THE UNIVERSITY OF ALBERTA

TEMPERATURE REGULATION IN THE EXTREMITIES OF RUMINANTS

by

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies for acceptance, a thesis entitled "Temperature Regulation in the Extremities of Ruminants" submitted by Allen Arnold Meyer, B.Sc. (Ag.), in partial fulfilment of the requirements for the degree of Master of Science.

ABSTRACT

Cattle and sheep were used in experiments conducted to study the nature and control of heat loss from the extremities. Skin temperature measurements of the tail of oxen were incorporated into thermal equations to determine heat loss and blood flow during cold exposure. Measurements were made of the surface temperatures of the legs and ears of sheep during cold exposure trials involving the infusion of noradrenaline or pitressin. Other measurements included blood pressure, heart rate and partial pressures of oxygen in arterial and venous blood. Sympathectomy of the left ear was carried out as a further technique in measuring cold induced vasodilation (CIVD) during cold exposure.

Minimum skin temperature in the tail of cattle was reached at an air temperature of about -7°C. It was assumed though not demonstrated that at air temperatures below about -7°C local blood flow increased as a consequence of local vasodilation. The extent of the increase in blood flow resulting from extreme local vasodilation occurring during reactive hyperaemia was in excess of that which, it was predicted would be required to prevent local tissue freezing at an air temperature of -40°C.

Sheep trials demonstrated the direct stimulatory action of noradrenaline on the heart. Noradrenaline infusion depressed the hunting response. In the sympathectomized ear however noradrenaline infusion caused sufficient vasoconstriction to cool the ear and induce 'hunting'. It was concluded that CIVD is due to a change in the balance between vasoconstriction caused by the sympathetic transmitter substance noradrenaline, and vasodilation caused by accumulation of vasodilatory substances.

ACKNOWLEDGEMENTS

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INTRODUCTION

The livestock producing regions of Western Canada experience an extreme temperature variation between seasons. Animals kept outside or in unheated buildings required elaborate control mechanisms to regulate and maintain homeothermy. Temperature regulation in the extremities involves mechanisms unlike those occurring in the rest of the body since their surface area to mass ratio is relatively large and they possess little capacity to produce heat *in situ*. In a cold environment compensations are made by decreasing blood flow to the extremities to a level which permits the tissues to be maintained at a temperature considerably below deep body temperature but above freezing point. Although heat loss from extremities is reduced by this means, it is not eliminated. Extremity temperatures approach a minimum close to the freezing point. Thereafter heat loss will increase linearly with decreasing ambient temperatures. To maintain this minimum temperature against a decreasing ambient temperature, blood flow must increase. It does so by a process called 'cold induced vasodilation' (CIVD). Temperature measurements show that CIVD can occur slowly or quite suddenly, in either case increasing blood flow in an attempt to maintain extremities above freezing. It would be desirable to know what the mechanisms are which control CIVD. It would also be desirable to know the extent to which heat loss from extremities contributes to total sensible heat loss.

LITERATURE REVIEW

Newton's law states that the amount of sensible heat exchange between an object and its surroundings is proportional to the difference in temperature between that object and its surroundings (Hardy, 1949). The greater the difference in temperature, the greater the sensible heat exchange. Thus thermoregulation is dependent in part on the ability of body areas to change temperatures.

Extremities, defined for purposes of this study, are the ears, facial areas (cheeks and nose), tail, and those parts of the limbs below the ventral limits of the trunk. It is a well established fact (Greenfield, 1963; Molyneux, 1965; Blaxter et al., 1959; Whittow, 1962) that the extremities play an important role in this heat exchange continually taking place between the body and the environment. Due to low thermal conductivity of body tissue (Greenfield, 1963), blood circulation is essential in conveying heat produced in the muscular areas and organs comprising the body core to the surface for elimination by conduction, convection, radiation, or evaporation.

Assuming, as does Greenfield (1963), a mean skin thickness of 1.2 mm, and a maximum flow of 90 cc/100 cc of tissue/min (Burton, 1939), maximal blood flow for the fingers would be 1080 $cc/m^2/min$. Blood flow through the skin has been measured in various other ways including calorimetry; comparison of one limb flow to another in which skin flow has been stopped by iontophoresis of adrenaline; indirect methods, i.e. change in oxygen content of blood leaving skin assuming constant metabolic rate in skin; measuring skin temperature for qualitative indication of blood flow; changes in thermal conductivity of skin; rate

of clearance of sodium-24 from an injection site (Greenfield, 1963).

Blood is the most important source of heat to extremities and blood flow can vary by 100-200 fold. Forster, Ferris and Day (1946) used a combination plethysmograph and calorimeter to measure hand blood flows in subjects exposed to varying temperatures. He reports blood flow to range from 0.15 cc/100 cc of hand tissue/min at an ambient temperature of 15.3°C to 31.7 cc/100 cc/min at an ambient temperature of 38°C. He mentions that values obtained during the cold exposures were lower than those usually reported, but points out that the whole body was exposed to the ambient temperature and not just the hand.

Indications from measurements of skin temperature would suggest two types of control over peripheral circulation on exposure to cold. One is the 'hunting' response first described by Lewis (1930), which appears as a sudden relaxation of constricted areas allowing for an increase in temperature. This is then followed by another vasoconstriction. This phenomenon can occur throughout experimental cold exposure.

The second is a continuous proportional control type of response where blood supply is regulated to keep the area at a steady temperature (Webster and Blaxter, 1966).

The areas reaching the lowest temperatures and exhibiting the hunting response described above have a greater density of specialized structures called arteriovenous (A/V) anastomoses than other areas of the body of rabbits (Grant, 1930), and sheep (Molyneux, 1965). Grant (1930) observed these structures to constrict in rabbit ears on adrenaline injection and dilate with acetylcholine. They also dilated

during the hunting response. The entire increase in blood flow during hunting in the rabbit was found by Edwards (1967) to come through the A/V anastomoses.

Hurley and Mescon (1956) described a cholinergic nerve supply around A/V anastomoses in man, and hypothesized a direct cholinergic control of their vasodilation. Assuming a muscarine-like effect, atropine should abolish any vasodilation. However Duff et al.(1953) recorded normal cold induced vasodilation (CIVD) in the fully atropinized finger and concluded that acetylcholine involvement in the reaction was unlikely.

Lewis (1930) found that sympathectomy did not alter the 'hunting' response. However after degeneration of sensory nerves he was not able to obtain a response in the denervated hand. From this he attributed the hunting response to a sensory axon nerve reflex mechanism, the stimulus for which being the release of a histamine-like substance from the skin upon injury by cold. The axon reflex will be discussed in more detail later.

Grant, Bland and Camp (1932) observed CIVD in the rabbits ear, similar to that described in the hand by Lewis (1930). This was not abolished by denervation and subsequent degeneration of sensory nerves. Greenfield, Shepherd and Whelan (1951), using subjects whose sensory nerve supply had been cut and had probably degenerated, came to the conclusion that the hunting response "... does not depend on the integrity of somatic sensory nerve fibers, although when they are intact the response is much larger than when they are not."

Experiments were carried out later by Greenfield, Shepherd

and Whelan (1952) to determine if anesthetic solutions of xylocaine or novocaine could, upon local injection, abolish CIVD. Although smaller than in unanesthetized hand, hunts occured, thus substantiating their earlier conclusion that a local axon reflex is not necessary for cold vasodilation.

Ingram and Whittow (1963) reported an increased blood pressure and heart rate preceding the CIVD in ears of the ox. The blood pressure increase was greater preceding the first increase in skin temperature than preceding the following increases. They suggested this due to less synchronous increases in temperature of the ears. They also suggested that the effect of the increased blood pressure was to cause an increased blood flow through the ear by forcibly dilating the arteriovenous anastamoses.

Lewis (1930) found CIVD was not synchronous in corresponding fingers of the left and right hands. Greenfield, Shepherd and Whelan (1951) found that if the corresponding fingers on the hands of an individual were immersed in ice water, the second going in five minutes after the first, that the onset of vasodilation was only dependent on the time of immersion. This asynchrony would throw doubt on the possibility of blood pressure controlling hunting.

Hertzman and Roth (1942) described an experiment in wnich the finger of one hand was cooled with the remaining fingers on that and the other hand acting as controls. He observed that the initial constriction in the cooled finger was accompanied by a smaller constriction in control fingers and concluded this constriction, in control fingers, due to vasoconstrictor reflexes. The dilation

following cooling could be limited to the cooled finger while the others maintained a high vasoconstrictor tone. Also during dilation vasoconstrictor response could be elicited. At times however " ... definite evidence of vasoconstrictor paralysis in the chilled finger was obtained." Hertzman and Roth concluded, "The reactive dilation, which follows in the chilled finger within three to eight minutes after the application of cold occurs independently of the vasomotor system."

Gaskell and Hoeppner (1967) infused noradrenaline into an individual with one hand kept warm (34°C) and the other cool (22°C), and found the warm hand more reactive to the vasoconstrictor effect of noradrenaline, as indicated by a larger increase in critical opening pressure in this hand.

Using isolated ulnar arteries of bullocks Keatinge (1958) demonstrated that when cooled below 10°C these arteries ceased to contract on exposure to adrenaline, histamine, or pitressin. On rewarming to 37°C however these arteries contracted. Keatinge proposed that, "The development of vasoconstriction, the hunting reaction in cold fingers, could well be explained by the partial recovery of the contractile power of the vessels following the vasodilation and the consequent return of warm blood to the skin."

Keatinge (1961) exposed one index finger to iontophoresis of noradrenaline and adrenaline and used the other as a control. Iontophoresis caused a complete cessation of blood flow as measured by heat-flow calorimetry. Both fingers were then immersed in ice water. The control finger decreased blood flow over the first five

minutes then dilated and proceded with characterisitic hunting. The finger exposed to iontophoresis continued to decrease blood flow a while longer, then dilated slowly. The CIVD following immersion was clearly suppressed by iontophoresis of noradrenaline. Keatinge's conclusion that this dilation was a direct consequence of a reduced effect of noradrenaline on cold tissue does not necessarily follow.

The peripheral conducting system characterisitic of autoregulation may play a part in CIVD (Hilton, 1962). The four more popular explanations of autoregulation are as follows: (1) active vasomotion due to change in transmural pressure (myogenic theory); (2) active vasomotion due to change in oxygen concentration; (3) active vasomotion due to change in concentration of vasodilating metabolites; and (4) passive vasomotion due to change of interstitial fluid pressures (Haddy and Scott, 1964).

Folkow (1949) has presented evidence in support of the myogenic theory that increased blood pressure causes an increase in tone of the small blood vessels and a reduced blood pressure causes a decrease in tone.

With regard to the chemical theories (2) and (3) Hilton (1962) proposed two questions: (i) What is the nature of the chemical stimulus derived from the cells of the active organ, and (ii) how is the vasodilation, initiated locally by this stimulus, conducted to the larger arteriole vessels which cannot be reached by diffusion alone?" Haddy and Scott (1968) acknowledged the evidence that autoregulation in the vascular bed exists and that metabolically linked chemical mediation is likely, although the exact chemical or chemicals involved

have as yet to be properly identified. If the autoregulatory mechanism is considered to be non-nervous, the possibility of a direct conduction system through the smooth muscle of the artery wall might be considered (Hilton, 1959).

Using the forelimb vascular system of the dog, Haddy and Scott (1964) could find no change in interstitial fluid pressures to either a change in flow rate or to a change in venous pressure. The limb during these procedures displayed autoregulation.

The polypeptide bradykinin and related plasma kinins exert a very strong vasodilator action. Chapman, Goodell and Wolff (1959) demonstrated an increase in plasma kinin during the red flare phase of the triple response initiated by injection of histamine into human skin. At the same time plasma kinin forming enzyme showed up in the perfusate. These results indicated a possible role for bradykinin or related plasma kinins in the vasodilation associated with the axon reflex (Lewis, 1960).

Chapman et al. (1959) listed three possible mechanisms which may cause kinin formation.

(1) Blood contains an enzyme (plasmin) which may enter interstitial spaces by some 'unknown' vasodilator mechanism which then secondarily produces plasma kinins.

(2) Neurogenic influences may release metabolites that convert plasminogen already present in extracellular fluid to the active proteolytic enzyme, plasmin, which then results in polypeptide formation.

(3) A neurogenic humoral agent, such as acetylcholine, may alter the membrane permeability of a variety of cells to release intracellular

proteolytic enzymes, which then act on protein in extracellular fluid to produce polypeptides of the bradykinin type, as well as other potent agents, such as histamine and serotonin.

Sympathectomies have been used in an attempt to decide upon the existence of an active vasodilator innervation to blood vessels in the extremities (Lewis, 1930; Grant et al., 1932, Grant, 1935) as well as a curative surgical procedure (Grimson and Durham, 1946). There generally occurs a recovery in "tone" of the denervated vessels wnicn may be sufficient to cause constriction in cool surroundings a few days after sympathectomy (Grant, 1935).

Possible reasons for the return toward the preoperative state are discussed by Grimson and Durham (1946) and listed as follows:

(1) Sympathetic nerve regeneration.

(2) Increased irritability of smooth muscle after sympathectomy.

(3) Recovery of intrinsic peripheral vascular tone.

Perkins et al. (1948) found incomplete agreement with respect to the responsible mechanism.

Antidromic vasodilation produced by stimulation of the peripheral end of a cut sensory nerve was attributed to liberation of a chemical transmitter (Lewis and Marvin, 1926). Both acetylcholine and histamine have been suggested as the possible transmitter substance (Wybauw, 1936; Wybauw, 1938b; Kwiatkowski, 1943). Atropine should abolish or eserine potentiate the reaction if acetylcholine (M-like action) is responsible, and mepyramine abolish the reaction if histamine is responsible. Holton and Perry (1951) found negative results with these chemicals and concluded that the transmitter

substance is neither acetylcholine nor histamine.

Lewis and Marvin (1926) indicated that the duration of the antidromic vasodilation lasted 5 to 10 minutes. Holton and Perry (1951) agreed, and suggested the latency of the response after the stimulation, and duration are further evidence that neither acetycholine nor histamine are responsible as they are both readily destroyed.

Noradrenaline is a powerful vasoconstrictor agent. Its action on vessels in extremities of sheep have been observed as a reduction in temperature (Webster et al., 1969).

Infusing noradrenaline into humans caused no significant change in cardiac output (Fowler et al., 1951), or only a slight increase in average cardiac output (Patel, Lange and Hecht, 1958) as reported by Harris and Heath (1962). Duration of infusion of noradrenaline was not directly reported, however assumptions from the methods indicate approximately a 5 minute duration. Concentrations were 0.2 to 0.4 micrograms of noradrenaline/kilo/min.

During continuous infusion of noradrenaline blood pressure has been shown to decline from the initial pressor level. Using a rat with one limb perfused at a controlled, constant blood supply, Gillespie and Muir (1967) found a decline in systemic blood pressure during constant infusion of noradrenaline but no decline in blood pressure in the perfused limb. They assumed that the reduced systemic pressor effect was due entirely to a diminished cardiac output, since the vasoconstrictor effect of noradrenaline in the perfused limb was observed to persist.

Pitressin exhibits the property attributed to the pressor fraction of posterior pituitary extract. Levinson and Essex (1943) described a vasoconstrictor effect in the rabbit ear on injection of pitressin.

Studying the anesthetized rabbit, cat and dog, Holtz (1932) found that pitressin had different actions in different species. In the cat, pitressin caused a rise in pulmonary arterial pressure, while in the rabbit and dog a fall in pressure occurred.

Infusing pitressin in dogs, Drepanas et al. (1961) found an increase in systemic arterial blood pressure, and reduced cardiac output (58% of control) lasting from 45 to 60 minutes after pitressin stopped. There was a drop in coronary blood flow corresponding with bradycardia and cardiac arrhythmias. This supports the conclusion of Green, Wégria and Boyer (1942) that pitressin depressed the contractile effort of the myocardium by impedance of coronary blood flow in dogs.

