Care of the Complex Cardiac Medical & Surgical Patient

Fun and Focused CMC-CSC Review

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The CMC 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. The content of the CMC exam is described in the test plan. The CMC exam focuses on adult patient populations. One hundred percent (100%) of the exam focuses on clinical judgment.

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

CSC 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

Study Help

- Dodge, T. Fast Facts for the Cardiac Surgery Nurse. Springer Publishing
- www.aacn.org
Study Apps to Download

- Pinterest

Disclaimer: Cheryl has not thoroughly tried these to be able to rate them... just passing on tips from others who have used them.

Let's Start!

CMC-CSC Test Blueprint

<table>
<thead>
<tr>
<th>CMC</th>
<th>CSC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute Coronary Syndrome</td>
<td></td>
</tr>
</tbody>
</table>

Correlate the Coronary Arteries

- Inferior – RCA – II, III, AVF
- Septal – LAD – V1, V2
- Anterior – LAD – V3, V4
- Lateral – Circumflex – I, AVL, V5, V6

Cardiac Anatomy

Atherosclerosis
**Cascade effects of atherosclerotic plaque rupture**

- Platelet aggregation
- Fibrin accumulation
- Thrombus formation
- Bleeding into the plaque
- Vasospasm

**Evolutinal Changes of an Acute Myocardial Infarction**

**Time Is Muscle**

**Muscle is Ejection Fraction**

**Ejection Fraction is Quality of Life**

**Target**

**Door to Balloon < 90 minutes**  
(Class 1, Level A)  
*or*  
**Door to Needle < 30 minutes**  
(Class 1, Level B)

ACC/AHA 2013 Guidelines for Management of STEMI

**CRUSADING towards a GOAL**

**Door to EKG**

10 minutes or less  
(Class 1: Level C)

**EKG Changes with MI: Ischemia**

- Ischemia < 20 Minutes  
  Lack of oxygen to the myocardial muscle
  ∗ Peaked T Waves
  ∗ Inverted T Waves
  ∗ ST Segment Depression

Ischemia = screaming for oxygen
EKG Changes with MI: Injury

Injury 20 - 40 minutes
When the period of ischemia is prolonged more than a few minutes, ischemic areas of the heart become damaged (injured)

- ST segment elevation

![Graph showing EKG changes with injury](image)

The ST Segment

- From the end of the QRS complex to the beginning of the T wave
- Should be at the baseline

![Graph showing the ST segment](image)

Measurement of ST-Segment Deviation

- STEMI: ≥1 mm ST-segment elevation in 2 leads.*
- NSTEMI/UA: ≥0.5 mm ST-segment ischemic depression in 2 leads.*
  
  *Anatomically (regionally) contiguous leads.

Source: AHA ACLS EP

EKG Changes with MI: Infarction

Infarction > 1 - 2 hours

- Abnormal Q waves
  - > 1/3 the height of R wave in that lead
  - > 0.03 ms wide

Normal Q wave
- 1st downward deflection of QRS

Abnormal (significant Q waves)

![Graph showing EKG changes with infarction](image)

Normal QRS complex – The Q wave

- Q wave is the first negative deflection after the p wave
- Always first may or may not be there
- Comes first in the alphabet
- There are normal and abnormal Q waves

Normal QRS complex – The R wave

- R wave is the first positive deflection after the p wave
- Always Rising above

![Graph showing normal QRS complexes](image)
Normal QRS complex – The S wave

- S wave is the second negative deflection after the R wave
- Slipping down
- Always after R wave like in the alphabet

EKG Changes with MI: Infarction

Infarction > 1 - 2 hours
- Abnormal Q waves
  - > 1/3 the height of R wave in that lead
  - > 0.03 ms wide

Normal Q wave
1st downward deflection of QRS

Evolution of STEMI

Evolving AMI:
EM #1 December 13 at 1701

EM #2 December 13 at 1823

EM #3 December 14 at 0630
Hyperacute Phase of MI
- Occurs within minutes to first hour of chest pain
- Very tall ST segment
- Tall peaked symmetrical T waves

Acute Phase of MI
- Occurs in the first 24 hours
- ST segment elevation returns to baseline within 24 hours
- T wave inversion occurs in 24 - 48 hours and stays for two weeks
- Q wave develops after 48 hours
- R wave decreases

Evolving Phase of MI
- Occurs in the first week
- ST segment returns to normal
- T wave is deeper and inverted
- Q wave deepens
- R wave decreases more

Resolving Phase of MI
- Occurs in second week
- ST segment returns to normal
- T wave stops inverting and stays that way for 2 weeks and then resolves and will be low voltage
- Q wave stops deepening and stays due to dead tissue
- R wave stops decreasing and stays due to dead tissue

CC: Admission EKG 1445
Positive EKG

- ST elevation > 0.1 mV (1 mm) in at least 2 contiguous precordial leads or at least 2 adjacent limb leads (STEMI) (Class 1, Level A)
- Transient ST Elevation > 0.5 mm
- ST depression > 0.5 mm (NSTEMI)
- T wave inversion > 0.2 mV (2 mm)
- New LBBB (Class 1, Level A)

LBBB = QRS > 0.12 sec
Negative QRS in V1 (carrot)

RBBB = QRS > 0.12 sec
Positive QRS in V1 (rabbit ears)

BBB = QRS > 0.12 sec
- LBBB = QRS > 0.12 sec
  Negative QRS in V1 (carrot)
- RBBB = QRS > 0.12 sec
  Positive QRS in V1 (rabbit ears)
NSTEMI
Non ST Segment Elevation MI

• No ST segment Elevation
• ST segment depression

STEMI
PCI < 90 minutes

Both elevated Tropionins

PCI within 24 - 48 hours

Differentiating MIs

Non-STEMI
• T wave inversion (ischemia)
• ST depression (injury)
• Laboratory values are diagnostic

STEMI
• T wave inversion (ischemia)
• ST elevation (injury)
• Q wave (infarct)
• Laboratory values

Treatment Strategies

STEMI
ST-segment elevation or new LBBB strongly suspicious for injury
Reperfusion
Lytics—PCI

