mode	II. Potential Effects of Failure	III. Potential Cause of Failure	IV. Management/Intervention	V. RPN				
				A. Harmfulness	B. Occurrence	C. Detectability	D. Frequency	E. Risk Priority
			2012 RISK: 37.5/375 * 100 =10.0% AVG RPN:2012	3.4	1.9	2.3	2.7	37.4
			2013 RISK: 36.8/375 * 100 = 9.81% AVG RPN:2013	3.4	1.8	2.4	2.5	36.8
			2014 RISK: 41.0/375 * 100 = 10.9% AVG RPN:2014	3.5	1.8	2.5	2.6	41.0
A. EQUIPMENT FAILURE								
A1. FAILURE: Roller pump failure to turn. 12/12/15	EFFECT: 1. Failure to initiate CPB or unintentional termination of CPB if arterial roller pump fails. a. No blood being delivered to patient b. Hypotension c. Acidosis d. Hypercapnea e. Hypoxia f. Need to hand crank pump g. Organ failure h. Death 2. Failure to initiate cardioplegia, ultrafiltration, ventricular venting or field suckers if secondary pumps fail.	CAUSE: 1.Human error due unfamiliarity with equipment or inadequate training. 2. Internal mechanical or electrical malfunction a. Power cable loose, disconnected or power supply failure b. Internal overload tripped due to over occlusion c. Pump motor, drive belt, main bearing or speed control failure. 3. Total OR power failure.	PRE-EMPTIVE MANAGEMENT: 1. Routine table top scenario discussion or wet lab simulation of a roller pump failure should be regularly performed at least annually or whenever new personnel or unfamiliar equipment is added. The discussion/simulation should include any servo-regulated alarm scenario that could disable the roller pump. 2. Use detailed rather than generalized checklists to reduce failures. Confirm checklist item with action accompanied by conscious out loud verbal repetition before check-off. 3. A flash light or other battery powered portable lighting should always be available ON THE PUMP and checked for proper function should there be an overall loss of power in the OR. Emergency flood lights in the OR do not satisfy this need. 4. All pump instrument stacks should have an uninterruptable DC battery power source in case the AC power source fails. Test for proper battery function and capacity during priming by temporarily disconnecting the AC power source. 5. Confirm by checklist the secure placement of wall plug and proper operation of individual components during set-up and prime. 6. During priming, listen for any unusual noises that might indicate an internal malfunction. 7. Adjust occlusion of each pump head according to accepted practices during set-up and prime. 8. Observe ‘Load’ light of each pump for excessive current use	5	1	5	3	75

			indicating over occlusion or internal power overload. 9. All pumps should undergo a manufacturer's or qualified clinical engineering routine maintenance regularly that specifically includes a battery check or replacement.. 10. Hand cranks should be available for each pump head. 11. A back-up, portable, battery powered centrifugal pump should be available to emergently take over from a malfunctioning arterial roller pump. The process of replacing a modular arterial roller pump with a back-up roller pump may require too much time to safely perform. 12. Back-up roller pumps should be available to replace ancillary roller pumps. 13. Secondary personnel (perfusion assistants or clinical perfusionists) should always be nearby to obtain back-up equipment and assist in emergency procedures. MANAGEMENT: 1. Power loss can be to the entire heart-lung unit or be localized to individual components of the heart-lung unit. 2. Immediately clamp venous line to prevent patient exsanguination in an arterial pump failure. 3. Begin hand cranking in the correct direction at appropriate RPMs before removing the venous line clamp. 4. Monitor flow with an independent flow meter. 5. The perfusionist and secondary personnel should each perform designated scenario-practiced tasks. 6. Check for displacement of electrical plug from wall power or at main connection to pump or circuit breaker if entire instrument stack becomes powerless. 7. If over occlusion or internal overload is suspected temporarily stop pump and loosen occlusion. 8. If occlusion mechanism is defective replace pump with back-up immediately. 9. If main bearing is frozen hand cranking will not be possible. Replace with back-up unit immediately. 10. If individual pump is powerless, turn pump switch off, reset circuit breaker and turn pump back on. 11. If pump is still inoperative, replace with back-up unit. 12. If the arterial pump fails, clamp arterial and venous lines and transfer raceway to a back-up centrifugal pump by cutting the raceway and attaching to the C-pump head.					

			13. If speed controller failure occurs, pump may not respond to speed control knob and even "runaway" at maximum RPMs. Turn off power to pump immediately, begin hand cranking. Replace with back-up unit. 14. Post-traumatic stress disorder therapy should be available if needed for the perfusionist and/or other surgery team members, particularly if the patient experiences an adverse outcome. Note: Specialized cardioplegia pumps may have no manual operation option should they fail unless specialized hand cranks have been supplied by the manufacturer.					
FAILURE: Mismatch of roller pump read out and actual blood flow delivered. 2/6/16.	EFFECT: 1. If arterial pump is affected, there may be inadequate blood supply to the patient causing a. Hypotension b. Acidosis c. Hypercapnea d. Hypoxia e. Shock and organ failure 2. There may be over perfusion to the patient causing a. Hypertension b. High arterial line pressure causing automatic pressure alarm and pump shut off. 3. Secondary pumps can fail to provide adequate cardioplegia, ultrafiltration, ventricular venting or field suckers function.	CAUSE: 1.Human error. 2.Check list error. 3.Roller-head occlusion not properly adjusted. 4.Pump read out not set to proper tubing size. 5.Defective pump operation.	PRE-EMPTIVE MANAGEMENT: 1. The blood flow of the arterial pump is continuously measured using a separate and independently operated Doppler flow meter to assure adequate flow if occlusion is too loose or pump calibration readout is incorrect. (* Without independent flow meter measurement the Occurrence RPN = 2.) 2. Confirm with checklist the proper tubing size calibration on the pump readout. Have secondary personnel confirm. (**Without double check Detectability RPN = 2.) 3. For flow calibration, if available on certain equipment, inter link setup process when setting BSA to tubing size to provide an Index flow as part of a visual check list reminder. 4. Adjust occlusion of each pump head according to accepted practices during set-up and prime. 5. Ritualize tasks in sequence, such as 1) tubing size, 2) occlusion, 3) venous reservoir level alarm, 4) arterial bubble alarm, etc. 6. Indications for over-occlusion can be 'Load' light or jammed tubing. 7. Indications for under occlusion can be failure to collapse raceway while clamped on the inflow side during pump high speed RPM set up testing. 8. Secondary personnel (perfusion assistants or clinical perfusionists) are always in attendance to obtain equipment and assist in emergency procedures. MANAGEMENT: 1. Common clinical indications for under-occlusion or improper calibration can be unexplained abnormal SVO2 values, hypotension, hypertension and acidosis. 2. Check tubing occlusion and calibration, then correct if needed. 3. If malocclusion is suspected temporarily stop pump and tighten or	3	1*	1**	3	9

			loosen pump head as necessary. 4. If occlusion mechanism is defective replace pump with back-up using appropriate caution. (If the arterial pump fails, clamp arterial and venous lines and transfer raceway to a back-up centrifugal pump by cutting the raceway and attaching to the C-pump head.)					
A 3. FAILURE: Failure to prevent roller pump causing spallation to the raceway tubing and sending foreign embolic material into the circulation and damaging cellular blood components. (7/14/16)	EFFECT: 1. Hemolysis 2. Hyperkalemia 3. Decreased hematocrit 4. Hematuria 5. Need for transfusion 6. Spallation of the tubing raceway. 7. Infusion of tubing particulates into the patient's arterial system. 8. Gas embolism 9. Splitting or jamming of the raceway and pump stoppage. 10. Hypoperfusion 11. Stroke 12. Organ failure 13. Bacterial contamination	CAUSE: 1.Over occlusion of pump rollers. 2.Use of silicone raceway tubing.	PRE-EMPTIVE MANAGEMENT: 1. Occlusion is the measurement of raceway tubing cavity cross section due to the compression exerted by a roller pump on the raceway tubing. 2. Spallation is the shredding of the inner lining of the raceway tubing. 3. Over occlusion: excessive roller compression to the point that blood is damaged or spallation results. 4. Heavy duty, medical grade, plasticized polyvinyl chloride tubing of proper durometer for raceway is utilized to combat spallation or prevent rupture if the occlusion is too tight. 5. Silicone tubing should not be used for raceway tubing. 6. Wet occlusion adjustment method: The occlusion on the arterial pump is adjusted during circuit priming using the meniscus level technique and/or using system pressure drop technique. Spall from the arterial pump must pass through any arterial line filter that is used before it enters the patient's arterial circulation.. (*The use of a arterial centrifugal pump eliminates spallation from that position and reduces the Harmfulness RPN to one making the total RPN 1x1x3x3 = 9.) 7. Dry occlusion adjustment method: The occlusion on the vent and suckers pumps is adjusted by clamping the dry inflow line, slowly turning the pump and adjusting roller tension until the inflow side collapses. Loosen the occlusion until the tubing refills with air. Then remove the clamp. Spall from the vent and sucker pumps must pass through the cardiotomy reservoir filter before entering the patient's circulation. 8. Over occlusion of 4:1, 1:4 or other dual tubing cardioplegia (CP) sets using a single pump: Larger 1/4" tube occlusion may need to be deliberately over tightened in order to occlude the smaller tube. Dual headed pumps or other proprietary pumps can prevent this deliberate over occlusion. Spall from the CP pump goes directly into the coronary arteries. (*With only a single head CP pump the Harmfulness RPN should be increased to three, making the total	2*	1	3**	3	18

			RPN 3x1x3x3 = 27.) 9. Tubing temperature changes can alter the occlusion and make the tubing stiffer which increases the risk of spallation. 10. Excessive roller bearing pressure on the raceway tubing results in a visual 'load' alarm on some pump consoles, prompting a loosening of the occlusion. 11. An independent Doppler flow meter is used to assure adequate flow if occlusion is too loose. After going on CPB, tighten the occlusion till the flow stabilizes on the flow meter. Then loosen the occlusion just until the blood flow drops slightly (Peek GJ, 1999). (Caution: There is a risk of finger injury while changing the occlusion as the pump turns. **Without an independent flowmeter the Detectability RPN should be increased to five, making the total RPN 2x1x5x3 = 30.) 12. Initially use a submicron filter pre-bypass to catch particulates and remove it before CPB. 13. Spall may still be generated after the submicron filter is removed. Recirculate the prime through the 20-40 micron cardiotomy filter to remove generated spall and other large particulates (Knopp EA, 1982). 14. Operate ancillary pumps as slowly as possible, even when dry. 15. If circumstances allow, utilize autotransfusion system cardiotomy reservoir and suction for field suckers. MANAGEMENT: 1.Clinical indications for over occlusion can be unexplained hyperkalemia and hematuria. (Caution: hyperkalemia and hematuria are more commonly caused by the administration of cardioplegia and hemolysis from foam generation due to excessive use of the ventricular vent and field suckers.) 2.If the raceway is damaged or split, terminate CPB and replace the raceway. 3.Alternate method: If the raceway is damaged or split, terminate CPB, remove the raceway from the roller pump and connect the remnants to a portable, battery powered centrifugal pump by cutting out the damaged portion of the raceway.					
A4. FAILURE: Centrifugal pump works during priming	EFFECT: 1.Inability to initiate bypass 2.Backflow with entrainment of air into the	CAUSE: 1. RPM too low for forward flow. 2. Level senor or auto clamp set incorrectly.	PRE-EMPTIVE MANAGEMENT: 1.All new critical equipment (including blood pumps) should be wet lab tested under as close to clinical conditions as possible prior to use. 2. Competency of all perfusionists should be fully documented prior	2	1	1	3	6

but when connected to the arterial cannula and bypass initiation is attempted , there is no forward flow. 12/12/15 .	aorta around aortic cannula purse stings. 3.Possible hypotension if patient inadvertently drains into the venous reservoir before bypass can be initiated.	3. Failure to verify line patency. 4. Line pressure control set too low. 5. Flow probe set or placed incorrectly. 6. Defective one way valve. 7. Purge or recirc line left open. 8. VAVD negative pressure set too negative. 9. Pump head magnetic coupling failure. 10. Down ramp safety feature values set out of the limit range.	to using new equipment clinically. 3.Maintain standby equipment necessary to change out a pump including adequate circuit tubing slack. 4.Centrifugal circuit should have a one way valve to prevent complications of backflow. 5.Centrifugal pumps heads should be tested against a high back pressure (300 mmHg) prior to the initiation of bypass. 6.Ensure adequate RPMs before initiating bypass. 7.Check alarm limit setting prior to bypass after servicing by maintenance personnel 8.Initiate alarm systems one by one after initiating bypass. MANAGEMENT: 1.Come off attempt to initiate bypass. If patient drained volume into venous reservoir, infuse volume by hand crank to prevent hypotension. 2.Aggressively hand crank pump head to determine if forward flow can be manually achieved. 3.Disable all alarm features, confirm forward flow via the recirculation line and then attempt to initiate bypass. 4. If failure still occurs splice in standby pump.					
A5. FAILURE: Sweep gas circuit (SGC) failure. 12/12/15	EFFECT: 1.Hypoxemia 2.Hypercapnea 3.Hypocapnea 4.Unnecessary oxygenator change out 5.Gas embolus 6.Room personnel exposed to anesthetic gas vapors. 6. Excessive or insufficient anesthesia.	CAUSE: 1.Defect, crack or leak in the oxygenator or sweep gas system. 2.Sweep gas system component (gas flowmeter, blender, anesthetic gas vaporizer) defective or connections loose. 3.Excessive scavenging of field CO2 flush by pump suckers can mimic oxygenator ventilation failure and hypercapnea. 4.Oxygenator not at lowest level in the blood circuit. 5.Obstruction of oxygenator gas outlet port.	PRE-EMPTIVE MANAGEMENT: 1.Pressure test the sweep gas circuit prior to CPB using an in-line pressure manometer to test for leaks (should be a checklist item). 2.Maximize sweep gas flow during priming to confirm that oxygenator outlet port is not obstructed. a.Only use oxygenators with secondary sweep gas exhaust ports. 3.Maintain an emergency O2 source; E-tank or O2 outlet from the wall or anesthesia machine with a gas line long enough to reach the oxygenator (checklist item). 4.Scavenge the sweep gas exhaust from oxygenator to the OR vacuum gas vent if an anesthetic gas vaporizer is used. 5.Perform visual assessment of the oxygenator exhalation port for a blood leak. 6.Visually confirm correct gas flow through air/O2 blender flow meter prior to CPB. 7.With blood prime visually confirm oxygenation of post-oxygenator blood by color change. 8.Consider the use of one or more of the following sensors:	3**	2	1*	3	18

		6.Oxygenator blood compartment pressure lower than the sweep gas compartment pressure. 7. Incorrect volatile anesthetic liquid added to vaporizer. 8.Empty anesthetic vaporizer. 9. Unnoticed flow meter setting causing excessive sweep gas flow. 10.Ineffective or non-functional (SGC) scavenge system.	a.O2 sensor on sweep gas affluent line. b.CO2 sensor in the oxygenator sweep gas exhaust port. c.Pressure manometer on the affluent sweep gas line. d.Blood SAO2, paO2 or paCO2 sensors sampling from the oxygenator outflow blood line. (*Without sensors the detectability RPN would be a 5, making the total RPN = 90). 9.Ensure that routine maintenance is performed on mechanical components; flowmeters, blender, anesthesia gas vaporizer, blender gas lines and wall connections, scavenge system, etc. 10.After routine maintenance by clinical engineering or manufacturer personnel on sweep gas circuit and its components, conduct a systems check before clinical use. MANAGEMENT: 1. If conditions suggest the failure of the oxygenator to work properly, begin using back-up O2 E-tank connected directly to the oxygenator to eliminate the sweep gas circuit as the cause. 2. If it is determined that the sweep gas circuit is the problem, have back-up personnel trouble shoot the SGC after back-up sweep gas system is initiated so the primary perfusionist can maintain focus on the CPB system. (**If there is no back-up help available increase the Harmfulness rating to 4.)					
B. DISPOSABLE COMPOMENT FAILURE								
B1. FAILURE: Antifoam embolization	EFFECT: Major organ infarction or impaired post bypass organ function	CAUSE: Washout of antifoam from large defoamer foreign surface area.	MANAGEMENT: There are no recent reports of complications from antifoam agents. Newer types of surface coatings have not been reported to mobilize into the blood. Many oxygenators and circuits use X-Coating, phosphorylcholine coating or Trillium coating which have not been reported to embolize into the patient. Stress on the oxygenator antifoam compartment is minimized by the judicious regulation of the ventricular vent and field sucker pumps which are the major cause of foaming in the CPB circuit.	1	1	5	3	15
B2. FAILURE: Cardiotomy or venous reservoir over pressurization or explosion. 12/12/15	EFFECT: 1. Reservoir subject to excessive positive pressure can explode or cause infusion of air up the venous line to the patient causing air embolus. 2. Cracked reservoir can	CAUSES: 1. Failure to properly vent reservoir. 2. Inattention to positive pressure levels both with and without VAVD. 3. Failure of vacuum control regulator or over	PRE-EMPTIVE MANAGEMENT: 1. Reservoir vent port is open and/or VAVD apparatus is safely applied, as confirmed by checklist item with action accompanied by conscious out loud verbal repetition before check-off. 2. Reservoir positive pressure is monitored by two separate pressure relief valves. 3. Pressure should also be monitored by transducer attached to the venous line or the top of the reservoir using a dedicated monitor. A	5	1	1	3	15

	cause loss of volume and interruption of bypass. 3. Exploding reservoir can expose healthcare personnel to plastic shrapnel and gross blood splatter contamination. 4. Risk of patient hypoperfusion, contamination and increased transfusion. 5. Loss of circulating volume causing interruption of bypass. 6. Massive gaseous embolism backing up the venous line and potentially to arterial circuit. 7. Death	pressurization by high speed operation of ventricular vent or cardiotomy sucker pumps. 4. Low level of vacuum during VAVD causing excessive pressure to develop.	dedicated monitor can be servo-regulated to control the sucker and vent pumps which are the two most likely to pressurize the reservoir. The RPN above is dependent on this configuration. (With pressure monitoring only, the RPN would be 5*2*1*3 = 30. Without pressure monitoring or servo-regulation the RPN would be 5*3*5*3 = 225.) 4. The reservoir negative pressure is gauged by a separate vacuum monitor if VAVD is used. 5. The negative pressure is limited to -40 mmHg or less. 6. The normal vacuum or positive pressure can be instantaneously released by removing the safety clamp on vacuum line. MANAGEMENT: 1. If an embolus is caused by air being pushed up the venous line by positive pressure on the reservoir, the venous return line should be immediately clamped and the patient removed from CPB, followed by treatment for air embolus if indicated. 2. If the reservoir should explode under pressure, emergently remove the patient from CPB and replace it. 3. Post-traumatic stress disorder (PTSD) therapy should be available if needed for the perfusionist or other surgery team members, particularly if the patient experiences an adverse outcome.					
B3. FAILURE: Component or connector failure leading to partial loss of circuit integrity but amenable to immediate repair. (5/23/16)	EFFECT: 1.Perfusionist's attention diverted. 2. Blood loss 3.Embolization 4.Hypoperfusion 5.Blood transfusion	CAUSE: 1.Plastic connector, stopcock or other component failure due to poor manufacturing. 2.Split or ruptured tubing. 3.Incorrect assembly or connection within circuit. 4.Clamping tubing over a connector. 5.Rolling the pump over a component during moving. 6.Exposure to high traffic area causing unintentional circuit contact damage. 7.Poor circuit or engineering design.	PRE-EMPTIVE MANAGEMENT: 1.After priming, the circuit is recirculated at high pressure to check for component failures before CPB is initiated. 2.Extreme care is taken when moving a pump with an assembled circuit. 3.Have replacement connectors and components readily available. 4.Have tools needed for repair readily available (clamps, sterile prep materials, sterile tubing cutters, tie bands, protective gloves, eye protection, etc.) 5.Have ancillary personnel readily available to assist. (*If no ancillary personnel immediately available, increase the Harmfulness RPN to 4.) MANAGEMENT: 1. Depending on the location of the component or connector, CPB may need to be emergently terminated and the part replaced.	*2	1	3	3	18
B4. FAILURE: Blood line	EFFECT: 1. Circuit discontinuity 2. Leakage and possible	CAUSE: 1. Tubing disconnecting from connector or circuit	PRE-EMPTIVE MANAGEMENT: 1. Check circuit components and connections during circuit step-up for defects.	4	1	3	3	36

rupture leading to partial loss of circuit integrity but amenable to immediate repair.	air embolus 3. Loss of perfusion 4. Blood loss 5. Hemodynamic instability 6. Death.	component. 2. Tubing cut by roller pump head.	2. Operate at maximum circuit flow and pressure during priming. 3. Test for pressure/pump servo-regulation during priming. 4. Ensure proper tubing position and occlusion in roller pump heads during priming. 4. Use tie bands on 3/8" or greater tubing. MANAGEMENT: 1. If arterial roller pump raceway is involved, stop CPB and clamp arterial and venous lines. 2. If ancillary pump raceway is involved, it may not be necessary to discontinued CPB. 2. Secure disconnected or damaged tubing. 3. Replace tubing damaged by roller head. 4. De-air circuit as needed and resume CPB.					
B5. FAILURE: Clotted CPB circuit; in whole or in part. 12/12/15	EFFECT: 1.Thrombus emboli shed from the circuit. 2.Clotted oxygenator. 3. Forced discontinuance of CPB. 4. Inability to re-institute CPB. 5. Hemodynamic instability 6. Profound shock 7. Stroke and/or organ failure. 8. Death	CAUSE: 1.Procedural: a.Failure to administer heparin before going on CPB b.Mislabeled syringe c.Wrong drug given d.Heparin not injected intravenously e.Low drug activity (old medication or heat exposure). 2. Heparin resistance: see Practice of Cardiac Anesthesia, ed. Little, Brown & Co., Boston., ©1990,Hensley & Martin, p. 218, Table 6-7and p. 553. a. Previous heparin use or ongoing infusion. b. Pregnancy or oral contraceptive use. c. Intra-aortic balloon pump. d. Shock. e. Streptokinase use.	PRE EMPTIVE MANAGEMENT: 1.Circuit prime should contain at least 1 unit heparin per ml of patient circulating blood volume. 2.Routinely check ACT before and after the administration of heparin. 3. If heparin resistance is suspected based on post-heparin ACT, give fresh frozen plasma or ATIII. Then re-check ACT prior to initiating CBP. (Heparin dose response testing may detect heparin resistance prior to CPB. If this testing is done the Occurrence and Detectability scores would be lower: 4*1*3*3 = 36 RPN.) 4. Monitor inflow pressure to the oxygenator and CP heat exchanger to evaluate for high pressure excursion from platelet fibrination. 5. Have replacement components and/or an entire replacement circuit immediately available as back-up. 6. Personnel administering heparin (or protamine) should communicate to the perfusionist and surgeon the type and amount of drug being given. Type and amount should be confirmed for proper dosage by a second person. Use closed-loop communication format for all crucial procedure steps. 7. If heparinized ACT is less than three times the baseline ACT inform surgeon and do not initiate CPB. 8. Give more heparin; re-check ACT. 9. Check ACT equipment prior to testing for proper operation or use different equipment. 10. Administer from a new heparin lot; re-check ACT. 11. Re-check ACT immediately after the initiation of CPB. 12. Re-test frequently for proper anticoagulation to detect the	4	2	4	3	96

