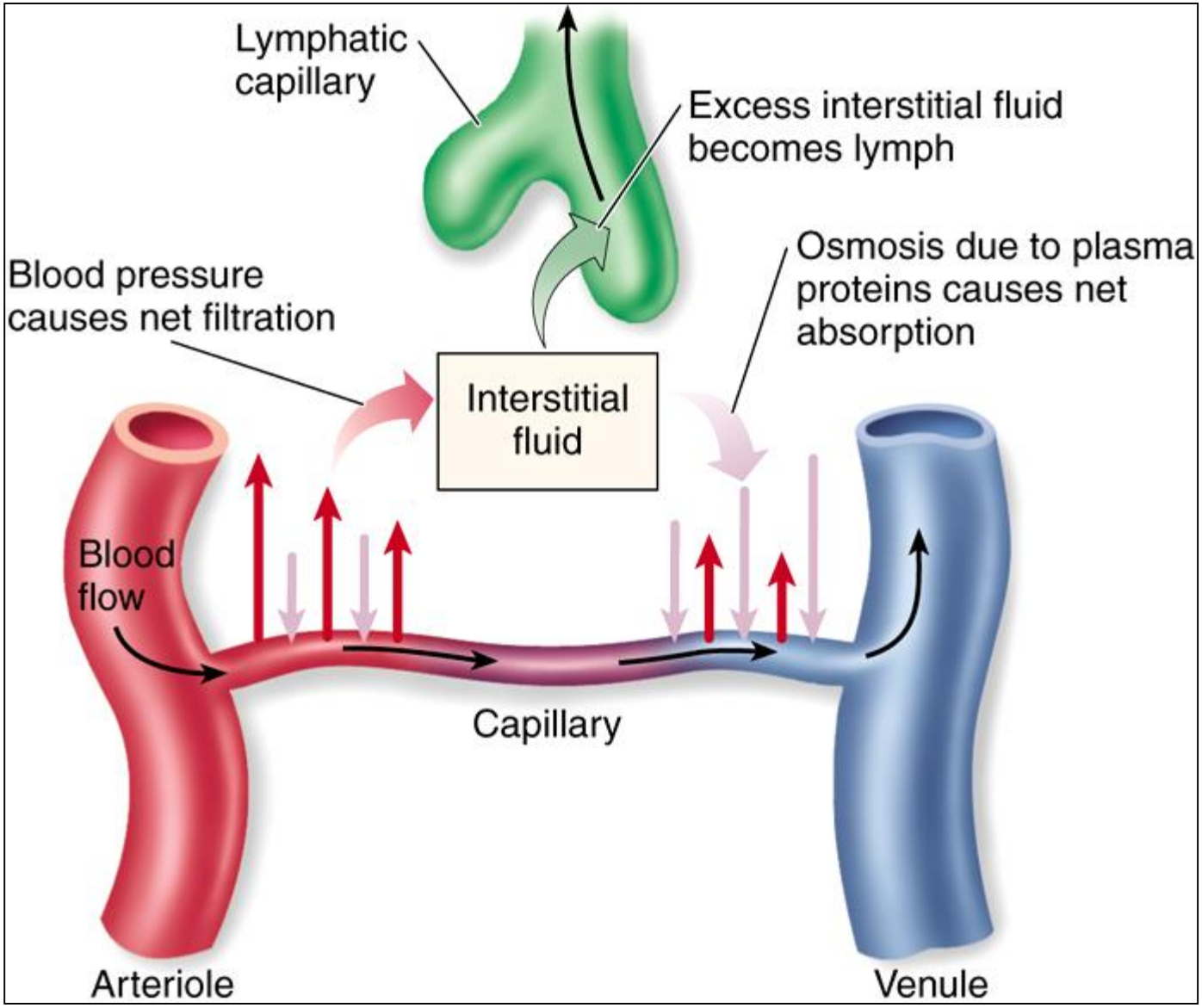
HEMODYNAMIC DISORDERS THROMBOSIS AND SHOCK

Robbins, Chapter 4

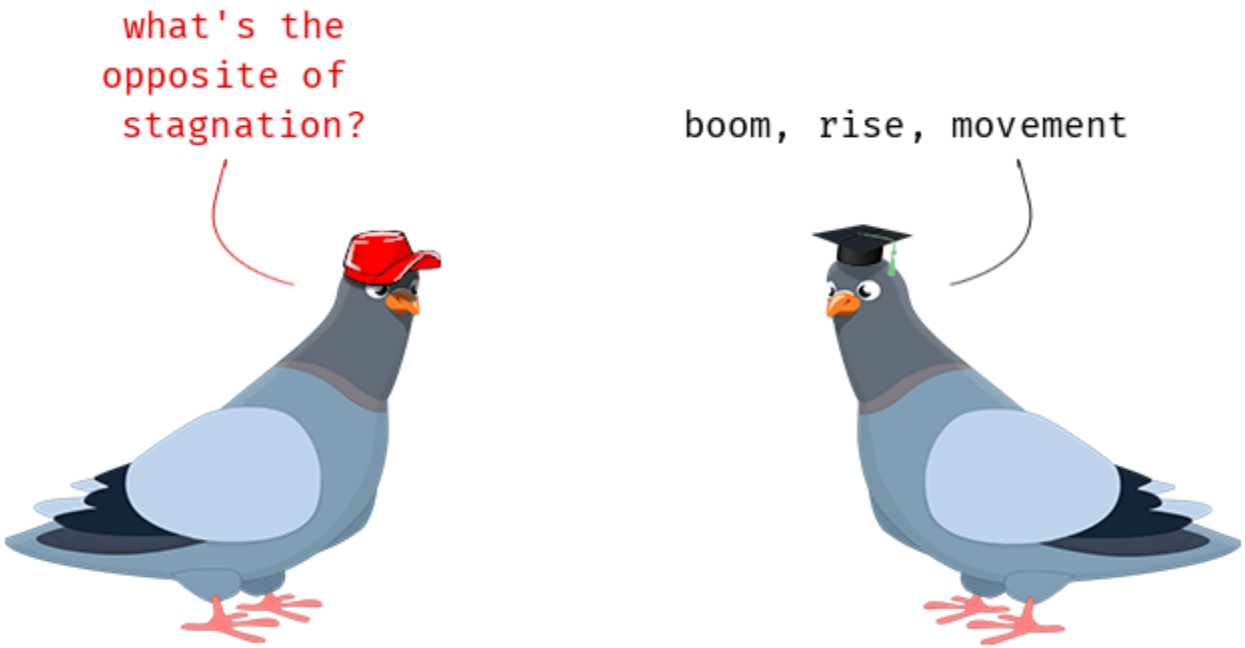
EDEMA (WATER AND ELECTROLYTE IMBALANCES)

Árpád Szállási

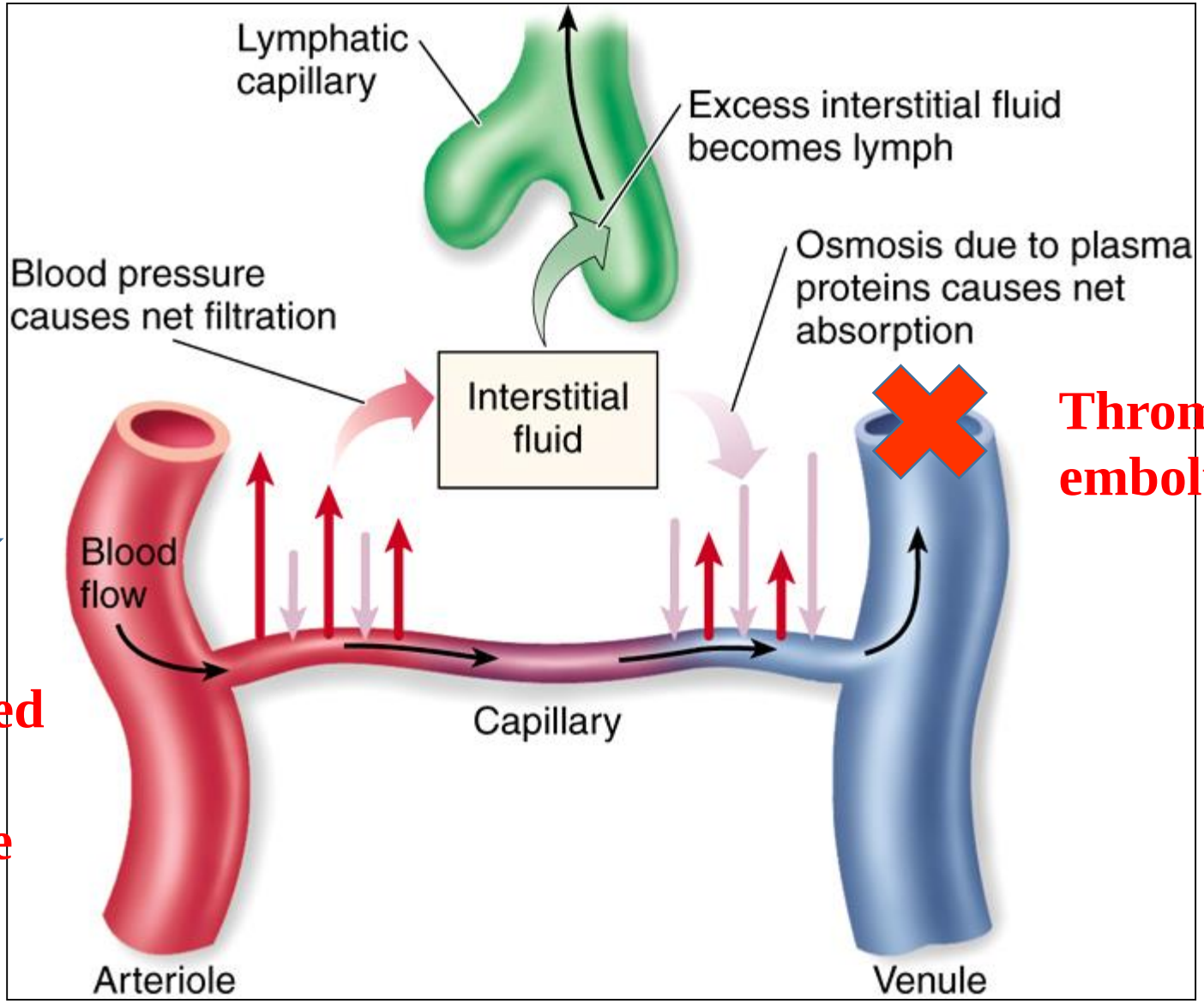
MICROCIRCULATION UNIT



STAGNATION = „THE STATE OF NOT MOVING OR FLOWING"

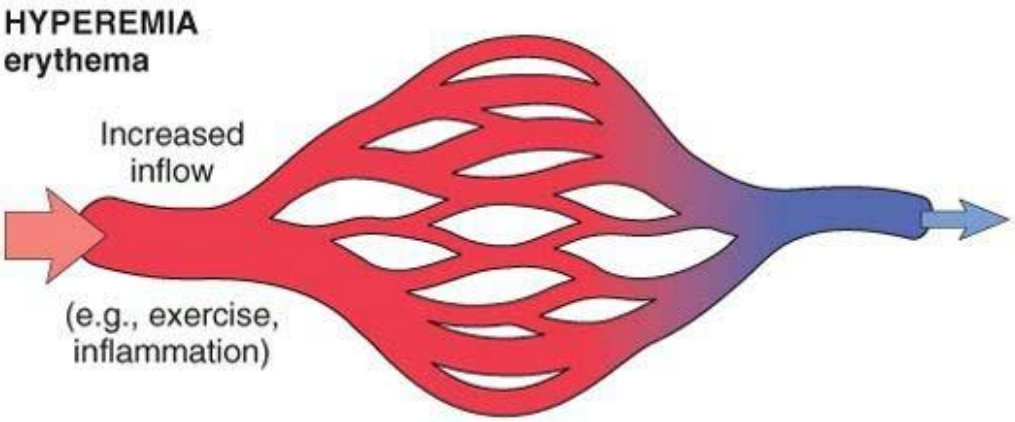


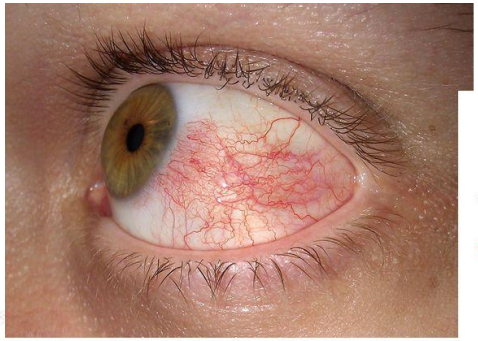
STAGNATION/CONGESTION



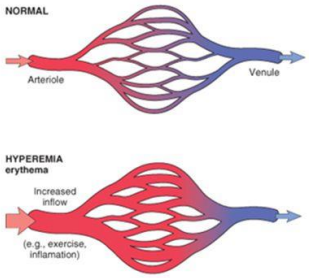
decreased blood pressure

Thrombus embolus





HYPEREMIA



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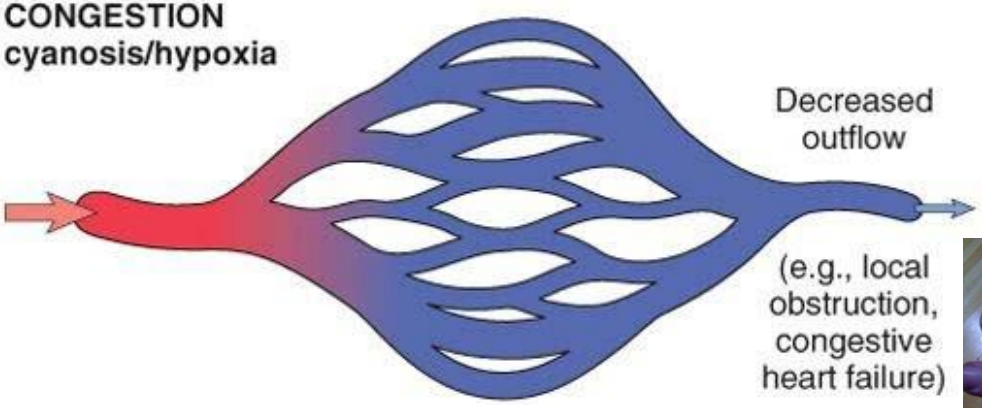
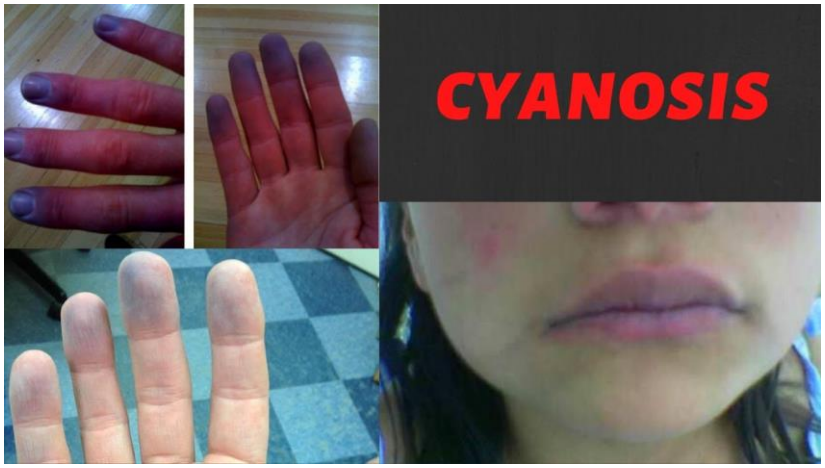
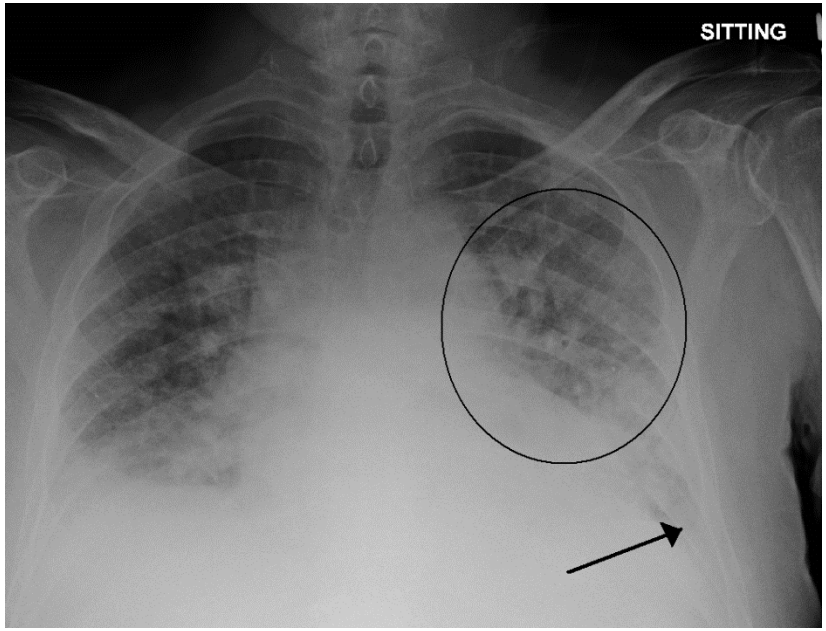
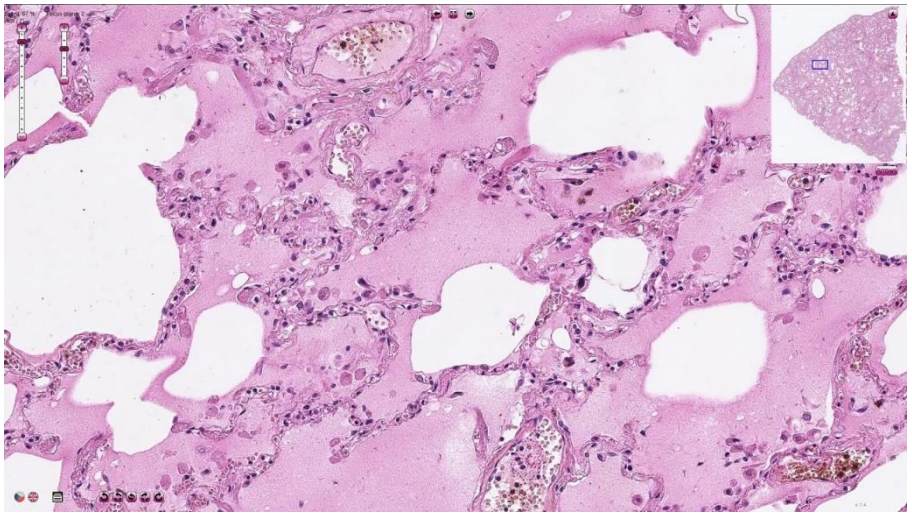
http://upload.wikimedia.org/wikipedia/commons/thumb/5/58/Hyperemia_conjunctiva.jpg/1024px-Hyperemia_conjunctiva.jpg





