PHYSIOLOGY OF HEMOSTASIS SYSTEM

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The hemostasis system is a complex, labile and multicomponent system that includes mechanisms that are necessary to achieve a certain result. One of these complex mechanisms is to stop bleeding from an injured vessel. , one of the second most important mechanisms is the mechanism that prevents the spread of this process to areas outside the damaged area. are considered to have inhibitors [44].

The main systems that provide hemostasis include the blood-vascular platelet system, the coagulation or plasma hemostasis system, and the protease and fibrinolysis systems that limit their high activity. can participate in functional mechanisms that contradict each other. The simultaneous participation of such a component in several processes causes physiological hemostasis to have the following characteristics:

- high activity of the system of hemostasis, that is, due to the positive functioning of the coagulation network, the activation of the feedback mechanism in relation to it leads to the repeated activation of this process.

- locality of the hemostasis system, i.e. non-spreading of the thrombus formation process in the area outside the damaged vessel due to the effect of the anticoagulation system.

- the fact that the activation of the hemostasis system is of a temporary nature, that is, the thrombus formed in the damaged vessel meets recanalization over time [45].

The primary reaction of the body to a vessel injury is the spasm of the vessel. Vasoconstriction slows down the blood flow in the injured area and, as a result, reduces the level of blood loss. At the same time as the blood circulation slows down, there is a local effect of the factors of the hemostasis system activated in the injured area. Blood in the injured area - spasm of blood vessels first occurs on the basis of a neuro-reflex mechanism, then it occurs under the influence of chemical mediators released from activated thrombocytes, that is, serotonin and thromboxane A2. The process of interaction of platelets with the damaged subendothelial structures of blood vessels occurs primarily through

adhesion to the damaged area with collagen. Adhesion of platelets occurs under the influence of Willebrand factor dissolved in blood plasma, and this factor It is activated when platelets come into contact with the damaged vascular subendothelium. As a result of the adhesion of platelets, their activation occurs. One of the signs of their activation is the release of secretory factors in the form of granules from platelets, that is, thromboxane A2, ADF and factors that stimulate platelet aggregation. Modern tests show that activated platelets secrete plasma hemostasis factors: factor VIII, Willebrand factor, fibronectin, beta-thromboglobulin and other platelet activators, and have a real anticoagulant effect that reverses this process. secreted platelet factor IV is also released. In addition, as a result of the activation of platelets, GPIIb-IIIa receptors are formed on their surface, and fibrinogen in the plasma binds to these receptors. The process of blood coagulation under the influence of plasma factors is based on the limited proteolysis of protein molecules of coagulation factors on the basis of a complex mechanism, and as a result, their active form is formed. The naming of blood coagulation factors with Roman numerals is accepted, and they are numbered from I to XIII. The active form of these factors the numbers are prefixed with the letter "a". The important aspect of blood clotting factors is that they are inactive or weakly activated in solution, and when they come into contact with the negatively charged phospholipid membrane, their activation occurs. As a result of the collision of platelets with the collagen in the damaged vessel wall, they are connected to them and a direct contact with the cell membrane is established. its activity is that it provides the connection of enzyme complexes, enzymes and their cofactors and substrates. Examples of such complexes are tenases (IXa+VIIIa-bCa+2+PF3), prothrombinases (Xa+Va+Ca+2+PF3) possible [46].

Such complexes are important for the formation of optimally structured enzyme molecules and their active centers, and under the influence of such active centers, the speed of the reaction is accelerated ten thousand times. Calcium ions participate in the stabilization of proteins involved in blood coagulation with a tertiary structure.

The most important and central link in the process of formation of a coagulation network is the activation of factor X. There are two mechanisms of activation of factor X. The first mechanism occurs as a result of the effect of factor IXa on the phospholipid membrane with the help of calcium ions. In this enzymatic process, factor VIIIa macromolecules act as a matrix and accelerate the enzymatic reaction thousands of times. This mechanism of hemostasis

associated with contact phase factors is called the "internal activation mechanism". This process is factor XI, which is a proenzyme of contact phase factors and serine proteases. factor XIII, high molecular kininogen, prekallikrein and kallikrein also participate in this process. A complex consisting of XII, XI, prekallikrein and high molecular kininogen is formed in the damaged vessel wall. In this process, a high molecular kininogen cofactor is considered and is important in the formation of the complex. The characteristic feature of the contact phase factors is that they are simultaneously activated and affect each other. As an example, it can be said that active under the influence of factor XII in the form, kallikrein is formed from prekallikrein, and in turn, kallikrein increases the maximum enzymatic activity of factor XII. As a result of the mechanism of internal activation of hemostasis, the formation of activated factor XI occurs in the plasma. Factor XI in the active state o in turn, together with calcium ions, it leads to the activation of factor IX. Activated factor IX activates factor X.

The second and main activation of the hemostasis system, that is, the activation of factor X, occurs with the participation of tissue factor (TF) and factor VIIa. A small amount of factor VIIa is constantly in the blood plasma, but it is tissue factor (TF) and calcium if it collides together with ions, its enzymatic activity increases several times. Tissue factor is considered an intermembrane protein. It is not normally present in blood plasma and endothelial cells. There are domains on the surface layer of this protein, which allow these proteins to bind to factor VIIa in the active state. Another important mechanism of factors of the hemostasis system is the activation of factors VII and Xa. In addition, the activation of factor VII is carried out under the influence of thrombin IXa, XII, and it is several times more important than the activated factor X. its inactive form is considered to be formed from prothrombin. This enzymatic process takes place with the participation of the "prothrombinase enzyme complex", which contains active factors such as Xa+Va+Ca+2+PF3. In this process, calcium ions ensure the binding of prothrombin to the phospholipid membrane. factor Va ensures the interaction of prothrombin with the active form of Xa. The above-mentioned "prothrombinase enzyme complex" accelerates the proteolysis process of prothrombin 500 thousand times. (α-thrombin) is formed. In the functional implementation of the coagulation network, it is important that the formation of thrombin affects and stimulates factors V, VII, XI involved in the formation of thrombin and thrombocytes through a positive feedback effect. In blood plasma after the formation of active thrombin, it affects fibrinogen in the

blood plasma and causes its molecule to be enzymatically split. After such molecular splitting, fibrinopeptides A and B are formed from Aa and Bp chains of fibrinogen. This is a feature of polymerization in the side chain of fibrinopeptides. after such polymerization, initial fibrin is formed in the formation of a fibrin net. A strong peptide bond is formed between the fibrin molecules formed by this enzyme, i.e. fibrin monomers standing next to each other. As a result, a fibrin mesh covering the damaged area is formed in the damaged vessel and blood clotting occurs.

The process of limiting blood coagulation in a damaged blood vessel is the function of the anticoagulation system, which has anticoagulation properties. This system includes factors that prevent the activation of groups of factors involved in blood coagulation. It takes place in the intact endothelium located adjacent to the damaged area of the vessel and leads to the formation of a thrombus only in the damaged area. Such mechanisms include: annexin V - competitive inhibition of factor X, which participates in blood coagulation, with the phospholipid membrane layer , serine protease inhibitors - form a covalent bond with the active centers of the enzyme factors involved in blood coagulation, and as a result lead to their irreversible deactivation. As a result of the interaction of thrombin in the blood plasma with thrombomodulin, it leads to the activation of protein C in the plasma. This activation In turn, it causes the inactivation of blood clotting factors V and VIII absorbed into the phospholipid membrane.

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