StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-.

Physiology, Temperature Regulation

Authors

Eva V. Osilla[1]; Jennifer L. Marsidi; Karlie R. Shumway[2]; Sandeep Sharma[3].

Affiliations

[1] University of South Alabama, DeBusk COM
[2] Henry Ford Health
[3] Mery Fitzgerald Hospital

Last Update: July 30, 2023.

Introduction

Thermoregulation is the maintenance of physiologic core body temperature by balancing heat generation with heat loss. A healthy individual will have a core body temperature of 37 +/- 0.5°C (98.6 +/- 0.9°F), the temperature range needed for the body's metabolic processes to function correctly.[1]

The human body's thermostat is the hypothalamic thermoregulatory center, which, more specifically, is located in the preoptic area of the hypothalamus. This center sets the body's set point and regulates temperature homeostasis. The hypothalamus contains temperature sensors, which receive information via nerve cells called thermoreceptors. The body has peripheral and central thermoreceptors. The peripheral thermoreceptors are located in the skin and sense surface temperatures, while central thermoreceptors are found in the viscera, spinal cord, and hypothalamus and sense the core temperature. Variations in body temperature activate these thermoreceptors, which inform the preoptic area of the hypothalamus. This area then activates heat regulation mechanisms to increase or decrease body temperature and return it to baseline.[2]

Issues of Concern

Disruption of the body's ability to thermoregulate can lead to temperatures that are too low (hypothermia) or too high (hyperthermia). Slight temperature variations can be reversible with behavior changes and physiologic responses, while extreme variations can ultimately lead to organ failure, coma, and/or death.

It is important to note that temperature varies throughout the body, with the core body temperature being higher and more stable and the skin temperature being lower and more variable due to external factors. Typically, the lowest body temperature occurs at 4 AM, and the highest body temperature occurs at 6 PM.[3]

Cellular Level

The definition of 'fever' is an elevation in core body temperature above a set point, which is set by the preoptic area of the hypothalamus in the thermoregulatory center. Numerous causes can precipitate a fever, including infection, inflammation, autoimmune processes, medications, or malignancy. These processes involve the release of immunological mediators, which trigger the thermoregulatory center of the hypothalamus, leading to an increase in the body's core temperature.[4]

The febrile response is mediated by pyrogens, substances that induce fever. Exogenous pyrogens originate outside the body and induce interleukins. Endogenous pyrogens originate inside the body and act on the thermoregulatory center in the hypothalamus. The primary endogenous pyrogens (cytokines) include interleukin-1 (IL-1) and interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-alpha).[5]

These pyrogens induce cyclooxygenase 2 (COX2), which catalyzes the formation of prostaglandins (PG) from arachidonic acid. Prostaglandin E2 (PGE2) then stimulates the release of neurotransmitters (cAMP), increasing body temperature. Interferon-gamma (IFN-gamma) is another proinflammatory cytokine that can directly cause fever via induction of IL-1 and synthesis of TNF.[6]

Development

The fetal hypothalamus and the thermoregulatory center of the hypothalamus form from the ventral diencephalon and are differentiated by approximately 9 to 10 weeks gestation.[7] Though central thermoregulatory mechanisms are present at birth, they are deemed both developmentally deficient and poorly differentiated, which places the newborn, especially those that are small and premature, at risk of severe heat loss and hypothermia.[8]

Newborns rely on nonshivering thermogenesis to metabolize brown adipose tissue (BAT) and release energy. BAT is well-vascularized tissue that develops at the beginning of the third trimester, at approximately week 27 of gestation. BAT is located around the kidneys, adrenal glands, scapulae, axilla, mediastinum, and spine. Newborns are more susceptible to cooling and hypothermia due to their high levels of heat loss by evaporation due to skin immaturity, elevated ratio of body surface area to weight, deficient amounts of subcutaneous adipose tissue for insulation, and poorly developed muscles. Additionally, immature respiratory and circulatory systems may impede proper temperature regulation. There is limited capacity for thermoregulation during the first few weeks of life, especially in preterm and low birthweight infants.[9]

Organ Systems Involved

The primary organs and organ systems that maintain thermoregulation include the brain (hypothalamus), skin, skeletal muscles, sweat glands, and the vascular, endocrine, and nervous systems.

