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This chapter reviews the importance of thermoregulation and temperature monitoring in anesthetized children. It addresses the significance of keeping children normothermic, and will help the novice understand why pediatric anesthesiologists become apoplectic when faced with the possibility that the small infant they are caring for may become hypothermic. The chapter also covers the management of postoperative fever, a common cause of concern in children.

NORMAL TEMPERATURE PHYSIOLOGY IN CHILDREN

Body temperature is a result of the balance between heat production by the major organs and heat loss to the environment. There is no standard for normal body temperature; individuals exhibit different temperatures, which will be influenced by the time of day, activity, etc. Like adults, children’s bodies contain different compartments that normally exhibit different levels of temperature. The core compartment is composed of the major organs and deep body tissues. The peripheral compartment is composed largely of the extremities. There normally exists a temperature gradient between the core and peripheral compartments that is largely maintained by peripheral vasoconstriction. When vasodilation occurs, during general or regional anesthesia, there is mixing of heat between the core and the peripheral compartments. This normally results in an overall decrease in core temperature. This phenomenon is commonly observed following induction of general anesthesia.

Being a homeotherm, when an infant is placed in a cooler than normal environment, he or she will consume oxygen and expend caloric energy to maintain a normal body temperature. The neutral thermal environment (NTE) is defined as the environmental temperature range that is most consistent with a normal body temperature with the most minimal metabolic heat production, measured as oxygen consumption. The clinical correlate of this is that if a small infant with lung disease has a pre-existing defect in transfer of oxygen into the bloodstream and excretion of carbon dioxide, he or she is more likely to suffer from cellular hypoxemia during hypothermia (i.e., when the infant’s oxygen consumption and metabolic rate are increased). Indeed, studies have documented differences in infant survival rates that depended on the temperature in the incubator. In one study, with an increase in incubator temperature from 85°F to 89°F there was an accompanying 15% increase in survival.

Generally, the smaller and younger the infant, the higher the environmental temperature required to achieve the NTE. Graphs and tables are published that list the temperature required to maintain infants in the NTE based on weight and gestational age. For example, for newborns weighing between 1 kg and 3 kg, this temperature can exceed 85°F (29.4°C). The anesthesiologist caring for small infants should attempt to replicate these conditions in the operating room, in the area immediately surrounding the infant. For a naked infant lying supine on an open platform, it is estimated that the abdominal skin
temperature should be between 36.5°C and 37°C to approximate the conditions required to be in the NTE. Within a narrow environmental temperature range (i.e., the NTE) the human infant’s oxygen consumption is lowest, meaning that it is expending the minimal amount of energy to maintain normothermia (Fig. 15-1). If this environmental temperature is lowered slightly (point “A” in Fig. 15-1), the infant is able to use its compensatory mechanisms (vasoconstriction, brown fat oxidation) to maintain normothermia. However, once this thermoregulatory range is exceeded (at the lower limit in the figure point “B”) the infant’s thermal protective mechanisms can no longer sustain normothermia and its core temperature will begin to drop. Eventually, oxygen consumption will also begin to decrease because of impairment of the temperature regulation center. Just because an infant’s temperature is relatively normal does not mean that it is still residing in the NTE. In fact, its thermal protective mechanisms may be quite active and on the verge of failing to maintain normothermia. When the infant’s body temperature begins to fall, it is an indication that the thermal stress has been so severe that its normal thermal compensatory mechanisms are being overpowered. Furthermore, the presence of either hypoxemia or hypoglycemia impairs the metabolic response to hypothermia, resulting in a more dramatic decrease in body temperature.

