STUDIES OF THERMOREGULATORY MECHANISMS IN SHEEP

A Thesis

Presented to the

Faculty of Graduate Studies and Research

The University of Manitoba

In Partial Fulfillment

of the Requirements for the Degree

Doctor of Philosophy

by

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April 1967

ACKNOWLEDGEMENTS

The writer wishes to thank Dr. E. W. Stringam, Chairman of the Department of Animal Science, for placing the facilities of the Department at his disposal. The assistance and guidance of Dr. G. D. Phillips, Associate Professor of Animal Science, during the course of this work, and in the preparation of this manuscript is gratefully acknowledged.

Financial support for this project from the Horned Cattle Purchases Fund of the Manitoba Government and from the National Research Council of Canada in the form of an Assistantship is gratefully acknowledged.

ABSTRACT

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Experiments were conducted on 4 Suffolk ewes to determine the distribution of peripheral thermal receptors on the skin and in the respiratory passages. The sheep were fitted with a carotid loop and tracheotomy tube, and were maintained in a holding room containing a modified metabolism crate provided with feed and water troughs. The temperature of the holding room ranged from 20°C to 22°C. Measurements were made of the temperature of the blood in the carotid artery and in the jugular vein during exposures to hot and cool environments, in a climatic chamber, during localized heating of the perineal and flank regions. In addition the ear and lumbar skin temperatures were recorded. Measurements were also made of respiratory frequency and heart rates.

When an adequate area of fleece (3000 cm^2) was sheared on the thoraco-lumbar region, a marked decrease in respiratory frequency occurred at an ambient temperature of 20°C and 55% relative humidity. At ambient temperatures of 20°C, heating the perineal region of unshorn ewes to 40°C resulted in the initiation of polypnoea.

Perineal heat treatment in a cool environment (15°C and 10°C) also resulted in an increased respiratory frequency but this increase was of lesser magnitude than that obtained at 20°C ambient temperature. The respiratory responses to heating the perineal region were maintained despite a marked fall in the temperature of the carotid blood. Partially shorn sheep no longer panted when the perineal region was heated to 40°C. The rise in respiratory frequency following perineal heat treatment of unshorn sheep was attributed to the stimulation of warm receptors located in the perineal region. The lower respiratory frequency of partially shorn sheep at 20°C ambient temperature and the absence of onset of polypnoea following perineal heat treatment at all ambient temperatures studied is attributed to the stimulation of cold receptors located beneath the fleece.

Short term exposure to rising ambient temperature resulted in an immediate onset of polypnoea in unshorn sheep, whereas in partially shorn sheep, there was a 20-30 minute delay in the onset of polypnoea. After this delay, there was an abrupt rise in respiratory frequency which then approached that of unshorn sheep.

In an attempt to study the presence of thermal receptors in the naso-buccal passages, the sheep were fitted with a two-way tracheal canula. A face mask with its own temperature controlled air supply was placed on

the animal so as to vary the temperature of the air surrounding the naso-buccal region. Meanwhile the animal breathed ambient air ($20^{o}C$) through the distal end of the two-way canula. Circulation of warm air ($40^{o}C$) through the naso-buccal passages at ambient temperatures of $20^{o}C$ was accompanied by an immediate rise in respiratory frequency, reflex vasodilatation, and increased heart rates. Conversely cooling the naso-buccal air to $10^{o}C$ after polypnoea had been fully established at an ambient temperature of $40^{o}C$ resulted in a marked depression of respiratory response. The changes in respiratory frequency following naso-buccal heating or cooling was not accompanied by any changes in the carotid blood temperature. It was therefore concluded that warm and cold receptors are located in the naso-buccal passages whose stimulation results in the reflex onset of polypnoea or a depression in respiratory response in a polypnoeic sheep.

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INTRODUCTION

It has long been known that the body temperature of homeotherms under varying degrees of climatic stress is the net result of the balance between heat production and heat loss. The balance is achieved through changes in physical and chemical processes brought about through the action of neural mechanisms.

One of the major discoveries of thermoregulation was that thermal homeostasis may be disturbed by lesions of the brain in the hypothalamic region and this discovery proved that the principal mechanisms of mammalian temperature regulation are neural. The hypothalamus is sensitive to temperature changes occurring in itself, since an increase in hypothalamic temperature induces panting and cutaneous vasodilatation, whereas a decrease in temperature initiates shivering and vasoconstriction.

It has been shown in humans, that the mechanism of temperature regulation is of hypothalamic origin and that the heat regulating centres are capable of controlling body temperature without afferent sensory impulses. Man regulates his body temperature remarkably well by enhanced sweating rates under hot and humid conditions. The situation is somewhat different in sheep, which do not sweat as much as humans do, although evaporative heat loss does take place from the skin. In hot surroundings, the excess heat in sheep is mainly dissipated by panting.

Until recently, there has been some doubt whether the stimulus for the panting originates from the central or the peripheral thermal receptors. It is now an established fact that, in sheep, the onset of panting in response to a sharp rise in ambient temperature occurs even in the absence of any rise in the temperature of the blood supplying the brain, thus suggesting that the stimulus to thermal panting could be of peripheral origin. Respiratory rates in sheep can be depressed by shearing which suggests the existence of cold receptors beneath the fleece. Conversely, panting has been initiated in a ram by the exposure of the scrotal skin to a warm environment and the panting persisted even though there was a $2^{o}C$ fall in deep body temperature. Similarly, heating the mammary region of female goats induced panting but this was accompanied by a rise in rectal temperature. This suggested the existence of warm receptors in the inguinal region, although there is an apparent contradiction between these later two findings. One leads to the conclusion that there is an inverse relationship between body temperature and respiration rates, whereas the other implies a direct relationship.

The present study was undertaken to examine the distribution of peripheral thermal receptors on the skin of ewes. In view of the prevailing uncertainty about the role of respiratory passages in thermoregulation, an

attempt was made to localize the distribution of thermal receptors in the respiratory passages. Experiments were also conducted to confirm the existence of cold receptors in the skin beneath the fleece. The temperature of the blood in the carotid artery of unanaesthetized sheep was kept under continuous observation in order to see whether or not the changes brought about by local heating or cooling of certain regions of the skin and naso-buccal area were correlated with various body temperatures, respiration rates, and heart rates.

LITERATURE REVIEW

PRESENT CONCEPT OF BODY TEMPERATURE

Body temperature can be measured at various sites on the animal body; e.g. oral, rectal and axillary temperatures are often used clinically to indicate deep body temperature. In classical physiological studies concerned with heat and temperature, rectal measurements have been the favoured method for obtaining deep body temperatures in mammals. However, recent studies indicate that rectal temperature is inadequate as a measure of deep body temperature, especially during sudden fluctuations in the heat content of the body. Benzinger (1961) showed that when humans were subjected to drastic changes of temperature either internally or externally, the temperatures in three different cranial locations, namely, the anterior ethmoidal septum, Rosemuller's fossa on the stem of the internal carotid artery, and the tympanic membrane, ran parallel to one another but the rectal temperature on the other hand very poorly paralleled the cranial temperatures. Similarly Bligh (1957a) found that both rectal and bicarotid blood temperatures started to rise in sheep when the ambient temperature was raised from 20°C and 17mg/l absolute humidity to 40°C and 45 mg/l absolute humidity. However, when the humidity was lowered at the end of an experiment at 40°C with high humidity, rectal temperature continued to rise or remained steady for 10 minutes while bicarotid

blood temperature started to fall immediately. These results indicate that changes in the heat content of the body are not immediately reflected in the rectal tissue. The temperature of any tissue is governed mainly by the temperature of the arterial blood supply and the rate of blood flow to it, its local heat production, and the heat exchange with the surrounding tissue. Grayson (1951) observed that temperature in the rectum may be influenced by the rate of blood flow through it.

It is unlikely that the temperature of any one location is a true representation of deep body temperature, whereas that of the blood supplying the brain is likely to provide a better measure of the changes which affect central thermoregulatory mechanisms. However, it was not until Bligh (1957a) successfully implanted thermocouples in the bicarotid trunk of the calf that it became possible to record the temperature of the blood flowing to the brain, for prolonged periods, in a conscious animal.

In an attempt to find reproducible results indicating the relationship between the response of sweating and internal and cutaneous stimuli, Benzinger and Taylor (1963) found the tympanic membrane of the ear to be a reliable index of deep body temperature. His non-surgical technique does not readily lend itself to animal research because each animal has to be thoroughly trained to permit this kind of treatment.

Hypothalamic temperature is generally considered to be a more reliable index of body temperature (Benzinger and Taylor, 1963). But introduction of temperature sensing elements directly into the hypothalamic region may well produce a variable degree of injury and consequently affect the thermoregulatory mechanisms. In view of these limitations, carotid blood temperature has gained considerable recognition as an index of average body temperature, since the temperature of the arterial blood leaving the heart can be considered as the mean temperature of the circulating blood.

HEAT EXCHANGE MECHANISMS

Physiologists generally refer to rectal and skin temperatures as core and shell temperatures, respectively. Heat is produced as a result of metabolic activity in the body and is conveyed to the skin surface. Depending on the thickness of the skin and the ambient temperature, heat is lost to the atmosphere by way of radiation, conduction, convection and evaporation at the skin surface.

Circulatory adjustments in heat exchange

One of the most important function of the circulatory system is the regulation of heat transfer from the metabolically active tissues to the body surface. The circulation is ideally suited for this function in that the heat conductivity and the specific heat of blood

are high.

Forster et al. (1946) observed that vasodilatation in a human hand begins at an ambient temperature of 22°C. Hertzman et al. (1946) showed that within the hand, the rate of circulation in the digits was relatively greater than that in the metacarpal region of the hand. Eichna et al. (1950) have shown that the overall blood flow to the skin varied from 0.16 l/m^2/minute in a nude resting man at an ambient temperature of 28°C to 2.6 l/m^2/minute in men working in an extremely hot environment.

Peripheral blood flow in ruminants has not been investigated but certain deductions have been made from surface temperature studies. Findlay and Beakley (1954) exposed an Ayrshire calf to an environmental temperature of 12°C. This temperature was then raised slowly to 20°C. The rectal and ear temperatures were measured repeatedly at 5 minute intervals. A 2°C rise in ambient temperature (T_A) from 18°C to 20°C was accompanied by a 13°C rise in ear skin temperature (T_{ES}), that is from 22°C to 35°C, while the rectal temperature (T_R) remained fairly stable at 38°C. The ratios of thermal conductances from ear to environment ($T_{ES}-T_A$) and from core to shell (T_R-T_{ES}) were calculated. The authors observed that a 2°C rise in environmental temperature was accompanied by a 20-fold increase in blood flow to the ear. Whittow (1962) made

a detailed study of the skin temperatures of the extremities of the ox at ambient temperatures ranging from -5°C to 45°C. Between ambient temperatures of -5°C and 20°C, large variations occurred in the skin temperatures of dewlap, ears and shank. The skin temperature at the base of the tail was relatively greater than at the tip. When an inflated cuff was placed around the forearm at ambient temperatures of 0°C to 20°C, there was a 0.5°C to 8.3°C fall in skin temperature of shank and pastern regions. However, skin temperatures on the corresponding regions of the opposite leg did not change during the same period. When ambient temperatures ranged from 35°C to 45°C, skin temperatures closely followed the ambient temperature. These changes suggest the importance of circulatory adjustments in thermoregulation.

Blaxter et al. (1959) made a detailed study of sensible heat loss in sheep at ambient temperatures ranging from 8°C to 38°C and partitioned the heat losses due to radiation and convection. Heat losses due to radiation and convection increased with decreasing ambient temperatures at the rate of 50 and 35 Cal/m^2/24 hr/°C, respectively. Their experiments also demonstrated that convection losses increased with body movements during shivering and panting. The tissue conductance of heat increased with rising ambient temperature. During the initial stages of shivering, between ambient

temperatures of 18°C to 23°C, the tissue conductance was minimum (225 Cal/m^2/24 hr/°C) and it then increased markedly to 310 Cal/m^2/24 hr/°C, when shivering was fully established below ambient temperatures of 18°C. These changes in the thermal conductivity of tissues between the initial stages of shivering and when shivering was fully established were attributed to changes in heat production resulting from contraction of fascial muscles and redistribution of blood to appendages. These findings indicate that there are wide variations in blood flow to different skin regions.

Insulating effect of fleece and hair

MacFarlane et al. (1958) observed that, in sheep, a fleece length of 4 cm could produce a temperature gradient of 45°C. The temperature at the tip of the wool was 87°C while standing in hot sun, whereas that of the skin surface was only 42°C.

Blaxter et al. (1959) studied the effects of environmental temperature on the energy metabolism and heat exchange of sheep in the shorn condition and with varying lengths of fleece. Heat production remained fairly constant in fleeced sheep (10-12 cm fleece) at ambient temperatures of 11°C to 33°C, whereas in shorn sheep (1 mm fleece) there was a linear increase in heat production as the ambient temperature decreased from 38°C to 11°C. They calculated the critical temperature

of sheep with different fleece lengths and found that heavy fleece depressed the critical temperature of the animal. For example, sheep with 1 mm fleece had a critical temperature of -3°C. Since critical temperature is the temperature below which the animal must increase its heat production to maintain constant body temperature (Brody, 1941), these variations in critical temperature of shorn and unshorn sheep clearly demonstrate the effectiveness of fleece as an insulating layer.

Schmidt-Nielsen et al. (1957) found that camels with natural fur have a lower water intake than when they were shorn. Since water expenditure by the animal body increases with heat load, their experiments provided an indirect evidence of the effectiveness of the animal's coat under heat stress.

Evaporative heat loss from the skin surface

Panting has been regarded as the primary mechanism on which cattle depended for heat loss in hot and humid environments. Worstell and Brody (1953) expressed the opinion that cattle, unlike man, do not sweat. However, Robinson and Klemm (1953), Knapp and Robinson (1954) and McDowell et al. (1954) claimed that an effective mechanism for moisture vaporization from skin must also exist, since total heat loss was always greater than that accounted for by respiratory evaporative loss alone. Estimates of the rate of surface evaporation was obtained

by aereated capsules applied to the body surface (McDowell et al. 1961). They found that under dry conditions (20% relative humidity), the rate of surface evaporation increased with the rise of temperature from 30°C to 45°C whereas at 80% relative humidity the surface evaporation increased between ambient temperatures of 28.3°C to 32.7°C but decreased at 35°C. McLean (1963) investigated the relative efficiency of various body areas for surface evaporation by the aereated capsule method and found that the rate of evaporation was highest at the shoulder region, followed by the neck, hindquarter and dorsal regions of the trunk. The evaporation from the ventral body surface was significantly less than that from the dorsal surface of the body.

The evidence cited above suggests that evaporative losses through the skin are associated with sweat gland activity and distribution over the body surface. Ferguson and Dowling (1955) and Dowling (1958) clearly demonstrated that the sweat glands in cattle are functional and do respond to thermal stimuli. Subsequently, the secretory nature of these glands was confirmed by several workers (Findlay and Jenkinson, 1960; Taneja, 1956). Morphological studies of the sweat glands were conducted by different workers (Yamane and Ono, 1936; Findlay and Yang, 1950; Carter and Dowling, 1954; Nay and Hayman, 1956; Nay, 1959). Nay and Hayman (1956) compared the

sweat glands of the Bos taurus and Bos indicus species. They found that Bos indicus had much larger and more numerous sweat glands. They also observed that the sweat glands in Bos indicus were more superficially located than those in Bos taurus. The following table points out vast differences in sweat gland activity of sheep, cattle and man.

TABLE 1

A COMPARISON OF THE NUMBER OF SWEAT GLANDS AND THE RATE OF SWEATING IN SHEEP, CATTLE AND MAN.

Species	Sweat glands (no./cm^2)	Rate of sweating ($g/m^2/hr$)
Sheep[a]	290	32
Cattle[b]	1000	588
Man[c]	150	2000

Although the values in Table 1 are approximate, they do provide interesting information. The sweat glands of sheep are less efficient than those of cattle while those of the human are highly efficient in the mobilization of water and subsequent evaporative cooling.

[a]Brook and Short (1960)
[b]McDowell et al. (1954); Nay and Hayman (1956)
[c]Kuno (1956)

Allen (1962) studied the sweating rates of Bos indicus, Jersey and Bos indicus x Jersey crosses. There was almost a linear increase in sweating rates with rise in skin temperature from 32.2°C to 37.7°C in Jerseys, whereas Bos indicus did not show a corresponding increase until the skin temperature had risen to 35°C, after which the increase was linear. This delay in the onset of sweating of Bos indicus indicates that the threshold stimulus required for the initiation of the sweating response was greater than that of the Jersey.

The evaporative heat loss from the skin of sheep has not been studied as extensively. Alexander and Brook (1960) measured the evaporative moisture from the skin and respiratory passages of lambs (5 to 15 days old) in a closed-circuit calorimeter. The moisture vaporization was approximately the same from both channels at ambient temperatures below 30°C but at ambient temperatures above 30°C the evaporation increased from both channels, increasing more rapidly from the respiratory tract than from the skin. When cutaneous evaporation was blocked by increasing ambient humidity, at ambient temperatures ranging from 40°C to 41°C, respiratory evaporation increased. However, when the respiratory moisture loss was minimized by raising the humidity of the inspired air almost to saturation, while maintaining normal ambient humidity, there was little or no increase in

evaporative heat loss from the skin and rectal temperature increased rapidly by about 2°C. Brook and Short (1960) determined the total evaporation from the skin by the dessicated capsule method in various breeds of sheep using 4 Merino mutant sheep without sweat glands as controls. There was no significant difference (10.2 g/m^2/hr) as regards total evaporative moisture, at 20°C ambient temperature and a water vapour pressure of 12.5 mm Hg, between the experimental and control group but the difference at 40°C ambient temperature and a water vapour pressure of 28.1 mm Hg was significant (32.1 g/m^2/hr). This difference was ascribed to sweating. There was no significant increase in sweating rate as a result of further increase in ambient temperature.

These findings and those of Alexander and Brook (1960) and Knapp and Robinson (1954) suggest that, in sheep, sweat can contribute substantially to evaporative cooling until the ambient temperature approaches that of the body. Once the sensible heat loss mechanisms are blocked by high ambient temperatures, panting apparently becomes the more important channel of heat loss.

Evaporative heat loss from the respiratory passages

Evaporation from the respiratory surface is not a source of heat loss, in man, at higher ambient temperatures. According to Bazett (1949), respiratory response in humans showed no indication of direct cooling effect

with respect to the ascending ambient temperature. Therefore, it may be assumed that the respiratory apparatus of humans is not of great physiological importance as regards temperature regulation at high ambient temperature. On the other hand, in ruminants like cattle and sheep, the respiratory frequency increases by 5 to 10 fold in response to a rise in ambient temperature. Findlay (1954) showed that, in cattle, the respiration rate increased with a rise in ambient temperature and finally reached a maximum of 110/minute at ambient temperature of 37.8°C. Bligh (1957a), working with sheep, found similar increases in the respiration rates. If a condition of high humidity was superimposed upon an already high ambient temperature, there was a further increase in respiration rate.

There appears to be no general agreement as to the amount of heat lost through vaporization from the respiratory passage of ruminants. Kibler and Brody (1956) estimated that about 35% of the heat produced in cattle was dissipated from the respiratory tract of a panting animal by vaporization. Measurements made by mask techniques (McLean, 1963) showed that respiratory evaporative cooling accounted for only 25% to 35% in cattle. Lee (1950) measured the respiratory rate as well as the respiratory volume in sheep and estimated the rate of evaporation, assuming that the expired air was fully saturated at

body temperature. His results indicate that the rate of evaporative moisture loss from the respiratory passages, during panting, could be as high as 1 g/minute. Knapp and Robinson (1954) carried out a comparative study of evaporation from the respiratory passages of a Jersey cow and a Corriedale ewe. When both animals were subjected to similar environmental conditions ranging from 30°C to 45°C, respiratory evaporative heat loss in the cow amounted to about 1/5 to 1/9 of the total heat loss, whereas in the sheep it amounted to about 1/3 of the total heat loss. This suggests that the respiratory cooling mechanism is of greater importance in sheep than in cattle.

There has been some doubt as to the location of the site of evaporative heat loss in the respiratory tract. The site of evaporative cooling was investigated by Bligh (1957b). He measured the temperature of the blood in the pulmonary artery (T_p) and bicarotid trunk (T_B) and found no differences between T_p-T_B, even when the calf was panting. He therefore suggested the term 'evaporative cooling in the respiratory passages' instead of the usual term 'evaporative cooling in the lungs'. Ingram and Whittow (1962) observed that large increases in the respiratory rate were associated with a significant fall in jugular blood temperature and thus corraborated Bligh's (1957b) findings that evaporative cooling occurs

in the upper respiratory tract.

THERMOREGULATORY MECHANISMS

One of the major discoveries in the field of temperature regulation was that whenever hypothalamic lesions were present, thermal homeostasis was eliminated (Ott, 1877). This was followed by Meyer's (1913) postulation of the 'dual centre' theory of hypothalamic control of heat production and heat loss. Since then, the existence of thermoregulatory centres in the hypothalamus has been established for several species of mammals (Ranson, 1940). In the cat and monkey (Magoun et al. 1938; Beaton et al. 1941) local heating of the region between the optic chiasma and the anterior commissure was accompanied by vasodilatation, an increase in respiratory rate, and sweating. In the ox, heating of a discrete region of the hypothalamus caused panting and vasodilatation (Findlay and Ingram, 1961). In goats, Andersson and Persson (1957) demonstrated the existence of a discrete heat loss centre in the hypothalamus (preoptic area), electrical stimulation of which results in the initiation of panting and vasodilatation. Stimulation of these areas for a prolonged period led to 10°C fall in rectal temperature. The same worker (Andersson, 1957) observed that electrical stimulation in the vicinity of the septum pellucidum produced shivering, peripheral vasoconstriction, and piloerection.

