A COMPARISON OF THERMOREGULATION IN A NUMBER OF EAST AFRICAN ANIMALS

by

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DECLARATION

I hereby declare that this Thesis has been composed by myself and has not been accepted in any previous application for a Degree. The work of which this is a record has been done by myself and all sources of information have been specifically acknowledged by means of references.

CONTENTS

SUMMARY

Three primate species, the greater galago (*Galago crassicaudatus*) the baboon (*Papio cynocephalus*) and the chimpanzee (*Pan satyrus*) were exposed to environmental temperatures of up to 45°C in a climatic chamber, and their thermoregulatory responses were measured. In the field the thermoregulatory responses to the changes in solar radiation load through the day were measured in the elephant (*Loxodonta africana*), the black rhinoceros (*Diceros bicornis*), the white rhinoceros (*Ceralotherium simum*) and the zebra (*Equus burchelli*).

It was necessary to tranquillise the baboons and the chimpanzees with phencyclidine hydrochloride in order to measure cutaneous moisture loss (C.M.L.) and rectal temperature. Initial experiments were, therefore, carried out to ensure that the tranquilliser was not:

1. having any significant effect on the ability of these animals to thermoregulate.
2. inhibiting their response to sympathetic and parasympathetic drug administration.

The tranquilliser was found to have a minimal effect in this respect.

The baboon and the chimpanzee increased both their C.M.L. (i.e. sweated) and respiration rate (i.e. panted) on heat exposure. This is in contrast to the galago which was found to use panting as its

only method of evaporative heat loss. All the animals were able to maintain a fairly stable rectal temperature on exposure to 40°C.

The administration of adrenaline, noradrenaline, acetylcholine, and atropine, both intradermally and intra-venously, showed that the sweat glands in the baboon and the chimpanzee were under cholinergic control. In the galago no sweat gland activity could be initiated by these drugs or by local or generalised heating of the animal.

A skin sample was taken from each animal and the sweat gland structure described.

The measurements of C.M.L. from the rhinoceros and the elephant produced the following results.

1. An increase in insensible C.M.L. between two sets of experiments twelve months apart. It is suggested that this is associated with the state of hydration or, possibly, with the age difference of the animals.

2. A very high C.M.L. at 07.00 hrs. This was significantly increased at mid-day in the rhinoceros, i.e. it was sweating; but showed no significant change during the day in the elephant.

The rhinoceros also showed an increase in respiration rate and, therefore, appears to thermoregulate by both sweating and panting. Rectal temperature through the day in this animal varied by 1.0°C (c.f. Bligh and Harthorn 1963), while the

diurnal air temperature variation was between 19°C and 31°C.

It was found, however, that the elephant neither sweats nor pants. It is suggested that it was able to maintain a relatively stable body temperature in the present experiments (maximum diurnal rectal temperature variation, 2.0°C, diurnal air temperature variation 12.0°C) by the following means:

1. non-evaporative heat loss (convection, conduction, radiation).
2. a high "insensible" moisture loss through the skin.
3. by means of its ears to lose heat, more especially in the cooler part of the day.
4. by its behavioural pattern (e.g. shade seeking, bathing, and spraying with exogenous water).

The zebra was found to have an increase in C.M.L. during the day, like other equines, and also an increase in respiration rate. Rectal temperature increase in these animals varied between 0.6°C and 2.9°C as air temperature increased from 20°C to 31°C.

The results obtained are then discussed on a comparative basis.

ACKNOWLEDGEMENTS

This work has only been possible as the result of the patience and co-operation of many people, to all of whom I am sincerely grateful. In attempting to mention them individually I would be more than likely to omit somebody who, even in some small way, has helped me considerably.

Therefore, I hope it will suffice to mention the Kenya National Parks who kindly allowed the use of the elephant, the rhinoceros and the chimpanzees. More specifically, Messrs. David Sheldrick, Peter Jenkins and Sammy Ngethe, the Kenya National Park Wardens who actually had to put up with me and my attempts to collect the reported data. Also the Medical Research Council, Nairobi, for their generosity in making the baboons available for use.

Throughout the progress of this thesis it has been watched, criticised and helped in the right direction by Professor David Robertshaw. To him I must offer my special thanks for the patience and understanding he has shown in offering his advice. Also Doc. Alan Walker who has shown a keen interest in this work and has been ever ready to discuss any points that have arisen from it. I hope that the assembly of this work, the solutions it has tried to offer and the new questions it poses is as satisfying to them as it is to me.

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INTRODUCTION

Animals indigenous to East Africa are potentially exposed to a high air temperature and a high level of solar radiation. The behaviour of the animal can, however, modify this considerably. High levels of solar radiation can be avoided by seeking the shade and high air temperatures by burrowing under the ground. Consequently, the environment that an animal experiences is often less stressful than might be expected from the overall ambient conditions, Weiss and Lantios (1961), Cabanac (1972). Even so, to maintain a stable body temperature (homeothermy) any animal must balance the heat inputs to the body (from the environment and metabolic heat load) with its heat output to the environment.

As long as air temperature is less than body temperature there can be a loss of heat to the environment by convection. Radiant heat loss will depend on the radiant temperature difference of the animal and its surroundings. Conductive heat transfer only occurs at points of physical contact between the animal and its environment; unless the animal is lying down, the area of contact and thus heat loss, is minimal. As the air temperature increases the amount of heat lost by convection decreases and when air temperature is higher than body temperature, the flow of heat is reversed and the animal gains heat from the environment. There comes a critical

air temperature, therefore, at which the animal is unable to dissipate its metabolic heat production by non-evaporative means. At this point, the metabolic heat must be lost by evaporation of water to prevent a rise in body temperature. As air temperatures in East Africa frequently approach and exceed this critical value the indigenous mammals are very much dependent on evaporative water loss in order to maintain a stable body temperature.

This evaporative heat loss can occur from the upper respiratory tract or from the general body surface. The rate of heat loss from the former is proportional to the ventilation rate and the vapour pressure difference between the surface of the respiratory tract and the atmosphere (Richards 1970). Although the rate of heat loss could be increased by raising the temperature of the surface, and thus increasing the vapour pressure gradient, the more effective way is to increase it by raising the ventilation rate. This can be variously classified as 'panting', 'thermal polypnoea', or 'thermal tachypnoea'. However, the efficiency of panting as a means of dissipating heat must be limited by the increased heat production of the respiratory muscles. It has been suggested that this efficiency decreases as body size increases so that many animals whose adult body weight is greater than 100kg have to rely on an increased evaporative heat loss from the skin.

This supplements panting and is necessary for the maintenance of a relatively stable body temperature on heat exposure (Robertshaw 1971).

The loss of evaporative heat from the skin is proportional to the vapour pressure gradient between the skin and the environment. This can be increased by raising skin temperature through alterations in cutaneous vaso-motor tone. It is not, however, limited by the area available for evaporation or by any efficiency considerations, as is panting.

A degree of thermoregulation is achieved in many reptiles by behavioural and non evaporative means (Richards 1970, Dawson 1972). The evaporative heat loss mechanisms which have developed to supplement this can be found in two classes of animals, the birds and the mammals. It is likely that these physiological temperature regulating mechanisms have evolved independantly in these classes since their radiation from a common, non-homeothermic ancestor, some 2-300 million years ago - an example of convergent evolution. Within the mammals it appears that thermoregulatory sweating has also undergone convergent evolution as it is found in the larger terrestial mammals (especially those indigenous to tropical climates) and in simian primates (Robertshaw 1972). In the other mammalian species panting is still the only evaporative heat loss mechanism used in thermoregulation - unless saliva spreading is considered

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significant (Dawson, 1972).

In the present study a number of mammals indigenous to East Africa have been investigated in an attempt to fit them into this broad general picture of thermoregulation which is emerging as a result of current research. Four of the species which were used are large terrestrial mammals, the white rhinoceros (Ceralotherium simum), black rhinoceros (Diceros bicornis), the elephant (Loxodonta africana), and the zebra (Equus burchelli). Three species of primates were also used, the galago (Galago crassicaudatus), the baboon (Papio cynocephalus), and the chimpansee (Pan satyrus).

It has been found that elephants have no sweat glands (Young 1972) and thus they cannot utilise sweating as a heat loss mechanism. Yet they are able to maintain a relatively stable body temperature (Young 1972) (Buss and Walker 1965). It is not known, however, what thermoregulatory mechanisms this animal uses to achieve this.

The rhinoceros is known to have adrenergic sweat glands (Robertshaw 1972). However, it has been reported by Allbrook, Harthorn, Luck and Wright (1958) that these are non-functional. They also reported a diurnal temperature variation of 3.0°C, but no other work has been done to substantiate this or to investigate the ways in which the rhinoceros loses heat.

A number of workers, Schmidt-Nielsen, Schmidt-

Nielsen, Jarnum and Houpt (1957); Allen and Bligh (1969); Robertshaw and Taylor (1969); Bullard, Dill and Yousef (1971); Maloiy (1972) have investigated thermoregulation in the genus Equus. These animals (the donkey and the horse) have been found to sweat on heat exposure; the reports on panting, however, are not so clear. Maloiy (1972) recorded panting on heat exposure of donkeys in a climatic chamber, while Schmidt-Nielsen (1964) reported that they did not pant on heat exposure in the desert. The zebra also belongs to this genus yet no work has been done to see if its thermoregulatory mechanisms are the same. As it is normally exposed to a much more severe thermal environment than most of the other members of the genus, it might be expected to differ, at least in the magnitude of these heat mechanisms.

It is known that the prosimian species Nycticebus coucang and Perodicticus potto pant on heat exposure, even though they have sweat glands in their skin (Aoki 1962). The galago, like all prosimians, also has sweat glands (Montagna and Yun 1962), (Montagna and Ellis 1963). It was, therefore, intended to see if these have any thermoregulatory significance. It is known that man never pants on heat exposure (Robertshaw 1972) so that active evaporative heat loss occurs entirely from the skin. It has not been ascertained, however, at what stage

in primate evolution panting, which is so important in prosimians, ceased to be used as an evaporative heat loss mechanism.

In trying to answer this question, it was hoped that it might become a little clearer if thermoregulation in existing primate species could be investigated. Previous research in this field has been rather limited and the results obtained have produced a rather confused picture.

A number of workers have investigated thermoregulation in rhesus monkeys. Although Whitford (1972) reports that they are able to tolerate 44°C with "no apparent disruption of their temperature regulating mechanisms", Folk (1966), quoted by Newman (1970), found that with their arms extended at an air temperature of 38°C there was an "explosive" rise in rectal temperature. With arms along the side of the body, however, there was only a slight rise in rectal temperature. Folk reported that there was no visual evidence of sweating, but made no mention of changes in respiration rate. It is quite possible, therefore, that there was an increase in C.M.L., if this was evaporating immediately then no visual moisture would have been apparent. The one rhesus monkey that Robinson and Morris (1957) investigated maintained a fairly stable body temperature on exposure to 40°C. As there was no increase in respiration rate they

"inferred" that the stability in body temperature must have been due to an increased C.M.L. Newman (1970) also mentions the work of Hardy (1955) on cebus monkeys. He found a doubling in evaporative water loss but did not partition this into its cutaneous and respiratory components.

Recently, two other sets of workers, Stitt and Hardy (1970) and Nakayama, Hari, Nagasaka and Tokura (1971) have investigated thermoregulation in the squirrel monkey and the japanese monkey respectively. Both found an increase in cutaneous and respiratory moisture loss, though the latter was rather small and of limited thermoregulatory benefit.

Robertshaw (personal communication) reports that stump-tailed macaque monkeys show an increase in both C.M.L. and respiration rate on heat exposure. However, the increase in respiratory water loss is negligible.

Previous work on the two simian species used in the present study, the baboon and the chimpansee is also rather limited. Funkhouser, Higgins, Adams and Snow (1967) report that baboons both sweat and pant on exposure to an air temperature of 45°C, but this was insufficient to prevent a rise in mean rectal temperature from 38.2°C to 40.6°C in their experiments. Although Newman, Cummings, Miller and Wright (1970) also found an increase in C.M.L. on exposure of baboons to 43°C, they found no increase

in respiration rate or rectal temperature. Similarly, Slonim (reported by Funkhouser et al 1967) found no increase in rectal temperature in baboons exposed to 40°C. It is not known, however, whether all these reported differences in the thermoregulatory responses of these animals can entirely be explained by the environmental temperature differences.

In the chimpanzee overall evaporative water loss doubles on increasing environmental temperature from 18°C to 38°C (Whitford 1972 and personal communication). In certain specific "sweating areas" C.M.L. increased by ten to twenty times. However, respiration rate was not measured and it is not known if any of the reported increase in evaporative water loss results from an increased respiration rate.

Much work has been carried out on the anatomy of primate sweat glands by Montagna and his associates, at the Oregon Regional Primate Research Centre, Oregon, U.S.A. In some of this work they also briefly investigated the control of the glands they found. The results of this and much other work on sweat gland control is extensively reviewed by Robertshaw (1972). It is well established that the eccrine (atrichial) glands of man are controlled by cholinergic, sympathetic nerves. Montagna and Yun (1962, 1963) found similar well developed, eccrine glands in the baboon and the chimpanzee.

These also respond to cholinergic stimulation in the chimpanzee. However, in the baboon their control mechanism is not known.

