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**Bestaev G.G., Slepushkin V.D.**

**CLINICAL PHYSIOLOGY OF THE CIRCULATION**

**(Tutorial)**

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**CLINICAL PHYSIOLOGY OF BLOOD CIRCULATION**

**(Tutorial)**



**Владикавказ**

**The authors:**

*Bestaev Georgy Givievich* —Associate Professor of the Department, Candidate of Medical Sciences

*Slepushkin Vitaly Dmitrievich*- Head. department, doctor of medical sciences, professor

**English translation:**

*Shulga Elena Vitalievna*- Candidate of Philological Sciences, Associate Professor

**Reviewer:**

*Lebedeva Elena Anatolievna* —doctor of medical sciences, professor of Rostov medical university

**Authors:**

*George Bestaev*-dozent

*Vitaly Slepushkin*-Head of the Department, Doctor of Medical Sciences, Professor

**The English translation:**

Elena Shulga – candidate of Philological Sciences, Associate Professor

**Review:**

*Elena Lebedeva* -professor of Rostov Medical University

The manual is intended for senior students, including foreign students studying in English, residents, graduate students.

## CLINICAL PHYSIOLOGY OF THE CIRCULATION

The circulatory system is a closed hydrodynamic system filled with a heterogeneous fluid and driven by a pump - the heart.

The main tasks of blood circulation are:

1. Delivery to tissues: oxygen, nutrients and salts, hormones and other active substances;
2. Removal from tissues: carbon dioxide and other end products metabolism;
3. Participation in heat transfer.

Functional divisions of the cardiovascular system

In the functional classification of the Swedish physiologist B. Folkov (1976 d.) provides for the division of the circulatory system into "links or elements connected in series":

one. *Heart* A pump that pumps blood rhythmically into the arteries.

2. *Elastically tensile vessels*, which transform the periodic ejection of blood from the heart into a uniform blood flow (aorta with its departments, pulmonary artery).

3. *Resistive vessels* (vessels of resistance) - precapillary (in mainly arterioles) and post-capillary sections (venules), which together create a general resistance to blood flow in the vessels of organs.

four. *Precapillary sphincters*— specialized department the smallest arterial vessels, contraction of the smooth muscle cells of these sphincters can lead to blockage of the lumen of small vessels. These vessels regulate the amount of blood flow in the capillary bed.

5. *exchange vessels*, or true capillaries, where blood contacts with tissue due to the huge surfaces of the capillary bed. Here the main function of the cardiovascular system is realized - the exchange between blood and tissues.

6. *Shunt vessels* (arteriovenous anastomoses), the presence which have not been proven for all tissues.

7. *capacitive vessels*, in which changes in clearance, even so small, which do not have a significant impact on the overall

resistance, cause pronounced changes in the distribution of blood and the magnitude of its inflow to the heart - the venous section of the system.

The circulatory system can be conditionally divided into a number of components. The functional divisions of the circulatory system are classified as follows:

1. Macrocirculation:

- heart pump;
- vessels-buffers (arteries);
- vessels - containers (veins).

2. Microcirculation:

- vessels of resistance or distribution (arterioles and venules);
- exchange vessels (capillaries);
- vessel shunts (arteriovenous anastomoses).

1. Blood: volume and rheology

Blood is a liquid medium that not only transports respiratory gases, energy, plastic and regulatory substrates in the body, but also acts as a coolant between the core of the body and its shell. Being a suspension of cells, it is the so-called. a typical non-Newtonian fluid whose viscosity is directly related to the applied shear stress. With regard to hemodynamics, the main characteristics of blood are its active volume (volume of circulating blood, abbr. BCC) and rheological, i.e. viscous flow properties. At normal values of blood flow velocity, the ratio of plasma volumes and formed elements in the contents of the vessel is set by its diameter. Consequently, the hematocrit of the vascular system will already depend on the structure of this vascular bed - the length and specific gravity of vessels of various diameters.