Until recently it was not clear whether the hypothalamic centres respond to changes in the temperature of its own environment in the intact animal or whether nervous excitation of peripheral origin can be an adequate stimulus to activate thermoregulatory mechanisms. It was assumed that in an intact animal an increase in body temperature activates a coordinated heat loss mechanism involving panting, cutaneous vasodilatation, and sweating (Folkow et al. 1949). As early as 1879, Sihler demonstrated that the normal stimulus to panting in the dog was entirely of reflex nature, originating from the periphery. Randall, Deering and Dougherty (1948) showed that the stimulus to sweating in man can be entirely due to peripheral temperature stimulation. Beakley and Findlay (1955) showed that, in the calf, a moderate thermal rise of 10°C in ambient temperature, from 20°C, caused an increase in respiration rate. This rise in respiration rate was not accompanied by any rise in rectal temperature. The lack of rise in rectal temperature during panting suggested that the stimulus to thermal polypnoea could also be of peripheral origin. A number of investigations, aimed at determining the extent to which physiological temperature regulation is central or peripheral, have been undertaken (Hemingway, 1938; Forster and Ferguson, 1952; Lim and Grodins, 1955; Andersson et al. 1956; Fusco et al. 1961). However, these studies have tended to indicate both a central and a peri-

pheral thermal control mechanism may be involved.

In ruminants, the thermosensory receptors in the skin appear to play an important role in thermal panting when the animals are subjected to heat stress. When calves were subjected to high ambient temperatures, Bligh (1957c) noted that panting started even in the absence of any increase in the temperature of the blood supply to the brain. He observed (Bligh, 1959) a similar reaction in sheep. These findings indicate that the thermal stimulus to panting could be entirely peripheral in origin just as stimulation of the thermosensory nerve endings in human skin induce sweating (Kerslake, 1955).

Waites (1962) observed that at an ambient temperature of 18°C, heating the scrotum of a fully fleeced ram to over 36°C evoked polypnoea which immediately stopped when the stimulus was withdrawn. Linzell and Bligh (1961) observed similar responses when the mammary region of goats were subjected to heating. Bligh (1963b) made some interesting observations on the respiratory rates of shorn and unshorn sheep. At chamber temperatures of 20°C, he found that the respiratory rate in shorn sheep was distinctly lower than that in unshorn sheep. When the ambient temperature was raised from 20°C to 42°C, which was accomplished in about 30 minutes, there was an immediate rise in the respiratory rate of the unshorn sheep, whereas no such response occurred in the shorn sheep

for a period of 30 to 40 minutes. After this period there was a sudden onset of panting in the shorn sheep and the differences in respiratory rates of shorn and unshorn sheep were insignificant from then on. The lower respiratory rate of shorn sheep at an ambient temperature of 20°C and the delay in the onset of panting after the temperature had reached 42°C were observed even in unshorn sheep when an area of 2500 cm^2 was closely shorn on any region of their trunk. The depression in respiratory rates in shorn sheep at 20°C was attributed to the presence of cold receptors on the skin and their subsequent stimulation. The delay in the onset of panting at an ambient temperature of 42°C was believed to be the carry over effect of the preceeding stimulation of cold receptors, thus causing a temporary nervous block via the hypothalamus on the warm receptor impulses leading to induction of polypnoea. The onset of panting after the delay was attributed to the withdrawal of the aforesaid temporary block between warm receptor drive and respiratory centres. These findings and those of Waites (1962) showed that thermal polypnoea could be induced by peripheral warm receptors alone. The warm receptors were concentrated at specific areas over the body surface, whereas the cold receptors were uniformly distributed over the external surface of the body.

MATERIALS AND METHODS

Animals

Four well-trained, adult Suffolk ewes weighing between 80-95 kg were used as experimental animals. Selection of the animals was made on the basis of observations that all animals had similar pulse rate, rectal temperature and respiration rates.

Management

The experimental animals were maintained in a holding room containing a modified metabolism crate (figure 1) provided with feed and water troughs. After the necessary surgical preparations, the sheep were brought into the holding room and maintained there, except when subjected to short term thermal treatments in the climatic chamber, for the duration of the experimental period. The temperature of the holding room ranged from 20°C to 22°C with 53 to 55% relative humidity. The holding room was well lighted and ventilated.

The animals were fed approximately 250 g of concentrate (supplemented with cobalt-iodized salt) and 500 g of good quality chopped hay in the morning between 8.30 and 9.00 a.m. and about 500 g of hay in the afternoon between 4.30 and 5.00 p.m. Water was provided *ad libitum*. Freedom of movement within the crate and the walk to and from the climatic chamber, about 25 yards, was their

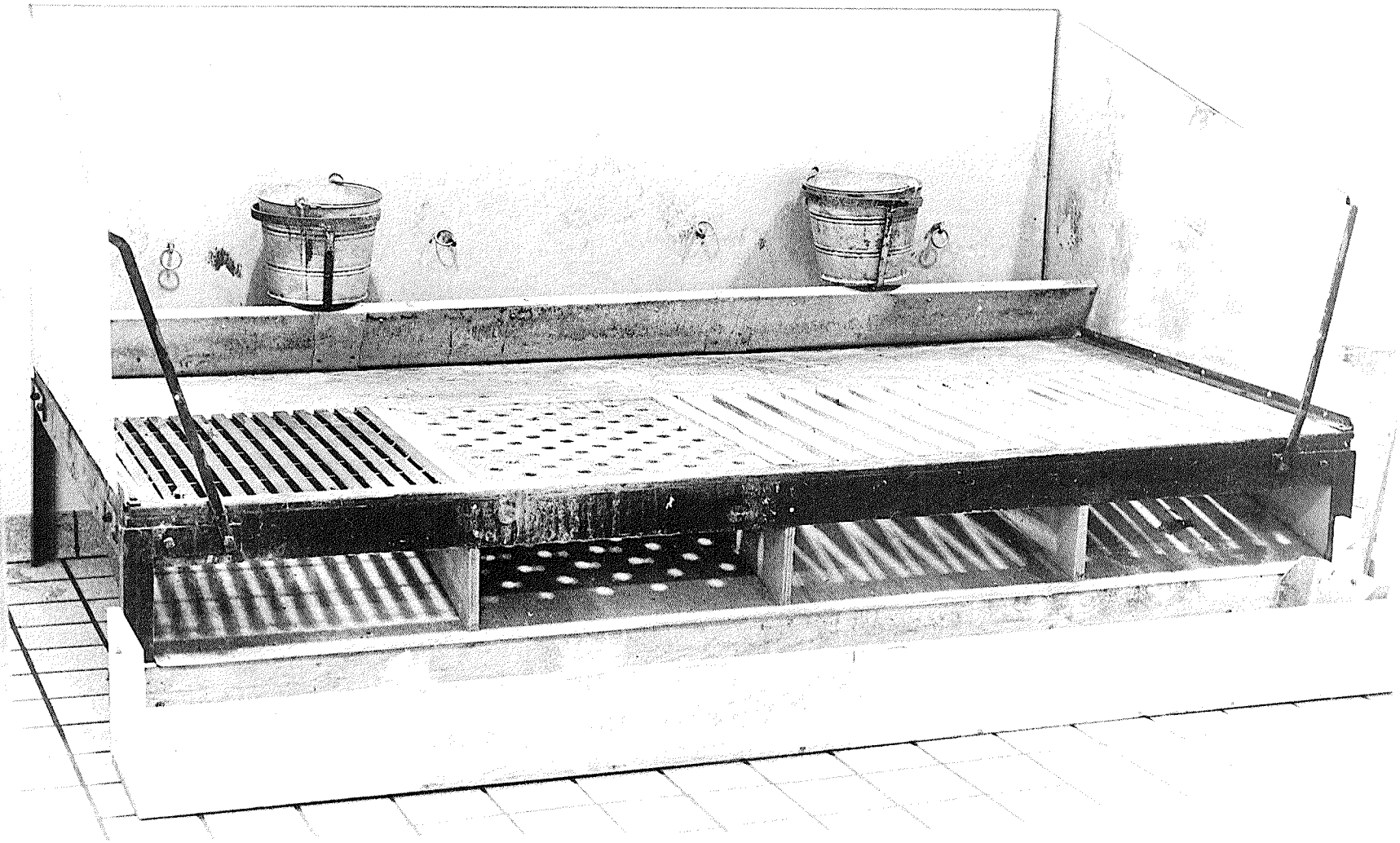

Fig. 1. Modified metabolism crate.

only exercise.

The sheep were sheared partially or fully, as required for the various experiments, with 'Oster' small animal clippers (Model A2) with a fine (size 40) head. The fleece was sheared evenly so that only about 2 mm of wool fibres remained above the skin surface. The clippers were applied twice weekly, so that a 'shorn' animal had a standardized coat length throughout an experimental period. When re-insulation was necessary, the sheep was covered with a sheep skin coat tailored to fit the shorn area. The coat was held in position by the aid of leather straps.

Experimental

Experiments were conducted after the animals had fully recovered from surgical procedures and had adjusted to the new conditions in the holding room. For each experiment, the sheep was walked into the climatic chamber, whose temperature and relative humidity was preset, and placed in a metabolism crate. On the day of the experiment the test animal was not given food or water in the morning. During each experiment, the skin, carotid and venous blood and rectal temperatures were recorded. In some experiments, the tracheal temperatures were also monitored. Respiration and heart rates were also recorded. All the above mentioned measurements were recorded at intervals of 5 minutes. After the various measurements

were recorded, which did not exceed 3 hours in most experiments, the animal was provided with feed and water. The experiments were conducted in such a way that each sheep was used every fourth day. This schedule permitted sufficient time for the animal to recover from carotid and venous punctures.

Plan of experiment

Preliminary experiments

Experiments were conducted to study the effect of removal of fleece in stages upon respiratory rates and various tissue temperatures at ambient temperatures of 20°C and 55% relative humidity. An area of about 1000 cm^2 (50 x 20 cm) was closely shorn on the thoraco-lumbar region. The shorn area was progressively increased upto 3000 cm^2 (60 x 50 cm).

Experimental schedule

The experimental schedule has been divided, for convenience, into 3 types of experiments.

Type 1

In the first type of experiments, the mammary region[1] was heated by an electric heating pad[2] (35 x 30 cm) to about 40°C to examine the distribution of warm receptors on the naked skin regions of the body. The heat treatment was carried out in the climatic chamber at 20/55, 15/55 and 10/55 (dry bulb temperature, °C/ relative humidity, %). In

[1]Henceforth called perineal region
[2]G. W. Endress Co., Ltd., Brantford, Ontario, Canada

subsequent experiments, a shorn area (900 cm^2) on the trunk or flank region, was heated to about 40°C to determine the effect on respiratory patterns and various body temperatures. In an attempt to study the role of fleece in thermoregulation during perineal heating, an area of 3000 cm^2 was closely shorn on the thoraco-lumbar region.

Type 2

The second type of experiments were conducted to study the effects of rise and fall in ambient temperature on cardio-respiratory activities and various body temperatures. The animals spent the first 30 minutes in the chamber preset at 20°C and 55% relative humidity. At the end of 30 minutes, the chamber temperature was raised to 40°C and maintained at that level for the duration of the experiment. The chamber temperature rose slowly at the rate of 0.5°C/minute. Relative humidity varied between 40 and 50% when the chamber temperature was rising. After attaining 40°C, the relative humidity of the chamber remained close to 52%. After thermal polypnoea had been established at 40°C and 55% relative humidity, the chamber temperature was lowered to 10°C. There was a 15°C fall in chamber temperature during the first 10 minutes and a further 15°C fall in the next 30 minutes. The relative humidity was not controlled during fall in chamber temperature and varied between 50 and 60%.

In order to study the distribution of cold receptors

on the thoraco-lumbar region, a cooling pad was applied to a shorn area (900 cm^2) on the lumbar or thoracic region, after thermal polypnoea had been established at chamber temperature of 40°C and 55% relative humidity. The pad was perfused with a mixture of alcohol and dry ice, so as to lower the skin temperature to about 10°C.

Type 3

In the final type of experiments, an attempt was made to investigate the distribution of thermal receptors in the respiratory tract. A face mask enclosing the naso-buccal area with a continuous controlled air supply, was used. The temperature of the air supplied to the face mask could be controlled independently. For these experiments, the animals were fitted with modified tracheal canulae. The pulmonary end of the canula was connected to a polythene tubing (vol-100 cc) to compensate for the dead space in the trachea and the naso-buccal cavity. This was done in order to prevent the CO_2 effect on respiration rates. The animals spent the first 30 minutes wearing the face mask in the climatic chamber which was preset at 20°C and 55% relative humidity. After 30 minutes, the temperature of the naso-buccal air was increased to approximately 40°C. The relative humidity of the naso-buccal air was not controlled and varied between 75 and 85%. In some experiments, the naso-buccal area was subjected to infra-red irradiation until the naso-buccal skin temperature reached

40°C to see whether this region harbors any thermal receptors. An attempt was also made to study the distribution of cold receptors in the respiratory tract. Each animal spent 12-14 hours at 40°C ambient temperature. After thermal polypnoea had been established, the naso-buccal air temperature was lowered to 10°C.

Climatic chamber[1]

Test exposures were carried out in a climatic chamber in which dry and wet bulb temperatures could be held at a constant level. The climatic chamber has internal dimensions of 2.54 m by 1.83 m and 2.54 m high. Some of the relevant features of the chamber are mentioned below.

Temperature control	0 to 40°C ± 1°C
Humidity control	20 to 90% ± 2%
Air circulation	4000 c. ft./min
Fresh air and exhaust	50 c. ft./min
Light control	0 to 3500 ft. cd.

Air circulation within the chamber was maintained by two centrifugal type fans. Flexibility of air circulation could be achieved by shutting down one fan completely. Constant temperature was maintained by modulating the flow of chilled glycol solution through a cooling coil located within the climatic chamber. The glycol chiller, glycol circulating pump and the refrigeration condensing unit were controlled automatically and provided a constant chilled glycol supply as required by the control system.

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[1]Constructed by Coldstream Products Ltd., Winnipeg, Canada

A programming type of temperature controller[1] controlled heating and cooling and maintained a predetermined temperature.

Humidity was provided by pneumatic water sprays located in the air plenum. The water sprays were controlled from the wet-bulb temperature cam in the recording controller. Humidity control was limited by a $0^{o}C$ wet-bulb temperature.

Surgical procedures

Carotid loop

The surgical procedure described by Bone et al. (1962) was followed for the preparation of carotid loop. The method is briefly described below.

After anaesthetising the animal with Nembutal, the right external carotid artery was approached through a 10-12.5 cm long incision through the skin and subcutaneous tissue, at a level anterior to the bifurcation of the Cleido-occipitalis and Cleido-mastoideus muscles and extending anteriorly along the ventral border of the Cleido-mastoideus muscle to a point 5 cm posterior at the ramus of the mandible. Approximately 12-14 cm of the carotid artery was exposed by blunt dissection along the ventral border of the Cleido-occipitalis muscle. The vagus nerve which lies on the dorsomedial aspect of the artery within the carotid sheath was bluntly dissected from the artery

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[1]Bulletins 1A 202 & 1E 200. Environmental control manual, Coldstream Products, Winnipeg, Canada

without causing undue damage to the nerve. The smaller branches of the carotid artery were clamped, ligated and severed.

A second incision about 10 cm long, approximately 4 cm above and parallel to the first incision was made through skin and subcutaneous tissue, in such a way that the midpoints of the first and second incisions were at the same level. The skin between the two incisions was freed from the body by blunt dissection resulting in a 10 cm flap of skin connected to the body at both ends. The external carotid artery was then brought to the surface of the body and the two free edges of the first and second incisions were sutured with interrupted sutures of 0 to 00 chromic catgut, leaving about 2.5 cm unsutured portion at each end. The carotid artery was then enclosed in the flap of skin produced between first and second incisions and the edges of the flap were then sutured with interrupted sutures. Thus, the skin flap served as a tube for carotid artery. The free edges at both ends of the tube were then apposed and closed with interrupted sutures. The completed operation resulted in a carotid loop (figure 2) of approximately 9 cm long with adequate space beneath it for 3 fingers.

Permanent tracheotomy

The sheep were also fitted with stainless steel tracheotomy tubes (Field pattern; 11 mm lumen). The trachea

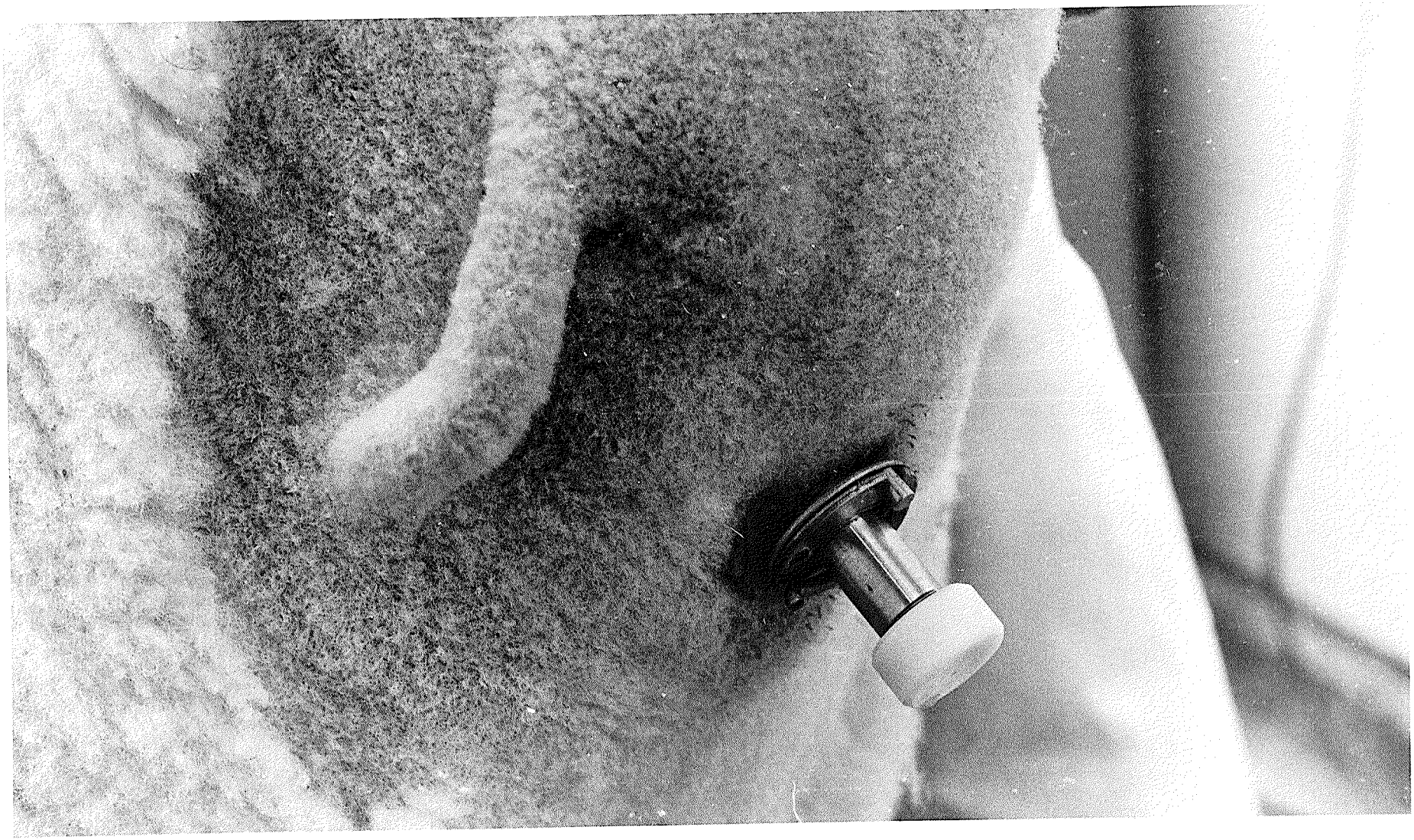

Fig. 2. Sheep fitted with carotid loop and Field pattern tracheotomy tube.

was approached by a midventral incision about 2.5 cm long through the skin and subcutaneous tissue starting from the IV or V ring of the trachea and extending posteriorly. The muscles were separated and haemorrhage was controlled. A circular piece of cartilage from two adjoining rings was removed, without cutting the entire width of either ring. The tracheotomy tube was introduced into the opening made.

Independent naso-buccal circulation assembly[1]

The assembly (figure 3) consists of a well insulated wooden box fitted with the following components.

a. Three gallon liquid container made of galvanized tin.

b. 1000 watt immersion heating coil.

c. Simmerstat,[2] energy regulator, used for the control of water temperature.

d. Motor driven 4 blade fan with 15 cm blades.

e. 'Midget' centrifugal pump,[3] measuring 18.5 cm x 8.9 cm x 8.2 cm with a capacity of 7 gallons/min, powered by a 1/20 h.p. motor, and

f. Automobile type radiator having dimensions of 20.3 cm x 19.0 cm x 5.0 cm.

