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ACUTE HEAT/EXERCISE STRESS IN RATS: EFFECTS ON FLUID AND ELECTR--ETC(U)
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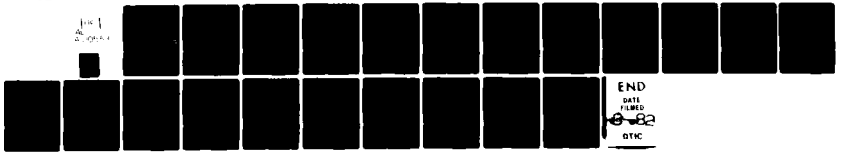

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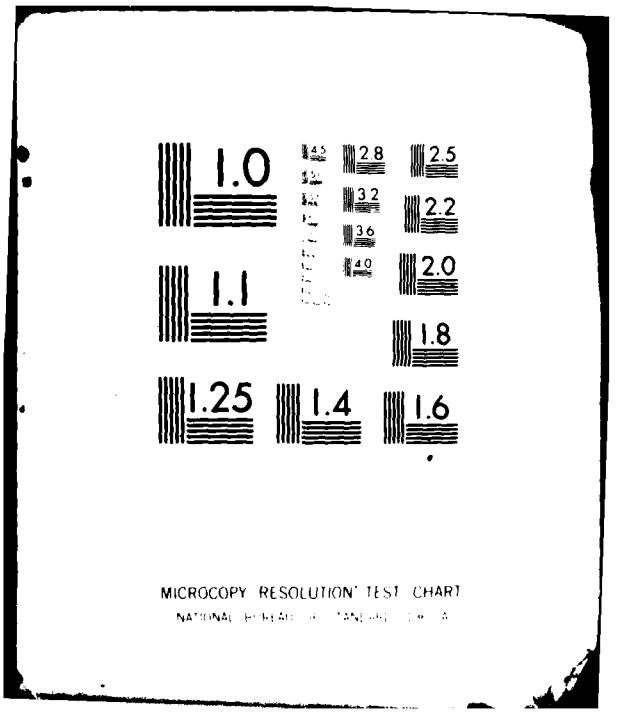
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Adult, male rats were exercised (level treadmill, 9.14 m/min) in the heat (35°C, 30-35% rh) until hyperthermic exhaustion (Tre = 42.5°-43°C) ensued. Blood samples (0.3 ml) were taken immediately prior to exercise in the heat (time 0), when Tre reached 40°C (8.25 min treadmill time), when hyperthermic exhaustion occurred, and again when Tre decreased to 40°C. Plasma was separated and assayed for aldosterone, angiotensin I (plasma renin activity), and antidiuretic hormone (arginine-vasopressin). Plasma aldosterone levels were significantly ($p < .001$) elevated after 8 min of exercise and remained

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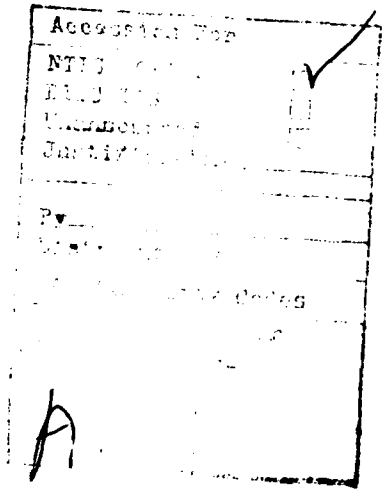
increased throughout the exercise and recovery periods. Alternatively, plasma angiotensin I levels were unaffected during exercise, but increased significantly ($p < .001$) during the recovery period. Arginine-vasopressin concentrations were likewise significantly ($p < .05$) elevated by completion of the exhaustive run and continued to increase during the recovery period. We attributed these elevations in levels of the fluid and electrolyte regulatory hormones to part of an adaptational response to maintain or increase plasma volume during exercise in the heat. Significant ($p < .001$) decrements in hematocrit ratio occurred during the exercise interval. However, we were unable to relate the ultimate ability of the animal to survive the heat/exercise-induced injury to the intensity of its hormonal response pattern to exercise in the heat.



