case studies

Post-swim orthostatic intolerance in a marathon swimmer

J. BRYAN FINLAY, A. FREDERICK HARTMAN, and ROBERT C. WEIR

Department of Medical Biophysics, University of Western Ontario, London, Ontario, CANADA N6A 5C1; and New England Health Associates, Laconia, NH 03246

ABSTRACT

FINLAY, J. B., A. F. HARTMAN, and R. C. WEIR. Post-swim orthostatic intolerance in a marathon swimmer. *Med. Sci. Sports Exerc.*, Vol. 27, No. 9, pp. 1231–1237, 1995. Two swims (1993 and 1994) are described which led to post-swim orthostatic intolerance and one episode of syncope in a 50/51-yr-old well-trained and experienced marathon swimmer. The swims of 33 km and 38 km took 12 h 30 s and 17 h 35 min, respectively. Water temperature in each swim was above 23°C and rectal core-temperature stayed above 37.0°C. Air temperatures differed, ranging from 23° to 37°C and 15° to 21°C, respectively. Regular fluid consumption totalled approximately 5.0 and 6.0 l, respectively. Fifteen minutes after completing the 1993 swim, the swimmer experienced orthostatic intolerance and fainted at the lakeside; hospital tests revealed an elevated creatine phosphokinase (CK) of 521 $U \cdot l^{-1}$. The 1994 swim was abandoned due to severe muscle cramps and CK was found to be markedly elevated at 909 $U \cdot l^{-1}$. Orthostatic intolerance was recorded in both cases; however, no cardiac abnormalities were found. After overnight rest and intravenous saline infusions of 3.0 and 1.5 l, respectively, the orthostatic intolerance was relieved. Based on previous descriptions of exercise-associated collapse in marathon runners, the swimmer's orthostatic intolerance and syncope are attributed to blood pooling in his legs due to inactivation of the venous muscle pump on completion of the swim.

CORE TEMPERATURE, ENDURANCE, EXERCISE-ASSOCIATED COLLAPSE, ISOTONIC DEHYDRATION, SYNCOPE, MUSCLE PUMP

Reports of orthostatic intolerance, subsequent to marathon swims, appear to be absent in the literature; however, shifts in body fluid and associated orthostatic intolerance (hypotension) have been reported with either laboratory-based water immersion (1,9,11), bed rest (9,17,18), space flight (9,11,18,32), or lower-body negative pressure (17). Both orthostatic intolerance and exercise-associated collapse (EAC), including syncope, have been reported in marathon runners (14) and in cases of intensive, prolonged exercise (8). This report presents a case study of post-swim orthostatic intolerance and a single episode of syncope in an experienced marathon swimmer after solo swims of 12 h and 17 h 35 min in warm water of 23°–26°C.

Solo Swims of Ontario Inc. (SSO) is a provincially funded governing body, in Ontario (Canada), established to regulate the safety of cross-lake (52 km) swims in Lake Ontario. Due to the geographical nature of this lake, hypothermia can be a major concern for officials monitoring such swims. Although visible symptoms of hypothermia have been described (12), separating the effects of cold from those of muscular/mental fatigue in the marathon swimmer can present the SSO officials with a major challenge. As with the control of many other sports, no physical contact is allowed with the swimmer during the swim. Consequently, in 1993, SSO undertook a project to monitor the core temperatures of several volunteer marathon swimmers. It was hoped that such measurements (unpublished, Finlay, 1995) might provide better judgment in deciding whether to terminate a swimmer's attempt on the basis of hypothermia and/or rate-of-loss of core temperature. Although not involving hypothermia, the current report of orthostatic intolerance and post-swim syncope stems from this SSO project, and the associated core-temperature measurements are relevant to the findings.

CASE REPORT

Subject. A white, Caucasian, male swimmer is the subject of this report and he provided informed consent to

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take part in this study, as well as permitting use of his hospital data and personal data related to the two reported swims. The swimmer was 50 yr old for the first swim in 1993, and was 51 by the time of the second swim. His past achievements include: Lake Winnipeg in 1963, 29 km, 9 h 57 min; Lake Manitoba in 1964, 55 km, 24 h 17 m; Lake Ontario in 1989, 51 km, 22 h 8 min; Lake Couchiching/Simcoe in 1991, 56 km, 25 h 8 min; and Lake Erie in 1992, 43 km, 23 h 42 min, with water temperatures typically in the range of 18°–24°C.

The course. These reported solo swims took place in Lake Winnipesaukee, NH, located 184 m above sea level. The route for the first swim (July 8, 1993) was from Center Harbor to Alton Bay, a distance of 33 km. The second swim (July 15, 1994) was a two-way attempt, starting from Center Harbor and turning at Alton Bay.