Hot air was produced by circulating hot water through the radiator. When cool air was required, the water in the container was replaced by a mixture of dry ice and alcohol. The desired air temperature could be obtained by

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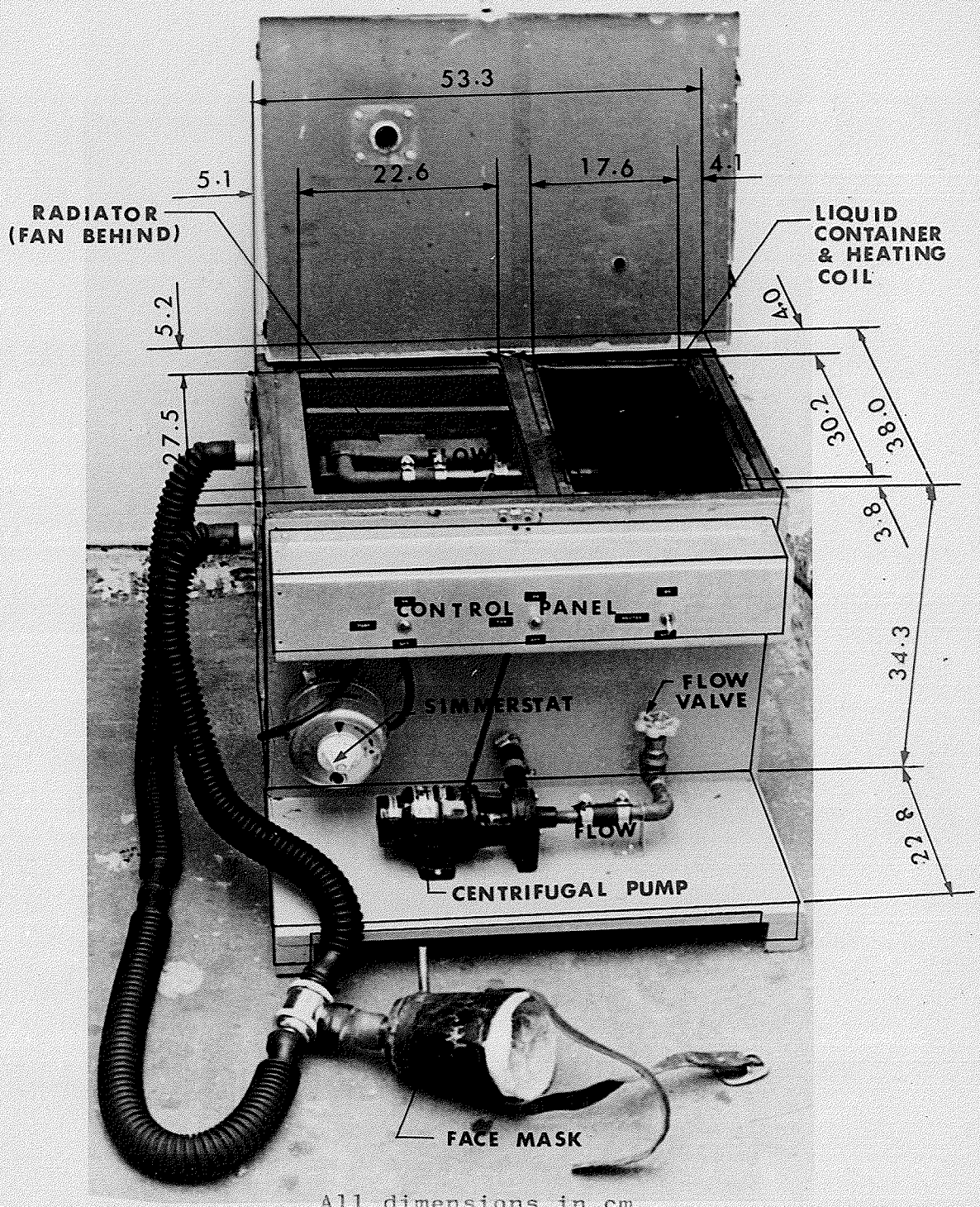
[1]Assembled at the Implement shed, Animal Science Department, University of Manitoba, Winnipeg, Canada.
[2]Electrothermal Engineering Ltd., London E7, England.
[3]Eastern Industries, Hamden, Connecticut, U.S.A.



All dimensions in cm

Fig. 3. Independent naso-buccal circulation assembly.

regulating the temperature of the liquid and the liquid flow through the radiator. The inlet and outlet from the circulation chamber were connected to a face mask, enclosing the naso-buccal area, through a two-way valve.

Preparation of a modified tracheal canula

In order to study the distribution of thermal receptors in the respiratory passages, the Field pattern tracheotomy tube was replaced by a modified tracheal canula with a diaphragm between the upper and lower ends of the tube. The modified tracheal tube (figure 4b) was prepared from the tracheal tube mold (figure 4a), similar to the one described by Waldo and Hoernicke (1961) with slight modifications. The mold was made in the machine shop[1] using round steel rods of appropriate dimensions. The right angle joints were held together by screws through the base of a cold-rolled steel bar. The upper surface of the upper right angle joints were ground off, so as to facilitate easier insertion of the resulting plastic cast into the trachea. The plastic cast was prepared according to the method described by Waldo and Hoernicke (1961).

Recording systems

Carotid blood temperatures. An enamel-insulated copper-constantan thermocouple (30 S.W.G.), heat sealed at the junction head, was introduced into the carotid artery through an 18 gauge hypodermic needle and passed down the

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[1]Department of Animal Science, University of Manitoba, Winnipeg, Canada.

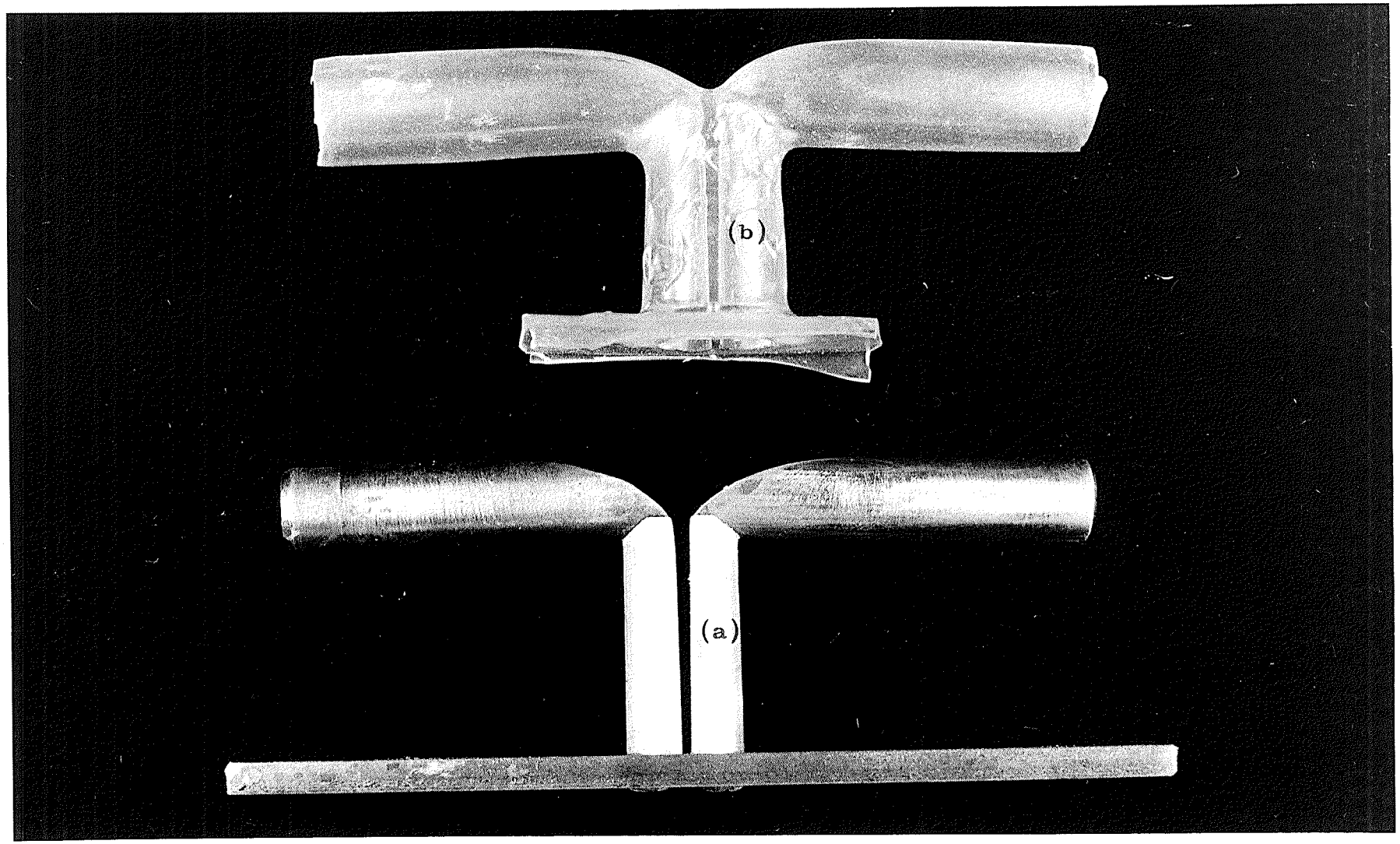

Fig. 4. Modified tracheal canula. (a) tracheal tube mold. (b) plastic cast made from mold.

lumen of the artery until the thermojunction was estimated to be lying approximately 5-7 cm from the bicarotid trunk. After insertion of thermocouple, the hypodermic needle was withdrawn and the thermocouple was held in position by adhesive tape wound around the thermocouple and the carotid loop. The lead wires were connected to a 'Micrograph' recorder[1] BD-I series. The calibrated accuracy of the recording instrument was 0.01°C and the response time for full scale travel of 21 cm chart was less than a second. The thermocouples for measuring carotid blood temperatures were calibrated over a temperature range of 35°C to 45°C against a standard glass and mercury thermometer with known NBS[2] calibration to within 0.01°C, in a well stirred water bath. This calibration held both when the temperature of the bath was constant and when it was rising or falling at the rate of 1°C/min, which is in excess of the highest rate of rise or fall of carotid blood temperature that occurred in any of the experiments.

Jugular blood temperatures were measured with a thermistor probe (time constant-0.6 sec) embedded in the tip of a 22 gauge hypodermic needle, 10 cm in length. The jugular temperatures were recorded by inserting the hypodermic probe into the jugular vein in the left, midneck

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[1]Kipp & Zonen, Delft, Holland.

[2]National Bureau of Standards

region. The position of the needle in the vein was confirmed by a sudden rise in the direct recording thermistor bridge instrument as the needle passed through the skin into the vein. The needle was held in this position with the help of adhesive tape.

Skin temperatures were measured on the lumbar region and the pinna of the left ear (each site was $1\frac{1}{2}$ cm in diameter). The hair at each location was clipped and the skin shaved to promote good contact by a 'Banjo' type thermistor (time constant-0.8 sec) employed for measuring skin temperature. The flat end of the probe containing the sensing element was held in close contact with the skin by adhesive rubber latex at each location.

Rectal temperatures were measured with a rectal thermistor probe (time constant-0.8 sec). The portion inserted into the rectum was 10 cm long.

Tracheal temperatures were measured with a tubular thermistor probe (time constant-3.2 sec). The probe was inserted through the tracheal canula. The tip of the probe containing the sensing element was lying free without touching the tracheal wall.

Air temperatures of the independent air circulation chamber were measured by a thermistor embedded in the tip of a 5/32" diameter stainless steel tube, 10 cm in length (time constant-3.7 sec).

All the aforesaid thermistor probes were plugged into a direct reading bridge thermistor instrument (YSI

12- channel telethermometer[1]). The whole system was accurate to within 0.1°C.

Respiration rates were measured by a Physiograph.[2] The recording channel consists essentially of 3 parts, the transducer (impedence pneumograph), the processor (amplifier), and the reproducer (recorder). The impedence pneumograph picks up the mechanical movements of the flank through needle electrodes fixed on the flank and converts them into an electrical signal. The amplifier enlarges the signal and forwards it to the recording pen, which reproduces the signal in the form of a graphic record.

Heart rates were determined from electrocardiograms with the aid of the Physiograph. Two plate electrodes were fastened on either side of the chest by means of an elastic rubber strap. The wool beneath the plate electrodes was closely shorn and good contact established between electrodes and skin by means of electrode paste. The lead wires were connected to cardiac pre-amplifier which in turn was forwarded to the recording system of the Physiograph.

The heart and respiration rates were recorded for a continuous period of 2 minutes at 5 minute intervals. The graphic records were later analysed and expressed as heart and respiration rates/minute for each 5 minutes.

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[1]Yellow Springs Instrument Co., Yellow Springs, Ohio, U.S.A.

[2]E & M Intrument Co., Inc. Houston, Texas 77021, U.S.A.

RESULTS

RESPONSES OF UNSHORN AND SHORN SHEEP

The changes in the respiratory rates of partially shorn and unshorn sheep are presented in table 2. The mean values of the respiratory rates show a gradual decrease as the shorn area was progressively increased from 1000 cm^2 to 3000 cm^2. The maximum depression in respiratory rates occurred when an area of 3000 cm^2 was closely shorn.

If the shorn area (3000 cm^2) was covered with a sheep skin coat (5 cm wool length) overnight and during the experiment, the respiratory rates reverted to that of the unshorn sheep (table 2). Reexposing the shorn area resulted in an immediate depression of respiratory rates.

The respiratory rates decreased from a steady rate of 29-31/minute in unshorn sheep (10 cm fleece) to 21-23/minute in fully shorn sheep. The depression in respiratory rate was very similar to that described by Bligh (1963b). Since there was practically no difference in the respiratory rates of fully shorn sheep and partially shorn sheep (3000 cm^2 shorn), whenever the effect of fleece on respiratory responses were studied, under various test conditions, the latter were used.

The various tissue temperatures and heart rates of partially shorn and unshorn sheep are presented in table 3.

TABLE 2

REMOVAL OF FLEECE IN STAGES ON MEAN* RESPIRATORY FREQUENCY

Time (minutes)	Unshorn	Area shorn (cm^2)						
		1000	1500	2000	2500	3000	fully shorn	3000 (covered with sheep skin coat)
0	30	30	30	27	27	22	22	30
30	31	29	30	28	29	23	22	29
60	31	30	28	29	29	22	22	31
90	30	31	28	29	26	23	23	30
120	31	29	29	28	28	23	22	29
150	31	31	28	28	28	23	22	30
180	31	29	27	29	27	22	21	29

*Mean of 3 experiments on each of 4 sheep

TABLE 3

MEAN* VALUES OF VARIOUS BODY TISSUE TEMPERATURES (°C) AND HEART RATES(PER MINUTE) OF UNSHORN AND PARTIALLY SHORN (3000 cm^2) SHEEP AT AMBIENT TEMPERATURE OF 20°C AND 55% RELATIVE HUMIDITY

	Unshorn		Partially shorn	
	Mean	±S.D.	Mean	±S.D.
Carotid blood temperature	38.51	0.04	38.61	0.05
Jugular blood temperature	38.44	0.07	38.51	0.06
Lumbar skin temperature	38.17	0.27	32.67	0.43
Rectal temperature	39.28	0.09	39.46	0.10
Heart rate	70	1.40	70	2.00

*Mean of 3 experiments on each of 4 sheep

There were no marked differences in the carotid and jugular blood temperatures of unshorn and partially shorn sheep. The skin temperature at the lumbar region in unshorn sheep was about 2.11°C and 1.34°C lower than the rectal and carotid blood temperature, respectively. However, the lumbar skin temperature of partially shorn sheep was markedly lower (4.5°C) than that of unshorn sheep.

EFFECT OF HEATING THE PERINEAL REGION

The perineal heat treatment was carried out in the climatic chamber at 20/55, 15/55 and 10/55 (dry bulb temperature, °C/ relative humidity, %) on unshorn and partially shorn sheep (3000 cm^2). The cardio-respiratory activities and various tissue temperature responses were recorded. The perineal skin was heated to about 40°C at a rate of 0.5°C/minute.

Responses at 20°C/55%

The behaviour of the cardio-respiratory activities and carotid blood temperatures before and after heating the perineal region of unshorn and partially shorn sheep are illustrated in figure 5. The immediate effect of heating the perineal skin of fully fleeced sheep was an increase in the respiratory rate. Prior to heating, the mean perineal skin temperatures and respiratory rates were 30.80°C and 31/minute, respectively. The respiratory response started almost immediately following perineal

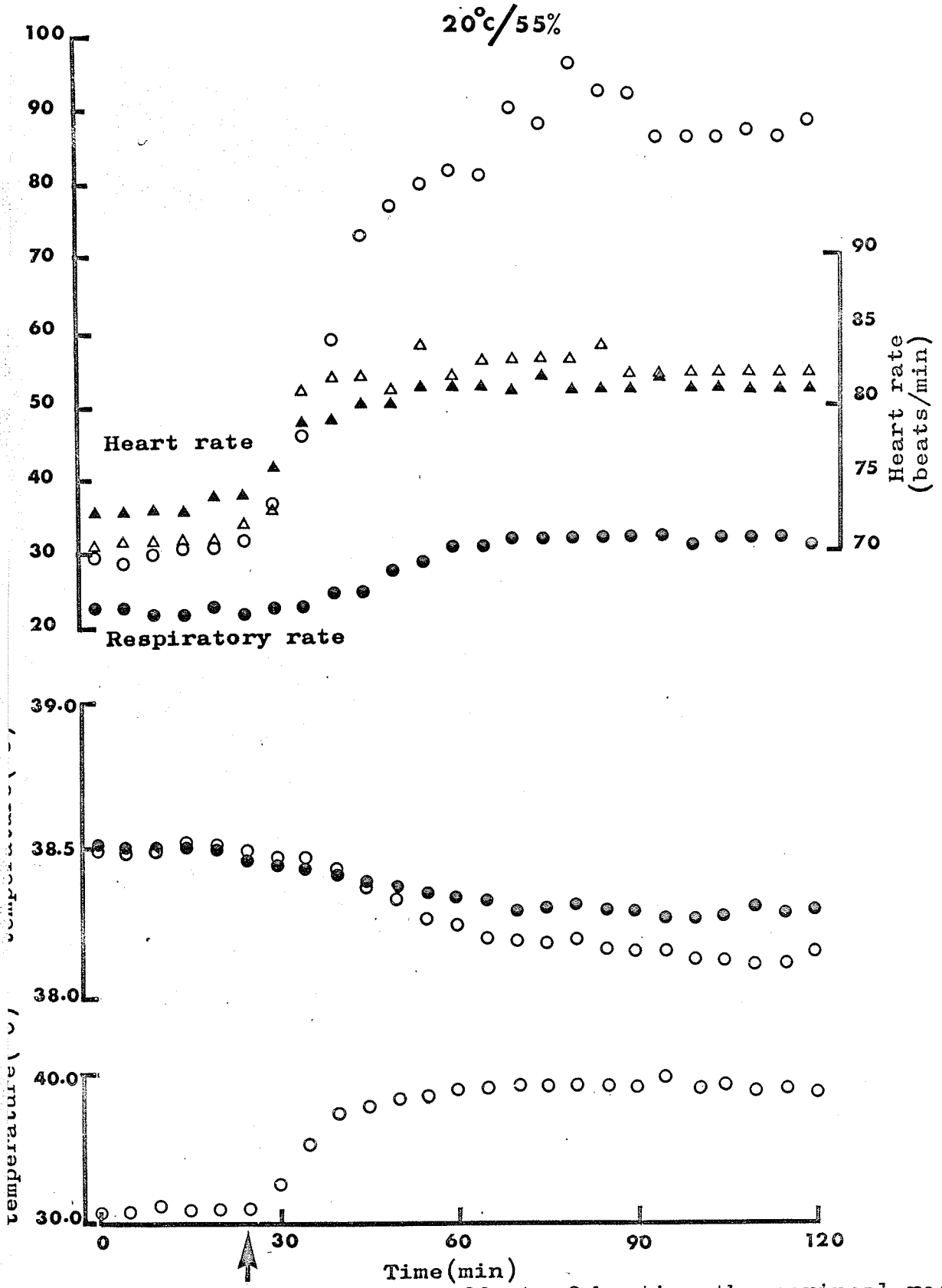

Fig.5. A comparison of the effect of heating the perineal region of unshorn(open symbols) and partially shorn(black symbols)sheep on cardio-respiratory activities and carotid blood temperature. Each plot is the mean of 3 experiments on each of 4 sheep. The arrow marks the point where perineal heat treatment was applied.

heating. Between perineal temperatures of 31°C and 38°C, the respiratory rate increased rapidly at the rate of 8 respirations/°C rise in perineal skin temperature. Peak values (96 respirations/minute) were attained 20 minutes after the perineal skin temperatures had reached 39°C. From then on there were slight fluctuations in respiratory rates during the remainder of the experimental period. In partially shorn sheep, only a small rise in respiratory rates occurred after a delay of about 20-25 minutes. The peak (32 respirations/minute) did not exceed the respiratory rate of unshorn sheep prior to perineal heat treatment.

The mean heart rates of unshorn sheep were 71 beats/minute before perineal heat treatment and increased to 84 beats/minute following perineal heating. The rise in heart rates closely paralleled the rise in perineal skin temperature and levelled off at perineal skin temperature of 38°C. The heart rates of partially shorn sheep followed those of unshorn sheep in all experiments.

The mean carotid blood temperature before perineal heat treatment in unshorn sheep was 38.51°C (figure 5). Following perineal heating, there was a gradual decline in carotid blood temperature to a minimum of 38.15°C. The decline was more pronounced during the initial stages of perineal heating, when the perineal skin temperature was rising from 32°C to 39°C. From then onwards there were slight variations during the remainder of the experimental

period. The marked fall in carotid temperature during initial stages of perineal heating was associated with a rapid rise in respiratory rates during the same period. Partially shorn sheep also showed a decline in carotid blood temperature. The decline was of lesser magnitude than that of unshorn sheep and extended over a longer period of time.

Jugular blood temperature (figure 6) showed a sharp decline of 0.30°C after perineal heat treatment in unshorn sheep, which is somewhat similar to the fall of carotid blood temperature. Partially shorn sheep also showed a decline following perineal heat treatment but the decline was more gradual and extended over the entire experimental period.

The lumbar skin temperature of both unshorn and partially shorn sheep showed a gradual decline (figure 6). The mean decline in unshorn and partially shorn sheep being 1°C and 2°C, respectively.

