Plasma $K^+$ dynamics and implications during and following intense rowing exercise

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POTASSIUM ($K^+$) homeostasis is essential for myocardial, neural, and skeletal muscle function. Plasma $K^+$ concentration ([K$^+$]) depends on the highly regulated interplay of renal and nonrenal mechanisms, via neurohumoral, metabolic, and ionic regulation, spanning a time course of seconds to months (55). Nonetheless, the circulating [K$^+$] is highly dynamic, being acutely perturbed by the normal daily activities of diet and, more particularly, exercise. Numerous studies have revealed that plasma [K$^+$] rises markedly during exercise and is primarily dependent upon exercise intensity, regardless of the exercise mode, or whether [K$^+$] is measured in arterial or venous plasma (22, 25, 32, 54). Importantly, plasma [K$^+$] also appears to increase in proportion to the contracting muscle mass, as evidenced from cross-sectional comparisons utilizing different exercise modalities (14). For example, arterial plasma [K$^+$] ([K$^+$]a) rose only slightly to 4.19 mM during exhaustive contractions of the finger flexor muscles (50), to 4.5 and 5.4 mM during submaximal arm cranking and leg cycling, respectively (53), to 6–7 mM during intense sprint cycling (26, 28) but to as high as 9 mM during exhaustive 60-s treadmill running (32). The higher [K$^+$]a with a large contracting muscle mass is most likely due to a greater $K^+$ release; however, the mass of noncontracting muscle is also important, since inactive muscle extracts $K^+$ and thereby lowers [K$^+$]a during exercise (14, 26). Thus a greater hyperkalemia during intense exercise with a large muscle mass may be a consequence of both a greater $K^+$ release from contracting muscle and a reduced $K^+$ clearance due to a smaller mass of inactive muscle.

If sprinting-type exercise could be sustained, even further increases in [K$^+$] might have occurred in those studies where very high [K$^+$] was obtained; however, exercise at these high intensities could only be sustained for short periods of time (32). Alternately, it is possible that the larger contracting muscle mass may induce a greater overall activation of Na$^+$,$K^+$-ATPase molecules simply due to excitation effects in more muscles (9) and may also lead to a reduced relative exercise intensity for each muscle group. Together these would result in a lesser rate of $K^+$ release from the contracting muscles and a lower circulating [K$^+$].

Rowing is an extremely demanding sport, utilizing a large muscle mass, contracting at high intensity and sustained for 5–7 min; during all-out trials, world-class oarsmen may achieve an oxygen consumption (V$\text{O}_2$) of 6.0 l/min, a venous blood lactate concentration of 26 mM, and a decline in pH to 6.85 (35). Large increases in [K$^+$]a with rowing might then also be anticipated, but in elite rowers, [K$^+$]a was only 6 mM at the completion of a 2,000-m trial (34). However, as a single time point measure in that study, the time course of changes in [K$^+$]a was not described, and it is unclear whether these values might have declined from higher levels earlier in exercise, or alternately, even reflect a late increase in [K$^+$]a due to the typical final surge in power output toward the end of a rowing trial. A peak venous plasma [K$^+$] of 6.1 mM was reported during incremental rowing in six renal transplant recipients (33), although patients with chronic kidney disease already exhibit poor exercise capacity and excessive exercise hyperkalemia (45). Thus the [K$^+$] dynamics during rowing have not yet been defined and are important to resolve. The major aim of this research was therefore to investigate the effects of exhaustive rowing exercise, which combines a large contract-
ing muscle mass and high exercise intensity, on arterial K⁺ regulation in humans. We specifically investigated the magnitude and time course changes of arterial [K⁺] during intense rowing. Since muscular fatigue is a feature of such intense exercise (46) and intramuscular K⁺ disturbances have been linked to fatigue (27), we also investigated the relationships during exercise between hyperkalemia, power output, and the electromyographic (EMG) activity of active muscles.

