Cardiac Surgery
Pearls of Wisdom
Fun and Focused
Class C150M605

Objectives
- Relate hemodynamic concepts of preload, afterload and contractility to medication management of cardiac surgery patients.
- Discuss assessment cues and management of cardiac surgery patients to prevent and treat complications associated with cardiac surgery.
- Differentiate the plan of care for cardiac surgery patients with coronary artery bypass surgery and valvular surgery/repair.

Learn something new
And have fun 😊

Are you certified?
1. CCRN
2. CCRN-CSC
3. CSC
4. Other
5. Certification "Wannabe"

Speaker Disclosures
- AACN Speaker Bureau
- Cross Country/Vyne Education Speaker Bureau
- Novartis Speaker Bureau
- Handouts will be available at www.cherylherrmann.com

Where in the World is Peoria?
“Will it Play in Peoria?”

polling
Number of years as Cardiac Surgery Nurse?
1. Less than 1 year
2. 1–2 years
3. 3–5 years
4. 6–10 years
5. >10 years–almost ancient 😊

Polling

CSC Exam Content
The CSC exam is a 2-hour test consisting of 90 multiple-choice items. Of the 90 items, 75 are scored and 15 are used to gather statistical data on item performance for future exams. Please see the test plan for more information. The CSC exam focuses on adult populations. One hundred percent (100%) of the exam focuses on clinical judgment.

www.aacn.org

Exam Blueprint
- Cardiovascular Patient Care Problems (33%)
- Other Patient Care Problems (24%)
- Nursing Interventions (33%)
- Monitoring & Diagnostics (9%)

Care of the Cardiac Surgery Patient
first 48 hours Post op

Resources/Study Books
- Dodge, T. Fast Facts for the Cardiac surgery nurse. Springer Publishing
- www.aacn.org

Cardiovascular Patient Care Problems (33%)
30 questions
Not studying for CSC
“No worries”

Fun and Focused
1. Valvular Surgery
2. Optimizing Cardiac Output
   • Preload, Afterload, Contractility, Heart Rate
   • Pharmacology
   • Hemodynamic Case Studies and Practice
3. Triad of Disaster – Preventing & Treating Complications

Let’s Start!

How has Cardiac Surgery Changed?

Last Decade, isolated CABG ↓ from 73% to 57% (Vahanian et al, 2011)

Valvular Surgery
- Increased from 16–22%
- 43% AVR + CABG
- 38% MVR + CABG
- 1997–2006 – percentage of valve surgery patients > 80 y/o increased from 13–20%
- Mitral Regurgitation often associated with HF

Source: Vahanian et al. 2011
The Killer Event!

Stop the complications before they start!

Valvular Surgery
Valvular Heart Disease

- An acquired or congenital disorder of a cardiac valve
- Characterized by
  - Stenosis (obstruction)
  - Regurgitation (backward flow)
- Can occur acutely
- Typically is a chronic progressive disorder
- Causes a significant impact on quality of life
- Medical management delays the inevitable surgery for replacement/repair
- Prosthetic valve creates new problems

Aortic Valve

- Has three leaflets or cusps
- Cusps close as the pressure in the aorta becomes greater than the pressure in the left ventricle.

Aortic Stenosis

- Aortic valve will not open completely
- Restricts flow of blood from left ventricle to aorta
- Most common valve lesion in USA

1. Small opening causes ↓ blood flow and ↓ CO
2. ↑ Afterload
3. ↑ Workload in Left ventricle
4. ↑ Pressure in LV
5. LV hypertrophy

Surgical Treatment for Aortic Valve Disease

- Aortic Valve Replacement (mainstay)
  - Avoid hypertension and stress on suture line
- Aortic Valve Repair (not mainstream)
- Transcatheter Aortic Valve Replacement (TVAR)

Aortic Regurgitation/Insufficiency

- Aortic valve fails to close completely
- Backflow of blood into the left ventricle during diastole
- Severe AI – most frequently caused by bicuspid valve

1. Volume overload leads to compensatory mechanisms
  - Left ventricular hypertrophy
  - ↑ End-diastolic volume which allows normal EF despite ↑ afterload.
  - ↑ LV afterload as the ↑ volume ejected into the high pressured aorta.

Transcatheter Aortic Valve Replacement (TVAR)

- Trileaflet bioprosthesis mounted on a balloon catheter delivered through the arterial system via a guidewire. Device is inserted into the midpoint of the native valve
Trans Apical vs Trans Femoral

- Femoral most common

https://www.youtube.com/watch?v=ztJ3cc2EOmM

TAVR

- Pro’s
  - Less invasive than traditional AVR’s
  - No sternotomy or cardiopulmonary bypass
  - Less ventilation time or extubated in OR
  - Shorter ICU length of stay and often discharged within 48 hours postop

- Con’s
  - Elderly population with comorbidities
  - Higher risk for delirium due to sedation or pain management
  - Screening for physical therapy

Post Op TVAR Femoral

- Usually extubated in OR, if not within 2–4 hrs postop
- Monitor bilateral puncture sites – hold pressure if oozing or bleeding
- Monitor pulses distal to insertion site due to the large catheters and embolization risk
- Monitor neuro assessment due to high risk for strokes
- Maintain SBP between 100mmHg – 130mmHg
  - May use beta blockers or other vasodilators for hypertension
- Discontinue Arterial line after extubation and venous sheath when ACT <180
- Internal Jugular discontinued on POD 1 and transferred to Telemetry
- All patients assessed for rehab upon transfer from ICU

Post Op TVAR Apical Postop

- Monitor hemodynamics, neuro assessment, urine output, & chest drainage same as an open sternotomy incision
- Wean to extubate within 6 hours of anesthesia end time. Encourage incentive spirometer every hour while awake
- Discontinue femoral lines after extubation
- Ice chips and advance diet as tolerated
- Up in chair early am and ambulate with physical therapy or nurses 3–4 times/day
- Discontinue PA catheter and arterial line POD 1

