



Surgical Liver Disease

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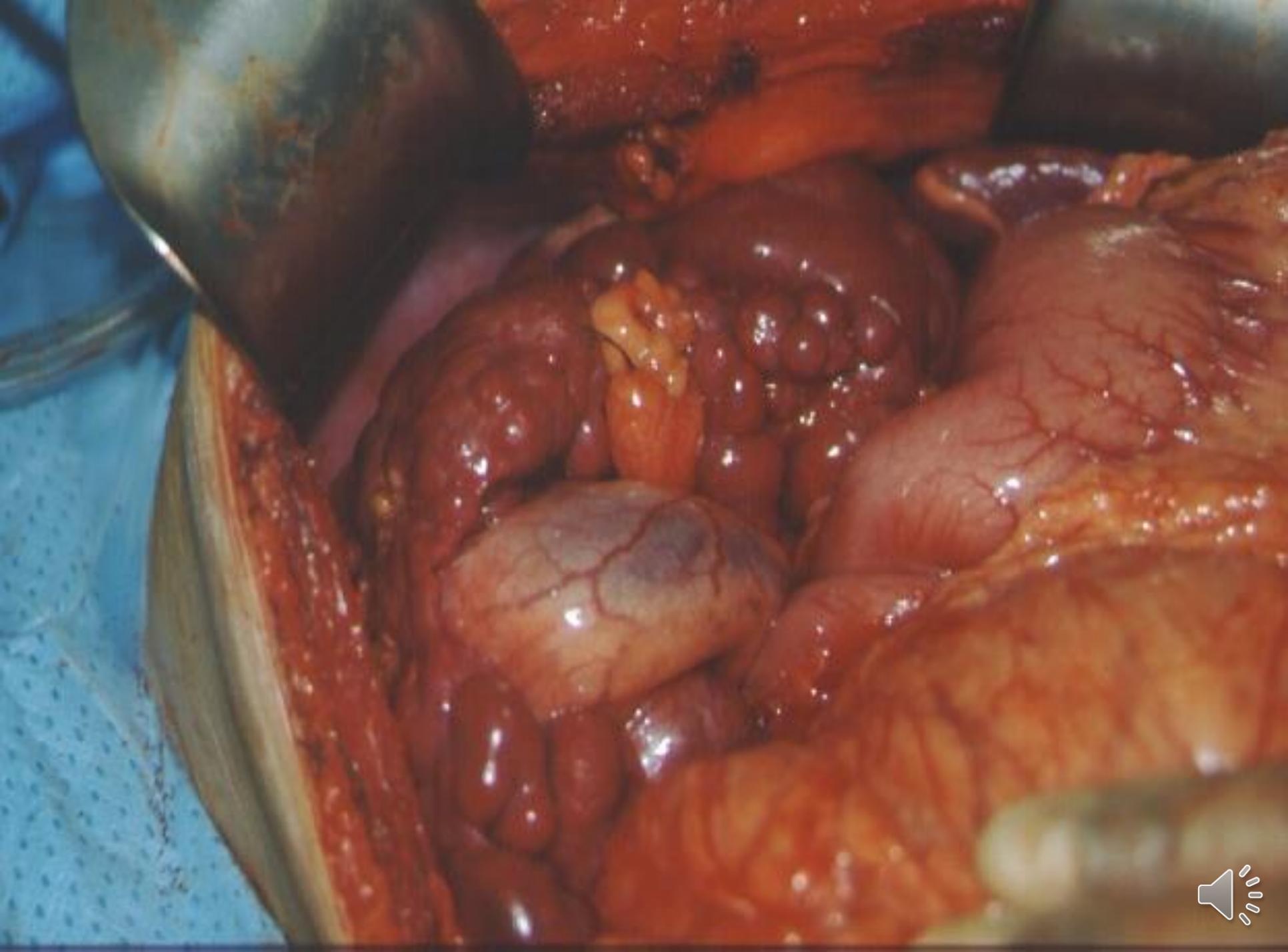
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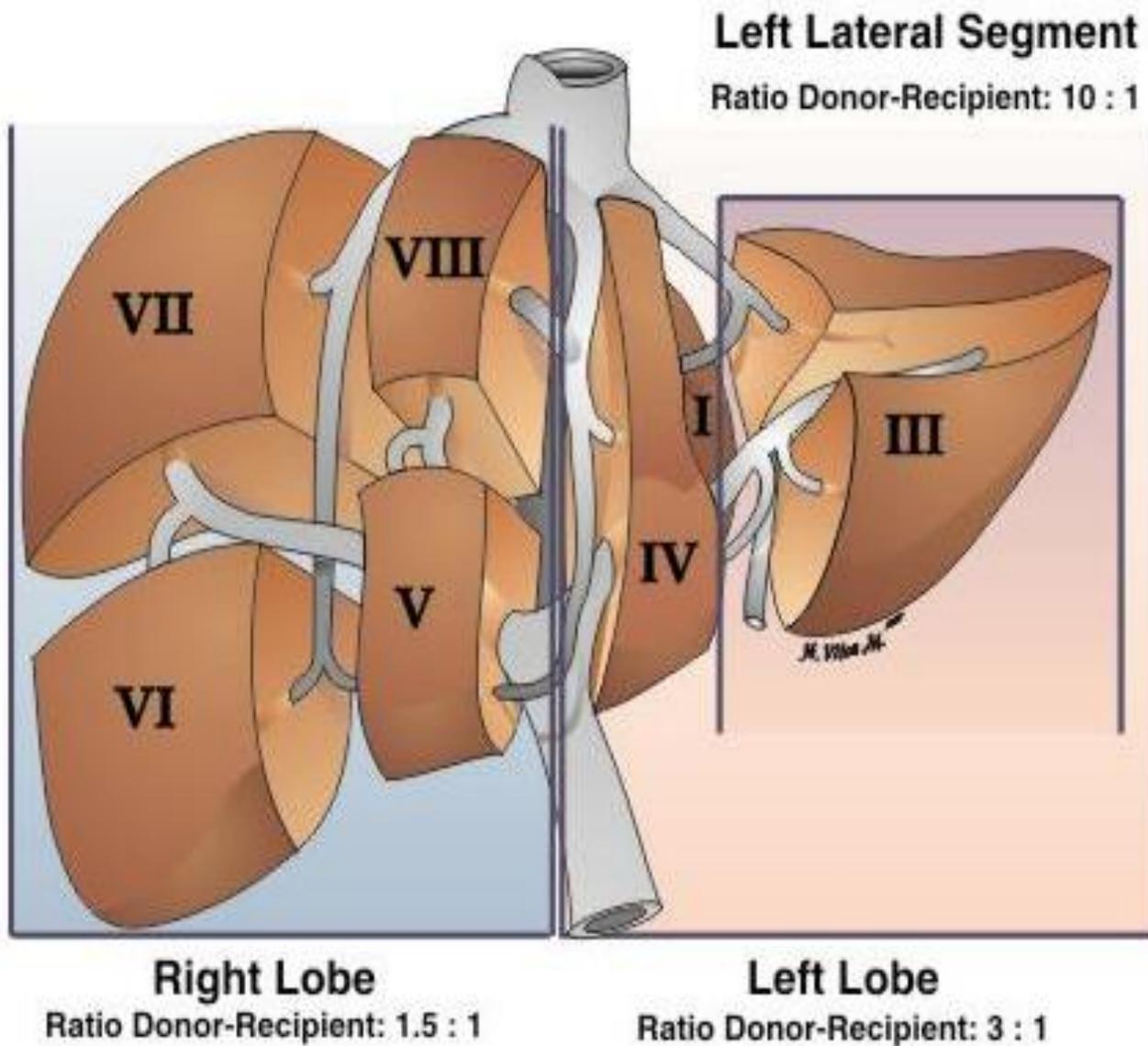




