CONTROL OF PERI-OPERATIVE SHIVERING

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In homeothermic species a thermoregulatory system co-ordinates defenses against cold and heat to maintain internal body temperature within a narrow range, thus optimizing normal physiologic and metabolic function. The combination of anaesthetic induced thermoregulatory impairment and exposure to cool environment makes most unwarmed surgical patients hypothermic. Although shivering is but one consequence of peri-operative hypothermia and rarely the most serious it occurs frequently (40-60% after volatile anaesthetics) and it remains poorly understood. While cold induced thermoregulatory shivering remains an obvious etiology, the phenomenon has also been attributed to numerous other causes.

THERMOREGULATION AND ANAESTHESIA:

Core body temperature is normally maintained within the narrow range of 36.7 -37.1 degrees C, even in the presence of widely varying environmental temperatures (-10 to 50 degree C).

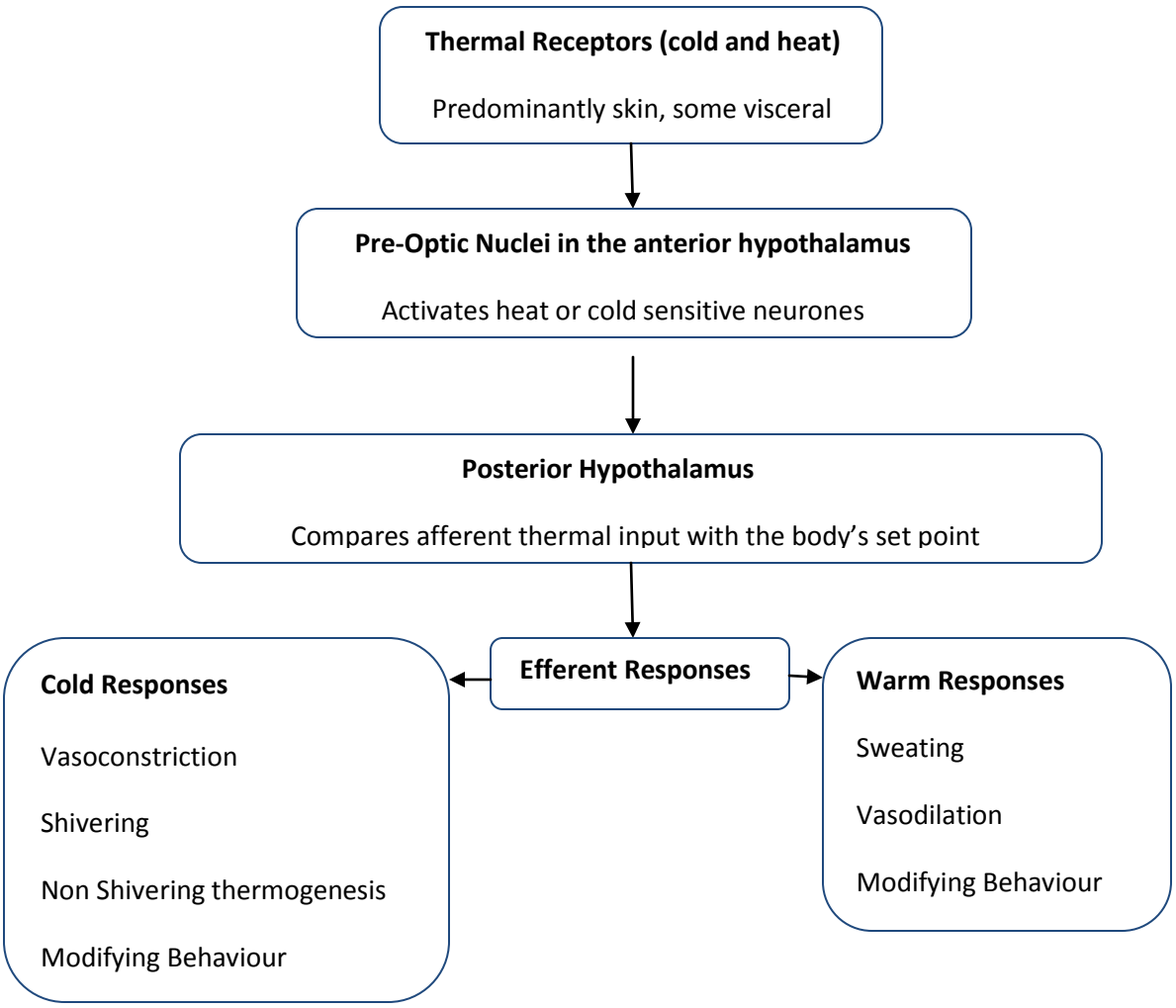
HEAT BALANCE:

Heat is lost from the body by four processes – convection, radiation, conduction and evaporation. Heat loss by radiation is related to the temperature difference between the patient and the ambient environment. Convective heat loss occurs when the layer of body next to skin moves or is disturbed, thereby removing its insulating properties. Classical physiological teaching is that the proportion of total heat loss caused by each mechanism is: radiation-40%, convection- 30%, Conduction-5%, evaporation-25%, but these proportions may change markedly for patients anaesthetized in the theatre environment.

PHYSIOLOGY:

Thermoregulation is achieved by a physiological control system consisting of peripheral and central thermo receptors, an integrating control centre and efferent response systems. Afferent thermal input comes from anatomically distinct cold and heat receptors, located predominantly in the skin, but also centrally. The central control mechanism situated in the hypothalamus determines mean body temperature by integrating the signals from peripheral and core structures and comparing the mean body temperature with a predetermined 'set point' temperature. In humans the efferent responses to effect change in body may be classified as behavioral and autonomic.

Autonomic mechanisms involve control of cutaneous vascular smooth muscle tone, shivering and non shivering thermogenesis and sweating.



Afferent Thermal Signals:

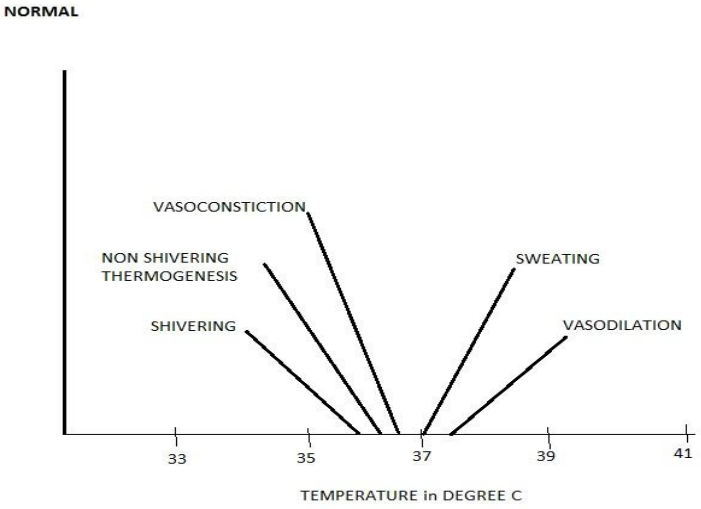
The afferent thermal input may be central or peripheral. Thermo sensitive receptors located in the skin and mucous membranes mediate thermal sensation and contribute to thermoregulation reflexes. Cold specific receptors are innervated by A-delta fibers. Heat receptors are innervated by C fibers. Cold receptors in the skin outnumber heat receptors 10 fold and are the major mechanism by which the body protects itself from cold temperatures. Afferent input from these cold receptors in the skin is transmitted to the posterior hypothalamus. In addition to the peripheral cold receptors, there are central cold receptors in the hypothalamus, although its effects are masked by the predominant peripheral influence.

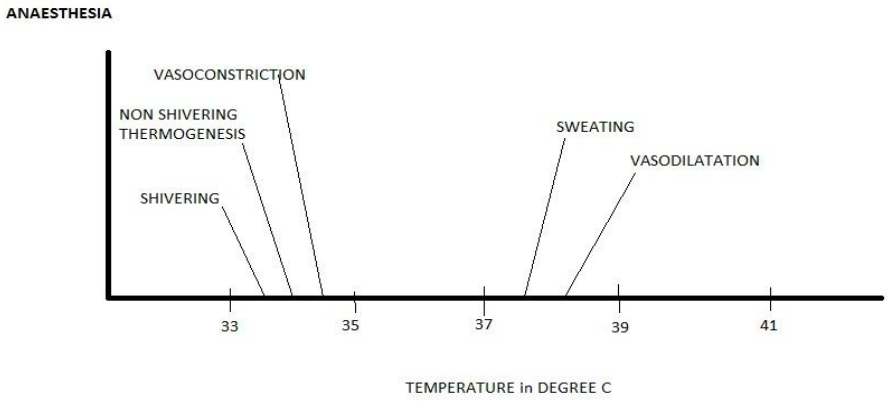
Central Integration: The Hypothalamus:

The afferent mechanisms provide feedback to temperature regulating centres in the hypothalamus. The pre-optic are of the hypothalamus contains temperature sensitive and temperature insensitive neurons. The temperature sensitive neurons which predominate by 4:1 , increase their discharge rate in response to increased local heat and this activates heat loss mechanisms. Conversely cold sensitive neurons increase their rate of discharge in response to cooling. Neurones sensitive to local thermal stimuli also

exist in the posterior hypothalamus, reticular formation, medulla and spinal cord. Detection of cold differs from detection of heat, in that the principal mechanism of detection of cold is input from cutaneous cold receptors.

The posterior hypothalamus integrates cold afferent signals from the periphery with heat sensitive stimulation from the pre-optic area of the hypothalamus and instigates the effector responses. The set point or physiological 'thermostat' of the thermoregulatory system is the temperature at which the system requires zero action to maintain that temperature (36.7-37.1 degree C) . The limits of this range represent the threshold at which cold or heat responses are instigated, and hence it has been termed the 'interthreshold range'. Normally, it is no more than 0.4 C, but is increased to 4.0 degree C during GA.





Effector Responses:

The thermoregulatory responses are characterized by altered behavior (quantitatively the most effective mechanism), a vasomotor response (consisting of vasoconstriction and pilo-erection or vasodilatation and sweating) and shivering with increased basal metabolic rate. In the conscious individual, behavioral modification is more powerful than the autonomic mechanisms. When the hypothalamic thermostat indicates an excessively cool body temperature, impulses pass from the hypothalamus to the cerebral cortex to give the individual the sensation of feeling cold. The result is modified behavior, such as increased motor activity, moving to warmer surroundings or adding additional clothing. The central behavioral response to cold is based largely on cutaneous thermal signals. When the set point temperature range has been breached, autonomic effector responses are activated.

Shivering and Non-Shivering Thermogenesis:

Adjacent to the centre in the posterior hypothalamus on which the impulses from the cold receptors impinge, there is a motor centre for shivering. It is normally inhibited by impulses from the heat sensitive areas in the anterior hypothalamus. But when cold impulses exceed a certain rate, the motor centre for shivering becomes activated by 'spill over' of signals and it sends impulses bilaterally into the spinal cord. Initially, this increases the tone of skeletal muscles throughout the body. But when this muscle tone increases above a specific level, shivering is observed.

Two patterns of muscular activity, seen in electromyographic studies, contribute to the phenomenon of post anaesthetic shivering; first, atonic pattern (4-8 cycles/min) characteristic of the response to hypothermia in awake patients, is observer, and then a phasic (6-7 Hz) pattern resembling clonus.

The elicitation of non-shivering thermogenesis is an important mechanism in increasing heat production, particularly in neonates. Non-shivering thermogenesis occurs mainly in brown adipose tissue(BAT). This subtype of adipose tissue contains large number of mitochondria in its cells and these are supplied by an extensive SNS innervations. When sympathetic stimulation occurs, oxidative metabolism of the mitochondria is stimulated. However, it is uncoupled to phosphorylation, so that heat is produced instead of generating ATP. In adults, the amount of BAT is small, and non-shivering thermogenesis increases the rate of heat production by less than 10-15%. In infants, it may double heat production.

Measurement of Temperature:

Core temperature may be evaluated by an infra red thermometer at the tympanic membrane and by thermistors positioned in the distal oesophagus , nasopharynx, or pulmonary artery. Skin surface varies with ambient temperature and induction of anaesthesia and is usually also measured with a thermistor, or alternatively with a liquid crystal thermometer. Measurement of the temperature at the skin surface has also been used to estimate the core temperature by heating the sensor to eliminate the core surface temperature gradient.