NSTEMI
ST-segment depression/dynamic T-wave inversion; strongly suspicious for ischemia
Antplatelet
Therapy

Angina/Unstable Angina
Normal or nondiagnostic ECG; chest pain strongly suspicious for ischemia
Risk
Stratification

Acute Coronary Syndrome (ACS)

• Umbrella term for a group of thrombotic coronary artery disease conditions that cause myocardial ischemia
• These syndromes represent progression of occlusion in the involved coronary artery
  • STEMI (ST segment Elevation Myocardial Infarction)
  • NSTEMI (Non-ST Segment Elevation Myocardial Infarction)
  • Unstable Angina
Types of Angina

<table>
<thead>
<tr>
<th>Type of Angina</th>
<th>Description</th>
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<tbody>
<tr>
<td>Exertional Angina (4 Es)</td>
<td>Pain with increased myocardial oxygen demand (Exertion, Eating, Extreme Emotions, Exposure to Cold)</td>
</tr>
<tr>
<td>Prinzmetal's Angina or Variant Angina</td>
<td>Pain at rest, during sleep or without evidence of provocation</td>
</tr>
<tr>
<td>Stable Angina</td>
<td>Exertional angina with consistent symptoms—typically relieved with rest or cessation of cause and possibly NTG</td>
</tr>
<tr>
<td>Unstable Angina (crescendo or preinfarction angina)</td>
<td>Partially occluding thrombus, Recent onset (within 2 months), Severely limits activity, May occur at rest, RX with Anti-platelets, Fibrinolytic therapy is not effective</td>
</tr>
</tbody>
</table>

12 Lead EKG
Understanding Lead Placement

To Learn 12 Lead EKG
You MUST pick them up and LOOK at them!

Leads Are Like Pictures
Camera is on the positive lead

The 12 Leads
Bipolar Leads
Each lead has two poles: One positive & one negative
I, II, III

Unipolar Leads
Only one lead is physically positive. Negative lead is not a specific site on the body
AVR, AVL, AVF, V1-V6
Cube Concept of Left Ventricle

Each face of the cube represents a different part of the left ventricle.

LIMB LEADS I, II, III

- Also referred to as extremity leads due to placement on the body
- Record electrical forces two points equidistant from the heart.
- Each lead has two poles: one positive & one negative
- Two leads to give the picture
- Current travels Negative to Positive to create the electrical complex
- 12 Lead EKG Reads or takes the picture from the positive electrode to the heart

Lead I

Views the heart from left arm to right arm
Area: Lateral
Artery: Circumflex

Lead II

Views the heart from left leg to right arm
Area: Inferior
Artery: RCA

Lead III

Views the heart from left leg to left arm
Area: Inferior
Artery: RCA

Einthoven’s Triangle

By connecting the electrodes of the limb leads, the Einthoven’s Triangle is formed.
Augmented Limb Leads
AVR, AVL, AVF

- Records electrical activity between the center of the heart and an extremity
- Since these leads are low voltage they are artificially augmented
- Unipolar leads: Negative pole is the heart

AVR
Augmented Voltage Right
- Views electrical activity directed rightward.
- Very minimal rightward electrical activity occurs.
- Looks at great vessels not myocardium
- Configuration should be negative

AVL
Augmented Voltage Left
- Views the heart from the left arm to the mid-point between right arm & right leg
- Area: Lateral
- Artery: Circumflex

AVF
Augmented VoltageFoot
- Views the heart from the feet to the chin
- Area: Inferior
- Artery: RCA

The Precordial System
Chest Leads V1 – V6

- Records electrical activity of the heart by placing electrodes on the anterior chest wall
- Heart is the negative pole
- Positive pole is where the electrode is placed
- Unipolar leads
**Precordial Leads Placement**

- **V1**: 4th intercostal space (ICS), right sternal border (septum)
- **V2**: 4th ICS, left sternal border (septum)
- **V3**: Midway between V2 and V4 (anterior)
- **V4**: 5th ICS, left midclavicular line (anterior)
- **V5**: 5th ICS, left anterior axillary line (lateral)
- **V6**: 5th ICS, left midaxillary line (lateral)

**R Wave Transition**

**R: Rises above baseline**

- **V1 & V2**: Views the septum of the heart
  - Area: Septal
  - Artery: LAD

- **V3 & V4**: Views the anterior area of the left ventricle
  - Area: Anterior
  - Artery: LAD

- **V5 & V6**: Views the lateral area of the left ventricle
  - Area: Lateral
  - Artery: Circumflex
Cardiac Anatomy in Relation to Coronary Artery

Reciprocal Changes
Reciprocal Leads that are opposite the damaged area will show opposite EKG Changes

- Mirror Image
- Two different electrodes viewing AMI from opposite angles
- Example: Take photo of male from front and from back – still a male but different view

Reciprocal Changes
Secondary Changes

- Ischemia, Injury, and infarction are primary changes
- Reciprocal changes are secondary changes
- Reciprocal Leads that are opposite the damaged area will show opposite EKG Changes
- Reciprocal changes = confirm primary changes
Reciprocal Changes
Reciprocal Leads that are opposite the damaged area will show opposite EKG Changes

- If you see ST segment depression, look in opposite leads for primary changes
- If you see tall R waves in the V leads, question if this is an old posterior AMI and look for Q waves in the inferior leads

Differential Diagnosis
12 Lead EKG in Acute Coronary Syndrome

Right Coronary Artery RCA
Inferior Wall
II, III, AVF

Occluded RCA
RCA post stent

Inferior Injury

Old Inferior Infarction
**Inferior AMI**
- Involves right ventricle – may also get right ventricular infarct
- Need lots of fluids to increase preload since RV is involved
- Arrhythmias= Blocks
- RCA wraps around the back of the heart and changes to PDA. Typically have inferior –posterior AMI.
- Inferior Posterior AMI:
  - ST Elevation: II, III, AVF and
  - ST depression V1, V2, V3

