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THE PATTERN AND CONTROL OF SWEATING IN THE SHEEP AND THE GOAT

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SUMMARY

1. The pattern and control of sweating in one breed of goat and six breeds of sheep have been studied.

2. Heat exposure of both sheep and goats resulted in periodic discharges of moisture on to the surface of the skin of the shorn mid-side. The frequency of discharge showed considerable variation between individual animals, varying from less than 1/hr to 14/hr. Approximate counts of the number of active sweat glands suggested that the same glands were involved at each discharge.

3. The amount of moisture produced at each discharge declined upon continued heat exposure. The rate of decline was independent of the frequency of discharge.

4. Adreno-medullary denervation had no effect on the pattern of sweating in either the sheep or the goat.

5. Intravenous adrenaline administration (5 μg/kg body wt.) caused the sweat glands to discharge, but noradrenaline had no effect at the same dose.

6. Thermal sweating was inhibited by bethanidine and phenoxybenzamine but not by propranolol. Sweating induced by intravenous adrenaline administration was blocked by phenoxybenzamine but not by bethanidine or propranolol.

7. It is concluded that sweating on the mid-side of the sheep and goat is controlled by an adrenergic mechanism, that secretion from the adrenal medulla under conditions of mild heat stress does not stimulate the glands and that sweating is mediated by adrenergic α-receptors.

INTRODUCTION

The sweat glands of both the mid-side (Bligh, 1961) and the scrotum (Waites & Voglmayr, 1963) of sheep respond to a heat stimulus and to the intravenous administration of adrenaline. Waites & Voglmayr (1963) also

demonstrated that the response of the scrotal sweat glands to a heat stimulus is much reduced by sympathetic denervation, and concluded that sweating in this species is controlled by an adrenergic mechanism. Kimura & Aoki (1962) came to a similar conclusion about the sweat glands of the goat. However, the role of the adrenal medulla in heat-induced sweating in these species has not been ascertained.

Findlay & Robertshaw (1965), using specific autonomic blocking drugs, together with adreno-medullary denervation, were able to show that sweating in the ox is controlled by an adrenergic mechanism and that although adrenaline is a potent sudomotor stimulus the adrenal medulla is not involved in heat-induced sweat gland activity. This was confirmed by Robertshaw & Whittow (1966), who showed that exposure to a hot, dry environment was not associated with any change in the level of plasma catecholamines although sweat gland activity was high.

The experiments reported in this paper were designed to examine, using autonomic blocking drugs, the mechanism of sweating in the sheep and goat and to assess the role of the adrenal medulla, using the technique of adreno-medullary denervation. Large differences in the pattern of sweating were noted between the various individuals that were studied.

METHODS

Two adult castrated male and three female British Saanen goats were used, and also two castrated male sheep of each of the following breeds: Suffolk, Kent, Welsh Mountain, Scottish Blackface, Hampshire and Cheviot. Each animal was exposed at intervals of not less than 48 hr in a climatic chamber (Findlay, McLean & Bennet, 1959) to air temperatures of 20, 30 or 40 °C at a vapour pressure of 9–10 mm Hg for 2 hr. On the day of experiment the morning feed was withheld until after the experiment. Changes in cutaneous moisture loss were detected by means of a ventilated capsule (McLean, 1963), using a flow rate of 0·7 l./min, placed on an area of shaved skin overlying the 8th and 9th ribs and approximately midway between the thoracic spinous processes and the sternum. Results were expressed as the difference in temperature between the two wet bulb thermocouples (Δ° C). The appearance of water on the skin surface was also detected visually using the starch-iodine method of Wada (1950).

All the drugs were administered through a polyethylene cannula inserted aseptically into either the right or left jugular vein on the day of experiment. In two animals (one sheep and one goat) bilateral adreno-medullary denervation was performed at a single operation by a method similar to that described for the ox by Findlay & Robertshaw (1965). Surgery was performed 8–12 weeks before experiments began. All surgical operations were carried out aseptically under thiopentone sodium and halothane anaesthesia.