Function

Thermoregulation is a homeostatic process that maintains a steady internal body temperature despite changes in external conditions. Maintaining a body temperature within a tight range (between 36.5 to 37.5°C) allows for the enzymes and immune responses of the body to maintain proper functionality.[10]

Mechanism

The mechanism of thermoregulation involves afferent sensing, central control, and efferent responses. Peripheral and central thermoreceptors sense an increase or decrease in body temperature and send this information to the hypothalamus. The body then responds with multiple mechanisms to either dissipate or

generate heat based on the body's needs. The physiological and behavioral responses to the activation of thermoreceptors are as follows.[11]

Increased Body Temperature

The body responds by dissipating heat via:

- Activating sympathetic cholinergic fibers innervating sweat glands, leading to increased sweat and increased heat loss
- Inhibiting sympathetic activity in blood vessels of the skin, causing blood to be shunted to the skin and an increased heat loss
- Decreasing the release of catecholamines from the adrenal glands and thyroid hormones from the hypothalamus, leading to a reduced metabolic rate
- Behavioral changes include reducing movements, adopting an open body position, removing clothing, and reducing appetite

Decreased Body Temperature

The body responds by generating heat via:

- Activating the sympathetic nervous system which causes vasoconstriction of skin arterioles, causing blood to bypass the skin and leading to a decreased loss of heat. Additionally, adrenal glands will release catecholamines (epinephrine, norepinephrine), leading to increased metabolic rate and heat production. Piloerection (goosebumps) also occur, leading to heat-trapping.
- Releasing thyroid hormones from the hypothalamus causes an increased metabolic rate and subsequent heat production.
- Activating the primary motor center in the posterior hypothalamus causes skeletal muscle contraction and shivering, leading to increased heat production.
- Non-shivering thermogenesis using brown adipose tissue (BAT) in the first six months of life.
- Behavioral changes include increased movements, adopting a closed body position, adding clothing, and an increased appetite.

The methods by which heat is lost from the skin to the external environment occur via radiation, conduction, convection, and evaporation.

Radiation

Heat loss via radiation occurs in the form of infrared rays and accounts for approximately 60% of total body heat loss. When the body temperature exceeds the surrounding temperature, heat is radiated from the body in greater quantity than is radiated to the body.

Conduction and Convection

Heat loss via conduction occurs through the air (approximately 15%) or by direct contact with a solid object (approximately 3%). After heat is conducted into the air, it is carried away by air currents (convection). A minute amount of convection almost always occurs, with a 15% heat loss through air encompassing both conduction and convection.

Evaporation

Heat loss via evaporation of sweat is regulated by the amount and rate of sweating and accounts for approximately 22% of total body heat loss. 0.58 kilocalories of heat is lost for each gram of evaporated water. Even when not sweating, water still evaporates from the skin and lungs at a rate of 600 to 700 mL/day, causing continual heat loss.

Related Testing

The thermoregulatory sweat test (TST) is a specific clinical test used to diagnose certain conditions that cause abnormal temperature regulation and defects in sweat production in the body. The TST assesses a patient's central and autonomic nervous system to determine if the thermoregulatory centers are working correctly by measuring a patient's ability to produce sweat in a controlled, heated, and humid environment. To perform a TST, the patient is first coated in an indicator powder that will change color when sweat is produced. They are then placed in a chamber that slowly rises in temperature. The patient's sweat pattern results are then documented by digital photography.[12]

Abnormal TST patterns can indicate a dysfunction in the autonomic nervous system (ANS), with certain differentials including hyperhidrosis, small fiber and autonomic neuropathies, multiple system atrophy, Parkinson disease with autonomic dysfunction, and pure autonomic failure.[13]

Methods of testing and monitoring temperature are critical in determining if there is dysfunction in the thermoregulatory processes of the body. The most accurate and precise measurement is desired in order to diagnose, monitor, and treat patients at both outpatient and inpatient levels. Listed below are temperature monitoring sites throughout the human body, from most accurate and precise to poor. Sites close to highly perfused organs or great vessels are deemed the most reliable in measuring core temperature.