Acute Heat/Exercise Stress in Rats: Effects on Fluid and Electrolyte Regulatory Hormones

Ralph Francesconi and Milton Mager

Running Title: Heat/exercise stress: hormonal responses

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Abstract

Adult, male rats were exercised (level treadmill, 9.14 m/min) in the heat (35°C, 30-35% rh) until hyperthermic exhaustion (T_{re} = 42.5° - 43°C) ensued. Blood samples (0.3 ml) were taken immediately prior to exercise in the heat (time 0), when T_{re} reached 40°C (8.25 min treadmill time), when hyperthermic exhaustion occurred, and again when T_{re} decreased to 40°C. Plasma was separated and assayed for aldosterone, angiotensin I (plasma renin activity), and antidiuretic hormone (arginine-vasopressin). Plasma aldosterone levels were significantly ($p < .001$) elevated after 8 min of exercise and remained increased throughout the exercise and recovery periods. Alternatively, plasma angiotensin I levels were unaffected during exercise, but increased significantly ($p < .001$) during the recovery period. Arginine-vasopressin concentrations were likewise significantly ($p < .05$) elevated by completion of the exhaustive run and continued to increase during the recovery period. We attributed these elevations in levels of the fluid and electrolyte regulatory hormones to part of an adaptational response to maintain or increase plasma volume during exercise in the heat. Significant ($p < .001$) decrements in hematocrit ratio occurred during the exercise interval. However, we were unable to relate the ultimate ability of the animal to survive the heat/exercise-induced injury to the intensity of its hormonal response pattern to exercise in the heat.

Key Words: angiotensin, aldosterone, vasopressin

Introduction

In several earlier reports from our laboratory we had demonstrated (12-14) in rats that a combined regimen of moderate exercise (9.14 m/min, level treadmill) and intense heat (35°C ambient) commonly elicited elevations in rectal temperatures (T_{re}) to 42.5° - 43°C. Generally, we observed that this range of T_{re} proved fatal in at least 50% of both experimental and control animals. In their earlier work on the development of a rat heatstroke model, Hubbard et. al. (20, 21) reported even higher mortality rates when T_{re} was held at these levels for longer periods. Because of the wide range of the effects of heat/exercise injury, we became interested in whether adaptational hormonal responses might be predictive of the rats' ability to withstand this extreme level of body temperature.

Acute and chronic exposure to environmental heat usually elicits substantial effects on fluid and electrolyte regulating hormones. Thus, it has been reported by various investigators that exposure of sedentary humans (1,8,11,22) to extreme heat stress has been characterized by significant increments in circulating levels of aldosterone, antidiuretic hormone (vasopressin), and renin-angiotensin. While Finbert et. al. have reported that both natural (10) and artificial (9) heat acclimatization attenuated the normal heat-induced elevation in plasma renin activity, Davies et. al. (7) found no effects of acclimatization on the increments of either plasma renin activity or aldosterone, but these responses were reduced by saline consumption. In an earlier study Braun et. al. (2) demonstrated that exogenous aldosterone administration to human test volunteers had significant effects on several indices of heat acclimatization during exercise in the heat.

Similar responsiveness of these hormones has been noted during exercise at more moderate environmental temperatures. Melin et. al. (26) observed significant increments in levels of aldosterone, renin activity, and

vasopressin in both trained and untrained men after exercise at 25°C. The same workers (15) demonstrated that levels of arginine-vasopressin and aldosterone were unaffected by training, but that resting levels of plasma renin activity were reduced by the training regimen. Convertino et. al. (5) attributed hypervolemic responses to exercise training to significant elevations in plasma renin activity and arginine-vasopressin during exercise. In an attempt to separate the thermal and exercise factors leading to these increments in plasma volume, the same workers (6) demonstrated greater exercise-induced hypervolemia than thermal-induced hypervolemia, partially attributable to larger increments in vasopressin responses to exercise.

Clearly, either thermal or exercise stress, singularly or in combination, is highly effective in eliciting general increments in circulating levels of these fluid regulatory hormones. Because of the apparent universality of these responses and the acknowledged importance (27,29,30) of body fluid adaptations in successfully withstanding extreme heat and exercise stress, we investigated the responses of plasma levels of aldosterone, renin activity, and vasopressin in heat/exercise injured rats. It was our intention not only to characterize further these responses during the actual time of heat exposure and exercise, but also to examine retrospectively the role of such effects on the ability of the animal to survive the heat/exercise injury.

