LACTIC ACID IN REST AND WORK AT HIGH ALTITUDE

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The present paper sets forth blood lactic acid values obtained from four groups of subjects at high altitude: members of our party in Chile, residents of 'Quilcha, Chile (altitude 5.34 km.), members of our party at Leadville, and residents at Leadville.

The Chilean party was divided into two groups. The first group, consisting of six men, was engaged for approximately six weeks setting up a mobile laboratory at Chuquicamata at an altitude of 2.81 km. A regular program of work on a bicycle ergometer, mountain climbing, and long walks was followed during this period. The second party from sea level, four men, then joined the first, and observations were immediately begun.

The concentration of lactic acid was determined both in arterial blood drawn in the morning before rising and in blood drawn from the antecubital vein after measured performances on the bicycle ergometer. There were 16 days of observations at an altitude of 2.81 km., 8 days at 3.66 km., 12 days at 4.70 km., and 3 weeks at 5.34 km. During this last period parties of two spent from 1 to 6 days at 6.14 km., the top camp. Lactic acid was determined in blood obtained in rest at this altitude, but it was not feasible to transport the ergometer for exercise studies. Values in rest were obtained on six residents at 5.34 km., five of whom were laborers at the sulphur mine, 5.70 km.

The Leadville party consisted of eight members who spent from one to two months at 3.0 km. altitude. Three members spent four days on the top of Mt. Elbert, 4.39 km. Resting lactic values only are presented for

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the Leadville party. Work values have been previously published (Dill, Edwards, Fölling, et al., 1931). Resting values were obtained on three residents at 3.0 km. altitude. One of them had spent eight months, and two, several years at this altitude. Lactic acid was determined on venous or arterial blood after one hour of rest.

Lactic acid was determined by the method of Friedemann, Cotonio and Schaffer (1927). That the method holds at high altitude was determined by recovery at sea level of lactic acid under an air pressure of 390 mm., equivalent to an altitude of about 5.5 km. Values in this paper are in milli-equivalents of lactic acid per liter of blood, i.e., mgm. per cent divided by 9.

The lactic acid in rest. Since the observations of Araki (1890, 1891), that acid, which he demonstrated to be lactic, appears in the organism in conditions of anoxemia, there have been varying reports concerning the extent to which lactic acid accumulates in rest during oxygen want. Ryffel (1910) detected only a small accumulation in the blood of man after breathing 12 per cent oxygen for four hours. Later Laquer (1919), in a study of blood lactic acid values in men and dogs found that increases of only a few milligrams per cent occur on going to 2.9 km., with no further changes up to 4.56 km. Barcroft (1925) reports that two subjects had a threefold increase on Monta Rosa: at sea level their values were 1.3 and 1.4 m.-eq., at 2.9 km., 1.9 and 2.0 m.-eq., and at 4.50 km., 4.0 and 4.3 m.-eq. Blood was drawn the morning after arrival. Jervell (1928) found no increase in lactic acid until 7.5 per cent oxygen was breathed, the equivalent of 6.10 km. altitude. More recently Bock, Dill and Edwards (1932) found little or no increase in several subjects after breathing for one hour a mixture of oxygen equivalent to an altitude of 6.70 km. Baicenko and Krestownikoff (1933) found that the lactic acid of two subjects varied between 2.2 and 3.9 m.-eq. on 7 of 10 days at 4.20 km. Their values at Leningrad were 1.0 and 1.6 m.-eq.

