

Cognitive Aids for Congenital Cardiac Anesthesia

Cognitive aids are designed to prompt users to complete a series of tasks and can take many different forms. We have designed a series of cognitive aids for congenital cardiac anesthesia for common and uncommon scenarios to try to capture experience and wisdom not published elsewhere. Congenital cardiac anesthesia involves taking care of very complex patients in many rare and challenging scenarios. Each individual cardiac anesthesiologist may only experience one or two of these emergency situations in a career. And for individuals who are training, our colleagues starting out in practice, or for those who have a low patient volume of cardiac anesthesia this resource may be invaluable. However, for even the most experienced amongst us, it can be helpful to review these aids to ensure that nothing has been forgotten. These cognitive aids are meant as a supplement to good clinical decision making, existing knowledge, and local experience. We encourage you to use them and to help us grow this resource over time. We have tried to be as accurate and complete as possible. However, if you notice any omissions or inaccuracies, please let us know so we can update the cards accordingly.

Thank you for downloading these cards. We encourage you to print, laminate, and post these in your operating rooms, cardiac cath labs, and other areas where you take care of patients with congenital heart disease.

A huge thank you to the CCAS. These cards were created by the Quality and Safety Committee, but there were many others who have contributed cards or edits. In alphabetical order:

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Topic List

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Veno-Arterial ECMO Cannulation
Vasoplegia



A Section of the Society for Pediatric Anesthesia

Activated Clotting Time (ACT) Inadequate

Related cards: bivalrudin

Immediate actions:
Notify team

Treatments to Consider

- Check that heparin was administered
 - Ideally use a CVL
 - Confirm aspiration of blood and flushed completely
- Give additional heparin bolus
 - (1/2 or same heparin dose as initial dose)
- Recheck ACT
- Transfuse FFP - 10 ml/kg OR Give ATIII (antithrombin) concentrate – 10-50 units/kg empirically
- Recheck ACT

Other considerations

- Send blood sample for ATIII levels
 - Sodium citrate tube (blue top)
- Double check heparin vial expiration date or administer additional dose from a new vial
- Consider alternative anticoagulant
 - E.g. Bivalrudin

Summary

Heparin binds and accelerates the activity of antithrombin III(ATIII) which then inhibits the activated clotting factors involved in the clotting sequence, mainly Xa and thrombin.

Activated clotting time is a whole blood clotting time test that initiates clotting and measures the time until clot formation. It is a point-of-care test. Results can be affected by several factors including platelet count & function, lupus anticoagulants, factor deficiency, blood volume, warfarin use, technique employed, ambient temperature and hemodilution.

Generally, an ACT >300 seconds is the minimum required to prevent clot formation in the oxygenator circuit. Thus, an ACT above 400-480 seconds (based on institutional protocol) is targeted as a safety margin before initiating CPB. One possible cause of an inability to achieve adequate ACT levels is due to inadequate AT III levels. AT concentrate or fresh frozen plasma may be given to increase these levels with institutional preferences/availability guiding the most appropriate therapy.

Selected References

Bull BS, et al. Heparin therapy during extracorporeal circulation. I. Problems inherent in existing heparin protocols. *J Thorac Cardiovas Surg* 1975; 69: 674-84

Arachchilage DRJ, et al. Use of antithrombin concentrate for acquired antithrombin deficiency in acutely unwell children receiving unfractionated heparin. *Semin Thromb Hemost*. 2019; 45: 859-864

Air Embolism

Related cards: sepsis, protamine reaction, air embolus, blood transfusion reaction, anaphylaxis

Immediate actions:

Call for help, Notify team, "Stop the Pump"

Treatments to Consider

- Stop CPB; Clamp lines and shut off suction lines
- Steep Trendelenburg (head DOWN)
- 100% FiO₂
- Cool patient, ice on head
- Remove arterial cannula to de-air patient, de-air circuit
- Retrograde regional cerebral perfusion:
 - a) Connect arterial line to SVC cannula, suckers for blood return
 - b) Flow at 15cc/kg/min (peds) , 500-800 cc/min (adult) until air is gone
 - c) Consider intermittent bilateral carotid compression
- Resume antegrade perfusion
 - a) Maintain deep hypothermia (18-24°C) for 30-60 min
 - b) Keep flows on CPB as high as possible, induce hypertension with medications as necessary
 - c) Consider steroids
 - d) Maintain PaCO₂ 35-45, FiO₂ 100% until 6 hours post
 - e) Deepen anesthetic
 - consider barbiturates
- Slowly warm to 34-35°C
 - Keep temperature gradient small (<8°C)

Other considerations

- Involve ICU and neurology
- Timing of CT head
- Hyperbaric oxygen therapy may be considered

Diagnosis

- Detection of air in heart by TEE
- Abrupt drop in cerebral NIRS
- Visualization of air in cannulas or bubble detector alarm on CPB

Causes

- Ejection during de-airing on open beating heart
- Vortexing, emptying or pressurizing venous reservoir
- Failure of positive pressure release valve
- Obstruction of reservoir vent or gas outlet port
- Reversed flow in vent or arterial line
- Unintended unclamped arterial line
- Leak or kink upstream from roller pump

Summary

Air embolism on CPB can lead to air in the cerebral circulation which can cause severe neurologic damage and/or death. A quick multi-faceted response, often including retrograde regional cerebral perfusion, is necessary to reduce adverse effects. A summary of steps includes:

- Promptly identify the air embolism and cause
- Limit the damage
- Decrease metabolic oxygen consumption
- Decrease inflammatory response to the insult
- Increase tissue perfusion and oxygen delivery
- Consider retrograde cerebral perfusion

Selected References

- Quintero OL, et al. Successful management of massive air embolism during cardiopulmonary bypass using multimodal neuroprotection strategies. *Semin Cardiothorac Vasc Anesth.* 2019; 23: 324-32
- Van der Zee MP, et al. Fatal air embolism during cardiopulmonary bypass: analysis of an incident and prevention measures. *Interact CardioVasc Thorac Surg.* 2014;19:875-57
- Sahu MK, et al. Successful Management of a case of massive air embolism from cardiopulmonary bypass with retrograde cerebral perfusion in a child. *J of Cardiothorac Vasc Anesth.* 2006; 20 :80-81

Air Lock on CPB

Related cards: air embolism

Immediate actions

Notify team, Call for help

Treatments to Consider

- Check
 - Search for source of venous outflow line air
 - loose atrial purse string stitch
 - atrial tear
 - open intravenous access
 - Venous cannula placement
 - Look for IVC/SVC/RA damage
- Actions
 - Identify and repair/address cause of air entry
 - Slow or stop pump flow to prevent venous reservoir emptying
 - Perfusion will clamp venous line at the reservoir
 - Perfusion will administer volume into venous reservoir
 - Surgeon/Perfusion will re-prime the venous line
 - Suggest additional suture around venous catheter

Causes

Air enters the venous outflow line at the surgical field, creating an interruption in venous gravity drainage. Results in complete cessation of flow to venous reservoir.

Signs/Symptoms

- CBP venous reservoir alarm may sound
- Decrease in MAP due to decreased pump forward flow
- Increase in CVP

Consequences

- Inability of perfusion to delivery arterial flow to patient
- Decreased MAPs
- Decreased delivery of oxygen

Summary

An air lock occurs if air entering the venous outflow line results in complete cessation of flow to the venous reservoir. The loss of venous outflow necessitates immediate slowing, even cessation, of pump flow to prevent emptying the reservoir. After an air lock is recognized, the source of venous outflow line air must be identified and repaired before re-establishing full bypass.

Selected References

- Mora-Mangano C, et al. Cardiopulmonary bypass and the anesthesiologist - McGill. Retrieved November 21, 2021, from https://www.mcgill.ca/anesthesia/files/anesthesia/wk_6a_kaplan_22_cpb1.pdf
- Cheung AT, et al. Management of cardiopulmonary bypass. UpToDate. Retrieved November 21, 2021, from <https://www.uptodate.com/contents/management-of-cardiopulmonary-bypass>

Anaphylaxis

Related cards: sepsis, protamine reaction, air embolus, blood transfusion reaction

Immediate actions:
Call for Help, Notify team

Treatments to Consider

- Epinephrine boluses (1-10 mcg/kg IV) as needed and/or infusion
- Volume as necessary
- If refractory hypotension, consider:
 - Vasopressin 0.01 units/kg IV
- Stop all other medication administration if possible
- Steroid: methylprednisolone 2mg/kg (MAX 100mg)
- Histamine blockade: Diphenhydramine 1 mg/kg IV/IO (MAX 50 mg) or Famotidine 0.25 mg/kg IV (MAX 20 mg) or Ranitidine 1 mg/kg IV
- Consider echocardiography to rule out other causes

Other considerations

- Send a Tryptase level as soon as possible (if on CPB, send again once off CPB)
- Common allergens: antibiotics, paralytics, protamine, chlorhexidine, blood products, volume expanders, latex, aprotinin, thrombin/gelatin hemostatic agents (e.g. Floseal)
- Allergy consult & follow-up

Signs

- Bronchospasm: ↑PIP or ↓TV or ↓EtCO₂
- Refractory hypotension
- Arrhythmias or cardiac arrest
- Rash
- On CPB:
 - inadequate venous return, high flows, low MAPs

Summary

Anaphylaxis may be encountered due to the large number of medications and substances to which a patient is exposed during cardiac procedures. Anaphylaxis may be difficult to diagnose due to multiple other causes of cardiopulmonary instability. Although cardiopulmonary bypass has been suggested as a therapy there are also case reports of refractory hypotension on bypass. The mainstay of treatment for anaphylaxis is epinephrine for mast cell stabilization.

