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Effect of Different Cycling Frequencies during Incremental Exercise on the Venous Plasma Potassium Concentration in Humans

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Summary

The effect of different muscle shortening velocity was studied during cycling at a pedalling rate of 60 and 120 rev.min^{-1} on the $[K^+]_v$ in 21 healthy young men (aged 22.5±2.2 years, body mass 72.7±6.4 kg, VO_2max 3.720±0.426 l · min^{-1}) performing an incremental exercise test until exhaustion. The power output increased by 30 W every 3 min, using an electrically controlled ergometer Ergoline 800S (see Zoladz *et al.* J. Physiol. 488: 211-217, 1995). The test was performed twice: once at a cycling frequency of 60 rev.min^{-1} (test A) and a few days later at frequency of 120 rev.min^{-1} (test B). At rest and at the end of each step (i.e. the last 15 s) antecubital venous blood samples for $[K^+]_v$ were taken. Gas exchange variables were measured continuously (breath-by-breath) using Oxycon Champion Jaeger. The pre-exercise $[K^+]_v$ in both tests was not significantly different amounting to 4.24±0.36 mmol.l^{-1} in test A, and 4.37±0.45 mmol.l^{-1} in test B. However, the $[K^+]_v$ during cycling at 120 rev.min^{-1} was significantly higher ($p<0.001$, ANOVA for repeated measurements) at each power output when compared to cycling at 60 rev.min^{-1}. The maximal power output reached 293±31 W in test A which was significantly higher ($p<0.001$) than in test B, which amounted to 223±40 W. The VO_2max values in both tests reached 3.720±0.426 l.min^{-1} *vs* 3.777±0.514 l.min^{-1}. These values were not significantly different. When the $[K^+]_v$ was measured during incremental cycling exercise, a linear increase in $[K^+]_v$ was observed in both tests. However, a significant ($p<0.05$) upward shift in the $[K^+]_v$ and a % VO_2max relationship was detected during cycling at 120 rev.min^{-1}. The $[K^+]_v$ measured at the VO_2max level in tests A and B amounted to 6.00±0.47 mmol.l^{-1} *vs* 6.04±0.41 mmol.l^{-1}, respectively. This difference was not significant. It can thus be concluded that a) generation of the same external mechanical power output during cycling at a pedaling rate of 120 rev.min^{-1} causes significantly higher $[K^+]_v$ changes than when cycling at 60 rev.min^{-1}, b) the increase of venous plasma potassium concentration during dynamic incremental exercise is linearly related to the metabolic cost of work expressed by the percentage of VO_2max (increase as reported previously by Vollestad *et al.* J. Physiol. Lond. 475: 359-368, 1994), c) there is a tendency towards upward shift in the $[K^+]_v$ and % VO_2max relation during cycling at 120 rev.min^{-1} when compared to cycling at 60 rev.min^{-1}.

Key words

Cycling • Exercise • Muscle fatigue • Oxygen uptake • Plasma potassium

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Introduction

The loss of potassium from exercising muscles attracted many researchers already back in the 1920s. Among the first who showed that human muscle loses potassium during exercise were Ewig and Wiener (1928). Subsequently Fenn and Cobb (1936) and again Fenn (1938) confirmed this finding using animal muscle preparation.

The interest in the study of muscle potassium shifts has been stimulated by the idea that its loss from the working muscle may play a key role in the origin of muscle fatigue (Sjøgaard 1990, 1991, 1996). The precise mechanism, however, responsible for the loss of potassium from the muscle, as well as its significance in the phenomenon of muscle fatigue, are still not completely understood (for review see Sejersted and Sjøgaard 2000).

It was reported that the rate of release of K^+ from the working muscle in men is linearly related to the power output (see Vollestad *et al.* 1994). During short lasting high intensity exercise, values of potassium concentration $[K^+]$ as high as 9 mmol.l^{-1} were reported (Medbo and Sejersted 1990, 1994).