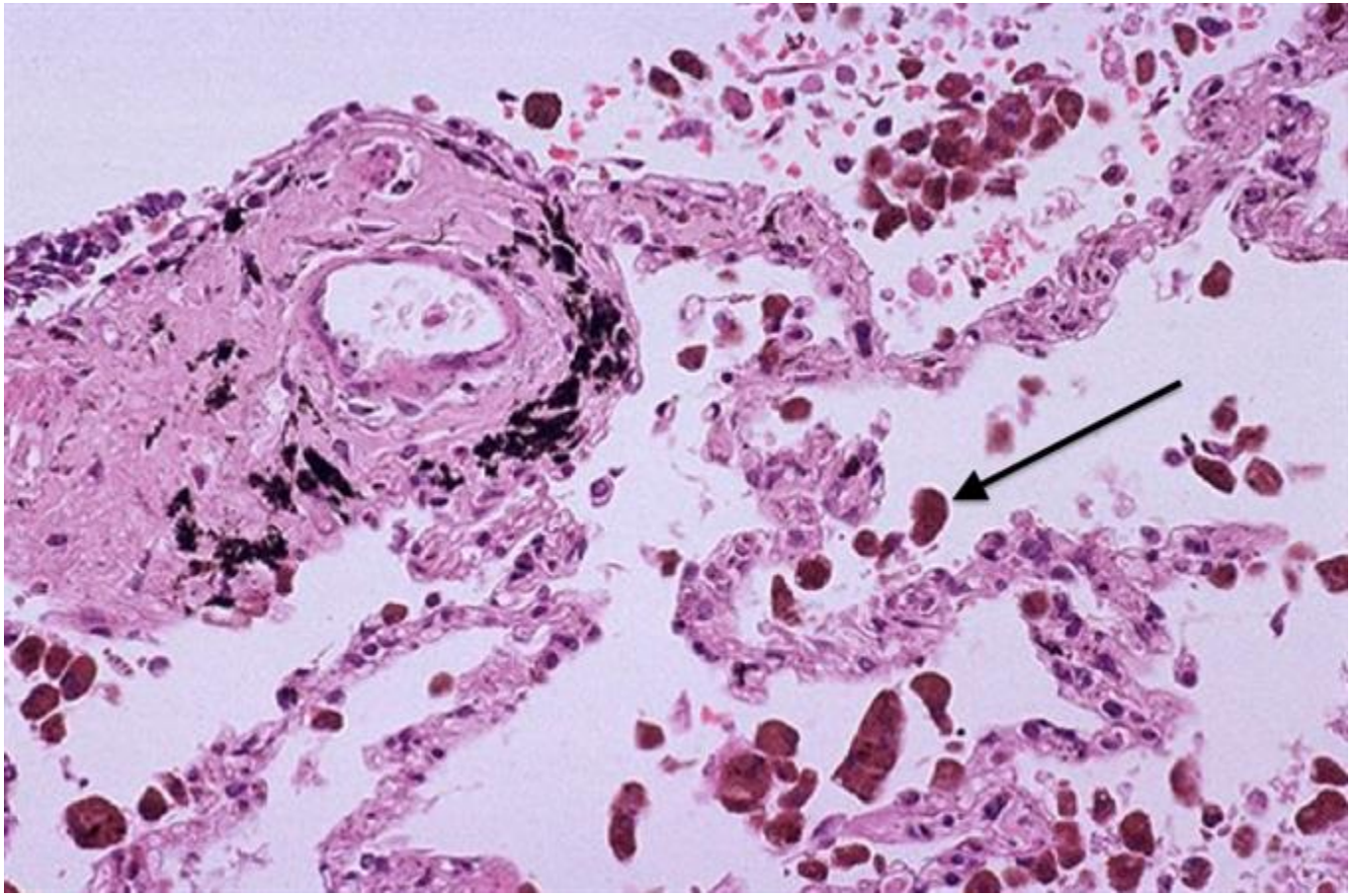
Pulmonary congestion



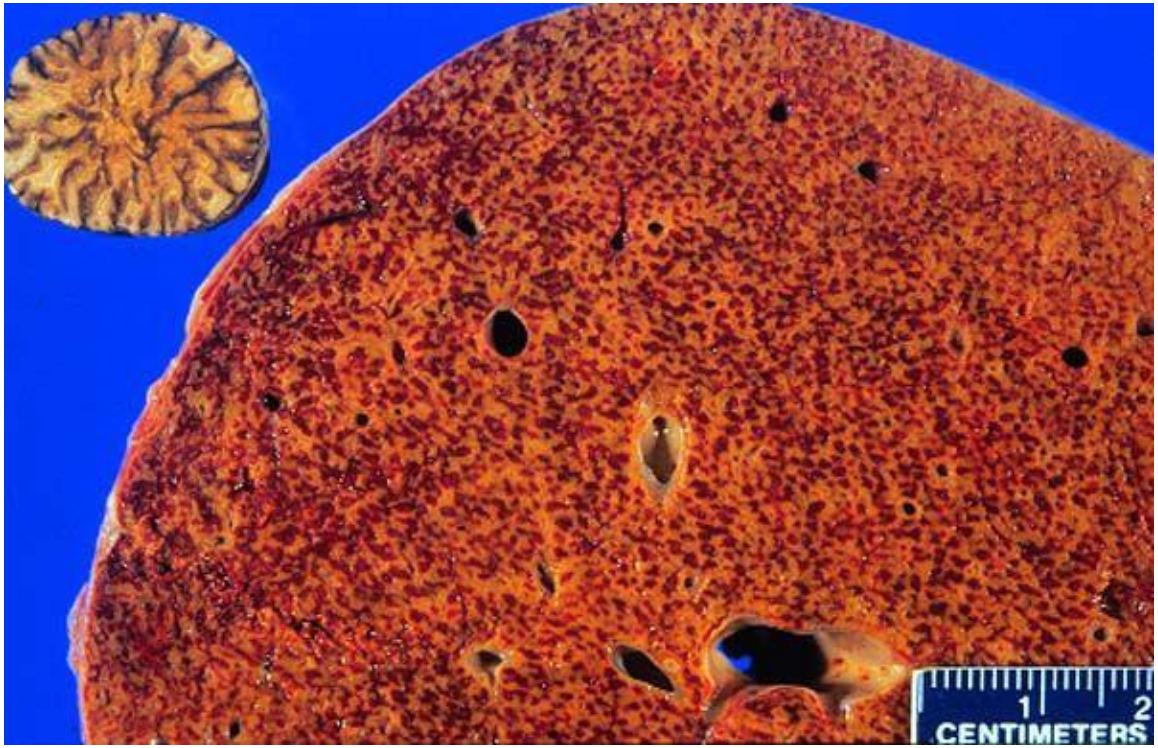


Pulmonary edema as complication of routine dental treatments performed under general anesthesia

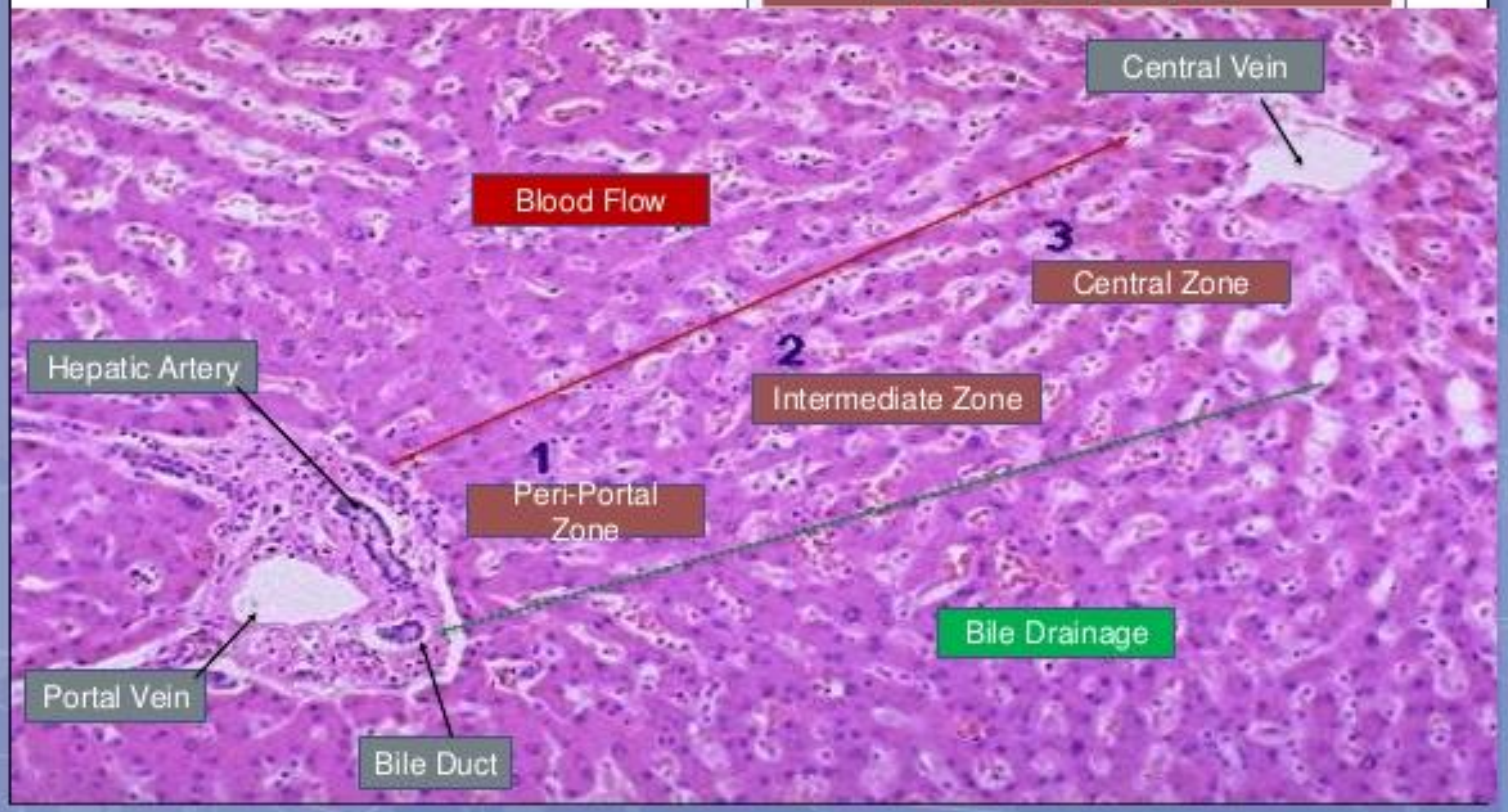
„heart failure cells”



Chronic hepatic congestion, „nutmeg liver”



Zones of Liver

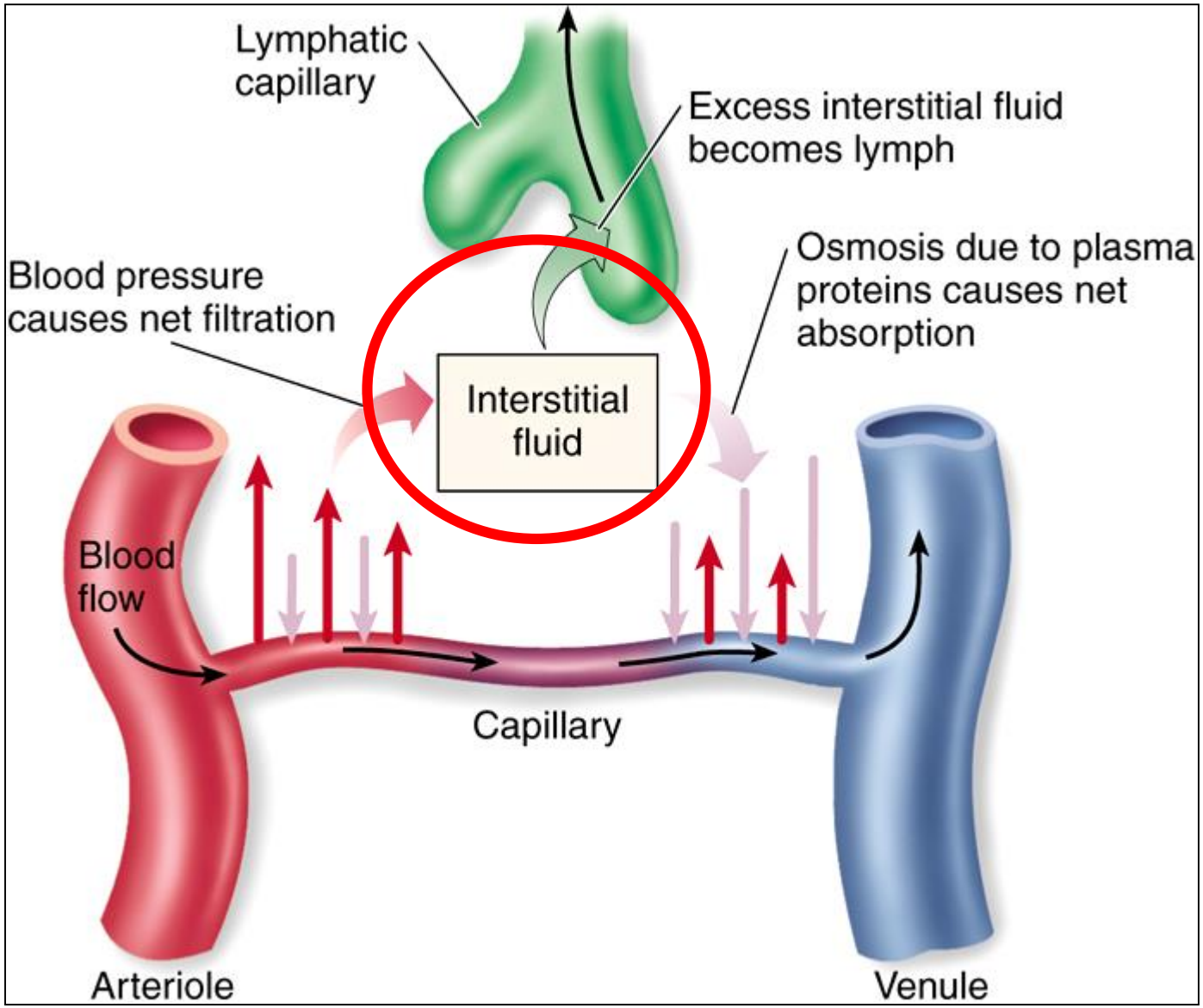


Nutmeg Liver:

- Chronic Passive Congestion – Heart failure.
- Central zone (Zone-3) – congestion and necrosis.
- Hemorrhage – RBCs in zone-3 - Mottled appearance (nutmeg).
- Symptoms similar to chronic hepatitis, Ascites, distende d abdomen, ankle edema, Hepatic encephalopathy, confusion.

οἴδημα

Edema = accumulated/increased interstitial fluid



EDEMA

Accumulated, extravasal fluid with low protein content (transudate)

- in the interstitial tissues
- freely in body cavities

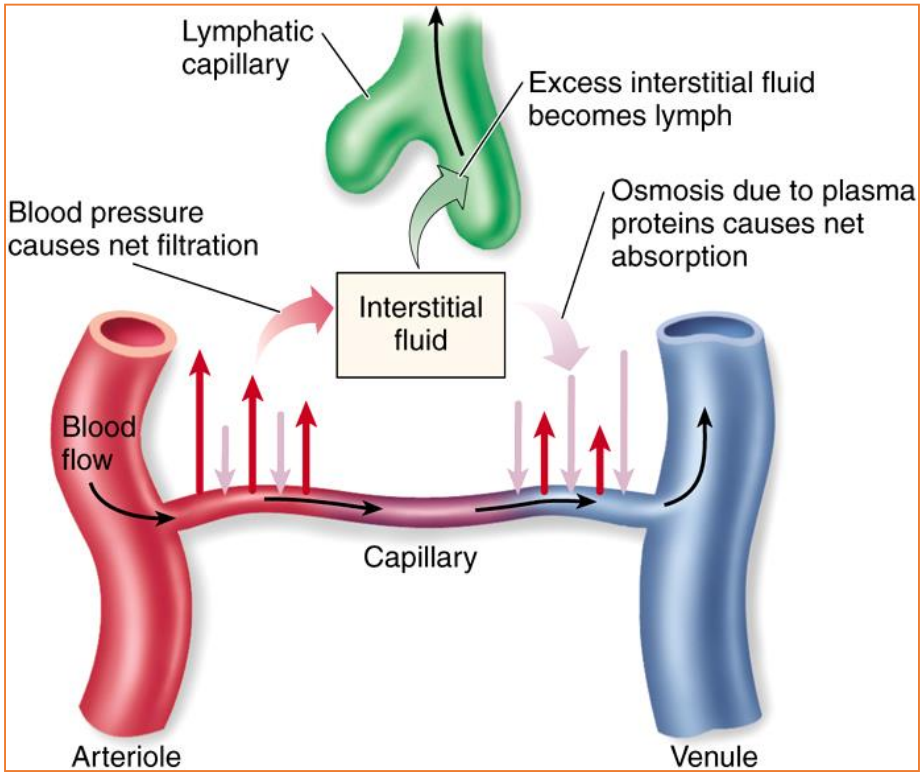
Special gravity: < 1.012

Origin: disturbed balance in the microcircular unit

Determinants:

- intravasal hydrostatic pressure
- colloidosmotic pressure in the vessels
- tissue colloidosmotic pressure
- lymphatic circulation

EDEMA FORMATION



- decreased colloidosmotic pressure in the blood
- increased capillary permeability
- increased hydrostatic pressure in the venous part
- lymphatic insufficiency
- sodium and water retention

