Hypertension

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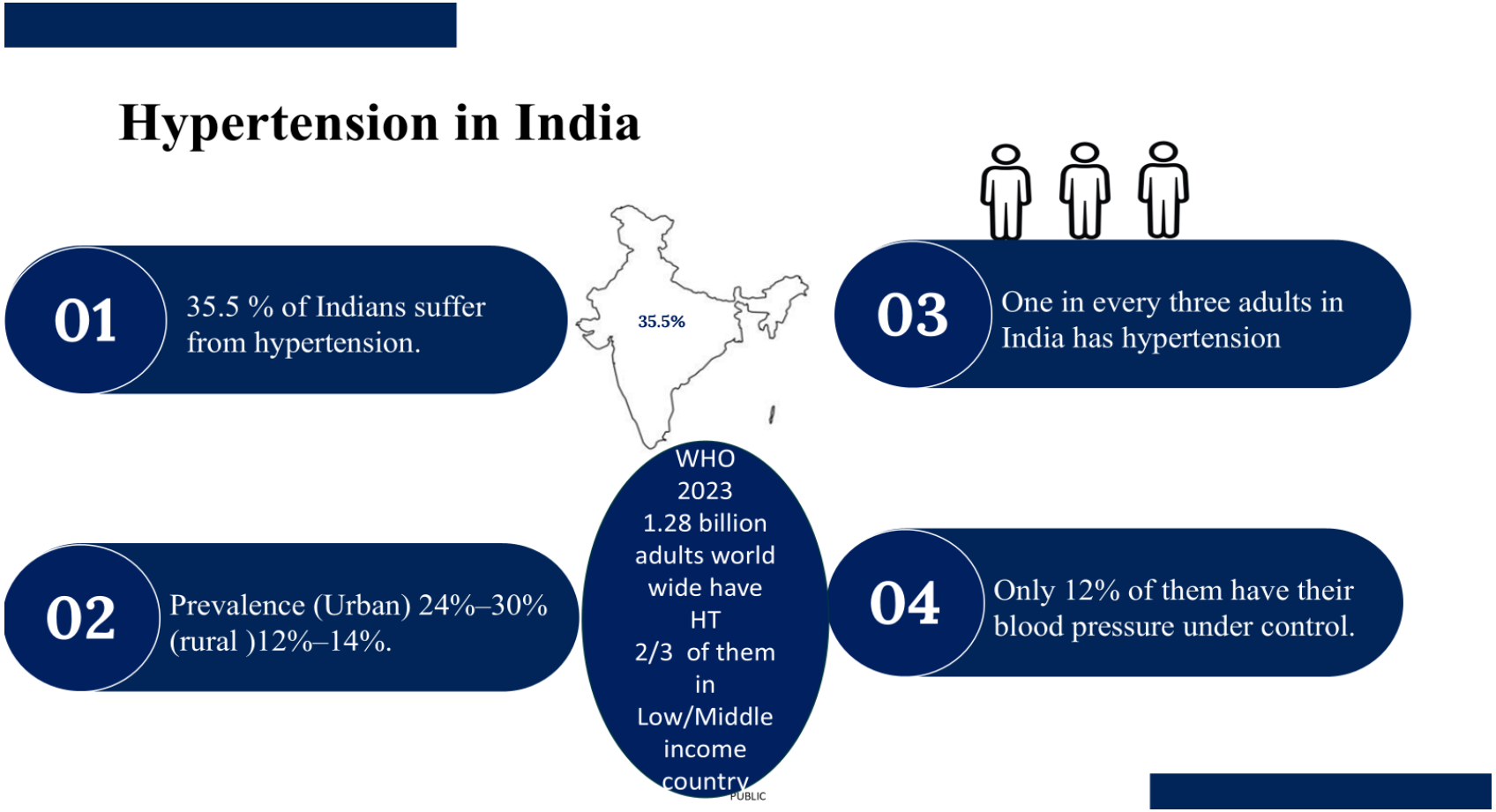
Learning Objectives

- Definition of HT
- Measurement of BP
- White coat ,Masked HT
- Pathogenesis of HT
- Secondary Hypertension
- Management

Define ---HT?

- Hypertension is defined as that level of Blood pressure at which institution of Antihypertensives will reduce Blood pressure related morbidity and mortality.

