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ORAL, RECTAL AND OESOPHAGEAL TEMPERATURES AND SOME FACTORS AFFECTING THEM IN MAN

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It is now well known that there is no single body temperature and that the temperature of different tissues may vary widely. Nevertheless, the relative constancy of temperatures recorded in the mouth, rectum and axilla suggests that there may be a temperature in the body which is finely regulated. This temperature is, presumably, that existing at the central temperature receptors, but the precise location of these structures in man remains unknown. Information about this temperature and its changes in response to changes in body heat will therefore continue to be sought from convenient orifices such as the mouth, oesophagus and rectum. This paper compares the temperatures at these sites in man, and the ways in which they are affected by changes in conditions that occur commonly in experiments.

METHODS

Temperature was measured every minute with copper-constantan wire thermocouples (copper gauge 36, constantan gauge 34). The wires were contained in polythene tubing of 1·5 mm external diameter with the junction either enclosed within the tubing or protruding a few mm beyond its end. For measuring rectal temperature a hollow bulb was constructed of thin silver 4 cm in length and 1·8 cm in diameter; a copper-constantan junction was soldered to the inside of the bulb. The bulb was attached to polythene tubing 3·0 mm in external diameter which contained the wire leads and steel wire to reduce its flexibility. In the mouth, in the rectum or in a water-bath the behaviour of the bulb was identical with that of a wire thermocouple. In a water-bath the thermocouples responded more rapidly than a mercury thermometer, and the readings corresponded within 0·03° C. The largest difference observed between a thermocouple and a mercury thermometer in the mouth wa s 0·05° C, and this msy have been partly due to slight differences in position of the instruments.

The reference junction was contained in a paraffin-filled Thermos flask which remained immersed in a thermostatically controlled water-bath at 35° C. Under these circumstances variations in the reference temperature did not exceed 0·02° C during any experiment. The circuit included a resistance of 100 Ω, a moving-coil galvanometer and a switch-board so constructed that all its contacts were of copper or constantan for the appropriate thermocouple wires. Additional junctions

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were unavoidably included with the brass connexions to the 100 Ω resistance and the galvanometer, but the thermoelectric potentials arising in them were small. Increasing the room temperature by 1° C produced a false reduction of temperature readings from the thermocouples of 0·05° C. The galvanometer produced a full response in 7 sec and a scale deflexion of about 36 mm for each degree centigrade temperature change so that a deflexion equivalent to 0·015° C was measurable. Calibrations with a mercury thermometer confirmed that relatively rapid changes of 0·015° C could be measured; calibrations at the same room temperature were identical and were not repeated before each experiment. The room temperature did not change by more than 1° C during any one experiment.

The oral thermocouple was inserted under the tongue and the subject instructed not to move the tongue away from the floor of the mouth; the lips were then sealed with adhesive tape. The tip of the bulb thermocouple was introduced 10–15 cm beyond the anus. By its intimate contact with the whole circumference of the rectal mucosa it was hoped to avoid interference by faeces and to integrate to some extent the temperature differences at various sites described by Mead & Bonmarito (1949). The oesophageal junction was swallowed to a distance of 47 cm from the lips. Radiological examination showed that in this position the thermocouple tip lay immediately above the cardiac sphincter. A steady state of temperature was usually obtained 5 min after insertion of the rectal and oesophageal thermocouples. In the mouth a steady temperature record was not obtained until the junction had been in place for 10–20 min.

In some experiments changes in cutaneous vasomotor tone were followed with the hand calorimeter described by Greenfield & Scarborough (1949).

Where mean figures are quoted, they are accompanied by their standard errors.

