Review Article

Health Risks and Interventions in Exertional Heat Stress

Dieter Leyk, Joachim Hoitz, Clemens Becker, Karl Jochen Glitz, Kai Nestler, Claus Piekarski

Summary

Background: With climate change, heat waves are expected to become more frequent in the near future. Already, on average more than 25 000 "heat deaths" are estimated to occur in Europe every year. However, heat stress and heat illnesses arise not just when ambient temperatures are high. Physical exertion increases heat production within the organism many times over; if not enough heat is lost, there is a risk of exertional heat stress. This review article discusses contributing factors, at-risk groups, and the diagnosis and treatment of heat illnesses.

Methods: A selective literature search was carried out on PubMed. Current guidelines and expert recommendations were also included.

Results: Apart from muscular heat production (>70% of converted energy), there are other factors that singly or in combination can give rise to heat stress: clothing, climate/acclimatization, and individual factors. Through its insulating properties, clothing reduces the evaporation of sweat (the most effective physiological cooling mechanism). A sudden heat wave, or changing the climate zone (as with air travel), increases the risk of a heat-related health event. Overweight, low fitness level, acute infections, illness, dehydration, and other factors also reduce heat tolerance. In addition to children, older people are particularly at risk because of their reduced physiological adaptability, (multi-)morbidity, and intake of prescription drugs. A heat illness can progress suddenly to life-threatening heat stroke. Successful treatment depends on rapid diagnosis and cooling the body down as quickly as possible. The aim is to reduce core body temperature to <40 °C within 30 minutes.

Conclusion: Immediately effective cooling interventions are the only causal treatment for heat stroke. Time once lost cannot be made up. Prevention (acclimatization, reduced exposure, etc.) and terminating the heat stress in good time (e.g., stopping work) are better than any cure.

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Time and again, heatwaves claim lives. In Europe, on average, over 25 000 heat-related deaths are believed to occur every year (e1–e4). With climate change, these sudden episodes of extreme weather are expected to be more frequent (e5, e6), impacting a population that is largely insufficiently acclimatized.

However, heat stress and health risks do not only arise when environmental temperatures are high; they can be triggered by physical exertion—even at apparently low-risk temperatures (1–3). Intense and rapid heat production takes place in the working muscles, such that at a high intensity of activity core body temperatures of over 39 °C can be reached within 20 minutes (4–6). Whether at work, in leisure pursuits, or during sporting activity, physical exertion can lead to overheating of the organism (heat stress) and to heat illness; higher environmental temperatures further increase the risk (7–13).

This review article focuses on:

- The main factors (physical exertion, clothing, environmental conditions, and individual characteristics) that singly or in combination can trigger heat stress in an organism (1–3, 14, 15);
- Population groups at increased risk of a heat-related health event; and
- The diagnosis and treatment of heat illness.

Methods

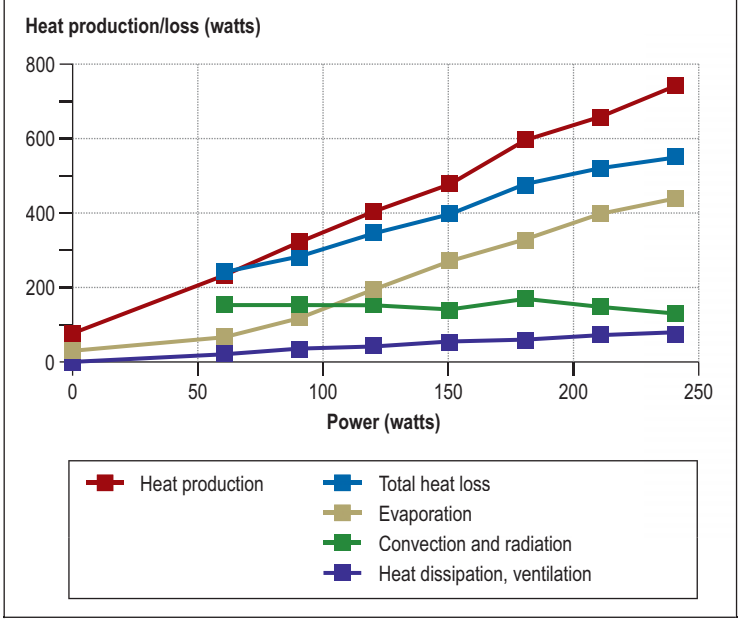
A selective literature search was carried out on PubMed for heat illness associated with the following topic clusters: climate, risk factors, prevention, treatment, and diagnosis. Guidelines and expert recommendations (16) were also included. Search terms and the search procedure were as shown in the *eBox* and the *eTable*.

