Cardiac Surgery
Pearls of Wisdom
Fun and Focused
Class M180M613

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Objectives
- Differentiate plan of care for cardiac surgery patients with CABG and valvular surgery/repair
- Relate hemodynamic concepts of preload, afterload, and contractility to pharmacological treatments in cardiac surgery patients
- Discuss assessment cues and management of cardiac surgery patients to prevent and treat complications associated with cardiac surgery

And have fun 😊

Not studying for CSC
“No worries”

Fun and Focused
1. Coronary Bypass Surgery and 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!

Coronary Artery Bypass Surgery
Venous Conduits or grafts
- Saphenous vein
  - With long leg incisions, graft vulnerable to platelet aggregation so need **antithrombotic** therapy to prevent graft closure
  - Persantine & ASA

Arterial Grafts/conduits
- Longer patency than venous grafts
- LIMA/RIMA
- Radial artery
- Gastroepiploic Artery (stomach)

LIMA/RIMA
- LIMA – LAD
- RIMA – RCA
- Resistant to atherosclerosis
- Only one anastomosis

LIMA/RIMA Complications:
- Phrenic nerve devascularization – LIMA
- Can cause inability or delayed vent weaning
- Spasm (ST segment changes)
  - Diltiazem or Nitroglycerin
  - Steal syndrome
  - Sternal ischemia
- Brachial plexus injury
  - Limp or paralyzed arm, lack of muscle control in upper extremity
  - Pulmonary complications due to pleural dissection
  - Pleural effusion

Radial Artery conduit
**Preop**
- Assess ulnar function by doing Allen’s test
- **Positive Allen’s test > 6 seconds** = contraindication to radial artery use
- Doppler tests to assess for collateral circulation

**Contraindications**
- Manual labor using hands
- Stroke with upper extremity weakness
- Reynaud’s disease

Radial Artery conduit Post op
- May have drain
- Assess the 6 “Ps” for arterial blood flow
  - Pain
  - Pulselessness
  - Pallor
  - Paresthesia
  - Paralysis
  - Polar (cold)
- May experience loss of motor strength and numbness (6 months)
  - Encourage hand/fist exercises
- Spasms
  - Diltiazem
Cardiopulmonary Bypass (CPB) vs OPCAB

Cardiopulmonary Bypass (CPB)
- Utilize cardioplegic to stop heart
  - High in potassium, bicarb
  - May need to pace until cardioplegia wears off
  - Mannitol to decrease brain edema
  - Mannitol causes diuresis post op

Cardiopulmonary Bypass (CPB) Complications
- Systemic inflammatory response syndrome (SIRS)
  - Vasoactive substances generated
  - Fluid retention and fluid shifts
  - Organ dysfunction
  - Coagulation disruption
- Utilize hypothermia to ↓ myocardial O₂ demand
  - Rewarming contributes to vasodilatation and can worsen effects of SIRS
- Nonpulsatile
  - Neurologic dysfunction
  - Bleeding due to effect on RBCs and platelets and decreasing coagulation factors
  - Renal failure
- Heparin complications
  - Bleeding or HIT

Cardiopulmonary Bypass (CPB) Advantages
- Less cerebral hypoperfusion
- Less embolization
- Less SIRS from CPB
- Less bleeding
  - Use about 1/3 to ½ less heparin than onpump CABG

Off Pump Coronary Artery Bypass: OPCAB (beating heart surgery)
- No Cardioplegia – heart is still beating
- May be utilized for
  - Medial sternotomy
  - Able to do four or five vessel revascularization
  - MIDCAB
  - Only able to do one or two vessel revascularization
  - Robotic (ROBOCAB)
- Mild hypothermia so less bleeding from hypothermia

Mannitol to decrease brain edema
- Mannitol causes diuresis post op
Off Pump CABG

MICCS (Minimally Invasive Cardiac Surgery)
Types:
- Mini-thoracotomy incision without use of CPB (MIDCAB)
- Endoscopic approach with CPB utilizing femoral cannulation
- Robotic
Disadvantages:
- Unable to access posterior heart for revascularization
Contraindication:
- Difficulty locating the LAD

MIDCAB (Minimally Invasive Direct Coronary Artery Bypass)
- Mini-thoracotomy incision without use of CPB
  - 3-4 inch incision made between the ribs
  - Heart is beating
- Utilized for LAD and RCA
- Only able to revascularize one or two vessels
- Unable to access posterior heart for revascularization

MIDCAB Advantages
- Less pain
- Earlier mobilization
- Shorter ICU LOS and hospital LOS
- Decreased sternal wound infections
- Lower mortality and morbidity

Robot-Assisted Coronary Artery Bypass

Robotic Heart Surgery
- MV Repair
- MVR
- AVR
- Myxoma
- VSD
- TV repair
- CABG
Robotics Cannulation

- Right femoral artery
- Right femoral vein and right jujular
- Monitor during OR for tissue perfusion
- If high risk, put in a catheter to perfuse the right leg
- May be occlusive & ischemia to the right leg
- DO NOT put warming blanket over right leg
- -- let warm naturally

Robotics Intubation

- Intubate with double lumen ET tube so can drop the right lung
- The right lung becomes atelectic with no blood flow
- When reinflate may get “reperfusion syndrome” or bleeding

Robotics

- Pain is from spreading the ribs and the chest tubes
- Exercise/Activity to patient discomfort
- Most bleed very little

MICS Preop Teaching

- Decreased amount of post op pain
- Aggressive pulmonary toilet
- Early ambulation
- Earlier discharge

MICS Nursing Care – Post op First 15 minutes

- Mostly same as those with sternotomy
- In depth report
- Assessment of vital signs, PA pressures, labs, U/O, CT output
- Hemodynamic stability
- Level lines – connect CT to suction
- Warm patient if had CPR (Hypothermia)

MICS Nursing Care – Post op The Next 15 minutes

- Preliminary assessment for clinical issues
  - Bleeding, hyper/hypotension, agitation, arrhythmias
- Head to toe assessment
- Rewarming
MICS Specific Complications

- Typically related to the more technically challenging nature of these procedures and the procedure related stress on the heart.
  - Dysrhythmias – A Fib, VT
  - Hypotension
  - MI
  - Bleeding
  - Brain Injury

MICS Ventilatory Support

- May extubate in OR
- Extubate 3–6 hours
- Extubation criteria
  - ABCs within parameters
  - Hemodynamically stable
  - Normal CXR
  - Normothermia
  - CT output < 100 ml/hour prior to extubate
  - UO > 1 ml/kg prior to extubate

Cardiac Surgery Recovery

<table>
<thead>
<tr>
<th>Procedure</th>
<th>ICU LOS</th>
<th>Hospital LOS</th>
<th>Return to normal activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>MIDCAB</td>
<td>1 day</td>
<td>3 days</td>
<td>2 weeks</td>
</tr>
<tr>
<td>OPCAB</td>
<td>1 day</td>
<td>5–7 days</td>
<td>2–3 months</td>
</tr>
<tr>
<td>Traditional CABG</td>
<td>1–3 days</td>
<td>5–10 days</td>
<td>2–3 months</td>
</tr>
</tbody>
</table>

Rewarming — all cardiac surgery patients

- Causes vasodilation → ↓ BP and filling pressures
- Use volume and pressors
- May get postop cognitive impairment due to cerebral hyperthermia if warmed too fast

Hypothermia

More common with CPB

- Bleeding, platelet dysfunction and impairment of the coagulation cascade
- May stimulation the SNS leading to:
  - Hemodynamic instability
  - Dysrhythmias
  - Vasoconstriction, hypertension, and increased SVR
- Shivering
  - Increased oxygen consumption (↓ SVO₂) and CO₂ production
  - Adrenergic stimulation
  - Use Demerol to treat

Bleeding risk factors

- Greater risk if CPB - more heparin
- Hemodilution
- Fibrinolysis
- Hypothermia
- If off pump patient is bleeding, it is usually surgical in nature
- Dark blood = venous or older blood
- Bright red (warm) blood = arterial or fresh blood
Valvular Heart Disease

<table>
<thead>
<tr>
<th>Congenital</th>
<th>Acquired</th>
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<tbody>
<tr>
<td>Bicuspid vs tricuspid valve</td>
<td>Ischemic CAD</td>
</tr>
<tr>
<td>Marfans – connective tissue disorder</td>
<td>Degenerative changes with aging</td>
</tr>
<tr>
<td></td>
<td>Rheumatic changes</td>
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<td></td>
<td>Infective endocarditis</td>
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<td></td>
<td>Neoplasm</td>
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<td></td>
<td>Thrombosis</td>
</tr>
</tbody>
</table>

Valvular Heart Disease

- Mitral
- Aortic
- Tricuspid
- Pulmonic

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

Valvular Heart Disease

- Rheumatic heart disease
- Degenerative diseases
- Infective endocarditis

Valvular Heart Disease

- Cardiac compensatory mechanisms can maintain stability for years before symptoms occur.
- Key is early diagnosis to prevent the long-term consequences
  - Pulmonary hypertension
  - Heart Failure
  - Atrial fibrillation
  - Thromboembolism
- Important to understand
  - The structure and function of the valves
  - The causes and treatments of each disorder
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

