Events during Diastole

Remember:
The cardiac muscle gets its perfusion during diastole.
Perfusion is determined by coronary perfusion pressure:
Coronary Perfusion Pressure = Diastolic BP – PCWP
Normal: 60 - 80 mmHg

Events during Systole

During systole the cardiac muscle has to overcome the pressures ahead of each chamber:
The right ventricle meets low to no pressure in the lungs.
The left ventricle must overcome the diastolic pressure in the aorta.

Hemodynamics

Cardiac Output = HR x Stroke Volume (SV)

Normal Cardiac Output:
4-8 liters / minute

Compensation for Decreased Cardiac Output

- ↓ cardiac output → ↑ heart rate.
- Heart rate is controlled by stimulation of both the sympathetic and parasympathetic nervous system.

Heart Rate
Cardiac Output
Preload
Afterload
Stroke Volume
Contractility
Muscle Synchrony
**Stroke Volume (SV)**

- **Stroke Volume** - the amount of volume ejected by the ventricle with each systolic contraction
- **OR**, amount of blood ejected by the heart with each beat
  - Normal: 60-130ml
  - \( SV = \frac{CO}{HR} \times 1000 \)

**Preload**

- The force on the ventricle during relaxation (diastole)
- Primary determinant is the **volume** of blood filling the ventricle
- **Right Ventricle** = RVEDP (Right heart preload):
  - Right Atrial Pressure (RAP); CVP
  - Normal values: 2-6mmHg
- **Left Ventricle** = LVEDP (Left heart preload):
  - PCWP, PAWP, or PAOP
  - Pulmonary Artery Diastolic (PAD)
  - Left Atrial Pressure (LAP)
  - Normal values: 8-12mmHg

**Afterload**

- **Afterload** — the ventricular force or pressure required to overcome impedance to ejection.
- As impedance \( \uparrow \), ejection velocity and SV \( \downarrow \), while ventricular workload & O2 consumption \( \uparrow \)
- **Systemic vascular resistance (SVR)**: Left
  - Normal SVR: 900-1400 dynes /sec/cm\(^5\)
- **Pulmonary vascular resistance (PVR)**: Right
  - Normal PVR: 50-250 dynes /sec/cm\(^5\)

**Contractility**

- Defined as the squeezing force generated by the ventricles.
- Refers to both the pressures of this ejection and the amount of blood ejected.
- Manipulate with **inotropic** drugs

**Cardiac Index**

- Cardiac Index — a more precise expression of CO, which takes into account the patient’s size
  - Normal CI = 2.5-4.2 L/min
  - CI < 2.0 is considered **cardiogenic shock**
  - 1.8-2.2 is low perfusion

- Calculate:
  - \( CI = \frac{CO}{BSA} \)

**Supply & Demand**

- **Supply & Demand**
  - Coronary artery patency
  - Diastolic filling time
  - Diastolic pressure
  - Hemoglobin
  - Arterial oxygen saturation
  - Oxygen extraction by the tissue
- **Demand**
  - Heart rate
  - Preload
  - Afterload
  - Contractility

**Best global measure is CO**

**Normal ejection fraction is 50 – 70 %**
**SVO₂**
- SVO₂ – amount of oxygen in the mixed venous blood in the pulmonary artery
- Normal SVO₂: 70-75% (range 60-80%)
- An estimate of the amount of oxygen returning to the cardiopulmonary circulation
- Reflects the patient’s ability to balance O₂ supply and demand at the tissue level

**Factors of SvO₂ & ScvO₂**

**Question**
Which of the following is a normal compensatory response to a decrease in cardiac output?
- a. Increased oxygen delivery
- b. Decreased oxygen consumption
- c. Increased oxygen extraction
- d. Decreased serum lactate

**Cardiac Assessment**
- Hemodynamic Monitoring
- ECG Interpretation
- Heart Sounds

**Arterial Waveform**
- Systolic Ejection (A)
  - Peak of waveform
  - Normal 90-140
- Diastole (C)
  - Lowest portion of waveform
  - Normal 60-90
- Dicrotic Notch (B)
  - Closure of Aortic valve
Peripheral Arterial Waveform

Review: Right Atrial Waveform
- The PA catheter is threaded manually to the right atrium. A continuous pressure reading will demonstrate a CVP/RA waveform

Normal RA: 2-6 mmHg

Preload
- What Decreases:
  - Hypovolemia
  - Position Change
  - Vasodilation
  - Right Heart Damage
  - Atrial Arrhythmias
  - Pericardial Effusion
  - PEEP
  - Tension Pneumothorax
- What Increases:
  - Vasoconstriction
  - ↑ fluid volume
  - ↑ ventricular filling time
  - Bradycardia

CVP waveform
- Pressure measurement is taken from the a waveform (mean) at end-expiration
- Normal RA/CVP: 2-6 mmHg

Right Ventricular Waveform
- The RV is very irritable and ectopy is a potential complication during PA catheter insertion
- Watch the monitor closely for Ventricular Tachycardia

Normal RV:
Sys: 15-30
Dia: 2-6 mmHg

Pulmonary Artery Waveform
- As the PA catheter floats through the pulmonic valve and into pulmonary circulation, the pulmonary systolic pressure remains similar to RV systolic pressure
- The catheter should stay here during continuous monitoring.

Normal PA:
Sys: 20-30 mmHg
Dia: 5-10
Mean: 10-20
Pulmonary Capillary Wedge Pressure

- Advance until it becomes lodged in a pulmonary artery slightly smaller at than the inflated balloon.
- No blood flows distal to the catheter tip.
- This pressure, the pulmonary artery occlusion pressure (POAP), reflects LV pressure when the mitral valve is open.
- Resembles CVP waveform. The a wave falls later in the T-P cycle.

**Normal PCWP:** 4 – 12 mmHg

The PCWP should be 1-4 mmHg **lower than** the PAD. IT SHOULD NEVER BE HIGHER!

Phlebostatic axis

- Head of the bed can range from flat to 60 degrees
- PA pressures may be significantly different in patients in a lateral position.
- Allow 5 minutes for stabilization after changing the patient’s position

Technical Factors: Effect of Patient Respirations

- If the patient is on a mechanical ventilator, the positive pressure "pushes up" the PA tracing. “Ventilator Valleys”
  - A PEEP > 10 will artificially elevate PA pressures
- If patient is breathing spontaneously, the negative pressure “pulls down” the PA tracing. “Spontaneous sky”
- The most accurate reading is obtained at respiratory end expiration.

Technical Factor: Mechanical Ventilation

Afterload

- Normal SVR = 900-1400 dynes/sec/cm³
  \[ SVR = \frac{MAP - CVP}{CO} \times 80 \]
- MAP = systolic BP + (2) diastolic BP
  \[ \frac{3}{CO} \]
  - Example:
    - BP = 120/80 (93)
    - CVP = 5
    - CO = 5
    - SVR = [(93 - 5) / 5] x 80 = 1406
Pulmonary Vascular Resistance (PVR)

- Pulmonary Vascular Resistance (PVR) reflects blood flow through the pulmonary circulation.
- The resistance is influenced by the pulmonary capillaries & arteries.
- Normal PVR = 50-250 dynes/sec/cm
  
  \[ PVR = \frac{MPAP - PCWP}{80} \]

ECG Monitoring

Treatment of Significant Arrhythmias

Conduction System

Electrical System of the Heart

Refractory Period

KEY CONCEPT: An electrical stimulus landing on the T wave, may cause disorganized ventricular contractions OR VENTRICULAR FIBRILLATION.