Hypertension in India

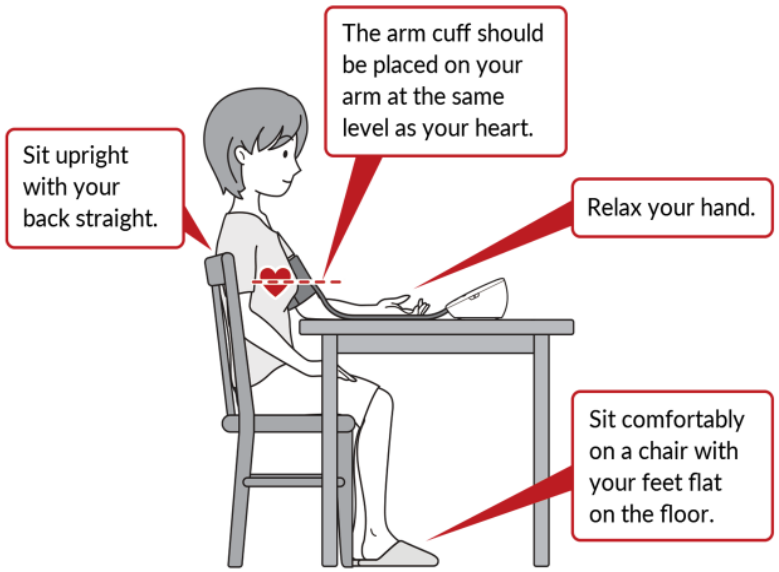


CUFFLESS BLOOD PRESSURE MONITORING

Accurate BP Measurement

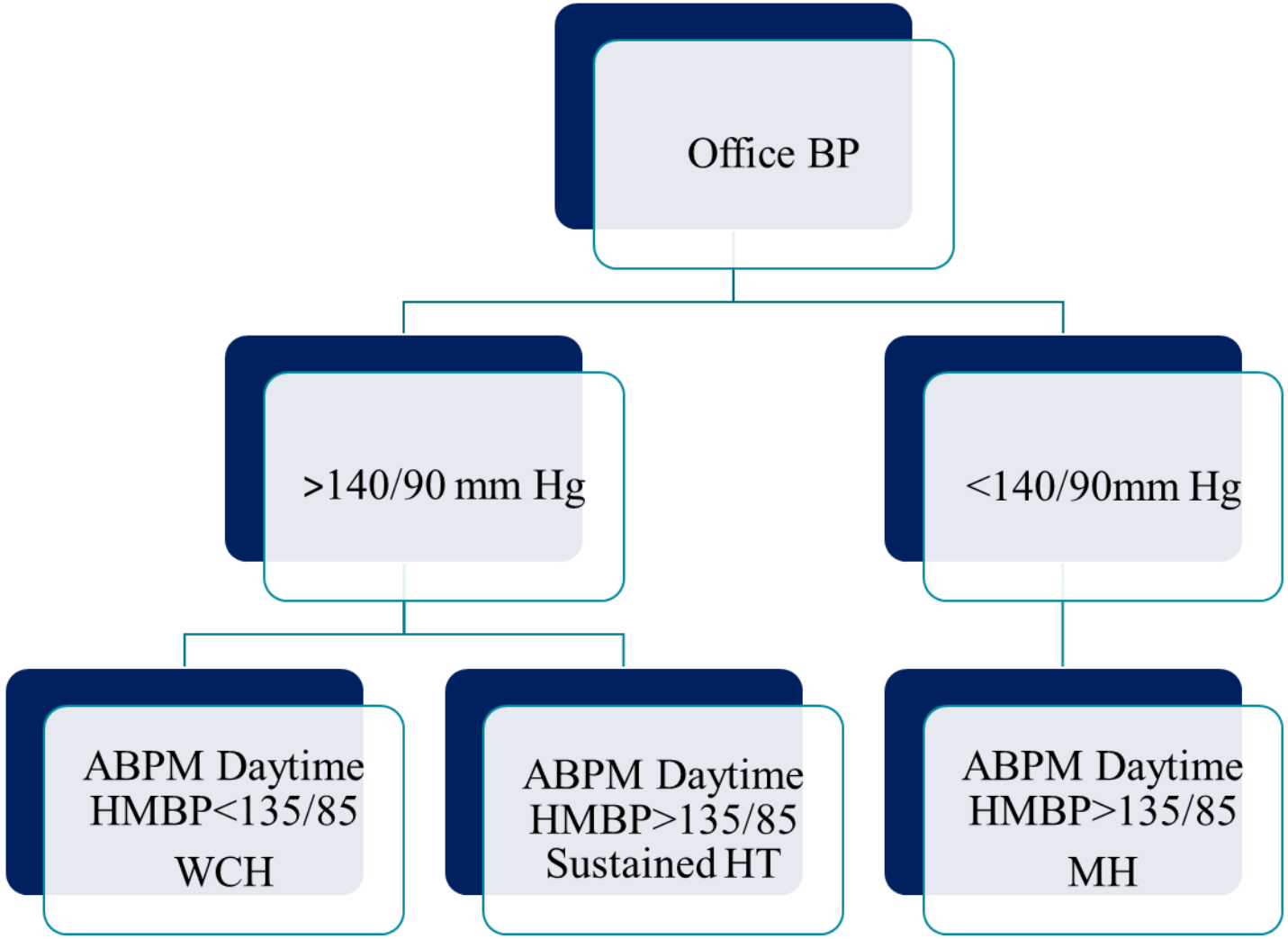
- Recommends office-based BP measurements for diagnosis
- Standardized ,Validated, cuffed devices should be used
- 2 separate office visits within 4 wks (except grade 3 HT /HMOD

