Pulmonology

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PA Certification and Recertification Exam Review
June 3-6, 2014
Part I:
Infectious Disorders

• Influenza
• Acute Bronchitis
• Pneumonia
• Tuberculosis
• Epiglottitis
• Pertussis
Influenza

Background:
occurs as epidemics or pandemics (type A)
most frequently in fall / winter

Etiology:
orthomyxovirus
three antigenic subtypes
   A & B (A & B are similar clinically)
   C (milder)
transmitted via large resp droplets; incubation 1-4d
Type A further divided into subtypes based on:
   hemagglutinin (H)
   neuraminidase (N)
Influenza

Clinical Findings:
epithelial necrosis leading to bacterial superinfection (esp. with pneumococcus or S. aureus)
*abrupt* onset, fever, chills, headache, coryza, myalgias (esp. back and legs), sore throat, proteinuria, leukopenia, cervical lymphadenopathy

Diagnosis:
usually clinical (aka presumptive)
rapid Ag tests (nasal/pharyngeal)
fever and cough in areas of epidemic:
positive predictive value of 80%
## Influenza

<table>
<thead>
<tr>
<th></th>
<th>Prophylaxis</th>
<th>Treatment (w/i 48 hrs)</th>
<th>Ages</th>
</tr>
</thead>
<tbody>
<tr>
<td>amantadine</td>
<td>A</td>
<td>not recommended</td>
<td>&gt; 1</td>
</tr>
<tr>
<td>rimantadine</td>
<td>A</td>
<td>not recommended</td>
<td>&gt; 1</td>
</tr>
<tr>
<td>oseltamivir (Tamiflu)</td>
<td>A/B</td>
<td>A/B</td>
<td>&gt; 1*</td>
</tr>
<tr>
<td>zanamivir (Relenza)</td>
<td>A/B</td>
<td>A/B</td>
<td>&gt; 7*</td>
</tr>
</tbody>
</table>
Influenza

Prevention (85% with annual vaccines)

Influenza A/B vaccine for:

- over >50
- any adult or child with chronic medical problems (esp. cardiac & respiratory)
- nursing home residents
- healthcare workers
- pregnant women
- children 6-59 months
- household contacts of those above

Contraindications to vaccines: allergy to eggs, acute febrile illness, thrombocytopenia
Influenza

*Watch For…. Reye’s syndrome:*

children with A/B and VZV treated with salicylates
presents with hepatic and CNS complications
Acute Bronchitis (aka tracheobronchitis)

Etiology:
>90% are viral (rhino, corona, RSV, etc)
inflammation of airways (trachea, bronchi, bronchioles) characterized by cough

Clinical Findings:
cough with or without sputum (color not predictive of bacteria), fever, or substernal discomfort
expiratory rhonchi or wheezes

Labs/Diagnosis:
CXR: absence of markings
but.....true bronchitis difficult to distinguish from pneumonia...get a CXR!
Acute Bronchitis (*aka* tracheobronchitis)

Treatment:

antibiotics are only indicated for the following:

- elderly
- cardiopulm diseases + cough > 7-10 d
- immunocompromised

What is appropriate?

treat symptoms (OTC meds +/-)
bronchodilators if airflow obstruction
cough can persist

3 weeks in 50% of patients
>1 month in 25%
Community Acquired Pneumonia (CAP)

Background:
#1 infectious cause of death in US; #8 overall
generally acquired via aspiration of previously
colonized upper airway
acquired in the home or non-hospital environment

Etiology:
bacteria isolated more than viruses (e.g. influenza,
RSV, adenovirus, parainfluenza)

$S.\ pneumoniae$ (m.c. bacterial) > $H.\ influenzae$ > $M.\ cat$

Atypical: $Legionella$, $Mycoplasma$, & $Chlamydia$
Community Acquired Pneumonia (CAP)

Clinical Findings:

- most common signs are tachycardia/tachypnea
- fever/hypothermia; rigor/sweats;
- new cough + / ─ sputum, dyspnea
- altered breath sounds/rales
- dullness to percussion with consolidation/effusion

*but*.....chest exam alone not accurate to confirm/exclude diagnosis
Community Acquired Pneumonia (CAP)

Labs/Diagnosis: *clinical diagnosis!*
- sputum gram stain
- sputum C&S (less sensitive and specific)
- CXR: patchy, segmental lobar, multi lobar consolidation
  - no pattern is pathognomonic
- Ag studies (*Legionella, Pneumococcus*, respiratory viruses)
- blood cultures x 2 (if hospitalized)
- procalcitonin to distinguish viral vs bacterial
A very prominent pneumonia of the middle lobe of the right lung


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Community Acquired Pneumonia (CAP)

Treatment:
- outpatient: doxycycline, erythromycin macrolides (clarithro >> azithro) respiratory fluoroquinolones (if comorbid conditions)
- inpatient: coverage of *S. pneu*mo and *Legionella*  ceftriaxone (cefotaxime) plus macrolide respiratory fluoroquinolones (non ICU)

hospitalization for CAP?
- clinical judgment
- PORT classification
- consider if: age > 50 with co-morbidities, altered mental status, or hemodynamically unstable
Community Acquired Pneumonia (CAP)

Prevention: pneumococcal vaccine
  age > 65 or co-morbid conditions

Remember…

▪ Expect improvement in 48 - 72 hrs with the right antibiotic
▪ CXR may worsen but patient improves clinically
▪ Fever can last 2-5d with pneumococcus; longer with others
▪ Rales can persist > 7 days in up to 40% of patients
▪ CXR may not clear for several weeks
▪ If patient not responding to initial therapy…
  consider: virus, TB, resistant organism, *Pneumocystis* or non-infectious illness
# Community Acquired Pneumonia (CAP)

