# Sprint training enhances ionic regulation during intense exercise in men

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1. This study investigated the effects of 7 weeks of sprint training on changes in electrolyte concentrations and acid–base status in arterial and femoral venous blood, during and following maximal exercise for 30 s on an isokinetic cycle ergometer.

2. Six healthy males performed maximal exercise, before and after training. Blood samples were drawn simultaneously from brachial arterial and femoral venous catheters, at rest, during the final 10 s of exercise and during 10 min of recovery, and analysed for whole blood and plasma ions and acid–base variables.

3. Maximal exercise performance was enhanced after training, with a 13% increase in total work output and a 14% less decline in power output during maximal cycling.

4. The acute changes in plasma volume, ions and acid–base variables during maximal exercise were similar to previous observations. Sprint training did not influence the decline in plasma volume during or following maximal exercise. After training, maximal exercise was accompanied by lower arterial and femoral venous plasma $[K^+]$ and $[Na^+]$ across all measurement times ($P < 0{\cdot}05$). Arterial plasma lactate concentration ($[Lac^-]$) was greater ($P < 0{\cdot}05$), but femoral venous plasma $[Lac^-]$ was unchanged by training.

5. Net release into, or uptake of ions from plasma passing through the exercising muscle was assessed by arteriovenous concentration differences, corrected for fluid movements. $K^+$ release into plasma during exercise, and a small net $K^+$ uptake from plasma 1 min post-exercise ($P < 0{\cdot}05$), were unchanged by training. A net $Na^+$ loss from plasma during exercise ($P < 0{\cdot}05$) tended to be reduced after training ($P < 0{\cdot}06$). Release of $Lac^-$ into plasma during and after exercise ($P < 0{\cdot}05$) was unchanged by training.

6. Arterial and venous plasma strong ion difference ([SID]; $[SID] = [Na^+] + [K^+] - [Lac^-] - [Cl^-]$) were lower after training (mean differences) by 2·7 and 1·8 mmol $l^{-1}$, respectively ($P < 0{\cdot}05$). Arterial and femoral venous $CO_2$ tensions and arterial plasma $[HCO_3^-]$ were lower after training (mean differences) by 1·7 mmHg, 4·5 mmHg and 1·2 mmol $l^{-1}$, respectively ($P < 0{\cdot}05$), with arterial plasma $[H^+]$ being greater after training by 2·2 nmol $l^{-1}$ ($P < 0{\cdot}05$).

7. The acute changes in whole blood volume and ion concentrations during maximal exercise were similar to previous observations. Arterial and femoral whole blood $[K^+]$ and $[Cl^-]$ were increased, whilst $[Na^+]$ was lower, across all observation times after training ($P < 0{\cdot}05$).

8. Net uptake or release of ions by exercising muscle was assessed by arteriovenous whole blood concentration differences, corrected for fluid movements. A net $K^+$ uptake by muscle occurred at all times, including exercise, but this was not significantly different after training. An increased net $Na^+$ uptake by muscle occurred during exercise ($P < 0{\cdot}05$) with greater $Na^+$ uptake after training ($P < 0{\cdot}05$). Net muscle $Lac^-$ release and $Cl^-$ uptake occurred at all times ($P < 0{\cdot}05$) and were unchanged by training.

9. Sprint training improved muscle ion regulation, associated with increased intense exercise performance, at the expense of a greater systemic acidosis. Increased muscle $Na^+$ and $K^+$ uptake by muscle during the final seconds of exercise after training are consistent with a greater activation of the muscle $Na^+$–$K^+$ pump, reduced cellular $K^+$ loss and the observed lesser rate of fatigue. The greater plasma acidosis found after sprint training was caused by a lower arterial plasma [SID] due to lower plasma $[K^+]$ and $[Na^+]$, and higher plasma $[Lac^-]$.

High-intensity exercise causes dramatic changes in fluid volumes, metabolites and ion concentrations in contracting skeletal muscle, which may impair muscular performance. During intense exercise, water, $Na^+$ and $Cl^-$ ions shift from plasma into exercising muscle, whilst $K^+$ and lactate ($Lac^-$) ions exit contracting muscle (Fenn, 1936; Lindinger & Heigenhauser, 1991; Lindinger, Heigenhauser, McKelvie & Jones, 1992). The contribution of these fluid and ionic changes to muscular fatigue during exercise is complex.

The fluid influx during exercise increases muscle intracellular and interstitial volumes, reducing muscle intracellular and interstitial ion and metabolite concentrations and concentrating plasma ions and metabolites (Sjøgaard & Saltin, 1982; Hermansen, Orheim & Sejersted, 1984; Medbø & Sejersted, 1985; Lindinger & Heigenhauser, 1991). The marked cellular $K^+$ efflux may cause muscle membrane depolarization and reduced excitability (Sjøgaard, Adams & Saltin, 1985; Kowalchuk, Heigenhauser, Lindinger, Sutton & Jones, 1988), whilst dramatic increases in [$Lac^-$] in muscle and in blood play a dominant role in the ensuing intracellular and systemic acidoses (Hermansen & Osnes, 1972; Hermansen *et al.* 1984; Medbø & Sejersted, 1985). Cellular $Na^+$ influx with intense contractions may result in a doubling of intracellular [$Na^+$], which may attenuate fatigue through activation of the $Na^+$–$K^+$ pump, thereby restricting cellular $K^+$ loss, as well as through constraining the intracellular acidosis (Fenn, 1936; Sjøgaard *et al.* 1985; Juel, 1986; Lindinger & Heigenhauser, 1991; Clausen & Nielsen, 1994). Any increase in intracellular water will exacerbate the contribution of reduced intracellular [$K^+$] to fatigue, but will also attenuate the contribution of elevated intracellular [$Lac^-$]. Changes in plasma ion concentrations during intense exercise are associated with a marked increase in plasma [$H^+$] (Kowalchuk *et al.* 1988; Jones, 1990; McKelvie, Lindinger, Heigenhauser & Jones, 1991; Lindinger *et al.* 1992). These ionic and metabolic changes are most pronounced with high-intensity exercise, which is also associated with the rapid development of fatigue (McCartney, Heigenhauser, Sargeant & Jones, 1983; Hermansen *et al.* 1984; Jones *et al.* 1985; McCartney, Spriet, Heigenhauser, Kowalchuk, Sutton & Jones, 1986; Kowalchuk *et al.* 1988; Medbø & Sejersted, 1990; Lindinger & Heigenhauser, 1991; Vøllestad, Hallén & Sejersted, 1994), suggesting a causal relationship between these severe intramuscular ionic and acid–base disturbances and fatigue. It is therefore likely that training at high-intensity work rates ('sprint training') will provide a large adaptive stimulus to regulatory mechanisms controlling ionic homeostasis. Such training-induced changes might then potentially decrease the rate of fatigue development during exercise.

Several longitudinal sprint training studies have demonstrated adaptive ionic regulatory mechanisms, associated with improved high-intensity exercise performance. Muscle $H^+$ regulation is enhanced after sprint training, with an increased *in vivo* muscle buffering capacity in humans (Sharp, Costill, Fink & King, 1986), and an increased muscle lactate transport capacity in rats (Pilegaard, Juel & Wibrand, 1993). Whole body $K^+$ regulation during intense exercise is improved after sprint training, with an increased skeletal muscle $Na^+$–$K^+$ pump concentration and an attenuated rise in plasma [$K^+$] relative to work output (McKenna, Schmidt, Hargreaves, Cameron, Skinner & Kjeldsen, 1993). However, none of the reported longitudinal studies have comprehensively investigated the effects of sprint training on fluid and ion homeostasis and the associated development of fatigue during high-intensity exercise. The present study therefore investigated the effects of sprint training on arteriovenous ion concentration differences across the exercising leg during maximal sprinting exercise. Arteriovenous ion differences were corrected for fluid shifts to assess the effects of intense training on fluid and ion net fluxes between contracting skeletal muscle and blood. We hypothesized that sprint training would modify the marked fluid and ionic exchange between muscle and blood during maximal exercise and thereby enhance sprint performance. Specifically, we hypothesized that this improved performance would be associated with augmented net muscle $Lac^-$ release and $Na^+$ uptake, as well as attenuated net muscle $K^+$ loss and fluid uptake. Plasma acid–base status was investigated because these ionic changes would potentially result in a more severe plasma acidosis, which might be self-limiting and reduce their potential benefits.

## METHODS

### Subjects

Six healthy males participated after being informed of risks associated with the study and giving written informed consent. All procedures were approved by the McMaster University Ethics Committee. The subjects were not highly trained and none participated in regular physical activities outside the training programme. Physical characteristics (means ± S.E.M.) of the subjects were: age, 21·2 ± 0·6 years; height, 178·4 ± 1·7 cm; body mass, 74·4 ± 2·6 kg; and maximal oxygen consumption ($\dot{V}_{O_2,max}$) on a cycle ergometer, 3·54 ± 0·18 l min$^{-1}$.

### Experimental overview

Each subject performed one maximal 30 s exercise bout on an isokinetic cycle ergometer, before and after sprint training. Blood samples were drawn simultaneously from a brachial artery and a femoral vein at rest, during exercise and in recovery and analysed for fluid shifts, plasma and whole blood ion concentrations and acid–base status. A further maximal 30 s sprint bout was also performed on a separate occasion before and after training, in which breath-by-breath ventilation and gas exchange were measured during and following exercise; these data are reported in the accompanying paper (McKenna, Heigenhauser, McKelvie, Obminski, MacDougall & Jones, 1997).

### Sprint training protocol

Subjects completed 7 weeks of sprint training, undertaking three sessions per week; five subjects completed all twenty-one training sessions and one completed all but one session. The sprint training protocol was adapted from Sharp *et al.* (1986), who demonstrated

improved muscle [$H^+$] regulation after training. Training was conducted in a laboratory under supervision and was performed on mechanically braked cycle ergometers (Monark, Varberg, Sweden). The flywheel tension was set at 0·075 kg (kg body mass)$^{-1}$ and remained constant for the duration of the training programme; subjects pedalled as fast as possible against the flywheel resistance, with the pedalling rate decreasing during the bout. Each training session consisted of several bouts of maximal exercise for 30 s separated by 4 min recovery periods. In the first week, four bouts per session were used with 4 min of recovery; the number of bouts was increased by two per session each week, up to ten per session at week 4, after which the recovery time was shortened by 30 s each week until week 6. Training was well tolerated, except for nausea felt by several subjects in the first few sessions.

