Journal of Medical Sciences and Health Care Research
ISSN: 3068-9104

PRIME OPEN ACCESS

Volume 3, Issue 3

Research Article

Date of Submission: 10 Jul, 2026
Date of Acceptance: 19 Aug, 2026
Date of Publication: 07 Sep, 2026

Hyperthermia Occurs When the Body Overheats

Siniša Franjić*

Independent Researcher, Europe

***Corresponding Author:** Siniša Franjić, Independent Researcher, Europe.

Citation: Franjić, S. (2026). Hyperthermia Occurs When the Body Overheats. *J Med Sci Health Care Res, 3*(3), 01-05.

Abstract
Hyperthermia refers to a hazardous rise in body temperature that transcends the normal range, caused by excessive generation, intake of heat, or inadequate dissipation, frequently due to extreme environmental conditions, dehydration, vigorous exercise, certain medications, and it does not react to fever-reducing medications. This condition has the potential to cause heat stroke and necessitates prompt cooling and medical care; however, hyperthermia is also employed therapeutically to eliminate cancerous cells.

Keywords: Hyperthermia, Fever, MT, Heat Stroke, Health

Introduction

Fever is characterized by an elevation in body temperature due to a pathophysiological reaction that raises the body's typical thermoregulatory threshold [1]. This threshold can fluctuate based on factors such as age, sex, the time of day, and several other influences. While there isn't a single, universally accepted definition for what constitutes a fever, a common benchmark is a temperature of ≥ 38. 3°C. Regardless of the specific cutoff used, it's crucial to acknowledge that body temperature can differ based on the measurement method employed. Since measurements taken from peripheral sites like the armpit or skin can fall short of reflecting core temperature by at least 1°C, consistent monitoring of core temperature is advisable for patients who are susceptible to severe temperature increases and when accurate temperature regulation is necessary.

Fever represents a variant of hyperthermia induced by a reconfiguration of thermoregulation, typically from infection, and is defined as a core temperature exceeding 38.3°C [2]. Hyperpyrexia refers to a temperature that reaches or exceeds 40°C. Heatstroke is a medical condition that arises from exposure to significantly elevated environmental temperatures, particularly if it is unusual for the individual, and when the body's ability to regulate temperature is compromised. This can result from:

- Damage to the hypothalamus, such as from tumors, surgical procedures, strokes, or infections of the central nervous system.
- Increased heat production, which occurs in 15%–50% of surgical patients and signifies a postoperative inflammatory reaction.

- Sepsis.
- Exercise.
- Drug-induced conditions, including malignant hyperthermia, neuroleptic malignant syndrome, salicylate toxicity, cocaine toxicity, and ingestion of methylenedioxymethylamfetamine.
- Hyperthyroidism.
- Pheochromocytoma.
- Status Epilepticus.
- Tetanus.

- Hindered heat loss, which can be due to:

- Autonomic Neuropathy.
- Drug-induced effects from anticholinergic medications, phenothiazines, or neuroleptic malignant syndrome.
- Dehydration.
- Excessive heat application during anesthesia.

Symptoms of heatstroke include confusion, headaches, loss of consciousness, absence of sweating, and temperatures exceeding 40. 6°C. Hyperventilation may lead to metabolic acidosis, seizures, and cardiovascular issues, as well as multiple organ dysfunction syndrome.

Hyperthermia

In a clinical setting, distinguishing between fever and hyperthermia can be challenging [1]. Hyperthermia arises when body temperature is raised while the thermoregulatory set point stays within the normal range. It results from exposure to heat in the environment, increased internal heat production, and impaired heat dissipation.