**Left Anterior Descending Artery LAD**
- Anterior Wall V3 & V4
- Septal Wall V1 & V2

**Precordial Leads – Know normal**

**Anterior-septal Injury**

**Old Anterior Infarction**
Occluded High LAD LAD post stent

**Anterior AMI**
- Lose the most muscle mass
- Usually have the lowest EF
- Arrhythmias = VT or VF

Stress Test – Angio found 3 occlusions in the LAD

**Circumflex Artery Cx**
- Lateral Wall
- I, AVL V5 & V6

**High Lateral Injury**

**Anterior Lateral Injury**
Lateral Ischemia

Posterior Descending Artery PDA
Posterior Wall
Reciprocal Changes

- Usually see with Inferior AMI as RCA wraps around the back of the heart and changes to PDA
- Reciprocal Changes V1, V2, V3 (ST segment depression or Tall R Waves)
- 18 Lead EKG

Inferior & Posterior Injury

Mirror Changes

Old Inferior-posterior Infarction

Mirror Changes

Lead Placement: Right-Sided ECG

Right-Sided 12-Lead ECG: Patient With Inferior ST-Segment Changes
18 Lead EKG

Anterior Chest

V4R = 5th ICS at RMCL
V5R = 5th ICS at RAAL
V6R = 5th ICS at RMAL

Posterior Chest

V7 = 5th ICS at Left Posterior axillary line
V8 = Halfway between V7 & V9
V9 = 5th ICS next to vertebral column

Right Chest and Posterior EKG

Pattern to Read EKG

Be consistent

- Rate & Rhythm
- QRS Interval V1 – for RBBB or LBBB
- QT interval
- Normal Depolarization – If not, why not
- ST & T waves
- What lead is abnormal and what other lead goes with it
- Evaluate axis
- Evaluate for hypertrophy

Example & Analysis Time

- Peaked T waves
- T Wave inversion
- ST Depression
- ST Elevation
- Q waves
- Type of AMI
- Coronary Artery Involved
48 y/o male has crushing chest pain
Calls 911

Door to PCI time = 49 minutes
- Initial CK = 72 IU/L, CK MB = 1.0 ng/ml
  Troponin = < 0.4 ng/ml
- 8 hours later CK MB = 2.8 ng/ml,
  Troponin = 0.58 ng/ml
- 12 hours later CK MB = 3.3 ng/ml,
  Troponin = 0.51ng/ml

Case Study: 42 year old male comes to ED (wife drives him)
- Came to ED due to c/o substernal burning pain that radiates up chest to both arms.
- Becomes SOB with Chest pain
- Episodes last approx 10 minutes at a time.
- Episodes occur more when lying flat.
- Episodes have been occurring for last 4 months.
- Had a negative stress test & normal GI workup.
- Denies any drug use of cocaine or other medications
- Quit Smoking 4 months ago. No other past medical history
- Father had some cardiac problems when he was in his 50s or 60s --- history unclear.

- Pain free on arrival to ED
- Alert, Oriented
- Skin Warm/dry
- When laid down for EKG developed chest pain
- BP 122/77, HR 87, RR 20 SpO2 99%
- Chest pain 7/10
- Weight: 70 kg
This 12 Lead was done when he was lying down and complaining of chest pain on Feb 24 at 1333.
- Patient is SOB & in severe pain at the time of the EKG.
- First time EKG done during chest pain

Chest pain resolved when sat up
- BP 118/56, HR 74, RR 20

Serial troponin levels & lipid levels ordered
- Troponin < 0.4 ng/ml
- CK = 71
- Total Cholesterol = 161
- Triglycerides = 66
- HDL = 35
- LDL = 113

Called cardiologist
- 1st EKG STEMI that resolved after a few minutes.
- Admit patient to CVICU. Started on ASA, plavix, heparin drip, nitroglycerin drip, and lopressor
- Hold cardiac cath for now as pain free with normal EKG

Cardiac Cath Feb 25
Initial Injection of RCA
- 70% Occlusion
Cardiac Cath Feb 25
RCA after administration of Intracoronary Nitroglycerin

Management
- Diltiazem 180 mg
- Nitroglycerin 0.4 mg Transdermal patch. Apply at bedtime and remove at 10 am.
- Two days later, stated, “I am finally sleeping at night!”
- Discharged with
  - Diltiazem 180 mg daily
  - Nitroglycerin 0.4 mg Transdermal patch at HS

Time is Muscle
59 minutes Door to PCI Time!
Benchmark < 90 minutes

Treatment Strategies

Cascade effects of atherosclerotic plaque rupture
- Platelet aggregation
- Fibrin accumulation
- Thrombus formation
- Bleeding into the plaque
- Vasospasm

ARTERY OCCULUSION
Clotting Cascade is like a freeway with multiple entrances.

Complementary Mode of Action between Clopidogrel and ASA

Antiplatelets

P2Y12 (ADP) platelet inhibitor
- Clopidogrel (Plavix®)
- Prasugrel (Effient®)
- Ticagrelor (Brilinta®)

Inhibition of platelet COX-1
- ASA

IIB/IIIa Receptor Inhibitors
- Abciximab (Reopro)
- Eptifibatide (Integrilin)
- Tirofiban (Aggrastat)

Oral Anti-coagulants

Direct thrombin inhibitor
- Dabigatran (Pradaxa)

Factor Xa inhibitor
- Rivaroxaban (Xarelto)
- Apixaban (Eliquis)

Vitamin K Antagonist
- Warfarin (Coumadin)
### What's the common complication of all the anticoagulants?

Bleeding!

### Other Causes of STEMI
- Coronary vessel spasm
- Coronary emboli
- Vasculitis
- Severe chest trauma

### Heart Attack Signs & Symptoms for Males
- Chest Pain
- Pain radiating down arms
- Jaw Pain
- Sweating
- Nausea

### Heart Attack Signs & Symptoms for Women
- “Atypical” Chest Pain
- Shortness of Breath/ Trouble Breathing
- Tingling of Fingers
- Extreme Fatigue
- Heartburn / Nausea
- Sweating
- Dizziness
- Feeling of Apprehension or Impending Doom

Even if they recognize the symptoms, women hesitate to wall 911 and get to the hospital 40 to 60 minutes later than men.

