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A modern classification of the exercise-related heat illnesses

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Summary This article proposes a novel framework classification for the heat illnesses. It argues that heat stroke is the only described condition that is truly a "heat illness" since it is the only condition in which there is clear evidence for a pathological elevation of the core body temperature. If this is correct the non-descript terms such as heat fatigue, heat exhaustion and heat syncopy should be removed from the modern lexicon. Since the evidence is that most cases of post-exercise collapse are due to the development of postural hypotension immediately on the cessation of exercise, it is further proposed that more specific terms such as exercise-associated postural hypotension should be used, when appropriate, to replace the non-descript terms such as heat exhaustion, heat fatigue or heat syncopy. Furthermore this novel classification acknowledges that heat stroke may occur in some as a result of accelerated rates of endogenous heat production (thermogenesis). It also suggests that the elevated body temperature alone may not be the sole cause of fatal outcomes in heat stroke but that toxic chemicals released from damaged muscles by the processes causing this accelerated thermogenesis may also be involved.

A popular perception is that any athlete who collapses during or after exercise in the heat should be diagnosed as suffering from a "heat illness" provided she or he does not have an obvious and well-defined medical condition such as cardiac arrest. Terms such as heat cramps, heat syncope, heat exhaustion, heat fatigue, siriasis, heat stroke and even dehydration and sunstroke are usually included in this classification.[1–5] In some traditional classifications, a further distinction is made between "heat illnesses" due either to salt or water deficiency.[3–6]

But the proper practice of medicine requires that each disease must be uniquely identifiable by a specific set of symptoms, signs and laboratory and other findings that distinguish it from all other known conditions. For, if they are to be managed properly, diseases with different causes may require different treatments. It helps not, for example, to treat a patient who presents with a cough for pneumococcal pneumonia if, in fact, she has cardiac failure. If the condition is not correctly defined, it cannot be properly diagnosed. Without correct diagnosis, the condition cannot be treated optimally so that either under- or overtreatment may result. The result of inappropriate treatment

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could include a fatal outcome as might occur, for example, when an athlete suffering from fluid overload is treated for "dehydration".[7–9]

Initial attempts to classify the "heat illnesses" seem to have been made by the medical teams caring for the British troops fighting in the Middle East in the First World War.[1] With few modifications, many of the terms adopted since then, including heat cramps, heat exhaustion and heat fatigue, are still in current use. But is this appropriate? Is there new information which suggests that this classification should be modernised especially when used to describe medical conditions occurring in athletes participating in currently popular single day endurance sports like marathon and ultra-marathon running races and Ironman triathlons, or even multi-day adventure races?

For example, the original classifications were based on clinical findings in military personnel who were exposed to extreme, unrelenting dry (desert) or wet (jungle) heat for weeks, months or years on end, during which they performed moderate amounts of daily exercise. These overstressed soldiers also had no relief from nocturnal heat since their accommodations were without air conditioning. Already in the 1930s it was found that the incidence of heat collapse was lessened when those who constructed the Hoover Dam on the Colorado river (USA) in the heat of the day, slept in air-conditioned bungalows at night.[10]

Yet modern athletes, whether they exercise for many hours in ultra-endurance events like the Ironman Triathlon or for much shorter durations of minutes to hours in other popular competitive or recreational activities, suffer no such inconvenience. They can escape the heat at all times when they are not training or competing. Their primary goal may also be different. Some wish to perform a single maximal effort on one chosen day, not repeated daily bouts of low intensity exercise as was the case for the military heat casualties described in the original studies. I propose that there is now sufficient information to allow a radical revision of the traditional classification of those exercise-associated "heat illnesses" which are commonly encountered in modern endurance athletes competing for many hours in the heat.

To what does the term "heat" apply?

The first issue that requires attention is the meaning of the word "heat" in the term "heat illness". As traditionally understood, it seems that the term "heat illness" refers purely to an "illness" that develops during exposure to an environment that is other than cold; much as the term "cold injury" might be used generically to describe any injury that occurs exclusively on exposure to a cold environment. But there is one important distinction. Whereas the most common cold injury, frostbite, can occur only in very cold environments since extreme cold is the direct and only cause of this injury, the so-called "heat illnesses" including even heatstroke, can occur in temperate environments[11] perhaps because it is easier to sustain a higher exercise intensity in cooler conditions. Thus any direct causal link between the environmental conditions and the "heat illness" is rather less obvious than is the case with cold injuries.

