PESI Health Care Seminar
All Things Cardiac: Day 1

IMPACTING OUTCOMES
ONE PROFESSIONAL
AND ONE PATIENT
AT A TIME

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www.cardionursing.com

THOUGHT FOR THE DAY

“Professional nursing practice can only advance as much as individual nurses are aware that a knowledge gap exists in their practice, feel empowered to access further learning, and integrate evidence based competencies into their professional practice to provide safe, effective, efficient, patient centered, equitable care.”

www.tigersummit.com
Heart Sounds
An essential assessment tool!

Auscultatory Areas

- Aortic area (2NSB)
- Pulmonic area (2LSB)
- Erb's point (3LSB)
- Tricuspid area (5LSB)
- Mitral area (5LMCL)
The Cardiac Cycle

Ventricular Diastole

Ventricular Systole

Murmurs

- High blood flow through a normal or abnormal valve
- Forward flow through a narrowed or irregular orifice into a dilated chamber or vessel
- Backward or regurgitant flow through an incompetent valve
Murmur Fundamentals

• Stenotic Murmurs
  – Valve does not open appropriately
  – Heard during the part of the cardiac cycle when the valve is open

• Regurgitant Murmurs
  – Valve does not close appropriately
  – Heard during the part of the cardiac cycle when the valve is to be closed

Systolic Murmurs: What is Happening During Systole

• Tricuspid and Mitral Valve Closed
  – Tricuspid Regurgitation
  – Mitral Regurgitation

• Pulmonic and Aortic Valve Open
  – Pulmonic Stenosis
  – Aortic Stenosis
AORTIC Stenosis
Systolic Ejection Murmur

• May be present before any significant hemodynamic changes occur
• More severe AS ➔ longer murmur
• **Timing:** Midsystolic
• **Location:** Best heard over aortic area
• **Radiation:** Toward neck and shoulders
  – May radiate to apex
• **Configuration:** Crescendo-decrescendo
• **Pitch:** Medium to high
• **Quality:** Harsh

Mitral Regurgitation

• **Timing:** Holosystolic
• **Location:** Mitral area
• **Radiation:** To the left axilla
• **Configuration:** Plateau
• **Pitch:** High
• **Quality:** Blowing, harsh or musical
Which of these valvular disorders can develop acutely?

BETWEEN AORTIC STENOSIS AND MITRAL REGURGITATION:

Diastolic Murmurs: What is Happening During Diastole

- **Tricuspid and Mitral Valves Open**
  - Tricuspid Stenosis
  - Mitral Stenosis

- **Pulmonic and Aortic Valves Closed**
  - Pulmonic Regurgitation
  - Aortic Regurgitation
Diastolic Murmurs
Mitral Stenosis

- **Timing:**
  - Holodiastolic if severe MS
  - Mid to Late diastole if moderate MS
- **Location:** Apex
- **Configuration:** Crescendo
- **Pitch:** Low
- **Quality:** Rumbling
- Best heard with patient in left lateral position
- Increases with isometric exercise, and expiration

Aortic Regurgitation

- **Diastolic Murmur of AR**
  - Length of murmur correlates severity of AR
  - **Timing:** Early diastole
  - **Location:** left sternal boarder
    - 3rd, 4th ICS
  - **Radiation:** Towards apex
  - **Configuration:** Decrescendo
  - **Pitch:** High
  - **Quality:** Blowing
  - **Patient Position:** Sitting and leaning forward at end expiration
  - **Intensity:** Increases with increased peripheral vascular resistance: Squatting, exercising, hand gripping
Which of these valvular disorders can develop acutely?

BETWEEN MITRAL STENOSIS AND AORTIC REGURGITATION:

Blood Pressure Monitoring

• Systolic: Maximum pressure when blood is expelled from the left ventricle
  — Represents stroke volume

• Diastolic: Measures rate of flow of ejected blood and vessel elasticity
  — Represents state of arterioles

• Pulse Pressure: Difference between systolic and diastolic pressure

• Mean pressure (MAP): calculated; pressure that determines end organ perfusion
Blood Pressure Assessment

- Variation of up to 15mm Hg between arms is normal
- BP in legs - 10 mm Hg higher than arms

Etiology of Hypotension

\[
\text{Cardiac Output} \times \text{SVR} = \text{Blood Pressure}
\]
BP = CO x SVR

• Low BP could be due to:
  — Low CO
    • HR too slow or too fast
    • Preload too low or too high
    • Contractility low
  — Low SVR
    • Vasodilation due to sepsis, anaphylaxis, altered neurological function, drugs

Use of Pulse Pressure

• PP < 35 with tachycardia (C.O. problem)
  — Early sign of inadequate blood volume
  — Will also be seen with cardiogenic shock
  — Vasoconstriction is compensatory

• PP > 35 with tachycardia (SVR problem)
  — Early sign sepsis
  — Vasodilation is primary pathology
Comparison of 2 Hypotensive Patients

<table>
<thead>
<tr>
<th>Blood Pressure</th>
<th>History</th>
<th>Cause</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>88/70</td>
<td>History of CABG with LVEF 25% Presents with SOB Pulmonary edema on CXR</td>
<td>Decreased C.O.</td>
<td>Treatment may actually involve afterload reduction to increase cardiac output</td>
</tr>
<tr>
<td></td>
<td>No vasopressor!</td>
<td></td>
<td>Fluid and Vasopressors</td>
</tr>
<tr>
<td>82/30</td>
<td>1 week history of upper respiratory symptoms Presents with confusion CXR infiltrate LLL WBC 15,000, Fever</td>
<td>Decreased SVR</td>
<td>Treatment is focused on filling tank and restoring vascular tone</td>
</tr>
</tbody>
</table>

Pulsus Paradoxus

- Patient is placed in a semirecumbent position
- Respirations should be normal
- BP cuff inflated to at least 20 mm Hg above the systolic pressure
- Slowly deflated until the first Korotkoff sounds are heard only during expiration.
  - Pulsus paradoxus is present at this pressure reading, if the cuff is not further deflated and the first Korotkoff sound is not audible during inspiration.
- As the cuff is further deflated, the point at which the first Korotkoff sound is audible during both inspiration and expiration is recorded.

- If the difference between the first and second measurement is greater than 12 mm Hg, an abnormal pulsus paradoxus is present.

May be present in cardiac tamponade.

(Yarlagadda, Chakri, 2005 Cardiac Tamponade. Retrieved 3-22-06 from www.emedicine.com)
May be present in volume overload, tricuspid regurgitation, or obstructive shock (cardiac tamponade, tension pneumothorax, and large PE)

Measuring JV Pulsation / Pressure

- Raise HOB 30 – 45 degrees
  - Internal jugular preferred
  - May use external
- Use tangential light
- Use centimeter ruler
- Difficult to assess if HR>100

- Normal JV pulsation level is ≤ 3 cm above the sternal angle
### Jugular Vein

| No pulsations palpable.                  | Palpable pulsations.                      |
| Pulsations obliterated by pressure above the clavicle. | Pulsations not obliterated by pressure above the clavicle. |
| **Level of pulse wave decreased on inspiration; increased on expiration.** | No effects of respiration on pulse. |
| **Usually two pulsations per systole** (x and y descents). | One pulsation per systole. |
| Prominent descents. | Descents not prominent. |
| Pulsations sometimes more prominent with abdominal pressure. | No effect of abdominal pressure on pulsations. |

### Carotid Artery

- Lying flat to verify location of jugular
- Sitting or standing patient up to see top of column

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**Additional assessment tips:**
- Lying flat to verify location of jugular
- Sitting or standing patient up to see top of column
Cardiac Output = Heart Rate $\times$ Stroke Volume

- Preload
- Afterload
- Contractility

Same four components also determine myocardial oxygen demand

Etiology of Decreased Cardiac Output:

$$\text{Stroke Volume} \times \text{HR} = \text{Cardiac Output}$$
Determinants of Myocardial Performance

- **Stroke Volume**
  - Preload
  - Afterload
  - Contractility

- **Heart Rate**

  - **Synergy**
  - **Synchrony**

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**Preload**

- The ventricle is **preloaded** with blood at the end of diastole: * Creates stretch on myocardial muscles fibers*
- Determined by:
  - Volume of blood filling the ventricle at end of diastole
  - Greater the volume the greater the stretch (muscle fiber length)
  - Greater the stretch the greater the contraction
  - Greater the contraction the greater cardiac output

**TO A POINT**
Non-Invasive Preload Assessment

Right Ventricle
- JVD
- Hepatojugular reflux

Less Specific
- Peripheral edema
- Weight

Left Ventricle
- Lungs sounds
  - Clear lungs do not rule out volume overload
  - Role of lymph system
- CXR
  - Vascular congestion
  - Interstitial edema
  - Pulmonary edema
- Orthopnea / Bendopnea / PND
- S₃
- Hypoxemia
  - Diffusion abnormality

Less Specific
- Decrease in Blood Pressure and Urine Output
  - Because stroke volume falls

Factors Influencing Preload
- Body Position
- Venous Tone
- Intrathoracic pressure
- Intrapericardial pressure
- Dysrhythmias
- Atrial Kick
- LV Function

- Circulating blood volume
  - Hypervolemia
  - Hypovolemia
  - Third spacing

- Size of Container
  - Sepsis
  - Anaphylaxis
  - Venous vasodilators
Afterload

- After the ventricle is loaded:
- Pressure ventricle needs to overcome to eject blood volume
- Right ventricle: Pulmonary vascular resistance major component
- Left ventricle: Systemic vascular resistance major component
  - Other components
    - Valve compliance
    - Viscosity of blood
    - Aortic and arterial wall compliance

Afterload Assessment

- Left ventricle:
  - SVR
  - Pulse pressure and DBP
  - HTN = increased afterload
  - Hypotension does not = decreased afterload
    - Vasoconstriction is compensatory
- Right ventricle:
  - PVR
  - PA pressure from echo
  - Hypoxemia causes pulmonary vasoconstriction
  - Positive pressure ventilation and PEEP increase work load of left ventricle
More on Pulse Pressure

- Vascular tone is affected by:
  - Large vessel compliance
  - Peripheral vascular resistance (smaller vessels)

- Vessel resistance changes more quickly than large vessel compliance

- Increased resistance = increased DBP and narrow pulse pressure

Causes of Increased LV Afterload
- Arterial vasoconstrictors
- Hypertension
- Aortic valve stenosis
- Increased blood viscosity
- Hypothermia
- Compensatory vasoconstriction from hypotension in shock

Causes of Decreased LV Afterload
- Arterial vasodilators
- Hyperthermia
- Vasogenic shock states (sepsis and anaphylactic) where the body cannot compensate with vasoconstriction
- Aortic Regurgitation – hyperdynamic cardiac output therefore lowering systemic vascular resistance
Contractility

- **By definition:** Ability of myocardium to contract independent of preload or afterload
  - Velocity and extent of myocardial fiber shortening
  - Inotropic state

- Physiologically is related to degree of myocardial fiber stretch (preload) and wall tension (afterload).

- ↑ contractility
  - ↑ myocardial workload
  - ↑ myocardial oxygen consumption

Important Points about Contractility

- No accurate way to measure contractility

**Noninvasive Assessment: Ejection Fraction**

- Low cardiac output does not necessarily mean diminished contractility (i.e. hypovolemia)

- Correct preload and afterload problems first in a patient with a low ejection fraction.

- Increasing contractility with medications will also increase myocardial oxygen demand.
Factors Altering Contractility

- Decreased contractility
  - Excessive preload or afterload
  - Drugs – negative inotropes
  - Myocardial damage
  - Ischemia
  - Cardiomyopathy
  - Hypothyroidism
  - Changes in ionic environment: hypoxia, acidosis or electrolyte imbalance

- Increased contractility
  - Drugs
    - Positive inotropes
  - Hyperthyroidism
  - Adrenal Medulla Tumor

Heart Rate

- Mathematically heart rate increases cardiac output

- Physiological limit where increased heart rate will decrease cardiac output due to decreased filling time (decreased preload)
Backwards Failure: Pulmonary Congestion

Forwards Failure: Hypoperfusion

<table>
<thead>
<tr>
<th>Warm and Dry</th>
<th>Warm and Wet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal Perfusion</td>
<td>Normal Perfusion</td>
</tr>
<tr>
<td>No Congestion</td>
<td>Congestion</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cold and Dry</th>
<th>Cold and Wet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low Perfusion</td>
<td>Low Perfusion</td>
</tr>
<tr>
<td>No Congestion</td>
<td>Congestion</td>
</tr>
</tbody>
</table>
Treatment

Congestion with Adequate Perfusion
• Reduce Preload

Hypoperfusion with No Congestion
• Increase contractility
  – Must have adequate preload

Hypoperfusion with Congestion
• Reduce Afterload

Pharmacological Options for INCREASING Preload

| Volume expanders | • Isotonic crystalloids such as 0.9% saline or lactated ringsers
  • Colloids such as albumin
  • Blood and/or blood products |
|------------------|---------------------------------------------------------------------|
| Decrease dose or stop diuretics or drugs that cause venous vasodilatation. | • Decrease or stop medications such as: loop diuretics, intravenous nitroglycerin, neseritide, and morphine sulfate
  (Venous vasodilatation pools blood away from the heart and decreases preload – direct impact on right sided preload) |

Exercise also increases venous return to the heart.
## Pharmacological Options for DECREASING Preload

**Stop or decrease fluid**

<table>
<thead>
<tr>
<th>Diuretics</th>
<th>• A loop diuretic such as furosemide eliminates circulating volume</th>
</tr>
</thead>
<tbody>
<tr>
<td>Venous Vasodilators</td>
<td>• Intravenous nitroglycerin, neseritide, or morphine sulfate (Venous vasodilation pools blood away from the heart and decreases preload)</td>
</tr>
<tr>
<td>ACE Inhibitors or Angiotensin II Receptor Blockers</td>
<td>• Interrupt renin-angiotensin-aldosterone system. (RAAS). Aldosterone secretion is decreased and there is less sodium and water retention.</td>
</tr>
</tbody>
</table>
| Aldosterone antagonists | • Spironolactone or epleranone  
• Directly block aldosterone and there is decreased sodium and water retention. |

## Pharmacological Options for INCREASING Afterload

**Vasopressor** is the term given to medications used to increase afterload.

| Sympathomimetics stimulating the alpha receptors of the sympathetic nervous system | • Dopamine  
• Norepinephrine  
• Phenylephrine  
• Epinephrine |
|-----------------------------------------------------------------------------|--------------------------------------------------|
| Arginine Vasopressin | • Vasoconstrictive and antidiuretic effect  
• Restores catecholamine sensitivity |
**Pharmacological Options for DECREASING Afterload**

<table>
<thead>
<tr>
<th>All therapies involve arterial vasodilatation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Smooth muscle relaxants</strong></td>
</tr>
<tr>
<td>• Nipride</td>
</tr>
<tr>
<td>• Hydralazine</td>
</tr>
<tr>
<td><strong>Calcium channel blockers</strong></td>
</tr>
<tr>
<td>• Dihydropyridines (ending in “ine”) calcium channel blockers such as amlodipine</td>
</tr>
<tr>
<td><strong>Alpha₁ receptor blockers</strong></td>
</tr>
<tr>
<td>• Labetolol (combination alpha and beta blocker)</td>
</tr>
<tr>
<td>• Prazoxin, Terazosin</td>
</tr>
<tr>
<td><strong>Central anti-adrenergics</strong></td>
</tr>
<tr>
<td><strong>Peripheral anti-adrenergics</strong></td>
</tr>
<tr>
<td>Clonidine, Methyldopa</td>
</tr>
<tr>
<td>Risperine, Guanthidine</td>
</tr>
<tr>
<td><strong>ACE Inhibitors</strong></td>
</tr>
<tr>
<td><strong>Angiotensin II Receptor Blockers (ARBs)</strong></td>
</tr>
<tr>
<td>• Interrupt the RAAS and limit production of angiotensin II a potent arterial vasoconstrictor</td>
</tr>
<tr>
<td>• Medications ending in “pril”</td>
</tr>
<tr>
<td>• Directly block the effects angiotensin II</td>
</tr>
<tr>
<td>• Medications ending in “sartan”</td>
</tr>
<tr>
<td><strong>Phosodiesterase Inhibitors</strong></td>
</tr>
<tr>
<td><strong>(PDE Inhibitors)</strong></td>
</tr>
<tr>
<td>• Milrinone</td>
</tr>
<tr>
<td>• Is used as an intravenous inotrope but also has arterial vasodilator properties</td>
</tr>
</tbody>
</table>