Accurate BP Measurement



- No talking, caffeine, smoking, exercise(30 min)
- Bladder should be empty
- Bp cuff -80% of arm circumference at level of rt atrium
- Triplicate measures- average of last 2 readings .
- Both arms (first visit) .Use the arm with higher reading.
- For auscultatory determinations, use a palpated estimate of radial pulse obliteration pressure to estimate SBP. Inflate the cuff 20–30 mm Hg above this level, deflate the cuff pressure 2 mm Hg per second

White Coat Hypertension (WCH)/Masked Hypertension(MH)

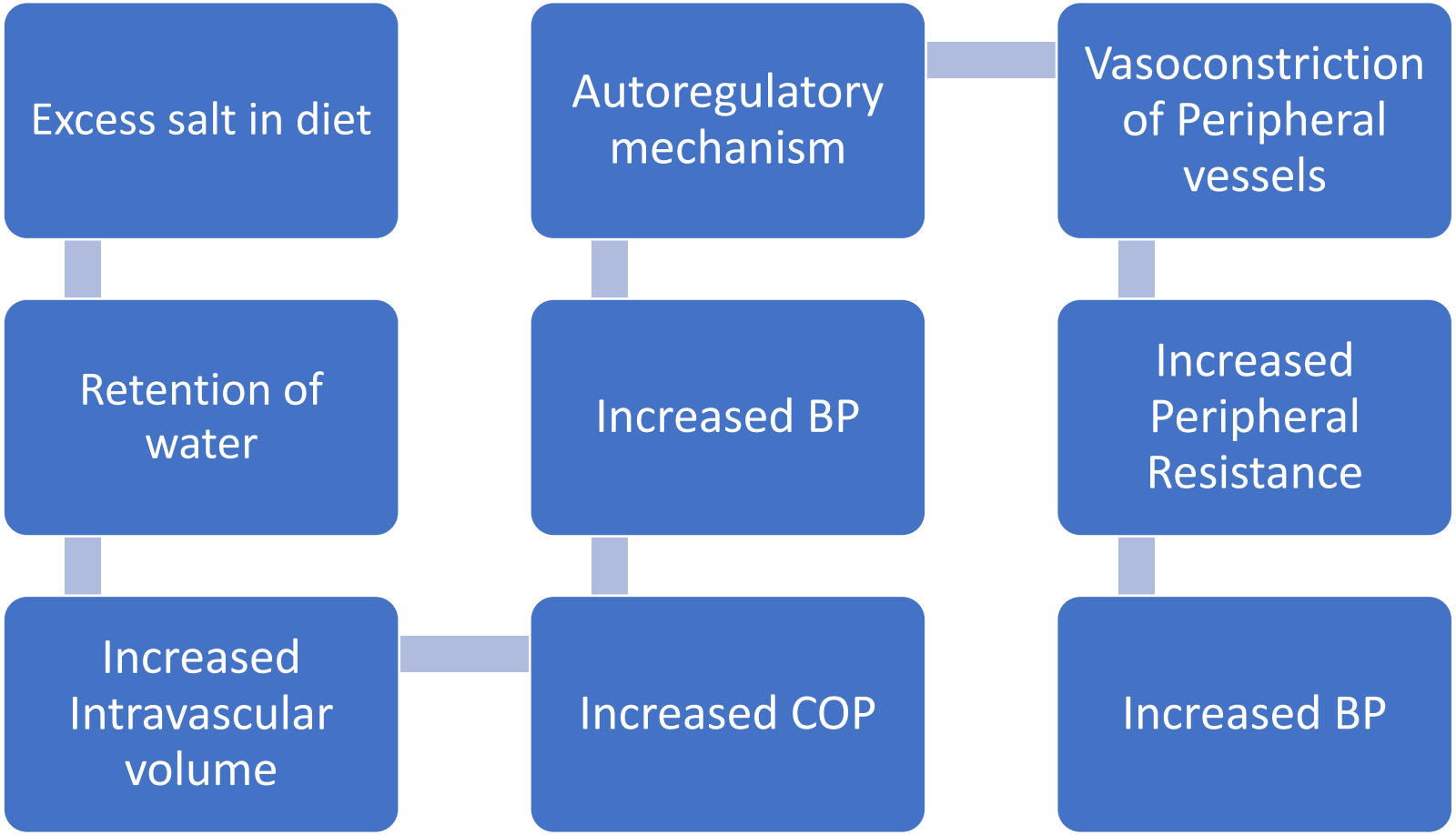


Similarities &Differences Between ACC/AHA and ESH Guidelines on Hypertension

Guideline difference	ACC/AHA 2017	2023 ESH
Hypertension Definition mmHg	> 130/80	>140/90 mmHg
Normal BP Ranges mmHg	Normal <120/80 Elevated 120-129/<80	Optimal <120/80 Normal 120-129/80-84 High Normal 130-139/85-89
Hypertension mmHg	Stage 1 -130-139/80-89 Stage 2->140/90	Grade 1-140-159/90-99 Grade 2-160-179/100-109 Grade3->180/110
BP Targets mmHg		
18-64 yrs	<130/80	<130/80
65-79 yrs	<130/80	<140/80
>80 yrs	<130/80	<140-150/ 80