### Time is Muscle
Treatment as getting ready for PCI

- **Oxygen** (Class 1, Level B)
- **ASA 162 mg if not given in ambulance** (Class I, Level C)
- **Betablocker**: Metoprolol 5 mg IV q 5 min x 3 doses. Hold if SBP < 90, notify MD if held. (Class 1, Level A)
- **Nitroglycerin**: NTG 0.4 mg SL x 3 or IV NTG (Class I, Level C)
- **Morphine 2 - 4 Mg IV q 5 - 15 min for pain relief** (Class I, Level C)

What if PCI is not available?

- Treat with fibrinolytic therapy within 30 minutes of hospital presentation
  - If unable undergo PCI within 90 minutes of first medical contact
  - unless fibrinolytic therapy is contraindicated.

AMI CORE Measures within 1st 30 – 90 minutes

- Artery opened with thrombolytic within 30 min of hospital arrival
- Artery opened within 90 min of ED arrival with PCI (percutaneous coronary intervention – stent)
  - 90 minutes criteria also includes inpatients that may have chest pain
- Artery opened with PCI within 45 min of hospital arrival for patients that transfer from another hospital
- Initial EKG to ED transfer to Cath Lab within 30 minutes

Other initial AMI Core Measures

- Aspirin and Beta blocker given within 24hrs of arrival unless contra or on Coumadin therapy.
- Lipid level drawn
- ASA daily

AMI Discharge CORE Measures

- ASA
- ACE inhibitor if EF < 40%
- Beta blocker
- Lipid lowering med if LDL > 100
- Smoking Cessation Counseling

ST Fingerprint & Precordial Leads Placement

<table>
<thead>
<tr>
<th>ACS Patients with STEMI and/or Stent</th>
<th>NonCardiac Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inferior/RCA – V1 or V2</td>
<td>Lead II &amp; V1 or V2</td>
</tr>
<tr>
<td>Anterior/LAD – V3 or V4</td>
<td></td>
</tr>
<tr>
<td>Lateral/Circumflex – V5 or V6</td>
<td></td>
</tr>
<tr>
<td>V1 - 4th intercostal space (ICS)</td>
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<tr>
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<td>V4 - 5th ICS, left midclavicular line</td>
<td>V4 – 5th ICS, left midclavicular line (anterior)</td>
</tr>
<tr>
<td>V5 – 5th ICS, left anterior axillary</td>
<td>V5 – 5th ICS, left anterior axillary line (anterior)</td>
</tr>
<tr>
<td>line (lateral)</td>
<td>V6 – 5th ICS, left midaxillary line (lateral)</td>
</tr>
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AACN Practice Alert
Ventricular remodeling in the infarcted area

- Dilation & ventricular wall thinning
- Increased wall stress on the healthy myocardium
- Sets the stage for Heart Failure
- ACE Inhibitors reduce remodeling & prevent the progression of heart failure

Heart Failure is the nation's most rapidly growing cardiac problem.

- About 22% males & 46% female MI patients will be disabled with Heart Failure within 6 years.

50% Heart Failure Patients die within 5 years of HF diagnosis

Goals After Myocardial Infarction

Reducing the risk of another heart attack
- ASA
- Antithrombotic therapy
- Beta-blockers
- Statins
- ACE inhibitors

Reducing the risk of heart failure
- ACE inhibitors
- Aldosterone antagonists
- Beta-blockers

Reducing the risk of sudden cardiac death
- ICD therapy

Lipid – Lowering Agents

- Statins
  - atorvastatin (Lipitor)
  - cerivastatin (Baycol)
  - fluvastatin (Lescol)
  - lovastatin (Mevacor)
  - pravastatin (Pravachol)
  - simvastatin (Zocor)
- Fibric Acid Derivatives
  - gemfibrozil (Lopid)
  - micronized fenofibrate (Tricor)
  - clofibrate (Atromid-S)
- Bile Acid Resins
  - colestipol (Colestid)
  - cholestyramine (Questran, Questran Light, Prevalite, LoCholest)
  - colesevelam (Welchol)
- Niacin (Niaspan and other various brands)

Psychosocial Aspect AMI

I don't want to take medicine
Will it stop next time?
No smoking!!
Who will pay the bills?
Cardiac Rehab? Exercised?
What about relations with my wife?
I'm only 55
Will I die before I see my grandchildren?

Cardiac Rehabilitation

Josie Harrington, RN
Lead Registered Nurse
Optimizing Cardiac Output

Cardiac 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
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...

Renin-Angiotensin-Aldosterone System (RAAS)
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↓
Growth Factor Potent Vasoconstrictor Adrenal Cortex
Increase B/P Increase SVR
Distal Renal Tubule
Increase H2O & Na++ Reabsorption
Excretes K+ for Na+

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

ARBs “Sartans”
• Candesartan  Atacand
• Irbesartan  Avapro
• Losartan  Cozaar
• Valsartan  Diovan
• Telmisartan  Micardis
• Eprosartan  Teveten
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-tuc-ky

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 as 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.
   - Ventricles 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 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 $CI = \frac{CO}{BSA}$

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**

**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**

**The Effect of Cardiac Meds on Heart Rate**

<table>
<thead>
<tr>
<th>Increase HR</th>
<th>Decrease HR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atropine</td>
<td>Beta Blockers</td>
</tr>
<tr>
<td>Dopamine/Intopin</td>
<td>Calcium Channel Blockers</td>
</tr>
<tr>
<td>Epinephrine/Adrenalin</td>
<td></td>
</tr>
<tr>
<td>Norepinephrine/Levophed</td>
<td></td>
</tr>
<tr>
<td>Dobutamine/Dobutrex</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Slight Increase HR</th>
<th>No effect on HR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Milrinone/Primacor</td>
<td>Phenylephrine/Neo-synephrine</td>
</tr>
<tr>
<td></td>
<td>Vasopressin/Pitressin</td>
</tr>
</tbody>
</table>
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 - 130 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: Sited in Cardiac Surgery Essentials, page 148