		f. Antithrombin III deficiency. g. Disseminated intravascular coagulation. h. Infective endocarditis i. Intracardiac thrombus j. Elderly patient. 3. Platelet fibrination of oxygenator or CP heat exchanger. 4. Inadvertent protamine administration to the pump circuit. 5. Returning shed blood to the extracorporeal circuit after the administration of protamine. 6. Recalcification of a blood prime before adding heparin. 7. Perioperative use of aprotinin or Factor VII.	degradation of heparin effect. 13. Increase ACT testing frequency with hemoconcentration due to circulating heparin removal by ultrafiltration. 14. Use heightened vigilance when aprotinin or Factor VII used prior to, during or immediately after CPB, particularly on REDO cases and when re-heparinization is needed to re-establish CPB. 15. Terminate CPB and the use of cardiotomy suckers before the administration of protamine. MANAGEMENT: 1. If circuit clotting is noticed during CPB consider change out of components or entire circuit before catastrophic failure occurs. 2. If catastrophic failure does occur, the anesthesiologist, surgeon and surgical team should begin resuscitative efforts while the perfusionist prepares back-up circuit for use. 3. Post-traumatic stress disorder (PTSD) therapy should be available if needed for the perfusionist and/or other surgery team members, particularly if the patient experiences an adverse outcome.					
B6. FAILURE: Loss of circuit integrity not amenable to immediate repair.	EFFECT: 1. Termination of CPB 2. Blood loss, possibly extensive 3. Inability to re-establish CPB 4. In ability to wean to patient's own cardiac support 5. Death	CAUSE: 1. Tubing, connector or component separation/breakage due to over pressurization from inadvertent clamping of the arterial line distal to a positive displacement pump. 2. Tube cut by the hinge of a tubing clamp 3. Roller head raceway rupture due to over-occlusion 4. Foreign object in pump head 5. Faulty tubing, connector or component.	PRE-EMPTIVE MANAGEMENT: 1. Maximize system pressure and flow during priming to stress test for component failure before CPB. 2. Document stress testing on checklist prior to initiation of CPB. 3. Have backup system readily available. 4. Have ancillary personnel readily available to assist. MANAGEMENT 1. Terminate CPB. 2. Replace components or replace circuit 3. Remove air. 4. Reestablish CPB.	5	1	3	3	45

		6. Hard shell venous reservoir rupture due to over pressurization or excessive vacuum. a. Malfunctioning pressure release valve during vacuum assisted venous drainage b. Obstructed cardiotomy vent port 7. Isofluorane spillage on polycarbonate components						
B7. FAILURE: Pull-off or blow-off failure between a blood line and a connector or blood port. 3.16.16	EFFECT: 1.Interruption of CPB. 2.Blood loss; exsanguination. 3.Circuit contamination 2. Embolization 3. Hypoperfusion 4. Blood transfusion	CAUSE: 1.Failure to secure tubing to connector or blood port. Without a proper match between tubing (durometer and wall thickness) and connectors, tubing can be a) pulled off due to traction-type tension, b) blown off from a pressure spike, c) or leak. 2.Shape and placement of barbs combined with tubing flexibility determine the force required to connect the blood line. If it is too difficult or too easy to push the blood tubing on, the tubing will not grip properly causing a leak or disconnect. 3.A connector barb or blood port barb is a sharpened ridge or bump that is used to grip the inside of the blood line and seal the connection. As a tube is pushed over the barb	PRE-EMPTIVE MANAGEMENT: 1.Utilize pre-assembled circuits with manufacturer-made connections when possible. 2.Apply cable ties at vulnerable connections. 3.Double check circuit for potential disconnects if equipment is transported. 4.After priming, the circuit is recirculated at high pressure to check for potential pull-off or blow-off sites. 5.Test circuit for pressure/pump servo-regulation during priming to prevent blow-off. 6.Ensure proper tubing position and occlusion in roller pump heads during priming. 7.At the minimum, use cable ties on 3/8” or greater connections. 8. Use checklist to confirm that all connections have been tested and are secure prior to CPB. MANAGEMENT: 1.Depending on the location of the disconnection, CPB may need to be emergently terminated and the disconnection secured, being careful to minimize the risk of blood loss, contamination and embolus. 2. If arterial roller pump raceway or centrifugal pump connections are involved, stop CPB and clamp arterial and venous lines until repaired. 3. If ancillary pump raceway is involved, it may not be necessary to discontinued CPB. 4. Secure disconnection. 5.De-air circuit as needed and resume CPB. 11.A cable tie remover tool should be available should an emergency	4	1	3	3	36

		it expands, gripping and sealing the connection as the tube returns to its original diameter behind the barb. 4.Multi-barbed connectors are more difficult to properly connect and may require significant strength to properly secure. 5.Improperly placed cable ties may be over the tubing but not behind the barb; the place needed to provide additional holding power. 6.Pull-off resistance: Blood lines tend to contract and grab more tightly when pulled. But the tensile strength characteristics will differ for different sizes and grades of tubing and connectors and may allow a blood line and connector to unexpectedly disengage with minimal pulling force. 7.Blow-off resistance: Spikes in hydraulic pressure make blood lines expand, potentially loosening the grip of the barb. Larger blood lines (3/8" and ½") are more susceptible to elevated pressure blow-off than smaller lines (1/4" and 3/16"). 8.A mold seam that produces a slight imperfection on the	arise wherein the ties need removal. 12.Glue bonding of the tubing to the connector should be avoided should an emergency arise requiring the tubing to be removed.					

		connector or blood port may cause a failure. 9.Blood lines and connectors that are wet before assembly are more prone to pull-off or blow-off. 10.Certain blood line coatings may be more prone to pull-off or blow-off.						
B8. FAILURE: Failure of integrated cardiotomy filter due to unexplained obstruction.	EFFECT: 1. IV infusion or blood flow on affluent filter side slowed or stopped. 2. Cardiotomy pressurized as evidenced by audible indication when luer lock cap removed. 3. Affluent filter chamber filled with blood causing reduced circulating volume. 4. Cardiotomy suckers and vent use greatly reduced or stopped 5. Inability to clear operative field of blood. 7. Danger of cardiac distention. 8. Cardiotomy reservoir subject to excessive positive pressure on the affluent side can crack or even explode (See CPB FMEA #6: Cardiotomy/venous reservoir over pressurization http://www.amsect.org/pa	CAUSE: Cause may be unknown but likely similar to high pressure excursions often seen in oxygenators (See CPB FMEA # 30 High pressure excursion <http://www.amsect.org/page/fmea-archives>): 1. Physiologic: a. Platelet and fibrin deposition on filter due to patient heparin resistance. b. Cryofibrinogen can occur in up to 7% of patients and may precipitate on cool cardiotomy filter partially blocking blood flow. c. Mannitol crystals may precipitate on filter surfaces partially blocking blood flow. 2. Manufacturing defect: a. Cardiotomy reservoir and filter sub-assembly incorrectly assembled and at least partially obstructed to fluid passage.	PRE-EMPTIVE: 1. Infuse crystalloid prime through the affluent side of the cardiotomy filter while running the suckers and vent pumps at high speed to check for obstruction caused by mechanical defect or abnormal moisture blockage during priming procedure. 2. Add heparin to the circuit and recirculate some through the cardiotomy filter. 3. Prior to use ensure that the caps on top of the reservoir are not stuck on the ports. Ensuring that the caps on a reservoir are able to come off easily in the case of over pressurization should help avoid problems. 4. Add pressure monitoring of affluent cardiotomy filter: a. Attach a pressure vail with manometer to an affluent filter luer lock OR b. Attach a 10-20 ml syringe with the plunger loosened to an affluent filter luer lock. Plunger is pushed upwards if pre-filter pressure increases OR c. Attach an unclamped, empty, crystalloid prime bag to the affluent side of the filter. This would be the least effective monitor and may inflate even when the filter is unobstructed. 5. Have a plan and supplies readily available pump side should cardiotomy reservoir change out become necessary. Change out should be planned without interrupting CPB. 6. Ensure that knowledgeable assistance is immediately available should change out be necessary. MANAGEMENT: 1. Increase blood temperature if possible. This may reverse filter obstruction caused by cryoprecipitate or mannitol crystals.	*3	1	*1	3	9

	ge/fmea-archives) 9. Cracked reservoir can cause loss of blood volume and interruption of bypass. 10. Exploding reservoir can expose healthcare personnel to plastic shrapnel and gross blood splatter contamination. 11. Forced premature termination of procedure. 12. Forced cardiotomy reservoir component change out. 13. Danger of emboli, hypotension and organ damage as a result of component change out and interruption of perfusion.		2. Change cardiotomy reservoir. Plan for change out should include supplies necessary to connect new cardiotomy reservoir to old venous reservoir, bypassing the old cardiotomy filter, preferably without coming off CPB. *I would give this Harmfulness failure an RPN of 3 if change out does not interrupt CPB and adequate help is available. If change out requires CPB termination OR adequate help is not available, then the Harmfulness RPN would be 4. If change out requires CPB termination AND no adequate help is available, the Harmfulness RPN should be 5. **The Detectability RPN equals 1 if manometer pressure monitoring of the affluent filter is used. If a syringe plunger pressure monitor is used, the detectability would be 2. If an empty crystalloid solution bag pressure monitor is used, the detectability would be 4. If no pressure monitoring is used at all, the detectability would be a 5. The total RPN for this failure is very low if Pre-Emptive Management which includes a plan to change out the cardiotomy reservoir without interrupting CPB, if adequate help is immediately available and if manometer pressure monitoring is used: 3*1*1*3 = 9. On the other hand, if the change out plan requires interrupting CPB, there is no help immediately available and no pressure monitoring is used the RPN would be 5*1*5*3 = 75.)					
C. BLOOD LEAKS								
C1. FAILURE: Heat exchanger leak in the oxygenator or cardioplegia heat exchangers. 12.12.15	EFFECT: 1. Unexplained increase in circuit volume 2. Unexplained increase in K+ (due to hemolysis of RBCs) 3. Unexplained decrease in hematocrit. 4. Unexplained acidosis due to water dilution of HCO3 in the blood 5. Unexplained hyponatremia due to water dilution blood Na+.	CAUSE: 1.Manufacturing defect. 2.Damage during transport or storage.	PRE-EMPTIVE MANAGEMENT: 1. Discard units with any damaged or suspicious packaging prior to use. 2. Maximize water system pressure and flow prior to priming to stress test for heat exchanger failure before CPB. Observe for water entry into dry blood circuit during testing. 3. Document lot#, serial # and stress testing on checklist prior to initiation of CPB. Stress testing my not detect a small leak (Hawkins, 2014). 4. Have backup heat exchanger component system readily available. 5. Have ancillary personnel readily available to assist. 6. Perform regular cleaning/decontamination of H/C water bath to reduce microbial contamination. MANAGEMENT:	4	1	3	3	36

	6. Hematuria 7. Blood visualized in the water lines. 8. Microbial contamination and systemic infection post-operatively, possibly fatal 9. Hypothermia from discontinued use of the water heating system. 10. Multi-systemic organ failure. 11. Death.		1.Immediately turn off heater/cooler and disconnect water lines. 2.Notify surgeon and anesthesiology of emergency and that emergency cooling cannot be performed. 3.Splice in new oxygenator/heat exchanger in parallel using a PRONTO line (Parallel Replacement of the Oxygenator that is Not Transferring Oxygen . Loss of the HE eliminates the ability to cool the patient prior to oxygenator change out. A PRONTO line allows for change out without coming off CPB). 4.Without a PRONTO line, perform series change out of the oxygenator/heat exchanger. (Increase Harmfulness score to 5; RPN = 5*1*3*3 = 45) 5.Replace cardioplegia set. 6.Consider entire circuit change out if practicable. 7.Send blood for culture and sensitivity. 8.Obtain stat blood electrolytes and free hemoglobin 9.Consider mannitol, antibiotics 10.Complete incident report. 11.Save oxygenator or CP HE for investigation 12.Post-traumatic stress disorder (PTSD) therapy should be available if needed for the perfusionist or other surgery team members, particularly if the patient experiences an adverse outcome.					
D. INADEQUATE VENOUS RETURN								
D1. FAILURE: Inadequate venous drainage caused by anatomical abnormality . (4.15.16)	EFFECT: 1. Inability to establish CPB at normal blood flow. 2. Hypotension	CAUSE: 1. Undiagnosed left SVC 2. Undiagnosed anomalous systemic venous return 3. Undiagnosed interrupted IVC with azygos extension 4. Undiagnosed hepatic veins entering right atrium and/or left atrium directly 5. Undiagnosed arterial to venous (AV) shunt a. Undiagnosed PDA b. Uncontrolled systemic to pulmonary shunts c. Arterial cannula flipped into left ventricle 6. Persistent left superior	PRE-EMPTIVE MANAGEMENT: 1. *Perform thorough anatomical examination by ECHO and/or cardiac catheterization prior to CPB. If the examination is abbreviated, the Detectability RPN would be higher. 2. Consider using neck or femoral venous cannulation if SVC or IVC are intact. 3. If using hard shell venous reservoir, maintain vacuum assisted venous drainage capability to augment venous . MANAGEMENT: 1. Establish CPB by atrial cannulation. 2. Add additional cannulae as anatomy and conditions warrant.	2	2	1*	1	4

		vena cava (PLSVC) draining into coronary sinus with absence of an innominate vein. 7. PLSVC draining into the left atrium with the presence of an innominate vein and hypoplastic right SVC. 8. Left atrial isomerism with interrupted IVC and azygos continuation. 9. Left atrial isomerism with abdominal situs inversus a. PLSVC draining into a left-sided morphologically RA. b. IVC located on the left side of the spine with hepatic veins & the right atrium located on the left side.							
D2. FAILURE: Inadequate venous drainage caused by non-anatomical blood line obstruction. 12/12/15	EFFECT: 1. Immediate decrease in venous return blood flow rate or failure to establish CPB. 2. Increase central venous pressure 3. Decreased arteriovenous pressure gradient to vital organs.	CAUSE: 1. Air lock in venous blood line 2. Manual lifting the heart 3. Too small venous cannulae 4. Kinked venous line 5. Misplaced vena caval tourniquet 6. Misplaced or entrapped venous cannula a. Superior vena cava cannula misplaced in azygos vein b. Inferior vena cava cannula misplaced in hepatic vein	PRE-EMPTIVE MANAGEMENT: 1. Have trans-esophageal ECHO available on every case to examine cannula position, atrial and venous anatomy. 2. Monitor CVP to detect pressure build-up caused by physical obstruction to venous drainage. 3. Inspect inflow venous port for man-made debris during circuit set-up (a checklist item?). 4.I f using hard shell venous reservoir, maintain vacuum assisted venous drainage capability to prevent air lock or to augment venous return if small cannulae are used. 5. Monitor cerebral oximetry. 6. Maintain the plasma K+ at the upper limit of normal to help prevent fluid sequestering. Low K+ is associated with splanchnic system fluid retention. 7. Train all table personnel ahead of time in venous line obstruction and have them scan for obstruction problems, especially newer team members. The obstruction may be hidden under drapes, Mayo stand,	2	4	3	3	72

			other tubing, etc. Personnel may unknowingly be leaning on the tube. 8. Incorporation of a venous flow probe may help to detect and correct venous return problems sooner and reduce the RPN; 2*4*2*3 = 48. MANAGEMENT: 1. If internal sequestering of fluid is suspected of causing fluid loss, add additional fluid to the system to restore reservoir level. a. Maintain high normal K+ levels. b. Use 25% albumin or mannitol rather than crystalloid if hemodilution thought to be the cause of fluid loss. c. If mannitol is used do not let the serum osmolality exceed 300 mosmoles/L. 2. Have all personnel at the table scan for venous line obstruction especially newer members. The obstruction may be hidden under drapes, Mayo stand, other tubing, etc. Personnel may unknowingly be leaning on the tube. 3. With surgeon's consent, tilt table in various ways to see if venous return improves. 4. If an undetermined obstruction is the cause, terminate CPB, if possible, investigate or recannulate and correct obstruction. 5.If obstruction cannot be resolved, open atrium and establish sucker bypass. 6.Consider initiating hypothermia so flow can be safely reduced. 7.Reconsider venous cannulae selection, venous line diameter and drop height.					
E. AIR IN THE CIRCUIT								
E1. FAILURE: Hard shell venous reservoir empties. 3/16/16	EFFECT: 1.Air is pumped through circuit with a roller pump and potentially to the patient causing a gross air embolism: a. brain infarction. b. organ dysfunction. c. minor/major disabilities. d. death. 2. A centrifugal pump is de-primed by air and forward flow stops causing:	CAUSE: 1.Human error; lack of attention. 2.Inadequate safety equipment. 3.Inadequate circuit design. 4.Failure of safety systems.	PRE-EMPTIVE MANAGEMENT: 1.Checklist items for level detector and blood line bubble detectors. 2. Activate a level sensor and blood line bubble detectors to automatically turn off the arterial roller pump if the blood level in the reservoir gets too low. 3. The use of a centrifugal (C) pump greatly reduces the possibility of pumping air to the patient from an empty venous reservoir. 4. If using a C-pump with a line clamp, the level sensor can automatically clamp the arterial line to prevent air entry into the patient. 5. If using a C-pump, circuit access should be designed to allow for quick and easy air removal and re-priming of the pump head. 6.Certain hollow fiber oxygenators may filter out some air pumped into them. Certain silicone or silicone coated hollow fiber	5	1*	1*	3	15

	a. brain hypoxia b. organ dysfunction. c. minor/major disabilities. d. death.		oxygenators will not filter air 7.Arterial line bubble trap/filter with air purge line removes air that gets past the oxygenator. (* Not using an arterial bubble trap/filter would increase the Occurrence by one point.) 8. The arterial line has a final bubble monitor to detect any remaining bubbles traveling to the patient. 9. A bubble monitor placed on the venous reservoir effluent line will act as a back-up alarm should the level detector fail. (* Not using a back-up monitor would increase the Detectability by one point.) 9. The cardioplegia circuit may draw air from the emptied circuit and should contain a bubble trap and air detection alarm with automated shut-off, if available. (* Not using a cardioplegia air/bubble alarm pump stop link would increase the Detectability by one point.) 10. Consider heart team management practice of gross air embolus by an FMEA table top discussion or by simulation on a regular basis. Include emergency transport to a hyperbaric chamber. MANAGEMENT: If gross air embolus is suspected: 1. Stop CPB immediately & clamp arterial and venous lines. 2. Place patient in steep Trendelenburg position. 3. Remove aortic cannula. 4. De-air aortic cannula and circuit components. 5. At surgeon's option, perform retrograde SVC perfusion at 100% FiO2 for 1-2 minutes. 6. Utilize intermittent carotid compression during retrograde perfusion, if possible, by anesthesia. 7. Pack head in ice. 8. Institute antegrade deep hypothermic perfusion for 40 minutes at 100% FiO2. 9. Express coronary air if present by massage and cardiac manipulation. 10. Consider administration of barbiturates and steroids by anesthesia. 11. Maintain FiO2 of 100% and sedation for 6 hours after CPB. 12. Maintain negative or siphon pressure on venous line. If C-pump de-primes and stops forward flow: 1. Stop CPB immediately & clamp arterial and venous lines. 2. De-air and re-prime pump head. 3. Resume CPB employing reperfusion tactics (cooling, head ice,					

			steroids, etc.) as needed depending on temperature and time length of flow stoppage. Post-traumatic stress disorder (PTSD) therapy should be available if needed for the perfusionist or other surgery team members, particularly if the patient experiences an adverse outcome.					
E 2. FAILURE Gross air/gas embolism or gaseous microemboli (GME) into the patient [11/19/12]	EFFECT: 1. Gross air/gas emboli entry into the patients' circulation 2. Coronary artery occlusion 3. Cerebral artery occlusion 4. Systemic artery occlusion 5. Vital organ damage. 6. GME leading to diffuse tissue ischemia.	CAUSE: 1. Air entry into the pump arterial line. a. Vortexing or emptying venous reservoir b. Obstruction of gas outlet port c. Leak or kink upstream from a roller pump d. Unclamped arterial line with pump creep or accidental restart e. Reversed flow in arterial line, particularly during A-V modified ultrafiltration f. Sudden loss of MAP during clampless open heart repair through a thoracotomy g. Oxygenator blood compartment pressure lower than the sweep gas compartment pressure h. Oxygenator blood compartment pressure exceeding 500 mmHg rupturing fibers and allowing air entry 2. Air entry into left heart a. Air entry from left atrium and left ventricle residual air pockets after cardiotomy.	PRE-EMPTIVE MANAGEMENT: Use all routine precautions to prevent gas emboli during CPB. 1. Checklist item for level detector and arterial line bubble detector. 2. De-bubble arterial line before connection to cannula 3. Use bubble trap and purge system 4. Prevent de-priming of venous reservoir with adequate circuit volume and level sensor alarm. 5. Use ventricular vent to remove air from the left ventricle 6. Minimize air/blood interface in sucker and vent 7. Minimize vacuum assisted venous drainage pressure. 8. Use a venous reservoir level detector 9. Use an arterial line bubble detector 10. Continuously monitor venous/cardiotomy reservoir for positive pressure using whistling pressure relief values on cardiotomy reservoir and VAVD line to vacuum gauge. 11. Maximize sweep gas flow during priming to confirm that oxygenator outlet port is not obstructed 12. Operate ventricular vent pump during priming to confirm correct directional flow operation13. Use ventricular vent safety valve in vent line. 13. Administer no cardiac stimulants with open/beating heart to provoke ejection during de-airing. 14. Keep fiber bundle of oxygenator as the lowest part of the circuit set-up 15. Keep fiber bundle pressure less than 500 mmHg. MANAGEMENT: If gross air entry is suspected: 1. Stop CPB immediately & clamp arterial line. 2. Place patient in steep Trendelenburg position. 3. Remove aortic cannula. 4. De-air aortic cannula and circuit components. 5. At surgeon's option, perform retrograde SVC perfusion at 100% FiO2 for 1-2 minutes. 6. Use intermittent carotid compression during retrograde perfusion,	4	2	2	3	48

		b. Ejection during de-airing; open beating heart c. Reversed flow in ventricular vent line 3. Air entry into right heart a. Pressurized venous line and venous reservoir. b. Positive pressure release valve failure during vacuum assisted drainage c. Excessive vacuum on right heart aspirating air from central or peripheral IV lines d. Obstruction of venous reservoir exhaust port 4. Air entry from cardioplegia line a. Air into the coronary arteries or aorta from the cardioplegia line during the administration. b. Air entry into the right atrium during modified ultrafiltration. 5. Air entry from other potential sources a. Siphon/vacuum aspiration of air during redo sternotomy due to intrusion into cardiac chamber b. Ruptured blood line c. Runaway pump head d. Cavitation e. Excessive blood/air interface in suckers or vent lines	if possible, by anesthesia. 7. Pack head in ice. 8. Institute antegrade deep hypothermic perfusion for 40 minutes at 100% FiO2. 9. Express coronary air if present by massage and cardiac manipulation. 10. Consider administration of barbiturates and steroids by anesthesia. 11. Maintain FiO2 of 100% and sedation for 6 hours after CPB. 12. Maintain negative or siphon pressure on venous line. MANAGEMENT: If GME entry is suspected: 1. Institute 100% FiO2 for remainder of the case to off-gas nitrogen. 2. Increase venous reservoir volume depth. 3. Minimize suspected source of GME a. Reduce sucker/vent pump speed b. Reduce vacuum assist pressure c. Minimize perfusionist interventions directly into blood circuit.					