Selected References:

- Levy J, et al. Anaphylaxis during cardiac surgery: Implications for Clinicians. *Anesthesia & Analgesia*. 2008;106:392-403
- Fords SA, et al. Anaphylactic or anaphylactoid reactions in patients undergoing cardiac surgery. *Cardiothorac Vasc Anesth*. 2001;15:684-8
- Weissgerber AJ. Methylene blue for refractory hypotension: a case report. *AANA J*. 1008;76:271-4
- Harper NJN, et al. Suspected Anaphylactic Reactions Associated with Anaesthesia. *Anaesthesia*. 2009; 64:199-21

Aortic Cannula Dislodgment

Related cards: air embolism on CPB, aortic dissection

Immediate actions:

Notify Surgical team, Notify Perfusion, Call for Help

Treatments to Consider

- Assess the surgical field for blood loss, MAP, CVP, NIRS
- Transfuse volume/PRBC's as needed in PIVs, CVL
- Call for ice for the head and cool room and patient as able
- Support blood pressure with inotropes/vasopressors
- Consider head down position to minimize risk of air embolism
Use echo to assess for intracardiac & intraaortic air (if concern for air, see air embolism card)
- Surgical team should occlude site of cannula dislodgement to decrease blood loss
- If on CPB, assess site and re-insert cannula; if unable, assess for other sites for cannulation
- If occurs pre- or post-CPB, repair the site & assess for further injury (dissection)
- Perfusion should stop the pump, clamp aortic line, & clamp venous line to stop draining the patient if on CPB; clamp the vent
- Note that the venous line can be used to transfuse retrograde if occurs pre- or post-CPB
- Perfusion should give a new cannula to surgical team for re-insertion

Causes

- Technical issue: loose purse string suture, shallow placement
- Equipment failure (cannula defect)
- Aortic injury

Signs/Symptoms

- Decrease in MAP
- Blood pooling in field
- Decreased venous return to CPB
- Decrease in cerebral and somatic NIRS

Consequences

- Massive blood loss
- Decreased cerebral and vital organ perfusion
- Risk of air embolism & cerebral injury
- Injury to aorta

Summary

Aortic cannula dislodgement during cardiopulmonary bypass is a rare event, and should be considered preventable in most circumstances as it will generally relate to a technical or equipment failure. If this occurs in the pre-or post-bypass phase, the primary goal must be to obtain source control of bleeding and repair the site of injury while supporting the patient's circulation. However, if this occurs on cardiopulmonary bypass, the priority must be on replacing the aortic cannula as efficiently as possible, as it will be unlikely to be able to quickly regain perfusion. Transfuse, cool, and maintain systemic perfusion pressures as able.

Selected References

- Gargava A, et al. Aortic cannula tip dislodgement: A rare complication. *Annals of cardiac anaesthesia*. Retrieved November 22, 2021, from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7879888/>
- Singh RS, et al. Disconnection of the tip of the aortic cannula during cardiopulmonary bypass. *Interactive CardioVascular and Thoracic Surgery*, 2009;8:296–300

Aortic Dissection

Related cards: hypertension/hypotension on CPB, aortic cannula dislodgement, abnormal NIRS on CPB, sepsis

Immediate actions:

Call for help, Notify team

Treatments to Consider

- Minimize wall stress:
 - Reduce MAP and pump flows to lowest acceptable levels
 - Best first agent may be esmolol or labetalol
- Separation from CPB and establishment of alternative cannulation site
- Hypothermia for cerebral protection and to decrease metabolic demand
- Diagnosis
 - Type A (ascending or arch) vs. Type B (descending)
 - Visual inspection
 - Epicardial echo for ascending, TEE for arch/descending
 - Type will determine surgical intervention

Other considerations

- Rare in children
- Risk increased with large aortas and/or hypertension
 - e.g. Marfans, Ehlers-Danlos, Williams syndrome
- Diagnosis: Visual inspection, aortotomy, TEE, epi-aortic ultrasound, NIRS asymmetry
- Perfusion plan may involve deep hypothermic circulatory arrest, antegrade cerebral perfusion, retrograde cerebral perfusion depending on location and extent of injury

Signs

- Systemic hypotension that is inadequately responsive to vasoconstrictors and increased pump flow
- Systemic hypoperfusion (e.g. oliguria/anuria, acidemia, low NIRS)
- Visual inspection of aorta: blue discoloration, bleeding, hematoma, increasing diameter
- Acute aortic insufficiency, wall motion abnormalities, ST segment changes
- CPB: High arterial line pressure, inadequate venous return

Summary

Intraoperative iatrogenic aortic dissection is a rare but potentially fatal complication. It occurs in 0.12-0.35% of adult cardiac surgery cases. Known predisposing factors include connective tissue disease, hypertension, aortic pathology, atherosclerosis, and preoperative steroids, but can occur in the absence of disease.

Selected References

- Troianos CA, et al. Transesophageal echocardiographic diagnosis of aortic dissection during cardiac surgery. *Anesthesiology*. 1991;75:149-53
- Mora-Mangano C, et al. Cardiopulmonary Bypass and the Anesthesiologist. From *Kaplan's Essentials of Cardiac Anesthesia* (2nd Ed), p. 513-545
- Wilkey BJ, et al. Anesthetic Considerations for Surgery on the Aortic Arch. *Seminars in Cardiothoracic and Vascular Anesthesia*. 2016;20:265-272
- Hibino N, et al. Intraoperative Aortic Dissection in Pediatric Heart Surgery. *Asian Cardiovasc Thorac Ann*. 2006;14:e55-7.
- Gukop P, et al. Principles for Management of Intraoperative Acute Type A Aortic Dissection. *Aorta*. 2015;3:191-4

Atrial Septal Defect Device Embolization

Related cards:

Immediate actions:

Call for Help, Notify Interventional Cardiologist, and/or Cardiac Surgical Team

Treatments to Consider

- Call for x-ray and/or echocardiography to confirm diagnosis
Note: Percutaneous retrieval will likely be the 1st technique attempted to remove a migrated device if the patient is hemodynamically stable; CPB or ECMO may be necessary if retrieval attempt fails or patient is hemodynamically unstable.
- Consider that the patient will likely need to be moved to the catheterization laboratory
- Start supplemental oxygen and consider intubation
- Establish appropriate access
 - PIVs +/- Arterial line +/- CVL
 - Call for PRBC to be available
 - Transthoracic or transesophageal echocardiography:
 - Identify device location, damage to cardiac structures (valves), function
- Supportive medical management
 - Start vasopressors and/or inotropes to support the hemodynamics
 - Strongly consider anticoagulation: 100 Units/kg of heparin due to thrombus risk

Signs and Symptoms

- Arrhythmias (supraventricular or ventricular)
- Chest pain or palpitations
- Hemodynamic instability
- Hypoxia
- Cardiac Arrest
- May also be asymptomatic

Risk Factors for Embolization

- Large ASD with small aortic rim
- Under or oversizing of the device
- Inadequate device placement
- Valsalva post device placement
 - Coughing, vomiting

Other Potential Procedural Risks (ASD Device Closure)

- Air embolism
- Vascular trauma from large sheaths
- Thrombus, CVA
- Obstruction of systemic venous return or pulmonary vein(s)
- Aortic perforation or atrial perforation
- Infective endocarditis
- Arrhythmias
- Damage to mitral/tricuspid valve structures

Summary

There are different FDA-approved devices for ASD closure; all are retrievable. Migration of the device is rare, with a reported rate of 0.4-1.1%. Device migration usually occurs during the first 24 hours after placement. The ASD device can migrate to: right or left atrium, main pulmonary artery (89%) or branch pulmonary arteries, left, left side of the heart (LVOT, aortic arch), and aorta. Embolization to the PA, LVOT, or aorta can be life threatening.

Selected References

- Wen B, et al. Emergent surgical removal of a migrated atrial septal defect occluder: case report. *J Cardiothorac Surg* 2020; 15:227
- Kallstrom E, et al. Migration of an Atrial Septal Occluder Device to the Transverse Aortic Arch Detected With Echocardiography. *Journal of Diagnostic Medical Sonography*. 2017;33:210-215
- Shirodkar S, et al. Successful retrieval of migrated Amplatzer septal occluder. *Ann Pediatr Cardiol*. 2010;3:83-6
- Nagre SW. Surgical removal of embolized atrial septal defect device from pulmonary artery. *J Thorac Cardiovasc Surg*. 2015;150:e55-7

Bivalirudin (Procedural Anticoagulation)

Related Cards: clot formation on cardiopulmonary bypass, HIT, failure to achieve adequate ACT

Indications for Bivalirudin Use

Cath Lab

- Heparin induced thrombocytopenia (HIT)
- h/o HIT (regardless of antibody status)
- Consider for: AT3 deficiency, heparin-refractory thrombosis

Cardiac Surgery with CPB

- HIT

PK/PD of Bivalirudin

Drug Class: Direct Thrombin Inhibitor

Co-Factors/Antagonists: None

Metabolism: Proteolytic Cleavage 80%, Renal 20%

Half-Life:

- Normal Renal Function – Mod. Dysfunction: 25-34 min
- Severe Renal Dysfunction: 57 min, Dialysis-Dependent: 3.5H
- Pediatric Patients: 15-18 min (age-dependent)

Monitoring: ACT, aPTT

Suggested Bivalirudin Dosing

Cath Lab

- Bolus: 0.75 mg/kg, additional 0.3 mg/kg if needed
- Infusion: 1.75 mg/kg/hr
- For STEMI: consider continuing infusion post-cath (up to 4H)

Cardiac Surgery with CPB

- Bolus: 1.5 mg/kg, additional 0.5-1 mg/kg if needed
- Infusion: 2.5 mg/kg/hr
- Pump Prime: 50mg

Monitoring

Cath Lab: ACT monitoring is controversial, best practice is to check ACT once 5 min after bolus/infusion to ensure anticoagulation effect.

CPB: ACT 2x baseline or > 400s

Safety Considerations

- Avoid stagnant blood (e.g. lines/sheaths, pleural cavities)
- Anticipate need for dosing up-titration in pediatric patients, during large-volume transfusion, and with continuous ultrafiltration on CPB
- Consider dosing infusion dosing reduction in > moderate renal dysfunction (CrCl < 30 mL/min, 1 mg/kg/h; patients who are dialysis-dependent, 0.25 mg/kg/h)
- Consider post-cath infusion in high-risk patients (e.g. STEMI, small and/or stented shunts, pulmonary vein interventions)
- Anticipate longer hemostasis times
- For CPB cases, anticipate and plan for post-CPB transfusion needs (no protamine, cannulas will come out regardless of ongoing transfusion need)
- Anticipate higher ACTs; avoid treating unless concern for bleeding outweighs risk of thrombosis
- ACT and aPTT are positively correlated with bivalirudin concentration but the dose-response relationship is non-linear and the correlation coefficients are poor, especially for non-standard patient populations
- Periprocedural ACT is unrelated to ischemic or hemorrhagic complications (in adults)

Please Note: FDA-Approved Indications - For use with aspirin in [adult] patients: 1. with unstable angina undergoing percutaneous transluminal angioplasty (PTCA), 2. Undergoing percutaneous coronary intervention (PCI) with provisional use of glycoprotein IIb/IIIa (GPI) as in the REPLACE-2 study, 3. With, or at risk of, heparin-induced thrombocytopenia (HIT) or heparin-induced thrombocytopenia and thrombosis syndrome (HITTS), undergoing PCI.