Responses at 15°C/55%

The respiratory rates increased at ambient temperature of 15°C following perineal heating of unshorn sheep (figure 7) but the responses differed from those in sheep exposed to 20°C ambient temperature in that there was a delay of 15 minutes before the onset of panting. The respiratory rates increased rapidly after this delay and reached a maximum of 76/minute. In partially shorn sheep, there was a small rise in respiratory rates

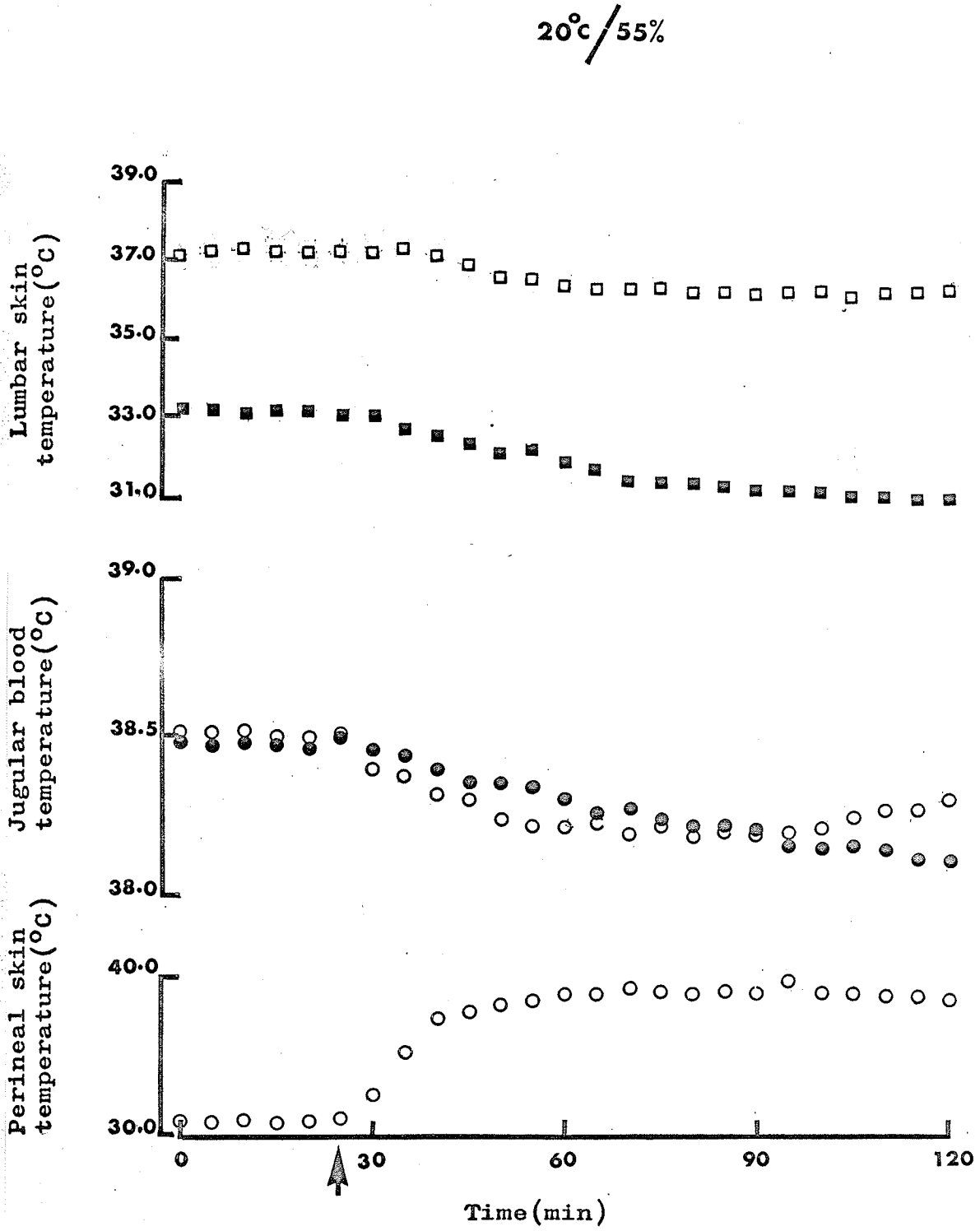

Fig.6. A comparison of the effect of heating the perineal region of unshorn(open symbols) and partially shorn(black symbols) sheep upon lumbar skin and jugular blood temperatures. Each plot is the mean of 3 experiments on each of 4 sheep. The arrow marks the point where perineal heat treatment was applied.

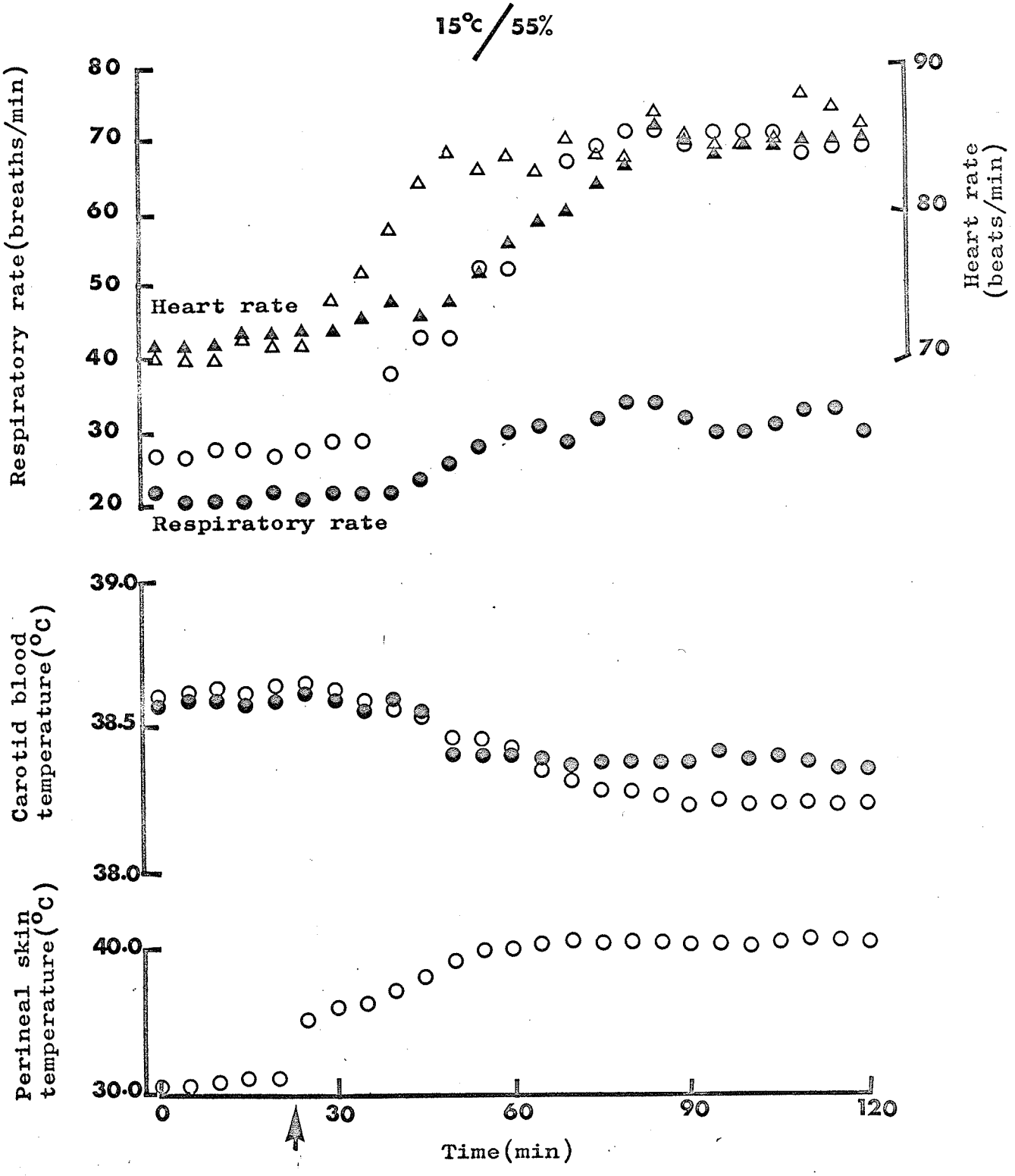

Fig.7. A comparison of the effect of heating the perineal region of unshorn(open symbols) and partially shorn(black symbols) sheep on the cardio-respiratory activities and carotid blood temperature. Each plot is the mean of 3 experiments on each of 4 sheep. The arrow marks the point where perineal heat treatment was applied.

following perineal heating but this did not exceed that of unshorn sheep before perineal heat treatment. The heart rates of unshorn sheep rose from a pretreatment level of 72 beats/minute to 87 beats/minute following perineal heat treatment. This rise was rapid and reached a level of 84 beats/minute even before perineal skin temperature had reached 39°C. In partially shorn sheep, the rise in heart rate was gradual and approximated that of unshorn sheep about an hour after perineal heating had commenced.

A decline in carotid blood temperature, similar to that of the 20°C ambient temperature experiment, occurred following perineal heat treatment at 15°C, but the decline was more pronounced and extended over a period of 80 minutes. In partially shorn sheep there was a sharp fall in carotid blood temperature after a 10 minute delay, and the temperature remained fairly steady with only minor fluctuations.

Jugular blood temperatures also declined gradually by 0.35°C in both unshorn and partially shorn sheep (figure 8). The decline was more pronounced during initial stages of perineal heat treatment in unshorn sheep whereas, in partially shorn sheep, the decline occurred during later stages.

The lumbar skin temperature was almost the same before and after perineal heating in unshorn sheep (figure 8) but the ear skin temperature rose from 19°C

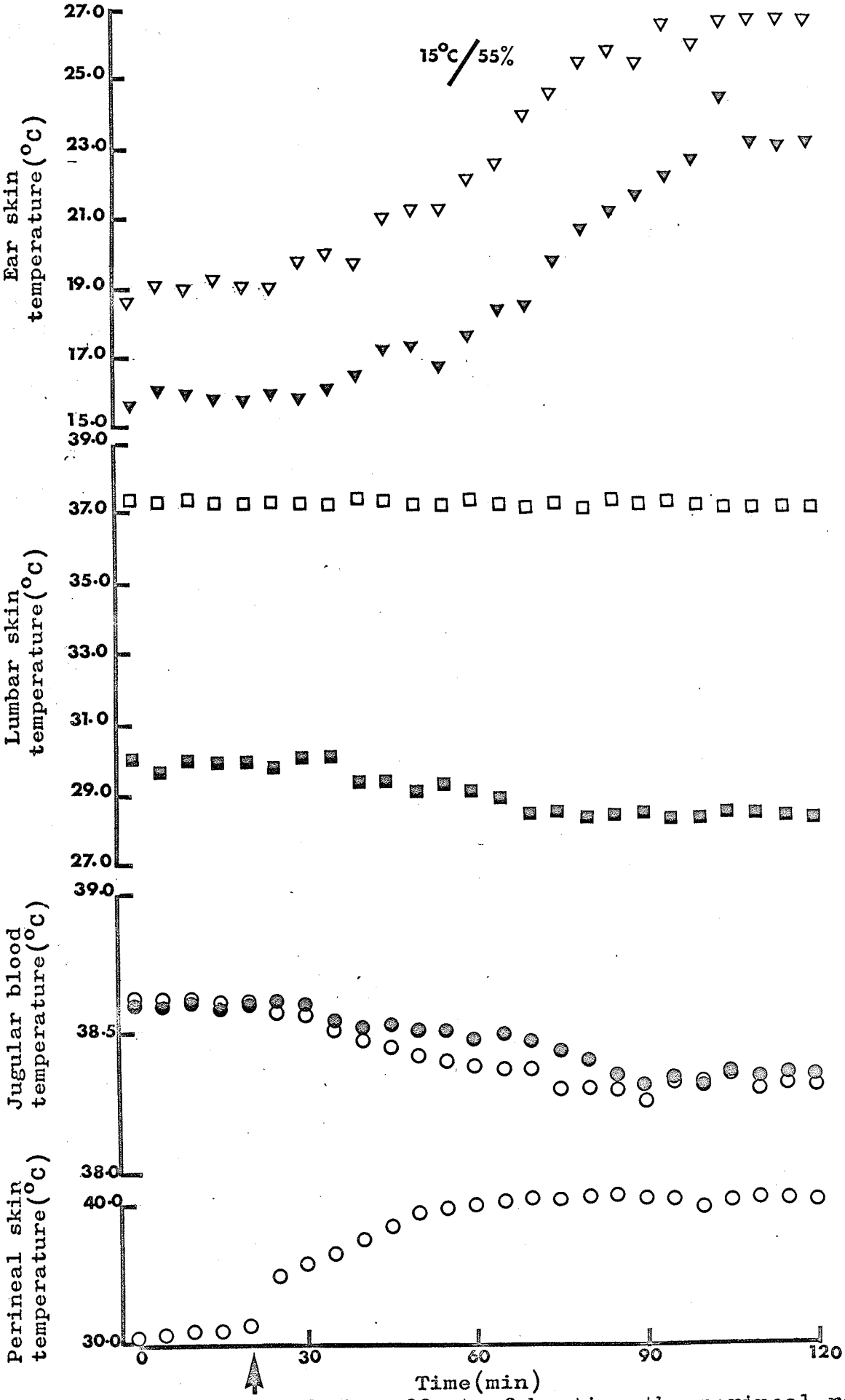

Fig.8. A comparison of the effect of heating the perineal region of unshorn(open symbols) and partially shorn(black symbols) sheep on lumbar and ear skin, and jugular blood temperatures. Each plot is the mean of 3 experiments on each of 4 sheep.The arrow marks the point where perineal heat treatment was applied.

before heating to 26°C following heating. The increase occurred immediately following perineal heating and extended over a period of 60 minutes. The lumbar skin temperature declined following heat treatment in partially shorn animals but ear skin temperature increased by 8°C. The rise in ear skin temperature closely paralleled that of unshorn sheep.

Responses at 10°C/55%

The respiratory response of unshorn sheep before and after perineal heating is illustrated in figure 9. The mean respiratory rate at 10°C of unshorn sheep which was 25 respirations/minute before perineal heat treatment increased to 63 respirations/minute following perineal heating. The respiratory responses at 10°C differed from those at 15°C ambient temperature in the following respects: At 10°C ambient temperature (a) a slightly lower mean rate was observed before perineal heat treatment, (b) the onset of polypnoea due to perineal heating was delayed for a period of 30 minutes and (c) the respiratory responses obtained following perineal heat treatment were of lesser magnitude. In partially shorn sheep, there were no differences in respiratory frequency before and after perineal heat treatment. Heart rates, however, increased both in unshorn and partially shorn sheep following perineal heating.

There was a progressive decline in carotid blood

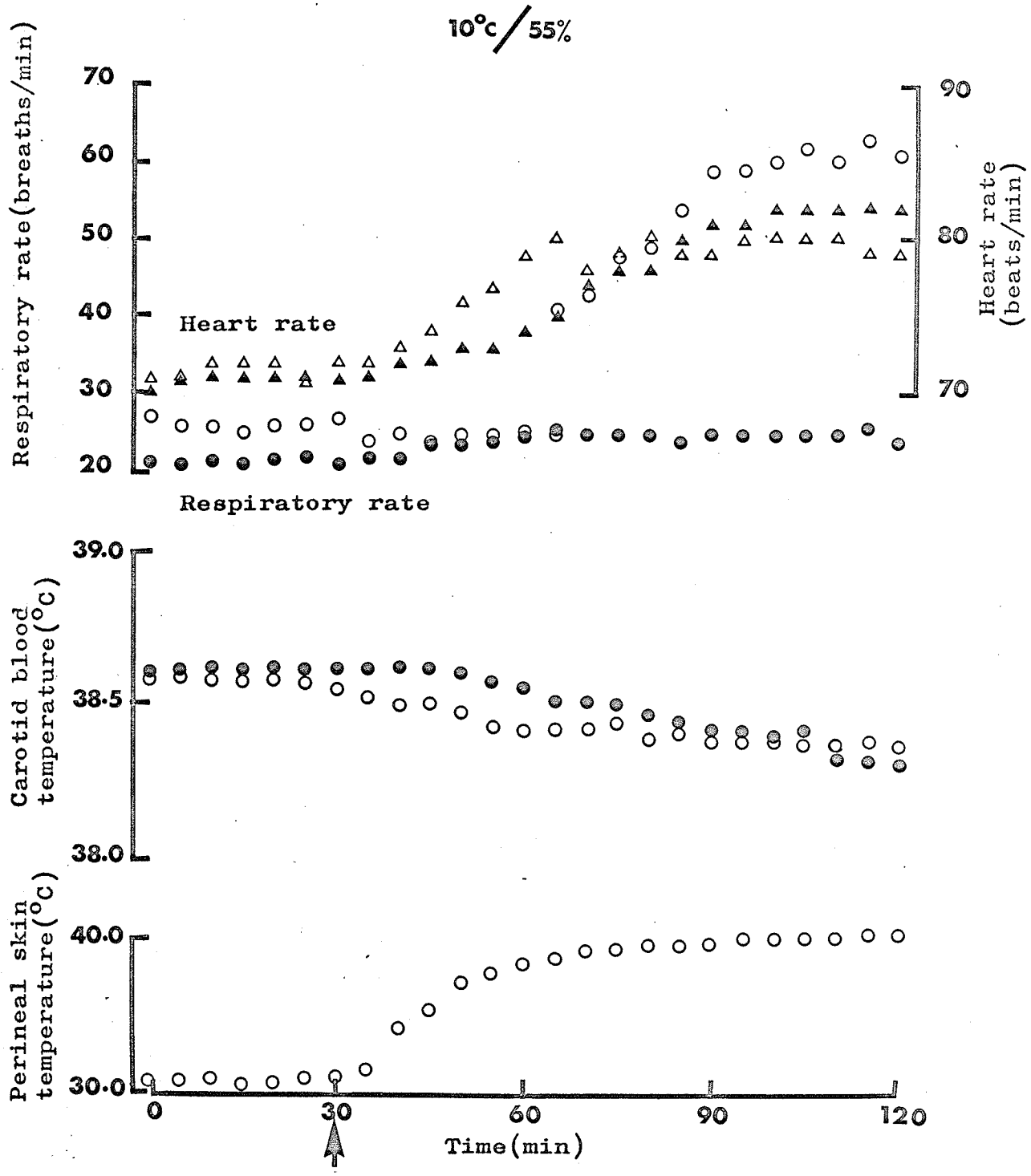

Fig.9. A comparison of the effect of heating the perineal region of unshorn(open symbols) and partially shorn(black symbols) sheep upon the cardio-respiratory activities and carotid blood temperature. Each plot is the mean of 3 experiments on each of 4 sheep. The arrow marks the point where perineal heat treatment was applied.

temperature following perineal heating of unshorn sheep. The maximum fall of 0.20°C, occurred immediately following perineal heat treatment. It is interesting to note that the carotid blood temperature declined before the onset of panting in unshorn sheep. In partially shorn sheep, which did not show any respiratory responses, the carotid blood temperature also declined sharply (0.20°C).

Jugular blood temperature of unshorn and partially shorn sheep (figure 10) closely paralleled each other before and after perineal heat treatment.

A concise account of the changes in the temperature of the blood and skin, and cardio-respiratory responses at various ambient conditions before and during perineal heat treatment of unshorn and partially shorn sheep are given in tables 4 and 5 respectively. In general, perineal heating resulted in the initiation of polypnoea in unshorn sheep, whereas in partially shorn sheep no such responses were observed. With a decrease in ambient temperature from 20°C to 10°C, unshorn sheep showed a delay in the initiation of polypnoea and a decrease in the magnitude of respiratory response, following perineal heat treatment.

Perineal heat treatment resulted in a marked rise in respiratory frequency, ear skin temperature and heart beats of unshorn sheep at all ambient temperatures, whereas in partially shorn sheep the ear skin temperature and heart beats also increased but was not accompanied by a similar

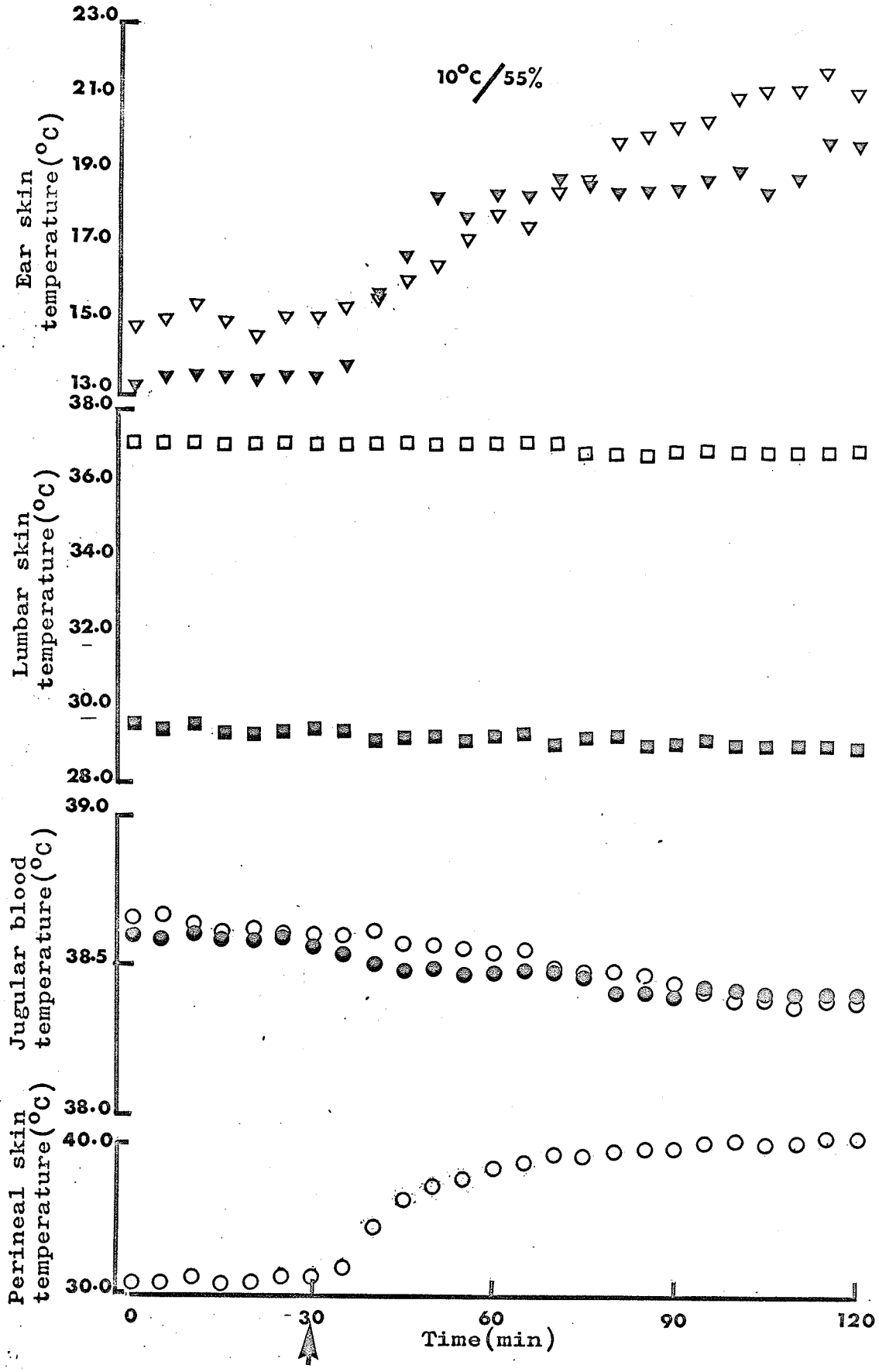

Fig.10. A comparison of the effect of heating the perineal region of unshorn(open symbols) and partially shorn(black symbols) sheep upon lumbar and ear skin, and jugular blood temperatures. Each plot is the mean of 3 experiments on each of 4 sheep. The arrow marks the point where perineal heat treatment was applied.

