ORIGINAL ARTICLE

Serum electrolyte concentrations and hydration status are not associated with exercise associated muscle cramping (EAMC) in distance runners

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Accepted 8 July 2003
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Objectives: To determine whether acute exercise associated muscle cramping (EAMC) in distance runners is related to changes in serum electrolyte concentrations and hydration status.
Methods: A cohort of 72 runners participating in an ultra-distance road race was followed up for the development of EAMC. All subjects were weighed before and immediately after the race. Blood samples were taken before the race, immediately after the race, and 60 minutes after the race. Blood samples were analysed for glucose, protein, sodium, potassium, calcium, and magnesium concentrations, as well as serum osmolality, haemoglobin, and packed cell volume. Runners who suffered from acute EAMC during the race formed the cramp group (cramp, n = 21), while runners with no history of EAMC during the race formed the control group (control, n = 22).
Results: There were no significant differences between the two groups for pre-race or post-race body weight, per cent change in body weight, blood volume, plasma volume, or red cell volume. The immediate post-race serum sodium concentration was significantly lower (p = 0.004) in the cramp group (mean (SD), 139.8 (3.1) mmol/l) than in the control group (142.3 (2.1) mmol/l). The immediate post-race serum magnesium concentration was significantly higher (p = 0.03) in the cramp group (0.73 (0.06) mmol/l) than in the control group (0.67 (0.08) mmol/l).
Conclusions: There are no clinically significant alterations in serum electrolyte concentrations and there is no alteration in hydration status in runners with EAMC participating in an ultra-distance race.

E xercise associated muscle cramping (EAMC) can be defined as "a painful, involuntary contraction of skeletal muscle that occurs during or immediately after exercise."[1] Numerous studies have documented the serum electrolyte changes that occur with endurance exercise.[2–6] Serum electrolyte and fluid disturbances have been associated with the development of muscle cramps in certain clinical conditions.[7–13] It is therefore often assumed that EAMC is also caused by fluid imbalances, in particular dehydration, and serum electrolyte abnormalities.[14–17] This assumption is common,[15 16 18–20] despite the fact that very few studies have examined the relation between changes in serum electrolyte concentrations and the development of EAMC. There are no well conducted studies that have documented a relation between dehydration and muscle cramping in athletes.

A prospective study of marathon runners showed no association between EAMC and plasma volume changes or changes in serum sodium, potassium, calcium, phosphate, bicarbonate, urea, or creatinine concentrations.[21] Two limitations of this study were that serum magnesium concentrations were not determined and that serum electrolyte concentrations were not documented in the recovery period after the race. If abnormal serum electrolyte concentrations return to normal during recovery, and this correlates with clinical recovery from EAMC, it would support the hypothesis that abnormal serum electrolyte concentrations are related to EAMC.

Magnesium has been shown to play an important role in muscle and nerve function.[22 23] Magnesium is also often promoted, mostly by the industry, as the most important electrolyte supplement for preventing skeletal muscle cramping in athletes. Thus any study examining the relation between EAMC and changes in serum electrolyte concentrations should include measurement of serum magnesium.

The relation between serum electrolytes, dehydration, and EAMC has been reported in two other case series. There was no association between EAMC and serum potassium concentrations in cyclists who cycled for between two and a half and five hours, and no association between dehydration (per cent body weight loss) and uncontrolled muscle contraction in 44 triathletes.[24 25] Small subject numbers and the lack of any control groups were limitations of those case series.

There is clearly a lack of research documenting the association between EAMC, dehydration, and serum electrolyte status. The relation between EAMC and fluid and electrolytes during the recovery phase from acute cramping after exercise has also not been documented. This relation is particularly important to document because a disassociation between recovery from EAMC and changes in serum electrolyte concentrations would strongly support the hypothesis that changes in serum electrolytes are not related to the aetiology of EAMC.

Our aim in this study was therefore to document the relation between the development of EAMC in ultra-distance runners and concomitant changes in serum electrolyte concentrations and hydration status.

