

CHANGES IN THE FATTY ACID COMPOSITION OF LIPIDS
IN THE BLOOD PLASMA AND CERTAIN ORGANS IN TRAUMATIC
SHOCK

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The fatty acid composition of the total lipids of the myocardium, lungs, liver, intestine, adipose tissue, and blood plasma was investigated in the course of development of traumatic shock produced in dogs by crushing the soft tissues of the thigh. The development of shock is accompanied by an increase in the relative percentage of linoleic and arachidonic acids in the blood plasma and certain organs; close correlation was found between the linoleic acid concentrations in the adipose tissue and blood. A deficiency of oleic acid also was observed in the lipids of the organs studied.

KEY WORDS: traumatic shock: fatty acids.

Investigations [3, 6, 10] have shown definite changes in lipid metabolism after trauma and blood loss. However, little is known of the conversions of individual fatty acids in traumatic shock although there is evidence of a broad spectrum of their biological defects [2, 7, 8].

The dynamics of the fatty acid composition of the lipids of the blood plasma and of certain organs with an active role in the utilization and conversion of lipids (liver, lungs, myocardium, adipose tissue, mucous membrane of the small intestine) during experimental traumatic shock was studied in the investigation described below.

EXPERIMENTAL METHOD

Traumatic shock was produced in dogs by crushing the soft tissues of the thigh. The blood pressure and oxygen saturation of the blood (in %) were recorded. Lipids were isolated by the method of Folch et al. [4] and hydrolyzed; the fatty acids were methylated in an acid medium. The methyl esters of the fatty acids were analyzed by gas-liquid chromatography on the Tsvet-4 and Tsvet-5 chromatographs with an ionization-flame detector. The stationary phase was 30% polypropylene-glycol adipate. The carrier gas was hydrogen. The temperature of the column was 202°C and of the vaporizer 275°C; the column consisted of a copper capillary tube (0.35 mm in diameter and 40 m long). Tissue samples were investigated immediately and 1 and 3 h after trauma. A group of intact animals served as the control. In some cases biopsy of the subcutaneous fat was carried out before and after trauma.

EXPERIMENTAL RESULTS

Immediately after trauma the blood pressure fell to 50-60 mm Hg and remained within these limits until the end of the experiment; the oxygen saturation of the venous blood fell considerably, whereas the oxygen saturation of the arterial blood remained within the initial limits until the animal was in a state of agony.

An increase in the content of linoleic acid in the lipids of the plasma and certain organs (myocardium and lungs) was observed 1 h after the fall of blood pressure. During the same period the content of arachidonic acid in the lipids of the myocardium, liver, and plasma increased. As regards oleic acid, this was deficient in the lipids of the myocardium, liver, and intestine. No significant changes took place in the composition of the fatty acids of the lipids of the adipose tissue of the different groups of animals (Table 1). However, when samples of subcutaneous fat were taken from one animal, in the course of the experiment the content of linoleic acid fell (18.1% initially, 12.6% during shock; $P < 0.05$), and close negative correlation was observed between

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TABLE 1. Changes in Content (in %) of Higher Fatty Acids in Lipids of Blood Plasma and Internal Organs during Traumatic Shock ($M \pm m$)

Fatty acids	Left ventricle			Lungs	
	control (n=6)	shock		control (n=8)	shock 1 h (n=8)
		1 h (n=8)	3 h (n=9)		
Palmitic 16:0	21.4 \pm 1.85	18.6 \pm 1.42	19.4 \pm 1.04	26.9 \pm 1.20	26.4 \pm 1.47
Palmitoleic 16:1	2.23 \pm 0.34	3.35 \pm 0.46	2.0 \pm 0.23	3.5 \pm 0.34	4.1 \pm 0.25
Stearic 18:0	22.9 \pm 2.01	18.1 \pm 2.25	20.0 \pm 1.93	17.9 \pm 1.2	17.1 \pm 0.85
Oleic 18:1	37.6 \pm 2.74	26.1 \pm 3.9*	36.5 \pm 3.08	38.3 \pm 3.5	30.4 \pm 0.87
Linoleic 18:2	12.6 \pm 3.15	23.7 \pm 1.31*	17.9 \pm 1.62	7.8 \pm 1.43	13.1 \pm 1.60*
Arachidonic 20:4	3.31 \pm 1.3	10.1 \pm 1.4*	4.1 \pm 0.68	5.6 \pm 1.83	8.8 \pm 1.07

Fatty acids	Lungs	Liver			Blood Plasma
	shock 3 h (n=11)	control (n=6)	shock		shock (n=7)
			1 h (n=4)	3 h (n=10)	
Palmitic 16:0	31.6 \pm 1.27*	22.6 \pm 1.38	16.2 \pm 3.79	20.8 \pm 1.49	21.2 \pm 3.6
Palmitoleic 16:1	2.6 \pm 0.3	1.4 \pm 0.13	1.74 \pm 0.49	1.3 \pm 0.18	1.8 \pm 0.3
Stearic 18:0	19.9 \pm 1.3	31.5 \pm 2.24	20.1 \pm 5.57	29.7 \pm 1.38	30.0 \pm 3.1
Oleic 18:1	26.3 \pm 1.1*	26.5 \pm 1.91	21.8 \pm 2.09*	25.4 \pm 2.39	31.6 \pm 1.4
Linoleic 18:2	10.8 \pm 0.92	13.2 \pm 2.79	18.2 \pm 1.26	16.5 \pm 0.91	10.6 \pm 3.7
Arachidonic 20:4	8.7 \pm 0.93	4.8 \pm 1.48	12.9 \pm 2.51*	7.3 \pm 1.29	4.8 \pm 1.5

* $P \leq 0.05$.

the linoleic acid content in the blood plasma and in the adipose tissue ($r = -0.764$, $P < 0.05$). This correlation demonstrates that one possible cause of the increase in the blood plasma concentration of linoleic acid is its net outflow from the fat deposits. The increase in the relative percentage of arachidonic acid in the blood plasma and liver can also be considered to be evidence of its increased formation in the liver and subsequent transport into the blood.

In traumatic shock a variety of changes is thus observed in the fatty acid pool of the lipids. Effects of arachidonic acid discovered comparatively recently [2, 7, 9] and its role as precursor of the prostaglandins suggest that the changes observed may be of great pathogenetic importance.

The differences in utilization of the fatty acids may also largely determine the changes in the fatty acid spectrum of the lipids. For instance the dog's heart utilizes predominantly oleic acid [5]. An oleic acid deficiency is observed in rats with experimental hypertension [1]. During physical exertion oleic acid metabolism has been found to take place more intensively than palmitic acid metabolism [11]. The decrease in the oleic acid content discovered in these experiments, it can tentatively be suggested, may be connected with its high ability to be utilized by the tissues, which is manifested particularly clearly under the conditions of hypotension and hypoxia associated with shock.

Shock hypotension is thus accompanied by distinctive conversions of the higher fatty acids, including the essential fatty acids, and this may perhaps have an important influence on the course of the shock.

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