TABLE 4

MEAN* CHANGES IN TEMPERATURE (°C) OF INTRAVASCULAR AND SKIN, AND RESPIRATORY AND HEART RATES OF UNSHORN SHEEP BEFORE AND DURING PERINEAL HEATING AT VARIOUS AMBIENT TEMPERATURES

	Before heat treatment						During heat treatment					
Ambient temperature(°C)	20		15		10		20		15		10	
Relative humidity(%)	55		55		55		55		55		55	
	Mean	±S.D.	Mean	±S.D.	Mean	±S.D.	Mean	±S.D.	Mean	±S.D.	Mean	±S.D.
Carotid blood temperature	38.49	0.06	38.65	0.03	38.55	0.02	38.16	0.07	38.23	0.02	38.37	0.03
Jugular blood temperature	38.51	0.08	38.63	0.04	38.60	0.04	38.27	0.04	38.33	0.04	38.37	0.03
Lumbar skin temperature	38.21	0.26	37.33	0.08	37.7	0.08	36.21	0.35	37.15	0.59	36.80	0.07
Ear skin temperature	-	-	19.00	1.98	15.08	0.66	-	-	26.75	1.51	21.09	0.98
Heart rate per minute	72	1.40	71	1.73	72	1.20	82	2.30	88	1.00	79	1.00
Respirations per minute	32	2.40	27	1.00	25	1.41	86	8.80	68	2.57	61	4.59

*Mean of 3 experiments on each of 4 sheep

TABLE 5

MEAN* CHANGES IN TEMPERATURE (°C) OF INTRAVASCULAR AND SKIN, AND RESPIRATORY AND HEART RATES OF PARTIALLY SHORN SHEEP BEFORE AND DURING PERINEAL HEATING AT VARIOUS AMBIENT TEMPERATURES

	Before heat treatment						During heat treatment					
Ambient temperature (°C)	20		15		10		20		15		10	
Relative humidity (%)	55		55		55		55		55		55	
	Mean	±S.D.	Mean	±S.D.	Mean	±S.D.	Mean	±S.D.	Mean	±S.D.	Mean	±S.D.
Carotid blood temperature	38.48	0.08	38.59	0.08	38.63	0.07	38.28	0.04	38.38	0.10	38.32	0.04
Jugular blood temperature	38.46	0.08	38.63	0.06	38.57	0.04	38.10	0.12	38.34	0.09	38.39	0.03
Lumbar skin temperature	33.10	0.64	29.79	0.68	29.54	0.50	31.06	0.44	28.59	0.71	28.96	0.47
Ear skin temperature	-	-	15.80	1.36	13.47	0.98	-	-	22.98	2.08	19.69	2.70
Heart rate per minute	74	1.00	72	1.00	71	1.78	81	2.00	85	2.86	82	1.00
Respirations per minute	22	1.70	22	1.83	21	0.63	32	3.60	33	3.45	24	1.82

*Mean of 3 experiments on each of 4 sheep

rise in respiratory frequency. The carotid blood temperatures declined both in unshorn and partially shorn sheep at all ambient temperatures but the magnitude of decline was greater in unshorn sheep than in shorn sheep. The onset of polypnoea in unshorn sheep as a result of perineal heating, in spite of a decline in carotid blood temperature, indicates the presence of thermal receptors in the perineal region.

EFFECT OF HEATING THE FLANK REGION

Experiments were conducted to see whether flank heating, at an ambient temperature of 20°C had any effect on respiratory patterns and various tissue temperatures. An area of about 900 cm^2 (30 x 30 cm) was closely shorn and heated to 40°C. The results are presented in figure 11. No marked rise in respiratory rate occurred following flank heating. Prior to heat treatment the respiratory rate ranged from 28-30 respirations/minute and reached a maximum of only 37/minute following heat treatment. The rise in ear skin temperature of 4°C during flank heating, indicated a condition of vasodilatation. The rise in the ear skin temperature was accompanied by a slight fall (0.15°C) in carotid blood temperature.

SHORT TERM EXPOSURE TO RISING AMBIENT TEMPERATURE

Each sheep spent the first 30 minutes in the climatic chamber preset at 20°C and 55% relative humidity. The chamber temperature was then raised to about 40°C and

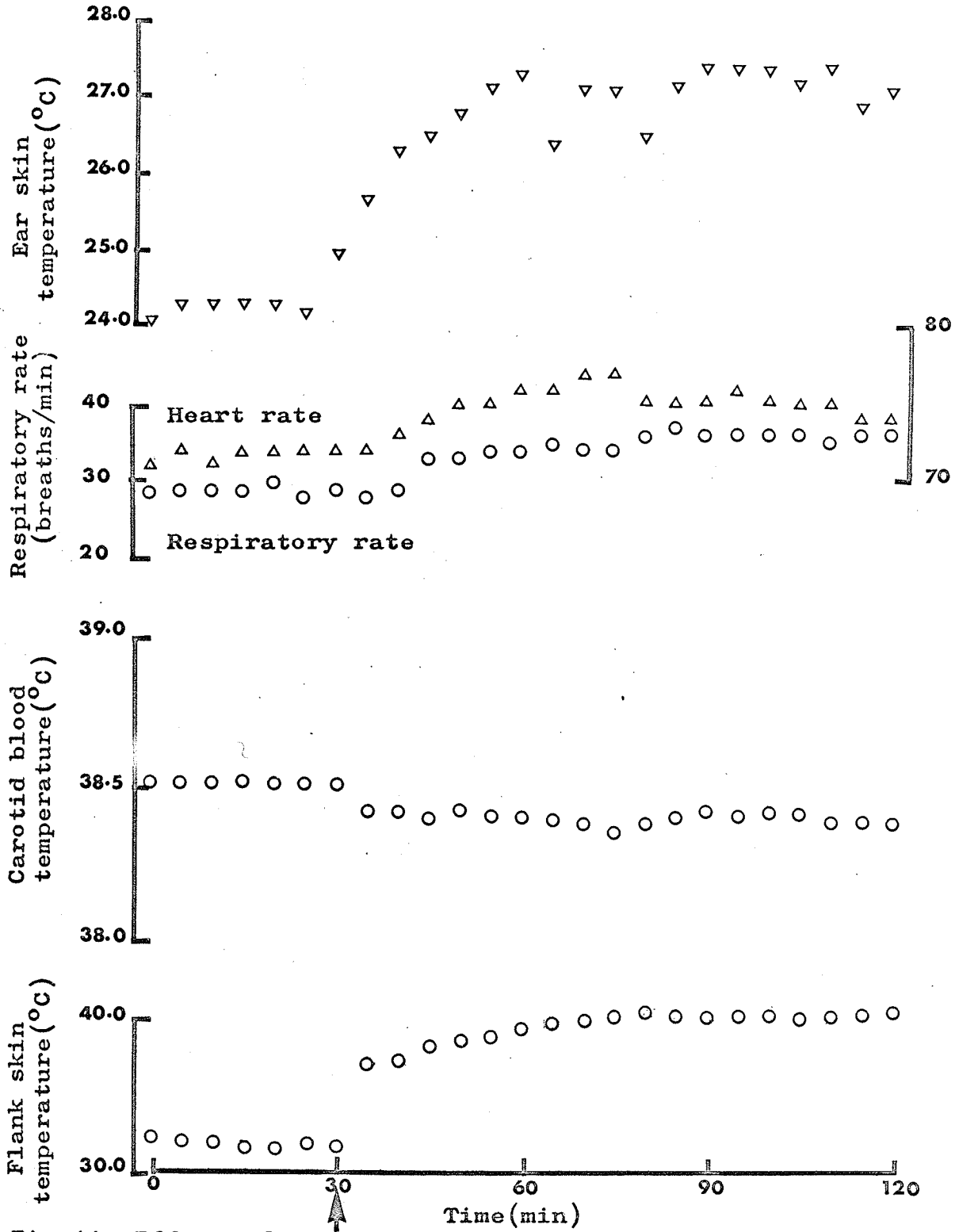

Fig.11. Effect of heating the flank skin of unshorn sheep upon cardio-respiratory activities, ear skin and carotid blood temperatures. Each plot is the mean of 2 experiments on each of 4 sheep. The arrow marks the point where flank heat treatment was applied.

maintained at that level for 90 minutes. The chamber temperature rose at the rate of 0.5°C/minute. Relative humidity was not controlled when the chamber temperature was rising and varied between 40 and 50%. After attaining 40°C, the relative humidity of the chamber remained close to 52%.

Figure 12 gives the mean changes in the respiratory rates, heart rates and carotid blood temperature of unshorn and partially shorn sheep, under the above conditions. The mean respiratory rate of unshorn sheep was 31/minute at 20°C and progressively increased reaching a maximum of 110/minute at 40°C. In partially shorn sheep the respiratory rate was 21 to 22/minute at 20°C and continued to be within this range for about 45 minutes, after the ambient temperature began to rise. After this delay, there was an abrupt rise and the respiratory rate then approached that(110 respirations/minute) of unshorn sheep.

The carotid blood temperature of unshorn sheep varied between 38.43°C and 38.53°C at 20°C ambient temperature and increased only after the onset of polypnoea. Conversely, the rise in carotid blood temperature in partially shorn sheep preceded the respiratory rise.

The skin temperatures (figure 13) at the lumbar region of unshorn sheep increased by only 1°C during the rise in ambient temperature and were 2°C lower than the ambient temperature during the final stages of the experi-

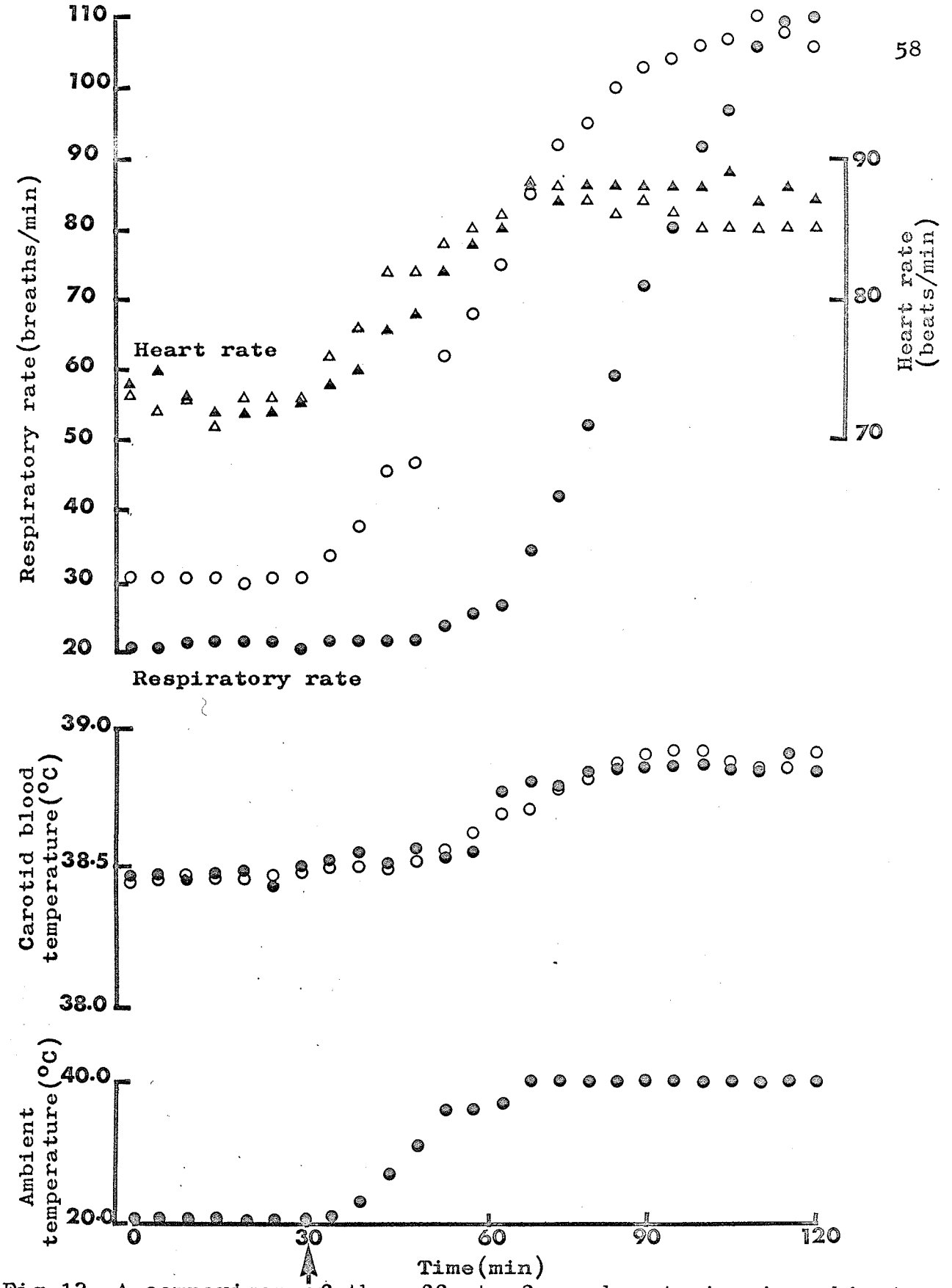

Fig.12. A comparison of the effect of an abrupt rise in ambient temperature from 20°C to 40°C in unshorn(open symbols) and partially shorn(black symbols) sheep upon cardio-respiratory activities and carotid blood temperature. Each plot is the mean of 3 experiments on each of 4 sheep. The arrow marks the point where ambient temperature started to rise.

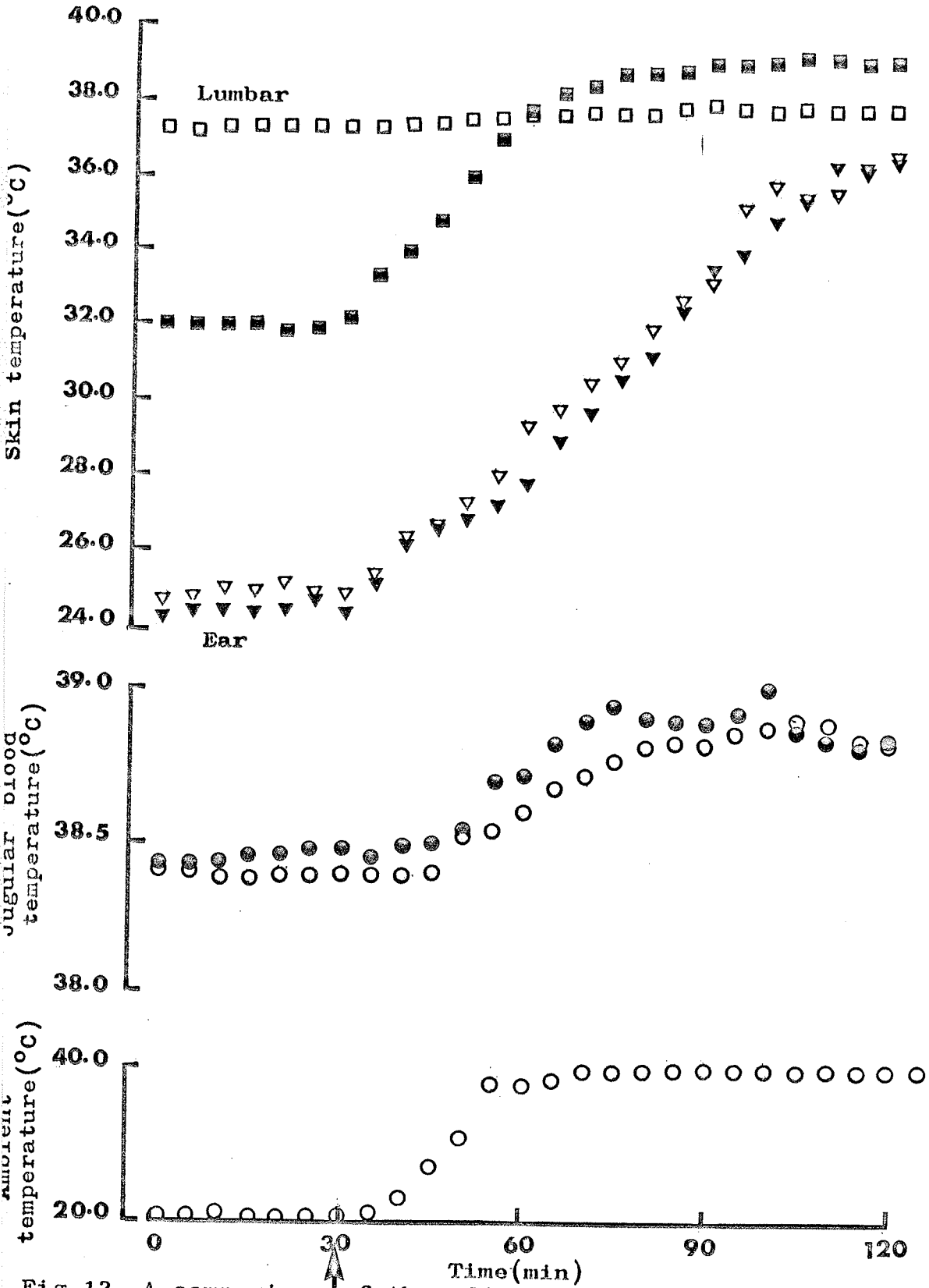

Fig.13. A comparison of the effect of an abrupt rise in ambient temperature from 20°C to 40°C in unshorn(open symbols) and partially shorn(black symbols) sheep, upon lumbar and ear skin, and jugular blood temperatures. Each plot is the mean of 3 experiments on each of 4 sheep. The arrow marks the point where the ambient temperature started to rise.

mental period. The lumbar skin temperature of partially shorn sheep, however, closely paralleled the rise in ambient temperature, and in some experiments almost equalled the ambient temperature. Polypnoea was initiated at lumbar skin temperatures of 38°C in partially shorn sheep, and, almost at the same point of the experimental period, an abrupt rise in carotid blood temperature occurred. The ear skin temperature of both unshorn and partially shorn sheep rose markedly along with the rise in ambient temperature from 20°C to 40°C.

There was a slight (0.15°C) rise in jugular blood temperature of unshorn sheep, following the rise in ambient temperature, and it paralleled the rise in carotid blood temperature. The rise in jugular blood temperature of partially shorn sheep was more pronounced and reached a maximum of 38.80°C during the final stages of the experiment.

EFFECT OF DECREASE IN AMBIENT TEMPERATURE

In this series of experiments the controls of the climatic chamber were set to 40°C and 55% relative humidity. After environmental equilibrium was established the sheep was introduced into the chamber and a 2 hour acclimation period allowed. After panting was fully established, the chamber temperature was lowered to 10°C and maintained at that level for 60 minutes. The chamber temperature declined by 15°C in the first 10 minutes and by an additional 15°C

in the next 30 minutes.

The mean changes in the cardio-respiratory activities and various tissue temperatures of unshorn and partially shorn sheep when the chamber temperature was lowered from 40°C to 10°C are shown in figure 14. There were large variations in respiratory rate (107-119/minute) of partially shorn sheep at ambient temperatures of 40°C. When the ambient temperature was lowered, there was a fall in the respiratory rate within the first 10 minutes. The respiratory rate continued to decline until the ambient temperature had reached 10°C. Thereafter it levelled off showing only slight fluctuations. In unshorn sheep, however, there was a delay of about 10 minutes before any decline was apparent in respiratory rate and the rate of decline was more gradual and extended over a longer period of time. The decline in respiratory rate continued even after the ambient temperature had reached 10°C and approached 33-36 respirations/minute during the final 30 minutes of the experiment.

The depression in respiratory rate following the fall in ambient temperature was accompanied by a decline in carotid blood temperature of both unshorn and partially shorn sheep. However, the decline in carotid blood temperature was more pronounced in partially shorn sheep (figure 14).

