

Changes in Plasma Potassium During Graded Aerobic Exercise and Two Hours of Recovery

by

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Plasma potassium increases with exercise intensity. Subjects (n=8) were monitored for changes in plasma potassium while exercising at progressively increasing steady-state intensities and for two hours of recovery. Plasma potassium was significantly increased at 100% of $\dot{V}O_{2peak}$ compared to 20% and 40% ($p < 0.01$). Plasma potassium at 60 and 120 minutes of recovery from exercise was significantly higher than 6 minutes post exercise ($p < 0.015$). These results support the supposition that high-intensity exercise may lead to hyperkalemia, and also indicates that increases in $[K^+]$ occur up to two hours after the cessation of exercise, a newly reported phenomenon. Although, high levels of plasma potassium are known to cause cardiac abnormalities and related events, exercise induced changes in normal healthy adults are not currently believed to have clinical implications

Key words: plasma, potassium, aerobic exercise, recovery.

Introduction

Contraction of skeletal muscle during exercise causes a release of potassium ($[K^+]$) into the interstitial space, which, in turn, causes plasma $[K^+]$ to increase rapidly with the onset of exercise and clearance at exercise cessation (Hirche, Schumacher & Hagemann, 1980; Medbo & Sejersted, 1990). This increase in $[K^+]$ potentially puts some exercisers at risk for cardiac events due to hyperkalemia (Ettinger, 1974). Previous studies have monitored increases in $[K^+]$ commensurate with exercise intensity (Busse, 1991; Lidinger, 1994; Medbo & Sejersted, 1990). Medbo & Sejersted (1990) demonstrated no significant difference between arterial and venous $[K^+]$ in exhaustive treadmill running for 1 minute. A few studies have investigated plasma $[K^+]$ changes during short anaerobic exercise (Medbo & Sejersted, 1990), progressive aerobic exercise (Busse, 1991) and

long duration steady state aerobic exercise (Lidinger, 1994). All previous studies have recorded increases in $[K^+]$ commensurate with exercise intensity. All studies which recorded $[K^+]$ in recovery from exercise, have reported a rapid decrease in plasma $[K^+]$ below resting values, with a gradual return to resting level within 10-30 minutes post-exercise (Busse, 1991; Medbo & Sejersted, 1990; Vollestad, 1994). However, none of these studies have combined varying intensities and short-term steady state exercise. This may be important because of the dissimilar time required to attain an equilibrium of metabolic status and ionic constituents in the blood during exercise.

While a number of studies have investigated the dynamics of plasma $[K^+]$ during exercise, there is an apparent dearth of data regarding the accumulation of $[K^+]$ in the bloodstream greater than 30 minutes after exercise. The goal of this study is to verify the previously reported data regarding venous plasma $[K^+]$ changes during exercise and explore the altera-

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tions in plasma $[K^+]$ two hours after the cessation of a progressive exercise test.

Methods

Subjects

Eight male ($n=4$) and female ($n=4$) participants ages 21-38 were recruited for this study, all gave written informed consent in accordance with the Helsinki Declaration. The participants were 68.4 ± 8.1 kg in mass, 173 ± 10 cm in height and had a VO_{2peak} of 62.3 ± 7.4 mL/kg/min.

Testing Protocol

All exercise tests were performed at 7:30 am. Participants reported to the laboratory on the day of their exercise test after refraining from vigorous exercise for 24 hours. Each participant then had a 20 gauge catheter inserted into an antecubital vein. The participant's oxygen uptake was recorded (to ensure subject had not consumed food more than 2 hours prior to testing) and a 3 mL blood sample was obtained at the end of a 5 minute seated rest period. A saline flush was used to keep the catheter patent. Hematocrit (Hct) was determined and the remainder of the blood was centrifuged at $4^\circ C$, and the plasma extracted. All hemolyzed samples were discarded.

The exercise test consisted of 5 minute steady-state segments of ergometer cycling (Monark 828E cycle ergometer; Varberg, Sweden) at 80 rpm, starting at 40 W/min and progressively increasing by 40 Watt increments. At the end of each exercise stage, a blood draw was performed. The exercise segments were alternated with 5 minute recovery periods, designed to ensure that the $[K^+]$ measurements attained were not obscured by an additive effect of exercise from the previous stage. The exercise session was completed once the participant's pedal frequency dropped below 70 rpm for a 30 second period.

After exercise, the participant had blood collected at minutes 3, 6, 9, 30, 60 and 120 of recovery. The participants were allowed to consume water during this recovery period, but remained seated through-

out the time period.

Instrumentation

Oxygen uptake was measured using the Parvo-Medics TrueMax 2400 Metabolic System. Hematocrit was determined using the micro-capillary tube method. Whole blood lactate was measured upon completion of exercise to verify maximal exertion (>6.0 mmol/L; Accutrend Analyzer, Roche, Mannheim, DL). Plasma potassium concentrations $[K^+]$ were determined using the Vitros DT60 Chemistry system (Ortho-Clinical, Johnson & Johnson, New York, NY, USA). Hyperkalemia was classified as >5.5 mmol/L (Lewis, 2009).

