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CHANGES OF ARTERIAL BLOOD PRESSURE AND HEART RATE IN THE OX (*BOS TAURUS*) WITH CHANGES OF BODY TEMPERATURE

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In the ox an increase of respiratory rate and minute volume occurs in response to localized heating of the hypothalamus (Ingram & Whittow, 1961, 1962*a*). Respiratory rate increases also when the skin is heated, in the absence of any change in the temperature of the blood in the bicarotid trunk or of the brain (Bligh, 1957; Findlay & Ingram, 1961). In addition, the respiratory rate and minute volume increase when the temperature of the blood in the bicarotid trunk, of the brain, and of the skin increase during exposure to high environmental temperatures combined with high humidity or infra-red irradiation (Ingram & Whittow, 1962*a*, *b*).

It has been suggested (Ingram & Whittow, 1962*a*) that these changes in respiratory activity are brought about by the separate or combined stimulation of central receptors in the brain and peripheral receptors in the skin. However, it is possible also that the responses are related, in part, to changes of arterial blood pressure which are known to alter respiration (Heymans & Bouckaert, 1930; Daly & Schweitzer, 1951; Aviado & Schmidt, 1955; Daly & Daly, 1957). In support of this possibility is the fact that increases in respiratory rate are usually associated with an increase in the skin temperature of the extremities (Ingram & Whittow, 1962*a*, *b*; Whittow, 1962). There is evidence that the increase in the skin temperatures of the extremities is brought about by an increased blood flow through the extremities (Whittow, 1962), and it is possible that the increased blood flow is associated with a change of arterial blood pressure. Furthermore, an increase in the skin temperatures of the extremities elicited by the administration of vasodilator drugs, which are known to cause changes of arterial blood pressure in the ox (Whittow, unpublished observations), results in stimulation of respiration (Whittow, 1962). There is also evidence that, in other species, heating the entire body or the blood going to the head, and electrical or thermal stimulation of the hypothalamus

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are accompanied by changes of arterial blood pressure (Wiggers & Orias, 1932; Rodbard, 1948; Newman & Wolstencroft, 1960; Wilson, Clarke, Smith & Rushmer, 1961). Further, Newman & Wolstencroft (1960) have postulated that stimulation of respiratory activity in the hyperthermic dog and calf might be secondary to a fall in blood pressure.

The present investigation was designed to find out if variations in the arterial blood pressure of the ox occurred with different forms of heating.

METHODS

Seven Ayrshire bull calves aged 8–12 months were used. They were exposed in a climatic room (Findlay, McLean & Bennet, 1959) to the following environmental conditions: (i) − 5·0° C, dry-bulb (DB) temperature; − 6·5° C, wet-bulb (WB) temperature. (ii) 10·0° C, DB; 8·0° C, WB. (iii) 15·0° C, DB; 11·5° C, WB. (iv) 20·0° C, DB; 13·5° C, WB. (v) 40·0° C, DB; 22·5° C, WB. During exposure to environments of − 5·0° C, 15·0° C and 40·0° C, the hypothalamus was heated to a temperature of 41·5° C for 3 min.

In separate experiments at environmental temperatures of 10·0 or 15·0° C four 250 W infra-red lights were shone on the rump and flanks of each animal as described previously (Whittow, 1962; Ingram & Whittow, 1962*b*).

In other experiments at an environmental temperature of 40·0° C the humidity of the air was increased to give a wet-bulb temperature of 38·5° C. The humidity was kept high until the body temperature of the animal was 41·8° C, when the humidity but not the dry-bulb temperature was reduced to its previous level.

In all experiments measurements were made of arterial blood pressure, heart rate and of the temperature of the hypothalamus. In most experiments respiratory rates were measured and in some of them respiratory minute volumes were measured also. The skin temperatures of both ears and both shanks were also measured except under hot, humid conditions.