E3. FAILURE: Cardiotomy/venous reservoir defoamer filter fails to remove air from the entering the blood in the venous reservoir resulting excessive foaming. [1/27/16]	EFFECT: 1. Air embolus causing organ damage, hypoperfusion, discontinuation of bypass, and increased transfusion donor exposure.	CAUSE: 1.The defoaming capacity of the cardiotomy filter can be exceeded by excessive field sucker or ventricular vent flow. 2.The blood/air interface results in blood foaming. 3.Dangerous foam build-up can occur within seconds in extreme situations. 4.Excessive foaming may be associated with low heparin dose response (HDR),despite ACT > 400sec pre-CPB, by partially clotting the defoaming filter and decreasing its effectiveness. 5.Albumin added to the cardiotomy reservoir may initiate foaming. 6.Patients with volume over load conditions such as valve repair or transplants may have 30%-50% increased blood volume needing better heparin management to prevent defoamer filter clotting.	PRE-EMPTIVE MANAGEMENT: 1.Special circumstances may indicate the need for increased field sucker or ventricular vent use, such as redo procedures in patients with excessive collateral circulation 2.In these instances, larger oxygenator/cardiotomy units that exceed the patient's calculated cardiac output may be utilized. 3.These larger units have the capability to handle increased defoaming needs. 4.Using coated circuits may maintain defoamer function longer. 5.Add piggy-back cardiotomy reservoir as needed to catch and remove foam. 6.*Heparin dose response testing may detect the potential for cardiotomy filter clotting which can lead to foaming. With such testing the Detectability RPN would be 1. 7.Notify surgeon if the risk of excessive foaming is developing. 8.Surgical intervention such as ablation of collateral vessels may help to reduce the excessive cardiotomy blood flow. MANAGEMENT: If excessive foaming develops in the cardiotomy reservoir: 1. Slow the field sucker or vent pump speed, if possible. 2. Remove vacuum assist. 3. Allow excess foam in the cardiotomy to overflow from the ventilation port. 4. Add piggy-back cardiotomy reservoir as needed to catch and remove excess foam. 5. If a closed system is in use, and the bag receives foam, it can be evacuated into the cell saver with very light vacuum. 4. Add fluid volume to the cardiotomy reservoir to increase bubble buoyancy and reduce the egress of bubbles from the venous reservoir. 5. Use 100% oxygen in the sweep gas to minimize nitrogen entrainment and convert bubbles from nitrogen to oxygen. 6. Reduce arterial blood flow to prevent air embolus. 7. Consider cooling the patient.	4	1	2*	3	24
E 4. FAILURE Gross air/gas embolism in coronary artery. [12/10/12]	EFFECT: 1. Acute ECG changes 2. Cardiac arrhythmia 3. Myocardial infarction 4. Decreased contractility 5. Low cardiac output	CAUSE: 1. Gas embolus from aortic cannula. 2. Gas embolus from cardioplegia administration. When the pressure on very	PRE-EMPTIVE MANAGEMENT: Use all routine precautions to prevent gas emboli during CPB. 1. De-bubble arterial line before connection to cannula 2. Use bubble trap and purge system 3. Prevent de-priming of venous reservoir with adequate circuit volume and level sensor alarm.	3	3	4	3	108

	6. Ventricular fibrillation 7. Death	cold blood or crystalloid in the cardioplegia set is suddenly released gas emboli can instantaneously form and be transmitted undetected into the coronary arteries. 3. Gas embolus from residual air in the ventricle, atrium or pulmonary veins.	4. Use ventricular vent to remove air from the left ventricle 5. Minimize air/blood interface in sucker and vent 6. Minimize vacuum assisted venous drainage pressure. 7. Use a venous reservoir level detector 8. Use an arterial line bubble detector 9. Continuously monitor venous/cardiotomy reservoir for positive pressure using whistling pressure relief values on cardiotomy reservoir and VAVD line to vacuum gauge. 10. Maximize sweep gas flow during priming to confirm that oxygenator outlet port is not obstructed 11. Operate ventricular vent pump during priming to confirm correct directional flow operation 6. Use ventricular vent safety valve in vent line. 7. Administer no cardiac stimulants with open/beating heart to provoke ejection during de-airing. 8. Keep fiber bundle of oxygenator as the lowest part of the circuit set-up 9. Keep fiber bundle pressure less than 500 mmHg. 10. Use trans-esophageal echocardiography to monitor air in the left heart prior to weaning. 11. Do not allow the sudden release of pressure on very cold blood or crystalloid in the cardioplegia set. This can instantaneously form bubbles that can be transmitted into the coronary arteries. MANAGEMENT: After weaning from CPB, if there are acute ECG changes, reduced cardiac contractility and/or frequent ventricular extra systoles coronary gross gas embolus should be suspected: 1. Utilize 100% FiO2 on ventilator and sweep gas, increase systolic pressure and vasodilate the coronary arteries with medication if possible. Return to CPB immediately if cardiac output is unsustainable. 2. Place patient in steep Trendelenburg position. 3. If it can be seen, express coronary air by massage and cardiac manipulation. 4. Maintain FiO2 of 100% during CPB. 5. Antegrade syringe coronary de-airing: a. Attach a syringe (50 mls for adults down to 3 mls for infants) to the cardioplegia cannula or root vent. b. Holding the syringe vertically, aspirate blood from the aorta to fill the syringe.					

			c. Apply the aortic cross clamp. d. Gently and gradually depress the syringe plunger to push the embolus out in an antegrade manner. e. Repeat a. through d. if necessary. 6. Retrograde coronary de-airing: a. reinsert coronary sinus cardioplegia cannula b. Cross clamp the aorta c. Open the aorta root vent. d. Gently and gradually flush the embolus out in a retrograde manner. e. Repeat a. through d. if necessary.					
E5. FAILURE: the sudden appearance of massive air embolism in the right heart during bypass initiation that has no definable source. 12/12/15	EFFECT: 1. Air embolus crossing to the left heart while it is still beating. 2. Temporary organ dysfunction 3. Permanent neurological and other vital organ damage. 4. Death.	1.Deviation from standard procedure 2.Air entry originating in the CPB circuit such as unnoticed pressurization of the venous reservoir caused by blocked reservoir vent. 3.Air entry during venous cannulation around purse strings when venous siphon is applied. 4.Air entry originating from central IV or peripheral IV lines with air being sucked in from a loose or broken line connector when venous siphon is applied.	PRE-EMPTIVE MANAGEMENT: 1.Follow procedural checklist. Double check central line/peripheral line connection integrity. 2.Minimize perioperative distractions. 3.Maintain situational awareness during periods of access to patient vasculature. 4.Cerebral monitor may detect trouble early and guide emergency management. Without a cerebral monitor the detectability RPN should be a 3 which raises the total RPN from 30 to 45. 5.Initiate CPB with arterial flow before removing venous clamp. 6.Monitor reservoir pressure; set positive pressure alarm and other safety devices and avoid priming w/ dry venous line. 7.Use of soft shell venous (bag) reservoir would further reduce the risk of retrograde venous air embolus. MANAGEMENT: An air embolus isolated to the right side in a heart with intact septum may not require all the steps listed below. If an unexpected air embolus develops and air is seen in the heart with possible embolus to the left heart: 1.Go to 100% on sweep and ventilation. DC nitrous oxide and anesthetic agents. 2.Trendelenburg position the patient.. 3.Stop bypass if possible, as it may be the source of the air. 4.Transfer aortic cannula to SVC. Debubble cannula if necessary. 5.Retro grade flow to SVC at 40 mmHg max for 1-4 minutes. 6.Apply intermittent carotid compression 7.Ice to head. Start core cooling immediately. 8.Watch aortic cannula site for diminishing air expulsion. 9.When no additional air is expelled, resume antegrade CPB at 20 degrees for at least 45 min. Maintain 80-100 mmHg arterial pressure.	5	1	2	3	30

			10.Manually strip visible air from coronaries through the coronary circulation. 11.Consider medications: neosynephrine, mannitol, barbiturate, Propofol, steroids. 12.Continue ventilating with 100% oxygen for at least 6 hours for nitrogen removal. 13.Continue treatment with mannitol and steroids for 48 hours post-op. 14.Use hyperbaric chamber post-op if immediately available. 15.Post-traumatic stress disorder (PTSD) therapy should be available if needed for the perfusionist or other surgery team members, particularly if the patient experiences an adverse outcome.					
F. WATER HEATER/COOLER FAILURE								
F 1. FAILURE: Heater/cooler (H/C) water hose disconnected or ruptured. (7.14.16)	EFFECT: 1. Non-sterile water spray over sterile operative field. 2. Potential for infection. 3. Potential for water damage to electrical equipment.	CAUSE: 1. Failure to secure water lines properly to oxygenator or cardioplegia heat exchangers. 2. Disconnecting water lines with the water pump running. 3. Loose adjustable hose clamp or other types of tubing connectors or leaking 'O' ring seal. 4. Tubing blow off under pressure. 5. Tubing pull off if pump rolled over tubing during case. 6. Water tube or connection defect.	PRE-EMPTIVE MANAGEMENT: 1. Checklist item for securing water lines to heat exchangers and testing for leaks. 2. Maximize water system pressure and flow prior to priming to stress test for component failure before CPB. 3. Document stress testing on checklist prior to initiation of CPB. 4. Have backup H/C system readily available. 5. Have ancillary personnel readily available to assist. 6. Perform routine maintenance on the H/C system and water lines. 7. If maintenance personnel are responsible for hose replacement and other repairs, the perfusionist should test the H/C for function and leaks prior to bringing it into a clinical area and using it clinically. 8. Have applicable tools and replacement parts readily available for quick repairs, particularly if no backup equipment is readily available.. MANAGEMENT 1. Tighten or replace components or replace water circuit.	1	1	1	3	3
F2. FAILURE: Water heater/cooler equipment fails to heat or cool.	EFFECT: 1. Inability to cool or warm the patient during the procedure. 2. Protracted period required for CPB	CAUSE: Internal mechanical or electrical malfunction 1. Power cable loose, disconnected or power supply failure	PRE-EMPTIVE MANAGEMENT: 1. Manipulate water system for temperature control prior to priming to stress test for failure before CPB. 2. Document stress testing on checklist prior to initiation of CPB. 3. Have backup heater/cooler system readily available. 4. Have ancillary personnel readily available to assist.	1	1	1	3	3

[11/19/12]		2. Internal overload tripped due to heater or compressor malfunction. 3. Compressor failure to cool water 4. Heater failure to warm water	5. Perform manufacturer recommend maintenance and cleaning. MANAGEMENT 1. Replace with a back-up unit. 2. Recirculate and warm or cool water prior to attachment to oxygenator or cardioplegia heat exchanger to prevent sudden temperature changes.					
F3. FAILURE: Contaminated water from the heater/cooler (H/C) may: 1. enter the sterile parts of the oxygenator or cardioplegia heat exchangers (H/E) 2. transmit bacteria through the air (aerosolized) from the H/C water pump or compressor cooling fans exhaust vent into the environment and the patient 3. be transmitted by the operator coming in contact with contaminated H/C water and then	EFFECT: 1.Nontuberculous Mycobacteria (NTM) or other bacterial infections related to cardiothoracic surgeries. 2.Some patients may not present with infections for several months or years after their surgical procedure. 3.This diagnostic delay may cause a failure to communicate critical information in a timely manner to the perfusionist. 4.Hospital staff does not seem to be at risk for NTM or other bacterial infections from H/Cs. 5. Indeterminate risk to patient welfare which may include death: a.A CDC survey found an overall surgical site infection rate of 1.9% with a mortality rate of 3% of those infected patients. b.Deep sternal wound infection complication after median sternotomy has a frequency of 1 to 5%	CAUSE: 1.NTM organisms as well as other bacteria are widespread in nature and can be found in soil and water, including tap water sources. 2.NTM bacteria are typically not harmful, but in rare cases may cause infections in very ill patients and/or in individuals with compromised immune systems. 3.Other bacteria such as Pseudomonas aeruginosa are known pathogens causing serious and fatal infections.	PRE-EMPTIVE MANAGEMENT: 1.Follow standard universal precautions. 2.* **Strictly adhere to the cleaning and disinfection instructions provided in the manufacturer's Instructions for Use (IFU). 3.*Do not use tap water to rinse, fill, refill or top-off water tanks since this may introduce NTM and other organisms. 4.*Use only sterile water or water passed through a filter of 0.22 microns or less. 5.*Ice used for patient cooling should be from sterile water or water passed through a 0.22 micron or smaller filter. 6.*Deionized water and sterile water created through reverse osmosis may corrode the metal components of the H/C. 7.*Direct the H/Cs vent exhaust away from the surgical field to reduce aerosolizing H/C tank water into the sterile field. 8.*Establish regular cleaning, disinfection and maintenance schedules for H/C according to the manufacturer's IFU. 9.*Develop quality control program for maintenance, cleaning, and disinfection of H/Cs. 10. Consider installation of an ultraviolet light water sanitizer in the H/C water lines. MANAGEMENT: 1.*Immediately remove from service any H/Cs with discoloration or cloudiness (biofilm) in the water lines and circuit components which may indicate bacterial growth. 2.*Consult in-house Infection Control officials for follow up measures. 3.*Report events of H/C contamination to the manufacturer. 4.*Consider performing environmental, air, and water sampling and monitoring if H/C contamination is suspected. (Environmental monitoring requires specialized expertise and equipment to collect and process samples, which may not be feasible in all facilities.)	3	1	5	3	45

introducing the bacteria into the blood circuit by routine contact. (12/22/15)	with a mortality rate ranging from 10% to 47%. c. There is no systematic reporting of NTM infections and precise incidence data are lacking. Several state health departments report that the number of isolates of NTM has surpassed the number of M. tuberculosis isolates. (De Groote M, Huitt G. Infections Due to Rapidly Growing Mycobacteria. Clinical Infectious Diseases Volume 42, Issue 12, pgs 1756-1763.)		5.**Health care facilities should follow their internal procedures for notifying and culturing patients if they suspect infection associated with H/C. 6.*Submit a report to the manufacturer and to the FDA via MedWatch if H/C contamination has led to patient infections. 7.*Hospitals obligated to the FDA's user facility reporting requirements should follow the reporting procedures established by their facilities. 8.*Perfusionists should submit voluntary reports of infection transmission associated with H/C or reports describing difficulty following the manufacturers' IFU to the Medical Device Reporting process.					
G. OXYGENATION/VENTILATION FAILURE								
G1. FAILURE: Oxygenator gas exchange failure [11/19/12]	EFFECT: 1. Failure to oxygenate the blood effectively. a. Blood exiting the oxygenator will appear dark red or black. b. SAO2 and SVO2 will decrease. 2. Failure to remove CO2. a. Blood pH will decrease due to increasing pCO2. 3. Patient hypoxemia 4. Patient hypercapnea	CAUSE: 1. Sweep gas system connections loose. 2. Defect, crack or leak in the oxygenator or sweep gas system 3. Failure of oxygen gas supply. 4. Condensation within the hollow fibers of the oxygenator 4. Clotted oxygenator. 5. Gas exchange failure in oxygenator due to fibrous, platelet and cellular accumulations on the gas exchange surface which can increase resistance to blood flow and abrogate the blood path	PRE-EMPTIVE MANAGEMENT: 1. Pressure test the sweep gas circuit prior to CPB to test for leaks. 2. Maintain an emergency O2 source (E tank or O2 outlet from the anesthesia machine) with a line if the sweep gas system fails. 3. The sweep gas is scavenged from oxygenator to the OR vacuum gas vent. This allows for visual assessment of the oxygenator exhalation port for a blood leak. It also removes any volatile anesthetics that may be used during CPB. 4. Visually confirm gas flow through flow air/O2 mixer flow meter. 5. With blood prime visually confirm oxygenation of post-oxygenator blood by color change. 6. Use continual monitoring of oxygenator gas exhaust w/ CO2 sensor to confirm adequate sweep gas flow. 7. Pre-coat membrane with albumin during priming to prevent excessive platelet adhesion and membrane clogging at initiation of bypass. 8. Maintain an activated clotting time of at least 3X baseline to prevent oxygenator clotting. MANAGEMENT: 1. Maximize sweep gas FiO2 2. Stop any CO2 titration	4*	2	3	3	72

			3. Increase sweep gas flow rate. 4. Initiate use of emergency O2 from the anesthesia machine or an O2 E cylinder. 5. Pressure check for sweep gas circuit for leaks at connections, blender, flow meter & along length of tubing. 6. Tap on oxygenator to displace condensation that might be plugging the gas channel. 7. Maintain or initiate hypothermia if possible. 8. Splice in new oxygenator in parallel using a PRONTO line (Parallel Replacement of the Oxygenator that is Not Transferring Oxygen) 9. Without a PRONTO line, perform series change out of the oxygenator. (*Increase Harmfulness RPN to 5; RPN = 5*2*3*3 = 90)					
G2. FAILURE: Inadequate cerebral oxygenation as indicated by cerebral oximetry. 12/12/15	EFFECT: 1. Greater than 20% drop from baseline or a decline to less than 50%. 2. Cerebral hypoxia and subsequent brain damage.	CAUSE: 1.Improperly placed or loose sensors. 2.Improper aortic cannula placement. 3.Inadequate perfusion pressure. 4.Inadequate pump blood flow 5.Low paO2 6.CO2 imbalance 7.Inadequate anesthesia 8.Hemodilution 9.Diabetes 10.Severe cerebrovascular disease	PRE-EMPTIVE MANAGEMENT: 1.The cerebral oximetry monitor is the only pre-emptive intervention available to prevent or quickly reverse cerebral oxygen desaturation. Without it the RPN would be 4*3*5*3 = 180. 2.Ensure that sensors are properly placed. 3. Record intervention to reverse desaturation. MANAGEMENT: 1.Check head & cannula position. 2.Increase the mean arterial pressure. 3.Increase pump flow rate. 4.Increase systemic oxygenation. 5.Increase paCO2. 6.Increase volatile anesthetic depth or administer IV anesthetic bolus. 7.Increase hematocrit by ultrafiltration 8.Consider hypothermia. 9.Consider PRBC transfusion for low hematocrit. 10. In cases w/ diabetes or severe cerebrovascular disease, all these interventions may fail. This warns of a higher risk of post-op neurologic deficits or stroke.	4	3	1	3	36
G 3. FAILURE: Failure to prevent the development of high pressure	EFFECT: 1.Interruption of CPB due to elevated back pressure from the oxygenator on the roller or centrifugal pump. 2.Change out of the	CAUSE: 1.Platelet and fibrin deposition on fiber bundle due to patient heparin resistance, particularly in oxygenators with plastic fiber heat exchangers, may	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 to prevent platelet/fibrinogen and lipid adsorption. Albumin coating will not prevent precipitation of cryofibrinogen.	3*	1	5	3	45

excursion (HPE) during CPB. 3/17/16	oxygenator due to uncontrollable back pressure. 2. Embolization, hypoperfusion, blood loss, or contamination during oxygenator change out.	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	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. 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.					

