Cardiovascular Surgery 101: The Basics of Postoperative Care

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None
History

- Pre-op Hemodynamics
- Operation
- Ischemic Time
- Complications
- Inotropes
Examination

- ABC’s - Endotracheal tube/ventilator
- EKG
- Pulse Oximetry
- Blood Pressure
- Color / Peripheral pulses / Temperature of extremities
- Chest tubes
- Foley catheter / Urine o/p
- Inotropes - what drugs/infusions running/correct dosages
Chest X-Ray

- ET tube
- NGT
- Chest tubes
- Lung fields-fluid/air
- Heart shadow
- Swan catheter tip
- IABP tip
- Mediastinal width
Laboratory

- ABG
- K+
- Hct
- Bleeding-PT/INR, PTT, Platelets
- Impaired renal Function → Cr
Assessment of perfusion
How do you do it?

What is “Shock”? 
Blood Pressure
LE temperature and pulses
Urine output
Cardiac output
Metabolic Shock

- Acid/Base balance
- Anaerobic metabolism $\rightarrow$ Lactic acidosis $\rightarrow$ Base deficit
- Mixed Venous (SVO2)
- $SvO2 = SaO2 - VO2 / (Hgb \times 1.39 \times CO) \times 10$
Acute Management of Hemodynamics

R/O Mechanical causes of cardiogenic shock

- Tension pneumothorax
- Hypoxia
- Tamponade
- Pressors inadvertently d/c’d
- Acute MI-thrombosed graft
- Bleeding- open line/intraabdominal hemorrhage from femoral access
Determinedants of Cardiac Output

- Stroke volume \times \text{heart rate}
- Stroke volume
  - preload
  - afterload
  - contractility
Hemodynamic Management –
Strategy to Optimize Cardiac Function

- Rate
- Rhythm
- Preload
- Afterload
- Contractility
Rate

- Optimal rate 90-100 bpm
- Atrial pace if AV node functioning
- A-V pace if some degree of heart block
Rhythm

- Identify rhythm
- If HR > 120 → slow rate pharmacologically
  - Inadequate diastolic filling
- If lack of A-V synchrony → A-V sequentially pace
  - to optimize atrial transport
Preload

Maximizing Frank-Starling relationship

If CVP or PCWP < 15 → volume
- crystalloid
- colloid
- blood

If CVP or PCWP > 15
- afterload or contractility
Afterload

- Resistance (increases work)
  - SVR = \(\frac{\text{MAP} - \text{CVP}}{80}\) 800-1200 dyne-sec/cm\(^5\)

  - CO
  - SVR \textit{increased} \(\rightarrow\) low CO \(\rightarrow\) hypertensive \(\rightarrow\)
    - reduce afterload \((\text{NTG/SNP})\)

  - SVR \textit{decreased} \(\rightarrow\) high CO \(\rightarrow\) hypotensive

  \(\rightarrow\)

  - increase afterload

  \((\text{vasopressin, phenylephrine, norepinephrine})\)
Contractility

Persistent low BP and low CO despite optimization of above parameters.

The only remaining reason for poor CO is intrinsic myocardial dysfunction.

This is closely associated with ischemia/reperfusion. Injury of muscle with troponin leak.

There is a fairly predictable time line of myocardial recovery (majority but not all cases).
Contractility

Pharmacologic support-Pressors

- **Dopamine**- 3-8 ug/kg/min-mild inotropy, huge chronotropy and increasing alpha

- **Dobutamine**- 2.5- 10 ug/kg/min-mod chronotropy and w/ moderate pulmonary and peripheral vasodilatory properties. +/- chronotropy

- **Epinephrine**- 0.01- 0.1 ug/kg/min- potent inotropy, mild chronotropy, increasing alpha

- **Milrinone**- excellent for right heart failure as it is a more potent pulmonary vasodilator than dobutamine. Also has potent systemic vasodilating properties so often used in conjunction w/ Epinephrine. Moderate inotropy as well.
Mechanical Support

- Intra-aortic ballon pump (IABP)
  - reduces afterload
  - increases diastolic coronary perfusion pressure

- Left ventricular assist device
  - temporary or permanent

- Bi-ventricular device
  - short term or long term

- Extracorporeal membrane oxygentation (ECMO)
Questions?