Obstructive Shock States: Recognition and Clinical Decision Making

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Obstructive Shock States: Recognition and Clinical Decision Making

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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

Cardiac Diastole (Atrial & Ventricular):
Early Passive Ventricular Filling
Atrial Systole & Ventricular Diastole:
Late Active Ventricular 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

0 Tricuspid and Mitral Valve Closed
  0 Tricuspid Regurgitation
  0 Mitral Regurgitation

0 Pulmonic and Aortic Valve Open
  0 Pulmonic Stenosis
  0 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

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**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
Aortic Regurgitation

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

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 15 mm 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
(Systolic BP – Diastolic BP)

- 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?
Pulsus Paradoxus

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 JV 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 ≤ 3 cm above the sternal angle
- Sternal angle is 5 cm above right atrium
- Normal RA pressure ≤ 8 cm H$_2$O
- 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?

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
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

- 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

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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

Who is at risk?
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
  - Ipsalateral 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
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 (eg, 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
Genetic Disorders

- Bicuspid Aortic Valve
- Turner Syndrome

Aortic Valve

Bicuspid Trileaflet

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
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

- Aortic regurgitation from retrograde dissection involving aortic valve or from aortic dilatation.
- MI from retrograde coronary artery dissection.
- Cardiac tamponade from ascending aorta or aortic arch rupture.
- Intrapleural rupture from descending aortic dissection ruptures into intrapleural space – most commonly left sided.
- Retroperitoneal bleed from rupture of abdominal aorta dissection.
- Stroke from brachial artery compromise.
- 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

1. Marfan Syndrome
2. Connective tissue disease*
3. Family history of aortic disease
4. Known aortic valve disease
5. Recent aortic manipulation (surgical or catheter-based)
6. Known thoracic aortic aneurysm
7. 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.

High Risk Pain Features

Chest, back, or abdominal pain features described as pain that:

1. is abrupt or instantaneous in onset.
2. is severe in intensity.
3. 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

[Diagram showing auscultatory areas]
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.

**Expedited aortic imaging**

- TEE (preferred if clinically unstable)
- CT scan (image entire aorta: chest to pelvis)
- MR (image entire aorta: chest to pelvis)

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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.
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? 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
Pulmonary Embolism

Who is at risk?

- 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
- 2nd most common cause of sudden death
- 3rd most common cause of death in hospitalized patient
  - 80% of unexpected hospital deaths
- Often recurrent
### Risk Factors for DVT

- Prolonged immobilization
- Recent trauma
- Plaster casts
- Burns
- Orthopedic/spine surgery
- Central venous catheters
- Pregnancy
- Oral contraceptives
- Varicose veins
- Phlebitis
- Obesity
- Dehydration/hypovolemia
- Polycythemia vera
- Sickle cell disease
- Behcet’s disease
- Deficiency in protein C, protein S, or antithrombin III
- Factor V Leiden mutation
- Heart failure
- Myocardial infarction
- COPD
- Stroke
- HIV/AIDS
- Malignancy
- Shock

*Source: Ouellette, Harrington, & Kamangar, 2013*

*Obesity is most common preventable cause of DVT.*

### Risk Factors for PE in Hospitalized Patient

- Admitted to the medical intensive care unit
- Admitted with pulmonary disease,
- Post myocardial infarction
- Post cardiopulmonary bypass surgery

*(Ouellette, Harrington, & Kamangar, 2013)*
Pulmonary Embolism

Acute

- Located centrally within the vessel lumen or causes vessel occlusion
- Results in distention of vessel wall

Chronic

- Adjoins to vessel wall
- Reduces vessel diameter by > 50%
- Recannulization through thrombus

Pulmonary Embolism

Central

- Main pulmonary artery, the left and right main pulmonary arteries, the anterior trunk, the right and left interlobar arteries, the left upper lobe trunk, the right middle lobe artery, and the right and left lower lobe arteries
- Can cause massive PE

Peripheral

- Segmental and subsegmental arteries of the three lobes of the right lung, the two lobes of the left lung, and the lingula (a projection of the upper lobe of left lung)
- Pain by initiating inflammation close to the parietal pleura.
Massive PE

- Present in less than 5% of patients presenting with PE (Kucher, Rossi, De Rosa, & Goldhaber, 2006).
- Involves both the right and left pulmonary arteries or causes hemodynamic collapse
- Presenting systolic BP of < 90 mmHg
- Mortality rates range from 30% to 60% and most deaths occur within the first 1 to 2 hours (Ouellette et al., 2013; Wood, 2002).
DVT occurs at valves of vein due to physiological abnormality
Clot can embolize or grow to occlude the vein
Embolized clot returns to right heart and into pulmonary vasculature
Lower lobes frequently affected due to increased perfusion
Additional **humoral response**
Pathophysiology

0 Increased PVR
  0 Proximal clots
  0 Substances (thromboxane A and serotonin) released in humoral response also cause vasoconstriction
  0 PA pressures double to compensate
  0 Increased work load of RV
    0 Right heart failure
    0 Leftward shift of septum
    0 Right coronary branches can be compressed

