**Lecture-1**

**Arterial blood pressure**: means the *force exerted by the blood against any unit area of the vessel wall*. Blood pressure almost always is measured in millimeters of mercury (mm Hg) because the mercury manometer has been used as the standard reference for measuring pressure. The pressure in the aorta and in the brachial and other large arteries in a young adult human rises to a peak value **(systolic pressure)** of about 120 mm Hg during each heart cycle and falls to a minimum **(diastolic pressure)** of about 70 mm Hg . Systolic pressure is produced by ejection of blood into aorta during left ventricular systole while diastolic pressure is produced as a result of the elastic recoil of the aorta during ventricular diastole. The arterial pressure is conventionally written as systolic pressure over diastolic pressure, for example, 120/70 mm Hg.

arterial pressure is the product of the cardiac output and the peripheral resistance, it is affected by conditions that affect either or both of these factors.

BP=CO\*PVR ( CO: cardiac output, PVR: peripheral vascular resistance)

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| The **pulse pressure,** the difference between the systolic and diastolic pressures, is normally about 40 mm Hg. The **mean blood pressure** is the average pressure throughout the cardiac cycle.  Mean BP=diastolic BP+1/3pulse pressure. Figure-1  Image6 |

Figure-1 Arterial blood pressure curve

**Methods of Measuring Blood Pressure*:***

**1-Invasive method:** a cannula is inserted into an artery, the arterial pressure can be measured directly with a mercury manometer or a suitably calibrated strain gauge.

**2-Non invasive methods**: BP measured by **sphygmomanometer**. It includes :

I-palpatory method: The cuff is wrapped around the arm .the radial pulse is palpated then the cuff is inflated till the pulse disappears which indicate complete obstruction of brachial artery, then the cuff is slowly deflated till radial pulse becomes palpable. by this methods only systolic BP is measured.

II-Auscultatory method: The arterial blood pressure in human is routinely measured by the **auscultatory method.** An inflatable cuff **(Riva–Rocci cuff)** attached to a mercury manometer **(sphygmomanometer)** is wrapped around the arm and a stethoscope is placed over the brachial artery at the elbow. The cuff is rapidly inflated until the pressure is well above the expected systolic pressure in the brachial artery. The artery is occluded by the cuff, and no sound is heard with the stethoscope. The pressure in the cuff is then lowered slowly. At the point at which systolic pressure in the artery just exceeds the cuff pressure, a spurt of blood passes and certain sounds called **Korotkoff sounds are heard.** The sounds of Korotkoff are produced by changing laminar blood flow into turbulent flow in the brachial artery.The first sound heard represent systolic pressure(phase1 of Korotkoff sound). Then the sound becomes louder,then dull, muffled and finally disappear. Diastolic pressure represented when the sound disappears.

**Phases of Korotkoff sounds are:**

- Phase1, tapping sound.

-Phase2, louder sound.

-Phase3,dull.

-Phase4, muffled sound.

-Phase5, disappeared.

In hyper dynamic conditions (e.g) pregnancy, children, and some disease conditions (e.g) hyperthyriodisim ,aortic insufficiency ,diastolic blood pressure represented by phase4.

**Regulation of arterial blood pressure**

arterial pressure is regulated not by a single pressure controlling system but instead by several interrelated systems:

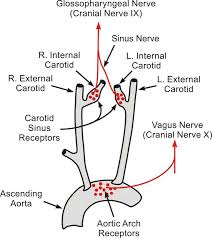
-**Short term mechanisms.**

**-Long term mechanisms.**

**Short term mechanism:** these mechanisms act within few seconds of alteration of BP. They are (**1) the baroreceptor feedback mechanism**: Baroreceptors are stretch receptors that are extremely abundant in (a) the wall of each internal carotid artery slightly above the carotid bifurcation,

an area known as the *carotid sinus,* and (b) the wall of the aortic arch.

signals from the “carotid baroreceptors” are transmitted through *glossopharyngeal nerves*  to the medulla. Signals from the“aortic baroreceptors” in the arch of the aorta are transmitted through the *vagus nerves* also to the medulla. When arterial BP increases cause stimulation of baroreceptors ,so signals transmitted to the medulla, secondary signals *inhibit the vasoconstrictor center* of the medulla and *stimulate cardiac inhibitory center(CIC) and vasodilator center(VDC).* The net effects are (1) *vasodilatation* of the veins and arterioles . (2) *decreased heart rate* and *strength of heart contraction.* Therefore, excitation of the baroreceptors by high pressure in the arteries reflexly *causes the arterial pressure to decrease* because of both a decrease in peripheral resistance and a decrease in cardiac output. Conversely, low pressure has opposite effects by inhibition of baroreceptors causing the pressure to rise back toward the normal.Figure-2.



vasoconstriction

Increased HR and contractility of heart

baroreceptors

Vasomotor center

Decreased BP

Increased BP

Fig-2 Baroreceptor system for controlling arterial pressure.