<table>
<thead>
<tr>
<th>Organism</th>
<th>Symptoms</th>
<th>Associated Conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>M. pneumoniae</em></td>
<td>- low grade fever</td>
<td>- Rabbits</td>
</tr>
<tr>
<td></td>
<td>- cough</td>
<td>- F. tularensis</td>
</tr>
<tr>
<td></td>
<td>- bullous myringitis</td>
<td>- Post splenectomy</td>
</tr>
<tr>
<td></td>
<td>- cold agglutinins</td>
<td>- Encapsulated organism</td>
</tr>
<tr>
<td><em>P. jiroveci (PCP)</em></td>
<td>- slow onset</td>
<td>- S. pneumo</td>
</tr>
<tr>
<td></td>
<td>- increased LDH</td>
<td>- H. flu</td>
</tr>
<tr>
<td></td>
<td>- more hypoxemic</td>
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</tr>
<tr>
<td></td>
<td>- than CXR seems</td>
<td></td>
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<tr>
<td></td>
<td>- “ground glass”  infiltrates</td>
<td></td>
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<tr>
<td><em>C. psittaci</em></td>
<td></td>
<td>- Leukemia</td>
</tr>
<tr>
<td></td>
<td>- psittacine birds</td>
<td>- RSV</td>
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<tr>
<td></td>
<td>- Zoonotic disease</td>
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<tr>
<td><em>S. pneumoniae</em></td>
<td>- single rigor</td>
<td>- COPD</td>
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<tr>
<td></td>
<td>- rust colored sputum</td>
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<tr>
<td><em>alcoholics</em></td>
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<tr>
<td></td>
<td>- K. pneumoniae: currant jelly sputum (dark red mucoid)</td>
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<tr>
<td><em>cystic fibrosis</em></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>- Pseudomonas</td>
<td></td>
</tr>
<tr>
<td><em>college student</em></td>
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<tr>
<td></td>
<td>- Mycoplasma</td>
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<td></td>
<td>- Chlamydia</td>
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<tr>
<td><em>air conditioning / aerosolized water</em></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>- Legionella</td>
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<tr>
<td><em>HIV/AIDS</em></td>
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<tr>
<td></td>
<td>- P. jiroveci</td>
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<tr>
<td><em>rabbits</em></td>
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Background:
- Onset of pneumonia > 72 hours after admission
- Highest risk: ICU patients on ventilation
- #2 cause of hospital acquired infection
- Mortality from 20-50%

Etiology:
- Variable geographically based on patient risk factors
- Pseudomonas, *S. aureus*, Klebsiella, *E. coli*, *Enterobacter*
Pneumonia: Nosocomial (HCAP)

Clinical Findings (same as CAP):
- most common signs are tachycardia/tachypnea
- fever/hypothermia; rigor/sweats; new cough + / — sputum, dyspnea
- altered breath sounds/rales; dullness to percussion with effusion

Diagnosis (CDC use for epidemiology):
- onset in 72 hours
- PE with rales/dullness or infiltrate on CXR
- one of following:
  - purulent sputum
  - isolated pathogen
  - Ab titers
  - histopathologic evidence of pneumonia
Pneumonia: Nosocomial (HCAP)

Labs/Diagnosis:
  blood culture
  sputum +/-
  CXR evidence of new infiltrate if VAP

Treatment:
  varies with organism, CXR findings, and Abx sensitivities; **NO UNIFORM CONSENSUS**
  empirical initially with broad coverage
    cefepime
    ticarcillin/clavulanic acid
    piperacillin/tazobactam
    meropenem
Pneumonia: HIV Related

Background:

*Pneumocystis jiroveci* (formerly PCP)

Etiology:

most common opportunistic infection assoc. with AIDS (CD4 < 200)
also occurs in patients with CA, malnourished, immunosuppressed

Clinical Findings:

typically sub acute in presentation
fever, tachypnea, SOB, non-productive cough
Pneumonia: HIV Related

Labs/Diagnosis:
- difficult to diagnose due to non-specific symptoms (fever, cough, SOB)
- CXR: cornerstone of diagnosis with diffuse or peri-hilar infiltrates
  - *no effusions seen*
- lymphopenia with low CD4 count
- sputum if possible to isolate the organism
- bronchoalveolar lavage

Treatment: TMP/SMX (or pentamidine, atovaquone, others)

Other: extremely high mortality (near 100%) if not tx primary prophylaxis
- TMP/SMX
- all AIDS patients with CD4 < 200
Tuberculosis

Background:
overall, 10% infected with TB will develop the disease

Primary TB
95% become Latent TB Infection (LTBI)
not considered infectious
cannot spread TB
asymptomatic
but have inactive TB in their body
5% become Progressive Primary TB (PPTB)

Secondary
usually reactivation TB develops from LTBI

Etiology: *M. tuberculosis*: transmitted by resp. droplets
Tuberculosis

Clinical Findings:

- may be asymptomatic

* cough is most common symptom *

- classic symptom complex: fever, drenching night sweats, anorexia, weight loss

- common pulmonary symptoms
  - cough, pleuritic chest pain, SOB, hemoptysis

*post-tussive rales are classic*

Diagnosis:

- CXR, sputum culture, acid fast stain of sputum smear

*organism necessary to obtain susceptibilities*
Tuberculosis

Labs:  Sputum: AFB
PPD:   measure induration, *not erythema*
       *positive indicates exposure* not necessarily active ds
CXR:   Primary:
       homogeneous infiltrates
       hilar/paratracheal lymph node enlargement
       segmental atelectasis
       cavitations with progressive disease (PPTB)
Reactivation:
       fibrocavitary apical ds., nodules, infiltrates
       posterior and apical segments of RUL
       apical-posterior segments of LUL
       superior segments of LL
       miliary pattern in hematogenous dissemination
Tuberculosis

*Ghon/Ranke* complexes: healed primary infection

Biopsy:
  caseating granulomas (aka necrotizing granulomas) is the histologic hallmark

Miscellaneous:
  Pott’s Disease: extrapulmonary TB (tuberculous spondylitis)
    m.c. in thoracic spine
An AP CXR of a patient with advanced bilateral pulmonary tuberculosis. It reveals the presence of bilateral pulmonary infiltrate (white triangles), and “caving formation“ (black arrows) present in the right apical region.