The lumbar skin temperature (figure 15) of partially shorn sheep at an ambient temperature of 40°C was

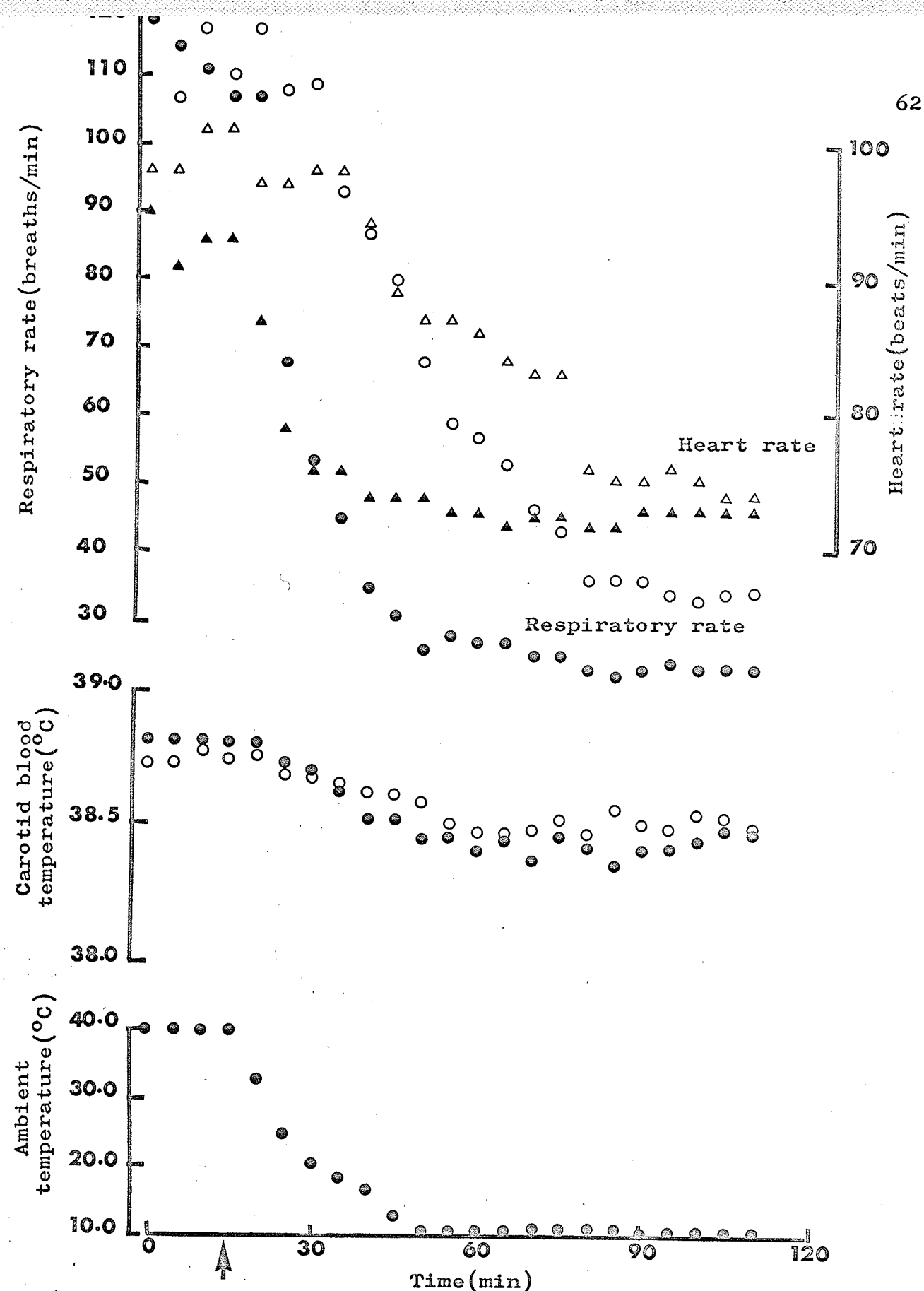

Fig.14. A comparison of the effect of an abrupt fall in ambient temperature from 40°C to 10°C after thermal polypnoea was established in unshorn(open symbols) and partially shorn(black symbols) sheep, upon cardio-respiratory activities and carotid blood temperature. Each plot is the mean of 3 experiments on each of 4 sheep. The arrow marks the point where the ambient temperature started to fall.

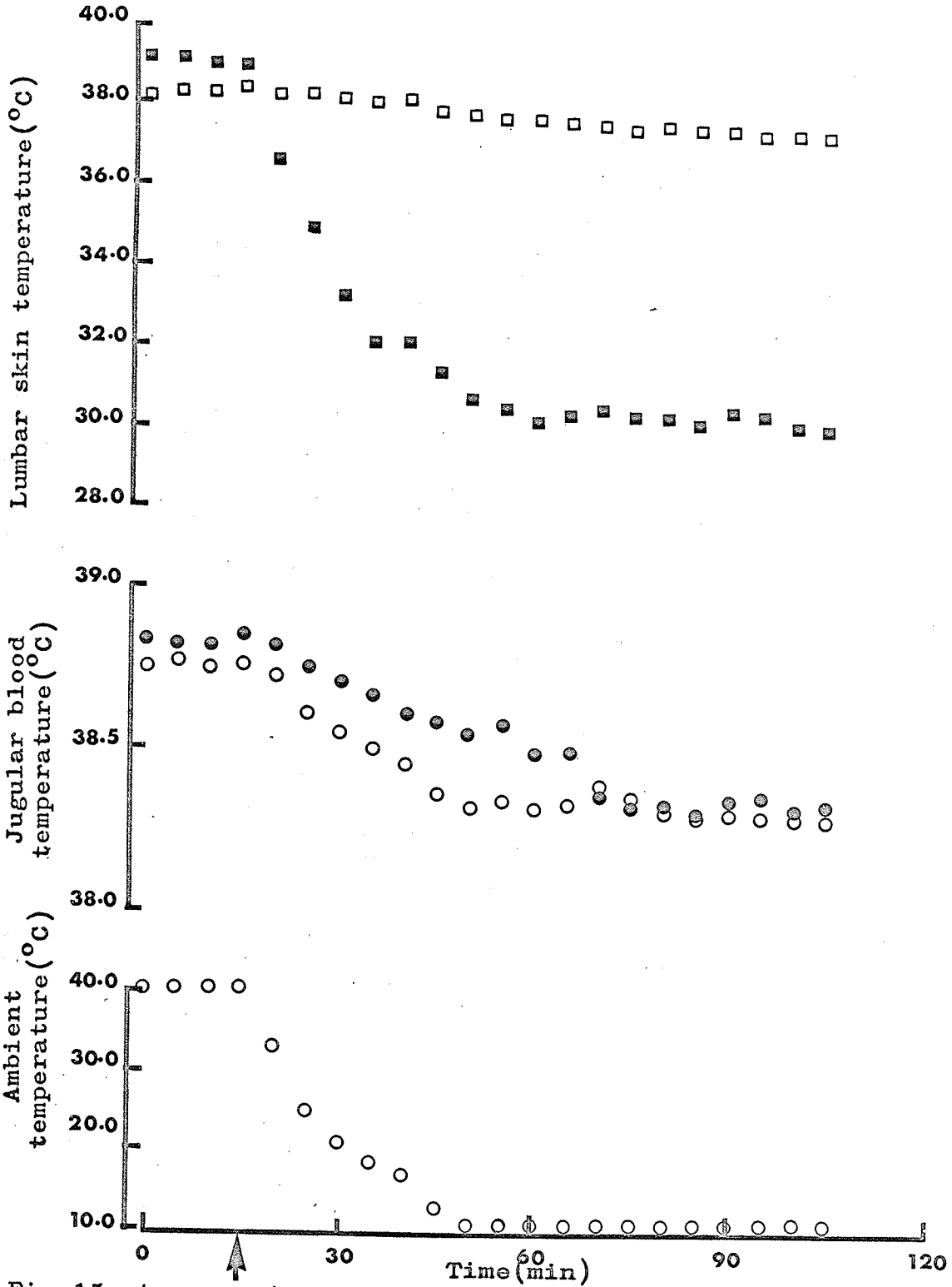

Fig.15. A comparison of the effect of an abrupt fall in ambient temperature from 40°C to 10°C after thermal polypnoea was established in unshorn(open symbols) and partially shorn(black symbols) sheep, upon lumbar skin and jugular blood temperature. Each plot is the mean of 3 experiments on each of 4 sheep. The arrow marks the point where ambient temperature started to fall.

1°C higher than that of the unshorn sheep. As the ambient temperature declined, the lumbar skin temperature of partially shorn sheep declined sharply and this was accompanied by a similar drop in respiratory frequency. The decline in lumbar skin temperature and respiratory rate was almost complete at an ambient temperature of 10°C. However, unshorn sheep showed only a 1°C decline in lumbar skin temperature.

The jugular blood temperature of partially shorn sheep was at a slightly higher level (38.83°C) than that of the unshorn sheep (38.75°C) at 40°C ambient temperature. When the ambient temperature was lowered to 10°C, the jugular blood temperatures of both unshorn and partially shorn sheep declined markedly.

EFFECTS OF LOCALIZED COOLING OF THE TRUNK REGION

Short term exposures to varying ambient temperatures indicated that a fall in ambient temperature was accompanied by a simultaneous fall in respiratory frequency and lumbar skin temperature of partially shorn sheep but a delayed respiratory depression and absence of any change in the lumbar skin temperature of unshorn sheep. When the ambient temperature was raised from 20°C to 40°C, polypnoea was induced in unshorn sheep even in the absence of any changes in the carotid blood temperature. In partially shorn sheep, polypnoea was preceded by a slight rise in carotid blood temperature and a marked

increase in lumbar skin temperature. Since it was not clear whether the onset of polypnoea in partially shorn sheep was due to a slight rise in carotid blood temperature or to a rise in lumbar skin temperature, an area of approximately 900 cm^2 (30 x 30 cm) on the trunk region was cooled to about 10°C, after thermal polypnoea had been established, to see if any changes occur in the respiratory rates and carotid blood temperature. The pattern of mean respiratory rates and carotid blood temperature are illustrated in figure 16. Upon cooling the lumbar region, there was an immediate drop in the respiratory frequency from 108 -113/minute to 40-45/minute. The decline was complete at a lumbar skin temperature of 10°C, fluctuated slightly for about 30 minutes, and again approached a level of 80/minute even though the lumbar skin temperature was maintained at 10°C. It was interesting to note that these changes in respiratory rates, brought about by lumbar cooling, were not accompanied by any marked changes in the carotid blood temperature throughout the experimental period.

INDEPENDENT NASO BUCCAL AIR CIRCULATION EXPERIMENTS

Effect of raising naso-buccal air temperature

Experiments were conducted in which the air in the face mask was raised to 40°C but the ambient temperature in the chamber was maintained at 20°C and 55% relative humidity. The typical changes in respiration and heart

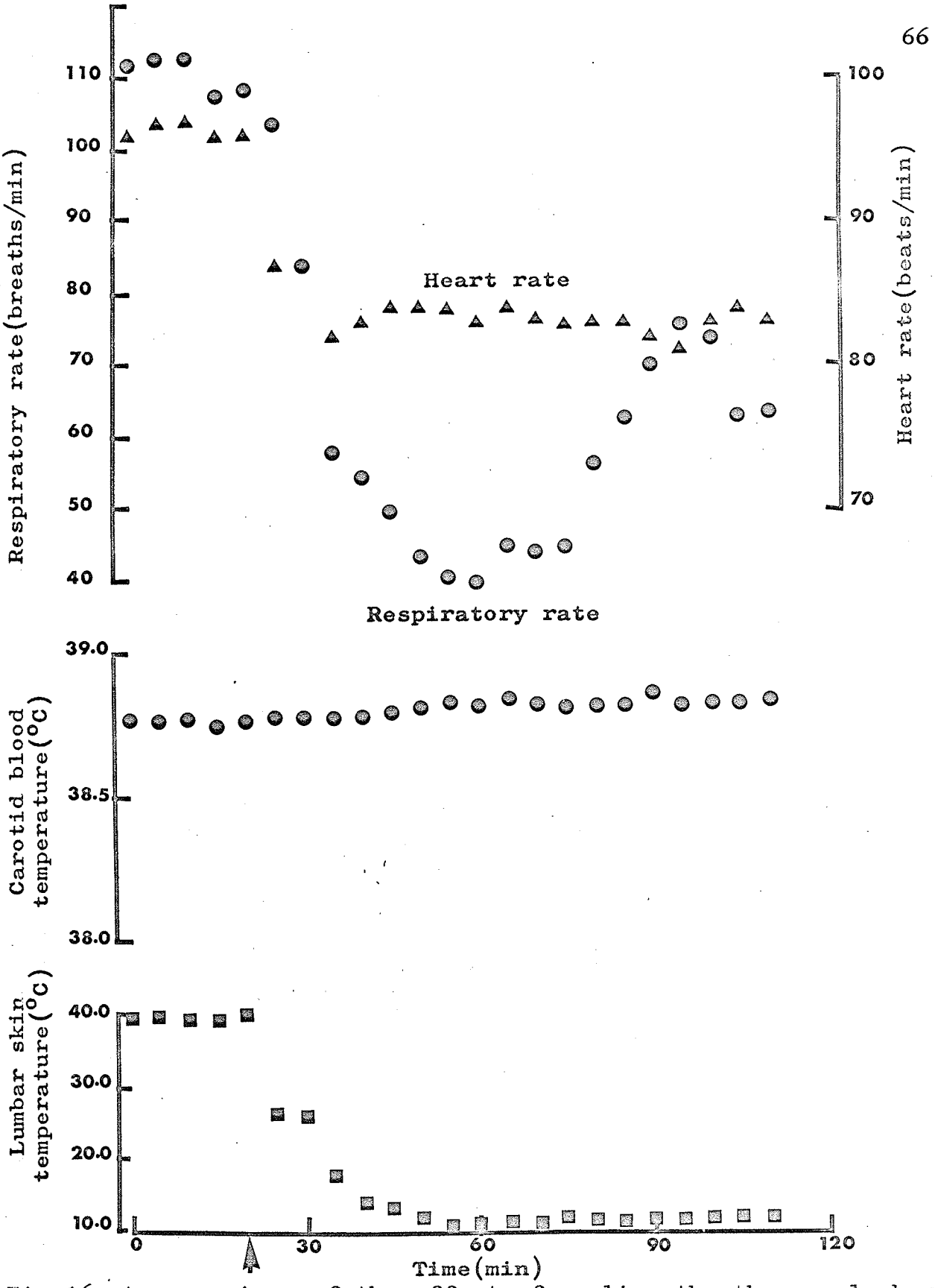

Fig.16. A comparison of the effect of cooling the thoraco-lumbar in partially shorn sheep after thermal polypnoea was established at 40°C ambient temperature, upon the cardio-respiratory activities and carotid blood temperature. Each plot is the average of 2 experiments on each of 4 sheep. The arrow marks the point where thoraco-lumbar cooling commenced.

rates and carotid blood temperatures in unshorn and partially shorn sheep are presented in figure 17. Following the increase in the temperature of the naso-buccal air, in unshorn sheep, the respiration rates increased almost immediately from a pretreatment range of 30-32/minute to a maximum of 80/minute at the end of 60-65 minutes and levelled off thereafter. Partially shorn sheep showed only small respiratory responses, the maximum rise being 34/minute. The onset of polypnoea in unshorn sheep was accompanied by a slight decline (0.15°C) in carotid blood temperature whereas in partially shorn sheep the decline was more pronounced (0.25°C).

While there was a decline in carotid blood temperature, the temperature of the jugular blood (figure 18) increased slightly (0.15°C) in both unshorn and partially shorn sheep. The ear skin temperature increased steadily from a mean of 24.5°C to a mean of 29.0°C in unshorn sheep. A similar increase occurred in partially shorn sheep but was of a lesser magnitude.

It was not clear from independent naso-buccal circulation studies whether the initiation of polypnoea was due to thermal stimulation of the naso-buccal region or the anterior tracheal region. In order to localize the thermoreceptor site, experiments were conducted on unshorn and partially shorn sheep, where only the naso-buccal region was heated to about 40°C while the chamber

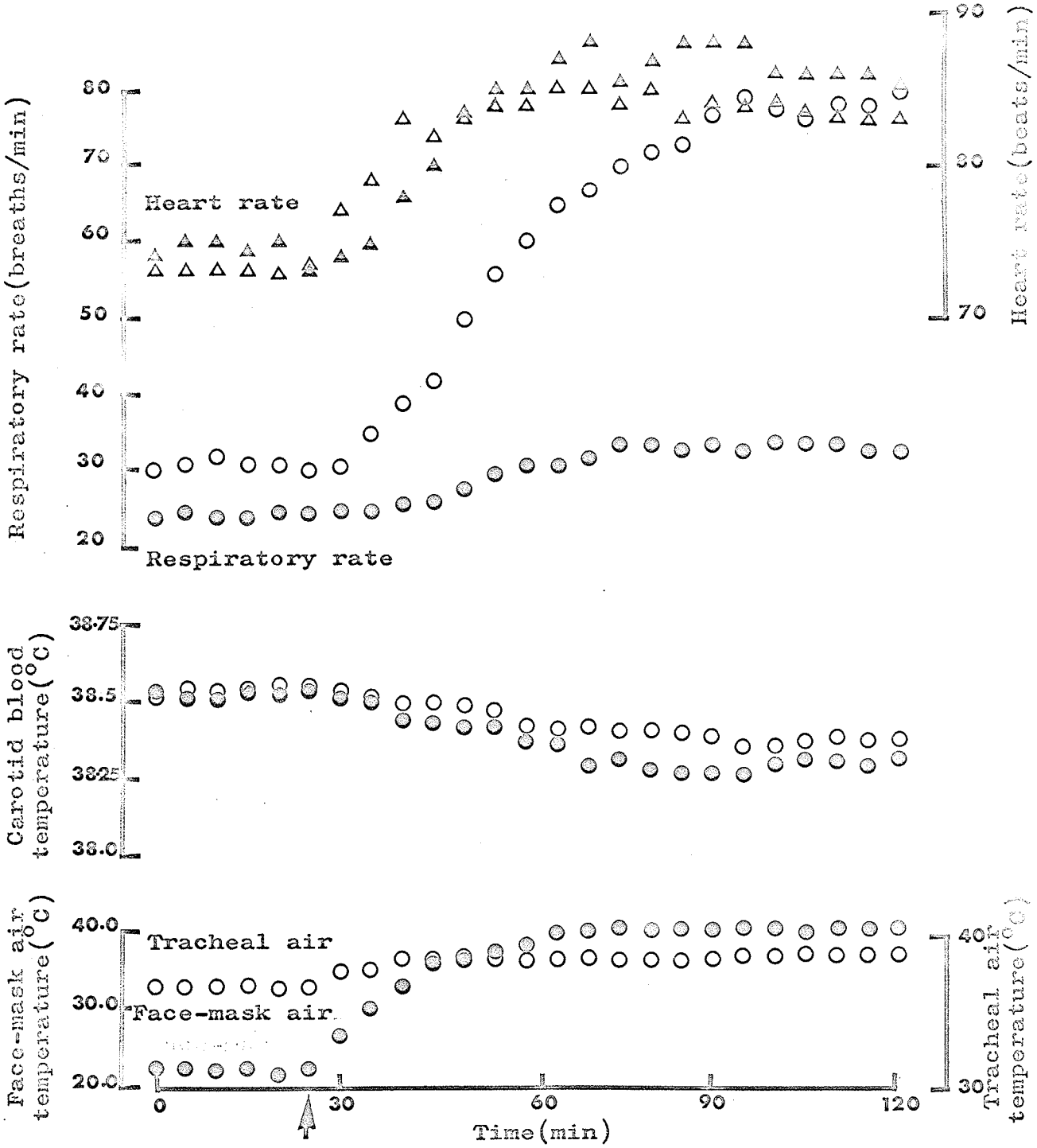

Fig.17. A comparison of the effect of raising the temperature of the face-mask air to 40°C at ambient temperature of 20°C, in unshorn(open symbols) and partially shorn(black symbols) sheep, upon the cardio-respiratory activities and carotid blood temperature. Each plot is the mean of 3 experiments on each of 4 sheep. The arrow marks the point where the face-mask air temperature was raised.

