CCC Review
Cardiovascular System

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CCC– www.aacn.org

Exam Content
- Cardiovascular – 18%
- Pulmonary – 17%
- Endocrine / Hematology / GI / Renal / Integumentary – 20%
- Musculoskeletal / Neurology / Psychosocial – 13%
- Multisystem – 14%
- Professional Caring and Ethical Practice – 20%
Cardiovascular Section – 18%

- Acute coronary syndromes
- Acute MI
- Acute peripheral vascular insufficiency
- Acute pulmonary edema
- Arterial/venous occlusion
- Cardiac/vascular cath
- Cardiogenic shock
- Dysrhythmias
- Heart failure
- Hypertensive crisis
- Conduction defects
- Structural heart defects (valves, congenital)
- Ruptured/dissecting aneurysm
- Physical assessment
- Cardiac monitoring
- Rhythm interpretation
- Hemodynamic monitoring
- 12 lead ECG
- Cardioversion/defibrillation
- CVP/A-lines/PA catheter
- Cardiac pharmacology
- Cardiovascular emergencies
- IABP
- Coronary interventions

Basic Concepts You Need to Know

**Physiology**

**Pharmacology**

**Assessment**

To help you think your way through whatever questions are asked
**Definitions**

- **Cardiac Output**: Volume of blood ejected by the ventricle each minute
  - Normal: 4–8 liters/minute
- **Cardiac Index**: Adjustment made for body size
  - Normal cardiac index: 2.5–4 liters/minute/m²
- **Stroke Volume**: Volume of blood ejected with each beat.
  - Normal: 60–120 ml / beat
  - Systolic BP as non invasive indicator
- **Ejection Fraction**: Percent of blood ejected from the ventricle
  - Normal: 55% to 70%

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**Right side versus left side systems**

[Diagram showing blood circulation through the heart and lung system]

Anatomically the heart sits between the two lungs, but physiologically the lungs sit between the right heart and the left heart.
Determinants of Cardiac Output

\[ \text{CO} = \text{HR} \times \text{SV} \]

**Preload**
- Ventricular fiber length determined by volume.
  - RV assessed by CVP, JVD. LV assessed by PAOP, lung sounds.
  - Increased by volume
  - Decreased by diuretics & venous dilators (NTG, ACEI, ARBs)

**Afterload**
- Work that ventricle has to do to eject volume.
  - RV assessed by PVR, LV assessed by SVR.
  - Increased by SNS stimulation (compensatory), vaso-pressor drugs (norepinephrine, phenylephrine, dopamine, vasopressin)
  - Decreased by vasodilator drugs (nitroprusside, milrinone, ACEI, ARBs)

**Contractility**
- Efficiency of fiber shortening regardless of length. Evaluated by ejection fraction.
  - Increased by positive inotropic drugs (dopamine, dobutamine, epinephrine, norepinephrine)
  - Decreased by negative inotropic drugs (Ca++ blockers, beta blockers, others)

Conditions That Alter Preload

**Hypovolemia** (low preload)
- Hemorrhage
- Dehydration
- Burns
- Overdiuresis
- Third Spacing (low albumin, capillary leak secondary to CPB, ARDs)

**High Preload**
- Hypervolemia (overhydration)
- Heart failure
- Renal disease
- Pulmonary HTN
- Tamponade
- Tension pneumo

**Altered Size of Vascular Space** (relative hypovolemia)
- Sepsis
- Neurogenic shock
- Spinal or epidural anesthesia
- Anaphylaxis
- Rewarming after cardiac surgery
- Venous dilating drugs
  - NTG
  - ACEI, ARBs
  - Ca++ blockers
Conditions That Alter Afterload

➤ **Vasodilation**
  - Sepsis
  - Spinal or epidural anesthesia
  - Anaphylaxis
  - Rewarming after cardiac surgery
  - Arterial dilating drugs
    • Nipride
    • ACEI
    • ARBs
    • Milrinone
    • Ca++ channel blockers
    • Antihypertensives

➤ **Vasoconstriction**
  - Hypertension
  - SNS stimulation
  - Compensatory vasoconstriction (hypothermia, shock)
  - Drugs
    • Phenylephrine (Neosynephrine)
    • Norepinephrine (Levophed)
    • High-dose dopamine
    • Epinephrine
    • Vasopressin

Conditions That Alter Contractility

➤ **Increase**
  - Pheochromocytoma (adrenal medulla tumor)
  - Hyperthyroidism
  - Positive inotropic drugs
    • Dobutamine
    • Dopamine
    • Norepinephrine (Levophed)
    • Milrinone
    • Digoxin

➤ **Decrease**
  - Myocardial infarction
  - Cardiomyopathy
  - Ischemia
  - Hypoxia
  - Acidosis
  - Negative inotropic drugs
    • Beta blockers
    • Ca++ blockers
    • Antiarrhythmics
    • Some chemo agents
    • Some anesthetics, sedatives
Therapy to Alter CO

$\text{CO} = \text{HR} \times \text{SV}$

- Atropine
- Epinephrine
- Pacing

- Beta blockers
- Ca++ blockers (diltiazem, verapamil)
- Antiarrhythmics
- Adenosine
- Digoxin

Preload

- Fluids
- Blood products
- Volume expanders

Afterload

Diuretics

Venous Dilators:
- NTG
- ACEI
- ARBs
- Nesiritide (Natrecor)
- Morphine

Contractility
CO = HR X SV

Preload — Afterload — Contractility

**Arterial Dilators:**
- Nitroprusside (Nipride)
- Milrinone (Primacor)
- Ca++ blockers
- Antihypertensives
- ACEI
- ARBs
- Nesiritide (Natrecor)

**Vasopressors:**
- Norepinephrine
- Dopamine (high dose)
- Epinephrine
- Neosynephrine
- Vasopressin

**Dobutamine**
- Dopamine
- Milrinone
- Digoxin

**Beta blockers**
- Ca++ blockers
- Antiarrhythmics
- Chemo agents
- Anesthetics
- Propofol

When a goal is to decrease contractility, use beta blockers or Ca++ channel blockers.
Body’s Response to Decrease in CO

- Sympathetic NS Stimulation
  - Increased HR = tachycardia
  - Vasoconstriction
    - Increased diastolic BP
    - Decreased pulse pressure
    - Cool skin
    - Poor capillary refill
    - Decreased urine output

Signs of hypoperfusion

Trend HR & diastolic BP over time: early sign of decompensation
BP = CO x SVR

- BP value does not tell you WHY the BP is low – must evaluate determinants of BP and treat the cause
- 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, drugs, anaphylaxis

**BP Control**

- **Baroreceptor Reflex**
  - Baroreceptors – pressure sensitive cells located in carotid sinus and aortic arch
  - Decreased BP → stimulates vasomotor center in brain to cause *vasoconstriction* and inhibits vagus nerve to ↑HR → ↑BP
  - Increased BP → inhibits vasomotor center to cause *vasodilation* and stimulates vagus nerve to ↓HR → ↓BP
- CSM and Valsalva maneuver act like ↑BP to stimulate vagus nerve and slow HR (diagnosis or treatment of tachycardias)
- Baroreceptor injury, edema, or stimulation with carotid surgery or stenting can cause inappropriate hypotension & bradycardia due to baroreceptor stimulation
Evaluating Blood Pressure

- **Systolic BP** is affected by
  - LV stroke volume (preload, afterload, contractility)
  - Peak rate of LV ejection (related to contractility)
  - Distensibility of blood vessel walls
    - How stiff vs how compliant: stiff walls = higher pressure
- **Diastolic BP** is affected by peripheral vascular resistance (arteriolar tone)
  - A rise in DBP is the first BP change – due to compensatory VC

- **Pulse Pressure**
  - Difference between systolic and diastolic BP
    - Normal = 40 mmHg (120/80 = 40 mmHg PP)
  - Influenced by *stroke volume and arterial compliance* (beat-to-beat change in pulse pressure or SBP reflects SV)
  - ↓ PP is an early sign of ↓ cardiac output (PP of < 30 mmHg is sign of advanced heart failure)
  - PP can be *increased* due to high stroke volume (↑preload, ↑contractility = ↑systolic BP) or high vascular compliance (vasodilation = ↓diastolic BP)
  - PP can be *decreased* due to low stroke volume (↓preload, ↓contractility = ↓systolic BP) or low vascular compliance (vasoconstriction = ↑diastolic BP)
- **Pulsus paradoxus** – exaggerated decrease in systolic BP during inspiration (normal is < 10 mmHg drop during inspiration)
  - Inspiration increases venous return to right heart and RV expands into pericardial space to accommodate volume.
  - With tamponade, the increased venous return can only be accommodated by the RV pushing the septum into the LV, which decreases LV filling and LV stroke volume and causes a > 10 mmHg drop in systolic BP during inspiration.
  - ↓RV filling leads to ↓LV filling & ↓systolic BP

To measure: have patient breathe normally. Gradually deflate cuff until first sound is heard during expiration, then note when sounds heard during both inspiration and expiration.
  - Example: 140/120/70

- **Pulsus Paradoxus on Arterial Line**
  - > 10mmHg drop in systolic BP during inspiration

BP = 136/116/84

- Pulsus paradoxus can be seen in:
  - Cardiac tamponade/constrictive pericarditis
  - Asthma/COPD, PE, tension pneumothorax
  - Restrictive cardiomyopathy
  - RV infarction
  - Hypovolemic shock
Drug Therapy to Decrease BP

\[ \downarrow \text{BP} = \downarrow \text{CO} \times \downarrow \text{SVR} \]

**Drugs to \( \downarrow \text{CO} \)**
- Diuretics
  - ↓preload
- Beta Blockers ("olols")
  - ↓HR and contractility
- Calcium Channel Blockers
  - ↓HR and contractility

**Drugs to \( \downarrow \text{SVR} \)**
- Peripheral Alpha Blockers (prazosin, terazosin, regitine etc)
- Direct Arterial Dilators (hydralazine, minoxidil)
- ACEI ("prils"), ARBs ("sartans")
- PDE inhibitors (milrinone)
- Calcium Channel Blockers ("pines")
- ↑nitric oxide in vascular tissue (nitroprusside, nitrates)
- Centrally Acting Agents (clonidine, guanabenz, guanfacine)

Drug Therapy to Increase BP

\[ \uparrow \text{BP} = \uparrow \text{CO} \times \uparrow \text{SVR} \]

**Drugs to \( \uparrow \text{CO} \)**
- Volume
  - NS, LR
  - Volume expanders
  - Blood Products
- Inotropes
  - Dobutamine
  - Dopamine
  - Milrinone

**Drugs to \( \uparrow \text{SVR} \)**
- Vasopressors
  - Norepinephrine (Levophed)
  - Phenylephrine (Neosynephrine)
  - Vasopressin
  - Epinephrine
  - Dopamine
Balancing $O_2$ Supply & Demand

**Demand**
- **Preload**
  - Diuretics
  - NTG
  - ACEI
  - ARBs
  - Aldosterone blockers
  - Nesiritide
  - Morphine
- **Afterload**
  - Ca++ blockers
  - ACEI
  - ARBs
  - NTG
  - Arterial dilators
  - IABP
- **Contractility**
  - Beta blockers
  - Ca++ blockers

**Supply**
- **$O_2$**
  - Drugs to blood flow
- **Open occluded coronaries**
  - Fibrinolytics
  - PTCA
  - Stents
  - Atherectomy
  - Rotablation
  - CABG

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Tachycardia is bad because it decreases coronary perfusion time and ventricular filling time and it increases myocardial $O_2$ demand.

