Test Questions…in theory…

Destruction of alveoli is a hallmark of
- Asthma
- **Emphysema**
- Chronic bronchitis
- Pleuresy

COPD can be which:
- Asthma and emphysema
- Asthma and chronic bronchitis
- Asthma and pneumoconiosis
**Emphysema and chronic bronchitis.**

*Note: Obstructive lung diseases make it hard to get the air out and will eventually turn into emphysema. Could be obstructed by gunk or obstructed functionally (no elasticity, for instance which causes an obstruction and air doesn’t go out right.)*

*Upon inhale, we create negative pressure by expanding the ribcage. Exhaling is just relaxation, allowing air to flow out. If you loose elasticity, you must force the air out, creating high pressure in alveoli sacs, damaging them.*

Which of the following is not a pneumoconiosis
- Asbestosis
- Silicosis
- Bariliosis
- **Aspergilliosis**

Name 4 risk factors for atherosclerosis (there are 6-7 factors generally accepted)

Name 2 causes of 2ndary hypertension
- Renal disease (renal artery stenosis, glomerulonephritis), tumors excreting hormones which cause bp to go up – adrenal overload (renin, aldosterone, angiotensin II). Pheochromocytoma, preeclampsia, hyperthyroidism, CHF and anything that leads to it like aortic stenosis, etc.

Know the risk factors for Colon Cancer
- Age: over 50
- Genetic predisposition
- History of other disorders such as: inflammatory bowel disease, familial polyposis, polyps, etc.
- Diet: high in sugars and fats, low in fiber, deficient in Vitamin A/C/E
- Smoker
- Overweight
- Physical
- Heavy alcohol use
- Type II diabetes
  (Symptoms: blood in the stool, change in bowel habits – diarrhea, constipation, decreased stool caliber)
What are the causes of anicteric jaundice?
   Colestatic jaundice or neonatal jaundice. Icteric jaundice or icterus is hepatitis, most commonly alcoholic hepatitis, but also seen in viral hep.
Viral Hepatitis
This encompasses Hep A, B, and C. There is also hepatitis caused by toxins. The types of hepatitis differ in modes of transmission, incubation periods, degree of damage, whether it goes chronic and/or carrier state.

**Hep A**
- fecal/oral, not blood borne. Likes to hang out in GI tract, escaping thru fecal matter. People downstream eat lettuce or food prep’d by someone who didn’t disinfect properly, etc.
- Incubation = 2-6 weeks. Abrupt onset – sx include: fever, malaise, nausea/vomiting, anorexia, ab pain, jaundice, dark urine.

**Hep B**
- blood borne and other body fluids – semen and to a lesser degree saliva. Transmitted sexually, via needles, etc. Less transmitted via casual contact.
- Incubation: longer than hep A, usually 2 months or so. SX = RUQ pain with tender feeling liver, can develop chronic with cirrhosis. Sometimes no sx at all, just go directly to carrier stage.
- Vaccine is available and recommended for kids, and at risk pops such as HCW’s.
- Most with Hep b are chronic, no sx. Disease is usually found during eval for elevated liver enzymes.

**Hep C**
- blood borne and other body fluids. Most common cause of chronic hep, cirrhosis, hepatocellular carcinoma worldwide.
- There are 6 genotypes of the damn thing, making it hard to get our immune system to recognize them all.
- More often transmitted via needles. Incubation is similar to B, average 50 days or so. Jaundice is uncommon, so fulminant hep is rare. Chronic hep and cirrhosis, carcinoma.
- There’s no vaccine for C.

Damage to the liver cells come in 2 flavors:
- Direct cellular injury
- Immune response against the virus – the higher degree the immune rsyp, the higher the cellular damage. Then again, the higher eradication of the virus, the less likely you’re going to go chronic/carrier.

The infection:
- Prodromal phase
  - Feels like a very bad flu
- Icteric phase
  - Really the completion of the prodromal phase. “Icterus” means yellow sclera due to increased bilirubin in eyes, skin. Isn’t quite jaundice at this point.
Convalescent phase

Alcohol-induced Liver Disease
These aren’t really different types of disease, but stages in progression of the illness.

1. Fatty Liver Disease
   Stage 1 – as the cells are damaged, they are replaced by fat cells. Early stage, assoc’d with
   swelling of the liver. Liver is working fairly well, but elevated liver enzymes, brief episode of
   jaundice, frequent nausea

   Metabolic syndrome can also cause fatty liver disease.
   - Obesity
   - Hyperlipidemia
   - Diabetes/insulin resistance – high insulin levels in the bloodstream, very inefficient
     at getting glucose/insulin into the cells.

