

Liver Disease

Let's Review the Liver!

Anatomy

- Largest internal organ in the body
- Divided into right and left lobes (right is largest and is further divided into caudate and quadrate lobes)
- Within the lobes are smaller units called lobules which consist of hepatocytes (liver cells)
- The hepatocytes are the functional cells of the liver, the remaining mass is made up of Kupffer cells, stellate cells, endothelial cells, blood vessels, bile duct cells and supporting structures.

Hepatic Blood Supply

- Obtains dual blood supply from the portal vein and hepatic artery...this is very unique! A vein is located between two capillary beds. The hepatic vein collects blood from capillaries in visceral structures located in the abdomen and empties it into the liver.
- The liver gets approximately 1500 ml of blood/minute. This comes out to 13% of total body blood supply. The hepatic artery supplies 25% (around 300 ml/min), and the portal vein supplies 75% (around 1050 ml./min). Because the portal vein supplies most of the blood supply, you can imagine why it's so devastating when that vein gets backed up...lots and lots of blood with no where to go (except back into the portal system).
- The hepatic vein empties into the inferior vena cava
- The liver stores 450 ml of blood due to pressure differences between the hepatic vein and the vena cava.
- In CHF, blood backs up and accumulates in the liver. That can't be good!
- Hepatic portal vein takes all the flow from other vessels....it there are problems it's going to back up to all these other organs. (see slide, it's the blue veins)
- Our biggest concern is with cirrhosis or fatty liver, and we can't get blood flow to go back out through the hepatic vein and on through the system.

Digestive Role

- Produces bile (main fxn). Bile emulsifies fats and stimulates peristalsis (more on bile below)
- Conveys bile from the gallbladder (where bile is stored) until it enters the duodenum at the Sphincter of Oddi through the common bile duct
- Processes and stores fats, carbohydrates and proteins
- Processes and stores vitamins and minerals (fat-soluble vitamins A,D,E,K, B12, copper and iron...iron is in the form of ferritin). Vit A is necessary for retinal fxn and epithelial cellular growth; Vit B-12 is necessary for DNA synthesis, normal RBCs and nerve fxn (Pernicious anemia is d/t a lack of Vit B-12); Vit K is necessary for clotting.
- Synthesizes cholesterol
- Produces triglycerides

What is bile?

Bile is an alkaline fluid composed of cholesterol, phospholipids, bile salts, bile pigments (bilirubin), proteins and electrolytes. The liver produces 600-1200 ml of bile a day 97% of which is water). It flows through the canaliculi, into the common bile duct, goes through the Sphincter of Oddi and empties into the duodenum. Bile is stored in the gallbladder, where it is concentrated, and discharged into the duodenum when fatty chyme enters from the stomach. The function of bile is to emulsify fats, facilitating their digestion in the small intestine by pancreatic lipase. It is also necessary for transport of cholesterol via fatty acids and the transport of fat soluble vitamins (ADEK). Bile also stimulates peristalsis. 94% of the bile salts that go into the intestines is reabsorbed into the portal circulation for recycling.

Terms To Know

Glycogenesis = formation of glycogen from glucose
 Lipogenesis = formation of fat from CHO
 Glycogenolysis = breaking down of glycogen
 Gluconeogenesis = AA + glycerol + lactic acid = glucose

Metabolism Role (carbohydrates, fats and proteins)

- Carbohydrates
 - production of glucose from proteins and fats

- results in glycogen storage
- Helps maintain normal blood glucose levels through glycogenesis, lipogenesis, glycogenolysis and gluconeogenesis.
- Protein (will affect diet teaching for patient, also very important!)
 - breakdowns dietary proteins
 - albumin synthesis (keeps oncotic pressure high, controls fluid/electrolyte balance...leads to ascites)
 - formation of urea from ammonia (you make a lot of waste products, specifically ammonia. It is the liver's job to turn ammonia to urea so you can pee it out. Too much ammonia and the pt will have severe neurological defects and look like they are in a coma. Need ammonia labs!, electrolytes)
 - synthesis of clotting factors (want to look at PT, PTT, PLT, fibrinogen levels, H&H)
- Fats
 - Dietary triglycerides are metabolized by the liver into fatty acids that are used for: oxidation and energy production; metabolized to ketones; synthesis of cholesterol and phospholipids; formation of triglycerides from dietary lipids, cho and proteins; formation of lipoproteins (triglyceride + protein + cholesterol + phospholipid); the fatty acids released from adipose tissues are used by the liver for the same purposes
- Metabolizes hormones such as mineralocorticoids, glucocorticoids and sex hormones (see detoxification)