Five animals had an indwelling radio-frequency (approx. 6 Mc/sec) heater and a thermocouple in the heat-sensitive region of the hypothalamus between the optic chiasma and the anterior commissure. These instruments were inserted aseptically, as previously described (Ingram & Whittow, 1962*a*) and the exact position of the thermocouple and electrodes was determined at autopsy. In addition, a polythene catheter (approximately 50 cm long) with a bore of 1·5 mm and an external diameter of 2·0 mm was inserted surgically into the left common carotid artery so that its tip lay in free-flowing blood in the bicarotid trunk. The catheter was brought out through the skin and attached by means of a Luer-Lok adaptor to a metal three-way tap which was mounted on a small Perspex plate. The Perspex plate was sewn to the surface of the skin of the neck. When pressure measurements were not being made a constant infusion of NaCl solution (0·9 g/100 ml.) containing heparin (5 i.u./ml.) was given at the rate of 30 ml./hr through the carotid catheter.

Measurements of arterial blood pressure were made by connecting the tap on the neck of the animal to a Statham P 23 Db pressure transducer by means of a catheter (approximately 90 cm long) filled with heparinized saline. The catheter had the same internal diameter as that implanted in the carotid artery. The transducer was rigidly mounted at the level of the animal's heart. The Statham transducer was used in conjunction with a MR 501 pre-amplifier (A.E.I.). The frequency response of this system was estimated to be linear ($\pm 5\%$) to at least 12 c/sec. Arterial blood pressures were either read directly from the dial of the pre-amplifier or they were displayed on a Cossor cathode-ray oscilloscope and a photographic record of the blood pressure wave taken. In view of the possible distortion of the blood-pressure wave form by the use of long catheters (Fry, 1960) reference is made only to mean blood pressures in this paper. Heart rates were counted from the records of arterial blood pressure.

Respiratory rates were measured by the counter described by Nisbet (1958). Respiratory tidal volumes were measured by means of a pneumotachometer (Nisbet, 1956). Skin and rectal temperatures were measured as previously described (Whittow, 1962).

RESULTS

Position of heating electrodes and thermocouple in the brain

A post-mortem examination of the brain of animal *F* could not be performed. With the exception of animal *A*, in which the electrodes were found to lie approximately 0·5 cm rostral to the optic chiasma, the tips of the electrodes were sited between the optic chiasma and the anterior commissure.

Effects of exposure to a cold environment (−5·0° *C*, *DB*; −6·5° *C*, *WB*)

When the animals were first exposed to this environment the skin temperatures of the ears and shanks decreased. The skin temperatures of the shanks continued to diminish until the end of the experimental period, which varied from 3 to 7 hr in different experiments. However, after exposure for approximately 20 min the skin temperatures of the ears increased by 10·0–25·0° C, and then diminished again. This change in the skin temperature of the ears occurred periodically throughout the experiment. Immediately before the initial increase in the skin temperatures of the ears there was an increase of mean arterial blood pressure, and an increase of heart rate (Fig. 1). The temperature of the hypothalamus sometimes increased slightly (0·1–0·3° C) before the skin temperature of the ears increased. When the skin temperatures of the ears increased the increase in the temperature of the hypothalamus was either arrested or the temperature diminished.

Effects of increasing the humidity of the air in a hot environment (40·0° *C*, *DB*; 22·5° *C*, *WB*)

After the wet-bulb temperature of the air had been increased to 38·5° C the temperature of the hypothalamus increased rapidly. The arterial blood pressure also increased, until the temperature of the hypothalamus was between 40·0 and 41·0° C. At temperatures higher than approximately 40·5° C there was little further increase of blood pressure, in seven out of nine experiments. The increase in mean blood pressure varied from 16 to 80 mm Hg in different experiments, the average increase being 40 mm Hg.

During the period of high humidity the heart rate increased progressively as described by Beakley & Findlay (1955). Respiratory rates also increased, but when the temperature of the hypothalamus was between 40·3 and 41·3° C, the respiratory rates began to decrease, and immediately

