HYPERPYREXIA

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Although the syndrome of hyperpyrexia has long been recognised in tropical medicine, it seems probable that the diagnosis of hyperpyrexia is still frequently missed and patients die who might easily have been saved. For hyperpyrexia is usually rapidly fatal if untreated, but when recognised and treated early can almost invariably be controlled. The main reasons for failure to diagnose this dangerous condition in its early stages are unawareness of the predisposing conditions and characteristic signs, and continuing confusion about the meaning of the term. It is certainly a mistake to define hyperpyrexia solely in terms of objective measurements of body temperature, because the physiological and pathological effects of specific internal body temperatures vary greatly from species to species, between individuals of the same species, and even from time to time in a single individual. It is better perhaps to define hyperpyrexia in simple functional terms, as a state in which the internal temperature of the body is so high as to interfere with the physiological mechanisms which normally regulate the body temperature. In clinical practice this makes the diagnosis of hyperpyrexia a matter of medical judgement.

All birds and mammals have relatively narrow optimal ranges of internal body temperature, normally maintained by complex temperature regulating mechanisms which can modify both heat loss and heat production. Heat production is a by-product of all metabolic processes, an essential by-product when the external environmental temperature is below the optimal body temperature but just another product for excretion at other times. During the course of evolution most warm blooded animals have developed highly effective measures against heat loss when the normal habitat is colder than the optimal body temperature. By keeping heat loss to a minimum in cold climates they have generally avoided the wasteful necessity of metabolism purely to produce heat, and have thereby conserved energy and reduced their food requirements. Nevertheless, increased heat production, for example by shivering and other muscular activity, is often an important response to sub-optimal internal temperatures. The reverse effect of reduced heat production in above optimal body temperatures is less marked, and usually does not occur at all. It is true that the bird or mammal with a fever tends to rest, thereby reducing muscular activity and consequent muscular heat production to a minimum, but as the body temperature rises above the optimal range there is an accompanying rise in general metabolism and heat production. The most important temperature regulating response in fever is therefore increased heat loss.

In clinical hyperpyrexia there is invariably some disturbance of the heat loss mechanisms, and it is necessary to consider the various ways that heat is eliminated from the body. When the external environmental temperature is lower than the body temperature heat is lost during respiration. The expired air is warmer than the inspired air, and in addition the higher water content of the expired air gives it a higher specific heat, thereby increasing the refrigerant efficiency of respiration. The evaporation of water into the breath from the surfaces lining the respiratory tract removes the latent heat of evaporation of the water from the body. This loss of heat by evaporation is so great in dry atmospheric conditions that even when the inspired air is considerably above body temperature, the overall effect of respiration may still result in effective heat loss. In hyperpyrexia the breathing tends to become shallow and increased in rate. The actual volume of respired air falls, so does its water content, and there is reduced heat loss in respiration.

The human body gives off heat by radiation and receives heat from the surroundings in the same way, the summated effect being proportional to the relative surface temperatures of the body and its external environment. But this form of heat loss is only susceptible to control in so far as the external environment can be changed. In fever the sick dog lies in the shade, and in human hyperpyrexia it is obviously desirable to avoid the direct sun or other sources of excessive radiant heat. Heat loss by radiation however, is not quantitatively and physiologically important in hyperpyrexia.

The human skin is an efficient natural heat conductor, a good excuse for the considerable ingenuity which has been applied to its artificial insulation. In hyperpyrexia there is usually a generalised dilatation of the blood vessels in the skin, and with increased circulation the exposed skin tends to reach temperatures close to the internal body temperature. But as with

radiation, actual heat loss by conduction is usually rather insignificant in hyperpyrexia. If the patient rests, the supporting structures usually are or become at least as hot as the patient's skin, and heat loss by conduction is thereby self-limited. In practice heat loss by conduction is rarely of much importance under normal conditions or in hyperpyrexia unless the body is more or less immersed in water.

