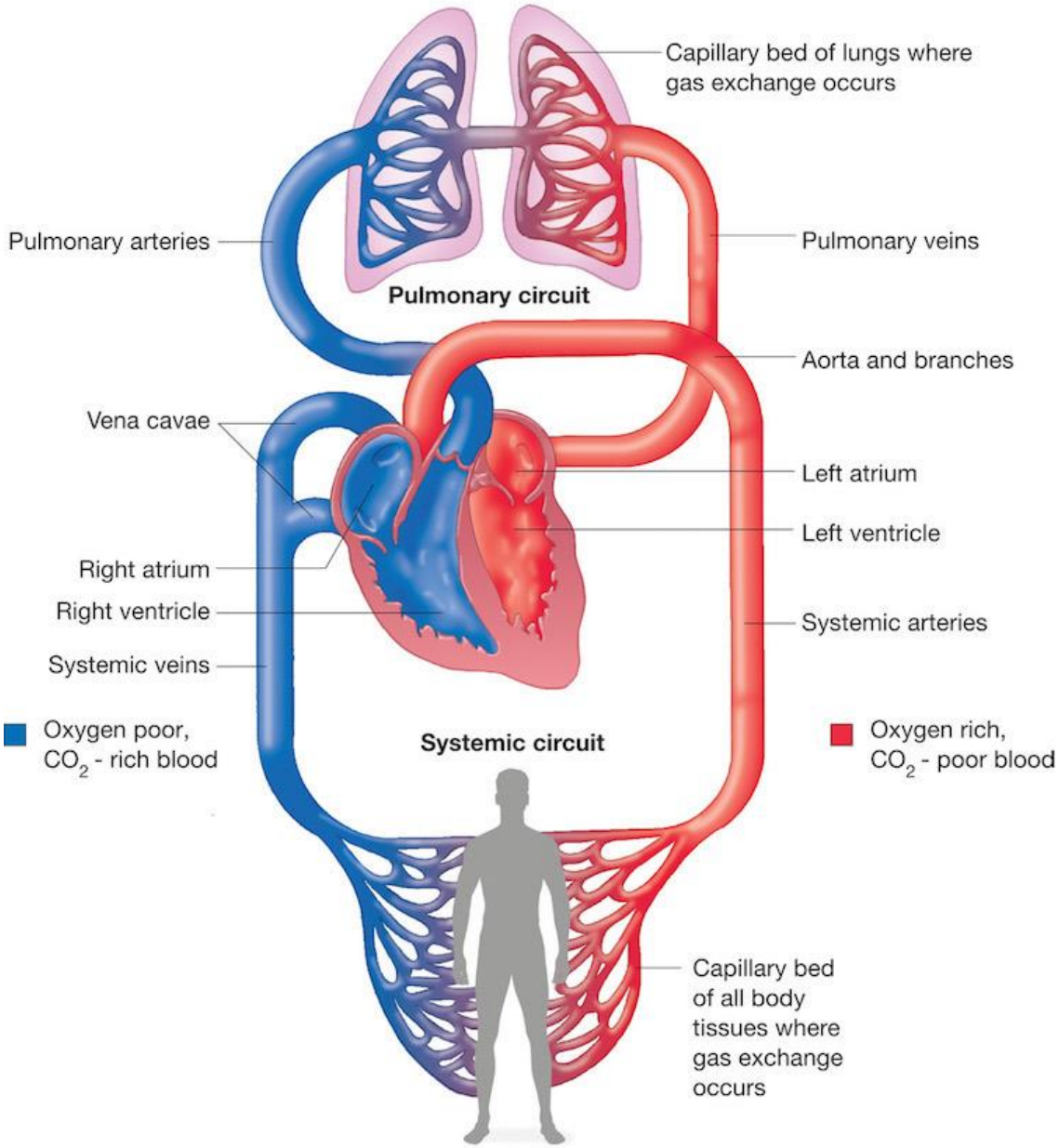
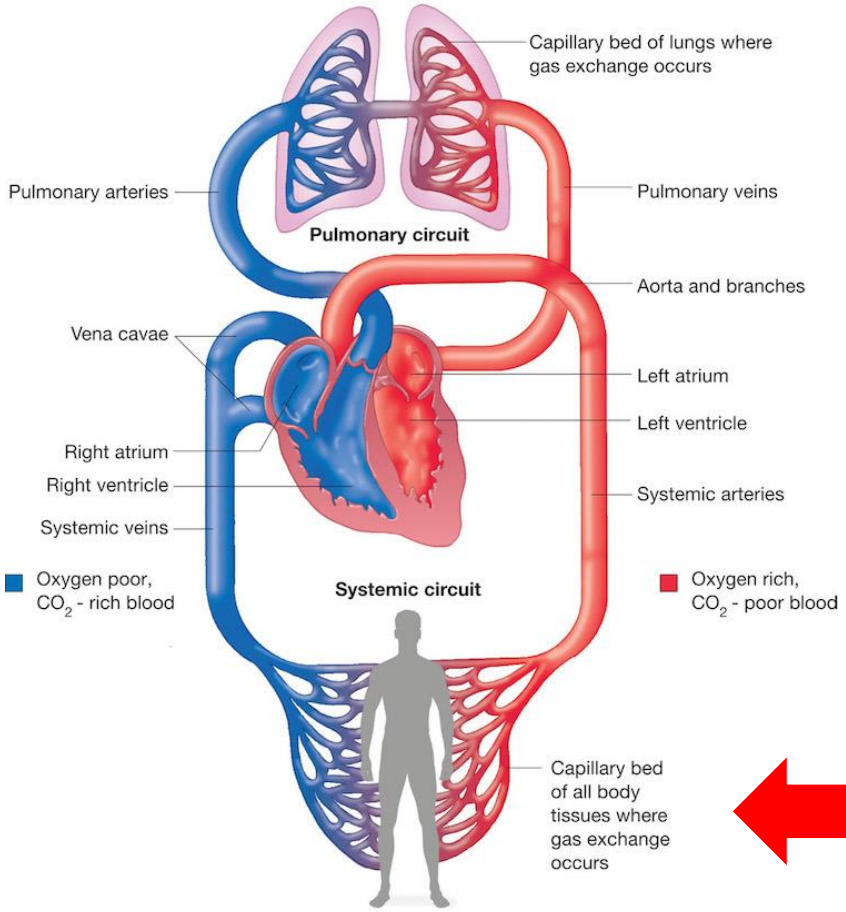
ACTIVE & PASSIVE HYPEREMIAS

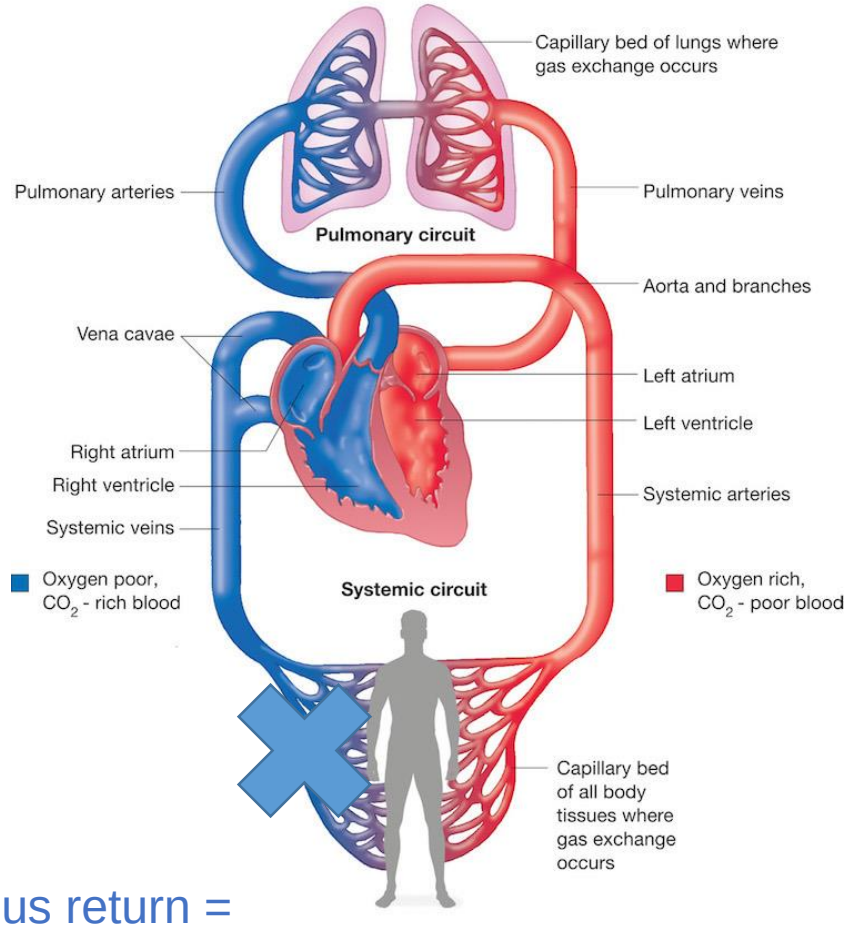
Árpád Szállási

Hyperemia: „excess of blood in the vessels supplying an organ or part of the body”


Capillary bed of lungs where gas exchange occurs
Pulmonary arteries
Pulmonary veins
Pulmonary circuit
Aorta and branches
Vena cavae
Left atrium
Left ventricle
Right atrium
Right ventricle
Systemic arteries
Systemic veins
Oxygen poor, CO_2 - rich blood
Systemic circuit
Oxygen rich, CO_2 - poor blood
Capillary bed of all body tissues where gas exchange occurs



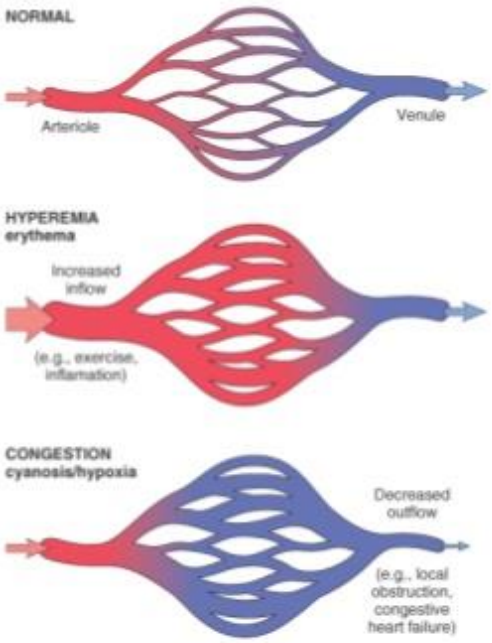
Increased arterial blood flow = active/functional hyperemia



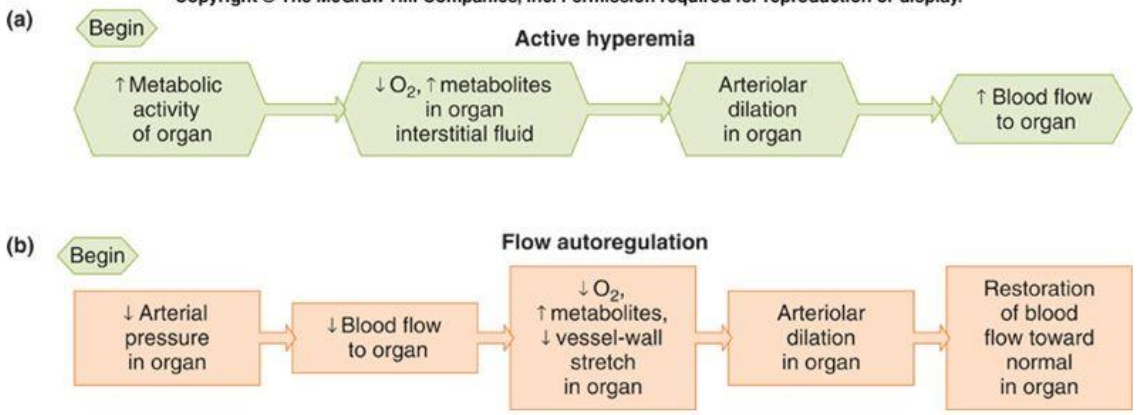
Blocked venous return = passive hyperemia

Hyperemia

- A local increased volume of blood in a particular tissue
  - Dilatation of small vessels
- Two types
  - Arterial (active) hyperemia-hyperemia
    - from augmented blood flow due to arteriolar dilation
  - Venous hyperemia-congestion, cyanosis
    - a *passive process* resulting from impaired venous return out of a tissue
    - closely related to the development of edema
      - so that congestion and edema commonly occur together



Local control of organ blood flow



Active hyperemia and flow autoregulation differ in their cause but both result in the production of the same local signals that provoke vasodilation.

Hyperemia

Active

- loss of sympathetic activity
- vasoactive compounds

↓

vasodilatation

-inflammations (rubor, calor) – (NO, PGE2)
-excercise
-eating
-psychological effects (blushing)
-hormones (postmenopausal)
-carcinoid flush
-reperfusion
-medicinal drugs (Na-nitrite, blue tablet)

Passive (congestion)

- venous block

a.) local

venous thrombus
venous compression
torsion
invagination
vena cava superior syndr.

b.) systemic

right sided heart failure
chronic cor pulmonale

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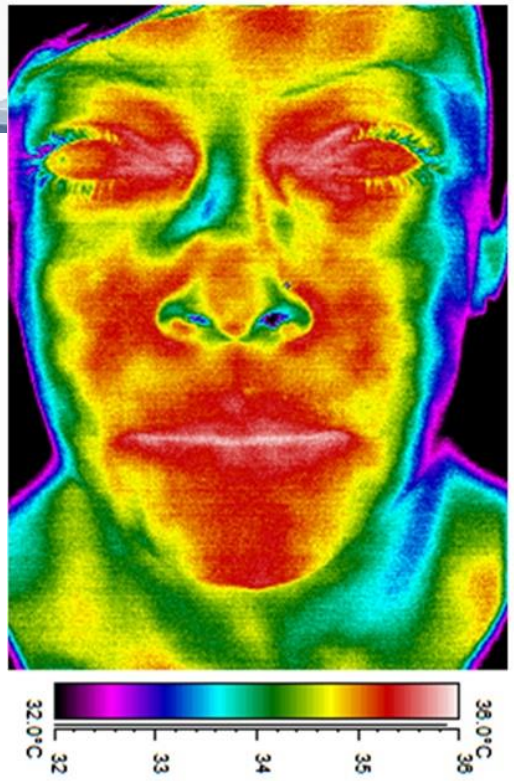
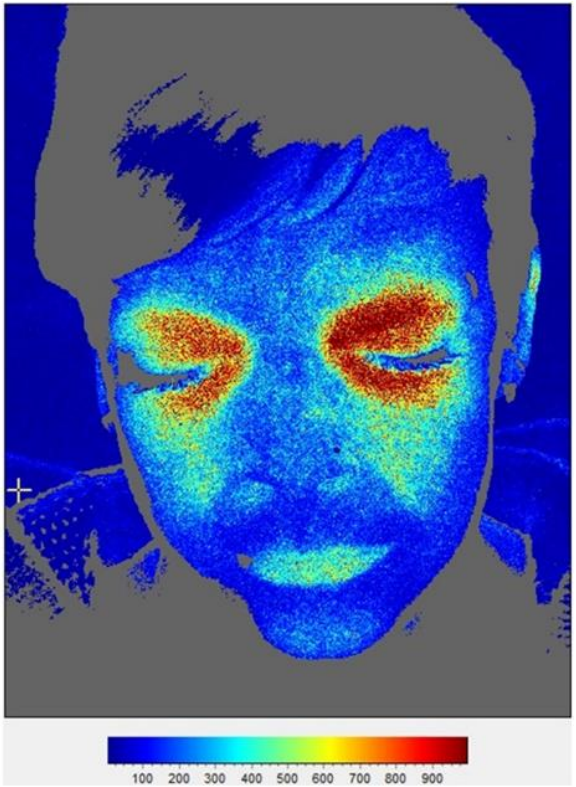
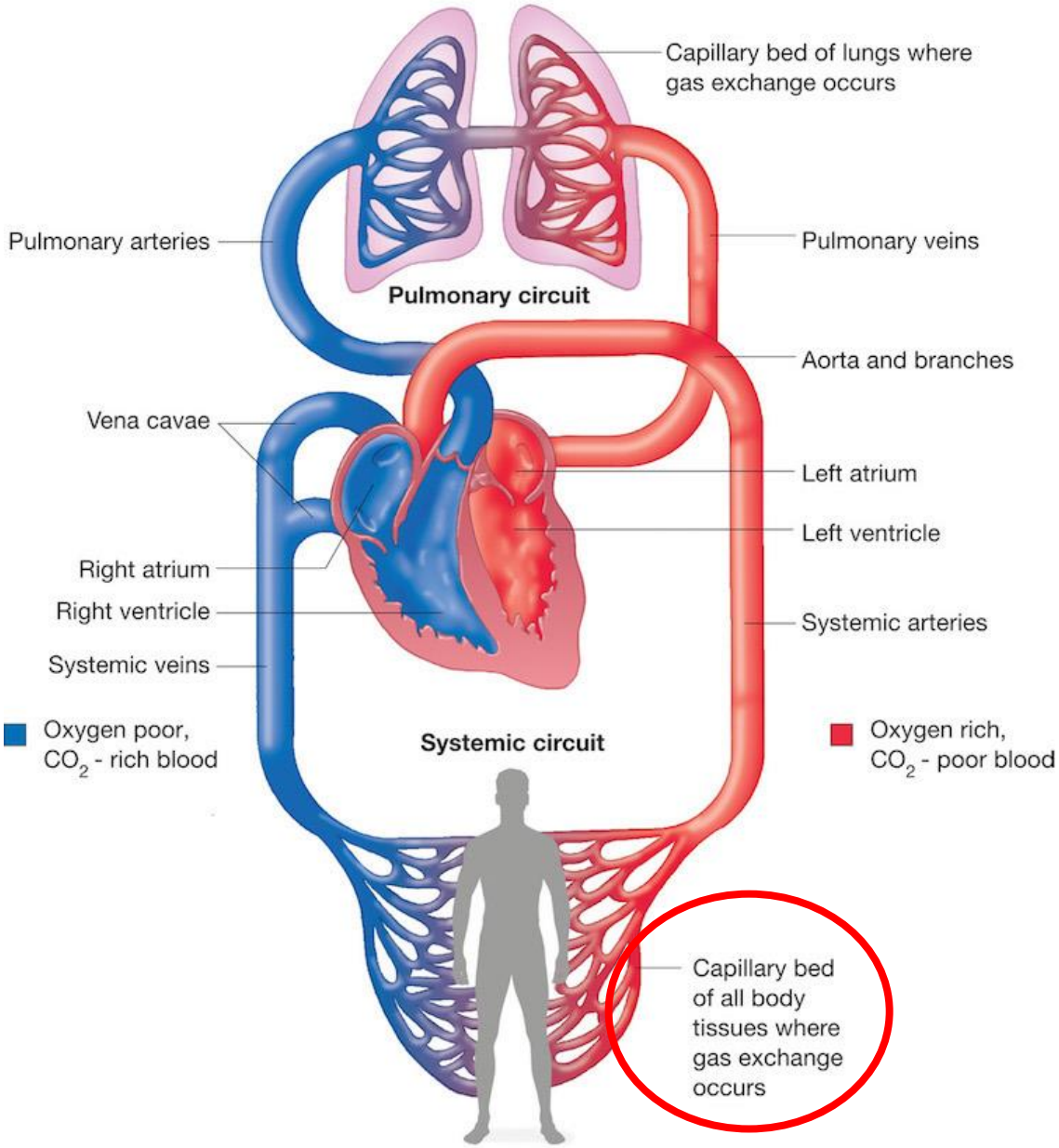

