

HYPOTHERMIA AND TEMPERATURE REGULATION **CONSIDERATIONS DURING ANESTHESIA**

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INTRODUCTION

For all us who administer general anesthesia on a daily basis, postoperative patient's sensation of cold, development of shakes and shivering are extremely common events that have occurred to most of our patients. From my first days of residency anesthesia delivery experience, I can remember these events occurred as part of the anesthetic case, and I expected them to occur regularly. I believed then that these were unescapable side effects which were part of the recovery from anesthesia and were almost unavoidable consequences. After fifteen years of anesthesia experience and having gained more knowledge about what happens to the patient after induction, I now know that my first impression of postoperative heat loss during surgery is still unavoidable, but the inadvertent perioperative and postoperative hypothermia indeed is largely preventable. Hypothermia remains the most frequent complication of surgery under anesthesia.

DEFINITIONS

Homothermia is the highly evolved physiologic characteristic feature of certain animals which allows them to maintain their internal body temperatures nearly constant. Mammals and birds belong to this group of animals which possess a sophisticated and intrinsically complex thermoregulatory mechanism allowing them to usually maintain a core body temperature within $\pm 0.5^{\circ}\text{C}$. Normothermia can be best defined as the range of body core temperature between $36.5 - 37.5^{\circ}\text{C} \pm 0.5^{\circ}\text{C}$. Any core body temperature below 1°C in "Homothermics" is therefore considered hypothermia. Hypothermia is defined as a core temperature less than 36°C . Mild hypothermia is defined as ranging from 1°C to 2°C below body core temperature¹ while moderate hypothermia constitutes a body core temperature of 35°C . Severe hypothermia is a body core temperature below 35°C ² while any temperature below 28°C is considered deep hypothermia where consciousness is lost, sinoatrial pacing becomes erratic, ventricular irritability increases, and below 26°C rigidity and myoclonus ensues.¹

SOURCES OF HEAT LOSS

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anesthetic induced changes in the body's regulatory heat mechanisms in combination with exposure of cold operating room and dental operatory environments will precipitate hypothermia in patients while undergoing both regional and general anesthesia. All surfaces above absolute zero (i.e. 273 °K or -273 °C) radiate heat just as all surfaces absorb heat from their surroundings. Physically speaking, heat transfer is proportional to the fourth power of the absolute temperature difference between surfaces.¹ Heat loss during anesthesia will occur from patient to environment from several sources (i.e. physical processes) which are radiation, evaporation, conduction and convection.³

Radiation

Energy transmitted by waves transferred through a medium.⁴ Accounts for 60% of heat loss in human. This loss of radiant heat is generally dependent on cutaneous blood flow and the body's exposed surfaces to the environment. This is the major source of heat loss in most surgical patients.

Evaporation

The physical process of converting a liquid or a solid into vapor.⁵ This phenomenon accounts for 20% of the heat loss. Heat loss occurs from the energy needed to vaporize liquid from mucosal, serosal surfaces, skin and lungs. Evaporative heat loss is dependant on exposed body surface area and the relative humidity of ambient gas (air). Respiratory system heat loss constitutes a small amount of the heat loss during surgery.⁶ Sweating is the most efficient process of evaporative heat loss patient's have, but diaphoresis does not generally occur to any appreciable extent during anesthesia.¹ Major open wound exposure during surgery can, however, account for a significant heat loss. This circumstance however is rarely the case in dental and/or oral maxillofacial surgery.

