



Hypertension

● Objectives:

- To be able to recognize the definition of hypertension
- To be able to identify the Stages of Hypertension
- To find out the complication of Hypertension
- To learn how to measure blood pressure
- To acquire knowledge on how to treat hypertension

[Color index : **Important** | **Notes** | Extra]

● Resources:

- 435 slides (The doctor said that the slides are more than enough)
- For further reading : Kumar & Clark's clinical medicine (Page 1046) or Davidson (Page 607).

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"Medicine is an art, nobody can deny it."

Hypertension:

- High blood pressure is only one of several cardiovascular risk factors that require attention
- Optimal blood pressure below 120/80 Reason for considering 120/80 as the optimal BP because complications like stroke, IHD and Renal failure start when the systolic blood pressure exceeds 115
- **BP greater than 140/90 in general population.**
- **BP greater than 130/80 in diabetics and those with renal disease.**

European Society of Nephrology Classification of Blood Pressure Levels		
Category	Systolic blood pressure (mmHg)	Diastolic blood pressure (mmHg)
Optimal blood pressure	<120	<80
Normal blood pressure	<130	<85
High-normal blood pressure	130-139	85-89
Grade 1 hypertension (mild)	140-159	90-99
Grade 2 hypertension (moderate)	160-179	100-109
Grade 3 hypertension (severe)	>/= 180	>/= 110
Isolated systolic hypertension	>140	<90

Epidemiology:

- Worldwide high BP affects **more than 40%** of adults **older than** the age of **25** years
- **Onset ranges between 25 to 55 years, mainly in 40 to 50**
- Global BP control remains at 32.5%. **The old population has high prevalence of HTN**
- The **fourth** most common **cause of death worldwide.**
- Directly and indirectly responsible for >20% of all deaths.
- 29-30% (about 66 million, 1 out of every 3) incidence of all over world.
- Occurs over 30% of people older than 65.
- The overall **prevalence** of hypertension in **Saudi Arabia** was **25.5%**. The Risk is increased among men, and with age, obesity, diabetes, and hypercholesterolemia.¹ **Awareness is limited that is why you depend on screening**

Prevalence of Hypertension:

Looking into gender, age and BMI, NHANES III suggests that the high prevalence of HTN is currently driven **by two phenomena**

- **The increased age of population**
- **The growing prevalence of obesity**

As well as **High dietary salt intake**

¹ A national survey performed in Saudi Arabia 2011 over 4758 adult participants showed that only 44.7% of HTN patients were aware of it and 32% of HTN receiving pharmacotherapy. Unfortunately, only 12% were controlled.

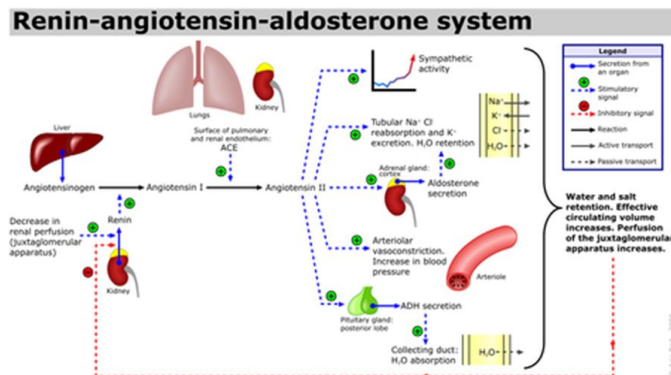
Pathophysiology of hypertension

Blood pressure equation:

BP= cardiac output x systemic vascular resistance = (stroke volume x heart rate) x systemic vascular resistance
HTN happens due to ↑ cardiac output and/ or ↑ systemic vascular resistance

The main mechanisms:

- An overactive **Renin angiotensin system** leads to **vasoconstriction** and **retention of sodium and water** → increase in blood volume → **hypertension**.
- An overactive **sympathetic nervous system** → **increased stress responses**. Some people have over-activated sympathetic system which can be due to stress or environmental and genetic factors.
- **Blunting of pressure-natriuresis**². It is supposed that the ingested salt is excreted within 24 hours by the kidneys (normally); high salt intake increases plasma volume. In this case (blunting) they retain salt and cannot excrete it -due to unknown cause- although their kidney function is normal .
- **Variation of cardiovascular development** When the cardiovascular of some people is not well developed, they become more sensitive to high salt or sympathetic activity.
- **Variation of renal development**. Normally The kidney contains 800,000 to 1,200,000 nephrons.
 - 1) People who are born during starvation generally have less nephrons, so they can't excrete salt and water properly → they get retention and end up with HTN.
 - 2) When people get obese the kidney becomes smaller when compared to the body and can't tolerate salt increase.
- **Elevated intracellular Na or Ca** . For the action potential to occur in any muscle Na, followed by Ca, are needed inside the cell while K is outside the cell. In this case they have more Na inside the muscles → contract the vessels (vasoconstricted) → high BP



