Learning is FUN!!

Practice is Joy!
Prepared to handle emergencies with calm, cool, critical thinking!
3 Components

✓ Key Assessment Tools
✓ Integration with Obstructive Shock and Mechanical Emergencies
✓ Pulling it All Together
Heart Sounds
An essential assessment tool!
Auscultatory Areas

- Aortic area (2RSB)
- Pulmonic area (2LSB)
- Erb’s point (3LSB)
- Tricuspid area (5LSB)
- Mitral area (5LMCL)
Cardiac Cycle

**Ventricular Diastole**
- Phase I: Early Passive Ventricular Filling
- Phase II: Late Active Ventricular Filling

**Ventricular Systole**
- Phase I: Isovolumic Contraction
- Phase II: Ejection
Cardiac Diastole (Atrial and Ventricular): Early Passive Filling
Atrial Systole & Ventricular Diastole: Late Active Filling

Atrial Kick
Beginning Ventricular Systole: Isovolumic Contraction
Ventricular Systole: Ejection
Murmurs

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

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

0 Regurgitant Murmurs
  0 Valve does not close appropriately
  0 Heard during the part of the cardiac cycle when the valve is to be closed
Systolic Murmurs: What is Happening During Systole

- Tricuspid and Mitral Valve Closed
  - Tricuspid Regurgitation
  - Mitral Regurgitation

- Pulmonic and Aortic Valve Open
  - Pulmonic Stenosis
  - Aortic Stenosis
Aortic Stenosis
Systolic Ejection Murmur

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

- **Timing:** Holosystolic
- **Location:** Mitral area
- **Radiation:** To the left axilla
- **Configuration:** Plateau
- **Pitch:** High
- **Quality:** Blowing, harsh or musical
Between aortic stenosis and mitral regurgitation:

Which of these valvular disorders can develop acutely?
Diastolic Murmurs: What is Happening During Diastole

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

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

- **Timing:**
  - Holodiastolic if severe MS
  - Mid to Late diastole if moderate MS

- **Location:** Apex

- **Configuration:** Crescendo

- **Pitch:** Low

- **Quality:** Rumbling

- Best heard with patient in left lateral position

- Increases with isometric exercise, and expiration
Diastolic Murmurs
Aortic Regurgitation

- **Timing**: Early diastole
- **Location**: LSB 3rd ICS
- **Radiation**: Toward apex
- **Configuration**: Decrescendo
- **Pitch**: High
- **Quality**: Blowing
Between mitral stenosis and aortic regurgitation:

Which of these valvular disorders can develop acutely?
When you have Tachycardia Ask Yourself:

Why is my patient compensating?

Look to the pulse pressure for your answer.
Blood Pressure Monitoring

- **Systolic**: Maximum pressure when blood is expelled from the left ventricle
  - Represents stroke volume

- **Diastolic**: Measures rate of flow of ejected blood and vessel elasticity
  - Represents state of arterioles

- **Pulse Pressure**: Difference between systolic and diastolic pressure
Blood Pressure Assessment

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

- Low BP could be due to:
  - Low CO
    - HR too slow or too fast
    - Preload too low or too high
    - Contractility low
  - Low SVR
    - Vasodilation due to sepsis, anaphylaxis, altered neurological function, drugs
Use of Pulse Pressure

PP < 35 with tachycardia (in absence of beta blocker)

- Increased vascular tone is usually associated with compensation for low SV
  - Hypovolemic shock
    - Early sign of inadequate blood volume
  - Acute Cardiogenic shock

PP > 35 with tachycardia

- Decreased vascular tone is usually due to abnormally pathology
  - Sepsis
  - Anaphylaxis
  - Altered neurological control
Blood Pressure:
CO x SVR

BP: 88/70
- Is problem low cardiac output or low SVR
- How to treat?

BP: 82/30
- Is problem low cardiac output or low SVR
- How to treat?
To measure the pulsus paradoxus, patients are often placed in a semirecumbent position; respirations should be normal. The blood pressure cuff is inflated to at least 20 mm Hg above the systolic pressure and slowly deflated until the first Korotkoff sounds are heard only during expiration. At this pressure reading, if the cuff is not further deflated and a pulsus paradoxus is present, the first Korotkoff sound is not audible during inspiration. As the cuff is further deflated, the point at which the first Korotkoff sound is audible during both inspiration and expiration is recorded. If the difference between the first and second measurement is greater than 12 mm Hg, an abnormal pulsus paradoxus is present.