It was already suggested many years ago by Fenn (1938) that K^+ loss during muscle activity is proportional to the intensity and frequency of muscle contractions. In a number of studies, venous plasma potassium concentrations during cycling exercise were found to correlate closely with the power output or oxygen uptake (Tibes *et al.* 1976, Yasuda *et al.* 1992, Vollestad *et al.* 1994). It is well known that the generation of power output is a result of muscle force and muscle shortening velocity (for review see Sargeant 1999). Therefore, it seemed of interest to ascertain what is the effect of different muscle shortening velocity on the exercise-induced increase of $[K^+]_v$. Surprisingly, no attention has been paid in the various studies performed so far to the influence of the cycling frequency under controlled power output, on the $[K^+]_v$ in humans. For this reason, we studied the effect of cycling at a pedaling rate of 60 and 120 rev.min^{-1} on $[K^+]_v$ in human subjects during an incremental exercise protocols.

Methods

In this study, 21 healthy young men aged 22.5±2.2 years, with body mass of 72.7±6.4 kg, VO_2max 3.720±0.426 l.min^{-1}, performed an incremental exercise test until exhaustion. The power output increased by 30 W every 3 min, using an electrically controlled ergometer Ergoline 800S (see Zoladz *et al.* 1995). The test was performed twice: once at a cycling frequency of 60 rev.min^{-1} (test A) and a few days later at a frequency of 120 rev.min^{-1} (test B).

At rest and at the end of each step (i.e. the last 15 s) antecubital venous blood samples were withdrawn for $[K^+]_v$ estimations. Blood samples were taken using Abbot Int-Catheter Ireland (18 G/1.2 x 45 mm) inserted into the antecubital vein about 30 min prior to the onset of the exercise. The catheter was connected with an extension set using "T" Adapter SL Abbot Ireland (a tube 10 cm long). Immediately before the blood samples were withdrawn for appropriate analysis, blood samples (1 ml) were taken in order to eliminate the blood from the catheter and the T - set. No agents or liquids that could alter the potassium changes were used.

Plasma venous potassium concentration $[K^+]_v$ was determined using Chiron Diagnostics 644 $Na^+/K^+/Cl^-$ analyzer, U.K. Plasma lactate concentration ($[La]_{pl}$) was measured using an automatic analyzer (Ektachem XR 700, Kodak, USA). Blood hydrogen ion concentration ($[H^+]_b$) was assessed with a Ciba-Corning 248 analyzer (England). The hematocrit value (Ht) was determined using an automatic hematological analyzer, Baker 9000 USA. Gas exchange variables were measured continuously (breath-by-breath) using Oxycon Champion Jaeger.

The study was approved by the local ethical committee and all subjects gave their informed, written consent.

Statistical analysis

Values represent means ± S.D. Statistical significance was tested by the paired t-test, as well as by the ANOVA for repeated measurements. The relationship between $\Delta[K^+]_v$) and % VO_2 was tested using F test model ANCOVA.

Results

The pre-exercise $[K^+]_v$ values in test A and B did not differ significantly, being 4.24±0.36 and 4.37±0.45 mmol.l^{-1}, respectively. However, during cycling at 120 rev.min^{-1} in the range of power output from 30-180 W (in which a complete set of data was obtained), the $[K^+]_v$ was significantly higher ($p<0.001$, ANOVA for repeated measurements) as compared to cycling at 60

rev.min^{-1} (Fig. 1). We also determined the exercise induced changes in hematocrit values (Ht). The pre-exercise Ht values determined before cycling at 60 and 120 rev.min^{-1} were very similar (46.8±2.2 and 46.6±2.8, respectively). At the final stage of both tests, a significant increase in Ht of 6 % was observed ($p<0.001$). Nevertheless, Ht determined at the final stage of cycling at 60 and 120 rev.min^{-1} were almost identical, being 49.7±2.2 and 49.9±2.3, respectively.

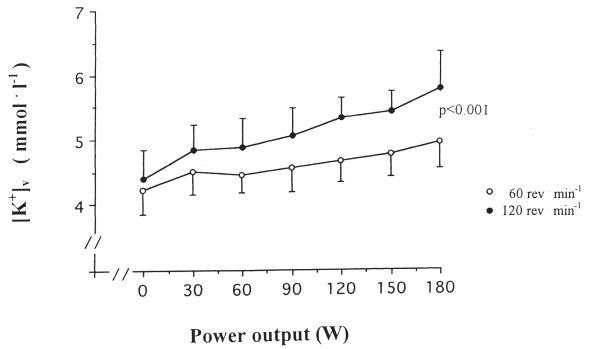

Fig. 1. *Venous plasma potassium concentration $[K^+]_v$ during incremental cycling exercise performed at 60 and at 120 rev.min^{-1} in relation to power output.*

It should be noticed that the maximal power output of 293±31 W attained during cycling at 60 rev.min^{-1} was significantly higher ($p<0.001$) than the maximal power output (223±40 W) reached at 120 rev.min^{-1}. The VO_2max values amounted to 3720±0.426 l.min^{-1} *vs* 3777±0.514 l.min^{-1}, respectively, in test A and B, which did not differ significantly.