Best

- Site: pulmonary artery
- Instrument: Swan-Ganz catheter
- Advantages: precise and repeatable
- Disadvantages: invasive and restricted to ICUs

In the clinical setting, the core temperature measured by a pulmonary artery Swan-Ganz catheter is the most precise technique for measuring temperature, as the pulmonary artery carries blood directly from the body's core.[1]

- Site: esophagus
- Instrument: long esophageal thermistor
- Advantages: easy to use, repeatable, widely available

- Disadvantages: high latency

Due to its location near the left atrium and left ventricle, the esophagus is a preferred location to determine temperature measurement for intubated patients and is strongly associated with the temperature of the pulmonary artery. Temperature measurement via esophageal thermistor may be favored due to its quick reaction to changes in core temperature. Deciding which gold standard temperature measurement to use in the clinical setting depends on the patient's status and available resources.

Good

- Site: nasopharynx
- Instrument: nasopharyngeal temperature probe
- Advantages: widely available and easy to use
- Disadvantages: high risk of measurement error due to suboptimal positioning
    - Previous studies have reported that nurses and residents optimally positioned nasopharyngeal temperature probes in the upper or mid-nasopharynx in only 41 to 43% of cases.[14]

The temperature of the central blood measured by a pulmonary artery catheter is not routinely used due to its restriction to intensive care units, so a nasopharynx temperature probe is commonly used to monitor temperature during general anesthesia due to its proximity to the internal carotid artery (ICA).[15]

- Site: urinary bladder
- Instrument: urinary catheter
- Advantages: repeatable and precise
- Disadvantages: high latency

For critically ill patients whose condition requires an indwelling urinary catheter, one with temperature-sensing abilities will allow both continuous drainage of urine and continuous measurement of their body temperature without the need for additional equipment.[16] Of note, temperature-sensing urinary catheters perform well during steady thermal states. They do not perform well during rapid thermal changes, as seen during cardiopulmonary bypass, in which rapid cooling and rewarming occur.[17]

- Site: rectum
- Instrument: rectal probe
- Advantages: repeatable and precise, widely available, easy to use
- Disadvantages: high latency

Once widely used, especially in children, research has now shown rectal temperatures to be a poor indicator of accurate core body temperature. The rectal probe must be properly introduced to a depth of 15 cm or greater to allow the temperature sensors to be near the large arteries of the pelvic region. Rectal temperature readings have shown to be higher than those in other parts of the body and are a measurement that is reliable only in conditions of normothermia. Research has demonstrated a considerable delay in rectal temperature results, especially during rapid temperature changes, and many things can alter a rectal temperature reading, such as rectal inflammation or the presence of feces.

Additionally, pain and discomfort of the patient typically occur during the insertion of the rectal probe, especially in those with rectal or perirectal infections or inflammation. Patients may experience a feeling of fullness and desire to defecate, and the insertion process can be scary and psychologically harmful for children or those mentally disabled. Not only has rectal thermometry been associated with delayed recordings in changing temperatures, but it has also been linked to the spread of enteric pathogens.[18][19]

- Site: tympanic membrane
- Instrument: specially insulated thermistor
- Advantages: repeatable, precise, and a good indicator of brain core temperature
- Disadvantages: high risk of measurement error

When performed correctly, temperature readings via the tympanic membrane are quick and straightforward, with the readings only slightly affected by environmental temperature. This method of temperature reading is safer than oral or rectal thermometers and is the most accurate commercially available system to measure core body temperature regularly. Interestingly, when placed in the left ear, the tympanic thermometer provides a more accurate body temperature reading.[20] A study comparing pulmonary artery temperature measurement to other methods showed that infrared ear thermometers offered a comparatively close assessment of pulmonary artery temperature, making them the gold standard for outpatient and home use.