Materials and Methods

Adult male rats (CD, Charles River Breeding Laboratories, Wilmington, MA 325-400 g) were used in all experiments. Upon arrival at the laboratory rats were kept in holding rooms in wire-bottomed cages (one animal/cage) at 22°C ± 1°C with free access to food (Charles River Laboratory Chow) and water. Automatic

fluorescent lighting (on, 0600-1800 hr) was maintained throughout, and entrained normal diurnal/nocturnal periodicities of body temperature. On the day prior to an experiment each animal was fitted with a permanent indwelling Silastic catheter in the external jugular vein for rapid, convenient blood sampling. Rats usually recovered from the surgery within 1-2 hrs, and no effects of surgery on the ability to exercise in the heat were noted.

Prior to experimentation a thermistor (Yellow Springs Instruments, Yellow Springs, OH) was inserted to a depth of 7 cm beyond the anal sphincter for monitoring core temperature (T_{re}); the probe was securely fixed in place so that its position was unaffected by exercise. Following this and immediately prior to commencing exercise in the heat, 0.3 ml blood was removed, hematocrit was measured, and the remaining blood was centrifuged (3-4^{o}C, 10000 g), the plasma removed, deep-frozen (-30^{o}C), and stored for subsequent assay. Following removal of the control blood sample (time 0), the rat was quickly transferred to a large stainless steel chamber (3m x 4m) which was maintained at 35^{o}C $\pm$ 0.5^{o}C, and the animal began treadmill exercise (9.14 m/min, 0 angle of incline). When the T_{re} reached 40^{o}C (approximately 8 min into the run) a second blood sample (also 0.3 ml) was taken and processed exactly as the first. The rat continued running until hyperthermic exhaustion (T_{re} = 42.5 - 43^{o}C, approximately 28 min on treadmill) ensued at which time the run was terminated and a third blood sample removed. The animal was then returned to a thermoneutral (28) environment (22^{o}C) and allowed to remain sedentary with continual monitoring of T_{re}. When T_{re} fell to 40^{o}C (approximately 34 min after the completion of the exercise), a final blood sample was obtained. It should be noted that because we wished to minimize the volume of blood taken at each sampling time, we repeated the entire experimental procedure for two separate groups of animals with an n of 15 in each group. Plasma samples of the first group were analyzed

for plasma renin activity and vasopressin while aldosterone was quantitated in the plasma collected from the second group; all experimental procedures were identical between groups.

Quantitative analyses of aliquots of the frozen plasma were performed for aldosterone, renin activity, and arginine-vasopressin. Aldosterone radioimmunoassay test kits were purchased from Damon Diagnostics, Needham, MA; the test kits were manufactured by International CIS, Sorin-Biomedica, Saluggia, Italy. The procedure used was that described in their technical bulletin; ordinarily 100 µl plasma was used for this assay. Angiotensin I (plasma renin activity) was analyzed using radioimmunoassay test kits manufactured by New England Nuclear, No. Billerica, MA, by methods described in their technical bulletin; for this assay 25 µl was required. Finally, arginine-vasopressin was assayed essentially by the methods of Hammer (18); ^{125}I arginine-vasopressin was purchased from New England Nuclear Corporation, and vasopressin antibody was obtained from Calbiochem, Inc. Generally, 25 µl plasma was used for this assay.

Statistical analyses were performed by a single factor analysis of variance; critical differences were established by Tukey's test (24,25). The null hypothesis was rejected at $p < .05$.

Results

Fig. 1 demonstrates the effects of exercise in the heat on plasma levels of aldosterone. Statistical analysis confirmed what is apparent from this graph: that after only 8.25 min (mean time required to achieve $T_{re} = 40^{\circ}C$) of exercise in the heat plasma levels of aldosterone rose significantly and remained elevated throughout the exercise and into the recovery period ($p < .001$, time 0 vs all other

sampling times). However, there were no differences noted in response levels among the latter 3 sampling intervals (50.4, 57.3, and 46.7 ng/dl, respectively, control = 14.9 ng/dl).