Potential TVAR Complications

- Complete Heart Block due to Aortic Valve edema.
- Hypotension
  - Monitor amount of sedation or vasodilating medications for cause of hypotension
- Check groin sites for bleeding, lower abdomen for signs of retroperitoneal bleed, check peripheral vascular pulses
- Monitor Labs (Hgb/Ht)
- Vasovagal response
- Stroke
  - Assess neuro status with VS’s

Mitral Valve

- Large anterior leaflet
- Small posterior leaflet
- Chordae tendineae and papillary muscles prevent the prolapse of valve leaflets into left atrium during systole
Mitral Stenosis

- Mitral valve will not open completely
- Restricts flow of blood from left atrium to LV

- Small opening causes ↓ blood flow and ↓ CO
- ↑ workload in Left Atrium
- ↑ pressure in LA
- LA dilation & hypertrophy
- ↑ in LA pressure → backflow into pulmonary artery
- Leads to pulmonary hypertension, congestion, right ventricular hypertrophy and right sided heart failure

- LV size and contractility = normal in MS

Mitral Regurgitation

- Mitral valve fails to close completely
- Blood is propelled backward into the LA during systole

- During systole, a portion of blood is ejected back into the LA
- ↓ blood in LV → ↓ CO
- ↑ blood in LA → ↑ LA pressures → pulmonary congestion and ↑ pulmonary pressures → RV hypertrophy
- During diastole, blood continues to flow into LV → ↑ LV volume
- LV hypertrophy

- MR = LA enlargement, Left or Right Ventricular Failure

Post op MVR and repair for MS

- Assess for pulmonary hypertension
- Increased PVR leads to RV failure
- Increased CVP = possible RV decompression
- TEE to assess for RV and LV function
- Dobutamine, Milrinone, Norepinephrine to increase contractility of RV and ↓ PVR
- Fluid administration
- PAD does not reflect LA filling pressures related to pulmonary hypertension – Wedge more accurate
- PA catheter may be placed farther in related to dilated pulmonary arteries
- IABP usually not indicated as no LV dysfunction but RV dysfunction

Post op for Mitral Valve Repair for MR

- Immediate ↑ SVR due to no backflow of blood in LA
- Pulmonary hypertension & Myocardial hibernation take time to reverse
- Inotropes (Milrinone, Dobutamine)
- IABP
- Monitor for RV failure

Mitral Valve Repair vs Mitral Valve Replacement

- Repair preserves native valve
- Repair is favored due to disadvantages of prosthetic valves
  - No anticoagulation needed for repair
- Technically more difficult
  - Depends on degree of regurgitation,
  - Pathophysiology of the regurgitation
  - LV function,
  - Ability of surgeon

Valve Replacement Considerations

<table>
<thead>
<tr>
<th></th>
<th>Tissue</th>
<th>Mechanical</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Over 65 yo</td>
<td>Under 65</td>
</tr>
<tr>
<td>Longevity</td>
<td>10–15 years</td>
<td>Potentially Lifetime</td>
</tr>
<tr>
<td>Anticoagulation</td>
<td>Aspirin lifelong</td>
<td>Warfarin lifelong</td>
</tr>
<tr>
<td></td>
<td>Warfarin lifelong</td>
<td>Warfarin lifelong</td>
</tr>
<tr>
<td>Reoperation risk</td>
<td>Patient dependent</td>
<td>As low as 1% risk lifetime</td>
</tr>
</tbody>
</table>

4/22/2017
Postoperative Valve Considerations
- Physical examination
  - Normal prosthetic heart valve sounds:
    - Mechanical valves:
      - Loud, high-frequency, metallic closing sound
    - Soft opening sound
  - Tissue valves:
    - Closing similar to those of native valves
  - New onset murmurs is a concern
    - Murmurs – though hard to hear – would raise suspicion

Postoperative Valve Considerations
- Embolic complications
  - Stroke
  - TIA
- Anticoagulant-related hemorrhage
  - Hemorrhage site – brain, abdomen, etc.
- Dysrhythmia
  - AV Block
  - Atrial dysrhythmias

Prosthetic Valve Endocarditis
- Blood borne bacterial traveling to the heart and growing on the valve
- Dental or other procedures may provoke bacteremia

What is SBE?
Subacute bacterial endocarditis
- Antibiotic prophylaxis is indicated for the following high-risk cardiac conditions:
  - Prosthetic cardiac valve
  - History of infective endocarditis
  - Congenital heart disease (CHD)
  - Cardiac transplantation recipients with cardiac valvular disease
- For these procedures
  - Dental
  - Invasive respiratory (bronch)

Standard general prophylaxis
- Amoxicillin
  - Adult dose: 2 g PO
  - Pediatric dose: 50 mg/kg PO; not to exceed 2 g/dose
  - Administer once as a single dose 30–60 min before the procedure.
- Amoxicillin, Clindamycin, Cephalexin, Cefazolin, or Ceftriazone
  - May be used if allergic or unable to take oral
  - See guidelines for specific doses
Case Study

- Ms Leaky, a 47 y/o. had a MVR. Today on POD #4, she is being transferred to the progressive care unit.

At 1508 Ms Leaky’s rhythm changes to this.

The left ventricle is normal size in which valvular disease?

1. Aortic Stenosis
2. Aortic Insufficiency
3. Mitral Stenosis
4. Mitral Regurgitation

Damage to His bundle may result in BBB or CHB

The left ventricle is normal size in which valvular disease?

