

Clinical and biochemical characteristics of collapsed ultramarathon runners

LUCY-MAY HOLTZHAUSEN, TIMOTHY DAVID NOAKES,
BETTINA KRONING, MARNÉ DE KLERK, MIMI ROBERTS,
and ROBIN EMSLEY

*Liberty Life Chair of Exercise and Sports Science and
MRC/UCT Bioenergetics of Exercise Research Unit,
Department of Physiology,
University of Cape Town Medical School,
Cape Town, SOUTH AFRICA; and
Department of Psychiatry,
University of Stellenbosch,
Stellenbosch, SOUTH AFRICA*

ABSTRACT

HOLTZHAUSEN, L.-M., T. D. NOAKES, B. KRONING, M. DE KLERK, M. ROBERTS, and R. EMSLEY. Clinical and biochemical characteristics of collapsed ultramarathon runners. *Med. Sci. Sports Exerc.*, Vol. 26, No. 9, pp. 1095–1101, 1994. To evaluate the characteristics of runners with exercise-associated collapse (EAC), we studied the time of onset of collapse, rectal temperatures, cardiovascular status, and incidence of readily identifiable medical conditions in 46 male athletes who collapsed during or after a 56-km ultramarathon footrace run on a cool day. Data were compared with 65 control runners who did not collapse in the same race. Weight changes during recovery were studied in a subsample of both groups. The majority (85%) of runners with EAC collapsed after they had completed the race; rectal temperatures ($38.5 \pm 1.3^\circ\text{C}$, mean \pm SD; range 35.5–42.0°C) and supine heart rates ($87.5 \pm 17.2 \text{ min}^{-1}$; range 60–138) were only modestly elevated. Postrace serum sodium concentrations, changes in plasma volume, and mass during recovery were not significantly different from values in control runners. We conclude that: (i) most cases of EAC (85%) occur after the finish line; (ii) runners collapsing during the race are more likely to have a readily identifiable medical condition than runners collapsing after the finish line; (iii) runners collapse most frequently near cutoff times for medals and race closure times; and (iv) 16% of EAC casualties and 19% of control runners have identifiable biochemical abnormalities.

EXERCISE-ASSOCIATED COLLAPSE, DEHYDRATION, FLUID STATUS, POSTURAL HYPOTENSION, HYPOTHERMIA

Mass participation in endurance events lasting more than 2 h had led to an alarming increase in the number of athletes presenting with exercise-associated collapse (EAC) (1,12,26,31). Although EAC is one of the commonest disorders presenting at the medical facilities supporting such events (1,12,31), it is the least understood. EAC is not in itself a diagnosis as it gives no indication of the cause of the condition. Rather

it describes the main complaint, which is an inability to stand or walk unaided as a result of light-headedness, faintness, dizziness, or syncope (26,27,31). More severe symptoms usually resolve in the recumbent position. In some cases a medical condition such as exercise-induced hypoglycemia or exertional heatstroke (27) may be readily identified. But in the majority of cases no clear-cut diagnosis is apparent (27).

Uncertainty regarding the true nature of this condition has led to the charge that the majority of such cases may be receiving inappropriate management (21). Since it has traditionally been believed that most collapsed runners suffer from exercise-induced dehydration with or without hyperthermia (1,12,13,37), intravenous rehydration has traditionally been considered the first line of therapy for patients with EAC (10).

We have recently reported that some ultramarathon runners with EAC treated with intravenous fluids developed iatrogenic hyponatremia (16,24), suggesting that they may have been euhydrated or even overhydrated rather than dehydrated (16,22) at the finish of their race. Clearly, the underlying cause of EAC, even though probably multifactorial, must be established so that the safest and most appropriate treatment protocols can be developed.

This study was designed to identify the clinical and biochemical characteristics of EAC and determine the role that factors such as dehydration, postural hypotension, and hypoglycemia might play in its pathogenesis.

MATERIALS AND METHODS

The study was undertaken in the medical tent situated at the finish of the 1990 56-km Two Oceans Marathon that is run annually in Cape Town, South Africa. Written informed consent was obtained from all subjects on ad-

mission to the trial, which had been approved by the Research and Ethics Committee of the Faculty of Medicine of the University of Cape Town.

Thirty-eight male runners (0.006% of the 6334 race finishers) who collapsed after the finish line were admitted to the medical tent. Another eight runners (0.001% of the race finishers) who collapsed during the race and not at the finish were also admitted to the medical tent and included in the study.