Selected References

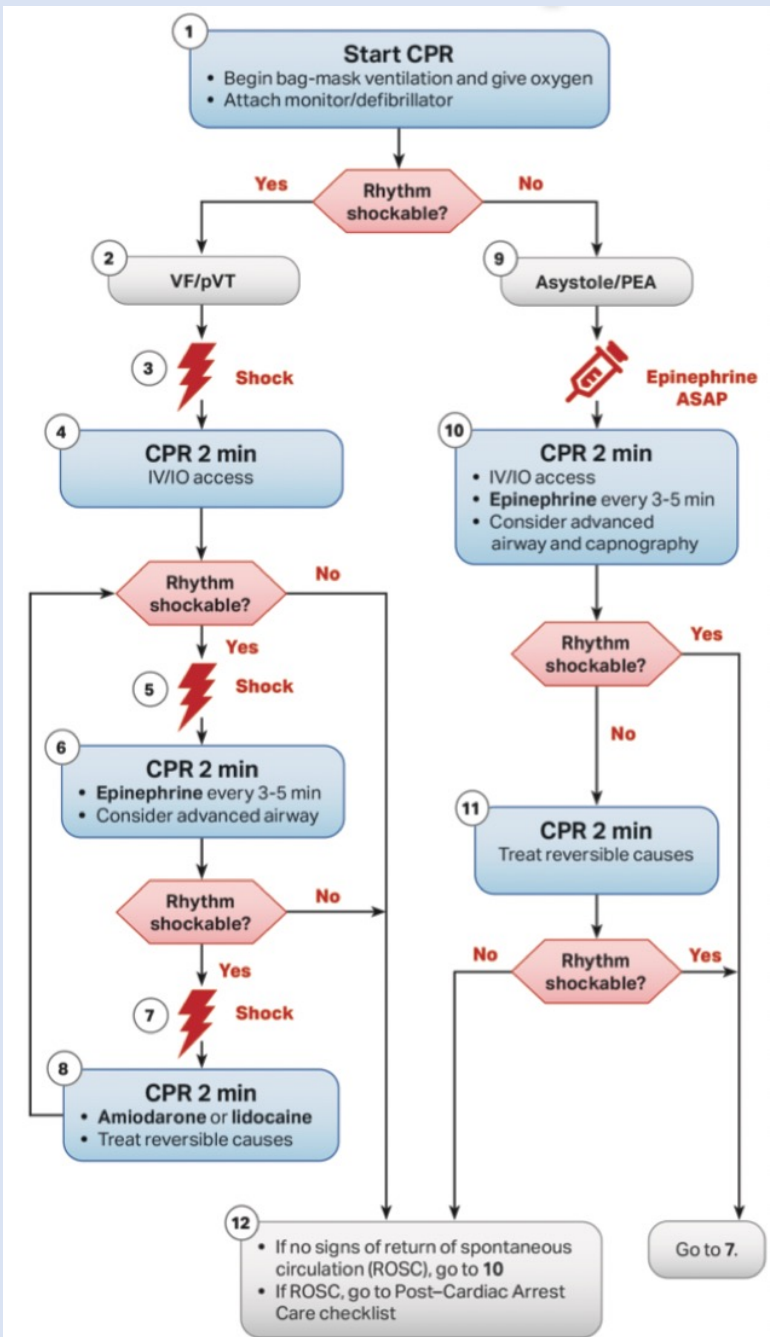
https://www.accessdata.fda.gov/drugsatfda_docs/label/2016/020873s036lbl.pdf

Zaleski KL, et al. Bivalirudin for Pediatric Procedural Anticoagulation: A Narrative Review. *Anesth Analg*. 2019;128:43-55

Van Cott EM, et al. Laboratory Monitoring of Parenteral Direct Thrombin Inhibitors. *Semin Thromb Hemost*. 2017;43:270-276

Cardiac Arrest in Cardiac Patients

Related cards: VA ECMO cannulation, junctional ectopic tachycardia



Immediate actions

- Call for Help, Call "code blue", activate ECMO team early
- Start CPR, call for board if on stretcher or bed to maximize CPR effectiveness, call for code cart
- Call for nitric oxide early if hypoxemic arrest
- Consider early chest exploration for patients immediate postop
- Call for echocardiography to help with diagnosis
- Call for blood if not present
- Assess for presence of pacemaker wires and call for pacer

Reversible causes – H's & T's

Hypovolemia (bleeding/other)	Tension pneumothorax
Hypoxia	Tamponade (cardiac)
Hydrogen ion (acidosis)	Toxins
Hypoglycemia	Thrombosis (pulmonary)
Hypo-/hyperkalemia	Thrombosis (coronary)
Hypothermia	

Special considerations for congenital heart disease

Shunted single ventricle patients

- Consider bolus of heparin if thrombus is suspected
- If severe hypotension, give volume, add additional inotropes

Single ventricle – Bidirectional Glenn or Fontan

- Adequate ventricular preload is dependent on PVR, consider volume resuscitation
- Consider respiratory acidosis (pH 7.3-7.35) to promote vasodilation and enhance flow through the Glenn

Pulmonary hypertension

- 100% FiO₂, inhaled nitric oxide, treat presumed acidosis with sodium bicarbonate
- Inotropes for RV support and also to avoid systemic hypotension which may cause RV ischemia
- Consider if adequate sedation, paralysis due to reactive pulmonary vasculature
- Consider use of vasopressin (?less pulmonary vasoconstriction)

Obstructed pulmonary venous return – TAPVC, MS, or PVS

- May require urgent cath or surgical procedures

Cardiopulmonary Bypass Pump Failure

Related cards: electrical failure on CPB

Immediate actions

- Notify team, Call for help, Say “Off bypass – bypass machine failure”

Treatments to consider

- Prepare for Separation from CPB
 - Resume ventilation
 - Inotropic agents as required
 - Transfuse blood and volume
- Work with perfusionist
 - Hand crank to manually perfuse, keep venous sats >70%
 - Obtain backup drive console
- If must separate from bypass, clamp arterial and venous CPB lines to prevent exsanguination or embolism

Other considerations

- Look for Reversible Causes
 - Check switches, AC plug
 - Check connections
 - Check clamps
 - Check roller pumps free
- Hand Crank Manual Perfusion
 - Maximum 15 min per operator
- Discuss abandoning procedure if possible

Signs

- Pump alarm, electrical failure, reservoir level rise, decreased RPM, hypotension, low NIRS, clot, pump noise

Summary

Sudden pump failure on CPB may be due to complete power loss, alarm-triggered pump stop, human error, or mechanical failures. The primary goals are prevention of exsanguination/embolism, continuation of manual perfusion or separation from bypass if conditions permit. If no immediate correctable cause can be found, a backup drive console should be obtained and utilized or separation from bypass should occur.

Selected References

Sadleir P, et al. The Perfusion Crisis Manual: Edition 1.0. Leeuwin Press, 2020

Delayed Emergence

Related cards: hyponatremia

Differential Diagnosis

- Hypothermia
- Neurologic
 - Stroke: Ischemic, hemorrhagic, or embolic
 - Seizure, status epilepticus, or post-ictal state
- Pharmacologic
 - Prolonged action of anesthetic agents or analgesics (i.e. opioids, benzodiazepines, propofol, volatile anesthetics)
 - Reduced drug metabolism/clearance due to liver failure, renal insufficiency or hypothyroidism
 - Drug interactions
 - Inadequate reversal of neuromuscular blockade
- Metabolic abnormalities
 - Hypoglycemia/hyperglycemia
 - Hyponatremia/hyponatremia
 - Hypocalcemia
 - Acidosis

Initial Workup and Management

- If currently intubated, keep endotracheal tube in place. If extubated, consider bag mask ventilation
- Ensure adequate blood pressure, heart rate/rhythm, and oxygen saturation
- Check patient temperature and use active warming devices if necessary
- Verify anesthetic agents have been discontinued
- Ensure adequate reversal of neuromuscular blockade by checking train-of-four and re-dosing reversal agents if indicated
- Review sedative medications administered during the anesthetic and possible dosing errors. Consider reversal of opioids and/or benzodiazepines if indicated (flumazenil, naloxone)

Next Steps in Management

- Conduct a focused neurological exam: Assess pupils, gaze preference, presence of spontaneous ventilation or movements, presence of brainstem reflexes (cough, gag)
- Check blood sugar and administer glucose or insulin as appropriate
- Draw an ABG to evaluate for hypocapnia, sodium abnormalities, or acidosis and correct appropriately
- If condition has not improved, notify the surgeon/proceduralist and discuss likelihood of possible neurologic event during the case.
 - Consider calling stroke alert and/or urgent neurology consult
 - Consider urgent head imaging – CT, MRI
 - In neonates, head ultrasound may also be used

Final Steps in Management

- Supportive care and transport to the ICU for further workup including continuous EEG, a complete metabolic profile and calcium level to evaluate for renal and liver function, and a thyroid function panel
- If related to an embolic stroke, there may be an opportunity for a interventional radiology intervention or pharmacologic thrombolysis – in these cases, this must be recognized as early as possible to ensure good neurologic recovery

Selected References

Cascella M, et al. Delayed Emergence from Anesthesia: What We Know and How We Act. *Local and regional anesthesia journal*. 2020;13:195-206

Tzabazis A, et al. Delayed emergence after anesthesia. *Journal of clinical anesthesia*. 2015;27:353–360

Alejandro A, et al. Chapter 55 - *Neurologic Disorders and Anesthesia*, Editor(s): Michael J. Aminoff, S. Andrew Josephson, *Aminoff's Neurology and General Medicine (Sixth Edition)*, Academic Press, 2021, 979-989.