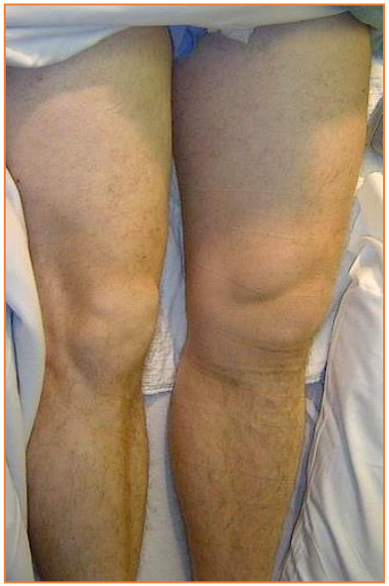
EDEMA

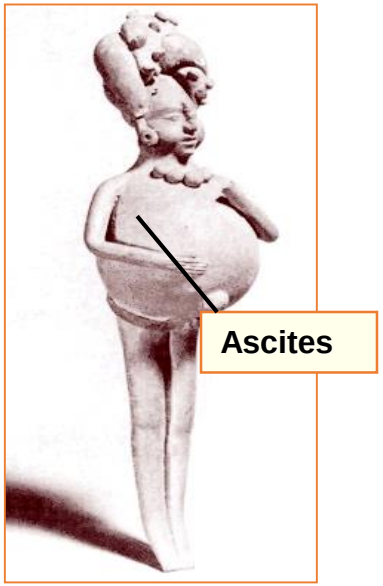
LOCALIZED

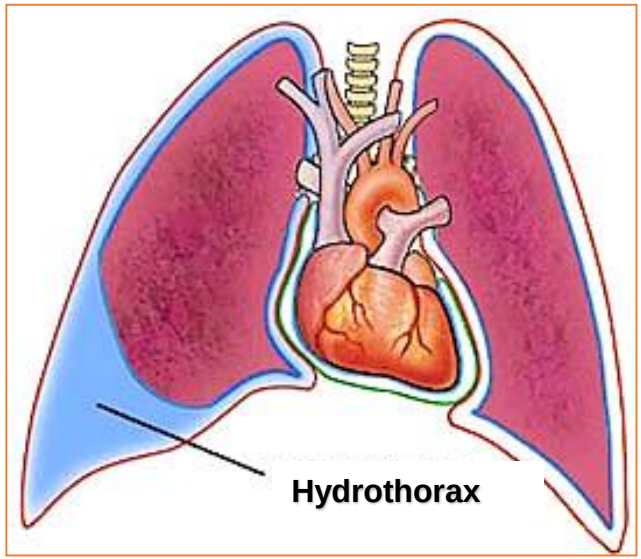
- disturbed venous outflow
- disturbed lymphatic circulation
- inflammation
- allergy
- hypoxia
- cerebral space-occupying lesions

GENERALIZED

- hypalbuminemia
- right sided heart failure
- Rh (ABO) incompatibility, infection







LOCALIZED EDEMA

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- disturbed lymphatic circulation
- inflammation
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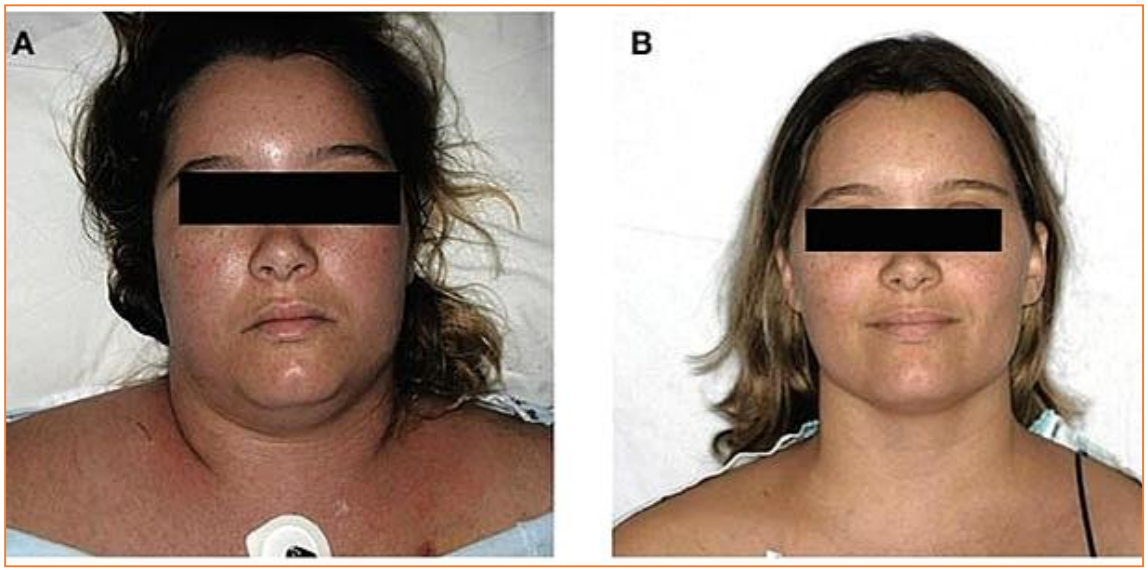
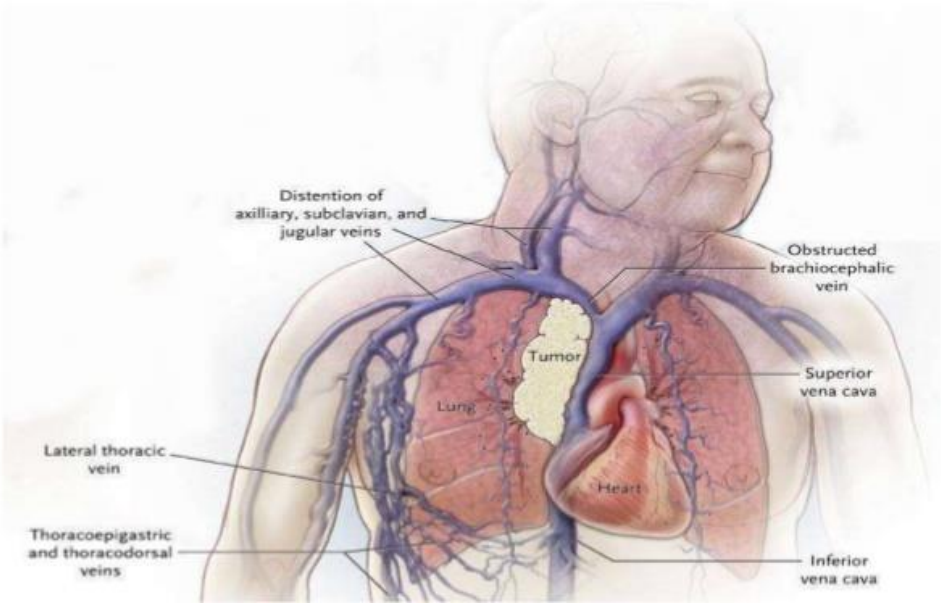
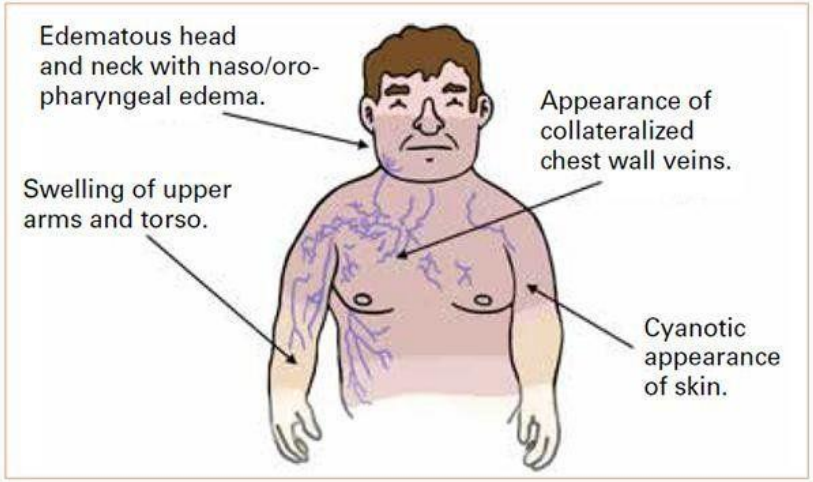
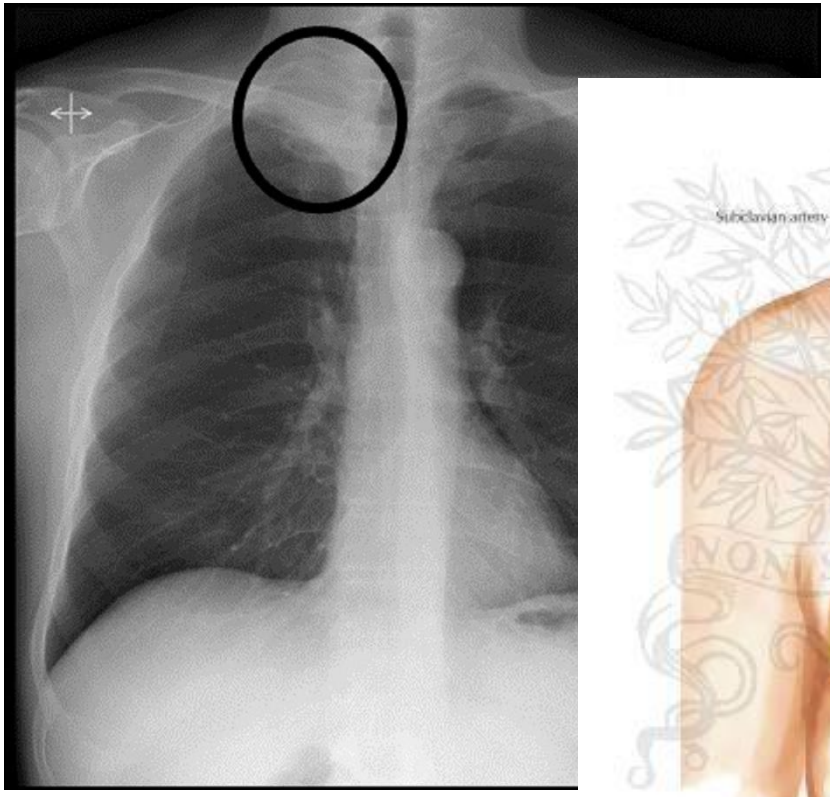


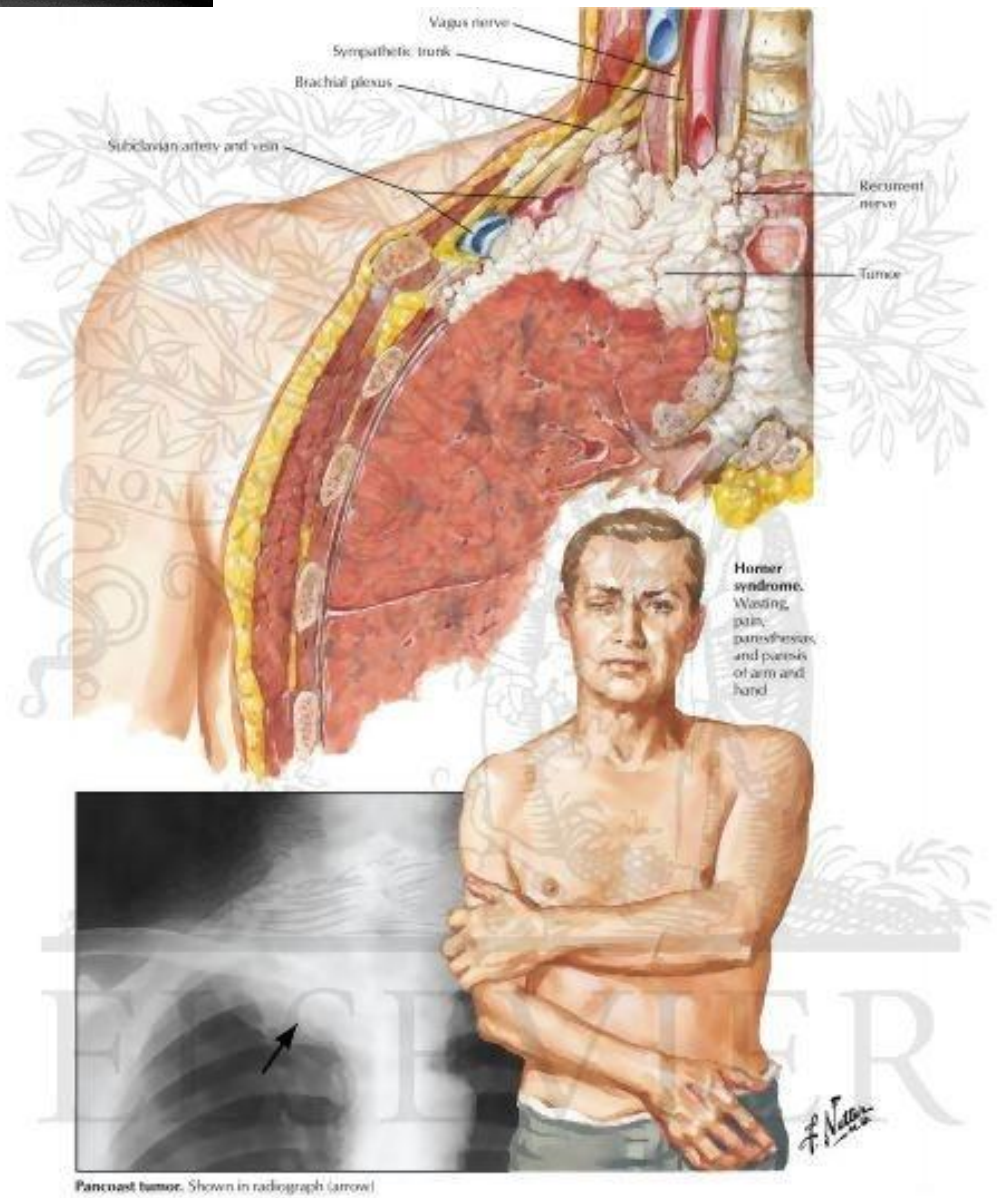
superior vena cava syndrome

SUPERIOR VENA CAVA SYNDROME





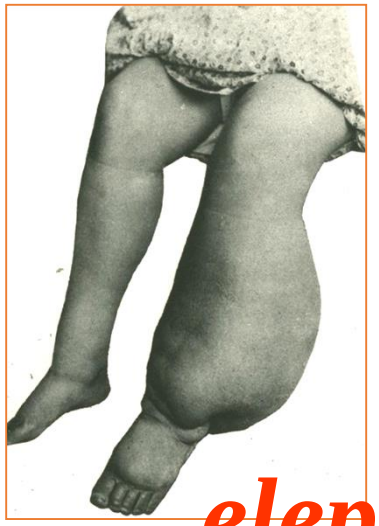




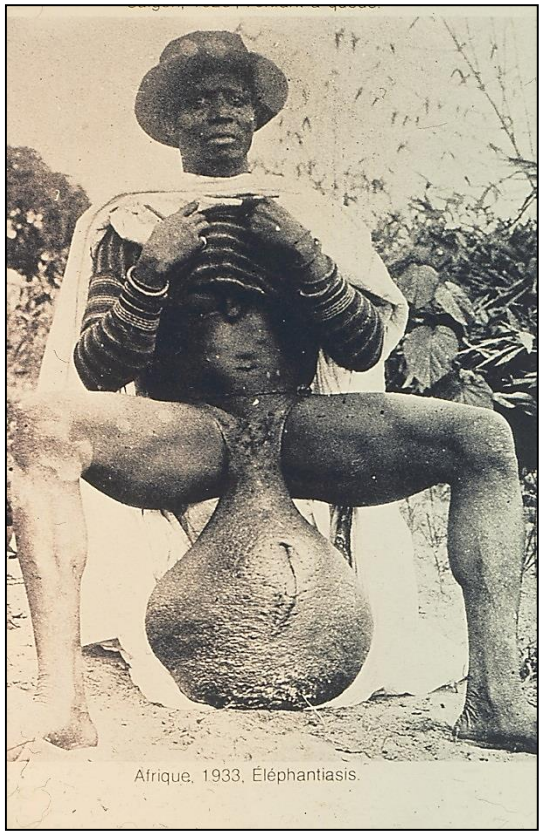
Pancoast tumor. Shown in radiograph (arrow)

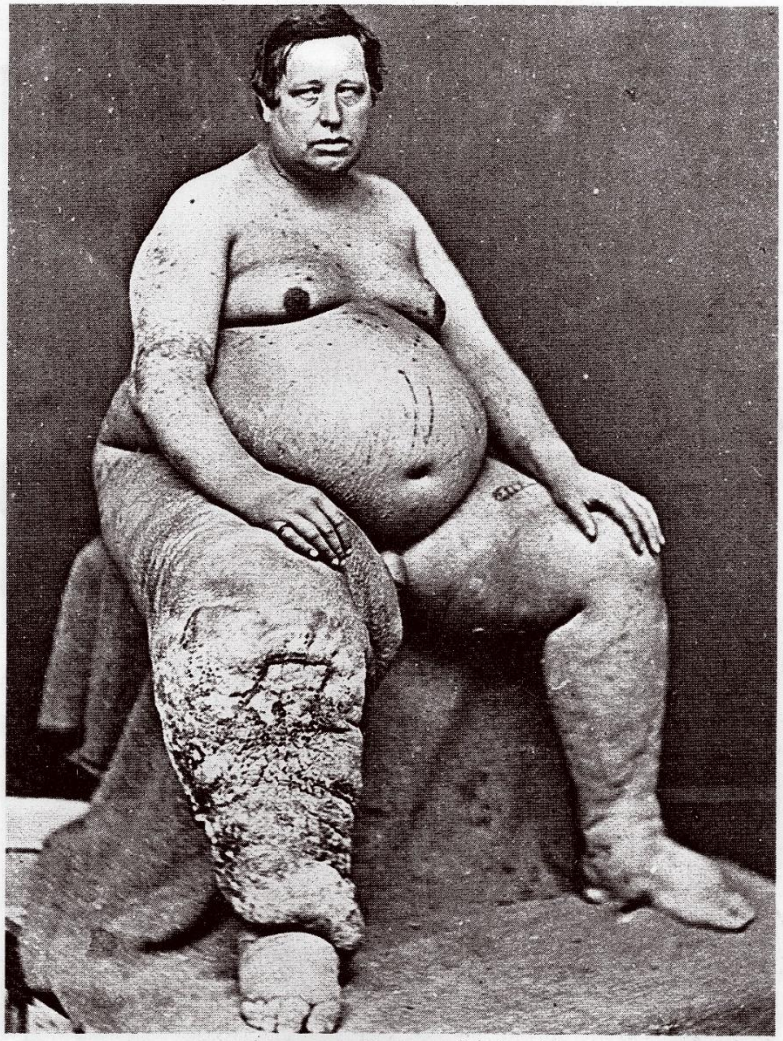
LOCALIZED EDEMA

- disturbed venous outflow
- disturbed lymphatic circulation = lymphedema
- inflammation
- allergy
- hypoxia
- cerebral space-occupying lesions