		Effect or Triboelectric Charging (Montoya JP 1992, Shchipunov YA 1991, Alberts MS 2009, Snijders J 1999, Cohen J 1971)						
H. PROCEDUREAL FAILURE								
H1. FAILURE: Separation of arterial line or raceway tubing line. [11/19/12]	EFFECT: 1. Loss of prime and blood 2. Risk of air emboli 3. Temporary loss of circulation.	CAUSE: 1. Failure of high pressure alarm to stop arterial pump; safety system failure. 2. Improper placement of a clamp on arterial line; operator failure. 3. Obstruction or twist in the arterial blood line post-pump, causing a sudden increase in line pressure	PRE-EMPTIVE MANAGEMENT: 1. Circuit pressure monitor on the arterial line is set at 500 mmHg to stop the pump before the tubing separates. 2. Larger blood lines are secured to the various circuit components by additional tie straps. MANAGEMENT: 1. Stop the pump. 2. Clamp the blood line 3. Secure the line as needed. 4. Add circuit volume or recirculate the circuit to remove air, as needed.	3	1	3	3	27
H2. FAILURE: Separation of cardioplegia line. [11/19/12]	EFFECT: 1. Loss of prime and blood 2. Risk of air emboli 3. Temporary loss of cardioplegia capability.	CAUSE: 1. Failure of high pressure alarm to stop cardioplegia pump; safety system failure. 2. Improper placement of a clamp on cardioplegia line; operator failure. 3. Obstruction or twist in the cardioplegia blood line post-pump, causing a sudden increase in line pressure.	PRE-EMPTIVE MANAGEMENT: 1. Cardioplegia circuit pressure monitor is set at 500 mmHg to stop the pump before the tubing separates. 2. Larger blood lines are secured to the various circuit components by additional tie straps. MANAGEMENT: 1. Stop the cardioplegia pump. 2. Clamp the blood line 3. Secure the line as needed. 4. Add circuit volume or recirculate the circuit to remove air, as needed.	1	1	3	3	9
H3. FAILURE: Inadequate anticoagulation by heparin administration.	EFFECT: 1. Clotting of circuit during the procedure with loss of cardiopulmonary support. 2. Thrombotic emboli infused into the patient.	CAUSE: 1. Procedural: Failure to administer heparin before going on CPB a. Heparin administration forgotten. b. Mislabeled syringe c. Wrong drug given	PRE-EMPTIVE MANAGEMENT: 1. Routinely check activated clotting time (ACT) before and after the administration of heparin. 2. Anesthesiologist and perfusionist should both verbally agree that heparin has been given in the correct dose prior to CPB. 3. If heparinized ACT is less than two times the baseline ACT inform surgeon and do not recommend initiating CPB. 4. Give more heparin; re-check ACT.	3	2	1	3	18

		d. Heparin not injected intra-vascularly e. Low activity (old medication or heat exposure). 2. Heparin resistance: see Practice of Cardiac Anesthesia, ed. Little, Brown & Co., Boston., ©1990,Hensley & Martin, p. 218, Table 6-7and p. 553. a. Previous heparin use or ongoing infusion. b. Pregnancy or oral contraceptive use. c. Intra-aortic balloon pump. d. Shock. e. Streptokinase use. f. Antithrombin III deficiency. g. Disseminated intravascular coagulation. h. Infective endocarditis i. Elderly patient.	5. Check ACT testing equipment for proper operation or use different equipment. 6. Administer from a new heparin lot; re-check ACT. MANAGEMENT: 1. Re-check ACT immediately after the initiation of CPB. 2. If heparin resistance is suspected administer fresh frozen plasma. 3. Re-check ACT. 4. If circuit clotting noticed consider change out of components or entire circuit.					
H4. FAILURE Excessive arterial perfusion cannula pressure.	EFFECT: 1. Failure to initiate CPB 2. Damage to the aorta. 3. Hypoperfusion 4. Death	CAUSES: 1. Small aortic cannula 2. Arterial cannula mal-positioned or cannula is too large or small. 3. Too small of an aortotomy 4. Fibrotic vessel wall 5. Calcific plaque 6. Cannulation of an aortic false lumen. 7. Aortic dissection.	PRE-EMPTIVE MANAGEMENT: 1. Confirm appropriately sized arterial cannula with surgeon prior to insertion 2. After insertion of arterial cannula but before initiation of CPB monitor static arterial line pressure for pulsatile wave pattern and correlate patient's blood pressure to the arterial line pressure. 3. At initiation of CPB monitor arterial line pressure for sudden or unexpected increases before removing venous line clamp. MANAGEMENT: 1. If arterial line pressure is high after the initiation of CPB, reduce flow if needed and check for cannula torque at insertion site. 2. Resume flow and re-evaluate arterial pressure. 3. If pressure remains excessive stop CPB and replace arterial	5	1	1	3	15

			cannula with larger or smaller one or select a different cannula site. 4. Re-evaluate again and re-cannulate as necessary. 5. Perform intraoperative ECHO to check for aortic abnormalities. 5. Consider an alternate cannulation site.					
H5. FAILURE Reverse cannulation: 1) by reversal of arterial and venous cannula position or 2) the inappropriate sequence of first inserting the venous cannula with the potential for exsanguination into the pump before arterial cannula insertion.	EFFECT: 1. Improper operation of the CPB pump 2. Hemodynamic instability 3. Flaccid aorta and tense vena cava by palpation 3. Cardiac damage 4. Unintentional exsanguination 5. Hypoperfusion 6. Death	CAUSES: 1. Reversal of arterial and venous cannulae sites; failure to follow proper cannula assembly and insertion sequence. 2. Reversal order of cannulation; venous cannula inserted first with potential for exsanguination into the pump before insertion of arterial cannula is completed. 3. May be masked by AV shunts, malformations and septal defects.	PRE-EMPTIVE MANAGEMENT: 1. Clamp and divide the arterial-venous loop at the appropriate position 2. Attach venous cannula assembly to venous line 3. Confirm correct configuration by filling venous cannulae with crystalloid from syringe attached to venous sample port of oxygenator. 4. Surgeon always performs arterial cannulation first, either aortic or femoral artery cannulation. 5. Once tubing is connected to the arterial cannula, monitor the arterial line pressure for appropriate pressure reading. Notify surgeon of status of the pressure in the line before initiating CPB. MANAGEMENT: 1. If patient exsanquinates into pump via the venous line before insertion of the arterial cannula, immediately clamp venous line between venous sample port and venous reservoir. 2. Connect feed line from bubble trap to venous line sample port. 3. With the arterial line clamped, transfer the purge line from the bubble trap from the cardiotomy reservoir to the venous line sample port and refill the heart retrograde through the venous line. 4. Clamp the bubble trap purge line upon initiation of CPB and return it to the cardiotomy reservoir. 5. If arterial and venous cannulae positions are reversed, stop CPB immediately and clamp arterial and venous lines. 6. Steep Trendelenburg. 7. Reduce cerebral damage; consider steroids, mannitol, barbiturates and hypothermia.	5	1	1	3	15
H6. FAILURE Cephalic artery hyperperfusion. (12/22/15)	EFFECT: 1.Vaporous cavitation from trapped arterial cannula turbulent flow with formation of gaseous emboli. 2. Misdirected or	CAUSE: 1.Arterial cannula positioned or perfusion jet directed into a carotid artery. 2.Arterial cannula too small for flow resulting in	PRE-EMPTIVE MANAGEMENT: 1.Once tubing is connected to the arterial cannula, monitor the arterial line pressure for appropriate pressure reading. 2.Perform test turn to check for cannula obstruction. 3.Notify surgeon of status of the pressure in the line before initiating CPB and as flow is increased. 4.Observe cerebral oximetry monitor for any change after	3	1	1*	3	9

	inadequate blood flow to the brain or other organs. 3. Unilateral facial edema 4. Lacrimation 5. Petechiae 6. Serosanguineous otorrhea 7. Rhinorrhea 8. Metabolic acidosis 9. Cerebral edema 10. Carotid arterial rupture 11. Carotid intimal flap obstructing arterial flow. 12. Blanching of the face 13. Pupillary dilation 14. Conjunctival chemosis. 15. Low BP measured by left radial or femoral arterial catheter. 16. Unequal cerebral O2 concentration by cerebral oximetry. 17. Post-operative delirium. 18. Post-operative brain damage. 19. Death	excessive force of high pressure blood jet.	cannulation. 5.Use TEE to check arterial cannula position. 6.Check by Anesthesia for bilateral carotid pulses and bruit before and after cannulation and for excessive or unbalanced bruit after the initiation of CPB. 7.Transcranial Doppler (TCD), if available, can be utilized to detect vaporous cerebral emboli, improper cannulation or improper clamping of aortic arch vessels. (* The Detectability RPN equals 1 only if all pre-emptive management processes are used: pressure/flow assessment, cerebral oximetry, TEE, carotid palpation/auscultation and TCD. Add one point for each item not used. If no pre-emptive management processes are used the Detectability RPN would be 5, making the total RPN 45, five times higher risk.) MANAGEMENT: 1. Reposition arterial cannula. 2. Consider mannitol, steroids, barbiturates & hypothermia to reduce cerebral damage.					
H7. FAILURE: Undersized aortic cannula used for cannulation.	EFFECT: 1. High velocity blood jet may cause intimal damage & occlude vessel 2. RBC lysis from shear stress 3. Cavitated out gassing from turbulent blood flow 4. Inadequate blood flow due to high pressure alarm.	CAUSE: 1. Incorrect selection of cannula for anticipated blood flow. 2. Failure to anticipate super normal blood flow. 3. Aorta size or exposure may limit the cannulae size.	PRE-EMPTIVE MANAGEMENT: 1. Select aortic cannula size based on anticipated blood flow. The need to compensate for aortic to pulmonary collaterals may require a cardiac index 1.5 times greater than normal. Other conditions may also require a larger cannula. 2. Anticipate an additional femoral or axillary cannulation if the aorta is abnormally small or difficult to cannulae with a large-enough cannula. MANAGEMENT: 1. Replace aortic cannula if CPB can be temporarily terminated. 2. Reduce temperature to allow for a reduction in blood flow.	3	1	1	3	9

	5. Physiologic consequences associated with under perfusion.							
H8. FAILURE: Aortic purse string failure and cannula dislodgement	EFFECT: 1. Aortic dehiscence 2. Immediate termination of CPB 3. Severe blood loss 4. Critical hypotension 5. Death	CAUSE: 1. Broken purse string suture 2. Purse string tissue pull through due to inadequate placement or tissue friability.	PRE-EMPTIVE MANAGEMENT: 1. Use two purse strings for aortic cannulation. 2. Use femoral cannulation if aortic tissue appears friable. MANAGEMENT: 1. Control aortotomy, replace suture and aortic cannula if CPB can be temporarily terminated. 2. Place patient in steep Trendelenburg's position. 3. Consider mannitol, steroids, barbiturates & hypothermia to reduce cerebral damage. 4. Side clamp dehisced aorta and convert to femoral or neck vessel cannulation.	3	1	1	3	9
H9. FAILURE: Perfusionist skills decay. 12/12/15	EFFECT: 1. Temporary loss of training, acquired skills and knowledge. 2. Errors due to loss of speed and accuracy. 3. Decay in personal quality performance of tasks. 4. Indeterminate risk to patient welfare.	CAUSE: 1. Lack of recent experience. 2. Extended interval such as vacation, illness or leave of absence impairs skill retention. 3. Excessive mental stress or pain. 4. Failures of routine (closed loop tasks) more affected: a. check list failures b. sequenced task failures c. less sensitivity to performance variations 5. Failures of tracking and problem solving are less affected by temporary skill loss. a. these tasks are not defined by routine start and end b. there is better retention of skill to address these types of issues.	PRE-EMPTIVE MANAGEMENT: 1. Use detailed rather than generalized checklists to reduce failures. Confirm checklist item with action accompanied by conscious out loud verbal repetition before check-off. 2. Use active case peer review of newly returned perfusionists. 3. Maintain a policy requiring active proctoring for perfusionists returning from extended leave. 4. Establish a support system for the perfusion team during clinical practice with active assessment by the chief perfusionist. 5. Strive to develop a simulation system to help maintain competency. MANAGEMENT: 1. Factors that affect performance after a temporary loss of skill include complexity of the skill and motivation of the perfusionist. 2. Focus on the immediate task at hand. 3. Get support personnel help for an objective assessment.	2	3	2	3	36

H 10. FAILURE: Perfusionist's inability to deal with fear in a stressful situation.	EFFECT: 1.Inability to make correct decisions or take effective action due to fear. 2.A delay or failure to communicate critical information. 3. Indeterminate risk to patient welfare.	CAUSE: 4.Even an experienced perfusionist can feel a level of fear caused by a set of circumstances that develop beyond his or her control. 5.Fearful intimidation develops between experienced and less experienced team members. 2. Fear has four increasing levels of severity: a. Apprehension is the controllable worry about a future mishap. b. Stress is a state of mental, emotional or physical tension that requires a mental, emotional or physical adjustment or response. c. Anxiety is an uncomfortable nervousness involving self-doubt about one's actions to control an imminent event or an uncertain outcome. d. Panic is a surge of overwhelming fear causing unthinking or irrational behavior.	PRE-EMPTIVE MANAGEMENT: 11.Obtain the proper educational preparation and training prior to beginning a stressful situation. 12.Do not attempt aspecific task unless properly trained and qualified. 13.During training or orientation and with a competent mentor, expose the student or inexperienced perfusionist to as many difficult situations as possible. 14.Emphasize multitasking, developing organizational skills, improving communication skills and teaching team work building. 15.Maintain up-to-date and readily available P&P and surgeon preference manuals for all procedures. 16.Perform frequent skills checkoff for emergent procedures. 17.Perform annual competency review. 18.Maintain up-to-date continuing education and evidence based practice. 19.Provide strong clinical sites for training programs. 20.Maintain the mental and emotional preparation that anticipates difficulty prior to a situation becoming stressful; i.e., have good situational awareness. 21.Emergency situations pose the greatest risk of fear because there is less time for preparation, less resource staff availability and often occur during off-hour periods. 22.Have support personnel available whenever possible. 23. Consult with experienced perfusionists prior to beginning a known stressful situation whenever possible. (*If no support personnel or experienced perfusionists are available increase the Harmfulness RPN to 3.) 24.Implement routine team training to prevent intimidation and teach ways to handle bullying from other team members. 25.Avoid all unnecessary distractions, i.e., cell phone calls, loud music and excessive jocularity. 26.Have available stress management resources for holistic approach to maintaining team member long term mental and physical health.. (**Fear and insecurity may be hidden by the facade of confidence, making self-fear more difficult to detect or an RPN of 4, low.) MANAGEMENT: 9.Focus on the immediate task at hand particularly in a rapidly developing or explosive situation.	1*	1	1**	3	3

			10.Prioritize other tasks as they develop. 11.Get support personnel help for an objective assessment of the situation before panic sets in. 12.Try to physically complete the task even if it is with detailed instructions from support personnel. Completing a stressful task can be valuable experience. 13.Post-traumatic stress disorder therapy should be available if needed for the perfusionist or other team members, particularly if the patient experiences an adverse outcome.					
H11. FAILURE: Rupture of the coronary sinus (CS) with retrograde cardioplegia (RCP).(Rev. 1/2/16)	1. Inadequate myocardial protection. 2. Increased morbidity with additional surgical procedure to repair coronary sinus. 3. Death.	1. Damage to CS during insertion of RCP cannula. 2. Damage to CS during infusion of RCP because of high CS pressure. 3. Migration and misplacement of the RCP cannula during manipulation of heart. 4. Overinflating of balloon. 5. Loss of focus by the surgeon or perfusionist to quickly recognize inappropriate pressures. 6. When utilizing a multi-perfusion device, ports left open inadvertently giving false pressure and flow readings to the intended RCP target area. 7. Women low body mass index*. 8. Overly forceful insertion due to CS web*. 9. Fragility of vessels in thin patients*. 10. Small CS*. 11. Elderly patients have friable tissues and are more liable to rupture. 12. Frequency of CS	PRE-EMPTIVE MANAGEMENT: 1.Care during insertion of RCP cannula to avoid trauma to CS. 2.If the patient is not on CPB during insertion of the RCP cannula, the perfusionist should monitor EKG closely for ectopy; risk of patient going into V-Tach. 3.Monitor of MAP during insertion of RCP cannula for loss of cardiac output (if patient is not on CPB) secondary to positioning and manipulation of the heart. 4.Perfusionist should be prepared to go on CPB emergently to provide cardiac support. 5.If the patient is on CPB when the surgeon is inserting the RCP cannula, the perfusionist should assist the surgeon by providing adequate filling of the right heart to assist in palpating the CS and placing the RCP cannula. 6.Once the RCP cannula is placed, the CVP line should be re-zeroed. 7. If CS pressure >20 mmHg after zeroing and before RCP infusion, the cannula may be wedged in either the greater cardiac vein or posterior descending vein. 8. If CS pressure < 20 mmHg during RCP infusion, the balloon may not be occluding the CS, the RCP cannula may have slipped back into the right atrium, or there may be an anomalous left superior vena cava or unroofed CS. 9. Initiate RCP flow slowly starting at 2.5%-5% of total calculated cardiac output while monitoring RCP line pressure and CS pressure. 10.Monitor CS pressure during RCP infusion with a target pressure of 25-30 mmHg and a safe pressure range of 20-40 mmHg. 11.Pressure >40-50 mmHg suggests that the CS is becoming distended or that the RCP cannula has entered a coronary vein, both of which increase the chance of the sinus rupturing. 12.Should the pressure spike the perfusionist should immediately STOP the delivery.	5	1*	4	3*	60

		rupture is about 1%* with an experienced team.	13. The perfusionist should confirm that the surgeon sees the cardioplegia coming out of the aorta if the aorta is open. 14. If the aorta is closed the aortic root vent should always be on. Without proper venting the coronary arteries could dissect. 15. Maintain meticulous communication among the team with each delivery. 16. If the Frequency of use is common (3*) then the Occurrence will be low (1*) and the total RPN will be 60. However it the Frequency of use is low (1*) then the Occurrence will be high (4*) and the total RPN will be 80. MANAGEMENT: 1.Since retrograde flow is so dependent upon adequate pressure and flow, any disparity in either pressure or flow should cause concern. Some examples would be: a) high flow with small pressure increase b) barely able to flow with very high pressures. 2.Discontinue RCP cardioplegia and repair CS. REFERENCES: 1. Drinkwater DC Jr, Cushen CK, Laks H, Buckberg GD. The use of combined antegrade-retrograde infusion of blood cardioplegic solution in pediatric patients undergoing heart operations. J Thorac Cardiovasc Surg. 1992; 104(5): 1349-55. 2. Hensley FA Jr, Martin DE. A Practical Approach to Cardiac Anesthesia, 2nd Ed. Boston: Little, Brown and Company; 1995. 3. Gravlee GP, Davis RF, Utley JR. Cardiopulmonary Bypass, Principles and Practice. Baltimore: Williams & Wilkins; 1993. 4. Lich BV, Brown DM. The Manual of Clinical Perfusion, 2nd Ed. Fort Myers, FL: Perfusion.com; 2004.					
H12. FAILURE: Inadequate myocardial protection of right ventricle after retrograde cardioplegia(by DZ 11/13)	1.Right ventricular dysfunction post-CPB. 2. Need for mechanical circulatory support post CPB.	1. Right coronary veins drain into the right atrium, not the coronary sinus so retrograde cardioplegia administration may be poorly distributed to the right ventricle	PRE-EMPTIVE MANAGEMENT: 1. Use other cardioplegia delivery methods with retrograde (i.e. antegrade or coronary ostial administration) to help with distribution of cardioplegia solution and adequate myocardial protection. 2. May not want to use retrograde cardioplegia in children with single ventricle anatomy. REFERENCES: 1. Drinkwater DC Jr, Cushen CK, Laks H, Buckberg GD. The use of combined antegrade-retrograde infusion of blood cardioplegic solution in pediatric patients undergoing heart operations. J Thorac Cardiovasc Surg. 1992; 104(5): 1349-55.	4	1	4	1	16

