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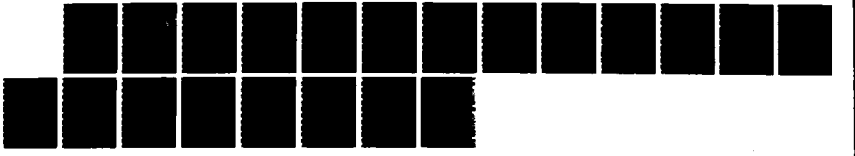

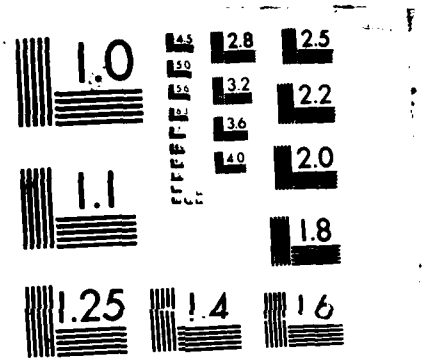

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A 32 year-old male (S.H.), monitored during an 8-day heat acclimation (HA) investigation unexpectedly exhibited heat intolerance and heat exhaustion. Thirteen other males completed HA without indications of either heat intolerance or heat exhaustion. Because S.H. responded normally to HA on days 1 - 4, the intervention of an unknown host factor on days 5 - 8 was suggested. S.H.'s heat exhaustion episode (day 8) was apparently forewarned by loss of body weight and increased ΔHR, ΔT_{sk} (days 5 - 8) and ΔT_{re} (days 7 - 8) during daily 90 min trials. His symptoms indicated classical salt depletion heat exhaustion, but the calculated salt deficit ($<0.1 g\ NaCl \cdot kg^{-1}$ body weight) was mild. Post-heat exhaustion serum enzyme levels were either normal (ALT, AST) or acutely elevated (CPK). Blood beta-endorphin and cortisol levels were 6 times and 2 times greater than control values, respectively. This case report is unique because physiological measurements and blood analyses were performed before, during, and after heat intolerance and heat exhaustion.

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Heat Intolerance, Heat Exhaustion Monitored:

A Case Report

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[4 tables, 0 figures]

Abstract

A 32 year-old male (S.H.), monitored during an 8-day heat acclimation (HA) investigation, unexpectedly exhibited heat intolerance and heat exhaustion. Thirteen other males completed HA without indications of either heat intolerance or heat exhaustion. Because S.H. responded normally to HA on days 1 - 4, the intervention of an unknown host factor on days 5 - 8 was suggested. S.H.'s heat exhaustion episode (day 8) was apparently forewarned by loss of body weight and increased ΔHR, ΔT_{sk} (days 5 - 8) and ΔT_{re} (days 7 - 8) during daily 90 min trials. His symptoms indicated classical salt depletion heat exhaustion, but the calculated salt deficit (< 0.1g $NaCl \cdot kg^{-1}$ body weight) was mild. Post-heat exhaustion serum enzyme levels were either normal (ALT, AST) or acutely elevated (CPK). Blood beta-endorphin and cortisol levels were 6 times and 2 times greater than control values, respectively. This case report is unique because physiological measurements and blood analyses were performed before, during, and after heat intolerance and heat exhaustion.

KEY WORDS: heat acclimation, exercise; sodium; potassium; plasma volume; rectal temperature; heart rate, sweat; serum enzymes; beta-endorphin; aldosterone; plasma renin; cortisol

Introduction

Heat intolerance is defined as the inability to adapt to work in a hot environment (10). Although temporary heat intolerance has been reported (8), controlled studies which reveal the course and/or cause(s) of heat intolerance do not exist. Similarly, studies describing the hematological and biochemical status of patients at the onset of heat exhaustion or heat stroke do not exist. Our understanding of these heat illnesses has been derived primarily from prospective studies and anecdotal reports. For these reasons, the following case report is presented. It involves observations made on a 32 year old male during an eight-day heat acclimation (HA) investigation. This subject unexpectedly exhibited classical heat exhaustion symptoms (day 8) and a gradual degradation of physiological responses (days 5 - 8) which we defined as heat intolerance. Because (a) he was monitored on successive days of heat exposure, (b) blood chemistry and hematological measurements were performed before and after the heat exhaustion episode, and (c) values of 13 normal subjects were recorded for comparison, this report clarifies heat intolerance, heat exhaustion, and the possible relationships between them.

