Ambulatory Blood Pressure (ABP) Regulation

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The regulation of arterial blood pressure is essential in order to maintain perfusion to the vital organs. While a drop in blood pressure is dangerous as no blood flows to the brain, at the same time a high blood pressure may result in various unfavorable outcomes such as cerebrovascular accidents and myocardial infarction.

Regulation of Arterial Blood Pressure

The arterial blood pressure is regulated by a variety of feedback mechanisms. These **mechanisms operate through specific receptors present in the blood vessels and the heart,** which, in turn, send signals to the specific areas of the brain, responsible for blood pressure control. After the interpretation of the signals, the brain sends impulses via the components of the **autonomic nervous system**.

A rapidly acting mechanism for regulation of blood pressure

Mostly the nervous control mechanism:

- Baroreceptor feedback mechanism
- Central nervous system ischemic mechanism
- Chemoreceptor mechanism

The intermediate-acting mechanism for control of blood pressure:

- Renin-angiotensin vasoconstrictor mechanism
- Stress relaxation of the vasculature
- Fluid shift across the capillary for adjustment of blood volume

The long-term mechanism for control of blood pressure:

- Renal blood volume pressure control mechanism

Autonomic Nervous System

The Autonomic Nervous System consists of the sympathetic and the parasympathetic nervous system. While the sympathetic stimulation plays a major role in the regulation of arterial blood pressure, the parasympathetic system is equally essential in the regulation of cardiac activity.

Sympathetic nervous system

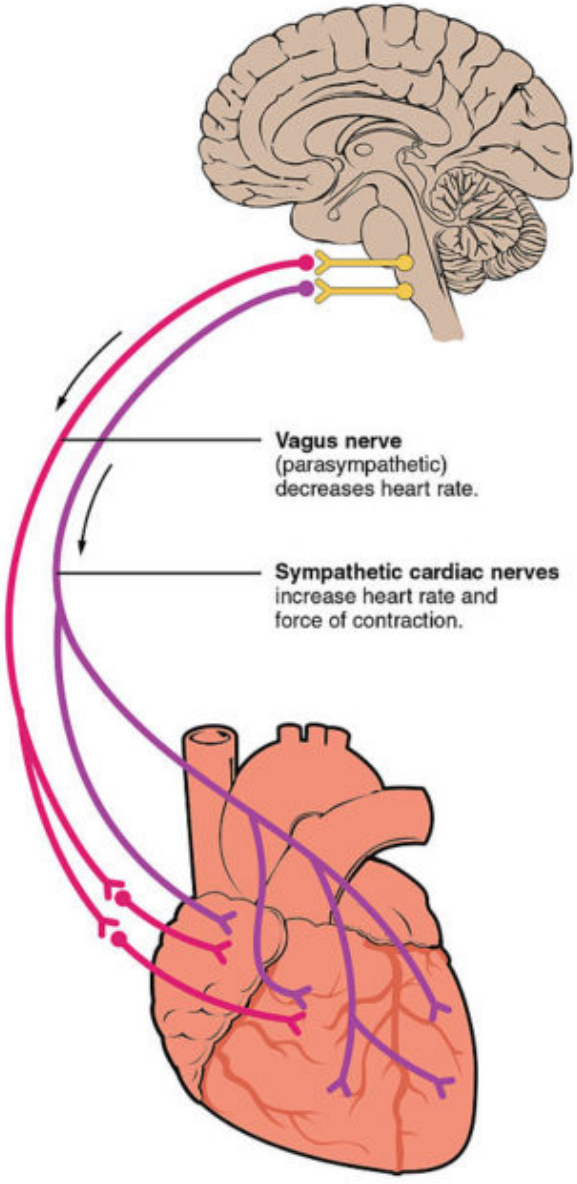


Image: “Autonomic innervation of the heart” by OpenStax College. License: CC BY-SA 3.0

The vasomotor fibers of the sympathetic nervous system arise from the thoracic and the

first two lumbar spinal nerves. They pass through the sympathetic chain and innervate the vasculature, heart, and viscera through specific sympathetic nerves. In addition, they also innervate the peripheral vasculature through the spinal nerves.