(Yarlagadda, Chakri, 2005 Cardiac Tamponade. Retrieved 3-22-06 from www.emedicine.com)
JVP (Jugular Venous Pulsation)

- Reflects volume and pressure in right side of heart
- Visual inspection
- HOB 30 - 45 degree angle
  - 45 degree angle will cause venous pulsation to rise 1 to 3 cm above the manubrium in internal jugular
Measuring JVP Pulsation

- Use targeted light
- Use centimeter ruler
  - Measure distance from angle of Louis to top column of blood
  - Draw imaginary horizontal line from column to sternal angle
Jugular Pulsation and Estimation of Right Atrial Pressure

- Normal pulsation level is $\leq 3$ cm above the sternal angle
- Sternal angle is 5 cm above right atrium
- Normal RA pressure $\leq 8$ cm $H_2O$
- Jugular venous pulsation $> 3$ cm above sternal angle
  - Increased blood volume (elevated RV preload)
  - Usually RV failure
- Other etiologies:
  - Tricuspid valve regurgitation
  - Pulmonary hypertension

Tamponade
Assessment Integration by Disease Process

Obstructive Shock and Mechanical Emergencies
Cardiac Tamponade

Who is at risk?
Classic Pericarditis
Pericardial Effusion

- Abnormal amount and/or type of fluid in the pericardial space
- Acute or chronic
- Increase capillary permeability due to inflammation may cause fluid leak into pericardial space
  - >120cc can cause tamponade if rapid
  - 2 Liters may not cause tamponade if slow
Pericardial Effusion – Signs /Symptoms

- Friction Rub
- Tachycardia
- Decreased breath sounds – if subsequent pleural effusions
- Pulsus Alternans
- Chest Pain
  - *Sit up and lean forward with pericarditis*

Pulsus alternans

- Pulses have large amplitude beats followed by pulses of small amplitude
- Rhythm remains normal
Cardiac Tamponade

- Clinical syndrome caused by accumulation of fluid in the pericardial space

- Results in reduction in ventricular filling and ultimately hemodynamic compromise

- Differentiation between pericardial effusion and tamponade is hemodynamic status.
Signs and Symptoms

- Same as with pericarditis and pericardial effusion
- Feeling of impending doom
- Beck’s Triad
  - Hypotension, Distended neck veins, Muffled heart sounds
- Equalization of filling pressures (RAP, PAD, PAOP within 5mm of each other)
- Pulses paradoxus
  - Also observed in constrictive pericarditis, severe obstructive pulmonary disease, restrictive cardiomyopathy, PE, and RV infarct with shock.

Echocardiogram
Cardiac Tamponade: Treatment

- Tamponade
  - Oxygen
  - Volume expansion
  - Bedrest with leg elevation
  - Dobutamine (increase pump without increasing SVR)
  - Avoid positive pressure mechanical ventilation (decreases venous return)

**Pericardiocentesis**
Percutaneous Surgical window *

![Image of pericardiocentesis](https://via.placeholder.com/150)
Trauma
Mechanical Ventilation
Chest Tubes

Tension Pneumothorax

Who is at risk?
Tension Pneumothorax

- Accumulation of air into the pleural space without a means of escape causes complete lung collapse and potential mediastinal shift

**Etiology**
- Blunt trauma
- Positive pressure mechanical ventilation
- Clamped or clotted water seal drainage system
- Airtight dressing on open pneumothorax
Hypotension with Mechanical Ventilation

- Sedation
- Conversion to positive pressure ventilation.
  - Assure adequate circulating fluid volume
- Development of auto PEEP
  - Increase expiration time
- Tension Pneumothorax
  - Chest tube required
Tension Pneumothorax

- **Pathophysiology**
  - Air rushes in—cannot escape pleural space
  - Creates positive pressure in pleural space
  - Ipsilateral lung collapse
  - Mediastinal shift
    - Contralateral lung compression
    - Potential tearing of thoracic aorta

- **Can also compress heart decrease RV filling**

- **Shock**
Tension Pneumothorax: Signs and Symptoms

- Anxiety / agitation
- Diminished / absent breath sounds
- Dyspnea
- Tachypnea
- If mediastinal shift:
  - Tracheal shift away from affected side
    - LATE SIGN
  - JVD
  - Hypotension
Tension Pneumothorax