Premature Ventricular Contractions

The complexes have a QRS ≥ .12 seconds.

Significance:

- PVCs can occur in healthy persons with normal hearts and no apparent cause.
- Patients can be asymptomatic or feel "racing heart" / skipped beats.
- Frequent PVCs increase risk of fatal arrhythmias × 5.
- Treat: More than 6/min, multi-focal, R on T configuration

Question

The cardiac monitor shows the rhythm below for your patient. Which of the following medications might the physician order?

- a. Atropine
- b. Adenocard
- c. Cardizem
- d. Amiodarone
Amiodarone

- **Dosage:**
  - Non-VT/V.fib: 150 mg IV over 10 min.
  - Pulseless VT/V.fib: 300mg IV bolus

- **Adverse effects:**
  - Hypotension and bradycardia are common during initial bolus.
  - May be prevented by slowing the rate of infusion.

A patient with which of the following is at greatest risk for torsades de pointes?

- a. Depressed ST segment
- b. Tall, tented T waves
- c. Prolonged QT interval
- d. u-wave

Second Line Drug: Lidocaine

- **Administration:**
  - Bolus: 1.0-1.5 mg/kg IVP; may repeat in 5-10 minutes to max of 3mg/kg
  - Infusion: 1-4 mg/min

- Does not prolong QT

- **Adverse effects:**
  - Confusion (most common), seizures, tremors.

Too Fast

Sinus Tachycardia

- **Rate:** 100-150 bpm
- **Significance:**
  - If very fast, the heart cannot refill & results in ↓CO

  **TREAT THE CAUSE!**

Supraventricular Tachycardia

- **Characteristics:**
  - Rate: Rapid! Usually 160-250. May start / stop abruptly.
  - QRS is normal looking.
  - No bizarre, early, or late beats.

- **Too fast to see a P wave.**
- **Significance:**
  - Must treat if prolonged

This tachycardia originates above the ventricles, but below the SA node.
SVT Treatment

Try VAGAL maneuvers. If not effective, administer:

**ADENOSINE**
Indicated for stable SVT unresponsive to vagal maneuvers.
Dose: 6 mg IV PUSH
12 mg IV PUSH
12 mg IV PUSH

- Adenosine depresses sinus & AV node activity.
- HALF LIFE: 10 seconds
- Not effective in ventricular rhythms.

Atrial Flutter

**Significance:**
- Consider this a hazardous rhythm because it can suddenly change to a rapid ventricular response.
- If patient is stable, no initial treatment.
- If ventricular rate is rapid, treatment is required.

Atrial Flutter

**Significance:**
- Consider this a hazardous rhythm because it can suddenly change to a rapid ventricular response.
- If patient is stable, no initial treatment.
- If ventricular rate is rapid, treatment is required.

Atrial Fibrillation

R-R interval is **always** irregular

**Significance:**
- If stable or chronic may be tolerated.
- If patient has symptoms, treatment will be required.
- Consider hazardous because ventricular rate can suddenly ↑.
- Also lose atrial “kick”, which is 20% of the CO

Irregular Narrow Complex Tachycardia: Control of Rate

- Rate can be controlled by:
  - Beta Blockers
  - Calcium Channel Blockers
  - Amiodarone *

*Not considered a first line agent for narrow complex tachycardias

To Control Rate, Use Selective β-Blockers

- **Non-Selective:**
  - Propranolol (Inderal)
  - Nadolol (Corgard)

- **Selective agents:**
  - Atenolol (Tenormin)
  - Betaxolol (Zebeta)
  - Metoprolol (Lopressor)

- **Vasodilatory, Non-selective**
  - Labetalol (Normodyne)
  - Carvedilol (Coreg)

- **Shortest half-life:**
  - Esmolol

Question

A 69-year-old patient presents to the ED with complaints of palpitations and irregular heart beats for the last three of days. The cardiac monitor shows atrial fibrillation, a heart rate of 136 beats/min. His blood pressure is 124/76 mm Hg. Which of the following medications would the physician likely order?

- a. Lidocaine
- b. Cardizem
- c. Corvert
- d. Adenocard
To Control Rate: Use Calcium Channel Blockers

- Slows AV node conduction & prolongs AV nodal refractoriness
- Example: Diltiazem
- Do not use in:
  - Drug-induced tachycardia
  - Heart blocks
  - Concurrent use of Beta blockers.

Ventricular Tachycardia
Wide & bizarre QRS (≥ .12 sec)

Significance:
- Treatment is REQUIRED
- Pulse or NO Pulse?
- Pulse & STABLE? Use AMIODARONE
- Pulse & UNSTABLE ➔ ELECTRICAL CARDIOVERSION

Question

The nurse should perform which of the following interventions for a patient with chest pain, hypotension, and tachycardia at a rate of 180 beats/min?

a. Administer amiodarone 150mg IV over 10 min
b. Administer adenosine 6 mg rapid IVP
c. Perform synchronized cardioversion
d. Defibrillate with 300 joules

Electrical Cardioversion

Immediate electrical cardioversion is indicated for a patient with serious signs & symptoms related to tachycardia.

Synchronized Cardioversion: Energy Selection

- Start with 100 joules.

- Push the Synch button!

Synchronized Cardioversion: Energy Selection

- If the rhythm does not change, recharge to 200 joules & repeat
- Repeat with 300 joules & 360 joules, if needed.

- Complications include:
  - Deterioration into ventricular fibrillation
  - Embolization of a thrombus
Synchronized Cardioversion: Pre-Medication
- For awake, alert patients who are hemodynamically stable, pre-medicate with both a **sedative** and a **analgesic**
  - Sedatives
    - Diazepam
    - Midazolam
    - Etomidate
  - Analgesics
    - Fentanyl
    - Morphine
    - Merperidine

Watch for apnea & hypoventilation after sedation. Frequent vital signs are required before & after cardioversion.

Sinus Bradycardia
- **Characteristics:**
  - HR > 60
  - All intervals within normal limits except rate
- **Significance:**
  - May be normal in healthy, young patient.
  - Treat symptomatic bradycardias!

Too Slow

3°Heart Block
- **Prevent to PACE!**
- New guidelines: chronotropic drips

Symptomatic Bradycardia
- **Treat Bradycardia with BRADE**
  - **Atropine:** 0.5 mg IV push
    Repeat every 3 – 5 min to total of 0.04 mg/kg (3 mg)
  - **Dopamine:** 2 to 10 mcg/kg/min
  - **Epinephrine gtt:** Start at 1 mcg/min and titrate to patient response

Temporary Pacemakers
- Use an external generator. 3 types:
  - Transcutaneous
  - Transvenous
  - Epicardial
Transcutaneous Pacemaker

Transvenous Pacemaker

Epicardial Pacemaker

Lead Placement

Temporary Generator

External Pacing

- Pacing produces an electrical artifact on the strip called a spike.
- QRS appears wide & bizarre (like a PVC)

Pacing Codes

VVI

- **Pacing** in Ventricle only
- **Sensing** in Ventricle only
- **Response** will be inhibited if it senses activity in the Ventricle

