



# Hypertension

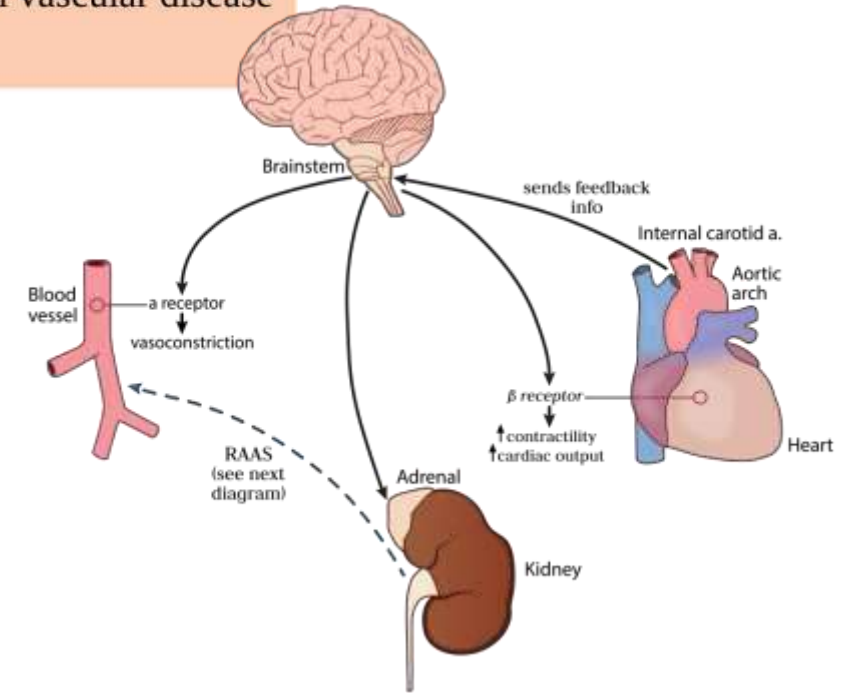
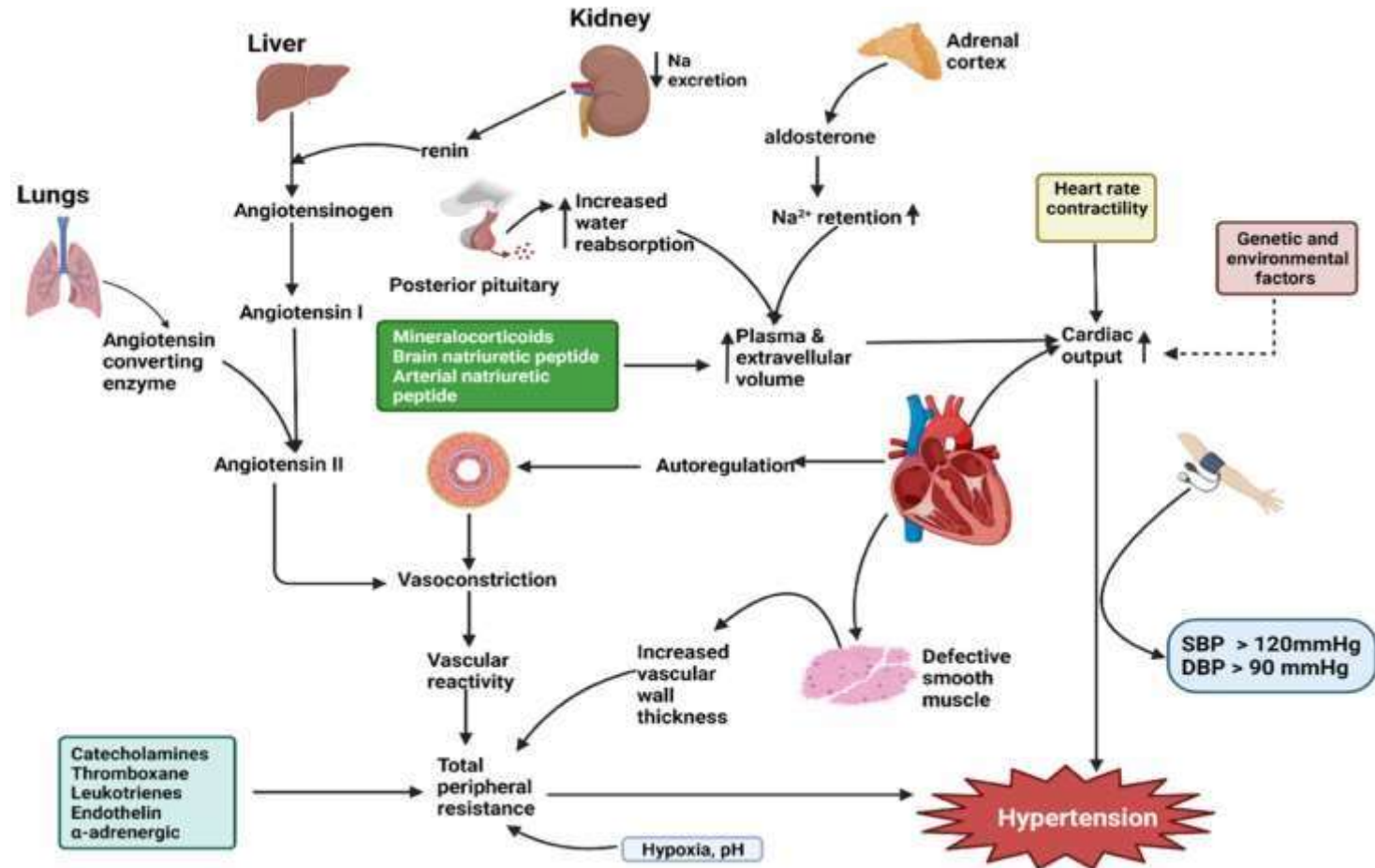


# Hypertension

- Condition in which blood pressure is regulated maladaptively , elevating blood pressure **over 140/90mmHg**
- Isolated systolic hypertension: systolic blood pressure is elevated, diastolic is not
- Isolated diastolic hypertension: diastolic blood pressure is elevated, systolic is not

## Pathophysiology of the Effects of HTN on the Heart

- Increased systemic vascular resistance (afterload) → concentric left ventricle hypertrophy (LVH) → decreased LV function. As a result, the chamber dilates → symptoms and signs of heart failure.
- HTN accelerates atherosclerosis, leading to a higher incidence of CAD (as well as peripheral vascular disease and stroke).



# PATHO-GENESIS OF ESSENTIAL HTN

Resistance small arteriols <1mm diameter.

- Vascular Intimal layer proliferation
- Muscular layer wall Thickening.
- Reduced vascular lumen diameter.
- Secondary Calcium and Hyaline deposition .
- Ending in vascular- ATHEROMA- atherosclerosis.

Increased peripheral vascular resistance Tissues hypo-perfusion and tissues ischemia. Arteriolar wall micro- aneurysm formation

# PATHO-GENESIS OF ESSENTIAL HTN

Larger arteriols  $>1\text{mm}$  diameter-

- Thickened internal elastic lamina.
- Smooth muscles wall hypertrophy .
- Collagen- fibrous tissues formation- Fibrosis.
- Arterial wall calcification.ONION - SKIN appearance.
- Blood vessels becomes dilated tortuous With loss of wall compliance.

# AETIOLOGY-MULTI-FACTORIAL

HTN- a complex interaction between Genetics and Environment –life style factors.

95% pt. Idiopathic HTN

5% pt. Secondary HTN

Essential - Idiopathic- HTN-No specific underlying cause can be identified.

# CAUSES

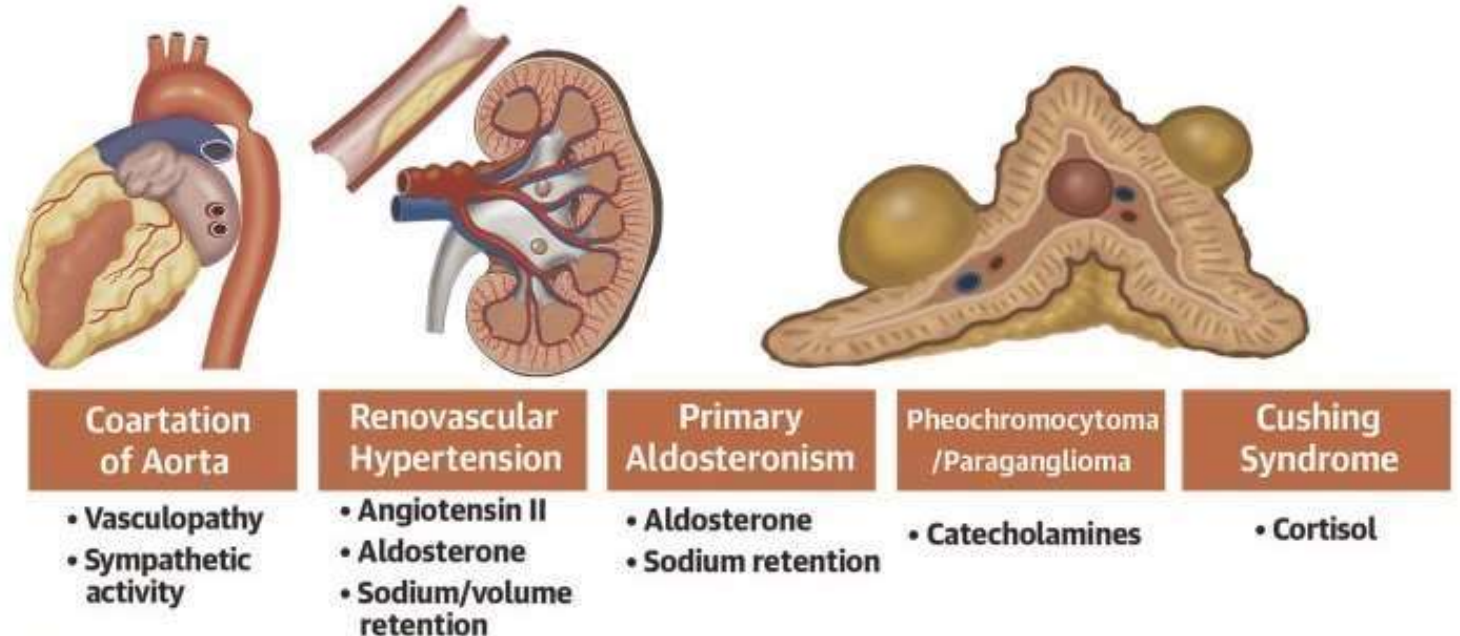
## Primary/essential hypertension

- Most cases (90%)