Conflicting evidence brought out in this literature review indicates an incomplete understanding of the controlling mechanism underlying CIVD in extremities.

A quantitative analysis of the contribution made by the extremities to total heat loss in the ruminant does not appear in the literature.

This project was designed to study control of CIVD and provide a quantitative analysis of heat loss contributed by extremities to total heat loss of the ruminant.

EXPERIMENTAL

1. Objectives

This study was designed to investigate the contribution of the extremities to total heat loss in ruminants exposed to sub freezing air temperatures, to estimate the blood flow to the extremities during cold induced vasodilation (CIVD) and to study the control mechanisms involved in the regulation of blood flow to the extremities.

2. Animals and Management

Two Holstein steers Claude and Denis, both weighing about 440 kg were used in the first series of experiments designed to estimate heat loss from the tail. These animals had been handled extensively prior to these trials and were accustomed to all experimental procedures. They were kept on summer pasture between experiments. Each animal was restrained in an individual stanchion during cold exposures. An apparatus (Fig. 1) designed to hold the tail away from the body was strapped on the animal during experiments.

In the second series of experiments two Lincoln ewes were kept in individual holding crates and maintained at approximately 20°C except during a cold acclimation period lasting six weeks. Alfalfa-brome hay was fed *ad lib* twice a day. Fleeces were 2 cm in depth at the start of the noradrenaline infusion trial. Fleeces were trimmed back to 2 cm for the cold acclimation period.

In each sheep one carotid artery had been exteriorized prior to these experiments and they had received an extensive training program

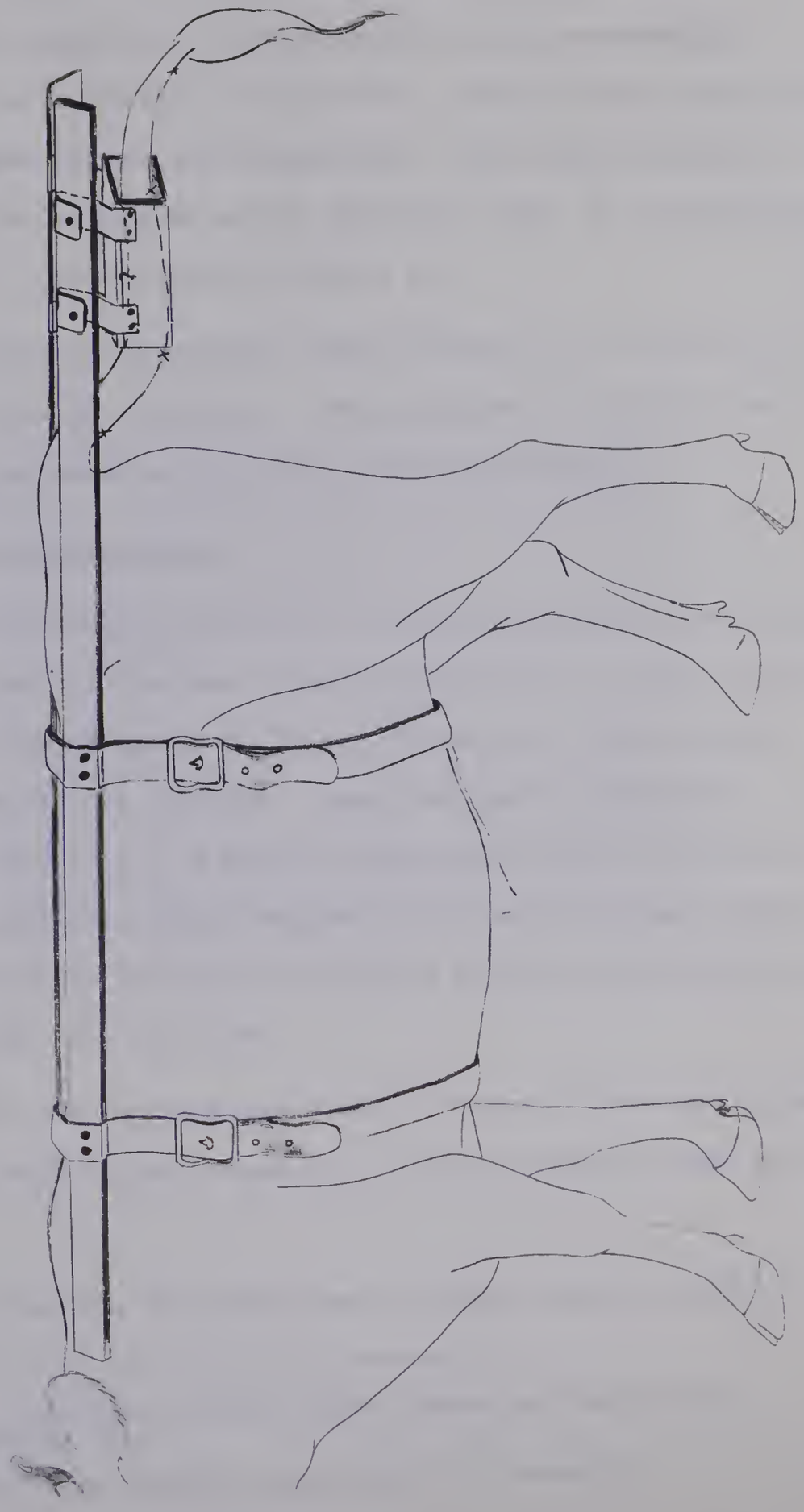

FIGURE 1 - View of ox with apparatus holding tail out from body.

consisting of at least four preliminary trials for each sheep.

One sheep died of cardiac arrest during noradrenaline infusion on the fifth week of acclimation. The surviving sheep and one Suffolk wether were used subsequently. They were maintained at 20°C and fed alfalfa-brome hay ad lib twice a day. At the beginning of the trials, fleece depth was about 2 cm.

During all experiments sheep were held in a crate (Fig. 2) positioned in front of a mirror. The mirror had a definite calming effect most necessary during blood pressure measurements.

3. Temperature Measurements

A Honeywell Electronik 16[1] recorder was used for measuring tail temperatures in the oxen. The machine was set to triple print on six positions and single print on two others for a total of eight different temperature readings. Copper-constantan thermocouple junctions backed by a 1 cm diameter polyethylene disk were glued in position and held by winding surgical tape over the tip and around the tail. Thermocouple wire was fed through a porthole and attached to the recorder outside the cold room.

The tail was held away from the body as illustrated in Fig. 1. This was to prevent direct conduction of heat between the body and the tail.

A Speedomax W[2] recorder set to double print six different

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[1]Honeywell - Industrial Products Group, Wayne and Windrim Aves., Philadelphia 44, Pa.

[2]Leeds and Northrup (Canada) Ltd., Toronto 15, Ontario.

Figure 2. View of holding crate used during series 2.

temperatures was used in experiments with sheep. Copper-constantan junctions were glued into position and held with tape. The recorder was again placed outside the cold room.

In experiments involving measurements of blood pressure and heart rate a thermistor was attached to the ear and connected to a telethermometer[3]. The output of the telethermometer was coupled to a Sanborn 350-1300 C preamplifier[4]. Coccygeal venous temperature was measured from a 30 gauge thermocouple previously chronically implanted into one vein at the base of the tail under posterior epidural anaesthesia. The thermocouple was drawn up to the dorsal side of the tail through a small hole bored in a coccygeal vertebra. This was done to reduce the risk of contamination from feces.

4. Direct Calorimetry

The tails of Claude and Denis, and tails from two other Holstein-type animals were obtained at slaughter and used to determine, by direct calorimetry, the rate of heat loss from the dead tail on immersion in a water bath. Surface area of each tail was obtained by Simpson's rule*. The tails were then clipped, weighed and run through the following experiments.

An insulated cylindrical tank was designed such that a tail might be submerged along with an immersible pump and attached hose. The immersible pump provided thorough mixing of water. A dead tail was

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[3] Yellow Springs Instrument Co., Yellow Springs, Ohio.

[4] Hewlett-Packard (Canada Ltd., Montreal, Quebec.

* (Handbook of Chemistry and Physics, 47th edition, 1966-1967).

warmed to 40°C in a stirred water bath nearby and plunged into the tank containing water at room temperature (Fig. 3).

Continuous temperature tracings of T_C (temperature of tail core), T_S (temperature of tail surface), and T_{H_2O} (temperature of water in tank) were taken for 15 minutes on a Honeywell Electronik 19[1] and an Esterline Angus (model Speed Servo)[5].

T_S and T_C were taken at points 1, 4, 10 and 16/20 of the distance from the base to the tip of the tail. Thermocouples recording T_C were inserted into the center of the tail. T_{H_2O} was taken at four positions inside the length of tubing leading from the immersible pump.

Each set of four thermocouples was hooked in series with a reference temperature of 0°C (Fig. 4), and temperature measurements obtained from the EMF's generated thereby.

Trial experiments without the dead tails indicated no change in temperature of water in tank. Heat loss (H) from tails to water was therefore calculated by multiplying the known mass (M) of water (g) by the °C rise in temperature of water, ΔT_{H_2O} or

$$H = M_{H_2O} \cdot \Delta T_{H_2O} \quad \text{.....(1)}$$

The specific heat of the tail could be calculated from

$$H = \rho \cdot \Delta T_t \cdot M \text{ (Hardy, 1949)} \quad \text{.....(2)}$$

where

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[5]Esterline Angus Instrument Co., Inc., Indianapolis, Indiana.

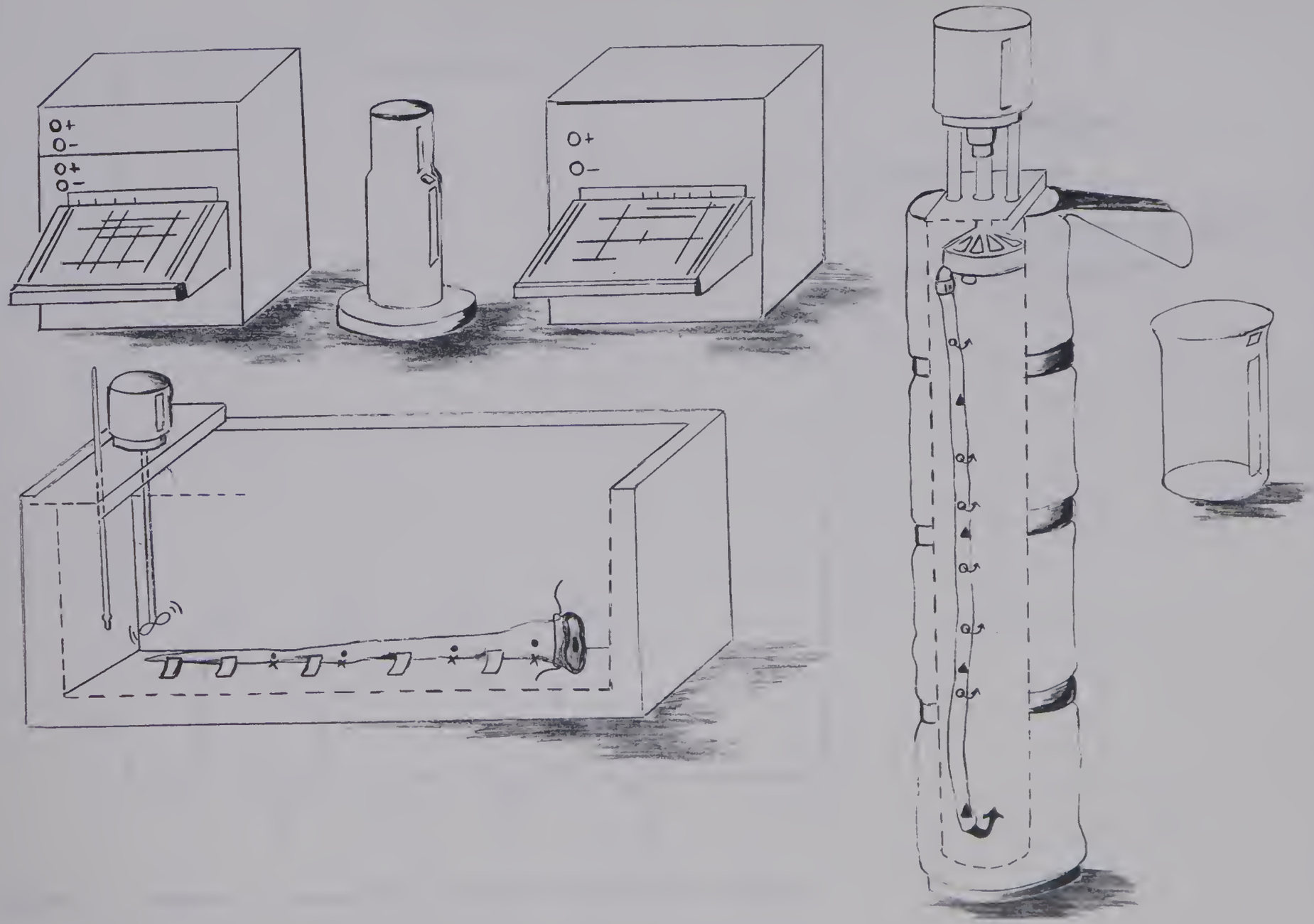

Figure 3. View of apparatus measuring heat loss from dead tail.

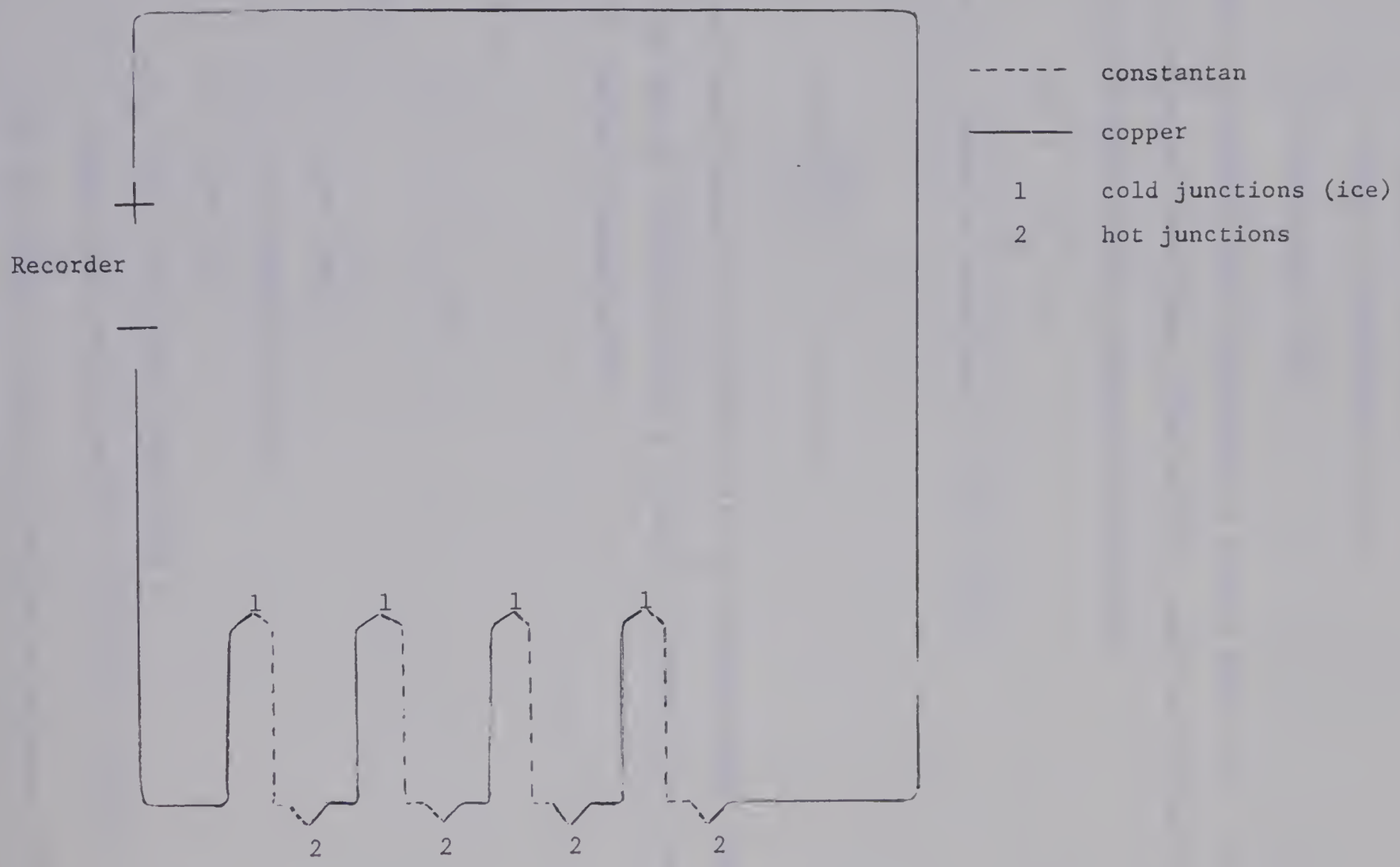

Figure 4. Diagram of thermocouple circuit connected in series.