Difficult Ventilation Post-CPB (Pulmonary Causes)

Related cards: hypoxia post CPB, hypercyanotic spells, pulmonary hypertensive crisis

Immediate actions:

Call for help, Notify team

Treatments to Consider

- If hypoxic
 - Increase FiO₂, manually ventilate, check ABG
 - Recruitment maneuvers to decrease atelectasis
 - Adding or altering PEEP strategy
- Check ventilator and ventilation settings using patient-machine
 - Assess for right mainstem intubation
 - Suction ETT for patency, blood
 - Look for kinking or dislodgement of ETT or circuit
 - Assess for circuit disconnections
- Bronchodilators – albuterol or epinephrine

Other considerations

- Fiberoptic bronchoscopy to confirm ETT position
- If adequate minute ventilation, but remains hypercarbic consider a CXR to look at lung fields; consider metabolic causes (e.g. malignant hyperthermia)
 - TRALI and/or TACO → pulmonary edema
 - Chest tube insertion may be required for large effusions and/or pneumothorax
- If adequate ventilation, but remains hypoxic, consider therapies to improve pulmonary blood flow (bronchodilators and/or pulmonary vasodilators (nitric oxide)) and also low mixed venous saturation (increase cardiac output and check hemoglobin levels)
- ICU ventilator or oscillator/JET ventilator in extreme cases

Signs

- Hypercapnea
- Cyanosis, hypoxia
- Poor chest rise, low/no lung movement
- Poor tidal volume despite high airway pressures

Summary

There are many etiologies for poor ventilation post-cardiopulmonary bypass. A systematic way to ensure nothing is overlooked is to group potential etiologies into equipment/mechanical, pulmonary, and thoracic. Equipment failures can occur with the ventilator, breathing circuit, and/or ETT. Some may be as simple as an unnoticed disconnection or kinking. Pulmonary complications can include atelectasis, bronchospasm, edema, and hemorrhage. Alveolar recruitment maneuvers, increased PEEP, and ETT suctioning are some first-line approaches. Thoracic etiologies to be addressed include effusions, pneumothorax, and diaphragm paralysis. Changes in respiratory compliance and PaCO₂ : ETCO₂ gradient changes should be monitored to assess improving or worsening ventilation.

Selected References

Badenes R, et al. Postoperative pulmonary dysfunction and mechanical ventilation in cardiac surgery. *Crit Care Res Pract.* 2015;2015:420-513

Bignami E, et al. Mechanical ventilation management during cardiothoracic surgery: an open challenge. *Ann Transl Med.* 2018;6:380

Electrical Failure During Cardiopulmonary Bypass

Related cards: CPB pump failure

Immediate actions:

Notify team, Call for help, Say "Bypass machine failure"

Treatments to consider

- Determine Extent of Power Failure
 - If widespread, use anesthesia power failure checklist
 - Consider stopping procedure if possible
- Prepare for Separation from CPB
 - Ventilation using anesthesia machine if power available, else hand ventilation using wall oxygen
 - Inotropic/vasoactive agents as required
- Obtain lights (flashlights)
 - Assure venous reservoir is visible
- Work with perfusionist
 - Check battery backup working
 - Minimize battery load
 - Consider hand-cranking suckers early
 - If have battery failure
 - Hand crank pump to venous sat >70%
 - Look for Reversible Causes
 - Check switches, AC plug, call engineering

Other considerations

- Loss of Warming/Cooling only
 - Portable generator to run heater/cooler
- Hand Crank Manual Perfusion
 - Maximum 15 min per operator

Signs

- Pump alarm, electrical failure, reservoir level rise, decreased RPM, hypotension, low NIRS, clot, pump noise

Summary

Electrical failure may be due to CPB console fault or failure of supply to the CPB machine wall socket. Priority is assuring pump operation via battery backup or hand-cranking. Separation from bypass should occur as soon as possible with ongoing electrical failure. The anesthesiologist will closely work with perfusionist and surgical team to assure adequate monitoring, assist with lighting, and optimize conditions for separation from bypass. If electrical failure is widespread, additional actions to address anesthesia machine/equipment failure are required.

Selected References

Sadleir, P. et al. *The Perfusion Crisis Manual: Edition 1.0.* Leeuwin Press, 2020

Troianos, C. Complete Electrical Failure during Cardiopulmonary Bypass. *Anesthesiology* 1995 (82):298-302

Heparin-Induced Thrombocytopenia (HIT)

Related cards: bivalrudin

Definition: antibodies to heparin-platelet factor 4 (PF4) complex, inducing platelet activation, thrombocytopenia, and venous and arterial thrombosis

Risks: treatment duration (longer ↑ risk), repeat exposures, unfractionated heparin > low-molecular weight heparin

Diagnosis:

4Ts	2 Points	1 Point	0 Points
Thrombocytopenia	>50% decline and platelet nadir $>20 \times 10^9/L$ and no surgery within the preceding 3 days	>50% platelet fall but surgery in last 3 days or Platelet fall that does not fit score 2 or 0	<30% platelet fall or Any platelet nadir $<10 \times 10^9/L$
Timing	Platelets fall 5-10 days after start of heparin or Platelet fall within 1 day of start of heparin and exposure to heparin within the past 5-30 days	Onset unclear or Platelet fall after day 10 or Platelet fall within <1 day of starting heparin (if exposed to heparin within the past 30-100 days)	Platelet fall within <4 days without exposure to heparin in the past 100 days
Thrombosis	Proven thrombosis, skin necrosis, or anaphylactoid reaction to systemic heparin bolus	Progressive, recurrent, or suspected venous thrombosis, or red skin lesions at heparin injection site	Thrombosis not suspected
Other Causes	None evident	Possible	Probable

Probability of HIT: Scores: 0-3 (low) 4-5 (intermediate) 6-8 (high)

Laboratory Studies -- Obtain if 4T score >3

- Serological assay (ELISA) - Detects antibody to PF4-heparin; highly sensitive, most rapid to result (~hours)
- Functional Assay (Serotonin Release Assay) - Detects PF4-heparin-IgG activation of platelets; gold standard, longer time to result (~days)

Treatments to consider

- Stop all heparin exposure (low-molecular weight heparin (LMWH), flushes, carrier solutions, heparin-coated catheters)
- Consider direct thrombin inhibitor (DTI) initiation, even if no current signs of thrombosis. Avoid Warfarin initiation.
- Hematology consult may be helpful
- Administration of platelets is not routinely recommended, and may precipitate thrombosis

Considerations for Cardiac Bypass and Catheterization

- Postpone surgical procedure during acute HIT episode if possible
- Use alternatives to heparin such as bivalirudin (see card)
- If HIT episode remote (>100 days) **AND** HIT antibody negative, consider heparin use for intraoperative setting only, with close monitoring of platelet count due to possibility of recurrence of HIT after additional heparin exposure

Selected references

Takemoto C et al. Heparin-Induced Thrombocytopenia Screening and Management in Pediatric Patients. Hematology AM Soc Hematol Educ Program 2011; 2011:162-9

Zaleski KL et al. Bivalirudin for Pediatric Procedural Anticoagulation: A Narrative Review. *Anesth Analg*. 2019;128:43–55

Alsoufi B, et al. Heparin-induced thrombocytopenia (HIT) in pediatric cardiac surgery: an emerging cause of morbidity and mortality. *Semin Thorac Cardiovasc Surg Pediatr Card Surg Annu*. 2004;7:155-71

Vakil NH, Kanaan AO, Donovan JL. Heparin-induced thrombocytopenia in the pediatric population: a review of current literature. *J Pediatr Pharmacol Ther*. 2012 Jan;17:12-30

Hypertension or Hypotension on CPB

Related cards: anaphylaxis, transfusion reaction

Immediate actions:

Notify team, Consider calling for help

Perfusion Pressure = MAP - CVP

Recommended CPB MAP Ranges Based on Age

- Neonate (< 1 month)
 - MAP 30 - 45 mmHg
- Child (1 – 10 years)
 - MAP 40 - 50 mmHg
- Adolescent (10 – 16 years)
 - MAP 50 – 70 mmHg
- Adult (> 16 years)
 - MAP 60 – 90 mmHg

Causes/Differential Diagnoses

- Inaccurate transducer height or transducer requires rezeroing
- Arterial line positional or dampened
- Hypertension
 - Arterial cannula malposition/kinking
 - Excessive flows on CPB
 - Inadequate anesthesia
 - Vasoactive agents infusing (increased SVR)
- Hypotension
 - Arterial cannula malposition
 - Aortic/Arterial dissection
 - Anaphylaxis
 - Bleeding, low hematocrit
 - Gas embolism (arterial/systemic/coronary)
 - Reversed cannulation
 - Cannula disconnection
 - Venous air lock
 - Inadequate venous drainage from patient
 - Collateral flow, shunting (PDA, AP collaterals)
 - Vasoplegia (decreased SVR)
 - Vasodilating agents infusing (Nitroglycerin)
 - High volatile agent/inappropriate anesthesia

Signs/Symptoms of Hyper- and Hypotension on CPB

- High or low MAPs displayed on monitoring
- Increasing lactate levels
- Change in NIRS from baseline levels

Treatments to Consider

- Check
 - Transducer height
 - Arterial line pressure on CPB
 - Aortic cannula position
 - Adequate venous drainage
 - Cannula for kinking
 - Check arterial inflow connected to arterial cannula
 - Medications Infusing (Vasoactive Agents vs. Vasodilators)
 - Hematocrit
- Actions
 - Re-zero the arterial line
 - Echo – TEE or epicardial to look at cannula position
 - Reposition aortic cannula
 - Increase/decrease anesthetic
 - Add vasoactive agent if hypotensive
Norepinephrine, Phenylephrine or Vasopressin
 - Add vasodilator if hypertensive
Nitroprusside, Hydralazine, Phentolamine
 - In hypotension, consider empiric steroids
 - Increase/decrease CPB flows
 - If unexplained, consider cooling more on CPB until source is found

Summary

- Monitor MAP, NIRS, lactates, and urine output on bypass
- Alert team with any concerning changes
- Communicate regarding medication administration

Selected References

Matte, GS. Perfusion for congenital heart surgery: Notes on cardiopulmonary bypass for a complex patient population.