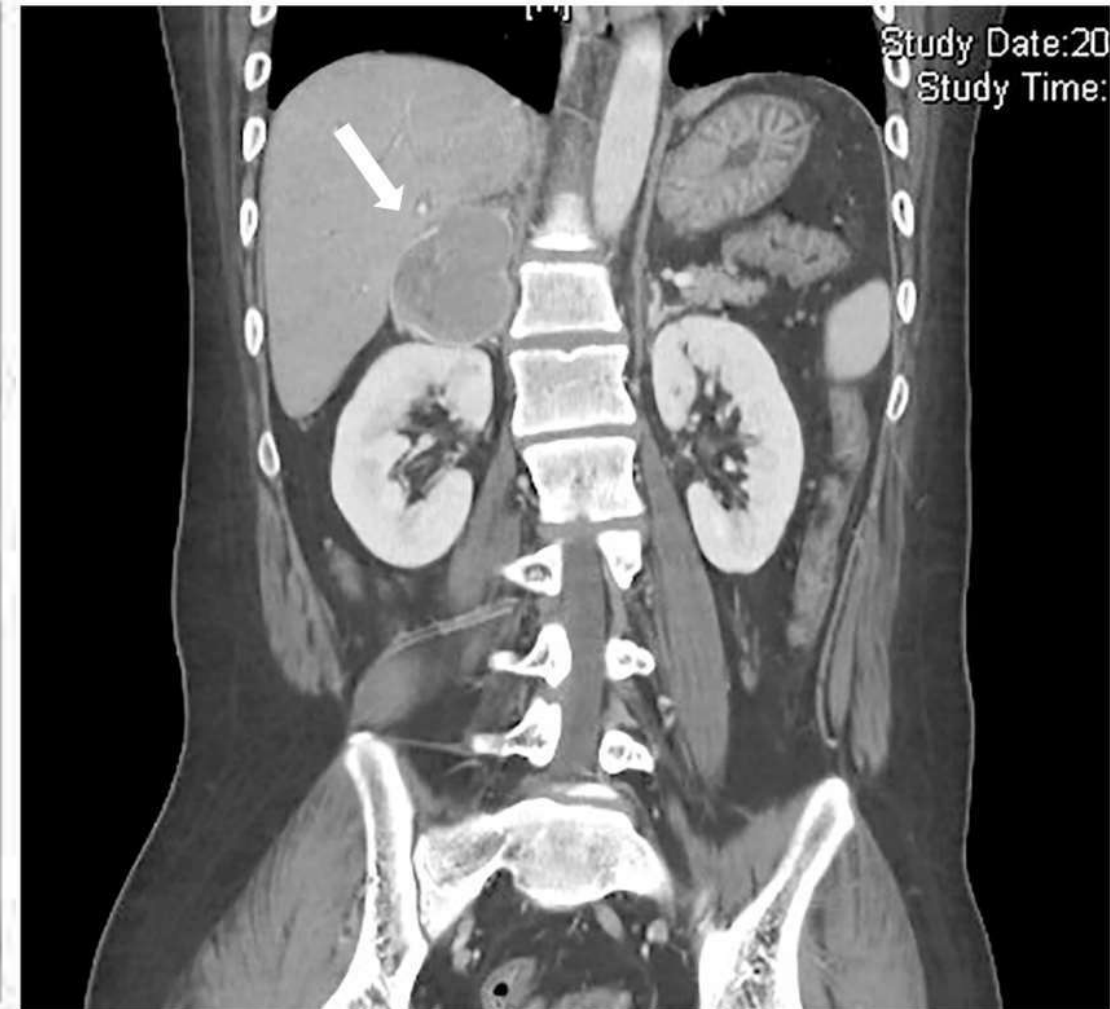
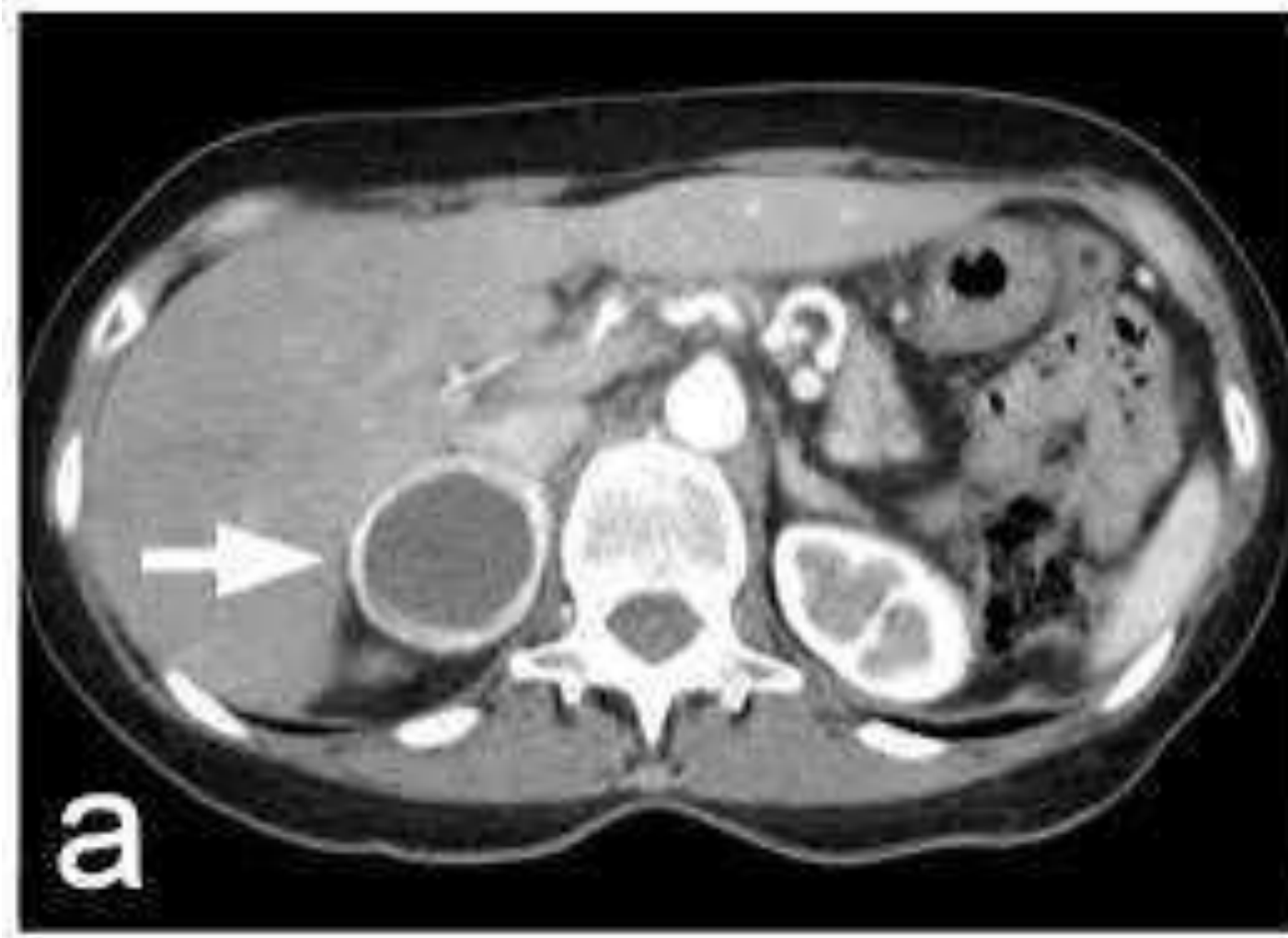
## Secondary hypertension

- Known etiology, often reversible
- **Renovascular hypertension:** anything partially obstructing blood flow to kidneys (e.g. atherosclerosis, vasculitis, fibromuscular dysplasia).
  - Decreased blood flow to kidneys → kidneys secrete renin → renin converts angiotensinogen to angiotensin I → angiotensin converting enzyme converts angiotensin I to angiotensin II (active) → angiotensin II effects:
    - **Vasoconstriction:** directly increases blood pressure
    - **Stimulation of sodium reabsorption:** increases water reabsorption
    - Stimulation of adrenal cortex to release aldosterone from adrenal cortex → aldosterone increases reabsorption of sodium + water → increased volume → high blood pressure

- **Primary hyperaldosteronism:** increased aldosterone → increased reabsorption of sodium + water → increased volume → increased blood pressure
- **Nonsteroidal anti-inflammatory drugs (NSAIDs)** → inhibit cyclooxygenase in kidneys → ↓ production of PGE-2 (renal vasodilator) → vasoconstriction of afferent arterioles in kidneys → ↓ renal blood flow,