Conduction

The transfer of energy via sound, heat, nerve impulses or electricity.⁷ The heat transferred from a warm to a cool object (medium). This heat loss accounts for 5% of human heat loss and is proportional to the area of the body exposed, the relative difference in temperature between the mediums or surfaces, and thermal conductivity of these. This heat loss occurs only when two mediums are in direct apposition (touching) to each other. Generally, this constitutes a small amount of the loss during surgery.¹

Convection

The transmission of heat in liquids and gases by a circulation carried on by bulk movement of the heated particles to a cooler area.⁸ This phenomenon is a special type of conduction heat loss

through gases. The loss accounts for 15% of the body's heat loss by conduction to a moving gas. It is proportional to the square root of the air speed, and it is the second most important process of heat loss in the anesthetic patient.¹ The high air flows of 10 to 15 room-volume changes per hour, like laminar flows in orthopedic operating rooms, can result in significant heat losses.³

THERMOREGULATORY CONCEPTS

The human body's temperature regulation system is under the control of the autonomic nervous system. The temperature control occurs through known physiologic positive and negative feedback systems which correct any temperature perturbation from its normal or "preset" value. The body's ability to regulate temperature occurs via the skin's thermal input, from the hypothalamus and extrahypothalamic portions of the brain, deep tissues and spinal cord.⁹ During exposure to hot or cold environments, temperature sensors provide important information to central regulatory centers in the anterior hypothalamus which have certain temperature thresholds that maintain the body normothermic. The complex interrelated interacting elements of the thermoregulatory system work conceptually in three physiologically distinct phases as a cohesive entity. These phases are afferent sensing, central control and efferent response.¹

Afferent Sensing

Afferent input occurs from thermal receptive cells throughout most of the body. Temperature sensitive cells are different for heat and cold temperature cells. Cold signals travel mainly via A δ (delta) nerve fibers and warm input travels by unmyelinated C fibers which also sense and conduct pain sensation.¹⁰ This is why the body is unable to discriminate between sharp pain and intense heat. The cutaneous information is then transmitted via the anterior spinothalamic tracts of the spinal cord to the hypothalamus. The skin and spinal cord contribute roughly 20% of the total body's thermal input information.¹¹

Central Control

The hypothalamus is the center of the thermoregulatory site in the brain which is mainly responsible for integrating most of the temperature input information and coordinating the different autonomic functions which will allow the body to autoregulate itself in order to maintain a homothermic level. Approximately 80% of this thermal input is derived from core body temperature.^{12,13} There is evidence however that some thermoregulatory functions occur at the level of the spinal cord not involving the hypothalamus.¹ How the body determines absolute thresholds is not known, but it appears the mechanisms could be mediated by neurotransmitters such as norepinephrine, dopamine, 5-hydroxytryptamine, acetylcholine, prostaglandin E₁ and other neuropeptides. Factors such as circadian (daily) rhythm, exercise, food intake, infection, hypothyroidism, hyperthyroidism, women's menstrual cycle, anesthetics and other drugs such as alcohol, sedatives and/or nicotine can alter the threshold.^{1,14}

Efferent Response

The body's effect to changes in the "predetermined" normal values of temperature will activate effector mechanism response by increasing metabolic heat production. They can also cause

alteration of environmental heat loss by activating behavioral changes to regulate and modify the patient's circumstances to compensate for mechanisms which the body cannot control. Such changes include dressing appropriately, modifying the environment's temperature, assuming bodily positions which diminish heat loss or increasing the body's skin surface apposition (e.g. fetal position, bringing arms centrally into the body), and increasing voluntary movement to develop a greater amount of heat production via metabolic means or through friction. Obviously, behavioral environmental regulation cannot be considered an option of thermal regulation initiated by an anesthetized patients. Such behavioral or circumstantial changes would have to be instituted by the anesthesia and/or surgical personnel involved in the case. These responses or considerations will be discussed later since some of these will help prevent hypothermia.