Effect of General Anaesthesia on Thermoregulation:

General anaesthesia causes thermoreguatory impairment characterized by an increase in heat response threshold and a decrease in cold response threshold, such as the normal inter threshold response range (between which no effector response occurs) is increased from approximately).4-4 degree C. Both heat response and cold response thresholds are affected. All general anaesthetic agents impair thermoregulatory responses to a similar, but not identical, extent. All the currently used volatile agents decrease the vasoconstriction and shivering thresholds. Mild hypothermia during general follows a distinctive pattern and occurs in three phases: first, an initial rapid decrease in core temperature of approximately 1 degree C over the first hour; second, a slower linear decrease to 34-35 degree C; and third, a core temperature plateau (or thermal equilibrium) is then reached, where heat lost to the periphery equals heat gained from core metabolic heat production.

The initial rapid reduction in core temperature is greater than that which would be explained by a lowering of metabolic rate and heat loss, and is in fact attributable to core to peripheral redistribution of body heat. Mean body temperature and body heat content remains constant during tis first hour. The subsequent core temperature plateau largely results from thermoregulatory vasoconstriction, triggered by a core temperature of 33-35 degree C.

Effect of regional Anaesthesia on Thermoregulation:

Both epidural and spinal anaesthesia decrease, to a similar extent, vasoconstriction and shivering thresholds, but not as much as GA. Because local anaesthetics administered into the sub-arachnoid or epidural space do not obviously interact with the hypothalamic control centres. However, it is inconsistent with the thermoregulatory impairment caused by the effects of the regional block on afferent thermal information.

In contrast with general anaesthesia, where the heat produced by shivering is unchanged, heat produced by epidural anaesthesia is reduced by approximately by 60%. This occurs because shivering above the upper limit of block does not compensate for the inability of muscles below the block to engage in shivering. As with general anaesthesia, core hypothermia (by 0.6-1.5 degree C) occurs during the first hour or so after epidural anaesthesia because of core to peripheral redistribution of body heat from the epidural induced vasodilation. However with prolonged epidural anaesthesia, the degree of core hypothermia is less than after GA, because of vasoconstriction above the level of block. Shivering during regional anaesthesia, in common with that of GA, is usually preceded by core hypothermia and vasoconstriction above the level of block.

After the core to peripheral redistribution of body heat, the degree of subsequent hypothermia depends on the balance of cutaneous heat loss and the rate of metabolic heat production. During epidural anaesthesia, heat loss may be accelerated by reduced vasoconstriction caused by the block. Hence, heat loss continues unawaited during epidural anaestheia, despite the activation of the effector mechanisms, above the level of block. This is seen especially where GA and Epidural are combined.

Consequences of Peri-Operative mild hypothermia:

In particular circumstances hypothermia may have a protective effect in terms of reducing basal metabolic rate. The use of moderate hypothermia is routine practice in many centres during cardio pulmonary bypass. It is generally agreed, however, that the deleterious consequences of mild hypothermia outweigh the potential benefits, with evidence emerging that hypothermia per se is responsible for adverse post-operative outcomes; in particulat hypothermic patients are more likely to have post-operative wound infection than normal patients. The initial 3-4 hrs after bacterial contamination is thought to be crucial in determining if clinical infection ensues. In vitro studies suggest that platelet function and coagulation are impaired by hypothermia, and mildly hypothermic patients lose > 25% more blood in the peri-operative period than do normothermic patients. In addition, peri-operative thermal discomfort is often remembered by patients as the worst aspect of their peri-operative experience.

CONSEQUENCES OF PERI-OPERATIVE MILD HYPOTHERMIA

- Increased wound infection
- Increased surgical bleeding
- Increased incidence of myocardial infarction and malignant arrhythmias
- Delayed recovery from anaesthesia
- Excessive SNS stimulation on waking
- Prolonged drug metabolism
- Negative nitrogen balance
- Impaired immune function
- Patient discomfort

Post Anesthetic Shivering:

Post anaesthetic shivering affects 5-65% of patients after general anaesthesia and 33% during epidural regional anaesthesia. It is usually defined as readily detectable fasciculation or tremor of face, jaw, head, trunk or extremities lasting longer than 15s. Apart from the obvious discomfort, post anaesthetic shivering, in common with hypothermia is associate with a number of potential delitrious sequelae. Post anaesthetic shivering is usually preceded by core hypothermia and vasoconstriction.