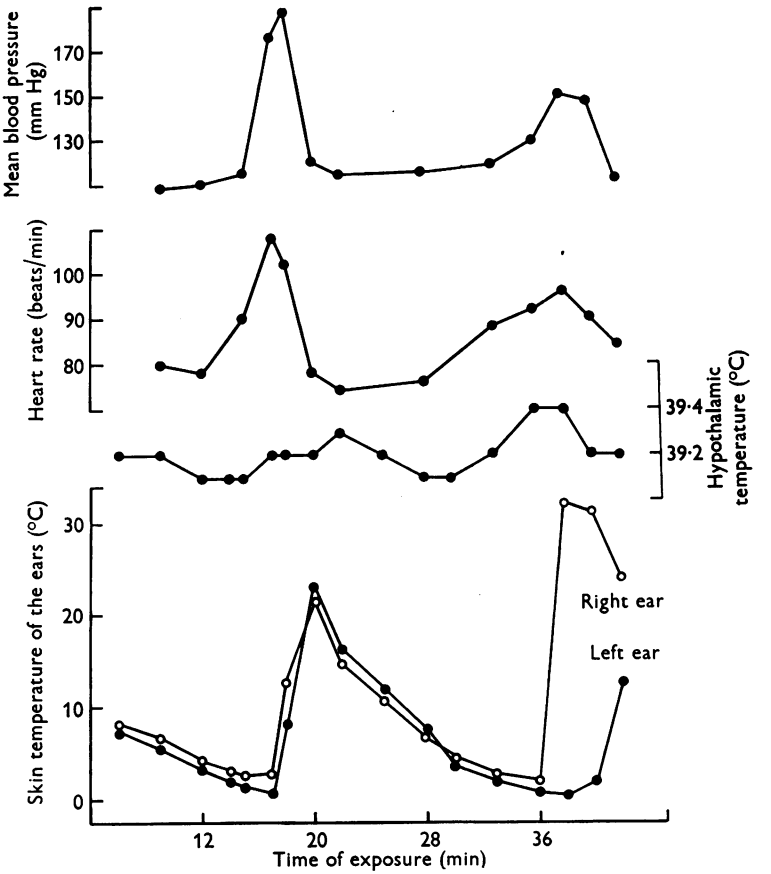

Fig. 1. Calf *A*, body wt. 141 kg. Changes in arterial blood pressure, heart rate and the temperature of the hypothalamus associated with spontaneous increases in the skin temperatures of the ears at an environmental temperature (DB) of −5° C.

TABLE 1. Effects of heating the hypothalamus to a temperature of 41·5° C for 3 min at different environmental temperatures. Mean results (±S.E.) of twenty experiments on four animals

Environmental temperature (° C, DB)	Temperature of the hypothalamus before heating (° C)	Mean B.P. (mm Hg)		Heart rate (beats/min)		Respiratory rate (respirations/min)	
		Before heating	Increase during heating	Before heating	Increase during heating	Before heating	Increase during heating
−5·0	38·2±0·26	130±2·6	20±4·3	103±4·9	14±5·8	18±1·6	129±13·9
15·0	38·5±0·15	123±7·3	29±11·3	111±5·7	20±3·4	35±4·3	115±14·9
40·0	39·0±0·29	129±5·9	38±12·3	115±8·8	44±14·8	163±12·4	38±12·0

before the animals were removed from the climatic room the decrease amounted to 13–58 beats/min. A similar reduction of respiratory rate has been observed before in calves (Findlay, 1957).

Effects of localized heating of the hypothalamus

The mean hypothalamic temperatures, blood pressures, heart rates and respiratory rates immediately before the hypothalamus was heated, together with the mean increases during heating, are given in Table 1. In all experiments in which it was measured the respiratory rate increased when the hypothalamus was heated.

Environmental temperature $-5{\cdot}0°$ *C, DB.* At an environmental temperature of $-5{\cdot}0°$ C heating the hypothalamus to a temperature of $41{\cdot}5°$ C for 3 min resulted in an increase of arterial blood pressure in eighteen out of nineteen experiments. In one animal (*B*) the response of the blood pressure to heating of the hypothalamus was diphasic, consisting of a transient increase followed by a diminution to, or below, the level before the hypothalamus was heated. The heart rate increased on fourteen out of nineteen occasions.

In twenty-six trials heating of the hypothalamus resulted in an increase in the skin temperatures of the shanks in only five instances. In these five instances the skin temperatures of the shanks were initially greater than $10{\cdot}0°$ C.