32.0°C
32
33
34
35
36
38.0°C


100 200 300 400 500 600 700 800 900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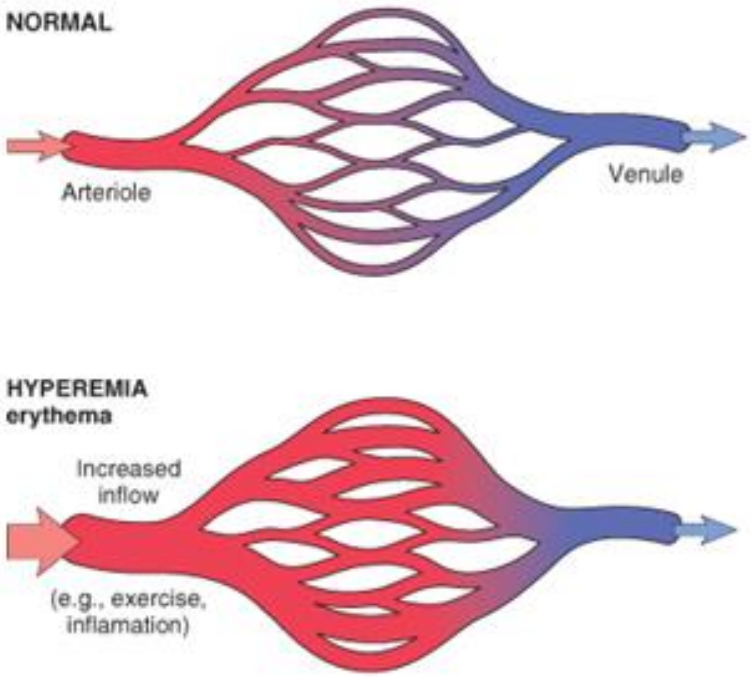
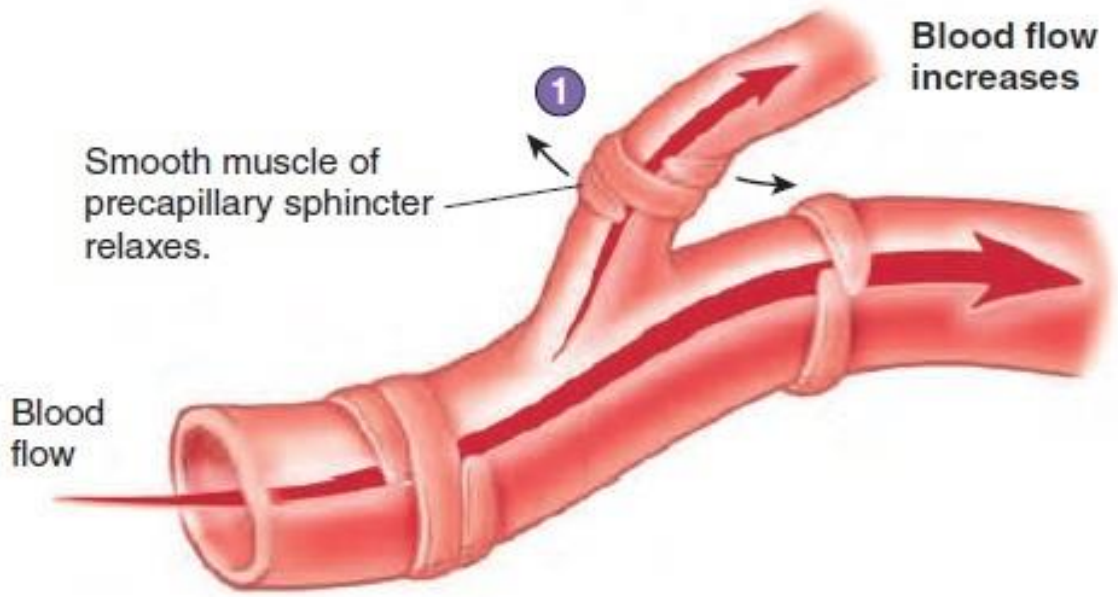
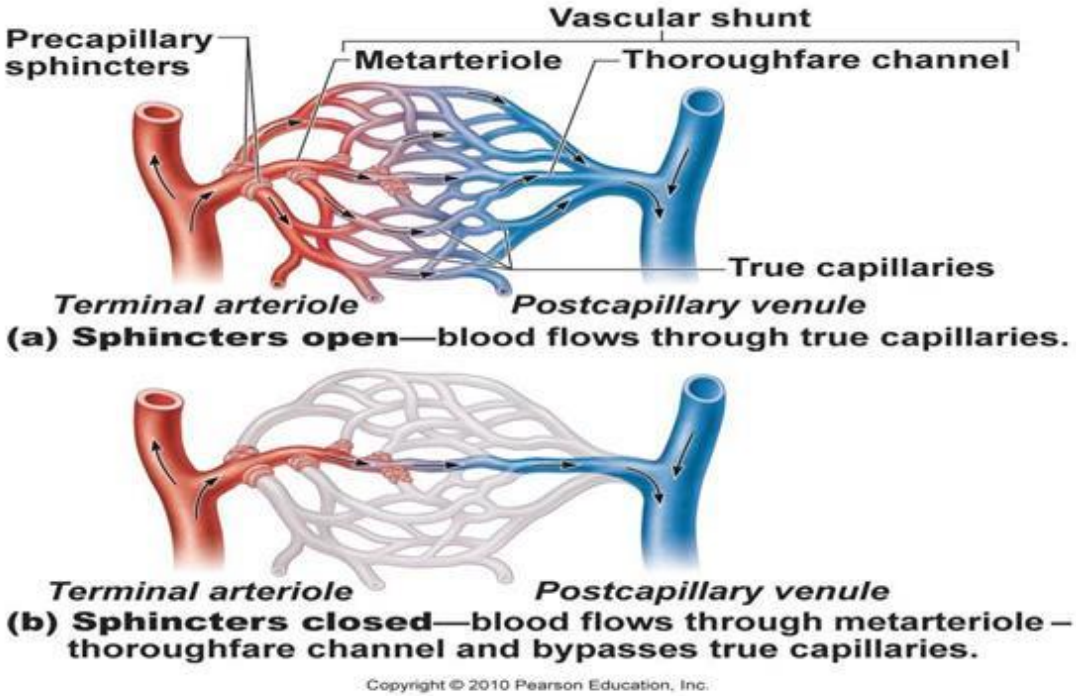
HYPEREMIA

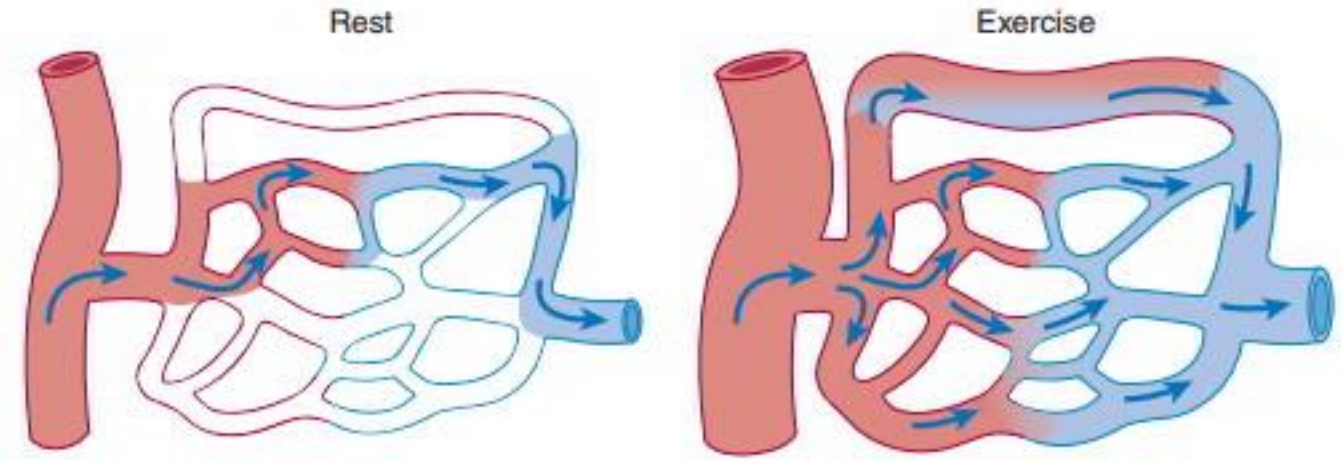

NORMAL
Arteriole
Venule
HYPEREMIA
erythema
Increased
inflow
(e.g., exercise,
inflamation)



1 Relaxation of precapillary sphincters.
Precapillary sphincters relax as the tissue concentration of O_2 and nutrients, such as glucose, amino acids, and fatty acids, decreases. The precapillary sphincters also relax as CO_2 concentration increases and pH decreases.

Precapillary sphincters can shunt blood past a capillary bed




Rest
Exercise

Hyperemia

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-excercise

-eating

-psychological effects (blushing)

-hormones (postmenopausal)

-carcinoid flush

-reperfusion

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Passive (congestion)

- venous block

a.) local

venous thrombus

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right sided heart failure

chronic cor pulmonale

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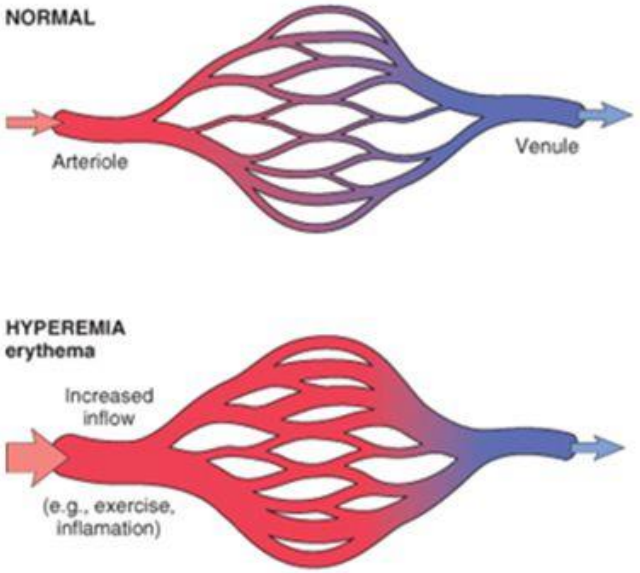

http://2.bp.blogspot.com/-rdBezbztfrM/TWOQ8us2sVI/AAAAAAAACs0/_Q1kbP0ZRo4/s1600/hyperemia.png

http://upload.wikimedia.org/wikipedia/commons/thumb/5/58/Hyperemia_conjunctiva.jpg/1024px-Hyperemia_conjunctiva.jpg

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CARCINOID TUMORS

* SLOW GROWING
* SOME METASTASIZE
* COMMON PRIMARY SITES
  - SMALL/LARGE INTESTINES
  - STOMACH
  - PANCREAS
  - LIVER

CARCINOID SYNDROME

CARCINOID TUMOR

NEUROENDOCRINE CELLS

- ↑SEROTONIN
- ↑HISTAMINE
- ↑BRADYKININ

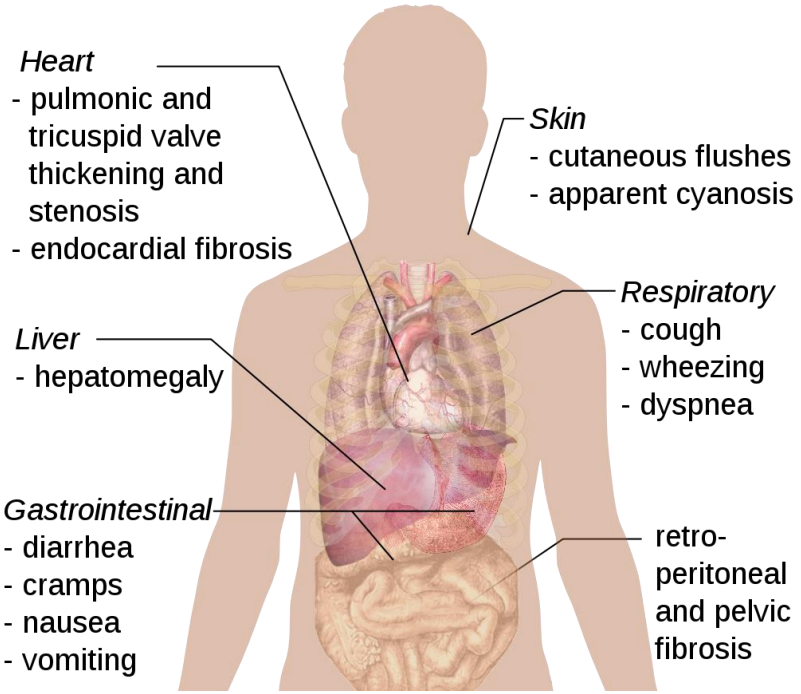
SET OF SYMPTOMS

- DIARRHEA
- SHORTNESS OF BREATH
- FLUSHING
- ITCHING

DIAGNOSIS

- OCTREOTIDE SCAN
- 5-HYDROXYINDOLEACETIC ACID (Urine)
- ↓NIACIN (Blood)

Carcinoid syndrome



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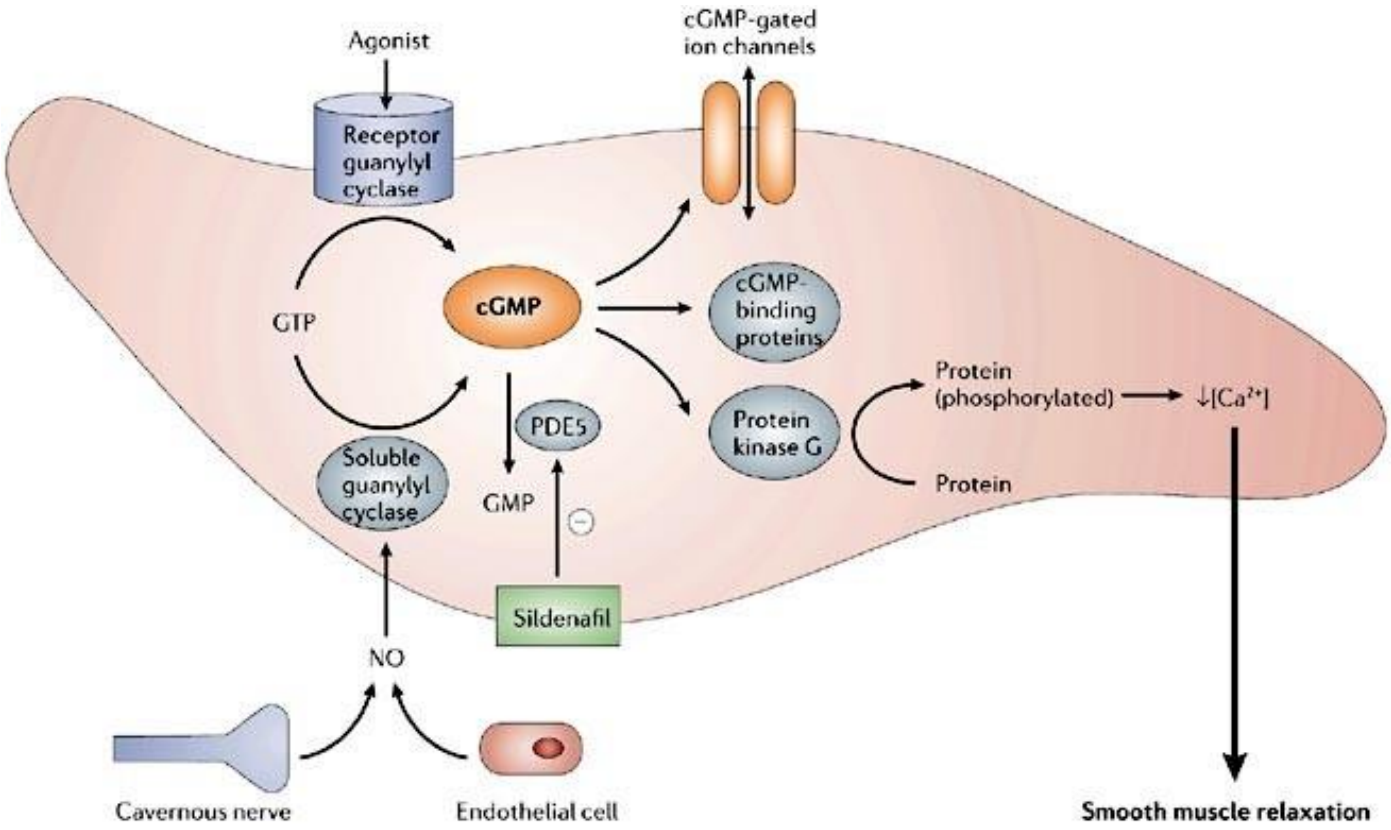
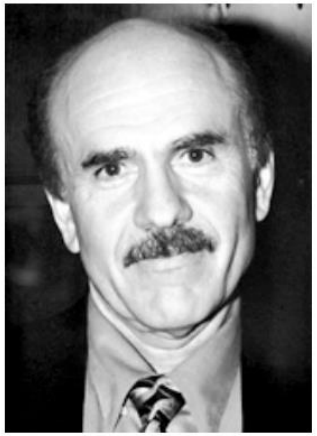
Pfizer


NDC 0069-4210-30
30 Tablets
Viagra™
50
(sildenafil citrate) tablets
50 mg*
Distributed by
Pfizer Labs
Division of Pfizer Inc, NY, NY 10017



The Nobel Prize in Physiology or Medicine 1998

Robert F. Furchgott
Prize share: 1/3



Louis J. Ignarro
Prize share: 1/3

Ferid Murad
Prize share: 1/3

The Nobel Prize in Physiology or Medicine 1998 was awarded jointly to Robert F. Furchgott, Louis J. Ignarro and Ferid Murad *"for their discoveries concerning nitric oxide as a signalling molecule in the cardiovascular system"*.

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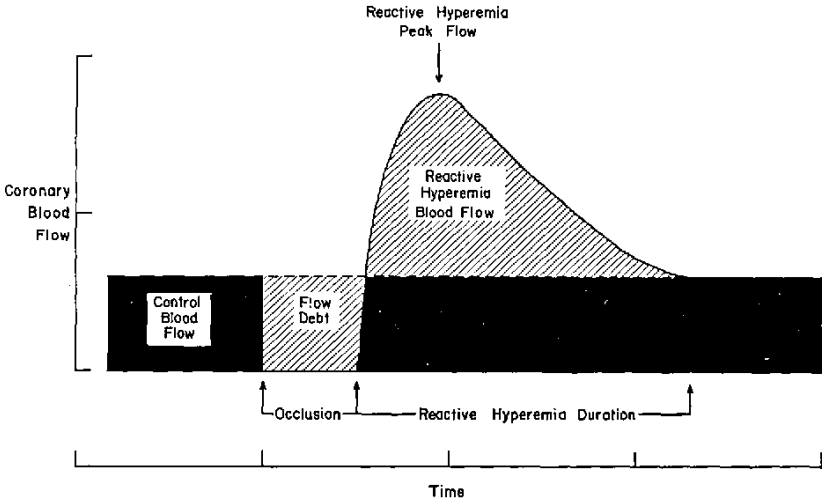
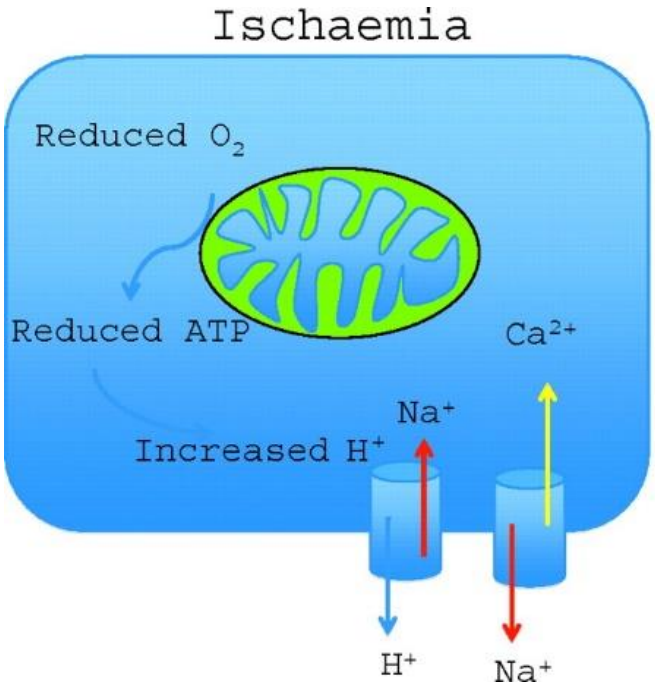
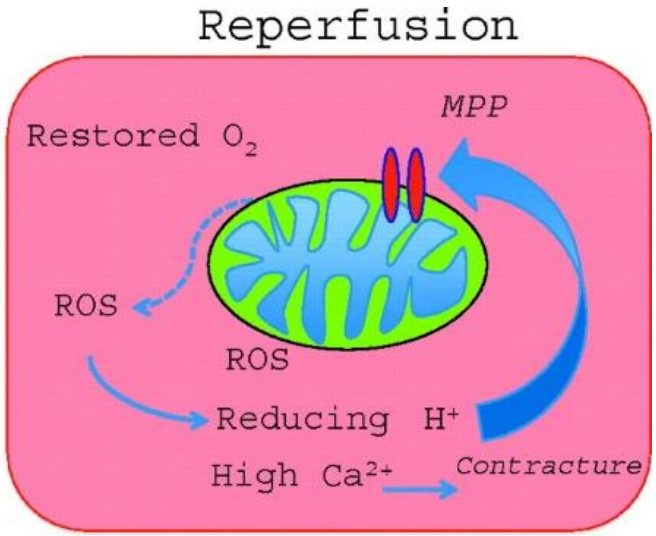
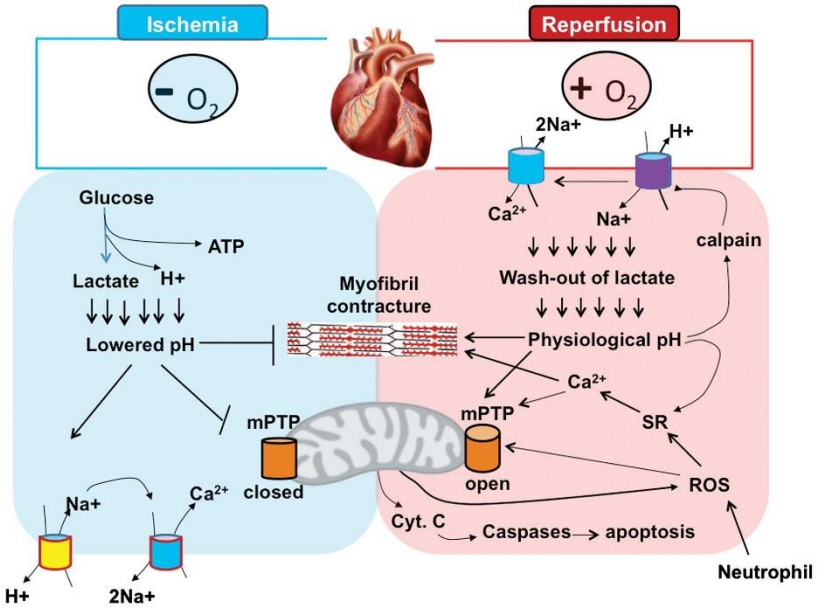

Reactive Hyperemia
Peak Flow
Coronary
Blood
Flow
Reactive
Hyperemia
Blood Flow
Control
Blood
Flow
Flow
Debt
Occlusion
Reactive Hyperemia Duration
Time