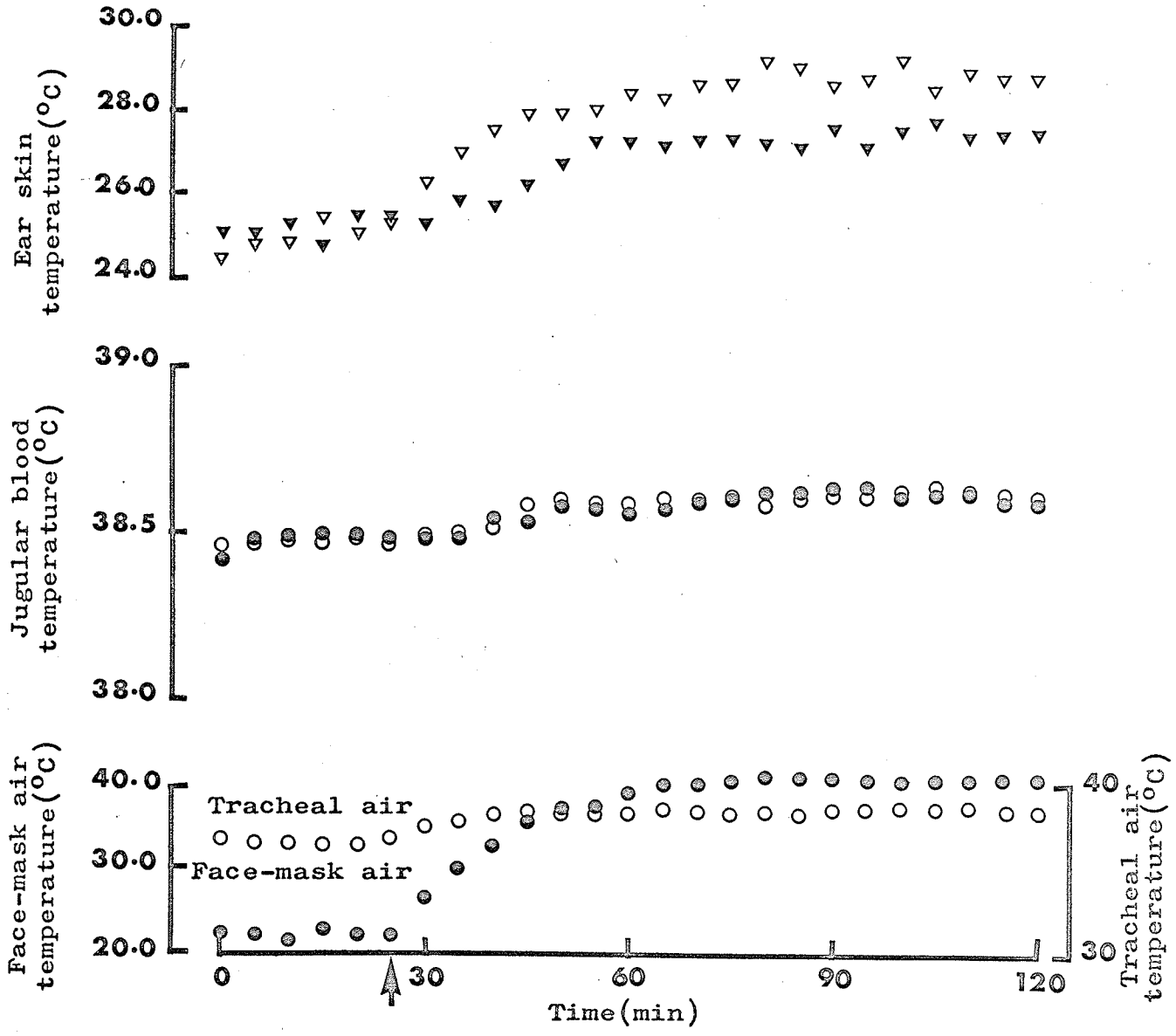

Fig.18. A comparison of the effect of raising the temperature of the face-mask air to 40°C at ambient temperature of 20°C, in unshorn(open symbols) and partially shorn(black symbols) sheep, upon the ear skin and jugular blood temperatures. Each plot is the mean of 3 experiments on each of 4 sheep. The arrow marks the point where the face-mask air temperature was raised.

temperature remained at 20°C. The changes in respiratory rate and carotid blood temperature following infra-red heating of the naso-buccal region are presented in figure 19. The muzzle temperature varied from 32.0°C to 34.5°C prior to infra-red heating and when this area was heated to 40°C, which was accomplished in 5 minutes, there was a gradual rise in respiratory rates which reached a maximum of 60/minute in unshorn sheep. Partially shorn sheep gave only small respiratory responses which were of no physiological significance. In both unshorn and partially shorn sheep, there was a moderate rise in heart rates. The carotid blood temperature of unshorn and partially shorn sheep remained almost steady throughout the experimental period.

Effect of lowering the naso-buccal air temperature

The naso-buccal air temperature was lowered to about 10°C after thermal polypnoea had been established at an ambient temperature of 40°C and 55% relative humidity. The ambient temperature was maintained at 40°C during the naso-buccal cooling. At ambient temperatures of 40°C, the respiratory rates varied between 103 and 111/minute in both unshorn and partially shorn sheep (figure 20). Following naso-buccal cooling, there was a moderate decline of 30-40 respirations/minute. The depressed respiratory rates were maintained for a period of 60-70 minutes and showed a slight rise during the later stages of the

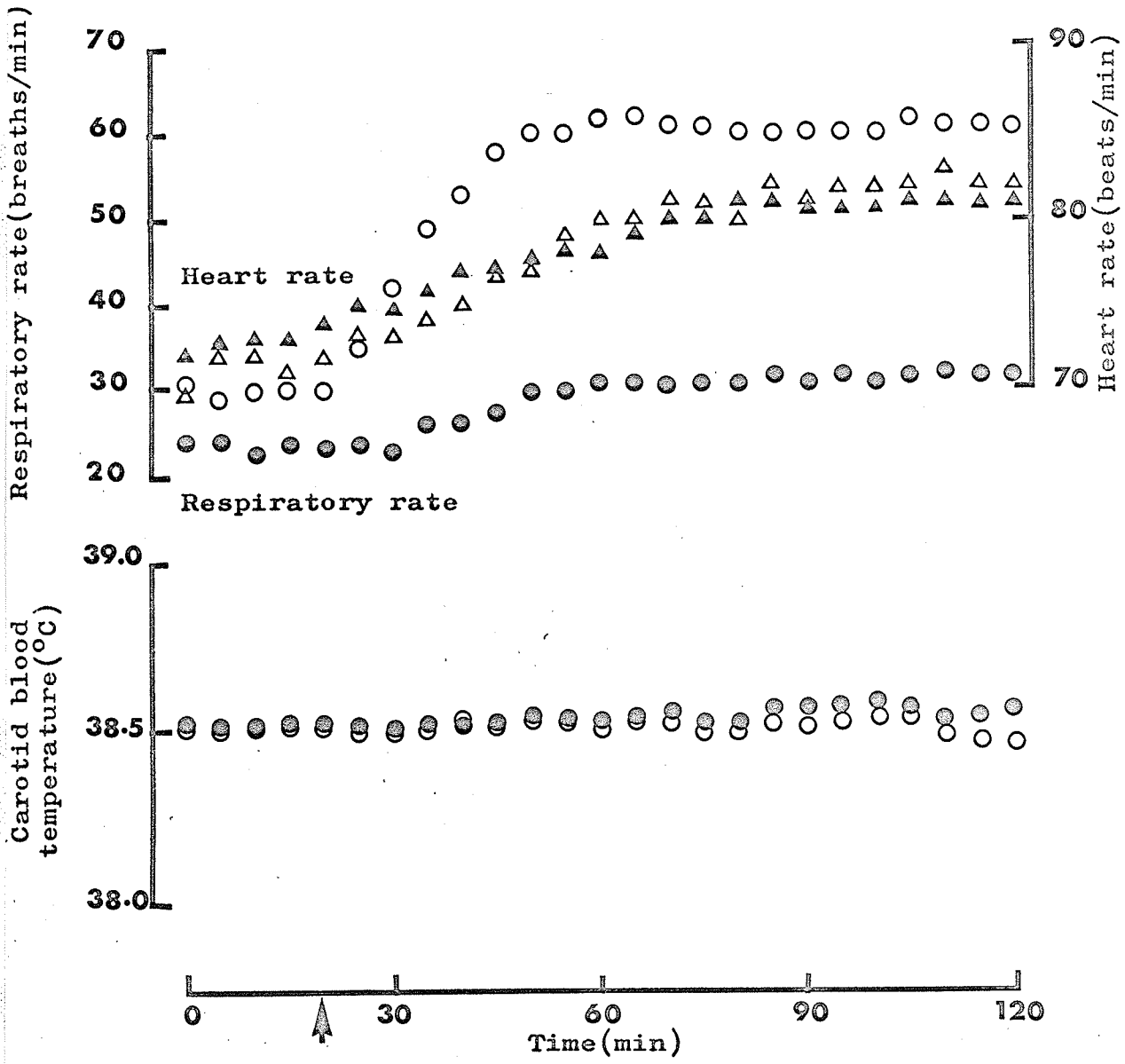

Fig.19. The effect of infra-red irradiation of the naso-buccal region at ambient temperature of 20°C in unshorn(open symbols) and partially shorn(black symbols) sheep, upon cardio-respiratory activity and carotid blood temperature. Each plot is the mean of 2 experiments on each of 4 sheep. The arrow marks the point where heat treatment was applied.

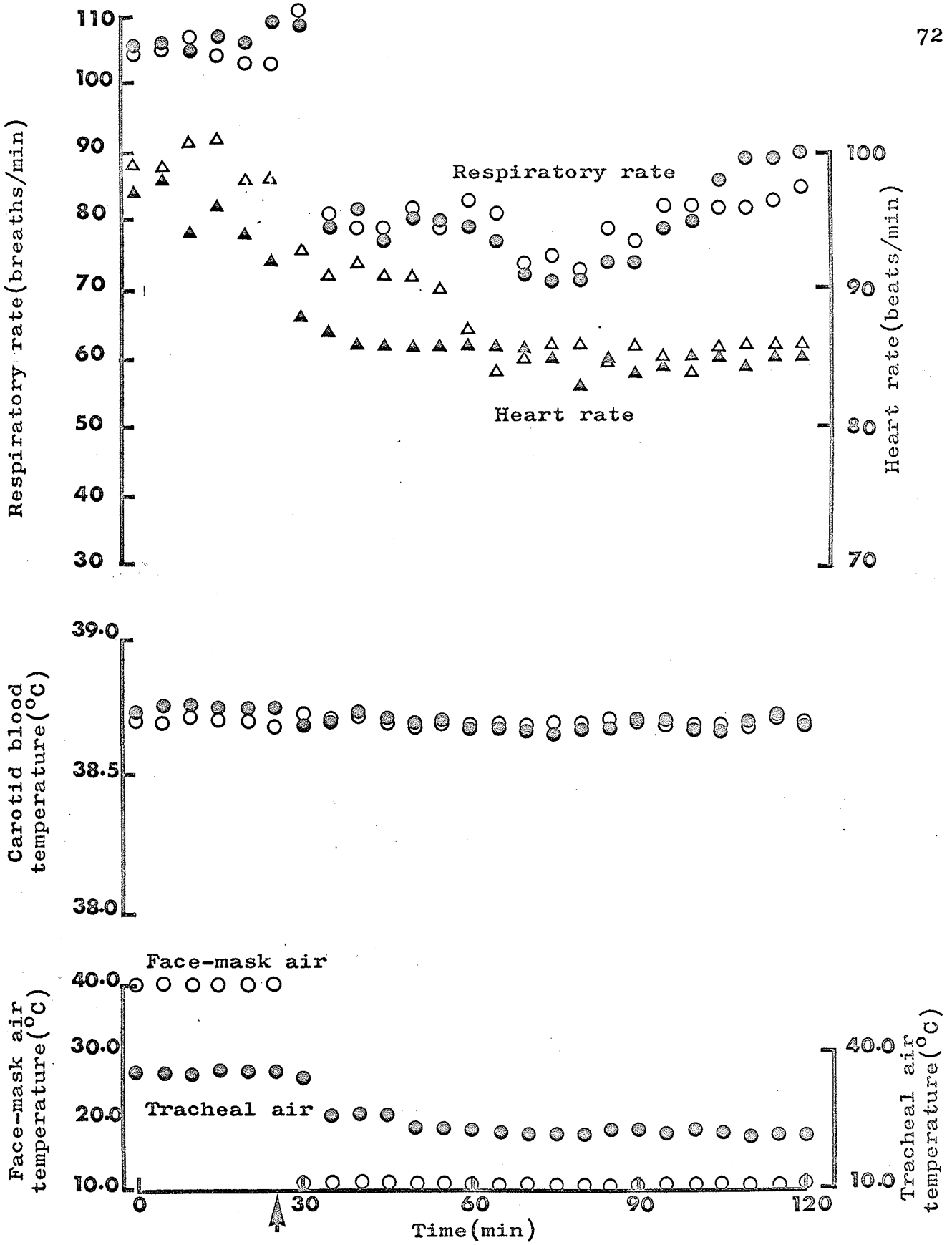

Fig.20. A comparison of the effect of lowering the temperature of air in the face-mask to 10°C at ambient temperature of 20°C, in unshorn(open symbols) and partially shorn(black symbols) sheep, upon the cardio-respiratory activity and carotid blood temperature. Each plot is the mean of 2 experiments on each of 4 sheep. The arrow marks the point where the face-mask air temperature was lowered.

experiment in both unshorn and partially shorn sheep, even though the mask temperature was maintained at 10°C. The heart rates of unshorn sheep were in the range of 98-101 beats/minute before lowering the naso-buccal air temperature and decreased to 84 beats/minute following naso-buccal cooling. The decline continued for about 35-40 minutes after which it remained fairly steady at 85 beats/minute. The heart rates of partially shorn sheep also declined following naso-buccal cooling but the decline was complete within 15-20 minutes after cooling.

While the cardio-respiratory activities responded to changes in naso-buccal air temperature, the carotid blood temperature showed no marked changes throughout the experiment in both unshorn and partially shorn sheep. The jugular blood temperature (figure 21) on the other hand, showed a marked decline in unshorn and partially shorn sheep following naso-buccal cooling. The decline in jugular blood temperature was expected, because the jugular vein drains the naso-buccal and tracheal regions, whose temperatures were markedly lowered by naso-buccal cooling. The temperature of the skin on the lumbar region did not show any marked changes following naso-buccal cooling in both unshorn and partially shorn sheep.

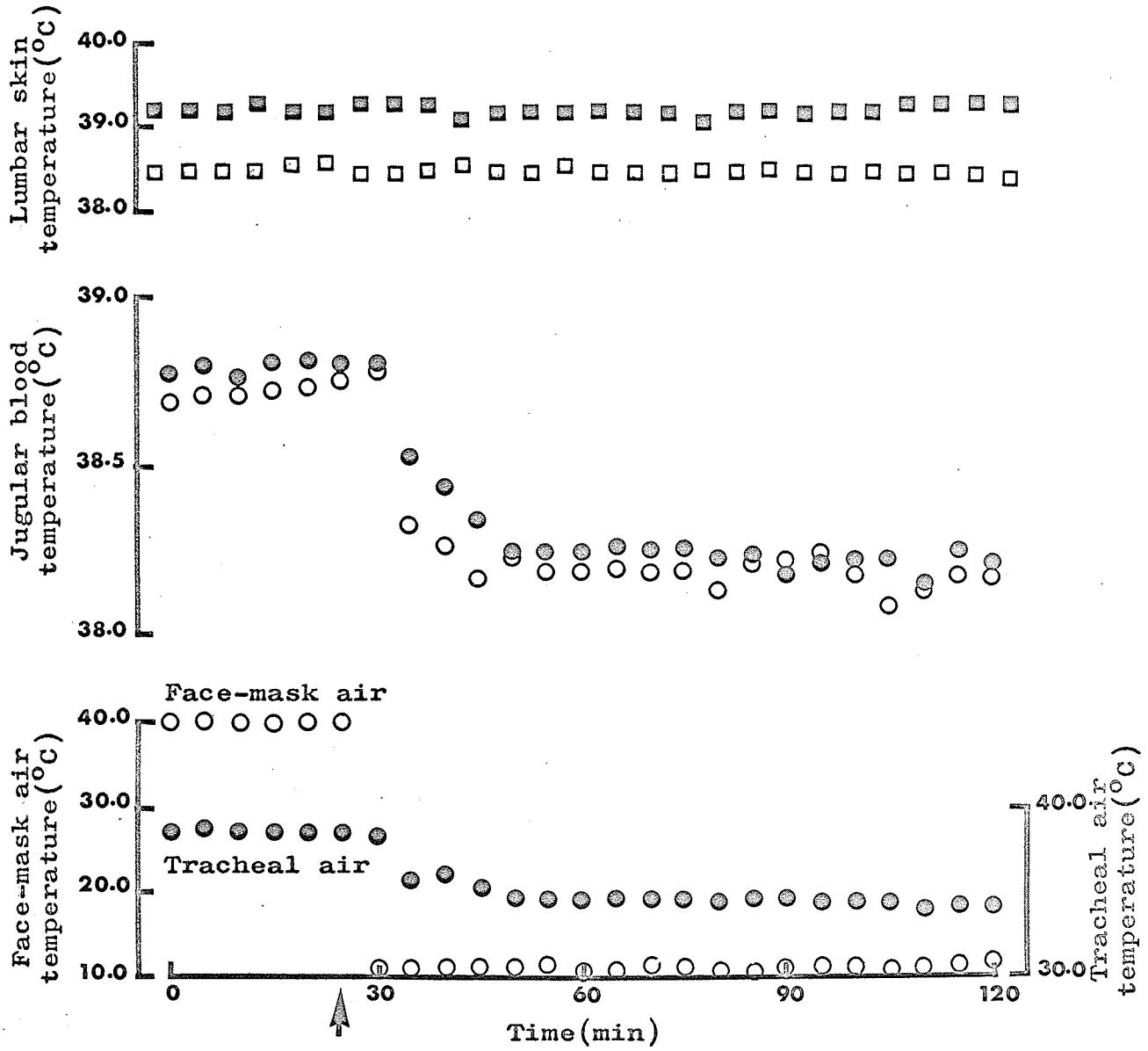

Fig.21. A comparison of the effect of lowering the temperature of air in the face-mask to 10°C at ambient temperature of 20°C, in unshorn(open symbols) and partially shorn(black symbols) sheep, upon the lumbar skin and jugular blood temperature. Each plot is the mean of 2 experiments on each of 4 sheep. The arrow marks the point where the face-mask air temperature was lowered.

DISCUSSION

It was observed that removal of an adequate area of fleece (3000 cm^2 or more) by close shearing on the thoraco-lumbar region resulted in a depression of respiratory rates. The respiratory responses to this shearing are in general agreement with those reported by Klemm (1962), Eyal (1963) and Bligh (1963b). Bligh (1963b) observed that the trunk skin temperatures of a closely shorn sheep were about 8-10°C lower than that of unshorn sheep at an ambient temperature of 20°C and this reduction in trunk skin temperature was not accompanied by any change in the temperature of the blood supplying the brain. Further, the respiratory rate reverted to that of unshorn sheep when the shorn area was covered with an insulating coat. He therefore suggested that the respiratory depression was brought about by stimulation of cutaneous cold receptors in the trunk region which were stimulated when the skin temperature was lowered. In the present study, the lumbar skin temperature dropped from 37°C to 31°C when an area of about 2000 cm^2 on the thoraco-lumbar region was closely shorn but this was not accompanied by respiratory depression. The depression occurred only when an area of 3000 cm^2 or more was closely shorn, at an ambient temperature of 20°C. However, when a shorn area of only 900 cm^2 on the thoraco-lumbar surface was cooled to about 10°C in

a polypnoeic animal, a depression in respiratory response occurred. Thus the respiratory depression appears to depend on two factors: (a) the extent of area sheared, and (b) the intensity of cold receptor stimulation. It is quite possible, therefore, that although cold receptors are stimulated when an area of 2000 cm^2 is closely shorn, additional cold receptor recruitment is necessary for the cold receptor drive to be effective in inducing respiratory depression. This additional cold receptor recruitment is achieved only when an additional 1000 cm^2 is closely shorn.

RESPONSES TO LOCALIZED HEATING

Heating the perineal region caused a rapid increase in respiratory frequency of unshorn sheep, and this rise was accompanied by a 0.30°C fall in carotid blood temperature. Waites (1962) observed a similar rapid rise in respiratory frequency when the scrotal skin of rams was heated to over 36°C despite a 2°C fall in deep body temperature. Linzell and Bligh (1961) also obtained a rise in respiratory frequency when hot air (45°C) was passed over the mammary glands of goats, but this rise in respiratory frequency was accompanied by a rise in deep body temperature. Subjecting flank skin to similar treatment did not result in the initiation of polypnoea. As regards the initiation of polypnoea, these results are in agreement with those of Waites (1962) and Linzell and Bligh (1961)

suggesting that the inguinal skin harbors warm receptors and that the stimulation of these receptors induces reflex polypnoea.

No marked rise in respiratory rate occurred in partially shorn sheep, following perineal heating, but the fall in the carotid blood temperature was not as pronounced as in unshorn sheep. The greater decline in the carotid blood temperature of unshorn sheep is due to a combination of respiratory evaporative cooling and peripheral vasodilatation, whereas in partially shorn sheep the decline is of lesser magnitude since it is due to peripheral vasodilatation alone. Apparently the only difference in responses, between unshorn and partially shorn sheep following perineal heating, was the absence of respiratory rise in the latter. The inability of a partially shorn sheep to pant following perineal heating could be due to a persistent cold receptor stimulation originating from the sheared area on the back region. Fusco et al. (1961) made continuous measurements of heat production in the dog after localized heating of the hypothalamus at environmental temperatures of 14°C, 26°C and 29°C. At all environmental temperatures hypothalamic heating caused 1.0°C to 1.3°C fall in deep body temperature. Heat balance during hypothalamic heating was achieved through vigorous panting at 29°C and by a 35% reduction in heat production at 14°C. Benzinger et al. (1963) described experiments in which lowering of

the skin temperature of humans, from 22°C to 18°C, resulted in an immediate increase in oxygen consumption indicating increased metabolic activity. It may be presumed from the present study of partially shorn sheep, that when the cold receptors of the lumbar skin region were stimulated the animals increased their heat production in an attempt to achieve thermal balance. This view is substantiated by the observation that partially shorn animals exposed to 15°C and 10°C ambient temperature shivered markedly, but when the perineal skin was heated the shivering ceased, resulting in decreased heat production. The decreased heat production coupled with increased peripheral vasodilatation resulted in a fall of carotid blood temperature.

The results also demonstrate that the changes in the pattern of respiration of unshorn sheep, after perineal heating, also depend on the ambient temperature to which they are subjected. For instance, when the ambient temperature was lowered from 20°C to 10°C, the pattern of respiratory frequency shows two distinct features: (1) a depression in respiratory frequency before perineal heat treatment and (2) a delay in the onset of polypnoea following perineal heat treatment.