„elephantiasis”

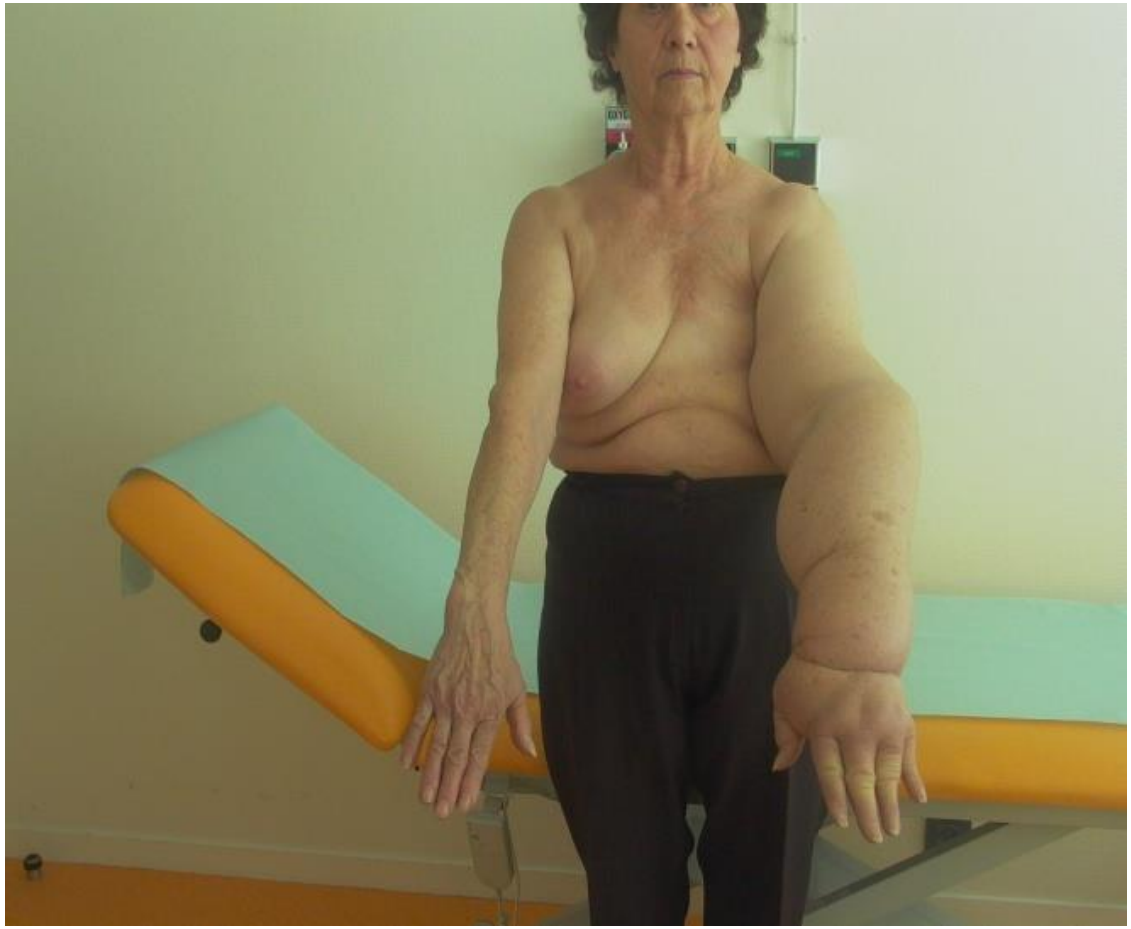




Anonyme. Femme éléphant.

What is Lymphatic Filariasis

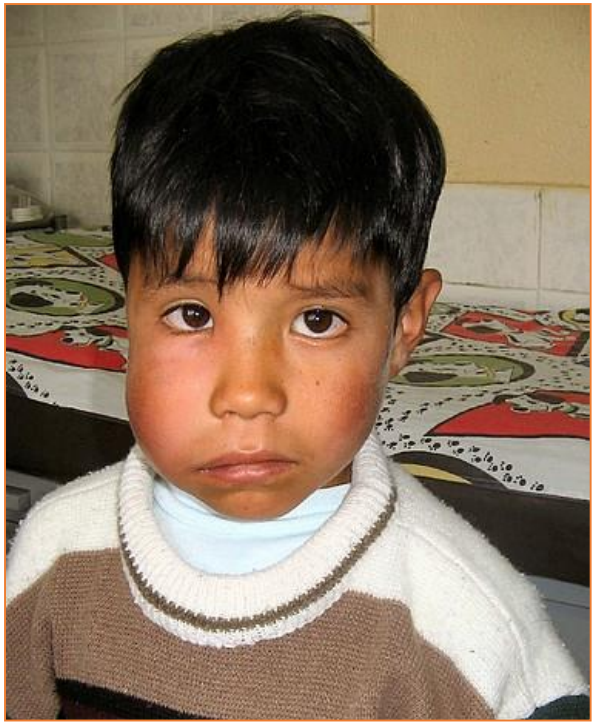
- Lymphatic filariasis is a vector-borne parasitic disease that is endemic in many tropical and subtropical countries. The disease is caused by thread-like, parasitic filarial worms: *Wuchereria bancrofti, Brugia malayi,* and *Brugia timori.*
- *W. bancrofti* is most widely spread and is responsible for more than 90% of the infections.



Lymphedema after mastectomy – remember, „once an edema, a lifetime of edema” (no cure)

LOCALIZED EDEMA

- disturbed venous outflow
- disturbed lymphatic circulation
- inflammation
- allergy
- hypoxia
-cerebral space-occupying lesions



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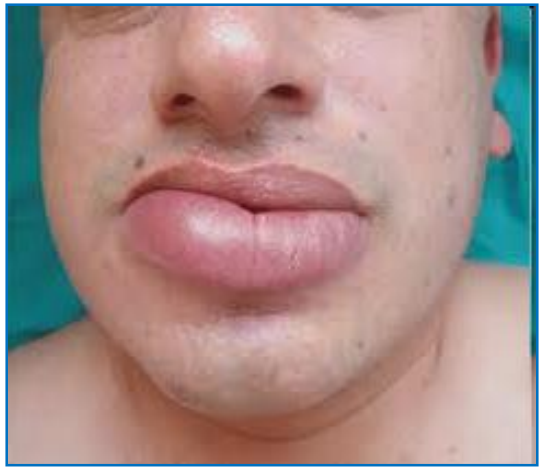
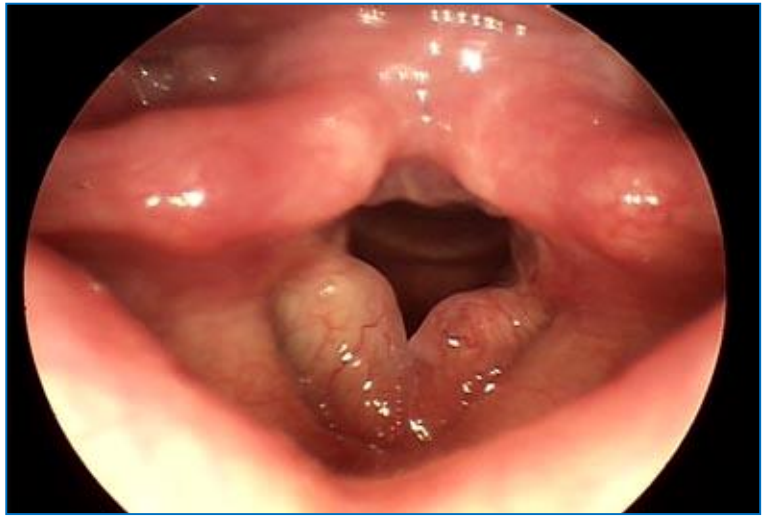
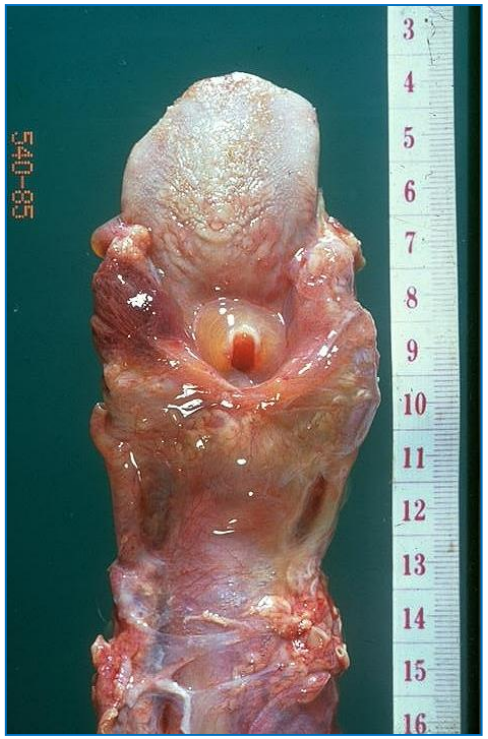
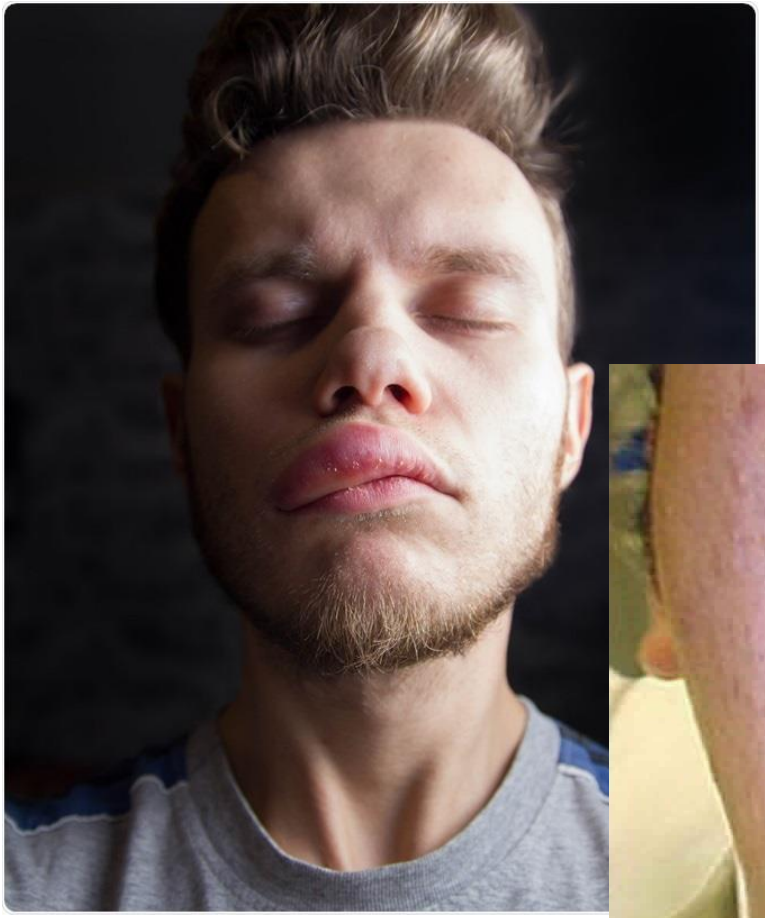
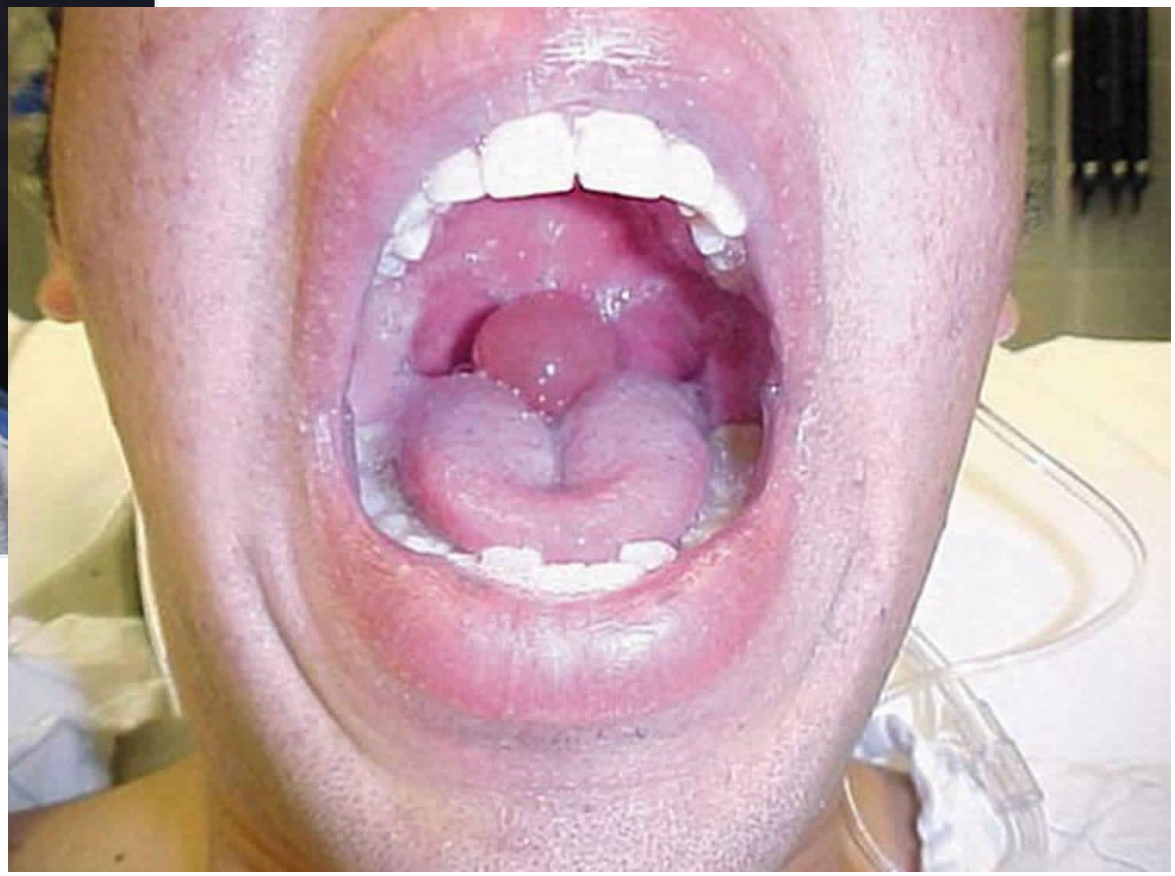






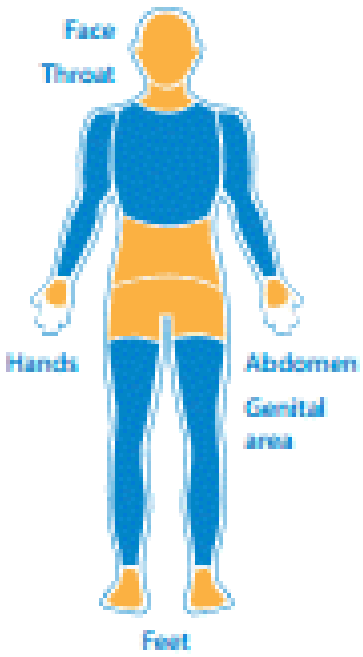




Quincke’s edema = angioneurotic uvular edema

What is Hereditary Angioedema (HAE)?

HAE is a rare inherited condition characterized by painful, recurring attacks of swelling in parts of the body including:[1,2]



It is the result of a problem with a protein called C1 esterase inhibitor.