Mean plasma levels of angiotensin I are depicted in Fig. 2. The results indicate that during the actual treadmill run there were no significant changes in circulating angiotensin I levels. However, in the blood sample taken after completion of the treadmill run (recovery period) there did occur a highly significant ($p < .001$) increment in the hormone. This elevation represents an approximate 40% increase over the mean level recorded during the course of the treadmill run. The effects of exercise in the heat on plasma levels of arginine-vasopressin are illustrated in Fig. 3. Clearly, at the end of the treadmill run levels of vasopressin significantly ($p < .05$) increased and remained elevated ($p < .05$) into the recovery period. At the first sampling time during the treadmill run (8.25 min) no significant ($p > .05$) differences from time 0 control levels were observed.

Discussion

Results of the present investigation confirmed the efficacy of combined heat and exercise stress in eliciting generalized increments in levels of fluid and electrolyte regulatory hormones. Of particular interest was the significant ($p < .001$) elevation of plasma aldosterone levels after approximately 8 min of exercise in the heat. We attributed this acute response to heat/exercise stress to a generalized sympathicoadrenocortical response. Earlier, Collins and Weiner (4) reviewed the evidence that acute exposure to extreme heat stress resulted in increments in circulating levels of 17-hydroxycorticosteroids as a result of pituitary-adrenal stimulation. Later work (23) indicated that the adrenocortical

response to heat stress may be tempered by continued and repeated exposure to the heat stress. Evidence is also available which indicates that the adrenocortical response to exercise stress may be similarly reduced during habituation. This observation could be attributed to a reduction in the physiological stress of exercise concomitant with increases in physical fitness levels (19,32,34). Thus, the extremely rapid and intense increment in aldosterone levels in the current studies could be the result of the combined exercise and heat stress; in addition, these rats had not been previously exposed to either stress regimen.

In addition to its role in vasoconstriction, the renin-angiotensin system has been implicated in the control of aldosterone secretion (16). However, the current data clearly indicated that increments in aldosterone levels preceded responses in angiotensin I. This observation would also lend support to the hypothesis that the acute effects on aldosterone levels were due to a generalized sympathicoadrenocortical stress response.

Ordinarily, the current regimen, including mild exercise, intense heat, and short duration of the treadmill run, effects a hemodilution when post-run hematocrits are compared with pre-run hematocrits (13). Indeed, in the present study mean value for hematocrit ratio pre-run was 46.7 (SE = 0.4, n = 30) while for post-run blood samples the value dropped to 44.1 (SE = 0.4, n = 30, $p < .001$, paired t test). In a short duration experiment as this, these variations in hematocrit value may be related to circulating plasma volume (17,33). Thus, it appears reasonable to conclude that the observed alterations in hormonal levels were associated with a physiological response designed to maintain or increase plasma volume during exercise in the heat.

Another factor which should be considered in evaluating the present data is the effect of combined exercise and heat stress on the metabolic clearance rates

of these hormones. For example, in a controlled hyperthermia study Collins et. al. (3) reported that the metabolic clearance rate of aldosterone was reduced by 26% when body temperature was raised by 1.08°C. This was approximately equivalent to the reduction in hepatic blood flow. Much earlier, Rowell et. al. (29) had demonstrated large decrements in estimated hepatic blood flow, particularly during periods of strenuous physical activity. Thus, it is probable that the combined effects of heat exposure and exercise reduce even further the metabolic clearance rate of these hormones, and hence contribute to the acute and significant elevations generally observed in these experiments.

We have concluded from these studies that a regimen of exercise in a hot environment elicited significant increments in circulating levels of all three fluid and electrolyte regulatory hormones under investigation. The responses of these hormones may be partially responsible for the maintenance of plasma volume generally observed under these conditions. However, we were unable to relate the intensity of such hormonal responses with the ultimate survival of the animals. Thus, while attainment of T_{re} in the range of 42.5° - 43°C during exercise in the heat generally was effective in eliciting significant hormonal responses, these responses were unrelated to the intensity of the heat/exercise injury incurred.

Figure Legend

Figure 1 depicts the effects of exercise (9.14 m/min, level treadmill) in the heat (35°C) on plasma levels of aldosterone. Mean values ± standard errors of the mean are recorded for an n of 15. Times at which the blood samples were obtained are noted on the abscissa.