Pathogenesis of HT

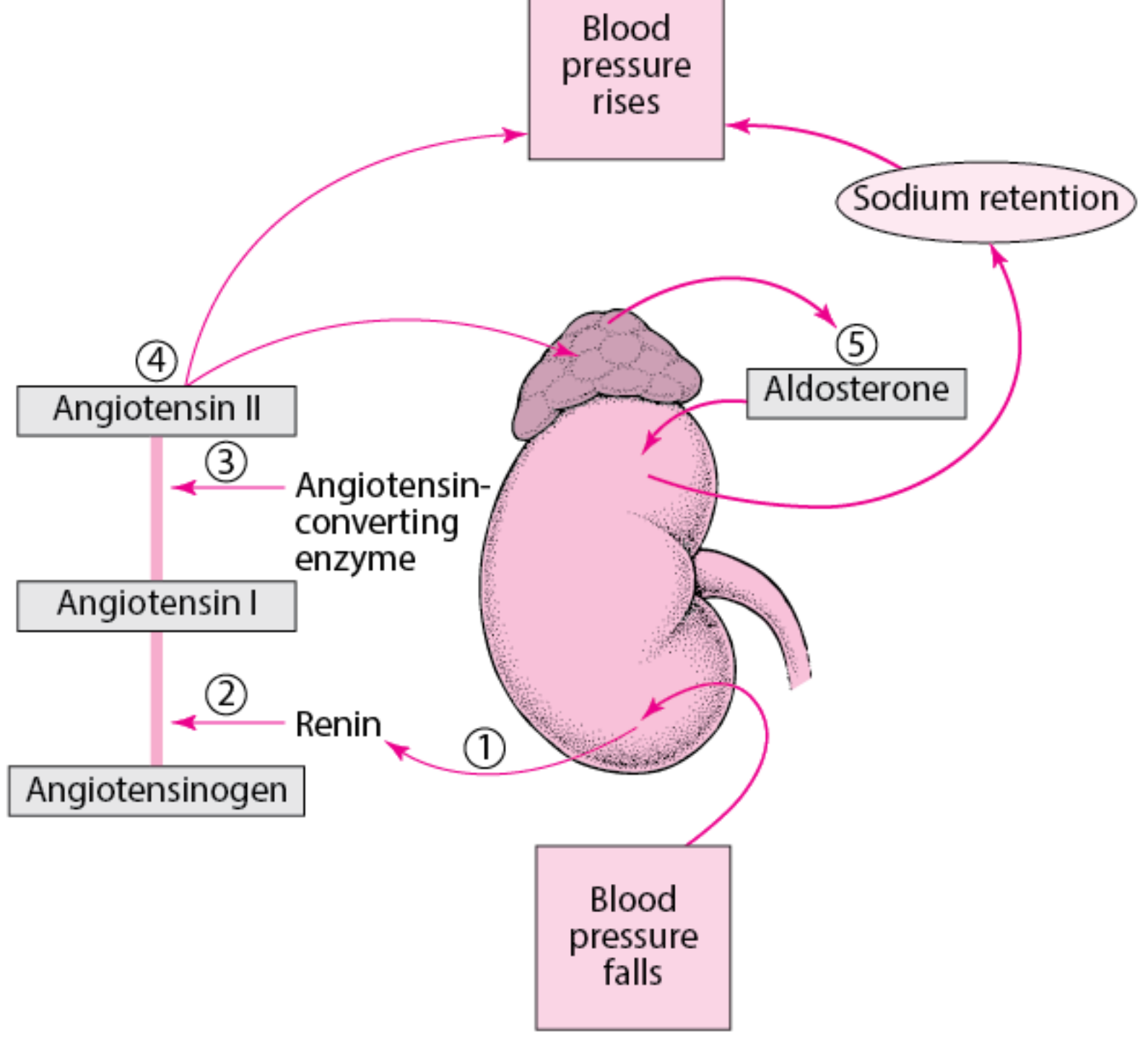
Salt and HT


Excess salt in diet
Retention of water
Increased Intravascular volume
Increased COP
Autoregulatory mechanism
Increased BP
Vasoconstriction of Peripheral vessels
Increased Peripheral Resistance
Increased BP

ANS

- Alpha receptors -- Norepinephrine
- Beta receptors -- Epinephrine
- Alpha1 –Post synaptic smooth muscles –Vasoconstriction
- Alpha2 –Presynaptic region activation causes negative feedback –inhibits Norepinephrine
- Beta1 activation cause increase in HR and force of contraction
- Beta 2 activation causes Vasodilatation
- Arterial baroreflex is mediated by nerve endings in carotid sinus and Aotic arch ,gets stimulated by increase in BP which reduces Sympathetic system activity

Renin Angiotensin system



Vascular mechanism

- Decrease in lumen of small arterioles
- Arterial stiffness
- Plaque

Types of HT

Primary HT

- Genetic plus Enviormental

Secondary HT

- Have some cause

When to suspect secondary Hypertension??