The NASPE/BPEG Generic (NBG) Code

<table>
<thead>
<tr>
<th>Position</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
<th>V</th>
</tr>
</thead>
<tbody>
<tr>
<td>Category</td>
<td>Chamber(s) Paced</td>
<td>Chamber(s) Sensed</td>
<td>Response to Sensing</td>
<td>Programmability Rate Modulation</td>
<td>Antiarrhythmia Function(s)</td>
</tr>
<tr>
<td>0 = None</td>
<td>A = Atrium</td>
<td>V = Ventricle</td>
<td>D = Dual (A+V)</td>
<td>O = None</td>
<td>P = Pacing</td>
</tr>
<tr>
<td>A = Atrium</td>
<td>V = Ventricle</td>
<td>I = Inhibited</td>
<td>D = Dual (T+I)</td>
<td>M = Multiprogrammable</td>
<td>S = Shock</td>
</tr>
<tr>
<td>V = Ventricle</td>
<td>V = Ventricle</td>
<td>D = Dual (A+V)</td>
<td>C = Communicating</td>
<td>R = Rate Modulation</td>
<td>D = Dual (P+S)</td>
</tr>
</tbody>
</table>

Note: Positions I through III are used exclusively for anti-bradyarrhythmia function

Sensitivity

- the number, sensitivity (more sensitive)
Troubleshooting

- Failure to capture
- Improper Sensing
- Loss of Output

Loss of Capture

Electrical stimuli delivered by the pacemaker does not initiate depolarization of the atria or ventricle

Undersensing

Failure of the pacemaker to sense intrinsic R-waves or intrinsic P-waves

Leads to OVER PACING

Undersensing: Causes

- Battery depletion
- Decreased QRS voltage
- Fusion beat
- Dislodged/fractured lead
- Inappropriate sensitivity setting

Undersensing

What to do:
Increase the sensitivity by lowering the number (lower the fence)
Oversensing

Inhibition of the pacemaker by events the pacemaker should ignore

Leads to UNDER PACING

What to do:
• Eliminate interference
• Adjust sensitivity: Make less sensitive by increasing the number (raise the fence)

No Output

Pacemaker fails to emit stimuli at the programmed intervals

• Battery depletion/pacemaker off
• Oversensing
• Faulty cable connection
• Dislodged/fractured lead

Ventricular Fibrillation

Significance:
Requires immediate defibrillation & CPR!

Defibrillation

Energy Requirements for adults:
• If using a biphasic defibrillator:
  • 150-200 joules initially
  • For second and subsequent shocks, use the same energy or higher
• If using a monophasic defibrillators:
  • Select a dose of 360 joules for all shocks
Epinephrine

Administer 1 mg (10 ml of 1:10,000) every 3–5 minutes

- Stimulation of alpha adrenergic receptors: ↑ peripheral vasoconstriction & ↑ coronary & cerebral blood flow
- Makes ventricular fibrillation more responsive to defibrillation

Epinephrine: Special considerations / Cautions

- May be given via ETT
- Instill 2 to 2.5 mg into ETT
- If given IV, be sure to flush with 20 ml of fluid or elevated arm

Vasopressin

- Can be substituted for the first or second dose of epinephrine
- Give 40 units IV bolus.

- Effects:
  - Coronary perfusion pressure
  - Vital organ blood flow
  - Cerebral oxygen delivery

Pulseless Electrical Activity

- Any organized rhythm that DOES NOT PRODUCE A PULSE.
- Characteristics: Variable
- Significance:
  - Immediate CPR is required.
  - Look for possible reversible causes.

Asystole

PULSELESS ARREST / ASYSOLE

- Continue CPR and administer:
  - Epinephrine 1 mg IV, every 3–5 minutes

- Atropine 1 mg IV, every 3–5 minutes (up to 3 doses) (OLD guideline)
**PULSELESS ARREST / ASYSALE**

No electrical therapy is indicated. Move quickly to identify reversible causes:

**Hs:**
- Hypothermia,
- Hypoxemia,
- Hypoglycemia,
- Hypovolemia,
- Hydrogen ions (acidosis),
- Hypo-hyperkalemia

**Ts:**
- Toxins,
- Tamponade,
- Tension pneumothorax,
- Thrombosis (cardiac and pulmonary),
- Trauma
- Tablet (overdose)

**Therapeutic Hypothermia**

- Recommended for witnessed pulseless VT & VF arrests
- May be beneficial in PEA/Asystole arrests
- Cool to 32-34°C for 12-24 hours
- Prevent shivering

**Potential complications**
- Infection
- Bradycardia
- Electrolyte imbalances
  - Potassium
  - Calcium
  - Phosphorus
  - Magnesium
  - Hyperglycemia

**Heart Sounds**

- **S3:** ventricular gallop
  - Normal in children
  - Adults: ↑ LVEDP (preload); LV failure
- **Summation gallop**
  - S4, S1, S2, S3
  - CHF, anemia, ischemic hearts

**Systolic murmurs**
- Aortic stenosis
- Pulmonic stenosis
- Mitral insufficiency
- Tricuspid insufficiency

**Diastolic murmurs**
- Aortic insufficiency
- Pulmonic insufficiency
- Mitral stenosis
- Tricuspid stenosis

**Acute Coronary Syndromes**

**Risk Factors**

- Cigarette smoking
- Hypertension
  - SBP > 160 mm Hg
  - DBP > 95 mm Hg
- Hyperlipidemia
  - Total chol >240 mg/dL
  - HDL <35 mg/dL
- Obesity
- Diabetes Mellitus
- Gender/Age
- Sedentary lifestyle
- Family history
Classification of Infarct

- **STEMI**: ST segment elevation
- **Non-STEMI**: Non-ST segment elevation infarct
- The goal is to open the artery to restore blood flow.
- **TIME IS MUSCLE**
  - Want to treat within 12 hours
  - Late restoration of patent artery still increases survival

Coronary Arteries

- Right coronary (RCA)
- Left coronary (LCA)
- Left anterior descending (LAD)
- Circumflex (CF)

Supplies blood to:

- Right Coronary Artery
  - SA node - 55% hearts
  - AV node - 90% hearts
  - RA & RV muscle
  - Bundle of His
  - Inferior-posterior wall of LV
  - 1/3 septum, posterior fascicle of left bundle branch
  - Inferior surface RV

- Left Anterior Descending Coronary Artery
  - Anterior/lateral surface LV
  - Anterior 2/3 intraventricular septum
  - R bundle branch
  - Anterior L bundle branch
  - LV papillary muscle

Left Circumflex

Supplies blood to:

- AV node - 10% hearts
- SA node in 45% hearts
- Lateral posterior surface of LV
- Portion of posterior wall

Ischemia: T wave Changes

- The normal T wave has the following characteristics:
  - Asymmetry
  - The T wave is normally positive in leads I, II, V3 to V6
  - The T wave is less than 2/3 the height of the preceding R wave.
Abnormal T Waves: Ischemia

- If a T wave is flipped and symmetrical, it suggests ischemia.

Abnormal T Waves: Potassium

- T waves that are tall and peaked suggest elevated potassium levels.

The ST segment

- The key thing to identify with the ST segment is its relationship to the baseline.
- The normal ST segment is isoelectric or level with the baseline.

Abnormal ST Segments: Ischemia

- ST depression suggests ischemia in the area viewed by that lead.
- ST depression is considered significant if:
  - Depression > 1 mm below the baseline
  - Depression is seen in 2 or more leads facing the same area of the heart

ST Depression occurs in < 20 minutes of ischemia.