Ischaemia
Reduced O_2
Reduced ATP
Ca^{2+}
Na^+
Increased H^+
H^+
Na^+


Reperfusion
MPP
Restored O_2
ROS
ROS
Reducing H^+
High Ca^{2+}
Contracture

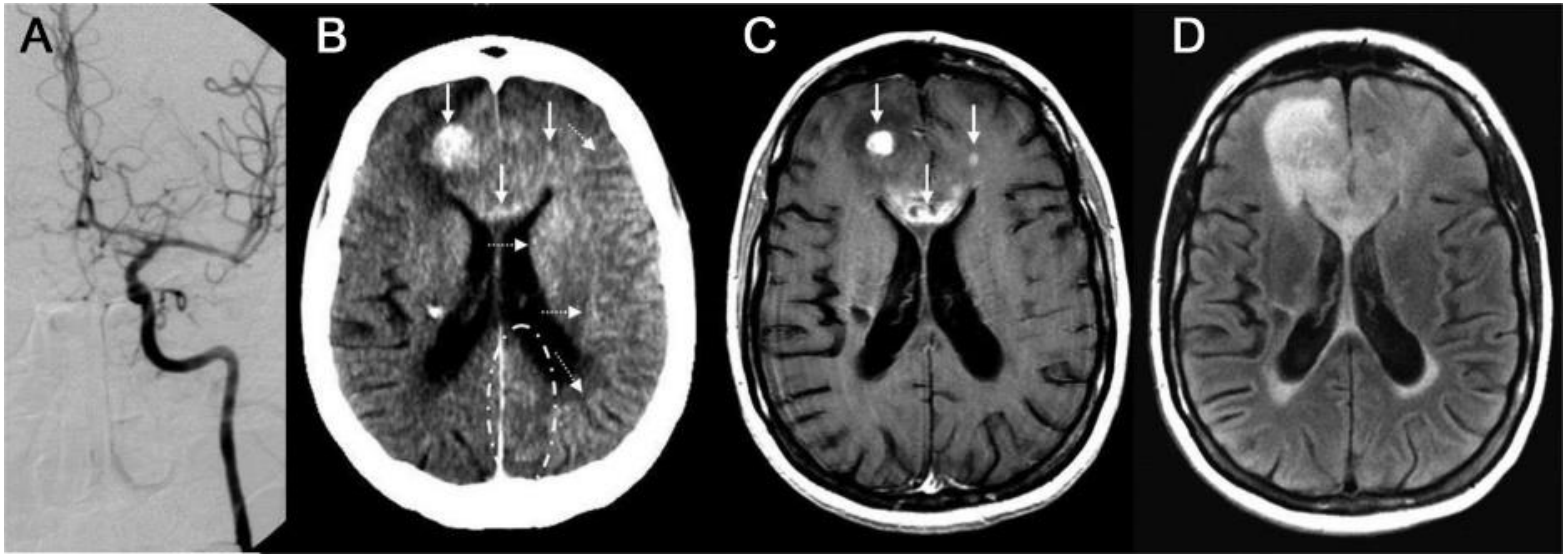


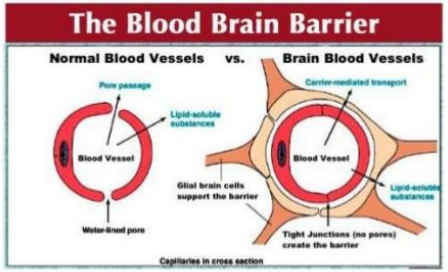
BOX 2

Reperfusion Injury[1,5–9,12]

- Oxygen is reintroduced into the affected area.
- XO forms superoxide radicals.
- Superoxide liberates free iron, causing creation of hydroxyl ROS.
- ROS damage macromolecules (i.e., DNA, RNA) and cause endothelial injury, microvascular dysfunction, and apoptosis.
- Neutrophils overwhelm the affected areas.
- The inflammatory process accelerates.
- Excessive cytokines are produced.


wiseGEEK


A
B
C
D



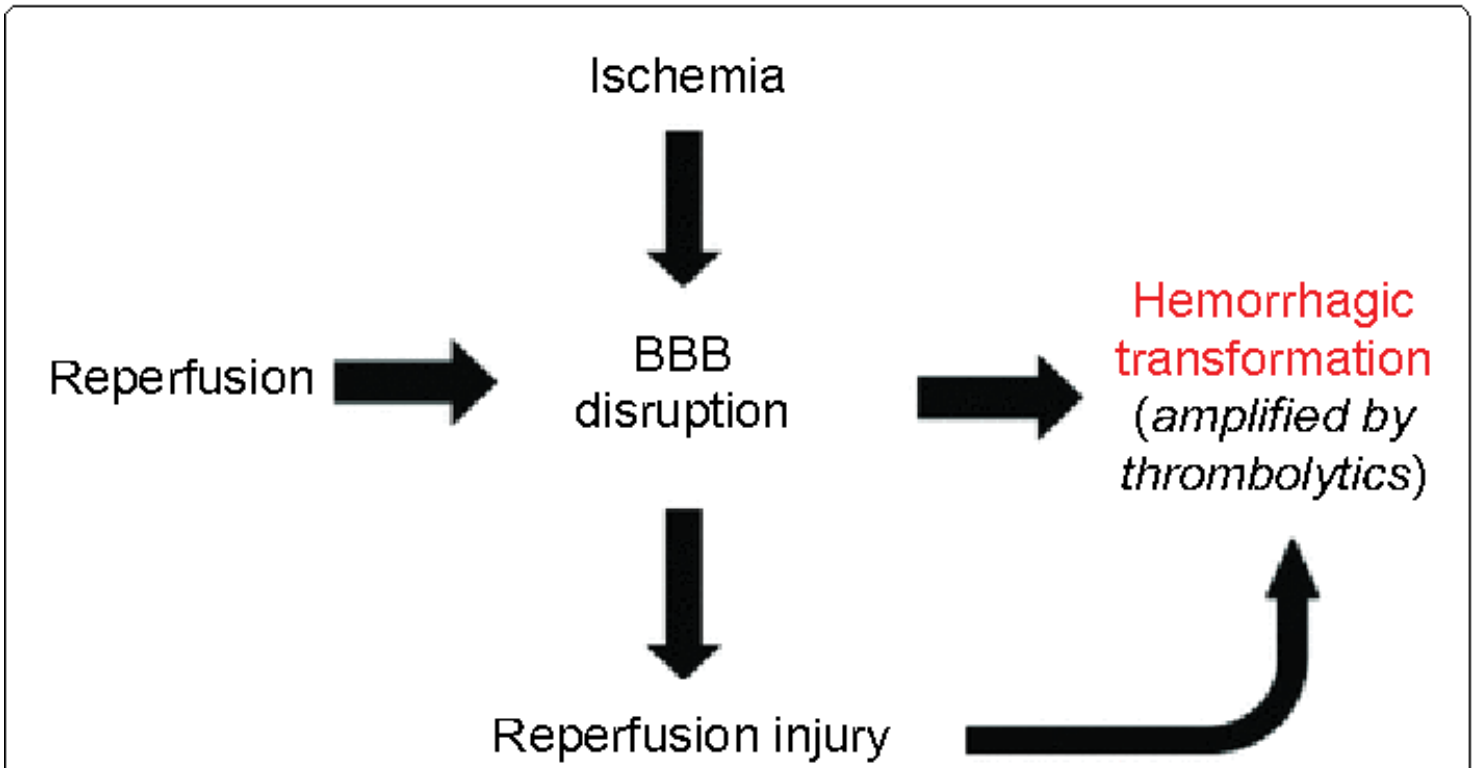
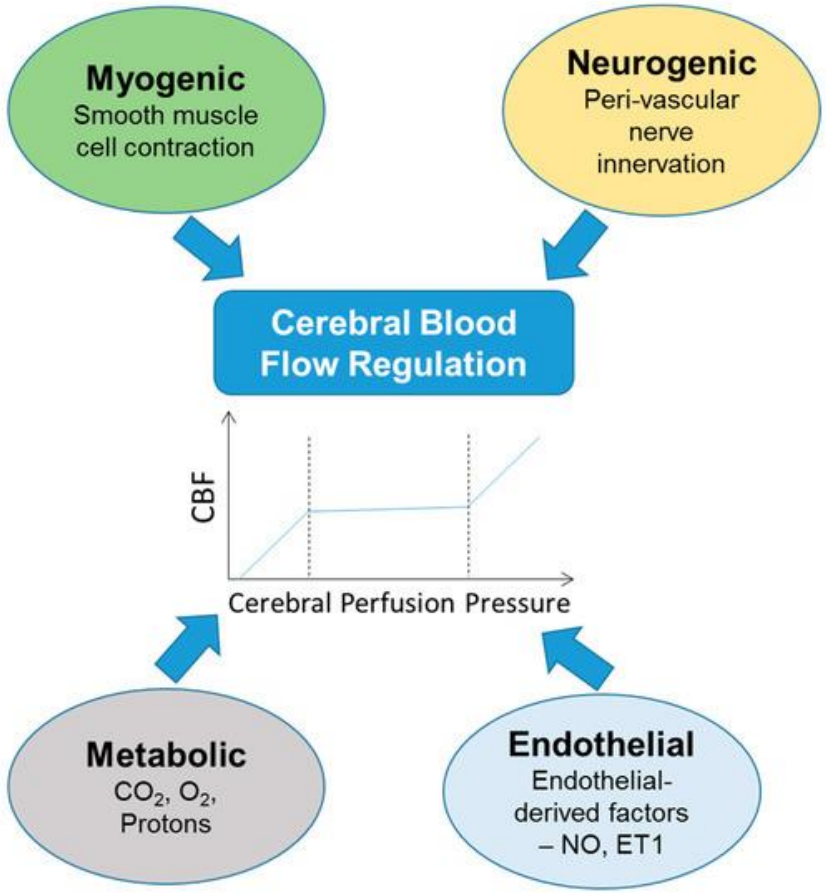


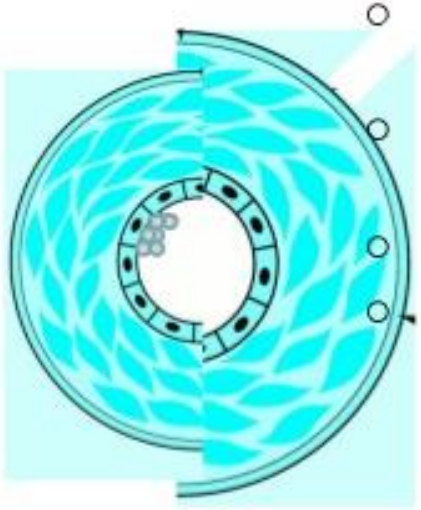
Figure 1: Schematic representation of blood–brain barrier (BBB) changes in


Myogenic
Smooth muscle cell contraction
Neurogenic
Peri-vascular nerve innervation
Cerebral Blood Flow Regulation
CBF
Cerebral Perfusion Pressure
Metabolic
CO_2, O_2, Protons
Endothelial
Endothelial-derived factors – NO, ET1

Local Control : Humoral

Vasoconstrictors

- Serotonin (5-HT) (from platelets)
- Thromboxane A2 (from platelets)
- Endothelin (from endothelial cells)



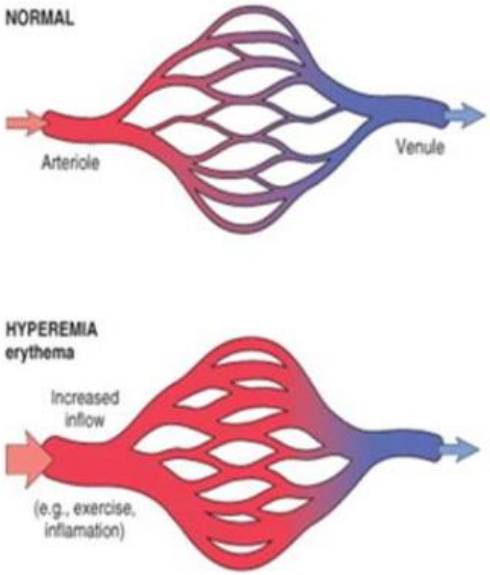
Vasodilators

- Nitric oxide (from endothelial cells)
- Prostacyclin (from endothelial cells)
- Histamine
- Vasodilator metabolites (VDMs) from tissue metabolism

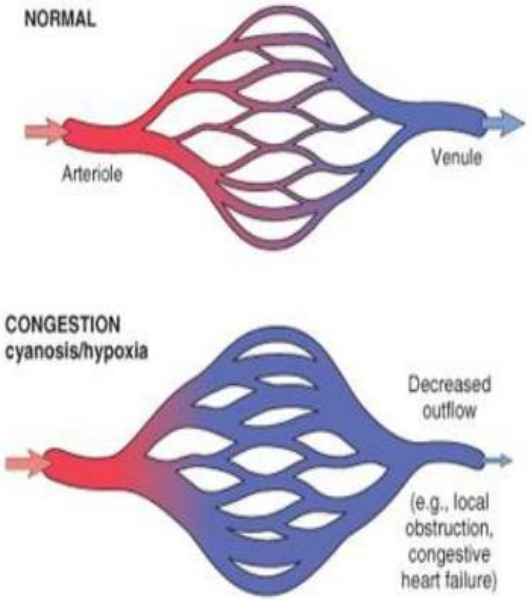
Buerger's Postural Test

- The patient is in supine position. Ask the patient to raise the leg one after the other keeping the knees straight. The legs of normal individual remain pink even if raised to 90°. But in case of an ischaemic limb elevation to a certain degree will cause marked pallor.
- The angle between the limb at which such pallor appears and the horizontal plane is known as Buerger's angle or vascular angle. Vascular angle < 30° - severe indicates arterial occlusion

HYPEREMIA



CONGESTION



Hyperaemia vs Congestion

Both = Increased volume of blood in tissue

Hyperaemia	Congestion
• active process	• passive process
• arteriolar dilation	• impaired venous outflow
• Affected organ- pink/Red	• Affected organ- bluish
• e.g. skeletal muscle during exercise (physiologic), inflammation (pathologic)	• e.g. cardiac failure (systemic), venous obstruction (local)
	• Local / systemic

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vena cava superior syndr.

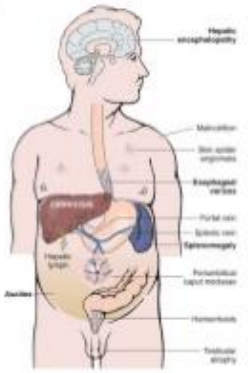
b.) systemic

right sided heart failure

chronic cor pulmonale

Local venous hyperemia

- In isolated venous obstruction - some organs
- Varied severity, depends on:
  - The time of appearance of congestion
    - gradually/suddenly
  - The presence of collaterals, anastomoses
- Examples
  - Peripheral venous failure of the legs –valve failure of the veins
  - Liver ciirhosis –portal hypertension
    - the formation of portosystemic venous shunts, with the rise in portal venous pressure
      - veins around and within the rectum (manifest as hemorrhoids),
      - the cardioesophageal junction (producing esophagogastric varices),
      - the retroperitoneum -periumbilical and abdominal wall collaterals-caput medusae
  - Acute venous obstruction + (-) collaterals ⇒ haemorrhagic necrosis
    - V. mesenterica –gut
    - V. lienalis
    - V. renalis



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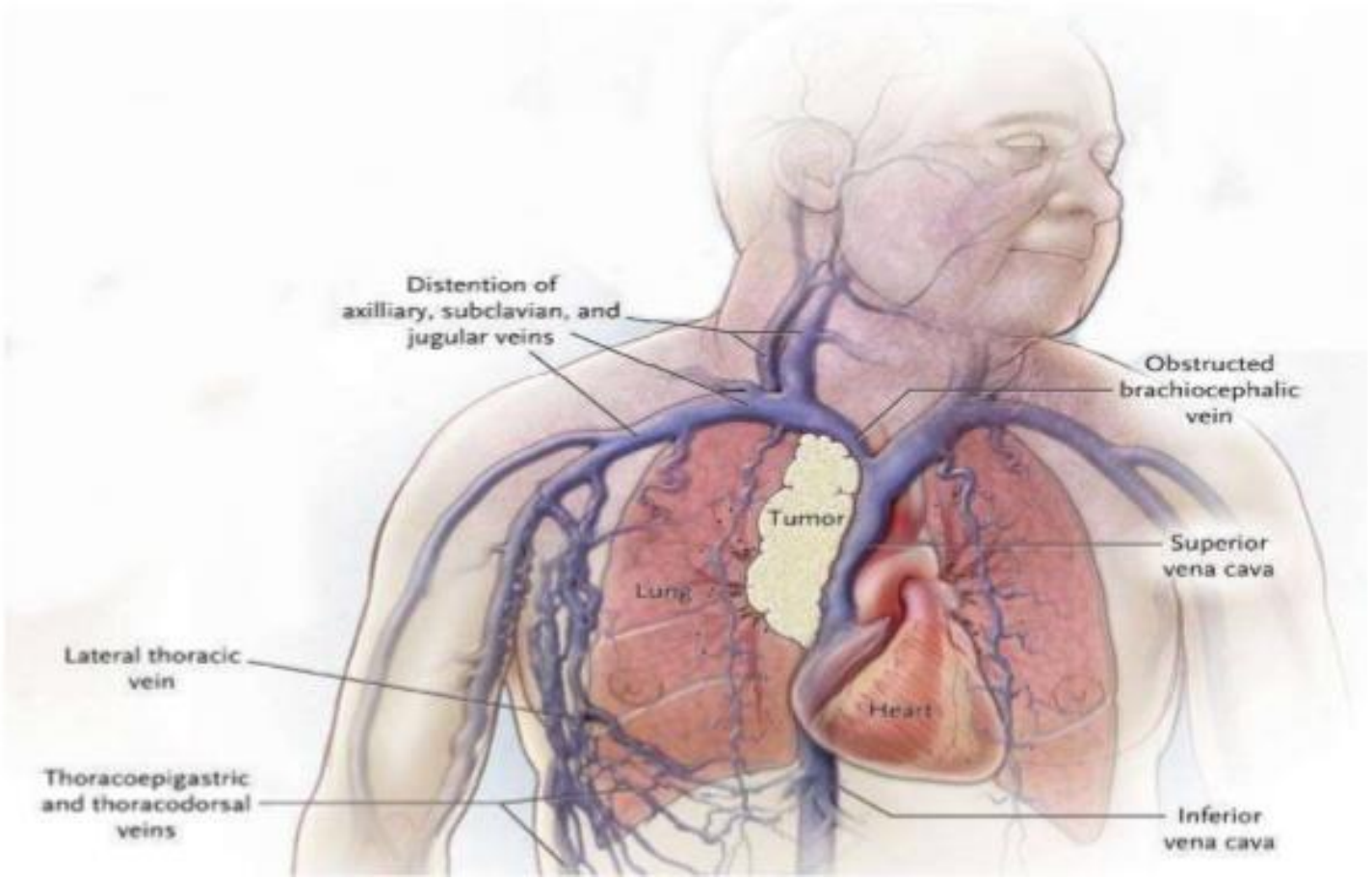
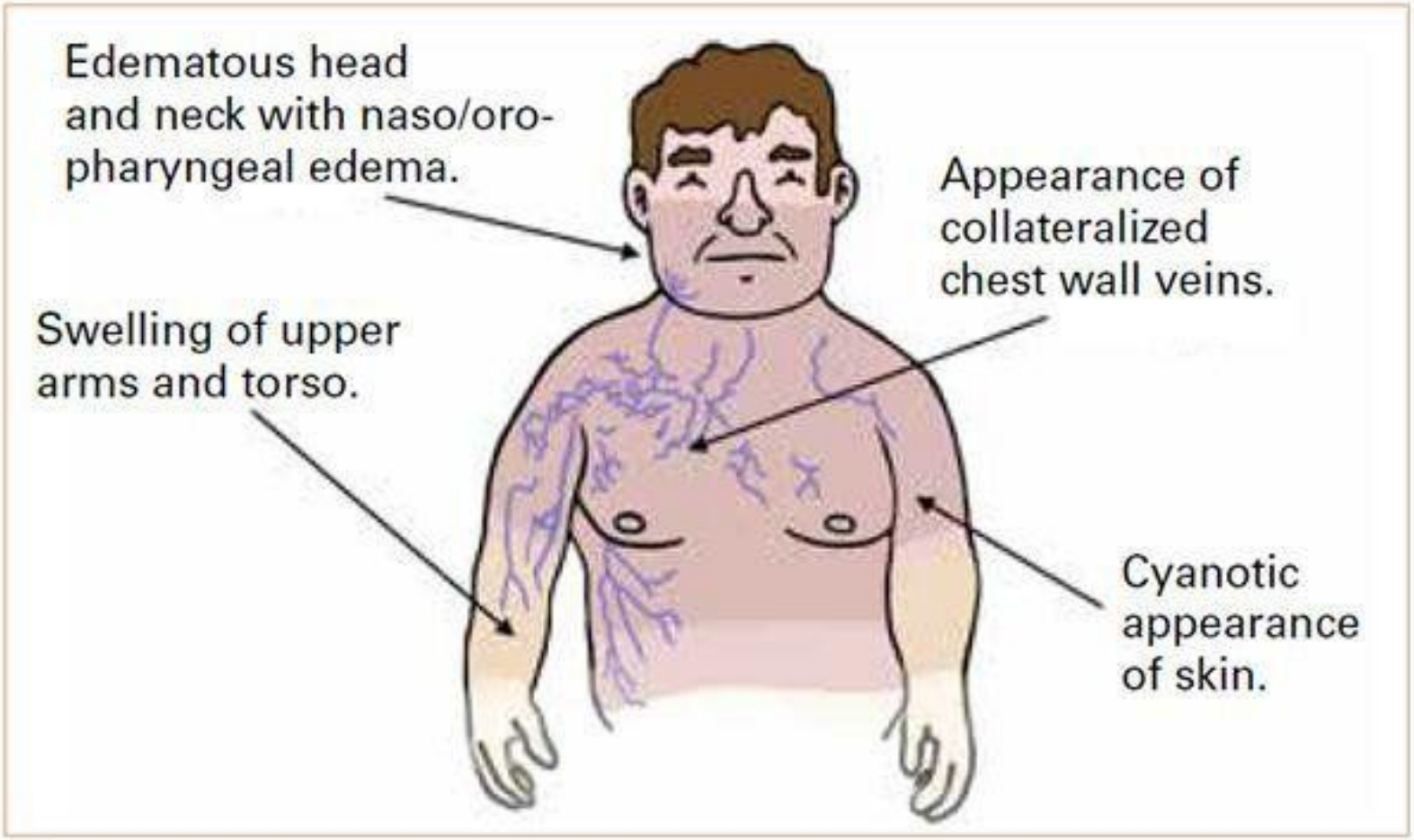
SUPERIOR VENA CAVA SYNDROME



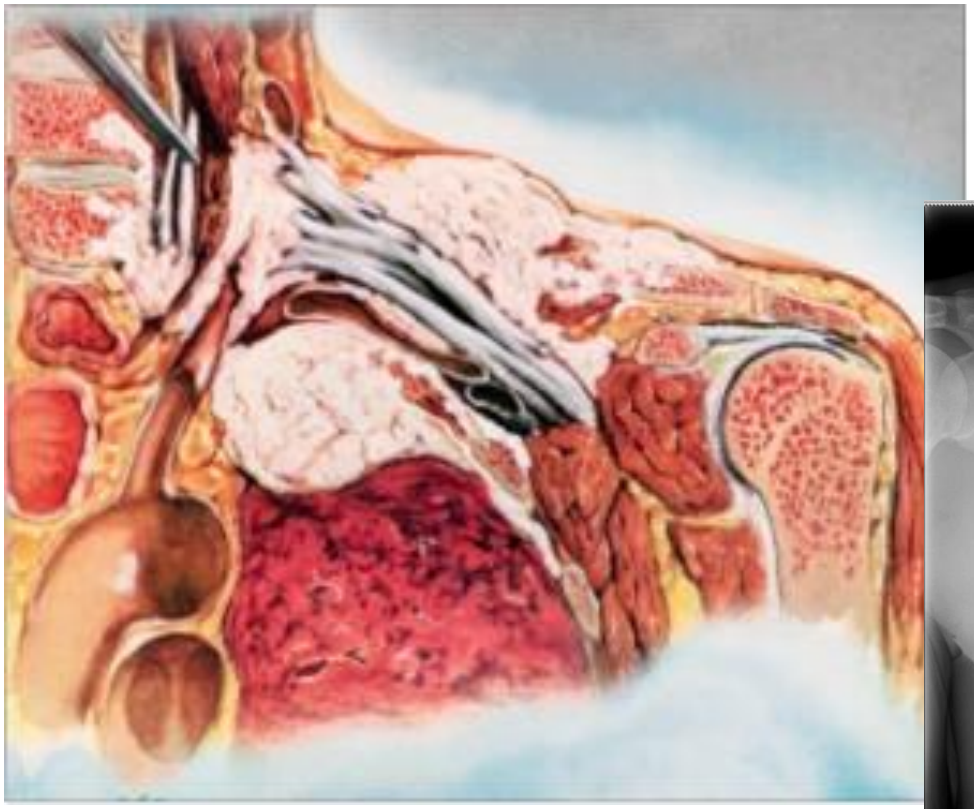

Edematous head and neck with naso/oro-pharyngeal edema.
Swelling of upper arms and torso.
Appearance of collateralized chest wall veins.
Cyanotic appearance of skin.