			2. Hensley FA Jr, Martin DE. A Practical Approach to Cardiac Anesthesia, 2nd Ed. Boston: Little, Brown and Company; 1995. 3. Gravlee GP, Davis RF, Utley JR. Cardiopulmonary Bypass, Principles and Practice. Baltimore: Williams & Wilkins; 1993. 4. Lich BV, Brown DM. The Manual of Clinical Perfusion, 2nd Ed. Fort Myers, FL: Perfusion.com; 2004.					
H13. FAILURE: Failure to monitor and maintain the appropriate fluid balance during cardiopulmonary bypass (CPB) and modified ultrafiltration (MUF). 12/12/15 .	EFFECT: 1.Excessive fluid administration can lead to hemodilution, causing: a.low hematocrit b.low albumin c.low coagulation factor concentration d.pulmonary edema e.unnecessary RBC transfusion. f.portal hypertension in patients with liver disease 2.Excessive fluid removal leading to low pre-load and low cardiac output may make weaning from CPB difficult. 3.If weaning is accomplished, excessive fluid removal may result in post-CPB or post-op hypotension or delay diuresis.	CAUSE: 1.Patient morbidity and likelihood of transfusion are associated with low plasma protein concentration. 2.Hemorrhage and administered fluids decrease both hematocrit and plasma proteins. 3.Fluid used for CPB prime and anesthesia management represents a significant fraction of total blood volume. 4.Infusion of washed, salvaged blood or donor red blood cells raises hematocrit, but further dilutes clotting factors. 5.If dilution is excessive, coagulopathy may ensue. 6.Patients with the smallest blood volumes are at highest risk. 7.Patients with excessive intraoperative fluid balance have more ICU complications and higher hospital mortality. 8.A positive fluid balance in adults of as little as 500 mls (+7 mls/kg in a 70 kg patient) on CPB is	PRE-EMPTIVE MANAGEMENT: 1.Monitor I&O during CPB to measure the net fluid balance. (* If there is no accurate I&O monitoring, the detectability value would be 5, resulting in a RPN of 225.) 2.A computerized spreadsheet with the necessary categories and calculations can be used real-time during CPB and MUF and to give situational awareness to fluid balance (Grist 2011). 3.The fluid balance can be adjusted by adding or removing fluid to achieve a specific goal at the end of CPB/MUF. 4. If pre-op testing indicates a lower than normal COP or albumin, consider adding albumin to the prime. MANAGEMENT: 1.A CPB/MUF fluid balance goal of negative 20 ml/kg should be achievable for most patients. This does not include fluid given by anesthesia pre-CPB. 2.Negative fluid balances can be achieved with slow, continuous ultrafiltration during the CPB time span. 3.Patients with excessive fluid accumulation in the pre-CPB period from CHF, resuscitation or liberal anesthesia rehydration may require additional fluid removal. 4.The need for excessive fluid administration during weaning resulting in a positive fluid balance may indicate myocardial failure in varying degrees or a detrimental change in pulmonary or systemic vascular resistance (McKiernan, 2005). 5.Patients with stiff, hypertrophied ventricles, as seen in Tetralogy of Fallot or left ventricular outflow tract obstruction, may require a positive fluid balance to enhance ventricular preload (Krayenbuehl, 1988, Romand 1995). 6.A zero or negative fluid balance is associated with decreased mortality and implies that there was no need for fluid resuscitation at the termination of CPB/MUF with the exceptions listed in #5. 7.Excess fluid removal (balances of negative 40 mls/kg or greater), even with uncomplicated weaning from CPB, may trigger a	3	5	1*	3	45

		associated with an increased length of stay and the need for blood transfusion in adults (Toraman 2004). 9.A positive fluid balance from excessive crystalloid may mask acute kidney injury (AKI) by diluting creatinine/BUN values after cardiac surgery. However, AKI is not consistently associated with fluid overload. 10.In adults, a positive fluid balance after CPB is associated with higher hospital mortality and is independent of diuretic administration, diuretic response, and type of surgery. 11.Early postoperative fluid overload is independently associated with worse outcomes in pediatric cardiac surgery patients who are 2 weeks to 18 years old. 12. Excessive amounts of low sodium cardioplegia solution may disrupt electrolytes and make appropriate fluid balance harder to achieve.	hypotensive episode in the post-CPB or post-op period (Grist 2011). 8.Before adding crystalloid to compensate for low venous return, evaluate quality parameters (SVO2, cerebral saturation, MAP, base balance, lactate, etc.) to determine if a lower cardiac index can be used for pump flow.					
H 14. FAILURE: acute, iatrogenic	EFFECT: 1.Hypotension 2.Loss of venous return 3.Elevated pump arterial	CAUSE: 1.Dissection incidence is 0.06-0.09 for ascending AO cannulation.	PRE-EMPTIVE MANAGEMENT: 1.The perfusionist should always be in the room during sternotomy. 2.Lower the mean arterial pressure (MAP) during cannulation/ decannulation and application/removal of AO clamps. Lowering	5	1	5	3	75

ascending dissection of the aorta (AO) after initiating cardiopulmonary bypass (CPB). 12/12/15	line pressure 4.Oliguria 5.Dilated pupils 6.BIS (bispectral index) changes 7.EEG changes 8.Cerebral oximeter changes 9.Transcranial Doppler (TCD) changes 10.ECG changes 115.Systemic acidosis 12.AO blue discoloration 13.AO distension 14.Bleeding from needle holes, arterial incisions, and cannulation sites. 15.Intra-luminal blood within split wall of the AO 16.Dissection usually in the direction of flow 17.Loss of pump flow. 18.Acute rupture of the AO with uncontrollable hemorrhage. 19.Death.	2.AO cannulation site most common. 3.Other sites a.AO cross clamp b.Antegrade CP cannula c.Partial occluding AO clamp d.Anastomosis of coronary grafts e.Aortotomy. 4.Dissection due to: a.Direct trauma from cannula insertion or manipulation. b.Indirect trauma from the high velocity jet lifting atheromatous endothelium 5.Risk factors include: a.Patients with an existing aneurysm or a history of one. b. Location of cannulation c. Advanced age (pediatric dissections also occur) d. Chronic hypertension e. Diseased/dilated AO f. Atherosclerosis g. Cystic medial necrosis h. Hypertension during decannulation i. Application/removal of AO clamps j. Cannulation technique. k. Antegrade CP cannulation l. High line back pressure during CP administration.	MAP during cannulation is an anesthesia responsibility. If the pressure is high the perfusionist must exercise due diligence for a dissection possibility. 3.Use extreme care with the insertion of a properly sized AO cannulae 4.Check for pulsatility and correlation of pump arterial line pressure to the MAP. 5.Check for resistance/line pressure with a pump test infusion before CPB 6.Use a narrowly set high pressure servo regulation audible alarm with pump shut off 7.During retrograde arterial auto-priming observe fluid return flow for normalcy. Hesitation or slow return may indicate an arterial cannula tip trapped in the AO wall. 8.Have TEE or epiaortic scanning available to monitor the AO PRN. 9.Evaluate hypotension causes during the initiation of CPB. If there is a loss of volume, ask the surgeon to quickly assess. 10.Do not commit patient to CPB support by quickly cooling, giving cardioplegia or opening the heart until dissection is ruled out. 11.Do not release venous line quickly. Release it slowly while infusing at the same time, keeping the volume in the patient should a dissection occur. If the patient's blood volume drains into the venous reservoir there is no way to quickly return it to the patient if the AO dissects. 12.Continue to visually inspect the AO cannulation site. 13.Patients with a history of aortic aneurysm are usually on beta blockers and always on antihypertensive agents and are safer using a lower MAP. 14.If using a centrifugal pump, do not clamp arterial pump line and quickly release at high RPMs. This increases shear stress by fluid jet and may dissect a weak AO wall. 15.With high risk patients consider: a. having femoral cannula, connectors and extra tubing readily available and in the room. b. having blood PRBC's checked and in the room c. having a hemoconcentrator available d. having extra heparin drawn up and available e. having extra IV fluids on the pump MANAGEMENT:					

			1.When the ascending aorta is the site of the initial cannulation, CPB should be stopped immediately and flow reinitiated via the femoral, subclavian or innominate artery (after assuring that the dissection has not extended into these vessels) or re-cannulate into the true lumen in the AO arch through the flap (perhaps with the aid of ultrasound and a guidewire). 2.With femoral cannulation, asses both forward flow and retrograde flow to confirm that the cannula is not in a false lumen. 3.After CPB is safely established consider the initiation of deep hypothermia in anticipation of need for circulatory arrest to repair the ascending dissection. 4.Use cerebral protection by selective cerebral perfusion if the dissection is extensive and involves the arch vessels. 5.Repair may only require closed exclusion suturing or more complex patch or graft replacement of the ascending AO and arch.					
H 15. FAILURE: Perfusionist related transfusion error on cardiopulmonary bypass (CPB). (12/22/15) .	EFFECT: (not necessarily in order of importance). 1. An error triggers an incident report and possible sentinel event (SE) review. 2.An SE triggers a root cause analysis, a Joint Commission citation, a review of perfusion practices by internal and outside assessors, and potentially a significant monetary penalty. 3.An error may trigger an indefensible civil law suite with significant monetary damages. 4.An error may cause a transfusion reaction: non-hemolytic or hemolytic a.hemoglobinuria b.DIC c.↓BP d.↑HR	CAUSE: 1.Human error. 2.Failure to follow approved hospital transfusion policy and procedure (P&P). 3.Approved hospital or blood bank P&P not suitable for transfusion procedure on CPB. 4. An incompatible blood transfusion leads to a potentially massive activation of the immune and clotting systems causing shock, kidney failure, circulatory collapse, and death. 5.Idiopathic transfusion reaction of unknown cause.	PRE-EMPTIVE MANAGEMENT: 1.Develop specific P&P for transfusion during CPB approved by blood bank medical director or equivalent. 2.Stipulate acceptable variations from general AABB approved transfusion P&P in the perfusion transfusion P&P; key points source*. a.Ensure proper consent obtained. b.Clinical indication documented c.Filter type (Pall, cardiotomy, etc.). d.Speed of transfusion. e.Transfusion line rinse fluid type (Plasmalyte, Normosol, LR, NS, etc). f.Confirm physician's order, product and recipient g.Policy requires double verification of patient identification and product labeling prior to transfusion and confirms that a perfusionist can verify blood products. h. Provide correct storage of blood product in OR before use. i.Vitals monitored and documented by perfusion staff on perfusion record. j.Completed transfusion documented on perfusion record. MANAGEMENT: 1.Call for help if transfusion reaction is suspected. 2.Stop transfusion. 3.Disconnect donor product and IV tubing.	5	2	2	2	60

	e.bronchospasm f.erythema g.urticaria h.fever i.angioedema j.bronchospasm k.pulmonary edema l.shock m.anaphylaxis n.organ failure o.death		4.Examine blood product ID and determine if correct patient. 5.Send remaining product to blood bank. 6.Document incident per institutional policy 7.Maintain intravascular volume. 8.Maintain urine output at least 1-2 mL/kg/h. 9.Prepare for cardiovascular instability: extracorporeal support. 10.Send patient blood and urine sample to laboratory. 11.Consider the following medications: a.Furosemide 0.1 mg/kg b.Mannitol 0.5 grams/kg (2 mL/kg of 25% mannitol) c.Dopamine (2-4 mcg/kg/min) d.Epinephrine 10 mcg/kg IV e.Diphenhydramine 1 mg/kg IV f.Hydrocortisone 2-5 mg/kg *http://www.jointcommission.org/assets/1/6/PBM_Implementation_Guide_20110624.pdf					
H 16. FAILURE: Failure to prevent human fatigue from extended work schedule, lack of sleep, or sleep apnea of personnel operating CPB. (Much of this material comes from the Health Care Worker Fatigue & Patient Safety. The Joint Commission Sentinel Event Alert, Issue 48,	EFFECT: Fatigue from extended shift work, lack of sleep or sleep apnea can lead to: 1.Lapses in attention. 2.Inability to stay focused. 3.Reduced motivation. 4.Compromised problem solving. 5.Confusion. 6.Irritability. 7.Memory lapses. 8.Impaired communication. 9.Slowed or faulty information processing and judgment. 10. Diminished reaction time. 11. Indifference and loss of empathy. 12. A fatigue related minor error was reported	CAUSE: 1.Fatigue can have physical, mental, and/or emotional causes: Physical: Examples – lack of sleep, poor nutrition, dehydration, pain, illness, untreated sleep apnea. Mental: Examples – depression, stress. Emotional: Examples – fear, relationship disturbances. 2. Extended shift length, excessive work schedule or untreated sleep apnea can result in the health care worker being three times more likely to make an error in patient care. 3. Work shifts of 24 hours result in 36% more serious adverse events than shifts	PRE-EMPTIVE MANAGEMENT: 1.Assess perfusion staff schedule for fatigue-related risks. a.Average hours worked per employee. b.Length of shift. c.Number of off-shift hours. d. Number of consecutive work shifts e. Update staffing policies with staff suggestions to minimize potential for fatigue. 2.Any staff exhibiting frequent daytime drowsiness should seek evaluation for sleep apnea. 3.Provide opportunities for staff to express concerns about fatigue; i.e., work place counseling. 4. Give support to staff when appropriate concerns about fatigue are raised and take action to address those concerns. 5.Encourage teamwork as a strategy to support staff working extended hours to protect patients from potential harm: a.Ensure that relief personnel are prompt. b.Patient hand-offs are high risk for errors, especially for fatigued staff. Assess hand-off procedures to ensure patient safety. 6.Perfusionists with sleep apnea should seek treatment since they might experience symptoms of fatigue without an excessive work schedule or mental/emotional cause. MANAGEMENT:	3*	3*	3*	3*	81

December 14, 2011) (4.15.16)	by 66% of perfusionists and 6.7% admitted to a serious accident during CPB (Trew 2011). 13.Patient or perfusionist injury of indeterminate magnitude. 14.Work schedules averaging 45+ hrs/week over 10 years or untreated sleep apnea greatly increase the risk of developing cardiovascular disease (Conway 2016) (Yaggi 2016).	of 16 hours or less. This includes 61% more sharps injuries after the 20th consecutive hour of work and 300% more fatigue-related preventable adverse events that lead to patient death. 4.Work weeks averaging 45+ hrs/week over 10 years greatly increase the risk of developing cardiovascular disease.	1. Implement a fatigue management plan with countermeasures for fighting fatigue: a. Engage in animated conversations with others (not just listening and nodding) b. Engage in physical activity or change body position (even if it is just standing or stretching). c. Utilize sensory stimulation (enhanced lighting, room temperature change, wash hands and face, slow breathing with pursed lips, place cool rag to back of neck). d. Drink fluids to be well hydrated. e. Consume small, high energy snacks. f. Discourage distractions. g. If possible, take short naps (less than 45 minutes). h. Use strategic caffeine consumption. Don't use caffeine if already alert. Avoid caffeine near a sleep period. Perfusionists should only consider the ingestion of caffeine (up to 1000 mg per day) as an alertness-enhancing strategy when the situation offers no other viable alternative. Caffeine should be used judiciously and only when it is truly needed to reduce the impact of fatigue. 2. Maximize fatigue countermeasures effectiveness by using different combinations or sequences of countermeasures. 3.Use a system of independent double checks for critical or complex tasks. 4. Consider fatigue as a possible contributing factor when reviewing all adverse events. *These RPNs are entirely dependent upon individual perfusion program staffing and work schedules. Programs with minimal staffing and longer work hours are going to be riskier than programs with adequate staffing redundancy.					
H 17. FAILURE: Failure to prevent an impaired perfusionist from performing clinical activities. (5/27/16)	EFFECT: 1.The perfusionist is unable to practice with reasonable skill and safety. 2.The potential for a lethal complication is increased when a perfusionist is impaired.	CAUSE: Impairment is most commonly caused by: 1.physical illness 2.mental illness 3.emotional stress 4.loss of motor skills 5.loss of cognitive functioning 6.drug abuse 7.alcohol abuse (Human fatigue, fear and	PRE-EMPTIVE: 1.Each perfusionist should review the organization's policy and procedures (P&P) for identifying the impaired perfusionist. 2. 3.P&P should contain steps to relieve an impaired perfusionist from duty if necessary. 4.P&P should contain a substance abuse and employee assistance program referral process. 5.Maintain situational awareness of staff members for impairment symptoms as follows: A.Immediate physical symptoms	5	*1	*4	3	60

		skills decay can temporarily impair a perfusionist, but they are discussed in separate FMEAs.)	1.In ability to stand or walk normally 2.Red or watery eyes 3.Stuffy, draining nose or excessive sneezing 4.Slow, slurred, garbled or rapid speech 5.Excessive fidgetiness 6.Discolored, pale or red face or skin 7.Altered mental state or demeanor 8.Loss of bowel or bladder control 9.GI disturbances leading to vomiting. 10.Smell of alcohol 11.Overt intoxication 12.Needle marks B.Work-related symptoms: 1.Late to appointments; increased absences; unknown whereabouts 2. Unusual pump set-up times, either very early or very late 3. Increase in OR/surgical staff complaints 4. Increase in secrecy 5. Decrease in quality of care; careless decisions 6.Incorrect or incomplete charting 7. Decrease in productivity or efficiency 8. Increase in conflicts with other perfusionists or OR/surgical staff personnel. 9.Increase in irritability and aggression 10.Failure to respond to “on call” situations 11.Past erratic job history C.Home related symptoms: 1.Withdrawal from family, friends, and community 2. Legal trouble (i.e, DUI, drug or domestic violence arrest) 3. Increase in accidents 4. Increase in medical problems and doctor's visits 5. Increase in aggression, agitation, and overt conflict with family and friends 6.Financial difficulties 7. Deterioration of personal hygiene 8. Emotional disturbances; depression, anxiety, and moodiness D.Institute a system utilizing a secondary perfusionist capable of recognizing and relieving and impaired perfusionist or use specially trained perfusion assistants who can actively seek help and notify management.					

			* This total RPN (60) is based on the presence of secondary personnel participating during CPB. If the perfusionist is working solo without secondary personnel the Occurrence and Detectability RPNs would both be increased by 1 to give a Total RPN of 5*2*5*3 = 150. MANAGEMENT: 1.Carefully document any changes in the suspected impaired perfusionist's behaviors. 2.Avoid any enabling behavior such as frequently covering call or completing work details for the impaired perfusionist. 3.Confront the perfusionist or notify the manager of suspicions. Any confrontation should include resources to aid the impaired perfusionist. 4.Relieve an impaired perfusionist from duty if necessary. There are many laws and regulations pertaining to temporarily removing an employee from duty as a result of impairment. The assistance of Human Resources (HR) should always be sought in these situations. 5.Resuming clinical duty may require a Fitness-for-Duty Certification. http://mrsc.org/getmedia/0EC1F355-A290-484F-8659-ECC9E0404BA8/m58fitness.aspx. 6.Consider other issues a.Loss of confidentiality b.Loss of trust and respect of manager or other perfusionists c.Fear of losing job and license d.Stigma of having a physical, mental, emotional or addictive impairment e.Reluctance of other perfusionists to get involved. 7.If behavior is repeated and the situation warrants it, be prepared to terminate the impaired perfusionist's employment by proper procedure under the direction of HR.					
H 18. FAILURE: Failure to prevent gross blood contamination to the perfusionist's	EFFECT: A.The risk of emotional trauma to the perfusionist has an indeterminate severity. B.The risk of infection to the perfusionist has an indeterminate severity.	CAUSE: Gross blood contamination is always accidental and unexpected. Certain situations are more likely to result in contamination: 1.During tear down and disposal of used perfusion-	PRE-EMPTIVE MANAGEMENT: 1.The emergence of AIDS led to the 1985 CDC recommendation and the 1991 OSHA implementation of universal precautions using PPE for protection from fluid and blood borne pathogens. <https://www.osha.gov/SLTC/etools/eyeandface/employer/requirements.html> 2.Perfusionists should receive ongoing biohazard safety training including PPE.	3*	1	5	3	45

face. (7/14/16)	1. acute risk depends upon: a.the pathogen concentration. b.the perfusionist's resistance based on current health, predisposing diseases, age, sex, and genetic heritage. c.the portal of entry; inhalation, ingestion, mucous membrane, skin or direct inoculation. d.the virulence of the organism. 2.Chronic risk: infection can affect organ systems for extended periods causing death years after exposure. 3.Most severe infection hazards: hepatitis B virus (HBV), and human immunodeficiency virus (HIV). (Biological agents--Refer to OSHA Instruction CPL 2-2.44B: Enforcement Procedures for Occupational Exposure to HBV and HIV.)	related circuits. 2.During emergent repair or component replacement of a perfusion-related circuit. 3.After accidental pressurization of circuit with sudden, uncontrolled release of pressure by unclamping or circuit rupture. 4.Most operating room personal protective equipment (PPE) is inadequate to protect the perfusionist's face against a large splash or projectile spillage originating from the CPB pump.	3.Installation of an eyewash station should be required in any area with high risk for blood and bloody fluid splashes due to perfusion-related procedures or clean-up and disposal of CPB, ECMO, autotransfusion and dialysis circuits being performed in the area. (* If there are no nearby eyewash stations, the Harmfulness RPN should be increased to 5, making the Toal RPN 5x1x5x3 = 75). 4.Eyewash stations should be available within 55 feet of potential accident sites (American National Standards Institute; ANSI). 5.The eyewash station should deliver tepid water (60-100 degrees F) at a rate of 1.5 L/min for 15 minutes. 6.Eyewash stations should be designed to deliver fluid to both eyes simultaneously with hands free. MANAGEMENT: Perfusionists should wash affected areas (face, eyes, nose and mouth) immediately after direct contact with blood or other body fluid. 1.Go to the eyewash station . 2.Push the lever. (An ANSI compliant unit will activate with one single motion, the dust covers will pop off and the flushing water will begin to flow out from the faucet heads. Once activated, the unit will stay on hands free.) 3.Get eyes directly in the stream of the flushing water. 4.Hold eyes open with fingers. (An ANSI compliant unit will be hands free.) 5.Roll eyes. 6.Rinse eyes, mouth, nose and entire face as needed. 7.Flush for fifteen minutes 8.Gently take out contact lenses while flushing. Don't delay the flushing to take lenses out. 9.Afterwards seek medical assistance and document the exposure according to the employer's policies. 10.Post-traumatic stress disorder (PTSD) therapy should be available if needed for the perfusionist or other employees, particularly if the employees experience an adverse outcome like infection or emotional trauma.					
H 19. FAILURE: Failure of checklist use	EFFECT: Indeterminate risk to the patient, ranging from insignificant to lethal, depending on the	CAUSE: 1.Action error: These errors occur when a frequent and routine task becomes a rote,	PRE-EMPTIVE MANAGEMENT: 1.Checklists are executed using a verbal challenge and response format. a.Verbalize task, even if being the sole operator. Verbalizing the task	3	2	2*	3	36

to prevent errors during CPB. (7/14/16)	specific error.	autonomic and habitual action that bypasses the active thought process. These errors also occur if attention is diverted resulting in an incomplete or unintended action. a.Slip: Error of commission failure. b.Lapse: Error of omission failure. 2.Non-compliance error: a.Routine failure: Deliberate deviation from procedures. Attitude: "I like my way better." b.Situational failure: Taking short cuts or failing to properly follow procedure to save time or effort on a particular case. Attitude: "I need to catch up with the surgeon." c.Exceptional failure: A well-meaning, but misguided procedural error on many cases. Attitude: "I am under pressure by my boss to get the job done." 3.Exacerbating factors: a.Fatigue b.Stress c.Hunger d.Illness 4.Perfusion checklists also fail to catch errors due to their poor design, poor wording or being too short or overly long.	helps to stimulate the thought process and inhibit the autonomic response. b.Physically complete task c.Check mark the item on the list only after the task is complete using repeat verbalization. 2.Avoid irrelevant distractions and interruptions when possible. Otherwise complete a specific checklist task before addressing the outside distraction. 3.Ensure sufficient time to complete checklist. 4.Employ a double check confirmation process with secondary personnel. (*Without double check confirmation the Detectability RPN should be increased to 4, making the Total RPN 3*2*4*3 = 72.) 5.Perform time out before cutting the AV loop. a.Describe modifications to circuit and procedure based on surgeon preference. b.Describe any circuit prime lab test results required during priming, reporting any abnormalities. c.Describe any contingency perfusion support preparations anticipated based on the patient condition; blood availability, auto-transfusion, IABP/ ECMO/VAD readiness, secondary personnel availability, etc. d.Be prepared to delay CPB implementation if circumstances warrant. 6.Ensure compliance to checklist procedure by staff: a.Raise awareness of purpose and non-compliance consequences. b.Employ frequent audits and active review to document compliance. c.Revise checklists annually or as necessary with staff input. 7.Either the checklist or the perfusion record should list the type and serial numbers of the equipment used and the type and lot numbers of supplies used. 8.Consider checklist use during the entire peri-operative period; pre-bypass, CPB initiation, CPB termination, post CPB with potential resumption or use of other support methods. MANAGEMENT: 1.If a checklist related failure occurs: a. Trouble shoot the failure as needed. b.Notify Risk Manager after the case of the need to perform a Root Cause Analysis if appropriate. c.Prepare a Failure Mode and Effects Analysis to prevent future incidents, modifying the checklist and its use.					