Aortic Stenosis

Pathophysiology

1. Small opening causes ↓ blood flow and ↓ CO
2. ↑ Afterload
3. ↑ Workload in Left ventricle
4. ↑ Pressure in LV
5. LV hypertrophy
6. LV becomes stiff
7. High LV pressures leads to LV failure and left atrial enlargement
8. High pressures transmitted to the lungs leads to pulmonary hypertension, pulmonary edema and right sided failure

LV size and contractility = abnormal in AS

Clinical Presentation

- Heart Sounds
  - Systolic ejection murmur (harsh) at the right sternal border radiating to neck
- CXR
  - May be normal
- EKG
  - Left ventricular hypertrophy with severe AS
  - Left Atrial enlargement, Left axis deviation, Left BBB

Syncope --- Systolic ejection harsh murmur

Surgical Treatment for Aortic Valve Disease

- Aortic Valve Replacement (mainstay)
- Aortic Valve Repair (not mainstream)
- Percutaneous or Transapical replacement

Post op AS

- Inotropes rarely needed except in patient with impaired LV function – may be caused by myocardial stunning
- Avoid ↑ BP in immediate post-op period – “thin” aorta in bicuspid valve patients more likely to rupture under high pressure
Aortic Regurgitation/Insufficiency

- Aortic valve fails to close completely
- Backflow of blood into the left ventricle during diastole

**Causes**
- Pathological conditions of the aortic valve cusps or aortic root
- Valve cusps
- Rheumatic heart disease
- Infective endocarditis
- Aortic Root
- Hypertension
- Aortic dissection
- Marfan’s Syndrome
- Syphilis
- Severe AI – most frequently caused by bicuspid valve

**Pathophysiology**
1. Volume overload leads to compensatory mechanisms:
   - Left ventricular hypertrophy
   - ↑ End-diastolic volume which allows normal EF despite ↑ afterload.
2. ↑ LV afterload as the ↑ volume ejected into the high pressured aorta.
3. Ventricular wall thickening with dilatation to accommodate volume overload.
4. Ventricular wall thickens without enlargement but with diminished capacity to accommodate pressure overload.
5. The balance between afterload excess, preload reserve, and hypertrophy may continue for decades
6. Symptoms occur when this balance can’t be maintained and there is a reduction in EF and diastolic function

AI – unique with both volume & pressure overload

Aortic Regurgitation/Insufficiency

- **Clinical Presentation**
  - May be asymptomatic for years
  - Fatigue
  - Dyspnea on exertion
  - Angina
  - Palpitations
  - Widen pulse pressure
  - > 50 mmHg

- **Heart Sounds**
  - Decrescendo diastolic blowing murmur – best heard in upright position and leaning forward.
  - CXR
    - Enlarged heart
    - Dilatation of proximal aorta
  - EKG
    - Left ventricular hypertrophy

Aortic Regurgitation/Insufficiency

- **Clinical Presentation**
  - Austin Flint murmur
  - Decrescendo diastolic blowing murmur
  - Hill Sign
    - Systolic BP in lower extremities is at least 20mmHg higher than arms
  - Duroziez sign
    - Systolic and diastolic bruit heard when femoral artery is compressed by stethoscope
  - Corrigan pulse
    - ↑ volume and rate of rise of the radial pulse when the wrist is elevated perpendicular to the body of a supine patient
  - de Musset sign
    - Bobbing of the head with each systolic beat

Aortic Regurgitation

- Valve leaflets fail to close tightly
- Imposes large volume → ↑ end diastolic volume (preload) = ↑ LVEDP and ↓ CO
- Present with HF = LV failure
- Audible diastolic blowing murmur

Post op AVR for AR

- Due to dilated ventricle use
  - IV vasodilators
  - Inotropic support to promote ventricular emptying
    - Milrinone
    - Dobutamine
    - IABP
Post op Aortic Valve Replacement
- Maintain preload to maintain SV
- Treat arrhythmias quickly
- Keep HR as close to normal – avoid bradycardia
- Avoid increases in SVR
- Decrease HTN to reduce wall stress and ↑ LV function
- Use myocardial depressants (CCB & BB) with caution
- Beta agonists are beneficial

AVR
Choice of Replacement Device
- Age less than 60–65
  - Mechanical prosthesis: need for coumadin
- Age greater than 60–65
  - Tissue prosthesis/no coumadin/issue of longevity of prosthesis

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

Mitral Valve
- Cause
  - Mostly rheumatic fever
  - Some calcified degenerative disease
  - Continuous, slow progressive disease with symptoms appearing 20–40 years after rheumatic fever

Mitral Stenosis
- Pathophysiology
  1. Small opening causes ↓ blood flow and ↓ CO
  2. ↑ workload in Left Atrium
  3. ↑ pressure in LA
  4. LA dilation & hypertrophy
  5. May lead to A fib
  6. LA blood flow stagnant
  7. May cause clot formation & thromboembolism
  8. ↑ in LA pressure → backflow into pulmonary artery
  9. Leads to pulmonary hypertension, congestion, right ventricular hypertrophy and right sided heart failure

Mitral Stenosis
- LV size and contractility = normal in MS

Mitral Stenosis
- Clinical Presentation
  - Mild MS
    - Dyspnea on exertion from pulmonary congestion
  - Moderate MS
    - Fatigue
    - Paroxysmal nocturnal dyspnea
    - Atrial fibrillation
  - Severe MS
    - Dyspnea on mild exertion or at rest

Mitral Stenosis
- Pulmonary congestion & right sided failure signs
  - Ruddy face (mitral facies)
  - Heart Sounds
    - Loud first heart sound S1 (closing snap)
    - Mid-diastolic murmur/rumble at apex
    - Opening snap
  - Right sided S3 & S4
  - Right ventricular heave at sternum
  - CXR
    - Left atrial and RV enlargement
    - Pulmonary congestion
  - EKG
    - LA enlargement: Notched P waves, P mitrale
    - Right ventricular hypertrophy
    - Tall R waves V1 & V2
Mitral Stenosis
Clinical manifestations that affect post op care

- Nt LV function
- Pulmonary Hypertension
- RV failure
- Tricuspid insufficiency
- High left atrial pressures and pulmonary pressures
- Low CO and pulmonary congestion

Mitral Valve Replacement

Mitral Commisurotomy for MS

- Percutaneous mitral balloon valvulotomy (PMBV)

Mitral Regurgitation

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

Mitral Regurgitation/Insufficiency

Pathophysiology

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

MR = LA enlargement, Left or 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

Causes
- Anything that affects any part of the MV
- Mitral annulus
- Valve leaflets
- Chord tendineae
- Papillary muscle
- Mitral Valve Prolapse
- Rheumatic heart disease
- Infective endocarditis
- Cardiomyopathy
- Ischemic heart disease

Pathophysiology

1. During systole, a portion of blood is ejected back into the LA
2. ↓ blood in LV → ↓ CO
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4. During diastole, blood continues to flow into LV → ↑ LV volume
5. LV hypertrophy

MR = LA enlargement, Left or Ventricular Failure
Mitral Regurgitation

Clinical Presentation

- May be asymptomatic for years
- Initial symptoms
  - Left sided failure
  - Dyspnea on exertion
  - Cough
  - Peripheral Edema
  - Palpitations from new onset of A fib

Heart Sounds
- Holosystolic murmur – high pitched, blowing at apex
- S2 – widely split
- Displaced PMI laterally
- Maybe right sided S3 & S4

CXR
- Cardiomegaly
- LA enlargement
- LV hypertrophy
- Possible pulmonary congestion

EKG
- LA enlargement P mitrale
- Left and/or right ventricular hypertrophy
- Large QRS complexes

Endocarditis risk is RARE

Mitral Regurgitation

- Backjet of blood into left atrium
- Large left atrium
- ↑ LVEDP and ↓ afterload

Mitral Valve Repair for MR

- Annuloplasty
- Open commissurotomy
- Primary, anterior and posterior leaflet repair
- Based on the type, extent, and location of the defect of the mitral valve.