Muscles as heat engines

The combination of heat with physical work puts enormous stress on the human organism and can lead to sudden loss of performance and threats to health (1, 3, 17–19). However, the organism can also become overheated in ambient temperatures below 0° C: for example, during ski patrols in an ambient temperature of –8 °C, core body temperatures of over 38 °C have been measured (1, 20). In a few cross-country skiers, core temperatures of over 40 °C have been recorded (1, 4).

Muscle work can lead to a rate of heat production that is more than ten times that in the resting state.

FIGURE 1



Heat metabolism: heat production and heat dissipation at rest and during increasing physical exertion (60 minutes on a cycle ergometer) in an unclothed human being at an ambient temperature of 22° C (adapted from [e7, e8])

As with a heat engine, by far the greatest part of the energy converted within the muscle is released in the form of heat (>70%) (1, 21–24). During running, the mechanically usable part of the energy is 25% at most. In domestic work or occupational activities, such as carrying loads or moving about in protective clothing, the efficiency is much lower (1, 19 , 25, 26). Because humans have a low tolerance (37 °C to around 40 °C) for increases in core body temperature, under conditions of heat even moderate physical work can result in heat illness, which may be as extreme as heat stroke, without warning (2, 18).

Insulating effect of clothing

Insulation provided by clothing should be variably adapted to ambient environmental conditions (e.g., in heat waves) in order to avoid additional thermal stress. Where there is a particularly high need for protection, however, such stress can be unavoidable even in temperate conditions (1, 27), e.g., in sport (football, fencing) or in certain occupations (e.g., police, firefighters, the military, the chemical industry). Furthermore, the weight and movement restrictions imposed by protective clothing and equipment can lead to increased muscle work and metabolic heat production (28).

Figure 1 shows heat production and dissipation at rest and during physical work. When protective clothing is worn, the most important and most efficient (70% to 80%) physiological cooling mechanism during physical work—heat loss through the evaporation of sweat—is very greatly restricted (1, 27). As a result of this, high rates of metabolic heat production can result in fatal heat stroke (29, 30). No representative data exist as to the incidence of fatal heat stroke in persons carrying out physical work in protective clothing.

Climate and acclimatization

When protective clothing is worn, even small amounts of thermal environmental stress increase the risk of heat illness (1, 9, 17–19, 29, 31, 32). In addition to air temperature, humidity, wind speed, and heat radiation are all significant climatic factors. Thermal environmental indices have been created to reduce various combinations of these effect sizes into a single value. For outdoor heat exposure, the WBGT (wet bulb globe temperature) index is widely used (16, 33). It is used to estimate risk in order to reduce heat-related health events at work (16) or during sport (34).

Through acclimatization (classic signs of adaptation: increased sweat rate, lower heart rate and core body temperature), heat tolerance can be improved and heat stress more easily compensated (35–38). However, it takes about 7 to 10 days for acclimatization to occur (18, 37, 39, 40). Sudden heat waves (on land) or changing climate zones (air travel) increase the risk of a heat-related event due to lack of or insufficient acclimatization (e1, e9, e10).

Risk factors in the individual and at-risk groups

Heat tolerance can vary greatly within a single person: acute infections and disease, dehydration, disturbances in electrolyte levels, overmotivation, insufficient acclimatization, or intake of prescription drugs all make heat illness more likely (1, 15, 26, 29, 32, e11, e12). Everyday observation, too, reveals variations in the ability to tolerate heat stress. For example, there are wide variations between individuals as to how much they sweat. Children are much more vulnerable than adults to high heat stress (1, e13–e15), because they have lower sweat rates, both absolute and relative (i.e., relative to their body surface area) (e14, e16). Despite sex-related differences (women have more subcutaneous fat tissue, later onset of sweat secretion, menstruation-related changes in core body temperature, etc.), men and women appear to have similar abilities to withstand heat stress (1, 19, e17–e19; on the ability of children and women to withstand heat stress, see review articles e13, e20, e21).