Poor

- Site: axilla, oral, and body surface
- Advantages: widely available and easy to use
- Disadvantages: inaccurate

While oral probes are widely used, they are highly inaccurate. Oral temperature readings do not accurately reflect core body temperature, as many significant factors influence readings, including air temperature, ingestion of fluids, food, or tobacco, and probe placement. Oral temperature probes should not be used in emergency situations when temperature readings should be highly accurate, such as in exertional heat stroke, as the temperature readings will grossly underestimate temperature and delay proper diagnosis and treatment.[21]

Pathophysiology

Disorders of thermoregulation and associated autonomic pathways can increase the risk of cold or heat-related illnesses. Such disorders include small fiber neuropathies, spinal cord injuries, central nervous system (CNS) disorders, and endocrine disorders.[22] Conditions that cause decreased sweating (hypohidrosis) or a complete lack of sweating (anhidrosis) can lead to severe overheating, as the body is unable to sweat and thermoregulate in times of extreme heat. For example, this can be seen in patients with Sjögren syndrome, as this disorder can lead to chronic inflammatory atrophy of sweat glands and can result in decreased sweating.[23]

CNS disorders leading to hypo- or anhidrosis may include multiple sclerosis (MS), Parkinson disease (PD), and Shy-Drager syndrome, while peripheral neuropathies leading to hypo- or anhidrosis include diabetes mellitus, Fabry disease, Guillain-Barre syndrome, and Ross syndrome.

Whether selectively or disproportionally, many peripheral neuropathies affect autonomic fibers, including those involved in eccrine gland innervation. The most prevalent peripheral neuropathy in developed countries is diabetic neuropathy, which affects approximately 50% of diabetic patients, of which 10% experience autonomic neuropathy such as distal anhidrosis. Fabry disease is an X-linked lysosomal storage disorder in which the peripheral nervous system can be affected, presenting as neuropathic pain and reduced sensation to cold and heat.[24]

Guillain-Barré syndrome is an autoimmune response affecting the peripheral nerves, which can lead to various autonomic nerve dysfunctions, including anhidrosis. Ross syndrome is a rare disorder of the peripheral nervous system characterized by a triad of segmental anhidrosis, tonic pupil, and hyporeflexia. Other small fiber neuropathies that can cause thermoregulatory problems include hereditary, toxic, drug-related, paraneoplastic, autoimmune, and idiopathic neuropathies.

Multiple sclerosis (MS) is a progressive neurological disorder characterized by disruption of axonal myelin in the CNS, leading to nerve scarring and lesions. MS lesions can also occur in the region of the brain responsible for thermoregulation. MS can impair the neural control of autonomic and endocrine functions. Approximately 70% of patients affected by MS note a temporary worsening of clinical signs and symptoms when exposed to heat.[25] Additionally, MS may produce impaired neural control of autonomic and endocrine functions, causing neural-induced changes in sweat glands and a lack of sweating.[25]

Parkinson disease (PD) patients can experience both hypohidrosis and hyperhidrosis, with the latter being more prevalent. The exact pathophysiology is still unclear but is thought to be related to the dysfunction of the autonomic nervous system seen in PD.[26] Shy-Drager syndrome is a multiple-system atrophy disorder mainly characterized by idiopathic orthostatic hypotension but also includes anhidrosis, iris atrophy, bowel incontinence, and rigidity.

Impaired thermoregulation is a well-known complication seen in individuals with a spinal cord injury (especially those with an injury about the level of T6), traumatic brain injury, or stroke. Spinal cord injury patients experience a loss of connections between the hypothalamus and its motor and sensory projections. Additionally, a high spinal cord injury leaves most of the skin without physical sensation, leaving these patients unable to sense hot or cold temperatures and adjust their surroundings accordingly. Furthermore, there is a lack of sympathetic outflow, which leads to a loss of vasodilation and/or vasoconstriction and, therefore, an inability to conserve or lose heat when the core temperature changes. Heat production is reduced due to less muscle mass, and heat loss is reduced due to the redistribution of blood and a decreased sweating capacity below the level of the spinal lesion.[27]

Clinical Significance

Hyperthermic syndromes caused by medications include serotonin syndrome, neuroleptic malignant syndrome, anticholinergic toxidrome, and malignant hyperthermia.

Serotonin syndrome develops from the use of serotonergic antidepressants, and it is typically seen within 24 hours of starting or changing therapy. Physical exam findings include hyperthermia, hyperreflexia, and myoclonus. Treatment includes stopping the offending drug, cooling methods, and administering cyproheptadine, a 5HT-2 receptor antagonist.

