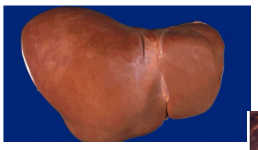
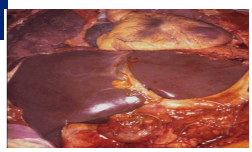
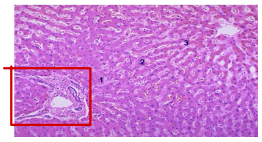
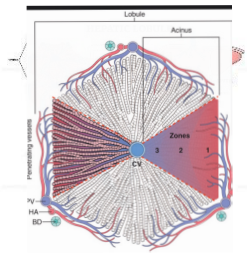
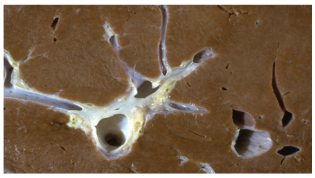




Patho 1

Liver

Net weight	1400-1600 g (2.5./. of body weight)
Blood supply	Portal vein: 60-70./. (MAIN) Hepatic artery: 30-40./.
Microstructure (we need to know the structure because any change→ disease) *central vein is not dilated, and not surrounded by fibrosis Liver zones  	Hexagonal lobules→ 1 hexagonal lobule (functional unit) (centre of hexagon has central vein)→6 acini. 1 acinus→ Portal areas (each area has 3 structures, hepatic art. Portal vein and bile duct) + 3 zones. These zones are in the parenchyma (within the hepatocytes) Zone 1: periportal areas (close to the vascular supply) (surrounding portal tract) Zone 2: intermediate between zone one and three Zone 3: Pericentral area (close to central vein) We divide them into zones because some diseases favor beginning at a certain zone, but this doesn't mean only one zone will be affected, but it can be helpful in diagnosis at beginning of disease
Colour	Brown
Surfaces	Covered by abdominal fat, shiny and smooth
Consistency	-Homogenous (all liver parts have the same colour and structure)
Hepatocyte arrangement *clinical note; if the arrangement changes from plate like arrangement→ disease Cross section of normal liver 	-The parenchyma is organized into plates of hepatocytes, hepatocytes are radially oriented around the terminal hepatic vein(central vein) -Vascular sinusoids present between cords of hepatocytes - Arranged in plates (One cell thick lines ,so they can be in close relation with blood supply) separated by sinusoids (vascular spaces filled with portal blood) TO SUM UP= hepatocytes—basement membrane and one layer close to sinusoids—sinusoids -Hepatocytes show only minimal variation in the overall size but nuclei may vary in size number and ploidy especially with advancing age
Biopsy	We need biopsy 1- diagnosis (liver disease might show similar clinical presentations) 2-follow up (after treatment we follow up to check if the patient responded to treatment or the if the liver reached the chronic phase) We need to check the size/appearance of hepatocytes and their arrangement in plates + check the sinusoids and blood vessels TYPES: -Needle biopsy (using a needle) -Cord biopsy -Wedge biopsy (through abdomen and by taking a part of liver)

Functions of liver

Notes

Metabolic	Glucose	
Synthetic	albumin and clotting factors, proteins, enzymes, BILE	In many diseases, we can see altered liver functions, like in excessive bleeding we have problem in synthesizing clotting factors
Detoxification	External toxins: Drugs Internal toxins: hormones, NH3	If we suspect a liver disease, we need to make sure the cause isn't drugs (IMP), so we don't exaggerate the disease even more by giving a contraindicated drug
Storage	Glycogen, triglycerides, Fe, Cu, vitamins	Increase in storage = injury/ disease
Excretory	bile	Excreted to small intestine, in some liver diseases, patients suffer from inability to secrete bile, that leads to stasis and accumulation in hepatocytes → injury

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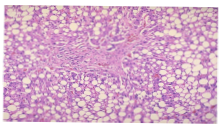
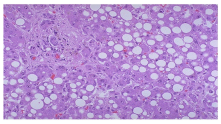
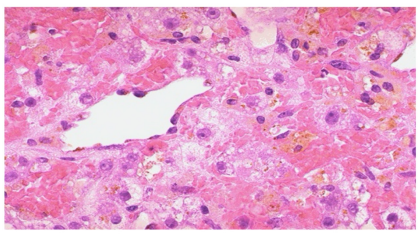
Note: * diagnosis of liver diseases is mandatory, and the first thing we do is to exclude the cause being because of a drug, to give the right drug, we can diagnose liver diseases by detecting liver changes and lab tests.