Treatment

- **Oxygen (100%)**
- **Emergency decompression**
  - Perpendicular insertion of large bore needle
  - Second anterior space at mid clavicular line
  - Flutter valve to prevent atmospheric air from entering into the space
- **Chest Tube**
Pulmonary Embolus

Who is at risk?
Risk Factors for PE

- **Stasis of blood**
  - Prolonged immobilization after surgical procedures
  - Plaster casts
  - Venous obstruction
  - Heart failure / Shock / Hypovolemia
  - Varicose veins
  - Obesity

- **Hypercoagulability**
  - Polycythemia vera
  - Sickle cell disease
  - Malignancy
  - Pregnancy
  - Recent trauma
  - Oral contraceptives

- **Injury to the vascular endothelium**
  - Central venous and arterial catheters
  - Phlebitis
VTE: Incidence and Impact

- Approximately 2 million VTEs occur every year
- Each year 1 in 1000 will experience his/her first VTE
  - One third manifest pulmonary embolism (PE, with or without deep vein thrombosis [DVT])
- Death within 1 month of diagnosis:
  - 6% of DVT cases
  - 12% of PE cases
- Recurrent DVT:
  - 17% of DVT patients 2 years after initial treatment
  - 30% of DVT patients 8 to 10 years after initial Tx
Consequences of VTE: High Incidence of Mortality

- 37% of 1333 patients died on the day of onset or were dead at discovery of VTE
- PE cause of death in 10% of hospitalized patients
- 14% of hip fracture patients die of PE
  - Mortality significantly less when surgery performed within 24h of admission
- PE from DVT is leading cause of preventable hospital mortality
  - 20% of untreated proximal DVT will result in PE
  - 10-20% of these PE will be fatal
  - Anticoagulation decreases mortality 5 to 10 fold
Pulmonary Embolism

- Obstruction of blood flow to one or more arteries of the lung by a thrombus (other emboli – fat, air, amniotic fluid) lodged in a pulmonary vessel
- Lower lobes frequently affected due to increased perfusion
Pulmonary Embolism: Pathophysiology

- > 90% of thrombus develop in deep veins of iliofemoral system
  - Can also originate in the right side of the heart, pelvic veins, and axillary or subclavian veins.
  - Another source is around indwelling catheters.
- Thrombus formation leads to platelet adhesiveness and release of serotonin (vasoconstrictor).
- Dislodgement of thrombus
  - Intravascular pressure changes (standing, massaging legs, fluid challenge, valsalva maneuver).
  - Natural clot dissolution (7-10 days after development).
Pulmonary Embolism: Pathophysiology

- Clot lodges in pulmonary vessels
- Ventilation continues but perfusion decreases
  - Increase in alveolar dead space
  - Alveolar CO2 decreases
- Over perfusion of uninvolved lung results in a decreased V/Q ratio
  - Hypoxemia can occur due to ventilation / perfusion mismatching.
- Decreased blood flow damages type II pneumocytes, which results in a decrease in surfactant production. (atelectasis)
  - Pulmonary edema can develop as secondary complication
- Increased PVR can lead to pulmonary hypertension and potential acute cor pulmonale.
- Cardiogenic shock can occur as the result of right-ventricular failure.
Pulmonary Embolus: Clinical Presentation

<table>
<thead>
<tr>
<th>Large to massive when 50% of pulmonary artery bed is occluded</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Impending doom</td>
</tr>
<tr>
<td>- Hypoxemia</td>
</tr>
<tr>
<td>- Syncope</td>
</tr>
<tr>
<td>- Sign and symptoms of right heart strain or right-ventricular failure</td>
</tr>
<tr>
<td>- Signs of right-ventricular strain on ECG.</td>
</tr>
<tr>
<td>- Sudden shock</td>
</tr>
<tr>
<td>- Pulseless electrical activity</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Medium-sized emboli</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Dyspnea</td>
</tr>
<tr>
<td>- Substernal chest discomfort/pleuritic chest pain</td>
</tr>
<tr>
<td>- Many non-specific signs</td>
</tr>
<tr>
<td>- Tachypnea</td>
</tr>
<tr>
<td>- Tachycardia</td>
</tr>
<tr>
<td>- Rales</td>
</tr>
<tr>
<td>- Accentuated 2nd heart sound</td>
</tr>
<tr>
<td>- Respiratory alkalosis</td>
</tr>
</tbody>
</table>
Acute Cor Pulmonale from PE