The human infant is born with a well-developed temperature regulating system. However, small infants are prone to hypothermia in cold environments and hyperthermia in overly warm environments mainly because of their relatively large surface area-to-volume ratio. The body surface area-to-volume ratio of a tiny premature infant is three to five times higher than an adult’s, and the heat loss per unit body mass is about four times that of the adult. Because of less subcutaneous fat (i.e., less insulation), the range of the environmental temperature in which the infant is able to maintain normothermia is severely limited when compared to the adult. For example, in a naked pediatric anesthesiologist, the lower limit of this control range is approximately 0°C (32°F) whereas for the full-term infant it is 20–23°C (68–73.4°F). Therefore, the temperature within the infant’s immediate surrounding area in the OR should be maintained at a minimum of approximately 75°F since subcutaneous fat is formed mainly in the third trimester of gestation, infants born prematurely are even more at risk for poikilothermic-type behavior.

**NORMAL COMPENSATION FOR HYPOTHERMIA**

When body temperature begins to vary just slightly away (about ± 0.2°C) from the physiologic set-point, involuntary compensatory mechanisms will attempt to return the body’s temperature back to normal. There are a number of these compensatory mechanisms. Those that are most important in small children and that are most different from adults will be reviewed.

**Behavioral Mechanisms**

When most of us feel cold, we instinctively seek a warmer location, or voluntarily increase our muscle activity to generate heat, or even put on more layers of clothing. The small infant does not have the capability to do any of these (although it is often heard in the ICU that babies will instinctively situate themselves toward the warmest part of the isolette).

**Shivering**

Adults and older children have the capability to shiver—the high-intensity involuntary rhythmic muscle activity that is probably the most significant means by which adults produce heat. Young children do not have the capability of efficient shivering. Once anesthetized (without muscle paralysis) efficient shivering in adults is greatly attenuated until the process of awakening.

**Nonshivering Thermogenesis**

Nonshivering thermogenesis describes a cold-induced increase in oxygen consumption and heat production.
that is not inhibited by muscle relaxants. In small infants, nonshivering thermogenesis is probably the most important means of heat production in a cool environment. The thermogenic effector organ – brown fat – is the most significant contributor to nonshivering thermogenesis in the small infant. In the human infant, brown fat accounts for 2–6% of total body weight and is located in the abdominal cavity surrounding the kidneys and adrenal glands, in the mediastinum, and between the scapulae. As opposed to the more abundant white fat, brown fat cells are rich in mitochondria, contain a dense capillary network, and are richly innervated with sympathetic nerve endings. When norepinephrine release is stimulated by sympathetic activity, triglycerides are hydrolyzed to free fatty acids and glycerol, with heat production resulting from enhanced oxygen consumption. Immediately after an infant is exposed to a cold stimulus the metabolic rate begins to increase, even before core body temperature decreases. Even a mild cold stimulus such as unheated preoxygenation can trigger the onset of an increase in metabolic heat production. In infants exposed to a cold environment, nonshivering thermogenesis is capable of doubling the metabolic rate. However, the drop in temperature required to initiate nonshivering thermogenesis is unknown. In one study of infants anesthetized with propofol and fentanyl, there was a lack of nonshivering thermogenesis with a temperature drop of 2°C.

**Thermoregulatory Vasoconstriction**

Thermoregulatory vasoconstriction occurs in the peripheral compartments in response to cold receptors on the skin. It serves to limit heat loss to the environment. In children undergoing abdominal surgery with isoflurane anesthesia, thermoregulatory vasoconstriction is attenuated by an average of about 2.5°C less than the unanesthetized state (Fig. 15-2). This is similar to the values found in unanesthetized adults. A similar study using halothane as the maintenance agent demonstrated a higher temperature at which thermoregulatory vasoconstriction was triggered (about 1–2°C difference in triggering threshold). These values are higher than for adults anesthetized with halothane.