H = heat loss from tail to water

ρ = specific heat of tail

ΔT_t = change in temperature of tail

M = mass of dead tail

However ΔT_t cannot easily be measured in live or dead tails. In a body of relatively small mass, like the tail, heat loss during cooling can also be expressed with reasonable accuracy by,

$$H = f \cdot \Delta T_s \cdot M \quad \text{.....(3)}$$

where ΔT_s = change in temperature of skin surface.

Then

$$f = \frac{\rho \cdot \Delta T_t}{\Delta T_s} \quad \text{.....(4)}$$

The heat loss constant for the live tail (C_s) during maximal vasoconstriction can then be determined using ΔT_s and f to estimate heat loss per unit of time as in equation (3).

Then

$$H_t = C_s (T_s - T_a)t \cdot A \quad \text{.....(5)}$$

where

t = time (sec)

T_a = air temperature (°C)

A = area (m^2)

H_t = Heat loss (kcal) during time t

C_s = Heat loss constant using T_s ($kcal/m^2 \cdot °C.sec$)

Heat loss from the dead tail can also be expressed directly in terms of Newton's cooling constant.

$$H_t = C_c (T_c - T_s)\ t \cdot A \text{ (Hardy, 1949)} \quad \ldots\ldots\ldots (6)$$

where

C_c = heat loss constant using core temperature ($kcal/m^2 \cdot °C \cdot sec$)

T_c = temperature of tail core (mean temperature of implanted thermocouples)

T_s = temperature of tail surface

t = time (sec)

A = area (m^2)

Implementing C_c found from dead tails and taking $T_{s/c}$ (subcutaneous temperature at position #2) as T_c of live tails during maximal vasoconstriction C_s' (heat loss constant for live tail using surface temperature) may be found by equating

$$H_t = C_c (T_{s/c} - T_s)\ t \cdot A$$
$$= C_s' (T_s - T_a)\ t \cdot A \quad \ldots\ldots\ldots\ldots\ldots\ldots (7)$$

Equation (7) is essentially the same as equation (5). The accuracy of C_s' however depends on the accuracy with which $T_{s/c}$ reflects T_c, which must be determined by experiment.

Total heat loss from the live tail (H_t) may now be found during any cooling interval using (5) or (7). The portion of H_t due to an actual reduction or increase in temperature of the surface (ΔT_s) may be called H_x and is found from (3).

$$H_x = f \cdot \Delta T_s \cdot M \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots (8)$$

The only appreciable source of heat to the tail is that supplied by the blood (H_s), and is given by

$$H_t \pm H_x = H_s \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (9)$$

Blood flow may then be calculated from the formula

$$H_s = S\ (T_{in} - T_{out})\ F \quad \text{(Hardy, 1949)} \qquad \ldots\ldots\ldots\ldots (10)$$

where

S = specific heat of blood (0.89) (Mendlowitz, 1948)

T_{in} = rectal temperature taken as temperature of arterial blood entering tail

T_{out} = temperature of venous blood leaving the tail

F = blood flow (ml/min)

$(T_{in} - T_{out})$ may be expressed as ΔT_b

5. Blood Pressure Measurements

The exteriorized carotid artery was catheterized with an intramedic polyethylene catheter (PE - 190/536)[6] via a 13 gauge bleeding needle. The catheter was connected to a three way tap tied to the top of the halter by wire twists, and flushed with heparinized saline[7] (10 U.S.P. units of heparin per ml of physiological saline). Surgical tape and gauze formed a secure bond between the carotid loop and the catheter to prevent it pulling out. The excess catheter was coiled and secured against the animal's neck with 'curls' of wool and wire twists. This technique prevented snagging of the catheter as well as providing it protection against freezing.

Seven feet of tubing, filled with heparinized saline, connected the three way tap with a pressure transducer[4] outside the cold room. A Sanborn 350-1100 C carrier preamplifier[4] recorded blood

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[6] Becton, Dickinson and Company, Parsippany, N.J. 07054.

[7] Riker Pharmaceutical Company Ltd., Cooksville, Ontario.

pressure.

Medical grade polyethylene tubing (no. RX 079 I.D. .079)[8] was used for the blood pressure line except for a flexible 12 inch piece of tygon (I.D. .079)[9] proximal to the animal to prevent kinking.

The line was positioned inside a tygon tube (I.D. 3/8) insulated with a layer of cloth, a heating tape (75 watt, 15 ft)[10], a fiberglass strip, polyethylene, and masking tape, in that order. One end of the tygon tube was attached to the holding crate in a position above the animal's head.

A collar was made with the loose end of the heating tape and fastened around the animal's neck. This collar provided enough heat to keep the catheter and tap from freezing.

At very low air temperatures an additional feature was required to prevent freezing. An insulative layer consisting of wool glued to dental dam rubber was wrapped around the tygon line attached to the animal. Also a large (30" × 15" × 2 1/2") piece of fiberglass wrapped in cheesecloth was placed against the neck in place of the collar.

6. Recording Heart Rate

Heart rate was obtained either from the ECG using surface electrodes attached to a Sanborn 350-2700 C high gain preamplifier[4], or from pulse pressure measured on the Sanborn 350-1100 C carrier pre-amplifier.

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[8] Becton, Dickinson and Company, Rutherford, N.J.

[9] U.S. Stoneware Co., Akron, Ohio.

[10] Heat Tape Inc., 1812 S. Halsted St., Chicago Heights, Ill.

When surface electrodes were used three were required, one on the back above the second or third thoracic vertebra and one one each side, just above the elbow, on the same circumference.

Intervals between successive beats, and thus rapid changes in heart rate were displayed using a Sanborn 350-3400 A cardiotach. pre-amplifier[4].

7. Measurement of pO_2

Arterial and venous pO_2 was measured with the Blood Micro System (type BMS3), PHM71 acid base analyzer and pO_2 electrode (type E5046)[11].

Animals were catheterized in the carotid artery and jugular vein as described previously. Heparinized 5 cc syringes were used to draw blood. Samples were taken at the beginning of experiments and every 10 minutes thereafter. These were sealed and immediately placed in ice water until analysis.

8. Infusion of Drugs

Drugs were infused into the jugular vein (catheterized as described previously).

A tygon infusion line (I.D. .079) was placed next to the blood pressure line and connected in similar fashion to an infusion/withdrawal pump (model 600-950 V[12] or model 950[13]) kept outside the cold room.

Noradrenaline (levophed[14]) and pitressin[15] were prepared at

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[11]Radiometer, Copenhagen, Denmark.

[12]Harvard Apparatus Co., Dover, Mass.

[13]Harvard Apparatus Co., Millis, Mass.

[14]Winthrop Laboratories, Aurora, Ontario.

[15]Parke, Davis and Co., Ltd., Brockville, Ontario.

appropriate concentrations in a physiological saline solution infused at 4.3 ml/min.

9. Sympathectomy

The left ears of one Lincoln ewe and one Suffolk wether were sympathectomized by removal of about 2 cm of the left vago-sympathetic trunk in the region of the neck. These operations were performed under general anaesthesia with Nembutal[16] and normal aseptic procedures were followed. Pronounced vasodilation of the left ear was apparent within minutes of section of the sympathetic nerves.

10. Experiments

Two series of experiments were performed.

Series 1

In these experiments two steers, Claude and Denis were used to investigate the pattern of cold induced vasodilation in the tail of the ox, and to estimate the nature and extent of changes in blood flow to the tail associated with this phenomenon.

In all experiments the tail was held out from the body (Fig. 1) and 4 thermocouples were attached to the ventral skin surface of the tail at points distal to the junction of the tail and body by 1/20, 4/20, 10/20 and 16/20 of the length of the tail. A single measurement of subcutaneous temperature was made deep to the 2nd proximal skin thermocouple in 1(b) and 1(c).

1(a). In six trials each animal was introduced to a refrigerated room at an air temperature of about +18°C. Once skin temperature measurements had stabilized, air temperature was reduced as

rapidly as possible to about -18°C. The experiment ran for 1 to 2 hours after the onset of cooling.

1(b). In four trials on each animal, air temperature was reduced from +18°C to -7°C in three stages. Initially air temperature was cooled slowly to +4°C over a period of 40 min and then held at 4°C for a further 40 min. At that time air temperature was rapidly reduced to -1°C and held at that for 40 min, then further cooled to -7°C and maintained for 120 min.

After 120 min at -7°C, a constriction was applied at the base of the tail for 20 minutes. A small rubber cork was fitted into the protective space created by two parallel lines of cartilage running ventrally alongside the main coccygeal artery. A rubber band was then tied tightly around the cork and the tail to obtain a constriction. After 20 min the obstruction was removed and temperatures recorded for another 20 minutes.

1(c). Measurements were made of skin temperatures at four sites, tail subcutaneous, rectal and coccygeal venous temperatures in four trials on Claude and three on Denis in which air temperature was reduced in stepwise fashion as above. Arterial constriction was not applied in these trials. Coccygeal venous temperature measurements from Denis could not be obtained after the first experiment due to a broken thermocouple. The anatomical structure at the base of Denis's tail was such that complete arterial occlusion was not possible.

1(d). The tails of Claude and Denis, obtained post mortem and two other tails were used to determine heat loss constants by direct calorimetry as described above.

Series 2

In these experiments three sheep were used to study some aspects of the control mechanisms involved in thermoregulation in the extremities (legs and ears) of sheep exposed to air temperatures below freezing point.

2(a). Measurements were made of blood pressure, heart rate, and skin surface temperature at three sites, on the left and right ears and on the left fore shank in two sheep, A and B. Blood pressure and heart rate were recorded from the continuous trace at 5 min intervals. Blood pressure was always recorded as systolic pressure over diastolic pressure. In experiments where samples of arterial and venous blood were taken for blood gas analysis, blood pressure was not measured.

For cold acclimation animals were shorn and continuously exposed to an ambient temperature of 4°C, decreased by 1°C approximately every 3 1/2 days, in an attempt to provide a constant intensity of cold stress for the sheep as their fleece was growing.

The trial lasted 14 weeks with 6 weeks allowed for cold acclimation, and involved 8 experiments (5 with blood pressure, 3 with blood gas analysis) prior to cold exposure, 12 (6 with blood pressure, 6 with blood gas analysis) during and 8 (4 with blood pressure, 4 with blood gas analysis) after cold exposure.

Each experiment lasted 1 1/2 hours after the point at which both heart rate and skin temperatures leveled off following introduction into a cold room. The first experiment was run at an ambient temperature of -4°C. In following experiments air temperature was

progressively lowered by 2°C each week for 4 weeks until cold acclimation began. Experiments then started at -6°C and air temperature was lowered each week as above until -16°C was reached. Subsequent trials were conducted at -16°C.

After taking a steady heart rate for 30 min noradrenaline was infused at a rate of 1 microg/kg/min for 30 minutes.

Animals were catheterized only once a week, and catheters left in the animal overnight. Blood pressures were taken the first day and blood gas analysis done the next.

Prior to the main trial six blank saline infusions were done on each sheep. These did not involve blood gas analysis.

Sheep A died the third week into cold exposure. Two blood gas analysis trials with sheep B were lost due to equipment breakdowns. During cold exposure one catheterization was not possible in sheep B.

2(b). Temperature measurements were taken from skin surface of left and right ears and left and right fore shanks of two sheep, B and C exposed usually for 3 hours to an ambient temperature of about -25°C. Blood pressure and heart rate were taken during two experiments in sheep B after sympathectomy.

Noradrenaline was usually infused for at least 100 minutes at 1 microg/kg/min. After using a test* sheep to determine dosage rate, Pitressin was infused in four experiments at 1 P.U./kg/min for 15 and 30 min and at 3 P.U./kg/min for 30 and 40 min respectively in sheep B.

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*Pitressin was administered at various dosages to an extra sheep with one carotid artery exteriorized for blood pressure measurement.

Four experiments with sheep B and seven with sheep C were done prior to sympathectomy, with 12 following in sheep B. Only four runs were obtained from sheep C after sympathectomy.

RESULTS and DISCUSSION

Results

Series 1

The surface area and mass of dead and live tails appear in Table 1. Mass of live tails was obtained from surface area assuming a constant ratio of mass to surface area in live and dead tails.

To eliminate errors in transfer of tail from water bath to tank, in series 1(d), readings commenced two min after immersion. They were taken from the continuous temperature records at one minute intervals thereafter, for eight minutes.

Figures for heat loss from dead tails (H), f and C_c, as determined from formulae (1), (3) and (6), respectively, appear in Table 2. There was considerable variation in C_c and f between individual preparations. Subsequent calculations of heat loss from live tails were based on the individual values for C_c and f obtained for Claude and Denis.

In experiments on live animals (series 1(a), 1(b) and 1(c)) seven records were taken over any twenty minute interval for each thermocouple junction except one. Eleven readings were recorded for $T_{s/c}$. An example of the treatment of data obtained over a typical twenty minute interval appears in Table 3.

Table 3 and Fig. 5 clearly show the temperature gradient occurring from position #1 on the tail to position #4 (proximal to distal). Position #3 and #4 sometimes displayed 'hunting' to a small extent in the latter part of experiments 1(b) and 1(c).

Results of trials involving coccygeal artery occlusion appear

TABLE 1. Surface areas and mass of tails from Claude, Denis and two Holstein cows (H_1 and H_2) .

	Claude	Denis	H_1	H_2
Surface area (dead) ($m^2 \times 10^{-1}$)	0.81	0.96	0.88	0.75
Mass (dead) (kgm)	0.94	1.13	1.03	0.85
Surfacearea (live) ($m^2 \times 10^{-1}$)	0.73	0.68		
Mass (live) (kgm)[1]	0.85	0.81		

[1]Calculated from surface area (live) assuming a constant ratio of mass to surface area in dead and live tails.

TABLE 2. Constants describing heat loss from dead tails to water derived by direct calorimetry.