Following intense exercise, [K⁺]₀ typically falls below resting levels, proportional to exercise intensity (32, 54), and this is probably due to Na⁺,K⁺-ATPase-mediated K⁺ reuptake by previously active and inactive muscles (26, 31, 32). Studies in isolated rat muscles have shown Na⁺,K⁺-ATPase activation is sustained following contractions (37, 38). Most human exercise studies do not report the time course of postexercise [K⁺]₀ beyond 10–20 min, but lowered [K⁺]₀ can persist for up to 90 min into recovery (25). A postexercise hypokalemia may be of broader interest given that hypokalemia has been linked to cardiac arrhythmias and sudden death following exercise in susceptible individuals (20). Given the high intensity and large active muscle mass during maximal rowing, a large and sustained decline in [K⁺]₀ might also be anticipated, but this is currently unknown. Hence the second major aim of the study was to investigate the magnitude and time course of the decline in [K⁺]₀ following exhaustive rowing. It was hypothesized that 1) rowing would be characterized by a marked [K⁺]₀ that is sustained during exercise, associated with a decline in power output and changes in the temporal and spectral characteristics of the EMG signals; and 2) rowing would also be followed by a pronounced and prolonged reduction in [K⁺]₀ postexercise.

METHODS

Subjects

Ten healthy subjects, comprising 7 males and 3 females, gave written informed consent prior to participating in the study (age, 24.7 ± 5.3 yr; height, 1.80 ± 0.09 m; body mass, 81.4 ± 12.3 kg, mean ± SD). All were recreationally active; four were actively rowing at club level, three had previous rowing experience competing at club level, but were no longer engaged in the sport, and three had gymnasium-based rowing experience and were recreationally engaged in rowing exercise. Individuals that were recruited were competitive rather than elite rowers, since elite rowers would likely exhibit upregulated muscle Na⁺,K⁺-ATPase content, as this is a typical training adaptation evident across all training types (11, 12) and which might then attenuate the exercise hyperkalemia (29). The study was approved by the Victoria University Human Research Ethics Committee.

Rowing Ergometer Test

Subjects were initially familiarized on a rowing ergometer (Concept 2 Model E, PM4 monitor) by rowing for 1,000 m at ~50% of maximal effort. Subjects avoided vigorous exercise, caffeine, and alcohol and maintained their normal dietary habits for 24 h preceding the experimental trial. Participants completed a 2,000-m “all-out” maximal trial on the rowing ergometer. This distance was selected as it is the standard in race competition. No warm-up was undertaken, as this would likely activate skeletal muscle Na⁺,K⁺-ATPase and attenuate the rise in plasma [K⁺] (49). Subjects were instructed to complete the 2,000 m in the shortest time possible and were given verbal encouragement to give a maximal effort throughout the trial, but were free to select their own pace. Average and instantaneous power output, distance covered, and stroke rate data were recorded every 30 s during the trial.

Cardiorespiratory Measures

Heart rate was measured continuously during and after the trial by 12-lead ECG (Model X-Scribe Stress Test System, Mortara Instrument, Milwaukie, WI). Pulmonary respiratory data were also obtained continuously throughout exercise. Subjects breathed into a Hans-Rudolph two-way nonrebreathing valve, with the expired gas passing through low-resistance plastic tubing into a 4-liter mixing chamber. Mixed expired oxygen (O₂) and carbon dioxide (CO₂) fractions were continuously analyzed by rapidly responding O₂ and CO₂ analyzers (Ametek S-3A/II and Ametek CD-3A, Pittsburgh, PA), and expired volumes were derived from a flow transducer (KL Engineering K520) as previously described (23). V̇O₂, CO₂ output, (V̇CO₂), and respiratory exchange ratio (RER) were calculated every 1 min on a PC (Turbo fit). The ventilometer and gas analyzers were calibrated before each test with a standard 3-liter syringe and precision references gases, respectively.

