OHR - ICU POST CARDIAC SURGERY

INITIAL ASSESSMENT

- Handoff
  - Prioritize a handoff in complex or non-straightforward cases
- Perform a neurologic examination as soon as possible
  - Minimize sedation while ensuring adequate analgesia
- Hemodynamics and tissue perfusion
  - Systemic, pulmonary, and cardiac filling pressures
  - SV and CI
  - Skin perfusion, UO, lactate, mixed SvO2, and CMP
- Clinical assessment of venous congestion and if needed, ultrasonographic assessment
  - Physical examination, fluid balance and the presence of vascular congestion on CXR are useful tests to identify patients with volume overload but have several limitations
  - Ultrasonographic findings of presence of pulmonary B-lines, IVC collapsibility and internal jugular or femoral venous pressure may perform better than clinical assessment but have also several limitations
  - VExUS (Venous Excess Ultrasound Score) reflects the interaction between venous return and cardiac function, **not pure volume status**.
- Gas exchange function, respiratory mechanics, and acid-base status assessment
  - ABGs, Vt, PEEP, FiO2, and pulmonary pressures (peak, plateau, and driving pressures)
- Coagulation and bleeding assessment
  - CBC, INR/PTT, and chest/mediastinal tubes output

MANAGEMENT

Rewarm passively covering the patient with warm blankets vs. Bair hugger

- Rewarming frequently causes vasodilation with hypotension that may need a vasopressor or additional volume resuscitation

Cardiovascular

The goal is optimization without any interventionally reversible cause, or as a bridge until true source control is achieved.

Therapeutic strategies

- Typically MAP goals between 60-90 and SBP 90-140 mmHg
  - Swings are common; intervene based on trends and avoid overreaction
  - Baseline HTN, RV dysfunction, or renal failure may need higher MAPs

    - Baseline LV dysfunction, MV repair, regurgitant valvular lesions, active bleeding, or vulnerable aortic suture lines may need lower MAPs
    - It is important to identify which variable that affect the BP is compromised and treat accordingly
        - BP = CO (SV x HR) x SVR
        - Assess pressures interfaces

- Inotropes and vasopressors increase myocardial demand and are arrhythmogenic for which should be used judiciously to optimize end organ perfusion.
    - Dynamic hemodynamic assessment with PA catheter is of paramount importance
    - Echocardiography is essential for better understanding of the hemodynamic picture
        - TTE may have very limited views in the early post-op period and TEE may be needed
        - LVEF is a load-dependent variable, hence, an inherent measure of coupling between contractility and afterload.
            - A normal LVEF does not necessarily equate to an adequate CO which requires an adequate SV, not only a normal EF.
        - LVOT-VTI can be used to estimate SV, such that an adequate VTI (>18 cm2) likely rules out significant uncoupling between LV function and afterload.
    - Escalating levels of inotropes and vasopressors should call for consideration for:
        - Percutaneous mechanical circulatory support (pMCS)
        - Evaluation of dynamic LVOT obstruction (DLVOTO) by echocardiogram defined as a gradient pressure across the LVOT >30 mmHg
            - Consider DLVOTO in any patient with shock and a “great-looking EF” and lack of clinical response to increasing dose of vasopressors.
            - DLVOTO can be identified by presence of SAM on M-mode and mosaic of colors suggesting high-velocity flow on continuous flow Doppler. Look for aortic valve mid-systolic notching or fluttering and also, we may see the classic dagger-shaped profile

LV or biventricular failure

- Usually treated with inopressors
    - Low dose epinephrine and/or norepinephrine (0.02 mcg/kg/min-0.08 mcg/kg/min)

- May need inodilators
    - Dobutamine (2.5 to 20 mcg/Kg/min)
        - More tachycardia
    - Milrinone (0.125 to 0.75 mcg/Kg/min)
        - More effective at decreasing PVR and associated with less tachycardia
        - However, the onset of effect is longer, and the effect lasts longer
    - If inodilators are used, they require simultaneous inopressors (epinephrine and/or norepinephrine) and may need vasopressors to prevent hypotension
        - Vasopressin 0.01 – 0.04 U/min (increases SVR but not PVR))
        - Avoid phenylephrine (unless dynamic LVOT obstruction)
            - It can decrease bypass graft flow, worsen pulmonary pressures, and less potent compared to norepinephrine and vasopressin
    - Consider digoxin for patients with Afib and RVR
        - 0.125 – 0.250 mg IV q6h x2. Thereafter daily adjusted by renal function