### Maximal sprint performance

Subjects performed maximal sprint exercise at 100 r.p.m. before and after the sprint training programme on an isokinetic cycle ergometer, previously described in detail (McCartney *et al.* 1983). Torque was calibrated statically before each test (McCartney *et al.* 1983). The subject was seated at a comfortable saddle height, with feet secured to the pedals by toeclips and tape. The subject was instructed to catch up to the predetermined crank velocity of 100 r.p.m., before exerting maximal force on the pedals for the next 30 s. The crank velocity was held constant by an electrically controlled 2·2 kW DC electric motor. The torque generated during each pedal stroke was detected by strain gauges bonded to the pedal crank and transferred via a brass slip ring and Wheatstone bridge circuit to a computer (PDP 11-03; Digital Equipment). The torque signals were sampled every 10 ms, with the peak torque, peak instantaneous power, maximal average power and work done calculated for every pedal stroke. The total work in 30 s was also calculated from the sum of all pedal strokes and the percentage decline in peak power from maximal values early in the test to the lowest at the end of the test was expressed as the fatigue index.

### Subject preparation

Subjects were familiarized with the experimental procedures and reported to the laboratory in the morning, at least 2 h after a light breakfast. A local anaesthetic (2% lignocaine injection) was applied before catheterization of a brachial artery and femoral vein, as previously described (Kowalchuk *et al.* 1988). The catheters were kept patent by a slow saline (0·9% NaCl) infusion throughout the experiment. Subjects were seated on a chair for at least 10 min before resting blood samples were drawn simultaneously from the arterial (a) and venous (v) catheters. The subject then mounted the isokinetic cycle ergometer and performed maximal exercise for 30 s (McCartney *et al.* 1983). Blood samples were drawn during the final 10 s of the exercise bout and at 1, 2, 5 and 10 min of recovery. The exercise and first two recovery blood samples were obtained with the subject seated on the cycle ergometer; the final two recovery blood samples were obtained with the subject seated in a chair. In two of the six subjects it proved impossible to obtain venous blood samples within the last 10 s of exercise; thus for this observation point $n = 4$; for all other times $n = 6$. Owing to the limited sample volume obtained, $n = 5$ for whole blood $K^+$ and $Na^+$ analyses, with $n = 3$ during exercise.

### Blood processing

Eight millilitres of blood were drawn into syringes containing lithium-heparin and mixed well. Air bubbles were expelled, and 1·5 ml of blood was removed for haematocrit determinations and, following three freeze–thaw cycles, whole blood (wb) electrolyte analyses. The syringe was recapped tightly and placed on ice for subsequent analyses of $P_{CO_2}$, $P_{O_2}$ and pH (Corning 178 pH/Blood Gas Analyser). Haematocrit (Hct) was measured in duplicate using heparinized microhaematocrit tubes centrifuged for 15 min at 10 000 r.p.m. (IEC model MB micro-haematocrit centrifuge). Haemoglobin concentration ([Hb]) was determined photometrically in duplicate (Radiometer OSM2 Hemoximeter). Following the blood gas and pH determinations, the syringes were kept anaerobic whilst being centrifuged at 4 °C for 11 min at 2500 r.p.m., 5000 *g* (Sorval GLC 1 centrifuge); the separated plasma was analysed for protein and electrolyte concentrations. Plasma protein concentration ([$PPr^-$]) was measured in duplicate with a refractometer (Atago 331). Plasma and whole blood [$Na^+$] and [$K^+$] were measured with ion- selective electrodes (Radiometer KNA1). Plasma and whole blood [$Lac^-$] were measured by an enzymatic fluorimetric technique, after deproteinization of 200 $\mu$l of plasma or blood with 400 $\mu$l of cold 7% perchloric acid. Plasma and whole blood [$Cl^-$] were determined in duplicate by colorimetric titration (Buchler-Cotlove Chloridometer 4-2008). Analysers were calibrated immediately before and during the analyses with precision standards in the range of the measurements. Mean differences between duplicate measure ments (means ± S.E.M.) in plasma were: [$PPr^-$], 0·0 ± 0·01 g dl$^{-1}$; [$Lac^-$], 0·1 ± 0·12 mmol l$^{-1}$; [$Cl^-$], −0·2 ± 0·25 mmol l$^{-1}$; pH 0·00 ± 0·001; $P_{CO_2}$, 0·36 ± 0·11 mmHg; $P_{O_2}$, 0·96 ± 0·34 mmHg; and [Hb], −0·14 ± 0·04 g dl$^{-1}$. Analytical precision for plasma [$Na^+$] (0·6 ± 0·4 mmol l$^{-1}$) and [$K^+$] (0·02 ± 0·04 mmol l$^{-1}$) was previously published (Kowalchuk *et al.* 1988). Mean differences between duplicate measures in whole blood (means ± S.E.M. (coefficient of variation)) were: [$K^+$], 0·20 ± 0·25 mmol l$^{-1}$ (2·6%); [$Na^+$], 0·54 ± 0·45 mmol l$^{-1}$ (3·0%); [$Cl^-$], 0·37 ± 0·32 mmol l$^{-1}$ (2·3%); and [$Lac^-$], 0·1 ± 0·10 mmol l$^{-1}$ (3·9%).

### Calculations

Changes in arterial plasma volume ($\Delta PV_a$) and blood volume ($\Delta BV_a$) from resting levels and changes in venous compared with arterial plasma ($\Delta PV_{a-v}$) and blood ($\Delta BV_{a-v}$) were calculated during and following exercise from changes in [Hb] and Hct (Harrison, 1985). These were calculated to determine the effects of fluid shifts on ion concentrations in plasma and blood during and following exercise. All arteriovenous differences for plasma ions (in mmol l$^{-1}$) and proteins (in g dl$^{-1}$) were corrected for $\Delta PV_{a-v}$, using the equation: $[ion]_{a-v} = ([ion]_a/(1 + \Delta PV_{a-v})) - [ion]_v$. A similar correction was made for whole blood ions using $\Delta BV_{a-v}$. The rise in plasma [$K^+$] during exercise ($\Delta[K^+]$, in mmol l$^{-1}$) was calculated as the difference between resting and peak exercise plasma [$K^+$] and was expressed relative to cumulative 30 s work output, as the $\Delta[K^+]$ : work ratio ($\mu$mol l$^{-1}$ kJ$^{-1}$). Plasma strong ion difference ([SID], in mmol l$^{-1}$) was calculated as: $[SID] = [K^+] + [Na^+] - [Lac^-] - [Cl^-]$. Plasma total weak acids ([$A_{tot}$], in mmol l$^{-1}$) were calculated as: 2·45 × [$PPr^-$] (Rossing, Maffeo & Fencl, 1986). Plasma [$H^+$] was derived from the measured pH as the antilog.

### Statistics

All data are reported as means ± S.E.M. Training effects on sprint exercise performance were analysed using Student's paired, two-tailed $t$ test. All other data were analysed using three-way analysis of variance (measurement time, training and subject), with subsequent Newman–Keuls *post hoc* analyses. Interactions between time and training are referred to only when significant, and direct comparisons of pre- *versus* post-training at a given time were performed only when a significant interaction effect was found. A significance level of $P < 0·05$ was accepted for all statistical analyses.

**Table 1. Changes in maximal 30 s exercise performance on an isokinetic cycle ergometer (100 r.p.m.), before and after sprint training**

| | Maximal peak torque (N m) | Peak instantaneous power (W) | Maximal average power (W) | Cumulative work (kJ) | Fatigue index (%) |
|---|---|---|---|---|---|
| Pre-training | 137·1 ± 5·4 | 1469·7 ± 57·6 | 950·7 ± 47·4 | 19·0 ± 0·9 | 59·8 ± 2·7 |
| Post-training | 137·9 ± 8·8 | 1498·7 ± 99·5 | 998·1 ± 59·2 | 21·4 ± 1·4 | 45·6 ± 2·5 |
| Percentage change | 0 | 2·0 | 5·0 | 12·6* | −14·2* |

Values are means ± S.E.M. $n = 6$. * $P < 0·05$.

## RESULTS

### Maximal sprint performance

Cumulative work performed during the 30 s sprint bout was significantly increased after sprint training by 13% (Table 1). This was due to better maintenance of power output during exercise, as evidenced by a reduction in the fatigue index after training by 14% ($P < 0·05$). There were no significant differences in maximal peak torque, peak instantaneous power or maximal average power after training (Table 1).

### Haemoglobin, haematocrit and plasma protein

Arterial and venous [Hb] were greater than at rest during and following exercise ($P < 0·05$), but were unchanged after training (Table 2). Arterial and venous Hct increased above resting values by 1 min post-exercise and remained elevated ($P < 0·05$, Table 2). After training, both $Hct_a$ and $Hct_v$ were increased by a mean difference of 0·8% ($P < 0·05$), but the arteriovenous difference was unchanged. Both $[PPr^-]_a$ and $[PPr^-]_v$ increased to a peak at 2 min post-exercise and remained elevated ($P < 0·05$, Table 2). After training, $[PPr^-]_a$ was unchanged ($P = 0·10$), but $[PPr^-]_v$ was lower by a mean difference of 0·2 g dl$^{-1}$ ($P < 0·05$). After correction for $\Delta PV_{a-v}$ (see below), $[PPr^-]_{a-v}$ during exercise was more positive than at rest ($P < 0·05$, Table 2), indicating a net loss of protein across the leg during exercise. There was a significant interaction between training and time, with a greater $[PPr^-]_{a-v}$ at 2 and 5 min post-exercise after training ($P < 0·05$), indicating a greater net loss of protein from plasma after training.