RESULTS

Comparison of temperatures under the tongue, in the oesophagus and rectum

In ninety-three experiments on forty seated subjects at room temperatures of 19–24° C, in the different experiments simultaneous sublingual and rectal temperatures were compared over a stable 10 min period. Sublingual temperature was always lower than rectal temperature, the mean difference being $0{\cdot}35 \pm 0{\cdot}01$° C. In twenty-four observations on seven subjects, the oesophageal temperature 47 cm from the lips was $0{\cdot}09 \pm 0{\cdot}015$° C higher than the sublingual, and $0{\cdot}24 \pm 0{\cdot}02$° C lower than the rectal temperature. Fig. 1 illustrates the considerable temperature gradient along the length of the oesophagus. The initial temperature record is from the oesophagus at a point 50 cm from the lips and, after attaining a steady temperature, the thermocouple is withdrawn in five steps of 5 cm each. The temperature is seen to fall as the thermocouple ascends the oesophagus until at a position 25 cm from the lips the temperature is 0·37° C lower than that at the original depth. Similar results were obtained in two further subjects although the temperature changes between 50 and 35 cm were variable. In seven experiments on three subjects, temperature was measured in the oesophagus at 47 cm and 30 cm from the lips; in all it was found to be lower at the 30 cm level, with a mean difference of $0{\cdot}49 \pm 0{\cdot}05$° C.

Temperature changes at rest

In subjects quietly seated in constant environmental conditions, the temperature at all sites tended to fall slowly. In eleven subjects the difference between the initial and final temperatures was found in twenty-five basal periods, lasting from 30 to 70 min. The sublingual temperature rose by 0·02 to 0·12° C in eight experiments, in only three of which did the rise exceed 0·05° C. In three cases the temperature did not change but in the remainder it fell; the largest fall was 0·42° C. The rectal temperature rose in one experiment by 0·14° C and in two cases by 0·02° C. There was no change in one instance and in the remainder the temperature fell; the largest fall was 0·61° C.

Temporary fluctuations in sublingual and rectal temperatures were rare and always less than 0·06° C. The temperature in the oesophagus showed larger fluctuations, and although the lips were sealed fell by a maximum of 0·14° C with swallowing and rose by 0·06° C with eructation.

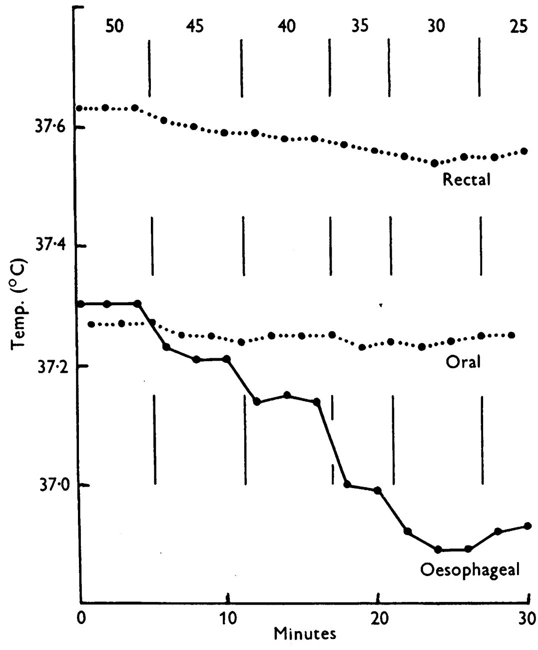

Fig. 1. The temperature gradient along the oesophagus. The tip of the thermocouple was withdrawn in steps, and its distance from the lips in cm is indicated by the figures above the graph. The abscissa represents time in minutes and the ordinate temperature in °C.

Postural changes

The effect on temperature of changes in posture was observed in six experiments on three subjects. The subject remained standing, without support until temperatures had attained a steady state, and then he lay down. After 30–45 min in the horizontal position he resumed a standing posture. Fig. 2 gives a typical example. The subject was initially standing upright, and lay down at the 19th min. There was a parallel fall of about 0·4° C in temperatures. On standing up again at the 64th min the temperatures rose, the oral more steeply than the rectal, ultimately reaching levels similar to their original ones. In all cases there was a rapid fall of oral and rectal temperatures on lying down; the sublingual fall ranged from 0·21 to 0·63° C and the rectal from 0·25 to 0·63° C. In the horizontal position a stable temperature was not usually reached within the experimental period. In three of these experiments temperature measurements were continued for at least 30 min after the subject stood up again. In all cases a gradual rise in temperature was noted but a stable level was not always attained during the period of observation.