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**Pharmacological Options for INCREASING Contractility**

**Inotrope** is the term used for medications used to increase contractility

| Sympathomimetics stimulating the β1 receptors of the sympathetic nervous system | Doebutamine: most commonly used because it is predominant beta one stimulator |
|---------------------------------------------------------------------------------| Other sympathomimetics may have inotropic properties even if not used primarily for an inotropic purpose |
| Phosodiesterase Inhibitors                                                      |
| **(PDE Inhibitors)**                                                            |
| • Milrinone                                                                     |
| • Is used as an intravenous inotrope but also has arterial vasodilator properties |
| Cardiac Glycoside                                                               |
| • Digoxin                                                                       |
| • Weak inotrope and is never used intravenously to support left ventricular dysfunction. Exerts weak inotropic properties when given orally. |
### Pharmacological Options for DECREASING Contractility

<table>
<thead>
<tr>
<th>Beta Blockers blocking the β₁ receptors of the sympathetic nervous system</th>
<th>Metoprolol</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Carvedilol</td>
</tr>
<tr>
<td></td>
<td>“lol” medications</td>
</tr>
<tr>
<td>Calcium Channel Blockers</td>
<td>Diltiazem</td>
</tr>
<tr>
<td></td>
<td>Verapamil</td>
</tr>
</tbody>
</table>

### Pharmacological Options for INCREASING Heart Rate

<table>
<thead>
<tr>
<th>Parasympatholytic (lyses the parasympathetic nervous system)</th>
<th>Atropine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sympathomimetics stimulating the β₁ receptors of the sympathetic nervous system</td>
<td>Epinephrine</td>
</tr>
<tr>
<td></td>
<td>Dopamine</td>
</tr>
</tbody>
</table>

Note: The non-pharmacological intervention of pacing the heart with either an external, temporary or permanent pacemaker is often the preferred method of increasing the heart rate to a set and controlled rate.
Pharmacological Options for DECREASING Heart Rate

1. Beta Blockers blocking the β₁ receptors of the sympathetic nervous system
   - "olol" medications
   - Class II antiarrhythmics

2. Calcium Channel Blockers
   - Diltiazem / Verapamil
   - Class IV antiarrhythmic

3. Cardiac Glycoside
   - Digoxin

4. Unclassified antiarrhythmic
   - Adenosine: Slows conduction through the AV node

5. Other antiarrhythmics
   - Class I and Class III antiarrhythmics
   - Used to establish and/or maintain a normal rhythm and therefore control heart rate

Let’s Clear Up Some Terminology

• **Vasopressors**
  – Term given to any medication in any class that is used to **increase left ventricular afterload** (systemic vascular resistance)

• **Inotropes**
  – Term given to any medication in any class that is used to **increase myocardial contractility**
    - Increase mortality
    - Used in shock / decompensation when other treatments fail
    - Used as bridge to transplant or palliation
Sympathetic Nervous System

- Fight or flight

**Alpha_1 Receptors**
- Vasoconstriction of vessels

**Beta_2 Receptors** (Heart)
- Increased heart rate
  - Chronotropic Response
- Increased conductivity
  - Dromotropic Response
- Increased contractility
  - Inotropic Response
- Increased automaticity

**Beta_2 Receptors** (Vesseles, Lungs)
- Bronchodilation
- Peripheral Vasodilatation

Sympathomimetics

Sympathetic

- Beta 1
- Beta 2

Parasympathetic

- Alpha 1
- Vagal Response
### Epinephrine

**Endogenous catecholamine**

| What receptors are stimulated: | β₁ and β₂  
| Alpha receptors |
|-------------------------------|----------------|
| What are the resultant actions: | Increase contractility (+inotrope) β₂  
|                                | Increased heart rate (+chronotrope) β₁  
|                                | Bronchodilation β₂  
|                                | Selective vasoconstriction (alpha) |
| When and why do we use:        | ACLS first line drug for cardiac standstill- 1 mg every 3 to 5 min  
|                                | Hypotension or profound bradycardia  
|                                | 0.1 to 0.5 mcg/kg/min (7 to 35 mcg/min if 70 kg)  
|                                | Anaphylaxis  
|                                | 0.3 mg SQ/IM - may repeat dose  
|                                | Bronchodilator  
|                                | 0.3 to 0.5 mg SQ q 20 min (inhalation and nebulization also) |
| What are special nursing considerations: | Onset instant; Peak 20 minutes, SQ onset 5 to 10 min, inhalation 1 min  
|                                | Increases lactate release  
|                                | Phentolamine for extravasation: 5 to 10 mg diluted in 10ml  
|                                | 0.9 NS, inject into area with fine needle  
|                                | Leave cannula and needle in place, aspirate not flush line, dry warm compress |

### Dobutamine

**Synthetic Compound**

| What receptors are stimulated: | Primarily β₁  
|                                | Some alpha₁ receptor stimulation  
|                                | Some β₂ stimulation  
|                                | Modest β₂ (more β₂ than alpha₁) |
| What are the resultant actions: | Increase contractility (+ inotrope) (β₁)  
|                                | Increase AV node conduction  
|                                | Modest vasodilation |
| When and why do we use:        | **Used as an inotrope** (resultant preload reduction) with modest afterload reduction (ACC / AHA Guidelines for Heart Failure*) |
| What are special nursing considerations: | VT, atrial tachyarrhythmias  
|                                | Onset 1 to 2 minutes; Peak 10 minutes  
|                                | Half-life 2 minutes  
|                                | Note: Blood pressure response is variable; β₂ causes vasodilatation; β₁ increases cardiac output and may increase BP |
Continuous intravenous dobutamine is associated with an increased risk of death in patients with advanced heart failure: Insights from the Flolan International Randomized Survival Trial (FIRST)

Intermittent dobutamine treatment in patients with chronic refractory congestive heart failure: a randomized, double-blind, placebo-controlled study. (PMID:9663183)

Intermittent dobutamine infusions in patients with refractory CHF have no effect on the need for hospitalization or on survival.
### Dopamine

**What receptors are stimulated:**
- **Dopaminergic** at low doses (0.5-2.0 mcg/kg/min)
- β₁ also at moderate doses (2.0-10.0 mcg/kg/min)
- Pure alpha stimulation at high doses > 10 mcg/kg/min

**What are the resultant actions:**
- Increase GFR at low doses
- Increase contractility at moderate doses (greater effects on contractility than heart rate)
- Vasoconstriction (alpha) at high doses

**When and why do we use:**
- Refractory hypotension / shock
  - *Not indicated for routine treatment or prevention of acute renal failure*

**What are special nursing considerations:**
- Onset: 1-2 minutes; Peak 10 minutes
- Maximal effects @20/mcg/kg/min
- Large IV line or central line
- Phentolamine for extravasation: 5 to 10 mg diluted in 10ml 0.9 NS, inject into area with fine needle
- Leave cannula and needle in place, aspirate not flush line, dry warm compress

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### Norepinephrine

**What receptors are stimulated:**
- Primarily alpha stimulation
  - Some β₁
    - (In lower doses β₁ can be more dominant)

**What are the resultant actions:**
- Potent vasoconstrictor (increased afterload)
  - Some increased contractility (+inotrope)

**When and why do we use:**
- Refractory hypotension / shock
  - (used as a vasopressor but will have inotropic properties)

**What are special nursing considerations:**
- Onset: rapid; very short half-life
  - Duration 1-2 minutes (BP checks q2 minutes while titrating)
- Large IV line or central line
- Phentolamine for extravasation: 5 to 10 mg diluted in 10ml 0.9 NS, inject into area with fine needle
- Leave cannula and needle in place, aspirate not flush line, dry warm compress
## Phenylephrine

| What receptors are stimulated: | Direct effect: Dominant alpha stimulation  
No substantial β₁ effect at therapeutic doses  
Indirect effect: Releases norepinephrine |
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>What are the resultant actions:</td>
<td>Vasoconstriction (increased afterload)</td>
</tr>
<tr>
<td>When and why do we use:</td>
<td>As a vasopressor for Unresponsive hypotension</td>
</tr>
</tbody>
</table>
| What are special nursing considerations: | Pressor effect occurs almost immediately  
Persists for 10 to 15 minutes |

### Pearls for Practice

- Titrate up based on onset of action & peak action
- Wean based on duration of action / half life
- Consider systolic, diastolic, pulse pressure and mean
- Vasoconstriction with a reduced ejection fraction increases work load of failing left ventricle!
Comparison of Dopamine to Norepinephrine in Shock

- Backer et al.
- Multi Center Randomized Controlled Trial
- New England Journal of Medicine

- There were no significant differences between the groups in the rate of death at 28 days or in the rates of death in the ICU, in the hospital, at 6 months, or at 12 months

- More patients with arrhythmia in the dopamine group

- Rate of death was higher in predefined subgroup analysis for patients with cardiogenic shock treated with dopamine.

Arginine Vasopressin:
Non Sympathomimetic Vasopressor

- Vasoconstrictive effects
  - Allowing for regional vasodilation

- Antidiuretic effects

- Restoration of catecholamine sensitivity

- Low dose exogenous
  - 0.04 units / min

- Use in refractory shock
Other Considerations in Refractory Shock

• Also consider methylene blue
  – Off label use
  – 1.5 to 2.0 mg/kg over 20 to 60 minutes x 1
  – Effects are seen 1 to 2 hours after administration
  – Direct inhibitory effect on endothelial nitric oxide synthase by oxygenation of enzyme bound ferrous iron
  – Also reduces vasorelaxation by blocking formation of cGMP through binding iron in the heme complex

• Also consider adrenal insufficiency as cause
  – Hydrocortisone
  – Off label use
  – 100 mg initial IV bolus followed by additional dosing over 24 hours – then taper

Milrinone: Phosphodiesterase Inhibitors

• Creates + inotropic effect by increasing availability of calcium
  • Inhibits the degradation of cyclic AMP which is indirectly responsible for increasing the influx of calcium through the calcium channel

• Smooth muscle relaxant (venous and arterial vasodilator)

• Indications:
  – Refractory heart failure (can be used in combination with dobutamine)
  – Left ventricular failure in MI
  – Patients waiting transplant

• Side Effects:
  – Ventricular arrhythmias
  – Atrial tachyarrhythmias

• Nursing Considerations:
  – Onset IV: Immediate
  – Peak: 10 minutes
Milrinone

Used as an Inotrope
BUT.....

Preload Reduction

Also has......

Afterload Reduction

OPTIME Trial

- Milrinone approved by FDA based on hemodynamic data
- Future trials need to address outcome data
- OPTIME
  - Prospective trial, randomized, placebo controlled
  - 951 patients
  - Patients had indication for but not all required inotrope for end organ perfusion.
  - Results: No difference in LOS, No difference in subjective improvement
  - Treatment failures more common in milrinone group due to hypotension, more atrial fibrillation in milrinone
  - Not powered for mortality differences
- Conclusion: Hemodynamic improvement does not translate into clinical improvement
A Closer Look at Venous Versus Arterial Vasodilators

Venous Vasodilators

Decrease Preload

Arterial Vasodilators

Decrease Afterload

Nesiritide (Natrecor)

- Recombinant form of human B type natriuretic peptide (BNP)
  - BNP is a naturally occurring cardiac neurohormone secreted by the heart in the body’s response to heart failure
  - BNP allows the heart to participate in the regulation of vascular tone and extracellular volume status

- The BNP system and the renin-angiotensin system counteract each other in heart failure

Onset: 15 minutes
Peak: Within an hour
Longer ½ life compared to NTG / Nitroprusside
Nesiritide (Natrecor)

- Balanced arterial and venous vasodilatation
  - Causes rapid reduction in right and left sided ventricular filling pressures (preload reduction)
  - Reduces afterload

- Indicated for acutely decompensated heart failure patients who have dyspnea at rest
  - Patient must have systolic BP > 90 mmHg
  - PAOP should be estimated to be > 20 mmHg

Nesiritide (Natrecor)

- Prime IV tubing with 5 ml of solution – prior to bolus or infusion
- Given by IV bolus and maintenance infusion
  - Bolus 2mcg/kg
  - Infusion 0.01mcg/kg/min - usually 24-48 hours

- Bolus to be taken from reconstituted IV bag and not from vial
- Caution with higher doses

Monitor BP, hemodynamic assessment, urine output, and renal function closely during administration.
Intravenous Nesiritide, a Natriuretic Peptide, in the Treatment of Decompensated Congestive Heart Failure (2000)

- Nesiritide lowered PAOP over 6 hours by 6 to 9.6 mmHg (P < 0.001)
- Improvement in global clinical status (P < 0.001)
- Improvement in dyspnea (P < 0.001)
- Improvement in fatigue (P < 0.001)

Most common side effect: Dose related hypotension, usually asymptomatic

Next: Safety Concerns

  - In the 3 trials, 485 patients were randomized to nesiritide and 377 to control therapy. Death within 30 days tended to occur more often among patients randomized to nesiritide therapy (35 [7.2%] of 485 vs 15 [4.0%] of 377 patients; risk ratio from meta-analysis.
  - P value did not achieve .05 or less.
Nesiritide in Cardiovascular Surgery

- **Effects of Perioperative Nesiritide in Patients With Left Ventricular Dysfunction Undergoing Cardiac Surgery: The NAPA Trial**

- **Conclusions**: Nesiritide in the setting of CABG with CPB is associated with improved postoperative renal function and possibly enhanced survival.

Randomized Trial

- **Effect of Nesiritide in Patients with Acute Decompensated Heart Failure (ASCEND-HF)**
  - O’Connor et al.
  - July 7 2011

  - 7141 patients

  - Nesiritide was not associated with an increase or a decrease in the rate of death and rehospitalization.
  - It was not associated with a worsening of renal function, but it was associated with an increase in rates of hypotension.

