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THE ROLE OF THE SYMPATHO-ADRENAL SYSTEM IN THE CONTROL OF SWEATING IN THE OX (*BOS TAURUS*)

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Muto (1916) reported that intravenous pilocarpine administration produced marked sweating in the calf, and concluded that the sweat glands of the ox were cholinergic. Ferguson & Dowling (1955) observed the formation of sweat in response to intradermal injections of adrenaline, and Taneja (1959) showed that Dibenamine inhibited this response and, in a hot environment, reduced the moisture loss from the skin. Findlay & Jenkinson (1964) confirmed that intradermal injections of adrenaline stimulated the sweat glands, and Ingram, McLean & Whittow (1963) showed that intravenous adrenaline injections markedly increased cutaneous moisture loss which they attributed to sweat gland activity. From their results on the effects of hypothalamic heating on cutaneous moisture loss Ingram *et al.* (1963) suggested that the control of sweat secretion in the ox was similar to that described by Evans (1957) for the horse, whose sweat glands have no sudomotor nerve fibres and are normally controlled by increases in plasma-adrenaline concentration or increases in cutaneous blood flow.

The experiments described here were undertaken to examine the threshold response of bovine sweat glands to intradermal injection of drugs, to examine the role of the sympathetic nervous system and assess the importance of the adrenal medulla in the control of thermal sweating in the ox, and to study the effects of drugs with specific autonomic blocking effects on sweat gland activity.

A preliminary report of this work has been published (Findlay & Robertshaw, 1964).

METHODS

Eleven castrated Ayrshire bull calves aged between 5 and 12 months were used. For the intradermal tests the animals were placed in a climatic room (Findlay, McLean & Bennet, 1959) at 15° C dry bulb (D.B.) temperature, 10° C wet bulb (W.B.) temperature. In the experiments where cutaneous moisture evaporation was measured the animals were exposed to conditions of 0/−1, 5/3, 10/5·5, 15/9·5, 20/13, 25/16·5, 30/18·5, 35/20·5 and 40/21 (D.B. ° C/W.B. ° C).

The animals were fed twice daily on a concentrate ration with hay and water *ad lib.* On

the day of experiment the morning feed was withheld until after the experiment. Cutaneous moisture loss was continuously recorded using the ventilated capsule method of McLean (1963*a*). Skin temperature measurements were continuously recorded from a spring-mounted 40 s.w.g. copper–constantan thermocouple within the capsule (McLean, 1963*a*). Measurements of rectal temperature were continuously recorded from a 40 s.w.g. copper-constantan thermocouple inserted 10 cm into the rectum.

The appearance of water on the skin surface was detected visually by the starch–iodine method of Wada (1950). Areas of unpigmented skin were clipped leaving the hair about 1 mm in length. At an air temperature of 15° C spontaneous sweating was not observed. Intradermal injections were given by 1·0 ml. tuberculin syringes, fitted with 26 s.w.g. 12 mm needles. The tip of the needle usually penetrated about 2 mm into the dermis. The

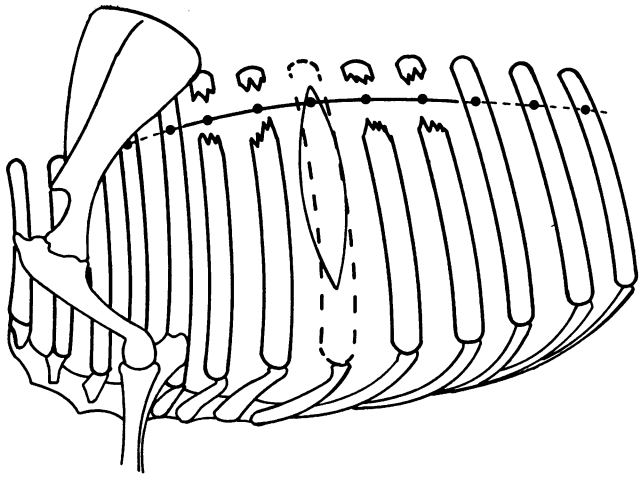

Fig. 1. Diagram showing the surgical approach for removal of thoracic sympathetic ganglia numbers 6–10. The interrupted line indicates the parts of the chain left intact and the continuous line represents the parts of the chain removed. The eighth rib, which was resected, is shown by the dotted outline.

solutions of the drugs were diluted with sterile normal saline (NaCl 0·9 g/100 ml.) and frequent control injections of normal saline were made throughout the experiments. In all the experiments 0·1 ml. of solution was administered, Drugs were administered through polythene cannulae inserted aseptically into either the right or left jugular vein on the day of the experiment. In some experiments atropine was injected intra-arterially through a polythene cannula inserted in the right carotid artery. The artery had been exteriorized (Robertshaw, 1963) at a previous operation.