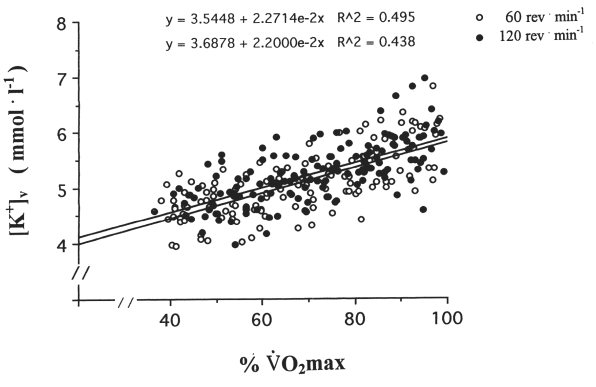

Fig. 2. *Venous plasma potassium concentration $[K^+]_v$ during incremental cycling exercise performed at 60 and at 120 rev. min^{-1} in relation to the percentage of maximal oxygen uptake (% VO_2max).*

When expressing the $[K^+]_v$ measured during incremental cycling exercise in relation to power output (Fig 1) and in relation to the percentage of VO_2max (Fig. 2), only minor differences between $[K^+]_v$ in both tests were observed. However, a noticeable and significant ($p<0.05$) upward shift in the $[K^+]_v$ / %VO_2max relationship was detected during cycling at 120 rev.min^{-1} (test F in the ANCOVA model) (Fig. 2). Nevertheless, the $[K^+]_v$ measured at VO_2max in both tests was 6.00±0.47 mmol.l^{-1} *vs.* 6.04±0.41 mmol.l^{-1} at 60 vs. 120 rev.min^{-1} respectively, which was not significantly different.

We also examined the increase of plasma potassium ($\Delta[K^+]_v$) in relation to the net increase in oxygen uptake (ΔVO_2) measured at the end of exhaustion during cycling at 60 and 120 rev.min^{-1} (Figs 3 A,B). Figure 3 C shows the relationship between ($\Delta[K^+]_v$) and (ΔVO_2) for all subjects and for both frequencies of cycling. In all the panels, a positive significant correlation $p<0.05$) between ($\Delta[K^+]_v$) and (ΔVO_2) was found.

Discussion

Plasma potassium concentrations in humans during various modes of exercise has been studied in several experiments (for review see Sejersted and Sjøgaard 2000). It was found in a number of studies that the venous plasma potassium concentration during cycling exercise closely correlates with the power output or oxygen uptake (Tibes *et al.* 1976, Yasuda *et al.* 1992, Vollestad *et al.* 1994). However, since the muscle mechanical power output is the result of muscle force and of muscle shortening velocity, a certain magnitude of mechanical power output may be obtained by a different contribution of muscle force and muscle shortening velocity (for review see Sargeant 1999). It has been shown that muscle shortening velocities have a strong effect on the power generating capabilities as well as on the energy turnover in working muscles (see e.g. Woledge *et al.* 1985, Sargeant 1999, Bottinelli *et al.* 1999). However, as far as we know, no data have been reported to show the effect of slow and fast muscle shortening velocities on $[K^+]_v$ during incremental cycling exercise in man. For this reason we examined the effect of cycling at 60 and 120 rev.min^{-1} during incremental cycling exercise on the $[K^+]_v$ in human subjects.

The main finding of our study is that the generation of the same external mechanical power output during cycling exercise at 120 rev.min^{-1} was accompanied by a significantly higher plasma potassium

concentration ($p<0.001$) than at 60 rev.min^{-1} (see Fig. 1). This may suggest that higher muscle shortening velocity applied at a given power output causes greater $[K^+]_v$.