	Interval (min)	Claude	Denis	H_1	H_2
Change in temperature of tail surface (ΔT_s)	0-5	6.5	8.7	8.5	6.7
	0-10	8.5	10.6	10.0	8.6
	2-7	3.2	4.4	3.2	3.4
	2-10	4.4	5.0	4.0	4.4
Heat loss from tail to water[1] (H, kcal)	0-5	3.614	3.447	5.601	5.529
	0-10	5.560	5.515	8.334	6.772
	2-7	2.919	2.895	4.918	2.073
	2-10	3.753	4.136	6.011	3.040
"Heat content" (f)[2]	0-5	0.59	0.35	0.64	0.97
	0-10	0.70	0.46	0.81	0.93
	2-7	0.97	0.58	1.49	0.72
	2-10	0.91	0.73	1.46	0.81
Heat loss constant[3] (C_c kcal/m^2·°C·sec)	0-5	0.0279	0.0112	0.0444	0.0425
	0-10	0.0265	0.0175	0.0396	0.0290
	2-7	0.0249	0.0160	0.0367	0.0168
	2-10	0.0235	0.0161	0.0340	0.0163

[1]From equation (1).

[2]From equation (3).

[3]From equation (6).

As a large error is obviously created by including the first 2 minutes, only values obtained in the 2-10 minute interval are used.

TABLE 3. An example of the results obtained in one 20-min period of a single experiment in series 1(c), and treatment of the data.

Interval (min)	T_{rectal} (°C)	T_A (°C)	T_s at positions #1 (°C)	#2 (°C)	#3 (°C)	#4 (°C)	$T_{I/V}$ (°C)	$T_{s/c}$ (°C)
140	39.4	-6.7	33.3	29.7	26.1	24.4	30.3	33.8
	39.4	-6.7	33.6	30.0	25.6	23.9	30.2	33.5
	39.4	-6.7	33.6	30.0	23.9	21.7	29.4	33.5
150	39.4	-6.7	32.2	27.8	21.1	19.4	29.4	33.6
	39.4	-6.7	33.6	26.7	20.0	17.5	29.2	32.9
	39.4	-6.7	33.1	26.1	18.9	15.6	29.4	33.3
160	39.4	-6.9	33.3	24.7	16.9	13.1	29.3	32.7
								32.2
Average	39.4	-6.7	33.3	27.8	21.8	19.4	29.6	33.3
								31.9
								32.8
								33.1

T_s at time 140 = average of 33.3 + 29.7 + 26.1 + 24.4 = 28.4°C.
T_s at time 160 = average of 33.3 + 24.7 + 16.9 + 13.1 = 22.0°C.

ΔT_s = 6.4°C.

T_s = average of 19.4 + 21.8 + 27.8 + 33.3 = 25.6°C.
T_s = -6.7°C.
T_A = 29.6°C.
$T_{I.V.}$ = 39.4°C.
T_{rectal}

TABLE 3. (Continued...)

Total heat loss, $H_t = C_s (T_s - T_a)\ t \cdot A$ ---------- (5)

$= 4.7 \times 10^{-3} [25.6 - (-6.7)]\ 20 \cdot 60 \cdot 0.73 \times 10^{-1}$

$= 13.24$ kcal/20 min

Heat loss from ΔT_s, $H_x = f \cdot \Delta T_s \cdot M$ ---------- (8)

$= 0.91 \cdot 6.4 \cdot 0.85$

$= 4.93$ kcal/20 min

Heat gain from blood, $H_s = H_t \pm H_x$ ---------- (9)

$= 13.24 - 4.93$

$= 8.31$ kcal/20 min

Blood flow, $$F = \frac{H_s}{*S(T_{in} - T_{out})}$$ ---------- (10)

$$F = \frac{8.31}{0.89\ (39.4 - 29.6)}$$

$F = 48$ ml/min

*Taken as 0.89 (Mendlowitz, 1948).

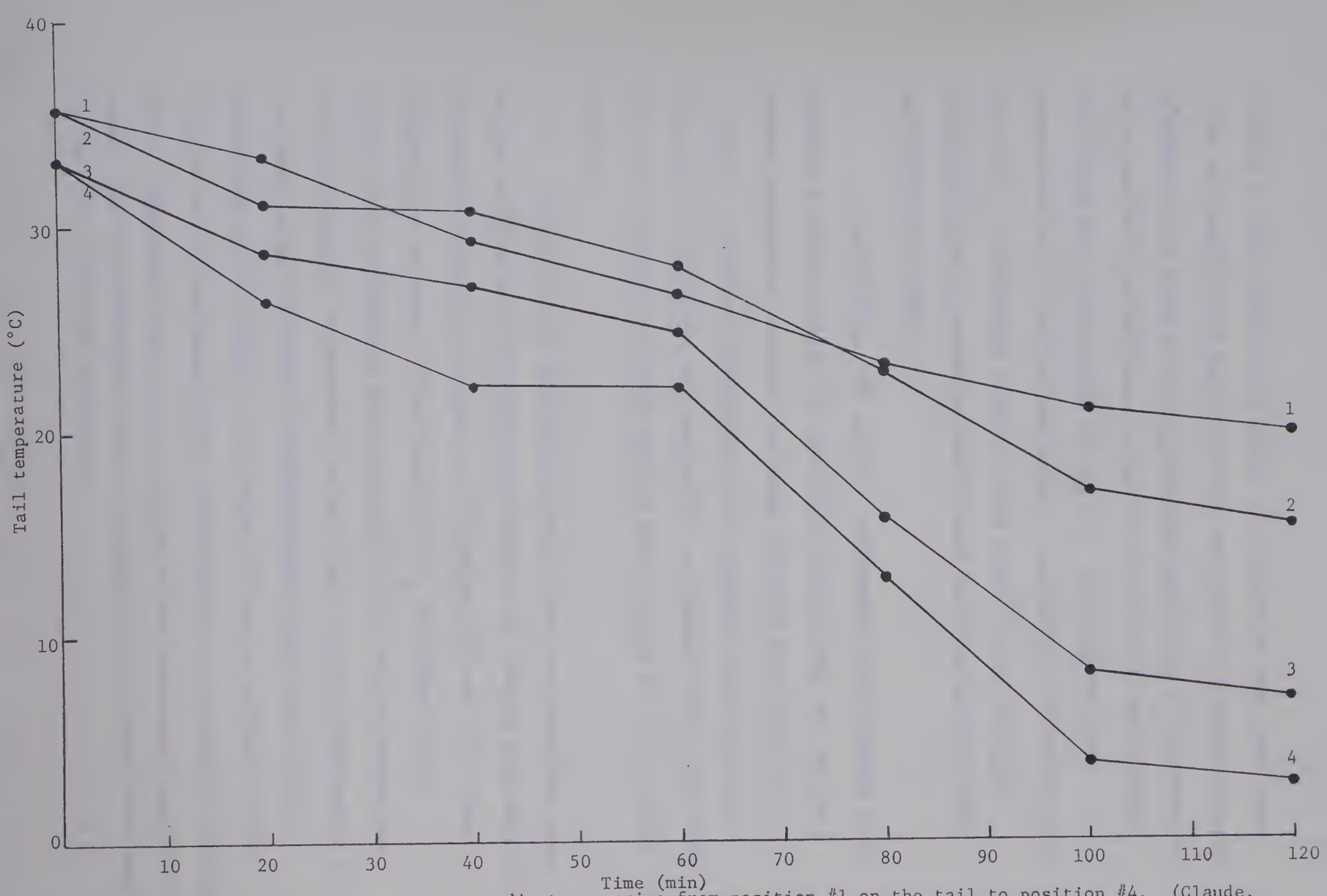

Figure 5. Example of the temperature gradient occurring from position #1 on the tail to position #4. (Claude, series 1(a) #2).

in Table 4. Only results from Claude are included in this table since it was not possible with Denis to obtain any degree of arterial occlusion as manifest by a rapid fall in T_s following application of the tourniquet. The values for C_s' as determined using (7) were obviously more deviant than values for C_s found using (3) and (5). This suggests that measurements of $T_{s/c}$ did not accurately or consistently reflect the true value of T_c. The average value of 0.0047 kcal/m^2·°C·sec for C_s was subsequently used in determinations of total heat loss and blood flow as illustrated in Table 3.

In both series 1(b) and 1(c), experiments which involved step cooling, a definite pattern for heat loss occurred (Fig. 6). As the ambient temperature decreased the animal increased heat loss till at a constant low ambient temperature (-7°C) a maximum heat loss occurred after which it decreased and leveled off. An example of heat loss from the tail in series 1(a), which involved rapid cooling to -17°C, appears in Fig. 7.

Results of all experiments (Table 5) show that mean skin temperatures in the tails at air temperatures below -7°C, (series 1(a)) were not appreciably different from those at -7°C (series 1(b) and (c)). This suggests that at about -7°C in these trials T_s had fallen to its lowest value and therefore vasoconstriction in the tail was maximal and heat loss minimal. Unfortunately in the trials involving measurements of coccygeal venous temperature and thus coccygeal venous blood flow, air temperature was not allowed to fall below -7°C and no increase in blood flow due to CIVD was demonstrated. Nevertheless it is reasonable to assume that a considerable and prolonged decrease in room temperature below -7°C would cause an increased heat loss and necessitate an increased blood supply to the tail to maintain temperature.

TABLE 4. Constants describing heat loss from tails during coccygeal artery occlusion.

Position	Temperature measurements (°C) during 20 min arterial constriction			Heat loss constant kcal/m^2·°C·sec	
	ΔT_s	$(T_s - T_a)$	$(T_{s/c} - T_s)$	C_s	C'_s
1	9.2	18.6	7.6	0.0043	0.0096
2	11.2	21.9	11.0	0.0045	0.0118
3	11.3	20.1	7.7	0.0049	0.0090
4	8.5	14.5	3.1	0.0051	0.0050
			Mean	0.0047	0.0089
			(S.E.)	±0.0002	±0.0014

C_s determined from equations 3 and 5.

C'_s determined from equation 7.

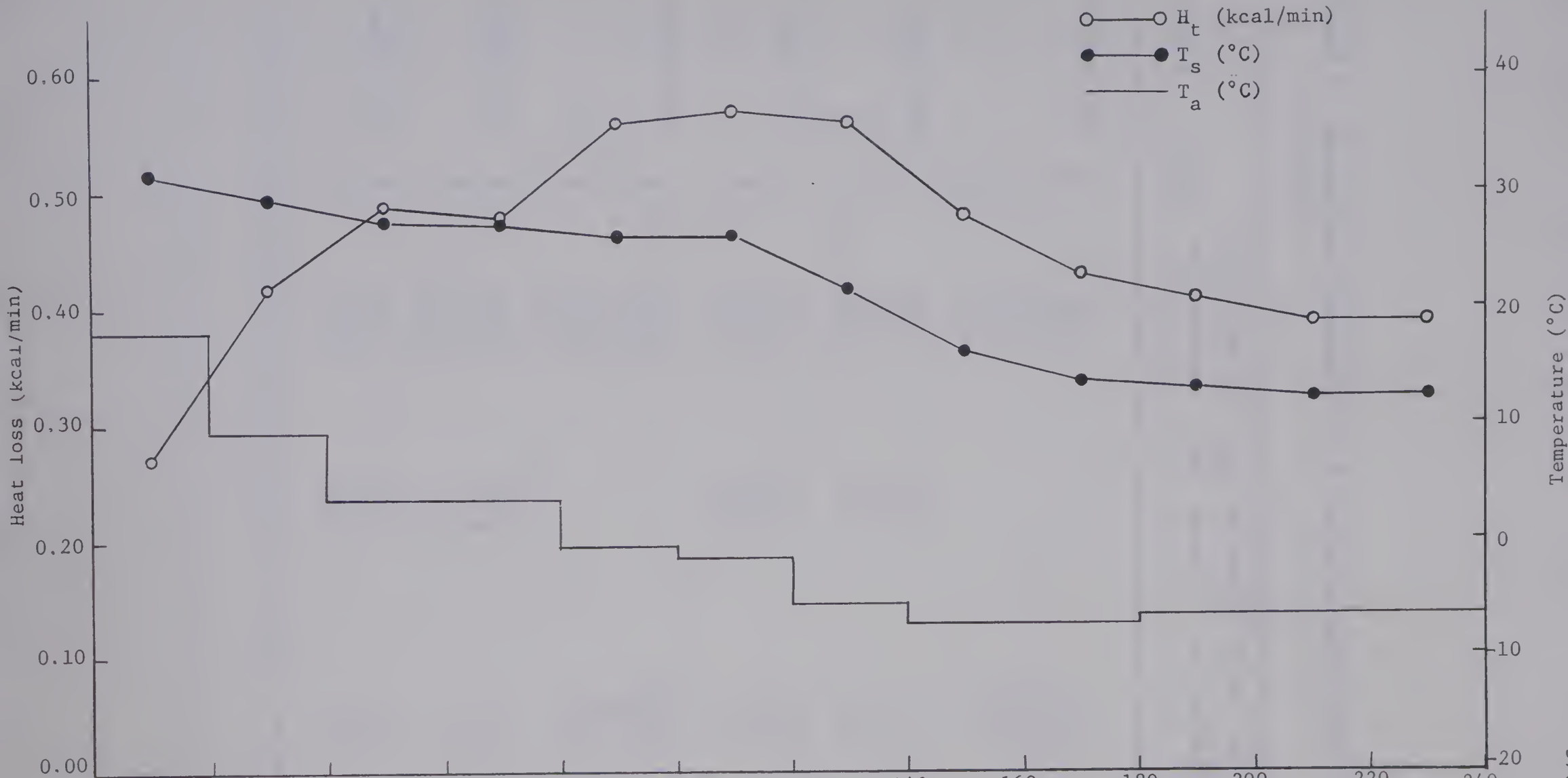

Figure 6. Example of heat loss (H_t) and skin temperature (T_s) from tail on step cooling (series 1(b) #4).

TABLE 5. Minimum skin temperature (T_s) reached in series 1(a), 1(b) and 1(c).