Natural laws will generally dictate the physical process many biochemical systems tend to follow. Considering free energy and entropy as common thermodynamic entities at play in any heat exchanging process, any physiological modifications that require the least amount of energy to achieve the necessary result will most certainly be initiated first by the body. The adages "The shortest distance between two points is a straight line, and haste makes waste" certainly shed light on this evolutionary concept. Therefore, the anticipated response which will be initiated first is an energy-efficient autonomic mechanism such as vasoconstriction which is initially maximized before any other metabolically-costly response is started. Cutaneous vasoconstriction is the most consistently used autonomic effector mechanism since metabolic heat is lost primarily through convection and radiation from skin surfaces, and consequently reduces heat loss.¹

Metabolic effector changes include nonshivering and shivering thermogenesis. Therefore, a core temperature below the thresholds for response to cold provokes an autonomic effector response of vasoconstriction first followed by nonshivering thermogenesis, and finally shivering. Core temperature exceeding the hyperthermic threshold produces active vasodilation and sweating. No thermoregulatory responses are initiated when core body temperatures are between these thresholds.^{15,16} The intensity of nonshivering thermogenesis increases linearly to the difference between the mean body temperature and its threshold. It doubles heat production in infants¹⁷ and increases only slightly in adults.¹⁸ The major site sources in adults of energy for nonshivering thermogenesis are the skeletal muscle and brown fat tissue. This process is metabolically controlled by norepinephrine primarily which is released from adrenergic nerve terminals and mediated locally by an uncoupling protein.¹⁹

Shivering can increase metabolic heat production 50% to 100% in adults as long as it is sustained. The increase in heat is small as compared to exercise which could reach 500%, but this is clearly not an option for the patient under anesthesia. Newborns do not shiver, and this mechanism does not develop until several years of age.¹ They have a greater surface area to body weight ratio which allows them to lose body heat more readily. Infants and newborns respond to cold stress by increasing norepinephrine production which enhances metabolism of brown fat, produces pulmonary and peripheral vasoconstriction, which if it becomes profound, right-to-left shunting, hypoxemia and metabolic acidosis can result.²⁰

Sweating which is mediated by sympathetic postganglionic cholinergic (parasympathetic) nerves is an active (i.e. energy consuming) process and is prevented by atropine administration and nerve blockade.²¹ Sweating is the only mechanism by which the body can dissipate heat in an environment exceeding body core temperature since all other mechanisms are temperature-gradient dependent to physically allow for heat transfer. Fortunately, it is an extremely efficient process. Active vasodilation is mediated by an unknown factor released by the sweat glands, and requires intact sweat gland function which is also inhibited by nerve blocks. In addition, the maximum vasodilation threshold is generally delayed until core body temperature is well above that of triggering the maximum sweating intensity.¹ Sweating may occur in response to anxiety, pain, hypercarbia, noxious stimuli in the presence of inadequate anesthesia, vagal reaction or as a thermoregulatory response to hyperthermia.³

THERMOREGULATION DURING ANESTHESIA

All general anesthetics and regional anesthesia can impair normal autonomic thermoregulatory control.³ Volatile and nonvolatile anesthetics predispose patient's to heat loss because of their vasodilatory properties.²² Most narcotics reduce vasoconstriction mechanisms for heat conservation because of their sympatholytic properties. Muscle relaxants reduce muscle tone and prevent shivering. Regional anesthesia produces sympathetic blockade, muscle relaxation, and sensory blockade of peripheral thermal receptors inhibiting compensatory thermal responses. The only anesthetic drug that minimally influences thermoregulatory control is midazolam (Versed[®]).²³

General Anesthesia

Temperature declines most dramatically during the first ½ of anesthesia.^{1, 16} This makes hypothermia not only a real possibility but almost an inescapable reality. For all anesthetic providers who administer IV anesthesia beyond midazolam, your patients will most likely develop some degree of hypothermia, if no countermeasures are provided, as the mere consequence of this anesthetic delivery. Ergo, the common patient's perception or feeling of postoperative cold sensation and/or shivering is real! Core temperature usually decreases in a very characteristic pattern of an initial rapid decrease during the first ½ hour followed by a slow linear reduction in core temperature up to 2 hours after induction. Finally, core temperature will stabilize and subsequently remain virtually unchanged after 3-4 hours from induction. Each of these three portions of the intraoperative drop in body core temperature has a different etiology in their development.

