PATHOLOGY TEAM 436

MEDICINE
KING SAUD UNIVERSITY

Hypertension

Revised by
شوق الأحمري & طراد الوكيل

Objectives:

1. Know the etiology, risk factors of hypertension,
2. Know the types of hypertension
3. Know the possible complications of untreated hypertension

Black: Doctor's slides.

Red: important!

Green: Doctor's notes.

Grey: Extra.

Purple: Female's slides.

Blue: Male's slides.

HYPERTENSION - INTRODUCTION

https://www.youtube.com/watch?v=9CKihqqIokI

- Common problem (25% of population)
- Asymptomatic until late- Silent Killer – painless –
- Leading risk factor – MI & Stroke
- Complications alert to diagnosis but late…

- In the early stages of HTN there are few or no symptoms.
- Hypertension is an important factor which contributes in development of:
  - Coronary heart disease.
  - Cerebrovascular accidents (stroke)
  - Cardiac hypertrophy
  - Congestive heart failure.
  - Aortic dissection.
  - Renal failure.
  - Retinopathy

DEFINITION

It is sustained diastolic pressure more than 90 mm Hg or a sustained systolic pressure more than 140 mm Hg **(>140/90)**

RISK FACTORS FOR HYPERTENSION

- **Hereditary**, Genetics-family history (most common)
- **Race**: African-Americans
- **Gender:** Men & postmenopausal women
- **Age**
- **Obesity**
- **Diet**, particularly sodium intake
- **lifestyle-stressful**
- **Heavy alcohol consumption**
- **Diabetes**
- **Use of oral contraceptives**[1] **'for long term'**
- **Sedentary or inactive lifestyle**

CLASSIFICATION:

1- BASED ON ETIOLOGY/CAUSE

- **Primary/Essential Hypertension (95%):** Mechanisms largely unknown, it is **idiopathic**[2]
- **Secondary Hypertension (5-10%):** it can be due to pathology in the renal, endocrine, vascular, or neurogenic systems.

Table 9–2 Types and Causes of Hypertension (Systolic and Diastolic)

Essential Hypertension
Accounts for 90% to 95% of all cases
Secondary Hypertension
Renal
Acute glomerulonephritis[3]
Chronic renal disease
Polycystic disease*
Renal artery stenosis**
Renal vasculitis
Renin-producing tumors
Endocrine
Adrenocortical hyperfunction (Cushing syndrome, primary aldosteronism, congenital adrenal hyperplasia, licorice ingestion)
Exogenous hormones (glucocorticoids, estrogen [including pregnancy-induced and oral contraceptives], sympathomimetics and tyramine-containing foods, monoamine oxidase inhibitors)
Pheochromocytoma[4]
Acromegaly[5]
Hypothyroidism (myxedema)
Hyperthyroidism (thyrotoxicosis)
Pregnancy-induced (pre-eclampsia)
Cardiovascular
Coarctation of aorta -Narrowing of the aorta-
Polyarteritis nodosa[6]
Increased intravascular volume
Increased cardiac output
Rigidity of the aorta
Neurologic
Psychogenic
Increased intracranial pressure
Sleep apnea (Pauses in breathing or shallow breaths while you sleep)
Acute stress, including surgery

Dr. Sufia: You should know all of them but with stress on Renal causes☺

* is an inherited kidney disorder. It causes fluid-filled cysts to form in the kidneys. PKD may impair kidney function and eventually cause kidney failure.

1 حبوب منع الحمل

2 An **idiopathy** is any disease with unknown cause or mechanism of apparently spontaneous Origin.

3. Glomerulonephritis refers to an inflammation of the glomerulus, which is the unit involved in filtration in the kidney.

4. Pheochromocytoma is a rare tumor of adrenal gland tissue. It results in the release of too much epinephrine and norepinephrine, hormones that control heart rate, metabolism, and blood pressure.

5. Acromegaly is a disorder that results from excess growth hormone (GH) after the growth plates have closed.[3] The initial symptom is typically enlargement of the hands and feet.

