REVIEW

Heat Strain in Tennis: a Selected Review of the Literature (Part 1)

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This is a first of a two part selected review of the literature on heat strain pertaining to tennis. The aim of this article is to present current knowledge on body temperature limitations during performance, acclimation/acclimatization issues, and aetiology of heat related disorders and predisposing factors contributing to heat strain. Part II of this review will be focusing on athlete sweat rate losses, fluid intakes and the issue of exertional hyponatremia, as well as methodologies to measure internal core temperature, current methodologies on cooling techniques and recommendations and guidelines currently in place in Grand Slams and Tour (ATP/WTA) events.

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Introduction

Exercise in a hot/humid environment poses a potentially stressful challenge to the maintenance of normal body temperature and fluid homeostasis.[1] Heat stressed-induced hyperthermia may occur in athletes whether they are participating in prolonged continuous submaximal exercise, such as a marathon or triathlon, or intermittent high-intensity exercise,[2] such as tennis. Modern competitive tennis is a year-round professional sport.
As suggested by Dawson et al.,[3] professional tennis players participate in a wide variety of climatic conditions from cool and dry conditions to hot and/or humid conditions. Recent anecdotal cases of players suffering from heat stress during tournament play have highlighted the need for scientific investigation into how players cope whilst playing in hot and/or humid conditions.
Research into exercise performance in hot environments and hyperthermia is vast.
A recent search using the terms "exercise, hyperthermia, heat stress" on Pub Med database returned a combined total of over 1000 articles. The aim of this review is to highlight, using selected papers, current knowledge about exercising in the heat with particular relevance to the tennis player, method of measuring core temperature, and current guidelines for playing tennis in hot/humid condition.

Heat production and its effects during exercise

Humans are homeothermic. At rest, and in non-extreme conditions, internal (core) body temperature remains steady within a small range of approximately 36.1 to 37.8°C (97.0 to 100°F).[4] Changes in core body temperature may move outside this range during prolonged heavy exercise, illness or extreme environmental conditions.
Heat production is a necessary by-product of metabolism. During tennis the metabolic rate, and consequently heat produced, increases substantially from that at rest.[6] Several authors have predicted that during exercise under warm to hot conditions the amount of energy released as heat (the lowest form of energy) ranges between 60 to 80%.[5,6] Sports Medicine Australia[7] have suggested that during exercise in warm to hot conditions an athlete may produce 15 to 20 times the amount of heat compared to rest.
Heat produced from metabolism can be transferred to the environment by conduction, convection, radiation, or evaporation. Heat loss through conduction and convection in air is relatively small, whereas radiation is the primary method for discharging the body's excess heat at rest, and evaporation of sweat is the primary avenue for heat dissipation during exercise.[4] Dawson et al.[3] have suggested that thermoregulatory and cardiovascular abilities are limited when exercising in hot and/or humid conditions, particularly when large water deficits occur in the body.
Exercising in relative humidity may also exacerbate the problem of releasing heat from the body as high relatively humidity restricts the vaporisation of sweat and promotes rapid dehydration.[3]
Exercise, such as tennis, under hot/humid conditions promotes competition between the active muscles and the skin for the limited blood supply. The redistribution of blood reduces central blood volume, stroke volume and a compensatory rise in heart rate, also known as cardiac drift.[8] When exercising in hot and/or humid conditions, the above competition between the cardio-vascular and muscular systems results in inadequate supplies of blood flow. Consequently the body is unable to dissipate heat produced[9] and increasing the risk of overheating. The loss of body water and electrolytes when exercising in the heat causes dehydration and hyperosmolarity, both of which have negative effects on circulation and temperature regulation; Nielsen and Nybo[8] have suggested that high core temperature may be an underlying factor causing fatigue during prolong exercise in the heat.