**Table 2. Haemoglobin concentration, haematocrit and plasma protein concentration at rest, during and following maximal exercise, before and after sprint training**

| | Rest | Exercise | 1 min | 2 min | 5 min | 10 min |
|---|---|---|---|---|---|---|
| Pre-training | | | | | | |
| $[Hb]_a$ (g dl$^{-1}$)* | 15·6 ± 0·3 | 16·5 ± 0·4 | 16·9 ± 0·3 | 17·0 ± 0·3 | 16·8 ± 0·3 | 16·4 ± 0·3 |
| $[Hb]_v$ (g dl$^{-1}$)* | 15·9 ± 0·3 | 18·0 ± 0·2 | 17·3 ± 0·3 | 17·3 ± 0·3 | 17·1 ± 0·3 | 17·0 ± 0·5 |
| $Hct_a$ (%)* | 46·5 ± 0·6 | 46·8 ± 1·2 | 49·2 ± 0·8 | 49·9 ± 0·6 | 49·3 ± 0·7 | 48·8 ± 0·5 |
| $Hct_v$ (%)* | 46·4 ± 0·8 | 50·1 ± 0·4 | 50·3 ± 0·4 | 50·2 ± 0·7 | 49·7 ± 0·9 | 49·4 ± 0·6 |
| $[PPr^-]_a$ (g dl$^{-1}$)* | 6·9 ± 0·2 | 7·5 ± 0·3 | 7·9 ± 0·2 | 8·0 ± 0·2 | 7·7 ± 0·2 | 7·5 ± 0·2 |
| $[PPr^-]_v$ (g dl$^{-1}$)* | 7·1 ± 0·2 | 8·3 ± 0·3 | 8·4 ± 0·3 | 8·4 ± 0·3 | 8·0 ± 0·2 | 7·6 ± 0·2 |
| $[PPr^-]_{a-v}$ (g dl$^{-1}$)* | 0·0 ± 0·1 | 0·4 ± 0·2 | −0·1 ± 0·1 | −0·2 ± 0·1 | 0·0 ± 0·1 | 0·3 ± 0·1 |
| Post-training | | | | | | |
| $[Hb]_a$ (g dl$^{-1}$) | 15·6 ± 0·2 | 16·5 ± 0·2 | 17·0 ± 0·3 | 16·9 ± 0·2 | 16·6 ± 0·2 | 16·6 ± 0·2 |
| $[Hb]_v$ (g dl$^{-1}$) | 15·9 ± 0·2 | 17·7 ± 0·3 | 17·2 ± 0·2 | 17·5 ± 0·3 | 16·8 ± 0·2 | 16·7 ± 0·2 |
| $Hct_a$ (%)† | 46·8 ± 0·6 | 48·3 ± 0·4 | 49·3 ± 0·2 | 50·3 ± 0·4 | 50·3 ± 0·5 | 49·8 ± 0·4 |
| $Hct_v$ (%)† | 47·2 ± 0·3 | 49·0 ± 0·4 | 50·4 ± 0·3 | 51·1 ± 0·2 | 51·2 ± 0·5 | 50·2 ± 0·4 |
| $[PPr^-]_a$ (g dl$^{-1}$) | 6·9 ± 0·2 | 7·5 ± 0·2 | 7·8 ± 0·2 | 7·9 ± 0·2 | 7·7 ± 0·2 | 7·3 ± 0·2 |
| $[PPr^-]_v$ (g dl$^{-1}$)† | 7·0 ± 0·2 | 7·9 ± 0·4 | 8·2 ± 0·3 | 8·2 ± 0·3 | 7·8 ± 0·2 | 7·5 ± 0·2 |
| $[PPr^-]_{a-v}$ (g dl$^{-1}$)‡ | 0·1 ± 0·1 | 0·2 ± 0·1 | 0·0 ± 0·1 | 0·1 ± 0·1 | 0·2 ± 0·1 | −0·1 ± 0·1 |

$n = 6$, except for venous samples and arteriovenous differences during exercise, where $n = 4$. * Significant time main effect ($P < 0·05$); † significant training main effect ($P < 0·05$); ‡ significant time by training interaction ($P < 0·05$). Details are reported in the text.

**Table 3. Percentage change in plasma volume (ΔPV) and blood volume (ΔBV), during and following maximal exercise, before and after sprint training**

| | Rest | Exercise | 1 min | 2 min | 5 min | 10 min |
|---|---|---|---|---|---|---|
| Pre-training | | | | | | |
| $\Delta PV_a$ * | 0 | −6·7 ± 1·3 | −12·4 ± 1·3 | −14·3 ± 1·1 | −11·9 ± 1·3 | −8·6 ± 1·7 |
| $\Delta PV_v$ * | 0 | −14·8 ± 1·3 | −14·4 ± 0·6 | −14·3 ± 0·6 | −12·3 ± 1·9 | −11·2 ± 2·8 |
| $\Delta PV_{a-v}$ * | −2·0 ± 1·2 | −14·2 ± 3·2 | −4·3 ± 1·4 | −2·0 ± 0·7 | −2·5 ± 1·1 | −4·9 ± 1·9 |
| $\Delta BV_a$ * | 0 | −5·4 ± 0·8 | −7·8 ± 0·7 | −8·5 ± 1·1 | −7·1 ± 0·8 | −4·7 ± 1·2 |
| $\Delta BV_v$ * | 0 | −11·2 ± 1·0 | −7·9 ± 0·7 | −7·8 ± 0·6 | −6·6 ± 0·6 | −5·4 ± 1·5 |
| $\Delta BV_{a-v}$ * | −2·2 ± 0·4 | −8·8 ± 1·1 | −2·3 ± 0·7 | −1·4 ± 1·1 | −1·7 ± 0·8 | −3·0 ± 1·2 |
| Post-training | | | | | | |
| $\Delta PV_a$ | 0 | −8·2 ± 0·6 | −12·6 ± 1·8 | −14·1 ± 1·2 | −12·4 ± 0·8 | −11·6 ± 1·6 |
| $\Delta PV_v$ | 0 | −15·1 ± 1·9 | −12·6 ± 1·3 | −14·8 ± 1·2 | −11·9 ± 1·5 | −9·2 ± 2·2 |
| $\Delta PV_{a-v}$ | −2·8 ± 1·1 | −9·1 ± 1·8 | −3·5 ± 0·6 | −4·3 ± 1·0 | −3·0 ± 1·0 | −0·9 ± 0·9 |
| $\Delta BV_a$ | 0 | −5·6 ± 0·3 | −8·4 ± 1·2 | −8·2 ± 0·7 | −6·4 ± 0·8 | −6·4 ± 1·5 |
| $\Delta BV_v$ | 0 | −11·1 ± 1·8 | −7·6 ± 0·7 | −8·7 ± 1·2 | −5·5 ± 0·7 | −4·5 ± 1·5 |
| $\Delta BV_{a-v}$ | −2·2 ± 0·7 | −7·6 ± 1·2 | −1·4 ± 0·7 | −2·8 ± 0·8 | −1·2 ± 0·6 | −0·3 ± 1·1 |

Sample sizes as in Table 2. * Significant time main effect ($P < 0·05$). Details are reported in the text.

## Changes in plasma and blood volumes with exercise

Arterial and venous PV declined during exercise and early recovery and remained depressed thereafter ($P < 0·05$, Table 3), with no significant training effects. The arterio-venous decline in PV ($\Delta PV_{a-v}$) became more negative during exercise ($P < 0·05$, Table 3), indicating a net fall in PV across the exercising leg, but there was a return to pre-exercise values by 1 min post-exercise. Although no significant training effect was found, there was a trend in $\Delta PV_{a-v}$ that suggested a reduced decline during exercise after training ($P < 0·08$). Similar, but quantitatively smaller, results were found for ΔBV as for ΔPV during exercise and after training ($P < 0·05$, Table 3). The $\Delta BV_{a-v}$ fell markedly during exercise ($P < 0·05$), recovered by 1 min post-exercise, and was not modified after training.

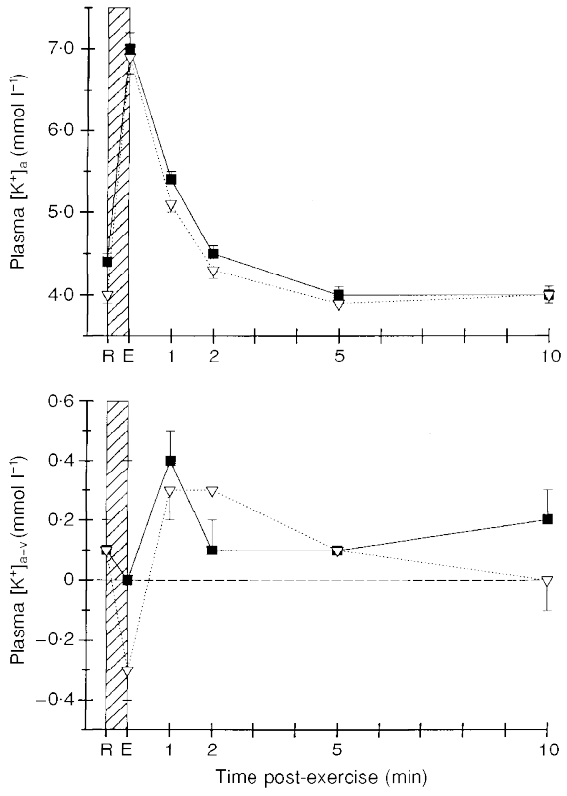


**Figure 1. Arterial plasma [$K^+$] (top) and arteriovenous plasma [$K^+$] differences (bottom)**

Measurements were obtained at rest (R), during (denoted by hatched bar here and in subsequent figures) and after exercise (E), before (■) and after (▽) sprint training. Arteriovenous differences are corrected for the decline in plasma volume; values are means ± S.E.M.; $n = 6$ except arteriovenous difference during exercise when $n = 4$. Dashed line indicates zero arteriovenous difference.

**Table 4. Arterial plasma strong ion concentrations and acid–base variables at rest, during and following intense exercise, before and after sprint training**

