التخدير-كورس اول

LIVER DISEASE AND ANESTHESIA

جامعـــــــة ســـــــــاوة الاهلية

كليـــــة التقنيـــات الصحيـة والطبيـة

قسم تقنيـــات التخديـــــــر

المرحلـــــــــــــة الثالثة.

Sawa University

College of health and medical techniques

Department of Anesthesia

3rd Stage cours 1

نظـــــــري محاضرة رقم 7

Theoretical Lecture No. 7

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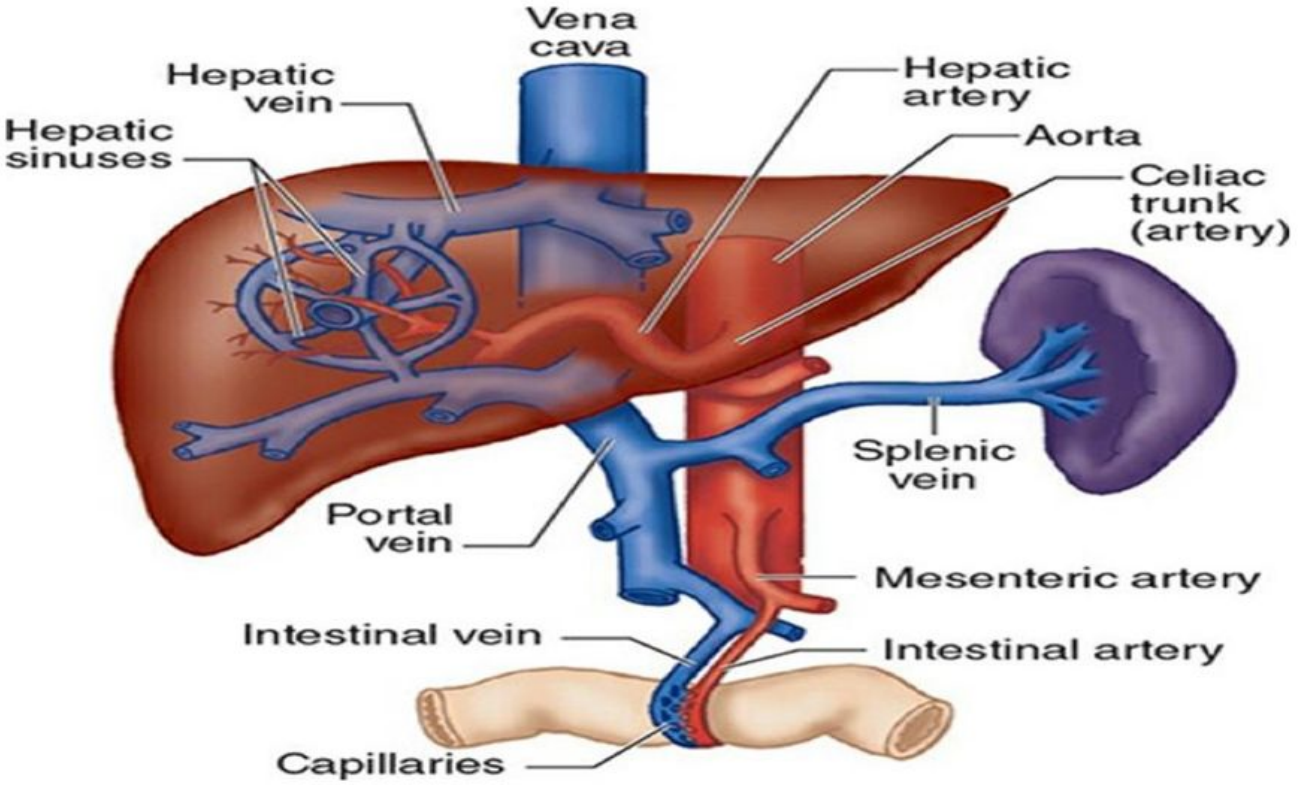
FUNCTIONAL ANATOMY

weighing approximately **1500 g** in adults.

It is separated by the falciform ligament into **right** and **left** anatomic lobes.

The liver is made up of **50,000** to **100,000** discrete anatomic units called lobules.


