



Advanced Physiology

Lec.1 Physiology of Cardiovascular system

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Introduction

The cardiovascular system comprises:

- 1- **pump**: the heart, which comprises **two atria** (reservoirs for blood and booster pumps to augment ventricular filling) and **two ventricles** (pumps). It consists of two pumps in parallel, with **synchronised actions**.
- 2- **high pressure distribution circuit**: the elastic arteries, i.e. the **aorta** and its major branches, serve a transport function. The muscular arteries serve a distributive function.
- 3- **Exchange vessels**: the capillaries, which are 8–10 µm in diameter. Capillaries can possess either continuous endothelium (muscle, heart, liver), fenestrated endothelium (gastrointestinal tract, renal glomeruli), or discontinuous endothelium (liver, spleen).
- 4- **low pressure collection and return circuit**. The system allows rapid transport of oxygen, nutrients, hormones and waste products throughout the body. The structure of the components is related to function across the vascular tree.

The fetal circulation

This has the following characteristics:

- 1- The right and left ventricles function in parallel;
- 2- The placenta provides for gas and metabolite exchange;
- 3- The parallel circulation is maintained by shunts at the ductus venosus, foramen ovale and the ductus arteriosus.
- 4- Oxygenated blood flows from the placenta via the ductus venosus and inferior vena cava into the right atrium, where it is deflected by the crista dividens and the eustachian valve across the foramen ovale into the left atrium and thence into the left ventricle. It is then ejected into the ascending aorta.
- 5- Deoxygenated blood from the superior vena cava flows into the right atrium, being primarily directed into the right ventricle and ejected into the pulmonary artery.
- 6- Mixing of the venous returns occurs.
- 7- The pulmonary circulation is a high-impedance and low-flow system.

8- The placental circulation is a low-impedance and high-flow system
At birth, a transitional circulation ensues, with the following changes:
A reduction in pulmonary vascular resistance secondary to expansion of the lungs and an increase in arterial pO₂.
An increase in systemic vascular resistance caused by removal of the low resistance placental circulation.
The systemic vascular resistance exceeds the pulmonary vascular resistance.
A left-to-right shunt through the ductus arteriosus, with progressive closure.
Functional closure of the foramen ovale as a result of raised left atrial pressure and volume.
Closure of the ductus venosus as a result of removal of the placenta from the circulation.
The adult circulation consists of two chambers, the right and left ventricles, in series, with two interposed vascular beds (systemic and pulmonary).

Control of blood volume

***Blood volume control mechanism**

Afferent limb

1- Volume sensors

Low-pressure baroreceptors (venous side stretch receptors)

Cardiac atria

Roots of great veins

Cardiopulmonary receptors

Left ventricle

Pulmonary vascular bed

High-pressure baroreceptors (arterial side stretch receptors)

Carotid sinus (glossopharyngeal nerve)

Aortic arch (vagus nerve)

2- Intrarenal baroreceptor system: juxtaglomerular apparatus

3- Hepatic and central nervous system(CNS) sensors

Central control system

1_ Cardiovascular centre

Pressor system: lateral pathway of descending reticular system

Depressor system: medial pathway of descending reticular system

2_ Nucleus of the tractus solitarius

3_ Hypothalamus

Efferent mechanisms

Those regulating renal sodium excretion and controlling extracellular fluid volume

1_ Glomerular filtration rate

2_ Physical factors

At proximal tubule level

Beyond proximal tubule level

3- Humoral effector mechanisms

Renin–angiotensin–aldosterone system

Vasopressin

Catecholamines

Prostaglandins: PGE₂, PGI₂, thromboxane

Kinin–kallikrein system

Atrial natriuretic peptide (ANP)

Endothelium-derived factors

4- Renal sympathetic nerves

Effects of blood loss

The effects of the rapid loss of one litre of blood (20% total blood volume) in an adult can be listed in sequence as consisting of:

1- Blood loss leading to reduced venous return, reduced right atrial pressure and reduced cardiac output.

2- Immediate baroreceptor reflex activation. A reduced discharge rate of baroreceptors in carotid sinus and aortic arch leads to reduced afferent input to Control of blood volume the medullary cardiovascular centre causing a reduction in parasympathetic, and increase in sympathetic, activity. This is manifested by tachycardia, increased myocardial contractility and arteriolar constriction.

3- A slower hypothalamic–pituitary–adrenal response. Reduced renal blood flow stimulates intrarenal baroreceptors with renin release from the juxtaglomerular apparatus. Angiotensin II which results causes arteriolar constriction and stimulates aldosterone and arginine vasopressin (antidiuretic hormone;ADH) release. Arginine vasopressin release is also stimulated by reduced extracellular fluid volume acting via atrial stretch receptors.

4- Redistribution of cardiac output from skin, muscle, viscera to heart and brain.

