Last edited: 10/30/2024

HYPERTENSION

I. PATHOPHYSIOLOGY
A. Primary Hypertension
B. Secondary Hypertension
C. Staging of Hypertension

II. COMPLICATIONS
A. Cardiovascular Disease
B. Neurologic Disease
C. Renal Disease
D. Retinal Disease

III. DIAGNOSTIC APPROACH
BP Measurement

IV. TREATMENT
A. Treatment Guideline
B. Antihypertensive Based on Comorbidities
C. Treatment of Hypertensive Crisis

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I. PATHOPHYSIOLOGY

A. Primary Hypertension

- Also known as **Essential Hypertension**
- Accounts for approximately **90% of cases**
- Presents in those from ages **25** to **55**

Mechanisms

a) Vessel Wall Thickening

- **Pathophysiology:**
  - Older patients → Calcium deposits in vessels → **Thick-walled vessel** → ↓Luminal diameter → ↑Systemic vascular resistance (SVR) → ↑BP

Risk Factors:

- Older age
- Smoking
- Diabetes

b) ↑Vasoconstriction

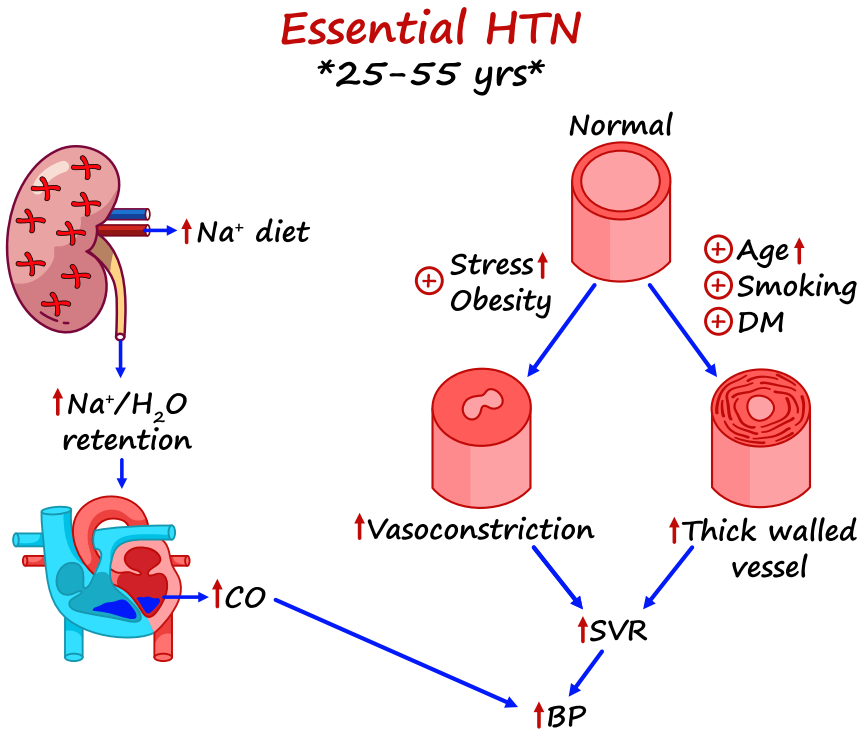
- **Pathophysiology:**
  - ↑Vasoconstriction → ↑SVR → ↑BP

Risk Factors:

- ↑Stress:
  - Type A personality
  - Exhibits a stressful lifestyle
- Obesity:
  - Modifiable risk factor
  - Causes ↑Cytokine release that can stimulate vasoconstriction

c) ↑Sodium Diet

- **Pathophysiology:**
  - ↑Na^+ diet causing the kidneys to have difficulty secreting Na^+ → ↑Serum Na^+ → H_2O retention → ↑Preload → ↑SV → ↑CO → ↑BP



Secondary Hypertension

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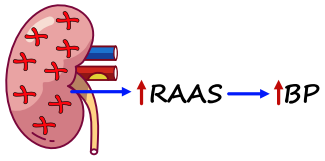
- Patients with secondary hypertension commonly have **Refractory Hypertension**
  - Administration of 3 or more antihypertensives → Unable to control hypertension