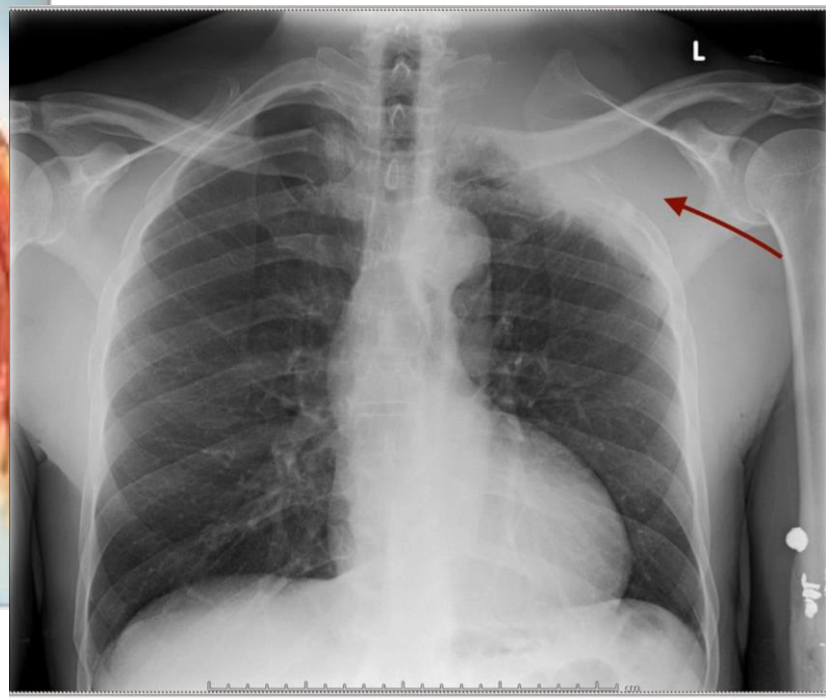
Horner sydrome (oculosympathetic paresis): miosis, ptosis, facial anhidrosis, enopthalmos

PEMBERTON'S SIGN

- **PROCEDURE:-**
  - ASK THE PATIENT TO RAISE BOTH RHE ARMS OVER THE HEAD TOUCHING THE EARS AND MAINTAIN IT FOR 2-3 MINS.
- **INTERPRETATION:-**
  - POSITIVE
  - NEGATIVE

Pancoast Tumor





Pancoast Tumor

Cancerous growth at the apex (top) of the lungs

1st described by Henry Pancoast in 1924

Constitute 5% of all lung cancers

Risk factors include smoking & asbestos exposure

95% of the tumors are non-small cell cancers

Symptoms are malaise, fever, weight loss, Horner syndrome & fatigue

Diagnosed by history, CT scan, MRI & biopsy

Treated by radiation therapy, chemotherapy & surgery

Spread to nearby tissues (ribs, nerves & vertebrae) in 30% cases

Complications are superior vena cava syndrome & thoracic outlet syndrome

5-year survival rate is 30%

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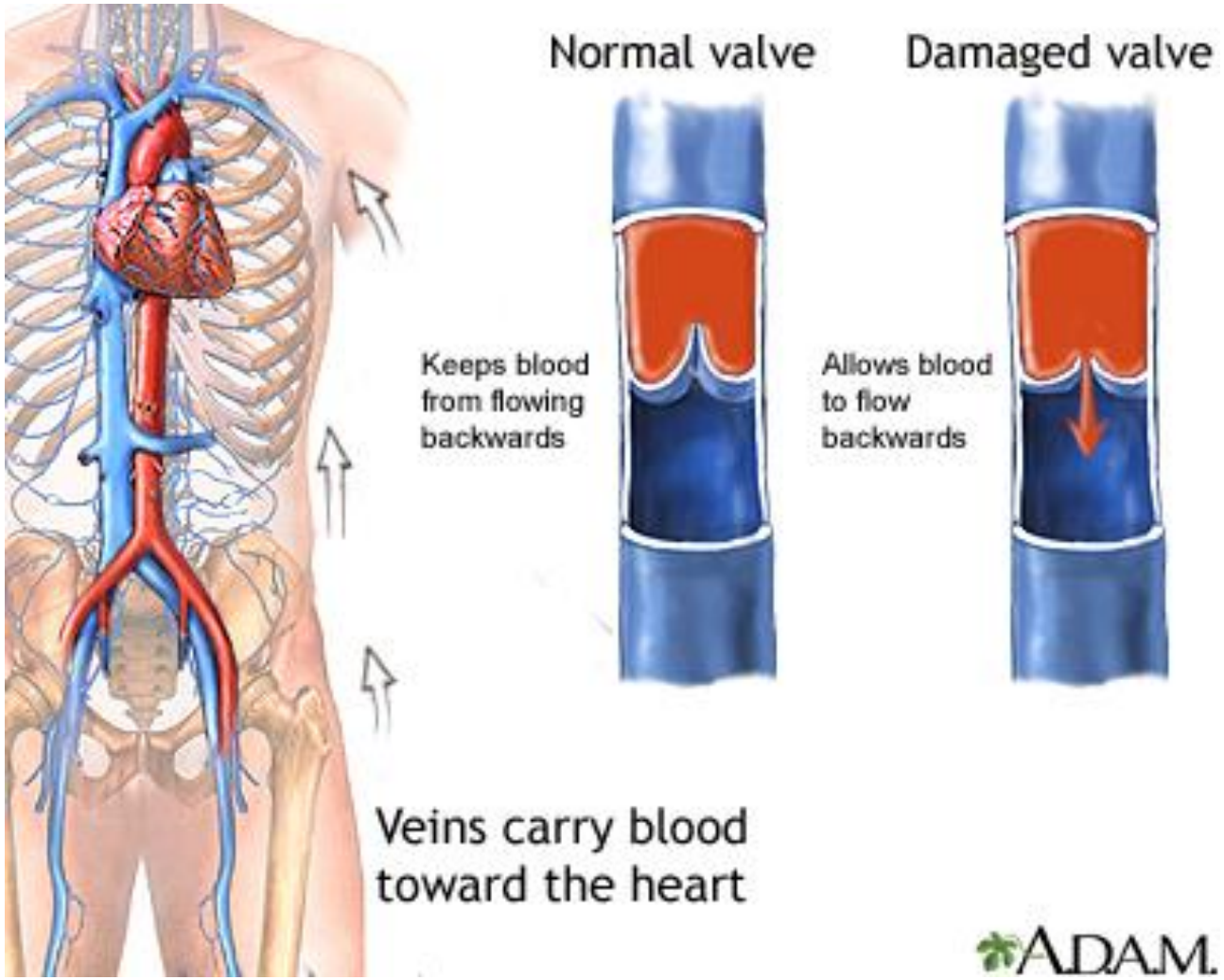
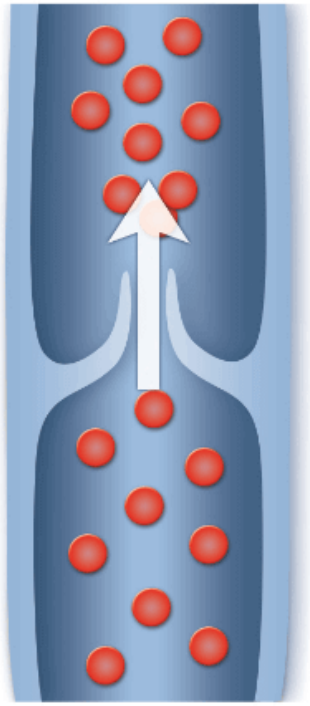
Ulcus cruris venosum

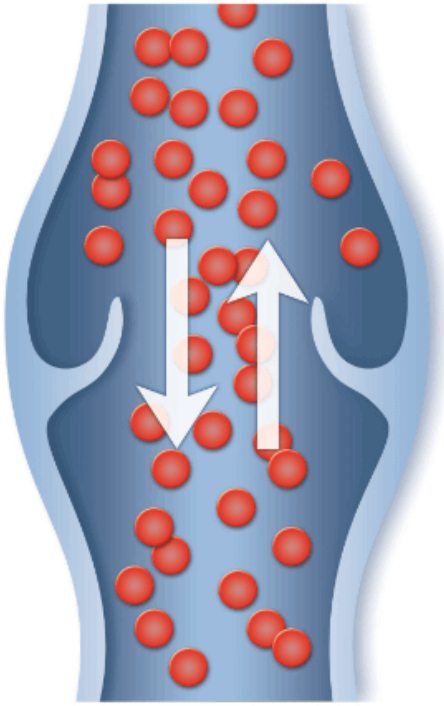

Normal valve
Damaged valve
Keeps blood from flowing backwards
Allows blood to flow backwards
Veins carry blood toward the heart
A.D.A.M.

Healthy Vein Valve



Healthy valves keep blood moving in one direction

Diseased Vein Valve



Diseased valves cause blood to move in both directions, elevating venous pressure

Hyperemia

Active

- loss of sympathetic activity
- vasoactive compounds

↓

vasodilatation

-inflammations (rubor, calor) – (NO, PGE2)

-excercise

-eating

-psychological effects (blushing)

-hormones (postmenopausal)

-carcinoid flush

-reperfusion

-medicinal drugs (Na-nitrite, blue tablet)

Passive (congestion)

- venous block

a.) local

venous thrombus

venous compression

torsion

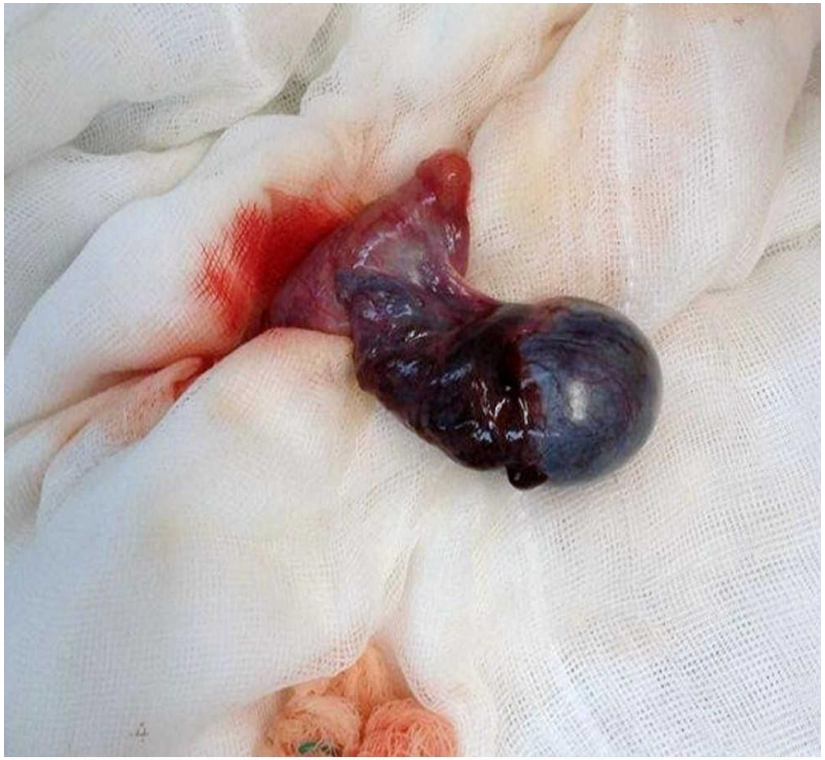
invagination

vena cava superior syndr.

b.) systemic

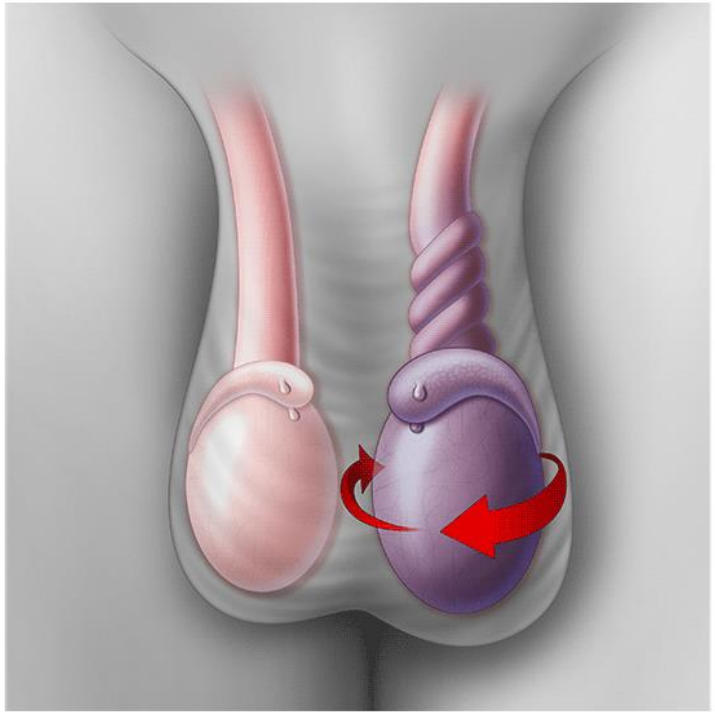
right sided heart failure

chronic cor pulmonale



Cir Cir. 2017;85:432-5

Torsion



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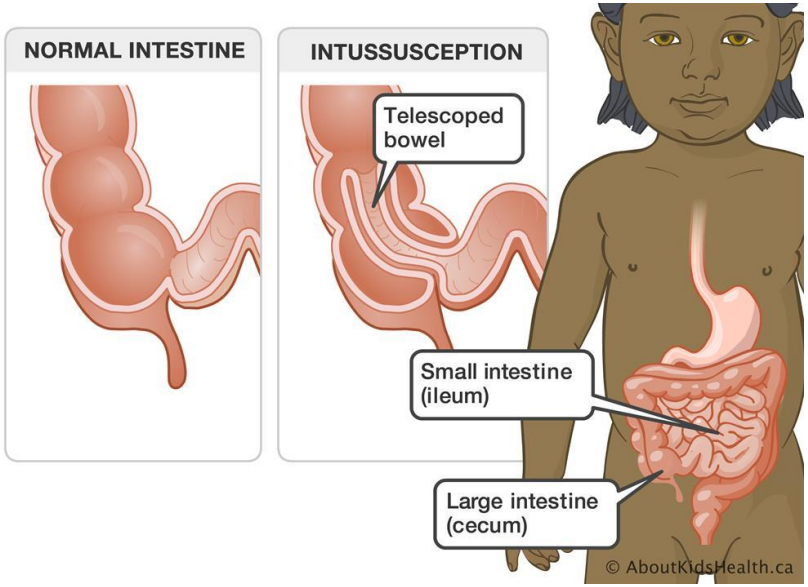
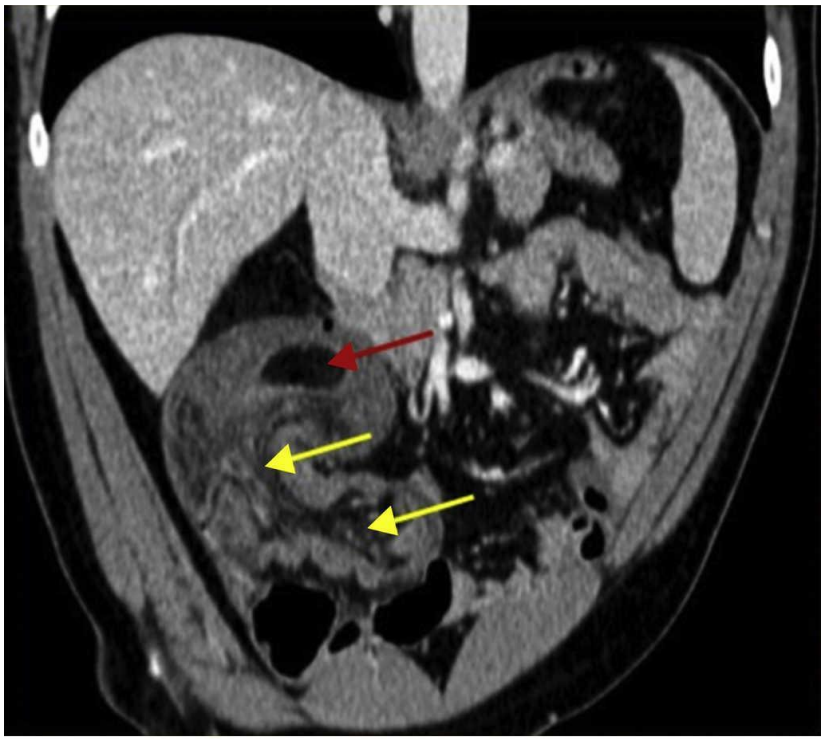
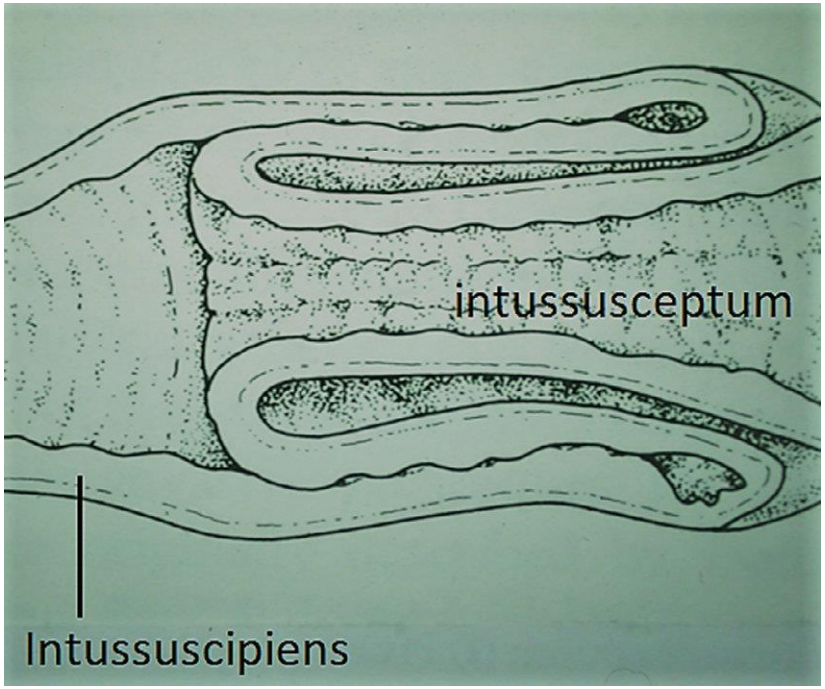