The first portion of the rapid decrease in core temperature develops due to shifting in body heat from the core to the periphery which leads to vasodilation. Simply speaking, hypothermia results from internal redistribution of the body core heat to the extremities and peripheral tissues. All general anesthetics reduce the threshold for response to hypothermia from 37°C to 33-35°C.¹ In order to understand why there is this significant drop in temperature, one must know that the body core temperature is isolated in 50% of the body's mass, mainly in the trunk and

head. The remaining body mass is normally 2-4°C cooler than core temperature. This temperature gradient is normally maintained by thermoregulatory vasoconstriction in awake individuals²⁴ resulting in a normal extremity and skin temperature of 31 - 35°C and 28 - 32°C, respectively.

Following core-to-peripheral redistribution there is a slower reduction in core temperature that results simply from heat loss exceeding heat production. The final portion represents a passive thermal steady-state where heat production equals heat loss. This almost constant temperature state, or plateau, occurs either in patients that are maintained relatively warm or with those individuals who have active peripheral vasoconstriction triggered by core temperatures of 33-35°C.²⁵

Regional Anesthesia

Any regional anesthesia produces a similar pattern of heat loss and hypothermia as that of general anesthesia. Epidural and spinal anesthesia decrease the threshold for triggering vasoconstriction and shivering about 0.6°C.²⁶ As in general anesthesia, regional anesthesia lowers the shivering and vasoconstrictive thresholds via central effect. In addition, regional anesthesia through its peripheral blocking effects also prevent shivering and vasoconstriction due to the interruption or lack of sensation of cold from the periphery.^{1,2} The brain interprets this as a warming, and it is this combination of vasodilation and blocked cold sensory input that results in a paradoxical experience of patient's having sustained significant heat loss with shivering, yet they do not feel cold. In addition, most of these patients receive supplemental sedatives and narcotics to lessen anxiety and more comfortably undergo their procedures which most likely increases their hypothermia. Because core temperature monitoring remains rare during regional anesthesia, substantial hypothermia commonly goes undetected.²⁷

HYPOTHERMICALLY INDUCED ANESTHETIC PHYSIOLOGIC CHANGES

Hypothermia is associated with a multitude of physiologic organ system effects which can either be beneficial or detrimental depending on the surgery in which the event occurs, the desired effect needed for any given procedure under anesthesia, and the degree of hypothermia. Management of temperature effects thus depends not only in understanding its effects, limitations and complications in order to beneficially and safely apply hypothermia for an appropriate therapeutic outcomes or for the prevention of its severe complications.

A number of physiologic changes are associated with hypothermia which include cardiovascular, respiratory, hepatic, renal, neurologic, metabolic, hematologic, immunologic, drug pharmacology, shivering and wound healing.^{1,2,3,28,40,41,46} Many organs have poor compensation (reserve) for hypothermia making a limited stressful anesthetic or surgical episode potentially dangerous. In addition, hypothermic physiologic changes can be of significant clinical importance in certain uncommon medical conditions in which prevention is critical in insuring that catastrophic surgical and anesthetic outcomes are not encountered. Conditions such as

sickle cell disease,²⁹ hypokalemic periodic paralysis,³⁰ pulmonary alveolar proteinosis (PAP),³¹ and connective tissue diseases such as scleroderma³² require special anesthetic considerations. These commonly associated effects include:

Cardiovascular - Increased pulse and blood pressure (postoperatively), systemic vascular resistance (SVR), contractility, ventricular dysrhythmias and irritability, myocardial depression and secretion of catecholamines. Cardiac output and heart rate are decreased (intraoperative).

Respiratory - Strength is diminished at body core temperature of less than 33°C, but CO₂ ventilatory response is unaffected.

Hepatic - Blood flow and function are diminished which will decrease significantly the metabolism of some drugs.

Renal - Decrease in renal blood flow due to increase in renal vascular resistance. Inhibition of tubular resorption maintains normal urinary volume until progressively lower temperatures inhibit reabsorption of sodium and potassium and an antidiuretic hormone (ADH) mediated diuresis results. Plasma electrolyte usually remain normal.

