Physiological Aspects of Heat Exposure as Applied to Flying

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HEAT stress is an important problem encountered during flying in tropics. It has assumed special importance in view of the emphasis now attached to low level high speed flying by deep penetration aircraft. No doubt, the modern aircraft provide for air conditioning of cockpit but since the heat exchangers and cold air units in most aircraft depend on the ram-air for cooling, they are not efficient under high ambient temperatures. Cabin thermal conditions in some of our aircraft during low level high speed flying have been reported to be most unsatisfactory and above the limits of human tolerance from operational point of view. Heat exposure to an environment of 55°C DB, 43°C WB and air velocity of 1.2 metre per second and a wall temperature of 55°C leads to a stage of collapse after exposure for 30 minutes even in acclimatized subjects. Deterioration of efficiency, however, sets in much earlier. It is well known that this degree of heat stress exists in the cockpits of some of our aircraft during summer months. The cockpits get fully heated up specially in aircraft parked in the open. Occupants of such cockpits are thus exposed to these stressful environments for durations which are physiologically hazardous and have a natural tendency to raise their body temperatures. The body has an inbuilt mechanism which attempts to regulate the body temperature whenever it shifts one way or the other but in the heated confines of a cockpit this mechanism is rendered inefficient. The important mechanisms which enable the body to lose heat in the hot environment are radiation, evaporation and respiratory heat exchange. Thermal sensing neurones are present in the periphery and in the hypothalamus. The hypothalamus coordinates the information on temperature changes and has neurones responsible for heat dissipation, conservation and production. Both types of neurones are linked together in such a way that when one is active the other one is inactive. These neurones respond to changes in the local brain temperature and to the neural inputs from spinal cord and peripheral thermoreceptors. During heat stress, the hypothalamus triggers the thermoregulatory effector mechanism via the heat loss neurones at the same time inhibiting the heat production/conservation neurones. The net effect is cutaneous vasodilatation, sweating and panting causing heat loss by radiation, evaporation and respiratory heat exchange. Cutaneous vasodilatation augments blood flow to the skin. It has been seen that the blood flow to the fingers can be increased from 1ml/min/100 gm of tissue to 90 ml/min/100 gm on exposure to heat At an ambient temperature of 34°C, blood flow through skin may increase to as much as 12% of cardiac output. An increased cardiac output ensures a rapid blood flow through the dilated cutaneous vessels and thus the heat is transported from the core to the body surface from where it can be dissipated by radiation. Simultaneously with the vasodilatation, an increase in circulating blood volume occurs. In an unacclimatized man the total blood volume may increase 10% in 2 to 4 hours on exposure to heat of a severity sufficient to cause a diffuse cutaneous vasodilatation. This increase in volume is brought about by fluid drawn from other tissues like skin, muscles and liver into the blood.

This causes a drop in haematocrit and in concentration of plasma proteins. Later, however, haemoconcentration occurs with a decrease in blood volume as a result of copious sweating. In a standing posture, the total area of skin exposed to heat radiation is obviously much greater than in sitting cramped up in the cockpit, dressed in full flying clothing. The situation is further complicated by the fact that the environmental temperature is considerably higher than the skin temperature. Above an ambient temperature of 36°C, the body gains heat by radiation from the environment instead of losing it. This heat together with the metabolic heat is dissipated principally by sweat evaporation from the skin and to a certain extent by increased respiratory activity by promoting respiratory heat exchange. Heat dissipated through evaporation of 1 ml of water from the skin and lungs amounts to 0.58 K cals. Even at ordinary room temperature when there is no obvious perspiration, heat loss through evaporation from skin and lungs amounts to about 17 K cal per hour. About two-third of it is lost through insensible perspiration and the remainder through the respiratory passages. At higher temperatures, heat loss by evaporation from the skin increases tremendously. During severe heat stress, sweating rate may approach as much as 1.6 litres per hour which, if evaporated in toto, would mean a loss of 90 K cal of heat per hour. It stands to reason that this evaporative cooling would diminish under high humidity conditions and in the closed and confined space of the cockpit. The heat disturbances therefore would set in at relatively lower temperatures under these situations.

Heat stresses the cardiovascular system to the maximum. Heart rate increases and so does the stroke volume leading to an overall rise in cardiac output which again is assisted by a fall in peripheral vascular resistance. The resultant effect is an increase in heat loss through radiation and sweat evaporation. The heart rate rises almost linearly with the heat storage. The degree to which the cardiovascular system can take the strain, however, is limited. Tolerance limit is reached when the heart rate rises to 180/min and the core temperature reaches 39.2°C and syncope follows. Well before these limits are reached, the aircrew performance starts deteriorating.

Exposure to heat also brings about an increase in pulmonary ventilation through an increase in tidal volume, the respiratory rate may not change and may even fall. This may lead to hypocapnia responsible for some of the symptoms of heat intolerance. The increased respiratory activity is responsible for about 10% heat loss due to respiratory heat exchange.

Acclimatization to heat occurs in 10 days, and is associated with lower pulse rate, a lower body temperature, an increased blood flow through the skin and a higher sweat rate when working in heat. These indicate a decreased strain in the face of continuing environmental stress. Increased sweating capacity is the result of local training of sweat glands causing an increased secretion in response to the central drive, i.e., the elevation of central temperature. While the sweat content of sodium and chloride are reduced during acclimatization, that of potassium remains elevated. This is because of increased aldosterone production brought about by acclimatization, which conserves sodium and chloride, but increases potassium excretion leading to a negative potassium balance. Loss of appetite which often occurs in summer leads to a further reduction in potassium intake. These factors produce hypokalemia with associated lethargy, weakness and lowering of the amplitude of T-wave of electrocardiogram, usual features of heat disorders. Fatigue, a common feature amongst aircrew after a couple of sorties in summer, is partly the consequence of potassium depletion. In general, aircrew based at air fields in North India who are acclimatized to heat, suffer in summer from a subjective feeling of heat around twentieth minute of exposure, when there is an increase in oral temperature by 1·4°C, mean skin temperature by 4 - 5°C and heart rate by about 50 beats/minute. Heat accumulates in the body and predisposes an individual to heat disorders much faster unless he has been exposed to comfortable environments in air conditioned rooms prior to the sortie.