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**Course: Medical Physiology**

**Assignment: Cardiovascular Physiology**

1. **Discuss the long-term regulation of mean arterial 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 synthesised 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. Write Short notes on the following**

**Pulmonary circulation**

Blood whose oxygen content has become partially depleted and whose carbon dioxide content has increased as a result of tissue metabolism, returns to the right atrium. This blood then enters the right ventricle, which pumps it into the pulmonary trunk and pulmonary arteries. The pulmonary arteries branch to transport blood to the lungs, where gas exchange occurs between the lung capillaries and the air sacs (alveoli) of the lungs. Oxygen diffuses from the air sacs to the capillary, while carbon dioxide diffuses in the opposite direction. The blood that returns to the left atrium by way of the pulmonary veins is therefore enriched in oxygen and partially depleted of carbon dioxide. The path of blood from the heart (right ventricle), through the lungs, and back to the heart (left atrium) completes one circuit which is known as the **Pulmonary circulation or Lesser circulation.**

**Circle of Willis**

The arteries which supply blood to the brain are derived from two internal carotid arteries and the basilar artery (formed by union of the right and left vertebral arteries). Branches of the internal carotid arteries and of basilar artery anastomose on the inferior surface of the brain to form the **circulus arteriosus** (circle of Willis).The circle of Willis is thus basically a free anastomoses between the two internal carotid arteries and the two vertebral arteries which equalize pressure on the arteries of the two sides. In this way, the **circulus arteriosus** allows blood that enters by either internal carotid or vertebral artery to be distributed to any part of both cerebral hemispheres. Six large arteries taking part in the formation of circle of Willis supply by their central and cortical branches to the brain substance**.**

**Splanchnic circulation**

Splanchnic circulation includes the combined vascular beds of the intestines, pancreas, spleen and liver. The main vessels which constitute the splanchnic circulation are: Arteries supplying the blood to the intestines, pancreas, spleen and liver include: Coeliac trunk is about 1 cm long and after arising from the abdominal aorta it divides into three main branches the left gastric artery hepatic artery and splenic artery, Superior mesenteric artery and Inferior mesenteric artery.

Hepatic portal system is formed by the veins draining blood from the abdominal part of the gastrointestinal tract (GIT). All these veins end in the portal vein. The portal vein supplies the blood collected from GIT to the liver by its right and left branches.

Hepatic veins are terminal parts of an elaborate venous tree that permeates the liver. The hepatic veins emerging from the liver tissue end in the inferior vena cava.

**SPLANCHNIC CIRCULATION: CHARACTERISTIC FEATURES**

During rest the abdominal GIT, viscera and liver receive about 1500 mL blood per minute (about 30% of cardiac output) via coeliac, superior mesenteric and inferior mesenteric arteries. If the entire GIT become simultaneously active, the splanchnic blood flow would have increased to about 4.0 L/ min. However, since during digestion and absorption, the GIT is sequentially activated, the maximum circulation is about 3.0 L/min. The unique feature of the splanchnic circulation is that the venous blood from GIT viscera is not directly carried to the heart through systemic veins, but is carried to the liver forming hepatic portal system.

For the purpose of discussion, the splanchnic circulation is considered to consist of three parts: Intestinal (mesenteric) circulation, Splenic circulation and Hepatic circulation.

**INTESTINAL CIRCULATION**

Intestinal or mesenteric circulation is constituted by the blood supplied to the intestines and pancreas (about 100 mL/min) by a series of parallel circulations via the branches of superior and inferior mesenteric arteries. Extensive anastomoses between the vessels constituting mesenteric circulation, but blockage of a large intestinal artery still leads to infarction. The blood flow to the intestinal mucosa is much more (about five times) than that of rest of the intestinal wall. During metabolic activity, the blood flow to GIT increases due to vagal activity (in the stomach), humoral activity, the local release of bradykinin from the mucosal glands and metabolites in the intestinal tract itself.