Causes

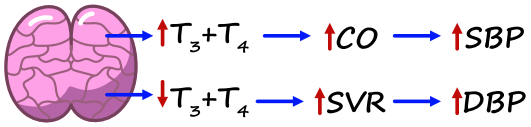
Mnemonic: RENALSS

Renal Disease

- **Pathophysiology:**
  - ↓GFR → ↑RAAS → ↑Vasoconstriction → ↑SVR → ↑BP
- **Causes:**
  - Renal vascular diseases such as **CKD** or **Renal Artery Stenosis**



Endocrine Disease



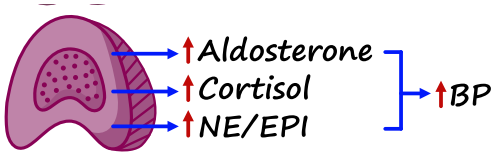
Thyroid

Hyperthyroidism

- **↑T3, T4** → ↑CO and ↑Contractility → **↑SBP**

Hypothyroidism

- **↓T3, T4** → ↑Vasoconstriction → **↑DBP**



Adrenal Glands

Hyperaldosteronism

- ↑Aldosterone → ↑Na^+ and water reabsorption → ↑BP

Cushing's Syndrome/Cushing's Disease

- ↑Cortisol → ↑Sympathetic activity → ↑BP

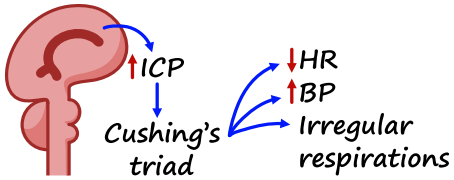
Pheochromocytoma

- ↑NE and Epi → ↑Sympathetic activity

Neurologic Disorders

- **Pathophysiology:**
  - A neurologic disorder that manifests with **↑Intracranial pressure (ICP)** → **Cushing's Triad**
  - Causes: Cerebral edema, CNS bleed, Hydrocephalus, CNS mass

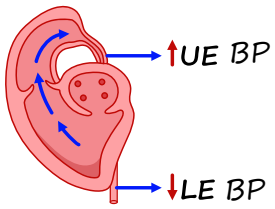
Cushing's Triad:
1) Bradycardia, 2) Hypertension, 3) Irregular Respirations



Aortic Diseases

Coarctation of the Aorta

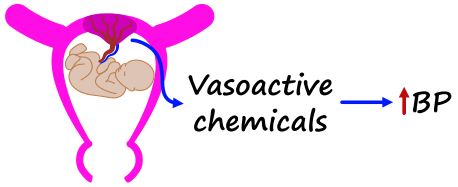
- **Pathophysiology:**
  - Narrowing of the aortic lumen after the left subclavian artery
    → ↑Pressure prior to the narrowed portion of the aorta
    → **↑Upper extremity BP and ↓Lower extremity BP**



Little People

Pre-Eclampsia and Eclampsia

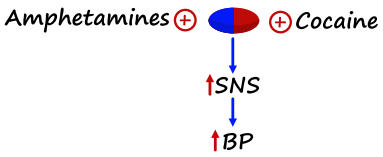
- **Pathophysiology:**
  - Insufficient placental blood supply → Placenta releases **vasoactive chemicals** → Vasoconstriction → ↑Maternal BP in pregnant patients in the 3rd trimester with **proteinuria**, **edema**, and **↑BP (Seizures also occur if presenting with eclampsia)**



Substances

Sympathomimetics

- **Pathophysiology:**
  - Sympathomimetics → Vasoconstriction → ↑BP along with Altered mental status, diaphoretic, hypertensive, tachycardic, with a history of drug abuse
  - Examples: Cocaine, Methamphetamine, PCP

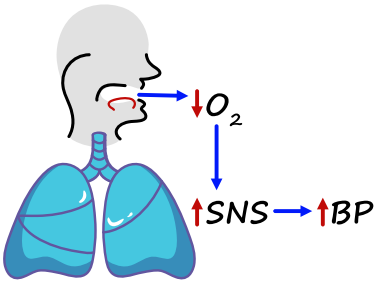


Sleep Apnea

- **Pathophysiology:**
  - Nocturnal hypoxia during periods of apnea during sleep → ↑Sympathetic nervous system → ↑Vasoconstriction leading to → **Refractory Hypertension**

Risk Factors and Clinical Features:

- Obese patients
- Witnessed periods of snoring and apnea



B. Staging of Hypertension

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Stage	SBP	DBP
Normal BP	<120	<80
Prehypertensive	<130	
Stage I	<140	<90
Stage II	>140	>90
HTN urgency (Ø target organ damage)	>180	>120
HTN emergency (target organ damage)		

