The blood pressure is monitored and regulated inside the body and is called mean arterial pressure (MAP). The clinical measurement of the blood pressure with a sphygmomanometer depicts the systolic and diastolic arterial pressures and gives relative information of the mean arterial pressure. MAP is the driving force that propels the blood to organ tissues. As all body tissues require oxygen for the process of respiration and energy, a constant supply of blood is mandatory. MAP, therefore, needs to be regulated for two reasons. Firstly, the driving force created by MAP should be sufficient enough to supply blood to the organs lying above the level of the heart, i.e., against gravity. Secondly, it should not be above the normal limits as it will then cause an extra load on the heart, which will have to pump blood with an extra force.

Determinants of Mean Arterial Pressure

Mean arterial pressure is not only affected by the components of the circulatory system, but other organs of various body systems are also involved. The two major determinants of the MAP are the **cardiac output** and the **peripheral resistance**. The relation between the MAP and its two determinants is given by the following equation:

\[
\text{Mean arterial pressure} = \text{Cardiac output} \times \text{total peripheral resistance}
\]

It is important to note here that the equation only shows the relationship of the MAP and its determinants. The actual calculation of the MAP is by the following formula:
Mean arterial pressure = Diastolic pressure + 1/3 pulse pressure

Cardiac Output

Cardiac output is defined as the volume of blood pumped by the heart in one minute. The greater the cardiac output, the greater will be the mean arterial pressure.

Cardiac output is dependent on two factors – stroke volume and heart rate.

\[ \text{Cardiac output} = \text{Heart rate} \times \text{Stroke volume} \]

Both the heart rate and the stroke volume are directly proportional to the cardiac output. However, under a constant value of cardiac output, they share an inverse relationship with each other.

Heart rate is the number of heartbeats per minute. As the heart rate increases, the cardiac output also increases. The heart rate is controlled by a relative balance between the parasympathetic and the sympathetic nervous stimulation.

The former decreases the heart rate, while the latter has the opposite effect. The accelerated heart rate in the sympathetic stimulation is because of the release of epinephrine by the sympathetic nerves. It acts on the cardiac muscle to increase the heart rate.

Stroke volume is the volume of blood pumped by the heart in one beat. It increases in response to sympathetic stimulation and by an increased venous return to the heart.

According to Frank-Starling law, the increase in venous return causes an increased ventricular filling of the heart and therefore greater stroke volume. Intrinsic factors affecting the stroke volume are the myocardial contractility and the size of the vascular compartment. It is calculated by the following formula:

\[ \text{Stroke volume} = \text{End-diastolic volume} - \text{End-systolic volume} \]

Factors which affect the venous return to the heart are:

Musculovenous pump: The soleus muscle of the leg acts as a pumping organ. When a person is doing any physical activity such as walking, running, skipping, etc., the soleus muscle contracts, which pushes the blood in the veins towards the heart. Therefore, physical activity promotes venous return. For the same reason, the soleus is known as the ‘second heart.’

Sympathetic stimulation of the veins increases vasomotor tone and therefore increases the venous return.
Respiratory pump: The pressure gradient between the supra-diaphragmatic and infra-diaphragmatic parts of inferior vena cava, produced by the inspiratory movement, increases the venous return. This pressure gradient is created by the decreased intra-thoracic pressure and the increased intra-abdominal pressure caused by the movement of the diaphragm during inspiration.

Vena cava compression: When the thoracic vena cava is compressed as in the Valsalva maneuver or during late pregnancy, it decreases the venous return.

Gravity: The gravity causes pooling of blood in the veins of lower parts of the body which creates a pressure gradient. This should practically increase the venous return. However, as the person stands up, the right atrial pressure decreases and so does the stroke volume, which eventually decreases the arterial pressure.

Right atrial pressure: As there is no valve present between the right atrium and the vena cava, any change in the pressure in the right atrium will alter the venous pressure.