Methods

At the time of these observations, subject S.H. was undergoing HA trials as a part of laboratory investigations; the techniques of these HA trials have been published elsewhere in detail (2). Prior to this study, S.H. had no known disease or metabolic disorder and had not experienced any form of heat illness. S.H. (age - 32 yr, height - 180 cm, weight - 110.47 kg, VO_2max - 41.40 $ml \cdot kg^{-1} \cdot min^{-1}$) was unacclimatized at the onset of eight

days of HA in a climatic chamber maintained at 41.2 ± 0.3 °C dry bulb, 39.0 ± 1.7 % RH. Thirteen other healthy males (mean ± SE: age - 28.4 ± 1.9 yr, height - 177 ± 2 cm, weight - 79.771 ± 3.784 kg, VO_2max - 45.74 ± 1.96 $ml \cdot kg^{-1} \cdot min^{-1}$) also completed this protocol. Daily 100 min HA trials consisted of intermittent treadmill running during nine exercise-rest periods of 2-10 min duration (2). Heart rate (HR), rectal temperature (T_{re}) and mean weighted skin temperature from three sites (T_{sk}) were monitored throughout each trial. If HR exceeded 180 $beats \cdot min^{-1}$ or if T_{re} exceeded 39.5°C, the trial was terminated. Treadmill speeds each day were selected by subjects (without knowledge of treadmill speed, HR, T_{re}, or T_{sk}), except that treadmill speeds selected on day 1 were duplicated on day 8. Total distance run each day ranged from 5.426 to 8.565 km. Water was consumed ad libitum during HA trials, and S.H. was encouraged to drink large quantities of water (not measured) during non-trial hours. Sweat rate ($g \cdot m^{-2} \cdot h^{-1}$) was calculated by using pre- vs post-exercise body weight differences. S.H. maintained a food consumption record for three days at the beginning of HA and three days at the end of HA. S.H. consumed an average of 197 mEq (4.52 g) of sodium (Na+) per 24 h; this was equivalent to 11.5 g $NaCl \cdot 24\ h^{-1}$.

All blood samples were taken from an antecubital vein. Change in plasma volume (ΔPV%) was calculated from triplicate microhematocrit and hemoglobin (Hycel, Houston, TX) measurements, using the method of Dill and Costill (5). Plasma samples were analyzed in duplicate for the immunoreactivities of cortisol (New England Nuclear, North Billerica, MA), beta-endorphin (Immuno Nuclear Corp, Stillwater, MN), aldosterone (International CIS, Cedex, France), renin activity (New England Nuclear), as

well as Na+ and K+ concentrations via flame photometry (Rainin Instruments, Woburn, MA).

Results

Because day 1 and day 8 trials were identical, a comparison of these trials (Table 1) clarified the physiological adaptations made during HA. S.H. was the only volunteer who experienced extreme difficulty in completing the day 8 trial, which he had previously completed on day 1. Treadmill speeds were decreased on days 7 and 8, at his request. He was removed from the climatic chamber during exercise period 9 on day 8 because T_{re} exceeded the predetermined guideline of 39.5°C and because he displayed classical symptoms of heat exhaustion: vomiting, fatigue, muscular weakness and abdominal cramps. S.H. had experienced no other heat illness symptoms during the previous 7 days of heat exposure.

TABLE 1

Table 1 describes physiological measurements recorded during the eight days of HA. During HA, two days of rest (without HA trials) were inserted between day 4 and day 5, to simulate a weekend without exercise-heat exposure for all subjects. The entering body weight of S.H. (Table 1) reflected this two day rest, in that he gained 2.39 kg between days 4 and 5. He then lost 5.44 kg body weight during the next 72 h (day 5 to day 8). The ΔHR, ΔT_{re} and ΔT_{sk} decreased from day 1 - 4, but tended to increase on days 5 to day 8 (ΔHR and ΔT_{sk}) and days 7 - 8 (ΔT_{re}). The sweat rate nearly doubled from day 1 to day 8 (408 vs 803 $g \cdot m^{-2} \cdot min^{-1}$), but much of this increase probably resulted from the increased central drive for sweating (T_{re}) on day 8.

