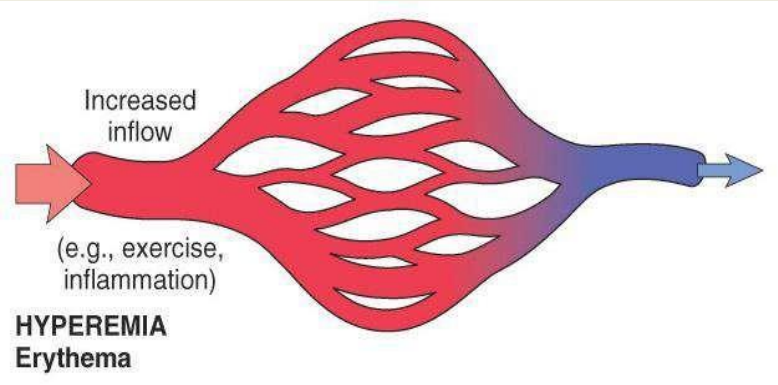
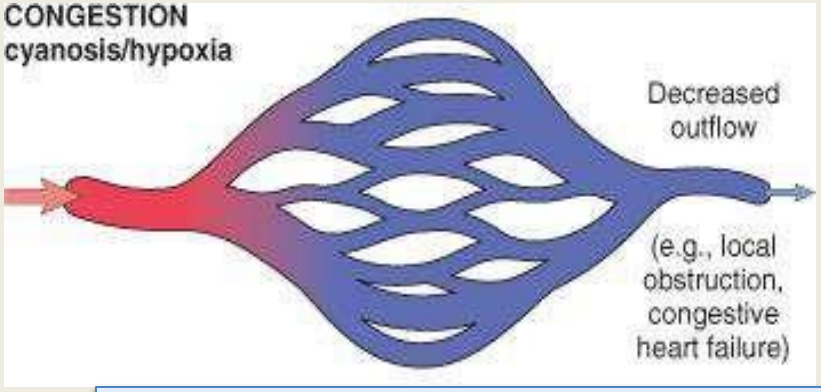
MJF COLLEGE OF VETERINARY AND ANIMAL SCIENCE, CHOMU, JAIPUR

DEPARTMENT OF VETERINARY PATHOLOGY

HYPEREMIA AND CONGESTION





ACTIVE HYPEREMIA

- Increased blood going into tissue
- Oxygenated (red)

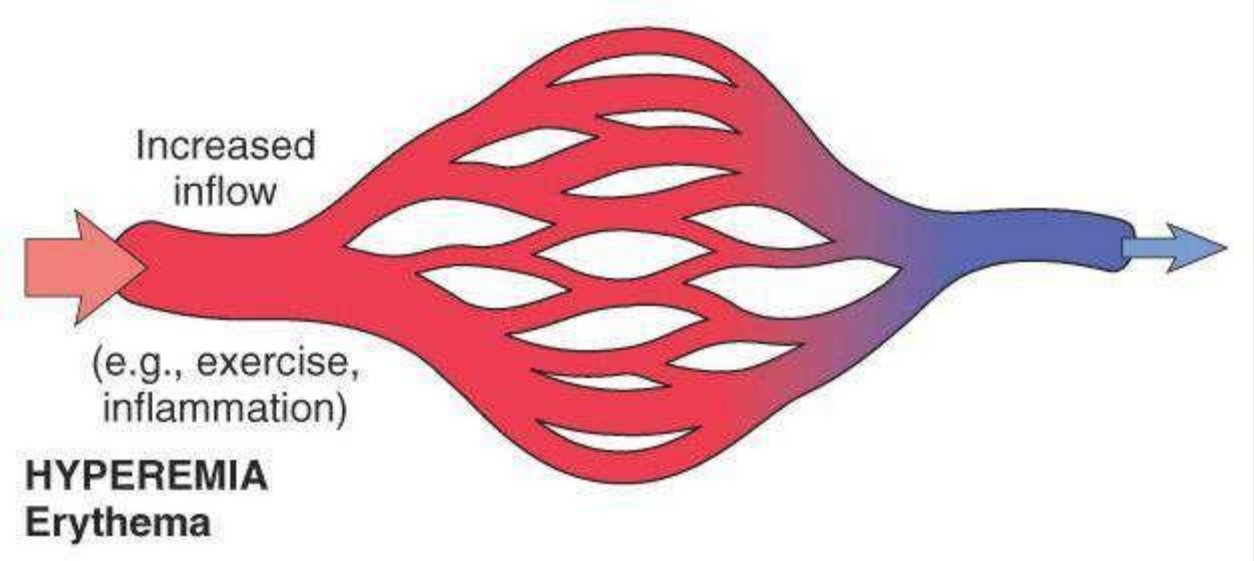
CONGESTION

- Decreased outflow of blood
- Not oxygenated (blue)

