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THE RECEPTORS CONCERNED IN THE RESPIRATORY RESPONSE TO HUMIDITY IN SHEEP AT HIGH AMBIENT TEMPERATURE

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The onset of panting in sheep in response to a rise in ambient temperature can occur in the absence of any rise in deep body temperature (Bligh, 1959). The suggestion that the thermal stimulus to panting is derived from peripheral thermoreceptors located at some relatively naked area or areas has been substantially confirmed by Waites (1961, 1962), whose results indicate that a fall of 2° C in deep body temperature does not reduce the effectiveness of the peripheral thermal stimulus. This apparent absence of any modifying influence of a change in deep body temperature is in contrast to the evidence of the integration of peripheral and deep-body thermal stimuli in the control of thermal polypnoea in the dog (Vlcek, 1937; Lim & Grodins, 1955), the goat (Andersson, Persson & Ström, 1960) and the ox (Ingram & Whittow, 1961).

The respiratory response of sheep to a high ambient temperature is greater when the ambient humidity is high (Lee & Robinson, 1941; Riek, Hardy, Lee & Carter, 1950). Under such circumstances the reduced evaporative capacity of the inspired air prevents or delays the re-establishment of thermal equilibrium and the deep body temperature rises.

The experiments reported in this paper were done to see whether the further rise in respiratory frequency which occurs when a condition of high ambient humidity is superimposed upon an already high ambient temperature could be causally related to the concomitant rise in deep body temperature. If this could be demonstrated to be so, there would be clear evidence of the involvement of both peripheral and deep body thermal sensitivity in the control of panting in sheep.

METHODS

Castrated male Welsh Mountain sheep weighing 24–30 kg were used. A polythene-clad thermocouple was first implanted within the lumen of the brachiocephalic trunk. The thermocouple wires were brought out through the skin in the neck region to terminate on a 'Perspex' button (Bligh, 1957*b*, 1959) so that the temperature of the blood supplying the

brain could be kept under continuous observation in the post-operative experiments on the conscious animal. In some of the animals a polyethylene cannula was inserted into the abdominal vena cava through a femoral vein, or into the thoracic vena cava through a jugular vein at the same operation. The cannula was filled with heparinized saline, stoppered and left *in situ*. It was flushed daily and remained patent for 3 weeks or longer.

After allowing a few days for recovery from the surgical procedures, experiments were made daily with interruptions at week-ends. For each experiment the sheep was brought to the laboratory in a trolley so as to minimize effects of activity and disturbance upon metabolic rate. It was tethered standing within tubular steel stanchions in a small climatic chamber in which air and wall temperatures and ambient humidity were controllable. The implanted thermocouple was connected to the temperature-measuring system. Thermocouples were also attached to the skin beneath the fleece in one or more locations with rubber latex beneath a small thin rubber patch, the fleece being closed over the thermocouple. Respiratory frequency was recorded by means of a stethograph belt and tambour on an ink-writing kymograph.

A face-mask enclosing the naso-buccal area, with a continuous air supply the temperature and humidity of which could be controlled independently of the conditions of the chamber, was used in some experiments. Also, when required, the cannula in the vena cava was connected through a two-way tap to a continuous saline drip apparatus containing a pyrogen-free isotonic solution of sodium chloride, and to a thermally regulated supply of the same saline solution which could be infused at up to 60 ml./min by a Dale–Schuster pump. The temperature of the infused fluid was measured by a thermocouple at a point immediately before the cannula passed through the skin.

The dry-bulb and wet-bulb temperatures of the well-stirred air passing through the chamber, and of the mask air when the mask was worn, together with the temperatures of the brachiocephalic trunk blood, the skin beneath the fleece, and of the infused fluid, were recorded with an accuracy of 0·1° C at intervals of 5 min throughout each experiment. Respiratory frequency was also recorded for about 2 min out of every 5 min. All recordings were made at a point remote from the chamber, and the animal was left undisturbed. Each experiment lasted about 3 hr and during this time the animal had no access to food or water.

The basic experimental procedure was to keep the environment at $20 \pm 1°$ C for 30 min after the thermocouples and stethograph belt had been attached to the animal. Air and wall temperatures were then raised to 42° C over a period of 20 min, and these temperatures were maintained for a further 60 min, during which time the relative humidity was uncontrolled, but remained at about 20 %. The relative humidity of the air in the chamber was then raised abruptly to 75 % with no change in air or wall temperatures, and maintained at that level for 45 min. During the 60 min period at an ambient temperature of 42° C with low humidity, respiratory frequency increased asymptotically to 160–210 breaths/min, and this response was not necessarily accompanied by any change in the temperature of the blood in the brachiocephalic trunk (Bligh, 1959). The present experiments are concerned only with the effects of the increase in ambient humidity at a steady ambient temperature of 42° C. Variations in the experimental procedure were as follows:

Variations in humidity. In a series of 36 experiments on seven sheep the level of humidity during the final phase of each experiment was varied between 25 and 75 % r.h., the selected level of humidity being maintained for the full 45 min in each experiment.