TABLE 2

Table 2 presents pre-exercise and post-exercise plasma constituents of S.H. on days 1, 4 and 8 of HA. The ΔPV% of S.H. on day 8 was 5%

TABLE 3

greater than on day 1. Table 3 describes the day 1 and day 8 values of the other subjects who completed this protocol (n = 13); these subjects responded as expected, during HA trials.

TABLE 4

Table 4 presents serum enzyme concentrations and plasma beta-endorphin immunoreactivity of S.H. on day 8. Of the three serum enzymes measured, only CPK was outside the normal range (2 - 3 min post-exercise). Day 8 post-heat exhaustion beta-endorphin levels were elevated 6 - 9 times above the levels measured on days 1 and 4.

Discussion

HEAT INTOLERANCE

The physiological measurements of S.H. indicated that his trials on days 1 - 4 progressed much like the trials of 13 other males. The deterioration of HA adaptations (Table 1) in S.H., however, did not follow the usual course of HA events (Table 3); this deterioration was observed in body weight, ΔHR and ΔT_{sk} (days 5 - 8) before it was observed in ΔT_{re} (days 7 and 8) or ΔPV% (day 8). These events are defined herein as heat intolerance, using a modification of Strydom's definition (10). Many other factors (e.g. VO_2max, surface area-to-mass ratio, age) have been correlated with heat intolerance, but none of these are likely explanations because S.H. responded normally to heat exposure until day 5 (Table 1). This fact implies that an unknown host factor intervened between days 5 and 8. The impact of a bacterial/viral illness on heat tolerance has been described by Assia et al. (4) and Keren et al. (8), but a pre-exercise fever was not

measured in S.H. before any HA trial. The possible role of accumulated fatigue or energy depletion (e.g. glycogen depletion) in S.H. are recognized.

HEAT EXHAUSTION

Marriott (9) utilized anecdotal observations to divide salt depletion heat exhaustion into three clinical grades: early, moderately severe and very severe. Early salt depletion (deficit of 0.5 g NaCl·kg body weight^{-1}) was associated with fatigue, dizziness, and negligible urinary chloride. The symptoms of moderately severe salt depletion (deficit of 0.5 - 0.75 g NaCl·kg body weight^{-1}) were described as fatigue, dizziness, negligible urinary chloride, nausea, cramps and perhaps vomiting. Very severe salt depletion (deficit of more than 0.75 g NaCl·kg body weight^{-1}) was associated with all of the above symptoms plus systolic hypotension and possibly oligaemic shock. Marriott also examined pure water *depletion* heat exhaustion. He divided this disorder into into three clinical grades: <u>early</u> (deficit of 2 % body weight) which involves thirst; <u>moderately severe</u> (deficit of 6 % body weight) which is typified by intense thirst, dry mouth, scanty urine and rapid pulse; and <u>very severe</u> (deficit of 7 % body weight) which includes all of the above symptoms plus impairment of mental and physical capacities.

The symptoms which S.H. exhibited on day 8 of HA (e.g. vomiting, fatigue, muscular weakness and abdominal cramps) were indicative of moderately severe salt depletion heat exhaustion, and not primary water depletion heat exhaustion, as described by Marriott. In calculating S.H.'s actual Na+ and NaCl deficit, it was assumed that 20 per cent of body weight was extracellular fluid (ECF). Therefore, the day 1 ECF of S.H. (110.47 kg body weight) equalled 22.09 L and the day 8 ECF equalled 21.37 L. Using the pre-exercise plasma Na+ values for day 1 and day 8 (141

and 138 $mEq \cdot L^{-1}$, respectively), it was calculated that the 8-day Na+ deficit which S.H. experienced was 166 mEq Na+ (3.8 g Na+). This 8-day deficit was equivalent to 9.7 g NaCl (< 0.1 g $NaCl \cdot kg^{-1}$ body weight)--considerably lower than Marriott's value for moderately severe salt depletion heat exhaustion. We conclude that either Marriott's salt deficit levels require refinement or the symptoms associated with salt and/or water depletion heat exhaustion must be reexamined. In support of this conclusion, observations made during field studies (Hubbard, unpublished observation) also suggest that Marriott's heat exhaustion categories are invalid because patients often present symptoms of moderately severe salt depletion heat exhaustion, yet are not clinically salt depleted.

