Obstructive Pulmonary Diseases (OPDs)

Asthma - Acute OPD

Chronic Obstructive Pulmonary Diseases (COPDs)

Ex. Emphysema

Results from breakdown of elastin fibers in the alveolar walls.

Responsible for elasticity of the lung tissue (ability of lung tissue to return to its normal shape + volume after being stretched during inflation).

Loss of the elastin fibers diminishes ability of the lungs to move air back out during exhalation.
Progressive trapping of air in the lungs leads to hyperinflation of the lungs → Barrel chest

Also, people with emphysema have:

- Dyspnea
- Increasing hypoxemia as disease progresses
- Increasing hypercapnia as disease progresses.

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Respiratory failure and death

Treatments:

- Cessation of smoking
- Bronchodilators
- Breathing exercises
- O2 therapy
- Lung transplant
Restrictive Lung Diseases (RLD)
aka. Interstitial Pulmonary Diseases (IPDs)

In IPDs the lung tissue has a decreased compliance (doesn't stretch as easily as normal lung tissue).

Increases difficulty in bringing air into the lungs during inhalation.

Primary cause of IPDs is buildup of scar tissue in the lungs due to exposure to environmental agents:
- Cigarette smoke
- Asbestos
- Coal dust
- etc.

Ex. Black lung disease, Asbestosis
Scarf tissue doesn't stretch as easily as normal lung tissue

Resists lung inflation

To compensate, people with IPDs breathe faster and shallower

Prevents hypercapnia, but doesn't help to decrease hypoxemia

Initially people with IPDs have decreased capacity for physical activity due to decreased O2 levels in their blood.

As the disease progresses, eventually hypercapnia develops, leading to resp. acidosis and respiratory failure.
Treatment:
- Remove the cause of the disease
- Use of corticosteroids to reduce inflammation as much as possible
- O₂ therapy to reduce hypoxemia
- Lung transplant

Lung Cancers

Leading cause of cancer death in U.S.

Originates from epithelial cells that line the respiratory airways.

Caused by chronic irritation of the respiratory epithelium, or exposure of the respiratory epithelium to airborne carcinogens, most commonly resulting from smoking.
The high incidence of death from lung cancer is due to the fact that they are relatively fast progressing and tend to metastasize to other organs.

- Lungs are highly vascularized
- Lungs have lots of lymph vessels & lymph nodes
- Provide routes for metastatic cancer cells to spread to other parts of the body.

Four major histologic of lung cancer:
- Squamous cell carcinoma
- Adenocarcinoma
- Large cell carcinoma
small cell carcinomas

Squamous cell lung carcinomas

Accounts for ~30% of lung cancers

Presence of squamous cells in the resp. epithelium is result of metaplasia.

In squamous cell carcinoma, the squamous cells begin dividing uncontrollably.

Most often starts in the primary and secondary bronchi (hilar bronchi) and spreads into the deeper airways.

Because of this, the symptoms of lung cancer (blood in sputum, cough, dyspnea) tend to show up early in the progression of the disease. So, this type of lung cancer is generally caught
in its early stages. Also, this type of lung cancer is slow growing and doesn't metastasize until later in the progression of the disease. All of which are good with regard to the prognosis.

Adenocarcinoma

Accounts for ~35-40% of lung cancers. Most common among women and non-smokers. Derived from the gland cells of the resp. epithelium - originate most often in the peripheral airways.
- Are slow growing
- Tend to metastasize early in development of the disease.

Prognosis is bad, because they don't show symptoms until later and metastasize early in the progression of the disease.

**Large Cell Carcinoma**

Account for 10-15% of lung cancers.

The cancerous cells are relatively large in size and are undifferentiated.
- Develop in either peripheral or hilar airways.
- Grow relatively rapidly.
- metastasize early in progression of the disease

Small cell carcinoma (oat cell carcinoma). Look like an oat flake.

Originates from neuroendocrine cells scattered sparsely in the resp. epithelium.

The cancer cells in this type of lung cancer can secrete neurohormones (ADH and ACTH).

Typically first symptoms result from ectopic secretion of these neurohormones.
- Tumors in small cell carcinoma are fast growing and invasive.
- Metastasis occurs early in progression of the disease.

Treatment:
- Surgical removal of the tumor.
- Radiation and/or chemotherapy.

Squamous cell + adenocarcinomas have a 5yr survival rate of ~25% depending on how early the cancer is diagnosed.

Large cell carcinoma has a 5yr survival rate of 2-3%.

Small cell carcinoma has poorest prognosis with death generally occurring...
within 2 yrs of diagnosis.

