

Hypertension Review

MEDICINE COURSE 442: 2020 - 2021
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Outline

- Presentations
 - Introduction
 - Evidence
 - Definitions
 - Summary

Case 1

- 35-year-old woman was referred to your clinic after undergoing screening for employment. Her blood pressure was found to be high (149/93 mmHg). She has no complaints and is otherwise healthy.
- PMH: nil
- Meds: nil
- FHx: High BP in father and brother
- SHx: Single, works as a nurse, no illicit drug use.

Case 1

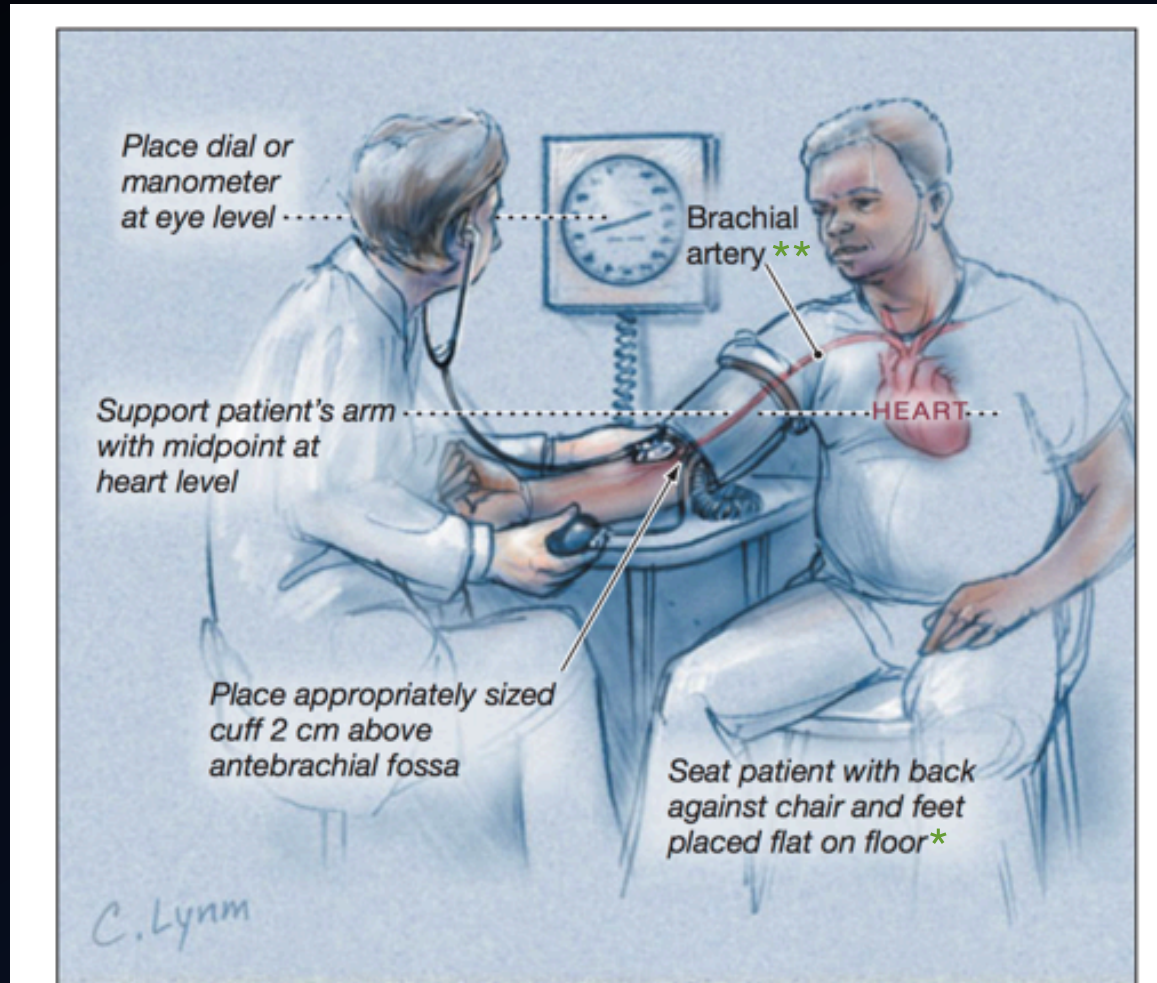
- On physical exam: BMI 29, looks well, not in distress
- BP: 145/93, HR: 78
- CVS: S1 + S2 + 0
- Chest: clear
- Abd: Soft, Lax, no bruit
- Rheum and Neuro: normal

Case 1

- What is your next step in management?
 - A. Advise for lifestyle modification and follow up in 3 months
 - B. Repeat blood pressure measurement in the office after 2 weeks
 - C. Perform a 24-hour ambulatory blood pressure monitor
 - D. Ask for home blood pressure readings over one week

ANS: C because she has family history so this is most correct, but B and D could also be correct

How is blood pressure measured? classic OSCE station



**avoid wrist measurement

*crossing legs will increase blood pressure

Figure 23-1 Clinical Measurement of Indirect Blood Pressure
See Table 23-2 for appropriate cuff sizing.

Other types blood pressure measurement devices :

- Mercury based manometer (phasing out)
- Oscillometer based measurements (most automated BP machines)

Epidemiology

- 1 in 3 adults (>20 years old) have high blood pressure
- Incidence increases with aging
- 5 – 10% of all hypertension is secondary
- Searching for secondary hypertension is expensive and cumbersome *This is why we have guidelines to minimize wasting resources*
- Need better selection of who to screen and offer specific treatments

Before 1960 they always associated HTN with bad outcomes, then on 1960 this study came out in the journal of medical association: they treated people with BP 115-129 and compared patients with active treatment and no treatment.

Effects of Treatment on Morbidity in Hypertension

Results in Patients With Diastolic Blood Pressures
Averaging 115 Through 129 mm Hg

Veterans Administration Cooperative Study Group on Antihypertensive Agents

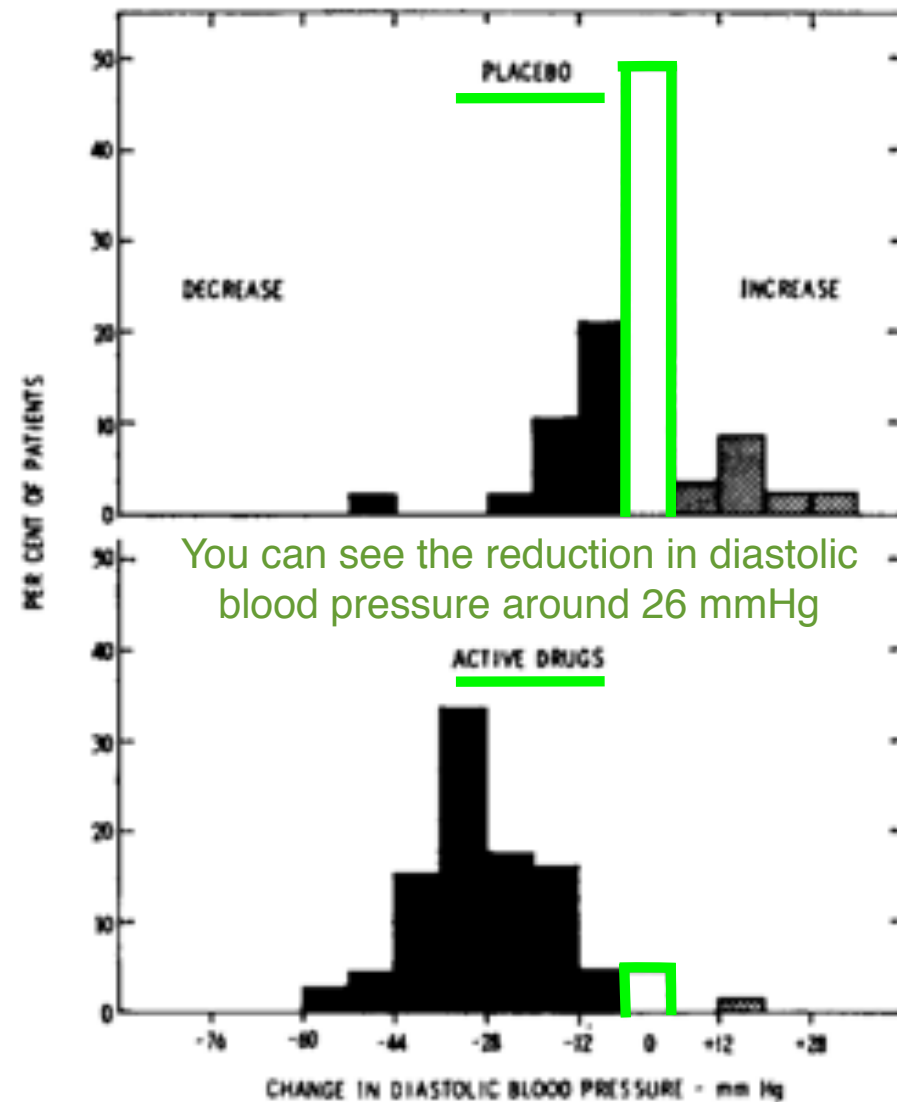
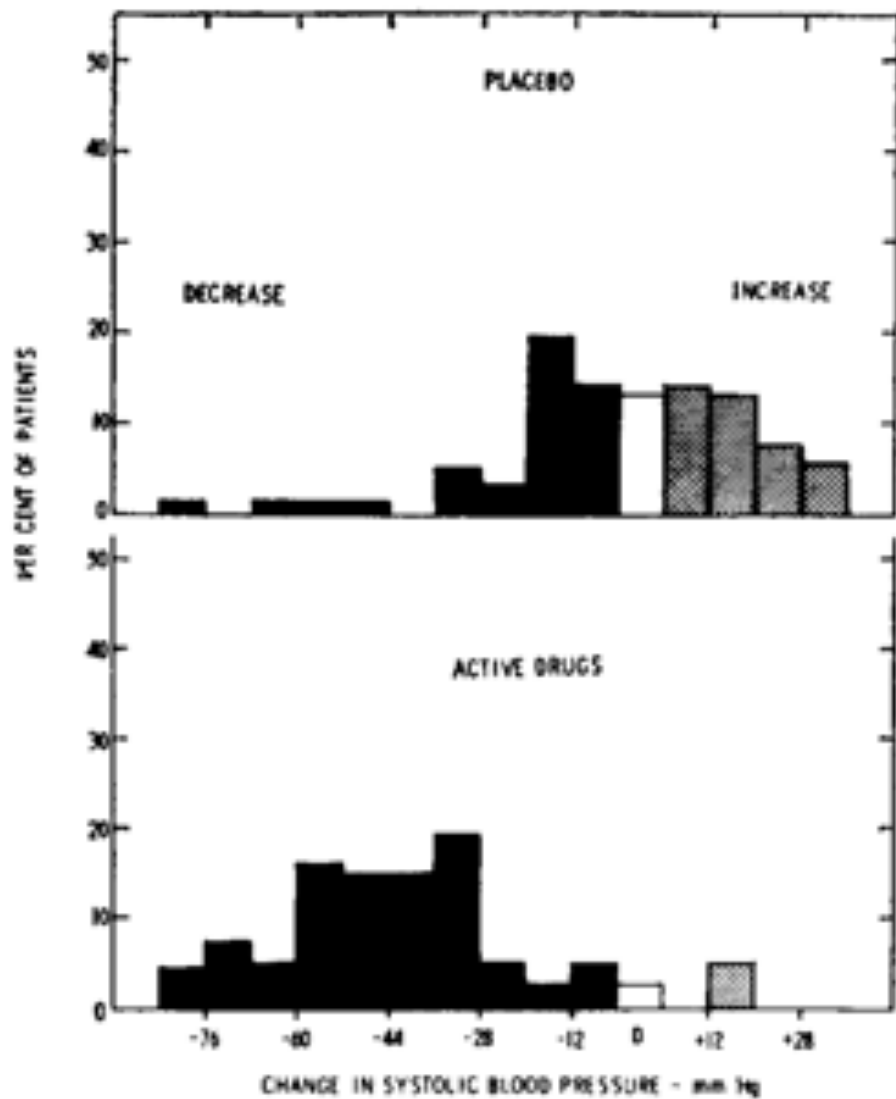
A group of 143 male hypertensive patients with diastolic blood pressures (at the clinic) averaging between 115 and 129 mm Hg were randomly assigned to either active (hydrochlorothiazide plus reserpine plus hydralazine hydrochloride) or placebo treatment. Twenty-seven severe, complicating events developed in the placebo-treated patients as compared to two in the active group. Four deaths occurred in the placebo-treated group and none in the actively treated patients. Other complications in the placebo group included grade 3 or 4 hypertensive retinopathy, congestive heart failure, increasing azotemia, cerebrovascular thrombosis, transient ischemic attacks, cerebral hemorrhage, myocardial infarction, and severely elevated blood pressure. Severe complications in the active-treatment group were one cerebrovascular thrombosis and one case of multiple drug toxicity. Male patients with diastolic blood pressures averaging 115 mm Hg or above represent a high-risk group in which antihypertensive therapy exerts a significant beneficial effect.