SERUM ANALYSES

The serum enzyme concentrations following heat exhaustion (Table 4) indicated no transaminase values outside the normal range and little or no liver damage. CPK levels, however, were slightly elevated to 152.5 $U \cdot L^{-1}$, at 2 - 3 min post-exercise. This elevation was considerably higher than the same time point on day 1 (39.79 $U \cdot L^{-1}$) and day 4 (29.41 $U \cdot L^{-1}$). Because CPK is not present in the liver, we speculate that this CPK elevation originated primarily in skeletal muscle. Records of 3 heat stroke patients (Armstrong, unpublished observation) indicate that CPK may peak 4 days following severe heat injury and that ALT and AST may peak between 2 - 5 days after such an event. Therefore, it is possible that S.H. experienced ALT and AST elevations several days after the final time point in Table 4 (not measured).

It has been reported that blood beta-endorphin levels are valid markers of relative exercise-heat stress (7). The pre-exercise vs post-exercise beta-

endorphin levels of S.H. on days 1, 4 and 8 support this observation (Table 4). The post-heat exhaustion levels of beta-endorphin on day 8 were 6 - 9 times greater than comparable post-exercise levels on days 1 and 4. Similarly, the cortisol level following heat exhaustion (Table 2, day 8)--an index of general stress--was more than twice the comparable day 1 and day 4 cortisol concentrations. Appenzeller et al. (1) recently reported elevated beta-endorphin levels in 18 heat stroke patients who participated in the Hajj desert pilgrimage to Mecca in 1982. The present case study, to our knowledge, is the first report of elevated beta-endorphin concentrations following heat exhaustion. The level which S.H. reached (263.3 $pg \cdot ml^{-1}$) was considerably lower than the mean post-heat stroke level published by Appenzeller et al. (1073 ± 428 $pg \cdot ml^{-1}$), although the values in that study ranged from 58 - 5000 $pg \cdot ml^{-1}$. The importance of circulating beta-endorphin following severe heat injury is emphasized by these data and a potential therapy utilizing opioids is suggested.

FLUID-ELECTROLYTE BALANCE

Cardiovascular insufficiency during strenuous exercise in the heat, as evidenced by ΔHR and ΔPV% played a role in the heat exhaustion symptoms observed in S.H. His PV loss on day 8 (-16.2 %) was 51 % greater than on day 1 (-10.7 %). In contrast, the 13 males who underwent HA showed improvements in HR and ΔPV% from day 1 to day 8 (Table 3). The findings of Armstrong et al. (3) clarify this observation. They observed 9 healthy males during two 8 day HA protocols (via treadmill walking), while consuming either a high Na+ diet or a low Na+ diet (L). L resulted in significantly lower PV and significantly higher HR and T_{re} on days 3 - 6 of HA, in a manner similar to S.H. on days 5 - 8 (Tables 1

and 2). These researchers concluded that subjects were at greater risk of heat injury during days 3 - 6 of L. Interestingly, L (98 mEq Na+·24 h^{-1}) resulted in a mean 8-day Na+ deficit of -230 mEq Na+·8 d^{-1}, compared to S.H.'s calculated 8-day deficit of -166mEq Na+·8 d^{-1} (above). In addition, Armstrong et al. (3) observed significant changes in muscle water and muscle Na+ during L; this implied altered membrane permeability.

THE ENERGY-DEPLETION MODEL

In a recent review (6), we hypothesized that at least three factors commonly associated with exercise in the heat (e.g. hyperthermia, dehydration and exercise-induced acidosis) superimpose an energy drain upon all the cells of the body, via increased sodium pump activity. First, hyperthermia increases the kinetic energy of ions in solution, increases diffusion, increases the permeability of the cell to Na^+ ions, and thereby stimulates the sodium pump. Second, dehydration stimulates sodium pump activity because the rate of net sodium diffusion is proportional to the concentration outside the cell minus the concentration inside the cell. Third, exercise-induced intracellular acidosis stimulates a Na^+ - H^+ exchange across the cell membrane that, while regulating intracellular pH, further increases intracellular Na^+. We hypothesized that such an increase in intracellular sodium stimulates cell swelling, the Na^+ pump, heat production and probably contributes to fatigue. A reduction in metabolic efficiency, subsequent to an increase in sodium permeability, also is hypothesized by this model. Named the Energy-Depletion Model, this concept presents the rate of body heat gain, increases in membrane permeability, cellular energy depletion, and reduced endurance capacity as a vicious cycle. A unique feature of this Energy Depletion Model is that it depicts heat injury as an

insult to all cells of the body, and not solely as localized damage to thermoregulatory centers, in agreement with empirical observations.

