CLOTTING POWER AND PROTEIN COMPOSITION OF LYMPH AND BLOOD AFTER ACUTE BLOOD LOSS

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We have investigated the role of the lymphatic system in the development of the adaptive changes in the clotting power of the blood after acute blood loss.

EXPERIMENTAL METHODS

Experiments were carried out on 10 dogs weighing 7-20 kg, in a fasting state, and anesthetized with morphine and hexobarbital. Acute blood loss was evoked by rapid withdrawal of 26% of the blood volume from the femoral artery. Blood for investigation was taken from the same artery with glass, silicone-coated cannulas. Lymph was collected through polythene cannulas from the thoracic duct at its point of entry into the left venous angle. In some experiments lymph from the left cervical lymphatic trunk was investigated at the same time. The clotting time of the blood and lymph was measured in a Bazaron's apparatus with automatic temperature control before bleeding and 5, 10, 20, 30, 45 and 60 min thereafter. The initial clotting time was taken as 100%. The velocity of lymph flow was measured by the number of drops of lymph flowing from the cannula per minute. The protein concentration in the sera of the blood and lymph taken after the same time intervals was determined by means of a type IRF-22 refractometer. The protein and lymphoprotein composition of these sera was investigated by the method of electrophoresis on paper.

EXPERIMENTAL RESULTS

The value of the test indices before bleeding are given in the table. It is interesting to note that the relative concentration of β-lipoproteins in the lymph of the thoracic duct was higher than that in the blood, whereas the relative proportions of α- and β-lipoproteins in the cervical lymph were approximately the same as those observed in the blood.

Lymph from the thoracic duct and cervical lymphatic trunk before bleeding did not clot within more than 1-8 h on the surface of the paraffin wax in a Bazaron's apparatus at 37°. In glass test tubes clotting began within 10-15 min. This means that the slow clotting of the lymph was not caused by a deficiency of factors XII and XI. In all probability the lowered coagulability of the lymph was caused by a deficiency of platelets, the phospholipid component of which is essential for thromboplastin formation.

After bleeding, in all the experiments save one the clotting time was shortened: 10 min after bleeding the clotting time was lowered on the average by 26.55% ($P < 0.02$), 20 min after – 23.28% ($P < 0.05$), and 45 min after – by 39.48% ($P < 0.02$). One hour after bleeding this index was close to its initial level. In contrast to the blood, the clotting time of the lymph was unchanged after bleeding. When the cannula was kept in the thoracic duct for long periods, in individual cases the clotting time of the lymph was shortened, as a result of the formation of a thrombus in the cannula. Such artefacts were overcome by changing the cannula.

The velocity of the lymph flow underwent phased changes: 5-10 min after bleeding it was reduced by 39.8% ($p < 0.001$), after 20-30 min it was partially restored ($p > 0.05$), and 45-60 min after it was again reduced by 57.1% ($P < 0.001$). In particular, it should be noted that in these investigation no fluid was injected parenterally, as is usually given to stimulate lymph formation. The slowing of the lymph flow was associated, firstly, with a

Clotting Power and Protein Composition of Lymph and Blood of Dogs before Bleeding

Test material	Clotting time	Velocity of lymph flow (number of drops per minute)	Protein (in g%)	Protein fractions (in %)					Lipoprotein fraction (in %)	
				albumins	globulins				α	β
					α_1	α_2	β	γ		
Lymph	1—8 hr	11,3 ±1,5	4,251 ±0,375	51,57 ±2,37	6,60 ±0,58	12,74 ±1,68	21,90 ±1,72	7,19 ±0,86	38,97 ±2,29	61,03 ±2,29
Blood	1017 ±115,1 sec	--	7,525 ±0,375	48,14 ±1,89	6,81 ±1,52	13,24 ±1,51	25,28 ±2,15	6,53 ±1,19	69,91 ±3,78	37,09 ±3,78

decrease in the hydrostatic pressure of the blood and intensification of the movement of tissue fluid into the blood stream, and secondly, with spasm of the blood vessels after bleeding, leading to a decrease in their filtration surface.

The protein concentration in the blood serum fell 30 min after bleeding by 0.461 g% ($P < 0.002$) and 60 min thereafter by 0.521 g% ($P < 0.02$). No regular change in the protein concentration in the lymph was observed. The ration between the protein fractions in the blood and lymph after blood loss was essentially unchanged, disregarding the very slight increase in the concentration of the γ-globulin fractinn in the blood serum by 1.01 ± 0.38% ($P < 0.05$) after 45-60 min and the decrease in the concentration of the α_2-globulin fraction of the lymph serum by 1.61 ± 0.65%) ($P < 0.05$) after 30-60 min. The relative concentrations of the lipoprotein fractions in the blood and lymph were not appreciably changed after blood loss.

Most of the lymph passes through the thoracic duct and the left cervical lymphatic trunk into the blood stream. The disturbance of the lymph formation caused by cannulization of these principal collectors of lymph did not exclude the acceleration of the clotting of the blood after bleeding. Clotting of the lymph itself was not accelerated. Hence, the acceleration of the blood clotting after blood loss is not brought about by the arrival of factors stimulating coagulation of the blood through the lymph. It may be concluded from the absence of a phenomenon of acceleration of coagulation of the lymph that the factor causing hypercoagulation of the blood does not penetrate to any appreciable extent into the lymph.

SUMMARY

In experiments on dogs a study was made of the role played by the lymphatic system in the acceleration of blood coagulation after acute blood loss. After blood letting the clotting time decreased in all the experiments except one. As distinct from blood, the rate of lymph coagulation remained unchanged. Disturbed lymph circulation, caused by cannulzation of the chief lymph collectors, did not exclude acceleration of blood coagulation after blood letting. Thus, acceleration of blood coagulation following blood loss was not casued by the penetration of factors stimulating hemocoagulation through the lymph. The factor, causing blood hypercoagulation, does not gain access to the lymph in significant quantity.