HYPEREMIA

- Increased amount of blood in the arterial side of the vascular system
- Acute process,
  - Chronic hyperemia not occur
- Generalized or local

Appearance:



Acute Local Active Hyperemia

- increased amount of blood in the arterial system within a local area (leg, stomach, or lung)

Etiology

Physiologic
- Digestion
- Exercise
- Dissipate heat
- Neurovascul

ar (blushing)

Pathological
- Inflammation
- **"Hyperemia of Inflammation"**
- Often see associated edema

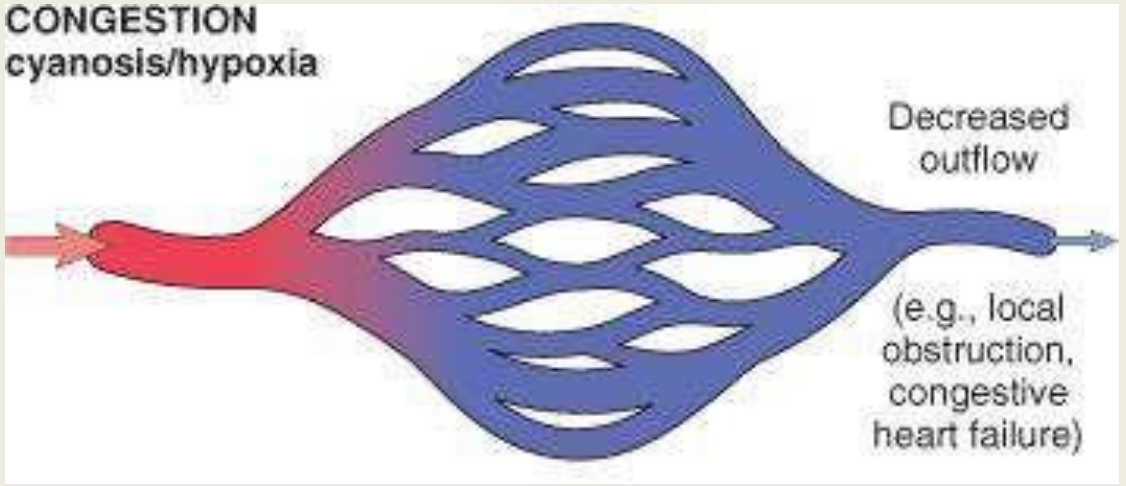
Congestion (Passive Hyperaemia)

- Passive engorgement of vascular beds due to decreased blood outflow
- **Duration:** Acute or Chronic
- **Extent:** Generalized or local

Appearance

- Tissue dark red to blue

/ black (cyanotic) due poorly oxygenated Hb



Acute General Passive Hyperaemia

- Sudden obstruction to the flow of blood in the heart or lungs.
- Short period of time

Etiology:

- Sudden death due to heart failure due to myocardial infarction
- Euthanasia with barbiturates – Dilation of sinusoids
- Fluid/blood/pus in pericardium or thorax - presses on the heart - prevents a normal diastole
- Blood accumulates in lung, spleen and liver