- Transient ECG changes
- **T wave inversion** (or other ECG signs of ischemia, injury, infarction) in both inferior and anteroseptal leads
- Elevated ST segments aVR and V1-V2

**Signs of Right Heart Strain**

- ST or atrial tachycardia (or fib / flutter)
- Prominent S waves in I and aVL
- **S1, Q3, T3**
- RAD or incomplete or complete RBBB
- Widespread S waves
- Prominent P waves in inferior leads
  *(right atrial strain)*
ECG in PE
Pulmonary Embolus: Treatment

- Prevent thrombus formation
  - Compression stockings that provide a 30-40 mm Hg or higher gradient
  - Low molecular weight heparin

- Heparin or other approved anticoagulant is the treatment of choice for reducing mortality in PE
  - Initiated prior to a confirmed diagnosis
  - Slows or prevents clot progression and decreases risk of further emboli

- Fibrinolytic therapy
  - Indicated in patients with hypotension, hypoxemia, or evidence of right-ventricular strain
  - Troponin levels can also be used to guide decision-making in patients with sub-massive PE

- Pulmonary embolectomy is a surgical option when fibrinolytic therapy is contraindicated.
Pulmonary Embolus: Treatment

- Oxygen is indicated, even in the absence of hypoxemia
- Pulmonary vasodilators to help reduce pulmonary vascular resistance
- Treat right-ventricular failure with fluids and inotropes

### Obstructive Shock

- Warfarin or other approved anticoagulant
  - 3 to 6 months if there is identifiable reversible risk factor
  - Minimum of 6 six months if there is no identifiable risk factor
  - Long term with recurrent PE or in patients with ongoing risk factors

- Surgical interruption of inferior vena cava with a filter
  - Patients with contraindication to anticoagulants.
  - Recurrent thromboembolism despite anticoagulant.
  - Survivor of massive PE
Aortic Dissection

Who is at risk?
Risk Factors for Development of Thoracic Aortic Dissection

Conditions Associated With Increased Aortic Wall Stress

- Hypertension, particularly if uncontrolled
- Pheochromocytoma
- Cocaine or other stimulant use
- Weight lifting or other Valsalva maneuver
- Trauma
- Deceleration or torsional injury (e.g., motor vehicle crash, fall)
- Coarctation of the aorta
Risk Factors for Development of Thoracic Aortic Dissection

Conditions Associated With Aortic Media Abnormalities

Genetic

- Marfan syndrome
- Ehlers-Danlos syndrome, vascular form
- Bicuspid aortic valve (including prior aortic valve replacement)
- Turner syndrome
- Loeys-Dietz syndrome
- Familial thoracic aortic aneurysm and dissection syndrome
Genetic Disorders

- Marfan Syndrome

- Ehlers-Danlos Syndrome, Vascular Form

![Image of Marfan Syndrome features: normal hands vs. elongated finger and arm bones.]

![Image of Ehlers-Danlos Syndrome, Vascular Form features.]

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Genetic Disorders

- Bicuspid Aortic Valve
- Turner Syndrome

Aortic Valve
- Bicuspid
- Trileaflet

Characteristics of Turner Syndrome:
- Short stature
- Low hairline
- Fold of skin
- Constriction of aorta
- Characteristic facial features
- Poor breast development
- Elbow deformity
- Rudimentary ovaries
- Gonadal streak (underdeveloped gonadal structures)
- No menstruation
- Brown spots (nevi)
- Shield-shaped thorax
- Widely spaced nipples
- Shortened metacarpal IV
- Small finger nails
Risk Factors for Development of Thoracic Aortic Dissection

Conditions Associated With Aortic Media Abnormalities (continued)

Inflammatory vasculitides
- Takayasu arteritis
- Giant cell arteritis
- Behçet arteritis

Other
- Pregnancy
- Autosomal dominant polycystic kidney disease
- Chronic corticosteroid or immunosuppression agent administration
- Infections involving the aortic wall either from bacteremia or extension of adjacent infection

Note: Information on this slide is adapted from Table 3 in full-text version of TAD Guidelines
Issue:
Patient’s are usually asymptomatic until a catastrophic event occurs. Therefore: Identifying disease in high risk patients while still stable is a priority.
Pathophysiology

- Intimal tear
- False channel
- Risk of rupture: outer wall
- Hematoma – occlusion of branch vessels
Classification of Dissections

