HYPERTENSIVE VASCULAR DISEASE
Cutoffs in diagnosing hypertension in clinical practice ➔ sustained diastolic pressures > 90 mm Hg, or sustained systolic pressures > 140 mm Hg

**Malignant hypertension**

➔ A small percentage of HTN patients (5%) 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 complications:

- stroke (CVD)
- multi-infarct dementia
- atherosclerotic coronary heart disease
- cardiac hypertrophy and heart failure (*hypertensive heart disease*)
- aortic dissection
- renal failure
## Essential HTN
Accounts for 90% to 95% of all cases

## Secondary HTN

### Renal
- 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
most cases (95%) are idiopathic (essential hypertension).

Most of the remaining cases (secondary hypertension) are due to primary renal disease, renal artery narrowing (renovascular hypertension), or adrenal disorders. The remainder are:

Several relatively rare single-gene disorders cause hypertension by affecting renal sodium resorption.

e.g. Gene defects in enzymes involved in aldosterone metabolism → increased aldosterone secretion, increased salt and water resorption, and plasma volume expansion

e.g. Mutations in proteins that affect sodium resorption (as in Liddle syndrome, which is caused by mutations in Na Channel)
- **Genetic factors**
- familial clustering of hypertension
- HTN has been linked to specific angiotensinogen polymorphisms and angiotensin II receptor variants; polymorphisms of the renin-angiotensin system.
- Susceptibility genes for essential hypertension are currently **unknown** but probably include those that control renal sodium absorption, etc.

- **Environmental factors**
- such as 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
Hyaline arteriolosclerosis

- associated with benign hypertension.
- marked by homogeneous, pink hyaline thickening of the arteriolar walls, and luminal narrowing.
- Results from 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.

Complications:
- in the kidneys ➔ nephrosclerosis (glomerular scarring).

Other causes of hyaline arteriolosclerosis (in absence of HTN):
1- elderly patients (normo-tensive)
2- diabetic mellitus
Hyperplastic arteriolosclerosis is more typical of severe hypertension. "onionskin," concentric, laminated thickening of arteriolar walls and luminal narrowing. The laminations consist of smooth muscle cells and thickened, reduplicated basement membrane. In malignant hypertension these changes are accompanied by fibrinoid deposits and 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) causing luminal obliteration.
DISORDERS OF BLOOD VESSEL HYPERREACTIVITY

Several disorders are characterized by inappropriate or exaggerated vasoconstriction of blood vessels:
1- Raynaud Phenomenon
2- Myocardial Vessel Vasospasm
1- Raynaud Phenomenon

- results from exaggerated vasoconstriction of arteries and arterioles in the extremities (the fingers and toes, but also sometimes the nose, earlobes, or lips).
- restricted blood flow induces paroxysmal pallor or cyanosis

- involved digits characteristically show "red-white-and-blue" color changes from most proximal to most distal (reflecting proximal vasodilation, central vasoconstriction, and more distal cyanosis, respectively).

- Raynaud phenomenon can be a primary entity or may be secondary to other disorders.
Primary Raynaud phenomenon

- (previously called Raynaud disease)
- caused by exaggerated vasomotor responses to cold or emotion (intrinsic hyperreactivity of medial smooth muscle cells)
- affects 3% to 5% of the general population and has a predilection for young women.
- Structural changes in the arterial walls are absent except late in the course, when intimal thickening may appear.
- The course usually is benign
- chronic cases may show atrophy of the skin, subcutaneous tissues, and muscles.
- Ulceration and ischemic gangrene are rare.
Secondary Raynaud phenomenon

- refers to vascular insufficiency due to arterial disease caused by other entities
- these include SLE, scleroderma, Buerger disease, or atherosclerosis.
- every patient with Raynaud phenomenon should be evaluated for these secondary causes
2- Myocardial Vessel Vasospasm

- Causes:
  1. vasoactive mediators $\rightarrow$ prolonged vascular contraction;
     $\rightarrow$ endogenous (e.g., epinephrine released by pheochromocytomas) or exogenous (cocaine or phenylephrine).
  2. Elevated thyroid hormone $\rightarrow$ increase sensitivity of vessels to catecholamines
  3. autoantibodies and T cells in scleroderma $\rightarrow$ vascular instability and vasospasm.
  4. extreme psychological stress (release of catecholamines)
Cardiac Raynaud

- When vasospasm of cardiac arterial or arteriolar beds is of sufficient duration (20 to 30 minutes), myocardial infarction occurs—Cardiac Raynaud

- Elevated levels of catechols:
  1. increase heart rate
  2. increase myocardial contractility
  3. coronary vasospasm.

- The outcome:
  1. myocardial infarction
  2. sudden cardiac death (fatal arrhythmia)
  3. ischemic dilated cardiomyopathy → so-called Takotsubo cardiomyopathy (also called "broken heart syndrome," because of association with emotional force).

- Histologic findings:
  - acute → microscopic areas of necrosis characterized by myocyte hypercontraction (contraction band necrosis)
  - subacute and chronic cases → microscopic foci of granulation tissue and/or scar