In four animals thoracic sympathetic ganglia nos. 6–10 and the intervening chain were removed on the left side. The eighth rib was partially resected and the parietal pleura over the sympathetic chain cut to allow access to the ganglia and chain (Fig. 1). After the operation the hair over the shaved area was allowed to grow to the same length as that on the right side of the thorax. The area of skin that had been sympathetically denervated by this procedure could be determined by placing the animal in an environment of approximately 0° C and observing the area that failed to show piloerection. Histological examination of skin biopsies taken from the denervated area confirmed the effectiveness of the procedure.

In two of the animals which had undergone sympathetic denervation, right and left adreno-medullary denervation was performed. The right and left adrenal glands were

denervated at separate operations, the animals being allowed to recover completely between operations. On both sides the splanchnic nerve was cut and 0·5 cm of the nerve removed. Small branches to the adrenal gland from the lumbar ganglia were also removed. Before adreno-medullary denervation intravenous insulin administration (1 u./kg body weight) produced no clinical signs of hypoglycaemia. Insulin administered at the same dosage after adreno-medullary denervation resulted in convulsions or coma within 1 hr. This was used as a test of the physiological effectiveness of the denervation. All surgical operations were carried out aseptically under 'Fluothane' (2-bromo-2-chloro-1,1,1-trifluoroethane, I.C.I.) anaesthesia.

The drugs used during these experiments were adrenaline hydrochloride, L-noradrenaline bitartrate, isoprenaline sulphate, nicotine sulphate, acetylcholine chloride, methacholine chloride, carbachol chloride, neostigmine methylsulphate, pilocarpine nitrate, physostigmine salicylate, atropine sulphate, ephedrine hydrochloride, Oxytocin Injection B.P., bethanidine sulphate, phenoxybenzamine hydrochloride, propanolol hydrochloride and tolazoline hydrochloride.

RESULTS

Dermal gland responses to intradermal injections

Dilutions of the various drugs in normal saline ranging from 10 mg/ml. to 1 ng/ml. of the active constituent were made and 0·1 ml. injected intradermally. Threshold concentrations, i.e. the smallest concentrations evoking sweat gland activity, were thus determined and provided a rough assessment of the sensitivity of the sweat glands. Results from eight animals are summarized in Table 1. Injections of saline occasionally produced a positive response immediately over the injection weal. These could be distinguished from true glandular activity, which showed a definite pattern around the injection site and appeared about 5 min after injection.

TABLE 1. The effect of intradermal injection (0·1 ml.) of various drugs on the sweat glands of the ox

Sweat detection by the starch–iodine method of Wada (1950).

Drug	Threshold dosage (in 0·1 ml.)
Adrenaline	1–10 ng
Noradrenaline	10–100 ng to 10–100 μg*
Isoprenaline	0·1–1 μg
Ephedrine	Negative
Nicotine	100 μg*
Acetylcholine	Negative †
Methacholine	Negative †
Carbachol	Negative †
Pilocarpine	Negative
Neostigmine	Negative
Physostigmine	Negative
Oxytocin	Negative

* Piloerection, † Oedema.

Adrenaline, noradrenaline, isoprenaline. The threshold concentration of adrenaline for all the animals was 1–10 ng/0·1 ml. In five animals the threshold concentration for noradrenaline was 10 ng/0·1 ml. to 100 ng/

0·1 ml.; in two animals 0·1 μg/0·1 ml. to 1 μg/0·1 ml., and in one animal 10 μg/0·1 ml. to 100 μg/0·1 ml. Isoprenaline was tested in only four animals and the threshold concentration was 0·1 μg/0·1 ml. to 1 μg/0·1 ml. The response was essentially similar in all experiments, glandular activity being confined to the area over and around the injection weal and along the course of the cutaneous lymphatic vessels. Noradrenaline injections also resulted in piloerection showing a distribution similar to that of sweat gland activity. When 10 mg tolazoline had first been administered subcutaneously there was a marked reduction in the threshold concentrations of these drugs and in the magnitude of their pilomotor effects. Sympathetic denervation, two months previously, did not affect the threshold concentration of adrenaline.

