Non-Cardiac Chest Part 2

Benjamin Schoener, MD
Clinical Assistant Professor
Central Michigan University EM Residency

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- 1.0 Signs, Symptoms and Presentations: 9%
- 2.0 Abdominal & Gastrointestinal Disorders: 8%
- 3.0 Cardiovascular Disorders: 10%
- 4.0 Cutaneous Disorders: 1%
- 5.0 Endocrine, Metabolic & Nutritional Disorders: 2%
- 6.0 Environmental Disorders: 3%
- 7.0 Head, Ear, Eye, Nose & Throat Disorders: 5%
- 8.0 Hematologic Disorders: 2%
- 9.0 Immune System Disorders: 2%
- 10.0 Systemic Infectious Disorders: 5%
- 11.0 Musculoskeletal Disorders (Non-traumatic): 3%
- 12.0 Nervous System Disorders: 5%
- 13.0 Obstetrics and Gynecology: 4%
- 14.0 Psychobehavioral Disorders: 4%
- 15.0 Renal and Urogenital Disorders: 3%
- 16.0 Thoracic-Respiratory Disorders: 6%
- 17.0 Toxicologic Disorders: 5%
- 18.0 Traumatic Disorders: 10%
- Appendix I: Procedures & Skills: 6%
- Appendix II: Other Components: 3%
- **Total: 100%**

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Angioedema Clinical Presentation

The lips, face, tongue, neck, extremities and/or genitalia can all be affected by angioedema

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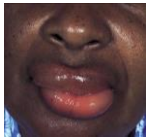
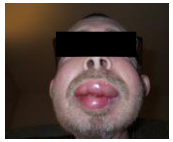

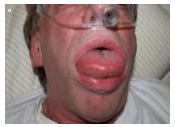

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Angioedema Pathophysiology

Angioedema is *not* always an IgE mediated allergic reaction to a stimulus

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Angioedema Clinical Presentation

The etiologies of angioedema (a *symptom*), include:

✓Hereditary Angioedema (HAE)
✓Acquired C1 Esterase Deficiency (ACID)
✓Allergic Reaction
✓ACE Inhibitor-associated angioedema

Additional patient history and physical examination will help narrow the differential dx

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Angioedema Clinical Presentation

Patients with Hereditary Angioedema (HAE) present with angioedema and **often** a family history (but **25% are de novo** mutations)

Some typical findings of an IgE mediated response (especially urticaria) are **typically** absent.

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The presence of itching and urticaria would suggest which diagnosis?

A. Hereditary Angioedema (HAE)
B. Acquired C1 Esterase Deficiency (ACID)
C. Allergic Reaction
D. ACE Inhibitor-associated angioedema
E. All of the above

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Angioedema Clinical Presentation

The presence of itching and urticaria suggest an allergic reaction.

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Angioedema Management

Fresh frozen plasma may be helpful in ACE Inhibitor associated angioedema.

Epinephrine may precipitate an acute coronary syndrome in this elderly, severely hypertensive patient with known coronary artery disease

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Angioedema Management

✓C1 esterase inhibitor [human] is indicated for HAE, not ACE inhibitor associated angioedema.

✓FFP, H1 and H2 blockers may be beneficial in ACE inhibitor associated angioedema.

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References:

US Hereditary Angioedema Association. Diagnosing HAE. http://www.haea.org/professionals/diagnosing-hae. Accessed online: 1/9/16.

Tran TP and Muelleman RL. Allergy, Hypersensitivity, and Anaphylaxis. In: Marx JA, Hockberger RS, Walls RM. eds. *Rosen's Emergency Medicine – Concepts and Clinical Practice*. 7th ed. Philadelphia, PA: Elsevier/Saunders, 2010

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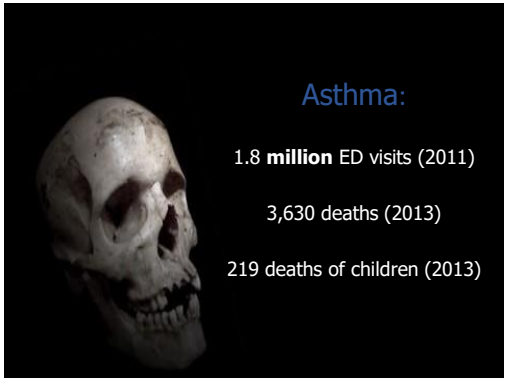

