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Acid-Base Balance and Electrolyte Concentration in Blood During Graded Exhausting Exercise in Non-Trained Subjects

J. M. González-Buitrago*, C. Martín**, X. Leibar***, F. J. Laso** and J. A. Navajo

Servicio de Análisis Clínicos
Hospital Virgen de la Vega
37007 Salamanca (Spain)

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Venous acid-base balance and electrolyte concentration during step-graded and exhausting exercise with a two-minute steady-state has been studied in a group of non-trained young men. The results showed a significant decrease in pH, pCO_2 and bicarbonate and a significant increase in lactate, potassium, inorganic phosphate and proteins during the exercise. The supervening acidosis had a large anion gap that was of proportion with the increase in lactate values. We suggest that the total sum of other anions such as proteins, phosphate, pyruvate, citrate, free fatty acids and aminoacids, as well as sodium-hydrogen exchange could account for this acidosis.

Key words: Acidosis, Anion gap, Standard base deficit, Bicarbonate, Lactate, Phosphate, Potassium.

ATP production during exhausting exercise rests mainly on anaerobic glycolysis with lactate formation, using muscle glycogen as precursor. Lactate production by skeletal muscle results in intracellular acidosis; in turn, lactate goes out to muscle cells and causes extracellular acidosis. Exhausting exercise acidosis was larger than that produced by lactic acid. This fact was associated with an inexplicably large anion gap (1, 4-6).

We have studied acid-base balance and electrolyte concentration during stepping and exhausting exercise with a two minute steady-state in a group of non-trained young men.

Materials and Methods

Subjects.— Seven male voluntary university students with a mean age of

* To whom all correspondence should be addressed.
** Departamento de Medicina. Hospital Clínico Universitario. 37007 Salamanca (Spain).
*** Departamento de Ciencias Biomédicas. IVEF-EKI. Vitoria (Spain).

21.6±0.5 years, a mean weight of 73.7±5.8 kg and a mean height of 179±8 cm were studied. All of the subjects had normal renal function, did not smoke or drink, and had no history of cardiorespiratory disease. Although the subjects performed sports sporadically, none of them had a systematic training. Informed consent was obtained from all subjects before the study.

Procedures.— The studies were performed early in the morning. After an eight-hour fasting period, a catheter was introduced in the antecubital vein. After a 30 min period of rest in a recumbent supine position the subjects began bicycle exercise on an electronically braked ergometer while attached to a cardiac monitor; each workload was performed for three minutes, and workloads were increased in increments of 25 W, with an initial workload of 25 W. Exercise finished when the subject considered himself exhausted and in every case after three minutes of bicycling with a workload of 200 W at least. At the end of exercise period, the subjects rested during a 30-min recovery period in a supine position. Heart rate, blood presure and EKG were monitored during the exercise and the recovery period. Blood samples were obtained at the beginning of the exercise, at 6 and 12 minutes of exercise, at the end of exercise (peak) and 5, 13, 21, and 30-min during the recovery period.

pH, pCO_2 and pO_2 were measured at rest, at peak exercise and at the end of recovery period in heparinized venous blood. Sodium, potassium, chloride, lactate, phosphate and total proteins were measured in every blood sample obtained. Lactate was measured in heparinized plasma centrifuged immediately after the extraction, while sodium, potassium, chloride, phospate and proteins were measured in serum.

Blood pH, pCO_2 and pO_2 were determined on an AVL 945 (AVL AG Schaffhausen, Switzerland) pH and blood gas analyzer. Bicarbonate and standard base deficit [SBD, or negative standard base excess (9)] concentrations were calculated from pH and blood gases using a pKa = 6.10, a CO_2 solubility coefficient of 0.031 mM/mmHg and assuming a hemoglobin concentration in the extracellular fluid of 50 g/l.

Plasma lactate concentration was determined with an enzymatic method (Monotest Lactate, Boehringer Mannheim), in a VP automatic analyzer (Abbott Laboratories). Serum potassium and sodium concentration were measured in the flame photometer Corning 490 (Corning Medical, Medfield MA, USA), that uses lithium as internal standard. Serum chloride concentration was measured by coulometric titration in the Corning 925 analyzer. Serum phosphate concentration was measured by molibdate UV method, with sample blank, in a Hitachi 705 automatic analyzer (Boehringer). Serum total protein concentration was measured by the biuret method in the Hitachi 705. Anion gap was calculated by the formula ($[Na^+] + [K^+]$) - ($[HCO_3^-] + [Cl^-]$). Serum anionic protein concentration was estimated by the formula in mmol/l = 10.3 (pH − 5.66) × (prot in g/dl) (3).