- Acute or chronic

**Type A Dissections:** Dissections involving the ascending aorta.

**Type B Dissections:** Dissections involving the descending thoracic aorta. These dissections begin distal to the left subclavian artery.
Complications of Dissection

0 Aortic regurgitation from retrograde dissection involving aortic valve or from aortic dilatation.
0 MI from retrograde coronary artery dissection.
0 Cardiac tamponade from ascending aorta or aortic arch rupture.
0 Intrapleural rupture from descending aortic dissection ruptures into intrapleural space – most commonly left sided.
0 Retroperitoneal bleed from rupture of abdominal aorta dissection.
0 Stroke from brachial artery compromise.
0 Paraplegia, reduced blood flow to kidneys, bowels, and lower extremities from compromise of arterial branches.
Clinical Presentation

Chest or back pain with variation in upper extremity blood pressure is key assessment finding in aortic dissection. Recurrent chest or back pain can indicate extension or rupture. The presence of aortic regurgitation in the setting of chest pain is also suspicious for aortic dissection.
Estimation of Pretest Risk of Thoracic Aortic Dissection

High Risk Conditions

- Marfan Syndrome
- Connective tissue disease*
- Family history of aortic disease
- Known aortic valve disease
- Recent aortic manipulation (surgical or catheter-based)
- Known thoracic aortic aneurysm
- Genetic conditions that predispose to AoD†

* Loeys-Dietz syndrome, vascular Ehlers-Danlos syndrome, Turner syndrome, or other connective tissue disease.

† Patients with mutations in genes known to predispose to thoracic aortic aneurysms and dissection, such as *FBN1, TGFBR1, TGFBR2, ACTA2*, and *MYH11*. 
Estimation of Pretest Risk of Thoracic Aortic Dissection

High Risk Pain Features

Chest, back, or abdominal pain features described as pain that:
• is abrupt or instantaneous in onset.
• is severe in intensity.
• has a ripping, tearing, stabbing, or sharp quality.
Estimation of Pretest Risk of Thoracic Aortic Dissection

High Risk Examination Features

• Pulse deficit
• Systolic BP limb differential > 20mm Hg
• Focal neurologic deficit
• Murmur of aortic regurgitation (new or not known to be old and in conjunction with pain)
Auscultatory Areas

Aortic area (2RSB)

1. Pulmonic area (2LSB)
2. Erb's point (3LSB)
3. Tricuspid area (5LSB)
4. Mitral area (5LMCL)
Diastolic Murmurs
Aortic Regurgitation

- **Timing**: Early diastole
- **Location**: LSB 3rd ICS
- **Radiation**: Toward apex
- **Configuration**: Decrescendo
- **Pitch**: High
- **Quality**: Blowing
Austin Flint Murmur

- Very severe chronic AR or acute AR
- Diastolic murmur: functional mitral stenosis
- Severe AR
  - Blood flow back through the aortic valve
  - Regurgitant volume presses on open anterior leaflet of mitral valve
  - Moves the leaflet towards the closed position
  - Functional Mitral Stenosis
- Timing: Mid diastolic
- Location: Cardiac apex
- Configuration: Plateau
- Pitch: Low pitch
- Quality: Rumbling
- Intensity: Soft
Risk-based Diagnostic Evaluation:
Patients with High Risk of TAD

Patients at high-risk for TAD are those that present with at least 2 high-risk features.

The recommended course of action for high-risk TAD patients is to seek immediate surgical consultation and arrange for expedited aortic imaging.

- TEE (preferred if clinically unstable)
- CT scan (image entire aorta: chest to pelvis)
- MR (image entire aorta: chest to pelvis)
MR Angiography: Acute Type B Aortic Dissection
Recommendations for Initial Management

Initial management of thoracic aortic dissection should be directed at decreasing aortic wall stress by controlling heart rate and blood pressure as follows:

a. In the absence of contraindications, intravenous beta blockade should be initiated and titrated to a target heart rate of 60 beats per minute or less.

b. In patients with clear contraindications to beta blockade, nondihydropyridine calcium channel–blocking agents should be used as an alternative for rate control.
c. If systolic blood pressures remain greater than 120mm Hg after adequate heart rate control has been obtained, then angiotensin-converting enzyme inhibitors and/or other vasodilators should be administered intravenously to further reduce blood pressure that maintains adequate end-organ perfusion.

d. Beta blockers should be used cautiously in the setting of acute aortic regurgitation because they will block the compensatory tachycardia
Recommendations for Initial Management

Vasodilator therapy should not be initiated prior to rate control so as to avoid associated reflex tachycardia that may increase aortic wall stress, leading to propagation or expansion of a thoracic aortic dissection.