Anatomy

- 4 Lobes:
 - Left
 - Right
 - Caudate
 - Quadrate
- 3 vessels
 - Hepatic portal vein
 - Hepatic artery
 - Hepatic vein







Aetiology

- Abnormal LFTs
- Non-alcoholic fatty liver disease
- Primary biliary sclerosis
- Primary sclerosing cholangitis
- Hepatocellular Carcinoma (rare)
- Metastasis (common)





Abnormal LFT's

- Asymptomatic elevation of ALT is most common problem
- If isolated and less than 3-fold elevation then stop alcohol or drug and recheck in 2-3 months
- If persistent then further workup is needed





ALT and AST

- Enzymes, found in Hepatocytes
- Released when liver cells damaged
- ALT is specific for liver injury
- AST (SGOT) is also found in skeletal and cardiac muscle





ALP and GGT

- Found in hepatocytes that line the bile canaliculi
- Level is raised in Biliary obstruction (causes stretch of the bile canaliculi)
- BUT also found in BONE and PLACENTA
- GGT is also found in bile canaliculi and therefore can be used in conjunction with Alk Phos for predicting liver origin
- BUT GGT can be raised by many drugs including Alcohol and therefore non specific





BILIRUBIN

- Water insoluble product of heme metabolism
- Taken up by liver and conjugated to become water soluble so it can be excreted in bile and into bowel.
- Patient looks Jaundiced if bilirubin >2.5 mg/dL
- If patient is vomiting GREEN, then they have bowel obstruction below the level of the Ampulla of Vater.





Direct and indirect bilirubin

- Prehepatic disease (e.g. hemolysis) causes high bilirubin which is non conjugated i.e. Indirect fraction higher
- Hepatic disease causes increased conjugated and unconjugated bilirubin
- Post hepatic disease e.g. Gallstones have increased conjugated (direct) bilirubin and lead to dark urine and pale stool.