There are three types of hereditary angioedema:

Type I

- 85% of cases[3]
- C1-INH is decreased or not present[4]

Type II

- 15% of cases[3]
- C1-INH is not working properly[4]

Type III

- Rare; prevalence is unknown[3]
- Diagnosed by genetic testing[4]

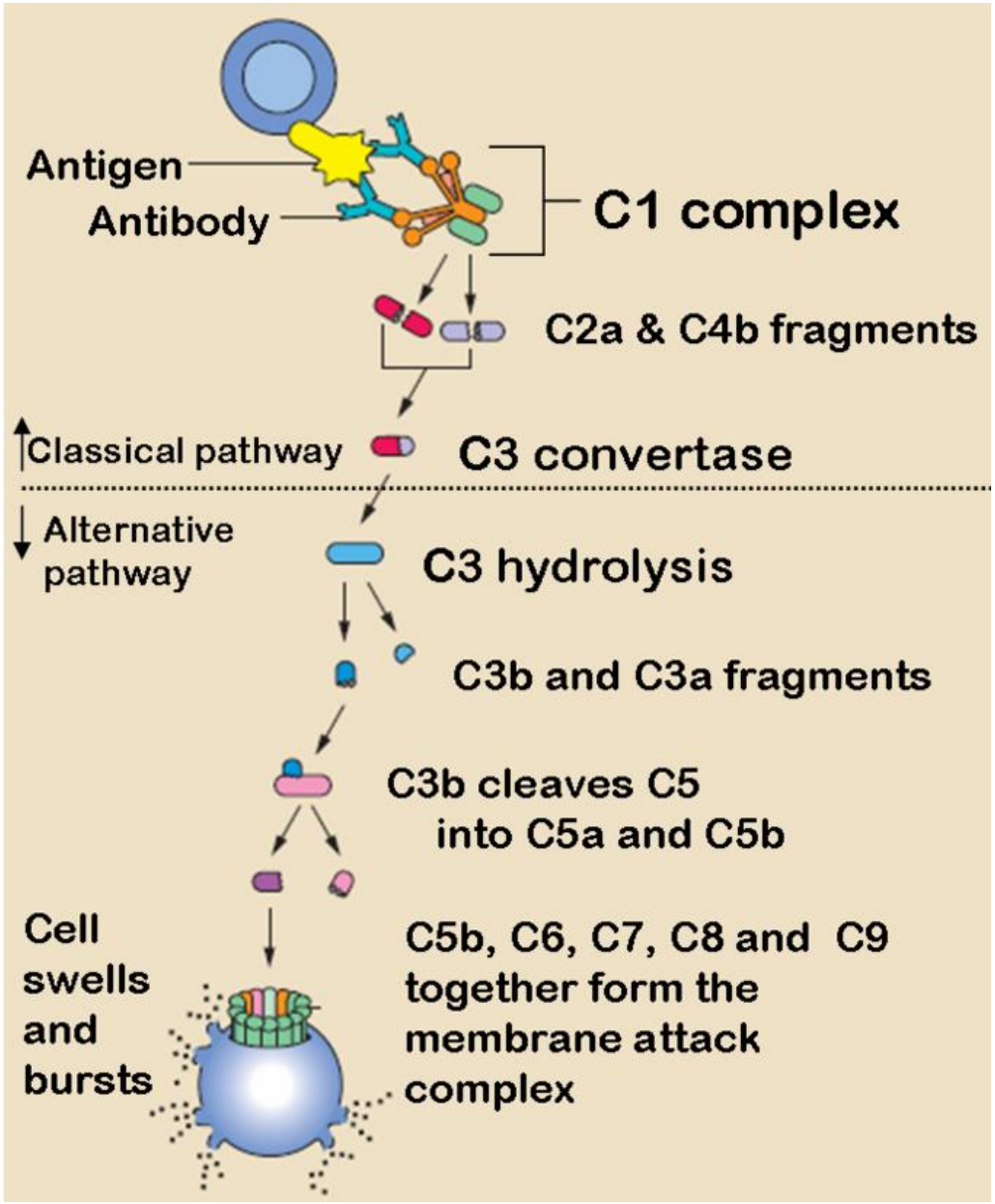
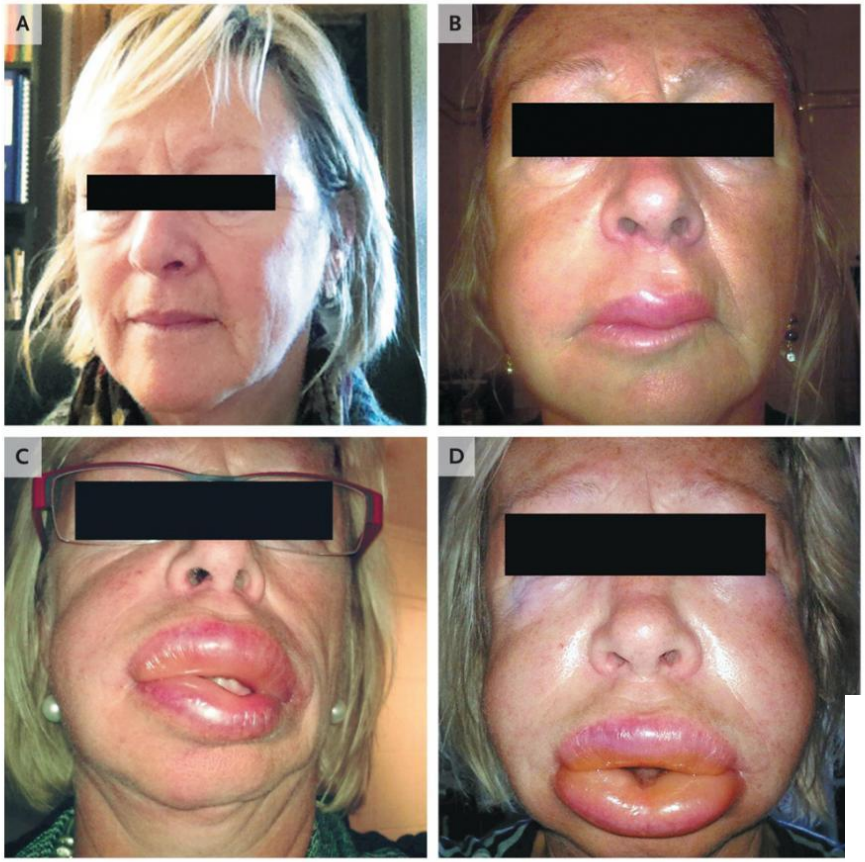

Antigen
Antibody
C1 complex
C2a & C4b fragments
Classical pathway
C3 convertase
Alternative pathway
C3 hydrolysis
C3b and C3a fragments
C3b cleaves C5 into C5a and C5b
Cell swells and bursts
C5b, C6, C7, C8 and C9 together form the membrane attack complex

FIGURE 1. Progressive Swelling Resulting From Angioedema Attack[14]



Images used with permission. Ebo DG, Bridts CH. Images in clinical medicine. Disfiguring angioedema. *N Engl J Med*. 2012;367(16):1539. doi: 10.1056/NEJMicm1200960.

LOCALIZED EDEMA

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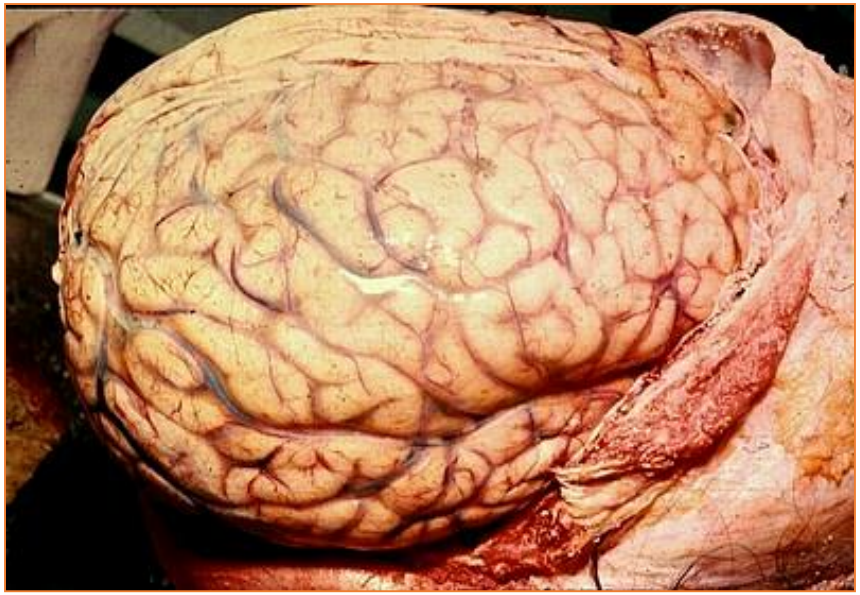
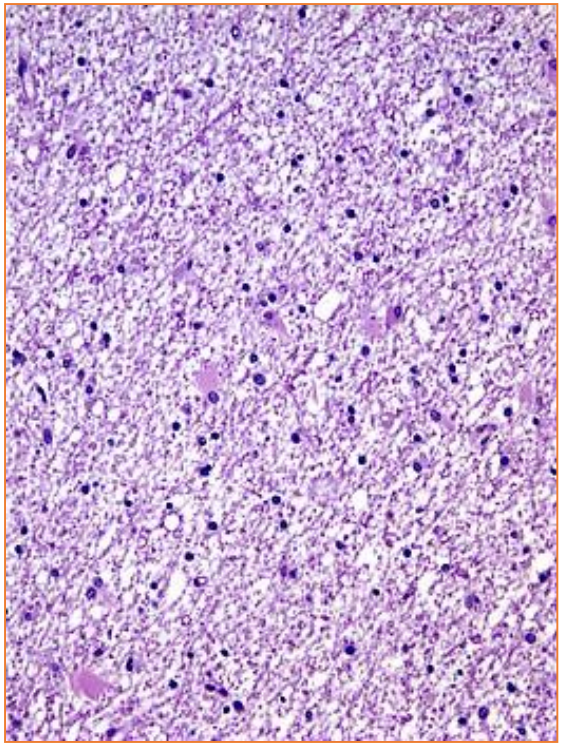
- disturbed lymphatic circulation
- inflammation
- allergy
- hypoxia
- cerebral space-occupying lesions





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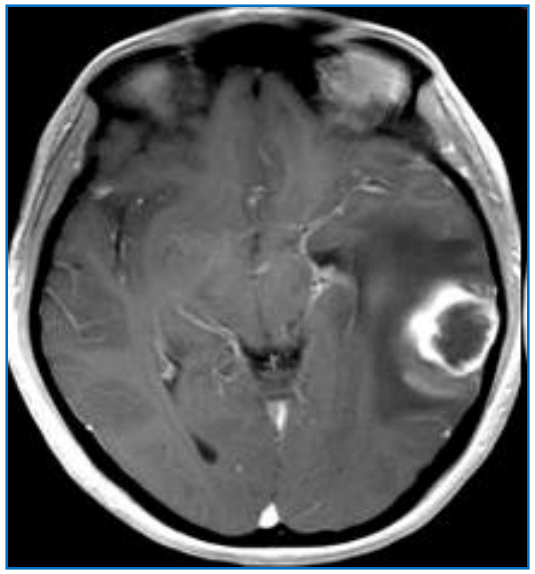


Perifocal edema (vasogenic)

brain tumors (primary, metastatic)

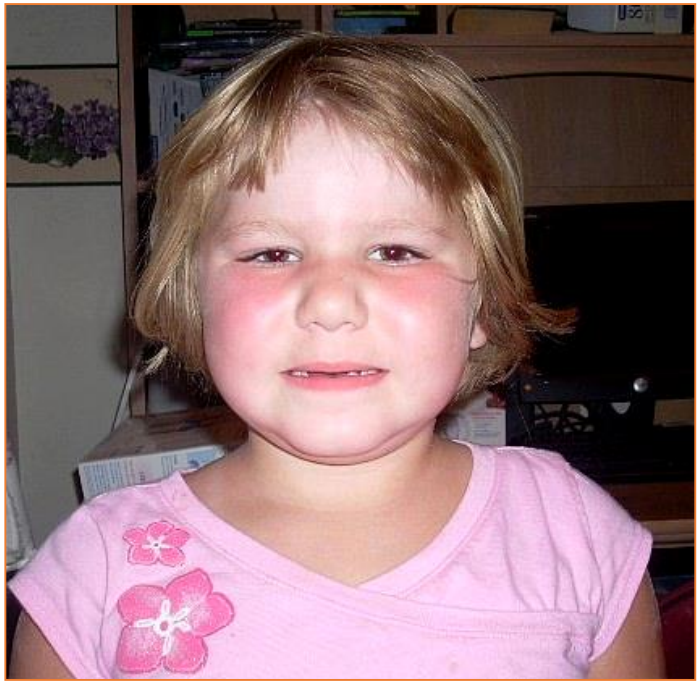
hemorrhage (apoplexia)

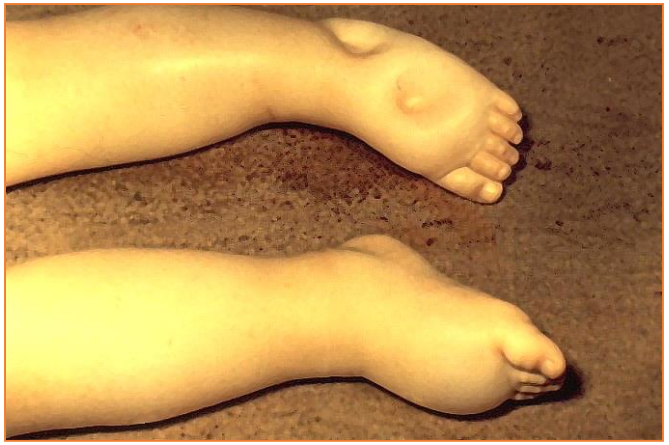
emollition

abscess (bacterial, fungal)

GENERALIZED EDEMA

- **Hypalbuminemia**
- **right sided heart failure**
- **Rh (ABO) incompatibility**





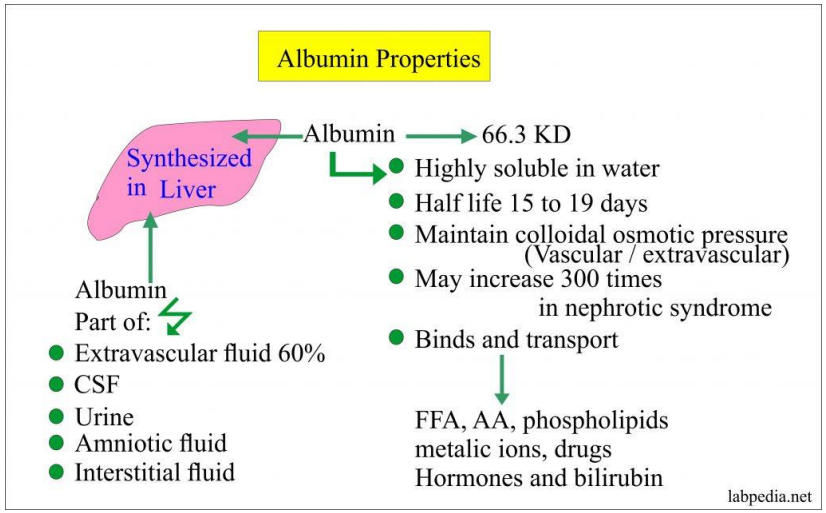
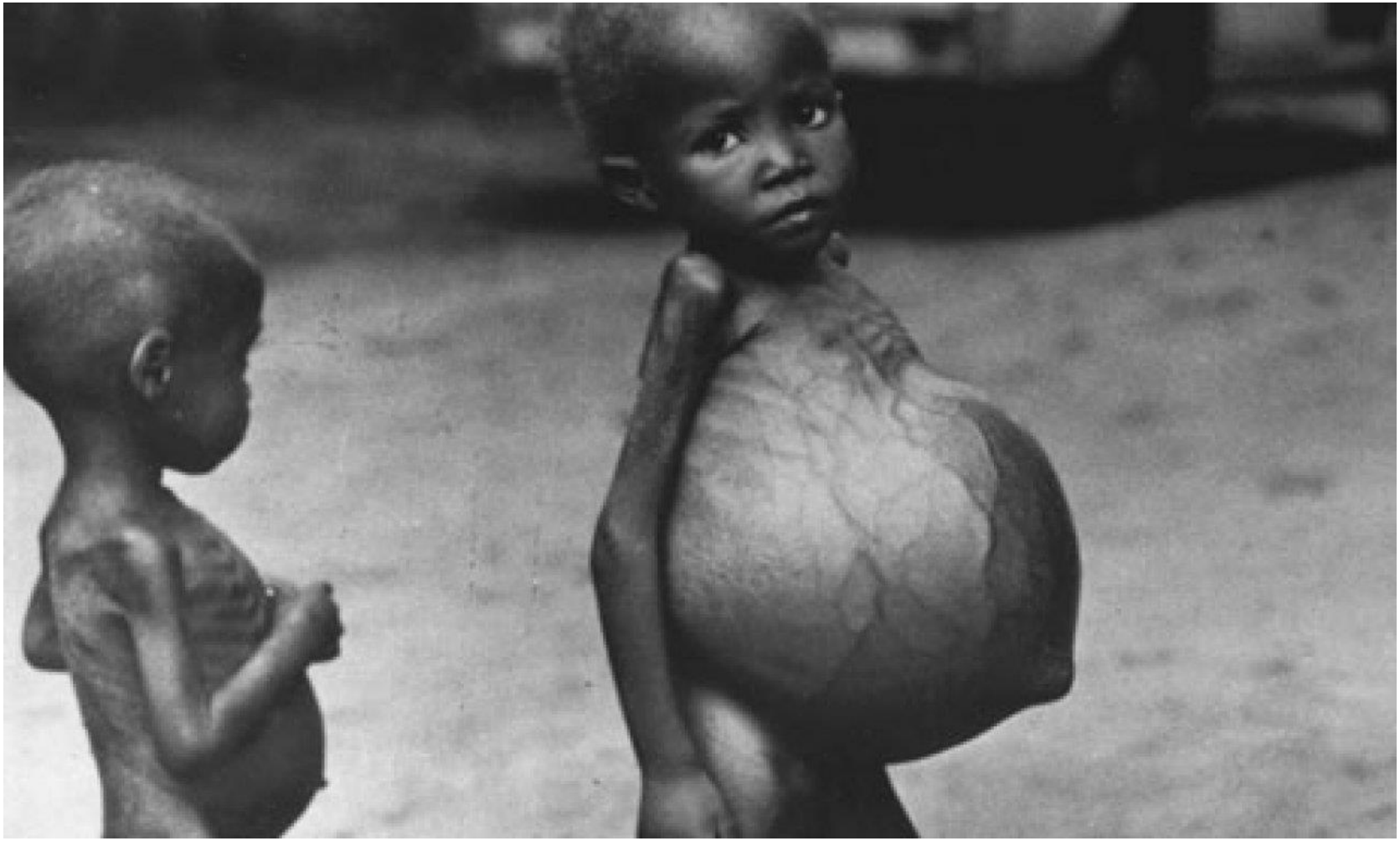


Table 1. Causes of Hypoalbuminemia Other Than PLE

◆ Impaired Synthesis ○ Chronic liver disease
◆ Increased Loss ○ Nephrotic syndrome
◆ Dilution ○ Volume overload in context of heart failure
◆ Inflammation ○ Acute inflammatory response (negative phase reactant) ○ Chronic inflammatory response

Malnutrition = „hunger edema”