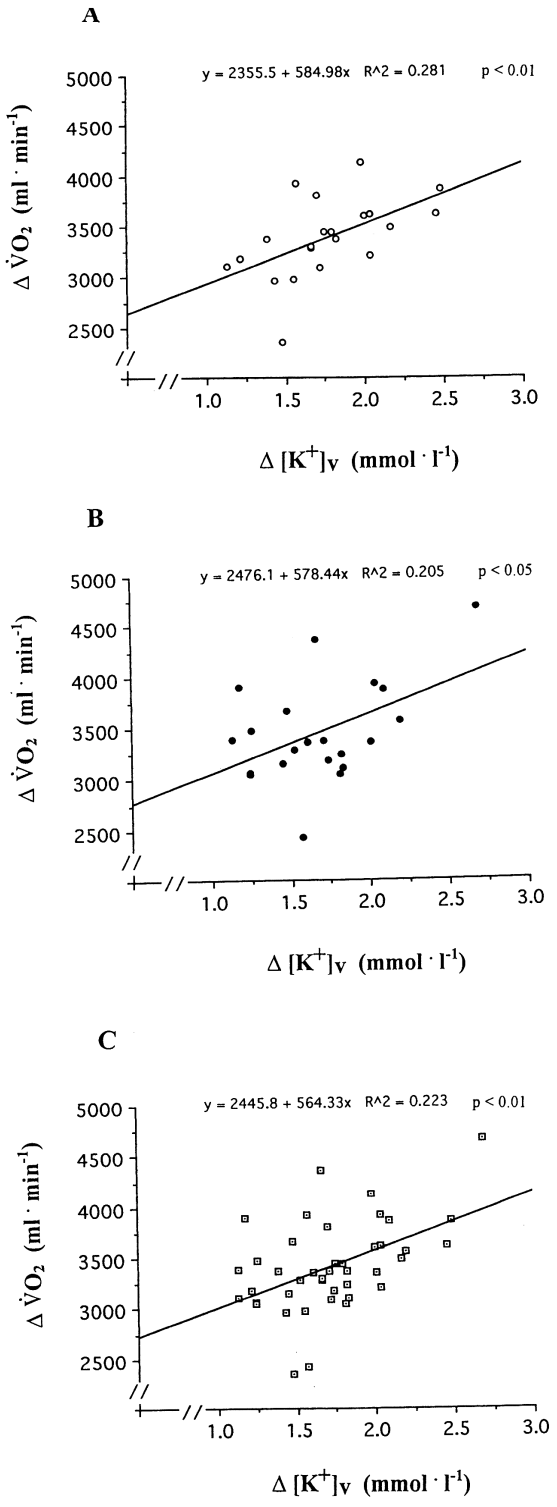

Fig. 3. *The net increase of plasma potassium ($\Delta[K^+]_v$) in relation to the net increase in oxygen uptake (ΔVO_2) measured at the end of exhaustion during cycling at 60 rev.min^{-1} (panel A), at 120 rev.min^{-1} (panel B) and combined data from both tests (panel C). The $\Delta[K^+]_v$ and the ΔVO_2 was calculated as the difference between the maximal values reached at exhaustion minus the pre-exercise values.*

However, one should be aware of the fact that, as was reported in a previous study (for review see e.g. Zoladz *et al.* 2000), the metabolic cost of work in this range of power output during cycling at 120 rev.min^{-1} is significantly higher than during cycling at 60 rev.min^{-1}. Hence, we have expressed the $[K^+]_v$ occurring during cycling at both frequencies of movement in relation to the metabolic cost of work (% of VO_2max). The increase in venous plasma potassium concentration during dynamic incremental exercise depends mainly upon the metabolic cost of work expressed by percentage of VO_2max, as is shown in Figure 2. Nevertheless, by analyzing the $[K^+]_v$ / %VO_2max relationship during cycling at 60 and 120 rev.min^{-1}, based on the F-test in the ANCOVA model, we have also found a significant tendency ($p<0.05$) towards an upward shift in the $[K^+]_v$ and the % VO_2max relation during cycling at 120 rev.min^{-1} when compared to cycling at 60 rev.min^{-1}.

Additionally, we have studied the relationship between the net increase of plasma potassium ($\Delta[K^+]_v$) in relation to the net increase in oxygen uptake (ΔVO_2) measured at exhaustive cycling exercise performed at 60 and 120 rev.min^{-1}. As presented in Figures 3A, 3B and 3C, significant positive correlation ($p<0.05$) between $\Delta[K^+]_v$ and ΔVO_2 was found. Our results are in agreement with the recent study by Tanabe *et al.* (1999) who have shown a similar relation in patients with chronic heart failure of various severity, in whom the peak VO_2 was below 25 ml.kg^{-1}.min^{-1}. These data taken together show that the potassium loss during exercise is strongly related to the metabolic cost of work and that the velocity of movements applied in our study have a relatively small effect on this relationship.