Temperature measurements. Prior to the start of the swim (2.5 h in 1993; 0.5 h in 1994), the swimmer inserted a miniature T-M core-temperature telemetry pill into his rectum, approximately 80–100 mm from the anus, transmitting at 27.795 MHz to a hand-held CH-3 receiver (Mini Mitter Co. Inc., P.O. Box 3386, Sunriver, OR 97707). Previous scientific reports (2,22) indicate that such an insertion-depth is associated with accurate measurement of core-temperature in humans. The transmitter pill was enclosed (knotted) in a latex condom (Shields X, extra thick, Ortho Pharmaceutical Canada Ltd., Don Mills, Ontario) with additional lubrication (Muko Lubricating Jelly, Ingram & Bell, Don Mills, Ontario). The pill was readily inserted by the swimmer wearing a latex glove lubricated with the same sterile, bacteriostatic, water-soluble clinical gel that was used on the condom. The swimmer encountered no problem in passing gas around the pill without dislodging it. The rim of the condom was outside the anus and facilitated easy post-swim removal of the telemetry pill.

During the swim, a 5.4-m-long half-wave dipole antenna was connected to the CH-3 receiver, to improve reception of the radio signal from the swimmer. The temperature-dependent pulse-periods of the transmitter were received by the CH-3 receiver and measured with a custom-designed, battery-operated, crystal-controlled (1 kHz) counter that provided a three-figure display of the pulse-period in milliseconds. This calibrated telemetry system permitted an accurate reading of the swimmer's rectal core-temperature within 10 s of the swimmer stopping to feed.

The swimmer's schedule involved feeding stops of up to 60 s every 0.5 or 1 h. During these stops, recordings were made of core temperature, air temperature, and water temperature. Figures 1 and 2 present the associated data for the 1993 and 1994 swims, respectively.

Skin protection. After inserting the telemetry pill, Coppertone no. 30 sunblock (Plough Inc., Memphis, TN) was liberally applied to the swimmer's legs, back, and neck. A second coating of sunblock (no. 12) was applied

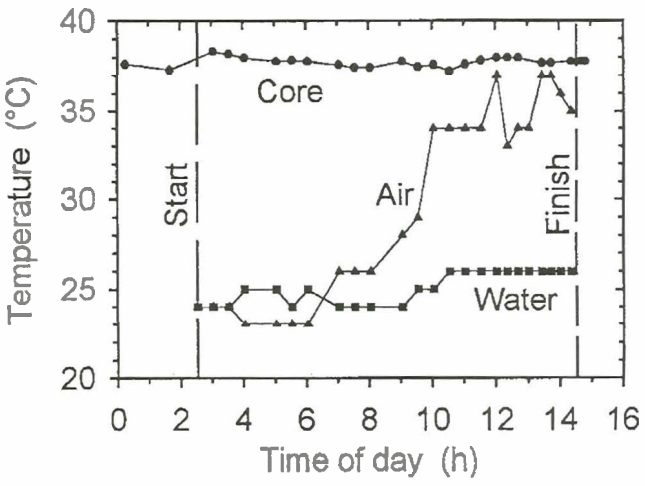

Figure 1—1993 swim: summary of data for air, water, and core body temperatures.

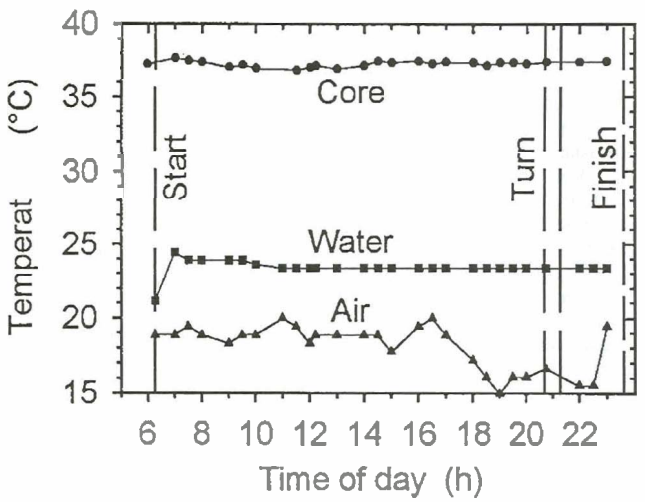

Figure 2—1994 swim: summary of data for air, water, and core body temperatures.

to these same areas 15 min before the start of the swim. The swimmer is light skinned, does not tan readily, and is known to sunburn easily. In fact, he had already been burnt mildly on his back 3 d before the 1993 swim. To prevent chafing, Vaseline was applied liberally around the neck, axillae, and swim suit.

To improve skin protection from the sun in 1994, zinc oxide cream (Ihle's Paste, Valmo Laboratory Inc., Montreal, Quebec, Canada) was applied to his back, neck, and the posterior surfaces of his legs.