H20. FAILURE: Failure to reinstitute CPB emergently due to an unexpected need for extracorporeal resuscitation in the immediate post-CPB or post-operative period. (8/15/17)	EFFECT: Failure to re-initiate CPB in a timely fashion can result in: 1. No oxygenated blood being pumped to the patient 2. Hypotension 3. Acidosis 4. Hypercapnea 5. Hypoxia 6. Organ failure 7. Death	CAUSE: 1. Premature takedown of CPB circuit. 2. No backup circuit ready for immediate use. 3. Absence of the perfusionist from the OR in the immediate post-CPB or post-operative period.	PRE-EMPTIVE: 1.Maintain CPB circuit for possible re-institution of emergent CPB: 2.Add heparin the circuit and recirculate AV loop. 3.Rinse ancillary lines with heparinized saline to prevent blood from drying and causing an embolus. 4.Cover AV loop and ancillary lines with sterile sheets if they are removed from the operative field. 5.Ensure perfusion personnel are immediately available in the critical post-CPB and post-operative periods. 6.Do not tear down circuit until patient is delivered to the ICU and evaluated for vital stability. MANAGEMENT: 1.If early CPB circuit tear down is performed, a back-up, assembled circuit on a pump should be immediately available. 2.If the perfusionist leaves the OR, a method of instant communication should be maintained and the perfusionist should be able to return within 3 minutes. *(If the CPB circuit is not maintained for immediate use, the Harmfulness RPN should be increased to 4. If no backup pump and circuit is immediately available, the Harmfulness RPN should be 5.) **(If the perfusionist is absent from the OR or cannot be immediately contacted or cannot return within 3 minutes, the Detectability RPN should be 5.) +(The total RPN for this failure is very low if Pre-Emptive Management is used: 2*1*1*3 = 6. The maximum RPN could be 75 if the recommended precautions are not taken.)	2*	1	1**	3	6+
I. SPECIAL & EMERGENT SITUATIONS								
I1. FAILURE: Various system failures - 1. Disposables 2. Equipment 3. Patient Info 4. Pump 5. Gas Supply 6. Blood Products 7. Lab &	EFFECT: Unknown serious consequences can occur.	CAUSE: The complexity of the equipment and procedures can result in the inadvertent over sight of a vital system preparation and operation. Failure on the part of personnel involved in the procedure to communicate can be a potential cause of failure.	PRE-EMPTIVE MANAGEMENT: There are seven formal steps to perfusion safety. 1. PROCEDURES: These are written instructions for a specific task performed in the safest, most effective manner. 2. SAFETY DEVICES: This is hardware used to prevent injury or accidents. 3. CHECKLISTS: These ensure consistency, completeness and compensate for limits of memory and attention. A checklist is utilized on all CPB cases and checked by two persons. Each of the 15 system failures listed is addressed by one or more items on the checklist. A Perfusion Data Sheet is a subsidiary form of the checklist and is utilized on all CPB cases to calculate the patients' blood flow and	5	5	5	3	375

Support Equipment 8. Circuit Set-up 9. Priming 10. Monitoring 11. Safety Devices 12. Drugs & Fluids 13. Pre Bypass 14. Post Bypass 15. Personnel			heart valve size. Preoperative lab values are also listed and the doses of drugs commonly given during CPB are automatically calculated. Data from this sheet is written on a dry erase board by the perfusion staff in the OR to inform all of the participants of the details of the operative procedure. Blood product availability is confirmed by direct communication with the Transfusion Services Laboratory before the procedure and then listed on the dry erase board. Additional details from the ECHO cardiogram, cardiac catheterization and surgeon's cardiac conference notes are added as well. The operative procedure is listed on the dry erase board and confirmed by the surgeon when s/he enters the room. 4. TROUBLE SHOOTING: This is problem solving for failures as they occur. Immediate assistance from secondary personnel (perfusion assistants or clinical perfusionists) is always available for consultation, to obtain equipment/circuit components and assist in emergency procedures. 5. ROOT CAUSE ANALYSIS (RCA): This identifies the cause of a serious failure and proposes actions and conditions that could have prevented the failure (Gritten Report http://www.scps.org.uk/pdfs/GrittenReport.pdf). This is a formal procedure carried out by hospital risk managers or outside assessors. 6. FAILURE MODE EFFECTS ANALYSIS (FMEA): This examines how a system can fail before the failure occurs. Development of this FMEA can assist in addressing system failures if pre-emptive management is unsuccessful. 7. DOCUMENTED COMPETENCY: Competency is the ability of personnel to apply their skill, knowledge, and experience to perform their duties correctly. Competency assessment is used to ensure that personnel are fulfilling their duties as required by the appropriate authority. Only qualified perfusionists are hired and their competency is assessed and documented by annual evaluations, frequent case reviews by their peers and annual re-certification requirements which include continuing education and documentation of cases performed.					
I2. FAILURE: Failure to estimate collateral blood flow, aortic	EFFECT: 1. Most congenital heart patients have collateral blood vessels that empty into the left heart with a risk of cardiac distention	CAUSE: The amount of blood removed from the heart by the left ventricular vent is a measure of blood which is returning to the heart and	MANAGEMENT: In instances of excessive vent flow, the perfusionist should overflow the calculated blood flow by an amount equal to the vent flow. The surgeon may also want to know the vent flow in order to assess the function of the aortic valve or assess any reduction in collateral flow after ablation of visible collateral vessels. After adjusting the vent	3	3	4	1	36

insufficiency and left ventricular vent blood flow leading to under perfusion	and damage. 2. Aortic valve insufficiency can also distend the ventricle.	bypassing the systemic vascular system during CPB. This may indicate a reduction in the effective arterial systemic blood flow of as much as 50% when the CPB is at the calculated flow. Blood regurgitated through an insufficient aortic valve into the left ventricle and removed by the vent also steals flow from the systemic circulation. In large patients with aortic insufficiency, large vent tubing should be used in anticipation of a high regurgitation rate of as much as 50% of the calculated blood flow. In biventricular patients, a reduction in the effective arterial systemic blood flow is indicated by a drop in the SVO2. However, collateral blood flow is fully oxygenated, having just come from the oxygenator. In patients with a common atrium or common ventricle, the collateral or regurgitated flow may mix with the systemic venous return. As this drains into the venous line of the pump, the SVO2 may appear as a normal or even elevated above normal, the true SVO2, which is	pump flow indicator to the correct tubing size of the vent, the vent flow can be estimated in two ways. 1. Watching the entry point of the vent tubing to the cardiotomy reservoir, adjust the vent pump flow until the blood begins to 'back suck' with each half revolution of the pump. Back suck appears when air within the cardiotomy reservoir moves backwards towards the pump head. This indicates that the pump is removing the maximum amount of blood provided to it by the ventricular vent cannula. The pump flow indicator would then be equivalent to the vent blood flow. 2. Watching the entry point of the vent tubing to the cardiotomy reservoir, adjust the vent pump flow until the blood and air interface within the tubing appears to be about 50/50; that is, a two inch column of blood is followed by a two inch column of air followed by a two inch column of blood, etc. The air within the vent tubing should be coming from the vent relief valve. This also indicates that the pump is removing the maximum amount of blood flow provided to it by the ventricular vent cannula. The pump flow indicator should be divided by two to get the true vent blood flow since half of the flow indicated is air. Watching the exit point at the “diamond” located in the ventricular vent line is also indicative of the vent flow arising from the heart. The diamond is a piece that is added to the ventricular vent line to accommodate increased blood flow from the heart. The diamond is constructed with a one-way valve in parallel with a ¼” piece of tubing located in the ventricular vent line. Flow through the diamond can be directed by clamp placement. If little return is coming through the ventricular vent line then the clamp may be placed on the ¼” side of the diamond. However, if a large amount of return is coming from the ventricular vent line, then a tubing clamp maybe placed above the one-way valve, which allows blood to travel solely through the ¼” tubing side of the diamond. In this configuration, the generation of excessive negative pressure will open the one-way valve’s relief mechanism allowing the entrainment of air into the ventricular vent sucker line. The estimation of ventricular vent flow maybe assessed by decreasing the pump flow until zero air is being entrained by the one-way valve relief mechanism. The digital readout of the ventricular vent pump will directly correlate to the ventricular vent pump flow. Additionally, the 50/50 method maybe used in this exact configuration. When using the 50/50 method, increase the ventricular vent pump until there is 50% blood to 50%					

		abnormally low, being masked. During this time period, the first sign of under perfusion may be a reduction in the base deficit and/or an increased lactate. There are three causes of arterial systemic blood return to the heart. 1. Patients with congenital heart disease may have developed aortic to pulmonary (AP) collaterals. If so, blood flow will shunt from the aorta to the pulmonary system and return to the heart without entering the systemic capillary bed. This may effectively reduce the arterial systemic blood flow, particularly if the heart is cross clamped or not beating effectively. 2. In patients with aortic valve insufficiency, some of the CPB blood flow will regurgitate into the heart and be removed by the left ventricular vent. Should this occur, the arterial systemic blood flow will be effectively reduced. Once the heart is cross clamped, the vent flow should be greatly diminished. 3. Patients may have both AP collaterals and aortic insuffici ency.	air. The flow arising from the heart is 50% of the digital ventricular pump readout.					

I3. FAILURE: Failure to recognize the presence of major aortic to pulmonary collateral arteries (MAPCAs). (4.15.16)	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 hypoxic cardiac lesions and naturally develop 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, hypoplasic right and left hearts, univentricular anatomy or Tetrology 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. Patients at increased risk of MAPCAs: 1. Adults with congenital heart disease are at elevated risk of having MAPCAs even if the lesion was repaired in childhood. 2. Fontan completion patients, particularly those older than two years and those who have failed a	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. 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 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 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 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	5	3	4*	1	60

		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 cannulation may cause poor perfusion above the diaphragm.	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. 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. 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: 1. Maximize blood flow estimating 1.5 times the normal calculated blood flow. 2. Maximize ventricular vent flow (if vent is used) and calculate MAPCA flow to the lungs by measuring vent flow. 3. Maximize sweep gas FiO2. 4. Maximize hematocrit till base deficit subsides or until hematocrit reaches 35%. 5. 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 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.					
I4. FAILURE: Hemodynamic instability due to the presence of residual muscular ventricular septal defects (VSD's) during arteriovenous modified ultrafiltration (MUF).	EFFECT: Residual VSDs can result in right ventricular overload causing hemodynamic instability.	CAUSE: The presence of residual (usually muscular) VSDs after a biventricular repair may result in acute right heart failure during MUF. During MUF, blood is aspirated from the ascending aorta via the CPB arterial line, pumped through a hemoconcentrator (HC) and returned to the patient's right atrium via a venous cannula. In effect this causes a L > R shunt that increases the workload of the right heart. Normally, this is negated by the reduction in pulmonary vascular resistance caused by excess water removal from the lungs by the hyperoncotic MUF blood passing through the pulmonary vessels. The beneficial effects are manifested by a decreased RA pressure and increased systemic blood pressure. However, additional L > R shunting from residual VSD's may critically overload the right heart, resulting in an	MANAGEMENT: 1. If possible, identify the presence of residual VSD's preoperatively or during post-CPB echocardiogram. 2. Initially, limit MUF blood flow to < 20% of calculated cardiac output, with half of the MUF flow being compensated by infusion from the CPB circuit. 3. Watch for appropriate response to MUF in the form of lower RA pressure and increasing systemic blood pressure. 4. Slow or stop MUF if hemodynamics failure to improve.	3	1	4	1	12

		increase in RA pressure due to failure of the RV, a reduction in pulmonary blood flow, reduced LA pressure and corresponding reduction in LV output. Patients at increased risk of right heart failure during MUF include infants with biventricular anatomy or repair with residual VSD's and whose effective MUF blood flow comprises a significant portion (>20%) of their calculated cardiac output.						
I5. FAILURE: Inadequately prepared donor PRBC for infants less than 4 months of age for perioperative transfusion and pump prime.	EFFECT: Improperly prepared PRBC can increase the risks for febrile non-hemolytic transfusion reactions (FNHTR), viral infection, hyperosmotic exposure to kidneys and the brain and transfusion associated graft vs host disease (TA-GVHD). Although rare, TA-GVHD usually occurs 10-14 days post transfusion with clinical features of fever, skin rash, hepatitis, diarrhea and pancytopenia. It is fatal in more than 90% of cases.	CAUSE: Transfusion Services Laboratory guidelines require that PRBC units for infants less than 4 months old receive special treatment prior to transfusion. The entire blood preparation process requires at least 45 minutes and often longer. In the acute setting of open heart surgery and the immediate post-operative period, obtaining PRBC units rapidly can be of great importance. 1. Each unit is leukocyte depleted to: a. minimize FNHTR. b. prevent of transmission of viral infections. 2. Each unit is spun down and the excess plasma and	MANAGEMENT: During the preparation for open heart surgery on an infant less than 4 months old, the perfusionist will - 1. Inquire of the CMH TSL the number of PRBC units on hand. 2. Two units will be ordered for preparation; 1 for pump prime and a 2nd unit for storage in the OR refrigerator. Both these units will undergo the preparation previously described. 3. During the course of the surgery, if the 2nd unit of PRBC is removed from the OR refrigerator either by Perfusion Services or Anesthesia, Perfusion Services will call the CMH TSL and order another PRBC unit to be prepared immediately and brought to the OR refrigerator as soon as possible. 4. Any PRBC unit with the preservative expressed will expire within 24 hours under refrigeration. This means that a 3rd unit may expire unused before its normal outdate. Regardless of this, the PRBC unit needs to be prepared for possible emergent transfusion in the immediate post-operative period.	3	2	1	2	12

		preservative (usually AS-1) is removed due to the hyperosmotic exposure from dextrose, adenine and mannitol. A neonate receiving large volumes of hyperosmotic fluid, such as from a pump prime, is at risk for kidney and brain damage. 3. Finally, the PRBC unit is irradiated to reduce TA-GVHD which occurs when donor lymphocytes from transfused blood engraft in the recipient and cause disease.						
I6. FAILURE: Hemodynamic instability due to the presence of hypertrophic cardiomyopathy (HCM), idiopathic hypertrophic subaortic stenosis (IHSS) and its variants. 12/12/15	EFFECT: 1.Hemodynamic instability 2. Iatrogenic myocardial damage 3. Failure to wean from CPB 4. The need for extended ventricular support 5. Death	CAUSE: 1.HCM is a form of congenital cardiomyopathy; a condition in which the heart ventricular muscle becomes abnormally thickened without a compensating increase in perfused capillary density (PCD). 2.The thickening makes it harder for the heart to work and the reduced PCD impairs oxygen distribution to the myocytes. 3.HCM causes the size of the ventricular chamber to shrink. So the heart must work harder to pump a normal amount of blood per minute because there is a smaller stroke volume. 4.The thickening of the	PRE-EMPTIVE MANAGEMENT: Do not attempt surgery on these patients without the availability of adequate mechanical ventricular support and necessary back-up personnel. 1.Myocardial protection is difficult with HCM. 2.Management on CPB involves maximizing oxygen delivery to the heart, optimizing the electrolytes and maximizing myocardial protection during cross clamping. 3.Weaning from CPB may prove difficult. 4.External defibrillation pads should be attached prior to prep and drape. 5.Hypotension during anesthesia induction can reduce coronary artery perfusion and cause fibrillation. 6. Consideration should be given to using a blood prime to prevent excessive hemodilution. 7.Use bi-caval cannulation to reduce warm blood return to the heart. 8. Increase coronary perfusion pressure to at least over 40 mmHg before and after cross clamping. 9. Expect to give cardioplegia at a greater frequency and possibly in larger amounts. 10. This may require hemoconcentration to reduce hemodilution from cardioplegia crystalloid. 11. Patients with a secondary diagnosis of coronary artery disease, even if it is not clinically significant, may benefit from frequent	5	2	4	1	40

		heart muscle may, at times, completely block the normal flow of blood out of the heart. 5.This is called IHSS and is a variant of HCM that involves the thickening of the interventricular septum. 6. HCM may also make it harder for the heart valves to work by obstructing their function. 7.The condition is seen in people of all ages. 8.Younger people are likely to have a more severe form of HCM. 9. In people over age 60, HCM is often associated with mild hypertension. 10.Patients with this heart condition have extremely fragile myocardium that must be carefully protected. 11.Despite the hearty appearance of the thickened myocardium, these hearts are quite fragile and do not tolerate periods of hypotension from general anesthesia or ischemia such as during aortic cross clamping with cardioplegia. 12.After cross clamping, the excessive bulk of the myocardium makes it difficult for the cold cardioplegia to cool the heart effectively. 13. The ambient	retrograde CP administration. 12. The protective effect of single dose CP solutions like HTK in HCM patients is unknown. 13. If cooling the patient, delay cross clamping until the target temperature is reached to aid in cooling the myocardium. 14. Prior to cross-clamp removal, increase the K+ to 4.5 mEq/L and the iCa to 1.4 mmoles/L. 15. Use 100% oxygen in the oxygenator sweep gas to maximize capillary oxygen distribution vectors in the myocardium particularly if the hemoglobin is low. 16. Increase the hematocrit to 40% before termination of CPB. This will require the availability of extra PRBC units, particularly in the larger patient. 17. Anticipate the need for higher than normal ventricular filling pressures; =/> CVP 15 mmHg. 18. Be prepared for ventricular support in the form of an intra-aortic balloon pump, VAD/biVAD or ECMO. MANAGEMENT: 1. Failure to wean from CPB will necessitate ventricular support for an indeterminate period. 2. If patient weans from CPB and transits to the ICU, be prepared for extracorporeal support (ECPR) should patient have sudden cardiac arrest in the post-op period.					

		temperature within the chest and relatively warmer venous blood returning to the heart and any pulmonary collaterals add to the difficulty in keeping the myocardium cool.						
I7. FAILURE: Failure to wean from CPB due to acute pulmonary hypertension (APH) crisis. (4/15/16)	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 surgery and in 20-30% of patients receiving an LVAD. (* These VAD patients would have an Occurrence RPN = 4.) The presence of RV failure after CPB has been associated with a mortality of 44% to 86%. 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,	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: a.have inhaled nitric oxide (iNO) readily available b. Consider steroid administration to attenuate inflammatory response. c.have ECLS readily available 3.If APH is suspected, prior to weaning: a.Hyperventilate with sweep gas and ventilator. b.Consider additional alkalization with NaHCO3 prior to weaning. c.Administer 100% oxygen by ventilator and sweep gas. d. Consider high frequency ventilation. e.Check venous blood gas for elevated pvCO2 before and during weaning. f. For COPD patient, consider return to base line blood gas even if abnormal. g.Provide additional preload fluid. 4. Perform arteriovenous modified ultrafiltration to target the pulmonary capillary bed and remove pulmonary edema. 5.Consider NIRS monitoring, especially if patient has carotid artery disease.. 6.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	5	1*	3**	3	45

