5. D. D. Matsievskii, G. G. Chichkanov, and V. B. Chumburidze, Kardiologiya, No. 1, 133 (1978).
6. N. V. Sanotskaya, Fiziol. Zh. SSSR, 52, No. 5, 556 (1965).
7. N. V. Sanotskaya, "Analysis of mechanisms determining the partial pressure of oxygen in the tissues," Author's Abstract of Doctoral Dissertation, Moscow (1976).
8. N. V. Sanotskaya and D. D. Matsievskii, Byull. Éksp. Biol. Med., No. 12, 119 (1982).
9. A. V. Trubetskoi and V. L. Golubykh, Fiziol. Zh. SSSR, No. 8, 1123 (1982).
10. F. L. Belloni, Cardiovasc. Res., 13, 63 (1979).
11. R. M. Bern, Physiol. Rev., 44, 1 (1964).
12. D. E. Gregg, E. Khouri, and C. R. Rayford, Circ. Res., 16, 102 (1965).
13. D. E. Gregg, Circulation, 27, 1128 (1963).

TREND OF CHANGES IN CATECHOLAMINE CONTENT AND LIPID FATTY ACID COMPOSITION OF THE LUNGS IN TRAUMATIC SHOCK

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Profound disturbances of hormone, mediator, and enzyme metabolism in the lungs in various types of shock are now familiar. Considerable shifts have been found in lipid metabolism and the content of surfactants of lipid nature in the lungs has been shown to be reduced in shock [2, 3, 9, 11, 13]. Changes in metabolism in the lungs are closely interconnected with the balance between several hormones and mediators that are actively removed by the lungs from the blood stream or are synthesized in them [10, 12, 14, 15].

Considering the importance of the sympathico-adrenal system in the pathogenesis of traumatic shock it was decided to study to what extent changes in its activity are reflected in the metabolic status of the lungs. The aim of this investigation was a comparative study of trends in catecholamine content and lipid fatty acid composition in the lungs at different stages of traumatic shock.

EXPERIMENTAL METHOD

Experiments were carried out on 64 adult male dogs. Traumatic shock was produced by the method described previously [8]. Intact animals (series 1) served as the control. The animals were killed at once (series 2), 1 h after trauma (series 3), or in the terminal stage of shock (series 4). Adrenalin and noradrenalin in tissues of the lungs, pulmonary arteries and veins, and also in the blood samples taken from the carotid artery (arterial blood) and the right atrium (venous blood) were determined quantitatively by the method in [5]. Lipids were extracted from the lungs by a chloroform–methanol mixture (2:1 by volume) and triglycerides and nonesterified fatty acids (NEFA) were obtained by thin-layer chromatography on silica-gel; the fatty acid composition of the lipids was studied by gas–liquid chromatography [4].

EXPERIMENTAL RESULTS

The adrenalin concentration in the lungs remained unchanged throughout the experiments, but in the pulmonary arteries it rose immediately after trauma. A considerable increase in the adrenalin concentration in the pulmonary veins, observed throughout the torpid phase of shock, should be noted. The noradrenalin level in the lungs fell immediately after trauma, returned to the initial value after 1 h, and fell again considerably in the terminal period. Similar changes in noradrenalin concentrations also were observed in tissues of the pulmonary arteries and veins (Table 1).

Comparison of the catecholamine concentrations in arterial and venous blood shows that immediately and 1 h after trauma the venous blood adrenalin level was higher than the

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TABLE 1. Trend of Changes in Catecholamine Concentrations (in μg/g tissue) in Tissues of Lungs and Pulmonary Arteries and Veins in Traumatic Shock (M ± m)

Test object	Control	Immediately after trauma	1 h after trauma	Terminal period
Lungs	0,05±0,008 / 0,306±0,031	0,09±0,02 / 0,148±0,02*	0,06±0,02 / 0,257±0,095	0,11±0,034 / 0,014±0,009*
Pulmonary arteries	0,08±0,008 / 0,553±0,14	0,290±0,58* / 0,189±0,073*	0,06±0,019 / 0,576±0,14	0,09±0,02 / 0,140±0,075*
Pulmonary veins	0,05±0,012 / 0,376±0,061	0,21±0,05* / 0,167±0,051*	0,15±0,03* / 0,436±0,120	0,22±0,04* / 0,199±0,052*

Legend. Numerator gives adrenalin, denominator noradrenalin concentration. *P < 0.05 compared with control.

TABLE 2. Trend of Changes in Catecholamine Concentration (in μg/liter) in Arterial and Venous Blood in Traumatic Shock (M ± m)

Blood	Initial background (after catheterization of vessels)	Immediately after trauma	1 h after trauma	Terminal period
		Adrenalin		
Arterial	1,8±0,4	1,2±0,18	1,0±0,17	—
Venous	1,9±0,8	2,8±0,72*	3,0±0,7*	16,9±5,65
		Noradrenalin		
Arterial	1,5±0,2	1,2±0,29	3,2±0,7	—
Venous	1,4±0,2	2,7±0,7	0,21±0,1*	6,1±2,1

*P < 0.05 compared with arterial at the same time.

arterial. The noradrenalin concentration in arterial blood 1 h after trauma was higher than in venous (Table 2).