Despite the fact that the maximal external mechanical power output reached during cycling at the pedaling rate of 120 rev.min^{-1} was significantly lower than during cycling at 60 rev.min^{-1}, the maximal values of plasma potassium concentrations measured at exhaustion in both exercise protocols were not significantly different (6.04±0.41 and 6.00±0.47 mmol.l^{-1}, respectively). Moreover, the $\Delta[K^+]_v$ values in both tests did not differ being during cycling at 60 and 120 rev.min^{-1} 1.76±0.36 and 1.68±0.39 mmol.l^{-1}, respectively.

The obtained results of $[K^+]_v$ at exhaustion are close to the data found in the literature for normal young healthy subjects (see Sejersted and Sjøgaard 2000). Occasionally, in individual cases in our study the $[K^+]_v$

exceeded 7.00 mmol.l^{-1}. These values of $[K^+]_v$ are lower than the maximal values reported in the literature (as high as 9 mmol.l^{-1}) measured in a small group of subjects after very high intensity short-term exercise (see Medbo and Sejersted 1985, 1990). Interestingly, in the study of Tanabe *et al.* (1999), the plasma potassium concentration ($[K^+]_a$ at the end of exercise measured in most severe chronic heart failure patients with the lowest VO_2max (13.8±1.7 ml.kg^{-1}.min^{-1}) was below 5 mmol.l^{-1}.

It is postulated that the loss of muscle potassium is directly involved in the mechanism of muscle fatigue (for review see Sejersted and Sjøgaard 2000). In the present study, a faster rate of plasma potassium accumulation occurring during cycling at 120 rev.min^{-1} was accompanied by early fatigue. The difference between the final power output reached during cycling at 120 rev.min^{-1} (223±40 W) and at 60 rev.min^{-1} (293±31 W) was significant ($p<0.001$), although the VO_2max values reached in both tests A and B were not significantly different (3.720±0.426 l.min^{-1} *vs* 3.777±0.514 l.min^{-1}, respectively. This could suggest that the early loss of potassium from the working muscle may indeed be directly involved in the early fatigue that occurs during cycling at 120 rev.min^{-1}.

In the present study, we also followed other blood parameters including lactate and $[H^+]$. At the stage of exhaustion during cycling at 60 and 120 rev·min^{-1} plasma lactate concentration was not significantly different in both tests (10.10±2.32 mmol.l^{-1} vs 10.91±2.16 mmol.l^{-1} respectively). Similarly $[H^+]$ measured at exhaustion during cycling at 120 rev.min^{-1} amounting to 58.3±6.40 nmol·l^{-1} was not significantly different when compared to its level measured at exhaustion during cycling at 60 rev.min^{-1} (which amounted to 60.6±8.1 nmol·l^{-1}). These results illustrate that early fatigue during cycling at 120 rev.min^{-1}, accompanied by a faster rate of plasma potassium accumulation was also accompanied by early acidosis. We therefore postulate that the faster rate of plasma potassium accumulation may be an important factor, but not the only cause of early fatigue that occurred during cycling at 120 rev·min^{-1}. We think that a number of other factors including increase of internal work, greater recruitment of fast fatigue-sensitive motor units already at low mechanical power output, early acidosis and early glycogen depletion in the fast fatigue-sensitive motor units (for review see Zoladz *et al.* 2000) could also be responsible for the early fatigue that had occurred during cycling at the pedalling rate of 120 rev.min^{-1}.

The main findings of our study may be summarized as follows: a) generation of the same external mechanical power output during cycling at a pedaling rate of 120 rev·min^{-1} causes significantly higher $[K^+]_v$ than when cycling at 60 rev·min^{-1}, b) the increase of venous plasma potassium concentration during dynamic incremental exercise is linearly related to the metabolic cost of work expressed by the percentage of VO_2max, as reported previously by Vollestad *et al.* (1994); c) there is an upward shift in the % VO_2max and $[K^+]_v$ relation during cycling at 120 rev·min^{-1} when compared to cycling at 60 rev·min^{-1}.

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