Subcutaneous injections of 1 mg atropine under the site of intradermal injection had no effect on the action of these drugs.

Nicotine produced a positive response in three out of five animals at concentrations of 100 μg/0·1 ml. and was associated with piloerection. Animals showing a positive response failed to show the response with every injection.

Other drugs tested were acetylcholine, methacholine, carbachol, neostigmine, pilocarpine, physostigmine, ephedrine and oxytocin.

In concentrations up to 1 mg/0·1 ml. none of these showed any sudomotor or pilomotor activity. Acetylcholine, methacholine and carbachol all produced marked oedema around the injection site. Previous subcutaneous injection of atropine below the site of intradermal injection inhibited this oedematous reaction.

The effect of intradermal injection of oxytocin was examined because of the well-developed layer of myoepithelial cells present in bovine sweat glands (Goodall & Yang, 1952).

Cutaneous moisture loss and environmental temperature

In two animals measurements of cutaneous moisture loss were made at temperatures ranging from 0 to 40° C (D.B.). Capsules were placed on either side of the thorax in the same transverse plane and approximately 50 cm from the spinous processes of the thoracic vertebrae. Measurements were made 4–6 hr after entry of the animal into the room to ensure approximately steady-state conditions. Steady-state conditions were judged by constancy of moisture loss and of rectal temperature. The results from such an experiment are shown in Fig. 2. They confirm the findings of McLean (1963*b*) that at higher ambient temperatures cutaneous moisture loss increases with increasing ambient temperature. In one animal the increased moisture loss was apparent at 20° C and in the other animal it did not occur until 25° C Moisture losses at temperatures of 0–15° C were

essentially similar. These experiments demonstrated that evaporative losses from either side of the thorax are virtually identical, and this fact was used as justification for further experiments in which the left side of the thorax was sympathetically denervated, each animal acting as its own control.

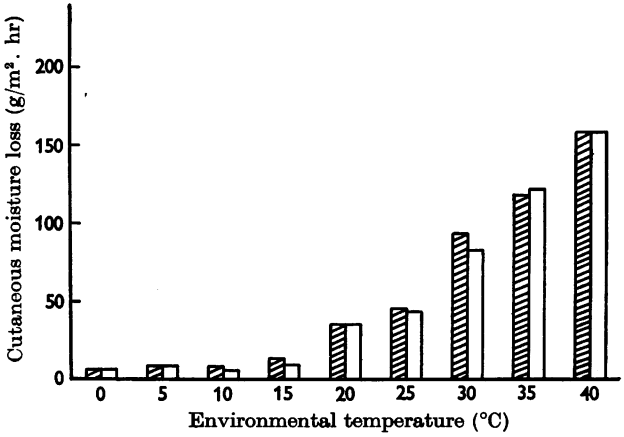

Fig. 2. Cutaneous moisture loss measured at various environmental temperatures in a normal animal. Shaded areas, left thorax; open areas, right thorax.

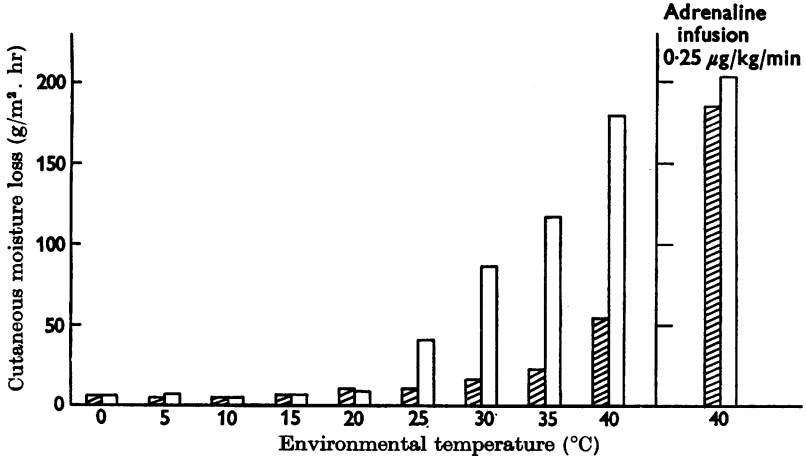

Fig. 3. Cutaneous moisture loss measured at various environmental temperatures from right (open areas) and left (shaded areas) sides of the thorax. The left thorax had been sympathetically denervated.