Older people

Older people in particular (from the age of 75) are at risk on hot days and during heat waves (e22–e25). Most heat-related deaths in Germany (2001–2015) are estimated to have occurred in 2003 (n = 7600), 2006 (n = 6200), and 2015 (n = 6100), and most were in this age group (e22). The main causes are the higher prevalence of chronic disease and the reduced physiological adaptability of this population group (e26). For example, skin blood flow is lower in old age and the redistribution of blood volume from retroperitoneal venous networks to the skin capillary bed is decreased

(e27, e28). Older people also sweat later and less than younger ones (e29, e30). As a result of these changes, less heat can be dissipated through the skin in old age (e31). Disease can further restrict thermoregulation. For example, in patients with cardiac insufficiency, the thermophysiological increase in skin blood flow may be reduced, because it requires cardiac output to be increased while at the same time maintaining adequate blood pressure.

Heat waves at the beginning of the summer generally result in higher death rates than those at the end of the summer (e32). Important risk factors for higher mortality include advanced age; low social status; addictive disorders; restricted mobility; the presence of pulmonary, cardiovascular, or psychogeriatric disease; and chronic renal disease (e33). People who live high up in a building or live alone also have a statistically increased risk (e34). It is now widely accepted that heat stress is particularly dangerous to old, frail, and often (multi-) morbid people and this is a generalized problem in geriatrics (e26).

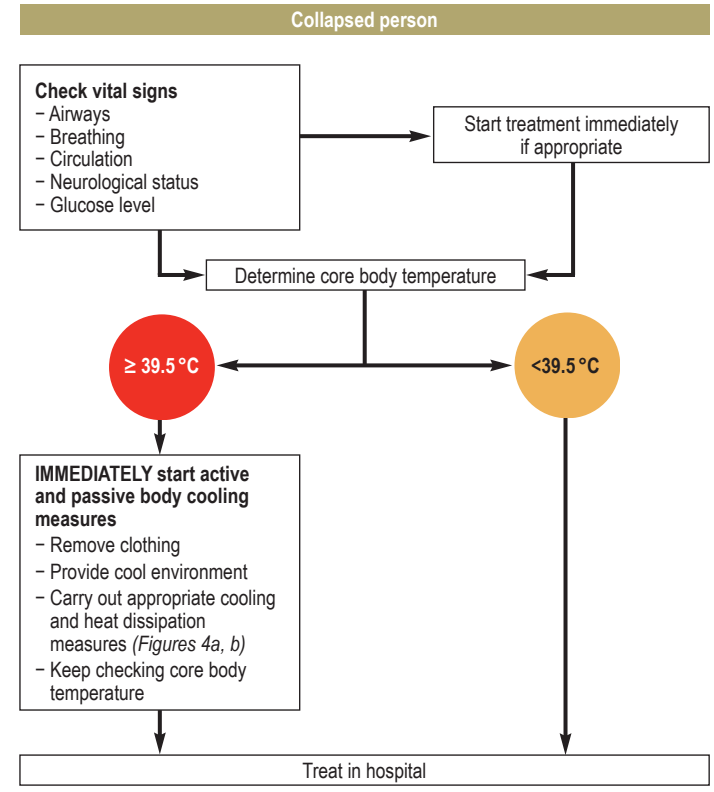
One thing that is not universally known is that prescription drugs can also damage heat resilience (e35). Medical drugs can interfere with at least five important defense mechanisms. The perception of thirst, for example, can be impaired by ACE inhibitors (e36). Opioids, serotonin reuptake inhibitors, carbamazepine, anticholinergics, and tricyclic antidepressants can impair central thermoregulation (e34, e37). Hypohidrosis can be triggered by antimuscarinic agents such as anticholinergics, tricyclic antidepressants, or antipsychotics (e38). Sympathomimetics, by causing cutaneous vasoconstriction, can affect the regulation of skin blood flow (e39). In patients being treated with sedatives (e.g., benzodiazepines, opioids), early recognition of warning symptoms is harder because they already have altered attention and alertness (e26).