2. Vascular bed

The state of the vessels and, in particular, the systemic vascular tone, are of interest to us in two aspects. On the one hand, the predominantly neurogenic tone of venules and small veins forms the very vascular capacity, which normally contains up to 75–80% of the BCC and allows physiologists to call the veins of the systemic circulation, together with the pulmonary circulation, a system of high volume and low pressure. On the other hand, the tone of arterioles, which depends to a greater extent on local metabolic influences, determines the magnitude of systemic vascular resistance.

### 3. Cardiac output and its determinants

The minute volume of blood circulation (MOV, minute volume of the heart, cardiac output, cardiac output - the terms are equivalent) is formed as the sum of stroke volumes of blood (SVK, one-time cardiac outputs) per minute. Thus, CO is a derivative of stroke volume and heart rate (HR). The unit of measure for CB is l/min.

The stroke volume of the blood, in turn, is determined by the difference in the phase volumes of the ventricles at the end of diastole (end-diastolic volume, EDV) and the end of systole (end-systolic volume, ESV).

The normal values of the IOC are 4 - 6 liters. In order to neutralize the influence of body size and make the indicator more comparable, A. Grollman proposed a cardiac index (CI) - the ratio of the IOC to the body surface area (normally  $2.8 \pm 4.2 \text{ l} / \text{m}^2/\text{min}$ ).

End diastolic volume (EDV) represents yourself consistent result of (a) infusion of blood under the action of filling pressure (central venous pressure for the right, pulmonary vein pressure for the left heart) into the ventricle with a given compliance and b) active "recharging" of the ventricle by atrial systole. The diastolic distensibility of the ventricle depends on its passive mechanical rigidity; there is a term compliance (compliance - English) of the ventricle.

The filling pressure of the ventricle characterizes venous return. In the strict sense, the latter indicator has the dimension of the flow and for the right ventricle depends on: a) the ratio of the volume of circulating blood and the tone of the venous (capacitive) section of the vascular bed, which normally contains 60-80% of the BCC, b) the pressure gradient between the peripheral veins and the right atrium and c) end-systolic volume (ESV) values at which diastole begins.

With an increasing EDV and a stretching muscle fiber, the contraction energy and stroke volume also increase to the point of overstretch, beyond which the stroke volume begins to decrease, as, for example, in heart failure. Increased preload leads to an increase in CO. Preload depends on the return of venous blood, which is influenced by body position, intrathoracic pressure, circulating blood volume and vascular tone of the venous system.

The influence of the CSR is realized through the magnitude of the passive tension of the ventricular wall at the beginning of diastole, which, according to the P.-S. Laplace (1807), *ceteris paribus*, is directly proportional to the cube root of the KSO. Thus, closing the circle, CSR characterizes the adequacy of the systolic function to the applied preload. It is important that the system

venous return - "the fulcrum of blood circulation" (A. Guyton, 1973) - is directed to the right heart, while systemic perfusion is determined by the left. Depending on the rhythm, EDV is determined either by the sum of a) the volume at which the elastic recoil of the ventricle is balanced with the filling pressure and b) the systolic volume of the atrium, or the sum of the same systolic volume of the atrium (b) with the volume at which diastole ended due to the onset of atrial systole (a). The latter situation characterizes a more frequent rhythm, and in the limit is the mechanism of the fall in cardiac output during tachycardia. The implementation of one of the two described options depends on whether the diastolic time is sufficient to balance the ventricular compliance with the filling pressure (when the flow becomes zero on its own), or the next atrial systole begins at non-zero flow through the atrioventricular valves. The decisive input parameters are: a) filling pressure, b) ventricular compliance and c) duration of diastole, i.e. the difference between the duration of the cardiocycle (the period of the heart rhythm) and the duration of the mechanical systole.

The duration of the ejection period, in turn, is determined by the ratio of contractility (inotropic status) and afterload, which determines the pressure in the ventricular outflow tract against which myocardial fibers perform their mechanical work. The inotropic status, further, depends on: a) the length of the fibers (myofibrils) at the beginning of the contraction process and b) the sum of other factors affecting their functional state.

