Temperature Management in Critically ill Patients

Essay

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Introduction

Critically ill patients are defined as those patients who are at high risk for actual or potential life-threatening health problems. The more critically ill the patient is the more likely he or she is to be highly vulnerable, unstable and complex, thereby requiring intense and vigilant nursing care ***(Hines et al, 2010)***.

Thermoregulation is a dynamic part of homeostasis controlled by the anterior hypothalamus. Temperature in mammals will vary diurnally within a narrow range and temperatures outside this can have a profound effect at both a biological and clinical level. Biochemical sequelae can include acid–base disturbance, electrolyte derangement, fluid shifts, alteration of intra- and extracellular enzymatic function, and variations in energy production and consumption. Clinical manifestations can also vary widely with potential neurological, cardiorespiratory, metabolic and infective compromise ***(Matthew Faulds and Tim Meekings, 2013).***

In the critically ill population, thermoregulation is often disrupted. This may be inadvertent (e.g.

hypothermia in a trauma patient), part of a disease process such as fever or for therapeutic benefit, such as induced hypothermia after cardiac arrest. The close monitoring, manipulation, and regulation of body temperature forms an important part of the care of the critically ill patient ***(Matthew Faulds and Tim Meekings, 2013).***

Older adults (≥ 65 years) and persons with chronic diseases are at risk of heat and cold related mortality and morbidity during extreme ambient temperatures. Even slight changes in temperature can adversely affect these populations because of their weakened physiological adaptability and socioeconomic factors ***(Rebecca S. Noe, et al, 2012).***

Due to its protective effect on the brain and the myocardium, hypothermia therapy (HT) has been extensively studied in cardiac arrest patients with coma as well as in patients presenting with acute myocardial infarction (MI) ***(Cédric Delhaye et al, 2012).***

Therapeutic hypothermia (TH) aims to improve the outcome by reducing metabolic oxygen demands. In the operating theatre, TH can be instituted for neuroprotection before primary

insult. Trials looking at TH in traumatic brain injury and ischaemic stroke have produced equivocal results to date and consequently use in these conditions is not recommended at present ***(Matthew Faulds and Tim Meekings, 2013).***

Patients surviving out-of-hospital cardiac arrest have shown better survival if treated with therapeutic hypothermia, and initial data now suggest that most of the patients who do survive after hypothermia have preserved cognitive function and are able to return to work. The use of hypothermia has improved the survival rate for resuscitated cardiac arrest patients from about 1 in 6 to around 1 in 2, and it has now become the standard of care for patients with resuscitated cardiac arrest, some had cognitive impairment, but that has always been the case after resuscitated cardiac arrest. That percentage does not appear to be higher after hypothermia ***(Sue Hughes, 2013).***

Temperature is not only an important clinical marker of severity of illness but also an independent predictor of morbidity and mortality in critically ill patients. Close monitoring and regulation to avoid extremes of body temperature is particularly important in the critically ill patient.

This will prevent the uncontrolled disruption of homeostasis and associated subsequent organ dysfunction and failure ***(Matthew Faulds and Tim Meekings, 2013).***

Aim of the work

The aim of this essay is to discuss temperature regulation in critically ill patients and its management .

Pathophysiology of temperature regulation

Human body is able to regulate internal body temperature within a narrow range near 37°C, despite wide variations in environmental temperature. The range of temperatures that living cells and tissues can tolerate without harm extends from just above freezing to nearly 45°c ***(Witzmann, 2009)***.

In a healthy individual, body temperature is kept constant in a very small range between 36.5°C and 37.5°C "thermo neutral zone". Very perfect regulation of body temperature, necessary for optimal progress of enzymatic reactions, is developed in all homoiothermic animals. It doesn't apply to poikilothermic animals ***(Epstein and Anna, 2006)***.