In table 1 are included summaries of the resting lactic acid values of the Chilean and the Leadville parties. At the 2.81 km. stop of the Chilean party the average, 1.2, suggests a slight rise over the sea-level average, 0.93. Four men, on the morning after arrival at this altitude from sea level, had an average of 1.5, 1.7 being the highest value obtained. After five weeks at this altitude, four men averaged 1.5. After nine weeks, four men averaged 1.0. Two men, however, after two weeks fell from 1.7 and 1.5 to 0.7 and 0.7. No unbroken altitude increase of over 1.05 km. followed this initial one of 2.81 km., which may account for this slight increase not being noted at the upper stations. On the contrary, at the upper stations the averages remain at the sea-level value, even at 6.14 km. Yet at the 2.81 km. station the arterial saturation was diminished by only a few per cent, while at the 6.14 km. station arterial saturations ranged between 55 and 70 per cent.
It is possible that this initial slight rise may be a reflection of acclimatization processes going on, and that it might again be seen if after acclimatization at 2.81 km. one went directly to 6.14 km.

It is obvious, however, that blood lactic acid in resting man is little affected by changes of altitudes compatible with life, and that after a period of time, sufficient presumably for acclimatization, sea-level values are found. This is strikingly illustrated by a comparison of the sea-level values in table 1 with those of the Chilean residents at 5.34 km.

The Leadville averages agree closely with the Chilean averages. Even better agreement might be expected if the conditions of rest for both

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<td>Lactic acid in rest</td>
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<td>Chilean Expedition, 1935</td>
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<td>Leadville expedition, 1929*</td>
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<td>(Venous and arterial bloods after 1 hour rest)</td>
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* Previously unpublished data.

...parties had been the same. Because of the time elapsed before the laboratory was set up at Leadville, we have no evidence regarding changes which may have occurred there during early acclimatization.

The lactic acid in work. There is little in the literature regarding blood lactic acid values in work at high altitudes. Laquer (1922) found in one subject that bicycle riding for one hour may produce a slightly higher level of lactic acid at 1.56 km. and 2.5 km. than at sea level. Baicenko and Krestownikoff (1933) found that immediately after a climb to 5.63 km. the lactic acid was 15.2 m.-eq. Results of experiments in low-pressure chambers and breathing low oxygen mixtures will be mentioned later.

In the present experiments the subjects worked for ten minutes at a
constant rate on the bicycle ergometer, and blood samples were drawn at the end of exercise from the antecubital vein. Figures 1 and 2 summarize the data. The concentration of lactic acid has been plotted against the kilogram pressure on the wheel. This manner of presentation has been adopted for the reason that metabolic rates in an individual varied little for a given output of work regardless of altitude. The metabolic changes are discussed by Christensen, Edwards, Forbes and Matthews (in press). Points on the figures represent physical performance and, as shown in the key, the altitude of each experiment. A curve representing the individual's lactic acid levels for various grades of work at sea level has been drawn for each individual except Matthews, who has not been available as a subject.

Figure 1 presents the performances of four members acclimatized to 2.81 km. for six weeks before the experiments were commenced. There is no evidence of extra lactic acid accumulation for a given grade of work in these men even at 5.34 km., except for a few points at 3.66 km. on E. H. C. and A. B. K. In all these men there are in many instances smaller increases in lactic acid for a given grade of work at the higher altitudes than had been observed at sea level, especially in the harder grades of work. In two cases, E. H. C., 2.81 km. altitude, 3.0 kgm. load, and W. H. F., 4.70 km. altitude, 2.25 kgm. load, the rise was distinctly less, but this, however, may be related to the fact that work could only be continued for five minutes. We are not disposed to attach much significance to subnormal rises in lactic acid, but it seems highly significant that for a given grade of work anoxemia does not produce a greater than normal increase in lactic acid.

Figure 2 presents the performance of three men who showed distinct tendencies to reach higher levels of lactic acid at 2.81 km. than at sea level. In the case of H. T. E. the experiment was carried out after 37 days at this altitude. No further experiments on the ergometer were performed on this subject. Those on D. B. D. and J. H. T. were begun the day after their arrival from sea level.