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Asthma Management

- ✓IV access for severe exacerbations
- ✓Pulse oximetry
- ✓Maintain SaO2 >90%
- ✓Nebulized albuterol solution (short acting inhaled B2 agonist)
- ✓Systemic corticosteroids for all moderate to severe attacks
- ✓Ipratropium bromide may be added to first 3 albuterol treatments for severe exacerbations
- ✓Methylxanthines (theophylline) and Leukotriene modifiers (montelukast) not recommended in acute setting

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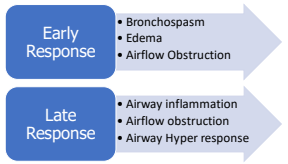
Asthma Pathophysiology



Adapted from Figure 71-3 Rosen Ch 71 p 890

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Asthma Pathophysiology

Asthma **is** a chronic inflammatory condition associated with bronchial hyper responsiveness and some reversibility

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Asthma Management

- ✓IV steroids are **not** more effective than PO steroids … unless the patient cannot tolerate oral intake
- ✓Give steroids promptly in severe attacks
- ✓Effects begin within hours (peak at 24 hours)

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Asthma Clinical Presentation

Wheezing does **not** correlate with disease severity and may be absent in a patient in extremis

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Asthma Management

- ✓When possible, PEFR or FEV1 should be measured in acute exacerbations. % Patient's personal best is most helpful
- ✓ABGs are rarely clinically useful in this setting
- ✓Patients who do not respond to usual therapy have 15% incidence of radiographically identifiable pulmonary complications (pneumothorax, pneumomediastinum)
- ✓ECG is not routinely indicated unless the patient is older or cardiovascular disease suspected

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Asthma Management

You should order **magnesium sulfate 2 gram IVPB over 20 minutes** to relax bronchial smooth muscle and dilate the airways

- ✓Subcutaneous adrenergics (epinephrine and terbutaline) do not have an advantage over aerosol but may be considered
- ✓There is no role for salmeterol or leukotriene inhibitors in the acute setting

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Asthma Clinical Presentation

Risk factors for death from asthma:

Prior Severe Exacerbation	>2 MDI albuterol canisters per month	Inner-city residence	Cardiovascular disease
2+ hospitalizations in year	Difficulty determining asthma severity	Severe psychosocial problems	Chronic lung disease
3+ ED visits in year	Low socioeconomic status	Cocaine and/or heroin use	Psychiatric disease

Adapted from Box 71-1 Rosen Ch 71 p. 893

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Asthma Management

- ✓Ketamine has bronchodilator effects (watch for increased secretions and emergency reactions)
- ✓CPAP or BIPAP may improve oxygenation and reduce fatigue
- ✓Heliox reduces airway resistance

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Asthma Management

Acute asthma should be aggressively treated to avoid hypoxia (maternal and fetal)

Maternal and fetal risk of uncontrolled asthma = high

If systemic corticosteroids used, continuous fetal monitoring is recommended at delivery

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Beware the now "calm" and "relaxed" patient with a severe asthma exacerbation

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Beware the normal ABG in a severe asthma exacerbation

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Asthma Management

Coma and apnea are absolute indications for intubation in asthma

Exhaustion, hypoxemia, altered mental status are indications for intubation

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Asthma Management

Ketamine is the preferred RSI induction agent for asthma and may be used with muscle paralysis (rocuronium 1mg/kg or succinylcholine 1.5mg/kg)

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Asthma Management

Adequate oxygenation and ventilation should be provided while minimizing elevated airway pressures, barotrauma and hypotension.

