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Physiological Mechanisms Of Hemostasis In Living Organisms.

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ABSTRACT

The blood and constant hemostasis have great physiological significance in different biological objects. Hemostasis is carried out by three interacting functional-structural components: the walls of blood vessels, platelets and plasma enzyme systems - coagulation, anticoagulant and fibrinolytic. The process of platelet activation under the action of aggregation inducers involves the interaction of the aggregate with the receptors of the plasma membrane and the signal transmission into the cell, then the transformation of the signal with the participation of secondary messengers and the release of Ca^{2+} ions into the cytoplasm. Thereafter, external manifestations of the cell response follow, including aggregation and the release reaction of chemicals from the cell. Under physiological conditions, the formation of atrombogenic substances in the endothelium predominates over the formation of thrombogenes, which provides for the inhibition of platelet aggregation and hemocoagulation. This preserves the liquid state of the blood in damage to the vascular wall. Under normal conditions, blood coagulation does not occur on the surface of the endothelium. Transformation of the surface of the endothelium from anticoagulant to procoagulant is induced by a tissue factor that activates factor VII, accelerates the activation of factor X and thus triggers an "external" blood clotting pathway.

Keywords: hemostasis, platelets, hemocoagulation, anticoagulation, vascular wall.

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The basis for maintaining social stability in many regions of the world is the solution of the food problem [1,2,3]. In view of the fact that cattle are a major source of milk and meat in many countries of the world, great attention should be paid to the study of its physiology [4].

Recently, it has become clear that blood and mechanisms of maintaining homeostasis, namely hemostasis, have great physiological significance in different biological objects [5,6]. This biological system, providing, on the one hand, the preservation of the liquid state of circulating blood, and on the other hand, prevention and cupping bleeding is considered to be functionally critical [7]. This duality of the opposite functions, which are necessary for the preservation of the organism's life, predetermines the conjugate participation in the mechanisms of hemostasis of various morphological structures and biochemical processes, the multistage nature of their interaction, the functioning of self-acceleration and self-inhibition, activation and inactivation mechanisms at all stages [8,9].

The most important system of hemostasis is to maintain normal blood flow and, at the same time, prevent and stop bleeding in vessels of any caliber. Therefore, the efficiency of this system largely depends on the effectiveness of blood supply to tissues, prevention and relief of hemorrhage [10], thrombosis, ischemia and infarction of organs. protection against dissemination of bacteria and toxins from the lesions of the body, etc. [eleven]. All this determines the most important general biological significance of the hemostatic system and the very significant role of disorders in it in the pathogenesis of the vast majority of diseases [12, 13].

Hemostasis is carried out by three interacting functional-structural components:

- walls of blood vessels;
- blood cells, in the first place, platelets;
- plasma enzyme systems - coagulation, anticoagulant and fibrinolytic [14,15].

The blood vessels and blood cells (platelets and, in part, erythrocytes) react first in response to the damage. In connection with this, the vascular-platelet reaction to damage is designated as "primary hemostasis", and the process of blood coagulation as "secondary", providing the final hemostasis, forming a full thrombus, capable of supporting the body's homeostasis [16,17].

Platelets, non-nuclear blood cells, are the basis of primary hemostasis. Intact platelets are in the form of smooth biconvex disks with a diameter of 2-5 μm. Their membrane, having a thickness of 7-8 nm, consists of polar phospholipids and proteins. Hydrocarbon residues of glycoproteins and glycolipids of the plasma membrane form an outer shell, called a glycocalysis [18, 19].

Thrombocytes have three types of granules: α-granules, dense granules and lysosomes, as well as mitochondria, vacuoles, peroxisomes, the Golgi apparatus. Dense granules contain ADP, ATP, serotonin, pyrophosphate, Ca^{2+} ions; α-granules - growth factor, B-thromboglobulin, factor VIII, von Willebrand factor V, fibrinogen factor, thrombospondin, fibronectin, lysosomal granules - phosphatases, arylsulfatases, acid hydrolases [20].

Thrombocytes have a leading role in the primary arrest of bleeding from microvessels. Circulating in the bloodstream, they practically do not interact with each other and the vascular endothelium. In the case of blood vessel damage, platelets are exposed to various substances that initiate the adhesion and aggregation processes that result in the formation of a platelet plug [21].

When binding the inducer to the receptor on the surface of platelets, aggregation of platelets develops, which is preceded by the stage of their activation - a change in shape from discoid to spherical and the formation of pseudopodia. It is the activated forms of platelets that interact with each other, form aggregates and discard the contents of the granules. Activation of platelets proceeds along several paths. The first pathway involves the metabolism of arachidonic acid and the formation of thromboxane A_2. The second route is associated with the metabolism of phosphatidylinositols and the formation of phosphatidic acid. The third way is probably due to the release of the lysolecithin phospholipid component of the platelet membrane, called the platelet activation factor. An important role is also played by the mechanisms of activation of cAMP, which controls the level of Ca^{2+} ions in the cytoplasm [22].

