Care of the Complex Cardiac Medical & Surgical Patient

Fun and Focused
CMC-CSC Review

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BEACON AWARD OF EXCELLENCE

CMC Exam Content

- 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.

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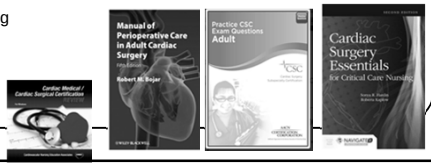
CSC Exam Blueprint

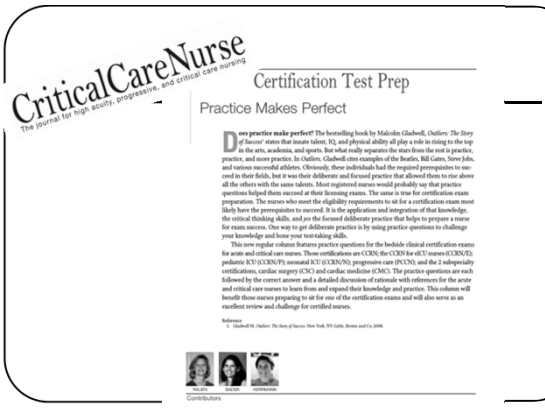
- Cardiovascular Patient Care Problems (33%)
- Other Patient Care Problems (24%)
- Nursing Interventions (33%)
- Monitoring & Diagnostics (9%)

Care of the Cardiac Surgery Patient first 48 hours Post op

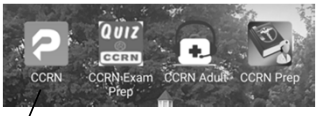
Study Help

- Bojar, r. (2011). Manual of Perioperative Care in Adult Cardiac Surgery. 5th ed. West Sussex, UK: Wiley-blackwell.
- Hardin, S, & Kaplow, R. (2016). *Cardiac Surgery Essentials for Critical Care Nursing*, 2nd ed. Jones & Bartlett.
- Todd, B. (2005). *Cardiothoracic Surgical Nursing Secrets.* Mosby/Elsevier.
- Dodge, T. *Fast Facts for the Cardiac surgery nurse*. Springer Publishing
- www.aacn.org





Study Apps to Download

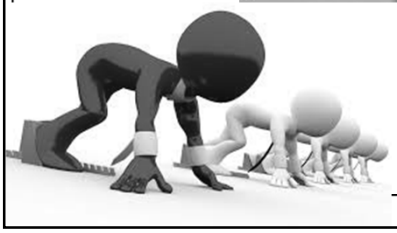


This green one can be set to send a question every day

- 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

CMC

I. CARDIOVASCULAR PATIENT CARE PROBLEMS (47%)
- A. Acute Coronary Syndrome
  1. ST segment elevation myocardial infarction
  2. Non-ST segment elevation myocardial infarction
  3. Unstable angina
- B. Dysrhythmias
  1. Lethal ventricular dysrhythmias
  2. Bradydysrhythmias
  3. Tachydysrhythmias
  4. Conduction defects and blocks

CSC

- Acute Coronary Syndrome

Correlate the Coronary Arteries

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

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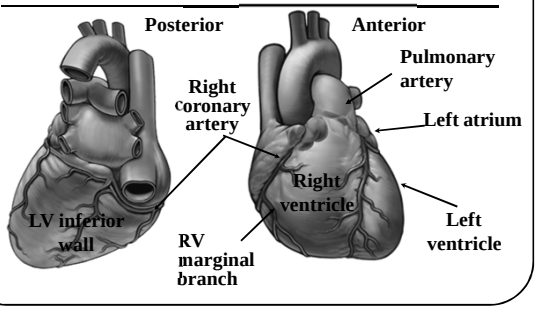
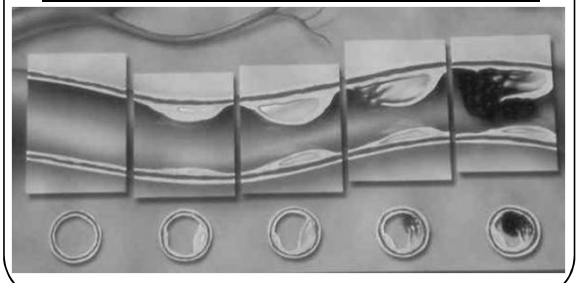
Cardiac Anatomy

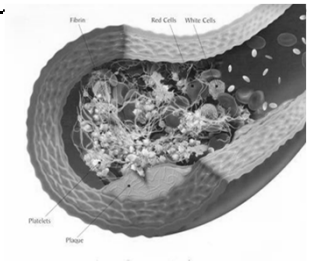


Atherosclerosis

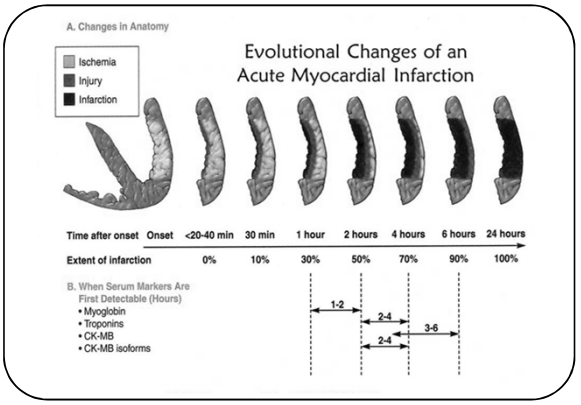


Cascade effects of atherosclerotic plaque rupture

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



ARTERY OCCULUSION



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

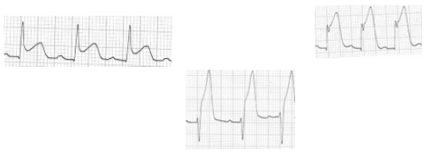
Ischemia < 20 Minutes
Lack of oxygen to the myocardial muscle
- **Peaked T Waves**
- **Inverted T Waves**
- **ST Segment Depression**

Ischemia = screaming for oxygen

EKG Changes with MI: Injury

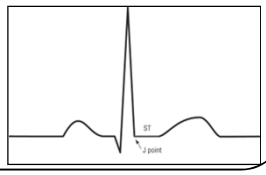
Injury 20 - 40 minutes
When the period of ischemia is prolong more than a few minutes, ischemic areas of the heart become damaged (injured)

- **ST segment elevation**

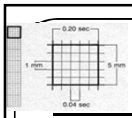


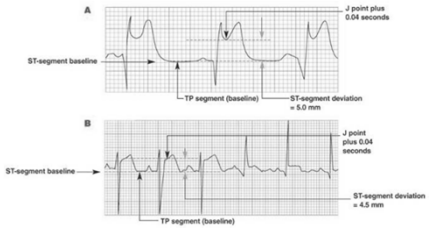
The ST Segment

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



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.

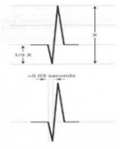
Source: AHA ACLS EP

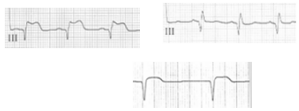
EKG Changes with MI: Infarction

Infarction > 1 - 2 hours

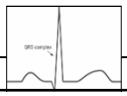
- **Abnormal Q waves**
  - **> 1/3 the height of R wave in that lead**
    or
  - **> 0.03 ms wide**

Abnormal (significant Q waves)

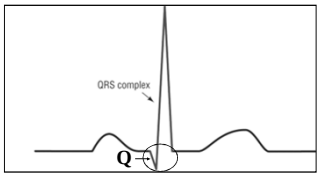




Normal Q wave
1st downward deflection of QRS

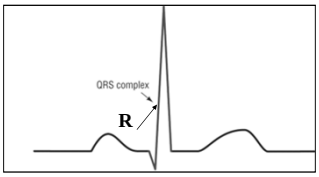


Normal QRS complex – The Q wave



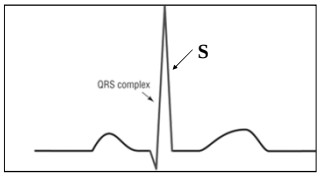
- Q wave is the first negative deflection after the p wave
- Always first may or may not be there.
- Comes first in the alphabet
- There are normal and abnormal Q waves

Normal QRS complex – The R wave



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

Normal QRS complex – The S wave

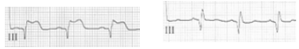


- S wave is the second negative deflection after the R wave
- *Slipping* down
- Always after R wave like in the alphabet

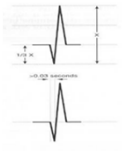
EKG Changes with MI: Infarction

Infarction > 1 - 2 hours

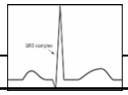
- **Abnormal Q waves**
  - **> 1/3 the height of R wave in that lead**
    or
  - **> 0.03 ms wide**



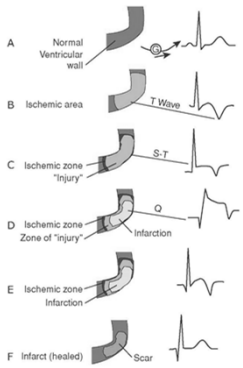
Abnormal (significant Q waves)



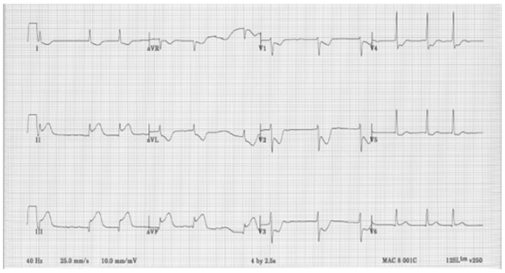
Normal Q wave
1st downward deflection of QRS



Evolution of STEMI

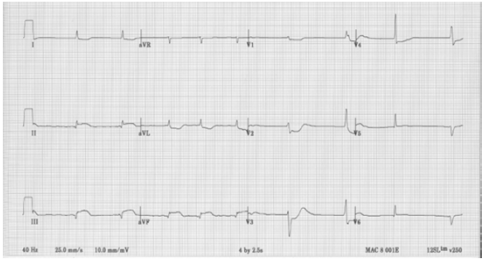


Evolving AMI:
EM #1 December 13 at 1701

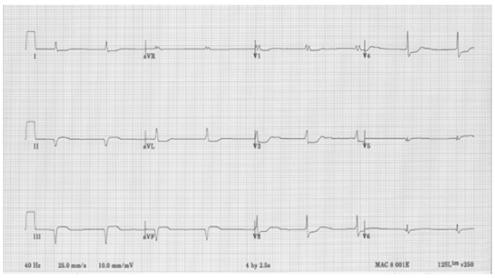


EM # 2 December 13 at 1823

EM #3 December 14 at 0630

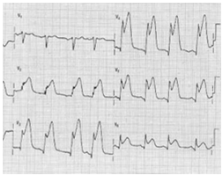


EM # 4 December 15 0600

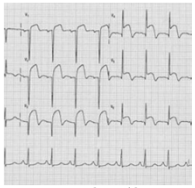


Hyperacute Phase of MI

- Occurs within minutes to first hour of chest pain
- Very tall ST segment
- Tall peaked symmetrical T waves