Normal thermoregulation involves a dynamic balance between heat production and control of heat loss, with the aim of providing a constant core temperature. This is achieved in part by adjustment of central thermo genesis, and in part by maintaining a differential temperature gradient between the body core temperature and the peripheries directly exposed to the environment, the amount of heat gained from or lost to the environment is closely and rapidly regulated in response to changing circumstances ***(Macario and Dexter, 2003)***.

Tissue temperature is important for two reasons:

First, temperature extremes injure tissue directly. High temperatures alter the three-dimensional structure of protein molecules, even though the sequence of amino acids is unchanged. Such alteration of protein structure is called denaturation. Denaturation inactivates

a cell's proteins and injures or kills the cell. Injury occurs at tissue temperatures higher than about 45°C, which is also the point at which heating the skin becomes painful. The severity of injury depends on the temperature to which the tissue is heated and its duration ***(Rodney and David, 2012)***.

Cold also can injure tissues. As a water-based solution freezes, ice crystals consisting of pure water form, so that all dissolved substances in the solution are left in the unfrozen liquid. Therefore, as more ice forms, the remaining liquid becomes more and more concentrated. Freezing damages cells as ice crystals mechanically injure the cell. The increase in solute concentration of the cytoplasm as ice forms denatures the proteins by removing their water of hydration, increasing the ionic strength of the cytoplasm and causing other changes in the physicochemical environment in the cytoplasm ***(Rodney and David, 2012)***.

Second, temperature changes profoundly alter biological function through specific effects on such specialized functions as electrochemical properties and fluidity of cell membranes and through a general effect on most chemical reaction rates. Increasing temperature by 10°C increases the reaction rate by a factor of two to three. For any particular reaction, the ratio of the rates at two temperatures 10°C apart is called the Q_{10} for that reaction, and the effect of temperature on reaction rate is called the Q_{10} effect. The Q_{10} effect is clinically important in managing patients who have high fevers and are receiving fluid and nutrition intravenously. A commonly used rule is that a patient's fluid and calorie needs are increased 13% above normal for each 1°C of fever ***(Rodney and David, 2012)***.

In healthy individuals, the body temperature (oral temperature) is somewhere between 36°c and 37°c. It slightly increases during the day since the morning (from 6:00 a.m.),the peak reached at 6:00 to 10:00 p.m. the lowest temperature is between 2:00 and 4:00 a.m. Diurnal variations depend on the activity throughout the day. Diurnal variations don't change in persons that work at night and sleep during the day. Such a diurnal variation is also kept when fever occurs. Fever reaches the peak in the evening, and in the morning even a very sick patient may have almost normal temperature ***(Guyton, 2006)***.

Body temperature changes are more intensive in young person than in old people. The temperature may slightly or temporarily increase in hot environment. Physical activity may also increase the body temperature. In extreme effort, the increase may be very high. The temperature may increase slightly if vasodilatation, Hyperventilation and other compensation mechanisms fail. Small increase in temperature may occur if the surrounding temperature is lower or the jogging is done early in the morning ***(Guyton, 2006)***.

Central thermoregulatory ability can be impaired in such situations as stroke, CNS trauma, infection, tumor, or hemorrhage and in uremia. Impaired control of peripheral vasculature through autonomic dysfunction can also play a part in diabetes. Reduced heat production occurs in endocrinopathies such as hypothyroidism, hypoadrenalism and hypopitutarism. Also some pharmacological agents can cause central thermoregulatory failure for example: barbiturates, opioids, tricyclicantidepressants, and benzodiazepine ***(Kelly et al, 2006)***.

Body temperature is the result of a balance between heat production and heat loss. Heat is a product of the body's natural metabolic processes, but as it is produced, it is also lost to the environment. Regulation of body temperature occurs through a negative feedback system in the central nervous system, primarily the hypothalamus. As a result, body temperature is maintained within normal range, ensuring a constant rate of metabolism, enhanced nervous system conduction, and optimal skeletal muscle contraction ***(Kelly et al, 2006)***.