The drugs used in these experiments were adrenaline hydrochloride, (−)-noradrenaline bitartrate, isoprenaline sulphate, bethanidine sulphate, phenoxybenzamine hydrochloride and propranolol hydrochloride.

RESULTS

Effect of heat exposure

Goats. Five experiments were performed on each of the five animals used. A small but constant amount of moisture evaporation was detected after fixing the capsule on to the skin (e.g. the separation between the dotted lines over minutes 0–20 in Fig. 1). In all the animals tested, upon exposure to an environment of 40° C, there occurred, after varying intervals of time, a sudden increase in skin moisture loss which quickly returned to the initial level. Areas of skin treated with starch and iodine, as described by Wada (1950), showed the sudden appearance of a large number of blue dots coincident with the sudden increase in cutaneous moisture evaporation. Further increases in moisture loss occurred at fairly regular intervals, but the amount of moisture produced declined until the initial rate of moisture loss was achieved and further discrete discharges were not detectable after 60–80 min. An intravenous injection of 5 μg adrenaline/kg body wt. at this time had no effect on cutaneous moisture loss. Figure 1 shows the results from such an experiment. Approximate counts of the number of active sweat glands of adjacent areas of skin suggested that discharges after the initial discharge involved the same glands, since the number of active glands per unit area did not increase. The frequency of discharge of the sweat glands was similar in all the goats tested varying from 7 to 10/hr.

Sheep. Four experiments were performed on each of twelve animals. Sweat gland activity in sheep was basically similar to that seen in the goat but the frequency of discharge of sweat glands varied greatly between individual animals and between breeds. Figure 2 shows the cutaneous moisture evaporation of two animals exposed simultaneously to an environment of 40° C and was typical of the variation in the pattern of sweating observed. One animal had a pattern of sweat gland activity similar to that seen in the goat whereas the other animal did not show discrete discharges and sweating appeared to be continuous. However, further experiments performed in an environment of 30° C on this animal showed a pattern of sweating similar to that seen in other animals, in that individual discharges could be detected (Fig. 3). Figure 3 also shows that the onset of sweat gland activity occurred, in some cases, as soon as the animal entered the climatic room.

All animals showed a decrease in the amount of moisture produced at each discharge. The frequency of discharge varied from less than 1/hr to 14/hr with a mean of 4·9/hr.

Effect of adreno-medullary denervation. Adreno-medullary denervation had no effect on either the pattern or frequency of sweat gland discharge in either the sheep or the goat.

Effect of drugs. Each animal was exposed once to an environment of 20° C. This did not result in any spontaneous discharge of moisture on to the surface of the skin. Noradrenaline (5 μg/kg body wt.) given by single

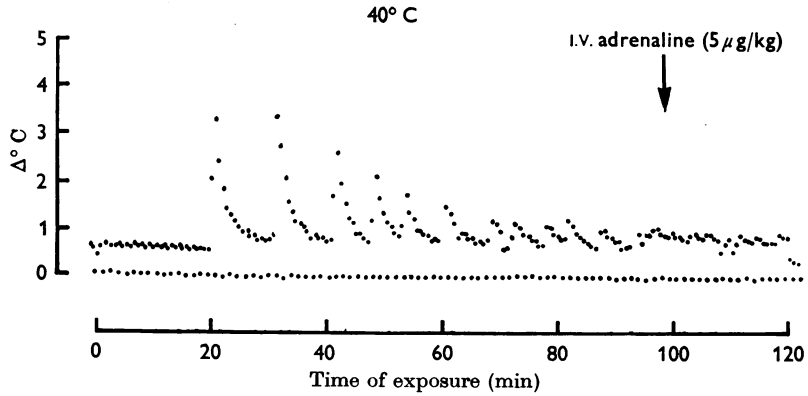

Fig. 1. Record of cutaneous moisture loss (Δ° C), recorded at 40 sec intervals from the mid-side of a goat exposed to an air temperature of 40° C and humidity of 9–10 mm Hg. Δ° C is the difference between the outputs of the wet bulb thermocouples in the test and the reference air streams (upper and lower dotted lines).