6.Classic polyarthritis nodosa (PAN or c-PAN) is a systemic vasculitis characterized by necrotizing inflammatory lesions that affect medium-sized and small muscular arteries, preferentially at vessel bifurcations, resulting in microaneurysm formation, aneurysmal rupture with hemorrhage, thrombosis, and, consequently, organ ischemia or infarction.

2- BASED ON CLINICAL FEATURES:

Benign:

1. The BP is at modest level (not very high).
2. Fairly stable over years to decades.
3. Compatible with long life.
4. it can be idiopathic HTN or secondary HTN.

Malignant (5%): Emergency

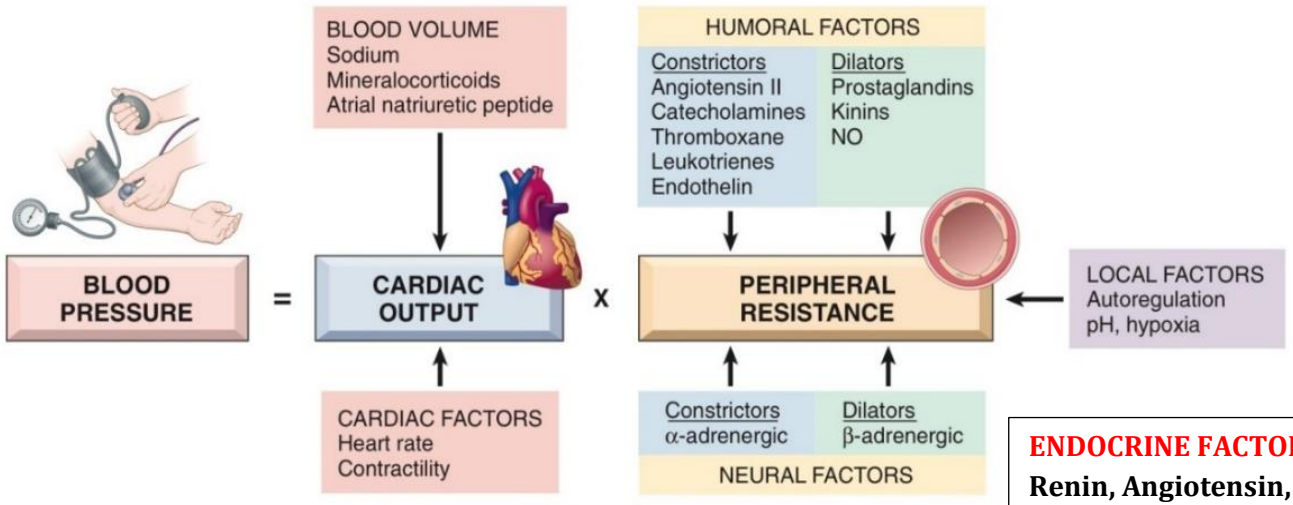
1. Rapid rising of BP which often leads to end organ damage (Kidney, Retina, Brain and Heart.).
2. It can complicate any type of HTN (i.e. essential or secondary).
3. It is seen in 5% of HTNsive patients.
4. The diastolic pressure is usually over 120mmHg.
5. It is associated with:
    - Widespread arterial necrosis and thrombosis
    - Rapid development renal failure
    - Retinal hemorrhage and exudate, with/without papilledema
    - Hypertensive encephalopathy[3]
    - Left ventricular failure
    - Leads to death in 1 or 2 years if untreated.

Regulation of blood pressure:

https://www.youtube.com/watch?v=tANEMMGoy9k&spfreload=10
https://www.youtube.com/watch?v=HzfAfsZVtYU

Two hemodynamic variables are involved in the regulation of BP: cardiac output and peripheral vascular resistance. **BP = Cardiac Output x Peripheral Resistance**