Runners with EAC had their vital signs assessed within 5 min of their admission to the medical tent. Measurement included rectal temperature, supine and erect blood pressures, and pulse rates measured with a Dinamap 8100T monitor (Johnson and Johnson, Johannesburg, South Africa). Blood pressure was first measured in the supine position. The runner was then asked to assume the standing, erect position and the supine blood pressure was taken within 1 min of assuming the erect posture.

Blood samples were then taken from an antecubital vein. These were processed immediately by centrifugation at 2000 revolutions·min⁻¹ for 10 min and the serum stored frozen at -20°C until analysis. The unclotted samples were analyzed within 24 h.

Collapsed athletes were then treated with oral fluids and elevation of the pelvis and lower limbs. Ice packs were placed over the abdomen and extremities in runners ($N = 6$) with rectal temperatures greater than 39.5°C. Intravenous (IV) fluid therapy was given if there was no clinical response to this treatment within 20 min. As a result, 12 (26%) collapsed runners were given IV therapy. All recovered fully and none required hospitalization.

On discharge the runners were weighed on a Seca electronic scale (Vogel and Halke, Hamburg, Germany). They were given a plastic 2-l container in which to urinate over the next 24 h and a fluid balance chart to record their fluid intake over the same period. They were asked to return the next day to be reweighed and to have repeat blood samples and blood pressure measurements taken.

A control group of 65 male runners, who completed the race without collapsing, volunteered to participate in the study and gave their written informed consent. These runners were recruited by randomly asking for volunteers as they crossed the finish line. They were weighed and blood samples were taken as above within 10 min of finishing the race. For logistical reasons, rectal temperatures and blood pressure measurements were not taken in this group. They too were asked to return the following day with a 24-h urine sample and fluid balance chart.

Forty-eight control runners and 15 runners with EAC returned the following day. The low compliance among runners with EAC was probably because most (74%) came from distant cities and returned home soon after the race finish. This very low return of study group participants is a major drawback of this study and may have

introduced some bias into the study, at least for data collected 24 h after the race.

The blood samples were analyzed for plasma and urinary electrolytes by flame photometry (Model 543 Beckman Instruments Inc., Galway, Ireland); creatinine by the Jaffe reaction measured on a spectrophotometer (Model 919 Beckman Instruments Inc.); chloride by CMT10 chloride titrator (Beckman Instruments Inc.); serum albumin and total protein by Technicon SMAC autoanalyzer (Beckman Instruments Inc.); and osmolality using an automatic osmometer (Osmotte A, Precision Instruments Inc., Newton, MA). Plasma renin activity was determined by the radioimmunoassay of generated angiotensin I using a ¹²⁵iodine gammacoat plasma renin activity immunoassay kit (CA-553). Aldosterone activity was obtained by a coated-tube nonextraction method (Diagnostic Products Corporation, Los Angeles, CA). Vasopressin activity was determined by a radioimmunoassay method developed locally. Blood drawn for hemoglobin concentration, hematocrit, and white cell count were analyzed with a Coulter S+2 automatic cell counter (Miami, FL).

Plasma volume changes were calculated according to the equations of Dill and Costill (7).

During the race the wet bulb globe temperature (WBGT) index was measured with a locally designed instrument.

Statistical Methods

The results were calculated as the mean and standard deviation. Statistical significance (P) was determined as follows: (i) for single, within group postrace and 24 h later differences with a paired Student's t -test using two-tailed P values corrected for unequal variance, and (ii) for multiple, between-group differences with a one-way analysis of variance and an unpaired Student's t -test using P values corrected for the total possible number of comparisons.

RESULTS

Both the start and finish of the race are situated geographically at the same point—a latitude of 33° 59'S and a longitude of 18° 24'. The race start was at 0600 h with cutoff times at the standard marathon distance (42.2 km) of 4.5 h and at the finish of 6 h. Runners not achieving these cutoff times are asked to stop running and could not continue the race.

Environmental conditions during the race were mild; the WBGT index measured 13.2°C at the start of the race. This rose to 15.6°C 3 h later and to 18.9°C at the cutoff finish time of 6 h.

Eight runners collapsed before finishing the race. All had identifiable medical conditions (Table 1) and were excluded from further analysis. The etiology of the gas-

TABLE 1. Clinical diagnoses in eight runners who collapsed before finishing a 56-km ultramarathon.

Hypoglycemia (<3.8 mmol·l ⁻¹)	3
Gastroenteritis	3
Angina	1
Asthma	1

troenteritis, manifest by severe nausea, vomiting, and diarrhea in three runners, was not established.