Base treatment goals on highest blood pressure reading.
**Acute Aortic Dissection Management Pathway**

**Initial management of aortic wall stress**

1. **Rate/Pressure Control**
   - Intravenous beta blockade or Labetalol
     - (If contraindication to beta blockade substitute diltiazem or verapamil)
     - Titrate to heart rate <60

2. **Pain Control**
   - Intravenous opiates
     - Titrate to pain control

3. **BP Control**
   - Intravenous vasodilator
     - Titrate to BP <120mm HG
     - (Goal is lowest possible BP that maintains adequate end organ perfusion)

---

Hypotension or shock state?

- No

- Yes

Systolic BP >120mm HG?

- No

- Yes

**Secondary pressure control**

Anatomic based management
Acute Aortic Dissection Management Pathway

Anatomic based management

Type A dissection

1. Urgent surgical consultation +
   Arrange for expedited operative management

2. Intravenous fluid bolus
   • Titrate to MAP of 70mm HG or Euvolemia
   (If still hypotensive begin intravenous vasopressor agents)

3. Review imaging study for:
   • Pericardial tamponade
   • Contained rupture
   • Severe aortic insufficiency

Type B dissection

1. Intravenous fluid bolus
   • Titrate to MAP of 70mm HG or Euvolemia
   (If still hypotensive begin intravenous vasopressor agents)

2. Evaluate etiology of hypotension
   • Review imaging study for evidence of contained rupture
   • Consider TTE to evaluate cardiac function

3. Urgent surgical consultation
Occlusion of RCA by Aortic Dissection
Myocardial Rupture

Who is at Risk?
Mechanical Rupture

- Cardiac tamponade from free wall rupture
- Formation of left ventricular diverticulum or pseudoaneurysm from free wall rupture
- Left to right shunt from septal rupture
- Acute mitral regurgitation from papillary muscle rupture.

- 10% of MIs
- 15% of in hospital deaths after MI
- Without surgical intervention, the mortality rate for rupture is > 80% at two weeks.
- Two high risk periods
  - 1st 24 hours
  - Within 1st week (3 to 5 days)
- Associated with delayed fibrinolytics and late presentation
Impacting Rates of Rupture

Timely Reperfusion

- Beta blockers as soon as possible after MI unless contraindicated
- Blood pressure control in hypertensive patients
- Avoidance of non-steroidal anti-inflammatory agents.
Ventricular Septal Rupture

- Without reperfusion average time frame is 5 days (2 to 8 days) post MI
- With fibrinolytic therapy post common time frame is within 24 hours

- Septum receives blood supply from branches of LAD and PDA arteries (apical septum)
- 60% ruptures with anterior MI and 40% with inferior posterior (posterior (inferior-basal)septum)

- Can be one large or a series of smaller defects
Ventricular Septal Defect or Rupture

- **Timing:** Holosystolic
- **Location:** Left lower sternal border
- **Radiation:** Widely throughout the precordium
- **Configuration:** Plateau
- **Pitch:** High
- **Quality:** Harsh / Loud

New loud holosystolic murmur (+ thrill)
## Ventricular Septal Defect

<table>
<thead>
<tr>
<th>Left to Right Shunt</th>
<th>Medical Management</th>
<th>Surgical Considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Poor systemic perfusion</td>
<td>• IABP</td>
<td>• Direct closure or closure with patch</td>
</tr>
<tr>
<td>• Right ventricular volume overload</td>
<td>• IV nitroprusside for MAP 60 to 75 mmHg.</td>
<td>• Higher risk with posterior defects</td>
</tr>
<tr>
<td>• Heart failure</td>
<td>• Do not reduce pulmonary vascular resistance more than systemic vascular resistance.</td>
<td>• Higher mortality when done urgently</td>
</tr>
<tr>
<td>• Shock</td>
<td></td>
<td>• If unstable – mortality is higher when surgery delayed</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• State of right and left ventricle affect hemodynamic status.</td>
</tr>
</tbody>
</table>
Papillary Muscle Rupture
Papillary Muscle Rupture

5% Acute MI Mortality
Complete transection = death.