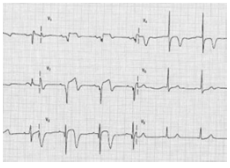
Within minutes



Later – within 1st hour

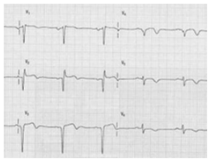
Acute Phase of MI

- 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



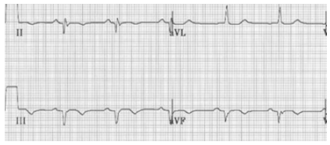
Evolving Phase of MI

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

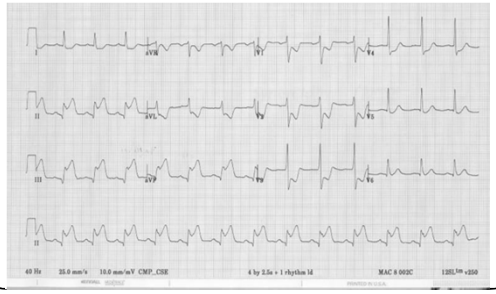


Resolving Phase of MI

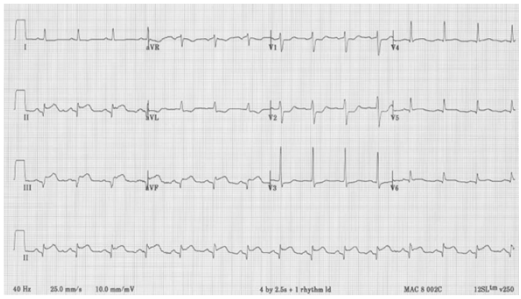
- Occurs in second week
- ST segment returns to normal
- T wave stops inverting and stays that way for 2 weeks and then resolves and will be low voltage
- Q wave stops deepening and stays due to dead tissue
- R wave stops decreasing and stays due to dead tissue



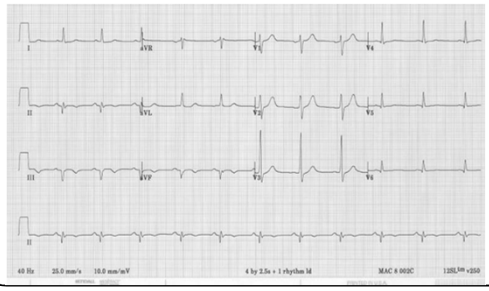
CC: Admission EKG 1445



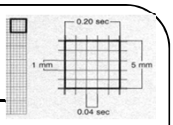
CC: 3 hours later



CC: 28 hours later



Positive EKG

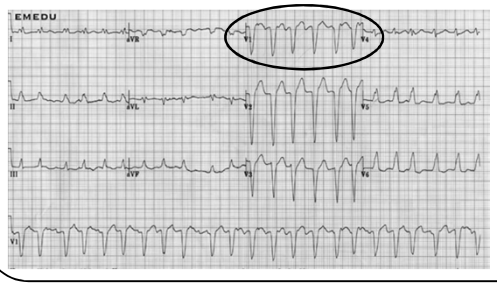


- ST elevation > O.1mV (1 mm) in at least 2 contiguous precordial leads or at least 2 adjacent limb leads (STEMI) (Class 1, Level A)

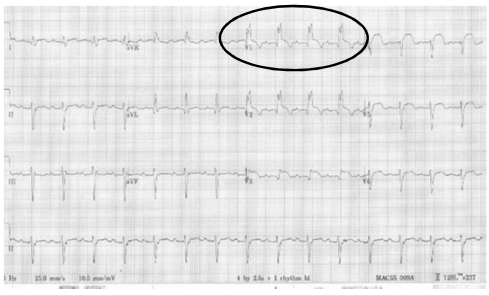
I lateral	aVR	V_1 septal	V_4 anterior
II inferior	aVL lateral	V_2 septal	V_5 lateral
III inferior	aVF inferior	V_3 anterior	V_6 lateral

- 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)

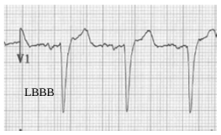


RBBB = QRS > 0.12 sec
Positive QRS in V1 (rabbit ears)

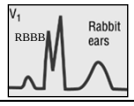


BBB = QRS > 0.12sec

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



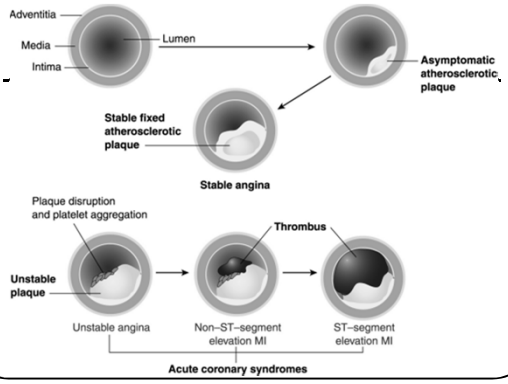
- RBBB = QRS > 0.12 sec
  Positive QRS in V1
  (rabbit ears)



NSTEMI

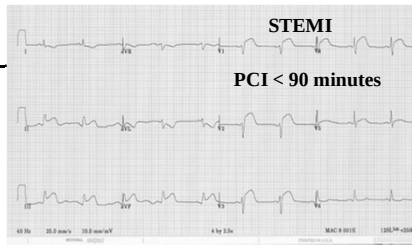
Non ST Segment Elevation MI

- No ST segment Elevation
- ST segment depression

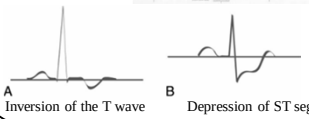


Both elevated Troponins

NSTEMI



PCI within 24 - 48 hours



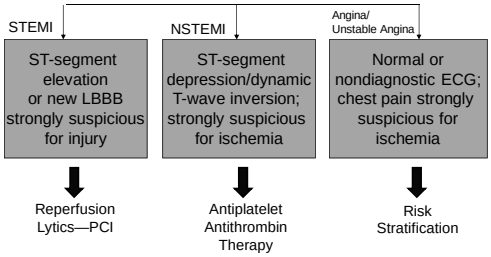
Inversion of the T wave

Depression of ST segment

Differentiating MIs

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

Treatment Strategies



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**

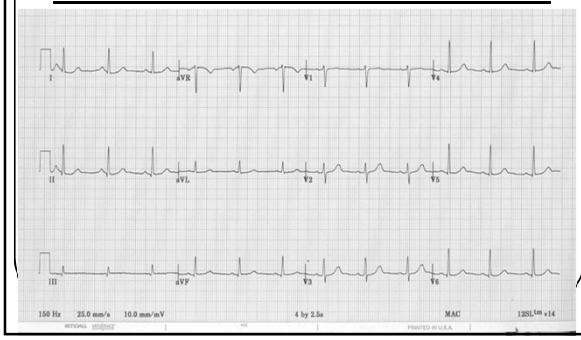
Types of Angina	Definition
Angina	Myocardial Anoxia
Exertional Angina (4 Es) (usually sign of atherosclerosis)	Pain with increased myocardial oxygen demand • Exertion • Eating • Extreme Emotions • Exposure to Cold
Prinzmetal's Angina or Variant Angina (thought to be a coronary spasm)	Pain at rest, during sleep or without evidence of provocation
Stable Angina	Exertional angina with consistent symptoms –typically relieved with rest or cessation of cause and possibly NTG
Unstable Angina (crescendo or preinfarction angina) Partially occluding thrombus	Recent onset (within 2 months) Severely limits activity Differs from the person's "typical exertional angina" May occur at rest RX with Anti-platelets Fibrinolytic therapy *is not* effective

12 Lead EKG

Understanding Lead Placement

12 Lead EKG 101

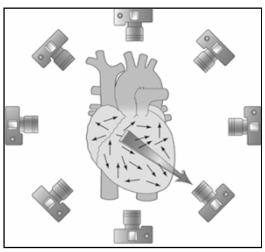
Learn the *Normal* so you can detect the abnormal



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

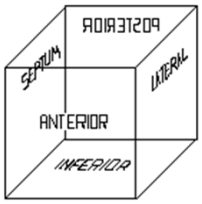
I, II, III

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

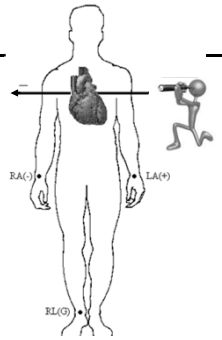
- **Also referred to as extremity leads due to placement on the body**
- **Record electrical forces two points equidistant from the heart.**
- **Each lead has two poles: one positive & one negative**
- **Two leads to give the picture**
- **Current travels Negative to Positive to create the electrical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

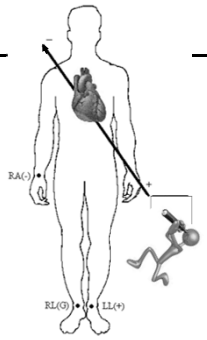


Lead II

Views the heart from left leg to right arm

Area: Inferior

Artery: RCA

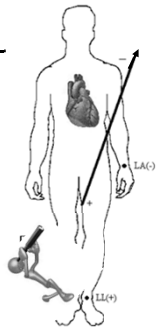


Lead III

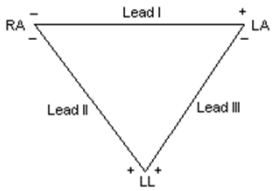
Views the heart from left leg to left arm

Area: Inferior

Artery: RCA



Einthoven's Triangle



By connecting the electrodes of the limb leads, the Einthoven's Triangle is formed.

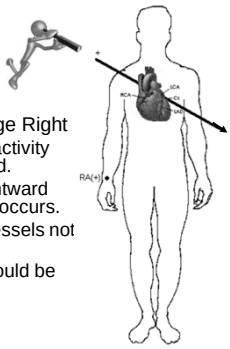
Augmented Limb Leads AVR, AVL, AVF

- 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



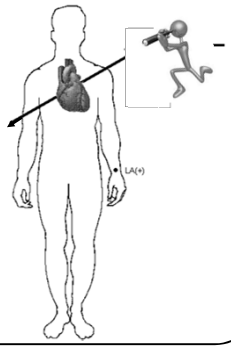
AVL

Augmented Voltage Left

Views the heart from the left arm to the mid-point between right arm & right leg

Area: Lateral

Artery: Circumflex



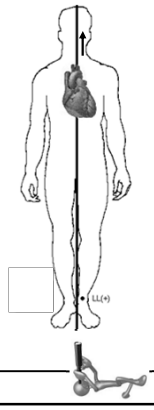
AVF

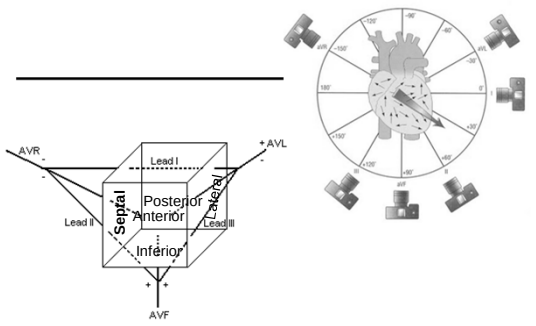
Augmented Voltage Foot

Views the heart from the feet to the chin

Area: Inferior

Artery: RCA

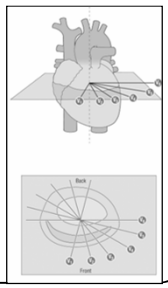




Correlation of the leads to the areas of left ventricle by superimposing Einthoven's Triangle over the cube.

