

Hypothermia with Anesthesia

Essay

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قالوا

لَسْبَحَانَكَ لَا عِلْمَ لَنَا
إِلَّا مَا عَلَّمْتَنَا إِنَّكَ أَنْتَ
الْعَلِيمُ الْعَظِيمُ

صدق الله العظيم

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LIST OF ABBREVIATIONS

ALF	Acute liver failure
ARDS	Adult respiratory distress syndrome
ASA	American Society of Anesthesiology
ATP	Adenosine triphosphate
BBB	Blood brain barrier
C	Celsius
CABG	Coronary artery bypass graft
Cm	Centimeter
CPB	Cardiopulmonary bypass
DHCA	Deep Hypothermic Circulatory Arrest
F	Fahrenheit
h	Hour
HMEs	Heat moisture exchangers
Hz	Hertz
ICH	Intracerebral hemorrhage
ICU	Intensive care unit
IV	Intravenous
Kcal	Kilocalorie
kg	Kilogram
KJ	Kilojoules

LLitter
mgMilligram
minMinute
mlMilliliter
mmHgMillimeter mercury
OLTOrthotopic liver transplantation
PGI₂Prostaglandin I₂
sSecond
μgMicrogram

INTRODUCTION

Hypothermia during anesthesia is the most common perioperative thermal disturbance (*Sessler, 2008*).

Hypothermia is defined as a core temperature $<35^{\circ}\text{C}$ and may be classified according to severity based on temperatures below this reading (*Hanania and Zimmerman, 2005*).

All general anesthetics produce a profound reduction in the core temperature triggering cold defenses including arterio-venous shunt vasoconstriction and post operative shivering (*Sessler, 2008*).

Skin temperature rises and falls with the temperature of a patient's surroundings. However, the temperature of deep body tissues, that is, the core temperature, remains relatively constant. This is due to a remarkable thermoregulatory system that is conventionally organized into three components: afferent sensing, central control, and efferent responses (*Guyton and Hall, 2006*).

Body temperature is normally tightly regulated, more than blood pressure or heart rate. The control system is complex and involves parallel positive- and negative-feedback systems. These systems are widely distributed and every part of the autonomic nervous system share in. It is not uncommon for

patients to become cold and exhibit uncontrollable episodes of shaking and shivering. These events are both troubling and perplexing to the anesthesia provider (*Diaz and Becker, 2010*).

Heat loss occurs primarily from the skin of a patient to the environment through several processes, including radiation, conduction and convection, and evaporation (*Guyton and Hall, 2006*).

Body temperature should be monitored in most patients undergoing general anesthesia exceeding 30 minutes in duration and in all patients whose surgery lasts longer than one hour. Core temperature monitoring (*e.g.*, tympanic membrane, pulmonary artery, distal esophagus, and nasopharynx) is used to monitor intraoperative hypothermia, prevent overheating, and facilitate detection of malignant hyperthermia (*Sessler, 2008*).

Hypothermia may cause significant discomfort in the awake patient. Recovery is prolonged not only because a sense of coldness alters mentation and delays awakening, but because drug metabolism is reduced (*Diaz and Becker, 2010*).

Thermoregulatory responses are impaired by general anesthesia. Intraoperative core body temperature changes are thus largely determined by patients' environments. Because the typical operating room is cold and because factors associated with surgery increase heat loss, perioperative hypothermia is common. Mild hypothermia (33-35°C) provides substantial

protection from tissue ischemia and hypoxemia (*Illievich et al., 1994*).

Surgery typically involves exposure to a cold environment, administration of unwarmed intravenous fluids, and evaporation from within surgical incisions. However, these factors alone would not usually cause hypothermia; instead, thermoregulatory defenses would normally maintain core temperature in the face of comparable environmental stress. That hypothermia is typical in unwarmed surgical patients reflects a failure of effective thermoregulatory defenses (*Sessler, 2008*).

AIM OF THE ESSAY

The aim of this essay is to focus on hypothermia with anesthesia, thermoregulation during general and regional anesthesia, and how to manage perioperative thermal changes.