- ✓low tidal volume 6-8ml/kg
- ✓low ventilation rate (<10 breaths/min)
- ✓high inspiratory flow rates

→ Permissive Hypercapnia

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Asthma Management

Squeezing of the lateral chest may relieve breath stacking

In the setting of abruptly high airway pressures followed by cardiac arrest, the patient may have tension pneumothorax requiring tube thoracostomy

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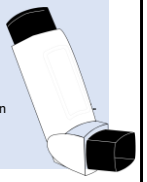
The patient's sister also has asthma and thinks you're an amazing doctor so asks you how to use her Metered-Dose inhaler properly. You tell her:

- ✓ Remove the cap from the MDI container
- ✓ Assemble the MDI and hold upright
- ✓ Shake the canister
- ✓ Place the mouthpiece loosely between the teeth
- ✓ Exhale fully
- ✓ Activate the inhaler at the beginning of a slow inhalation 6 seconds
- ✓ Hold breath 10 seconds
- ✓ Wait 1 minute before reuse



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http://www.cdc.gov/asthma/most_recent_data.htm. Accessed on: 12/19/15

http://www.cdc.gov/asthma/asthma_stats/default.htm. Accessed on: 12/19/15.

Nowak RM and Tokarski GF. Asthma. In: Marx JA, Hockberger RS, Walls RM, eds. *Rosen's Emergency Medicine – Concepts and Clinical Practice*, 7th ed. Philadelphia, PA: Elsevier/Saunders, 2010

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COPD Management

Characteristics of a COPD exacerbation:

- ✓change in the patient's baseline dyspnea, cough, or sputum
- ✓beyond day to day variations
- ✓abrupt in onset
- ✓may require change in medications

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COPD Pathophysiology

Cigarette smoking **is** the most significant risk factor for the development of COPD. However, only a **minority** of smokers develop COPD

~

airway obstruction + obliteration of pulmonary vasculature

↓

poor gas exchange

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COPD Management

The change in pulse oximetry from patient's baseline is more important than absolute levels.

Indications for admission for COPD are:

- ✓ significant worsening from baseline
- ✓ inadequate response to ED therapies
- ✓ significant comorbidities
- ✓ worsening hypoxia or hypercarbia from baseline
- ✓ insufficient home resources

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COPD Management

Beta agonists and anticholinergics are **first line** therapy for COPD exacerbation

Corticosteroids lead to a decrease in relapse and dyspnea

Antibiotics are recommended for:

- ✓increase in sputum purulence and increased dyspnea or sputum volume
- ✓for any COPD patient requiring ventilation (including non-invasive)

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COPD Management

Initial ED testing for this patient should include:

- ✓ chest radiograph to rule out pneumothorax and coexisting cardiac pathology
- ✓ sputum cultures have limited value unless you expect a superinfection
- ✓ ABGs are of limited value, especially if you do not know the patient's baseline

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COPD Management

Non-invasive ventilation can be very effective in avoiding intubation, increasing pH and lowering PCO2.

Contraindications for non-invasive ventilation include:

- ✓ Respiratory arrest
- ✓ Cardiovascular instability
- ✓ Uncooperative patient
- ✓ Upper airway obstruction
- ✓ High risk for aspiration
- ✓ Recent facial or gastroesophageal surgery
- ✓ Facial trauma
- ✓ Non fitting mask

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COPD Pathophysiology

Additional possible etiologies of a COPD exacerbation include:

- ✓atelectasis
- ✓pneumonia
- ✓pulmonary compression (obesity, ascites, pleural effusion, gastric distension)
- ✓trauma
- ✓neuromuscular disorders
- ✓other chronic pulmonary diseases
- ✓noncompliance

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COPD Clinical Presentation

Stage	Characteristics
0: At Risk	Chronic cough and sputum
	Normal Spirometry
I: Mild COPD	FEV1/FVC <70%
	FEV1 ≥80% predicted
II: Moderate COPD	FEV1/FVC <70%
	FEV1 30-80% predicted
III: Severe COPD	FEV1/FVC <70%
	FEV1 <30% predicted

Adapted from Table 73-1 Rosen 7th Ed

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The 2 interventions that alter the progression of COPD and reduce mortality are: **smoking cessation** and **oxygen therapy**.