Source: [http://phil.cdc.gov/phil/home.asp](http://phil.cdc.gov/phil/home.asp) ID#: 2543 US Department of Health and Human Services

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## Tuberculosis

<table>
<thead>
<tr>
<th>Classification Of Positive Tuberculin Skin Test Reactions</th>
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</thead>
<tbody>
<tr>
<td><strong>Reaction Size</strong></td>
</tr>
</tbody>
</table>
| ≥ 5 mm                                                   | 1. HIV positive persons  
2. Recent contacts of those with active TB  
3. Persons with evidence of TB on CXR  
4. Immunosuppressed patients on steroids |
| ≥ 10 mm                                                  | 1. Recent immigrants from countries with high rate of TB infection  
2. HIV negative injection drug users  
3. Mycobacteriology lab personnel  
4. Residents/employees of high risk congregate settings  
5. Persons with certain medical conditions: DM, silicosis, CRF, etc.  
6. Children < 4 years of age  
7. Infants, children, adolescents exposed to adults at high risk |
| ≥ 15 mm                                                  | 1. Persons with no risk factors for TB |
Measure induration (not erythema) at 48-72 hours


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Tuberculosis

Treatment:

LTBI: (treat only after active TB is ruled out!)
*INH x 9 months or
PZA and RIF x 2 months or
*RIF x 4 months (only if in contact with TB resistant persons)

Active TB:
INH/RIF/PZA/EMB x 2 months…then,
INH/RIF x 4 months (if sensitive)
Tuberculosis

Anti-tuberculous class specific side effects

INH → hepatitis, peripheral neuropathy  
co-administer Vitamin B6 (pyridoxine)
RIF → hepatitis, flu syndrome, orange body fluids
EMB → optic neuritis, red-green vision loss
Tuberculosis

Treatment Considerations

- Multiple drugs are necessary
- Sensitivity testing is important
- Single daily dose is effective
- Prolonged treatment may be necessary
- Ensure compliance
Epiglottitis (supraglottitis)

Etiology: viral or bacterial

Signs/Symptoms: rapidly developing sore throat or odynophagia out of proportion to clinical findings

Labs: laryngoscopy, lateral films (thumb print sign)

Treatment: ceftizoxime or cefuroxime; dexamethasone
Pertussis

Background:  
• usually affects infants and young children  
• incidence increasing in adults (27% of all cases)  
• protection from childhood vaccines wears off

Etiology:  
• *Bordatella pertussis*  
• transmitted via respiratory droplets  
• incubation 6-20 days (most often 7 days)

Clinical Findings:  
• resembles common cold/bronchitis  
• “whoop” rare in adults
Pertussis

Labs:  
- PCR is current diagnostic standard
- more sensitive than culture

Treatment:  
- antibiotics to eradicate organism \textit{but does not alter course of illness}
  - erythromycin, azithromycin, clarithromycin or TMP-SMX

Prevention:  
vaccination with Tdap (instead of Td)

Prophylaxis:  
same as treatment when given within 3 weeks of onset of cough in index case
A 72 year-old male patient presents c/o acute onset of fever, with pleuritic chest pain, a single rigor, and rust colored sputum. CXR is normal. What is the most likely etiologic agent?

A. H. influenzae  
B. K. pneumoniae  
C. M. pneumoniae  
D. P. aeruginosa  
E. S. pneumoniae
Which of the following regimens is most appropriate for the treatment of active TB in immunocompetent patients?

A. INH x 6 months
B. INH+RIF x 6 months
C. INH+RIF+PZA+EMB x 2 months then INH+RIF x 4 months
D. PZA+EMB x 2 months then INH+RIF x 4 months
Part II:
Neoplastic Diseases

- Pulmonary Nodules
- Bronchogenic Carcinoma
- Carcinoid Tumors
- Metastatic (Secondary) Tumors
Solitary Pulmonary Nodule

Background:
  a.k.a. coin lesion, lung nodule
  lesion < 3cm (if > 3cm = “mass”)
  40% are malignant

Etiology:

  most are infectious granulomas: (old or active TB, fungal infection, foreign body reaction)
  carcinoma
  hamartoma
  metastasis (usually multiple)
  bronchial adenoma (95% are carcinoid tumors)
Solitary Pulmonary Nodule

Clinical Findings: most are asymptomatic

Labs/Diagnosis:
- CXR: lesion < 3 cm, isolated, rounded opacity surrounded by normal lung
- old radiographs for comparison? compare size (doubling time)
  - larger 30-500 days → malignancy
  - rapid growth < 30 days → infection
  - no growth in 2 yrs → benign
Solitary Pulmonary Nodule

Labs/Diagnosis:

CT: determine nature, location, progression, extent evaluate with CT and biopsy for diagnosis

- smooth, well defined → often benign
- ill defined/lobular → suggests CA
- spiculated/peripheral halo → often CA
# Solitary Pulmonary Nodule

## Solitary Nodule Comparison

<table>
<thead>
<tr>
<th></th>
<th>malignant</th>
<th>benign</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>age</strong></td>
<td>&gt; 45-50</td>
<td>&lt; 35</td>
</tr>
<tr>
<td><strong>calcifications</strong></td>
<td>absent to irregular calcifications</td>
<td>central/uniform/laminated or popcorn like</td>
</tr>
<tr>
<td><strong>size</strong></td>
<td>&gt;2cm</td>
<td>&lt;2cm</td>
</tr>
<tr>
<td><strong>old films</strong></td>
<td>new or larger</td>
<td>no change</td>
</tr>
<tr>
<td><strong>margins</strong></td>
<td>irregular</td>
<td>regular</td>
</tr>
</tbody>
</table>
Solitary Pulmonary Nodule

Treatment:

>35 years old: resect unless no change in 2 years

<35 years old, lesion is unchanged, can repeat study in 3-6 months
Bronchogenic CA

Background

90% of lung CA is bronchogenic leading cause of cancer deaths in men and women more deaths from lung cancer then colon, breast, and prostate combined!