- Resistant hypertension -persistent blood pressure greater than 140/90 mm Hg despite using optimal doses of at least three anti-hypertensive from different classes, that includes a diuretic
- An acute rise in blood pressure in a patient who had previously stable pressures.
- Hypertension before the age of 30 years, who do not have any other risk factors for hypertension, e.g., obesity, family history, etc.
- Severe hypertension (BP greater than 180/110 mm Hg) with end-organ damage like acute kidney injury, neurological manifestations, flash pulmonary edema, hypertensive retinopathy, left ventricular hypertrophy, etc.
- Associated with electrolyte disorders like hypokalemia or metabolic alkalosis
- Age of onset of hypertension before puberty.
- Non-dipping or reverse dipping patterns while monitoring 24-hour ambulatory blood pressure. Normally, the blood pressure at night is lower than the blood pressure during the day

Secondary HT – Causes?

Secondary hypertension

Renal hypertension
- bilateral renal disease
  - acute and chronic glomerulonephritis
  - chronic interstitial nephritis
  - cystic kidney disease
  - diabetic nephropathy
  - collagen–vascular disorders
- unilateral renal disease
  - congenital hypoplasia
  - vesicoureteral reflux
  - unilateral hydronephrosis
  - postradiation nephritis
- renovascular disease (unilateral or bilateral)
  - atherosclerotic renal artery stenosis
  - fibromuscular dysplasia
  - rare causes
- renin-producing tumor
- kidney transplantation

Endocrine hypertension
- Cushing syndrome
- mineralocorticoid hypertension
- pheochromocytoma
- hyperthyroidism
- hypothyroidism
- primary hyperparathyroidism
- acromegaly
- neuroendocrine tumor (carcinoid)

Cardiovascular hypertension
- coarctation of the aorta
- increased cardiac output

Neurogenic hypertension
- increased intracranial pressure
- sleep apnea syndrome
- acute porphyria
- lead intoxication

Hypertension in pregnancy

Toxic agent-induced and drug-induced hypertension
- contraceptives
- nonsteroidal antiinflammatory drugs (NSAIDs)
- sympathicomimetics
- erythropoietin
- cyclosporine
- alcohol
- amphetamine
- cocaine
- anabolic steroids

Renal disease

- CRF- 80%HT
- Glomerular Diseases have more HT then Interstitial diseases
- Nephrosclerosis --HT ?
- Proteinuria >1000mg/day ,Active urine sediments –Renal Disease