By far the most important mechanism for the spontaneous control of pyrexia in human beings is the secretion and evaporation of sweat. And in clinical hyperpyrexia there is invariably inhibition of sweat secretion, producing a dry skin. Irrespective of dehydration or salt depletion, sweating is always inhibited if a high internal body temperature persists too long. This reaction rarely occurs with body temperatures below 103°F, is common at 105°F, and is almost invariable above 107°F. When sweating ceases in fever the most important method of heat loss ceases and the temperature commonly rises abruptly even higher. Signs of dysfunction of the nervous system and cardiovascular system soon appear and death supervenes, unless the temperature is reduced by artificial means or sweating can be induced.

PREDISPOSING CAUSES OF HYPERPYREXIA

Exposure to very high environmental temperatures, particularly in the unacclimatised, may alone be sufficient to cause hyperpyrexia. However in an otherwise fit person the exposure to high climatic temperatures usually has to be prolonged for several hours and other factors are equally important such as dehydration, enclosure in a confined space, high atmospheric humidity and clothing preventing the normal evaporation of sweat. The commonest causes of hyperpyrexia are infective fevers, particularly those associated with delirium, restlessness and increased muscular activity. Cerebral malaria, tetanus and the meningitides are particularly liable to produce hyperpyrexia and precautions against this complication are an important part of the management of these conditions. Rarer causes of hyperpyrexia are staphylococcal septicaemia, streptococcal erisipelas, mid-brain damage and thyrotoxic crisis. But almost any infection in the tropics combined with factors reducing the secretion and evaporation of sweat may occasionally result in hyperpyrexia.

Unfortunately, hyperpyrexia is often precipitated by misplaced kindness and hygienic considerations. Many patients with high fevers at times feel cold, and it is common to see patients shivering under a pile of blankets with body temperatures as high as 104°F. In such cases the patients do not necessarily know what is best and the desire for more bedclothes should be resisted. Usually a few words of explanation will reassure the patients and the feeling of cold will pass off. But in some cases sedation is advisable, particularly in treating transfusion reactions and delirious fevers. In hospital most patients are nursed on rubber undersheets which greatly reduce heat loss from the body, and this is certainly one of the main precipitating causes of hyperpyrexia among hospital admissions. The tendency to replace flock mattresses by rubber foam ones will no doubt increase this problem. A comfortable, porous and easily cleanable mattress with good heat conducting properties is badly needed for hospitals in the tropics, and indeed would be an immediate success everywhere.

The modern tendency to treat most serious infections with more or less appropriate antibiotics has unfortunately led to a neglect of older and well tested remedies. In particular there is little doubt that aspirin is still a valuable drug in treating fever and its diaphoretic action is a protection against the development of hyperpyrexia.

THE SIGNS OF HYPERPYREXIA

As mentioned earlier, hyperpyrexia may occur in some patients with quite moderate fevers. Other patients can stand high fevers for long periods without evidence of any permanent ill effects. Nevertheless, functional hyperpyrexia is so common with fevers exceeding 105°F in the tropics that it is wise to treat all such patients as for hyperpyrexia. Unfortunately the routine method of taking the temperature orally with a thermometer under the tongue is liable to grave errors. Many patients with high fever take periodical cool drinks, or breath through the mouth. Patients in shock may have mouth temperatures several degrees below the true internal temperature. Many are uncooperative and do not really keep the thermometer closely under the tongue. Without doubt many of these pitfalls of taking temperatures in the mouth can be avoided by careful forethought and attention to technique. But in hospital practice, routine temperatures are usually taken by the overworked most junior nurses and are not adequately reliable for the detection of incipient or actual hyperpyrexia. The axillary temperature is less unreliable, but whenever hyperpyrexia is likely to develop or is suspected rectal temperatures

are essential. The temperature may rise very fast to dangerous levels in some fevers and four hourly temperature checks, even if taken rectally, are inadequate in those conditions, such as cerebral malaria, pyogenic meningitis and uncontrolled tetanus, which commonly result in hyperpyrexia. Although it means considerable labour and inconvenience, two hourly rectal temperatures in these three conditions actually save work for the nursing staff for it may only take a few minutes to sponge a rising temperature down from 104°F while two hours or so are commonly required at 106°F. And of course the aim should be to prevent hyperpyrexia, rather than to repeatedly treat it.