Stage I: ↑BP plus comorbidities may consider initiation of medications
Stage II: Two or more readings of ↑BP warrant antihypertensive medication

Remember:
The presence of **target organ damage** → **Hypertensive Emergency!**
Especially in the setting of elevated BP (>180/>120)

19:43

COMPLICATIONS

- **Target organ damage** is classified as:
  - Cardiovascular disease
  - Neurologic disease
  - Renal disease
  - Retinal disease

C. Cardiovascular Disease

1. Heart Failure (HFpEF)

- **Pathophysiology:**
  - Severe and acute ↑BP → **↑Afterload** → Left ventricle has to work harder → **LV hypertrophy** → ↓Ventricular filling → **Diastolic Heart Failure** or **HFpEF**
- **Presents with the following:**
  - **Flash pulmonary edema**
    - Acute development of Hypoxia, ↑RR/WOB

2. Atherosclerosis

- **Pathophysiology:**
  - ↑BP → Triggers **atherosclerosis of blood vessels**

a) CAD/MI

- **Stable CAD Pathophysiology:**
  - Coronary plaques → ↓Coronary supply → Angina in the setting of ↑O2 demand during exertion
- **ACS or MI Pathophysiology:**
  - **Coronary plaque rupture** → Coronary occlusion → Reduces O_2 perfusion significantly → MI
- **Presentation of MI:**
  - Angina
  - ECG with ST changes
  - Troponin elevation

b) PAD or CLI

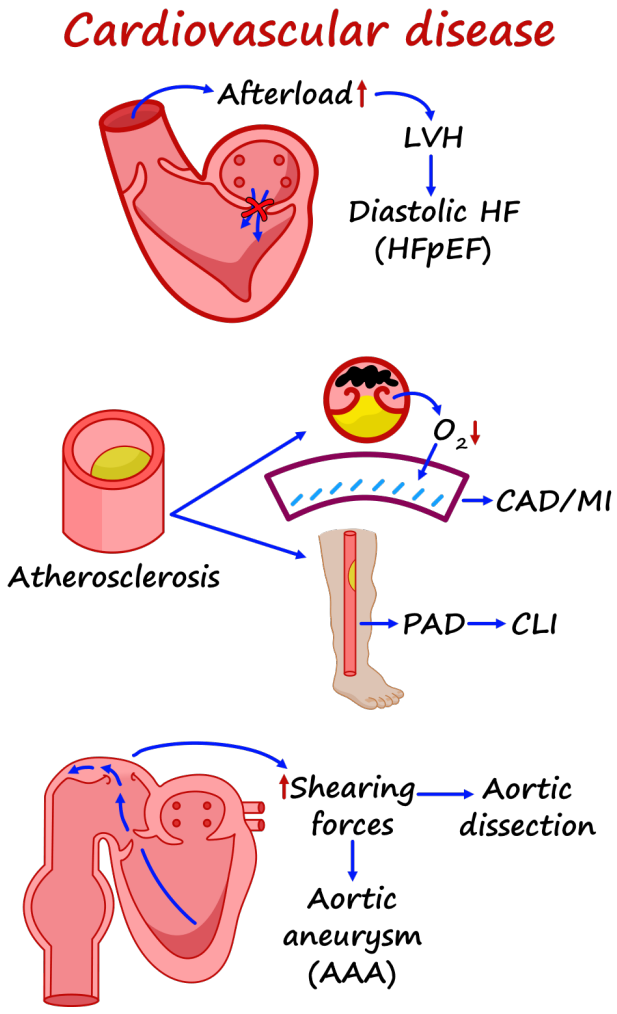
- **Pathophysiology:**
  - LE arterial plaque → ↓Blood supply to **LE muscles and skin**
- **Presentation of PAD:**
  - LE pain, ↓Pulses, LE pallor, arterial ulcers, claudication

3. Aortic Dissection

- **Pathophysiology:**
  - ↑BP causes shearing forces in the aortic walls → Intimal tear → Blood moves within the false lumen → **Aortic Dissection**
- **Presentation of Aortic Dissection:**
  - Ripping or tearing chest pain
  - Asymmetric BP and pulses

4. Aortic Aneurysm

- **Pathophysiology:**
  - Vasa vasorum damage → ↓Supply to the aortic wall → Weakening of aortic wall → Dilation of aorta → **Aortic Aneurysm (AAA most common)**
- **Presentation of AAA:**
  - Usually asymptomatic → Requiring surveillance via U/S



