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# Critical Care Nursing of Infants and Children

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# 14 Thermal Regulation

Mary Frances D. Pate



Heat is a natural byproduct of metabolism. It is constantly produced and continuously lost to the environment. When the quantity of heat produced is equal to the amount lost, homeostasis exists. If heat production and heat loss are not in balance, body temperature will rise or fall.

Normal thermoregulatory function serves to maintain body temperature within a narrow range. Both environmental and maturational factors can cause or contribute to *ineffective thermoregulation*—the inability to maintain normal body temperature in the presence of adverse or changing environmental factors.[1] Critically ill infants and children are at high risk for ineffective thermoregulation resulting from both environmental and maturational factors (Table 14-1). Critical care nurses play an essential role in protecting patients from adverse environmental factors and in identifying patients at risk from maturational factors that may impair effective thermoregulation.

Many patients in the pediatric intensive care unit (PICU) may experience a *risk for altered body temperature*—the state in which the individual is at risk for failing to maintain body temperature within a normal range.[1] Abnormal body temperature may be the result of illness (such as infection, surgery, or shock) or its therapy.

Internal factors that contribute to or are risk factors for altered body temperature include pathophysiologic and treatment-related factors. For example, traumatic brain injury or congenital central nervous system (CNS) malformations may produce recurrent, transient elevations in body temperature. In contrast, certain hypothalamic lesions produced by cerebrovascular hemorrhage, neurosurgical procedures, or tumors may result in low body temperature, resulting from decreased ability to produce heat. Furthermore, a depressed or injured CNS results in a diminished response to cold and minimizes shivering as a means of generating heat.

Shock states limit peripheral perfusion in response to hypotension and endogenous catecholamine-mediated subcutaneous vasoconstriction. These responses reduce peripheral blood flow, thereby diminishing heat dissipation and resulting in increased core temperature.

Treatment-related factors leading to alterations in body temperature include medications (e.g., vasopressors or sedatives), parenteral fluids or blood transfusions, renal dialysis, anesthesia, and surgery. For instance, anesthesia inhibits most of the body's heat-producing and heat-conserving mechanisms. When infants or children are unconscious or paralyzed (either pharmacologically or nonpharmacologically), postural changes are no longer possible because muscular activity is obliterated. Anesthetic agents may also depress the hypothalamic thermoregulatory center,

causing vasodilation and depression of the metabolic rate, thereby reducing heat production and increasing heat loss.[2] Induced hypothermia for extracorporeal circulation, exposure to cold ambient temperature, and exposure of the thoracic cavity during open heart surgery are specific treatment-related risk factors for infants and children. The consequences of intraoperative hypothermia are often not manifested until the postoperative phase. Small incremental drops in temperature markedly increase oxygen consumption at a time when oxygen supply to tissues is marginal, resulting in hypoxemia.[3]

Pediatric critical care nurses are in a position to appreciate a variety of environmental, situational, and individual factors that can result in either altered body temperature or ineffective thermal regulation. Care of infants and children based on an understanding of the dynamic interface between the patient and the environment can maximize the nurse's ability to assist patients in maintaining a normal body temperature despite the stress of illness. Regardless of the thermoregulatory issue with which the nurse is confronted, interventions that are evidence based are critical to the well-being of the patient.

**TABLE 14-1 Factors Related to Ineffective Thermoregulation**

| Environmental | Maturational |
|---|---|
| Changing environmental temperature | Extremes of age |
| Insufficient heating or humidification | Large ratio of body surface area to body mass |
| Physical contact with or proximity to cold or warm objects | Metabolic immaturity and decreased heat production |
| Wet or exposed body surfaces | Rapid metabolic rate |
| Excessive or insufficient clothing or coverings | Thin layer of subcutaneous fat |

## ESSENTIAL PHYSIOLOGY OF TEMPERATURE REGULATION

### Physiologic Control of Body Temperature

Heat production and heat loss are controlled in two states. First, the transfer of heat to the body skin surface from the central core establishes an internal thermal gradient. Second, heat is dissipated from the skin surface to the surrounding environment. This balance is critical to normal thermoregulatory function and is summarized in Fig. 14-1.

Body temperature regulation is controlled almost exclusively by intricate nervous system feedback mechanisms located in the hypothalamus. Heat-sensitive neurons located in the preoptic area of the hypothalamus are the body's most influential temperature receptors. These receptors respond to rising temperature by increasing their impulse output and to falling temperature by decreasing their output.

Additional temperature receptors found in the skin consist of both warmth and cold receptors. There are 4 to 10 times as many cold as warmth receptors. These receptors convey nerve impulses to the hypothalamus, where the information is used to regulate body temperature. Receptors in the spinal cord itself, the abdomen, and other internal body structures also transmit signals, primarily cold signals, to the CNS to help in temperature control. Peripheral thermoreceptors dispatch signals to the posterior hypothalamus, where they are integrated to control heat loss and heat production.[4] This "hypothalamic thermostat" is the primary temperature control mechanism in the body.

**The Body's Response to Heat.** Overheating of the hypothalamic thermostatic area increases the pace of heat loss by two essential processes. The first prompts the sweat glands to boost evaporative heat loss from the body. The second inhibits sympathetic centers in the posterior hypothalamus. This reaction allows vasodilation and, consequently, increased heat loss from the skin.[4]

**The Body's Response to Cold.** When the body cools down to a normal temperature (37° C), several mechanisms reduce heat loss and escalate heat production. Vasoconstriction of the epidermal vessels is one of the body's earliest

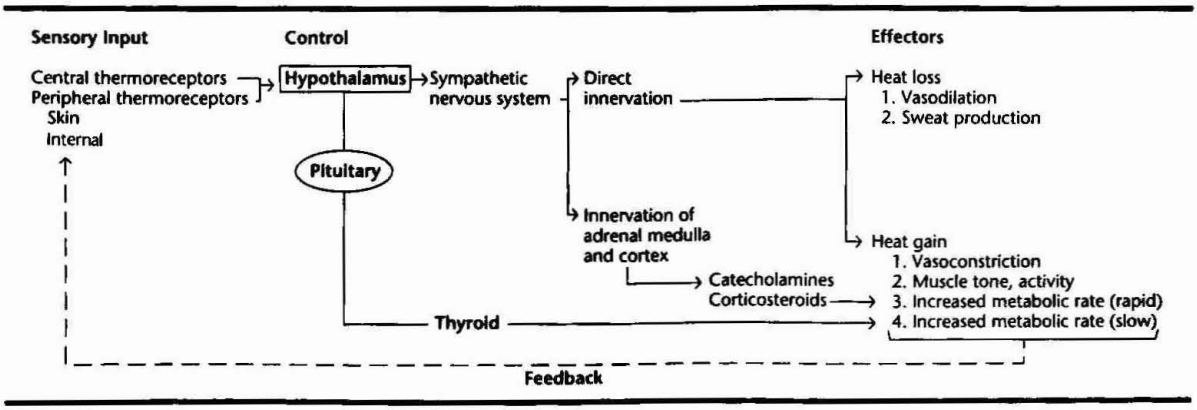


**Fig. 14-1** Thermoregulation. (Reprinted by permission of *Neonatal Netw* 13:15, 1994, Fig. 1.)

efforts to enhance heat conservation. The posterior hypothalamus mobilizes the sympathetic nervous system and initiates powerful vasoconstriction throughout the body. This effect decreases conduction of heat from the internal core to the skin. When vasoconstriction develops, the only heat loss that persists is that via the fat insulators of the skin. Vasoconstriction can diminish heat loss eightfold and can very forcefully conserve heat. When the temperature of the hypothalamic thermostat falls below normal body temperature, the elimination of sweating is absolute. This response arrests evaporative cooling except for insensible evaporation (e.g., from the respiratory tract).[4]

The body also increases heat production in the event of cold stress. This occurs in three distinct ways when body temperature drops below 37° C. First, hypothalamic stimulation of shivering occurs. The primary motor center for shivering is located in the posterior hypothalamus. Cold stress stimulates and heat inhibits this nerve center. When muscle tone is increased to a critical level in response to cold stress, shivering begins. As a result, heat production can increase 4 to 5 times the normal amount.

Second, chemical thermogenesis commences. The rate of cellular metabolism increases as a result of sympathetic stimulation or circulating epinephrine. In the adult, this generally accounts for an increase in heat production of no more than 10% to 15%. In infants, however, chemical thermogenesis can increase the rate of heat production as much as 100% and is a crucial mechanism.[4]

Thermogenesis in infants is different than in older children or adults. In infants, brown fat is the biochemical substance used in chemical thermogenesis. Brown fat cells are approximately one-half the size of white fat cells. Brown fat is found in the subcutaneous tissue, adjacent to the major blood vessels of the neck, abdomen, and thorax, between the scapulae, and in large quantities in the suprarenal areas. At birth, 2% to 6% of the infant's body weight consists of brown fat.[2] Brown fat cells contain finely scattered lipid droplets and are rich in cytoplasm in mitochondria, which facilitates energy transformation and heat production.