Blood Sampling and Analyses

A cannula (Arrow Quick Flash, 20 G) was inserted into the radial artery, attached to an extension tubing and stopcock set (ITL Arterial Kit- LS084), which was affixed to the forearm and upper arm, and then attached to a pressurized bag of sterile isotonic saline, placed on a mobile stand adjacent to the rowing ergometer. A separate extension set was attached to the stopcock to enable rapid, repeated blood sampling without hindrance while subjects rowed at high intensity and stroke frequency. After cannulation, subjects rested supine for 20 min and the initial resting sample was then obtained, which was at 10 min prior to exercise commencement. Subjects were then seated on the adjacent rowing ergometer where a seated preexercise sample was taken ~2 min before exercise commencement. Blood samples (~3.6 ml) were taken on the rowing ergometer at 30-s intervals throughout exercise, immediately prior to the completion of 2,000 m rowing, and at 1 and 2 min postexercise; the participant was then moved to an adjacent couch for supine rest, and further samples were then taken at 5, 10, 20, and 30 min recovery. Two samples were taken at each time point. The first sample (~0.6 ml) was drawn into a heparinized blood gas syringe (Siemens, New York) and immediately analyzed for plasma electrolyte (K⁺, Na⁺, and Cl⁻) concentrations and acid-base status using an automated blood gas analyzer (Rapid Point 405 Siemens Medical Solutions and Diagnostics, Tarrytown, NY). An additional sample (~3 ml) was collected in a plain syringe and immediately distributed into two separate Eppendorf tubes, used for duplicate analyses of hematocrit (Hct) and hemoglobin concentration ([Hb]) (Sysmex K800 TOA Medical Electronics, Kobe, Japan), and blood glucose and lactate ([Lac⁻]) concentrations (YSI 2300 Stat Plus Analyser, YSI, Yellow Springs, OH). All auto-analyzers were calibrated before and during measurements using standard manufacturer calibration procedures. Duplicates of the resting plasma [K⁺] were 3.86 ± 0.12 vs. 3.86 ± 0.11 mM (CV 2.9%). Comparison of measures using blood gas syringe vs. plasma immediately separated and frozen for later analysis revealed no difference between measures (0.05 ± 0.23 mM, NS, n = 215, r² = 0.96). The change in plasma volume (ΔPVs) from rest in the supine position and from preexercise levels in the seated position was calculated during and after exercise from [Hb] and Hct as earlier described (16) ([Hb] and Hct data not shown). Approximately 80–100 ml of blood (around 2–2.5% blood volume) was sampled during the trial. Renal K⁺ excretion was not measured here but is unlikely to substantially contribute to regulation of the circulating [K⁺] during intense rowing exercise, due to both the decline in urinary K⁺ excretion with intense exercise and the very low rate of this compared with skeletal muscle K⁺ release (45).
Electromyography (EMG)

The surface electromyographic (sEMG) signals of seven muscles were recorded continuously during exercise (TeleMyo DTS, Noraxon) for analyses of muscle activation. The sEMG signals were recorded from the left side of the body for five lower limb muscles [gastrocnemius medialis (GAS), vastus lateralis (VL), biceps femoris long head (BF), and rectus femoris (RF)]; two upper limb muscles [brachial radialis (BR) and biceps brachii (BB)]; and one trunk muscle [lumbar erector spinae (LES)]. Previous studies have shown that these muscles are all highly activated during rowing (41, 42). Before placing the sEMG electrodes, the local area was shaved, and the skin lightly abraded and cleansed with an alcohol swab to reduce skin impedance. Electrodes were then affixed to the skin longitudinally over the area of greatest muscle bulk and aligned parallel to the underlying muscle fiber direction, following SENIAM’s recommendations (Surface EMG for Non-Invasive Assessment of Muscles) (18). Prior to the start of the experiment, the subjects were asked to alternate contraction and relaxation of the different muscles to enable the experimenter to visually confirm a good sEMG signal-to-noise ratio for each muscle. The sEMG signals were sampled at 1,500 Hz and then processed using Noraxon software (MyoResearch XP version 1.07.41) according to the following procedure: 1) all raw sEMG signals were band-pass filtered (10–500 Hz); 2) the filtered EMG signals were full-wave rectified and smoothed over a 50-ms time window; 3) absolute integrated (iEMG) values (in mV) of each muscle were calculated over 10% intervals of the rowing total time (i.e., deciles of the exercise duration); 4) the amplitude of all iEMG values (in mV) was expressed as a percentage of the maximal amplitude of the iEMG value (in mV) calculated for the corresponding muscle during the effort (44); and 5) the normalized iEMG values (%iEMGmax) of the seven muscles were then summed to obtain a sum-iEMG value that represented the overall activity of the muscles activated to produce this whole body effort (2, 5, 41). In addition to the temporal analysis of the sEMG signals, a spectral analysis was completed, according to the following procedure: 1) all raw sEMG signals were band-pass filtered (10–500 Hz); 2) the median frequencies of the sEMG signals of each muscle were calculated over decile intervals of the exercise duration; and 3) the average value of the median frequencies measured for the seven muscles was calculated.