5-year survival is 15%
cigarette smoking is #1 risk factor
Bronchogenic CA

Classification Scheme

SCLC: early mets & aggressive clinical course
assumes micro metastases at presentation

NSCLC: (adeno, squamous, large cell)
slower spreading
more amenable to treatment (i.e., surgery)

Clinical Findings

often presents in 50s-70s
cough, dyspnea, hemoptysis, anorexia, weight loss
Bronchogenic CA

Main histological types

**Squamous** (25-35% of cases)
- keratinization w/ keratin “pearl” formation
- centrally located, intraluminal mass
- hemoptysis is common

**Adenocarcinoma** (*m. c. with 35-40% of cases*)
- peripheral mass or nodules

**Large Cell** (5-10% of cases)
- heterogeneous group of undifferentiated tumors w/ large cells usually peripherally
- doesn’t fit in other groups
- fast doubling rates

**Small Cell** (15-20% of cases)
- bronchial origin begins centrally, infiltrating to cause bronchial narrowing/obstruction without a discreet luminal mass
Bronchogenic CA

Labs/Diagnosis:
  cytology & biopsy
  TNM classification only applies to NSCLC

Treatment:
  depends on type/extent of disease
  surgery, chemo, radiation

Other:
  common sites of metastases:
    bone, brain, adrenal glands, liver
# Bronchogenic CA

<table>
<thead>
<tr>
<th>S.P.H.E.R.E. of Lung CA Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SVC Syndrome</strong></td>
</tr>
<tr>
<td>compression of SVC: plethora, H/A, mental status changes</td>
</tr>
<tr>
<td><strong>Pancoasts Tumor</strong></td>
</tr>
<tr>
<td>tumor of the lung apex</td>
</tr>
<tr>
<td>causes Horner’s syndrome and shoulder pain</td>
</tr>
<tr>
<td>affects brachial plexus &amp; cervical sympathetic n.</td>
</tr>
<tr>
<td><strong>Horner’s Syndrome</strong></td>
</tr>
<tr>
<td>unilateral facial anhidrosis, ptosis, miosis</td>
</tr>
<tr>
<td><strong>Endocrine</strong></td>
</tr>
<tr>
<td>Carcinoid syndrome: flushing, diarrhea, telangiectasias</td>
</tr>
<tr>
<td><strong>Recurrent Laryngeal Symptoms</strong></td>
</tr>
<tr>
<td>hoarseness</td>
</tr>
<tr>
<td><strong>Effusions</strong></td>
</tr>
<tr>
<td>exudative</td>
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</tbody>
</table>
### Bronchogenic CA

#### Paraneoplastic Syndromes

<table>
<thead>
<tr>
<th>Classification</th>
<th>Syndrome</th>
<th>Histological Type</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endocrine/Metabolic</td>
<td>Cushing’s Syndrome</td>
<td>Small Cell</td>
</tr>
<tr>
<td></td>
<td>SIADH</td>
<td>Small Cell</td>
</tr>
<tr>
<td></td>
<td>Hypercalcemia</td>
<td>Squamous Cell</td>
</tr>
<tr>
<td></td>
<td>Gynecomastia</td>
<td>Large Cell</td>
</tr>
<tr>
<td>Neuromuscular</td>
<td>Peripheral neuropathy</td>
<td>Small Cell</td>
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<tr>
<td></td>
<td>Myesthenia (Eaton-Lambert)</td>
<td>Small Cell</td>
</tr>
<tr>
<td></td>
<td>Cerebellar Degeneration</td>
<td>Small Cell</td>
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<tr>
<td>Cardiovascular</td>
<td>Thrombophlebitis</td>
<td>Adenocarcinoma</td>
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<tr>
<td>Hematologic</td>
<td>Anemia</td>
<td>All</td>
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<tr>
<td></td>
<td>DIC</td>
<td>All</td>
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<tr>
<td></td>
<td>Eosinophilia</td>
<td>All</td>
</tr>
<tr>
<td></td>
<td>Thrombocytosis</td>
<td>All</td>
</tr>
<tr>
<td>Cutaneous</td>
<td>Acanthosis nigricans</td>
<td>All</td>
</tr>
</tbody>
</table>
Carcinoid Tumor

Background:
aka “carcinoid adenomas”, “bronchial gland tumors”
well-differentiated neuroendocrine tumors
men = women; usually under age 60
m.c. location: GI tract; also lung

Etiology:
low-grade malignant neoplasms
pedunculated / sessile growth in the central bronchi
Carcinoid Tumor

Clinical Findings:
- usually asymptomatic
- localized bronchial obstruction
- hemoptysis, cough, focal wheezing, recurrent pneumonia
- carcinoid syndrome
  - flushing, diarrhea, wheezing, hypotension
  - occurs in 10% of patients

Labs/Diagnosis:
- CT and octreotide scintigraphy for localization
- Bronch / CT → surgery

Treatment:
- surgical excision
- octreotide for symptoms
- most are resistant to radiation and chemotherapy
Mesothelioma

Background: primary tumors from pleural lining (80%) or peritoneum (20%)

Etiology: *history of asbestos exposure*

Clinical Findings: insidious onset of SOB, non-pleuritic chest pain, weight loss; dullness to percussion, decreased breath sounds, digital clubbing
Mesothelioma

Labs/Diagnosis:
- pleural fluid is exudative and hemorrhagic
- CXR reveals nodular, irregular, unilateral pleural thickening, and effusion
- video assisted thoracic surgery (VATS): biopsy

Treatment:
- none that are effective
- some do chemo/radiation

Other: five year survival is less than 5%
Malignant mesothelioma marked by yellow arrows

Source: http://commons.wikimedia.org/wiki/File:Tumor_Mesothelioma2_legend.jpg

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Secondary Lung Cancer

represents extra-pulmonary metastases
most frequently primary’s that mets to lung:
  breast, liver, colon
almost any CA can spread to lungs
imaging reveals multiple nodules/masses
diagnose and treat the primary tumor
Which of the following is the most common type of lung cancer in non-smokers?

A. Adenocarcinoma
B. Bronchoalveolar
C. Large cell
D. Small cell
E. Squamous cell
For which of the following types of lung cancer is surgery **not** indicated?