- Aortic Stenosis
- Aortic Insufficiency
- Mitral Stenosis
- Mitral Regurgitation

ANSWER

1. Mitral Stenosis
2. Mitral Regurgitation
3. Mitral Stenosis

Valvular Pearls

<table>
<thead>
<tr>
<th>Heart Sounds</th>
<th>MS</th>
<th>MR</th>
<th>AS</th>
<th>AR/AI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mid-diasstic murmur at the apex</td>
<td>S3, S4</td>
<td>RV heave</td>
<td>Systolic ejection murmur</td>
<td>Heart failure</td>
</tr>
<tr>
<td>Holosystolic murmur</td>
<td>High pitched Widely split S2 S3, S4</td>
<td>Systolic ejection murmur</td>
<td>Mitral valve prolapse</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>MS</th>
<th>MR</th>
<th>AS</th>
<th>AR/AI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyspnea</td>
<td>Pulmonary Hypertension</td>
<td>Pulmonary symptoms</td>
<td>Syncope</td>
<td>Dyspnea</td>
</tr>
<tr>
<td>Peripheral edema</td>
<td>Cough</td>
<td>LV failure</td>
<td>Holosystolic murmur at right sternal border</td>
<td>Decrescendo decrescendo blowing murmur</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Atrial size</th>
<th>MS</th>
<th>MR</th>
<th>AS</th>
<th>AR/AI</th>
</tr>
</thead>
<tbody>
<tr>
<td>LA enlarged</td>
<td>LA enlarged</td>
<td>LA enlarged</td>
<td>LA enlarged</td>
<td>LA enlarged</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Ventricular Size</th>
<th>MS</th>
<th>MR</th>
<th>AS</th>
<th>AR/AI</th>
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</thead>
<tbody>
<tr>
<td>LV normal</td>
<td>LV enlarged</td>
<td>LV enlarged</td>
<td>LV enlarged</td>
<td>LV enlarged</td>
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</tbody>
</table>
Valvular Surgery Pearls

Aortic Valve

<table>
<thead>
<tr>
<th>Aortic Stenosis</th>
<th>Aortic Regurgitation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preop</td>
<td></td>
</tr>
<tr>
<td>LV hypertrophy</td>
<td>LV hypertrophy</td>
</tr>
<tr>
<td>↑ SVR</td>
<td></td>
</tr>
<tr>
<td>s/s heart failure</td>
<td></td>
</tr>
<tr>
<td>Post op</td>
<td></td>
</tr>
<tr>
<td>LV may not anticipate ↑ in SVR and continue to pump hard</td>
<td>IV vasodilators to ↓ SVR</td>
</tr>
<tr>
<td>Avoid hypertension and stress on suture line</td>
<td>promote emptying LV: Milrinone/Dobutamine IABP</td>
</tr>
</tbody>
</table>

Optimizing Cardiac Output

Cardiac Surgery
Hemodynamics/Medications

Valvular Surgery Pearls

Mitral Valve

<table>
<thead>
<tr>
<th>Mitral Stenosis</th>
<th>Mitral Regurgitation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preop</td>
<td></td>
</tr>
<tr>
<td>NS LV function</td>
<td>Pulmonary Hypertension</td>
</tr>
<tr>
<td>RV failure</td>
<td>High atrial &amp; pulmonary pressures</td>
</tr>
<tr>
<td>Pulmonary congestion</td>
<td></td>
</tr>
<tr>
<td>Post op</td>
<td></td>
</tr>
<tr>
<td>Assess pulmonary hypertension (PVR)</td>
<td>Immediate ↑ SVR due to no backflow of blood in LA</td>
</tr>
<tr>
<td>Dobutamine or Milrinone</td>
<td>Pulmonary hypertension &amp; myocardial hibernation take time to reverse</td>
</tr>
<tr>
<td>- Norepinephrine to ↑ contractility of RV &amp; ↑ PVR</td>
<td>Inotropes (Milrinone, Dobutamine) + epinephrine</td>
</tr>
<tr>
<td>Fluids</td>
<td>IABP Monitor for RV failure</td>
</tr>
<tr>
<td>- CVP may indicate RV decompression</td>
<td>Treat atrial fibrillation</td>
</tr>
</tbody>
</table>

Terms used to describe Cardiac Drug Effects

- **Inotropic**: Effect on contractility
  - Positive = increase in contractility
  - Negative = decrease in contractility
- **Chronotropic**: Effect on Heart Rate
  - Positive = increase in Heart Rate
  - Negative = decrease in Heart Rate
- **Dromotropic**: Effect on Conductivity
  - Positive = increase in conductivity
  - Negative = decrease in conductivity

Cardiac Index

\[ CI = \frac{CO}{BSA} \]

- Cardiac output divided by body surface area (BSA)
  - Normal range = 2.5 - 4 l/min/m²
  - Subclinical: 2.2 - 2.7 l/min/m²
  - Low perfusion: 1.8 - 2.2 l/min/m²
  - Shock < < 1.8 l/min/m²

Is a cardiac output of 4.2 l/min. adequate for both Mrs. A, a 5 ft. 98 lb. woman and Mr. B, a 6 ft. 2 in., 240 lb. man?
By using formula CI = CO/BSA

Mrs. A’s BSA is 1.36 m². Her CI is determined to be 3.08 l/min/m².

Mr. B has a BSA of 2.34 m², therefore his CI falls below the normal level of 1.79 l/min/m².

Determinants of Cardiac Output

Cardiac Output = Heart Rate x Stroke Volume

Determinants of CO:

Rate/Rhythm

Low
- Pacemaker
- Atropine
- Isuprel
- Dopamine

High
- Beta blockers
- Calcium channel blockers
- Other

Heart Rate

- Increasing Heart Rate is the fastest way to increase CO.
- Overtime, it is not the most efficient way.
- Optimal HR is 60 – 80 bpm

How Cardiac Meds effect Heart Rate

How Cardiac Meds effect Heart Rate

- Dopamine Hydrochloride (Intropin)
- Epinephrine (Adrenalin)
- Norepinephrine Bitartrate (Levophed)
- Phenylephrine (Neo-Synephrine)
- Vasopressin (Pitressin)
- Nitroprusside (Nipride)
- Nitroglycerin (Tridil)
- Dobutamine Hydrochloride (Dobutrex)
- Digitalis (Digoxin, Lanoxin)
- Milrinone (Primacor)
- Calcium Chloride
- Amiodarone Hydrochloride (Cordarone)
- Lidocaine (Xylocaine)
- Atropine Sulfate
- ACE Inhibitors
- Beta Blockers
- Diltiazem (Cardizem)
- Nicardipine (Cardene)

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The Effect of Cardiac Meds on Heart Rate

**Increase HR**
- Atropine
- Dopamine/Intopin
- Epinephrine/Adrenaline
- Norepinephrine/Levophed
- Dobutamine/Dobutrex

**Decrease HR**
- Beta Blockers
- Calcium Channel Blockers

**Slight Increase HR**
- Milrinone/Primacor

**No effect on HR**
- Phenylephrine/Neo-synephrine
- Vasopressin/Pitressin

Know Normal Values!