ST Segment Elevation: Injury

- ST elevation is considered significant if:
  - Elevation is > 1 mm in the limb leads or > 2 mm in the precordial leads
  - Elevation occurs in two or more leads facing the same area of the heart

ST elevation occurs within 20-40 minutes after injury occurs.
**Q Waves: Infarction**

- Pathological Q wave represents an area of dead tissue or infarction.
  - The Q wave is > 0.04 seconds wide
  - The Q wave is \( \frac{1}{4} \) of the height of the R wave that follows.

- Q waves usually take up 24 hours to develop.
  - They do not reveal when the infarction occurred.
  - Early development of Q waves predicts a large infarction.

**Pathologic Q Waves**

- CPK – 3 isoenzymes
  - CPK-MB is more specific to myocardium. Normal values:
    - CK (Total) - Males < 180, Females <150
    - CK MB Mass - < 8.0
    - CK Relative Index - < 4.0
    - Can be falsely elevated in renal failure, skeletal muscle injury, marathon runners
  - Elevations do not occur for up to 6 hours after injury.

- Troponin I
  - Elevations start within 3 hours after ischemic event.
  - Normal value: < 0.6
  - Remains elevated longer than CPK-MB
  - Highly sensitive markers for cardiac injury

**Case Study**


<table>
<thead>
<tr>
<th>Biomarker</th>
<th>1230</th>
<th>1900</th>
<th>0445</th>
</tr>
</thead>
<tbody>
<tr>
<td>Troponin I</td>
<td>&lt;0.04</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CK</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CK-MB</td>
<td>2.8</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Risk Stratification**

- **Initial Interventions**
  - Targeted history
  - Oxygen, Monitor, IV (Oh, MY!)
  - 12 lead ECG
  - Vital signs
  - Serum Markers
  - MONA

**Goal:**
- Door to Drug: 30 minutes
- Door to Balloon: 90 minutes
Using the 12 Lead ECG: Four Column Layout

- 12 Lead ECG machines layout the various leads in 4 standard columns:

<table>
<thead>
<tr>
<th>I</th>
<th>Lateral</th>
<th>AVR None</th>
<th>V1 Septal</th>
<th>V4 Anterior</th>
</tr>
</thead>
<tbody>
<tr>
<td>II</td>
<td>Inferior</td>
<td>AVL Lateral</td>
<td>V2 Septal</td>
<td>V5 Lateral</td>
</tr>
<tr>
<td>III</td>
<td>Inferior</td>
<td>AVF Inferior</td>
<td>V3 Anterior</td>
<td>V6 Lateral</td>
</tr>
</tbody>
</table>

Using the 12 Lead ECG: Localization of Problems

<table>
<thead>
<tr>
<th>Affected Part</th>
<th>Vessel(s) Involved</th>
<th>Leads</th>
<th>Electrical Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior</td>
<td>LAD</td>
<td>V3, V4</td>
<td>PVC, SVT, 2° AV Mobitz II, CHB, BBB, Hemiblocks</td>
</tr>
<tr>
<td>Inferior</td>
<td>RCA</td>
<td>II, III, avF</td>
<td>PVC, SB, ST, JR, PMC, Atrial Fib, 2° AV Mobitz II (Wenckebach)</td>
</tr>
<tr>
<td>Lateral</td>
<td>Circumflex</td>
<td>V5, V6, I, avL</td>
<td>PVC, SB, ST, PAC, A. Fib</td>
</tr>
<tr>
<td>Septal</td>
<td>LAD</td>
<td>V1, V2</td>
<td>Same as Anterior</td>
</tr>
<tr>
<td>Posterior</td>
<td>Circumflex, RCA</td>
<td>Reciprocal changes, V1, V2</td>
<td>Same as Inferior</td>
</tr>
<tr>
<td>Right Ventricular</td>
<td>RCA</td>
<td>V4R</td>
<td></td>
</tr>
</tbody>
</table>

12 Lead ECG Layout

Note: This one has 2 leads of rhythm strips

Localizing Injury

TREATMENT
**Treatment**

<table>
<thead>
<tr>
<th>STEMI</th>
<th>Non-STEMI</th>
<th>Non-Diagnostic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time from onset &lt;12 hr?</td>
<td>NO</td>
<td>Admit to monitored bed</td>
</tr>
</tbody>
</table>

**Reperfusion:**
- Door to needle: < 30 min
- Door to balloon: < 90 min

- NTG
- Beta Blockers
- ACE Inhibitors
- Statin Therapy
- Heparin
- Clopidogrel

**Early Invasive Therapy within 48 hr of AMI**

**MONA Greets Them at the Door**

- **Oxygen:**
  - Continue for at least initial 6 hours, then DC if oxygen saturation is >90%.
  - Nitroglycerin: Dilates coronary arteries to increase blood flow to heart.
  - ↓ preload by venous dilation. Give:

- **Aspirin:** Inhibits platelet aggregation.
  - Give 162-325 mg as soon as patient arrives in ED.
  - CHEW.

- **NTG**
- Beta Blockers
- ACE Inhibitors
- Statin Therapy
- Heparin
- Clopidogrel

**Adjuvant Medications**

- β receptor blocking agents
- Clopidogrel (Plavix); Prasugrel (Effient)
- Heparin, Lovenox
- ACE Inhibitors & ARBs
- Statins

**Re-Perfusion Strategy: Fibrinolytics**

- Breaks up the fibrin network that binds clots together

**Indications:**
- ST elevation in 2 or more leads
- Onset of symptoms < 12 hours
- MAY CAUSE DEATH FROM BRAIN HEMORRHAGE

**Agents available:** alteplase (tPA, Activase), reteplase (Retavase), streptokinase (Streptase), tenecteplase (TNKase)

**Question**

Which of the following lab results should be reported to the cardiologist for a patient with ACS who is scheduled to go for cardiac catheterization & possible PCI?

- a. aPTT 65 sec
- b. Troponin 0.2ng/ml
- c. Serum K+ 4.9 mEq/L
- d. Serum creatinine 2.3 mg/dL

If a Blockage is found:

Multiple options are available

- Balloon angioplasty
- Coronary atherectomy
- Stent placement
- Laser

**Early Invasive Therapy within 48 hr of AMI**
β – Blockers
• Mechanism of action:
  • Blocks catecholamines from binding to β-adrenergic receptors.
  • ↓ heart rate, blood pressure, and myocardial contractility
  • ↓ AV nodal conduction
  • ↓ incidence of primary ventricular fibrillation
• Absolute Contraindications
  • Severe CHF
  • SBP < 100 mm HG
  • Acute asthma
  • 2nd or 3rd degree heart block
• Cautions
  • Mild / moderate CHF
  • Heart rate <60
  • History of asthma
  • Insulin dependent diabetes
  • Severe Peripheral Vascular Disease

Heparin vs. Enoxaparin (Lovenox)
• Unfractionated heparin
  • Given as a continuous infusion
  • Keep PTT within target range of 50 to 70 (1.5 to 2x baseline)
  • Reversal agent: Protamine sulfate
• Low molecular weight- Enoxaparin
  • Given as an injection every 12 hours
• DO NOT administer either within 6 hours of non-specific fibrinolytic