Series	Trial	T_s in last 20 min interval °C	$T_{s/c}$ in last 20 min interval °C	T_a in last 20 min interval °C	Duration of experiment min
Claude 1(a)	1	14.2		-17.2	120
	2	11.1		-17.2	120
	3	14.8		-12.2	60
	4	13.5		-12.0	60
	5	19.7		-10.2	60
	6	12.8		-17.8	100
Claude 1(b)	1	16.6	27.5	- 7.4	240
	2	23.5	34.2	- 6.7	240
	3	20.5	25.0	- 7.1	240
	4	12.6	15.3	- 6.6	240
Claude 1(c)	1	19.2	31.3	- 6.8	240
	2	15.9	15.3	- 6.8	240
	3	15.1	15.0	- 6.7	240
	4	16.3	24.2	- 6.4	240
Denis 1(a)	1	10.3		-17.9	160
	2	20.4		-12.2	60
	3	13.3		-15.5	80
	4	12.9		-11.1	60
	5	14.6		-11.4	60
	6	17.0		- 7.4	60
Denis 1(b)	1	10.3	13.6	- 7.2	120
	2	13.9	12.9	- 7.3	120
	3	12.1	7.6	- 7.0	120
	4	10.0	9.0	- 6.5	120
Denis 1(c)	1	13.4	12.7	- 6.7	120
	2	10.3	9.2	- 6.5	120
	3	8.9	10.4	- 6.7	120

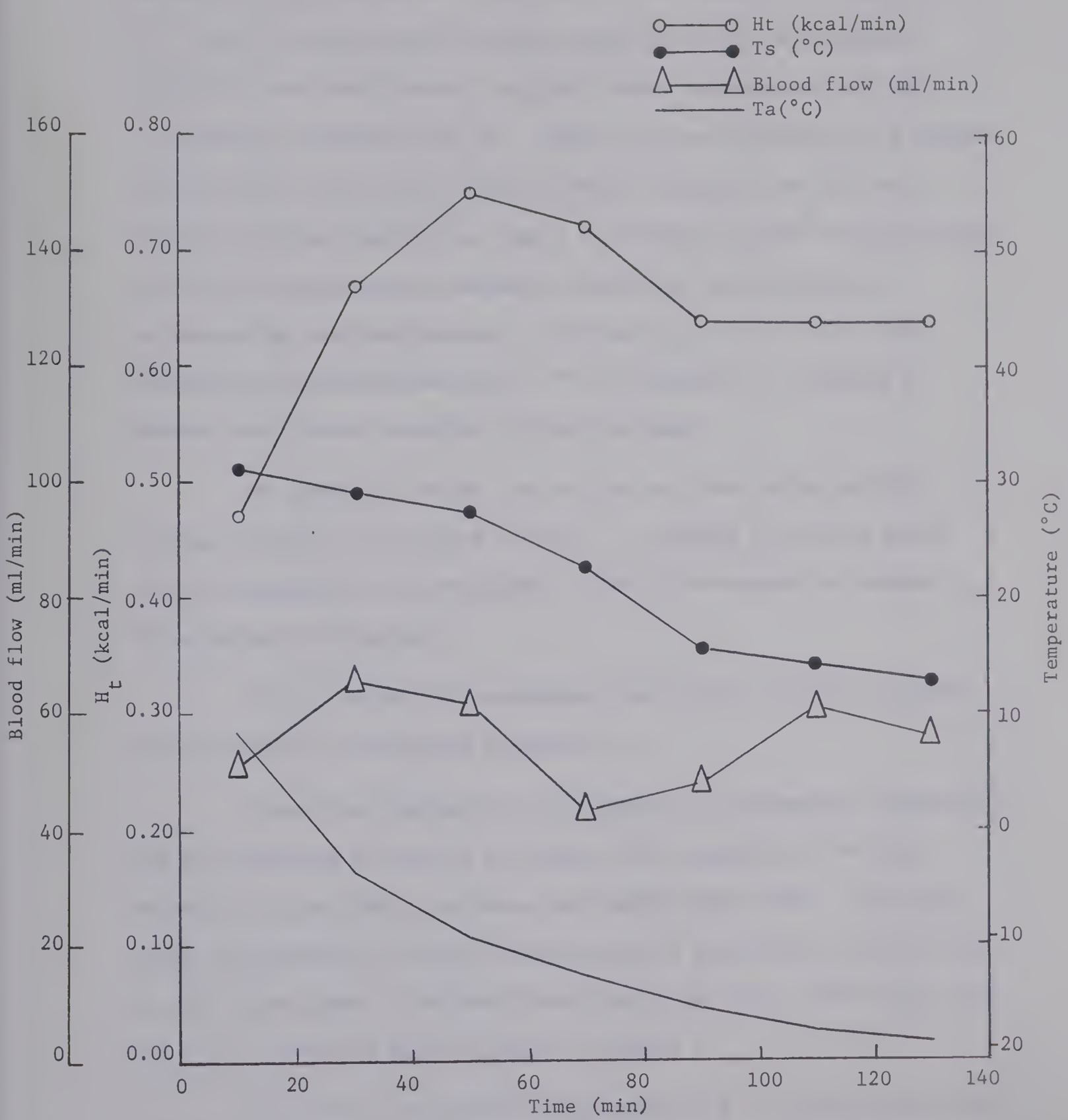

Figure 7. Example of blood flow, heat loss (H_t) and skin temperature (T_s) occurring on step cooling of tail (series 1(a) #1).

During the time taken by the tail to reach minimum skin temperature, T_s reacted as a function of room temperature (Figs. 6 and 7). After the tail reached minimum temperature and local thermal equilibrium was established, coccygeal venous temperature and thus ΔT_b remained constant (Fig. 8). Since ΔT_b must certainly be a direct function of T_s and since T_s reacts during cooling of the tail as a function of room temperature, then ΔT_b obtained in step cooling during series 1(c) can be used to estimate blood flow from the tail at corresponding room temperatures in series 1(a) or 1(b). For room temperatures reaching lower than -7°C the maximum ΔT_b obtained at thermal equilibrium in series 1(c) may be used.

An example of blood flow to the tail associated with fast cooling (series 1(a)) appears in Fig. 7. Although T_s did not settle out at a minimum in this experiment, blood flow started to increase 70 min after cold exposure.

During series 1(c) estimated blood flow to the tail ranged from 21 ml/min to 120 ml/min (Appendix 1).

"Reactive hyperaemia is independent of any nervous connections and may therefore be used as an index of the capacity of the blood vessels to dilate"(Bell, Davidson and Scarborough, 1968). The 10 min reactive hyperaemia following constriction in series 1(b) may therefore be used as an index of maximum blood flow to the tail. The results for H_t, H_x, H_s and blood flow (F) appear in Table 6.

There was a considerable variation in F. In calculating blood flow during reactive hyperaemia, ΔT_b was taken from the values obtained during series 1(c) in which arterial occlusion was not applied.

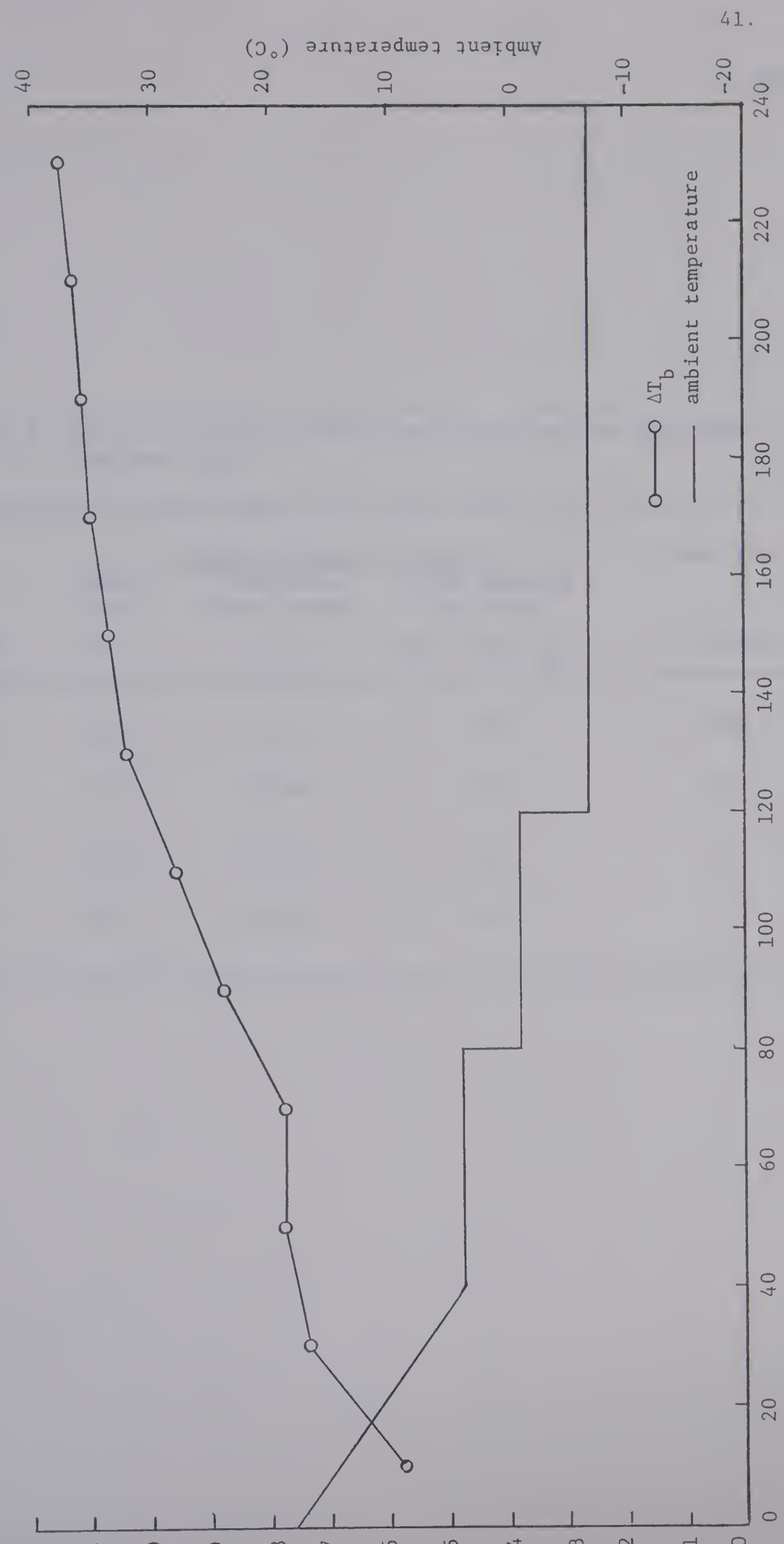

Figure 8. Average temperature difference between arterial and venous blood (ΔT_b) taken during the same time intervals in four trials in series 1(c).

TABLE 6. Heat exchange and blood flow during reactive hyperaemia (series 1(b)).

Trial	Heat exchange (kcal/min)			Blood flow
	Heat loss	Change in heat content	Heat supplied by blood	
	H_t	H_x	$H_s = (H_t + H_x)$	F, ml/min
1	0.50	1.13	1.63	160
2	0.44	0.86	1.30	127
3	0.40	0.87	1.27	125
4	0.29	0.66	0.95	93

Estimated blood flow was less in series 1(c) than following reactive hyperaemia in 1(b). It is probable therefore that the values for ΔT_b from 1(c) overestimate actual ΔT_b in 1(b) and thus underestimate blood flow during reactive hyperaemia. Also slight differences in tail temperature between experiments in series 1(b) could exaggerate the variation due to ΔT_b. Therefore it is reasonable to assume that the higher values obtained for F (Table 6) more closely approximately maximal blood flow of which the tail is capable.

Discussion

Series 1

Maximal blood flow to the tail.

The values f found for each dead tail are functions of the specific heat of that tail. They should however be higher than true specific heat as, with M constant, T_s in formula (3) will be lower than T_t in formula (2). The function f varied considerably from tail to tail (Table 2(a)) as expected since specific heat does vary greatly between individuals (Burton and Edholm, 1955).

Forster's (1946) maximal recorded value for blood flow was 31.7 cc/100 cc of hand tissue/min at an ambient temperature of 38°C. This may be compared with blood flow/m^2 in the tail as follows. Let

X = possible blood flow to the tail

V = volume of tail (determined by displacement from dead tail)

x = blood flow in hand (Forster, 1946)

v = volume of hand

Then

$$\frac{X}{V} = \frac{x}{v}$$

$$\frac{X}{825} = \frac{31.7}{100}$$

$$X = 262 \text{ ml/min}$$

However values obtained for maximum blood flow during reactive hyperaemia (Table 6) are lower. This is very likely due to the value taken for ΔT_b being high. The difference between arterial and venous blood temperature (ΔT_b) would likely be less than 11.5°C, since this

value was taken during a lower flow than that occuring during reactive hyperaemia.

A faster flow during reactive hyperaemia would not allow blood to loose as much heat to the tail as with lower flow rates.

Mentioned in the review of literature is a maximal blood flow for fingers of 1080 cc/m^2, min as determined using Greenfield's (1963) reasoning. This calculation is questionable as Greenfield is converting cc of total hand tissue into skin area. The volume of the hand contains a considerable proportion of bone which has a relative meagre vascular supply compared to skin. Greenfield's reasoning would then likely lead to a low estimate of maximum blood flow to skin.

Using the maximum blood flow to the tail estimated to be 160 ml/min, and knowing area of the tail to be 0.0732 m^2 then maximum blood flow during reactive hyperaemia in the present experiments was 2186 ml/m^2 min. Extrapolation from Forster's figures would give 3580 ml/m^2 min.

The results of the experiments may be used to estimate blood flow to the tail at an environmental temperature of -40°C (T_s minimal and constant) from

$$H_t = C_s (T_s - T_a)\, t \cdot A \qquad (7)$$

where

C_s = 0.0047 kcal/m^2·°C·sec (Table 2(b))

t = 60 sec

A = 0.732 × 10^{-1} m^2

T_a = -40°C

T_s = 13°C

T_s was obtained from an average of minimum temperatures reached in series 1. Carrying out the above calculation heat loss (H_t) from the tail was found to be 1.1 kcal/min.

The blood flow required to maintain this temperature is found from formula (10) as follows:

$$H_s = S \cdot \Delta T_b \cdot F \quad \text{or} \quad F = \frac{H_s}{S \, \Delta \, T_b} \qquad \text{.....(10)}$$

where

H_s = 1.1 kcal/min

S = 0.89 (Mendlowitz, 1948)

ΔT_b = 11.5 (maximum found during last 120 min in series 3)

Blood flow was found to be 107 ml/min. This indicates that blood flow to the tail extended from the body in still air at -40°C is less than that which can be achieved during reactive hyperaemia and can be accounted for entirely by peripheral vasomotor control.

Contribution of extremities to sensible heat loss in cattle.

From measurements carried out at this laboratory using Simpson's rule surface area of extremities was found to comprise 25% of the total surface of oxen. Using Mitchell's (1928) equation $0.09 \, W^{0.67}$, surface area for Claude was calculated to be 5.28 m^2. Area contributed by extremities then equals 1.32 m^2.

One may assess the contribution of the extremities to total heat loss from formula 7 using the data given in Fig. 6.

$$H_t = C_s \, (T_s - T_a) \, t \cdot A$$

when

C_s = 0.0047 kcal/m^2·°C·sec

t = 24 h

A = 1.32 m^2

T_a = -7°C

T_s = +13°C

then

H_t = 10.7 Mcal

Total heat production at this temperature would, depending on food intake and total thermal insulation be of the order of 2.5 Mcal/m^2·24 h or 13.2 Mcal/24 h. The estimate of 10.7 Mcal/24 h heat loss from the extremities is clearly much too high. This may be, in part, attributable to the fact that the extremities do not loose heat as a series of independent cylinders, but in fact, radiate heat to one another. The effective surface for radiant and convective heat loss will be considerably less than total surface area. It is, however, probable that the estimate of C_s is also too high. This raises serious doubts as to the absolute accuracy of calculations based on C_s. It does not however affect the estimates of relative heat loss and blood flow at different air temperatures.

Results

Series 2(a)

Examples of blood pressure (BP) and heart rate (HR) recorded during a typical trial appear in Fig. 9. Three one half hour periods are referred to as pre-noradrenaline (pre NA), 'during NA' and 'post NA'.

Blood pressure increased markedly on noradrenaline infusion. The tendency for BP to decline from the initial pressor level appears as expected. The systolic/diastolic interval increased from 110/80 to 150/90. This clearly reflects the direct effect of NA on the force of contraction of the myocardium. HR initially decreased on infusing NA but after approximately 10 minutes increased steadily (Fig. 9).

BP did not appear to change significantly during any one, or between six, blank saline infusion trials for sheep A or B (Fig. 10). HR was very variable during these initial trials which probably indicated that the animals were relatively unaccustomed to the experimental procedures at this time (Fig. 11).

The 14 trials with sheep B, run before, during and after the period of prolonged cold exposure are shown in Fig. 12. It appeared that BP increased about 10 mm Hg during the first 2 weeks exposure to cold. The increase in BP during NA infusion was rather less. HR in the 'pre NA' and 'post NA' periods, though much more variable, appeared to show a similar trend (Fig. 13).

