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# CLOTS OF BLOOD PLASMA

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*Clots of blood plasma with a decreased content of thrombocytes were investigated on a special tearing machine. The strength and deformation characteristics of the samples have been determined. The data obtained are of interest to biomechanicians and doctors. They supplement the pathogenetic views of thromboses.*

Problems of changing the shapes and dimensions of various structures in living organisms have always attracted the attention of researchers. By now a large number of data on the mechanical properties of the majority of tissues have been accumulated [1–4]. At the same time, some formations in the human organism are not rheologically understood.

The mortality from cardiovascular diseases is very high at present. In many cases, the chief reason for a fatal outcome is thrombosis of vessels, i.e., termination of the blood supply of a tissue with some blood which changed its aggregate state as a result of coagulation.

The rheological properties of fibrinous structures, among which are first of all blood clots, thrombi, and thromboemboles, are practically not understood [5–7].

Under natural conditions, these structures are subjected to complex loadings: tension, torsion, shear, compression, and their combinations. In this case, the biological expediancy (hemostatic clots) or pathogenicity (thrombi and thromboemboles) of the existence of fibrinous formations are determined in many respects by their strength.

The majority of rheological investigations of coagulated blood were carried out on electrographs. These devices make it possible to investigate only blood clots and only within the limits of their insignificant elastic deformations. With them, it is impossible to determine one of the main indices of any structure — strength; therefore, it is beyond sufficient reason to advocate that elastometry gives very valuable information to a doctor.

Because of this, we have carried out investigations on determination of the strength and deformation characteristics of fibrinous structures.

For the object of investigation we have selected human plasma with a decreased content of thrombocytes. This is explained by the fact that the strength basis of the clots and thrombi is a net of insoluble polymerized protein — fibrin. However, fibrin in the initial state is dissolved in plasma. The form elements of blood (erythrocytes, leukocytes, and thrombocytes) can only modify the polymeric structure. Moreover, it has been shown earlier [8] that even thrombocytic retraction does not strengthen the clots.

The plasma for the experiments was obtained from the blood of donors. To prevent coagulation, we stabilized it with a solution of sodium citrate and then removed erythrocytes, leukocytes, and most of the thrombocytes from it by centrifugation in the generally accepted manner.

The clots were tested on a special measuring setup, the main part of which was a tearing machine [9].

In the process of investigation, the substrate (plasma) was filled in a transparent sectional cuvette-form that was placed in a thermostabilizing ($37^{o}$C) liquid. As the latter, we used an isotonic solution of sodium chloride.

The coagulation of the plasma, initiated by addition of calcium chloride, led to the formation of a clot in the cuvette. The mean time of coagulation was $381 \pm 73$ sec. The sample was set in the vertical position in the setup; its working part had the form of a cylinder 6 mm in diameter and 30 mm in length. The upper and lower semispherical parts of the clot fixed it to the mobile (at the bottom) and immobile (at the top) anchors of the tearing machine.

After completion of coagulation, the cuvette-form was opened and the sample found itself in the liquid in the suspended state, as this takes place under natural conditions where the body and the red tail of a thrombus are found in the cavity of a vessel.

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Then the clot was stretched to rupture with a velocity of 11 mm/min, i.e, the tests were static in character.

The mechanical stresses arising in the clot as a result of stretching were transferred through the upper anchor to the rod of a 6MKh1S movable-electrode tube. The latter was a part of a bridge circuit, a signal from which was amplified and fed into a plotting device. In such a way, we have investigated more than thirty samples.

The samples were significantly deformed in the process of testing. The specific elongation was very large and was equal to $2.03 \pm 0.17$ at the instant of rupture. The lateral deformation was extremely large. At an initial diameter of 6 mm, this index was equal to $0.7 \pm 0.1$ mm at the instant of rupture, i.e., it accounted for only 12% of the initial value. The postrupture diameter had a tendency to increase ($0.8 \pm 0.2$ mm).

The volume of the clot decreased irreversibly in the process of stretching. At the instant of rupture this index was equal to only $4.1 \pm 1.3\%$ of its initial value. In this case, the Poisson ratio was $0.46 \pm 0.5$. With the use of a small optical magnification, it was well seen how liquid (serum) was pressed out from the sample in the process of stretching. The rupture force was equal to $209 \pm 73$ mN. Since the initial and rupture diameters of the clots differed markedly, the rupture stress was calculated in two ways: its "conditional" value was calculated for the initial diameter and its "true" value was calculated for the diameter at the instant of rupture. The conditional rupture stress was $7.4 \pm 2.6$ mN/mm$^2$ and the true rupture stress was $550.5 \pm 336.2$ mN/mm$^2$, i.e., the first stress accounted for only 1.3% of the second.