Or does the term "heat" refer to an illness that occurs when the body heat content is pathologically increased? But this also cannot be so since there is now very good evidence that most athletes diagnosed with any of the so-called "heat illnesses" other than heat stroke, do not have a pathological elevation of their core body temperatures at the time of collapse. For example, we[12] showed that the range of rectal temperatures in collapsed ultra-marathon runners is no different from those measured in asymptomatic marathon runners in many earlier studies.[13–17] Conversely the recent study of Byrne et al.[18] showed that the intestinal temperatures of 12 of 18 asymptomatic runners completing a 21 km race in the heat were above 40 °C and two were above 41 °C. This confirms that a rectal temperature in excess of 41 °C is frequently found in completely asymptomatic humans during vigorous exercise in the heat.[19] Thus this degree of temperature elevation cannot, by itself, be considered diagnostic of a "heat illness". The corollary is that a temperature below about 41.5 °C cannot be the exclusive cause of exercise-associated collapse and hence, even when present, is not sufficient to warrant a diagnosis of "heat illness". But confusion can clearly arise if a subject with a core temperature in excess of 41 °C also has postural hypotension as this could lead to an inappropriate diagnosis of heatstroke if the differentiating features are not appreciated.

Thus my central argument is that the term "heat illness" should be used only when there is clear evidence for abnormal body heat retention during exercise so that the core temperature is elevated, usually above 41.5 °C. This abnormal heat storage could result either (i) from a failure to lose an adequate amount of heat produced during exercise causing a gradual and progressive increase in body temperature, or (ii) as a result of abnormally high rates of (endogenous) heat production by the body, or (iii) a combination of (i) and (ii). If this definition is correct, then the first absolute diagnostic

criterion for "heat illness" is a core temperature elevated above the highest values found in otherwise asymptomatic athletes, that is, usually above 41.0–41.5 °C. However, on occasion, the temperatures of patients with heat stroke may be lower than 41 °C. Thus when the clinical picture is entirely compatible with the diagnosis, heat stroke should not be excluded simply because the temperature does not exceed 41 °C.

The second criterion is that the elevated core temperature must be associated with evidence for an altered cerebral function, in which case the presumptive diagnosis is heat stroke.

Why do most persons with "heat illness" collapse even if they are not hot?

According to this argument,[20] persons who collapse during or after exercise but who retain their mental faculties and whose body temperatures are not above at least 41.0 °C, should not be diagnosed as suffering from a "heat illness". If this is correct, then the common terms such as heat cramps, heat syncope, heat exhaustion, heat fatigue and sunstroke should be removed from the general classification since, in popular use, they are always used to describe a group of non-descript and ill-defined "illnesses"[21] in athletes whose body temperatures are not higher than peak values measured in other asymptomatic athletes (who would normally constitute the control group).

Rather, the diagnosis of "heat illness" should be reserved only for athletes in whom there is clear evidence for a pathological increase in body heat storage, as occurs in heat stroke. The increase is clearly pathological when the other features classically used to diagnose heat stroke are also present, in particular abnormal cerebral function including altered behaviour, an alteration in the level of consciousness or coma. But in the absence of evidence for abnormal heat retention and altered cerebral function, there can be no justification for the continued use of any of the poorly defined terms such as heat fatigue, heat exhaustion, sunstroke and heat syncope, for example. The question then becomes: what causes athletes to collapse especially after exercise even though they are not suffering from a "heat illness" as now defined since their core body temperatures are within the normal range?

A crucial observation made by us more than a decade ago was that approximately 75% of subjects seen in the medical tent at the finish of an ultra-marathon race had collapsed after they had finished the race.[12] Those who collapsed during the race were more likely to have significant medical conditions including heat stroke. More recent data from the Twin Cities Marathon in the USA suggests that as many as 99% of subjects treated in the medical facility at the race finish had collapsed after they had completed the race.[22] Clearly the usual explanation that such collapses are always due solely to "dehydration" cannot be correct since, had the athletes developed cardiovascular "strain" with a low cardiac output as a result of "dehydration" – a currently popular explanation[23] – they should have collapsed before the finish line when their cardiac outputs were higher and therefore more likely to be "strained". Similarly collapses are common in the Sydney City to Surf Race of 14 km even though the activity may last for only 60–80 min during which (short) time significant dehydration cannot occur.[24]

Next we found that a large proportion of subjects developed postural hypotension after completing an 80 km ultra-marathon. Their blood pressures were more normal when they lay supine.[25] Empirically we discovered that athletes with postural hypotension after exercise became asymptomatic immediately they were placed in the head-down (Trendellenberg) position with their legs and pelvis elevated above the level of the heart.