Statistical Analyses

All data were analyzed using a one-way ANOVA with repeated measures for time. Statistical comparisons were made against both the supine rest sample and the seated preexercise sample to take into account the two postures used at different time points during the trial, comprising seated (exercise, plus 1 and 2 min recovery) and supine (rest plus 5–30 min recovery). Data are presented as means ± SD, and statistical significance was accepted at $P < 0.05$. Correlations were determined using Pearson’s product-moment correlation. Average power output, iEMG, and median frequency values were calculated every 30 s during rowing to coincide with plasma $\left[K^+\right]_e$ measures; the $\left[K^+\right]_e$ and EMG data were then analyzed over decile time intervals (i.e., first decile represents 0–10% total rowing time, tenth decile represents 90–100% time). Effect sizes (ES) and 95% confidence intervals were assessed using Cohen’s $d$, to determine meaningfulness of results; ES thresholds were defined as trivial ($<0.2$), small (0.6), moderate (1.2), large (2.0), very large (4.0), and extremely large ($>4.0$) (1).

RESULTS

Exercise Time, Power Output, and Cardiorespiratory Measures

The time to complete the 2,000-m rowing trial was 7.26 ± 0.59 min (range 6.35–8.28 min). The exercise was intense, evidenced by peak cardiorespiratory values, with peak minute ventilation ($\dot{V}E_{peak}$) of 121.2 ± 26.7 l/min, peak heart rate (HR$_{peak}$) of 176 ± 10 beats/min, and peak VO$_2$ ($\dot{V}O_2$$_{peak}$) of 4.32 ± 0.78 l/min; the time course of VE, HR, and VO$_2$ during rowing are shown in Table 1. The instantaneous power output at 30 s was 326 ± 81 W; on an absolute time scale power tended to decline slightly until 4 min to 242 ± 53 W and was then unchanged at 260 ± 70 W at end exercise with a small effect size suggesting a possible rise (ES, $-0.49$ ± 0.30, Table 1). However, since exercise time varied, power output was also calculated on a decile time scale; this analysis revealed power output (%maximum) decreased by 16.5% from the first to the fourth decile ($P < 0.05$) and remained 19.9% depressed at the ninth decile ($P < 0.05$). Power output increased slightly between the ninth and tenth deciles [3.4% NS, ES = 0.27 (−0.62 to 1.14)], but although not significant, remained 16.2% less than the first decile [ES = −1.58 (−2.51 to −0.52)].

Arterial Plasma Electrolytes

Fluid shifts. The PV$_a$ declined slightly (4%) from supine rest (−10 min) to seated preexercise (0 min, $P < 0.05$) and continued to decline during exercise, with $\Delta$PV$_a$ reaching a nadir of $-9.7 ± 2.3$% at 6.5 min exercise and remaining low at end exercise ($P < 0.001$), indicating hemoconcentration. Postexercise, $\Delta$PV$_a$ recovered progressively but remained below rest (−10 min) at 10 min postexercise ($P < 0.001$) and below preexercise (0 min) at 5 min postexercise ($P < 0.001$, Fig. 1), indicating that fluid balance was not restored within this time period.

Potassium. Plasma $\left[K^+\right]_e$ increased slightly from supine rest to seated preexercise (0 min, $P < 0.05$), increased progressively until 90 s during exercise, reaching 6.13 ± 0.46 mM (range 5.66–7.01 mM, $P < 0.001$), and remained unchanged from this level thereafter during exercise ($P < 0.001$, Fig. 1). Following exercise, plasma $\left[K^+\right]_e$ decreased rapidly, falling to below rest ($P < 0.001$) and preexercise ($P < 0.05$) by 1 min, reaching a nadir at 5 min postexercise of 3.33 ± 0.22 mM ($P < 0.001$, range 3.03–3.76 mM), remaining below resting levels at 20 min postexercise ($P < 0.05$), and tending to be less than rest at 30 min as suggested by a small effect size [ES = −0.62 ± 0.30].

Table 1. Respiratory, cardiovascular, and power output during a 2,000-m rowing “all-out” ergometer trial

<table>
<thead>
<tr>
<th>Time (min):</th>
<th>1.0</th>
<th>2.0</th>
<th>3.0</th>
<th>4.0</th>
<th>5.0</th>
<th>6.0</th>
<th>End Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Power, W</td>
<td>314 ± 76</td>
<td>284 ± 62</td>
<td>258 ± 60</td>
<td>242 ± 53</td>
<td>246 ± 59</td>
<td>253 ± 60</td>
<td>260 ± 70</td>
</tr>
<tr>
<td>VO$_2$, l/min</td>
<td>3.85 ± 0.76</td>
<td>4.22 ± 0.80</td>
<td>4.31 ± 0.83</td>
<td>4.22 ± 0.82</td>
<td>4.29 ± 0.84</td>
<td>4.32 ± 0.78</td>
<td>4.20 ± 0.82</td>
</tr>
<tr>
<td>VE, l/min</td>
<td>79.1 ± 17.2</td>
<td>111.1 ± 30.0</td>
<td>117.4 ± 33.7</td>
<td>117.2 ± 31.9</td>
<td>119.0 ± 24.9</td>
<td>121.2 ± 26.7</td>
<td>117.0 ± 24.9</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>158 ± 11</td>
<td>162 ± 9</td>
<td>166 ± 9</td>
<td>169 ± 9</td>
<td>172 ± 10</td>
<td>175 ± 9</td>
<td>176 ± 10</td>
</tr>
</tbody>
</table>