Chronic General Passive Hyperaemia

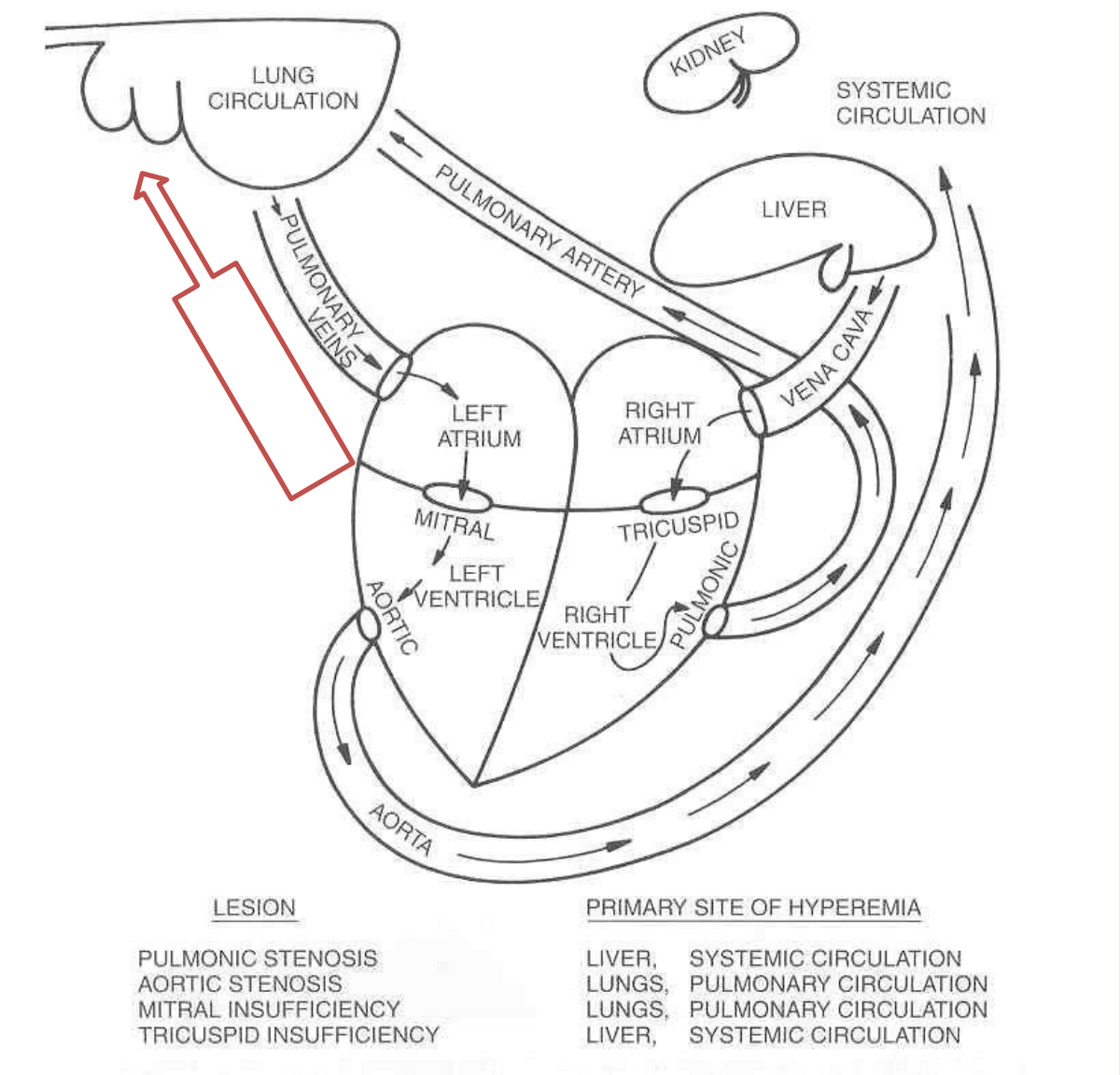
- Persists for a long period of time
- Permanent alterations (atrophy and fibrosis)

Etiology

- Often associated with pathology in heart or lungs
- Stenosis or defect of a valvular opening
- Traumatic pericarditis in cattle