In sheep B the BP difference between the first 10 and last 10 minutes of NA infusion tended to increase as the experimental series

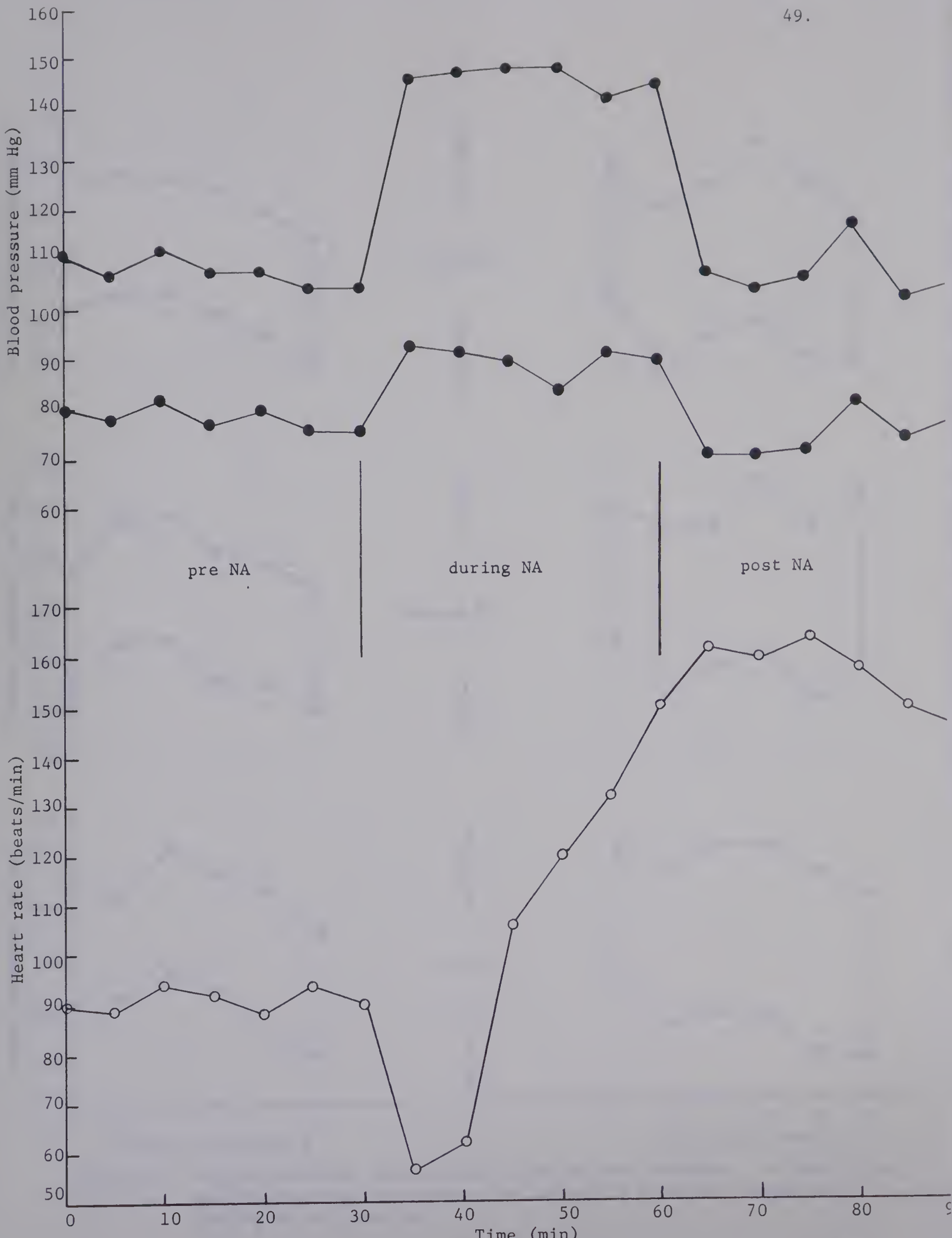

Figure 9. Example of blood pressure and heart rate (sheep B, series 2(a) #3).

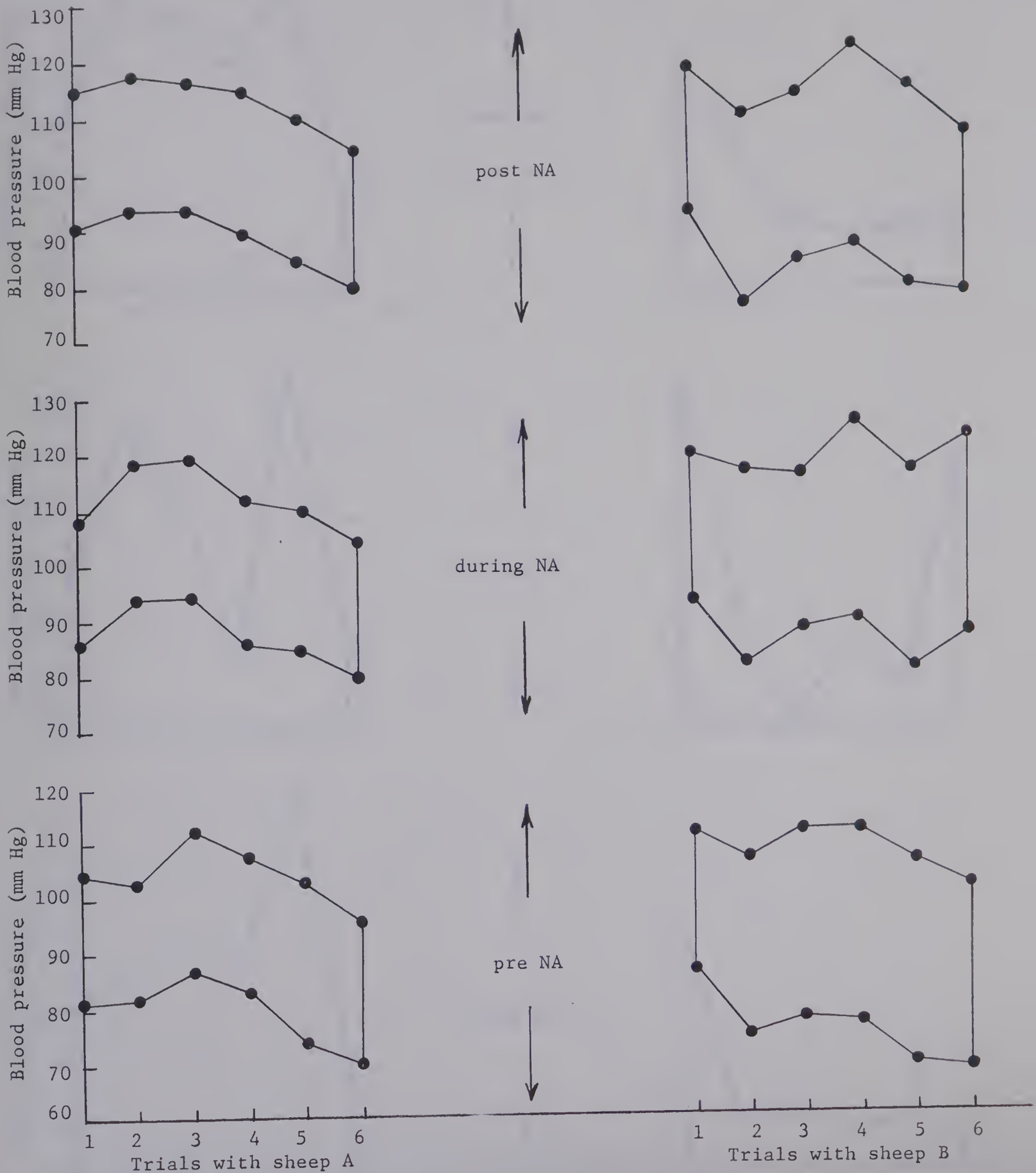

Figure 10. Blood pressures taken during blank saline infusions. In each trial BP was taken as an average over intervals described as pre NA, during NA and post NA.

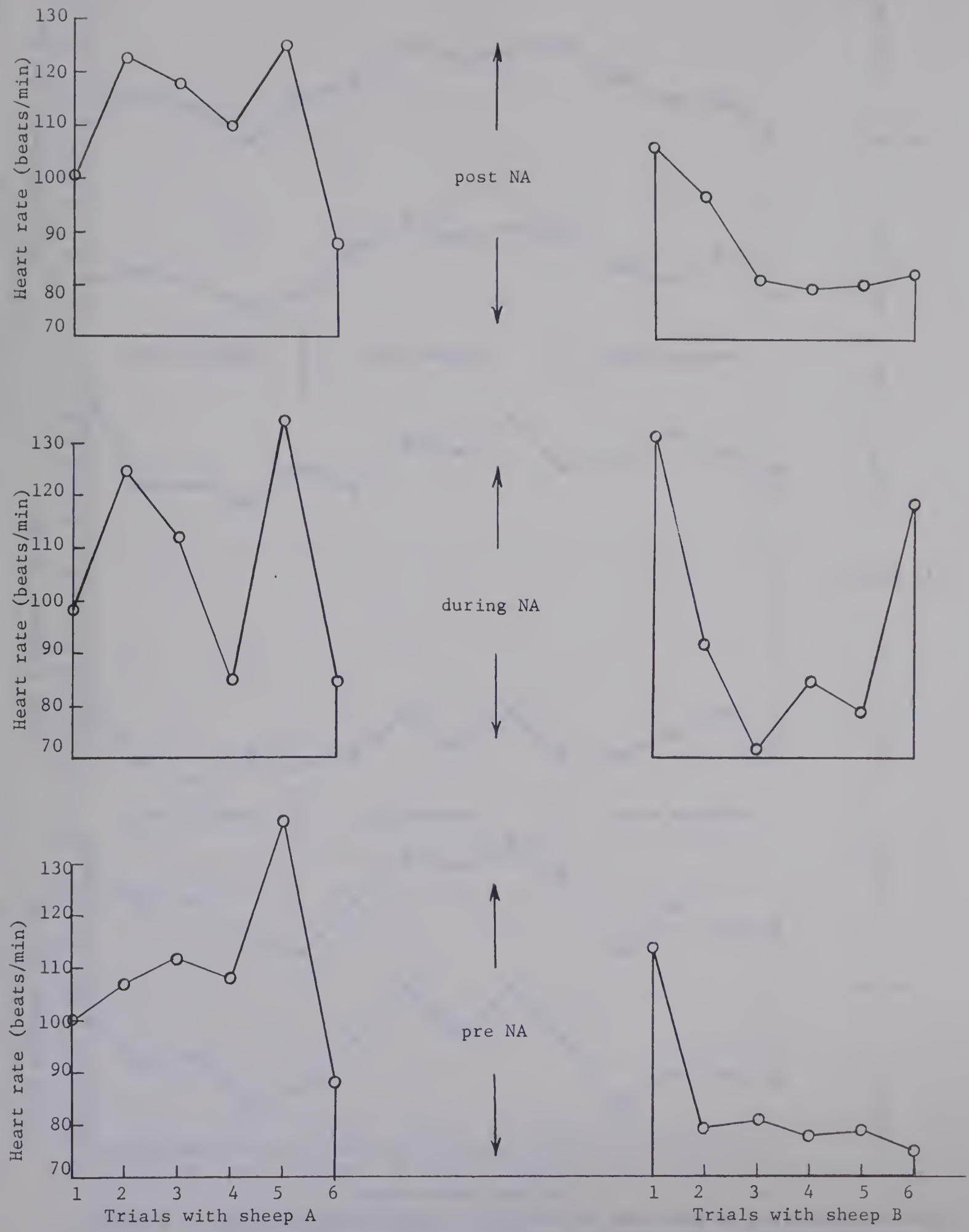

Figure 11. Heart rates taken during blank saline infusions. In each trial HR was taken as an average over intervals described as pre NA, during NA and post NA.

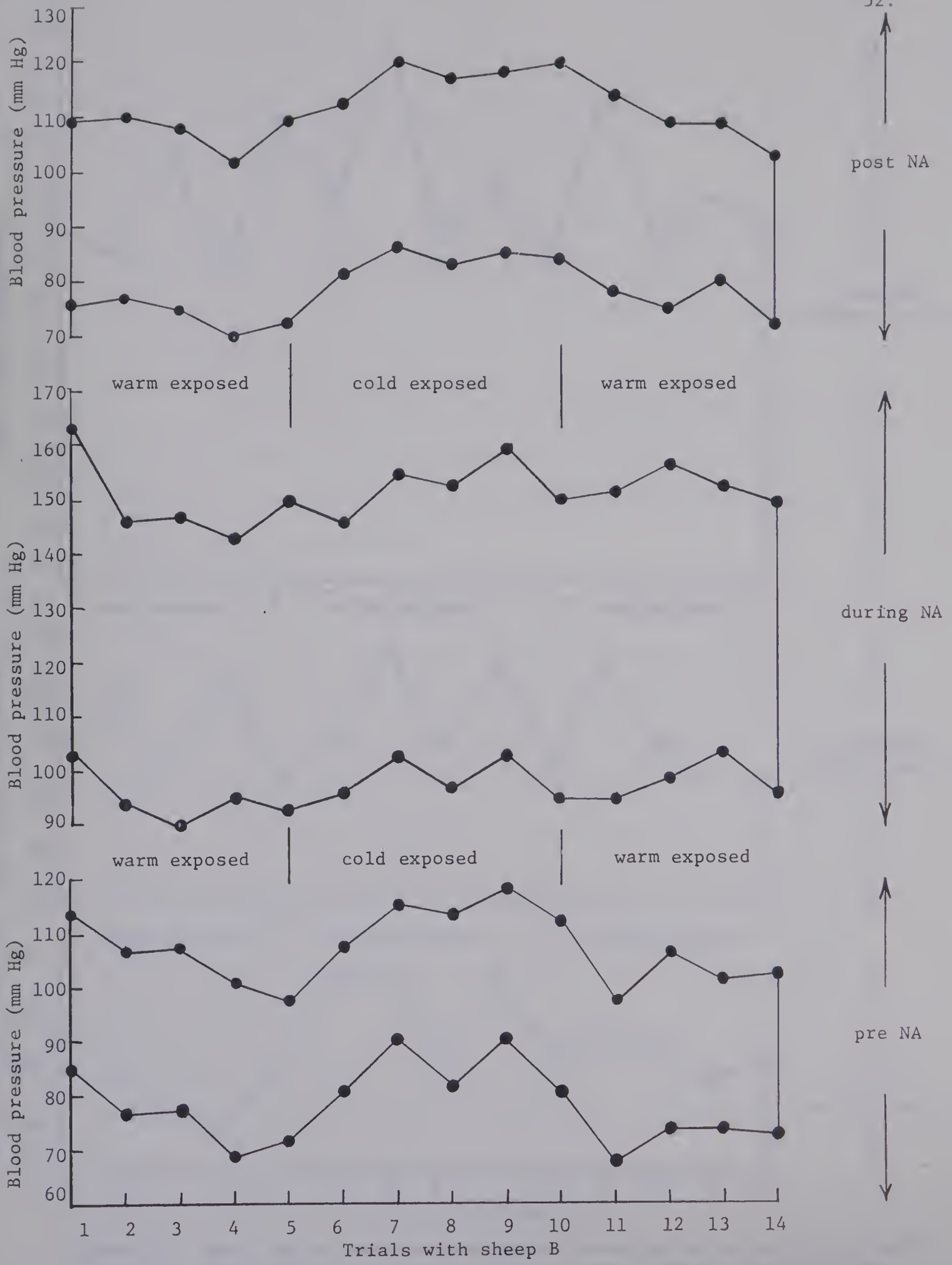

Figure 12. Blood pressure taken during trials involving NA infusion. In each trial BP was taken as an average over intervals described as pre NA, during NA and post NA.

