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THE INITIATION OF THERMAL POLYPNOEA IN THE CALF

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There is good evidence for an anatomically discreet 'heat-loss' centre in the hypothalamic region of the brain. Electrical stimulation of this area has been shown to simulate closely the effect of a sudden rise in environmental temperature, causing the rapid onset of polypnoea and cutaneous vasodilatation in the conscious goat (Andersson, Grant & Larsson, 1956), and cutaneous vasodilatation alone in the anaesthetized cat (Ström, 1950*b*). The temperature sensitivity of this centre has also been demonstrated (e.g. Magoun, Harrison, Brobeck & Ranson, 1938; Folkow, Ström & Unväs, 1949; Ström, 1950*a*, *b*), but it is not yet clear whether the centre responds to changes in the temperature of its own environment in the intact animal, or whether nervous excitation of peripheral origin can be an adequate stimulus to activate heat loss mechanisms. The assumption, based upon the temperature sensitivity of the 'heat loss' centre, that 'in the intact animal an increase in body temperature activities a co-ordinate heat loss mechanism including polypnoea or panting, cutaneous vasodilatation and sweating' (Folkow *et al.* 1949) is open to doubt. Randall, Deering & Dougherty (1948) have shown that the stimulus to sweating in man, and Kerslake (1955) has shown that the control of the rate of sweat secretion, can both be attributed entirely to peripheral temperature stimulation. Beakley & Findlay (1955*b*) showed that in the calf a moderate thermal stimulus of less than 30° C air temperature caused an increase in respiration rate unaccompanied by any rise in rectal temperature, and suggested that the stimulus to thermal polypnoea is also peripheral in origin.

This latter observation supports the conclusion of Sihler (1879, 1880) that the normal stimulus to panting in the dog must be entirely reflex from the periphery. However, most other studies of thermal polypnoea, while inconclusive, have tended to favour a central as well as a peripheral thermal component. Hammouda (1933), Vlcek (1937), Hemingway (1938), and Lim & Grodins (1955) have doubted the existence of a purely peripheral reflex, and

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conclude that the initiation of panting is a mixed phenomenon involving both an elevation of the temperature of the blood and nervous impulses of cutaneous origin. An attempt by Forster & Ferguson (1952) to observe the actual temperature in the central region of temperature regulation during the initiation of thermal polypnoea in cats, by means of thermistors implanted within the hypothalamic tissue, was inconclusive. With some animals a rise in hypothalamic temperature occurred before the onset of panting, while with others panting preceded a rise in temperature. There has, in fact, been no clear evidence that a change in the temperature of the blood supply to the brain must occur before thermal polypnoea is initiated.

In the experiments reported here the temperature of the blood in the bicarotid trunk of the unanaesthetized calf was kept under observation while the animal was subjected to a sudden increase in environmental temperature. It was then possible to determine whether or not the onset of polypnoea occurred before there was any change in the temperature of the blood supply to the brain.

METHODS

Three Ayrshire bull calves, 4–6 months old, were used. A polythene-clad copper-constantan thermocouple was inserted into a carotid artery so that the thermojunction lay below the point of bifurcation of the bicarotid trunk into the common carotid arteries. The thermocouple wires were brought to a terminal button sewn to the skin of the neck region. When the animal was taken into the climatic chamber, the thermocouple could be connected to the temperature-measuring system of the chamber, while at other times, the animal was able to move about freely in its loose-box. This technique has already been described (Bligh, 1957*a*, *b*).

The calf was taken into the chamber which had its temperature pre-set at 20° C. Skin temperature was measured on both flanks by means of fine copper-constantan thermocouples (Beakley & Findlay, 1955*a*), and respiration rate was recorded by a stethograph. Air temperature (T_A), bicarotid-trunk blood temperature (T_B), skin temperature (T_S) and respiration rate were recorded at intervals of 5 min throughout the experiment. After 60 min at 20° C the air temperature was raised as rapidly as possible (about 1° C/min) to 40° C, and maintained at this temperature for 100 min. This procedure was repeated six times on each animal.

After six experiments on each animal, a final experiment, following the same pattern, was conducted under pentobarbitone sodium anaesthesia (20 mg/kg intravenously for induction and 4 mg/kg/hr intravenously for maintenance). At the conclusion of this experiment, the calf was killed and the position and condition of the bicarotid-trunk thermocouple was determined. In each case, the couple was found to be lying in free-flowing blood in the bicarotid trunk, close to the point of origin of the vertebral arteries, and free of any blood or fibrin clot.