The **sympathetic innervation of the small arteries and arterioles causes an increase in peripheral resistance and a rise in arterial blood pressure**. In veins, when sympathetic stimulation causes vasoconstriction, the volume of blood in the veins decreases and the venous return to the heart increases. In the heart, it causes an increase in heart rate and force of cardiac contractility, which increases the stroke volume. The overall result is the rise in cardiac output.

The **sympathetic fibers are mainly vasoconstrictor in nature**, however, a few vasodilator fibers are also present, depending upon the type of organ and tissues. The neurotransmitter which is secreted by the vasoconstrictor fibers of the sympathetic nerves is norepinephrine, which acts on the alpha receptors of the vascular smooth muscles to cause vasoconstriction.

When the sympathetic vasoconstrictor signals are sent to the vascular smooth muscles, the **impulses are also transmitted to the adrenal medulla**. The adrenal medulla secretes epinephrine and norepinephrine. While norepinephrine only causes vasoconstriction, epinephrine can sometimes cause vasodilation by the activation of beta receptors.

Parasympathetic nervous system

As discussed earlier, the parasympathetic fibers are important in controlling the heart rate. The **vagus nerve** carries the parasympathetic fibers to the heart. Its stimulation causes a decrease in heart rate as well as the cardiac contractility.

Vasomotor Center

The vasomotor center is **located bilaterally in the reticular substance of the medulla and the lower one-third of the pons**. The impulses are transmitted to the autonomic nerve fibers through the **spinal cord**.

The vasomotor area is further subdivided into the following regions:

- **The vasoconstrictor area**, also known as C1, distributes its fibers throughout the cord. The neurons in these fibers release norepinephrine and activate the vasoconstrictor fibers of the sympathetic nervous system. Under normal conditions, this area continuously stimulates these fibers at a rate of 1—2 impulses per second. This continuous firing is called sympathetic vasoconstrictor tone and the partial state of constriction of the blood vessels is called the vasomotor tone.
- **The vasodilator area**, also referred to as A1, sends inhibitory signals to the vasoconstrictor area, hence causing vasodilation.
- **The sensory area**, also called A2, receives sensory signals from the glossopharyngeal and the vagus nerves. The output signals control the activities of the vasoconstrictor and the vasodilator area.

The lateral portion of the vasomotor center also controls the cardiac activity by sending excitatory impulses to the sympathetic fibers, while the medial portion sends signals via the vagus nerve, thereby decreasing cardiac activity.