Medications to Prevent Platelet Aggregation
• Glycoprotein IIB/IIIC Inhibitors
  • (Reopro, Aggrastat, Integrelin)
  • Inhibit platelet aggregation
  • Shown to decrease mortality & should be used in all patients with non-STEMI as soon as possible in conjunction with aspirin, heparin, clopidogrel, and early PCI
• Clopidogrel (Plavix):
  • Irreversible inhibition of platelet aggregates
  • Start 300 mg dose at the time of reperfusion, continue 75 mg daily dose for 8 days.
  • May need to withhold for 5 – 7 days prior to CABG

ACE Inhibitors
• Mechanism of action
  • Reduces BP by inhibiting angiotensin-converting enzyme
  • Alters left ventricular remodeling that occurs post MI by inhibiting tissue ACE
  • Lowers peripheral vascular resistance by vasodilation
  • Reduces mortality and CHF after an acute MI

RAS & ACE Inhibitors
• Decreased renal blood flow
• JG cells stimulated
• Renin released
• Reaction with angiotensinogen
• Increased blood vol., Na+, H₂O retention
• Aldosterone secretion
• Angiotensin I then II production
• Improved renal blood flow
• Increased arterial blood pressure
• Vasoconstriction
Statins
- ↓ complications such as reinfarction, recurrent angina, and arrhythmias
- ↓ both total cholesterol and LDL.
- Stabilize platelets, improve endothelial function, & inhibit inflammatory response.
- Should be started within 24 hours

MI Complications
- Dysrhythmias most common complication
- ½ of the deaths occur within 1st hour of symptoms -- usually due to v. fib
- Heart failure – Can range from mild dysfunction to CHF to cardiogenic shock.
- RV infarction: occurs with inferior / posterior MI. Incidence around 40%
- Patients with RV infarct need VOLUME & are very sensitive to NTG
- Emboli: Thrombi form on inner wall at site of MI. Most common with large anterior MI.
- Papillary muscle rupture

Question
Your MI patient suddenly develops ↓ LOC, a weak, thready pulse, and bilateral posterior crackles in the lower lung fields. VS: 78/46; HR 139; RR 25 u/o < 30 ml for the last hour O2 sat 89% on 4L NC
You suspect
a. Stroke
b. ARDS
c. Pulmonary embolus
d. Cardiogenic shock

Assistance to the Failing Ventricle: Intra Aortic Balloon Counterpulsation

IABP Placement
- When the IABP balloon is properly placed, it is just distal to the left subclavian artery.
- If the IABP balloon is displaced proximally, it can occlude the take-off of the left subclavian artery
- If the IABP balloon is displaced distally, it can occlude the take-off of the renal arteries

IABP- How does it work?
- Inflation during diastole
  - ↑ Aortic root pressure
  - ↑ Aortic diastolic pressure
  - ↑ Coronary perfusion pressure
  - CPP = Aortic diastolic pressure – myocardial wall tension
  - ↑ Oxygen supply
  - ↑ Stroke volume = ↓ Heart rate
IABP- How does it work?

- Deflation immediately prior to systole
  - ↓ Aortic end-diastolic pressure
  - ↓ Impedance to ejection
  - ↓ Afterload
  - ↓ Oxygen demand

Primary Effect of IABP Therapy

Supply

Demand

- Supply – Balloon Inflation
- Demand – Balloon Deflation

Indications

1. Cardiogenic shock
2. Refractory ventricular failure
3. Unstable refractory angina / impending infarction
4. Mechanical complications due to acute myocardial infarction
5. Ischemia related to intractable ventricular arrhythmias
6. Cardiac support for high risk general surgical and coronary angiography / angioplasty patients
7. Septic shock
8. Weaning from cardiopulmonary bypass
9. Intra-operative pulsatile flow generation
10. Support for failed angioplasty and valvuloplasty

Contraindications

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

Question

An IABP is used to manage cardiogenic shock in order to increase

a. Coronary perfusion during systole
b. Myocardial oxygen supply
c. LV filling volume
d. LV systolic pressure

Congestive Heart Failure: Definition

- Systolic failure: ↓ cardiac contractility
- Diastolic failure: impaired cardiac relaxation and abnormal ventricular filling.
- The heart is unable to generate a cardiac output sufficient to meet the demands of the body.
- Manifested by:
  - Poor tissue perfusion and congestion of the vascular beds
  - Symptoms of inadequate tissue perfusion
Pathophysiology: Systolic Dysfunction

- Impaired LV contractility results in reduced ejection fraction (EF < 40%)
- Ventricle is dilated, thin walled
- Etiology:
  - Acute Myocardial Infarction
  - Coronary Artery Disease, Hypertension
  - Valvular Heart Disease
  - Toxins
  - Endocrine
  - Congenital

Dilated Heart (Systolic Failure)

Pathophysiology: Diastolic Dysfunction

- Normal systolic function with the impaired ability of the ventricle to relax (lusitropy) and fill with blood
- ↑ filling pressures result due to stiff ventricles
- Ventricle is thickened and concentrically hypertrophied
- Etiology:
  - Hypertension, coronary artery disease
  - Cardiomyopathy
  - Aortic Stenosis
  - Infiltrative diseases
    - Sarcoidosis
    - Amyloidosis

Hypertrophic Heart (Diastolic Failure)

Chronic Heart Failure: Pathophysiology

- Myocardial Injury — Fall in LV Performance
- Activation of RAAS, SNS
- Myocardial Toxicity
- Peripheral vasoconstriction
- Hemodynamic alterations
- Remodeling and progressive worsening LV function
- Heart Failure Symptoms
- Morbidity Mortality

Left Ventricular Failure

- Signs of Decreased CO
  - Fatigue, poor exercise tolerance
  - Tachycardia
  - Narrow pulse pressure
  - Cool, pale, diaphoretic
  - Altered mental status
  - Decreased urine output

Right Ventricular Failure

- Dependent edema
- RAP/CVP ↑
- Fluid vol. Backs into portal system
- GI s/s
- Ascites
- Nocturia
- Weakness, fatigue
- Murmur of TR

Signs of Elevated Pulmonary Pressure

- Dyspnea, tachypnea
- Orthopnea
- Cough, wheeze
- Hypoxia
- Respiratory Alkalosis
- Crackles, rhonchi
  - S₃, S₄

What kind of heart failure does this man have— Right or Left?
Classification of Heart Disease

<table>
<thead>
<tr>
<th>ACC/AHA Heart Failure Stage</th>
<th>NYHA Functional Class</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>At high risk for heart failure, but no symptoms or structural heart disease</td>
</tr>
<tr>
<td>B</td>
<td>Structural heart disease, but no symptoms of heart failure</td>
</tr>
<tr>
<td>C</td>
<td>Structural heart disease with prior or current symptoms of heart failure</td>
</tr>
<tr>
<td>D</td>
<td>Refractory heart failure requiring specialized interventions</td>
</tr>
</tbody>
</table>

Compensatory Mechanisms

- In the short term, compensatory mechanisms maintain cardiac output.
- In the long term, all of these factors trigger a process of pathologic growth and remodeling.
  - Include:
    - Stimulation of SNS
    - Activation of Renin-Angiotensin-Aldosterone System
    - Hypertrophy

VENTRICULAR REMODELING

- Decrease contractility → decreased stroke volume → Activation of SNS & RAAS
- Increased ventricular volumes
- Increased ventricular pressures
- Increased ventricular wall tension
- Thinning of ventricle wall & chamber dilation
- Fibrous tissue deposits