Neuroleptic Malignant Syndrome (NMS) develops from using neuroleptics (dopamine antagonists), with signs and symptoms typically occurring within the first few weeks of treatment.[28] Physical exam findings include hyperthermia, lead pipe rigidity, and hyporeflexia. Treatment includes stopping the offending drug, cooling methods, and administering a D2 agonist (bromocriptine).

Anticholinergic toxidrome is caused by ingesting medications with anticholinergic properties, including antihistamines, antidepressants, Parkinson drugs, mydriatics, antispasmodics, and antipsychotics. Physical exam findings include hyperthermia, flushing, anhidrosis, dry mucous membranes, mydriasis, urinary retention, and altered mental status. Treatment is generally supportive and symptom-specific, with the administration of physostigmine utilized for severe toxicity.[29]

Malignant Hyperthermia (MH) occurs due to a genetic alteration of ryanodine receptor 1 (RYR1) in the muscle cells, leading to skeletal muscle hypermetabolism upon exposure to depolarizing muscle relaxants (succinylcholine), halogenated anesthetics (halothane, isoflurane, desflurane, enflurane, ether, or sevoflurane), or, rarely, excessive heat or vigorous exercise.[30]

In MH, once a cell depolarizes, the defective RYR1 becomes hyperactivated, causing excessive calcium release, inappropriate muscle contraction, and increased metabolic rate, all leading to excessive heat production. The treatment of MH is to stop the offending drug and immediately administer dantrolene, a postsynaptic muscle relaxant.[31] Rapidly cooling the patient, giving 100% oxygen, and regulating metabolic acidosis are also important.

Additionally, some medications can inhibit sweating and increase the risk of thermoregulatory dysfunction. Carbonic anhydrase inhibitors, such as acetazolamide and topiramate, can cause transient hypohidrosis and lead to heat intolerance, especially in children. M3 anticholinergic agents such as bladder antispasmodics, tricyclic antidepressants, and neuroleptics can also lead to heat intolerance.

Drugs such as salicylate and methyl salicylate can cause hyperthermia by uncoupling oxidative phosphorylation. Lastly, the recreational use of psychomotor stimulants is known to frequently cause hyperthermia. These drugs include amphetamine, methamphetamine, cocaine, heroin, and 3,4-methylenedioxy-methamphetamine, also known as MDMA or ecstasy.

Hyperthyroidism is a condition of an overactive thyroid gland that can lead to altered thermoregulation. An overactive thyroid gland releases excess T4 and T3, hormones that affect the basal metabolic rate of cells. Excess T4 and T3 lead to an increased basal metabolic rate, thus increasing the body temperature, ATP turnover, and oxygen consumption.

Fever is an elevation in body temperature due to changes in the hypothalamic set-point.

Below is a summary of the categorization of fever. Based on the source, these figures may have slight variations.[4]

- **Low grade:** 37.3 to 38.0°C (99.1 to 100.4°F)

- **Moderate grade:** 38.1 to 39.0°C (100.6 to 102.2°F)
- **High grade:** 39.1 to 41°C (102.4 to 105.8°F)
- **Hyperthermia:** Greater than 41 C (105.8°F)

A fever occurs when pyrogens act on the hypothalamus and release prostaglandins, which increase the hypothalamic set-point, causing the body temperature to rise and reach a new baseline. The benefits of a fever include inhibiting bacterial growth by making growing conditions less favorable and increasing the efficiency of immune cells.

Aspirin reduces fever by inhibiting prostaglandin production

Hyperthermia is an unregulated elevated body temperature due to an imbalance between heat loss and heat production. Interleukins are not involved in hyperthermia, as they are in fever, which is why there is a normal hypothalamic set-point in hyperthermia. This differs from fever, in which the hypothalamic set-point is elevated.

Heat Exhaustion is a heat-related illness caused by excessive sweating, which leads to loss of water and electrolytes and a decreased blood volume. Without replacement of water and electrolytes, this will lead to decreased arterial pressure and feelings of dizziness, muscle cramps, and fainting.