Control of vasomotor center by higher nervous centers

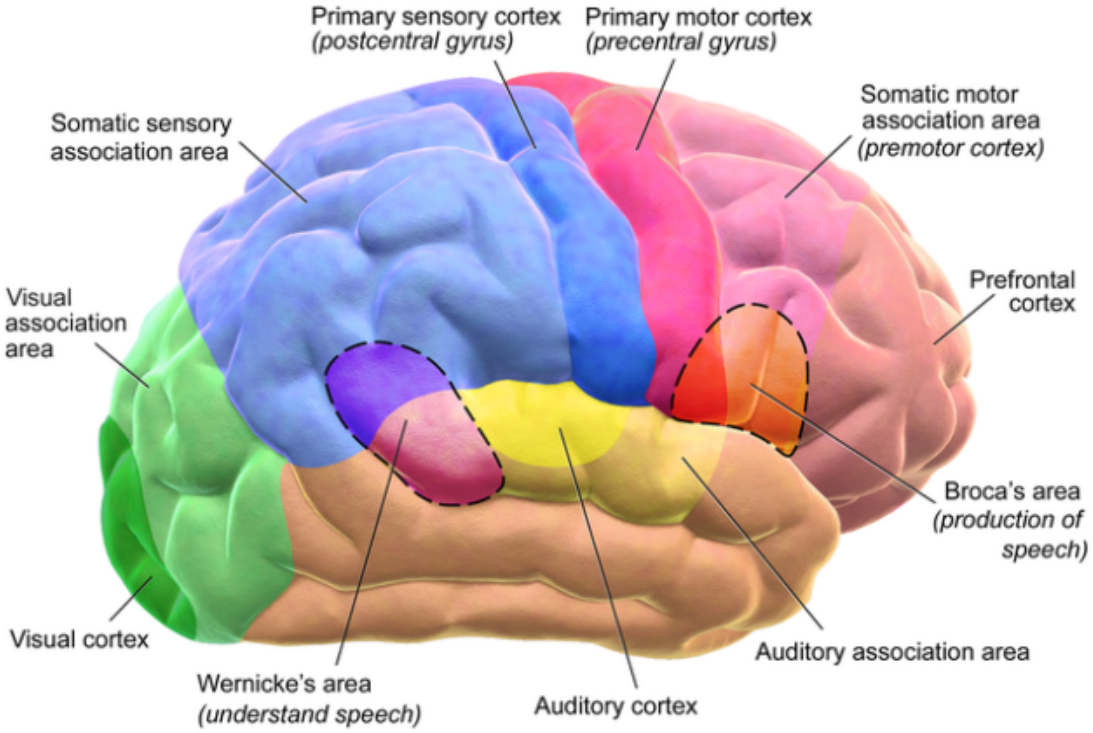


Image: "Functional areas of the human brain" by BruceBlaus. License: CC BY-SA 3.0

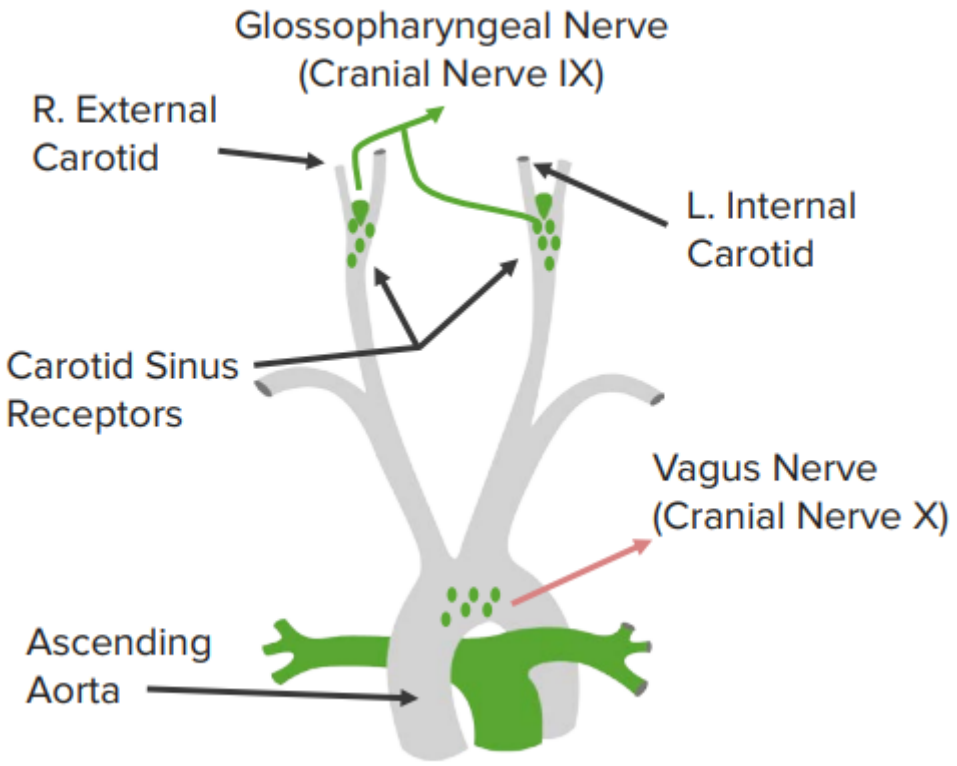
The vasomotor center is under control of higher nervous centers:

1. **The reticular substance** of the pons, mesencephalon, and diencephalon can either excite or inhibit the vasomotor center.
2. **The posterolateral portion** of the hypothalamus causes excitation of the vasomotor center while the anterior portion can either cause excitation or inhibition.
3. **Different parts of the cerebral cortex** such as the motor cortex, anterior temporal lobe, orbital areas of the frontal cortex, the amygdala, the anterior part of the cingulate gyrus, the septum and the gyrus can either excite or inhibit the vasomotor center.