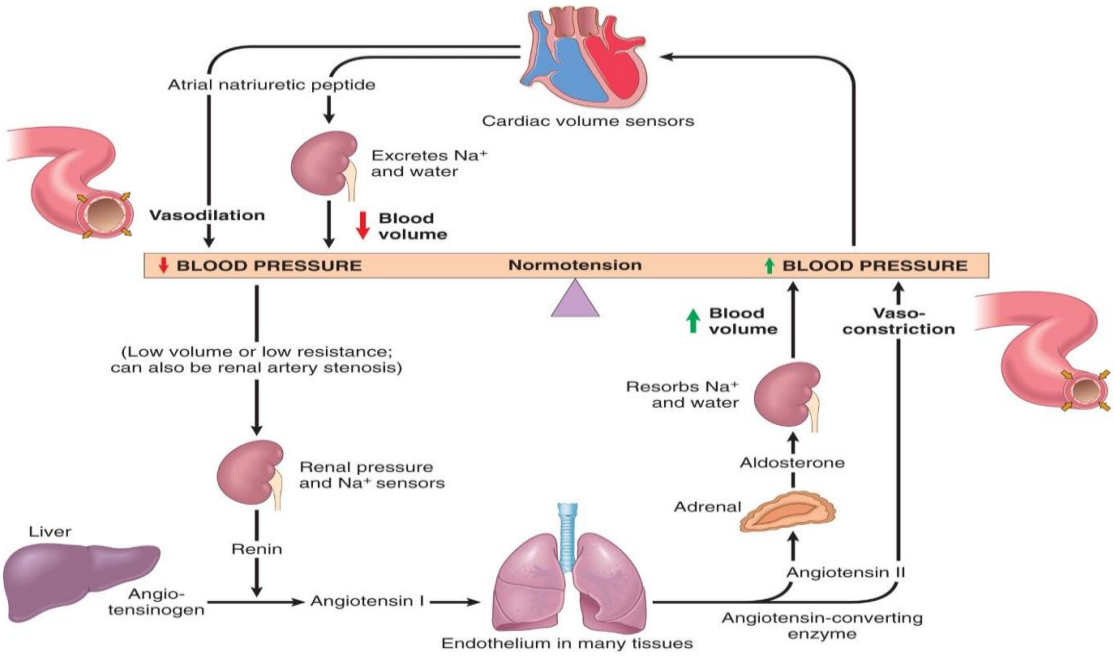
- **Cardiac output:** is affected by blood volume and is dependent on sodium concentrations.
- **Peripheral resistance:** it is the resistance of the arteries to blood flow. As the arteries constrict, the resistance increases and as they dilate, resistance decreases. Is regulated predominantly at the level of the arterioles (also known as resistance vessels) and is influenced by neural and hormonal inputs. It is determined by three factors:
  1- Autonomic activity: sympathetic activity constricts peripheral arteries.
  2- Pharmacologic agents: vasoconstrictor drugs increase resistance while vasodilator drugs decrease it.
  3- Blood viscosity: increased viscosity increases resistance.
- Normal BP is maintained by a balance between factors that induce vasoconstriction (e.g., angiotensin II and catecholamines) and factors that induce vasodilation (e.g., kinins, prostaglandins, and nitric oxide). An increased blood flow in the arterioles induces vasoconstriction to protect tissues against hyperperfusion. C.O itself increases constriction.



ENDOCRINE FACTORS:
Renin, Angiotensin, ADH, Aldosterone

[3] A metabolic, Toxic, or degenerative disease of the brain.

Extra



*Because of the **difference** between Dr. Sufia & Dr. Ahmed in the way the explained the pathogenesis. We wrote the two ways starting with Dr.Ahmed way because it is shorter.

Pathogenesis*:

1. **Defect in sodium excretion:** Decreased Na excretion causes an obligatory **increase in fluid volume** and **increased cardiac output**, thereby elevating blood pressure. At the new higher blood pressure, the kidneys excrete additional Na. Thus, a new steady state of Na excretion is achieved, but at the expense of an elevated blood pressure.
2. Defect in cell membrane function (Na/Ca transport)
3. Increased sympathetic/ vasoconstrictive response

Essential HTN occurs when the relationship between cardiac output and peripheral resistance is altered. Multiple **genetic and environmental factors** ultimately **increase the cardiac output and/or peripheral resistance (BP = Cardiac Output x Peripheral Resistance)**

1. Genetic factors: There is a strong genetic component (family history). A genetic effect is involved in making people more susceptible or less susceptible to high salt diet etc.

high salt>high water>high C.O>increase risk of hypertension.