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**Sympathetic Nervous System & Sympathomimetic Drugs**

**Alpha Receptors**
- (Arteries & Veins)
  - Neosynephrine
  - Vasoconstriction
  - Dopamine
  - Epinephrine
  - Norepinephrine

**Beta Receptors**
- (Heart)
  - Isoproterenol
  - Dobutamine
  - Contractility
  - Heart rate
  - Automaticity
  - Conduction velocity
  - Renin release
- (Arteries, Veins)
  - Beta 2
  - Vasodilation
  - Bronchodilation
## Adrenergic Effects of Sympathomimetic Drugs

<table>
<thead>
<tr>
<th>Drug</th>
<th>Alpha-1 (vasoconstriction)</th>
<th>Beta-1 (cardiac stimulation)</th>
<th>Beta-2 (vasodilation)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epinephrine</td>
<td>++ (&gt;0.2 mcg/kg/min)</td>
<td>+++</td>
<td>+++ (&lt;0.01 mcg/kg/min)</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>+++</td>
<td>++++</td>
<td>+</td>
</tr>
<tr>
<td>Dopamine</td>
<td>++ (&gt;10 mcg/kg/min)</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>0/+</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>++++</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Isoproterenol</td>
<td>0</td>
<td>++++</td>
<td>+++</td>
</tr>
</tbody>
</table>

## Hemodynamic Effects of Vasoactive Drugs

<table>
<thead>
<tr>
<th>Drug</th>
<th>CO/CI</th>
<th>HR</th>
<th>PWP</th>
<th>SVR</th>
<th>MAP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dopamine</td>
<td>↑↑↑↑</td>
<td>↑↑↑</td>
<td>↓</td>
<td>↔↑</td>
<td>↑</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>↑↑↑↑</td>
<td>↑↑</td>
<td>↓</td>
<td>↔↑</td>
<td>↑</td>
</tr>
<tr>
<td>Levophed</td>
<td>↑</td>
<td>↑↑</td>
<td>↔↑</td>
<td>↑↑↑↑</td>
<td>↑↑↑</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>↑</td>
<td>↑↑</td>
<td>↔↑</td>
<td>↑↑↑↑</td>
<td>↑↑</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>↔</td>
<td>↔</td>
<td>↑↑</td>
<td>↑↑↑↑</td>
<td>↑↑↑</td>
</tr>
<tr>
<td>Nitroprusside</td>
<td>↑</td>
<td>↔↑</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Nitroglycerine</td>
<td>↔</td>
<td>↔</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Isoproterenol</td>
<td>↑</td>
<td>↔</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Nesiritide</td>
<td>↑</td>
<td>↔</td>
<td>↓</td>
<td>↓</td>
<td>↔</td>
</tr>
</tbody>
</table>
Effects of Beta Blockers

Beta Receptors

Beta_1
(Heart)

Beta_2
(Arteries, Veins)
(Lungs)

All Beta Blockers:

- ↑ Heart rate
- ↑ Contractility
- ↑ Automaticity
- ↑ Conduction velocity
- ↑ Renin release (kidney)

Non-cardiodeselective Beta Blockers:

- ↓ Vasodilation
- ↓ Bronchodilation

Mechanism of Action

<table>
<thead>
<tr>
<th>Use</th>
<th>Mechanism of Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>↓ heart rate = ↓ CO = ↓ BP, ↓ contractility = ↓ CO = ↓ BP, ↓ renin release in kidney = ↓ angiotensin II formation</td>
</tr>
<tr>
<td></td>
<td>BP = CO \times SVR</td>
</tr>
<tr>
<td>Classic Angina</td>
<td>↓ O_2 demand by ↓ HR, ↓ contractility, ↓ BP, ↓ O_2 supply by ↓ HR which ↓ diastolic filling time and ↓ coronary perfusion time</td>
</tr>
<tr>
<td>Acute Coronary Syndromes</td>
<td>↓ automaticity in ventricle so ↓ risk of VF early in MI, preserves ischemic myocardium by ↓ O_2 demands ↓ mortality rates</td>
</tr>
<tr>
<td>Heart Failure</td>
<td>Upregulation of beta receptors, decrease circulating vasoconstrictors, ↓ LV remodeling, improve O_2 supply &amp; demand, ↓ SCD and A Fib</td>
</tr>
<tr>
<td>Arrhythmias</td>
<td>↓ automaticity so ↓ VT and VF, ↓ AV conduction to slow ventricular rate in A Fib or flutter, may terminate SVTs</td>
</tr>
<tr>
<td>Hypertrophic Cardiomyopathy</td>
<td>↓ contractility so reduces outflow track obstruction, ↓ HR allows longer diastolic filling time, more blood in ventricle decreases outflow tract obstruction</td>
</tr>
</tbody>
</table>
**Effects of Ca\(^{++}\) Channel Blockers**

- **Heart:**
  - ↓ heart rate (except nifedipine–like agents)
  - ↓ AV conduction velocity
  - ↓ contractility (especially verapamil)

- **Blood Vessels:**
  - Coronary vasodilation (prevent vasospasm)
  - Peripheral vasodilation (afterload reduction)
    - Dihydropyridines have mostly peripheral vascular effect

**CCB and BB have the same effects on the heart but opposite effects on blood vessels. Monitor for bradycardia, AV block, hypotension, worsening HF**

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**Clinical Uses of Ca\(^{++}\) Channel Blockers**

<table>
<thead>
<tr>
<th>Use</th>
<th>Mechanism of Action</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Angina:</strong></td>
<td>Prevents vasoconstriction by decreasing amount of Ca(^{++}) available for contraction. Coronary vasodilation increases collateral blood flow. ↓ MVO(_2) by ↓HR, ↓contractility, ↓afterload</td>
</tr>
<tr>
<td>Coronary Spasm</td>
<td></td>
</tr>
<tr>
<td>Classic Angina</td>
<td></td>
</tr>
<tr>
<td><strong>Hypertension</strong></td>
<td>BP = CO x SVR</td>
</tr>
<tr>
<td></td>
<td>↓CO by ↓contractility and HR</td>
</tr>
<tr>
<td></td>
<td>↓SVR by vasodilation</td>
</tr>
<tr>
<td><strong>Arrhythmias: SVT</strong></td>
<td>Slows AV conduction so ↓ventricular response to atrial fib &amp; flutter. Can terminate AV nodal active arrhythmias.</td>
</tr>
<tr>
<td><strong>Hypertrophic</strong></td>
<td>↓ contractility lessens outflow tract obstruction. ↓ HR allows longer diastolic filling time, more blood in ventricle keeps outflow tract open</td>
</tr>
<tr>
<td><strong>Cardiomyopathy</strong></td>
<td></td>
</tr>
</tbody>
</table>
Role of Kidney in HF

Renin-Angiotensin-Aldosterone System

↓ Renal blood flow (↓ BP, ↓ Na⁺, diuresis)
  ↓ Renin release
  Angiotensinogen → Angiotensin I
    (converting enzyme) ↓ Angiotensin II
      Vasoconstriction
      Aldosterone release
        ↑ Na⁺ & H₂O retention
          ↑ BP & Organ perfusion
When there is decreased LV function and cardiac output:

- Kidneys think body is hypovolemic → Na⁺ & H₂O retention
- Baroreceptors think body is hypotensive → peripheral vasoconstriction, release of vasopressin (causes H₂O retention and hyponatremia)

Drugs to Block the RAAS

↓Renal blood flow

**Beta Blockers**

Renin release

**Renin Blockers**

Angiotensinogen → Angiotensin I

(ACE Inhibitors) → Angiotensin II

Angiotensin Receptor Blockers (ARBs)

Vasoconstriction → Aldosterone release

**Aldosterone Blockers** → Na⁺ & H₂O retention

↑BP & Organ perfusion
Indications for ACEI/ARBs

- Heart Failure – all stages

- Hypertension
  - Especially effective in patients with renal disease or diabetes

- Acute MI
  - Early phase for high risk patients to prevent HF
  - All post-MI patients with reduced EF

Side Effects of ACE Inhibitors

- Cough (5–20%) – due to increased bradykinin level
- Hypotension – due to arterial and venous dilation
- Hyperkalemia (3%) – due to decreased aldosterone (which increases K⁺ reabsorption)
- Angioedema (0.1–0.7%) – due to vasodilation and increased vascular permeability resulting from increased bradykinin level
- Decreased glomerular filtration in some patients with renal disease or heart failure – due to dilation of efferent arteriole and hypotension which reduce glomerular perfusion press
  - May also protect basement membrane in the glomerulus in diabetics
Patient Assessment & Monitoring

- Normal and Abnormal Physical Assessment Findings
- Cardiac Monitoring
  - Lead placement
  - Arrhythmia recognition
- Hemodynamic Monitoring
  - Arterial line
  - Pulmonary artery catheter
  - Cardiac output
  - SVO₂ monitoring

Assessment of Chest Pain

- Location
- Radiation
  - Does it go anywhere?
- Character or Quality
  - Sharp, dull, achy, burning, pressure
  - Closed fist (Levine sign) = cardiac pain
- Duration
- Type of Onset
  - Sudden or gradual
- Aggravating Factors
  - What makes it worse?
  - Position changes, breathing, activity
- Relieving Factors
  - What makes it better?
  - Position changes, rest
- Associated Symptoms
  - Sweating, dyspnea, palpitations, dizziness
### Characteristics of Cardiac Pain

<table>
<thead>
<tr>
<th>Location</th>
<th>Radiation</th>
<th>Quality</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angina</td>
<td>Substernal, preordial, diffuse across chest</td>
<td>Shoulders, either arm, jaw, teeth, neck, back, epigastric area</td>
<td>Tight, squeezing, pressure, heavy, crushing</td>
</tr>
<tr>
<td>MI</td>
<td>Same</td>
<td>Same or anginal equivalents</td>
<td>Often more severe</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Angina</th>
<th>Precipitating Factors</th>
<th>Aggravating Factors</th>
<th>Relieving Factors</th>
<th>Associated Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exertion, emotions, heavy meal, cold weather, stress. Unstable angina can occur at rest</td>
<td>Exertion</td>
<td>Constant, no relation to moving or breathing</td>
<td>Rest, NTG (stable angina)</td>
<td>Often none; can be same as MI</td>
</tr>
<tr>
<td>MI</td>
<td>May occur at rest</td>
<td></td>
<td>Sometimes NTG, narcotics</td>
<td>Sweating, nausea, flushing, dizziness, palpitations, dyspnea</td>
</tr>
</tbody>
</table>

#### Evaluating Neck Veins

- Blood in jugular veins assumes level corresponding with right atrial pressure so estimates CVP
  - Right jugular vein reflects pressure best
  - Normal CVP = <9 cm H₂O (2–6 mmHg)
- Position patient so internal jugular is visible (45 degrees)
  - Normal JVD level is no more than 3 cm above the sternal angle
  - Angle of Louis is 5 cm above right atrium
  - Place ruler vertically at Angle of Louis
  - Measure in cm how far above Angle of Louis the neck vein is visible
  - Add this measurement to 5 = estimated CVP
Neck veins are distended whenever CVP is high or something interferes with RV filling:
RV failure, cardiac tamponade, restrictive pericarditis, restrictive cardiomyopathy, tension pneumothorax

More on Neck Veins
- Abdominojugular reflux – seen in RV failure
  - Press on upper right quadrant for 30–60 seconds while watching neck veins
  - Rise in neck veins of 1 cm or more = positive abdominojugular reflux
- Kussmaul’s sign (paradoxical elevation of jugular venous pressure during inspiration)
  - Normally the neck veins empty during inspiration as intrathoracic pressure drops and venous return to the heart increases
  - In “restrictive” disease (pericardial effusion, constrictive pericarditis, cardiomyopathy, diastolic HF) the heart cannot handle the increased venous return so neck veins elevate when venous return increases during inspiration

CVP = 12 cm H₂O
Heart Sound Listening Areas

- Aortic
  - Second right ICS
- Pulmonic
  - Second left ICS
- Tricuspid
  - Left bottom of sternum
- Mitral
  - Apex area, mid-clavicular line

S₃ Gallop

- Occurs during passive ventricular filling early in diastole before atria contract.
- Heard after S₂ (Lub – Dup – ta)
- Is early sign of LV systolic failure (LV S₃) or pulmonary hypertension (RV S₃) and occurs with mitral, aortic or tricuspid insufficiency.
- Can be heard in healthy children and young adults to age 40.

