• 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 cyanosis (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 \( \rightarrow \) 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 erythematosus (SLE)**
- Thromboangitis obliterans (TAO)
- Ergot poisoning (vasoconstriction)
- Cryoglobulinemia (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 relieved 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) \times \text{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 $\rightarrow$ 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.