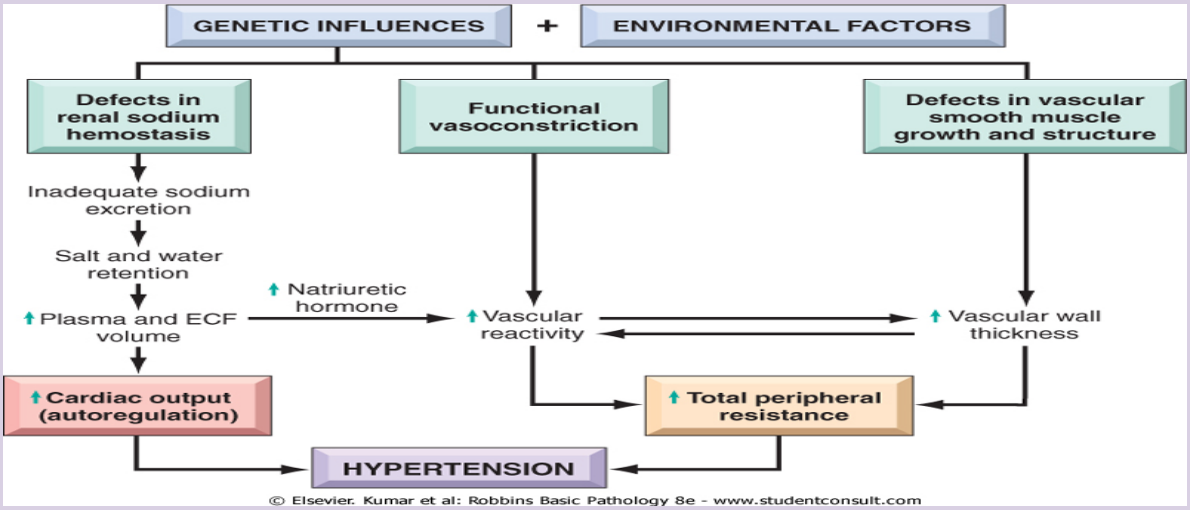
A. Defect in renal sodium homeostasis: reduced renal sodium excretion is a key initiating event in most forms of essential hypertension. This decreased sodium excretion will result in increase in fluid volume
B. Functional vasoconstriction: abnormality in vascular tone such as increased sympathetic stimulation will cause vasoconstriction leading to increased peripheral resistance.
C. Structural abnormality in vascular smooth muscle also leads to increased peripheral resistance.

Both B,C increases PR

D. Also rare gene disorders can cause HTN by increasing renal sodium resorption e.g. Liddle syndrome. Liddle syndrome is an inherited autosomal dominant type of HTN, that begins in childhood. It is caused by mutations of the epithelial sodium channel protein (ENaC) which leads to increased sodium reabsorption in in the renal tubules (followed by water), which leads to hypertension. Reabsorption of sodium is also correlates with potassium loss (**hypokalemia**).

2. Environmental factors: stress, obesity, smoking, physical inactivity, and heavy consumption of salt also play a role. هي نفسها الهاي ريسكس