“I believe”  S₁  S₂  S₃  S₁  S₂  S₃
**S₄ Gallop**

- Occurs during atrial contraction as blood is ejected into a noncompliant ventricle at the end of diastole
  - Absent in atrial fibrillation
- Heard before $S_1$ (ta – Lub – Dup)
- Is a sign of LV diastolic dysfunction
  - Often heard in acute MI due to decreased compliance
  - Common with hypertensive heart disease, aortic stenosis, hypertrophic cardiomyopathy

```
S₄ S₁ S₂
```

“Believe me”

---

**Murmurs**

- **Stenosis** is a murmur heard when a valve is supposed to be open (does not develop acutely)
- **Regurgitation** is a murmur heard when a valve is supposed to be closed (can occur acutely)

```
In systole, which valves are supposed to be open?
Which are supposed to be closed?
```

```
In diastole, which valves are supposed to be open?
Which are supposed to be closed?
```
Systolic Murmurs

- **S1** MMMMM **S2**
- Tricuspid Regurgitation
- Mitral Regurgitation
- Pulmonic Stenosis
- Aortic Stenosis
- VSD (ventricular septal defect)
- HOCM (obstructive cardiomyopathy)

Blood flowing backwards through valves that are supposed to be closed.

Blood flowing forwards through valves with narrowed orifice (stenosis).

Mitral regurgitation is a systolic murmur that can occur acutely with papillary muscle ischemia or rupture in MI.

Diastolic Murmurs

- **S1** S2 MMMMMM
- Tricuspid Stenosis
- Mitral Stenosis
- Pulmonic Regurgitation
- Aortic Regurgitation

Blood flowing forwards through valves with narrowed orifice (stenosis).

Blood flowing backwards through valves that are supposed to be closed.

Aortic regurgitation is a diastolic murmur that can occur acutely with aortic arch dissection involving aortic valve.
Carol Jacobson

**Invasive Hemodynamic Monitoring**

With balloon deflated:
- Proximal port monitors CVP
- Distal port monitors pulmonary artery (PA) systolic & diastolic pressure

With balloon inflated (wedged):
- Proximal port monitors CVP
- Distal port monitors LA pressure which reflects LV end diastolic pressure (referred to as PWP or PAOP)

**Normal Cardiac Pressures**

Nickle  Dime  Quarter  Dollar
- **CVP** measures right atrial pressure and is used as a clinical indicator of **RV preload**
  - Normal CVP = 2–6 mmHg
- **PAOP** reflects left atrial pressure and is used as a clinical indicator of **LV preload**
  - Normal PWP = 8–12 mmHg

We assume that the pressures measured are a reflection of volume in the ventricles which indicates preload, but pressure can be higher when ventricles are noncompliant. **Pressure does not equal volume!**
- **PVR** is a calculated value based on mean PA pressure and PWP and is used as a clinical indicator of RV afterload
  - Normal PVR = < 250 dynes
- **SVR** is a calculated value based on mean arterial pressure and CVP and is used as a clinical indicator of LV afterload
  - Normal SVR = 800–1200 dynes

In order for these calculated values to be accurate, the parameters they are based on need to be accurate: CVP & PWP

**AACN Practice Alert for Pulmonary Artery Pressure Monitoring – 2009**

- Perform square waveform test each shift and whenever system disturbed (blood samples, disconnection)
- Position patient supine with HOB between 0° – 60°, lateral, or prone (allow 15 minutes to stabilize before reading pressures)
- Level stopcock air–fluid interface to phlebostatic axis
- Use graphic tracing that includes the ECG
- Take measurements at end expiration

**New practice alert 2016**
Marking the Phlebostatic Axis

- Phlebostatic axis is 4th intercostal space at mid anterior-posterior chest level (left atrial level)
- System needs to be zeroed and leveled at the phlebostatic axis
- Measure accurately and mark on chest

Zeroing and Leveling

- **Zeroing** tells the monitoring system that atmospheric pressure is “zero”
  - It removes the effect of hydrostatic pressure in the tubing system and establishes a baseline of zero so all pressure recorded by the system is patient pressure
- **Leveling** means that the stopcock that was used to zero must be at the phlebostatic axis for all pressure readings
  - Keep the transducer at the phlebostatic axis while monitoring pressure
Right Atrial Waveforms

- a wave = atrial contraction and follows P wave
- c wave = closure of tricuspid valve
- v wave = atrial filling and follows QRS

Record the mean of the “a” wave for the CVP

2-6 mmHg

Right Ventricular Waveforms

The monitor is never right on the RV diastolic pressure!

- It reads the lowest point
- We want to record the plateau (true RVEDP)

15-30
2-6

Systolic

Diastolic
The monitor is usually right about PA pressures

- Same “a”, “c”, and “v” waves as CVP
- Further removed from the ECG waves
  - “a” wave near end of QRS
  - “v” wave after T wave

Record the mean of the “a” wave for the PWP
Normal PAD–PAW gradient is 1 – 4 mmHg

Causes of large PAD – PWP gradient:
• Severe COPD, ARDS, PE
  (primary pulmonary problems not due to LV failure)

Intrathoracic pressure changes during inspiration can cause the waveform to swing
• Measure pressures at end-expiration
• Find inspiration and measure right before
End Expiration in Spontaneous Breathing

- During inspiration, intrathoracic pressure drops
- Inspiration is a negative dip in the waveform
- Read just before the negative inspiratory dip

End Expiration in Ventilator Breath

- Positive pressure ventilation increases intrathoracic pressure
- Inspiration is a rise in waveform
- Read pressures just before the inspiratory rise
This patient is breathing spontaneously. Where do you read his PAOP?

This patient is on mechanical ventilation. Where do you read his CVP?
Cardiac Output Measurement

- **Intermittent Method**
  - Injection of known amount of solution with known temperature into RA → solution travels through RV into PA → temp of blood measured in PA by thermistor near distal port
  - The resultant change in temperature is plotted on a time-temperature curve
  - The area under the curve is indirectly proportional to CO: when cardiac output is low, it takes more time for the temperature to return to baseline and produces a larger area under the curve. With high cardiac output, the cooler injectate is carried more quickly through the heart, and the temperature returns to baseline faster. This produces a smaller area under the curve.

- **Continuous Method**
  - Uses special PA catheter with a heat filament that warms blood → blood travels through RV into PA → temp of blood measured in PA by thermistor near the distal port
  - CO calculated based on temperature change
  - CO values are updated about every 60 seconds and displayed graphically on the monitor
SvO₂ Monitoring

- Oximetry PA Catheter
  - PA catheter with fiberoptic port that emits light and reflects amount of Hb saturated with O₂ in the pulmonary artery.
  - Measures mixed venous O₂ saturation (SvO₂) from entire body
  - Normal is 60% – 80%

- Central venous oximetry catheter
  - CVP catheter in SVC with fiberoptic port that emits light and reflects amount of Hb saturated with O₂
  - Measures O₂ saturation in venous blood returning from head and upper extremities (ScvO₂)
  - Normal is 65% – 85%

Complications of PA Catheters

- Insertion
  - Pneumothorax
  - Vessel trauma – bleeding, PA rupture
  - Infection – insertion site, risk of endocarditis, sepsis
  - Valve damage – tricuspid, pulmonic
  - Arrhythmias – VT, RBBB (big problem if preexisting LBBB)

- Maintenance
  - Pulmonary infarction – if catheter migrates to small branch
    - Continuously monitor waveform
    - Limit time of balloon inflation
  - Thrombosis – catheter acts as a site for thrombus formation
  - Air embolism – ruptured balloon
    - Use no more than 1.5 cc air, never actively deflate balloon

Use associated with ↓mortality in severe HF and cardiogenic shock but not in other patient populations
IABP

- **Indications**
  - Cardiogenic shock
  - Intractable angina
  - Weaning from CPB
  - Adjunctive therapy with thrombolysis or PCI
  - Prophylaxis in patients with severe left main stenosis or critical aortic stenosis prior to surgery

- **Insertion**
  - Percutaneous femoral artery
  - Positioned in descending aorta below subclavian artery and above renal arteries

- **Hemodynamic effects**
  - **Inflation** during diastole improves coronary, brain, and peripheral blood flow by pushing blood retrograde toward aortic arch and coronary arteries and forwards toward periphery.
  - **Deflation** during systole reduces afterload by creating an “empty space” in aorta and reduces pressure that LV has to pump against.
Carol Jacobson

Contraindications to IABP Therapy

- **Absolute contraindications**
  - Aortic regurgitation
  - Thoracic or abdominal aortic aneurysm
  - Aortic stents
  - Bleeding disorders
  - Severe peripheral vascular disease
  - Bilateral fem-pop bypass grafts

- **Relative contraindications**
  - Calcific aortic or iliac disease
  - Peripheral arterial disease
  - Thrombocytopenia
IABP Complications & Monitoring

- Limb ischemia
  - Monitor limb for the 6 Ps

- Vascular laceration
  - Observe insertion site for bleeding or hematoma

- Major bleeding due to platelet trauma, anticoagulation or vascular injury
  - Monitor platelets and Hct

- Thrombosis and embolization
  - Monitor for the 6 Ps

- Catheter dislodgement up or down
  - Monitor and document left radial pulse
  - Monitor urine output – sudden decrease can indicate migration

- Infection

Cardiac Monitoring & Arrhythmia Interpretation

- Arrhythmia monitoring (on test blueprint)
- ST segment monitoring
- QT interval monitoring
What is the best bedside monitoring lead for cardiac arrhythmia recognition?

A. Lead II
B. Lead III
C. Lead V₁
D. It doesn’t matter as long as you can see P waves and QRS complexes

Best Leads for Arrhythmia Monitoring

- V₁ is recommended as lead of choice
- V₆ is recommended as second choice if V₁ not possible
- MCL₁ and MCL₆ can be substituted if true V leads not possible, but they are not as accurate as a true V lead

- When there is a question about a rhythm, the best lead is a 12 Lead ECG!
Best Practice for Bedside Arrhythmia Monitoring

- Use multiple leads whenever possible
  - Continuous 12 lead monitoring is the best
  - A 5–lead system is better than a 3–lead system
  - If two leads are available, use V₁ as primary lead and Lead II as second lead (according to AACN practice alert)
  - When given a choice of MCL₁ or V₁ – choose V₁
- Place electrodes where they are supposed to be and connect leads correctly!!
- Proper skin preparation is important
  - Scrub with rough washcloth until pink and dry
  - Use alcohol to dry if oily or diaphoretic
  - Shave excess hair
  - Make sure electrodes are not dry

Lead Placement for a 5–Wire System

- Arm electrodes on shoulders close to where arm joins torso
  - Front, top, or back of shoulder OK
- Leg electrodes low on thorax or on hips
- Chest electrode placed according to V lead desired
  - V₁ = 4th right intercostal space
  - V₆ = straight line from V₄, left mid–axillary line
Dyshytmias

- Bradycardias
  - Sick sinus syndrome
  - AV blocks
    - 2nd degree Type I – Wenckebach, occurs in AV node
    - 2nd degree Type II – occur below AV node
    - 3rd degree (complete)

- Supraventricular Tachyarrhythmias
  - Atrial fib/flutter
  - SVT (AV nodal reentry, circus movement tachycardia in WPW)

- Ventricular Tachyarrhythmias
  - VT (monomorphic, polymorphic)
  - Torsades
  - V Fib
AV Blocks & Treatment