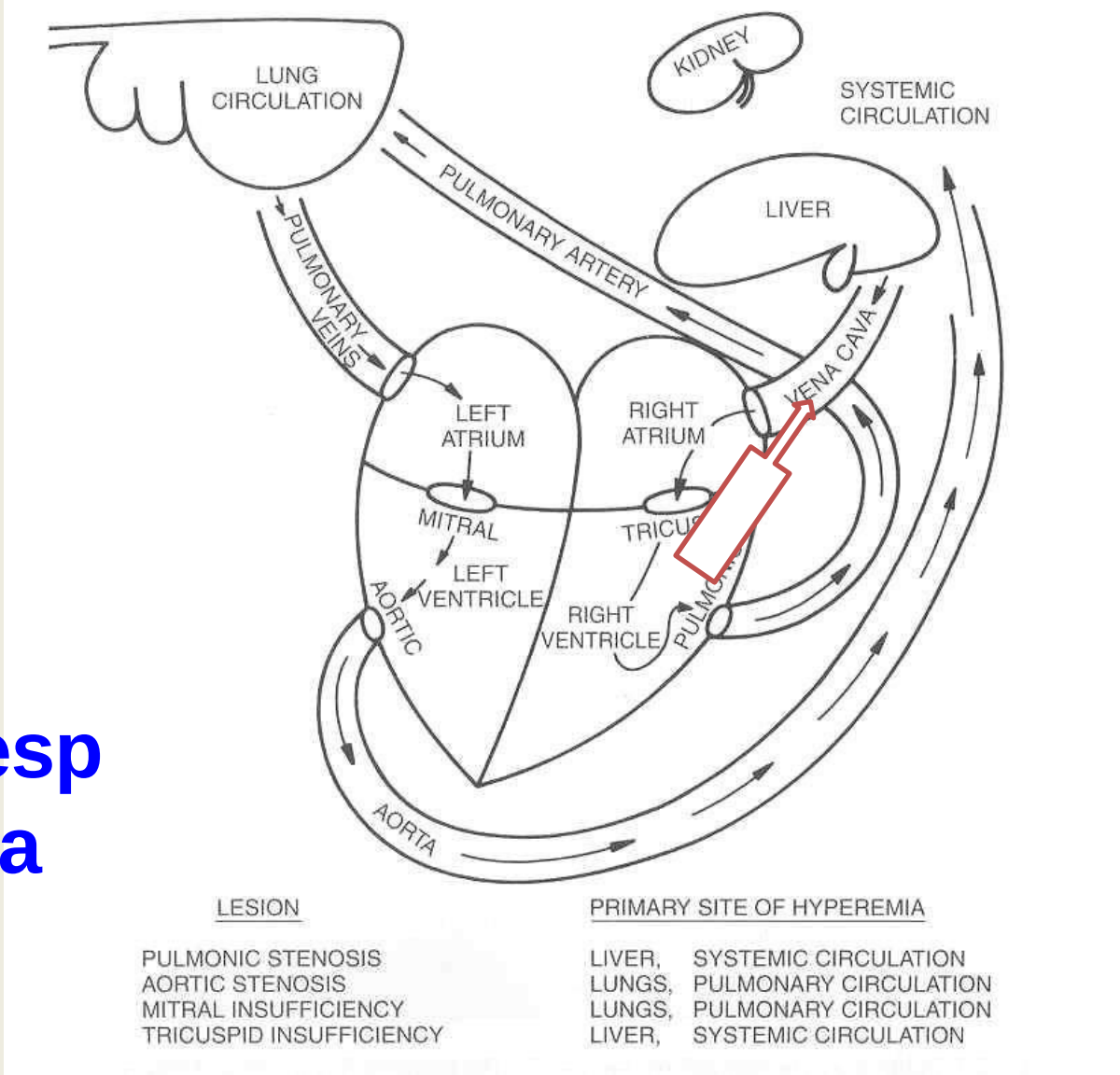
Left-sided Heart Failure

- congestion (& edema) of lungs



Right-sided Heart Failure

- systemic congestion (esp liver) & edema (eg ascites)



Chronic Local Passive Hyperaemia

- Amount of blood that persists for a long time
- Permanent tissue changes (atrophy and fibrosis) in the area

Etiology:

1. External pressure: Enlarging neoplasms, lymph nodes, and abscesses.
2. Obstruction within a vein, such as caused by a thrombus.