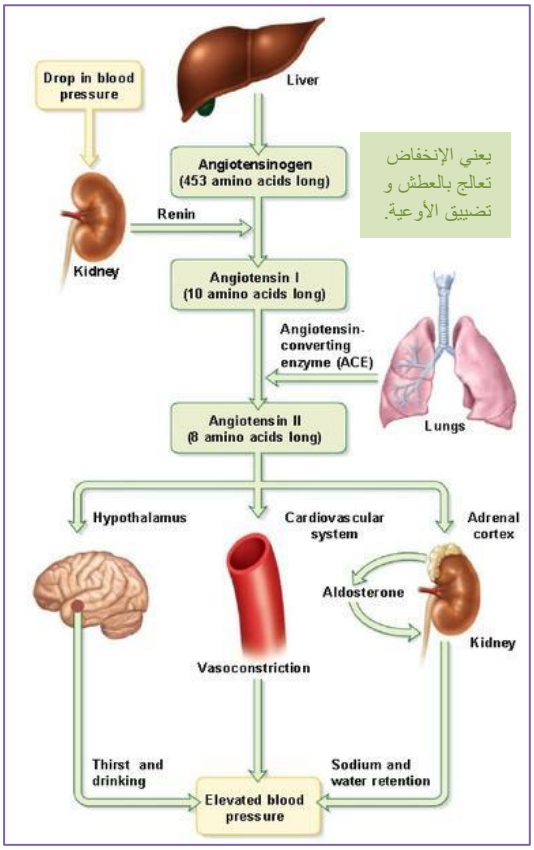
NOTE: In hypertension, both increased blood volume and increased peripheral resistance contribute to the increased pressure. However reduced renal sodium excretion in the presence of normal arterial pressure (initially) is probably a key initiating event. 'the most trigger'



Atrial natriuretic peptide / factor / Hormone

(Cardionatrine / Cardiodilatine / atriopeptin) عكس الألدوستيرون بعمله

- It is a protein (polypeptide) hormone secreted by the heart muscle cells in the atria of heart (atrial myocytes). 'Right side of the heart'
- It is a powerful vasodilator and is involved in the homeostatic balance of body water, sodium, potassium and fat.
- It is released in response to high blood volume. It acts to reduce the water, sodium and adipose loads on the circulatory system, thereby reducing blood pressure.
- It has exactly the opposite function of the aldosterone secreted by the zona glomerulosa[4].
- **In the kidney:**
  - decreases sodium reabsorption and increases water loss.
  - Inhibits renin secretion, thereby inhibiting the renin–angiotensin–aldosterone system.
- **In adrenal gland:**
  - Reduces aldosterone secretion by the zona glomerulosa of the adrenal cortex.
- **In arterioles:** Promotes vasodilatation.
- **In adipose tissue:** Increases the release of free fatty acids from adipose tissue



ENDOCRINE FACTORS: role of renin-angiotensin- aldosterone in regulating BP

[4] The zona glomerulosa of the adrenal gland is the most superficial layer of adrenal cortex, lying directly the renal capsule.

Dr. Sufia: We like to ask here☺.

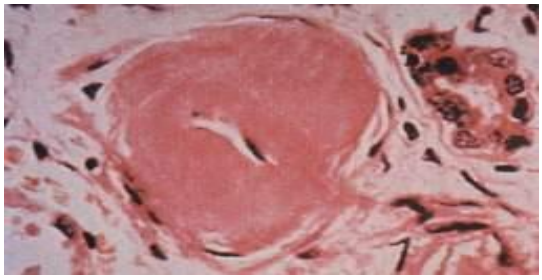
انتبهوا للي تحته خط ممكن يلخبطونكم فيها.

Morphology:

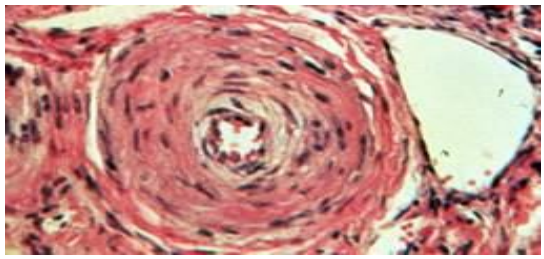
- *Large Blood Vessels (Macroangiopathy)*
  ***Ather*osclerosis**. HT is a major risk factor in AS.

- *Small Blood Vessels (Microangiopathy)*
  - ***Arterio*sclerosis**.
    1. Hyaline arteriolosclerosis*:
       - Characteristic of benign hypertension.
       - Can also be seen in elderly without hypertension and in diabetic patients.
       - Leads to benign nephrosclerosis due to diffuse renal ischemia.
    2. Hyperplastic arteriolosclerosis**:
       - Characteristic of malignant hypertension.
       - Can show (**onion-skinning**)*** on histology causing luminal obliteration of vascular lumen
       - May be associated with necrotizing arteriolitis (fibrinoid necrosis).