✓ 1st Degree
  • All P waves conduct with longer than normal PRI
    • Normal PR = 0.12 – 0.20 sec
    • No treatment – observe

1st degree block: PR = .28 sec

✓ 2nd Degree
  • Type I (Wenckebach) – block occurs in AV node
    • Common with inferior wall MI and drugs that slow AV conduction: beta blockers, Ca++ blockers, digoxin
    • Usually no treatment needed, atropine or temp pacing may be needed for symptomatic bradycardia
  • Type II – below AV node, more dangerous
    • Occurs with anterior wall MI and chronic conduction system disease
    • May need temporary or permanent pacing
    • Atropine doesn’t work

2nd degree, Type I: gradual ↑ in PRI on consecutively conducted beats before blocked P wave

2nd degree, Type II: constant PRI before blocked P wave
3rd Degree – complete block
- Can occur with any MI
  - Inferior wall block usually in AV node and can have junctional escape pacemaker
  - Anterior wall block is below AV node with ventricular escape pacemaker
- Temporary pacing usually indicated in MI
- Permanent pacing if doesn’t resolve
- Atropine doesn’t work

3rd degree: no relationship between P waves and QRS, junctional escape pacemaker

3rd degree: no relationship between P waves and QRS, ventricular escape pacemaker

Treatment of Symptomatic Bradycardia
- Atropine 0.5 IV
  - ↑ rate of sinus node or junctional pacemaker
  - Speeds AV conduction if there is any
  - Does not work for 3rd degree block or Type II block
- Pacing
  - External
  - Temporary transvenous or epicardial
  - Permanent

Indications for Transvenous Pacing
- Symptomatic bradycardia not responsive to atropine
- Bradycardia with acute MI
- Bradycardia following cardiac surgery
- New bifascicular block or alternating RBBB and LBBB
Temporary Pacemakers

- External Pacing via two large adhesive pads placed on the chest. (Intended for emergency use and is only used until transvenous pacing can be established)

- Transvenous pacing – lead inserted via a peripheral or central vein into the RV apex for ventricular pacing

- Epicardial pacing – pacing leads attached to the atria and/or ventricles during cardiac surgery and pulled through the chest wall so they can be accessed when needed

Main Functions of a Pacing System

- **Capture** – ability of the pacing stimulus to depolarize the chamber being paced.
  - ECG shows pacing spikes immediately followed by either a P wave or a QRS complex

- **Sensing** – ability of the pacemaker to recognize and respond to intrinsic cardiac depolarizations.
Loss of Capture

Are pacer spikes present? Yes
Capture? Total loss of capture

To correct loss of capture:
• Increase the mA
• Reposition the patient

Loss of Sensing

Intrinsic beats not sensed

To correct loss of sensing:
• Increase the sensitivity
Managing Patients with Temporary Pacemakers

- **External (transthoracic)**
  - Sedation and pain meds – it hurts!
  - Cardiac monitoring for proper capture & sensing (spike may be so big it obscures QRS)
  - Monitor pulse with each paced beat: doppler on radial or antecubital; or pulse oximeter on finger

- **Transvenous**
  - Cardiac monitoring for proper capture & sensing
  - Monitor insertion site for infection
  - If femoral insertion, keep patient in bed with HOB no higher than 30°
  - Keep pulse generator away from patient!

Narrow QRS Tachycardias

**Atrial Tachycardia**
- P waves visible in front of QRS or may be hidden in T wave
- Atrial rhythm is regular: 150-250
- Ventricular rate usually same (unless AV block present)

**Atrial Flutter**
- Atrial rate 250-350 with flutter waves
- Ventricular rhythm regular or irregular
- Ventricular rate depends on amount of AV block: 150 or lower

**Atrial Fibrillation**
- Atrial rate very fast with irregular fibr waves
- Ventricular rhythm always irregular
- Ventricular rate depends on amount of AV block: can be up to 200 or so

**Rate Control:**
- Ca²⁺ blockers (verapamil, diltiazem)
- Beta blockers

**Adenosine** is not appropriate
Narrow QRS Tachycardias

- AV Nodal Reentry Tachycardia
  - Regular at rates 140-200
  - P waves usually not seen (sometimes peek out at end of QRS)
  - Term “SVT” is appropriate
  - AV node is part of reentry circuit that maintains the tachycardia

- AV Reentry Tachycardia
  - Regular at rates 140-280
  - P waves not easily seen – appear in ST segment when seen
  - Term “SVT” is appropriate
  - AV node is part of reentry circuit that maintains the tachycardia

Vagal maneuver

- Adenosine – drug of choice
- Beta blockers
- Ca++ blockers

Hemodynamic consequences

- Loss of atrial kick
- Rapid ventricular rate \( \downarrow \) SV and CO
- Risk of thrombus formation and embolization \( \rightarrow \) stroke

Treatment

- Rate Control – AV nodal blocking drugs
  - Beta blockers, Ca++ blockers (verapamil, diltiazem), digoxin in HF patients
- Rhythm Control
  - Cardioversion – electrical or chemical: ibutilide, flecainide, dofetilide, propafenone, amiodarone
  - Maintenance of NSR with antiarrhythmic drugs: amiodarone, dronedarone, flecainide, propafenone, sotalol

- Anticoagulation
- Ablation procedures
- MAZE procedure

Atrial Fibrillation
**Causes of a Wide QRS Tachycardia**

- SVT with Bundle Branch Block
- Ventricular Tachycardia
- Accessory Pathway with Antegrade Conduction

**Monomorphic VT**

**Treatment:**
- Defibrillate if pulseless
- Sedate and cardiovert if hemodynamically unstable but not unconscious (usually start with 100 joules)
  - Amiodarone is drug of choice
  - Lidocaine still OK (works best in ischemic VT)
  - ICD is treatment of choice for recurrent VT not responsive to drug therapy and high risk for SCD
Polymorphic VT
(normal QT interval)

Treatment:
- Treat ischemia
- Correct electrolytes
- Beta blockers (anti-ischemic, ↓SNS)
- Lidocaine often effective if ischemic VT
- Amiodarone OK if QT is normal
- Defibrillate if it becomes sustained with loss of consciousness (200 J biphasic, 360 J monophasic)

QT = 320 ms
QTc = 400 ms

Torsades de Pointes
(polymorphic VT with long QT interval)

Treatment:
- Defibrillate if sustained with loss of consciousness
- Correct underlying cause
  - DC suspicious drugs (antiarrhythmics, antidepressants, anti-acids, some antibiotics, haldol, many others - www.crediblemeds.org)
  - Correct electrolyte imbalances (K⁺, Mg++)
  - Sudden bradycardia
- IV Magnesium
- Overdrive pacing at rates of around 100 bpm

Risk of torsades greatest with QTc ≥ 500 ms (.50 sec)
Ventricular Fibrillation

Treatment (ACLS):
- Defibrillate with 200 joules (biphasic) or 360 joules (monophasic)
- If rhythm continues: immediate CPR beginning with compressions (30:2 compression/ventilation ratio)
- Epinephrine 1 mg IV q 3–5 minutes
- Amiodarone 300 mg IVP OR Lidocaine 1–1.5 mg/kg

Narrow QRS
- Stable
- Unstable
  - Vagal maneuver
  - Adenosine 6mg
    - Repeat with 12mg if necessary
  - Beta blocker
  - Ca++ blocker (diltiazem)

Wide QRS
- Unstable
- Stable
  - Can try adenosine (if regular and monomorphic)
  - Antiarrhythmic
    - Amiodarone
    - Procainamide
    - Sotalol

Cardiovert
Defibrillation & Cardioversion

- Stops ALL electrical activity in heart
- Fastest pacemaker wakes up first – should be sinus node
- Keep pads away from pacemaker/ICD pulse generator

**Defibrillation**
- Unsynchronized shock delivered anywhere in cardiac cycle
- Used for VF and pulseless VT
- Biphasic defibrillator: start with 120 joules and escalate to 200 joules if necessary
- Anterolateral pad placement is fastest

**Cardioversion**
- Shock synchronized on QRS to avoid hitting T wave
  - Make sure synch marker is on QRS
- Used for VT with pulse, unstable atrial fib/flutter, unstable SVT
- Elective for atrial fib/flutter
- Antero-posterior pad placement most effective
- Energies vary: 50-200 joules

---

### Bundle Branch Blocks

<table>
<thead>
<tr>
<th></th>
<th>(V_1)</th>
<th>(V_6)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Normal Conduction</strong></td>
<td><img src="image" alt="Normal Conduction" /></td>
<td><img src="image" alt="Normal Conduction" /></td>
</tr>
<tr>
<td>QRS = 0.06 - 0.10 sec</td>
<td>(V_1) = rS</td>
<td>(V_6) = qR or R</td>
</tr>
<tr>
<td><strong>LBBB</strong></td>
<td><img src="image" alt="LBBB" /></td>
<td><img src="image" alt="LBBB" /></td>
</tr>
<tr>
<td>QRS = 0.12 or more</td>
<td>(V_1) = rR'</td>
<td>(V_6) = qRs (wide S wave)</td>
</tr>
<tr>
<td><strong>RBBB</strong></td>
<td><img src="image" alt="RBBB" /></td>
<td><img src="image" alt="RBBB" /></td>
</tr>
<tr>
<td>QRS = 0.12 or more</td>
<td>(V_1) = QS</td>
<td>(V_6) = wide R wave</td>
</tr>
</tbody>
</table>
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Sinus tachycardia with LBBB

Atrial fib with RBBB

Antiarrhythmic Drugs

Speed Sinus Rate:
- Atropine
- Epinephrine

Slow Sinus Rate:
- Beta blockers
- Ca++ blockers
- Digoxin
- Antiarrhythmics

Speed AV Conduction:
- Atropine
- Epinephrine

Slow AV Conduction:
- Beta blockers
- Ca++ blockers
- Adenosine
- Digoxin
- Vagal Maneuvers

Suppress Atrial Arrhythmias:
- Procainamide
- Flecainide
- Propafenone
- Sotalol
- Amiodarone
- Dronedarone
- Ibutilide
- Dofetilide

Slow or Block AP:
- Procainamide
- Flecainide
- Amiodarone (oral only)
- Propafenone
- Sotalol

Suppress Ventricular Rhythms:
- Lidocaine
- Procainamide
- Amiodarone
- Sotalol
- Magnesium

www.cardionursing.com
AACN Practice Alert: Alarm Management – 2013

- Provide proper skin preparation for ECG electrodes.
- Change ECG electrodes daily
- Customize alarm parameters and levels on ECG monitors
- Customize delay and threshold settings on O2 sat (SpO2) monitors
- Provide initial and ongoing education about devices with alarms
- Establish interprofessional teams to address issues related to alarms – policies and procedures
- Monitor only those patients with clinical indications for monitoring

Acute Coronary Syndromes

♥Unstable Angina
♥Non–ST–Elevation MI (NSTEMI)
♥ST Elevation MI (STEMI)

New guidelines refer to NSTE–ACS which includes UA & NSTEMI
Acute Coronary Syndrome

No serum marker release

Serum marker release

Ischemia:
- Stable Angina
- Unstable Angina

Injury:
- Non-Q-Wave MI
- Q-Wave MI

ECG: No ST Elevation
ECG: ST Elevation

Pathogenesis of ACS

Plaque rupture
- Spontaneous
- Induced by PCI

Platelet activation

Platelet aggregation
Carol Jacobson

**Antiplatelet Drugs**

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

- Heparin
- Bivalirudin (Angiomax)
- Dabigatran (Pradaxa)

