The Fate of Potassium Ions Released from Contractile Muscle during Repeated Supramaximal Exercise

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Keywords
supramaximal exercise, potassium ion, acidification, blood compartments, renal secretion

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Figures: 7
References: 14

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Background

Intense muscle contractions causes considerable changes in the intracellular concentration of electrolytes. In working muscle cells, each action potential is associated with an influx of Na+ from extracellular compartment into inwards of cells and an efflux of K+ from cytoplasm to extra cellular fluid [1].

The release of K+ from muscle leads to a decrease in membrane potential and it is considered as an important factor in the development of muscle fatigue. It is obvious that the restoration of Na+-K+ gradients between extra- and intracellular compartments is necessary before the subsequent action of a muscle. Restoration in muscle cells of Na+ and K+ gradients can be done by Na+-K+ pump’s action. The Na+-K+ pump (Na+-K+ - ATP-ase) action is the major mechanism allowing accumulation of K+ in cells and Na+ in the extracellular fluid. But the Na+-K+ pump activity requires ATP for active transportation of ions. The active removal of Na+ from the cell and the active uptake of K+ into cells depends on the activity of Na+-K+ - ATP-ase [2, 3].

During the muscle contraction, which uses up a high rate of energy, the locally available ATP supply may be limited to the extent that ion transportation and other cellular processes demanding energy cannot continue their activity at proper rates. Due to a decreased activity of Na+-K+ - ATP-ase, the rapid increase in K+ concentration in plasma occurred as a response to muscular contraction. It is known that K+ efflux achieves values 7–16 mmol/g wet tissue per action potential [4]. The extracellular fluid K+ concentration is normally in the range of 3.35–4.45 mmol/l. Increased concentration of plasma K+ is a threat to the resting membrane potential. Under these circumstances the depolarization of the resting potential can take place, which, by itself should increase neuromuscular excitability.

It is interesting that an increased level of plasma K+ is not a long-lasting phenomenon, but following continued exercise, concentration of plasma K+ rapidly returns to the resting values.

In such a case, the question appears what mechanism regulates such a rapid distribution of potassium ions in blood and where they are accumulated or removed.

The purpose of this paper was to examine the fate of K+ released from contracting muscles during supramaximal exercise repeated three times, which is known to be associated with a large efflux of K+ and lactate into plasma.

Blood may be considered as a two-compartment system comprised of red blood cells (RBC) and plasma in a ratio about 45%: 55 % at rest. Some authors proved active transportation of K+ through cells membrane of erythrocytes [5, 6].

In our work we investigated the fate of K+ released from contracting muscles during supramaximal exercise, taking in consideration:

- the contribution of erythrocytes to the control of the plasma concentration of K+ under exercise and postexercise conditions,
- the renal secretion of K+ to urine.

Material and Methods

Nineteen healthy young physically active men volunteered for the study. The mean (±SE) age, height and weight for the group were respectively: 22.6±1.77 yr, 180.9±5.8 cm and 89.2±11.7 kg (Table 1). The Ethical Committee of Scientific Research at the University of Medical School approved of the experimental protocol.

All experiments were conducted in the morning. Exercise was performed on a mechanical, Monark cycloergometer with automatic counting of pedal revolution. Subjects performed a warm-up lasting 5-min. before the specific test, to promote specific physiologic and motor adaptation.
After 5-min. rest, subjects performed the specific 30-s test (Wingate anaerobic test) repeated three times with 7-min. rest between each exercise bout. During the test subjects pedalled as fast as possible against a resistance of 0.075 kp per kg body mass. The maximal speed was maintained through the 30-s period.

During the test important indices characterizing anaerobic capacity were measured. Blood samples were collected in heparinized capillary tubes at rest and immediately after each exercise bout and 15-min. after the final exercise bout. Blood samples were stored on ice for analysis of: \( \text{Na}^+ \), \( \text{K}^+ \), \( \text{Cl}^- \) concentration in plasma and whole blood, acid-base balance parameters and hematocrite. Ions concentrations in plasma and parameters of blood acid-base balance were measured immediately after blood collection. Samples of blood were frozen and thawed three times to lyse erythrocyte cell membranes before determination of \( \text{Na}^+ \), \( \text{K}^+ \), \( \text{Cl}^- \) concentration in whole blood. Ions concentration in plasma and lysed whole blood extracts were measured using ion selective electrodes (Ciba-Corning 644 \( \text{Na}^+ /\text{K}^+ \). \( \text{Cl}^- \) Analyzer). Parameters of blood acid-base status were determined by using pH/oxygen Ciba-Corning Analyser 238.

Whole blood and plasma ion concentrations were used to calculate the ion concentration of erythrocytes [7]. Urine was collected during 24 h before the exercise and 1 h, and 24 h after the completion of the last exercise bout. Urine volume was measured and urinary potassium ion concentration was determined by using ion-selective electrode in Ciba- Corning 644 \( \text{Na}^+ /\text{K}^+ \). \( \text{Cl}^- \) Analyzer.

All data are presented as means with SEM. The statistical significance of difference was ascertained using Student's \( t \) test for paired observations.