  - Neseritide cannot be recommended for routine use.
2013: ROSE Trial

- Low dose dopamine and low dose nesiritide against placebo for enhance decongestion
  - Neither enhanced decongestion or improved renal function

- Difference in outcome between preserved and reduced ejection fraction (Sub Study analysis 2014)
  - Low dose dopamine and low dose nesiritide impacted urine output in patients with HFrEF

Nitroglycerin

- Mixed venous and arterial vasodilator
  - Dosage < 1mcg/kg/min = venous vasodilator
  - Dosage > 1mcg/kg/min = arterial and venous vasodilator

- Primarily used as venodilator to quickly reduce preload
  - Ideal in HF accompanied by ischemia, hypertension, or mitral valve regurgitation
  - Sublingual tablets provide high enough dosage to dilate arteries and veins
Nitroglycerin

• Side Effects:
  – H/A,
  – Hypotension,
  – Flushing
  – Tachyphylaxis
    • 20% resistance even at high doses
• Caution in patients with tachycardia or bradycardia – can cause paradoxical bradycardia

• Nursing Considerations:
  – Contraindicated with Sildenafil like drugs
  – Caution (all venous vasodilators) with:
    • Hypertrophic cardiomyopathy, aortic stenosis, mitral stenosis, any severe diastolic dysfunction, right ventricular MI
  – Treat H/A with pain meds and decrease dose
  – Onset IV: 1-2 minutes

Caution with Vasodilators
Nitroprusside

- Mixed venous and arterial dilator (primarily arterial)
- Decreases BP, SVR, PVR, PAOP, RAP
- Uses:
  - Hypertensive crisis
  - CHF
  - Acute Mitral Regurgitation
  - Other Indications for Afterload Reduction
- Side Effects:
  - Hypotension
  - Thiocyanate toxicity: tinnitus, blurred vision, delirium, seizures, muscle twitching, absent reflexes, dilated pupils [several days – high doses]
- Nursing Considerations:
  - Onset: 1-2 minutes
  - Duration: 1-10 minutes
  - Monitor BP carefully - arterial line encouraged

Drugs Used to Alter Clotting in ACS

- Fibrinolytics
  - STEMI
  - tPA
    - Alteplase
    - Retaplace
    - Tenecteplase
  - Streptokinase (no longer used)
- Antiplatelets
  - STEMI / NonSTEMI / UA
  - GP IIb/ IIIa Inhibitors
    - Eptifibitide (Integrelin)
    - Tirofiban (Aggrastat)
    - Abciximab (Repro)
  - ADP Receptor Blockers
    - Clopidogrel
    - Prasugrel
    - Ticagrelor
  - Thromboxane A2 Inhibitor
    - ASA

- Anticoagulants
  - STEMI / NonSTEMI / UA
  - Unfractionated Heparin
  - Low Molecular Weight Heparin
  - Direct Thrombin Inhibitors
  - Factor Xa Inhibitors
Clot Formation: Clotting Cascade

**Intrinsic Pathway**
- Initiated by vascular injury and direct exposure to collagen
  - Site of activated platelet
  - Site of endothelial damage
  - Subendothelial layer where collagen is exposed
- From initiation to a clot is 2-6 minutes
- Measured by APTT

**Extrinsic Pathway**
- Initiated by endothelial release (secondary to tissue injury) of thromboplastin tissue factor
- From initiation to clot is 15 to 20 seconds
- Measured by Protime

---

A clot can be produced by activation of either the intrinsic or extrinsic pathway.

---

The Clotting Cascade

**Intrinsic Pathway**
- XII → XIIa
- XI → Xla
- IX → IXa
- X → Xa
- II Prothrombin → IIa Thrombin
- Fibrinogen → Fibrin → Fibrin Stable Clot

**Extrinsic Pathway**
- VII → VIIa
- Activated platelets
  - Endo /tissue damage
- Phospholipid
  - Calcium
  - Factor VIIIa
- Thromboplastin Tissue Factor III
  - Activated platelets
  - Endo /tissue damage

**Common Pathway**
The Clotting Cascade

Intrinsic Pathway

XII → XIIa

XI → Xa

Extrinsic Pathway

TF III → VII

VIIa

Common Pathway

II Prothrombin → X

Xa

IIa Thrombin

Fibrinogen

Fibrin

Fibrin Stable Clot

Anticoagulants

- Unfractionated Heparin
  - Heparin by Weight
  - STEMI, NonSTEMI, UA
  - Mortality benefit
- Low Molecular Weight Heparin
  - STEMI, NonSTEMI, UA
- Direct Thrombin Inhibitors
  - If history of HIT, PCI NonSTEMI
- Factor Xa Inhibitors
  - Not in PCI
- Warfarin (Vitamin K antagonist)
- Dabigatran (Direct thrombin inhibitor)
- Rivaroxaban (Factor Xa inhibitor)
- Apixaban (Factor Xa inhibitor)
- Edoxaban (Factor Xa inhibitor)
Unfractionated Heparin

- Works in the intrinsic and common pathway
- Antithrombin activator that inhibits factors Xa and IIa (thrombin)
  - Antithrombin III lyses factor Xa and thrombin and inhibits clotting
  - When heparin binds with antithrombin III the inhibition is increased 1000 times
- Anticoagulation is almost instant
- \(\frac{1}{2}\) life relatively short
- Antidote: Protamine 1 mg per 100 units
- Concern that unfractionated heparin results in platelet activation - although thrombin is a strong platelet activator and heparin is an antithrombin drug
More About Heparin

- Baseline aPTT, PT/INR, platelets and CBC
- Different dose and aPTT target for ACS versus venous thrombotic event
- Increased bleeding can occur with renal failure
  - Heparin has dual clearance mechanism

- aPTT (activated partial thromboplastin time) is used to monitor effectiveness and safety
  - Goal is aPTT 1.5-2Xs the control
  - Weight based heparin dosing reaches goal 90% of time compared to 77% with standard therapy
- OR – Anti factor Xa levels
  - 0.3-0.7 IU/ml

Complications of Heparin

- Bleeding
- Mild thrombocytopenia – HIT 1
  - Mild thrombocytopenia occurs in 10-20% of patients
- Severe thrombocytopenia – HIT 2
  - Occurs in 1-2% of patients
  - Heparin Induced Thrombocytopenia (HIT)
  - Platelet aggregation resulting in venous or arterial thrombosis (HITT – Thrombocytopenia with thrombosis)
  - Determining patients at risk is unpredictable
  - Generally occurs 5 to 10 days after initiation of heparin
    - Could be sooner if recent exposure to heparin
  - DC heparin if platelets fall below 100,000 (or > 50% reduction from baseline)
  - Severe thrombocytopenia is due to an immune response
More on Heparin Induced Thrombocytopenia

- Immune system forms antibodies against heparin when bound to protein platelet factor 4 [PF4] – antibodies bind to PF4-heparin complexes and induce platelet activation
  - Immunoassay identify antibodies against PF4
    - Detected in ELISA testing
  - Function Assay
    - Heparin-induced platelet aggregation assay (HIPA)
    - Platelet activation test
- HIT antibodies are usually IgG class
  - Take 5 days to form
  - IgG antibodies associated with platelet activation and increased thrombin generation
  - Antibodies not necessarily associated with thrombotic risk
  - Can disappear 3 months after exposure
- Antibodies bind to platelets and trigger the development of thrombosis.

Treatment of HIT

1. Discontinue and avoid all heparin.
2. Give a non-heparin alternative anticoagulant: Direct thrombin inhibitors.
3. Postpone warfarin pending substantial platelet count recovery (give vitamin K if warfarin has already been started). Warfarin is associated with protein C deficiency and increased risk for microthrombosis
4. Avoid platelet transfusions – leads to platelet activation.
5. Test for HIT antibodies (ELISA and washed assay)
Low Molecular Weight Heparin

- Enoxaparin, dalteparin, tinzaparin, and nadoparin
- Smaller in size
  - Antithrombin by inhibiting factor Xa
  - Causes less inactivation of thrombin and less bleeding than standard heparin
  - More predictable anticoagulant response
  - No need to monitor APTT
    - Anti Xa levels can be drawn 4 hours after SQ dose
  - Lower incidence of heparin induced thrombocytopenia
  - Less platelet activation concern than with UFH

Low Molecular Weight Heparin

- Can be self administered with Sub – Q administration
  - Full length of 27 gauge ½ needle (prepackaged) should be injected
  - Skin fold held until needle withdrawn
  - Use anterolateral or posterolateral walls of abdomen
  - Rotate sites frequently
  - Do not massage site
- ½ life 4-6 hours
- Protamine reverses 60% of drug effect
- Renal failure results in increased risk of bleeding because LMWH is renally cleared
  - Special dosing for chronic renal insufficiency with enoxaparin
Enoxaparin Dosing

- Prevention of DVT
  - 40 mg SC daily in most situations
  - 30 mg SC daily for renal adjustment (CR Clearance < 30 ml/min)

- NSTE-ACS (or as adjunct in STEMI)
  - 1 mg/kg SC q12 hours
  - 1mg/kg SC daily if CR Clearance < 30 ml/min
  - IV loading dose of 30 mg – in select patients
  - Continued for duration of stay or until PCI

- Embolism with Atrial Fib
  - 1 mg/kg SC q12 hours

- Venous thrombosis / DVT
  - 1mg/kg SC q12 or 1.5 mg/kg daily depending of specific circumstances

Direct Thrombin Inhibitor

- Ability to inactivate fibrin bound thrombin
- Less binding to plasma proteins, therefore more reliable anticoagulation effect
- Indications
  - HIT /HITT
  - Approved in NSTE-ACS (Use only in patients with early invasive strategy) / PCI
    - Non inferior to heparin with a GPIIb/IIIa with less bleeding
    - Monitor in the cath lab with ACT
- Lipirudin / desirudin (hirudin)
- Argatroban
- Bivalirudin*
  - 0.10 mg/kg loading
  - 0.25 mg/kg per hour
  - Until diagnostic angiography or PCI is performed
**Synthetic Factor Xa Inhibitor**

**Fondaparinux (Arixtra)**

- Neutralizes Factor Xa and interrupts the clotting cascade
- Does not inhibit thrombin
- No reported HIT / HITT
- Indications
  - Venous thromboembolism and PE
  - DVT prophylaxis
  - ACS
- Contraindicated in severe renal dysfunction
- No laboratory monitoring is needed – PT/aPTT not sensitive
- No antidote (Recombinant factor VIIa can help reverse anticoagulation effect)

**Synthetic Factor Xa Inhibitor**

- DVT Prophylaxis: 2.5mg SC once daily in adults > 50 kg
- ACS
  - 2.5 mg SC daily for duration of hospital stay up to 8 days or until time of revascularization
  - If STEMI an initial dose 2.5mg should be given IV before starting daily SC
  - *Cannot be used as sole anticoagulant during PCI – add DTI or UH*
- Acute DVT or PE – weight based for between 5mg and 10mg SC daily
  - Can use as a bridge for 5-7 days if warfarin is long term anticoagulation choice
  - Start warfarin on day 1 or 2 but continue Fondaparinux for at least 24 hours after therapeutic INR is achieved
Figure 1. Master Treatment Algorithm for Duration of P2Y₁₂ Inhibitor Therapy in Patients With CAD Treated With DAPT

2016 ACC/AHA Guideline Focused Update on Duration of Dual Antiplatelet Therapy in Patients With Coronary Artery Disease.
### Factors Used to Calculate a “DAPT Score”

<table>
<thead>
<tr>
<th>Variable</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age ≥75 years</td>
<td>-2</td>
</tr>
<tr>
<td>Age 65 to &lt;75 years</td>
<td>-1</td>
</tr>
<tr>
<td>Age &lt;65 years</td>
<td>0</td>
</tr>
<tr>
<td>Current cigarette smoker</td>
<td>1</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1</td>
</tr>
<tr>
<td>MI at presentation</td>
<td>1</td>
</tr>
<tr>
<td>Prior PCI or prior MI</td>
<td>1</td>
</tr>
<tr>
<td>Stent diameter &lt;3 mm</td>
<td>1</td>
</tr>
<tr>
<td>Paclitaxel-eluting stent</td>
<td>1</td>
</tr>
<tr>
<td>CHF or LVEF &lt;30%</td>
<td>2</td>
</tr>
<tr>
<td>Saphenous vein graft PCI</td>
<td>2</td>
</tr>
</tbody>
</table>

A score of ≥2 is associated with a favorable benefit/risk ratio for prolonged DAPT while a score of <2 is associated with an unfavorable benefit/risk ratio.


### Aspirin

- Diminishes platelet reactivity
- Produces rapid clinical antithrombotic effect caused by immediate and near-total inhibition of thromboxane A2 production (released with vascular injury).
  - Thromboxane A2 is a potent vasoconstrictor
- Inhibits COX1 and COX2
  - NSAIDS reversibly bind to COX1 preventing inhibition by ASA and may cause prothrombotic events
- Inhibits the endothelium’s production of prostaglandin I2 which decreases platelet aggregation and induces vasodilation.
  - Prostaglandin I2 is also involved in inflammation.
- Reduces mortality
- Increase myocardial oxygen supply

**Use in ACS**
- Administered as soon as possible after presentation
- Initial dose: 162 mg to 325 mg chewed (non-enteric coated)
- Long Term: 81 mg daily
- If ASA intolerant load with clopidogrel and then daily dose
P2Y\textsubscript{12} Receptor Inhibitors / ADP Receptor Blockers

- **Thienopyridines:**
  - *Irreversibly* inhibits P2Y\textsubscript{12} receptor
    - Clopidogrel
    - Prasugrel

- **Non thienopyridine:**
  - *Reversibly* binds to P2Y\textsubscript{12} receptor
    - Ticagrelor

- Adenosine Diphosphate (ADP) - Stored in platelets and released upon platelet activation

- ADP interacts with P2Y\textsubscript{12} chemoreceptors to enhance adhesiveness and aggregation of platelets through the activation of the GP IIb/IIIa pathway

### Comparison of P2Y\textsubscript{12} Inhibitors

<table>
<thead>
<tr>
<th>Clopidogrel (Thienopyrodine)</th>
<th>Prasugrel (Thienopyrodine)</th>
<th>Ticagrelor (Non-Thienopyrodine)</th>
</tr>
</thead>
<tbody>
<tr>
<td>600 mg initial dose 75 mg daily</td>
<td>60 mg initial dose 10mg daily</td>
<td>180mg initial dose 90mg twice daily</td>
</tr>
</tbody>
</table>

- Alternative to ASA therapy in those who cannot take ASA
- Option in NSTE-ACS as the second antiplatelet agent; Recommended agent in elective PCI
- Approved 2009
- Triton TIMI 38 (compared to clopidogrel)
  - All patients to cath lab
  - Significant reduction in risk but also **significant increase in major bleeding**
- Approved 2011
- PLATO trial
  - Better anti-ischemic effect compared to clopidogrel
  - No significant increase in major bleeding

- **Issue of Non Responders**
- Prodrug: 2 step process
- *CYP2C19* isoenzyme responsible for almost half of the first step formation
- 3 major genetic polymorphisms are associated with loss of function
- Potential concern with PPIs: pantoprazole has less *CYP2C19* inhibition
- When tested in patients being managed medically (Trilogy) – no significant difference in outcomes compared to clopidogrel
  - Used only in patients having PCI
  - Cannot use: (bleeding risk)
    - ≥ 75 years old
    - <60 KG
    - Previous CVA / TIA
- Prodrug but not as dependent on *CYP2C19* isoenzyme
- **Not a pro drug**
- Higher recommendation for ticagrelor in NSTE over clopidogrel in either ischemia guided or early invasive option
- BID dosing concern for adherence
- Cannot take with more than 100 mg of ASA
- Hold 5 days for major surgery despite shorter ½ life
Guideline Directed Medical Therapy for ACS / CAD

**Mortality Reducing Agents**
- Dual Antiplatelet Therapy
  - ASA
  - Clopidogrel/Prasugrel/Ticagrelor
- Beta Blocker
- ACE Inhibitor
  - Based on additional criteria
- Eplerenone
  - Based on additional criteria
- Statin
  - Regardless of baseline LDL-C

**Additional Medications to Control Angina in Medical Management**
- Calcium channel blockers
- Long acting nitrate
- Ranolazine (Ranexa)
- SL Nitroglycerin

Guideline Directed Medical Therapy for Heart Failure with Reduced LVEF

**Mortality Reducing Agents**
- Evidence Based Beta Blockers
- ACE Inhibitor / ARBs / ARNIs
- Aldosterone antagonists
- Hydralazine and isosorbide dinitrate

**Additional Medications for Symptom Improvement / Hospitalization Reduction**
- Loop diuretics
- Thiazide diuretics
- Digoxin
- Ivabradine
Beta Blockers in ACS

• Immediate as well as long term mortality benefit
• Immediate beta-blocker therapy
  – Reduces the magnitude of infarction and incidence of associated complications
    • Decreases myocardial oxygen demand
  – Reduces rate of reinfarction
  – Reduces frequency of life-threatening ventricular tachyarrhythmias.
• Long term benefit post ACS
  – Decreases myocardial oxygen demand
  • HR Benefit
  – Enhances overall well being
  – Slows disease progression
  – Inhibits ventricular remodeling and apoptosis
  – Inhibits adverse effects of SNS
  – Reduces mortality and repeat hospitalizations
  – ? length of benefit