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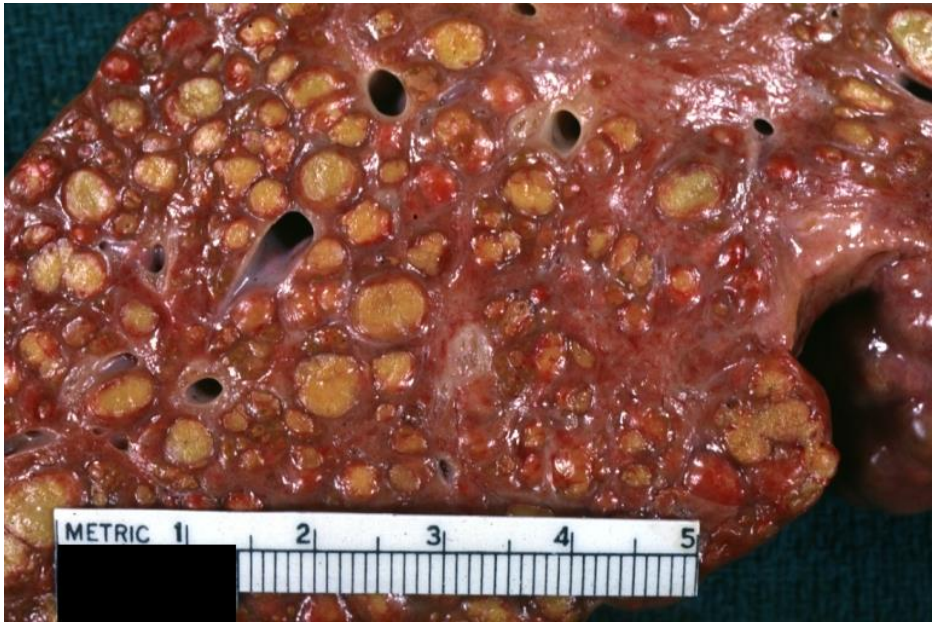
METRIC 1
2
3
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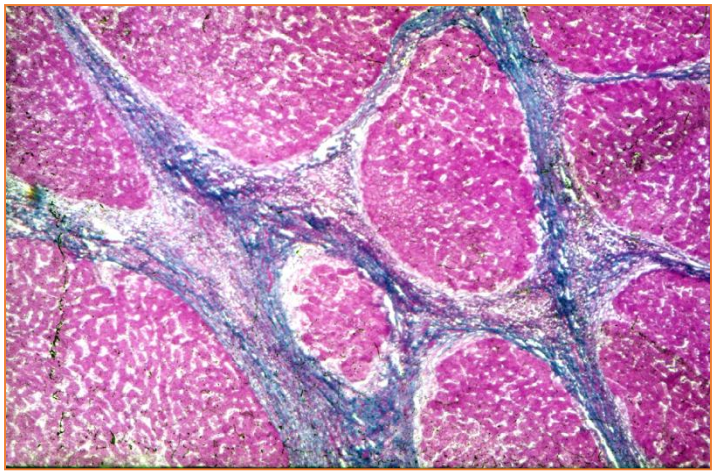
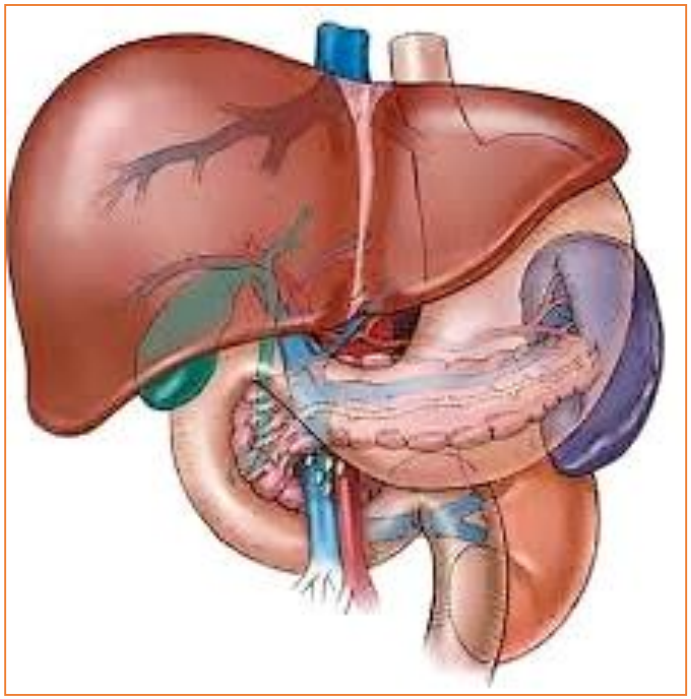
5





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Nephrotic Syndrome

Filtration Barrier

Podocytopathies

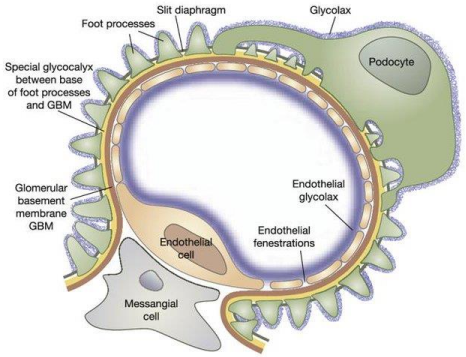
1. **MCD**
2. **FSGS**
   - Primary
   - Secondary
     Drugs
     Infectious
     Malignancy
     Adaptative (FSGS)

Immunocomplex Deposits

1. **Membranous**
   - Primary (PLA2R)
   - Secondary
     - Drugs
     - Infectious
     - Rheuma (Lupus)
     - Malignancy
2. **Membranoproliferative**
   - Complement dysregulation
   - Infectious
   - Autoimmune/Rheuma
   - Dysproteinemia

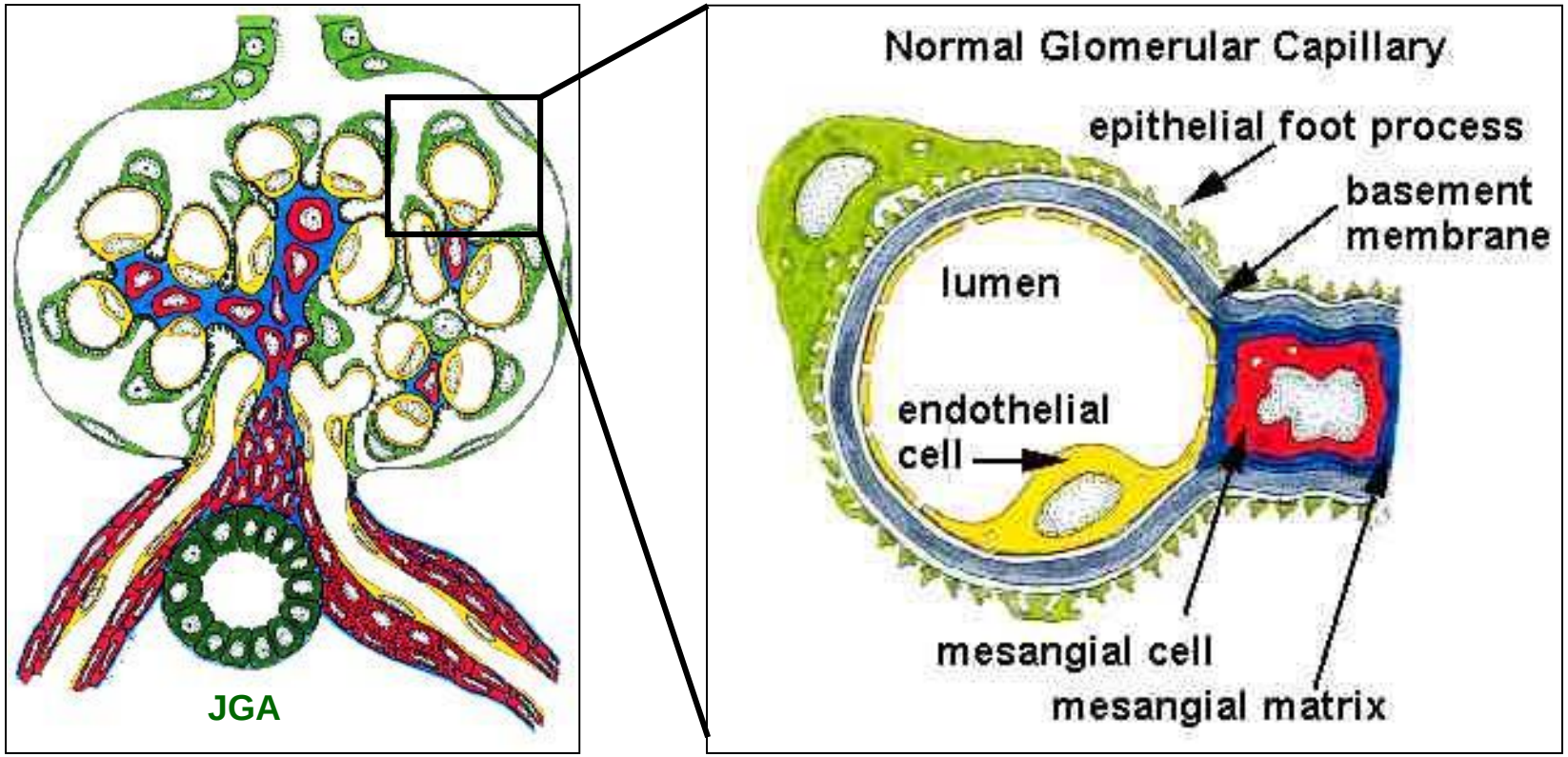
Other substances Deposits

1. **Diabetes**
2. **Amyloid**



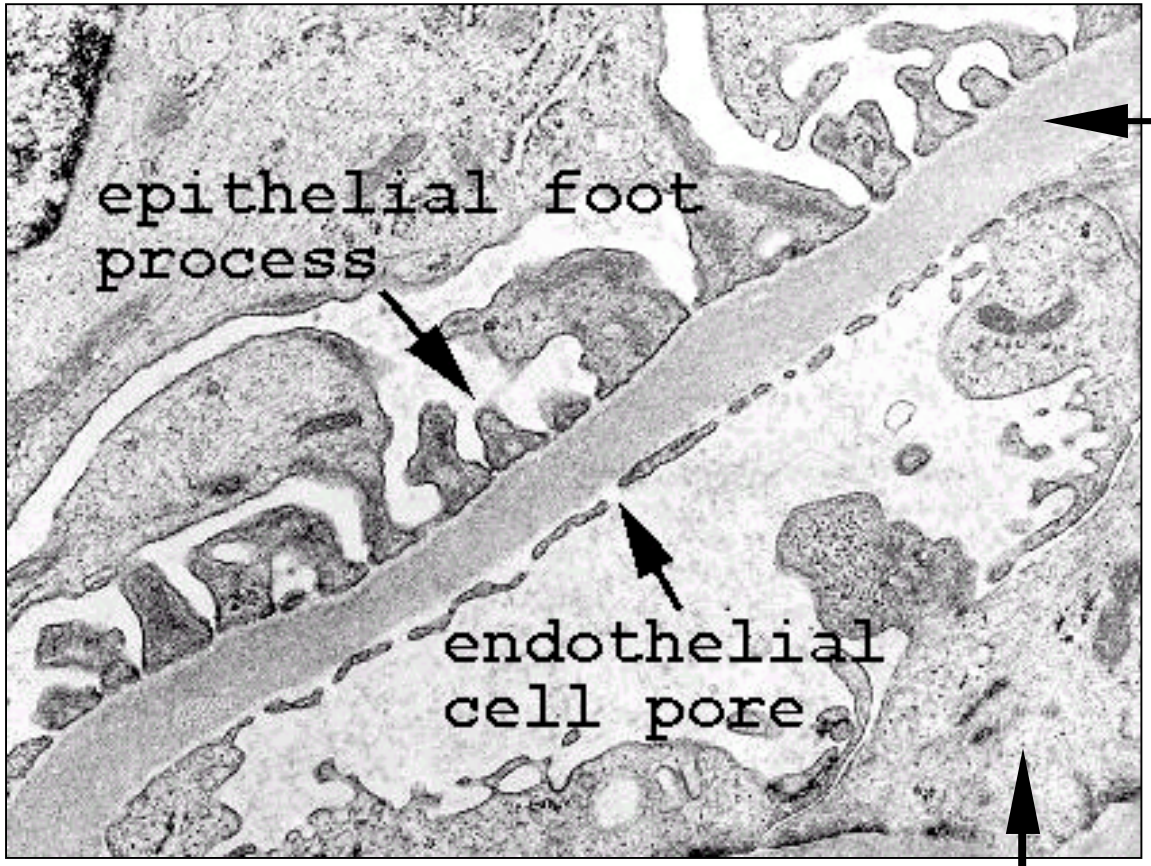
Structure of the glomerulus

urinary pole



vascular pole

Electron microscopical structure of the glomerulus



Pathological diagnosis of glomerular diseases

Light microscopy: HE, PAS, Mallory, Jones (silver-methenamine)

Immunofluorescence: Ig-s, complement

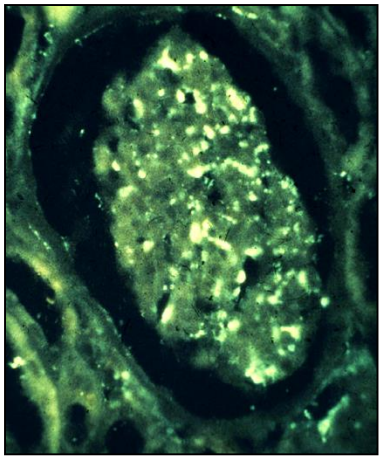
Electron microscopy

Basic terms:

Diffuse: > 80%
Focal: < 80%

Global: whole glomerulus is involved
Segmental: part of glomerulus is involved

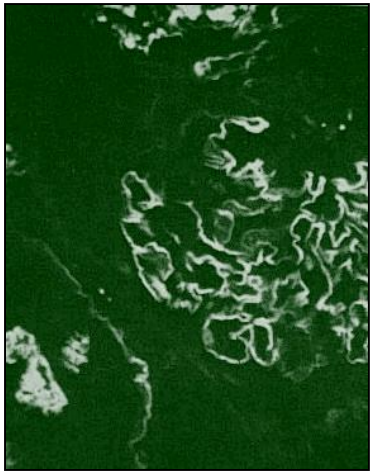
The pathological alterations are confined to the glomeruli, the other renal structures are involved secondarily



Basic mechanisms:

1. **Immuncomplex-deposition in the basement membrane**

   IF: granular deposition



2. **Antiglomerular basement membrane antibodies**

   IF: linear deposition

3. **Alternative pathway: hypocomplementemia,**

Minimal change

1. Primary glomerular diseases:

Minimal change

Poststreptococcal glomerulonephritis

Focal segmental glomerulosclerosis

Membranous glomerulonephritis

Membranoproliferative glomerulonephritis

IgA nephropathy (Berger-disease) –

- Schönlein-Henoch purpura

2. Glomerular involvement in systemic diseases:

Goodpasture syndrome

SLE

Amyloidosis

Childhood disease

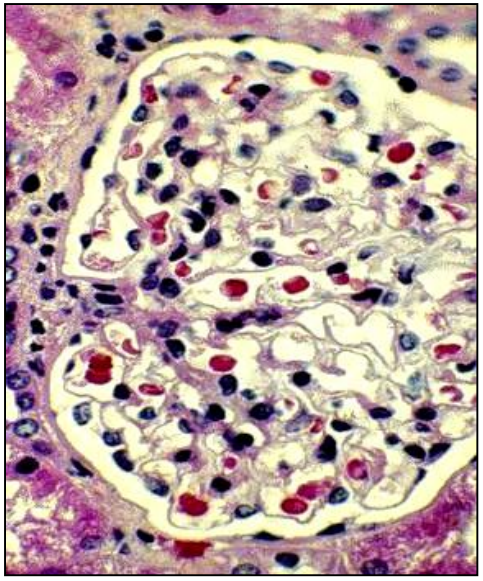
Nephrotic syndrome

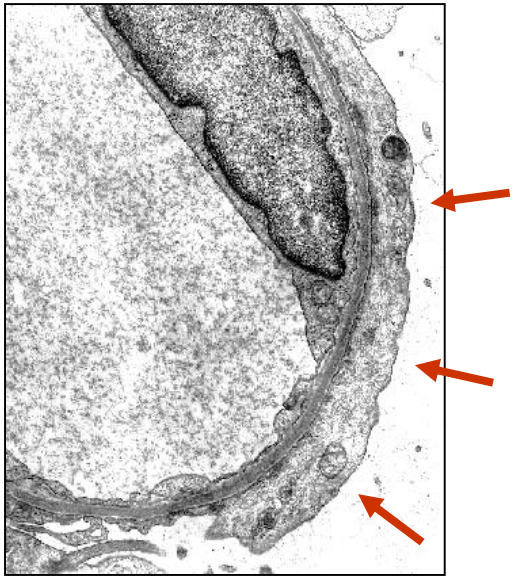
Cytokine-production by the T-lymphocytes

Light microscopy: no alteration at all

IF: negative

Diagnosis: electron microscopy (fusion of the podocyte foot processes)





Clinical presentations of glomerular diseases

- asymptomatic proteinuria
- nephrotic syndrome
  (proteinuria, hypoproteinemia, edema, hyperlipidemia)
- asymptomatic hematuria
- nephritis (nephritic) syndrome
  (hematuria, proteinuria, hypertension, GFR ↓, renal failure)
- crescentic glomerulonephritis
  (nephritis with rapidly progressive renal failure)
- chronic glomerulonephritis
  (chronic progression of renal failure)
- end stage renal disease – chronic renal insufficiency, uremia
  (irreversible renal failure)

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Minimal change

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2. Glomerular involvement in systemic diseases:

Goodpasture syndrome

SLE

Amyloidosis

Focal segmental glomerulosclerosis

Young adults

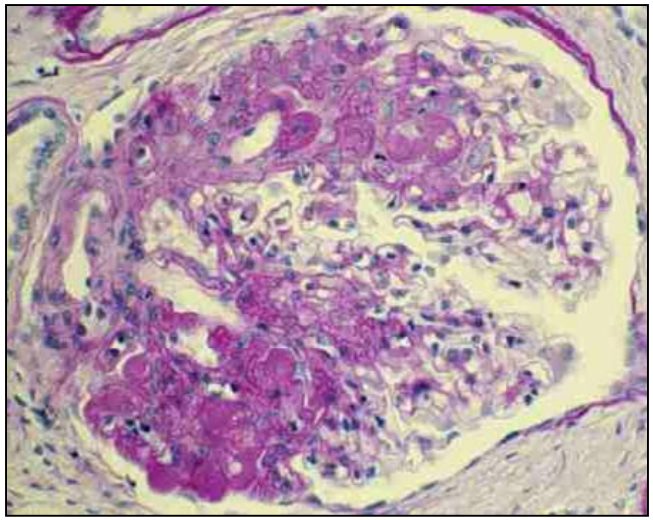
Nephrotic syndrome

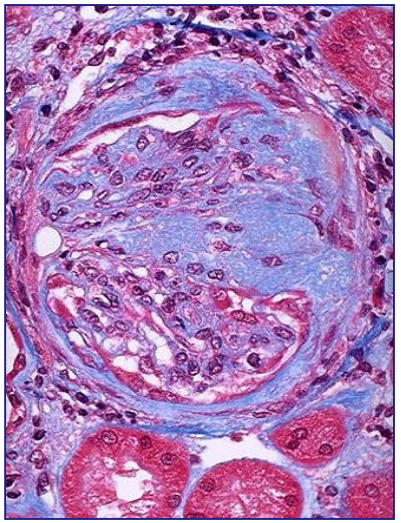
Slowly progressive, may recur after kindey Tx

Light microscopy: focally, segmentally sclerotic glomeruli

IF: granular IgM, C3

Electron microscopy: fusion of the foot processes





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2. Glomerular involvement in systemic diseases:

Goodpasture syndrome

SLE

Amyloidosis

Membranous glomerulonephritis

Nephrotic syndrome in adults!