**COMPLICATIONS OF HYPOTHERMIA IN THE SMALL INFANT**

Other than the direct depressant effects of hypothermia on cerebral and cardiovascular function, hypothermia sets into motion a variety of physiological compensation mechanisms that increase oxygen consumption and may ultimately adversely affect normal physiology (Fig. 15-3). Cooling results in release of norepinephrine. This, along with the direct effects of hypothermia, results in widespread vasoconstriction. Peripheral vasoconstriction may restrict oxygen delivery to tissues and cause cellular hypoxia manifested as a metabolic acidosis. Pulmonary vasoconstriction will increase pulmonary arterial pressures, and cause increased susceptibility to right-to-left shunting at the atrial level through a patent foramen and through a patent ductus arteriosus. This will in turn cause additional peripheral tissue hypoxia.

**Figure 15-2** The central thermoregulatory threshold in 32 healthy children and infants undergoing abdominal surgery. Although there was a trend towards increased threshold temperatures in smaller patients, differences between the groups were not statistically significant. (Reproduced with permission from Bissonnette B, Sessler DI: Thermoregulatory threshold in infants and children anesthetized with isoflurane and caudal bupivacaine. *Anesthesiology* 73:1114–1118, 1990.)

**Figure 15-3** Vicious cycle that develops when a neonate is hypothermic. (Reproduced with permission from Klaus MH, Fanaroff AA: *Care of the High Risk Neonate*, 5th edn, WB Saunders, Philadelphia, 2001.)
Mild hypothermia (34–36°C) in healthy infants and children during peripheral procedures probably does not result in adverse effects, and does not influence postoperative recovery indices. Postoperative shivering is uncommon in children. In one audit of 376 children, the incidence of postoperative shivering was 14.4%, and was associated with age > 10 years, inhalation induction of anesthesia, use of atropine, use of opioids, duration of anesthesia > 40 minutes, and perioperative decrease in body temperature.

**HEAT LOSS DURING ANESTHESIA**

After induction of general anesthesia, an initial decrease in core temperature results from the redistribution of heat from the core to the periphery. This is largely caused by a combination of direct vasodilation by the anesthetic agents and an anesthetic-induced inhibition of thermoregulatory vasoconstriction that occurs at a lower than normal core temperature. In children the administration of general anesthesia blunts the ability for the central nervous system to trigger compensatory vasoconstriction by approximately 2.5°C, as compared with approximately 0.2°C in the unanesthetized state. This threshold is similar to that of adults. Since infants and small children have a relatively greater proportion of their body mass contained in the core compartment, they may, at least initially, lose proportionately less heat because of redistribution of core heat to the periphery. Their relatively small extremities will not absorb as much heat from the core compared to an adult.

Following this initial decrease in core temperature from redistribution, infants will likely continue to lose heat to the environment at a faster pace than older children and adults. This is mainly caused by their relatively large surface area-to-volume ratio, paucity of subcutaneous fat, immature epidermal barrier, and limited capacity for metabolic heat production. In addition, there is a relatively greater contribution to body cooling from unwarmed intravenous solutions and sterile irrigating solutions.

**Mechanisms of Heat Loss to the Environment**

*Radiation* is the process by which heat is lost from the child to any colder surrounding structures (e.g., walls in the OR) by the transfer of photons, and is not influenced by the temperature of the surrounding air. Radiation normally accounts for the greatest percentage of heat lost during anesthesia. During transport of a neonate to and from the operating room, heat lost through radiation can be decreased by use of a double-shelled incubator, or another type of barrier between the infant and the surrounding incubator walls, such as a blanket.

*Conduction* refers to the direct transfer of heat between contiguous structures. Examples include loss of heat from the child to the operating room table, or the hypothermic effect of infusion of cool intravenous fluids. Because of the relatively larger surface area-to-volume ratio of infants, conduction may influence heat loss more than in older children or adults. Heat lost by conduction is reduced by using a warming mattress underneath the child, increasing the ambient temperature in the operating room, use of a forced warm air blanket on nonsurgical areas of the body, and warming infused intravenous fluids and sterile prep solutions.

*Convection* is the loss of heat by the movement of air flowing past the surface of the skin. Heat lost through convection is minimized by ensuring that exposed parts of the child are covered as much as possible.