Without reperfusion: 2 to 7 days.
Reperfusion era: Median time 13 hours.

Most common with inferior posterior STEMI: Posteromedial papillary muscle receives blood supply from posterior descending artery.
Vicious Cycle

- Acute mitral valve regurgitation ➔ Backward flow ➔ Pulmonary edema
- Decreased forward flow ➔ Acute decrease in cardiac output
- Increased SVR to compensate
- More blood flow to area of least resistance
- Further decrease in cardiac output ➔ further increase in SVR
- Increase in pulmonary edema
- Further decrease in cardiac output ➔ Further increase in SVR
- Vicious Cycle
Systolic Murmurs
Mitral Regurgitation

- **Timing:** Holosystolic
- **Location:** Mitral area
- **Radiation:** To the left axilla
- **Configuration:** Plateau
- **Pitch:** High
- **Quality:** Blowing, harsh or musical

New holosystolic murmur: Often radiating to LSB.
OR
May be absent due to equalizing pressures.

If acute pulmonary edema and shock – suspect papillary muscle rupture or ischemia in absence of murmur.
Papillary Muscle Rupture

Medical Management
- Afterload reduction similar to septal rupture.

Surgical Intervention
- Mitral valve repair or replacement required.
- Operative mortality 20% - superior to medical management.
ECG 1 of 3
Free Wall Rupture

**Incidence:**

More common than ventricular septal rupture or papillary muscle rupture.

**Warning Signs:**

Recurrent chest pain and signs of regional pericarditis.

**Consequences:**

Hemipericardium, tamponade, electromechanical dissociation, and death.
Risk Factors for Free Wall Rupture

- First MI, often relatively small
- Anterior lateral wall MI
- Advanced age
- Female gender
- Hypertension during acute phase
- Absence of collateral blood flow
- Q waves on ECG
- Use of corticosteroids or non-steroidal anti-inflammatory agents (NSAIDs)
- Administration of fibrinolytics > 11 hours after symptom onset.
- Post infarction angina
Myocardial Free Wall Rupture

- Post-infarction regional pericarditis most often precedes rupture

T Wave Patterns in Post-infarction Regional Pericarditis

- Persistently positive T waves 48 hours after an MI
- Premature reversal of T wave inversion to positive
- ST segment reelevation
## ECG Changes After STEMI

<table>
<thead>
<tr>
<th>Non Reperfused</th>
<th>Reperfused</th>
</tr>
</thead>
<tbody>
<tr>
<td>• T wave enlargement</td>
<td>0 Earlier ST normalization and stabilization</td>
</tr>
<tr>
<td>• ST elevation</td>
<td>0 T wave inversion may accelerate</td>
</tr>
<tr>
<td>• Q wave formation or loss of R wave amplitude</td>
<td>0 Terminal T wave inversion initially</td>
</tr>
<tr>
<td>• ST stabilization</td>
<td>0 T waves deepen symmetrically over time</td>
</tr>
<tr>
<td>• <strong>T wave inversion</strong> <em>(within 48 - 72 hours)</em> before ST resolution</td>
<td>0 Q wave development is less pronounced or even absent</td>
</tr>
<tr>
<td>• T waves stays inverted for period of time <em>(takes weeks to months)</em></td>
<td></td>
</tr>
<tr>
<td>• Possible disappearance of Q waves</td>
<td></td>
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</tbody>
</table>
**Linking Knowledge to Practice**

- When recurrent chest pain occurs after a myocardial infarction, especially when accompanied by ST segment elevation, it is important to differentiate between re-infarction, acute in-stent thrombosis, and pericarditis. Understanding the characteristics of pericardial pain can greatly assist in the differentiation.
Types of Free Wall Rupture

Within 24 hours: Full thickness rupture.

1 to 3 days post MI: Erosion of myocardium.