Wiley Blackwell. 2015

Hypercyanotic Spells

Related cards: pulmonary hypertensive crisis, inadequate ventilation post CPB

Immediate actions:

Call for help, Notify team

Treatments to Consider

- Without IV Access
 - Calm child if agitated
 - Knees to chest position to increase SVR
 - Supplemental O₂
 - Sedation via intramuscular or intranasal routes
- With IV Access
 - In addition to above:
 - IV fluids, 10-20 ml/kg
 - Consider use of PRBC if cyanosis does not resolve
 - Intravenous sedation (midazolam, fentanyl, morphine)
 - Phenylephrine 1-10 mcg/kg IV
 - Beta-blocking agents (usually preventative therapy)
 - May require intubation with paralysis, ensure adequate ventilation

Other considerations

- May occur during prolonged fasting
- For patients with a secure airway, deepened sedation and/or muscle relaxation may be required
- Ensure that the cyanosis is not related to the airway
 - Right main stem intubation
 - Bronchospasm, laryngospasm, obstruction
 - Kinked or obstructed ETT
 - Pulmonary edema, hemothorax, pneumothorax
- Extreme hypercyanotic spells which do not reverse can result in a decision to intervene: prostaglandins and/or PDA stent in neonates, RVOT stent or emergency surgical repair, mechanical support such as ECMO

Signs

- Cyanosis (blue lips, gingivae, nail beds)
- Hypoxia (low SpO₂)
- Hyperpnea
- Agitation

Summary

Hypercyanotic spells are classically described in patients with tetralogy of Fallot (TOF) and are characterized by a paroxysm of hyperpnea, irritability or agitation, and prolonged crying, leading to worsening cyanosis. These can occur under sedation/anesthesia from catecholamine effects on the right ventricular outflow tract (RVOT). The pathophysiologic effects in TOF are largely determined by the degree of RVOT and not the VSD. Treatments are aimed at reversing/diminishing the right to left shunt by increasing SVR and alleviating dynamic RVOT obstruction.

Selected References

- Wise-Faberowski L, et al. Tetralogy of Fallot: Everything you wanted to know but were afraid to ask. *Pediatr Anesth*. 2019;29:475-482
- Sharkey AM, et al. Tetralogy of Fallot: Anatomic variants and their impact on surgical management. *Semin Cardiothorac Vasc Anesth*. 2012;16:88-96
- Tsze DS, et al. Treatment of tetralogy of Fallot hypoxic spell with intranasal fentanyl. *Pediatrics*. 2014;134:e266-269

Hyperkalemia

Related cards: cardiac arrest

Immediate actions:

Call for Help, Inform team, Consider calling ICU

Initial Diagnosis & Findings

- >6.5 mEq/L neonates/infants; >5.5 mEq/L children/adults
- Repeat and confirm labs
 - Consider arterial line to be able to sample without hemolysis
- Get EKG if feasible
 - Peaked T waves, shortened QT, ST segment depression

Treatments to Consider if non-bypass, or pre/post bypass

- Stop any potassium being administered (TPN, IV fluids, KCl, blood)
- Administer calcium gluconate 50-100 mg/kg (max 2 grams)
- Consider sodium bicarbonate (1-2 mEq/kg IV)
- Consider
 - IV insulin (0.1 – 0.2 U/kg) (max 10 units) AND
 - IV Dextrose (0.5g/kg) (max 50g)
- Consider albuterol
- Continue to monitor glucose hourly
- If PRBC required, use cell saver to remove potassium

Treatments to Consider on cardiopulmonary bypass

- Ultrafiltration may be used to removed potassium

Attempt to eliminate potassium

- Kayexalate – PO/PR 1g/kg
- Diuretics (furosemide 0.5-1mg/kg)
- Hemodialysis if refractory
- Call ICU for consult and consider taking to higher level of care for monitoring/treatment

Special Considerations: Digoxin Toxicity

- Digoxin blocks the sodium/potassium ATPase pump
- Digoxin toxicity can cause hyperkalemia
- Causes of digoxin toxicity:
 - Hypokalemia, hypermagnesemia, hypercalcemia
 - Overdose
 - Drug interactions: verapamil, macrolides, and antifungals
 - EKG may include VT/VF, progressive bradycardia or heart block, prolonged PR interval, downsloping ST depression in inferolateral leads on EKG
- Treatments:
 - Normalize potassium
 - Avoid calcium (possibility of myocardial injury)
 - Digibind /DigiFab (antibody)

Hyponatremia

Related cards: delayed emergence

Immediate actions:

ABCs if seizure

If preop: Consider delay of procedure to normalize sodium if possible in moderate or severe hyponatremia

If intraop: Consider treatments below

Hyponatremia defined as plasma sodium <134 mEq/L

Mild Na >125: usually asymptomatic

Moderate (Na 120-125) or gradual onset: confusion, muscle cramps, lethargy, anorexia, nausea

Severe (Na <120) or sudden onset: Seizures, coma

Risk of rapid correction: central pontine myelinolysis, cerebral edema, seizures

Treatments to Consider

- Recheck sodium, send serum sample for formal labs
- Review all fluids pt is receiving, including CPB prime
- Severe hyponatremia or neurologic symptoms:
 - Hypertonic saline 3% 1-2ml/kg/hr until Na>125
 - Loop diuretics (furosemide)
 - Sodium bicarbonate 0.5-1 mEq/kg boluses
- Avoid rapid overcorrection (0.5-1 mEq/hr, <6 mEq in 24 h)
- If SIADH – treat underlying cause
- If Addison's – treat with steroids
- If proceeding with urgent case and pt hyponatremic, discuss with the perfusionist: may require addition of free water or 5% dextrose to lower prime sodium concentration
- If contrast agents are to be administered (cardiac cath) consider frequent sampling to monitor for sodium

Serum Osmolality mOsm/kg H ₂ O		Causes			Treatment
High >295	Hypertonic hyponatremia	Severe hyperglycemia with dehydration, mannitol, radiocontrast administration			
Normal 280-295	Isotonic hyponatremia	Pseudohyponatremia: hyperlipidemia, hyperproteinemia			
Low <280	Hypotonic hyponatremia	Hypovolemic (↓ sodium & ↓ TBW)	Urinary sodium >20 mEq/L	Renal loss (diuretics, Addisons)	Isotonic saline
			Urinary sodium <20 mEq/L	Extra renal loss (Vomiting, diarrhea, 3 rd spacing)	Isotonic saline
		Hypervolemic (↑ TBW)	Urinary sodium >20 mEq/L	Renal failure	Fluid & sodium restriction
			Urinary sodium <20 mEq/L	Heart failure, cirrhosis, nephrosis, hypoalbuminemia	Diuresis, fluid & sodium restriction
		Euvolemic	Urinary osmolality >100mOsm/kg	SIADH, hypothyroidism, adrenal insufficiency, stress, amphetamines	Fluid restriction & tx of underlying cause
			Urinary osmolality <100 mOsm/kg	Primary polydipsia, low solute intake	Fluid restriction

Hypoxemia on Cardiopulmonary Bypass

Related cards: electrical failure on CPB, cardiopulmonary bypass pump failure

Immediate actions:

Notify team, Call for help

Treatments to consider

- Confirm blood gas analyzer with arterial blood gas measurement if possible or compare to arterial & venous circuit color; examine pulse oximetry and NIRS values
- Begin ventilation using anesthetic machine and 100% FiO₂
- If heart is beating, consider attempting to come off bypass
- Work with perfusionist
 - Confirm the correct gas source (oxygen) and blender settings if in use
 - Confirm function of flow meter
 - Ensure adequate arterial blood flow & arterial pressure
 - Obtain additional E cylinder of oxygen
 - Possibility of oxygenator failure --> need additional perfusion help to obtain replacement oxygenator. If required discuss with team coming off CPB vs. cooling for circulatory arrest to exchange oxygenator (+/- venous reservoir)

Other considerations

- Consider unusual diagnoses
 - Malignant hyperthermia
 - Anaphylaxis

Signs

- Pump alarm, electrical failure, reservoir level rise, decreased RPM, hypotension, low NIRS, clot, pump noise

Summary

Sudden pump failure on CPB may be due to complete power loss, alarm-triggered pump stop, human error, or mechanical failures. The primary goals are prevention of exsanguination/embolism, continuation of manual perfusion or separation from bypass if conditions permit. If no immediate correctable cause can be found, a backup drive console should be obtained and utilized or separation from bypass should occur.

Selected References

Sadleir P, et al. The Perfusion Crisis Manual: Edition 1.0. Leeuwin Press, 2020

Junctional Ectopic Tachycardia

Related cards: cardiac arrest

Immediate Actions:

Notify team

Consider differential diagnosis of supraventricular tachycardias

Signs

- Tachyarrhythmia with HR generally > 170 BPM
- Narrow QRS complex (or QRS complex similar to patient's baseline sinus rhythm)
- Atrioventricular dissociation, with ventricular rate faster than atrial rate

Treatments to Consider

- Confirm diagnosis with adenosine or EKG – may be difficult in the operating room
- Cool patient to low normothermia (35.5-36 C)
- Consider additional sedation
- Minimize inotropic infusions as much as possible
- Electrical cardioversion or adenosine usually ineffective, as there is no AV nodal reentry; it may address other SVTs
- Medication treatments
 - Check and correct any electrolyte disturbances
 - Magnesium repletion (goal 4-4.5 mmol/L)
 - Consider acetaminophen IV bolus to aid cooling
 - Dexmedetomidine can be attempted
 - Consider early EP involvement to guide antiarrhythmic choices which may include:
 - Common: Amiodarone, procainamide
 - Less common: propafenone, esmolol, propranolol, sotalol, ivabradine
- Attempt to overdrive pace tachyarrhythmia (atrio-ventricular)
- If refractory and unstable arrhythmia, consider ECMO
- Even if JET resolves, consider leaving chest open +/- tourniquets if patient had been unstable with arrhythmia

Other considerations

- Perioperative JET is often self-terminating within 72 hours of surgery.
- Perioperative JET is separate phenomenon from congenital JET.

Pathophysiology

Focal ectopic tachyarrhythmia with abnormal automaticity (not a re-entry circuit) of cardiac conduction system tissue in the AV node or proximal bundle of His, attributed to direct or ischemic injury to, traction on, or swelling of the AV node or interventricular septum.

Preoperative and Surgical Risk Factors

- Young age (< 6 months)
- Longer duration of CPB and aortic cross clamping
- Surgeries that affect the proximal ventricular conduction system – VSD, TOF, CAVC

Intraoperative Risk Factors

- Hyperthermia
- Elevated endogenous or exogenous catecholamines
 - High dose dopamine and epinephrine gtts
- Electrolyte abnormalities (especially hypomagnesemia)

Summary

Perioperative junctional ectopic tachycardia (JET) is the most common tachyarrhythmia following congenital cardiac surgery. JET carries significantly increased risk of perioperative major morbidity and mortality. JET is a focal ectopic tachyarrhythmia due to abnormal automaticity of AV nodal or proximal ventricular conduction system tissue, which is attributed to traction on, edema of, and/or direct surgical injury to these tissues. Therapeutic interventions target low normothermia, minimized exogenous catecholamines, serum electrolyte correction (especially magnesium), and antiarrhythmics with AV pacing.