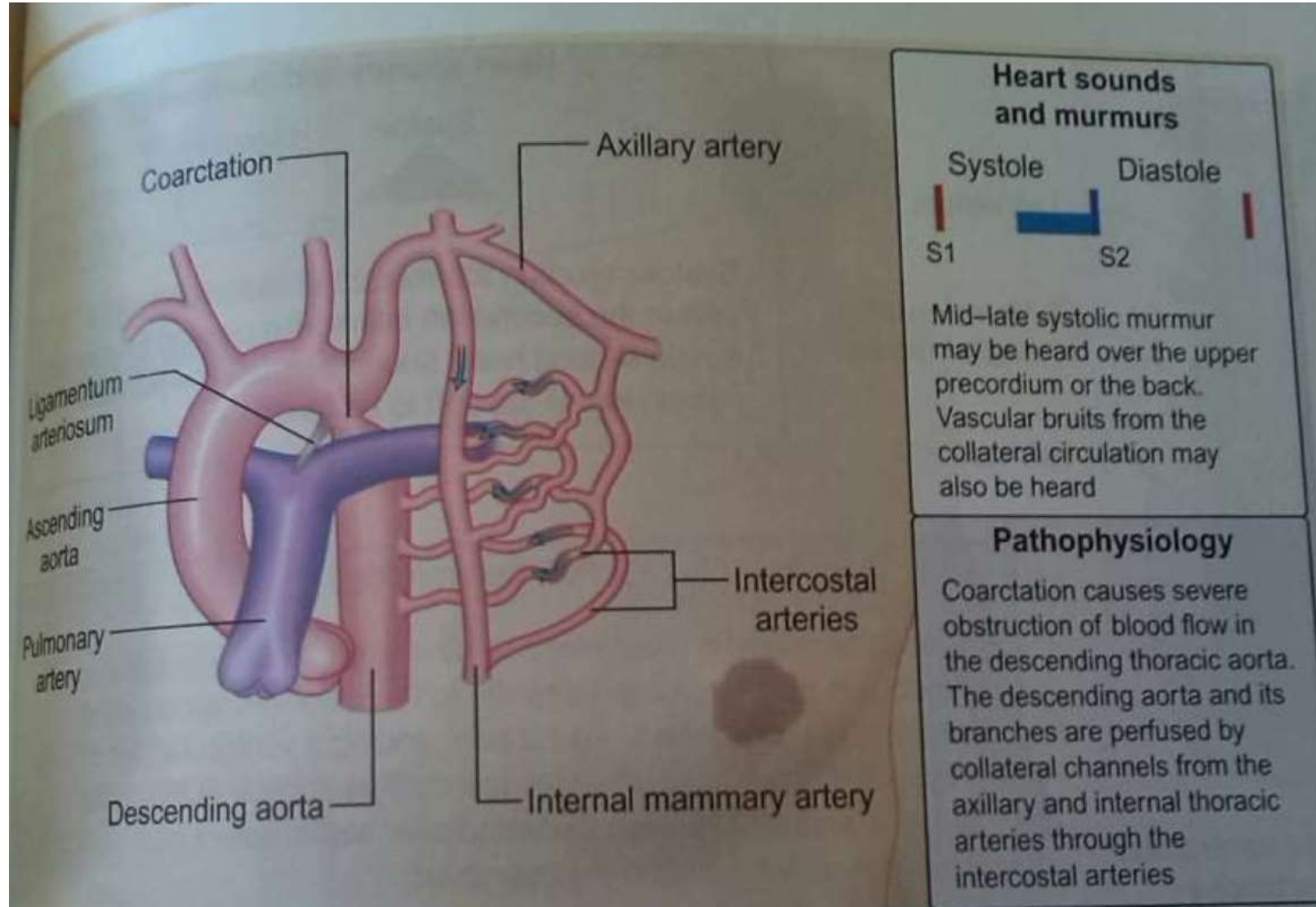
# PHAEOCHROMOCYTOMA



# Conn's adenoma



# CO-ARCTATION-OF -AORTA



- **Preeclampsia/eclampsia in pregnancy**: mechanism unknown
- **Chronic kidney disease**: fluid overload, sodium retention
- **Medications**: oral contraceptives, decongestants, estrogen , appetite suppressants, chronic steroids, tricyclic antidepressants (TCAS), nonsteroidal anti-inflammatory drugs (NSAIDs)
  - **Coarctation of the aorta**
  - **Cocaine**, other stimulants
  - **Obstructive sleep apnea** (OSA)

**Quick**

**HIT**



Birth control pills are the most common secondary cause of HTN in young women.

# Risk factors

1. **Age**—both systolic and diastolic BP increase with age
2. **Gender**—more common in men (gap narrows over age 60 years); men have higher complication rates
3. **Race**—it is twice as common in African-American patients than in Caucasian patients; African-American patients have higher complication rates (stroke, renal failure, heart disease)
4. **Obesity, sedentary lifestyle, dyslipidemia**
5. **Family history**
6. **Increased sodium intake**—this correlates with increased prevalence in large populations, but not in individuals; individual susceptibility to the effects of high salt intake varies
7. **Alcohol**

# Genetics factor

GENETIC FACTORS HTN has complex genetic disorders large number of Genes may be involved in HTN

1 RENIN- ANGIOTENSIN - ALDOSTERON -SYSTEM- GENE HIGH- -  
RENIN- HTN- YOUNG.LOW RENIN- HTN- ELDERLY- BLACK

2 ADRENERGIC RECEPTORS- GENE-Peripheral vascular resistance-  
and vascular tone

3 VASCULAR ENDOTHELIAL FUNCTIONS –GENE-Vasoconstrictors-  
Cytokines Angiotensin-II- Endothelin- Thromboxin A2.Vasodilators –  
Cytokines Prostaglandin- NO- Prostacyclin

4 Na- and salt Sensitivity –GENE - Salt Sensitive HTN

5 Metabolic GENES-

Regulator of insulin receptors .

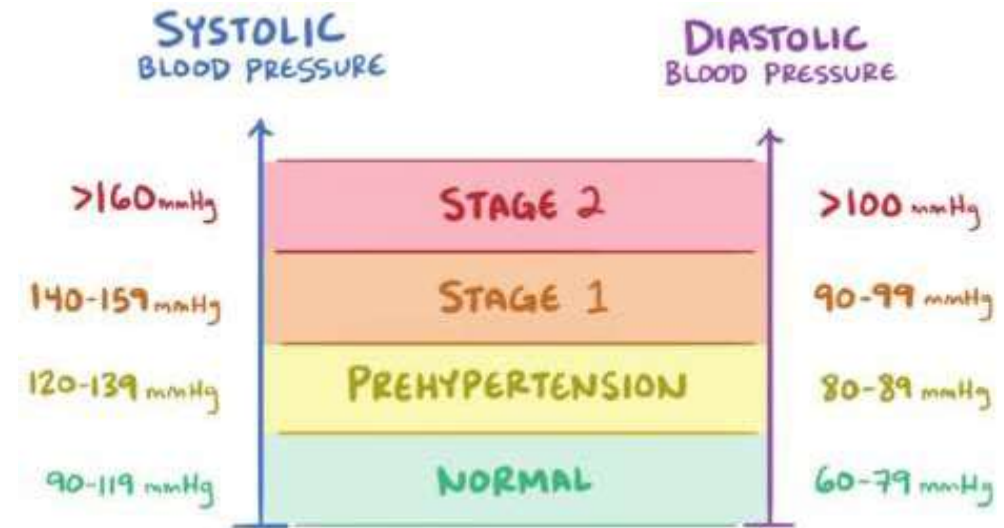
Hyperinsulinemia and insulin Resistance.

SYNDROME-X- Metabolic syn.

Marked central Obesity- Dyslipidemia- DMT2- HTN

# Classification (for adults >18 years)

- **Normal** : systolic BP < 120 and diastolic BP < 80
- **Elevate** : dsystolic BP 120 to 139 or diastolic BP 80 to 89
- **Stage I** ( Mild ) : systolic BP 140 to 159 or diastolic BP 90 to 99
- **Stage II** ( Moderate ) : systolic BP  $\geq 160$  or diastolic BP  $\geq 100$
- **Stage III** ( Severe ) : systoloc BP > 180 or diastolic > 110
- **Isolated systolic hypertension** :
  - Grade I : systolic 140-159 , diastolic < 90
  - Grade II : systolic > 160 , diastolic < 90



| Category                       | Systolic BP<br>(mmHg) | Diastolic BP<br>(mmHg) |
|--------------------------------|-----------------------|------------------------|
| BP                             |                       |                        |
| Optimal                        | < 120                 | < 80                   |
| Normal                         | < 130                 | 85                     |
| High normal                    | 130 – 139             | 85 – 89                |
| Hypertension                   |                       |                        |
| Grade 1 (mild)                 | 140 – 159             | 90 – 99                |
| Grade 2 (moderate)             | 160 – 179             | 100 – 109              |
| Grade 3 (severe)               | > 180                 | > 110                  |
| Isolated systolic hypertension |                       |                        |
| Grade 1                        | 140 – 159             | < 90                   |
| Grade 2                        | > 160                 | < 90                   |

# Definitions

- **Hypertensive urgency** : severe HTN (typically **systolic BP >180** and/or **diastolic BP >120**) in an asymptomatic patient
- **Hypertensive emergency**: severe HTN with end-organ damage (e.g., neurologic changes, myocardial ischemia, aortic dissection)
- **White coat hypertension**: elevated blood pressure when measured in the office setting, but not out of the office
- **Masked hypertension**: elevated blood pressure outside the office, but does not meet criteria when measured in the office.

## Quick HIT

HTN is an asymptomatic disease (“silent killer”)—it causes insidious damage to the following target organs: heart, eyes, CNS, and kidneys.

- **CHF is a common end-result of untreated HTN** as LVH occurs.
- Most deaths due to HTN are ultimately due to MI or CHF.
- HTN predisposes the patient to peripheral artery disease (PAD).
- HTN is associated with increased incidence of abdominal aortic aneurysm (AAA) and **aortic dissection**.



# Complications

1. The major complications of HTN are **cardiac complications** (coronary artery disease [CAD], CHF with left ventricular hypertrophy [LVH]), stroke, and renal failure. These account for the majority of deaths associated with untreated HTN.



# Main complications of persistent High blood pressure

## Brain:

- Cerebrovascular accident (*strokes*)
- Hypertensive encephalopathy:
  - confusion*
  - headache*
  - convulsion*