Excretory Role

- Excretes bile
- Excretes cholesterol
- Converts ammonia to urea
- Detoxifies drugs, hormones and other foreign substances (more on this below)

Detoxification

The liver detoxifies **ammonia**, which is produced in the gut by bacteria after de-amination of ingested high protein foods or GI bleeds. It is produced in the liver from de-amination of amino acids (NH₃). Ammonia is HIGHLY TOXIC to body tissues, especially neurons! It is converted by the liver into urea that is excreted through the kidneys. With liver disease, urea synthesis is often depressed. This leads to increased ammonia levels and decreased BUN.

80-90% of alcohol is detoxed by the liver...so be kind to your liver and don't drink so much!

Hormones are also detoxified by the liver. (is detoxified a word?) This involves de-amination of insulin and glucagon; deiodination of thyroxine and triiodothyronine; glucocorticoids are transformed to water soluble form; ADH and aldosterone detoxification; estrogen and androgen detoxification.

Drugs are also a biggie. Dr. Brady listed amphetamines, many sedatives, and barbituates except for phenobarbital and barbitol (not sure if she meant these are detoxed by liver, or these drugs damage liver?)

Hematologic Role

- Stores blood! In case of rt-side heart failure or valvular problems, excess blood can be stored in the liver. In the case of hemorrhage, the liver can release blood (approx 450 ml) to maintain circulatory volume)
- Synthesizes all but two clotting factors and synthesizes bilirubin (discussed elsewhere in more detail)

What is bilirubin anyway?

When RBCs break down they are pulled out of circulation by macrophages at the cellular level and by the spleen, liver and bone marrow at the organ level. The amino acids and iron are recycled and put back into the bone marrow for construction of new RBCs. The heme group of the old falling-apart RBC does not go to the bone marrow for recycling. Instead, it is converted to bilirubin and transported to the liver where it is incorporated into bile. Some of this goes to the digestive system to be excreted in feces, and some goes in the blood stream so it can travel to the kidneys and be excreted in the urine.

What do bilirubin levels mean?

- Total bilirubin: high levels warrant more testing
- Unconjugated /indirect: high levels indicate large hemolysis or problems with intrahepatic cells
- Conjugated/direct: high levels indicate problems with the bile flow, suggestive for biliary tract obstruction

Geriatric Liver Considerations (as patient ages, things become even more important b/c...)

- ↓ in # and size of hepatic cells
- ↑ in fibrous tissue = less protein synthesis (part of why they dont heal as quickly)
- ↓ # of enzymes to metabolize drugs (drug dosages have to be adjusted)
- Lab Data remains normal many times.

Clinical Manifestations of Altered Liver Function

The liver has multiple functions, so when it's not working well the dysfunction will be manifested in multiple body systems.

Common S/S: lethargy
 anemia (because of B-12 problem)
 asterixis (a shaking of nerve from high ammonia levels)
 jaundice (bile salts not being processed correctly)
 fever (inflammatory process)
 dyspnea
 dyspepsia (comes from lack of good blood flow, so blood backs up, leading to...
 bloating, N/V, anorexia, abd pain, weight loss or gain)

What is Jaundice? (also called Icterus)

Jaundice is yellow pigmentation of the sclerae, skin & tissues due to excessive bile pigments in blood. It occurs when serum bilirubin levels are > 2.0 - 2.5 mg/dl (normal levels are 0.1 - 1.2 mg/dl). It's major causes are:

- Excessive destruction of RBCs (will see ↑ unconjugated bili)
- Impaired uptake of bilirubin by the liver (intra-hepatic problem, will see ↑ unconjugated bili)
- Decreased conjugation of bilirubin (intra-hepatic problem, will see ↑ unconjugated bili)
- Obstruction of bile flow in the hepatic lobules, or the intrahepatic or extrahepatic bile ducts (will see ↑ conjugated bilirubin levels)

When the problem is the liver not being able to take up bilirubin correctly, you will have dark urine. If jaundiced and urine also dark, then not processing bilirubin in liver...bile causes problems with skin changes and it is dumped out into system and into the urine. (normally it goes to stool) U/A is another lab you will need! **Important!**

Type of Jaundice	Characteristics
Pre-hepatic jaundice	the jaundice is MILD, stools are NORMAL, elevated unconjugated bilirubin and there is NO bilirubin in the urine.
Intrahepatic or hepatocellular jaundice	both conjugated and unconjugated levels are elevated and the urine is DARK.
post-hepatic or obstructive jaundice (AKA cholestatic jaundice)	the conjugated bilirubin levels are elevated, the stools are clay colored, the urine is dark, serum alkaline phosphatase is elevated, and amino transferase increases, bile salts are elevated leading to pruritis.

Liver Function Radiological tests

- Ultra sound or Ultrasonography (looks at fluid levels) of abdomen
- Computed tomography (CT) of Abd
- Magnetic resonance imaging (MRI) of Abd
- Liver Biopsy
- Paracentesis (abdominal tap...one way to pull off fluid. Don't want to do more than 1.5 to 2 L, don't do it too fast)
Look in ATI. Can also give albumin and lasix to get rid of fluid.
- Endoscope (GI Scope procedure)
- Angiography and portal pressure measurements

Nursing Responsibilities/Patient Teaching with Tests/Interventions

- Ultrasound/CT/MRI: Big issue is that they have to go FLAT into the scanner. Worry about breathing! Make sure you have a portable pulse ox on them, and portable oxygen on while in scanner. MRI takes 45 minutes. Also make sure they can follow directions (may not be able to d/t ammonia issues) to hold breath when they need to. Most scanners cannot handle over 300 pounds...so what do you do? They do ultrasound, x-ray instead.
- Liver Biopsy: Very concerned about bleeding...watch H&H, watch vitals. Stop any aspirin or Plavix 7 days prior, no alcohol for a week prior, for 2 weeks after should not be doing any lifting, no driving for 24 hours after, watch the dressing for signs of bleeding. Position on right side for 1-2 hours on BR to put pressure on the liver.
- Endoscopy: Big issues are NPO prior, consent, conscious sedation...so watch airway during recovery.
- Paracentesis: Only remove up to 1 L at a time. The most determining factor in the use of paracentesis is respiratory distress. When a large amount of protein-rich fluid is removed, fluid will shift between the intravascular space and the interstitial tissue causing hypovolemia...this is the major factor that determines the rate at which the fluid can safely be removed (may be over several hours). Tell pt they may experience pain or pressure at site.
 - Nursing interventions for paracentesis (just the non-obvious ones): take baseline vitals including weight and abdominal girth, have the client void prior to the procedure (or put in a Foley), explain that local anesthetics used at the site, administer sedation if ordered, position client sitting with legs dangling and arms resting on bedside table or supine (depends on tolerance).
 - After the procedure (just the non-obvious ones): maintain pressure at needle-insertion site for several minutes, re-do vitals and compare, monitor temp q 4 for 48 hours, give IV fluids or albumin as ordered, place pt on unaffected side for 1-2 hours after, document what the fluid looked like and how much.
 - If the site continues to leak, apply dry sterile gauze and change as often as necessary.
 - Diuretics may be prescribed to control fluid volume, may need K supplements as a result. Monitor K and -lytes.
 - Lab tests afterward are: albumin, protein, glucose, amylase, BUN, creatinine
 - To prevent hypovolemia: slow drainage, administration of plasma expanders (albumin); monitor for signs (tachyC, hypoT, pallor, diaphoresis, dizziness)
 - Bladder perforation is rare but possible; symptoms are hematuria, low or no urine output, suprapubic pain/distention, symptoms of cystitis, fever; if suspected, call the MD!
 - Peritonitis can occur d./t injury to intestines from needle insertion; symptoms include sharp, constant abdominal pain, fever, nausea, vomiting and diminished or absent bowel sounds; if suspected, call the MD!
- Angiography and portal pressure measurements: The pt is usually under sedation, and to go through angiography they have a groin site so watch for bleeding there.