It should also be noted that heat affects pharmacokinetics by means of various mechanisms, and thus influences the effective level (concentration) of an active substance in patients (e35). Local heat, for example, can quadruple cutaneous blood flow, increasing the systemic availability of transcutaneously administered drugs (e.g., opioid patches) (e40). The same is true of subcutaneously administered drugs (such as insulin), which are more rapidly released with increased temperature and have a correspondingly stronger effect. Renal and hepatic blood flow may diminish by around one third (e35). The latter affects the bioavailability of orally administered substances with high hepatic extraction rates (i.e., substances with a high first-pass effect), such as beta-blockers.

Overweight and low physical fitness

Overweight and a low fitness level reduce heat tolerance markedly (1–3, 14, 15, 19, 29). Impressive results were presented by Bedno et al., who studied the occurrence of heat illness in 9455 male US army recruits during their first 180 days of service. These authors showed that fitness and weight status are independently associated with the occurrence of heat illness. In comparison to trained normal-weight recruits, untrained normal-weight recruits had twice the risk of heat illness. Trained overweight recruits had an almost four-fold increased risk, and untrained overweight recruits an almost eight-fold increased risk of a heat event (2, e41).

FIGURE 2



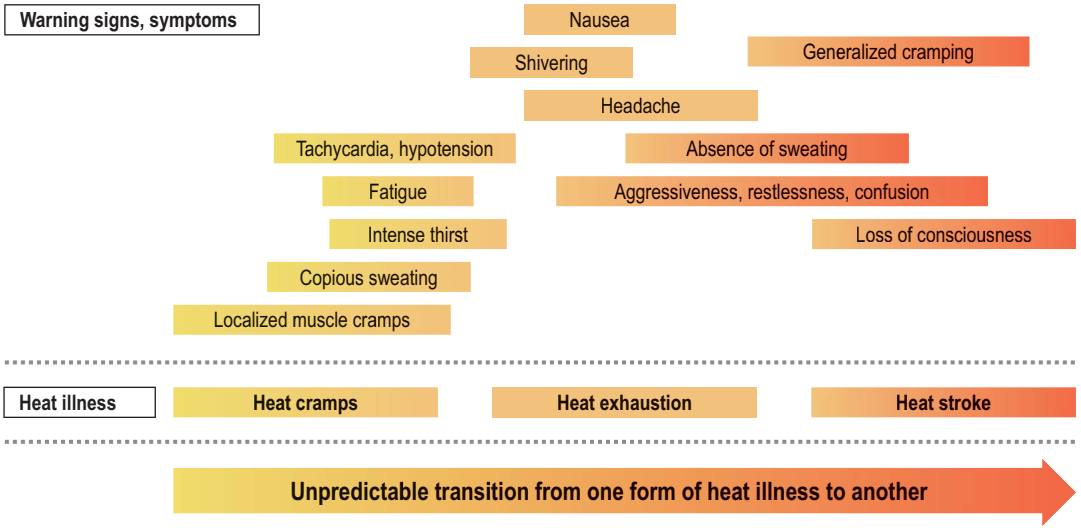
Flowchart for use in a case of suspected exertional heat stroke (36, e57–e59)

Risk of heat stress in sport and at work

Physical exertion leads to a considerable increase in heat production. In athletes during maximal exertion, rectal temperatures of >41 °C have been reported (6, e42–e45). Most of these reports come from clinical case reports. In studies, testing usually has to be stopped before a core body temperature of 39.0 °C (16) is reached.

In long-distance events (marathon or half-marathon), considerable fluid/electrolyte loss can occur, even in temperate conditions, as can exertional heat stress (10, 11, 36, e46). Dangerous heat stress can also occur in other popular sports (such as tennis or football), for example if older people or those with health-related risk factors play to the limit of their capacity (e46, e47). In some areas of professional sport (2019 Australian Open tennis tournament in

FIGURE 3



Symptoms of heat illnesses. Not all the symptoms may appear, and they may not appear in any given order. The various forms of heat illness may develop independently and apparently without warning; i.e., this is not a regular sequence of recognizable stages of illness (adapted from [e78, e81]).

Melbourne; 2014 Football World Championship in Brazil), heat/cooling-down breaks have been introduced (e48–e50). Increased risks exist in other sports in which protective clothing is used (fencing, motor racing, and others). Every year, heat stress–related deaths are seen in American football (e45, e51, e52).