HYPEREMIA AND CONGESTION **EDEMA**

HEMORRHAGE

HEMOSTASIS

THROMBOSIS

EMBOLISM

INFRACTION

SHOCK

Edema

- **Definition**

  abnormal (excess) accumulation fluid in interstitial tissue spaces or body cavities

Gross Appearance of Edema

- organs wet (gelatinous) and heavy.
- organs swollen and fluid may weep from cut surface
- may be yellow

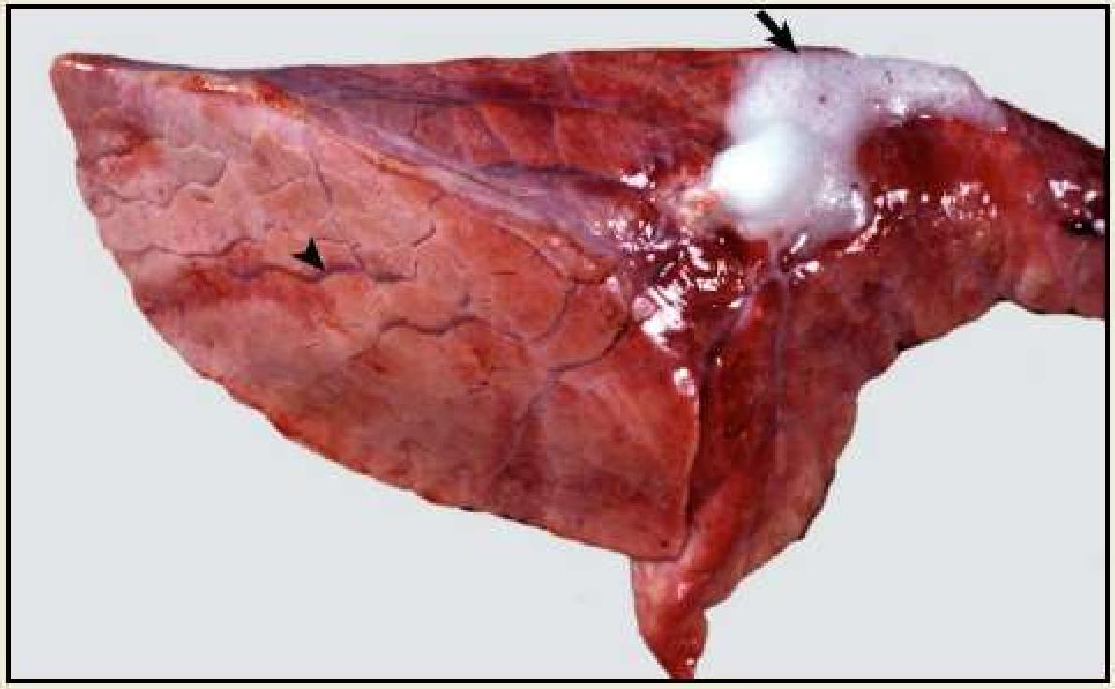
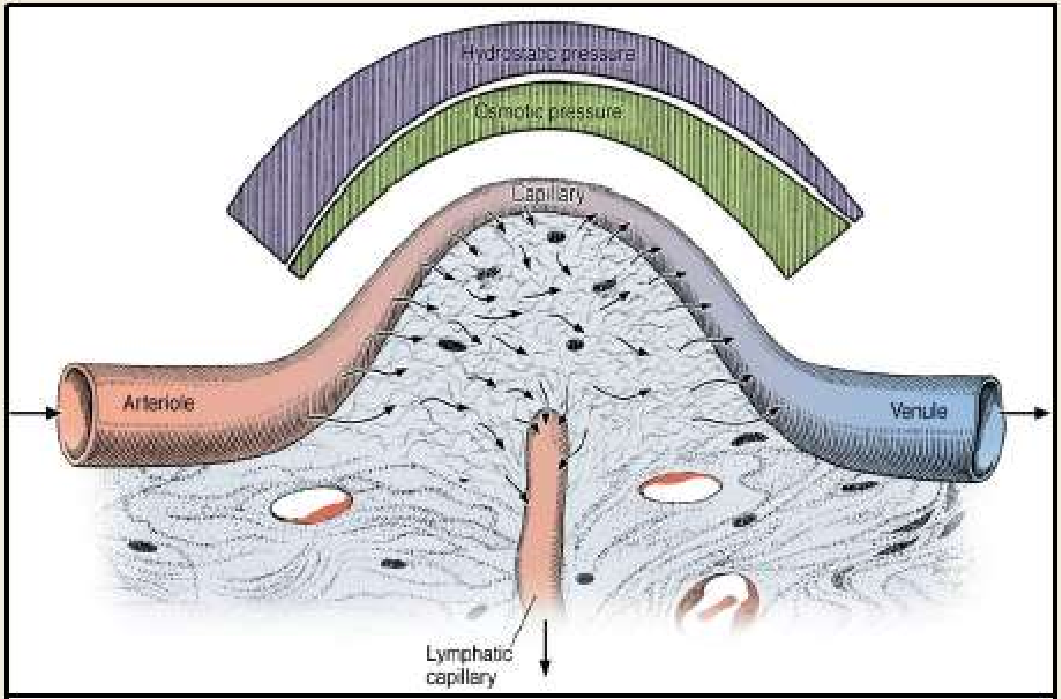


