- 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.
  - <u>Angiotensin II increases BP by:</u>
    - 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 → <u>increase in diastolic blood pressure</u>.

- 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.