Late: At border between MI and normal myocardium.
# Free Wall Rupture

<table>
<thead>
<tr>
<th>Presentation</th>
</tr>
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<tbody>
<tr>
<td>Sudden chest pain associated with coughing or straining.</td>
</tr>
<tr>
<td>Transient bradycardia and other signs and symptoms of increased vagal tone prior to rupture.</td>
</tr>
<tr>
<td>May have signs and symptoms of cardiac tamponade.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Requires urgent surgical repair.</td>
</tr>
<tr>
<td>Resection of the infarcted area</td>
</tr>
<tr>
<td>Closure of the ruptured area with a patch or with the use of biological glues</td>
</tr>
<tr>
<td>An emergency pericardiocentesis may be required to stabilize the patient (not a definitive treatment)</td>
</tr>
</tbody>
</table>
• Contained rupture by the parietal pericardium.
• Outer wall of the pseudoaneurysm is formed by the parietal pericardium and thrombus that lines the parietal wall.
• Pseudoaneurysm communicates with the left ventricle through a narrow neck.
• To-and-fro murmur may be heard.
• May result in persistent ST elevation on the ECG.
• Represents the chronic phase of a free wall rupture.
• May be clinically silent but at high risk for rupture leading to hypovolemic shock.
• Emergency surgery is considered.
The next 2 slides show the following:
1. Admission ECG for a patient with an anteroseptal / lateral wall STEMI.
2. ECG post intervention for same patient.
   1. Note: The T waves have not yet inverted post intervention. Ideally T waves will begin to invert after an intervention showing evidence of reperfusion.

**REMEMBER:** T wave must invert within 48-72 hours after a STEMI (the sooner the better). Failure of T waves to invert after a STEMI is indicative of post infarction regional pericarditis and the patient is at higher risk for myocardial rupture.
AGE IS NOT ENTERED, ASSUMED TO BE 50 YEARS OLD FOR PURPOSE OF ECG INTERPRETATION

SINUS RHYTHM

ANTEROLATERAL INFARCT, ACUTE

Q >35ms, ST >0.20mV, V2-V6

Fac: EMERGENCY DEPT (01)

- ABNORMAL ECG -

Unconfirmed Diagnosis

>>>>> Acute MI <<<<<
BORDERLINE LEFT ATRIAL ABNORMALITY
ANTEROLATERAL INFARCT, POSSIBLY ACUTE

Post Intervention

- ABNORMAL ECG -

PRELIMINARY-MD MUST REVIEW
The strip below assessing ST segments in V3 was done 48 hours post STEMI (same patient as previous 2 ECGs.). The failure of the T waves to invert is indicative of post infarction regional pericarditis with increased risk of myocardial rupture. The patient was hypotensive, which raises the concern for cardiac tamponade as the etiology of the hypotension. This assessment finding was communicated to the cardiologist.

The patient’s echocardiogram showed a large pericardial effusion and the patient subsequently underwent a surgical pericardial window.
ECG 1 of 3 Day 1 5:30 am
ECG 2 of 3 Day 2 8:30 am
ECG 3 of 3 Day 7 4:45 am
Emergency Decision Making

Checklist for Practice
Cardiac Tamponade

Risk factors: (Trauma, Post OHS / procedure, MI - lateral wall, HTN during acute phase, late presentation)

Checklist

- Beck’s triad (hypotension / JVD / muffled heart sounds)
- Pulses Paradoxus
- Pulses / Electrical Alternans
Tension Pneumothorax

Risk Factors: (Trauma, Conversion to positive pressure ventilation, existing chest tube)

Checklist

- √ Diminished to absent lung sounds
- √ Hypotension
- √ JVD
- √ Mediastinal shift (very late sign)
Pulmonary Embolus

**Risk Factors:** (Venous stasis, hypercoagulability, injury to vascular endothelium, any hospitalized patient without pharmacological prophylaxis)

**Checklist**

- √ Tachypnea (most common sign)
- √ Respiratory Alkalosis
- √ ECG signs: Right axis deviation, RBBB, Tall P waves inferior leads, T wave inversion (limb and precordial leads), Prominent S waves 1 and aVL, S1, Q3, T3
- √ ST / New atrial arrhythmia
Aortic Dissection

Risk Factors: HTN, aortic cannulation, genetic abnormalities, known aortic valve disease, known thoracic aneurysm

Checklist

√ Tearing or ripping description of chest or back pain
√ Diastolic murmur of aortic regurgitation
√ Bilateral arm BP variation
√ 4 extremity pulse variation
√ Neurological deficit or Co-existing Inferior MI
Mechanical Complications of MI

- **Septal Rupture**
  - High Risk
  - Check List
    - Anteroseptal MI
    - Loud holosystolic murmur
    - RV volume overload
    - Systemic hypoperfusion

- **Papillary Muscle Rupture**
  - High Risk
  - Check List
    - Inferior posterior or posterior MI
    - Holosystolic murmur (can be absent)
    - Acute pulmonary edema
You can make a difference