Fig. 2-10 (McGavin) Pulmonary edema, lung, pig. The lung failed to collapse and has a firm rubbery texture attributable to edema fluid in alveoli and the interstitium. Note the prominent interlobular septa caused by edema (arrowhead) and the frothy edema fluid exuding from the bronchus (arrow).

Starlings Equation

- hydrostatic pressure in the vascular system (+ interstitial osmotic pressure) moves fluid out of the vascular system

- the osmotic pressure of the plasma proteins (+ some tissue hydrostatic pressure) contains the fluid within the vascular system



	ARTERIOLAR	VENULAR
PLASMA HYDROSTATIC PRESSURE	* 30 mm Hg	* 17 mm Hg
TISSUE HYDROSTATIC PRESSURE	8 mm Hg	8 mm Hg
PLASMA COLLOIDAL-OSMOTIC PRESSURE	25 mm Hg	25 mm Hg
TISSUE COLLOIDAL-OSMOTIC PRESSURE	10 mm Hg	10 mm Hg
	(30 – 8) — (25 – 10) = 7 mm Hg **Net filtration pressure**	(17 – 8) — (25 – 10) = 6 mm Hg **Net absorption pressure**

Hydrostatic pressure at the arterial end of the capillary	=	45 mm Hg (mercury)
Osmotic pressure at the arterial end of the capillary	=	30 mm Hg
		15 mm rate of fluid flow into the tissues
Osmotic pressure at the venular end of the capillary	=	30 mm Hg
Hydrostatic pressure at the venular end of the capillary	=	15 mm Hg
		15 mm rate of fluid flow into the vein

Mechanisms of

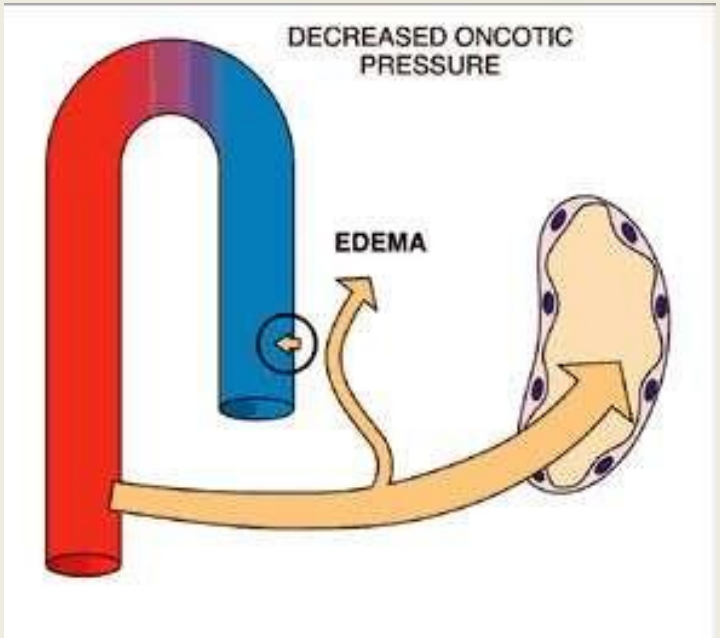
1. Increased hydrostatic pressure

Etiology

- **Portal hypertension** (e.g., right-side heart failure, hepatic fibrosis)
- **Pulmonary hypertension** (e.g., left-side heart failure, high altitude disease)
- **Localized venous obstruction** (e.g., gastric dilation and volvulus, intestinal volvulus and torsion, uterine torsion or prolapse, venous thrombosis)
- **Fluid overload** (e.g., iatrogenic, sodium retention with renal disease)
- **Hyperemia** (e.g., inflammation, physiologic)