The effects of sympathetic denervation on cutaneous moisture evaporation

Two experiments were performed on each of four animals in which part of the left side of the thorax had been sympathetically denervated. Figure 3 shows the results from such an experiment. On the control (right) side

a marked increase in cutaneous moisture loss occurred at temperatures above 25° C from 9·0 g/m².hr at 15° C to 179·6 g/m².hr at 40° C. On the denervated (left) side, however, the cutaneous moisture loss increased from 8·0 g/m².hr at 15° C to 54·0 g/m².hr at 40° C.

In order to test that the glands on the denervated side were still functional, infusions of adrenaline or noradrenaline were made into animals subjected to an environment of 40° C. Adrenaline was infused at the rate of 0·25 μg/kg.min and the results of such an experiment are shown in Fig. 3. There was a marked increase in evaporative loss on the denervated side and a smaller increase on the innervated side. Noradrenaline was infused at a rate of 0·5 μg/kg.min and produced no effect on either the denervated or control areas.

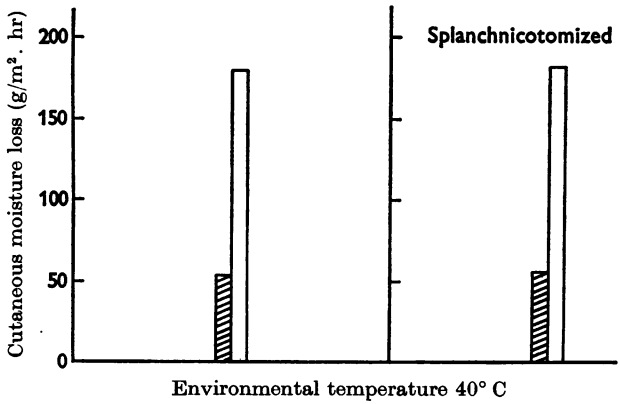

Fig. 4. Cutaneous moisture loss from the right (open areas) and left (shaded areas) sides of the thorax before and after splanchnicotomy. The left side had been sympathetically denervated.

The effects of adreno-medullary denervation

Cutaneous moisture loss was measured in two animals which had undergone a bilateral splanchnicotomy. Measurements were made in an environment of 40° C. Figure 4 shows the results from such an experiment. Splanchnicotomy had no effect on the cutaneous moisture loss from either the denervated or fully innervated areas of skin.

The effects of drugs

Three drugs which are known to have various actions on the sympathoadrenal system were tested. Each drug was tested on three occasions on two animals. An animal exposed to an environment of 30° C D.B. showed an increase in cutaneous moisture loss which usually reached a constant amount after 90 min. When adrenaline was then infused (0·25 μg/kg.min)

a further increase occurred which persisted during the infusion, and then declined afterwards (Fig. 5).

Bethanidine, an adrenergic-neurone blocking agent (Boura & Green, 1963), intravenously administered at a dosage of 1 mg/kg body weight, prevented the increase in cutaneous moisture loss caused by exposure to a temperature of 30° C but not that caused by adrenaline infusion (Fig. 5).

