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Review Paper

ACID-BASE AND ION REGULATION DURING EXERCISE WITH EMPHASIS ON HORSES

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Summary: Exercise induced intracellular and extracellular acidosis requires a number of homeostatic adaptations to return acid base status to its resting level. The purpose of this review is to briefly describe the quantitative approach to these homeostatic adaptations, and to describe intracellular (intramuscular and intraerythrocyte) and extracellular (plasma) ion kinetics during exercise. Special consideration is given to horses.
Ions and carbon dioxide (CO_2) movement between muscle and plasma and output of CO_2 by the respiratory system play a critical role in acid base homeostasis. The skeletal muscle acidosis is largely driven by a fall in intramuscular strong ion difference due to increase in intramuscular lactate concentration (La^-) and reduction in intramuscular potassium concentration (K^+). The increase in intramuscular hydrogen ion concentration is buffered by a reduction in creatine phosphate concentration (CrP^{2-}), and by a small change in the apparent equilibrium constant (K_A) for weak acid buffers. Diffusion of La^- into venous blood and its associated metabolism, re-uptake of K^+, and diffusion of CO_2 from muscle and its pulmonary elimination contribute to the resolution of acidosis. CrP^{2-} is resynthesized, and K_A reverts to its resting value.

Key words: exercise - physiology; muscles - physiology; acid - base equilibrium; acidosis, lactic; horses

Introduction

Rapid increases in ATP turnover during intense, short-term exercise result from substrate and oxidative phosphorylation (1, 2, 3, 4, 5, 6), which causes a series of metabolic and ionic events that contribute to changes in acid base status. Ion and CO_2 movement between muscle and plasma play an essential role in that process. Metabolic processes driven by exercise necessitate rapid increases in gas exchange within the active skeletal muscle and across the lung. They decrease (mildly) intramuscular/intracellular bicarbonate ($_m[HCO_3^-]$) and increase intracellular hydrogen ion concentration ($_m[H^+]$). Changes in $_m[HCO_3^-]$ and $_m[H^+]$ are due to increases in intracellular lactate concentration ($_m[La^-]$) and loss of intracellular potassium concentration ($_m[K^+]$), which reduces the intracellular strong ion difference ($_m[SID]$), thereby generating carbonic acid (1, 7).

Most ionic events during exercise share great similarities between species studied. Horses are specially adapted to exercise and have been extensively studied for acid base physiology. Only differences in acid base regulation that are very particular to horses are emphasized in the text below.

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A quantitative approach to acid-base chemistry

The application of a physicochemical approach to the regulation of acid-base status in intra- and extracellular space clarifies the links between fluid and electrolyte control and the physiological and biochemical events occurring during exercise in muscle, circulation, and the lungs (8, 9, 10). It quantifies the relative contributions of three independent variables: strong ion difference (SID), weak electrolyte concentrations (A_{tot}), and the partial pressure of CO_2 (pCO_2) to changes in dependent variables ($[H^+]$, $[HCO_3^-]$) in aqueous solutions. Changes in $[H^+]$ can be achieved only by changing one or more of these three independent variables. The system is constrained by three fundamental physical laws: conservation of mass (equations 2 & 9), electro-neutrality (equation 12) and the equilibrium constraints on dissociation reactions (equations 5 - 8).

Strong ions are by definition electrolytes that, based on their K_A, completely dissociate in physiological aqueous solutions at physiological $[H^+]$. The net effect of the presence of strong ions can be expressed in terms of the difference between the total concentration of strong base cations and strong acid anions. This is termed strong ion difference (SID):

$$[SID^+] = \Sigma\ [\text{strong cations}] - \Sigma\ [\text{strong anions}] \quad (1)$$