In the world of work, many kinds of industrial jobs are carried out in hot work environments (e.g., steel, glass, and ceramics production). Less well known is heat exposure in work environments where high humidity is added to the ambient thermal stress (kitchens, laundries, sculleries, etc.). The protective clothing worn by firefighters, police, military personnel, and also some medical personnel (barrier nursing, etc.), which insulates and impedes the loss of heat through evaporation, also leads to the risk of overheating, requiring restrictions on the length of time for which they can be worn (e53) or else microclimate body cooling (27). All over the world, people working in civil engineering, or in agricultural or forestry work, are at risk of heat illness (7, 8) and the carcinogenic effects of UV light (e54, e55).

Diagnosis and treatment of heat illness

Heat stroke, the most dangerous form of heat illness, can be successfully treated if the condition is diagnosed as quickly as possible and cooling measures are started before the patient is transferred to hospital (*Figure 2*). When heat stroke is suspected, it is extremely important to monitor core body temperature, preferably rectally (e56). There is no single agreed method of clinically determining mean skin temperature, but this should still be considered, in addition to skin color and the presence or absence of sweating, to assess thermoregulatory status (body core versus body shell). Typical symptoms of heat illness are shown in *Figure 3*; the different forms of heat illness can arise independently, and sudden deterioration can occur (17, e57). For example, there may be a sudden transition from red skin color (skin blood flow present) to pallor (circulation centralized to the core). In suspected cases, nothing, not even rectal temperature measurement, should be allowed to delay the immediate start of cooling treatment (36, e57–e59).

Sunstroke

Direct sun on the uncovered head for a long period can lead to heat stress of the brain, resulting in inflammation of the meninges or even to brain edema (e60). Sunstroke should be seen as primarily a localized condition and is not directly caused by a significant rise in body temperature (e57, e61). Depending on severity, symptoms range from overheating of the head with headache, dizziness, restlessness, nausea, and meningism to altered consciousness and cerebral seizures (e60, e62, e63).

Heat cramps

Heat cramps usually affect the local working muscles, and like heat edema, heat rash, and heat collapse/syncope they are regarded as a mild form of heat illness (e64). Heat cramps are painful muscle contractions and cramps during physical exertion; typically there is no systemic build-up of heat and core body temperature is often normal (e65). In sports that involve much running (e.g., football, marathons), calf cramps are common, whereas in tennis it may be the muscles of the forearm and/or hand that are affected (37, e66). The key element

is the combination of intense sweating, electrolyte loss, and a negative fluid balance (e67, e68). Insufficient acclimatization with an elevated electrolyte concentration in the sweat increases the risk of heat cramps (e69). The symptoms of heat cramp, including weakness, headache, and nausea, can occur simultaneously with heat exhaustion (40).

Heat collapse/syncope

Heat collapse/syncope is a risk especially in people who stand for a long time in a hot environment (e64). The heat leads first to dehydration and to redistribution of blood into peripheral sections of the circulation, especially with a strong increase in skin blood flow. This can cause a drop in blood pressure and cerebral blood flow, triggering syncope (e70). Laying the patient flat (with or without elevation of the legs), loosening the clothing, moving the patient into a cool environment, and if necessary giving infusions, will quickly bring this relatively minor health event under control.

Heat exhaustion and heat stroke

Excessive sweating and dehydration during physical exertion in a warm environment are typical of heat exhaustion (e71). Usually only low-grade cerebral symptoms are present (e.g., dizziness). Immediate interventions include heat dissipation (removing clothing, cooling measures), intravenous fluid administration, and monitoring of vital signs (34). Monitoring core body temperature and the cerebral status is extremely important, as heat exhaustion can occasionally progress to heat stroke.

At core body temperatures >40.0 °C, endothelial cells become increasingly damaged, resulting in capillary leak (e72, e73). In heat stroke, this is the trigger for pathophysiological processes with systemic effects that can culminate in multiorgan failure (e72–e74). The generalized endothelial cell damage has effects on various organ systems, and can lead to multiple organ failure via the systemic inflammatory response (SIRS) (e72, e73, e75, e76). Predisposing factors and symptoms of exertional heat stroke are shown in the *Box* and in *Figure 3* (further information is provided in [15, e77–e80]).