Malignant Hyperthermia

Related cards: sepsis, protamine reaction, air embolus, blood transfusion reaction, anaphylaxis

Immediate actions

Call for help, Notify team, Obtain MH cart

Treatments to Consider

- Stop volatile vapor/succinylcholine administration
 - Stop volatile anesthetic administration on CPB circuit; remove vaporizer from CPB circuit
- Switch to TIVA
- Place charcoal filter in anesthesia circuit. Turn O2 flow to 10 L/min; Decontaminate CPB oxygenator with 100% FIO2 10L/min
- Give dantrolene (post synaptic muscle relaxant)
 - 2.5 mg/kg IV every 5 minutes until symptoms resolve
 - Dose same if patient is on the CPBP circuit. If no response to 10 mg/kg Dantrolene, consider other diagnoses
 - Dantrolone/Revonto: Assign dedicated personnel to mix formulation of dantrolene (20 mg/vial) with 60 mL of sterile water (0.33mg/cc)
 - Ryanodex: 250 mg is mixed with 5 mL sterile water (50mg/cc)
- Cool Patient
 - Consider cooling on CPBP circuit to 35-36 C as a temporizing measure if extreme hemodynamic instability occurs.
 - Apply ice externally
 - Address the rising PaCO2/ respiratory acidosis
 - Increase sweep if still on CPB; Hyperventilate if off CPBP
- **MHAUS # 1-800-644-9737**

Other considerations

- Metabolic acidosis: sodium bicarbonate 1-2 mEq/kg IV
- Hyperkalemia
 - Calcium gluconate 30 mg/kg or calcium chloride 10 mg/kg IV
 - Sodium Bicarbonate 1-2 mEq/kg IV
 - Regular insulin 0.1 units/kg IV (max 10 units) and dextrose 0.5-1g/kg IV
 - If on CPB: ultrafiltration to remove potassium
- VT or Afib
 - Do not use calcium channel blocker
 - Amiodarone 5 mg/kg
- Cardiac Arrest
 - Begin CPR or cardiac massage; consider resuming CPB or transitioning to ECMO
- Myoglobinuria
 - Maintain UOP >2 ml/kg/hr; Furosemide 0.5- 1 mg/kg

Signs

- ↑ HR, ↑ PaCo₂ with ↑ sweep on CPBP circuit
- ↑ lactate, metabolic acidosis, ↓ MVO₂. Hyperkalemia
- Hyperthermia after termination of CPBP
- Rhabdomyolysis, ↑ CK, myoglobinuria, skeletal muscle rigidity, DIC
- Hemodynamic instability that rapidly resolves after Dantrolene
- Differential Diagnosis: Sepsis, NMS, serotonin, syndrome, myopathy, pheochromocytoma

Summary

Potentially lethal hypermetabolic syndrome that is triggered after susceptible patients receive a volatile anesthetic and/or succinylcholine. Administration of triggering substances leads to a surge of calcium release from the sarcoplasmic reticulum due to the presence of an abnormal ryanodine receptor. This causes a subsequent dramatic increase in oxygen consumption, carbon dioxide production, massive release of muscle enzymes, and lactic acidosis. Medical management is cessation of the trigger, administration of dantrolene and resultant symptomatic medical management.

NIRS Abnormal on CPB

Related cards: aortic dissection, sepsis, hypertension or hypotension on CPB

Immediate actions

Notify team, Consider calling for help

Treatments to Consider

- Discuss with surgeon – visually inspect the field
- Discussion with perfusion – adequacy of CPB run (flows, mixed venous, hematocrit, line pressures/venous drainage)
- Check
 - Position and adherence of sensors, cables and connections
 - Head position and characteristics of head (color, swelling, fontanelle)
 - Cannula position (visually or TEE)
 - Appropriate placement of clamps, tourniquets
 - Hemoglobin, PaO₂, PaCO₂, blood pressure
- Possible actions if NIRS low
 - Increase MAP
 - Increase arterial pump flow rate
 - Increase PaO₂
 - Increase PaCO₂ > 45
 - Increase anesthetic depth
 - Transfuse PRBCs
 - Greater hypothermia

Other considerations

- Unilateral decrease NIRS
 - More likely to be related to mechanical cause
 - Head position or cannula position
 - Seizure/stroke
- Bilateral decrease in NIRS
 - Systemic issues – hemoglobin, blood pressure, etc.

Causes

- Mechanical obstruction of venous return
 - Position of venous cannula
 - Neck positioning
- Hemodilution, anemia, abrupt blood loss
- Hypotension
- Hypoxia or hypocarbia
- Increased metabolic demand (seizure)
- If measuring bilateral NIRS and one side drops, may have incorrect position of aortic cannula causing streaming of blood flow away from vessels on that side (can also occur when doing regional cerebral perfusion)

Summary

Regional cerebral saturation as measured by near infrared spectroscopy (NIRS) is used to continuously monitor cerebral perfusion and oxygen delivery during cardiac surgery.

Responding quickly to reverse abrupt decreases in NIRS during CPB may help restore optimal cerebral perfusion and oxygen delivery and in turn, prevent neurologic morbidity and long-term adverse cognitive issues in children.

Selected References

Williams GD, et al. Brain monitoring and protection during pediatric cardiac surgery. *Seminars in Cardiothorac Vasc Anesth.* 2007;11:23-33

Tobias JD. Cerebral oxygenation monitoring: near-infrared spectroscopy. *Expert Rev Med Devices.* 2006;3:235-43

Kussman BD, et al. An evaluation of bilateral monitoring of cerebral oxygen saturation during pediatric cardiac surgery *Anesth Analg.* 2005;101:1294-1300

Andropoulos DB, et al. Neurological monitoring for congenital heart surgery. *Anesth Analg.* 2004;99:1365-75

Pulmonary Hypertensive Crisis

Related cards: hypercyanotic spells, inadequate ventilation post CPB

Immediate actions:

Call for help, Notify team

Treatments to Consider

- Stop procedure and/or stimulation if at all possible
- Increase FiO_2
- Inhaled nitric oxide, inhaled/intravenous prostanoids
- Give additional sedation (opioids or benzodiazepines), consider muscle relaxants
- Correct hypercarbia
- Administer sodium bicarbonate if metabolic acidosis
- Inotropes/vasopressors:
 - Epinephrine/dopamine may improve RV function but excessive tachycardia may be detrimental
 - Norepinephrine may improve SVR but may also raise PVR
 - Vasopressin may improve SVR, possibly lower PVR
 - Milrinone may lower PVR but hypotension possible
- Consideration for other supportive strategies such as ECMO

Other considerations

- Poor neurologic outcomes with CPR and ECMO in pulmonary hypertension
- Avoid precipitants of increased PVR
 - Hypoxia
 - Hypercarbia
 - Cold
 - Acidosis
 - Light anesthesia/pain

Signs

- Cyanosis (blue lips, gingivae, nail beds)
- Hypoxia (low SpO₂)
- Agitation
- Hypotension
- Bradycardia and/or cardiac arrest

Summary

The right ventricle cannot handle acute increases in pulmonary artery pressure or pulmonary vascular resistance (PVR), and therefore acute pulmonary hypertensive crises can lead to circulatory collapse requiring mechanical circulatory support. Immediate efforts should be made to reduce PVR, remove any inciting stimuli, and provide cardiocirculatory support. Acute therapies should be weaned while instituting long-term therapies, as abrupt cessation of pulmonary vasodilatory therapy may result in rebound pulmonary hypertensive events.

Selected References

- Abman SH, et al. Pediatric Pulmonary Hypertension: Guidelines From the American Heart Association and American Thoracic Society. *Circulation*. 2015;132(21):2037-99
- Kaestner M, et al. Pulmonary hypertension in the intensive care unit. *Heart* 2016; 102 Suppl 2:ii57-66
- Bernier ML, et al. Spectrum of Current Management of Pediatric Pulmonary Hypertensive Crisis. *Crit Care Explor*. 2019;1(8):e0037

Reoperative Sternotomy

Review of previous operative notes, CXR, or CT scan	Previously placed bypass grafts, RV to PA conduit, aortopexy
	Prior injury to cardiac and extracardiac structures
	Prior gortex or other membrane placed
Level of risk*	Aorta or conduit adherence or erosion into the sternum
	Hypertensive, dilated RV abutting sternum
	Dilated or aneurysmal aorta or coronary
Identify structures at risk for injury	Innominate vein (especially if pacing leads present)
	Dilated RV (PHTN)
	RA, PA
	Aorta, coronary arteries
Evaluation of vessel patency (Ultrasound, cath or CT scan)	Internal jugular vein
	Axillary artery
	Femoral or iliac vein
	Femoral or iliac artery
Anesthesia preparation#	Large bore IV, RIC or sheath, rapid infuser (Belmont or Level I) for >40kg, pressure bag, arterial line, CVL
	Blood in the room before incision
	TEE in place
	Cell saver available, perfusionist in room
	Inotropes & vasopressors primed and ready to start before incision
	Availability of ice (e.g. need for circulatory arrest)
	Neuro monitors (cerebral oximetry ± EEG)
	Notify blood bank of a potential need for large volume PRBC or massive transfusion protocol activation (larger patients), cell saver available

Potential Initial Cannulation strategies	Peripheral cannulation and CPB initiation before reentry
	Cutdown on vessels before reentry
	Wire placement before reentry
Possible Sites of Cannulation	Femoral artery/vein, carotid artery/internal jugular, innominate artery

Venous or right/pulmonary heart injury management

Surgery	Packing, manual compression of the chest
Anesthesia	Volume replacement
	Follow gases closely transfuse if needed
	Check TEE and rule out the air in the left heart

Arterial or systemic/left heart injury management

Surgery	Cannulate and initiate CPB for decompression
	Need for deep hypothermia & circulatory arrest
	May require LV drain or systemic potassium if fibrillates and ventricle distends
Anesthesia	Call for help
	Obtain additional PRBC (matched PRBC or emergency release or massive transfusion activation – based on local practice). Use cell saver blood as able.
	Transfuse as needed via rapid infuser
	Start vasopressor & inotropes to support BP
	Place ice on head if circulatory arrest planned
	Assess for risk of air embolism (see card)

* Based on CTA findings but confirm with surgeon

‡ Discuss with surgeon appropriate A-line and CVP, including potential CPB cannulation sites

Avoid unnecessary personnel in high-risk sternotomy cases, and always keep a quiet environment to facilitate communication. Two anesthesia practitioners are needed in this case until sternotomy is achieved safely.

