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Hypothermia in the Newborn

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With induced hypothermia, body temperature can go down to about 17°C and tissue metabolic needs are reduced to a bare minimum; circulatory arrest can be tolerated for 60 minutes in this temperature range, and the procedure offers a bloodless field of particular value in cardiac surgery. One disadvantage with hypothermia is redistribution of blood flow causing certain organs such as skeletal muscle to be poorly perfused and therefore warmer than other parts with a consequent intolerance of oxygen deprivation. Other problems include acid-base disturbances and possible neurologic defect directly related to the duration of the period of circulatory arrest. (Tyson, 1975). In the newborn, however, hypothermia is likely to produce catastrophic sequelae with a high fatality rate, particularly with temperatures below 32°C in the first day of life.(Arniel and Kerr, 1963). There is an increased mortality rate when temperatures drop below 36°C but the reasons for this are not clear. (Sinerman 58, 1963). Thermoregulation breaks down below 32° and may not recover for a few days, during which time the infant, readily assumes the environmental temperature and may also develop a bleeding tendency. (Cockburn & Drillien, 1974).

Immediately after birth the abdominal skin temperature is the same as deep core temperature. (Miller and Oliver 1966); if cooling is allowed to occur, the skin temperature drops to more than 2° below core temperature. (Silverman and Agate, 1961). The metabolic rate increases, a metabolic acidosis ensues, and energy stores are depleted. (Gundy, 1964). When temperatures approach 25°C bradycardia is manifest; the cardiac cycle is lengthened at the expense of the resting phase, (Brewin 1964; Ree 1964). Below 25°C ventricular fibrillation sets in, and cardiac arrest at 15 C (Brewin, 1974).

Neonates lose temperature very rapidly after delivery especially if uncovered and unattended; the temperature may reach as low as 34°C without suspicion. (Miller, 1966). As far back as 1889, Henoch described *oedema of the newborn* associated with cold injury. The first signs include a feeble cry, a cold skin, failure to feed well, oedema, and erytherma of face and the extremities. The PCV rises and blood sugar diminishes; the latter may be particularly severe during re-warming. (Brooke, 1972; Clockburn and Drillien, 197?; Mann and Elliott, 1957). In fatal cases, petechiae and pulmonary and gastro-intestinal haemorrhages are features at autopsy. Diffuse bleeding may be obvious before death, and are usually associated with convulsions, oliguria and oedema. IVP demonstrates a low renal perfusion. (Mann, 1957).

Hypothermia is one of the factors which may trigger off the coagulation cascade which leads to disseminated intravascular coagulopathy. The neonate's blood is hyperviscous and hypercoagulable; the PCV is normally very high, in the region of 50 to 60%. Associated asphyxia, acidosis or sepsis may trigger off generalised coagulation more readily in the presence of hypothermia, and the organs commonly involved include the nervous system, the gastro-intestinal tract and the kidneys; with the consumption of coagulation factors, there develops a generalised bleeding tendency, with haematemesis, melaena, haemoptysis and haematuria. Fatality rates are extremely high. (Cockburn and Drillien, 1974).

Heat production by the neonate is restricted to metabolic activity as heat derived from muscular movements and shivering are negligible. This thermogenetic tissue is brown fat (Dawkins, 1964) which forms the major part of the 160 grams of the fat per kilo of the newborn (Widdowson, 1950) and is richly supplied with blood vessels, receiving as much as a fourth of the total cardiac output. (Heim and Hull, 1966). Temperature regulation is under the control of the hypothalamus, which in response to cold blood perfusing it and to stimuli from cold sensors in the skin, causes (through the autonomic nervous system) a release of norepinephrine which causes metabolic reactions in brown fat. Triglycerides are broken down to glycerol and fatty acids; the latter are either released into the blood stream, are oxidised or are re-esterified with heat production and calorie consumption. (Cloherty and Startk, 1980; Cockburn and Drillien, 1974).

Thermoregulation by the hypothalamus is in part mediated through TSH which increases in its plasma concentration on exposure of the newborn to cold stress, and this elevated TSH level causes an increase in thyroid activity with heat production. (Cockburn and Drillien, 1974).