## Blood:

- Elevated sugar levels

## Retina of eye:

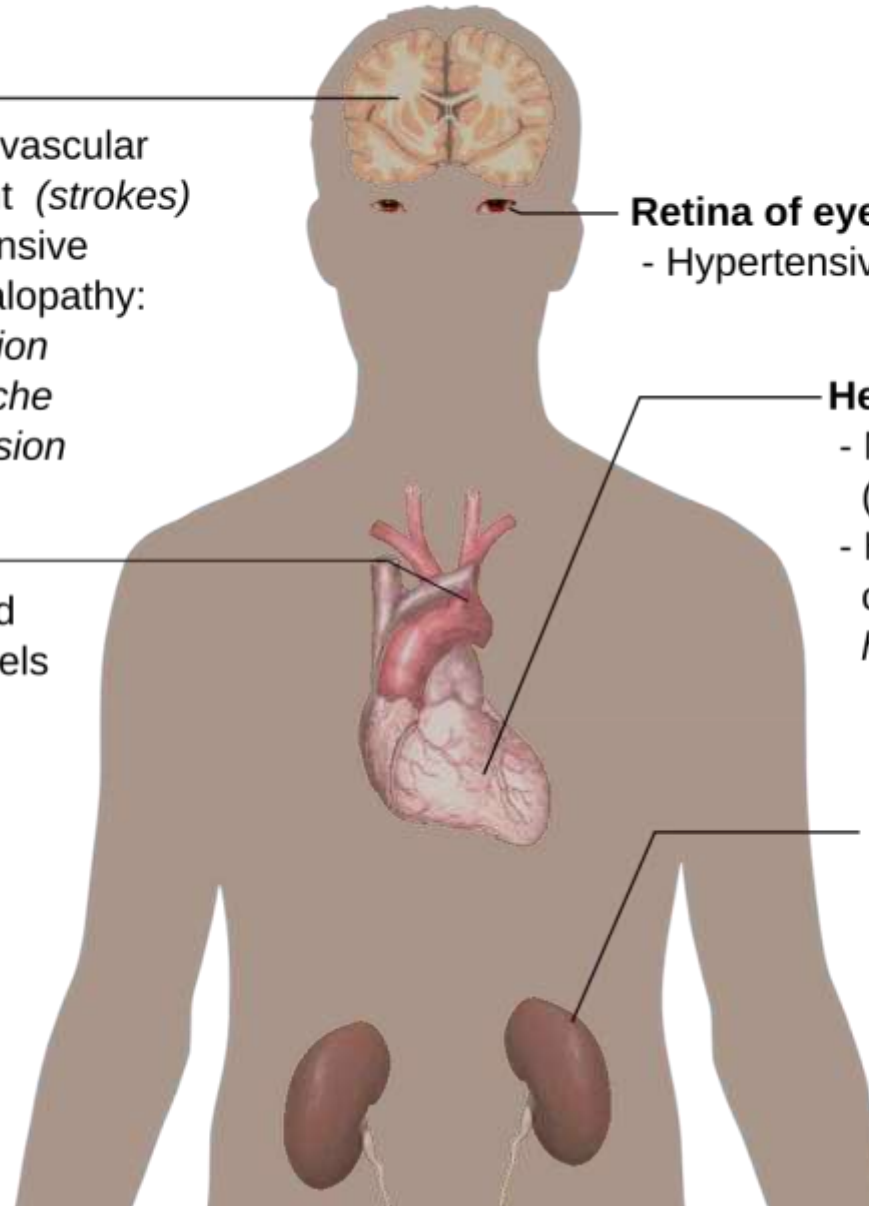
- Hypertensive retinopathy

## Heart:

- Myocardial infarction (*heart attack*)
- Hypertensive cardiomyopathy:  
*heart failure*

## Kidneys:

- Hypertensive nephropathy:  
*chronic renal failure*



## 2. Target organ damage:

- a. **Cardiovascular system** Effects on the heart are most important. HTN is a major risk factor for CAD, with resultant angina and MI.
- b. **Eyes (retinal changes)** Early changes—arteriovenous nicking (discontinuity in the retinal vein secondary to thickened arterial walls) and cotton wool spots (infarction of the nerve fiber layer in the retina) can cause visual disturbances and scotomata (Figure 12-1). More serious disease—hemorrhages and exudates. Papilledema—an ominous finding seen with severely elevated BP.

# Hypertensive retinopathy

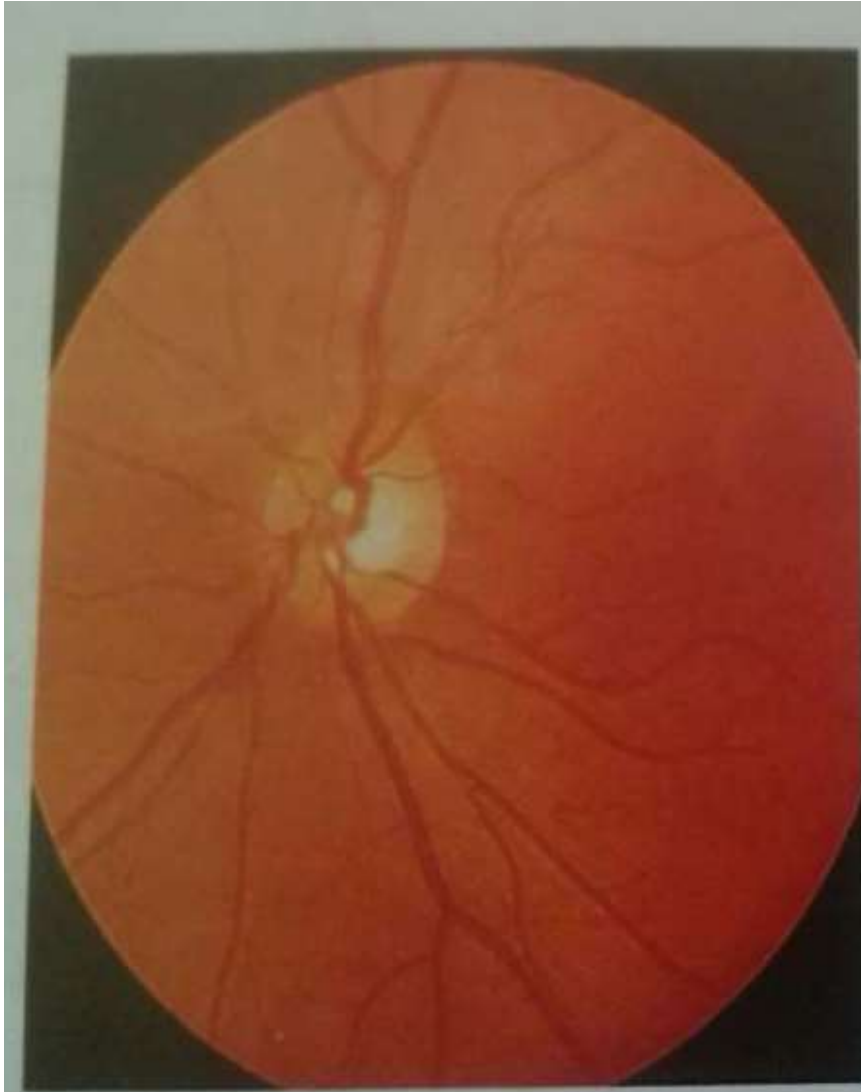
**Grade 1** Arteriolar thickening, tortuosity and increased reflectiveness (“silver wiring”).

**Grade 2** Grade 1 plus constriction of veins at arterial crossings (“arteriovenous nipping”).

**Grade 3** Grade 2 plus evidence of retinal ischaemia (flame – shaped or blot haemorrhages and “cotton wool” exudates).

**Grade 4** Grade 3 plus papilloedema

# HTN-RETINOPATHY



A



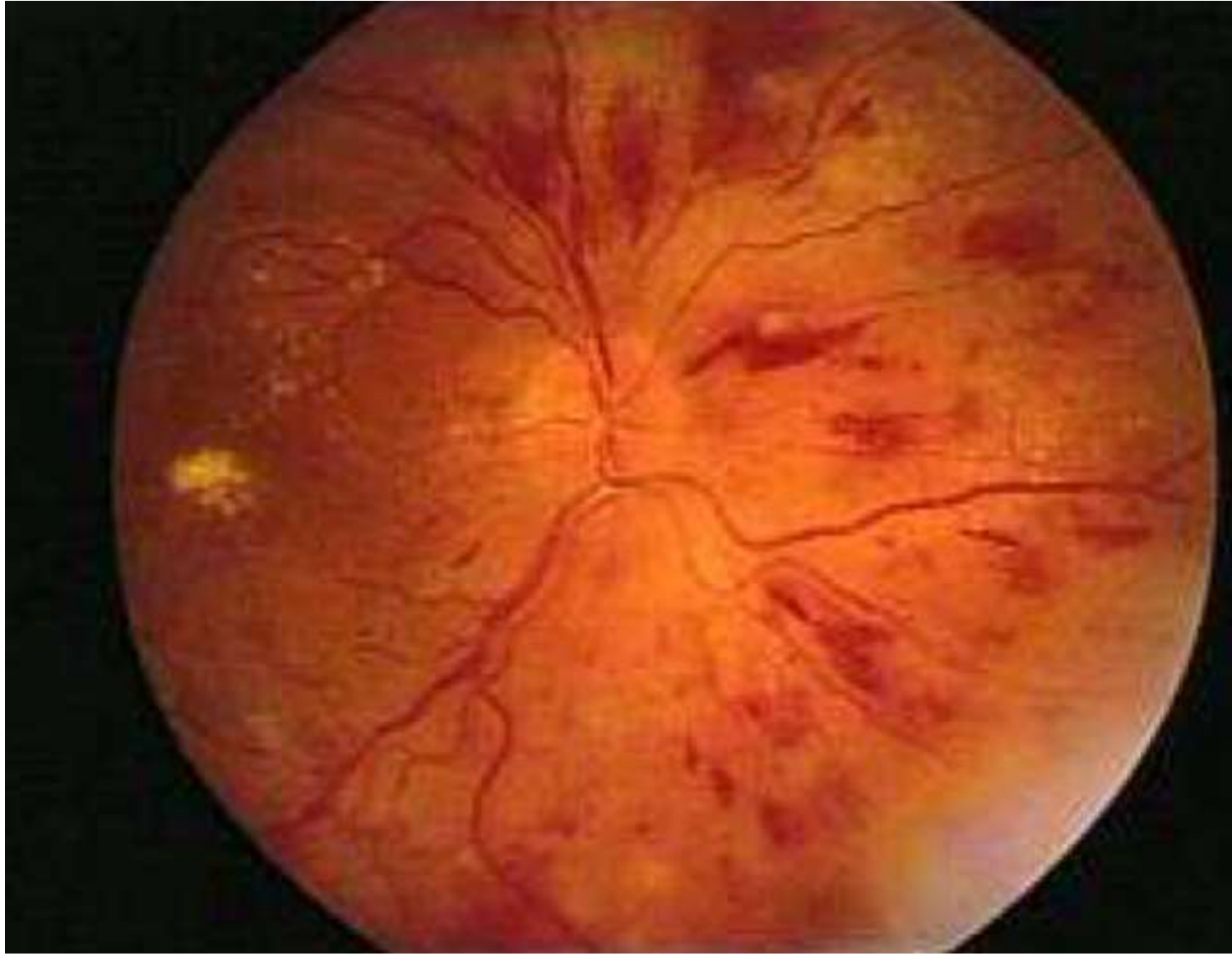
B

# HTN-RETINOPATHY



Figure 14.117 Fundus showing hypertensive changes: Grade 4 retinopathy with papilloedema, haemorrhages and exudates.