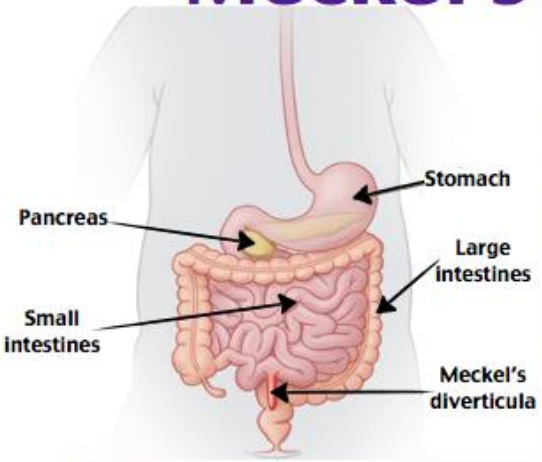


Rev Gastroenterol Mex. 2018;83:457–9

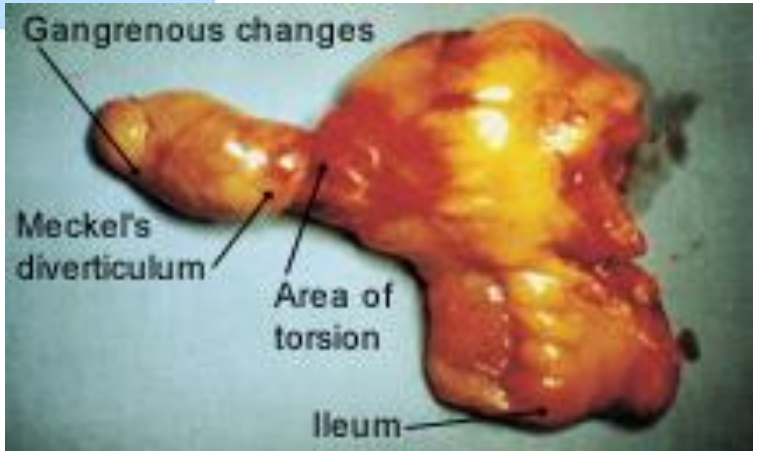


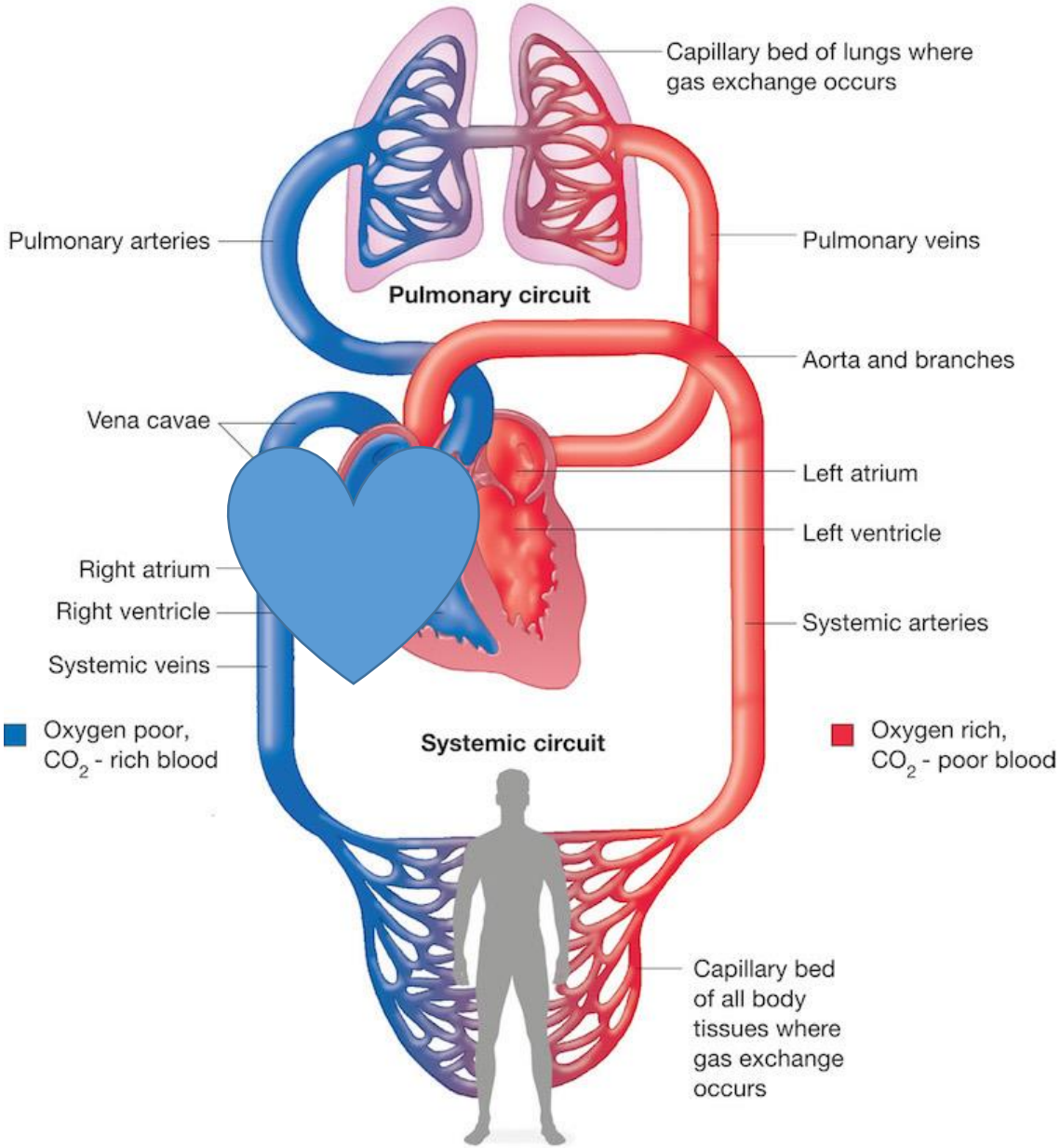
Hemangioma
Inflammatory polyp
Kaposi sarcoma
Lipoma
Meckel diverticulum
Neurofibroma
Peutz–Jegher polyp
Tuberculosis
Submucosal hemorrhages from unregulated anticoagulation
Enteric malignant
Adenocarcinoma
Carcinoid tumor
Leiomyosarcoma
Lymphoma
Metastatic carcinoma (melanoma most common)
Malignant GIST
Neuroendocrine tumor

Meckel's Diverticulum



- True diverticula of **all 3 layers** of the small intestines
- Caused by incomplete obliteration of the **vitelline duct**
- **Rule of 2s**: 2-years-old, 2 ft from ileocecal valve, 2 in long, 2% of population
- Usually asymptomatic
- **Painless bleeding** due to ulcer caused by **heterotopic gastric tissue**
- Obstruction caused by intussusception, volvulus, hernia
- Nuclear medicine scan (**Meckel's scan**)




Capillary bed of lungs where gas exchange occurs
Pulmonary arteries
Pulmonary veins
Pulmonary circuit
Aorta and branches
Vena cavae
Left atrium
Left ventricle
Right atrium
Right ventricle
Systemic arteries
Systemic veins
Oxygen poor, CO_2 - rich blood
Systemic circuit
Oxygen rich, CO_2 - poor blood
Capillary bed of all body tissues where gas exchange occurs


RIGHT SIDED
HEART
FAILURE


LEFT SIDED FAILURE
• Paroxysmal Nocturnal Dyspnea
• Elevated Pulmonary Capillary Wedge Pressure
• Pulmonary Congestion
- Cough
- Crackles
- Wheezes
- Blood-Tinged Sputum
- Tachypnea
• Restlessness
• Confusion
• Orthopnea
• Tachycardia
• Exertional Dyspnea
• Fatigue
• Cyanosis


RIGHT SIDED FAILURE
(Cor Pulmonale)
• Fatigue
• ↑ Peripheral Venous Pressure
• Ascites
• Enlarged Liver & Spleen
• May be secondary to chronic pulmonary problems
• Distended Jugular Veins
• Anorexia & Complaints of GI Distress
• Weight Gain
• Dependent Edema

LEFT SIDED FAILURE

- SOB/DOE
- Crackles/rales at bases
- Tachypnea
- Diaphoresis
- Weight gain
- Fatigue
- Extra heart sounds
- Mental status changes
- Capillary refill >3 sec

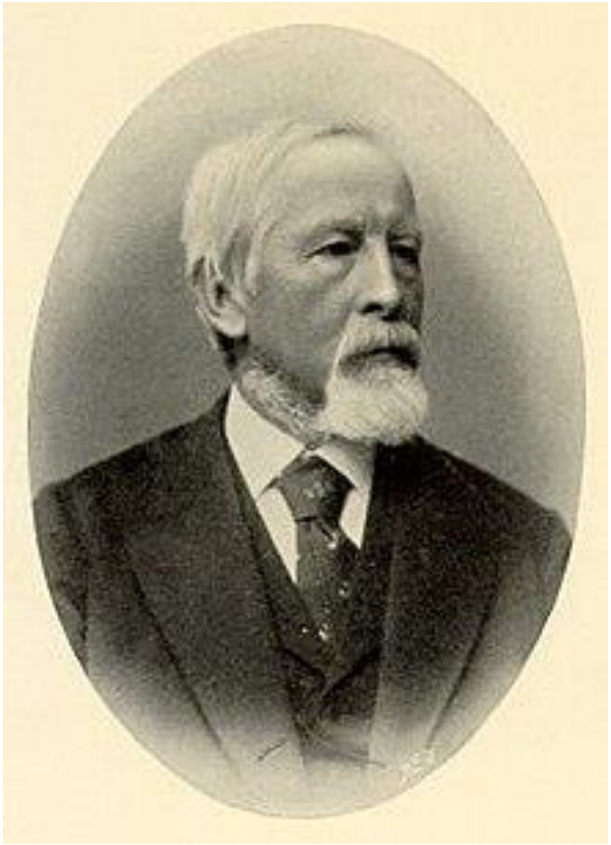
RIGHT SIDED FAILURE

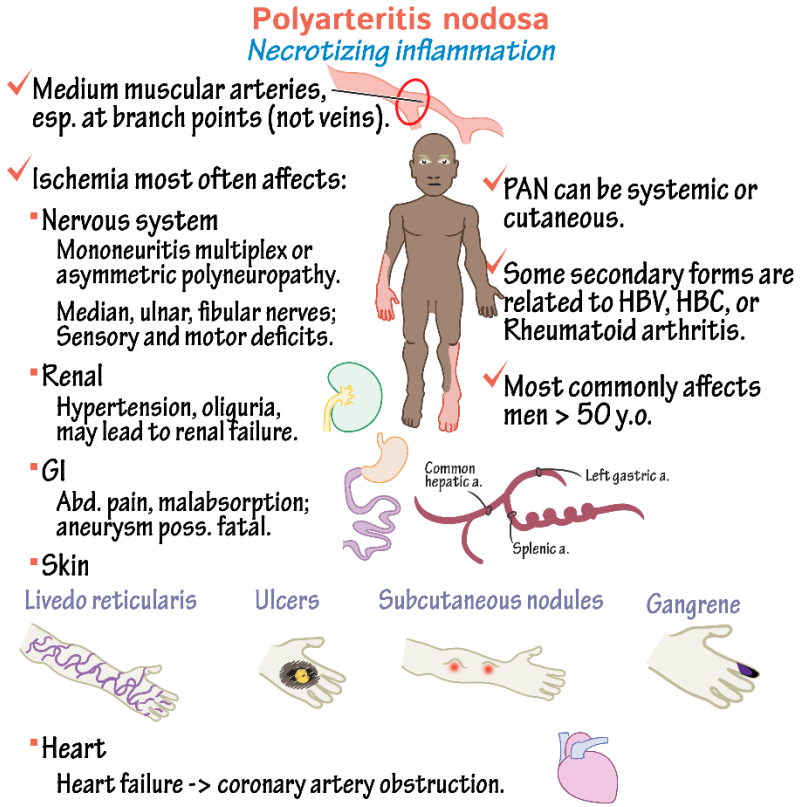
- Hepatomegaly
- Splenomegaly
- Ascites
- Dependant pitting edema
- JVD (Kussmaul's sign)
- Weight gain
- Anorexia
- Extra heart sounds

1. What is the differential for Kussmaul's sign?

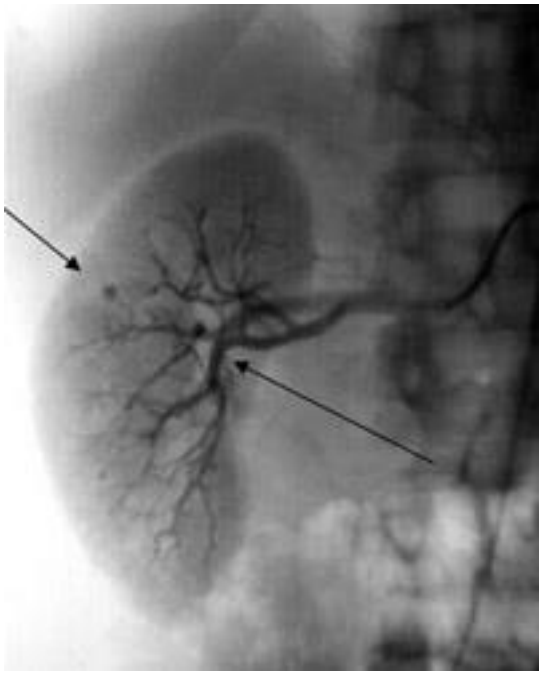
Typically the JVP ⬇ with inspiration as the negative intrathoracic pressure "sucks in" blood from the vena cava. When there is a lack of ⬇ or an ⬆ in JVP with inspiration, this finding is called **Kussmaul's sign.** Kussmaul's sign reflects conditions where there is **RV dysfunction, impaired RV filling and ⬆ RA pressure.**

Kussmaul's sign is classically associated with **constrictive pericarditis.** Other conditions where we may see a positive Kussmaul's sign include **restrictive cardiomyopathy**, RV predominant infarction, massive PE, tricuspid stenosis, and **severe TR.**

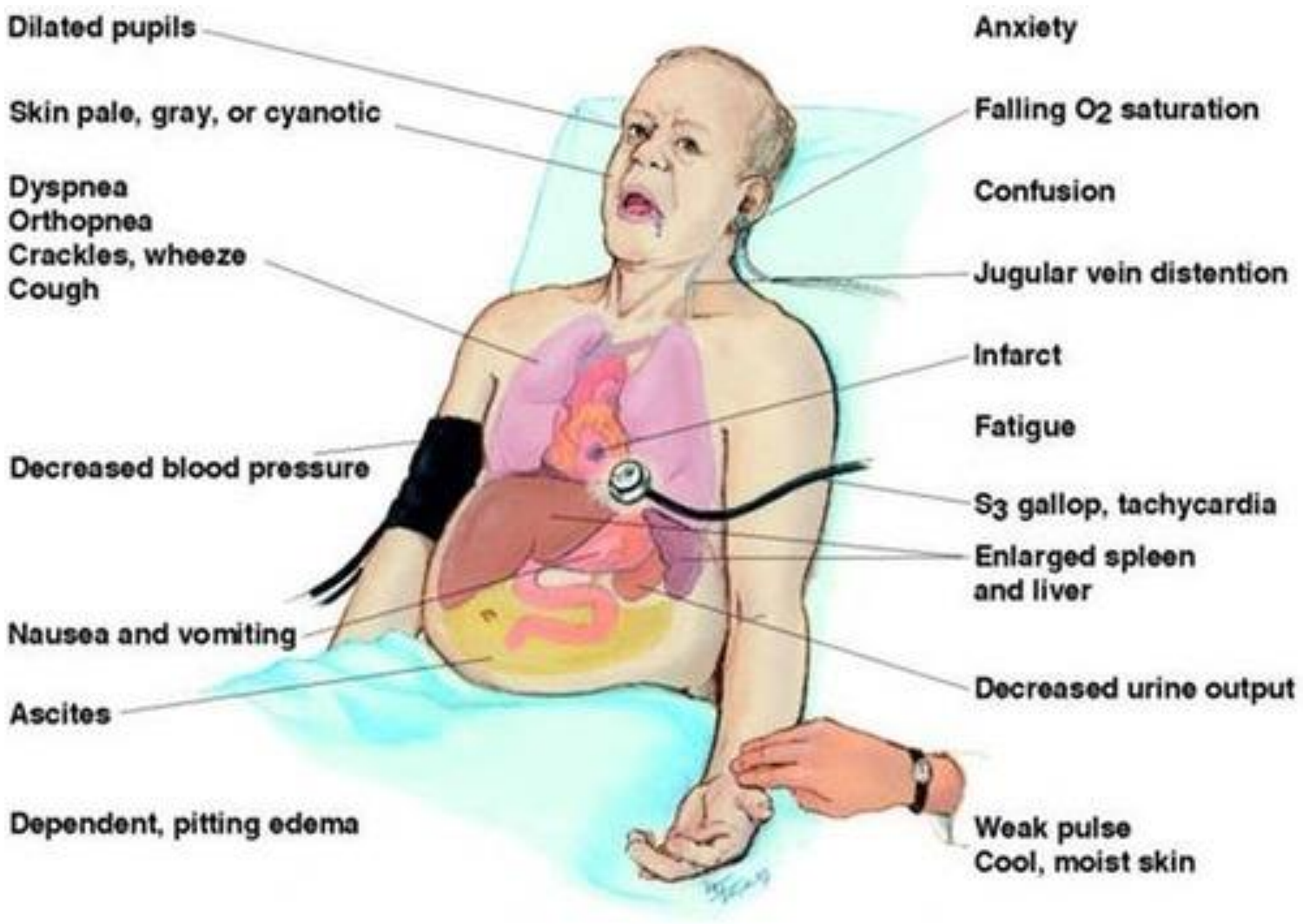




Kussmaul disease (polyarteritis nodosa)




Skin


Dilated pupils
Skin pale, gray, or cyanotic
Dyspnea
Orthopnea
Crackles, wheeze
Cough
Decreased blood pressure
Nausea and vomiting
Ascites
Dependent, pitting edema
Anxiety
Falling O_2 saturation
Confusion
Jugular vein distention
Infarct
Fatigue
S_3 gallop, tachycardia
Enlarged spleen and liver
Decreased urine output
Weak pulse
Cool, moist skin

Right sided heart failure

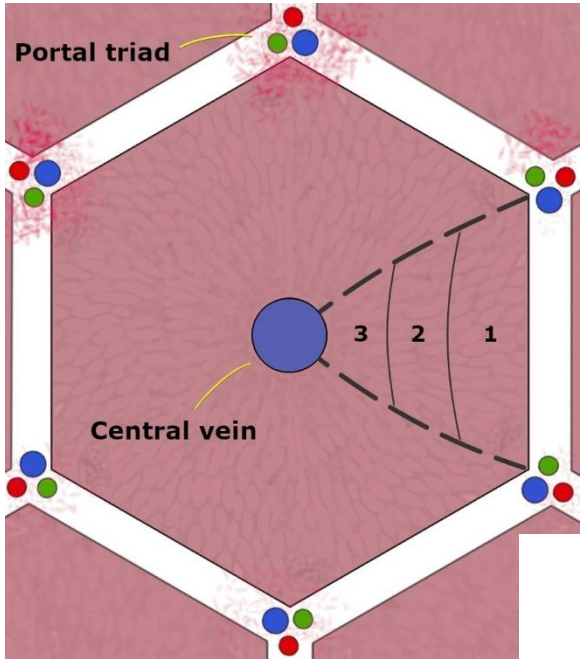
visceromegaly (hepatomegaly, splenomegaly)

