HYPERTENSIVE VASCULAR DISEASE

Hypertension can be classified according to
-the readings (benign hypertension(HTN) and malignant HTN)
-and to etiology(essential HTN and secondary HTN)

Benign Hypertension(HTN)

- Cutoffs in diagnosing hypertension in clinical practice➔ ***sustained*** **diastolic pressures >90 mm Hg, and/or *sustained* systolic pressures >140 mm Hg**

- ***Malignant hypertension*** *(has nothing to do with tumors)*

→ A small percentage of HTN patients (5%) present with a rapidly rising blood pressure that, **if untreated, leads to death within 1 to 2 years.**

➔ **systolic pressures > 200 mm Hg or diastolic pressures > 120 mm Hg**

→ associated with **renal failure** and **retinal hemorrhages**

→ **most commonly is superimposed on preexisting benign hypertension**
(especially in those who don't take their medication probably, or have a bad diet)

Because it can damage the vital organs in a short period of time:
-the brain → cerebrovascular disease (e.g. stoke or dementia)
-cardiovascular sys. →heart failure or MI
-renal failure

Hypertension (HTN) has the following complications:

These compilation can happen in both malignant hypertension (HTN)or benign HTN, and the only difference is that the progression is allot slower in benign HTN

- stroke (CVD)
- multi-infarct dementia
- atherosclerotic coronary heart disease
- cardiac hypertrophy and heart failure (*hypertensive heart disease*)
- aortic dissection
- renal failure

Types of hypertension

1- essential/primary/idopathic hypertension: most cases (95%) are idiopathic.

2- **secondary hypertension :** (5%) Most of the remaining cases **(**are **most commonly** due to renal disease, or renal artery narrowing **(called renovascular hypertension),** and to a lesser degree many other conditions….

Essential HTN
Accounts for 90% to 95% of all cases

Secondary HTN

Renal
Acute glomerulonephritis
Chronic renal disease
Polycystic disease
Renal artery stenosis
Renal vasculitis
Renin-producing tumors

Endocrine
Adrenocortical hyperfunction (Cushing syndrome, primary aldosteronism, CAH
licorice ingestion)
Exogenous hormones (glucocorticoids, estrogen sympathomimetics
monoamine oxidase inhibitors)
Pheochromocytoma
Acromegaly
Hypothyroidism (myxedema)
Hyperthyroidism (thyrotoxicosis)
Pregnancy-induced (pre-eclampsia)

The doctor didn't read them but asked us to read them :P

Cardiovascular
Coarctation of aorta
Polyarteritis nodosa
Increased intravascular volume
Increased cardiac output
Rigidity of the aorta

Neurologic
Psychogenic
Increased intracranial pressure
Sleep apnea
Acute stress, including surgery

- ***Pathogenesis of essential HTN***
- **Genetic factors**
- **familial clustering of hypertension**
- HTN has been linked to specific angiotensinogen polymorphisms and angiotensin II receptor variants; polymorphisms of the renin-angiotensin system. Susceptibility genes for essential hypertension are currently **unknown** but probably include those that control renal sodium absorption(abnormalities in the genes that control electrolytes channels), etc.

Its not a necessity to have one of these to develop essential HTN (these are just hypothesizes)

Pathogenesis of essential HTN

- **Environmental factors**
- such as stress, obesity, smoking, physical inactivity, and high levels of salt consumption, modify the impact of genetic determinants.
- Evidence linking dietary sodium intake with the prevalence of hypertension in different population groups is particularly strong.

Morphology

- **HTN is associated with arteriolosclerosis (small arterial disease)**
- **Two forms of small blood vessel disease are hypertension-related:**