**Results**

Tab. 1. Characteristics of the examined subjects. Changes in absolute values for peak power (PP), total work (TW) and fatigue index (FI) during three times repeated 30s Wingate Anaerobic Test

<table>
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<td>25</td>
<td>75</td>
<td>796</td>
<td>740</td>
<td>879</td>
<td>18.47</td>
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<td>J.T.</td>
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<td>1090</td>
<td>1004</td>
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<td>K.Ł.</td>
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<td>1225</td>
<td>1100</td>
<td>1000</td>
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<td>1094</td>
<td>1100</td>
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<td>891</td>
<td>19.71</td>
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<tr>
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<td>720</td>
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<td>579</td>
<td>17.52</td>
<td>18.09</td>
<td>16.81</td>
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<td>895</td>
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<td>16.86</td>
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<td>188.49</td>
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</table>
**Performance.** During the repeated 30s Wingate anaerobic test values of Peak Power decreased from 967.95 W in the first bout to 959.2 W and 900.32 W in the second and the third bouts of exercise, respectively (Table 1). The same effect could be seen in the amount of total work. Values of total work were reduced from 21.99 KJ in the first bout to 20.72 KJ in the second repetition of the Wingate Test (Table 1).

**Potassium ions concentrations:** $K^+$ concentration in whole blood increased significantly from 72.33±7.54 mmol/l at rest to 80.27±6.12 mmol/l after three repetitions of the Wingate Test. Changes were statistically significant ($p=0.007$) (Fig. 1). After 15 min of recovery, the whole blood $K^+$ concentration decreased almost to the rest values.

Plasma $K^+$ concentration increased from 4.75 mmol/l at rest to 6.26 mmol/l after the first bout, but during 7 min recovery returned to normal. At the end of the second exercise bout and the third one plasma $K^+$ concentration remained significantly elevated comparing with resting values (Fig. 2).

![Fig. 1. Changes in blood potassium ion concentration induced by exercise test](image1.png)

![Fig. 2. Changes in plasma potassium ion during exercise test and recovery](image2.png)

![Fig. 3. Erythrocyte potassium ion concentration correlated with blood potassium ion concentration](image3.png)

Potassium concentration in erythrocytes increased during exercise and is correlated with potassium ion concentration in blood ($p=0.00002$, $r=0.8139$) (Fig. 3). During 15 min of recovery, arising the increment of the loss of erythrocytes potassium ion was correlated with the increment of
values of blood BE and HCO$_3^-$ concentration (Fig 4, 5). The recovery lasting fifteen minutes was sufficient to lower potassium ion concentration to the pre-exercise level.

**Blood acid-base balance.** Repeated supramaximal exercise caused changes in values of blood acid-base status. A significant decrease in blood pH was observed from 7.40±0.01 at rest to 7.21±0.04, 7.15±0.05, 7.13±0.04 at the end of the first, the second and the third exercise bouts, respectively. Also, significant changes in blood buffers were shown in the figures (Fig. 6, 7).

**Potassium ion concentration in urine:** Potassium ion concentration measured in urine collected during 1h and 24 h recovery after the exercise test was lower than in urine at rest. The concentration of urine potassium was 67.16±13.0 mmol/l and 31.23±12.53 mmol/l at the rest and 24 h after exercise, respectively.

**Discussion/ Conclusions**

The most important finding of this study was that a rapid increase in K$^+$ concentration in plasma during repeating supramaximal exercise did not lead to elevating of urine excretion of potassium ion. It was possible due to the participation of plasma and erythrocytes (RBC) (i.e. two
compartments of blood) in lowering large changes in plasma of potassium ion concentration released from contracting muscles.

Taking into consideration the obtained results, we can notice a protection against loss of potassium from the human body.

The quantity of K\(^+\) released during contractile action depends on the intensity of the exercise and the size of the muscle groups involved in contractions [8]. Our results show that the concentration of K\(^+\) is moderated by its uptake or release by RBCs. These observations were consistent with results of other authors [9, 10]. Transportation of K\(^+\) by human RBC is possible in both directions across the plasma membrane due to three mechanisms. The primary mechanism is active transportation by the Na\(^+\)-K\(^+\) pump [10, 11] and the second one – active transportation by the Na\(^+\)-K\(^+\)-2Cl\(^-\) cotransporter, while the third one – transportation across the K\(^+\)-ATP-sensitive channel [5, 6, 10, 11]. All mechanisms are used to move ions into RBCs.

Taking in consideration the fact that conditions after the exercise were favorable to activation of Na\(^+\)-K\(^+\) - ATP-ase due to increased concentration of catecholamines, insulin, or lactic acid [3, 12, 13], this enables us to suggest the mentioned above first mechanism as a regulating K\(^+\) transportation into RBCs.

In this study we showed not only the important role of RBC, but also renal system in K\(^+\) conservation. We observed that concentration of potassium ions in urine after supramaximal exercise was lower than at rest.

Our results are consistent with results by Medbo and Sejersted [14]. It is considered that such a rapid decrease in plasma K\(^+\) concentration is possible not only due to hiding it in erythrocytes but also to returning to muscles [5]. Described in this study turnover of K\(^+\) released from contractile muscle is of practical importance.

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