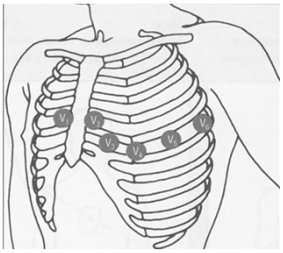
The Precordial System Chest Leads V1 – V6

- 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

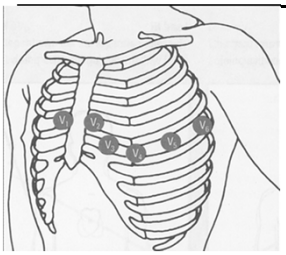


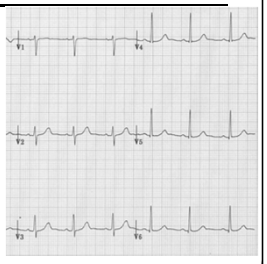
Precordial Leads Placement

- **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)



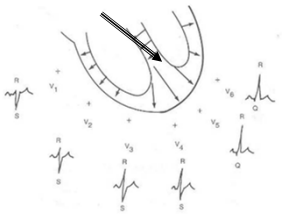
Precordial Leads

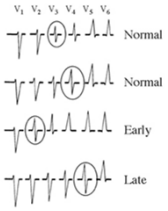




R Wave Transition

R: Rises above baseline



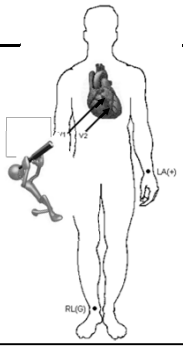


V1 & V2

Views the septum of the heart

Area: Septal

Artery: LAD

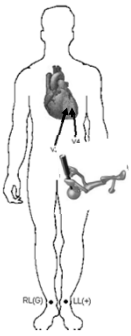


V3 & V4

Views the anterior area of the left ventricle

Area: Anterior

Artery: LAD

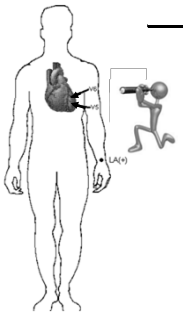


V5 & V6

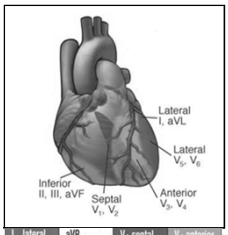
Views the lateral area of the left ventricle

Area: Lateral

Artery: Circumflex



Cardiac Anatomy in Relation to Coronary Artery



I lateral	aVR	V_1 septal	V_4 anterior
II inferior	aVL lateral	V_2 septal	V_5 lateral
III inferior	aVF inferior	V_3 anterior	V_6 lateral

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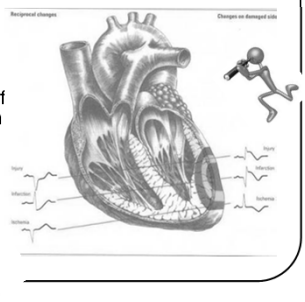
ACLS pocket cards or poster

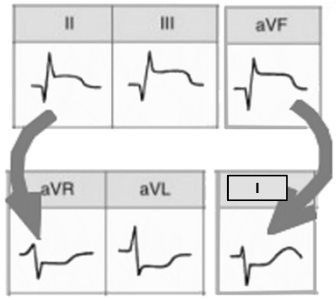
Reciprocal Changes

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

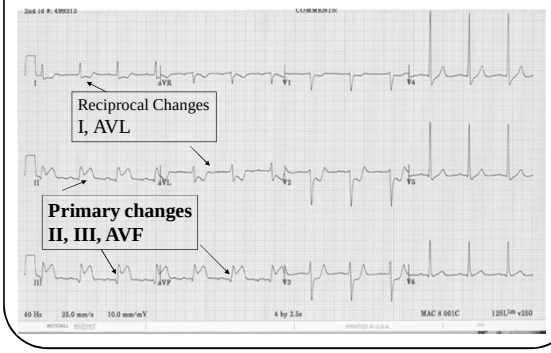
- Mirror Image
- Two different electrodes viewing AMI from opposite angles
- Example: Take photo of male from front and from back – still a male but different view



Inferior STEMI

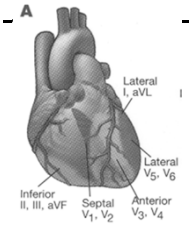


Reciprocal leads



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*



- Inferior ↔ Lateral
  - II, III, AVF ↔ I, AVL, V5, V6
- Anterior ↔ Posterior
  - V1 – V4

I lateral	aVR	V_1 septal	V_4 anterior
II inferior	aVL lateral	V_2 septal	V_5 lateral
III inferior	aVF inferior	V_3 anterior	V_6 lateral

Reciprocal Changes

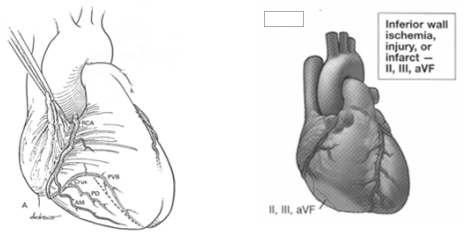
Reciprocal Leads that are opposite the damaged area will show opposite EKG Changes

- 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

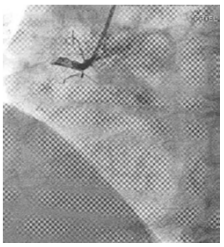
Differential Diagnosis

12 Lead EKG in Acute Coronary Syndrome

Right Coronary Artery RCA
Inferior Wall
II, III, AVF



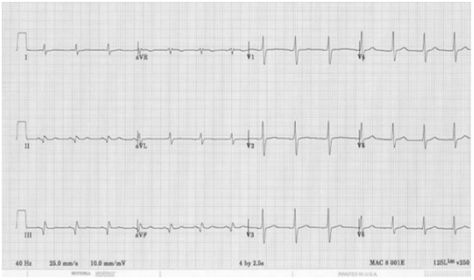
Occluded RCA



RCA post stent

Inferior Injury

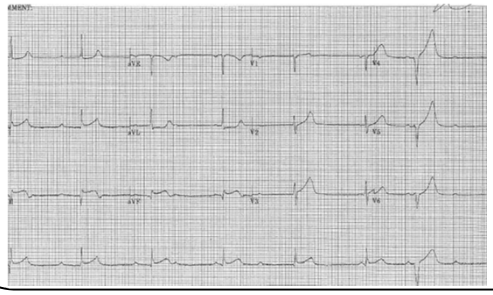
Old Inferior Infarction



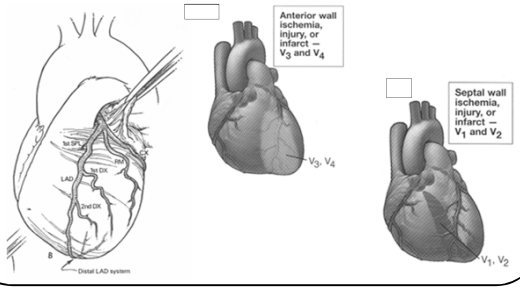
Inferior AMI

- 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

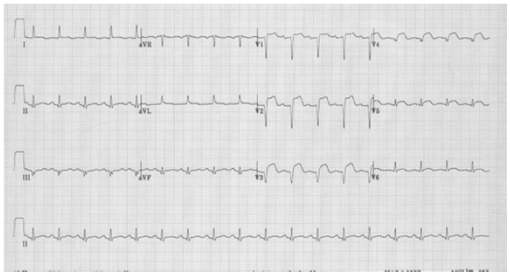
Inferior AMI
What's the rhythm?



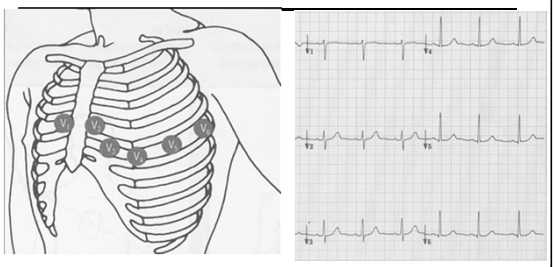
Left Anterior Descending Artery LAD
Anterior Wall V3 & V4
Septal Wall V1 & V2



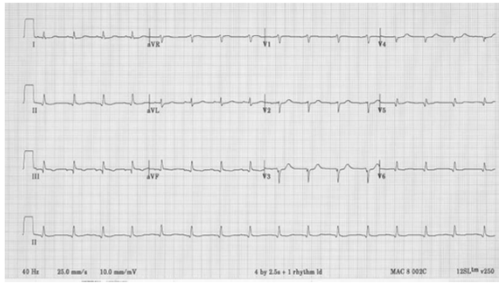
Anterior-septal Injury



Precordial Leads – Know normal

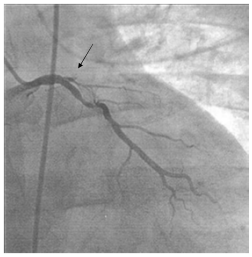


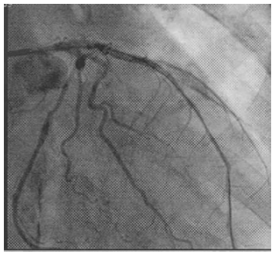
Old Anterior Infarction



Occluded High LAD

LAD post stent

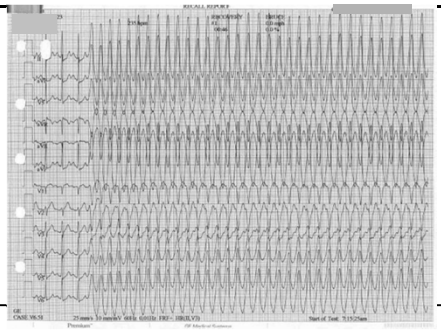




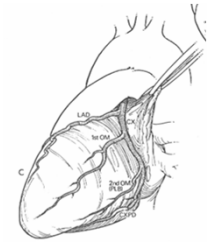
Anterior AMI

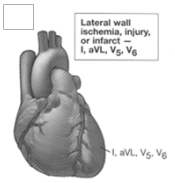
- Lose the most muscle mass
- Usually have the lowest EF
- Arrhythmias = VT or VF