Figure 2 illustrates the effects of exercise (9.14 m/min, level treadmill) in the heat (35°C) on circulating levels of angiotensin I (plasma renin activity) (n = 15, mean ± SE). Times at which blood samples were drawn are noted on the abscissa.

Figure 3 displays the effects of exercise (9.14 m/min, level treadmill) in the heat (35°C) on circulating levels of arginine-vasopressin (antidiuretic hormone) (n = 15, mean ± SE). Times at which blood samples were drawn are noted on the abscissa.

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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 an official department of the Army position, policy, or decision, unless so designated by other official documentation.

In conducting the research described in this report, the investigators adhered to the Guide for Laboratory Animal Facilities and Care, as promulgated by the Committee on the Guide for Laboratory Animal Facilities and Care of the Institute of Laboratory Animal Resources, National Academy of Sciences-National Research Council.

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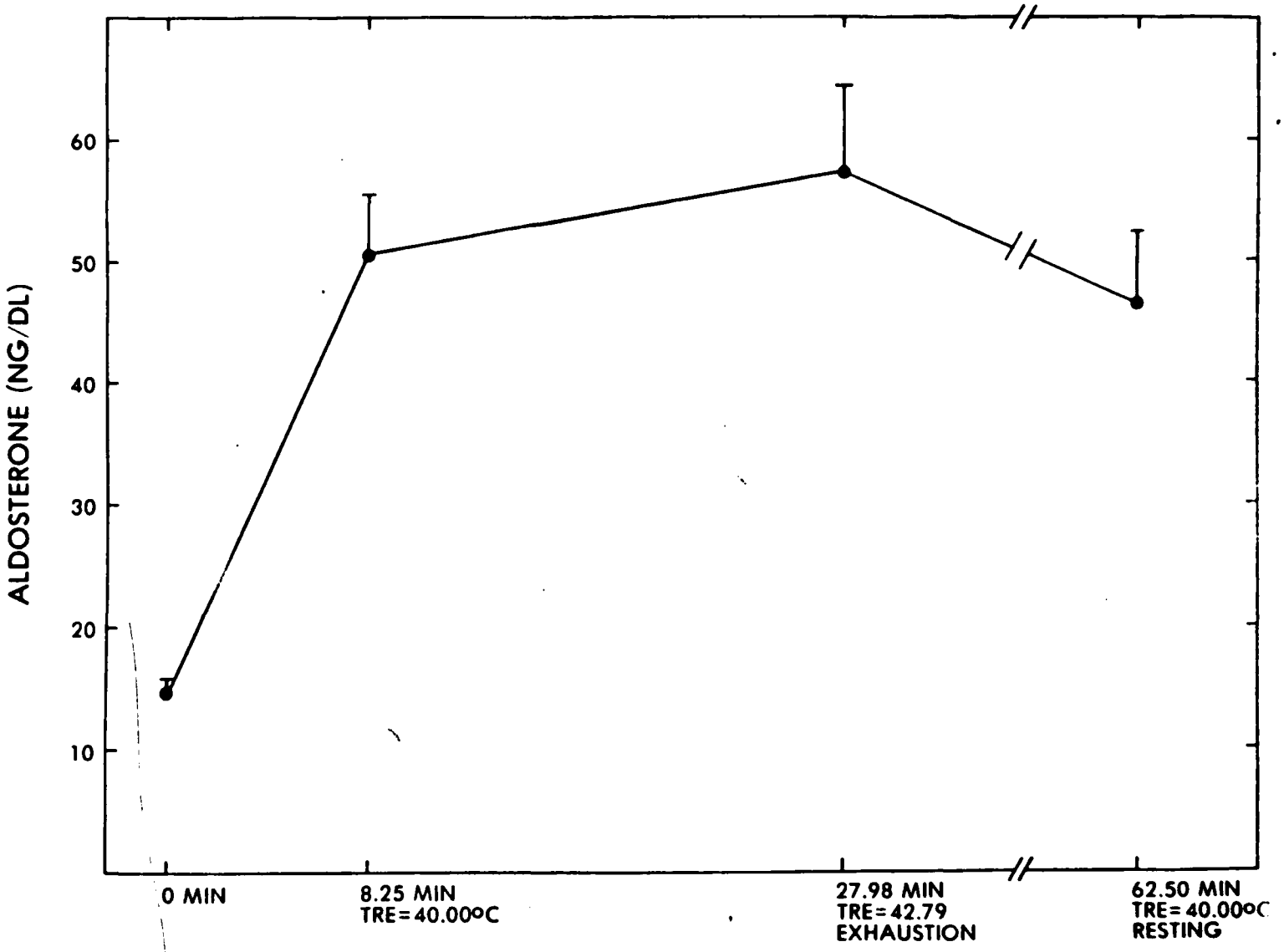
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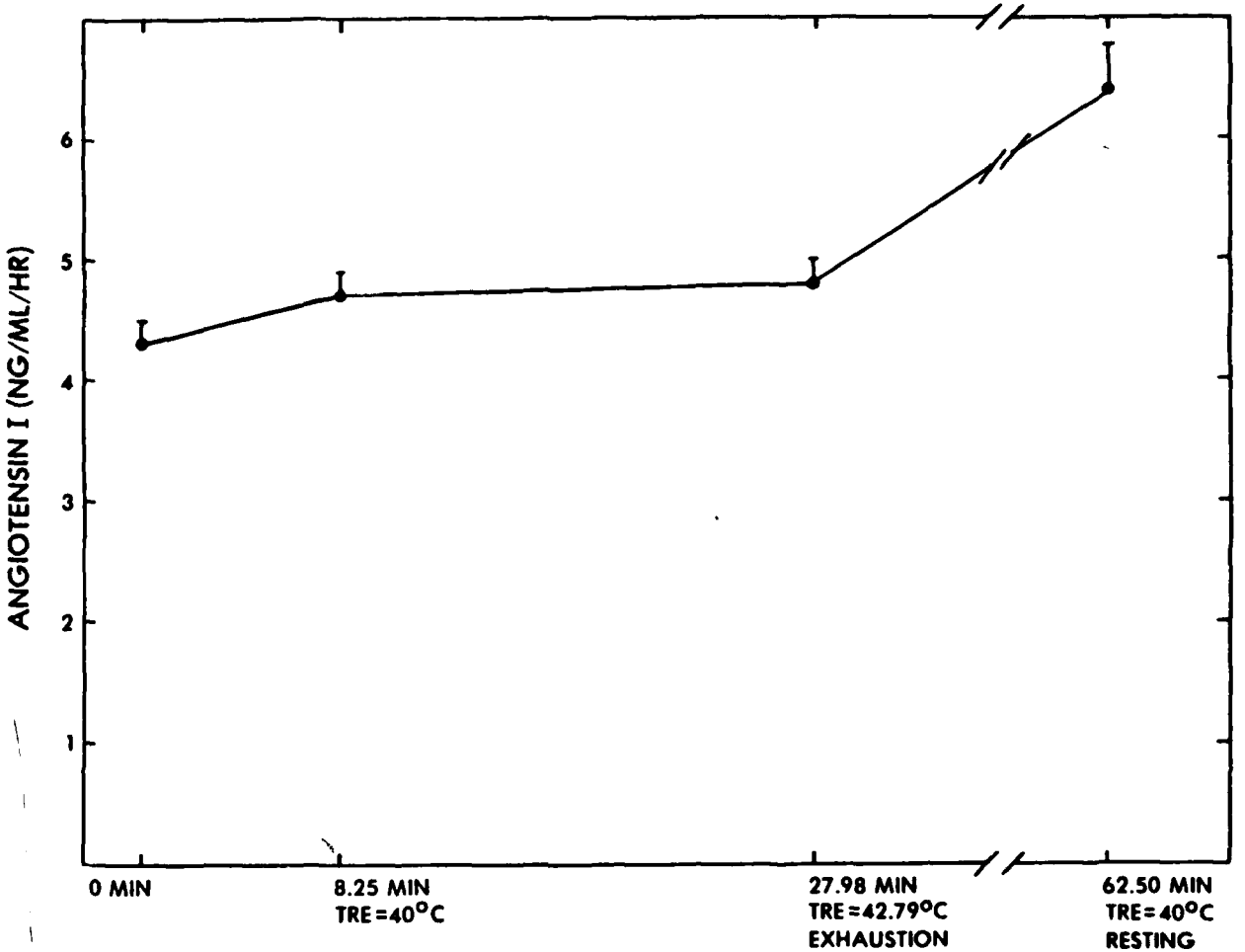