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References

Swadron SS and Mandavia DP. Chronic obstructive pulmonary disease. In: Marx JA, Hockberger RS, Walls RM. eds. *Rosen's Emergency Medicine – Concepts and Clinical Practice*. 7th ed. Philadelphia, PA: Elsevier/Saunders, 2010

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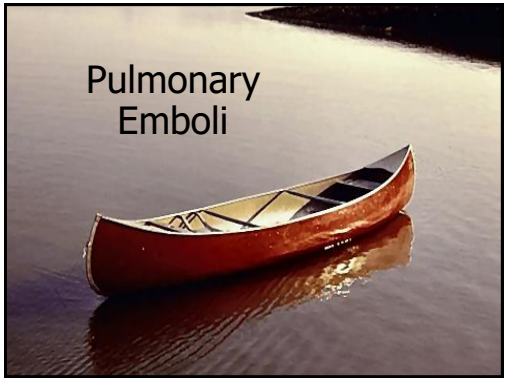

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PE Clinical Presentation

✓The PERC Rule risk stratifies (<2%) but does not "rule out" PE

✓Clinician must have a **low suspicion for PE** and the patient must fulfill **all** of the 8 criteria

1) Age <50
2) Pulse ox > 94% on room air
3) HR <100 bpm
4) No prior venous thromboembolism
5) No recent major surgery or trauma
6) No hemopytysis
7) No estrogen use
8) No unilateral leg swelling

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PE Epidemiology

The overall 30 day mortality rate for ED patients with PE is approximately 10%

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PE Clinical Presentation

~90% of patients with PE have some sensation of dyspnea
~70% of patients with PE have chest pain
~50% of patients with PE have tachycardia

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PE Management

EKG findings associated with PE include:
✓ S1 Q3 T3
✓ Tachycardia
✓ RBBB pattern
✓ Inverted T waves in V1-V4
✓ P pulmonale

Chest radiograph findings associated with PE include:
✓ Hampton's Hump and Westermark Sign

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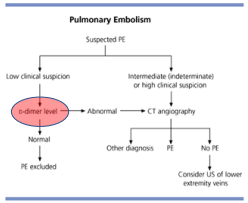
PE Clinical Presentation



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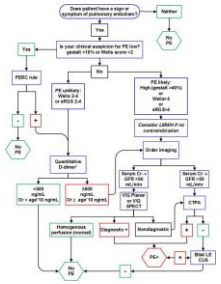

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PE Clinical Presentation

Well's PE Criteria

Clinical Factor	Points
Clinical DVT	3
PE as likely or more likely	3
Immobilization >3 days or surgery in past 4 wks	1.5
Previous DVT or PE	1.5
Heart rate > 100 bpm	1
Active malignancy	1
Hemoptysis	1

< 2 = low risk / 2-6 = moderate risk / > 6 = high risk

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PE Management

In the non-pregnant patient with normal renal function, CT angiogram is currently the test of choice for PE.

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PE Management

Heparin dosing for pulmonary embolism is:
Load 80 units/kg IV, then initiate infusion at 18 units/kg/hr

Enoxaparin dosing for pulmonary embolism is:
1mg/kg subcutaneous every 12 hours

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PE Management

Fibrinolysis is reasonable for pts with massive PE and acceptable risk of bleeding complications (IIa/B)

Fibrinolysis may be considered for pts with submassive PE judged to have clinical evidence of adverse prognosis (hemodynamic instability, worsening resp. insufficiency, severe RV dysfunction, or major myocardial necrosis) and low risk of bleeding complications (IIb/C)

Fibrinolysis is not recommended for patients with submassive PE with only mild dysfunction, i.e. low risk PEs (III/B)

Fibrinolysis is not recommended for undifferentiated cardiac arrest (III/B)

http://emcrit.org/misc/aha-pulmonary-embolism-guidelines-2011/

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References

http://emcrit.org/misc/aha-pulmonary-embolism-guidelines-2011. Accessed: 12/19/15.

Kline JA and Runyon MS. Pulmonary embolism and deep vein thrombosis. In: Marx JA, Hockberger RS, Walls RM. eds. *Rosen's Emergency Medicine – Concepts and Clinical Practice*. 7th ed. Philadelphia, PA: Elsevier/Saunders, 2010

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... and that does it for

Non-Cardiac Chest 2

best of luck on the exam!

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