Abnormal ALP

- Hepatic
 - PBC (middle aged women)
 - PSC (IBD history)
 - Gallbladder/stone disease
 - Meds (tetracyclines, OCP's, ceftriaxone)
 - Infiltrative liver disease (sarcoid, TB, CA)
- Pregnancy
- Bone (Mets or Paget's disease)





Hepatology Pearls

- Hepatitis: $\uparrow\uparrow$ AST and ALT
- Cholestasis: $\uparrow\uparrow$ TB and ALP
- ALT more specific than AST
- Measures of function: ALB, Coags, Bilirubin
- Alcoholic hepatitis $AST > ALT$ 2-3:1 (NASH with cirrhosis also)





TYPICAL PATTERNS

- HEPATOCELLULAR

- Increased transaminases

- Viral Hepatitis
- Drugs/alcohol
- Autoimmune
- NASH
- Hemochromatosis

- CHOLESTATIC

- Increased Alk Phos and Bilirubin

- Also may cause increased transaminases

- Gallstones
- Primary Biliary Cirrhosis
- Sclerosing Cholangitis
- Peri ampullary tumors





Abnormal LFT's

- Mildly high ALP or TB without evidence of biliary dz, think infiltrative (TB, sarcoid, fungal) or metastatic disease
- Workup mainly by history and risk factors
- Image or biopsy for diagnostic purposes is not always needed





Abnormal ALP

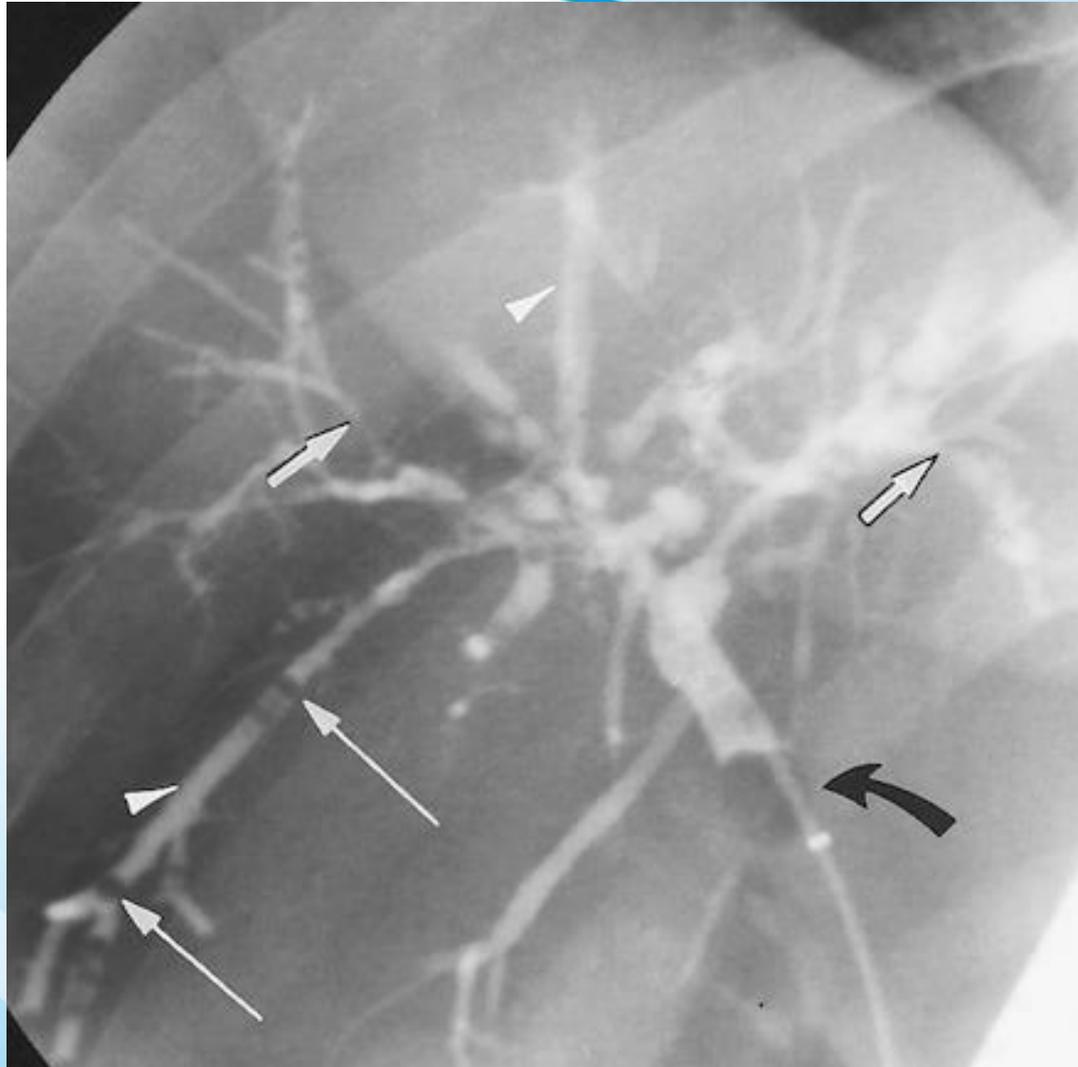
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Primary Biliary Cirrhosis (PBC)