Beta Blockers at Presentation

• DO NOT administer in acute presentation IF:
  – STEMI precipitated by cocaine
    • Risk of exacerbating coronary spasm
  – Heart blocks
    • 1st degree AV block with PR ≥ 0.24 sec
    • 2nd or 3rd degree AV block
  – Heart rate < 60 BPM
  – SBP < 100 mm Hg
  – Moderate LV failure is present (signs of HF or shock)
  – Active asthma or reactive airway disease
A Closer Look at Beta Blockers

Decreases Myocardial Oxygen Demand

- Decrease HR
- Decrease Contractility

\[ \text{Blood pressure} = \text{CO} \times \text{SVR} \]

Beta Blockers

- **Nonselective**: Block both Beta_1 and Beta_2
  - Propranolol (Inderal)
  - Timolol (Blocadren)
  - Nandolol (Corgard)
  - Sotolol (Betapace)
  - Labetolol (Normodyne, Trandate) (also alpha blockade)
  - Carvedilol (Coreg) (also alpha blockade)

- **Cardio selective**: Block Beta 1
  - Acebutolol (Sectral)
  - Metoprolol tartrate (Lopressor)
  - Metoprolol succinate (Toprol XL)
  - Atenolol (Tenormin)
  - Esmolol (Breviblock)
  - Bisoprolol (Z Beta)
  - Nebivolol (Bystol) (also nitric oxide vasodilatory properties)
Medication Pearls

• Start beta blocker prior ACE-inhibitor
  – Beta blockers most important medication to reduce mortality early
  – However, cannot be given in patients at high risk for shock

• Do not start beta blocker and ACE-I at the same time

• Hold ACE inhibitors for BP < 100 mm Hg systolic or < 30 mm Hg below baseline.
  – Ideally ACE-I should be initiated within 24 hours

Beta Blockers
Recommended by Disease State

Post MI
• Atenolol
• Carvedilol ★
• Metoprolol ★
• Propanolol
• Timololol

Heart Failure
• Bisoprolol
• Carvedilol ★
• Metoprolol
  Succinate (XL) ★

Use GDMT for Heart Failure in all patients with ACS and LVEF < 40%.
Evidence Based Beta Blocker

• Cannot assume class effect
  • Bisoprolol – β1
    – CIBIS III randomized trial – 2005 (enalapril)
  • Metoprolol succinate - β1
    – MERIT-HF randomized trial – 1999 (placebo)
  • Carvedilol - β1, β2, α1
    – CAPRICORN randomized trial – 2001 (placebo)
    – COMET randomized trial – 2003 (metoprolol tartrate)

Point 1: Why do we use them when they decrease contractility?
  – Inhibits adverse effects of SNS
    • Decrease myocardial oxygen consumption
      – Decreases HR
      – Decreases contractility (however, benefit outweighs)
    – Inhibits ventricular remodeling and apoptosis
    – Slows disease progression
    – Can improve LVEF
    – Decrease mortality/hospitalization

When to initiate?
  – Do not initiate in an acutely decompensated patient
    • Remember you are giving a negative inotrope
  – Can be initiated in hospital for HF admission if inotropic therapy not required
  – If decompensation occurs on a beta blocker it is generally not stopped unless inotrope is needed. Dose may need to be decreased.
Beta Blocker Considerations

- Even better in combination with ACE Inhibitor
  - Started after initiation of ACE-I but before getting to target dose of ACE-I
- Must be used with diuretic if any recent or current fluid retention
- Start very low doses with gradual up-titration
- Titration to target dose essential
  - Nursing Practice Consideration: Educate patients regarding initial expectation of fatigue.
- Pearl: If hypotension – consider spacing of medications or decrease in diuretic dose
- Pearl: Fatigue may be multifactorial – address over diuresis, sleep apnea and screen for depression

Renin-Angiotensin Aldosterone System (RAAS)

↓ Renal Blood Flow / Perfusion

Renin Release → Angiotensinogen → Angiotensin I → Angiotensin II → Angiotensin Converting Enzyme → Aldosterone Release Na⁺ and H₂O Retention → ↑ BP → ↑ Renal Perfusion

Angiotensin Receptor Blockers

Vasoconstriction

ACE Inhibitors

Aldosterone Antagonists
A Closer Look at ACE Inhibitors and Angiotensin II Receptor Blockers

• Angiotensin-converting enzyme inhibitors ("pril" medications)
  – Captopril, Enalapril, Lisinopril, Quinapril, Ramipril, Benazepril, Fosinopril

• Angiotensin II Receptor Blockers ("sartan" medications)
  – Losartan, Irbesartan, Candesartan, Telmisartan, Valsartan, Eprosartan

ACE Inhibitors impact afterload and preload because they **block the vasoconstrictive effects of angiotensin II by preventing its formation**

  – Very important in reducing workload of left ventricle in systolic dysfunction
  – Decrease systemic vascular resistance without reflex stimulation of heart rate and contractility

ACE Inhibitors have additional benefit in preload reduction by blocking the effects of aldosterone release
A Closer Look at ACE Inhibitors

• Overall cardioprotective, vasculoprotective effect, and renal protective
  – Prevents ventricular remodeling
  – Reduce mortality in patients with systolic heart failure
  – Reduction of left ventricular mass in LV hypertrophy
  – Slows progression of both renal disease in diabetes and hypertensive nephrosclerosis

Cough in ACE-I

• Increases bradykinin release and can produce cough
  – Release of bradykinin causes constriction of non-vascular smooth muscle in bronchus
  – Cough is side effect in 10-20% of patients
• Need to assure cough is not sign of worsening heart failure
• Patient should be changed to an angiotensin receptor blocker (ARB) if unable to tolerate cough

Other contraindications to ACE Inhibitors are also contraindications to ARBs.
ACE Inhibitor and Angioedema

- Secondary to excessive accumulation of bradykinin
- Occurs in 0.7% of treated patients
- Likely genetic – non histamine angioedema
- Usually soon after administration but could occur after years of use
- African Americans have a 4-5x greater risk
- Women have a 2x higher risk than men
- Class effect reaction:
  - Absolute contraindication to further ACE I use

Bradykinin Facts:
- 9-Amino Acid Peptic Chain
- Causes vasodilation
- Causes naturesis
- Broken down by angiotensin converting enzyme
- Therefore: ACE I ↑ release of bradykinin

Treatment:
- Stop ACE I
- Antihistamines / corticosteroids not effective
- Epinephrine only if there is airway compromise
- FFP 2-4 units – suppresses bradykinin inhibits edema progression
- Ecallantide - suppresses bradykinin generation
- Icatibant - bradykinin B2 receptor antagonist

ACE Inhibitors and Renal Function:
Sorting Out the Confusion

- Renal protective in chronic kidney disease
- However, can cause acute kidney injury (AKI) in patient’s at risk (i.e. low stroke volume) due to preventing the compensatory mechanism of efferent vasoconstriction
  - When there is decreased blood flow into the glomerulus via the afferent arterioles, the efferent arterioles constrict to raise glomerular filtration pressure on the back end
  - ACE-I prevent efferent vasoconstriction
- Creatinine can be allowed to be 35% above baseline without stopping the drug.
  - As forward flow to the glomerulus improves – there is less need for efferent vasoconstriction to compensate and glomerular filtration will stabilize
- Will cause acute renal failure in patients with bilateral renal artery stenosis
  - Dilation of efferent glomerular arterioles with no ability to dilate afferent arterioles which results in decreased glomerular filtration
  - In bilateral renal artery stenosis there is fixed flow into the glomerulus – an improvement in stroke volume will not improve flow into the glomerulus
ACE Inhibitors and GFR

ACE Inhibitor Monitoring and Contraindications

- Assess renal function and potassium within 1 to 2 weeks of initiation if outpatient
- High risk features for AKI: diabetes, hyponatremia, hypotension, azotemia, potassium supplementation, combination with aldosterone antagonist.

- **Note**: difference between AKI (hold regardless of creatinine) and CKD may give until creatinine of > 3.0

ESRD: ACE Inhibition ok. SBP most often limiting factor. Need reasonable SBP for dialysis.

**Cautions/Contraindications**
- Bilateral renal artery stenosis
- Creatinine > 3 mg/dL
- Potassium > 5.0 mEq/L
- Systolic BP < 80 mmHg
Angiotensin Receptor Blockers
End in “SARTAN”

- ACE Inhibitors remain the first choice for inhibition of RAAS
- ARB’s are a reasonable alternative to ACE Inhibitor if intolerant to ACE Inhibitor due to cough or angioedema
- Reasonable alternative to ACE I as 1st line therapy for patients with mild / moderate HF & reduced LVEF, especially if already take ARB for other reason (HTN)
- Directly blocks angiotensin II

- Combination of ACE I and ARB – not recommended

New class of medication: ARNI
- Angiotensin Receptor Blocker with Neprilysin Inhibitor
- Combo drug: sacubitril (Neprilysin Inhibitor) with valsartan (ARB)

PARADIGM-HF Trial
- Multinational, randomized, double-blind trial
- Comparing ENTRESTO with enalapril
- 8,442 adult patients with symptomatic chronic heart failure (NYHA class II–IV) and systolic dysfunction (left ventricular ejection fraction ≤40%).
- Results:
  - 20% reduction in the rate of death or hospitalization for heart failure
  - 16% reduction in the rate of all-cause death compared to enalapril at 3.5 years of follow-up.
Endogenous Vasoactive Peptides
- Naturetic peptides
- Adrenomedullin
- Bradykinin
- Substance P
- Calcitonin gene-related peptide

↓ Neurohormonal activation
↓ Vascular tone
↓ Cardiac fibrosis, hypertrophy
↓ Sodium retention

Neprilysin Inhibitor

Inactivates Vasoactive Peptides

Angiotensin Neprilysin Inhibition With LCZ696 Doubles Effect on Cardiovascular Death of Current Inhibitors of the Renin-Angiotensin System

Effect of ARB vs placebo derived from CHARM-Alternative trial
Effect of ACE inhibitor vs placebo derived from SOLVD-Treatment trial
Effect of LCZ696 vs ACE inhibitor derived from PARADIGM-HF trial
Entresto Dosing

Valsartan in Entresto is more bioavailable in Entresto than valsartan alone.

Dosing equivalents:

Valsartan In Entresto = Valsartan alone
- 24 mg in Entresto = 40 mg alone
- 49 mg in Entresto = 80 mg alone
- 97 mg in Entresto = 160 mg alone

Tiered Dosing:

Sacubitril /Valsartan (Paradigm HF listed doses)
- 24 mg/26 mg (50 mg)
- 49 mg/51 mg (100 mg)
- 97 mg/103 mg (200 mg)

Entresto

- Do not administer with ACE I
  - Increased risk of angioedema
  - Stop ACE I for 36 hours before starting Entresto
  - Do not administer in patients with history of angioedema
- Monitor kidney function, blood pressure and potassium levels
- BNP levels will not be accurate with Entresto but pro-BNP levels may be used
### Side Effect Profile

<table>
<thead>
<tr>
<th>Entresto</th>
<th>Enalapril</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypotension 18%</td>
<td>Hypotension 12%</td>
</tr>
<tr>
<td>Hyperkalemia 12%</td>
<td>Hyperkalemia 14%</td>
</tr>
<tr>
<td>Cough 9%</td>
<td>Cough 13%</td>
</tr>
<tr>
<td>Dizziness 6%</td>
<td>Dizziness 5%</td>
</tr>
<tr>
<td>Renal failure/AKI 5%</td>
<td>Renal failure/AKI 5%</td>
</tr>
</tbody>
</table>

**Hypotension pearl:**
Decrease diuretic prior to starting.

### Potential Concerns Related to Entresto

- **Alzheimer’s dementia**
  - Neprilysin breaks down the amyloid beta protein that is believed to collect in the brain and cause Alzheimer's dementia
  - Data from the Paradigm HF trial included no adverse events related to dementia
  - Clinical trials are being completed to assess for increased incidence of Alzheimer’s in this population

- **Macular degeneration**
  - Amyloid beta can also accumulate in the eye contributing to macular degeneration
  - Same concerns as with Alzheimer’s dementia

- **Prostrate Cancer**
  - Neprilysin suppression may contribute to tumor progression
ACC/AHA/HFSA Guideline Update
Recommendations for RAS Inhibition with ACE I or ARB or ARNI (Stage C HFrEF)

<table>
<thead>
<tr>
<th>COR</th>
<th>LOE</th>
<th>Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>ACE I: A</td>
<td>Inhibition of the RAS with ACE I OR ARB OR ARNI in conjunction with evidence-based betablocker, and aldosterone antagonist in selected patients, is recommended for patients with chronic HFrEF to ↓ mortality and morbidity.</td>
</tr>
<tr>
<td></td>
<td>ARB: A</td>
<td></td>
</tr>
<tr>
<td></td>
<td>ARNI: B-R</td>
<td></td>
</tr>
</tbody>
</table>

COR= Class of Recommendation (strength); green is recommended (Strong)
LOE = Level of Evidence (Quality); A = high quality evidence; B = moderated quality evidence; R = randomized


Aldosterone Antagonists

- Mineralocorticoid Receptor Antagonist (MRA)
- ACC/AHA Class IA Recommendation
- LVEF < 35% with NYHA Class II-IV Heart Failure to reduce mortality and morbidity

Diuretic effect is not primary reason for administration.

RALES Trial (1999) – 1663 pts
- NYHA Class III-IV
- LVEF ≤ 35%
- Standard rx. vs standard rx. with spironolactone
- 30% ↓ in mortality
- 35% ↓ in hospitalization

EMPHASIS-HF (2011) – 2737 pts
- NYHA Class II
- LVEF ≤ 35%
- Standard rx. vs standard rx. with epleranone
- 24% ↓ in all cause mortality
- 42% ↓ in HF hospitalization
Clinical Effects of Aldosterone

- Promotes retention of sodium
- Promotes loss of potassium and magnesium
- Potentiates catecholamines
- Inhibits the parasympathetic nervous system
- Decreases arterial compliance
- Promotes direct remodeling
- Has prothrombotic properties
- Causes vascular inflammation and injury

Lab Monitoring

- Potassium, sodium and renal function checked 2-3 days after initiation and again at 7 days then at the one month mark. If stable then every 3 months.
- Adding or increasing ACE I or ARB should increase potassium and creatinine surveillance.

Clinical Considerations

- Potassium sparing medications
- Any potassium supplements should be stopped after initiating - consider potassium based salt substitutes
- Counsel patients about avoiding foods high in potassium
- Prior to initiation:
  - Creatinine should be < 2.5 mg/dl (< 2.0 women)
  - Potassium should be < 5.0 mEq/L
  - No hyponatremia
Spironolactone (Aldactone)
• Non selective aldosterone blocker
  – Blocks aldosterone and androgen; stimulates progesterone
• RALES Trial

Eplerenone (Inspra)
• Selective aldosterone receptor antagonist
• EMPHASIS-HF
• EPHESUS (Post MI)

Major side effect: gynecomastia (10%), sexual dysfunction and menstrual problems due to non selectivity

Eliminates most gynecomastia and sexual side effects associated with aldactone

Hydralazine & Isosorbide Dinitrate

• The combination of hydralazine and isosorbide dinitrate (ISDN) is recommended to REDUCE MORBIDITY AND MORTALITY for:
  – Self-described African Americans with NYHA class III–IV HFrEF receiving optimal therapy with ACE inhibitors and beta blockers, unless contraindicated (Class I, LOE: A)
  – Anyone with current or prior symptomatic HFrEF who cannot be given an ACE inhibitor or ARB because of drug intolerance, hypotension, or renal insufficiency, unless contraindicated (Class IIa, LOE: B)

Self-identified African Americans are thought to have a less active renin-angiotensin system and a lower bioavailability of nitric oxide than those self-identified as white.