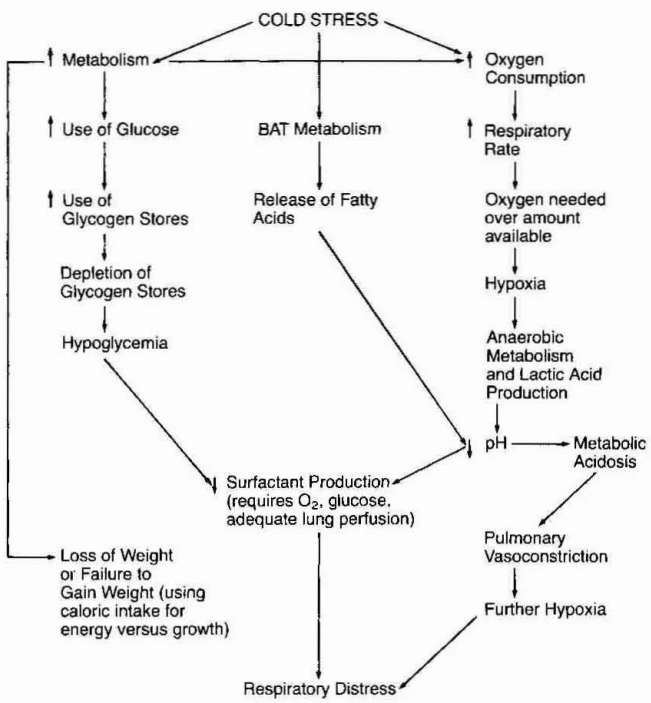


**Fig. 14-2** Physiologic consequences of cold stress in infants. *BAT,* Brown adipose tissue. (From Blackburn ST, Loper DL: *Maternal, fetal, and neonatal physiology,* Philadelphia, 1992, WB Saunders, p 692.)

Cold stress produces a release of norepinephrine and thyroid hormones. This response in turn triggers a lipolytic process in the brown fat stores. Triglycerides in the fat are broken down into fatty acids and glycerol. These fatty acids then enter the thermogenic pathways that produce the common pool of metabolic acids. Besides thermogenesis, glycolysis may be stimulated, resulting in a transient increase in serum glucose levels. Because infants are unable to shiver or actively alter their environment, they depend on nonshivering (chemical) thermogenesis, that is, use of brown fat, to increase heat production.[5]

If cold stress is sustained, increased thyroxine production results in an elevated rate of cellular metabolism throughout the body. This mechanism requires several weeks to become operative, and thus it cannot be considered a primary response to cold stress.[4] (See Fig. 14-2 for a summary of physiologic consequences of cold stress.)

## Behavioral Control of Body Temperature

The most obvious thermoregulatory responses are behavioral.[6] When the temperature of the preoptic area of the hypothalamus rises, this produces the sense of being warm; cooling of the skin and possibly other receptors produces the awareness of being cold. Older children and adolescents who experience either of these sensations usually take steps to reestablish a feeling of comfort.

Effective behavioral control of temperature depends on both an intact sensory-motor system and an ability to communicate perceptions. For example, regulation of body temperature is inadequate below the level at which the sympathetic nerves leave the cord in spinal cord transection. This occurs because the hypothalamus can no longer control skin blood flow or the degree to which sweating is possible. To maintain thermal homeostasis, the affected person needs to rely on responses to cold and hot sensations in the region of the head to make suitable behavioral and environmental adaptations.[4]

Critically ill infants and children have a limited ability to alter their environment in response to their perception of temperature variations. Moreover, their ability to communicate their perceptions is often limited by their developmental stage and the severity of their illness.

## Mechanisms of Thermoregulation

The normal body temperature measured rectally is 37° C. Practitioners have assumed that rectal temperatures are 1° C higher and that axillary temperatures are 1° C lower than

**Box 14-1**

**Comparable Clinical Temperatures in a Resting Afebrile Subject With Rectal Temperature as a Reference**

Rectal approximately 37° C
Oral 0.3°-0.5° C lower than rectal
Esophageal 0.2° C lower than rectal
Pulmonary artery 0.2°-0.3° C lower than rectal
Tympanic membrane 0.05°-0.25° C lower than rectal
Bladder temperature 0.1°-0.2° C lower than rectal
Axillary temperature 0.6°-0.8° C lower than rectal

From Holtzclaw BJ: Monitoring body temperature, *AACN Clin Issues Crit Care Nurs* 4:49, 1993; Holtzclaw BJ: New trends in thermometry for the patient in the ICU, *Crit Care Nurs Q* 21:18, 1998.

oral temperatures. However, studies have demonstrated that the difference is considerably less[7] (Box 14-1). The core body temperature varies by 1.1° C during the day, with the highest temperature occurring in late afternoon or evening and the lowest occurring around 4 AM. Presumably, this holds true in critically ill infants and children. Once thought to be the result of exogenous factors, such as muscular exercise or feeding activity, it is now clear that these periodic fluctuations in temperature are the result of the operation of an endogenous system.[8] Despite the influence of certain conditions that alter the regulation of body temperature, among them fever, the human organism is capable of immense thermoregulatory adaptations and changes.

Circadian cycles influence childhood temperature. Temperature variation is one of the earliest rhythms to develop in infants and begins to appear after the first week of life. These rhythm changes develop progressively until the age of 5 years, when the adult pattern is present.[8]

The stability of body temperature varies inversely with the size of the body. This means that in infants with a rapid metabolic rate and somewhat large body surface area, the oral and rectal temperatures may vary through a range perhaps twice that of adults.[9] Because of the infant's small size, increased ratio of body surface area to mass, and elevated thermal conduction rate, the thermoregulatory ability of infants is restricted and easily overwhelmed by the environment.[2] Conversely, adults exhibit very stable body temperature mechanisms.

**Heat Loss.** The amount of heat loss that occurs varies according to atmospheric conditions, such as the speed of air currents passing over the body and the relative humidity of the air. The common modes of heat loss are via (1) radiation, conduction, and convection from the skin; (2) evaporation of sweat and insensible perspiration; (3) warming and humidifying of inspired air; and (4) urine and stool. Only the first two are under direct physiologic control. Of the sources under direct control, radiation accounts for approximately 50% of the total heat loss and convection, about 15%. Most of the remainder (about 30%) occurs through evaporation of water.[9]

Radiation is the loss of heat that radiates from the body to surroundings that are cooler than the body itself. It involves the transfer of heat between two objects, independent of the environmental temperature. The difference in temperature between the body and objects in the environment directly affects the rate at which a body cools via radiation.[9] For example, radiative heat losses can occur even when nude infants are in warm but transparent, single-walled incubators, particularly when near a cold wall or window. The total radiating surface of infants or children also influences heat loss. In fact, radiative heat loss is proportionately greater in smaller infants and children and represents the most serious source of heat loss for this group.[2]

Conductive heat loss involves the transfer of heat between two surfaces that are in direct contact with each other. The intensity of conductive heat loss depends on the temperature gradient between the body and the surface it contacts, the total body surface area, and the conductivity of the material contacting the body. In addition, physiologic factors influencing conductive heat loss are the velocity of cutaneous blood flow and the thickness of the body's subcutaneous insulating tissue.[2]

Convection is simply the movement of air. Heat loss by convection refers to heat conducted to the air and then carried away by convection currents. Insignificant amounts of convection always occur because heated air naturally rises away from the body. The degree of convective heat loss depends on several conditions, including the temperature of the air, volume of airflow, and the specific heat of the flowing air. Exposure of infants or children to drafts or increased airflow causes convective heat loss and is a stimulus for increased oxygen consumption.

Evaporative losses, primarily through the skin and lungs, account for a significant portion of heat loss. Infants are particularly vulnerable because their skin is thinner than that of older children, which increases evaporative losses. When water evaporates from the skin, 0.58 calories of heat are lost for every gram of water that evaporates. Under normal conditions, nearly 20% of the total body heat loss occurs from evaporation. Little can be done about this in terms of body temperature regulation because evaporative heat loss results from continuous diffusion of water molecules at any body temperature. However, excessive evaporative loss can be controlled by regulation of sweating, primarily by environmental manipulation.