Basic concepts

Core temperature

The body core consists of the essential organs: brain, heart, lungs, liver and kidneys. These organs are maintained at an almost uniform temperature. Core temperature is measured by temperature sensitive neurons in the great veins, spinal cord, abdominal viscera and the hypothalamus. Peripheral thermoreceptors (free nerve endings) are found mainly on the body surface; there are ten times more cold-sensitive nerve endings than heat-sensitive nerve endings ***(Dan et al, 2011)***.

Normal temperature

Normal body temperature is 37°C (±0.5°C). There is a diurnal variation of core temperature within individuals of a similar magnitude; in women, there is also a monthly variation because core temperature rises around the time of ovulation. Temperature rises with increasing physical activity ***(Dan et al, 2011).***

Integration of thermal information

The many neural, environmental and hormonal signals involved in temperature regulation are integrated in the pre-optic anterior hypothalamus. The hypothalamus has a 'set point' of acceptable core body temperature ('thermoneutral zone') and deviation from this setpoint by ±0.2°C initiates reflex activity to gain or lose heat by a variety of negative feedback-controlled methods **(*Rodney and David, 2012*).**

Physiology of temperature regulation

Heat Dissipation

Overheating occurs more often in the adult than in the neonate because of fever, exercise, and exposure to excessive temperatures, but the infant has a more limited ability to compensate for heat stress. The body responds to overheating by vasodilatation in the skin, which increases heat loss from the blood. Vasodilatation happens when the anterior hypothalamus senses an increase in temperature, whether it is central or peripheral, and inhibits the posterior hypothalamus from sending sympathetic impulses that cause vasoconstriction ***(Leslie, 2012).***

At certain core body temperatures, which may vary slightly among individuals, the anterior hypothalamus will send signals through the autonomic nervous system and release acetylcholine to the sweat glands in the skin to produce sweat. Sweating causes an evaporative heat loss, and the adult is able to remove 10 times the basal rate of heat production ***(Guyton & Hall, 2006)***.

The neonate, when exposed to excessive temperatures, may vasodilate but has a limited ability to sweat. In fact, the individual sweat glands of the infant are capable of only one third of the response of the adult sweat glands ***(Hey et al, 1999)***.

The infant born at less than 32 weeks gestation has no ability to sweat, but this ability matures rapidly after birth. The sweat glands in the forehead are the most mature and may be the only visible signs of sweat ***(Baumgart, 1996).***

A third mechanism of heat dissipation occurs when there is a change in body position that increases the amount of skin surface area exposed to the environment so heat can be disseminated by convective heat loss. The overheated adult or infant will assume a flaccid extended position to increase heat transfer to the environment from the skin ***(Baumgart, 1996)***.

Heat production

There are a limited number of ways that man can produce heat, or increase his heat production. Basal heat production (heat production associated with the maintenance of life with energy cost of breathing, heartbeat, maintenance of cell membrane potentials ,etc.) amounts to between 1000 and 2000 kcal/24 hours dependent on sex (less in women, who have proportionately more fat than men), declines with age and is dependent on body size (table 1). The human body is able to respond to cold stress sensed in the skin receptors and hypothalamus through voluntary muscular activity, shivering, and chemical or non shivering thermogenesis ***(Uebel, 2007)***.

The skin receptors first sense a cooler environmental temperature, and through the posterior hypothalamus, by sympathetic nervous control, norepinephrine is released, which will cause the blood vessels in the skin to constrict. Vasoconstriction constitutes one aspect of voluntary muscle control to overcome hypothermia. Another aspect is the ability to produce movement, which increases metabolism, which in turn increases heat production. The body can also assume a flexed position to decrease the amount of skin exposed to the cooler environment, effectively conserving heat for the body ***(Leslie, 2012)***.

Table (1): Mechanisms of heat production

Mechanism	***Effect***
Basal metabolism	Minimum heat production (metabolism) for maintenance of life, Magnitude depends on body size, age and sex
Muscle contraction	Shivering and voluntary (behavioral) activity
Dietary induced thermogenesis	Heat production rises by 10-15% following nutrient (food) intake, particularly marked with protein
Non shivering	Hibernating animals and