The patient in hyperpyrexia usually has a dry flushed skin, with sometimes slight cyanosis of the extremities. The exposed skin may feel cool to the touch, but if the hand is inserted under the patient the unexposed skin feels unpleasantly hot to the touch. With experience the internal temperature can be gauged in this manner with remarkable accuracy. The respiration is usually shallow and the rate is high, even in the absence of abnormal chest signs. There is a pronounced tachycardia, and pansystolic cardiac murmurs can often be heard on auscultation. With the onset of hyperpyrexia, even patients without previous evidence of mental changes tend to become restless and even delirious. Epileptiform convulsions may occur, and are common in children with hyperpyrexia. Older patients usually pass into coma at 107-108°F, and children at slightly higher temperatures. If the patient passes into a state of shock, the pulse becomes very weak, and the skin even if unexposed tends to become cool, while the respiration may become stertorous or intermittent.

The clinical suspicion of hyperpyrexia is based upon the finding of dry skin, very hot to the touch where unexposed, with a rapid shallow respiration. It should always be confirmed by taking the temperature of the patient rectally.

THE TREATMENT AND MANAGEMENT OF HYPERPYREXIA

This is a medical emergency. The treatment of hyperpyrexia should never be left entirely in the hands of a junior nurse, but should be supervised by a trained nurse or doctor with full authority to force quick and effective action.

The immediate aim of treatment should be to reduce the internal body temperature as quickly as possible to about 105°F and then more slowly to about 101°F. If the temperature is allowed to drop precipitously to 101°F or below a state of shock commonly supervenes, and although this vasomotor collapse may respond to the intravenous administration of noradrenaline it is sometimes fatal.

Several effective methods have been used to reduce the fever in hyperpyrexia, but most of them are not available when required in an emergency. For example the various forms of heart lung machine developed for open heart surgery can easily be adapted to pass the patient's blood through a refrigerating coil and so reduce the body temperature to any level required. Similarly the disposable coil artificial kidney will quickly cool the body temperature if the dialysing fluid is maintained at a low temperature. The intravenous administration of one or two litres of ice-cold normal saline is another dramatic method of initiating cooling, and is not inappropriate since most patients with hyperpyrexia are somewhat dehydrated. Each litre of ice-cold saline infused will reduce the body temperature by about one degree Fahrenheit in an average hyperpyrexial adult. But in practice the ready cooled saline is rarely available when it is wanted. Hyperpyrexia has sometimes been treated by immersing the patient in a bath of cold water, and if the patient is fully conscious and able to get into a bath without help this is safe and a very effective method of cooling. Once in the bath the temperature of the water can be gradually reduced by adding ice, if it is available, and the whole procedure is not uncomfortable. However, most patients with hyperpyrexia have impaired consciousness and may even be delirious and uncooperative. It is difficult to hold a limp or struggling patient in a bath of water without some risk of the wet body slipping and drowning, and on the whole this method of cooling patients with hyperpyrexia is not recommended.

Hyperpyrexia usually occurs where there is a lack of facilities to treat the condition, or in hospital due to lack of nursing care and neglect. In these circumstances there are three basic requirements. understanding, water and a good draught. Without understanding treatment is usually inefficient, ineffective, and wasteful in time and labour. Water is required to moisten the skin. The water need not necessarily be very cold, though this is preferable, and nor are vast amounts required. Used intelligently, 2 or 3 litres of water applied to the skin and evaporated therefrom are usually ample to reduce the fever of hyperpyrexia to safe levels. The importance of a good draught cannot be overemphasised. Evaporation can only continue