RESULTS

The mean values for all eighteen experiments are shown in Fig. 1*A*. There was no upward change in T_B during the first 30 min after T_A was raised. T_B then started to rise quite slowly (about 0·36° C/hr). On the other hand, both T_S and respiration rate had started to increase within 5 min of T_A being raised. Respiration rate increased from 38 to 106/min during the first 30 min and there was no alteration in the rate of increase when T_B started to rise

slowly. The individual experiments did not differ from this over-all picture in any essential point. Fig. 1*B* shows the results for one individual experiment as an example. In some instances, the onset of panting was delayed for as long as 15 min after the rise in T_A, but even so the increase in respiration rate preceded any rise in T_B. Sometimes there was even a slight fall in T_B coincident with the increase in respiration rate. With one animal on one occasion the climatic chamber experiment was made before the thermocouple was placed in the bicarotid trunk, and the surgical procedure was shown to have no effect upon the initiation of panting.

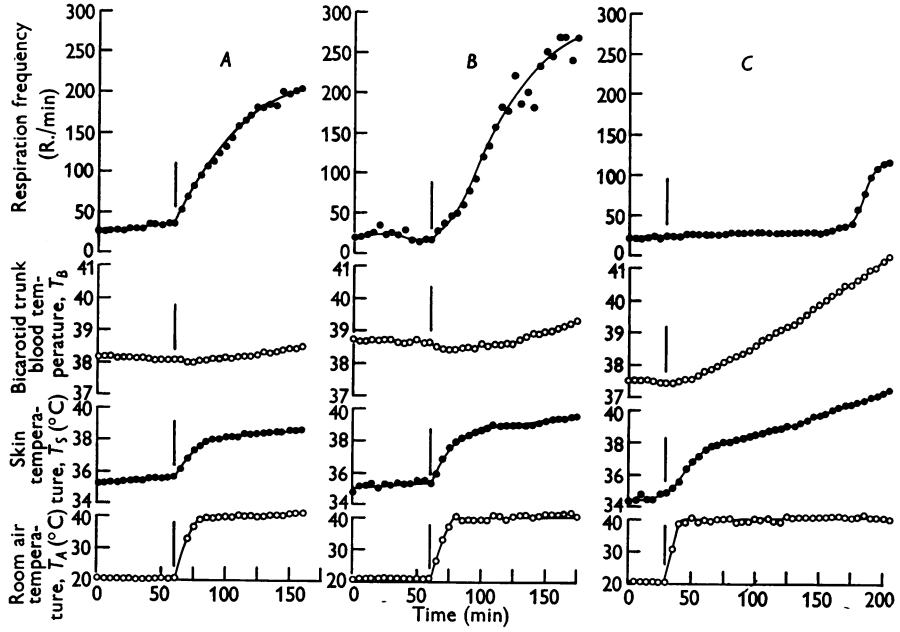

Fig. 1. (*A*) The onset of thermal polypnoea in the unanaesthetized calf; the mean obtained from eighteen experiments (six on each of three animals). (*B*) The onset of thermal polypnoea in the unanaesthetized calf; a typical single experiment. (*C*) The onset of thermal polypnoea in the anaesthetized calf; a typical experiment. The vertical lines indicate the points at which room air temperature starts to rise.

The terminal experiments performed under pentobarbitone sodium anaesthesia gave results in marked contrast to those obtained from the unanaesthetized animal. A typical result is shown in Fig. 1*C*. When T_A was raised, T_B started to rise almost immediately and continued to rise throughout the experiment at the rate of about 1·4° C/hr, but there was no change in respiration rate until T_A was about 40·5° C when a sudden increase in respiration rate occurred.

DISCUSSION

In some studies a distinction has been made between the initiation of thermal polypnoea and the onset of panting. In others, the two terms are regarded as synonymous. This is confusing as it is not always clear whether the moment to which body temperatures are being related is that at which respiration rate starts to increase, or the moment, which varies according to definition, at which the already polypnoeic animal is deemed to be panting. This study is concerned with the thermal stimulus which causes respiration rate to rise abruptly from a previously steady level; the point of initiation is taken to be that at which respiration rate starts to rise.