D. Neurologic Disease

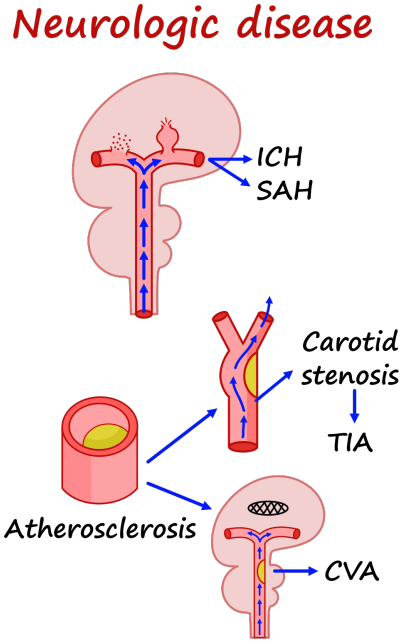
25:36

1. Transient Ischemic Attack or Cerebrovascular Accident

- **Pathophysiology:**
  - o **Carotid, Vertebral, and Intracranial** blood vessels become **atherosclerotic** → ↓O2 supply to the brain → Cerebral ischemia and potential infarction if plaque ruptures
- **Presentation of TIA or CVA:**
  - o **Neurologic deficits** dependent on vascular territory affected

2. Intracerebral or Subarachnoid Hemorrhage

- **Pathophysiology:**
  - o Acute ↑BP → Rupture of cerebral vessels → Blood accumulates in parenchyma (ICH) or subarachnoid space (SAH)
- **Presentation of ICH or SAH:**
  - o Headache
  - o **Neurologic deficits** dependent on vascular territory affected



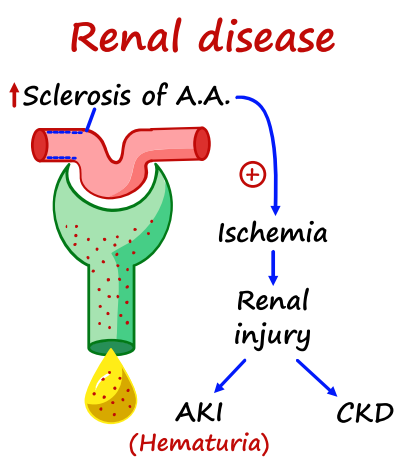
E. Renal Disease

27:42

a) AKI or CKD

- **Pathophysiology:**
  - o ↑BP → ↑GFR → **Thickening** or **sclerosis of the afferent arteriole** to compensate for ↑GFR → ↓ Blood flow to the glomerulus → Ischemia → Renal injury (**AKI** or **CKD**)

Acute Kidney Injury (AKI)	Chronic Kidney Disease (CKD)
● Abrupt ↑ in creatinine	● ↓GFR over time
● Hematuria	● Albuminuria



F. Retinal Disease

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- **Pathophysiology:**
  - o **Sclerosis of the vessels** leading to ↓O_2 supply of the retinal tissues → **Small hemorrhaging** → **↑Edema**
- **Presentation of Retinopathy:**
  - o Blurry vision
  - o Progressive vision loss

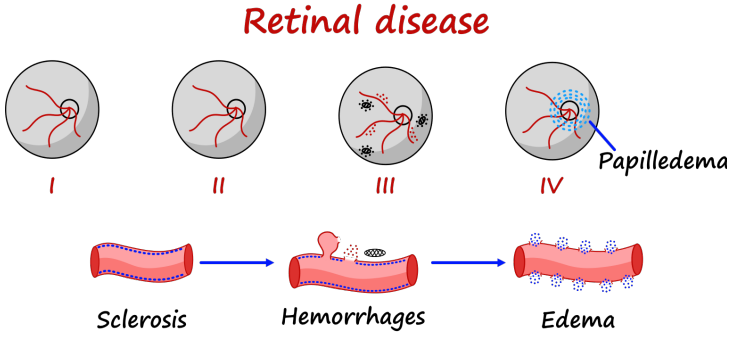

Table 1. Staging of Hypertensive Retinopathy.

