THE ACID-BASE EQUILIBRIUM OF THE BLOOD IN EXERCISE

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An increased blood lactate in humans during exercise is accompanied by a decrease in base bound as bicarbonate consequently causing a decrease in the CO₂ combining capacity of the blood. The reaction

\[ \text{BHCO}_3^- + \text{Lactic acid} \rightarrow \text{B(lactate)} + \text{H}_2\text{CO}_3 \]

and the associated extra output of CO₂ through the lungs acts as one of the principal buffering mechanisms of the body. However observations upon the relation of the magnitude of changes in lactate and CO₂ capacity have been contradictory. Mellanby and Thomas (1920) and Evans (1922), by addition of lactic acid to drawn blood, found that the decline in CO₂ content was less than the increase in blood lactate. Results of similar experiments performed in this laboratory have shown close agreement yet the picture is not identical with that seen in blood drawn after exercise. In six observations on blood drawn after exercise, Barr, Himwich and Green (1923) obtained wide variations and found a greater change in blood lactate than in CO₂ capacity in only two cases. Dill, Talbott and Edwards (1930) found in general a greater decline in CO₂ capacity of the blood. Dennig et al. (1931) found approximately equal changes when the blood lactate rose to 10 mEq. per liter. Robinson and Harmon (1941) found that the decreases in CO₂ capacity, at physiologically high concentrations of blood lactate, were smaller than the corresponding increases in lactate.

By further study of this problem we have attempted to relate changes in blood lactate, CO₂ capacity, and serum pH and to determine the rôle of the various mechanisms in buffering acid as it is accumulated during exercise. Samples of blood were drawn from human subjects in the basal state and after exercise on the same day. Various intensities of exercise, which consisted of running on a motor driven treadmill or in competitive races, were used to produce different concentrations of blood lactic acid in the men. For comparison of changes in pH, CO₂ capacity, lactate, and related changes in available base, arterial blood samples were drawn under oil and treated with heparin. Several samples of venous blood were used in the comparison of the variations in lactate concentration with those of CO₂ capacity. Blood lactate was determined by the method of Edwards (1938), and plasma protein by micro Kjeldahl analysis. HbO₂ and CO₂ capacity were determined by equilibration of blood with O₂ and CO₂ pressures of 200 and 40 mm. Hg respectively at 37°C. as described by Dill in Henderson’s book (1928). Analyses of blood samples for both content and capacity of HbO₂ and CO₂ were done on the Van Slyke apparatus. The pH values of arterial blood samples were calculated by means of the Henderson-Hasselbalch equation and some over the entire range of values

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obtained were checked by the electrometric method as described by Dill, Daly and Forbes (1937). The two methods checked each other very closely. The term "CO₂ capacity" as used here is defined as the CO₂ content of oxygenated whole blood at 37°C and 40 mm Hg CO₂ tension.

Results. Figure 1 reveals a distinct relationship between the increase in lactate (Δ lactate) and the decrease in CO₂ capacity (Δ CO₂ capacity). Increases of lactate up to 4 mEq. per liter are accompanied by approximately equivalent decreases in CO₂ capacity. In this range almost all of the base used in neutralization of the acid is obtained from base bound as bicarbonate. However, as the concentration of base bound as bicarbonate is decreased beyond this point by higher lactate concentrations the ratio of \( \frac{\Delta \text{CO}_2 \text{ capacity}}{\Delta \text{lactate}} \) becomes progressively smaller. With the accumulation of lactic acid the pH of the blood decreases and approaches the isoelectric point of the blood proteins decreasing their base binding capacity and releasing base for neutralizing the acid. Thus as the concentration of lactic acid becomes higher the plasma proteins and hemoglobin account for a greater fraction of the base used to neutralize the accumulated acid.

Calculations of changes in the arterial blood samples have revealed the relation of increases in lactate concentration to decreases in available base from bicarbonate, cell proteins, and plasma proteins. Decreases in base bound as bicarbonate in the whole blood (Δ BHCO₃)ₙ were calculated from values of the
CO₂ tension and content. Base contributed by plasma proteins (Δ BPₚ) has been calculated from the data of Van Slyke, Hastings, Hiller and Sendroy (1928).

\[ \text{BP}_p = 0.104 \text{ (gram protein)} (\text{pH}_e - 5.08) \]

Grams of plasma proteins per liter of blood were calculated from protein analysis of plasma and hematocrit determination of plasma and cell volumes. Similarly the decrease in base bound by protein in the cells (Δ BPₚ) has been calculated from the formula for oxygenated cells derived by Dill, Edwards and Consolazio (1937).

\[ \text{BP}_c = \text{HbO}_2 [ -0.5 (\text{pH}_c)^2 + 10.625 \text{pH}_c - 48.46 ] \]

In Table 1 have been tabulated the changes found in 15 arterial blood samples drawn after exercise. The increases in blood lactic acid (Δ lactate) are about equivalent to corresponding combined decreases in available base according to the equation

\[ \Delta \text{ lactate} = \Delta (\text{BHCO}_₃)_b + \Delta \text{BP}_p + \Delta \text{BP}_c \]

This relationship is represented in figure 2.
Figure 3 shows changes in the pH of arterial blood serum (\(\Delta pH_a\)) as related to \(\Delta\) lactate and \(\Delta CO_2\) capacity. Throughout the range of lactate concentrations studied the ratio of \(\frac{\Delta pH_a}{\Delta\text{ lactate}}\) is almost constant being only slightly greater at high values of lactate. In contrast the ratio of \(\frac{\Delta pH_a}{\Delta CO_2\text{ capacity}}\) steadily increases as the \(CO_2\) capacity is lowered. This again demonstrates the decreased buffering action of bicarbonate as the concentration is lowered.

The maximum \(\Delta pH_a\) of \(-0.40\) pH units was a decrease, measured in a well-trained athlete, from a basal \(pH_a\) value of \(7.37\) to a \(pH_a\) of \(6.97\) after work. Bock, Field and Adair (1923) and others have measured similarly low \(pH_a\) values in diabetic coma. It has been our experience that lactate values of \(22\) mEq. per liter are not uncommon in trained athletes after hard races. In such cases the arterial \(pH_a\) probably drops to about \(7.0\) yet these athletes show no ill effects aside from a breathlessness after the race from which they soon recover.

**SUMMARY**

Blood samples drawn from human subjects in the basal state and after various intensities of exercise were analyzed for \(O_2\) and \(CO_2\) capacity, lactic acid, plasma proteins, and serum pH. Comparison of increases in lactate concentration (\(\Delta\) lactate) and decreases in \(CO_2\) capacity (\(\Delta CO_2\) capacity) up to \(4\) mEq. per liter showed approximately equivalent changes. Beyond this point decreases in \(CO_2\) capacity became progressively smaller than corresponding increases in lactate concentration. Base for neutralization of the lactic acid at low concentrations was obtained principally from bicarbonate. At the higher lactate concentrations hemoglobin and plasma proteins accounted for an increased fraction of this base. Decreases in serum pH of arterial blood (\(\Delta pH_a\)) varied directly with \(\Delta\) lactate and \(\Delta CO_2\) capacity. The maximum blood lactate value measured was \(22\) mEq. per liter with a corresponding serum pH of \(6.97\).

**REFERENCES**

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