The sweat glands over the general body surface of prosimians are classified as apocrine (Montagna and Ellis 1963), and respond more readily to adrenergic than to cholinergic stimulation. In the Lorisidae (which includes the galago) these and the eccrine glands, found on the friction pads, have many characteristics in common (Ellis and Montagna 1963). The apocrine glands in this family are, however, different from the apocrine glands found in man and the higher primates (Montagna 1956). Differences can be identified histochemically (presence of phosphorylase, and of cholinesterase in nerves) anatomically (structure of secretory and myoepithelial cells) and physiologically (mode of action).

The present work, therefore, was carried out to try to clarify some of the points which have arisen from the work reviewed above, viz:

1. How do the elephant and the rhinoceros thermoregulate, bearing in mind that the sweat glands in the rhinoceros are large, and well developed, while the elephant has none?
2. Does the zebra thermoregulate in a manner similar to the other members of the genus Equus?
3. Are the sweat glands in the greater galago of thermoregulatory importance and how are they

controlled?

4. How does the baboon compare to the chimpanzee with respect to its thermoregulatory mechanisms, and are they different to man?

The limitation in all this work has been availability of an insufficient number of animals in each species, and the intractability of those that were used. However, it was hoped that in answering these questions and thus identifying the avenues of evaporative heat loss in these species, it would assist in the understanding of comparative thermoregulation in mammals.

MATERIALS AND METHODS

Date and Location

Work on the three primate species was carried out at the Department of Animal Physiology, University of Nairobi, Kenya, between October 1970 and December 1971. Work on the three ungulate species mentioned was carried out in the field at Tsavo National Park, Kenya, Meru National Park, Kenya, and the field station of the Department of Animal Physiology at Athi River, Kenya. The field work was carried out at various times, depending on weather conditions, between March 1971 and May 1972.

Housing, maintenance and handling of animals in Nairobi

1) Galago (Galago crassicaudatus)

Work carried out in Nairobi involved the use of one sexually mature male and one sexually mature female greater galago, Galago crassicaudatus. These were caught locally in Kiambu District in September 1970, and at the termination of the experiments they were still in good health and were transferred to the Animal Orphanage attached to Nairobi National Park. The animals were housed in a room (4m x 7m x 7m) at the Department of Animal Physiology for the duration of the work. This room contained a small box (30cm x 80cm x 40cm) which was suspended from the wire mesh ceiling of the

room, and the animals were always observed to be sleeping in this during the day. Access to this box was from one of a number of tree branches also placed in the room, all of which were used for climbing by the animals. The room was illuminated by two 40 watt fluorescent lights which were switched on each morning at 8 a.m. and off at 4.30 p.m. by the animal attendant on duty. A 15 watt bulb was left switched on at night to provide sufficient light for the nocturnal activities of these animals.

They were fed fresh fruit, fresh meat and milk to which a proprietary vitamin mixture was added. When first caught the animals were completely wild and after considerable training they would come down from their sleeping box and be fed by hand by the author. Provided a leather glove was worn, they could be handled sufficiently to apply the equipment. Once this was in place the animals did not appear to be worried by it.

2. Baboon (Papio cynocephalus)

Work was carried out on a number of yellow baboons, Papio cynocephalus. These belonged to Medical Research Council unit attached to the Kenyatta National Hospital, Nairobi, where they were normally housed. They had previously been used by research workers on various projects,

but at the time of the experiments, they had not been used for several months and were in a normal, healthy condition. When required, they were placed in a "press back" cage which allowed the animal to be restricted completely while 1mg/1kg of tranquilisor (phencyclidine) was given. They were then transported to the Department of Animal Physiology in a tranquilised state. Normally they were kept at the Department of Animal Physiology for less than a week during which time they were kept carefully locked in the "press back" cages unless tranquilised and removed for experiments. If used for experiment in an untranquilised state the whole cage was moved into the climatic chamber. Four animals were used in this work, two four year old males weighing 18.5 kg and 18.0 kg, a two and a half year old female weighing 9.5 kg, and a four year old female weighing 15.5 kg. They were fed fresh fruit and vegetables, but appeared to require no extra liquid, and remained in good health at all times. They all exhibited a very aggressive behaviour which required considerable care in moving and housing them, and, consequently, handling was impossible without tranquilisation.

3. Chimpanzee (Pan satyrus)

The two chimpanzees (Pan satyrus) used were normally kept at the Animal Orphanage attached to

Nairobi National Park, Langata, Nairobi, where they were on view to the general public. One was a two year old male weighing 13 kg and the other a 5 year old female weighing 35 kg. Both had been household pets and were tame. It was very easy to move the smaller male animal to the Department of Animal Physiology, where he was then housed for brief periods. He was fed fresh fruit, fresh vegetables, bread and milk. It was, however, found to be necessary to tranquilise the animal in order to do some of the experiments as otherwise its "primate curiosity" made it impossible to keep any experimental equipment in place. The larger female was considered too powerful to handle untranquilised and on only one occasion was it used for an experiment. On this occasion, the tranquiliser was given before it was moved from its normal housing at the Animal Orphanage. It was then transported, the experiment completed, and the animal returned before the tranquiliser had time to wear off.

Management of animals in field experiments

a) Tsavo National Park

Field work was carried out in Tsavo National Park, Kenya, in March 1971 and in February and April 1972. The park is situated 200 miles S.E. of Nairobi at an altitude of 360 metres. The

National Park warden at Tsavo has a number of abandoned or injured game animals in his care, and it was possible to use one of the elephants, Loxodonta africana, and a black rhinoceros, Diceros bicornis. These were two and two and a half years old respectively and were completely tame, as they had been hand reared since they were found abandoned in the park some two years previously. They were normally herded in the park during the day together with a number of other similar animals. Readings were taken (a) before they were let out from their overnight enclosure, (b) when they returned to it at mid-day and (c) before being shut in at night. The animals tolerated the equipment used.

b) Meru National Park

Field experiments at Meru National Park were carried out on a mature female, white rhinoceros, Ceralotherium simum, during December 1971 and February 1972. The park is on the border of the Northern Frontier District of Kenya, 200 miles North of Nairobi at an altitude of 1100 metres. The animal was one of a group of six that had been translocated from South Africa in 1966, the survivers of which are all presently in Meru National Park. Of these, the two surviving females are closely guarded while out grazing in the Park during the day. They are kept in an enclosure

overnight and also return to it from 11.00 hrs. to 15.00 hrs. Readings could thus be taken at the same time of day as the other field experiments mentioned above. Although the animals are not completely tame they tolerated the equipment for short periods provided no sudden movement or noise was made. While out grazing they permitted an approach close enough to see respiratory movements.

c) Athi River

The two zebras, Equus burchelli, used were kept at the field station of the Department of Animal Physiology at Athi River. This is 25 miles S.E. of Nairobi at an altitude of 1,500 metres. The animals live in semi-natural conditions in 2 to 3 acre paddocks where the natural grazing and browsing is supplemented by lucerne and hay. The animals are allowed water ad libitum. Normally the animals have free range within these paddocks along with a hartebeest and two wildebeest. For the experiments the zebras were brought into a quarter of an acre paddock which contained a crush for restraining the animals movements. The two animals, a two year old, 204 kg female, and a two and a half year old 248 kg female, had been previously trained to stand in the crush and tolerate the application of experimental equipment. Normally they were let out into the smaller

paddock between hourly readings to minimise the stress which results from handling such animals. While in the crush they were fed lucerne hay for the same reason.

Experiments to test the tranquiliser

Experiments were carried out to ensure that the tranquiliser, phenycyclidine hydrochloride (Sernylan, Parke Davies) did not suppress the action of adrenaline, noradrenaline and acetylcholine. This was done by monitoring the changes in blood pressure caused by these drugs before and after the administration of the tranquiliser. Rats and rabbits which had previously been kept in a hygenic, well fed condition in small animal accommodation belonging to the Department of Animal Physiology at the University of Nairobi were used in these experiments.

The rabbits were first anaesthetised with ether, the skull was then opened and a mid-collicular decerebration carried out. The animal was then attached to an animal respirator (Phillips & Bird Incorp., Richmond, Virginia U.S.A.) for the duration of the experiment to maintain respiration at a normal level. Blood pressure was monitored by inserting a small polyvinyl cannula (Portex Ltd., Hythe, England) into the femoral artery. This was then filled with heparinised saline solution and

attach to a mercury manometer. A tracing of changes in blood pressure was made on a revolving, smoked kymograph drum. The drugs were administered through a similar cannula inserted into the femoral vein. Each drug was washed in with heparinished saline, and at the least 20 mins was allowed between the administration of the different drugs.

The rats used were first anaesthetised with ether and afterwards with Ethyl Carbamate (urethane). The blood pressure was measured as mentioned above from the carotid artery. The drugs were administered through another polyvinyl cannula inserted into the ipsilateral jugular vein. At the termination of the experiments all animals were destroyed.

Environmental control and measurement

Initial work with the galagos was carried out using a small galvanised iron chamber measuring 31 cm x 31 cm x 32 cms which was heated in a water bath. The water bath was heated to the required temperature by a 0.84 watt Braun water heater and the bath was completely surrounded by polystyrene foam two inches thick to prevent excessive loss of heat. The chamber was continuously ventilated using a Medcalf (Potters Bar, England) low volume rotary pump. Dry bulb temperature was measured by a 30 gauge Iron/Constantine thermocouple (Revere Corporation, Wallingford, Connecticut) suspended

inside the chamber. This was connected to a Honeywell, 12 channel recorder (Honeywell, Fort Washington, Pa. USA) and had been previously calibrated in a water bath. Wet bulb temperature was measured by a similar thermocouple which had been placed inside a moistened cigarette lighter wick. One end of the wick was immersed in a small container of water and the thermocouple was placed adjacent to the ventilating exit. This thermocouple was attached to the same Honeywell 12 channel recorder. The temperature inside the chamber could be lowered below ambient temperature if required by the addition of ice to the water bath. It was possible to increase the temperature as required and this was maintained to $\pm$ 0.5°C.

Later work was carried out in a climatic room built at the Department of Animal Physiology during January and February 1971. The room which measured 4m x 2½m x 3m, was heated by a Schmidt and Sohne (Munich, Germany) air conditioning unit and controlled to $\pm$ 1.5°C by a domestic thermostat. The heater was switched on four to six hours prior to the commencement of any experiment, to ensure that the walls, floor and air were all at the required temperature. The humidity within the room could be controlled by adjusting an exhaust fan and two air inlets. The wet and dry bulb temperatures were measured using a pair of Casella (London) thermometers or a Casella (London) Assman Hygrometer.

Wet and dry bulb temperatures in the field were measured using a Casella (London) whirling hygrometer.

Infra Red Heating

Infra red heating of a localised skin area was also used as a stimulus to sweating. A 250 watt Phillips Infra red bulb was placed 20 to 30 cms away from the previously prepared area. This area had been shaved of any hair and starch and iodine applied (see below). Heat was applied for 5 to 10 minutes through a hole in a piece of polystyrene so that a distinct circle of heat was produced on the experimental area. Any sweating then showed up as black dots within this circle of heat.

Measurement of body temperatures

The body temperatures of the galagos exposed to high environmental temperatures in the small climatic chamber were continuously monitored using a 30 gauge Iron/Constantan thermocouple (Revere Corporation Wallingford, Connecticut). This was enclosed in a vinyl sheath and attached to a Leeds Northrup Amar recorder, and calibrated in a water bath. When required for use it was inserted three to four cms into the rectum, and if necessary taped to the tail of the animal. This did not appear to disturb the animal in any way as it would

sit quietly with the thermocouple in place.

The body temperatures of the chimpansees and the baboons could be measured using clinical thermometers provided the animals were tranquilised. These were inserted 3-4 cms into the rectum and left in position for two to three minutes before a reading was taken. The aggressive behaviour of the baboons prevented this in their untranquilised state, while the smaller chimpansee also refused to tolerate the thermometer if untranquilised. To overcome this problem in the baboon a temperature sensitive thermistor, after a design by McGinnis (1968) was inserted aseptically into the peritoneal cavity. The surgery was carried out by Prof. D. Robertshaw of the Department of Animal Physiology. The baboons were tranquilised and given subcutaneous and intramuscular local anaesthesia (2% procaine) and the telemetric device inserted through a small, ventral mid line incision. The entire transmitter, powered by a Ray-o-vac R212 mercury battery and sealed in beeswax, measured 2.0 cm in diameter and 2.5 cm in length and appeared to cause the animal no discomfort. It was inserted a week before any experiments were carried out, by the time of the experiments the animals had completely recovered.

The signal it produced was a series of clicks, the frequency of which was linearly related to its temperature, and could be received on any medium

wave frequency radio. As the telemeter had previously been calibrated in a stirred, known-temperature water bath, by measuring the number of clicks per minute the temperature of the abdominal cavity could be calculated. The device functioned perfectly during the two to three weeks it was in place, and was easily removed, when the experiments were completed, using the same surgical procedure. Both animals operated on are still in excellent health. The transmitters remained close to the site of insertion and became surrounded by omentum. They did not, therefore, move around the peritoneal cavity.

The body temperature of the animals in the field was measured using a clinical thermometer. This was attached to a piece of plastic tubing and inserted 8-9 cms into the rectum. It was left in place for 2-3 minutes before a reading was taken.