The initial length of myofibrils, which depends on their stretching by the end of diastole, reflects one of the modern interpretations of the concept *preload*: *ceteris paribus*, it is she who determines the size of the active contact surface of the actin and myosin sarcomere filaments in the starting conditions of contraction.

The ejection fraction ( $EF = SVK/EDV$ ), as it is clear from the above, characterizes not contractility as such, but its adequacy to the current values of pre- and afterload. Thus, ESR is the result of a dynamic balance between the tension of the contracting myocardium and the pressure it creates in the outflow tract.

Today, the end-systolic tension of the ventricular wall, which has the dimension of pressure, is considered an accurate measure of afterload. The pressure in the outflow tract, which also characterizes the afterload of the ventricle, is determined by the rate of blood ejection into systole and the hydrodynamic resistance of the vascular system. To characterize the latter, indicators of general vascular resistance are introduced; peripheral (OPSS) - for a large circle of blood circulation and pulmonary (OLSS) - for a small one.

Since the growth of body dimensions, other things being equal, increases the total lumen of the vessels, the indices are calculated not as a ratio, but as the product of the resistance values by the surface area of the body or, which gives the same result as the ratio of the pressure difference to the flow, referred to the surface area ( i.e. replacing the IOC-1 multiplier with SI-1).

The normal values of OPSS are 900–1500 dyn·s·cm<sup>-5</sup>; the norm for OLSS is 25–150 dyn·s·cm<sup>-5</sup>.

*Arterial pressure* is an integral value, the components and which are determined by the volumetric blood flow velocity and vascular resistance. Therefore, systemic arterial pressure is the resultant value of cardiac output (CO) and total peripheral vascular resistance (TPVR).

In relation to arterial pressure, systolic, diastolic, pulse and mean pressure are distinguished. Systolic - occurs in the arteries during the systole of the left ventricle of the heart, diastolic - during its diastole, the difference between the systolic and diastolic pressures characterizes the pulse pressure. There is also an average pressure (MAP), which is an average (not arithmetic) value between systolic and diastolic pressures, which would be capable, in the absence of pulse fluctuations in blood pressure, to give the same hemodynamic effect that occurs with natural, oscillating blood movement. SBP is determined by the formula:  $SBP = (BP_{syst} + 2BP_{diast}) / 3$  and equal 80-90 mmHg

#### Literature:

- 1.** Lebedinsky K.M., Bautin A.E., Bestaev G.G. etc. Blood circulation and anesthesia. Publisher: Man, 2015.
- 2.** Bunyatyan A.A., Trekova N.A., Meshcheryakov A.V. etc. Guide to cardioanesthesiology. - M.: MIA, 2005. - 688 p.
- 3.** Bullock J., Boyle J., Wang MB, Ajello RR Physiology. - John Wiley & Sons, 1984. - 392 p.

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1. Delivery of oxygen, nutrients and salts, hormones and other active substances to the tissues;
2. Removal from tissues: carbon dioxide and other end products of metabolism;
3. Participation in heat transfer.

Functional departments of the cardiovascular system

The functional classification of the Swedish physiologist B. Folkov (1976) provides for the division of the circulatory system into "sequentially connected links or elements":

one. *The heart* is a pump that rhythmically releases blood into the vessels.

2. *Elastic-stretchable vessels* that turn the periodic release of blood from the heart into a uniform blood flow (the aorta with its departments, the pulmonary artery).

3. *Resistive vessels*—resistance vessels) - precapillary (mainly arterioles) and postcapillary sections (venules), which together create a general resistance to blood flow in the vessels of the organs.

four. *Precapillary sphincters*—a specialized department of the smallest arterial vessels, the contraction of the smooth muscle cells of these sphincters can lead to the overlap of the lumen of small vessels. These vessels regulate the volume of blood flow in the capillary bed.

5. *Exchange vessels*, or true capillaries, where the blood contacts the tissue due to the huge surfaces of the capillary bed. Here the main function of the cardiovascular system is realized—the exchange between blood and tissues.

6. *Bypass vessels* (arteriovenous anastomoses), the presence of which is not proven for all tissues.