| | Rest | Exercise | 1 min | 2 min | 5 min | 10 min |
|---|---|---|---|---|---|---|
| Pre-training | | | | | | |
| $[K^+]_a$ (mmol $l^{-1}$)* | 4·4 ± 0·1 | 7·0 ± 0·2 | 5·4 ± 0·1 | 4·5 ± 0·1 | 4·0 ± 0·1 | 4·0 ± 0·1 |
| $[Na^+]_a$ (mmol $l^{-1}$)* | 139·7 ± 0·4 | 145·8 ± 0·6 | 148·3 ± 1·0 | 147·0 ± 1·0 | 143·8 ± 1·0 | 141·5 ± 0·7 |
| $[Lac^-]_a$ (mmol $l^{-1}$)* | 1·3 ± 0·2 | 6·4 ± 0·6 | 18·1 ± 1·1 | 20·6 ± 1·0 | 20·6 ± 1·3 | 19·0 ± 1·4 |
| $[Cl^-]_a$ (mmol $l^{-1}$)* | 97·3 ± 1·3 | 103·9 ± 2·0 | 101·3 ± 1·7 | 100·3 ± 1·6 | 97·8 ± 1·2 | 96·5 ± 0·9 |
| $[SID]_a$ (mmol $l^{-1}$)* | 45·4 ± 1·3 | 42·5 ± 2·0 | 34·4 ± 1·6 | 30·6 ± 1·6 | 29·6 ± 1·3 | 30·1 ± 1·4 |
| $P_{a,CO_2}$ (mmHg)* | 43·9 ± 0·7 | 38·2 ± 1·6 | 37·6 ± 1·7 | 34·0 ± 1·3 | 31·6 ± 1·5 | 32·8 ± 1·6 |
| $[A_{tot}]_a$ (mmol $l^{-1}$)* | 16·8 ± 0·5 | 18·3 ± 0·6 | 19·2 ± 0·6 | 19·3 ± 0·6 | 18·7 ± 0·5 | 18·2 ± 0·5 |
| $[H^+]_a$ (nmol $l^{-1}$)* | 39·3 ± 0·3 | 39·8 ± 1·4 | 58·3 ± 1·6 | 62·7 ± 2·1 | 65·8 ± 3·6 | 65·8 ± 4·3 |
| $[HCO_3^-]_a$ (mmol $l^{-1}$)* | 27·5 ± 0·4 | 23·7 ± 0·6 | 15·9 ± 0·8 | 13·4 ± 0·6 | 11·9 ± 0·6 | 12·5 ± 0·8 |
| Post-training | | | | | | |
| $[K^+]_a$ (mmol $l^{-1}$)† | 4·0 ± 0·1 | 6·9 ± 0·2 | 5·1 ± 0·1 | 4·3 ± 0·1 | 3·9 ± 0·0 | 4·0 ± 0·1 |
| $[Na^+]_a$ (mmol $l^{-1}$)† | 138·7 ± 0·8 | 145·2 ± 0·7 | 146·8 ± 1·2 | 145·2 ± 1·4 | 142·0 ± 0·9 | 138·8 ± 1·9 |
| $[Lac^-]_a$ (mmol $l^{-1}$)† | 1·5 ± 0·2 | 7·3 ± 1·0 | 20·2 ± 1·3 | 21·4 ± 1·3 | 21·7 ± 1·4 | 19·9 ± 1·5 |
| $[Cl^-]_a$ (mmol $l^{-1}$) | 97·1 ± 1·5 | 103·7 ± 1·1 | 102·4 ± 0·8 | 99·7 ± 0·9 | 98·1 ± 0·8 | 95·3 ± 1·5 |
| $[SID]_a$ (mmol $l^{-1}$)† | 44·1 ± 1·3 | 41·2 ± 1·4 | 29·3 ± 1·2 | 28·3 ± 0·9 | 26·1 ± 0·8 | 27·6 ± 0·9 |
| $P_{a,CO_2}$ (mmHg)† | 41·9 ± 1·2 | 38·2 ± 1·3 | 35·4 ± 1·1 | 31·6 ± 1·0 | 29·4 ± 1·6 | 31·4 ± 1·1 |
| $[A_{tot}]_a$ (mmol $l^{-1}$) | 16·6 ± 0·4 | 18·4 ± 0·6 | 18·9 ± 0·5 | 19·1 ± 0·5 | 18·6 ± 0·5 | 17·8 ± 0·4 |
| $[H^+]_a$ (nmol $l^{-1}$)† | 38·6 ± 0·4 | 41·3 ± 1·3 | 61·0 ± 1·6 | 65·7 ± 2·1 | 69·8 ± 3·1 | 68·1 ± 3·0 |
| $[HCO_3^-]_a$ (mmol $l^{-1}$)† | 26·8 ± 0·8 | 22·7 ± 0·6 | 14·3 ± 0·4 | 11·9 ± 0·4 | 10·4 ± 0·7 | 11·4 ± 0·7 |

* Significant time main effect ($P < 0·05$); † significant training main effect ($P < 0·05$). Details are reported in the text.

**Table 5. Femoral venous plasma strong ion concentrations and acid–base variables at rest, during and following intense exercise, before and after sprint training**

| | Rest | Exercise | 1 min | 2 min | 5 min | 10 min |
|---|---|---|---|---|---|---|
| Pre-training | | | | | | |
| $[K^+]_v$ (mmol $l^{-1}$)* | 4·4 ± 0·1 | 8·2 ± 0·2 | 5·3 ± 0·1 | 4·5 ± 0·1 | 4·1 ± 0·1 | 4·1 ± 0·1 |
| $[Na^+]_v$ (mmol $l^{-1}$)* | 140·5 ± 0·3 | 155·8 ± 1·4 | 153·5 ± 1·1 | 149·8 ± 1·3 | 145·0 ± 0·9 | 141·5 ± 0·9 |
| $[Lac^-]_v$ (mmol $l^{-1}$)* | 1·5 ± 0·2 | 11·5 ± 2·1 | 23·1 ± 1·3 | 24·7 ± 1·1 | 23·9 ± 0·8 | 21·9 ± 1·4 |
| $[Cl^-]_v$ (mmol $l^{-1}$)* | 96·1 ± 1·1 | 101·8 ± 1·2 | 96·9 ± 1·4 | 96·2 ± 1·5 | 94·8 ± 1·4 | 93·7 ± 1·2 |
| $[SID]_v$ (mmol $l^{-1}$)* | 47·3 ± 1·4 | 50·7 ± 1·9 | 38·7 ± 1·6 | 33·5 ± 1·3 | 30·3 ± 1·6 | 30·0 ± 1·6 |
| $P_{v,CO_2}$ (mmHg)* | 48·2 ± 1·3 | 102·8 ± 4·4 | 97·2 ± 3·1 | 69·4 ± 4·7 | 54·0 ± 3·5 | 46·9 ± 3·6 |
| $[A_{tot}]_v$ (mmol $l^{-1}$)* | 17·1 ± 0·5 | 20·0 ± 0·8 | 20·3 ± 0·6 | 20·3 ± 0·6 | 19·3 ± 0·6 | 18·5 ± 0·5 |
| $[H^+]_v$ (nmol $l^{-1}$)* | 40·9 ± 0·7 | 77·0 ± 7·6 | 107·3 ± 5·2 | 98·0 ± 7·0 | 89·9 ± 6·2 | 80·3 ± 6·0 |
| $[HCO_3^-]_v$ (mmol $l^{-1}$)* | 29·0 ± 0·5 | 33·2 ± 1·3 | 22·5 ± 1·2 | 17·4 ± 0·5 | 14·8 ± 0·3 | 14·4 ± 0·7 |
| Post-training | | | | | | |
| $[K^+]_v$ (mmol $l^{-1}$)† | 4·0 ± 0·1 | 7·8 ± 0·4 | 5·0 ± 0·1 | 4·2 ± 0·0 | 3·9 ± 0·1 | 4·1 ± 0·1 |
| $[Na^+]_v$ (mmol $l^{-1}$)†‡ | 139·3 ± 0·6 | 152·8 ± 1·8 | 150·8 ± 1·6 | 147·3 ± 1·2 | 143·7 ± 1·0 | 140·8 ± 0·9 |
| $[Lac^-]_v$ (mmol $l^{-1}$) | 1·7 ± 0·2 | 12·0 ± 3·3 | 23·9 ± 1·7 | 25·6 ± 1·7 | 24·4 ± 1·6 | 21·6 ± 1·6 |
| $[Cl^-]_v$ (mmol $l^{-1}$)‡ | 96·6 ± 0·9 | 100·1 ± 1·1 | 97·0 ± 1·1 | 95·6 ± 1·8 | 93·8 ± 1·4 | 93·8 ± 1·1 |
| $[SID]_v$ (mmol $l^{-1}$)†‡ | 45·1 ± 1·0 | 48·5 ± 2·2 | 34·9 ± 1·7 | 30·4 ± 1·1 | 29·5 ± 1·1 | 29·5 ± 0·9 |
| $P_{v,CO_2}$ (mmHg)† | 47·3 ± 1·5 | 93·4 ± 7·4 | 89·3 ± 4·2 | 64·4 ± 3·0 | 51·0 ± 3·2 | 46·2 ± 3·5 |
| $[A_{tot}]_v$ (mmol $l^{-1}$)† | 16·9 ± 0·5 | 19·1 ± 1·1 | 19·7 ± 0·6 | 19·8 ± 0·6 | 18·8 ± 0·5 | 18·3 ± 0·5 |
| $[H^+]_v$ (nmol $l^{-1}$) | 40·9 ± 0·8 | 73·2 ± 7·8 | 109·5 ± 4·0 | 103·7 ± 6·3 | 94·8 ± 5·4 | 81·6 ± 5·1 |
| $[HCO_3^-]_v$ (mmol $l^{-1}$)† | 28·5 ± 0·9 | 31·5 ± 1·2 | 20·2 ± 0·8 | 15·4 ± 0·4 | 13·3 ± 0·7 | 13·9 ± 0·7 |

Sample size as in Table 2. * Significant time main effect ($P < 0·05$); † significant training main effect ($P < 0·05$); ‡ time by training interaction ($P < 0·08$). Details are reported in the text.

**Table 6. Whole blood (wb) strong ion concentrations at rest, during and following maximal exercise, before and after sprint training**