METHODS

Subjects

This prospective cohort study was conducted at the Two Oceans Ultra-marathon, a 56 km road race held annually in Cape Town, South Africa. The ethics and research committee of the University of Cape Town Medical School approved the study.

All runners who registered for the race were considered potential subjects. In a pre-race advertisement campaign in

the press and during registration for the race, a cohort of 72 runners was recruited. Forty five of these had a history of regularly suffering from EAMC and 27 had no previous experience of muscle cramping. A regular history of EAMC was defined as having a history of EAMC during at least two of every six consecutive races. Inclusion criteria were that all subjects should be male between the ages of 20 and 60, have a history of at least two years of active running as a registered runner, have no history of medical illness, and have no history of chronic or recent medicinal drug use.

A personal interview was conducted with each runner during pre-race registration by one of the investigators (JN). A questionnaire was first administered to the runners to elicit personal details, medical history, and any history of EAMC. A personal interview followed during which the information in the questionnaire was confirmed and all testing procedures were carefully explained. Written informed consent was obtained from each subject.

On race day, 21 runners from the initial subgroup of 45 runners with a history of cramping, who were all part of the cohort of 72 runners, suffered acute EAMC during or within 60 minutes of completing the race and formed the ''cramp'' group (n = 21). Data were collected from 22 of the 27 runners who had no past history of cramping but who were all part of the initial cohort of 72 runners. These runners formed the control group (n = 22) and they had no past history of EAMC and did not suffer any form of muscle cramping during or after the race. Twenty nine runners of the original cohort of 72 were excluded for various reasons: 16 failed to comply with the protocol during the race, nine had incomplete blood samples, and four drank significant amounts of fluid after completing the race but before arriving for immediate post-race blood sample collection.

Pre-race assessment

Pre-race weighing was conducted on the morning of the race. All runners were weighed at least 75 minutes before the start of the race (0600 hours). Body mass was measured in full race attire (running shorts, vest, and shoes). An electronic scale (Soehnle, Germany) was used for all body mass measurements. The scale was calibrated before weighing sessions. Subjects were instructed not to drink any fluids or consume any food between the weighing procedure and the start of the race.

Pre-race blood samples were collected from all subjects in the 75 minutes before the start of the race. Blood samples were taken from the antecubital veins, with subjects in the seated position. All the blood samples were clearly coded and stored for later analysis. Blood samples were analysed for haemoglobin concentration, packed cell volume, plasma proteins, serum sodium, serum potassium, serum total calcium, serum total magnesium, serum osmolality, and plasma glucose.

The temperature on race day ranged from 14.3°C to 23.8°C, with a relative humidity of 47%.

Post-race assessment

All subjects reported to the medical tent within five minutes of completing the race and before drinking any fluid or emptying their bladder. On arrival at the medical tent, blood samples were immediately collected from all the subjects. These samples were analysed for haemoglobin concentration, packed cell volume, plasma proteins, serum sodium, serum potassium, serum total calcium, serum total magnesium, serum osmolality, and plasma glucose. Thereafter subjects were weighed in full race attire (running shorts, vest, and shoes) as before the race. Subjects were requested to return 60 minutes later for a repeat blood sample and an interview to obtain race cramp history. Runners were allowed to consume liquids ad libitum in this 60 minute post-race period.

A second post-race blood sample was collected from subjects at 60 minutes after the race. This blood sample was analysed for serum sodium, serum potassium, serum total calcium, serum total magnesium, serum osmolality, and plasma glucose.

Race cramp history

All subjects were requested to monitor muscle cramp status during the race. These data were documented at the 60 minute post-race interview. The following data were collected from the EAMC group: muscle groups that cramped, the race distance when cramps started, the duration of cramping bouts, the recurrence of cramping bouts, cramping severity, and relieving factors for an acute cramp.