Geometric mitral reconstruction (GMR)

- Annuloplasty ring is used to restore a more normal mitral valve anatomy
- Results in significant increases in EF and reduction in NYHA symptomatology.
- Indicated for patients with cardiomyopathy and MR

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
Mitral Valve Repair/Quadrangular Resection

- 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

Mitral Valve Repair vs Mitral Valve Replacement

- Rheumatic
- Endocarditis (esp IV drug abuser)
- Functional (most common form): secondary to left sided pathology often accompanied by pulm HTN
- Congenital: AV canal, VSD, Ebsteins, Myxoma

Surgery for Tricuspid Valve Disease

- Primary indication for tricuspid valve repair is severe TR in patients requiring surgery for mitral valve disease
- Tricuspid stenosis: don’t see often
- TR: with left sided lesion, right sided failure, mod to severe TR

Tricuspid Valve Disease

- Repair with annuloplasty ring (MC3) or sewn (DeVega)
- Replace (often with PPM)
- Excision (IV drug abuser)

Valve Replacement

<table>
<thead>
<tr>
<th>Mechanical</th>
<th>Biological/tissue</th>
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<tbody>
<tr>
<td>Man–made from metal alloys, pyrolite carbon and Dacron</td>
<td></td>
</tr>
<tr>
<td>More durable and last longer</td>
<td></td>
</tr>
<tr>
<td>Increased risk of thrombotic events</td>
<td></td>
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<tr>
<td>Need long-term anticoagulation therapy</td>
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<tr>
<td>Assess age and medication compliance</td>
<td></td>
</tr>
<tr>
<td>Constructed from bovine, porcine, and human cardiac tissue and may contain some man–made products</td>
<td></td>
</tr>
<tr>
<td>Less durable – tendency towards early calcification, tissue degeneration, and stiffening of the leaflets</td>
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</tr>
<tr>
<td>Do not require long–term anticoagulation therapy</td>
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</tbody>
</table>
Prosthetic Valve Thrombosis
- Diminished or absence of crisp, clicking sounds of mechanical valves
- Presence of new murmurs
- Pulmonary congestion
- Poor peripheral perfusion
- Systemic embolization
- May have acute deterioration

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?
- 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.
- **Ampicillin, Clindamycin, Cephalexin, Cefazolin, or Ceftriazone**
  - May be used if allergic or unable to take oral
  - See guidelines for specific doses

Infective Valve Endocarditis
- Medical therapy is first line treatment
- Surgery indicated for those patients with acute infective valve endocarditis and life-threatening heart failure or cardiogenic shock

Complications of heart valve surgery
- Venous thrombotic events
- AV Block
- Atrial dysrhythmias
- Heart failure
- Neurological complications
  - Stroke
  - TIA
- Renal insufficiency
- Respiratory insufficiency
- Myocardial infarction
- Sternal wound infections
Ms Leaky, a 47 y/o, had a MVR. Today on POD #4, she is being transferred to the progressive care unit.

What is the rhythm?
1. Sinus Tachycardia
2. Atrial Flutter
3. Sinus Rhythm
4. SVT

Valvular Surgery
Aortic Valve

<table>
<thead>
<tr>
<th>Aortic Stenosis</th>
<th>Aortic Regurgitation</th>
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<tr>
<td>Preop LV hypertrophy</td>
<td>LV hypertrophy</td>
</tr>
<tr>
<td>Post op LV may not anticipate in SVR and continue to pump hard</td>
<td>IV vasodilators to LV SVR</td>
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Valvular Surgery
Mitral Valve

<table>
<thead>
<tr>
<th>Mitral Stenosis</th>
<th>Mitral Regurgitation</th>
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</thead>
<tbody>
<tr>
<td>Preop Nx LV function</td>
<td>Enlarged left atrium</td>
</tr>
<tr>
<td></td>
<td>Both common to have atrial fibrillation</td>
</tr>
<tr>
<td>Post op Assess pulmonary hypertension (PVR)</td>
<td>Immediate I SVR due to no backflow of blood in LA</td>
</tr>
<tr>
<td></td>
<td>Pulmonary hypertension &amp; myocardial hibernation take time to reverse</td>
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<tr>
<td></td>
<td>Inotropes (Milrinone, Dobutamine) = epinephrine</td>
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<tr>
<td></td>
<td>IABP Monitor for RV failure</td>
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<tr>
<td></td>
<td>Treat atrial fibrillation</td>
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### Tables

<table>
<thead>
<tr>
<th>Heart Sounds</th>
<th>MS</th>
<th>MR</th>
<th>AS</th>
<th>AR/AI</th>
</tr>
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<tbody>
<tr>
<td>MS: Mid diastolic murmur at apex 5, 4</td>
<td>Holosystolic murmur high pitched mildly split S2 5, 4</td>
<td></td>
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<td></td>
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<tr>
<td>MR: Dypsnea Pulmonary Hypertension Pulmonary symptoms</td>
<td>Systolic injection murmur harsh at right sternal border</td>
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</tr>
<tr>
<td>AS: Periheral Edema Cough LV failure New onset AFib</td>
<td>Decreased RBCs diastolic blowing murmur - best heard sitting upright</td>
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<td></td>
</tr>
<tr>
<td>AR/AI: Syncope Dyspnea Angina Cyanosis Wide pulse pressure &gt; 50 mmHg</td>
<td>Breath sounds, auscultation</td>
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<table>
<thead>
<tr>
<th>Atrial Size</th>
<th>LA enlarged</th>
<th>LA enlarged</th>
</tr>
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<tbody>
<tr>
<td>Ventricular Size</td>
<td>LV normal</td>
<td>LV enlarged</td>
</tr>
</tbody>
</table>

| Ventricular Size | LV normal | LV enlarged |

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<th>LV hypertrophy</th>
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<td>LV may not anticipate in SVR and continue to pump hard</td>
<td>IV vasodilators to LV SVR</td>
</tr>
<tr>
<td>Avoid hypertension and stress on suture line</td>
<td>Inotropes to promote emptying LV: Milrinone/Dobutamine, IABP</td>
</tr>
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### Diagrams

- **Damage to His bundle may result in BBB or CHB**
- **Ms Leaky, a 47 y/o, had a MVR. Today on POD #4, she is being transferred to the progressive care unit.**

### Notes

- **Valvular Surgery Aortic Valve**
  - Preop LV hypertrophy
  - Post op LV may not anticipate in SVR and continue to pump hard

- **Valvular Surgery Mitral Valve**
  - Preop Nx LV function
  - Post op Assess pulmonary hypertension (PVR)
Cardiac Surgery
Hemodynamics/Medications

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

β-Blockers
Limit the donkey’s speed, thus saving energy

Beta Blocker “Olols”
Beta Blockade of the Sympathetic Nervous System

- Decrease oxygen demand
  - ↓ HR & contractility
  - Vasodilate
  - ↓ Afterload
  - ↓ O₂ wasteage

- Antiarrhythmic effect
- Increase oxygen supply
  - Increased diastolic perfusion
  - Less exercise vasoconstriction

Side effect: May promote spasm in vasospastic angina

Beta Blockers “Olols”
- Acebutolol Sectral
- Atenolol Tenormin
- Betaxolol Kerlone
- Bisoprolol Zabeta
- Metoprolol Lopressor
- Nadolol Corgard
- Pindolol Visken
- Propanolol Inderal
- Timolol Blocadren

ACE Inhibitors/ARBs
Reduce the number of sacks on the wagon

RAAS Blockers Dilation & Diuresis
Body’s Response to Low Perfusion

Renin-Angiotensin-Aldosterone System (RAAS) Kicks in...

- Low Cardiac Output/Hypotension/Hypovolemia
- Decreased Renal perfusion
- Afferent Arteriole (baroreceptors)
- Release Renin (a messenger)
- Go to Liver to stimulate Angiotensin I production
- Angiotensin I goes to the Lung
  - Angiotension Converting Enzyme (ACE) located in the pulmonary vascular membrane
  - Converts Angiotensin I to Angiotensin II
  - Angiotensin II
  - Growth Factor
  - Potent Vasoconstrictor
  - Adrenal Cortex
  - Increases B/P
  - Aldosterone
  - Increases SVR
  - Distal Renal Tubule
  - Increases Na & K+ Reabsorption
  - Excretes K+ for Na+

ACE Inhibitors “Prils”
- Benazepril Lotensin
- Captopril Capoten
- Lisinopril Zestril Prinivil
- Quinapril Accupril
- Ramipril Altace

The Cardiac Cycle

Systole and Diastole

1. Rapid Ventricular Filling

- Pressure in the atria overcomes the pressure in the ventricles; the A-V valves open.
- First third of diastole.
- S3 would be heard here --- right after dub-- sounds like Ken-tucky
2. Active Ventricular Filling

- "Atrial kick" forcing 30% more blood into the ventricles.
- Last third of diastole.
- S4 would be heard here -- sounds like Ten-nes-see - produced by vibration of atria contracting

3. Isometric Contraction

- Pressure in ventricles overcomes pressure in the atria.
- Blood tries to flow back in to the atria
- A-V valves slam shut; S1 is heard --The lub in normal lub dub - heard loudest at Apex
- Semilunar valves have not yet opened; all cardiac valves are closed.

4. Ventricular Systole

- Pressure in ventricles overcomes that in the aorta and pulmonary artery.
- Semilunar valves open.
- Ventrices contract.
- Blood is ejected forcibly into aorta and pulmonary artery.

5. Isometric Relaxation

- At the end of systole, ventricles begin to relax.
- Pressure in aorta and pulmonary artery increases; pressure in ventricles decreases.
- Blood attempts to rush back in to the ventricles.
- Semilunar valves slam shut; S2 is heard - the dub in normal lub - dub; heard loudest at the base
- A-V valves are not yet open. All cardiac valves are closed again.