Liver produces angiotensinogen when the kidney produces renin, especially in ischemia, lower perfusion or even when BP drops a little. Renin will react with angiotensinogen to form angiotensin I which is converted to angiotensin II by the angiotensin converting enzyme (ACE).

Angiotensin II is a very strong vasoconstrictor . It produces hypertension, works on aldosterone and leads to salt and water retention in the kidney. Patients with HTN may have an overactive renin angiotensin system when compared to other people.

² The **pressure-natriuresis** mechanism, whereby increases in renal perfusion **pressure** lead to decreases in sodium reabsorption and increases in sodium excretion.

Types of hypertension:

1. Essential Hypertension: The exact mechanism is unknown

In **90%-95%** of cases, but no cause can be found (**primary** hypertension). More common in black people. It results from **environmental** and **genetic** factors (more than 50 genes)

- Risk factors:
 - Obesity, metabolic syndrome, DM
 - Excessive salt intake, low potassium intake. ↑ plasma volume .
 - Excessive alcohol intake. ↑ sympathetic activity
 - Polycythemia. ↑ RBCs → ↑ blood volume
 - Lack of exercise. Exercise leads to vasodilatation of blood vessels which help in lowering BP
 - Family history of essential HTN. **The main risk factor**
 - **Vitamin D deficiency.** They have more renin-angiotensin activity. Vit D helps to lower BP, so when vit D levels are corrected there will be decreased activation of RAAS which in turn improves BP.
 - Aging .Blood vessels get stiff (lose compliance) with age. So, they can't dilate → HTN
 - Smoking; increases risk of complication. It increases BP temporarily for hours. However it increases the risk of complication for example the risk of stroke increase in smokers three times
 - Caffeine increases the BP temporarily acutely but is not a risk factor for the development of chronic essential HTN.

2. Secondary hypertension:

- Account for **5 to 10 %**
- Expected if :
 - **Onset of high blood pressure before age 30 or after age 55** In pts <40 you always have to rule out 2ry HTN
 - **Severe or resistant** hypertension
- The Possible etiologies are :
 - **Primary renal disease.** In any kidney disease or injury:
 1. Due to loss of glomeruli → they can't excrete salt and water →HTN
 2. Increase in sympathetic → ↑ in BP.
 3. Increase production of renin → ↑ BP
 - **Oral contraceptives.** In female after age of 35 if they use OCP more than two years → ↑ angiotensinogen production from liver. They need 6 months at least to get normal BP after stopping the contraceptive
 - **Sleep apnea syndrome.** Around 30% . In any patient presents with HTN you have to rule out sleep apnea. The mechanism is that they have snoring; suddenly they get desaturation → relax muscles in the neck usually it happened in obese people and this muscles lead to obstruction of respiratory system → they wake up from night. This can happen for many times in night. If not treated properly they can get: Car accident (daytime sleeping) or Arrhythmias
 - **Primary hyperaldosteronism.**
 - ↑ Aldosterone lead to HTN mainly because it helps in reabsorbing salt and excreting K. Primary hyperaldosteronism is known as Conn's syndrome.
 - **Renovascular disease.** Renal artery stenosis
 - **Cushing syndrome.** Due to ↑ in corticosteroid. It lead to HTN and hyperglycemia patients present with round face, trunk obesity and stria.
 - **Pheochromocytoma.** Pheochromocytoma patients will have sudden headache, palpitation, sweating and get high BP stays for mins then the BP get back to normal but they will be tired. In investigation you will find high catecholamine.

- Other endocrine disorders **hyperparathyroidism, hyperthyroidism and hypothyroidism.**
- Coarctation of the aorta. **Aortic arch narrowing**
- **Medications:** NSAID **Na and water retention**, Cyclosporin **Vasoconstriction**, decongestant **Vasoconstriction** , erythropoiesis-stimulating agent

Stages Of Hypertension

Stages:³⁴

Diagnosis	systolic	diastolic
Normal	<120	<80
Prehypertension	120-139	80-89
Stage 1 HTN (mild) ⁵	140-159	90-99
Stage 2 HTN (moderate)	> = 160	> = 100
Hypertensive emergency	> 180	> 120

Hypertensive Emergency: It can occur due to improper treatment or pt stopping treatment for 3 to 6 months

Severe hypertension that develops rapidly (**Diastolic** blood pressure **above 120 mmHg**), that results in end organ damage (MI,Stroke,AKI,CHF). Both systolic and diastolic are increased. However, we can't depend on systolic in old patients since it increases with age.