- 1050 self-described African American patients
- NYHA Class III or IV
- LVEF < 35% or < 45 with a dilated left ventricle
- Currently on standard therapy with BB for at least 3 months prior to enrollment
- Randomized to standard therapy + placebo (1 tablet TID) or standard therapy + 37.5mg of hydralazine and 20mg of ISDN (combined in one tablet TID)
- Dose increased to 2 tablets TID if no side effects with 1 tablet TID
- Trial ended early due to significantly higher rate of mortality in placebo group
  - 43% reduction in rate of death from any cause
  - 33% reduction in the rate of first hospitalization for heart failure in treatment group
  - Significant improvement in quality of life score in treatment group

Initiation:
- Hydralazine 37.5 mg / ISDN 20mg 3 times daily

Target dose:
- Total DAILY dose of Hydralazine 225 mg (75mg TID) and ISDN 120 mg (40mg TID)

Bidil – combo drug:
- Hydralazine 37.5mg / ISDN 20mg
- 1 up to 2 tablets TID

Adherence difficult

Adverse Reaction
- Headache
- Dizziness
- GI complaints

Consider slower titration to enhance tolerance
Digoxin

• Stage C Recommendations
  – Added in patients with persistent symptoms already on ACE Inhibitor, Beta-blocker and diuretic
• Positive inotropic effect – weak effect
• Enzyme inhibition in noncardiac tissues – reduces sympathetic flow
• **Improved symptoms**, exercise tolerance and quality of life
• No reduction in mortality
• Beta-blocker better for rate control
• Low dose: 0.125mg daily
• No need for loading dose
• Narrow therapeutic range in HF
  – 0.5 to 0.9 ng/ml

The ECG is a graphic recording of electrical activity spreading through the heart
  – 12 lead ECG provides 12 different views of electrical activity
  – Each bedside monitoring lead provides one view
The Electronics

Action Potential of Cardiac Cells

- Phase 0: Rapid depolarization – Sodium Influx (beginning of QRS complex)
- Phase 1: Brief, rapid initiation of repolarization

The Electronics

- Phase 2: Slowing of the repolarization – Calcium Influx – correlates with ST segment
- Phase 3: Sudden acceleration in the rate of repolarization - Potassium Efflux – Correlates with T wave
- Phase 4: Resting membrane potential
Electrical Conduction Pathway

- SA Node
- Interatrial pathways
- AV Node
- Bundle of His
- AV Junction
- Right and Left Bundle Branches
- Anterior and Posterior Fascicles
- Purkinje Fibers

WAVES and COMPLEXES

- P wave: atrial depolarization
- QRS: ventricular depolarization
- T wave: ventricular repolarization
- PR interval: AV conduction time
- QRS width: intraventricular conduction time
- ST Segment: sustained ventricular depolarization
- QT interval: used to reflect ventricular repolarization time
ECG Paper – Horizontal Axis

Normal speed:
• 25 mm / sec
• Smallest box 1mm x 1mm
• 1 small box 0.04 sec
• 1 large box 0.20 sec
• 5 large boxes 1.0 sec

Quick Method for Estimating HR
• Count number of large boxes between R waves (used for regular rhythms)
  1 = 300  2 = 150  3 = 100  4 = 75  5 = 60  6 = 50  7 = 43  8 = 37  9 = 33  10 = 30
ECG Paper – Vertical Axis

- Voltage or amplitude
- Measured in millivolts (mV) or millimeters (mm)
- EKG machine calibrated so that 1 mV produces a deflection measuring exactly 10 mm tall
- 1 small box = 1 mm high
- 1 large box = 5 mm high
**Dual and Single Electrode Leads**

**Dual Electrode Leads**
- One positive electrode
- One negative electrode
- Records difference in electrical potential between selected electrodes
- Leads I, II, and III

**Single Electrode Leads**
- One positive electrode
- One negative reference point
  - Zero electrical potential
  - Center of heart
- Leads aVR, aVL, aVF
- V1-V6

**Note:** All leads are bipolar.

---

**Frontal vs. Horizontal Planes**

- **Frontal plane:**
  - Leads I, II, III,
  - aVF, aVL, aVF
- **Horizontal plane:**
  - V leads
Consider the positive pole of each lead as “the camera” (exploring electrode).

Positive pole is where the camera is located. Negative pole tells the camera which way to look.
The Ground

- Note: Nothing travels toward the right leg as a positive electrode.
- The right leg is the ground used to absorb any excess electrical activity.

Standard Limb Leads
Leads I, II, III

Each Standard Limb Lead uses 2 surface electrodes per lead
Standard Limb Leads (Dual Electrode Leads)
Leads I, II, III

- Lead 1: RA - Left Arm + High Lateral Wall LV
- Lead 2: RA - Left leg + Inferior Wall LV
- Lead 3: LA - Left leg + Inferior Wall LV

Augmented Limb Leads
Leads aVR, aVL, aVF

Goldberberg’s Leads
Amplified to provide a signal that is useful.

Each Augmented Limb Lead uses one surface electrode per lead.
Augmented Limb Leads (Single Electrode Leads)  
\( aVR, aVL, aVF \)

Not specific to the LV

INFERIOR WALL of LV

CHST (Precordial) Leads (Single Electrode Leads)
**Electrode Placement**

**Chest (Precordial) Leads**

- **Lead V₁**
  - 4th ICS, RSB
- **Lead V₂**
  - 4th ICS, LSB
- **Lead V₃**
  - Midway Between V₂ & V₄
- **Lead V₄**
  - L. midclavicular line, 5th ICS
- **Lead V₅**
  - L. anterior axillary line, same level as V₄
- **Lead V₆**
  - L. midaxillary line, same level as V₄

---

**6 Precordial (Chest) Leads**

<table>
<thead>
<tr>
<th></th>
<th>V1</th>
<th>V4</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>+ 4th ICS, RSB</td>
<td>+ L MCL, 5th ICS</td>
</tr>
<tr>
<td></td>
<td>Septum</td>
<td>Anterior Wall LV</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>V2</th>
<th>V5</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>+ 4th ICS, LSB</td>
<td>+ L anterior axillary, same level as V₄</td>
</tr>
<tr>
<td></td>
<td>Septum</td>
<td>Low Lateral Wall LV</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>V3</th>
<th>V6</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>+ Midway Between V₂ &amp; V₄</td>
<td>+ L midaxillary line, same level as V₄</td>
</tr>
<tr>
<td></td>
<td>Anterior Wall LV</td>
<td>Low Lateral Wall LV</td>
</tr>
<tr>
<td><strong>Lead 1</strong></td>
<td><strong>aVR</strong></td>
<td><strong>V1</strong></td>
</tr>
<tr>
<td>-----------</td>
<td>---------</td>
<td>--------</td>
</tr>
<tr>
<td>RA – Left Arm + High Lateral Wall</td>
<td>Right Arm +</td>
<td>+ 4th ICS, RSB Septum</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Lead 2</strong></th>
<th><strong>aVL</strong></th>
<th><strong>V2</strong></th>
<th><strong>V5</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>RA – Left leg + Inferior Wall</td>
<td>Left Arm + High Lateral Wall</td>
<td>+ 4th ICS, LSB Septum</td>
<td>+ L anterior axillary, same level as V4 Low Lateral Wall</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Lead 3</strong></th>
<th><strong>aVF</strong></th>
<th><strong>V3</strong></th>
<th><strong>V6</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>LA – Left leg + Inferior Wall</td>
<td>Left Leg + Inferior Wall</td>
<td>+ Midway Between V2 &amp; V4 Anterior Wall</td>
<td>+ L midaxillary line, same level as V4 Low Lateral Wall</td>
</tr>
</tbody>
</table>

Any lead with a “V” use the heart as the negative reference point.

---

**Note!**

- Posterior wall of the left ventricle and the right ventricle are not captured on the standard 12 lead ECG.
The Importance of the Positive Electrode: Reason 2
Waves Record Differently When Depolarization is Moving Toward or Away from a Camera

1. Septum depolarizes from left to right
2. Both ventricles depolarize from endocardium to epicardium
3. Basal portions of ventricles depolarize last
4. Mean direction of depolarization is downward, leftward, and posterior

How Leads Record

- If positive electrode sees depolarization approaching it, it records an upright complex
- If positive electrode sees depolarization heading away from it, it records a negative complex.

If depolarization is proceeding perpendicular to a lead, a biphasic waveform is recorded: no net deflection.
A Closer Look at Lead I

- **Lead 1 Normals**
  - P waves: Upright and gently rounded
  - QRS Complex: Upright
  - T Waves: Upright and smaller than QRS

A Closer Look at Lead II

- **Lead II normals**
  - P wave: upright and gently rounded
  - QRS: upright
  - T wave: upright and smaller than QRS

**Note:** Must be upright
A Closer Look at Lead III

- Lead III normals
  - P wave: upright and gently rounded
  - QRS Complex: Usually upright
    - However, can occasionally record as negative
  - T wave: Generally upright
    - However, can have occasionally record as inverted

A Closer Look at aVR

- aVR Normals
  - P wave: inverted
  - QRS: inverted (rSr’ or rS)
  - T wave: inverted

Note: Must be negative
A Closer Look at aVL

- **aVL Normals**
  - P waves: Upright or inverted
  - QRS: Upright or inverted
  - T wave: Upright or inverted (but no down sloping of ST)

A Closer Look at aVF

- **aVF Normals**
  - P waves: upright and gently rounded
  - QRS: Upright
  - T wave: Upright and smaller than QRS
A Closer Look at V1

- Normal V1
  - P wave: inverted, upright or biphasic
  - QRS: inverted with rS pattern
  - T waves: inverted or upright

A Closer Look at V2

- V2 Normals
  - P waves: upright
  - QRS: inverted; rS pattern
  - T waves: usually upright
A Closer Look at V3

- V3 Normals
  - P wave: upright
  - QRS: equiphasic; RS pattern
  - T waves: Upright

A Closer Look at V4

- V4 Normals
  - P Wave: Upright
  - QRS: Upright; qRs
  - T wave: Upright
A Closer Look at V5

- **V5 Normals**
  - P wave: Upright
  - QRS: upright; qRs pattern
  - T wave: Upright

A Closer Look at V6

- **V6 Normals**
  - P wave: upright
  - QRS: upright; qRs pattern
  - T wave: upright
The R wave becomes taller and the S wave becomes smaller as the electrode is moved from right to left. This pattern is called R wave progression.

### Normal V1-6: R Wave Progression

<table>
<thead>
<tr>
<th>Lead 1</th>
<th>aVR</th>
<th>V1</th>
<th>V4</th>
</tr>
</thead>
<tbody>
<tr>
<td>+ Left Arm</td>
<td>+ Right Arm</td>
<td>+ 4th ICS, RSB</td>
<td>+ L MCL, 5th ICS</td>
</tr>
<tr>
<td>High Lateral Wall</td>
<td></td>
<td>Septal Wall</td>
<td>Anterior Wall</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lead 2</td>
<td>aVL</td>
<td>V2</td>
<td>V5</td>
</tr>
<tr>
<td>+ Left Leg</td>
<td>+ Left Arm</td>
<td>+ 4th ICS, LSB</td>
<td>+ L anterior</td>
</tr>
<tr>
<td>Inferior Wall</td>
<td>High Lateral Wall</td>
<td>Septal Wall</td>
<td>axillary, same</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>level as V₄</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Low Lateral Wall</td>
</tr>
<tr>
<td>Lead 3</td>
<td>aVF</td>
<td>V3</td>
<td>V6</td>
</tr>
<tr>
<td>+ Left Leg</td>
<td>+ Left Leg</td>
<td>+ Midway Between</td>
<td>+ L midaxillary</td>
</tr>
<tr>
<td>Inferior Wall</td>
<td>Inferior Wall</td>
<td>V₂ &amp; V₄</td>
<td>line, same level</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Anterior Wall</td>
<td>as V₄</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Low Lateral Wall</td>
</tr>
</tbody>
</table>
Normal P Wave Criteria

• Smooth and rounded
• No more than 2.5 mm in height
• No more than .11 seconds in duration
• Upstroke of P wave represents right atrial depolarization
• Down stroke of P wave represents left atrial depolarization

P Waves must be upright in Lead II in sinus rhythm

QRS Complex

• Not every QRS complex contains a Q wave, R wave and S wave!!
• Normal QRS width 0.05 to 0.10 sec
• Q – always negative (below baseline)
  – Normal Q not wider than 0.03 sec
• R – first positive above the baseline
• R’ – second positive above the baseline
• S – negative deflection following R wave or second component to entirely – complex

Remember: The alphabet says Q then R then S
Let's Practice

ST Segment

- In limb leads the ST segment is normally isoelectric but may be slightly elevated or depressed by less than 1mm.
- In precordial leads ST segment elevation may be present (not more than 1 to 2.5 mm) in many people.)
The “J” Point

- Point where the QRS complex and the ST segment meet.

T Waves

- Represents ventricular repolarization
- Slightly asymmetrical
- Usually upright in leads where QRS is upright
- Also – expect upright T waves in chest leads V2-V6
- Most likely abnormal if inverted in two contiguous leads
- T wave should be subordinate to the QRS it follows
Welcome Back!

Understanding the Electrical Axis of the Heart

• Direction (depolarization) of the mean electrical impulse of the heart

• Down and to the left
More About Axis

- Axis is determined by the sum of all electrical activity
- As depolarization moves through the conduction pathway the direction is constantly changing; however, the overall thrust of activity is in one direction
- Most of the electrical activity is directed towards the left ventricle due to the size of the myocardium required to eject blood

Axis Wheel
Axis Quadrants:

Adults:
-30° to +90°

Normal Axis

Note: Normal axis is extended into the left quadrant by 30 degrees.
**Axis Quadrants:**

**Leftward Axis and Left Axis Deviation**

**Causes:**

- Aging (changes in left anterior fascicular conduction)
- Left ventricular hypertrophy
- Acute MI (peri-infarction block)
- Congenital heart disease
- Hyperkalemia
- **Marked**: Associated with left anterior fascicular block

**Diagram:**

- Adults: 
  - -30° to -45°: Moderate
  - -45° to -90°: Marked

**Axis Quadrants:**

**Right Axis Deviation**

**Causes:**

- Infants / Children
- RV Hypertrophy
- Chronic Lung Disease
- Pulmonary Hypertension
- **Pulmonary Embolus**
- **Marked**: Often associated with left posterior fascicular block

**Diagram:**

- +90° to +120°: Moderate
- +120° to +180°: Marked
Axis Quadrants:

Extreme (Right Superior) Axis Deviation

Causes:
- Ventricular Tachycardia
- Other significant conduction abnormalities
Let Your Hands Determine Axis

- Use Lead I and aVF
- Left hand represents QRS in Lead I
- Right hand represents QRS in aVF
- Fingertips will point in the same direction as the QRS complex

Normal Axis:
+0 to +90 Degrees

- Lead I: Upright QRS
- aVF: Upright QRS
- It’s always “normal” to be on the up and up
Right Axis Deviation:
+90 to +180 Degrees

• Lead I: Downward QRS

• aVF: Upward QRS

• Your right hand is up so this is “right”
  axis deviation

Left Axis Deviation:
0 to –90 degrees

• Lead I: Upright QRS

• aVF: Downward QRS

• Your left hand is up so this is “left”
  axis deviation
Extreme (Right Superior) Axis:
-90 to -180 Degrees

- Lead I: Downward QRS
- aVF: Downward QRS
- Fingertips are both facing downward therefore the axis is "down and out" (opposite of normal)

Axis Practice
**Note:** Left axis can be a normal shift leftward or a true left axis deviation.

