Cardiovascular Emergencies: Rapid Recognition and Response

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PREPARED TO HANDLE EMERGENCIES WITH CALM, COOL, CRITICAL THINKING!
Murmur Fundamentals

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

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

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

• Pulmonic and Aortic Valve Open
  – Pulmonic Stenosis
  – Aortic Stenosis

Which of these valvular disorders can develop acutely?

BETWEEN AORTIC STENOSIS AND MITRAL REGURGITATION:
**Mitral Regurgitation**

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

**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
Which of these valvular disorders can develop acutely?

BETWEEN MITRAL STENOSIS AND 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
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

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

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

Etiology of Hypotension

Cardiac Output $\times$ SVR $\rightarrow$ Blood Pressure
Use of Pulse Pressure

- PP < 35 with tachycardia (C.O. problem)
  - Early sign of hypovolemia
  - Will also be seen with cardiogenic shock
  - Vasoconstriction is compensatory

- PP > 35 with tachycardia (SVR problem)
  - Early sign sepsis
  - Vasodilation is primary pathology

JVD (Jugular Venous Distension)

May be present in cardiac tamponade, tension pneumothorax, and large PE.
Assessment Integration by Disease Process

Obstructive Shock and Mechanical Emergencies

Cardiac Tamponade

Who is at risk?

Trauma
Post CABG
Post MI
Pericarditis / Effusion
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
  - Want to sit up and lean forward if 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.
Cardiac Tamponade: 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, tension pneumothorax, severe obstructive pulmonary disease, restrictive cardiomyopathy, PE, and RV infarct with shock.

Echocardiogram
Cardiac Tamponade: Treatment

- Supportive
  - 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 *

*Who is at risk?*

- 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

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

**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

Case Example!
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

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

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
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.
Pathophisiology

- 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.
- Intraperitoneal 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.
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.

Estimation of Pretest Risk of Thoracic Aortic Dissection

High Risk Pain Features

2. 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)

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)
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.
Recommendations for Initial Management

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

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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

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

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

**Systolic BP >120mm HG?**
- **Secondary pressure control**
  - BP Control
    - Intravenous vasodilator
    - Titrate to BP <120mm HG (Goal is lowest possible BP that maintains adequate end organ perfusion)

**Hypotension or shock state?**
- **Yes**
  - **Anatomic based management**
- **No**

**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
Pulmonary Embolus

Who is at risk?
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
- 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

* 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)

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**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).
Pathophysiology

- 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

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

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

Clinical Presentation

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

- Potential diagnosis in any patient with respiratory symptoms in whom there is not another clear etiology
Physical Exam Findings

- The most common physical sign, present in almost everyone with PE, is tachypnea (defined as respiratory rate > 16 per minute)
- Other:
  - Dyspnea, rales, cough, hemoptysis
  - Accentuated 2nd heart sound, presence of right sided S3 or S4, new systolic murmur of tricuspid regurgitation
  - Tachycardia, low grade fever, diaphoresis
  - Signs of thrombophlebitis, lower extremity peripheral edema
  - 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**
  - 90% PE come from thrombus of the deep veins.
- **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
ECG in PE

S1, Q3, T3

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
Emergency Decision Making

Checklist for Practice

Cardiac Tamponade

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

Checklist

- VBeck’s triad (hypotension / JVD / muffled heart sounds
- V Pulses Paradoxus
- V 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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<tr>
<td>√ Diminished to absent lung sounds</td>
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<tr>
<td>√ Hypotension</td>
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<tr>
<td>√ JVD</td>
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<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>
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<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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