		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.	b. administer narcotic 2.Repair PPM if present. 3.Consider pulmonary vasodilators: a.iNO b.Milrinone c.Nitroglycerine d.Nitroprusside e.Prostaglandin f.Prostacyclin 4.For COPD patient, consider return to base line blood gas even if abnormal. 5.Utilize inotropes sparingly to prevent excessive systemic vasoconstriction that could limit blood flow if ECLS is required: a.dopamine b.dobutamine c.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.					
I8. FAILURE: The failure to properly transfer a patient on extracorporeal membrane oxygenation (ECMO) to cardio-pulmonary bypass (CPB) in preparation for a surgical procedure with possible return to ECMO.	EFFECT: 1. Hemodynamic instability 2. Loss of perfusion 3. Inadequate provision for utilization of all of the components of the CPB pump to perform the surgical procedure.	CAUSE: 1. Poor planning. 2. Communication failure between perfusionist and surgical personnel. 3. Lack of written procedure.	PRE-EMPTIVE MANAGEMENT: Preparation of the modified CPB circuit prior to priming: 1. The CPB circuit is fully assembled in the usual manner. 2. A second arteriovenous loop (AV loop) is connected to the first AV loop as follows, see Fig. 1: 3. Insert a "Y" connector just below the cardioplegia blood takeoff line on the first AV loop. 4. Connect the arterial line of a second AV loop to the "Y" connector. 5. Insert a second "Y" connector just above the venous sample port on the first AV loop. 6. Connect the venous line of a second AV loop to the "Y" connector. 7. Secure the connections as necessary with tie straps. 8. Prime the CPB in the usual fashion with crystalloid and blood, if necessary, ensuring that both AV loops are completely filled and any bubbles removed. MANAGEMENT: 1. The ECMO pump and the CPB pump should each have a dedicated	3	1	5	1	15

			perfusionist. 2. After the patient is placed on the OR table and is being prepared for surgery, connect AV loop #1 to the ECMO blood lines using all appropriate clamping and connector procedures. This should be performed before the sterile drapes are applied. 3. The blood lines should be kept long enough to secure them to the OR table and position them in such a way as to provide unimpeded access to the table by the surgeon or other surgical personnel. 4. This process should be performed if the patient has a neck cannulation, chest cannulation or femoral cannulation. 5. The CPB pump should then takeover the function of the ECMO pump. 6. Extreme care needs to be taken assure the proper placement of clamps on both sets of arterial and venous lines throughout the procedure to prevent the inadvertent misdirection of blood flow. 7. The ECMO pump lines should be connected and the circuit recirculated and fully heparinzed should the ECMO pump be needed later. 8. For transferring the patient from the CPB pump to the ECMO pump. the ECMO pump and the CPB pump should each have a dedicated perfusionist. 9. After the procedure is completed and the patient needs to be placed back on the ECMO pump the determination should be made as to which set of arterial and venous lines needs to be reattached to the ECMO pump. This depends on which cannulae the surgeon wishes to utilize for ECMO. 10. Discussion and agreement between the surgeon, the CPB perfusionist and the ECMO perfusionist should confirm what lines need to be transferred.					
I9. FAILURE: Left heart bypass using the open heart pump.	EFFECT: Certain procedures can result in hemodynamic instability unless the left heart is bypassed while the right heart and lungs continue to function normally.	CAUSE: Some procedures on the distal aorta require that blood flow distal to the operative site be stopped by vascular clamp. During this time the open heart pump can be utilized to provide perfusion to the spinal cord and abdominal organs. During bypass, perfusion is	MANAGEMENT: Arterial cannulation is normally placed in the femoral artery, but it can also be placed in the descending aorta distal to the operative site. Venous cannulation is normally placed in the right atrium via the femoral vein, but it can also be made directly in the right or left atria. 1. CPB flow should be initiated at approximately 1/2 of the calculated blood flow. The heart should NOT be drained of blood. 2. Venous return should be restricted by the perfusionist to prevent exsanguination of the right heart. The right and left heart must continue to function to provide perfusion to the arms, head and brain. 3. Normal ventilation should be maintained by anesthesia.	3	1	1	1	3

		maintained to the head arteries by the patient's own pulmonary and cardiac function. While perfusion to the abdomen and spinal cord is provided by the open heart pump.	4. Normothermia will be maintained unless directed by the surgeon. 5. Arterial pressure monitoring should include a right radial artery and femoral or leg arterial pressure lines. 6. NIRS monitoring should be utilized on the head and flank. 7. Perfusion and discontinuation of bypass should be done in such as way as to prevent hypotension in the head and arms.					
I10. FAILURE: Inadequate cerebral perfusion indicated by cerebral oximetry (NIRS monitor)	EFFECT: 1. Greater than 20% drop from baseline or a decline to less than 50%. 2. Cerebral hypoxia and subsequent brain damage.	CAUSE: 1. Improper aortic cannula placement. 2. Inadequate perfusion pressure. 3. Inadequate pump blood flow 4. Low paO2 5. CO2 imbalance 6. Inadequate anesthesia 7. Hemodilution	MANAGEMENT: 1. Check head & cannula position. 2. Increase the mean arterial pressure. 3. Increase pump flow rate. 4. Increase systemic oxygenation. 5. Increase PaCO2 > 45 mmHg. 6. Increase volatile anesthetic depth or administer propofol bolus. 7. Increase hematocrit by ultrafiltration 8. Consider hypothermia. 9. Consider PRBC transfusion for Hct less than 23%.	4	3	1	3	36
I11. FAILURE: Too large of a blood to air interface	EFFECT: Systemic inflammatory response syndrome (SIRS) activated.	CAUSE: 1. Too large of CPB circuit used. 2. Open reservoir design with excessive ventricular vent/field sucker blood flow.	PRE-EMPTIVE MANAGEMENT: 1. Four different circuit sizes, based on the patients' body surface area, are utilized to minimize surface area exposure. 2. Steriods are added to the prime on all cases to reduce SIRS response. 3. Blood prime is washed to remove unwanted metabolites, excess glucose, excess K+ and harmful preservatives.	2	5	1	3	30
I12. FAILURE: Divergent practice by multiple perfusionists with loss of consistency in performance and outcomes.	EFFECT: Different perfusionists have different outcomes in morbidity and mortality.	CAUSE: 1.Management failure to compel perfusionists to perform in a uniform manner consistent with past evaluation of morbidity and mortality outcomes. 2.Lack of case management reviews leads to variation in perfusion practice. 3. Failure to review standardized procedures at least annually can result in	PRE-EMPTIVE MANAGEMENT: Perfusion quality control is a comprehensive team process that establishes and maintains a high standard of consistent perfusion practice that strives to ensure high quality patient care. Perfusion quality control is divided into 4 main components: 1. Morbidity and mortality discussion. 2. Outcomes review. 3. Cardiopulmonary Bypass Case Management Review 4. Yearly review and update of procedures. Deviation from accepted procedures by individual perfusionists is not accepted without a thorough evaluation and adoption by the entire perfusion team.	2	3	1	3	18

		the individual modifications. 4.Self-centered diversity of practice subverts routinization of technique and appropriate instinctual actions in emergent situations.						
I 13. FAILURE: Failure of communication between the perfusionist and other team members. (7/14/16).	EFFECT: Indeterminate effect ranging from insignificant to lethal.	CAUSE: 1.Lack of formalized communication techniques. 2.Ineffective timeout tool that does not include role identification of team members (who is responsible for what, particularly on teams with frequent member changes). 3.Differences in educational level, training and experience among team members. 4.Disruptive or distracting behaviors. 5.Tense emotional climate or the presence of conflict between team members, i.e. fear of rebuke or ridicule. 6.Pre-occupation with non-relevant matters. 7.Institutional or leadership impediments to implementation of formal communication training, particularly for new team members. 8.Poor operating room ergonomics; line of sight, lighting, acoustics, noise level, temperature	PRE-EMPTIVE: 1.Implement checklists (perfusion and surgical). 2.Utilize effective time out tools prior to each critical process: examples, pre-incision, pre-CPB, CPB termination, moving patient for transport. 3. Verbal communication of patient and procedural information between individual team members should include an affirmative verbal, repeat response. 4.Formal handoff tools should be implemented during transfer of patients between providers. 5.Implement training involving all team members to improve structured communication, situational awareness and leadership. 6.Conduct event scenario training (i.e. FMEA discussion) for non-routine events on a regular basis that includes all team members. 7.Conduct routine simulation exercises for all team members if practical. 8.Conduct prospective studies of teamwork and communication that investigate optimal communication models. MANAGEMENT: 1.There is no management technique which can mitigate a communication failure other than immediately addressing the consequence of the failure. 2.Following a miscommunication involving patient harm or potential harm, root cause analysis should be conducted and an FMEA developed. 3.Post-traumatic stress disorder (PTSD) therapy should be available if needed for perfusionists or other employees, particularly if the patient experiences an adverse outcome.	5	5	5	3	375

		variations, equipment positioning, etc.						
I 14. FAILURE: Failure to take proper precautions for the sickle cell disease (SCD), sickle cell trait (SCT) or thalassemia patient undergoing CPB to prevent post-op sickle cell crisis. 12/12/15	EFFECT: 1.The presence of a significant fraction of hemoglobin S (Hgb S) before CPB poses an increased risk for: a. Hemolysis b.Sickling c. Capillary obstruction d. Shortened RBC life e. Reduced oxygen-carrying capacity. 2. Common postoperative complications include: a. Hemorrhage b. Stroke c. Renal failure d. Death.	CAUSE: 1.SCD/SCT patients who require cardiac surgery are at risk of a potentially fatal sickling crisis, which may be induced by hypothermia, hypoxia, acidosis, or low-flow states. 2.Hemoglobinopathies (mainly sickle cell anemia and thalassemia) are autosomal-recessive inherited disorders. 3.Approximately 5% of the whole world population carries a potentially pathological gene. 4.Erythrocytes containing high amounts of Hgb S undergo multiple sickling and de-sickling events, eventually resulting in hemolysis and anemia. 5.These deformed cells have an increased tendency to adhere to the vascular endothelium, leading to occlusion of small vessels and causing organ damage. 6. Anticipate pulmonary complications upon weaning due to sickling of residual Hgb S RBCs within the pulmonary vasculature that was under perfused during CPB. 7.There is no consensus on absolute safe values of Hgb	PRE-EMPTIVE MANAGEMENT: 1.Consider the early initiation of hydroxyurea, erythropoietin, folic acid, pentoxifylline and oral antibiotic administration in workup prior to non-emergent surgery as part of a comprehensive perioperative blood management program and infection prevention. 2.Modify the routine perioperative management strategies for SCD/SCT cardiac surgery patients: a. Keep warm in cool OR environment. b. Sedate ASAP to avoid anxiety and stress. c. Hydrate ASAP with IV solution to make-up for NPO deficit. d. Provide supplemental O2 ASAP. 3.CPB setup should be designed to perform exchange transfusion at the initiation of CPB. 4.If practical, the CPB circuit volume should be at least equal to the patient's blood volume plus the circuit prime volume in known SCD patients to facilitate exchange transfusion. This may be difficult to accomplish in a large patient with upwards of 3 L blood volume. 5.The goal is to reduce Hgb S levels to below 30%. 6.Prime the pump with donor RBC and plasma in the volume necessary to replace as much of the patient's own blood volume as is practical. Add enough heparin to the prime to make up for the heparin removed in the exchange transfusion. 7.The blood prime should be normalized as much as possible for pH, HCO3, base balance, Na+, K+ and glucose. 8.Upon the initiation of CPB, collect the patient's venous return blood into a collection bag system or separate cardiotomy reservoir. 9.Salvage the plasma from the collected patient blood using intraoperative plasmapheresis by an autotransfusion device. 10.Transfuse the salvaged plasma into the pump and hemoconcentrate it to an appropriate volume. 11.Discard the collected patient RBCs. 12.Maintain 100% oxygen sweep gas FiO2. 13.Normalize the both the arterial and venous pH. 14.Minimize the need for hypothermia as much as possible. 15. Consider diuresis to treat potential excessive hemolysis. 16.Monitor for Hgb S as needed. MANAGEMENT:	4	1	4	1	16

		S in patients undergoing surgery. 8. Preoperative Hgb S may be as high as 45%. 9.The literature suggests that the level of Hgb S should be reduced to <30% for major surgical procedures or even to 5% for cardiac surgery before or at the time of surgery. 10.The classic precipitating factors for sickling include: a. Stress b. Exposure to cold c. Dehydration d. Infections e. Hypoxia f. Inflammatory cascades g. Acidosis. 11.SCD/SCT is frequently seen among Africans, Afro-Caribbeans, east Indians, and the Middle East and Southern Europe Mediterranean populations. 12.SCD/SCT patients requiring CPB are rare; between 0.29% and 0.41%, or 1 out of every 300-400 CPB patients.	If CPB is to be initiated without exchange transfusion: 1. Hemodilute the patient to the hematocrit of 25%. 2. Immediately begin exchange transfusion. 3. Maintain 100% oxygen sweep gas FiO2. 4. Normalize the both the arterial and venous pH. 8. Minimize the need for hypothermia as much as possible. 9. Monitor for Hgb S as needed.					
I 15. FAILURE: Failure to recognize Raynaud's phenomenon in a patient and implement	Raynaud's phenomenon causes arterial vasospasm of the digital arteries, often precipitated by cold stress. There may also be vasospasm in internal organs, particularly the brain, during or after CPB.	Raynaud's phenomenon is a vascular disease occurring in 3%–5% of the general population. There are two types: "Raynaud's disease" which is idiopathic and "Raynaud's syndrome" which has a stressful trigger	MANAGEMENT: Cerebral NIRS monitoring should be used for patients with Raynaud's phenomenon undergoing cardiac surgery. 1. During CPB a precipitous decrease in the bilateral rSo2 (NIRS values) may be seen. 2. Pulse oximetry waveform may be unobtainable after pulsatility returns, even on different digits. 3. If Raynaud's is suspected, a single dose of 40-µg nitroglycerin should be given intravenously to treat the vasospasm. If successful,	3	1	4	1	12

early treatment.	Vasospasm in the digits can lead to necrosis and gangrene. In the brain it can lead to hypoxia/anoxia by NIRS measurement.	factor. Attacks are characterized by episodes of vasospasm primarily affecting the digits. These attacks are usually triggered by cold temperatures or a stressful situation. The fingers and less often the toes are affected, but the ear lobes, lips, nose, and nipples may also be involved. Although controversial, vasospasm affecting the central nervous system or other vital organs has been suggested. One study found that individuals with Raynaud's syndrome have a more than twofold increased frequency of heart disease and an almost threefold increase in stroke. CPB using temperature variations has the potential to precipitate Raynaud's attacks. CNS vasoconstriction might accompany peripheral vasoconstriction during CPB as a result of core temperature changes or vasopressor use. Raynaud's may include the following: 1. Platelet activation - Present in both Raynaud's disease and syndrome, this leads to increased levels of the vasoconstrictors thromboxane and serotonin	NIRS will improve and pulse oximetery will return. 4. Begin a nitroglycerin infusion to maintain adequate NIRS levels. The use of milrinone is not suggested as a therapeutic treatment option for Raynaud's phenomenon.					

		2. Defective fibrinolysis - Found primarily in syndrome patients, this can lead to fibrin deposition and obstruction of vasculature 3. WBC activation - Found in both Raynaud's phenomenon, this can lead to damage from oxidative stress 4. Decreased RBC deformability - Found in the syndrome, this can lead to impairment of blood flow. Increased blood viscosity - Found in both, this can lead to impaired blood flow. 5. Oxidative stress - Triggered by ischemic episodes; decreased levels of antioxidants is found in both types.						
I 16. FAILURE: Failure to recognize antiphospholipid antibodies (APA) that can lead to antiphospholipid syndrome (APS), an autoimmune disease, which causes varying degrees of clotting to occur in the	EFFECT: 1. APS is defined by blood vessel thrombosis occurring in the presence of APA. 2. Induction of a transient hypercoagulability state despite ongoing anticoagulant therapy. 3. Catastrophic exacerbation of APS. 4. Bleeding complications in the perioperative period due to excessive anticoagulation and/or thrombocytopenia. 5. Greater than normal	CAUSE: 1. Surgery increases the risk of thrombosis from APS that can precipitate varying degrees of clotting due to withdrawal of oral anticoagulants. 2. An autoimmune disease, APS can occur as an isolated condition or can be associated with connective tissue diseases, such as systemic lupus erythematosus (SLE). 3. APS is thought to occur in 1-5% of asymptomatic patients.	PRE-EMPTIVE MANAGEMENT: 1.Antiphospholipid antibodies are detected by functional coagulation assay: the lupus anticoagulant (LAC) and/or by solid phase assays: anti-cardiolipin (aCL), or anti-β2 glycoprotein I (anti-β2GPI) antibody tests. *In the absence of obvious autoimmune disease these tests may not be performed making APS difficult to detect before thrombosis occurs; a Detectability RPN of 5 resulting in a total RPN of 50. 2.There is no consensus regarding the optimal perioperative management of anticoagulation in APS. However keeping to an absolute minimum the time periods without anticoagulation is recommended. 3.Patients with APS are at increased risk for thrombosis and adequate anticoagulation is of vital importance during cardiopulmonary bypass (CPB). 4.A successful outcome requires multidisciplinary management in order to prevent thrombotic or bleeding complications and to manage	5	1	1*	2	10

perioperative period of cardiopulmonary bypass (CPB). 2/4/16	morbidity due to clotting or bleeding in the post-CPB period. 4. Cardiac problems associated with APS include a. heart valve disease, valvular thickening, dysfunction and vegetation The mitral valve is the most often involved. b. coronary thrombosis c. ventricular hypertrophy or dysfunction d. intracardiac thrombi e. pulmonary hypertension. 5. Estimates of APS morbidity and mortality associated with cardiopulmonary bypass vary, but have been reported as high as 84% morbidity (postoperative thrombosis or bleeding) and 63% mortality. 6. Individual case reports of cardiac surgical patients often describe thrombotic or bleeding complications including early graft occlusion, hemothorax, pulmonary emboli, and limb ischemia. 7. Death due to complications of clotting or bleeding.	7. Minor alterations in anticoagulant therapy, infection, or a surgical insult may trigger widespread thrombosis. 8. Deep hypothermic circulatory arrest during cardiac surgery increases the risk due to the combination of blood stasis and changed enzymatic activity associated with the temperature changes. 9. The outer surface of the red cell membrane is composed of electrically neutral phosphorylcholine. APA reacts to the negatively charged phosphatidylserine phospholipids (PPs) located on the inside of the red cell membrane. PPs are exposed when the red cell hemolyzes. APA patients have varying degrees of clot because of the varying degrees of natural hemolysis in the body.	perioperative anticoagulation. MANAGEMENT: 1. APS often interferes with in vitro tests of hemostasis by impeding the binding of coagulation proteins to phospholipid surfaces, especially during CPB when blood contacts the extracorporeal surfaces and the coagulation cascade is stimulated. 2. To prevent clotting, unfractionated heparin is administered before CPB. Heparin concentrations of greater than/equal to 3 u/ml ± 1 are generally accepted as therapeutic for CPB , but individual patient responses to a standardized heparin dose vary. 3. Heparin activity is assessed using the activated clotting time (ACT) which is a phospholipid dependent test. The ACT may be prolonged by the APA. In the normal patient, a heparin level of 3 u/ml ± 1 of blood typically produces a kaolin ACT of more than 450 seconds. Low molecular weight heparin is attractive in this setting as it causes a highly predictable anticoagulant effect for a given dose, decreasing the need for monitoring. 4. Suggested alternative methods for monitoring anticoagulation during CPB in APS patients include empirically doubling the baseline ACT or to reach an ACT twice the upper limit of normal, obtaining heparin concentrations by protamine titration, performing anti-factor Xa assays, or performing heparin/ACT titration curves preoperatively to determine patient specific target ACT levels. 5. Minimizing actions that contribute to hemolysis may reduce the effects of APS in the post-CPB period. 6. Patients need to remain anticoagulated post-CPB because the higher levels of hemolysis caused by CPB can stimulate clotting.					
I 17 FAILURE: Failure to	EFFECT: 1.De-ranged electrolytes including:	CAUSE: Swiss hypothermia classification:	PRE-EMPTIVE MANAGEMENT: 1.There is no pre-emptive management to prevent accidental hypothermia. 2.Peripheral vascular vasoconstriction may make arterial pressure	5	1	5	1	25

revive a patients form severe, accidental , non-drowning hypothermia. Swiss hypothermia classification: I: Awake and shivering (usually 35-32 C). II: Reduced conscience without shivering. (32-28 C). III:Uunconscious without shivering but with vital signs. (28-24 C). IV: Without vital signs. (<24). V: Dead by hypothermi. (< 13 C).	a.Low pH b.Low bicarbonate (HCO3) c.Negative base balance d.High potassium (K+) e.High sodium (Na+) usually due to excessive Na HCO3 administration during pre-pump resuscitation. f.High osmolarity: >300 milliosmoles usually due to hypernatremia. g.Excess administration of epinephrine, vasopressin and calcium may prevent adequate ECLS perfusion and aggravate reperfusion injury. 2.Pulmonary edema, partially from pre-pump resuscitation fluid administration. 3.Systemic capillary leak syndrome resulting in hypotension and anasarca. 4.Failure to wean from CPB. 5.Extended ECLS support required after CPB. 6.Coagulopathy. 7.Extensive brain and other organ damage. 8.Death	I: Awake and shivering (usually 35-32 C). II: Reduced conscience, no shivering. (32-28 C). III: Unconscious, no shivering & w/ VS (28-24 C). IV: No VS. (<24). V: Dead by hypothermia. (< 13 C). Accidental hypothermia classification: Mild; > 34C (932.F), <36C Moderate; >30C (86F), <34C Severe; <30C 1.Usually environmentally induced including immersion in water but not submersion (drowning). 2.Frequent contributing factors: a.Trauma b.Drug overdose c.Alcohol consumption d.Hypoglycemia e.Advanced age (adults) f.Low body mass index (children) 3.Non-drowning hypothermia slowly precipitates bradycardia to cardiac arrest limiting organ (brain) hypoxia compared to drowning. 4.Poor perfusion during severe hypothermia leads to physiologic disturbances	monitoring and pulse oximetry unreliable. 3. Prior to CPB, use slow, methodical manual or automated chest compressions (adult 40-50 bpm) to provide better peak pressure to the brain with a hypothermic vasoconstricted periphery. 4.Consider cerebral oximetry monitor even though a normal baseline is unobtainable. 5.Consider taking patient to surgical suite to implement extracorporeal support with all the anesthesia and CPB accoutrements. MANAGEMENT: 1.Prepare for sternal, femoral (adult) and neck (child) cannulation. Unknown anatomy may prevent femoral or neck cannulation. 2.Do not delay ECLS to wait for arterial monitor or central line placement. 3.Opt for CPB equipment over ECMO if time allows. CPB offers greater flexibility for temperature control, ZBUF use and circuit volume manipulation. 4. Use esophageal thermometer for core temperature measurement. 5.With a functioning peripheral arterial monitor; MAP ≥50 torr goal (adult). 6.Perform ongoing tests for electrolytes and osmolarity. 7.Electrolyte rebalance; use aggressive ZBUF of ½ NS w/ 50 mEq NaHCO3/L added. Reduces K+ and restores HCO3 without increasing osmolarity from hypernatremia. 8.Monitor venoarterial CO2 gradient to assess CO2 tissue retention; <15 torr goal. 9.Hemodilute to 25 % Hct to improve capillary perfusion during rewarming. 10.If no early return of cardiac function, perfuse lungs with gentle CPR or open massage. This helps to normalize pulmonary vasculature physiology. 11.Don't fully rewarm; target temperature = 33C +/- 1. Maintain mild hypothermia for 24-48 hours. 12.If ventricular tach (VT) or V fibrillation (VF) is present, defibrillation should be attempted once. Proper time/temperature to attempt defib is unknown, but repeat attempt at 30C. 13.During hypothermia, drug metabolism may be reduced. Medications (epinephrine, vasopressin) given during earlier resuscitation efforts could accumulate to toxic levels in the peripheral circulation.					