*Evaporation* describes the loss of heat by the energy depleted when water is dissipated from exposed surfaces of the body, such as the skin, visceral organs, and respiratory epithelium. Evaporative heat loss is minimized by humidification of inspired gases, covering exposed skin surfaces, and using warmed sterile prep solutions.

**Prevention and Treatment of Perioperative Hypothermia**

Preoperative warming of the extremities is perhaps the most effective method for prevention of the initial decrease in temperature due to redistribution. However, this is not practical in most children. Therefore, more effective means must be utilized to prevent large decreases in temperature. Every attempt should be made to achieve cutaneous warming by covering all exposed areas with sheets or blankets. This will significantly decrease radiant, convective, and evaporative heat loss. Many institutions utilize radiant warmers, which are kept over the infant during induction of anesthesia and placement of lines and monitors. These devices may help prevent heat loss via evaporation and conduction of heat to the surrounding cold air. Airway humidification can prevent evaporation. There are two methods with which to humidify the airway. The simplest is by using a heat and moisture exchanger (HME) to passively trap the patient's own heat and moisture within the airway. The second is the placement of an active humidification device within the anesthesia breathing circuit. This device can both prevent heat loss and add heat to the child's body via the respiratory tract. However, its use is probably not warranted for peripheral surgery of relatively short duration. In a study of 20 infants weighing between 5 kg and 30 kg who were randomized to receive active humidification, passive humidification, or none, there was good correlation between humidification and core body temperature, but no difference between active and passive humidification.
Heated water-filled mattresses are routinely used to prevent conductive heat loss from the infant to the OR table and to transfer heat to the infant. However, its safe temperature range is narrow – below 35°C infants may lose heat to the mattress, and above 38°C there is the possibility of overheating and burns. It is most effective in infants weighing less than 10 kg. All infusions and sterile scrubbing and preparation solutions should also be appropriately warmed to prevent conductive heat loss within and around the child’s body. The flow of warm air across a child’s skin produced by forced-air warming blankets prevents heat loss to the environment and may even effectively warm patients via radiant shielding and convection. Of all devices, these are one of the most effective and should be used in all cases where hypothermia is possible.

Lastly, one of the most effective methods for prevention of hypothermia in small children is warming the OR environment. Eventually, the walls of the OR will also become warmed with consequent decrease in loss of radiant heat. One must be sensitive, however, to the comfort of the surgeons and nurses under the hot operating lights. Therefore, the OR should be maintained warm, but then, once the child is covered with blankets and a forced-air warming blanket, the air temperature can be turned down for comfort, after ensuring that the child’s core temperature is in the satisfactory range.

**Where Temperature Should be Measured**

Most studies demonstrate that axillary temperature measurement is as efficacious as tympanic, nasopharyngeal, or esophageal sites. However, when hypothermia is possible, true representations of core temperature are preferred, such as distal esophageal, rectal, and nasopharyngeal locations. Tympanic membrane temperatures are also reliable but fear of injuring the tympanic membrane dissuades most anesthesiologists from choosing this route. When using distal esophageal temperature monitoring in neonates and small infants during a laparotomy, one must ensure that the tip of the temperature probe has not entered the stomach – falsely elevated readings can result from warming of the stomach if directly exposed to the heat of the overhead lights or warm irrigation solution. Disposable skin temperature devices are generally not considered to be useful in the perioperative environment where diagnosis of temperature alteration is clinically important.

**Regional Anesthesia**

Regional anesthesia may also contribute to onset and maintenance of hypothermia by vasodilation within the affected extremities and subsequent redistribution of heat from the core component. Furthermore, inhibition of peripheral sympathetic tone may prevent thermoregulatory vasoconstriction and inhibit heat production by utilization of brown fat. However, few studies in children have been performed to assess the influence of regional anesthesia on intraoperative temperature regulation. A study in children anesthetized with halothane demonstrated that caudal anesthesia with bupivacaine had no effect on the threshold for thermoregulatory vasoconstriction.