5- Starling forces, which are responsible for tissue fluid reabsorption into the plasma compartment.

Movement out of capillary:

Capillary hydrostatic pressure

Interstitial fluid colloid osmotic pressure

Fluid retention within the capillary:

Plasma colloid osmotic pressure

Interstitial space hydrostatic pressure

6- Carotid chemoreceptor response: reduced blood flow to the aortic and carotid bodies, with reduced oxygen in the peripheral chemoreceptor tissues, leading to hyperventilation.

Blood pressure

Systemic arterial blood pressure is related to cardiac output and to total peripheral resistance (in turn related to the number and calibre of small arteries and arterioles, and to blood viscosity). Arterial hypertension represents a disproportion between these two components.

Blood pressure comprises:

A steady component, **the mean arterial pressure**, which is related to small arterial and arteriolar resistance. This in turn depends on a combination of neural mechanisms (sympathetic and parasympathetic), hormonal mechanisms (renin–angiotensin–aldosterone mechanism) and local transmitters (nitric oxide), all of which affect arteriolar calibre.

A pulsatile component, the **pulse pressure**, which depends on arterial stiffness and the timing of reflected waves. It represents the oscillation around mean pressure, extending from systolic to diastolic pressures.

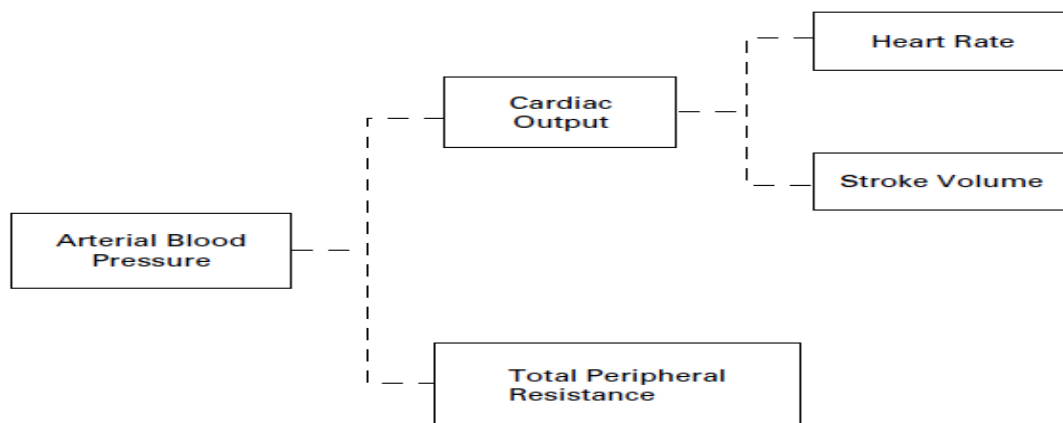


Figure 6.1 Determinants of arterial blood pressure.

Mean arterial pressure is equal to cardiac output multiplied by total peripheral resistance. Pulse pressure is equal to systolic minus diastolic pressure. It is directly proportional to stroke volume and inversely proportional to compliance. Pulse pressure increases with age due to reduced compliance.

Determinants of arterial blood pressure

Physical

Blood volume in arterial system

Elastic characteristics (compliance)

Physiological

Cardiac output

Peripheral resistance

Systolic blood pressure depends on myocardial contractility, the compliance of the great vessels and on diastolic blood pressure. Diastolic blood pressure depends on systemic vascular resistance, peripheral run-off and on heart rate.

Factors regulating blood pressure

Short-term regulation

Baroreceptors comprise a negative feedback system incorporating stretch receptors sensitive to both mean pressure and rate of change of pressure (elevations in blood pressure increase the rate of firing). Under physiological conditions, baroreceptor firing exerts a tonic inhibitory influence on the sympathetic outflow from the medulla oblongata. High-pressure baroreceptors are found in the carotid sinus, at the bifurcation of the common carotid artery and in the aortic sinus in the arch of the aorta.

1-Hypothalamus.

2-Arterial chemoreceptors.

3-Vomiting centre.

4-Pulmonary stretch receptors.

5-Ventricular mechanoreceptors.

6-Atrial stretch receptors.

Longer-term regulation

Alterations in blood volume and osmolality.

Hormonal regulation of blood pressure.

This is achieved by a balance of vasopressor and vasodepressor hormones, which include the following:

Vasopressor hormones:

Renin-angiotensin-aldosterone system.

Arginine vasopressin.

Catecholamines.

Endothelins (ETs).

Vasodepressor hormones:

Natriuretic peptides: ANP, brain type natriuretic peptide (BNP).

Kinin–kallikrein system.

Prostaglandins (PGI₂, prostacyclin).

Medullipine system.

Adrenomedullin.

Nitric oxide.