# HTN-RETINOPATHY PAPILLOEDEMA





## Target Organ Damage

- Heart—LVH, MI, CHF
- Brain—stroke, TIA
- Chronic kidney disease
- Peripheral arterial disease, AAA, aortic dissection
- Retinopathy

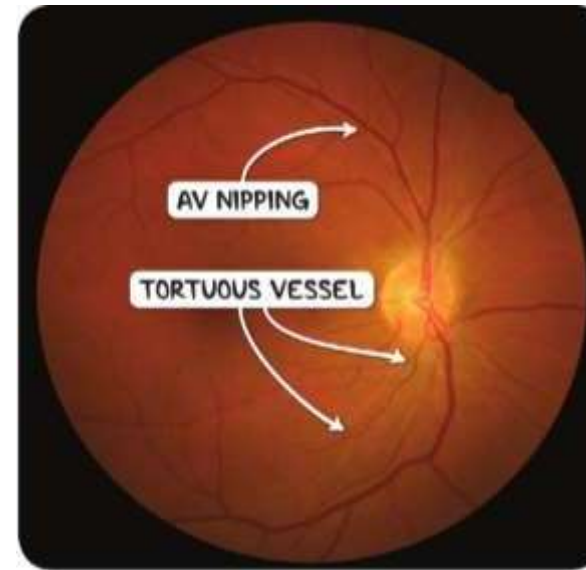
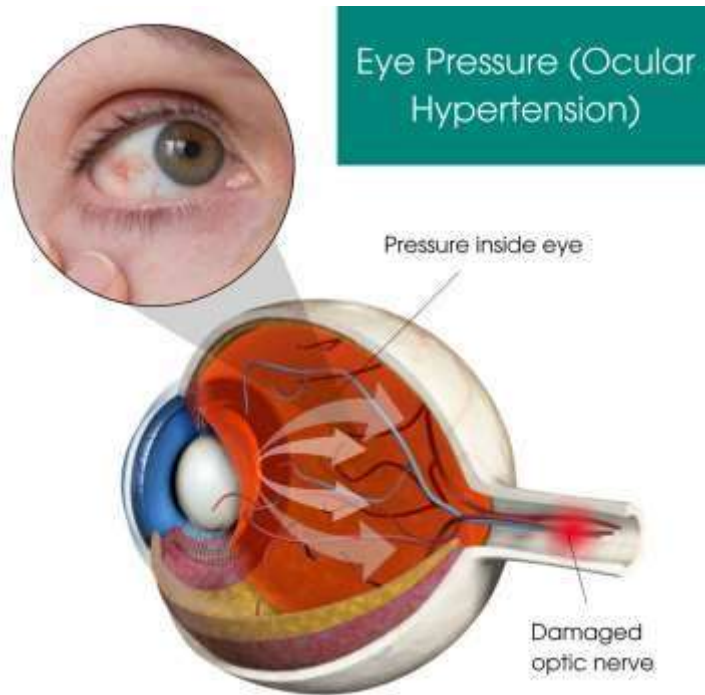


**Figure 11.3** Retinal photograph demonstrating changes of hypertensive retinopathy (AV nipping and tortuous vessels).

c. **CNS** Increased incidence of intracerebral hemorrhage. Increased incidence of other stroke subtypes as well (transient ischemic attacks [TIAs], ischemic stroke, and lacunar stroke).

Hypertensive encephalopathy when BP is severely elevated (uncommon).

d. **Kidney** Arteriosclerosis of afferent and efferent arterioles and glomerulus —called nephrosclerosis . Decreased GFR and dysfunction of tubules—with eventual renal failure.

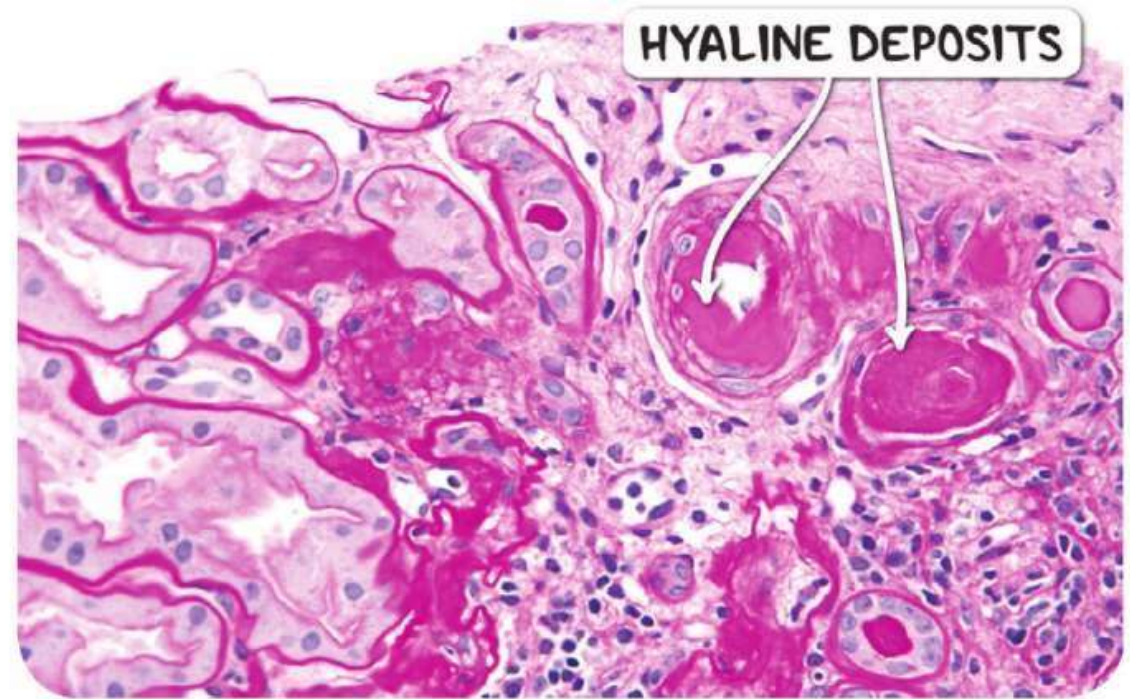


**Figure 11.4** Histological appearance of renal artery hyalinosis; a manifestation of hypertensive renal disease.

## **Quick HIT**

### **Goals in Evaluating a Patient With HTN**

- Look for secondary causes (may be treatable).
- Assess damage to target organs (heart, kidneys, eyes, CNS).
- Assess overall cardiovascular risk.
- Make therapeutic decisions based on the above.

## **Quick HIT**

### **Definition of HTN**

BP >130/80 in general population (including patients with diabetes and renal disease)

# HTN- ELDERLY

More than 50% of people > 60y. are HTN

- HTN Age related disease. LOW RENIN HTN Isolated Systolic HTN is common- Atherosclerosis. They are high risk group patients FOR STROKE - IHD- MI- HF-CKD- Peripheral vascular disease.
- DRUG of choice Hydrochlorthiazide diuretics + Calcium channel blockers
- AGE > 80 years- BP- TARGET -140/90 - Consider CO- Morbidity. SYMPATHETIC OVER DRIVE- HTN- SMOKER—OBESE-YOUNG- <50Y SYMPATHETIC STIMULATION - VASOCONSTRICTION- HIGH RENIN- TACHYCARDIA- resting HR>80 -p/min. diastolic HTN-
- TREATMENT- B- BLOCKER- CENTRALLY ACTING DRUGS-MOXODINE.

# HYPERTENSION IN PREGNANCYBP

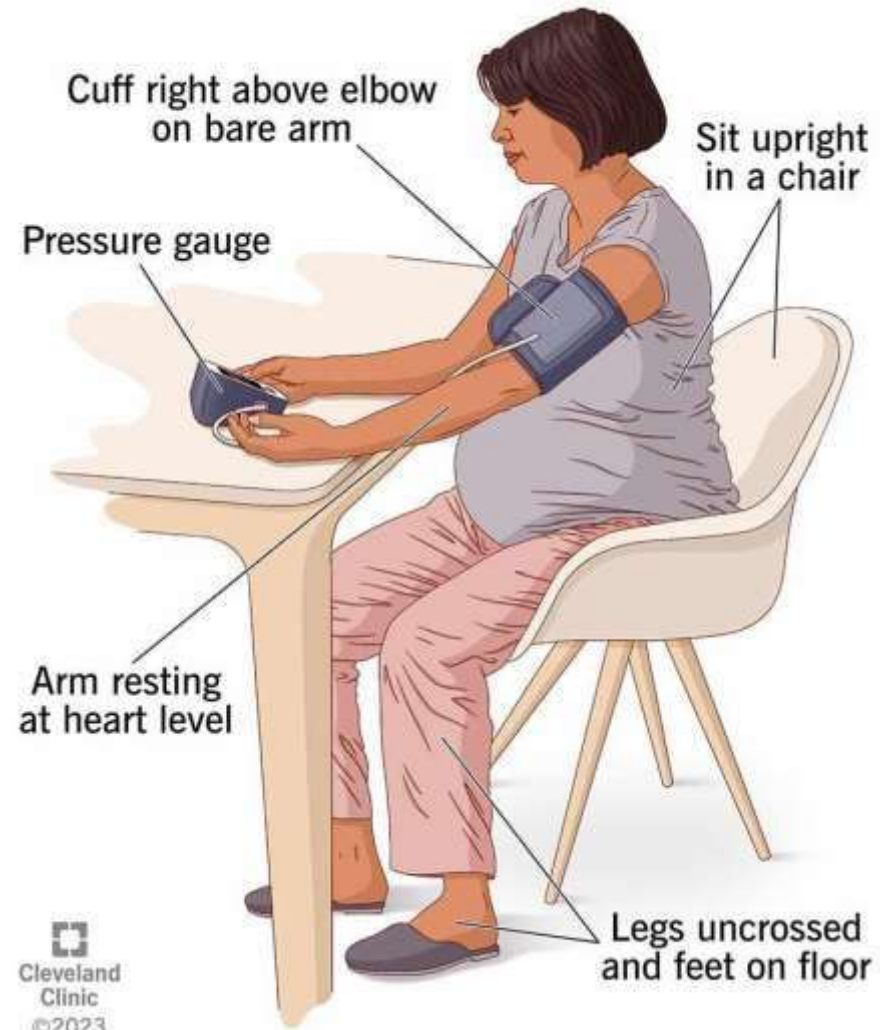
- 1 Chronic -HTN- pre- existing before 20 weeks of gestation.
- 2 Gestational HTN-is BP >140/90 in 2nd trimester in previously Normotensive women NO proteinuria.
- 3 Pre-Eclampsia -HTN-after 20 weeks of gestation+ proteinuria.
- 4 Eclampsia- HTN + grand mal seizuresleg edema- proteinuria >300mg/24hours .
- 5 HELLP syndrome – sever pre-eclampsia +Hemolytic anemia - Elevated liver enzymes- Low plat. ACEI- ARABs – TERATOGENIC- CONTRA-INDICATED.First line Methyldopa  
.Second line- Nifedipine - Labetalol- metoprolol-THIAZIDE.