Stress Test – Angio found 3 occlusions in the LAD

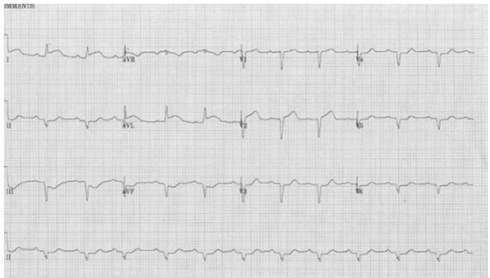


Circumflex Artery Cx
Lateral Wall
I, AVL V5 & V6

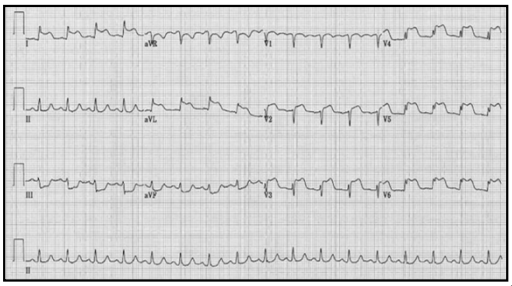




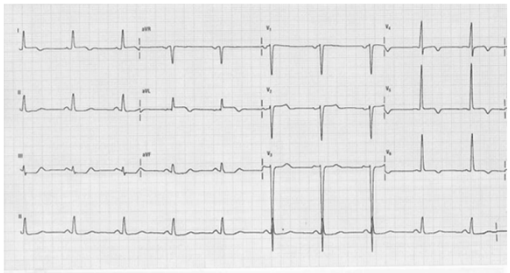
High Lateral Injury



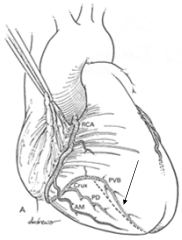
Anterior Lateral Injury



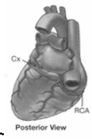
Lateral Ischemia



Posterior Descending Artery PDA Posterior Wall Reciprocal Changes

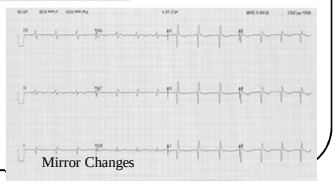


- Usually see with Inferior AMI as RCA wraps around the back of the heart and changes to PDA
- Reciprocal Changes V1 , V2, V3
  (ST segment depression or Tall R Waves)
- 18 Lead EKG

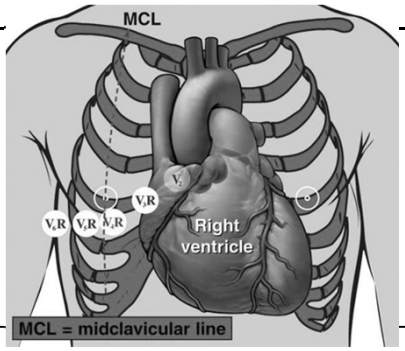


Inferior & Posterior Injury

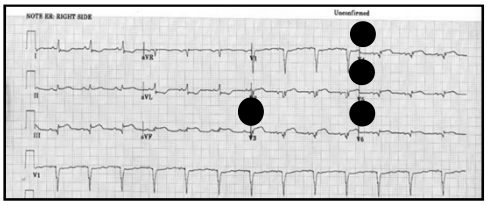
Old Inferior-posterior Infarction



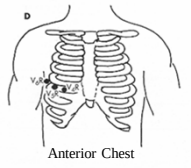
Lead Placement: Right-Sided ECG



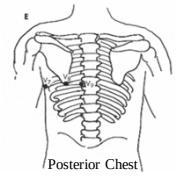
Right-Sided 12-Lead ECG: Patient With Inferior ST-Segment Changes



18 Lead EKG



Anterior Chest

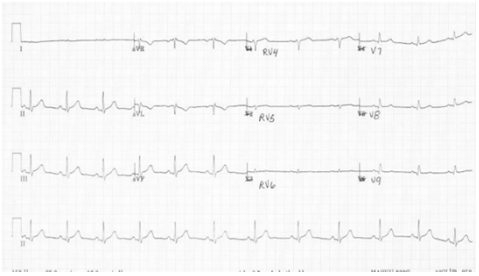


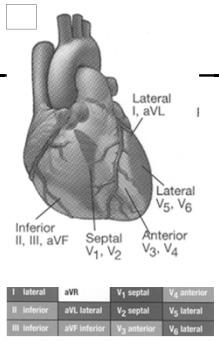
Posterior Chest

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

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





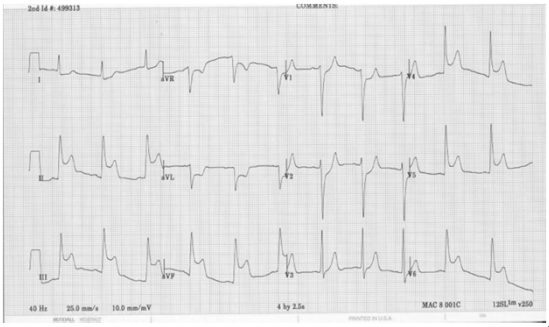
I lateral	aVR	V_1 septal	V_4 anterior
II inferior	aVL lateral	V_2 septal	V_5 lateral
III inferior	aVF inferior	V_3 anterior	V_6 lateral

Pattern to Read EKG
Be consistent

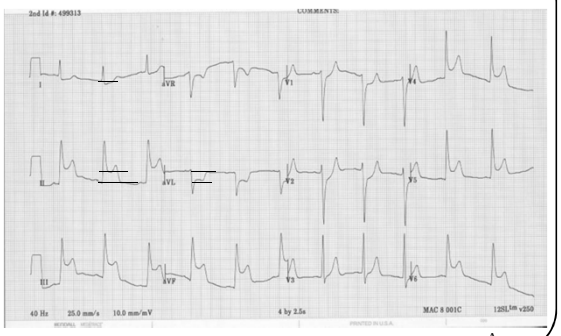
- Rate & Rhythm
- QRS Interval V1 – for RBBB or LBBB
- QT interval
- Normal Depolarization – If not, why not
- ST & T waves
- What lead is abnormal and what other lead goes with it
- Evaluate axis
- Evaluate for hypertrophy

Example & Analysis Time

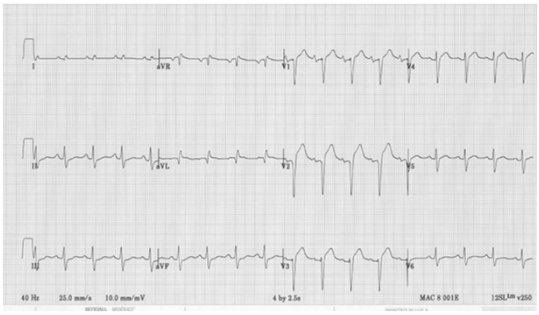
Peaked T waves
T Wave inversion
ST Depression
ST Elevation
Q waves
Type of AMI
Coronary Artery Involved



A

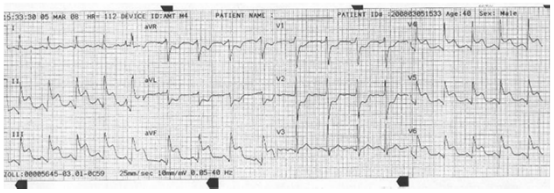


A



B

48 y/o male has crushing chest pain Calls 911



E

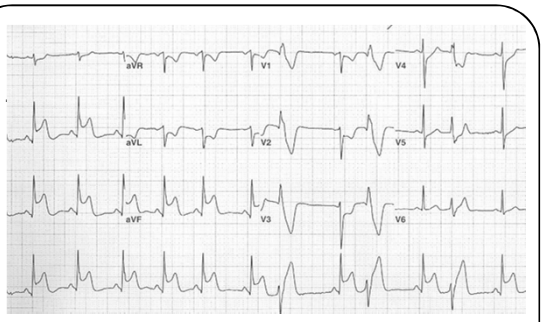
Door to PCI time = 49 minutes

- 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.

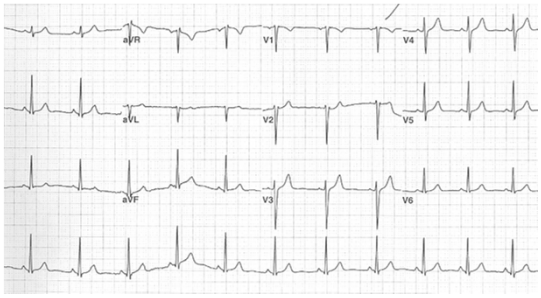
- Pain free on arrival to ED
- Alert, Oriented
- Skin Warm/dry
- When laid down for EKG developed chest pain
- BP 122/77, HR 87, RR 20 SpO2 99%
- Chest pain 7/10
- Weight: 70 kg



- **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**

F

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



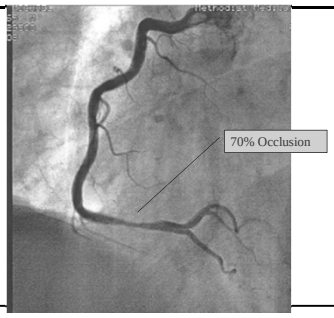
- **At 1339 on 2-24 (6 minutes later), the chest pain was gone. Pt was sitting up at the time. This is the 12 Lead EKG.**

G

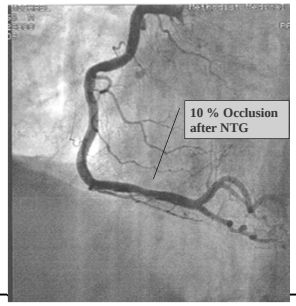
- **Serial troponin levels & lipid levels ordered**
- **Troponin < 0.4 ng/ml**
- **CK = 71**
- **Total Cholesterol = 161**
- **Triglycerides = 66**
- **HDL = 35**
- **LDL = 113**

- 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

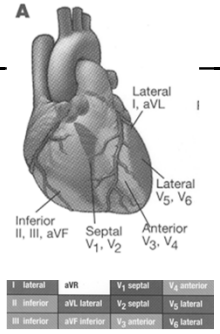


Cardiac Cath Feb 25
RCA after administration of Intracoronary Nitroglycerin



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

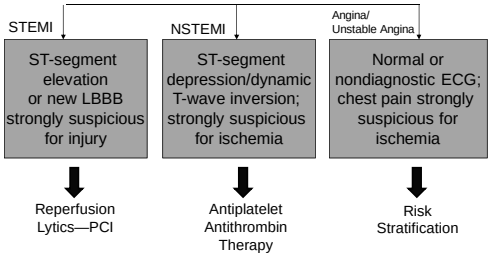
59 minutes Door to PCI Time!