Vena cava
Hepatic vein
Hepatic sinuses
Hepatic artery
Aorta
Celiac trunk (artery)
Splenic vein
Portal vein
Mesenteric artery
Intestinal vein
Intestinal artery
Capillaries

Hepatic blood flow

- **20%** of cardiac output

Hepatic artery

- **30%** of hepatic blood flow
- **90%** of oxygen deliver

Portal vein

- **70%** of hepatic blood flow
- **10%** of oxygen deliver

HEPATIC FUNCTION

- **Metabolic:** metabolism of carbohydrate, lipid, amino acid, ammonia formation, interconversion of sugars and metabolism of drugs
- **Regulation** of blood glucose
- **Synthesis** of protein and clotting factors
- **Secretory:** secretion of bile in the intestine, and conjugation of bilirubin.
- **Detoxification:** detoxification of drugs, steroids, thyroid hormones, and endogenous metabolites.
- **Storage:** storage of glycogen, B12, iron, and vitamin A

LIVER FUNCTIONS TESTS

The most commonly performed liver tests are neither sensitive nor specific.

*No one laboratory test evaluates overall hepatic function

Many “liver function” tests, such as serum **transaminase measurements,** reflect hepatocellular integrity more than hepatic function.

Liver tests that do assess hepatic synthetic function include **serum albumin, prothrombin time (PT)** or **international normalized ratio (INR), serum cholesterol**, and **plasm pseudocholinesterase**.

Serum Bilirubin

The normal total bilirubin concentration, composed of **conjugated (direct)**, **water soluble**, and **unconjugated (indirect) lipid-soluble forms**, is less than **1.5 mg/dL (<25 mmol/L)** and reflects the balance between bilirubin production and excretion.

Jaundice

is usually clinically obvious when total bilirubin exceeds **3 mg/dL.**

conjugated hyperbilirubin is may reflect hepatocellular dysfunction, congenital or acquired intrahepatic cholestasis, or extrahepatic biliary obstruction

unconjugated may be seen with hemolysis or with congenital or acquired defects in bilirubin conjugation.

Unconjugated bilirubin is neurotoxic, and high levels may produce encephalopathy

Serum Aminotransferases (Transaminases)

These enzymes are released into the circulation as a result of hepatocellular injury or death.

Two types:

- aspartate aminotransferase (AST)
- alanine aminotransferase (ALT)

Serum Alkaline Phosphatase

Test	Normal Range (Seconds)
PT (Prothrombin Time)	11–13.5
PTT (Partial Thromboplastin Time)	25–35
INR (International Normalized Ratio)	0.8–1.1

Serum Albumin

The normal serum albumin concentration is **3.5 to 5.5 g/dL.**
Because its half-life is
approximately **2 to 3 weeks**, albumin concentration may initially be normal with acute liver disease.

Albumin values less than **2.5 g/dL** are generally indicative of chronic liver disease, **acute stress, or severe malnutrition.**

Hypoalbuminemia caused by
-malnutrition,
-nephrotic syndrome,
-malabsorptive states
-late pregnancy

Blood Ammonia

Significant elevations of blood ammonia levels usually reflect disruption of hepatic urea synthesis.

- Normal levels are **47 to 65 mmol/L (80–110 mg/dL).**

Marked elevations usually reflect severe hepatocellular damage and may cause encephalopathy.

LIVER DISORDER

Obstructive disorders primarily affect the **biliary excretion of substances**

parenchymal disorders result in generalized **hepatocellular dysfunction LFTs** measure the concentrations of various proteins and enzymes in the blood that are either produced by liver cells or released when liver cells are damaged.

DIAGNOSIS OF LIVER DISEASE

- -History
- -Examination
- -Investigation

- Ascites and distended abdomen
- Enlarged liver
- Depression
- Seizures
- Weight loss
- Jaundice

ACUTE LIVER FAILURE

defined as the rapid development of:

1. jaundice,
2. coagulopathy,
3. encephalopathy, disease.

Common causes

-paracetamol overdose

-viral hepatitis.

CHRONIC LIVER DISEASE

The chronic liver disease involves a disease process of progressive destruction and regeneration of the liver parenchyma leading to fibrosis and cirrhosis.

Common causes include viral hepatitis B and C, and toxins such as alcohol.

The common complications of chronic liver disease such as ascites, hyper-splensism and a collateral venous circulation producing lower oesophageal and gastric varices, all result from portal hypertension.

PHYSIOLOGICAL CHANGES ASSOCIATED WITH LIVER DISEASE

1. **Renal:** renal Impairment is most commonly due to dehydration, sepsis, or nephrotoxic drugs.

