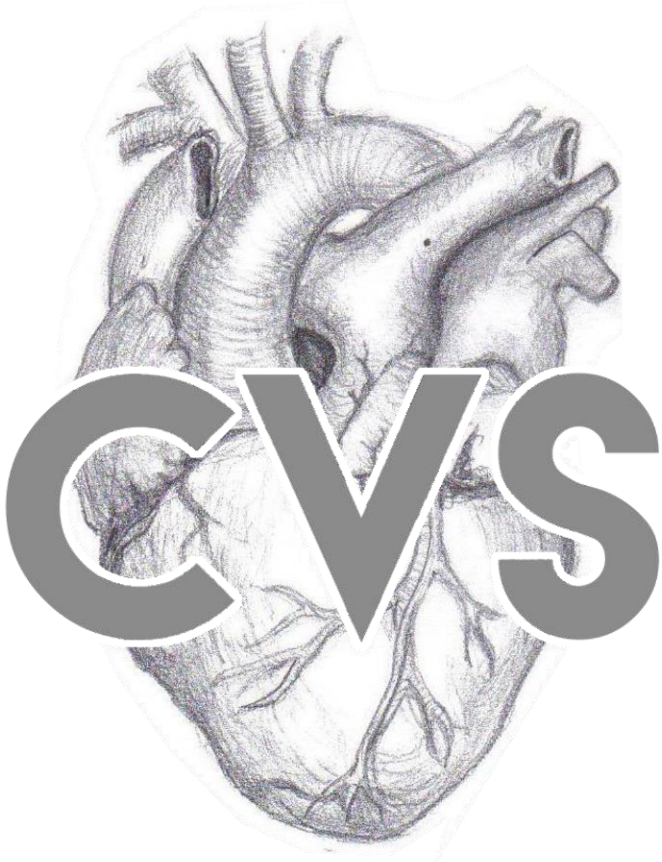

CVS

ANATOMY / HISTOLOGY

☑ Sheet ○ Slide ○ Handout

Number

9

Subject

Heart Failure & Portal Circulation

Done By

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Corrected by

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Doctor

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Date: 2/11/2016

Price:

بسم الله الرحمن الرحيم

Heart Failure Types

☆ Left heart failure:

*Remember: Left half of the heart receives blood from right half!

Left ventricle unable to pump the total volume of blood it receives from the right side (less ejection fraction!)

- Causes of left ventricle failure:
  1. MI fibrous tissue ⇨ weak ventricle
  2. systemic hypertension
  3. aortic valve stenosis or “insufficiency” widened

- Heart fibers hypertrophy without blood supply in pathological states
- While in athletes hearts hypertrophies with increased blood supply (forming of vessels and capillaries too!)

- Backward effect:
  - ⇨ Decreased emptying of left ventricle “increase EDV instead of 130 becomes 140-150”
  - ⇨ blood collects in left atrium
  - ⇨ then to pulmonary veins
  - ⇨ then back to pulmonary circulation
  - ⇨ causes congestion of lungs with blood
  - ⇨ increase pressure inside lungs capillary beds
  - ⇨ which causes pulmonary edema due to increased filtration,
  - ⇨ then pulmonary hypertension due vasoconstriction by defensive mechanism of the lung ,
  - ⇨ **Right ventricle** faces higher pressure!

- Forward effect:
  - ⇨ less CO,
  - ⇨ mean arterial pressure becomes lower, "mean arterial pressure and resistance related to CO"

$$MAP = CO \times TPR$$

  - ⇨ less blood to tissues (hypoxia)
  - ⇨ kidney ischemic -> Renin-Angiotensin-Aldosterone system gets activated
  - ⇨ Angiotensin II (powerful vasoconstrictor) -> vasoconstriction,
  - ⇨ Aldosterone -> water sodium retention-> increase ECF -> blood volume increased -> increased pressure!
  - ⇨ This process will fail eventually (Temporary compensatory effect) by increasing the volume yet the ventricle is still failing "**aldosterone** also **<u>decrease K</u>** conc."

- Symptoms of left side failure:

- fatigue "just like anemia",
- muscle weakness "due to low potassium by aldosterone",
- dizziness "because low amounts of blood to the brain"

*Patient with <u>acute heart failure</u> and infarction the blood congest in the lungs causes **Dyspnea** "defective gas exchange" no oxygenation!