Grade I	AV nicking
Grade II	Constricted vessels
Grade III	Cotton wool spots
Grade IV	Blurring of the optic disc due to **papilledema**
*In the chronic stages, you can see AV nicking, constriction of vessels, and cotton wool spots. Acutely, there should be close monitoring for papilledema	

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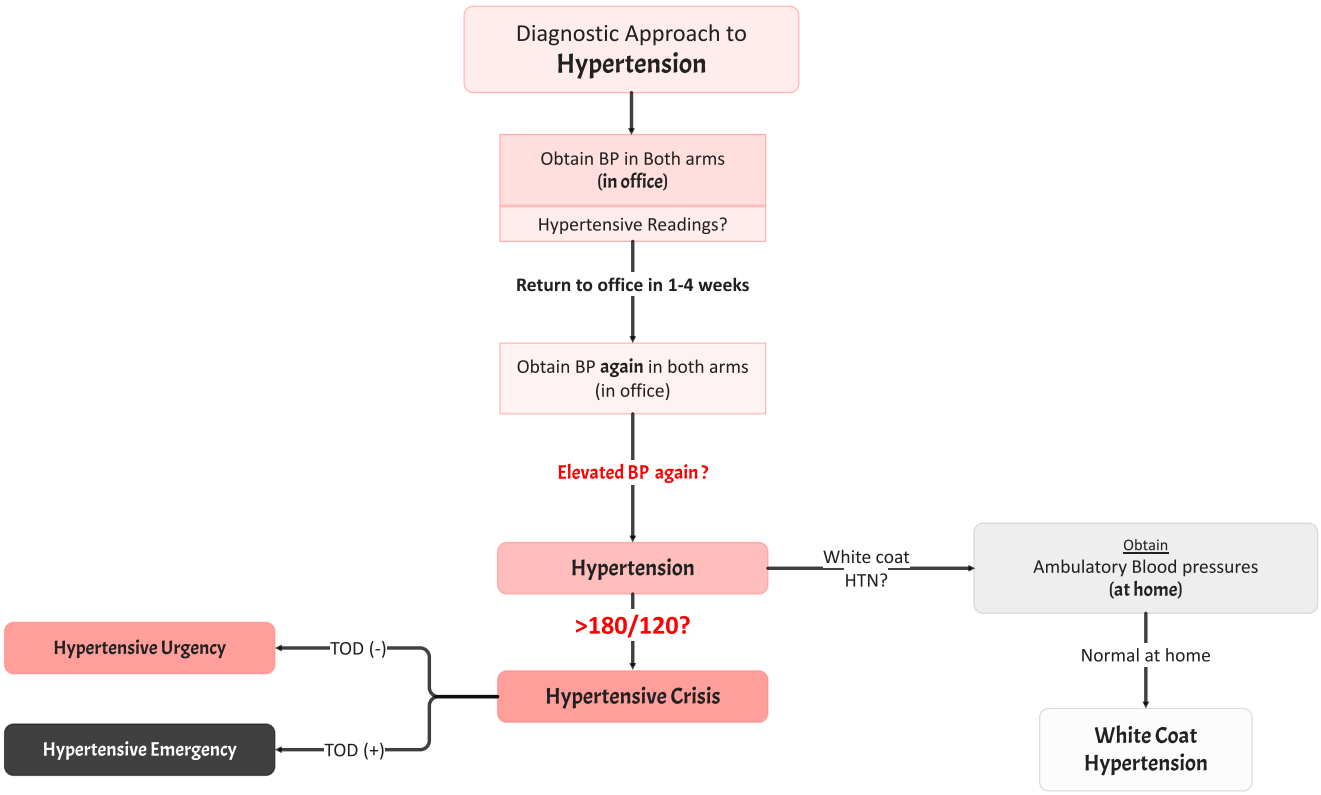
II. DIAGNOSTIC APPROACH



BP Measurement

- Obtain BP in **both arms**
- Are there hypertensive readings?
  - Yes? → Return to office in 1-4 weeks

Upon Follow-Up:

- Recheck BP in **both arms**
- ↑BP reading? → **Hypertension**

Hypertension requires ≥ 2 readings of elevated blood pressure in two spaced-out office visits.

White-Coat Hypertension

- **White-Coat Hypertension:**
  - Blood pressure readings at a health care provider's office are higher than they are in other settings, such as at home
- Check **ambulatory BP** at home
- Normal at home? → **White-Coat Hypertension**

Hypertensive Urgency and Hypertensive Emergency

- If the **BP** is **> 180/120**, suspect a **Hypertensive Crisis**
  - (-) TOD → **Hypertensive Urgency**
  - (+) TOD → **Hypertensive Emergency**

33:48

III. TREATMENT

A. Treatment Guideline

Table 2. Treatment of hypertension according to staging.