Statistical Analysis

All $[K^+]$ values were first converted to change from baseline value (Δ scores) because of the wide range of resting values. Since all subjects did not perform the same number of stages during the exercise test, values based on intensity were interpolated. The chosen intensities were, 20, 40, 60, 80 and 100% of VO_{2peak} . VO_{2peak} was defined as the highest VO_2 level attained during the exercise test. Using Matlab, a cubic or 4th order polynomial spline was applied to the raw data (R^2 range: 0.58-0.98; R^2 mean: 0.82). The $[K^+]$ change from baseline ($\Delta[K^+]$) was then interpolated via the applied spline for each subject. The Hct data was interpolated using a linear regression method (R^2 range: 0.44-0.88; R^2 mean: 0.70).

The $\Delta[K^+]$ during exercise and recovery were analyzed via separate one-way repeated measures ANOVAs since exercise was scaled for intensity and recovery for time. The global alpha-level of 0.05 was Bonferroni adjusted for each comparison calculated in exercise and recovery.

Results

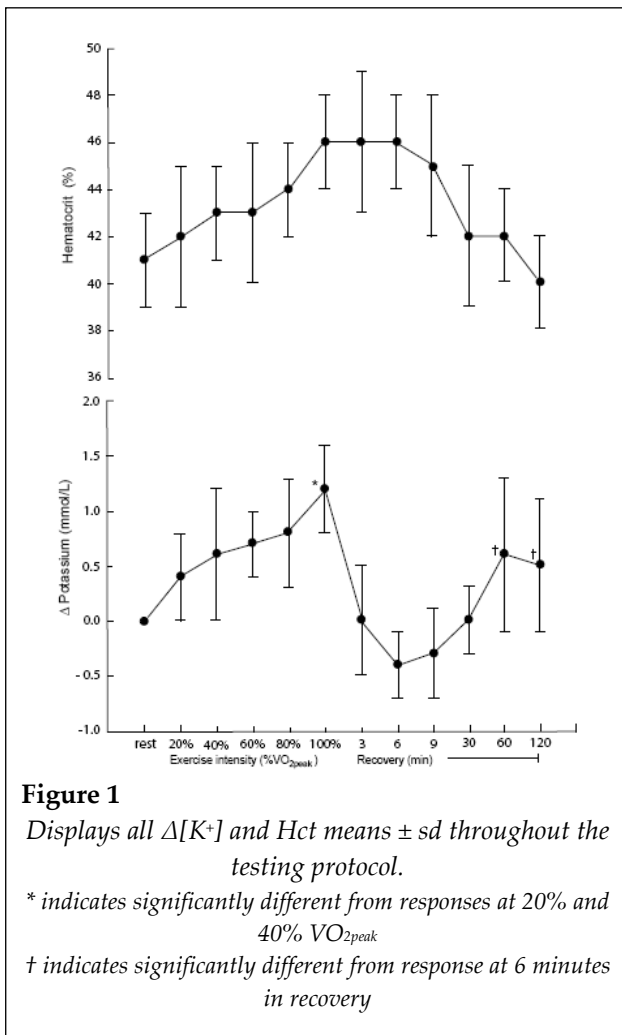
Figure 1 depicts the $\Delta[K^+]$ and Hct means throughout the testing protocol. Hct data is presented for visual analysis only and $\Delta[K^+]$ was not corrected for changes in Hct. Mean resting $[K^+]$ was 4.2 ± 0.2 mmol/L (Mean \pm SD). The $\Delta[K^+]$ at 100%

Table 1

Displays raw $[K^+]$ values (mmol/L) at rest and in recovery

Resting $[K^+]$	Minutes in Recovery Post-Exercise					
	3 Minutes	6 Minutes	9 Minutes	30 Minutes	60 Minutes	120 Minutes
4.2 \pm 0.2	4.2 \pm 0.4	3.9 \pm 0.3	4.0 \pm 0.4	4.2 \pm 0.4	4.8 \pm 0.6	4.8 \pm 0.5*

*indicates significant difference from resting value ($P=0.028$)



VO_{2peak} was significantly higher than the 20% and 40% VO_{2peak} responses ($p < 0.01$). Six of eight subjects were hyperkalemic at 100% VO_{2peak} (5.7 ± 0.4 mmol/L). By three-minutes in recovery, $\Delta[K^+]$ returned to resting values. At 60 and 120 minutes in recovery, $\Delta[K^+]$ had increased to 0.6 and 0.5 mmol/L above resting values, respectively. The recovery $\Delta[K^+]$ at 6 minutes was significantly lower than at 60 and 120 minutes ($p < 0.015$). At 60 minutes of recovery, one subject was again hyperkalemic. The unexpected ANOVA results in recovery warranted further analysis with paired-samples t-tests examining the raw $[K^+]$ values at baseline vs. 60 minutes and 120 minutes in recovery (Table 1). $[K^+]$ was significantly different from baseline at 120 minutes in recovery ($p = 0.028$) and nearly significant at 60 minutes in recovery ($p = 0.056$). The changes in potassium during recovery were not solely attributable to hemoconcentration since $[K^+]$ and Hct responses in recovery followed differing time courses (Figure 1).