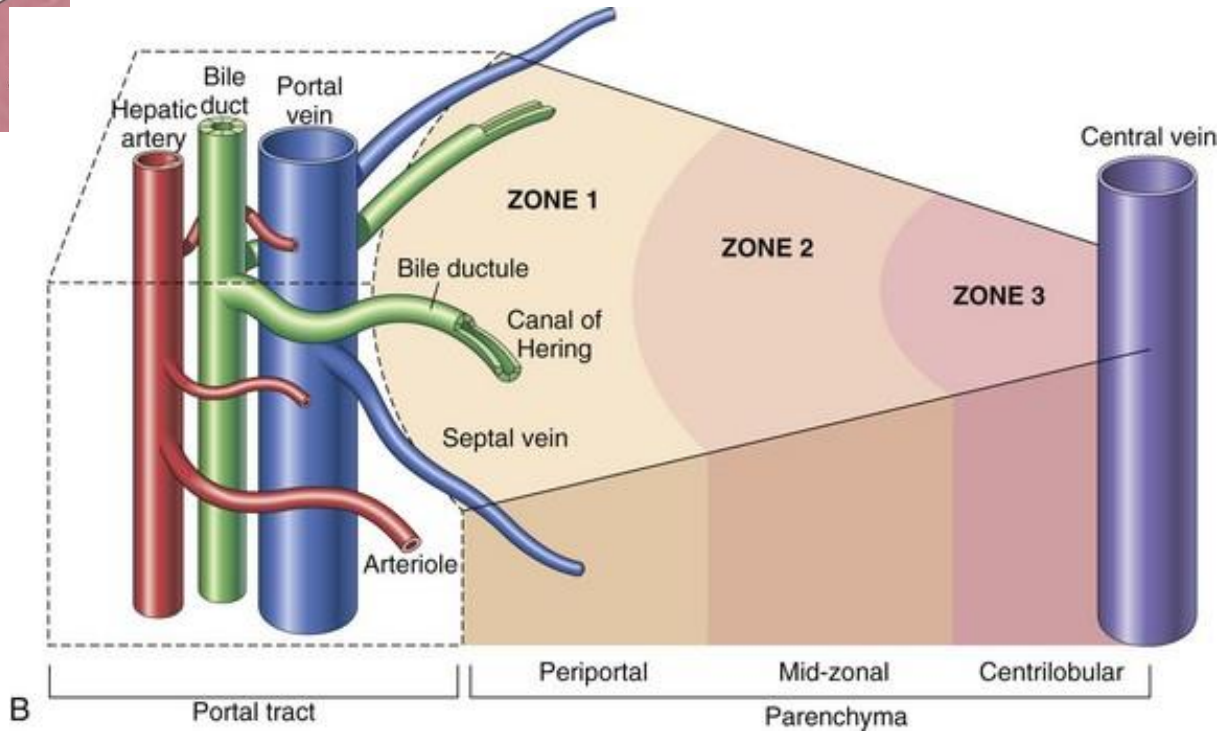
congestive gastritis, duodenitis

Kidney: dilatation of stellate veins, livid pyramids

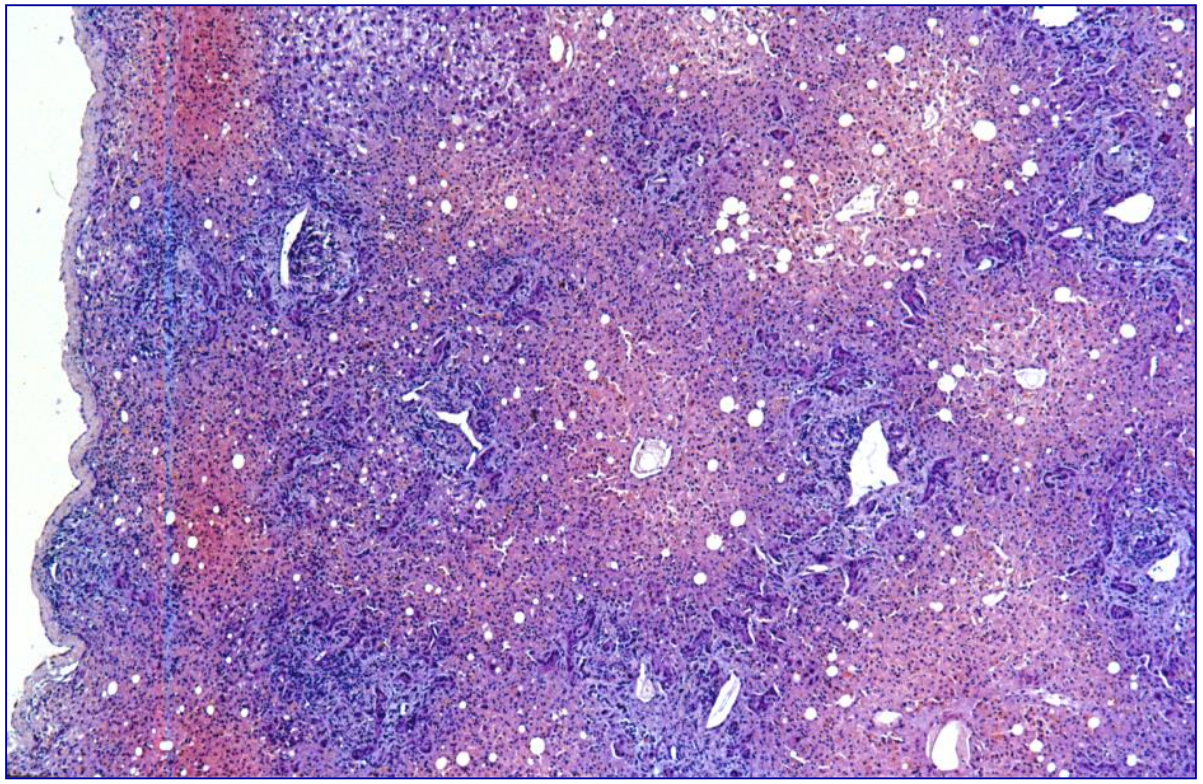
cyanosis

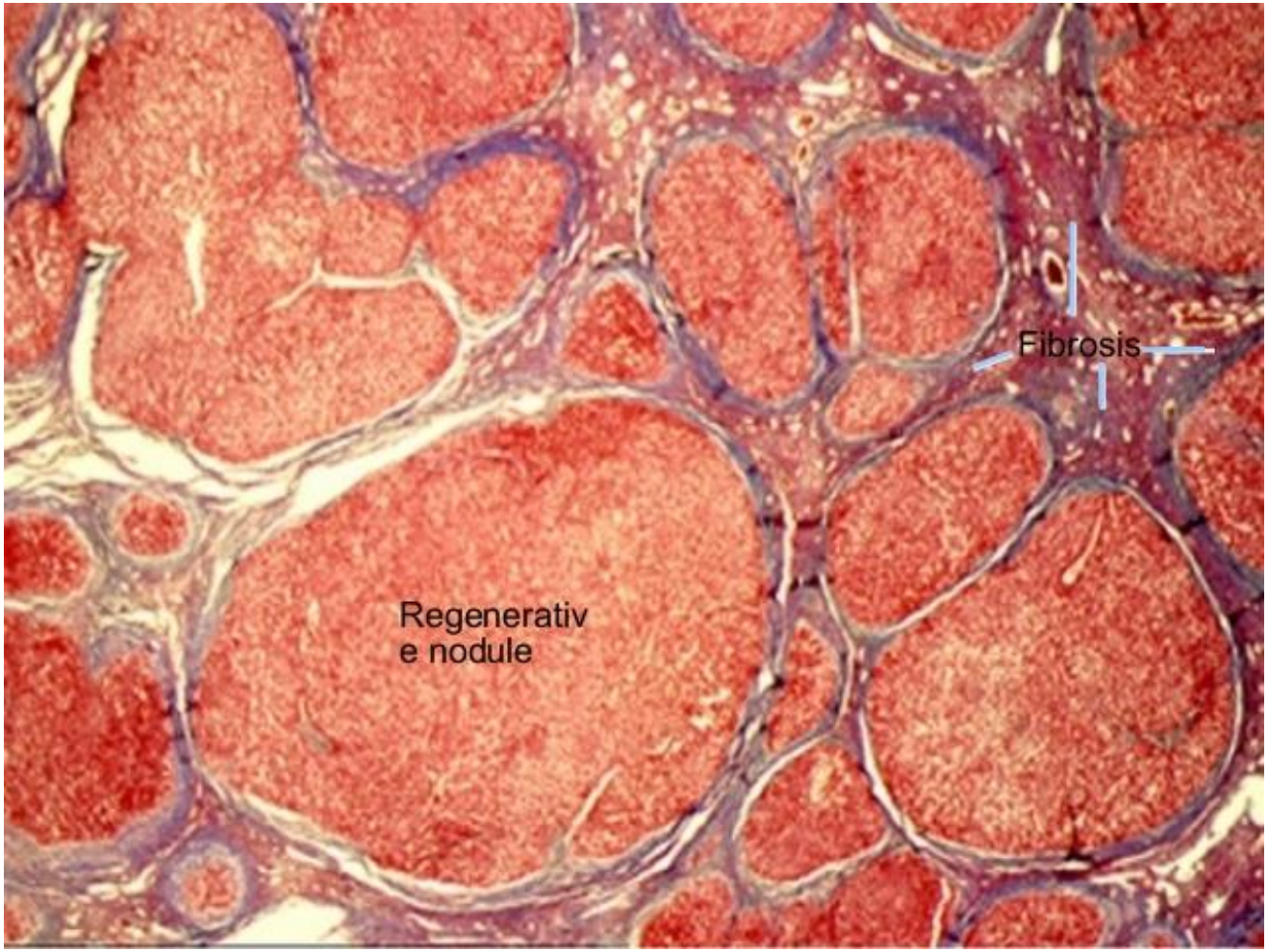
(oedemas)

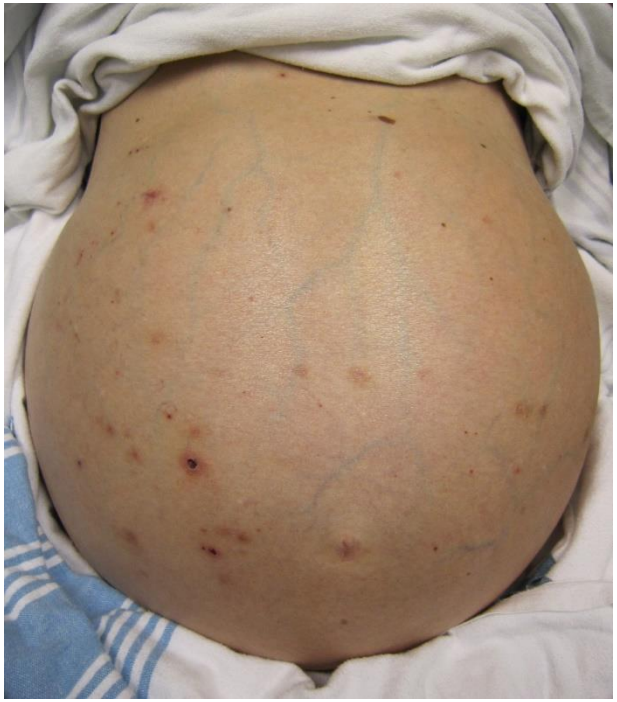

Portal triad
3
2
1
Central vein


Hepatic artery
Bile duct
Portal vein
ZONE 1
ZONE 2
ZONE 3
Central vein
Bile ductule
Canal of Hering
Septal vein
Arteriole
B
Portal tract
Periportal
Mid-zonal
Centrilobular
Parenchyma


1
2
CENTIMETERS

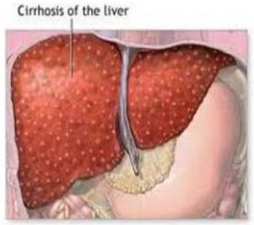


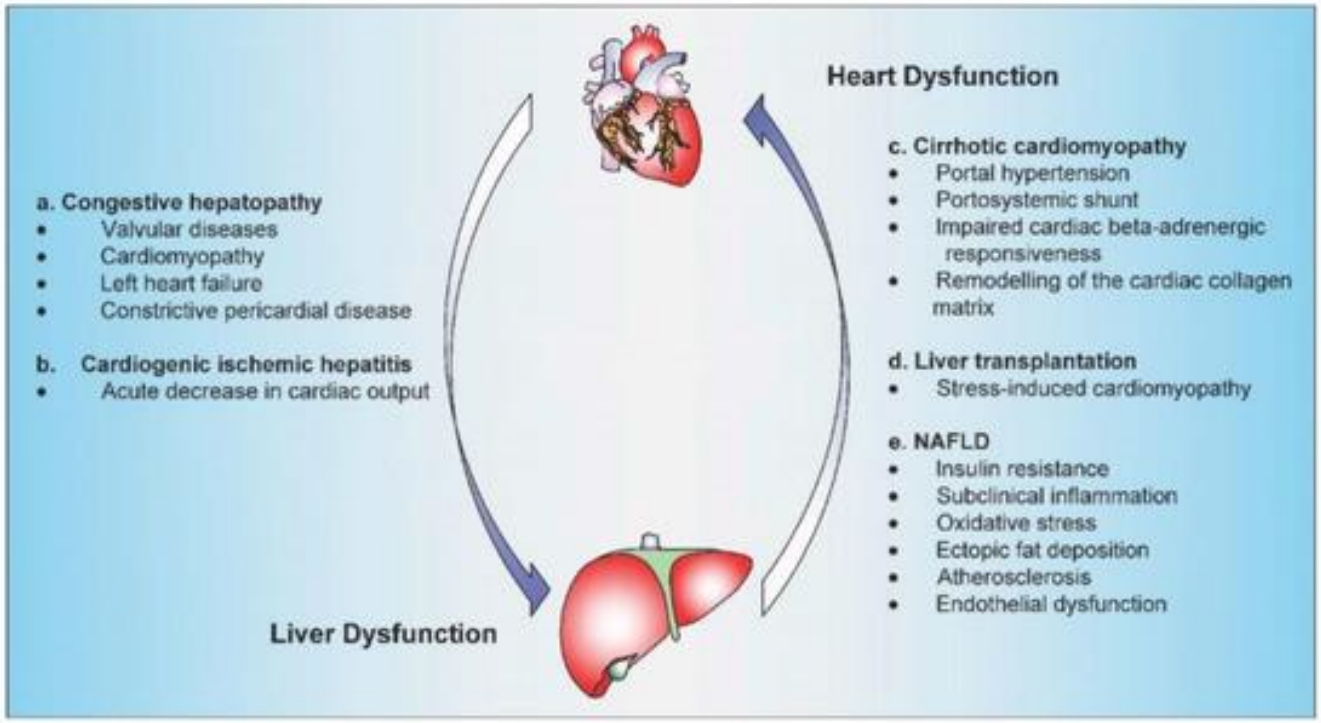

Fibrosis
Regenerativ
e nodule

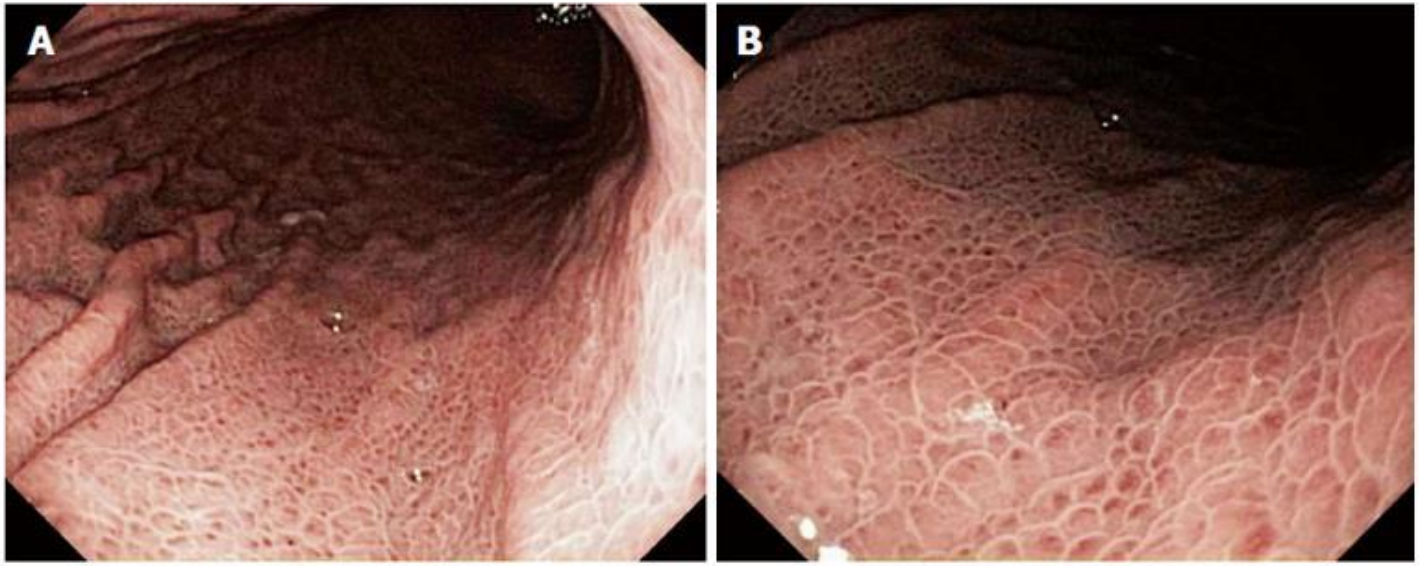


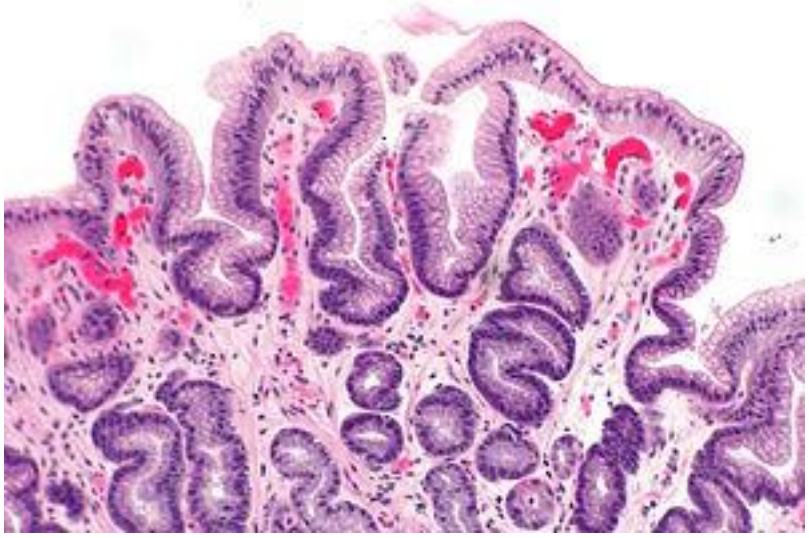

cm 1
2
3

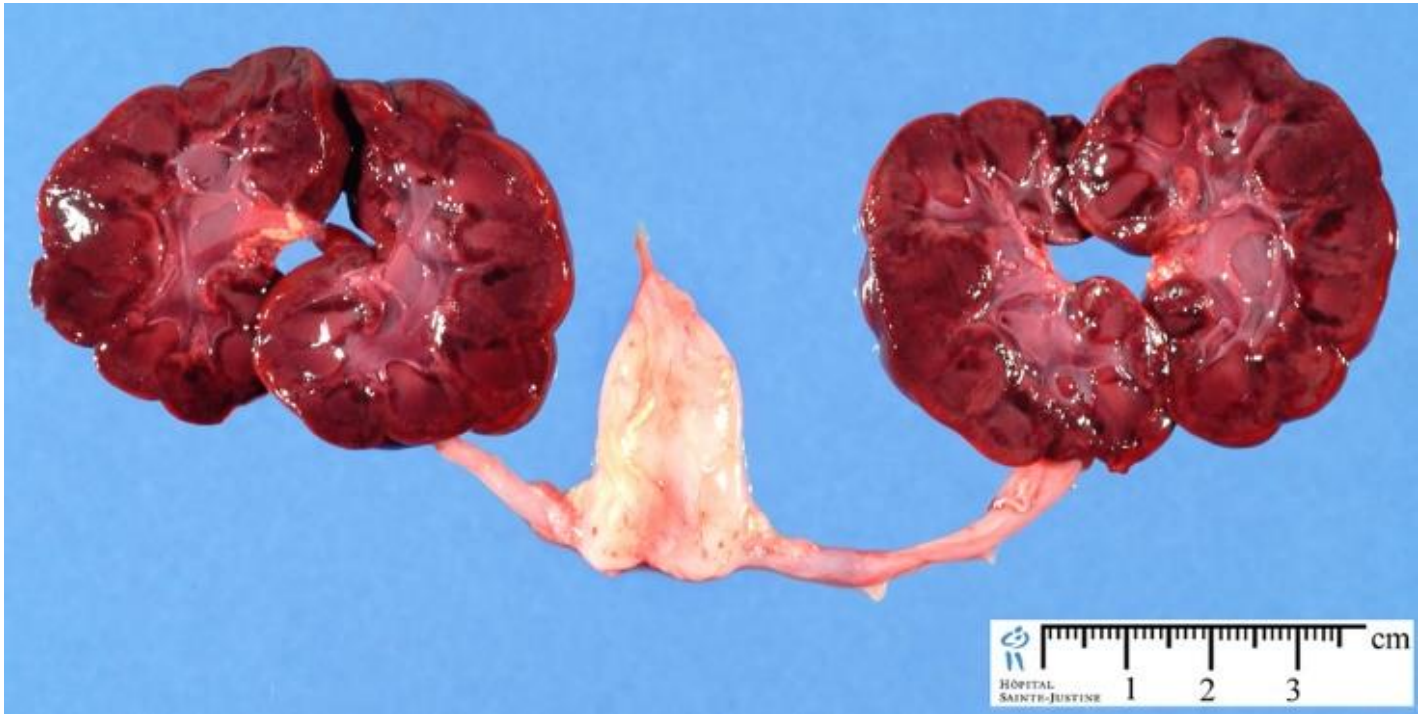
Cardiac Cirrhosis






A
B




cm
1
2
3
HÔPITAL
SAINTE-JUSTINE

Cor pulmonale:

Hypertrophy and/or dilatation of the right side of the heart as a result of pulmonary hypertension (valve diseases, left heart problems excluded!!)

1. Acute cor pulmonale (massive pulmonary embolus, air embolism,
   amniotic fluid embolization

2. Chronic cor pulmonale

   a. diseases of the lung parenchyma

   (chronic bronchitis, emphysema, bronchiectasis, lung fibrosis, sarcoidosis, massive tbc)

   b. diseases of the chest wall impairing the respiration

   (kyphoscoliosis, pleural callus, extreme obesity)

   c. diseases of the lung vasculature

   (multiplex pulmonary microembolisation)

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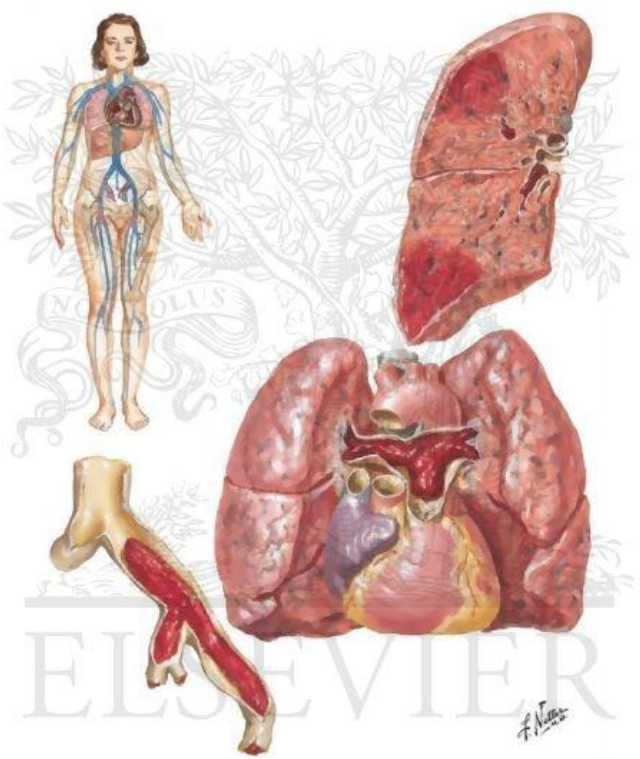
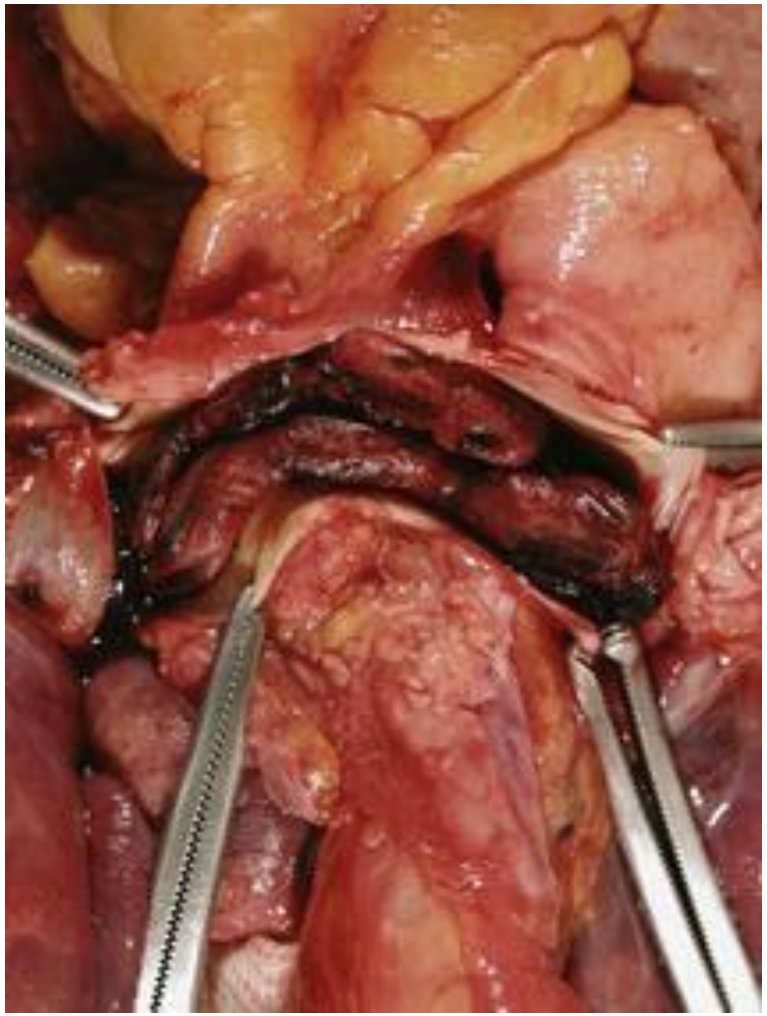
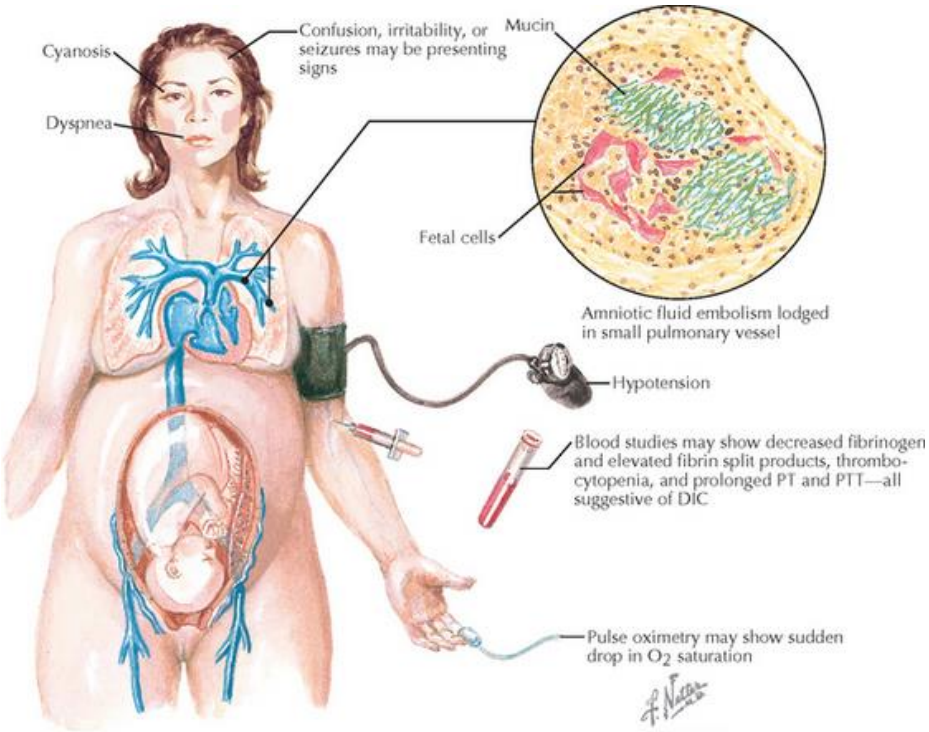
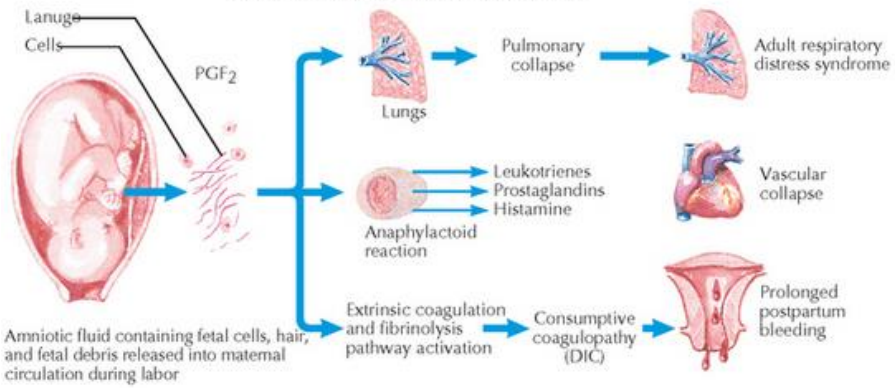