Myocardial Hypertrophy

- Late compensatory mechanism
- Larger cells less contractile & require more oxygen
- Ischemia leads to fibrosis and necrosis

Pillars of Treatment:
Drug Therapy using the A-B-C-Ds

- **ACE inhibitors**: Block RAAS
- **Beta blockers**: Block SNS
- **↑ Contractility**: Digitalis
- **Diuretics, Vasodilators**: 
  - ↓ Preload & ↓ Afterload

Pharmacologic

**Systolic Treatment**
- Loop diuretics
- Spironolactone
- Beta Blockers
- ACE inhibitors
- Digoxin

**Diastolic Treatment**
- Diuretics
- Beta Blockers
- ACE inhibitors
- Calcium Channel Blockers
Angiotensin II Receptor Antagonists: ARBs

- Indicated for patients who cannot tolerate ACE Inhibitors.
- Act directly on angiotensin receptors. Increased selectivity & specificity by directly blocking angiotensin receptors
  - Example: Losartan
  - ↓ both preload and afterload
  - Fewer side effects

Beta Blocker Selection

- Beta blockers recommended for heart failure include:
  - Carvedilol (a non-selective agent)
  - Metoprolol: Long acting
  - Bisoprolol
  - Doses may need to be titrated gradually over a period of several weeks.

Preload: Diuretics

GOAL: To restore fluid balance

- Beneficial effects
  - Excrete sodium and water
  - Decrease CVP
  - Symptom relief
  - Decrease shortness of breath & edema

- Detrimental effects
  - Electrolyte imbalances
  - Decrease renal function
  - Activates RAAS
  - Hypotension

Use Daily Weight For diuresis monitoring

Preload / Afterload: Vasodilators

- Vasodilators that target Preload:
  - Nitroglycerin
  - Morphine
- Vasodilators that target Afterload:
  - Nipride
  - NTG at > 50mcg/min

Contractility

- Digoxin: Used for inotropic support
- Dobutamine: Used short term
- Milrinone: Used for patients who develop tolerance to dobutamine

BNP: Brain Natriuretic Peptide

- Natriuretic peptides are a family of naturally occurring molecules.
- Actions:
  - ↓ cardiac preload by causing vasodilatation & ↑ vascular capacitance.
  - ↓ sympathetic tone.
  - Induce natriuresis (sodium excretion) by actions on renal vasculature and tubules.
**BNP Measurement**

- Measurement of BNP can differentiate patients with heart failure from patients without heart failure.
  - BNP level >100 is indicative of HF
  - May also be elevated in right sided failure due to pulmonary disease and end stage renal disease.
  - May see false negative in patients with flash pulmonary edema and heart failure due to mitral regurgitation.

**Natrecor (Nesiritide)**

- Synthetic natriuretic peptide (BNP)
- Approved for IV use in acute decompensated heart failure
  - ↓ pulmonary pressures
  - Improves dyspnea
  - Contraindicated with systolic BP < 90
  - Monitor closely for hypotension and arrhythmias

**Adjunct Therapies**

- All AT RISK patients should be on:
  - Beta blockers
  - ACE
  - Statins – Believed to decrease inflammatory response
- May also require:
  - Anticoagulants – If patient develops atrial fibrillation
  - Antiarrhythmics
  - Physical conditioning
- Consider:
  - Biventricular Pacing / Cardiac Re-synchronization
  - IABP
  - Assist Devices
  - Transplant

**Left Ventricular Assist Device**

**Heart Transplant**

**Question**

A patient status-post heart transplant develops second degree heart block, Type II. VS: 86/40; HR 44; RR 24. The patient c/o chest pain. Skin is warm and dry. Breath sounds clear. You should

a. Prepare for transcutaneously pacing
b. Draw labs to determine if patient is rejecting the heart
c. Administer 0.5 mg atropine
d. Reassure the patient that what he is experiencing is normal after a heart transplant
Acute rejection in cardiac transplantation is diagnosed by:

a. ECG  
b. Chest x-ray  
c. Echocardiography  
d. Endomyocardial biopsy

Acute Inflammatory Diseases

- Myocarditis
- Endocarditis
- Pericarditis

Myocarditis

- Inflammation of the myocardium
- Etiology: viral, bacterial, parasitic, fungal, radiation to chest
- Acute or chronic
- S/S: chest soreness, burning; ↑ with inspiration; syncope; fever; S₃, S₄;
- Dx: ↑ WBC; ↑ CK-MB; Cardiomegaly; diffuse ST & T wave abnormalities; SVT or ventricular dysrhythmias

Question

Which of the following medication regimens would be most appropriate to relieve chest pain in a patient with diagnosis of myocarditis?

a. NTG 1/150 grains sublingual  
b. Furosemide 40 mg IV  
c. Ibuprofen 800 mg PO  
d. Morphine sulphate 2 mg IV

Infective Endocarditis

- Definition: Inflammation of the endocardium, usually occurring in the membranous lining of the heart valves
- Etiology:
  - Congenital or acquired heart disease
  - Invasive monitoring (PA catheters, temporary pacing wires, etc)
  - Cardiac surgery
  - IV catheters / dialysis access devices
  - GI / GU tubes (Foley cath)
  - Dental procedures / poor oral hygiene

Question

The valve most often effected by infective endocarditis is:

a. Mitral  
b. Aortic  
c. Tricuspid  
d. Pulmonary
Infective Endocarditis:
Presentation:
- Infectious symptoms (fever, chills, malaise)
- Fatigue, night sweats, anorexia, weight loss
- Symptoms of heart failure
- New murmur (aortic insufficiency most common)
- Vascular signs:
  - Splinter hemorrhages
  - Petechiae on chest, oral mucosa, abdomen
  - Janeway lesions: flat, erythematous lesion on palm, soles of feet
  - Roth's spots: round white lesions on retina
  - Osler's nodes: painful nodules on fingers, toes

Infectious Endocarditis:
Diagnosis:
- Blood cultures; TEE
Management:
- Prevention:
  - Patients with valve disease should receive antibiotics prior to any invasive procedure
  - Good hygiene
  - No street drugs
  - Decrease myocardial oxygen demand
  - Control and treat infection
  - Antibiotics for 6 – 8 weeks
  - Monitor for complications
  - Systemic embol
  - Valve surgery may be required

Pericarditis:
Definition:
Inflammatory process involving the visceral or parietal pericardium

Pericarditis:
Etiology:
- Idiopathic
- Post MI – May be acute (within 7 days) or Dressler's syndrome (occurs 2 wks or more later)
- Trauma
- Infection
- Radiation
- Drugs

Pericarditis:
Presentation:
- Sharp, stabbing pain that radiates to the left shoulder
- Aggravated by inspiration, supine position
- Relieved by sitting up or leaning forward
- Dyspnea, tachypnea
- Tachycardia
- Murmur / rub
- Fever
Diagnosis:
- Troponin elevated, ↑WBC
- Diffuse ST elevation in all leads

Pericarditis - Management:
- Management
  - Non-steroidal anti-inflammatory drugs
  - Treat cause
  - Monitor for complications:
    - TAMPOONADE
    - Dyshrhythmias
    - Anticipate pericardiocentesis or pericardial window