Determinants of Cardiac Output

Cardiac Output = Heart Rate x Stroke Volume

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

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

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 10mm 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</th>
<th>High</th>
</tr>
</thead>
<tbody>
<tr>
<td>Volume</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Diuretics</td>
</tr>
<tr>
<td></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

How Cardiac Meds effect preload

- Vasoconstrictors will increase preload when started
- Vasodilators will decrease preload when started
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 dynes/sec/cm^-5
- Pulmonary Vascular Resistance (PVR) reflects RV afterload
  - Normal Range = 20-120 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: 800-1500 dyne.sec.cm^-5

SVR

< 800 = vasodilated

> 1500 = vasoconstricted

High afterload (SVR) → heart is working harder

CO and SVR

\( SVR = \frac{MAP - CVP}{CO} \times 80 \)

CO \(\underline{\text{SVR}}\)

A "Teeter-Totter" Relationship
CO and SVR

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

A “Teeter–Totter” Relationship

Most hypovolemic patients will 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:
\[PVR = \frac{MPA - PCW}{CO} \times 80\]

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
  - Scleroderma
- Vascular destruction
- Emphysema
- Pulmonary interstitial fibrosis
- Vascular Compression
  - Pneumothorax, hemotorax
- Tumor mass

Humoral Substances
- Histamine, angiotensin, fibrinopeptides
- Prostaglandin E\(_2\)
- Prostacyclin
- Sildenafil (Viagra)

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
  - Vasocostriction
  - Vasopressors
  - Hypothermia
  - Catecholamine stimulation from surgery
How Cardiac Meds effect Afterload

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
- Nitroglycerin/Tridil
- Beta Blockers
- Nicardipine/Cardene
- ACE Inhibitors

Slight Decrease Afterload
- Milrinone/Primacor

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
  - 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 CVP, PAO, PAOP</td>
<td>Diuretics Venous Vasodilation</td>
</tr>
<tr>
<td>Vasopressors</td>
<td>AFTERLOAD SVR, PVR</td>
<td>Vasodilators Calcium Channel Blockers IABP Valve Surgery</td>
</tr>
<tr>
<td>Optimize preload</td>
<td>CONTRACTION (CO/CI indirect measurement)</td>
<td>------</td>
</tr>
<tr>
<td>Pacemaker Atropine Isuprel Dopamine</td>
<td>RATE/RHYTHM Beta Blockers Calcium Channel Blockers</td>
<td></td>
</tr>
</tbody>
</table>

Pearls

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

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
- Neuro-synephrine→used to ↑ SVR when hypotension exists with normal CO
- Vasopression→ Refractory vasodilatory shock, ↓ SVR


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


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/inamrinone


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/mums if satisfactory CO
- Blood transfusion if Hematocrit < 26%
- IABP if refractory to pharmacologic interventions
- Ventricular Assist device if no response to above

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

Medications for Low Cardiac Output

**Alpha Receptors**
- Arteries & Veins

**Beta Receptors**
- Heart
- Bronchodilation

### Table 17.5: Hemodynamic Effects of Vasomotor Medications

<table>
<thead>
<tr>
<th>Medication</th>
<th>SVR</th>
<th>HR</th>
<th>PCW</th>
<th>CI</th>
<th>MAP</th>
<th>SvO₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dopamine</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Milrinone</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Norepinephrine/Levophed</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Isoproterenol</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Cardium Thrdms</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Neosynephrine</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Dopamine</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Norepinephrine/Levophed</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Phentolamine</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Norepinephrine/Levophed</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

1. Increase; 0. No change; -1, variable effect. The relative effect is indicated by the magnitude of arrow.

### Principles of **SvO₂** Monitoring

### Principles of Reflection Spectrophotometry
An "early warning" system
Evaluate efficacy of therapeutic interventions
Identify detrimental consequences of "patient care"

Does the Oxygen Supply meet the Oxygen Demand?

Fink Equation Rewritten

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

Normal Value is \(60 - 80\%\)

SvO2 Values

<table>
<thead>
<tr>
<th>Saturation (%)</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>80 or &gt;</td>
<td>Sepsis, 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.

If oxygen demand exceeds oxygen delivery

- Without oxygen, lactate cannot be reutilized through the Krebs's cycle and will accumulate in the cells leading to progressive metabolic acidosis, cellular injury and dysfunction, and eventually cellular death.

Compensatory Mechanisms to meet O\(_2\) Demands

Primary
- \(\uparrow\) Cardiac Output

Secondary
- \(\downarrow\) SvO\(_2\)

Final
- Anaerobic Metabolism

Four determinants of SvO\(_2\):

- Hemoglobin
- Cardiac output
- Arterial saturation (SaO\(_2\))
- Oxygen consumption (VO\(_2\))

\[\text{SvO}_2 < 60\]

Increase in O\(_2\) Demand
- \(\uparrow\) O\(_2\) Consumption
  - Hyperthermia
  - Seizures
  - Pain
  - Shivering

Decrease in O\(_2\) Delivery
- \(\downarrow\) Hemoglobin
  - Bleeding, anemia
- \(\downarrow\) Oxygen Saturation (SaO\(_2\))
  - Hypoxia
  - Suctioning
  - Pulmonary infiltrates
  - Decreased ventilation
- \(\downarrow\) Cardiac Output
  - Hypovolemia
  - Hypotension
  - Arrhythmias
  - Cardiogenic shock

\[\text{SvO}_2 > 80\]

Decrease in O\(_2\) Demand
- \(\downarrow\) O\(_2\) Consumption
  - Hyperthermia
  - Anesthesia
  - Pharmacological Paralysis
  - Sepsis (peripheral shunting)

Increase in O\(_2\) Delivery
- \(\uparrow\) Fi O\(_2\)
If SvO₂ < 60 for 15 minutes or longer, the tissues are not getting enough oxygen — call for further orders.

