CHANGES IN THE BLOOD COAGULATION SYSTEM IN RABBITS RATS, AND DOGS IN SUDDEN DEATH

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In 1761 Morgagni found that the blood of people who died suddenly lost its capacity to coagulate. This was explained by the rapid disappearance of fibrinogen which is lysed by fibrinolysin [1, 2, 11]. However, it has been pointed out in a number of works recently that in sudden death it is not fibrinogenolysis that develops, but intravascular coagulation of the blood with subsequent fribrinolysis [4, 14].

The purpose of this investigation was to explain these contradictions.

METHOD

Acute asphyxia resulting in death was caused by squeezing the trachea (of 14 anesthesized dogs) and by applying a rope loop on the neck (18 dogs, 15 rabbits, and 25 rats). In another group of experiments (10 dogs and 10 rats) the animals were killed by a hammer stroke on the occipital region of the skull with subsequent destruction of the medulla oblongata by a needle.

The blood of the dogs and rabbits was obtained in the initial state, at the moment of clinical death, and 10 and 25 min after it. In the blood we determined the coagulation time [12], recalcification time [5], plasma tolerrance to heparin [14], fibrinogen concentration [1, 5], and the fibrinolytic activity of the blood [8, 9]. The obtained data were statistically processed (see Table).

To elicit the intravascular thrombi of the dogs, the great vessels (superior and inferior venae cava, vena iliaca, vena femoralis, aorta) and the heart were isolated by ligatures, extracted, and sectioned 25,40, and 60 min and 6, 12, and 24 h after clinical death. The rabbits were autopsied after 30 min and 12 h and the rats after 1 and 24 h.

RESULTS

In dogs at the moment of clinical death from asphyxia, blood coagulation was accelerated twofold, plasma tolerance to heparin increased 1.2 times, and the fibrinolytic activity was intensified 1.5-fold (see Table). By the 25th min after death, these changes became more pronounced. The fibrinogen concentration dropped twofold only by the 25th min after death. Unlike the dogs, in rabbits the fibrinolytic activity of the blood increased more slowly.

In the asphyxiated dogs total thrombi were detected only 60 min after death. The thrombi did not disappear within 24 h, but became more friable.

In the asphyxiated rabbits thrombi were found after 30 min only in the right ventricle of the heart and after 12 h in all large and medium vessels and in the heart; in the rats the thrombi were found in the heart after 60 min and were absent after 24 h; thrombi were present in dogs and rats that died from trauma and were autopsied after 1 and 24 h.

Death from asphyxiation increased the tendency of the blood to coagulate, at first owing to a drop of its anticoagulant activity and later as a consequence of the entrance of tissue thromboplastin into it [14], which led to the formation of thrombi and a drop in the concentration of fibrinogen. A marked increase in fibrinolytic activity of the blood depended at first on the reflex entrance of plasminogen activators into the blood [8, 9, 11] and later on the

Change in the Indexes of the Blood Coagulating System of Rabbits and Dogs in Instantaneous Death from Asphyxia

Index	Statistical index	Initial data		Clinical death	Time after clinical death			
					10 min		25 min	
		dogs	rabbits	dogs	dogs	rabbits	dogs	rabbits
Coagulation time (in sec)	M	240 (32) [1]	—	138 (10)	102 (10)	—	90 (5)	—
	$\pm m$	0,6	—	12,0	7,8	—	3,0	—
	P		—	<0,001	<0,001	—	<0,001	—
Plasma recalcification time (in sec)	M	109 (32)	97 (10)	65 (10)	45 (10)	74 (10)	41 (5)	24 (10)
	$\pm m$	3,0	5,3	4,0	4,0	3,3	5,0	1,9
	P			<0,001	<0,001	<0,01	<0,001	<0,001
Plasma tolerance to heparin (in sec)	M	247 (32)	159 (10)	198 (10)	116 (10)	130 (10)	106 (5)	25 (10)
	$\pm m$	12,0	7,3	13,0	19,0	7,1	23,0	2,1
	P			<0,02	<0,001	<0,02	<0,01	<0,001
Fibrinogen conc. after Gachev (in mg %)	M	464 (32)	460 (10)	394 (10)	358 (10)	440 (10)	309 (5)	395 (10)
	$\pm m$	15,0	2,4	32,0	12,6	1,1	11,7	1,1
	P			<0,1	<0,001	<0,001	<0,01	<0,001
Fibrinogen conc. after Rutberg (in mg %)	M	392 (32)	260 (10)	349 (10)	300 (10)	238 (10)	249 (5)	233 (10)
	$\pm m$	11,6	7,8	22,9	14,6	5,6	5,5	6,7
	P			<0,2	<0,01	<0,05	<0,001	<0,05
Fibrinolytic activity after Coons (in %)	M	18 (32)	8 (10)	31 (10)	42 (10)	13 (10)	50 (5)	18 (10)
	$\pm m$	1,2	1,1	3,4	2,5	1,2	0,8	1,1
	P			<0,01	<0,02	<0,001	<0,001	<0,001
Fibrinolytic activity after Kowalski (in min)	M	133 (32)	—	96 (10)	50 (10)	—	50 (5)	—
	$\pm m$	8,2	—	11,0	8,0	—	14,0	—
	P		—	<0,05	<0,001	—	<0,05	—

[1] The number of animals is indicated in parentheses.

transfer into the blood of lysokinases, proactivators, and activators of plasminogen from the destroyed blood and tissue cells. A normal content of fibrinogen in the blood at the moment of clinical death indicates that fibrinogenolysis does not occur in the body at this time. A comparison of the changes of the thrombi in the corpses of the animals that died from asphyxia and in those that died from trauma permits the conclusion that the conditions for thrombolysis in corpses of rats that died from asphyxia are more favorable than in the corpses of rabbits and dogs; death of the animals from trauma did not create such conditions and thrombolysis did not occur.

SUMMARY

In dogs and rabbits death from asphyxia was accompanied by accelerated blood coagulation, increased tolerance of the plasma to heparin and intensified fibrinolytic activity of the blood. Twenty five minutes later these changes were enhanced and fibrinogen concentration dropped. With the death of dogs from asphyxia and trauma, thrombi were revealed in 60 min; in the first case these thrombi were subjected to a greater destruction within the 24 h. Postmortem examination of rats in 60 min demonstrated the presence of thrombi in the heart. No thrombi were present 24 h later in rats which died of asphyxia, whereas in those which died of a trauma the thrombi were retained. With the death occurring as a result of asphyxia more favorable conditions are created for thrombolysis than after death from trauma.

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