Heat loss, or a mix of factors, suggests that the body's natural temperature-regulating processes have been exceeded. Increased body temperature, whether due to fever or hyperthermia, is frequently observed in individuals admitted to the intensive care unit (ICU). While infections should always be a primary consideration with elevated body temperatures, a wide array of noninfectious causes should also be taken into account. Although numerous patients experience fever during their stay in the ICU, ongoing fever is rare, except in those with neurological issues. For patients without such issues, fever usually resolves in one to two days unless there is a collection of pus that has not been drained. There are some exceptions worth noting. Persistent high fevers lasting days or weeks are often linked to influenza pneumonitis and are typically seen in patients suffering from severe necrotizing pancreatitis. Fever is notably frequent and often enduring among patients with various neurological conditions, such as subarachnoid hemorrhage, stroke, and traumatic brain injury. In younger individuals with severe traumatic brain injuries, managing persistent high fevers, along with tachycardia and hypertension, can become challenging during the weeks following the initial brain injury. One rare neurological disorder associated with consistently high body temperature is anti–N-methyl-D-aspartate (NMDA) receptor autoantibody encephalitis. This condition is important to highlight because individuals affected may require extended intensive care, and significant elevation of body temperature can sometimes occur as part of the illness.

A patient in the ICU with an elevated body temperature should undergo a clinical evaluation while considering the differential diagnosis. Although fever can be present in patients with significant myocardial infarctions and thromboembolic disorders, the mere presence of fever does not justify extensive diagnostic testing for these conditions, as the specificity is low. Since fever is a major indicator of infection and sepsis is a common and frequently deadly complication in critical care, it is crucial to monitor for any new or worsening organ dysfunction alongside potential infection sources. While other diagnostic options must always be explored, the appearance of elevated body temperature in a critically ill patient serves as a vital prompt to investigate for sepsis. In this context, the temperature threshold for initiating such investigations should be lowered as the clinician's assessment of infection likelihood increases. If there is a high enough suspicion of infection, sepsis related investigations should proceed regardless of the body temperature, and antibiotics should be administered without delay.

In addition to identifying and addressing the cause of an elevated body temperature, an important consideration for healthcare providers is deciding whether to take action to lower the temperature. The clinician must evaluate both the level of temperature increase and the strength of evidence supporting intervention for the specific medical condition at hand.

A hyperthermia diagnosis signifies that the body's ability to regulate temperature has been compromised. Immediate action through physical cooling is necessary. The greater the increase in body temperature observed in a hyperthermia patient, the faster and more forcefully intervention should occur. Should malignant hyperthermia be diagnosed, dantrolene must be given without delay.

It is crucial to differentiate between hyperthermia and fever, as hyperthermia can quickly become life-threatening and typically does not improve with antipyretic medications [3]. Unfortunately, there is no quick method to differentiate the two. Often, hyperthermia is identified based on recent events leading to a rise in core temperature, such as exposure to heat or the administration of medications that disrupt thermoregulation. Besides the patient's medical history, certain physical signs of hyperthermia can alert healthcare providers. For instance, patients experiencing heat stroke or those using medications that inhibit sweating may exhibit skin that is both hot and dry. Additionally, while antipyretics fail to lower the temperature in hyperthermia, they usually lead to a temperature reduction in fever, and even in cases of hyperpyrexia, when sufficient doses of acetaminophen or aspirin are given.

Body Temperature

The typical oral body temperature measured around midmorning averages at 36. 7°C (with a range of 36–37. 4°C) [4]. This range reflects a mean value alongside two standard deviations, capturing 95% of the population considered normal

(normal daily temperature fluctuation is about 0. 5–1°C).

A standard rectal or vaginal temperature reading is generally 0. 5°C greater than the oral temperature, while an axillary temperature is 0. 5°C lower. Nonetheless, a normal reading from a peripheral thermometer (tympanic membrane, temporal artery, axillary, oral) does not necessarily rule out a fever. Rectal temperature measurements are more dependable than oral methods for ruling out fever, especially in individuals who breathe through their mouths, those who are tachypneic, or in critical care scenarios where a rectal probe can be utilized to identify fever.

The regulation of body temperature occurs through various processes involving heat loss and heat generation, managed by the hypothalamic thermoregulatory center [5].

- Hyperthermia arises when the body's mechanisms for releasing heat are surpassed, when there is excessive heat production, or through a combination of these factors. Essentially, the body's inherent cooling processes fail to lower the temperature to the intact hypothalamic "set point. "

On the other hand, fever is characterized by an elevation of the hypothalamic set point, often triggered by infection or inflammation. Individuals with a fever may feel cold and shiver because their actual body temperature is lower than the set point. The physiological response at 37 °C (98. 6 °F) is similar to that of 35 °C (95 °F) when the set point is raised to 39 °C (102. 2 °F), since in both instances the body temperature remains 2 °C below the designated "set point. " As the "set point" normalizes, the individual begins to feel warm and sweat. The process of a fever "breaking" represents the body's natural mechanism to reduce temperature back to the updated set point. This section primarily examines hyperthermia, rather than fevers.