- Middle-aged woman
- F:M 9:1
- Elevated alkaline phosphatase
- Symptom: itching!
 - Later: jaundice, osteomalacia, osteoporosis
- Xanthomas/xanthelasmas
 - hypercholesterolemia with low risk CAD
- Diagnosis:
 - antimitochondrial antibody (AMA): 98%
 - liver bx: Chronic granulomatous inflammation of interlobular bile ducts
- Tx:
 - Early: Ursodeoxycholic acid
 - Late: liver transplant







Primary Sclerosing Cholangitis (PSC)

- Male: female 2:1
- Intra and Extrahepatic inflammation and sclerosis of the biliary tree
- Strong Association (75%) with IBD (Ulcerative colitis)
- Cholestatic enzyme pattern (TB and ALP)
- ANCA: +ve 80%
- Dx: ERCP
- Cholangiocarcinoma: 15-30%
- Tx:
 - Stenting of dominant strictures
 - Liver Transplant





NASH/NAFLD

- 5% population
- Risk Factors:
 - Obesity
 - DM
 - Hyperlipidemia
 - Pregnancy (high oestrogen)
 - TPN
- Dx:
 - USS: fatty infiltrariopon, hepatomegaly
 - Histology: Steatosis, Necrosis
- 20-30% progress to cirrhosis
- Tx: Wt Loss (>15%), Exercise, Control of DM, Lipid Lowering Agents