The process of platelet activation under the action of aggregation inducers involves the interaction of the aggregate with the receptors of the plasma membrane and the signal transmission into the cell, then the transformation of the signal with the participation of secondary messengers and the release of Ca^{2+} ions into the cytoplasm. After that, external manifestations of the cell response follow, including aggregation and the release of chemicals from the cell [23].

According to modern concepts, the leading role in the regulation of platelet functions and coagulation is assigned to the vascular endothelium [24,25].

Substances secreted by the endothelium and participating in hemostasis can, to a certain extent, be conditionally divided into two groups - thrombogenic and atrombogenic. The substances inducing adhesion and aggregation of platelets include von Willebrand factor, platelet activation factor, ADP, thromboxane A_2. Adhesion of platelets to the endothelium and subendothelial matrix - the initial stage of hemostasis and thrombosis. In the norm of adhesion of platelets to intact endothelium does not occur, and in conditions of pathology adhesion is limited, as a rule, to the area of damage to the vascular wall adjacent to the area. This is due to the formation of prostatycline, nitric oxide, ecto-ADPase and other factors that inhibit platelet adhesion and aggregation by endothelial cells [26].

Under physiological conditions, the formation of anthrombogenic substances in the endothelium predominates over the formation of thrombogenes, which ensures the preservation of the liquid state of the blood in damage to the vascular wall, including minor, accidental, which may occur in normal conditions [27]. Under normal conditions, blood coagulation does not occur on the surface of the endothelium. Transformation of the surface of the endothelium from anticoagulant to procoagulant is induced by tissue factor, which activates factor VII, accelerates activation of factor X and thus triggers an "external" way of blood coagulation [28].

The surrounding endothelial matrix contains heparin sulfate and other glycosaminoglycans that increase the activity of a cell-bound or matrix-bound antithrombin III (AT III) [29].

The tissue-derived plasminogen activator (t-PA) formed in the endothelium and secreted from it and its inhibitor is PAI-I. t-PA, like the von Willebrand factor, is secreted continuously, but the "ejection" of it from endotheliocytes can dramatically increase in certain situations. PAI-I is also constantly produced and secreted by endotheliocytes. In the blood and subcellular matrix, PAI-I is bound to the adhesive glycoprotein vigronectin. In this complex, the biological half-life of PAI-I increases by a factor of 2-4. In pathological processes, the content of PAI-I in the blood was increased [30].

A feature of hemocoagulation is that the activation and interaction of coagulation factors at almost all stages of the process occur on free plasma phospholipid membranes [31].

Blood coagulation can function by two mechanisms:

- internal, in which the sequential activation of factors XII, XI, IX + VIII, X + V and II is observed;
- external (rapid), which is triggered by the entry into the blood from outside the tissue factor (factor III), which includes apoprotein III and phospholipid.

Factor III and factor VIIa form an active complex, under the influence of which they are activated in the presence of calcium ions and phospholipid membranes X, V and II. The activated factor X not only converts prothrombin (factor II) to thrombin (factor Pa), but retrogradely activates the factor III factor VIIa complex.

Both ways are closed on the factor X, after which they close and until the formation of fibrin merge into a single stream. However, the external and internal mechanisms of the initial stage of blood clotting are not completely separated from each other. They interact between themselves by mutual activation of factors XII and VII, VII and IX. Factor X retrogradely activates factor VII in combination with factor III and Ca^{2+} [32].

The most important role in maintaining the liquid state of blood is played by a system of physiological anticoagulants, which includes cellular and humoral components. To the cellular components that ensure the maintenance of blood in the liquid state in circulation, first of all, cells of the reticulo-endogelial system and

hepatocytes specifically removing the activated factors of blood coagulation and fibrinogen without any influence on their predecessors. The humoral component consists of physiological anticoagulants that inactivate active clotting factors. Among them, the most significant for practice are antithrombin III, proteins C and S.

The enzyme system that causes progressive asymmetric cleavage of fibrinogen and fibrin is referred to as a fibrinolytic or plasmin system. The main active principle of this system is the proteolytic enzyme-plasmin [33].

Despite the accumulated information on hemostasis, its functional state in productive animals in different periods of ontogenesis has not been studied enough. The formation of platelet functions of the vascular wall and coagulation hemostasis is not defined in physiological conditions, the peculiarities of their functioning with deviations from homeostasis, and approaches to their correction have not been determined.

CONCLUSION

It becomes clear that blood has a great physiological significance in different biological objects, the optimal state of which is maintained by hemostasis. This biological system, providing, on the one hand, the preservation of the liquid state of circulating blood, and on the other hand, prevention and cupping bleeding. Hemostasis is carried out by three interacting functional-structural components: the walls of blood vessels; cells of blood, in the first place, by thrombocytes; plasma enzyme systems - coagulation, anticoagulant and fibrinolytic.

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