Counter-current system exists in the capillaries and venules in a villus, i.e. the direction of blood flow in the capillaries and venules in a villus is opposite to that in the main arteriole. This system permits diffusion of O2 from the ascending arterial limb of villi into the descending venous limb. In this way, at low flow rates substantial amount of O2 from the arterioles is shifted to the venules near the base of villi resulting in decrease in O2 supply to the mucosal cells at the tips of villi. When intestinal blood flow is very low, the transfer of O2 from arterioles to venules is exaggerated and may cause extensive necrosis of intestinal villi.

**SPLENIC CIRCULATION**

Splenic artery which is a branch of coeliac trunk supplies about 200 mL of blood/min to the spleen during rest via its splenic branches which enter the hilum of the spleen.

Spleen serves as a reservoir of blood. In spleen, two structures are involved in the storage of blood, namely splenic venous sinuses and splenic pulp. The small arteries and arterioles open directly into the splenic venous sinuses. Due to dilatation of venous sinuses, a large amount of blood is stored in spleen and the spleen distends. The capillaries of the splenic pulp are highly permeable. So, lot of blood cells pass through the capillary membrane and are stored in the pulp. The constriction of splenic venous sinuses by the sympathetic stimulation causes release of blood into the circulation.

**HEPATIC CIRCULATION**

Characteristic features of hepatic circulation

Source of blood. Liver receives about 1500 mL blood/min from two sources:

Hepatic artery which is a branch of coeliac trunk supplies about 20–25% (300–400 mL) of the total blood which caters to the metabolic requirements of the liver tissue.

Portal vein which collects blood from the mesenteric and splenic vascular bed, supplies about 75–80% (1100– 1200 mL/min) of the total blood. The hepatic and portal blood streams meet in the sinusoids.

Functional unit of liver

The functional unit of liver is acinus. There are about 10,000 acini in human liver. Thus, each acinus is at the end of vascular stalk containing terminal branches of portal vein, hepatic arteries and bile ducts. Blood flows from these terminal vessels into the sinusoids, which represent the capillary network of the liver. The sinusoids radiate towards the periphery of acinus, where they drain into the terminal branches of hepatic veins. Blood from these terminal hepatic venules drains into progressively larger branches of the hepatic veins, which are tributaries of the inferior vena cava.

**Zones of acinus**. Each acinus can be considered to have three zones: 1, 2, and 3 based on the pattern of vessels in the acinus described above. The blood supply to different zones of acinus is:

 Zone 1 refers to the central portion of acinus immediately surrounding the terminal hepatic arteriole and terminal portal venule. This zone is well oxygenated. Enzymes involved in oxidative metabolism and glucogenesis predominate here. Zone 2, i.e. the intermediate zone which is present in between zone 1 and 3 is moderately well oxygenated. It contains a mixed complement of enzymes. Zone 3 refers to the most peripheral part of the acinus. It is least well oxygenated and most susceptible to an anoxic injury. It is rich in enzymes involved in glycolysis, lipid and drug metabolism.

Regulation of hepatic circulation

1. Autoregulation. The hepatic arterial blood flow is autoregulated and the portal blood flow is not autoregulated. As described above the hepatic arterial blood flow changes reciprocally with the portal blood flow and that the adenosine is involved in this adjustment.

2. Functional hyperaemia of the intestinal tract after meals is associated with an increased portal blood flow to liver.

3. Neural regulation. The hepatic vessels are innervated by the noradrenergic sympathetic nerve fibres. The liver serves as a blood reservoir, storing about 400 mL of blood in its sinusoids. The sympathetic nerves constrict the presinusoidal resistance vessels in the portal venous system and hepatic arterial system. As described in the neural control of intestinal blood flow, the neural effects on capacitance vessels are more important. Sympathetic stimulation causes a marked reduction in the capacitance of the portal system and other splanchnic capacitance vessels and mobilizes about 1 L of blood towards the heart in less than a minute. In severe shock, hepatic blood flow gets reduced markedly and may produce patchy necrosis of the liver.