**PERIOPERATIVE HYPERThERMIA**

Intraoperative hyperthermia is probably less common than hypothermia, yet it occurs regularly in children. It is associated with peripheral or head/neck procedures where there is minimal heat or fluid loss and warming maneuvers are being used. Examples include ear/nose/throat (ENT) and dental surgeries as well as procedures on the distal limbs. For these types of procedures, it is usually sufficient to use a blanket that covers the child’s body, and perhaps a warming blanket underneath the child on the OR table. Use of a forced warm blanket in these situations will usually contribute to hyperthermia.

Although relatively common, postoperative fever (generally defined as a core body temperature greater than 37.5°C) in children is a consistent cause of concern. Surgeons worry about wound infections and anesthesiologists worry about postoperative signs of malignant hyperthermia. The fact is, however, that postoperative fever is extremely common in children, and is rarely due to either wound infection or malignant hyperthermia. The precise cause of postoperative fever is unknown, but it is theorized to be a transient adjustment of the body temperature “set-point” as a response to the surgical stress. An audit of 150 consecutive pediatric urologic patients revealed that 74% aged less than 1 year and 28% aged greater than 4 years exhibited postoperative fever, none of whom was otherwise clinically ill. Similar incidences have been reported in the pediatric orthopedic, plastic, and tonsillectomy populations. A study that examined fever patterns in 150 children following inguinal hernia repair demonstrated that one-third of the children had a fever of greater than 37.5°C on the evening of the surgery. No studies in any particular surgical specialty indicate that fever is a marker of a serious clinical entity.

When asked to evaluate a child with postoperative fever, the anesthesiologist should review the anesthetic and surgical events as a prelude to determining the cause. Obvious signs of surgical infection (e.g., redness or exudate at the surgical site) should be sought. The child should be evaluated for concomitant upper respiratory tract illness or middle ear infection that may have been present preoperatively. Abnormal lung sounds should prompt investigation of possible lower respiratory tract infection. If the child is ill-appearing, intravenous hydration should be continued
and the child evaluated for overnight hospital admission. Malignant hyperthermia is extremely rare in this setting. One possible clue that a malignant hyperthermia episode might be occurring is a child demonstrating a large minute ventilation that is out of proportion to the child’s clinical status.

**ADDITIONAL ARTICLES TO KNOW**


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**Article To Know**


In this classic article from 1973, Dr Goudsouzian and his colleagues from the Massachusetts General Hospital set out to evaluate the usefulness of a warming blanket placed underneath infants and children above 18 months of age during general anesthesia. The warming blanket was set to a temperature of 40°C and the patients were randomized to the warming blanket group or a control group. Lower esophageal temperatures were recorded every 15 minutes following induction of general anesthesia. When measurements on 25 children were collected, the authors plotted their results on a scatter diagram and observed an obvious difference between children less than or greater than 0.5 m² in surface area. The two groups were then separated and their temperatures throughout the case were graphed separately.

Fig. 15-4A represents 13 children <0.5 m² in surface area, in which 7 were placed on warming blankets. There is an obvious difference in esophageal temperature throughout the case. This difference does not exist in the children >0.5 m² in surface area (Fig. 15-4B). The authors postulated that the results are due to smaller infants having a greater ratio of surface area to volume, and thus able to reap greater benefits from the warming blanket, which prevented conductive heat loss to the operating table. In general, children less than 0.5 m² in surface area weigh less than 10 kg.

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**Figure 15-4** Changes in esophageal temperature (mean ± SD) in infants and children: patients with

(A) <0.5 m² and (B) >0.5 m² of body surface area. (Reproduced with permission from Goudsouzian NG et al.: *Anesthesiology* 39:351–353, 1973.)