Heat Stroke is a temperature >40°C (104°F) accompanied by dry, hot skin and central nervous system abnormalities such as convulsions, delirium, and/or coma. An alternative definition is that heatstroke is a form of hyperthermia associated with a systemic inflammatory response that leads to multiorgan dysfunction, especially encephalopathy.[32]

Heat stroke can be exertional or non-exertional. Exertional heat stroke tends to occur in athletes, outdoor laborers, those in the military, and anyone performing rigorous physical activity in hot environments. Non-exertional heatstroke tends to develop in elderly individuals and those with obesity, diabetes, heart disease, renal disease, hypertension, dementia, and alcoholism.

Neurogenic fever, or posttraumatic hyperthermia, is a non-infectious fever in a brain injury patient, especially in those with a hypothalamic injury, stroke, or lesion. This is a diagnosis of exclusion.[33]

Hypothermia is an involuntary drop in core body temperature below 35°C (95°F). Mild hypothermia is defined as a core temperature of 32 to 35°C (89.6 to 95°F), moderate hypothermia is 28 to 32°C (82.4 to 89.6°F), severe hypothermia is 24 to 28°C (75.2 to 82.4°F), and pulseless or profound hypothermia < 24°C (75.2°F). In addition to prolonged cold exposure, impaired thermoregulation is a leading cause of hypothermia. Hypothermia caused by impaired thermoregulation can be due to skin disorders, cerebrovascular accidents, neurodegenerative disorders, peripheral neuropathies, spinal cord injuries leading to improper peripheral vasodilation, and drug misuse.[34]

Hypothermia can also be caused by endocrine disorders such as diabetes, hypothyroidism, hypoadrenalism, and hypopituitarism. Those most at risk for hypothermia include elderly and trauma patients, those who are mentally ill, and those who abuse alcohol or drugs. Decreased metabolic rates from malnourishment, severe burns, and hypoglycemia can also cause hypothermia.[35]

Thermoregulation and Age

Clinically, caregivers must remember that patients of extreme spectrums of age (infants and elderly persons) are at higher risk for thermoregulation dysfunction, especially when ill. For example, while heat production and heat loss mechanisms are functional in neonates and children, these processes are easily exhaustible, leading to the development of hypothermia or hyperthermia. Elderly patients have a resting body temperature that is typically lower than that of young adults, by approximately 0.23°C, and they exhibit decreased thermosensitivity, causing delayed or insufficient responses to thermal changes leading them to be more susceptible to hypothermia or hyperthermia.[36]

Of clinical importance is evaluating the mental state of patients. Infection-induced fevers are often absent, and an impaired mental state can be the dominant feature of infection. One may experience hypothermia and severe infection, so it is important not to rely solely on whether a patient has a fever or not to determine if they have an infection.[37]

Hot Flashes

As a woman ages, they will undergo menopause and typically experience hot flashes caused by estrogen withdrawal. Of note, no consistent correlation has been found between plasma estrogen levels and the severity of a woman's symptoms.[37]

Robert Freedman, a professor of psychiatry and obstetrics and gynecology at Wayne State University, has found that women who experience hot flashes tend to demonstrate a more narrow hypothalamic temperature zone between the upper and lower threshold temperatures involved in heat loss and heat production, respectively, which leads to an increased likelihood of crossing these thresholds and developing sweats and chills associated with hot flashes. A pre-menopausal woman has an interthreshold zone of about 0.4°C, in which temperature fluctuations between 0 and 0.4°C will not trigger compensatory chills, flushing, or sweating. However, in a menopausal woman, this threshold zone is significantly reduced, leading to even minute fluctuations in body temperature initiating thermoregulatory responses.[38]

Careful and accurate monitoring of core body temperature is of great importance in patients with thermoregulatory dysfunction to provide appropriate treatment, improve their quality of life, and prevent serious complications from arising.

Review Questions

- Access free multiple choice questions on this topic.
- Comment on this article.

References

1. Hymczak H, Gołąb A, Mendrala K, Plicner D, Darocha T, Podsiadło P, Hudziak D, Gocoł R, Kosiński S. Core Temperature Measurement-Principles of Correct Measurement, Problems, and Complications. Int J Environ Res Public Health. 2021 Oct 10;18(20) [PMC free article: PMC8535559] [PubMed: 34682351]
2. Tabarean I, Morrison B, Marcondes MC, Bartfai T, Conti B. Hypothalamic and dietary control of temperature-mediated longevity. Ageing Res Rev. 2010 Jan;9(1):41-50. [PMC free article: PMC2818054] [PubMed: 19631766]
3.