Pathophysiology

0 Increased V/Q ratio (alveolar dead space)
  0 Alveolar shrinkage (↓ CO₂ – damage Type 2 alveolar cells – loss of surfactant – atelectasis – non cardiac pulmonary edema
  0 Decreased V/Q ratio to other areas due to redistribution of blood flow
  0 Hypoxemia due to V/Q mismatching
  0 Increased minute ventilation to compensate for increased dead space – respiratory alkalosis – however, hypercapnea in massive
  0 Pulmonary infarction rare due to dual blood supply
Clinical Presentation

0 Pleuritic chest pain, shortness of breath, and hypoxemia is not present in the majority of patients
0 May have no respiratory complaint
0 Atypical presentation: flank pain, abdominal pain, delirium, syncope, and seizures

0 Potential diagnosis in any patient with respiratory symptoms in whom there is not another clear etiology

Physical Exam Findings

0 The most common physical sign, present in almost everyone with PE, is tachypnea (defined as respiratory rate > 16 per minute)
0 Other:
   0 Dyspnea, rales, cough, hemoptysis
   0 Accentuated 2nd heart sound, presence of right sided S3 or S4, new systolic murmur of tricuspid regurgitation
   0 Tachycardia, low grade fever, diaphoresis
   0 Signs of thrombophlebitis, lower extremity peripheral edema
   0 Hypoxemia, cyanosis
**More on Assessment**

**Massive PE**
- Shock presentation

**Multiple Emboli**
- More signs of pulmonary hypertension and cor pulmonale

**Diagnosis**
- The modified Wells Prediction Rule and the simplified revised Geneva Scoring System – can be used to exclude PE in the presence of a normal D-dimer (Douma et al., 2011).
- Cardiac troponins will be elevated in half of patients with moderate to large PE (Konstantinides, 2008).
- **Use of ultrasound to rule out DVT.**
- **Computed tomography angiography (CTA) has become the standard test for the diagnosis of PE.**
- VQ scan is used as alternative.
ECG in PE

- Changes in only 20% of pts
- Non specific
  - ST or atrial fibrillation
  - Small T wave inversion in limb and chest leads
- S1,Q3,T3
- RV hypertrophy
- Right axis deviation

Other:
- Large R waves in V1 and V2
- Deep S waves in leads V5 and V6
- Right atrial enlargement (tall P waves in lead II or dominant first ½ of P wave in V1)
- Incomplete right bundle branch block (RBBB)
- Delayed intrinsicoid deflection in leads V1 and V2
Treatment

- Treatment with anticoagulation in non-massive PE reduces mortality to less than 5%
- Full parenteral anticoagulation with UFH, LMWH, or fondaparinux is the priority in any patient with suspected or confirmed PE.
  - Intravenous unfractionated heparin is the drug of choice in massive PE, in patients with renal failure, and when there is concern about subcutaneous absorption.
    - An initial bolus of 80 U/kg followed by an infusion of 18 U/kg/hour
- Long term anticoagulation for at least 3 months
  - Warfarin is preferred in patients without active cancer. LMWH is preferred in patients with active cancer. Recommendations may change with more evidence of newer agents.
  - Fondaparinux and Oral factor Xa inhibitors are also use (Ouellette et al., 2013).
Treatment

- Fibrinolytic therapy is indicated in patients with a low risk for bleeding who present with hemodynamic compromise as evidenced by systolic BP < 90 mmHg.
- Catheter based pulmonary embolectomy or surgical pulmonary embolectomy are options when fibrinolytic therapy is contraindicated or when fibrinolytic therapy has failed.

Treatment

- Compression stockings are recommended for a minimum of 2 years after a DVT
  - 30 to 40 mmHg
- Chronic thromboembolic pulmonary hypertension requires long term anticoagulation. May also be candidates for a pulmonary thromboendarterectomy.
- IVC Filter:
  - Absolute contraindication to anticoagulation
  - Post survival of massive PE where subsequent PE will prove fatal
  - Presence of venous thromboembolism with adequate anticoagulation
  - May be retrievable in certain conditions
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)

<table>
<thead>
<tr>
<th>Checklist</th>
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<tbody>
<tr>
<td>√Diminished to absent lung sounds</td>
</tr>
<tr>
<td>√Hypotension</td>
</tr>
<tr>
<td>√JVD</td>
</tr>
<tr>
<td>√Mediastinal shift (very late sign)</td>
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## Pulmonary Embolus

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

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<th>Checklist</th>
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<tbody>
<tr>
<td>√Tachypnea (most common sign)</td>
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<tr>
<td>√Respiratory Alkalosis</td>
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<tr>
<td>√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</td>
</tr>
<tr>
<td>√ST / New atrial arrhythmia</td>
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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

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A Final Thought

*Impact every patient and family on their journey and provide safe passage by meeting them where they are, connecting with them in a meaningful way, and delivering care with wisdom and intention.*

- Karen
BE THE BEST THAT YOU CAN BE EVERY DAY. YOUR PATIENTS ARE COUNTING ON IT!

Handouts are available on the NTI Network today and will be available next week at www.cardionursing.com

THANK YOU!!! Have a great NTI!