APPLIED ASPECTS

Blood supplied by hepatic arteries to the liver does not take part in portal circulation. This blood supplies oxygen and nutrients to the liver cells. Obstruction of the portal vein or its tributaries causes increased blood pressure in portal venous system, a condition known as portal hypertension. This results in enlargement of spleen, oesophageal vein (varices) and formation of haemorrhoids (piles) in the rectum. Haemorrhage from oesophageal varices can be fatal.

**Coronary circulation**

The heart receives its own supply of blood from the coronary arteries. Two major coronary arteries branch off from the aorta near the point where the aorta and the left ventricle meet. These arteries and their branches supply all parts of the heart muscle with blood.

Left Main Coronary Artery (also called the left main trunk). **The left main coronary artery branches into:**

* Circumflex artery
* Left Anterior Descending artery (LAD)

**The left coronary arteries supply:**

* Circumflex artery - supplies blood to the left atrium, side and back of the left ventricle
* Left Anterior Descending artery (LAD) - supplies the front and bottom of the left ventricle and the front of the septum

### Right Coronary Artery (RCA)

**The right coronary artery branches into:**

* Right marginal artery
* Posterior descending artery

**The right coronary artery supplies:**

* Right atrium
* Right ventricle
* Bottom portion of both ventricles and back of the septum

The main portion of the right coronary artery provides blood to the right side of the heart, which pumps blood to the lungs. The rest of the right coronary artery and its main branch, the posterior descending artery, together with the branches of the circumflex artery, run across the surface of the heart's underside, supplying the bottom portion of the left ventricle and back of the septum.

### **What is coronary artery disease?**

Coronary artery disease is the narrowing or blockage of the [coronary arteries](https://my.clevelandclinic.org/health/articles/heart-blood-vessels-coronary-arteries), usually caused by atherosclerosis. Atherosclerosis (sometimes called "hardening" or "clogging" of the arteries) is the buildup of cholesterol and fatty deposits (called plaques) on the inner walls of the arteries. These plaques can restrict blood flow to the heart muscle by physically clogging the artery or by causing abnormal artery tone and function.

Without an adequate blood supply, the heart becomes starved of oxygen and the vital nutrients it needs to work properly. This can cause chest pain called angina. If the blood supply to a portion of the heart muscle is cut off entirely, or if the energy demands of the heart become much greater than its blood supply, a [heart attack](https://my.clevelandclinic.org/health/articles/cad-heart-attack) (injury to the heart muscle) may occur.

### **Who is affected by coronary artery disease?**

Heart disease is the leading cause of death among men and women in the United States. Coronary artery disease affects 16.5 million Americans. The American Heart Association (AHA) estimates that someone in the US has a heart attack about every 40 seconds. In addition, for patients with no risk factors for heart disease, the lifetime risk of having cardiovascular disease is 3.6% for men and less than 1% for women. Having 2 or more risk factors increase the lifetime risk of cardiovascular disease to 37.5% for men and 18.3% in women..

### **What are acute coronary syndromes?**

**Unstable angina:** This may be a new symptom or a change from stable angina. The angina may occur more frequently, occur more easily at rest, feel more severe, or last longer. Although this can often be relieved with oral medications (such as nitroglycerin), it is unstable and may progress to a heart attack. Usually, more intense medical treatment or a procedure is required to treat unstable angina.

**Non-ST segment elevation myocardial infarction (NSTEMI):** This type of heart attack, or MI, does not cause major changes on an electrocardiogram (ECG). However, chemical markers in the blood indicate that damage has occurred to the heart muscle. In NSTEMI, the blockage may be partial or temporary, so the extent of the damage is usually relatively small.

**ST segment elevation myocardial infarction (STEMI):** This type of heart attack, or MI, is caused by a sudden blockage in blood supply. It affects a large area of the heart muscle and causes changes on the ECG as well as in blood levels of key chemical markers.

Although some people have symptoms that indicate they may soon develop acute coronary syndrome, some may have no symptoms until something happens, and still, others have no symptoms of acute coronary syndrome at all.