It is clear from these experiments that the initiation of thermal polypnoea in the unanaesthetized calf can occur before there is any upward change in the temperature of the blood supply to the hypothalamic centres. By inference, the necessary thermal stimulus is entirely peripheral in origin. This conclusion is in general agreement with that of Sihler (1880), who showed that the stimulus to panting in the conscious dog acts peripherally, and of Richet (1898) who also found no rise, and sometimes even a slight fall, in rectal temperature when the unanaesthetized dog started to pant.

Richet further showed that in the anaesthetized dog, there was no rise in respiration rate until the rectal temperature reached 41·7° C. The behaviour of the anaesthetized calf is similar. There was no increase in respiration rate until T_B reached 40·5° C but at that temperature panting began. In both the unanaesthetized dog and calf respiration rates have been observed to reach peak values long before such levels of deep body temperature were attained. Direct temperature sensitivity of the hypothalamic centre could have played no part in the normal initiation of thermal polypnoea. Hemingway (1938) objected that, under anaesthesia, the direct temperature sensitivity of the C.N.S. may be depressed, but Sihler (1880) seems to have covered this point when he showed the dependence of the peripheral stimulus upon nervous pathways to the C.N.S. He divided the cord of the conscious dog at the lower cervical level, and then heated the trunk and lower limbs. Panting then no longer occurred until the deep body temperature had risen to about 42° C which is close to the temperature at which panting starts in the anaesthetized dog.

The temperature at which thermal polypnoea is initiated by the direct application of heat to the hypothalamic 'heat loss' centre is also considerably higher than normal deep body temperature. Magoun *et al.* (1938) found it necessary to heat this area in the dog to between 40·5° and 43° C in order to induce panting. Folkow *et al.* (1949) obtained peripheral vasodilation in the cat by raising the hypothalamic temperature by 0·5–1·0° C but this did not cause panting.

There has been no report of a rise in deep body temperature to 40° C or more before the onset of polypnoea in the conscious animal. Most recent investigators have concluded that the thermal stimulus to panting, while largely peripheral, is probably not entirely so (Hammouda, 1933; Vlcek, 1937; Hemingway, 1938; Lim & Grodins, 1955; Andersson *et al.* 1956). However, the evidence for the existence of a direct central thermal component in the initiation of polypnoea is based upon quite small changes in rectal temperature which in some instances have preceded an increase in respiration rate (Hemingway, 1938) or which have occurred before an already polypnoeic animal started to 'pant' (Andersson *et al.* 1956). It is also based on the inconclusive observations of Forster & Ferguson (1952) that in five of seven cats the hypothalamic temperature increased by up to 1·25° C before panting started, while in the other two cats panting occurred before there was any rise in hypothalamic temperature. Against this evidence are the observations of Sihler (1880), Richet (1898) and Hammouda (1933) that thermal polypnoea started before there was any rise in rectal temperature.

It does not follow that, if the thermal stimulus to panting is entirely peripheral, the initiation of thermal polypnoea will always precede any rise in deep body temperature. This occurs only when the peripheral stimulus is adequate. In a preliminary experiment on the calf, when room air temperature was raised quite slowly the point of onset of polypnoea was not then sharply defined, and the temperature of the blood supply to the brain had started to rise before respiration frequency had deviated sufficiently from its previous level to be called polypnoeic. However, there is no reason to assume that this increase in deep body temperature was instrumental in the onset of polypnoea. When the increase in environmental temperature is gradual, accommodation of the temperature sensitive nerve endings may prevent an adequate stimulus to panting from reaching the C.N.S. until after the progressive decline in non-evaporative heat loss channels has caused a rise in deep body temperature.

The conclusion must be not only that the thermal stimulus leading to polypnoea is entirely peripheral in the calf, but also that there is no clear evidence for a central as well as a peripheral component to the stimulus in other panting mammalia which have been studied. The thermal stimulus to polypnoea in the dog is almost certainly without a deep body component, despite the contrary views of some recent investigators.

An advantage of an entirely peripheral stimulus to thermal polypnoea is that it permits the animal to increase the evaporative heat loss from the respiratory system as soon as an adverse increase in environmental temperature takes place, and before there is any appreciable rise in the heat content of the body. In this way the rapid onset of thermal polypnoea serves as a buffer mechanism which prevents any sudden rise in deep body temperature, rather than an adjusting mechanism to counter such a change.