SvO₂ Algorithm

If 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

ScvO₂
Central Venous Oxygen Saturation
- Oxygen saturation measured from central venous catheter
- Normal > 70%
- < 70% extracting more oxygen than normal
- Assess for trends as with SvO₂
- May not reflect global hypoxia
Causes of Abnormal Filling Pressures

- Low PAOP/PAD
  - Bleeding
  - Third space fluid loss
  - RV failure
  - Massive PE

- High PAOP/PAD
  - LV Dysfunction
  - Systemic Hypertension
  - Constrictive pericarditis

- Low CVP
  - Bleeding
  - Third space fluid loss

- High CVP
  - RV Dysfunction
  - Pulmonary hypertension
  - PE
  - Tamponade
  - Constrictive pericarditis

Draw arrows to indicated 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>CD/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>PAD</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/SVRI</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
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<td></td>
<td></td>
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<tr>
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<tr>
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<td></td>
<td></td>
</tr>
<tr>
<td>SVR/SVRI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PVR/PVRI</td>
<td></td>
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</tr>
<tr>
<td>PAD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SV/SVI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SVR/SVRI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PVR/PVRI</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Let’s Practice!

Hemodynamics

Hypovolemia

LV Failure
# RV Failure

<table>
<thead>
<tr>
<th>Hypothesis</th>
<th>Fluid Overload</th>
<th>LV Failure</th>
<th>RV Failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO/Cl</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>CVP</td>
<td>↓</td>
<td>↑ Normal</td>
<td>↑ Normal</td>
</tr>
<tr>
<td>PAD</td>
<td>↓</td>
<td>↑ Normal</td>
<td>↑ Normal</td>
</tr>
<tr>
<td>SV/SVI</td>
<td>↓</td>
<td>↑ Normal</td>
<td>↑ Normal</td>
</tr>
<tr>
<td>SVR/SVRI</td>
<td>Normal/Increased</td>
<td>Normal</td>
<td>↑ Normal</td>
</tr>
<tr>
<td>PVR/PVRI</td>
<td>Normal</td>
<td>Normal</td>
<td>↑</td>
</tr>
</tbody>
</table>

# RV & LV Failure

<table>
<thead>
<tr>
<th>Hypothesis</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/Cl</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>CVP</td>
<td>↓</td>
<td>↑ Normal</td>
<td>↑ Normal</td>
<td>↑</td>
</tr>
<tr>
<td>PAD</td>
<td>↓</td>
<td>↑ Normal</td>
<td>↑ Normal</td>
<td>↑</td>
</tr>
<tr>
<td>SV/SVI</td>
<td>↓</td>
<td>↑ Normal</td>
<td>↑ Normal</td>
<td>↑</td>
</tr>
<tr>
<td>SVR/SVRI</td>
<td>Normal/Increased</td>
<td>Normal</td>
<td>↑ Normal</td>
<td>↑</td>
</tr>
<tr>
<td>PVR/PVRI</td>
<td>Normal</td>
<td>Normal</td>
<td>↑</td>
<td>↑</td>
</tr>
</tbody>
</table>

# What’s abnormal?

<table>
<thead>
<tr>
<th>Hypothesis</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/Cl</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>CVP</td>
<td>↓</td>
<td>↑ Normal</td>
<td>↑ Normal</td>
<td>↑</td>
</tr>
<tr>
<td>PAD</td>
<td>↓</td>
<td>↑ Normal</td>
<td>↑ Normal</td>
<td>↑</td>
</tr>
<tr>
<td>SV/SVI</td>
<td>↓</td>
<td>↑ Normal</td>
<td>↑ Normal</td>
<td>↑</td>
</tr>
<tr>
<td>SVR/SVRI</td>
<td>Normal/Increased</td>
<td>Normal</td>
<td>↑ Normal</td>
<td>↑</td>
</tr>
<tr>
<td>PVR/PVRI</td>
<td>Normal</td>
<td>Normal</td>
<td>↑</td>
<td>↑</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
What's abnormal?

<table>
<thead>
<tr>
<th>CABG on admission</th>
<th>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>
</tr>
<tr>
<td>HR</td>
<td>85</td>
<td>88</td>
</tr>
<tr>
<td>SV$_{O_2}$</td>
<td>38</td>
<td>39</td>
</tr>
<tr>
<td>CVP</td>
<td>9</td>
<td>10</td>
</tr>
<tr>
<td>PAS/PAD</td>
<td>26/16</td>
<td>28/18</td>
</tr>
<tr>
<td>PAM</td>
<td>21</td>
<td>23</td>
</tr>
<tr>
<td>PAW</td>
<td>20</td>
<td>21</td>
</tr>
<tr>
<td>SV</td>
<td>44</td>
<td>56</td>
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<tr>
<td>SVR</td>
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<td>1055</td>
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<tr>
<td>SVRI</td>
<td>1779</td>
<td>2166</td>
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<tr>
<td>PVR</td>
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<td>33</td>
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<tr>
<td>PVR1</td>
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<td>68</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?

- Fluid

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

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
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<td>92/57</td>
</tr>
<tr>
<td>MAP</td>
<td>88</td>
</tr>
<tr>
<td>HR</td>
<td>125</td>
</tr>
<tr>
<td>PAS/PAD</td>
<td>37/26</td>
</tr>
<tr>
<td>CVP</td>
<td>19</td>
</tr>
<tr>
<td>SV$_{O_2}$</td>
<td>38</td>
</tr>
<tr>
<td>CI</td>
<td>1.6</td>
</tr>
<tr>
<td>SVR</td>
<td>1031</td>
</tr>
<tr>
<td>SpO$_2$</td>
<td>92</td>
</tr>
<tr>
<td>SV</td>
<td>30</td>
</tr>
<tr>
<td>UO</td>
<td>30</td>
</tr>
</tbody>
</table>

T
Case 1: Identify abnormal hemodynamic parameters and what you would do?