At the higher altitudes these two men no longer reached higher levels of lactic acid than at sea level. Even H. T. E., who showed the most pronounced excess lactic acid formation, probably approached his sea-level curve finally, since at the end of the expedition he had the record number of climbs to the top camp, 6.14 km., a climb that every member found exhausting. On a previous expedition, Dill, Edwards, Fölling et al. (1931) this man finally reached his sea-level performance curve after eight weeks at 3.00 km. On this previous expedition, D. B. D. and J. H. T. showed a lag in approaching their sea-level curves. This lag may be related to physical training since all members of the present expedition, H. T. E. excepted, were in excellent physical condition.
The failure to show higher levels of lactic acid at the higher altitudes not only in various intensities of work but also in rest, may be due to the fact that no single rise after the initial one of 2.81 km. exceeded 1.05 km., or it may mean that once acclimatization has begun at lower altitudes, acclimatization to higher altitudes takes place more readily. In any case, sea-level lactic acid values are eventually reached at any altitude, at least up to 6.14 km. It is significant that, as in the case of the resting lactic acid, the increases over sea-level values occur at the initial station, 2.81 km., where arterial saturation was diminished only a few per cent, yet there were no increases at upper stations where arterial saturations were as low as 55 per cent.

The arrows on the graphs of figures 1 and 2 indicate the maximum load that each subject could pull for 10 minutes at the designated altitude.
For example, E. H. C., figure 1, could ride with no more than 1.5 kgm. load for 10 minutes at 5.34 km. altitude. The lactic acid accumulated was the same as at sea level for this grade of work, 1.7 m.-eq. Yet at sea level he was capable of riding at the same rate with double the load, accumulating 7.1 m.-eq. of lactic acid. In other words, the ability to perform work is lessened progressively with increase in altitude, and also, with acclimatization, the ability to accumulate lactic acid is lessened. Work experiments of Clark-Kennedy and Owen (1927) performed at sea level while breathing mixtures of low oxygen pressure, led to the conclusion that “the degree of acidosis is proportional not only to the grade of work but inversely to the oxygen tension of the inspired air.” The latter part of this statement is completely at variance with our observations on fully acclimatized subjects. Chamber experiments, unless the subjects are in good physical training and have been allowed sufficient time for acclimatization, may be poor substitutes for field studies on men leading an active life.

The inability to produce appreciable amounts of lactic acid at very high altitudes after acclimatization is illustrated in an experiment on D. B. D. and H. T. E. after 5 and 17 days respectively at 5.34 km. After riding mules to 5.70 km. they climbed intermittently for more than an hour, stopping frequently because of exhaustion. On arrival at the top camp, 6.14 km., the lactic acid values were only 1.76 and 1.83 m.-eq. That the values on D. B. D. and H. T. E. are probably maximal is suggested by the paper of Margaria and Edwards (1934) in which they found that in exhausting intermittent exercise of this type the highest attainable lactic acid values are soon reached. Further work is then carried on, as postulated by Margaria, Edwards and Dill (1933) by an alactacid mechanism.

At 6.14 km. altitude arterial saturations were between 55 and 70 per cent. In this range the oxygen dissociation curve is steep. Therefore, a large accumulation of lactic acid would result in a large diminution in the already low arterial saturation. Accordingly, this inability to increase lactic acid seems to reflect a protective mechanism. It is difficult to correlate these findings with the 15.2 m.-eq. of lactic acid found by Baicenko and Krestownikoff (1933) previously mentioned.

That the protective mechanism is very stable after acclimatization is shown by an experiment on E. H. C. The subject after one week at 5.34 km. breathed throughout the experiment sea-level equivalent of oxygen while riding with 3.0 kgm. pressure, a task he had been quite capable of carrying on at sea level for 10 minutes. In 2 minutes and 42 seconds he stopped in an acute state of exhaustion which persisted several minutes. Yet the lactic acid level reached was only 1.67 m.-eq., compared to 7.1 m.-eq. after working for 10 minutes at this rate at sea level. As can be seen from his performance curve, figure 1, the 1.67 m.-eq is almost
identical with the lactic acid for half the load when exhaustion was reached after 10 minutes at this altitude. There was only a slight rise (to 2.01 m.-eq.) after 5½ minutes of recovery.