| | Rest | Exercise | 1 min | 2 min | 5 min | 10 min |
|---|---|---|---|---|---|---|
| Pre-training | | | | | | |
| $[K^+]_{wb,a}$ * | 53·7 ± 1·1 | 58·9 ± 1·1 | 59·2 ± 1·1 | 58·8 ± 1·4 | 57·1 ± 1·3 | 55·7 ± 1·2 |
| $[K^+]_{wb,v}$ * | 54·2 ± 1·0 | 64·0 ± 0·8 | 60·6 ± 1·4 | 59·3 ± 1·0 | 57·5 ± 1·1 | 55·6 ± 1·1 |
| $[K^+]_{wb,a-v}$ | 0·8 ± 0·4 | 1·2 ± 0·6 | 0·3 ± 0·6 | 0·7 ± 1·1 | 0·4 ± 0·5 | 1·3 ± 0·2 |
| $[Na^+]_{wb,a}$ * | 94·0 ± 1·0 | 96·2 ± 1·5 | 96·6 ± 1·5 | 95·8 ± 1·6 | 93·0 ± 1·2 | 93·5 ± 1·5 |
| $[Na^+]_{wb,v}$ * | 93·0 ± 1·1 | 100·7 ± 2·3 | 98·8 ± 0·9 | 96·4 ± 0·8 | 93·4 ± 1·1 | 95·6 ± 1·5 |
| $[Na^+]_{wb,a-v}$ * | 3·3 ± 0·4 | 4·7 ± 1·3 | 0·3 ± 1·5 | −0·5 ± 1·7 | 0·9 ± 1·2 | −0·1 ± 2·5 |
| $[Lac^-]_{wb,a}$ * | 1·5 ± 0·1 | 5·1 ± 0·5 | 13·2 ± 0·7 | 15·0 ± 0·8 | 16·2 ± 1·0 | 15·4 ± 1·2 |
| $[Lac^-]_{wb,v}$ * | 1·7 ± 0·2 | 8·9 ± 1·3 | 16·9 ± 0·8 | 18·2 ± 1·0 | 18·5 ± 0·8 | 16·8 ± 1·2 |
| $[Lac^-]_{wb,a-v}$ * | −0·1 ± 0·1 | −3·5 ± 1·0 | −3·3 ± 0·4 | −3·0 ± 0·7 | −2·0 ± 0·8 | −0·7 ± 0·5 |
| $[Cl^-]_{wb,a}$ * | 78·5 ± 0·7 | 82·9 ± 1·4 | 84·8 ± 1·3 | 84·0 ± 1·1 | 81·9 ± 1·3 | 80·6 ± 1·5 |
| $[Cl^-]_{wb,v}$ * | 77·9 ± 0·7 | 85·0 ± 2·1 | 84·7 ± 0·6 | 83·8 ± 0·8 | 82·5 ± 0·9 | 81·9 ± 0·8 |
| $[Cl^-]_{wb,a-v}$ | 2·4 ± 0·8 | 6·0 ± 1·4 | 2·1 ± 1·0 | 1·5 ± 1·7 | 0·9 ± 1·3 | 1·9 ± 2·4 |
| Post-training | | | | | | |
| $[K^+]_{wb,a}$ † | 55·3 ± 0·4 | 62·2 ± 1·0 | 61·9 ± 0·9 | 60·6 ± 0·9 | 59·6 ± 1·0 | 58·0 ± 1·2 |
| $[K^+]_{wb,v}$ † | 56·0 ± 0·6 | 65·6 ± 0·9 | 62·4 ± 0·7 | 62·0 ± 0·9 | 59·0 ± 1·3 | 57·6 ± 1·2 |
| $[K^+]_{wb,a-v}$ | 0·3 ± 0·5 | 2·6 ± 1·0 | 0·8 ± 1·2 | −0·1 ± 1·2 | 1·2 ± 0·8 | 0·6 ± 0·7 |
| $[Na^+]_{wb,a}$ † | 93·0 ± 1·0 | 95·6 ± 1·2 | 96·6 ± 0·9 | 93·5 ± 2·9 | 91·3 ± 1·7 | 91·0 ± 1·2 |
| $[Na^+]_{wb,v}$ † | 93·6 ± 1·9 | 100·0 ± 3·1 | 96·4 ± 1·5 | 94·2 ± 1·6 | 90·4 ± 1·2 | 90·4 ± 1·5 |
| $[Na^+]_{wb,a-v}$ † | 2·1 ± 0·8 | 8·3 ± 2·0 | 1·0 ± 0·7 | 3·2 ± 1·1 | 2·0 ± 0·2 | 1·3 ± 0·8 |
| $[Lac^-]_{wb,a}$ | 1·1 ± 0·1 | 5·0 ± 0·6 | 14·2 ± 1·0 | 15·3 ± 0·9 | 16·4 ± 1·0 | 15·6 ± 1·2 |
| $[Lac^-]_{wb,v}$ | 1·3 ± 0·1 | 8·6 ± 2·2 | 17·1 ± 1·6 | 18·7 ± 1·4 | 19·1 ± 1·3 | 16·8 ± 1·3 |
| $[Lac^-]_{wb,a-v}$ | −0·2 ± 0·1 | −4·1 ± 1·9 | −2·8 ± 0·6 | −2·9 ± 0·6 | −2·5 ± 0·6 | −1·1 ± 0·4 |
| $[Cl^-]_{wb,a}$ † | 82·7 ± 1·4 | 87·9 ± 1·7 | 88·9 ± 1·7 | 87·7 ± 1·4 | 86·9 ± 1·7 | 87·3 ± 2·3 |
| $[Cl^-]_{wb,v}$ † | 80·6 ± 1·1 | 92·6 ± 2·9 | 88·3 ± 1·7 | 90·0 ± 2·2 | 87·7 ± 2·1 | 84·3 ± 0·9 |
| $[Cl^-]_{wb,a-v}$ | 4·0 ± 0·7 | 2·4 ± 3·7 | 1·8 ± 1·3 | 0·2 ± 2·0 | 0·3 ± 0·6 | 3·3 ± 2·2 |

All values are in millimoles per litre. Sample size: $[Lac^-]$ and $[Cl^-]$, $n = 6$; $[K^+]$ and $[Na^+]$, $n = 5$, except for venous samples and arteriovenous differences during exercise, where $[Lac^-]$ and $[Cl^-]$, $n = 4$; $[K^+]$ and $[Na^+]$, $n = 3$. * Significant time main effect ($P < 0·05$); † significant training main effect ($P < 0·05$). Details are reported in the text.

## Plasma and whole blood electrolytes

### Potassium

**Plasma.** Peak plasma $[K^+]_a$ and $[K^+]_v$ values were found during exercise; both declined rapidly following exercise and returned to resting values by 5 and 2 min post-exercise, respectively ($P < 0·05$, Fig. 1 and Tables 4 and 5). Both $[K^+]_a$ and $[K^+]_v$ were lower after training, by mean differences of 0·2 and 0·3 mmol $l^{-1}$, respectively ($P < 0·05$). Changes in $[K^+]_{a-v}$ indicated a small net release of $K^+$ into plasma during exercise and a small net loss from plasma at 1 min post-exercise ($P < 0·05$, Fig. 1), but no significant training main effects were found for $[K^+]_{a-v}$. The $\Delta[K^+]_a$ and $\Delta[K^+]_v$ were unchanged after training (arterial: pre-training, 2·2 mmol $l^{-1}$ *vs.* post-training, 2·4 mmol $l^{-1}$; venous: pre-training, 2·5 mmol $l^{-1}$ *vs.* post-training, 2·8 mmol $l^{-1}$). The calculated $\Delta[K^+]$ : work ratios were not significantly different after training (arterial: pre-training, 115·0 ± 7·5 $\mu$mol $l^{-1}$ $kJ^{-1}$ *vs.* post-training, 112·9 ± 10·8 $\mu$mol $l^{-1}$ $kJ^{-1}$; venous: pre-training, 141·9 ± 5·1 $\mu$mol $l^{-1}$ $kJ^{-1}$ *vs.* post-training, 145·0 ± 23·3 $\mu$mol $l^{-1}$ $kJ^{-1}$).

**Whole blood.** Both arterial whole blood $[K^+]$ ($[K^+]_{wb,a}$) and femoral venous whole blood $[K^+]$ ($[K^+]_{wb,v}$) peaked during exercise ($P < 0·05$) and then declined progressively, but remained elevated at 10 min post-exercise ($P < 0·05$, Table 6 and Fig. 2). Both $[K^+]_{wb,a}$ and $[K^+]_{wb,v}$ were increased after training, by mean differences of 2·4 and 1·9 mmol $l^{-1}$, respectively ($P < 0·05$, Table 6 and Fig. 2). The uncorrected arteriovenous $[K^+]_{wb}$ difference was more negative during exercise than at other time points ($P < 0·05$), suggesting $K^+$ release from muscle during exercise. However, after correction for $\Delta BV_{a-v}$, $[K^+]_{wb,a-v}$ did not differ between observation time points and was not significantly different after training (Fig. 2); $[K^+]_{wb,a-v}$ was positive at all times, suggesting a net muscle $K^+$ uptake.

### Sodium

**Plasma.** Both plasma $[Na^+]_a$ and $[Na^+]_v$ rose during exercise and reached peak values at 1 min post-exercise and during exercise, respectively ($P < 0·05$); both returned to resting values by 10 min post-exercise (Fig. 3 and Tables 4 and 5).

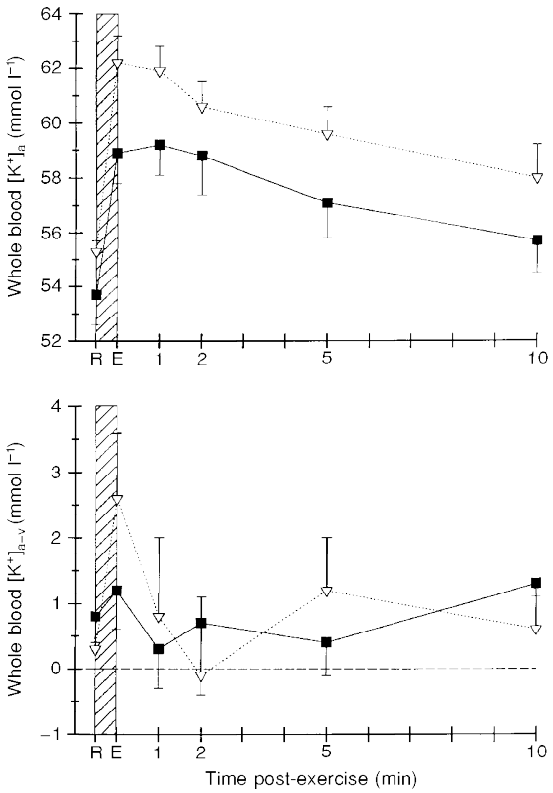


**Figure 2. Arterial whole blood [$K^+$] (top) and arteriovenous whole blood [$K^+$] differences (bottom)**
Measurements were obtained at rest (R), during and after exercise (E), before (■) and after (▽) sprint training. Arteriovenous differences are corrected for the arteriovenous decline in blood volume. Sample size as in Table 6. Dashed line indicates zero arteriovenous difference.

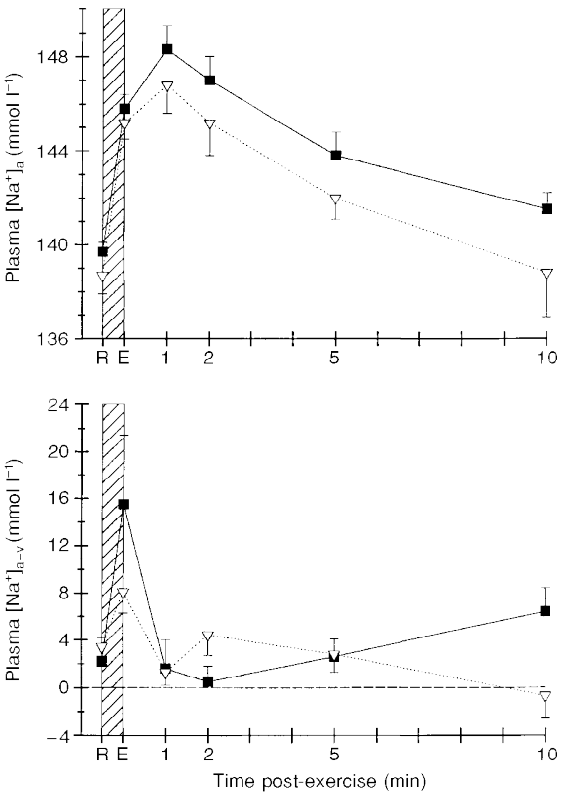


**Figure 3. Arterial plasma [$Na^+$] (top) and arteriovenous plasma [$Na^+$] differences (bottom)**
Measurements were obtained at rest (R), during and after maximal exercise (E), before (■) and after (▽) sprint training. Sample size and corrections as in Fig. 1. Dashed line indicates zero arteriovenous difference.

Plasma $[Na^+]_{a-v}$ increased during exercise, indicating a greater net loss of $Na^+$ from plasma than at rest ($P < 0{\cdot}05$, Fig. 3); recovery $[Na^+]_{a-v}$ was not significantly different from rest. Both $[Na^+]_a$ and $[Na^+]_v$ were lower after training, by mean differences of 1·6 and 1·9 mmol $l^{-1}$, respectively ($P < 0{\cdot}05$). Although no significant training effects were found for $[Na^+]_{a-v}$ at any given time of observation, the interaction between time and training ($P < 0{\cdot}06$) suggested a greater net $Na^+$ loss from plasma across the leg during exercise after training.