Discussion

The findings of our study confirm previous findings that plasma $[K^+]$ increases with exercise (Busse, 1991; Lindinger, 1994; Medbo & Sejersted, 1990). However, our data was obtained in response to progressing stages of steady-state exercise which allows for equilibrium between metabolic and ionic events. Thus, our study adds to the mounting evidence that increases in plasma $[K^+]$ are related to the intensity of exercise.

Our findings suggest that during recovery there is a biphasic response, with an initial decline, possibly below resting baseline, followed by a delayed increase above resting baseline. This biphasic response is not related to hemodynamics, since Hct values declined continuously throughout the two-hour recovery; a different pattern than $\Delta[K^+]$. The initial rapid reduction in $[K^+]$ during the first 30-min of recovery has been previously reported (Busse, 1991; Medbo & Sejersted, 1990; Vollestad, 1994). However, the delayed increase one-to-two hours into recovery has not been previously noted. Medbo and Sejersted (1990) reported that after five, repeated bouts of anaerobic exercise there was a tendency for $[K^+]$ to increase slightly above baseline by 20 minutes of recovery. The greater total amount of exercise volume in our study may have exacerbated the recovery response. The same group (Medbo & Sejersted, 1994) also reported differences between aerobic and sprint-trained athletes in regards to resting and peak $[K^+]$ levels, hypothesizing this was an effect of Na^+/K^+ pump density. While our subjects were all aerobically trained, it is unclear how this training affected the extended recovery response observed. Differences in training or lack of training may be a potential area for future K^+ mechanics research.

It is unlikely that increases in $[K^+]$ 1-2 hours after cessation of exercise are a result of skeletal muscle mechanisms. Lindinger et al. (1992) extensively studied $[K^+]$ dynamics in regards to arterio-venous differences from the whole-blood, plasma and erythrocyte compartments. While the limited scope of the present study can not discount the compartmental factors presented by Lindinger et al., we believe the most plausible explanation for the unexpected rise in $[K^+]$ 1-2 hours after exercise is the acid-base homeostatic action in the kidneys. It has long been known that both acute respiratory and metabolic acidosis are associated with increases in $[H^+]$ and decreases in $[K^+]$ urinary excretion (Schultze, 1973). The exact mechanism by which the acid/base environment in the blood impacts K^+ transport at the distal tubule

has been debated; however, the association between increases in blood $[H^+]$, as often occurs during maximal aerobic exercise, and decreases in urinary output of $[K^+]$ are likely related to our unexpected $\Delta[K^+]$ rise hours after the cessation of exercise. As the kidneys attempt to equilibrate blood pH after exercise, it is possible that more H^+ ions are excreted into the urinary duct and more K^+ ions are reabsorbed back into the bloodstream. Barker et. al. (1957) and Elkinton et. al. (1955) demonstrated that plasma pH decreases as small as 0.08 result in significant decreases in urinary K^+ excretion. The subjects in our study had a mean blood lactate response of 11.1 ± 2.4 mmol/L at maximal exertion, making it very possible that pH had decreased by 0.08 or more (Kato, 2005). Further work is necessary to examine this proposed hypothesis.

The elevation of $[K^+]$ in the bloodstream hours after the cessation of exercise has important medical implications as hyperkalemia at 6.0 mmol/L can cause a notable shortening of the QT segment and increase in T-wave amplitude; hyperkalemia as great as 8.0 mmol/L can cause deadly cardiac arrhythmias (Ettinger, 1974). The exercise induced increases in blood potassium hours after exercise may be further exacerbated by medications known to increase $[K^+]$

in some patients. ACE inhibitors and some diuretics are known to increase serum $[K^+]$ in patients with normal and impaired renal function (Textor, 1982). Though patients undergoing therapy with an ACE inhibitor and performing strenuous aerobic exercise are likely rare, the pharmaceutical can be used to treat type-1 diabetes, migraines and high blood pressure, none of which are exclusion criteria for aerobic exercise. Recreational use of diuretics as an ergogenic aid in sports may also increase the occurrence of hyperkalemia. Non-steroidal anti-inflammatory drugs are commonly used in athletic populations, and an uncommon side effect is hyperkalemia (Schlondorff, 1993). The use of these medications in conjunction with vigorous exercise has the potential to cause both benign transient arrhythmias as well as more serious cardiac complications in susceptible populations. Although these implications exist, none of our subjects exhibited hyperkalemic complications during either exercise or recovery. As graded exercise tests have been used extensively in research with few reported complications, such a clinically evident hyperkalemic response may be limited to extended bouts of exercise and/or those using select medications.

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Acknowledgement

The authors would like to express their sincere thanks to all participants in this study, Kyle Leppert for assistance with sample processing and Kelly Tenan for technical artwork advising.

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