Ingram and Whittow (1962) have shown that the magnitude of respiratory response, when the hypothalamus was heated, depended on both the temperature to which the hypothalamus was heated and the environmental temperature

to which the animal was subjected. In their experiments, small respiratory responses were observed until the hypothalamic temperature reached 41.5°C at an environmental temperature of 0°C. A greater respiratory response occurred at a hypothalamic temperature of 40.5°C and environmental temperature of 15°C. However, at an environmental temperature of 30°C, the response was maximal although the hypothalamus was heated to only 39.5°C. The perineal skin, in the present study, was heated to 40°C at all ambient temperatures (20°C, 15°C and 10°C) and invariably in all experiments there was a progressive decline in the carotid blood temperature.

Bligh (1962) observed that cold saline infusion into the thoracic or abdominal vena cava caused a substantial depression of respiratory rates and he suggested the possible existence of cold sensitive receptors between the point of infusion in the vena cava and the left side of the heart. The respiratory depression in the present investigation was not due to stimulation of these receptors, since jugular blood temperature before perineal heat treatment was about 0.1°C higher at an ambient temperature of 10°C than at an ambient temperature of 20°C.

Blaxter et al. (1959) found the critical temperature of sheep with 12 cm fleece to be 0.3°C. The unshorn sheep in the present experiments had a fleece of 10 cm long and thus the critical temperature of these sheep

would be expected to be in the vicinity of 0°C. Further, the fleeced sheep did not shiver when exposed to 15°C and 10°C ambient temperature. It is unlikely, therefore, that the respiratory depression was due to a change in the critical temperature of the animal, since the fleece length at ambient temperatures of 20°C and 10°C remained virtually the same.

The decrease in the magnitude of respiratory frequency of unshorn sheep at lower ambient temperatures before and after perineal heating was not dependent upon the temperature of the blood supply to the brain. It is suggested, therefore, that the respiratory depression could be due to stimulation of cold receptors in the naso-buccal or anterior tracheal region. Hensel and Zotterman (1951b) studied the frequency of electrical discharges from isolated thermoreceptive fibres in the tongue of the cat, in response to cold stimulation. In their experiments the receptor temperature was lowered in 3 successive steps of 2°C each. At each step the frequency of electrical discharges responded with a transitory rise and then settled at a new elevated level. It is quite possible that, in the present study, there was a rise in the frequency of electrical discharges from the naso-buccal cold receptors when the ambient temperature was lowered from 20°C to 10°C, thus resulting in depressed respiratory frequency.

If the warm receptors in the perineal region are

stimulated while the cold receptor drive is persisting, a situation may be attained wherein the thermoregulatory centres may receive conflicting messages from both cold and warm receptors. An interference of this nature could cause a delay in the onset of polypnoea at lower ambient temperatures.

A progressive increase in the skin temperature of the ear after perineal heating must have been caused by local periodic increase in blood flow because similar increases were not observed in lumbar skin temperature of both shorn and unshorn sheep. The reflex increase in blood flow to the ear is accompanied by a similar increase in heart rate. These circulatory adjustments following perineal heating were observed both in unshorn and partially shorn sheep at environmental temperatures of 15°C and 10°C. The rise in ear skin temperature and heart rates were also observed when flank skin was heated but this was of a lesser magnitude. Kerslake and Cooper (1950) observed reflex vasodilatation in the fingers of the hand in response to heating skin areas remote from the hand itself. The same authors (Cooper and Kerslake, 1955) found that the reflex increase in blood flow is preceded by a reflex increase in heart rate. They concluded that this reflex vasodilatation resulting from indirect heating was due to the excitation of cutaneous thermal receptors. Thus the rise in the ear skin temperature of both unshorn and

partially shorn sheep in the present study could be due to stimulation of the cutaneous warm receptors.

RESPONSES TO RAPID CHANGES IN AMBIENT TEMPERATURE

The rapid rise in the respiratory frequency of unshorn sheep which occurred in response to a rise in ambient temperature, was not related to changes in the temperature of the blood in the carotid artery. But often the temperature of the blood in the carotid artery and jugular vein increased after polypnoea had been initiated. The present observations confirm the suggestion of Bligh (1957c) and Waites (1962) and Findlay and Ingram (1961) that the stimulus for the initiation of polypnoea could be entirely of peripheral origin.

The respiratory responses of partially shorn sheep to rising ambient temperature are in general agreement with those reported by Bligh (1963b). A persistent delay of 20-30 minutes in the onset of polypnoea, following a rise in ambient temperature from 20°C to 40°C, occurred in all partially shorn sheep. The respiratory rates then rose rapidly after the lumbar skin temperature had attained 39°C and approached that of unshorn sheep. Bligh (1963b) observed that the respiratory depression in shorn sheep at 20°C ambient temperature was due to the exposure of cold receptors on the sheared surface. Besides causing respiratory depression, the cold receptor stimulus at the same time causes a temporary build up of 'blocking factor'

in or between the thermoregulatory and respiratory centres. The 'blocking factor' prevents the warm receptor drive from acting upon the respiratory centre and causes a temporary delay in the onset of polypnoea. This seems to have been the case in the present study. Since the skin temperature of partially shorn sheep closely paralleled the rise in room temperature which counteracts or causes the withdrawal of cold receptor drive, an immediate cessation of shivering, and an abrupt rise in respiratory frequency results.

It has already been demonstrated that polypnoea can occur without any change in hypothalamic temperature (Findlay and Ingram, 1961) and the temperature of the blood supply to the brain (Bligh, 1957c). Findlay and Ingram (1961) also observed that if the body temperature was raised to above 40°C by infra-red irradiation, the higher respiratory rates were maintained even after irradiation had ceased. In the present experiments there was a 0.4°C rise in carotid blood temperature during terminal stages of the experiment in all sheep. It would thus appear that once the cold receptor drive is withdrawn or overcome, the higher respiratory frequency is maintained by the hypothalamic thermoregulatory centres.

The rapid fall in respiratory rates of partially shorn sheep, when the ambient temperature is lowered from 40°C to 10°C, is attributed to a similar fall in

the lumbar skin temperature. This in turn is probably transmitted to the blood. The delayed respiratory decline in unshorn sheep could be due to a slower decline in the temperature of the blood supply to the brain. The present study also indicates that continuous stimulation of the cold receptors in a polypnoeic sheep did not result in continuous depression of respiratory frequency. This phenomenon may be due to the fact that the temperature of the blood supply to the brain remained comparatively at a higher level, at 40°C ambient temperature, and did not show marked changes following cold receptor stimulation. It is, therefore, suggested that the rise in respiratory frequency in response to a rise in ambient temperature has two components, namely, peripheral and central. The peripheral control depends on the stimulation of peripheral warm receptors which serves as an immediate defensive mechanism whereas hypothalamic control takes over at a later stage when the environmental conditions continue to be adverse.

THERMAL SENSITIVITY OF THE UPPER RESPIRATORY TRACT

The respiratory behaviour of unshorn sheep when the naso-buccal air was heated suggests the existence of warm receptors in this region. Sihler (1880) conducted experiments to study the role of respiratory passages in the initiation of panting in dogs. He found that breathing warm and moist air did not result in panting.

Beakley and Findlay (1955) suggested that, in bovines, there may be thermal receptors in the respiratory tract. Nisbet (1955) confirmed this view by demonstrating the existence of many sensory nerve endings in the bovine muzzle. Bligh (1959) observed that heating the naso-buccal passage of the bovine only provides a feeble stimulus to panting. In the present experiments, heating the naso-buccal region results in an increase of respiratory rates, but the magnitude of response is about 25% less than when the naso-buccal air is heated. These results support the view of Richet (1898) that initiation of polypnoea results from stimulation of cutaneous nerve endings of the naso-buccal region.

The humidity of the inspired air during independent naso-buccal air circulation studies could not be controlled and remained high during these experiments. Consequently, the rise in respiratory frequency was markedly greater. These observations confirm Bligh's (1963a) findings that the respiratory response of sheep to a high ambient temperature is greater when the humidity is high. It is quite likely that there is either an enhancement of the impulse frequency of the warm receptors when the humidity of the inspired air is raised, or a modification in the response of pressure or touch receptors in the mucous membrane of the naso-buccal and tracheal region when the vapour pressure of the inspired

air is altered. It is reasonable to assume that, although some degree of receptor specificity exists, the possibility cannot be ignored that some fibres transmit information relating to more than one type of cutaneous sensation. Hunt and McIntyre (1960) observed that the responses of touch units to mechanical stimulus can be influenced by adequate heating of the skin.

In the present experiments, the temperature of the blood supply to the brain declined in response to naso-buccal heating and the changes in ear and skin temperature were similar to those of perineal heating. Furthermore, partially shorn sheep showed no marked respiratory responses and yet their carotid blood and ear skin temperatures declined just as in unshorn sheep. It is, therefore, suggested that initiation of polypnoea is of reflex nature and is due to the stimulation of warm receptors located in the naso-buccal region. The specific nature of these receptors remains unknown. However, judging from their ability to increase respiratory rate, heart rate, and the vasomotor reflexes elicited in the ear, it is presumed that they are similar to those located in the perineal region. The specificity of cutaneous sensory mechanisms has been a problem of great controversy. Hensel et al. (1960) cautioned that the concept of 'specific' sensory fibres or receptors has two quite different aspects, namely, the specific sensation obtained by stimulation

of the receptor and the response of a receptor to a specific stimulus. Since certain cutaneous and other receptors respond to thermal as well as other stimuli (Hensel and Zotterman, 1951a; Lele and Weddell, 1959) the classification of sensory nerve endings as thermoreceptors seems to be arbitrary.

Naso-buccal cooling, in the present study, resulted in a marked depression of respiratory frequency and the depression was transient even though the cold stimulus was maintained. The transient depression which occurred in all animals suggests the existence of receptors sensitive to cold in the naso-buccal passage. These results are in agreement with those of Kleiber and Regan (1935) who also observed that breating cold air at a temperature of 4°C to 15°C at ambient temperatures of 32°C to 37°C caused a marked decrease in the respiratory rate of cows.

Hensel and Zotterman (1951a) recorded the action potentials in the lingual nerve of cats and dogs during cooling of the tongue surface. Their experiments suggest that the frequency of cold impulses remain fairly unchanged as long as the temperature of the tongue surface is maintained at a constant lower temperature. In the present investigation, the tracheal air temperature remained fairly constant at 19°C. After cooling had commenced, the cold receptor drive must have persisted. The inability of the cold receptor drive to maintain the depressed

respiratory activity, at an ambient temperature of 40°C, during naso-buccal cooling, and the absense of any decline in the temperature of the blood supply to the brain, which was about 0.35°C higher than at an ambient temperature of 20°C, suggests that brain temperature assumes a much greater significance at higher body temperatures. Holmes, Newman and Wolstencroft (1958) demonstrated that, besides the hypothalamus, heating of structures in the hind brain also causes a rise in respiratory rates. Thus a rise in the temperature of the blood to the brain could influence respiration rates by its effect on more than one centre.

GENERAL DISCUSSION

The present study has shown that localized heating (perineal or naso-buccal) and cooling (lumbar skin or naso-buccal) of fully fleeced sheep can initiate thermoregulatory responses. The decline in the carotid blood temperature following perineal and naso-buccal heating, the depression in respiratory rate after polypnoea had been established during thoraco-lumbar and naso-buccal cooling, and the absence of any rise in carotid blood temperature before the onset of polypnoea, when the ambient temperature was raised from 20°C to 40°C, clearly emphasises the contribution of peripheral thermal drives in maintaining thermal balance.

The respiratory and tissue temperature responses of unshorn and partially shorn sheep, when the ambient temperature is raised from 20°C to 40°C, are similar to those reported by Bligh (1963b). He explained that the depression of respiratory frequency, in shorn sheep, at 20°C ambient temperature, was due to stimulation of the cold receptors on the trunk skin region and at the same time there could be a build up of 'blocking factor' in or between the thermoregulatory and respiratory centres. The 'blocking factor' somehow prevents the warm receptor drive from acting upon the respiratory centre. This results in a delayed onset of polypnoea when the ambient tempera-

ture is raised to 40°C. When the cold receptor drive is withdrawn by rising ambient temperatures, the intensity of the 'blocking factor' tends to decline producing an abrupt onset of polypnoea. The reduction in the intensity of this 'blocking factor' is governed by (a) the duration of cold exposure (b) the degree of intensity attained by the 'blocking factor' during cold exposure and (c) may depend also on the intensity of the warm receptor stimulus. The intensity of the 'blocking factor' determines only the duration of the respiratory delay and not the degree of depression. Thus, when the intensity of the 'blocking factor' has declined to zero, the normal warm receptor drive reaches the respiratory centre causing an abrupt onset of polypnoea.

Based on the responses of peripheral warm and cold receptor stimulation, a model similar to the one proposed by Bligh (1963b), with slight modifications, but which is consistent with his interpretation, is presented in figure 22.

Stimulation of the warm receptors by perineal or naso-buccal heating results in the onset of polypnoea, increased heart rate, cessation of shivering and peripheral vasodilatation (as indicated by marked rise in ear skin temperatures at 15°C and 10°C ambient temperature). When the ambient temperature was lowered from 20°C to 10°C, there was a delay in the onset of polypnoea following perineal heating and a decrease in the magnitude of respiratory

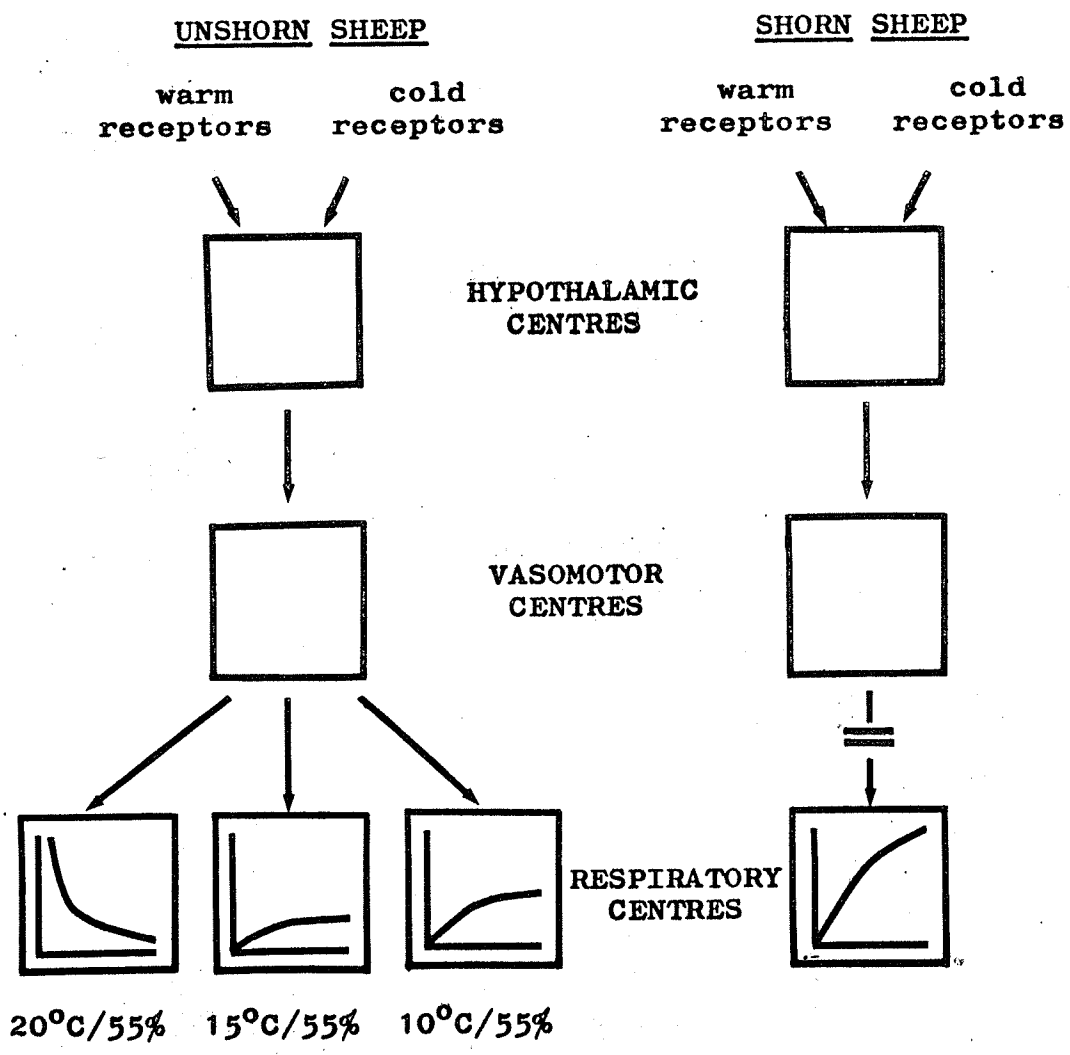

Fig.22. A diagrammatic representation of the proposed model. Abscissa of insert graph, time; ordinate, intensity of blocking factor.

frequency during polypnoea. It is suggested that these responses at lower ambient temperature were brought about by stimulation of cold receptors in the naso-buccal cavity. The duration of cold exposure before perineal heat treatment and the intensity of warm receptor stimulus were identical at all ambient temperatures (20°C, 15°C and 10°C) and yet the delay in the onset of polypnoea increased with decreasing ambient temperature. Thus, only the intensity of the cold receptor drive from the naso-buccal region must have been varied. The present observations show that this is actually the case since there is a progressive depression in the respiratory frequency of unshorn sheep, before perineal heat treatment, at lower ambient temperatures.

On the other hand, close shearing of 3000 cm^2 or more on the thoraco-lumbar region caused a marked depression of respiratory frequency at all ambient temperatures. Subsequent warm receptor stimulation did not show any marked respiratory responses but resulted in increased heart rate and peripheral vasodilatation. Thus, the delay in the onset of polypnoea of unshorn sheep and a subsequent decrease in the magnitude of respiratory frequency following perineal heat treatment at lower ambient temperatures (15°C and 10°C) could be due to a build up of a 'blocking factor', similar to the one described by Bligh (1963b). Since the only variable was the intensity of the cold receptor drive, which appears to increase with decrease

in ambient temperature, it is suggested that the intensity of the cold receptor drive determines not only the level attained by the 'blocking factor' but also its release upon warm receptor stimulation. In partially shorn sheep, there is a greater recruitment of cold receptor drive orginating both from the trunk surface and the naso-buccal region. This increases the intensity of the 'blocking factor' and it does not decline, even after warm receptor stimulation. The intensity of the 'blocking factor' is further increased when the ambient temperature is lowered to 10°C. The decrease in the respiratory rate of partially shorn sheep with decrease in ambient temperature following warm receptor stimulation supports this view.

It is interesting to note that the perineal heating of unshorn and partially shorn sheep results in reflex vasodilatation as indicated by marked rise in ear skin temperature and increased heart rate. It is, therefore, suggested that the block interferes only with the respiration centre in partially shorn and unshorn sheep. Heat loss from the skin surface is not interfered with.

SUMMARY

1. Four Suffolk ewes were used as experimental animals to study the distribution of peripheral thermal receptors on the skin and in the respiratory passages.

2. The effect of close shearing of the fleece upon the respiratory frequency of ewes at ambient temperature of 20°C and 55% relative humidity is described.

3. Removal of an adequate area of fleece(3000 cm^2 or more) on the thoraco-lumbar region resulted in a depression of respiratory response. The respiratory depression in partially shorn sheep was attributed to the exposure of cold receptors located beneath the fleece.

4. Measurements have been made of the cardio-respiratory activities, ear and lumbar skin temperatures, and temperature of the blood in the carotid artery and jugular vein of partially shorn and unshorn sheep during perineal heating at ambient temperatures of 20/15, 15/55 and 10/55 (dry bulb temperature, °C/ relative humidity, %).

5. Perineal heating resulted in a marked rise in respiratory and heart rates of unshorn sheep, whereas, in partially shorn sheep, the heart rates increased without being accompanied by a similar rise in respiratory rates.

6. With decrease in ambient temperature from 20°C to 10°C, there was a decrease in respiratory response of unshorn sheep during perineal heat treatment, suggesting

that the magnitude of respiratory response during perineal heat treatment depends partly on the environmental temperature to which the sheep is exposed.

7. The carotid blood temperature declined following perineal heat treatment at all ambient temperatures studied both in unshorn and partially shorn sheep, but the magnitude of decline was greater in unshorn than in partially shorn sheep.

8. The rise in respiratory rates following perineal heating of unshorn sheep at all ambient temperatures, inspite of a decline in carotid blood temperature and the absence of similar responses during flank heating, is attributed to the stimulation of warm receptors in the perineal region.

9. Experiments involving short term exposures to rising ambient temperature resulted in an immediate initiation of polypnoea in unshorn sheep. In partially shorn sheep, there was a 20-30 minute delay in the onset of polypnoea. The delay in the onset of polypnoea in partially shorn sheep during rising ambient temperatures, the small respiratory responses obtained following perineal heat treatment of partially shorn sheep, and a variable depression of respiratory responses when the lumbar skin (900 cm^2) temperature was reduced to 10°C at 40°C ambient temperature, is attributed to the exposure of cold receptors distributed on the skin beneath

the fleece.

10. Experiments with a face mask in which the temperature of the air in the face mask was raised to 40°C while the ambient temperature in the chamber was maintained at 20°C, resulted in a marked increase in respiratory frequency and a slight decline in carotid blood temperature of unshorn sheep. Partially shorn sheep showed only small respiratory responses.

11. Localized infra-red irradiation of the naso-buccal area of unshorn sheep also resulted in an increased respiratory rate.

12. It is suggested that the initiation of polypnoea during infra-red irradiation of the naso-buccal region and following rise in the temperature of the air in the face mask is due to stimulation of warm receptors in the upper respiratory tract.

13. Cooling the naso-buccal air in the face mask to 10°C after thermal polypnoea had been established at an ambient temperature of 40°C resulted in a moderate decline of 30-40 respirations/minute. This decline was attributed to the stimulation of cold receptors located in the upper respiratory tract.

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