In very uncommon instances, damage to the hypothalamus could lead to issues with temperature regulation.

- The typical range for core body temperature is maintained between 36 and 38 °C (96. 8–100. 4 °F), while regulatory systems are significantly less efficient when temperatures exceed 40 °C (104 °F).
- When body temperature elevates, there are mainly four physiological reactions: expansion of blood vessels, heightened perspiration, a reduction in heat generation, and changes in behavior.

The methods through which heat is transferred away from the body include:
- **Radiation:** The movement of heat from a hotter object to a cooler one through electromagnetic waves.
- **Conduction:** The exchange of heat between two surfaces that are in direct touch with one another.
- **Convection:** The transfer of heat by air or liquid that flows over another object's surface.
- **Evaporation:** The loss of heat via the vaporization of moisture or sweat.

MH

A condition initially identified by Denborough in 1961, characterized by elevated temperature and stiffness during anesthesia [2]. The reported occurrence ranges from 1 in 5,000 to 1 in 200,000. It arises from unusual skeletal muscle contractions along with increased metabolism affecting both muscle and other tissues. It is inherited in an autosomal dominant manner; in 50% to 70% of families affected, the genetic predisposition is traced to the long arm of chromosome 19, associated with the ryanodine/dihydropyridine receptor complex located at the T-tubule and sarcoplasmic reticulum junction in striated muscle. This receptor controls the movement of calcium in and out of the sarcoplasmic reticulum; however, in MH, this regulation fails, causing an excessive influx of calcium that prompts uncontrolled muscle contractions. The susceptibility to MH is also genetically linked to central core disease, a rare type of muscular dystrophy. Several additional gene mutations have been associated, which complicates the reliability of genetic testing for MH.

MH can be triggered by exposure to specific agents, especially volatile anesthetics and suxamethonium, although it is believed that just a single dose of suxamethonium alone does not induce the syndrome. It might occur in individuals who have previously undergone anesthesia without complications. Stress and vigorous exercise may also instigate MH. Therefore, patients' reactions to triggering substances can vary over time. Responses have been documented as late as 11 hours after surgery.

This condition is most frequently seen in younger individuals undergoing surgical procedures on bones and muscles, including trauma operations. It remains unclear if this is indicative of a pre-existing muscle abnormality that makes them more prone to trauma. Surgical procedures in young patients often address issues such as squints and orthopedic conditions, and the increased prevalence in this demographic may simply reflect their first experience with anesthesia in at-risk individuals.

Heat Stroke

The most critical condition associated with heat stress is heat stroke [6]. Although there is some similarity between heat stroke and heat exhaustion, those with heat stroke experience hyperthermia along with central nervous system disturbances. In studies involving populations, this diagnosis has a mortality risk of up to 10%. Heat stroke is classified

as a medical emergency and may manifest through symptoms like loss of appetite, dizziness, extreme tiredness, headaches, general discomfort, nausea, vision changes, or weakness. More alarming signs consist of the absence of sweating, abnormal heart rhythms, liver failure, high body temperature, neurologic symptoms (such as lack of coordination, unconsciousness, confusion, irritability, seizures), fluid accumulation in the lungs, kidney failure, muscle breakdown, shock, rapid heart rate, and fast breathing. Core body temperature frequently ranges from 40° to 44°C, with some instances showing even higher readings. Although the absence of sweating is a characteristic feature of classic heat stroke, it should not be considered a definitive indicator, as some individuals may still sweat while being affected by this condition.

In severe cases, heat stroke can result in dysfunction of multiple organs. Elements linked to high mortality rates include delays in seeking medical help, postponing the start of treatment, and increased severity of illness at the time of presentation.