		that may or may not be reversible during CPB. 5. Excessive osmolarity from NaHCO3 or mannitol administration may result in organ damage: >320 = kidney damage, >360 = brain damage. 6.Reperfusion and rewarming with CPB may precipitate a lethal reperfusion injury.	14.Consider phenylephrine, lasix, steroids during rewarming. 15.Rewarm at 1 degree/15 minutes or slower if correction of physiology lags. 16.Prepare to implement ECMO for at least 24 hours after rewarming and after the correction of electrolytes and other physiology.					
I 18. FAILURE: Failure to prevent post pump chorea from developing after CPB using deep hypothermic circulatory arrest (DHCA). (5/23/16)	EFFECT: 1.The encephalopathy ranges from transient and mild to persistent and severe. 2. Develops within two weeks after CPB. 3. Causes delayed development in: a. memory b. attention c. language skills d. motor skills e. IQ 4. Neurological symptoms: a. choreoathetosis b. dystonia c. hypotonia d. obtundation 5. Death	CAUSE: 1.Most commonly occurs in children but does occur in adults. (* If the patient is a child the occurrence is about 1.2% of cases, so the Occurrence RPN should be increased to 2.) 2.The delayed onset may reflect the time required for the diffuse cellular atrophy to mature. 3.High risk patient characteristics: a.often have pre-existing developmental delay. b.undergo longer CPB times at lower temperatures. c.are more likely to have had DHCA with a cooling time <20 minutes and alpha-stat pH management during CPB (Levin et al. 2005). 4.Exact etiology and pathophysiology is	PRE-EMPTIVE MANAGEMENT: 1.pH-stat gas strategy with hyperoxia during CPB cooling maximizes capillary perfusion and shifts the oxy-hemoglobin dissociation curve to the right for better oxygen release to the tissues. This allows maximum tissue oxygen loading prior to arrest. 2.Pre-arrest preparation by oxygen loading of tissues in combination with pH stat gas strategy can delay the conversion from aerobic to anaerobic metabolism, thereby extending the safe DHCA time. 3.pH-stat gas strategy with hyperoxia during CPB cooling results in 85% less acid production during the hypothermic arrest period than normoxia with alpha-stat gas strategy. (Pearl at al, 2000). 4. Traditional methods of DHCA rely on reaching a specified temperature without consideration of impaired O2-hemoglobin disassociation, dissolved O2 utilization or tissue oxygen loading. 5.Cooling for at least 20 minutes, reaching a temperature of 18C and a pvO2 of 300+mmHg may provide the most favorable conditions for DHCA of the brain. 6.Rewarming with normoxia may reduce the risk of reperfusion injury in an acidotic brain after a prolonged circulatory arrest. MANAGEMENT: Since post pump chorea develops several days after surgery and because there is no clear cut way to determine if it will develop during CPB, there is no perfusion management strategy.	3	1*	5	2	30

		unknown, but it is thought that chorea and similar neurological complications most likely result from non-embolic hypoxia-ischaemia within diffuse cellular areas rather than global hypoperfusion of the brain. 5.Area of damage is thought to be near the basal ganglia, but multiple EEGs and MRIs have not confirmed this. 6.Hemodilution during CPB reduces the amount of hemoglobin-bound O2 for use. 7.At profound temperatures, the affinity of O2 for hemoglobin greatly increases and impairs the disassociation of O2. 8.Low CO2 concentration (alpha-stat gas control) further impairs the disassociation of O2 from hemoglobin. 9.Dissolved O2 is the primary source of O2 for brain tissue at profound hypothermia (Dexter 1997). 10.Low FiO2 sweep gas will fully saturate hemoglobin but reduces dissolved O2. This may result in diffuse cellular hypoxia even in the presence of hyperoxemia. (du Plessis et al, 1995).						

J. HYPOTENSION DURING CPB								
J1. FAILURE: Failure to prevent refractory hypotension following CPB initiation. (12/22/15) .	EFFECT: 1. Hypotension 2. Indeterminate effect on organ systems.	CAUSE: 1. Hemodilution causing low blood viscosity. 2. Catecholamine dilution. 3. Atrial natriuretic factor increase. 4. Loss of pulsatility. 5. Aortic baroreceptor vasodilation. 6. Chelation of iCa+2 by citrate in the pump prime or cardioplegia solution. 7. Dilution of iCa+2 by a calcium free pump prime.	PRE-EMPTIVE MANAGEMENT: 1. Draining the venous system slowly and initiating bypass rapidly may stymie hypotension development. MANAGEMENT: 1. Increase blood flow 2. Increase blood viscosity by ultrafiltration 3. Reduce arterial pCO2 for systemic vasoconstriction 3. Consider giving phenylephrine. 4. Consider giving norepinephrine.	3	4	1	3	36
J2. FAILURE Hypotension following phenylephrine and/or norepinephrine administration: level II. (12/22/15)	EFFECT: 1. Refractory hypotension despite adequate blood flow 2. Inadequate perfusion of vital organs 3. Temporary or permanent organ damage 4. Failure to wean from CPB. 5. Preoperative use of ACE inhibitors is associated with increased postop morbidity possibly related to hypotension during CPB.	CAUSE: 1.Bradykinin accumulation or release during CPB. A history of ACE inhibitor usage, particularly in large doses, is associated with bradykinin release during CPB. 2.Preoperative ACE inhibitor use among smokers further potentiates the kinin response to CPB and is causes hypotension during CPB. 3. During CPB a significant rise in kallikrein also leads to the formation of bradykinin. Since the primary site of bradykinin breakdown is the lungs, and the bypass circuit removes the lungs from the circulation, the primary site for removal of excess	PRE-EMPTIVE MANAGEMENT; There are no pre-emptive management processes for this failure. Patients taking ACE inhibitors have a higher mortality after heart surgery. However this may simply co-inside with sicker patients who need such medication. Stopping this medication prior to heart surgery is controversial and should be taken on a case-by-case basis. (* Patients who are taking ACE inhibitors are more frequently associated with Level II hypotension, but their frequency in the population is less. Such patients would have an RPN of only 4*4*2*2 = 64 as a group, but the Occurrence RPN for individual patients is higher than those not taking ACE inhibitors.) MANAGEMENT: 1.After utilizing all management interventions for Level I hypotension consider: a.Epinephrine bolus and/or drip (100 mcgm/1000 mls NS. b.Arginine Vasopressin (AVP), an endogenous antidiuretic hormone essential for cardiovascular homeostasis and released during baroreflex response. It constricts vascular smooth muscle. Adult Dosage: 1 – 2 units bolus; 1 – 4 units/hr. Pediatric Dosage: 0.0003-0.002 units/kg/min. 2.Failure to wean from CPB will necessitate ventricular support for an indeterminate period. 3.If patient weans from CPB and transits to the ICU, be prepared for	4	3*	2	3*	72

		bradykinin is lost leading to its general accumulation. 4. Reaction to artificial surfaces or some types of membranes; i.e. polyacrylonitrile or cuprophane membranes used in hemodialyzers or ultrafiltrators. 5. Increased bradykinin contributes to protamine associated hypotension in ACE inhibitor patients after CPB.	extracorporeal support (ECPR) should patient have sudden cardiac arrest in the post-op period.					
J3. FAILURE: Failure to prevent hypotension following phenylephrine, norepinephrine , epinephrine or vasopressin administration: level III. (12/22/15)	EFFECT: 1. Refractory hypotension despite adequate blood flow and treatment with phenylephrine, norepinephrine, epinephrine and vasopressin. 2. Inadequate perfusion of vital organs 3. Temporary or permanent organ damage 4. Failure to wean from CPB. 5. Death	CAUSE: 1. CPB associated vasoplegia of unknown origin. 2. In severe sepsis, excessive formation of NO & c-GMP are associated with profound vasodilatation, hyporeactivity to catecholamines, & myocardial depression. CPB may initiate a systemic inflammatory response syndrome (SIRS) similar to severe sepsis. CPB SIRS causes endothelial production and release of NO & c-GMP, causing profound hypotension; essentially an anaphylactic response to CPB. 3. Anaphylaxis to antibiotics. 4. Transfusion reaction.	PRE-EMPTIVE MANAGEMENT: 1.Precaution: Methylene blue (MB) can be used to treat vasoplegia, but it may be contraindicated in patients taking selective serotonin reuptake inhibitors (SSRIs). 2.Heart failure patients and chronically ill patients may take SSRIs for depression. 3.MB may induce serotonin syndrome as a result of its effect on monoamine oxidase activity. 4.Symptoms of serotonin syndrome: a.Agitation b.Confusion c.Tachycardia d.Hypertension e.Pupil dilation f.Muscular spasms or rigidity g.Diaphoresis h.Diarrhea 5.SSRIs: a.Citalopram (Celexa) b.Escitalopram (Lexapro) c.Fluoxetine (Prozac) d.Fluvoxamine (Luvox) e.Paroxetine (Paxil, Pexeva) f.Sertraline (Zoloft) g.Vilazodone (Viibryd)	5	2	3	3	90

		5. Bradykinin is the mediator of hypotensive symptoms in hereditary angioedema (HAE) patients. 6. Persistent LSVC 7. Poorly protected hypertrophic heart (see section on hypertrophic heart) 8. Intraop MI 9. Alpha gal allergy from tick bite 10. Lidocaine overdose or acute toxicity. 11. Mastocytosis: degranulation of mast cells causing SEVERE anaphylaxis. Stimulated by opiate derivatives, NSAIDs, alcohol and hypothermia. 12. Protamine reaction 13. Histamine reaction 14. Unknown drug allergy or reaction	MANAGEMENT: 1. CPB initiation & cardioplegia delivery can cause a precipitous & refractory drop in the SVR of patients in septic shock. 2. Boluses of α-1 agonists or vasopressin usually reverse this drop in SVR. But, in some cases, this may not normalize SVR. 3. MB, a NO & c-GMP inhibitor, can reverse severe vasodilatory shock. MB inhibits both constitutive & inducible nitric oxide synthase (c-NOS & i-NOS). The additional effect of MB on soluble guanylyl cyclase adds to the inhibition of the NO/c-GMP pathway. 4. Adult Dosage: 2 mg/kg over 15-20 min 5. Pediatric Dosage: 1 mg/kg during a 1-hour period 6. Epinephrine and Benadryl if antibiotic anaphylaxis is suspected. 7. Consider steroids if inflammatory response is suspected. 8. Consider high-dose intravenous hydroxocobalamin (Vit B12) as an alternative to MB especially in patients taking SSRIs. Vitamin B12 treats vasoplegia by the binding of nitric oxide (NO) and directly inhibiting NO synthase and guanylate cyclase. (Roderique JD et al, 2014). 9. High flow ventricular support may be necessary to wean from CPB or utilized in the immediate postop period. PRECAUTIONS: 1. MB skin staining & discoloration is known to interfere with pulse & cerebral oximetry. 2. MB blood discoloration is known to interfere with near IR spectroscopy used to measure vSAT in the ECC. 3. MB is contraindicated during pregnancy. 4. MB has the potential to cause hemolytic anemia & hyperbilirubinemia in the newborn. 5. Other safety concerns include oximeter interference, pulmonary hypertension, neurotoxicity, arrhythmias, and potentially altered coronary, mesenteric, and renal perfusion.					
K. HEMORRHAGE								
K1. FAILURE: Hemorrhage secondary to redo sternotomy.	EFFECT: 1. Uncontrollable exsanguination 2. Hypovolemic shock 3. Coronary ischemia 4. Irreversible arrhythmia 5. Cardiac arrest 6. Death	CAUSE: 1.A redo sternotomy carries a great risk of massive hemorrhage. 2.This is because of adhesion of the heart and associated structures to the underside of the sternum.	PRE-EMPTIVE MANAGEMENT: 1.The perfusionist should be in the room during sternotomy as a standard of practice. 2.Read the cardiac catheterization report to confirm that there is no problem with entering the femoral vessels and that the inferior vena cava is continuous from the femoral vein to the right atrium. 3.Hand off the pump lines before opening the sternum for redo cases. 4.If available, have a heparin dose response test performed pre-CPB	5	2	1	2	20

		3.The process of dissection through the sternum may disrupt vital structures that can lead to sudden and excessive hemorrhage. 4.Excessive scarring may prevent rapid entry into the chest to control the hemorrhage. 5.Radiographic evidence of this may be obvious on a lateral CXR.	to estimate the heparin dose if there is no time for ACT confirmation should rapid exsanguination occur. Expect some redo patients to have heparin resistance or other blood dyscrasia. 5.Be prepared for fem-fem or right neck CPB by selecting appropriate arterial and venous cannulae capable of carrying as great a blood flow as vessel size will allow. Have cannulae and any associated accessories in the room before the sternotomy incision is started. In addition to the desired size cannula, a smaller size cannula should also be in the room should the desired sizes not accommodate the vessels. Extra tubing should be in the room to modify the AV loop as needed to accommodate cannula placement. Extra straight and Y-connectors should be in the room. 6.A dedicated autotransfusionist should be prepared to supply two aspiration/anticoagulation lines with two cardiotomy reservoirs. The autotransfusion equipment should be modified to collect any rapid blood loss and quickly pump it to the open heart pump. The processing mode should be changed from a Quality Wash mode or High Wash mode to the Emergent mode to expedite blood return to the patient, should exsanguination occur from the field. 7.A second scrub nurse should remain at the field until the sternum is safely opened and the aortic and venous cannulation stitches are in place or until fem-fem or right neck CPB is initiated. The second scrub nurse should also standby to change cannulae or reconfigure the tubing at the direction of the surgeon. (Second assist personnel like a medical student or resident should not displace a knowledgeable scrub nurse who can quickly make the necessary circuit modifications until the danger of uncontrollable hemorrhage has passed.) 8.The perfusionist should be aware if any danger of over distention of the heart exists due to aortic valve insufficiency, sudden fibrillation or poor ejection fraction. Once started CPB should be conducted appropriately to each situation until the chest is opened and the surgeon is in control of the over distention risk. 9.Extra 40 micron blood filters should be in the room. A 40 micron blood filter should not be used for more than four units of autotransfusion or donor blood. 10.Additional vials of heparin should be available with additional heparin drawn up and ready to give into the pump in an emergency. This is in addition to the normal amount of heparin in the prime. 11.Prior to the chest being opened, the perfusionist should confirm					

			that blood products are in the room and properly checked. 12.The perfusionist should have additional IV fluids, additional drugs, and extra tubing clamps on the pump. 13.Extra perfusion personnel should stand by during any redo sternotomy. MANAGEMENT: 1.Should a sudden sternal hemorrhage occur, the patient should be heparinized immediately in preparation for CPB and to prevent clotting of blood collected by the autotransfusion equipment or the pump suckers. The anticoagulant drip on the autotransfusion equipment should be opened completely to prevent the clotting of the rapidly shed blood. If a hemorrhage is large enough, the heparin given by the anesthesiologist might be lost to the field or to the autotransfusion system and not reach the entire blood volume. If the patient is exsanguinating, give extra heparin into the pump while going on CPB. If a hemorrhage occurs (or even before a hemorrhage can occur), the surgeon may place the patient on cardiopulmonary bypass (CPB) by cannulation of a femoral artery and femoral vein or the right carotid artery and right internal jugular vein if the patient weights less than 15 kg. 2.After the arterial cannula is placed, CPB can be initiated and any hemorrhage from the sternotomy can be suctioned into the CPB pump (sucker bypass) and returned to the patient. The hemodynamics can be partially or fully controlled in this way. However the high speed use of the suckers will generate excessive gaseous microemboli. During this time, the sweep gas should be maintained at 100% oxygen to off-gas the nitrogen microemboli entering the patient's circulation from the pump. 3.If the blood loss compromises the patient's hemodynamics before CPB can be initiated, a large bore IV tubing extension should be passed from a bubble free site on open heart pump to a central line access in the patient. The open heart pump can then be used to rapidly re-infuse fluid into the patient to restore hemodynamic stability through this line. (Ensure that the pressure servo-regulation system will prevent rupture of the circuit by a high infusion pressure.) As blood is collected by the sterile cardiotomy suction or autotransfusion equipment it should be pumped unprocessed into the open heart pump and re-infused through the large bore IV tubing to the patient until fem-fem or right neck CPB can be established. If the					

			loss of patient blood volume is rapid and extensive, peripheral cannulation might be difficult due to circulatory collapse. Rapid re-infusion of shed blood may be necessary to aid in peripheral dilation and cannulation. Note how long the mean arterial pressure was decreased. 4.Vacuum assist needs to be available, but do not implement vacuum assist until authorized by the surgeon if neck or femoral cannulation is utilized. If a major structure of the heart has been breached during sternal entry excessive bleeding may ensue. But the implementation of vacuum assist venous drainage may decompress the heart to the point of sucking air in through the sternal breach. If this occurs in patients with common atria or common ventricle, an air embolus could be pumped into the aorta by the heart. 5.Save blood bags and mark them with the time of administration until charting can be caught up. 6.If you give more than four processed units of autotransfusion blood, consider giving FFP to restore clotting factors. 7.Calcium levels should also be monitored and calcium given if large volumes of citrated donor blood are administered. 8.Monitor urine output. If large quantities of blood products have been given, mannitol should be administered to flush out free hemoglobin in the kidneys. 9.Charting is second to patient care, but it is important to note times. Ask for assistance from nursing or other personnel to write or chart. 10.Coagulation factors should be measured prior to coming off CPB. If possible, draw a thromboelastography test to assess the clotting status of the patient.					
K2. FAILURE: Acute exsanguination of patient at the initiation of CPB.	EFFECT: 1. Blood loss 2. Hemodynamic instability 3. Organ damage. 4. Death	CAUSE: 1. Arterial cannula (aortic or femoral) trapped in vessel wall or perforated through arterial wall 2. Arterial cannula dislodges at the initiation of CPB limiting forward arterial blood flow while patient blood volume drains into venous reservoir.	PRE-EMPTIVE MANAGEMENT: 1. Assess pressure fluctuation on cannula after placement. 2. Monitor arterial line pressure for continuous normal fluctuation. 3. Compare cannula pressure with mean arterial pressure. 4. Notify surgeon of any irregularities before or at the initiation of CPB. 5. Do not initiate CPB until surgeon revises arterial cannula placement. MANAGEMENT: 1. Prevent or limit exsanguination of the patient's blood into the CPB circuit should forward arterial blood flow have to be stopped. 2. Clamp arterial line and venous return line immediately.	4	1	4	3	48

			3. Connect purge line or some other large bore tubing from the arterial side of the CPB circuit to the venous sample port on the venous line. 4. Pump blood from the venous reservoir up the venous line as quickly as possible to return circulating volume to the patient. 5. Do not initiate CPB until surgeon revises arterial cannula placement.					
L. ELECTROLYTE CONTROL								
L 1. FAILURE Low bicarbonate (HCO3) level during CPB. (5/23/19)	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	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 NaHCO3 added 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	2	3	2	3	36

		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.	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.					

L 2. FAILURE Failure to regulate blood ionized calcium (iCa+2) as necessary. (7/14/16)	EFFECT: 1. Hemodynamic instability 2. Cardiac distention. 3. Risk of overdose. 4. Brain damage. 5. Death.	CAUSE: 1.Serum and extracellular ionized calcium (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	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. a.An ampule of 10 ml/10% calcium gluconate contains 8.9 mg/mL (4.65 mEq/gm) of elemental calcium. b.An ampule of 10ml/10% calcium chloride contains 27.2 mg/mL (13.6 mEq/gm) of elemental calcium. 3.CaCl2 is more caustic and may cause intravascular tissue damage or tissue necrosis with extravasation. 4.CaGluc must be metabolized in the liver before it becomes bioavailable. 5.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	5	2	2*	3	60

		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.	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 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.					
L3. FAILURE: Failure to prevent hyperkalemia. 3/16/16	EFFECT: 1. ECG progresses from peaked T waves and shortened QT to lengthening PR and loss of P waves followed by QRS	CAUSES: 1.Cardiac repolarization failure. 2.Excess administration of high potassium cardioplegia (HPCP) solution.	PRE-EMPTIVE MANAGEMENT: 1. Administer appropriate HPCP dose during appropriate time period 2. Double clamp HPCP source tubing when not in use. 3. Fill cardioplegia holding container with only enough HPCP solution to administer the appropriate dose. 4. Maintain adequate perfusion pressure during full flow period for	2	2	2	3	24

	widening with sine wave morphology. 2. Cardiac ventricular fibrillation, wide complex PEA and asystole. 3. Failure to wean from CPB.	3.Accidental overdose of HPCP solution. 4.Patient with end-stage renal failure or acute renal failure. 4. Excessive hemolysis of red blood cells. 5. Excessive administration of banked red blood cells that have not been washed. 6. Heat exchanger leak. 7. Procedurally required low flow state during normothermia or hypothermia reduces renal function and may lead to hyperkalemia.	proper renal function. 5. Minimize air/blood interface in suckers & vent by using only the minimum pump speed needed. 5. Use only minimum vacuum assist needed. 6. Use fresh (less than 7 days old) RBCs for transfusion if available or washed cells. 7. Test for heat exchanger (HE) leak prior to CPB (see HE leak under oxygenator heat exchanger failure). 8. Monitor urine output during CPB w/ a goal of 1-3 ml/kg/hr. MANAGEMENT: 1. Intravenous calcium chloride or gluconate can quickly block hyperkalemia effect on cardiac myocytes by restoring a balanced electrical gradient across the cellular membrane. 2. Administer diuretics prophylactically if urine output < 1 ml/kg/hr during CPB. 3. Alkalize blood with NaHCO3. 4. Administer normal saline in 250 mls aliquots and ultrafiltrate excess volume; (zero balance ultrafiltration, ZBUF). 5. Recheck K+ level and repeat 2, 3 and 4 as necessary. 6. If K+ fails to drop consider glucose-insulin therapy. 7. Treat anemia or high K+ from hemolysis by ultrafiltration (UF) fluid removal and/or added RBCs. 8. Use diuresis; furosemide if renal perfusion is good and mannitol +UF if it is not. 8. Change out oxygenator if the heat exchanger is defective using a PRONTO line. If a PRONTO line is not in standard use, increase the Harmfulness RPN to 4. This would give a total RPN of 4*2*2*3 = 48. 9. Hemodialysis during or immediately after CPB.					