without signs of accelerated hypertension at admission whose diastolic blood pressures prior to treatment averaged 115 through 129 mm Hg.

Plan of Investigation

All patients were hospitalized for the initial work-up. Male patients whose diastolic blood pressures from the fourth through the sixth day of hospitalization averaged 90 through 129 mm Hg without treatment were considered for admission to the prerandomization trial period.

Severity was evaluated in five categories. These were the average diastolic blood pressure during hospitalization and the degree of clinically detectable hypertensive damage in the following four target organs: the optic fundi, the brain, heart, and kidneys. Severity of damage in each category was graded on a scale from 0 (no detectable abnormality) to 4 (most severe changes). The criteria used



You can see the reduction in diastolic blood pressure around 26 mmHg

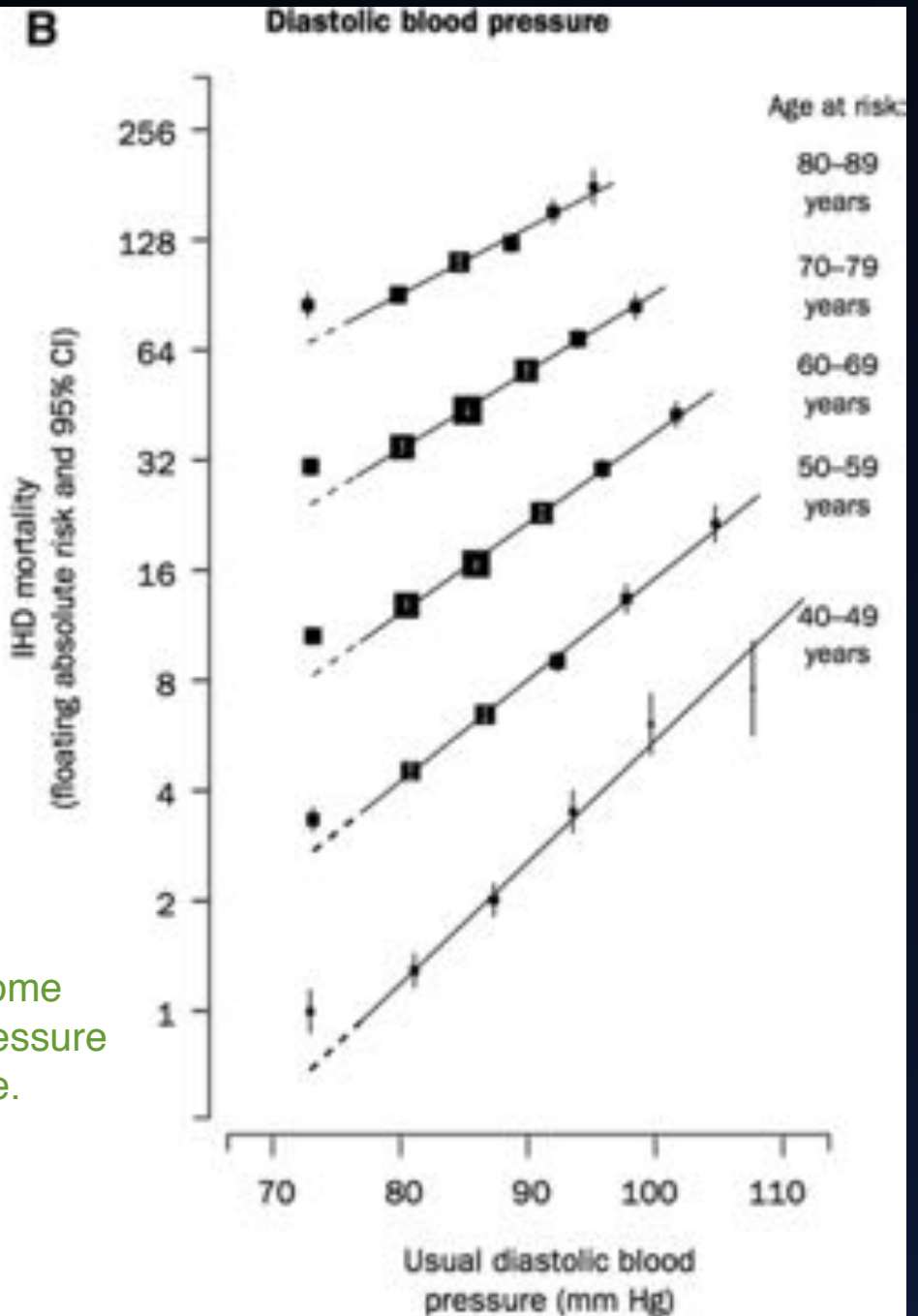
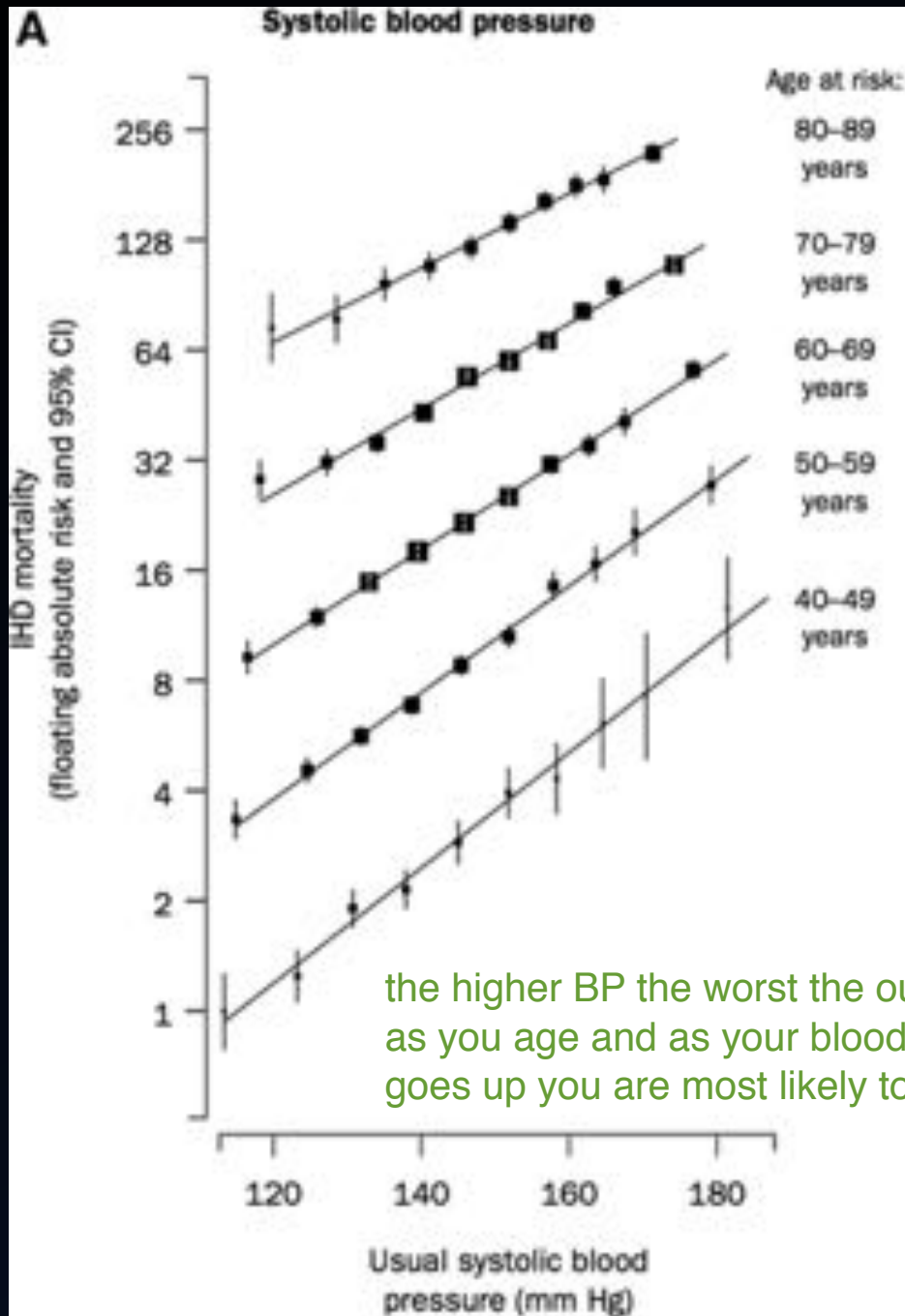
Changes in systolic (*left*) and diastolic blood pressure (*right*) after four months of treatment in 57 patients given placebos (*above*) and 68 patients treated with hydrochlorothiazide plus reserpine plus hydralazine (*below*).

