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Exertional Heatstroke: An International Perspective

An Introduction: The Role of Exercise
in the Etiology of Exertional Heatstroke

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Running Head: Introduction

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ABSTRACT

Exertional heatstroke usually occurs among healthy, fit individuals who are motivated to perform strenuous exercise because of peer pressure, discipline, or athletic competition. In fact, exertional heatstroke deaths among high school athletes in the United States (ranked third only to head and neck injuries and heart failures), and the reluctance of race directors to cancel or postpone mass participation road races during periods of severe heat and humidity, emphasize a continuing need for widespread dissemination of information. The purposes of this symposium are to (a) convey essential information to the members of ACSM who are involved in exercise prescription, patient care, or athletic training, and (b) disseminate recent scientific theories and research concerning exertional heatstroke.

Key words: heat acclimation, thermoregulation, hyperthermia, cardiovascular

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AN INTRODUCTION:

In non-heat acclimated humans, exercise in the heat produces an initial period of cardiovascular instability characterized by increases in body temperature, heart rate, plasma volume and decreased stroke volume (25). Following successful acclimation, there is a return to cardiovascular stability manifested by an increase in plasma volume (25), increased stroke volume, and decreased heart rate (27). In rats, there is a similar increase in plasma volume and increase in rectal temperature during the initial phase of acclimation (12) followed later by elevated stroke volume and cardiac output (13). The mechanism of rapid plasma volume increase in rats at rest in the heat probably results from a decrease in hydrostatic pressure brought about by decreased tissue perfusion (14). The increase in cardiac output does not appear to occur in some other species (10,11).

This affect of heat stress on the cardiovascular system produces a strain resulting from increased demand for cardiac output to transfer heat and water for evaporative cooling to superficial, vasodilated vascular beds in the skin (25) and peripheral venous anastomoses (10). This hemodynamic displacement of blood to the periphery is aggravated by the gravitational displacement of blood volume due to upright posture. When skeletal muscle vasodilates to supply the

increased demands for blood flow in support of muscle metabolism, the combined demands for flow exceed the capacity of the heart as a pump. As a result, these competing demands for flow among vascular beds represents a major regulatory problem (26) in order to maintain blood pressure, aerobic exercise capacity and thermoregulation within normal limits. This inherent competition between blood pressure maintenance, support for muscle metabolism and thermoregulation represents one of the greatest physiological challenges to homeostasis under conditions of normal daily life.

The existence of human heat illness as a phenomenon, the diversity or spectrum of the characteristic symptoms (5) and the resultant morbidity and mortality clearly establish the inherent risks of homeostatic failure during thermoregulation as well as the biological variability of the individual response to the combined stresses of heat and exercise. Moreover, the mere occurrence of these heat-induced disorders while exercise or exertion is ongoing suggests that the control of hemodynamic stability often takes precedence over thermoregulation. In other words, exercise performance is often maintained even as the risk of heat injury increases. In contrast to the more familiar sensations of fatigue or exhaustion (2), young, highly motivated individuals are often unaware of the seriousness of their hyperthermia. For example, Gilat et al (8) presented the rectal

temperatures of eight volunteers marching eight hours with a load of 35 kg. Six of the volunteers had rectal temperatures in excess of 40.6°C (mean = 41.1 C, n=8) and all were sweating profusely. So subtle are the evolving symptoms of hyperthermia that only three had signs of restlessness and euphoria.

Body temperature increases with heat stress and exercise; in fact, during athletic events rectal temperatures of 40-42 C° (104-107.6°F) are not uncommon (6,24,33). These elevated levels often exceed both the survival limits (35-40°C) of efficient thermoregulation in addition to the normal internal temperatures (36-38°C) as estimated by Stolwijk (31). We have previously reported that a temperature of 40.4°C (17,19) represented a threshold hyperthermia above which heatstroke mortalities occur in exercised heat-stressed rats. Cabanac (4) estimates that the highest temperature tolerated by the brain is probably 40.5°C. The mean core temperature of rats at exhaustion which produced a 50% mortality rate within 24h (LD50) was 41.5 ± 0.1 (SE). These results suggested a continuum of risk and a probability of mortality directly related to the core temperature at collapse. In humans, the rise in core temperature during exercise up to 90% of maximal oxygen consumption was more nearly in proportion to relative work load than to absolute work load (9).

Heatstroke is a condition caused by excessive rise in body temperature brought about by either "overloading" or "failure" of the thermoregulatory system during exposure to heat stress (3). "Overloading" occurs when the rate of heat production or storage exceeds the rate of heat loss. Under these conditions, heatstroke can occur even in the presence of profuse sweating. In such circumstances, the longer that exercise and the rise in rectal temperature could be sustained, the higher the core temperature would rise prior to collapse. From this perspective, the early onset of heat exhaustion and collapse in some susceptible individuals could preclude, in reality, a more serious episode of heatstroke. Gisolfi and Wenger (9) have estimated that highly trained athletes can produce metabolic heat in excess of 1,033 kcal/h. Under conditions in which heat dissipation is severely limited (e.g., highly humid conditions or in vapor impermeable clothing), the rate of heat storage from this kind of effort (0.3 C/min) could produce heatstroke within 15 minutes.

