HYPERTENSIVE VASCULAR DISEASE

Arteriolosclerosis

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***

  → 5% of HTN patients 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

Hypertension (HTN) has the following potential complications:

- 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 (idiopathic) hypertension (95%)

2- **secondary hypertension:** Most are due to renal disease, or renal artery narrowing

(= renovascular hypertension), and to a lesser degree are due to many other conditions….

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

2- Secondary HTN:

Renal (most common of secondary)

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)

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**
- angiotensinogen polymorphisms and angiotensin II receptor variants; polymorphisms of the renin-angiotensin system.
- ? Susceptibility genes for essential hypertension: genes that control renal sodium absorption, etc.

Pathogenesis of essential HTN

- **Environmental factors**
- 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

1- Hyaline arteriolosclerosis

- **with benign hypertension.**
- **homogeneous, pink hyaline thickening of the arteriolar walls; luminal narrowing.**
- **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.**

- **Hyaline arteriolosclerosis: Complications**

- **Most significant in kidneys➔ nephrosclerosis (glomerular scarring).**

- **Other causes hyaline arteriolosclerosis:**

1- elderly patients (normo-tensive)

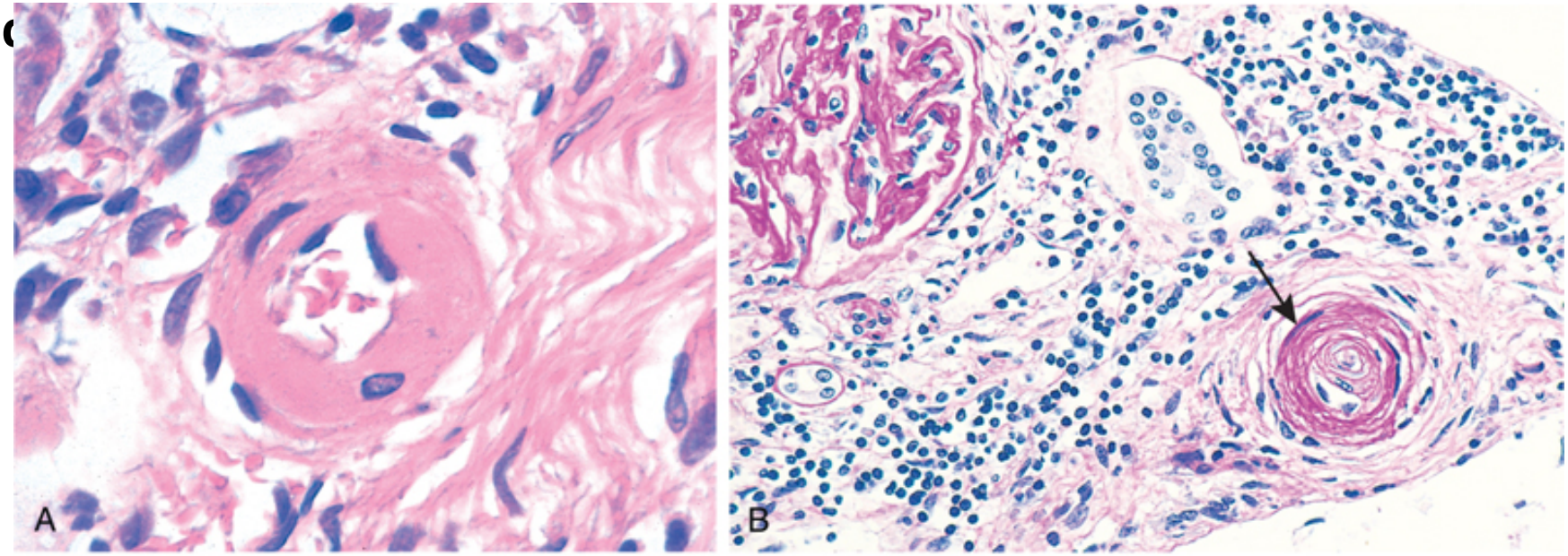
2- diabetis mellitus

2- Hyperplastic arteriolosclerosis

- With **severe (malignant)** hypertension.
- "**onionskin**" concentric laminated thickening of arteriolar walls→luminal narrowing.
- = smooth muscle cells and thickened, reduplicated basement membrane.
- In malignant hypertension → fibrinoid vessel wall necrosis **(necrotizing arteriolitis)**, which are particularly prominent in the kidney