There were no differences between runners with EAC and controls (C) in age (EAC: 33 ± 8 vs control C: 32 ± 8 (mean ± SD) yr), weight (EAC: 69.8 ± 10.4 vs C: 70.8 ± 8.9 kg), or race finishing time (EAC: 272 ± 53 vs C: 276 ± 42 min). When compared with the group of total finishers, the finishing times of runners with EAC were abnormally distributed with increased representation in the groups finishing between 225 and 240 min (4-h silver medal cutoff time) and between 345 and 360 min (race finish cutoff time). Runners with EAC were underrepresented in the groups finishing between 270 and 345 min (Fig. 1).

Table 2 lists the clinical findings in the runners with EAC at the time of collapse and 24 h later. No such data were collected in the control group. The mean pulse rate at the time of collapse was only moderately elevated but was significantly lower 24 h later. Mean supine blood pressure was essentially normal but fell by an average of 10 mm Hg on assuming the erect position. This posture-related drop in systolic blood pressure was not present 24 h later at which time systolic blood pressure in the supine and erect positions, and diastolic blood pressure in the erect position had all increased significantly (Table 2).

Postrace rectal temperatures ranged from 35.5–42.0°C (Fig. 2); 25% of values fell below 37°C and only 2.5% were above 40.5°C. Rectal temperatures, which were significantly lower 24 h after the race (Table 2), did not correlate with finishing time (r = 0.004); the highest rectal temperature was measured in one of the slowest

TABLE 2. Cardiovascular measurements and rectal temperatures in runners with EAC immediately after the race and 24 h later.

	5 min after Race		24 h after Race	
	N	Mean ± SD	N	Mean ± SD
Heart rate (beats·min ⁻¹)				
Supine	35	88 ± 17	12	61 ± 8*
Erect	35	106 ± 14	12	72 ± 5*
Systolic blood pressure (mm Hg)				
Supine	34	121 ± 17	12	132 ± 9*
Erect	34	111 ± 16	12	134 ± 12*
Diastolic blood pressure (mm Hg)				
Supine	34	68 ± 17	12	67 ± 11
Erect	34	66 ± 16	12	78 ± 11*
Rectal temperature (°C)	33	38.5 ± 1.3	12	37 ± 0.8*

* P < 0.03 5 min after vs 24 h after the race.

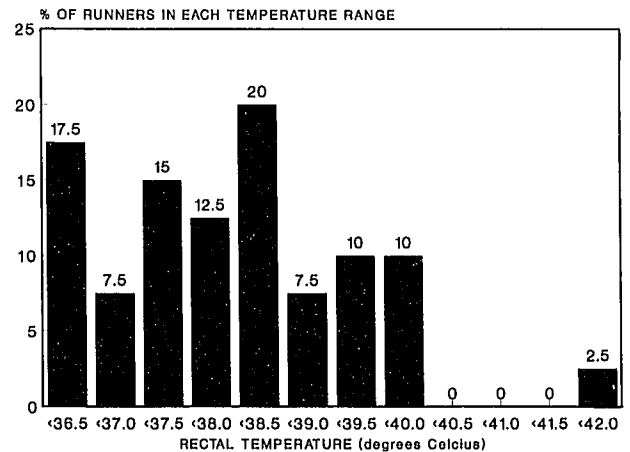


Figure 2—Distribution of postrace rectal temperatures in runners with EAC. Note that the rectal temperature of only one runner (2.5%) was greater than 40.5°C, whereas temperatures were subnormal (>37.0°C) in 17.5% of collapsed runners.

runners. Conversely, the lowest temperature (35.5°C) was measured in an athlete who completed the race in 259 min.

Table 3 shows that serum sodium, potassium, chloride, and glucose concentrations were not different between controls and runners with EAC, nor were they significantly altered 24 h later in either group. Hemoglobin, hematocrit, white cell counts, and total protein concentrations were also not different between groups. White cell counts and total protein concentrations fell in both groups during recovery. Serum renin activity and vasopressin concentrations showed a wide variance in both groups but were not significantly different between groups.