Neurologic - Decreased cerebral blood flow, increased cerebrovascular resistance, decreased minimum alveolar concentration, delayed emergence from anesthesia due to direct depressant effects of hypothermia, altered mental sensorium to include drowsiness and confusion.

Metabolic - Decreased metabolic rate, decreased tissue perfusion leading to metabolic acidosis, and hyperglycemia from catecholamines may occur. Increased oxygen consumption may occur due to shivering postoperatively.

Hematologic - Increased blood viscosity, thrombocytopenia, leftward shift of the hemoglobin dissociation curve causing increase difficulty of oxygen unloading from hemoglobin leading to hypoxia, alterations in coagulation via impaired platelet function, decreased coagulation factor activity leading to a greater intraoperative bleeding and blood loss.

Immunologic - Impaired immune system function increasing rate of postoperative wound infection.

Drug Pharmacology - Decreased hepatic blood flow, and metabolism coupled with decreased renal blood flow and clearance result in decreased anesthetic requirement, delayed awakening due to reduced rates of drug clearance.

Shivering and Wound Healing - Increased shivering which can increase heat production by 100% - 300% with concomitant oxygen consumption up to 500% and increased production of CO₂. Vasoconstriction and the reduced delivery of oxygen to injured tissues also leads to a delay in wound healing and a significant rate of postoperative infection rate.

Special Medical Conditions - Hypothermia can precipitate a sickle cell crisis in sickle cell anemia patient since sickling of erythrocytes occur with decreasing arterial oxygen tension.³³ Cold is a major triggering cause of paralysis in hypokalemic periodic paralysis.²⁹ Because of the abnormal accumulation of amorphous lipoproteineaceous matter in the alveoli normally produced by PAP patients, these patients have a restrictive pattern with decreasing lung volumes, static lung compliance and diffusing capacity predisposing them to hypoxemia because of their decreased functional residual capacity and intrapulmonary shunts. In these patients, hypothermia will severely aggravate their hypoxemia leading to serious ventilatory problems. In

addition, pulmonary lavage is normally performed to improve arterial saturation via double lumen endotracheal tubes. The solutions used for lavage can further exacerbate a temperature drop if not appropriately warmed.³¹ In scleroderma patients, because of their vasospastic phenomenon and difficulty with ventilatory effort, cold will precipitate Raynaud's phenomenon making blood pressure taking difficult and predisposing them also to respiratory complications.²⁹ Moreover, with peripheral temperatures already being lower than normal because of their preexisting diminished peripheral vascular blood flow, hypothermia would make core-to-peripheral redistribution effect of temperature more profound.

HYPOTHERMIA BENEFITS AND COMPLICATIONS

Benefits

It has been known that a substantial protection against cerebral ischemia and hypoxia can be gained by providing a 1°C - 3°C hypothermia. The potential protection provided by reduced core temperature (i.e. ≈34°C) is so great that it has been used very successfully in many neurosurgery cases and in other procedures in which tissue ischemia can be anticipated. This principal becomes a justifiable and established cerebral protective anesthetic technique to enhance cerebral tolerance during circulatory arrest procedures (e.g. cardiopulmonary bypass).²⁸ Possible mechanism of these effects are largely due to a reduction in cerebral metabolic rate (CMR) by reduction in energy utilization as measured by cerebral metabolic rate of oxygen (CMRO₂),³⁴ decreased release excitatory neurotransmitters, inhibition of activation of protein kinase C(early), DNA transcription, apoptosis, proinflammatory cytokines, and finally preservation of late postischemic enzyme function mainly protein kinase C.^{35,36} CMRO₂ is decreased 7% for every 1°C reduction in temperature.³⁷ In addition hypothermia is associated with improved intracranial pressures (ICP) and cerebral perfusion pressure(CPP).²⁸

