Perioperative Temperature Regulation

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One of the most precise and, at the same time, complex physiological body function is the maintenance of a constant temperature varying only within narrow limits and being almost independent of external environmental conditions. Coordination and integration between peripheral and central temperature sensitivity and between heat producing as well as heat loosing processes constitute determining factors for important functions like coagulation and immunology. This paper presents the normal temperature physiology and how this is changed by general and/or regional anesthesia. Temperature monitoring and prevention of hypothermia during surgery will also be discussed.

Normal Temperature Balance

Temperature regulation is governed from the anterior parts of hypothalamus and core body temperature varies only within ±0.2 °C from the individual set point or thermoneutral zone.[1] Outside this range, compensatory mechanisms are initiated. Peripheral sensitivity for environmental temperatures is regulated via cold and warm receptors in the skin as well as in different organs of the body. Afferent cold signals are transmitted via A-delta fibers while heat is mediated via unmyelinated C-fibers. The narrow thermoneutral zone is influenced by several different factors such as age, physical condition, endocrine factors, different neurotransmittors, pyrogens and drugs.

Compensatory Mechanisms

There are very important peripheral arterio-venous shunts in the fingers and toes as well as in the nose. Via these shunts, heat loss as well as heat preservation can occur without disturbing the nutritive capillary blood flow.[2] When core body temperature increases, peripheral vessels dilate initially and thereafter sweating increases to enhance heat loss. During hypothermia, the first line of defense is peripheral vasoconstriction followed by non-shivering thermogenesis which in adults mainly is mediated via thyroid heat production. In neonates and infants less than 6 months of age non-shivering thermogenesis occurs in brown adipose tissue.[3] This is a highly specialized tissue with a rich sympathetic innervation and a high blood flow. Via an unique mitochondrial mechanism, that uncouples ATP production and enhances oxygen consumption, heat production is increased: a heat producing mechanism that neonates and infants have in common with hibernating animals. The next compensatory mechanism against hypothermia, after vasoconstriction and non-shivering thermogenesis, is skeletal muscle shivering, a most uncomfortable and painful condition. In addition, shivering is also known to be inefficient as far as heat production is concerned and increases the workload on the cardiovascular system.[4,5]

Heat Production and Metabolism

In order to maintain a constant core body temperature a certain surplus of heat must be produced. Energy expenditure must therefore overcome, by about 10 %, the basal metabolic rate. Normally, this is achieved by additional food intake and hence by nutrient-induced thermogenesis. The detailed knowledge of how and where in the body, nutrients stimulate heat production is not completely delineated. Heat production is most effective from amino acids and proteins that have a thermic effect of 30-40 %. Corresponding values for carbohydrates and fat are 6-9 % and 0-2 % respectively.[6] Hence, from a thermogenetic point of view, fat and lipids are relatively unimportant.

Temperature Regulation During Anesthesia

General Anesthesia

Temperature balance is disturbed during general anesthesia. The thermoneutral zone is increased tenfold. Compensatory mechanisms that set in after 0.2 °C of hypothermia in the awake state are not activated during general anesthesia until the core body temperature has decreased by 2-2.5 °C.[7] This is because anesthetic agents decrease metabolism and energy expenditure, i.e. heat production. This drug effect is further enhanced by the parallel peripheral vasodilatation during general anesthesia resulting in a redistribution of heat.

Intravenous as well as inhaled anesthetic agents therefore result in decreased heat production, increased heat loss and decreasing core body temperature. The non-shivering thermogenesis from brown adipose tissue is reduced in

parallel with the decreased energy expenditure caused by anesthetic agents.[8,9] As in the awake state, the first compensatory mechanism against a pronounced, uncontrolled, temperature drop is vasoconstriction. This function is dose-related and sets in, at ordinary anesthetic levels for surgical procedures, when the core temperature has decreased by 2 to 2.5 °C. All anesthetic agents also reduce shivering from skeletal muscle, a compensatory mechanism that is completely eliminated by muscle relaxation. There are, however, recent findings suggesting that skeletal muscles have a capacity for non-shivering thermogenesis. An uncoupling protein has been detected.[10,11] This means that possibilities for non-shivering thermogenesis exist also in skeletal muscle.

Regional Anesthesia

Central regional blocks, i.e. spinal and epidural anesthesia, change afferent as well as efferent nerve impulses as well as those regulating core body temperature. During central blocks, the thermoneutral zone is widened 3-4 times from the ±0.2 ºC in the un-blocked state to 0.6-0.8 °C.[7] Most probably, this is caused by an incorrect peripheral afferent information in parallel with a sympathetic blockade that results in vasodilatation and heat loss. Vasoconstriction and shivering are both inhibited in the blocked region.

When a central regional block is combined with general anesthesia, hypothermia is greater than during general anesthesia alone. Hence, the effect of the two anesthetic techniques is additive. It takes longer, postoperatively, to regain normal core body temperature after combined general and central regional anesthetic technique. Therefore, it is of the greatest importance that body temperature is monitored carefully so that hypothermia can be adequately prevented.