Immediate initiation of cooling is the only causal treatment (*Figure 4a, b*). The longer core body temperature remains above >40 °C, the poorer the expected outcome (36, e76, e82, e83). The aim of treatment is to bring down the core body temperature below 40 °C within 30 minutes (the "golden half hour") (e84). Whole-body immersion in iced water is the recommended intervention with the most rapid cooling effect (*Figure 4a, b*). Because the start of treatment is time-critical, it should be carried out as fast as possible: clothing can be removed once the patient is immersed (e85). Other, less effective ways of cooling include immersion in tepid water, immersion of the torso, cold packs on the torso, and so on (e23, e79, e86–e89). The success of treatment depends on the greatest possible temperature gradient between the body core and the cooling intervention (e90). Patients with exertional heat stroke are typically young and without pre-existing cardiovascular disease. No cardiac events due to cooling interventions have been described. Once core body temperature has reached 38–39 °C, the cooling interventions should be halted so as to avoid a further temperature drop due to blood returning from the periphery to the core (afterdrop) (e91). When cooling measures in the form of ice water immersion are implemented without delay, further treatment purely on an outpatient basis has been described (e92–e97).

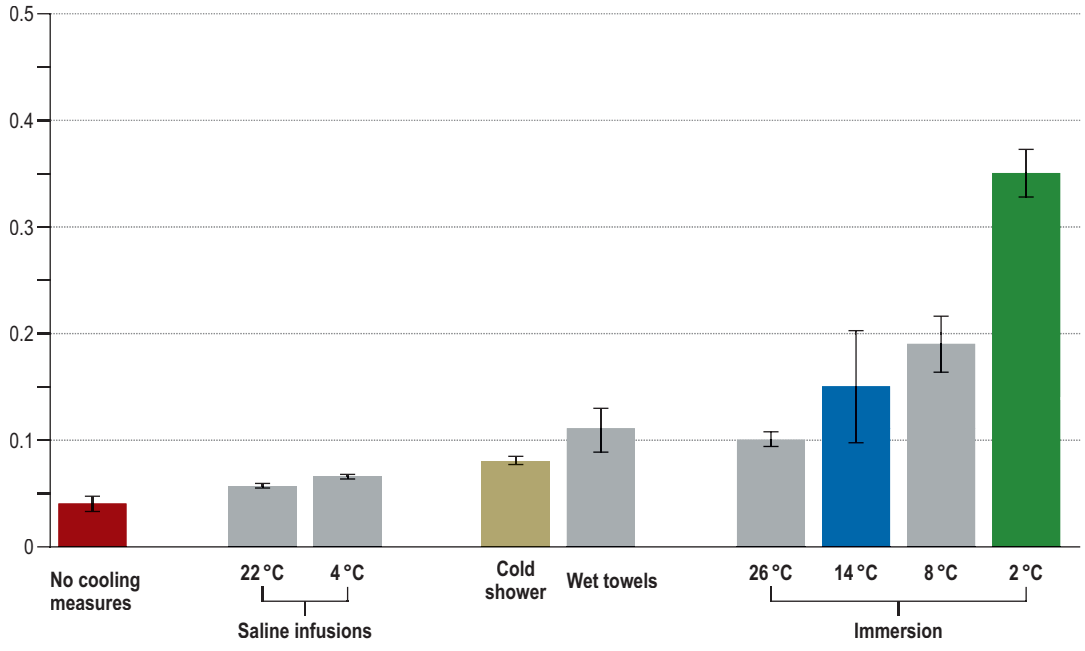
If there is a delay in starting treatment, or symptoms are initially misinterpreted, morbidity and mortality rise sharply (e83, e98). Lost time cannot be made up. In patients admitted to emergency departments, cooling measures should be continued with monitoring of core body temperature at close intervals until it has reached 38–39 °C (e23, e87, e99, e100). The clinical (inpatient) course—especially in cases when the start of treatment has been

BOX

Predisposing factors (with examples) of exertional heat stroke (adapted from [15, e79, e80])