This diminished ability to accumulate lactic acid and this failure to utilize oxygen reflect marked changes in the organism after acclimatization. Immediately there occurs to one, by way of explanation, the fall in alkali reserve. At sea level, however, when the reserve is diminished to the level at which E. H. C.'s was at 5.34 km., one can still carry on work that reduces the reserve to a much lower value chiefly through the accumulation of lactic acid. In the case of E. H. C. there exists a slight

increase in pHs; in the case of the subject at sea level, a decrease. Bearing in mind, however, the principles of the system as a physico-chemical unit as illustrated by L. J. Henderson (1928) we cannot argue merely from a pH viewpoint.

Figure 3 contains the highest lactic acids attained by each individual at the various altitudes. With increase in altitudes there is a decrease to remarkably low values at 6.14 km. It is obvious from the figure and the explanatory footnote that higher lactic acids can be attained by climbing intermittently to exhaustion than by continuous exercise to exhaustion on the ergometer. This is illustrated by the two points for W. H. F., v, (v),
at 4.70 km. After each stop while climbing intermittently at high altitudes a rapid recovery takes place within a few minutes. This recovery is hardly suggestive of the recovery following a large accumulation of lactic acid in the body. Often one stops with the feeling that it is physically impossible to take another step. Yet, if the legs were the cause of stopping, through accumulation of lactic acid, obviously II. T. E., ⟨⟩ 5.34 km., figure 3, and D. B. D., ⟨⟩, and H. T. E., ⟨⟩, 6.14 km., figure 3, would have reached much higher levels of lactic acid, since the legs represent such a large portion of the body muscles, and time for the diffusion of the lactic acid was ample. A muscle such as the heart or the diaphragm, however, could become exhausted from the accumulation of lactic acid with little effect on the level of lactic acid in the body as a whole. The intense respiratory difficulty experienced in the region of the diaphragm during the pauses suggests the involvement of the diaphragmatic muscle. It may be that the protective mechanism or the limiting factor in work at high altitudes is the circulatory failure to the diaphragm or the heart.

This leaves unexplained, however, the failure of E. H. C. to utilize the added oxygen, and no explanation is offered here. It may be that the problem is one of utilization rather than of transportation. If so, we become involved in the rôle of myoglobin and the complexity of tissue chemistry. It is not impossible that we are dealing with an enzyme.

It is of interest, in view of the findings in this paper, that Weiss and Ellis (1935) enumerate several of the classical symptoms of exhaustion at high altitudes in cases of cardiac decompensation during work at sea level. Only small amounts of lactic acid could be accumulated. Comparing such performances with those of normals reaching the same low levels of lactic acid, he concludes that "when disturbances in the lactic acid production and peripheral circulation occur, they are the result and not the cause of heart failure."

CONCLUSION

Resting lactic acid values determined on blood drawn in the morning before rising show an initial slight rise over sea-level values on going to high altitudes. Sea-level values are found after acclimatization even at 6.14 km., where arterial saturations range between 55 and 70 per cent.

Standard work performances, on first going to high altitudes, produce greater rises in blood lactic acid than at sea level. After acclimatization lactic acid values similar to those at sea level are found for a given performance. The ability to perform work is lessened progressively with increase in altitude, hence also the ability to accumulate lactic acid. Only slight increases over rest values of lactic acid are found during work at 6.14 km.

The inability to accumulate large amounts of lactic acid at high alti-
tudes suggests a protective mechanism preventing an already low arterial saturation from becoming markedly lower by shift of the \( O_2 \) dissociation curve through acid effect. It may be that the protective mechanism lies in an inadequate oxygen supply to essential muscles, e.g., the diaphragm or the heart.

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