Lell B, Brandts CH, Graninger W, Kremsner PG. The circadian rhythm of body temperature is preserved during malarial fever. Wien Klin Wochenschr. 2000 Dec 07;112(23):1014-5. [PubMed: 11190710]
4. Balli S, Shumway KR, Sharan S. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Sep 4, 2023. Physiology, Fever. [PubMed: 32966005]
5. Luheshi G, Rothwell N. Cytokines and fever. Int Arch Allergy Immunol. 1996 Apr;109(4):301-7. [PubMed: 8634513]
6. Mühl H, Pfeilschifter J. Anti-inflammatory properties of pro-inflammatory interferon-gamma. Int Immunopharmacol. 2003 Sep;3(9):1247-55. [PubMed: 12890422]
7. Howland MA, Sandman CA, Glynn LM. Developmental origins of the human hypothalamic-pituitary-adrenal axis. Expert Rev Endocrinol Metab. 2017 Sep;12(5):321-339. [PMC free article: PMC6334849] [PubMed: 30058893]
8. Asakura H. Fetal and neonatal thermoregulation. J Nippon Med Sch. 2004 Dec;71(6):360-70. [PubMed: 15673956]
9. Lubkowska A, Szymański S, Chudecka M. Surface Body Temperature of Full-Term Healthy Newborns Immediately after Birth-Pilot Study. Int J Environ Res Public Health. 2019 Apr 12;16(8) [PMC free article: PMC6518189] [PubMed: 31013692]
10. Morrison SF, Nakamura K. Central Mechanisms for Thermoregulation. Annu Rev Physiol. 2019 Feb 10;81:285-308. [PubMed: 30256726]
11. Tansey EA, Johnson CD. Recent advances in thermoregulation. Adv Physiol Educ. 2015 Sep;39(3):139-48. [PubMed: 26330029]
12. Illigens BM, Gibbons CH. Sweat testing to evaluate autonomic function. Clin Auton Res. 2009 Apr;19(2):79-87. [PMC free article: PMC3046462] [PubMed: 18989618]
13. Davis MD, Genebriera J, Sandroni P, Fealey RD. Thermoregulatory sweat testing in patients with erythromelalgia. Arch Dermatol. 2006 Dec;142(12):1583-8. [PubMed: 17178984]
14. Lee J, Lim H, Son KG, Ko S. Optimal nasopharyngeal temperature probe placement. Anesth Analg. 2014 Oct;119(4):875-879. [PubMed: 25025586]
15. Lim H, Kim B, Kim DC, Lee SK, Ko S. A comparison of the temperature difference according to the placement of a nasopharyngeal temperature probe. Korean J Anesthesiol. 2016 Aug;69(4):357-61. [PMC free article: PMC4967630] [PubMed: 27482312]
16. Fallis WM. Monitoring urinary bladder temperature in the intensive care unit: state of the science. Am J Crit Care. 2002 Jan;11(1):38-45; quiz 47. [PubMed: 11785556]
17. Fallis WM. Monitoring bladder temperatures in the OR. AORN J. 2002 Sep;76(3):467-76, 481-6, 488-9. [PubMed: 12227290]
18. Robinson J, Charlton J, Seal R, Spady D, Joffres MR. Oesophageal, rectal, axillary, tympanic and pulmonary artery temperatures during cardiac surgery. Can J Anaesth. 1998 Apr;45(4):317-23. [PubMed: 9597204]
19. Livornese LL, Dias S, Samel C, Romanowski B, Taylor S, May P, Pitsakis P, Woods G, Kaye D, Levison ME. Hospital-acquired infection with vancomycin-resistant Enterococcus faecium transmitted by electronic thermometers. Ann Intern Med. 1992 Jul 15;117(2):112-6. [PubMed: 1605425]
20. Mah AJ, Ghazi Zadeh L, Khoshnam Tehrani M, Askari S, Gandjbakhche AH, Shadgan B. Studying the Accuracy and Function of Different Thermometry Techniques for Measuring Body Temperature. Biology (Basel). 2021 Dec 15;10(12) [PMC free article: PMC8698704] [PubMed: 34943242]