# Diagnosis

# BP measurement

- a. Unless the patient has severe HTN or evidence of end-organ damage, never diagnose HTN on the basis of one BP reading. Establish the diagnosis on the basis of at least two readings over 4 or more weeks apart, ideally using home blood pressure monitoring in addition to office readings
- b. Observe the following to obtain an accurate BP reading  
The arm should be at heart level, and the patient should be seated comfortably  
Have the patient sit quietly for at least 5 minutes before measuring BP  
Make sure the patient has not ingested caffeine or smoked cigarettes in the past 30 minutes (both elevate BP temporarily)  
Use a cuff of adequate size (a cuff that is too small can falsely elevate BP readings).  
The bladder within the cuff should encircle at least 80% of the arm.

## Blood Pressure Measurement



2. Order the following laboratory tests to evaluate target organ damage and assess overall cardiovascular risk

a. **Urinalysis**

b. **Chemistry panel**: serum K1, BUN, Cr

c. **Fasting glucose** (if patient is diabetic, check for microalbuminuria )

d. **Lipid panel**

e. **ECG**

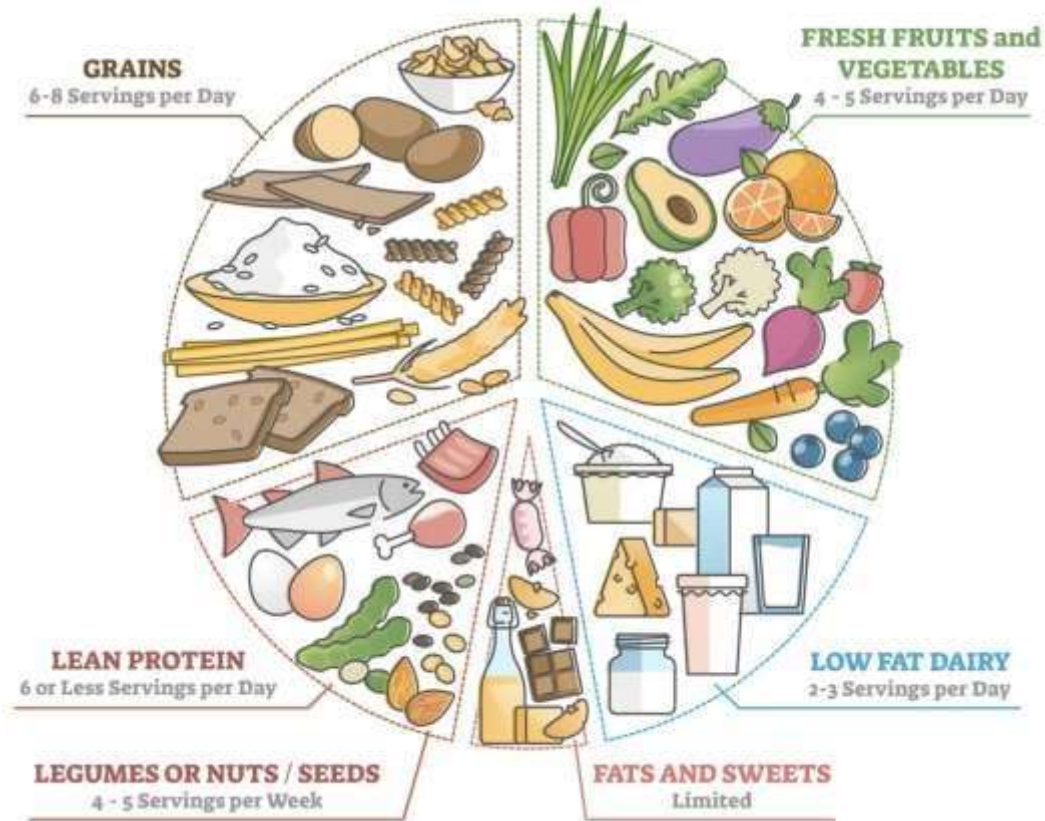
3. If the history and physical examination (H&P) or laboratory tests suggest a secondary cause of HTN, order appropriate tests.

# Treatment

1. **Lifestyle changes**, listed in order of effect on BP reduction:
  - a. **Weight loss** lowers BP significantly. In patients with central obesity (who often have coexisting diabetes, hyperlipidemia, and other risk factors), weight loss is particularly important because multiple risk factors are reduced concomitantly.
  - b. **Follow a low-saturated-fat diet** rich in fruits, vegetables, and low-fat dairy products (DASH diet).
  - c. **Exercise regularly**. Regular aerobic exercise can lower BP (and reduce overall cardiovascular risk).

# The DASH Diet

DIETARY APPROACHES TO STOP HYPERTENSION



**d. Reduce salt intake.** Reduction in dietary salt has been shown to reduce BP. Recommend either a no-added-salt diet (4 g sodium/day) or a low-sodium diet (2 g/day).

**e. Avoid excessive alcohol consumption.** Alcohol has a pressor action, and excessive use can increase BP.

**f. Others—stop unnecessary medications** that may contribute to HTN. Engage in appropriate stress management practices.

**Medication that  
elevate blood  
pressure**

Nonsteroidal anti-inflammatory agents

Corticosteroids

Anabolic steroids

Serotonin reuptake inhibitors

Serotonin-norepinephrine reuptake inhibitors

Tricyclic antidepressants

Monoamine oxidase inhibitors

Cyclosporine

Tacrolimus

Erythropoietin

Methylphenidate

Amphetamines

Decongestants

Oral contraceptives

# Pharmacological therapy

Typically a thiazide diuretic, calcium channelblocker, or ACE inhibitor/ARB is used as first line.

**a. Thiazide diuretics** Mechanism is as a diuretic by inhibiting NaCl reabsorption in the distal convoluted tubule. Chlorthalidone is often used as a first-line option, since it was studied in major trials such as ALLHAT. A good option in patients with osteoporosis (increases calcium reabsorption in the nephron).

**b.  $\beta$ -Blockers**—decrease HR and cardiac output and decrease renin release. A good option in patients with CHF, CAD, or atrial fibrillation; a poor option in patients with obstructive lung disease, heart block, or depression.

c. **ACE inhibitors** and ARBs ACE inhibitors work by inhibiting the renin–angiotensin aldosterone system (RAAS) by blocking the conversion of angiotensin I to angiotensin II. Angiotensin II normally causes vasoconstriction, aldosterone release, and ventricular remodeling. ARBs work by directly blocking angiotensin II receptors. ACE also acts to degrade bradykinin, so inhibition results in excess levels in the lung that can cause a chronic dry cough. Preferred in all diabetic patients because of their protective effect on kidneys; also a good option for patients with CHF, CAD. ACE inhibitors and ARBs should not be used in combination.

**Quick**

**HIT**



Treatment with ACE inhibitors and ARBs is associated with decreased risk of new-onset diabetes in patients with HTN.

- d. **Calcium channel blockers**—cause vasodilation of arteriolar vasculature.
- e.  **$\alpha$ -Blockers**—decrease arteriolar resistance. May be of benefit if the patient has concurrent benign prostatic hyperplasia (BPH) but these are not considered first- or second-line agents.
- f. **Vasodilators** (hydralazine and minoxidil)—not commonly used; typically given in combination with  $\beta$ -blockers and diuretics to patients with refractory HTN.
- g. **Mineralocorticoid receptor antagonist** (e.g., spironolactone, eplerenone)—great option to add on in cases of resistant hypertension.

**Quick**

**HIT**



Always obtain a pregnancy test in reproductive age women before starting an antihypertensive medication. Thiazides, ACE inhibitors, calcium channel blockers, and ARBs are contraindicated in pregnancy.  $\beta$ -Blockers and hydralazine are safe.

| Class                                                    | Mechanism                                            | Adverse effects                                             | Compelling indication                                                                                      | Compelling contra-indication                                   | Monitoring                                    | Example                             |
|----------------------------------------------------------|------------------------------------------------------|-------------------------------------------------------------|------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------|-----------------------------------------------|-------------------------------------|
| <b>Angiotensin-converting enzyme (ACE) inhibitor</b>     | Reduce angiotensin II vasoconstriction               | Cough<br>Angio-oedema<br>Hyperkalaemia                      |                                                                                                            | Bilateral RAS<br>Angio-oedema                                  | eGFR, K                                       | Ramipril 2.5–10 mg daily            |
| <b>Angiotensin II receptor blockers (ARB)</b>            | Reduce angiotensin II vasoconstriction               | Hyperkalaemia                                               |                                                                                                            | Bilateral RAS                                                  | eGFR, K                                       | Losartan 25–100 mg daily            |
| <b>Dihydropyridine calcium-channel blocker (DHP CCB)</b> | Peripheral vasodilation                              | Constipation, headache, oedema                              | Nifedipine/amlo-dipine recommended for use in pregnancy                                                    |                                                                |                                               | Amlodipine 5–10 mg daily            |
| <b>Non-DHP CCB</b>                                       | Peripheral vasodilation                              | Constipation, bradycardia                                   |                                                                                                            | High grade atrioven-tricular block<br>Bradycardia              | Heart rate                                    | Verapamil 120–480 mg daily          |
| <b>Thiazide-like diuretic</b>                            | Vasodilation, salt/water loss                        | Hyponatraemia, hypokalaemia, hyperuricaemia, hyperglycaemia | Heart failure                                                                                              | Gout                                                           | Serum electrolytes, HbA <sub>1c</sub> , urate | Indapamide 1.5–2.5 mg daily         |
| <b>Potassium-sparing diuretic</b>                        | Salt/water loss                                      | Hyponatraemia, hyperkalaemia, gynaecomastia                 | Primary hyperaldosteronism<br>Resistant hypertension                                                       | eGFR <30 mL/min<br>K >5.0 mmol/L                               | Serum electrolytes                            | Spironolactone 25–50 mg daily       |
| <b>Beta-blocker</b>                                      | Vasodilation through renin inhibition, sympatholysis | Bradycardia, sleep disturbance, bronchospasm                | Heart failure with reduced ejection fraction<br>Ischaemic heart disease<br>Labetalol licensed in pregnancy | Asthma<br>High-grade atrioven-tricular block<br>Bradycardia    | Heart rate                                    | Bisoprolol 2.5–10 mg daily          |
| <b>Alpha-blocker</b>                                     | Venodilation                                         | Postural hypotension, urine incontinence, oedema            | Suspected pheochromocytoma<br>Benign prostatic hypertrophy                                                 | Urinary incontinence<br>Postural hypotension<br>Heart failure  |                                               | Doxazosin 1–8 mg twice daily        |
| <b>Central</b>                                           | Sympatholysis                                        | Depression, drowsiness, dry mouth                           | Methyldopa licensed in pregnancy                                                                           | Mood disorders<br>Heart failure with reduced ejection fraction | Rebound hypertension on cessation             | Methyldopa 250–500 mg 3 times daily |

