NAME: EWHRUDJAKPOR OGHENERUKEVWE FAVOUR

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**Long-Term Regulation of Blood Pressure**

There are several physiological mechanisms that regulate blood pressure in the long-term, the first of which is the renin-angiotensin-aldosterone system**(RAAS)**.

**Renin-Angiotensin-Aldosterone System (RAAS)**

*Renin* is a peptide hormone released by the granular cells of the**juxtaglomerular apparatus** in the kidney. It is released in response to:

* Sympathetic stimulation
* Reduced sodium-chloride delivery to the distal convoluted tubule
* Decreased blood flow to the kidney

Renin facilitates the conversion of angiotensinogen to angiotensin I which is then converted to angiotensin II using angiotensin-converting enzyme**(ACE)**.

Angiotensin II is a potent vasoconstrictor. It acts directly on the kidney to increase sodium reabsorption in the proximal convoluted tubule. Sodium is reabsorbed via the sodium-hydrogen exchanger. *Angiotensin II* also promotes release of **aldosterone**.

ACE also breaks down a substance called **bradykinin** which is a potent vasodilator. Therefore, the breakdown of bradykinin potentiates the overall constricting effect.

Aldosterone promotes salt and water retention by acting at the distal convoluted tubule to increase expression of **epithelial** sodium channels. Furthermore, aldosterone increases the activity of the basolateral sodium-potassium ATP-ase, thus**i**ncreasing the electrochemical gradien**t** for movement of sodium ions.

More sodium collects in the kidney tissue and water then follows by osmosis. This results in decreased water excretion and therefore increased blood volume and thus blood pressure.

**Anti-Diuretic Hormone (ADH)**

The second mechanism by which blood pressure is regulated is release of Anti Diuretic Hormone (ADH) from the OVLT of the hypothalamus in response to **thirst** or an increased plasma osmolarity.

ADH acts to increase the permeability of the collecting duct to water by inserting **aquaporin channels** (AQP2) into the apical membrane.

It also stimulates sodium reabsorption from the thick ascending limb of the loop of Henle. This increases water reabsorption thus increasing plasma volume and decreasing osmolarity.

**Further Control of Blood Pressure**

Other factors that can affect long-term regulation of blood pressure are natriuretic peptides. These include:

* Atrial natriuretic peptide**(ANP)** is synthesized and stored in cardiac myocytes. It is released when the atria are stretched, indicating of high blood pressure.
* ANP acts to promote sodium excretion. It dilates the **afferent arteriole** of the glomerulus, increasing blood flow (GFR). Moreover, ANP inhibits sodium reabsorption along the nephron. Conversely, ANP secretion is low when blood pressure is low.
* **Prostaglandins** act as local vasodilators to increase GFR and reduce sodium reabsorption. They also act to prevent excessive vasoconstriction triggered by the sympathetic nervous and renin-angiotensin-aldosterone systems.

2)a) Pulmonary circulation is the system of transportation that shunts de oxygenated blood from the heart to the lungs to be re-saturated with oxygen before being dispersed into systemic circulation.De-oygenated blood from the lower half of the body enters the heart from the inferior vena cava while de-oxygenated blood from the upper body is delivered to the heart via the superior vena cava. Both the superior and inferior vena cava empty blood into the right atrium. Blood flows through the tricuspid valve into the right ventricle. It then flows through the pulmonary valve into the pulmonary artery before it is delivered to the lungs.

**Clinical Significance**

A number of medical conditions can affect the pulmonary circulation.

* [Pulmonary hypertension](https://en.wikipedia.org/wiki/Pulmonary_hypertension) describes an increase in resistance in the pulmonary arteries
* [Pulmonary embolus](https://en.wikipedia.org/wiki/Pulmonary_embolus) is a [blood clot](https://en.wikipedia.org/wiki/Thrombus), usually from a [deep vein thrombosis](https://en.wikipedia.org/wiki/Deep_vein_thrombosis) that has lodged in the pulmonary vasculature. It can cause difficulty breathing or chest pain, is usually diagnosed through a [CT pulmonary angiography](https://en.wikipedia.org/wiki/CT_pulmonary_angiography) or [V/Q scan](https://en.wikipedia.org/wiki/V/Q_scan), and is often treated with [anticoagulants](https://en.wikipedia.org/wiki/Anticoagulants) such as [heparin](https://en.wikipedia.org/wiki/Heparin) and [warfarin](https://en.wikipedia.org/wiki/Warfarin).
* [Cardiac shunt](https://en.wikipedia.org/wiki/Cardiac_shunt) is an unnatural connection between parts of the heart that leads to blood flow that bypasses the lungs.
* [Vascular resistance](https://en.wikipedia.org/wiki/Vascular_resistance)
* [Pulmonary shunt](https://en.wikipedia.org/wiki/Pulmonary_shunt)

b)**Circle of Willis**

The circle of Willis is a circulatory anastomosis that supplies blood to the brain and surrounding structures. It is formed by 2 arteries namely; the internal carotid arteries and the vertebro-basilar system.