Finally we retrieved the study of Barcroft and Edholm[26] which seemed better able to explain (i) why exercise-associated collapse can occur precipitously almost the moment endurance athletes stop exercising; (ii) why a compensatory tachycardia is not a usual feature of this form of hypotension,[27] and (iii) why the symptoms can be reversed almost instantaneously when subjects lie supine in the head-down position.

These authors originally proposed the existence of a right atrial reflex, subsequently termed the Barcroft–Edholm reflex,[27] that induces a potent vasodilator reflex in skeletal muscle whenever the right atrial pressure falls either too rapidly or below some critical value. This reflex would be activated shortly after the termination of prolonged exercise in the heat as a result of the sudden withdrawal of the lower limb muscle pump as running or walking stops. This causes blood to accumulate in the dilated veins of the lower limb with a reduction in the central blood volume and the right atrial pressure. The reflex is atavistic since it compounds rather than corrects the hypotension caused by this redistribution of blood volume into the veins in the lower limbs.

We concluded that the rapidity with which the symptoms of post-exercise collapse are reversed when the legs are elevated, suggests that this

right atrial reflex probably contributes to post-exercise postural hypotension. Lifting the legs may reverse the hypotension not only by increasing the venous return and hence the cardiac output, the more usual explanation, but also by instantaneously increasing the right atrial pressure. This would immediately reverse the Barcroft/Edholm reflex[26] thereby removing the added vasodilation that this reflex causes in skeletal muscle and perhaps elsewhere. On the other hand, if elevation of the lower limbs does not reverse the symptoms of hypotension, then one or more other factors may be involved. The most likely factor would be ventricular dysfunction although, when severe (>10–15%), dehydration might conceivably also play a role.

However the modern evidence is that postural hypotension develops after exercise as a result of some or all of the following exercise-induced changes in blood pressure regulation: a resetting of the arterial baroreflex to lower pressures after exercise[28]; impaired sympathetic vascular regulation,[29] and H_1 and H_2-receptor mediated vasodilation.[30–32]

The important clinical point is the following: many athletes will develop symptoms only after they stop exercising as a result of the sudden development of post-exercise postural hypotension. Indeed, one might argue that this is the more "normal" state after exercise. When these subjects lie down, their blood pressures are likely to be low as a result of peripheral vasodilation[33] with a normal or elevated cardiac output. Lifting the legs and pelvis will rapidly reverse any symptoms that are present when lying. However, symptoms may well recur if patients attempt to stand too soon after their collapse and before this state of low peripheral vascular resistance has been reversed.

I propose that this is the condition which, in the past, has been incorrectly called heat exhaustion, heat fatigue, heat syncope or "dehydration" amongst others, since its real aetiology was not understood.

But now that we understand that postural hypotension is common especially after prolonged exercise in the heat, it becomes possible to suggest that this condition probably explains almost all cases of benign collapse that occur shortly after the termination of exercise in athletes who are not suffering from another obvious medical condition. This state fulfils all the diagnostic criteria for the condition term exercise-associated collapse as first proposed by Roberts,[34] and which is the most common diagnosis in the Twin Cities marathon.[22] But since this is purely a physiological state rather than a medical condition, it might perhaps be better simply to name it according to the prinicipal physiological change that causes it.

A proposed new classification of "heat illnesses" commonly encountered in modern endurance athletes

I have argued that there is no value in continuing to use terms such as heat fatigue and heat exhaustion since there is no evidence that abnormal heat retention plays any role in these incorrectly termed "heat illnesses". Nor do these non-descript terms advance the understanding of the pathophysiology of this condition. Nor should the term dehydration ever be used either to define these conditions or to imply their aetiology since there is no randomized controlled clinical trials to establish whether or not dehydration plays any role in these conditions.[35] Rather the clear evidence is that a state of low peripheral vascular resistance is the usual consequence of prolonged exercise and that this is the likely mechanism for most cases of collapse from "heat illness" after exercise. Perhaps the most appropriate term for this condition would be exercise-associated postural hypotension.[33]

Table 1 Proposed new framework classification for the so-called "heat illnesses"

1. Conditions in which there is a pathological increase in body heat storage
Heatstroke
(a) Exercise and environmentally induced heat stroke (EEHS)
(i) Exercise-induced heat stroke due to inadequate heat loss during exercise
(ii) Exercise-induced heat stroke due to excessive endogenous heat production during exercise
(iii) Combination of (i) and (ii)
(b) Other causes of heat stroke unrelated to exercise
2. Conditions in which there is no pathological increase in body heat storage
Exercise-associated postural hypotension
Other conditions not specific to exercise