from the skin as long as the water saturated air near the skin is constantly replaced by fresh unsaturated air. The patient should be stripped. It is no time for modesty, and in addition the patient should be placed to expose as much skin to free circulation of air as possible. A bed is better than the floor, but several chairs or a wickerwork chaise-longue are better still to support the body. The patient's skin should be continually moistened with wet cloths so that as much as possible of the skin surface is damp and cooled by evaporation of water into the atmosphere. The rectal temperature should be taken every 10 to 15 minutes, and if no thermometer is available the examiner's finger introduced into the patient's rectum is a better guide to body temperature than none at all. In severe hyperpyrexia it is always advisable to use ice-cold water, if available, for sponging the skin, and a fan blowing directly on and across the patient is a great help. If hyperpyrexia is treated indoors, the room must be airy and well ventilated. An open veranda is always preferable to a room, unless the room is strongly air-conditioned. The patient should be kept well away from walls, and screens should only be used if essential and then should be as far from the patient as possible and placed so as to allow full benefit of any natural air currents. Even under good conditions in hospital, with an electric fan, plenty of ice-cold water, and hard working nurses, it may occasionally be as long as an hour before this treatment begins to reduce the fever in hyperpyrexia. But with patience and perseverance almost every case of hyperpyrexia can be controlled by these simple measures. Ice-cold enemata are probably never indicated in the management of hyperpyrexia. Apart from preventing the simple and accurate measurement of body temperature in the rectum, these very cold enemata seem to precipitate shock. The common mistakes that impair the cooling effectiveness of cold sponging are nursing the patient on a soft bed covered by a machintosh, having the bed near a wall or in a corner, having screens closely around the bed, having the patient covered by a towel, having too many useless onlookers around the bed, and having no natural or artificial breeze blowing over the patient.

If and when the temperature does begin to fall, the sponging should be continued vigorously until the rectal temperature is down to 103°F, and then less energetically until 101°F is reached, or in the absence of a thermometer until the rectum just feels comfortably warm to the examining finger. A careful check should be kept on the patient's temperature even after the temperature has successfully responded to treatment, because unless the original causes of the hyperpyrexia can be treated the condition is of course likely to recur. It is a reasonable precaution to take temperatures two hourly in all patients for 24 hours after a hyperpyrexial attack.

Whatever the initial cause of hyperpyrexia, pneumonia frequently follows the successful reduction of temperate by cold sponging unless prophylactic treatment is given. The routine administration of 1 million units of procaine penicillin during the sponging has all but eliminated this complication. No doubt the tetracyclines, chloramphenical and erythromycin would do as well.

The main remaining hazard following hyperpyrexia is cardiac failure. This seems to be due to the specific effects of fever on the myocardium, although the onset of symptoms and signs of cardiac involvement may be delayed as long as 48 hours after the fever has been reduced. This complication of hyperpyrexia is frequently as fatal as it is unexpected. A 12 year old girl with staphylococcus aureus septicaemia developed fever with a rectal temperature of 110.2°F (the thermometer was later checked and found to be accurate!). Following effective emergency sponging and antibiotic therapy the child's temperature fell and was maintained between 99.2° and 100.4°F. 24 hours after the temperature of 110.2° had been registered the child was sitting up in bed and eating well. She had no cardiac irregularity and no signs of cardiac failure. Yet the following day she developed increasing ventricular extrasystoles and signs of left heart failure. She died in cardiac failure over 2 days after she had been in hyperpyrexia although her causal septicaemia seemed to be well under control. The same sequence of cardiac failure ushered in by increasing extrasystoles has been observed following hyperpyrexia due to tetanus, viral encephalo-meningitis, malaria and pyelitis. Yet primary cardiac failure is an uncommon complication of these conditions when there has been no hyperpyrexia, and so the relationship of cardiac failure to hyperpyrexia per se seems probable. Once this complication occurs it is often fatal and does not respond to strict rest, corticosteroid therapy or digitalis. It seems reasonable however to keep all patients on strict bed rest for at least three days after hyperpyrexia to reduce cardiac work and strain to the minimum.

SUMMARY

Hyperpyrexia is defined in functional terms. It is often unrecognised, and is usually fatal if untreated. The diagnosis should be suspected when a patient has a hot dry skin and rapid shallow respiration, whatever the reported oral temperature. Cold sponging is effective if carried out in a good draught. The complications of relapse, pneumonia and cardiac failure can be minimised if certain precautions are observed.