Besides transepidermal evaporation, the respiratory system also serves as a route of evaporative heat loss. Evaporative losses via the respiratory tract are higher in infants as a result of their higher minute ventilation (the product of respiratory rate and tidal volume) in relation to body weight. Adequate environmental humidity minimizes evaporative losses from the lungs and skin surfaces. Physical factors affecting the rate of evaporation include relative humidity, velocity of airflow, and minute ventilation. Physiologic factors include infants' ability to sweat and their rate of minute ventilation.[2]

The neutral thermal zone of infants is the range of ambient temperatures at which the metabolic rate is minimal and temperature regulation is achieved by nonevaporative physical processes.[2] The neutral thermal environment (NTE) is further described as the ambient temperature and

humidity in which the control of body temperature is achieved by vasomotor adjustments, with minimal oxygen consumption and heat production. This narrow range of temperatures in infants varies with gestational age, postnatal age, weight, and clothing. With increasing age and weight, the NTE widens, and lower environmental temperatures are tolerated.[10]

**Sweating.** Sweating is an important means of controlling heat balance. Full-term infants, for example, begin to sweat with rectal temperatures of 37.5° to 37.9° C and an ambient temperature higher than 35° C. As body temperature rises, the anterior part of the hypothalamus is stimulated. The impulses from this area are transmitted through the anatomic pathways to the spinal cord and through the sympathetic outflow to the sweat glands in the skin throughout the body.[4] When the body temperature increases as little as 1° C, the sweat glands secrete large amounts of sweat to the skin surface.[11] This reaction produces evaporative cooling of the body. The rate of sweating varies according to environmental factors.

Excessive sweating can deplete extracellular fluid levels of electrolytes, particularly sodium and chloride. Cholinergic sympathetic nerve fibers ending on or near the glandular sweat cells elicit the secretions, which contain large amounts of sodium chloride. Similar to its effect on the renal tubules, aldosterone works in the sweat glands by augmenting the rate of active reabsorption of sodium by the ducts. This process also carries chloride with it because of the electrical gradient that develops across the epithelium with the reabsorption of sodium. Aldosterone can minimize the loss of sodium chloride in sweat when the plasma concentration is already low. Becoming acclimated to the heat can diminish this loss because of increased aldosterone production resulting from decreased salt reserves in the body.[4]

## NURSING INTERVENTIONS TO MAINTAIN NORMOTHERMIA

Nurses can help to maintain the body temperature of critically ill infants and children within normal limits primarily by managing external factors. The environment may be manipulated based on the principles of conduction, convection, radiation, evaporation, and the impact of each on body temperature. Factors such as ambient air temperature, humidity, airflow velocity, and the temperature of objects in direct contact with children's skin are all considered part of the environment. Each of these factors should be considered when making alterations to support an NTE.

Keeping infants and children (especially their heads), clothing, and bed linens dry can minimize evaporative losses. Humidifying and warming inspired gases can minimize evaporative and conductive losses from respiratory mucosa. In contrast, gases may be humidified and cooled when infants or children are hyperthermic. High humidity tends to reduce insensible water losses and evaporative losses; however, it also encourages the growth of gram-negative bacilli on the skin, including *Escherichia coli* and *Pseudomonas aeruginosa.* Hence, a relative level of humidity, approximately 50%, provides optimal environmental conditions.[12]

Conductive heat losses can be reduced by ensuring that cold surfaces are not in direct contact with children's skin and by using various types of thermal insulation, such as blankets and head coverings. Nurses can position children to avoid drafts and maintain the environmental temperature within the neutral thermal zone to avoid convective losses. Increasing the room temperature, using external heating devices, and applying thermal insulators, such as plastic or aluminized plastic sheeting, can minimize radiant heat loss.

## Assessment and Maintenance of Normal Body Temperature

The medical and surgical treatment of critically ill or injured infants and children often aggravates heat loss through skin and body cavity exposure, administration of cold intravenous fluids and blood products, and anesthetic administration. Hypothermia may contribute to inaccuracies in patient assessment and may complicate resuscitation of injured infants or children. Because the risk for alterations in body temperature are greater for critically ill infants and children, assessment of temperature and the body's thermoregulatory capabilities must be accurate and ongoing.

Frequent monitoring of critically ill infants' or children's body temperatures is necessary to establish baseline parameters and to guide nursing interventions. Temperature should be measured at recommended intervals for age and condition. Various techniques are used to measure core, regional, and skin temperatures in critically ill infants and children. Each is evaluated based on its relative advantages and disadvantages. The method selected is based on individual patient needs and the net balance of advantages and disadvantages of the system accurately monitoring body temperature. The site selected for estimating infants' or children's temperatures is recorded and used consistently in serial measurements. Because of the inaccuracy of many thermometers in clinical use, the same thermometer is used consistently for an individual patient. To decrease the possibility of inaccuracy, electronic and infrared devices are regularly scheduled for calibration.[13]

Internationally, the Celsius scale is the standard of temperature measurement. However, in the United States, the Fahrenheit scale is still widely used. Nurses may not be comfortable with converting temperatures from one scale to another and also may not be aware of the severity of temperature changes when measured on the Celsius scale.[13]

**Core Temperature Monitoring.** The core or central temperature is the temperature of the blood flowing through the branches of the carotid arteries to the hypothalamus. Pulmonary artery catheters and esophageal, tympanic, or nasopharyngeal probes monitor the temperature of blood, which approximates the temperature of the carotid artery and may be used for continuous assessment of core temperature.[14] Although useful, esophageal and nasopharyngeal temperature probes are not routinely used in PICU settings. Urinary bladder temperature (UBT), using a urinary catheter with an indwelling temperature-sensing

element, may also be measured. Tympanic thermometers have been found to be less accurate than esophageal readings but more accurate than rectal, axillary, or bladder readings for core temperature over a wide range of temperatures in pediatric surgery patients.[15]

The accepted standard for measurement of core body temperature is a thermistor in the pulmonary artery.[16] Pulmonary artery temperatures are approximately 0.2° lower than blood returning from the brain.[15] Because of the invasive nature, pulmonary artery temperature is only assessed in critically ill infants and children who also require advanced hemodynamic monitoring.

**Peripheral Temperature Monitoring.** Noncentral temperature measurement does not reflect core temperature but, rather, regional temperature. Regional temperature is affected by a variety of factors that may affect regional blood flow, such as intravascular volume, vascular tone, or environmental conditions. These methods are convenient and useful in monitoring changes and trends in temperature but lack the accuracy of most core temperature techniques. In contrast to core temperature reading techniques, regional temperature monitoring can detect physiologic decompensation in response to persistent hypothermia or hyperthermia. When hypothermia persists, for example, children's increased metabolism fails to compensate for the body cooling and results in regional blood flow shifts, causing metabolic acidosis and, eventually, apnea.[14]

Generally, regional temperatures are measured using electronic thermometers. Temperature measuring sites include the oral, rectal, and axillary locations. In the critical care setting, the oral route is infrequently used, whereas rectal or axillary routes predominate. Measuring rectal temperature is generally unnecessary. The risk of rectal perforation, cross-contamination, and the repeated invasiveness of the procedure are probably not warranted, particularly in young infants.[7,10] Rectal temperature probes can be influenced by the presence of stool in the rectum, which can act as an insulator and produce markedly delayed responses to core temperature changes.[2] In addition, relying solely on rectal temperature measurements that may not reflect rapid changes in core temperature may lead to delayed recognition of temperature extremes. Rectal probes and temperatures are always avoided in infants and children with inflammatory bowel disease, absolute neutropenia, or evidence of coagulation disorders or thrombocytopenia. Axillary temperatures are less hazardous to patients but have been shown to underestimate core temperature and be affected by ambient temperature.[7]

Skin temperature may be measured either by electronic thermometers or by electronic skin temperature thermistor probes that provide continuous assessment. They are a useful adjunct to other standard temperature-measuring devices but can be affected by poor perfusion, equipment dysfunction, or improper application. Critically ill patients who require the use of overbed warmers or warming blankets are continuously monitored for skin temperature with appropriate alarms for underheating or overheating. When skin temperature is continuously monitored, temperature is also measured periodically with an electronic thermometer.

For critically ill infants or children, a combination of temperature-measuring techniques is often indicated. This approach is necessary to detect variations in temperature that occur in response to physiologic dysfunction associated with critical illness or injury. Because significant differences may occur between peripheral and core temperatures, critical care nurses require skill in interpreting the implications of these differences considering the patient's physiologic status.

## Assessment of Temperature Imbalance

Wide variations in temperature produce alterations in cardiac output, oxygen consumption, and insensible water losses. With hyperthermia, heart rate increases, whereas with hypothermia, heart rate commonly decreases. Blood pressure can also be affected. For instance, blood pressure and cardiac output drop precipitously as hypothermia becomes more severe. Cardiac dysrhythmias, such as conduction delays and abnormal atrial and ventricular rhythms, may be evident. Respiratory rates may also vary. For example, hypothermic infants may experience periods of apnea or shallow breathing; in contrast, infants or children with fever or hyperthermia may become tachypneic.

Neurologic function may be impaired when infants or children experience temperature imbalance. For example, severe hypothermia (a core temperature lower than 35° C) may complicate neurologic assessment as pupils dilate, level of consciousness declines, reflexes and respirations diminish, and varying degrees of amnesia occur. Cerebral blood flow has been estimated to decrease 6% to 7% for every 1° C decrease in body temperature.[17] This is of particular concern for infants or children who have experienced multiple trauma or multiorgan dysfunction syndrome. All patients with hypothermia are monitored for changes in level of consciousness, signs of irritability or lethargy, diminished ability to arouse, and changes in muscle tone. Hence, measures to promote thermal neutrality are instituted as early as possible to ensure accurate neurologic assessment. Seizures may occur after periods of hypothermia as a result of ischemic brain injury and cerebral edema.