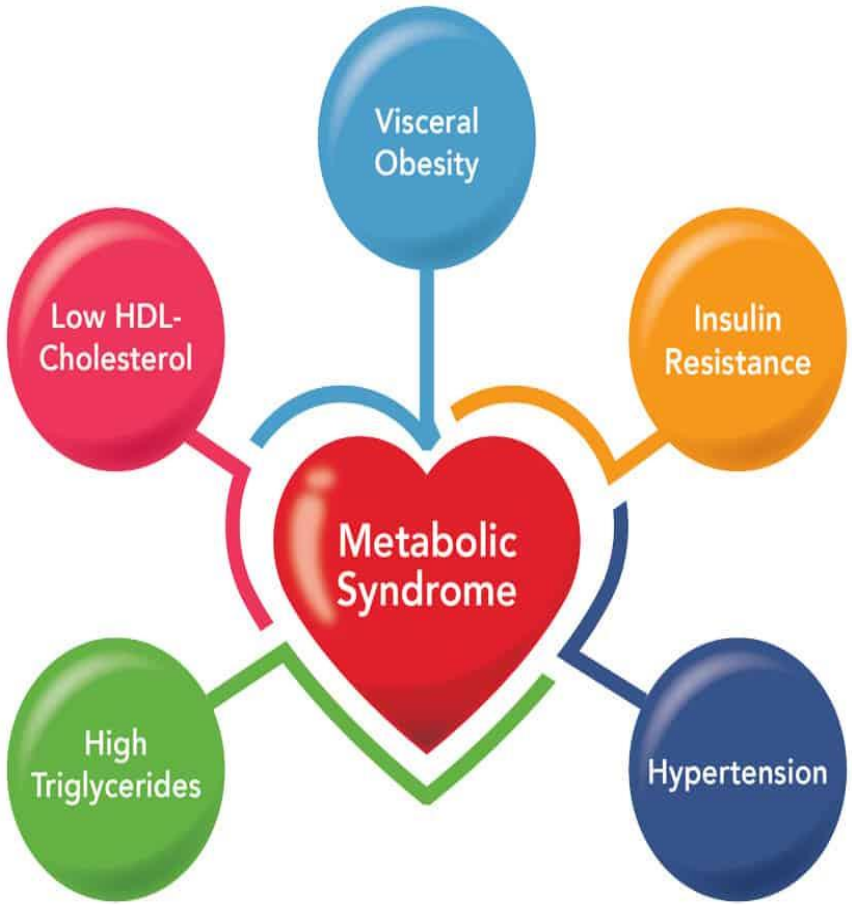
Renovascular HT

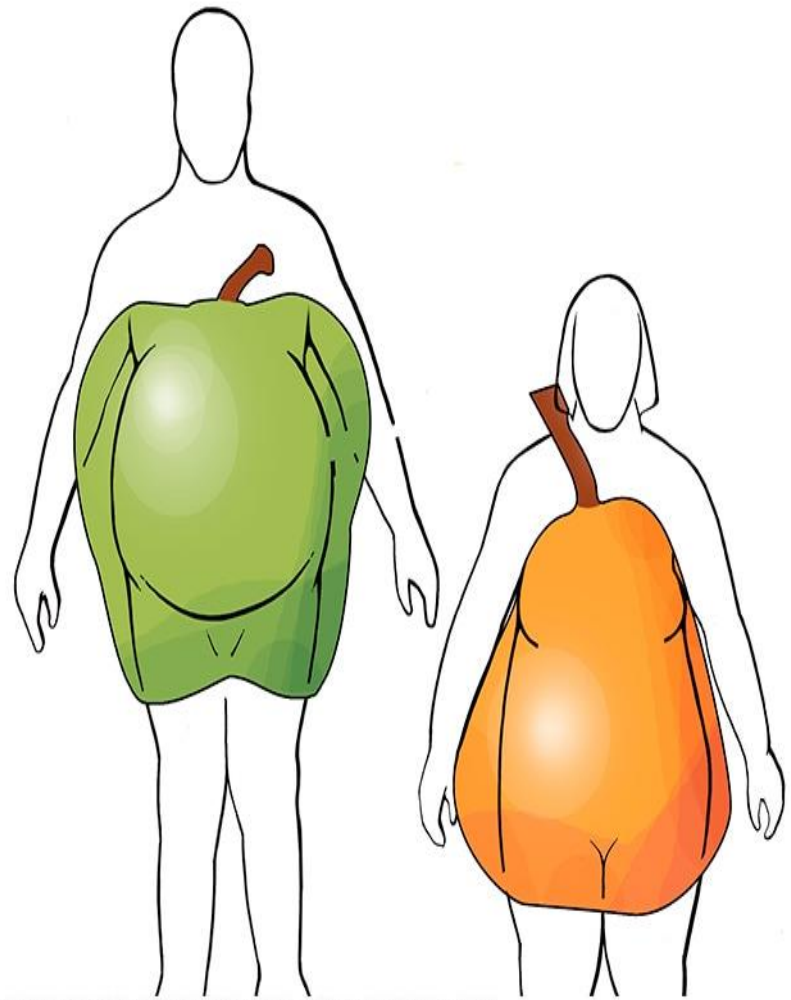
- Renal artery stenosis –Arteriosclerotic,Fibromuscular dysplasia
- When to suspect?-HT, ACE Inhibitor worsens HT
- How to Diagnose?-DTPA Scan,Renal Angiography
- How to Rx?-PTRA,Stent,Surgical repair

Hyperaldosteronism

- Hypokalaemic Alkalosis
- Polyuria,Polydipsia
- PA/PRA >30:1 -Screening test

What is Metabolic Syndrome?





- History /Clinical Examination/ Investigations.

- HMOD.

ECG
UACR
Sr Creatinine, e GFR
Extended Screening
Echo
cfPWV/baPWV
Carotid artery USG
Coronary artery calcium score

Kidney ,Aorta Usg
ABI
Retina microvascular changes
Brain Imaging
Cognitive MMSE

Management

Life Style Changes

01 Wt loss –Low Calorie Diet
Each Kg Wt loss causes reduction of SBP and DBP by 1 mmHg

02 Exercise 150-300 minutes of aerobic exercise/wk

03 Sodium intake less then <5.8 gm/day reduces SBP/DBP by 5/2 mmHg
Sodium 2 gm =5 gm Nacl

04 Increase K in diet but not excess

05 DASH Diet

06 Stop Alcohol ,Smoking

Drug Therapy When and What to start ?(with Life Style Changes)

- BP >140/90 mmHg. (18-79 years)
- CAD and BP ≥130/80 mm Hg/ Estimated 10-year risk of atherosclerotic CVD >10% Hg. (ACC)
- >80 years when SBP is >160 mm Hg(ESH), whereas the ACC/AHA guideline does not make this distinction. BP >140/90 mmHg Rx can be considered.
- Single pill combination therapy. ACC/AHA recommends this approach for patients with stage 2 hypertension, those with BP >20/10 mm Hg above their target BP, and Black patients.
- Monotherapy -Grade 1 HT with low CV risk, High normal BP with high CV risk, Frailty/adv age

Initial Therapies - Four Major Classes.
The ESH guidelines continue to include Beta-Blockers

- Angiotensin Converting Enzyme Inhibitors (ACEIs)
- Angiotensin-Receptor Blockers (ARBs)
- Thiazide/Thiazide likes Diuretics
- Calcium Channel Blockers (CCBs).