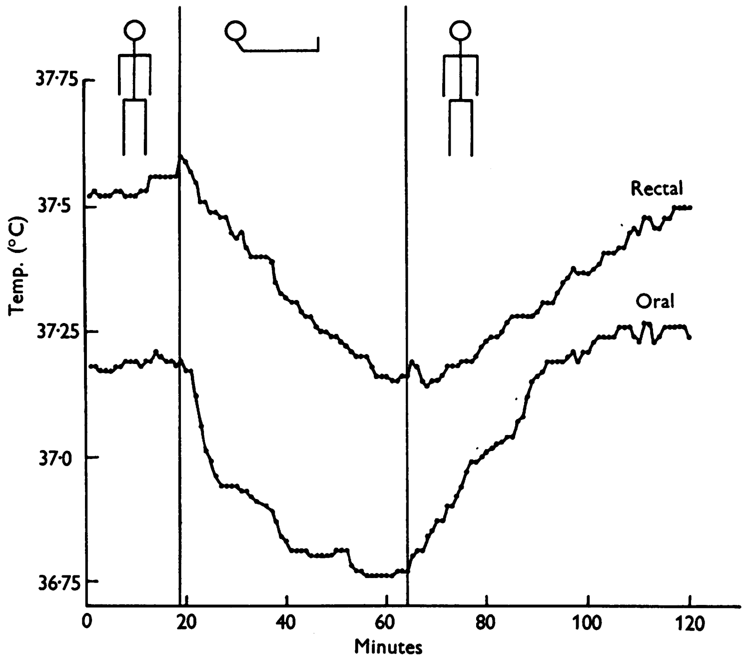

Fig. 2. Oral and rectal temperatures during change of posture. The symbols indicate the position of the subject.

Oesophageal temperature was measured in three experiments and behaved in the same way as the sublingual. Heat elimination from the hand was also followed during these experiments; it fell progressively on lying down with a further temporary fall on standing up again.

The influence of a general cutaneous vasoconstriction and vasodilatation

In five experiments on three subjects the effect on the sublingual temperature was observed of a stimulus provoking reflex cutaneous vasoconstriction. The right forearm was placed in a water-bath at 37° C to prevent heat loss from that limb. When temperatures and heat elimination from the other hand were stable, a cuff around the upper part of the immersed arm was inflated to 200 mm Hg; this arm was then transferred to a bath at 12–14° C where it remained for at least 10 min. In all experiments the heat elimination from the other hand fell sharply and the sublingual temperature rose by 0·03–0·11° C; in two instances there was an initial small fall in temperature of 0·02 and 0·03° C lasting for 2 min. Fig. 3 illustrates a typical experiment and shows the fall of heat elimination and the rise of the sublingual temperature after the cold stimulus. On release of the cuff there is a profound fall in sublingual temperature and a further fall of heat elimination. The effect of this procedure on heat elimination was the same as that observed by Pickering (1932).