2. Alcoholic hepatitis
   This is the active liver disease, often wake throwing up. Will still have lots of fatty liver tissue
   and fatty liver disease, but also has an inflamed liver, will be jaundiced more days than not. Liver
   is soft here in the pre-cirrhosis stage.
   The disease is reversible up to the cirrhosis stage. Livers work in independent “cells” so that if
   part is affected the rest carries on.

3. Cirrhosis
   This is the scarring of the liver, liver is shrinking at least in segments. Doesn’t occur everywhere
   at once, but in spots. Liver gets very hard. Obstx of the blood flow in the portal vein (portal
   hypertension)—veins get engorged with blood and will ooze blood. Blockage of the portal vein
   causes backflow and retention of fluid in the abdomen. Ascites. Veins engorged with blood will
   cause esophageal varices (hemorrhoid in the esophagus).
   a. Portal hypertension
   b. Biliary obstruction, exposing liver cells to destructive effects of static ble.
   c. Loss of liver cells.

Pg 937-38.
Manifestations:
   - Ascites, edema, jaundice
   - Hepatosplenomegaly-thrombocytopenia
   - Portal hypertension, esophageal varices, caput medusaa
   - Impaired production of clotting factors – albumin.
**Kidney Stones**

Kidney stones are also called Nephrolithiasis. Glomerulus → tubule (further filtering, mostly KI is deciding how much water/sodium/potassium to retain and resorb → nephrons.

Kidney’s main job is to filter chem impurities out the blood. Spleen filters out the blood for cellular elements that are no longer viable: RBC’s mostly.

Kidney: blood thru the glomerulus, a mechanical and chemical type of filter. The most common problem is the obstruction of the upper tract. As blood filters, get a very concentrated preurinary product in the renal pelvis where it stays until it travels down the ureter to be excreted. If the renal pelvis is partially obstx’d or is just slow flowing, stones can form. Usually do so due to anatomical irregularities.

Calcium oxylate is the most common kind of stone, not due to too much ingestion of calcium though as the body will probably absorb or poop it out. That said, overingestion of antacids like Tums will push someone into a high alkylosis which will dump calcium into the urine and could be candidate for kidney stones.

Urate stones is another type – purine buildup. Similar to gout, but in the kidney. Can decrease risk of stones by changing diet, unlike people with calcium oxylate stones. See pg 815. If you alkalinize the urine there is less risk of the acidic stones. These stones tend to be crystaline in nature and are like little pointy grassburs. Youch.

People with frequent episodes of pyelonephritis = kidney stones. Also, if you have stones, higher risk of infection.

Manifestations:

- Colicky pain – comes/goes.
  - Due to dilation of the ureter or collecting system due to obstruction.
  - Assoc’d with stones that are 1-5mm in diameter that pass thru.
  - Pain is acute, intermittent, severe in the flank and upper-outer quad ab pain. Radiates to ab, blader, perineum, scrotum
  - Cold, clammy skin with n/v.
- Non-colicky pain – usually a precursor to the colicky pain
  - Assoc with distention of renal pelvis, calyces
  - Dull, deep ache in flank (hypochondriac pain) or back.
- Hematuria – can be gross or microscopic. Kinda common that pt gets treated for kidney infection and gets antibiotics. Can test with a dipstick – just a trace of leukocytes with more blood then probably isn’t infection. Infection would show as higher WBCs than blood.

DX: can’t see on xray very well. IVP and ultrasound are better.
Kidney Infections

UTI’s
Cystitis is a lower tract infection. Painful, but not life threatening. Often caused by bacteria already living in your system.

Remember the longer the urine is retained increases the risk. Dehydration is another big old risk – again, low flow. Antibiotics is another cause – kills off the normal flora, makes growth of abnormal bacteria more likely.

Antibiotics widely prescribed for UTI because stops the progression of the bacterial spread to the kidney. Stewart says douche with plain water and acidophilus…TK says hell no. Hmmm.

Pyelonephritis: upper tract infections (kidney). This does not = UTI! Becomes septic quickly – goes from kidney to blood rapidly.

Antibiotic time:
  Fever – 102+
  Flank pain
  WBCs in urine

See Ch 35 – Disorders of Renal Function.pdf

Most common organisms in UTI:
  • gram negative rods – which populate the bowel.
  • Gram positive rods – usually more in the upper body.
  • E-coli is the most common.

Women are more likely to get UTI’s due to the short ureter length. Also, dude peepee’s are easier to keep clean. Note that the urethra is normally colonized by bacteria—supposed to be there! Moral: wash after sex, but only with water, not soap or antibacterials!