Abbreviations: RV, right ventricle; RA, right atrium; PHTN, pulmonary hypertension; PA, pulmonary artery; TEE, transesophageal echocardiography; BP, blood pressure.

Selected References

Pahwa S, et al. High-Risk Reoperative Sternotomy-How We Do It, How We Teach It. *World J Pediatr Congenit Heart Surg.* 2020;11:459-465

Morales DL, et al. Repeat sternotomy in congenital heart surgery: no longer a risk factor. *Ann Thorac Surg.* 2008;86:897-902

Mangukia CV, et al. Management of iatrogenic RV injury - RV packing and CPB through PTFE graft attached to femoral artery. *Ann Pediatr Cardiol.* 2015;8:53-5

ROTEM

Related cards: massive transfusion

Indications for ROTEM use

- Evaluate coagulopathy in a bleeding patient
- Guide transfusion strategies in trauma, major surgery, obstetric hemorrhage, and massive transfusion

ROTEM Parameters

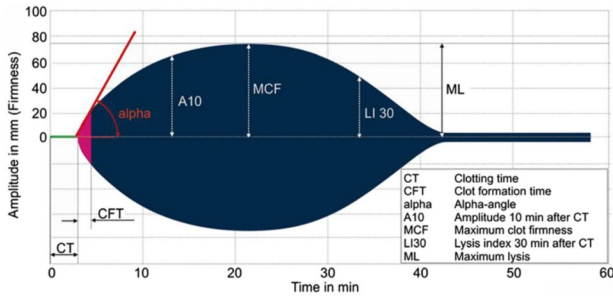


Image adapted from Tanaka et al (2012)

Interpretation of ROTEM

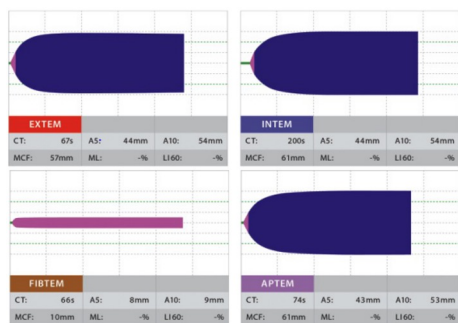
Parameter	Definition	Problem (if abnormal)	Treatment
CT	Time to initiation of clotting to 2mm	Coagulation factors	CT Prolonged (EXTEM, INTEM): consider FFP
CFT	Time of clotting from 2mm to 20mm	Platelets and/or fibrinogen	MCF Low (EXTEM, INTEM): Also low FIBTEM MCF: consider cryo or fibrinogen concentrate Normal FIBTEM MCF: consider platelets
A10, A20	Clot firmness at 10min, 20min		
MCF	Maximum clot amplitude during test		
LI30	Ratio of amplitude/MCF, lysis index at 30 min	Hyperfibrinolysis	ML >15% (EXTEM, INTEM): consider TXA
ML	Maximum lysis		

Limitations of ROTEM

- Does not account for thrombocytopenia or platelet dysfunction which must be separately tested (e.g. antiplatelet drugs or von Willebrand disease)
- Normal ranges vary slightly by age. One study found prolonged INTEM CT, shorter EXTEM CT, and overall shorter CFT, greater A10, and greater MCF in patients age <3 months compared to older children (up to 16 years)

Functions of ROTEM assays

- EXTEM: Assess clot dynamics through activation of extrinsic pathway
- INTEM: Assess influence of heparin and clot dynamics through activation of intrinsic pathway
- FIBTEM: Assess fibrinogen status and fibrin polymerization after blocking platelet contribution
- HEPTTEM: Assess intrinsic pathway after neutralizing influence of heparin
- APTEM: Assess clot stability after inhibiting fibrinolysis



Normal ROTEM

Image from Deaton, Ben

[https://www.nmthoracic.org/components/com_rseventspro/assets/images/files/DEATON-ROTEM%20in%20Bleeding%20Patient.pdf]

Summary

Use ROTEM in an actively bleeding patient to guide planned transfusion with rapid (<20 minute) results. ROTEM should be supplemented by platelet function tests for comprehensive interpretation. ROTEM guided transfusion has been shown to reduce mortality, morbidity, and transfusion requirements when compared to conventional coagulation assays.

Selected References

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Shunt Dependent Single Ventricle for Noncardiac Surgery

Related cards: Superior cavopulmonary shunt (Glenn) for noncardiac surgery

Immediate actions

Call for help, Notify team

Ask surgeon to stop

If does not resolve, call a code, echo, ECMO

Acute Intraoperative Hypotension – Treatments to Consider

- Check for rhythm /ST changes; if not in NSR, consider calling EP
- Consider IV fluid administration
- Inotropes may be necessary – epinephrine, dopamine, calcium
- Check ABG/VBG for anemia and acidosis – treat if found
- Consider that may have excessive pulmonary blood flow – lower oxygen to keep saturations >80% and avoid hypoxemia
- Echocardiography for refractory hypotension
- Consider other causes of hypotension
 - Excessive sedation, anaphylaxis, drug error, bleeding

Acute Intraoperative Hypoxemia - Treatments to Consider

- Check machine to patient including:
 - Put on 100% FiO₂, check vent & connections, suction ETT, listen for breath sounds, consider FOB or CXR to help diagnosis
- Check blood pressure, rhythm (If not NSR, call EP)
- Consider giving bolus of inotropic support (epi or calcium)
- Auscultate the chest for the presence of a shunt murmur. If none, try to evaluate with echocardiography. If profound desaturation, give empiric heparin bolus (~75-100units/kg). If there has been surgical blood loss, consider giving blood. If not, consider crystalloid or albumin.
- Perform maneuvers to ensure PVR low – increase FiO₂, moderate hyperventilation, check for acidosis and treat, consider adding inhaled nitric oxide

Physiologic goals

Target saturations are 75-85%

MAPs >40-60 mmHg

Description of shunted single ventricle physiology

Blood flow

- Systemic and pulmonary venous return mix in the common atrium
- Blood flows through the atrioventricular valve
- Single systemic ventricle pumps blood into the neo-aorta, then systemically
- Pulmonary blood flow is provided by a systemic-pulmonary shunt (e.g. a Blalock-Taussig (BT) or other central shunt), a right ventricular to pulmonary artery conduit (Sano) or a PDA stent

Specific Surgical Considerations

Laparoscopic surgery:

- Pneumoperitoneum has several physiological effects
 - Carbon dioxide absorption (increases PVR)
 - Ventilation impairment leading to PVR increase from increased basal atelectasis
 - Increased afterload
 - Decreased preload from reduction in IVC venous return resulting in hypotension
 - Increased heart rate
- Limit insufflation pressures (ideally less than 10mmHg) and total time; consider arterial line to monitor
- Careful attention to oxygenation and ventilation

Most noncardiac surgery should be delayed or deferred in patients with shunt dependent single ventricle physiology unless absolutely necessary. Consideration for a postoperative intensive care unit bed should be made for recovery following any anesthetic. For any major procedure, additional monitoring including arterial lines and/or NIRS should be

Superior Cavopulmonary Shunt (Glenn) for NonCardiac Surgery

Related cards: Stage I palliation for noncardiac surgery

Immediate actions

Call for help, Notify team

Ask surgeon to stop and deflate any CO2 if laparoscopic
If does not resolve, call a code, echo, and ECMO

Acute Intraoperative Hypotension - Treatments to Consider

- Check for rhythm; if not in NSR, consider calling EP
- IV fluid administration (crystalloids are appropriate) to maintain adequate preload
- Inotropes may be necessary
- Check ABG/VBG for anemia
- Echocardiography for refractory hypotension
- Consider other causes of hypotension
 - Excessive sedation, anaphylaxis, drug error, bleeding

Acute Intraoperative Hypoxemia - Treatments to Consider

- Stop the procedure, call for help
- Assess ventilation
 - Consider CXR and/or using fiberoptic bronchoscopy
 - ETT displacement (?right mainstem), obstruction, pneumothorax, equipment (ventilator) failure, others
- Lower the PVR
 - Optimize ventilation
 - Check ABG or VBG for CO2 levels
 - Consider nitric oxide to further lower PVR
- Assess the cardiac output
 - Consider additional fluid, inotropes
 - Echocardiography – function, effusion, thrombus
- Check for anemia
 - Check ABG or VBG
 - If no other causes found, consider transfusion to achieve Hct >40

Physiologic Goals

- Target Saturations should be 75-85%
- Low-normal pulmonary vascular resistance (PBF is passive)
- Maintain preload, NSR, normal/low normal SVR

Description

Stage 2 palliation of single ventricle (also known as Bidirectional Glenn (BDG)) involves the creation superior cavopulmonary anastomosis to provide pulmonary blood flow
Path of blood flow

- The single ventricle ejects blood systemically through the neo-aorta
- Venous blood from head, neck and arms is routed through the pulmonary artery via the superior cavopulmonary anastomosis
- Oxygenated blood returns via the pulmonary veins to the common atrium
- Venous blood from the lower body drains via the inferior vena cava (IVC) to the common atrium to mix with oxygenated blood returning from the lungs

Specific Surgical Considerations

- Consider having lower body venous access if bleeding anticipated

Laparoscopic surgery:

- Pneumoperitoneum has several physiological effects
 - Carbon dioxide absorption (increases PVR)
 - Ventilation impairment leading to PVR increase from increased basal atelectasis
 - Increased afterload
 - Decreased preload from reduction in IVC venous return resulting in hypotension
 - Increased heart rate
- Limit insufflation pressures (ideally less than 10mmHg) and total time; consider arterial line to monitor
- Careful attention to oxygenation and ventilation

Neurosurgery:

- High venous pressures (due to SVC connection to PA), predisposes patients to increased bleeding risk

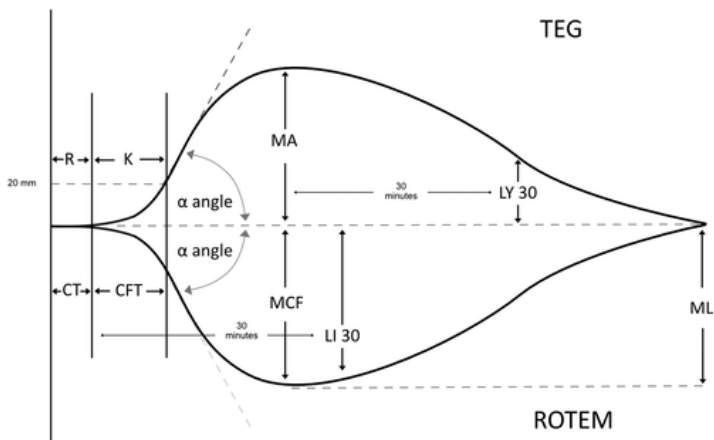
Thromboelastography (TEG)

Related cards: ROTEM, massive transfusion

TEG measures the relative impedance to the rotational movement of a pin in a cup due to clot formation and breakdown. It is a point of care, functional assay to assess coagulopathy.