Cold saline infusion. In 25 experiments on ten animals isotonic saline at 20–28° C was infused into the thoracic or abdominal vena cava during the first 17–30 min at high humidity (75 % r.h.). The rate of infusion was adjusted manually so as to prevent any rise or fall in the temperature of the blood in the brachiocephalic trunk. At the end of this period the infusion was stopped while the high humidity was maintained to the end of the experiment. Control experiments in which the same quantity of saline was infused into the vena cava at approximately the same rate, but at deep body temperature, were made on each animal.

Mask experiments. In a series of 6 experiments on four sheep the humidity of the chamber was raised to 75 % r.h. at 42° C while the air supply to the mask was maintained at 20 % r.h. at 42° C. In a converse series of 11 experiments on the same four sheep, the relative humidity was raised to 75 % in the mask, while that in the chamber, still at 42° C, remained low. Control experiments in which no mask was worn and the relative humidity raised to 75 % at 42° C were also made on each of these animals.

Warm saline infusion. In 15 experiments on five animals, the ambient humidity was not raised after 60 min at 42° C, but saline at about 48° C was infused into the vena cava at 40–60 ml./min during 20 min.

A post-mortem examination was made on each sheep to determine the condition and position of the implanted thermocouple and cannula. In each animal the thermocouple was found to have been correctly placed in the lumen of the patent brachiocephalic trunk.

RESULTS

A typical result of the abrupt rise in humidity from 20 to 75 % r.h. at 42° C ambient temperature is shown in Fig. 1. During the 45 min following the rise in humidity, brachiocephalic trunk temperature rose steadily from 39·6 to 41·4° C and respiratory frequency increased from 160/min to

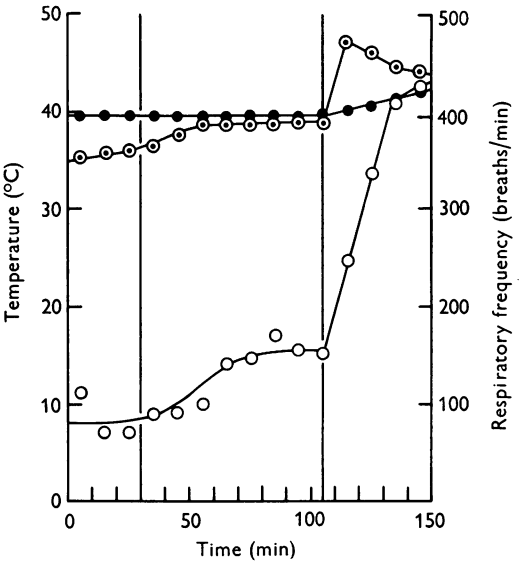

Fig. 1. Effect of high ambient temperature and humidity upon the respiratory frequency (○), brachiocephalic trunk temperature (●), and skin temperature at one point beneath the fleece on the back (⊙), of a Welsh Mountain sheep. At the first vertical line ambient conditions were changed from 22° C and 45 % r.h. to 42° C and 22 % r.h. At the second vertical line the ambient humidity was raised to 75 % r.h. with no change in ambient temperature.

420/min. Within 5–10 min of the increase in humidity the temperature of the skin beneath the fleece, as measured at one point on the back, rose from 39 to 47° C, and then slowly declined. In 50 similar experiments on

twenty-seven animals the mean rise in respiratory frequency was 192/min (S.D. ± 47) during the high-humidity period, and this was invariably accompanied by a steady rise in deep body temperature. Both the rise in respiratory frequency and in deep body temperature were of the same order when the experiment was repeated on the same animal, but the rate of rise, the peak value and the rate of decay of the subfleece skin temperature were quite unpredictable. This secondary rise in respiratory frequency could, if thermally induced, result from the stimulation of temperature-sensitive receptors, either at some deep body location or at the periphery.

Variations in subfleece skin temperature

In some experiments there was a rise of only 1–2° C in subfleece skin temperature when ambient humidity was raised. In other experiments the rise exceeded 12° C (Table 1), but there was no obvious relation between the changes in skin temperature and respiratory frequency. When up to 6 thermocouples were distributed over the skin beneath the fleece, it was found that, while the rise in temperature occurred over the entire fleece-covered surface, the variability of this response, already noted between animals and between experiments on the same animal, could occur to the same extent between positions in a single experiment. After close shearing, so that less than 2 mm of the wool fibres projected from the follicles, there was no rise in skin temperature when ambient humidity was raised, but the rise in deep body temperature and the increase in respiratory frequency were not modified by shearing.