**Clinical Pearl:**
The key is to look at lead II. If the QRS is upright in lead II leftward axis is within normal limits. If the QRS in lead II is not upright then left axis deviation is present.
Conduction System Review

• Left Bundle Branch
  – Left anterior fascicle
  – Left posterior fascicle
• Right Bundle Branch

• Ventricular conduction time increases with increased heart size
• Measured on ECG by earliest onset to latest offset in all leads
Normal Depolarization

- QRS .06-.10 sec

Bundle Branch Block

- QRS complex is 0.12 sec or greater (in adults)
- Fixed or heart rate dependent
- Causes
  - Structural abnormalities
  - Functional (relative refractory period)
Right Bundle Branch Block

- \( V_1 = rsR' \)
- \( V_6 = qRS \)
- QRS = .12 sec or more

Causes

- Myocardial infarction
- Disease of right side of the heart
- **Pulmonary Embolism**
- Myocarditis
- Congenital heart disease
- Atrial septal defects

Note: Right bundle is more vulnerable than left bundle due to single fascicle.
Right Bundle Branch Block

• **V1**
  - Triphasic complex rsR' (rSr', rSR') pattern
  - Or an M shaped R wave with right peak taller
  - Or a qR pattern (In patients with septal infarct)

• **V6**
  - Triphasic complex
  - Large R is maintained
  - qRs with wide S waves
  Note: Also a wide S wave in lead 1

![ECG Diagram](image)
Left Bundle Branch Block

V₁ = QS
V₁ = rS
V₆ = wide R
QRS = .12 sec or more

Causes

- Coronary artery disease
- Hypertension
- Left ventricular hypertrophy
- Cardiomyopathy
Left Bundle Branch Block

- V1
  - Wide QS or rS complex - negative
  - Slick downstroke
  - Time to nadir <0.06 sec

- V6
  - Large R wave is maintained
  - Wide R wave with no initial septal q wave

Additional diagnostic criteria include:
Broad slurred or notched R waves in leads 1,aVL, V5, and V6.
In limb leads the ST segment is normally isoelectric but may be slightly elevated or depressed by less than 1mm.

In precordial leads ST segment elevation is normally not more than 1 to 2 mm (small elevation normal in many people).

**Clinical Application:**
1) Do not accept any elevation in limb leads
2) Do not accept any depression in chest leads

### Thresholds for ST Elevation

<table>
<thead>
<tr>
<th>Gender</th>
<th>Age</th>
<th>Leads</th>
<th>Threshold</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td>≥ 40</td>
<td>V2, V3</td>
<td>2 mm</td>
</tr>
<tr>
<td>Men</td>
<td>≥ 40</td>
<td>All except V2, V3</td>
<td>1 mm</td>
</tr>
<tr>
<td>Men</td>
<td>&lt; 40</td>
<td>V2, V3</td>
<td>2.5 mm</td>
</tr>
<tr>
<td>Women</td>
<td>All ages</td>
<td>V2, V3</td>
<td>1.5 mm</td>
</tr>
<tr>
<td>Women</td>
<td>All ages</td>
<td>All except V2, V3</td>
<td>1 mm</td>
</tr>
<tr>
<td>Men</td>
<td>≥ 30</td>
<td>V3R, V4</td>
<td>0.5 mm</td>
</tr>
<tr>
<td>Men</td>
<td>&lt; 30</td>
<td>V3R, V4R</td>
<td>1 mm</td>
</tr>
<tr>
<td>Women</td>
<td>All ages</td>
<td>V3R, V4R</td>
<td>0.5 mm</td>
</tr>
<tr>
<td>Men</td>
<td>All ages</td>
<td>V7 thru V9</td>
<td>0.5 mm</td>
</tr>
<tr>
<td>Women</td>
<td>All ages</td>
<td>V7 thru V9</td>
<td>0.5 mm</td>
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</table>
Thresholds for ST Depression

<table>
<thead>
<tr>
<th>Gender</th>
<th>Age</th>
<th>Leads</th>
<th>Threshold</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td>All ages</td>
<td>V2, V3</td>
<td>0.5 mm</td>
</tr>
<tr>
<td>Women</td>
<td>All ages</td>
<td>V2, V3</td>
<td>0.5 mm</td>
</tr>
<tr>
<td>Men</td>
<td>All ages</td>
<td>All except V2, V3</td>
<td>1 mm</td>
</tr>
<tr>
<td>Women</td>
<td>All ages</td>
<td>All except V2, V3</td>
<td>1 mm</td>
</tr>
</tbody>
</table>

The “J” Point

- Point where the QRS complex and the ST segment meet.

Clinical Application: There can be ST segment elevation with no J point elevation.
T Waves

• Represents ventricular repolarization
• Slightly asymmetrical
• Usually upright
• Most likely abnormal if inverted in two contiguous leads
• Not normally > than 5mm (limb leads) to 10 mm (precordial) high

Clinical Application:
3) Never tolerate a T wave that is too big in any lead.

Never Tolerate a T Wave that is Too Big
2 hours later

ECG Assessment Priorities
When Assessing for Injury or Ischemia

1) Assess for ST segment elevation first
   – ST elevation and need for reperfusion

2) Assess for T wave inversion next
   – Non STEMI or
   – Unstable angina
     • ischemia

3) Assess for ST segment depression thirdly
   – Supply and demand ischemia (often in V5 regardless of vessel occlusion)
   – OR – reciprocal changes to ST elevation
   – Clinical application: Supply and demand ischemia is typically not the primary problem in patients at rest

4) Assess patient symptoms

5) Correlate with history and risk factors

NonSTEMI: Troponin Abnormally Elevated
Unstable Angina: Troponin Normal
Hyperacute T Wave

Note: Hyperacute T waves can occur within 2 minutes of a coronary occlusion.

Hyperacute T Waves
Post Hyperacute T Waves

J Point Elevation
• T wave should be positive in lead I and II
• **Normal inversion is rare in V2 – V6**
• Inversion in lead III, aVL and aVF may be normal
• Inversion in V1 is common - always compare to previous ECG

**T Wave Inversion Associated With Ischemia/Infarction**
- Deep T wave Inversion
- Disproportionate T wave Inversion (in relation to QRS voltage)
- New or changing T wave Inversion
- QTc usually increased

Note: ST elevation is often subtle in the limb leads in an acute MI.
T wave inversion is a “warning” for ACS (either unstable angina or NonSTEMI) unless……………

- T wave inversion occurs after a STEMI
  - After a STEMI T wave inversion is expected
  - Terminal T wave inversion is a sign of reperfusion after a STEMI
  - Symmetrical T wave inversion will develop after terminal T inversion
Terminal T Wave Inversion: A Sign of Reperfusion

- “Wellen’s Warning” when seen in chest leads (V2-V3) of undiagnosed patient
  - Represents LAD occlusion that spontaneously reperfused prior to the ECG (lesion at risk for reocclusion)
  - Seen on ECG done during pain free period
  - Can be UA / NSTEMI

This pattern frequently occurs after successful reperfusion in STEMI s. It is an expected finding after reperfusion and not a warning!

Practice ECG 1 of 3
Can be seen in exercise stress testing with supply and demand ischemia. Often seen with left ventricular hypertrophy.

Clinical Pearls

**ECG**
- Be suspicious of horizontal ST segment depression in patient at rest.
- Suspect left main disease (or significant 3 vessel disease) when diffuse depression and ST elevation in lead aVR (and V1 to lesser extent)

**Presentation**
- Assess for reasons for supply and demand ischemia at rest (i.e. low hemoglobin).
- Rule out medical reason for falls, motor vehicle crashes, and other trauma (i.e. syncope or near syncope due to cardiac cause).
Practice ECG
Admitted with fall. Fracture femur admitted to surgical floor.

Practice ECG
Developed chest pain in PACU.
Cardiac Risk Factors

- Non-Modifiable Risk Factors
  - Previous history
  - Family history
    - 1st degree relative (parents, siblings)
    - Men < 55; Women < 65
  - Age
  - Gender
  - Socioeconomic Factors and Ethnicity

9 easily measured and potentially modifiable risk factors account for over 90% of the risk of an initial acute MI
- Smoking
- Hypertension
- Dyslipidemia
- Diabetes
- Obesity
- Metabolic Syndrome
- Inactivity
- Alcohol

Mortality Rate Age > 40 years:
1 year: F - 23%, M - 18%
5 year: F - 43%, M - 33%

Other Pertinent History

- CAD
- Cerebral Vascular Disease
- Peripheral Vascular Disease

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Stable Angina

- **Typical angina** is defined as angina that meets all three of the following characteristics:
  - Substernal chest discomfort with a characteristic quality and duration
  - Provoked by exertion or emotional stress
  - Relieved by rest or nitroglycerin.

- **Atypical angina** is defined as angina meeting two of the characteristics of typical angina

- **Non cardiac chest pain** is defined as chest pain with none or only one of the characteristics of typical angina.
Unstable Angina

- Caused by unstable or ruptured plaque that causes abrupt closure of a coronary artery which may spontaneously reperfuse.
- Occurs with minimal exertion or at rest
- Angina that increases in severity or is very severe on first presentation
- OR increased dose of nitroglycerin is required to achieve relief (Progressive angina)

Characteristics of Angina

**Sensation of pressure, tightness, heaviness, burning, or squeezing.**

- Usually no described as a sharp or stabbing pain (? women)
- Should not worsen with changes in position or respiration.

**Location behind the sternum and in the upper back, shoulder, arm, jaw, or epigastric area.**

- Not usually located in the middle to lower abdomen and does usually not radiate to the lower extremities.

**Associated symptoms (or stand alone symptoms) of dyspnea, nausea, palpitations, or diaphoresis.**

**Duration typically defined in minutes.**

- Not typically defined in seconds or hours.

CAUTION WHEN ASKING THE PATIENT ABOUT “PAIN”!
**Assessment of Angina**

- **Quality:**
  - Use the word “discomfort” or “symptoms” when assessing
  - Many patients with dyspnea or chest pressure deny the presence of pain.
- **Location:**
  - Assessment of location includes radiation of symptoms.
- **Time:**
  - Both the time of onset and duration of symptoms
- **Aggravating and alleviating factors:**
  - Key in differentiating stable from unstable angina.
- **Reproducibility:**
  - Reproducibility of chest pain by applying pressure to the chest wall suggests a musculoskeletal etiology.
  - Does not completely rule out the presence of angina.

**Angina and CAD in Patient with Diabetes**

- Autonomic dysfunction can affect symptoms experienced with angina
- Less likely to experience pain
- Approximately 20 - 25% of all patients presenting with ACS have diabetes
- More severe multi-vessel disease
- Greater proportion of ulcerated plaques resulting in intracoronary thrombi
- Higher rates of complications from ACS, higher mortality, and high rates of sudden death
Angina in Women

- Delay presenting with symptoms
- Attribute symptoms to other non-cardiac causes
- Presentation
  - More epigastric discomfort
  - Less specific complaints: dyspnea or fatigue
  - More atypical (sharp) chest pain
    - WISE Study: 65% of women presented with atypical symptoms
- Symptoms of discomfort from nose to navel should be evaluated for presence of CAD
- Less documented stenotic disease of major epicardial coronary arteries
  - Altered microvascular and endothelial function
  - Downstream microembolization

More on Women and Heart Disease

- Stable angina is often initial presentation
- Women with Non-STEMI and unstable angina are older than men and have more co-morbid conditions (diabetes and HTN)
- The average age for first MI is 64.7 years for men and 72.2 years for women (Go et al., 2013)
  - Female sex is a risk factor for mortality in STEMI
  - Women receive less evidence based therapies including reperfusion
**Angina in the Elderly**

- Generalized symptoms
  - Dyspnea, diaphoresis, N&V, and syncope
  - Confusion, weakness
- Symptoms often attributed to the aging process
  - Importance of assessment with activity tolerance
- Don’t complain about chest pain
  - 37% of patients > 65
  - 42% of patients > 75 years
  - 75% of those > 85 years
- Silent MIs account for 60% of MIs in those > 85 years of age
  - STEMI
    - < 65 years = 90% pain
    - > 85 years = 57% pain

**Acute MI Symptoms**

- Symptoms occur spontaneously and are not relieved by rest or nitroglycerin
- Chest pressure or discomfort may be accompanied by nausea, vomiting, or diaphoresis
- Patient may have hemodynamic instability or cardiac arrest from ventricular fibrillation
ECG Evolution of a STEMI

• Evolutionary changes occur due to prolongation of the cardiac action potential in regions of stunned myocardium adjacent to the necrotic area.
• Prolongation in repolarization affects the voltage and polarity of the T wave.
• Ventricular remodeling can produce differences in electrical fields that may be responsible for the evolutionary changes.

ECG Evolution of a STEMI

T wave inversion within first 3 days predictive of large amount of stunned myocardium.
  – Early terminal T wave inversion can occur with successful reperfusion.
  – T waves typically stay inverted for weeks to months
  – Persistent negative T waves or an increase in number of negative T waves during the 6 month period post discharge is associated with less regional wall motion recovery, more ventricular enlargement, and decreased left ventricular function over time.
• ST segment back to baseline occurs as result of myocardial cell death and reduction in injury current.
  – One of final evolutionary changes.
  – Abrupt resolution of ST elevation indicates reperfusion.
  – Persistent ST elevation is predictor of adverse outcomes.
ECG 3 of 3 Day 7 4:45 am

ST Evolution: Pseudo Normalization

Reoclusian
- Terminal T-wave inversion
- Pseudo-normalization of T-wave
- Increased ST elevation
Myocardial Free Wall Rupture

- Post-infarction regional pericarditis most often precedes rupture

T Wave Patterns in Post-infarction Regional Pericarditis

- Persistently positive T waves 48 hours after an MI
- Premature reversal of T wave inversion to positive
- ST segment reelevation

Reciprocal Changes

- Primary Change is most important – look for:
  - ST Elevation: ACS (STEMI)
  - T Wave Inversion: ACS (Non STEMI or UA)
  - ST Depression (ischemia)
- Reciprocal Changes
  - ST segment depression in leads reciprocal (opposite) those with ST elevation
  - Reciprocal changes can help confirm primary changes

Clinical Application:
Before calling ST segment depression ischemia – double check the reciprocal leads for missed ST segment elevation.
Specific Types of MIs

Nuances
Anterior MI

- Proximal LAD
  - Proximal to first diagonal
    - Anterolateral
  - Proximal to first septal perforator
    - Anteroseptal
- Mid LAD
  - Anterior MI

LAD: Anterior Wall, High Lateral Wall, Septum

Complications of Anterior MI

- Myocardium at risk
- Mortality and morbidity
- Post Infarction ejection fraction

Urgency for Reperfusion!
Complications of Anterior MI

- Tachycardia
  - Sinus tachycardia
  - Atrial tachycardia
  - Ventricular tachycardia
- Right BBB and left anterior hemiblock
- Complete heart block
- Ventricular septal defect
  - New loud systolic murmur
- Cardiogenic shock
- Long term ventricular modeling and heart failure

ECG Changes: Anterior MI

- ST Elevation
  - $V_3, V_4$ Anterior Wall
  - $V_1, V_2$ Septum

Various locations in the LAD will affect ST deviation vector with varying results on ST segment changes in the limb leads.

For Example: Occlusion proximal to diagonal branch results in ST elevation in Leads I and aVL.

For Example: Occlusion proximal to septal perforator shifts vector to right and results in additional ST elevation in Lead aVR.
Practice ECG

Practice ECG
Anterior Wall MI with RBBB
Inferior MI

• RCA occlusion 80% to 85% of time
  – Marginal branch: Right ventricle
  – Posterior descending artery = Posterior wall of LV
    • Concept of right versus left dominant

Clinical application: Assess right sided leads in patients with inferior MI.

Inferior MI

• Variations
  • Inferior posterior
  • Inferior with RV
  • Inferior posterior and RV
  • Inferolateral (often with circumflex)

• Complications
  • Sinus Bradycardia, 1st degree and 2nd Degree HB Type I
  • Increased parasympathetic activity
  • Papillary muscle rupture with posterior wall involvement
  • RV failure with RV involvement
ST Changes in Inferior MI

• ST elevation leads II, III, aVF
  – Lead III > II (RCA occlusion)
  – Lead II > III (Circumflex occlusion)

• **ST depression in aVL**

• ST elevation ≥ 0.5mm in inferior leads should be considered abnormal until proven otherwise
Practice ECG

Practice ECG 1 of 2

ECG in route via squad – ASA given
High Grade AV Block with RCA Occlusion

ECG 1of 3
Lateral Wall MI

- Lateral wall MIs are frequently associated with anterior, inferior, or posterior wall MIs
- However – when isolated are frequently missed
- ST elevation may be < 1 mm
- ST elevation may only be in aVL

Myocardium at Risk and Mortality Benefit Warrant Reperfusion Therapy
Coronary Artery Distribution to the Lateral Wall

- Lateral Wall
  - First diagonal branch of LAD (Leads 1, aVL)
  - Obtuse marginal branches of Circumflex (V₅, V₆)

ST Elevation with circumflex occlusion

ST elevation with RCA or LAD occlusion

Practice ECG
Note: MRI studies suggest that ECG evidence of posterior injury may actually reflect more anatomical injury of the lateral wall. However, ECG interpretation guidelines recommend we continue to refer to this type of MI as posterior.

