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Hyperthermic Fatigue Precedes a Rapid Reduction in Serum Sodium in an Ironman Triathlete: A Case Report

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Bradley A. Wall, and Kazunori Nosaka**

Purpose: To monitor the hydration, core temperature, and speed (pace) of a triathlete performing an Ironman triathlon. **Methods:** A 35-year-old experienced male triathlete participated in the Western Australian Ironman triathlon on December 1, 2006. The participant was monitored for blood Na^+ concentration before the race (PRE), at the transitions (T1 and T2), halfway through the run (R21), and after the race (POST; 2hPOST). Core body temperature (T_{c} ; pill telemetry) was recorded continuously, and running speed (s3 stride sensor) was measured during the run. **Results:** The participant completed the race in 11 h 38 min, in hot conditions ($26.6 \pm 5.8^{\circ}\text{C}$; $42 \pm 19\%$ rel. humidity). His T_{c} increased from 37.0 to 38.6°C during the 57-min swim, and averaged 38.4°C during the 335-min bike ($33.5 \text{ km}\cdot\text{h}^{-1}$). After running at $12.4 \text{ km}\cdot\text{h}^{-1}$ for 50 min in the heat (33.1°C), T_{c} increased to 39.4°C , before slowing to $10.0 \text{ km}\cdot\text{h}^{-1}$ for 20 min. T_{c} decreased to 38.9°C until he experienced severe leg cramps, after which speed diminished to $6 \text{ km}\cdot\text{h}^{-1}$ and T_{c} fell to 38.0°C . The athlete's blood Na^+ was constant from PRE to T2 ($139\text{--}140 \text{ mEq}\cdot\text{L}^{-1}$), but fell to $131 \text{ mEq}\cdot\text{L}^{-1}$ at R21, $133 \text{ mEq}\cdot\text{L}^{-1}$ at POST, and $128 \text{ mEq}\cdot\text{L}^{-1}$ at 2hPOST. The athlete consumed 9.25 L of fluid from PRE to T2, 6.25 L from T2 to POST, and lost 2% of his body mass, indicating sweat losses greater than 15.5 L. **Conclusion:** This athlete slowed during the run phase following attainment of a critically high T_{c} and experienced an unusually rapid reduction in blood Na^+ that preceded cramping, despite presenting with signs of dehydration.

Keywords: core temperature, hyponatremia, dehydration, triathlon

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Hyperthermia elicits a marked lowering of exercise intensity, especially once critical core temperatures (T_c) are reached.¹ However, field data examining the associations among hydration status, blood sodium concentration, T_c , and performance are limited.² We had the opportunity to collect measures of hydration status, T_c , and race speed in an experienced Ironman triathlete as he performed throughout a warm Ironman Western Australia triathlon.

Methods

The participant, a 35-year-old experienced male triathlete (10 Ironman triathlons: best time, 10 h 14 min) competed in his 11th Ironman triathlon in Busselton, WA, on December 1, 2006. Four days before the race, the athlete's body mass, height, percent body fat (DEXA), cycling VO_{2max} , and peak power output were measured to be 75.0 kg, 178 cm, 10.6%, 67.0 mL·kg⁻¹·min⁻¹, and 432 W, respectively. From participation in previous research, the subject's sweat rate and critical T_c were determined to be 1.42 L·h⁻¹ and 39.1°C, respectively.

Air temperature during the Ironman ranged from 15.6°C to 33.1°C, with an average value of 26.6°C. Core temperature (CorTemp; HQInc., Palmetto, FL), heart rate (Polar RS800sd), and running speed (s3 stride sensor; Polar Electro, Finland) were recorded at 10-s increments throughout the race. Body mass, blood and urine osmolality (Advanced Instruments, Inc., Needham Heights, MA), urine specific gravity (Nippon 503, Japan), and plasma aldosterone concentration (SPAC-S, T.F.B. Co., Tokyo, Japan) were measured before and after the race. Fingertip blood samples were taken from the athlete 4 d before the race (PRE1), the evening before the race (PRE2), at the transitions (T1 and T2), 21 km into the run (R21), after the race (POST), and 2 h after the race (2hPOST) and measured for sodium (Na⁺) concentration (iSTAT Corporation, East Windsor, NJ). The subject was permitted to eat and drink ad libitum during the race but made detailed mental notes of his food and fluid consumption (type and quantity) throughout the race (reported in Table 1). The subject was of the mindset that he should drink fluids throughout the event as dictated by his thirst,³ and did so accordingly.

Table 1 Estimated Na⁺ and K⁺ intake based on food consumption during the event

Source	Amount	Na ⁺ Content (mEq)	K ⁺ Content (mEq)
Gatorade	7.5 L	150	23.3
Coca-Cola	1.5 L	10	0
Carbo-Shotz	7	11	3.2
Powergel	1	9	0.5
Banana	1	0	11.3
Endura	750 mL	10.7	12
Sustagen	250 mL	6.5	18.5
Total		197.2 (~4.5 g)	68 (~2.7 g)

Results

The athlete completed the race in 11 h, 38 min, and 41 s (swim 1:07:03; cycle 5:25:06; run 5:06:31; h:min:s). The subject's T_{c} , heart rate, and speed over the duration of the Ironman event are presented in Figure 1. Total fluid consumption during the race was 15.5 L, consisting of 9.25 L consumed from the start to T2 (1.71 L·h⁻¹) and 6.25 L consumed from T2 to the finish (1.23 L·h⁻¹). The athlete's blood [Na⁺] was 141 and 140 mEq·L⁻¹ at PRE1 and PRE2, 139 mEq·L⁻¹ at both T1 and T2, 131 mEq·L⁻¹ at R21, 133 mEq·L⁻¹ at POST1 and 128 mEq·L⁻¹ at POST2. At the finish, the subjects' body mass (73.5 kg) was 2% lower than his prerace body mass (75.0 kg), indicating sweat losses of ~16.5 L (1.42 L·h⁻¹). Urine specific gravity and osmolality and serum aldosterone levels increased from 1.015, 465 mOsm·kg⁻¹, and 15.7 ng·dL⁻¹ before the race, to 1.025, 619 mOsm·kg⁻¹, and 157.7 ng·dL⁻¹ at the finish, indicating mild dehydration.