Mainly idiopathic

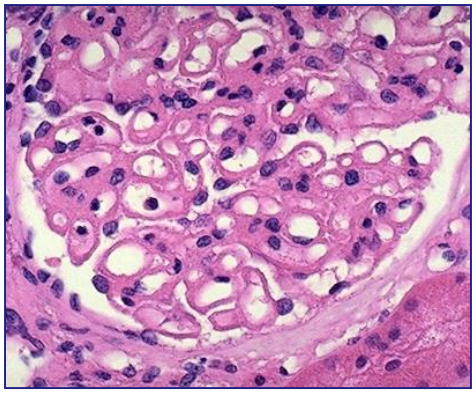
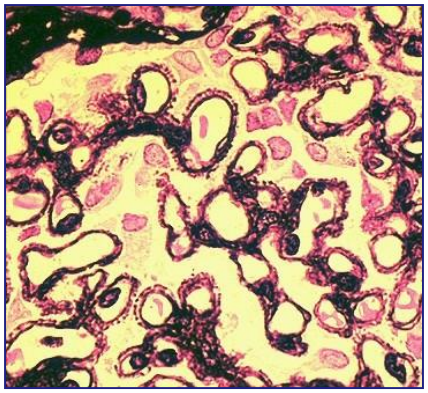
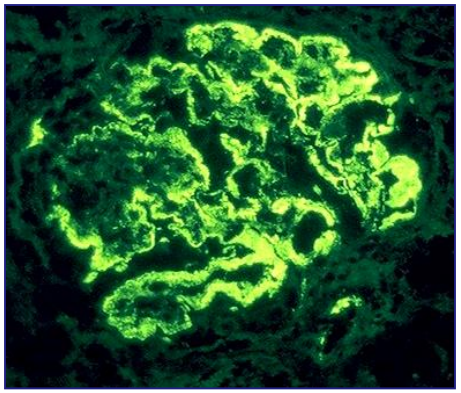
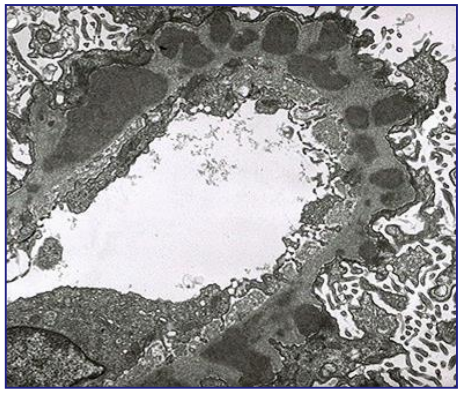
Frequently paraneoplastic, can be linked to infections

Thickened, prominent capillary loops

Spikes

IF: granular, mainly IgG immuncomplexes

Electron microscopy: dense immune deposits

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2. Glomerular involvement in systemic diseases:

Goodpasture syndrome

SLE

Amyloidosis

Goodpasture syndrome

Type II hypersensitivity reaction

Pulmonary hemorrhage + renal involvement

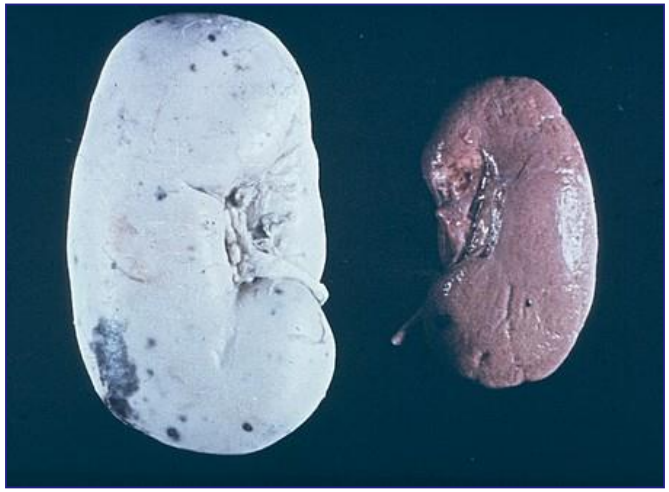
Antiglomerular basement membrane antibodies

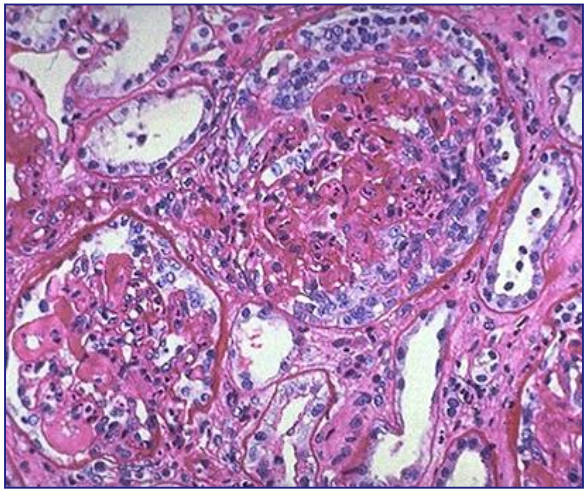
Nephrosis syndrome, rapidly progressive course

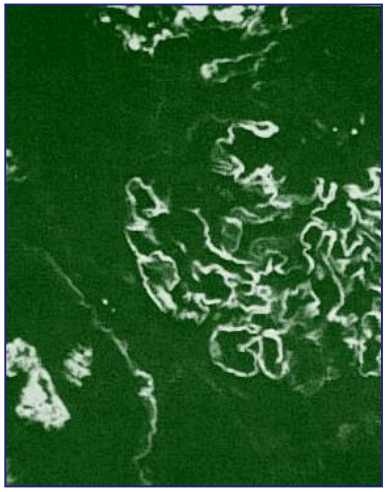
Gross: large, white kidneys

Light microscopy: cellular crescents

IF: linear antiglomerular basement membrane Ab-s

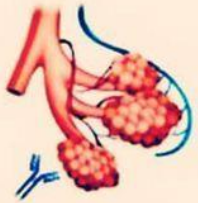






Goodpasture's Syndrome

→ Small vessel vasculitis



Glomerular **P**ulmonary

Anti-glomerular basement membrane antibodies (antiGBM)

Clinical

- Cough
- Dyspnea
- Hemoptysis
- Glomerulonephritis

Diagnosis

- Renal and lung biopsy showing **anti-glomerular basement membrane antibodies (antiGBM)**

Management

- Supportive
- Prednisone
- Cyclophosphamide
- Plasmapheresis

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IgA nephropathy (Berger-disease) –

- Schönlein-Henoch purpura

2. Glomerular involvement in systemic diseases:

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Amyloidosis

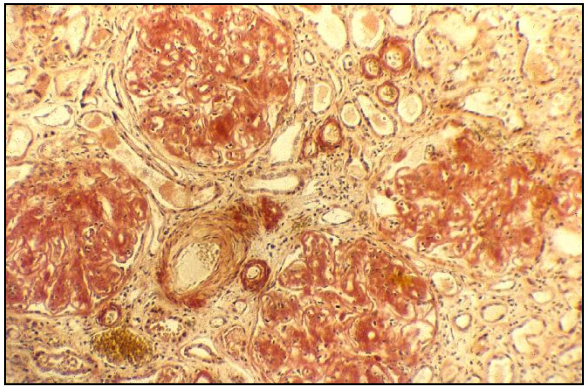
Amyloidosis

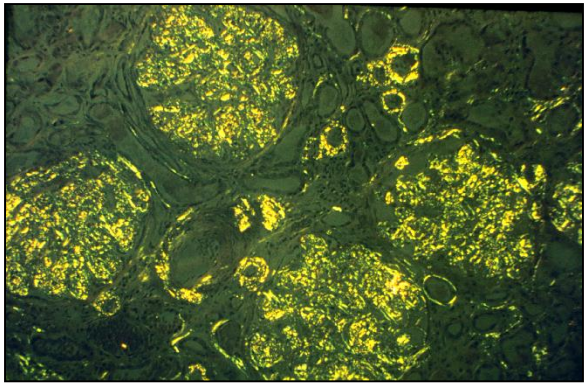
Nephrotic syndrome

Light microscopy: eosinophilic, homogeneous material

(capillaries, vessel walls)

Special stain: Congo red + polarization





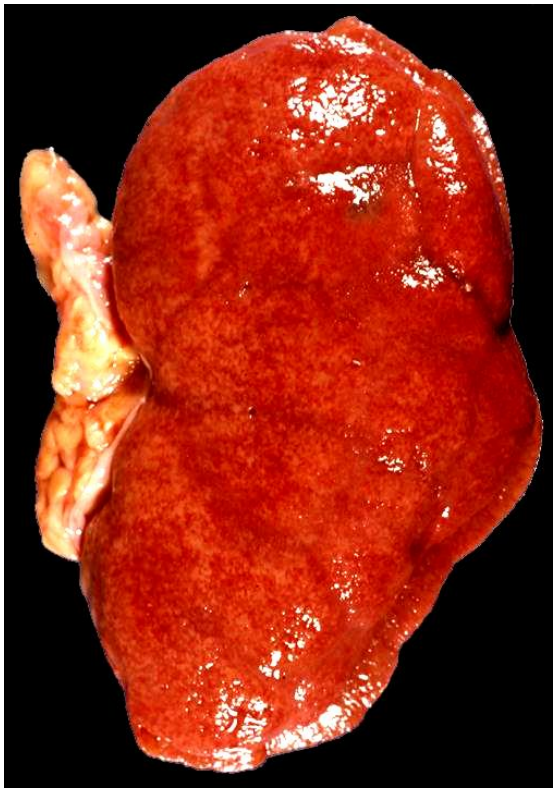
Kimmelstiel-Wilson syndrome

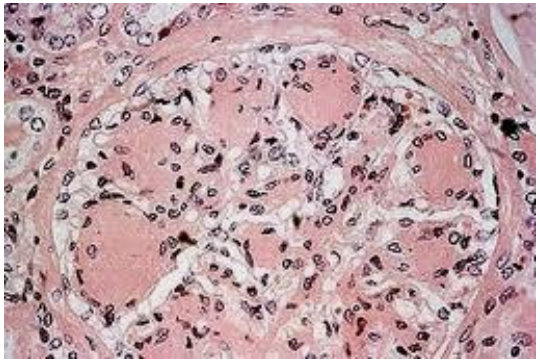
Nephrotic syndrome at the late stage of diabetes mellitus

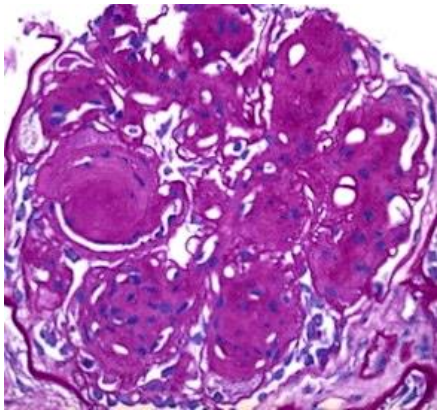
(10-20 ys)

FM: nodular glomerulosclerosis

IF: no immune deposition







Paul Kimmelstiel and Clifford Wilson

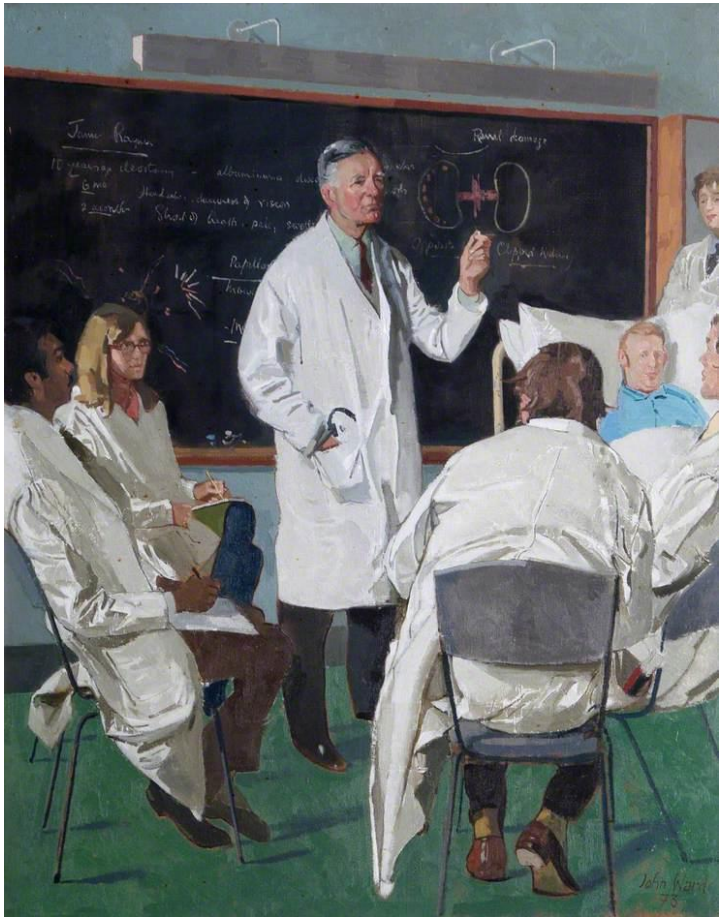
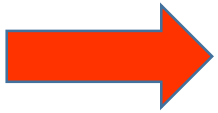


Table 1. Causes of Hypoalbuminemia Other Than PLE

- Impaired Synthesis
  - Chronic liver disease
- Increased Loss
  - Nephrotic syndrome
- Dilution
  - Volume overload in context of heart failure
- Inflammation
  - Acute inflammatory response (negative phase reactant)
  - Chronic inflammatory response



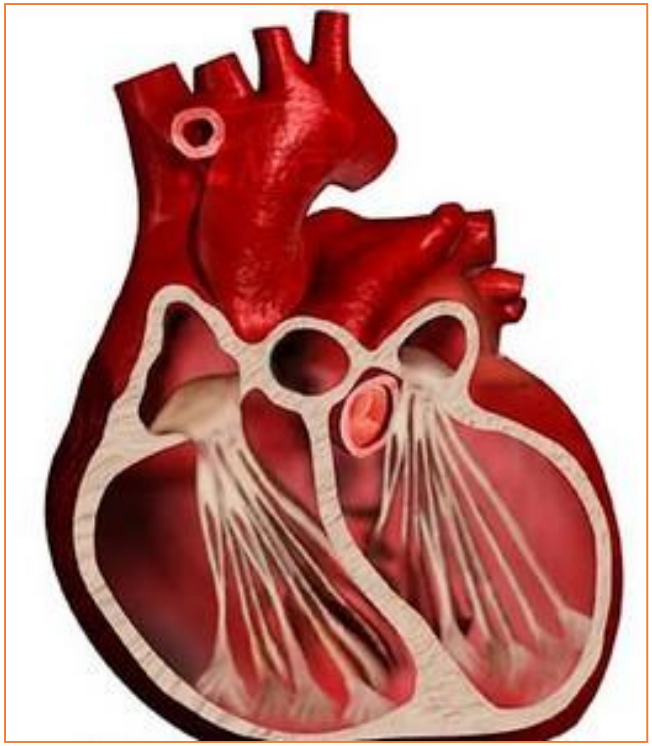
Volume overload due to right-sided heart failure