The rise in subfleece temperature, which is very similar to that described by Bligh (1961*a*) when the sweat glands of the sheep discharge spontaneously and synchronously over the entire fleece-covered surface, is almost certainly due to the exothermic effect of moisture on wool (Hedges, 1926; King & Cassie, 1940; Speakman, 1944), as the rise also occurred when a piece of excised sheep skin with the fleece intact was subjected to the same change in relative humidity, but was absent following the removal of the fleece from both the living animal and the piece of excised skin.

The variability of this subfleece thermal response to humidity is no doubt a reflexion of such variable physical factors as the initial state of hydration of the fleece, its permeability to moisture and its thermal conductivity, which will determine the quantity and rate of heat production and its rate of dissipation and which are unlikely to be uniform.

The effects of different levels of humidity upon respiratory frequency and deep body temperature

Seven sheep were exposed to ambient relative humidities between 25 and 75 % at 42° C; some of the results of a typical series on one sheep are given in Fig. 2. Both brachiocephalic trunk temperature and respiratory frequency increased with ambient humidity. The relation between the humidity level and the respiratory frequency after 45 min, and between the rise in brachiocephalic trunk temperature and the respiratory frequency after 45 min (Fig. 3) were approximately linear.

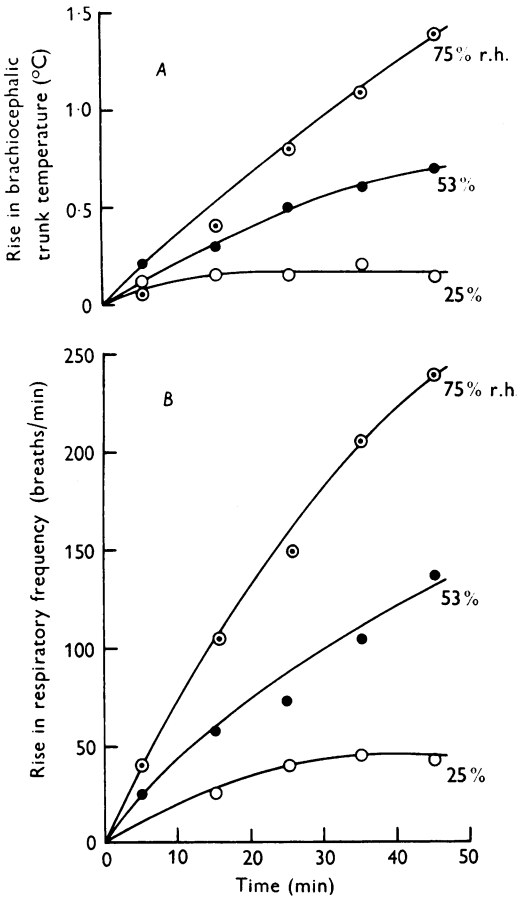

Fig. 2. 3 out of a series of experiments on one sheep in which the ambient humidity was raised from 20 % r.h. at 42° C to 25 % (○), 53 % (●) and 75 % r.h. (⊙) with no change in ambient temperature. *A* shows the rise in brachiocephalic trunk blood temperature and *B* shows the rise in respiratory frequency following the rise in ambient humidity.

This close relation between humidity and respiratory frequency could be the direct consequence of the rise in deep body temperature. However, the latter is an inevitable physical consequence of the humidity change and a causal relation between deep body temperature and respiratory frequency cannot be assumed.

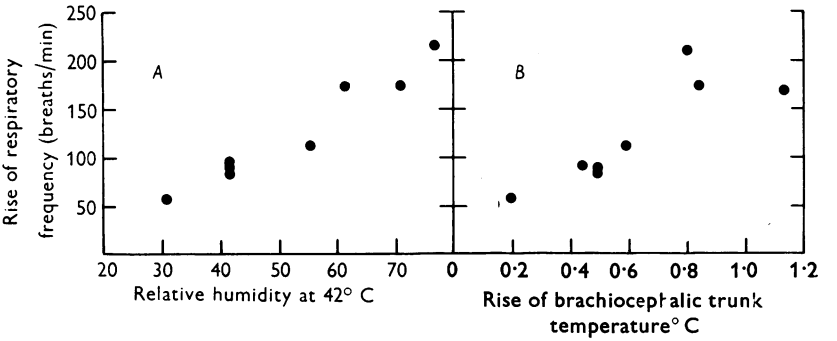

Fig. 3. A series of experiments on one sheep in which the ambient humidity was raised to various levels at 42° C (see Fig. 2). Ordinates: the rise in respiratory frequency after 45 min at the high humidity. Abscissae: *A* the relative humidity at 42° C ambient temperature; *B*, the rise in brachiocephalic trunk blood temperature during the same period.