The skin temperature of the rhinoceros and the elephant was measured using an Infra-red field thermometer, Type PRT-10 (Barnes Engineering Co., Stanford, Conn. U.S.A.). This was based on the original design of Gates (1968) and had a sensitivity to wavelengths of radiation within the range of 6.9 to 20 microns.

Measurement of cutaneous moisture loss (C.M.L.)

A quantitative assessment of C.M.L. of all the species used was made using a dessicant capsule

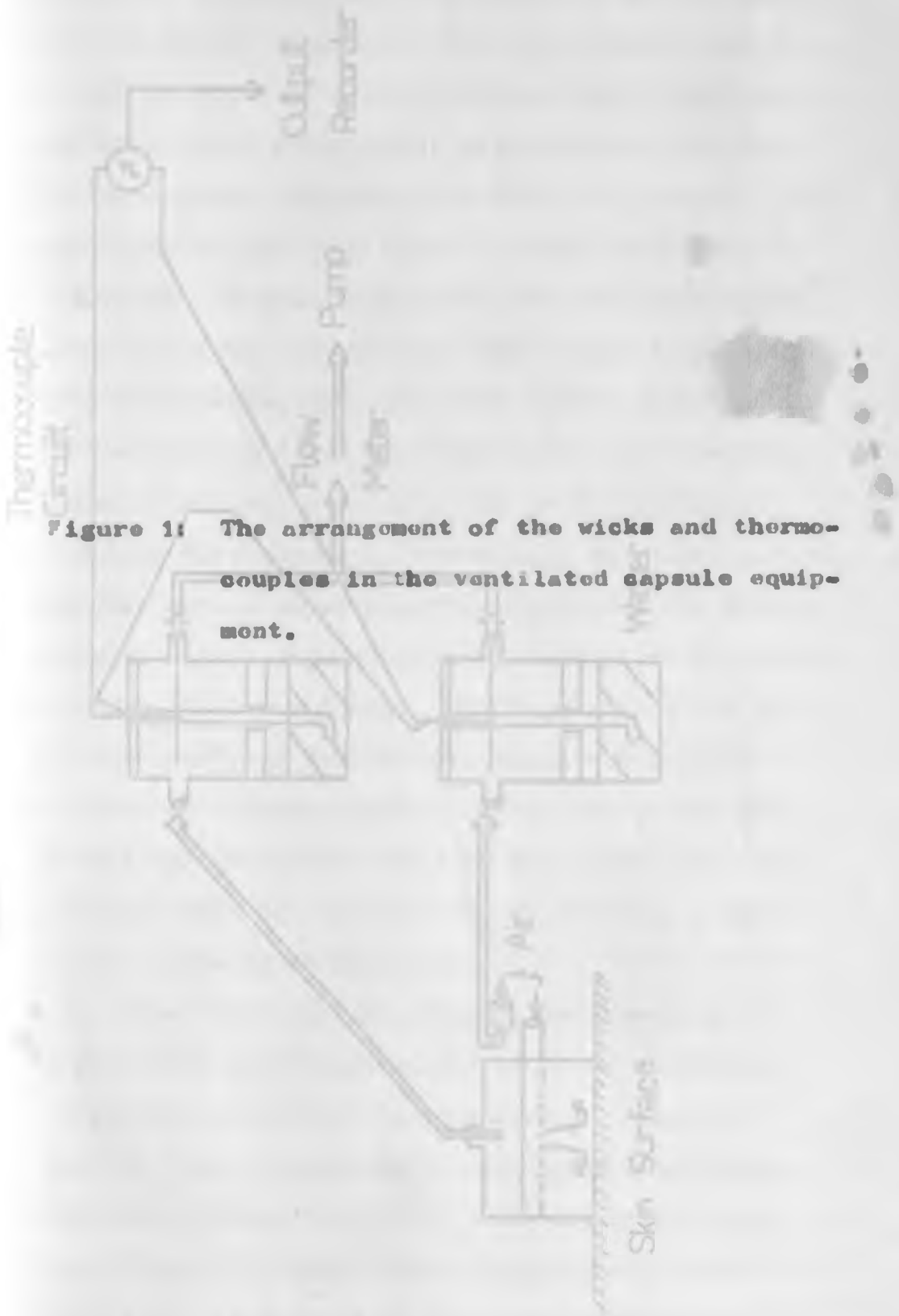
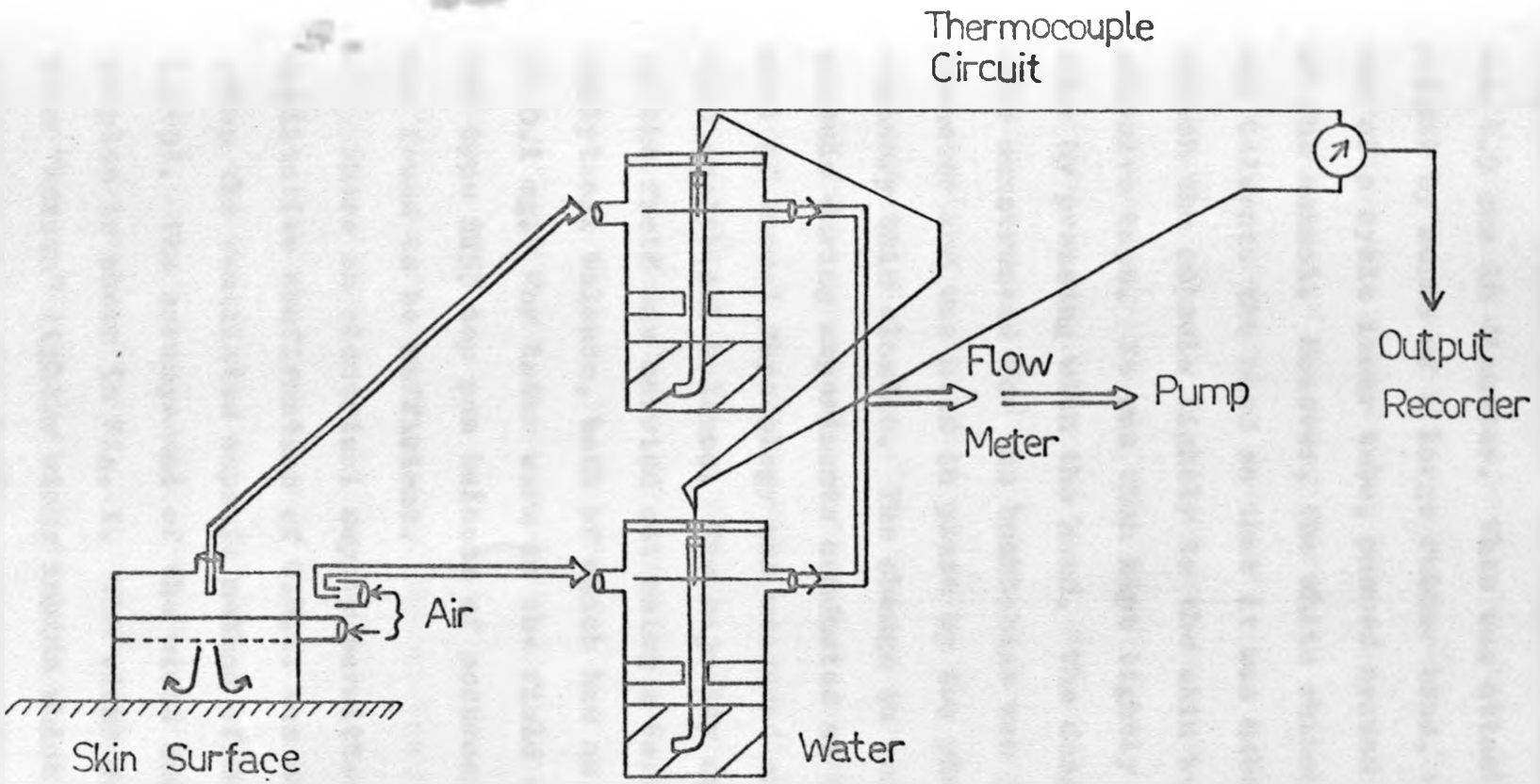


Figure 1: The arrangement of the wicks and thermocouples in the ventilated capsule equipment.


Thermocouple
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Air
Skin Surface
Water

(Taylor & Lyneman 1969). The capsule used normally was 4.9 cms in diameter. This was attached to the animal by means of a large rubber band, constructed out of a cycle inner tube, passed around the body of the animal. However, the white rhinoceros would not tolerate the band so that it was necessary to attach the capsule lightly to the skin with wide adhesive tape. It was then kept tightly against the skin by pressing with the hand. The dessicant capsule constructed for the bushbabies was 3 cms in diameter and was held in place by two pieces of ordinary thin elastic. The change in weight of the capsule during experiments conducted at the Department of Animal Physiology was measured on a Mettler H.18 analytical balance. The majority of the work in the field was carried out using a Sartorius analytical balance, both of which had an accuracy of 0.1 mg. For later work in the field a Sartorius type 2255 top pan balance of accuracy 1 mg was found to be sufficient.

Where an electrical supply permitted, a qualitative confirmation of C.M.L. was obtained using the ventilated capsule method of Maclean (1963). The arrangement of the wicks and thermocouples is shown in Fig. 1. The wicks were made from "Ronson" lighter wicks inside which there were a single pair of 38 gauge Copper/Constantan thermocouples (Dural Plastics and Eng. Pty., Dural,

N.S.W., Australia). The cup was 5.8 cms in diameter and the equipment was ventilated by a 240 volt Edwards Hi-Vacuum Pump (Edwards Ltd., Crawley, Sussex). The ventilation rate was controlled to 1.2 litres per minute by means of an M.F.G. 1100 rotameter (Rotameter Manufact. Co., Richmond, England). It was necessary to construct a smaller ventilated capsule for the bushbabies. This measured 3 cms in diameter and was ventilated at a rate of 1 litre per minute. To increase the sensitivity of the equipment when this was used the single pair of thermocouples in the wicks was replaced by 5 pairs of 48 gauge Copper/Constantan thermocouples (Dural Plastic and Engineering Pty., Dural, N.S.W., Australia) connected in series. The output from these, or the single thermocouplepair, was connected to a Kent Mark 3, 36 range millivolt recordor (Kent Industrial Instruments Ltd., Luton, Bedfordshire).

In experiments carried out at the Department of Animal Physiology it was possible to use the mains electricity to power the pump and recorder. At the field station at Athi River a Honda portable generator was used to provide the power.

The wicks and thermocouples were, whenever possible, placed in the same temperature conditions to which the animals were exposed. The recorder, pump, and other equipment were then placed outside

the climatic chamber, or shaded from the sun when in the field. This was impossible using the small climatic chamber so the wicks and thermocouples were placed as close as possible to the point of exit of the air streams from the chamber. By doing this it was hoped to minimise any cooling of the tubes containing the air prior to the contact of the air with the thermocouple/wick system.

The Starch-Iodine method of Wada (1950) was also used as a qualitative method of assessing C.M.L. Iodine in alcohol was applied to the skin, allowed to dry, and covered with a mixture of Starch and Castor Oil. Any activity of the sweat glands extruded moisture onto the surface of the skin. In the area to which the mixture had been applied the water, starch and iodine combined together, so that active sweat glands appeared as purple/black spots. Starch-Iodine filter papers were produced by soaking filter papers in a starch solution. After drying, they were dipped in a solution of iodine in alcohol and the alcohol allowed to evaporate inside a dessicator. If stored in a dessicator they could then be used to detect active sweat glands. If the papers were held against the skin the same purple/black colouration appeared where moisture was being produced.

Measurement of respiration rate

In all the animals used, the respiration rate

could normally be measured by counting flank or chest movements with the aid of a stopwatch. Respiration rates were, where possible, recorded as the average of three or four measurements taken over half a minute.

Computation (for ventilated capsule method)

An attempt was made to simplify and speed up the calculation of a quantitative value for C.M.L. which could be obtained from the millivolt reading off the Kent recorder. A computer programme in Fortran IV for use on the ICL 1900 computer belonging to the University of Nairobi Computing Centre was produced to make this possible (Appendix 1).

Injection of Drugs (1) Intradermal

The local actions of sympathetic and parasympathetic drugs were investigated in the galagos and the baboons by giving intradermal injections of adrenaline, noradrenaline, carbachol, acetylcholine, and pilocarpine. The experimental area used in the galagos was over the 6th and 10th ribs in the dorsal quarter of the body; in the baboon it was an area over the last four ribs in the ventral quarter. The area was shaved and an iodine solution applied to it. After this had dried the required concentrations of the drug to be used were then injected intradermally, using a 24 gauge

needle. The position of each injection was noted and starch in castor oil placed over and around the injection wheal. Any sweating reaction was noted.

(2) Systemic

Systemic injections of these drugs were given into the short saphenous vein, where it comes to the surface over the gastrocnemius muscle. This skin was first shaved, then sterilised using alcohol before the drug was administered using a 24 gauge needle. Any reaction to the drug was measured by a ventilated or dessicant capsule placed over the areas referred to in the previous paragraph.

Histology

Wherever possible, a sample of skin was removed from the experimental area for histological examination. The baboon and the galago were first tranquillised and the area to be sampled injected with local anaesthetic (2% procaine). A small sample was then removed using a forceps and scalpel, and immediately placed in a 5% formalin solution.