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

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



EDEMA

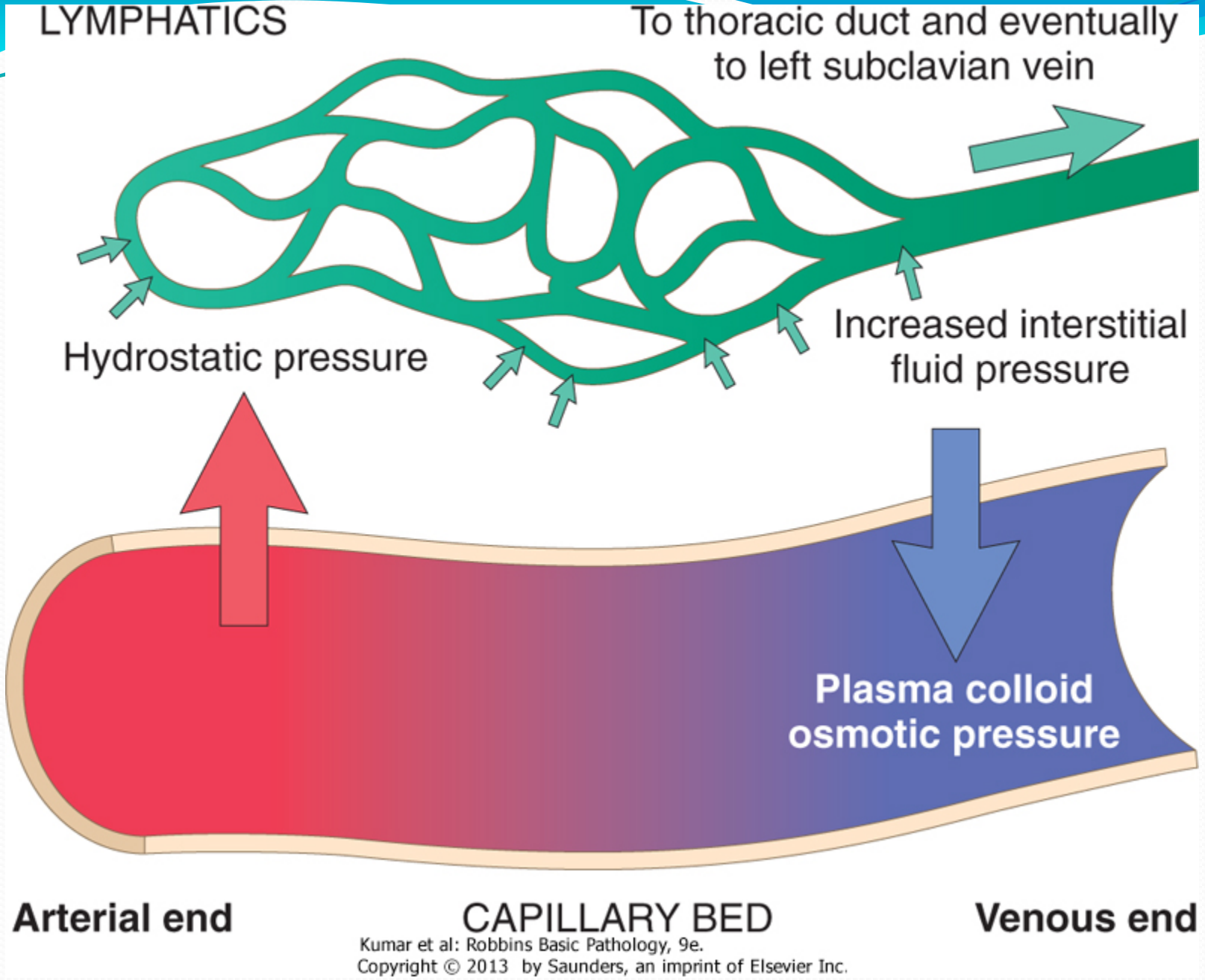
EDEMA

- 60% of lean body wt. = water
  - →(2/3) intracellular.
  - →(1/3) extracellular (interstitial fluid)
  - →5% blood plasma.

} water

- ***edema* = accumulation of interstitial fluid within tissues.**

- **Edema ≠ Extravascular fluid collection in body cavities:**
  - pleural cavity (*hydrothorax*)
  - the pericardial cavity (*hydropericardium*)
  - peritoneal cavity (*hydroperitoneum*, or *ascites*).



Mechanisms of edema

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

Clinical Correlation of edema

- **Subcutaneous edema**:

- the most common;
- important to recognize as it signals potential underlying cardiac or renal disease
- Can impair wound healing or the clearance of infections.

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- **Brain edema:**

- life-threatening→ brain *herniation* (extrude) e.g. through the foramen magnum.

- **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