**IIb-IIIa Inhibitors**

- Reopro
- Integrilin (eptifibatide)
- Aggrastat (tirofiban)
Extrinsic Pathway

Plaque rupture

$\xrightarrow{Tissue\ Factor}$

$X$ 

Xa

$\xleftarrow{Prothrombin\ Activator}$

$Ca^{++}$

$\xrightarrow{Prothrombin}$

$\xrightarrow{Thrombin}$

Factors formed in liver and inhibited by warfarin: IX, X, VII, II

Fibrinogen $\xrightarrow{Ca^{++}}$ Fibrin $\rightarrow$ Thrombus

Indirect Xa inhibitor: fondaparinux (Arixtra)

Heparin (indirect)

LMWH: enoxaparin (Lovenox)

dalteparin (Fragmin)

Direct Xa inhibitor: apixaban (Eliquis)

rivaroxaban (Xarelto)

edoxaban (Savaysa)

Heparin (indirect)

Direct thrombin inhibitors: argatroban (Novastat)

bivalirudin (Angiomax)

lepirudin (Refludan)

dabigatran (Pradaxa)

Symptoms

- Chest pressure, tightness (don't ask about “pain”) in center of chest
- Chest discomfort spreading to shoulders, neck, arms, back, jaw
- Anginal equivalents: unexplained fatigue, SOB, flu-like symptoms, atypical pain
  - Women, diabetics, elderly

Biomarkers – Troponin (CK-MB no longer recommended)

- Elevated in STEMI
- Normal in NSTEMI/UA

ECG

- STEMI = ST elevation
- UA/NSTEMI = ST depression or T wave inversion
Troponins

- Troponins I and T are more sensitive in detection of myocardial injury than CK-MB
- Are cardiac specific but not disease specific. Can be elevated with any kind of cardiac injury.
- Levels begin to rise 2–3 hours after onset of acute MI
- Peak in 12 – 48 hours depending on reperfusion status
  - Higher peaks and earlier return to baseline with reperfusion
  - Higher peaks with larger MIs
- Remain elevated for up to 10 days

ECG Diagnosis of ACS

- STEMI
  ST elevation in leads facing infarcted tissue

- UA or NSTEMI
  ST depression or T wave inversion
RCA Supplies:
- Inferior wall
- Posterior wall
- Right ventricle
- Septum (posterior 1/3)
- Sinus node (60%)
- AV node (90%)

LAD Supplies:
- Anterior wall
- Septum (anterior 2/3)

Circumflex Supplies:
- Lateral wall
- In 10% - inferior/posterior walls
- AV node (10%)
The 12 Leads

- Lateral/Circ
- Inferior/RCA
- Septal/LAD
- Anterior/LAD

ECG Diagnosis of STEMI

Normal: 

- STEMI
  - ST elevation 1mm or more in 2 contiguous leads
  - New LBBB
  - True posterior MI (ST elevation in posterior leads)
Anterior wall STEMI

- LAD or left main occlusion
- ST elevation V1–V4
- Monitor for LV failure, BBB, Type II AV blocks, ventricular arrhythmias

Inferior wall STEMI

- RCA (sometimes circumflex)
- ST elevation II, III, aVF (record right side leads)
- Monitor for bradycardia, Type I AV block (Wenckebach), N/V, hypotension
Lateral Wall MI
- Circumflex artery
- ST elevation I, aVL, and/or V5, V6
- Often associated with anterior or posterior MI
- Monitor for atrial arrhythmias, papillary muscle dysfunction or rupture

Posterior Wall MI
- Usually RCA, sometimes circ
- Reciprocal ST depression V1-V3 (record posterior leads V7-V9)
- Often with inferior or lateral MI
- Watch for papillary muscle rupture
Right Ventricular Infarction

- Occurs in about 40–45% of inferior MIs
- 12 Lead ECG Clues:
  - ST elevation in II, III, AVF, V₁ (can’t count on V₁)
  - Should record right side chest leads V₄₋₆R

- RV infarction results in decreased LV filling and signs of LV forwards failure
  - Signs of high RV pressure (JVD) and low LV pressure (clear lungs, hypotension)

- Treatment of RV infarction
  - Fluids
  - Avoid preload reduction (NTG, MS)
  - Inotropes if necessary
  - Dual chamber pacing if possible rather than VVI

RVMI
- RCA
- ST elevation II, III, aVF, V₁ (record right side leads)
- Reciprocal ST depression I, aVL
- Watch for bradycardia, AV block, tachycardia related to ↓SV due to ↓LV filling
### Table: ECG Indications of Myocardial Infarction

<table>
<thead>
<tr>
<th>Infarction Site</th>
<th>Indicative Changes (ST elevation)</th>
<th>Reciprocal Changes (ST depression—not always seen)</th>
<th>Complications</th>
</tr>
</thead>
</table>
| **Anterior**    | V₁–V₄ (not necessarily all of these leads)  
ARD with proximal LAD occlusion | II, III, AVF, VS with proximal LAD occlusion | LV failure most common. Right or left BBB, hemiblocks, bifascicular blocks, Type II 2° AV block, VT, VF, atrial arrhythmias. |
| **Septal**      | V₁, V₂ | VS with proximal LAD occlusion | BBB, hemiblocks. |
| **Anterolateral** | V₁–V₆, I, AVL | II, III, AVF | LV failure most common. Right or left BBB, hemiblocks, bifascicular blocks, Type II 2° AV block, VT, VF, atrial arrhythmias. |
| **Inferior**    | II, III, AVF | I, AVL | Bradycardia (sinus, junctional rhythm), 1° AV block, Type I 2° AV block, 3° AV block with junctional escape rhythm, LBBB, VT, VF, atrial arrhythmias. |
| **Posterior**   | Posterior leads V₈, V₉ | V₁–V₃ | Same as inferior. |
| **Lateral**     | I, AVL, V₅, V₆ | II, III, AVF | Usually occurs in combination with anterior or posterior so similar complications. |
| **Right ventricle** | Right side leads V₅R–V₆R | V₅, V₆ | Same as inferior. RV failure, low cardiac output. Need volume. |

### ECG Diagnosis of NSTE–ACS

**Normal:**

![Normal ECG](https://via.placeholder.com/150)

**UA or NSTEMI**

- **ST depression**
- **T wave inversion**

![UA or NSTEMI ECG](https://via.placeholder.com/150)
Management of NSTEMI similar to STEMI with anti-ischemic and antiplatelet drugs. High risk patients should go to cath lab for intervention ASAP.

ACS: Initial Assessment & Management

- ASA 162-325 mg ASAP with onset of chest pain
- Oxygen if O₂ sat < 90%, heart failure, dyspnea
- 12 Lead ECG interpreted within 10 minutes
- NTG 0.4 mg SL q 5 min x 3 for ongoing chest pain
  - IV for persistent ischemia, HF, or HTN
  - Ask about Viagra or Levitra within 24 hrs or Cialis within 48 hours
  - Avoid with RV infarction or SBP < 90mmHg
- Morphine 2-8 mg IV q 5-15 minutes if needed for pain
  - Avoid with RV infarcts
Management of **STEMI** (2013 guidelines)

**Primary PCI** is the recommended therapy for reperfusion

- Only option if contraindications to fibrinolysis
- Best option for cardiogenic shock or acute severe HF (irrespective of time delay from symptom onset)

Time from FMC (EMS system) to device (balloon inflation or thrombectomy) should be 90 minutes or less

**Non-PCI Capable Hospital?**

- **Transfer to a PCI-capable** hospital for primary PCI is recommended with a FMC-to-device time of 120 minutes or less (from EMS to balloon inflation)

- **Fibrinolysis** when PCI cannot be performed within 120 minutes of FMC

- Door to drug time within **30 minutes of arrival at hospital**
Fibrinolytic Therapy

- **tPA (Alteplase)**
  - 15 mg bolus IV, then 0.75 mg/Kg over next 30 minutes, then 0.5 mg/Kg over next 60 minutes
- **rtPA (Reteplase)**
  - 10 units x 2 over 30 minutes
- **TNK–tpa (Tenecteplase)**
  - Weight based bolus over 10–15 minutes
- **APSAC (Anistreplase)**
  - 30 units IV over 2–5 minutes

- **Contraindications**
  - Active bleeding
  - Suspected aortic dissection
  - Any prior intracerebral hemorrhage
  - Known structural cerebral vascular lesion or malignant intracranial neoplasm
  - Ischemic stroke within 3 months EXCEPT acute ischemic stroke within 3 hours
  - Significant closed head or facial trauma within 3 months

- **Complications**
  - Bleeding
    - 1% intracranial bleeding
    - < 5% major bleed (>10 point drop in HCT or requiring transfusion)

---

Cardiac Catheterization

**Left heart cath** to evaluate coronary arteries, LV function, measure LV & aortic pressures, evaluate mitral & aortic valves, perform PCI procedures and catheter-based valve procedures. Access via:
- Femoral artery
- Radial artery
- Brachial artery

**Right heart cath** to evaluate right heart function, measure PA pressures and CO, detect left-to-right shunt, evaluate pulmonic and tricuspid valves, perform EPS. Access via:
- Femoral vein
- Internal jugular vein
- Subclavian vein
Radial vs Femoral Approach

Radial Artery
- **Advantages**
  - Less bleeding – easily compressible artery
  - More comfortable for patients
  - Short bedrest time and LOS
  - Fewer access site complications
- **Disadvantages**
  - Technically more difficult – steep learning curve
  - Smaller artery – can’t accommodate larger catheters or devices
  - Prone to spasm

Femoral Artery
- **Advantages**
  - Large artery can accommodate larger catheters and devices
  - Easier to perform
- **Disadvantages**
  - More bleeding – harder to get hemostasis
  - Uncomfortable for patients
  - Longer bedrest times
  - More access site complications (pseudoaneurysms, AV fistula, retroperitoneal bleed)

Percutaneous Coronary Interventions (PCI)
- PTCA (angioplasty)
- Stents
- Atherectomy
- Rotablation
Problems with Stents

Restenosis

- Due to intimal hyperplasia (excess tissue growth around struts of stent) and local vessel injury from procedure
- DES decrease restenosis rate by about 75% compared to BMS
  - Zotarolimus, everolimus – immunosuppressive and antiproliferative properties to prevent intimal hyperplasia – released over days to a year
  - Newer stents have much lower risk of restenosis (everolimus may be lowest)

Thrombosis

- Clot formation at site of stent struts
  - Occurs equally with BMS and DES
  - BMS re-thrombosis usually occurs within 24–48 hours
  - DES re-thrombosis usually occurs within a year
  - Late re-thrombosis (after 1 year) more common with DES when antiplatelet therapy (clopidogrel, etc) is stopped
- Presents as acute myocardial ischemia or infarction and is a medical emergency
Prevention of thrombosis

- Anticoagulation (heparin, bivalirudin during procedure)
- Dual antiplatelet therapy (DAPT) with clopidogrel or prasugrel or ticagrelor and ASA for 1 year after procedure

Risk of re-thrombosis with DES if dual antiplatelet drugs DC’d before 1 year (even for short period of time)

Nursing Care After PCI

- Main Concerns
  - Restenosis
  - Groin Management
  - Anticoagulation, antiplatelets
- Routine Monitoring
  - Continuous ECG & ST segment
  - VS, groin checks, pedal pulses (q 15” x four, then q 30” x two, then as indicated)
- Encourage fluids (contrast nephropathy)

- Monitor for complications
  - Cardiac tamponade (Beck’s triad: JVD, hypotension, muffled heart sounds)
  - Retroperitoneal bleeding (back pain, hypotension)
  - Hematoma or pseudo-aneurysm at access site
  - Distal embolization of clot into extremity (6 P’s)
  - Reocclusion of coronary artery (chest pain, ST elevation)
  - Contrast nephropathy (increase in plasma creatinine of more than 0.5 mg/dL above baseline within 12–24 hours of procedure)

Same monitoring is needed following peripheral vascular procedures or carotid stenting
Discharge Medications Following ACS