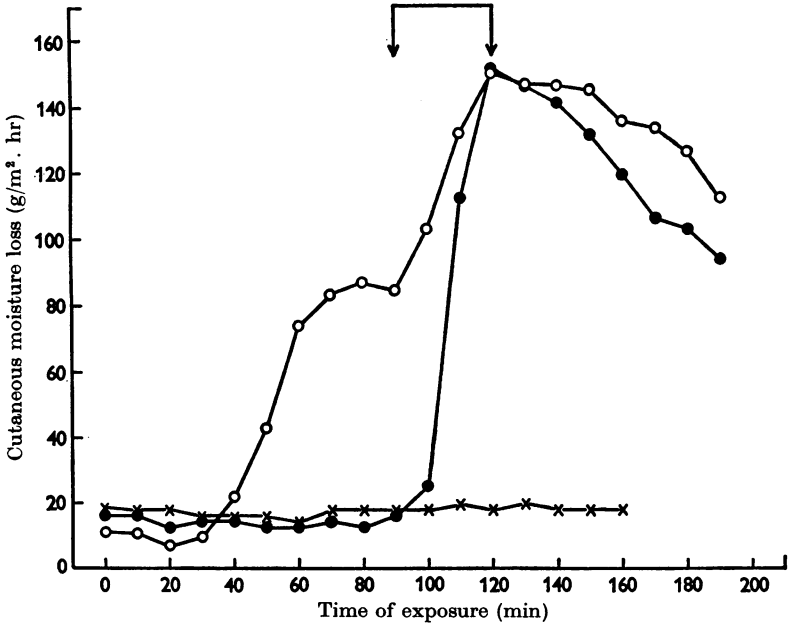

Fig. 5. Cutaneous moisture loss measured from the skin of the thorax of one animal exposed to an environment of 30° C; (○) control experiment; (●) after bethanidine (1 mg/kg); (– × –) after phenoxybenzamine (3 mg/kg); (↓‾↓) adrenaline infusion 0·25 μg/kg.min.

Phenoxybenzamine, an α-receptor blocking agent (Nickerson, 1959), intravenously administered at a dosage of 3 mg/kg body weight, prevented the increase in cutaneous moisture loss caused by exposure to a temperature of 30° C and that resulting from adrenaline infusion (Fig. 5).

Propanolol, a β-receptor blocking agent (Black, Crowther, Shanks, Smith & Dornhorst, 1964), intravenously administered at a dosage of 1 mg/kg body weight, had no effect on the increase in cutaneous moisture loss due to temperature or that resulting from adrenaline infusion. This dose of propanolol completely abolished the increase in heart rate produced by a single intravenous injection of 0·3 μg isoprenaline/kg body weight.

The intravenous or intra-arterial administration of 80 μg atropine/kg had no effect on either the heat-induced increase or the adrenaline-induced increase of cutaneous moisture loss.

Vapour pressure gradient across the skin

The vapour pressure gradient across the skin, i.e. the difference between saturation vapour pressure at skin temperature and ambient vapour pressure, is shown plotted for one animal (Fig. 6). The active secretion of water from normal skin is clearly seen while moisture loss from denervated skin is similar though reduced in amount and occurring at a higher vapour-pressure gradient. This higher vapour-pressure gradient is due to the higher skin temperatures of anhidrotic skin compared with normal skin. Phenoxybenzamine or bethanidine administration results in a lower cutaneous moisture loss than that occurring from denervated skin.

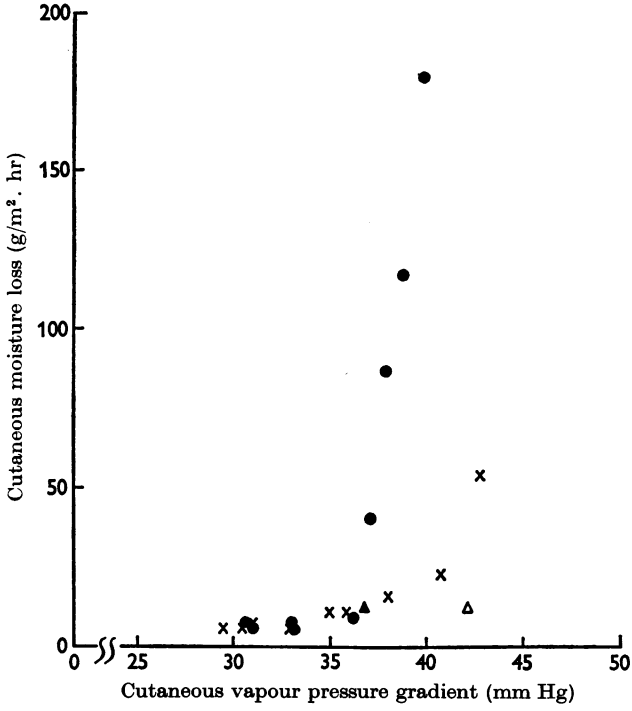

Fig. 6. The relation between cutaneous moisture loss and cutaneous vapour-pressure gradient at various environmental temperatures. All measurements are taken from one animal: (●), normal skin; (×), sympathectomized skin; (▲), after bethanidine administration; (△), after phenoxybenzamine administration.