Muscle groups were listed as quadriceps, hamstrings, gastrocnemius, and other. Race distance was listed as less than 28 km, 28 to 42 km, or more than 42 km. Landmarks familiar to all Two Oceans marathon runners were used to identify these distances that relate to the first half, the third quarter, and the last quarter of the race, respectively. Duration of EAMC was recorded as lasting for less than 30 seconds, 30 to 120 seconds, and more than 120 seconds. Recurrence of EAMC referred to the number of repeat episodes of the muscle cramp after the first episode. This was quantified as one to two recurrences, three to four recurrences, or more than four recurrences until completion of the race.

Cramp severity was quantified according to the time the runner was forced to stop or run slowly and the ability to continue running afterwards. Any cramping where the runner could continue running or walking within five minutes was classified as mild. Cramping that required the runner to stop for 10 to 15 minutes but with resumption of walking or running was classified as moderate, and cramping that required stopping for more than 15 minutes with inability to resume a comfortable walking or running pace was classified as severe. Relieving factors included stretching the cramping muscle, massaging the cramping muscle, and slowing of running pace.

Criteria for discharge

Discharge criteria were that subjects had to be pain-free, able to walk freely, and have suffered no cramping activity for 30 minutes.

Analysis of blood samples

A centrifuge and two large cooler boxes filled with ice were set up in a section of the medical tent for immediate preparation and storage of blood samples. Serum was obtained for sodium, potassium, calcium, magnesium, and osmolality analysis. Blood for glucose analysis was obtained in tubes containing potassium oxalate/sodium fluoride. Blood for haemoglobin and packed cell volume analysis was obtained in tubes containing K3 EDTA 15%. Aliquots of whole blood were removed from the purple top vacutainers and kept for later analysis of packed cell volume and haemoglobin. The remainder of the vacutainer was kept on ice until centrifugation at 2500 rpm for 10 minutes. This separation of serum was within 45 minutes of obtaining the blood sample.

Storage of serum samples was done at −20°C in well sealed tubes until analysis. This storage was achieved within 90 minutes of blood sampling. The blood samples were stored at the Physiology Laboratories at the University of Cape Town (UCT) Medical School. All analyses were completed within 10 days after collection. All samples were analysed in duplicate

with suitable standards and random intervals between samples.

Serum sodium and serum potassium determinations were done using ion selective electrodes on an ionised sodium/potassium analyser (KNA1, Radiometer, Copenhagen, Denmark). Serum total calcium and total magnesium concentrations were determined using atomic absorption spectrophotometry (Varian AA 1275 series atomic absorption spectrophotometer, Varian Techtron, Musgrave, Australia). The serum samples were diluted 1:40 with 0.1% lanthanum chloride solution. The wavelengths of determination were 422.7 nm for calcium and 285.2 nm for magnesium. Serum osmolality determinations were done using an automatic osmometer (Osmette A, Precision Systems, Newton, Massachusetts, USA). Particular care was taken to ensure with storage of samples before measurement of serum osmolality. Storage may result in water evaporation, causing an error of measurement. All samples were stored in paraffin wax sealed Eppendorf tubes until analysis could be undertaken. Plasma glucose determinations were done on fluoride/oxalated plasma using an automated glucose analyser (Beckman Glucose Analyzer 2, Beckman Instruments, Brea, California, USA). This glucose analyser was based upon an enzymatic glucose oxidase technique. Serum protein determinations were done on the serum samples using the standard Biuret method.

Haemoglobin determinations were done on whole blood samples using the standard cyanmethaemoglobin technique (Drabkins solution) for haemoglobin estimation. Packed cell volume determinations were carried out in duplicate using a microhaematocrit centrifuge.

