EFFECT OF METABOLIC ALKALOSIS ON HIGH-INTENSITY PERFORMANCE IN SPRINT AND ENDURANCE ATHLETES

Simon Green*, Darrin Street*, Michael McNeiry**

* School of Human Movement Studies, Queensland University of Technology, Brisbane, Australia.
** Department of Human Movement Studies, The University of Queensland, Brisbane, Australia.

INTRODUCTION

The ergogenic effect associated with ingesting an alkali prior to high-intensity performance is probably mediated by an improvement in muscle acid-base and electrolyte regulation (Lindinger et al., 1990). However, ergogenesis is not always observed following alkali ingestion, and there is evidence that its presence depends on both the alkali dose and exercise duration (e.g., McNaughton & Cedaro, 1992). Given that the contractile function of fast-twitch fibres is impaired by acidosis to a greater extent than slow twitch fibres (Donaldson, 1983), then the size of the ergogenic effect might also depend upon the relative contribution of the recruitment of fast-twitch muscle to performance. On this basis, this study tested the hypothesis that alkalosis would improve performance to a greater extent in sprint athletes compared with endurance athletes.

METHODS

Four sprint-pursuit cyclists and one sprint runner (S: age = 21.6 ± 4.7 y; weight = 75.3 ± 8.6 kg) and four endurance cyclists (E: age = 21.0 ± 3.6 y; weight = 70.9 ± 12.5 kg) were tested on three separate occasions. On the initial occasion the peak power and total work done (i.e. anaerobic work capacity) during an all-out 30 s cycle test, as well as the ventilatory threshold (Tvent) during a ramp (0.25 Ws⁻¹) test, were measured. During the next two sessions, each subject cycled at a submaximal intensity (i.e. Tvent + 40 W) to exhaustion 30 min after either a placebo (P: 179 mg kg⁻¹ of NaCl) or sodium citrate (SC: 300 mg kg⁻¹) were orally administered over 60 min. The treatments were assigned in a randomised and double-blind manner. Arterialised-venous blood samples were taken prior to and following ingestion, as well as during min 2, 3, 6 and immediately following exercise. Oxygen consumption during exercise was measured using the Douglas bag method at min 2, 3, 6 and exhaustion, and VO2 drift was determined as the difference in VO2 between the sixth and third minutes.

RESULTS

Peak power and anaerobic work capacity (AWC) were greater (p < 0.05) in S (13.1 ± 1.3 W kg⁻¹; 312 ± 28 J kg⁻¹) compared with E (10.0 ± 0.8 W; 272 ± 15 J kg⁻¹). Alkalosis did not increase (p > 0.05) time to exhaustion (TE) in S or E, despite that an 8% increase (P = 0.11; two-tailed) occurred in S (Table 1). There was no interaction between group and treatment for TE, and the change in TE was not correlated (p > 0.05) with peak power or AWC. In contrast, the VU2 drift was larger in S than E (p = 0.02) and was correlated with peak power (r = 0.71) and AWC (r = 0.78). There was a group-by-treatment interaction (p = 0.02) for VU2 drift that was a function of both a reduction in VO2 drift under alkalosis in S (p = 0.06) and an opposing increase (p > 0.05) in E (Table 1). Peak VO2 was not different between either P and SC (5.09 ± 0.45 and 5.13 ± 0.60 /min⁻¹) or groups S and H (4.98 ± 0.35 and 5.28 ± 0.66 /min⁻¹). The change in plasma [La⁺] from pre-exercise to exhaustion was higher (p = 0.05) for S and E under...
SC compared with P (16.6 ± 3.3 vs 13.5 ± 3.0 mmolL⁻¹). There was no correlation (p > 0.05) between either the change in plasma [La⁺] between min 6 and 3 and VO₂ drift, or the alkalosis-induced change in either plasma [La⁺] between min 6 and 3 or between pre-exercise and exhaustion and change in VOi drift (r = 0.52 or 0.26). There was, however, a stronger correlation (p = 0.06 to 0.1) between the change in plasma [K⁺] over min 3 to 6, or from pre-exercise to exhaustion, and the change in VC⁰⁻² drift (r = 0.62 or 0.67). Alkalosis decreased (p < 0.05) the peak plasma [K⁺] (5.7 ± 0.3 mmol L⁻¹) compared with placebo (6.1 ± 0.3 mmol L⁻¹). There was also a group-by-treatment interaction (p = 0.02) for the change in plasma [K⁺]: alkalosis decreased the exercise-induced increase in plasma [K⁺] in S (1.43 ± 0.35 vs 1.81 ± 0.22 mmolL⁻¹), whereas this variable was increased in E (2.03 ± 0.35 vs 1.67 ± 0.16 mmolL⁻¹). There was also a moderate, but non-significant, correlation (r = -0.56; p = 0.15) between the change in performance and change in plasma [K⁺] induced by alkalosis.

Table 1. Physiological and performance data on five sprint (S) and four endurance (E) athletes prior to and during exhaustive cycling at T

<table>
<thead>
<tr>
<th>Group</th>
<th>Treatment</th>
<th>Pre-ex. pH</th>
<th>TE(s)</th>
<th>VO₂ drift (m/)</th>
<th>Post-ex blood [La]</th>
</tr>
</thead>
<tbody>
<tr>
<td>S</td>
<td>P</td>
<td>7.40 ± 0.03</td>
<td>500 ±71</td>
<td>576 ± 90**</td>
<td>14.9 ± 3.4</td>
</tr>
<tr>
<td></td>
<td>SC</td>
<td>7.47 ± 0.05*</td>
<td>542 ± 99</td>
<td>448 ± 169</td>
<td>16.8 ± 3.6</td>
</tr>
<tr>
<td>E</td>
<td>P</td>
<td>7.39 ± 0.02</td>
<td>433 ± 105</td>
<td>217 ± 12</td>
<td>14.9 ±1.71</td>
</tr>
<tr>
<td></td>
<td>SC</td>
<td>7.50 ± 0.08*</td>
<td>440 ± 132</td>
<td>450 ± 145</td>
<td>19.8 ± 2.9</td>
</tr>
</tbody>
</table>

* different (p < 0.05) from placebo; ** different from E for the same condition.

DISCUSSION

The present findings do not support the hypothesis that the effect of metabolic alkalosis on high-intensity performance is greater in sprint as compared with endurance athletes. This was despite the group-by-treatment interactions for both VO₂ drift and plasma [K⁺]. The relationships observed between alkalosis and these two variables supports other evidence (Lindinger et al., 1990) that alkalosis improves the intracellular regulation of K⁺, resulting in lower plasma [K⁺], and suggests that K⁺ loss from muscle might also influence the slow component of VC⁰⁻² observed at higher exercise intensities.

REFERENCES