Therapeutic hypothermia (deliberately induced) has been also shown to unequivocally improve outcome from cardiac arrest recovery.^{38,39} Induction of therapeutic hypothermia during surgery is relatively easy because of the anesthetic effects which impair thermoregulatory response. Hypothermia decreases the whole body metabolic rate by 8% per °C to about half the normal rate at 28°C,¹ oxygen demand drops and those tissues with normally have high oxygen consumption rates, like the brain, have a proportionally greater drop in reduction of oxygen use. This "lowered" metabolic rate allows for aerobic metabolism to continue through greater periods of compromised oxygen supply. Toxic waste production decreases in proportion to the decline in metabolic rate. Ischemic changes makes the body switch to anaerobic metabolism which is extremely inefficient and will not provide adequate energy supply. This will in turn cause tissue damage due to toxic metabolic byproducts such as superoxide radicals and lactate. Finally, hypothermia decreases the triggering of malignant hyperthermia and reduces the severity of the event once triggered.

Complications

The three most common complications associated with mild hypothermia are a three-fold increase (i.e. 300%) in morbid myocardial events,⁴² a three-fold increase in the risk of surgical

wounds infection and prolonged hospitalization,⁴³ and finally, increased blood loss and transfusion requirements.⁴⁴

Myocardial ischemia is one of the leading cause of unanticipated perioperative death. Hypothermia through increase in heart rate, blood pressure, oxygen consumption, shunting and catecholamine release will easily predispose patients to morbid myocardial events. Hypothermia most likely contributes to wound infections by both impairing immune function and by the thermoregulatory vasoconstriction which in turn diminishes oxygen delivery to surgical sites. Fever will increase white blood cell diapedesis and mobilization, therefore being protective for infections. Infections are aggravated when a systemically induced fever is prevented. Mild hypothermia hampers coagulation. Local temperature seems to be the important factor in causing a cold-induced defect in platelet function⁴⁰ and not core temperature. In addition, hypothermia induces impairment on enzymes of the coagulation cascade. Drug metabolism is markedly decreased with hypothermia. With a constant infusion of propofol, an increase of about 30% more than normal in plasma concentration is seen in patient's who are 3°C hypothermic.⁴⁵ Muscle relaxants and volatile anesthetic pharmacodynamics and pharmacokinetics are altered. Minimum alveolar concentration (MAC) is reduced 5% per °C, and the duration of action of vecuronium is more than doubled by a 2°C reduction in core temperature.¹

In the postoperative period, mild hypothermia, and even at the lower levels of normothermia, cause significant discomfort in the awake patient. Hypothermia will delay the recovery of patients.^{41,46} by decreased metabolic rate of drugs, delaying awakening and altering mentation. Harmful effects also result from physiologic efforts of rewarming. Vasoconstriction exacerbates hypertension. Increased oxygen consumption due shivering and nonshivering thermogenesis, not only worsens the effects of pulmonary shunt on arterial oxygen content but demands increases in cardiac output that patients compromised by cardiac disease or age may not be able to achieve. This situation is extremely important to address in our cardiac patients to ensure that during recovery a postoperative mild hypothermia does not tip a patient into cardiac distress, even though intraoperative anesthetic or surgical adverse problems have not occurred! The costs of perioperative hypothermia vary and can range from price of an extra cotton blanket to increased patient morbidity and mortality. Studies have shown that a body temperature averaging 1.5°C below normal cause cumulative adverse outcomes which added \$2,500 to \$7,000 per surgical patient to hospitalization costs.²

The incidence of postanesthetic shivering-like tremors is as high as 40%. Postanesthetic shivering is a thermoregulatory response to intraoperative hypothermia which is always preceded by core hypothermia and arteriovenous shunt vasoconstriction.¹ Shivering can cause up to a 500% increase in oxygen consumption.³

TEMPERATURE MONITORING

The preferred sites for core temperature monitoring include, tympanic membrane, pulmonary artery, distal portion of the esophagus and nasopharynx. These sites, although not necessarily easily accessible, will prevent over heating and will allow detection fairly accurately of malignant hyperthermia. These sites constitute anatomical areas of highly perfused tissues whose temperature is uniform and high in comparison to the rest of the body.¹ Oral, axillary, rectal and bladder are sites which core body temperatures can be estimated fairly accurately. Skin surface temperature are not considered accurate or reliable because of the great fluctuations and variable offset.