As described above, the skin temperatures of the ears increased spontaneously in a periodic manner at an environmental temperature of $-5{\cdot}0°$ C. The effect of heating the hypothalamus on the skin temperatures of the ears depended upon whether the skin temperature of the ear was increasing or decreasing spontaneously. When the skin temperature was increasing, heating the hypothalamus caused the temperature of the ear to increase to a level which was higher than that attained during spontaneous increases of skin temperature in the absence of hypothalamic heating. When the skin temperature of the ear was decreasing, heating the hypothalamus had no effect.

Environmental temperature $15{\cdot}0°$ *C, DB.* With the exception of animal *B*, in which a fall of arterial blood pressure was the predominant response to heating of the hypothalamus, the arterial blood pressure increased when the hypothalamus was heated at an environmental temperature of $15{\cdot}0°$ C. The heart rate increased in all (nineteen) experiments and the increase was greater than that which occurred at an environmental temperature of $-5{\cdot}0°$ C. When the skin temperatures of the ears and shanks were initially low, i.e. less than $20{\cdot}0°$ C, the skin temperatures increased when the hypothalamus was heated.

Environmental temperature 40·0° *C, DB.* The arterial blood pressure increased when the hypothalamus was heated at an environmental temperature of 40·0° C. In two animals (*B* and *C*) the mean blood pressure decreased by 11–18 mm Hg before it increased. The heart rate also increased, and the increase of heart rate was greater than that which occurred when the hypothalamus was heated at an environmental temperature of 15·0° C. At an environmental temperature of 40·0° C, the skin temperatures of the ears and shanks were high and heating the hypothalamus had little or no effect on them.

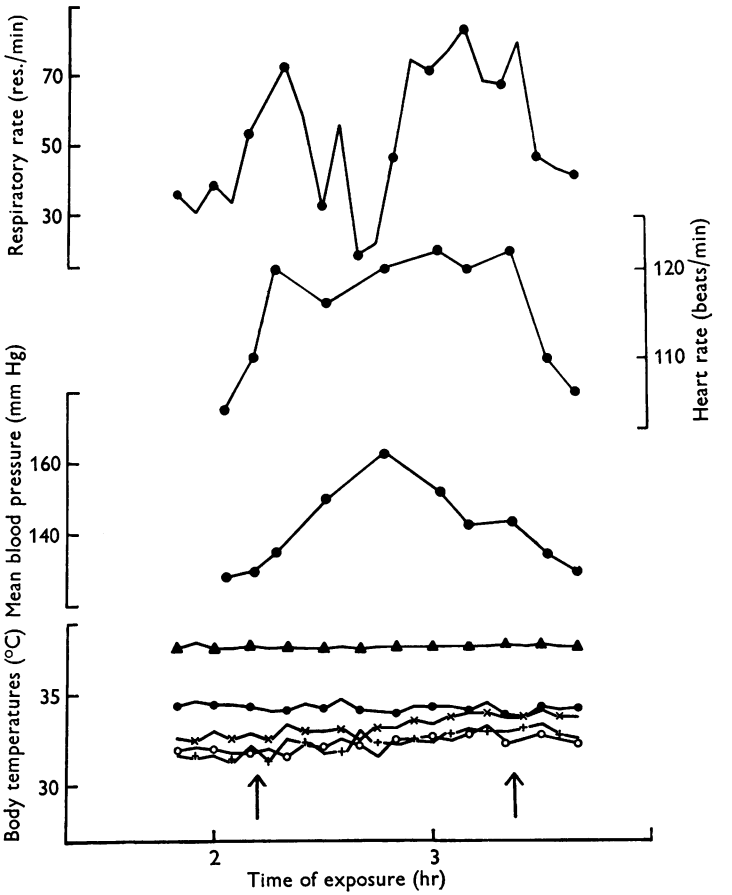

Fig. 2. Calf *B*, body wt. 162 kg. Effects of infra-red irradiation of the rump and flanks on the body temperatures, blood pressures, heart rates and respiratory rates. Infra-red irradiation was applied for the period between the two arrows. Environmental temperature 15·0° C, DB. Body temperatures: ▲, temperature of the hypothalamus; ●, skin temperature of the left ear; ○, skin temperature of the right ear; +, skin temperature of the left shank; ×, skin temperature of the right shank.