☆ Right side failure:

Right ventricle unable to pump total volume of blood received from veins (venous return)

*Normally **CO** should be equal to **venous return**

- Causes of right ventricle failure
  1. <u>Main cause right vent failure is left vent failure!</u> *"transforms From left side to right side"*

  Other causes:
  2. COPD,
  3. Congenital heart defect, especially those that involve more blood to the lungs and pulmonary hypertension

- Backward effect:

  - ⇨ Blood goes back to right atrium
  - ⇨ Then to systemic veins!
  - ⇨ Hepatomegaly and splenomegaly!
  - ⇨ collection in veins then capillaries causes increased pressure
  - ⇨ Transudation and **Edema** in the limbs "systemic veins" (called **dependent edema** because it depends on the gravity)!
  - ⇨ If patient is lying edema happens in the lower back

- Forward effect:
  - ⇨ right ventricle is unable to pump blood properly,
  - ⇨ less blood coming from the lungs to left atrium
  - ⇨ less blood to the left ventricle which leads to **less CO**
  - ⇨ Same forward effect of left side failure mentioned earlier happens!

Note: Patient with left side failure complains from Dyspnea "orthopnea", after a while it transforms to right side failure! Patient will be relieved from the dyspnea and lung edema but he will have lower limb edema!!

"He will think he is healed but he isn't!!" pay attention to this! Change in symptoms

Congestion in neck veins:

- ❖ Because blood is back to SVC from right atrium, then to brachiocephalic ⇨ internal jugular ⇨ **External jugular** which will be visible when congested, "due to increased central venous pressure"

External jugular vein will be visible although it's covered by superficial fascia and platysma!

Other causes of external jugular congestion is **tricuspid stenosis** and **pericardial effusion** (3 causes)

*pericardial effusion: fluid around the heart inside the pericardium will put pressure on the heart, right ventricle will start to fail and blood accumulate in rt. Atrium and so on

*Important:

If patient had:

Bleeding -> low central venous pressure

Right side failure -> high central venous pressure!

Portal Circulation

**Begins with capillaries and ends in capillaries !*

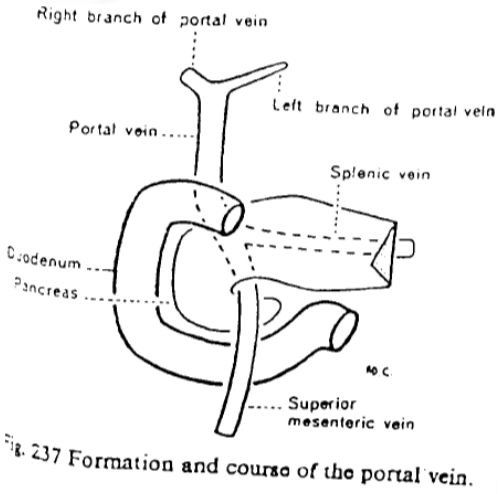


Fig. 237 Formation and course of the portal vein.

Hepatic Portal Vein begins in capillaries of the intestine and ends in sinusoids capillaries in the liver! "Begins as a vein then ends as an artery" the doctor said it like this!

"Portal circulation also occurs in the pituitary gland and the kidney "

☆ Course:

Portal vein is formed **posterior to the neck of the pancreases** by union of:

1. **Splenic Vein**
2. **Superior Mesenteric Vein**

So Portal vein receives blood from: the Stomach + small bowel + large bowel + spleen + pancreas + gallbladder

Splenic vein drains from the spleen

Superior mesenteric drains from **Midgut**: (lower half of duodenum + jejunum + ileum + ascending colon + part of transverse colon)

Inferior mesenteric drains blood from **Hind gut:** (descending colon + sigmoid) and opens in **splenic vein!**

- It ascends behind the 1st part of the duodenum to enter the free margin of lesser omentum.
- It enters the Porta hepatis where it divide into right and left branches, it is devoid of valves.