Modification in Treatment Regimens.—Of the 73

Table 4.—Incidence of Mortality and Morbidity

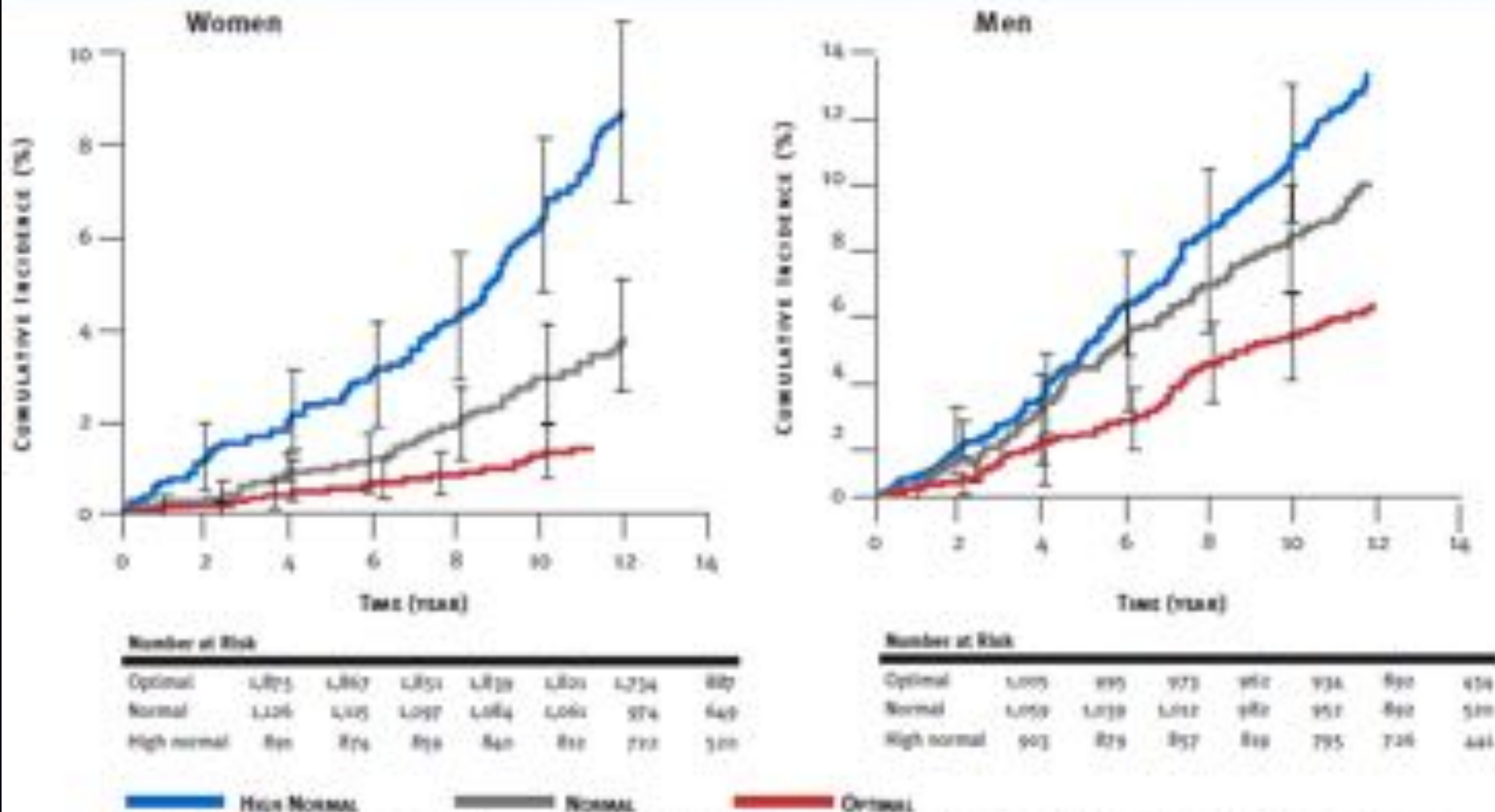
	Placebo-Treated Patients	Actively Treated Patients
Deaths	4	0
Class A events (stroke, heart failure, MI, death)	10	0
Subtotal	14	0
Other treatment failures	7	1
Total terminating events	21	1
Class B events (nonterminating)	6	1
Total	27	2

people who are treated had better outcome.



the higher BP the worst the outcome as you age and as your blood pressure goes up you are most likely to die.

Figure 11. Impact of high normal blood pressure on the risk of cardiovascular disease



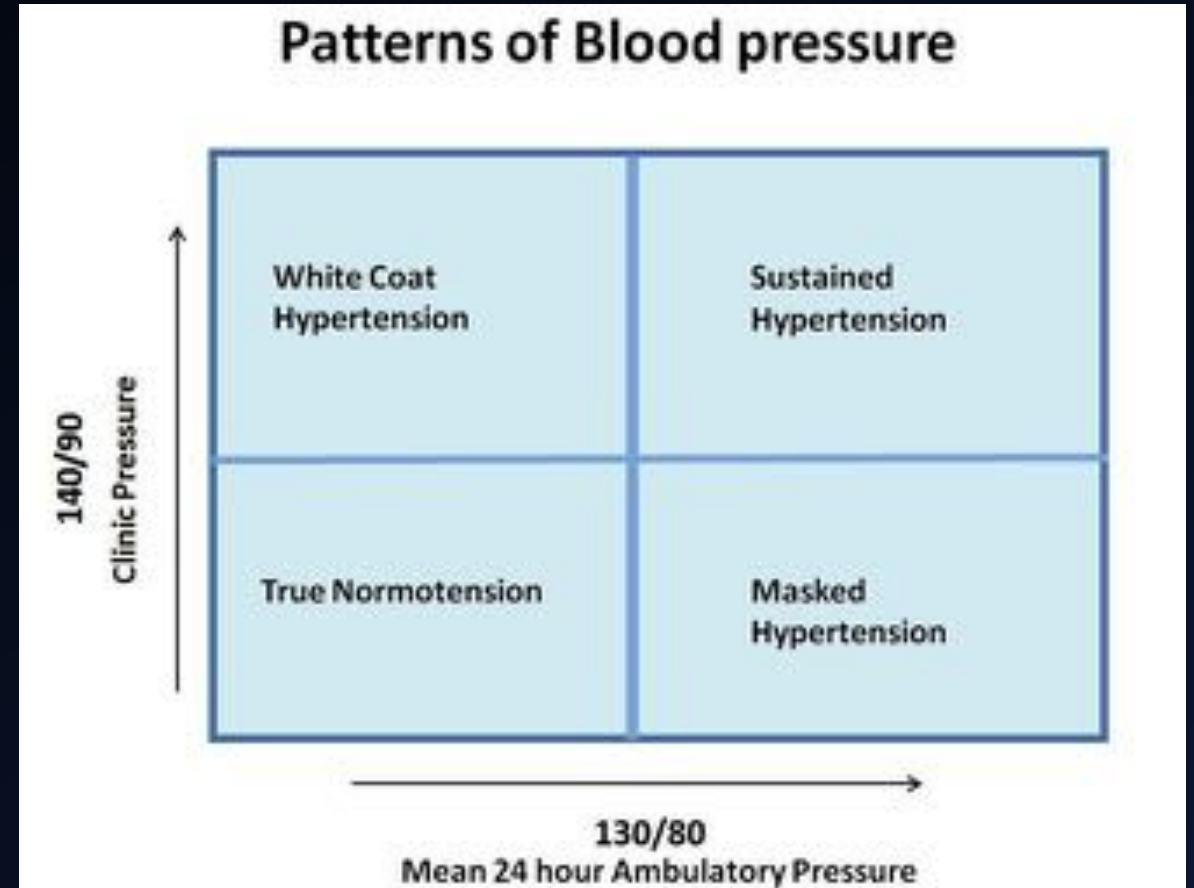
Cumulative incidence of cardiovascular events in women (panel A) and men (panel B) without hypertension, according to blood pressure category at the base-line examination. Vertical bars indicate 95 percent confidence intervals. Optimal BP is defined here as a systolic pressure of <120 mmHg and a diastolic pressure of <80 mmHg. Normal BP is a systolic pressure of 120-129 mmHg or a diastolic pressure of 80-84 mmHg. High-normal BP is a systolic pressure of 130-139 mmHg or a diastolic pressure of 85-89 mmHg. If the systolic and diastolic pressure readings for a subject were in different categories, the higher of the two categories was used.