**All acute coronary syndromes require emergency evaluation and treatment.**

### **Collateral Circulation**

As the size of the blockage in a coronary artery increases, the narrowed coronary artery may develop "**collateral circulation**." Collateral circulation is the development of new blood vessels that reroute blood flow around the blockage. However, during times of increased exertion or stress, the new arteries may not be able to supply enough oxygen-rich blood to the heart muscle.

### **What is ischemia**?

[Ischemia](https://my.clevelandclinic.org/health/diseases/17848-myocardial-ischemia) is a condition described as "cramping of the heart muscle." Ischemia occurs when the narrowed coronary artery reaches a point where it cannot supply enough oxygen-rich blood to meet the heart's needs. The heart muscle becomes "starved" for oxygen-rich blood to meet the heart's needs. The heart muscle becomes "starved" for oxygen.

Ischemia of the heart can be compared to a cramp in the leg. When someone exercises for a very long time, the muscles in the legs cramp up because they're starved for oxygen and nutrients. Your heart, also a muscle, needs oxygen and nutrients to keep working. If the heart muscle's blood supply is inadequate to meet its needs, ischemia occurs, and you may feel chest pain or other symptoms.

Ischemia is most likely to occur when the heart demands extra oxygen. This is most common during exertion (activity), eating, excitement or stress, or exposure to cold.

When ischemia is relieved in less than 10 minutes with rest or medications, you may be told you have "stable coronary artery disease" or "stable angina." Coronary artery disease can progress to a point where ischemia occurs even at rest.

Ischemia and even a heart attack can occur without any warning signs and is called "silent" ischemia. Silent ischemia can occur among all people with heart disease, though it is more common among people with diabetes..

### **What are the risk factors for coronary artery disease?**

**Non-modifiable risk factors** (those that cannot be changed) include:

* **Male gender**. Men have a greater risk of heart attack than women do, and men have heart attacks earlier in life than women. However, beginning at age 70, the risk is equal for men and women.
* **Advanced age**. Coronary artery disease is more likely to occur as you get older, especially after Age 65.
* **Family history of heart disease**. You have an increased risk of developing heart disease if you have a parent with a history of heart disease, especially if they were diagnosed before Age 50. Ask your doctor when it's appropriate for you to start screenings for heart disease so it can be detected and treated early.
* **Race**. African Americans have more severe high blood pressure than Caucasians and, therefore, have a higher risk of heart disease. The risk of heart disease is also higher among Mexican Americans, American Indians, native Hawaiians, and some Asian Americans. This is partly due to higher rates of obesity and diabetes in these populations.

**Modifiable risk factors** (those you can treat or control) include:

* Cigarette **smoking** and exposure to tobacco smoke
* **High blood cholesterol and high triglycerides** – especially high LDL ("bad") cholesterol over 100 mg/dL and low HDL ("good") cholesterol under 40 mg/dL. Some patients who have existing heart or blood vessel disease, and other patients who have a very high risk, should aim for an LDL level less than 70 mg/dL. Your doctor can provide specific guidelines.
* **High blood pressure** (140/90 mmHg or higher)
* Uncontrolled **diabetes** **(HbA1c >7.0)**
* **Physical inactivity**
* Being **overweight** (body mass index [BMI] 25–29 kg/m2) or being obese (BMI higher than 30 kg/m2)
* **NOTE**: How your weight is distributed is important. Your waist measurement is one way to determine fat distribution. Your waist circumference is the measurement of your waist, just above your navel. The risk of cardiovascular disease increases with a waist measurement of over 35 inches in women and over 40 inches in men.
* Uncontrolled **stress or anger**
* [Unhealthy Diet](https://my.clevelandclinic.org/health/articles/mediterranean-diet-heart-health)

The more risk factors you have, the greater your risk of developing coronary artery disease.

### **What causes the coronary arteries to narrow**?



Your coronary arteries are shaped like hollow tubes through which blood can flow freely. The muscular walls of the coronary arteries are normally smooth and elastic and are lined with a layer of cells called the **endothelium**. The endothelium provides a physical barrier between the blood stream and the coronary artery walls, while regulating the function of the artery by releasing chemical signals in response to various stimuli.