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac Output (CO)</td>
<td>4 - 8 l/min</td>
</tr>
<tr>
<td>Cardiac Index (CI)</td>
<td>2.5 – 4.2 l/min/m²</td>
</tr>
<tr>
<td>Right atrial pressure (CVP)</td>
<td>0 – 8 mmHg</td>
</tr>
<tr>
<td>Pulmonary artery pressure (PAS/PAD)</td>
<td>15 – 30/6 -12 mmHg</td>
</tr>
<tr>
<td>Pulmonary artery occlusive pressure</td>
<td>4 – 12 mmHg</td>
</tr>
<tr>
<td>Systemic vascular resistance (SVR)</td>
<td>770 – 1500 dyne/sec/cm²</td>
</tr>
<tr>
<td>Pulmonary vascular resistance (PVR)</td>
<td>20 – 120 dyne/sec/cm²</td>
</tr>
<tr>
<td>Stroke Volume (SV)</td>
<td>60 - 120 mL/beat</td>
</tr>
<tr>
<td>Stroke Volume Index (SVI)</td>
<td>30 – 65 mL/beat/m²</td>
</tr>
<tr>
<td>Arterial oxygenation saturation</td>
<td>95 – 100 %</td>
</tr>
<tr>
<td>Venous oxygenation saturation</td>
<td>60 – 80 %</td>
</tr>
</tbody>
</table>

Source: Stated in Cardiac Surgery Essentials, page 148.

Determinants of Cardiac Output

**Cardiac Output = Heart Rate x Stroke Volume**

- Preload
- Afterload
- Contractility

Stroke Volume Optimization

**BP = CO x SVR**

- CO = Stroke Volume + Heart Rate
- Body compensates to keep BP normal
  - ↓ SV causes ↑ HR
  - ↓ CO causes ↑ SVR
- Thus, BP does not change until late.

Why Stroke Volume Optimization?

- Stroke volume is the first parameter that changes before...
  - Tissue hypoperfusion and
  - Organ dysfunction
Order of Events

1. Stroke Volume Decreases
   - HR compensated to keep CO normal
2. Cardiac Output Decreases
   - HR compensation fails
   - Vasoconstriction (↑ SVR)
   - BP remains the same
3. Increased oxygen extraction of hemoglobin
   - Peripherally initially (StO$_2$)
   - Central Later (SvO$_2$)
4. Blood Pressure, Urine Output Change

Stroke Volume (SV)
Stroke Volume Index (SVI)

- SV: Volume of blood ejected with each beat
  - Normal SV: 60 – 100ml
- SVI: the amount of blood pumped with each beat indexed to BSA
  - Normal SVI: 33 – 47 ml/m2
  - Very powerful indicator of ventricular function

Hemodynamics 101

Interpretation of SV/SI

- If low, the cause may be:
  - Inadequate fluid volume: bleeding
  - Impaired ventricular contractility: MI
  - Increased SVR (afterload or resistance to ejection)
  - Cardiac valve dysfunction: mitral regurgitation
- If high, the cause may be:
  - Fluid overload
  - Low vascular resistance: sepsis
SV Pearls

- As HR goes up, SV is going down
- CVP is not a stand alone measure for volume. Use SV
- Volume first, then inotrope

Assessing Fluid Responsiveness

- If SV increases by 10% after fluid bolus = volume responsive.
- Keep increasing fluids until SV does not increase by 10%
- Then may need inotrope to push fluids around
- If SV does not increase by 10% after fluid bolus = contractility problem
- Add inotrope

Which patient is volume responsive?

<table>
<thead>
<tr>
<th></th>
<th>A</th>
<th>B</th>
<th>C</th>
</tr>
</thead>
<tbody>
<tr>
<td>SV</td>
<td>40</td>
<td>40</td>
<td>40</td>
</tr>
<tr>
<td>SV after 500ml bolus</td>
<td>43</td>
<td>45</td>
<td>50</td>
</tr>
</tbody>
</table>

Which patient is volume responsive?

<table>
<thead>
<tr>
<th></th>
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<tr>
<td>SV after 500ml bolus</td>
<td>43</td>
<td>45</td>
<td>50</td>
</tr>
</tbody>
</table>
Case Study

- 60 y/o admitted to ICU for sepsis related to above knee amputation 6 weeks ago.
- PMH
  - Atrial Fibrillation
  - COPD
  - Heart Failure with EF 45%
  - End Stage Renal Disease – on hemodialysis
  - Type 2 Diabetes

Treated per Sepsis bundles and responded. Transferred to Progressive unit 4 days after admission. It is now 16 hours after transfer from the ICU

- **Vital Signs: 4 hours earlier**
  - BP 91/52
  - HR 99 A Fib
  - RR 18 T 98.5 oral

- **Vital Signs Now**
  - BP 60/ doppled
  - HR 128 A Fib
  - RR 20

- Patient was dialyzed yesterday. BP typically drops to 70 – 80s post dialysis.

- Renal MD concerned about giving too much fluid as patient goes into pulmonary edema quickly.
- MD thinking about transferring the patient back to the ICU?
- What do you want to do?

USCOM

- SV 19
- SVI 9.2
- CO/CI 2.4/1.2

USCOM

- SV 19
- SVI 9.2
- CO/CI 2.4/1.2

- Was he fluid responsive?
- 500 ml Saline given
- BP 78/45
- SV 31
USCOM

- SV 19
- CO/CI 2.4/1.2
- 500 ml Saline given to total 1 liter given
- BP 98/50
- HR 88
- SV 49
- SVI 23
- CO/CI 4.1/2.0

Pt was sitting up eating lunch after the boluses and prevented a return to the ICU.