In a similar manner, we calculated the coefficients of elasticity. The conditional Young modulus was equal to $3.6 \pm 1.6$ mN/mm$^2$ and the true one was equal to $271.2 \pm 187.0$ mN/mm$^2$. The coefficient of stiffness was $3.41 \pm 1.5$ mN/mm at the instant of rupture. The specific work of deformation before the rupture of the sample was characterized by the value $7.52 \pm 3.23$ $\mu$J/mm$^3$.

In all cases, the load–elongation diagrams were presented by ascending straight lines passing through the origin of coordinates.

Of some interest is comparison of the data obtained with the characteristics of other tissues in the organism [1–4]. It is important first of all to perform comparison with the protein fibrous structures that form the strength basis of the vascular wall, i.e., collagen and elastin. Before the rupture, elastin elongates by 100–200% and collagen only by 10%. By this index, plasma clots approach the elastic fibers.

The comparison by the value of the Young modulus was performed in a similar manner. However, for the collagen fibers the Young modulus exceeds the values obtained in our work by a factor of $10^4$–$10^5$. For the arterial vessels the true Young modulus is commensurable on the whole with that for the plasma clots. This is also true for the comparison of the modulus of elasticity of the smooth-muscular cells found in the excited state with the results of our investigations.

The true rupture stress of the arterial vessels is approximately ten times larger than that of the plasma clots.

For the majority of biological objects investigated and described in the literature [1–4], the load–deformation graphs represent ascending curves. Their nonlinearity is concentrated on the initial portions. The main portions of the curves, including the limiting values, are well approximated by straight lines. The curves obtained in our work were ascending rectilinear in character on all the portions.

In analyzing the processes occurring in a sample in the case of its deformation and rupture it is necessary to take into account the fact that clots and thrombi are complex colloidal systems. They may at times be classified with gels and at other times with gelatins. The basis for the first and for the second is a spatial net of the insoluble protein polymerizing in the process of coagulation — fibrin — which is a strength basis of the clots or thrombi. The fibrin net occupies a small part of the volume of an object (of the order of one or less than one percent). The interfilament space is filled with liquid (serum), which represents a multicomponent complex solution that is simultaneously intrinsic for low-molecular substances and colloidal for a number of high-molecular compounds.

If fibrin is formed in a stationary liquid, the direction of the fibrin filaments is isotropic, which is clearly seen in the electronic microphotographs [10–12].

We believe that when the sample was stretched, the fibrin filaments approached each other, liquid was pressed out from the interfilament spaces to the environment, and the density of the clot increased. The displacement of the filaments was irreversible in character and, probably, led to their longitudinal (relative to the load) orientation dominating the transverse one, i.e., the stretching could cause an anisotropy of the net. In our opinion, this is responsible for the increase in the length of the clots and the decrease in their transverse dimensions.

In attempting to assign the objects investigated to any types of materials (plastic, brittle, and so on) in accordance with the classifications accepted in study of strength of materials and in rheology we faced a number of difficulties. In particular, the rectilinear (to the point of rupture) load–elongation graphs obtained in our experiments are characteristic of solid brittle bodies. At the same time, the latter are characterized by very small relative deformations, which was not observed in our experiments.

Of course, the fact that the volume and transverse dimensions of the samples decreased irreversibly and significantly, whereas their length increased, is evidence of the plastic deformation. Nevertheless, on the graphs there are no portions pointing to the plastic process.

Close inspection of the conditions of the experiments conducted and the data obtained in them shows that they are not extraordinarily abstracted. A typical arterial thrombus, the length of which is often equal to several tens of centimeters or more, is attached to the wall of a vessel by the white head the basis of which is formed by erythrocytes. The prevailing parts of the thrombus, i.e., its body and tail, which account for more than 90% of the mass of this formation, are in a suspended state in the blood flow. In the flow they are acted upon by different forces, the most obvious of which is the stretching force. The data obtained allow the suggestion that the stretching loads can cause gradual change in the shape of the thrombus and its elongation. The fibrin net will compress due to the pressing-out of liquid from the filament spaces. This can lead, first, to the appearance of an anisotropy in the orientation of the filaments and a strengthening of the structure and, second, to an increase in the fibrinolytic resistance of the thrombus, since the penetrability of the compressed fibrin net decreases, which hampers the access of the lytic agents to the substrate.

These suggestions are indirectly supported by the well-known fact that the old thrombi are more resistant to the lytic therapy that the fresh ones [13]. The venous and arterial thrombi are also different in structure and fibrinolytic stability [7, 1 3, 14]. The arterial thrombi that are subjected to significant intensive hydraulic actions are longer, more dense, and more difficult to remove with the help of fibrinolytics.

Thus, in the present work we have determined the mechanical characteristics of plasma clots with a decreased content of thrombocytes and established that the samples investigated can be assigned with great caution to elastoplastic bodies. The data obtained can supplement the etiopathogenetic views of thromboses and be useful in conducting the fibrinolytic therapy.

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