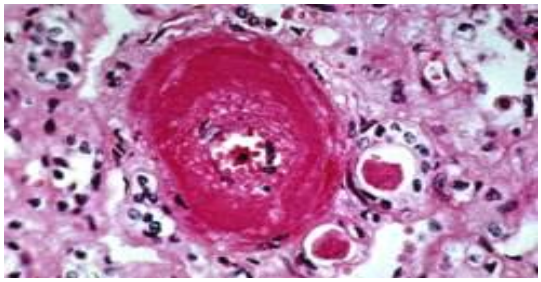
*Hyaline/Benign Arteriolosclerosis



**Hyperplastic/Malignant Arteriolosclerosis

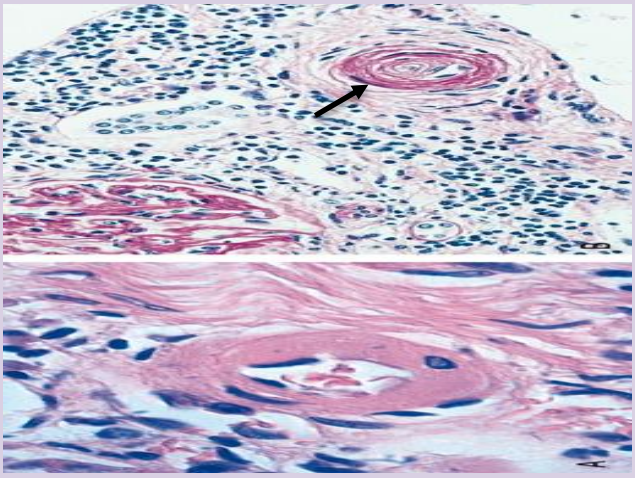


***Onion skinning



Vascular pathology in hypertension:

- **Hyaline arteriolosclerosis:** hyalinosis of arteriolar wall with narrowing of lumen
- **B. Hyperplastic arteriolosclerosis (onion skinning)** causing luminal obliteration [5](arrow) of vascular lumen with secondary ischemic changes, manifested by wrinkling of the glomerular capillary vessels at the upper left (periodic acid–Schiff [PAS] stain).



Left ventricular cardiac hypertrophy (left sided hypertensive cardiomyopathy/ hypertensive heart disease):

- Longstanding poorly treated HTN leads to left sided hypertensive heart disease.
- Hypertrophy of the heart is an adaptive response to pressure overload due to HTN.
- HTN induces left ventricular pressure overload, which leads to hypertrophy of the left ventricle with increase in the weight of the heart. The free LV wall is > 2cm and the weight of the heart is > 500 grams.

[5] Complete removal, as by disease, degeneration, surgical procedure, or irradiation, or by filling up of a space with fibrous tissue or inflammation.

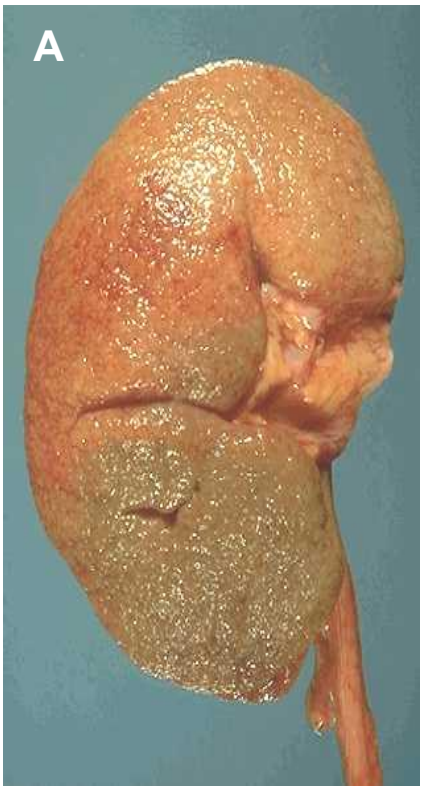
Complications/Organ damage in HTN: After 20-25 years

Cardiovascular

- Left ventricular hypertrophy (left sided hypertensive cardiomyopathy/ hypertensive heart disease).
- Coronary heart disease.
- Aortic dissection.