SVV
Stroke Volume Variation

- Only use
  - if on controlled rate ventilator
  - Regular rhythm (no Afib)
- Normal < 10-15%
  - > 15% likely to respond to fluids
  - 10 – 15% -- probably hypovolemic
- Value should decrease as give volume
- As SV ↑, SVV should ↓

Determinants of Cardiac Output

Cardiac Output = Heart Rate x Stroke Volume

- Preload
- Afterload
- Contractility

Preload

Myocardial Fiber-Stretch

How full is the tank (heart)?

Empty

Full
**Clinical Measurement of PRELOAD**

- **LEFT VENTRICLE = LVEDP**
  - Pulmonary Artery Wedge Pressure: 8-12 mm Hg
  - Pulmonary Artery Diastolic: 8-15 mm Hg

- **RIGHT VENTRICLE = RAP**
  - Right Atrial Pressure measures the pre-load of RV [normal range 2-5 mm Hg]
  - CVP 4 to 10 mm Hg

**Decreased Preload**

- **Etiology**
  - Hypovolemia
  - Arrhythmias
  - Loss of “Atrial Kick”
  - Venous Vasodilation

- **Cardiac Surgery Specific**
  - Underlying cardiac disease
  - Medications (preop, anesthesia, & vasoactive agents)
  - Procedural induced hypothermia
  - Rewarming
  - Bleeding

**Preload**

<table>
<thead>
<tr>
<th>Low Volume</th>
<th>High Volume</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diuretics</td>
<td>Venous vasodilators</td>
</tr>
</tbody>
</table>

**Decreased Preload**

- Anticipate that Cardiac Surgery patients will have a decrease in blood and plasma volume (preload) within the 1st 24 hours post op
- Watch for hypovolemia from rewarming and third spacing!
- FLUID- FLUID- FLUID
  - Drugs don’t work if there isn’t anything to pump!

**Which CABG patient needs volume?**

1. CVP 8 mm Hg, SI 35 ml/beat/M²
2. CVP 8 mm Hg, SI 42 ml/beat/M²
3. CVP 8 mm Hg, SI 20 ml/beat/M²

**Answer**

3. CVP 8 mm Hg and SI 20 ml/beat/M²
How Cardiac Meds effect preload

- Vasoconstrictors will increase preload when started
- Vasodilators will decrease preload when started

Afterload

- Afterload is the pressure the ventricle has to generate to overcome resistance to ejection.
- Any resistance against which the ventricle must pump in order to eject its volume

Afterload is measured as SVR and PVR

- Systemic Vascular Resistance (SVR) reflects LV afterload
  - Normal Range = 800-1500 dyne/sec/cm-5
- Pulmonary Vascular Resistance (PVR) reflects RV afterload
  - Normal Range = 20-120 dyne/sec/cm-5

Systemic Vascular Resistance (SVR)

Definition:
A measurement of impedance to left ventricular ejection.

Equation: $SVR = \frac{MAP - CVP}{CO} \times 80$

Normal Range: 800-1500 dyne.sec.cm\(^5\)
SVR
< 800 = vasodilated
> 1500 = vasoconstricted
High afterload (SVR) → heart is working harder

**CO and SVR**

\[
SVR = \frac{MAP - CVP}{CO} \times 80
\]

A "Teeter - Totter" Relationship

Most Hypovolemic patients have high SVR due to low SV causing low CO. However, it is misleading to say the patient is dry if the SVR is high.

**Pulmonary Vascular Resistance (PVR)**

Definition:
A measurement of impedance to right ventricular ejection.

Equation:
\[
PVR = \frac{MPA - PCW}{CO} \times 80
\]

Normal Range: 20 - 120 dyne.sec.cm\(^{-5}\)

**Factors That Increase Pulmonary Vascular Resistance**

- **Chemical Stimuli**
  - Alveolar hypoxia
  - Acidosis
  - Hypercapnia
- **Pharmacologic Agents**
  - Epinephrine
  - Norepinephrine
  - Dobutamine
  - Phenylephrine
- **Hyperinflation**
  - Mechanical Ventilation
  - Continuous Positive Airway Pressure (CPAP)
  - Positive End Expiratory Pressure (PEEP)
- **Pathologic Factors**
  - Vascular Blockage
    - Pulmonary emboli, air bubbles, tumor mass
  - Vascular wall disease
    - Sclerosis, endarteritis, polyarteritis, scleroderma
  - Vascular destruction
    - Emphysema
  - Vascular compression
    - Pneumothorax, hemothorax
  - Tumor mass
- **Humoral Substances**
  - Histamine, angiotensin, fibrinopeptides
  - Prostaglandin F\(_2\alpha\)
  - Serotonin
Factors That **Decrease** Pulmonary Vascular Resistance

<table>
<thead>
<tr>
<th>Pharmacologic Agents</th>
<th>Humoral Substances</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Oxygen</td>
<td>• Acetylcholine</td>
</tr>
<tr>
<td>• Isoproterenol</td>
<td>• Bradykinin</td>
</tr>
<tr>
<td>• Aminophylline</td>
<td>• Prostaglandin E</td>
</tr>
<tr>
<td>• Calcium channel blocking agents</td>
<td>• Prostacyclin</td>
</tr>
<tr>
<td>• Nitrous Oxide</td>
<td>• Sildenafil (Viagra)</td>
</tr>
</tbody>
</table>