Response to localized infra-red irradiation of the trunk

Infra-red irradiation of the rump and flanks of the animals at environmental temperatures of 10·0 and 15·0° C resulted in an increase of arterial blood pressure and of respiratory rate, in all (seven) experiments. The heart rate usually increased also. At an environmental temperature of 10·0° C the skin temperatures of the extremities were initially low, in most instances, but they increased during infra-red irradiation. When the skin temperatures of the extremities increased, the increase in blood pressure was less than when the skin temperatures of the extremities were already high before infra-red irradiation. An illustration of the changes observed in one animal is given in Fig. 2. The temperature of the hypothalamus sometimes increased during infra-red irradiation but the increase in blood pressure also occurred when the temperature of the hypothalamus did not increase or even when it decreased.

DISCUSSION

The increase in respiratory rate which occurred with localized heating of either the hypothalamus or the skin, and with an increase of the humidity of the air under hot conditions, was always associated with an increase of arterial blood pressure. In other species an increase of arterial blood pressure usually results in a diminution of respiratory activity (Aviado & Schmidt, 1955). It is unlikely therefore that the increase of arterial blood pressure contributed to the increased respiratory rate. The possibility remains that there was a causal relationship between the increase of arterial blood pressure and the decrease in respiratory rate which occurred when the body temperature was between 40·0 and 41·0° C in a hot, humid environment. Similarly, the increase in respiratory rate during heating of the hypothalamus decreased with increasing environmental temperature, whereas the response of the blood pressure was greater at the higher environmental temperature (Table 1). However, the present results do not allow any firm conclusion to be made about the nature of this possible relation.

An increase of arterial blood pressure during exposure to heat has been observed before, in the rat (Richardson, Cooper & Pinakatt, 1962), the dog (Wiggers & Orias, 1932) and in man (Burch & Hyman, 1956). Localized heating of the hypothalamus of the turtle increased arterial blood pressure (Rodbard, 1948) and a similar response can be elicited by electrical stimulation of the hypothalamus of the unanaesthetized dog (Wilson *et al.* 1961). On the other hand, Newman & Wolstencroft (1960), who heated the blood in the carotid arteries of anaesthetized cats, reported no change

of blood pressure until the temperature of the brain was between 41 and 42° C, when the blood pressure decreased. However, the temperature sensitivity of the hypothalamus was not involved in their experiments because they obtained similar results in decerebrate cats.

The increase in heart rate which occurred in response to localized heating of the hypothalamus, like the increase in blood pressure, seemed to depend upon the environmental temperature. The greater increase in blood pressure and heart rate at the higher environmental temperatures could be related to the higher levels of body temperature before the hypothalamus was heated.

The increase of arterial blood pressure which followed localized heating of the hypothalamus or the skin was usually associated with an increase in the skin temperature of the extremities. This means that the increased blood pressure was not due to an increase in cutaneous vascular resistance in the extremities. Moreover, the blood pressure increased when the skin temperatures were already elevated. The increase of blood presure which occurred after the wet-bulb temperature of the air had been increased, at an environmental temperature of 40·0° C, might be related to an increase in blood viscosity, because the haematocrit increases when calves are exposed to hot, humid conditions (Bianca, 1957). There is also some evidence that the blood volumes of calves increase when they are exposed to a hot, humid environment (Bianca, 1957). An increase of blood volume, if it occurred, might also have contributed to the increase of blood pressure observed in the present experiments. An increase of cardiac output in cattle during exposure to a hot environment has been described by Pichaicharnarong (1960). An increased cardiac output could be a factor causing the increased blood pressure in the present experiments.