When we have resistance in the systemic circulation> increase contraction> LV hypertrophy. نفس النادي لما نتدرب كثير تكبر العضلة

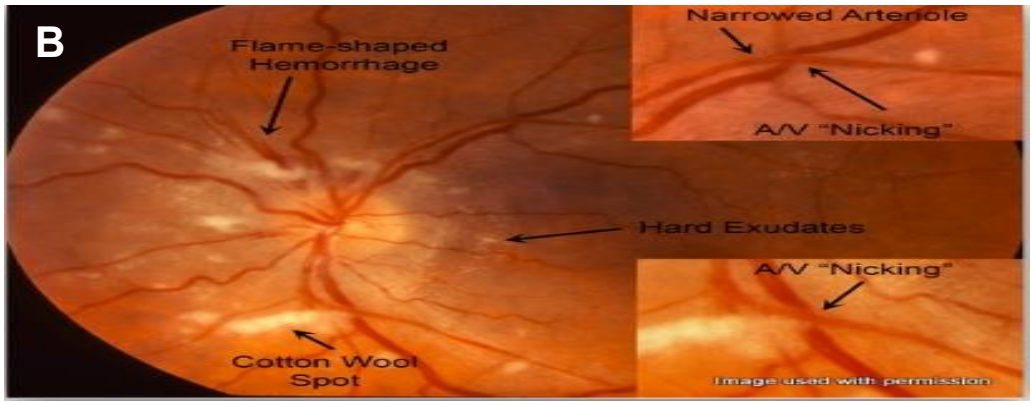
Kidney:



- Benign nephrosclerosis (photo A)
- Renal failure in untreated or in malignant hypertension

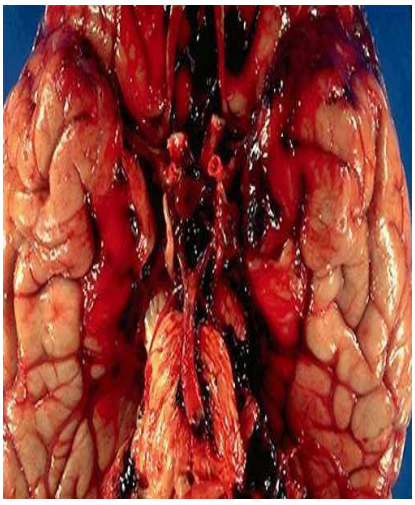
Eyes:

- Hypertensive retinopathy (photo B) is especially seen in malignant hypertension.

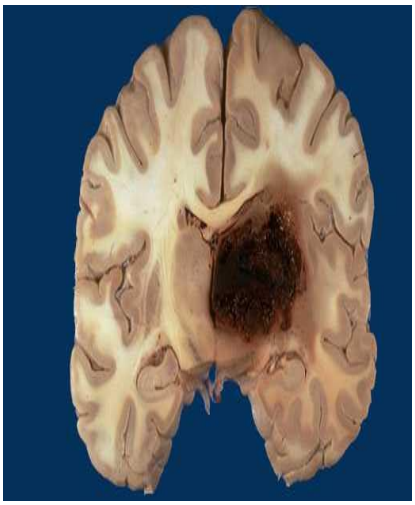


Brain: (Comes suddenly)

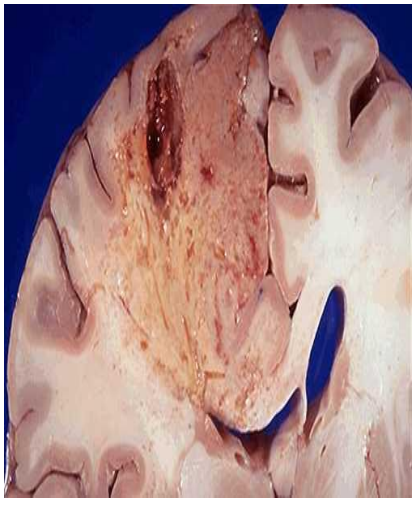
- Haemorrhage, infarction leading to Cerebrovascular accidents