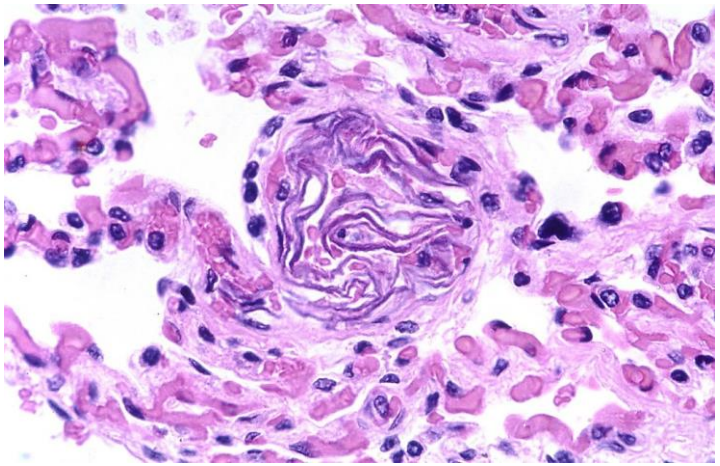


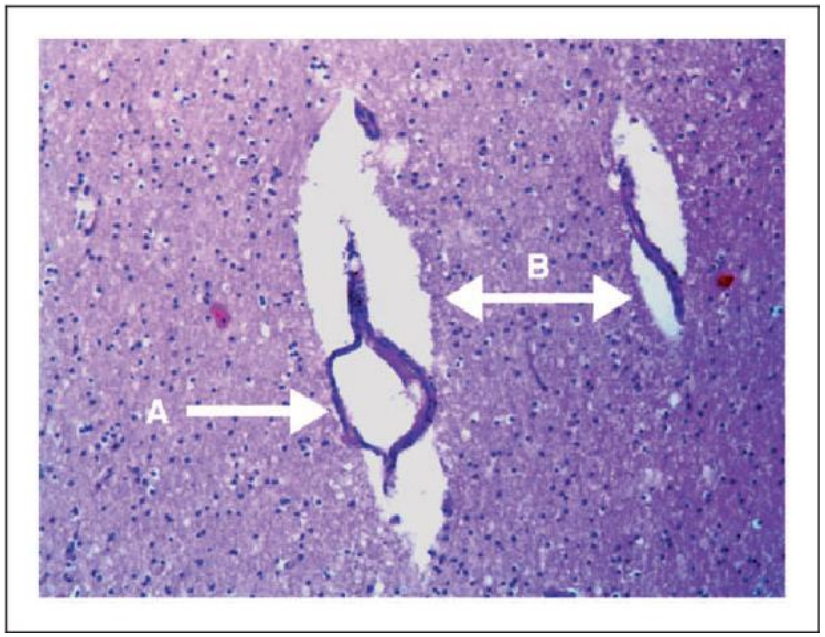
Sudden onset of dyspnea and shock during labor resulting from release of amniotic fluid and fetal material into maternal circulation

Clinical Features of Amniotic Fluid Embolism



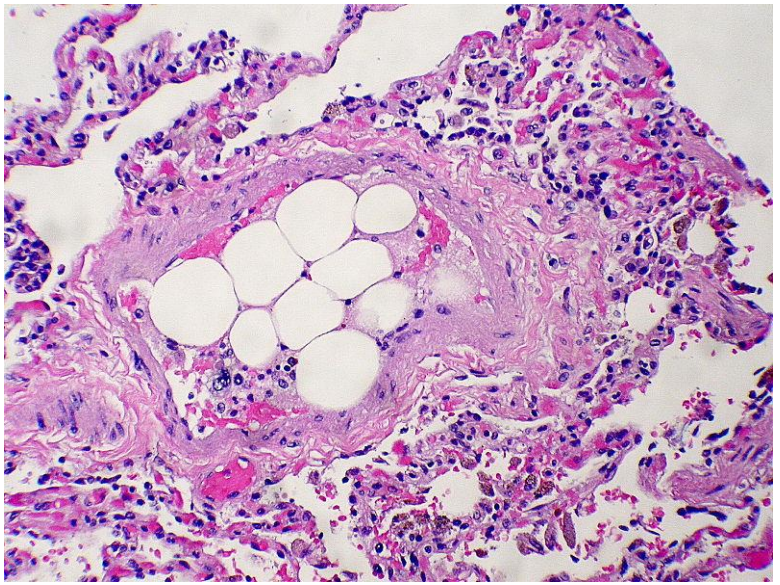
DIC, disseminated intravascular coagulation; PT, prothrombin time; PTT, partial thromboplastin time.





Fatal air embolism

Bone marrow embolism



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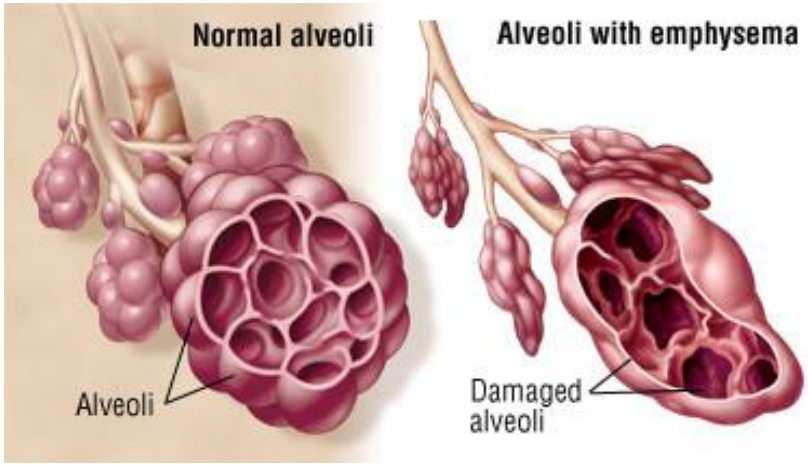
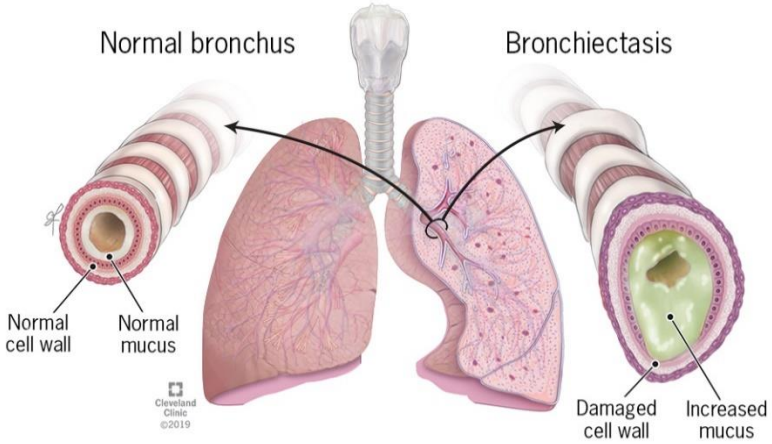
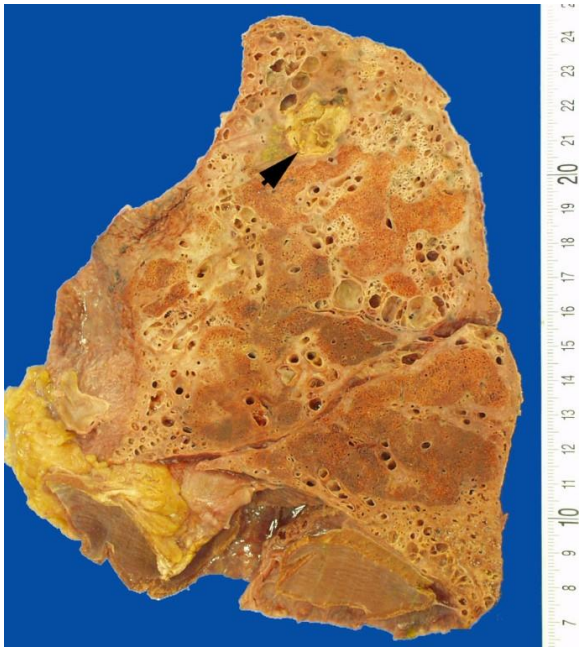
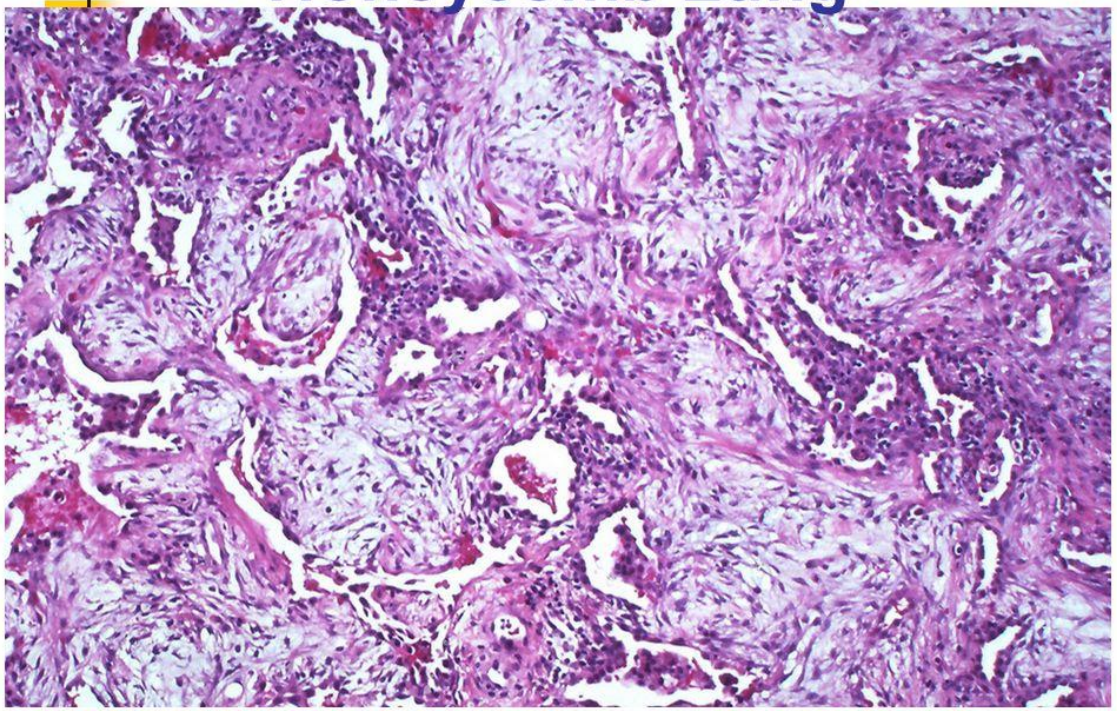

Normal alveoli
Alveoli with emphysema
Alveoli
Damaged alveoli

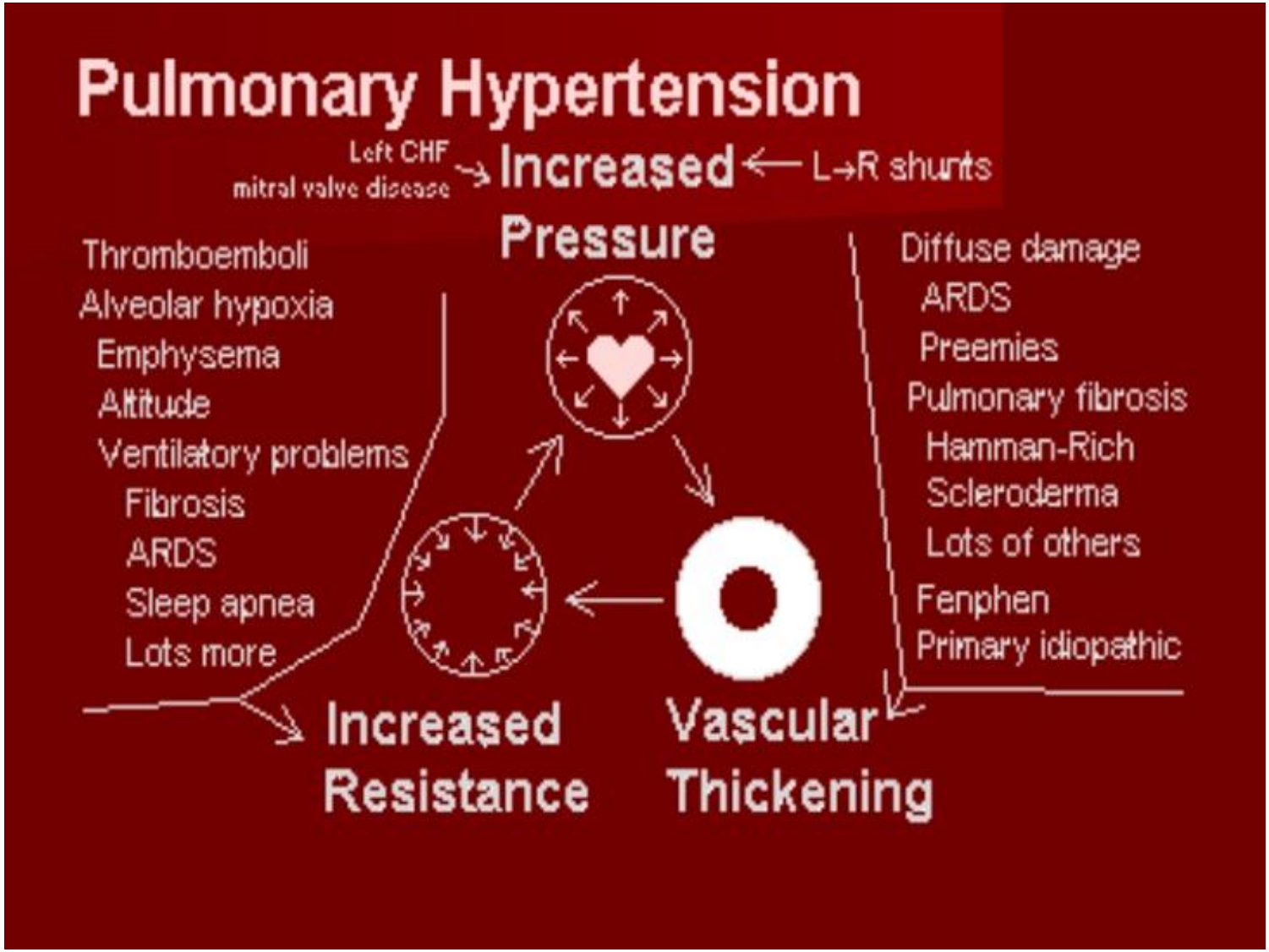

cm
1
2
3
4
5
6
SPECIMEN A-73-309
DATE


Normal bronchus
Bronchiectasis
Normal cell wall
Normal mucus
Damaged cell wall
Increased mucus
Cleveland Clinic ©2019

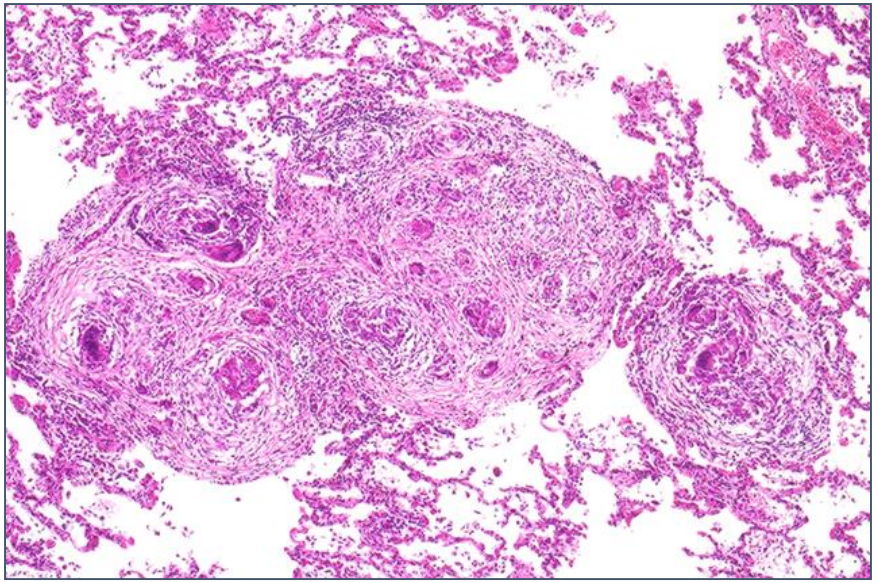


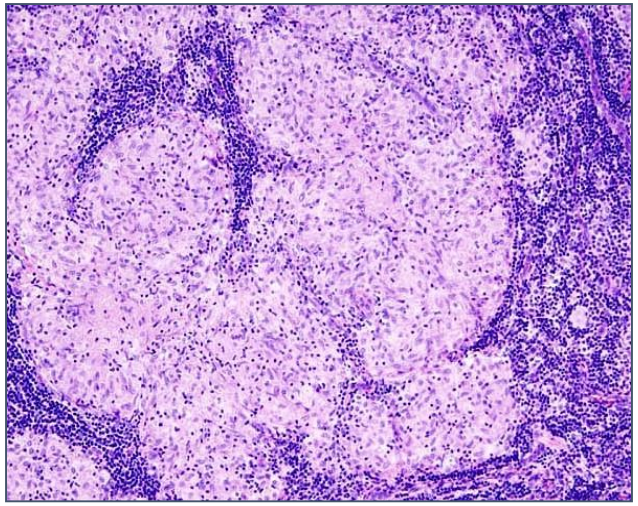
Advanced Interstitial Fibrosis
"Honeycomb Lung"

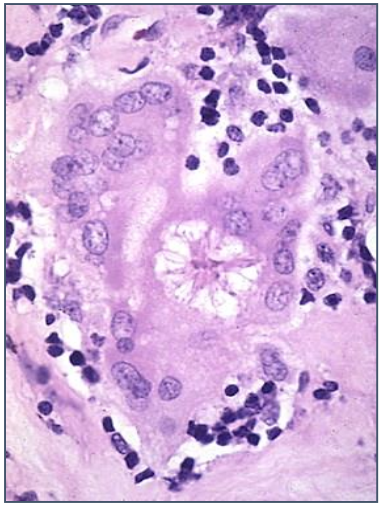


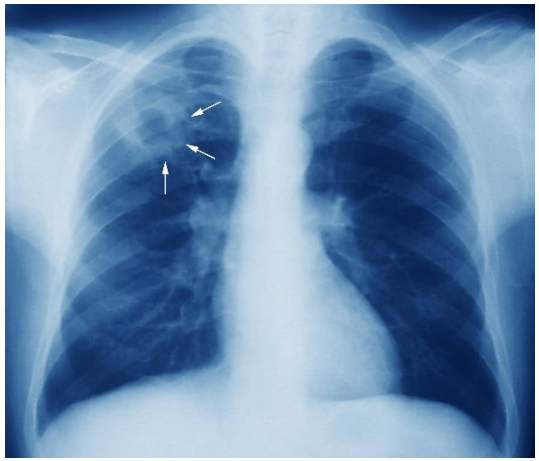

Pulmonary Hypertension
Left CHF
mitral valve disease
Increased
L→R shunts
Pressure
Thromboemboli
Alveolar hypoxia
Emphysema
Altitude
Ventilatory problems
Fibrosis
ARDS
Sleep apnea
Lots more
Diffuse damage
ARDS
Preemies
Pulmonary fibrosis
Hamman-Rich
Scleroderma
Lots of others
Fenphen
Primary idiopathic
Increased
Resistance
Vascular
Thickening