Feeding. Each feed involved consumption of either 200–300 ml beef-broth soup, 235 ml of blueberry Boost (Mead Johnson Canada Ltd., Division of Bristol-Myers Squibb Canada Inc., Belleville, Ontario), fruit (nectarine, banana, and canned peaches), 200–300 ml of the electrolyte-and-carbohydrate drink Endura® (Unipro-Metagenics®, San Clemente, CA), or 200–300 ml of the

nutritional supplement Opti HealthGain™. The principal constituents per gram of Endura® are 821.9 mg carbohydrate, 6.58 mg magnesium proteinate, 4.93 mg potassium chloride, 3.84 mg phosphorus proteinate, 3.40 mg chloride, 2.52 mg sodium, and 1.64 mg calcium. Opti HealthGain™ is high in carbohydrates (in the form of glucose polymers and pure crystalline fructose) and contains predigested protein derived from lactalbumin.

The volume of voided urine was not measured; however, the swimmer voided urine on average every hour, with a greater frequency in the early stages of each swim. The swimmer experienced no need to defecate. The total fluid consumptions during the 1993 and 1994 swims were 5.5 ± 0.5 l (23 feeds) and 6.0 ± 0.5 l (27 feeds), respectively.

Swim regulations. These swims were conducted according to regulations of internationally recognized bodies such as the English Channel Swimming Association, the British Long Distance Swimming Association, and SSO. Under these rules, the swimmer is allowed to wear a cap, goggles, grease, and a noncellular swim suit. A two-way swim requires the swimmer to exit the water at the end of the first leg of the swim, and to return immediately to the water, where (s)he may rest for up to 10 min before having to resume swimming. No contact is permitted with the swimmer during the swim or turn-around. Apart from representing a solo attempt to swim the lake, each of these swims was conducted to raise funds for the Drug Abuse Resistance Education (DARE) program in New Hampshire.

1993 swim. Prior to the swim on July 8, 1993, New Hampshire had experienced a heat wave (daily temperatures exceeding 32.2°C) for more than a week and the lake temperature had risen to 24°–26°C. The swim began at 02:33 h. By 08:30 h, the swimmer was already complaining of the sun on his neck and, with a clear blue sky and temperatures over 30°C, this problem persisted for the remainder of the swim. For the last 4 h of the swim, all except two of the 11 feeds involved Endura®, and the swimmer responded with renewed energy for at least 15 min after each of these feeds. The average pace throughout the swim was 2.75 km · h^{-1} and, although the swimmer experienced fatigue during the final 4 h, a following breeze for the final 3 h caused waves of 20–100-mm, which aided the swimmer's progress.

Within 30 min of having entered the water, the swimmer's core temperature rose 1°C to 38.3°C. Within a further 60 min, his temperature settled below 38°C and ranged between 37.3° and 38.0°C for the remainder of the swim, registering 37.8°C at the completion of the swim.

The swimmer completed the swim in 12 h 30 s and, with prior arrangement by radio, he was immediately given a T-shirt and hat. The numerous news media, which had followed him on the lake for the final 8 km, gathered around the waterside with microphones and cameras. The swimmer was elated, standing ankle-deep in the water and joking with the news media while answering their questions. After approximately 15 min of questions, the swimmer came over to the inflatable lifeguard boat which had accompanied him, sat down saying that he wanted to lie down, and proceeded to close his eyes. His coach said he could not go to sleep and encouraged him to get up to walk to the shore, at which time he fainted.

TABLE 1. Cardiac and blood data related to each swim.

Parameter	Units	Reference Range	1993 Hospital	1994 Turn	1994 Hospital
Blood pressure	mm Hg	110/70	106/60	110/65	Detailed
Heart rate	beats·min^{-1}	52	65	68	in the text
Hemoglobin	g·100 ml^{-1}	14–18	15.7	16.3	
Hematocrit	%	42–52		47.8	
White cell count	$\times 10^6$·ml^{-1}	4.8–10.8	15.0 *H*	23.7 *H*	
Calcium	mg·100 ml^{-1}	8.5–11.0			9.4
Magnesium	mg·100 ml^{-1}	1.8–3.0			2.2
BUN	mg·100 ml^{-1}	6–26	26	25	25
Creatinine	mg·100 ml^{-1}	0.5–1.4			0.9
Glucose	mg·100 ml^{-1}	70–128	80	119	119
CK	U·l^{-1}	30–170	521. *H*		909. *H*
Sodium	mM·l^{-1}	135–145	135	136	134. *L*
Potassium	mM·l^{-1}	3.5–5.0	4.0	4.0	4.0
Chloride	mM·l^{-1}	95–107	100	94. *L*	92. *L*
Enzymatic CO_2	mM·l^{-1}	22–31		27	30
Anion gap (K^+)	mM·l^{-1}	7–23		20	16

BUN = blood urea nitrogen; CK = creatine phosphokinase.
H (high) and *L* (low) signify data that are above or below the Reference Range respectively.

