CHANGES IN OXIDATIVE PROCESSES IN BURN SHOCK

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During the first few hours of burn shock in rabbits the arteriovenous oxygen difference increases whereas the activity of oxidative enzymes (cytochrome oxidase) in the mitochondria of the organs is unchanged at this time. A shift of the blood pH to the acid side and the accumulation of incompletely oxidized products are evidently due to disturbances of the blood circulation.

There is no doubt of the existence of tissue hypoxia during burn shock. Under clinical and experimental conditions the accumulation of incompletely oxidized products has been consistently determined during the first few hours after severe burns [1, 11]. The mechanism of this phenomenon has been explained by disturbances of external respiration, of oxygen transport, and of the hemodynamics [5, 8, 11] although other workers consider that the chief cause of the hypoxia is a primary inhibition of oxido-reductases (cytochrome oxidase) in the tissues [1, 2, 7].

To study oxidative processes during burn shock parallel tests were made of the acid-base balance and concentration of incompletely oxidized products in the blood and of the activity of oxido-reductases in the blood and organs of the same animals.

EXPERIMENTAL METHOD

A third degree burn covering 20% of the body surface (boiling water, exposure 40 sec) was inflicted on 25 male rabbits weighing 2.5-3 kg. The animals developed severe shock accompanied by a reduction in the minute volume of the heart and leading to death after 5-6 h. Before the burn was inflicted cannulas were introduced into the carotid artery and jugular vein of the rabbits. Before burning, 30 min, and 3 h, 30 min after burning estimations were made of the pH of the blood, the partial pressure of oxygen and oxygen saturation of the arterial and venous blood, the lactic and pyruvic acid concentrations in the blood [9, 10], activity of lactate and malate dehydrogenases in the blood [6, 13], and cytochrome oxidase activity in the erythrocytes [3] and in mitochondria of the liver, kidneys, and skeletal and heart muscles [12].

EXPERIMENTAL RESULTS AND DISCUSSION

The results given in Table 1 show that 30 min after burning the pH of the blood was lowered, the lactic acid concentration in the blood was raised, while the oxygen saturation of the venous blood was reduced. During the next 3 h the concentration of organic acids in the blood remained at the same level but the oxygen saturation of the venous blood continued to diminish.

The increase in the arteriovenous oxygen difference in burns is regarded as an adaptive reaction of the tissues aimed at overcoming the metabolic acidosis and the oxygen deficiency which develop as the result of a disturbance of the peripheral circulation [4]. In this context it is interesting to examine the activity of the oxide-reductases in several tissues. Lactate dehydrogenase activity in the blood during the period of burn shock increased (from 242 units in the control to 380-417 units in the experi-

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TABLE 1. Acid-Base Balance and Organic Acids of the Blood in Burn Shock (M \pm m)

Experimental conditions				PO ₂	
		Lactic acid (in mg %)	(in mg%)	artery	
Control (original state)	7,34±0,02	36,38±2,42	1,08±0,16	87±1,8	
30 min after burning	7,03±0,03 <0,001	$69,41\pm2,71$ <0,001	1,43±0,14 >0,1	88 ± 0.4 >0.5	
3 h 30 min after burning P	6,92±0,001 <0,001	70,0±6,3 <0,001	1,25±0,09 >0,1	$82,5\pm 6$ >0,5	

Experimental conditions	PO ₂	HbO:		Arterio-	
	vein	artery	vein	venous difference	
Control (original state) Shock:	41±2,6	95,2±0,5	61±5	35±5	
30 min after burning P 3 h 30 min after burning P	27±2 <0,01 23±1,4 <0,001 -	85,6±2,5 >0,05 79±5 <0,05	23,6±4 <0,001 14±2,5 <0,001	62,2±4 <0,001 60±3 <0,01	

TABLE 2. Cytochrome Oxidase Activity in Mitochondria of Organs (in mg indophenol/mg protein) and in Erythrocytes (conventional units) of Rabbits during Burn Shock (M ±m)

Experimental conditions	Liver	Kidney	Heart	Muscles	Erythro- cytes
Control Shock:	1,84±0,12	1,38±0,08	1,33±0,07	2,05±0,17	2,4±0,14
30 min after burning	1,43±0,14	1,39±0,08	1,33±0,1	2,36±0,20	2,1±0,1
3 h 30 min after	<0,05 2,08±0,8	>0,05 1,42±0,06	>0.2 $1,46\pm0.05$	>0.5 2.18 ± 0.32	>0,2 $2,4\pm0,15$
b urnin g P	>0,2	>0,5	>0,2	>0,5	>0,2

mental series) while malate dehydrogenase activity decreased (from 2.45 to 1.3-0.8 units). Cytochrome oxidase activity was determined in the erythrocytes and mitochondria of the organs (Table 2). The results showed that the activity of cytochrome oxidase, an enzyme of the final stage of tissue respiration, underwent no significant change in the organs of the burned animals during the first hours after injury. Cytochrome oxidase activity fell 30 min after burning only in the mitochondria of the liver, and 3 h after the onset of shock it returned to normal. The hypoxia developing during the first hours of burn shock was evidently caused by primary disturbances of the blood circulation rather than by changes in activity of the tissue oxidative enzymes.

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