Cirrhosis:
Pathogenesis and Complications

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Infections
• Hepatitis

Auto-Immune
• AIH, PBC, PSC

Toxin
• Ethanol

Metabolic Dx
• NAFLD

Genetic Dx
• HH, Wilson's, A1AT

Note on abbreviations:
- **-AIH:** Auto-Immune Hepatitis
- **-PBC:** Primary Biliary Cirrhosis
- **-PSC:** Primary Sclerosing Cholangitis
- **-NAFLD:** Non-Alcoholic Fatty Liver Disease
- **-HH:** Hereditary Hemochromatosis
- **-A1AT:** Alpha-1 Anti-Trypsin

(These disorders, as well as all other causes of Liver Cirrhosis, will be described in subsequent slides)

Death of hepatocytes, inflammatory destruction of normal hepatic architecture → Scarring, fibrosis

Liver is highly regenerative, but here it must regenerate within extensively scarred/fibrotic tissue, forming nodules of poorly-functioning cells → disrupting hepatic vasculature, biliary production/excretion, and other liver functions.

Cirrhosis

↑ resistance to blood flow through fibrotic liver

Hepatocellular carcinoma (HCC) (85% of HCCs occur in background of cirrhosis)

↓ liver function **("liver insufficiency")**

Portal hypertension:
↑ blood pressure in the hepatic circulation

Kideys retain more water & Na+ to ↑ blood volume

Blood backs up into the collateral venous system

↑ hydrostatic pressure in abdominal capillaries

↓ effective blood volume felt by kidneys

↑ vasodilators (NO, CO)

Into esophageal varices:
• Hemorrhage could cause an **Upper GI Bleed!**

Into rectal varices:
• Hemorrhage could cause an **Lower GI Bleed!**

Into the spleen: Splenomegaly
(In turn, a congested, enlarged spleen could lead to ↑ trapping of blood cells within, which could cause pancytopenia)

Fluid exudes from plasma in the capillaries

Fluid exudation into peritoneal cavity→ **Ascites**

Low Albumin Synthesis

↓ oncotic pressure in systemic capillaries

Fluid exudes from plasma in the capillaries into interstitial tissues

Edema

Liver unable to synthesize clotting factors or anti-coagulant proteins

Low measured clotting factors 10, 5, 2, etc; low Protein C, Protein S, antithrombin, etc

Usually the ↓ in pro- and anti-coagulants are in balance, no coagulopathy results

Liver unable to remove toxins from body

Toxins (i.e. NH3) build up, cross blood-brain-barrier

Encephalopathy (degeneration of neurological function: confusion, asterixis, etc)

• ↓ conjugation of bilirubin
• ↓ secretion of conjugated bilirubin into bile duct canaliculi
• ↓ drainage of conjugated bilirubin out of the ducts

When bilirubin > 40-50: **Jaundice, Sclera icterus**

Legend: = pathophysiology = mechanism = sign/symptom/lab finding = complications

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Signs (Stigmata) of chronic liver disease

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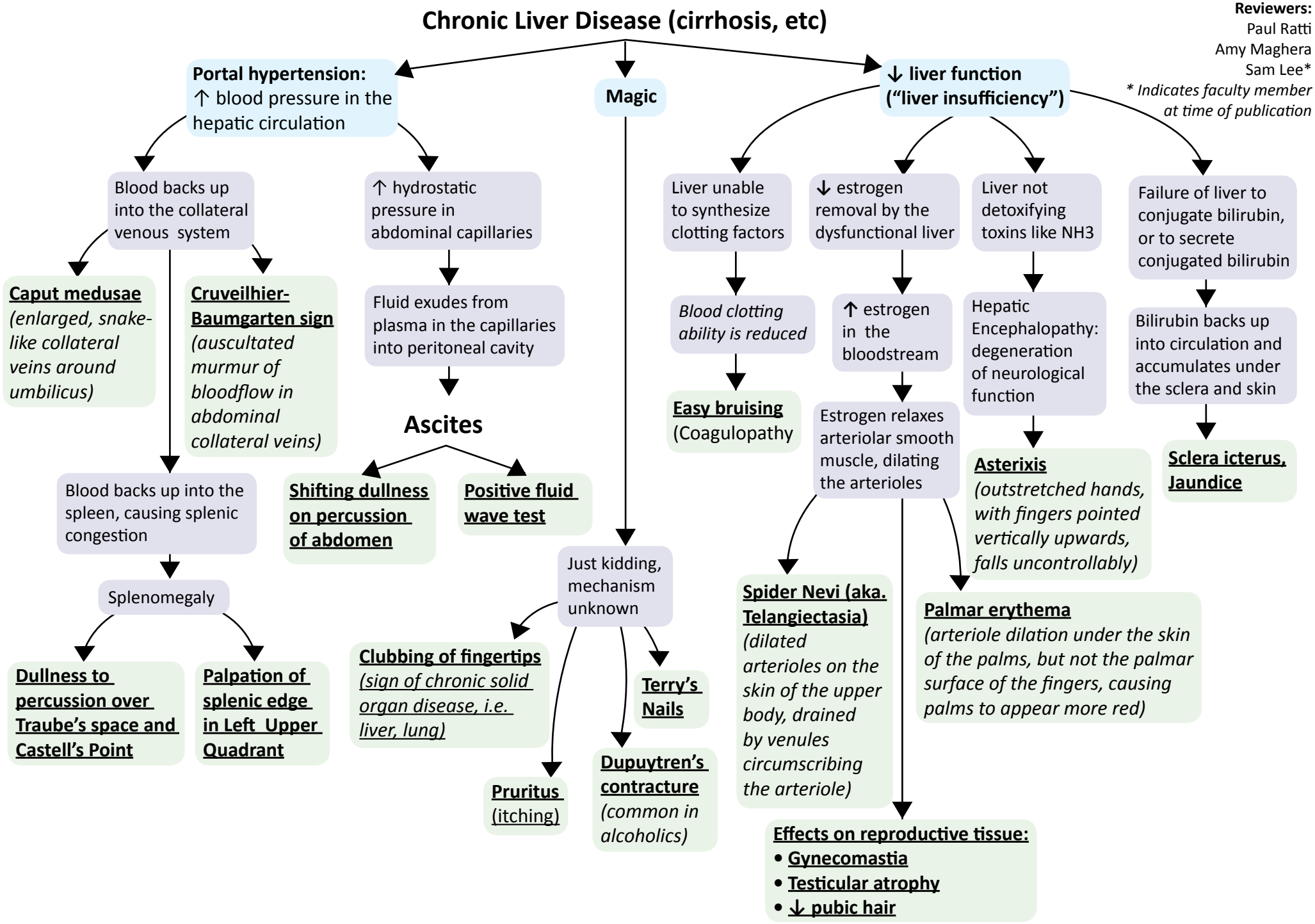

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