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Blood Lactates in Various Anoxemic States.

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In the course of an investigation on the general subject of the oxidative aspect of functional metabolism which is being carried on in this laboratory, it seemed desirable to test the validity of the hypothesis of A. V. Hill[1] that lactic acid serves as a "governor" of recovery oxidation. To this end two techniques have been developed for the production in the anesthetized dog of anoxemic states, which have been shown by Fletcher and Hopkins,[2] and others, to be the sole cause of excess tissue lactates. Throughout, dogs anesthetized by intraperitoneal injection of amytal were employed. Samples of blood for analysis were drawn from the femoral artery. The recently published method of Friedemann, Cotonio and Shaffer[3] was used for the determination of lactic acid in the blood so obtained.

Artificial Exercise. Two sheet brass electrodes were implanted in the subcutaneous tissues over the midcervical and sacral regions of the spinal cord. By means of faradic stimulation, a regular series of short, vigorous, tetanic contractions, involving the muscles of the trunk and extremities, were produced over a period of 15 minutes. During this intense muscular activity the oxygen consumption of the animal increased eight-fold. The observed changes in blood lactates are shown in Table I.

Subnormal Oxygen Supply. The animal was caused to rebreathe from a closed system, from which the CO_2 was absorbed and to which oxygen was added at a rate below that at which it was consumed by the anesthetized dog. Animals so maintained over periods up to an hour developed deep cyanosis and marked hyperpnea. The observed changes in blood lactates are shown in Table II.

Since the hyperpnea evoked in these experiments itself constituted fairly vigorous muscular activity, the possibility arose that the blood lactate increase might result from the contractile process rather than simple general anoxemia. Accordingly, the procedures were repeated on curarized dogs, artificial ventilation from the closed system being employed. The observed changes in blood lactates are shown in Table III.

We have shown that artificial exercise in the anesthetized dog produces rises in blood lactates comparable to those demonstrated by Hill and co-workers in man after voluntary muscular work. Furthermore, in one experiment, a definite increase of oxygen con-

TABLE I.
Blood lactates after artificial exercise.
(Figures give mg. of lactic acid per 100 cc. blood.)

Exp. No.	Control before exercise	Time after exercise				
		0 min.	15 min.	30 min.	45 min.	60 min
A 2	25.4	42.0	20.8	—	—	
A 3	23.9	51.6	58.5	51.0	—	
A 4	36.2	98.2	103.6	—	59.1	
A 5	19.5	42.5	47.7	—	29.9	
B 11	19.3	40.0	—	—	—	20.3

TABLE II.
Blood lactates after anoxemia.

Exp. No.	Control before anoxemia	Duration of anoxemia	Time after anoxemia in minutes					
			0	15	30	45	60	75
B 6	16.4	18 min	37.0					
		30 min.	29.4					
		48 min.	43.70					
		60 min.	51.30					
B 7	15.1	47 min.	61.81	—	42.47	—	—	26.5 after 2½ hrs.
B 11	19.3	51 min.	58.4	—	27.20	—	19.3	—

TABLE III.
Blood lactates after anoxemia in the curarized dog.

Experiment No.	Control before anoxemia	Duration of anoxemia	Time after anoxemia in minutes			
			0	15	30	45
B 9	14.3	23 min.	64.3			
B 10	29.3	21 min.	47.0			
		49 min.	54.6	79.2	—	47.5
	After curare and operation 33.2					

sumption over the resting value was noted after artificial exercise. These facts indicate a sufficiently close relation between the 2 forms of exercise to permit an analysis conducted by our technique to be of general application.

We have also shown that a subnormal oxygen supply results in a definite and considerable increase in blood lactates, and that this increase is not due solely to the functional metabolism of the respiratory muscles.

[1] Hill, A. V., The Herter Lectures, 1924, p. 102.

[2] Fletcher, W. M., and Hopkins, F. G., *J. Physiol.*, 1906-7, xxxv, 247.

[3] Friedemann, T. E., Cotonio, M., and Shaffer, P. A., *J. Biol. Chem.*, 1927, lxxiii, 335.