THE RESPIRATORY CENTER

- See Fig 29-23.
- 2 Groups of neurons in the medulla:
  - **1) Dorsal-** Controls inspiration- moves the diaphragm via the phrenic nerve- “C 3-4-5 keep the diaphragm alive.”
  - **2) Ventral-** control inspiration and expiration via the intercostal and abdominal muscles.
- Also involved: the pneumotaxic center and the apneustic center in the pons.
THE RESPIRATORY CENTER

- Control via:
- 1) **An automatic component**- via the chemoreceptors and the lung receptors. More later.
- 2) **A voluntary component**- integrates breathing w/ speaking, singing, blowing etc., once initiated by the motor cortex, by causing a temporary cessation of automatic breathing.
CHEMORECEPTORS

- Monitor levels of O2 and CO2 → transmitted to the resp. center → ventilation adjusted to maintain normal O2 and CO2.

- 2 TYPES:
  - 1) CENTRAL.
  - 2) PERIPHERAL.
CENTRAL CHEMORECEPTORS

- Located in the respiratory center in the medulla.
- Sense changes in CO2.
- **Read the text re how this is mediated by ↑ H+ in the CSF.**
- Sensitive to **short-term** changes in CO2.
- Long term → H+ is buffered by bicarb, so the stimulus from ↑ CO2 is wiped out.
- Now, can respond only to ↓ O2 in the peripheral chemoreceptors.
PERIPHERAL CHEMORECEPTORS

- Located in the carotid and aortic bodies.
- Sense changes in O2.
- Exert little control until PaO2 is below 60 mm Hg
- Patients w/ chronic CO2 retention rely on hypoxia rather than hypercapnia to drive respiration → giving O2 to these patients can depress respiration.
LUNG RECEPTORS

- Sense changes in airway resistance and lung expansion.
- 3 types of receptors:
  1) **Stretch receptors** - adjust resp. rate (and work) acc. to lung compliance and airway resistance.
  2) **Irritant receptors** - bronchoconstriction & shallow breathing in resp. to irritants, sighing, yawning.
  3) **Juxtacapillary** or J receptors - rapid, shallow breathing in resp. to pulm. congestion, edema.
THE COUGH REFLEX.

- Protection against “foreign stuff”: irritants, secretions.
- Mediated by receptors in the tracheobronchial wall.
- Receptors $\rightarrow$ vagus n. $\rightarrow$ medulla $\rightarrow$ deep inspiration $\rightarrow$ closed glottis $\rightarrow$ contraction of abd. & exp. muscles $\rightarrow$ expulsive cough.
- Interfered w/ by lotsa stuff $\rightarrow$ see text $\rightarrow$ weakened m., paralysis, drugs, etc.
DYSPNEA

- Dyspnea = SOB = breathlessness.
- The perception of difficult, labored breathing.
- **3 general categories of disease:**
  1) **Respiratory disease**: pneumonia, asthma, COPD.
  2) **Cardiac disease** resulting in pulmonary congestion: CHF.
  3) **Neuromuscular disorders**: myasthenia, M.D.
DYSPNEA

- Cause unknown- see text for 4 theories.
- Best measure / assessment of breathing ability → change in level of daily activity.
SINUSITIS

- SINUSES: frontal ethmoid, sphenoid, and maxillary.
- Most common cause: conditions that obstruct drainage thru the ostia, such as w/ URI or allergies.
- Also: polyps, barotrauma, swimming, etc.
- 2 EXCITING FLAVORS:
- ACUTE & CHRONIC.
ACUTE SINUSITIS

- ACUTE VIRAL SINUSITIS- resolves within 5-10 days.
- ACUTE BACTERIAL SINUSITIS- Sx’s worsen after 5 days or persist beyond 10. Congestion pressure, headache, purulent drainage, possibly fever.
- RECURRENT ACUTE SINUSITIS- 4 or more episodes within 12 months.
ACUTE SINUSITIS

- A lot of cases of acute bacterial sinusitis begin as a viral URI or acute viral sinusitis, and the fluid within the sinuses from this becomes secondarily infected, leading to acute bacterial sinusitis.