Lab Test of Liver Function

AST	0-35 units/L	Liver, skeletal & heart muscle. ↑ with INFLAMMATION; not specific for liver
ALT	4-36 units/L	shows parenchymal inflammation; SPECIFIC for liver
GGT (alcohol)	8-38 units/L	liver cells and kidney fxn; ↑ with alcohol-related problems
ALP	20-80	ALKALINE PHOSPHATASE; ↑ with obstruction
LDH	100-190 units/L	heart, kidneys, liver, skeletal, RBCs, brain and lungs
Ammonia	80-110 units/L	byproduct of metabolism that is normally converted to urea by liver. when high you will have a change in LOC, shaking (asterixis); will be ↑
Cholesterol	< 200 mg	↓ or ↑ in severe hepatocellular disease
Phospholipid	150-250 mg/dL	
Albumin	3.5-5 /dL	↓ in liver disease (cirrhosis, alcoholism, hepatitis)
BUN	8-22	↓ with severe liver disease; BUN & Cr ↑ with hepatorenal syndrome
PT	11-12.5 seconds	↑ with liver dysfunction
PTT	68-82 seconds	↑ with severe liver disease / Cirrhosis
PLT	130-400	either ↓ or WNL but poor quality; d/t storage in spleen and distribution with portal hypertension...they get squished and misshapen.
Bili Total	0.3-1 mg/dL	↑ in liver disease
Direct Bili	0.1 -0.3 mg/dL	CONJUGATED; ↑ with obstruction of ducts
Indirect Bili	0.2 - 0.8 mg/dL	UNCONJUGATED; ↑ d/t reduced parenchymal surface and hemolysis
Urine Bili	0 - 0.2 mg/dL	↑ in hepatocellular disorders and biliary track obstruction
Urobilinogen	0.3 - 3.5 mg/dL	↑ in hepatocellular disorders and biliary track obstruction
Fecal urobilinogen	75 - 275 EU/100g	
Na/K		decrease or increase
Glucose	70-110 mg/dL	↑ with pancreatic obstruction; ↓ with severe liver disease
H & H		↓ d/t severe anemia and hemorrhage

Causative Factors of Impaired Liver Function

Drugs

- Alcohol (ETOH) most common in US males
- Toxins (mushrooms,) most common in dveloping countries
- Pharmacological agents (Tylenol, Isoniazide)
- Elderly have increased incidence of toxicity with lower doses

Viral

- Hepatitis A,B,C,D
- Herpes Simplex
- Adenovirus
- Cytamegaolvirus
- Epstein-Bar

Common Disorders of the Liver

Hepatitis (hepatocellular injury)

Hepatitis is the most common **acute problem** of the liver. It is an acute process with stages that cause heptocellular injury. It involves widespread inflammation of liver cells which alters the structure and function of liver cells causing degeneration or necrosis. No bueno!

Cirrhosis

It is important to note that cirrhosis can happen for reasons other than alcoholism...it can also occur due to hepatitis or a viral infection. This is a **chronic**, progressive disease characterized by diffuse destruction and regeneration of parenchymal cells. The ongoing destruction results in scarring and loss of function. Blood flow doesn't go through hepatic vein as it should...you start to see backup into the venous system of GI tract...the veins dont like a lot of pressure so they bleed easily. Voila...a GI bleed!