- **ASA** (indefinitely)
- **Plavix or Brilinta or Effient** (at least 12 months)
- **Beta Blocker** (indefinitely)
- **ACEI** (ARB if ACEI intolerant)
- **Statin** (high dose indefinitely)

Plavix = clopidogrel, Brilinta = ticagrelor, Effient = prasugrel

Complications of MI

- **Arrhythmias**
  - **Inferior MI (usually RCA):** sinus bradycardia, atrial arrhythmias, 1\textsuperscript{st} degree block, Wenckebach, 3\textsuperscript{rd} degree (often with junctional escape rhythm), ventricular arrhythmias
  - **Anterior MI (LAD):** type II AV block, 3\textsuperscript{rd} degree block with ventricular escape rhythm, ventricular arrhythmias

- **Pump Failure**
  - Especially with anterior MI
  - The more muscle lost the more severe the pump failure
- **Right Ventricular Infarction**
  - 40% incidence with inferior or posterior MI
- **Pericarditis – can occur early or late**
  - Widespread ST elevation
  - Pain mimics MI, relieved by leaning forward
- **Thromboembolism**
  - Mural thrombi at site of infarction, especially large anterior MI
  - In atria during atrial fibrillation
  - Prevention:
    - Anticoagulation with heparin during PCI and for minimum of 48 hours
    - Anticoagulation with LMWH or fondaparinux for duration of hospitalization up to 8 days

---

### Mechanical Complications

- **LV Free Wall Rupture**
- **Papillary Muscle Rupture**
- **Septal Rupture**
Myocardial Rupture (all can cause cardiogenic shock)

- LV free wall – more common with 1st MI and large MI
  - Persistent ST elevation and persistent or recurrent chest pain; sudden profound right heart failure and shock, often progresses rapidly to PEA & death
  - Management: emergent echo-guided pericardiocentesis, surgery if blood in pericardium; fluids, inotropes, vasopressors, IABP

- Papillary muscle – posterior papillary muscle most common (only one blood supply from PDA), causes acute mitral regurgitation
  - Acute onset of hypotension and pulmonary edema, new systolic murmur, large V waves in PWP tracing
  - Management: afterload reduction (nitroprusside, IABP), diuretics, nitrates, emergent mitral valve repair if possible

- Septal – can occur with any MI, especially wrap-around LAD (ST elevation in anterior & inferior leads) and RVMI
  - Sudden onset of hypotension, RV failure due to L to R shunt, new systolic murmur
  - Management: vasodilators, inotropes, IABP; urgent surgery if cardiogenic shock, delayed surgery if no shock and stabilized medically

New systolic murmur with chest pain is suspicious for papillary muscle or septal rupture

Cardiac Tamponade

- Can occur after MI or after cardiac surgery

- Beck’s Triad
  - JVD – may increase with inspiration (Kussmaul’s sign)
  - Hypotension
  - Muffled heart sounds

- Tachycardia, decreased ECG voltage
- Narrowing of pulse pressure
- Pulsus paradoxus
- Equalizing CVP, PAD, PWP and drop in CI

- Management
  - Pericardiocentesis
  - Surgery if fluid is blood
  - Return to OR in post op CABG patient
Cardiogenic shock (pump failure)

- **Persistent hypotension** (systolic blood pressure <80 to 90 mmHg or mean arterial pressure 30 mmHg lower than baseline) with **severe reduction in the cardiac index** (<1.8 L/ min/m² without support or <2.2 L/min/m² with support) **with adequate or increased LV filling pressure** (PWP >15).
  - 15% have CS on presentation
  - 85% develop CS during hospitalization
  - Mortality rate used to be 80–90% prior to reperfusion era, now around 42–48%
- Causes: extensive infarction, RVMI, mechanical complications (ruptures)

Management of Cardiogenic Shock

- Emergency revascularization with either PCI or CABG
- Fibrinolytic therapy for patients within 24 hours of MI if revascularization is not feasible.
- Usual medical treatment of STEMI except for no beta blockers or other negative inotropes
- Inotropic and vasopressor support
  - Norepinephrine better than dopamine (higher 28 day mortality and more arrhythmias with dopamine)
  - Dobutamine or milrinone not recommended if hypotension but OK if BP can tolerate
- IABP
- Volume (especially with RVMI) or diuretics to keep PWP optimal (usually around 18–20 mmHg)
Heart Failure

Acute Pulmonary Edema

Shock

- Hypovolemic (volume problem)
- Cardiogenic (pump problem)
- Distributive (vessel problem)
- Obstructive (obstruction to cardiac filling or ejection)

Backwards Failure

Ventricular pressures rise

- **High LV pressure** is transmitted backwards into lungs (pulmonary congestion)
- **High RV pressure** is transmitted backwards into venous system (systemic venous congestion)
Forwards Failure

- Ventricles fail to pump well in a forward direction
  - **LV forwards failure** results in peripheral hypoperfusion
  - **RV forwards failure** results in failure to adequately fill the LV

Systolic Dysfunction (HFrEF)

- Impaired LV contractility results in reduced ejection fraction (< 40%), increasing end-diastolic volume and pressure
- Ventricle is dilated, thin-walled
- Due to **volume overload**
  - MI (↓ CO results in fluid retention by kidneys)
  - Aortic or mitral regurgitation
  - Congenital defects
Diastolic Dysfunction (HFpEF)

- Normal systolic function with impaired ability of the ventricle to relax and fill with blood
- Results in increased filling pressures due to stiff, noncompliant ventricles
- Increased ventricular wall thickness without dilation of chamber
- Due to **pressure overload**
  - Chronic HTN
  - Aortic stenosis
  - Hypertrophic cardiomyopathy

Pathophysiology of Heart Failure

**Physiologic Change**

<table>
<thead>
<tr>
<th>Down: ability of LV to pump blood</th>
<th>Up: LVEDV &amp; LVEDP</th>
<th>Up: Left atrial pressure</th>
</tr>
</thead>
</table>

**Signs & Symptoms**

<table>
<thead>
<tr>
<th>Fatigue, chest pain (if coronary arteries underperfused)</th>
<th>S3 and/or S4 gallop</th>
<th>Atrial arrhythmias (atrial fib)</th>
</tr>
</thead>
<tbody>
<tr>
<td>S3 and/or S4 gallop</td>
<td>Atrial arrhythmias (atrial fib)</td>
<td></td>
</tr>
</tbody>
</table>
Physiologic Change

↑ pressure in pulmonary capillaries & pulmonary artery

Leaking of fluid from pulmonary capillaries into lungs

↑ right ventricular pressure

↑ right atrial pressure

Backup of blood into systemic veins

Signs & Symptoms

↑ PA pressure & PAD

Crackles, SOB, cough, orthopnea, wheezing, hypoxia

Sternal heave, RV S₃

↑ CVP, ↑ neck veins

Peripheral edema, + abdominojugular test

Compensatory Mechanisms in Heart Failure

↓ Contractility (MI, ischemia, cardiomyopathy)

↓ Stroke Volume

↑ SNS Activation

↑ Heart rate

↑ Contractility

↑ SVR (vasoconstriction)

↑ Renin–Angiotensin – Aldosterone System

↑ Blood Volume (Na⁺ & H₂O reabsorption)

↑ SVR (vasoconstriction)

↑ CO
### 3 Clinical Profiles of Hospitalized Patients

- Patients with volume overload  
  - Pulmonary &/or systemic congestion
- Patients with profound depression of cardiac output  
  - Hypotension, renal insufficiency, other signs of hypoperfusion
- Patients with signs and symptoms of both fluid overload and shock

Most common reason for hospitalization: noncompliance with medical therapy! Other reasons for decompensation: acute MI, uncorrected hypertension, atrial fibrillation (or other arrhythmias), negative inotropic drugs, PE, NSAIDS, excessive alcohol or illicit drug use, diabetes, pneumonia, viral illness, hyper/hypothyroidism.

---

### Profiles of Advanced HF

<table>
<thead>
<tr>
<th>Congestion at Rest</th>
<th>Are lungs wet or dry? (orthopnea, JVP, rales, S3)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Dry</td>
</tr>
<tr>
<td></td>
<td>Wet</td>
</tr>
<tr>
<td>Warm</td>
<td>Warm and Dry (normal hemodynamics)</td>
</tr>
<tr>
<td></td>
<td>No congestion</td>
</tr>
<tr>
<td></td>
<td>No hypoperfusion</td>
</tr>
<tr>
<td></td>
<td>Wet</td>
</tr>
<tr>
<td></td>
<td>Warm and Wet (backwards failure)</td>
</tr>
<tr>
<td></td>
<td>Congestion</td>
</tr>
<tr>
<td></td>
<td>No hypoperfusion</td>
</tr>
<tr>
<td>Low Perfusion at Rest</td>
<td>Most patients present this way</td>
</tr>
<tr>
<td>Are extremities warm or cold?</td>
<td></td>
</tr>
<tr>
<td>Warm</td>
<td>Cold and Dry (forwards failure)</td>
</tr>
<tr>
<td></td>
<td>No congestion</td>
</tr>
<tr>
<td></td>
<td>Hypoperfusion</td>
</tr>
<tr>
<td></td>
<td>Cold and Wet (cardiogenic shock)</td>
</tr>
<tr>
<td></td>
<td>Congestion</td>
</tr>
<tr>
<td></td>
<td>Hypoperfusion</td>
</tr>
<tr>
<td></td>
<td>Least common presentation</td>
</tr>
<tr>
<td>PWP 18</td>
<td></td>
</tr>
</tbody>
</table>

Nohria et al. (2002). Medical Management of Advanced Heart Failure. *JAMA* 287, 628-640
Your patient is admitted for severe SOB, diaphoresis, and fatigue. Assessment reveals: BP 156/90, atrial fibrillation at rate in 90’s, S3 gallop, RR = 24 and struggling to breathe, crackles half way up bilaterally, neck veins elevated 5 cm above sternal angle, skin is warm and diaphoretic, 2+ pitting edema to knees. What clinical subset is he in?

A. Subset 1: warm & dry  
B. Subset 2: warm and wet  
C. Subset 3: cold and dry  
D. Subset 4: cold and wet

What therapy is indicated for him at this time?

A. Dobutamine drip & furosemide  
B. NTG drip & furosemide  
C. Nitroprusside drip  
D. Metoprolol
Therapies for Advanced HF

Congestion at Rest

Low Perfusion at Rest

Is skin warm or cold?