Weak electrolytes are only partially dissociated in H_2O at physiological $[H^+]$. A_{tot} is used to express the total available anionic charge of the weak electrolytes, which consist of associated (HA) and dissociated (A^-) forms:

$$[A_{tot}] = [A^-] + [HA] \quad (2)$$

Carbon dioxide, a major end product of cell metabolism is, under physiological temperature, $[H^+]$, and pressure, moderately soluble in H_2O. It also reacts with H_2O to form several other solute compounds, all of whose concentrations are dependent variables. The amount of dissolved CO_2 (dCO_2) is directly proportional to its partial pressure (pCO_2) in the gas phase and its solubility coefficient (SCO_2):

$$dCO_2 = SCO_2\ (pCO_2) \quad (3)$$

During exercise CO_2 moves down its partial pressure gradient from a working muscle into circulation and is then removed through the respiratory system. Dissolved CO_2 reacts with H_2O to form carbonic acid (H_2CO_3), which further dissociates into H^+ and HCO_3^- (hydration of CO_2); HCO_3^- then further dissociates to form H^+ and CO_3^{2-}. The process is catalyzed by the enzyme carbonic anhydrase (CA):

$$H_2O + CO_2 \xleftrightarrow{CA} H_2CO_3 \leftrightarrow H^+ + HCO_3^- \leftrightarrow H^+ + CO_3^{2-} \quad (4)$$

The dissociation of the H_2CO_3 can also be formed as the mass action equation:

$$K_a\ [dCO_2] = [H^+] + [HCO_3^-] \quad (5)$$

Where K_a is the dissociation constant, incorporating the dissociation constants for hydration and dehydration of the CO_2. Based on Equation (3), the following can be substituted:

$$K_a\ [SCO_2\ (pCO_2)] = [H^+] + [HCO_3^-] \quad (6)$$

The dissociation constant incorporating hydration and dehydration of CO_2 (K_a) can be combined with SCO_2 to form the constant K_c:

$$K_c\ [pCO_2] = [H^+] + [HCO_3^-] \quad (7)$$

As mentioned above, HCO_3^- further dissociates to form H^+ and CO_3^{2-}:

$$K_3\ [HCO_3^-] = [H^+] + [CO_3^{2-}] \quad (8)$$

Where K_3 is the equilibrium dissociation constant for HCO_3^-.

H^+ homeostasis in physiological aqueous solutions is most readily described in H_2O:

$$H_2O \leftrightarrow OH^- + H^+ \quad (9)$$

The law of mass action further on transforms Equation 9:

$$Kw\ [H_2O] = [H^+]\ [OH^-] \quad (10)$$

The concentration of H_2O is 10^9 greater than $[H^+]$ and the dissociation constant of water (K_w) is small. Therefore, $[H_2O]$ itself can be considered a constant (K'_w):

$$K'w = [H^+]\ [OH^-] \quad (11)$$

K'_w changes with temperature; therefore, temperature changes will manipulate the $[H^+]$. The increase in temperature will increase $[H^+]$, and vice versa.

Based on the information above water interacts with the weak electrolyte system (Equation 2), as well as the CO_2 system (Equations 7 and 8). It will also interact with strong electrolytes:

$$[SID] + [H^+] - [HCO_3^-] - [A^-] - [CO_3^{2-}] - [OH^-] = 0 \quad (12)$$

The above equations can be rearranged and combined into a single equation for $[H^+]$ in terms of independent variables and the equilibrium constants of each system that interacts in the solution:

$$[H^+]^4 + (K_A + [SID])\ [H^+]^3 + (K_A \times ([SID] - [A_{tot}]) - (K_C \times pCO_2 + K'w))\ [H^+]^2 - (K_A \times (K_C \times pCO_2 + K'w) + K_3 \times K_C \times pCO_2))\ [H^+] - (K_A \times K_3 \times K_C \times pCO_2) = 0 \quad (13)$$

In conclusion, in the above series of equations independent variables (SID, A_{tot}, pCO_2) interact with the concentration of four dependent variables (H^+, HCO_3^-, CO_3^{2-}, OH^-) employing three fundamental physical laws: conservation of mass, electro-neutrality, and equilibrium dissociation of weak acids and water (8, 9, 10, 11).

Ion regulation in exercise - intracellular events

The intramuscular acidosis develops mainly due to a large fall in $_m$[SID], secondary to increases in $_m[La^-]$ and reductions in $_m[K^+]$. Further, the $_m[H^+]$ is increased by a reduction in $_m[CrP^{2-}]$, and partly due to a large increase in $_mPCO_2$. Intramuscular A_{tot} may (12) or may not (7) contribute to the intracellular acid-base changes. The rise in $_m[H^+]$ is also modulated by a change in the K_A for weak acid buffers (7).