Cardiac Cycle

- Occurs every 0.8 seconds (HR=80)
- 0.3 seconds for systole
- 0.5 seconds for diastole

Tachycardia decreases diastole time
**Principles of Muscle Function**

---

**Frank-Starling Law**

*The longer the muscle is stretched in diastole, to a point, the stronger the contraction in the next systole.*

---

**Cardiac Output**

- A measurement of flow of the volume of blood pumped by the heart each minute.
- Cardiac Output = Stroke Volume x Heart Rate
  
  \[
  CO = SV \times HR
  \]

- Normal range 4-8 liters/minute

---

**Cardiac Index (CI)**

- Cardiac output that has been indexed to body surface area (BSA)
- Hemodynamic parameters can vary with the size of an individual. Therefore it is easier to interpret values which have been indexed by body surface area (BSA)
- BSA (m$^2$) = weight (kg) x height (cm) x 0.007184
  
  or

  - BSA can be calculated from height/weight nomogram

---

**Cardiac Index**

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

- Cardiac output divided by body surface area (BSA)

- Normal range = 2.5 – 4 l/min/m$^2$
- Subclinical: 2.2 - 2.7 l/min/m$^2$
- Low perfusion: 1.8 - 2.2 l/min/m$^2$
- Shock < < 1.8 l/min/m$^2$

---

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 $CI = \frac{CO}{BSA}$

Mrs. A’s BSA is 1.36 $m^2$. Her CI is determined to be 3.08 l/min/m$^2$.

Mr. B has a BSA of 2.34 $m^2$, therefore his CI falls below the normal level of 1.79 l/min/m$^2$.

Determinants of Cardiac Output

Cardiac Output = Heart Rate x Stroke Volume

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)
How Cardiac Meds effect Heart Rate

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

Determinants of CO:

Rate/Rhythm

Low
Pacemaker
Atropine
Isuprel
Dopamine

High
Beta blockers
Calcium channel blockers
Other
The Effect of Cardiac Meds on Heart Rate

**Increase HR**
- Atropine
- Dopamine/Intopin
- Epinephrine/Adrenalin
- Norepinephrine/Levophed

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

**Slight Increase HR**
- Phenylephrine/
- Neo-synephrine
- Vasopressin/Pitressin
- Dobutamine/Dobutrex
- Milrinone/Primacor

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 (PAP/PAD)</td>
<td>15 - 30/8 - 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**

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

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

Preload

Myocardial Fiber-Stretch
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

- **Low**
  - Volume
- **High**
  - Diuretics
  - Venous vasodilators

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!

Preload

**Common Reasons - Heart Cannot Fill**

- Persistent Tachycardia
- PEEP/CPAP
  - If patient is on PEEP, every time he takes a breath, it tamponades the heart.
  - The heart cannot fill or eject.
Interpretation of the CVP
- Reflects right-sided heart diastolic function and volume status
- Normal 2 - 6 mm Hg
- Assess with SV/SI
  - > 6 mm Hg usually reflects right ventricular failure if the SV/SI is low
  - < 2 mm Hg usually reflects hypovolemia if SV/SI is low

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

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; pushing...

Straw vs. Garden Hose
Afterload is measured as SVR and PVR
- Systemic Vascular Resistance (SVR) reflects LV afterload
  - Normal Range = 800-1400 dynes/sec/cm-5
- Pulmonary Vascular Resistance (PVR) reflects RV afterload
  - Normal Range = 40-220 dynes/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: 900-1400 dyne.sec.cm5

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

CO and SVR
Most Hypovolemic patients with have a 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: \[ PVR = \frac{MPA - PCW \times 80}{CO} \]

Normal Range: 40 - 220 dyne.sec.cm^5

Factors That Increase Pulmonary Vascular Resistance

- Chemical Stimuli
  - Alveolar hypoxia
  - Acidosis
  - Hypercapnia

- Pharmacologic Agents
  - Epinephrine
  - Norepinephrine
  - Dobutamine

- 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
    - Thrombus, endarteritis, polyarteritis, sclerosis

- Vascular destruction
  - Amyloidosis

- Pulmonary interstitial fibrosis

- Vascular Compressions
  - Pneumothorax, hemothorax

- Humoral Substances (Pathologic Factors)
  - Histamine, angiotensin, fibrinopeptides
  - Prostaglandin \( F_2\alpha \)
  - Serotonin

Factors That Decrease Pulmonary Vascular Resistance

- Pharmacologic Agents
  - Oxygen
  - Isoproterenol
  - Aminophylline
  - Calcium channel blocking agents
  - Nitrous Oxide

- Humoral Substances
  - Acetylcholine
  - Bradykinin
  - Prostaglandin E
  - Prostacyclin
  - Sildenafil (Viagra)

Afterload

- Decreased
  - Vasodilation
    - Vasodilation from rewarming
    - Vasodilator therapies
    - Preop beta blockers
    - Sepsis

- Increased
  - Right
    - Pulmonary hypertension
    - Hypoxemia
    - Pulmonic stenosis
  - Left
    - Severe LV dysfunction
    - Vasoconstriction
    - Vasopressors
    - Hyperthermia
    - ↑ catecholamine simulation from surgery

How Cardiac Meds effect Afterload

- Increase Afterload
  - Dopamine/Intopin
  - Epinephrine/Adrenalin
  - Norepinephrine/Levophed
  - Phenytoin/Neo-synephrine
  - Vasopressin/Pitressin

- Minimal effect on afterload
  - Dobutamine/Dobutrex

- Decrease Afterload
  - Nitroprusside/Nipride
  - Arenal vasodilator
  - Nitroglycerin/Tridil
  - Venous vasodilator
  - Beta Blockers
  - Nicardipine/Cardene
  - ACE Inhibitors

- Slight Decrease Afterload
  - Milrinone/Primacor

The Effect of Cardiac Meds on Afterload
Afterload

Low
- Vasopressors
- Warming blanket
- Vasodilators
- Calcium channel blockers
- IABP

High

Contractility

Cardiac Squeeze

- Inotropic state of muscle
- Force & velocity of ventricular contractions
- Not directly measurable
- Independent of Starling mechanism

Increased Contractility

- Sympathetic stimulation
- Metabolic states:
  - Hypercalcemia
- Inotropic therapies:
  - Epinephrine
  - Dopamine
  - Digoxin
  - Calcium
  - Dobutamine
  - Milirone

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

<table>
<thead>
<tr>
<th>Increase Contractility</th>
<th>Decrease Contractility</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium</td>
<td>Beta Blockers</td>
</tr>
<tr>
<td>Dopamine/Intopin</td>
<td>Calcium Channel Blockers</td>
</tr>
<tr>
<td>Epinephrine/Adrenalin</td>
<td>Nicardipine/Cardene</td>
</tr>
<tr>
<td>Norepinephrine/Levophed</td>
<td>Lidocaine/Xylocaine</td>
</tr>
<tr>
<td>Phenylephrine/Neo-synephrine</td>
<td>Vasopressin/Pitressin</td>
</tr>
<tr>
<td>Vasopressin/Pitressin</td>
<td></td>
</tr>
<tr>
<td>Dobutamine/Dobutrex</td>
<td></td>
</tr>
<tr>
<td>Milrinone/Primacor</td>
<td></td>
</tr>
</tbody>
</table>

Treating Low Contractility

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

<table>
<thead>
<tr>
<th>LOW</th>
<th>CARDIAC OUTPUT Treatment Options</th>
<th>HIGH</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PRELOAD</td>
<td></td>
</tr>
<tr>
<td>Volume</td>
<td>CVP, PAO, PAP</td>
<td>Diuretics, Venous Vasodilation</td>
</tr>
<tr>
<td>Vasopressors</td>
<td>SVR_PVR</td>
<td>Vasodilators, Calcium Channel Blockers, IABP, Valve Surgery</td>
</tr>
<tr>
<td>Optimize preload</td>
<td>Inotropes, Calcium, Ventricular Assist Devices, Avoid/treat acidosis</td>
<td>CONTRACTILITY CO/CI indirect measurement</td>
</tr>
<tr>
<td>Pacer, Atropine, Dopamine</td>
<td>RATE/RHYTHM</td>
<td>Beta Blockers, Calcium Channel Blockers</td>
</tr>
</tbody>
</table>

Principles of SvO₂ Monitoring
SvO₂

- An “early warning” system
- Evaluate efficacy of therapeutic interventions
- Identify detrimental consequences of “patient care”

Does the Oxygen Supply meet the Oxygen Demand?

SvO₂ reflects the delicate balance between oxygen delivery and oxygen consumption

Fink Equation Rewritten

\[ \text{SvO}_2 = \frac{\text{SaO}_2 - \text{VO}_2}{\text{CO} \times \text{Hgb} \times 1.34 \times 10} \%
\]

Normal Value is \textbf{60 – 80\%}

SvO₂ Values

<table>
<thead>
<tr>
<th>Saturation %</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>80 or &gt;</td>
<td>Septic, L R Shunt</td>
</tr>
<tr>
<td></td>
<td>Excess inotrope</td>
</tr>
<tr>
<td></td>
<td>Hypothermia</td>
</tr>
<tr>
<td></td>
<td>Cell poisoning</td>
</tr>
<tr>
<td></td>
<td>Wedged catheter</td>
</tr>
<tr>
<td>60 – 80</td>
<td>Normal Range</td>
</tr>
<tr>
<td>60</td>
<td>Cardiac Decompensation</td>
</tr>
<tr>
<td>53</td>
<td>Lactic Acidosis</td>
</tr>
<tr>
<td>32</td>
<td>Unconsciousness</td>
</tr>
</tbody>
</table>

- A single isolated value out of context of other clinical information is probably not very helpful
If oxygen demand exceeds oxygen delivery

- If oxygen demand (the amount of oxygen required by the tissues to function aerobically) exceeds oxygen delivery, shock is present.
- The body must switch to anaerobic metabolism to continue producing energy albeit by an energy inefficient process (glycolysis) with lactic acid or "lactate" as a byproduct.