RV failure due to increased RV afterload

- Avoid hypoxemia, hypercapnia, and acidemia
- Usually requires combination of IVF, inodilators, inopressors, and vasopressors
    - Ensure adequate RV perfusion/MAP first with IVF and inopressors
    - Increase contractility and decrease PVR/RV afterload
        - Inodilators (milrinone usually preferred)
        - If refractory RV failure, consider inhaled pulmonary vasodilator therapy (iNO/Epoprostenol)

Arrythmias

- Pacing thresholds need to be checked ideally every shift
- Important to identify changing thresholds especially in those that are pacer-dependent

Bradycardia/conduction abnormalities

- Temporary epicardial atrial and ventricular pacing wires
    - Atrial pacing is preferred if AV conduction is intact
    - Sequential atrial-ventricular pacing if AV conduction is impaired
    - Ventricular pacing should only be used as backup/rescue
        - It may need to be used if there are no atrial wires placed or atrial wires no longer functional
- Consider pacemaker if AV block >7 days

Tachyarrhythmias

- Afib
    - B-blockers first line if not requiring vasoactive therapy
    - Antiarrhythmics, most commonly amiodarone (less negative inotropy)
    - Magnesium
    - Atrial pacing
    - Cardioversion for hemodynamic instability
    - Anticoagulation (AC)
        - Not indicated for <48 hr duration
        - If it is greater than 48 hr duration, it needs to weigh individual risk factors for post-op bleeding vs prevention of thrombotic events. Expert opinion recommends AC for a minimum of 4-6 weeks
- Ventricular arrhythmias
    - Are uncommon and should raise suspicion for ongoing ischemia (grafts or coronary button) with low threshold for cath lab for assessment
        - The coronary button refers to the portion of the coronary arteries that are surgically detached and reimplanted to the aorta on aortic root replacements
    - Antiarrhythmics (amiodarone/lidocaine/ibutilide)
    - Cardioversion/defibrillation
    - For refractory ventricular arrhythmias consider:
        - pMCS
        - Stellate ganglion block for patients with electrical storm defined as >3 episodes of sustained VT/VF within 24 h despite antiarrhythmic therapy

Vasodilatory shock

- Norepinephrine/vasopressin
- Oxygen scavengers
    - Hydroxocobalamin (preferred)
        - CYANOKIT 5 gr IV infusion over 15 minutes. A second dose may be used

    OR
    - Methylene blue
        - Avoid when using serotonergic meds or underlying G6PD or pulmonary hypertension
        - Initial IV bolus dose of 1-2 mg/kg over 10-20 minutes. In some cases, a maintenance infusion of 0.25-0.5 mg/kg/hour may be used depending on the patient's response and clinical condition

Hypovolemic shock

- Assess for bleeding and need for blood products replacement and/or surgical re-exploration
- If due to decreased effective intravascular volume (decreased preload and SV compensated by elevated SVR "dry and cold")
    - IVF resuscitation
    - On occasions, until volume replacement is achieved and there is no hypotension, vasodilators can be used
        - Short acting agents preferred: Clevidipine/Nitroglycerine
        - Nicardipine can be used but has longer half-life with risk for hemodynamic deterioration
        - Vasodilators can also decrease the risk of arterial graft vasospasm

Obstructive shock

- Assess with stat echocardiogram
    - If cardiac tamponade
        - IVF and inopressor support
        - Stat cardiac surgery and interventional cardiology evaluation
            - Surgical re-exploration
            - Emergent pericardiocentesis
    - If PE
        - Activate PERT
        - Systemic anticoagulation
        - Consider pMCS