The change in plasma volume (PV) during the exercise was calculated from values for hemoglobin (Hb) and hematocrit (Hct) using the method of DILL and COSTILL (2).

$$BV_A = BV_B\ (Hb_B/Hb_A)$$

$$CV_A = BV_B\ (Hct_A)$$

$$PV_A = BV_A - CV_A$$

$$\Delta PV,\% = 100\ (PV_A - PV_B)/PV_B$$

BV is blood volume and CV is cell volume; the subscripts B and A refer to be-

fore and after exercise, respectively, and BV_B was taken as 100.

Statistics.— All values are expressed as means ± SD. Changes from pre-exercise values were analyzed with Student's t-test for paired observations.

Results

Values for venous acid-base status and lactate concentration before beginning exercise, at peak of exercise and 30 min after finishing exercise are shown in table I. Venous pH decreases during exercise and is still below basal values 30 min after finishing exercise. At the peak of exercise venous pCO_2 has a lesser value than that at the beginning of exercise; 30 min after finishing exercise pCO_2 value is almost the same as the basal value. Bicarbonate concentration, calculated from pH and pCO_2, shows a significant decrease from basal value to peak value. Bicarbonate value 30 min after finishing exercise is similar to basal value.

Plasma lactate levels increase during exercise; during rest, after finishing exercise, lactate values decrease slowly and 30 min after finishing exercise the mean value is significantly higher than the basal one.

Percent change in plasma volume, electrolyte concentrations, anion gap (AG), protein concentration and concentration of anionic protein during exercise and during rest are shown in table II. Serum sodium concentration increases during exercise but this increase is not significant. Serum sodium concentration decreases to basal values 10 min after finishing exercise. Serum potassium concentration shows a significant increase during exercise. When exercise stopped, serum potassium fell abruptly to levels equal to rest. Serum chloride concentration increases with exercise but this increase is not significant. Basal values are reached immediately after finishing exercise. Serum inorganic phosphate shows a significant increase from the basal value to peak of exercise. Peak values decrease slowly to reach basal values 30 min after finishing exercise. Anion gap increases significantly during exercise. Basal values are reached 30 min after finishing exercise. Plasma protein concentration increases with exercise; the increase is 15.2 % more than basal values. Plasma protein concentration decreases slowly during rest to reach basal values. Anionic serum proteins concentration does not change significantly during exercise and rest.

Discussion

Exhausting exercise causes a shift of fluid out of the vascular space. The mean percent fall in plasma volume after exercise was greater than the increase of sodium and chloride concentrations, and

Table I. *Venous acid-base status and lactate concentration before beginning exercise, at «peak» exercise and 30 minutes after finishing exercise (n = 7).*

Date are mean ± SD. SBD is standard base deficit. Significance comparing pre-exercise: * $p < 0.001$; ** $p < 0.01$; *** $p < 0.05$.

	Pre-exercise	«Peak»	Post-exercise
pH	7.405 ± 0.024	7.245 ± 0.035*	7.368 ± 0.037***
pCO_2 (mmHg)	42.6 ± 2.9	33.1 ± 3.2*	40.2 ± 2.1*
HCO_3^- (mmol/l)	25.8 ± 0.7	14.4 ± 1.8*	24.0 ± 2.1*
SBD (mmol/l)	−1.53 ± 0.71	11.41 ± 3.16*	1.20 ± 0.80***
Lactate (mmol/l)	1.65 ± 0.38	8.02 ± 1.20*	3.78 ± 1.12**

Table II. *Plasma volume variation (ΔPV), electrolyte concentrations, anion gap (AG), protein concentration and concentration of anionic protein during exercise and during rest (n = 7).*

Data are mean ± SD. Significance comparing pre-exercise: * $p < 0.001$; ** $p < 0.01$; *** $p < 0.05$.

	ΔPV (%)	Na^+ (mmol/l)	Cl^- (mmol/l)	K^+ (mmol/l)	AG (mmol/l)	Phosphate (mmol/l)	Protein (g/l)	Anionic protein (mmol/l)
Pre-exercise		137.3 ± 3.7	98.4 ± 3.6	3.80 ± 0.12	16.7 ± 1.2	1.03 ± 0.10	69.9 ± 4.9	12.56 ± 1.05
Exercise								
1		137.3 ± 3.4	98.4 ± 3.6	4.14 ± 0.13*		1.06 ± 0.14	73.1 ± 3.0	
2		138.8 ± 3.3	100.1 ± 3.5	4.39 ± 0.24*		1.15 ± 0.15***	75.5 ± 2.7***	
3 (peak)	−6.0 ± 4.0	140.8 ± 3.5	100.2 ± 3.6	4.74 + 0.20*	28.8 ± 2.5*	1.45 ± 0.18*	80.5 ± 2.8*	13.22 ± 0.76
Rest								
1		136.8 ± 6.0	99.0 ± 3.7	3.89 ± 0.26		1.31 ± 0.19**	76.5 ± 2.3*	
2		137.6 ± 3.7	98.3 ± 3.7	3.89 ± 0.18		1.22 ± 0.19**	72.8 ± 1.7**	
3		137.4 ± 3.9	98.0 ± 3.9	3.89 ± 0.21		1.15 ± 0.15**	70.4 ± 2.2	
4	0.5 ± 1.1	137.6 ± 4.1	97.8 ± 3.6	3.86 ± 0.17	19.7 ± 1.7*	1.07 ± 0.15	69.9 ± 3.6	12.42 ± 0.92

lesser than the increase in concentration of the other measured quantities.