It can be either:

- **Malignant (accelerated) hypertension**⁶ Marked hypertension with encephalopathy and retinal hemorrhages, exudates, and/or papilledema The terms “malignant” and “accelerated” hypertension are difficult to distinguish clinically, with “malignant” usually referring to the more severe syndrome.

White Coat Hypertension:

- Approximately 20 to 25% of patients with mild office hypertension
- More common in elderly
- The diagnosis of mild hypertension should not be made until the blood pressure has been measured on **at least three to six visits.** Especially first and second stages when you can't diagnose it at the first visit.

³ Generally speaking, young people have high diastolic while old people have low diastolic and high systolic.

⁴An increase in systolic or diastolic indicates the presence of HTN.

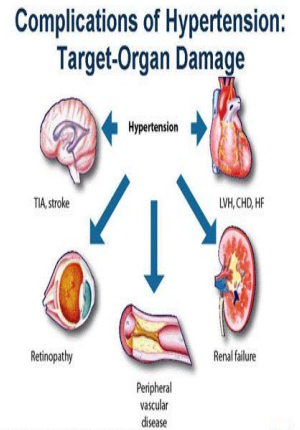
⁵ High normal because 50% will develop HTN within 5 years and if they have other risk factors like renal disease, DM, IHD or stroke you need to treat them early before they get into grade one

⁶ This rare condition may complicate hypertension of any aetiology and is characterised by accelerated microvascular damage with necrosis in the walls of small arteries and arterioles ('fibrinoid necrosis') and by intravascular thrombosis. The diagnosis is based on evidence of high BP and rapidly progressive end organ damage, such as retinopathy (grade 3 or 4), renal dysfunction (especially proteinuria) and/or hypertensive encephalopathy (see above). Left ventricular failure may occur and, if this is untreated, death occurs within months (Davidson).

Complication of Hypertension

- **Without treatment, high BP can lead to:**

- Stroke **60% of stroke are due to HTN (mainly thrombotic)**, Ischemia, Alzheimer's Disease, **retinal hemorrhage**
- CAD, arrhythmia **especially atrial fibrillation**, sudden death.
- Congestive heart failure, left ventricular hypertrophy
- **Blood flow become limited → Ischemia and necrosis → Can be source of arrhythmia and when the heart becomes stiff it can lead to CHF**
- Aortic dissection.
- Renal disease. **More than 50% of black people in renal dialysis are due to renal failure**
- Peripheral vascular disease.
- Hypertensive Emergency And Increase Emergency Morbidity



- These diseases account for significant disability, loss of productivity, and decreased quality of life .

- **Hypertensive retinopathy can be graded as the following:**

Grade	Description	Picture
I	Minimal narrowing of retinal arteries. Generalized arteriolar constriction-seen as `silver wiring ⁷ `, Copper wiring⁸ and Vascular tortuosities	
II	Narrowing of retinal arteries in conjunction with regions of focal narrowing and arterio-venous nicking ⁹ . (yellow arrow) Arteriovenous nicking	
III	Abnormalities seen in Grade 1 and II, as well as retinal hemorrhages, hard exudates and cotton wool spots . (yellow arrow) Flame-shaped hemorrhage	
IV	Papilledema from malignant hypertension Abnormalities encountered in Grades I through III, as well as swelling of the optic nerve head and macular star ¹⁰ . There is blurring of the borders of the optic disk with hemorrhages (yellow arrows) and exudates (white arrow)	

⁷ Retinal arterioles look white if they have become occluded

⁸ Retinal arterioles appear orange or yellow instead of red

⁹ **AV**, or **arteriovenous nicking** (also known as **arteriovenous nipping** in the UK) is the phenomenon where, on examination of the eye, a small artery (arteriole) is seen crossing a small vein (venule), which results in the compression of the vein with bulging on either side of the crossing.

¹⁰ The lipid-rich component of the exudate is further able to penetrate into the outer plexiform layer, creating what is clinically seen as a macular star pattern.