Mechanisms of

2. Decreased plasma colloidal osmotic pressure



Hydrostatic pressure at the arterial end	=	45 mm Hg
Osmotic pressure at the arterial end	=	20 mm Hg
		25 mm Hg rate of fluid flow into the tissues
Osmotic pressure at the venular end	=	20 mm Hg
Hydrostatic pressure at the venular end	=	15 mm Hg
		5 mm Hg rate of fluid flow into the vein

Net result: 25 mm Hg minus 5 mm Hg = 20 mm Hg. **Therefore, at the rate of 20 mm Hg fluid accumulates in the tissues. This type of oedema is severe.**

Causes of Hypoalbuminemia

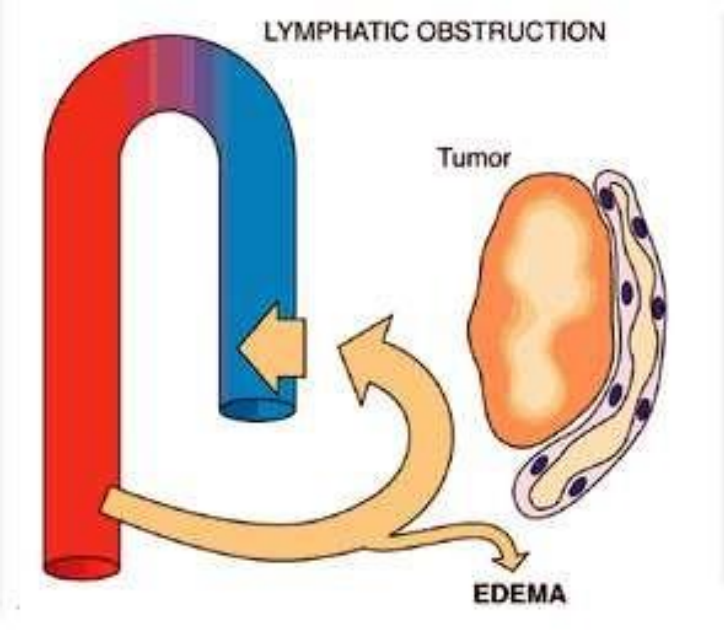
Proteins not absorbed: Starvation, Malabsorption

Proteins not produced : Liver disease

Proteins lost: Glomerular disease, Intestinal damage

Mechanisms of

3. Lymphatic obstruction



Hydrostatic pressure at the arterial end	=	45 mm Hg
Osmotic pressure at the arterial end	=	25 mm Hg
		20 mm Hg rate of fluid flow into the tissues
Osmotic pressure at the venular end	=	25 mm Hg
Hydrostatic pressure at the venular end	=	15 mm Hg
		10 mm Hg rate of fluid flow into the vein

Net result: 20 mm Hg minus 10 mm Hg = 10 mm Hg. **Therefore,** at the rate of 10 mm Hg fluid accumulates in the tissues.

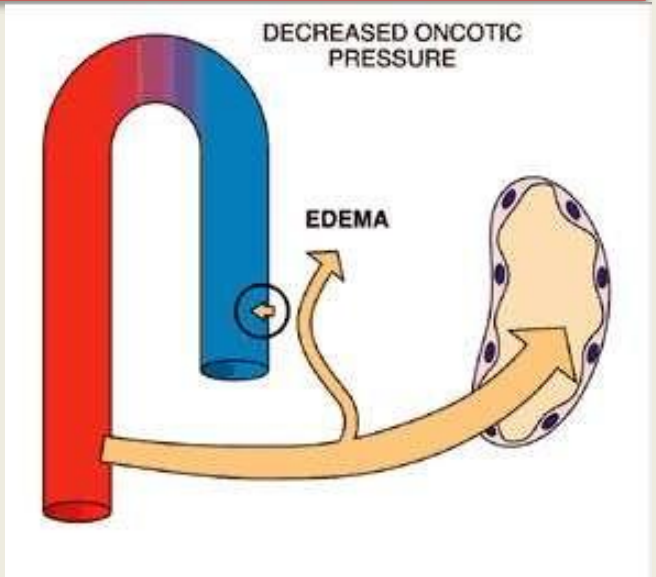
Causes of Lymphatic Obstruction (Lymphoedema) Damage / obstruction of lymphatics