- Coronary arteries and the posterior wall
  - RCA (responsible for Inferior / Posterior STEMI)
    - Posterior descending coronary artery
  - Circumflex (responsible for isolated posterior STEMI)
    - PDA
    - Marginal Branch
• Maximal ST depression $> 2$ mm in V1 – V3 may be 90% specific for posterior MI
• T waves usually remain upright
• Persistent ST depression is more commonly due to posterior STEMI than LAD disease........
• Anterior UA/ NonSTEMI is most likely when T wave inversion in present in V1-V4

• Reperfusion is indicated if true posterior MI is confirmed
  • Even with absence of ST elevation on standard 12 lead ECG
  • 3.3% to 8.5% of MIs are isolated posterior STEMI (Smith et al., 2002)
• Non reperfused posterior MI
  • Tall R waves in V1-V3

Remember: Increased risk for papillary muscle ischemia or rupture. May hear new holosystolic murmur.
• Approximately 1/3 of Inferior MIs
  – Occlusion proximal to marginal branch of RCA

**Recognition**

• > 0.5 mm ST elevation in V4R
  – Men < 30 years of age (> 1.0 mm ST elevation in V4R)

• Suspect when elevation in V1 but not in V2
  – However, cannot rely on if there is simultaneous RV and posterior involvement

• Combination of elevation in V4R and V1 is very specific to RV infarct

• Reciprocal changes to RV injury may be seen in low lateral leads
RV Infarct

• Implications
  – Increased short term mortality
  – RV can recover well if patient survives acute phase
• RV Failure = Decreased LV Preload = Decreased Stroke Volume = Hypotension
• Clinical presentation
  – Hypotension
  – Clear lungs
  – Signs of RV failure (increased CVP; jugular venous pressure)

RV Infarct

• Pathophysiology of RV infarct is complex
  – Ventricle can be noncompliant or distensible
• Treatment of hypotension in RV infarct
  – Avoid diuretics and venous vasodilators
  – IV fluids to CVP of 15 mmHg and PAOP of 15 mmHg (Brenner & Tschopp, 2009)
  – Caution with too much fluid:
    • Distendability
    • Septal displacement
  – Need for inotrope when fluid loading not successful
  – Atrial fibrillation and bradyarrhythmias need treated
Note: V1 and V2 may also be reversed with V1 on LSB and V2 on RSB.

If so, these are labeled V1R and V2R.

Right Ventricular Leads

May also record V₃R, V₄R, and V₅R.
Mirror image of normal

Posterior Leads

V7: Posterior axillary line
V8: Under tip of scapula
V9: Same level at paraspinal border
All posterior leads the same level as V6
Labeling the Additional Leads

Note: Verify that electronically recorded 12 leads are able to labeled correctly.

<table>
<thead>
<tr>
<th>Original Chest Leads</th>
<th>New Chest Leads</th>
</tr>
</thead>
<tbody>
<tr>
<td>V1</td>
<td>V4R</td>
</tr>
<tr>
<td>V2</td>
<td>V5R</td>
</tr>
<tr>
<td>V3</td>
<td>V6R</td>
</tr>
<tr>
<td>V4</td>
<td>V7</td>
</tr>
<tr>
<td>V5</td>
<td>V8</td>
</tr>
<tr>
<td>V6</td>
<td>V9</td>
</tr>
</tbody>
</table>

Right Sided and Posterior Quick Look on Bedside Monitor

Right Sided Lead

• Place electrode in V4R Position
  – 5th ICS Right MCL
• Attach V monitoring lead (Brown Lead) to electrode
• Assure monitor lead selector is on V
• Run strip and clearly mark “V4 Right Chest Lead”

Posterior Lead

• Place electrode in V8 position
  – Under tip of left scapula same level as V6
• Attach V monitoring lead (Brown Lead) to electrode
• Assure monitor lead selector is on V
• Run strip and clearly mark “V8 Posterior Lead”
• Inferior STEMI: ST Elevation in II, III, aVF
• Reciprocal depression in Leads I and aVL
• ST Depression in V2 and V3: Reciprocal changes from Posterior STEMI
• Ideal patient for a 16 lead ECG to assess for injury to the right ventricle and posterior wall of the left ventricle.

Same Patient as Previous 12 Lead:
Due to hypotension the point of care nurse used the V lead from bedside monitoring to record a V4R lead. This recording confirms RV injury and this knowledge was used to guide treatment.

ST segment elevation in lead V4R demonstrates high risk for high grade AV node block
(Wellen & Conover, 2006).
**Acute Coronary Syndrome (ACS)**

- No ST Elevation
- ST Elevation
- Non STEMI
- Unstable Angina
- STEMI

**Hospitalizations in the U.S. due to Acute Coronary Syndromes**

1, 190,000 Hospital Discharges with primary or secondary diagnosis of ACS

<table>
<thead>
<tr>
<th>UA/NSTEMI</th>
<th>STEMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>NRMI-4*: 71%</td>
<td>NRMI-4*: 29%</td>
</tr>
<tr>
<td>AHA Get with the Guidelines: 69%</td>
<td>AHA Get with the Guidelines: 31%</td>
</tr>
<tr>
<td>GRACE** Study: 62%</td>
<td>GRACE** Study: 38%</td>
</tr>
</tbody>
</table>

*NRMI-4: National Registry of Myocardial Infarction; **GRACE Study: Global Registry of Acute Coronary Events.
Acute Coronary Syndrome refers to any rupture of plaque or thrombotic event that leads to symptomatic ischemia or infarction.

Ruptured Plaque

- STEMI
- NonSTEMI / Unstable Angina

Pathophysiology of ACS

- Deposit of lipids, calcium, fibrin, and other cellular substances within the lining of the arteries.
- Initiates a progressive inflammatory response in an effort to heal the endothelium.
- End result of inflammatory process: the production of a fibrous atherosclerotic plaque.
- Plaque can progress to cause coronary stenosis.
- Plaque can also rupture prior to causing significant stenosis.
Plaque

- Stable plaque of stable angina
  - Thick fibrous caps separate the lipid core from the endothelium
  - Less complicated than vulnerable plaques
  - Tend to have smooth outlines

- Vulnerable plaque of ACS
  - Thin caps
  - Edge of the fibrous cap is a particularly vulnerable area and is commonly the location of ruptured plaque

- Limitations of stress testing
**STEMI**

29-38% of ACS patients
Complete occlusion of a vessel by a thrombus
Fibrin stable clot (red clot)
Classified more specifically by the portion of the left ventricle suffering injury.
Mortality is greatest within the first 24 to 48 hours of symptom onset

TREATMENT FOCUS = REPERFUSION

**NSTE – ACS**

Nationally under treated according to evidence based practice guidelines (Crusade Registry)
Pathophysiology often involves a platelet plug or white clot
Less stable clot
Opportunity for spontaneous reperfusion
NSTEMI differentiated from unstable angina by troponin levels

TREATMENT FOCUS = ANTIPLATELET THERAPY
Supply and Demand Mismatch

- Increase myocardial oxygen demand:
  - Hyperthermia
  - Hypertension
  - Tachycardia
  - Conditions producing over stimulation of the sympathetic nervous system (cocaine use, hyperthyroidism)

- Decrease myocardial oxygen delivery:
  - Anemia
  - Pulmonary disease.

- Increase myocardial oxygen demand and decrease myocardial oxygen supply:
  - Aortic stenosis
  - Hypertrophic cardiomyopathy

Elderly are at risk for secondary coronary events related to supply and demand imbalance.

Cardiac Biomarkers
Troponin I and T (cardiac troponins)

- Found only in cardiac muscle
- Most sensitive indicator of myocardial damage
  - Capable of diagnosing small amounts of myocardial necrosis not measured by rises in CK-MB levels
- Approximately 30% of patients with non-ST elevation and normal CKMB levels will test positive for Non-STEMI
- Of equal sensitivity and specificity
- Troponin remains elevated for a long period
  - Beneficial for late presentation
  - Challenging for re-infarction
- Positive troponin + ECG changes of injury / ischemia or ACS symptoms = INFARCT
Non infarct cardiac causes of elevated troponin: heart failure, left ventricular hypertrophy, tachyarrhythmias, pericarditis, cardiac trauma

- Non CAD causes of troponin elevation (sepsis, pulmonary emboli, chronic kidney disease, chemotherapy, respiratory failure, burns, neurological disease)
- Troponin I more specific in renal dysfunction
  - Patients with ESRD commonly have elevated troponin T
    - Not a false positive - relates to overall dysfunction of the cardiorenal system
  - < 10% of patients with ESRD have elevated troponin I in absence of ACS
- Elevated troponin levels are marker of risk and associated with an increased mortality – even when diagnosis is not myocardial infarction
- Degree of troponin elevation correlates with risk of death
- New high sensitivity troponin T

Cardiac Biomarker Summary

<table>
<thead>
<tr>
<th>Cardiac Biomarker</th>
<th>Specificity / Sensitivity</th>
<th>Rise</th>
<th>Peak</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>CK-MB</td>
<td>Highly specific</td>
<td>4 to 6 hours</td>
<td>18 to 24 hours</td>
<td>2 to 3 days</td>
</tr>
<tr>
<td>Troponin I or T</td>
<td>Highly specific and sensitive</td>
<td>4 to 6 hours</td>
<td>18 to 24 hours</td>
<td>10 or more days</td>
</tr>
</tbody>
</table>
Timing of Release of Various Biomarkers After Acute Myocardial Infarction

Medical Management of STEMI

- ASA: 325 mg (non enteric coated)
  - If fibrinolytic therapy – 162-325 mg
- P2Y12 inhibitor (loading dose before or at time of PCI)
  - If fibrinolytic therapy - clopidogrel only
- Anticoagulants (related to reperfusion strategy)
  - If fibrinolytic – weight based heparin x 48 hours
- Oral beta blockers ASAP
  - IV if hypertensive or tachycardic
- NTG – Sublingual vs IV
- Morphine Sulfate (Class I)
- Oxygen if hypoxemic (arterial oxygen saturation < 90%)
- High intensity statin therapy
- D/C NSAIDS
- ACE Inhibitors (within 24 hours)
  - Greatest benefit in anterior wall MI, LVEF < 40%, HTN, diabetes or chronic kidney disease
- Aldosterone Antagonists
  - Initiate within 7 days in those with LVEF <40%, HF, or diabetes
**Recommendation for Glucose Control**

- It is reasonable to use an insulin-based regimen to achieve and maintain glucose levels less than 180 mg/dL while avoiding hypoglycemia* for patients with STEMI with either a complicated or uncomplicated course.

---

**STEMI Management**

- **Reperfusion is number one treatment strategy**
  - Primary Coronary Intervention (PCI) preferred treatment strategy if within 90 minutes
    - Goal: 90 minutes from 1st medical contact
  - Fibrinolytics within 30 minutes of hospital presentation (or 30 minutes from EMS to fibrinolytics)

*Facilitated PCI with full dose fibrinolytics is not recommended.

*Rescue PCI may be done after failed fibrinolytics*
The Winner!

Interventional Revascularization: PCI
(Primary Coronary Intervention (in STEMI) or Percutaneous Coronary Intervention)

- PTCA: Percutaneous transluminal coronary angioplasty
- Coronary Stent
  - BMS: Bare metal
  - DES: Drug eluting
- Coronary Extraction Atherectomy – no longer routinely recommended

However:
Timely reperfusion is the priority over method of reperfusion.
Interventional Revascularization

- Indications
  - Reperfusion in STEMI
    - Strategy of choice if 90 minute reperfusion time
  - Unstable angina / NSTEMI
    - High risk features
  - Stable angina
    - Courage Trial -2007

- Contraindications
  - When antiplatelet therapy is contraindicated

- Complications
  - Abrupt closure
  - Dissection
  - In stent thrombosis (acute or late)
  - Down stream embolization
  - Emergency CABG
  - Bleeding or hematoma
  - Pseudoaneurysm*
  - Retroperitoneal Bleed
  - Arterial Embolus
  - Contrast nephropathy
  - Restenosis (late)
  - Coronary artery aneurysm (late)
  - MI
  - Stroke
  - Death
Reasons for Delayed or Missed Reperfusion Therapy

• Missed diagnosis of unequivocal ECG due to atypical symptoms
• Unrecognized unequivocal ECGs
• Delay in diagnosis of subtle ECGs or failure to perform serial ECGs
• Delay in administration of therapy or inappropriate abortion of treatment.
  – Resolution of pain alone is not an indication for aborting therapy. Look for 50-100% resolution of ST-segment elevation before considering suspending reperfusion therapy based on further evaluation.
Each community should develop a STEMI system of care consistent with minimum standards of AHA’s Mission Lifeline

Door to device time alone is not sufficient to further reduce mortality

The average time of presentation after symptom onset is 1.5 to 2.0 hours.

Patient populations with the longest delays are women, African Americans, and the elderly.

Nurses can make an impact through patient and community education and awareness campaigns.

60 minutes is the golden hour: Survival rates improve significantly.
Medical Management of NSTEMI – ACS

- Dual antiplatelet
- Anticoagulation
- Oxygen if $\text{SpO}_2 < 90$
- NTG
  - IV in first 48 hours for persistent ischemia, HTN, HF
  - Should not interfere with mortality reducing beta blockers or ace inhibitors
- MS (if NTG unsuccessful and other anti ischemic drugs on board )
- Beta Blockers (within 24 hours)
  - Start PO when hemodynamically stable
  - May use IV if hypertensive
- ACE Inhibitors (within 24 hours)
  - In select patients – pulmonary congestion or LVEF $\leq 40\%$ – may also be used in other patients
- High intensity statin
- DC – NSAIDS

Medical Supportive Therapy: Similar to STEMI

Priority Treatment is to Attack the Platelet
Attacking Platelet is number one treatment strategy

Two antiplatelets agents are indicated

There are 3 types of antiplatelet agents
- Aspirin
- P2Y12 Receptor Antagonists
- Intravenous GP IIb/IIIa Inhibitors

Dual antiplatelet therapy for invasive strategies in medium to high risk patients
- ASA (and one of following)
- P2Y12 / ADP Receptor blockers
  - Clopidogrel
  - Prasugrel
  - Ticagrelor * preferred over clopidogrel
- GP II b / III a Inhibitors
  (*eptifibatide, * tirofiban, abciximab)
  - * preferred agents
  - Used only in special circumstances

Antiplatelet Therapy also in conservative treatment
- Prasugrel not unless PCI is

Dual antiplatelet therapy is also used after STEMI and after any coronary intervention.
Stent Restenosis Compared to Stent Thrombosis

Early Invasive Option in NSTE-ACS Versus Ischemia Guided Treatment

• What is it?
  – Not waiting for failed medical treatment
  – Not waiting for + noninvasive test
  – Angiography with intent of revascularization
  – Done within 12 to 24 hours

Overall reduction in mortality and increased quality of life.
Early Invasive Strategy

- Initially stable patients with a high risk for clinical events
- Refractory angina
- Hemodynamic instability
- Electrical instability
- Excluded: very frail elderly, severe hepatic, renal or pulmonary disease / active or inoperable cancer
- Early invasive therapy is not recommended in patients with acute chest pain with a low likelihood of ACS
- Early invasive therapy is not recommended in patients who do not want to consent to revascularization.