TABLE 277-9 Examples of Oral Drugs Used in Treatment of Hypertension

DRUG CLASS	EXAMPLES	USUAL TOTAL DAILY DOSE[a] (DOSING FREQUENCY/DAY)	OTHER INDICATIONS	CONTRAINDICATIONS/CAUTIONS
Diuretics				
Thiazides	Hydrochlorothiazide	6.25–50 mg (1–2)		Diabetes, dyslipidemia, hyperuricemia, gout, hypokalemia
	Chlorthalidone	25–50 mg (1)		
Loop diuretics	Furosemide	40–80 mg (2–3)	CHF due to systolic dysfunction, CHF with preserved ejection fraction, renal failure	Diabetes, dyslipidemia, hyperuricemia, gout, hypokalemia
	Ethacrynic acid	50–100 mg (2–3)		
Aldosterone antagonists	Spironolactone	25–100 mg (1–2)	CHF, primary aldosteronism, resistant hypertension	Renal failure, hyperkalemia
	Eplerenone	50–100 mg (1–2)		
K^+ retaining	Amiloride	5–10 mg (1–2)	Liddle's syndrome	Renal failure, hyperkalemia
	Triamterene	50–100 mg (1–2)		
Beta blockers				Asthma, COPD, second- or third-degree heart block, sick-sinus syndrome
Cardioselective	Atenolol	25–100 mg (1)	Angina, CHF, post-MI, sinus tachycardia, ventricular tachyarrhythmias, thoracic aortic disease	
	Metoprolol	25–100 mg (1–2)		
Nonselective	Propranolol	40–160 mg (2)		
	Propranolol LA	60–180 (1)		
Combined alpha/beta	Labetalol	200–800 mg (2)		
	Carvedilol	12.5–50 mg (2)		
Alpha antagonists				
Selective	Prazosin	2–20 mg (2–3)	Prostatism	
	Doxazosin	1–16 mg (1)		
	Terazosin	1–10 mg (1–2)		
Nonselective	Phenoxybenzamine	20–120 mg (2–3)	Pheochromocytoma	

Sympatholytics				
Central	Clonidine	0.1–0.6 mg (2)		
	Clonidine patch	0.1–0.3 mg (1/week)		
	Methyldopa	250–1000 mg (2)		
	Reserpine	0.05–0.25 mg (1)		
	Guanfacine	0.5–2 mg (1)		
ACE inhibitors	Captopril	25–200 mg (2)	Post-MI, coronary syndromes, CHF, nephropathy	Acute renal failure, bilateral renal artery stenosis, pregnancy, hyperkalemia
	Lisinopril	10–40 mg (1)		
	Ramipril	2.5–20 mg (1–2)		
Angiotensin II antagonists	Losartan	25–100 mg (1–2)	CHF, nephropathy, ACE inhibitor cough	Renal failure, bilateral renal artery stenosis, pregnancy, hyperkalemia
	Valsartan	80–320 mg (1)		
	Candesartan	2–32 mg (1–2)		
Renin inhibitors	Aliskiren	150–300 mg (1)	Diabetic nephropathy	Pregnancy
Calcium antagonists				
Dihydropyridines	Nifedipine (long-acting)	30–60 mg (1)		
Nondihydropyridines	Verapamil (long-acting)	120–360 mg (1–2)	Post-MI, supraventricular tachycardias, angina	Second- or third-degree heart block
	Diltiazem (long-acting)	180–420 mg (1)		
Direct vasodilators	Hydralazine	25–100 mg (2)		Severe coronary artery disease
	Minoxidil	2.5–80 mg (1–2)		

[1]At the initiation of therapy, lower doses may be preferable for elderly patients and for select combinations of antihypertensive agents.

Abbreviations: ACE, angiotensin-converting enzyme; CHF, congestive heart failure; COPD, chronic obstructive pulmonary disease; MI, myocardial infarction.

- Preferred combinations are ARS Inhibitors plus CC Blockers or Thiazide Diuretics
- Beta blockers to be added in specific conditions
- CCB –DHP CCB can be used in HFrEF with caution –negative ionotropic effect.Non DHP CCB not to be used in HFrEF
- Thiazides fair well in Heart failure
- Alpha blockers cause Orthostatic Hypotension .Avoid in older people
- Central acting drugs have side effects use only in special conditions.
- MRA can be used in resistant hypertension after 3 drugs.
- Drug titration should be not very slow in high CV risk cases

Beta blocker controversy?