1- hyaline arteriolosclerosis

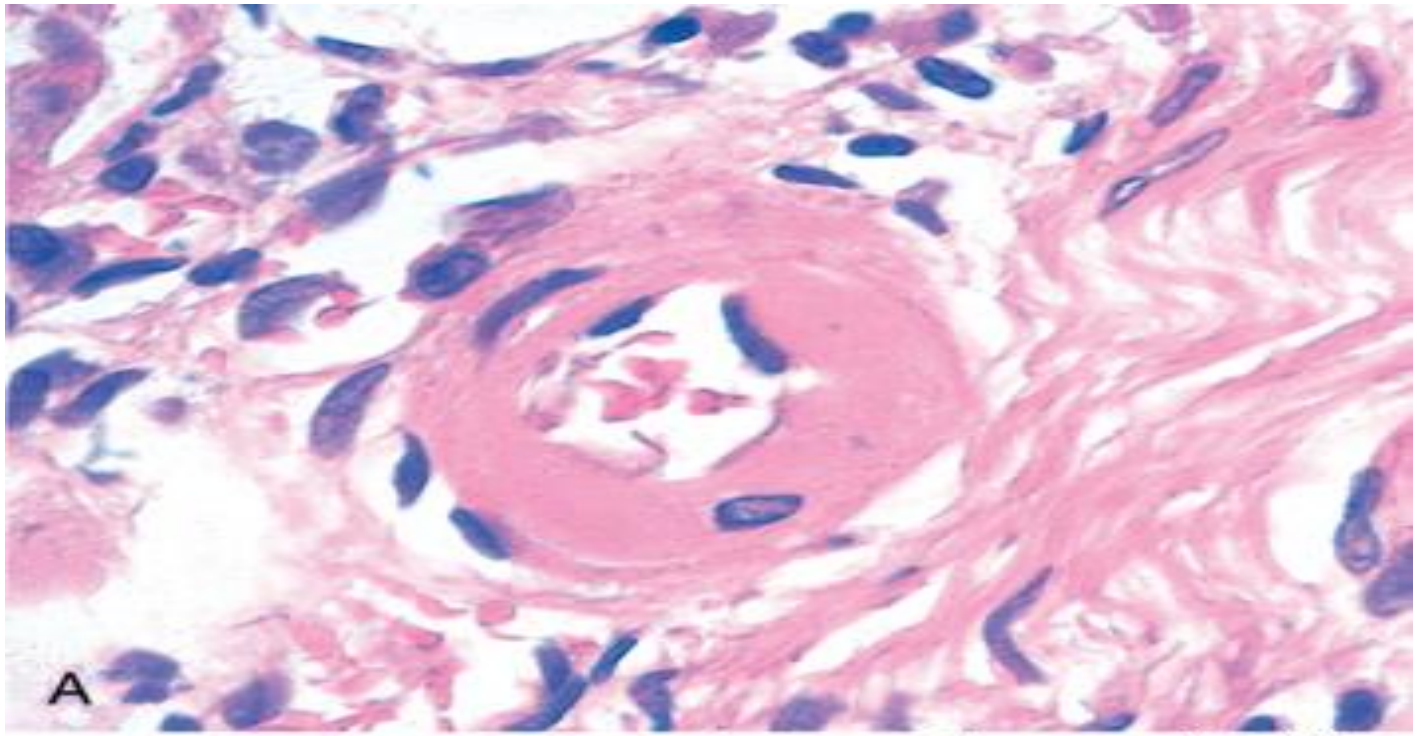
2- hyperplastic arteriolosclerosis

Remember:
-Arteriolosclerosis is most commonly caused by HTN but might also result from diabetes or the normal aging process
- It has a diffuse effect in all the vessels of the body

Hyaline arteriolosclerosis

- **associated with *benign hypertension*.**
- **marked by homogeneous, pink hyaline thickening of the arteriolar walls, and luminal narrowing.**
- **Results from leakage of plasma components across injured endothelial cells, into vessel walls and increased ECM production by smooth muscle cells in response to chronic hemodynamic stress.**

A, Hyaline arteriolosclerosis. The arteriolar wall is thickened with the deposition of amorphous proteinaceous material, and the lumen is markedly narrowed.



- **Complications:**

- **Most significant in the kidneys➔progressive atrophy of the kidney → chronic renal failure →<u>nephrosclerosis (glomerular scarring).</u>**

- **Other causes of hyaline arteriolosclerosis (in absence of HTN):**

1- elderly patients (normo-tensive)

2- diabetis mellitus

Nephrosclerosis is caused by an ischemic injury in the kidneys over a long period of time