Exhausting exercise leads to intramuscular lactic acidosis; lactate escapes to the blood causing extracellular acidosis. The total acid load in the extracellular fluid was calculated as standard base deficit using an average hemoglobin concentration of 50 g/l and assuming that the acid load was distributed in the extracellular space. In this study total plasma acid load was greater than could be accounted for by lactate. During exercise standard base deficit increased by 12.94 mmol/l, while plasma lactate concentration increased by 6.37 mmol/l. That is, plasma bicarbonate concentration decreased more than lactate concentration increased. Muscles appear to release hydrogen ions independently from and in excess of lactate (7, 8).

The anion gap provides information about ionic balance. The anion gap expresses the negative protein charges, phosphate and organic acids like lactate, pyruvate, citrate, free fatty acids and aminoacids. The increase of anion gap (12,1 mmol/l) is much greater than the increase of lactate (6.37 mmol/l); the increment of anion gap unexplained by lactate (5.73 mmol/l) appears to correspond roughly to the difference between base deficit and lactate concentration (6.57 mmol/l). The increased standard base deficit (SBD) unexplained by lactate may correspond to other anions or to a sodium/hydrogen ion exchange between blood and tissues. To evaluate both alternatives we have calculated the changes in anionic protein charges and measured electrolyte concentrations in blood plasma during exercise and during the recovery period.

Two factors affect the negative plasma protein charges during exercise. On one side, the net protein charge decreases secondarily to pH decrease. On the other side, increased protein concentration produced during exercise due to hemoconcentration increases the negative charges. The concentration of anionic serum proteins undergoes a net increase of 0.66 mmol/l, not large enough to give negative charges.

As far as sodium/hydrogen ions exchange is concerned, there appears to exist a serum sodium decrease when hemoconcentration is considered. However, serum sodium concentration changes paralleled chloride serum concentration changes (table II). Thus, parallel movements of these ions will not affect the anion gap.

In addition to proteins, the other anions which may increase during exercise are phosphate and organic ions such as pyruvate, citrate, free fatty acids and aminoacids. Phosphate changes during exercise are high, but the net negative charges increase at peak of maximal effort is 0.42 mmol/l, clearly lower than anion gap. Hyperphosphatemia probably results from a combination of hydrolysis of cellular phosphate esters during acidosis and a shift of this inorganic phosphate into the extracellular fluid. The normalization of the serum phosphate levels during rest suggests that extracellular phosphate shifted into cells during correction of acidosis.

Preliminary results indicate that during exhausting effort the increase in pyruvate, citrate and free fatty acids are around 0.5 mmol/l each. Therefore, total negative charges comprising anionic proteic charges, phosphate, pyruvate, citrate and free fatty acids is about 2.5 mmol/l, far away from the 6 mmol/l value of negative charges not ascribed to lactate. However, it must take into account that this value has been obtained from 9 measured or estimated quantities, each with its own errors.

In conclussion, acidosis induced during step-graded and exhausting effort with a 2-minute steady-state produces more hydrogen ions than lactate in the blood. There is the likelyhood that the total sum of other ions such as proteins, phosphate, pyruvate, citrate, free fatty acids and aminoacids, as well as hydrogen ions-sodium exchange coadjuvate in acidosis being

greater than that simply explained by the increase in lactate concentration.

Resumen

Se estudia en hombres jóvenes el balance ácido-base y la concentración de electrolitos en vena durante un ejercicio graduado y extenuante con un estado estacionario de dos minutos. Durante el ejercicio se produce un descenso significativo del pH, la pCO_2 y el bicarbonato y un aumento significativo de lactato, potasio, fosfato inorgánico y proteínas. La acidosis observada se acompaña de una laguna aniónica mayor que la justificada por el incremento de los valores de lactato. Se sugiere que la suma total de otros aniones como las proteínas, fosfatos, citrato, ácidos grasos libres y aminoácidos podrían explicar esta acidosis.

Palabras clave: Acidosis, Laguna aniónica, Déficit de base estándar, Bicarbonato, Lactato, Fosfato, Potasio.

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