Four determinants of SvO₂:
- Hemoglobin
- Cardiac output
- Arterial saturation (SaO₂)
- Oxygen consumption (VO₂)

Compensatory Mechanisms to meet O₂ Demands

- **Primary**
  - ↑ Cardiac Output

- **Secondary**
  - ↓ SvO₂

- **Final**
  - Anaerobic Metabolism

SvO₂ < 60

- Increase in O₂ Demand
  - ↑ O₂ Consumption
  - Hyperthermia
  - Seizures
  - Pain
  - Shivering

- Decrease in O₂ Delivery
  - ↓ Hemoglobin
  - Bleeding, anemia
  - ↓ Oxygen Saturation (SaO₂)
  - Hypoxia
  - Suctioning
  - Pulmonary infiltrates
  - Decreased ventilation
  - ↓ Cardiac Output
  - Hypovolemia
  - Hypotension
  - Arrhythmias
  - Cardiogenic shock

SvO₂ > 80

- Decrease in O₂ Demand
  - ↓ O₂ Consumption
  - Hypothermia
  - Anesthesia
  - Pharmacological Paralysis
  - Sepsis (peripheral shunting)

- Increase in O₂ Delivery
  - ↑ Fi O₂
If SVO₂ < 60 for 15 minutes or longer, the tissues are not getting enough oxygen — call for further orders

**SVO₂ Algorithm**

- **< 60**
  - SVO₂ < 60 or changes by 10% in 10 minutes or longer
  - Check O₂ supply
  - Check O₂ consumption
  - Assess for low CO/CI
  - Assess for low HCT
  - Draw serum lactate level
  - Recalibrate machine

- **> 80**
  - SVO₂ > 80
  - May be associated with CO/CI pathologic conditions, lab & x-ray
  - Increase O₂ concentration

---

**SVO₂ < 60 or changes by 10% for 10 minutes or longer**

- Check O₂ supply
- Check O₂ consumption
- Assess for low CO/CI
- Assess for low HCT
- Draw serum lactate level
- Recalibrate machine

---

**Hemodynamics**

Let’s Practice!
Pearls

- Make sure adequate preload before starting inotrope
- Low preload → 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 → potent vasoconstrictors
  - Decrease blood flow to microcirculation
  - ↑MvO₂

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


Why is the Heart Not Responding?

- Blood Pressure
- Cardiac Output
- SVR

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>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PAP</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SV/SVI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SVR/PVR</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
</tbody>
</table>

Hypovolemia
Fluid Overload
LV failure
RV failure
RV & LV failure
Sepsis
CO/CI
↓
N or ↑
↓
↓
↓
↓
CVP
↓
↓
Normal
↓
↓
↓
↓
Pao/PAD
↓
↓
↑
Normal
↓
↓
↓
↓
SVR/SVRI
Normal/increased
↓
↓
Normal
↓
↓
↓
↓
PVR/PVRI
Normal
Normal
Normal
↓
↓
↓
↓
What’s abnormal?

CABG on admission
Dopamine 2.5 mcg/kg/min
30 minutes later after 250 ml 5% albumin

CO/CI
3.7/1.8
SBP/DBP
115/53
MAP
71
HR
85
SvO2
38
CVP
9
PAS/PAD
26/16
PAM
21
PAW
20
SV
44
SVR
1339
PVR
22
PVRI
45

How do you want to treat?
1. Fluid
2. Increase dopamine
3. Decrease dopamine
4. Add another pressor

CABG on admission
Dopamine 2.5 mcg/kg/min
30 minutes later after 250 ml 5% albumin
36 hours later after 500 ml 5% albumin & Dopamine 1 mcg/kg/min

CO/CI
3.7/1.8
SBP/DBP
115/53
MAP
71
HR
85
SvO2
38
CVP
9
PAS/PAD
26/16
PAM
21
PAW
20
SV
44
SVR
1339
PVR
22
PVRI
45

What’s abnormal?
Case 1: Identify abnormal hemodynamic parameters and what you would do?

<table>
<thead>
<tr>
<th>2300</th>
</tr>
</thead>
<tbody>
<tr>
<td>Art BP</td>
</tr>
<tr>
<td>MAP</td>
</tr>
<tr>
<td>HR</td>
</tr>
<tr>
<td>PAS/PAD</td>
</tr>
<tr>
<td>CVP</td>
</tr>
<tr>
<td>SVO₂</td>
</tr>
<tr>
<td>CO</td>
</tr>
<tr>
<td>CI</td>
</tr>
<tr>
<td>SVR</td>
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<tr>
<td>SV</td>
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1. Treat hypovolemia
2. Treat tamponade
3. Treat cardiogenic shock
4. Treat fluid overload

Case 1 Answer: Tamponade. If cardiogenic shock would expect a higher SVR and CVP would be lower. Treatment—reexploration of chest.

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

Case 2 Answer: Tamponade. If cardiogenic shock would expect a higher SVR and CVP would be lower. Treatment—reexploration of chest. Note same patient as before only 11 hours later & did not go for reexploration and was treated with inotropes:
Depammine 2.5 mcg/kg/min, Epi 3.07 mcg/min Milrinone 0.5 mcg/kg/min. Did it help?

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Case 3: Identify abnormal hemodynamic parameters and what you would do?
Patient is on Dopamine 2.5 mcg/kg/min, Epi 3.07 mcg/min Milrinone 0.5 mcg/kg/min

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1. Give fluid
2. Wean Epinephrine
3. Wean Milrinone
4. Observe

Case 3 Answer: This is the same patient post removal of tamponade. SV is borderline low – may need some fluid even with high CVP. Wean intropes as HR ↑

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Case 4: Identify abnormal hemodynamic parameters and what you would do?

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1. Treat hypovolemia
2. Treat tamponade
3. Treat cardiogenic shock
4. Treat fluid overload
Case 4: Identify abnormal hemodynamic parameters and what you would do?

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1. Treat hypovolemia
2. ANSWER

Case 4 Answer:
Hypovolemia. Give fluids – 250 ml 5% Albumin Be careful when warming patient.

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Case 5: Identify abnormal hemodynamic parameters and what you would do?

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1. Fluids
2. Inotrope
3. Antihypertensive
4. Observe

Case 5 Answer:
Warm to decrease SVR. Fluids (check Hbg) for low SV, CI

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Case 6: Identify abnormal hemodynamic parameters and what you would do?

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Case 6: Identify abnormal hemodynamic parameters and what you would do?

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

Case 6 Answer:

Hypovolemia from bleeding. Give blood, check coags

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Case 7: After two units of pRBCs. Did it help? What would you?

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Case 7 Answer: Still hypovolemic – needs more blood/surgery to find bleeder.

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Case 7: After two units of pRBCs. Did it help? Are you happy? 1. Yes 2. No

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Case 7 Answer: Still hypovolemic – needs more blood/surgery to find bleeder.

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<td>PAS/PAD</td>
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<td>95</td>
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<td>SV</td>
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<td>UO</td>
<td>75</td>
</tr>
<tr>
<td>CT</td>
<td>300</td>
</tr>
</tbody>
</table>

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
Hemodynamic Case Studies

Mrs. C is a 72 y/o with Type 1 Diabetes and CAD. She had a CABG x 4. She had difficulty coming off cardiopulmonary bypass. An IABP was inserted in OR & is pumping at 1:1. Dopamine is at 5 mcg/kg/min and Epinephrine at .05mcg/kg/min. What treatments need to be ordered for each case scenario?

**Case 1**

- **B/P**: 82/52
- **HR**: 110
- **RR**: 12/12
- **Temp**: 35.6°C/96°F
- **PAS/PAD**: 25/10
- **CVP**: 4
- **SVR**: 800
- **CO**: 2.5
- **CI**: 1.5
- **SaO2**: 97
- **SvO2**: 56

1. Fluids
2. Inotropes
3. Nitrates to decrease afterload

**Case 2**

- **B/P**: 82/52
- **HR**: 90
- **RR**: 12/12
- **Temp**: 35.6°C/96°F
- **PAS/PAD**: 35/15
- **CVP**: 15
- **SVR**: 800
- **CO**: 2.5
- **CI**: 1.5
- **SaO2**: 97
- **SvO2**: 56

1. Fluids
2. Inotropes
3. Nitrates to decrease afterload

**Case 3**

- **B/P**: 82/52
- **HR**: 132
- **RR**: 12/12
- **Temp**: 35.6°C/96°F
- **PAS/PAD**: 35/15
- **CVP**: 15
- **SVR**: 3000
- **CO**: 2.5
- **CI**: 1.5
- **SaO2**: 97
- **SvO2**: 56