Clinical presentation:

- Asymptomatic
- Headache *It could be on any side but it is mainly occipital. In any event you depend on the screening.*
- Epistaxis
- Chest discomfort
- Symptoms of complications *Stroke, renal failure or CHF*

Screening :

- Every two years for people with systolic and diastolic pressure below 120 mmHg and 80 mmHg .
- Yearly for persons with **high risk** OR a **SBP of 130 to 139** mmHg OR **DBP of 85-89** mmHg .

Physical examination :

- Confirm the diagnosis of of hypertension.
- Look for secondary causes (may be treatable) .
- Assess damage to target organ (heart *4th heart sound*, kidneys *renal bruit*, Eyes *retinal changes*, CNS *↓ cognition*).
- Assess overall cardiovascular risk.
- Concomitant clinical conditions .

Methods of diagnosing hypertension:

Device			To Dx HTN		Notes
			Systolic	Dystolic	
Office BP	Non-automated device [non-AOBP]	Mercury Type	≥140	≥90	It will become obsolete
		Aneroid Type			
		Half automated device			
	Automated Device	Digital Type			The commonest one
Ambulatory BP		Daytime	≥135	≥85	For 24 hours during daytime as well as in nighttime. Then it will calculate the mean. <i>It will help to know the BP during sleep because if it does not drop the patient might get stroke. Daytime = awake / nighttime =sleep</i>
		Nighttime	≥120	≥70	
		Mean (24 h)	≥ 130	≥80	
Home BP Monitoring (HBPM)		Arm	≥135	≥85	Among the home devices it is the best
		Wrist			not recommend unless for obese people
		Finger			Not accurate but might help on emergency
AOBP (Automated office blood pressure) ¹¹			≥135	≥85	Recommended method. The BP is taking when the patient is comfortable for 5 times each reading for 2 mins and it measure the mean after drop the first reading.

¹¹ AOBP is taken for 5 times, regular automated office measuring is done only once

Diagnosis :

1) BP measurement :

- 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 a span of 4 or more weeks.***

- Observe the following to obtain an accurate BP reading :

1. The Back should be straight and the arm should be at heart level (whatever the position of the patient), and the patient should be seated comfortably.
2. Have the patient sit quietly for at least 5 minutes before measuring BP.
3. Make sure the patient has not ingested caffeine or smoked cigarettes in the past 30 minutes.
4. Use a cuff of adequate size. The standard bladder is 12 to 13 cm wide and 35 cm long. For large arm you use large bladder which its circumference is more than 32 cm (a cuff that is too small can falsely elevate BP readings). The bladder within the cuff should encircle at least 80% of the upper arm.
5. Use phase one (disappearance) **Korotkoff sounds¹²** to identify systolic BP. And phase four Korotkoff sounds to identify diastolic BP.
6. Take at least two BP measurements, spaced 1–2 min apart, and additional measurements if the first two are quite different. Consider the average BP if deemed appropriate.
7. Measure BP in both arms at first visit to detect possible differences. In this instance, take the arm with the higher value as the reference.

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

1. Urinalysis **To look for hematuria and proteinuria**, (if patient is diabetic, check for microalbuminuria)
2. Serum sodium (**In case of hyperaldosteronism the Na will be high**), serum potassium (**In hyperaldosteronism or Cushing syndrome K will be low**), creatinine, or the corresponding estimated GFR, calcium (**hyperparathyroidism leads to hypercalcemia**) and uric acid (**It can cause HTN if it increases**).
3. Fasting glucose and hematocrit **since that polycythemia is one of the risk factor**
4. Lipid profile, after 9- to 12-hour fast, that includes high density and low-density lipoprotein cholesterol, and triglycerides
5. ECG
6. Optional tests: Measurement of urinary albumin excretion or albumin/creatinine ratio
7. More extensive testing for identifiable causes is not generally indicated unless BP control is not achieved

3) If the history and physical examination or laboratory test suggest a secondary cause of HTN, order appropriate test.

4) Before starting treatment for hypertension, it is useful to evaluate the patient more thoroughly :

Risk factors by using **Framingham Risk Score¹³**

Asymptomatic organ damage ¹⁴

¹² Korotkoff sounds are blood flow sounds that healthcare providers observe while taking blood pressure with a sphygmomanometer over the brachial artery in the antecubital fossa. These sounds appear and disappear as the blood pressure cuff is **inflated** and deflated.

¹³ include age, male gender, smoking, dyslipidemia, glucose intolerance, obesity and family history of premature CVD.