Body temperature limitations to performance

The attainment of a high level of body temperature, or hyperthermia, has been proposed as the main factor limiting endurance performance in hot environments.[10,11] A number of investigations have suggested core body temperature of 40°C as the critical physiological limit for performance.[9,12] Hargreaves and Febbraio[12] reported on volitional fatigue occurring to exercising males in ambient temperature of 40°C and 10% relative humidity at 60% of VO_2max at a rectal core temperature of 40°C. Fatigue in this group of males, was not found to coincide with any reduction in cardiac output, leg (muscle) blood flow or substrate availability and utilization.
Lindinger [9] showed, in exercising males at 45%VO_2max in conditions of ambient temperature of 35°C and 87% relative humidity, exhaustion occurred at 45mins when core temperature (oesophageal measurement) reached 40°C. Nag et al.[13] examined 11 male subjects performing exercise in controlled conditions at 60%VO_2max between 38 to 49°C dry bulb temperature and 45 to 80% relative humidity and found in these subjects the tolerance limit was a core body temperature 39°C and heart rate attained 172 to 182 beats/minut. The authors concluded that subjects were only susceptible, showing 'unacceptable levels' of physiological and psychological reactions, to extreme heat conditions. They further suggest the consideration of time limits for prolonged work in extreme heat conditions being 80 to 85mins for individuals with 38 to 38.2°C core temperature or 40 to 45mins at 39°C core temperature.[13]
Even in trained subjects, core temperature of ~40°C also appears to be the physiological limit for termination of exercise. Neilsen et al.[11,13] demonstrated in trained athletes cessation of exercise due to fatigue at oesophageal temperatures of 39.7 and 39.9°C respectively. Gonalez-Alonso and colleagues[15] reported in cyclists, despite initial core body temperature, fatigue in all subjects occurred at a similar core body temperature (40.1-40.2°C).
Although a core temperature as high as ~ 40°C has been reported in the fatigue of highly trained endurance athletes during exercise-heat stress, several investigations have shown that untrained individuals cannot tolerate the same high core temperature as their endurance trained counterparts with rectal core temperatures of approximately 0.5-0.9°C lower in untrained individuals at point of exhaustion.[10,16]

Heat acclimatization

Heat acclimatization is described as the process of physiological and psychological adaptation to the new environment.[17] When accomplished in an artificially controlled environmental chamber, this process is known as heat acclimation.[51] It is generally accepted that the ability to tolerate exercising in the heat can be improved by a combination of exercise and repeated exposure.[7,17,18,19]
Mechanisms that occur during the acclimatization process include earlier onset of sweating and increase in sweat rate. Conversely, the electrolyte content of sweat decreases.[17] Primary physiological adaptations that occur includes an expansion of plasma volume, increased cutaneous blood flow, red-cell filterability, improved circulatory control; and reduced utilization of muscle glycogen.[17]
These adaptations contribute to a reduced core temperature and heart rate response for a given heat exposure and exercise intensity.[18]
It has been suggested that heat acclimatization may take between 10 to 14 days.[17]
However, Bailes et al.[19] suggests that athletes require between seven to 14 days to become heat acclimatized. Sports Medicine Australia[7] has suggested that adaptation to exercising in the heat are rapid, and in hot environments can occur after three to five days, however

full acclimatization can take 10 to 14 days. It should be noted that no mention is made whether these suggestions are consistent across sports.

High aerobic fitness accelerates development of acclimatization.[52,53] Aerobic exercise increases core temperature and elicits sweating even in a moderate environment, and aerobic training involving exercise at 70% of maximal oxygen uptake (VO2max) or greater[54,55] produce changes in the control of sweating similar to those produced by heat acclimatization. However, whether or not aerobic training in a temperate environment induces true heat acclimatization has yet to be resolved. In a critical review of the evidence and arguments on both sides of the issue, Gisolfi and Cohen [56] and Pandolf and colleagues[57] suggested that exercise training programs lasting two months or more in a temperate environment produce substantial improvement in exercise heat tolerance. However, exercise training alone has not been shown to produce a maximal state of exercise-heat tolerance.