A rapid increase in $_m[H^+]$, decrease in $_m[K^+]$, and the accumulation of $_m[La^-]$ contribute to changes in sarcolemmal and transverse tubular membrane potential, which further alters the $_mCa^{2+}$ homeostasis and contractile function of skeletal muscle (12, 13, 14). K^+ release from skeletal muscle depends on the intensity of contraction and is proportional to number of action potentials per unit of time (15, 16, 17, 18). In skeletal muscle, K^+ kinetics are regulated by Na^+-K^+ ATPase and K^+ channels (19, 20). During muscle contraction the high rate of K^+ efflux cannot be compensated by slower inward K^+ transport by the Na^+- K^+ ATPase; therefore, voltage gated K^+ channels that open during the repolarization phase of the action potential must primarily modulate the high rate of K^+ efflux (19). ATP sensitive K^+ (K_{ATP}) channels located in the sarcolemma may contribute to the net loss of K^+ from muscle (19, 21, 22, 23). It was originally suggested that the K_{ATP} channels are only activated in metabolically exhausted muscle fibers (24) and that the activity of the channels contributes to the decrease in force during fatigue (22). However, the fact that glibenclamide, the selective inhibitor of K_{ATP} channels, has no effect on interstitial $[K^+]$ during exercise, suggests that K_{ATP} channels are not important for K^+ release during muscle contractions in human muscle (25). Another group of K^+ channels, the large Ca^{2+} activated channels, are reported to be present in rat skeletal muscle T-tubules (26). The function of these channels in muscle is unknown (25).

La^- is removed from skeletal muscle by a bidirectional monocarboxylate carrier that is responsible for 70-80% of the La^- flux. La^- diffusion as undissociated lactic acid according to the prevailing transmembrane $[La^-]$ and $[H^+]$ gradients across the plasma membrane and nonspecific anion exchange, plays a minor roles in La^- removal from skeletal muscle (27, 28, 29, 30, 31).

While the intramuscular proton-buffering mechanisms contribute over the short term to the regulation of $_m[H^+]$, its restoration after exercise depends on the recovery of $_m[K^+]$ and on the removal of $_mLa^-$. Hence diffusion of La^- into venous blood and its associated metabolism, re-uptake of K^+, and diffusion of CO_2 from muscle and its pulmonary elimination accomplish the resolution of acidosis. CrP^{2-} is re-synthesized, and K_A reverts to its resting value (1, 6, 32). However, based on a quantitative approach to acid-base chemistry the recovery from intracellular acidosis develops as an interaction between SID, A_{tot}, and pCO_2 between the intracellular (ICF) and extracellular fluid (ECF), resulting in restoration of normal $_m[H^+]$. The restoration of $_m[H^+]$ occurs at the expense of increasing the $[H^+]$ in interstitial fluid, plasma, and erythrocytes. Plasma and erythrocytes contribute to recovery by redistributing CO_2 to the lungs and SID to other tissues. It may appear that erythrocytes provide a first line defense within the blood to attenuate the large and abrupt increase in plasma $[La^-]$ and $[H^+]$ that occur with high-intensity exercise (33, 34, 35, 36). However, with regards to $[K^+]$, erythrocyte $[K^+]$ ($_e[K^+]$) in studies by McKelvie et al. (33) and Lindinger et al. (34) was calculated from whole blood and plasma K^+ content as well as hematocrit. When $_e[K^+]$ is established from direct measurements of K^+ content in a fixed volume of sedimented (packed) cells, the changes in $_e[K^+]$ of arterial and venous blood during exercise appeared to be due to water shifts and not due to fluxes of K^+ between erythrocytes and plasma (37, 38). It has been proposed that contracting muscle and, partially, the inactive tissue, can take up K^+ probably by a combination of K^+ and hormone activation of the Na^+- K^+ pump (38). However, it appears that Juel *et al.* (38) neglected the importance of erythrocyte volume (EV), as changes in EV will influence the hematocrit and therefore [ion] (33).