1. Fluids
2. Inotropes
3. Nitrates to decrease afterload

**LOW**

**CARDIAC OUTPUT**

- Treatment Options

**HIGH**

<table>
<thead>
<tr>
<th>LOW</th>
<th>CARDIAC OUTPUT</th>
<th>HIGH</th>
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<tbody>
<tr>
<td>Volume</td>
<td><strong>PRELOAD</strong></td>
<td>Diuretics, Venous Vasodilation</td>
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<tr>
<td></td>
<td>CVP, PAD, PAOP</td>
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<tr>
<td>Vasopressors</td>
<td><strong>AFTERLOAD</strong></td>
<td>Vasodilators, Calcium Channel Blockers, IABP, Valve Surgery</td>
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<td></td>
<td>SVR, PVR</td>
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<tr>
<td>Optimize preload</td>
<td><strong>CONTRACTILITY</strong></td>
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<tr>
<td>Inotropes</td>
<td>CO/CI indirect measurement</td>
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<tr>
<td>Calcium</td>
<td>Ventricular Assist Devices</td>
<td></td>
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<tr>
<td>Avoid/treat acidosis</td>
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<tr>
<td>Pacer, Atropine</td>
<td><strong>RATE/RHYTHM</strong></td>
<td>Beta Blockers, Calcium Channel Blockers</td>
</tr>
<tr>
<td>Dopamine</td>
<td></td>
<td></td>
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</tbody>
</table>
**Post-Operative Nursing Cares Goals**

- Hemodynamic stability
- Adequate perfusion of vital organs
- Prevention of complications

**Trauma Triad of Death**

**Cardiac Surgery Triad of Disaster**

**Hypoperfusion Starts the Triad of Disaster**

**Causes of Heat Loss**

- Cooled during CPB
- Cold OR room
- Cool room and/or fan on
- Cold fluids
  - 1 unit of pRBC can lower body Temp 0.25°C
  - 1 liter of fluids unit can lower body Temp 0.5°C
- No blankets
- Head uncovered
Alarming Consequences of Hypothermia

1. Increased oxygen debt
   - Cold hemoglobin can not release oxygen to the cells
   - Left shift of the oxyhemoglobin dissociation curve
2. Increased lactic acid production
   - Change from aerobic to anaerobic metabolism
   - Leads to acidosis
3. Coagulopathy
   - Prolonged clotting cascade
   - Platelet dysfunction – platelets are extremely temperature dependent
   - Altered fibrinolytic system
4. Altered cardiovascular function
   - Decreased cardiac output
   - Risk of arrhythmias
   - Increased SVR due to vasoconstriction
5. Hyperglycemia
   - Decrease insulin production

Rewarming techniques

- Warm room – no fan
- Warm blankets – keep patient covered
- Bare Hugger
- Use blood warmer to give blood products
  - Have blood warmer and bare hugger in room

Cardiac Surgery Triad of Disaster

Coagulopathy

↑ LOS
Complications
Death

Metabolic Acidosis

Decreased myocardial performance

Alarming Consequences of Acidosis

- Decreased cardiac contractility & cardiac output
- Impaired response to catecholamine
  - (e intropes are not effective)
- Increased PVR
- Vasodilation - decreased SVR
- Bradycardia
- Increased arrhythmia risk
- Coagulopathy
- Compensatory hyperventilation.

Pathophysiology of Acidosis

Hypothermia causes peripheral vasoconstriction and impairs oxygen delivery to the tissues

Hypovolemia caused by bleeding reduces tissue perfusion

cellular metabolism changes from aerobic to anaerobic

serum lactate levels rise

metabolic acidosis develops

Identify Cause of Lactic Acidosis

- Type A Lactic Acidosis
- Type B Lactic Acidosis
Metabolic Acidosis

- Type B Lactic Acidosis
  - Occurs in the absence of tissue hypoxia
  - May be catecholamine-induced metabolic effect (especially with epinephrine)
  - May be caused by hyperglycemia & alterations in fatty acid metabolism

Injury from surgery disrupts normal regulatory mechanisms

Diabetics

Goal is to maintain normoglycemia

Non Diabetics

Elevation in glucose is temporary

- Plasma glucose elevates during surgery
- Insulin secretion is depressed during surgery

Elevated blood levels of catabolic hormones render patient resistant to insulin.
Metabolic changes are proportional to the degree of surgical stress.

Ketones form as a result of incomplete oxidation.

¬ Large quantities of body fluid are needed for renal excretion of glucose and ketones.
¬ This may result in hypovolemia.

The inability of kidneys to remove excess glucose with hypovolemia prompts further rise in glucose.

Metabolic Acidosis may develop within hours.

K+ increases as response to hyperglycemia.
K+ is redistributed from within the cell to the extracellular space as a result of increased plasma osmolality. Insulin corrects the abnormality.

If Potassium is LOW, Be Careful when giving...

♥ Insulin
♥ Calcium
♥ Digoxin

Serum glucose should not decrease more than 75 - 100 ml/dl per hour to prevent...

♥ Hypoglycemia
♥ Hypokalemia
♥ Cerebral Edema
Severe hypoglycemia can lead to coma and death!

Admission ABGs
What would you do?

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<thead>
<tr>
<th></th>
<th>Patient A</th>
<th>Patient B</th>
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</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.29</td>
<td>7.33</td>
</tr>
<tr>
<td>pCO₂</td>
<td>60</td>
<td>41</td>
</tr>
<tr>
<td>pO₂</td>
<td>132</td>
<td>100</td>
</tr>
<tr>
<td>TCO₂</td>
<td>31</td>
<td>22</td>
</tr>
<tr>
<td>O₂ %</td>
<td>98</td>
<td>98</td>
</tr>
<tr>
<td>BE</td>
<td>-1</td>
<td>-6</td>
</tr>
</tbody>
</table>

Metabolic Acidosis
Ongoing Metabolic Acidosis means something is not being perfused

- Type A Lactic Acidosis
  - Reflects impaired tissue oxygenation & anaerobic metabolism resulting from circulatory failure
  - The lactate ion more than the acidemia contributes to potential cardiovascular dysfunction

The Value of Lactate

- Serum lactate levels are used to assess the acid-base state and adequacy of tissue perfusion
- By product of anaerobic metabolism if tissue hypoxia (from hypoperfusion) exists
- A change from aerobic to anaerobic metabolism

- Lactate is primarily excreted by liver.
- Treatment: may use NaHCO₃, but it is truly only a “Band-Aid”. Must treat cause.

The Value of Lactate
Serial lactate levels predictor of perfusion

- Normal <2.5mmol/L
- Mild acidosis 2.5-4.9mmol/L (mortality 25-35%)
- Moderate acidosis 5.0-9.9mmol/L (mortality 60-75%)
- Severe acidosis > 10mmol/L (mortality > 95%)

Lactate Levels
“Surviving Sepsis 2014”

- **Normal 1-2**
  - Cells are alive & well
- **Moderate 2 – 4**
  - DECREASED cellular perfusion
  - Cells STRUGGLE to survive
  - May indicate severe sepsis
- **Severe > 4**
  - COMPLETE TISSUE HYPOXIA
  - Cells DIE
  - Hypotension refractory to adequate fluid resuscitation indicates septic shock

Serum Lactic Acid Levels

- May be the first indication that something is wrong
- Excess lactate demonstrates measurement of tissue oxygen debt
- Results in metabolic acidosis due to tissue hypoperfusion and “starvation”

Serum Lactic Acid Levels

- Increasing lactic acid levels mean the tissues are hypoperfused and patient is getting worse.
- Decreasing lactic acid levels mean the tissues are getting perfused and the patient is getting better.

Base deficit/excess

- Amount of total base (buffer) that is needed to achieve acid-base balance.
- BD/BE is depicted by HCo3-Cl, phosphates, sulfates, proteins and organic acids. To figure BD/BE, lab uses the PH, PaCO2 and Hct.
- **Normal -2 to +2**
- **If < - 2, the patient is not perfusing**

Base deficit

- **Normal -2 to +2**
- **Mild Hypoperfusion: -2 to -5**
- **Moderate Hypoperfusion: -6 to -14**
- **Severe Hypoperfusion: < -15**

Source: Davis J et al: Journal of Trauma, 1996. Davis K et al. Journal of Trauma, 2002

Which patient are you more concerned about?

<table>
<thead>
<tr>
<th></th>
<th>1.0600</th>
<th>2.0800</th>
</tr>
</thead>
<tbody>
<tr>
<td>PH</td>
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<td>7.35</td>
</tr>
<tr>
<td>PCO2</td>
<td>36</td>
<td>32</td>
</tr>
<tr>
<td>PO2</td>
<td>77</td>
<td>87</td>
</tr>
<tr>
<td>CO2</td>
<td>22</td>
<td>18</td>
</tr>
<tr>
<td>O2%</td>
<td>94</td>
<td>96</td>
</tr>
<tr>
<td>BE</td>
<td>-4</td>
<td>-8</td>
</tr>
</tbody>
</table>
Which patient are you more concerned about?

Answer 2

- BP is 80
- pH = 7.29 (metabolic acidosis)

You only have one line and you need to start a Bicarb drip and Dopamine drip. Which will you start first?
1. Bicarbonate drip
2. Dopamine drip

ANSWER

1. Bicarbonate drip

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

Cardiac Surgery Triad of Disaster

Coagulopathy

- Hypothermia
- Acidosis
- Underlying diseases
- Medications
- Dilation with fluids
High Risk Bleeding

- ASA, Plavix, Coumadin or other anticoagulants preop
- History of Liver Disease
- Alcohol use
- Abnormal clotting factors
- Herbals

Risk Factors for Bleeding:
- ASA, Plavix, Lowmox, Coumadin, Integrin, Reopro, Aggragace, TPA, ReoCare, or other anticoagulants preop
- Herbals that affect coagulation (Ginger Root, Grapes, Codenase, Ginseng, Garlic, Bilberry)
- History of liver disease
- Alcohol use
- Abnormal clotting factors
- If known heparin-antibody, do not give heparin

Notify MD if herbals have not been discontinued at least 5 days prior to OR
Notify OR & CVICU that patient at High risk for bleeding
Does surgery need to be delayed?
Do more blood products need to be ordered to be on hold?