While hypothermia is one factor in the etiology of the post anaesthetic shivering, not all patients who shiver or hypothermic . Studies on post-operative patients have indicated that male gender, age (16-60 yrs) and anti-cholinergic premedication are risk factor for the post anaesthetic shivering, while the intra-operative use of Pethidine virtually abolished it. The use of Propofol reduces the incidence of post-operative shivering compared with Thiopental.

CONSEQUENCES OF POST ANAESTHETIC SHIVERING
❖ Increased O2 consumption and CO2 production ❖ Catecholamine release and SNS stimulation ❖ Increased cardiac output , heart rate, BP ❖ Increased intra-ocular pressure ❖ Decreased SvO2 ❖ Lactic Acidosis ❖ Interference with monitoring ❖ Patient Discomfort

Physical, active and passive strategies for avoiding peri-operative hypothermia:

Preventing redistribution included hypothermia may be achieved by physical and pharmacological means. Redistribution of heat results when anaesthetic induced vasodilation allows heat to flow from the core to periphery down its concentration gradient. Pre-emptive skin surface warming does not increase core temperature but increases the body heat content, particularly in the legs and removes the gradient of heat loss via the skin. This approach s rarely used in clinical practice, however, because it requires one hour of prewarming.

Passive insulation, including cotton drapes has been used pre-operative to reduce heat loss to the environment. Because only 10% of metabolic heat production is lost on heating and humidifying inspired gases, this method is relatively inefficient in maintaining normothermia. Heat and moisture exchange filters retain significant amount of moisture and heat within the respiratory system, but are only 50% as effective as active mechanism. Ambient temperature determines the rate of heat loss by radiation and convection and maintains normothermia if close to initial, pre-induction, core temperature (36 degree C). However, this is usually impractical, as operating room staff usually find this temperature uncomfortable. Water mattresses are demonstrably ineffective at preventing heat loss, possibly because relatively little heat is lost from the back. Moreover decreased local tissue perfusion associated with local temperature of 40 degree C may lead to skin necrosis. Conductive loss may be reduced if intra-venous fluids are warmed before or during administration.

Forced air warming systems are undoubtedly the best way to maintain normothermia during long procedures and are particularly effective when used intra-operatively for vasodilated patients, allowing heat applied peripherally to be rapidly transferred to the core. Their use increases core temperature and reduces the incidence of post anaesthetic shivering.

STRATEGIES FOR PREVENTION AND TREATMENT OF PERI-OPERATIVE HYPOTHERMIA AND POST ANAESTHETIC SHIVERING
PREVENTION: ❖ Intra-operative use of forced air warming device ❖ Reflective ‘Space’ blankets ❖ Heating and humidifying inspired gases ❖ Increased ambient temperature ❖ Warmed i.v. fluids

TREATMENT:

- Pethidine 0.33 mg/kg i.v. or epidural (and other opiods to a lesser extent)
- Doxapram 1.5 mg/kg
- Clonidine 2 µg/kg
- Methylphenidate 0.1 mg/kg
- Physostigmine 0.04 mg/kg
- Ondansetron 0.1 mg/kg

Treatment of Post Operative Shivering:

Post operative shivering should not be treated in isolation from pre-operative hypothermia. Not all patients to shiver are hypothermic, but most are and successful treatment of shivering in these patients without concomitant mananagement of hypothermia may result in deepening hypothermia. However, the mainstay of symptomatic treatment of post-operative shivering is pharmacological.

A wide range of drugs is effective and it would be surprising if all worked on a single part of the thermo regulatory mechanism. Pethedine is remarkably effective in treating post-operative shivering, 25 mg being sufficient in the majority of adults. This is evidence suggesting this may be result of an action at the κ- opioid receptors.

One hypothesis for the mechanism of post anesthetic shivering is that because the brain recovers later than the spinal cord, uninhibited spinal clonic tremor occurs, resulting in shivering. Consistent with this hypothesis, Doxapram (a cerebral stimulant) has also shown to be an effective treatment. This is not as effective as Pethidine. Various drugs, mechanisms of action of which are unclear, are also effective. Physostigmine prevents the onset of post anaesthetic shivering, implying the cholinergic pathways are involved in the thermoregulatory mechanism that leads to shivering. Clonidine, an α2- adrenergic agonist and Ondansetron , a serotoninergic antagonist are also effective.