Values are means ± SD; $n = 10$ for power and heart rate; $n = 9$ for minute ventilation (VE) and oxygen consumption (VO$_2$). Exercise time to complete 2,000 m rowing was 7.26 ± 0.59 min.
plasma $[K^+]_a$ was below preexercise (0 min) values at 30 min postexercise ($P < 0.05$, Fig. 1).

To account for hemoconcentration effects, plasma $[K^+]_a$ was also corrected for the PV$_a$. The corrected $[K^+]_a$ at end exercise was 5.73 $\pm$ 0.51 mM, greater than at rest and preexercise ($P < 0.001$), while the nadir in corrected plasma $[K^+]_a$ was 3.18 $\pm$ 0.19 mM at 5 min postexercise, less than both rest and preexercise ($P < 0.001$).

Plasma $[K^+]_a$ was also expressed per decile time during exercise. Plasma $[K^+]_a$ (uncorrected for plasma volume shifts) was increased above seated preexercise in the first decile, continued to increase in the second decile to 6.12 $\pm$ 0.46 mM, and remained at similar concentrations for the remainder of exercise ($P < 0.001$, Fig. 2).

**Plasma $[K^+]$ and power output correlation.** A significant negative correlation was found between plasma $[K^+]$ and power output, with all data points included from 30 s until end exercise being 100%. Values are expressed as means $\pm$ SD; $n = 10$. $^*P < 0.05$, different from 10% decile for power output or median frequency. $^**P < 0.001$, different from seated preexercise (0 min). A: black circles represent plasma $[K^+]$; white triangles represent average power output. B: all data points from 30 s to end exercise were included in correlation analysis. $n = 10$ subjects; $n = 135$ data points, $r = -0.48$, $P < 0.001$, $Y = -0.0036x + 7.09$. C: black circles represent iEMG; white triangles represent median frequency.
exercise pooled for all individuals \((r = -0.48, P < 0.001, \text{Fig. 2})\). When examined on an individual level, an inverse relationship between plasma \([K^+]\) and power output was found for 9 of 10 individuals (slope = $-0.0146 \pm 0.0109 \text{ mmol} \cdot \text{l}^{-1} \cdot \text{W}^{-1}$; \(r^2 = 0.52 \pm 0.25, n = 10\)). The largest power output values were typically achieved in the first 1–2 min when plasma \([K^+]\) was still increasing or just reaching a plateau, while thereafter plasma \([K^+]\) was maintained and the power output declined.

**Chloride.** Plasma \([\text{Cl}^-]_a\) increased slightly from supine rest (106.6 ± 1.9 mM) to seated preexercise \((P < 0.05)\). At end exercise (108.9 ± 2.2 mM), \([\text{Cl}^-]_a\) remained elevated above supine rest and seated preexercise \((P < 0.001)\), but this slight increase was considerably less in relative terms than the decline in PV, indicating a considerable net \(\text{Cl}^-\) loss from the circulation \((\text{Fig. 3})\). Plasma \([\text{Cl}^-]_a\) declined to below rest at 20 min postexercise and below preexercise levels at 30 min postexercise \((P < 0.05)\).

**Sodium.** Plasma \([Na^+]_a\) increased slightly by 30 s exercise compared with supine rest (136.4 ± 2.7 mM) and seated preexercise \((P < 0.05)\) and was elevated throughout exercise, reaching a peak of 147.4 ± 3.9 mM at end exercise \((P < 0.001)\); this increase (8.4%) was slightly less than the relative decline in plasma volume, indicating some net \(\text{Na}^+\) loss from the circulation. Postexercise, plasma \([Na^+]_a\) decreased to below preexercise rest at 10 min postexercise \((P < 0.05)\) and below resting levels at 20 min \((P < 0.05)\) \((\text{Fig. 3})\).