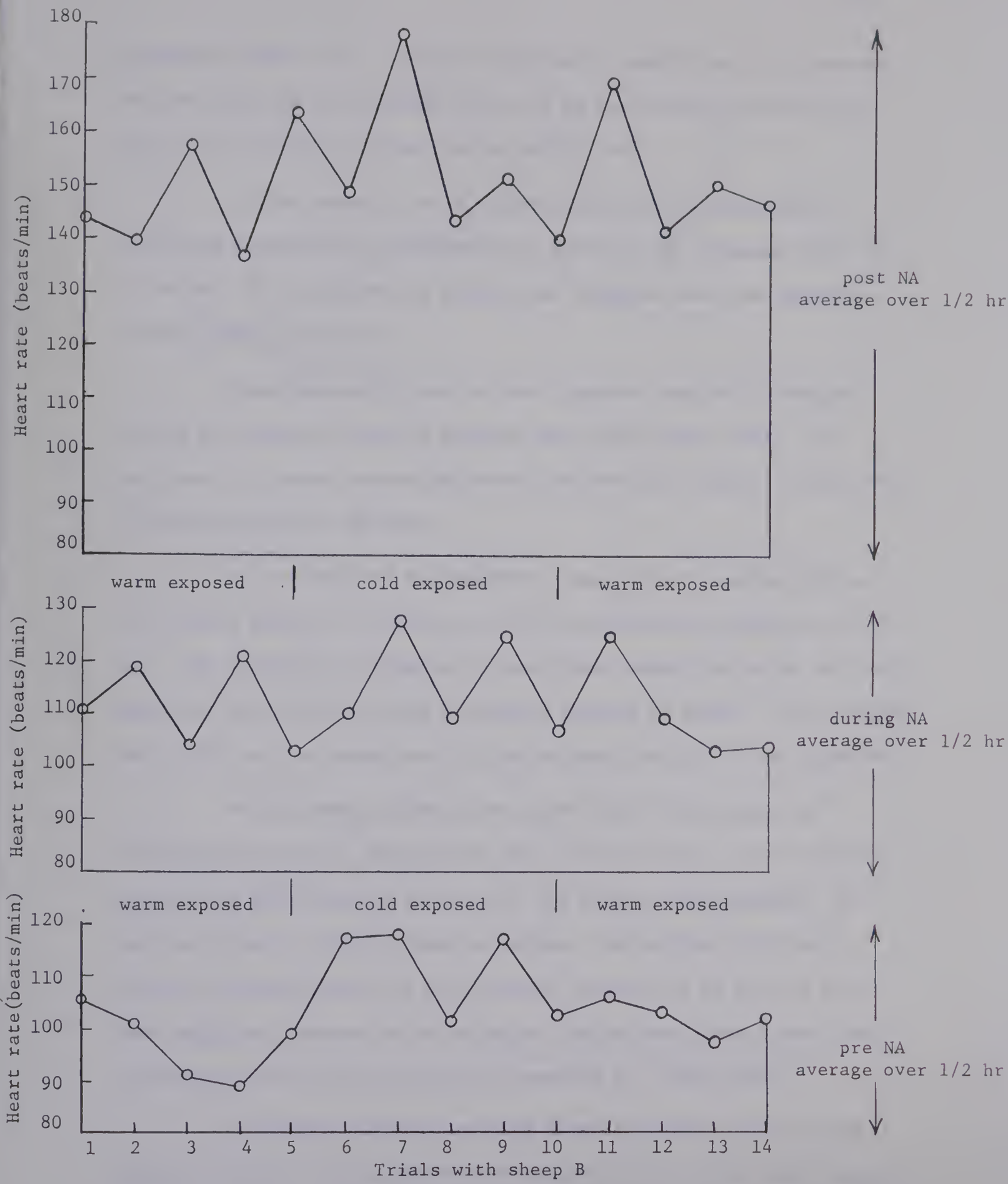

Figure 13. Heart rates taken during trials involving NA infusion. In each trial HR was taken as an average over intervals described as pre NA, during NA and post NA.

progressed (Fig. 14). Perhaps a progressive adaptation to NA infusion occured such that the pressor effect of NA was reduced in the latter part of the infusion but not in the initial part.

In both sheep A and B arterial-venous pO_2 differences decreased progressively throughout the period of NA infusion (Fig. 15). A recovery in pO_2 difference towards the original state was apparent after stopping infusion.

Since metabolic rate in sheep remains reasonably constant during NA infusion (Webster, Heitman, Hays and Olynyk, 1969), the decrease in arterial-venous pO_2 would indicate that cardiac output (CO) increases during NA infusion.

On starting the NA infusions a small 'hunt' lasting only a few minutes appeared frequently in the ears and less frequently in the leg. The incidence and degree of these hunts occurring in the left ear, right ear and left front leg of sheep B appears in Table 7. On stopping NA a brief fall in temperature of the extremities quite often appeared.

During these trials it was noted that if the sheep was disturbed by entry of the operator into the cold room, this frequently resulted in hunts similar to those at the start of NA infusion. BP was also noted to rise on these occasions. Hot saline (5-10 mls) injected intraarterially on one occasion resulted in BP rise of the same magnitude obtained on NA infusion. At the same time a small hunt occurred in each of the extremities measured for temperature.

Arrhythmia occurred in sheep B on NA infusion in both warm exposed periods. The incidence and duration of the missed beats appear in Table 7.

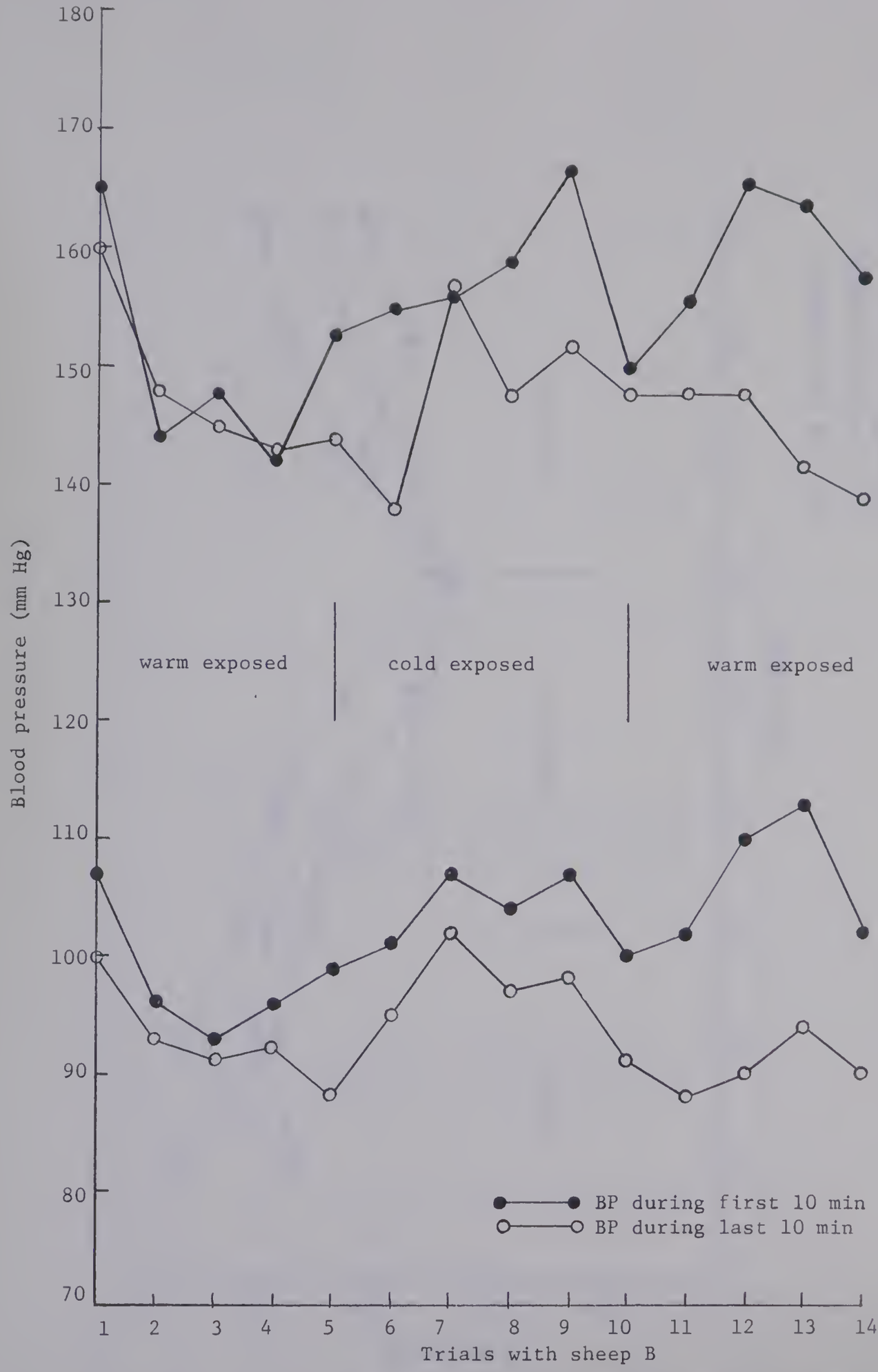

Figure 14. Blood pressure taken as an average over the first 10 and last 10 minutes of NA infusion and expressed as systole/diastole.

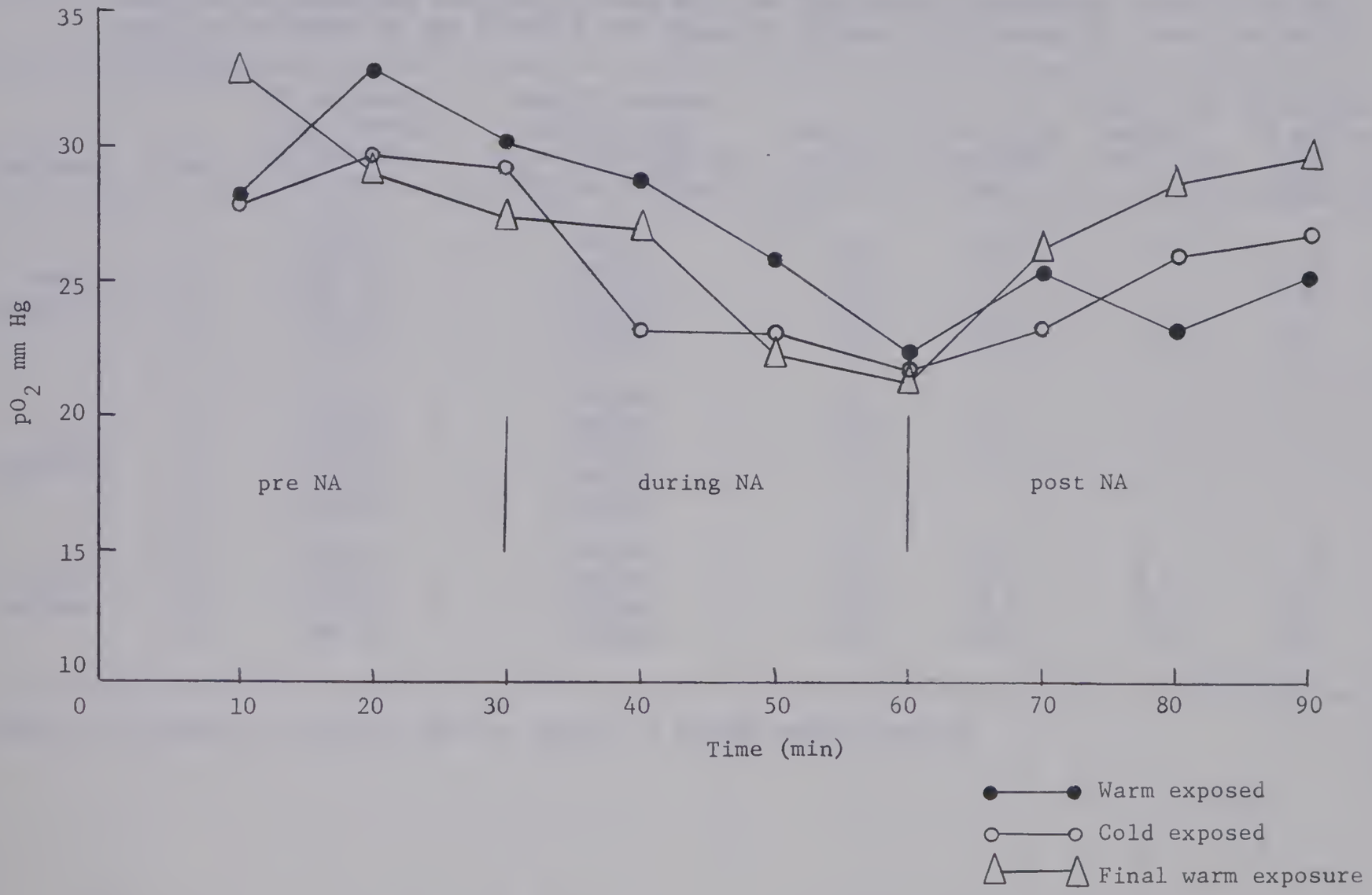

Figure 15. Arterial-venous pO_2 differences averaged at 10 minute intervals for the individual exposure periods during series 2(a) with sheep B. Three readings were taken prior to NA infusion, three during and three after.

Table 7. Incidence and degree of hunts which result from the large BP increase occurring at the start of NA infusions are listed along with the duration of arrhythmia (when it occurs) and the incidence of the missed beats (sheep B, series 2(a)) during the 30-min period of infusion.

Treatment	Trial	BP at point 5 min previous to infusion (mm Hg)	Max BP reached within 2 min after starting infusion (mm Hg)	Hunt (°C) in left ear	Hunt (°C) in right ear	Hunt (°C) in left fore leg	Duration of arrhythmia (min)	Incidence of missed beat
warm exposed	1	112/84	168/114	3.0	0.8	-	7	every 5
	2	100/72	160/114	2.8	4.5	-	2	"
	3	105/76	150/106	2.0	-	-	27	"
	4	99/67	142/107	2.3	2.3	-	20	"
	5	97/73	150/104	2.3	1.5	-	11	"
cold exposed	6	110/84	149/103	1.0	-	-	-	-
	7	118/93	156/114	1.0	1.0	-	-	-
	8	113/80	155/115	0.3	0.3	-	-	-
	9	118/90	168/118	-	-	-	-	-
	10	116/82	150/105	-	-	-	-	-
warm exposed	11	96/66	156/108	1.5	1.5	-	5	every 30
	12	104/71	170/119	4.5	1.5	2.0	6	every 60
	13	102/74	183/128	3.3	3.0	1.3	20	every 5
	14	98/70	177/120	4.5	2.5	1.0	15	every 5

Dashes in columns 4-7 indicate that no "hunts" or missed beats occurred.

Series 2(b)

Throughout this series the Lincoln ewe (B) showed very little hunting in either degree or incidence in the ear in comparison to the Suffolk wether (C). This difference in the two breeds has shown up regularly in other experiments at this laboratory.

A general feature of cooling (especially in sheep C) was the appearance of hunting first in the ears and later in the legs. By the time hunting started in the legs, however, the ears showed a decrease in both incidence and degree of hunting, sometimes stopping altogether. As a general rule the lower the temperature to which the skin dropped before each hunt, the greater the sharp increase in temperature during the hunt.

In these experiments NA often stopped or at least reduced the incidence of hunting (Fig. 16).