Oxygen consumption and tissue perfusion are important assessment parameters. Oxygen saturation using arterial or mixed venous blood can provide an important indicator of oxygen consumption. Often oxygen saturation of mixed venous blood indicates changes in physiologic status before changes in heart rate, pulmonary capillary wedge pressure (PCWP), or blood pressure are evident.[18] Supplemental oxygen may be necessary to combat hypoxemia, particularly in hypothermic infants and children.

Tissue perfusion assessed from skin color, temperature, and capillary refill must be routinely monitored. Pale, cool skin is an early manifestation of the vasoconstrictive response to cold stress or a decrease in core temperature. With hyperthermia, the skin may appear flushed as vasodilation occurs. Flushing may also come about because oxygen is not liberated from hemoglobin as readily when either low temperatures or overheating occurs.[19] Evidence of sweating is monitored in hyperthermic children.

Increased muscle activity is associated with heat production, whereas diminished muscle activity is often indicative of reduced heat production. Infants and children who are experiencing either elevated or reduced temperature may manifest changes in motor activity and therefore are monitored for such changes.

Routine assessment for the presence of shivering permits early detection and intervention. Shivering develops in a predictable fashion, beginning with masseter contractions and then proceeding to contractions of the trunk and long muscle groups. It culminates with generalized body shaking and teeth chattering.[20] Assessment for the presence of shivering includes palpation of the mandible for vibration and close inspection of facial, neck, and chest muscles for fasciculation. Core and peripheral temperatures are routinely assessed and compared. Assessment for shivering is continued until central and peripheral temperatures are normal.[18]

Fluid balance and renal function are carefully monitored when body temperature becomes deranged. Because the rate of fluid loss increases as a result of hyperthermia, adequate hydration is maintained. Adequate hydration prevents the complications of dehydration and promotes heat dissipation. When infants and children are hypothermic and peripherally vasoconstricted, fluids are carefully regulated during rewarming. When patients have vasoconstriction, fluid requirements are diminished; as warming occurs, the intravascular space expands, thereby increasing fluid requirements to maintain cardiac output. Infants' and children's ability to concentrate urine is impaired as hypothermia worsens. Acute tubular necrosis may occur as a result of diminished cardiac output and renal perfusion and myoglobinuria. Measurement of fluid balance enables nurses to prevent fluid overload or deficit. Fluid deficit following a period of hypothermia is often the result of increased insensible water loss during rewarming.

Laboratory data are routinely monitored to detect metabolic, biochemical, and hematologic derangements often associated with thermal instability. For example, excessive sweating can deplete extracellular fluid levels of electrolytes, particularly sodium and chloride. Serum electrolytes, blood urea nitrogen, creatinine, measures of acid-base balance, serum and urine osmolarity, hemoglobin, hematocrit, platelets, and other specific biochemical determinations are assessed regularly.

Hypothermia may cause metabolic acidosis. Acidosis coupled with hypothermia results in a left shift in the oxyhemoglobin dissociation curve, thereby impairing oxygen release at the tissue level. Arterial blood gases are monitored closely, and measures are instituted to prevent episodes of hypoxemia and acidosis. Hyperthermia can cause biochemical changes, depending on the underlying cause.

Hypoglycemia is another common finding in infants who experience alterations in temperature. This condition results from depletion of glycogen stores in the attempt to maintain core temperature in the normal range. In contrast, a transient hyperglycemic response is a common finding in older infants and children with alterations in temperature. This condition occurs as part of the body's response to stress, which liberates glucose to fuel the response.

Laboratory studies used in determining the source of fever or hyperthermia may include indirect and direct studies. Indirect studies, such as the white blood cell count and the erythrocyte sedimentation rate, reflect the body's response to infection. Indirect tests may serve as screening devices for identifying subgroups of infants and children at high risk of occult bacteremia (Table 14-2). Direct studies include blood and urine culture and sensitivity and rapid tests for detection of bacterial antigen. Direct tests allow detection of the specific causative organism.[21]

Other diagnostic tests may include cerebrospinal fluid examination or urinalysis. In addition, examinations such as radiographs; ultrasounds and computed tomographic (CT) scans; magnetic resonance imaging (MRI); or other nuclear medicine studies of the lungs, abdomen, and other organs may be indicated to determine the underlying cause of the temperature derangement.

## Thermoregulation Devices

Maintaining a neutral thermal environment and a normothermic body temperature are common nursing goals when caring for critically ill infants and children. Various thermoregulation devices are used regularly in the critical care setting. This is particularly crucial when transferring infants or children to other units within the hospital or to another facility.

**Warming Devices.** In addition to manipulating environmental conditions to alter the ambient temperature,

**Table 14-2 Risk of Occult Bacteremia**

| | Low Risk | High Risk |
|---|---|---|
| Age | >3 yr | <2 yr |
| Temperature | <39.4° C | >40° C |
| WBC count (per microliter) | >5000 and <15,000 | <5000 or >15,000 |
| Observational variables | Normal | Abnormal |
| | | History of contact with *Haemophilus influenzae* or *Neisseria meningitidis* |
| | | History of bacteremia |
| | | Immunologic impairment |

From Kline MW, Lorin MI: Fever without source. In Oski FA, DeAngelis CD, Feigin RD et al, eds: *Oski's principles and practice of pediatrics,* Philadelphia, 1999, Lippincott Williams & Wilkins, p 844.
*WBC,* White blood cell.

specialized equipment is often necessary to maintain an NTE. Various types of devices are used for this purpose. For infants, some type of closed warming device is commonly used, such as single- or double-walled incubators. These convection-warmed devices are used for thermal regulation of the infant's ambient air. Standard closed incubators control infant temperature by recirculation of warmed and humidified air. Infant size and postnatal age determine the temperature of the air in the incubator.[10] Plastic blankets or heat shields inside the incubator also reduce convective and evaporative losses. Disadvantages of this type of device include heat losses when the incubator is entered, potential variations in both incubator and infant temperatures during heating cycles, and diminished accessibility of infants for assessment and treatment.[22]

Open radiant warmers are useful to regulate temperature, particularly when infants and children require frequent monitoring and interventions. A radiant warmer consists of an electrically heated element that emits radiation within the infrared region of the electromagnetic spectrum. Radiation within this range allows optimal absorption of the energy by the skin. Heating of the skin causes vasodilation and increased blood flow to the skin. Moreover, it provides an avenue for heat transfer from the skin surface to the blood and eventually to deeper structures.

The advantages of radiant warmers include a superior servocontrol mechanism, greater consistency in surface temperature, improved patient access, and easier cleaning. The disadvantages associated with infrared radiation used in radiant warmers include risk of cataracts, flash burns of the skin, and heat stress.[22] In addition, radiant warmers promote insensible water loss, increase oxygen consumption, and slightly increase metabolic rate in infants, depending on weight and gestational age. Fluid requirements may be increased by 10% to 20%, particularly when radiant warmers are used in conjunction with phototherapy. Hence fluid requirements are adjusted depending on clinical and biochemical data. Radiant warmers are generally servocontrolled with the temperature probe attached to the abdomen.[19]

Servocontrolled devices automatically adjust heat output to maintain the temperature at a predetermined level in response to changes in patients' skin temperature. Some use an anterior abdominal wall temperature servocontrol mechanism to regulate skin temperature within a thermal-neutral range (36° to 36.5° C) by automatic air temperature control.[19] Core temperature is measured frequently when servocontrol is used to avoid overheating if the skin sensor loosens. In addition, accurate assessment of the infant's temperature may be compromised when a servocontrol device is used because the temperature is regulated to maintain the temperature at the predetermined level.

For older children, external heat sources, such as radiant warmers or heating blankets, are often used. Circulating water mattresses may be used to raise a patient's temperature and reduce conductive heat losses. Water-filled heating blankets are used judiciously in infants and children because when children are cold and peripherally vasoconstricted, the ability of surface capillaries to dissipate heat is diminished, increasing the risk for burns. Hence continuous monitoring of temperature and assessment of responses to interventions for rewarming are crucial to avoid tissue injury. The fluid temperature in the heating blanket should never exceed 39° C, and several layers of material are placed between the patient and the heating blanket to avoid burns.[23]

Forced air heating blankets have been shown to be the most clinically effective warming device intraoperatively.[24] This modality transfers the greatest amount of heat to the patient, when compared with other warming modalities.

When rapid rewarming is necessary, cardiopulmonary bypass may be used. This technique allows direct perfusion of the central circulation with warmed blood, reducing cardiac irritability and the risk of ventricular fibrillation and cardiac arrest. Alternatively, body cavities such as the chest, peritoneum, or gastrointestinal tract may be irrigated with warmed fluids.