A. Adenocarcinoma
B. Large cell
C. **Small cell**
D. Squamous cell
Part III:
Obstructive Pulmonary Disease

- Asthma
- Bronchiectasis
- Chronic Bronchitis
- Emphysema
Obstructive Pulmonary Disease

\[ \downarrow \text{FEV/FVC} \]

Normal / \[ \uparrow \text{TLC} \]
Asthma

Background:

“reversible” airway condition characterized by:

- acute inflammation
- bronchial hyper reactivity
- mucus plugging
- smooth muscle hypertrophy

Atopy is the strongest identifiable factor:

Atopic “triad”: wheeze, eczema, seasonal rhinitis
Asthma

Etiology:
  Precipitants: allergens (esp. dust and dust mites), exercise, URI, post nasal drip, GERD, meds (beta blocker, ACEI, ASA, NSAIDS), stress, cold air

Clinical Findings:
  episodic/chronic symptoms of airway obstruction
  breathlessness, cough, wheeze
  1/3 of children have no wheeze
  prolonged expiration/diffuse wheeze
# Asthma

## Classification Of Severity

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Nighttime Symptoms</th>
<th>Lung Function</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Intermittent</strong></td>
<td>&lt; 2x/week</td>
<td>&lt; 2x/month</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FEV1 &gt; 80% predicted</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FEV1/FVC normal</td>
</tr>
<tr>
<td><strong>Mild Persistent</strong></td>
<td>&gt; 2x/week but not daily</td>
<td>3-4x/month</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FEV1 &gt; 80% predicted</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FEV1/FVC normal</td>
</tr>
<tr>
<td><strong>Moderate Persistent</strong></td>
<td>Daily</td>
<td>&gt; 1x/week but not nightly</td>
</tr>
<tr>
<td></td>
<td>Daily use of beta agonist</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>FEV1 &gt; 60% but &lt; 80% pred.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FEV1/FVC reduced 5%</td>
</tr>
<tr>
<td><strong>Severe Persistent</strong></td>
<td>Throughout the day</td>
<td>Often 7x/week</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FEV1 &lt; 60% pred.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FEV1/FVC reduced &gt; 5%</td>
</tr>
</tbody>
</table>
Asthma

Labs/Diagnosis:
- ABGs: mild hypoxia and respiratory alkalosis
- Peak Flow: diminished
- CBC: eosinophilia
- CXR: hyperinflation
- spirometry (pre and post therapy)
  - decreased FEV1/FVC (<75%)

_definitive test:_ methacholine challenge test
  (aka bronchial provocation test)
  - FEV1 decreases by > 20%
- ≥ 10% ↑ FEV with bronchodilator therapy
Asthma

Treatment:

General
remove irritants
education on peak flow measurements
desensitization
oxygen

Pharmacological
Quick relief meds
INH beta 2 agonists (e.g. albuterol)
glucocorticoids (e.g. prednisone)
anticholinergics (e.g. ipratropium)
Asthma

Long-term control therapy

INH steroids (e.g. fluticasone, budesonide)
mainstay for **persistent** asthma

Long acting bronchodilators (LABA)
INH mast cell stabilizers (e.g. cromolyn)
INH beta 2 agonists (e.g. salmeterol):
good for mild persistent or EI asthma
leukotriene inhibitors (e.g. montelukast (Singulair))
phosphodiesterase inhibitors (e.g. theophylline)
Bronchiectasis

Background:
permanenent dilation/destruction of the bronchial walls

Etiology:
congenital: Cystic Fibrosis
acquired: recurrent infections (TB, fungal infection, lung abscess) obstruction (tumor)

Clinical Findings:
foul breath, chronic cough with purulent sputum, hemoptysis, recurrent pneumonia, weight loss, anemia, persistent basilar crackles
Bronchiectasis

Labs/Diagnosis:
  Sputum smear/culture
  \textit{CXR}: tram track lung markings
  honeycombing
  atelectasis
  CT (HRCT): diagnostic test of choice
  thickened bronchial walls with dilated airways

Clinical diagnosis with radiological support
Bronchiectasis

Treatment:

- ambulatory oxygen
- aggressive antibiotics (10-14 days):
  - guided by sputum cultures or
  - empiric therapy
    - amoxicillin
    - amox-clavulante (Augmentin)
    - TMP/SMX (Bactrim)
    - ciprofloxacin
- INH bronchodilators for maintenance and acute exacerbations
- lung transplantation
Lung CT with thin slices (1 mm) showing bronchiectasis in the lower lung lobes of a subject with type ZZ alpha-1-antitrypsin deficiency. There are no signs of emphysema.

COPD: Chronic Bronchitis/Emphysema

Background:
   airflow obstruction due to chronic bronchitis or emphysema
   most patients have features of both

**Emphysema:**
   permanent air space enlargement distal to
terminal bronchiole with alveolar wall destruction

**Chronic Bronchitis:**
   increased bronchial secretions
cough for > 3 months over at least 2 years
COPD: Chronic Bronchitis/Emphysema

Etiology

- smoking/exposure to tobacco (80%)
- environmental pollutants
- recurrent URI’s
- eosinophilia
- bronchial hyper responsiveness

Labs/Diagnosis:

- PFT: normal early in the disease
decreased FEV1/FVC occur later
increased RV and TLC
confirmed by biopsy

↑ Reid index: gland layer is > 50% of total bronchial wall
# COPD: Chronic Bronchitis/Emphysema

## COPD Comparisons

<table>
<thead>
<tr>
<th>Patient type</th>
<th>Emphysema Predominant</th>
<th>Bronchitis Predominant</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>“pink puffers”</td>
<td>“blue bloaters”</td>
</tr>
</tbody>
</table>

## Clinical Findings

<table>
<thead>
<tr>
<th>Emphysema Predominant</th>
<th>Bronchitis Predominant</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hallmark:</strong> exertional dyspnea</td>
<td>mild dyspnea <strong>chronic productive cough</strong></td>
</tr>
<tr>
<td>cough is rare</td>
<td>noisy lungs: rhonchi and wheeze</td>
</tr>
<tr>
<td>quiet lungs</td>
<td>peripheral edema</td>
</tr>
<tr>
<td>no peripheral edema</td>
<td>overweight and cyanotic</td>
</tr>
<tr>
<td>thin; recent weight loss</td>
<td></td>
</tr>
<tr>
<td>barrel chest</td>
<td></td>
</tr>
<tr>
<td>pursed lips breathing</td>
<td></td>
</tr>
<tr>
<td>hyperventilation</td>
<td></td>
</tr>
</tbody>
</table>