<table>
<thead>
<tr>
<th>2300</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Art BP</td>
<td>92/57</td>
</tr>
<tr>
<td>MAP</td>
<td>68</td>
</tr>
<tr>
<td>HR</td>
<td>125</td>
</tr>
<tr>
<td>PAP/PAD</td>
<td>37/26</td>
</tr>
<tr>
<td>CVP</td>
<td>19</td>
</tr>
<tr>
<td>SvO2</td>
<td>32</td>
</tr>
<tr>
<td>CO</td>
<td>3.8</td>
</tr>
<tr>
<td>CI</td>
<td>1.6</td>
</tr>
<tr>
<td>SVR</td>
<td>1031</td>
</tr>
<tr>
<td>SV</td>
<td>30</td>
</tr>
<tr>
<td>UO</td>
<td>30</td>
</tr>
</tbody>
</table>

1. Answer
2. Treat tamponade

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

<table>
<thead>
<tr>
<th>2300</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Art BP</td>
<td>92/57</td>
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<tr>
<td>MAP</td>
<td>68</td>
</tr>
<tr>
<td>HR</td>
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<td>32</td>
</tr>
<tr>
<td>CO</td>
<td>3.8</td>
</tr>
<tr>
<td>CI</td>
<td>1.6</td>
</tr>
<tr>
<td>SVR</td>
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<tr>
<td>SV</td>
<td>30</td>
</tr>
<tr>
<td>UO</td>
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</table>

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

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<tbody>
<tr>
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<td>118/71</td>
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<tr>
<td>MAP</td>
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<tr>
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<td>CVP</td>
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<td>SvO2</td>
<td>45</td>
</tr>
<tr>
<td>CO</td>
<td>4.2</td>
</tr>
<tr>
<td>CI</td>
<td>1.8</td>
</tr>
<tr>
<td>SVR</td>
<td>1316</td>
</tr>
<tr>
<td>SpO2</td>
<td>95</td>
</tr>
<tr>
<td>SV</td>
<td>39</td>
</tr>
<tr>
<td>UO</td>
<td>60</td>
</tr>
</tbody>
</table>

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 inotropic: Dopamine 2.5 mcg/kg/min, Epi 3.07 mcg/min Milrinone 0.5 mcg/kg/min. Did it help?

<table>
<thead>
<tr>
<th>1300</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Art BP</td>
<td>118/71</td>
</tr>
<tr>
<td>MAP</td>
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<td>CVP</td>
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</tr>
<tr>
<td>SvO2</td>
<td>45</td>
</tr>
<tr>
<td>CO</td>
<td>4.3</td>
</tr>
<tr>
<td>CI</td>
<td>1.8</td>
</tr>
<tr>
<td>SVR</td>
<td>1316</td>
</tr>
<tr>
<td>SpO2</td>
<td>95</td>
</tr>
<tr>
<td>SV</td>
<td>39</td>
</tr>
<tr>
<td>UO</td>
<td>60</td>
</tr>
</tbody>
</table>
**Case 3: Identify abnormal hemodynamic parameters and what you would do?**

<table>
<thead>
<tr>
<th>Time</th>
<th>Art BP</th>
<th>MAP</th>
<th>HR</th>
<th>Temp</th>
<th>PAS/PAD</th>
<th>CVP</th>
<th>SVO2</th>
<th>CO</th>
<th>CI</th>
<th>SVR</th>
<th>Svo2</th>
<th>SV</th>
<th>UO</th>
<th>CT</th>
</tr>
</thead>
<tbody>
<tr>
<td>2200</td>
<td>106/38</td>
<td>62</td>
<td>83</td>
<td>99 F</td>
<td>29/14</td>
<td>1.3</td>
<td>64</td>
<td>1.3</td>
<td>1.7</td>
<td>1186</td>
<td>64</td>
<td>39</td>
<td>375 per hour</td>
<td>60</td>
</tr>
</tbody>
</table>

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

**Case 3 Answer:**

Hypovolemia. Give fluids – 250 ml 5% Albumin
Be careful when warming patient

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

<table>
<thead>
<tr>
<th>Time</th>
<th>Art BP</th>
<th>MAP</th>
<th>HR</th>
<th>Temp</th>
<th>PAS/PAD</th>
<th>CVP</th>
<th>SVO2</th>
<th>CO</th>
<th>CI</th>
<th>SVR</th>
<th>Svo2</th>
<th>SV</th>
<th>UO</th>
<th>CT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Admission</td>
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<td>83</td>
<td>68</td>
<td>97</td>
<td>32/17</td>
<td>14</td>
<td>69</td>
<td>3.5</td>
<td>1.8</td>
<td>1685</td>
<td>68</td>
<td>51</td>
<td>750</td>
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</tbody>
</table>

1. Fluids
2. Inotrope
3. Antihypertensive
4. Observe
Case 5: Identify abnormal hemodynamic parameters and what you would do?

<table>
<thead>
<tr>
<th>Admission</th>
</tr>
</thead>
<tbody>
<tr>
<td>Art BP</td>
</tr>
<tr>
<td>MAP</td>
</tr>
<tr>
<td>HR</td>
</tr>
<tr>
<td>Temp</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>
</tr>
<tr>
<td>SpO2</td>
</tr>
<tr>
<td>SV</td>
</tr>
<tr>
<td>UD</td>
</tr>
<tr>
<td>CT</td>
</tr>
</tbody>
</table>

1. Fluids

2. ANSWER

Case 4 Answer:
Warm to decrease SVR. Fluids (check Hbg) for low SV, CI

<table>
<thead>
<tr>
<th>Admission</th>
</tr>
</thead>
<tbody>
<tr>
<td>Art BP</td>
</tr>
<tr>
<td>MAP</td>
</tr>
<tr>
<td>HR</td>
</tr>
<tr>
<td>Temp</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>
</tr>
<tr>
<td>SpO2</td>
</tr>
<tr>
<td>SV</td>
</tr>
<tr>
<td>UD</td>
</tr>
<tr>
<td>CT</td>
</tr>
</tbody>
</table>

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

<table>
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<tr>
<th>0500</th>
</tr>
</thead>
<tbody>
<tr>
<td>Art BP</td>
</tr>
<tr>
<td>MAP</td>
</tr>
<tr>
<td>HR</td>
</tr>
<tr>
<td>Temp</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>
</tr>
<tr>
<td>SpO2</td>
</tr>
<tr>
<td>SV</td>
</tr>
<tr>
<td>UD</td>
</tr>
<tr>
<td>CT</td>
</tr>
</tbody>
</table>