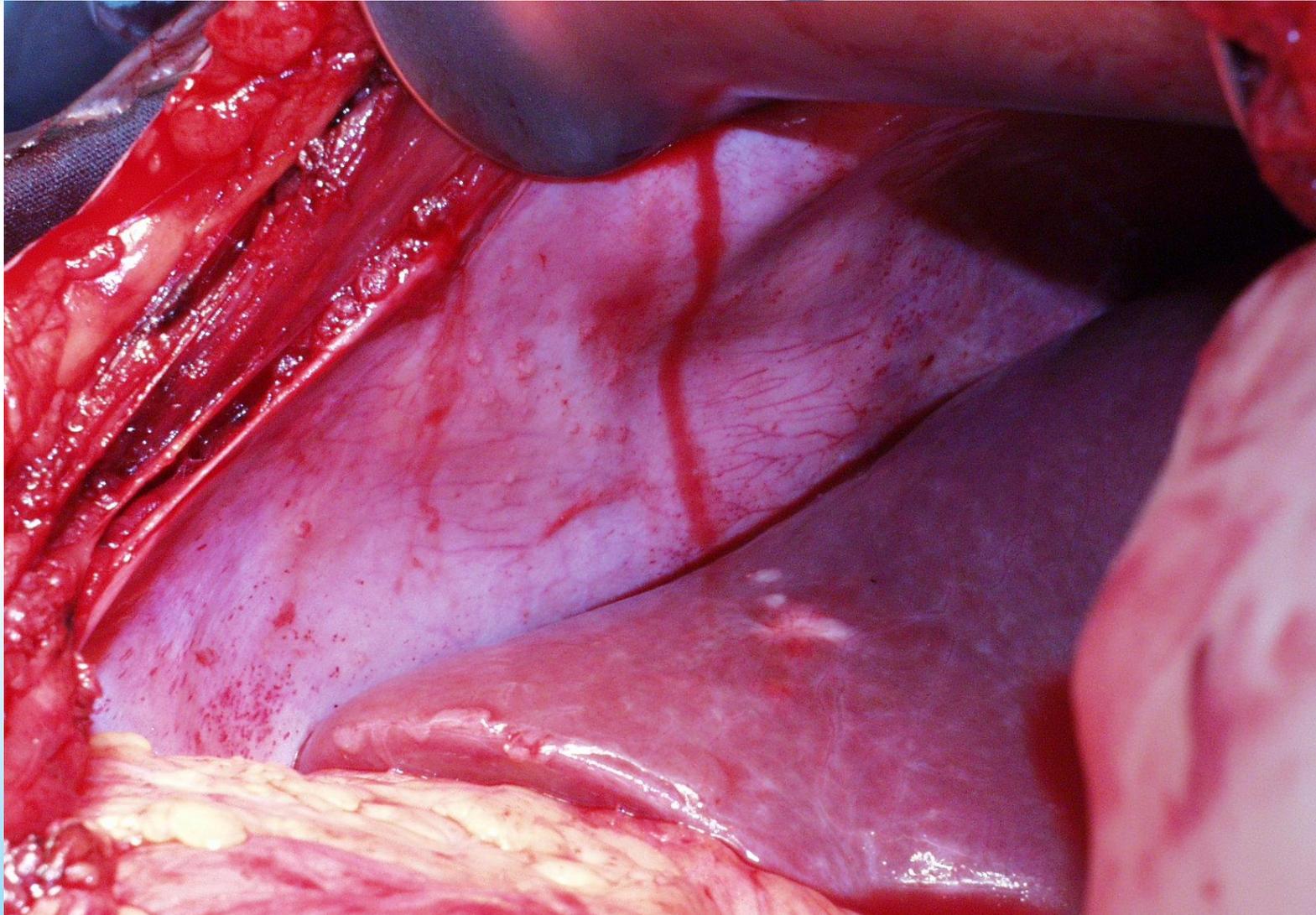


Hepatocellular Carcinoma

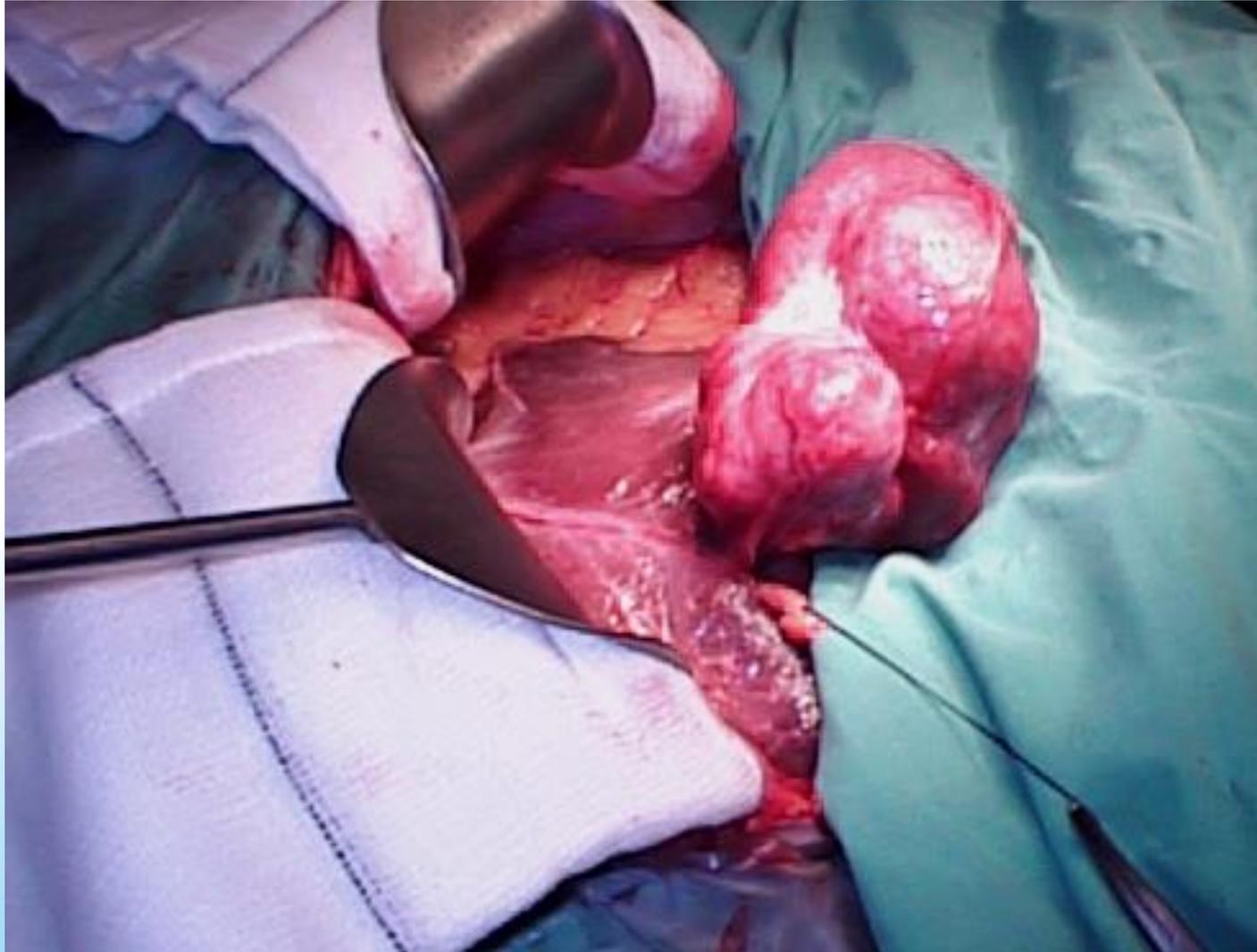
- Risk factors:
 - HCC > 75% in setting of cirrhosis
 - Hep B and C
 - Exposure to Aspergillus Flavus toxin
 - Aflatoxin (raw peanuts, raw peanut butter)
- Screening
 - Alphafetoprotein should be checked annually in patients with cirrhosis.
- Need USS if AFP > 100
- Less than 15% are resectable at diagnosis.