**Afterload**

<table>
<thead>
<tr>
<th>Decreased</th>
<th>Increased</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vasodilation</td>
<td>Right</td>
</tr>
<tr>
<td>Vasodilation from rewarming</td>
<td>Pulmonary hypertension</td>
</tr>
<tr>
<td>Vasodilator therapies</td>
<td>Hypoxemia</td>
</tr>
<tr>
<td>Presp beta blockers</td>
<td>Pulmonic stenosis</td>
</tr>
<tr>
<td>Sepsis</td>
<td>Left</td>
</tr>
<tr>
<td>Pulmonary hypertension</td>
<td>Severe LV dysfunction</td>
</tr>
<tr>
<td>Hypoxemia</td>
<td>Vasoconstriction</td>
</tr>
<tr>
<td>Pulmonic stenosis</td>
<td>Vasopressors</td>
</tr>
<tr>
<td>Hypothermia</td>
<td>↑ catecholamine simulation from surgery</td>
</tr>
</tbody>
</table>

The Effect of Cardiac Meds on Afterload

**Increase Afterload**

- Dopamine/Intopin
- Epinephrine/Adrenalin
- Norepinephrine/Levophed
- Phenylephrine/Neo-synephrine
- Vasopressin/Pitressin

**Minimal effect on afterload**

- Dobutamine/Dobutrex

**Decrease Afterload**

- Nitroprusside/Nipride
- Arterial vasodilator
- Nitroglycerin/Tridil
- Venous vasodilator
- Beta Blockers
- Nicardipine/Cardene
- ACE Inhibitors

**Slight Decrease Afterload**

- Milrinone/Primacor

**Afterload**

<table>
<thead>
<tr>
<th>Low</th>
<th>High</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vasopressors</td>
<td>Warming blanket</td>
</tr>
<tr>
<td>Vasodilators</td>
<td>Calcium channel blockers</td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td>IABP</td>
</tr>
</tbody>
</table>
Contractility
Cardiac Squeeze

Increased Contractility
- Sympathetic stimulation
- Metabolic states:
  - Hypercalcemia
- Inotropic therapies:
  - Epinephrine
  - Dopamine
  - Digoxin
  - Calcium
  - Dobutamine
  - Milrinone

Decreased Contractility
- Parasympathetic stimulation
- Negative inotropic therapies
  - Beta blockers
  - Calcium channel blockers
- Metabolic states:
  - Acidosis
  - Hyperkalemia
  - Myocardial ischemia/infarct
  - #1 negative inotope is acidosis!

Etiology of ↓ contractility
- Cardiac surgery
- Acidosis
- ↑ or ↓ preload
- ↑ afterload
- Factors that affect myocardial contractility directly
  - Ischemia
  - RV or LV failure
  - Aneurysms
  - Electrolyte imbalances
  - Tamponade

Acidosis is the #1 negative inotrope!
- Acidosis decreases cardiac contractility!
- Treat acidosis so inotropes work!

How Cardiac Meds effect Contractility
The Effect of Cardiac Meds on Contractility

**Increase Contractility**
- Calcium
- Dopamine/Intopin
- Epinephrine/Adrenalin
- Norepinephrine/Levophed
- Dobutamine/Dobutrex
- Milrinone/Primacor

**Decrease Contractility**
- Beta Blockers
- Calcium Channel Blockers
- Nicardipine/Cardene
- Lidocaine/Xylocaine

Treating Low Contractility

- Optimize preload & afterload
- Treat underlying causes
- Inotropes
- IABP
- Ventricular assist devices

Cardiac Output Pearls

<table>
<thead>
<tr>
<th>LOW</th>
<th>CARDIAC OUTPUT Treatment Options</th>
<th>HIGH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Volume</td>
<td>PRELOAD</td>
<td>CVP, PA, PAOP</td>
</tr>
<tr>
<td>Vasopressors</td>
<td>AFTERLOAD</td>
<td>SVR, PVR</td>
</tr>
<tr>
<td>Optimize preload &amp; afterload</td>
<td>CONTRACTILITY</td>
<td>CO/CI indirect measurement</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>RATE/RHYTHM</td>
<td>Beta Blockers</td>
</tr>
</tbody>
</table>

Pearls

- Make sure adequate preload before starting inotrope
- Low preload $\rightarrow$ FLUID
- Drugs don’t work if there isn’t anything to pump

Pearls – what to wean first?

- Wean medication that impacts the most stable parameter first
- Wean most potent medication first
- Vasopressin & Epinephrine $\rightarrow$ potent vasoconstrictors
  - Decrease blood flow to microcirculation
  - $\uparrow$ MvO$_2$

Drug Pearls

- Epinephrine $\rightarrow$ 1st line drug for borderline cardiac output
- Dopamine $\rightarrow$ 1st line drug for low CO state. Also useful to increase urine output
- Dobutamine $\rightarrow$ Most useful when CO is marginal & mild $\uparrow$ SVR. Moderate pulmonary dilator
- Milrinone $\rightarrow$ used for persistent low CO, RV dysfunction, diastolic dysfunction
- Norepinephrine $\rightarrow$ Low CO with low BP caused by low SVR
- Neo-synephrine $\rightarrow$ used to $\uparrow$ SVR when hypotension exists with normal CO
- Vasopression $\rightarrow$ Refractory vasodilatory shock, $\downarrow$ SVR

Pearls – Management of Low Cardiac Output Syndrome

- Look for non cardiac correctable causes (resp, acid/base, electrolytes)
- Treat ischemia or coronary spasm
- Optimize HR 90 – 100 bpm with pacing
- Control arrhythmias
- Assess CO & start inotrope if CI < 2
  - Epinephrine unless arrhythmias or tachycardia
  - Dopamine if low SVR or Dobutamine if high SVR
  - Milrinone/minstrinone

Pearls – Management of Low Cardiac Output Syndrome (cont)

- Start vasodilator if SVR >1500
  - Nitroprusside if high filling pressures, SVR, BP
  - Nitroglycerine if high filling pressures or evidence of coronary ischemia or spasm
- If SVR low
  - Norepinephrine if marginal CO
  - Phenylephrine if satisfactory CO
  - Vasopressin 0.01 – 0.07 units/minutes if satisfactory CO
- Blood transfusion if Hct < 26%
- IABP if refractory to pharmacologic interventions
- Ventricular Assist device if no response to above


Source Barbara McLean

Source Barbara McLean

Source Barbara McLean

Source Barbara McLean
If what you are doing isn’t working, change strategies!