- **Surgery / trauma (fibrosis)**
- **Neoplasm**
- **Inflammation (lymphangitis)**

Mechanisms of

4. Sodium and Water Retention

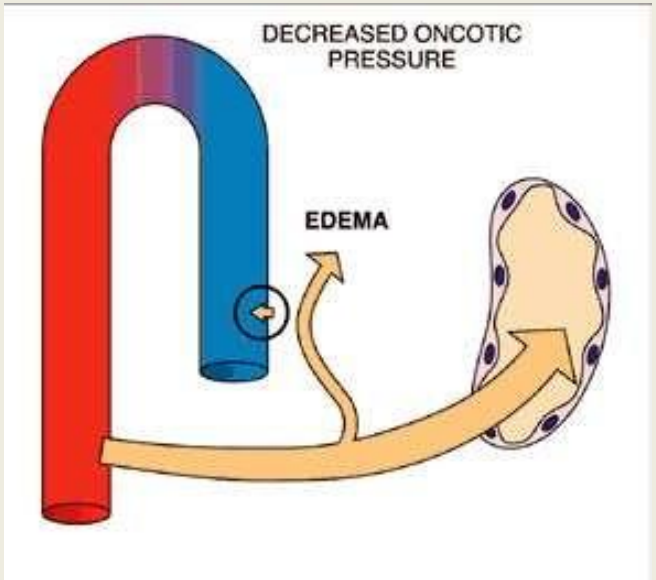
- Excessive retention of salt and water
- Expansion of the intravascular volume
- Increasing hydrostatic pressure
- Reducing plasma osmotic pressure



Cause

compromise renal function

Glomerulonephritis and *acute renal failure*



Edema of

1. **Increased hydrostatic pressure (venous)**
2. **Decreased plasma colloidal osmotic pressure**
3. **Lymphatic obstruction**
4. **Sodium and Water Retention**

Fluid Characteristics

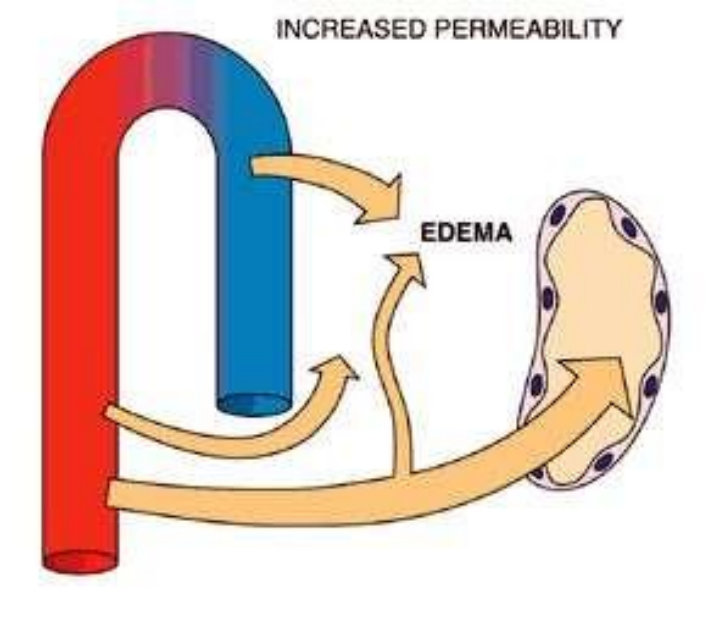
– **“Protein poor” (“Non-inflammatory edema”)**

–**Transudate**

- Low protein content < 30g/L
- Low specific gravity < 1.025
- Few nucleated cells <1.5 $X10^9$/L

Mechanisms of

5. Increased Vascular Permeability / Endothelial damage



Localized edema

Causes

- **Inflammatory / immune reactions**
- **"inflammatory edema"**
- **Viruses**
- **Bacteria**
- **Rickettsia (e.g., Cowdria)**
- **Type III hypersensitivity**
- **Toxins**

Fluid Characteristics

Protein-rich Exudate

- High protein content > 30g/L
- Specific gravity > 1.025
- Total nucleated cells > 7 X10^9/L