* any accumulation inside the hepatocytes (even if it was stored in its normal place) will cause hepatocyte injury.

Hepatic injury

Inflammation (hepatitis)	Can be **acute** (manifested by neutrophils) or **chronic**. Can be **infectious** or **non-infectious**
Degeneration	-**Ballooning degeneration** cells increase in size because something (mainly water due to electrolyte imbalances because of ATP failure (in hypoxia for eg), enters the cell and make it swell. early manifestation -**Feathery degeneration**: accumulation of fat -Can also be **because of iron, copper, biliary material accumulation** (depending on the underlying process of disease)
Steatosis (fatty change)   	NOT A DIAGNOSIS – IT'S A DESCRIPTION FOR WHAT IS GOINGIN THE LIVER, liver changes colour to yellow and it gets greasy, structure become compressed because hepatocytes enlarge, present in two very similar forms: -**Microvesicular** = small globules accumulated in cytoplasm e.g.= ALD, Reye syndrome, acute fatty change of pregnancy -**Macrovesicular**= large globules / 2 globules in cytoplasm, usually related to metabolic diseases or non-alcoholic fatty liver diseases , nucleus become peripheral and the cell gets narrower e.g.= DM and obesity
Necrosis *Very significant indicator to severe injury, Important mark for evaluation of disease process and follow up for patient *necrosis should be so extremely massive to cause necrosis *interface hepatitis; تشمل parenchyma *to say a cell is dead we have many indicators but mainly nucleus fragmentation or absence. Necrosis of liver 	Death of cells DEPENDING ON TYPE (imp give us info. About underlying cause) -**Coagulative necrosis** (vascular problem like thrombus) -**Councilman bodies** (individuals shrunken eosinophilic cells that are dead ,with pyknotic nucleus , there presence indicate that the liver has been exposed to injury, due to toxicity// vascular problems, sometimes when we examine the biopsy we don't see inflammation nor necrosis but we see these bodies, and they indicate a previous injury) -**Lytic necrosis (LIQUIFACTIVE)**(frequently related to infection) DEPENDING ON THE CAUSE **Ischemia** (can be systemic vascular problem or due to use of a certain drug for e.g), **shock** DEPENDING ON LOCATION -**Centrilobular necrosis** -**Midzonal** -**Periportal**: interface hepatitis (death of cells surrounding portal area usually due to inflammation) -**Focal**: piece meal necrosis (not used anymore, we call it interface hepatitis,), bridging necrosis -**Diffuse**: massive and submassive necrosis ; depending on injury and how the liver dealt with it, this type result from exposure to severe toxin , drugs (anesthetics) used in a short period of time , viral diseases (fulminant)
Regeneration *Hepatocytes have high regenerative capacity, functionally and morphologically, (only 5-10./. of hepatocytes can compensate)	it is evidenced by increased mitosis (**compensatory hyperplasia**) or cell cycle markers, the cells of the canal of the hering are the progenitor for hepatocytes and bile ducts cells(Oval cells). Cells will lack this ability when we have severe damage
Fibrosis *minimal amount is present in portal area and surrounding central vein. We don't have fibrosis in the parenchyma (basement membranes have collagen only) *any increase in fibrosis → process is irreversible going to chronicity (we look for it in follow ups because its imp)	-**portal or periportal fibrosis** -**pericentral** (around the central vein) - **Cellular fibrosis or fibrous tissue** may be deposited directly within the sinusoids around single or multiple hepatocytes -**bridging fibrosis (** fibrosis connecting 2 areas together, portal-portal, central-portal, central-central) , imp sign to be evaluated because → one of the initial signs that show the possibility to develop cirrhosis
Cirrhosis (micronodular & macronodular)	organized fibrosis, totally irreversible, اعراض لمدى الحياة
Ductular proliferation	In certain diseases, that primary affect biliary system → increased number of bile ducts, we accept 1-2 bile ducts in the portal area, any increase→ indicate proliferation due to obstruction (so when we take the biopsy we have to check what was the initial cause that lead to proliferation)