DISCUSSION

The sweat and sebaceous glands of bovine skin have a common secretory orifice at the base of each hair. The intradermal tests, therefore, do not differentiate between the secretions of these glands. However, the formation of the blue starch–iodine complex requires the presence of water, and

similarly the capsule method of measuring moisture loss from the skin will detect only water vapour. The increases in cutaneous moisture loss occurring as a result of raising the ambient temperature or of the intravenous administration of adrenaline are greater than can be accounted for by diffusion of water across the skin and hence some active water secretion from the skin surface is postulated. Histochemical studies of bovine dermal glands have shown that the sebaceous glands contain much lipid material whereas the sweat glands contain none (Yang, 1952). It might be expected, therefore, that if the increases in cutaneous moisture loss were due to sebaceous gland activity then the moisture appearing on the surface would be rich in lipid material. Findlay & Jenkinson (1964) were unable to detect any lipid material in droplets of moisture appearing on the skin after intravenous adrenaline administration. McDowell, McDaniel, Barrada & Lee (1961) have shown that inactivation of bovine sweat glands by formaldehyde iontophoresis considerably reduces cutaneous moisture loss. It seems likely, therefore, that the increase in cutaneous moisture loss that occurs on raising the ambient temperature, or after intravenous adrenaline administration, is due to sweat gland activity. McLean (1963*b*) has shown that cutaneous evaporative heat loss accounts for 84 % of total evaporative heat loss at ambient temperatures of about 40° C. Bovine sweat glands would thus appear to have a heat dissipating function similar to those of man.

The sudomotor effect of intradermal or intravenous adrenaline administration observed here confirms the findings of previous workers (Ferguson & Dowling, 1955; Taneja, 1959; Findlay & Jenkinson, 1964). The glands also respond to noradrenaline and isoprenaline. However, higher concentrations of these drugs are required to elicit a response than is required for adrenaline. Taneja (1959) was unable to demonstrate a sudorific action of noradrenaline. However, he used the ventilated capsule method of sweat detection, which is much less sensitive than the starch–iodine method. In this respect, therefore, bovine sweat glands resemble those of the horse (Evans & Smith, 1956) but differ in their responses to the parasympathomimetic drugs. In man the threshold of stimulation of the forearm sweat glands to adrenaline and noradrenaline is about the same as that of the ox (Chalmers & Keele, 1951). However, the sudomotor action of adrenaline and noradrenaline in man is probably of little physiological significance since adrenergic blocking agents fail to inhibit thermally-induced sweating (Chalmers & Keele, 1952).

In common with all other species studied, except the horse, sympathetic denervation of the skin of the ox abolished thermal sweating. Evans & Smith (1956) have shown that, in the horse, increases in cutaneous blood flow are associated with increases in sweat-gland activity and it might be

argued that the anhidrotic state of bovine sympathectomized skin may be the result of the inability of the cutaneous vessels actively to vasodilate. If this were so, then atropine would abolish or considerably reduce sweat-gland activity and bethanidine would increase sweat-gland activity. Since these drugs did not display these effects sudomotor activity resulting from changes in blood flow seems unlikely.

If denervation had been complete, the evaporative loss from the denervated side at high environmental temperatures should have been a measure of diffusion of moisture across the skin. Diffusion is a physical process controlled mainly by the vapour-pressure gradient across the skin (Buettner, 1953). The upward trend of the moisture loss from denervated skin (Fig. 6) could be due to a decrease in diffusion resistance at higher temperatures and this is known to occur in human skin (Buettner & Holmes, 1959). However, phenoxybenzamine administration resulted in a much lower moisture loss at a similar vapour-pressure gradient. Unless phenoxybenzamine also depressed the decrease in diffusion resistance occurring at higher temperatures, some other explanation for the difference must be found. A more likely explanation is that there is some residual sweat gland activity. This could be caused by four possible mechanisms; incomplete denervation, overspill of transmitter substance from adjacent innervated areas, supersensitivity to circulating adrenaline or a rise in circulating adrenaline levels. The experiments on adreno-medullary denervation were designed to examine the role of circulating adrenaline of adreno-medullary origin. If the residual sweat-gland activity of sympathectomized skin had been due to an increase in the circulating levels of adrenaline, then adreno-medullary denervation would have reduced this and also possibly would have reduced the degree of moisture loss from the fully innervated skin. Similarly, any increase in sensitivity of the denervated glands such that they responded to the normal concentration of adrenaline in the blood would not be apparent because of the greatly diminished output of catecholamines from the denervated adrenal medulla and the consequent low circulating levels (Vogt, 1952). However, adrenal medullary denervation failed to reduce the cutaneous moisture loss from either the denervated or fully innervated skin. Therefore, it must be concluded that, under the conditions of these experiments, either there was no increase in adreno-medullary secretion sufficient to cause any sudomotor activity, or the postulated sweat-gland activity from the denervated skin was not due to an increased sensitivity consequent upon denervation. The denervated glands did not show an increased sensitivity to intradermal injections of adrenaline, a finding contrary to that of Kimura & Aoki (1962) in the goat. It would seem, therefore, that the residual sweat-gland activity is due to incomplete denervation or possibly overspill of transmitter