Intravenous infusion of cold saline when ambient temperature and humidity were high

In 25 experiments on ten animals normal saline was infused into the vena cava at 20–28° C and at such a rate as to prevent any change in brachiocephalic trunk temperature during the first 20 min of the condition of high humidity. The wide variation between individual experiments in the pattern of respiratory frequency during this infusion period is indicated in Fig. 4, in which the two thin continuous lines represent the extremes of the respiratory responses obtained, and the thick solid line represents the mean response. Also shown is the mean respiratory pattern during control experiments on all ten animals, in which saline at deep body temperature (about 39–40° C) was infused into the vena cava at the same rate and under the same ambient conditions, and the mean respiratory pattern in experiments on the same animals in which there was no infusion of saline when the humidity was raised. The S.D. of the final values in these experiments are indicated.

The infusion of saline at deep body temperature caused no modification in the normal respiratory response to humidity. However, the infusion of cold saline produced this wide range of changes in the respiratory pattern. Sometimes the rise in respiratory frequency was of the order obtained in the control experiments, although there was no rise in brachiocephalic

trunk temperature. In other, seemingly identical, experiments there was a profound depression of respiratory frequency which was maintained during the infusion period. Whenever there was a depression or a diminished rate of rise of respiratory frequency during the infusion of cold saline, respiratory frequency increased rapidly to the normal high level under high humidity conditions as soon as the infusion of cold saline ceased.

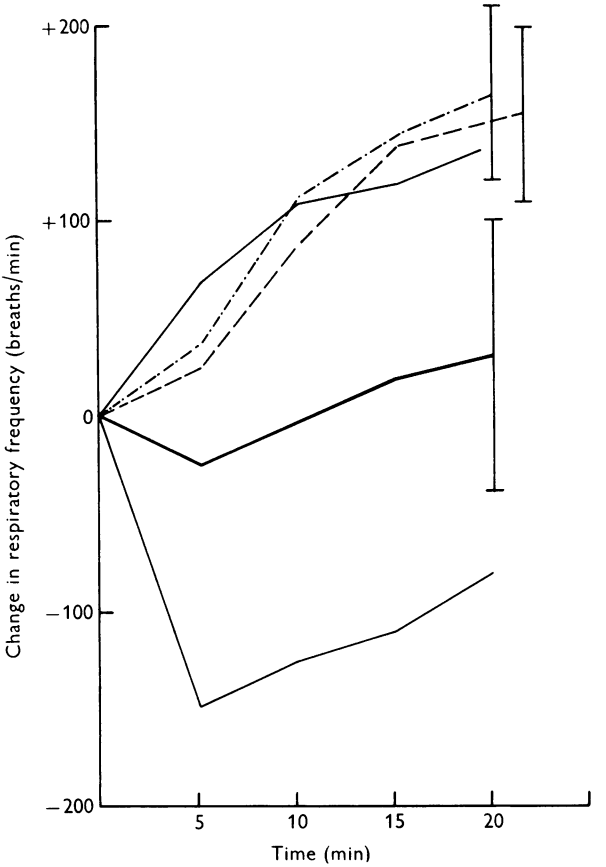

Fig. 4. The respiratory frequency of sheep at a high humidity (75 % r.h.) and an ambient temperature of 42° C while cold saline was infused into the vena cava at such a rate as to prevent any rise in brachiocephalic trunk temperature. Zero on the ordinate is the mean respiratory frequency during the 15 min immediately before the humidity was raised. The thin continuous lines represent two experiments in which the extremes of the range of respiratory responses were obtained. The thicker continuous line shows the mean respiratory response for 25 experiments on ten sheep. –·–·– the mean respiratory response when saline at deep body temperature was infused into the vena cava. ———, the mean response when no saline was infused in the same ten sheep when the humidity was raised. The standard deviations of the final mean values for the three types of experiment are indicated.