2. **Hepatorenal syndrome:** the reduced GFR and consequent decline in renal function caused by advanced liver disease.

3. **Hepatopulmonary syndrome:** This occurs when intrapulmonary vascular dilations

contribute to hypoxia in liver disease.

4. **Encephalopathy:** toxic metabolites build up (particularly ammonia), leading to progressive encephalopathy.

5. **Hematological changes:** Anemia may be present secondary to blood loss, hemolysis, bone marrow depression, or nutritional deficiency.

ANAESTHESIA IN PATIENTS WITH LIVER FAILURE

Pre-operative assessment

History
Examination
Investigation

1) **Full blood count:** to establish **anemia, thrombocytopenia,** or evidence of **infection**.

2) **Coagulation profile: bleeding time, clotting time, prothrombin time (PT).**

3) **Prothrombin time (PT):** as an indicator of hepatocellular function

4) **Renal function and electrolyte.**

5) **ECG and Echocardiography.**

6) **Chest x-rays and pulmonary function tests.**

7) **Liver function tests and Hepatitis screening.**

OPTIMIZING THE MEDICAL PROBLEM

- If PT > 1.5 times control – FFP
- Platelet < 50000 – platelet transfusion
- Fibrinogen < 1.0 g/l – cryoprecipitate
- Recombinant factor VIII – prophylaxis 7 therapeutic
- Correct electrolytes
- Hyponatremia; fluid restriction
- Anaemia – blood transfusion, aim for 10g/dl
- Sedative premed – avoided
  - H2 receptor antagonist : ranitidine
- **Ascites**
  - Paracentesis – massive ascites compromising the ventilation
- Spirinolactone (Aldactone)
- Dietary sodium restrictions

- **Encephalopathy** -Reduce nitrogen load
  - Lactulose
  - Neomycin
  - Protein restriction

PREMEDICATION

Depressed patients may not need preanesthetic premedication.

Avoid premedication in severely debilitated patients.

Prolonged drug effect - prolonged recovery.

Agents of choice: opioids +/- anticholinergics.

OPIOIDS

Morphine – delayed elimination d/t reduced hepatic blood flow & extraction ratio May precipitate hepatic. encephalopathy in pt – decompensated liver failure

Fentanyl – low dose (no active metabolites & renally excreted If repeated / large dose – will accumulate

Alfentanil – elimination reduce

Remifentanil – ideally suit intraoperatively

Metabolize by tissue & plasma esterase

Opioids reversible with opioid antagonists

INDUCTION

Preoxygenation of **2-3 minutes** prior to induction to prevent hypoxemia.

Inhalational induction preferable

(note that 99% of isoflurane is eliminated from respiration and not the liver).

Desflurane has the advantage of being the least metabolized and providing the quickest emergence.

Intravenous induction

Induction agent - titration of dose to avoid hypotension more important than the particular drug

benzodiazepines Increased sensitivity

Thiopental- dose should be reduce d/t reduce plasma proteins – increase unbound protein & prolong duration of action

Propofol – is the most commonly used (extra-hepatic metabolism but increases sensitivity to sedative & cardiorespiratory depressant so the dose should be reduced.

Etomidate – safely but little advantage over thiopental.

suxamethonium may have a prolonged duration of action due to reduced pseudocholinesterase concentrations slowing its metabolism

Vecuronium and rocuronium have a prolonged elimination phase in severe disease.

Atracurium and cisatracurium are better options as they are not reliant on hepatic excretion.

POST OPERATIVE PAIN RELIEF CAN BE CHALLENGING.

The role of regional techniques is very much restricted by the high incidence of coagulation defects. Placement of epidural catheters should be undertaken with caution and with careful consideration to timing of catheter removal.

Similarly, intra-muscular and subcutaneous injections can lead to haematoma formation and should be avoided.

Non-steroidal anti-inflammatory medications and their association with increased risk of GI bleeding, platelet dysfunction and nephrotoxicity mean they are often avoided.

DRUGS ASSOCIATED WITH LIVER DYSFUNCTION

- Valproate, amitriptyline, carbamazepine
- Tramadol - said to be safe with mild hepatic impairment
- Local anaesthetics – amides will have delayedelimination and accumulate
- Paracetamol if severe liver failure – avoid

Any questions?