Two methods were used to determine changes in hydration status of runners in this study. The first calculated changes in pre-race and post-race body mass measurements. The following formula was used to calculate the percentage change in body weight after the race: [pre-race weight−post-race weight]/pre-race weight×100. The extent of body weight loss was used as an index of dehydration.[23] [26]

The second method to assess hydration status used the formula that is applied to haematological data to calculate the changes in blood volume, plasma volume, and red cell volume that occurred during the race.[27] The changes were calculated by applying the Dill and Costill formula to the haematological data.[27]

Statistical analysis

Statistical analysis of the data was conducted at the Medical Research Council in Cape Town using SAS system software on a Sunsperc 10 Server computer (SAS Institute Inc, *SAS/STAT User's Guide*, version 6, 4th ed; Cary, North Carolina: SAS Institute Inc, 1989). Data were analysed using both parametric and non-parametric statistics. Where data were skewed, non-parametric statistics were used. The Wilcoxon two sample test was used for comparisons between groups and within groups. The Tukey-type multiple comparison post-hoc test was used to determine where the differences between the groups lay. The Wilcoxon sign rank test was used to test for within group differences. Significance was accepted at the 0.05 level.

RESULTS
Descriptive data

The average age (years), weight (kg), and finishing time (minutes) from the cramp and control groups are presented in table 1. There were no significant differences between the two groups for any of these variables.

Table 1 Age, weight, and finishing time for the cramp and control groups

Variable	Cramp group (n=21)	Control group (n=22)
Age (years)	36.6 (7.7)	42.4 (7.5)
Weight (kg)	77.7 (11.3)	72.8 (8.4)
Finishing time (min)	309.0 (41.3)	304.3 (35.5)

Values are mean (SD).

Race cramping history

The clinical characteristics and race history of muscle cramping in the cramp group are presented in table 2. The most common muscle groups reported by the cramp group were hamstring (48%) and quadriceps (38%). All the runners (100%) reported cramping in the latter half of the race, with most (67%) reporting cramping immediately after the race. Most cramps (62%) reportedly lasted longer than 30 seconds and most runners (71%) reported three or more cramping episodes. Most cramps (57%) were moderate to severe in intensity and were best relieved by slowing the running pace (76%) or passive stretching (52%).

Serum electrolyte concentrations and haematological data

The pre-race, immediate post-race, and 60 minute post-race results for serum sodium, potassium, total calcium, and total magnesium concentrations (mmol/l), serum osmolality (mmol/kg), plasma glucose concentration (mmol/l), plasma proteins (g/l), packed cell volume (%), and haemoglobin (g/dl) from the cramp and control groups are given in table 3.

There were no significant differences between cramp and control groups for pre-race, immediate post-race, and 60 minute post-race serum potassium and calcium concentrations, osmolality, plasma glucose, plasma proteins, packed cell volume, or haemoglobin results. The immediate post-race serum sodium concentration was lower (p = 0.004) in the cramp group (139.8 (3.1) mmol/l) than in the control group (142.3 (2.1) mmol/l). The immediate post-race serum magnesium concentration was higher (p = 0.03) in the cramp group (0.73 (0.1) mmol/l) than in the control group (0.67 (0.1) mmol/l).

Table 2 Clinical characteristics and race history of muscle cramping in the cramping group (n=21)

Category	Subcategory	Percentage
Muscle group	Quadriceps	38%
	Hamstring	48%
	Gastrocnemius	14%
	Other	5%
Race distance when cramp started	<28 km	0%
	28 to 42 km	33%
	>42 km	67%
Duration of cramping episode	<30 s	38%
	30 to 120 s	38%
	>120 s	24%
Number of recurring episodes	0	10%
	1–2	19%
	3–4	38%
	>4	33%
Cramping severity	Mild	43%
	Moderate/severe	57%
Relieving factors	Stretching	52%
	Massage	24%
	Slowing race pace	76%

Values are percentage of total group.