The rhinoceros skin sample was obtained from an animal killed in a collision with a lorry in Tsavo National Park. The elephant skin sample was obtained from a recently shot animal. Both animals had been dead for less than two hours when the samples were taken. As before, these were immed-

iately placed in 5% formalin solution.

All these skin samples were then dehydrated and embedded in paraffin wax (melting point 60°C). After sectioning they were stained with haemotoxylin and eosin, or masson trichrome, and mounted.

RESULTS

The effect of Sernylan on the response to sympathetic and parasympathetic drugs

A typical tracing from an experiment on an anaesthatised rat is shown in fig. 2 and Table 1. An increase in blood pressure was caused by the administration of 2µg/kg. of adrenaline or noradrenaline. The same dose of Acetylcholine caused a decrease in blood pressure.

The administration of the Sernylan (2µg/kg) itself caused an increase in blood pressure. This pressor effect declined in the twenty minutes following the injection to a level slightly above the initial pressure and remained elevated for the duration of the experiment. The administration of the same dose of adrenaline or noradrenaline as previously again caused an increase in blood pressure. However, the Sernylan potentiated this pressor response. The effect of the Acetylcholine was not changed by the Sernylan (fig. 3 Table 1).

These results confirm the findings of Hirt, Jarrot, O'Donnel and Wastell (1966) who attributed this potentiation of the pressor response to the action of the Sernylan on adrenergic α- receptors and also indirectly on the release of catecholamine stores. Therefore, it was concluded that the administration of Sernylan as a tranquilliser to the animals that might be used in this investigation

TABLE I

Blood pressure changes in the rat in response to 2µg/kg injections of adrenaline, nor adrenaline and acetylcholine before, and after, the administration of phencyclidine hydrochloride.

Drug	Blood Pressure (mm. Hg)		
	Before	Peak	Change
Nor Adrenaline	19	56	+ 37
Adrenaline	18	68	+ 50
Acetylcholine	18	7	- 11
Nor Adrenaline	28	100	+ 72
Adrenaline	28	111	+ 83
Acetycholine	28	17	- 11

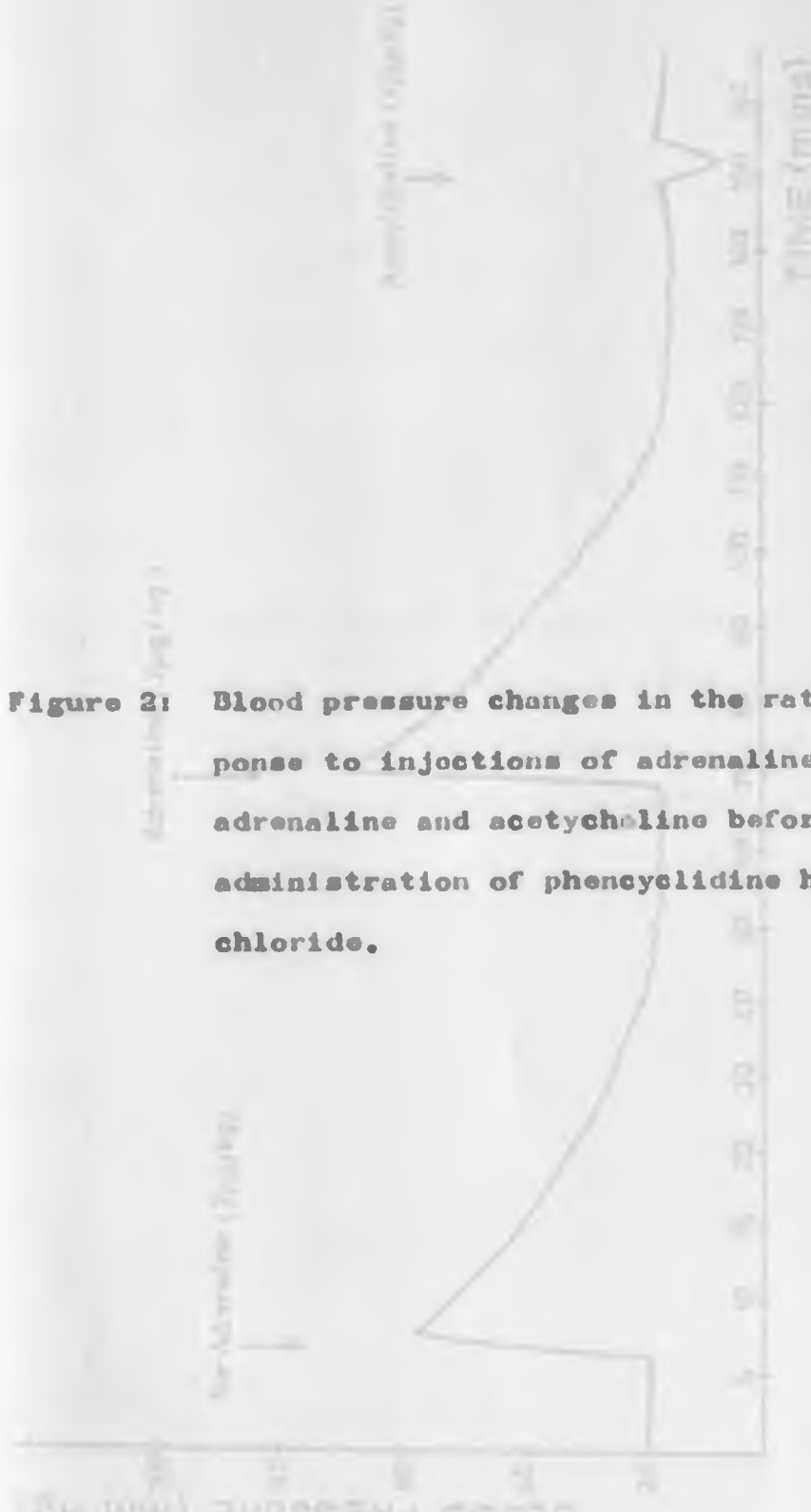
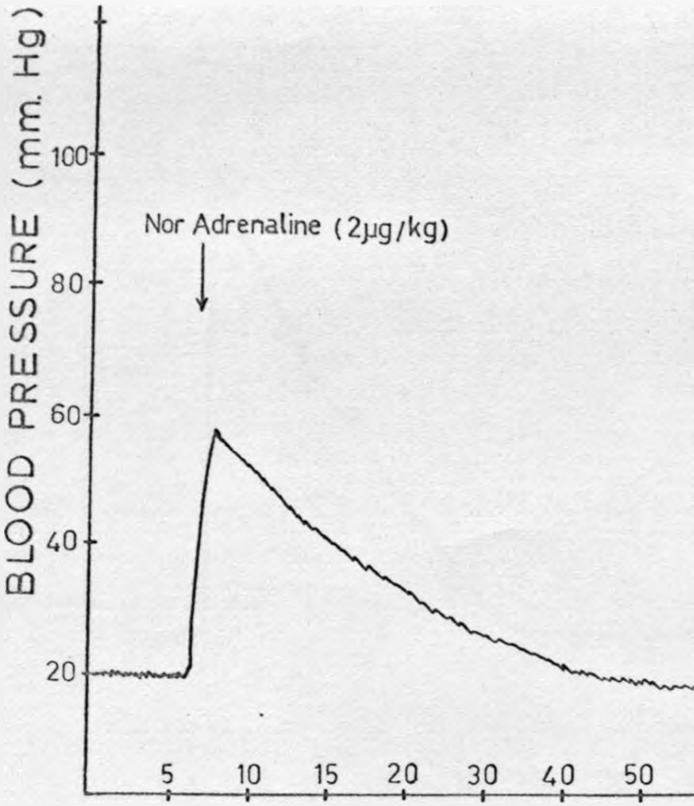
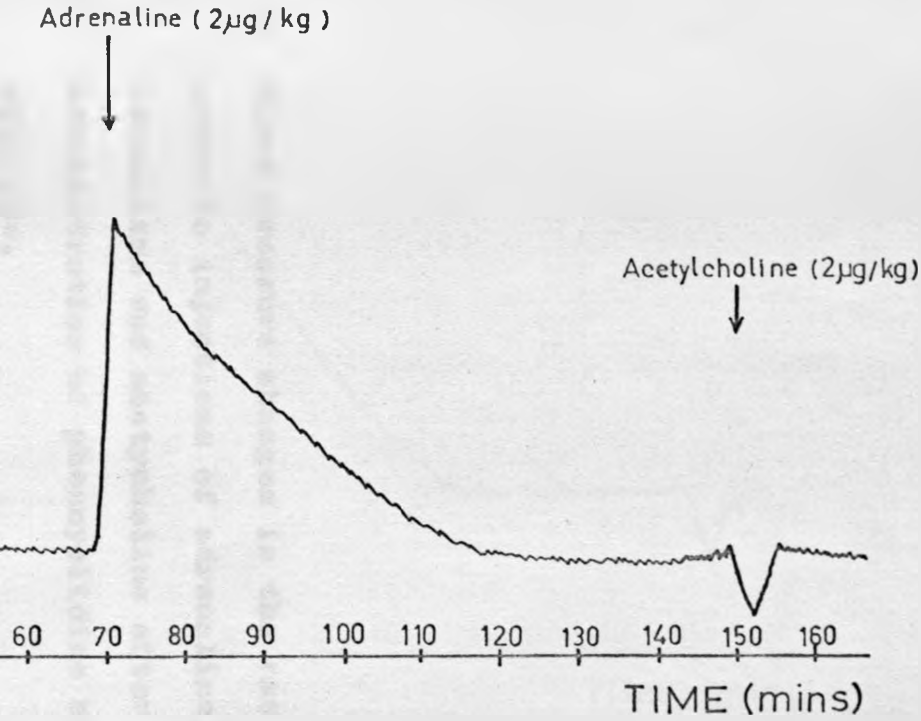


Figure 2: Blood pressure changes in the rat in response to injections of adrenaline, nor adrenaline and acetycholine before the administration of phencyclidine hydrochloride.


BLOOD PRESSURE (mm. Hg)
100
80
60
40
20
Nor Adrenaline (2µg/kg)
5
10
15
20
30
40
50


Adrenaline (2µg / kg)
Acetylcholine (2µg/kg)
60
70
80
90
100
110
120
130
140
150
160
TIME (mins)