Temperature of the sympathectomized ear in both sheep B and C remained at a high temperature (approximately 32°C) when they were subjected to the first two cooling trials (no NA). In the third trial with each sheep the sympathectomized ear remained at approximately 32°C until NA was infused at which time sheep B showed an initial fast drop in this ear of approximately 4°C then slowly cooled for approximately 40 min after which a very regular hunting set in for the duration of the NA infusion (Fig. 17). At no time did the sympathectomized ear reach the low temperature maintained by the normal innervated extremities. On stopping the infusion the sympathectomized ear temperature immediately rose to the pre infusion level and remained there for the duration of the experiment. There had been no return of sympathetic tone after six

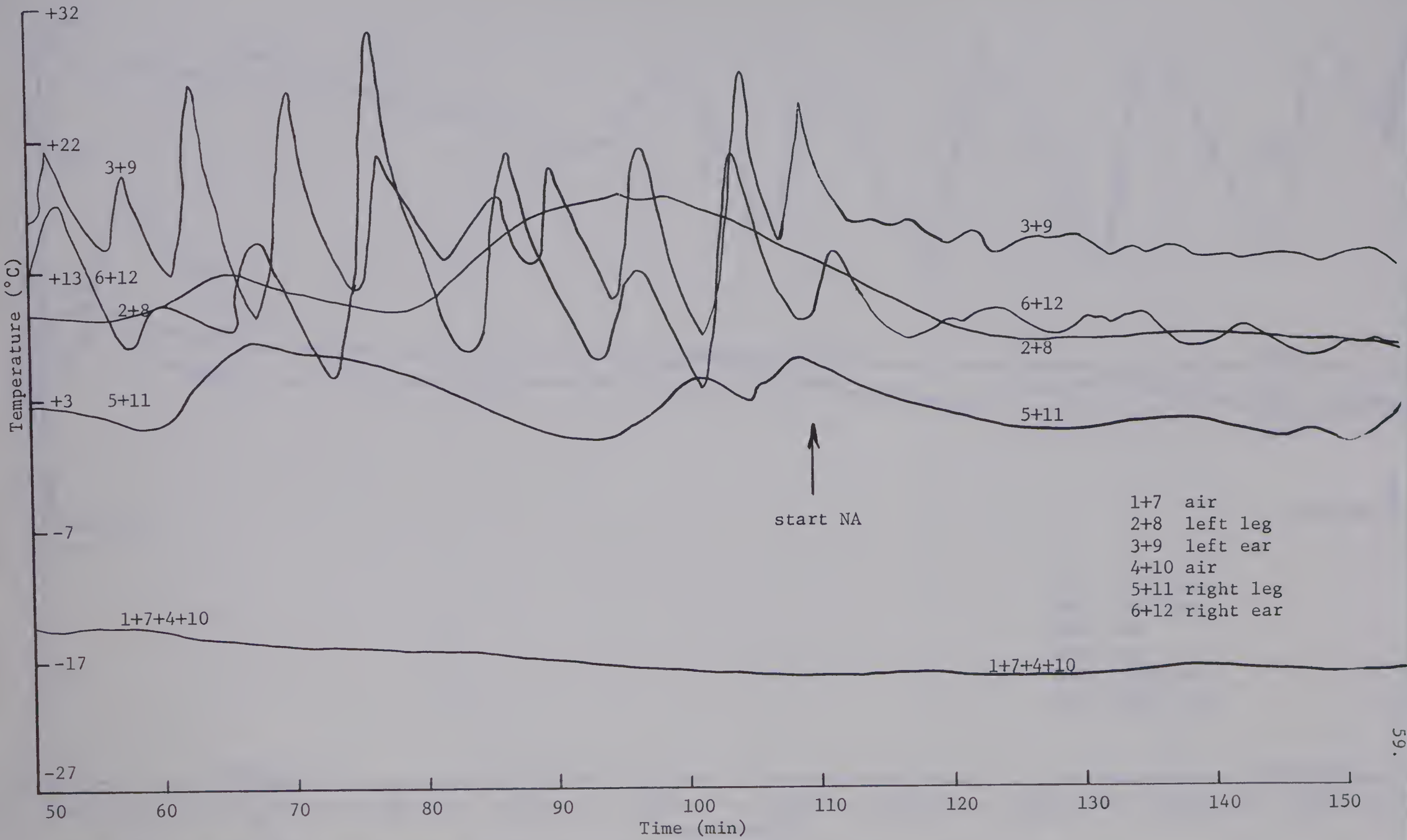

Figure 16. Depiction of inhibition imposed by NA on hunting. Sheep C prior to sympathectomy.

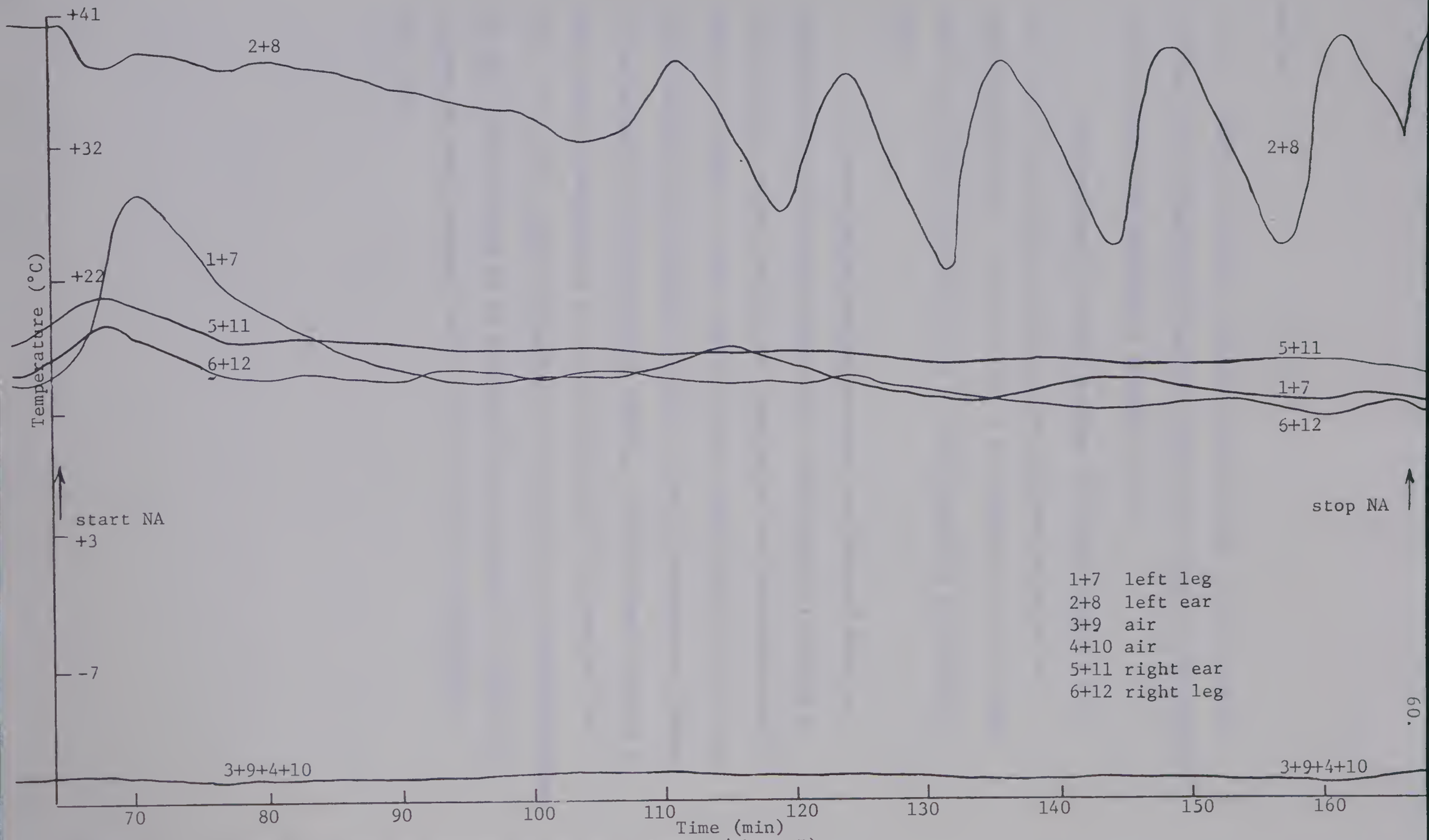

Figure 17. Hunting in sympathectomized ear on NA infusion (sheep B).

months.

Sheep C followed a similar pattern except on starting the NA infusion the sympathectomized ear cooled very rapidly (approximately 20°C in 4 min) then immediately started to hunt.

Pitressin even at large dosages (3 PU/ kg /min for one half hour) was not able to duplicate the response to NA in the sympathectomized ear. At the outset of the infusion the ear cooled slowly by 3°C and then warmed over a 10 min period to pre infusion level.

As a much bigger hunting response was obtained from the sympathectomized ear during series 2(b) a simultaneous recording of blood pressure, heart rate and ear temperature was taken from sheep B during NA infusion. During this trial (and series 2(a)) it was noted that increased blood pressure, whether a natural occurrence or induced by the operator, at times preceded hunting. It was however typical at these times to see hunting occur simultaneously in all extremities (Fig. 17). Of 10 'hunts' averaging 5.3°C in rise in the sympathectomized ear only 1 was preceded by an increase in blood pressure. Again this increase in blood pressure caused a hunt in both fore shanks and both ears.

Discussion

Series 2

The breed difference that showed up in hunting in the ears may be due to the great variation in the size of the ears. The Lincoln had short ears partially protected with a wool outcropping from the head. The Suffolk had much larger ears with no protection other than a light hair covering.

The difference in character of hunting between legs and ears (Webster and Blaxter, 1966) can be explained simply by the difference in mass and subsequent difference in rate of cooling. The legs on hunting took a relatively longer time to cool than the ears, thus taking a longer time to reach the 'trigger' temperature required to start the hunting response. Hunting in the leg thus appeared to be of the same nature as in the ear with the only difference being a much longer period required for cooling off the leg after the peak in a hunt had been reached.

The increased BP and HR seen before and after NA infusion in the sheep during prolonged cold exposure is possibly a feature of cold acclimation. The increase in metabolic rate during prolonged cold exposure would likely be associated with an increase in CO. If peripheral resistance remained the same BP would increase. The present results can no more than suggest this as an explanation.

Although hunting may result from an increase in systemic blood pressure, the results clearly illustrate that increased BP is not essential for this phenomenon.

NA infusion causes an increased cardiac output (CO) which persists over a one-half hour period in the sheep, a result contrary to that assumed by Gillespie and Muir (1967) to occur in the rat. This conclusion is based on two pieces of evidence. The A/V ΔpO_2 decreased during continuous NA infusion. Moreover systolic blood pressure increased by 40 mm Hg on NA infusion and diastolic BP increased only 10 mm Hg which strongly suggests that one effect of NA was to increase the force of contraction of the myocardium.

Heart rate usually decreased at the beginning of NA infusion and then increased progressively. The initial decrease can be attributed to the cardioinhibitory reflex consequent upon the initial sharp rise in BP. The subsequent increase in HR was usually associated with a steady fall in BP and may be attributed to a progressive decrease in cardioinhibitory tone from the baroreceptors allowing the NA to exert a direct accelerator effect on the heart.

The sudden increase in HR which occurred immediately after stopping NA infusion can be attributed entirely to a rate sensitive response transduced by the baroreceptors.

The vasoconstriction occurring in the normally innervated ear of a sheep exposed to cold can be attributed to the effect of noradrenaline. CIVD seems dependent on a change in the balance between vasoconstriction as caused by NA and an opposite vasodilatory force. CIVD may then be due: (1) to an increase in vasodilatory substance or substances, (2) to liberation of a noradrenaline inhibitor, (3) to a shut off of NA, (4) to a decreased sensitivity to NA, or any combination of (1), (2), (3) and (4).

During NA infusion the initial synchronous hunt in the normal innervated extremity was due to the increase in BP. Thereafter asynchronous hunts decreased in intensity and frequency. Noradrenaline may then have inhibited hunting by weighing the balance against the effects of the vasodilatory substance or by preventing a total shut off of NA.

In the sympathectomized ear there was no vasoconstriction prior to NA infusion. This confirms that cold induced vasoconstriction in the normal ear is due entirely to the sympathetic transmitter noradrenaline.

Noradrenaline infusion caused the sympathectomized ear to constrict thus provoking a larger than usual hunt. This hunt may have been due to a vasodilatory substance (1) or to a NA inhibitor (2). It cannot have been due to a shut off of NA (3) or a decreased sensitivity to NA (4).

These results taken together would suggest that at a particular ear temperature, or at a set time interval after a previous hunt the accumulation of vasodilator substance became sufficient to overwhelm the vasoconstrictor effect of NA to a point where arteriolar resistance dropped sufficiently to permit the entry of blood to the capillary bed and especially the A/V anastomoses. In the sympathectomized ear during NA infusion the effects of NA would be equally distributed, the A/V anastomoses would open more or less simultaneously and a large 'hunt' would ensue. In the normal ear continued NA induced vasoconstrictor tone would only allow small numbers of capillaries or A/V anastomoses to open at any one time. This suggests that the

vasodilatory force exerts a general effect on the ear such as could be expected from a buildup of vasodilatory metabolites. The vasoconstrictor effectors are likely firing in relays giving different intensities of vasoconstriction to different arterioles and thus providing the fine control of CIVD which does not show up as hunting and may be called continuous proportional control.

GENERAL SUMMARY

Experiments were carried out to investigate the nature and extent of control of heat loss from extremities of ruminants. Experiments with cattle were designed to investigate the heat loss from extremities and the associated requirements for, and control of blood flow. With sheep, experiments were designed for a concentrated study of the mechanisms involved in CIVD.

1(a). Results with cattle indicate that minimum skin temperature is reached in the tail at about -7°C. Calculated values four blood flow did not reveal any increase in flow attributable to CIVD at this temperature, but skin temperature measurements made at air temperatures down to -18°C suggested that prolonged exposure of the tail to temperatures considerably below -7°C would induce an increase in blood flow.

1(b). Measurements for maximum blood flow associated with reactive hyperaemia indicated a much greater flow through the skin of the tail than suggested from literature values. However the estimates made in these experiments for heat loss and consequently blood flow are questionable and probably too high.

1(c). Estimated blood flow to the tail at -40°C was much less than that demonstrated during reactive hyperaemia. This indicates that local regulation of blood flow to the extremities in cattle is sufficient to maintain the integrity of the tissues at temperatures as cold as those likely to be experienced in any existing areas of cattle production.

2(a). Results from sheep experiments show that NA infusion had a specific stimulating action on the heart by increasing force of contraction of the myocardium and thereby cardiact output. The pressor response

to NA infusion in the sheep decreased from its initial peak throughout the period of infusion. The increase in HR during NA infusion may be due to a progressive decrease in cardioinhibitory tone from the baroreceptors thus allowing NA to exert a direct accelerator effect on the heart.

2(b). NA infusion depressed the hunting response in the normal ear although in the sympathectomized ear its cooling effect allowing hunting to occur.

2(c). It was concluded that CIVD is due to a change in the balance between vasoconstriction caused by the sympathetic transmitter substance NA, and vasodilation caused by a build up of vasodilatory substances.

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Appendix 1

Total heat loss (H_t), change in heat content (H_x) and estimated blood flow (F) of the tail (Series 1(c)).

	1			2			3			4		
Interval min from start	H_t kcal/ min	H_x kcal/ min	F ml/ min	H_t kcal/ min	H_x kcal/ min	F ml/ min	H_t kcal/ min	H_x kcal/ min	F ml/ min	H_t kcal/ min	H_x kcal/ min	F ml/ min
0-20	5.74	3.00	21	6.72	2.70	43	6.64	2.16	55	7.05	2.54	40
20-40	8.69	1.00	46	10.09	0.62	81	9.55	0.85	89	10.25	0.54	68
40-60	9.80	1.23	49	11.23	0.46	86	11.07	0.92	95	11.23	0.46	73
60-80	9.68	+0.15	54	10.74	1.23	73	11.19	+0.54	120	10.78	0.39	63
80-100	11.40	0.39	55	11.77	1.39	70	11.52	3.47	64	12.10	1.46	70
100-120	11.60	0.31	53	11.60	1.46	66	11.15	+0.54	84	12.63	+0.39	75
120-140	13.20	0.77	56	12.34	2.54	59	13.57	+1.08	90	13.78	2.16	61
140-160	13.08	0.08	60	11.69	0.77	65	13.24	4.93	48	12.63	3.00	48
160-180	13.33	0.92	56	11.23	1.16	59	10.70	3.62	38	10.95	3.00	59
180-200	12.38	1.85	48	10.95	1.08	56	10.05	+0.39	57	10.66	1.23	45
200-220	11.19	2.46	40	9.92	1.46	48	9.72	2.77	36	9.68	+0.31	47
220-240	10.66	0.46	48	9.31	2.46	44	8.94	+0.77	50	9.31	1.69	34