**Whole blood.** $[Na^+]_{wb,a}$ and $[Na^+]_{wb,v}$ increased with a similar time course to changes in plasma $[Na^+]$ ($P < 0{\cdot}05$), but recovered more rapidly to resting levels by 5 and 2 min post-exercise, respectively (Table 6 and Fig. 4). The $[Na^+]_{wb,a-v}$ became more positive during exercise, indicating increased net $Na^+$ uptake across the leg ($P < 0{\cdot}05$, Table 6). Both $[Na^+]_{wb,a}$ ($P < 0{\cdot}06$) and $[Na^+]_{wb,v}$ ($P < 0{\cdot}05$) were lower after training by mean differences of 0·9 and 2·2 mmol $l^{-1}$, respectively (Table 6 and Fig. 4). The $[Na^+]_{wb,a-v}$ was greater after training by a mean difference of 1·8 mmol $l^{-1}$ ($P < 0{\cdot}05$), indicating increased net $Na^+$ uptake across the leg after training.

### Lactate

**Plasma.** Plasma $[Lac^-]_a$ and $[Lac^-]_v$ rose sharply during exercise and early recovery, peaking at 2 min post-exercise ($P < 0{\cdot}05$, Fig. 5 and Tables 4 and 5). Plasma $[Lac^-]_a$ remained unchanged thereafter, whereas $[Lac^-]_v$ declined slightly by 10 min post-exercise ($P < 0{\cdot}05$). The negative $[Lac^-]_{a-v}$ indicated a net release of $Lac^-$ into plasma at each time point ($P < 0{\cdot}05$, Fig. 5). The widest $[Lac^-]_{a-v}$ difference occurred during exercise ($P < 0{\cdot}05$): $-4{\cdot}5 \pm 1{\cdot}3$ mmol $l^{-1}$ before and $-5{\cdot}4 \pm 2{\cdot}5$ mmol $l^{-1}$ after training (n.s.). $[Lac^-]_a$ was greater at all experimental time points after training, by a mean difference of 1·0 mmol $l^{-1}$ ($P < 0{\cdot}05$), but no significant training effects were found for either $[Lac^-]_v$ or $[Lac^-]_{a-v}$.

**Whole blood.** Both $[Lac^-]_{wb,a}$ and $[Lac^-]_{wb,v}$ increased sharply during exercise and early recovery ($P < 0{\cdot}05$), peaking at 5 min post-exercise (Table 6). Net muscle $Lac^-$ release was found at all time points, with the widest $[Lac^-]_{wb,a-v}$ occurring during exercise ($P < 0{\cdot}05$): $-3{\cdot}5 \pm 1{\cdot}0$ mmol $l^{-1}$ before and $-4{\cdot}1 \pm 1{\cdot}9$ mmol $l^{-1}$ after training (n.s., Table 6). No training effects were found for $[Lac^-]_{wb,a}$, $[Lac^-]_{wb,v}$ or $[Lac^-]_{wb,a-v}$.

### Chloride

**Plasma.** Both $[Cl^-]_a$ and $[Cl^-]_v$ rose during exercise ($P < 0{\cdot}05$) and returned to resting values by 5 and 1 min post-exercise, respectively (Fig. 6 and Tables 4 and 5); $[Cl^-]_v$ fell below resting values at 5 and 10 min post-exercise ($P < 0{\cdot}05$, Table 5). Plasma $[Cl^-]_{a-v}$ became more positive during exercise and at 1 min post-exercise ($P < 0{\cdot}05$), indicating a net $Cl^-$ movement out of the plasma (Fig. 6). No significant training effects were found for $[Cl^-]_a$, $[Cl^-]_v$ or $[Cl^-]_{a-v}$. A significant interaction between time and training was found for $[Cl^-]_{a-v}$, with a tendency for a reduced net $Cl^-$ loss from plasma after training at 10 min post-exercise ($P < 0{\cdot}08$).

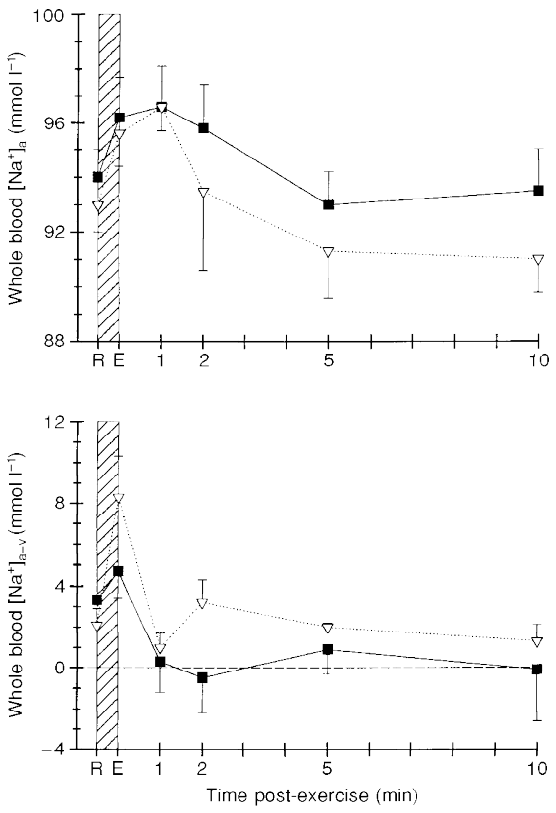


**Figure 4. Arterial whole blood $[Na^+]$ (top) and arteriovenous whole blood $[Na^+]$ differences (bottom)**
Measurements were obtained at rest (R), during and after maximal exercise (E), before (■) and after (▽) sprint training. Sample size and corrections as in Fig. 2. Dashed line indicates zero arteriovenous difference.

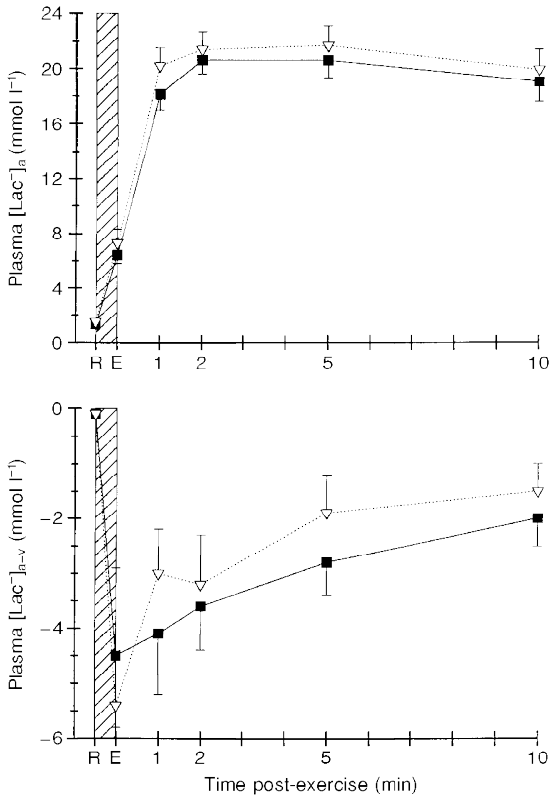


**Figure 5. Arterial plasma [Lac$^-$] (top) and arteriovenous plasma [Lac$^-$] differences (bottom)**

Measurements were obtained at rest (R), during and after maximal exercise (E), before (■) and after (▽) sprint training. Sample size and corrections as in Fig. 1.

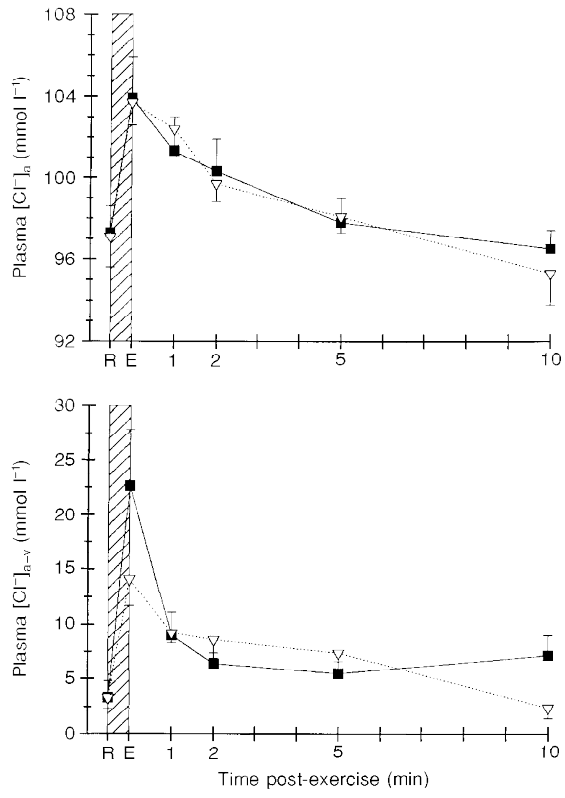


**Figure 6. Arterial plasma [Cl$^-$] (top) and arteriovenous plasma [Cl$^-$] differences (bottom)**

Measurements were obtained at rest (R), during and after maximal exercise (E), before (■) and after (▽) sprint training. Sample size and corrections as in Fig. 1.

**Whole blood.** $[Cl^-]_{wb,a}$ rose modestly during exercise and remained elevated throughout recovery ($P < 0{\cdot}05$, Table 6). A sharper rise was seen in $[Cl^-]_{wb,v}$ during exercise ($P < 0{\cdot}05$, Table 6). Both $[Cl^-]_{wb,a}$ and $[Cl^-]_{wb,v}$ were increased after training, by mean differences of 4·8 and 4·9 mmol $l^{-1}$, respectively ($P < 0{\cdot}05$). A positive $[Cl^-]_{wb,a-v}$ was found at all time points, suggesting net $Cl^-$ uptake across the leg; however, $[Cl^-]_{wb,a-v}$ did not differ significantly between observation time points and was not modified by training.

## Plasma acid–base variables

### Strong ion difference

Plasma $[SID]_a$ fell during exercise and recovery and remained depressed at 10 min post-exercise ($P < 0{\cdot}05$, Fig. 7). In contrast, plasma $[SID]_v$ increased during exercise, declined sharply to subresting levels by 1 min and remained depressed at 10 min post-exercise ($P < 0{\cdot}05$, Table 5). Compared with resting values, plasma $[SID]_{a-v}$ was more negative during exercise and at 1 min post-exercise ($P < 0{\cdot}05$, Fig. 7). After training, both $[SID]_a$ and $[SID]_v$ were lower at all experimental times after training, by mean differences of 2·7 and 1·8 mmol $l^{-1}$, respectively ($P < 0{\cdot}05$, Tables 4 and 5). There was a tendency for $[SID]_{a-v}$ to be less post-training during exercise ($P = 0{\cdot}06$), suggesting a reduced rise in [SID] across the leg during exercise.