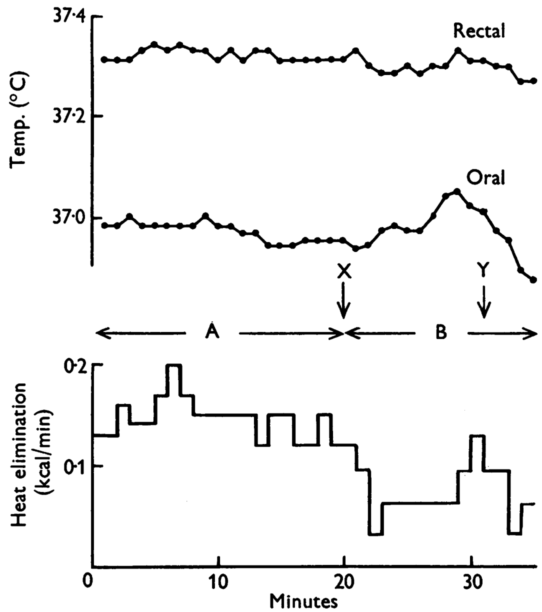

Fig. 3. Response of oral and rectal temperatures and heat elimination from the left hand to a stimulus provoking reflex cutaneous vasoconstriction. During the period A, the right arm was immersed to the elbow in a water-bath at 37° C. At *X* a cuff around the right upper arm was inflated to 200 mm Hg and the arm was transferred to a bath at 13° C where it remained during the period B. At *Y* the cuff was deflated.

In connexion with other work we have observed the rise in sublingual and rectal temperature after an intravenous injection of bacterial pyrogen in twenty-eight experiments on fifteen subjects. During the rise of rectal temperature and phase of intense cutaneous vasoconstriction, the sublingual temperature usually showed a parallel rise, but in five experiments it fell. In two of these experiments the oesophageal temperature was also followed and rose during the fall in sublingual temperature. We are satisfied that these falls in sublingual temperature were not caused by displacement of the thermocouple. The falls ranged from 0·61 to 1·56° C. They began 8–22 min and reached their lowest levels 24–42 min after the onset of the cutaneous vasoconstriction. In these instances the fall in sublingual temperature must be considered to be due to a greatly diminished oral blood flow over a long period.

Kerslake & Cooper (1950) have shown that radiant heat applied to a large area of skin produces a cutaneous vasodilatation mediated by cutaneous receptors. They further observed that this manoeuvre caused a fall in oral temperature (Cooper & Kerslake, 1948). We have repeated their observations in three subjects. In four experiments of this nature when the skin of the

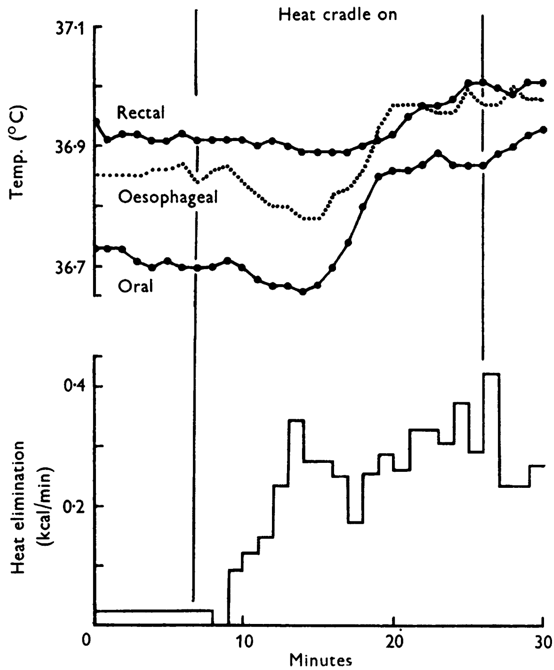

Fig. 4. Response of oral, rectal and oesophageal temperatures and heat elimination from the hand to exposure of the trunk to radiant heat.

trunk was exposed to a radiant heat cradle, the oral temperature fell by 0·03 to 0·10° C during the first 10 min after switching on the heat cradle and while the heat elimination was rising. Fig. 4 shows a typical example, and it can be seen that the temperature in the oesophagus 47 cm from the lips responded in a very similar fashion to the oral temperature. The response of oral temperature during reflex cutaneous vasodilatation and vasoconstriction may be interpreted as reflecting the changes in total body heat secondary to increase or decrease of total heat loss through the skin.

