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Cardiac electrophysiology during progressive and controlled dehydration: inferences from ECG analysis during steady-state exercise and recovery

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Abstract

When fluid intake is insufficient to match sweat losses, dehydration develops. It is well established that dehydration impacts unfavourably upon cardiovascular function, including cardiac output and peripheral blood flow (Gonzalez-Alonso et al., 1998). However, the limitations of cardiac electrophysiology in the dehydrated state are not known. In light of possible electrolyte imbalances, particularly when water deficit moves towards 7% of total body mass, it is worth considering the possibility of adverse conduction changes, as reflected within the electrocardiogram (ECG), may accompany electrolyte loss. In addition, the ECG can also be employed to investigate other cardiac limitations, such as myocardial ischaemia. Thus, in this project, set amongst a large dehydration study, basic cardiac conduction measures, and indicators of myocardial ischaemia (ST segment) were evaluated across a wide range of dehydration levels (1-7% water deficit).

Keywords

dehydration, inferences, ecg, analysis, steady, state, exercise, recovery, cardiac, during, electrophysiology, progressive, controlled

Disciplines

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CARDIAC ELECTROPHYSIOLOGY DURING PROGRESSIVE AND CONTROLLED DEHYDRATION: INFERENCES FROM ECG ANALYSIS DURING STEADY-STATE EXERCISE AND RECOVERY

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INTRODUCTION

When fluid intake is insufficient to match sweat losses, dehydration develops. It is well established that dehydration impacts unfavourably upon cardiovascular function, including cardiac output and peripheral blood flow (Gonzalez-Alonso *et al.,* 1998). However, the limitations of cardiac electrophysiology in the dehydrated state are not known. In light of possible electrolyte imbalances, particularly when water deficit moves towards 7% of total body mass, it is worth considering the possibility of adverse conduction changes, as reflected within the electrocardiogram (ECG), may accompany electrolyte loss. In addition, the ECG can also be employed to investigate other cardiac limitations, such as myocardial ischaemia. Thus, in this project, set amongst a large dehydration study, basic cardiac conduction measures, and indicators of myocardial ischaemia (ST segment) were evaluated across a wide range of dehydration levels (1-7% water deficit).

METHODS

Nine physically active men were progressively dehydrated to a 7% water deficit. This was achieved using the controlled-hyperthermia (isothermal clamping) technique in hot (35.6^{o}C (±0.4)) and humid conditions (56.0% relative humidity) by attempting to elevate and clamp the core temperature of each subject at approximately 38.5^{o}C (Taylor *et al*., 2009). The black globe temperature averaged 35.6^{o}C (±0.3) and wind velocity was <0.05 m.s^{-1}. Subjects wore only shorts and running shoes. Tests were conducted at approximately the same time of day for each person, using fully-hydrated subjects. The pre-experimental mean urine specific gravity across all trials was 1.006 (SD 0.005).

Prior to commencing heat exposure, data were collected while subjects rested (seated) in a thermoneutral laboratory (baseline). Hydration state was determined from the change in body mass, with data collected at the attainment of each 1% dehydration target. Just prior to reaching the target, steady-state cycling was performed at 40% of the maximal work rate for 5 min, followed by a 2-min recovery. Subjects were connected to a 12-lead electrocardiograph (Norav 1200, Norav Medical, Yokneam, Israel) during both of these phases. The ECG patterns were analysed using Norav 1200 software. Specifically, R-R time (ms), heart rate (b.min^{-1}), QRS interval (ms), QT interval (ms) and ST segment displacement (mm) were calculated from Lead II. Exercise data were analysed between the fourth and fifth minutes. Recovery data were analysed 2 min after ceasing exercise. Venous blood samples were collected prior to each trial, and again on reaching each dehydration target, but while subjects were in recovery, and with no change in posture. These samples were immediately analysed (ABL77 blood gas / electrolyte, Radiometer Pacific, Melbourne, Australia). A wide range of other physiological data were collected prior to each trial, and again on reaching each dehydration target. Some of these observations are

reported in other communications to this meeting (Machado-Moreira *et al*. 2009, Taylor *et al.*, 2009).