High Risk Bleeding

High Risk Bleeding

- Notify MD if herbals not discontinued at least 5 days prior to OR
- Notify OR & CVICU that patient at High Risk for bleeding
- Does surgery need to be delayed?
- Do more blood products need to be ordered to be on hold?

High Risk Bleeding

Change in Plan of Care- preop

- OR and CVICU MUST KNOW!
- Should surgery be delayed???
- Reopro – to reverse need to give 6 units of platelets
- Integrilin – affects platelets until half life wears off
- Plavix – should wait 5 days to have surgery

Preop
- Coag profile

Postop
- Consider daily PT/PPT
- Minimum Coag profile on POD #3
- Daily platele count if on Heparin
- Call for platelet count < 100,000
- Keep at least 2 units of PRBC’s on hold DOS

Coag profile (PT/PTT/Platelet count/Fibrinogen): Pre-op
- Platelet transfusions if patient received radiation

Aggraglace infusion
- Consider daily PT/PTT post-op
- Minimum Coag profile on POD #3 if high risk bleeding preop
- Daily platelet count if on Heparin
- Call for platelet count < 100,000
- Keep at least 2 units of PRBC’s on hold DOS

BLEEDING

BLOWN GRAFT
### Causes

<table>
<thead>
<tr>
<th>Mechanical Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Bleeding from suture lines</td>
</tr>
<tr>
<td>• Clip comes off graft</td>
</tr>
<tr>
<td>• Aortic or ventricular rupture</td>
</tr>
<tr>
<td>• Chest wall bleeders</td>
</tr>
<tr>
<td>Abnormal clotting factors due to</td>
</tr>
<tr>
<td>• Preop anticoagulant meds</td>
</tr>
<tr>
<td>• Systemic heparinization during CPB</td>
</tr>
<tr>
<td>• Breakdown of factors during CPB</td>
</tr>
</tbody>
</table>

### Signs & Symptoms

<table>
<thead>
<tr>
<th>CT bleeding &gt; 100 – 200 cc/hr</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low or labile B/P</td>
</tr>
<tr>
<td>Low CVP or PAD</td>
</tr>
<tr>
<td>Falling SvO(_2) and CO/CI</td>
</tr>
<tr>
<td>Abnormal clotting Factors</td>
</tr>
<tr>
<td>Bleeding from line sites, incisions</td>
</tr>
</tbody>
</table>

### Treatments

| Monitor CT output. May need to replace CT output cc for cc with packed cells |
| Keep sedated and keep B/P < 140 to prevent stress on suture lines           |
| Keep CT patent by gently milking and stripping                                |
| Use warming blanket to keep normal thermic.                                  |
| Hypothermia interferes with clotting factors                                |

### Treatment: Blood and Blood Products

| Give blood and blood products                                   |
| FFP for ↑ PT or PTT                                            |
| Platelet Phoresis for ↓ Platelet count                         |
| Cryoprecipitate for ↓ Fibrinogen level                         |
| Packed cells for ↓ H & H                                       |

### Rule of thumb

| Replace CT output ml for ml                                   |
| After every 4\(^{th}\) unit pRBCs                           |
| • Calcium Chloride                                          |
| • FFP                                                      |
Keep blood on HOLD --- communicate with blood bank that you have a bleeder

- May need to use type specific blood

Treatments

- Pharmacological Interventions
  - Protamine to reverse effects of systemic heparinization
  - Aminocaproic Acid (Amicar) to inhibit conversion of plasminogen to plasmin
  - Desmopressin to improve platelet function
- May need to return to surgery to repair mechanical cause of bleeding

81 y/o male CABG x 3 LAD, Marginal, PDA

Admission vital signs & labs.

What are your priorities?

Treat high glucose
Treat low Magnesium
Fluids for low CI

Postop recovery going well. Extutated at 0015.

Now what?

1. ROUTINE SURGERY – continue to monitor
2. BLEEDING – call surgeon
3. HYPERTENSIVE – start antihypertensive
It's 0500. What are your priorities?

- It is 0500, calculate the CT output and the blood products given…. Are you keeping up with the bleeding?

<table>
<thead>
<tr>
<th>Art B/P</th>
<th>MAP</th>
<th>HR</th>
<th>Temp</th>
<th>PAS/PAD</th>
<th>CO</th>
<th>CI</th>
<th>SVR</th>
<th>SpO2</th>
<th>CT</th>
<th>Fluid</th>
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<td>151/51</td>
<td>91</td>
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<td>97</td>
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<tr>
<td>113/43</td>
<td>66</td>
<td>101</td>
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<td>67/2</td>
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<td>5.8</td>
<td>758</td>
<td>93</td>
<td>300</td>
<td>190 cc</td>
</tr>
</tbody>
</table>

**Hypotension, 200 cc PRBC, 250 cc albumin, 325 cc PRBC, 200 cc platelets**

**Hypotension, 200 cc PRBC, 250 cc albumin, 325 cc PRBC, 200 cc platelets**

**Hypotension, 200 cc PRBC, 250 cc albumin, 325 cc PRBC, 200 cc platelets**

- 2nd Case: Based on these coag values, what blood products do you expect to be ordered?

- Bleeding patient summary of vital signs

<table>
<thead>
<tr>
<th></th>
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<td>50</td>
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<td>HR</td>
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<td>116/47</td>
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<td>SpO2</td>
<td>98</td>
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<td>98</td>
<td>98</td>
<td>98</td>
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<td>98</td>
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<tr>
<td>CT</td>
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<td>Fluid</td>
<td>200 cc PRBC</td>
<td>250 cc albumin</td>
<td>325 cc PRBC</td>
<td>200 cc platelets</td>
<td>325 cc PRBC</td>
<td>200 cc platelets</td>
<td>325 cc PRBC</td>
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</table>

**Hypotension, 200 cc PRBC, 250 cc albumin, 325 cc PRBC, 200 cc platelets**

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**Hypotension, 200 cc PRBC, 250 cc albumin, 325 cc PRBC, 200 cc platelets**

- 2nd Case: Based on these coag values, what blood products do you expect to be ordered?

Hgb 7.3 1. ANSWER

HCT 21.3 2.

Platelets 186 3. Packed cells, FFP,

PT 21.7 Cryoprecipitate

INR 2.23 4. Packed cells, FFP,

PTT 43.4 Cryoprecipitate, platelets

Fibrinogen 108

Lactic Acid 3.1

- Pt received 3 units of pRBCs
After 5 pRBCs, 1 platelet pheresis, 3 FFP, 10 cryo. Would you anticipate any further blood products?

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
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<th></th>
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<tbody>
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<td>Hbg</td>
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<td>28.1</td>
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<td>115</td>
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<td>18.7</td>
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<td>1.09</td>
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<tr>
<td>Lactic Acid</td>
<td>3.1</td>
<td>2.8</td>
</tr>
</tbody>
</table>

Cardiac Tamponade

- Compression of the heart due to collection of fluid or blood in the pericardial space

Cardiac Tamponade: Causes

- Blood accumulated in the chest from:
  - CTs clotted off and unable to drain excess blood
  - Epicardial wire removal
- May occur quickly within minutes of hours or may occur slowly over days or weeks

Cardiac Tamponade: Signs & Symptoms

- Hypotension
- Low urine output
- Rising & equalization of CVP & PAD
- Falling SVO₂, CO/CI
- Sudden decrease in CT output
- Widening mediastinum on CXR
- Neck Vein Distention
- Tachycardia
- Pulses Paradox > 20 mmHG
- Diminished heart sounds
- For tamponade that occurs slowly may also see these S/S:
  - Shortness of Breath
  - Chest Pain
  - Ischemic changes on EKG
  - Nausea

Cardiac Tamponade

- Beck’s Triad
  - Hypotension
  - Neck vein distention
  - Muffled heart sounds
Cardiac Tamponade: Treatment

- Urgent surgical exploration to evacuate excess blood & correct cause of the tamponade
- Bedside echo may be used to make differential diagnosis between tamponade & LV failure
- Administer fluids & inotropes or Calcium Chloride until patient can be returned to OR
- Prepare for possible exploration of chest at bedside

It's 2300, what do you want to do?

1. Fluids for hypovolemia
2. Surgery for tamponade
3. Inotropes for cardiogenic shock
4. Diuretics for fluid overload

**ANSWER**

It's 2300, what do you want to do?

2. Surgery for tamponade

TAMPOANDE!

