

A STUDY OF BODY TEMPERATURES OF ANAESTHETIZED MAN IN THE TROPICS

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SUMMARY

Body core and skin temperatures were measured in 20 African patients undergoing herniorrhaphy in hot and humid conditions; one half of the patients received halothane and the other half received diethyl ether. No difference was found between these two groups. Body core temperatures decreased even with an ambient temperature of 28.7 °C and a relative humidity of 72% and all sites reached a new thermal equilibrium at 30 min. It is suggested that the level of the re-established thermal equilibrium is a function of the skin to ambient thermal gradient, which depends on the failure of the countercurrent heat exchange mechanism as a result of redistribution of peripheral blood flow.

In a thermally neutral environment, general anaesthesia induces a modest reduction of body core temperature. If thermosensitive probes are attached to an anaesthetized patient at various sites it can be shown that, concurrent with the decrease of body core temperature, the skin temperature increases and the magnitude of the changes depends on ambient temperature (Foregger, 1943). The increase in skin temperature is a result of the peripheral vasodilatation and causes a net loss of heat from the body by physical transfer to the environment. If this heat loss is simply a result of radiation, conduction and convection, the total loss of heat from the body would correlate with the temperature gradient between the skin and the surroundings; thus in a cool environment there should be a greater heat loss than in a hot environment and if ambient temperature is similar to skin temperature, no heat loss could occur.

Evaporation of sweat from the body surface results in loss of heat because of the consumption of latent heat of evaporation. In a cool environment, anaesthetized patients are unlikely to lose much heat from sweating, especially if their body core temperature has decreased (Benzinger, 1969). In hot ambient conditions, sweating produces a more significant heat loss than the other physical methods (Fox, 1974). The thermal efficiency of sweating depending on the ambient relative humidity is ineffective if the air in direct contact with the skin is fully saturated with water.

Mild hypothermia is usual during general anaesthesia, although a few patients develop an increasing body core temperature even in cool ambient conditions (Stephen, 1961; Goldberg and Roe, 1966). Several possible causes of this pyrexia have been identified and these include hot carbon dioxide absorbers, inability to sweat after an antisialogogue has been given, mismatched blood transfusion, metabolic stimulation in poorly controlled thyrotoxicosis and in epilepsy, endogenous pyrogens in acute septicaemia and other infections such as malaria, and i.v. administration of exogenous pyrogens. Hot environmental conditions have been blamed for pyrexia during operation and hyperpyrexia after operation. If environment alone could be responsible for pyrexia during anaesthesia, it should be encountered in thermally uncontrolled operating rooms in Accra, Ghana.

A study of body temperatures was made in the hot and humid conditions prevailing in Accra to determine the influence of the environment on the temperature of anaesthetized patients. In order to facilitate comparisons between patients, a standardized surgical procedure was chosen, namely herniorrhaphy. This provided an opportunity to compare the influence of two different anaesthetic agents, diethyl ether and halothane, on body heat balance. The reason for this choice was that diethyl ether is known to cause a release of catecholamines and "ether convulsions" are associated with pyrexia. Halothane differs from ether in both these regards.

METHODS

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Twenty healthy male African patients between 19 and 44 years of age undergoing herniorrhaphy were studied. Ten unselected patients were anaesthetized using halothane, and 10 with diethyl ether. Pethidine

and atropine were given routinely for premedication and anaesthesia was induced with Althesin. The halothane group then received only nitrous oxide, oxygen and halothane from a Fluotec vaporizer via a Magill circuit and face mask. The ether group were given nitrous oxide, oxygen and diethyl ether from a Boyle bottle via a Magill circuit and face mask, but in order to avoid excitation 1.0% halothane was added to the inspired gas for about 2 min after induction.

Before induction of anaesthesia, thermistor probes (Yellow Springs) were placed as follows: rectum, tympanic membrane, brow, dorsum of thumb, dorsum of great toe, outer calf and outer thigh. The temperatures at these sites were read at intervals using a galvanometer to measure the unbalance of a Wheatstone bridge circuit to which each thermistor in turn was connected by means of a multipole switch. Before starting the study, all probes were calibrated using a mercury-in-glass solution thermometer. It was impossible to calibrate the Wheatstone bridge circuit because the probes had different resistances. Calibration curves were constructed for each probe and every temperature reading was corrected later against its respective calibration curve.

All patients received anaesthesia for at least 40 min and, to facilitate comparisons between groups, the temperatures at the various recording sites were plotted at 5-min intervals from the time of induction of anaesthesia.

Ambient temperature and relative humidity were measured using wet and dry mercury-in-glass thermometers (whirling type).

RESULTS

No significant difference was found between the halothane and the diethyl ether groups. The changes in the temperatures at the seven sites are illustrated in figures 1, 2 and 3, in which the results from the 10 patients in each anaesthetic group are averaged.