Accordingly a novel framework classification is proposed in Table 1. This new classification distinguishes between conditions in which there is clear evidence for a pathological rise in the core body temperature so that the diagnosis of "heat illness" is appropriate, from those in which the core body temperature is not abnormally elevated. Currently, other than some obvious medical conditions not specific to exercise, postural hypotension is the only known mechanism that would explain the collapse that occurs immediately on the cessation of exercise and which is the commonest cause of admission to the medical facility at endurance events. Hence the suggestion that, in the absence of a readily identifiable medical condition, the initial diagnosis in persons who require medical care after exercise in the heat and whose core body temperatures are not above about 41 °C, should be exercise-associated postural hypotension. The other diagnostic tests required to make this diagnosis have been described.[20]

In contrast heat illness should only be diagnosed when the core body temperature is pathologically elevated and there is also evidence for altered cerebral function. Under these conditions, the critical factor in treatment is to lower the body temperature as rapidly as possible.[36] However if future heat stroke fatalities are to be prevented, there is also the need to understand how the condition develops and why it occurs. This is why the new framework classification distinguishes different types of heat stroke cases according to the likely mechanism causing the excessive heat accumulation.

The traditional explanation is the heat stroke occurs in athletes who exercise too vigorously, thereby generating too much heat which they are unable to lose when the environmental conditions are severe. But this explanation ignores the clear evidence that, when given the choice, humans normally regulate their exercise intensity (work rate) specifically to insure that their body temperatures do not rise excessively.[37,38] But forced pacing, for example in military or some occupational settings, might force some humans to exercise beyond the safe limits imposed by these controls.

Thus a failure of this central regulation must be present in all persons who develop heat stroke[39] during either voluntary or imposed exercise. Furthermore, calculations indicate that the environmental conditions necessary to cause progressive heat accumulation during exercise are usually much more severe than those actually present when most cases of exercise-induce heat stroke occur. This has led to the suggestion that athletes who develop heat stroke during exercise must either (i) have an abnormally reduced capacity for heat loss or (ii) their normal capacity for heat loss is unable to cope because the rate of endogenous heat production is abnormally rapid.[39]

The practical point is that if an accelerated endogenous heat production (thermogenesis) is present in persons with heat stroke, then the mechanisms causing that thermogenesis may also be the factor(s) that causes the fatal outcome. This contrasts with the usual presumption that it is the heat alone that causes death in heat stroke. For the overlooked point is that most patients who die from heat stroke whilst receiving apparently appropriate treatment, succumb when their body temperatures are not greatly elevated. Rather it may be that these abnormal thermogenic processes are associated with an extensive skeletal muscle rhabdomyolysis which releases toxic biochemicals into the circulation. Perhaps it is these chemicals that contribute to the mechanisms causing multiple organ failures characteristic of heatstroke and which include disseminated intravascular coagulation, acute renal failure, adult respiratory distress syndrome and ultimately cardiac failure and cardiac arrest.

Hence focusing only on reducing the elevated body temperature, treats just one of the features of this condition. It may be that the novel interventions that will save lives are those that reverse the rhabdomyolysis and which prevent the leakage of toxic molecules from skeletal muscles into the circulation.

Yet, to my knowledge, this is not often considered an important goal in the treatment of heatstroke.

Practical implications

- Traditional classifications of the heat-illnesses include a range of "illnesses" that are poorly defined and in none of which is the core temperature abnormally elevated. This new classification proposes that heatstroke is the only true heat illness but that it may, on occasion, result from a state of elevated heat production within the body—heatstroke due to excessive endogenous heat production during exercise.
- Furthermore, this classification proposes that other causes of exercise-associated collapse should be classified according to the likely physiological mechanisms causing collapse. The most usual mechanism is postural hypotension.

- **According to this new classification, management of these conditions is simplified. Patients with heatstroke must be actively cooled until their core temperatures are ~38 °C. If there is evidence for excessive endogenous heat production, interventions may need also to focus on preventing or limiting rhabdomyolysis, which may be the ultimate cause of death in cases of heatstroke that fail to improve despite an appropriate reduction in the core body temperature.**
- **Similarly, there is currently no known treatment that rapidly reverses postural hypotension after prolonged exercise. Supportive treatment which increases venous return and increases the right atrial pressure, in particular nursing patients with their legs and pelvis above the level of the heart, is usually all that is required other than time and patience.**

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