Conquering Complications:
Not related to the Triad of Disaster

- Coagulopathy
- Cardiac Surgery
- Triad of Disaster

↑ LOS Complications
- Death

↑ LOS Complications
- Death

Metabolic Acidosis
- Decreased myocardial performance

Post Op Hypertension

Identify And Treat Cause
Post op Hypotension Causes
- Bradycardia/tachycardia
- Irregular rhythm/Arrhythmias
- Medications
- Volume Status
- SVR
- Contractility or Low Ejection Fraction
- SvO$_2$
- Tamponade
- Acidosis
- Hypoxia

Hypovolemia

<table>
<thead>
<tr>
<th>Bleeding pt</th>
<th>2400</th>
<th>700</th>
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<tbody>
<tr>
<td>Art B/P</td>
<td>149/44</td>
<td>91/38</td>
</tr>
<tr>
<td>HR</td>
<td>82</td>
<td>108</td>
</tr>
<tr>
<td>PAS/PA</td>
<td>28/12</td>
<td>20/12</td>
</tr>
<tr>
<td>CVP</td>
<td>12</td>
<td>6</td>
</tr>
<tr>
<td>SvO$_2$</td>
<td>71</td>
<td>59</td>
</tr>
<tr>
<td>CO</td>
<td>4.5</td>
<td>3.6</td>
</tr>
<tr>
<td>CI</td>
<td>2.3</td>
<td>1.8</td>
</tr>
<tr>
<td>SVR</td>
<td>1186</td>
<td>1006</td>
</tr>
<tr>
<td>SV</td>
<td>54</td>
<td>33</td>
</tr>
<tr>
<td>Temp</td>
<td>98.6</td>
<td>97</td>
</tr>
</tbody>
</table>

Volume Status

- Volume Depleted
  - CVP < 10 mm Hg
  - PAD < 15 mm Hg
  - SV < 60 ml/min
    - Patient specific
  - Early sign of hypovolemia = decreased pulse pressure

Volume Status

- HCT < 21-25 (surgeon specific)
  - Yes
    - Give PRBC per orders
  - No
    - 250 ml 5% Albumin per orders
    - Constantly reassess for fluid overload
      - Especially Decreased EF
      - History of Renal Insufficiency

Copious urine output and falling CVP and/or PAD

- Treatment:
  - Hespan or 5% albumin
  - Resist giving too much fluid, it can cause Hemodilution and cause progressive anemia and therefore dilute clotting factors causing mediastinal bleeding.
**Post op Hypotension**
- Bradycardia/tachycardia
- Irregular rhythm/Arrhythmias
- Medications
- Volume Status
- SVR
- Contractility or Low Ejection Fraction
- SvO$_2$
- Tamponade
- Acidosis
- Hypoxia

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**Cardiac Vasoplegic Syndrome post Cardiac Surgery**

Sara Caruso, RN, BSN, CCRN-CSC

---

65 year old male is admitted to CVICU post on-pump CABGx3, on Insulin gtt, Epinephrine drip, and precepx drip.

PMH: ½ ppd smoker x20 years, depression with use of SSRI's, EF = 40%

Admitting VS:
- Mean Arterial Pressure 60
- CVP 3
- CO/CI 4.5/2.5
- SVR 1012

RN administers 250ml of 5% albumin x2 with no change in SVR/CVP, Neosynephrine drip is started

1 hour post-op VS:
- Mean Arterial Pressure 42
- CVP 1
- CO/CI 4.2/2.1
- SVR 770

---

**What is Cardiac Vasoplegic Syndrome?**

- Arterial vasodilatory state resistant to the usual vasopressors post cardiac surgery
- Severe and persistent form of hypotension, tachycardia, increased/normal cardiac output, and decreased systemic vascular resistance (SVR) <800
- Low filling pressures that are poorly responsive or unresponsive to volume
- 8.8% of all patients post cardiac surgery
- Increased morbidity and mortality

---

**Why does this happen?**

- Huge inflammatory response post cardiac surgery
  - Cardiopulmonary bypass
  - Surgical trauma
  - Blood loss
  - Blood transfusions
  - Hypothermia
  - Neutralization of heparin with protamine

---

**At Risk population:**

- Preoperative chronic congestive heart failure
- Previous use of ACE inhibitors and Beta Blockers
- Use of pre and post Amiodarone and Phosphodiesterase inhibitors (Milrinone)
Treatment for Cardiac Vasoplegic Syndrome

Methylene Blue (Tetramethylthionin chloride)
- Interferes with the nitric oxide pathway (causes vasodilation) and inhibits the vasorelaxant effect on smooth muscle
- Can raise mean arterial pressures while minimizing the use of vasopressors
- Usually given pre-op or inter-op to prevent CVS
- Bolus dose of 1-2mg/kg over 10-20 min followed by an infusion of 0.25mg/kg/hr for 48-72 hours, do not exceed 2mg/kg

Vasopressin
- Treats refractory hypotension when used in conjunction with Methylene Blue
- Potent vasoconstrictor

Nursing Role
- Early identification of Cardiac Vasoplegic Syndrome
- Notification to cardiac surgeon
- DO NOT KEEP GIVING VOLUME!

Post op Hypotension
- Bradycardia/tachycardia
- Irregular rhythm/Arrhythmias
- Medications
- Volume Status
- SVR
- Contractility or Low Ejection Fraction
- SvO₂
- Tamponade
- Acidosis
- Hypoxia

Decreased Contractility
- Underlying Causes & treatment
  - Treat hypoxia
  - Treat low ionized Ca++ with Calcium drip
  - Treat acidosis with Bicarbonate
  - depresses myocardium
  - decreases effectiveness of inotropes

- Cause hypotension
- Low cardiac contractility
- Low SVR
- Acidosis
- Hypoxia
- Increased systemic vascular resistance
- Increased heart rate
- Decreased cardiac output
- Low CO/CI
- Low SVR
- Increased heart rate
- Low CO/CI
- Acidosis
- Hypoxia
- Increased systemic vascular resistance
- Increased heart rate
- Decreased cardiac output
Hypertension: Dangers

- Predispose to bleeding
- Suture line disruption
- Aortic Dissection
- Increased myocardial oxygen demand

Hypertension: Treatment

- Keep B/P < 140 - 150 mmHg
- Antihypertensive medication
  - Nitroglycerine
  - Cardene
  - Beta Blockers
  - Nitroprusside
- Analgesics or sedatives for pain or anxiety

Treatment of Cardiac Arrest

Cardiac Surgery Advanced Life Support

Cardiac Arrest...

- In the immediate postop recovery in a cardiac surgery patient is typically related to reversible causes
- Tamponade
- Bleeding
- Ventricular arrhythmias
- Blocks associated with conduction problems
- Survival to discharge can be up to 79%
  - If treated promptly

Pulseless Electrical Activity (PEA)

- Cardiac surgery patients who arrest with PEA are typically experiencing treatable causes
  - Hypovolemia -- severe
  - Hypoxia
  - Tamponade
  - Tension pneumothorax
- Prompt treatment results in good outcomes
  - To assess for causes of PEA/nonschockable rhythm
  - Consider the 4 “Hs” and 4 “Ts”
### Assess for Reversible Causes

<table>
<thead>
<tr>
<th>Four Hs</th>
<th>Four Ts</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoxia *</td>
<td>Tamponade *</td>
</tr>
<tr>
<td>Hypovolemia</td>
<td>Tension Pneumothorax</td>
</tr>
<tr>
<td>Hypokalemia/ Hyperkalemia</td>
<td>Thromboembolism</td>
</tr>
<tr>
<td>Hypothermia</td>
<td>Toxin</td>
</tr>
</tbody>
</table>

* = Most common causes of cardiac surgery arrests

### Treatment of PEA causes

<table>
<thead>
<tr>
<th>Hypoxia</th>
<th>Hypovolemia and Tamponade</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Treat per airway management and assessment</td>
<td>• Severe hypovolemia is typically due to bleeding</td>
</tr>
<tr>
<td></td>
<td>• Severe hypovolemia and tamponade both require emergent resternotomy to correct</td>
</tr>
</tbody>
</table>

### Steps to Ensure Adequate Airway and Ventilation

1. Check endotracheal tube (ET) position and end tidal carbon dioxide (EtCO₂) waveform and reading
2. Listen for an ETT airleak and verify that it is properly inflated
3. Listen and look for bilateral breath sounds.
   - Consider removing the patient from the ventilator and give 100% oxygen via bag-mask-valve to more easily assess lung sounds and determine lung compliance
   - If bilateral lung sounds are present, reconnect the ETT to ventilator.

### Prepare for Emergency Resternotomy

**Six Key Roles**

1. External cardiac massage
2. Management of airway and breathing
3. Defibrillation
4. Team leader
5. Medication administration
6. ICU nursing Coordinator

**You will save more lives by being METICULOUS than by being BRILLIANT**
Brush Up on….. (CSC)
- Other Patient Care Problems
- Extubation/ Respiratory Complications
- ABGS
- IABP
- Post Op Cardiac Surgery Arrhythmias
  - Atrial arrhythmias
  - Maze procedure
  - Antiarrhythmic meds
  - Ventricular arrhythmias
  - Bradyarrhythmia
  - Pacemakers

Next Steps
- Make a study action plan
- Set the target test date
- Get a study partner

CCRN or CSC
- By __________ (insert date)
  Place selfie here

Cardiac Surgery Pearls of Wisdom
Fun and Focused
Class M180M613