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| Key facts |
| Definition and function | Anastomosis between the anterior and posterior circulation. Provides arterial branches that vascularize the brain. |
| Anterior circulation | Common carotid -> internal carotid -> anterior cerebral artery (-> anterior communicating artery), middle cerebral artery  |
| Posterior circulation | Subclavian arteries -> vertebral arteries -> unite forming the basilar artery -> anterior inferior cerebellar, superior cerebellar, posterior cerebral arteries(->posterior communicating artery) |
| Circle of Willis | Polygonal anastomosis between: Internal carotid artery (branch of the common carotid)Anterior cerebral artery (branch of the internal carotid)Anterior communicating artery (branch of the anterior carotid, connects left and right anterior cerebral arteries)Posterior cerebral artery (branch of the basilar artery)Posterior communicating artery (branch of the posterior cerebral, connects the three cerebral arteries on the same side) |
| Clinical significance | Thrombosis, occlusion, aneurysm, rupture, infarction, ischemic attack, cerebral hemorrhage |

c) **Splanchic circulation**

The splanchnic circulation is composed of gastric, small intestinal, colonic, pancreatic, hepatic, and splenic circulations, arranged in parallel with one another. The three major arteries that supply the splanchnic organs, cellac and superior and inferior mesenteric, give rise to smaller arteries that anastomose extensively. The circulation of some splanchnic organs is complicated by the existence of an intramural circulation. Redistribution of total blood flow between intramural vascular circuits may be as important as total blood flow. Numerous extrinsic and intrinsic factors influence the splanchnic circulation. Extrinsic factors include general hemodynamic conditions of the cardiovascular system, autonomic nervous system, and circulating neurohumoral agents. Intrinsic mechanisms include special properties of the vasculature, local metabolites, intrinsic nerves, paracrine substances, and local hormones. The existence of a multiplicity of regulatory mechanisms provides overlapping controls and restricts radical changes in tissue perfusion.

d) **Coronary circulation**

**Coronary circulation**, part of the systemic [circulatory system](https://www.britannica.com/science/circulatory-system) that supplies blood to and provides drainage from the tissues of the [heart](https://www.britannica.com/science/heart). In the human heart, two coronary arteries arise from the [aorta](https://www.britannica.com/science/aorta) just beyond the semi lunar valves; during [diastole](https://www.britannica.com/science/diastole-heart-function), the increased aortic pressure above the valves forces blood into the coronary arteries and thence into the musculature of the heart. Deoxygenated blood is returned to the chambers of the heart via coronary veins; most of these converge to form the coronary [venous sinus](https://www.britannica.com/science/venous-sinus), which drains into the right [atrium](https://www.britannica.com/science/atrium-heart).

**E) Cutaneous Circulation**

The cutaneous circulation is the circulation and blood supply of the skin. The skin is not a very metabolically active tissue and has relatively small energy requirements, so its blood supply is different to that of other tissues.

Some of the circulating blood volume in the skin will flow through will flow through **arteriovenous anastomoses (AVAs)** instead of capillaries. AVAs serve a role in temperature regulation. In this article we shall consider the different adaptations of the cutaneous circulation, and its role in body temperature control.

**3)** The integrated response to severe exercise involves fourfold to fivefold increases in cardiac output, which are due primarily to increases in cardiac rate and to a lesser extent to augmentation of stroke volume. The increase in stroke volume is partly due to an increase in end-diastolic cardiac size (Frank-Starling mechanism) and secondarily due to a reduction in end-systolic cardiac size. The full role of the Frank-Starling mechanism is masked by the concomitant tachycardia. The reduction in end-systolic dimensions can be related to increased contractility, mediated by beta adrenergic stimulation. Beta adrenergic blockade prevents the inotropic response, the decrease in end-systolic dimensions, and approximately 50% of the tachycardia of exercise. The enhanced cardiac output is distributed preferentially to the exercising muscles including the heart. Blood flow to the heart increases fourfold to fivefold as well, mainly reflecting the augmented metabolic requirements of the myocardium due to near maximal increases in cardiac rate and contractility. Blood flow to the inactive viscera (e.g., kidney and gastrointestinal tract) is maintained during severe exercise in the normal dog. It is suggested that local auto regulatory mechanisms are responsible for maintained visceral flow in the face of neural and hormonal autonomic drive, which acts to constrict renal and mesenteric vessels and to reduce blood flow. However, in the presence of circulatory impairment, where oxygen delivery to the exercising muscles is impaired as occurs to complete heart block where normal heart rate increases during exercise are prevented, or in congestive right heart failure, where normal stroke volume increases during exercise are impaired, or in the presence of severe anemia, where oxygen-carrying capacity of the blood is limited, visceral blood flows are reduced drastically and blood is diverted to the exercising musculature. Thus,, visceral flow is normally maintained during severe exercise as long as all other compensatory mechanisms remain intact. However, when any other compensatory mechanism is disrupted (even the elimination of splenic reserve in the dog), reduction and diversion of visceral flow occur.