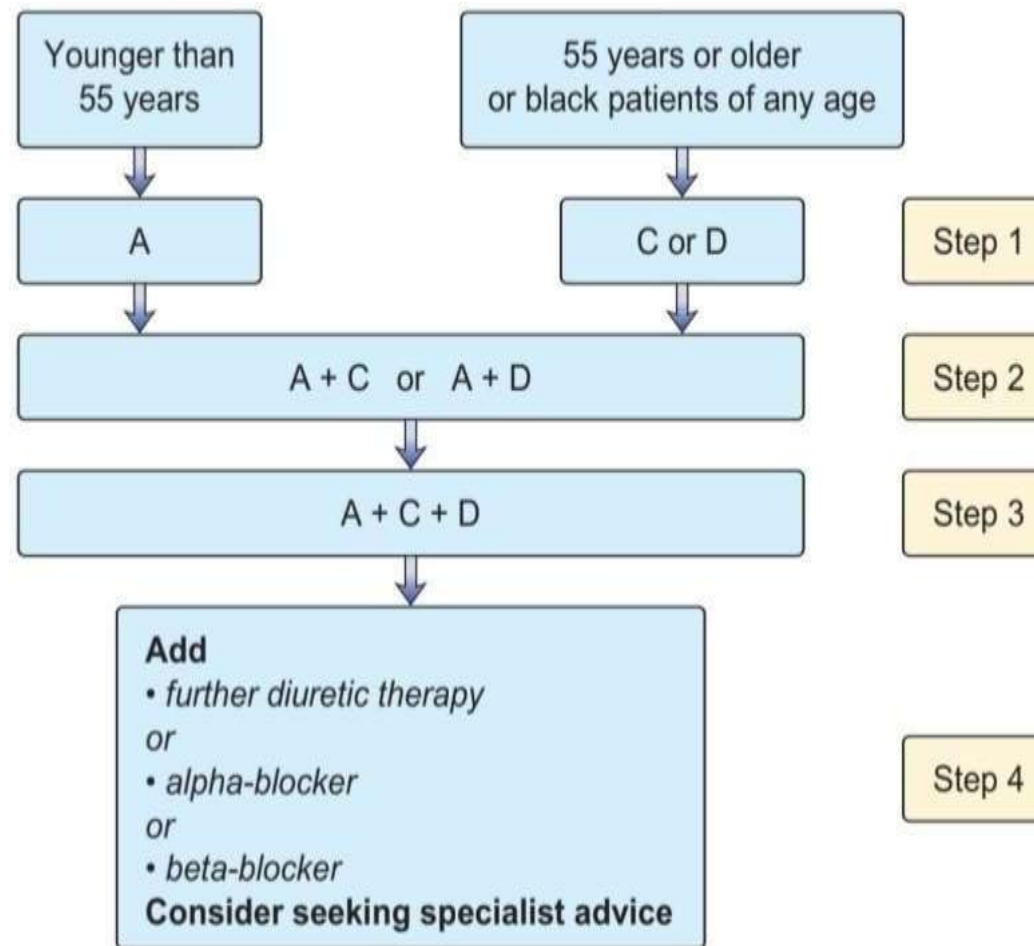
Blood pressure targets should be based on the patient's **cardiovascular risk, age, comorbidities** such as postural hypotension, etc.

- The ACC/AHA guidelines recommend a BP goal of  $<130/80$  mmHg for most patients, including older patients.
- Each of the antihypertensive agents is roughly equally effective in lowering BP. However, there is great variability in how patients respond to each drug.



The three classes of drugs that are used for initial monotherapy are **thiazide diuretics, long-acting calcium channel blockers** (most often a dihydropyridine), and **ACE inhibitors/ARBs**.  $\beta$ -Blockers are not commonly used as initial monotherapy in the absence of a specific indication because of adverse effects on some cardiovascular outcomes especially in elderly patients.





**Abbreviations:**

A = ACE inhibitor  
(angiotensin-II receptor antagonist if ACE intolerant)  
C = calcium-channel blocker  
D = thiazide-type diuretic

Black patients are those of African or Caribbean descent and not mixed-race, Asian or Chinese patients

## Refractory HTN

**Pts not controlled on a combination of three antihypertensive medications that are taken on reasonable doses and that include a diuretic are considered to have resistant HTN (once nonadherence and white coat effects eliminated)**

**BP that requires at least 4 medications to achieve control is considered CONTROLLED resistant HTN. Many pts who appear to have resistant HTN actually have pseudoresistance rather than true resistant. Pseudo resistance: -inaccurate BP measurement (use of small cuff, not allowing pt to rest before readings) -poor adherence to blood pressure medications -poor adherence to lifestyle and dietary approaches to lower BP -suboptimal antihypertensive Rx, due to either inadequate doses, inappropriate drug combinations or exclusion of a diuretic from antihypertensive regimen -white coat HTN**

One or more of the followings may contribute to True resistant HTN

- 1 extracellular volume expansion**
- 2 increased sympathetic activation**
- 3 ingestion of substances that inc BP like NSAIDS, decongestants or stimulants**
- 4 secondary HTN**

-ACCOMPLISH trial showed that treatment with antihypertensive combination therapy—the ACE inhibitor benazepril plus the calcium channel blocker amlodipine—was more effective than treatment with the ACE inhibitor plus diuretic.

Based on this trial, it is common practice to start monotherapy with either a calcium channel blocker or an ACE inhibitor, so that the other can be added if combination therapy is needed.

- If the patient's response to one agent is not adequate, there are two options: Increase the dose of the first agent to the maximum dose. OR Add a second medication (thiazide, calcium channel blocker, ACE inhibitor, or ARB); if target BP not achieved, increase the dose of each as necessary until the maximum dose is achieved.

- If a patient's response is still inadequate with two agents, consider a third agent and referral to an HTN specialist.

-The decision of when to start pharmacologic treatment is based on the patient's total cardiovascular risk, not just the elevation in BP. For any level of BP elevation, the presence of cardiovascular risk factors and/or comorbid conditions dramatically accelerates the risk from HTN, and therefore modifies the treatment plan. Estimation of overall risk depends on cardiovascular risk factors and clinical risk factors

# Hypertensive emergency

## **PATHOLOGY & CAUSES**

- Elevated blood pressure > 180/120mmHg with signs of acute end organ damage (e.g. encephalopathy, stroke, papilledema, myocardial infarction, heart failure, microangiopathic hemolytic anemia, etc.)
- Complication of poorly managed hypertension

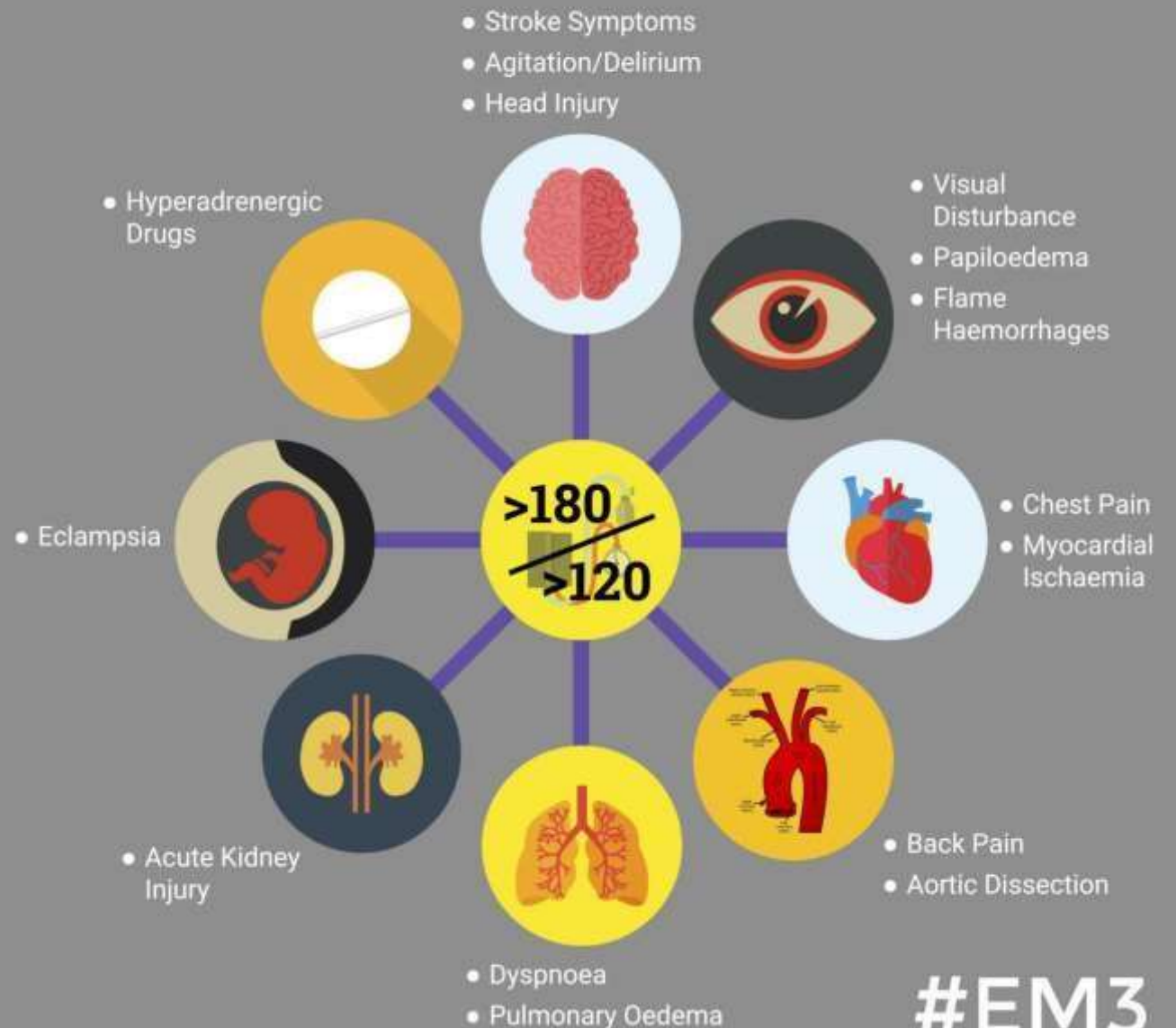
## **RISK FACTORS**

- Kidney failure, renovascular hypertension, stimulant abuse, medication non-adherence
- More common in young adults, particularly those of African descent