Coronary artery disease starts when you are very young. Before your teen years, the blood vessel walls begin to show streaks of fat. As you get older, the fat builds up, causing slight injury to your blood vessel walls. Other substances traveling through your blood stream, such as inflammatory cells, cellular waste products, proteins and calcium begin to stick to the vessel walls. The fat and other substances combine to form a material called **plaque.**

Over time, the inside of the arteries develop plaques of different sizes. Many of the plaque deposits are soft on the inside with a hard fibrous "cap" covering the outside. If the hard surface cracks or tears, the soft, fatty inside is exposed. Platelets (disc-shaped particles in the blood that aid clotting) come to the area, and blood clots form around the plaque. The endothelium can also become irritated and fail to function properly, causing the muscular artery to squeeze at inappropriate times. This causes the artery to narrow even more.

Sometimes, the blood clot breaks apart, and blood supply is restored. In other cases, the blood clot (**coronary thrombus**) may suddenly block the blood supply to the heart muscle (**coronary occlusion**), causing one of three serious conditions, called **acute coronary syndromes**.

Bottom of Form

**Cutaneous circulation**

The skin is the outer covering of the body and as such serves as the first line of defense against invasion by disease-causing organisms. The skin, as the interface between the internal and external environments, also helps to maintain a constant deep body temperature despite changes in the ambient (external) temperature—a process called thermoregulation. The thinness and large area of the skin make it an effective radiator of heat when the body temperature rises above the ambient temperature. The transfer of heat from the body to the external environment is aided by the flow of warm blood through capillary loops near the surface of the skin. Blood flow through the skin is adjusted to maintain deep body temperature at about 37 8 C (98.6 8 F). These adjustments are made by variations in the degree of constriction or dilation of ordinary arterioles and of unique arteriovenous anastomoses. These latter vessels, found predominantly in the fingertips, palms of the hands, toes, soles of the feet, ears, nose, and lips, shunt (divert) blood directly from arterioles to deep venules, thus bypassing superficial capillary loops. Both the ordinary arterioles and the arteriovenous anastomoses are innervated by sympathetic nerve fibers. When the ambient temperature is low, sympathetic nerves stimulate cutaneous vasoconstriction. Cutaneous blood flow is thus decreased, so that less heat will be lost from the body. Because the arteriovenous anastomoses also constrict, the skin may appear rosy because the blood is diverted to the superficial capillary loops. In spite of this rosy appearance, however, the total cutaneous blood flow and rate of heat loss is lower than under usual conditions. Skin can tolerate an extremely low blood flow in cold weather because its metabolic rate decreases when the ambient temperature decreases. In cold weather, therefore, the skin requires less blood. As a result of exposure to extreme cold, however, blood flow to the skin can be so severely restricted that the tissue dies; a condition known as **frostbite.** Blood flow to the skin can vary from less than 20 ml per minute at maximal vasoconstriction to as much as 3 to 4 L per minute at maximal vasodilation. As the temperature warms, cutaneous arterioles in the hands and feet dilate as a result of decreased sympathetic nerve activity. Continued warming causes dilation of arterioles in other areas of the skin. If the resulting increase in cutaneous blood flow is not sufficient to cool the body, sweat gland secretion may be stimulated. Perspiration helps cool the body as it evaporates from the surface of the skin. The sweat glands also secrete bradykinin, a polypeptide that stimulates vasodilation. In usual ambient temperatures, the cutaneous vascular resistance is high and the blood flow is low when a person is not exercising. In the pre-exercise state of fight or flight, sympathetic nerve activity reduces cutaneous blood flow still further. During exercise, however, the need to maintain a deep body temperature takes precedence over the need to maintain an adequate systemic blood pressure. As the body temperature rises during exercise, vasodilation in cutaneous vessels occurs together with vasodilation in the exercising muscles. This can cause an even greater lowering of total peripheral resistance during exercise.

