* **Functional vascular disorders**
* **Raynaud’s phenomenon**
* **Raynaud’s phenomenon**
* **Refers to**
* **Intermittent ,bilateral attacks of ischemia of the fingers or toes, and sometimes ears or nose.**
* **It clinically manifests as:**
* **Pallor (blanching) followed by cynosis (blue) followed by redness**
* **Occurs following exposure to cold and then rewarming.**
* **Sometimes attacks precipitated by emotional stimuli.**
* **Reflects**
* **Spasm of local small arteries or arterioles.**
* **Classified into two categories:**
* **Idiopathic Raynaud’s phenomenon or Raynaud’s disease**
* **Secondary Raynaud’s phenomenon**
* **Idiopathic Raynaud’s phenomenon or Raynaud’s disease**
* **Occurs as an isolated disorder.**
* **Typically occurs in young, otherwise healthy women.**
* **Of uncertain etiology, it reflects exaggerated vasomotor response to cold or emotion causing vasoconstriction.**
* **Fingers and toes become white 🡪 blue when exposed to cold.**
* **On warming , they turn red.**
* **Secondary Raynaud’s phenomenon**
* **Occurs as a part of a number of systemic disease of connective tissue etc.**
* **Secondary causes include:**
* **Systemic sclerosis (Scleroderma) \*\***
* **MC initial manifestation.**
* **CREST syndrome**
* **Systemic lupus erythethomatosus (SLE)\*\***
* **Thromboangitis obliterans (TAO)**
* **Ergot poisoning (vasoconstriction)**
* **Cryglobulinemia ( patients with RA or HCV)**
* **Secondary Raynaud’s phenomenon**
* **Clinical:**
* **Cold temperatures and stress are stimuli that may trigger the color changes of the fingers 🡪 white  blue  red**
* **Ears and nose cyanotic**
* **Often relived by warmth.**
* **Vessel changes:**
* **Normal initially**
* **Later – show thickening of intima and hypertrophy of tunica media**
* **Hypertension**
* **Defined as systolic blood pressure >140mm Hg and diastolic blood pressure >90 mm Hg for a sustained period.**
* **Hypertension predisposes to development of:**
* **Coronary artery disease**
* **Cerbro-vascular accidents**
* **Cardiac hypertrophy 🡪 heart failure**
* **Aortic dissection**
* **Renal failure**
* **Pathophysiology of HT**
* **Blood pressure (BP) = Cardiac output (CO) X Total peripheral resistance (TPR).**
* **Cardiac output (CO ) is dependent upon**
* **blood volume (equates with sodium homeostasis)**
* **force of contraction and**
* **Heart rate.**
* **Total peripheral resistance:**
* **Vasodilation: decreases TPR**
* **Vasoconstriction : increases TPR.**
* **Role of kidney in regulating BP**
* **The renin-angiotensin-aldosterone system.**
* **Renin (from JGC) converts plasma angiotensinogen into angiotensin I.**
* **Angiotensin I converted into Angiotensin II by ACE.**
* **Angiotensin II increases BP by:**
* **Increasing peripheral resistance**
* **Stimulation of aldosterone secretion 🡪 Na reabsorption**
* **Role of Sodium in hypertension**
* **Na retention 🡪 increase in plasma volume 🡪 increase in SV 🡪 increase in CO🡪 increase in systolic blood pressure.**
* **Excess sodium 🡪 enters smooth muscle cells of arterioles 🡪 opens calcium channels 🡪 contraction of SMC 🡪 vasoconstriction 🡪 increase in TPR 🡪 increase in diastolic blood pressure.**
* **Types of hypertension**
* **Essential**
* **Secondary**
* **Essential hypertension**
* **HT of unknown etiology**
* **Accounts for 95% of cases of HT**
* **More common in blacks**
* **Pathogenesis:**
* **reduced renal sodium excretion due to genetic factors**
* **vasoconstriction of arterioles due to unknown factors.**
* **Secondary hypertension**
* **Is secondary to known causes, including:**
* **Renal disease:**
* **Narrowing of renal arteries:**
* **Renovascular HT (MC).**
* **Glomerulonephritis, Polycystic renal disease**
* **Adrenal disease: Primary aldosteronism or Conn syndrome, Cushing syndrome, Pheochromocytoma.**
* **Thyroid disease: Grave’s disease.**
* **Coarctation of aorta**
* **Toxemia of pregnancy**
* **Renovascular hypertension**
* **Is the most common secondary cause of HT in adults.**
* **Pathologic features:**
* **Elderly men: atherosclerotic plaque partially blocks blood flow at the renal artery orifice.**
* **Young to middle aged women: fibromuscular hyperplasia (hyperplasia of SMC 🡪 narrow lumen)**
* **In either condition the affected kidney is small and shrunken owing to persistent ischemia.**
* **Renovascular hypertension**
* **Pathogenesis:**
* **Decreased renal arterial blood flow activates renin angiotensin aldosterone system**
* **Angiotensin II vasoconstricts peripheral resistance arterioles.**
* **Aldosterone increases sodium retention.**
* **Clinical findings:**
* **abrupt onset of HT: due to elevated plasma renin activity.**
* **Involved kidney has increased plasma renin activity in renal vein**
* **Presence of abdominal bruit**
* **due to turbulence of blood flow through the narrow renal artery.**
* **Complications of hypertension**
* **Cardiovascular: Concentric left ventricular hypertrophy (most common), acute MI.**
* **CNS: stroke due to an intracerebral hematoma or rupture of berry aneurysm**
* **Complications of hypertension**
* **Kidneys:**
* **Hyaline arteriolosclerosis:**
* **Narrows lumen of arterioles 🡪**
* **Ischemic injury 🡪**
* **Loss of renal parenchyma**
* **= benign Nephrosclerosis**
* **Shrunken kidney (cortical atrophy)**
* **Retina:**
* **hypertensive retinopathy with hemorrhages of retinal vessels, exudates, papilledema (swelling of the optic disc due to increased cerebral pressure)**
* **Malignant hypertension**
* **Occurs in 5% of patients with either**
* **essential or secondary HT.**
* **Death in 1-2 years if not treated.**
* **Characterized by:**
* **sudden increase in BP >240/>100 mmHg.**
* **Complications:**
* **Renal failure (hyperplastic arteriolosclerosis) , retinal hemorrhage, papilledema.**