Dynamic LVOT obstruction

Primary DLVOTO with preserved ventricular function

- Sepsis and hypovolemia are the usual underlying clinical conditions

1. *Optimize preload*
    - Stop diuretics
    - Give a 250–500 mL crystalloid bolus and reassess
2. *Increase afterload*
    - Phenylephrine or vasopressin are classically first-line agents, as they avoid β-agonism and act as pure afterload increasers without increasing inotropy
        - They may also induce reflexive bradycardia which could be helpful to increase diastolic filling time and augment LV preload
    - Norepinephrine-probably should be the second line. Mild β_1 effect—monitor closely for potential contribution to LVOTO if used (reported in case study).
    - Avoid epinephrine, beta effects can worsen LVOTO
    - Discontinue IABP: reduces LV afterload, worsens LVOTO. Caution that thrombus can form very quickly so either a transient pause or remove IABP ASAP
3. *Reduce contractility*

- Stop inotropes

4. Control heart rate – slower HR important to allow increased diastolic filling, especially in a hypertrophied ventricle
    - Keep HR 55 –70 bpm, but the important concept is to slow the heart rate compared to LVOTO presentation
    - If hypotensive and unstable after IV fluids, optimizing afterload, and stopping inotropes, consider initiation of short acting B-Blocker like Esmolol
5. Maintain sinus rhythm
    - Atrial fibrillation with loss of "atrial kick" can impair LV diastolic filling
        - Amiodarone or cardioversion for atrial fibrillation as indicated
    - Post–cardiac surgery without underlying SR
        - Prefer AAI pacing if AV conduction is intact
        - Use DDD if AV conduction is impaired
        - Avoid VVI pacing when possible

Secondary dynamic LVOTO with reduced RV function

In patients with susceptible LV, RV failure can be the physiological trigger leading to an "underfilled, compressed LV" that precipitates DLVOTO

- Usual underlying clinical conditions
    - Post- cardiotomy RV failure in patients with small, hypertrophied LV
    - PE with high risk or Intermediate-high risk mortality (AHA categories D or E)
    - Pulmonary hypertension with resultant RV-PA uncoupling
- Pathophysiology
    - Decreased LV preload due to decreased RV cardiac output
    - Ventricular interdependence due to dilated RV resulting in volume/pressure overload shifting the septum leftward in systole
        - The reduced LV preload and or ventricular interdependence can result in narrowing the LVOT geometry and promoting high velocities inducing SAM which is a classic permissive state for dynamic LVOTO/SAM
- Treatment
    - When dynamic LVOTO is secondary to RV failure, treating it like primary LVOTO can make things worse.
    - In this setting, indiscriminate volume loading may worsen RV dilation and pericardial constraint, further limiting LV preload, while beta-blockade can depress RV contractility and precipitate hemodynamic decompensation.
    - Fix the RV failure, and often the LVOTO resolves.

1. Treat the underlying etiology
2. Target a higher MAP which supports the RV perfusion while providing adequate LV afterload to reduce the LVOTO
    - Vasopressin works well because it raises SVR and BP without increasing PVR or RV afterload
    - Norepinephrine may be a good option as its mild β_1 effects can provide some inotropic support to a struggling RV
    - Phenylephrine should be avoided due to its propensity to increase PVR and worsen RV failure
3. Reduce RV afterload
    - Correct hypoxemia, hypercapnia and acidemia
    - Consider inhaled pulmonary vasodilators (Epoprostenol, nitric oxide not available to us, nitroglycerine by nebulization can be used as it will be converted to nitric oxide)
4. Consider diuresis, it may be beneficial when the RV is dilated and operating beyond its optimal Frank-Starling range
5. Consider inopressors as a last resort in severe RV failure with markedly reduced contractility if hemodynamics remains inadequate (low-dose epinephrine (0.01–0.03 mcg/kg/min is preferred with close bedside reassessment)
6. Pure inodilators (e.g., milrinone) should be avoided, as SVR/afterload reduction may worsen dynamic LVOT obstruction
7. Early referral for or initiation of ECMO to address the severe RV failure if the above measures fail to resolve the dynamic LVOTO, or if RV pharmacological support is worsening the situation

Pulmonary

Etiology of post-operative hypoxemia

- Mechanical
    - ET tube migration
    - Mucous plugging
    - Phrenic nerve injury
    - Pneumothorax, unlikely if chest tubes are functioning well
- Pulmonary edema
    - SIRS-like syndrome with capillary leak
    - Reperfusion injury to lungs especially if prolonged CPB
    - Volume overload especially if need for multiple blood products transfusion
    - Poor LV function