Source: Vasan RS, et al. Impact of high-normal blood pressure on risk of cardiovascular disease. *N Engl J Med* 2001;345:1297-7. Copyright 2001, Massachusetts Medical Society. All rights reserved.

even high normal BP has worse outcome than normal BP

Hypertension definitions

- Prehypertension
- Hypertension
 - Stages
 - Urgency
 - Emergency
- White Coat syndrome
- Masked Hypertension



Prehypertension: office BP 130-140/80-90 (this name is no longer used)

Masked hypertension: normal BP at office but high on home 24hr ambulatory

Criteria for HTN diagnosis

*most commonly the diagnosis is made using office BP measured in 2 or 3 settings, however if you have symptoms of HTN or evidence of hypertensive disease or end organ damage, you only need 1 high reading.

** this is diagnostic

Table 3

Thresholds values for office, home and ambulatory blood pressure measurement

Category	Systolic (mmHg)	Diastolic (mmHg)
Office BP *	≥140	≥90
Home BP**	≥135	≥85
Ambulatory BP		
24 h	≥130	≥80
Daytime (or awake)	≥135	≥85
Nighttime (or asleep)	≥120	≥70

Ambulatory BP measurement

1.2.2 If blood pressure measured in the clinic is 140/90 mmHg or higher:

- Take a second measurement during the consultation.
- If the second measurement is substantially different from the first, take a third measurement. People often have variable readings during the same visit

Record the lower of the last two measurements as the clinic blood pressure.
[2011]

1.2.3 If the clinic blood pressure is 140/90 mmHg or higher, offer ambulatory blood pressure monitoring (ABPM) to confirm the diagnosis of hypertension. [2011] *

1.2.4 If a person is unable to tolerate ABPM, home blood pressure monitoring (HBPM) is a suitable alternative to confirm the diagnosis of hypertension. [2011]

*Not always available and some patients may not tolerate it so u can replace it with home monitoring NICE

Ambulatory BP measurement

Table 4.4 Recommendations on methods of blood pressure measurement

Methods of measuring blood pressure	Grade of recommendation	Level of evidence
a. If clinic blood pressure is $\geq 140/90$ mmHg, or hypertension is suspected, ambulatory and/or home monitoring should be offered to confirm the blood pressure level.	Strong	I
b. Clinic blood pressure measures are recommended for use in absolute CVD risk calculators. If home or ambulatory blood pressure measures are used in absolute CVD risk calculators, risk may be inappropriately underestimated.	Strong	–
c. Procedures for ambulatory blood pressure monitoring should be adequately explained to patients. Those undertaking home measurements require appropriate training under qualified supervision.	Strong	I
d. Finger and/or wrist blood pressure measuring devices are not recommended. <i>avoid this!!</i>	Strong	–

Ambulatory BP

in sleep apnea or chronic kidney disease or DM is they have nocturnal hypertension (non-dipping.) typically during sleep BP should go down, in this case there is absence of this.

Australian guidelines

Table 4.2 Clinical indications for out-of-clinic blood pressure measurements

Clinical indications for out-of-clinic blood pressure measurements

Suspicion of white-coat hypertension

Suspicion of masked hypertension

Identified white-coat hypertension

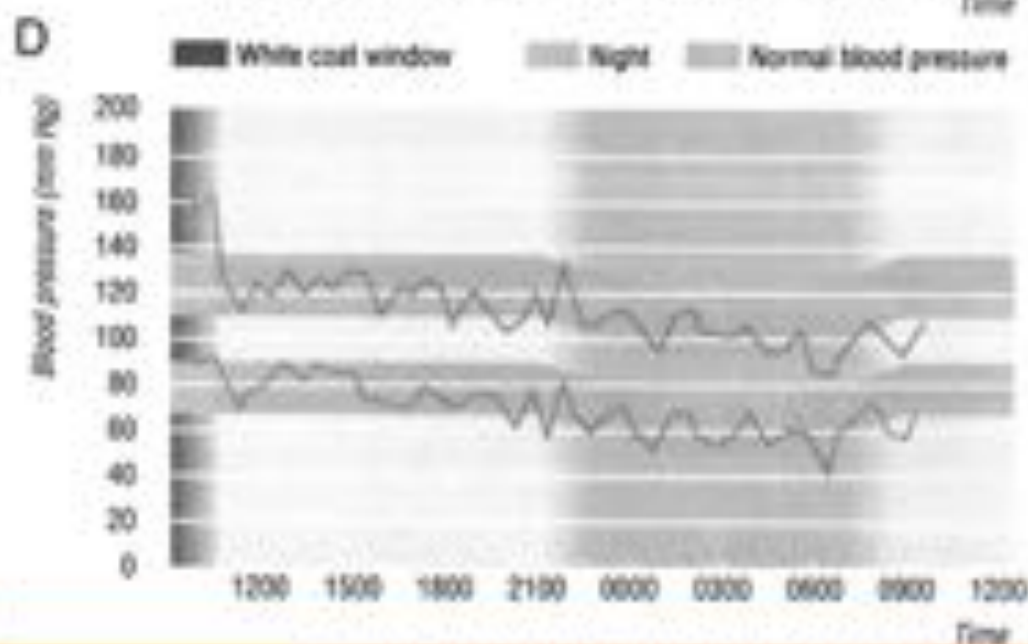
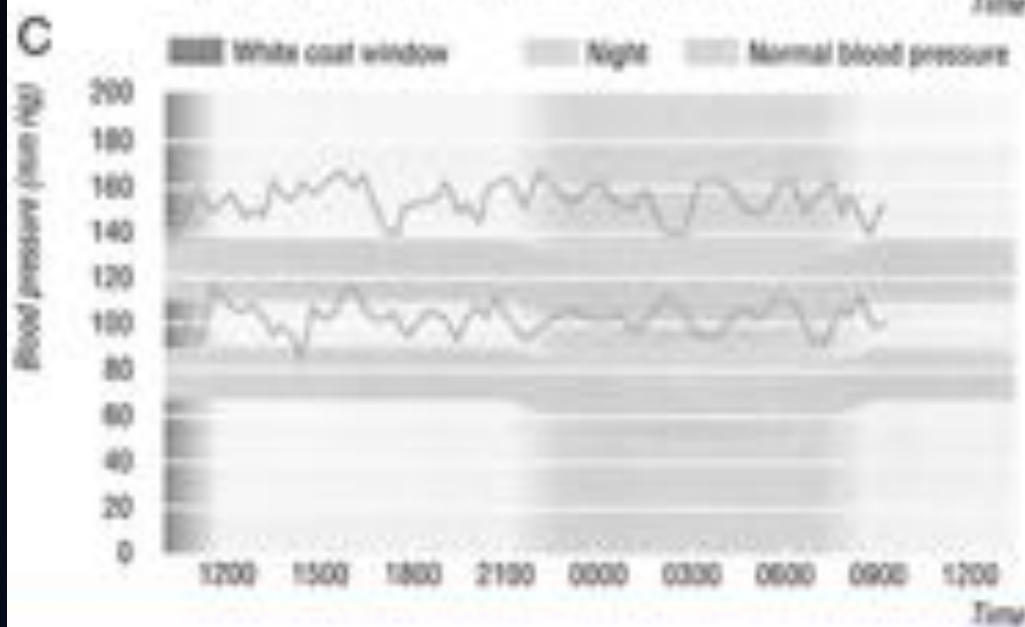
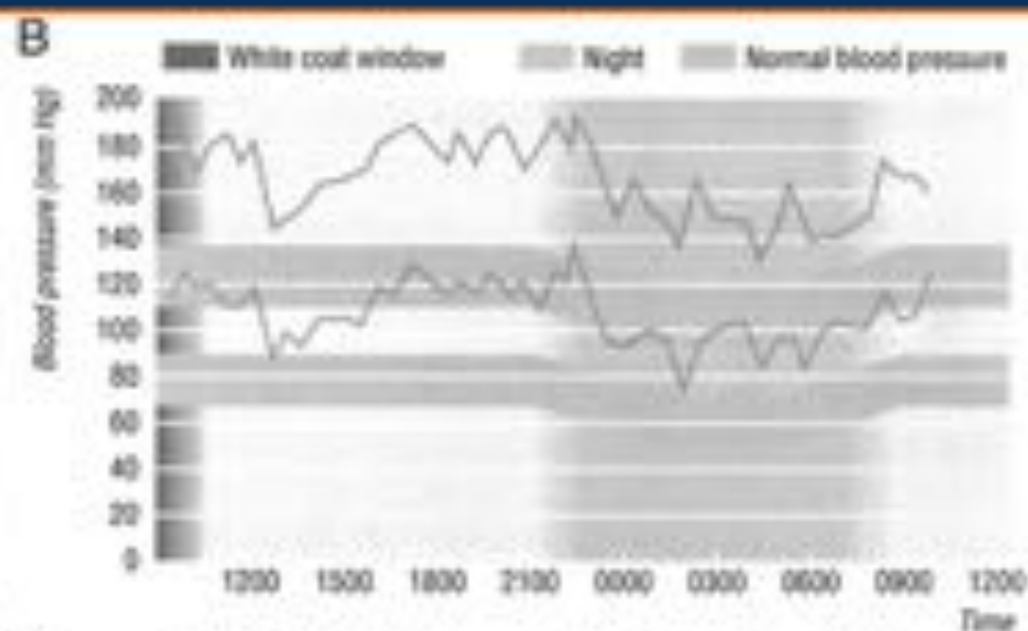
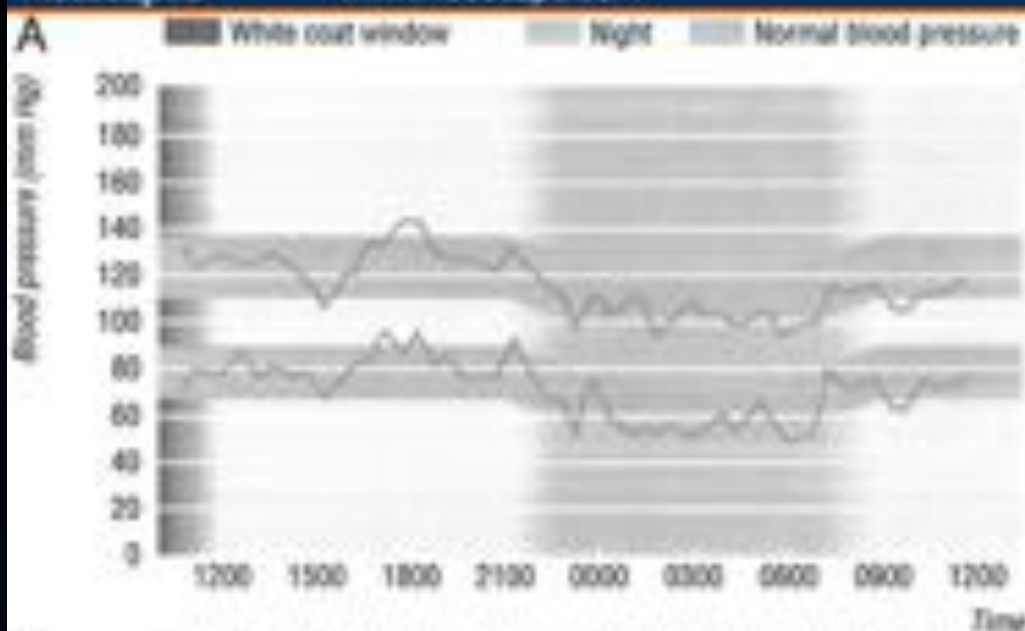
Marked variability of clinic or clinic and home blood pressure measurements