Immediately after trauma a decrease in the oleic acid concentration and an increase in the stearic acid level were observed in the lung triglycerides; the total unsaturated fatty acid content also increased, but later the triglyceride composition returned virtually to normal. The oleic acid concentration fell again in the terminal period but the steric acid level rose, with a parallel rise in the unsaturated fatty acids level. Similar changes also took place in the NEFA fraction. Immediately after trauma the oleic acid content fell, accompanied by a simultaneous rise in the palmitic acid level; in the terminal period the palmitic and stearic acids levels rose whereas the oleic acid level fell (Table 3).

Catecholamines are known to have a marked influence on lipid metabolism; in particular, they activate lipolysis and stimulate fatty acid oxidation; noradrenalin promotes utilization of oleic acid by the myocardium [6, 7]. At the same time the lungs play a special role in catecholamine metabolism; noradrenalin is taken up in the lungs from the blood stream, and this uptake is linked not only with neuronal assimilation of the mediator, but also with the function of the endothelial cells of the pulmonary capillaries and postcapillary venules, where it can be detected by autoradiography and fluorescence microscopy [12, 15].

Accumulation of adrenalin during shock in the pulmonary veins (throughout the experiment) and pulmonary artery (immediately after trauma), and the higher adrenalin level in venous blood than in arterial blood can be regarded as important facts. They may be linked with increased uptake of adrenalin by vessels of the pulmonary circulation, by analogy with the increased uptake of adrenalin by the myocardium observed in different types of stress. At the same time, lowering of the noradrenalin level in the vessels and tissues of the lungs and the increase in its concentration in arterial blood compared with venous are evidence that, at a

TABLE 3. Fatty Acid Composition of Triglycerides and NEFA in Lungs (in %) in Traumatic Shock (M ± m)

Fatty acids	Control	Immediately after trauma	1 h after trauma	Terminal period
		Triglyceride		
Palmitic	30±1,8	34±2,4	27±2,0	36±3,9
Palmitoleic	3±0,9	2±0,7	3±0,4	3±0,6
Stearic	22±2,5	36±5,5*	27±5,8	32±3,1*
Oleic	45±1,7	28±3,1*	42±3,2	29±3,9*
Saturated	52±1,5	70±3,1*	55±3,6	68±4,5*
Unsaturated	48±1,5	30±3,1*	45±3,6	32±4,5*
		Nonesterified fatty acids		
Palmitic	24±2,5	36±3,1*	—	44±1,4*
Palmitoleic	4±0,7	4±0,6	—	3±0,5
Stearic	20±4,6	32±4,2	—	35±1,4*
Oleic	53±2,5	28±5,1*	—	27±2,0*
Saturated	43±3,2	66±6,2*	—	80±1,8*
Unsaturated	57±3,2	34±6,2*	—	20±1,8*

*P < 0.05 compared with control.

certain stage of shock, additional portions of noradrenalin enter the blood stream, probably to supply mediator to the tissues which, as investigations have shown [1], are deficient in adrenalin. One of the consequences of the response of the sympathico-adrenal system in the lungs may evidently be changes in the fatty acid composition of their lipids. It must be pointed out that a decrease in the oleic acid content is observed in those lipids which are basically an energy-yielding substrate (triglycerides, NEFA). It can be tentatively suggested that this phenomenon is based on involvement of oleic acid (which is readily utilized) in energy metabolism; the mechanism triggering these reactions is the reorganization of catecholamine metabolism in the lungs.

LITERATURE CITED

1. A. A. Gorbov, "Adrenergic mechanisms of traumatic shock," Author's Abstract of Candidate's Dissertation, Rostov-on-Don (1979).
2. V. K. Kulagin, Pathological Physiology of Trauma and Shock [in Russian], Leningrad (1978).
3. G. S. Levin and Ts. L. Kamenetskaya, Lipid Metabolism in Blood Loss and Shock [in Russian], Tashkent (1982).
4. Techniques in Biological Chemistry [in Russian], Rostov-on-Don (1975).
5. Technical Problems in Clinical Pathophysiology of the Endocrine System [in Russian], Rostov-on-Don (1979).
6. F. Z. Meerson, Adaptation, Stress, and Prophylaxis [in Russian], Moscow (1981).
7. M. L. Mikhailov, Kardiologiya, No. 12, 88 (1972).
8. B. A. Saakov, V. K. Kulagin, and A. A. Zor'kin (eds.), Some Mechanisms of Formation of Traumatic Shock and Posthemorrhagic Hypotension [in Russian], Rostov-on-Don (1973).
9. V. E. Nikolaev, in: Mechanisms of Some Pathological Processes [in Russian], No. 6, Rostov-on-Don (1975), pp. 7-11.
10. O. G. Tishkin and N. A. Temur'yants, Fiziol. Zh. (Ukr.), No. 1, 80 (1982).
11. J. Cailar and J. Kienlen, Ann. Anesthésiol. Franc., 14, No. Spéc. 3, 2 (1973).
12. C. N. Gillis, Anesthesiology, 39, 626 (1973).
13. C. Mittermayer, U. N. Riede, U. Bleyl, et al., Verh. Dtsch. Ges. Path., 62, 11 (1978).
14. H. Naito and C. N. Gillis, Anesthesiology, 39, 575 (1973).
15. J. Said Sami, Circ. Res., 50, 325 (1982).