Stage	SBP (mmHg)	DBP (mmHg)	Treatment
Normal BP	< 120	< 80	**Lifestyle Modifications** (↓Weight, DASH diet, Exercise, ↓Na diet, ↓Alcohol)
Prehypertensive	< 130		
Stage 1	< 140	< 90	**Lifestyle Modifications** (↓Weight, DASH diet, Exercise, ↓Na diet, ↓Alcohol) Start Antihypertensives if **ASCVD risk ≥ 10%** (ACE-I/ARB, Thiazides, CCB)
Stage 2	> 140	> 90	**Lifestyle Modifications** (↓Weight, DASH diet, Exercise, ↓Na diet, ↓Alcohol) **Start Antihypertensives** (ACE-I/ARB, Thiazides, CCB)

Patients suspected of hypertension should undergo **lifestyle modification**

Normal or Prehypertensive

a) Lifestyle Modifications

- Lose weight
- DASH diet
- Exercise
- Reduce Na^+ diet
- Reduce alcohol

Stage I

- **Lifestyle modification +**

b) Antihypertensives

- ***If ASCVD risk ≥ 10%***
  - Indicative of ↑Risk of stroke, MI, CLI
    - Requires the initiation of antihypertensives
- Examples:
  - ACE-I or ARBs (most common)
  - Thiazides
  - Calcium-Channel Blockers (CCBs)
    - Amlodipine or Nifedipine

Stage 2

- **Lifestyle modification +**

c) Antihypertensives

- ***Start antihypertensive regimen***
  - Regardless of ASCVD risk
- Choice of one of the following:
  - ACE-I, ARBs, Thiazides, or CCB

If the patient is African-American, consider **CCBs and Thiazides** instead of ACE-I/ARBs because they have low-renin hypertension.

B. Antihypertensive Based on Comorbidities

35:52

1. Comorbidities:

Post-MI

Beta-Blockers Purpose:
- ↓Risk for V-Tach or V-Fib
- ↓Ventricular remodeling

ACE-I or ARB Purpose:
- ↓Ventricular remodeling

Heart Failure

Beta-Blockers Purpose:
- ↓Ventricular remodeling

ACE-I or ARB Purpose:
- ↓Ventricular remodeling

Aldosterone Antagonist Purpose:
- ↓Ventricular remodeling

Coronary Artery Disease

- Indicated for patients WITH Angina:

Beta-Blockers Purpose:
- ↓Contractility, ↓HR

CCB's Purpose:
- ↓Contractility, ↓HR and dilates coronary arteries

Nitrates Purpose:
- ↓Preload

Atrial Fibrillation

Beta-Blockers Purpose:
- ↓HR by blocking AV node

CCB's Purpose:
- ↓HR by blocking AV node

Diabetes Mellitus, Chronic Kidney Disease

ACE-I or ARB purpose
- ↓Proteinuria, ↓damage to the kidneys → Help prevent progression to CKD

Pregnancy
- **No teratogenic effects** → Safe for pregnancy

Mnemonic:

Healthy Moms Love Nifedipine
Hydralazine
Methyldopa
Labetalol
Nifedipine

Osteoporosis

Thiazides Purpose:
- Reabsorbs calcium across kidneys, maintaining a good serum calcium

Benign Prostatic Hyperplasia

Alpha-1 Antagonist Purpose:
- Causes vasodilation → ↓Preload and afterload

Table 3. Summary of Antihypertensives based on Comorbidities.

Disease	Optimal Antihypertensive
Post-MI	▪ **BB** (Metoprolol, Carvedilol) ▪ **ACE-I** or **ARB**
Heart Failure	▪ **BB** (Metoprolol, Carvedilol) ▪ **ACE-I** (Lisinopril) or **ARB** (Losartan) ▪ **Aldosterone Antagonist** (Spironolactone)
Coronary Artery Disease	▪ **BB** (Metoprolol, Carvedilol) ▪ **CCB** (Diltiazem, Verapamil or Amlodipine) ▪ **Nitrates** (ISDN or ISMN)
Atrial Fibrillation	▪ **BB** (Metoprolol, Carvedilol) ▪ **Non-DHP CCB** (Diltiazem or Verapamil)
Diabetes Mellitus Chronic Kidney Disease	▪ **ACE-I** (Lisinopril) or **ARB** (Losartan)
Pregnancy	Hydralazine Methyl dopa Labetalol Nifedipine
Osteoporosis	▪ **Thiazides** (HCTZ)
BPH	▪ **Alpha-1 Antagonist** (Tamsulosin, Doxazosin)