Viral Hepatitis

This is a world wide disease causing inflammation of the liver. It can be **acute or chronic**. There are several types of viral heptatitis (types: A, B, C, D, E, and G). The most common in the US is blood born hepatitis (Hep B, C, D), and 250,000 people are infected each year with some form of hepatitis. Persons with hepatitis are carriers but are also infectious, so they should never donate blood, organs or body tissues (unless it's a Hep-to-Hep transplant). Immunizations are available for Type A, and Type B ONLY! It is **caused by viruses, toxins or chemicals**. Dr. Brady listed some causes: rubella, varicella, retroviruses, yellow fever, adenovirus, & Epstein Barr Virus. She also said that Interferon is commonly given with viral infections.

Prodromal phase (flu-like)	Icteric phase (5-10 days later)	Signs of obstruction	Convalescent (2-3 week period)	Dx Testing
headache, fatigue, low-grade fever, N/V, arthralgia, myalgia	diarrhea/constipation, RUQ pain, lethargy, irritability, jaundice, severe pruritus	light colored stools, dark urine, jaundice, elevated bilirubin and LFTs, bleeding tendencies, anemia, transient hyperglycemia, palmar erythema, hepatomegaly	gradual regaining the sense of well-being, increased appetite, gradual decrease of jaundice, complete recovery is in 9 weeks for Hep A; 16 weeks for Hep B, C	Elevated liver enzymes: ALT, AST, ALP, total bilirubin, direct indirect

When liver enzymes peak and drop then increase again, this means chronic Hepatitis and poor prognosis.

The Table of Hepatitis

Factor	Transmission	incubation	severity	Prevent/Vac.
Hep. A	Fecal – oral Carrier state	30 days	low	Hygiene Hep A vac
Hep. B	Blood born Body fluids	12-14Wk	May be fatal	Hygiene/Univ perca/ HepBVac
Hep. C	Unprotected sex Drugs & Needles Health workers	6-7 Wk	Chronic hep	Univ Per. HCV interferon
Hep. D	Carrier state	Same as Hep. B	Co-infect with hep.B. risk for carcinoma	Univ Perc Hygiene Hep B vaccine
Hep. E	Fecal – oral Asia Africa, India & Mexico	40 days	Self limiting risk increase in pregnancy	Hygiene sanitation
Hep. F	Rare & difficult to diagnose due to lack of testing methods			
Hep. G	percutaneous	-	No liver disease	Hygiene

Serologic Markers and Antibodies for Hepatitis

- **Serologic markers** identify presence of virus (HAV, HBsAg, Anti-HBc IgM, HCV, HDV, HEV)
 - Serum HBsAg for longer than 6 months => chronic hepatitis or carrier.
- **Hepatitis anti-body** serum testing serum: Anti-HAV, HBsAb, Anti-HCV, Anti-HDV, Anti-HEV.
 - Presence of HBsAb indicates immunity to HBV either from recovery of Hep.B or successful immunity.

Medical and Nursing Management of Hepatitis

- Reduce fatigue (pace nursing activities, rest periods, short but frequent activity)
- Maintain nutritional and fluid balance (follow I/O, albumin levels, low protein diet, CHOs, small frequent meals)
- Reduce effects of hepatitis
 - Antiemetics, antihistamines, vitamin K, bile acid sequestrants (Cholyseramine... it binds up bile salts and reduces itchiness, immune globulin (not as common now, using more Interferon), vaccines for other Heps
 - Medications to avoid (chlorpromazine, aspirin, acetaminophen, phenothiazine and many sedatives) Sedatives tend to last longer in these pts.

Jaundice Management

- Medical management
 - Determine cause of jaundice
 - Reduce pruritis and maintain skin integrity
 - Oral cholestyramine resin bind salts in GI*
 - Antihistamines*
 - Phenobarbital enhance bile flow (not seen a lot)
- Nursing management
 - Impaired Skin Integrity, disturbed body image, ineffective health maintenance

Jaundice usually clears once you resolve the underlying condition. Usually takes 4-6 weeks.