### Carbon dioxide tension

Plasma arterial $P_{CO_2}$ ($P_{a,CO_2}$) fell during exercise and recovery, reaching a nadir at 5 min post-exercise ($P < 0{\cdot}05$, Table 4). In contrast, plasma venous $P_{CO_2}$ ($P_{v,CO_2}$) rose sharply to peak during exercise ($P < 0{\cdot}05$, Table 5), before declining to resting values at 5 min post-exercise. Significant training effects were found for $P_{a,CO_2}$ and $P_{v,CO_2}$, both being lower across all times after training, by mean differences of 1·7 and 4·5 mmHg, respectively ($P < 0{\cdot}05$).

### Plasma protein weak acid concentration

Plasma $[A_{tot}]_a$ and $[A_{tot}]_v$ were both elevated in exercise and recovery ($P < 0{\cdot}05$). After training, $[A_{tot}]_a$ was unchanged ($P = 0{\cdot}10$), but $[A_{tot}]_v$ and $[A_{tot}]_{a-v}$ were both reduced after training, by mean differences of 0·4 and 0·2 mmol $l^{-1}$, respectively ($P < 0{\cdot}05$, Tables 4 and 5).

### Hydrogen ion concentration

Plasma $[H^+]_a$ was unchanged during exercise but increased in recovery, peaking at 65·8 nmol $l^{-1}$ at 5 min post-exercise ($P < 0{\cdot}05$, Fig. 8 and Table 4). In contrast, $[H^+]_v$ increased during exercise ($P < 0{\cdot}05$) and reached a peak of 107·3 nmol $l^{-1}$ at 1 min post-exercise ($P < 0{\cdot}05$, Table 5). Plasma $[H^+]_{a-v}$ became increasingly negative during exercise and at 1 min post-exercise, remaining less than at rest at all times in recovery ($P < 0{\cdot}05$, Fig. 8). After training, $[H^+]_a$ was greater by a mean difference of 2·2 nmol $l^{-1}$ ($P < 0{\cdot}05$). No significant training effects were found for either $[H^+]_v$ or $[H^+]_{a-v}$.

### Bicarbonate concentration

Plasma $[HCO_3^-]_a$ fell sharply but $[HCO_3^-]_v$ increased during exercise; both declined in the first 5 min of recovery ($P < 0{\cdot}05$, Tables 4 and 5). Plasma $[HCO_3^-]_{a-v}$ was negative at all times, became more negative during exercise and returned to resting values at 10 min post-exercise ($P < 0{\cdot}05$).

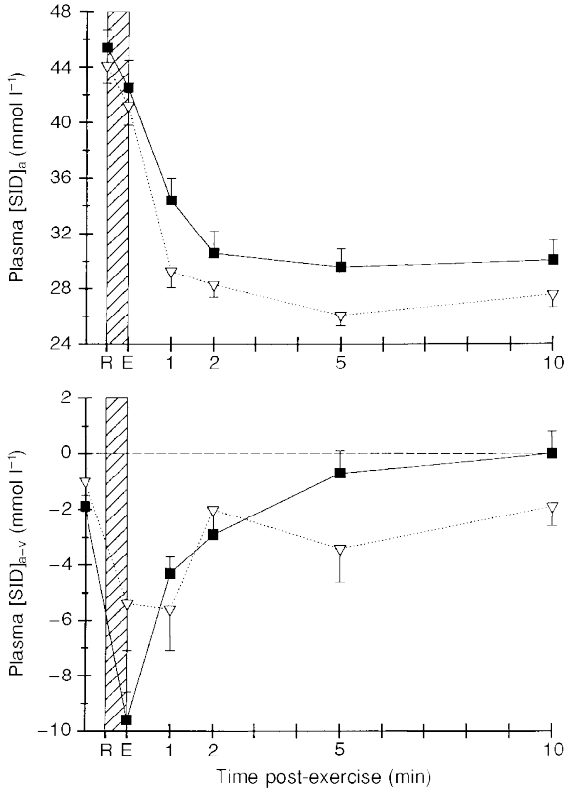


**Figure 7. Arterial plasma [SID] (top) and arteriovenous plasma [SID] difference (uncorrected; bottom)**

Measurements were obtained at rest (R), during and after maximal exercise (E), before (■) and after (▽) sprint training. Sample size as in Fig. 1. Dashed line indicates zero arteriovenous difference.

After training, $[HCO_3^-]_a$ and $[HCO_3^-]_v$ were both lower across all times, by mean differences of 1·2 and 1·6 mmol $l^{-1}$, respectively ($P < 0·05$). No significant training effect was found for $[HCO_3^-]_{a-v}$.

## DISCUSSION

Sprint training, comprising repeated 30 s bouts of maximal cycling exercise, with only brief recovery periods, enhanced sprint exercise performance; cumulative work output was increased and fatigue reduced, associated with altered muscle and vascular strong ion regulation. After training, arterio-femoral venous differences in whole blood ions indicated greater $Na^+$ uptake by the exercising leg; arterial and venous plasma $[K^+]$ and $[Na^+]$ were lower, whilst arterial plasma $[Lac^-]$ and $[H^+]$ were both higher.

### Training increased sprint exercise performance

Sprint training increased work output (by 13%) and reduced fatigue during the sprint bout, as evidenced by the 14% lower fatigue index after training. The magnitude of increase in work output after training was consistent with other sprint training studies (Sharp *et al.* 1986; Nevill, Boobis, Brooks & Williams, 1989; McKenna *et al.* 1993; Stathis, Febbraio, Carey & Snow, 1994). Although peak power output in the single exercise bout used in the present study was unchanged by training, a small but significant increase in peak power was found after training (4% increase, $P < 0·05$) when data from the two separate sprint tests completed before and after training were combined; these data are presented and discussed in the accompanying paper (McKenna *et al.* 1997). The reduced fatigability after training may be due to less fatigue in the central nervous system or in the contracting muscles, or both. Although it was not possible in this study to determine the relative contributions of central and peripheral factors to fatigue, central fatigue can probably account for only a small proportion of the fatigue during this type of exercise (Beelen, Sargeant, Jones & de Ruiter, 1995). Thus, the reduced fatigability found after training can most probably be explained by reduced local fatigue in the contracting muscles. In the absence of altered regulation, the enhanced 30 s work output after training might then be expected to induce greater fluid and ion shifts with exercise.

### Plasma volume and fluid shifts during intense exercise

Resting plasma and blood volumes were not measured in the present study, but $Hct_a$ was increased, whilst resting $[Hb]_a$ and $[PPr^-]_a$ were unchanged after sprint training. In a separate study, neither plasma nor blood volumes were altered following an identical sprint training programme (Haukka *et al.* 1994). Therefore it is quite unlikely that any changes in resting vascular volumes occurred after training in the present study. These findings indicate that sprint training did not induce the hypervolaemia usually associated with aerobic training, comprising increased PV and unchanged red cell mass (Convertino, 1991).

Increased fluid shifts during exercise might be expected after training owing to the greater 30 s work production, but all measured fluid shifts with exercise were unchanged

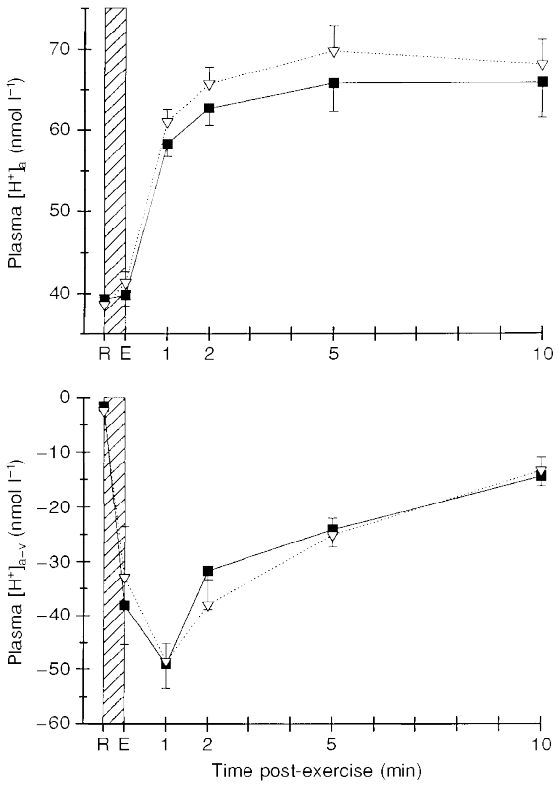


**Figure 8. Arterial plasma $[H^+]$ (top) and arteriovenous plasma $[H^+]$ difference (bottom)**

Measurements were obtained at rest (R), during and after maximal exercise (E), before (■) and after (▽) sprint training. Sample size as in Fig. 1.

after sprint training in the present study. Greater absolute fluid shifts during exercise occurred after five consecutive days of intermittent training, owing to an 11 % increase in resting PV (Green, Hughson, Thomson & Sharratt, 1987). It is likely that these early training adaptations do not persist for longer term training (Haukka *et al.* 1994). Since resting vascular volumes were most probably unchanged (Haukka *et al.* 1994) and the relative fluid shifts with exercise were unaffected by training in the present study, any training-induced differences in plasma or whole blood ion concentrations will reflect differences in net ion release into, or clearance from, plasma or blood.

## Improved potassium regulation

The effects of training on $[K^+]$ regulation and fatigue are of particular interest since increases in interstitial $[K^+]$ and reductions in muscle $[K^+]$ have been linked with muscular fatigue, through impairment of membrane excitation, as well as an increased intracellular $[H^+]$ (Sjøgaard *et al.* 1985; Lindinger & Heigenhauser, 1991). The changes in arterial and venous plasma $[K^+]$ with intense exercise were consistent with findings from other studies (Medbø & Sejersted, 1985, 1990; Kowalchuk *et al.* 1988; Lindinger *et al.* 1992; Vøllestad *et al.* 1994), and were unchanged after training despite the increased work output, suggesting an improved extracellular $K^+$ clearance after training. Consistent with this interpretation were the 0·2–0·3 mmol $l^{-1}$ lower values of arterial and venous plasma $[K^+]$ across all observation times after training. Medbø & Sejersted (1994) reported that the peak femoral venous $[K^+]$ during intense treadmill exercise did not differ between endurance and sprint athletes, but this finding is difficult to interpret, since both groups of athletes probably had improved mechanisms for $K^+$ regulation (Green, Chin, Ball-Burnett & Ranney, 1993; McKenna *et al.* 1993). However, the sprinters had a greater capacity to clear $K^+$, with a more rapid post-exercise decline in plasma $[K^+]$ (Medbø & Sejersted, 1994). Calculations based on the data of Medbø & Sejersted (1994) indicated a lower $\Delta[K^+]$: work ratio for the sprint athletes, consistent with lower ratios found during intermittent exercise after sprint training (McKenna *et al.* 1993). These $\Delta[K^+]$: work ratios were unchanged after training in the present study owing to the lower resting plasma $[K^+]$ after training.