The dehydration observed as weight loss in S.H. (days 5 - 8) can be interpreted as one event contributing to or initiating this vicious cycle. The heat exhaustion data of day 8 (Table 1) may be explained by increased heat storage and decreased work efficiency due to altered membrane permeability. If S.H.'s calculated 8-day deficit of -166 mEq Na^+ (see above) were distributed throughout the intracellular space (38.5 L), intracellular sodium concentration (14 mEq/L) would increase 1.3 fold (18.3 mEq/L). Since membrane pumping activity increases approximately as the third power of the sodium concentration, sodium pump activity and its rate of ATP hydrolysis would increase 2.2 fold. This energy drain is hypothesized to have contributed to the faster onset of fatigue on day 8, despite a lower work rate and a greater sweat rate. The imbalance in heat gain versus heat loss (ΔT_{re} in Table 1) is a matter of speculation, but an increase in anaerobic metabolism has not been ruled-out.

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The views, opinions, and/or findings contained in this report are those of the authors and should not be construed as official Department of the Army position, policy or decision, unless so designated by other official documentation.

Human subjects participated in these studies after giving their informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 for use of volunteers in research.

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References

1. Appenzeller O, Khogali M, Carr DB, Gumma K, Mustafa MKY, Jamjoom A, Skipper B. Makkah Hajj: heat stroke and endocrine responses. Ann. of Sports Med. 1986; 3:30-32.

2. Armstrong LE, RW Hubbard, JP DeLuca, EL Christensen. Self-paced heat acclimation procedures. Natick, MA: USARIEM, Technical Report No. T8/86, 1986.

3. Armstrong LE, Costill DL, Fink WJ. Changes in body water and electrolytes during heat acclimation: effects of dietary sodium. Aviat. Space Environ. Med. 1987; 58:143-48.

4. Assia E, Epstein Y, Shapiro Y. Fatal heat stroke after a short march at night: a case report. Aviat. Space Environ. Med. 1985; 56:441-442.

5. Dill DB, Costill DL. Calculation of percentage changes in volumes of blood, plasma and red cells in dehydration. J. Appl. Physiol. 1974; 37:247-248.

6. Hubbard RW, Matthew CB, Durkot MJ, Francesconi RP. Novel approaches to the pathophysiology of heat stroke: the energy depletion model. In: Proceedings of the Fourth Annual UA/EM-IRIEM Research Symposium: Environmental Emergencies, February 24, 1987. In press.

7. Kelso TB, Herbert WG, Gwazoawskas FC, Goss FL, Hess JL. Exercise-thermoregulatory stress and increased plasma beta-endorphin/beta-lipotropin in humans. J. Appl. Physiol. 1984; 57:444-449.

8. Keren G, Epstein Y, Magazanik A. Temporary heat intolerance in a heat stroke patient. Aviat. Space Environ. Med. 1981; 52:116-117.

9. Marriott HL. Water and Salt Depletion. Springfield, IL: Charles C. Thomas, 1950.

10. Strydom NB. Heat intolerance: It's detection and elimination in the mining industry. South African J. Sci. 1980; 76:154-156.

TABLE 1 - Physiological measurements of S.H. during daily heat acclimation trials.

MEASUREMENT (UNIT)	EXPERIMENTAL DAYS 1	2	3	4	5	6	7	8
Entering body weight (kg)	110.47	109.68	109.58	109.90	112.29	109.40	107.89	106.85
Entering urine specific gravity	1.024	1.021	1.023	1.024	1.024	1.026	1.026	1.022
Trial duration (min)	100	100	100	100	100	100	100	98 *
Distance run ($km \cdot 100\ min^{-1}$)	8.565	8.061	7.595	8.015	8.411	8.792	7.685	5.426+
ΔHR ($beats \cdot min^{-1}$)	79	81	75	65	84	88	77	96
ΔT_{re} (^{o}C)	+1.81	+2.27	+1.79	+1.79	+1.68	+1.67	+2.16	+2.45
ΔT_{sk} (^{o}C)	+3.42	+1.06	+1.19	+1.59	+1.97	+2.79	+1.99	+2.08
Sweat rate ($g \cdot m^{-2} \cdot h^{-1}$)	408	662	582	776	588	673	873	803

* - trial stopped prematurely

+ - treadmill speeds voluntarily reduced by S.H.