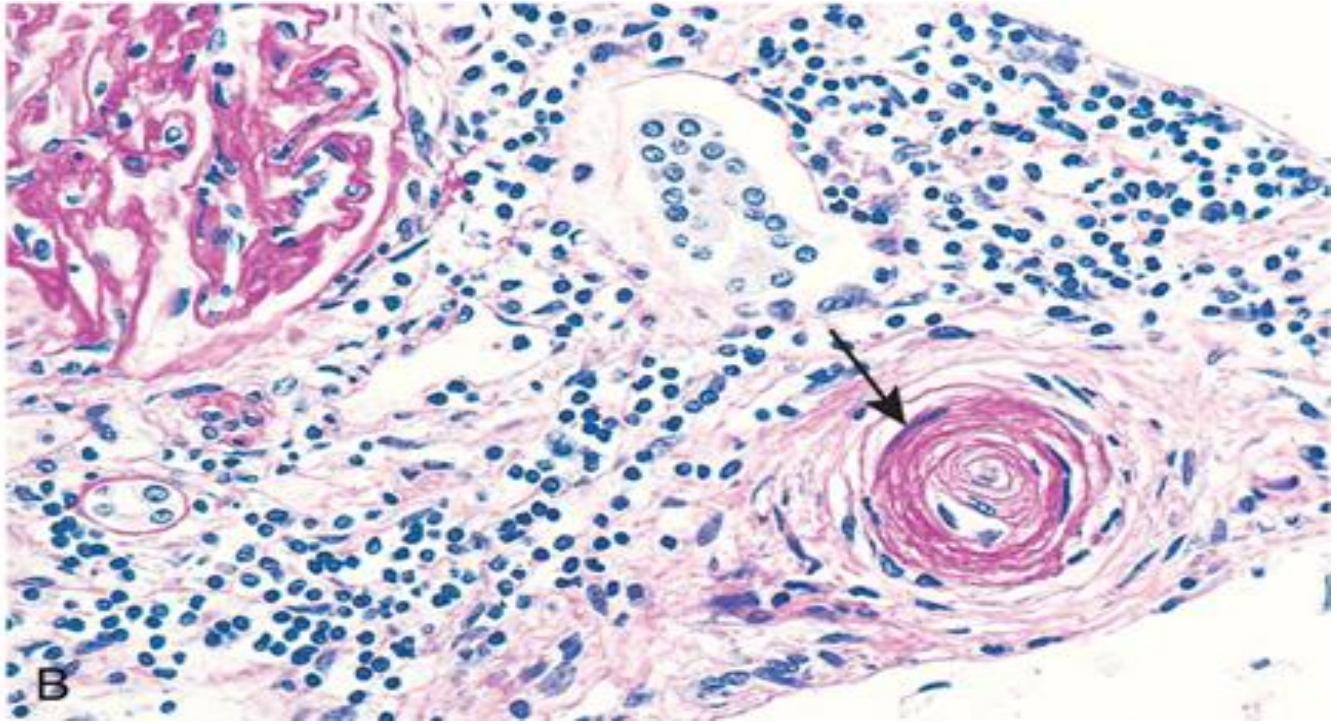
Hyperplastic arteriolosclerosis

Named hyperplastic because of the structural changes

- is more typical of **severe (malignant)** hypertension.
- "**onionskin**," concentric, laminated thickening of arteriolar walls and luminal narrowing.
- **The laminations consist of smooth muscle cells and thickened, reduplicated basement membrane.**
- In malignant hypertension these changes are accompanied by fibrinoid deposits and vessel wall necrosis **(necrotizing arteriolitis)**, which are particularly prominent in the kidney

How it is formed :
1-attacks of malignant HTN
2- micro damage In the endothelial and the basement membrane
3-repair → smooth muscle recruitment and **reduplication** of the basement membrane (deposition of new ECM)
4- several attacks of malignant HTN will lead to **onionskin** appearance(next slide).

B, Hyperplastic arteriolosclerosis ("onion-skinning") (*arrow*) causing luminal obliteration



DISORDERS OF BLOOD VESSEL HYPERREACTIVITY

- Several disorders are characterized by inappropriate or exaggerated vasoconstriction of blood vessels:

  1- Raynaud Phenomenon

  2- Myocardial Vessel Vasospasm

1- Raynaud Phenomenon

- results from exaggerated vasoconstriction (hyperactivity of the smooth muscles) of arteries and arterioles in the extremities (the fingers and toes, but also sometimes the nose, earlobes, or lips).

  -restricted blood flow induces paroxysmal pallor or cyanosis

- involved digits characteristically show "red-white-and-blue" color changes from most proximal to most distal (reflecting proximal vasodilation, central vasoconstriction, and more distal cyanosis, respectively).