hydropericardium

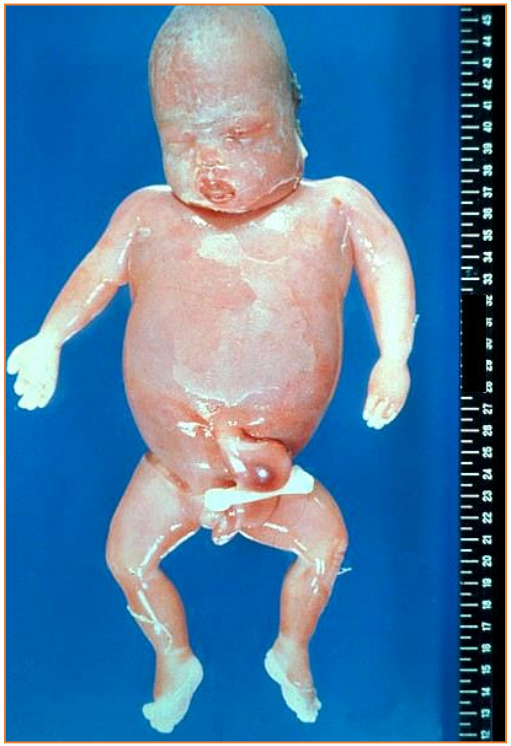
bilateral hydrothorax

ascites

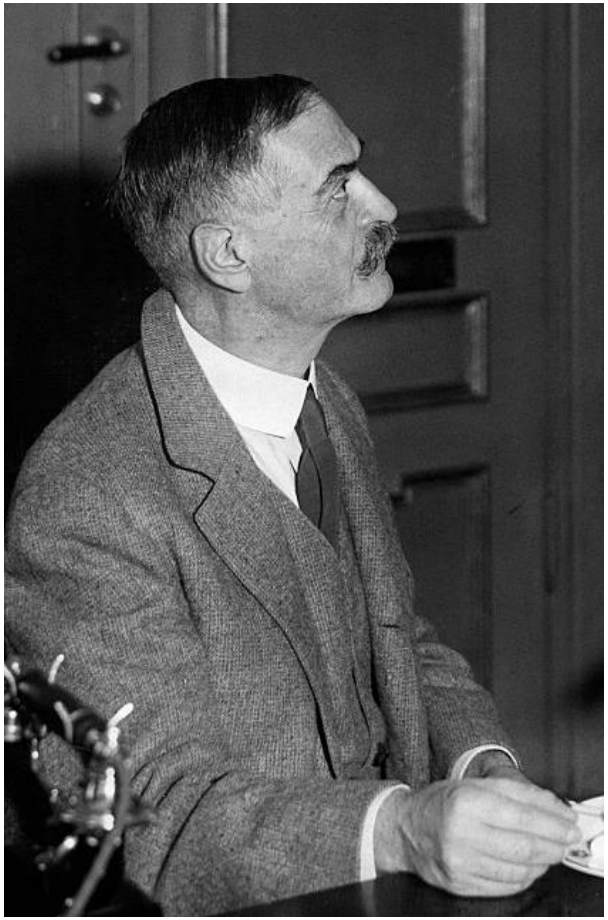
anasarca



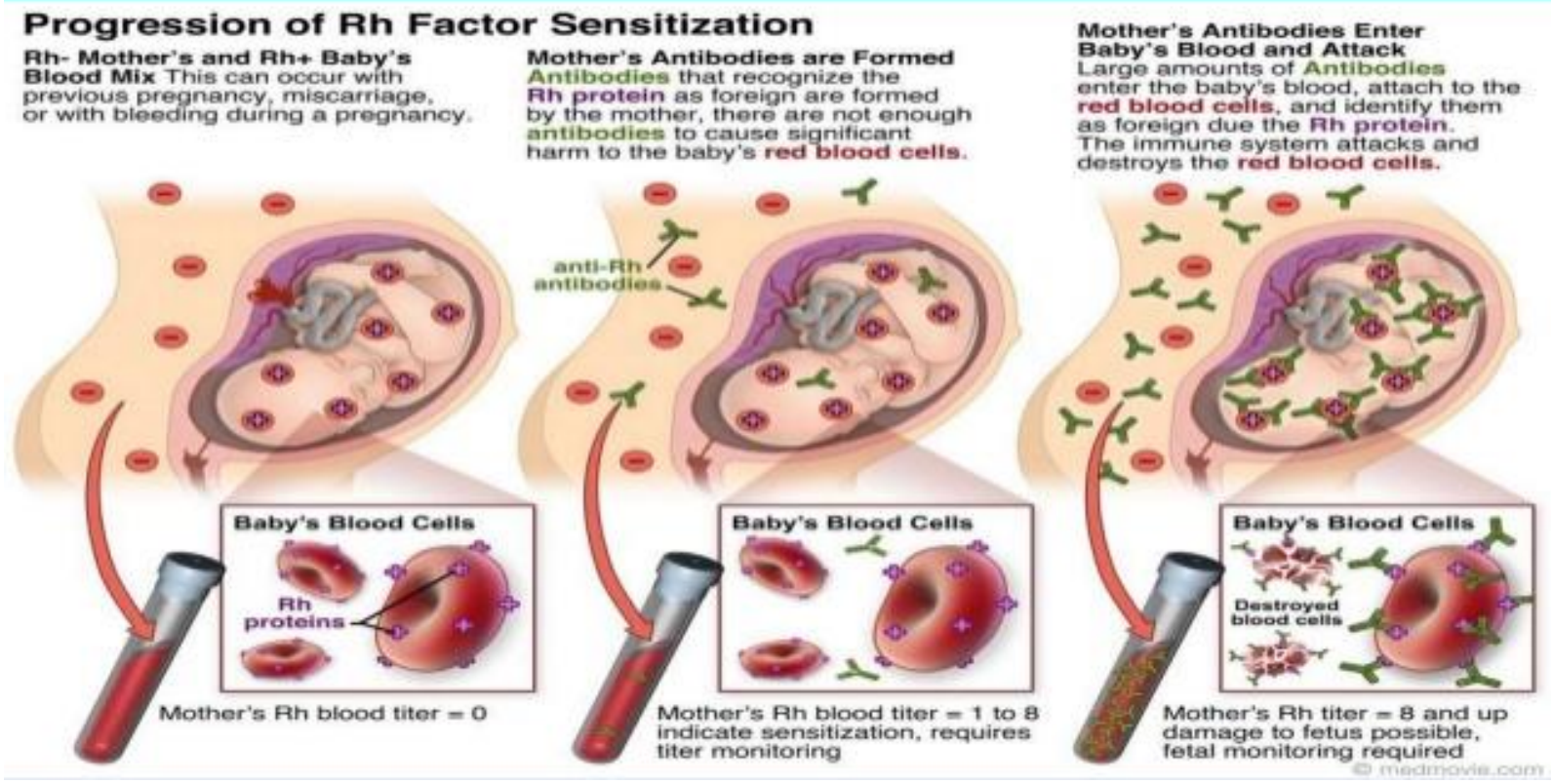
Rh incompatibility, Hemolytic disease of the newborn Erythroblastosis fetalis (*„hydrops fetalis*”)

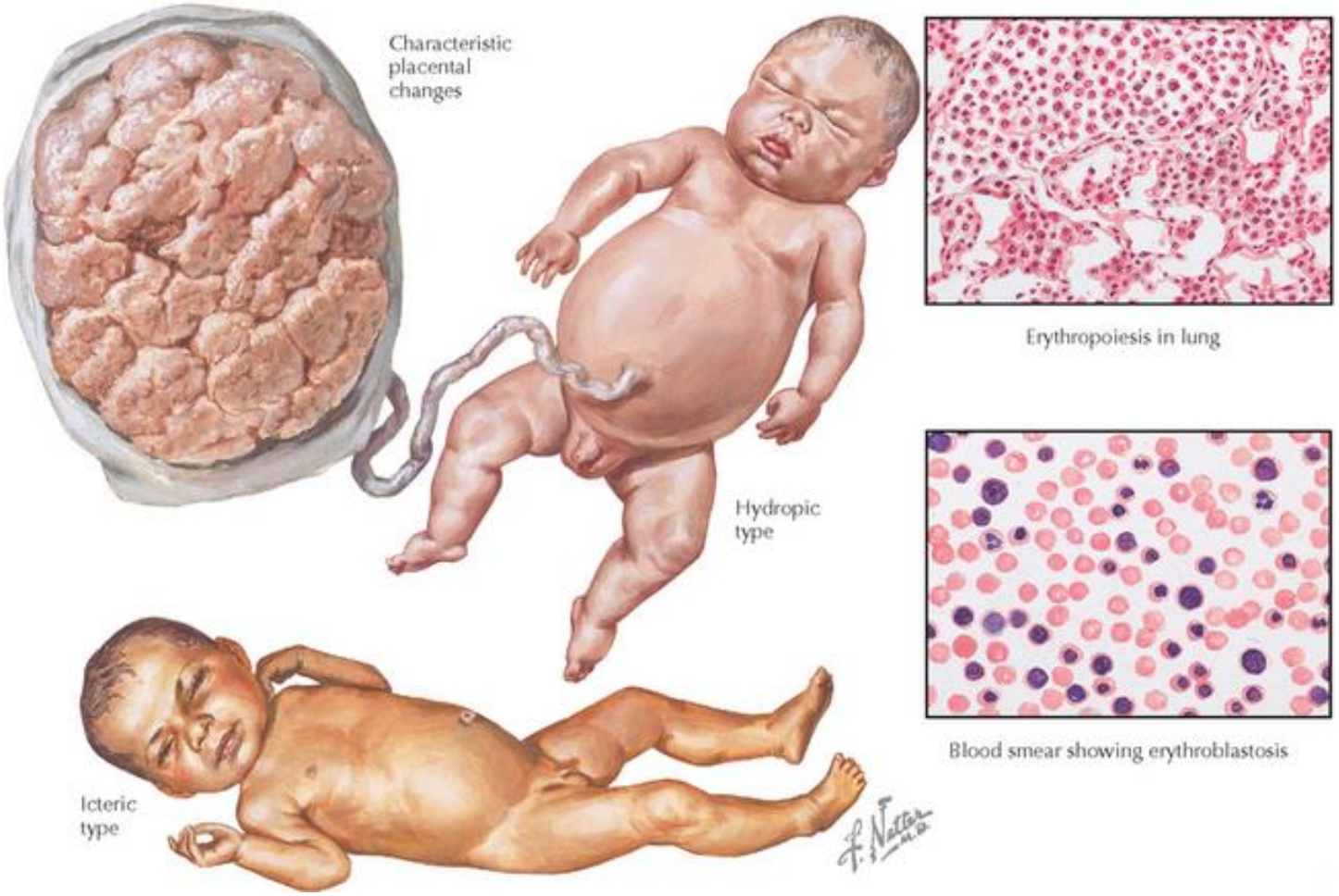


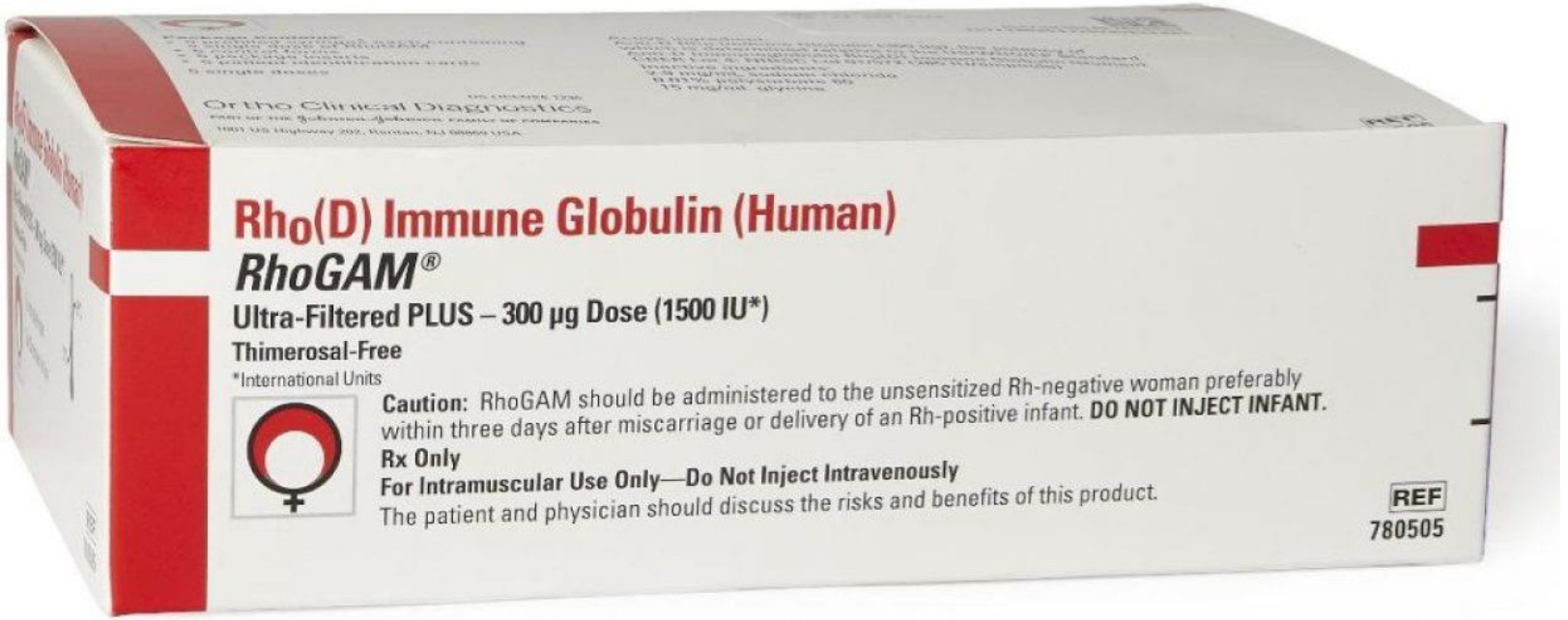
1940, Karl Landsteiner discovers the „rhesus factor”

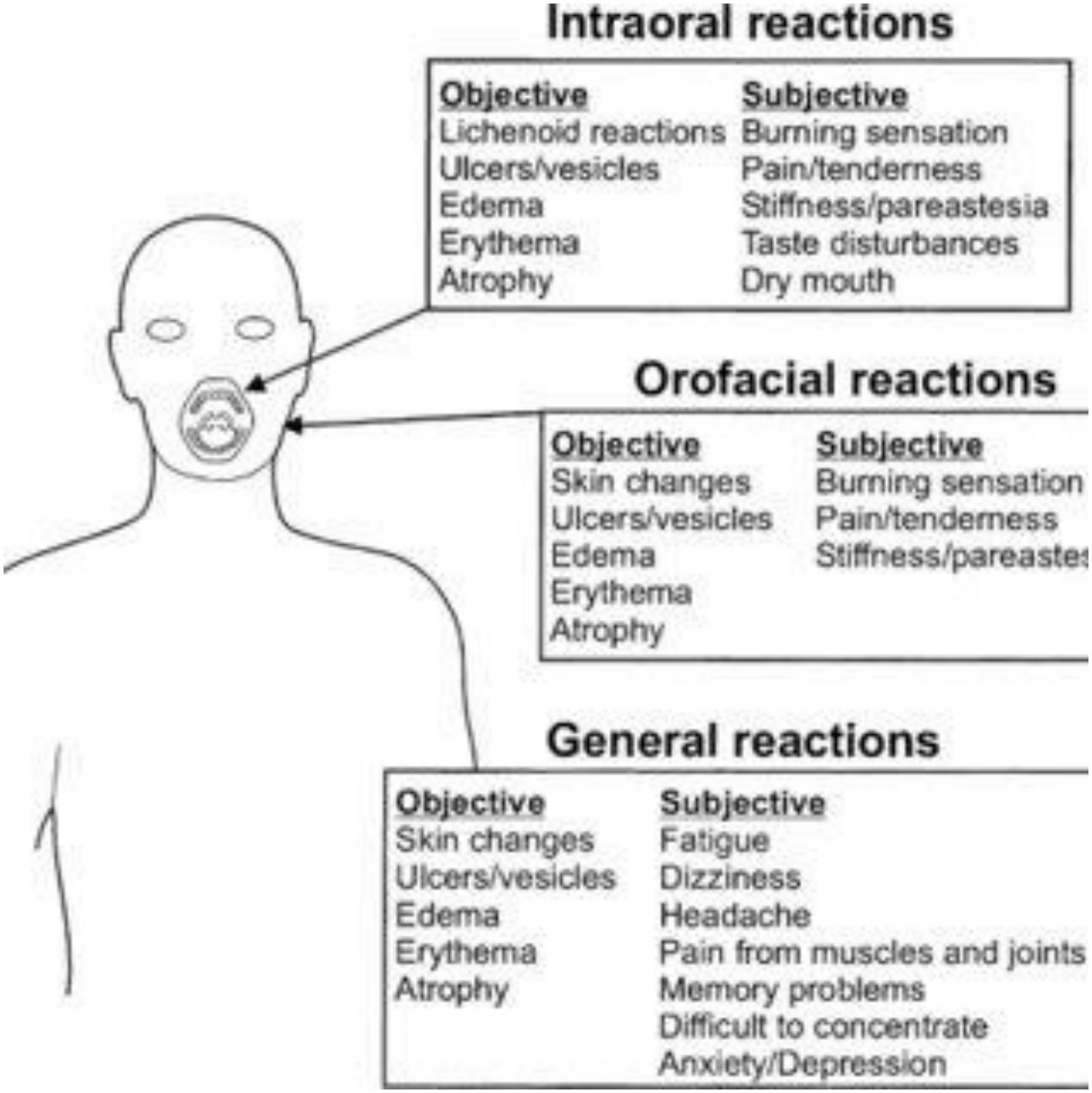


PATHOPHYSIOLOGY

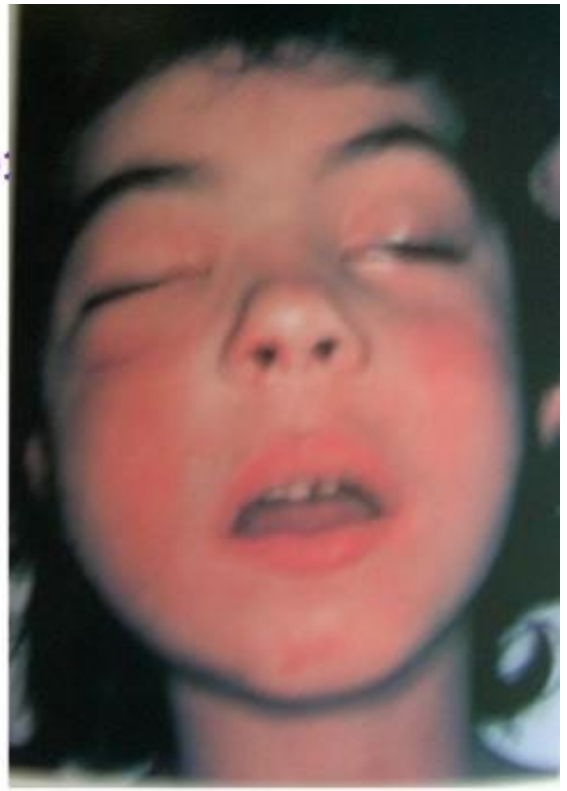



Characteristic placental changes
Hydropic type
Icteric type
Erythropoiesis in lung
Blood smear showing erythroblastosis


Ortho Clinical Diagnostics
Rho(D) Immune Globulin (Human)
RhoGAM®
Ultra-Filtered PLUS – 300 µg Dose (1500 IU*)
Thimerosal-Free
*International Units
Caution: RhoGAM should be administered to the unsensitized Rh-negative woman preferably within three days after miscarriage or delivery of an Rh-positive infant. DO NOT INJECT INFANT.
Rx Only
For Intramuscular Use Only—Do Not Inject Intravenously
The patient and physician should discuss the risks and benefits of this product.
REF
780505


Intraoral reactions
Objective
Lichenoid reactions
Ulcers/vesicles
Edema
Erythema
Atrophy
Subjective
Burning sensation
Pain/tenderness
Stiffness/pareastesia
Taste disturbances
Dry mouth
Orofacial reactions
Objective
Skin changes
Ulcers/vesicles
Edema
Erythema
Atrophy
Subjective
Burning sensation
Pain/tenderness
Stiffness/pareaste
General reactions
Objective
Skin changes
Ulcers/vesicles
Edema
Erythema
Atrophy
Subjective
Fatigue
Dizziness
Headache
Pain from muscles and joints
Memory problems
Difficult to concentrate
Anxiety/Depression

POST-OP OEDEMA



Excessive post-op edema occurs due to:

Tight suturing

Rough tissue handling.

Pulling on flaps.

Traumatic bone cutting.

MANAGEMENT:

Loosen sutures.

Steroids like dexamethazone or.[decadran 4mg I.V].
Hydrocortisone.