Table 3 Values for pre-race, immediate post-race, and 60 minute post-race serum sodium, potassium, total calcium, and total magnesium concentrations, serum osmolality, plasma glucose concentration, plasma proteins, packed cell volume, and haemoglobin results for the cramp and control groups, expressed as mean (SD) and median (1st and 3rd quartiles)

	Pre-race		Immediate post-race		60 min post-race	
Variable	Cramp (n = 21)	Control (n = 22)	Cramp (n = 21)	Control (n = 21)	Cramp (n = 13)	Control (n = 16)
Mean (SD)						
Sodium (mmol/l)	139.2 (2.1)	139.3 (2.0)	139.8 (3.1)*	142.3 (2.1)*	140.3 (1.9)	141.7 (1.7)
Potassium (mmol/l)	4.5 (0.4)	4.4 (0.4)	4.9 (0.6)	4.7 (0.5)	4.7 (0.5)	4.6 (0.5)
Calcium (mmol/l)	2.2 (0.1)	2.2 (0.1)	2.3 (0.2)	2.3 (0.1)	2.2 (0.3)	2.2 (0.2)
Magnesium (mmol/l)	0.81 (0.1)	0.83 (0.1)	0.73 (0.1)*	0.67 (0.1)*	0.75 (0.1)	0.73 (0.1)
Osmolality (mmol/kg)	284 (5)	282 (4)	280 (6)	284 (10)	284 (7)	283 (8)
Glucose (mmol/l)	6.3 (3.1)	6.1 (1.1)	6.8 (1.9)	6.5 (2.0)	6.3 (1.0)	6.5 (1.1)
Plasma proteins (g/l)	73.3 (5.3)	72.7 (3.9)	76.4 (5.2)	73.7 (15.5)		
PCV (%)	40.0 (4.0)	42.0 (4.0)	40.0 (3.0)	40.0 (3.0)		
Haemoglobin (g/dl)	15.5 (1.1)	15.5 (0.8)	15.7 (1.0)	15.5 (3.2)		
Median (1st and 3rd quartiles)						
Sodium (mmol/l)	139 (138, 140)	139 (138, 141)	140 (139, 142)*	143 (141, 144)*	141 (138, 141)	142 (141, 143)
Potassium (mmol/l)	4.5 (4.2, 4.6)	4.3 (4.2, 4.5)	4.9 (4.5, 5.4)	4.7 (4.5, 5.0)	4.8 (4.2, 5.0)	4.6 (4.3, 4.9)
Calcium (mmol/l)	2.2 (2.1, 2.2)	2.2 (2.1, 2.2)	2.3 (2.2, 2.4)	2.3 (2.2, 2.4)	2.3 (2.0, 2.4)	2.2 (2.1, 2.3)
Magnesium (mmol/l)	0.8 (0.8, 0.9)	0.8 (0.8, 0.9)	0.8 (0.7, 0.8)*	0.7 (0.6, 0.7)*	0.8 (0.7, 0.8)	0.7 (0.7, 0.8)
Osmolality (mmol/kg)	284 (280, 285)	282 (280, 286)	280 (277, 286)	283 (279, 291)	284 (279, 286)	285 (282, 287)
Glucose (mmol/l)	5.3 (4.7, 6.7)	5.8 (5.6, 7.1)	6.5 (5.6, 7.1)	6.7 (5.8, 8.1)	6.3 (5.9, 7.0)	6.6 (5.5, 7.4)
Plasma proteins (g/l)	71.9 (69.6, 74.3)	72.7 (71.1, 75.4)	75.0 (73.3, 78.8)	76.2 (75.0, 80.6)		
PCV (%)	40 (40, 40)	40 (40, 40)	40 (40, 40)	40 (40, 40)		
Haemoglobin (g/dl)	15.4 (14.6, 16.1)	15.7 (14.9, 16.1)	15.6 (15.2, 16.2)	16.2 (15.6, 16.9)		

*Significant difference in the immediate post-race values between the cramp and control groups (p<0.05).
PCV, packed cell volume.

Hydration status

The average per cent change in body weight, blood volume, plasma volume, and red cell volume between immediate post-race and pre-race from both the cramp and control groups are shown in table 4. There was no significant difference between the two groups for any of these variables.