The quantity of saline infused varied between 550 and 980 ml. and the mean rate of infusion varied between 21 and 57 ml./min. The temperature of the infused fluid, measured by a thermocouple just before the cannula passed through the skin, varied between 20 and 28° C. There were brief variations in the temperature of the blood flowing through the brachiocephalic trunk, but on no occasion did these exceed 0·2° C. An examination of these variable factors, which are recorded in Table 1, shows that

TABLE 1. Data from the individual experiments in which cold saline was infused into the venae cavae of sheep during 17–30 min of the high-humidity period

Expt. no.	Quantity of infused fluid (ml.)	Duration of infusion (min)	Mean rate of infusion (ml./min)	Mean temp. of infused fluid (° C)*	Max. deviation of T_B† during infusion (° C)		Mean change in respiration frequency (resp./min)*	Maximum rise in T_{SK}‡ (°C)
					−	+		
14/1	825	25	33	24	0·2	0·15	+110	13·6
14/3	875	30	29	23	0·1	0·0	+ 96	9·4
15/3	856	30	28·5	22	0·0	0·15	+ 25	7·6
15/5	950	30	31·5	25·5	0·1	0·05	− 15	5·2
16/2	840	30	28	23	0·0	0·1	+ 43	7·7
16/4	950	30	31·5	23	0·2	0·0	− 14	10·4
16/10	625	30	21	24	0·1	0·0	− 8	15·5
17/4	910	25	36	20	0·0	0·15	− 58	4·7
17/6	933	22	42·5	20	0·0	0·2	− 93	8·1
18/2	975	25	39	—	0·05	0·05	−123	> 15
18/5	980	24	41	28	0·15	0·0	+100	3·4
18/7	950	25	38	27	0·05	0·0	− 5	6·0
18/8	950	18	53	26·5	0·0	0·0	+ 34	8·6
18/9	974	27	36	23·5	0·05	0·05	− 52	10·6
18/10	976	17	57	30	0·1	0·1	− 4	9·4
19/7	980	19	51·5	24·5	0·2	0·0	− 32	4·9
20/2	800	22	36	25	0·05	0·0	+ 64	4·0
20/4	550	19	29	26·5	0·0	0·05	+ 52	6·6
20/11	590	20	29·5	24	0·05	0·05	+ 42	3·4
21/4	600	17	35	24	0·1	0·0	− 30	7·9
21/7	847	20	42	24·5	0·1	0·0	+ 46	4·0
22/2	770	20	38·5	21·5	0·15	0·0	− 19	6·6
23/2	730	18	40·5	26	0·0	0·0	+ 37	5·3
23/4	710	18	39·5	25	0·0	0·0	+ 12	4·7
23/7	726	17	42·5	28	0·0	0·0	+ 42	4·5

* Mean of values recorded at 5 min intervals during infusion.

† T_B = Brachiocephalic trunk blood temperature.

‡ T_{SK} = Skin temperature beneath the fleece at one position of the back of the trunk.

the variation in the respiratory pattern during the infusion of cold saline cannot be related to variations in the rate of infusion, to the total quantity or the temperature of the infused fluid or to such transitory variations as occurred in deep body temperature. Nor can it be related to the location of the internal orifice of the infusion cannula, which in some animals was found at the post-mortem examination to be lying in the anterior vena cava 1–2 cm above the entry into the right atrium, and in other animals in the posterior vena cava just above the level of the renal veins, or at some point between these two extremes.

As the respiratory depression was removed within 1 min when infusion ceased, and as the infusion of an equal volume of saline at the same rate but at deep body temperature was without effect, it can be concluded that the respiratory depression was due to the low temperature of the infusion fluid and not to any other physical or chemical disturbance caused by the intravenous injection of a large volume of saline. This thermal effect could not be attributed to a local peripheral stimulus where the infusion cannula passed through the skin, as the perfusion with cold fluid of a polyethylene cannula which passed through the skin to the femoral vein and out again was without effect upon the respiratory response to the rise in ambient humidity.

Face mask with an independent air supply

When, at an air temperature of 42° C in both mask and chamber, the relative humidity of the chamber was raised to 75 % while that in the mask remained low, the mean rise in respiratory frequency at the end of 45 min was 124/min (S.D. ± 30), or 73 % of that obtained in the control experiment when no mask was worn (169 min ± 35 S.D.). During the same period brachiocephalic trunk temperature rose 0·2 ± 0·2° C S.D. compared with a rise of 1·6 ± 0·26° C S.D. in the control experiment (Fig. 5). The data from one mask experiment were omitted from the calculation of the mean rise in brachiocephalic trunk temperature because the rise was much greater than that which occurred in the other 5 experiments. In this experiment, however, the rise in respiratory frequency was below the mean value. It is clear from Fig. 6 that the magnitude of the respiratory response under these conditions was not dependent upon the magnitude of the rise in deep body temperature. In one experiment brachiocephalic trunk temperature fell by 0·05° C during this period, while the rise in respiratory frequency was slightly in excess of the mean value.

This respiratory response was apparently due to a humidity effect upon the general body surface, as neither the mask wet-bulb temperature measurements nor the behaviour of the brachiocephalic trunk temperature indicated a leak of high-humidity air into the mask. This effect could result from the rise in subfleece skin temperature or from some other as yet unrecognized effect of the humidity change upon the animal.