Complications of Hepatitis

- Fulminant hepatitis progression of liver necrotic process. Major complications...
 - Jaundice
 - Hepatic encephalopathy occurs b/c of high ammonia levels, decreased LOC, changes in comprehension, don't know where they are, can go to full coma. Can give mds to bind that up (Lactulose)
 - Ascites: pt may need paracentesis, or give albumin/lasix
- Chronic hepatitis liver inflammation (drug dosages will need to be adjusted)
 - Chronic active hepatitis B, C are most common ones that cause this problem
 - B superimposed with D
- Chronic carrier state (develop carcinoma)
- Aplastic anemia a rare complication of hep. (has to deal with the whole problem of not being able to store vitamins normally, esp B-12... and Vit K synthesis)

Mortality from hepatitis complications increase with age, especially people over 60 (approaches 90%)

Chronic Hepatitis

- Chronic hepatitis is liver inflammation > 3- 6 months
- Chronic active hepatitis (B 5% & C 50-70%)
- B superimposed with D
- Elevation of AST & ALT > 6 months
- Drugs & toxins methyl dopa, nitrofurantoin, amiodarone & isoniazid
- Autoimmune following hep. A, Epstein Barr or Measles infections.
- Metabolic disorders (alcoholic hep.)

A client with liver disease should avoid: Acetaminophen!

Liver Cirrhosis

Cirrhosis is a chronic progressive disease characterized by widespread fibrosis (scarring) of the liver and nodular formation. In cirrhosis, the normal flow of blood, bile and hepatic metabolites is altered by fibrosis. The pt will have high unconjugated levels and back-up in portal hepatic vein. It is the 10th, leading cause of death in the US. Note that 45% of cases are alcohol related, which means most of the cases are not due to alcohol. Don't be all judgmental with your patients! Though cirrhosis can be life threatening, it can be controlled if discovered early.

Cirrhosis Classification by cause

- Alcoholic or Laennec's or Micro-nodular (more common in men, most common form in North America)
- Post-necrotic (macro-nodular or toxin induced, also malaria-treatment drugs)
 - World-wide cirrhosis; more common in women; causes = chronic viral Hep B/C/D, tylenol, Isoniazide
- Biliary (bile duct obstruction, bile stasis and hepatic fibrosis)
 - Primary = disordered immune response damages bile ducts resulting in intrahepatic obstruction
 - Secondary = partial or complete obstruction of the bile duct from gallstones, strictures, pancreatitis, tumors
- Cardiac (results from prolonged right-side heart failure or constrictive pericarditis)

Caput Medusae

Caput Medusae is a plexus of dilated veins around the umbilicus. It is seen in patients with portal hypertension that is usually a result of cirrhosis.

Medical Management

The main goal with cirrhosis is to treat underlying cause...

- Modify medication doses and trend liver labs of ALT AST Albumin
- Diet changes (low protein diet to decrease production of ammonia)
- Monitor for complications
 - GI bleed
 - Renal failure
- Prevent infection

Nursing Diagnoses for Cirrhosis

- Ineffective Tissue Perfusion
- Imbalanced Nutrition: Less Than Body Requirements
- Activity Intolerance
- Risk for Injury
- Ineffective Protection
- Ineffective Health Maintenance

Nursing Management for Cirrhosis

- Ineffective tissue perfusion related to **bleeding tendencies* and varices***
 - Monitor for bleeding (labs: H/H, Plat, PT/INR PTT)
 - Check VS & urine output > 0.5ml/kg/hr.
 - Prevent bleeding and protect pt from physical injury
 - Teach pt to avoid straining, vigorous nose blowing, use soft tooth brush & avoid flossing
- Imbalanced nutrition less than body requirements related to anorexia & decreased absorption
 - Modify diet low fat, low protein, low Na (b/c of ascites)
 - High calorie 2500-3000
 - Daily wt, I/O, calorie count
 - Vitamin supplements (A,D,E,K, folic acid, thiamin supplements)
- Ineffective protection related to alcohol abuse and inadequate nutrition and leukopenia.
 - Prevent systemic infection
 - Monitor for signs of infection
 - Monitor for signs of ascites
 - Daily abdominal girth, daily weight, fluid wave
 - Monitor for signs of encephalopathy (hallucinations, lethargy, not oriented, not cooperative)
 - Confusion, agitation...elevated ammonia levels so check the ammonia!
- Activity intolerance related to bed rest, fatigue and lack of energy
 - Rest periods.
 - Space activities
 - Balance rest and ADL's
- Ineffective health maintenance related to lack of knowledge
 - Explain the disease process
 - Implications for long term management
 - Abstinence from alcohol
 - Nutrition