Paramedics, who had been routinely requested for the end of the swim, were quick to take over. The swimmer was placed supine in the shade, chemical cold packs were positioned in his axillae and groin area, and oxygen was administered. The telemetry pill still indicated a rectal core-temperature of 37.8°C. After about 1 min, his eyes opened and he regained consciousness. Within 5 min, he was carried by stretcher to the ambulance and taken about 30 km to the Lakes Region General Hospital in Laconia NH. During this trip, he was readily conversing with the paramedics and discarded the oxygen mask.

On a stretcher in the Emergency Department, with rectal core-temperature still at 37.8°C and tympanic temperature at 36.8°C, his pulse was regular and normal at 65 beats · min^{-1}, and blood pressure (BP) in the supine position was 106/60 mm Hg. The swimmer's medical records showed that his normal BP was 110/70 mm Hg. A 2-h EKG (3-lead) and a 12-lead EKG revealed no cardiac abnormalities. The attending physician (A.F.H.) ordered an intravenous (IV) drip of 5% dextrose with half-normal saline and 20 mEq of potassium chloride added to each liter; this solution ran at 250 ml · h^{-1}. Previous personal experience in treating ultramarathoners has shown that such athletes need, and tolerate, high-volume fluid replacement. His hemoglobin and blood chemistry were normal (Table 1), except for a notably elevated level of creatine phosphokinase (CK) at 521 U · l^{-1} (over 3 times the maximum of the normal

range). This muscle-breakdown product, however, was a normal expectation after the thigh-muscle fatigue that the swimmer had experienced during the last 2 h of the swim, and was judged as not being cardiac related, i.e., since CK-MB (the cardiac fraction) was only 12 $U \cdot l^{-1}$ (normal).

The swimmer still experienced some light-headedness when he sat up on the stretcher, and this feeling was immediately relieved in the supine position. Consequently, he volunteered to be admitted to the hospital for the night. Two liters of IV solution had been administered in the emergency room and a further 4.0 l were administered during the night. While in the hospital, he received a total of 6.0 l of IV solution and voided 3.0 l. He was discharged from the hospital at 09:30 h on the following morning, was interviewed at the local radio station, and, although somewhat debilitated for the remainder of the day (again over 30°C), he encountered no subsequent ill effects.

1994 swim. While several weeks of warm weather produced a stable water-temperature of 23°-24°C, the weather during this swim was unfavorable. The sky was overcast throughout the attempt. A head wind for the first 11 h of the swim produced waves of 100–300 mm height. Light-to-heavy rain started after 8 h of the swim and continued for about 5 h. Air temperature ranged between 18° and 20°C, but dropped 3°C about 11 h into the swim.

The swimmer completed the first leg of the swim (33 km) in 14 h 20 min; however, for the last 8 h he had experienced increasing degrees of cramping in the anterior muscles of the thighs. Due to these cramps, the swimmer was unable to walk clear of the water at the turn at Alton Bay. He sat on a picnic bench that had been submerged in the water for this purpose, and spoke enthusiastically with the news media. A physician was in attendance and a paramedic took blood samples for subsequent analysis. The cool wind caused the swimmer to shiver uncontrollably, so a blanket was placed around his shoulders to protect him from the wind while he ate linguini and drank a total of 800 ml of hot tea with sugar but no milk. Without treatment, the severe thigh cramps would have prevented the swimmer starting the return trip; however, due to the publicity for this fund-raising event, about 200 people and news media were at the turning point, and the swimmer was intent on starting the return journey, with a goal of swimming as far as possible. Consequently, the swimmer's thighs were massaged by the attending sport-medicine physician, in an effort to relieve the severe cramping. Contact with the swimmer, and the 35 min stop, meant that a two-way swim would not be officially recognized; however, the return trip was started to aid the fund-raising campaign.

The swimmer started the return trip appearing quite fresh; he was no longer shivering and was free of his cramps. The cramping, however, resumed with increased severity after about 1 h, and the swimmer voluntarily left the water after 2 h 24 min of the return trip, having completed a further 5 km, for a total distance of 38 km. Paramedics on the attending power boat transported him to the Lakes Region General Hospital where he was admitted at 01:20 h.

At the hospital, analysis of his blood chemistry (Table 1) revealed marginally low levels of sodium (134 $mM \cdot l^{-1}$) and chloride (92 $mM \cdot l^{-1}$); however, CK at 909 $U \cdot l^{-1}$ was markedly elevated (over 5 times the maximum of the normal range; Table 1) and was attributed to the severe muscle cramps experienced by the swimmer for most of the final 12 h of the swim. All of the other monitored parameters of blood chemistry were normal.