When the relative humidity was raised to 75 % in the mask, while that in the chamber remained low at 42° C, the mean rise in respiratory frequency at the end of 45 min was 190/min ± 36 S.D., which was not significantly different from that in the control experiments in which no mask was worn (169/min ± 35 S.D.). At the same time the rise in brachiocephalic trunk temperature was 0·7 ± 0·2° C S.D. which was only 44 % of that in the control experiments (1·6 ± 0·26° C S.D.). The rise in respiratory

frequency when the humidity was raised in the mask was not accompanied by any rise in the temperature of the skin beneath the fleece.

In these last experiments the full respiratory response to the rise in humidity must have depended upon some sensory factor other than the rise in either subfleece or central temperature. The response would appear to depend upon an effect of the humidity change upon the nasobuccal or upper respiratory surfaces enclosed within the mask.

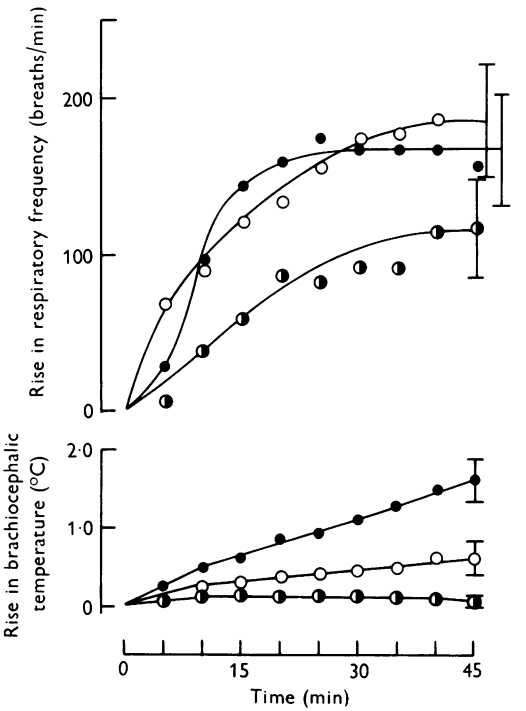

Fig. 5. Changes in respiratory frequency and brachiocephalic trunk temperature in experiments where a face-mask with an independent air supply was placed on the sheep. ●, mean values for control experiments on all four sheep in which no mask was worn when the humidity was raised. ○, mean values for all experiments on the four sheep in which the humidity was raised in the mask but not in the chamber. ◑, mean values of all experiments in which the humidity was raised in the chamber but not in the mask. The standard deviation of the final value is indicated for each series of experiments.

Intra vena-caval infusion of warm saline

In these experiments the ambient humidity was not raised, and ambient temperature remained at 42° C. The quantity of saline infused into the vena cava during 20 min varied between 700 and 1000 ml. The temperature of the infusion fluid, measured just external to the point where the cannula

passed through the skin, varied between 39 and 47° C. This temperature was, no doubt, modified by heat exchange with body tissues before the saline was discharged into the blood stream. The temperature of the brachiocephalic trunk blood was raised by up to 0·7° C during the infusion, and respiratory frequency was increased by up to 145 breaths/min. Under

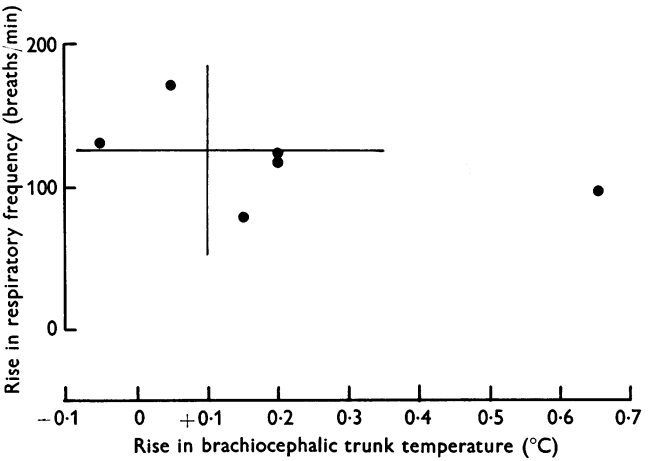

Fig. 6. Mask experiments on four sheep. Air in both chamber and mask maintained at 42° C. Humidity raised to 75 % in chamber alone. Abscissa: rise in brachiocephalic trunk after 45 min. Ordinate: rise in respiratory frequency during the 45 min of high humidity in the chamber. The thin lines indicate mean values, omitting values for the point at the extreme right of the graph.

the same conditions, the infusion of saline at deep body temperature (39–40° C) resulted in no change in brachiocephalic trunk temperature and no rise in respiratory frequency.

The rise in respiratory frequency at the end of the 20 min infusion period has been plotted against the rise in brachiocephalic trunk temperature in Fig. 7. There was apparently no relation between them.