Remember don't Biopsy if resectable

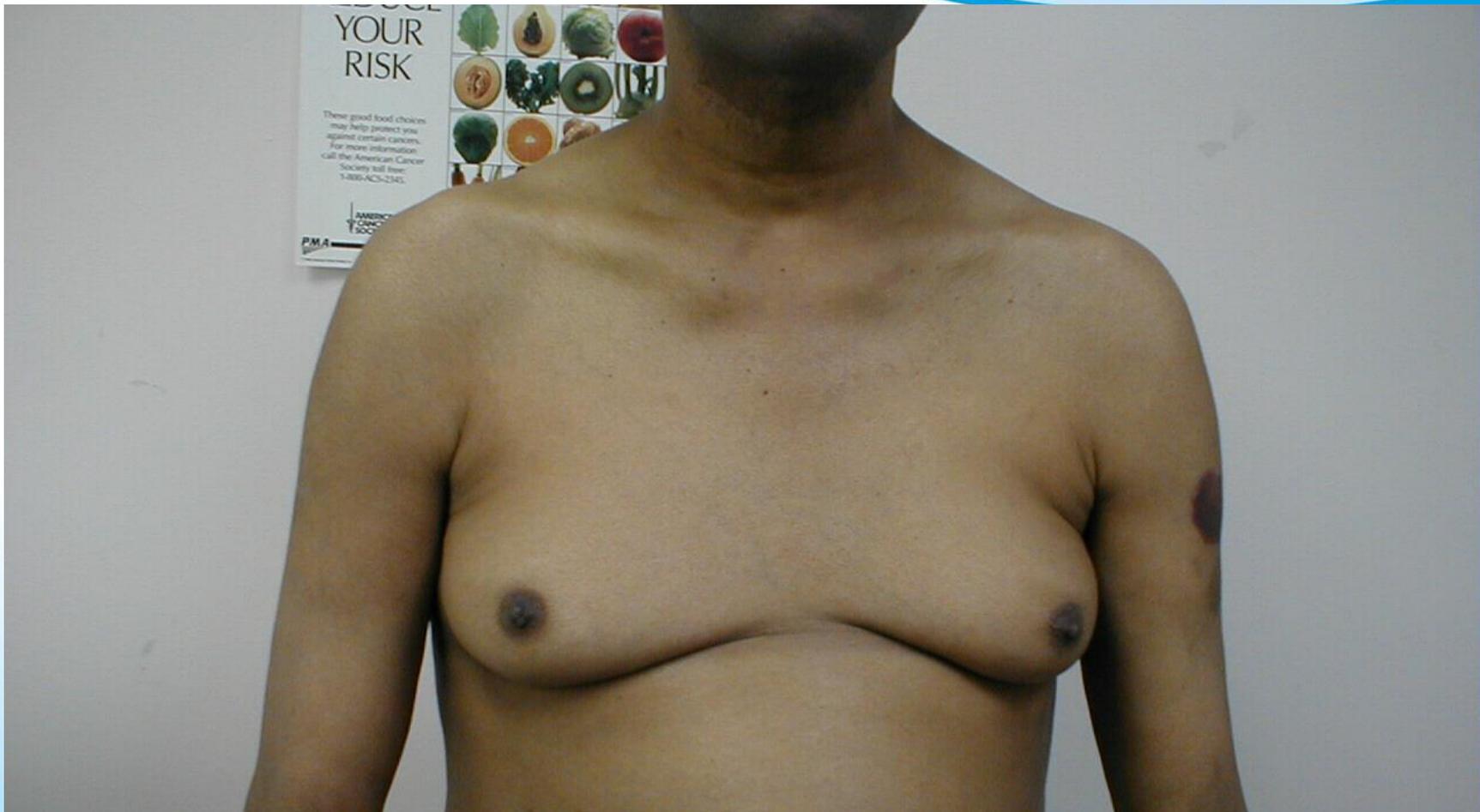


Non colorectal Tumours











Childs-Pugh classification

Child-Pugh classification of liver failure

	No of points		
	1	2	3
Bilirubin ($\mu\text{mol/l}$)	< 34	34-51	> 51
Albumin (g/l)	> 35	28-35	< 28
Prothrombin time	< 3	3-10	> 10
Ascites	None	Slight	Moderate to severe
Encephalopathy	None	Slight	Moderate to severe

Grade A = 5-6 points, grade B = 7-9 points, grade C = 10-15 points.