Analysis of the blood samples taken at the turning point at Alton Bay revealed a notably elevated white cell count ($23.7 \cdot 10^6$ ml^{-1}) and a marginally low level of chloride (94 $mM \cdot l^{-1}$), together with normal levels of hemoglobin and other blood-chemistry parameters (Table 1).

At 02:30 h, with heart rate (HR) 60 beats $\cdot$ min^{-1}, and BP 90/59 mm Hg, the swimmer felt light-headed while sitting to eat a meal of lasagna, and the problem was immediately relieved when he resumed a supine position. At 03:36 sitting, HR was 74 beats $\cdot$ min^{-1}, and BP 107/69 mm Hg. Sitting and dangling legs over the edge of the bed (03:39 h), HR was 72 beats $\cdot$ min^{-1}, and BP dropped to 88/64 mm Hg. Still in the same position (03:42 h), HR was 74 beats $\cdot$ min^{-1}, and BP was 96/63 mm Hg, so the swimmer attempted to stand; however, light-headedness forced him to return to a seated position. At 03:53 h, the swimmer stood briefly and stated that he felt better (HR was 77 beats $\cdot$ min^{-1}, and BP 88/65 mm Hg); however, subsequent light-headedness when standing led to the decision to stay overnight in the hospital.

The swimmer was discharged the following morning at 10:30 h with a standing HR of 79 beats $\cdot$ min^{-1}, BP 103/68 mm Hg, mean arterial BP of 83 mm Hg, and no further symptoms of orthostatic intolerance. During the 1994 hospital admission, 2.0 l of normal saline (0.9%, with no added sugars or salts) were infused into the swimmer and he voided 500 ml of urine.

DISCUSSION

This case report has identified that the major factors influencing the reported orthostatic intolerance experienced by this swimmer appear to be his degree of hydration, and the muscle-fatigue/cramps in his lower limbs.

Field data. Air temperatures were recorded from the open, inflatable boat that accompanied the swimmer and, during the period 11:00–14:30 h of the 1993 swim, were somewhat variable—due largely to the lack of shade in the boat, a light wind, and the high position of the sun

during that time. Water temperatures also varied slightly, depending on the depth of water. The technique and instrumentation employed in the recording of rectal temperatures, however, ensured accurate measurements (unpublished, Finlay, 1995).

Fluid balance. Sawka (30) has reported the effect of hypohydration during exercise in the heat in affecting thermoregulation through sweating and skin blood flow; however, the measurements of core temperature during and after the swim, together with the blood chemistry data, provide no indication of such hypohydration-induced problems in this swimmer.

Nadel (23) states that a loss of body water in excess of 3% of body weight constitutes clinical dehydration and is associated with the early signs of heat related disorders such as light-headedness and syncope. Post-swim sodium, potassium, and chloride levels in the blood were normal; and, while the loss of body fluid was not monitored during the reported swim, hospital-based stabilization of the swimmer, in the 1993 swim, involved a net infusion of 3.0 l of half-normal saline (containing 5% dextrose and 20 mEq of potassium chloride added to each liter) over a 19-h period, i.e., approximately 3.8% of the body weight of the 78 kg swimmer. These findings, therefore, suggest isotonic dehydration of the swimmer.

In this swimmer, it was subsequently clear from the data on fluid-administration and fluid-loss in the 1993 swim that he was somewhat dehydrated (albeit isotonically). Although fluid intake is clearly important for the well-being of marathoners, Noakes et al. (25) have cautioned against excessive fluid intake/infusion in causing hyponatremia and, in the case of post-exercise intravenous therapy, hyperglycemia in these athletes.

In the 1994 swim, there was an in-hospital net infusion of 1.5 l of normal saline. Given the generally cool temperatures and regular fluid-intake during the swim, clinical dehydration was not an important factor in the 1994 swim.

While post-swim orthostatic intolerance does not appear to have been reported previously in marathon swimmers, the similarity of reported shifts in body-fluid and the associated orthostatic intolerance (hypotension) with either water immersion (1,9,11), bed rest (9,17,18), space flight (9,11,18,32), or lower-body negative pressure (17) are all relevant to this case. As with this swimmer, measures of hemoglobin and hematocrit in these latter circumstances have often stayed within normal ranges while extravascular fluid volumes have changed notably (27). In a series of 7-h experiments, Simanonok and Bernauer (31) reported that subjects seated up to the neck in 35° ± 0.5°C water had a 365% increase in urine production, and a 200% increase in sodium excretion relative to control subjects in dry air. The mechanisms and phenomena associated with these fluid shifts have been summarized by Lloyd (19).