## CXR

<table>
<thead>
<tr>
<th>Emphysema Predominant</th>
<th>Bronchitis Predominant</th>
</tr>
</thead>
<tbody>
<tr>
<td>decreased lung markings at apices</td>
<td>increased interstitial markings at bases</td>
</tr>
<tr>
<td>flattened diaphragms</td>
<td></td>
</tr>
<tr>
<td>hyperinflation</td>
<td></td>
</tr>
<tr>
<td><strong>parenchymal bullae and blebs</strong></td>
<td>diaphragms not flattened</td>
</tr>
<tr>
<td>small, thin appearing heart</td>
<td></td>
</tr>
</tbody>
</table>
CXR of patient with severe emphysema


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COPD: Chronic Bronchitis/Emphysema

Treatment:

*smoking cessation*

*oxygen improves the natural history of the disease*

bronchodilators

  #1: *ipatropium*
  #2: short acting beta agonists: albuterol
  #3: theophylline

INH steroids?

antibiotic: for AECB and acute bronchitis

  TMP/SMX; augmentin/clavulanate, doxycycline

influenza and pneumococcal vaccines

Surgery: transplant, LVRS, bullectomy
Which of the following is the most common cause of chronic bronchitis?

A. Air pollution
B. Allergies
C. Alpha 1 antitrypsin deficiency
D. Pneumonia
E. Smoking

[Bar chart showing percentages: Air pollution 0%, Allergies 1%, Alpha 1 antitrypsin deficiency 0%, Pneumonia 1%, Smoking 98%]
A 12 year-old girl with cystic fibrosis since age 3 presents c/o purulent foul smelling sputum. CXR demonstrates tubular, air-filled structures that extend to near the end of the lung fields. The remainder of the lung fields appear normal. Which of the following is the most likely diagnosis?

A. asthma
B. **bronchiectasis**
C. chronic bronchitis
D. emphysema
E. pneumonia
A 60 year-old male smoker presents c/o SOB with mild exertion. He denies cough or chest pain. Exam reveals a thin male with an increased chest A:P diameter and use of accessory muscles. On auscultation, the chest is very quiet with no adventitious sounds. Which of the following is the most likely diagnosis?

A. asthma
B. bronchiectasis
C. chronic bronchitis
D. **emphysema**
E. tuberculosis
A 24 year old woman is having an acute asthma exacerbation. Which of the following medications would be most appropriate to administer?

A. INH cromolyn  
B. INH long acting beta-2 agonist  
C. INH short acting beta-2 agonist  
D. PO leukotriene modifier  
E. PO theophylline
Break
Part IV: Restrictive Pulmonary Diseases

- Idiopathic Pulmonary Fibrosis
- Pneumoconioses
- Sarcoidosis
Restrictive Pulmonary Diseases

Normal / ↑ FEV/FVC

↓ TLC
Idiopathic Pulmonary Fibrosis

Background:

- most common dx among pts with interstitial lung disease
- includes group of distinct histopathologic features
- ensure its truly idiopathic as most ILD are due to infection, drugs, environmental/occupational exposures

Etiology: unknown

Clinical Findings:

- insidious dry cough
- exertional dyspnea
- diffuse, fine, end insp. crackles/rales ("velcro" at bases)
- clubbing
Idiopathic Pulmonary Fibrosis

Labs/Diagnosis:

CXR/HRCT:
- low lung volumes
- patchy, diffuse fibrosis
- pleural honeycombing
- biopsy helps to exclude other causes

Treatment:

controversial
corticosteroids
interferon
Pneumoconioses

Chronic lung diseases
Differentiated by origin of the precipitating agent
Etiology:
  generally industrial
  inhalation of mineral or metal dusts
  fibrotic lung develops progressively from ingestion of the agents by macrophages leading to cell injury and death
Treatment:
  generally supportive
# Pneumoconioses

<table>
<thead>
<tr>
<th>Disease</th>
<th>Occupation</th>
<th>Diagnosis</th>
<th>Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>asbestosis</td>
<td>insulation, demolition, construction,</td>
<td>BX: asbestos bodies</td>
<td>increased risk of lung CA and <em>mesothelioma</em>, esp. if smoker</td>
</tr>
<tr>
<td></td>
<td></td>
<td>CXR: linear opacities at bases and pleural plaques</td>
<td></td>
</tr>
<tr>
<td>CWP</td>
<td>coal miner</td>
<td>CXR: nodular opacities at upper lung fields</td>
<td>progressive massive fibrosis</td>
</tr>
<tr>
<td>silicosis</td>
<td>miners, sand blasters, quarry workers, stone workers,</td>
<td>CXR: nodular opacities at upper lung fields</td>
<td>increased risk of <em>TB</em>; progressive massive fibrosis</td>
</tr>
<tr>
<td>berryliosis</td>
<td>high technology fields: aerospace, nuclear power, ceramics, foundries, tool &amp; die manufacturing</td>
<td>CXR: diffuse infiltrates and hilar adenopathy</td>
<td>needs chronic steroids</td>
</tr>
</tbody>
</table>
Sarcoidosis

Background:
↑ incidence in North American blacks & Northern European whites

Etiology:
systemic disease of unknown etiology

Clinical Findings:
malaise, fever, slowly progressing dyspnea, cough
pulmonary findings are limited
extra-pulmonary findings common:
erothe\thema nodosum
parotid gland enlargement
Sarcoidosis

Labs: ACE levels elevated
CXR → bilateral hilar adenopathy
hypercalcemia
Diagnosis: biopsy shows non-caseating granulomas
Treatment: prednisone
Other: “GRUELING”
- Granulomas
- RA
- Uveitis
- Erythema nodosum
- Lymphadenopathy
- Interstitial fibrosis
- Negative TB test
- Gammaglobulinemia
Which of the following is an example of a restrictive lung disease?

A. Asthma
B. Bronchiectasis
C. COPD
D. Cystic fibrosis
E. Sarcoidosis

Which is the most common restrictive lung disease?
Which of the following restrictive lung diseases has an increased risk of mesothelioma if the patient is a smoker?