The effects of hyperventilation on temperature

In eight experiments on five subjects maximal hyperventilation through the nose was carried out with room air for 1–3 min whilst the sublingual temperature was measured. The temperature fell in each case, the fall ranging from 0·04 to 0·19° C. In three of these experiments the rectal temperature was recorded and fell by 0·05° C in each case. On one occasion the temperature was measured both above and below the tongue. The supralingual temperature fell by 0·5° C and the sublingual by 0·05° C. The fall in rectal temperature during hyperventilation suggests that cooling occurs throughout the body and that the fall in sublingual temperature is due only partly to local cooling in the mouth.

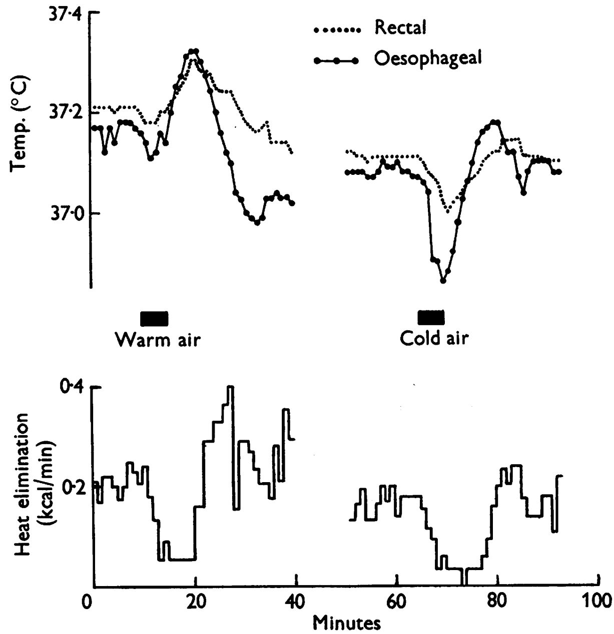

Fig. 5. The first part of the figure shows the response of oesophageal and rectal temperatures to hyperventilation for 5 min with air at 39 to 40° C saturated with water vapour. The second part shows a similar period of hyperventilation with room air at 23 to 24° C. Periods of hyperventilation are represented by solid blocks.

During hyperventilation we have observed, as have others (Bolton, Carmichael & Stürup, 1936), a cutaneous vasoconstriction as indicated by a fall in heat elimination from the hand. To confirm that the vasoconstriction is due to factors other than the fall in body temperature, the effect was observed of hyperventilation with water-saturated air at about body temperature. Fig. 5 illustrates an experiment in which the subject carried out maximal hyperventilation firstly with air at 39–40° C saturated with water vapour and secondly with room air at 23–24° C. In both experiments a marked fall in heat elimination occurred; in the first experiment rectal and oesophageal temperatures rose by 0·09 and 0·15° C respectively, whereas in the second the temperatures fell by 0·11 and 0·21° C respectively. This result agrees with the findings of Bolton *et al.* (1936) that the cutaneous vasoconstriction is unrelated to a fall in body temperature.

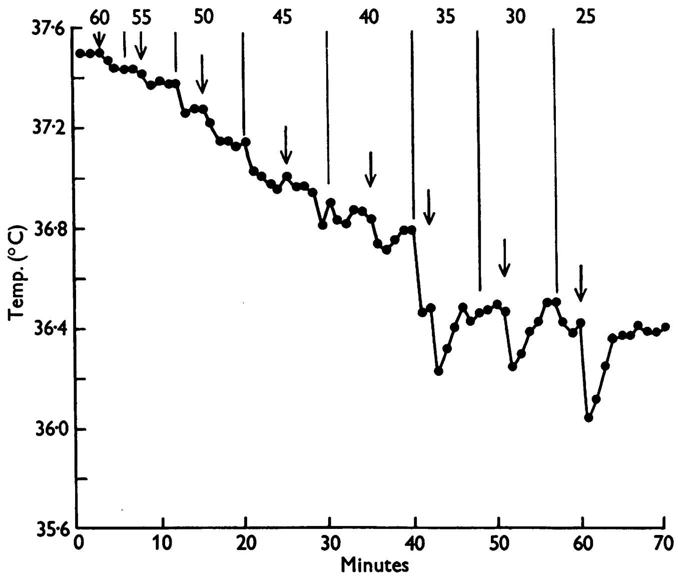

Fig. 6. The response of oesophageal temperature to hyperventilation. The figures above the graph indicate the position of the thermocouple in cm from the lips. Periods of hyperventilation for 30 sec are shown by the arrows.