Autonomic, postural, post-prandial and drug-induced hypotension when starting new medication

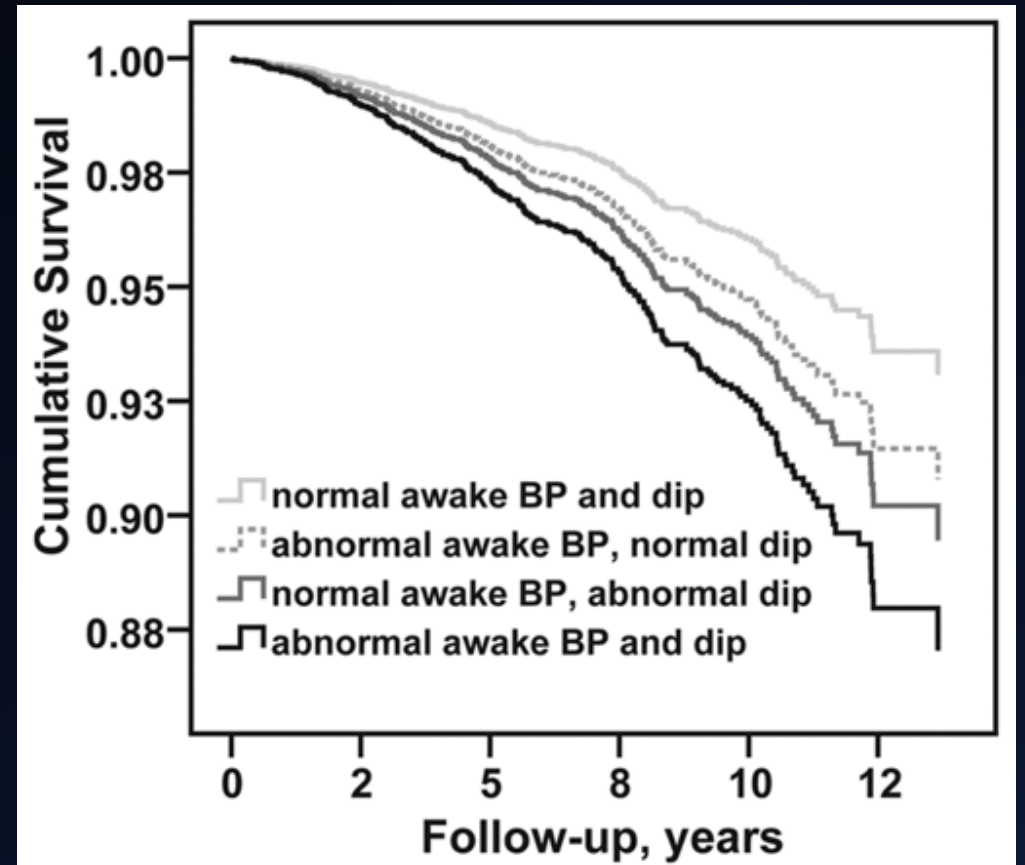
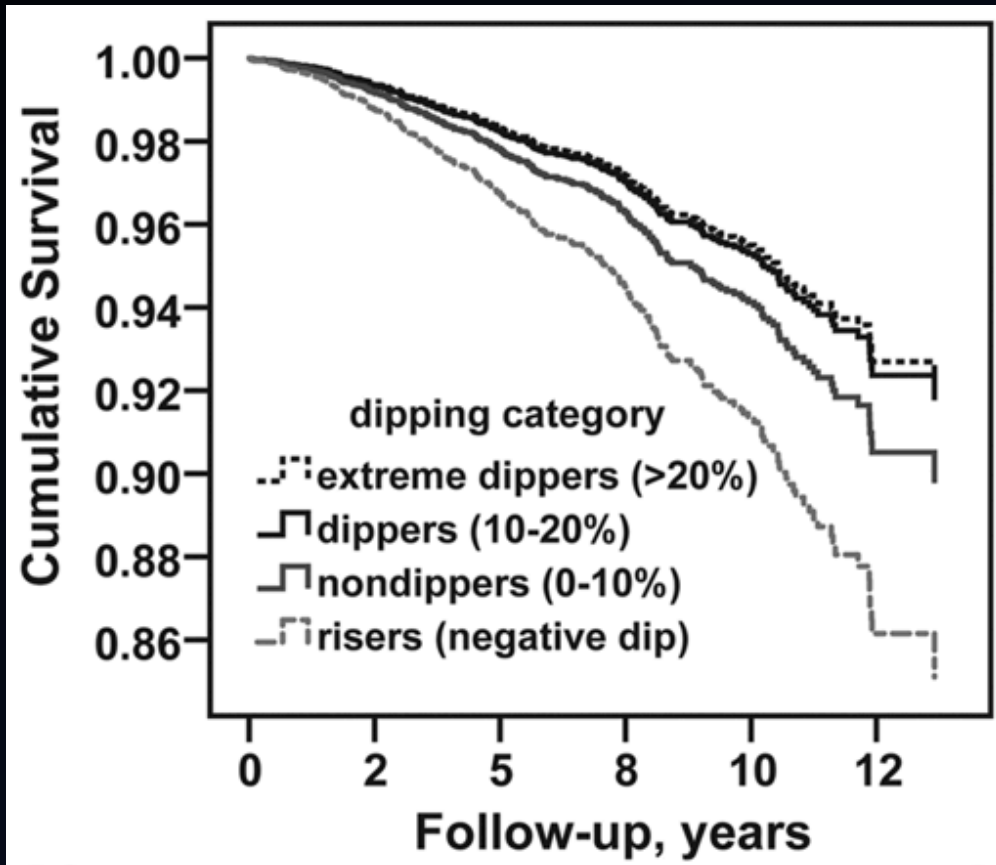
Identification of true resistant hypertension

Suspicion of nocturnal hypertension or absence of nocturnal dipping, for example in patients with sleep apnoea, chronic kidney disease or diabetes

Table adapted with permission from European Society of Hypertension guidelines²⁵ and Ambulatory blood pressure monitoring in Australia: 2011 consensus position statement.²³



Nocturnal Blood pressure



*Someone with HTN and they are young and have risk factors like hyperthyroidism, SLE, DM you should screen them for OSA