Heat stroke is divided into two categories: classic and exertional. Classic heat stroke typically occurs during periods of high temperature, is more prevalent among older and weakened individuals, and usually develops over days rather than in hours or minutes. The cause of this form is linked to exposure to external heat. In contrast, physical exertion is generally not a factor in classic heat stroke development. Patients are frequently unable to sweat due to the gradual onset of the condition. Exertional heat stroke usually affects healthy young adults who engage in vigorous activities in hot, humid conditions without adequate acclimatization. It arises from internal heat production (i. e., metabolic processes) and is often seen in athletes, firefighters, factory workers, and military trainees.

Poisoning

From a pathophysiological perspective, there are several mechanisms through which exposure to poison and subsequent toxicity can lead to hyperthermia [7]. Impairments in normal mental functioning, cognitive processes, and increased psychomotor activity may render patients oblivious to their body temperature or external heat. They might neglect to avoid physical activities in hot climates, become incapable of exiting the environment, or still exert themselves while confined. For instance, this can occur when a person becomes comatose in a parked car on a sunny day or collapses on a hot surface like asphalt, where heat absorption can happen swiftly. Such situations often arise with commonly abused substances like alcohol, cocaine, opioids, and PCP (pneumocystis carinii pneumonia). The heightened psychomotor agitation resulting from these drugs can lead to significant increases in heat production.

These incidents are understandably more prevalent during warmer months, such as summer in the Northern Hemisphere and winter in the Southern Hemisphere. There is a clear connection between fatalities due to cocaine-related hyperthermia and ambient temperatures: for example, in New York City, deaths linked to cocaine hyperthermia sharply increase during the hottest summer months and are seldom seen at other times. This pattern likely holds true for other substances that result in psychomotor agitation and elevated body temperature as well.

Other physiological processes leading to hyperthermia comprise the disconnection of oxidative phosphorylation, as observed in toxicity from salicylates or dinitrophenol; heightened metabolism resulting from thyroid hormone or thyroid extract toxicity; reduced sweating induced by antihistamines and anticholinergic medications; and blood vessel narrowing due to alpha-adrenergic agonists, which include substances like amphetamines, cocaine, pseudoephedrine, and other sympathomimetic agents.

Even though the distinct physiological processes causing hyperthermia vary, the preliminary acute management approaches remain largely consistent. The specific core temperature at which irreversible neurological damage may occur for any individual is uncertain; however, a core temperature of 107°F or 42°C necessitates swift cooling strategies, ideally through submersion in ice or an ice bath. Less effective techniques like tepid sponging, mist sprays, and fans are to be considered only in rare situations where true ice immersion is unachievable.

Generally, individuals experiencing hyperthermia due to psychomotor agitation, increased metabolism, or disassociated oxidative phosphorylation typically do not express discomfort while in an ice pack or ice bath. After a while, as their temperature drops, they might indicate feelings of coldness or discomfort, often aligning with achieving a temperature goal of 100–102°F. It is crucial to monitor these patients diligently to prevent cooling that falls below the normal body temperature range.

Management

Malignant hyperthermia represents a genuine emergency, with mortality rates linked to the extent and duration of the peak temperature [8]. Even slight delays in treatment can result in fatalities. Continuous temperature monitoring must occur until it is stabilized below 39°C. Any exposure to possibly harmful volatile anesthetic agents and neuromuscular blocking drugs should be halted as soon as possible, and if feasible, any surgical procedure should be discontinued. In rare instances where surgery cannot be halted, medications considered to be safe include nitrous oxide, barbiturates, benzodiazepines, pancuronium, and opioids. Initiating ventilation with 100% oxygen must happen without delay. Employing direct external cooling with a mist of fanned water helps dissipate heat while waiting for definitive treatment with dantrolene. Shivering instigated by external cooling measures can be managed through the administration of

benzodiazepines and nondepolarizing neuromuscular blockers.