21. Mazerolle SM, Ganio MS, Casa DJ, Vingren J, Klau J. Is oral temperature an accurate measurement of deep body temperature? A systematic review. J Athl Train. 2011 Sep-Oct;46(5):566-73. [PMC free article: PMC3418963] [PubMed: 22488144]
22. Cheshire WP. Thermoregulatory disorders and illness related to heat and cold stress. Auton Neurosci. 2016 Apr;196:91-104. [PubMed: 26794588]
23. Gadarowski MB, Pukhalskaya T, Farah R, Smoller BR. Acquired anhidrosis in a patient with Sjogren syndrome and silicone breast implants. JAAD Case Rep. 2020 May;6(5):414-416. [PMC free article: PMC7200200] [PubMed: 32382632]
24. Jung SE, Kim YC. Hypohidrosis: an early clue in the diagnosis of Fabry disease. Clin Exp Dermatol. 2015 Jun;40(4):444-5. [PubMed: 25546261]
25. Davis SL, Wilson TE, White AT, Frohman EM. Thermoregulation in multiple sclerosis. J Appl Physiol (1985). 2010 Nov;109(5):1531-7. [PMC free article: PMC2980380] [PubMed: 20671034]
26. van Wamelen DJ, Leta V, Podlewska AM, Wan YM, Krbot K, Jaakkola E, Martinez-Martin P, Rizos A, Parry M, Metta V, Ray Chaudhuri K. Exploring hyperhidrosis and related thermoregulatory symptoms as a possible clinical identifier for the dysautonomic subtype of Parkinson's disease. J Neurol. 2019 Jul;266(7):1736-1742. [PMC free article: PMC6586713] [PubMed: 30997572]
27. Price MJ, Trbovich M. Thermoregulation following spinal cord injury. Handb Clin Neurol. 2018;157:799-820. [PubMed: 30459042]
28. Berman BD. Neuroleptic malignant syndrome: a review for neurohospitalists. Neurohospitalist. 2011 Jan;1(1):41-7. [PMC free article: PMC3726098] [PubMed: 23983836]
29. Broderick ED, Metheny H, Crosby B. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Apr 30, 2023. Anticholinergic Toxicity. [PubMed: 30521219]
30. Rosenberg H, Pollock N, Schiemann A, Bulger T, Stowell K. Malignant hyperthermia: a review. Orphanet J Rare Dis. 2015 Aug 04;10:93. [PMC free article: PMC4524368] [PubMed: 26238698]
31. Ratto D, Joyner RW. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Apr 3, 2023. Dantrolene. [PubMed: 30571019]
32. Bouchama A, Knochel JP. Heat stroke. N Engl J Med. 2002 Jun 20;346(25):1978-88. [PubMed: 12075060]
33. Agrawal A, Timothy J, Thapa A. Neurogenic fever. Singapore Med J. 2007 Jun;48(6):492-4. [PubMed: 17538744]
34. Duong H, Patel G. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Jan 19, 2024. Hypothermia. [PubMed: 31424823]
35. Schieber AM, Ayres JS. Thermoregulation as a disease tolerance defense strategy. Pathog Dis. 2016 Dec;74(9) [PMC free article: PMC5975229] [PubMed: 27815313]
36. Geneva II, Cuzzo B, Fazili T, Javaid W. Normal Body Temperature: A Systematic Review. Open Forum Infect Dis. 2019 Apr;6(4):ofz032. [PMC free article: PMC6456186] [PubMed: 30976605]
37. Székely M, Garai J. Thermoregulation and age. Handb Clin Neurol. 2018;156:377-395. [PubMed: 30454602]
38. Freedman RR. Pathophysiology and treatment of menopausal hot flashes. Semin Reprod Med. 2005 May;23(2):117-25. [PubMed: 15852197]

Disclosure: Eva Osilla declares no relevant financial relationships with ineligible companies.

Disclosure: Jennifer Marsidi declares no relevant financial relationships with ineligible companies.

Disclosure: Karlie Shumway declares no relevant financial relationships with ineligible companies.

Disclosure: Sandeep Sharma declares no relevant financial relationships with ineligible companies.

Bookshelf ID: NBK507838 PMID: 29939615