Serum urea and creatinine concentrations and serum aldosterone concentrations were significantly higher in runners with EAC. Although the urea concentrations normalized 24 h later and were not significantly different between the two groups, the creatinine concentrations remained significantly higher in runners with EAC. Urine volume (EAC: 1.1 ± 0.5 vs C: 1.5 ± 0.6 l), fluid intake (EAC: 2.3 ± 1.1 vs C: 2.5 ± 1.4 l), percent change in plasma volume (EAC: 14 ± 8 vs C: 7 ± 10%), mass

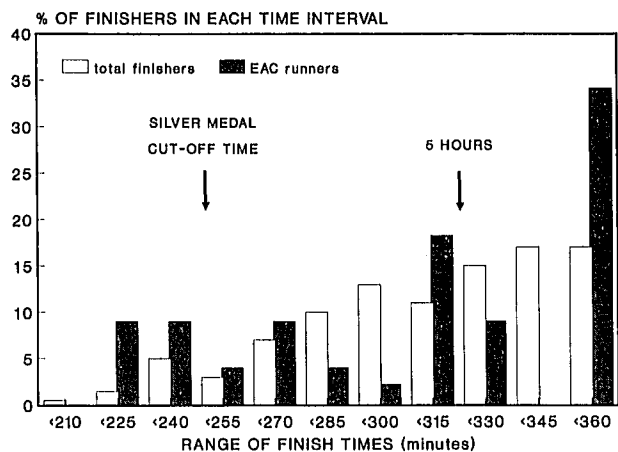


Figure 1—Comparison of distribution of finishing times of runners with EAC with that of all finishers in the 56-km ultramarathon. Note that runners with EAC were overrepresented in the groups finishing the race between 225 and 240 min and between 345 and 360 min.

TABLE 3. Blood biochemical and hematological measures in runners with EAC and in controls, 5 min and 24 h after a 56-km ultramarathon.

	5 min after Race				24 h after Race			
	Runners with EAC		Control Runners		Runners with EAC		Control Runners	
	N	Mean ± SD	N	Mean ± SD	N	Mean ± SD	N	Mean ± SD
Glucose (mmol·l ⁻¹)	38	5.8 ± 1.5	59	6.1 ± 1.2	12	4.9 ± 0.4	44	4.8 ± 0.8
Sodium (mmol·l ⁻¹)	37	141 ± 3	60	142 ± 4	12	137 ± 5	44	138 ± 4
Potassium (mmol·l ⁻¹)	37	4.8 ± 0.7	60	4.6 ± 0.4	12	4.3 ± 0.4	44	4.1 ± 0.3
Chloride (mmol·l ⁻¹)	37	102 ± 3	56	103 ± 4	10	102 ± 4	43	103 ± 3
Total protein (g·l ⁻¹)	37	73 ± 6	56	73 ± 4	10	60 ± 6*	43	65 ± 6
Urea (mmol·l ⁻¹)	37	7.4 ± 1.9*	60	6.8 ± 1.4	12	6.8 ± 2	44	6.5 ± 1.4
Creatinine (μmol·l ⁻¹)	36	141 ± 26**	60	123 ± 23	12	104 ± 13*	44	95 ± 13
Serum osmolality (mOsm·kg ⁻¹)	37	284 ± 5.6	56	285 ± 7.1	10	275 ± 11	43	278 ± 8
Plasma renin activity (mg·l ⁻¹ ·h ⁻¹)	20	18.1 ± 9.5	55	18 ± 10				
Vasopressin (pg·ml ⁻¹)	25	1434 ± 690	60	1278 ± 626				
Aldosterone (pmol·l ⁻¹)	25	5.8 ± 5.3*	64	3.8 ± 3.6				
Hemoglobin (g·dl ⁻¹)	37	15.8 ± 1	65	15.3 ± 1.2	9	15 ± 0.7	48	15 ± 1
White cell count (×10 ⁹ ·l ⁻¹)	37	17.6 ± 3.7	65	16.8 ± 3.6	9	7.7 ± 3.1	48	6.5 ± 1.8
Hematocrit (%)	37	45 ± 0.1	65	44 ± 0.1	9	43 ± 0.1	48	43 ± 0.1

* $P < 0.05$; ** $P < 0.01$ between controls and EAC runners.

changes (EAC: 1.3 ± 1.7 vs C: 1.4 ± 1.4 kg) during the 24 h after the race were the same in the subgroups that were studied.

Table 4 shows the prevalence of identifiable medical conditions in the control runners and those with EAC. Hypothermia (11%), hyponatremia (8%), and hyperthermia (7%) were the common diagnoses in runners with EAC, but hyponatremia (10%) was more common in the controls.

DISCUSSION

There were four principal findings in this study, all of which add to an understanding of the etiology of EAC.

First, 85% of all the studied runners collapsed after finishing the race. Hence, any theory must explain why the cessation of exercise is an essential etiological factor for EAC.