Dantrolene prevents excessive heat production by blocking the release of calcium from the sarcoplasmic reticulum. A starting dose of 2. 5 mg/kg should be given rapidly and may be repeated at intervals of 5 minutes until the symptoms diminish or the maximum safe dose of 10 mg/kg is achieved. Typically, when treatment and recognition are timely, improvement is seen quickly. (If there is no improvement despite doses of 10 mg/kg, it should prompt reconsideration of the diagnosis). After the initial treatment, additional doses of 1 mg/kg every 4 to 6 hours should be maintained for a duration of 36 to 48 hours. In cases where dantrolene is not effective or acts slowly, cooling methods applied to the surface of the body may be utilized. Dantrolene's inhibition of calcium release has the potential to induce muscle weakness that might be severe enough to hinder the ability to breathe spontaneously. Consequently, careful consideration is necessary when extubating patients who have received dantrolene treatment.

Patients experiencing malignant hyperthermia typically maintain a normal level of intravascular volume, which means their fluid needs are low. Although this is a topic of discussion, administering fluids to aid in urine production and using sodium bicarbonate for serum alkalinization could help prevent kidney damage from myoglobinuria. For hyperkalemia, standard treatments can be applied initially, including sodium bicarbonate to correct pH, insulin with glucose to help move potassium into cells, and calcium to stabilize cell membranes. Managing underlying issues is the most effective way to treat arrhythmias unrelated to hyperkalemia, although the use of lidocaine and β-blockers might be beneficial. Given the involvement of calcium metabolism issues in this condition, it is advisable to avoid the use of calcium channel blockers.

Conclusion

The human body operates optimally when its internal temperature remains in a secure, stable range. There are instances when the body overheats and cannot effectively dissipate heat. This hazardous increase in temperature is termed hyperthermia. Hyperthermia arises when the body becomes excessively hot and fails to release that heat in a timely manner. Consequently, internal body temperature rises, potentially affecting key organs, including the brain, heart, and kidneys. Hyperthermia is defined as a state where body temperature exceeds normal values, typically above 38 °C or 100. 4 °F. For the majority of individuals, the typical body temperature ranges from 36. 5 °C to 37. 5 °C or 97. 7–99. 5 °F. When temperature surpasses this range and continues to escalate, it can pose challenges to normal bodily functions. If hyperthermia is not addressed swiftly, it can become a threat to life.

References

1. Young, P. (2024). Fever and hyperthermia. In J.-L. Vincent, F. A. Moore, R. Bellomo, & J. J. Marini (Eds.), Textbook of critical care (8th ed., pp. 20–23). Elsevier.
2. Yentis, S. M., Hirsch, N. P., Ip, J. (2019). Anaesthesia, Intensive Care and Perioperative Medicine A-Z - An Encyclopaedia of Principles and Practice, Sixth Edition", Elsevier Limited, Edinburgh, UK, pp. 290; 369.
3. Dinarello, C. A., & Gelfand, J. A. (2005). Fever and hyperthermia. In D. L. Kasper, E. Braunwald, A. S. Fauci, S. L. Hauser, D. L. Longo, & J. L. Jameson (Eds.), Harrison's Principles of Internal Medicine, 16th Edition", The McGraw-Hill Companies, Inc., New York, USA, pp. 105.
4. Nadler, P. L., & Gonzales, R. (2025). Common symptoms. In M. A. Papadakis, M. W. Rabow, K. R. McQuaid, & M. Gandhi (Eds.), CURRENT medical diagnosis & treatment 2025 (64th ed., pp. 15–41). McGraw Hill.
5. Smith, B., & Michalik, M. (2019). Hyperthermia:"I'm Hot Blooded; Check It and See". In Case Studies in Emergency Medicine: LEARNing Rounds: Learn, Evaluate, Adopt, Right Now (pp. 275-284). Cham: Springer International Publishing.
6. Beltran, G. W. (2021). Heat-related illness. In D. C. Cone, J. H. Brice, T. R. Delbridge, & J. B. Myers (Eds.), Emergency medical services: Clinical practice and systems oversight (3rd ed., Vol. 1, pp. 403–409). Wiley.
7. Hoffman, R. J. (2011). The critically ill poisoned patient. In D. A. Farcy, W. C. Chiu, A. Flaxman, & J. P. Marshall (Eds.), Critical care emergency medicine (pp. 427–434). McGraw-Hill.
8. Marini, J. J., & Dries, D. J. (2018). Critical care medicine: the essentials and more. Lippincott Williams & Wilkins.