One of the advantages of the present study was that plasma and whole blood arteriovenous $[K^+]$ differences were measured, enabling interpretation of the effects of training on ionic fluxes between muscle and whole blood. Previous intense exercise studies have reported wide negative arteriovenous plasma $[K^+]$ differences during exercise and have interpreted these as net $K^+$ release from contracting muscle (Medbø & Sejersted, 1990; Juel, Bangsbo, Graham & Saltin, 1990; Rollett, Strange, Sjøgaard, Kiens & Saltin, 1990; Vøllestad *et al.* 1994). In the present study, a similar wide negative arteriovenous plasma $[K^+]$ difference was found during exercise, when the data were uncorrected for fluid shifts. However, since the net water and $K^+$ fluxes are in opposite directions across the muscle membrane during exercise (Watson, Garner & Ward, 1993), it is appropriate to correct the arteriovenous $[K^+]$ differences for the corresponding arteriovenous decline in plasma volume. After this correction, there was only a very small net $K^+$ release of 0·1–0·3 mmol $l^{-1}$ into plasma during the final seconds of exercise. In contrast to findings in plasma, the whole blood arteriovenous $[K^+]$ difference was positive during the final seconds of exercise. Together with the large increase in arterial plasma $[K^+]$, these data suggest that a net loss of $K^+$ occurred from contracting muscle early in exercise, but in the final seconds of exercise, when power output had declined by 40–60 %, this had reversed to a net muscle $K^+$ uptake. The $K^+$ uptake during exercise was not significantly different from that at other time points, but this is not surprising given the small sample size during exercise ($n = 3$) and measurement variability in the whole blood $[K^+]$ and [Hb] determinations. However, the arteriovenous whole blood $[K^+]$ differences were higher during exercise than at rest in each subject; also, the expected large increase in blood flow during exercise would greatly magnify the effects of any difference. $K^+$ uptake across the leg during the final seconds of sprint exercise is also consistent with the rapid uptake reported immediately following intense exercise (Lindinger *et al.* 1992) and was also greater after training in each subject. Taken with the probable increase in muscle blood flow during maximal sprint exercise after training (see McKenna *et al.* 1997), these findings suggest a substantial increase in exercise and post-exercise $K^+$ uptake after training. The reduced resting and recovery plasma $[K^+]$ and greater muscle $K^+$ uptake during exercise after sprint training are consistent with the increased $Na^+$,$K^+$-ATPase concentration in human skeletal muscle following an identical training programme (McKenna *et al.* 1993). It is also possible that an increased total $Na^+$,$K^+$-ATPase content, or $Na^+$,$K^+$-ATPase activation during exercise, in skeletal muscle and in other tissues may contribute to these findings (McKenna, 1995).

## Enhanced muscle sodium uptake

Net $Na^+$ shifts from arterial plasma occurred during and following exercise, consistent with earlier studies (Medbø & Sejersted, 1985), with the major $Na^+$ shift occurring during exercise as indicated by the greater $[Na^+]_{a-v}$ and the expected increase in muscle blood flow. We can conclude that $Na^+$ was taken up by the exercising leg muscle since the whole blood $[Na^+]_{a-v}$ was also increased during exercise. An interesting finding was the greater leg muscle $Na^+$ uptake after sprint training, particularly during exercise (77 %, $n = 3$). Although most $Na^+$ remains in the interstitial space, any increase in muscle intracellular $[Na^+]$ would constrain the rise in intracellular $[H^+]$ with exercise, by minimizing the fall in intracellular [SID] (Lindinger & Heigenhauser, 1991). This would be consistent with the smaller rise in muscle $[H^+]$ found at fatigue after sprint training (Sharp *et al.* 1986; Troup, Metzger & Fitts, 1986) and unchanged $[H^+]$ found by Nevill

*et al.* (1989). Also, an increased intracellular $[Na^+]$ would further activate muscle $Na^+$,$K^+$-ATPase, reducing cellular $K^+$ loss during exercise. Both of these changes would be consistent with the improved muscle performance found after sprint training. Both plasma $[Na^+]_a$ and $[Na^+]_v$ were lower after sprint training, consistent with the decreased plasma $[Na^+]_a$ reported at rest and during exercise after endurance training in calves (Fosha-Dolezal & Fedde, 1988).

## Unchanged muscle glycolysis

It is unlikely that muscle glycolysis during exercise was substantially greater after sprint training in the present study, with little change in circulating $[Lac^-]$ found after training. Although arterial plasma $[Lac^-]$ was 1 mmol $l^{-1}$ greater, none of the arterial whole blood $[Lac^-]$, femoral venous plasma or whole blood $[Lac^-]$, nor the arterio-femoral plasma or whole blood $[Lac^-]$ differences were significantly changed after training. These findings are discussed in detail in the accompanying paper (McKenna *et al.* 1997).

## Plasma $[H^+]$ regulation

Medbø & Sejersted (1985) compared acid–base responses to maximal intermittent exercise between sprint and endurance athletes. They found that sprint athletes generated a greater plasma acidosis, attributed to a greater blood lactate concentration, with a lower $P_{a,CO_2}$. However, it is difficult to conclude whether these greater responses were induced by sprint training, because both groups of athletes were highly trained and a cross-sectional study design was employed. These authors found a discrepancy between the rise in $[Lac^-]$ and rise in $[H^+]$, leading them to postulate that $H^+$ ion efflux from muscle was more rapid than for $Lac^-$ ions. To interpret plasma acid–base changes with exercise we used a physicochemical approach (Stewart, 1981), in which $[H^+]$ is dependent upon the strong ions, defined as being fully dissociated in plasma ($Na^+$, $K^+$, $Cl^-$ and $Lac^-$) and acting through the strong ion difference; carbon dioxide, acting through $P_{CO_2}$; and weak acid buffers in plasma proteins ($[A_{tot}]$). All these variables were measured to quantify their relative contributions to changes in $[H^+]$. The acute responses to maximal exercise in arterial and femoral venous plasma [SID], $P_{CO_2}$ and $[A_{tot}]$ in this study were similar to those reported previously (Kowalchuk *et al.* 1988; Lindinger *et al.* 1992). The sharp post-exercise rise in $[H^+]_a$ to a peak of 66–70 nmol $l^{-1}$ (pH 7·18–7·16) could be almost entirely attributed to the decline in $[SID]_a$, due mainly to the rise in $[Lac^-]_a$, although increases in $[Cl^-]_a$ also contributed; increases in $[K^+]_a$ and $[Na^+]_a$ acted to attenuate the decrease in $[SID]_a$. In contrast to arterial changes, a large increase in femoral venous $[H^+]$ to 73–77 nmol $l^{-1}$ (pH 7·14) was found during exercise, attributable mainly to the rise in $P_{v,CO_2}$. Later, following exercise, both $P_{v,CO_2}$ and $[SID]_v$ declined, indicating a reduced relative contribution of $P_{v,CO_2}$ to the elevated post-exercise plasma $[H^+]_v$, whilst the relative contribution of $[SID]_v$ increased, such that all of the rise in $[H^+]_v$ at 10 min post-exercise was due to the reduction in $[SID]_v$. $[SID]_v$ increased during exercise because the rise in $[Na^+]_v$ and $[K^+]_v$ exceeded that of $[Lac^-]_v$ and $[Cl^-]_v$, whereas the subsequent post-exercise decline in $[SID]_v$ was due mainly to the increased $[Lac^-]_v$.

These findings lead to two important conclusions: first, that the marked acidosis in arterial and venous plasma following maximal exercise is due to changes that vary according to the time post-exercise and the site (arterial or venous); and second, that consideration of the strong ion difference makes it clear that several ions, in addition to $[Lac^-]$, contribute to or modulate the acidosis.

After sprint training, exercise total work output was increased, accompanied by greater post-exercise rises in plasma $[H^+]_a$ and $[Lac^-]_a$ and declines in $P_{a,CO_2}$ and $[HCO_3^-]_a$. The peak increase in plasma $[H^+]_a$ observed following exercise was 4·0 nmol $l^{-1}$ (6%) higher after training, due almost entirely to the mean 2·7 mmol $l^{-1}$ larger decline in plasma $[SID]_a$. Minimum $[SID]_a$ before and after training was 29·6 and 26·1 mmol $l^{-1}$, respectively. These values correspond well to estimates of arterial [SID] from data in sprint athletes (Medbø & Sejersted, 1985), who displayed a 3–6 mmol $l^{-1}$ greater fall in [SID] than endurance athletes. The lower $[SID]_a$ after training in the present study was due not only to an increased $[Lac^-]_a$ but also to changes in other strong ions, particularly $[Na^+]_a$, which was lower at rest, during and following exercise. The lower mean resting $[SID]_a$ (1·3 mmol $l^{-1}$) after training was accounted for by decreases in both resting $[Na^+]_a$ (1·0 mmol $l^{-1}$) and resting $[K^+]_a$ (0·4 mmol $l^{-1}$). The reduction in post-exercise $[SID]_a$ after training was due to mean decreases in plasma $[Na^+]_a$ (1·6 mmol $l^{-1}$) and $[K^+]_a$ (0·2 mmol $l^{-1}$), and an elevation in plasma $[Lac^-]_a$ of 1·0 mmol $l^{-1}$. Thus, the greater $[H^+]_a$ after training was dependent upon the smaller rise in plasma $[Na^+]_a$ with exercise, in addition to the greater rise in plasma $[Lac^-]_a$. The greater decline in $[SID]_a$ and greater rise in $[H^+]_a$ with maximal exercise after training were partly compensated for by a greater reduction in $P_{a,CO_2}$, consistent with an increased pulmonary ventilation with maximal exercise after sprint training (McKenna *et al.* 1997).

## Conclusion

Sprint training increased the capacity for muscular work and improved muscle strong ion regulation during exercise. Increased muscle $Na^+$ and $K^+$ uptake during exercise after training are consistent with a greater activation of the muscle $Na^+$–$K^+$ pump, reduced cellular $K^+$ loss and the observed reduced rate of fatigue. There was little evidence of enhanced glycolysis after training, suggesting that the major energetic adaptation after sprint training was enhanced aerobic metabolism. A consequence of the improved muscle strong ion regulation was a lower arterial plasma [SID] due to lower plasma $[K^+]$ and $[Na^+]$, and higher plasma $[Lac^-]$, resulting in a greater plasma acidosis. Thus, sprint training improved muscle ion regulation, associated with increased intense exercise performance, at the expense of a greater systemic acidosis.

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