Second, the prevalence of identifiable medical conditions was significantly more in those who collapsed before finishing the race (100%—Table 1) than in those who collapsed at the race finish (34%—Table 4).

Third, runners collapse more frequently near cutoff times for medals and race closure, suggesting that extreme physical effort, perhaps beyond the capabilities and training status of the participants, may play a role in the etiology of this condition.

Fourth, there was a high but equal incidence of abnormal biochemical and clinical findings in the collapsed (16%) and control (19%) runners. Hence, the real sig-

nificance particularly of these biochemical changes including asymptomatic hyponatremia, which was present in 10% of control runners, is unclear. It must, however, be stressed that hyponatremia associated with symptoms, in particular alterations in the level of consciousness, is a potentially lethal condition (22) and should be suspected in collapsed subjects who do not respond to usual treatment.

We also found that the cardiovascular status of the runners with EAC was essentially normal in the supine position; the mean systolic blood pressure of 121 mm Hg and a mean heart rate of 88 beats·min⁻¹ are within the normal range for the postexercise recovery period. In contrast, patients with exercise-induced heatstroke or shock due to blood loss are markedly hypotensive and have heart rates in excess of 100 beats·min⁻¹ (11).

Although a definitive conclusion cannot be drawn as rectal temperatures were not measured in the control group, this study provides no evidence that abnormalities in body temperature, especially hyperthermia, were a critical factor in the genesis of EAC.

The distribution of rectal temperature in the runners with EAC (Fig. 2) was not different from that reported in marathon and ultramarathon runners who do not collapse after these events (17,23,25) and in runners with EAC (1,26,27,31). Only one runner had a rectal temperature in excess of 40°C (Fig. 2), but there were no other clinical features of heatstroke in that subject. Rectal temperatures in excess of 40°C are found commonly in asymptomatic subjects performing exercise of moderate to high intensity (14).

The finding that hypothermia (25%) was a more common diagnosis than hyperthermia can probably be explained by the relatively mild weather conditions prevailing during this particular race. There was no relationship between race time and the probability that hypothermia would develop; race finishing times of hypothermic runners varied from 215–360 min.

On the basis of these findings we suggest that the term

TABLE 4. Incidence of identifiable medical conditions in runners with EAC and in controls.

	Runners with EAC % of Total Sample	Control Runners % of Total Sample
Hypothermia (<36.4°C)	11	0
Hyponatremia (<135 mmol·l ⁻¹)	8	10
Hypoglycemia (<3.8 mmol·l ⁻¹)	8	3
Hyperthermia (>40°C)	7	0
Hypernatremia (>150 mmol·l ⁻¹)	0	3
Hyperosmolality (>300 mosm·kg ⁻¹)	0	3

“heat exhaustion” should not be considered synonymous with EAC, nor vice versa, as the rectal temperature is seldom markedly elevated in EAC (1,26,31) or in “heat exhaustion”. For the same reason, neither EAC nor “heat exhaustion” should be considered a mild form of exertional heatstroke (4,12,30) as there is no evidence for impaired thermoregulation and elevated rectal temperatures in persons with so-called “heat exhaustion.”

Perhaps the strongest indicator for the etiology of EAC in our subjects were the findings that (i) the great majority (85%) of such collapses occurred after the cessation of exercise and (ii) that all exhibited a postural change in blood pressure after the race (Table 2); this change was not present 24 h later. A potential link for these observations might be that the cessation of exercise causes inactivation of the muscle pump, which results in pooling of a sufficiently large volume of blood in the lower extremities and pelvis to cause circulatory decompensation leading to syncope. Any reduction in venous return resulting from blood pooling would be exacerbated by a number of additional variables.

First, a work-induced vasodilation in the muscles of the lower limb may further increase peripheral blood pooling. Also, the temperature-induced dilation of skin blood vessels resulting from the hot environment will further increase venous capacitance peripherally and therefore reduce the central blood volume (28).

This additional factor may explain why the incidence of EAC increases with the duration of exercise and is greatest after the longest races (20,21,26,31). Training, too, may attenuate the vasoconstrictor response to any hypotensive stress such as the sudden cessation of exercise particularly involving the lower limbs (32). Finally, fluid loss due to a combination of excessive sweating, diarrhea, or vomiting may further reduce central blood volume or venous return, or both.

Interesting, the early work of Adolph (2), Adolph and Fulton (3), Eichna and co-workers (8,9), Talbott et al. (33), and Weiner (34) drew attention to the possible role of postural hypotension in the etiology of “heat exhaustion” during or following exercise.