Ion regulation in exercise - intravascular/extracellular events

Changes in plasma [SID] occur as a consequence of fluid shifts between different compartments, and exchange of strong ions across the capillary and erythrocyte membrane. Changes in plasma volume between the vascular and extravascular compartments are forced by changes in hydrostatic and osmotic forces acting between these compartments. The increase in the plasma $[K^+]$ may be contributed to a decrease in plasma volume and an efflux of $[K^+]$ from the active skeletal muscle (33, 34, 37, 38). Plasma $[La^-]$ changes are balanced between release of La^- into circulation from the active muscle, and its uptake and metabolism (34, 39). Na^+ and Cl^-, despite their relative increase in plasma during exercise, seem to move out of the vascular space, as the relative increases in plasma is less than that predicted from the change in plasma volume only (34, 39).

In recovery plasma $[K^+]$ ($_p[K^+]$) decreases promptly and $_p[Na^+]$ increases as a consequence of a high rate of Na^+- K^+ ATPase activity in skeletal muscle and other tissues. Plasma $[La^-]$ decreases gradually as La^- continues to move from skeletal muscle down its concentration gradient. The magnitude of La^- movement is governed by the intensity of muscle work (1, 40, 41, 42, 43, 44, 45, 46, 47, 48). In horses, erythrocytes have been suggested to function as a lactate sink (35, 36); up to 50% of horse blood lactate is in erythrocytes (35, 49, 50), whereas the corresponding value in man is 17% (51).

Changes in A_{tot}, an ion equivalent of the total available amino acids from proteins, also influence the plasma acid base status (10, 52). In horses the anionic equivalent for plasma proteins is 0.21 mmol/L of plasma protein (53). The increase in $_p[A_{tot}]$ during exercise is accounted for by the decrease in plasma volume (7, 52).

Conclusion

Several ionic events regulate the acid base equilibrium in living organism. Both, the intracellular and extracellular compartments must be regulated in an integrated manner to enable strong ion exchange between the muscle, plasma, and erythrocytes, which contributes to the resolution of acidosis. Quantitative approach to acid base clarifies the acid base chemistry by recognizing the acid base independent variables (SID, PCO_2, A_{tot}) and their influence on acid base dependent variables ($[H^+]$, $[HCO_3^-]$). It quantifies the contribution of particular ions or CO_2 to acid base homeostasis at rest and during exercise.

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REGULACIJA ACIDOBAZNEGA RAVNOTEŽJA IN RAVNI IONOV PRI TELESNEM NAPORU S POSEBNIM OZIROM NA KONJE

M. Vengušt

Povzetek: Pri telesnih obremenitvah (športu) nastala acidoza znotraj in zunaj celičnega prostora zahteva številne fiziološke prilagoditve za uravnoteženje acidobaznega sistema na fiziološki nivo, ki je značilen za mirovanje. Namen tega prispevka je opisati kvantitativen pristop k razumevanju acidobazne fiziologije pri telesnih obremenitvah in razložiti dinamiko ionov znotraj (eritrociti, mišična celica) in zunaj celice (plazma), s posebnim poudarkom na konjih.

Prehajanje ionov in ogljikovega dioksida (CO_2) med mišicami in plazmo ter odstranjevanje CO_2 z dihanjem igrata pomembno vlogo pri ohranjanju acidobaznega ravnotežja. Acidozo oz. povečano koncentracijo vodika ([H^+]) v skeletni mišici povzroči znižanje razlike v koncentraciji močnih ionov (SID). SID se zniža zaradi povečane koncentracije laktata (La^-) in znižane koncentracije kalija (K^+) v mišici. Preveliko povečanje [H^+] v mišici pri obremenitvah pa se ublaži z zmanjšanjem koncentracije kreatin fosfata (CrP^{2-}) in z manjšo spremembo navidezne ravnotežnostne konstante (KA) šibkih kislin. Prehod La^- in CO_2 iz mišice v vensko kri ter porast K^+ v mišicah po prenehanju obremenitve prispevajo k odpravi acidoze. Koncentracija CrP^{2-} se obnovi, KA pa se vrne na svojo izhodiščno vrednost.

Ključne besede: napor - fiziologija; mišice - fiziologija; acido - bazno ravnotežje; acidoza, laktatna; konji