¹⁴ mainly involves left ventricular hypertrophy, evidence of vascular damage and microalbuminuria; CKD; CVD, DM

Management :

Lifestyle intervention	Pharmacological therapy
- High normal (SBP >130–139 mmHg, DBP 85–89 mmHg) -In high risk patients	If blood pressure is 140/90 mmHG
Diet : high consumption of vegetables and fruits ¹⁵ , low-fat diet, Vit D replacement	Thiazide diuretics
Regular physical exercise : 30min of moderate-intensity aerobic exercise 5-7 days/week	B-Blocker
Reduction of alcohol intake	ACE inhibitors
Reduction of dietary sodium intake: (5-6g/day) and use of low sodium salt	Angiotensin II receptor blockers (ARBs)
Smoking cessation	Calcium channel blockers
Weight reduction : BMI 25 kg/m	Vasodilators

Drugs	Indication	Side effects	Contraindication	Notes
Thiazide diuretics	- A good option in patients with osteoporosis - Secondary stroke prevention	- Hypokalemia - Hyerglycemia - Hyperuricemia	- Gout - Pregnancy	A good initial choice for African-American patients
B-blockers	1- A good option in patients with CHF,CAD or atrial fibrillation	- Bradycardia -bronchospasm -insomnia -fatigue	1-Asthma 2-COPD	1-Decrease HR and cardiac output and decrease renin release 2-B-Blocker safe in pregnancy
ACE inhibitors	1-preferred in all diabetic patients 2-A good option for patients with CHF,CAD	- Hyperkalemia - Cough -Acute renal failure	1-pregnacy 2-renovascular disease	—
Angiotensin II receptors blocker (ARBs)	ARBs have the same beneficial effects on the kidney in diabetic patients as ACE inhibitors	Hyperkalemia	Pregnancy	1- If the patient has cough we can alternate ACEI to ARBs 2-ACE inhibitors and ARBs should not be used in combination

¹⁵ As 7 to 8 servings per day of grain or grain products, 4 to 5 vegetable and 4 to 5 fruit

Calcium channel blockers	—	- Edema - Bradycardia - Tachycardia - Constipation	—	—
alpha -Blocker	1- If the patient has benign prostatic hyperplasia (BPH)	- 1st dose hypotension - headache - dizziness	Urinary incontinence	Not consider first - second-line agents
Vasodilators (hydralazine and minoxidil)	Combination with B-blocker and diuretics to patients with refractory HTN	- Tachycardia - Edema		Not commonly used

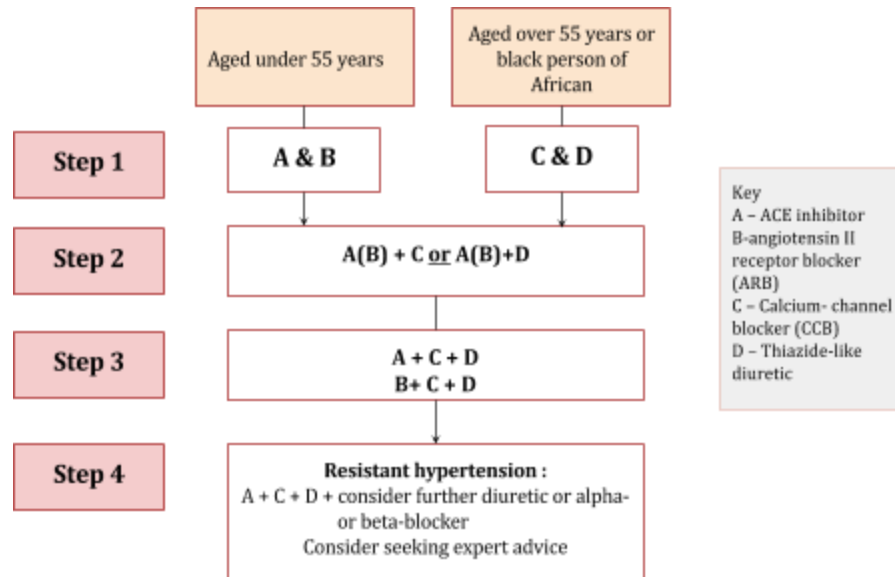
- Note : drugs with central sympatholytic action could cause drowsiness

Who should be treated ?