Reported techniques for combatting such fluid shifts and their associated adverse effects have included blood volume reduction by 15% (31), autogenic-feedback training (7), the use of 1.07% saline to produce hyperhydration (10) instead of the conventional use of 0.9% saline for rehydration (5), and the use of glycerol solutions (4,21,27). While an intentional 15% reduction of blood volume is generally not desirable in a performing athlete, each of the other three techniques offers some promise for the management of potential orthostatic intolerance due to fluid loss in a marathon swimmer. Clearly, such measures have to be viewed in association with the needs for ensuring an adequate fluid intake and electrolyte replacement during the swimming activity.

Muscle fatigue. In explaining post-exertional orthostatic hypotension and associated syncope after prolonged exercise of 7–9 h, Eichna et al. (8) proposed in 1947 that "The causative factor appears to be a pooling of the blood in the dependent lower extremities, presumably due to the failure of the muscular venopressor mechanism in the legs, plus a work induced dilatation of their vascular beds." More recently (1993), Holtzhausen et al. (14) have focused on the same explanation for a number of cases of EAC in ultramarathon runners. The venous muscle pump of the lower limb, in controlling the potential "pooling" of blood in the lower limbs, is an established and essential element in the maintenance of normal blood pressures (28). It is possible that warm water of 23°C or more may well contribute to the work-induced vasodilation of the vascular beds described by Eichna et al. (8).

Holtzhausen et al. (14) reported significantly elevated levels of serum creatinine and urea concentrations, measured post-race in marathon runners with EAC. Neither of these parameters (where measured) were elevated at the completion of either of our two reported swims; however, CK was markedly elevated at the completion of each swim, and the levels of CK reflected the increased muscle-cramping experienced in the second swim.

Both Holtzhausen et al. (14) and Wells et al. (34) have reported marked elevation of white cell counts (with mean peak values of $17–18 \cdot 10^6$ ml^{-1}) in both EAC individuals and control runners after marathon runs, and noted that these counts fell to the normal range within 24 h after the runs. Wells et al. (34) describe "margination" and an inflammatory response to local tissue injury as the possible reasons for these increased white cell counts; given the severe muscle cramps reported for the 1994 swim, the tissue-injury theory could well explain the notable elevation in the recorded white cell count of ($23.7 \cdot 10^6$ ml^{-1}).

To put these dehydration and muscle-fatigue findings into perspective, it should be noted that no such orthostatic intolerance had been recognized by this swimmer in swims of twice the duration (i.e., 24–25 h) that he had

completed during the previous 3 yr in both cooler air and water temperatures. While air temperature may be a contributing factor, it would appear that water temperature over 23°C may be the major feature affecting swimmer-dehydration, muscle fatigue, and associated orthostatic intolerance. Although cold-induced vasodilatation has been reported to cause fainting (24), the features associated with the syncope in the first swim of this reported case are not compatible with such a problem.

Sunburn. Severe sunburn will produce a long-term cutaneous vasodilation, which is known to be associated with a reduction in central blood volume (29). In the first swim, the swimmer experienced significant discomfort from sunburn, which undoubtedly contributed to his post-swim debilitation. Improved effectiveness of sunblocking creams has been reported anecdotally, through application of the cream for several days before the anticipated long exposure. While the swimmer in this report employed two coatings of sunblock, they were applied within 2.5 h of each other; however, the problem was exacerbated by an existing mild sunburn over the back of the neck and upper back as a result of a training swim 3 d previously. Our recent experience has shown that zinc-oxide cream (Ihle's Paste: a mixture of equal parts of zinc oxide, starch, anhydrous lanoline, and white petrolatum) remains in place during water-immersion up to at least 18 h, and is effective in preventing sunburn on critically sensitive areas such as the neck, back, upper thighs, and legs.

Core temperature. Although the normal body temperature measured orally is 37.0°C, Harnett et al. (12) indicate that the "normal" core temperature when measured rectally is 37.6°C, and that this normal core-temperature has a diurnal variation of 1°–2°C. A rectal temperature higher than the accepted normal oral temperature is compatible with measurements that we have taken on four separate swimmers. Keatinge (16) notes that, in cases of rapid cooling, rectal temperature can lag behind the temperature of the heart by 1°–2°C. The reported swim encountered quite steady water-temperatures and so should not have produced such a problem for monitoring during the swim; however, the "stable" rectal temperatures, monitored at the time of fainting, might not have been indicative of temperatures in the cerebral blood-flow (13). No "after drop" (13,15,20) was noted in the rectal temperature within 0.5 h of the swimmer leaving the water in the 1993 swim, and no "drop" was noted during the period of shivering at the turning point in the 1994 swim.