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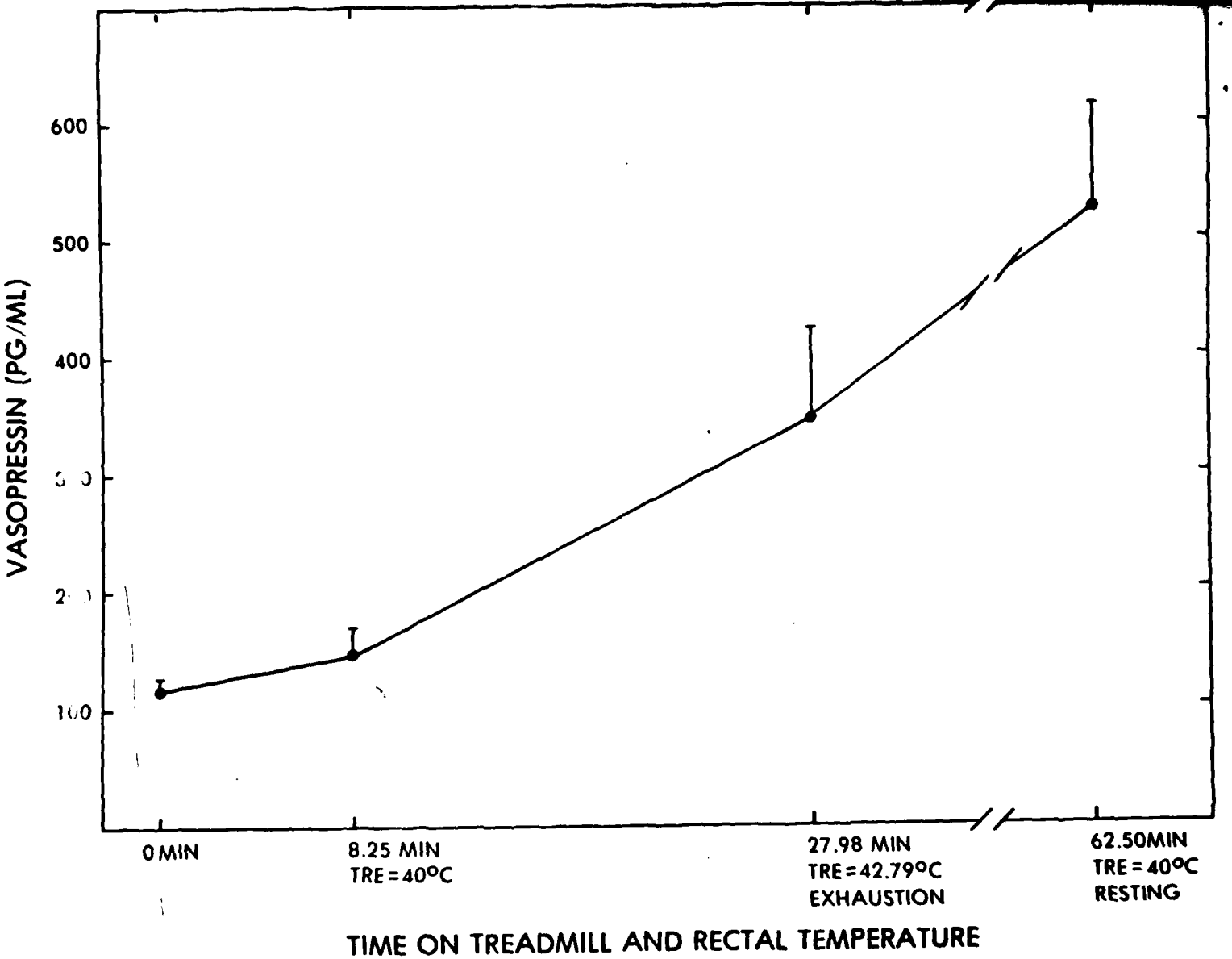

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