- Presentation of this:
CHRONIC SINUSITIS

- Signs / Sx’s- fever, pain absent. Congestion, fullness, post-nasal drip, hoarseness, cough, headache, unpleasant breath.
- Mucosal changes can become irreversible.
ETIOLOGY OF SINUSITIS

- ACUTE BACTERIAL - *H. flu.*, *Strep. pneumonia*.

- CHRONIC SINUSITIS - anaerobes, *Peptostreptococcus, Fusibacterium, Prevotella*, alone or in combination w/ aerobes such as *Strep.* or *Staph. aureus.*
COMPLICATIONS OF SINUSITIS

- Intracranial and orbital extension.
DIAGNOSIS OF SINUSITIS

- Criteria exist.
- Essentially, the Dx of a 1st episode of what appears to be straight-forward, uncomplicated sinusitis can be made on clinical grounds, without the need for imaging studies.
DIAGNOSIS OF SINUSITIS

- For cases that are recurrent, or do not seem straight-forward and uncomplicated, imaging studies are advised.
- This would be in order to confirm the Dx of the non-straight-forward case, or to look for reasons for recurrences: polyps, ostial occlusion, tumor, etc.
- Studies: CT, MRI, less so the “Sinus Series.”
THE PNEUMONIAS

- PNEUMONIA- inflammation of the lung parenchyma- alveoli, bronchioles.

- Most common cause of death from infection, 6th most common cause of death overall.

3 FLAVORS:

- 1) COMMUNITY-ACQUIRED.
- 2) HOSPITAL-ACQUIRED.
- 3) PNEUMONIA IN THE IMMUNOCOMPROMISED.
COMMUNITY-ACQUIRED PNEUMONIA

- As opposed to those acquired in a hospital, nursing home.


- See text re classification system by the American Thoracic Society.
COMMUNITY-ACQUIRED PNEUMONIA

- Signs / Sx’s: fever, malaise, chest discomfort, cough, increased resp rate, can progress to respiratory failure and sepsis.
HOSPITAL-ACQUIRED PNEUMONIA

- 2\textsuperscript{nd} most common hospital-acquired infection.
- 20-50\% mortality.
- 90\% are bacterial.
- Organisms: *Pseudomonas aeruginosa, Staph. aureus, Enterobacter, Klebsiella, E. coli, Serratia*.
- Highly antibiotic-resistant.
PNEUMONIA IN THE IMMUNOCOMPROMISED

- Defects of humoral immunity (B-Cell)- bacterial infections
- Defects of cellular immunity (T-Cell)- viruses, fungi, protozoa, mycobacteria.
- Neutropenia, impaired granulocyte function → *Staph. aureus, Aspergillus, gram-negative bacilli, Candida.*
- Time course: fulminant → bacterial; insidious → viral, fungal, protozoal, mycobacterial.
E = MICE$^2$

Einstein's cat.
TB

- PRIMARY VS. SECONDARY.

- PRIMARY = “LATENT” TB = INACTIVE = NON-CONTAGEOUS.

- SECONDARY = “ACTIVE” TB = REACTIVATION OF PRIMARY TB
TB

**PRIMARY TB**

- In a person w/ no prior exposure to TB.
- Inhaled droplet nuclei w/ the tubercle bacillus.
- Cell-mediated response $\rightarrow$ formation of a granuloma (Ghon’s focus) $\rightarrow$ tubercle $\rightarrow$ spread to hilar lymph nodes $\rightarrow$ caseous necrosis.
- Ghon’s complex- the granuloma + the lymph nodes.
TB

**PRIMARY TB**

- If cellular immunity intact $\rightarrow$ infection contained, remains latent / dormant $\rightarrow$ but can become active TB $\rightarrow$ active TB is infectious, latent is not.

- Treatment of latent TB (prophylaxis) greatly reduces the likelihood of progression to active TB.

- Usually asymptomatic $\rightarrow$ + PPD, calcifications on CXR, hilar adenopathy.