Overview. While this study was initiated as a prospective evaluation of core-temperature monitoring in the safety management of marathon swimmers, the associated data and clinical findings have led to a valuable retrospective evaluation of the factors affecting potential orthostatic intolerance in such athletes. Blood chemistry measured pre- and post-swim is essential to exclude the effects of electrolyte imbalance in such cases and, although providing largely negative findings in this case, they were diagnostically useful in confirming a normal balance of blood electrolytes. For a swim to be recognized officially, rules for marathon swimming dictate no contact with the swimmer during the swim (solo or competitive); however, monitoring of blood sugars in a diabetic has been allowed by SSO for safety reasons. While a swimmer's fluid intake may be readily recorded, measurement of urine excretion and/or its acquisition for chemical analysis both pose considerable challenges. Changes in body weight, however, could be readily recorded, with some allowance for changes in adhering water and/or grease.

Physical mechanisms for combatting orthostatic dizziness include either squatting or standing with legs-crossed (33). Had the swimmer and coach been aware of the cause of the light-headedness (with its relatively slow onset) at the end of the first swim, the swimmer would have been allowed to lie down or adopt one of these positions, and the episode of syncope could have been avoided.

Marathon swimming is generally associated with a cold-water endurance challenge that presents problems of potential hypothermia (15,26), a condition that is considered to occur at, and below, a core body temperature of 35°C in humans (3). While training can influence these responses, body composition appears to be associated with better correlations of an athlete's tolerance to cold (15,26). Initial immersion in cold water has been associated with an exercise-induced transient increase in core-temperature of up to 1.3°C, depending on factors such as water temperature, vigorousness of exercise, and the athlete's body composition (6,15). The two swims reported in this case, and our own unpublished measurements on two other well-trained marathon swimmers, have confirmed these figures.

CONCLUSION

The well-being of a marathon swimmer depends on maintenance of an adequate core body temperature, balanced levels of electrolytes, and a proper fluid-balance. While a swimmer may have consumed several liters of fluid during the swim, fluid imbalance may still exist due to urination, perspiration, and respiration. Post-swim, orthostatic intolerance may then develop from such fluid imbalance/shifts and should be recognized as a potential consequence of inactivation of the venous muscle pump of the lower limbs at the completion of the swim, especially when severe muscle fatigue has been present during the athletic effort. A swimmer's susceptibility to fluid-loss is most easily assessed during training swims by monitoring body-weight before and after the swim. As a preventive measure during marathon swims, therefore,

it would be appropriate for swimmers, coaches, trainers, swim-organizers, and/or attending physicians to monitor these changes in body-weight, particularly when training swims or previous swims have indicated a fluid-loss approaching or exceeding 3% of body-weight. In focusing on dehydration, the potential for significant problems of hyponatremia, caused by hyperhydration, should not be overlooked. Swimmers and their support crew should also be aware of the potential for, and consequences of, pooling of blood in the lower limbs following prolonged strenuous exercise; this knowledge should also include the simple physical mechanisms, such as squatting and leg-crossing, for combatting this hypotension.

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Bryan Finlay is from the Department of Medical Biophysics, University of Western Ontario, Canada and Frederick Hartman is from New England Health Associates, Laconia, New Hampshire.

Present address for R. C. Weir: c/o Solo Swims of Ontario Inc., P.O. Box 215, Station "R", Toronto, Ontario, Canada, M4G 3Z9.

Address for correspondence: Bryan Finlay, Medical Biophysics, Medical Sciences Building, University of Western Ontario, London ON N6A 5C1, Canada.