Needless to say, any person undergoing an inhalational or an intravenous anesthetic be it a sedation and/or general for more than 30 minutes, temperature should be monitored. Special group of patients such as pediatric and elderly patients need to be monitored. Finally, anyone undergoing regional anesthesia should be measured when changes in body temperature are intended, anticipated or suspected. Pediatric patients because of the relatively large surface area to body mass ratio are particularly prone to heat loss and have less effective compensatory mechanisms for preserving and generating body heat, need monitoring. Geriatric patients are also in this group of special patients because of age-related body fat atrophy, decrease metabolic function and a more severe central hypothermia is required to trigger autonomic thermoregulatory responses.⁴⁶

THERMAL PERIOPERATIVE CONSIDERATIONS AND HYPOTHERMIA PREVENTION

The best management for hypothermia, like most complications in anesthesia, is prevention. Intraoperative hypothermia can be minimized by any technique which limits cutaneous heat loss to the environment, evaporation from surgical sites and respiratory tract, conductive cooling from excessive gas flow rates, cold intravenous fluids or irrigating solutions. Remember that most of the anesthetized patient are poikilothermic, the exhibition of body temperatures which varies with the environmental temperature,⁴⁷ because they do not get sufficiently hypothermic to set off thermoregulatory responses. The initial 1-1.5 °C reduction in core temperature change is not possible to prevent. It will happen! Mean body temperature will decrease when heat loss to the environment is greater than metabolic heat production. The body will lose about 1°C per hour when the heat lost to the environment is greater than twice the metabolic production.¹ Normally, approximately 90% of the body's metabolic heat loss is through the skin surface. Therefore, the skin should become the site of focus in order to modulate thermal manipulation. Maintenance of intraoperative core temperature should be kept higher than 36°C. Prevention starts with preoperative preparation of the patient and the operating environment.

Pre-Warming

Inform your patient to dress appropriately the day of surgery. Having patients who are having dental and/or oral maxillofacial procedures can come in with warm appropriate clothing covering most of their body. Discourage the use of shorts, shirts which do not cover most of their torso

and upper extremities, and request socks. The patient can be pre-warmed before induction with forced-air systems (i.e. Bair hugger) to minimize the drop in core temperature from redistribution. With pre-warming the extremities which are normally about 2°C to 4°C lower than core temperature will require less heat to warm when the core-to-peripheral redistribution of heat occurs. It is an inexpensive way to reduce perioperative hypothermia. Cotton blankets, other than offering comfort to the patient, their warmth only lasts three to five minutes and will not increase peripheral body temperature. However, their use will at least minimize the normal heat loss by minimizing the patient's skin exposure.

Operating Room

The operating room temperature is the most important factor in influencing heat loss from the lost radiation and convection from skin, and by evaporation from surgical wounds. The operating room should be warmed greater than 24°C (i.e. 76 °F) during induction and while prepping and draping the patient. All patients become hypothermic if the room is below 21°C (i.e. 70 °F) . Once warming devices have been applied to the patient, the room temperature can be lowered to comfortable levels for the staff. Forced-air systems placed over patients are the most effective and provide both insulation and active cutaneous warming. Warming of patients via skin surface apposition is most effective intraoperatively since it is during this time that anesthetic-induced vasodilation occurs allowing heat to flow peripherally down a temperature gradient. Forced-air systems have been shown to preserve body heat and maintain normothermia, even during the longest and most invasive surgical procedures. Patient positioning is important in heat conservation. The more radially positioned the patient's extremities are the greater the heat loss. Fortunately, with most of our procedures being in the head and neck area, placing of the arms and legs medially and tucking the patients with blankets to maintain the extremities against the body will also diminish the amount of heat loss as well.