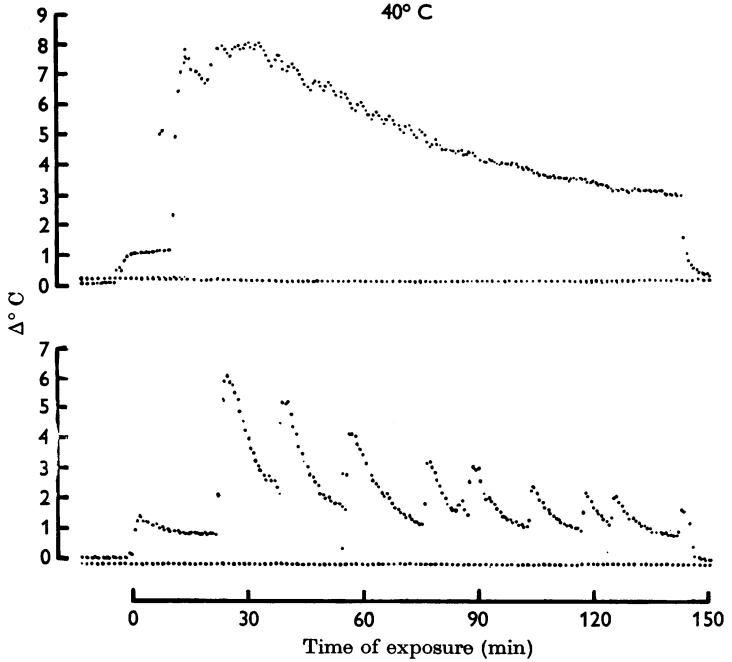

Fig. 2. Simultaneous records of cutaneous moisture loss (Δ° C) from the mid-side of a Suffolk sheep (upper record) and a Welsh Mountain sheep (lower record) exposed to an air temperature of 40° C and humidity of 9–10 mm Hg.

intravenous injections had no effect on the sweat glands. Subsequent infusion of adrenaline (0·5 μg/kg.min for 30 min) did not result in changes in evaporative loss, but a single intravenous injection of adrenaline (5 μg/kg body wt.) produced a sudden increase in cutaneous moisture loss.

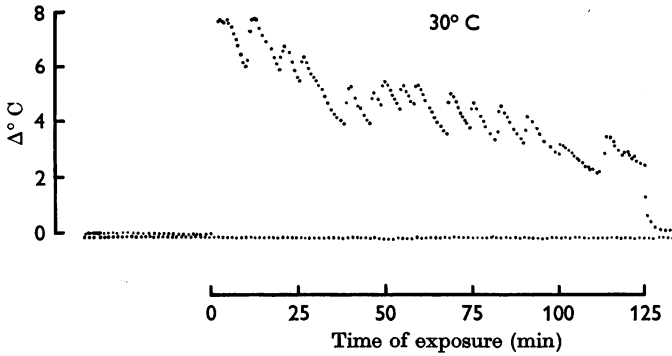

Fig. 3. Record of cutaneous moisture loss (Δ° C) from a Suffolk sheep (the same animal to which Fig. 2 refers) exposed to an air temperature of 30° C and humidity of 9–10 mm Hg.

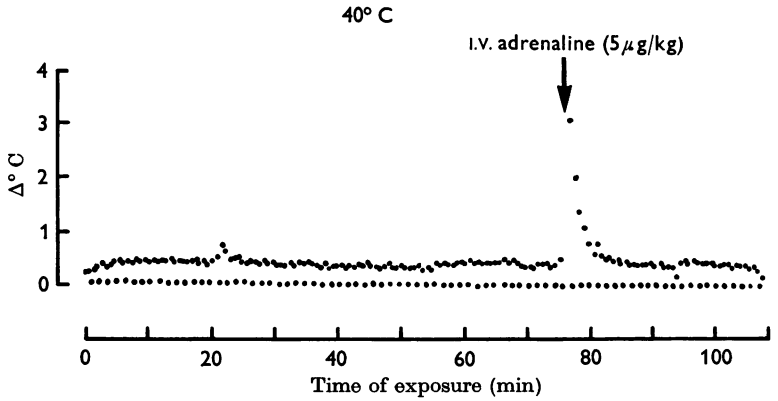

Fig. 4. Record of cutaneous moisture loss (Δ° C) from the mid-side of a goat exposed to an air temperature of 40° C and humidity of 9–10 mm Hg. Bethanidine (1 mg/kg body wt.) administered 1 hr before exposure.