**Cooling Devices.** When infants or children become hyperthermic, surface cooling techniques, such as removing heat-conserving clothing or blankets or packing in ice, may be used. Most commonly, external cooling blankets are applied, and in the majority of cases, the decision to use this modality is made by the critical care nurse.[25] A comparison of the effectiveness of posterior versus anterior positioning of the blanket and cooling effectiveness after placement is needed. If the blanket is placed posteriorly, care is taken to avoid decubitus ulcers. A recent development in cooling is the use of cold air blankets. These blankets are thought to be more comfortable for patients and may be more effective than circulating water blankets; however, this assumption has not been substantiated by research studies.[25] Whatever method is used for surface cooling, the patient's vital signs, perfusion, and skin integrity are assessed frequently. If additional temperature reduction is needed, body temperature can be reduced by core cooling. Lavage of gastric or peritoneal cavities and the administration of iced intravenous fluids can achieve this goal.

Extreme variation in temperature, hypothermia or hyperthermia, can result in death or serious injury, and thus alarm systems and range controls of all equipment used to regulate temperature require regular testing.

## ABNORMALITIES OF BODY TEMPERATURE REGULATION

### Hyperthermia

Hyperthermia is a state in which a person has a sustained elevation in body temperature (more than 37.8° C orally or 38.8° C rectally) because of internal or external factors.[1] Internal factors, such as fever, malignant hyperthermia, or heat-related illnesses, and external factors, such as extreme environmental conditions or accidental overheating, contribute to the development of hyperthermia. The most common cause of hyperthermia is fever. Although relatively uncommon, malignant hyperthermia may also necessitate a patient's admission to a critical care unit.

Fever is distinguished from other types of elevations in body temperature. First, the "set-point" is that temperature around which body temperature is regulated by the thermostat-like mechanism in the hypothalamus. "Hyperthermia" is that situation in which body temperature ex-

ceeds the set-point. This usually results from conditions producing more heat than the body can dissipate (e.g., in heatstroke, aspirin toxicity, or hyperthyroidism). "Fever" is an elevation in the set-point such that body temperature is regulated at a higher level.[26] In any discussion of fever, it is important to remember that fever is a *symptom,* not a disease, and should be viewed as reflecting an underlying disorder.

Fever may result from abnormalities in the brain itself, the presence of toxins that affect the brain's temperature control areas, infection, dehydration, or other causes.[4] Generally, fever results from a pyrogen-mediated elevation in the hypothalamic set-point. The major problems resulting in fever include an increase in the hypothalamic thermoregulatory set-point, excess heat production, and defective heat loss.[27]

Instrumental in resetting the hypothalamic thermostat are pyrogens, substances that cause the set-point to be increased. These pyrogens may be proteins, breakdown products of proteins, or certain other substances (e.g., lipopolysaccharide toxins secreted by bacteria). Pyrogens may be present during disease states. When the set-point is elevated, all the body's efforts turn to decreasing heat loss and increasing heat production. Heat production is increased via increased muscle tone, activity, and metabolic rate, whereas heat loss is decreased through peripheral vasoconstriction.[28] These changes help the body to reach its new temperature within hours.[4]

The pathophysiologic mechanism of fever includes the production of hormonelike mediators by macrophages and cells of the reticuloendothelial system. This results in (1) an increase in CNS production of prostaglandin $E_2$, which increases the hypothalamic set-point and temperature; (2) an increase in neutrophil release from the bone marrow; (3) a decrease in serum iron and zinc; (4) a change in hepatic protein production; and (5) an increased T-lymphocyte proliferation. Interleukin-1 (IL-1) is a substance common to these pathways.[29] Undesirable effects of fever are listed in Box 14-2.

For healthy infants and children, these demands pose no particular threat. For those with underlying disease, especially that involving the heart or lungs, the increased demands are potentially harmful or even fatal. In susceptible infants and children 6 months to 5 years old, fever can precipitate seizures. Generally, these seizures are benign, but they are very upsetting to both parents and children and may result in invasive, expensive, and probably unnecessary procedures.[28]

Febrile conditions share several characteristics. Chills occur when the hypothalamic set-point abruptly rises to a higher-than-normal level because of tissue destruction, presence of pyrogenic substances, or dehydration. As the body attempts to attain its new temperature setting, the blood temperature is lower than the set-point temperature for several hours. Autonomic responses to increased body temperature occur, such as chills, vasoconstriction, and shivering. When the blood temperature reaches the set-point temperature, the person feels neither hot nor cold. As long as the factor producing the fever continues, the body temperature is regulated normally but at a higher level. If the factor producing the fever is suddenly removed, the set-point abruptly decreases to its normal lower level. The body then feels "overheated" and reacts with intense sweating and hot skin, resulting from a general vasodilation caused to dissipate heat more quickly. This reaction is known as the "crisis" or "flush."[4]

Fever has several other causes. Traumatic brain injury or congenital CNS malformations can produce recurrent, transient elevations in body temperature. Other noninfectious causes of fever are (1) iatrogenic (e.g., heavy blankets, overdressing, mechanical); (2) thrombophlebitis, resulting from intravenous catheterization; (3) infusions of irritating fluids; and (4) endocrine disorders. Certain hypothalamic lesions produced by cerebrovascular hemorrhage, neurosurgical procedures, or tumors may produce decreased thermoregulatory ability. Drugs that produce fever include lysergic acid diethylamide (LSD), cocaine, amphetamines, phencyclidine (PCP), salicylates, anticholinergics, prostaglandin $E_1$, and tricyclic antidepressants.[30,31]

**Treatment of Fever.** The decision to treat fever can be difficult. An important principle is that not all fevers *need* to be treated; body temperature does not always need to be completely normal. The importance of fever as an indicator of disease, not as inherently harmful, should be expressed to the patient's significant others, who may be experiencing "fever paranoia."[32] Recommendations for treatment include (1) high fever (40° C or above), (2) fever in infants and children at risk for febrile seizures, (3) fever in infants and children with underlying neurologic or cardiopulmonary disease, or (4) fever in any situation in which heat illness (e.g., heatstroke) is suspected. Treating fever for patient comfort should not be condemned.[28] Once the decision to treat a fever has been made, the choice of a specific modality is based on a number of considerations. Because fever is the result of an elevated hypothalamic set-point, the most logical means of treating the fever is by restoring the set-point to a normal level. Aspirin, acetaminophen, and ibuprofen all work in this way. Aspirin and acetaminophen are equally effective at similar doses. Ibuprofen is effective at a slightly lower dose and has a longer duration of action. Given the minimal difference in the effectiveness of aspirin and acetaminophen, selection should be based on potential toxicities and cost rather than efficacy.[28]

In therapeutic doses, aspirin is the most toxic of the choices. Potentially serious side effects are gastritis, gastro-

**Box 14-2**
**Undesirable Effects of Fever**

Patient discomfort
Increased metabolic rate
Elevated oxygen consumption
Increased carbon dioxide production
Increased cardiovascular and pulmonary system demands

From Lorin MI: Pathogenesis of fever and its treatment. In Oski FA, DeAngelis CD, Feigin RD et al, eds: *Oski's principles and practice of pediatrics,* Philadelphia, 1999, Lippincott Williams & Wilkins, pp 848-850.

**Table 14-3 Use of External Cooling Methods for Treating Elevated Temperature**

| Cooling Method | Indications |
|---|---|
| Tepid sponging **instead of** antipyretic drugs | Very young infants<br>Severe liver disease<br>History of hypersensitivity to antipyretic drugs |
| Tepid sponging **plus** antipyretic drugs | High fever (>40° C)<br>History of febrile seizures, neurologic disorders, or brain damage<br>Infection plus suspicion of overheating<br>Septic shock* |
| Cold sponging **alone** | Heat illness |

From Lorin MI: Pathogenesis of fever and its treatment. In Oski FA, DeAngelis CD, Feigin RD et al, eds: *Oski's principles and practice of pediatrics*, Philadelphia, 1999, Lippincott Williams & Wilkins, p 850.
*May require cold sponging.

intestinal bleeding, diminished platelet functioning, decreased urinary sodium excretion, and lowered immune response. These effects are seen often with aspirin, less often with ibuprofen, and not at all with acetaminophen. In fact, acetaminophen has no side effects at therapeutic levels. However, one study found that when administered rectally, the absorption of acetaminophen was found to be erratic.[25] Aspirin, and possibly ibuprofen and naproxen, because of their pharmacologic similarity, has been implicated in the development of Reye's syndrome.[28]

Another method of fever reduction is external cooling, generally by sponging with tepid water. This may be used with or without the administration of antipyretic medications. Henker[25] provided evidence that the combination of acetaminophen with sponging was significantly more effective in decreasing temperature than acetaminophen alone. External cooling is the treatment of choice for heat-related illnesses. Its use in fever is generally recommended only if a heat-related illness may be the partial or total cause of the elevated body temperature[28] (Table 14-3).