substance from adjacent areas. In man, thoracolumbar sympathectomy has often been associated with a small amount of retained sudomotor activity which can be reduced by anterior rhizotomy (Ray & Console, 1948). Intermediate ganglia outside the sympathetic chain offer an alternative pathway for sympathetic innervation to cutaneous structures, but intermediate ganglia in the mid-thoracic areas are rare (Boyd, 1957). Since only a partial sympathectomy was performed in these experiments, it is possible that some fibres originated from the adjacent innervated areas, especially as collateral sprouting is known to occur in the sympathetic nervous system (Murray & Thompson, 1957).

Transmission at the sudomotor nerve endings in the paw of the cat is known to be cholinergic (Dale & Feldberg, 1934) and is not affected by bethanidine administration (Boura & Green, 1963). However, in the ox, bethanidine abolished thermal sweating, although the sweat glands still responded to intravenous infusion of adrenaline, a result similar to that seen after sympathetic denervation. Since bethanidine blocks adrenergic neurones it would appear that the sudomotor nerves of the ox are adrenergic.

Phenoxybenzamine inhibition of both adrenaline-induced and heat-induced sweating suggests that sweat-gland activity is mediated by α-receptors; this suggestion is in agreement with the finding of Taneja (1959). Similarly, lack of any effect with propanolol demonstrates that there is no β-receptor component of sudomotor activity.

These experiments, therefore, suggest that sweating in cattle may be controlled by an adrenergic mechanism requiring a sympathetic nerve supply, and that under moderate degrees of heat stress with increases in rectal temperature of 1–1·5° C there is no adreno-medullary component. Even if the adrenal medulla were activated at high environmental temperatures, adrenaline secretion would have very little effect, as shown by the comparatively small increase in moisture loss that resulted from adrenaline infusion (Fig. 3). The sweat glands of the goat (Kimura & Aoki, 1962) and those of the scrotum of the ram (Waites & Voglymayr, 1963) are probably controlled by a similar mechanism. Those of the horse, however, appear to have no nerve supply and to be controlled entirely by circulating levels of adrenaline (Evans, 1957) of adreno-medullary origin (Usenik, 1957).

The sudomotor mechanism of the ox differs from that of other species. In man, cat and dog sympathetic denervation results in a gradual diminution of the responsiveness of the sweat glands to local stimulation (Weiner & Hellman, 1960). This is not so in the ox since we found that, one year after denervation, the sweat glands still responded to the same degree to intradermal or intravenous adrenaline administration. This might suggest that cholinergic sudomotor end units differ from those of the ox.

SUMMARY

1. Experiments have been undertaken to study the mechanism of sweating in the ox.

2. Intradermal administration of various drugs showed that adrenaline is the most potent sudomotor drug. Noradrenaline, isoprenaline and nicotine sulphate also produced sweating but only at higher concentrations. Tolazoline partially inhibited the response but atropine had no effect. None of the parasympathomimetic drugs or oxytocin had any sudomotor activity.

3. Sympathetic denervation considerably reduced thermal sweating but did not abolish the response to intravenous or intradermal adrenaline.

4. Adreno-medullary denervation did not reduce the moisture loss from sympathetically denervated skin or from normal skin at environmental conditions up to 40° C D.B., 21° C W.B.

5. Thermal sweating was abolished by bethanidine and phenoxybenzamine but not by propanolol. Adrenaline-induced sweating was blocked by phenoxybenzamine but not by bethanidine or propanolol.

6. It is concluded that sweating in cattle is controlled by an adrenergic mechanism requiring intact sympathetic nerves, and that under mild heat stress adreno-medullary secretion does not stimulate the sweat glands. Sweating is mediated by α-receptors, there being no β-receptor component.

7. The results are discussed in relation to sudomotor control in other species.

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