- In HIV $\rightarrow$ progression to active disease w/out latent period.
TB

- **Miliary TB** → hematogenous dissemination → lesions similar to millet seeds → brain, kidneys, liver, meninges, marrow
TB

SECONDARY TB

- Reinfection, or reactivation of latent, primary TB.
- Reactivation occurs with impaired defenses.
- Cellular immunity $\rightarrow$ cavitation.
- Signs / Sx’s: fever, night sweats, anorexia, weight loss, fatigue. “Consumption.”
- Cough: dry initially, then productive $\rightarrow$ purulent, hemoptysis (late).
TB

- TB- a case scenario / example.
LUNG CANCER

RISK FACTORS

- Smoking.
- Industrial exposure- asbestos.
- Familial.
- Diets deficient in fruits and veggies, anti-oxidants.
LUNG CANCER

MANIFESTATIONS

- Variable.
- 3 Categories:
  1) Manifestations from involvement of the lung and adjacent structures.
  2) Manifestations from local and metastatic spread.
  3) Manifestations from paraneoplastic syndromes.
- Anorexia, weight loss, cough, hemoptysis
LUNG CANCER

INVolVEMENT OF THE LUNG AND ADJACENT STRUCTURES

- Local irritation, obstruction of airways, involvement of the mediastinum and pleura.
- Chronic cough, SOB, wheezing.
- Hemoptysis.
- Pain w/ involvement of the pleura, mediastinum, and vessels.
INvolvement of the Lung and Adjacent Structures

MediaStinal Involvement

- Hoarseness - recurrent laryngeal nerve.
- Dysphagia - pressure on the esophagus.
- Superior vena cava syndrome.

Pleural Involvement

- Pleural effusion → compression of the lung → atelectasis, dyspnea.
METASTATIC SPREAD

- Lymphatic, vascular spread.
- 50% have mets at time of Dx, eventually 90% develop metastatic disease.
- Brain, bone, liver.
- Will go into staging and survival rates in Biomedical Treatment of Disease I. In a nutshell, it ain’t good.
PARANEOPLASTIC SYNDROMES.

- Manifestations involving endocrine, neurologic, and connective tissue function.
- Related to cellular undifferentiation.
- Ectopic PTH- hypercalcemia.
- ACTH- Cushing’s.
- SIADH.
- Migratory thrombophlebitis, DIC.
- Paraneoplastic syndromes may precede and lead to the Dx.
There's a worm in my swill.

That's the early bird special.
PLEURAL PAIN

- Pleura- visceral and parietal.
- Accompanies pleuritis, from pleurisy, respiratory infections, etc.
- Abrupt in onset, sharp, unilateral, lower lateral aspect of the chest.
- Worse w/ chest movement (“pleuritic”)—coughing, deep breathing, from rubbing together of the inflamed pleural surfaces.
- Resp. are shallow and rapid w/ splinting.
PLEURAL PAIN

- Auscultation → pleural friction rub.
VS. NON-PLEURAL CHEST PAIN

- **Musculoskeletal**- from coughing, barfing, etc. Bilateral, inferior rib cage where abd. muscles insert. Worse w/ contractions of abdominal m. rather than chest m.

- **Bronchial**- substernal, dull, worse w/ coughing but not w/ deep inspiration (pleura not involved).

- **Myocardial**- substernal, not affected by respiration.
PLEURAL EFFUSION

- Abnormal collection of fluid in the pleural space.
- Normally only 10-20 cc in the pleural space, a “potential space” drained by lymphatics.
- Effusion develops when the rate of fluid formation exceeds the rate of removal.
- 5 exciting mechanisms.
PLEURAL EFFUSION

5 EXCITING MECHANISMS

1) ↑ capillary pressure- CHF.
2) ↑ capillary permeability- inflammation
3) ↓ osmotic pressure- from ↓ albumin.
4) ↑ negative intra-pleural pressure- atelectasis.
5) Impaired lymphatic drainage- metastatic cancer to the mediastinum.
PLEURAL EFFUSION