Sponging as a method of fever reduction usually adds nothing other than discomfort when used with previously well infants or children with non–life-threatening fever. When ice water is used, cooling is more rapid and more uncomfortable; therefore it is used only in the case of heat illness. Sponging is useful in infants or children with neurologic disorders because many have abnormal temperature control mechanisms and respond poorly to antipyretics. Sponging is preferable in infants and children with demonstrated hypersensitivity to antipyretics or in those who have liver disease. Sponging is normally done with tepid water (approximately 30° C). Alcohol is not used because the fumes may be absorbed through the lungs and possibly skin, and it may produce alcohol intoxication.[28] External cooling devices may also be effective in reducing body temperature.

Treatment of fever associated with suspected bacteremia generally includes antibiotics. Ideally, with the emergence of antibiotic-resistant organisms, blood cultures are obtained before instituting antibiotic therapy.[33] Specific antibiotic recommendations are directed at the most common bacterial pathogens. In any case, the patient is followed carefully to monitor the effectiveness of the treatment regimen. When infants and children are critically ill and febrile, parenteral antibiotic therapy may be initiated in tandem with the diagnostic workup. Infants younger than 30 days, children with underlying disorders that predispose them to serious bacterial infections, and children who appear toxic are generally treated with antibiotics before culture results are returned, to decrease the possibility of an overwhelming sepsis.[21] Moreover, if the source of the fever is determined to be infectious, proper infection control and therapeutic measures are initiated.

## Drug Fever

Fever may be a complication of drug therapy. This response to medications can increase the duration of hospital stay and the number of diagnostic tests performed.[34] Particular drugs associated with drug fever include phenytoin, histamine blockers, procainamide, and antibiotics, most notably sulfonamides.[35] Drug fever is considered if a clinically improved patient develops an unexplained fever after receiving drugs known to produce febrile reactions 7 to 10 days after their institution. Drugs that raise the basal metabolic rate, produce increased skeletal muscle activity, or lower cutaneous blood flow may produce an increase in body temperature that will normalize after the drug is stopped.[30] Drug-associated fever can be extremely elevated in some patients and can take up to 5 days to resolve after discontinuation of the drug.[35]

## Malignant Hyperthermia

Malignant hyperthermia (MH) is a hypermetabolic crisis triggered by the administration of a certain quantity of potent volatile anesthetic agents or a depolarizing muscle relaxant, usually succinylcholine (SCH). The mean age of a patient experiencing a MH episode is 22 years of age.[36] MH is a familial disease, but the mode of genetic transmission remains unclear. The syndrome was originally thought to be transmitted as an autosomal dominant trait. Further investigation suggests that the inheritance in most families is multigenetic with variable expression.[37] Reports of MH vary between countries. In the United States, the incidence is highest in the Midwest.[36]

The incidence of MH ranges from 1 in 14,000 pediatric patients to 1 in 40,000 adult patients. The difference in occurrence may occur because adults are often induced with sodium thiopental and a nondepolarizing muscle relaxant, both of which inhibit triggering of MH. Children, on the other hand, are often induced with halothane

followed by SCH, a combination that constitutes an effective trigger for MH.[37]

The site of the primary lesion implicated in the pathogenesis of MH is skeletal muscle and is related to disturbed calcium metabolism. Traditional theory is that a defect in the muscle cell membrane causes loss of control of intracellular ionized calcium levels, leading to an increase in calcium in skeletal muscle and abnormal muscle activity. As hyperthermia continues, the myoplasmic calcium concentration remains elevated, producing continued muscle contraction and heat production. A new developing theory is that MH is a disorder of the ion channels that control skeletal muscle, that is, a channelopathy.[38,39]

Initially, an anesthetic-induced increase in aerobic and anaerobic metabolism occurs, manifested by massive production of heat, carbon dioxide, and lactic acid. This results in respiratory and metabolic acidosis, along with a rapid increase in temperature. Tachycardia is accompanied by other signs of circulatory and metabolic stress. Abnormal muscle activity develops, which may progress to whole-body rigidity. An increase in muscle permeability produces increased serum levels of potassium, phosphorus, calcium, sodium, and creatine phosphokinase (CPK). Muscle edema develops, and an excessive release of myoglobin from muscle results in gross myoglobinemia. Disseminated intravascular coagulopathy and cardiac or renal failure may develop. Death may result from a combination of gross electrolyte disturbances, especially hyperkalemia, leading to cardiac failure.[37,38]

The clinical course of MH is extremely variable. It can be fulminant, rapidly progressing to metabolic acidosis and death if not diagnosed and treated promptly. Rarely, the onset can be delayed for some hours. The sequence and severity of clinical events depend on (1) the types and concentrations of anesthetics involved, (2) the nature and extent of underlying myopathy, and (3) the promptness of diagnosis and initiation of appropriate treatment.[43]

The first systemic effect of MH is increased metabolism (increased oxygen consumption and carbon dioxide production). The cardiovascular and respiratory systems respond to this increased demand by increasing their output. Therefore the first clinically evident signs and symptoms of MH are tachycardia and tachypnea. However, an increased end-tidal carbon dioxide value precedes these signs and symptoms[40] (Table 14-4).

Tachycardia occurs in 96% of all patients with MH within 30 minutes of anesthesia induction.[41] Rapid ventricular arrhythmias (e.g., bigeminy and ventricular tachycardia) may occur. Tachycardia and/or dysrhythmias usually occur before fever, and thus MH is suspected when these signs occur, unless there are other obvious causes for them. Cardiac arrhythmias result from the stress of MH on the myocardium, probably caused by the hypermetabolic state. The electrocardiographic tracing shows tall, peaked T waves, and/or ST-segment depression.[42]

Muscle rigidity may or may not occur. If seen, it usually occurs first in the muscles of the jaw, extremities, or chest usually after the administration of SCH. Instead of relaxing, the jaw tightens, making intubation and ventilation difficult or impossible.[42] Facial muscle fasciculation may be present. Rigidity then travels through other skeletal muscles.

**Table 14-4 Clinical Presentation of Malignant Hyperthermia**

| Clinical Findings | Laboratory Findings |
|---|---|
| Tachycardia | Marked elevation of end-tidal carbon dioxide |
| Tachypnea—spontaneous ventilation | Hypercarbia—central venous and arterial |
| Unstable blood pressure | Acidosis—respiratory and metabolic |
| Fever—rapid rise (1° C every 15 min) | Central venous and arterial desaturation |
| Sustained rise (to 43° C) | Hyperkalemia |
| Rigidity—especially trismus | Elevated creatine phosphokinase (CPK), myoglobinemia |
| Cyanosis—dark blood in surgical field, mottling of skin | |
| Profuse sweating | |

From Ryan JF: Malignant hyperthermia. In Ryan JF, Todres ID, Cote CJ, et al, eds: *A practice of anesthesia for infants and children*, ed 2, Philadelphia, 1993, WB Saunders, pp 417-428; Gronert GA, Antognini JF, Pessah IN: Malignant hyperthermia. In Miller RD, ed: *Anesthesia*, 2 vols, ed 5, New York, 2000, Churchill Livingstone, pp 1033-1052.

Fever is the clinical hallmark of MH and results from many biochemical derangements. It is a somewhat late sign and may not occur at all if dantrolene is promptly administered.[43] Without treatment, the body temperature can rise 1° C every 5 minutes and exceed 43° C.[38]

Patients undergoing anesthesia are observed for evidence of fever. Signs of fever during surgery include hot, flushed skin; a hot anesthetic rebreathing bag; and hot tissue around the operative site. A change in skin color may accompany the development of MH. Flushed, rosy skin (similar to the familiar "atropine flush") may occur from the increased production of body heat. To dissipate the heat, vasodilation occurs. The flushed skin subsequently becomes mottled and then cyanotic. Simultaneously, the surgeon notes dark blood at the operative site.[43]

Treatment of MH includes early recognition, discontinuation of the anesthetic agent, cooling, hyperventilation with 100% oxygen, restoration of acid-base balance, administration of medications to treat dysrhythmias, and relax skeletal muscle contractions.[38,44] All interventions are carried out simultaneously. In addition, dantrolene sodium is administered without delay because MH is potentially fatal if not treated immediately with this medication. The mortality rate from MH reached 70% before the use of dantrolene. Earlier diagnosis of MH and advent of dantrolene have decreased the mortality rate to less than 5%.[38]

Dantrolene is a lipid-soluble hydantoin derivative with direct effects on skeletal muscle. It is given intravenously, initially 2 mg/kg, increasing to a total of 10 mg/kg. Dosing in this manner provides therapeutic blood levels with a half-life of at least 10 hours in children and adults.[38]

## Nursing Care of Patients With Elevated Body Temperature

Hyperthermia may be treatable by nursing intervention alone (e.g., by correcting external causes such as inappropriate clothing for environmental conditions, exposure to the elements, or dehydration). In other cases, such as MH, nursing intervention alone is insufficient, and medical and other interventions may be necessary.