(Boeck) sarcoidosis









MYCOBACTERIUM TUBERCULOSIS (TB)

IMMUNE SYSTEM COMPROMISED] – AIDS or aging

↓

SPREADS TO UPPER LOBES] – Oxygenation greatest

↓

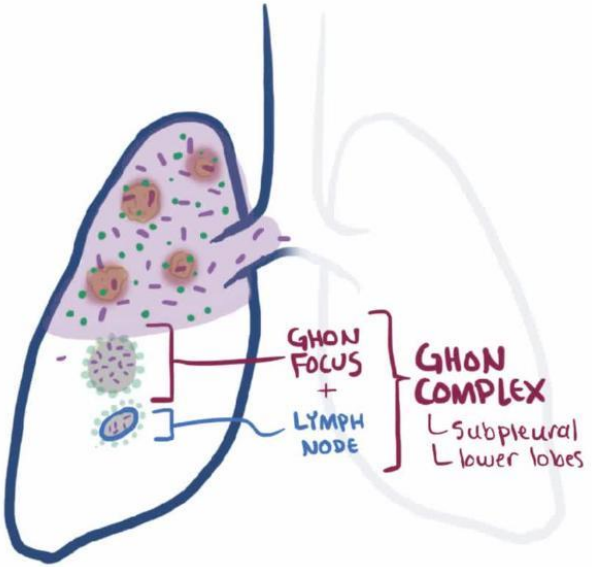
MEMORY T CELLS release CYTOKINES

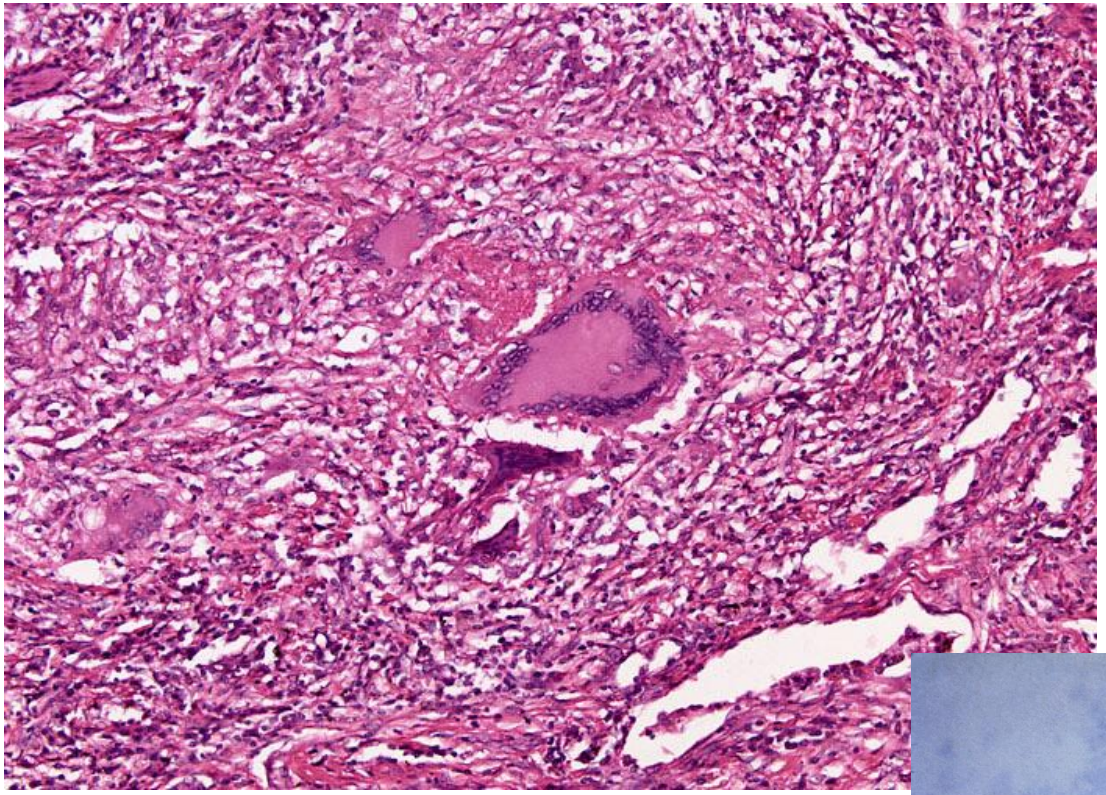
↓

(more) CASEOUS NECROSIS ⇝ CAVITATES

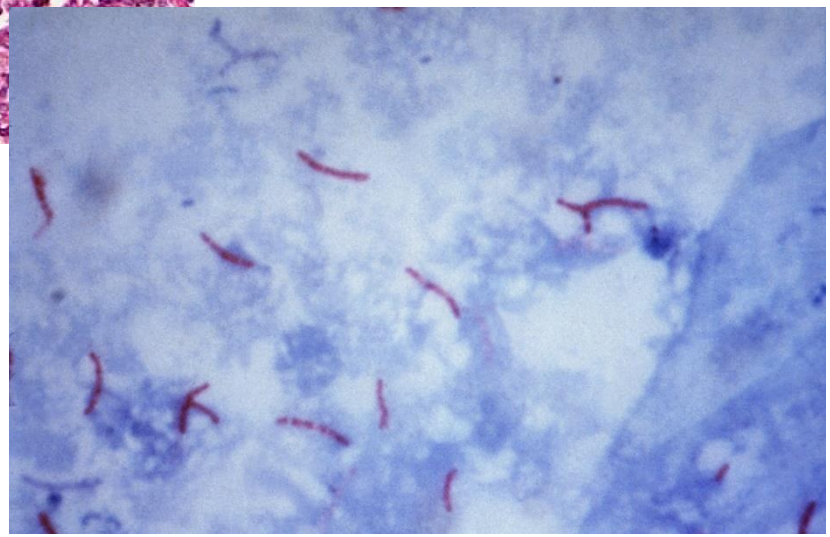
↓

TB DISSEMINATES (SPREADS)

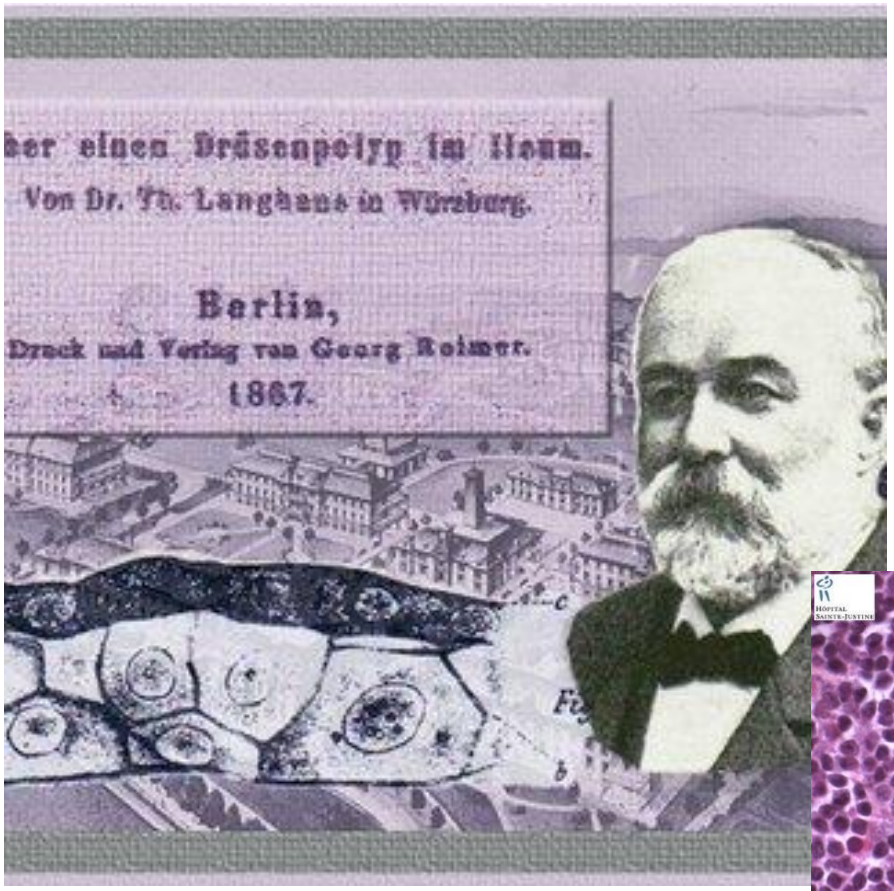


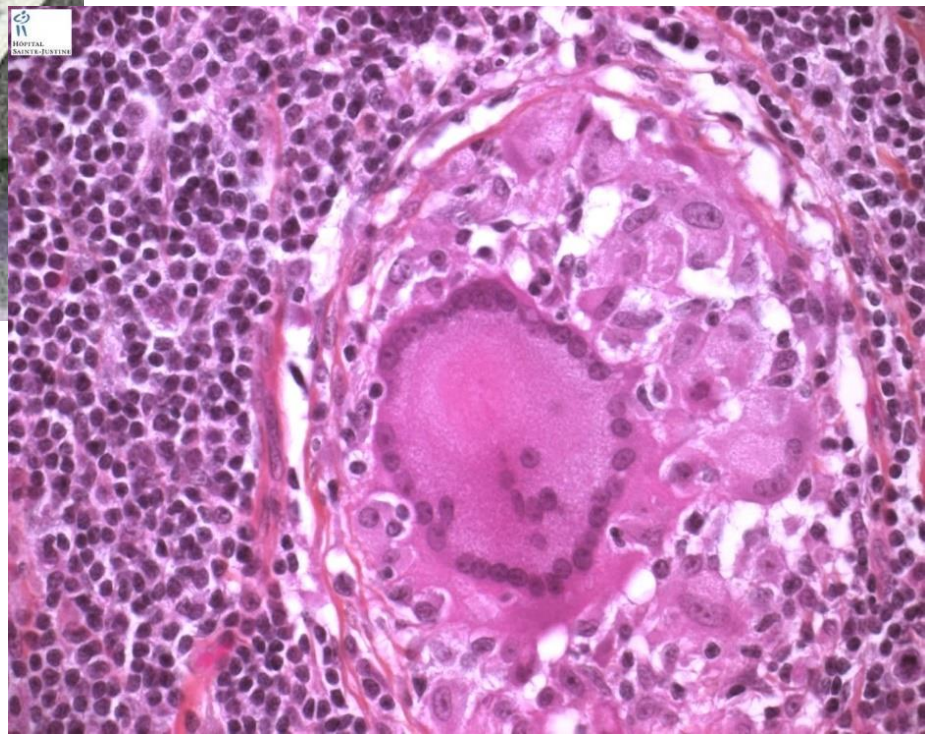


Langhans-type giant cell



Ziehl-Neelsen stain


ber einen Drüsenpolyp im Ileum.
Von Dr. Th. Langhans in Würzburg.
Berlin,
Druck und Verlag von Georg Reimer.
1867.



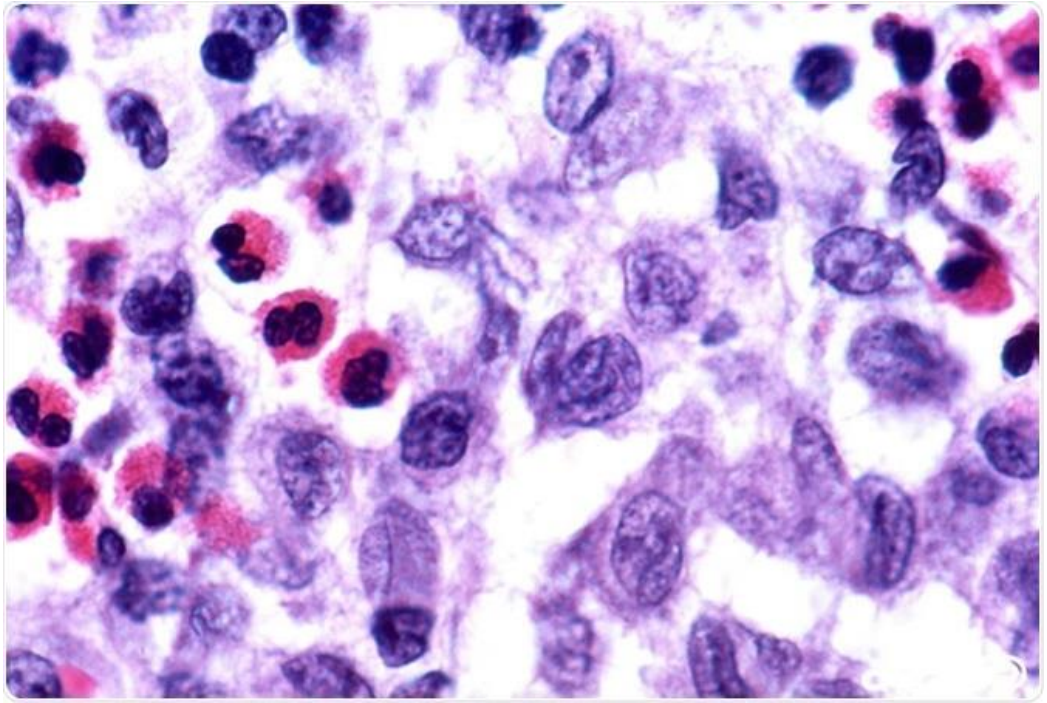
Paul LANGERHANS

1847 - 1888

Langerhans cell

Islets of Langerhans

eponymictionary



Cor pulmonale:

Hypertrophy and/or dilatation of the right side of the heart as a result of pulmonary hypertension (valve diseases, left heart problems excluded!!)

1. Acute cor pulmonale (massive pulmonary embolus, air embolism,
   amniotic fluid embolization

2. Chronic cor pulmonale

a. diseases of the lung parenchyma

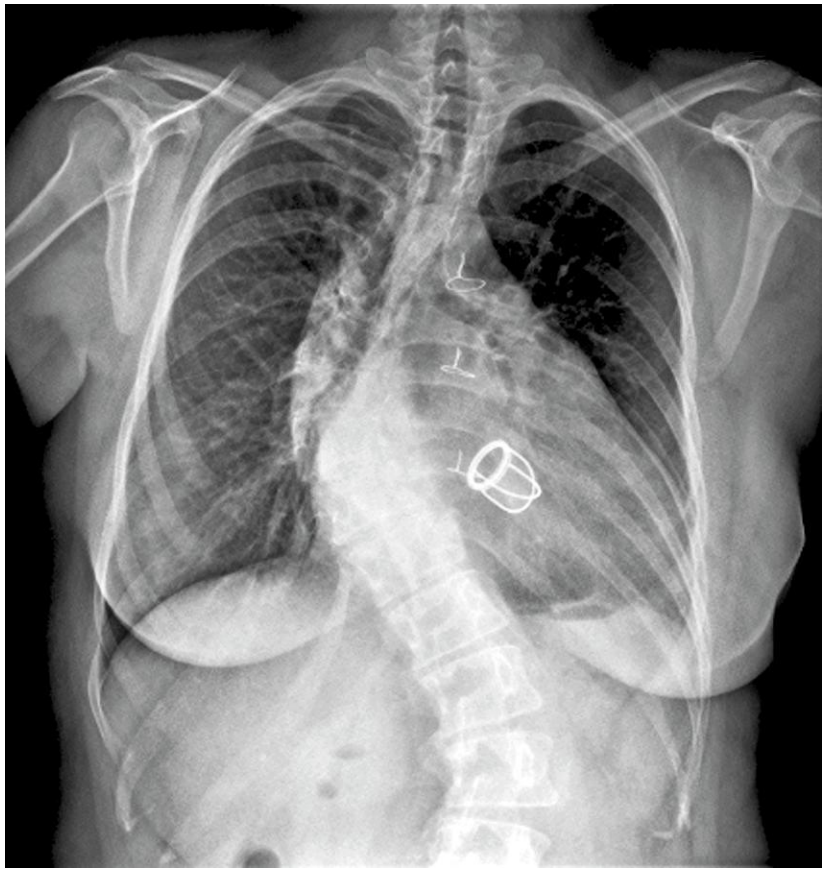
(chronic bronchitis, emphysema, bronchiectasis, lung fibrosis, sarcoidosis, massive tbc)

b. diseases of the chest wall impairing the respiration

(kyphoscoliosis, pleural callus, extreme obesity)

c. diseases of the lung vasculature

(multiplex pulmonary microembolisation)



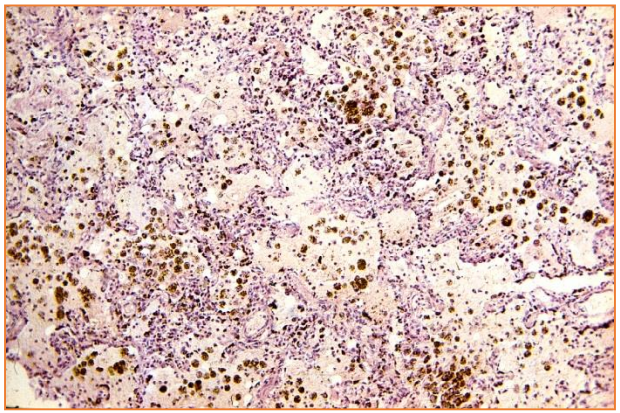

LIVERPOOL
YOU'LL NEVER
WALK ALONE

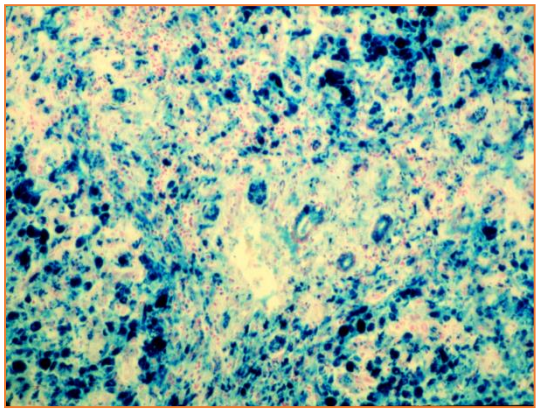

353 kg

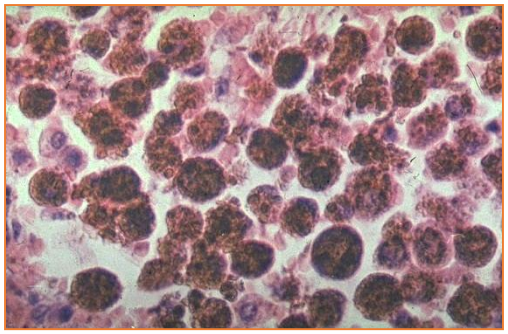
Consequences of chronic congestion

- organ enlargement (hepatomegaly, splenomegaly)
- induration (spleen, liver, lung, skin)
- atrophy (skin)
- necrosis (bowel, testicle)
- edema

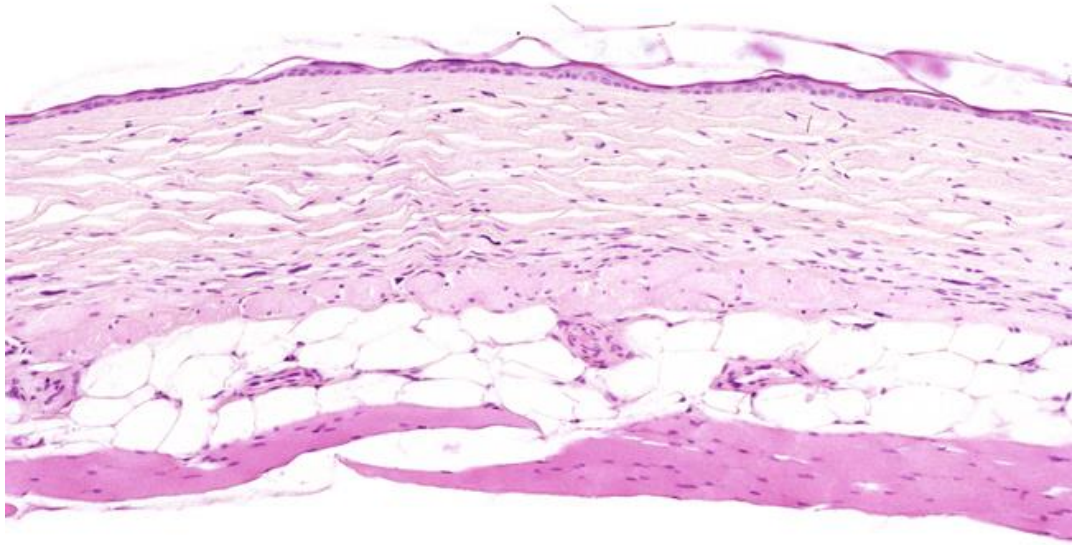
brown Induration of lungs





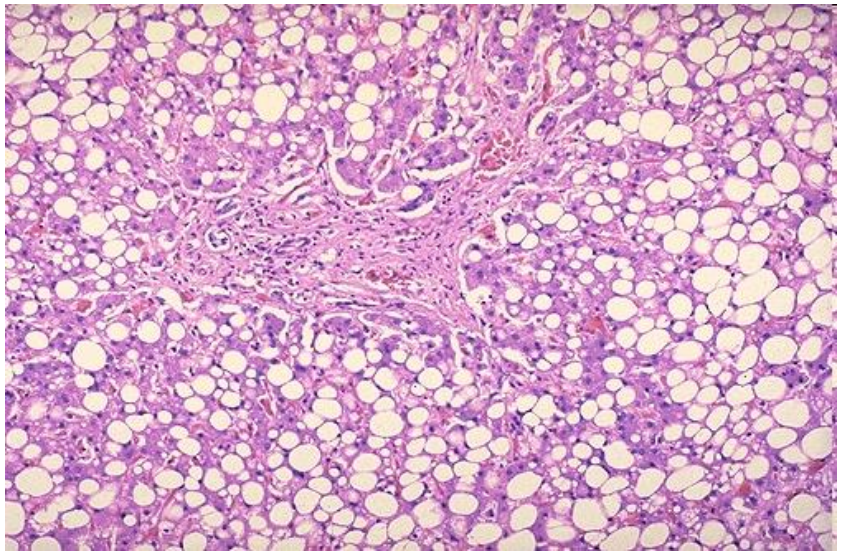


„heart failure cells”



4

postthrombotic syndrome



fatty degeneration

crural ulcer


ZetRix
hyperemia!