1. **Asbestosis**
2. Berryliosis
3. Coal workers pneumoconiosis
4. Silicosis
Part V: Pleural Diseases

- Pleural Effusion
- Pneumothorax
Pleural Effusions

Background

abnormal fluid collection in the pleural space
25% of effusions are associated with malignancy

Important to distinguish transudate from exudate
Pleural Effusions

Etiology: 5 types of effusions

exudates: “leaky capillaries”
these three cause 80%
para-pneumonic
malignancy
PE (but 20% as transudate)
infection (TB), malignancy, trauma

transudates: “intact capillaries”
CHF (90%), atelectasis, renal/liver ds. (cirrhosis)

empyemas: direct infection of an exudate
hemothorax: trauma
chylothorax: TB
Pleural Effusions

Clinical Findings:

- often asymptomatic
  
  *progressive dyspnea on exertion and pleuritic chest pain*

- presentation is variable
  
  - asymptomatic $\rightarrow$ small effusion
  
  - dyspnea/cough $\rightarrow$ large effusion

- percussion dullness
  
- decreased tactile fremitus
  
- diminished/absent breath sounds

- b/l (transudates) vs. unilateral (exudates)
Pleural Effusions

Labs:
- imaging helps define extent of effusion
  - lateral decubitus (free flowing vs. loculated fluid)
  - upright (blunting of costophrenic sulcus)
- CT scan for small effusions

Diagnosis:
- thoracentesis is the gold standard
  - send for protein, LDH, pH, total & cell counts, glucose cytology?
  - Gram stain with C & S?
Massive Left-Sided Pleural Effusion


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Pleural Effusions

Treatment:

Transudates:
- correct underlying condition
- therapeutic thoracentesis if severe dyspnea

Exudates:
- drainage for empyemas
- pleurodesis for malignant pleural effusions
Pleural Effusions

Other:

transudates vs. exudates (Light’s criteria)

exudate if meets any one of the following:

- pleural fluid protein/serum protein ratio > 0.5
- pleural fluid LDH / serum LDH ratio > 0.6
- pleural fluid LDH > 2/3 upper limit of normal for serum LDH (a cut-off value of 200 IU/L was used previously)
Pneumothorax

Background: accumulation of air in pleural space

Etiology/Classifications:

Spontaneous (1° or 2°)
- Primary: (PSP) occurs in absence of underlying ds. tall, thin males (rupture of apical blebs)
- Secondary: (SSP) underlying ds. COPD, asthma, CF, ILD

Traumatic: penetrating/blunt trauma (incl. iatrogenic)

Tension pneumothorax: medical emergency!
- penetrating trauma, CPR, pos pressure ventilation: lung collapse
  → contra lateral mediastinal shift
  → hypotension 2° impaired v. return
Pneumothorax

Clinical Findings:
  Spontaneous:
    ipsilateral, unilateral chest pain, sudden and pleuritic, dyspnea, cough
    absent/diminished breath sounds
    hyper resonance
    decreased tactile fremitus
    if small, exam is unimpressive
  Tension: (in addition to above…)
    respiratory distress, falling SaO2, hypotension, distended neck veins, tracheal deviation
Pneumothorax

Labs/Diagnosis:

*end expiratory chest film reveals visceral pleural air*

Tension: air on affected side with contralateral mediastinal shift

Treatment:

Primary spontaneous/secondary:

<15% diameter of hemithorax on CXR: rest, cough, chest pain relief, serial CXRs

>15%: chest tube plus above measures

Tension: immediate needle decompression 2nd ICS at MCL
Bilateral pneumothorax (larger arrows)


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Left tension pneumothorax

Source: http://clinicalcases.blogspot.com/2004/02/tension-pneumothorax.html

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A 54 year-old patient presents with acute onset of chest pain, SOB and hypotension. CXR reveals a mediastinal shift to the right. Which of the following is the most appropriate next step?

A. Admit to hospital for observation  
B. Insert an airway  
C. Order pulmonary consult  
D. Perform immediate needle decompression

3% 4% 0% 94%
Which of the following is the most likely cause of an transudative effusion?

A. Cirrhosis
B. Mesothelioma
C. Pneumonia
D. Tuberculosis
Part VI:
Pulmonary Circulation

• Pulmonary Thromboembolism
• Pulmonary Hypertension
• Cor Pulmonale
• ARDS
Rudolf Virchow by Hugo Vogel, 1861

Source: http://www.kunsttexte.de/download/bwt/werner.pdf

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Pulmonary Embolism (PE)

Background
- occlusion of pulmonary arterial circulation from an embolized substance
- #3 leading cause of death in hospitalized pts.

Risk Factors: (Dr. Rudolf Virchow’s triad)
- hypercoagulable state: (e.g. CA)
- venous stasis (e.g. prolonged rest/cast)
- vascular intimal inflammation / injury (e.g. surgery/trauma)
- surgical procedures: orthopedic, pelvic, abdominal CA, OCPs, pregnancy
Pulmonary Embolism (PE)

Etiology: most are from thrombus

- 95% deep calf veins that propagate proximal to popliteal / ileofemoral veins
- Risk of PE greater with proximal thrombus
- Others:?
  - air → central lines
  - amniotic fluid → active labor
  - fat → long bone (femur) fx

Negative workup in 25-50% patients for VTE
Pulmonary Embolism (PE)

Clinical Findings:

Homans’ sign: low sensitivity/specificity
calf pain with passive, forcible, dorsiflexion of
foot with knee flexed

variable, signs and symptoms are non-specific but <3% chance of PE in absence of dyspnea with
tachypnea or pleuritic chest pain

most emboli are clinically silent

most common symptom: dyspnea (sudden; 85% with RR>16) / pain on inspiration

(*consider PE in any hospitalized patient with acute SOB)

most common sign: tachycardia (60% with P > 100)
Pulmonary Embolism (PE)

Labs/Diagnostic Evaluation:

ECG: *not diagnostic!*
- sinus tachycardia (most common)
- atrial dysrhythmias, PEA
- S1-Q3-T3 (inverted T wave) is rare (20%)

ABGs: hypoxemia (but 15% have PaO2 > 80)