Risk Groups

It is patients at the extremes of age, i.e. neonates and elderly, who are most susceptible for temperature drops during anesthesia. Neonates have relatively large body surface areas in relation to body weight and with well perfused and thin skin they are, at least during the neonatal period, not very well isolated by subcutaneous fat. The neonate rapidly develops hypothermia. It is true that neonates have special heat producing tissue, brown fat, but this heat production is inhibited by anesthetic agents.[8,9] It is, in particular, premature neonates that have the highest risks for developing hypothermia and it is of the utmost importance that these babies are treated by an uninterrupted temperature discipline at all times during their flow through the surgical process.

In the elderly, there is less muscle mass and decreased muscle tone that results in a decreased heat production compared with younger individuals. Also, there is reduced capacity for peripheral vasoconstriction in the elderly. Furthermore, if shivering occurs they have a reduced cardiovascular tolerance for the relatively ineffective enhancement of oxygen consumption. In elderly patients, as in infants, careful temperature monitoring is important and, often, external heat must be provided during general as well as during regional anesthesia.

There are other patient groups that are susceptible to heat loss; in particular, those with burn injuries, hypothyroidism and cortical adrenal insufficiency.

Effects of Hypothermia

Hypothermia results in a reduced oxygen consumption (energy expenditure) which decreases by 7-8 % per °C. There are several indications for intraoperative hypothermia in neuro- as well as in cardiovascular surgery. This kind of deliberate hypothermia is not commented upon further in this article.

Anesthesia-induced hypothermia influences the function of most organ systems. Some effects of hypothermia are listed below:

- increased infection susceptibility
- disturbed blood coagulation
- delayed drug metabolism.

At a moderate degree of hypothermia leukocyte mobility and phagocytosing capacity is changed which weakens the line of defense against wound infections.[12] Reduced capillary blood flow, due to vasoconstriction, adds to this.

Increased bleeding and coagulation times in connection with postoperative hypothermia are not only caused by reduced activities of various coagulation factors but also by pathological thrombocyte aggregation and increased fibrinolysis.[13] Not only is there a higher incidence of disturbed coagulation in connection with hypothermia resulting in an increased need for blood transfusions,[14] but increased bleeding may also result in more frequently infected hematomas in the postoperative period.

It is also important to be aware of the longer duration of drug effects due to hypothermia. This is particularly important for potent analgesic agents and for neuromuscular blocking agents. It is well known that a train-of-four-ratio (TOF) of 0.7 in the postoperative period results in increased respiratory complications particularly in elderly patients subjected to intraabdominal surgery. This effect could be due to the lowered hypoxic ventilatory response caused by a small dose of non-depolarizing muscle relaxants.[15] However, it is more likely to be due to pharyngeal incoordination also caused by subparalytical doses (TOF 0.70) of non-depolarising muscle relaxants as recently shown by Eriksson et al.[16] Hence, complete reversal of non-depolarising muscle relaxation must be achieved before sending patients to the postoperative recovery unit, especially in hypothermic patients.

Shivering

For a long time, postoperative shivering was regarded as

almost normal and was noted to occur in 40 % of patients. The rate is now much lower due to improved hypothermia prevention. Most patients shiver at a core body temperature of 34°C and a third of patients have shivering at a body temperature of 36 °C. Shivering can cause complications such as wound rupture, increased postoperative bleeding, increased intracranial and intraocular pressures etc. Whenever shivering is noted it must be treated urgently. Meperidine as well as clonidine are good choices in addition to external heat.

Temperature Monitoring

There are several ways to monitor temperatures during anesthesia and surgery. It is preferable to measure two temperatures, one central and one peripheral. It is also important to know the temperature in the operating room to assess the actual temperature stress for the patient. You may choose to measure the central temperature rectally (at 10 cm) or in the lower third of the esophagus. Urinary bladder temperature is easily monitored using thermistor equipped urinary catheters. Ear temperatures can be complicated by effects on the ear drum. This site also has greater variability than other sites most likely because the temperature sensor is difficult to position correctly. Peripheral body temperature is best measured distally on the hand or the foot. When reliable measurements of central and peripheral body temperatures are used, this supplies not only information of the patient´s temperature balance but also of the peripheral circulation which is reflected by the difference between central and peripheral temperatures.

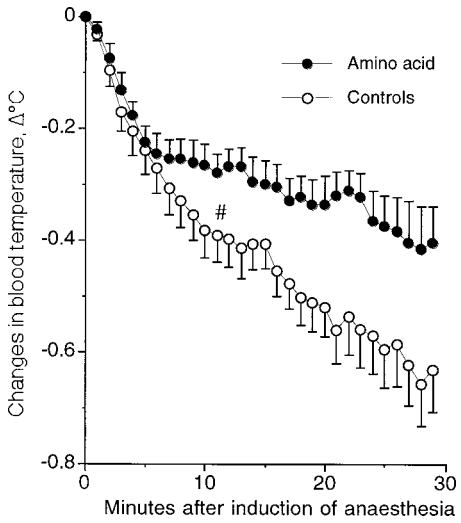

Figure 1: Changes in mixed venous blood temperature from baseline during the first 30 min of anesthesia. Vertical bars indicate SE of mean changes.
# $P<0.05$, statistically significant difference between the groups,occurring after 12 min.