  - Raynaud phenomenon can be a primary entity or may be secondary to other disorders

Primary Raynaud phenomenon

- **caused by exaggerated vasomotor responses to cold or emotional stress (intrinsic hyperreactivity of medial smooth muscle cells)**
- **affects 3% to 5% of the general population and has a predilection for young women.**
- ***Structural changes in the arterial walls are absent* except late in the course, when intimal thickening may appear.**
- **The course is usually *benign* *(clinically insignificant)***
- **chronic cases may show atrophy of the skin, subcutaneous tissues, and muscles.**
- **Ulceration and ischemic gangrene are rare.**

Secondary Raynaud phenomenon

- **refers to vascular insufficiency due to arterial disease that lead to hyperactivity of the smooth muscles caused by other entities**
- **these include SLE, scleroderma, Buerger disease, or atherosclerosis.**
- **every patient with Raynaud phenomenon should be evaluated for these secondary causes**

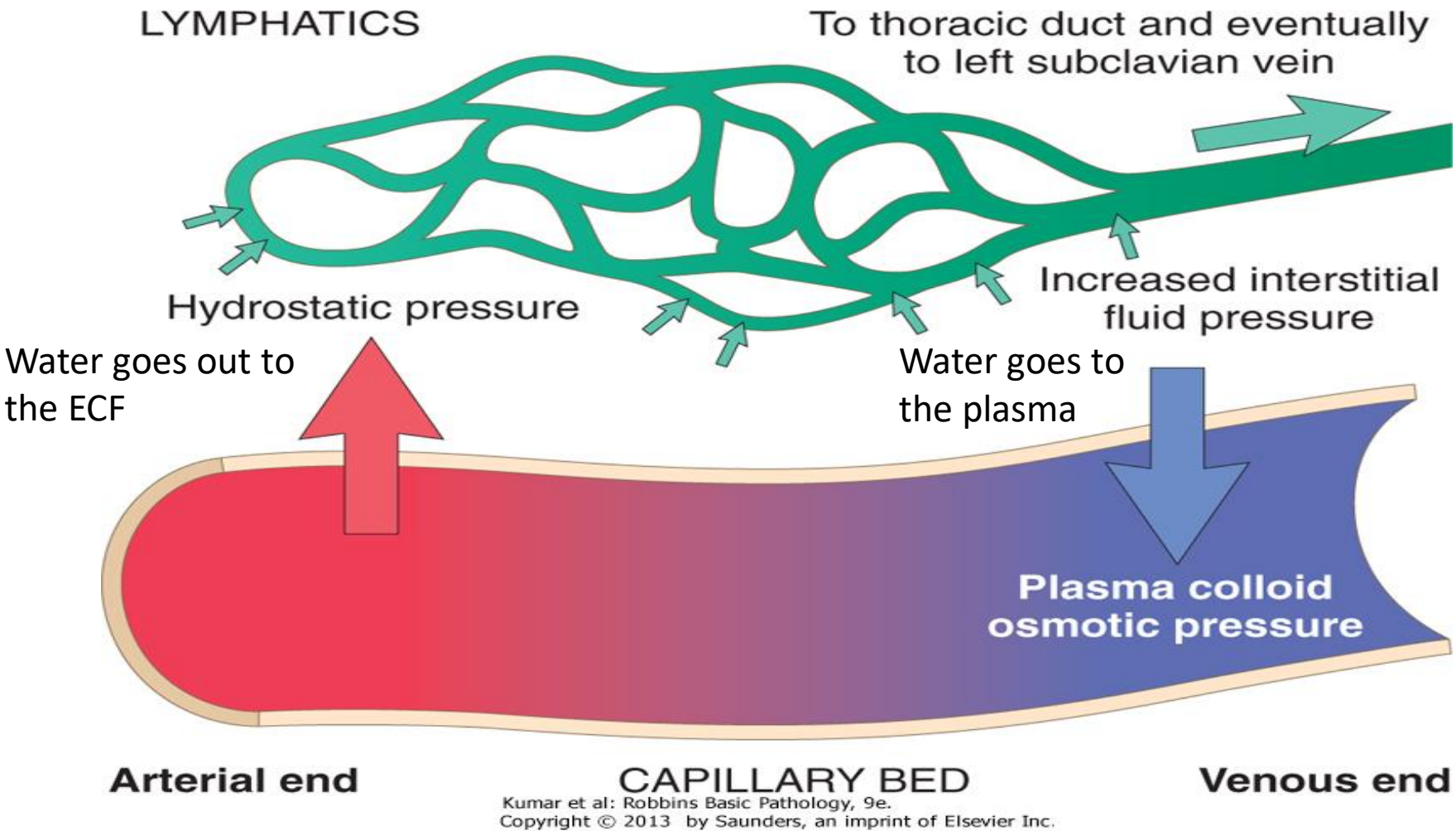
EDEMA

- 60% of lean body weight = water
  → (2/3) intracellular.
  → (1/3)extracellular (interstitial fluid)
  → 5% blood plasma.
- *edema* = an accumulation of interstitial fluid within tissues =accumulation of excess water in the interstitial fluid.
- Extravascular fluid collection in body cavities:
  - **pleural cavity (*hydrothorax*)**
  - **the pericardial cavity (*hydropericardium*)**
  - **peritoneal cavity (*hydroperitoneum*, or *ascites*).**
- *Anasarca* is severe, **generalized edema** marked by profound swelling of subcutaneous tissues and **accumulation of fluid in body cavities.**

not analogous to edema, cuz the water here accumulates in the cavities not in the interstitial fluid