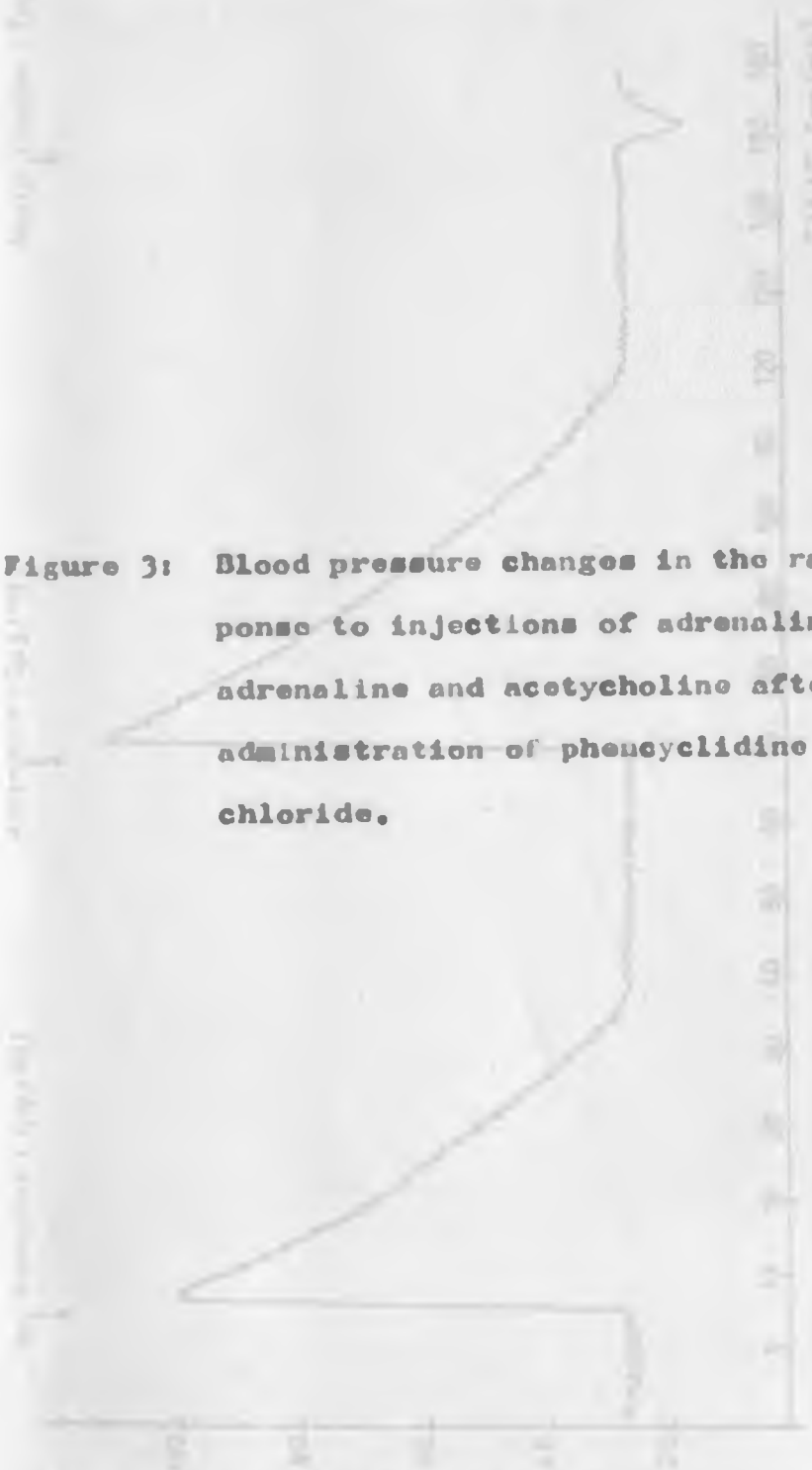
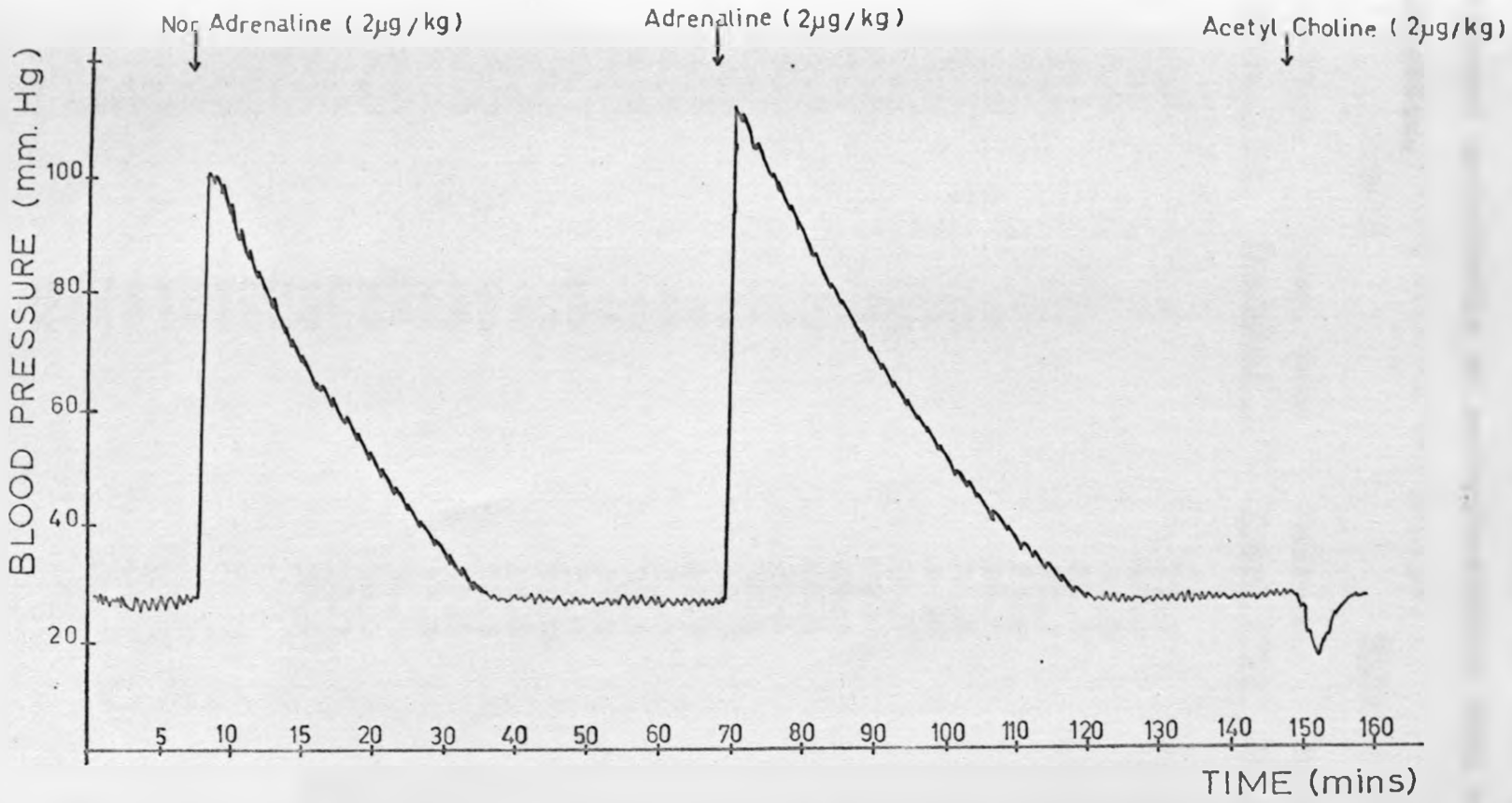


Figure 3: Blood pressure changes in the rat in response to injections of adrenaline, nor adrenaline and acetycholine after the administration of phencyclidine hydrochloride.


Nor Adrenaline (2µg / kg)
Adrenaline (2µg / kg)
Acetyl Choline (2µg/kg)
BLOOD PRESSURE (mm. Hg)
100
80
60
40
20
5
10
15
20
30
40
50
60
70
80
90
100
110
120
130
140
150
160
TIME (mins)

would not affect their qualitative response to subsequent sympathetic or parasympathetic drug administration.

TABLE 2

The effect of increasing environmental temperature on respiration rate of the galago.

Female		Male	
Environ. Temp. (°C)	Resp. Rate (no/min)	Environ. Temp. (°C)	Resp. Rate (no/min)
17.8	70	17.7	73
19.5	86	19.0	60
21.0	94	20.9	85
23.1	94	22.4	50
24.2	53	23.5	40
25.1	40	24.0	52
25.5	70	24.0	55
25.5	75	24.8	60
26.0	71	25.7	52
26.5	44	26.5	60
27.0	66	27.0	44
27.7	38	29.0	60
28.0	75	29.9	60
28.5	76	31.1	75
29.5	66	31.5	34
29.7	44	32.3	65
30.4	76	32.5	53
31.2	44	33.2	90
31.3	72	33.5	58
31.9	64	34.5	85
32.7	100	35.0	80
33.5	92	35.2	120
33.8	54	35.8	122
34.3	100	36.3	108
34.4	120	37.0	238
35.1	100	37.0	240
35.5	171	38.0	200
36.0	150	38.0	260
36.2	150	38.7	256
36.6	187	39.5	284
37.2	172		
37.3	170		
38.0	200		
38.1	222		
38.5	204		
39.0	200		
39.1	222		
40.0	210		
40.1	220		

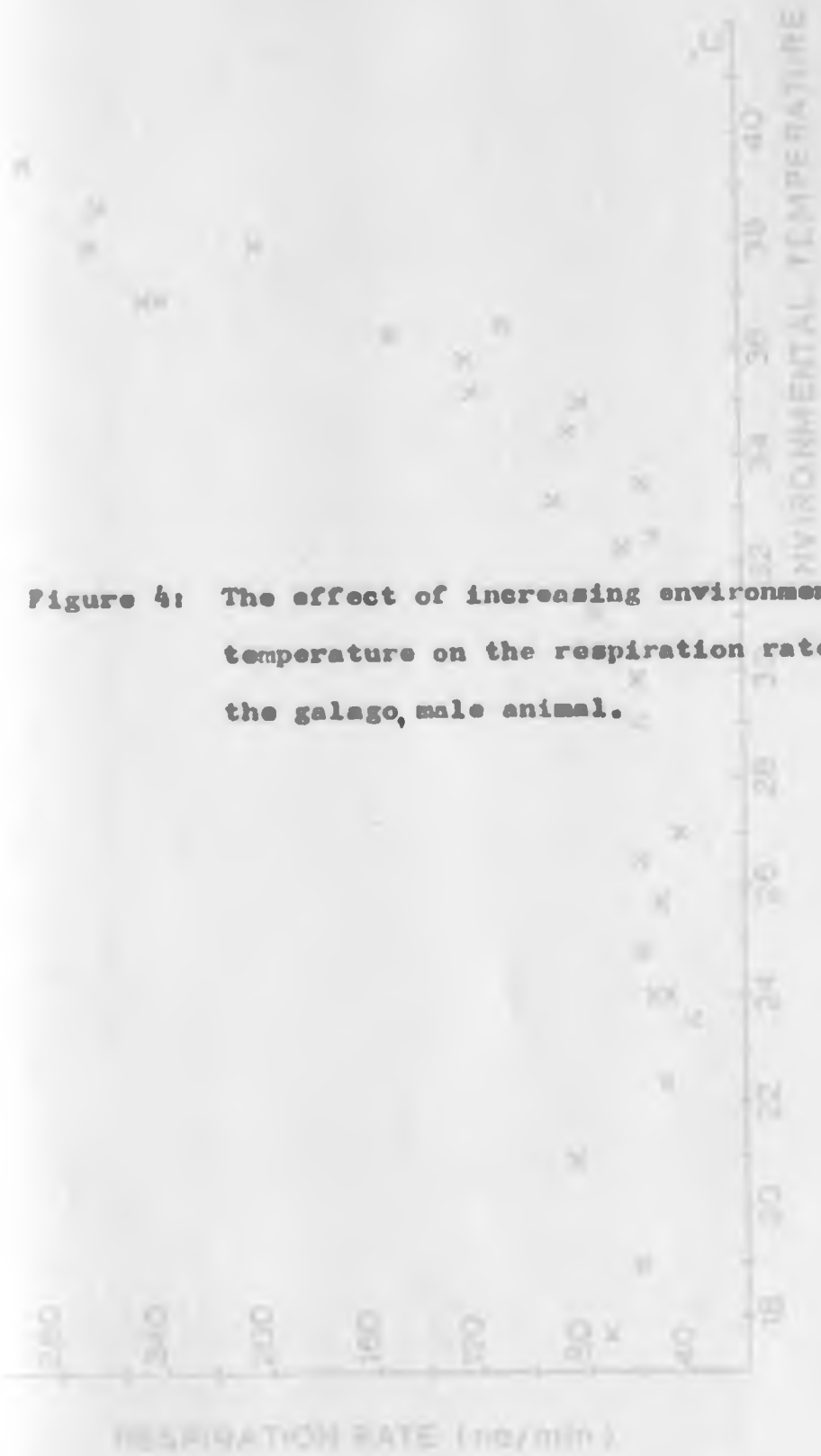
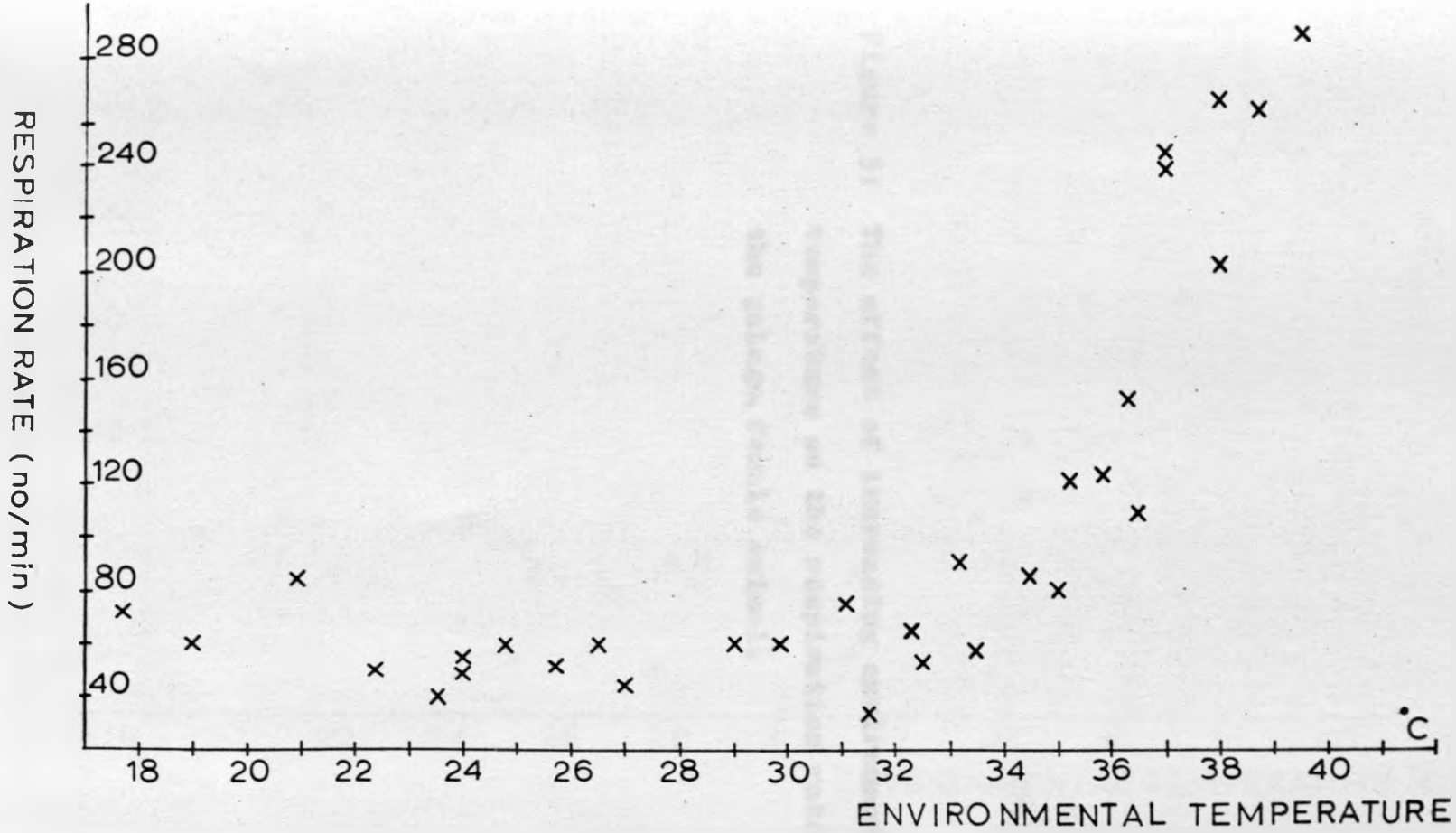


Figure 4: The effect of increasing environmental temperature on the respiration rate of the galago, male animal.