Elevated D-Dimer

Plasma levels of degraded fibrinogen

*negative D-Dimer (with low clinical suspicion) → strong evidence against DVT*
Pulmonary Embolism (PE)

CXR (abnormalities may be subtle / absent)

*m.c. abnormality is atelectasis at bases*

**Westermark’s Sign**: focal oligemia (vasoconstriction) in the embolized zone

**Hampton’s Hump**: (classic finding)

wedge shaped infarct

VQ scans:

“normal” practically rules out PE

“abnormal” is non specific
categorized along with CXR

normal/very low
low
intermediate
high probability
Chest Spiral CT (with and without contrast agent) showing multiple filling defects of principal branches, due to acute and chronic pulmonary embolism


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Pulmonary Embolism (PE)

Spiral CT (helical) angiography
more sensitive than VQ, but less than pulmonary arteriography
less sensitive in the distal segmental arteries

Pulmonary Arteriography
shows intraluminal filling defect
“Gold Standard”

LE Venous Doppler (not good for dx of PE)
most commonly used
incompressible veins (absence of “wink”)
90-94% sensitivity in proximal (less in distal)

Venography
“Gold Standard” for diagnosis of LE DVT
Pulmonary Embolism (PE)

Treatment:

anticoagulation: generally 3-6 months
heparin (UFH: sig is TID) → coumadin (INR 2-3x normal)
LMWH (sig is QD)
thrombolytic therapy (streptokinase, alteplase, urokinase): reserved for hemodynamically unstable patient
not generally recommended

IVC filter
surgery: only for saddle emboli
Pulmonary Embolism (PE)

Other:

Prevention:

combination of mechanical & pharmacological measures
early ambulation
intermittent pneumatic compression
low dose heparin
LMWH
Pulmonary Hypertension

Background:

- Pulmonary artery pressure rises to a level inappropriate for a given cardiac output
- Self-perpetuating once initiated
- Women > men
- 30-50 yo
Pulmonary Hypertension

Etiology:

primary (idiopathic) hypertension (PPH) is rare
most frequently: secondary pulmonary HTN (COPD, connective tissue disorder esp. scleroderma)
increased pulmonary venous pressure
constrictive pericarditis, LV failure, mitral stenosis, mediastinal disease compressing pulmonary veins
decreased area of pulmonary arterial bed
vasoconstriction
loss of vessels
lung resection, emphysema, ILD, CVD
vessel obstruction
Pulmonary Hypertension

Clinical Findings:
- dull/retrosternal chest pain (angina-like), dyspnea, fatigue, effort syncope
- difficult to diagnose early
- signs/symptoms are often related to underlying cause

Labs: polycythemia
- EKG: right axis deviation, RVH, RAE, right ventricular strain
Pulmonary Hypertension

Diagnosis: Multifactorial

Work-up:

- CXR/CT: increased vasculature
- PFTs: underlying airflow obstruction or restricted lung volumes
- ECHO: RVH, estimated pulmonary artery pressure
catheterization to determine degree of HTN
- others (VQ scan, serology: conn tissue disorders, etc.)
Pulmonary Hypertension

Treatment:

- underlying cause
  - oxygen if from COPD
  - anticoagulants if from emboli
  - diuretics/salt restriction for cor pulmonale
- vasodilators?
  - epoprostenol (PGI2)
  - prostacyclin
Cor Pulmonale

Background:
Failure of the right side of the heart caused by prolonged high blood pressure in the pulmonary artery (pulmonary HTN) and right ventricle of the heart. RV enlargement leads to RV failure

Etiology:
if acute think P.E.; if chronic think COPD pulmonary vascular disease (PE, vasculitis, ARDS) respiratory disease obstructive (asthma, COPD) restrictive (ILD, lung resection)
Cor Pulmonale

Clinical Findings:
  - fatigue, exertional dyspnea, and syncope with exertion
    increase in chest diameter
  - labored respiratory efforts with retractions of the chest wall
  - hyper resonance to percussion
  - diminished breath sounds
  - wheezing
  - distant heart sounds
  - cyanosis (rarely)
Cor Pulmonale

Labs/Diagnosis:

   CXR
   EKG: RAD > 30°; flat, inverted T waves in RV precordial leads

Treatment:

   oxygen
decrease pulm. Vasc. resistance and pulmonary HTN
treat underlying disorder
Acute Respiratory Distress Syndrome

Clinical Definition:
acute (12-18 hours) hypoxemic respiratory failure after a systemic or pulmonary insult without heart failure

Physiological Definition
bilateral diffuse pulmonary infiltrates
normal PCWP (<18 mmHg)
PaO2/FiO2 < 200

Etiology:
most common (one-third of patients): sepsis
others: toxic inhalation, near drowning, aspiration, etc.
Acute Respiratory Distress Syndrome

Clinical Findings:
- respiratory distress, tachypnea, fever, crackles, rhonchi

Labs:
- CXR: diffuse pulmonary infiltrates *that spares the costophrenic angles*
- air bronchograms in 80%
- *normal* heart size

Diagnosis: no biochemical tests to define ARDS
- clinical dx that excludes cardiogenic pulmonary edema
Acute Respiratory Distress Syndrome

Treatment:
- underlying cause *plus* supportive care
- support cardiac output with inotropes, cautious fluids
- mechanical ventilation
  - PEEP: lowest levels to recruit atelectic alveoli
  - PaO2 > 60
  - FIO2: < 60%
  - SaO2 > 90%

Other:
- ARDS mortality: 30-40%
- ARDS *plus* sepsis mortality: 90%
A 52 year-old patient presents to the ER with pleuritic chest pain, cough, dyspnea, and hemoptysis. On exam she is anxious with tachycardia and tachypnea. Lab work demonstrates an elevated D-dimer. CXR is normal. What is the most likely diagnosis?

A. Emphysema
B. Myocardial infarction
C. Pneumonia
D. Pulmonary embolism
E. Tension pneumothorax
Which of the following is not recommended for primary prevention of pulmonary emboli in the immediate post-op period?

A. Ambulation
B. Compression stockings
C. Heparin
D. Pneumatic compression
E. Thrombolytics
Good luck!