"Failure" of the thermoregulatory system suggests dysfunction in either central (hypothalamic) control or peripheral responses of heat loss mechanisms (sweating and vasodilation or skin blood flow). Human eccrine sweat glands behave physiologically and pharmacologically as if they were

cholinergic, muscarinic effectors. Active neurogenic vasodilation (7) is mediated by an, as yet, unidentified transmitter but may be linked to sweating (20). Atropine, the classic antimuscarinic agent, can traverse the blood-brain barrier and possesses both central and peripheral antimuscarinic activity. The peripheral and central anticholinergic syndrome accompanying drug overdose (1) bears striking resemblance (18) to heat illness (tachycardia, warm and dry skin, mydriasis, urinary retention, confusion, loss of consciousness etc.). Khogali (22) states that constricted and pinpoint pupils are present in 69% of heatstroke patients. The absence of mydriasis (widened pupils) argues against a diagnosis of anticholinergic poisoning. In the majority of cases (80%), the onset of heatstroke is sudden (30).

With prolonged exercise in the heat, the progressive displacement of blood volume into cutaneous veins and a shift of blood volume into dependent limbs lowers cardiac filling pressure and stroke volume. Heart rate must increase to maintain cardiac output. If core temperatures and skin blood flow become very high, cutaneous and splanchnic arterioles are unable to constrict enough to prevent venous pooling and a fall in blood pressure. In some individuals, the blood pressure falls gradually in proportion to the increased total vascular conductance of skin. These individuals may commonly collapse with lower core

temperatures and by definition be members of the heat exhaustion population.

In more resistant individuals, the increase in peripheral blood flow is, at first, adequately compensated by a decrease in splanchnic blood flow during moderate heat stress and dehydration. The abolishment of compensatory splanchnic vasoconstriction during the more severe thermal dehydration of non-acclimated rats has been demonstrated (13) for the first time. In this type of response in rats or humans, there would be an increased splanchnic conductance accompanied by a sudden fall in skin blood flow. This would produce a paradoxical fall in skin temperature at a time when core temperature was rising rapidly due to the diminished heat transfer capacity. The spiralling core temperature would make the occurrence of heatstroke more likely. Under experimental conditions resulting ultimately in a rapid rise in rectal temperature, a decrease in plasma volume in dogs (14) and a rapid fall in the tail skin temperature of running rats (16) appeared related to a spiralling increase in rectal temperature. This would suggest that a fall in the capacity for heat transfer and dissipation was, in part, responsible for the rapidly increasing hyperthermia.

More recent results provide further support for the hypothesis that a selective loss of compensatory splanchnic

vasoconstriction represents a key event in the pathophysiology of heatstroke (23). Progressive hyperthermia was accompanied by a rise in mean arterial blood pressure (MAP), which plateaued and then fell precipitously as core temperature exceeded 41.5°C. A temporal analysis of the regional vascular responses during the prodromal period using implanted Doppler flow probes indicated that increased superior mesenteric artery (SMA) resistance declined sharply 10-15 minutes prior to the precipitous fall in MAP. The time course of change in SMA resistance contributes to the fall in MAP but cannot account for its sudden decline as heart rate is rising. The latter suggests that the sudden fall in MAP must be associated with a decline in stroke volume and cardiac output. Whether myocardial failure is directly responsible for the decline in stroke volume and cardiac output is still debatable (32). Recently, Kielblock (21) characterized the cardiovascular response pattern in heatstroke as three distinct stages- compensation, crisis, and failure. The third stage was characterized by cardiac failure heralded by a fall in MAP. To quote the authors, "Although circulatory failure could ultimately be ascribed to cardiac failure, the trigger in all probability was the excessive reduction in total vascular resistance following the abolishment of compensatory splanchnic vasoconstriction" (21). It is interesting to note that a failure

in myocardial energy transformation reactions was also postulated.

The increased cardiovascular stability, lower heart rate, and increased stroke volume of the heat acclimatized individual represents, in part, an improved "cardiovascular reserve" (27). This state enables the individual to cope with the increased demand for cardiac output by increasing stroke volume rather than heart rate. A study by Horowitz and colleagues (15) using isolated rat hearts showed that acclimatization was accompanied by a change in the intrinsic properties of the cardiac muscle. This was characterized by "an increased compliance, reduced chamber stiffness, and a decrease in the tension developed for each volume load". As a result of acclimation, the pressure-volume diastolic curve was shifted significantly to the right allowing for increased filling volumes at equivalent filling pressures. The increased left ventricular compliance accommodates an increased stroke volume without an increase in end-diastolic pressure. This adaptation is seen as advantageous because it would support an increased cardiac output at lower heart rates. As Horowitz (15) points out, these results differ from the effects of training which increases the ventricular ejection fraction during systole without a change in diastolic function (29). The increased compliance of the acclimated heart also

differs from the adaptation associated with volume overload which increases stiffness (28).

This symposium has been organized to focus on the following areas: (a) exercise as a causative factor, (b) physiological systems involved, (c) symptoms and diagnosis, (d) differentiating between heatstroke and heat exhaustion, (e) treatment and management of heatstroke in the field, (f) whole body cooling techniques, (g) changes in symptoms during evacuation and hospitalization, (h) a new theory delineating the biochemical mechanisms involved in cell injury, (i) physiological and pathological factors underlying heat intolerance, (j) congenital and acquired heat intolerance, (k) time course and extent of recovery in patients, and (l) ability of recovering patients to acclimate to exercise heat exposure. Each speaker has been selected because of his scientific/clinical expertise in the area of his presentation.

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