From figure 1 it can be seen that the body core temperatures decreased initially but stabilized towards the end of the 40-min period. The earlier change was detected in the tympanic membrane temperature which preceded the decrease in rectal temperature by about 15 min. The temperatures at both sites decreased by a similar amount, although out of phase. The rectal temperature at the beginning and the end was 0.15 °C greater than the tympanic temperature.

Some skin temperatures showed a much greater change than the core temperature. In figure 2, it can be seen that the skin temperature on the dorsum of the great toe, which was less than 30 °C initially, showed

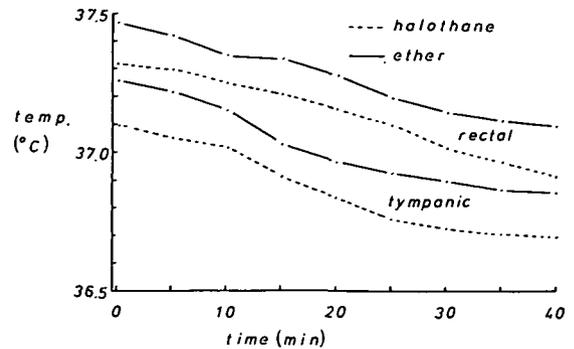


FIG. 1. Changes in body core temperatures during 40 min of anaesthesia; each point represents the grouped mean values for patients anaesthetized with either halothane or diethyl ether; there is no significant difference between the halothane and ether groups although both sites show a significant decrease.

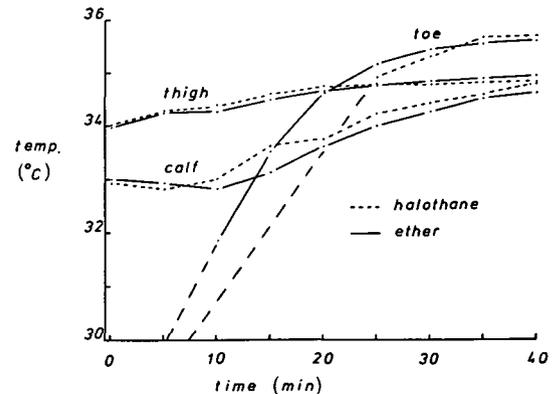


FIG. 2. Changes in distal skin temperatures; there is no significant difference between the halothane and ether groups.

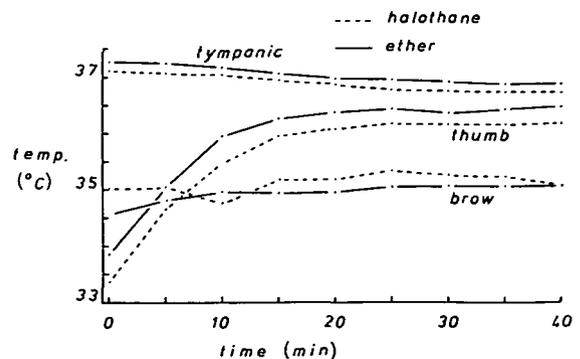


FIG. 3. Changes in temperature of body core (tympanic), proximal skin (brow) and distal skin (thumb); there is no significant difference between halothane and ether groups.

the most marked increase shortly after induction of anaesthesia. All the skin sites on the lower limb showed similar qualitative changes, but the more proximal sites showed least change.

The temperature of the dorsum of the thumb increased rapidly in a manner similar to that of the toe (fig. 3) and both these sites approached close to body core temperature, the difference being approximately 1 °C. The brow temperature showed the least change of all the skin sites and remained fairly stable throughout.

The mean ambient conditions throughout the study were a mean temperature of 28.7 ± 1.5 °C and relative humidity of $72.2 \pm 2.5\%$.

DISCUSSION

Throughout the study it was clear that there was no tendency for body core temperature to increase during anaesthesia in tropical conditions. In fact there was a small but definite decrease in body core temperature. This finding contrasts with that of Morris (1971), who suggested that for anaesthetized men the neutral thermal range is 24–26 °C and that body temperature will remain normal with ambient temperatures in excess of 21 °C (Morris and Wilkey, 1970). However, it is possible that the African is better adapted to these ambient conditions than is the European, and that he can still lose heat when thermal gradients are small. From these results there is no suggestion that adverse thermal environmental conditions can induce pyrexia during anaesthesia.

One interesting finding was the marked change in the peripheral temperatures following induction of anaesthesia. Using a different thermometer, it was possible to check that the dorsal toe temperature before anaesthesia was close to the ambient temperature. The rapid increase in toe temperature must reflect a great increase in blood flow. The change in the most peripheral site was greatest and a higher temperature was reached on the skin of the toe than more proximally on the calf and thigh. It is likely that the countercurrent mechanism for heat retention (Hardy, 1972) of the conscious person is abolished or severely attenuated. This mechanism alone could account for much of the heat loss from the body core in the anaesthetized subject.

