

Regulation of blood pressure

Definition of blood pressure

Blood pressure is the pressure exerted by the blood on the vessel wall and the heart. We usually mean the blood pressure in the arteries of the systemic volume. We recognize systolic (100-140 mmHg) and diastolic (60-90 mmHg) pressure. 1 mmHg can then be expressed as 0.133 kPa.

Blood pressure can be explained by several equations:

- $P = T / r$ - pressure is directly proportional to the tension of the vessel wall and inversely proportional to the radius of the vessel (Laplace's law).
- $P = Q \times R$ - pressure is directly proportional to blood flow and vascular resistance.

Pressure amplitude

calculated by subtracting the diastolic pressure from the systolic pressure. It is usually something around 50 mmHg.

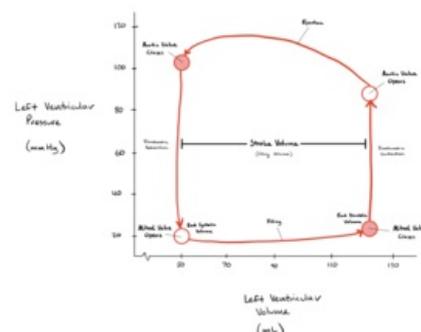
Mean arterial pressure

(MAP = mean arterial pressure) is the pressure averaged over time: $MAP = \text{diastolic pressure} + 1/3 \text{ of the pressure amplitude}$.

Blood pressure can be influenced by cardiac output, total vascular resistance, central venous pressure and blood volume.

Pressure changes during cardiac revolution

During the chamber filling phase (diastole), the volume of the chamber increases and the pressure increases by passive stretching of the walls (pressure increase is small, however). At the end of diastole, there is a so-called end-diastolic pressure in the chamber, which is less than 10 mmHg and is higher in the left ventricle (therefore it must have stronger muscle). At the MZ point, the atrioventricular valve closes as soon as the pressure in the ventricles exceeds the pressure in the atria. Next, an isometric contraction begins, during which the pressure in the chambers increases without changes in volume. At point AO, the semilunar valves open as soon as the pressure in the ventricles exceeds the pressure in the aorta and pulmonary artery. Next, the ejection phase occurs, during which blood is ejected into the aorta and lung. At point AZ, the semilunar valves close, and the pressure decreases further at constant volume.



Left ventricle pressure-volume diagram

Variations in the blood stream

Small arteries and arterioles account for up to 50% of the total peripheral resistance, while capillaries account for only around 25%. The pressure in the veins is very low, for that reason they serve as a reservoir for the blood, from which the blood is pumped to the whole organism if necessary.

Blood pressure regulation

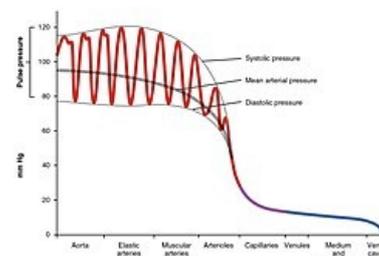
2 concepts are important for pressure regulation:

Vasoconstriction

In it, vasoconstrictor substances bind to the receptor and cause an increase in intracellular Ca^{2+} via the G-protein. This will result in depolarization of the membrane - entry of Ca^{2+} through L-type voltage-gated channels and at the same time second messengers will release Ca^{2+} from the sarcoplasmic reticulum. This will lead to a contraction of the smooth muscle in the vessel wall (greater force of contraction). Vasoconstrictor substances are, for example, endothelin 1, thromboxane A2, angiotensin II, noradrenaline (sympathetic).

Vasodilation

It removes Ca^{2+} , which goes out of the cell by Ca^{2+} -ATPase = PMCA and Ca^{2+} - Na^{+} exchange and also back into the ER (Ca^{2+} ATPase = SERCA). Vasodilators increase the level of guanosine monophosphate (cGMP) or adenosine monophosphate (cAMP), which through protein kinases stimulate SERCA and PMCA, so that the intracellular Ca^{2+}



Systemic Blood Pressure

content decreases and, in addition, the voltage-gated Ca^{2+} channel is inhibited (drugs verapamil and dihydropyridine). Vasodilating agents include nitric oxide and prostacyclin .

Acute regulation of blood pressure - baroreceptor reflex

The baroreceptors of the high pressure system are in the aorta and carotid sinus.

This system works by lowering the pressure in the arterial wall and reducing the activity of the baroreceptors. This will increase sympathetic nerve activity, which will increase heart rate and contractility, peripheral vasoconstriction and venoconstriction (eg, when the body is upright). This process will ensure that the pressure increases again.

More detailed information can be found on the Baroreflex page .

Long-term regulation of blood pressure

The amount of fluid excreted by the kidneys

If there is an increase in pressure, the filtration pressure in the kidneys will increase . This will increase the volume of urine, which will decrease the volume of extracellular fluid, i.e. blood. This will reduce the cardiac output.

Antidiuretic hormone

Vasoconstriction increases reabsorption of water in the glomeruli , thereby increasing blood volume and increasing venous return.

Aldosterone

It is produced by the adrenal cortex. Increases reabsorption of Na^+ and water. This increases blood volume and venous return . Thus, the cardiac output is increased and, in addition, the sensitivity to angiotensin II increases, thus the efficiency of the renin-angiotensin system increases .

The renin-angiotensin system

Renin is secreted by the kidney when blood flow decreases (sympathetic effect). It cleaves from alpha 2 globulin the decapeptide angiotensin I , which has no significant activity, but is converted in the lungs by angiotensin-converting enzyme to angiotensin II .

Angiotensin II has two main functions:

- vasoconstriction of arterioles ,
- water retention and Na^+ - shift of the functional renal curve.

The renin — angiotensin system therefore increases blood volume (and hence minute output) as well as peripheral resistance.

Local blood pressure management

Local control of blood pressure is the ability of tissues to independently regulate blood flow.

Autoregulation

Autoregulation is the ability to maintain constant flow within a pressure range of 50-170 mmHg through the myogenic response. Arterioles will constrict in response to vessel wall tension (activation of stretch-activated Ca^{2+} channels) via locally produced vasodilator factors (increased flow will dilute them, resulting in vasoconstriction).

Metabolic factors

The response to hyperemia (increased blood flow) that is caused by K^+ , CO_2 , and adenosine is vasodilation .

Topical hormones

During inflammation, histamine and bradykinin are released , which cause vasodilation and increase the permeability of blood vessels. As a result, swelling occurs. Leukocytes and antibodies penetrate into damaged tissues, increasing the activity of NO production (vasodilator).

Links

Related articles

- Blood pressure
- Blood pressure measurement
- Blood pressure monitoring

- Home blood pressure monitoring

Sources

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