Liver transplant

- Treatment for:
 - most end-stage liver disease
 - confined liver cancer
 - fulminant failure not responding to supportive measures
- Need to consider in
 - all decompensated cirrhotics
 - encephalopathy, ascites, variceal bleeding, albumin <2.5
 - Also indicated for intractable pruritis, such as in PBC
- Contraindicated
 - Active EtOH or drug use,
 - Metastatic CA,
 - severe lung or cardiac disease
 - HIV infection

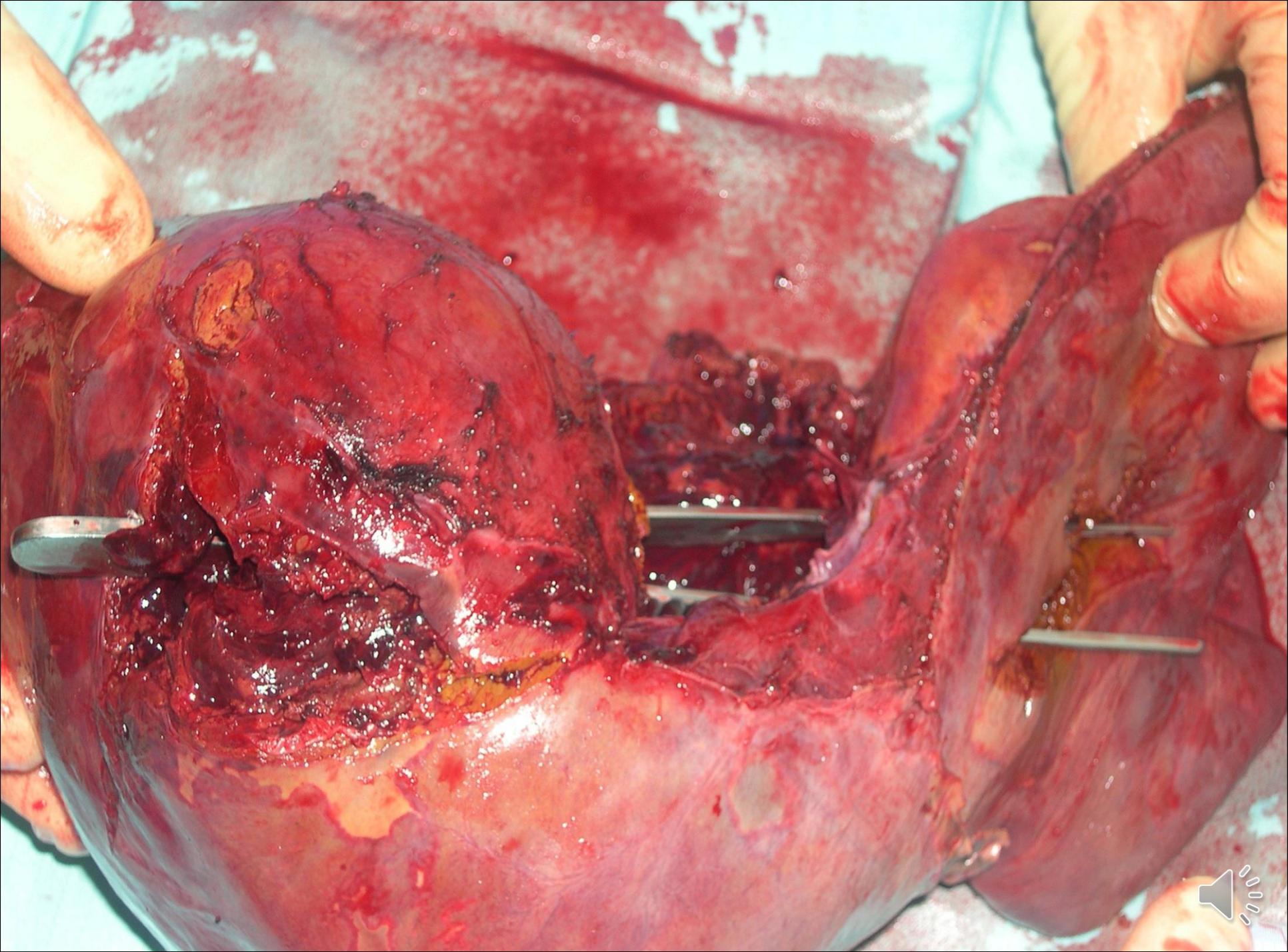




Liver trauma

- Commonest cause of death <30 years
- 3rd most common for all age groups
- Preventable
- Prompt assessment and treatment
- Trauma centres ?







Mechanism of liver trauma

BLUNT

Multiple injuries

Mechanism:

Direct

Shearing / rotational

Deceleration

PENETRATING

Gunshot

Stab wound





Management of liver trauma

Gunshot – operate

Stab wound –operate selectively

Blunt trauma – operate selectively





Liver Trauma (Grades)

- I Haematoma subcapsular, non expanding, <10% of surface
Laceration tear <1cm deep)
- II Haematoma non expanding, subcapsular 10-50%% of surface
intraparenchymal <2cm diameter
Laceration tear 1-3cm deep, <10cm long
- III Haematoma subcapsular, expanding, >50% of surface or
ruptured; intraparenchymal >2cm or expanding
Laceration tear >3cm deep
- IV Haematoma Ruptured intraparenchymal, bleeding
Laceration Disruption involving 25-50% of lobe
- V Laceration Disruption >50%
Vascular - Juxta hepatic venous injury (IVC or hepatic vein)
- VI Vascular Hepatic Avulsion



How does grading help ?