RESPIRATION RATE (no/min)
280
240
200
160
120
80
40
18
20
22
24
26
28
30
32
34
36
38
40
°C
ENVIRONMENTAL TEMPERATURE

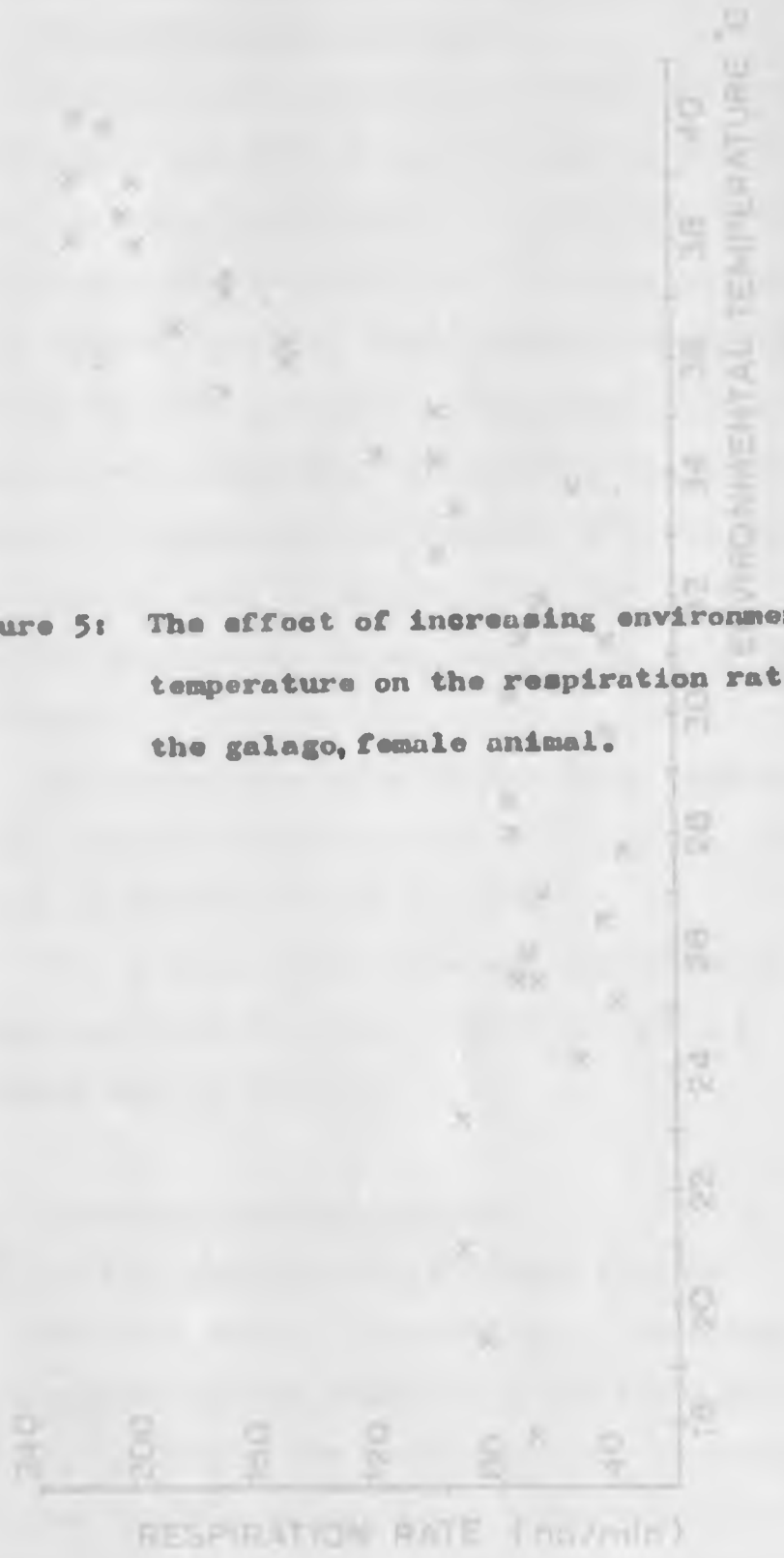
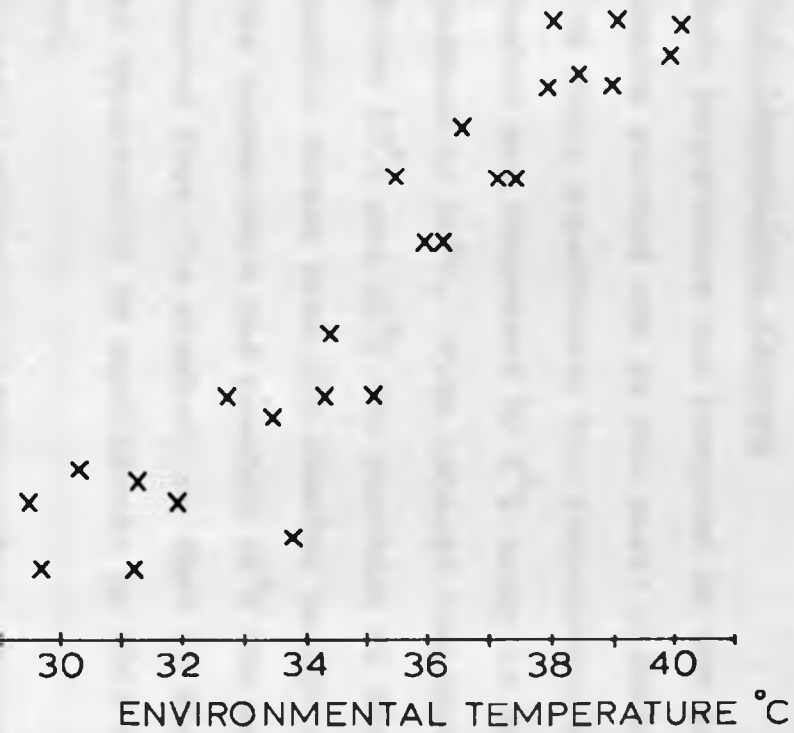


Figure 5: The effect of increasing environmental temperature on the respiration rate of the galago, female animal.

240
200
160
120
80
40
RESPIRATION RATE (no/min)
18
20
22
24
26
28


30
32
34
36
38
40
ENVIRONMENTAL TEMPERATURE °C

The effect of heat exposure on the galago

1. Body temperature changes

Body temperature was recorded in the seven experiments carried out in the small climatic chamber. In these experiments the temperature inside the chamber was increased by 1°C every 10 min. up to a maximum of 40°C. With ambient temperatures of between 17°C and 23°C the duration of the experiments varied from 170 minutes to 230 minutes. Once the temperature had reached 40°C the animal was removed from the chamber, so that it did not have an opportunity to equilibrate to this temperature.

Rectal temperatures during these experiments never increased by more than 0.6°C from a mean value of 38.4°C at 18°C to 20°C.

The animal always remained placid during these experiments, lying curled up with its tail wrapped around its body.

2. Respiration rate changes

(a) In the portable climatic chamber

In both animals the gradually increasing temperature in the chamber did not cause an increase in respiration rate until the temperature reached 33-34°C (figs. 4 and 5 and Table 2). Above this the increase in respiration rate with an increase in environmental temperature was linear,

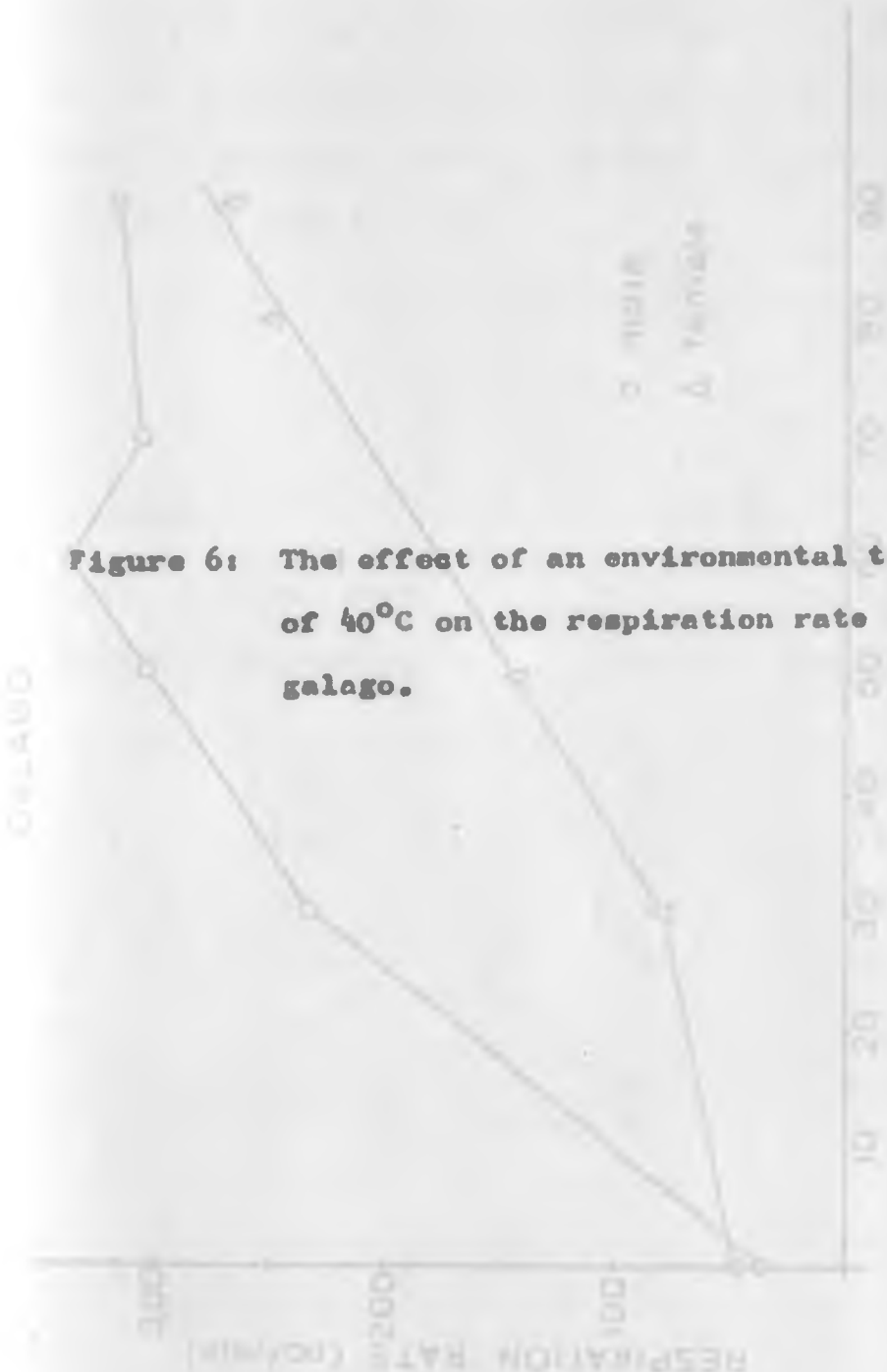


Figure 6: The effect of an environmental temperature of 40°C on the respiration rate of the galago.