Cardiac Tamponade:
- If fluid accumulates in the pericardial sac, there is potential for development of cardiac tamponade.
- Tamponade has a constrictive effect on the heart. Must be treated urgently!
**Question**

The classic triad (Beck’s triad) of symptoms of cardiac tamponade are:
- a. Rales, muffled heart sounds, bradycardia
- b. Widened pulse pressure, atrial dysrhythmias
- c. Tachycardia, hypotension, narrow pulse pressure
- d. Hypertension, flushing, pulse paradoxus

**Hypertension – Definitions**

- **Hypertension**: Elevation in BP above 140 / 90 on at least 3 separate occasions
- **Hypertensive Urgency**: Develops over days to weeks. Characterized by ↑ diastolic BP, but are usually not associated with end organ damage.
- **Hypertensive Crisis**: Elevation severe enough to cause the threat of immediate vascular necrosis and end organ damage. Usually > 180 / 120 or MAP over 150.
  - Accelerated: DBP >120
  - Malignant: DBP > 140
- **Hypertensive Emergency**: Develops over hours to days. BP must be lowered within minutes to an hour to reduce potential complications or new / progressive end organ damage.
  - **Hypertensive Encephalopathy**: BP > 250/ 150

**Principles of Treatment**

\[ BP = CO \times PVR \]

- All antihypertensives must act by:
  - ↓ cardiac output
  - ↓ peripheral vascular resistance.

**Hypertensive Crisis**

- **Goal**: Reduce cardiac & renal mortality and morbidity
- **Usually require ICU admission**
- **Reduce BP gradually to maintain cerebral perfusion.**
- **In hypertensive emergency:**
  - Decrease MAP 25% within first hour
  - Decrease to 160/ 100 within next 2-6 hours, then gradual reduction over next 24 hours
- **Agents:**
  - Sodium nitroprusside, fenoldopam, hydralazine
  - Hydralazine good in eclampsia

**Cardiomyopathy**

- **Dilated**
- **Hypertrophic (Idiopathic hypertrophic subaortic stenosis [IHSS])**
- **Restrictive**
Cardiomyopathy: Dilated
- Myocardial fibers degenerate & fibrotic changes occur
- Systolic and diastolic dysfunction
- Mitral Insufficiency
- Ventricular rhythms common cause of death

Cardiomyopathy: Hypertrophic
- ↑ mass & thickening of myocardium
- Fibrosis occurs
- Ventricles rigid & stiff
- LV chamber ↓ in size
- LA dilates
- May develop outflow obstruction

Cardiomyopathy: Restrictive
- Restricted filling of ventricles
- Etiology: Idiopathic; Amyloidosis
- ↓ compliance; LV stiff & can not dilate or contract well in systole
- LVEDP ↑; contractility ↓; ↓ CO → HF → Death

Cardiomyopathy: Rx
- ACE inhibitors
- B-blockers
- Antidysrhythmic agents
- Antibiotics (at risk for infectious endocarditis)
- DCM: + inotropes; diuretics
- HOCM: Avoid agents that ↓ preload; avoid + inotropes (can worsen obstruction)

Coronary Artery Bypass Grafting
- The internal mammary artery is frequently used because of its long-term patency.
- Other conduits include:
  - Saphenous veins
  - Radial artery
  - Gastroepiploic artery
  - Inferior epigastric artery
- CABG remains the treatment of choice for patients with three vessel coronary artery disease, complex lesions, or more than 75% occlusion of the left main.
Question
Upon turning the postoperative CABG patient, the nurse observes 200 ml of bloody drainage in the CT drainage system. The nurse should perform which of the following?
- Autotransfuse the patient
- Aggressively strip the CT tubing
- Obtain lab for coagulation studies
- Continue to monitor the patient’s CT drainage

Upon arrival to the ICU post-CABG, the patient has the following:
- BP 180/110
- HR 70, 100% paced
- RAP 6; PAD 10
- CO 3.5
- U/O 60 ml

What should the nurse do first?
- Bolus with 250ml to ↑ RAP & PAD
- ↑ NTG gtt by 6.6 mcg/min to ↓ BP
- ↑ pacer to 75 to improve CO
- Administer propranolol 1 mg IV to ↓ BP

After a mitral valve replacement, the patient is most likely to have which of the following as part of the patient’s plan of care?
- Ace-inhibitor
- Beta-blocker
- Positive inotropic agent
- Anticoagulant

Valve Surgery
- The primary cause for valve disease is rheumatic fever.
- Most commonly affected the aortic and mitral valves
- If allowed to persist, congestive heart failure can occur.
- Surgical repair can include replacement of the defective valve with either a mechanical or tissue valve.
  - Patients with mechanical valves will require lifetime anticoagulation

Cardiac Trauma
- Cardiac contusion is the most common blunt injury to the heart.
  - Rarely fatal, but should be suspected if the patient has anterior chest wall trauma and fractures of the sternum and ribs.
  - Signs are similar to MI with ST changes and elevation of enzymes
  - ECG changes: RBBB; ST; ventricular dysrhythmias
  - Right ventricle is most often affected
- Aortic rupture is the result of a blunt trauma deceleration injury

Question
Following a motor-vehicle accident, pericardial tamponade is suspected. Which of the following findings is consistent with traumatic tamponade?
- Muffled heart sounds
- Pericardiocentesis of 50ml of blood
- ST-segment depression in the limb leads
- Rales on auscultation
Which of the following findings is most indicative of a leaking abdominal aortic aneurysm?

a. Back pain  
b. Bounding peripheral pulses  
c. Intermittent claudication  
d. Nausea and vomiting

Aortic Aneurysms

• Thoracic or Abdominal.  
  Abdominal are more common (65%)  
• Two patterns:  
  - Weakness and bulging of the entire vessel wall  
  - Weakness and bulging within the vessel wall  
• Patients with an aneurysm may be asymptomatic  
  - Rupture of the aneurysm causes acute pain that radiates to the back, unrelieved by changes in position.  
  - Mortality from rupture is 90%. The only chance for survival is emergent repair.

Aortic Dissection

• Tear in the intimal layer of the aorta allows blood to flow into the medial layer, creating a false channel.  
  - It is usually accompanied by hypertension that is not well controlled.  
  - Most common is ascending aorta dissection, with many occurring only centimeters from the aortic valve.  
  - High risk of death from acute aortic regurgitation and pericardial tamponade.  
• Symptoms: Severe “tearing” or “ripping” chest pain, moves to the back as dissection progresses

Question

Post-endovascular AAA repair, a patient has the following findings: BP 124/76; HR 92; RR 18. Distal pulses 2+, cap refill 2 sec. Groin without s/s of hematoma. U/O is 20ml/hr for 2 hours. The most likely cause of the decreased u/o is:

a. Renal toxic effects of contrast agents  
b. Occlusion of the renal artery by the endograft  
c. Hypovolemia due to operative blood loss  
d. Hypovolemia due to retroperitoneal bleeding

Question

On PO day 2 after an AAA, your patient develops hypotension, tachycardia, abdominal distention, and ↑ WBC. These findings indicate

a. Post-op graft infection  
b. Ischemic colitis  
c. Aortic-enteric fistula  
d. Abdominal compartment syndrome

Your patient has a history of hypertension and CAD. Assessment reveals ↓ skin temperature of legs & feet, ↓ pulses & pallor when the legs are elevated & reddish-blue discoloration of the legs. Patient c/o leg pain. You suspect

a. Peripheral arterial disease  
b. Chronic venous insufficiency  
c. Acute arterial occlusion  
d. Deep vein thrombosis
Acute Peripheral Vascular Insufficiency