The impact of core hyperthermia on an already compromised patient can be deleterious. Oxygen consumption rises 10% to 12% for every 1° C temperature elevation.[45] The increased metabolic demand in response to hyperthermia may produce progressive metabolic acidosis, as oxygen delivery to the tissues is compromised.[46] Arterial blood gases and biochemical balance are monitored closely to detect acid-base imbalances and hypoxemia. Proper treatment of acidosis is promptly instituted to prevent untoward effects.

Sustained tachycardia in hyperthermic infants and children may compromise myocardial perfusion and diastolic filling and may lead to greater stress on an already compromised heart. Moreover, infants or children experiencing hyperthermia are observed for sweating and peripheral vasodilation, both of which greatly increase loss of heat from the skin.

Regardless of the measures instituted to reduce temperature, shivering is not stimulated. Shivering is a normal compensatory response to heat loss, but in the hemodynamically compromised patient, the effects can be deleterious. Shivering increases metabolic rate, carbon dioxide production, and myocardial oxygen consumption, all of which eventually increase the myocardial workload. Arterial oxygen saturation decreases and systemic vascular resistance and heart rate increase with shivering. In addition, oxygen consumption increases 500%,[47] and the production and accumulation of lactic acid accelerate, which may culminate in lactic acidosis.

Nurses focus on determining the proper combination of interventions to reduce temperature and avoid shivering. If shivering develops, measures are instituted to avoid the metabolic and hemodynamic consequences. Appropriate nursing interventions for shivering modify the rate of heat loss from the skin and interfere with the body's determination of heat loss.[48] Various techniques have been suggested. Intravenous narcotics have been used to suppress shivering but may also produce side effects such as nausea or hypotension.[49] An alternative is wrapping the extremities with towels during surface cooling with a hypothermia blanket.[48]

Psychologic support is particularly important when dealing with critically ill infants and children who are further distressed by both the discomfort of fever and its treatment. Interventions are based on the developmental stage and cognitive ability of the patient.

## Hypothermia

Hypothermia is defined as any core body temperature less than 35° C. Degrees of hypothermia are detailed in Table 14-5. At low body temperatures (below 34° C), the hypothalamus functions minimally, and below 29° C, it cannot regulate temperature at all. Loss of temperature-regulating capability produces a rapid decrease in body temperature and eventually results in death.[4] When temperature drops low enough to trigger thermoregulatory control mechanisms, shivering thermogenesis and a generalized catecholamine release occur. Responses of the sympathetic nervous system prompt many other physiologic responses to produce the diagnostic characteristics of hypothermia (Box 14-3).

Infants and children are among high-risk groups for hypothermia, especially if unconscious, immobile, sedated, or malnourished. Mild hypothermia often can be observed in infants and children admitted to critical care units because of cold ambient temperatures. Box 14-4 outlines predisposing factors. Moderate to severe hypothermia is often present in patients who have suffered trauma, exposure, drowning, ingestion of poisons, or shock. Infants or children with unexplained altered responsiveness are evaluated for hypothermia by measuring core temperature.[50]

Many pharmacologic agents may contribute to hypothermia. Phenothiazines and barbiturates exert a direct effect on the anterior hypothalamus, decreasing its responsive-

**Table 14-5 Levels of Clinical Hypothermia**

| Level | Temperature (° C) |
|---|---|
| Normothermia | 37 |
| Mild hypothermia | 32-35 |
| Moderate hypothermia | 28-32 |
| Severe hypothermia | <28 |

Heimbach D, Jurkovich GJ, Gentilello LM: Accidental hypothermia. In Grenvik A, Ayres SM, Holbrook PR et al, eds: *Textbook of critical care*, ed 4, Philadelphia, 1999, WB Saunders, pp 377-383.

**Box 14-3**
**Diagnostic Characteristics of Hypothermia**

**Major (80%-100% of Cases)**

Rectal temperature of <35.5° C
Cool skin
Pallor (moderate)
Shivering (mild)

**Minor (50%-79% of Cases)**

Mental confusion, drowsiness, restlessness
Decreased pulse and respirations
Cachexia, malnutrition

Data from Carpenito LJ: *Nursing diagnosis: application to clinical practice*, ed 8, Philadelphia, 2000, JB Lippincott, p 147.

ness to cold. Neuromuscular blocking agents and phenothiazines decrease the body's ability to engage in shivering thermogenesis. Vasodilators inhibit the peripheral vascular vasoconstrictor response and increase heat loss, thus decreasing temperature stability. Long-term use of vasopressors depletes catecholamine reserves and alters receptor function, thus impairing the peripheral vascular response to cold stress.[50]

One clinical phenomenon that may produce severe hypothermia is cold water drowning (i.e., drowning in freezing water). Even in warm climates, however, the temperature of pool water can be significantly lower than air temperature. Moderate water temperatures are lower than body temperature. The relatively large body surface area of infants and children predisposes them to rapid heat loss in water. As a consequence, small infants or children can become hypothermic even in relatively warm pool water in a moderate climate.

Therapeutic, induced, or controlled hypothermia is used to reduce metabolic demands during cardiac surgery. Induced hypothermia for cardiac surgery involves both systemic and cardiac cooling. Systemic hypothermia is achieved by cooling the blood as it circulates through the heat exchanger of the cardiopulmonary bypass pump. Cardiac hypothermia is achieved directly by a cooled perfusate. Initially, a regional temperature gradient occurs because the core (heart and brain) is cooled first and the peripheral tissues remain warm. Gradually, the skin temperature drops and eventually approximates the core temperature as heat is dissipated. During this time, the body's inherent thermoregulatory mechanisms cease, resulting in profound hypothermia.[51] Rewarming is initiated by warming the blood circulated through the body and discontinuing extracorporeal circulation. During this phase, the core is warmed first, and the regional and peripheral areas (rectum, bladder, and skin) remain cooler, creating another temperature gradient. As the patient's thermoregulatory function returns, the patient is vulnerable to shivering.

**Box 14-4**

**Factors Predisposing Infants and Children to Thermal Instability**

- Relatively large body surface area
- Relatively limited nutritional reserve
- Impaired cardiac, renal, hepatic, or endocrine function
- Impaired behavioral, neural, and endocrine responses (from underlying physical and physiologic states)
- Impaired neuroendocrine response (from pharmacologic agents)
- Cardiopulmonary resuscitation, anesthesia, or extended radiographic procedures

Data from Brink LW: Abnormalities in temperature regulation. In Levin DL, Morris FC, eds: *Essentials of pediatric intensive care,* New York, 1999, Churchill Livingstone, pp 548-559, and St Louis, Quality Medical Publishing; and Heimbach D, Jurkovich GJ, Gentilello LM: Accidental hypothermia. In Grenvik A, Ayres SM, Holbrook PR et al, eds: *Textbook of critical care,* ed 4, Philadelphia, 1999, WB Saunders, pp 377-383.

## Nursing Care of Patients With Decreased Body Temperature

Once the diagnosis of hypothermia is made, continuous core body temperature measurement is initiated while evaluating thoroughly for risk factors (Table 14-6) and potential complications. Important assessments following the diagnosis of hypothermia include (1) electrocardiographic monitoring (significant arrhythmias may occur because of myocardial irritability); (2) arterial blood pressure monitoring; and (3) frequent evaluation of acid-base status, serum electrolytes, and blood glucose levels. External and/or core rewarming is instituted promptly.

Interpretation of arterial blood gas results in the hypothermic patient may necessitate the use of correction curves or values on rewarmed specimens. This is recommended because low temperatures cause carbon dioxide solubility to change, forcing the oxygen dissociation curve to shift to the left. When a specimen is drawn from a hypothermic patient and warmed to 37° C, the solubility of carbon dioxide decreases, resulting in a higher $Paco_2$ and lower pH than exists in the patient. $Pao_2$ values are corrected for temperature because warming the blood increases the solubility of oxygen and results in $Pao_2$ values significantly higher than in the patient.[50] According to Shapiro and Cane,[52] however, if the patient's temperature is 35° to 39° C, little is to be gained in correcting blood gas values. If the patient's temperature falls outside this range, it may be clinically useful to correct blood gas values with an uncorrected $Pao_2$ less than 60 torr or an uncorrected $Paco_2$ less than 30 torr because these values may be higher than actual measurements.

Shivering thermogenesis begins at temperatures of 30° to 35° C. This condition results in a small increase in heat production, whereas oxygen consumption and metabolic rate increase significantly. Transient hyperglycemia may result from glycogenolysis in the liver and muscles. The catabolism of fat can produce ketosis. Lactate production ends in metabolic acidosis, and compensatory respiratory alkalosis follows. These changes peak at 34° to 35° C.[50]

As hypothermia deepens, shivering thermogenesis ceases. Nonshivering thermogenesis occurs as the core temperature falls below 30° C. Heat production and metabolic rate both fall below baseline requirements at this point.[50,53] Total oxygen consumption is proportionately decreased. There is a 6% fall in oxygen consumption for every degree Celsius that the core temperature decreases. However, the extent of reduction of metabolism varies in each organ system.[50] When the temperature is normal, oxygen consumption is highest in the kidney, which is the organ most rapidly affected by hypothermia.