- Porta hepatis: portal vein enters with hepatic artery and common hepatic duct goes out! (common hepatic duct with cystic duct makes **bile duct**)
- 1.5 liter per min enters the liver 80% portal 20% hepatic artery, so mixing of arterial blood and portal blood occurs in the liver!
- Then blood comes back to IVC through three hepatic veins

Relations with duodenum:

A. Infraduodenal part

1. Anteriorly: neck of pancreas.

2. Posteriorly: inferior vena cava.

B. Retroduodenal part

1. Anteriorly: (a) first part of duodenum (b) common bile duct; and (c) gastroduodenal artery.

2. Posteriorly: inferior vena cava. ...

C. **Supraduodenal part**, in the free margin of lesser omentum

1. Anteriorly: (a) hepatic artery; and (b) bile duct.

2. Posteriorly; inferior vena cava. Through epiploic foramen

3. *Surrounded by: (a) hepatic plexus of nerves; and (b) lymph nodes and lymphatics*

**Infraduodenal part of portal vein can be connected with IVC which lies behind it to treat portal hypertension*

Cirrhosis causes portal vein block ⇨ portal hypertension

Liver's main function is **detoxifying** the blood, if connection between IVC and the portal vein happened blood will go from the gut directly to the IVC which is dangerous especially for the brain!

Branches of Portal Vein:

1. The right branch is shorter and wider.
After receiving the cystic vein(ان وجد), it enters the right lobe of liver.

2. The left branch is longer and narrower.
It traverses the whole porta hepatis from right to the left end, and furnishes branches to the **caudate and quadrate** lobes. Just before entering the left lobe of liver, it receives:

(a) **Para-umbilical veins**; (b) ligamentum teres; and (c) ligamentum venosum

Caudate and quadrate lobes are considered anatomically from the right lobe but functionally they are from the left lobe that's why they receive blood from the left branch and their bile is going to the left hepatic duct

Note: the rule of 2s in the portal vein

The portal vein is **2** inches long, formed by **2** veins (splenic and superior mesenteric),
ends by **2** branches (to right and left lobes of the liver),
receives **2** main tributaries (right and left gastric veins)
and **2** ligaments are attached to its left branch (ligamentum teres and ligamentum venosum).

Around ligamentum teres there is a vein called **para-umbilical vein** which drains blood from the abdominal wall to the left branch of portal vein

"Ligamentum teres was the umbilical vein and ligamentum venosum was ductus venosus in the embrvo"

Tributaries to the portal vein:

These are:

(i) the splenic

(ii) superior mesenteric

(iii) left gastric

(iv) right gastric

(v) superior pancreaticoduodenal

(vi) cystic

(vii) para-umbilical veins

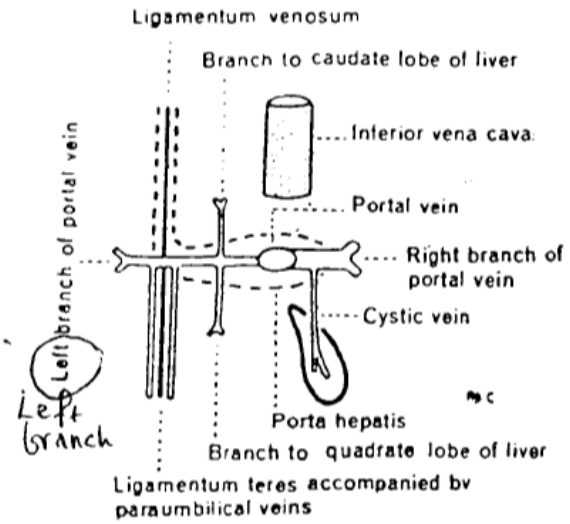


Fig. 238 Branches of the portal vein; their sub-branches and tributaries.

☆ Portosystemic anastomoses:

*Cirrhosis ⇨ portal hypertension,

In this case the portal veins tries to find other ways to drain to,

- **Causes of liver cirrhosis:**

1. Alcoholism
2. Schistosomiasis – also known as bilharzia

Sites of Anastomoses:

1. AT THE **LOWER END OF ESOPHAGUS** between:

a) esophageal tributaries of the left gastric vein (portal).

b) esophageal tributaries of the azygos vein (systemic"SVC").

In portal hypertension this anastomosis enlarges to form **Esophageal Varices** (dilated and tortuous veins in the sub-mucosa of lower end of esophagus). Its rupture leads to bleeding in the stomach **(haematemesis).**

If a patient came to the ER with a symptom of vomiting blood, don't think about bleeding from peptic ulcer , because rarely it will cause vomiting blood and most of the cases blood will go down the intestines and comes with the stool as dark stool!