7. *Capacitive vessels* in which changes in the lumen, even so small that they do not significantly affect the overall resistance, cause pronounced changes in the distribution of blood and the amount of its inflow to the heart - the venous part of the system.

The circulatory system can be divided into a number of components. The functional divisions of the circulatory system are classified as follows:



### 1. Macrocirculation:

- heart pump;
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- vessels-containers (veins).

### 2. Microcirculation:

- vessels of resistance or distribution (arterioles and venules);
- vessels of exchange (capillaries);
- shunt vessels (arteriovenous anastomoses).

### 1. Blood: volume and rheology

Blood is a liquid medium that not only transports respiratory gases, energy, plastic and regulatory substrates in the body, but also performs the functions of a heat carrier between the core of the body and its shell. Being a suspension of cells, it is a so-called typical non-Newtonian fluid, the viscosity of which directly depends on the applied shear stress. In relation to hemodynamics, the main characteristics of blood are its active volume (the volume of circulating blood, VCB) and rheological, ie visco-fluid properties. At normal values of the blood flow rate, the ratio of plasma volumes and shaped elements in the contents of the vessel is set by its diameter. Therefore, the hematocrit of the vascular system will depend on the structure of this vascular bed – the length and specific weight of vessels of different diameters.

### 2. Vascular bed

The state of the vessels and, in particular, the systemic vascular tone, are of interest to us in two aspects. On the one hand, the predominantly neurogenic tone of the venules and small veins forms the very vascular capacity that normally contains up to 75-80% of VCB and allows physiologists to call the veins of the large circle together with the small circle of blood circulation a system of high volume and low pressure. On the other hand, the tone of the arterioles, which depends more on local metabolic influences, determines the value of systemic vascular resistance.

### 3. Cardiac output and its determining factors

The minute volume of blood circulation (MVBC, minute volume of the heart, heart performance, cardiac output - the terms are equivalent) is formed as the sum of the shock volumes of blood (SVB, one-time heart performance) per

minute. Thus, the cardiac output is a derivative of the stroke volume and heart rate (HR). The unit of measurement of the cardiac output is l/min.

The SVB, in turn, is determined by the difference in the phase volumes of the ventricles at the end of the diastole (end-diastolic volume, EDV) and the end of the systole (end-systolic volume, ESV).

The normal values of the MVBC are 4-6 l. To neutralize the influence of body size and make the indicator more comparable, A. Grollman proposed the cardiac index - CI) - the ratio of the MVBC to the body surface area (normally  $2.8 \pm 4.2$  l/m<sup>2</sup> / min).

The end-diastolic volume (EDV) is the sequential result of (a) blood infusion under the action of filling pressure (central venous pressure for the right, pulmonary vein pressure for the left heart) into the ventricle with a given extensibility, and (b) active "reloading" of the ventricle by the atrial systole. The diastolic extensibility of the ventricle depends on its passive mechanical rigidity; there is a term compliance (pliability - eng.) of the ventricle.

Ventricular filling pressure characterizes venous return. In the strict sense, the latter indicator has the flow dimension and for the right ventricle depends on: a) the ratio of the volume of circulating blood and the tone of the venous (capacitive) section of the vascular bed, which normally contains 60- 80% of the circulating blood volume, b) the pressure gradient between the peripheral veins and the right atrium, and c) the value of the end-systolic volume (ESV), at which diastole begins.

With a growing EDV and stretching muscle fiber, the energy of contraction and shock volume also increase to the point of overstretch, after which the shock volume begins to decrease, as, for example, in heart failure. Increased preload leads to increased cardiac output. Preload depends on the return of venous blood, which is influenced by the position of the body, intra-thoracic pressure, the volume of circulating blood and the tone of the vessels of the venous system.

