Describe the normal functions of the liver and potential patient problems that can develop with liver abnormalities:
The normal functions of the liver are:
- Digestive Role: produces bile; processes and stores fats, CHO, proteins; processes and stores vitamins and minerals; synthesizes cholesterol; produces triglycerides
- Metabolism Role: produces glucose; glycogen storage; helps maintain normal blood glucose levels through glycolysis, lipogenesis, glycogenolysis and gluconeogenesis; synthesizes albumin; forms urea from ammonia; metabolizes hormones; forms lipoproteins
- Excretory Role: excretes bile; excretes cholesterol; converts ammonia to urea so it can be excreted
- Detoxification Role: detoxifies ammonia, alcohol, hormones, drugs
- Hematologic Role: synthesizes clotting factors, stores blood

Problems that can develop with liver abnormalities:
- portal hypertension
- ascites
- hepatic encephalopathy
- vitamin deficiency (mainly fat soluble ones)
- bleeding/hematologic disorders
- endocrine disorders
- skin disorders
- hepatorenal syndrome (a type of liver failure that also has renal failure as a bonus)
- esophageal varices, abdominal varices (caput medus)
- GI bleeds
- splenomegaly
- poor drug metabolism

Differentiate between the causative factors and preventable measures for Hep A through E
Hep A: cause = poop; prevention = wear gloves when handling feces; wash all fruits/veggies carefully using bottled water; don’t drink the water; get a vaccine
Hep B: cause = blood, body fluids; prevention = wear condoms, wear gloves (universal precautions), don’t get stuck by a needle, be cautious regarding tattooing and piercings, do not share personal items, don’t use IV drugs, get a vaccine
Hep C: same as Hep B except there is no vaccine for Hep C.
Hep D: cause = blood, body fluids; prevention = early recognition and treatment of Hep B can help prevent Hep D; same prevention as Hep B; get the Hep B vaccine
Hep E: cause = poop (Asia, Africa, India, Mexico); prevention = good sanitation, don’t drink the water, can boil or chlorinate the water.

Describe the clinical manifestations of impaired liver function
- lethargy
- anemia d/t GI bleed or B-12 problem
- asterixis d/t CNS alteration r/t high ammonia levels (liver is not converted ammonia to urea like it should)
- jaundice d/t high levels of bilirubin
- hypoglycemia d/t impaired gluconeogenesis, glycogenolysis and glycogenesis; impaired metabolism of protein also
- palmar erythema, spider angiomas testicular atrophy d/t androgen & estrogen detoxification problems
- fever d/t inflammatory response (just a guess)
- dyspnea d/t ascites and fluid shifts secondary to hypoalbumenemia
- dyspepsia (bloating, N/V, anorexia, abd pain) d/t ascites secondary to portal hypertension
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- weight loss or gain d/t esophageal varices making eating difficult or ascites leading to a feeling of fullness; weight gain due to fluid overload r/t ascites and edema secondary to decreased plasma proteins
- ascites d/t portal hypertension
- splenomegaly r/t portal hypertension
- edema r/t portal hypertension, ADH & aldosterone problems, altered metabolism of fats and proteins
- esophageal varices d/t portal hypertension
- superficial abd varices d/t portal hypertension
- hemorrhage d/t GI Bleeds and esophageal varices caused by portal hypertension
- confusion r/t elevated ammonia (hepatatic encephalopathy)
- respiratory acidosis (I’m not sure what causes this)
- foul breath (not sure why this happens...improper breakdown of things, I guess).

Discuss the significance of various laboratory data used to evaluate liver function

- ALT is elevated with parenchymal inflammation. This is specific for the liver.
- GGT is what you’d look at to see if there is an alcohol-related problem. Will be elevated
- AST is elevated with inflammation; not specific for liver
- ALP is elevated with obstruction; not specific for liver
- Ammonia will be elevated when the liver is not converted it to urea. Can lead to asterixis and hepatic encephalopathy
- LDH (lactic dehydrogenase) will be elevated in liver disease. Not specific for liver
- Cholesterol could be up or down. If down, then disease is severe.
- Albumin will be low d/t decreased synthesis.
- BUN will be low with severe liver disease. It will be high (along with Cr) for hepatorenal syndrome
- PT will be elevated with liver dysfunction d/t decreased clotting factors
- PTT will be elevated with severe liver disease d/t decreased clotting factors
- PLT will be low or WNL but poor quality d/t problems with the spleen and portal hypertension
- Bilirubin will be elevated in liver disease
- Na and K could be up or down
- Glucose will be increased with an obstruction; decreased with disease
- H&H will be decreased d/t severe anemia and hemorrhage

Create a nursing care plan to promote adaptation for adults with altered liver function that specifies dietary restrictions, comfort measures, and patient safety concerns.

- Dietary restrictions
  - low protein if hepatic encephalopathy
  - low sodium to reduce ascites
  - replace vitamins
- Medication restrictions
  - Hepatitis: no chlorpromazine (an antipsychotic and antiemetic), aspirin, acetaminophen, phenothiazines (a whole group of antipsychotic/antiemetics) and most sedatives
- Comfort measures
  - Reduce fatigue: pace nursing activities, give rest periods
  - Give antiemetics
  - Give antihistamines to treat pruiritis
- Patient safety
  - Seizure precautions if hepatic encephalopathy
  - Monitor for confusion
  - Monitor for bleeding, teach pt to avoid things that cause bleeding (straining, flossing, vigorous nose blowing, etc...)
Incorporate the special considerations for the elderly patient regarding liver function in creation of a nursing care plan.

Synthesize information on complications of portal hypertension to provide patient teaching on treatment plan for GI bleed that incorporates emergent procedures, diagnostic tests, and rational for TIPS intervention.

Portal hypertension causes:
- ascites
- splenomegaly
- edema
- esophageal varices
- superficial abdominal varices
- hemorrhage

Portal hypertension occurs when there is increased resistance to flow in the portal venous system. This increased resistance leads to abnormally high blood pressure in branches of the portal vein. The portal vein receives blood from the entire intestine and from the spleen, pancreas and gallbladder. As blood backs up into these systems problems occur such as those listed above.

Management of Portal Hypertension (I found this algorithm on a website)
1. Perform endoscopic variceal ligation or endoscopic sclerotherapy
2. If hemorrhage is not controlled, consider a TIPS procedure
   1. a shunt is placed between the portal vein and the hepatic vein
   2. it is a non-surgical way of placing a shunt...it is passed down through the jugular vein by a radiologist
   3. once in place it is opened up to reduce resistance to flow
3. If hemorrhage is not controlled, or TIPS is not available go with balloon tamponade (after this point you are going to start looking at their candidacy for transplantation)
4. If the problem persists, you’re looking at a portocaval shunt: The big problem with this procedure is that now a LOT of blood is not getting filtered/processed through the liver. In other words, 75% of the blood coming through the liver comes through the portal vein and it is now redirected.

Synthesize information on liver abnormalities to provide patient/family teaching on rationale for medications used in the treatment of liver failure.
- Lactulose will bind up ammonia so it can be pooped out.
- Neomycin will kill bacteria that produce ammonia as a by-product
- Diuretics will be used to help with ascites. Note that the pt with hepatorenal syndrome will not respond to diuretic therapy for ascites.
- Albumin increases oncotic pressure to reduce ascites
- Octeotide is used to decrease flow to blood vessels in the GI system (helps to control GI bleed)
- Folic acid, thiamine, vitamins, minerals are replaced
- Antihistamines for pruritis
- Beta blockers to reduce portal hypertension (not mentioned in lecture)