Discussion

This case study presents two original observations. First, once a critically high T_c was experienced during the run phase of the Ironman (~425 min into the race; 39.4°C),¹ pace slowed dramatically (~1.5 km·h⁻¹; Figure 1). To the authors' knowledge, this

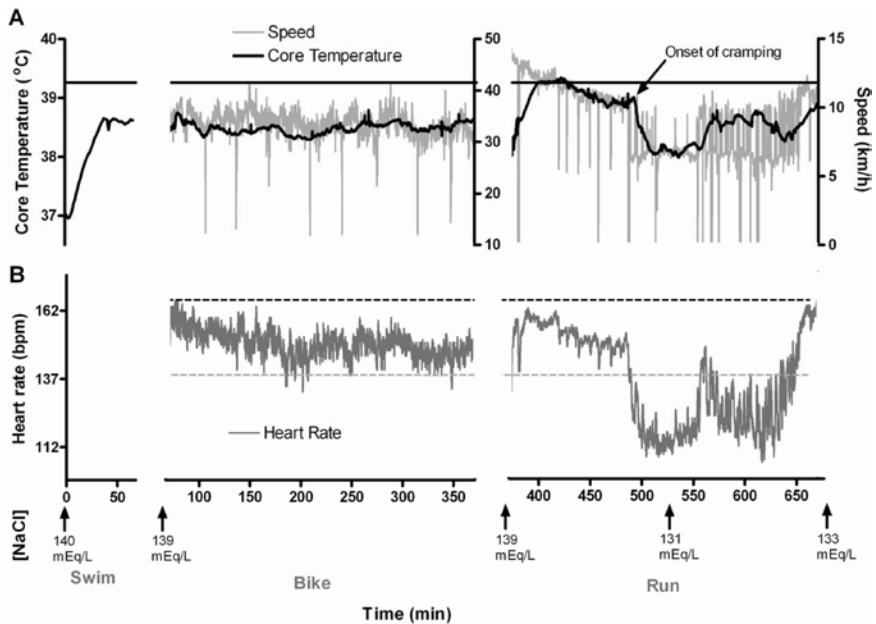


Figure 1 — Core temperature, speed (A), heart rate (B), and blood sodium concentration ([NaCl]) during the Ironman triathlon. The solid black line represents “critical” core temperature reached during laboratory time-to-exhaustion tests. The dashed black line represents heart rate at second ventilation threshold. The dashed gray line represents first ventilation threshold heart rate.

study is the first to show a decrease in running speed following attainment of a critically high T_{c} within a field setting. Another important finding was the matched response of T_{c} and running speed (Figure 1) despite only a 2% body mass loss following the race. This finding supports original observations made in marathon runners predicting metabolic rate and not hydration status as the main determinant of the T_{c} response.⁴ The fact that running speed slowed once T_{c} reached 39.4°C supports the notion that humans will lower or abolish motor output when T_{c} approaches “critical” levels.¹ Thus, the athlete in this study did not appear to select an appropriate pace at the beginning of the run in response to the rate of rise in T_{c} , as previously suggested.⁵

The second important finding was the symptomatic hyponatremia observed at 21 km of the marathon run. From this point until the finish, the athlete reported experiencing vertigo and cephalalgia while recording blood sodium levels ranging from 128 to 133 mEq·L⁻¹. As described by Beltrami et al,³ the usual cause of exercise-associated hyponatremia is the overconsumption of fluids. While estimated fluid volumes consumed during the cycle phase (1.71 L·h⁻¹) exceeded this athlete's sweat rate (1.42 L·h⁻¹), and therefore could have contributed, this athlete drank throughout the race according to his thirst.³ Another possible cause of the hyponatremia observed after the midpoint of the marathon run was a heat- and/or exercise-induced oversecretion of arginine vasopressin (AVP).⁶ Thus, the peak high core temperature (39.4°C) that likely caused the triathlete to slow may also have stimulated a nonosmotic AVP secretion, leading to fluid retention and a dilutional hyponatremia. This presumption is supported by both the increased post-race urine osmolality (619 mOsm·kg⁻¹) despite low blood sodium levels (128–133 mEq·L⁻¹), as well as the high aldosterone levels (157.7 ng·dL⁻¹) acting to preserve blood sodium. Interestingly, this same athlete experienced altitude sickness 10 mo after this Ironman event during an ascent to 5,500 m in the Himalayas, a relationship recently proposed by Ayus and Moritz.⁷

The positive pacing strategy chosen by this triathlete during both cycle and run phases is typical of ultraendurance racing.⁸ As shown in Figure 1, at commencement of both the cycle and run phases, when energy stores were presumed to be relatively high and T_{c} relatively low, exercise intensity was selected at a rate higher than what was able to be maintained throughout either phase. This chosen pace was below the second ventilatory threshold heart rate but above the first ventilatory threshold heart rate, and was lowered considerably during the run phase once critical T_{c} levels were reached (Figure 1). It is interesting to speculate how a lowered pace earlier on might have contributed to an improved performance. Indeed, had pace been lowered sooner in the run phase, the critical T_{c} attained might not have been reached, which may have prevented the likely nonosmotic oversecretion of AVP, and possibly delayed the onset of cramp occurrence in this athlete.

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