In the terminal experiments conducted under anaesthesia the response to peripheral stimulus was completely abolished. The bicarotid trunk blood temperature started to rise almost as soon as the air temperature was raised, although heat production must have been considerably less than in the conscious standing animal. This supports the suggestion that the rapid onset of polypnoea serves to prevent the increase in the deep body temperature.

SUMMARY

1. A thermocouple was implanted chronically within the bicarotid trunk of calves, and the animals exposed to a rapid increase in environmental temperature from 20 to 40° C.

2. A marked increase in the frequency of respiration took place before there was any increase in the temperature of the blood supplying the brain.

3. It is concluded that the thermal stimulus responsible for panting acts entirely at the periphery.

4. The view is expressed that the same may be true of other mammals that pant when exposed to heat stress.

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REFERENCES

ANDERSSON, B., GRANT, R. & LARSSON, S. (1956). Central control of heat loss mechanisms in the goat. *Acta physiol. scand.* **37**, 261–280.

BEAKLEY, W. R. & FINDLAY, J. D. (1955*a*). The effect of environmental temperature and humidity on the skin temperature of Ayrshire calves. *J. Agric. Sci.* **45**, 353–364.

BEAKLEY, W. R. & FINDLAY, J. D. (1955*b*). The effect of environmental temperature and humidity on the respiration rate of Ayrshire calves. *J. agric. Sci.* **45**, 452–460.

BLIGH, J. (1957*a*). The relationship between the temperature in the rectum and of the blood in the bicarotid trunk of the calf during exposure to heat stress. *J. Physiol.* **136**, 393–403.

BLIGH, J. (1957*b*). A comparison of the temperature of the blood in the pulmonary artery and bicarotid trunk of the calf during thermal polypnoea. *J. Physiol.* **136**, 404–412.

FOLKOW, B., STRÖM, G. & UNVÄS, B. (1949). Cutaneous vasodilatation elicited by local heating of the anterior hypothalamus in cats and dogs. *Acta physiol. scand.* **17**, 317–326.

FORSTER, R. E. & FERGUSON, T. B. (1952). Relationship between hypothalamic temperature and thermoregulatory effectors in unanaesthetized cat. *Amer. J. Physiol.* **169**, 255–269.

HAMMOUDA, M. (1933). The central and reflex mechanism of panting. *J. Physiol.* **77**, 319–336.

HEMINGWAY, A. (1938). The effect of rate of heating and environmental temperature on panting threshold temperatures of normal dogs heated by diathermy. *Amer. J. Physiol.* **122**, 511–519.

KERSLAKE, D. McK. (1955). Factors concerned in the regulation of sweat production in man. *J. Physiol.* **127**, 280–296.

LIM, P. K. & GRODINS, F. S. (1955). Control of thermal panting. *Amer. J. Physiol.* **180**, 445–449.

MAGOUN, H. W., HARRISON, F., BROBECK, J. R. & RANSON, S. W. (1938). Activation of heat loss mechanisms by local heating of the brain. *J. Neurophysiol.* **1**, 101–114.

RANDALL, W. C., DEERING, R. & DOUGHERTY, I. (1948). Reflex sweating and the inhibition of sweating by prolonged arterial occlusion. *J. appl. Physiol.* **1**, 53–59.

RICHET, C. (1898). Regulation of temperature by respiration. *Dictionnaire de Physiologie*, **3**, 175–181.

SIHLER, C. (1879). On the so-called heat-dyspnoea. *J. Physiol.* **2**, 191–201.

SIHLER, C. (1880). Some further observations on heat-dyspnoea. *J. Physiol.* **3**, 1–10.

STRÖM, G. (1950*a*). Influence of local thermal stimulation of the hypothalamus of the cat on cutaneous blood flow and respiratory rate. *Acta physiol. scand.* **20**, Suppl. 70, 47–76.

STRÖM, G. (1950*b*). Vasomotor responses to thermal and electrical stimulation of frontal lobe and hypothalamus. *Acta physiol. scand.* **20**, Suppl. 70, 83–112.

VLCEK, J. (1937). Sur la polypnée thermique réflexe. *C.R. Soc. Biol., Paris*, **126**, 637–640.