Adolph (2) noted that patients with “heat exhaustion” were “still producing sweat and keeping cool” but that they showed evidence of postural hypotension. Adolph and Fulton (3) concluded: “The peripheral blood vessels are greatly dilated during exposure to high temperatures, and this dilation continues indefinitely. The lack of a high resistance in the peripheral blood vessels prevents blood from returning to the heart. The heart rate increases steadily and rapidly, and is even able to increase the systolic blood pressure. In spite of this compensating activity on the part of the heart, the blood flow back to the heart finally becomes inadequate. At this point circulatory collapse or shock is complete, with faintness.” Adolph (2) also noted that patients with this condition

usually needed “merely to lie down to feel better. The shock-like circulatory failure appears to be the crucial element in this condition.”

Talbott et al. (33) also concluded that “heat prostration is associated with vasomotor collapse. It is known that peripheral dilation of blood vessels and tachycardia accompany exposure to high temperature. During muscular activity in high environmental temperatures, the peripheral dilation with vasomotor collapse may approach pathological proportions.”

Similarly, Eichna et al. (8) showed that 58% of subjects developed syncope on their first exposure to “exhausting” exercise in the heat. Despite the finding that the rectal temperatures were not greatly elevated (<38°C) and were similar to those measured in our subjects with EAC, those authors labeled the condition “heat exhaustion.” Others had also noted that the rectal temperature was either “submaximal or slightly elevated” in heat exhaustion (6). In common with the other early workers, Eichna et al. (9) proposed that postural hypotension explained the syncope present in their subjects with “heat exhaustion.” They concluded that this postural drop in blood pressure resulted from pooling of blood in the lower extremities on cessation of exercise due to a work-induced vasodilation in the lower limb muscles and inactivation of the muscle pump that aids venous return during exercise.

In an experimental study of heat collapse in South African gold miners, Weiner (34) concluded that the “collapse and the associated circulatory findings are explained as being due to extensive vasodilation of the peripheral vascular bed with consequent ‘pooling’ of the blood by the action of heat.” Oral temperatures measured during exercise showed that “a high degree of pyrexia is definitely not associated with heat collapse. The mouth temperature taken during work of subjects who subsequently collapsed is of the same order as those of non-collapsing individuals.”

Thus, the conclusion from these early studies must be that, historically, the terms “heat exhaustion,” “heat prostration,” or “heat syncope” were used only to describe a condition of collapse due to postural hypotension that develops in persons exercising in the heat. The terminology should not be misinterpreted to indicate that the collapse is caused by an elevated body temperature and is therefore a mild form of heatstroke. It would seem that the possibility that postural hypotension could explain EAC has been overlooked since these early studies (20,21).

We also found that runners most likely to collapse were those attempting to finish the race within certain cutoff times (Fig. 1). This suggests that extreme personal effort beyond the athlete’s capability and training level, brought on by the competitive nature of the event, may place undue stress on the athlete’s circulatory system that predisposes to syncope when the athlete stops suddenly

at the finish line. Interestingly, athletes with EAC had significantly higher serum creatinine and urea concentrations both immediately postrace and 24 h after the event (Table 3) than did controls. This could indicate that the EAC runners had exerted themselves to a greater extent and thus induced greater muscle damage than did controls.

The extent to which dehydration contributed to collapse in the runners with EAC could not be determined directly by this study. Published data provide little firm evidence that clinically severe dehydration, equal to a fall in body weight of 5–10%, develops routinely in athletes participating in prolonged exercise. Indeed most authors report no more than a 2–4% loss of body weight during prolonged exercise such as marathon running (12,15,17–20,23,35,36). Furthermore, levels of dehydration are usually less in those who have participated in events of even longer duration (29), possibly because these events are performed at a lower exercise intensity resulting in lower than expected sweat rates (23,25).

Our results suggest that high levels of dehydration alone are not the sole cause of EAC. First, it is difficult to understand why dehydration not sufficiently severe to cause collapse *during* exercise when the stress on the cardiovascular system is the greatest, should be a critical factor in the postexercise period as cardiovascular stress is reducing. Second, we found that 35% of the control runners gained more than 2.5 kg during the 24 h after the race. As the controls were asymptomatic, similar mass losses cannot by themselves have caused EAC in collapsed runners. Mass losses in that small subgroup of runners with EAC who returned for study was not different from that measured in controls. Third, postrace plasma renin activity and plasma vasopressin concentrations were the same in runners with EAC and controls and were 3 times normal resting values. Serum sodium concentrations were also similar in these groups. Even though plasma aldosterone concentrations were significantly higher in runners with EAC, the difference was small and of dubious physiological significance.