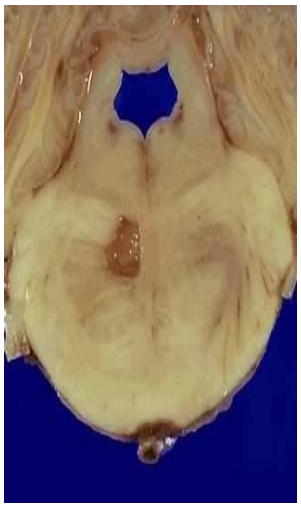
Subarachnoid Haemorrhage



Cerebral Hemorrhage

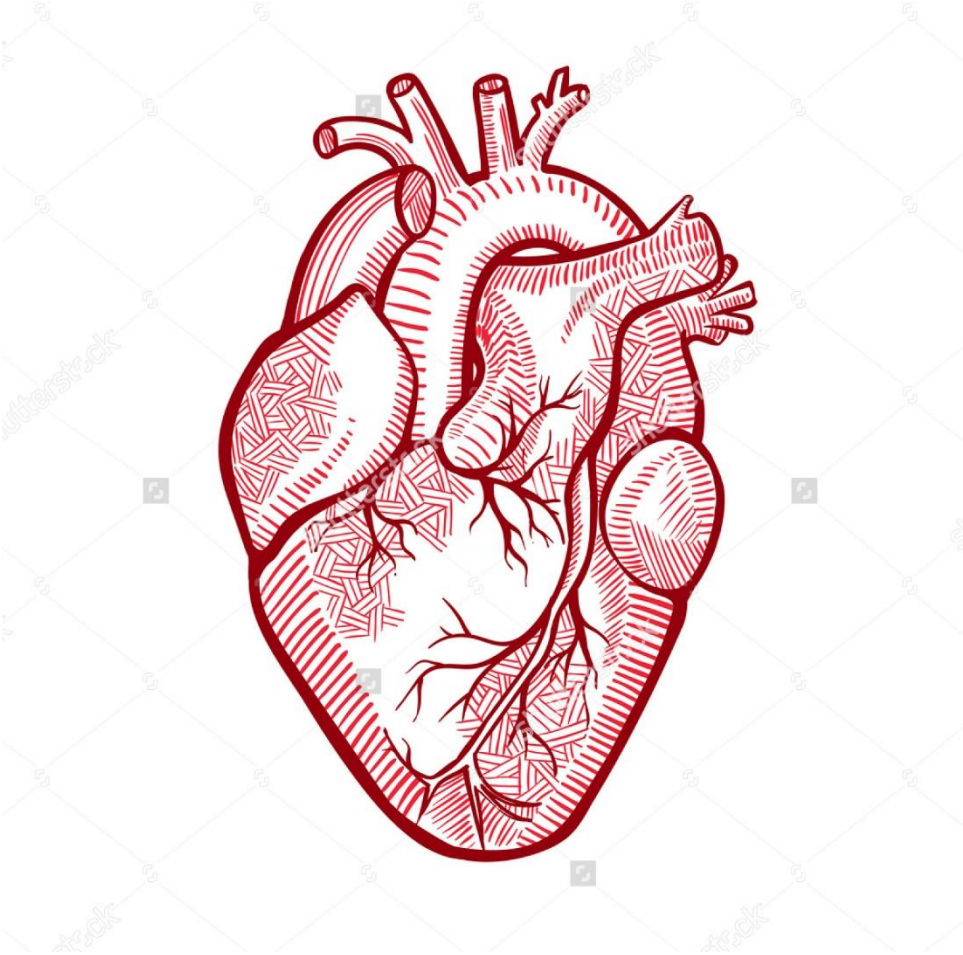


Cerebral infraction



Lacunar Infarct

"اللهم لا سهل إلا ما جعلته سهلًا و أنت تجعل الحزن إذا شئت سهلًا"



MCQs: https://onedrive.live.com/view.aspx?resid=E0947849CE6A90D1!132&ithint=file%2cpptx&app=PowerPoint&authkey=!ABtjf7EZ-CY2YdA

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