**Arterial Metabolic and Acid-Base Changes**

**pH.** Arterial plasma pH (\(\text{pH}_a\)) increased above supine rest \((-10 \text{ min})\) and seated preexercise \((0 \text{ min})\) during the initial 30 s of exercise \((P < 0.005)\), reflecting an initial pulmonary hyperventilation and consequent hypocapnia, with a sharp decline in arterial \(\text{PCO}_2\) \((\text{PaCO}_2)\) seen from 40.3 ± 2.7 to 33.6 ± 3.6 mmHg \((P < 0.005)\) and with \(\text{VE}\) already increased to 61.29 ± 3.85 l/min. The \(\text{pH}_a\) declined thereafter, reaching a nadir of 7.06 ± 0.08 units by 5 min postexercise \((P < 0.001)\), despite \(\text{PaCO}_2\) remaining low at 33.3 ± 3.9 mmHg. Recovery of \(\text{pH}_a\) was incomplete at 30 min postexercise \((P < 0.001)\, \text{Fig. 4}\).

**Lactate and glucose.** A considerable anaerobic contribution to exercise occurred, evidenced by blood \([\text{Lac}^-]_a\) increasing within the first 30 s and throughout exercise, reaching 10.87 ± 1.33 mmol/l at end exercise \((P < 0.001)\), subsequently decreasing during recovery, but remaining elevated at 30 min postexercise \((P < 0.001)\, \text{Fig. 4}\). Blood \([\text{glucose}]_a\) was unchanged during exercise, with sympathoadrenal-mediated liver glycogenolysis matched to rate of peripheral disposal. Blood \([\text{glucose}]_a\) then rapidly increased to a peak of 7.75 ± 1.19 mmol/l at 2 min postexercise \((P < 0.001)\), likely reflecting continued liver glycogenolysis exceeding a declining rate of peripheral disposal; thereafter blood \([\text{glucose}]_a\) declined but remained elevated at 20 min postexercise \((P < 0.05)\, \text{Fig. 4}\).

**sEMG**

The sum of iEMG from seven muscles averaged 73.25 ± 9.81% iEMGmax during exercise. When data were expressed in deciles of exercise time, the muscle iEMG tended to decline from 85.26 ± 4.45% iEMGmax during the first decile to 75.12 ± 6.88% iEMGmax during the second decile, but this and subsequent changes were not significant. The average median frequency was 63.50 ± 11.43 Hz during exercise. Median frequency expressed per decile time during exercise was 67.96 ± 11.40 Hz during the first decile; this decreased by 4.6% during the third decile and remained lower through to the sixth decile (−4.3%, \(P < 0.05)\) and was 5.5% lower at the eighth decile \((P < 0.05)\, \text{Fig. 2}\).

**DISCUSSION**

We utilized rowing as a model of high-intensity exercise utilizing a large contracting muscle mass, to study arterial plasma \(\text{K}^+\) dynamics during and following exercise, with two major findings. First, while \([\text{K}^+]_a\) showed the typical rapid increase during intense exercise, reaching ~6.1 mM after 90 s, \([\text{K}^+]_a\) was then sustained at this concentration plateau for the duration of exercise, lasting ~7.26 min. During this period of high \([\text{K}^+]_a\), muscle fatigue was evidenced by declines in each of the power output and average median frequency. Second, a
post-exercise hypokalemia occurred following rowing, with [K⁺]a falling to 3.3 mM and was sustained for 30 min postexercise.