TABLE 2 - Pre- and post-exercise plasma constituents of S.H. on days 1, 4 and 8 of heat acclimation.

MEASUREMENT (UNIT)	EXPERIMENTAL DAYS					
	1		4*		8	
	PRE-EX	POST-EX	PRE-EX	POST-EX	PRE-EX	POST-EX
Hematocrit (%)	46	48	---	---	47	51
Change in PV during exercise (ΔPV%)		-10.7		---		-16.2
Na+ ($mEq \cdot L^{-1}$)	141	139	140	142	138	141
K+ ($mEq \cdot L^{-1}$)	4.2	4.6	4.5	4.7	4.3	4.7
Plasma renin activity ($ng \cdot ml^{-1} \cdot h^{-1}$)	2.57	11.28	3.82	13.25	9.14	16.40
Aldosterone ($ng \cdot dl^{-1}$)	10.71	42.54	25.98	77.24	64.44	125.60
Cortisol ($ug \cdot dl^{-1}$)	8.30	11.71	8.63	10.02	9.55	25.98

* - day 4 involved treadmill speeds which were different from days 1 and 8

TABLE 3 - Day 1 and day 8 measurements (mean ± SE) of 13 males who underwent heat acclimation.

MEASUREMENT (UNITS)	EXPERIMENTAL DAYS 1	8	
Entering body weight (kg)	79.961 ± 3.761	79.575 ± 3.601	
Entering urine specific gravity	1.021 ± 0.022	1.022 ± 0.002	
Δ HR (beats·min^{-1})	82 ± 4	67 ± 6	*
Δ Tre (^{o}C)	2.17 ± 0.11	1.46 ± 0.19	*
Δ Tsk (^{o}C)	1.68 ± 0.21	1.29 ± 0.40	
Sweat rate (g·m^{-2}·h^{-1})	402 ± 35	380 ± 45	
Hematocrit (%) - pre-exercise	45 ± 1	45 ± 1	
- post-exercise	47 ± 1	45 ± 1	
Change in PV (Δ PV%)	- 7.1 ± 0.8	- 5.1 ± 1.1	
Plasma Na+ (mEq·L^{-1}) - pre-exercise	141 ± 1	140 ± 1	
- post-exercise	140 ± 3	141 ± 3	
Plasma K+ (mEq·L^{-1}) - pre-exercise	4.3 ± 0.1	4.4 ± 0.1	
- post-exercise	4.7 ± 0.1	4.8 ± 0.1	
Plasma renin activity - pre-exercise	1.30 ± 0.36	4.10 ± 0.70	*
(ng·ml^{-1}·h^{-1}) - post-exercise	12.70 ± 4.90	9.70 ± 1.17	*
Plasma aldosterone - pre-exercise	15.75 ± 2.81	28.16 ± 5.50	*
(ng·dl^{-1}) - post-exercise	80.01 ± 9.05	87.17 ± 12.96	
Plasma cortisol - pre-exercise	13.60 ± 2.72	11.67 ± 1.39	
(ng·dl^{-1}) - post-exercise	20.01 ± 2.08	14.65 ± 2.04	*