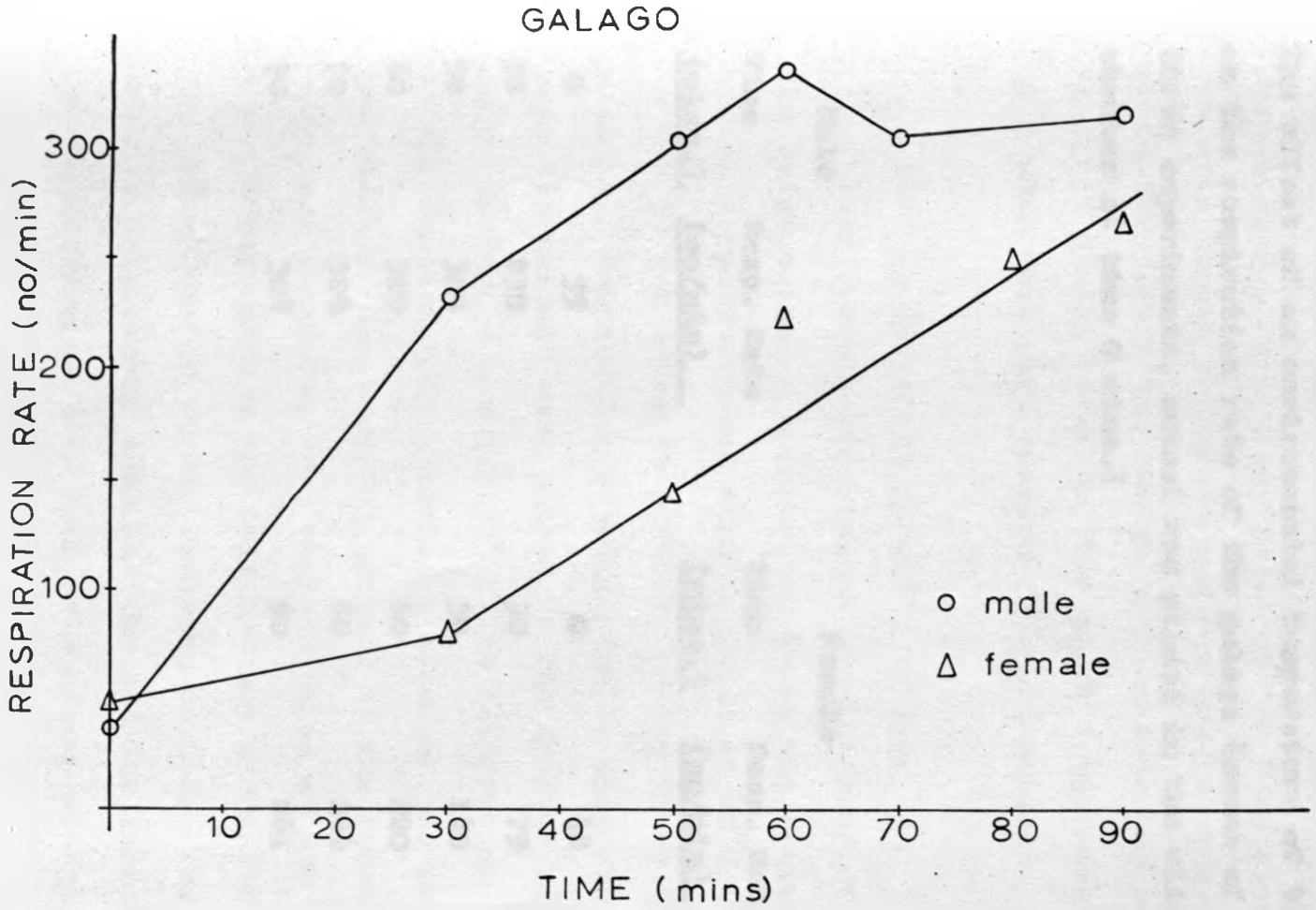

GALAGO
RESPIRATION RATE (no/min)
300
200
100
10
20
30
40
50
60
70
80
90
TIME (mins)
male
female

TABLE 3

The effect of an environmental temperature of 40°C on the respiration rate of the galago (means of three experiments, animal was placed in the climatic chamber at time 0 mins.)

Male		Female	
Time (mins.)	Resp. Rate (no/min)	Time (mins.)	Resp. Rate (no/min)
0	35	0	48
30	230	30	79
50	300	50	140
60	329	60	220
70	304	80	244
90	308	90	261

the least squares regression of this relationship being y=20.39x-585.06 for the female animal and y=29.14x-890.17 for the male animal. The correlations for these relationships being 0.946 and 0.895 respectively, both significant at the 0.1% level. The male animal therefore had the greater increase in respiration rate, though both animals had probably not equilibrated to this upper temperature of 40°C when they were removed from the chamber.

(b) In the large climatic chamber

Seven further experiments were carried out in the large climatic chamber, in which the environmental temperature was kept constant at 40°C (± 1.5°C) for exposure periods lasting up to 90 minutes. Respiration rates gradually increased once the animals were placed in the chamber as shown in fig. 6 and Table 3. The respiration rate of the smaller male stabilised at about 300 breaths per minute. The respiration rate of the female rose more slowly up to a maximum value of 250 breaths per minute, but this had not stabilised by the end of the 90 minute exposure period. The smaller male animal thus had the greater increase in respiration rate in both sets of experiments.

3. Cutaneous moisture loss changes

(a) In the portable climatic chamber

TABLE 4

The effect of environmental temperature on cutaneous moisture loss of the galago.

Dessicant Capsule

Animal	Environmental Temp. (°C)	C.M.L. ($g/m^2/hr$)
Female	17.5	34.2
"	17.5	17.4
"	18.0	26.2
"	18.5	22.9
"	18.5	22.9
		mean 24.7 (± 6.2 S.D.)
"	40.5	42.0
"	40.0	35.2
"	38.5	44.6
"	40.0	30.7
"	38.0	27.4
		mean 36.0 (± 7.3 S.D.)
Male	19.2	25.4
"	19.5	21.9
"	19.5	21.0
		mean 22.5 (± 2.5 S.D.)
"	40.0	53.5
"	40.0	38.6
"	40.0	36.5
"	41.0	33.3
"	41.0	29.0
"	39.0	24.6
		mean 35.9 (± 10.0 S.D.)

Ventilated Capsule

Female	17.0	8.7
"	39.0	35.6
"	19.0	59.1
"	39.5	45.3
Male	24.2	31.5
"	41.5	29.9

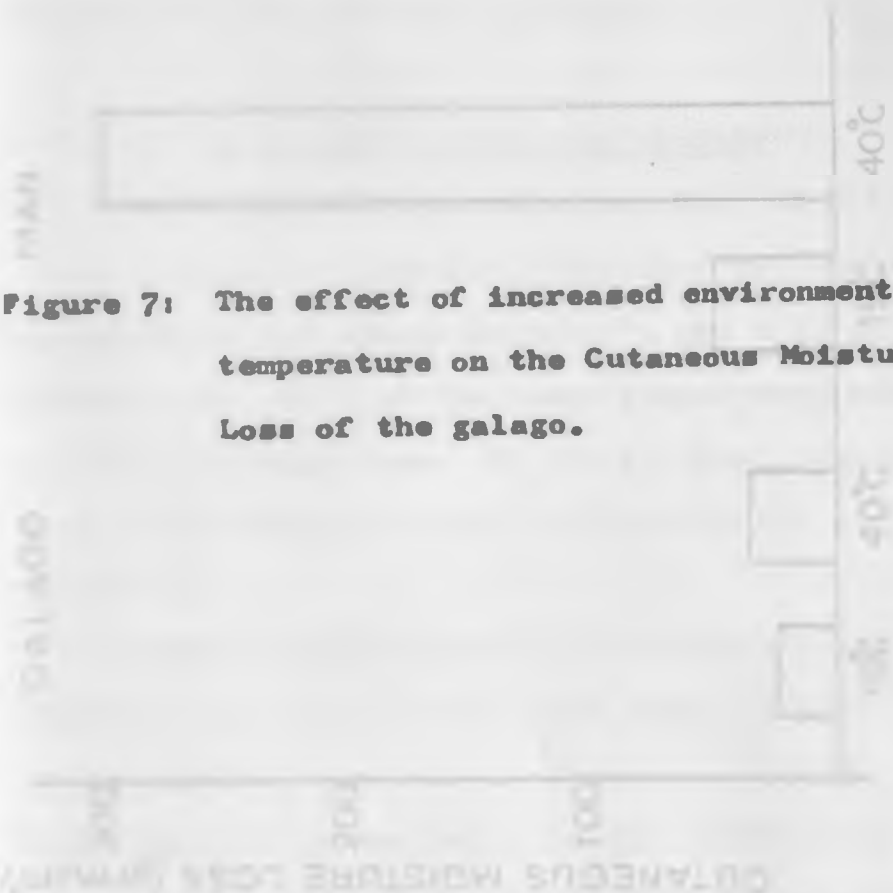


Figure 7: The effect of increased environmental temperature on the Cutaneous Moisture Loss of the galago.

CUTANEOUS MOISTURE LOSS (grm/m²/hr)
100
200
300
19°C
40°C
19°C
40°C
GALAGO
MAN

Due to the instability of the very sensitive thermocouple/wick system and possibly, due to the placement of these outside the chamber, the initial ventilated capsule results obtained were rather equivocal (see discussion of materials and methods). In some cases it appeared that the animals had an increased C.M.L. on heat exposure.

(b) In the large climatic chamber

The results of two experiments on each animal using a dessicant capsule, together with the control experiments, are shown in Table 4 and fig. 7. The increase in C.M.L. of the male animal when exposed to 40°C was significant at the 2% level, while that of the female animal was significant at the 5% level.

Further experiments using the ventilated capsule produced the results also shown in Table 4. In two of the experiments a decrease in C.M.L. was recorded on heat exposure, in the third there was a small increase. The mean difference between C.M.L. recorded at high and low temperatures is not significant.

4. Pharmacological stimulation

(a) Local

Adrenaline solutions in concentrations varying from 10^{-8} g/cc to 10^{-3} g/cc did not produce any

stimulation of the sweat glands. Acetylcholine solutions of the same concentrations also failed to stimulate them.

Injections of a 10^{-3} g/cc solution of Acetylcholine into the pads of the feet did, however, cause stimulation of the sweat glands present in this area.

It was noticed that the hair which grew again over the experimental area lacked any pigmentation. This was also reported by Findlay and Jenkinson (1964) after intradermal injections of adrenaline in cattle.

(b) Systemic

Adrenaline was injected intravenously in six experiments in amounts up to 10µg/kg. No sweat gland response was detected using the ventilated capsule.

5) Local infra-red heating

Two experiments on each of the animals failed to produce any sweat gland response to localised infra-red heating.

6) Skin sample

On completion of the above work two small skin samples were taken for histological examination.

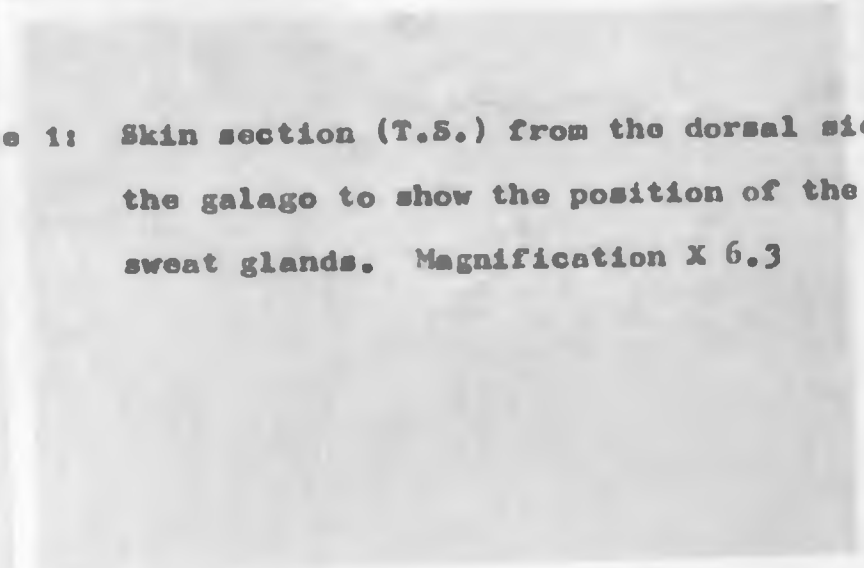
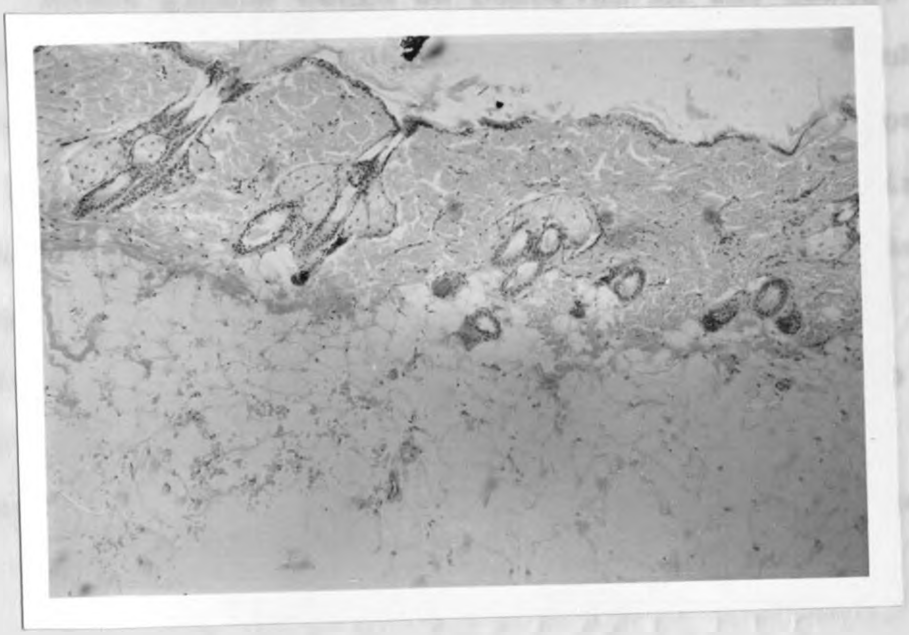
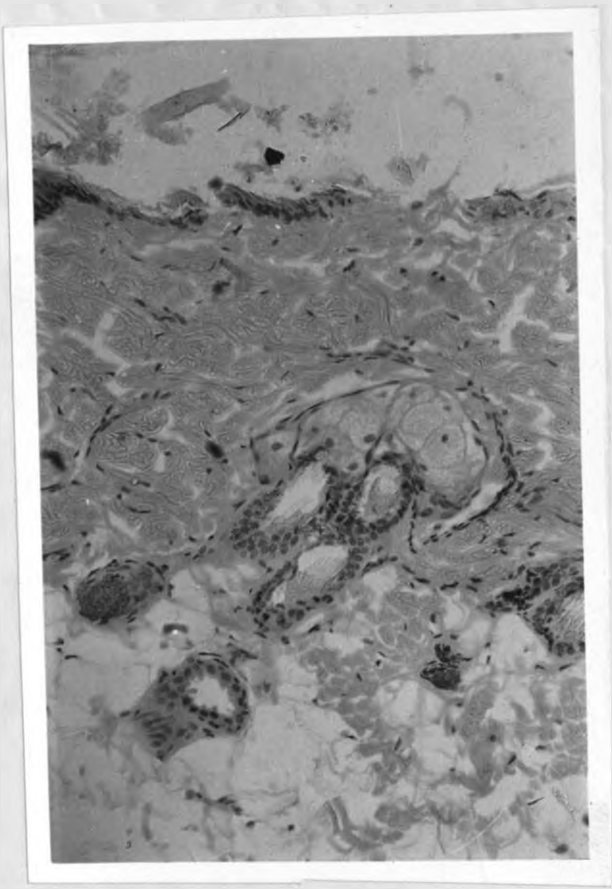


Plate 1: Skin section (T.S.) from the dorsal side of the galago to show the position of the sweat glands. Magnification X 6.3

Plate 2: The structure of a sweat gland in the galago (T.S.). Magnification X 16.0





Sweat glands could be observed in the lowest level of the dermis, Plate 1 and 2, and in the subcutaneous fat layer below this. These glands appear to have the typical gross configuration of apocrine glands (Schieffendecker 1917) and in Plate I the gland is seen adjacent to a sebacious gland of a hair follicle. The secretory cuboidal epithelium can be seen with apical, secretory projections. They are surrounded by a layer of myoepithelial cells.