- Intra-pulmonary shunt
    - Incomplete lung reinflation with atelectasis-typically lingula/LLL
    - Hemothorax/pleural effusion
    - Decreased chest wall compliance (sternum wired)
    - V/Q mismatch due to vasodilator (Nitroglycerine/Clevidipine/Nicardipine) inhibition of hypoxic pulmonary vasoconstriction
- Right to left intracardiac shunt due PFO opening by increased pulmonary pressures

Invasive mechanical ventilatory (IMV) support management

The goal is to liberate from IMV within 6 hours following ERAS recommendation and lung protective strategy.

- Vt 6 mL/kg PBW, with allowances for 4–8 mL/kg
- PEEP levels adjusted, guided by the PEEP-FiO2 table to keep plateau pressure <30 and driving pressure <15 using the lowest FIO2 possible
- Adjust vent to target PaO2/FiO2 ratio >300, PaO2 ≥60 mmHg with O2 Sat >90% and PaCO2 37- 45 mmHg
- Adjust sedation/analgesia targeting ANVPS ≤2 and RASS 1 to -2 and RASS -4 if hemodynamic instability
    - Spontaneous awakening trial (minimize sedation while ensuring adequate pain control) and neuro exam
    - If need to reinitiate sedation prioritize short-acting agents (propofol or dexmedetomidine)
    - Pain control to optimize patient's comfort, O2 supply-demand balance, pulmonary function by regaining FRC, and prevent delirium
- Multimodal analgesia – intravenous with transition to oral when able
        - Fentanyl IV boluses as needed
        - Opioid sparing agents
            - Acetaminophen, can consider Toradol if no renal dysfunction
            - Consider pain dose ketamine
- Promote early mobilization and physiotherapy within hours post-extubation

Criteria for liberation readiness

- Adequate hemodynamic stability with minimal inotropic or vasopressor support
- Satisfactory oxygenation and ventilation (e.g., PaO2/FiO2 ratio > 250 and normocapnia)
- Adequate pain control and absence of excessive bleeding
- Adequate level of consciousness to protect the airway (responsive and cooperative)

Neurovascular

Management of common neurological insults per UF Health Flagler Hospital protocols

- Acute ischemic and hemorrhagic strokes
    - Atheroembolism and microembolization
    - Watershed infarcts especially in those with carotid/vertebral stenosis
    - Air embolization
- Seizures (continuous EEG monitoring is recommended if the patient does not return to baseline or if nonconvulsive seizures are suspected). Etiologies include:
    - Tranexamic acid (TXA)-induced seizures
        - These are typically self-limited but can progress to status epilepticus. Notably, peak TXA concentrations in CSF occur after termination of the infusion, so seizures may still develop or worsen after the drug is stopped
        - Discontinuation of TXA infusion (if still running)
        - Propofol if needed
    - Perioperative stroke
    - Electrolyte abnormalities (sodium, calcium, magnesium) or hypoglycemia
- ICU delirium
- Postoperative cognitive dysfunction or decline

Hematology

Anemia and thrombocytopenia are expected

- Platelets count typically decreases by 50% with peak at 48-72 hrs

Excessive bleeding definition

- It is variable, in general be concerned if:
    - Chest tube output is >400 1st hour, >300 for first 2 h, > 200 for 3 consecutive hours
    - Regardless of the amount, if sudden bleeding increase or decreases (accumulating hemopericardium/hemothorax)

Excessive bleeding treatment

- Assure warming
- Tranexamic acid (TXA)
    - It has shown to decrease post-operative bleeding/transfusion requirements in patients post cardiac bypass
    - Typically initiated in the OR at end of case and infusing for several hours in ICU
    - High dose use has been associated with increased risk of seizures
- Correct coagulopathy
    - PRBC, target Hb >8 g/dl
    - Platelets, target platelets >50,000
    - FFP, target INR ≥1.5
    - Cryoprecipitate, target fibrinogen levels >150 mg/dL

- Replace calcium
- DDAVP 0.3 mcg/Kg dose IV over 30 min (antiplatelet therapy-related or uremia)
- Reverse heparin with protamine
- If there is no medical etiology for bleeding, consider surgical re-exploration