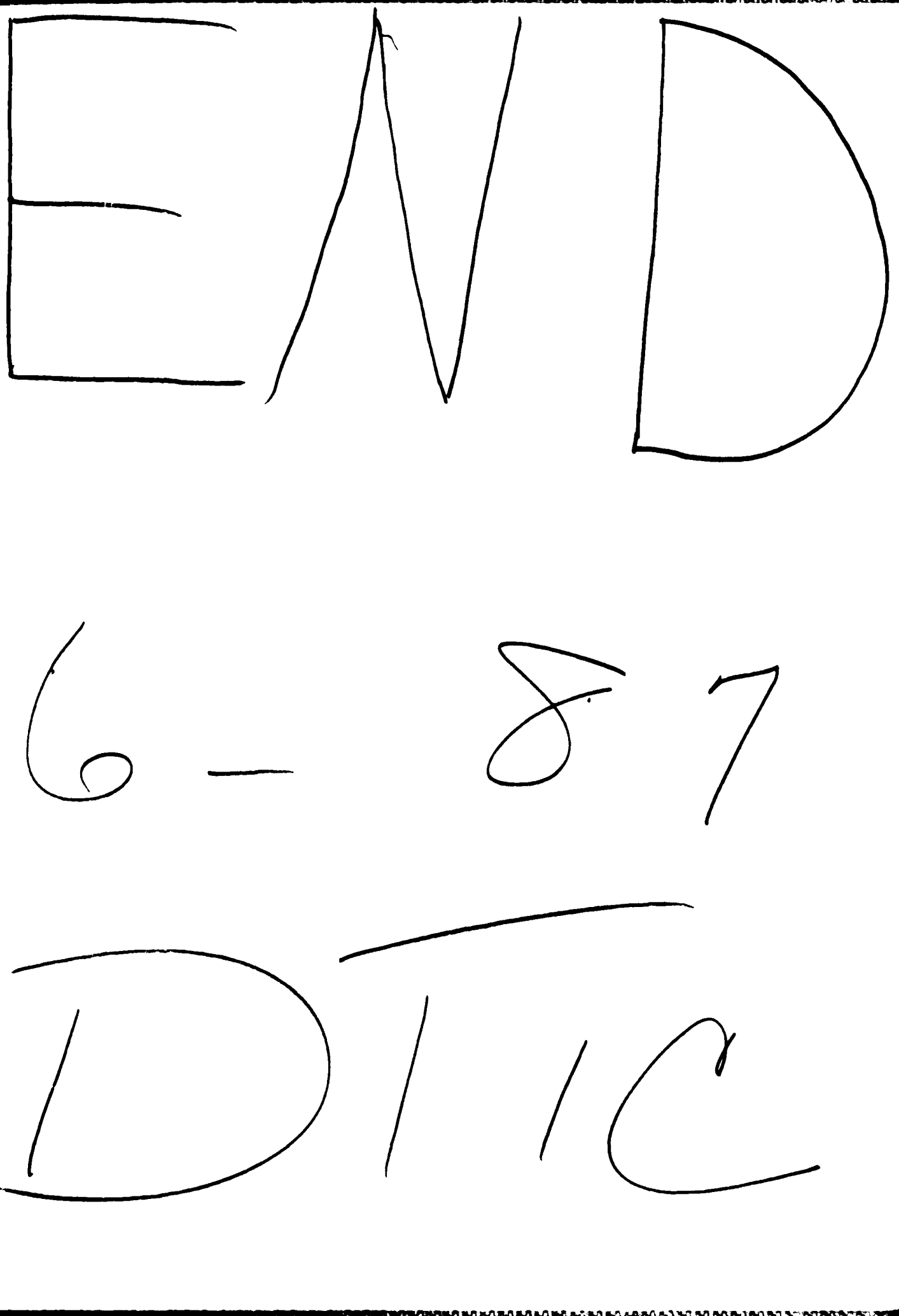
* - $p < .05$ when compared to day 1

TABLE 4 - Serum enzyme concentrations and plasma beta-endorphin immunoreactivity of S.H. on day 8 of heat acclimation trials.

MEASUREMENT (UNIT)	NORMAL RESTING RANGE	PRE-EXERCISE CONTROL	2-3 min POST EXERCISE	5.5 h POST EXERCISE	21 h POST EXERCISE
ALT ($U \cdot L^{-1}$)	8-30	7.8	23.5	14.8	9.6
AST ($U \cdot L^{-1}$)	7-27	14.1	20.7	16.9	14.2
CPK ($U \cdot L^{-1}$)	20-134	122.0	152.5 *	118.6	109.4
Beta-endorphin ($pg \cdot ml^{-1}$)	9-40	13	263 *+	---	---

* - outside normal range

+ - control and 2-3 min post-exercise beta-endorphin values were: 9 and 40 on day 1; 10 and 29 on day 4 of heat acclimation.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