Renal + Urologic Systems
Figure 28-1 Organs of the urinary system. (From Thibodeau GA, Patton KT: Anatomy & physiology, ed 5, St Louis, 2003, Mosby.)
Figure 28-2 Kidney structure. (From Thibodeau GA, Patton KT: Anatomy & physiology, ed 5, St Louis, 2003, Mosby.)
Nephrons and collecting ducts are the functional unit of the kidney; filter the blood and process the filtrate into urine.

**Figure 28-3 Components of nephron.** (From Thibodeau GA, Patton KT: Anatomy & physiology, ed 5, St Louis, 2003, Mosby.)
Filtration is non-specific, allowing all components of the blood except blood cells and protein to move into Bowman's capsule.
Glomerular filtration rate: rate of flow of solutes + fluid across the glomerular membrane = 180L/day total BV = 5L
To prevent loss of important solutes & water, most of the volume of the filtrate is reabsorbed as the filtrate moves along the nephron & down the collecting duct.

Dysfunctions of the Renal & Urologic Systems:
- Urinary Tract Obstructions (UTOs)
- Glomerular Disorders
- Renal Failure (Kidney Failure)

**UTOs**

Involve blockage of urine flow along the urinary tract, can occur anywhere along the urinary tract.
Can result from:
- Tumor along the urinary tract
- Enlargement of the prostate gland in men
- Bladder dysfunction
- Most common cause is formation of a renal caliculi (kidney stone) along the urinary tract

Most kidney stones form in the calyces or renal pelvis.

4 types of kidney stones:
- Calcium oxalate or calcium phosphate stones.
  Account for 80% of kidney stones
- Struvite stones - consist of magnesium ammonium phosphate
Account for 15% of kidney stones

- Uric acid kidney stones composed of uric acid crystals

Account for 7% of kidney stones

- Cysteine stones — composed of cysteine crystals

Theories of kidney stone formation

- Saturation theory — Stones form due to the super saturation of the urine with the stone components.

- Inhibitor theory — Stones form because of a deficiency of nephrocalcin in the urine
that normally inhibits Stone Formation

- Matrix theory - mucopolysaccharides secreted by the epithelial lining of the urinary tract are acting as centers for stone formation.

Most kidney stones are less than 5 mm in diameter and can pass through the urinary tract and be eliminated without intervention.

In cases of larger stones surgical removal may be necessary.

Or extracorporeal shockwave lithotripsy can be used to break the stone into smaller pieces.
Most commonly caused by immunologic mechanisms.

Antibodies are produced by the glomerular basement membranes.

Disorders involving the glomeruli and filtration.

Glomerular Disorders

Fluid intake is increased to decrease urinary concentration.

Dietary changes are made to reduce intake of those components.

Compounds are analyzed for what is needed in long-term treatment.

It is directed at the cause of the problem.
Antigen–Antibody complexes become trapped in the glomerular membrane triggering an inflammation that damages the glomerular membrane (glomerulonephritis).

The damage to the glomerular membrane results in Nephrotic syndrome:

- Damage to glomerular membrane
  - Blood cells + proteins cross the membrane and pass into the filtrate
  - Hematuria + proteinuria result
  - ↓ level of protein in the blood (hypoproteinemia)
↓ results in ↓ colloid osmotic pressure in the capillaries ↓

↓ Tissue edema ↓

↓ ↓ fluid volume of the blood ↓

↓ ↓ GFR ↓

↓ ↓ urine production (oliguria) ↓

Wastes accumulate in the blood rather than being filtered in the kidneys.

Treatment:
- Corticosteroids to minimize inflammation in the glomerular membrane
- Diuretics to reduce tissue edema
If there is an underlying infection, antibiotics are used.

Chronic glomerulonephritis is the leading cause of kidney failure. It accounts for 1/2 of all people on kidney dialysis.

In chronic glomerulonephritis, there is a progressive loss of glomeruli and a progressive decrease in the GFR.

Signs of kidney failure don't normally become apparent until renal function drops to about 25% of normal GFR.
So, as a consequence, the disease is not normally detected until the kidneys are severely damaged.

Treatment is directed at maintaining the remaining renal function through management of diet and fluid intake.

- Management of protein intake to reduce nitrogenous wastes. Reduces workload on the kidneys.

- Fluid intake has to be carefully regulated to avoid tissue edema.

- Na+ and K+ intake has to be carefully regulated.
Excess K+ intake can result in hyperkalemia (elevated K+ levels in the body fluids), which can disrupt membrane potentials required for normal functioning of the nervous system and heart muscle.

- Dialysis can be done to clear the blood of toxic wastes.
- Kidney transplant.