Fig. 6 illustrates an experiment in which the thermocouple was introduced down the oesophagus to a depth of 60 cm from the lips and then withdrawn 5 cm at a time in eight steps. At each level hyperventilation with room air was performed for periods of 30 sec. At a depth of 60 cm the thermocouple junction registered a fall of 0·06° C during hyperventilation, with similar falls at 55, 50 and 45 cm. The temperature fall at 40 cm from the lips was 0·10° C, and increased to 0·28° C at 35 cm, 0·22° C at 30 cm and 0·39° C at 25 cm. Radiological examination showed that in this subject the trachea was in apposition to the oesophagus as far as a point 34 cm from the lips. This behaviour of temperature in the upper part of the oesophagus may be influenced by its contiguity with the trachea.

The influence of muscular activity

Sublingual, oesophageal and rectal temperatures were recorded during rhythmical tensing of the thigh and leg muscles, carried out vigorously but *without* moving the limbs, at rates of 20–30 contractions per minute for 2–5 min. Six experiments on three subjects produced falls in sublingual temperatures ranging from 0·08 to 0·22° C. In one experiment the rectal temperature rose 0·03° C and in one it remained unchanged; the remainder showed falls ranging from 0·06 to 0·10° C. The oesophageal temperature in four of the experiments on two subjects fell by 0·20–0·33° C. In four of the experiments the heat elimination from the hand was measured and, although it was initially low in all, it fell further, indicating a cutaneous vasoconstriction.

DISCUSSION

The temperature of a tissue depends on the temperature of the arterial blood delivered to it, the local heat production, the heat exchange with its surroundings and the rate of blood flow through it. These are the variables that may be presumed to determine the relationship one to another of the different tissue temperatures and their responses to experimental interference. The occasional finding of a fall in sublingual temperature during the onset of fever, whilst temperatures elsewhere were rising, can be reasonably explained only by a profound reduction of blood flow through the mouth. Sublingual temperature was not disturbed in this manner by stimuli producing more moderate cutaneous vasoconstriction or vasodilatation, although it is by no means certain that there was an associated change in oral blood flow. Grayson (1951) has adduced evidence that temperature in the rectum may be influenced by the rate of blood flow through it so that even in this structure changes in the temperature recorded may result from alterations in blood flow alone.

In the resting subject rectal temperature is higher than oesophageal which in turn is higher than oral. Tanner (1951) reported a temperature difference between the mouth and rectum of $0{\cdot}395 \pm 0{\cdot}02°$ C; our findings are in close agreement. The fact that there are different opportunities for heat loss from these two sites does not fully explain their temperature difference. There are indeed three series of observations indicating that rectal temperature is governed by factors which have not hitherto been widely appreciated. In the first place, the temperature measured simultaneously at different sites in the rectum has been found to vary by as much as 1·5° F (Mead & Bonmarito, 1949). This suggests that the temperature may be influenced by the surrounding structures. Secondly, rectal temperature has been shown in animals to be 0·22 and 0·21° C higher than the temperature in the left atrium and pulmonary artery respectively (Mather, Nahas & Hemingway, 1953); in man the temperature in the right ventricle was very similar to that in the femoral artery but these temperatures were $0{\cdot}23 \pm 0{\cdot}035°$ C lower than in the rectum (Eichna, Berger, Rader & Becker, 1951). It must be supposed that local heat production in the rectum or adjacent tissues is an important factor, or, alternatively, that the arterial blood supplying it must previously have been in contact with structures at a higher temperature. Thirdly, it has been found (Gerbrandy, Snell & Cranston, to be published) that induced changes in total body heat are reflected more quickly and more regularly by sublingual temperature than by rectal temperatures. The variations in cutaneous vasomotor tone are also correlated more highly with changes in oral temperature than with temperature changes in the rectum. These are very strong grounds for considering the rectal temperature to be a poor guide to the temperature elsewhere in the body and particularly to changes occurring at the central temperature receptors; in the latter respect rectal is certainly inferior to sublingual temperature.