انخفاض درجه حراره الجسم المصاحب للتخدير

رسالة

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NORMAL THERMOREGULATION

Thermoregulation is similar to many other physiologic control systems in that the brain uses negative and positive feedback to minimize disorders from preset normal values. Since 1912, investigators have known that animals regulate body temperature poorly when the hypothalamus is destroyed. The importance of thermal input from the skin surface was recognized in the late 1950s when it was observed that mice placed in cold environment shivered without decreasing their hypothalamic temperatures. In the early 1960s, physiologists reported active thermoregulation in response to isolated warming and cooling at sites other than hypothalamus and skin surface, including extra hypothalamic portions of the brain, deep abdominal tissues, and the spinal cord (*Guyton and Hall, 2006*).

Thus, temperature is regulated by signals derived from nearly every type of tissue including hypothalamus, spinal cord, deep core tissue and skin surface (*Sessler, 1998*).

The processing of thermoregulatory information occurs in three phases: afferent thermal sensing, central regulation and efferent responses (*Sessler, 2000*).

Afferent input:

Temperature information is obtained from thermally sensitive cells throughout the body. There are both peripheral and central detectors of temperature. Peripheral temperature receptors are located in the skin, whereas the central ones are found in deep visceral tissues (e.g., spinal cord and hypothalamus). Peripheral thermoreceptors are the first to sense a decrease in environmental temperature (*Sessler, 2000*).

Cold- sensitive cells are anatomically and physiologically distinct from those that detect warmth. Warm receptors increase their firing rates when temperature increases and cold receptors increase their firing rates when temperature decreases. Cutaneous warm receptors rarely depolarize at normal skin temperatures and probably are important only during heat stress (*Guyton and Hall, 2006*).

The hypothalamus, other parts of the brain, the spinal cord, the deep abdominal and thoracic tissues and the skin surface each contribute roughly 20% of the total thermal input to the central regulatory system. Control of autonomic response is approximately 80% determined by thermal input from the core structures; in contrast a large fraction of input controlling behavioral responses is derived from the skin surface (*Sessler, 2000*).

Cold signals travel primarily via A delta nerve fibers and warm information travels by unmyelinated C fibers, although there is some overlap. The C fibers also detect and convey pain sensation, which is why intense heat cannot be differentiated from sharp pain (*Poulos, 1978*). Most ascending thermal information traverses the spinothalamic tracts in the anterior spinal cord but no single spinal tract is critical for conveying thermal information. Consequently, the entire anterior spinal cord must be destroyed to ablate thermoregulatory responses (*Simon, 1974*).

Central regulation:

The central regulation has a set point or threshold (approximately 37°C), i.e., that temperature above or below which a physiologic thermoregulatory response is triggered. There are thresholds for cold (vasoconstriction) as well as warm responses (vasodilatation, sweating) (*Sessler, 2000*).

Temperature is regulated by central structures (primarily hypothalamus) that compare mean body temperature (which is integrated thermal inputs from skin surface, neuroaxis and deep tissues) with threshold temperatures for each thermoregulatory response. Thresholds are usually expressed in terms of core temperature. No thermoregulatory responses are initiated when core temperature is between these thresholds, these temperature

identify the interthreshold range, which, in human, is only 0.2°C (*Sessler, 2000*).

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 (*Guyton and Hall, 2006*).

How the body determines absolute threshold temperature is unknown, but the mechanism appears to be mediated by norepinephrine, dopamine, acetylcholine, prostaglandin E1 and neuropeptides. The thresholds vary daily in both sexes (circadian rhythm) and monthly in women by ~ 0.5°C. Both sweating and vasoconstriction thresholds are 0.3 - 0.5°C higher in women than in men even during the follicular phase of the monthly cycle (first 10 days). Differences are even greater during the luteal phase (*Hessemer and Brück, 1985*).

Although regulated by the hypothalamus, most thermal information is preprocessed in the spinal cord and other parts of the central nervous system. It is likely that some thermoregulatory responses can be regulated by spinal cord