Algorithm for Management of Patients With Definite or Likely NSTE-ACS

- NSTE-ACS: Definite or Likely
  - Ischemia-Guided Strategy
    - Initiate DAPT and Anticoagulant Therapy
      1. ASA (Class I; LOE: A)
      2. P2Y12 inhibitor (in addition to ASA) (Class I; LOE: B):
        • Clopidogrel or
        • Ticagrelor
      3. Anticoagulant:
        • UFH (Class I; LOE: B) or
        • Enoxaparin (Class I; LOE: A) or
        • Fondaparinux† (Class I; LOE: B)
  - Early Invasive Strategy
    - Initiate DAPT and Anticoagulant Therapy
      1. ASA (Class I; LOE: A)
      2. P2Y12 inhibitor (in addition to ASA) (Class I; LOE: B):
        • Clopidogrel or
        • Ticagrelor
      3. Anticoagulant:
        • UFH (Class I; LOE: B) or
        • Enoxaparin (Class I; LOE: A) or
        • Fondaparinux† (Class I; LOE: B) or
        • Bivalirudin (Class I; LOE: B)

Can consider GPI in addition to ASA and P2Y12 inhibitor in high-risk (e.g., troponin positive) pts (Class IIb; LOE: B)
- Eptifibatide
- Tirofiban
**High Risk Features in UA / NSTEMI**

- Recurrent angina / ischemia  
  - Rest or low level activity with medical treatment
- Troponin +
- New or presumed new ST depression
- S&S HF or worsening mitral regurgitation
- High risk findings on noninvasive testing  
  - EF < 35%, large anterior perfusion defect, multiple perfusion defects)
- Hemodynamic instability
- Sustained VT
- PCI within 6 months
- Prior CABG
- Reduced LV Function
- High risk TIMI or GRACE Score

**Population > 75 years:**
80% are high risk

**Elderly:**
cancer, renal insufficiency, lung disease, anemia, and heart failure are common co morbid conditions

---

**Risk Assessment in NSTE-ACS**

**TIMI Risk Score**
- Age > 65
- 3 or > risk factors for CAD
- Prior 50% or > stenosis
- ST deviation on ECG
- 2 or > anginal events in previous 24 hours
- Use of ASA in prior 7 days
- Elevated cardiac biomarkers

**GRACE**
- Older age
- Killip class
- Systolic BP
- Cardiac arrest during presentation
- Serum creatinine
- Positive initial cardiac markers
- HR
### TIMI Risk Score* for NSTE-ACS

<table>
<thead>
<tr>
<th>TIMI Risk Score</th>
<th>All-Cause Mortality, New or Recurrent MI, or Severe Recurrent Ischemia Requiring Urgent Revascularization Through 14 d After Randomization, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–1</td>
<td>4.7</td>
</tr>
<tr>
<td>2</td>
<td>8.3</td>
</tr>
<tr>
<td>3</td>
<td>13.2</td>
</tr>
<tr>
<td>4</td>
<td>19.9</td>
</tr>
<tr>
<td>5</td>
<td>26.2</td>
</tr>
<tr>
<td>6–7</td>
<td>40.9</td>
</tr>
</tbody>
</table>

*The TIMI risk score is determined by the sum of the presence of 7 variables at admission; 1 point is given for each of the following variables: ≥65 y of age; ≥3 risk factors for CAD; prior coronary stenosis ≥50%; ST deviation on ECG; ≥2 anginal events in prior 24 h; use of aspirin in prior 7 d; and elevated cardiac biomarkers.

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### Short-Term Risk of Death/Nonfatal MI in Patients With UA/NSTEMI

<table>
<thead>
<tr>
<th>Feature</th>
<th>High Risk</th>
<th>Intermediate Risk</th>
<th>Low Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>History</td>
<td>≥ 1 of the features below must be present: Accelerating tempo of ischemic sx in preceding 48 h</td>
<td>No high-risk features, but must have 1 of the following: Prior MI, peripheral or cerebrovascular disease, or CABG; prior ASA use</td>
<td>No high- or intermediate-risk features but may have any features below:</td>
</tr>
<tr>
<td>Character of pain</td>
<td>Prolonged ongoing (&gt; 20 min) rest pain</td>
<td>• Prolonged (&gt; 20 min) rest angina, now resolved, w/ moderate/high likelihood of CAD • Rest angina (&gt; 20 min) or relieved with rest or sublingual NTG • Nocturnal angina • New-onset or progressive CCS class III/IV angina in past 2 wks w/o prolonged (&gt; 20 min) rest pain but with intermediate/high likelihood of CAD</td>
<td>• ↑ Angina frequency, severity or duration • Angina provoked at lower threshold • New onset angina with onset 2 wks to 2 mos prior to presentation</td>
</tr>
</tbody>
</table>
**Short-Term Risk of Death/Nonfatal MI in Patients With UA/NSTEMI, Continued**

<table>
<thead>
<tr>
<th>Feature</th>
<th>High risk</th>
<th>Intermediate risk</th>
<th>Low risk</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical findings</strong></td>
<td>• Pulmonary edema, most likely due to ischemia</td>
<td>Age &gt; 70 y</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• New/worsening MR murmur</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• S₃ or new/worsening rales</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Hypotension, bradycardia, tachycardia</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Age &gt; 75 y</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>ECG</strong></td>
<td>• Angina @ rest with transient ST-segment changes &gt; 0.5 mm</td>
<td>T-wave changes</td>
<td>Normal or unchanged ECG</td>
</tr>
<tr>
<td></td>
<td>• BBB, new/presumed new</td>
<td>Pathological Q-waves/resting ST-depression &lt; 1 mm in multiple lead groups (anterior, inferior, lateral)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Sustained VT</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Cardiac markers</strong></td>
<td>↑ Cardiac TnT, TnI, or CK-MB (e.g., TnT/Tnl &gt; 0.1 ng/mL)</td>
<td>Slightly ↑ cardiac TnT, TnI, or CK-MB (e.g., TnT &gt; 0.01, but &lt; 0.1 ng/mL)</td>
<td>Normal</td>
</tr>
</tbody>
</table>

Estimation of the short-term risk of death and nonfatal cardiac ischemic events in UA/NSTEMI is a complex multivariable problem that cannot be fully identified in a table such as this; this table is meant to offer general guidance & illustration rather than rigid algorithms. Braunwald E, et al. - AHCPR Publication No. 94-0602:1-154. Anderson JL, et al. J Am Coll Cardiol 2007;50:e1-e157, Table 7.

**Long Term Management of ACS**

**Medications to improve prognosis**

- **Aspirin**
  - ASA benefits > in those > 65 years
  - Long term benefit with 81 mg

- **Clopidogrel / Prasugrel / Ticagrelor**
  - Dual antiplatelet therapy in conservative management for 12 months
  - Higher risk of bleeding with dual antiplatelet therapy
    - No elderly sub group data for clopidogrel

- **Statins**
  - Have greater benefit in elderly for reduction of future MI and death than in younger patient populations

2017
Long Term Management of ACS

**Medications to improve prognosis**

- Beta-blockers
- ACE inhibitors
  - Definite in select patients / reasonable in all
  - ARBs if ACE-I intolerant
- Aldosterone antagonists
  - EF ≤ 40 with HF or diabetes

**Impact long term ventricular remodeling**
SL NTG Instruction

- No more than 1 dose of SL NTG
  - If chest discomfort is unimproved or is worsening 5 min after 1 NTG call 9-1-1 immediately before taking additional NTG.
  - May take additional NTG while waiting EMS.
  - Chew ASA while waiting EMS.

- In chronic stable angina if symptoms are significantly improved by 1 dose of NTG may repeat NTG every 5 min for a maximum of 3 doses and call 9-1-1 if symptoms have not resolved completely.

Stepped Care Approach To Pharmacologic Therapy for Musculoskeletal Symptoms with Known Cardiovascular Disease or Risk Factors for Ischemic Heart Disease

Add ASA 81 mg and PPI to patients at increased risk of thrombotic events *

- Acetaminophen, ASA, tramadol, narcotic analgesics (short term)
- Nonacetylated salicylates

- Regular monitoring for sustained hypertension or worsening of prior blood pressure control, edema, worsening renal function, or gastrointestinal bleeding.
- If these events occur, consider reduction of the dose or discontinuation of the offending drug, a different drug, or alternative therapeutic modalities, as dictated by clinical circumstances.

NSAIDS (except for ASA), whether nonselective or COX-2-selective agents increase risk of mortality, reinfection, hypertension, HF, and myocardial rupture

- NSAIDs with some COX-2 activity
- COX-2 Selective NSAIDs

Secondary Prevention: ACS and Stable CAD

- Smoking cessation
- Reduction of hyperlipidemia
  - LDL < 100 mg/dL or < 70 mg/dL (optimal)
- Hypertension control
  - <130/80 for kidney disease or diabetes
- Diabetes control Hb A1c < 7
- Physical activity minimum of 5 days / per week
  - 7 days recommended
- BMI 18.5 – 24.9 kg/mm²
- Phase II Cardiac Rehab
- Influenza Vaccine / Pneumonia Vaccine

Key Nursing Care Considerations

- Use oxygen for hypoxemia
- Assess response to beta-blocker therapy.
  - HR / BP
  - Arrhythmia control
- Assess for complications related to specific type of MI
  - Assess heart sounds for new holosystolic murmurs
    - Risk for myocardial rupture
  - Observe for signs of left ventricular dysfunction, including hypotension or clinical signs of heart failure.
  - Monitor ECG for conduction disturbances and arrhythmias.
Key Nursing Care Considerations

- Management of arterial access site
- Assessment for contrast nephropathy
- Restrict activity for the first 12 hours, and then begin Phase I Cardiac Rehabilitation (progressive mobility)
- Utilize cardiac monitoring
  - ST-segment monitoring
  - Uninterrupted monitoring for first 24-48 hours
- Address addiction to nicotine
  - Consideration for nicotine withdrawal
- Focus on holistic approach to anxiety reduction
  - Include the family. Family visits do not have a negative impact on vital signs or cardiac rhythm

Activity

- After the patient achieves a rehabilitation level equivalent with activities of daily living, he/she can begin a walking program
  - 3 to 4 METS
  - Should be by time of discharge
  - Begin walking 5 to 10 minutes at a time
- Patients should rate activity as moderate
- Shortness of breath means overexertion. Other signs of activity intolerance include: angina, dizziness, diaphoresis, prolonged fatigue, and nausea.
- The use of force to open windows or tight jar lids should be avoided in patients with lifting restrictions.
Linking Knowledge to Practice

✓ When providing instructions regarding weight lifting restrictions it is helpful to know the weight of common household items.

A gallon of milk = 8 pounds,
A bag of groceries between 5 & 10 pounds
A large basket of laundry 20 pounds or more.

Typical MET Levels of Common Activities

<table>
<thead>
<tr>
<th>Activity</th>
<th>MET Level</th>
<th>Activity</th>
<th>MET Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Washing, dressing, grooming</td>
<td>2.1</td>
<td>Grocery shopping</td>
<td>2.1</td>
</tr>
<tr>
<td>Light housework</td>
<td>2.51</td>
<td>Heavy equipment operation</td>
<td>6.0</td>
</tr>
<tr>
<td>Interior cleaning</td>
<td>3.01</td>
<td>Vehicle repair</td>
<td>2.93</td>
</tr>
<tr>
<td>Laundry</td>
<td>2.07</td>
<td>Walking (moderate to brisk)</td>
<td>3.0 to 5.0</td>
</tr>
<tr>
<td>Food preparation and clean up</td>
<td>2.54</td>
<td>Jogging / Running</td>
<td>6.3 to 8.0</td>
</tr>
<tr>
<td>Light shoveling (&lt;10 pounds)</td>
<td>6.0</td>
<td>Golfing (Pulling Clubs)</td>
<td>3.75 (4.3)</td>
</tr>
<tr>
<td>Lawn and garden</td>
<td>3.45 to 3.66</td>
<td>Strength training</td>
<td>3.0</td>
</tr>
<tr>
<td>Physical care for children</td>
<td>2.67 to 2.72</td>
<td>Dancing</td>
<td>4.5</td>
</tr>
<tr>
<td>Physical care for adults</td>
<td>2.89</td>
<td>Biking</td>
<td>8.0</td>
</tr>
</tbody>
</table>
Driving

- Driving requires only 1.5 to 3.0 METS.
- Most patients with an uncomplicated hospital course can drive 1 week after discharge. Driving instructions should be compliant with any existing state regulations.
- Patients should be accompanied when they resume driving and should avoid stressful driving situations such as night driving, rush hour, high speeds, and driving during heavy rain or snow.
- Driving should be delayed for 2 to 3 weeks in patients with a complicated myocardial infarction. This includes patients who had a cardiac arrest, hypotension, arrhythmias, or heart failure during hospitalization.

Travel

- Patients can usually travel by air within 2 weeks if accompanied by a travel companion, and if the patient has sublingual nitroglycerin
  - If free of all angina symptoms and complications of their myocardial infarction
- Patients should also have airport transportation assistance to avoid excessive stress and rushing in the airport
- Patients should also take precautions when traveling to avoid the development of deep vein thrombosis
Sex

• After an acute coronary syndrome, stable patients can resume sexual activity with their usual partner in one week to 10 days (Anderson et al., 2011).

• Patients are uncomfortable asking about resuming sexual relationships, so instructions regarding sexual activity should be included as a routine part of all discharge instructions.

• Patients with a history of angina during sexual relationships may be instructed to take nitroglycerin prior to engaging in sexual activities.

• The average intimate session ranges from 2.5-4 METS for most people.
  – Walking at 2 mph on level ground is 2.5 METS. Mowing the lawn with a power mower or walking at 3.5 mph is 4 METS. Climbing up a flight of stairs is 8 METS.
  – The biggest risk with sex in the cardiac patient is the possibility of arrhythmias, which is associated with sympathetic activity increased during arousal. Patients with uncontrolled or untreated hypertension need to discuss specific guidelines with their physician (Sotile & Cantor-Cooke, 2003).

Return to Work

• Low risk myocardial infarction (LVEF > 45%, successful revascularization with PCI, age < 70 years) can generally return to work after 2 weeks.

• Most myocardial infarction adverse events reach a low steady state at 10 weeks. This may guide decision making in some types of employment.

• Patients who need to return to physically demanding activities can have an exercise stress test that compares their performance on the stress test to the METs required for the activity. This will provide information about the ability and safety of engaging in activities based on the MET level achieved during exercise stress test. (Anderson et al., 2011).
Cardiac Rehabilitation

• Goals:
  – Increase functional capacity
  – Reduce disability
  – Improve quality of life
  – Modify cardiac risk factors
  – Reduce morbidity and mortality.

• Pooled data from a meta-analysis of studies involving the exercise portion of cardiac rehabilitation show a benefit of reduced all-cause mortality of approximately 25% when compared to usual care.

• In one study of over 600,000 Medicare patients, mortality rates were 21% to 34% lower in patients

Cardiac Rehabilitation

• Low-risk patients can implement an exercise prescription at home or in a community setting. Low-risk patients include those with absence of ischemia or arrhythmias on a stress test.

• High-risk patients should be in medically supervised exercise programs. They are defined as patients with ischemia or serious arrhythmias on a stress test.

• Under utilization of cardiac rehabilitation.
Treating the Whole Patient

• **Depression**
  – Approximately 1 in 5 patients hospitalized with MI have major depression. There is also evidence that depression continues for several months after discharge (Fihn et al., 2012; Bush et al., 2005).
  – There is strong evidence that patients who are depressed post MI have a higher rate of mortality from both cardiac and non-cardiac causes (Bush et al., 2005).

• **Anxiety and Stress**
  – In post MI patients, interventions to reduce stress can reduce recurrent cardiac events by as much as 35-75% (Gibbons et al., 2002).

• **Social Support**
• **Role Identity**

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Our Vision:

*Practice with Joy. Positively impact every patient and family on their journey; provide safe passage, meet them where they are, connect with them in a meaningful way, and delivering care with wisdom and intention.*

- Cindy and Karen