RESULTS

The pre-experimental standardisation procedures ensured that every subject presented in a euhydrated state (urine specific gravity: <1.021). Baseline data confirmed that the cardiovascular, thermal and body mass status of all subjects were successfully standardised at a physiologically normal basal state. The dehydration targets were secured with precision, and were achieved to within 0.06%.

Progressive dehydration sequentially increased haematocrit (baseline: 45.9% (±1.1); 7%: 50.4% (±1.5)), plasma potassium concentration (baseline: 3.9 mmol.L^{-1} (±0.1); 7%: 4.9 mmol.L^{-1} (±0.1)) and plasma sodium concentration (baseline: 138 mmol.L^{-1} (±0.4); 7%: 142 mmol.L^{-1} (±0.8); all P<0.05). Heart rate during both the exercise (1%: 135 b.min^{-1} (±4); 7%: 160 b.min^{-1} (±3)) and recovery conditions (1%: 109 b.min^{-1} (±6); 7%: 134 b.min^{-1} (±3)) increased by 25 b.min^{-1} at the 7% dehydration target (P<0.05).

There was no change in ventricular depolarisation (QRS) time during exercise (1%: 101 ms (±4); 7%: 100 ms (±4)) or recovery (1%: 95 ms (±7); 7%: 86 ms (±5)). Equally, ventricular repolarisation (QT) time was not significantly reduced. Exercising ST segment was elevated with successive dehydration targets to 5%, and then plateaued through to 7% (Figure 1). An overall elevation of 1.1 mm from rest was observed from 5-7% water deficit. During recovery, the ST segment remained elevated from 1-3% dehydration. However, from 4-7% there was a trend for it to return towards baseline (4%: 1.81 mm (±0.22); 5%: 1.67 mm (±0.29); 6%: 1.57 mm (±0.32); 7%: 1.43 mm (±0.4)).

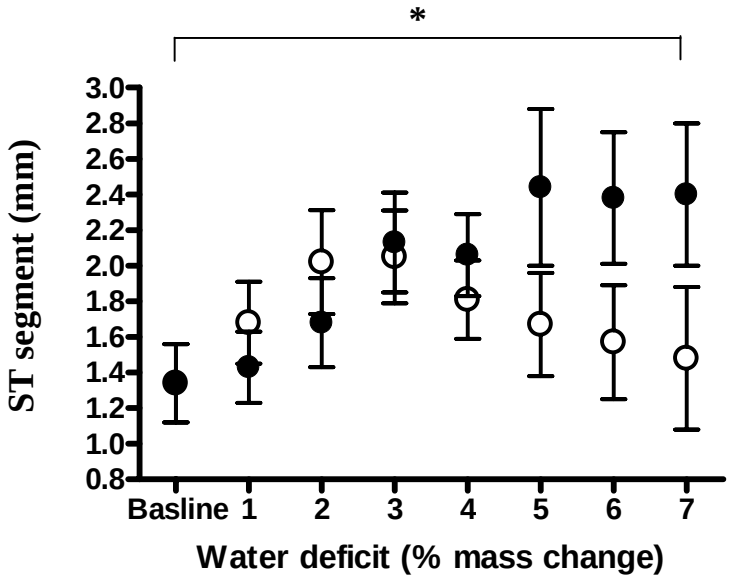

Figure 1: Exercising (●) and recovery (○) ST segment displacement (mm) measured at baseline, and then at each dehydration target. Data are means with standard errors of the means. * exercise ST segment differed from baseline (P<0.05).

CONCLUSIONS

The current project provided an evaluation of basic cardiac electrophysiology in the context of controlled and progressive dehydration to a 7% water deficit. Despite significantly increased blood sodium and potassium concentrations, there was no evidence that cardiac electrophysiology was impaired. In addition, there was also no clinical evidence of limited myocardial perfusion, with resulting myocardial ischaemia, since there was only a minor ST segment elevation across 1-7% water deficit.

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