<u>Low risk group</u>	<u>High risk group</u>
<ul style="list-style-type: none"> • If the visit 1 mean office systolic BP is 180mm Hg and/or Diastolic BP is 110mm Hg then hypertension is diagnosed. • At visit 2, mean office BP measurement is 140mm Hg systolic and/or 90mm Hg diastolic in patients with macrovascular target organ damage , diabetes mellitus , or chronic kidney disease (glomerular filtration rate < 60ml/min/1.73 m²). • At visit 3, mean office BP measurement is 160mm Hg systolic or 100 mm Hg diastolic . • At visit 4-5, mean office BP measurement is 140mm Hg systolic or 90mm Hg diastolic without risk factor. • Treat all cardiovascular risk factors. 	<ul style="list-style-type: none"> • Start Lifestyle change in pre-hypertension (130 – 139)/(85 – 89) mmHg • Drug therapy (If BP is 130/80 mmHg) • Congestive heart failure – Thiazide, ACE-1, Aldosterone antagonist , BB • Post Myocardial Infarction – BB, ACEi • Diabetes Mellitus – ACEi, ARBs, Thiazide, CCB • Chronic kidney disease – ACEi, ARBs, Thiazide • Stroke – CCB +ACEi

Drug therapy :

- A low dose of initial drug should be used, slowly titrating upward.
- Optimal formulation should provide 24-hour efficacy with once-daily dose.
- Combination therapies may provide additional efficacy with fewer adverse effects. For example:
 - ★ ACE inhibitors and diuretics.
 - ★ Calcium antagonists and ACE inhibitors (*more effective*).
 - ★ Angiotensin II receptor antagonists and diuretics



Nonpharmacological treatment:

- Hypertension Renal Denervation **Used for resistant HTN but results aren't good and still under study**
- An implantable device designed to activate baroreceptors to reduce blood pressure does not appear to reduce blood pressure. **Unfortunately, it does not succeed and it leads to thrombosis in the arteries. The theory now is to let it outside and get the stimulation**

Benefits of Lowering BP:

- Stroke incidence will reduce 35 to 40%
- Myocardial infraction will reduce 20 to 25%
- Heart failure will reduce 50%
- Renal failure will reduce 35 to 50%
- 2 mmHg decrease in mean systolic BP will lead to
 - 7% reduction in risk of ischemic heart disease mortality
 - 10% reduction in risk of stroke mortality

Follow-up And Monitoring :

- Patients should return for follow-up after 2- 4 weeks and adjustment of medications until the BP goal is reached.
- More frequent visits for stage 2 HTN or with complicating comorbid conditions.
- Serum potassium and creatinine monitored 1-2 times per year.

Cases

1) A 25-year-old male came complaining from headache. BP in several visits 150\105 mmhg. Which one of the following is the next most appropriate diagnostic step?

- a. Ambulatory BP monitor
- b. Ultrasound of the kidney
- c. BUN and Creatinine and electrolytes
- d. Liver function test

2) Which one is considered as secondary hypertension ?

- A. Cardiomyopathy
- B. Behcet disease
- C. Heavy smoking
- D. Conn's syndrome

3) A 53 male known to be hypertensive and he is taking 10 mg of ramipril for several months. Despite this, his BP is suboptimal. There is no compliance issue. Which one would be the best add-on therapy?

- a. ARBs
- b. Thiazide diuretic
- c. Beta-Blocker
- d. Ca-channel-blocker

4) A 48-year-old woman was diagnosed with essential hypertension and was commenced on treatment three months ago. She presented to you with a dry cough which has not been getting better despite taking anti-tussive and antibiotics. You assess the patient's medication history.

Which of the following antihypertensive medications is responsible for the patient's symptoms ?

- a. Lisinopril
- b. Atenolol
- c. Furosemide
- d. Amlodipine

5) A 34 year old man comes to your clinic with history of headache and dizziness for 2 months. His examination is unremarkable apart from repeated BP measurements of 200/100 mmHg. What is the most appropriate next step:

- a. Recheck his BP again in 1 month.
- b. starts low salt diet only.
- c. starts antihypertensive medications.
- d. order CT scan of his head.

6) A 55 years old male presented to outpatient clinic with history of fatigue found to have BP of 155/90 , no previous history of hypertension, no diabetes mellitus, with normal laboratory test.-What is the best next step for the patient?

- a. Start Anti hypertension with beta blockers.
- b. Repeat the blood pressure measurements after 3 days.
- c. Give out patients a follow up after 3 months.
- d. Reassure the patient.

7) A 77 women found to have elevated BP with typical reading of 162/84 mm Hg. Which one would be most appropriate?

- a. ACEI
- b. ARBs
- c. Thiazide diuretic
- d. Beta-Blocker

1	2	3	4	5	6	7
c	d	d	a	c	b	c