REFERENCES

- ADNER, M. M., J. J. SCARLET, J. CASEY, W. ROBINSON, and B. H. JONES. The Boston Marathon medical care team: ten years of experience. *Physician Sportsmed.* 16(July):99–109, 1988.
- ADOLPH, E. F. Physiological fitness for the desert. *Fed. Proc.* 2:158–164, 1943.
- ADOLPH, E. F. and W. B. FULTON. The effects of exposure to high temperatures upon the circulation in man. *Am. J. Physiol.* 67:573–588, 1924.
- ARMSTRONG, L. E., R. W. HUBBARD, W. J. KRAEMER, J. P. DE LUCA, and E. L. CHRISTENSEN. Signs and symptoms of heat exhaustion during strenuous exercise. *Ann. Sports Med.* 3:182–189, 1987.
- BETHELL, H., D. JEWELL, and P. BURKE. Medical hazards of a four-km fun run. *Br. J. Sports Med.* 25:181–182, 1991.
- DERRICK, E. D. Heat cramps and uremic cramps, with special reference to their treatment with sodium chloride. *Med. J. Aust.* 10:612–616, 1934.
- DILL, D. B. and D. L. COSTILL. Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. *J. Appl. Physiol.* 37:247–248, 1974.
- EICHNA, L. W., W. B. BEAN, W. F. ASHE, and N. NELSON. Performance in relation to environmental temperature. Reactions of normal men to hot humid (simulated jungle) environment. *Bull. Johns Hopkins Hosp.* 76:25–58, 1945.
- EICHNA, L. W., S. M. HORVATH, and W. B. BEAN. Postexertional orthostatic hypotension. *Am. J. Med. Sci.* 213:641–654, 1947.
- EICHNER, E. R. Sacred cows and straw men. *Physician Sportsmed.* 19(July):24, 1991.
- GREEN, D. M. and D. METHENY. The estimation of acute blood loss by the tilt test. *Surg. Gynecol. Obstet.* 84:1045–1050, 1947.
- HILLER, W. D. B., M. L. O'TOOLE, E. E. FORTRESS, R. H. LAIRD, P. C. IMBERT, and T. D. SISK. Medical and physiological considerations in triathlons. *Am. J. Sports Med.* 15:164–167, 1987.
- HUBBARD, R. W. and L. E. ARMSTRONG. Hyperthermia: new thoughts on an old problem. *Physician Sportsmed.* 17(June):97–113, 1989.
- HUGHSON, R. L., L. A. STAUDT, and J. M. MACKIE. Monitoring

In summary, possibly the most significant finding of this study was identification of the likely etiology of collapse on the basis of whether subjects collapsed before or after finishing the race. The vast majority collapsed after completing the race; although those runners had higher serum urea and creatinine concentrations than controls, there were no distinguishing biochemical characteristics indicating the likely nature of the condition. In contrast, all runners who collapsed before the race finish had identifiable medical conditions that were treatable by conventional medical protocols.

On the basis of these findings, we hypothesize that the most likely mechanism for EAC is a syncopal episode resulting from postural hypotension (5) caused by a sudden cessation of exercise and loss of the skeletal muscle pump in the lower limbs with pooling of blood in the pelvis and lower extremities. It is possible that a number of other factors, including mild dehydration, excessive racing effort, and a training-induced reduction in the vasoconstrictor response to any hypotensive stress, may contribute to EAC. Hyperthermia would likely be a more important factor in shorter races run at higher exercise intensities (25) under more severe environmental conditions.

The modern management of EAC should take account of these several possibilities, rather than assuming that one single factor, in particular dehydration, is the sole etiological factor for EAC.

This work was supported by the South African Medical Research Council, and the Nellie Atkinson and Harry Crossley Research Funds of the University of Cape Town.

The assistance of the Department of Hematology and the Renal Unit, Groote Schuur Hospital, in the analysis of blood samples is gratefully acknowledged.

Address for correspondence: Professor T. D. Noakes, MRC/UCT Bioenergetics of Exercise Research Unit, Department of Physiology, University of Cape Town Medical School, Observatory, 7925, South Africa.

