Last Word on Point: Counterpoint: Lactic acid is/is not the only physicochemical contributor to the acidosis of exercise

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TO THE EDITOR: We grounded our positions on arguments and quantitative considerations (2) while the counterpoint supporters often present only statements. Beginning with Van Slyke, we studied a bulk of acid base literature, therefore it is somewhat amusing that, according to Lindinger and Heigenhauser (4), “choose to ignore more than a century of physical, chemical, and physiological science.” And where are we “discarding the laws of mass action and electroneutrality”? Also we have not written “that intracellular Cl− repels lactate influx into erythrocytes”; this is the effect of the Donnan potential.

Some contributions show that Stewart’s conception is not helpful for understanding acidosis despite comparable results as with the traditional approach when calculating pH (3). One example is the production of ATP by the reaction between creatine phosphate and ADP consuming one H+., which is again liberated during splitting of ATP (6): CrP2− + ADP3− + H+ → Cr + ATP4−; ATP4− + H2O → ADP3− + HPO42− + H+.

But the buffer HPO42− (pK = 6.8) binds H+. Consequently pH rises, there is no contribution to exercise acidosis. In contrast to this traditional explanation fitting biochemical reaction equations, the formalistic Stewart approach describes a reduction of strong anions causing alkalosis (how?, which chemical reaction consumes H+?). And appearance of the weak acid H3PO4, which partly dissociates thus attenuating alkalosis. This latter reaction does not occur in reality. Also the lacking acidosis during exercise in McArdle patients cannot be attributed to a lower ATP production (Prakash, Ref. 5). If that is true, untrained subjects will not develop an acidosis as well.

One author (Gladden, Ref. 5) speculates about effects of changes in water properties on [H+] but basic chemistry explains dissociation of H+ from an acid by the molecular structure determining the distribution of charges and some influences of surrounding solved charges (ionic strength). This is a really mechanism-based approach also taking into account electroneutrality.

Some remarks in the letters are not correct. We do not ignore the effect of PCO2 (Rowlands, Ref. 5) as declared in the introduction of our contribution. Although PCO2 decreases in arterial blood, an increase in muscle and capillary/venous blood is not under dispute; we have therefore focused on nonrespiratory acidosis. The quotation “more (sic!) La− than H+ leaves the fibers but this effect is probably caused by temporary changes in extracellular buffer concentration . . .” in Jones’s (5) contribution is incomplete, correctly it is “During exercise apparently even more La− . . .” (1). After stopping exercise, when the extracellular volume normalizes, there results an almost 1:1 relation for H+ and La− entering or leaving the interstitial fluid.

Of course, in such a debate the positions are somewhat exaggerated. Fortunately, our controversy is not essentially based on differences in measurements but in calculation or interpretations. CO2 plays a role and small amounts of pyruvic and fatty acids are formed. Phosphate effects depend on actual pH. Shifts across membranes influence the amount of H+ in different compartments but cannot create new acids considering the whole body. Lactic acid remains the main cause of nonrespiratory acidosis.

REFERENCES