**You should do urine analysis

***Do duplex, CT or MRI, in textbooks they mention abdominal bruit but it is difficult to identify and not very specific

****Keep in mind in young women

Secondary HTN

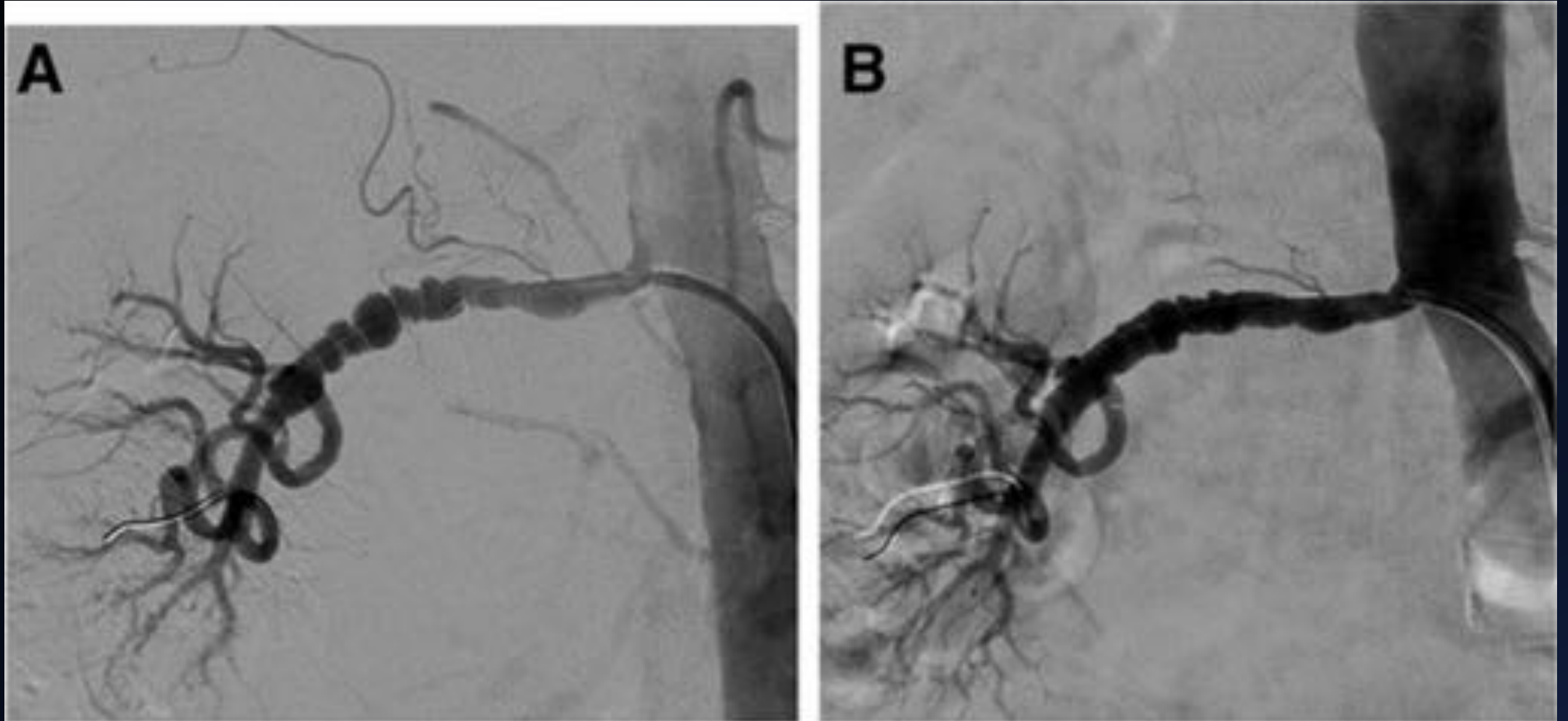
Table 1 Overview of the most common causes for secondary hypertension

Secondary cause	Prevalence ^a	Prevalence ^b	History	Screening	Clinical findings	Laboratory findings
Obstructive sleep apnoea *	>5–15%	>30%	Snoring, daytime sleepiness, morning headache, irritability	Screening questionnaire; polysomnography	↑ neck circumference; obesity; peripheral oedema	Not specific
Renal parenchymal disease**	1.6–8.0%	2–10%	Loss of good BP-control; diabetes; smoking; generalized atherosclerosis; previous renal failure; nocturia	Creatinine, ultrasound of the kidney	Peripheral oedema; pallor; loss of muscle mass	↑ Creatinine, proteinuria; ↓ Ca ²⁺ , ↑ K ⁺ , ↑ PO ₄
Renal artery stenosis ***	1.0–8.0%	2.5–20%	Generalized atherosclerosis; diabetes; smoking; recurrent flush pulmonary oedema	Duplex, or CT, or MRI, or angiography (drive by)	Abdominal bruits; peripheral vascular disease;	Secondary aldosteronism: ARR →; ↓ K ⁺ ; ↓ Na ⁺
Primary aldosteronism ****	1.4–10%	6–23%	Fatigue; constipation; polyuria, polydipsia	Aldosterone–renin ratio (ARR)	Muscle weakness	↓ K ⁺ ; ARR ↑
Thyroid disease ****	1–2%	1–3%	Hyperthyroidism; palpitations, weight loss, anxiety, heat intolerance; Hypothyroidism; weight gain, fatigue, constipation	TSH	Hyperthyroidism: tachycardia, AF; accentuated heart sounds; exophthalmus; Hypothyroidism: Bradycardia; muscle weakness; myxoedema	Hyperthyroidism: TSH ↓; FT4 and/or FT3 ↑; Hypothyroidism: TSH ↑; FT4 ↓; cholesterol ↑
Cushing's Syndrome	0.5%	<1.0%	Weight gain; impotence; fatigue; psychological changes; polydipsia and polyuria	24 h urinary cortisol; dexamethasone testing	Obesity, hirsutism, skin atrophy, Striae rubrae, muscle weakness, osteopenia	24 h urinary: cortisol ↑; Glucose ↑; Cholesterol ↑; K ⁺ ↓
Pheochromocytoma	0.2–0.5%	<1%	Headache; palpitations; flushing; anxiety	Plasma-metanephrines; 24 h urinary catecholamine	The 5 P's [†] : paroxysmal hypertension; pounding headache; perspiration; palpitations; pallor	metanephrines ↑
Coarctation of the aorta	<1%	<1%	Headache; nose bleeding; leg weakness or claudication	Cardiac ultrasound	Different BP (≥20/10 mmHg) between upper–lower extremities and/or between right–left arm; ↓ and delayed femoral pulsations; interscapular ejection murmur; rib notching on chest Rx	Not specific

Last 2 are not seen often but they can be stressful to the patient.

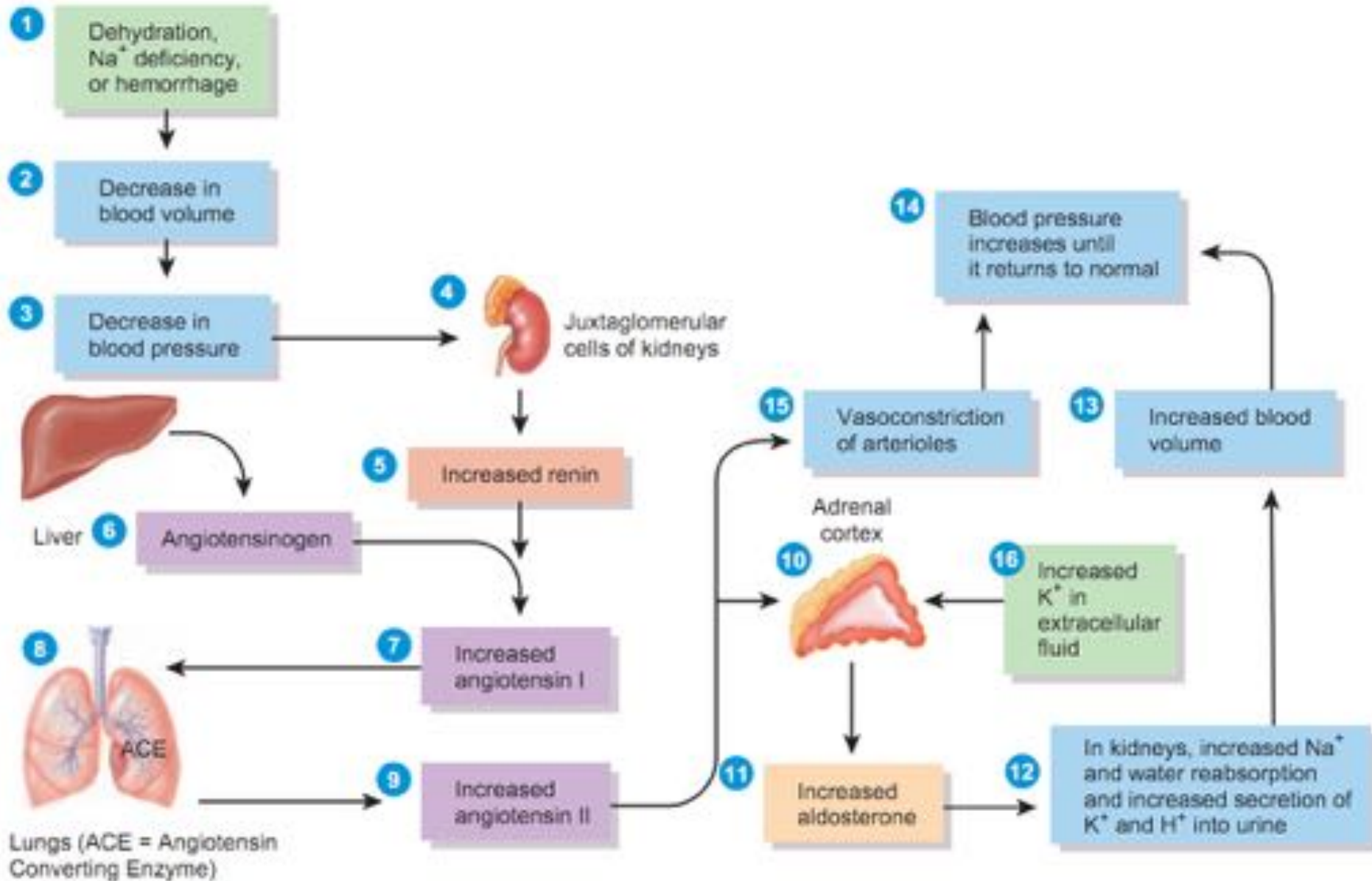
This is not her image

sign of fibromascular dysplasia



Regulation of aldosterone secretion by the renin–angiotensin–aldosterone (RAA) pathway.

Aldosterone helps regulate blood volume, blood pressure, and levels of Na^+ , K^+ , and H^+ in the blood.