Arterial Plasma K⁺ Dynamics During Rowing Exercise and the Role of Muscle Mass

While the rapid and early rise in plasma [K⁺] with intense exercise is well documented (30, 54), these findings are important as no previous studies have reported that arterial plasma [K⁺] can be maintained at this level during whole body exercise sustained for this duration. The hyperkalemia during the initial 90 s of exercise is attributed to a high rate of K⁺ release from active muscle exceeding the K⁺ clearance rate, which is mainly into contracting and inactive skeletal muscles. During submaximal exercise, the loss of K⁺ from muscle and rise in circulating [K⁺] are most pronounced within the first few minutes due to a delay in the activation of the Na⁺,K⁺-ATPase (15); plasma [K⁺] subsequently levels off, but at submaximal values. In contrast, maximal “sprinting” exercise induces increases in plasma [K⁺] until end exercise (54), and this also reflects a lag in the activation of the Na⁺,K⁺-ATPase (14). It was anticipated that during rowing exercise a plasma [K⁺] greater than 6.1 mM would have been achieved, since [K⁺]a can reach 7–8 mM during other forms of maximal exercise, such as cycling, which involves a lesser amount of active muscle mass (26, 48). While the arterial plasma [K⁺] measures during rowing are within the range of values reported for other intense exercise studies (48), these values were not as high as anticipated. One possible explanation why this did not occur may be the large adrenergic effect evident during heavy exercise involving a large muscle mass (3), since catecholamines stimulate the Na⁺,K⁺-ATPase in muscle and lower circulating [K⁺] during exercise (10). The large active muscle mass during rowing implies there is relatively little inactive muscle available to assist in K⁺ clearance during exercise, but this effect was probably outweighed by a greater overall activation of Na⁺,K⁺-ATPase in muscle due to the greater contracting muscle mass, thus contributing to enhanced K⁺ clearance (9). A further possibility is that the relative exercise intensity for each muscle group may be reduced with a large contracting muscle mass, with consequential lesser rate of K⁺ release in the contracting muscles. Given the high intensity and that maximal VO₂ (VO₂max) can typically be achieved during rowing as opposed to cycling, this seems less likely than enhanced rates of K⁺ clearance.

The impacts of elevated arterial plasma [K⁺] may be important as this will reflect elevated muscle interstitial [K⁺], which has been proposed to directly impair muscle cellular excitability (19). Muscle interstitial [K⁺] may reach 9–15 mM and exceed the plasma [K⁺] in venous effluent from contracting muscles by as much as 4–8 mM (36). Hence, it seems likely that the muscle interstitial [K⁺] reached during rowing must also be substantial.

An important observation in this regard was that while participants were able to tolerate rowing exercise with a sustained hyperkalemia for ~7 min, nonetheless, power output declined by between 17 and 21% during the fourth to ninth exercise deciles. This decrease in power output may be due to a combination of factors, including pacing, muscle fatigue, and reduced motivation. The decrease in power output associated with a decline in median frequency during the first to fourth decile of exercise is consistent with a possible pacing strategy used by the subjects (4, 13). This may be useful in reducing the recruitment of type II motor units and thus in minimizing the occurrence of early fatigue during exercise. However, the high exercise VO₂, Ve, HR, and [K⁺] during exercise, the 10-fold increase in blood [lactate]a, and drop in pHa to 7.1 all attest to a very high physiological stress throughout the rowing bout. Furthermore, perturbations in these variables were sustained throughout exercise which attests to a strong motivation required to achieve this. The sustained elevations in all of these variables during exercise suggests that the decline in power output cannot simply reflect a pacing strategy alone, but rather points to a more likely development of muscular fatigue. Whether these elevations in [K⁺]a and likely increases in muscle interstitial [K⁺] during rowing may contribute to fa-
tigue cannot, however, be concluded from these data. Correlation analysis revealed negative relationships between $[K^+]_a$ and power output for pooled data, as well as in all but one individual. This reflects both the highest power output being attained early in exercise when $[K^+]_a$ was still increasing and that power output subsequently fell while $[K^+]_a$ was then sustained.

Evidence against the entire decline in power output being due to fatigue was a tendency to a small increase in power output during the final decile, which indicates some additional capacity existed and suggests recruitment of additional muscle fibers occurred to maximize force and power output. Nevertheless, power output in the final exercise decile remained 16.2% less than that attained during the first decile [large effect size, $-1.58 (-2.51$ to $-0.52)$], and a high activation level of the muscles (i.e., $>70\%EMG_{Max}$) was maintained over the whole duration of the exercise. Analysis of the EMG activity of the active muscles revealed a decline in the EMG median frequency without concurrent diminution of the iEMG values. This suggests that declines in motor unit discharge rate, action potential propagation, and duration occurred during exercise (7). The EMG median frequency did not increase during the final interval of the exercise while iEMG and power output tended to increase, suggesting that fatigue was a likely causative factor in the markedly lowered power output during most of the rowing trial. The mechanism underlying this cannot be ascertained from the current data, but would be consistent with a reduced activation of, and action potential propagation in, Type II fibers. A decline in Type II fiber activation and/or excitability could be consistent with the greater $K^+$ loss evident in active muscles with predominantly Type II compared with those with Type I fibers (24, 51). More recent studies have, however, challenged the depressive role of $K^+$ disturbances in muscle fatigue, with the deleterious $K^+$ effects attenuated by acidosis mediated via reduced Cl$^-$ conductance (39, 40). The consequent effects, at least in isolated skeletal muscle, are that muscle action potentials, excitability, and force production are preserved at higher extracellular $[K^+]$ (6). What happens to muscle Cl$^-$ conductance in vivo during intense exercise is not known. During rowing exercise, plasma Cl$^-$ increased only slightly and to a lesser extent than the decline in plasma volume, indicating a net loss of Cl$^-$ from the circulation during exercise. This Cl$^-$ loss is consistent with other studies with intense exhaustive exercise (17, 47).