DISCUSSION

The two main findings of this study were first, that there is no relation between any clinically significant changes in serum concentrations of sodium, potassium, total calcium, and total magnesium and the development of EAMC in ultra-distance runners before or immediately after a race, or during the period of clinical recovery from EAMC; and second, that there is also no relation between the changes in hydration status (measured by changes in body weight, plasma volume, blood volume, or red cell volume) and the development of EAMC in ultra-distance runners during or immediately after a race. The results of our study also show that there is no relation between the development of EAMC in ultra-distance runners and changes in serum osmolality, blood glucose concentration, and the concentration of plasma proteins before and after a race.

The association between skeletal muscle cramping and disturbances of serum electrolyte concentrations has been well documented in a variety of medical conditions. These include plasma volume contraction and extracellular hypo-osmolality in patients undergoing haemodialysis, severe dehydration after sweating, vomiting or diarrhoea, hyponatraemia associated with whole body salt depletion, hypokalaemia and hyperkalaemia, hypocalcaemia, and more recently hypomagnesaemia.[9–12 28] In most instances, the postulated mechanism for the development of skeletal muscle cramping is related to alterations in neuromuscular excitability which can be induced by disturbances in serum electrolyte concentrations. Clinically, patients with these abnormalities present with increased generalised neuromuscular excitability, which can lead to generalised skeletal muscle cramping.

Numerous studies have shown that disturbances in serum electrolyte concentrations and hydration status can occur following ultra-distance running.[2–4 6] It is therefore commonly assumed that there is a relation between changes in serum electrolyte concentrations, hydration status, and the development of EAMC in distance runners.[15 16 18–20]

In the present study, serum electrolyte concentrations and hydration status were not associated with the clinical symptoms of EAMC. Small but statistically significant differences in serum sodium and magnesium concentrations between the cramping and control groups in the immediate post-race period in the present study are too small to be of clinical significance. Furthermore, the decrease in serum sodium concentration following the race in the cramp group is probably related to an increased fluid intake during the race in this group.[29] Although drinking patterns were not measured directly, increased drinking in the cramp group is likely because of the well publicised belief that cramping is caused by dehydration. The serum electrolyte concentrations observed in this study are also similar to those reported by others, and most importantly are too small to be of any clinical significance.[21 24 25]

This study was not designed to measure fluid balance in the runners; therefore precise data on the type and volumes of fluid ingested during the race, as well a specific losses (sweat, urine, faeces) during the race, are not presented. However, accurate and consistent data on the hydration status immediately after the race (body weight changes, changes in plasma volume, changes in blood volume) indicate that runners with EAMC were less dehydrated than

Table 4 Per cent change (pre-race to post-race) in body weight, blood volume, plasma volume, and red cell volume during the race for the cramp and control groups

Variable	Cramp group (n = 21)	Control group (n = 22)
Change in body weight (%)	−2.9 (1.2)	−3.6 (1.2)
Change in blood volume (%)	−1.3 (5.5)	−3.7 (4.0)
Change in plasma volume (%)	0.2 (6.3)	−0.7 (8.6)
Change in red cell volume (%)	−2.1 (16.2)	−8.3 (12.6)

Values are mean (SD).

non-cramping runners at the time of presentation. The per cent decrease in body weight (pre- to post-race) was less in the cramp group (2.9%) than in the control group (3.6%).

The absence of any relations between clinical recovery from EAMC and changes in serum electrolyte concentrations in the 60 minute period after the race also do not support the hypothesis that EAMC is associated with alterations in serum electrolyte concentrations. Finally, the clinical picture of EAMC is that of localised skeletal muscle cramping, and this differs substantially from the generalised skeletal muscle cramping observed in patients with serum electrolyte changes secondary to systemic disease.[9-12 28]

Conclusions
The results of our study do not support the common hypotheses that EAMC is associated with either changes in serum electrolyte concentrations or changes in hydration status following ultra-distance running. An alternative hypothesis to explain the aetiology of EAMC must therefore be sought.

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