Intravenous administration of 1 mg bethanidine/kg body wt. 1 hr before heat exposure inhibited sweat gland activity in twelve experiments on four sheep and two goats. Intravenous administration of 5 μg adrenaline/kg body wt., however, initiated an immediate discharge (Fig. 4). Noradrenaline (5 μg/kg body wt.) had no effect.

Intravenous administration of 3 mg phenoxybenzamine/kg body wt. 1 hr before heat exposure inhibited both heat-induced and adrenaline-induced sweating in one experiment on each of four sheep and two goats (Fig. 5).

Intravenous administration of propranolol (1 mg/kg body wt.) to two sheep and six goats immediately before exposure to 40° C had no effect on heat-induced sweating. This dose of propranolol completely abolished the increase in heart rate produced by a single intravenous injection of 0·3 μg isoprenaline/kg body wt.

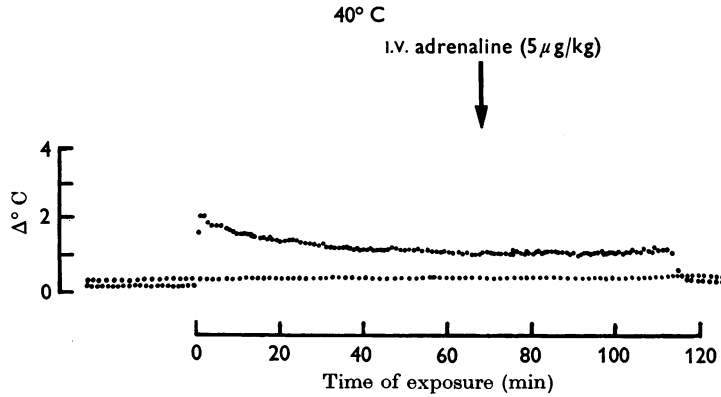

Fig. 5. Record of cutaneous moisture loss (Δ° C) from the mid-side of a goat exposed to an air temperature of 40° C and humidity of 9–10 mm Hg. Phenoxybenzamine (3 mg/kg body wt.) administered before exposure.

DISCUSSION

Bligh (1961) was the first to demonstrate conclusively that the sweat glands of sheep respond to a heat stimulus and that there is a synchronous discharge from all the sweat glands. The work of Kimura & Aoki (1962) suggests that the sweat glands of the goat may function in a similar manner and this has been verified in this work. Neither of these workers, however, noted a decrease in volume of sweat produced at each discharge in the presence of an unchanged thermal stress. Examination of the data of Waites & Voglmayr (1963) suggests that the scrotal sweat glands of the sheep do not show this phenomenon. Presumably, therefore, these glands are actively concerned in heat loss, whereas those on the remainder of the body surface contribute little to evaporative cooling because they are covered by fleece; furthermore the amount of cutaneous moisture declines with continued heat exposure. Sweat gland 'fatigue' has been noted in man but occurs usually in hot, humid conditions. Collins & Weiner (1962) suggest that the failure of sweating under these conditions may be due to obstruction of sweat gland ducts by excessive skin hydration although Peter & Wyndham (1966) claim to have shown that this is not so. Decline in activity under hot, dry conditions has not been observed in cattle. (D. Robertshaw, unpublished observations). Thus, the present experiments possibly provide the first demonstration of a decline in activity of sweat

glands under hot, dry environmental conditions. The fact that intravenous adrenaline administration was unable to stimulate the 'fatigued' glands after the activity had declined might suggest that the decline was due to metabolic exhaustion of the gland cells and not to failure of transmission at sudomotor nerve endings. Bligh (1967) has suggested that heat exposure or adrenaline administration results in myoepithelial contraction thereby emptying the glands of preformed secretion, there being no stimulation of glandular secretion. The decline in activity, therefore, would be due to the inability of the glandular cells to provide sufficient secretion for every contraction of the myoepithelial cells. However, it might be expected that within one animal the rate of decline would be related to the frequency of myoepithelial contraction. Examination of Figs. 2 and 3 where there is a marked difference in discharge frequency of the same animal shows that there is little difference in the rate of decline of moisture loss.

