

# CHANGES IN BLOOD AND LYMPH COAGULATION ACCOMPANYING ACUTE LOWER LIMB ARTERIAL OCCLUSION

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In acute occlusion of the main arteries of the lower limb evidence of a thrombotic state of the hemostasis system is observed: hypercoagulation, inhibition of fibrinolysis, and enhancement of adhesive and aggregative properties of the blood cells [2-8]. According to Mamedov's (1981) hypothesis, changes in the clotting, anticlotting, and fibrinolysis systems in all components of humoral transport (blood—tissue—lymph—blood) are linked and proceed in the same direction in different pathological states. On this assumption, and also in view of the absence of data on changes in coagulation in the lymph in this pathological form it was decided to make a comparative study of changes in the coagulability of the blood and lymph components of humoral transport in acute occlusion of the femoral artery.

## EXPERIMENTAL METHOD

Experiments were carried out on 20 mongrel dogs weighing 10-22 kg. A model of acute occlusion of the main lower limb artery was created by ligating the femoral artery at its origin. Tests on the animals were undertaken in the intact state and 3 h and 1, 3, and 7 days after ligation of the artery (five dogs at each time).

The state of coagulation of the blood and lymph was assessed by the use of the usual *in vitro* tests [1]: clotting time after Lee and White; heparin tolerance after Sigg; prothrombin index after Quick; thrombin time after Szirmai; heparin time after Abrosimov; antithrombin after Marbet and Winterstein, and fibrinogen concentration after Rutberg. Blood was taken from the jugular vein and lymph obtained by draining the thoracic duct. All operations were performed under pentobarbital anesthesia (30 mg/kg, intravenously).

## EXPERIMENTAL RESULTS

In the model of acute arterial occlusion hypercoagulation changes developed both in the blood and in the lymph (Table 1). No marked changes of blood or lymph coagulation were observed 3 h after ligation of the femoral artery, but after 1 and 3 days the coagulability of both biological media rose to a maximum. For instance, the blood clotting time on the 1st and 3rd days was shortened by 24 and 26% respectively, and the thrombin time by 35 and 37% respectively. The antithrombin level fell by 21 and 14% after 1 and 3 days whereas the heparin time fell by not more than 12%. Heparin tolerance was increased on the 1st and 3rd days by 26 and 28% respectively and the fibrinogen concentration was increased by 25%.

Changes in coagulability in the lymph were more marked than in the blood. For instance, the clotting time was reduced after 1 and 3 days by 49 and 37% respectively and the thrombin time was reduced by not more than 26%. Heparin tolerance was increased after 1 and 3 days by 47 and 50% and the fibrinogen concentration by 39 and 30% respectively.

A tendency toward normalization of these various parameters of blood and lymph coagulation was observed 7 days after ligation of the femoral artery.

To sum up the results, in acute arterial occlusion of the lower limb hypercoagulation changes are observed not only in the blood, but also in the lymphatic component of humoral transport. This conclusion is in full agreement with Mamedov's

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TABLE 1. Changes in Coagulation of Blood and Lymph in Dogs with Acute Occlusion of the Femoral Artery

Parameter	Data for intact animals	Acute occlusion of femoral artery							
		3 h	p	1 day	p	3 days	p	7 days	p
Clotting time (sec)	392,6±14,4	356,4±20,7	<0,2	300,0±25,9	<0,02	291,0±10,9	<0,001	387,0±21,9	Not fd.
	603,4±10,1	490,2±20,1	<0,001	309±30,2	<0,001	380,2±10,8	<0,001	449,8±23,1	<0,001
Heparin tolerance (sec)	130,6±10,0	135,2±8,1	Not fd.	96,8±4,8	<0,02	34,4±5,7	<0,02	118,2±7,7	<0,5
	203,8±8,06	186,8±8,3	<0,2	107,8±5,1	<0,001	101,6±5,8	<0,001	175,4±8,7	<0,05
Prothrombin index (%)	90,4±2,6	91,6±1,8	Not fd.	96,4±1,9	<0,2	96,2±2,9	<0,2	88,4±1,8	Not fd.
	77,4±3,5	78,6±2,4	Not fd.	83,0±1,8	<0,2	82,6±1,8	<0,5	79,6±1,1	Not fd.
Thrombin time (sec)	17,6±1,7	16,6±1,1	Not fd.	11,4±1,1	<0,02	11,0±0,9	<0,01	15,4±1,4	<0,2
	22,4±1,5	20,2±1,0	<0,5	11,0±0,7	<0,001	11,8±1,2	<0,001	17,8±1,6	<0,05
Heparin time (sec)	30,6±2,4	38,2±1,3	<0,05	26,8±1,2	<0,2	27,0±2,4	<0,5	28,8±3,1	Not fd.
	41,0±2,5	43,8±1,7	<0,5	30,2±1,7	<0,01	30,2±2,6	<0,02	36,6±2,1	<0,5
Antithrombin-III (sec)	20,2±1,2	20,8±1,2	Not fd.	16,0±1,3	<0,05	17,4±0,9	<0,1	18,2±1,9	<0,5
	24,0±1,1	29±2,7	<0,2	28,2±1,6	<0,5	20,6±1,2	<0,1	23,8±1,3	Not fd.
Fibrinogen concentration (mg/ml)	16,6±0,5	17±0,5	Not fd.	20,8±1,0	<0,01	21,6±0,8	<0,001	19,6±0,8	<0,02
	8,8±0,4	9,2±0,4	<0,5	12,2±0,4	<0,001	11,4±0,5	<0,01	10,6±0,5	<0,05

(1981) concept. An increase in the clotting potential in the biological media of the body evidently impairs drainage of the affected region along both venous and lymphatic channels, and, perhaps, to an even greater degree in the latter; this may promote accumulation of toxic products of disturbed metabolism and rapid enlargement of the zone of cellular destruction.

The results make lymphatic drainage imperative with the aim of stimulating the outflow of toxic products and this must be followed by detoxication treatment.

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