Case 1

- 35 F, BP 149/98 mmHg, FHx father, brother. No significant PMHx
- Labs: CBC normal, Urea 4, Cr 56, Na 143, K 3.1, Cl 89, HCO₃ 30
- LFT Normal, TSH Normal
- US Kidneys: R kidney 11.2 cm, L kidney 12 cm, Dopplers Normal renal artery flow

some evidence of hypokalemia and alkalosis and because of her age she might have primary aldosteronism so order aldosterone ratio

ARR

Table 2. Conditions That May Affect the Aldosterone-Renin Ratio (ARR)^a

Condition	Effect on PAC	Effect on PRA	Overall Effect on the ARR
Hypokalemia	Decreased	May be increased	Decreased
Potassium loading	Increased	May be decreased	Increased
Sodium restriction	Increased	Increased	Increased
Sodium loading	Decreased	Decreased	Decreased
Advanced age	Decreased	Decreased	Decreased
Renal impairment	Unchanged	Decreased	Increased
Pregnancy	Increased	Increased	Decreased
Luteal phase of menstrual cycle	Increased	Unchanged	Increased

ARR

It is very hard to interpret because most of the patients who have HTN are already on treatment, and most hypertensive treatment can alter the reading of the test so it is not the best test but it can give you some idea.
so whatever the reading is whether its high or low you need to do some confirmatory tests.

Table. Impact of Medications on the Aldosterone/Renin Ratio (ARR)¹

False-positive ARR	False-negative ARR
β -Adrenergic blockers	Potassium-wasting or -sparing diuretics
Central α 2 agonists (eg, clonidine, α -methyldopa)	ACE inhibitors
NSAIDs	Angiotensin II type 1 receptor blockers
Renin inhibitors	Calcium blockers (eg, dihydropyridine)

NSAIDs, nonsteroidal anti-inflammatory drugs; ACE, angiotensin-converting enzyme.

ARR results should be interpreted in light of the patient's age, sample collection conditions (eg, time of day, posture and length of time in that posture, sodium and potassium status, and medications being taken), as well as the patient's clinical history.

PA Confirmatory tests

- Try to inhibit Aldosterone (demonstrate that it is regulated)
 - Oral salt loading (1 g po od x3days) *it wont be supressed in case of PA*
 - NS infusion (500 ml)
 - Captopril challenge
 - Fludrocortisone + Na

PA testing

- Imaging
- Venous sampling invasive so its not done anymore except in specialized centers

Case 1

- Results:
 - High Aldosterone
 - Low Renin
- CT Abdomen: No adenoma, normal adrenal glands
- 24-hour K excretion: high
- What to do next? start aldosterone antagonist, like spironolactone or epleronone
start aldosterone antagonist, like spironolactone or epleronone
difference b/w is in young men avoid spironolactone cuz of gynecomastia and ?affects androgens
both of them is potassium sparing

Case 2

- A 67-year-old man presents to the emergency department with headache. He has a history of uncontrolled hypertension as he is not compliant on medications.
- On physical examination: He is conscious, tired but oriented.
- BP is 230/120 mmHg, HR 89 bpm, O2 saturation is 95% on RA. CVS reveals S1+S2+S4, Chest is clear.
- Remaining exam is unremarkable.

abnormal findings: High BP, headache, S4

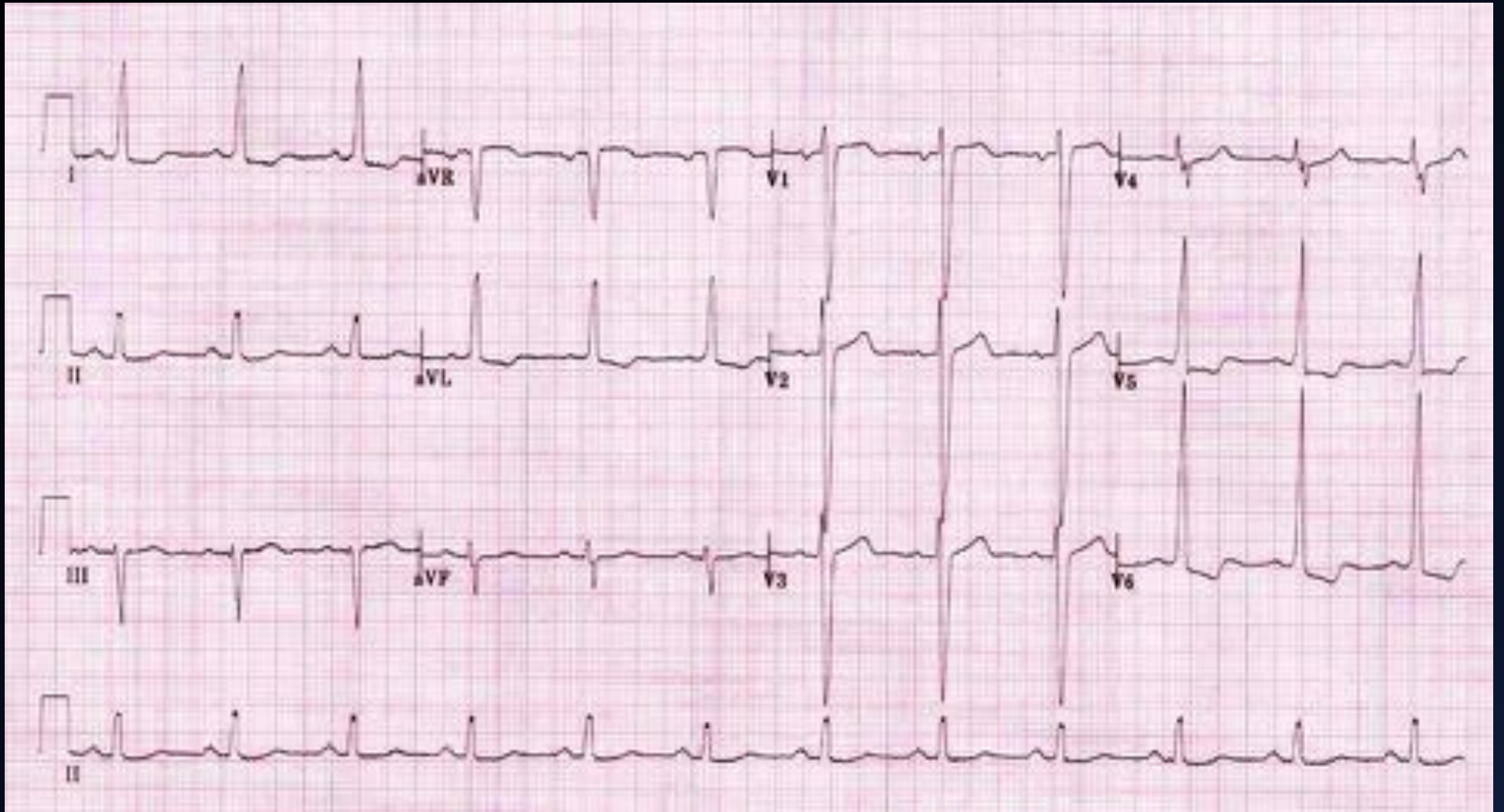
S4 indicates stiffness of ventricles

Case 2

- Which one of the following is the most appropriate next step in management?
 - A. ECG *ECG takes priority*
 - B. Fundoscopy
 - C. Urinalysis
 - D. CT Scan of the head *he has headache which is worrisome*
 - E. None of the above
 - F. All the above

Case 2 - ECG

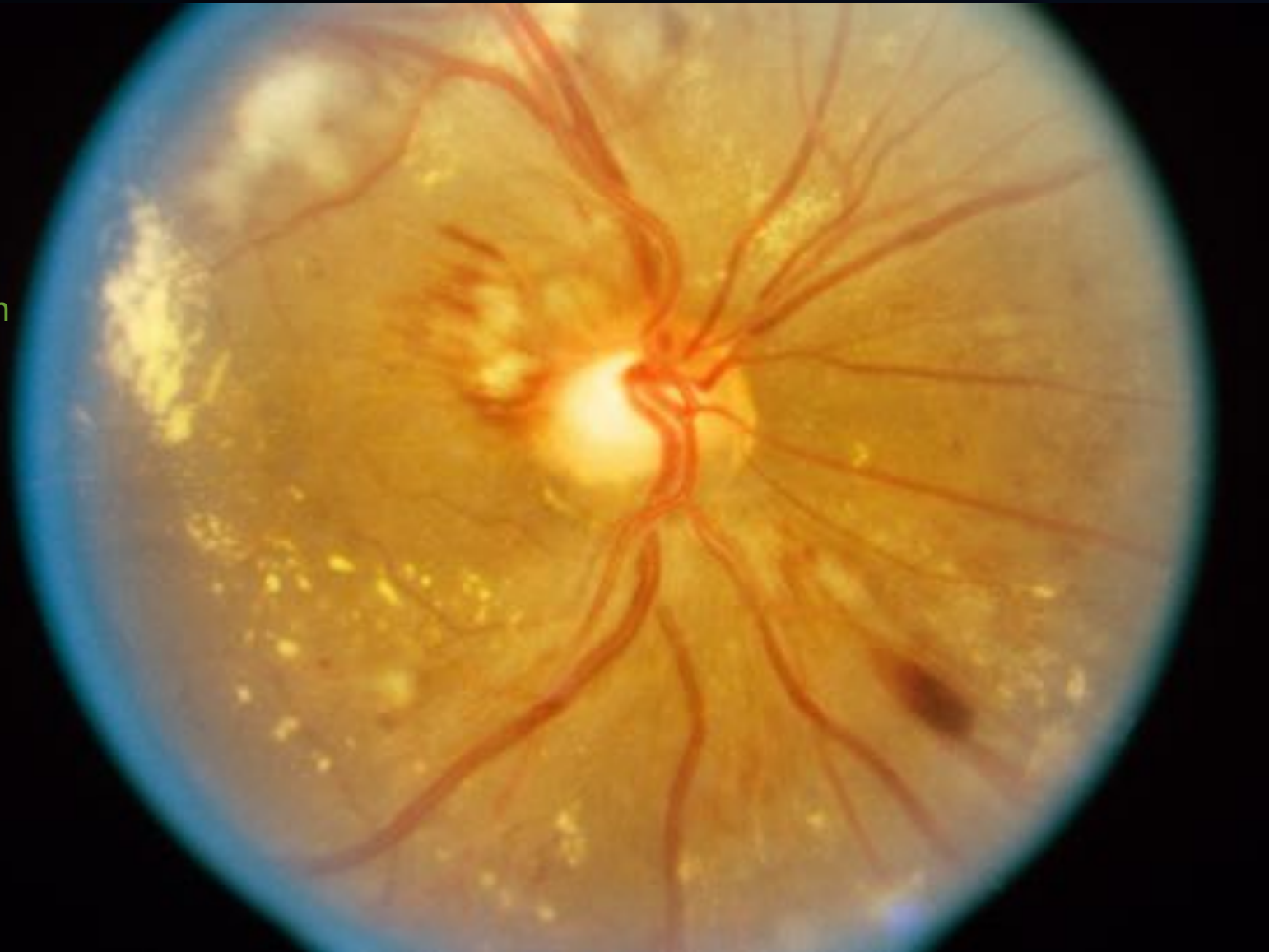
sinus rhythm, rate is around 60-70, there is left axis deviation (lead 2 and 3 or aVf)
prominent s wave in v2 and prominent r wave in v6 so it is left ventricular hypertrophy



Case 2 - Fundoscopy

Cotton wool spots, hemorrhage and optic edema and copper wire indicates hypertensive retinopathy.

malignant hypertension in previous terminology it is not used anymore.