The work of Bligh (1961) was confined to three animals of the Welsh Mountain breed of sheep. The present experiments have examined five other breeds of sheep, including examples of breeds adapted to hill conditions, e.g. Scottish Blackface, and those adapted to lowland conditions, e.g. Hampshire. Since there was no obvious difference between the breeds, although there was a big variation between the sweat gland response of individual animals, it cannot be stated that breeding and adaptation for hill or lowland environmental conditions is associated with any difference in the pattern of sweating. The continuous pattern of sweating seen in one animal at 40° C (Fig. 2) probably reflected a very high frequency of discharge which could not be detected by the measuring system, since exposure to an environment of 30° C (Fig. 3) resulted in a pattern of sweating similar to that shown by other animals. Examination of Fig. 2 does in fact show, initially, the presence of discrete discharges. Since exposure to an ambient temperature of 30° C resulted in a lower frequency of discharge than exposure to 40° C then the response would appear to be related to the intensity of the stimulus. Bligh (1961) made a similar observation and also noted that the initiation of sweat gland activity was independent of a rise in the temperature of the blood supplying the brain. This is consistent with the observation made in the present experiments that on some occasions sweat gland activity commenced immediately the animal entered the room before any rise in body temperature could occur (Fig. 3).

Waites & Voglmayr (1963) showed that the scrotal glands of the sheep respond to the intravenous administration of adrenaline or noradrenaline at similar dosage rates. However, in the present experiments although 5 μg adrenaline/kg body wt. was an effective sudomotor stimulus the same dose of noradrenaline had no effect. Thus the scrotal sweat glands of the

sheep differ in one other aspect from those of the general body surface in that they are sensitive to noradrenaline.

The fact that bethanidine, an adrenergic neurone blocking agent (Boura & Green, 1963) inhibited heat-induced but not adrenaline-induced sweating, reveals that sweating in these species is controlled by an adrenergic mechanism. In the goat (Kimura & Aoki, 1962) and sheep (Waites & Voglmayr, 1963) it has been shown that sympathetic denervation considerably reduces sweat gland activity and it has been concluded that the sweat glands are controlled by adrenergic nerves. In these species, therefore, the control of the sweat glands is similar to that of the ox (Findlay & Robertshaw, 1965).

Neither Kimura & Aoki (1962) nor Waites & Voglmayr (1963) were able to assess the role of the adrenal medulla in heat-induced sweating. Bethanidine does not inhibit the release of adreno-medullary hormones (Boura & Green, 1963). One might expect, therefore, that in animals treated with bethanidine increased sweating as a result of increased adrenaline secretion by the adrenal medulla would be detected, since intravenous injection of adrenaline into these animals produced a sweat gland response. Also, animals in which the adrenal medulla had been denervated showed normal sweat gland activity. It thus appears that although adrenaline is a potent sudomotor stimulant in the sheep and goat, adreno-medullary secretion plays no part in normal heat-induced sweating. Findlay & Robertshaw (1965) and Robertshaw & Whittow (1966) came to a similar conclusion with respect to the sweat glands of the ox. Sweating in the sheep and goat is also similar to that in the ox in that it is mediated by α-receptors, since both heat-induced and adrenaline-induced sweating could be inhibited by the α-receptor antagonist, phenoxybenzamine and was not affected by the β-receptor antagonist, propranolol.

Thus the sweat glands of the mid-side of the sheep and goat are basically similar to those of cattle in that they are adrenergic.

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