Wet
Preload Reduction
Diuretics (IV loops preferred)
Nitrates
Nesiritide
Ultrafiltration

Dry

Wet
Afterload Reduction
Hydralazine
Nitroprusside
Milrinone
Nesiritide

Inotropes

Inotropes

Dopamine
Dobutamine
Milrinone

Volume if hypovolemic

CI 2.2

PWP 18

Drug Therapy in Heart Failure

 próteses

Diuretics
- Loops preferred
- Thiazides
- K+ sparing

Inotropes
- Dobutamine
- Dopamine
- Milrinone
- Digoxin

Venous Dilatators
- NTG
- ACEI
- ARBs
- Nesiritide
- Morphine

Arterial Dilatators
- Nitroprusside
- Milrinone
- ACEI, ARBs
- Hydralazine
- Nesiritide

Beta Blockers
- Carvedilol
- Metoprolol
- Bisoprolol
**Types of Cardiomyopathies**

- **Dilated Cardiomyopathy**
  - Thinning of ventricular wall with dilation of chamber leading to systolic failure

- **Hypertrophic Cardiomyopathy**
  - Thickening of ventricular muscle with normal or reduced chamber size leading to diastolic failure, normal systolic function

- **Restrictive Cardiomyopathy**
  - Nondilated ventricles with restricted ventricular filling and normal systolic function

- **Idiopathic means no known cause**

**Dilated Cardiomyopathy (DCM)**

- Characterized by
  - **Systolic dysfunction** (EF <40%)
  - Ventricular dilation (one or both ventricles)

- Is main indication for cardiac transplant
  - Causes: genetic, viral myocarditis, alcohol/drugs, peripartum, chronic anemia, others

- Presents with signs of RV and LV failure

- Treatment
  - Treat cause if possible
  - Treat same as systolic HF:
    - Preload reduction: diuretics, ACEI, ARBs
    - Afterload reduction: ACEI, ARBs
    - Beta blockers: decrease risk of SCD and HF progression
    - Inotropes: digoxin long term
    - Anticoagulation
    - ICD / biventricular pacing, VADs (bridge to transplant)
Hypertrophic Cardiomyopathy (HCM)

- Genetic mutations of genes that control contractile proteins
- Diagnosed by
  - Unexplained cardiac hypertrophy – hypertrophy in the absence of an increased load (no aortic stenosis or HTN)
  - Small left ventricular cavity size
  - **Diastolic dysfunction** – stiff, noncompliant LV
  - Preserved or increased LV systolic function
  - HOCM = outflow tract obstruction by hypertrophied septum
- Causes: 60–80% are genetic; acquired secondary to aortic stenosis or chronic HTN
- Often presents as SCD

Management (symptom relief, prevent SCD)
- Avoid anything that can further decrease outflow tract size
  - Tachycardia
  - Hypovolemia
  - Increased contractility (no + inotropes)
  - Systemic vasodilation – facilitates collapse of outflow tract (no arterial dilators)

These are good:
- Beta blockers or Ca++ blockers to ↓ HR and contractility
- Antiarrhythmics – avoid AF if possible to preserve atrial kick
- Surgery (septal myectomy), alcohol ablation
- ICD to prevent SCD
Restrictive Cardiomyopathy (RCM)

- Characterized by
  - Increased stiffness of the ventricles leading to reduced diastolic filling (**diastolic dysfunction**)
  - Reduced ventricular volume
  - Normal LV wall thickness and chamber size
  - Normal systolic function
- Causes: genetic, infiltrative diseases (amyloidosis, sarcoidosis), chemotherapy, radiation
- Presents with HF **symptoms** due to high LV pressures backing up into pulmonary system and/or high RV pressures backing up into neck veins and venous system
- Management: supportive therapy to ↓ pulmonary & peripheral congestion
  - Diuretics (but avoid hypovolemia), beta blockers, Ca++ blockers
  - Maintain sinus rhythm – need atrial kick
  - Heart transplant for intractable HF

---

On blueprint: Structural Heart Defects

- Acquired
  - Valve disease

- Congenital
  - Bicuspid Valve
  - ASD
  - Patent Foramen Ovale (PFO)
  - VSD
Carol Jacobson

Valve Disease

AV Valves: Tricuspid & Mitral

Fibrous Ring (Annulus)
Leaflets
Chordae Tendineae
Papillary Muscles

Valve dysfunction can be due to calcification of leaflets, long chordae, papillary muscle ischemia, or dilation of annulus (HF, LA or LV dilation)
Valve Disease

- **Stenosis** = valve orifice narrows and forward flow is restricted. Murmur heard when valve is supposed to be open.
  - Causes **pressure overload** → hypertrophy of muscle
  - Does not occur acutely

- **Regurgitation** (or insufficiency or incompetence) = valve fails to close completely and allows backwards flow of blood. Murmur heard when valve is supposed to be closed.
  - Causes **volume overload** → dilation of chambers
  - Can occur acutely or develop over time

Compensatory changes occur over years to decades.
A new murmur that occurs acutely is **NOT** due to stenosis:
mitral or aortic regurgitation
Etiology of Valve Disease

- **Mitral Stenosis** – rheumatic fever is primary cause, calcification of annulus
- **Mitral Regurgitation** – rheumatic fever, endocarditis, trauma, papillary muscle dysfunction, LV dilation, Marfan’s Syndrome
- **Aortic Stenosis** – most common cause is calcification of valve leaflets of trileaflet valve or congenital bicuspid valve, rheumatic fever causing fusion of commissures.
- **Aortic Regurgitation** – rheumatic fever, endocarditis, Marfan’s Syndrome, aortic dissection involving valve

Aortic Stenosis
(Harsh late peaking systolic murmur)

- Most common cause is calcification of leaflets

↑ LV afterload ➔ ↑ LV pressure & hypertrophy ➔ stiff LV and ↑ LVEDP
LV wall thickens but cavity remains normal or small (diastolic dysfunction)
↑ LA pressure ➔ ↑ pulmonary pressure
Aortic Stenosis Symptoms
(develop gradually over decades)

❖ Classic Triad of Symptoms
  ▪ Angina
    • Left ventricular hypertrophy causes ↑ O₂ demand
  ▪ Syncope:
    • Normal hemodynamic response to exercise is arterial vasodilation → decreased SVR, and increased heart rate
    • Fixed obstruction of aortic valve does not allow increased cardiac output → hypotension → syncope
  ▪ Heart Failure
    • Diastolic dysfunction
    • Ultimately systolic dysfunction
  
  Once symptoms occur, risk of SCD increases and surgical Rx indicated

Aortic Stenosis
Normal size = 2–4 cm²

➢ Signs & Symptoms of ↓ LV ejection
  ▪ Effort syncope, fatigue
  ▪ Angina
  ▪ Pale, hairless skin
  ▪ Narrow pulse pressure
  ▪ Decreased urine output and mentation

➢ Signs & Symptoms of Pulmonary Congestion
  ▪ Dyspnea
  ▪ Crackles in lungs
  ▪ S₄ gallop (LA emptying into stiff LV)

➢ Treatment
  ▪ Aortic valve replacement recommended for symptomatic AS: mechanical or bioprosthetic valve
  ▪ TAVR: transcatheter AVR
  ▪ Medical Rx may be harmful: preload reduction with diuretics, ACEI or NTG can ↓ ventricular filling; afterload reduction can ↓ BP and coronary perfusion; beta blockers can ↓ HR to improve ventricular filling time but can also ↓ contractility and ability of LV to overcome restriction to forward flow
Volume overload of LV due to reflux from aorta during diastole.
LV cavity dilates to accommodate ↑ volume, and total SV increases (high systolic BP), but much of that SV regurgitates through incompetent aortic valve (low diastolic BP). Eventually systolic dysfunction and LV hypertrophy can occur.

**Acute AR:** trauma, endocarditis, dissecting arch aneurysm
- Presents as pulmonary edema or cardiogenic shock; SCD is common

**Signs & Symptoms of hyperdynamic circulation**
- Warm, flushed, reddish mucous membranes
- Wide pulse pressure (> 100 mmHg)
- Head bobbing (De–Musset sign)
- Strong rapid–rising pulse – aware of heart beat

**Treatment**
- Urgent valve replacement for acute AR and for severe AR with LV dysfunction
- Treat systolic HF with diuretics, ACEI/ARB, beta blocker, aldosterone blocker, digoxin
- Rx of HTN (ACEI/ARB, Ca++ blockers), angina, arrhythmias

No IABP!
Mitral Stenosis (Rumbling Diastolic Murmur)

↑ LA pressure → ↑ pulmonary pressure → ↑ RV pressure → ↑ RA pressure
↓ LV filling pressure = ↓ and fixed CO

Mitral Stenosis
Normal size = 4–5 cm²

- **Signs & Symptoms of ↑ LA Pressure**
  - SOB with exercise, emotional stress, pregnancy – anything that ↑ flow across valve
  - Pulmonary Edema
  - Pulmonary HTN
  - Atrial fib (poorly tolerated)

- **Signs & Symptoms of RV Failure**
  - ↑ CVP
  - ↑ neck veins
  - Liver engorgement
  - Peripheral edema

- **Exercise intolerance due to fixed CO**

- **Treatment depends of symptom severity**
  - Medical: beta blockers & Ca++ blockers to ↓ HR and improve ventricular filling; control ventricular rate and anticoagulate if AF occurs
  - Balloon valvotomy to open fused commissures
  - Surgical repair or replacement for severe stenosis
**Mitral Regurgitation**

*(High pitched, holosystolic murmur)*

- Backflow of blood into LA $\longrightarrow$ ↓ forward flow from LV
- ↑ preload of LA and LV $\longrightarrow$ atrial & ventricular dilation
- ↑ pulmonary capillary pressure $\longrightarrow$ ↑ RV pressure and RV failure

**Signs & Symptoms of ↑ LA and pulmonary pressures**
- SOB, orthopnea
- Rales
- Atrial fib
- Large “v” waves in PWP tracing

**Rapid onset with papillary muscle rupture = acute pulmonary edema & hemodynamic instability**

**Treatment**
- Acute MR: afterload reduction to ↓ pulmonary congestion and ↑ CO
- Standard Rx of HF: diuretics, ACEI/ARB, beta blocker, aldosterone blocker
- Standard Rx of HTN, A Fib
- Surgical repair or replacement for acute or severe MR
- Mitraclip or other catheter-based procedures
Congenital Structural Heart Disease

Bicuspid Aortic Valve

- Most common congenital defect in adults
- Valve can be functionally normal but begins to thicken and calcify by age 20s.
- Most patients eventually develop AS or AR
  - AS progressively develops and requires surgery in >75% of patients
  - Peak incidence of symptoms of AS occurs between ages 40–60
- Valve is susceptible to infective endocarditis
- Aortic valve replacement is the preferred treatment
  - Balloon valvotomy is an option in young patients

Atrial Septal Defect (ASD) & Patent Foramen Ovale (PFO)

- Second most common congenital defect in adults
- Usually left to right shunt through hole in septum but can reverse to right to left shunt (increases risk for stroke)
- Often asymptomatic until adulthood
- Potential complications: RV failure, atrial arrhythmias, paradoxical embolization (stroke), pulmonary hypertension.
- Indications for closure: right heart volume overload with or without symptoms (exercise intolerance, fatigue, dyspnea, heart failure, paradoxical emboli, arrhythmias).
- Percutaneous closure with Amplatzer or CardioSeal
  - ASA and clopidogrel for at least 6 months
- Surgical patch through mini thorocotomy or sternal incision
  - Beta blockers to ↓ risk of AF
  - Anticoagulation for several months to ↓ risk of thrombus on patch and if AF occurs.
**Patent Ductus Arteriosus (PDA)**
- Fetal vascular connection between the main pulmonary artery and the aorta – usually closes at birth.
- Causes left to right shunting from aorta into PA
- Symptoms depend on size of PDA: small can be asymptomatic, large creates continuous murmur; overload of pulmonary circulation, LA, and LV → LV dilation and dysfunction and pulmonary HTN.
- Large uncorrected PDA results in right to left shunting, cyanosis, heart failure, moderate risk of infective endocarditis.
- Percutaneous closure is preferred in adults unless very large PDA
  - Amplatzer device or coil
  - Antibiotic prophylaxis for 6 months
  - Surgical ligation for large PDA

**Ventricular Septal Defect (VSD)**
- Most common congenital defect at birth but only about 10% in adults (most close spontaneously)
- Moderate sized VSD causes left to right shunt, volume overload of pulmonary arteries, LA, LV.
- Large VDS cause pulmonary HTN → RV pressures can get so high that shunt reverses to right to left and cause cyanosis.
- High risk of infective endocarditis
- Surgical repair with patch closure
- Percutaneous repair with Amplatzer device
- Common post-op issues: RBBB, ventricular arrhythmias, AF, endocarditis, tricuspid regurg, persistent pulmonary hypertension
Peripheral Vascular Disease

- Acute arterial occlusion
  - Peripheral stents
  - Bypass grafts (fem-pop)

- Carotid artery stenosis
  - Carotid endarterectomy (CEA)
  - Carotid stent

- Ruptured/dissecting aortic aneurysms
Arterial Versus Venous Disease

**Acute Arterial Occlusion**
- Same risk factors as CAD
- Usually due to atherosclerosis with plaque rupture or to thromboembolism.
- Other causes: arterial trauma or dissection, vasospasm, compartment syndrome, hypercoagulable state.
- Presentation: 6 Ps
- Treatment: anticoagulation, catheter-based thrombolysis, thrombectomy, endovascular or surgical revascularization.