**(2) the chemoreceptor mechanism:** The *chemoreceptors* are chemosensitive cells to oxygen lack, carbon dioxide excess, and hydrogen ion excess. They are located in several small *chemoreceptor organs*  (two *carotid bodies,* one of which lies in the bifurcation of

each common carotid artery, and *aortic bodies* adjacent to the aorta). The chemoreceptors excite nerve fibers that pass through Hering nerve and vagus nerve into vasomotor center. Whenever the arterial pressure falls

below a critical level, the chemoreceptors become stimulated because diminished blood flow causes decreased oxygen as well as excess buildup of carbon dioxide and hydrogen ions that are not removed by the slowly

flowing blood.The signals transmitted from the chemoreceptors *excite* the vasomotor center, and this elevates the arterial pressure back toward normal. However, this chemoreceptor reflex is not a powerful arterial pressure controller until the arterial pressure falls below 80 mm Hg.

**(3) the central nervous system ischemic mechanism**: when blood flow to the vasomotor center in the lower brain stem becomes decreased severely enough to cause *cerebral ischemia—*the vasoconstrictor and cardioaccelerator neurons in the vasomotor center respond directly to the ischemia and maintain cerebral blood flow. This CNS ischemic response occur when BP falls below 60mmHg.

**-Long term mechanism:** involve the renin-angiotensin system,rennin is synthesized and stored in the juxtaglomerular cells of the kidney, these cells located in the wall of the afferent arterioles proximal to the glomeruli. :decreased BP stimulate secretion of rennin enzyme from kidney this enzyme act on angiotensinogen (synthesized by the liver) forming angiotensinI which is converted to angiotensinII by angiotensin converting enzyme which is occur in the lung .this angiotensinII has potent vasoconstrictor effect. angiotensinII has two principal effects that can elevate arterial pressure. The first of these, *vasoconstriction in many*

*areas of the body*, occurs rapidly. Vasoconstriction occurs intensely in the arterioles so increases the total peripheral resistance, thereby raising the arterial. The second principal means by which angiotensin increases the arterial pressure is to *decrease excretion* *of both salt and water* by the kidneys*.* This slowly increases the extracellular fluid volume, which then

increases the arterial pressure during subsequent hours and days.

AngiotensinII that is formed from rennin-angiotensin system causes the kidney retain both salt and

water in two major ways: (fig-3)

1. AngiotensinII acts directly on the kidneys to cause salt and water retention.

2. AngiotensinII stimulates the adrenal glands to secrete aldosterone, and the aldosterone in turn increases salt and water reabsorption by the kidney tubules.

Decreased arterial pressure

Renin(kidney)

Angiotensinogen Angiotensin I

ACE(lung)

ACE(lung)

AngiotensinII

Secretion of aldosterone from adrenal gland

Vasoconstriction

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**Figure -3 Renin angiotensin system**

Increased arterial blood pressure

Renal retention of salt and water

**Venous pressure:**

*central venous pressure*: it is the pressure in the right atrium as Blood from all the systemic veins flows into the right atrium of the heart; *Right atrial pressure is regulated by a balance between (1) the ability of the heart to pump blood out of the right atrium and ventricle into the lungs* . *(2)the tendency for blood to flow from the peripheral veins into the right atrium.* If the right heart is pumpingstrongly, the right atrial pressure decreases. Conversely,weakness of the heart elevates the right atrial pressure. Also, any effect that causes rapid inflow ofblood into the right atrium from the peripheral veinselevates the right atrial pressure.

***Peripheral venous pressure:***

***Muscle pump****:* every time one moves the legs by contraction of the muscles which compress the veins adjacent to them and this squeezes the blood out of the veins. the valves in the veins are arranged so that the direction of venous blood flow can be only toward the heart.

**Thoracic Pump**: During inspiration, the intrapleural pressure falls . This negative pressure causes drop in central venous pressure during inspiration which increase venous return. When the diaphragm descends during inspiration, intra-abdominal pressure rises, and this squeezes blood toward the heart because backflow into the leg veins is prevented by the venous valves.