- road racing in the heat. *Physician Sportsmed.* 11(May):94–105, 1983.
15. IRVING, R. A., T. D. NOAKES, C. BURGER, K. H. MYBURGH, D. QUERIDO, and R. VAN ZYL-SMIT. Plasma volume and renal function during and after ultramarathon running. *Med. Sci. Sports Exerc.* 22:165–170, 1990.
 16. IRVING, R. A., T. D. NOAKES, R. BUCK, et al. Evaluation of renal function and fluid homeostasis during recovery from exercise induced hyponatremia. *J. Appl. Physiol.* 70:342–348, 1991.
 17. MARON, M. B., S. M. HORVATH, and J. E. WILKERSON. Acute blood biochemical alterations in response to marathon running. *Eur. J. Appl. Physiol.* 34:173–181, 1975.
 18. MAUGHAN, R. J., P. H. WHITING, and R. J. L. DAVIDSON. Estimation of plasma volume changes during marathon running. *Br. J. Sports Med.* 19:138–141, 1985.
 19. NELSON, P. B., D. ELLIS, F. FU, M. D. BLOOM, and J. O' MALLEY. Fluid and electrolyte balance during a cool weather marathon. *Am. J. Sports Med.* 17:770–772, 1989.
 20. NOAKES, T. D. Why marathon runners collapse. *S. Afr. Med. J.* 73:569–571, 1988.
 21. NOAKES, T. D. The collapsed endurance athlete—time to rethink our management? *Sports Training, Med. Rehabil.* 2:171–191, 1991.
 22. NOAKES, T. D. The hyponatremia of exercise. *Int. J. Sport Nutr.* 2:205–228, 1992.
 23. NOAKES, T. D., B. A. ADAMS, C. GREEFF, T. LOTZ, and M. NATHAN. The danger of an inadequate water intake during prolonged exercise. A novel concept re-visited. *Eur. J. Appl. Physiol.* 57:210–219, 1988.
 24. NOAKES, T. D., N. BERLINSKI, E. SOLOMON, and L. M. WEIGHT. The biochemical results of intravenous fluid therapy in collapsed ultramarathon runners. *Physician Sportsmed.* 19(July):70–81, 1991.
 25. NOAKES, T. D., K. H. MYBURGH, J. H., DU PLESSIS, L. LANG, and M. LAMBERT. Metabolic rate, not percent dehydration, predicts rectal temperature after marathon running. *Med. Sci. Sports Exerc.* 23:443–449, 1991.
 26. RICHARDS, R. and D. RICHARDS. Exertion-induced heat exhaustion and other medical aspects of the City-to-Surf fun runs, 1978–1983. *Med. J. Aust.* 141:799–805, 1984.
 27. ROBERTS, W. O. Exercise-associated collapse in endurance events. A classification system. *Physician Sportsmed.* 17(May):49–57, 1989.
 28. ROWELL, L. B. *Human Circulation: Regulation During Physical Stress.* New York, Oxford University Press, 1986, pp. 174–212.
 29. RONTOYANNIS, G. P., T. SKOULIS, and K. N. PAVLOU. Energy balance in ultramarathon running. *Am. J. Clin. Nutr.* 49:976–979, 1989.
 30. ROOS, R. Medical coverage of endurance events. *Physician Sportsmed.* 15(November):140–146, 1987.
 31. SANDELL, R. C., M. D. PASCOE, and T. D. NOAKES. Factors associated with collapse following ultramarathon footraces. A preliminary study. *Physician Sportsmed.* 16(September):86–94, 1988.
 32. SMITH, M. L., H. M. GRAITZER, D. L. HUDSON, and P. B. RAVEN. Baroreflex function in endurance- and static-exercise trained men. *J. Appl. Physiol.* 64:585–591, 1988.
 33. TALBOTT, J. H., D. B. DILL, H. T., EDWARDS, E. H. STUMME, and W. V. CONSOLAZIO. The ill effects of heat upon workmen. *J. Ind. Hyg. Toxicol.* 19:258–274, 1937.
 34. WEINER, J. S. An experimental study of heat collapse. *J. Ind. Hyg. Toxicol.* 20:389–300, 1938.
 35. WELLS, C. L., J. R. STERN, and L. H. HECHT. Hematological changes following a marathon race in male and female runners. *Eur. J. Appl. Physiol.* 48:41–49, 1982.
 36. WHITING, P. H., R. J. MAUGHAN, and J. D. B. MILLER. Dehydration and serum biochemical changes in marathon runners. *Eur. J. Appl. Physiol.* 52:183–187, 1984.
 37. WYNDHAM, C. H. Heatstroke and hyperthermia in marathon runners. *Ann. N. Y. Acad. Sci.* 301:128–138, 1977.