Cardiovascular Sy	vstem-//-//- Sheet 1
Subject   Pharmacology	
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<u>\*\* please refer to the slides , I didn't mention what is written in the</u> <u>slides</u>

- Normal blood pressure → 120/80 (120→ systolic → when the heart contracted, 80→diastolic → when the heart relaxed)
- Hypertension → elevated the blood pressure above 140/90
  → It's the prehypertension state
- Two main contributing factors in the BP : cardiac output(CO) & peripheral vascular resistance (PVR)
- Increase the blood volume → will increase the pressure on the wall of blood vessels
- Increase the contraction of the smooth muscle of the blood vessels→ leading to narrowing the blood vessels → increase PVR→ increase BP
- Usually, it is asymptomatic disease → having the elevated BP but there is no symptoms → its bad condition → its called silent killer → because it has severe complications → could cause damage of certain organs:
  - 1- **Heart** → heart failure , hypertrophy, MI
  - 2- Blood vessels → change their structure ( thickness of their wall , hypertrophy ) → then there will be decrease the blood supply

to the orangs due to the narrowing in the blood vessels. Also, can affect the blood vessels in the brain  $\rightarrow$  causing stroke

- 3- Kidney  $\rightarrow$  can be damaged by having  $\rightarrow$  chronic kidney failure
- So, we <u>need to diagnose the hypertension early</u> to prevent these complications
- If these problems happen, they will persist along time
- to diagnose a patient with hypertension →we need the average of 14 reading during 7 days (twice a day, one in the morning and the other at night) → why we take the average reading ? because we have variation : during stress and excitement (over activation of sympathetic system) there will be high epinephrine(adrenaline) & norepinephrine → causing constriction of the heart → increase PVR → increase heart rate. According to these differences, at morning the BP will high BUT in the night will be low (circadian variation of blood pressure)
- multifactorial abnormalities can lead to high BP:
  - **1- genetics** (family history)
  - 2- stress (over activation of sympathetic)
  - 3- environment and diet( smoking → nicotine will bind to nicotinic receptors in the synapse ganglia in the neuromuscular junction ( of sympathetic and parasympathetic)→ the tone that is innervated the blood vessels → the sympathetic→ lead to constriction of the blood vessels → increase BP),,, salts → high Na+→ high water → high blood volume→ high pressure in the blood vessels → high venous blood return to the heart → high preload(increase the heart filling)→ high BP
- <u>secondary hypertension</u>→ a disease that will cause hypertension such as tumor in the adrenal gland
- BP variations:

**1-white coat**  $\rightarrow$  normal BP in the house when it is measured but high in the clinic  $\rightarrow$  due to stress

2-masked hypertension → people have hypertension but the don't know OR in the clinical have normal BP but in the house abnormal BP

- Women <u>before menopause</u> have low chance to develop hypertension than men → due to <u>estrogen</u> has vascular protective function BUT after menopause women and men have the same chance to develop hypertension
- Mortality is related to BP→ more associated with systolic pressure→ common in elderly BUT the diastolic hypertension, it is said to be common in the patient below 40
- Factors that can lead to increase the coronary heart diseases:
  - **1- Hypertension**
  - 2- High cholesterol
  - 3- High glucose
  - 4- Smoking
  - 5- Left ventricular hypertrophy (LVH)
- Ways to regulate BP:
  - 1- Minute to minute

Low BP $\rightarrow$  sense the baroreceptors $\rightarrow$  stimulate sympathetic pathway $\rightarrow$  release epinephrine and norepinephrine $\rightarrow$  bind to beta 1 adrenergic receptor in the heart  $\rightarrow$  increase heart rate $\rightarrow$ Increase cardiac output $\rightarrow$  increase the action of alpha receptors in smooth muscle of the blood vessels $\rightarrow$ vasoconstriction $\rightarrow$ increase PVR  $\rightarrow$  BP

2- Kidney →( for chronic hypotension)→lowering the renal blood flow→ release renin → release angiotensin ll ( neuropeptide)→ will bind to vascular smooth muscle receptors → causeing high PVR → high BP,,,,,OR angiotensin can bind to its receptors in the kidney → causing releasing of aldosterone → increase the reabsorption of Na+ and water→ increase the blood volume→increase BP\*\*Note: orthostatic hypotension→ due to change the position