THE EFFECT OF SODIUM BICARBONATE INGESTION ON THERMOREGULATORY FUNCTION DURING CYCLE ERGOMETRY

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Typically sodium bicarbonate ingestion has been employed to buffer the large reductions in muscle and blood pH induced by short-duration high-intensity exercise (Jones *et al.*, 1977; Sutton *et al.*, 1981). Recently, Lindinger *et al.* (1999) reported an expanded extracellular fluid (ECF), and subsequently greater PV (plasma volume), following sodium bicarbonate ingestion and suggested potential cardiovascular and thermoregulatory benefits from such ingestion. However, while the large dose of sodium resulted in greater ECF retention the elevated plasma sodium concentration could have detrimental effects on heat loss mechanisms (Greenleaf & Brock, 1980). This investigation determined whether sodium bicarbonate ingestion affected cardiovascular and thermoregulatory function during prolonged exercise. Ten male subjects exercise for 90 min at 62.5 ±1.3% O_{2peak} in an environment of 20.6 ±0.1°C and 50.0 ± 0.0% relative humidity. Subjects ingested either $NaHCO_3^-$ (0.3 $g \cdot kg^{-1}$ body mass) or empty capsules (placebo) over a 120-min period 60 min prior to exercise, with trials being presented in a blind balanced order 7 days apart. Arterialised-venous blood samples were drawn prior to ingestion, prior to exercise and after 15, 30, 60 and 90 min of exercise. Blood acid-base status was determined using an automated blood gas meter (ABL5, Radiometer). Changes in PV were estimated by measurement of haematocrit and haemoglobin. Serum $[Na^+]$ and $[K^+]$ were determined by flame photometry, while serum $[Cl^-]$ was assessed using the thiocyanate technique. Heart rate (HR; Polar Vantage NV, Finland), skin temperature and rectal temperature (T_{re}) were monitored continuously during exercise, and sweat loss was derived from the change in body mass (uncorrected for respiratory water loss and loss resulting from CO_2-O_2 exchange). Treatment effects were determined using a two-way ANOVA, with alpha set at 0.05. PV was expanded after $NaHCO_3^-$ ingestion (5.3 ±1.0%; $P<0.05$), with this greater PV being maintained throughout exercise. Resting serum $[Na^+]$ was greater after the $NaHCO_3^-$ treatment (140.2 ±1.1 versus 143.2 ±1.0 $mmol \cdot l^{-1}$; $P<0.05$), with this elevation also being maintained during the exercise bout. HR was unaffected by the $NaHCO_3^-$ ingestion (148 ±3 versus 150 ±4 $beats \cdot min^{-1}$; $P>0.05$) as was T_{re} (37.8 ±0.1°C versus 37.7 ±0.1°C; $P>0.05$). While sweat loss was suppressed in eight of the ten subjects following $NaHCO_3^-$ ingestion, significance was not reach (0.68 ±0.03 versus 0.71 ±0.04 $l \cdot h^{-1}$; $P=0.07$). The $NaHCO_3^-$ ingestion consequently elevated PV both at rest and during exercise, however this expansion did not influence exercising HR. Similarly the greater serum $[Na^+]$ did not augment the T_{re} elevation, however it seemed that sweat rate was marginally reduced. Therefore it would seem that the small change in sweat rate was not sufficient to impede heat loss from the body and the magnitude of PV expansion was inadequate to facilitate an attenuation of the HR response.

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