*Cold diuresis* is a term used to describe the renal response to cold. This response denotes adequate urine output despite a significant impairment in renal blood flow and glomerular filtration rate. Diuresis may continue despite systemic hypotension, dehydration, and hyperosmolarity, presumably because of a defect in renal tubular reabsorption of water.[50]

Changes in the cardiovascular system occur with hypothermia. The initial catecholamine-induced tachycardia is transient. During the phase of shivering thermogenesis,

**Table 14-6 Risk Factors for Hypothermia**

| Cause | Mechanism |
|---|---|
| **Exposure** | Increased heat loss, especially conductive heat loss (wet clothes or immersion) or convective losses (wind) |
| Trauma | |
| Drowning | |
| **CNS depression** | Direct central effect on thalamic temperature center |
| Head injury | |
| Cerebral hemorrhage, tumor, or infection | |
| **Drug-induced** | |
| Narcotics | CNS depression and vasodilation |
| Barbiturates | CNS depression |
| Phenothiazines | α-Adrenergic block, impaired shivering thermogenesis, lowered set-point |
| Alcohol | CNS depression (and associated trauma, exposure, and impaired behavioral responses) |
| General anesthesia | CNS depression with vasodilation |
| **Endocrine Abnormalities** | |
| Hypoglycemia | Impaired thermogenesis, limited metabolic response to cold |
| Hypothyroidism | |
| Hypopituitarism | Impaired hypothalamic response to cold |
| **Spinal Cord Transection** | Interrupted sensory afferent<br>Inability to sense cold<br>Impaired central reflex and behavioral responses |
| **Skin Disorders** | Increased transdermal water and heat losses |
| Erythrodermas | |
| Burns | |
| Stevens-Johnson syndrome | |
| **Therapeutic** | CNS depression |
| Treatment of Reye's syndrome | |
| Cardiopulmonary bypass | |

Data from Brink LW: Abnormalities in temperature regulation. In Levin DL, Morris FC, eds: *Essentials of pediatric intensive care*, New York, 1999, Churchill Livingstone, p 553, and St Louis, Quality Medical Publishing.
*CNS*, Central nervous system.

**Table 14-7 Cardiac Dysrhythmias in Hypothermia**

| Core Temperature | Arrhythmia |
|---|---|
| <34° C | Atrial fibrillation (more severe bradydysrhythmias noted with cooling) |
| <30° C | First-degree atrioventricular block |
| <20° C | Third-degree atrioventricular block |

Data from Brink LW: Abnormalities in temperature regulation. In Levin DL, Morris FC, eds: *Essentials of pediatric intensive care*, New York, 1999, Churchill Livingstone, p 553, and St Louis, Quality Medical Publishing.

there is a decrease in cardiac conductivity and automaticity and an increase in the refractory period.[50] Table 14-7 outlines the characteristic cardiac effects of various levels of hypothermia. These arrhythmias may not be treatable until core rewarming occurs. However, electrocardiographic monitoring may be useful in identifying the severity of hypothermia. The J-point elevation, for example, is potentially useful in diagnosing the severity of hypothermia.[50]

Another significant effect of hypothermia is hemodynamic. Both myocardial contractility and vasomotor tone are impaired by hypothermia. This effect may produce profound hemodynamic collapse. During rewarming, significant hypotension may occur in response to peripheral vasodilation. Severe hypothermia may make cardiac resuscitation impossible, and thus rewarming should continue during resuscitation.[54] Resuscitation is continued until a core temperature of *at least* 30° C has been obtained, particularly if the primary cause of the cardiac arrest is hypothermia. Resuscitation efforts are applied thoughtfully in the severely hypothermic child. Core temperature below 28° C places the child at high risk for ventricular fibrillation, which may be induced by cardiac compression. If the child presents with a nonarrest cardiac rhythm, chest compression is not implemented, even in the face of severe bradycardia. However, chest compression is necessitated in patients with asystole or ventricular fibrillation.[49] Hypothermic patients are not

declared legally dead until a core temperature of 32° C or greater is achieved.[55]

The respiratory system shows less uniform effects. Initially, cold stimulates tachypnea. Shivering thermogenesis may produce compensatory alkalosis; however, below 30° C, hypoventilation is often seen. Central apnea occurs as hypothermia progresses.[50] In addition, oxygen consumption rises and may produce hypoxia.

The CNS response varies with the degree of hypothermia. Mild to moderate hypothermia can produce confusion and behavioral changes. As the core temperature continues to drop, stupor worsens, and coma results. Below 26° C, unresponsiveness; flaccidity; and fixed, dilated pupils follow. The CNS can benefit from the reduced metabolic and oxygen demands that result from hypothermia. Factors that determine the degree of benefit include (1) degree and duration of hypothermia, (2) underlying disease processes, (3) cardiorespiratory status, and (4) prior or concomitant medication usage. Because the effects of hypothermia on the CNS may be profound, rewarming to a temperature higher than 35° C is recommended before evaluation of brain death is undertaken.[50]

Pharmacologic effects of hypothermia are varied. Moderate to severe hypothermia produces such a serious decrease in metabolic rate that oxygen consumption and the rate of biochemical reactions slow considerably. As a result, drug levels and effects are difficult to evaluate in hypothermic patients. Decreased cardiac output, dehydration, slowed hepatic metabolism, impaired glomerular filtration, and abnormal renal tubular filtration and reabsorption can all result in reduced drug clearance.[50] Hypothermia, for example, elevates the toxic dose of digitalis, whereas it decreases the inotropic dose. Potassium- and calcium-induced cardiac arrhythmias are possible because of increased myocardial sensitivity during hypothermia. Finally, temperatures below 26° C depress the cardiotonic effects of catecholamines; mild to moderate hypothermia, however, enhances their effects.[50] Other pharmacologic effects include heightened sensitivity to anesthetic agents and barbiturates. Both barbiturates (because of their depressant effect) and phenothiazines (because of their α-adrenergic blocking effects) potentiate hypothermia.

With body temperature below 30° C, hyperviscosity and hypercoagulability of the blood may occur. This results from a rising hematocrit level resulting from the cold diuresis that accompanies hypothermia. Infection is also a danger as a result of neutropenia, and coagulopathies can be accentuated because of thrombocytopenia.[50]

Generally, external warming devices are used to return the patient's temperature to the normal range in the case of mild to moderate hypothermia (i.e., core temperature of 30°to 35° C). Radiant warmers, heating blankets or pads, warmed blankets, and head coverings are commonly used. Reflective blankets (lightweight metallic blankets that reflect up to 80% of radiant heat to the body)[56] and buntings insulated with Thinsulate[57] have been recommended. A combination of modalities may be superior to a single rewarming technique.[58]

When instituting such measures, nurses are vigilant in their assessment of the patient's responses to the treatments. Radiant warmers are used only with the servocontrol option to avoid thermal injury to the skin. Heating pads and blankets and other warming devices are used with caution. Critically ill infants or children are not likely to be able to perceive a thermal injury or communicate it to nurses.

Severe hypothermia (core temperature lower than 30° C), for example, as a result of cold-water submersion, often requires active internal warming methods in addition to external warming measures. In such circumstances, measures such as heated humidified air, warmed intravenous fluids, and gastric or colonic lavage with warmed solutions or peritoneal dialysis may be implemented.[53] Extracorporeal rewarming (ECR) may be required in the most severe circumstances.[49] ECR has been advocated as a rewarming technique in hypothermic patients to reduce the problems of rewarming shock, dysrhythmias, and thermal injury associated with external warming devices.[17,59]

ECR diverts a significant portion of the patient's cardiac output through the extracorporeal membrane oxygenator and blood warmer. Gradual rewarming is facilitated by maintaining a warming gradient of approximately 10° C between the perfusate in the extracorporeal circuit and the patient's core temperature until body temperature reaches a normal range.[60] Slow rewarming avoids the sudden recirculation of cold, acidotic blood from the vasoconstricted peripheral vascular beds to the central circulation. This phenomenon, called "rewarming shock" or "afterdrop," is manifested by a continued decline in core temperature and serum pH after removal from the cold.[53] Because rapid rewarming increases the risk of ventricular fibrillation, gradual rewarming is the goal in any severely hypothermic patient, regardless of the intervention selected. Throughout the rewarming phase, the patient is closely monitored for cardiac dysrhythmias and coagulopathies resulting from systemic heparinization if ECR is used. Another option is the use of continuous arteriovenous rewarming. Femoral arterial and venous catheters and the patient's own blood pressure create a circulatory system that drives blood through a heat exchanger.[53]

## SUMMARY

The incidence of altered body temperature and ineffective thermoregulation is significant among patients in the PICU. The risks to physiologic stability in critically ill infants and children are high. Critical care nursing practice can correct environmental factors leading to altered body temperature, support thermoregulatory processes, provide physical comfort during interventions to normalize body temperature, and ensure physiologic stability in patients with altered body temperature or ineffective thermoregulation.

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