Hypokalemia After Rowing

An important finding was the rapid decline in plasma $[K^+]_a$ postexercise reaching a nadir of 3.33 mM at 5 min recovery. The postexercise decline in $[K^+]_a$ and consequent hypokalemia reflects the continuing reuptake of $K^+$ by previously active muscles, via elevated Na$^+$,K$^+$-ATPase activity, probably in part due to elevated levels of circulating catecholamines (21). A low postexercise $[K^+]$ was expected since the magnitude of decline in $[K^+]$ is proportional to exercise intensity (14, 25, 32, 54). This effect is highly likely also due to the large contracting muscle mass, since the decline in arterial $[K^+]$ from end exercise after rowing was larger (25-36%) at 2, 5, and 10 min postexercise than after cycling (45) and substantially (10-20-fold) greater than after forearm finger flexion exercise (50). The stress of this intense exercise with a very large contracting musculature was such that the $[K^+]_a$ did not return to preexercise level even at 30 min postexercise. While the importance of the Na$^+$,K$^+$-ATPase during muscle contraction is well recognized (9, 43), the potential impacts of continued Na$^+$,K$^+$-ATPase activity during recovery are less well understood. The extent of hypokalemia found in this study in healthy individuals has been linked to cardiac arrhythmias in patients with chronic conditions and is postulated as one possible factor in sudden death postexercise (8, 20, 52). It is possible that postexercise hypokalemia may induce a risk of arrhythmias in susceptible individuals. Interestingly, the $[K^+]$ corrected for plasma volume shifts highlighted that an even greater degree of hypokalemia would have been evident postexercise without the decline in plasma volume, with a nadir corrected $[K^+]$ of 3.18 mM rather than the observed 3.33 mM. Hence, fluid shifts may play an important protective role in recovery via attenuating the fall in plasma $[K^+]$ that would otherwise occur. While the resting plasma $[K^+]$ are on the lower end of the normative range, this likely reflects the careful postural controls, the healthy recreationally active participants, and possibly the $[K^+]_a$ measurement with ion-selective electrodes; these plasma $[K^+]$ are consistent with measures taken in our laboratory over many years.

Conclusions

High-intensity rowing exercise requires recruitment of a large contracting muscle mass and caused a marked elevation in arterial $[K^+]$ that was sustained for the final 6 min of a 2,000-m (7.26 min) rowing bout. This sustained arterial hyperkalemia during exercise reflects a balance between considerable $K^+$ release from and $K^+$ reuptake in contracting muscles and a restricted clearance of $K^+$ during exercise by inactive muscles due to the small amount of inactive muscle. The $[K^+]_a$ during rowing was however lower than anticipated compared with observed during maximal cycling or treadmill sprinting. This might reflect greater adrenergic activity and increased overall activation of Na$^+$,K$^+$-ATPase with a greater contracting muscle mass, and/or a lesser relative intensity of individual muscle groups, together resulting in overall reduced net $K^+$ loss from contracting muscles. While arterial plasma $[K^+]$ remained unchanged after 90 s of exercise, power output and EMG median frequency both declined while iEMG was unchanged, suggesting impaired muscle function was due to fatigue. This is consistent with a possible role of muscle $K^+$ disturbances in fatigue, especially in Type II muscle fibers. However, as muscle interstitial and intracellular ion concentrations, Cl$^-$ conductance, and membrane potential are not available during rowing exercise, it is not possible to ascertain whether these $K^+$ shifts are sufficiently high enough to cause muscle fatigue. Tendency to a small increase in power output at the end of the rowing effort, associated with an increased iEMG and no change in median frequency, indicates that more power is produced through a preferential activation of the slow muscle fibers resulting from fatigue. Finally, postexercise arterial $[K^+]$ fell to a mean value as low as 3.3 mM, and hypokalemia was sustained for 30 min following exercise, reflecting both the large muscle mass and high exercise intensity. Such low $[K^+]$ levels may pose a cardiovascular risk in those individuals susceptible to arrhythmias, but this requires further investigation.
DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS


REFERENCES