Anesthesia Circuits, Airway Heating and Humidification

Use closed, low flow semi-closed circuits to decrease evaporative losses. This provides rebreathing of gases that have been warmed and humidified by previously inhaled gases. If high flow rates are utilized use anesthetic circuit humidifiers to minimize evaporative losses from the lung. Because little heat is lost through respiration, active airway heating will only influence core body temperature minimally. However, this should still be done. If an open anesthetic circuit is used (nasal mask) minimize air flow rates and attempt to get the best mask seal possible.

Warming IV Fluids and Blood Products

Warming of fluids only will help minimize heat losses. Unfortunately, it is not possible to warm patients by administering heated fluids since these cannot be administered at temperatures above body temperatures due to the potential of protein denaturization. Warm fluids should be used to cut heat losses but they are really indicated for when large amounts of intravenous fluid replacement or blood and/or blood products are administered. A liter of fluid at room temperature will reduce the mean body temperature approximately 0.25°C.¹ Warming fluids involves either fluid warmers via the intravenous set line or with warming cabinets.

Postoperative Considerations

During the postoperative recovery period, the bodies thermal heat transfer situation is significantly different. As the anesthetic-induced peripheral vasodilation dissipates away, the thermoregulatory vasoconstriction ensues. Now, heat transfer from the periphery to the central core compartment is significantly impaired due to this vasoconstriction. Because the postoperative thermoregulatory vasoconstriction decreases peripheral-to-core heat transfer, applied warming to the skin is most effective during surgery when patients are vasodilated. Patients with regional anesthesia however warm faster than those recovering from general anesthesia. Hence, it is easier to maintain intraoperative normothermia than to re-warm patients postoperatively. Even a small decrease of 0.5°C may induce shivering postoperatively. Postoperative shivering should be treated with warming of the patient most effectively via forced-air systems and warm blankets to not only psychologically help make the patient feel better but institute physiologic measures to re-warm the patient. Intravenous administration of 12.5 - 25mg of meperidine IV or ketanserin IV can be use to treat the postoperative shivering caused by hypothermia.¹ The mechanism by which this works is by lowering the thermoregulatory threshold for shivering thermogenesis.

SUMMARY

Mild body core hypothermia is an extremely common during anesthesia and surgery. Want it or not it happens. The basic process occurs as a peripheral redistribution of warm central body heat to the extremities and peripheral tissues via anesthetic-induced vasodilation. The heat transfer occurs mostly via skin through radiation and convection. A temperature drop of about 1-2°C is common. Multiple physiologic effects of hypothermia occur which have significant potential detrimental effects on the patient's well-being. Major consequences of inadvertent hypothermia include morbid myocardial events, reduced resistance to surgical wound infection, impaired coagulation, delayed recovery, prolonged duration of drug action and postoperative thermal discomfort. Efforts to maintain intraoperative body core temperature higher than 36°C will keep patients normothermic and prevent significant complications.

Most of the beneficial effects of hypothermia do not direct apply to the dental and/or oral and maxillofacial surgery arena but were discussed anyhow to ensure full understanding of their uses. In contradistinction, most of the detrimental effects of hypothermia can and do affect our care for patients. It is because of this reason that it behooves us to ensure we not only understand but actively become involved with the management of this insidious, common problem of anesthesia. Because of the limited body surface area in which our healthcare specialty works and the fact that the majority of our procedures are not prolonged, we have been able to "dodge this problem" and not consistently confront this anesthetic complication. My intent in writing this article was to impress upon my dental colleagues who deliver anesthesia as well as the whole dental profession that we should not ignore or refuse to deal with this forlorn, common intraoperative and postoperative anesthetic side effect, but to clinically address it and treat it in order to improve the quality and safety of anesthesia delivery care.

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