Why beta-blockers should not be used as first choice in uncomplicated hypertension

Alberto Ranieri De Caterina 1, Antonio Maria Leone
Affiliations Expand
PMID: 20451690 DOI: 10.1016/j.amjcard.2009.12.068

Abstract
In the past 4 decades, beta blockers (BBs) have been widely used in the treatment of uncomplicated hypertension and are still recommended as first-line agents in national and international guidelines. Their putative cardioprotective properties, however, derive from the extrapolation into primary prevention of data relative to the reduction of mortality observed in the 1970s in patients with previous myocardial infarctions. In the past 5 years, a critical reanalysis of older trials, together with several meta-analyses, has shown that in patients with uncomplicated hypertension BBs exert a relatively weak effect in reducing stroke compared to placebo or no treatment, do not have any protective effect with regard to coronary artery disease and, compared to other drugs, such as calcium channel blockers, renin-angiotensin-aldosterone system inhibitors or thiazide diuretics, show evidence of worse outcomes, particularly with regard to stroke.

β blockers switched to first-line therapy in hypertension

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Cite
https://doi.org/10.1016/S0140-6736(23)01733-6
The Lancet, Volume 402, Issue 10414, 11–17 November 2023, Pages 1729-1730

The ESH guidelines and missed prevention opportunities

Summary
In their recent guidelines, the European Society of Hypertension upgraded β blockers, putting them on equal footing with thiazide diuretics, renin–angiotensin system blockers (eg, angiotensin-converting enzyme inhibitors and angiotensin receptor blockers), and calcium channel blockers. The reason offered for upgrading β blockers was the observation that they are often used for many other clinical conditions commonly encountered with hypertension. This upgrade would allow for the treatment of two conditions with a single drug (a so-called twofer). In most current national and international hypertension guidelines, β blockers are only considered to be an alternative when there are specific indications.

HT and comorbities

01

ESH
<140/90 mm Hg
CKD
<130/80 mm Hg
CAD, DM, and cerebrovascular disease

02

ACC/AHA guideline
<130/80 mm Hg
CKD, CAD, DM, and
cerebrovascular disease.

CVA and Blood Pressure

01

Hemorrhagic Stroke<6hrs of symptoms

consider lowering BP <140/90 mm Hg.

Reduces Haematoma expansion.

02

Hemorrhagic Stroke>6hrs of symptoms

1.SBP>220 lower it to 180 mmHg

2.SBP<220 mmHg slow reduction to 140mm Hg

03

Acute Ischemic stroke

If Thrombolysis indicated lower BP to <185/110 mmHg ,maintain <180/105

If not eligible for Thrombolysis reduce BP by 15% during first 24 hrs if <220/120mmHg

Renal Denervation Therapy

- 2018 ESH did not recommend Renal Denervation as there was lack of trials
- Now there are highest-quality, multicenter, randomized, blinded trials which used Ambulatory Blood Pressure (BP) as the primary outcome.
- Useful in Resistant hypertension(triple drug therapy, one of which is a diuretic) /cannot tolerate too many medications.
- No significant renal artery stenosis or worsening of renal function.
- The BP-lowering effect was sustained for up to 3 years. Benefit is like a single medication, roughly 5-10 mm Hg
- Patients must be fully informed about the risks and benefits of renal denervation when compared to drug therapy.

Renal Denervation Therapy

01

02

03

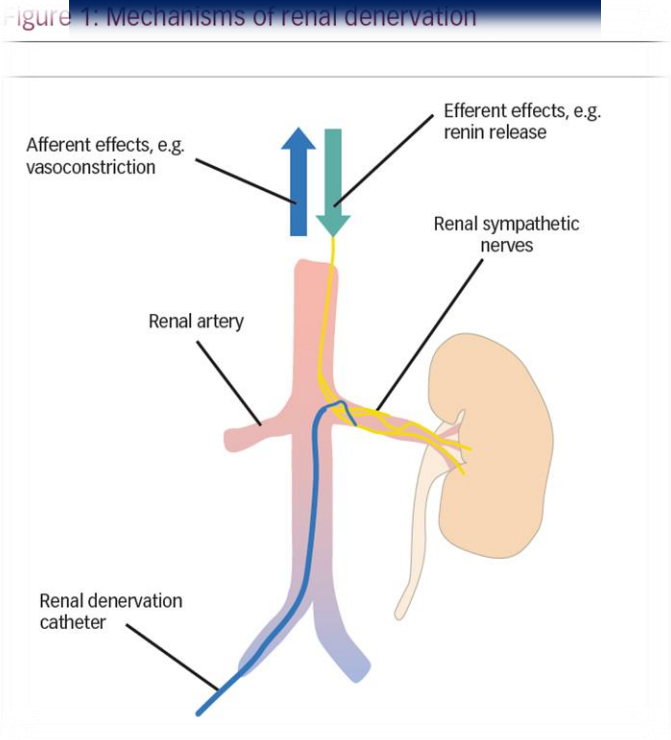

Figure 1: Mechanisms of renal denervation


Thank
You