Heat acclimatization does not appear to change the critical physiological limit of cessation of exercise. Hargreaves and Febbraio[12] found that amongst trained men exercising in hot and humid conditions until fatigue over 9 to 12 days, acclimatization did not change the core temperature at which fatigue occurred. However the exercise time required attaining the critical high core temperature was increased, suggesting acclimatization did take place in these subjects.

The benefits of acclimatization are reduced or undone by sleep loss, infection, and alcohol abuse,[58,59], salt depletion,[58] and dehydration.[58,60,61] Heat acclimatization gradually disappears without periodic heat exposure, although partial losses (for example, a few days' interval of non training) are easily regained.[59] The improvement in heart rate, which develops more rapidly, also is lost more rapidly than are the thermoregulatory improvements.[53, 62- 64] However, there is disparity in how long acclimatization persists. In an early study cited by Wenger,[57] acclimatization almost completely disappeared after 17 days without heat exposure. Conversely in another study, approximately three quarters of the improvement in heart rate and rectal temperature was retained after 18 days without heat exposure.[53] Bean, Eichna and others [65-66] were the first to show that physically fit subjects retain heat acclimatization longer; and warm weather may[66] or may not[64] favor resolution of acclimatization, although intermittent exposure to cold does not appear to hasten the loss of heat acclimatization.[67]

Heat related disorders (heat illness)

Heat illness is an umbrella term describing various forms including cramps, heat exhaustion and heat stroke.[18] Heat illness is generally caused where the required evaporative cooling necessary for the body to achieve a thermal steady state exceeds the maximum evaporative potential of the environment. Such a situation defines a condition of uncompensable heat stress[20] where the body continues to store heat and core temperature continues to rise to dangerously high levels. Further, exercising for prolonged periods without fluid intake increases the risk of heat illness.[6] Progressive dehydration during exercise in the heat reduces the sensitivity of the sweat rate/core temperature relationship and thus results in a relative hyperthermia and an earlier onset of fatigue.[5] Core temperature and heart rate have been identified as the two primary determinants of heat illness. Other measures include various combinations of heart rate, core and skin temperature and/or their rates of change, and sweat production. The traditional classification of heat illness defines three heat illness disorders: heat cramps, heat exhaustion and heat stroke. However, Binkley et al.[22] asserts this classification scheme omits several other heat- and activity-related illnesses, including heat syncope and exertional hyponatremia. Although heat illness is more likely to occur in hot, humid conditions, heat illness can occur in the absence of these environmental conditions.[22] Further, many cases of exertional heat illness are preventable and can be successfully treated if the condition is recognised and treated in appropriate time.[22,24]

Cramps

Heat cramping is the least serious of the heat disorders. Elchner[25] has suggested that sodium deficits may make motor nerve endings hyper-excitable, leading to involuntary and sustained contractions or cramping. Although considered 'least serious' heat cramps can produce excruciating pain and cause tennis players to discontinue play.

Syncope

Heat syncope, or orthostatic dizziness, can occur when a person is exposed to high environmental temperatures[26] and usually occurs in the first five days of acclimatisation before blood volume expansion.[27] It often occurs after standing for long periods of time, immediately after cessation of activity, or after rapid assumption of upright posture after resting or sitting.[22]

Exhaustion

Heat exhaustion is caused by the cardiovascular system's inability to adequately meet the body's needs to simultaneously dissipate heat through vasodilation as well as supply the blood to the working muscles.[22] Although the actual mechanisms causing heat stroke are not well understood, this condition is life-threatening.[22]

Heat Stroke

Exertional heat stroke occurs when the temperature regulation system is overwhelmed due to excessive endogenous heat production or inhibited heat loss in challenging environmental conditions[28] and can progress to complete thermoregulatory system failure.[26,29] Even if not fatal, the effects of heat stroke are often irreversible[19] including severe damage to the brain, liver and kidneys.[23]