Indications for TEG

- Evaluate coagulopathy in a bleeding patient
- Guide transfusion strategies in trauma, major surgery (cardiac surgery), obstetric hemorrhage, and massive transfusion



Consider TEG/ROTEM to guide management	Treatment options	Pattern recognition
R-Time/CT (Thrombin building)	If R-Time/CT prolonged → plasma (or PCC)	
MA/MCF (Clot firmness)	If MA/MCF is low → platelets or fibrinogen (cryoppt)	
LY30/ML (Clot lysis)	If LY30 is >3%/ ML >15% (hyperfibrinolysis) → TXA	
Normal		

	Boston Reference Range	Definition	Diagnosis	Treatment
Reaction Time (min)	4-9	Time to initiation of fibrin clot formation	Decreased clotting factors	FFP, protamine
Clot (K) Time (min)	1-3	Time to initiation of clotting to 2mm	Coagulation factors	FFP
Alpha Angle (degrees)	60-78	Rate at which fibrin cross-linking occurs	Decreased fibrinogen	Cryo, fibrinogen
Maximum Amplitude (mm)	52-69	Maximum strength of clot	Decreased platelet count or function vs. fibrinogen	Platelets +/- additional testing to distinguish cause (platelet count, INR,
Ly-30	0-10%	Degradation of clot 30 min after MA	Increased clot breakdown	Antifibrinolytics

Limitations of TEG

- Does not account for thrombocytopenia or platelet dysfunction which must be separately tested (e.g. antiplatelet drugs or von Willebrand disease)
- Normal ranges vary slightly by age
- Data for certain subpopulations including pediatric patients requiring ECMO or VADs remains limited

Selected References

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- Whiting D, Dinardo JA. TEG and ROTEM: Technology and clinical applications. *Am J Hematol.* 2014;89: 228–232

Transfusion Reaction

Related cards: hyper/hypotensive on CPB, anaphylaxis

Immediate actions:

Stop Transfusion, Verify Unit Number/Patient, Notify team & Blood Bank

Types of acute transfusion reactions

- Acute hemolytic
 - Timing: immediate
 - Cause: ABO incompatibility, clerical error
- Allergic/Anaphylactic
 - Timing: < 30 minutes
 - Cause: IgA deficient patient, recipient IgE antibodies vs. donor proteins
- TRALI (transfusion related acute lung injury)
 - Timing: immediate to hours
 - Cause: donor antibodies vs. recipient HLA antigens in the lung tissue, particularly plasma-containing components
- TACO (transfusion associated circulation overload)
 - Timing: < 12 hours
 - Cause: Very rapid and/or large volume transfusion
- Febrile
 - Timing: < 30 – 90 minutes
 - Cause: donor cytokine release
- Infectious/Transfusion associated sepsis
 - Timing: Within 60 minutes
 - Cause: bacterial or viral contamination

Signs/Symptoms

- Hypotension
- Hypertension (TACO)
- Tachycardia, Fever, Rash
- Coagulopathy, Bleeding, DIC
- Hemolysis, Drop in Hemoglobin Level
- Hemoglobinuria
- Pulmonary Edema (TRALI, TACO), hypoxemia
- Rash

Treatments to Consider

- Check
 - Unit number/confirm patient
 - ABO compatibility, Rh status
 - Order coagulation studies
- Actions
 - Stop transfusion
 - Support BP and vitals (volume, vasopressors)
 - Promote urine output with diuretics
 - Urine alkalinization (sodium bicarbonate)
 - Manage coagulopathy
 - Diphenhydramine (allergic reactions)

Consequences

- Intravascular hemolysis, coagulopathy, DIC
- End organ damage, renal failure
- Death

Summary

- Ensure products are checked prior to transfusion
- Use leukocyte-reduced products
- Use washed products if available
- If unwashed PRBC are not available, consider use of cell saver to wash PRBC
- Be cautious with rapid or large volume transfusion

Selected References

- Bandarenko N. Chapter 7: Adverse effects of blood transfusion. From Bandarenko N, ed. Blood transfusion therapy: a physician's handbook. 12th Ed. AABB; 2017:221-256
- Lich BV, et al. The Manual of Clinical Perfusion. 2nd Ed. Perfusion.com Inc; 2006
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Veno-Arterial ECMO Cannulation

Related cards: cardiac arrest

	Peripheral	Central
Efficient	>2 weeks postop Prior sternotomy	Open chest Sternotomy <2 weeks
Advantages	Fewer resources (people, equipment)	Control PDA Placement of LV vent
Risks	Vagus nerve Tracheal injury Loss of control of vessels into chest Inadequate flows	Bleeding Sepsis

Peripheral Cannulation:

- Need to know patency of peripheral vessels
- Usually >20kg femoral <20 kg neck
- Neck – need to think of contralateral vein – is it obstructed?
If so, need to decompress cephalad on cannulated side – especially in small kids
- Femoral – consider leg viability & consider distal arterial perfusion cannula; vein is usually okay unless clinical evidence of venous obstruction (compartment syndromes)

For critically unwell patients in which the chest is left open, tourniquets may be left at central cannulation sites to facilitate urgent ECMO cannulation if required later

Anesthetic Issues

- If need CPR, ensure minimal interruptions to compressions
- Manage airway with ETT if possible, EtCO₂ monitoring
- Anticoagulation:
 - Heparin ALWAYS in the ECMO circuit
 - May give patient heparin or not based on situation
 - Heparin bolus: 50-100 Units/kg
- Sedation and/or paralytics as necessary
- Consider decreasing or stopping pressors/inotropes once cannulated to ECMO due to risk of hypertension once achieve full flows
- Advise a TIME OUT prior to initiation of ECMO to ensure appropriate connections are made (A-A, V-V), heparin administered

Specific issues with congenital heart disease

Aortic insufficiency

- Can cause myocardial distention, LV/LA vent may result in circular shunt

Aortopulmonary shunts – PDA, PDA stents, BTS, MAPCAs

- Can result in pulmonary congestion
- Can be modulated with ECMO flows – inotropes can be used to keep heart ejecting, but may need to control surgically; risk of PA thrombosis if no flows to the lungs

Mustard, Senning, Fontan – central cannulation

- Single cannula may drain well from IVC
- Cannula in the baffle may obstruct and not drain well

Glenn

- RA cannula will decompress heart
- Need to decompress head with SVC cannulation
- Neck cannulation – need to be careful to not injure the PAs

Stage I

- Don't need to control the Sano

Consider venting when no LV ejection, distended LV or congested lungs

Options: creation of BAS in cath lab or LV/LA vent (prone to clotting/dislodgement)

Vasoplegia

Related cards: sepsis, anaphylaxis

Immediate actions

Notify team, Consider differential diagnosis for arterial hypotension on CPB

Treatments to Consider

- Ensure arterial line is working properly – rezero, assess for patency, check central aortic pressures if unsure
- If on CPB, check for status regarding flows, venous saturation, hematocrit, and medications given (phenolamine)
- Goal-directed fluid and blood product resuscitation, consider increasing hct to increase SVR if borderline
- Phenylephrine or norepinephrine infusion +/- vasopressin bolus or infusion
- Consider hydrocortisone
- Methylene blue (1-2 mg/kg IV bolus +/- infusion)
 - Inhibits NOS and cGMP
 - Interferes with oximetry labs and monitors
 - Contraindicated with other serotonergic medications and in G6PD deficiency
 - May ablate hypoxic pulmonary vasoconstriction
 - Can cause methemoglobinemia and hyperbilirubinemia
- Hydroxocobalamin (vit B12) (70 mcg/kg IV bolus, max 5g)
 - Inhibits NO and H₂S action in peripheral vascular smooth musculature
 - Repeat doses may cause multiorgan dysfunction related to increase in serum cobalt levels
 - Red color can interfere with oximetry & blood gas analysis

Signs

- Severe systemic vasodilation (low MAP with low SVR)
- Cardiac index is preserved or elevated (hypotension with high flows on cardiopulmonary bypass)

Pathophysiology

- Pathologic response to surgical trauma, blood contact with CPB surfaces, transfusion, and/or ischemia-reperfusion injury.
- Two cellular pathways cause inappropriate peripheral vasodilation:
 - Inducible nitric oxide synthase (iNOS) levels increase with systemic inflammation. iNOS creates nitric oxide (NO), which produces cyclic guanosine monophosphate (cGMP) which causes vascular smooth muscle relaxation.
 - iNOS creates NO. NO and hydrogen sulfide (H₂S) inhibit potassium channels in peripheral vascular smooth muscle cells, thus preventing inflow of calcium and vasoconstriction of vascular smooth muscle.

Other Considerations

- During rewarming, peripheral arterial hypotension may not reflect central arterial hypotension. Check a central arterial pressure if there is concern for refractory vasoplegia during rewarming.
- Thiamine, hydrocortisone, and ascorbic acid have been used in adults with VPS with anecdotal success.
- Methylene blue and hydroxocobalamin can be used in sequence in refractory cases.

Summary

Vasoplegic syndrome (VPS) is defined as systemic hypotension in the setting of decreased systemic vascular resistance (SVR) and normal or increased cardiac index (CI). The risk of VPS increases with numerous preoperative risk factors, particularly including the preoperative use of ACE-I, CCB, and amiodarone. VPS is often refractory to standard interventions to treat hypotension on CBP. Multimodal/multi-receptor therapy is recommended, including catecholaminergic and noncatecholaminergic vasopressor infusions and inhibitors of peripheral vasoculular smooth muscle relaxation (methylene blue and hydroxocobalamin). Perioperative vasoplegia, especially catecholamine-resistant VPS, carries significant increase in risk of perioperative major morbidity and mortality.