Reflex Mechanisms for Maintaining Normal Arterial Pressure

Multiple subconscious reflex mechanisms are present in our body which keeps the arterial pressure under control. Almost all of them work by the negative feedback mechanism. They are as follows:

The Baroreceptor Reflex



"Feedback Regulation of Autonomic Nerves by Arterial Baroreceptors" by Lecturio.

The baroreceptor reflex, also known as the pressure buffer system, is the most important reflex mechanism for the control of rapid changes in the arterial pressure.

Note: The baroreceptors, also known as the stretch receptors, are present in the wall of great arteries. They are extremely abundant in the wall of the internal carotid artery, above the level of bifurcation, where this area is known as the carotid sinus. In addition, the wall of the aortic arch also consists of a large number of baroreceptors.
The **carotid sinus baroreceptors are less sensitive to the pressure range of 0—60 mmHg**. However, they are highly stimulated by pressures of 60 mmHg and above. The aortic baroreceptors are sensitive to pressures above 30 mmHg. Because the baroreceptor system opposes either increases or decreases in arterial pressure, it is called a *pressure buffer system* and the nerves from the baroreceptors are called *buffer nerves.*

The long-term regulation of mean arterial pressure by the baroreceptors requires interaction with additional systems, principally the renal-body fluid-pressure control system (along with its associated nervous and hormonal mechanisms)

Once stimulated, the **baroreceptors send signals through the Hering's nerve to the glossopharyngeal nerve**, which finally reaches the sensory area of the vasomotor area of the brain. The response is the inhibition of the vasoconstrictor area and the excitation of the vagal nerve. This leads to the vasodilation of the arterioles and veins, decreased heart rate and the force of cardiac contractility.

When the arterial blood pressure goes back to the normal limits, the baroreceptors stop sending signals and the reflex response is stopped by the negative feedback mechanism. The **baroreceptors respond very rapidly to the changing arterial pressure**. The rate of impulse transmission is greater when the pressure is increasing such as the

systole and decreases when the pressure is dropping as in the diastolic phase.

Reduction in arterial blood pressure

A sudden change in posture, for example, when a person stands up from the supine or seated position, may result in a drop in arterial pressure. The **baroreceptors sense this reduction in arterial pressure immediately, which leads to activation of the sympathetic system**, thus causing vasoconstriction. The vasoconstriction increases the arterial pressure and prevents sudden loss of consciousness due to low arterial pressure (one of the causes of **syncope**).

The baroreceptor system becomes non-functional under long-term pressure changes. This is because the baroreceptors stop the transmission of impulses despite an increase in the arterial blood pressure. Within only 1—2 days, they reset themselves to the current pressure.

The Chemoreceptor Reflex

Just like the baroreceptors, chemoreceptors are also present in the wall of the carotid arteries and the arch of the aorta where they form the carotid and the aortic bodies respectively.

Unlike the baroreceptors, the chemoreceptors are **sensitive to the change in concentration of oxygen, carbon dioxide and hydrogen ions** in the arterial blood. The carotid and aortic bodies are supplied with a nutrient artery. If the arterial pressure drops, less blood is supplied to them and therefore they sense lack of oxygen and build-up of carbon dioxide and hydrogen ions in the blood.

The nerve fibers excited by the chemoreceptors follow the same pathway as that of the baroreceptor reflex. However, the **response, in this case, is the activation of the vasoconstrictor pathway**.

The chemoreceptor reflex is not a very potent mechanism in controlling the arterial pressure as the chemoreceptors are not activated unless the arterial pressure falls below 80mmHg.

Volume reflex

When the walls of the atria are stretched, it causes dilatation of the peripheral arterioles. They do detect simultaneous increases in pressure in the low-pressure areas of the circulation caused by an increase in volume, and they elicit reflexes parallel to the baroreceptor reflexes to make the total reflex system more potent for control of arterial pressure. This response is more potent in the afferent arterioles of the **kidney**. The glomerular pressure rises, causing more filtration of fluid and production of more urine. **Signals are simultaneously sent to the hypothalamus** to stop the release of antidiuretic hormone (ADH). Thus less water is reabsorbed from the kidneys causing arterial pressure to come back to the normal range.