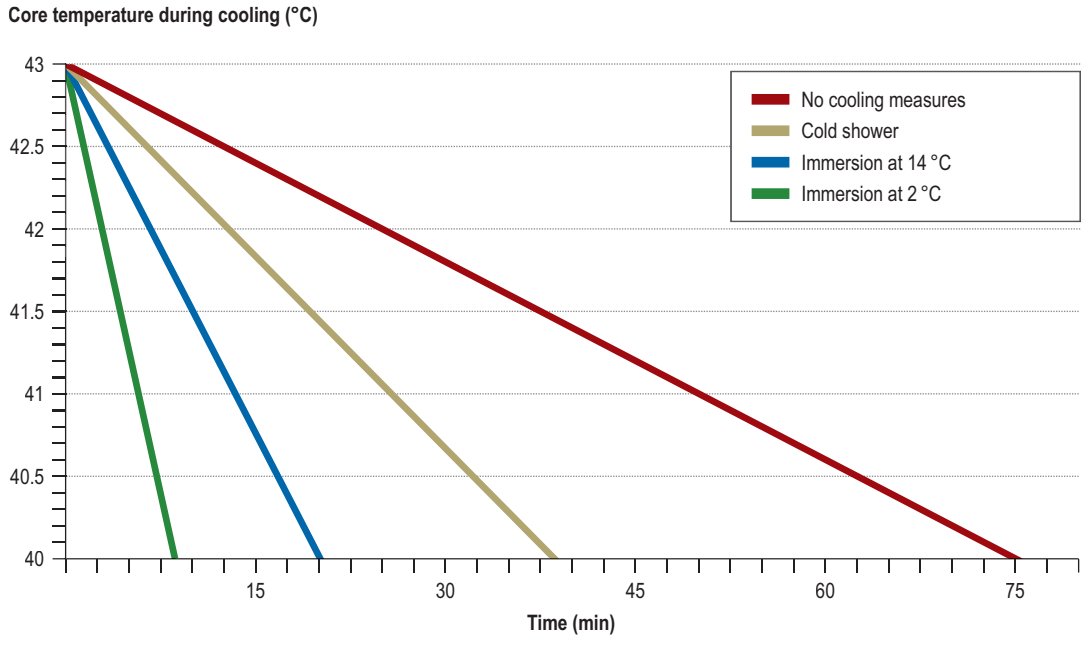
- **Environmental factors**
  - High environmental temperature
  - High humidity
  - No air movement
  - No shade
- **Constitutional factors**
  - Age
  - Overweight
  - Low fitness level
  - No heat acclimatization
  - Dehydration
  - Lack of sleep
- **Medical drugs and other substances**
  - Alcohol
  - Antihistamines
  - Benzodiazepines
  - Beta-blockers, and many more
- **Health status**
  - Viral infection
  - Diabetes mellitus
  - Feverish illness
  - Cardiovascular disease
- **Mental factors**
  - Strong desire to succeed/overmotivation accompanied by (as yet) insufficient performance capacity, e.g., in athletes and military personnel

FIGURE 4a



Rate of cooling of the human body using various cooling methods. The comparison is for general guidance only, since no standardized comparison is possible for the studies involved (e42, e93–e95, e97) in terms of initial body temperature and patient population characteristics (age, sex, body mass, body surface area to volume ratio, etc.). For the saline infusions, 2 × 1 L 0.9% saline solution was used.

FIGURE 4b



Model of reduction over time in core body temperature (adapted from [e96], assuming constant mean cooling rates [e93, e97]), the same initial temperature (43 °C), and the same time of treatment initiation. The chosen endpoint was the lower temperature threshold for cell damage in human beings (40 °C).

delayed—often requires all the options offered by intensive medicine, up to and including organ transplantation, because of the multiorgan failure (e101, e102). In cases where inpatient treatment is required, life expectancy appears to be shorter even when the patient makes a good initial recovery (e103). No pharmacological alternative to immediate cooling interventions exists. Dantrolene is not an option (e104). Antipyretics do not help either, since the problem is not a fever, but overheating caused by muscle work (e105).

Summary

Heat-related health events can develop quickly, especially during physical exertion (even at apparently innocuous ambient temperatures), and can lead to life-threatening heat stroke. Successful clinical treatment of heat stroke requires the core body temperature to be reduced below 40 °C within the first 30 minutes, and this requires aggressive cooling interventions. Prevention is better than cure with heat illness, and this means effective preventive measures (drinking, acclimatization, reduced exposure, etc.) (16), and terminating the heat stress (e.g., sport or work activity) in good time.

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Conflict of interest statement
The authors declare that no conflict of interest exists.