DISCUSSION

A preliminary examination of the physiological changes which occur when a high humidity is superimposed upon an already high ambient temperature (Fig. 1) suggests that the further rise in respiratory frequency might be due to the additional peripheral thermal stimulus resulting from the abrupt and transitory rise in subfleece skin temperature, or to the inevitable rise in deep body temperature when the evaporative capacity of the inspired air is drastically reduced.

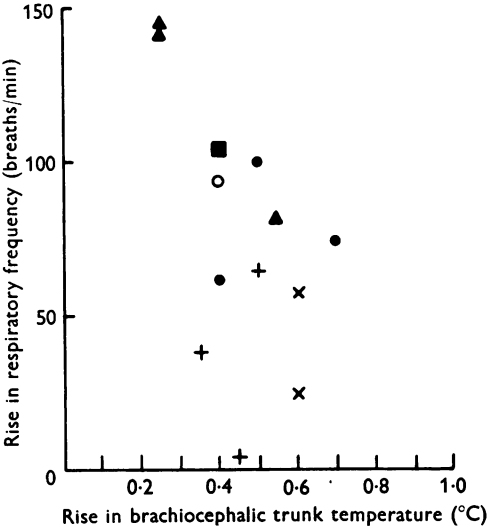

Fig. 7. Intravena-caval infusion of warm saline into the sheep at ambient conditions of 42° C and about 18 % r.h. Abscissa: rise in brachiocephalic trunk temperature at the end of the 20 min infusion period. Ordinate: secondary rise in respiratory frequency during this period. The various symbols indicate different animals.

The influence of subfleece skin temperature changes upon respiratory frequency

The rise in subfleece skin temperature, although transitory and variable, was often more than twice that which occurred when ambient temperature was raised from 20 to 42° C and where the increase in respiratory frequency has been attributed solely to the stimulation of peripheral receptors (Bligh, 1959). Waites (1961, 1962) has indicated that the peripheral warm receptors concerned in the control of panting are concentrated at specific and relatively naked areas and that there are few such receptors beneath the fleece. The rise in subfleece temperature in response to a humidity change, attributed to the exothermic effect of moisture upon wool, may not, therefore, give rise to a peripheral thermal stimulus equal to that when the surface temperature of the naked areas is raised to the same extent.

The conclusion that this rise in subfleece skin temperature is not an essential stimulus in the humidity-induced rise in respiratory frequency is supported by the observation that the removal of the fleece does not diminish the respiratory response to humidity, although there is then no rise in skin temperature. Furthermore, the normal respiratory response occurred when the humidity of the air supply to the mask alone was raised, although there was then no rise in skin temperature.

In the converse experiment, however, when the humidity was raised in the chamber but not in the mask, a substantial rise in respiratory frequency occurred and this was not necessarily accompanied by any rise in brachiocephalic trunk temperature. In these circumstances the rise in subfleece skin temperature may have served as an adequate peripheral thermal stimulus, although it must be concluded that such a stimulus is not an essential part of the normal stimulus to panting when the ambient humidity is raised.

The influence of deep body temperature changes upon respiratory frequency

Although the respiratory response was related to the rise in deep body temperature when the level to which ambient humidity was raised was varied, and although there was a marked rise in respiratory frequency when deep body temperature was raised by infusion into the vena cava of saline at a temperature in excess of that of the brachiocephalic trunk blood, it is not possible to attribute the respiratory responses to changes in ambient humidity solely to the rise in deep body temperature. When the humidity was raised in the mask but not in the chamber, the full respiratory response to humidity occurred, although the rise in deep body temperature was less than half that in control experiments (Fig. 5). When cold saline was infused into the vena cava at such a rate as to prevent any rise in brachiocephalic trunk temperature there was frequently a profound and sustained depression of respiratory frequency. On occasions, however, although there was no rise in deep body temperature, the rise in respiratory frequency when the ambient humidity was raised was within the range of values for control experiments. This indicates that the additional rise in respiratory frequency in response to the humidity change is not dependent upon change in deep body temperature, and that the magnitude of the respiratory response may not on occasions be influenced by the absence of a rise in deep body temperature.

The complex range of respiratory responses when cold saline was infused into the vena cava in order to eliminate the rise in deep body temperature when the ambient humidity was raised has already been discussed briefly (Bligh, 1961*b*). The depression of respiratory frequency which occurred in many of the experiments showed no correlation with any of the known variable factors, and it is concluded that the depression must be due to the low temperature of the infusate. As neither peripheral nor central thermoreceptors can be implicated, it is suggested that the influx of cold saline stimulated some cold-sensitive nerve endings located somewhere between the point of injection in the vena cava and the left side of the heart.

