

shown that no part of the hyperpnoea from exertion can be attributed to an anoxemia in the arterial blood, since there is a greater degree of oxygen saturation after exertion.⁶ Bayliss¹³ states, "We know that lactic acid is formed, and that the actual contractile process is not associated with the giving off of carbon dioxide, nor with the consumption of oxygen." In the absence of oxygen there is neither evolution of CO₂, nor consumption of oxygen, whereas both processes occur when oxygen is present. The lactic acid content of fresh resting muscle is 0.015%, while that of exhausted muscle is 0.3%. This is the fatigue maximum and is not further increased, however long the muscle is subjected to stimulation.⁵ The application of oxygen, however, causes the lactic acid to fall to its resting value, and the muscle regains its excitability. The oxygenation has restored the lactic acid to its precursor. This fact has been confirmed and extended by Hill, Peters, and Meyerhof.

The recovery process is likened by Hill³ and⁴ to an accumulator which requires recharging after usage, i.e., the lactic acid must be transformed back to its precursor, a process which requires a supply of energy. This energy comes from the combustion of foods by oxygen. All the oxygen that is used by a man during and after muscular exertion is employed in this "recovery process." The capacity for endurance depends upon the maximum available supply of oxygen, while the capacity for rapid, violent, short-lived effort depends upon the acid tolerance. Hill states that of the lactic acid set free during contraction one-fifth is oxidized to CO₂ and H₂O, and four-fifths⁷ is restored to the state of its precursor, which is presumably glycogen. There is probably an intermediary in the formation of lactic acid from glycogen, in hexose diphosphate (lactacidogen), which breaks down to lactic acid and phosphate.⁵ This contention by Embden, Meyerhof, and Evans is stated by Hill to require "wider and more direct verification." John Raymond Murlin has drawn attention to the phosphate precursor, and to the fact that the ingestion of cooking soda after fatigue greatly aids in lessening the lactic acid concentration.

In severe muscular exercise, sprinting

for example, lactic acid is being liberated in the muscles at the rate of four grams per second, and at the end of a 300-yard race there may be one-fourth pound of this potent substance free in the tissues.³ It is obvious then, that the body has been able to perform this enormous amount of work without a parallel removal of lactic acid, and the delayed oxidation in this "recovery process" is known as the "oxygen debt," which is limited to about 15 liters before complete exhaustion. The maximum "intake" is about 4 liters per minute, which Hill⁴ likens to the athlete's "income," while the "oxygen debt," the toleration of his muscles to lactic acid, is his "capital." In exhaustion, "capital" and "income" are completely spent. Using the oxygen after exercise in the "recovery process" makes very vigorous exercise possible.

It is known that even in the blood of a resting individual there are at least 5 to 20 mg. of lactic acid per 100 c.c. This is explained by the fact that even with a restricted amount of muscular activity some of the lactic acid diffuses out into the blood, and is only slowly oxidized. During the more severe forms of muscular activity, lactic acid is being produced at a much faster rate, and in spite of the facts made known by Anrep and Cannan⁸ (confirmed by Long)⁹ it tends to accumulate in the blood, and even be excreted in the urine in small amounts. (Ryffel, Campbell, Douglas, and Hobson.) The rate of this accumulation naturally depends on the severity of the exercise, and the efficiency of the oxidative processes to cope with it. It is found¹⁰ that in severe exertion it may go as high as 204 mg.%. Its passage to the blood stream makes possible, according to the concentration of the H-ion, a stimulation of the respiratory function up to the point of its maximum "intake." The fact of the high concentration of lactic acid in blood after severe exertion may have some significance in the later discussion of the results of certain experiments.

Hill⁴ has shown that the heat production is not continuous, but appears to be in two phases, (a) initial, including the onset, maintenance, and relaxation phases, and (b) a prolonged phase during the recovery process. The initial heat production is the same whether in oxy-