 **Discuss the cardiovascular adjustment that occurs during exercise**

Both breathing and pulse rate increase within one second of exercise, suggesting that the motor cortex responsible for originating the exercise also influences the cardiovascular adjustments to exercise. However, cardiovascular changes during exercise are also affected by sensory feedback from the contracting muscles and the baroreceptor reflexes. These mechanisms increase the activity of the sympathoadrenal system and reduce parasympathetic nerve activity during exercise. As a result, there is an increase in cardiac rate, stroke volume, and cardiac output. During exercise, the cardiac output can increase fivefold— from about 5 L per minute to about 25 L per minute. This is primarily due to an increase in cardiac rate. The cardiac rate, however, can increase only up to a maximum value, which is determined mainly by a person’s age. In well-trained athletes, the stroke volume can also increase significantly, allowing these individuals to achieve cardiac outputs during strenuous exercise up to six or seven times greater than their resting values. This high cardiac output results in increased oxygen delivery to the exercising muscles; this is the major reason for the much higher than average maximal oxygen uptake of elite athletes. In most people, the increase in stroke volume that occurs during exercise will not exceed 35%. The fact that the stroke volume can increase at all during exercise may at first be surprising, given that the heart has less time to fill with blood between beats when it is pumping faster. Despite the faster beat, however, the end-diastolic volume during exercise is not decreased. This is because the venous return is aided by the improved action of the skeletal muscle pumps and by increased respiratory movements of the diaphragm during exercise. The enddiastolic volume is not significantly changed during exercise, so any increase in stroke volume that occurs must be due to an increase in the proportion of blood ejected per stroke.

 The proportion of the end-diastolic volume ejected per stroke can increase from 60% at rest to as much as 90% during heavy exercise. This increased ejection fraction is produced by the increased contractility that results from sympathoadrenal stimulation. There also may be a decrease in total peripheral resistance as a result of vasodilation in the exercising skeletal muscles, which decreases the afterload and thus further augments the increase in stroke volume. Endurance training often results in a lowering of the resting cardiac rate and an increase in the resting stroke volume. The lowering of the resting cardiac rate results from a greater inhibition of the SA node by the vagus nerve. The increased resting stroke volume is believed to be due to an increase in blood volume; indeed, studies have shown that the blood volume can increase by about 500 ml after only eight days of training. These adaptations enable the trained athlete to produce a larger proportionate increase in cardiac output and achieve a higher absolute cardiac output during exercise. This large cardiac output is the major reason for the improved oxygen delivery to skeletal muscles that occurs as a result of endurance training.

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| Variable | Change  | Mechanism |
| Cardiac output | Increased | Increased cardiac rate and stroke volume |
| Cardiac rate | Increased | Increased sympathetic nerve activity and decreased activity of the vagus nerve |
| Stroke volume | Increased | Increased myocardial contractility due to stimulation by sympathoadrenal system; decreased total peripheral resistance |
| Totalperipheral resistance | Decreased | Vasodilation of arterioles in skeletal muscles (and in skin when thermoregulatory adjustments are needed) |
| Arterial blood pressure | Increased | Increased systolic and pulse pressure due primarily to increased cardiac output; diastolic pressure rises less due to decreased total peripheral resistance |
| End-diastolic volume | Unchanged | Decreased filling time at high cardiac rates is compensated for by increased venous pressure, increased activity of the skeletal muscle pump, and decreased intrathoracic pressure aiding the venous return |
| Blood flow to heart and muscles | Increased | Increased muscle metabolism produces intrinsic vasodilation; aided by increased cardiac output and increased vascular resistance in visceral organs |
| Blood flow to visceral organs | Decreased | Increased muscle metabolism produces intrinsic vasodilation; aided by increased cardiac output and increased vascular resistance in visceral organs |
| Blood flow to skin | Increased | Metabolic heat produced by exercising muscles produces reflex (involving hypothalamus) that reduces sympathetic constriction of arteriovenous shunts and arterioles |
| Blood flow to brain | Unchanged | Metabolic heat produced by exercising muscles produces reflex (involving hypothalamus) that reduces sympathetic constriction of arteriovenous shunts and arterioles |