Prevention of Hypothermia

During anesthesia, heat is lost via radiation, convection, conduction and evaporation. Quantitatively, the most important loss is from radiation which makes up for about 65 % of the total external heat loss. Next is convection that can measure up to 25 % of the total heat loss. Conduction influences heat loss only to a minor degree in the adult but could have a greater influence in infants and children. Evaporation, i.e. loss of high energy molecules when fluid is converted to gas, occurs both from open surfaces at the operating site and from the airways. During major surgical procedures, the high flux of air in the operating room together with cold inspired air result in increased heat loss via evaporation. Evaporation from the airways is reduced during low flows and when heat-moister exchange devices are used, particularly during pediatric anesthesia. Preterm and term newborns must always be transported to the operating room in an incubator that ought to be used for as long as possible during preparation for surgery. It is important, in all patients, to make sure that the utmost is done so that they do not loose heat unnecessarily during their journey from the ward, transport to surgery, during surgery and postoperatively. This means that the operating room temperature be set so that it is comfortable for the patient. Also, all fluids that are used during the procedure should be warmed.

In the awake subject, intravenous amino acids raise blood temperature to supranormal levels. An intravenous amino acid infusion in awake tetraplegic patients results in somewhat enhanced thermogenesis as compared with in normal awake subjects.[17] Recently, it has been discovered that infusion of ordinary amino acid solutions before and during anesthesia and surgery are able to diminish the temperature reduction during anesthesia (Fig. 1). The inhibition of the decrease in whole body heat content by the infusion of amino acids (Fig. 2) was equivalent to a five-fold enhanced thermogenesis compared with awake volunteers.[18] The enhanced amino acid induced oxidative metabolism and heat production found during anesthesia might, in analogy with the increased amino acid induced thermogenesis in tetraplegic patients with interrupted nerve signals, support the existence of a central inhibitory temperature regulating mechanism that is depressed by anesthesia. Amino acid induced thermogenesis during anesthesia did not take place in the splanchnic region, but was most likely in skeletal muscle. The question is - which mechanisms are involved? Certainly, the recently demonstrated existence of the uncoupling protein in skeletal muscle invites speculation that non-shivering thermogenesis from skeletal muscle is responsible for the increased amino acid induced heat production during anesthesia.

Perioperative Hypothermia and Outcome

Even slight hypothermia during anesthesia and surgery increases the incidence of postoperative wound infection,

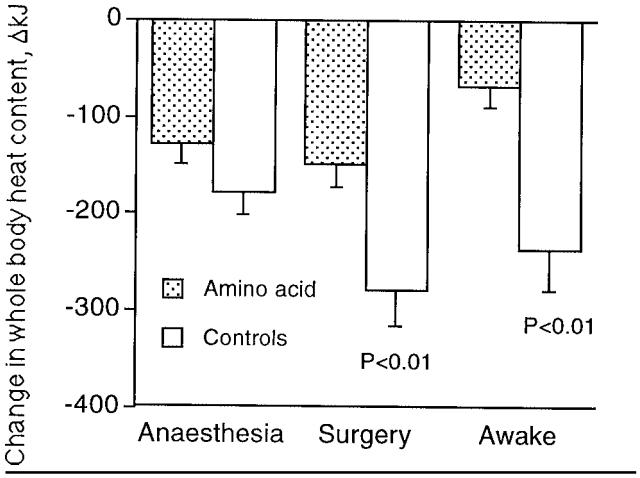

Figure 2: Changes in whole body heat content from baseline during anesthesia, surgery and at awakening. Vertical bars are SE of mean changes.
$P<0.01$, statistically significant differences between the groups.

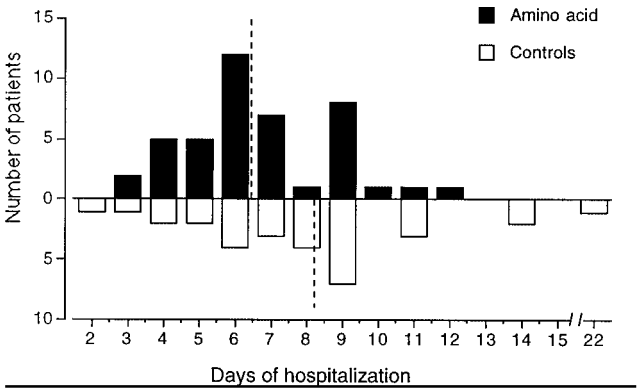

Figure 3: Days of hospitalization after abdominal surgery in 45 patients, receiving *iv* amino acids, and in 30 control patients. Dotted lines indicate mean values in each group.

results in enhanced bleeding and prolonged effects of potent anesthetics and muscle relaxants. Cardiac morbidity is increased in patients who are hypothermic during anesthesia and surgery.[19] It has also been shown that the duration of hospitalization is 2-3 days shorter when normothermia is maintained during anesthesia and surgery (Fig 3).[12,20]

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