Hyponatremia

Exertional hyponatremia once thought as a rare condition but has now emerged as an important cause of race-related death and life-threatening illness among endurance athletes (such as marathon runners).[50] It is defined as a serum-sodium level less than 130 mmol/L. Low serum-sodium levels usually occur when activity exceeds four hours.[26] Two, often-additive mechanisms are proposed: an athlete ingests water or low-solute drinks well beyond sweat losses (also known as water intoxication), or an athlete's sweat sodium losses are not adequately replaced.[30] Exertional hyponatremia can result in death if not treated properly. For a full listing of signs and symptoms of exertional heat illnesses the reader is referred to Binkley et al.[22]

There are a number of predisposing factors for exertional heat illness, these factors maybe classified either as intrinsic/non-environmental, extrinsic/ environmental, or predisposing medical conditions.[22,24] Intrinsic factors for exertional heat illness include the following: dehydration; poor physical condition; history of exertional heat illness;[31] inadequate heat acclimatization;[32] increased body mass index;[33] illness, such as fever;[34,35] dietary supplements; overzealousness, and reluctance to report problems, issues or illnesses, and barriers to evaporation, such as wearing rubber or plastic suits for "weight loss", and excessive or dark coloured clothing.[36,37] Extrinsic factors include ambient air temperature, relative humidity, air motion, and the amount of radiant heat from the sun or other sources, of which these can be calculated using the Wet Bulb Globe Temperature (WBGT). WBGT index can calculate the risk of heat illness. Other extrinsic factors contributing to heat illness can include intense or prolonged exercise with minimal breaks or inappropriate work/rest ratios, lack of education and awareness of heat illnesses among coaches and athletes, no emergency plan to identify and treat exertional heat illnesses, no access to shade during exercise or rest breaks, and minimal access to fluids before and during rest breaks.[24] Predisposing medical conditions add to the risk of heat illnesses including: malignant hyperthermia, where an autosomal dominant trait causes muscle rigidity, resulting in elevation of body temperature, due to the accelerated metabolic rate in skeletal muscle;[38] neuroleptic malignant syndrome, associated with neuroleptic agents and antipsychotic drugs giving rise to an unexpected idiopathic increase in core temperature during exercise;[39] arteriosclerotic vascular disease compromises cardiac output by thickening of the arterial walls;[40] and, scleroderma, skin disorder decreasing sweat production.[41]

Age has also been questioned with early investigations suggesting that the ability to exercise in the heat deteriorates with age.[42] More recently well-controlled studies[43,44] concluded that exercise-conditioned old and young males showed little difference in thermoregulation during exercise. It maybe suggested that earlier studies were not properly controlled for deconditioning and a lack of heat acclimatization, and may explain these studies findings of decreased heat tolerance in older people. For more information regarding age and thermoregulation, the reader is referred to Kenny.[45]

Gender has been considered as a predisposing factor and early research indicated that females were less tolerant exercising in hot conditions than males; however these studies did not match participants for physical fitness or size.[46] Marsh and Jenkins[46] assert that very little data exists to suggest that differences in thermoregulatory capability can be attributed to gender when there is adequate matching and control of factors such as maximal aerobic power, heat acclimation and menstrual cycle phase. In particular, increased progesterone levels during the luteal phase causes increases in both core and skin temperatures and alter the temperature at which sweating begins during exposure to both ambient and hot temperature. Elevated core temperature during this phase has been reported as high as 0.6 °C both at rest[47] and during exercise[48] in ambient temperatures, which are maintained in hot environments. Lower sweat rates found in females[46] are thought to be due to the →

larger surface area-to-mass ratio which would be a disadvantage when ambient temperatures become higher than the skin temperature as the body will gain heat via conduction and radiation.[46] Similarly, higher levels of body fat, such as that found in females, significantly influences thermoregulation.[46] However, McLellan[49] reported that males and females matched for body fatness do not exhibit differences in sweat rate, metabolic rate, heart rate or tolerance time to heat exposure. It should still be noted for those involved with tournament tennis, that some female tennis players may be at a disadvantage, due to their lower total body water and consequently greater potential for relative dehydration, in tournament venues that have high on-court evaporative heat losses.[6]

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