- HEMOTHORAX - BLOOD.
- EMPYEMA – PUS.
- CHYLOTHORAX - LYMPH.
PLEURAL EFFUSION

MANIFESTATIONS

- Vary according to the cause.
- Space-occupying, prevents lung expansion.
- Dyspnea, constant or pleuritic chest pain
- Mediastinal shift.
- Percussion → dullness, “shifting dullness,” flatness.
- Auscultation → ↓ breath sounds.
PLEURAL EFFUSION

The standard of care for a new pleural effusion is to tap it (thoracentesis) to evaluate the fluid and determine its characteristics: infection / TB, malignancy, transudate, exudate, etc. (more in Biomedical Treatment of Disease I).
PNEUMOTHORAX

- Mechanism- air escapes into the pleural space (a “potential space”) via rupture of a “bleb” on the surface of the lung, trauma to the lung, or trauma to the chest wall.

- 3 TYPES:
  1) SPONTANEOUS PNEUMOTHORAX.
  2) TRAUMATIC PNEUMOTHORAX.
  3) TENSION PNEUMOTHORAX.

- There’s also (4) IATROGENIC.
PNEUMOTHORAX

**SPONTANEOUS**

- **PRIMARY**- in otherwise healthy people, from rupture of a bleb. Cause of the bleb, why they rupture are unknown. More common in tall males.

- **SECONDARY**- More serious, occurs in people w/ underlying lung disease (damaged tissue → rupture): emphysema, asthma, CHF, etc., further compromises already compromised lung fxn.

- **CATAMENIAL**- occurs during menses. ? related to endometriosis on the pleura or diaphragm.
PNEUMOTHORAX

In these types, air accumulates in the pleura, while the lung “collapses,” until there is no longer a gradient forcing air in, or until collapse causes the leak to seal.
PNEUMOTHORAX

TRAUMATIC

- Penetrating (knife, bullet) or non-penetrating (broken rib) injuries.
- May also have hemothorax.
- Severity dependent upon the nature of the injury.
PNEUMOTHORAX

IATROGENIC

- From us poking a needle somewhere and penetrating the lung: thoracentesis, acupuncture, biopsy, etc.
- Can also occur from a difficult intubation and aggressive ventilation, where the damage can not only to the alveoli but to the trachea, bronchi, etc.
PNEUMOTHORAX

TENSION

- The “sucking chest wound.”
- Air accumulates rapidly in the pleural space but doesn’t escape, and continues to accumulate shifting the mediastinum (w/ the trachea etc) to the right, compression of the vena cava → impairment of venous return.
PNEUMOTHORAX

MANIFESTATIONS

- Depends on degree of the pneumo.
- Ipsilateral chest pain, dyspnea, tachypnea, ↑ HR
- Percussion → resonant / hyper-resonant.
- Auscultation → absent or ↓ breath sounds over the area.
- Tension pneumo → mediastinal shift to the opposite side.
- Hypoxia.
PNEUMOTHORAX

DIAGNOSIS

- Clinical exam, symptoms.
- CXR, CT Scan.
- ABG’s.
ASTHMA

- National Heart Lung & Blood Institute: “asthma is a **chronic inflammatory disorder** of the airways in which many cells and cellular elements play a role, in particular mast cells and eosinophils.”

- Recurrent episodes of airway obstruction characterized by wheezing, breathlessness, chest tightness, and a cough that is worse at night or in the early morning.
ASTHMA

EARLY / ACUTE-PHASE RESPONSE

- The bronchoconstriction.
- Develops within 10-20 minutes after the trigger, release of chemical mediators from IgE-coated mast cells.
- Increased vascular permeability, increased secretions → mucosal edema.
ASTHMA

LATE-PHASE RESPONSE

- The inflammation.
- 4-8 hours after the trigger.
- Inflammation $\rightarrow$ increased airway responsiveness $\rightarrow$ prolongs the attack $\rightarrow$ exacerbations.
- See text for details.
ASTHMA – THE CAUSES