Complications of cirrhosis

- Portal hypertension: increased resistance to flow in the portal venous system (> 12 mmHg; normal is 5-10 mmHg).
 - GI Bleed
 - Portosystemic shunt (bypass of the liver by the body's circulatory system)
 - Splenomegaly
- Ascites
- Hepatic encephalopathy

Portal Hypertension

Again, this is an increased resistance to flow in the portal venous system above 12 mm Hg (normal 5-10 mm Hg). It involves compression and destruction of the portal and hepatic veins due to increasingly fibrotic hepatic tissue. The result is resistance to normal flow of blood through the portal system. This leads to a persistent increase in portal vein pressure. Major complications include splenomegaly, esophageal varices leading to GI bleed and ascites.

Management of Hemorrhage in Portal Hypertension

- T/S (Type and Screen) immediately for high risk patients

FYI:

All patients with cirrhosis experience parenchymal necrosis, regeneration, and scarring. This process is slow and progressive and dependent upon the presence or absence of the stimulating factor.

- Fluids/Blood
- Sclerotherapy (ligate or clip?)
- Transjugular intrahepatic portosystemic shunt (TIPS)
- Vasopressin
- Beta-adrenergic-blocking agents (propranolol= inderal, metoprolol=lopressor, or nadolol = Corgard).
- Balloon tamponade (see picture at right)

The lecture had various photographs of different esophageal problems which I didn't use here because they kind of all looked the same to me.

The Portacaval Shunt is a procedure to decrease the risk of bleeding. As you can see from the picture, the portal vein is redirected straight into the SVC. This releases the pressure of portal hypertension since some of the blood is now going straight into the SVC and bypassing that high-pressure area. The drawback is that the blood doesn't get cleaned in the liver like it's supposed to. A person with this type of shunt will take a long time to metabolize drugs.

Another procedure that is done (but not very common) is a LeVeen peritoneovenous shunt. It moves fluid from the peritoneal cavity into the SVC.

Liver Failure (leads to...)

- Hematologic disorders
- Endocrine disorders
- Skin disorders
- Hepatorenal syndrome
- Hepatic encephalopathy

Hepatorenal Syndrome (a type of Liver Failure)

- Advanced liver failure with ascites → decrease renal perfusion → renal failure
- Progressive azotemia (elevated waste products)
- Increased serum Cr. Levels
- Oliguria and urine Na < 10mEq/L
- Azotemia & elevated levels of Ammonia → hepatic encephalopathy → coma.
- Pts will not respond to diuretic therapy for ascites

Hepatic Encephalopathy: Medical Management

- Identify and treat cause
- Reduce nitrogenous waste in blood
- Reduce bacteria in colon
- Maintain fluid volume balance

Liver Failure Management Medications (KNOW THESE MEDS!)

- Diuretics (Spironalactone, Lasix)
- Folic acid, thiamine, vitamins, minerals
- Albumin (increases oncotic pressure to reduce ascites)
- Lactulose (reduces blood ammonia levels)
- Neomycin
- Octreotide (used to decrease flow to blood vessels in GI system, helps to control GI bleed)
- IV infusion to stop internal hemorrhage and lower splanchnic blood flow
- Bolus: 100 mcg
- Infusion: 10 mcg/hr

Liver Failure Management

- Eliminating alcohol intake is very very important!
- Monitor for signs of bleeding (teach patient about bleeding precautions also)
- Preventing infection

- Providing sufficient carbohydrate & calories to prevent protein break down (low protein diet!)
- Correcting fluid & electrolyte imbalance...particularly hypokalemia
- Decrease GI NH₄ (ammonia) by controlling protein intake and administering lactulose to bind ammonia.
- Liver transplant may be the only effective treatment

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