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Key messages

- Even in moderate ambient temperatures, physical exertion can lead to exertional heat stress, dramatic loss of performance, and risks to health.
- The risk of heat-related health events is higher in sports and work activities involving intense metabolic heat production and/or in which heat dissipation is restricted.
- The combination of overweight with low physical fitness increases the risk of a heat-related health event by a factor of eight. Children and older people are also at increased risk.
- Heat tolerance can vary greatly within a single person: acute infections and disease, dehydration, insufficient acclimatization, intake of prescription drugs, and other factors can all cause problems with heat.
- The only causal treatment for heat stroke is to reduce core body temperature (<40 °C within 30 minutes); there is no pharmacological treatment (such as with dantrolene for malignant hyperthermia). In suspected cases, cooling measures should be started immediately.

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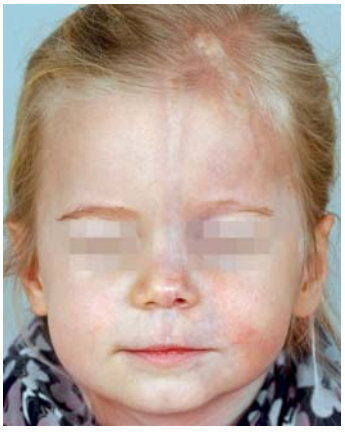
CLINICAL SNAPSHOT

Scleroderma en Coup de Sabre



A 3-year-old girl was brought to us with facial cutaneous lesions. The initial manifestation was an erythematous macula on the nose, followed 18 months later by formation of a hypopigmented atrophic scar extending from the hairline to the jaw. This was accompanied by craniofacial asymmetry and malposition of the jaw. Overall, the clinical and histological findings pointed to linear circumscribed scleroderma (scleroderma en coup de sabre). This is a sclerosing connective-tissue disease of the skin that may also affect other tissues such as bone. Scleroderma en coup de sabre can result in neurological symptoms such as headache or seizures and may even cause structural alterations in the brain. In our patient, gliosis in the area of the basal ganglia was diagnosed with the aid of magnetic resonance imaging. The differential diagnosis includes, for example, hemiatrophia faciei progressiva.
An attempt at treatment with systemic glucocorticoids and methotrexate is planned.

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eBOX

Method

The selective literature search was carried out on PubMed without restriction (no filters). The *eTable* shows the various search terms used during the primary search. Guidelines and expert recommendations were also included.

Search terms within the various topic clusters ("hyperthermia," "climate," "risk factors," and "prevention/treatment/diagnosis") were first combined into clusters using the OR operator. A total of four independent search strategies were followed by then combining each topic cluster separately with the superordinated main topic "heat illness" using the AND operator.

eTABLE

Search strategy for the heat illness topic cluster

Heat illness Heat illness/heat injury/heat stroke/heat strain/exertional heatstroke/exertional heat illness			
Hyperthermia	**Climate**	**Risk factors**	**Prevention/treatment/diagnosis**
Exercise-heat stress	Air motion	Body heat exchange	Acclimatisation
Exertional heat illness	Ambient temperature	Clothing	Acclimatization
Exertional heatstroke (EHS)	Climate	Dehydration	(Body) core temperature
Heat casualties	Climate change	Exercise	Body temperature regulation
Heat cramps	Climatic change	Heat production	Compensable heat stress (CHS)
Heat collapse	Climate indices	Metabolic heat production	Clothing
Heat exhaustion	Environmental heat stress index	Metabolic rate	Cooling
Heat illness	Heat radiation	Military	Dehydration
Heat injury	Heat waves	Motivation	Electrolyte replacement
Heat load	Humidity	Obesity	Environmental exposure
Heat-related illness	Temperature	Occupation	Fitness
Heat stress	Thermal regulatory model	Occupational exposure	Fluid replacement
Heat stress disorder	Universal thermal climate index (UTCI)	Overweight	Heat adaptation behavior
Heat stress syndrome	Wet bulb globe temperature (WBGT)	Physical activity	Heat exposure
Heat syncope	Wet bulb globe temperature index	Protective clothing	Heat (stress) management
Heat strain	Wind speed	Risk factor	Heat shock proteins
Heat stroke		Sports	Heat tolerance
Thermal strain		Thermal insulation	Hydration
Thermal stress		Work load	Hyponatremia
Uncompensable heat stress (UCHS)			Physical fitness
			Prevention
			Rehydration
			Rectal temperature
			Rest cycles
			SIRS
			Sweat evaporation
			Sweat rate
			Sweating rate
			Therapy
			Thermoregulation
			Work cycles