They say “the definition of insanity is doing the same thing over and over again and expecting different results.”

So why are you still doing it?

Medications for Low Cardiac Output

**Alpha Receptors**
- Noradrenaline
- Phenylephrine

**Beta Receptors**
- Dobutamine
- Isoproterenol

**Beta 1**
- Heart
- Contractility
- Conduction velocity
- Automaticity
- Renin release

**Beta 2**
- Arteries & veins
- Lungs, Kidney
- Vasodilation
- Bronchodilation

* = combined alpha & beta stimulation

**Vasodilation**
- Dopamine
- Epinephrine
- Norepinephrine (Levophed)
- Other
  - Vasopressin – Hormone (Stimulates V1 receptors)
  - Milrinone (Primacor) – phosphodiesterase inhibitor (+ inotrope, vasodilator)

**Vasoconstriction**
- Neosynephrine
- Phenylephrine

** = combined alpha & beta stimulation

**Draw arrows to indicate if the hemodynamic parameters would be increased, decreased or normal.**

<table>
<thead>
<tr>
<th>Hypovolemia</th>
<th>Fluid Overload</th>
<th>LV failure</th>
<th>RV failure</th>
<th>RV &amp; LV failure</th>
<th>Sepsis</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO/CI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CVP</td>
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<tr>
<td>PAD</td>
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</tr>
<tr>
<td>SW/SVI</td>
<td></td>
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<tr>
<td>SVW/SVRI</td>
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<tr>
<td>PVR/PVRI</td>
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</tr>
</tbody>
</table>

**Table 11.5 - Hemodynamic Effects of Vasopressor Medications**

<table>
<thead>
<tr>
<th>Medication</th>
<th>SVR</th>
<th>HR</th>
<th>PCWP</th>
<th>CI</th>
<th>MAP</th>
<th>M-O2</th>
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<tbody>
<tr>
<td>Dopamine</td>
<td></td>
<td></td>
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<tr>
<td>Epinephrine</td>
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<tr>
<td>Norepinephrine</td>
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<td></td>
</tr>
<tr>
<td>Milrinone/ Primacor</td>
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<td></td>
</tr>
<tr>
<td>Isoproterenol</td>
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<td></td>
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<tr>
<td>Carvedilol</td>
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<tr>
<td>Phentolamine</td>
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<td>Phenylephrine</td>
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<td>Vasopressin</td>
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</tr>
<tr>
<td>Methyl-xanthine</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

*↑ increased, ↓ decreased, ↔ no change, / variable effect. The relative effect is indicated by the number of arrows.

**Hypovolemia**

<table>
<thead>
<tr>
<th>Hypovolemia</th>
<th>CO/CI</th>
<th>CVP</th>
<th>PAD</th>
<th>SW/SVI</th>
<th>SVW/SVRI</th>
<th>PVR/PVRI</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO/CI</td>
<td>↓</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CVP</td>
<td></td>
<td>↓</td>
<td></td>
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</tr>
<tr>
<td>PAD</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>SVW/SVRI</td>
<td>Normal/Increased</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PVR/PVRI</td>
<td>Normal</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
# Fluid Overload

<table>
<thead>
<tr>
<th>Hypovolemia</th>
<th>Fluid Overload</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO/CI</td>
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<tr>
<td>CVP</td>
<td>↓</td>
</tr>
<tr>
<td>PAD</td>
<td>↓</td>
</tr>
<tr>
<td>SV/SVl</td>
<td>↓</td>
</tr>
<tr>
<td>SVr/SVrl</td>
<td>Normal/increased</td>
</tr>
<tr>
<td>PVR/PVrl</td>
<td>Normal</td>
</tr>
</tbody>
</table>

# LV Failure

<table>
<thead>
<tr>
<th>Hypovolemia</th>
<th>Fluid Overload</th>
<th>LV failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO/CI</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>CVP</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>PAD</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>SV/SVl</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>SVr/SVrl</td>
<td>Normal/increased</td>
<td></td>
</tr>
<tr>
<td>PVR/PVrl</td>
<td>Normal</td>
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</tbody>
</table>

# RV Failure

<table>
<thead>
<tr>
<th>Hypovolemia</th>
<th>Fluid Overload</th>
<th>LV failure</th>
<th>RV failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO/CI</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>CVP</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>PAD</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>SV/SVl</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>SVr/SVrl</td>
<td>Normal/increased</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PVR/PVrl</td>
<td>Normal</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

# RV & LV Failure

<table>
<thead>
<tr>
<th>Hypovolemia</th>
<th>Fluid Overload</th>
<th>LV failure</th>
<th>RV failure</th>
<th>RV &amp; LV failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO/CI</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
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<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>PAD</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>SV/SVl</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>SVr/SVrl</td>
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<tr>
<td>PVR/PVrl</td>
<td>Normal</td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

# Hemodynamics Let’s Practice!

Hemodynamics Let’s Practice!

http://pie.med.utoronto.ca/edwards
Case #1

What's abnormal?

<table>
<thead>
<tr>
<th>Case #1</th>
<th>CABG on admission (Dopamine 2.5 mcg/kg/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO/CI</td>
<td>3.7/1.8</td>
</tr>
<tr>
<td>SBP/DBP</td>
<td>115/53</td>
</tr>
<tr>
<td>MAP</td>
<td>71</td>
</tr>
<tr>
<td>HR</td>
<td>85</td>
</tr>
<tr>
<td>SvO₂</td>
<td>38</td>
</tr>
<tr>
<td>CVP</td>
<td>9</td>
</tr>
<tr>
<td>PAS/PAD</td>
<td>26/16</td>
</tr>
<tr>
<td>PAW</td>
<td>20</td>
</tr>
<tr>
<td>SV</td>
<td>44</td>
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<tr>
<td>SVR</td>
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<tr>
<td>SVRI</td>
<td>2779</td>
</tr>
<tr>
<td>PVR</td>
<td>22</td>
</tr>
<tr>
<td>PVRI</td>
<td>45</td>
</tr>
</tbody>
</table>

How do you want to treat?