Grade I and II generally can be managed non-operatively

Grade III - V generally require surgery

Grade VI is incompatible with survival





Predictors of success

Neither grade of injury and/or degree of haemoperitoneum on CT predict the outcome

Haemodynamic status of the patient is the most reliable predictor

Presence of a contrast blush on the vascular phase of the CT indicates need for interventional angiography





MANAGEMENT OF LIVER TRAUMA

PLAN - Early surgical

Bilateral subcostal incision

Vascular control

Pringle manoeuvre

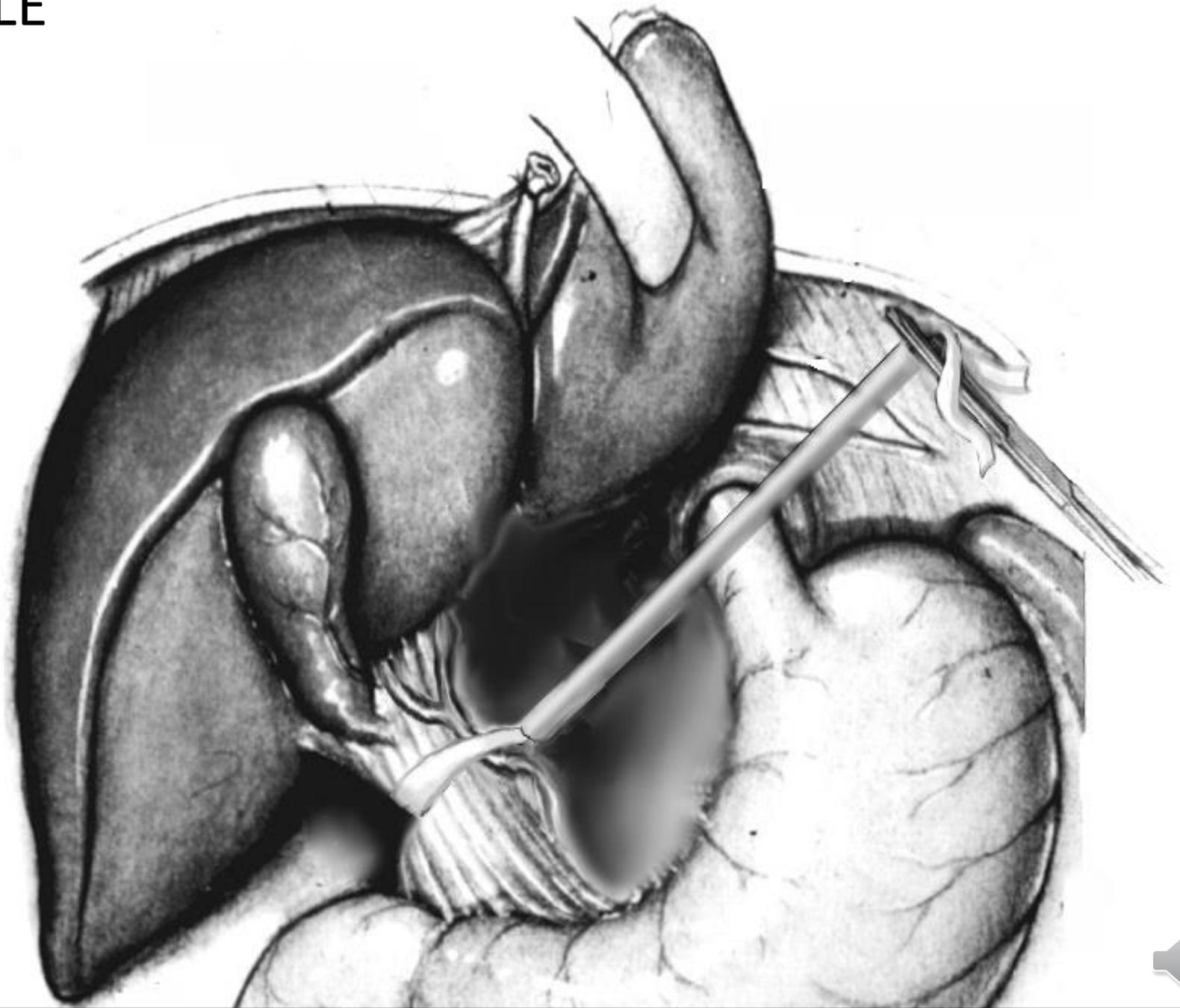
Aortic clamp / compression

Allow the anesthetist to resuscitate before continuing

Packing



PRINGLE





Surgical management

- Limited mobilization of the liver by dividing falciform ligament
- Place packs to compress injured liver
- Avoid compressing IVC
- Avoid lifting the liver up
- Avoid raised intra-abdominal pressure





Complications

- Sepsis
- Biliary leak or stricture
- Haemobilia
- Aneurysm
- Arterioportal fistula