Benchmark < 90 minutes

Methodist Heart, Lung and Vascular Institute

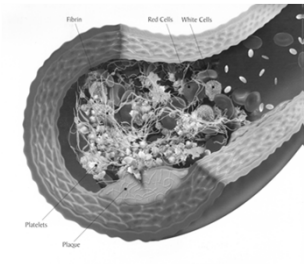
Taking You Well INTO THE FUTURE

Treatment Strategies



Cascade effects of atherosclerotic plaque rupture

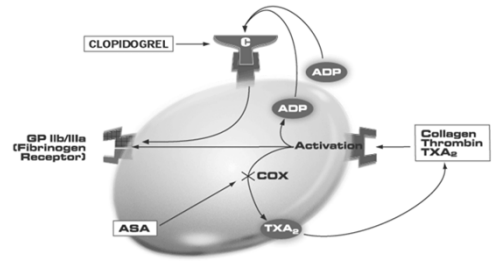
- Platelet aggregation
- Fibrin accumulation
- Thrombus formation
- Bleeding into the plaque
- Vasospasm



ARTERY OCCULUSION

Clotting Cascade is like a freeway with multiple entrances

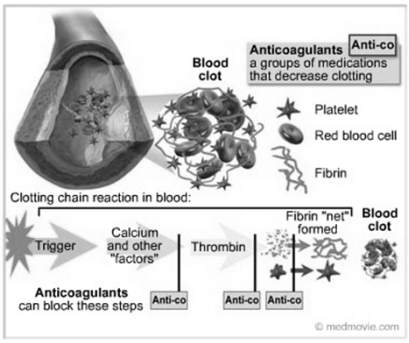
Complementary Mode of Action between Clopidogrel and ASA



COX, cyclooxygenase; ADP, adenosine diphosphate; TxA_2, thromboxane A_2

Schafer AI *Am J Med* 1996;101:199–209

Anticoagulants



Antiplatelets

P2Y12 (ADP) platelet inhibitor

- Clopidogrel (Plavix®)
- Prasugrel (Effient®)
- Ticagrelor (Brilinta®)

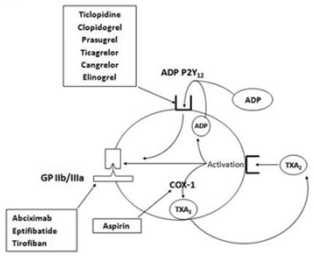
Inhibition of platelet COX-1

- ASA

IIB/IIIa Receptor Inhibitors

- Abciximab (Repro)
- Eptifibatide (Integrilin)
- Tirofiban (Aggrastat)

Figure 1



Oral Anticoagulants

Direct thrombin inhibitor

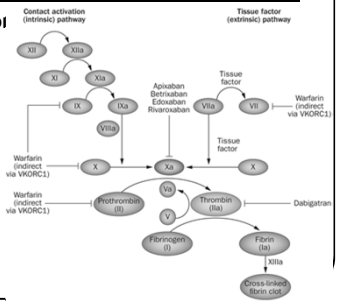
- Dabigatran (Pradaxa)

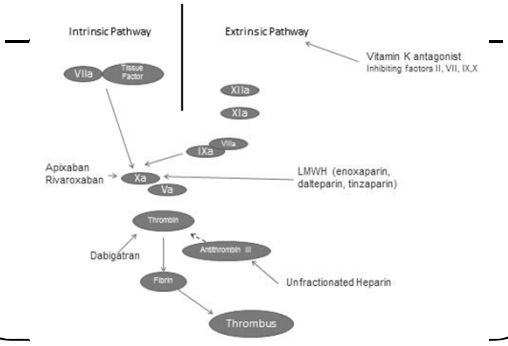
Factor Xa inhibitor

- Rivaroxaban (Xarelto)
- Apixaban (Eliquis)

Vitamin K Antagonist

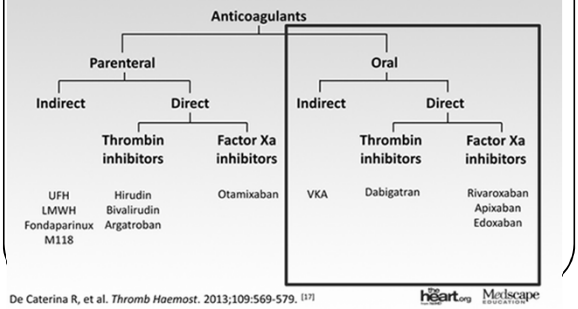
- Warfarin (Coumadin)





Currently Available Anticoagulants

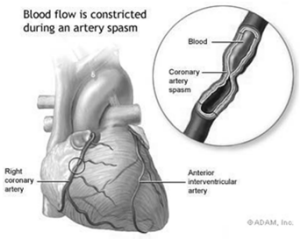
Classification based on route of administration and mode of action



De Caterina R, et al. *Thromb Haemost*. 2013;109:569-579. [17]

Other Causes of STEMI

- Coronary vessel spasm
- Coronary emboli
- Vasculitis
- Severe chest trauma



What's the common complication of all the anticoagulants?

Bleeding!

♥Heart Attack Signs & Symptoms for Males

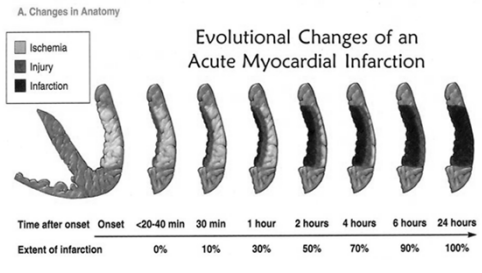
- **Chest Pain**
- **Pain radiating down arms**
- **Jaw Pain**
- **Sweating**
- **Nausea**

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 call 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
  Or
- Artery opened within 90 min of ED arrival with PCI (percutaneous coronary intervention – stent)
  90 minutes critieria also includes inpatients that may have chest pain
  Or
- 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

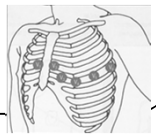
ACS Patients with STEMI and/or Stent

- **Inferior/RCA – V1 or V2**
- **Anterior/LAD – V3 or V4**
- **Lateral/Circumflex – V5 or V6**

NonCardiac Patients

- **Lead II & V1 or V2**

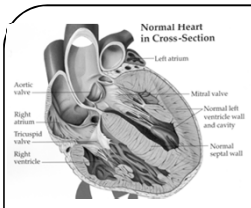
- **V1 4th intercostal space (ICS) right sternal border (septum)**
- **V2 4th ICS, left sternal border (septum)**
- **V3 Midway between V2 and V4 (anterior)**
- **V4 5th ICS, left midclavicular line (anterior)**
- **V5 5th ICS, left anterior axillary line (lateral)**
- **V6 5th ICS, left midaxillary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



AACN Practice Alert

Ventricular remodeling in the infarcted area

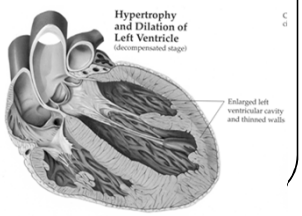
- Dilation & ventricular wall thinning
- Increased wall stress on the healthy myocardium
- Sets the stage for Heart Failure
- ACE Inhibitors reduce remodeling & prevent the progression of heart failure



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**

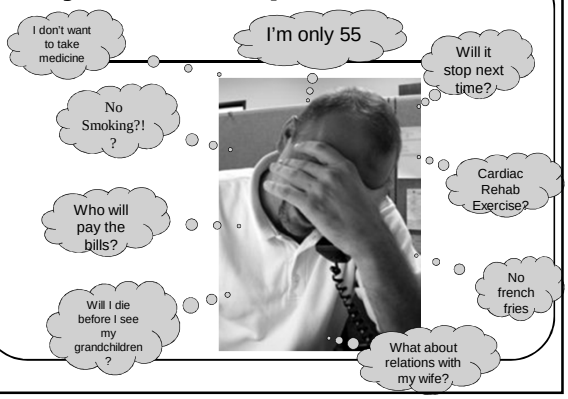
Reducing the risk of heart failure
- **ACE inhibitors**
- **Aldosterone antagonists**
- **Beta-blockers**

Reducing the risk of sudden cardiac death
- **ICD therapy**

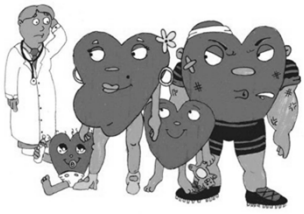
Lipid – Lowering Agents

- Statins
  - atorvastatin (Lipitor)
  - cerivastatin (Baychol)
  - 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



Optimizing Cardiac Output



Cardiac Hemodynamics/Medications

CMC

IV. MONITORING AND DIAGNOSTICS (16%)
- A. Cardiovascular
  1. Invasive hemodynamic monitoring (i.e., pulmonary artery catheter)
  2. Noninvasive hemodynamic monitoring
     - a. transesophageal echocardiography
     - b. transthoracic echocardiography
  3. ECG interpretation
     - a. ST segment monitoring
     - b. telemetry (e.g., dysrhythmia)
     - c. 12-lead ECG
  4. Blood studies
  5. Stress testing
     - a. exercise
     - b. pharmacologic
- B. Pulmonary
  1. Arterial blood gases (ABGs)
  2. Mixed venous gases
  3. Pulse oximetry

CSC

IV. MONITORING AND DIAGNOSTICS (5%)
- A. Hemodynamic Monitoring
  1. CVP monitoring (without pulmonary artery catheter)
  2. Mixed venous monitoring - SvO_2
  3. Standard pulmonary artery catheter
- B. Other Monitoring
  1. Pulse oximetry
- C. Diagnostics
  1. Arterial blood gas (ABG) interpretation
  2. ST segment monitoring
  3. Serum lactate
  4. 12-lead ECG interpretation
  5. Chest x-ray interpretation for:
     - a. placement of central line
     - b. placement of endotracheal tube
     - c. placement of IABP
     - d. placement of NG tube
     - e. pneumothorax
     - f. pulmonary edema

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**

ß-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
  - ↓ O_2 wasteage
- Antiarrhythmic effect
- Increase oxygen supply
  - Increased diastolic perfusion
  - Less exercise vasocontriction

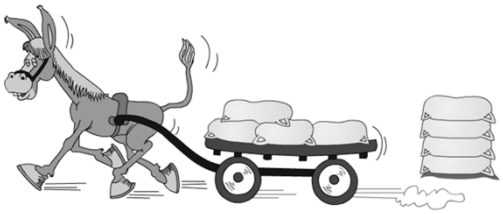
  Side effect: May promote spasm in vasospastic angina

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…**

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
↓
Angiotensin II
↓ ↓ ↓
Growth Factor | Potent Vasoconstrictor | Adrenal Cortex
↓ ↓
Increases B/P | Aldosterone
↓ ↓
Increases SVR | Distal Renal Tubule
↓
Increases H2O & Na++ Reabsorption
↓
Excretes K+ for Na+

Renin-Angiotensin-Aldosterone System (RAAS)

Low Cardiac Output/Hypotension/Hypovolemia
Decreased Renal perfusion
↓
Afferent Arteriole (baroreceptors)
↓
Release Renin (a messenger)
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Go to Liver to stimulate Angiotensin I production
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↓ ↓
Increases B/P | Aldosterone
↓ ↓
Increases SVR | Distal Renal Tubule
↓
Increases H2O & Na++ Reabsorption
↓
Excretes K+ for Na+

Dilatation & Diuresis

ACE Inhibitors "Prils"

Angiotensin II receptor Blockers "ARBs" "Sartans"

ACE Inhibitors "Prils"

- Benazepril Lotensin
- Captopril Capoten
- Lisinopril Zestril
  Prinivil
- Quinapril Accupril
- Ramipril Altace

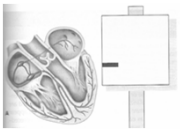
ARBs "Sartans"

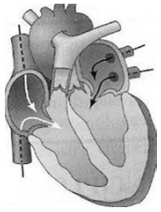
- Candesartan Atacand
- Irbesartan Avapro
- Losartan Cozaar
- Valsartan Diovan
- Telmisartan Micardis
- Eprosartan Teveten

The Cardiac Cycle

Systole and Diastole

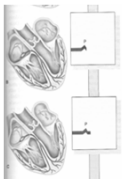
1. Rapid Ventricular Filling

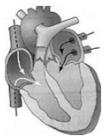




- Pressure in the atria overcomes the pressure in the ventricles; the A-V valves open.
- First third of diastole.
- S3 would be heard here --- right after dub-- sounds like Ken-tuc-ky

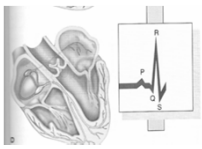
2. Active Ventricular Filling

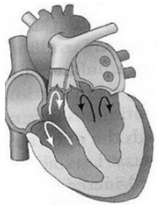




- "Atrial kick" forcing 30% more blood into the ventricles.
- Last third of diastole.
- S4 would be heard here -- sounds like Ten-nes-see - produced by vibration of atria contracting

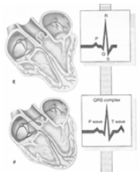
3. Isometric Contraction

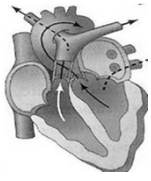




- Pressure in ventricles overcomes pressure in the atria.
- Blood tries to flow back in to the atria
- A-V valves slam shut; S1 is heard --The lub in normal lub dub - heard loudest as Apex
- Semilunar valves have not yet opened; all cardiac valves are closed.