The patient will have generalized edema + hydrothorax, ascites…etc

the balance of water is maintained by
Hydrostatic pressure → caused by water and electrolytes content (high in the arterial side)
Colloid osmotic pressure → caused by macro-molecules content
Lymphatic → collects water and opens in the venous circulation.



Increased Hydrostatic Pressure
Impaired Venous Return
Congestive heart failure; Constrictive pericarditis; Ascites (liver cirrhosis); Venous obstruction or compression; Thrombosis; External pressure (e.g., mass); Lower extremity inactivity with prolonged dependency
Arteriolar Dilation
Heat; Neurohumoral dysregulation
Reduced Plasma Osmotic Pressure (Hypoproteinemia)
Protein-losing glomerulopathies (nephrotic syndrome) Liver cirrhosis (ascites); Malnutrition; Protein-losing gastroenteropathy
Lymphatic Obstruction
Inflammatory; Neoplastic; Postsurgical; Postirradiation
Sodium Retention
Excessive salt intake with renal insufficiency Increased tubular reabsorption of sodium Renal hypoperfusion Increased renin-angiotensin-aldosterone secretion
Inflammation
Acute inflammation; Chronic inflammation; Angiogenesis

causes

-Increased vascular permeability → leakage of water
-the edema is usually localized

Increased Hydrostatic Pressure

- ***Local* : -impaired venous return-** e.g. DVT
- ***Generalized***: -***congestive heart failure*** *(most common):*

- reduced cardiac output leads →hypoperfusion of the kidneys→renin-angiotensin-aldosterone axis → sodium and water retention (*secondary hyperaldosteronism*).
- (vicious circle): fluid retention→ increased venous hydrostatic pressures→worsening edema.
- Treatment of generalized edema:

salt restriction

diuretics

aldosterone antagonists

Decreased venous return (venous congestion) → increased hydrostatic pressure

Reduced Plasma Osmotic Pressure

- common causes:

1- albumin is lost from the circulation

e.g. *nephrotic syndrome* → proteinuria (loss of albumin (and other plasma proteins) in the urine) .

2- albumin synthesized in inadequate amounts

e.g. **severe liver disease** (e.g., *cirrhosis*)

e.g. **protein malnutrition**

- Unfortunately, increased salt and water retention by the kidney not only fails to correct the plasma volume deficit but also exacerbates the edema, since the primary defect (low serum protein) persists

3- **gastrointestinal malabsorption**

Lymphatic Obstruction = lymphedema

- *Causes:*

1- localized obstruction caused by an inflammation.e.g. ***filariasis*** (so-called *elephantiasis*)

2- neoplastic conditions. E.g. breast cancer: Infiltration and obstruction of superficial lymphatics cause edema of the breast's overlying skin → *peau d'orange* (orange peel).

3- post surgical. e.g. breast cancer who undergo axillary lymph node resection and/or irradiation→ upper limb lymphedema

4- irradiation

Sodium and Water Retention

- leads to edema by increasing hydrostatic pressure (due to expansion of the intravascular volume) and reducing plasma osmotic pressure.
- causes: diseases that compromise renal function, including *poststreptococcal glomerulonephritis* and *acute renal failure*

Clinical Correlation

- **Subcutaneous edema**: the most common, **is important to recognize primarily because it signals potential underlying cardiac or renal disease**
- Gravity dependent edema→ while standing up it appears In the lower extremities and while laying(in the supine position) it will be around the sacrum.
- Can impair wound healing or the clearance of infections.
- **Pulmonary edema**

➢ Common causes:

- left ventricular failure - renal failure - ARDS
- inflammatory and infectious disorders of the lung.

➢ **can cause death by interfering with normal ventilatory function & impeding oxygen diffusion**

➢ creates a favorable environment for infections.

- **Brain edema**
- is life-threatening→ → **brain *herniation*** (extrude) through the foramen magnum.

Life threatening because it might compress the cardiovascular and the respiratory control centers