**Venous Thrombosis**
- Virchow’s Triad:
  - Blood stasis
  - Vascular endothelial injury
  - Inherited or acquired hypercoagulable state
- Risk factors for DVT and PE
  - Surgery, immobility
  - Central venous catheter
  - Trauma, malignancy
  - Obesity
  - Oral contraceptives, pregnancy
  - IV drug use, specific drugs
- Treatment: anticoagulation, thrombectomy, thrombolysis, IVC filter

The 6 P’s of Acute Limb Ischemia
- Pallor – pale, delayed capillary refill
- Polar – cold to touch
- Pain – usually located distally in the extremity, gradually increases in severity
- Pulseless – compare both sides
- Paresthesia – numbness, tingling sensations due to ischemic nerve dysfunction
- Paralysis – ischemic nerve dysfunction

- Indicate obstructed blood flow and should be reported immediately.
- Surgical or percutaneous intervention is often needed to salvage the limb.
- Monitor foot distal to groin insertion site with cardiac cath, IABP, peripheral vascular grafts (fem-fem, fem-pop, etc) or peripheral stent placement.
Treatment Options for Acute Limb Ischemia

- **Anticoagulation**
  - Heparin bolus and infusion immediately

- **Thrombolytic therapy**
  - Urokinase, rt-PA
  - Intra-arterial more effective than IV

- **Percutaneous revascularization**
  - Embolectomy or clot extraction
  - Angioplasty/stenting
  - Atherectomy

  Femoral access can be on one leg with intervention on the other leg. Need to monitor 6 P’s on both legs.

- **Surgical revascularization**
  - Bypass grafts: Fem–Pop, Fem–Fem
  - Endovascular stent grafts

- **Amputation**
  - For nonviable limb

Monitor for 6 Ps in both legs
Carotid Artery Stenosis

- Same risk factors as CAD
- Is a risk equivalent for CV disease
- Atherosclerosis most often occurs at bifurcation of common carotid artery
- Symptomatic carotid disease defined as:
  - Focal neurologic symptoms that are sudden in onset and referable to the appropriate carotid artery distribution (ipsilateral) within previous 6 months
  - TIA, transient monocular blindness, minor ischemic stroke
- Ischemic stroke or TIA can result from embolization, thrombosis, or decreased blood flow to brain
  - Neuro complications are 2nd most common cause of morbidity and mortality in post op cardiac patients – CEA with CABG if >50% stenosis in men and >70% in women
  - TIA precedes stroke in 50–70% of patients

Intensive medical management with statins, antiplatelet agents, treatment of hypertension and diabetes, and healthy lifestyle changes is preferred to revascularization in asymptomatic carotid artery disease

Treatment of Carotid Stenosis

- Endarterectomy (CEA)
  - Recommended over stenting if >70% stenosis in symptomatic patients and in patients >70 years
  - ASA 81–325 given pre-op and continued indefinitely
  - Clopidogrel OK if other indications (ACS, PAD)
  - Antibiotic prophylaxis prior to surgery – DC within 24h
  - Statins – ↓ risk of stroke

- Carotid Stenting (CAS)
  - Recommended in patients who are not surgical candidates and can be considered if >70% stenosis
  - BMS usually used (lower restenosis rate – larger arteries)
  - ASA or ASA & clopidogrel 48 hrs prior and at least 30 days after – ASA indefinitely
  - Antibiotic prophylaxis
  - Statins – ↓ risk of stroke

30 day stroke and death rate higher with CAS than with CEA
Postoperative Issues with CEA or Carotid Stenting

- Labile blood pressure
  - Due to manipulation and edema of carotid baroreceptors
    - Baroreceptor stimulation → reflex stimulation of vagus nerve (bradycardia) and peripheral vasodilation (hypotension)
  - Hypertension can cause suture line disruption, neck hematoma, or hyperperfusion syndrome
  - Hypotension can cause inadequate cerebral perfusion
    - May require vasopressors (neosynephrine)
    - Maintain SBP 100–150mmHg after CEA

- Bleeding
  - Hematoma in neck can compromise airway and contribute to hypotension → careful monitoring of airway & breathing

- Nerve injury
  - Facial nerves and vagus nerve potentially affected
  - Neuro checks with emphasis on face, tongue, eye movement
  - Hoarseness, impaired swallowing, abnormal gag reflex are signs of nerve damage

- Hyperperfusion syndrome
  - Due to re-established blood flow to previously low flow cerebral circulation (post-op hypertension is major risk factor)
  - Symptoms: ipsilateral headache, focal seizures, intracranial hemorrhage – evaluate with CT scan
  - Treatment: blood pressure control (nitroprusside, labetalol, NTG), anti-seizure drugs (phenytoin)
Types of Dissecting Aortic Aneurysms

- **Type A – Proximal**
  - Involves ascending aorta and arch – most dangerous
  - Tear can arise in ascending aorta, transverse arch, or descending aorta
  - Dissection can progress backwards to involve aortic valve and cause acute aortic insufficiency and cardiac tamponade
  - Dissection can involve carotid arteries and cause stroke
  - Requires early surgical repair with cardiopulmonary bypass

  New onset diastolic murmur can occur with acute aortic regurgitation

- **Type B – Distal**
  - Involves descending aorta
  - Tear arises distal to left subclavian artery
  - Dissection downwards can result in occlusion of major aortic branches and renal arteries
  - Medical management often better than surgery

- **Abdominal** – below diaphragm
  - Most common type of aneurysm
  - Infrarenal – aneurysm originates below the renal arteries
  - Juxtarenal – aneurysm originates at the level of the renal arteries
  - Suprarenal – aneurysm originates above the renal arteries
  - Risk of rupture increases with size >5.5 cm
Clinical Presentation

- **Pain**
  - Severe, sharp, stabbing posterior chest or back pain with distal dissection – often described as “tearing”
  - Anterior chest pain more common with ascending dissection
  - Abdominal pain with abdominal aneurysm
  - Can radiate anywhere in thorax or abdomen
  - May be associated with syncope, stroke, MI, or heart failure

- **Hypertension** – more common with Type B

- **Pulse deficit** – weak or absent carotid, brachial, or femoral pulse (take bilateral arm and leg BP)

- **BP difference between arms (>20 mmHg) if left subclavian or innominate/right subclavian artery involved**

- **Signs of occluded circulation: 6Ps**
  - Pallor, polar, pulseless, pain, paresthesia, paralysis

- **Symptoms of shock – hypotension, diaphoresis, tachycardia, cool clammy skin**

- **Diastolic murmur of aortic insufficiency**

- **MI can occur if coronary arteries involved in retrograde arch dissection**

- **Signs of cardiac tamponade if pericardial sac involved**

- **Oliguria due to renal artery involvement**

- **Cough, hoarseness if laryngeal nerve compressed**

- **Pulsatile abdominal mass with severe abdominal pain and hypotension may occur with abdominal aneurysm**
Management of Aortic Dissection

- Ascending aortic dissection is a surgical emergency
  - ↑ risk of acute aortic regurgitation, tamponade, MI
- Descending dissections treated medically unless occlusion of major branch, continued dissection, or evidence of rupture
  - Pain Control – morphine
  - BP Control – avoid hypertension and tachycardia
    - Goal is SBP 100–120 mmHg and HR <60 if possible
    - IV Beta blockers (to decrease HR & contractile strength)
    - Nitroprusside if SBP still >120 mmHg
    - Verapamil or diltiazem if beta blockers not tolerated
  - Evaluate cause of hypotension if present (bleeding, tamponade, heart failure)
    - Bleeding requires surgery

Aortic Aneurysm Repair

- Open Surgical Repair
  - Excision of involved segment and replacement with graft
  - Graft surrounding aneurysmal sections
- Percutaneous repair with endovascular stent grafts
  - Catheter based insertion of fabric covered stent into aorta to cover intimal flap and seal entry site of dissection from blood flow
  - Modular construction allows graft to extend into iliacs or femorals if necessary
Complications

- **Hemorrhage**
  - Aneurysm rupture, leaking grafts
  - Monitor hemodynamics, signs of low volume (low CVP, PWP, BP, UO)
  - Monitor abdominal girth
  - Monitor for increasing pain
- **Renal failure**
  - Dissection involving renal arteries or cross clamping aorta during surgery
  - Monitor UO, creatinine
- **MI**
  - Monitor chest pain, ECG changes, troponin
- **Bowel ischemia**
  - Dissection of mesenteric arteries or cross clamping during surgery
  - Monitor abdominal pain, diarrhea, white cell counts
- **Distal thromboembolism**
  - Embolization of debris from aortic manipulation
  - Monitor pedal pulses, extremity color, temp, sensation – 6 Ps
- **Spinal cord ischemia**
  - Compression of spinal vessels by aneurysm
  - Monitor sensation and movement of extremities
  - Monitor CNS function – mentation, LOC
- **Acute life-threatening elevations in BP**
  > 180/120 mm Hg with evidence of target organ damage
  - Retinal hemorrhages, exudates, or papilledema
  - Signs of cerebral edema caused by severe and sudden rises in blood pressure (hypertensive encephalopathy)
  - Presentation: headache, nausea, vomiting, and confusion
- **Other hypertensive emergencies**
  - Intracerebral hemorrhage
  - Acute MI or unstable angina
  - Acute LV failure with pulmonary edema
  - Dissecting aortic aneurysm
  - Eclampsia
  - Drugs: cocaine, amphetamines, MAO inhibitors, etc
Treatment of HTN Emergencies

- Admit to ICU
- Continuous BP monitoring

Goals of therapy for hypertensive emergencies
- Gradually reduce MAP by about 10% in the first hour and by a further 10% to 15% over the next 23 hours (maximum initial fall in BP should not exceed 25% in first 24 hours)
- IV drugs: nitroprusside, nicardipine, labetalol, esmolol, clevidipine, fenoldopam

Exceptions to gradually decreasing BP: aortic dissection, acute ischemic stroke or STEMI if candidate for thrombolysis

Therapy for Specific HTN Emergencies

- Dissecting aortic aneurysm
  - Rapidly reduce systolic BP to 100–120 mmHg within the first 20 minutes using a beta–blocker (esmolol) and a vasodilator (nitroprusside or clevidipine)

- Acute MI
  - Reduce BP more rapidly (to <180 systolic and <110 diastolic) to administer thrombolytics (NTG, esmolol, nicardipine, clevidipine)

- Acute heart failure & pulmonary edema
  - Loop diuretics and NTG or nitroprusside (avoid beta blockers and Ca++ blockers)

- Acute ischemic stroke
  - Reduce BP to <185 mmHg systolic and <110 mmHg diastolic if candidate for thrombolysis (labetalol or nicardipine are first line agents; nitroprusside if needed)
Drugs for HTN Emergencies

- **IV Vasodilators**
  - Nitroprusside (Nipride)
    - More arterial than venous dilation
  - Nicardipine (Ca++ blocker)
    - Not in acute HF (↓ contractility)
    - Caution with ischemia
  - Clevidipine (Ca++ blocker)
  - Fenoldopam (Corlepam)
  - Nitroglycerin
    - Mostly venous dilation
    - If MI or coronary ischemia
  - Enalaprilat (ACEI)
    - Acute LV failure

- **Adrenergic Inhibitors**
  - Labetalol (β & α blocker)
    - Not in acute HF
  - Esmolol (β blocker)
    - Not in acute HF
    - Aortic dissection
    - Perioperative HTN
  - Phentolamine (Regitine)
    - Catecholamine excess