Mild exercise without change in position leads to a fall of rectal, oesophageal and mouth temperature and to a fall in heat elimination from the hand. That an increase in total heat production and apparent decrease in total heat loss from the body should result in a fall in these temperatures is paradoxical. But there is a considerable increase in blood flow through the active muscles, with transfer of heat from the blood to the relatively cool muscles. A redistribution of body heat may accordingly account for the falls in oral, oesophageal and rectal temperatures in the early stages of exercise.

Temperature changes induced by posture are not so easily explained. Similar postural changes in rectal temperature were described by Kleitman & Doktorsky (1933) which they ascribed to alterations in heat production. Oxygen consumption in young women was shown to be approximately 16% greater standing than lying (Tepper & Hellebrandt, 1938) which appears insufficient to account for the magnitude and rapidity of the temperature changes. On lying down, there is an increased return of cool venous blood from the legs and on standing there is not only a decreased venous return but a reduction of heat loss from the legs consequent upon cutaneous vasoconstriction. The temperature changes may be related to these and other circulatory readjustments.

The effects of heat loss to the respired air are clearly shown by the overbreathing experiments. The fall in temperature in the upper part of the oesophagus was greater than that in its lower reaches. If the explanation of this temperature difference is found in the relationship of oesophagus to trachea, then it suggests that heat exchange may occur readily through the wall of the oesophagus. This raises the interesting possibility that temperature measurements of the aorta, left auricle and ventricle may be obtained from those parts of the oesophagus with which they are in close contact. The temperature difference between the lower oesophagus and rectum was found to be $0{\cdot}24 \pm 0{\cdot}02^\circ$ C which bears a close comparison with the finding (Eichna *et al.* 1951) that in man the rectal temperature was $0{\cdot}23 \pm 0{\cdot}035^\circ$ C higher than temperature in the right ventricle. As Bolton *et al.* (1936) showed, deep breathing is accompanied by a reduced skin blood flow that is independent of changes in body temperature. When the inspired air was at 37–38° C the cutaneous vasoconstriction was accompanied by an increase in oesophageal and rectal temperature.

It was frequently observed that strong sensory or emotional stimuli produced small transient falls in sublingual and rectal temperatures with reduction of heat elimination from the hand. These stimuli may cause hyperventilation or involuntary increase in muscle tone, and either or both of these factors may be responsible for the findings.

SUMMARY

1. In normal subjects at rest at room temperature of 19–24° C rectal temperature is $0{\cdot}35 \pm 0{\cdot}01°$ C higher than sublingual temperature when the lips are sealed.

2. Oesophageal temperature falls progressively from cardia to mouth, the chief change occurring at about 30 cm. Below this level oesophageal is intermediate between sublingual and rectal temperature.

3. Hyperventilation produces only a slight fall in rectal and sublingual temperatures but much greater falls in supralingual temperature and temperature measured in the oesophagus as far as the bifurcation of the trachea. The accompanying cutaneous vasoconstriction is not due to the fall of body temperature.

4. Standing is accompanied by a rise, lying by a fall in rectal, oesophageal and sublingual temperature.

5. Tensing of the leg muscles produces a fall in rectal, oesophageal and sublingual temperatures and a small vasoconstriction in the hand.

6. Mouth temperature rises during the transient vasoconstriction induced reflexly by cooling the skin; it falls sometimes during a powerful and persistent cutaneous vasoconstriction in the early stages of a pyrogenic reaction.

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