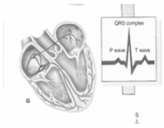
4. Ventricular Systole

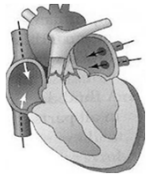




- Pressure in ventricles overcomes that in the aorta and pulmonary artery.
- Semilunar valves open.
- Ventricles contract.
- Blood is ejected forcibly into aorta and pulmonary artery.

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 nomral lub - dub; heard loudest at the base
- A-V valves are not yet open. *All* cardiac valves are closed again.

Cardiac Cycle

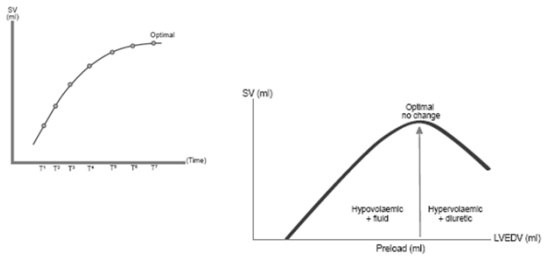
- Occurs every 0.8 seconds (HR=80)
- 0.3 seconds for systole
- 0.5 seconds for diastole

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 = 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?

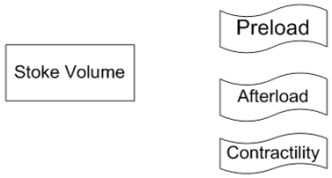
By using formula CI = CO/BSA

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

Determinants of CO:

Rate/Rhythm

Low	High
Pacemaker Atropine Isuprel Dopamine	Beta blockers Calcium channel blockers Other

How Cardiac Meds effect Heart Rate

The Effect of Cardiac Meds on Heart Rate

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

Decrease HR
- Beta Blockers
- Calcium Channel Blockers

Slight Increase HR

Milrinone/Primacor

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

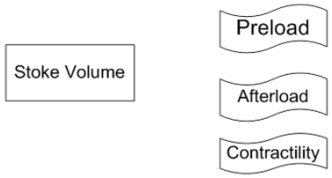
Know Normal Values!

Parameter	Normal Values
Cardiac Output (CO)	4 - 8 l/min
Cardiac Index (CI)	2.5 – 4.2 l/min/m^2
Right atrial pressure (CVP)	0 – 8 mmHg
Pulmonary artery pressure (PAS/PAD)	15 - 30/6 -12 mmHg
Pulmonary artery occlusive pressure	4 – 12 mmHg
Systemic vascular resistance (SVR)	770 – 1500 dyne/sec/cm^5
Pulmonary vascular resistance (PVR)	20 – 120 dyne/sec/cm^5
Stroke Volume (SV)	60 -130 mL/beat
Stroke Volume Index (SVI)	30 – 65 mL/beat/m^2
Arterial oxygenation saturation	95 – 100 %
Venous oxygenation saturation	60 – 80 %

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)

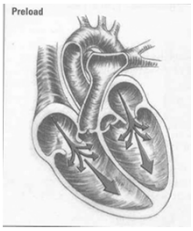
- SV: Volume of blood ejected with each beat
  - Normal SV: 60 – 100ml
- SVI: the amount of blood pumped with each beat indexed to BSA
  - Normal SVI: 33 – 47 ml/m2
  - Very powerful indicator of ventricular function

Interpretation of SV/SI

- If <u>low</u>, 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 <u>high</u>, 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

Low	High
Volume	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!

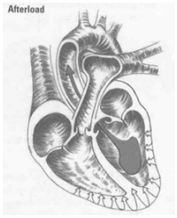
Which CABG patient needs volume?

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

How Cardiac Meds effect preload

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

Afterload



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

Afterload;
pushing…

Straw

VS.

Garden Hose

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.cm5

SVR

< 800 = vasodilated

> 1500 = vasoconstricted

High afterload (SVR)→ heart is working harder

CO and SVR

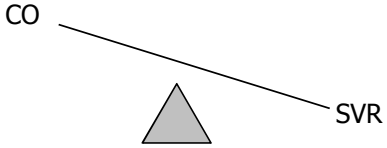
$SVR = \frac{MAP - CVP}{CO} \times 80$

CO SVR

A "Teeter –Totter" Relationship

CO and SVR

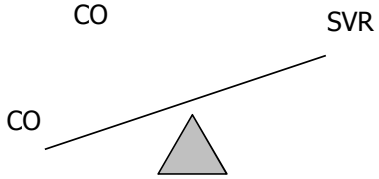
$$SVR = \frac{MAP - CVP}{CO} \times 80$$



A "Teeter –Totter" Relationship

CO and SVR

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



A "Teeter –Totter" Relationship

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.

$$\text{Equation: } PVR = \frac{MPA - PCW}{CO} \times 80$$

Normal Range: 20 - 120 dyne.sec.cm5

Factors That **Increase** Pulmonary Vascular Resistance

Chemical Stimuli
- Alveolar hypoxia
- Acidosis
- Hypercapnia

Pharmacologic Agents
- Epinephrine
- Norepinephrine
- Dobutamine
- Phenylephrine

Hyperinflation
- Mechanical Ventilation
- Continuous Positive Airway Pressure (CPAP)
- Positive End Expiratory Pressure (PEEP)

Pathologic Factors
- Vascular Blockage
  - Pulmonary emboli, air bubbles, tumor mass
- Vascular wall disease
  - Sclerosis, endarteritis, polyarteritis, scleroderma
- Vascular destruction
  - Emphysema
  - Pulmonary interstitial fibrosis
- Vascular Compression
  - Pneumothorax, hemothorax
  - Tumor mass

Humoral Substances
- 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	Increased
▪ Vasodilation ▪ Vasodilation from rewarming ▪ Vasodilator therapies ▪ Preop beta blockers ▪ Sepsis	▪ Right ▪ Pulmonary hypertension ▪ Hypoxemia ▪ Pulmonic stenosis ▪ Left ▪ Severe LV dysfunction ▪ Vasoconstriction ▪ Vasopressors ▪ Hypothermia ▪ ↑ catecholamine simulation 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

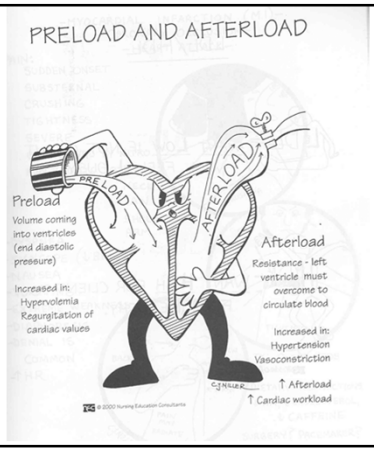
- Nitroprusside/Nipride
  - Arterial vasodilator
- Nitroglycerin/Tridil
  - Venous vasodilator
- Beta Blockers
- Nicardipine/Cardene
- ACE Inhibitors

Slight Decrease Afterload

- Milrinone/Primacor

Afterload

Low	High
▪ Vasopressors	▪ Warming blanket ▪ Vasodilators ▪ Calcium channel blockers ▪ IABP



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
  - Milirinone

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 intropes work!

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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

LOW	CARDIAC OUTPUT Treatment Options	HIGH
Volume	**PRELOAD** CVP, PAD, PAOP	Diuretics Venous Vasodilation
Vasopressors	**AFTERLOAD** SVR,PVR	Vasodilators Calcium Channel Blockers IABP Valve Surgery
Optimize preload Inotropes Calcium Ventricular Assist Devices Avoid/treat acidosis	**CONTRACTILITY** CO/CI indirect measurement	-----
Pacemaker Atropine Isuprel Dopamine	**RATE/RHYTHM**	Beta Blockers Calcium Channel Blockers

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
    - ↑MvO_2

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

Source: Bojar. R. 2011. Manual of Perioperative Care in Adult Cardiac Surgery,5th ed

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

Source: Bojar. R. 2011. Manual of Perioperative Care in Adult Cardiac Surgery,5th ed

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
  - Phenlephrine if satisfactory CO
  - Vasopressin 0.01 – 0.07 units/mins if satisfactory CO
- Blood transfusion if Hematocrit < 26%
- IABP if refractory to pharmacologic interventions
- Ventricular Assist device if no response to above

Source: Bojar. R. 2011. Manual of Perioperative Care in Adult Cardiac Surgery,5th ed

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

Medications for Low Cardiac Output

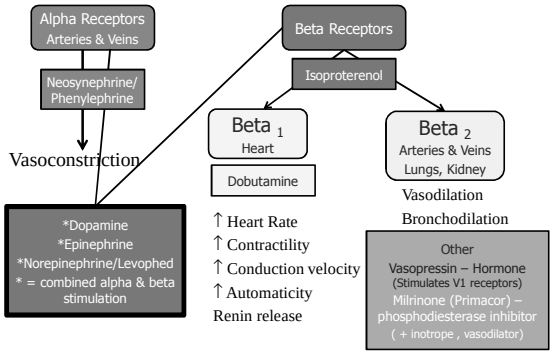


Table 11.5 • Hemodynamic Effects of Vasoactive Medications

Medication	SVR	HR	PCW	CI	MAP	MvO_2
Dopamine	↓↑	↑↑↑	↓↑	↑	↓↑	↑
Dobutamine	↓	↑↑↑	↓	↑	↓↔↑	↑↔
Epinephrine	↓↑	↑↑	↓↑	↑	↑	↑
Milrinone/ Inamrinone	↓↓	↑	↓	↑	↓	↓↑
Isoproterenol	↓↓	↑↑↑↑	↓	↑	↓↑	↑↑
Calcium chloride	↑	↔	↑	↑	↑↑	↑
Norepinephrine	↑↑	↑↑	↑↑	↑	↑↑↑	↑
Phenylephrine	↑↑	↔	↑	↔	↑↑	↔↑
Vasopressin	↑↑	↔	↑	↔	↑↑↑	↔↑
Nesiritide	↓	↔	↓↓	↑*	↓	↓↓

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

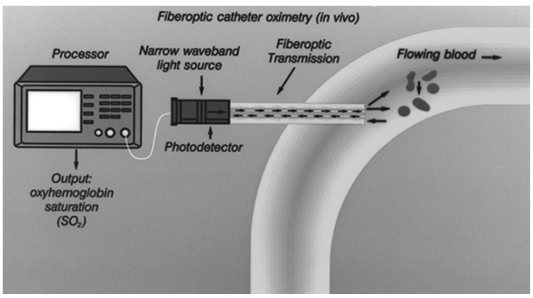
Note:
1. The effect may vary with dosage level (particularly dopamine and epinephrine, in which case the effect seen at low dose is indicated by the first arrow).
2. For some medications, an improvement in MAP may occur from the positive inotropic effect despite a reduction in SVR.
3. The effects of inamrinone, milrinone, and calcium are not mediated by α and β receptors.