1. Treat hypovolemia

2. ANSWER

Case 5 Answer:
Hypovolemia from bleeding. Give blood, check coags

<table>
<thead>
<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>Art BP</td>
</tr>
<tr>
<td>MAP</td>
</tr>
<tr>
<td>HR</td>
</tr>
<tr>
<td>Temp</td>
</tr>
<tr>
<td>PAS/PAD</td>
</tr>
<tr>
<td>CVP</td>
</tr>
<tr>
<td>SVo2</td>
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<td>CO</td>
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<td>SVR</td>
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</tr>
<tr>
<td>SV</td>
</tr>
<tr>
<td>UD</td>
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<tr>
<td>CT</td>
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</table>

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

<table>
<thead>
<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>Art BP</td>
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<td>SV</td>
</tr>
<tr>
<td>UD</td>
</tr>
<tr>
<td>CT</td>
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1. Treat hypovolemia

- ANSWER
Case 6: After two units of pRBCs. Did it help?

<table>
<thead>
<tr>
<th>Time</th>
<th>Art BP</th>
<th>MAP</th>
<th>HR</th>
<th>Temp</th>
<th>PAS/PAD</th>
<th>CVP</th>
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<th>CO</th>
<th>CI</th>
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<th>SpO2</th>
<th>SV</th>
<th>UO</th>
<th>CT</th>
</tr>
</thead>
<tbody>
<tr>
<td>0500</td>
<td>91/38</td>
<td>67</td>
<td>108</td>
<td>99</td>
<td>20/12</td>
<td>6</td>
<td>99</td>
<td>4.2</td>
<td>2.1</td>
<td>1180</td>
<td>93</td>
<td>43</td>
<td>40</td>
<td>200</td>
</tr>
<tr>
<td>0700</td>
<td>109/42</td>
<td>67</td>
<td>101</td>
<td>99</td>
<td>43/16</td>
<td>8</td>
<td>61</td>
<td>4.2</td>
<td>2.1</td>
<td>1180</td>
<td>95</td>
<td>43</td>
<td>40</td>
<td>100</td>
</tr>
</tbody>
</table>

Are you happy? 1. Yes  2. No

0500  | Art BP  | MAP   | HR    | Temp | PAS/PAD | CVP  | SVO2 | CO   | CI    | SVR   | SpO2 | SV  | UO  | CT   |
<table>
<thead>
<tr>
<th></th>
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<td>2.1</td>
<td>1180</td>
<td>95</td>
<td>43</td>
<td>40</td>
<td>100</td>
</tr>
</tbody>
</table>

Answer

2. No

Case 6 Answer: Still hypovolemic – needs more blood/surgery to find bleeder.

<table>
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<tr>
<th>Time</th>
<th>Art BP</th>
<th>MAP</th>
<th>HR</th>
<th>Temp</th>
<th>PAS/PAD</th>
<th>CVP</th>
<th>SVO2</th>
<th>CO</th>
<th>CI</th>
<th>SVR</th>
<th>SpO2</th>
<th>SV</th>
<th>UO</th>
<th>CT</th>
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<tr>
<td>0700</td>
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<td>67</td>
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<td>99</td>
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<td>4.2</td>
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<td>1180</td>
<td>95</td>
<td>43</td>
<td>40</td>
<td>100</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

The Mechanics of Balloon Counterpulsation
**IABP**

Inflation

↑ Coronary artery perfusion

Deflation

Decrease afterload

**Contraindications**

- Severe aortic insufficiency
- Abdominal or aortic aneurysm
- Severe calcific aorta-iliac disease or peripheral vascular disease

**Potential Side Effects and Complications**

- Limb ischemia
- Bleeding at the insertion site
- Thrombocytopenia
- Migration of the balloon catheter
- Balloon leak
- Infection
- Aortic dissection
- Compartment syndrome

**Arterial Waveform Variations During IABP Therapy**

**Timing Errors - Early Inflation**

Inflation of the IAB prior to aortic valve closure

Waveform Characteristics:

- Inflation of IAB prior to dicrotic notch
- Diastolic augmentation encroaches onto systole (may be unable to distinguish)

Physiologic Effects:

- Potential premature closure of aortic valve
- Potential increase in LVEDV and LVEDP or PCWP
- Increased left ventricular wall stress or afterload
- Aortic regurgitation
- Increased MVO₂ demand
### Timing Errors - Late Inflation

**Inflation of the IAB markedly after closure of the aortic valve**

**Waveform Characteristics:**
- Inflation of the IAB after the dicrotic notch
- Absence of sharp V
- Sub-optimal diastolic augmentation

**Physiologic Effects:**
- Sub-optimal coronary artery perfusion

### Timing Errors - Early Deflation

**Premature deflation of the IAB during the diastolic phase**

**Waveform Characteristics:**
- Deflation of IAB is seen as a sharp drop following diastolic augmentation
- Sub-optimal diastolic augmentation
- Assisted aortic end-diastolic pressure may be equal to or less than the unassisted aortic end-diastolic pressure
- Assisted systolic pressure may rise

**Physiologic Effects:**
- Sub-optimal coronary artery perfusion
- Angina may occur as a result of retrograde coronary blood flow
- Sub-optimal afterload reduction
- Increased MVO₂ demand

### Timing Errors - Late Deflation

**Deflation of the IAB as the aortic valve is beginning to open**

**Waveform Characteristics:**
- Assisted aortic end-diastolic pressure may be equal to the unassisted aortic end-diastolic pressure
- Rate of rise of assisted systole is prolonged
- Diastolic augmentation may appear widened

**Physiologic Effects:**
- Afterload reduction is essentially absent
- Increased MVO₂ consumption due to the left ventricle ejecting against a greater resistance and a prolonged isovolumetric contraction phase
- IAB may impede left ventricular ejection and increase the afterload