The effect of ESV is realized through the value of the passive stress of the ventricular wall at the beginning of the diastole, which, according to the law of P.-S. Laplace (1807), all other things being equal, is directly proportional to the cubic root of ESV. Thus, closing the circle, ESV characterizes the adequacy of the systolic function to the applied preload. It is important that the systemic venous

return - the "fulcrum of blood circulation" (A. Guyton, 1973) - is directed to the right heart, while the systemic perfusion is determined by the left. Depending on the rhythm, the BWT is determined either by the sum of a) the volume at which the elastic thrust of the ventricle is balanced with the filling pressure and b) the systolic volume of the atrium, or by the sum of the same systolic volume of the atrium (b) with the volume at which the diastole ended due to the onset of the atrial systole (a). The latter situation characterizes a more frequent rhythm, and in the limit is the mechanism of the fall of the SVB in tachycardia. The implementation of one of the two described options depends on whether the diastole time is sufficient to balance the elasticity of the ventricle with the filling pressure (when the flow independently becomes zero), or whether the next atrial systole begins with non-zero flow through the atrioventricular valves. The decisive input parameters are: a) the value of the filling pressure, b) the extensibility of the ventricle, and c) the duration of the diastole, ie the difference between the duration of the cardiocycle (the period of heart rhythm) and the duration of the mechanical systole.

The duration of the period of expulsion, in turn, is determined by the ratio of contractility (inotropic status) and afterload, which determines the pressure in the ventricular outlet tract against which the myocardial fibers perform their mechanical work. The inotropic status, further, depends on: a) the length of the fibers (myofibrils) at the beginning of the contraction process, and b) the sum of other factors affecting their functional state.

The initial length of myofibrils, which depends on their extension to the end of the diastole, reflects one of the modern interpretations of the concept of *preload*: all other things being equal, it determines the size of the active contact surface of the actin and myosin filaments of the sarcomere in the initial conditions of contraction.

The ejection fraction ( $EF = SVB/EDV$ ), as it is clear from the above, does not characterize contractility as such, but its adequacy to the current values of pre-and post-loading. Thus, ESV is the result of the dynamic equilibrium of the voltage of the contracting myocardium with the pressure created by it in the output tract.

The exact measure of afterload today is considered to be the end-systolic stress of the ventricular wall, which has the dimension of pressure. The pressure in the exit tract, which also characterizes the post-loading of the ventricle, is determined by the rate of expulsion of blood into the systole and the hydrodynamic resistance of the vascular system. To characterize the latter, the indicators of total

vascular resistance are introduced; peripheral (TVRP) - for a large circle of blood circulation and pulmonary (lung) (TLVR) - for a small one.

Since the increase in the size of the body, all other things being equal, increases the total lumen of the vessels, the indices are calculated not as a ratio, but as the product of the resistance values by the surface area of the body, or , which gives the same result, as the ratio of the pressure difference to the flow related to the surface area (ie, replacing the MVBS-1 multiplier with the cardiac index (CI)

- one).

The normal values of the TVRP are 900-1500  $\text{din} \cdot \text{s} \cdot \text{cm}^{-5}$ , the norm for the TLVR is considered to be 25-150  $\text{din} \cdot \text{s} \cdot \text{cm}^{-5}$ .

*blood pressure* is an integral quantity, the components and determinants of which are the volume velocity of blood flow and the resistance of blood vessels. Therefore, systemic blood pressure is the resulting value of cardiac output (CO) and total peripheral vascular resistance (TVRP).

In relation to blood pressure, there are systolic, diastolic, pulse and average pressures. Systolic-occurs in the arteries during the systole of the left ventricle of the heart, diastolic-during its diastole, the difference between the value of systolic and diastolic pressure characterizes the pulse pressure. There is also an average pressure (ABP), which is the average (not arithmetic) between the systolic and diastolic pressures, which would be able, in the absence of pulse fluctuations in blood pressure, to give the same hemodynamic effect that occurs with the natural, fluctuating movement of blood. The average is determined by the formula:  $\text{ABP} = (\text{BD syst} + 2\text{BD diast}) / 3$  and is equal to 80-90 mmHg.

### **Literature:**

**one.** Lebedinsky KM, Bautin AE, Bestaev GG, et al. Blood circulation and anesthesia. Publisher: Man, 2015.

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