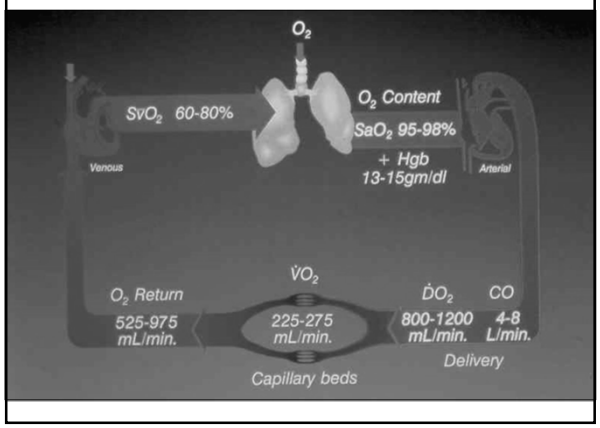
*indirect effect.

Source: Bojar. R. 2011. Manual of Perioperative Care in Adult Cardiac Surgery,5th ed

Principles of **SvO_2** Monitoring

Principles of Reflection Spectrophotometry

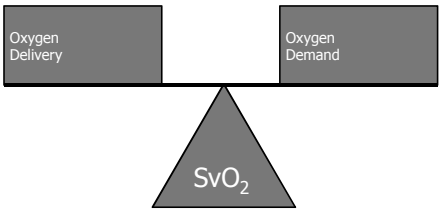




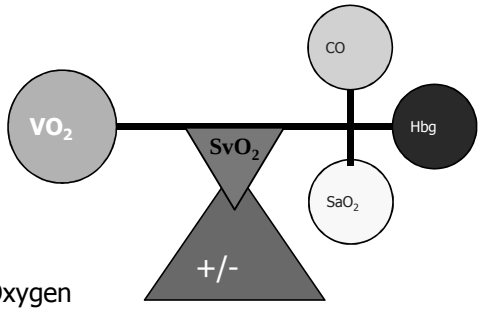
SvO_2

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

Does the Oxygen Supply meet the Oxygen Demand?



SvO_2 reflects the delicate balance between oxygen delivery and oxygen consumption



Oxygen Consumption – Oxygen Demand

Fink Equation Rewritten

$$\mathbf{SvO_2} = SaO_2 \text{ -- } \frac{VO_2}{CO \times Hgb \times 1.34 \times 10}\ \%$$

Normal Value is
60 – 80%

SvO_2 Values

Saturation %	Condition
80 or >	***Sepsis, L R Shunt Excess inotrope Hypothermia Cell poisoning Wedged catheter***
60 - 80	***Normal Range***
60	***Cardiac Decompensation***
53	***Lactic Acidosis***
32	***Unconsciousness***

- A single isolated value out of context of other clinical information is probably not very helpful

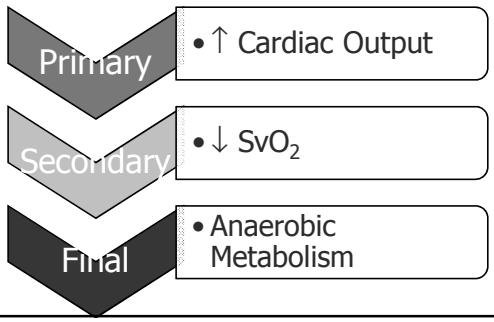
If oxygen demand exceeds oxygen delivery

- 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



Four determinants of SvO_2:

- Hemoglobin
- Cardiac output
- Arterial saturation (SaO_2)
- Oxygen consumption (VO_2)

SvO_2 < 60

Increase in O_2 Demand

- ↑ O_2 Consumption
  - Hyperthermia
  - Seizures
  - Pain
  - Shivering

Decrease in O_2 Delivery

- ↓ Hemoglobin
  - Bleeding, anemia
- ↓ Oxygen Saturation (SaO_2)
  - Hypoxia
  - Suctioning
  - Pulmonary infiltrates
  - Decreased ventilation
- ↓ Cardiac Output
  - Hypovolemia
  - Hypotension
  - Arrhythmias
  - Cardiogenic shock

SvO_2 > 80

Decrease in O_2 Demand

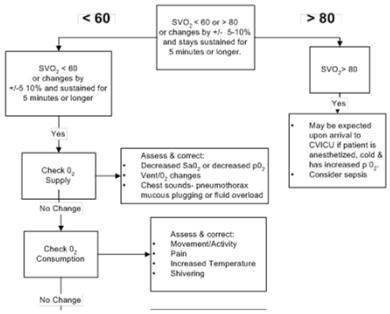
- ↓ O_2 Consumption
  - Hypothermia
  - Anesthesia
  - Pharmalogical Paralysis
  - Sepsis (peripheral shunting

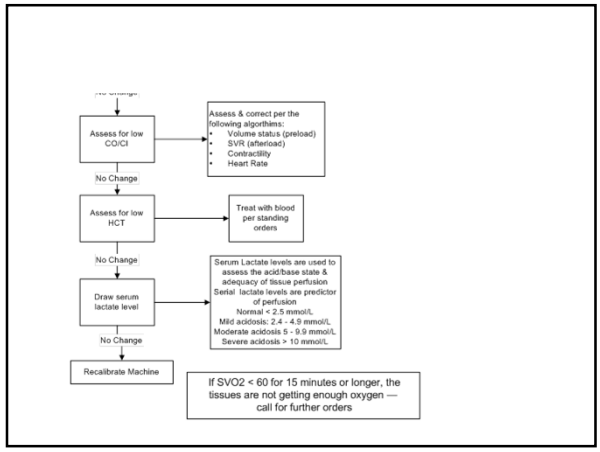
Increase in O_2 Delivery

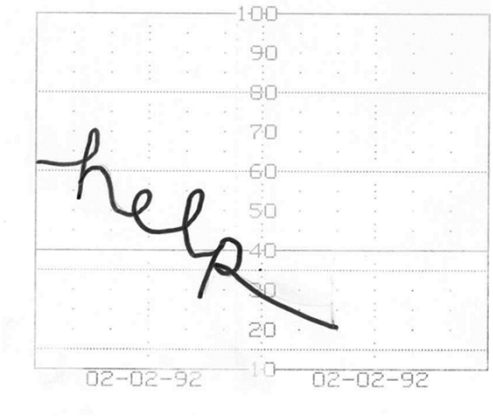
- ↑ Fi O_2

If SVO2 < 60 for 15 minutes or longer, the tissues are not getting enough oxygen — call for further orders

SvO_2 Algorithm







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

$ScvO_2$

Central Venous Oxygen Saturation

- Oxygen saturation measured from central venous catheter
- Normal > 70%
- < 70% extracting more oxygen than normal
- Assess for trends as with SvO_2
- 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 Dysfuntion
  - Systemic Hypertension
  - Constrictive pericarditis
- Low CVP
  - Bleeding
  - Third space fluid loss
- High CVP
  - RV Dysfunction
  - Pulmonary hypertension
  - PE
  - Tamponade
  - Constrictive pericarditis

Hemodynamics Let's Practice!

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

	Hypovolemia	Fluid Overload	LV failure	RV failure	RV & LV failure	Sepsis
CO/CI						
CVP						
PAD						
SV/SVI						
SVR/SVRI						
PVR/PVRI						

Hypovolemia

	Hypovolemia
CO/CI	↓
CVP	↓
PAD	↓
SV/SVI	↓
SVR/SVRI	Normal/increased
PVR/PVRI	Normal

Fluid overload

	Hypovolemia	Fluid Overload
CO/CI	↓	Nx or ↓
CVP	↓	↑
PAD	↓	↑
SV/SVI	↓	↑
SVR/SVRI	Normal/increased	Normal
PVR/PVRI	Normal	Normal

LV Failure

	Hypovolemia	Fluid Overload	LV failure
CO/CI	↓	Nx or ↓	↓
CVP	↓	↑	Normal
PAD	↓	↑	↑
SV/SVI	↓	↑	↓
SVR/SVRI	Normal/increased	Normal	↑
PVR/PVRI	Normal	Normal	Normal

RV Failure

	Hypovolemia	Fluid Overload	LV failure	RV failure
CO/CI	↓	Nx or ↓	↓	↓
CVP	↓	↑	Normal	↑
PAD	↓	↑	↑	Normal
SV/SVI	↓	↑	↓	↓
SVR/SVRI	Normal/increased	Normal	↑	Normal
PVR/PVRI	Normal	Normal	Normal	↑

RV & LV Failure

	Hypovolemia	Fluid Overload	LV failure	RV failure	RV & LV failure
CO/CI	↓	Nx or ↓	↓	↓	↓
CVP	↓	↑	Normal	↑	↑
PAD	↓	↑	↑	Normal	↑
SV/SVI	↓	↑	↓	↓	↓
SVR/SVRI	Normal/ increased	Normal	↑	Normal	↑
PVR/PVRI	Normal	Normal	Normal	↑	↑

	Hypovolemia	Fluid Overload	LV failure	RV failure	RV & LV failure	Sepsis
CO/CI	↓	Nx or ↓	↓	↓	↓	↑
CVP	↓	↑	Normal	↑	↑	↓
PAD	↓	↑	↑	Normal	↑	↓
SV/SVI	↓	↑	↓	↓	↓	↓
SVR/SVRI	Normal/ increased	Normal	↑	Normal	↑	↓
PVR/PVRI	Normal	Normal	Normal	↑	↑	↓

	CABG on admission Dopamine 2.5 mcg/kg/min
CO/CI	3.7/1.8
SBP/DBP	115/53
MAP	71
HR	85
$Sv0_2$	38
CVP	9
PAS/PAD	26/16
PAM	21
PAW	20
SV	44
SVR	1339
SVRI	2779
PVR	22
PVRI	45

What's abnormal?

	CABG on admission Dopamine 2.5 mcg/kg/min
CO/CI	3.7/1.8
SBP/DBP	115/53
MAP	71
HR	85
$Sv0_2$	38
CVP	9
PAS/PAD	26/16
PAM	21
PAW	20
SV	44
SVR	1339
SVRI	2779
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
CO/CI	3.7/1.8
SBP/DBP	115/53
MAP	71
HR	85
$Sv0_2$	38
CVP	9
PAS/PAD	26/16
PAM	21
PAW	20
SV	44
SVR	1339
SVRI	2779
PVR	22
PVRI	45

Answer How do you want to treat?