Abdominal compression reflex

Whenever the vasoconstrictor area of the brain is stimulated to send excitatory signals to the sympathetic nerves, the brain stem simultaneously sends impulses to the skeletal muscles of the body, particularly the abdominal muscles. The basal tone of the abdominal muscles increases which causes compression of the veins. This increases the venous

return to the heart, providing more blood to be pumped by the ventricles.

Central nervous system ischemic response

When the arterial pressure decreases below 60 mmHg, there is reduced blood flow to the vasomotor area which may lead to cerebral ischemia.

The neurons in the vasomotor area are strongly excited causing a potent excitation of the sympathetic nervous system. **Carbon dioxide is thought to be a major culprit** since decreased blood flow to the vasomotor areas will not flush away the carbon dioxide produced. Other substances such as lactic acid and other acidic substances also contribute to the production of such a response.

The sympathetic excitation, in this case, is so great that it causes full occlusion of the peripheral arterioles. The **response may last for as long as 10 minutes and the rise in arterial pressure can be as high as 250 mmHg**. The highest degree of response is produced when the arterial pressure falls to 20 mmHg. The central nervous system ischemic response is therefore sometimes called the ‘last ditch stand’.

The Renin-Angiotensin System: Its Role in Arterial Pressure Control

When the arterial pressure falls, intrinsic reactions in the kidneys themselves cause many of the pro-renin molecules in the JG cells to split and release renin. Most of the renin enters the renal blood and then passes out of the kidneys to circulate throughout the entire body.

During its persistence in the blood, angiotensin II has two principal effects that can elevate arterial pressure.

1. The first of these, *vasoconstriction in many areas of the body,* occurs rapidly. Vasoconstriction occurs intensely in the arterioles and much less so in the veins. Constriction of the arterioles increases the total peripheral resistance, thereby raising the arterial pressure.
2. The second principal means by which angiotensin II increases the arterial pressure is to *decrease excretion of both salt and water* by the kidneys. This long-term effect, acting through the extracellular fluid volume mechanism, is even more powerful than the acute vasoconstrictor mechanism in eventually raising the arterial pressure.

Angiotensin II causes the kidneys to retain both salt and water in two major ways:

- Angiotensin II acts directly on the kidneys to cause salt and water retention.
- Angiotensin II causes the adrenal glands to secrete aldosterone, and the aldosterone, in turn, increases salt and water reabsorption by the kidney tubules.

Thus both the direct effect of angiotensin on the kidney and its effect acting through aldosterone are important in long-term arterial pressure control.

Ambulatory Blood Pressure Monitoring

The measurement of the arterial blood pressure at regular intervals for a minimum

duration of 24 hours is called ambulatory blood pressure measurement. A **digital monitor is attached to the waist through an abdominal belt and the cuff is tied around the arm**. The patient is asked to move around and perform their daily routine tasks. Nocturnal blood pressure is usually 10% lower than daytime blood pressure. The disappearance of the decline in blood pressure at night less than 10 % from daytime is suggestive of increased cardiovascular risks and renal insufficiency.

The significance of monitoring ambulatory blood pressure is:

- **To avoid the effect of white coat hypertension**: some patients become anxious and panic whenever their blood pressure is measured.
- **To follow the variation in blood pressure during the day** and risk assessment of the **coronary heart disease**. It is also recommended before initiation of an anti-hypertensive drug.
- **To see the response to medications** if a patient is on multiple anti-hypertensive drugs.

Review Questions

The correct answers can be found below the references.

1. A patient was brought to the ER in an unconscious state and cold peripheries. On examination, pulses were barely palpable, HR 140/min, RR 20/min, temp 97 F and BP 55 systolic on the cardiac monitor. Baroreceptors of which area in the body are more sensitive to such low blood pressure?

A. Aortic arch
B. Carotid sinus
C. Thoracic duct
D. AV node
E. SA node

2. A trauma patient was brought in the surgical emergency after a road accident. He has been bleeding due to head wounds for the last hour. On examination, pulses were barely palpable, HR 110/min, RR 40 /min, temp 98 F and BP 60 systolic. Which of the following changes in the blood of this patient stimulated the chemoreceptor reflex of the body to such blood pressure?

A. Lack of oxygen
B. Excess of CO_2
C. Excessive accumulation of H^+
D. All of these
E. None of these

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Correct answers: 1A, 2D

Notes