- Acute Arterial Occlusion
- Peripheral Stents
- Medications: Antiplatelet; Thrombolytics
- Carotid Stenosis
- Endartarectomy

Question
After aorto-femoral bypass surgery for acute arterial occlusion, your patient has:
- CPK, K+ 5.9 mE/L
- PaO2 90
- PaCO2 24
- HCO3 19

This patient is at risk for developing:
- a. Dysrhythmias
- b. Graft occlusion
- c. Pulmonary embolus
- d. Heart Failure

Question
After femoral-popliteal bypass surgery, patient c/o pain unrelieved by narcotics. Dressing D & I. Skin warm, pale, & dry. Cap refill 2 sec. Distal pulses 2+. Motor strength slightly ↓. Gastrocnemius muscle slightly swollen & is “doughy” to palpation. These findings are consistent with:
- a. Compartment syndrome
- b. Graft occlusion
- c. Development of false aneurysm
- d. Heparin induced thrombocytopenia (HIT)

Question
A patient is receiving thrombolytics via continuous infusion for acute limb ischemia suddenly complains of pain in the affected extremity. The most appropriate nursing action includes:
- a. Administer narcotic pain medication to relieve the pain
- b. Elevate the affected extremity above the level of the heart
- c. Notify the MD that the patient may have compartment syndrome
- d. Discontinue the thrombolytic infusion and notify the MD

Structural Heart Defects

- Atrial septal defects
- Ventricular septal defects
- Valvular disease

Structural Heart Defects:
Atrial septal defects
- Left-to-right shunt resulting in right chamber overload
- RA, RV dilate
- Leads to pulmonary hypertension
- Dysrhythmias: Aflb, A.Flutter, PR prolongation, Incomplete RBBB. LAD

- Treatment:
  - Antibiotics to prevent endocarditis
  - Manage heart failure s/s
  - Surgical repair
Structural Heart Defects:
Ventricular Septal Defects
- Left-to-right shunt
- Pulmonary HTN
- S/S
  - Tachypnea
  - Sweating
  - Hemoptysis
  - Heart Failure
  - Holosystolic murmur
- Treatment
  - Antibiotics
  - Manage HF s/s
  - Surgical repair

Valvular Disease
- Mitral Regurgitation
- Mitral Stenosis
- Aortic Regurgitation
- Aortic Stenosis

Other Structural Defects
Coarctation of Aorta
- ↑ workload LA & LV
- Left-sided HF
- Tx
  - Ab
  - Tx HF
  - Surgical Closure
Patent Ductus Artiosus
- Narrowing of lumen & ↓ flow
- Tx
  - Ab
  - Antihypertensives
  - Surgery (1-5 yo)

Let’s Test Ourselves Some More!

Which of the following vasopressors is indicated for low CO syndrome when the desired effect is vasoconstriction without tachycardia?
- Norepinephrine (Levophed)
- Phenylephrine (Neosynephrine)
- Dopamine
- Epinephrine

Your patient c/o chest pain. The ECG shows ST elevation in Leads II, III, & aVF. You administer NTG 1/150 grains SL. The patient’s BP drops from 136/84 to 82/50 mmHg. The most likely cause of the decrease in BP is:
- Right ventricular MI
- Papillary muscle rupture
- Hypersensitivity to NTG
- Rupture of a ventricular free wall
Two hrs post-CABG, patient has these findings:
BP 80/62 (68)
CVP 15
PAD 14
CO 3
CT output 50ml/hr for 2 hrs.

These findings are consistent with:
- Normal post-op course
- Myocardial stunning
- Cardiac tamponade
- Perioperative MI

Your post-op patient has a HR 132, BP 80/66, PCWP 5, Hgb 8.
Which medication would help?
- Vasopressin 20 unit IVP
- Administer 1 unit of PRBC
- Administer 250ml NS bolus
- Start dopamine at 10mcg/kg/min

Which of the following medications will worsen the symptoms of hypertropic cardiomyopathy?
- Calcium channel blockers
- Amiodarone
- Beta Blockers
- Nitrates

Patient admitted to the ICU status post-cardiac arrest. Immediate management of this patient includes:
- Monitoring for hyperkalemia
- Maintaining a blood glucose 120-150mg/dL
- Initiating clinically induced hypothermia
- Monitoring labs for ↑ WBC & Plt counts

Coronary artery perfusion is dependent upon:
- Diastolic pressure
- Systolic pressure
- Afterload
- SVR

Chest pain that is NOT relieved by rest & NTG is called:
- Variant angina
- Stable angina
- Unstable angina
- Prinzmetal’s angina
The primary function of drug therapy with beta-blockers in heart failure is to:

a. Increase blood pressure
b. Block compensatory mechanisms
c. Increase urine output
d. Decrease dysrhythmias

A patient admitted with CHF has the following: BP 94/60, HR 125, RR 24, u/o 50 ml for 2 hours. Cool clammy & ↓ LOC. Which of the following would be included in the patient's plan of care?

a. Positive inotropes, diuretics, vasodilators
b. ACE-inhibitors, adenosine, beta-blockers
c. Beta-blockers, diuretics, calcium-channel blockers
d. Negative inotropes, digoxin, antidysrhythmics

A direct effect of sodium nitroprusside (Nipride) is to:

a. Decrease stroke volume
b. Increase venous return
c. Decrease afterload
d. Increase pulmonary vascular resistance

Which of the following medications best improves LV function?

a. Dopamine (Intropin)
b. Captopril (Capoten)
c. Digoxin (Lanoxin)
d. Procainamide (Pronestyl)

Which of the following findings is most consistent with high left ventricular filling pressure?

a. Sinus bradycardia
b. Diastolic murmur
c. Peripheral edema
d. Bibasilar crackles

Which of the following pulmonary artery catheter findings would be anticipated in a patient with chronic emphysema?

a. Increased RAP
b. Decreased RAP
c. Increased PCWP
d. Decreased PCWP
Which of the following is a contraindication to the use of an IABP?

a. Aortic valve insufficiency  
b. Mitral valve insufficiency  
c. Ventricular aneurysm  
d. Ventricular septal defect

Two hours after a permanent DDD pacemaker is inserted for SSS, the patient goes into atrial fibrillation. Which of the following pacing modes is indicated for this patient?

a. DVI  
b. DDD  
c. VAT  
d. VVI

In patients with angina, CCB are effective because they:

a. Inhibit vasodilation  
b. Increase myocardial contractility  
c. Increase venous return  
d. Reduce myocardial oxygen consumption

Your patient c/o chest pain that is sharp, constant, & worse when lying down. The pain lessens when sitting up & leaning forward. These symptoms are consistent with

a. Pulmonary embolism  
b. Abdominal aortic aneurysm  
c. Pericarditis  
d. Myocardial infarction

*New PCCN Cardiac Content

Starting 06-26-2013, content includes:

- Genetic Cardiac Disease
  - Long QT syndrome,
  - Brugada syndrome
- Minimally-invasive cardiac surgery (i.e., nonsternal approach)
- Vascular disease: minimally-invasive interventions (e.g., stents, endografts)
- Cardiomyopathies: stress-induced (e.g., Takotsubo)

Thanks

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Questions?

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