This explanation is similar to that offered by Blatteis (1960) for the shivering which resulted from the cold venous return from the hind limb of the dog when there was no change in brain temperature. The experiments of Hallwachs, Thauer & Usinger (1961) also indicate the existence of extra-cerebral sensitivity to cold in the deep tissues of the dog.

The possible influence of a sensory mechanism in the upper respiratory tract upon respiratory frequency

The regular relation between the change in ambient humidity and the rise in respiratory frequency must depend upon some stimulus other than either the rise in subfleece or deep body temperatures. In the mask experiments discussed above only the surfaces of the upper respiratory tract, including the naso-buccal areas, were exposed to the air at high humidity. The possibility of a process sensitive or responsive to changes in the humidity of the inspired air must be considered.

A possible mechanism could be an increase in the state of hydration of the epithelial cells acting mechanically upon interstitial pressure receptors. Another could be the effect of a rise in humidity of the inhaled air upon thermo-receptors located at the evaporative surfaces in the mouth and upper respiratory tract. It may be supposed that the temperature of these surfaces is depressed by evaporative cooling during panting and that a reduction in the evaporative capacity of the inspired air would result in a proportional rise in surface temperature.

Temperature-sensitive nerve endings similar to those described in the tongue of the dog (Hensel & Zotterman, 1951), if present in the sheep, could thus respond to changes in the humidity of the inspired air. In these circumstances the thermoreceptors would be behaving essentially as wet-bulb receptors. As the increase in respiratory frequency appears to be a thermoregulatory response to the change in humidity the latter hypothesis is preferred.

When the inspired air is heated without humidification there is no marked rise in respiratory frequency in the dog (Sihler, 1880), ox (Bligh, 1957*a*) or sheep (Bligh, 1959). However, the inspired air is warmed and humidified high in the respiratory pathway (McCutchen & Taylor, 1951; Cole, 1954) and the thermal effect upon the evaporative surfaces when the inspired air is raised from 20 to 42° C may be less profound than that which occurs when the humidity is raised at 42° C.

If these arguments are valid, it would be surprising if the humidity-sensitivity of the naso-buccal area was confined to the one species. A further rise in respiratory frequency when the humidity is raised at a high ambient temperature occurs in the ox (Beakley & Findlay, 1955) and the goat (Bligh, unpublished observation), while Lee, Robinson & Hines

(1941) and Robinson & Lee (1941*a*, *b*, *c*) have shown that the respiratory frequency of the rabbit, pig, cat and dog at a high ambient temperature is greater when the humidity is high, although the sensory factors have not been analysed in these other species.

From the experiments reported in this paper it appears that the linear relation between the humidity level and respiratory frequency is dependent upon a direct effect of the rise in humidity upon some sensory mechanism in the naso-buccal area or the upper respiratory tract. If this is so, it must follow that the other sensory components, although effective singly, are ineffective in competition with the stimulus arising in the buccal area and do not integrate with it to yield a greater respiratory response than results from the buccal stimulus alone.

It is concluded, therefore, that the rise in deep body temperature which accompanies an increase in humidity at a high ambient temperature is a consequence of the rise in the humidity of the inspired air, and that the concomitant rise in respiratory frequency is not causally related, even in part.

SUMMARY

1. When a condition of high relative humidity is superimposed upon an already high ambient temperature there is a further rise in the respiratory frequency of sheep which is regularly related both to the rise in humidity and the rise in deep body temperature. This is accompanied by an initial rise in skin temperature beneath the fleece which may exceed both deep body and ambient temperatures. The possible involvement of a deep body or peripheral thermal stimulus in bringing about this further rise in respiratory frequency has been examined.

2. The rise in subfleece skin temperature is attributed to the exothermic effect of moisture on wool fibres.

3. Experiments in which cold saline was infused intravenously to prevent the rise in deep body temperature show that the rise in respiratory frequency is not necessarily dependent upon a rise in deep body temperature.

4. A variable depression of respiratory frequency during the intravenous infusion of cold saline is attributed to the stimulation of temperature-sensitive nerve endings in or near the vena cava.

5. Experiments with a face-mask which allowed an independently conditioned air supply to the lungs confirm that the respiratory response to humidity is not tied to the change in deep body temperature, and also show that it is independent of the rise in subfleece skin temperature.

6. The linear relation between respiratory frequency and relative humidity apparently depends upon some sensory mechanism in the upper

respiratory tract, in the presence of which the cutaneous and deep body stimuli are without additional influence upon respiratory frequency.

7. It is suggested that buccal temperature receptors situated at evaporative surfaces may act as 'wet-bulb' receptors giving rise to a thermal stimulus related to the rise in the relative humidity of the inspired air.

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