The neonate loses heat through a variety of mechanisms. The loss from the body to the surface and thence to the environment is under vasomotor control. Heat is lost by radiation from the body surface, to a cold surface, such as the incubator wall; a heat shield will be warmed by the incubator heat and will diminish radiation losses. A cold surface under the newborn would again cause heat loss through conduction. Moving air such as in the delivery room, oxygen flowing on the face, or during transport to the nursery, will cause convection losses; these are also minimised by heat shields. Finally, an undried infant in the delivery room will loose heat by evaporation.

Low birth weight infants pose greater problems in thermoregulation. Their surface area is large in relation to their body weight, and hence heat is more readily lost through their surface. Subcutaneous fat is scarce and insulation inefficient. Brown fat is also sparse and readily consumed. Their ability to mobilise norepinephrine is limited, and consumption of enough energy for thermogenesis is inadequate. Furthermore, they are more prone to respiratory problems which decrease their ability to increase oxygen consumption for theromogenesis. On exposure to cold stress they undergo peripheral vasoconstriction with resultant anaerobic mechanisms for metabolic activity which in their turn cause or aggravates an acidosis. The low PH would cause pulomonary vessel vasoconstriction, right to left shunting and further hypoxaemia and anaerobic metabolism; a vicious cycle is produced. (Cloherty and Startk, 1980).

Glucose cannot readily enter the intracellular compartment during hypothermia, and the administration of glucose would only cause a hyperglycaemia. Fructose is better than glucose in this respect and is therefore used in performance to replenish supplies of energy. (Arniel and Kerr, 1963; Brewin, 1964).

It seems that in the first few days of life the newborn is not capable of reacting adequately to cold stress by increasing his metabolism of brown fat, (Hey, 1972). It is during this first week that complications from hypothermia are likely to arise. Surgery may be required for a congenital anomaly, and modern air-conditioned operating theatres predispose to lowering of the neonate's temperature during the intervention. With exchange transfusion there exists the additional contributing factor of cold blood being used for exchange. The environmental temperature is ideally maintained at between 28°to 32°C during interventions, and any infused blood should be prewarmed to 37°C prior to administration. (Cockburn and Drillien, 1974). Low temperatures in jaundiced babies increases competition for binding sites on albumin (Cloherty and Stark, 1980) and the risk of death and kernicterus is increased in hypothermic states. (Cockburn and Drillien, 1974). Co-existing hypoxia will aggravate the hypothermia and also cause inefficient utilisation of the limited glucose reserves, thereby increasing the risk of neuronal damage. (Ibid).

In a survey on early neonatal deaths during 1981, the following data was obtained. Total number of deaths were 33, 12 of which were under 28 weeks of gestation, and the rate per 1000 live births equated at 6.3. 12% were due to respiratory distress, 21% to congenital anomalies, 9% to maternal factors, another 9% to meconium aspiration, 6% to perinatal asphyxia, and 3% each for neonatal pneumonitis and intracranial haemorrhage. Hypothermia had been recorded in 15% of these infants who died, and this represents a significant fraction which was both preventable and at least partly contributory to demise.

With healthy infants, drying the skin and wrapping in a warm blanket suffices to prevent unnecessary heat losses; with ill infants and preterms it is also necessary to transport them in warm incubators and to use radiant heathers during resuscitation. The benefits of a heat shield have already been mentioned; this may also help to prevent apnoea which is related to sudden temperature changes. (Dailey et al, 1969; Perstein et al, 1970). Clothing the infant and lining the incubator with foil would render observation difficult and are therefore not recommended.

Servo-controlled incubators have helped in maintaining a neutral thermal environment, and tables exist which indicate the correct temperature for different gesttional ages and body weights. (Klaus and Flanaroff, 1973). Detached skin probes are a potential hazard because hyperthermia is usually precipitated thereby. Sepsis may be masked because of adequate temperature control, so that both dermal as well as core temperature are ideally recorded. (Cloherty and Stark, 1980).

Diazepam is increasingly being used at and around delivery time, and this drug causes problems in the newborn. Feeding is slow, sucking is poor, and lethargy is present; the response to cold stress is impaired ad this may easily predispose to hypothermia. (Ibid.)

Hypothermia in the newborn is largely preventable and is potentially lethal; the maxim "Prevention is better than cure" is particularly applicable here where no great expense nor too much time is required for the maintenance of normothermia in the vulnerable neonate.

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