- Complex interaction between genetic (atopic) factors and environmental factors.
- THE ATOPIC TRIGGERS: allergens from: dust mites, cockroaches, animal dander, pollens and molds. Also have other atopic/allergic problems: hay fever, hives, eczema.
- OTHER TRIGGERS: exercise; cold, infection, esp. viruses; inhaled irritants, tobacco smoke, strong odors, ozone (smog-related asthma); occupational fumes; NSAID’s and aspirin.
ASTHMA – THE CAUSES

- OTHER TRIGGERS: sulfites, non-selective beta blockers, emotional factors, hormones, GERD.
ASTHMA – SIGNS & Sx’S

- Mild to severe to **fatal**.
- Wheezing, tightness, dyspnea, fatigue cough.
- Worse at night.
- Prolonged expiratory phase (FEV1, PEV).
- Trapped air → hyperinflation.
- Cough is less effective → can’t clear secretions.
- ↓ effectiveness of ventilation → hypoxia, hypercapnia (ventilation-perfusion mismatch).
- Respiratory failure, **death**.
ASTHMA – THE DX

- Physical findings, history, PFT’s (obstructive defect).
- For early detection of exacerbationsm flare ups → peak flow meter.
- Remember “cardiac asthma” in new-onset asthma in an older patient w/ cardiac disease - they are developing CHF, not asthma.
ASTHMA

- DID I MENTION THAT ASTHMA IS AND CAN BE FATAL?

- HOW YOU SHOULD APPROACH THE ASTHMATIC →
“That’s all the alternative medicine your HMO will pay for.”
COPD

- A group of disorders characterized by chronic and recurrent obstruction of airflow
- Progressive, accompanied by inflammation.
- Most cases related to smoking.
- 4th leading cause of death in the U.S.
COPD

1) EMPHYSEMA

2) CHRONIC BRONCHITIS
COPD - EMPHYSEMA

- Loss of lung elasticity, abnormal enlargement of the air spaces distal to the terminal bronchioles, and destruction of alveolar walls and capillary beds.

- Breakdown of elastin and other alveolar wall components by enzymes released by an inflammatory reaction triggered by smoking.
COPD - EMPHYSEMA

- Destruction of alveoli $\rightarrow$ ↓ surface area available for gas exchange, loss of elasticity, airway collapse.
- Impaired expiratory flow, air trapping $\rightarrow$ proportionate loss of ventilation-perfusion.
- The “pink puffer.”
COPD - EMPHYSEMA

α-1 ANTI-TRYPSIN DEFICIENCY

- α-1 ANTI-TRYPSIN- an anti-protease, protects the lung from proteolytic enzyme destruction by trypsin, a protease whose job is to take out old and infirmed alveoli.
- Deficiency leads to alveolar destruction by trypsin, which is not inhibited.
- Genetic- 1% of cases of emphysema.
COPD – CHRONIC BRONCHITIS

- Airway obstruction caused by inflammation of the major and small airways.
- Edema and hyperplasia of submucosal glands $\rightarrow$ excess mucous production.
- Difficulty clearing mucous.
- Mismatch of ventilation-perfusion, the “blue bloaters.”
COPD - MANIFESTATIONS

- Most patients w/ COPD have elements of both emphysema and chronic bronchitis.
- Progressive change in pulmonary function → cough, sputum production, respiratory impairment → SOB, dyspnea, increased work to breath.
- Late → hypoxia, pulmonary hypertension, right-sided heart failure (cor pulmonale).
COPD - MANIFESTATIONS

- **EMPHYSEMA**: Pursed lips to increase resistance and prevent airway collapse (The Pink Puffers). Use of accessory muscles. Cyanosis develops late. Air trapping → Barrel chest.

- **CHRONIC BRONCHITIS**: SOB. Prolonged exp. phase, exp wheezes and crackles; cyanosis develops earlier → polycythemia. (The Blue Bloaters)
THE PNEUMOCONIOSES

- Various forms of occupational lung disease.
- Inhalation of inorganic dusts and particulates.
- See text for details.
I'd like to report an identity theft...

OK...what's your name?

Maybe you didn't hear me...