1. Fluid

	CABG on admission Dopamine 2.5 mcg/kg/min	30 minutes later after 250 ml 5% albumin
CO/CI	3.7/1.8	4.9/2.4
SBP/DBP	115/53	123/55
MAP	71	74
HR	85	88
$Sv0_2$	38	39
CVP	9	10
PAS/PAD	26/16	29/18
PAM	21	23
PAW	20	21
SV	44	56
SVR	1339	1055
SVRI	2779	2166
PVR	22	33
PVRI	45	68

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	4.9/2.4
SBP/DBP	115/53	123/55
MAP	71	74
HR	85	88
$Sv0_2$	38	39
CVP	9	10
PAS/PAD	26/16	29/18
PAM	21	23
PAW	20	21
SV	44	56
SVR	1339	1055
SVRI	2779	2166
PVR	22	33
PVRI	45	68

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
CO/CI	3.7/1.8	4.9/2.4
SBP/DBP	115/53	123/55
MAP	71	74
HR	85	88
$Sv0_2$	38	39
CVP	9	10
PAS/PAD	26/16	29/18
PAM	21	23
PAW	20	21
SV	44	56
SVR	1339	1055
SVRI	2779	2166
PVR	22	33
PVRI	45	68

Answer How do you want to treat?

1. Fluid

	CABG on admission Dopamine 2.5 mcg/kg/min	30 minutes later after 250 ml 5% albumin	36 hours later 500 ml 5% albumin & Dopamine 1 mcg/kg/min
CO/CI	3.7/1.8	4.9/2.4	6.5/3.1
SBP/DBP	115/53	123/55	133/40
MAP	71	74	69
HR	85	88	75
$Sv0_2$	38	39	55
CVP	9	10	12
PAS/PAD	26/16	29/18	40/19
PAM	21	23	27
PAW	20	21	26
SV	44	56	86
SVR	1339	1055	701
SVRI	2779	2166	1455
PVR	22	33	12
PVRI	45	68	26

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

	2300
Art BP	92/57
MAP	68
HR	125
PAS/PAD	37/26
CVP	19
SVO_2	32
CO	3.8
CI	1.6
SVR	1031
SpO_2	92
SV	30
UO	30

T

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

	2300
Art BP	92/57
MAP	68
HR	125
PAS/PAD	37/26
CVP	19
SVO_2	32
CO	3.8
CI	1.6
SVR	1031
SpO_2	92
SV	30
UO	30

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

T

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

	2300
Art BP	92/57
MAP	68
HR	125
PAS/PAD	37/26
CVP	19
SVO_2	32
CO	3.8
CI	1.6
SVR	1031
SpO_2	92
SV	30
UO	30

1. Answer
2. Treat tamponade

T

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

	2300
Art BP	92/57
MAP	68
HR	125
PAS/PAD	37/26
CVP	19
SVO_2	32
CO	3.8
CI	1.6
SVR	1031
SpO_2	92
SV	30
UO	30

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

	1300
Art BP	118/71
MAP	80
HR	107
PAS/PAD	37/26
CVP	23
SVO2	45
CO	4.2
CI	1.8
SVR	1316
SpO2	95
SV	39
UO	60

T2

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

	1300
Art BP	118/71
MAP	80
HR	107
PAS/PAD	37/26
CVP	23
SVO2	45
CO	4.2
CI	1.8
SVR	1316
SpO2	95
SV	39
UO	60

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

T2

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

	1300
Art BP	118/71
MAP	80
HR	107
PAS/PAD	37/26
CVP	23
SVO2	45
CO	4.2
CI	1.8
SVR	1316
SpO2	95
SV	39
UO	60

1. Answer
2. Treat tamponade

T2

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 intropes :

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

	1300
Art BP	118/71
MAP	80
HR	107
PAS/PAD	37/26
CVP	23
SVO2	45
CO	4.2
CI	1.8
SVR	1316
SpO2	95
SV	39
UO	60

Case 3: Identify abnormal hemodynamic parameters and what you would do?

	2200
Art BP	106/38
MAP	62
HR	83
Temp	99 F
PAS/PAD	29/14
CVP	13
SVO2	64
CO	3.3
CI	1.7
SVR	1186
SpO2	100
SV	39
UO	375 per hour
CT	60

H

Case 3: Identify abnormal hemodynamic parameters and what you would do?

	2200
Art BP	106/38
MAP	62
HR	83
Temp	97 F
PAS/PAD	29/14
CVP	13
SVO2	64
CO	3.3
CI	1.7
SVR	1186
SpO2	100
SV	39
UO	375 per hour
CT	60

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

H

Case 3: Identify abnormal hemodynamic parameters and what you would do?

	2200
Art BP	106/38
MAP	62
HR	83
Temp	97 F
PAS/PAD	29/14
CVP	13
SVO2	64
CO	3.3
CI	1.7
SVR	1186
SpO2	100
SV	39
UO	375 per hour
CT	60

1. Treat hypovolemia
2. ANSWER

H

Case 3 Answer:
Hypovolemia. Give fluids – 250 ml 5% Albumin
Be careful when warming patient

	2200
Art BP	106/38
MAP	62
HR	83
Temp	97 F
PAS/PAD	29/14
CVP	13
SVO2	64
CO	3.3
CI	1.7
SVR	1186
SpO2	100
SV	39
UO	375 per hour
CT	60

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

	Admission
Art BP	142/52
MAP	83
HR	68
Temp	97
PAS/PAD	32/17
CVP	14
SVO2	69
CO	3.5
CI	1.8
SVR	1685
SpO2	97
SV	51
UO	750
CT	210

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

	Admission
Art BP	142/52
MAP	83
HR	68
Temp	97
PAS/PAD	32/17
CVP	14
SVO2	69
CO	3.5
CI	1.8
SVR	1685
SpO2	97
SV	51
UO	750
CT	210

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

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

	Admission
Art BP	142/52
MAP	83
HR	68
Temp	97
PAS/PAD	32/17
CVP	14
SVO2	69
CO	3.5
CI	1.8
SVR	1685
SpO2	97
SV	51
UO	750
CT	210

1. Fluids
2. ANSWER

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

	Admission
Art BP	142/52
MAP	83
HR	68
Temp	97
PAS/PAD	32/17
CVP	14
SVO2	69
CO	3.5
CI	1.8
SVR	1685
SpO2	97
SV	51
UO	750
CT	210

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

	0500
Art BP	91/38
MAP	58
HR	108
Temp	99
PAS/PAD	20/12
CVP	6
SVO2	59
CO	3.6
CI	1.8
SVR	1006
SpO2	93
SV	33
UO	40
CT	200

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

	0500
Art BP	91/38
MAP	58
HR	108
Temp	98
PAS/PAD	20/12
CVP	6
SVO2	59
CO	3.6
CI	1.8
SVR	1006
SpO2	93
SV	33
UO	40
CT	200

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

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

	0500
Art BP	91/38
MAP	58
HR	108
Temp	98
PAS/PAD	20/12
CVP	6
SVO2	59
CO	3.6
CI	1.8
SVR	1006
SpO2	93
SV	33
UO	40
CT	200

1. Treat hypovolemia

- ANSWER

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

	0500
Art BP	91/38
MAP	58
HR	108
Temp	98
PAS/PAD	20/12
CVP	6
SVO2	59
CO	3.6
CI	1.8
SVR	1006
SpO2	93
SV	33
UO	40
CT	200

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

	0500
Art BP	91/38
MAP	58
HR	108
Temp	99
PAS/PAD	20/12
CVP	6
SVO2	59
CO	3.6
CI	1.8
SVR	1006
SpO2	93
SV	33
UO	40
CT	200

	0700
Art BP	109/42
MAP	67
HR	101
Temp	99
PAS/PAD	43/16
CVP	8
SVO2	61
CO	4.2
CI	2.1
SVR	1180
SpO2	95
SV	43
UO	75
CT	300

Case 6: After two units of pRBCs. Did it help?
Are you happy? 1. Yes 2. No

	0500
Art BP	91/38
MAP	58
HR	108
Temp	99
PAS/PAD	20/12
CVP	6
SVO2	59
CO	3.6
CI	1.8
SVR	1006
SpO2	93
SV	33
UO	40
CT	200

	0700
Art BP	109/42
MAP	67
HR	101
Temp	99
PAS/PAD	43/16
CVP	8
SVO2	61
CO	4.2
CI	2.1
SVR	1180
SpO2	95
SV	43
UO	75
CT	300

Answer

- 2. No

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

	0700
Art BP	109/42
MAP	67
HR	101
Temp	99
PAS/PAD	43/16
CVP	8
SVO2	61
CO	4.2
CI	2.1
SVR	1180
SpO2	95
SV	43
UO	75
CT	300

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

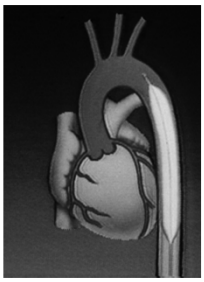
Don't PANIC!

GO WITH WHAT YOU KNOW!

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

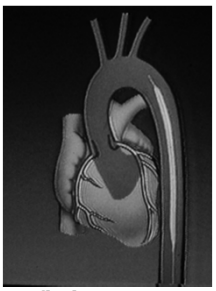
The Mechanics of Balloon Counterpulsation

IABP



Inflation

↑ **Coronary artery perfusion**



Deflation

Decrease afterload

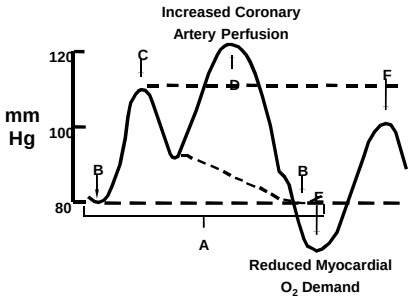
Contraindications

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

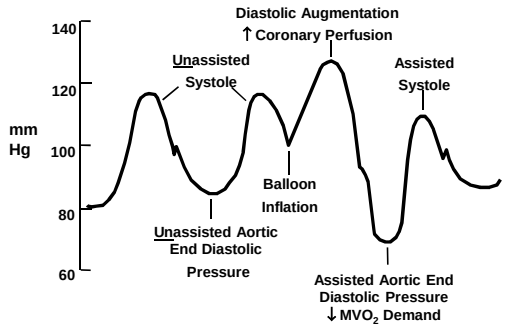
Potential Side Effects and Complications

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

1. Inflate at the beginning of diastole (dicrotic notch)
2. Deflate before ventricular systole



Arterial Waveform Variations During IABP Therapy

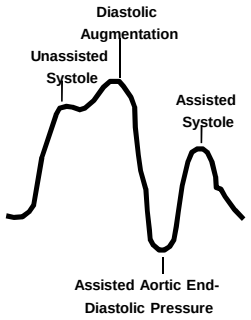


Timing Errors - Early Inflation

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 MVO_2 demand

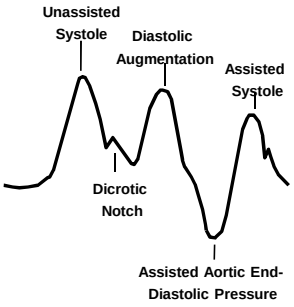


Timing Errors - Late Inflation

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

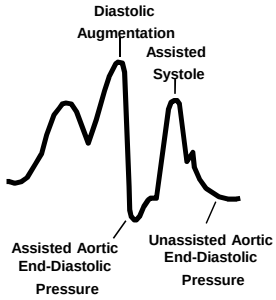


Timing Errors - Early Deflation

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 perfusion
- Potential for retrograde coronary and carotid blood flow
- Angina may occur as a result of retrograde coronary blood flow
- Sub-optimal afterload reduction
- Increased MVO_2 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 MVO_2 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