Case 2 – CT Scan - Brain

This is a normal CT head,
there is no hemorrhage.

in cerebral hemorrhage u
would see shifting and
compression of surrounding
tissue

Blood is white in CT u will see
hyper-density in the
distribution of the arteries .



Hypertensive Crisis it can be classified into 2:

Explanation in the next slide

	Definition	BP values
Hypertensive Emergencies ¹	Severe and acute elevation of blood pressure associated to a new onset or worsening organ damage* ²	SBP>180 mmHg and/or DBP>120 mmHg
Hypertensive Urgencies	Elevation of blood pressure values without clinical, laboratory or instrumental evidence of acute hypertensive organ damage*	

³* Organ damage: hypertensive encephalopathy, acute stroke, intracranial hemorrhage, acute coronary syndromes, pulmonary edema, aortic dissection, acute or rapidly progressing renal failure, sympathetic crises (i.e. cocaine toxicity/pheochromocytoma), eclampsia or severe pre-eclampsia **and HTN retinopathy**

- 1- Hypertensive emergency you have to admit the patient for treatment under monitoring.
- 2- worsening organ damage like a patient who has history of MI or IHD and is stable but now he complains of severe chest pain this is worsening of his angina.
- 3- End-organ damage can be classified as changes in CNS or CVS, renal changes, eye disease.

Eclampsia and pre-eclampsia are important and often forgotten.

- perioperative HTN is considered as hypertensive emergency and you need to treat them so they do not bleed to death.

Which one of the following treatments is given for Hypertensive Emergencies

- A. Metoprolol IV infusion it affects the rate of the heart mostly not the blood pressure
- B. Labetalol IV infusion
- C. Nifedipine IV infusion There is no IV formulation of nifepidine

Drugs for hypertensive emergencies and urgencies. Explanation in the next 2 slides

Agent	Action	Dosage	Onset	Duration	Adverse Effects	Comments
Hypertensive Emergencies						
1 Nicardipine (Cardene)	Calcium channel blocker	5 mg/h intravenously; may increase by 1-2.5 mg/h every 15 minutes to 15 mg/h	1-5 minutes	3-6 hours	Hypotension, tachycardia, headache.	May precipitate myocardial ischemia.
2 Clevidipine (Cleviprex)	Calcium channel blocker	1-2 mg/h intravenously initially; double rate every 90 seconds until near goal, then by smaller amounts every 5-10 minutes to a maximum of 32 mg/h	2-4 minutes	5-15 minutes	Headache, nausea, vomiting.	Lipid emulsion; contraindicated in patients with allergy to soy or egg.
3 Labetalol (Trandate)	Beta- and alpha-blocker	20-40 mg intravenously every 10 minutes to 300 mg; 2 mg/min infusion	5-10 minutes	1-6 hours	GI, hypotension, bronchospasm, bradycardia, heart block.	Avoid in acute LV systolic dysfunction, asthma. May be continued orally.
4 Esmolol (Brevibloc)	Beta-blocker	Loading dose 500 mcg/kg intravenously over 1 minute; maintenance, 25-200 mcg/kg/min	1-2 minutes	10-30 minutes	Bradycardia, nausea.	Avoid in acute LV systolic dysfunction, asthma. Weak antihypertensive.
5 Fenoldopam (Corlopam)	Dopamine receptor agonist	0.1-1.6 mcg/kg/min intravenously	4-5 minutes	< 10 minutes	Reflex tachycardia, hypotension, increased intraocular pressure.	May protect kidney function.
6 Enalaprilat (Vasotec)	ACE inhibitor	1.25 mg intravenously every 6 hours	15 minutes	6 hours or more	Excessive hypotension.	Additive with diuretics; may be continued orally.
7 Furosemide (Lasix)	Diuretic	10-80 mg orally	15 minutes	4 hours	Hypokalemia, hypotension.	Adjunct to vasodilator.
8 Hydralazine (Apresoline)	Vasodilator	5-20 mg intravenously; may repeat after 20 minutes	10-30 minutes	2-6 hours	Tachycardia, headache, GI.	Avoid in coronary artery disease, dissection. Rarely used except in pregnancy.
9 Nitroglycerin	Vasodilator	0.25-5 mcg/kg/min intravenously	2-5 minutes	3-5 minutes	Headache, nausea, hypotension, bradycardia.	Tolerance may develop. Useful primarily with myocardial ischemia.
10 Nitroprusside (Nitroprussil)	Vasodilator	0.25-10 mcg/kg/min intravenously	Seconds	3-5 minutes	GI, CNS; thiocyanate and cyanide toxicity, especially with renal and hepatic insufficiency; hypotension. Coronary steal, decreased cerebral blood flow, increased intracranial pressure.	No longer the first-line agent.
Hypertensive Urgencies						
11 Clonidine (Catapres)	Central sympatholytic	0.1-0.2 mg orally initially; then 0.1 mg every hour to 0.8 mg orally	30-60 minutes	6-8 hours	Sedation.	Rebound may occur.
12 Captopril (Capoten)	ACE inhibitor	12.5-25 mg orally	15-30 minutes	4-6 hours	Excessive hypotension.	
13 Nifedipine (Adalat, Procardia)	Calcium channel blocker	10 mg orally initially; may be repeated after 30 minutes	15 minutes	2-6 hours	Excessive hypotension, tachycardia, headache, angina, myocardial infarction, stroke.	Response unpredictable.

ACE, angiotensin-converting enzyme; CNS, central nervous system; GI, gastrointestinal; LV, left ventricular.

Source : Current Medical Diagnosis and Treatment 2018

Hypertensive emergency treatment:

- 1- do not confuse nicardapine with nifepidine, they are both CCB but we do not use nifepidine for hypertensive emergency because it only comes in an oral formulation.
- 3- The one we use the most is labetalol, it is widely available, the only down side is that it causes bradycardia, so if that happens switch to another agent.
- 4- Esmolol has a significant effect on the rhythm so avoid it in heart failure.
- 7- Furosemide is not used for HTN emergency, it is only used for special cases, like heart failure with HTN emergency.
- 8- Hydralazine is not often used because it is associated with tachycardia which can precipitate coronary artery disease.
- 9- Nitroglycerin can be used but you might have tolerance after 24 hrs.
- 10- Nitroprusside is associated with cyanide toxicity.

In summary:

- Labetalol is the first choice.
 - if nicardipine is available use it.
 - In case of heart failure use furosemide + labetalol.
- In hypertensive emergency you have to decrease the BP by 25 mmHg within 1 hour because of end organ damage, you start with 1 medication if the BP does not reach the goal within an hour then you increase the dose until the maximum, if still it is not improved you can add another agent.

Hypertensive urgency treatment:

- 13- CCB is the first choice of treatment because of lower risk of side effects.
 - 12- Captopril is used because it has a short half-life so it can build up in the system fast and it can be used every 8-12 hrs, but it can precipitate renal function.
 - 11- Clonidine is listed in the books but we don't use it because it has central activity, some people use it for anxiety related HTN.
- You have to decrease the BP gradually, because the organs are used to the high BP, so if you lower the BP fast it may lead to renal ischemia or cerebral ischemia

Summary

*do ECG, urine analysis, fundoscopy, echo, no need for CT head if they are asymptomatic.

**medications that cause HTN: NSAIDs very commonly, and OCP among young women. and also pseudoephedrine, cocaine, caffeine.
Most common and most important is NSAIDs and OCP.

***Cushing and hyperthyroidism

General clinical clues

- Age, Habitus comorbidities
- Resistant/severe hypertension
- Target organ damage *



24h ABPM

- Exclude white coat hypertension
- Exclude pseudo-resistance
- Dipping status, heart rate



Exclude other factors

- Exclude drug-related hypertension **
- Confirm therapy adherence



Screening for secondary forms

- Obstructive sleep apnoea
- Renal parenchymal/vascular disease
- Primary aldosteronism
- Other endocrine causes ***
- Aortic coarctatio

Summary Important slide

General clinical characteristics suggestive of secondary hypertension when to screen:

Early onset of hypertension (i.e. <30 years) in patients without other risk factors (i.e. family history, obesity, etc.); increased BP in prepubertal children

* Resistant hypertension (>140/90 mmHg despite three antihypertensive drugs including a diuretic)

Severe hypertension (>180/110 mmHg) or hypertensive emergencies

Sudden increase of BP in a previously stable patient

Non-dipping or reverse dipping during 24 h ambulatory BP monitoring

Presence of target organ damage (i.e. LVH, hypertensive retinopathy, etc.)

* Renal artery stenosis is often associated with resistant hypertension.