The stability of the brow temperature was surprising. Perhaps the time-honoured habit of "feeling the brow" may have some rationale, as it seems to be comparatively unaffected by anaesthesia. The brow is perhaps the best thermally adapted part of the skin and sweating often commences on the brow during

pyrexia. It may be that, even under anaesthesia, the brow skin is capable of thermal adaptation and this would be brought about largely by skin blood flow regulation.

After 20 min of anaesthesia, the changes in temperature at most sites showed a tendency to diminish and at 30 min little further change was seen; a new thermal equilibrium had been established. The operative site was too small to make a real contribution to continuing heat loss, and anaesthesia was stable. It is tempting to suggest that the hypothermic effect of anaesthesia is brought about initially by disturbing the thermal equilibrium by increased loss with cutaneous vasodilatation, and after a short period the body temperature control is re-established, but at a new level which depends on the thermal gradient between the skin and the environment.

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REFERENCES

- Benzinger, T. H. (1969). Heat regulation: Homeostasis of central temperature in man. *Physiol. Rev.*, **49**, 671.
 Foregger, R. (1943). Surface temperature during anaesthesia. *Anesthesiology*, **4**, 392.
 Fox, R. H. (1974). Temperature regulation with special reference to man; in *Recent Advances in Physiology*, (ed. R. J. Linden), p. 340. London: Churchill Livingstone.
 Goldberg, M. J., and Roe, C. F. (1966). Temperature changes during anaesthesia and operations. *Arch. Surg.*, **93**, 365.
 Hardy, R. N. (1972). Heat production and heat loss; in *Temperature and Animal Life*, p. 25. London: Edward Arnold.
 Morris, R. H. (1971). Operating room temperatures and anaesthetized paralysed patients. *Arch. Surg.*, **102**, 95.
 — Wilkey, B. R. (1970). The effects of ambient temperature during surgery not involving body cavities. *Anesthesiology*, **32**, 102.
 Stephen, C. R. (1961). Postoperative temperature changes. *Anesthesiology*, **22**, 795.

ETUDE SUR LES TEMPERATURES DU CORPS D'UN HOMME ANESTHESIE SOUS LES TROPQUES

RESUME

On a mesuré les températures du corps et de la peau de 20 malades africains subissant une herniorraphie dans un climat chaud et humide; la moitié des malades a été anesthésiée par l'halothane et l'autre moitié par l'éther diétyl. On n'a constaté aucune différence entre ces deux groupes. Les températures du corps ont baissé même par une température ambiante de 28,7 °C et une humidité relative de 72%; tous les points ont atteint un nouvel

équilibre thermique après 30 min. Il ressort de cela que le niveau de l'équilibre thermique rétabli est une fonction du rapport entre la température de la peau et la température ambiante qui dépend de l'insuffisance du mécanisme d'échange de chaleur à contre-courant par suite de la redistribution du débit sanguin périphérique.

STUDIE DER KÖRPERTEMPERATUREN NARKOTISIERTER PATIENTEN IN DEN TROPEN

ZUSAMMENFASSUNG

Innen- und Hauttemperaturen wurden bei 20 afrikanischen Patienten gemessen, die unter heiss-feuchten Klimabedingungen einer Bruchooperation unterzogen wurden, wobei die Hälfte von ihnen mit Halothan, die andere Hälfte mit Diäthyläther narkotisiert wurden. Zwischen den beiden Gruppen ergaben sich keine Unterschiede. Die Körperinnentemperaturen sanken bei einer Umgebungstemperatur von 28,7 °C und bei einer relativen Feuchtigkeit von 72% sogar ab, und überall wurde nach 30 Minuten ein neues thermales Gleichgewicht erreicht. Es scheint, dass die Höhe des wiederhergestellten thermalen Gleichgewichtes eine

Funktion des Verhältnisses zwischen Hauttemperatur und Umgebungstemperatur ist, das von dem Versagen des Gegenströmungs-Wärmeaustauschmechanismus' aufgrund der Veränderungen des peripheren Blutkreislaufes abhängig ist.

UN ESTUDIO DE TEMPERATURAS CORPORALES EN EL HOMBRE BAJO ANESTESIA EN LOS TROPICOS

SUMARIO

Se midieron las temperaturas cutánea e interior en 20 pacientes africanos sometidos a herniorafia en condiciones acuosas y húmedas; una mitad de los pacientes recibió halotano y la otra mitad recibió eter dietilo. No fué hallada diferencia alguna entre estos dos grupos. Las temperaturas interiores del cuerpo decayeron aún con una temperatura ambiental de 28,7 °C y una humedad relativa de 72%; todos los sectores alcanzaron un equilibrio térmico a los 30 min. Se sugiere que el nivel térmico equilibrado reestablecido es en función de la gradiente térmica entre la piel y el ambiente, que depende de la falla del mecanismo termopermutador contracorriente debida a la redistribución de la circulación sanguínea dérmica.