You should think about bleeding from ruptured esophageal varices

2. AT THE **LOWER END OF RECTUM** between:

a) Superior rectal vein (portal). "To Inferior mesenteric"

b) Middle and inferior rectal veins (systemic). "To Internal iliac"

Varices means portal hypertension!!

If this anastomosis enlarges, the veins in the sub-mucosa of the rectum become dilated and tortuous leading to formation of piles **(hemorrhoids).**

Note: hemorrhoids doesn't always mean portal Hypertension!!

Not all hemorrhoids are portal hypertension

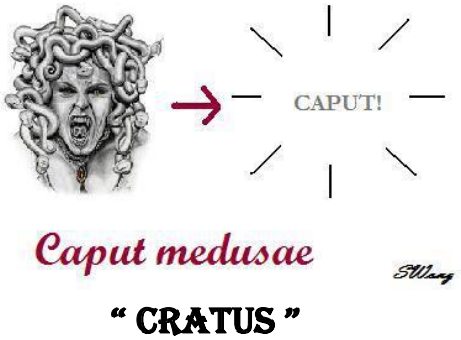
Hemorrhoids can occur without hypertension and persists as painless bleeding at the lower end of anal canal during defecation "blood with the stool" , it becomes painful if it goes out of the anus and thrombosis occurs in it!

3. AT THE **SKIN AROUND UMBILICUS** between

a) Para-umbilical veins (portal).

b) Veins of anterior abdominal wall (systemic).

Enlargement of this anastomosis leads to dilatation of the skin veins in a radial direction around the umbilicus, a condition called **(caput medusa).**



4. LESS EFFECTIVE SITES OF ANASTOMOSIS :

I. At the **bare area** of liver between :

a) Capillaries within the liver (portal).

b) Phrenic tributaries on the under surface of diaphragm (systemic).

II. On **posterior abdominal wall** between:

a) Lumbar veins (systemic).

b) Colic veins (portal).

*One of the ways to treat bleeding esophageal Varices is to connect the portal vein with the IVC (porosystemic anastomosis)

This way we reduce the bleeding of the varices, but now the toxins absorbed from the intestines will go directly to the systemic circulation without getting detoxified in the liver which is dangerous for the brain! Which may cause loss of concentration or dementia etc..

The Lecture Ends Here…

Revision of Last anatomy Lab:

Feeling the pulse:

In the hand:

Brachial artery can be felt Medial to tendon of biceps brachii

Radial can be felt on the distal end of radius lateral to flexor carpi radialis longus tendon

"between the tendons of the brachioradialis and flexor carpi radialis"

If there is pulse then there is blood flowing, no pulse no blood flow!

In the leg:

Inferior to mid iguinal point ⇨ femoral artery

Popliteal artery in the popliteal fossa

posterior tibial artery ⇨ behind medial malleolus

anterior tibial can't be felt but we can feel it's continuation the **dorsalis pedis** artery which lies lateral to extensor halluces longus,

if pulse is not felt doesn't always mean ischemic foot!!

We should check the posterior tibial too, if there is no pulse there then it's mostly ischemic!

Gradual obstruction of lower limb arteries by Atherosclerosis,

Risk factors: diabetic, smoker, obese, hypertension, sedentary lifestyle

Smoking is the Major risk factor

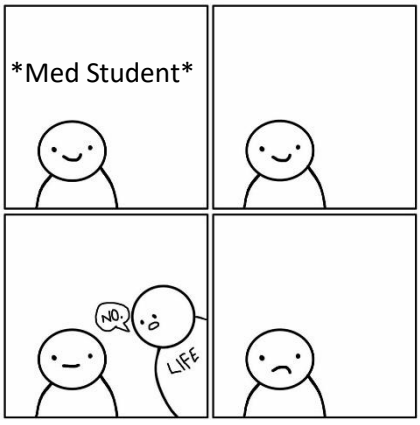
Sudden(acute) obstruction caused by thrombus on top of atherosclerosis!

If acute obstruction no pulse felt! Patient will suffer from **parasthesia** , pallor, polar (cold) "three P's"

Paresthesia is due to no blood to the nerve!

When patient walks he'll feel pain in the calf area, this Pain is due to accumulation of metabolites (mainly lactic acid) this is called **intermittent claudication** similar to angina in the heart

Le Fin.



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