REFERENCES

1. Arborelius, M., Jr., U. I. Balldin, B. Lilja, and C. E. G. Lundgren. Hemodynamic changes in man during immersion with the head above water. *Aerosp. Med.* 43:592–598, 1972.
2. Benedict, F. G. and Slack, E. P. In: *A Comparative Study of Temperature Fluctuations in Different Parts of the Human Body: Publication No. 155*, Washington, DC: Carnegie Institute of Washington, 1911, pp. 1–73.
3. Bligh, J. and K. G. Johnson. Glossary of terms for thermal physiology. *J. Appl. Physiol.* 35:941–961, 1973.
4. Bondar, R. L., M. S. Kassam, F. Stein, P. T. Dunphy, and M. L. Riedesel. Simultaneous transcranial Doppler and arterial blood pressure response to lower body negative pressure. *J. Clin. Pharmacol.* 34:584–589, 1994.
5. Bungo, M. W., J. B. Charles, and P. C. Johnson, Jr. Cardiovascular deconditioning during space flight and the use of saline as a countermeasure to orthostatic intolerance. *Aviat. Space Environ. Med.* 56:985–990, 1985.
6. Costill, D. L., P. J. Cahill, and D. Eddy. Metabolic responses to submaximal exercise in three water temperatures. *J. Appl. Physiol.* 22:628–632, 1967.
7. Cowings, P. S., W. B. Toscano, N. E. Miller, et al. Autogenic-feedback training: a potential treatment for orthostatic intolerance in aerospace crews. *J. Clin. Pharmacol.* 34:599–608, 1994.
8. Eichna, L. W., S. M. Horvath, and W. B. Bean. Post-exertional orthostatic hypotension. *Am. J. Med. Sci.* 213:641–654, 1947.
9. Frey, M. A. B., J. B. Charles, and D. E. Houston. Weightlessness and response to orthostatic stress. In: *Circulatory Response to the Upright Posture*, J. J. Smith. (Ed.). Boca Raton, FL: CRC Press, 1990, pp. 65–120.
10. Frey, M. A. B., C. Lathers, J. Davis, S. Fortney, and J. B. Charles. Cardiovascular responses to standing: Effect of hydration. *J. Clin. Pharmacol.* 34:387–393, 1994.
11. Gotshall, R. W., S. Yumikura, and L. A. Aten. Effect of the prelaunch position on the cardiovascular response to standing. *Aviat. Space Environ. Med.* 62:1132–1136, 1991.
12. Harnett, R. M., J. R. Pruitt, and F. R. Sias. A review of the literature concerning resuscitation from hypothermia: part I—the problem and general approaches. *Aviat. Space Environ. Med.* 54:425–434, 1983.
13. Hervey, G. R. The physiology of cold/wet survival. *J. R. Nav. Med. Serv.* 58:161- 170, 1972.
14. 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.* 26: 1095–1101, 1994.
15. Kanaar, A. C. and M. W. Hecht. Marathon swim training in a 74-yr-old man: personal experiences. *Med. Sci. Sports Exerc.* 24:490–494, 1992.
16. Keatinge, W. R. Consequences and treatment of hypothermia. In: *Survival in Cold Water*. Oxford: Blackwell Scientific Publications, 1969, pp. 63–74.
17. Lathers, C. M., J. B. Charles, V. S. Schneider, M. A. B. Frey, and S. Fortney. Use of lower body negative pressure to assess changes in heart rate response to orthostatic-like stress during 17 weeks of bed rest. *J. Clin. Pharmacol.* 34:563–570, 1994.
18. Lathers, C. M. and J. B. Charles. Orthostatic hypotension in patients, bed rest subjects, and astronauts. *J. Clin. Pharmacol.* 34:403–417, 1994.
19. Lloyd, E. L. Definitions and classification. In: *Hypothermia and Cold Stress*. London: Croom Helm, 1986, pp. 42–48.
20. Lloyd, E. L. Hypothermia. In: *Hypothermia and cold stress*. London: Croom Helm, 1986, pp. 49–54.
21. Lyons, T. P., M. L. Riedesel, L. E. Meuli, and T. W. Chick. Effects of glycerol-induced hyperhydration prior to exercise in the heat on sweating and core temperature. *Med. Sci. Sports Exerc.* 22:477–483, 1990.
22. Mead, J. and C. L. Bommarito. Reliability of rectal temperature as an index of internal body temperature. *J. Appl. Physiol.* 2:97–109, 1949.
23. Nadel, E. R. Body fluid and electrolyte balance during exercise: Competing demands with temperature regulation. In: *Thermal physiology*, J. R. S. Hales. (Ed.). New York: Raven Press, 1984, pp. 365–376.
24. Nelms, J. D. Adaptation to cold and cold injury. *J. R. Nav. Med. Serv.* 58:189–194, 1972.
25. Noakes, T. D., N. Berlinski, E. Solomon, and L. M. Weight. Collapsed runners: blood biochemical changes after IV fluid therapy. *Physician Sportsmed.* 19:70–81, 1991.
26. Pugh, L. G. C. E., O. G. Edholm, R. H. Fox, et al. A physiological study of Channel swimming. *Clin. Sci.* 19:257–273, 1960.
27. Riedesel, M. L., D. Y. Allen, G. T. Peake, and K. Al-Qattan. Hyperhydration with glycerol solutions. *J. Appl. Physiol.* 63: 2262–2268, 1987.
28. Rowell, L. B. Adjustments to upright posture and blood loss. In: *Human Circulation: Regulation During Physical Stress*. New York: Oxford University Press, 1986, pp. 137–173.
29. Rowell, L. B. Thermal stress. In: *Human Circulation: Regulation During Physical Stress*. New York: Oxford University Press, 1986, pp. 174–212.
30. Sawka, M. N. Physiological consequences of hypohydration: exercise performance and thermoregulation. *Med. Sci. Sports Exerc.* 24:657–670, 1992.
31. Simanonok, K. E. and E. Bernauer. Blood volume reduction counteracts fluid shifts in water immersion. *Aviat. Space Environ. Med.* 64:139–145, 1993.
32. Simanonok, K. E. and J. B. Charles. Space sickness and fluid shifts: a hypothesis. *J. Clin. Pharmacol.* 34:652–663, 1994.
33. Van Lieshout, J. J., A. D. J. Ten Harkel, and W. Wieling. Physical manoeuvres for combating orthostatic dizziness in autonomic failure. *Lancet* 339:897–898, 1992.
34. 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.