The increase in respiratory rate which occurred in the present investigation when the hypothalamus was heated at an environmental temperature of −5·0° C was greater than that observed at an environmental temperature of 0·0° C (Ingram & Whittow, 1962*a*). However, the initial temperature of the hypothalamus before heating was higher at an environmental temperature of −5·0° C than at 0·0° C. In the present investigation the increase in respiratory rate during heating of the hypothalamus, at an environmental temperature of 40·0° C, was less than that at −5·0 or 15·0° C. At an environmental temperature of 40·0° C, however, the respiratory rate before the hypothalamus was heated was very much higher than at −5·0 or 15·0° C. Respiratory rates during heating of the hypothalamus at an environmental temperature of 40·0° C were close to the maximal rates possible in calves, and thus the increase in respiratory rate which could occur as a result of heating the hypothalamus was limited. Furthermore, the temperature of the hypothalamus before heating

was higher at an environmental temperature of 40·0° C than at −5·0 or 15·0° C, and it has been observed before that the effect of heating the hypothalamus on respiratory rate diminishes if the environmental temperature exceeds approximately 30° C (Ingram & Whittow, 1962*a*).

Spontaneous increases in the skin temperatures of the ears of the ox during exposure to cold have been described before (Whittow, 1962; Ingram & Whittow, 1962*b*). A similar response occurs in the extremities of man and of other species (Burton & Edholm, 1955). An increase of arterial blood pressure immediately before the spontaneous increase of skin temperature has not been described before. The increase of blood pressure which preceded the initial increase in the skin temperature of the ears, after the animals were led into the cold room, was greater than the increase of blood pressure which preceded subsequent increases of skin temperature. This was probably because the increases of skin temperature in the two ears became less synchronous during subsequent increases of temperature. The cause of the increase in blood pressure and heart rate is not known. It is possible that the effect of the increased pressure was to cause an increase in the blood flow through the ear by the opening of arteriovenous anastomoses, which have been shown to be present in the ear of the ox (Goodall, 1955). This conclusion is in accord with the suggested mechanism of operation of arteriovenous anastomoses (Burton & Eagan, 1956). The absence of arteriovenous anastomoses in the skin immediately above the fore hooves of the ox (Nisbet, 1956) might explain why spontaneous increases of temperature were not observed in this region (Whittow, 1962).

Heating the hypothalamus, when the environmental temperature was −5·0° C, resulted in an increase in the skin temperature of the shanks only when the initial temperature of the shanks exceeded about 10° C. In a previous investigation heating the hypothalamus at an environmental temperature of 0° C caused an increase of the temperatures of the shanks, provided that the temperatures were initially greater than 15·0–20·0° C (Ingram & Whittow, 1962*a*). However, in the present experiments heating of the hypothalamus was maintained for 3 min, as opposed to 1 min in the earlier experiments. These results are of especial interest because Keatinge (1958) has found that isolated strips of ulnar artery from the shanks of the ox failed to respond to adrenaline, histamine or pitressin at temperatures below 10° C.

When hypothalamic heating coincided with a spontaneous increase in the skin temperature of the ear, at an environmental temperature of −5·0° C, the temperature of the ear always increased to a level which was higher than that attained during a spontaneous increase of the temperature of the ear in the absence of heating of the hypothalamus. It is possible

that the blood vessels of the ear were not maximally dilated during spontaneous increases of ear temperature or that heating of the hypothalamus resulted in the opening of different vessels in the ear. However, since heating the hypothalamus was accompanied by an increase of blood pressure it is possible also that an increased blood flow through the ear followed passively from an increase in the pressure gradient between the arteries and veins in the ear. The failure of heating of the hypothalamus to increase the skin temperature of the ear when the ear was cooling after a spontaneous increase of temperature suggests that the blood vessels of the ear are refractory to dilator stimuli for a time after they have dilated spontaneously. Conversely, Hertzman & Roth (1942) have claimed that, in man, vasoconstrictor reflexes cannot be elicited in the finger when the blood flow through the finger increases spontaneously during exposure to cold.

SUMMARY

1. The arterial blood pressure of the unanaesthetized ox has been measured during exposure to various environmental conditions, during localized heating of the hypothalamus and during localized infra-red irradiation of the skin.

2. An increase of arterial blood pressure and heart rate was elicited by an increase of environmental temperature and humidity, by localized heating of the hypothalamus and by infra-red irradiation of the skin.

3. In a cold environment ($-5{\cdot}0°$ C, DB) spontaneous increases in the skin temperatures of the ears were preceded by a marked increase of arterial blood pressure and heart rate.

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