1. Fluid
2. Increase dopamine
3. Decrease dopamine
4. Add another pressor

Answer

How do you want to treat?

1. Fluid

30 minutes later after 250 ml 5% albumin

<table>
<thead>
<tr>
<th>Case #1</th>
<th>CABG on admission (Dopamine 2.5 mcg/kg/min)</th>
<th>30 minutes later after 250 ml 5% albumin</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO/CI</td>
<td>3.7/1.8</td>
<td>4.9/2.4</td>
</tr>
<tr>
<td>SBP/DBP</td>
<td>115/53</td>
<td>123/55</td>
</tr>
<tr>
<td>MAP</td>
<td>71</td>
<td>74</td>
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<tr>
<td>HR</td>
<td>85</td>
<td>88</td>
</tr>
<tr>
<td>SvO₂</td>
<td>38</td>
<td>39</td>
</tr>
<tr>
<td>CVP</td>
<td>9</td>
<td>10</td>
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<tr>
<td>PAS/PAD</td>
<td>26/16</td>
<td>29/18</td>
</tr>
<tr>
<td>PAW</td>
<td>20</td>
<td>21</td>
</tr>
<tr>
<td>SV</td>
<td>44</td>
<td>46</td>
</tr>
<tr>
<td>SVR</td>
<td>1339</td>
<td>1055</td>
</tr>
<tr>
<td>SVRI</td>
<td>2779</td>
<td>2166</td>
</tr>
<tr>
<td>PVR</td>
<td>22</td>
<td>33</td>
</tr>
<tr>
<td>PVRI</td>
<td>45</td>
<td>68</td>
</tr>
</tbody>
</table>
CABG on admission

Dopamine 2.5 mcg/kg/min
30 minutes later after 250 cc 5% albumin & Dopamine 1 mcg/kg/min

| CO/CI | 3.7/1.8 | 4.9/2.4 | 6.5/3.1 |
| SP/BP | 115/53  | 123/55  | 133/40  |
| MAP   | 71      | 74      | 69      |
| HR    | 85      | 88      | 75      |
| SV0   | 38      | 39      | 55      |
| CVP   | 9       | 10      | 12      |
| PAS/PAD | 260/16 | 28/18   | 40/19   |
| PAM   | 21      | 23      | 27      |
| SVW   | 20      | 21      | 26      |
| SV    | 44      | 56      | 86      |
| SVR   | 1339    | 1055    | 701     |
| SVRI  | 2779    | 2166    | 1495    |
| PVR   | 22      | 33      | 12      |
| PVRI  | 45      | 68      | 26      |

36 hours later after 500 cc 5% albumin & Dopamine 1 mcg/kg/min

| CO/CI | 3.3/1.5 |
| SP/BP | 107/47  |
| MAP   | 66      |
| HR    | 67      |
| SV0   | 62      |
| CVP   | 10      |
| PAS/PAD | 37/19 | |
| PAM   | 26      |
| SV    | 50      |
| SVR   | 1259    |
| PVR   | 179     |

Case 2: Identify abnormal hemodynamic parameters and what you would do?
Dopamine 2.5 mcg/kg/min, Epi 3.07 mcg/min, Milrinone 0.5 mcg/kg/min

1. Treat hypovolemia
2. Treat tamponade
3. Treat cardiogenic shock
4. Treat fluid overload

Case 2: Identify abnormal hemodynamic parameters and what you would do?

1300

Art BP 118/71
MAP 80
HR 107
PAS/PAD 37/26
CVP 23
SV0 45
CI 4.2
CO 4.4
SVR 1316
SpO2 95
SV 39
UO 60

Case 2 Answer: Tamponade. If cardiogenic shock would expect a higher SVR and CVP would be lower. Treatment= reexploration of chest.

Dopamine 2.5 mcg/kg/min, Epi 3.07 mcg/min, Milrinone 0.5 mcg/kg/min.

What’s abnormal?
- NSTEMI 2 days ago with EF 30%
- PMI
- Stent to RCA = 5 years ago
- Moderate COPD
- Smoker
- Diabetes
- RBBB
How do you want to treat?

1. Fluid
2. Increase phenylephrine
3. Decrease phenylephrine
4. Add another pressor

- NSTEMI 2 days ago with EF 30%
- PMH
  - Stent to RCA = 5 years ago
  - Moderate COPD
  - Smoker
  - Diabetes
  - RBBB

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250 ml 5% albumin x 2 = 500 ml Phenylephrine at 50mcg/min

Was he fluid responsive to the 500 ml Albumin?

1. Yes
2. No

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250 ml 5% albumin x 2 = 500 ml Phenylephrine at 50mcg/min

He needs something to pump the fluid (increase the contractility). What do you want to use?

1. Dopamine
2. Increase Phenylephrine
3. Epinephrine
4. Milrinone
5. Calcium

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250 ml 5% albumin x 2 = 500 ml Phenylephrine at 50mcg/min

Was he fluid responsive to the 500 ml Albumin?

1. No

He needs something to pump the fluid (increase the contractility). What do you want to use?

1. Dopamine
2. Increase Phenylephrine
3. Epinephrine
4. Milrinone
Drug Pearls

- Epinephrine → 1st line drug for borderline cardiac output
- Dopamine→ 1st line drug for low CO state. Also useful to increase urine output
- Dobutamine→ Most useful when CO is marginal & mild ↑ SVR. Moderate pulmonary dilator
- Milrinone→ used for persistent low CO, RV dysfunction, diastolic dysfunction
- Norepinephrine→Low CO with low BP caused by low SVR
- Neo-synephrine→Used to ↑ SVR when hypotension exists with normal CO
- Vasopression→ Refractory vasodilatory shock, ↓ SVR


What do you want to do?
1. Fluids
2. Pressors

What if you have one hemodynamic value you can't remember the normal?

Don't PANIC!

GO WITH WHAT YOU KNOW!

Practice!
http://pie.med.utoronto.ca/edwards