

## Physiology of Blood Vessels and Blood Pressure

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**Blood pressure is monitored and regulated within the body and known as mean arterial pressure (MAP). The clinical measurement of blood pressure using 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 blood to organ tissues. Since body tissues require oxygen for the process of respiration and energy, constant blood supply is mandatory. MAP, therefore, needs to be regulated for two reasons: Firstly, the driving force created by MAP should be adequate to supply blood to the organs above the heart level, i.e. against gravity. Secondly, the MAP should not be above normal limits as it will increase the load on the heart.**



### Determinants of Mean Arterial Pressure

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

$$\text{Mean arterial pressure} = \text{cardiac output} \times \text{total peripheral resistance}$$

It is important to note that the equation only represents the relationship between the MAP and its determinants. The actual MAP can be calculated using 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 per minute. The greater the cardiac output, the greater is the mean arterial pressure.

Cardiac output is dependent on stroke volume and heart rate and can be represented as follows:

$$\text{Cardiac output} = \text{heart rate} \times \text{stroke volume}$$

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

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

The former decreases heart rate, while the latter has the opposite effect. Accelerated heart rate during sympathetic stimulation is due to the release of **epinephrine** by the sympathetic nerves, which acts on the cardiac muscle to increase heart rate.

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

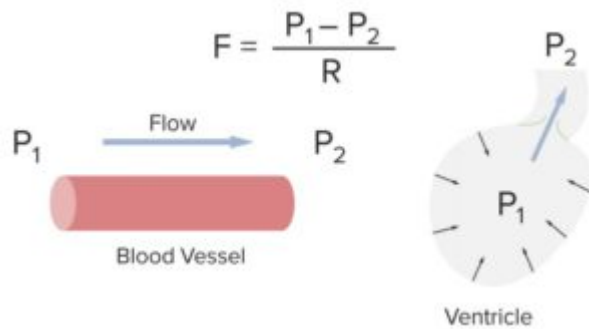
According to the Frank-Starling law, an increase in venous return causes an increased ventricular filling of the heart and therefore results in greater stroke volume. Intrinsic factors affecting stroke volume are **myocardial contractility** and size of the vascular compartment. It is calculated using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

$$\text{Stroke volume} = \text{end-diastolic volume} - \text{end-systolic volume}$$

Factors that affect venous return to the heart are as follows:

**Musculovenous pump:** The soleus muscle of the leg acts as a pump. When an individual is involved in physical activities such as walking, running, or skipping, the soleus muscle contracts and increases venous blood flow to the heart. Therefore, [physical activity](#) promotes venous return. Owing to the pumping action of the soleus muscle, it is often known as the 'second heart.'

**Sympathetic stimulation of the veins** increases **vasomotor tone** and therefore increases venous return.



"Pressure Gradient" Image created by Lecturio

**Respiratory pump:** The pressure gradient between the supradiaphragmatic and infradiaphragmatic regions of the inferior vena cava resulting from the inspiratory movement increases venous return. This pressure gradient is created by decreased intra-thoracic pressure and increased intra-abdominal pressure caused by the movement of the diaphragm during inspiration.

**Vena cava compression:** Compression of the thoracic vena cava during the Valsalva maneuver or late pregnancy impedes venous return.

**Gravity:** Gravity causes pooling of blood in the veins of the lower extremities and creates a pressure gradient. This phenomenon should ideally increase venous return; however, when an individual stands up, right atrial pressure and stroke volume decrease, which eventually decreases arterial pressure.

**Right atrial pressure:** Due to the absence of a valve between the right atrium and vena cava, any pressure change in the right atrium alters venous pressure.

**Cardiac suction effect:** The cardiac suction effect increases venous return since it tends to draw more blood from the blood vessels into the ventricles.

**Blood volume:** The increase in blood volume increases venous return. Blood volume depends on the shift of fluids present in the vascular and interstitial compartments. It also depends on the extent of salt and water retention by the [kidneys](#) due to the action of **vasopressin** and the **renin-angiotensin-aldosterone system**.

## Peripheral Resistance

The resistance of arteries to blood flow is termed as peripheral resistance. As peripheral resistance increases, the mean arterial pressure increases correspondingly. Factors affecting peripheral resistance are arteriolar radius, blood viscosity, and vessel structure.

As arteriolar radius decreases, peripheral resistance increases, as does arterial pressure. In this situation, blood flow to tissues decreases.

**Arteriolar radius** is influenced by local metabolic control, which may cause vasodilation and increase blood flow to tissues, or result in vasoconstriction.

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

**Blood viscosity** is affected by the presence of cellular components in blood. The greater the percentage of [cellular components](#), the greater is the blood viscosity and resistance to blood flow. Thus, as blood viscosity increases, the peripheral resistance

correspondingly increases.

Aging causes a decrease in the [elasticity of arteries](#); therefore, they do not effectively compensate for an increase in blood pressure when there is an increase in peripheral resistance.

## Hormones Affecting Arterial Blood Pressure

### The renin-angiotensin-aldosterone system

The renin-angiotensin-aldosterone system regulates **plasma sodium levels** and **arterial blood pressure**. It is activated following a drop in blood pressure, which is sensed by the [baroreceptors](#), or if there is decreased renal perfusion.

Once activated, the **juxtaglomerular cells** secrete **renin**, which converts angiotensinogen to angiotensin I. Angiotensin-converting enzyme, 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 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 collecting ducts of the [kidneys](#) to increase sodium reabsorption.

Angiotensin II stimulates the **pituitary gland** to secrete **antidiuretic hormone (ADH)**, also known as vasopressin. ADH then acts on the collecting tubules to increase water reabsorption by the formation of aquaporins on the luminal surface of epithelial cells.

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

### Epinephrine and Norepinephrine

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

**Norepinephrine** induces **vasoconstriction** by stimulating the  $\alpha$ -receptors in [blood vessels](#).

**Epinephrine** causes **vasodilation** at low concentrations (by activation of  $\beta$ -2 receptors) and **vasoconstriction** at high concentrations (by activation of  $\alpha$ -receptors). The final outcome is an increase in cardiac output.

### Erythropoietin

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

# References

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