HYPOTHERMIA AND TEMPERATURE REGULATION CONSIDERATIONS DURING ANESTHESIA

by Marcos Díaz, D.D.S.

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 ± 0.5□C. Normothermia can be best defined as the range of body core temperature between 36.5 - 37.5□C ± 0.5□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 temperature[1] 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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nesthetic 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

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 infants[17] 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 along 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 loose 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 mechanism 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 individuals[24] 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 PHYISOLOGIC 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