2. Other Things to Consider:

- Avoid **β-blockers** in COPD, asthma and acute heart failure (AHF)
  - Can worsen cardiogenic shock
- Avoid **Non-DHP CCBs** in AHF
- Avoid **ACE-I, ARBs, AA** in acute kidney injury and hyperkalemia
  - Can ↑K^+ and creatinine
- Avoid **Thiazides** in gout
  - Possibility of ↑Uric acid

C. Treatment of Hypertensive Crisis

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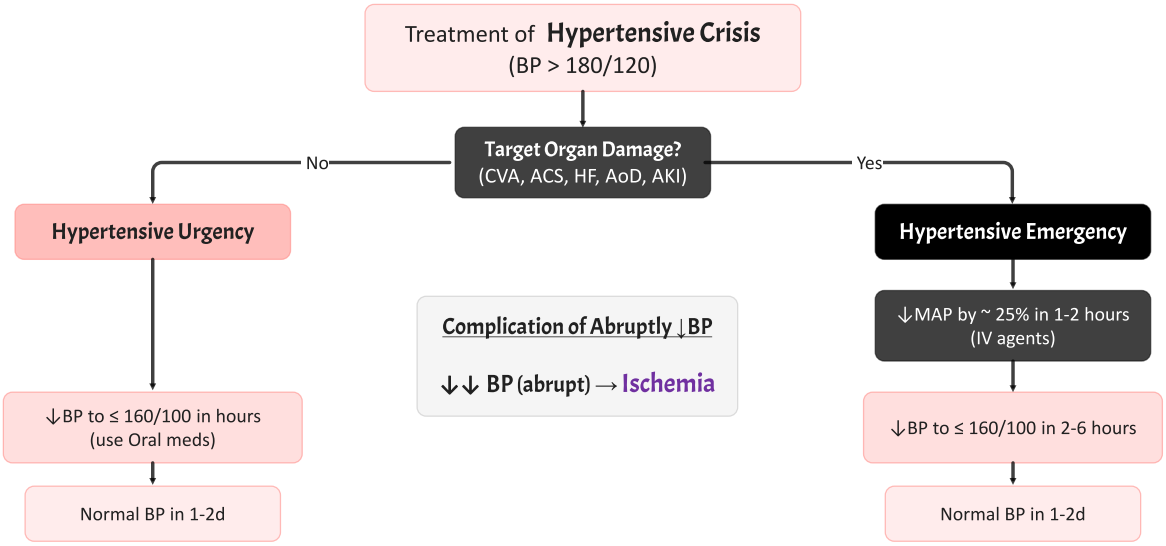

- **Do they have target organ damage?**
  - Neurologic deficits?
  - Confusion and encephalopathy?
  - Chest pain and ECG changes?
  - Pulmonary edema or signs of acute HF?
  - Aortic dissection or aneurysm?
  - AKI?

1. Hypertensive Urgency

- (-) TOD
- Aim to **↓BP to ≤ 160/100** in 1-2 hours through oral meds
- Goal → Normalize BP in 1-2 days
  - Abrupt drop in BP can cause ischemia → Monitor closely

2. Hypertensive Emergency

- (+) TOD
- Aim to **↓ MAP by appx 25% in 1-2 hours** through IV agents
- Goal → Normalize BP in 1-2 days

3. Complications of Abrupt ↓ in BP

- Developing Ischemia → Worsening organ malperfusion and TOD

4. IV Antihypertensives in HTN Emergency

Infusion or IV Push	IV agent
Infusion (titratable)	**Nicardipine** (common)
	Esmolol (Good in cardiac disease)
	Nitroprusside (Avoid) ↑ Risk of CN toxicity with prolonged infusions (Lactic Acidosis can develop as a result)
IV push	**Hydralazine**
	Labetalol

a) Infusion

- Titratable and controlled

Nicardipine
  - Most commonly utilized infusion (inpatient setting)

Esmolol
  - Good in cardiac diseases such as CAD, MI, or Aortic Disease

Nitroprusside
  - Caution due to the risk of lactic acidosis

b) IV Push

- If BP does not require close titration:
  - **Hydralazine**
  - **Labetalol**