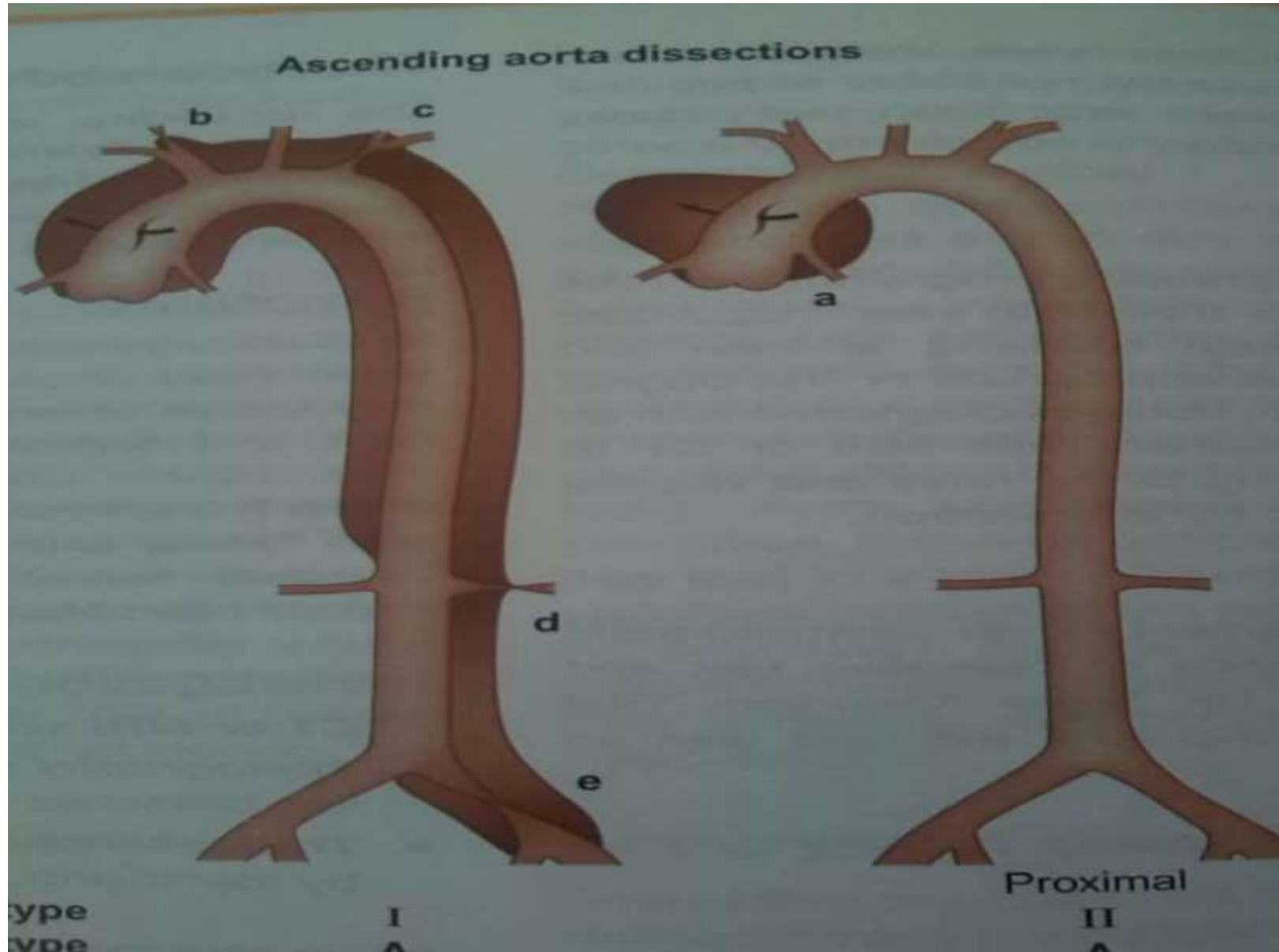
## **COMPLICATIONS**

- Neurological complications (stroke, seizures), myocardial infarction, kidney failure, permanent blindness, pulmonary edema

# Hypertensive Emergencies:

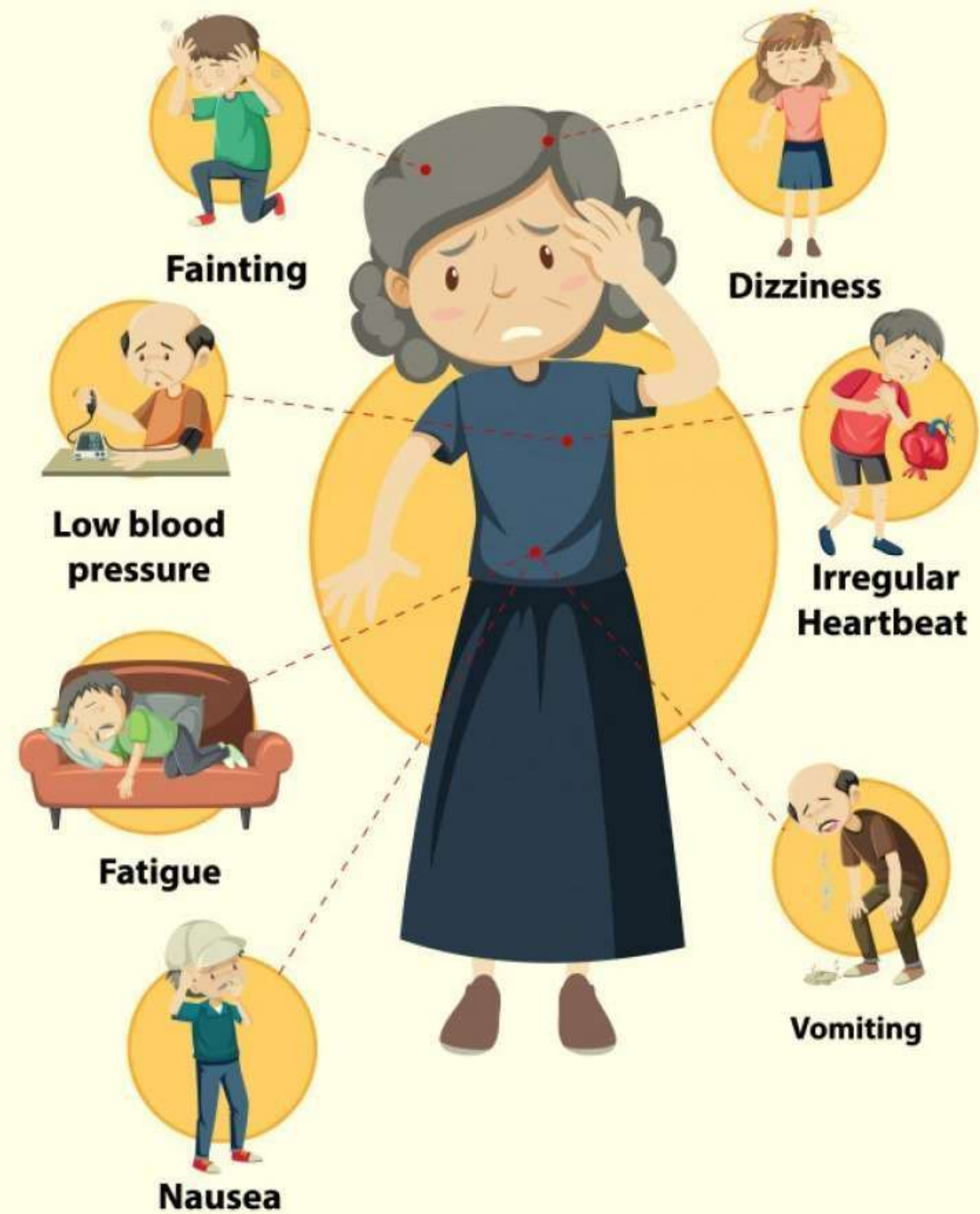


# MALIGNANT HTN



## SIGNS & SYMPTOMS

- Blood pressure > 180/120mmHg, signs of end-organ damage
  - Blurred vision, altered mental state, chest pain, headache, nausea, vomiting, numbness in extremities, oliguria, seizure, dyspnea, weakness, papilledema



# DIAGNOSIS

## OTHER DIAGNOSTICS

- Sphygmomanometer
  - Blood pressure > 180/120mmHg
- Evaluation to identify at-risk target organ
  - Electrocardiography (heart)
  - Chest X-ray (heart, lungs)
  - Urinalysis (kidneys)
  - Serum electrolytes, serum creatinine (kidneys)
  - Cardiac enzymes (heart)
  - CT scan of brain (if brain suspected → neurologic symptoms, retinopathy)
  - Contrast-enhanced CT scan of chest (if aortic dissection suspected)



# TREATMENT

- Treatment varies case-by-case, dependent on affected organ

## MEDICATIONS

- Unwise to lower blood pressure too quickly/ too much, as this can lower cerebral perfusion excessively
  - Most cases: mean arterial pressure (MAP) should be reduced using intravenous medication 10–20 % in first hour, then 5–15% over the following 23 hrs. Specific medications used dependent on case

## OTHER INTERVENTIONS

- Exceptions to most cases: acute phase ischemic stroke (not lowered unless specific conditions met)
  - Acute aortic dissection (rapid lowering), intracerebral hypertension (variable)