Cardiac suction effect: The cardiac suction effect increases the venous return, as it tends to pull more blood from the vessel into the ventricles.

Blood volume: An increase in the blood volume increases the venous return. The volume of blood depends upon the percentage of blood and shift of fluid present in the vascular and interstitial compartment. It also depends upon the salt and water retention by the kidneys, under the action of vasopressin hormone and renin-angiotensin-aldosterone system.

Peripheral Resistance

The resistance of the arteries to the blood flow is called the peripheral resistance. As the peripheral resistance increases, the mean arterial pressure also increases. Factors which affect the peripheral resistance are the arteriolar radius, blood viscosity, and vessel structure.

Arteriolar radius: As the radius of the artery decreases, the peripheral resistance increases and so does the arterial pressure. In this situation, the blood flow to the tissues will also decrease.

The arteriolar radius is influenced by the local metabolic control, which may cause vasodilation to increase the flow of blood to the tissues or it may result in vasoconstriction.

Sympathetic stimulation and the release of epinephrine also result in vasoconstriction. Similarly, other hormones such as vasopressin and angiotensin II
increase peripheral resistance by decreasing the radius of the arteries.

**Blood viscosity** refers to the presence of cellular components in the blood. The greater the percentage of the *cellular components*, the greater will be the blood viscosity and hindrance in the blood flow. As the blood viscosity increases, the peripheral resistance also increases.

**Vessel structure**: With increasing age, the elastic tissue content of the arteries decreases. Arteries, therefore, do not effectively compensate for the increased blood pressure because of an increase in the peripheral resistance.

### Hormones Affecting Arterial Blood Pressure

#### The renin-angiotensin-aldosterone system

The renin-angiotensin-aldosterone system is a hormone system that regulates the plasma level of sodium and the arterial blood pressure. It is either activated as a result of a drop in blood pressure, which is sensed by the *baroreceptors*, or if there is decreased renal perfusion.

Once activated, the *juxtaglomerular cells* secrete an enzyme called *renin*. Renin converts angiotensinogen to angiotensin I. An enzyme called angiotensin-converting enzyme (ACE), found in the capillaries of the *lungs*, converts angiotensin I to angiotensin II.

Angiotensin II causes vasoconstriction of the arterioles. This increases peripheral resistance and an increase in the systemic arterial blood pressure. It also causes a release of *aldosterone* from the *zona glomerulosa* of the *adrenal cortex*. Aldosterone acts on the distal convoluted tubules and the collecting duct of the *kidney* to increase the reabsorption of sodium ions.

It stimulates the *pituitary gland* to secrete an *antidiuretic hormone (ADH)*, also known as vasopressin. ADH also acts on the collecting tubules to increase water reabsorption by the formation of aqua-porins on the luminal surface of epithelial cells.

The final outcome of the activation of the renin-angiotensin-aldosterone system is the increase in peripheral resistance, salt and water retention which will cause an increase in arterial blood pressure.

#### Epinephrine and Norepinephrine

A decrease in blood volume causes a release of epinephrine and norepinephrine by the *adrenal gland*. Both epinephrine and norepinephrine tend to increase arterial blood pressure by increasing the heart rate and the cardiac force of contractility.

*Norepinephrine* is mainly responsible for the *vasoconstriction* because of its stimulation of the alpha receptors present on *blood vessels*.

*Epinephrine*, on the other hand, causes *vasodilation* at low concentration (by activation of beta-2 receptors) and *vasoconstriction* at high concentration (by activation of alpha receptors). The final response is the increase in cardiac output.
Erythropoietin

Erythropoietin is a hormone secreted by the kidneys in response to low hemoglobin levels in the blood. Erythropoietin acts on the hematopoietic stem cells in the bone marrow and stimulates the production of red blood cells. An increased number of red blood cells in the plasma will increase the plasma viscosity which will increase the peripheral resistance and finally the arterial pressure.

References


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