TABLE 5

Rectal and abdominal temperatures recorded in the baboon. (Tr and Tab respectively).

	Duration of Exposure to 40 C	Animal 3	Animal 2
Experiment 1A			
Change in Tr.	50 mins	0.9	
Change in Tab.	" "	0.8	
1B			
Change in Tr.	70 mins		0.5
Change in Tab.	" "		0.3
1C			
Change in Tr.	105 mins	1.6	
Change in Tab.	" "	1.3	
1D			
Change in Tr.	90 mins		0.9
Change in Tab.	" "		0.9
Experiment 2A			
Change in Tab.			
Tranquillised	90 mins	0.9	
Untranquillised	" "	0.6	
2B			
Change in Tab.			
Tranquillised	80 mins		0.3
Untranquillised	" "		0.2
2C			
Change in Tab.			
Tranquillised	60 mins	0.8	
Untranquillised	" "	0.4	

The effect of heat exposure on the baboon

Previous results have shown (page 30) that Sernylan does not suppress the actions of sympathetic or parasympathetic drugs. It was not know, however, if it had any effect on the ability of an animal to overcome any heat stress imposed upon it. By recording changes in body temperature, in an untranquillised and a tranquillised animal, during similar heat exposure periods and then comparing the changes, any effect of the tranquilliser on thermoregulation would become evident. However, in the untranquillised animal, the only measurement of body temperature that could be made was abdominal temperature. It was, therefore, necessary to obtain simultaneous measurements of rectal and abdominal temperature on exposure to an air temperature of 40°C to see if abdominal temperature gave a reasonable indication of body temperature. If this was the case a direct comparison of abdominal temperature changes in the tranquillised and untranquillised animals could be made.

1. Body temperature changes

(a) Effect of the tranquilliser

Table 5 shows the simultaneous measurements of abdominal and rectal temperature mentioned above (Experiment 1A,B,C,D). These results show that there is no significant difference between the

TABLE 6

The effect of an environmental temperature of 40°C on the rectal temperature of baboon I (the animal was placed in the climatic chamber at time 0 mins.)

Time (Mins)	Rectal Temperature (°C) Expt. I	Expt. 2	Expt. 3	Expt. 4
0	38.5	39.5	39.5	
10		39.1		
15	38.2		39.3	39.55
20		39.1		
25			39.3	
30	38.4	39.2		40.0
35			39.5	
40	38.7			
45		39.5		40.1
50	38.8		39.95	
60	39.0		40.1	40.0
65		39.6		
70	39.2		40.0	
75		39.7		
80			39.9	
85	39.7			39.6
90			39.9	
95	39.95	39.8		
100			40.0	
110	39.9		40.0	
115	39.9			
120	39.9			

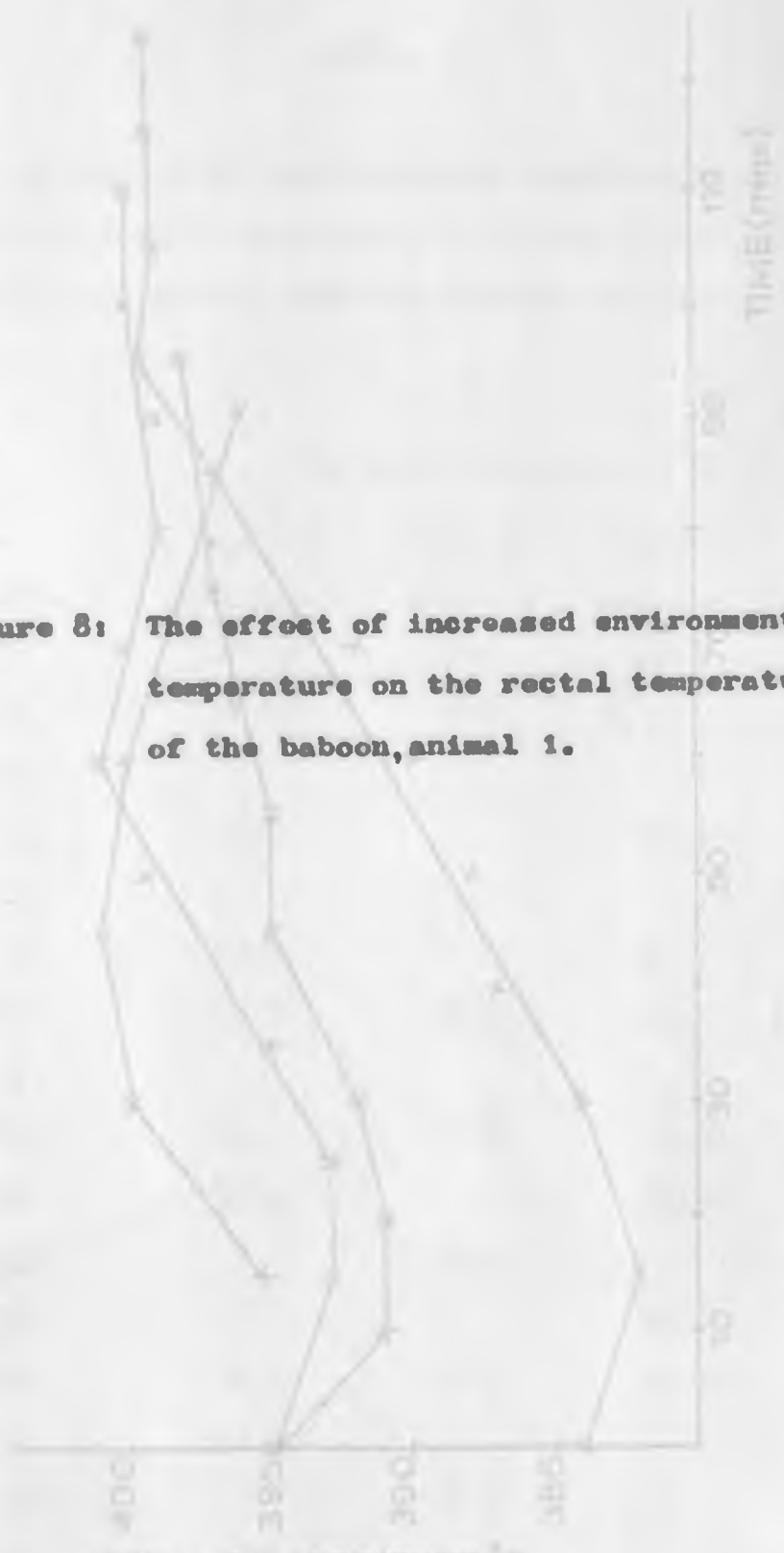
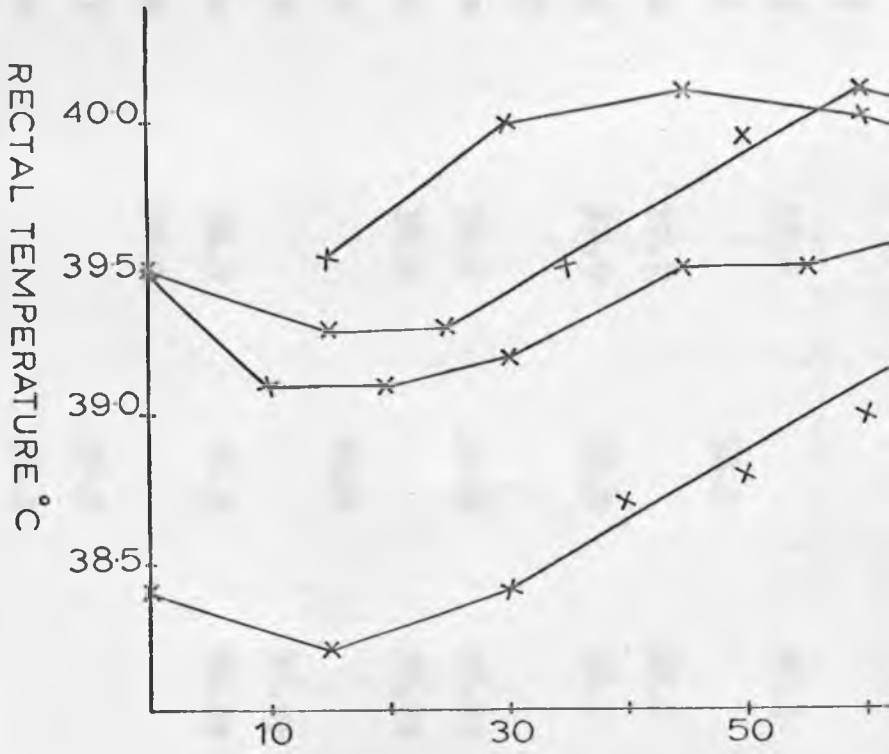


Figure 8: The effect of increased environmental temperature on the rectal temperature of the baboon, animal 1.


RECTAL TEMPERATURE °C
40·0
39·5
39·0
38·5
10
30
50

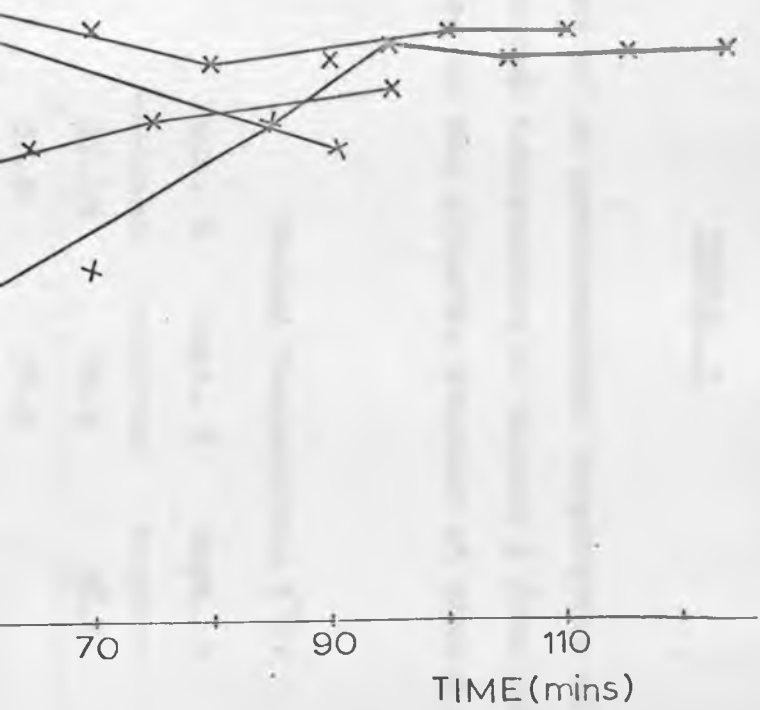

70
90
110
TIME (mins)

TABLE 7

The effect of an environmental temperature of 40°C on the rectal temperature of baboon 2 (the animal was placed in the climatic chamber at time 0 mins.)

Time (mins)	Rectal Temperature (°C) Expt. 1	Expt. 2	Expt. 3	Expt. 4
0	37.15	37.9	38.1	38.8
10	37.2	38.3		
15				39.1
20	37.6		38.3	
25		38.7		
30	37.7		38.6	39.3
40	37.9	38.9	38.7	
45				39.3
50	38.1	38.8	38.85	
60	38.2		38.85	39.1
65		39.0		
70			38.75	38.9
80	38.4	38.7	38.85	
90	38.1			
95		38.5		38.7
110		38.3		38.8
120		38.5		38.8

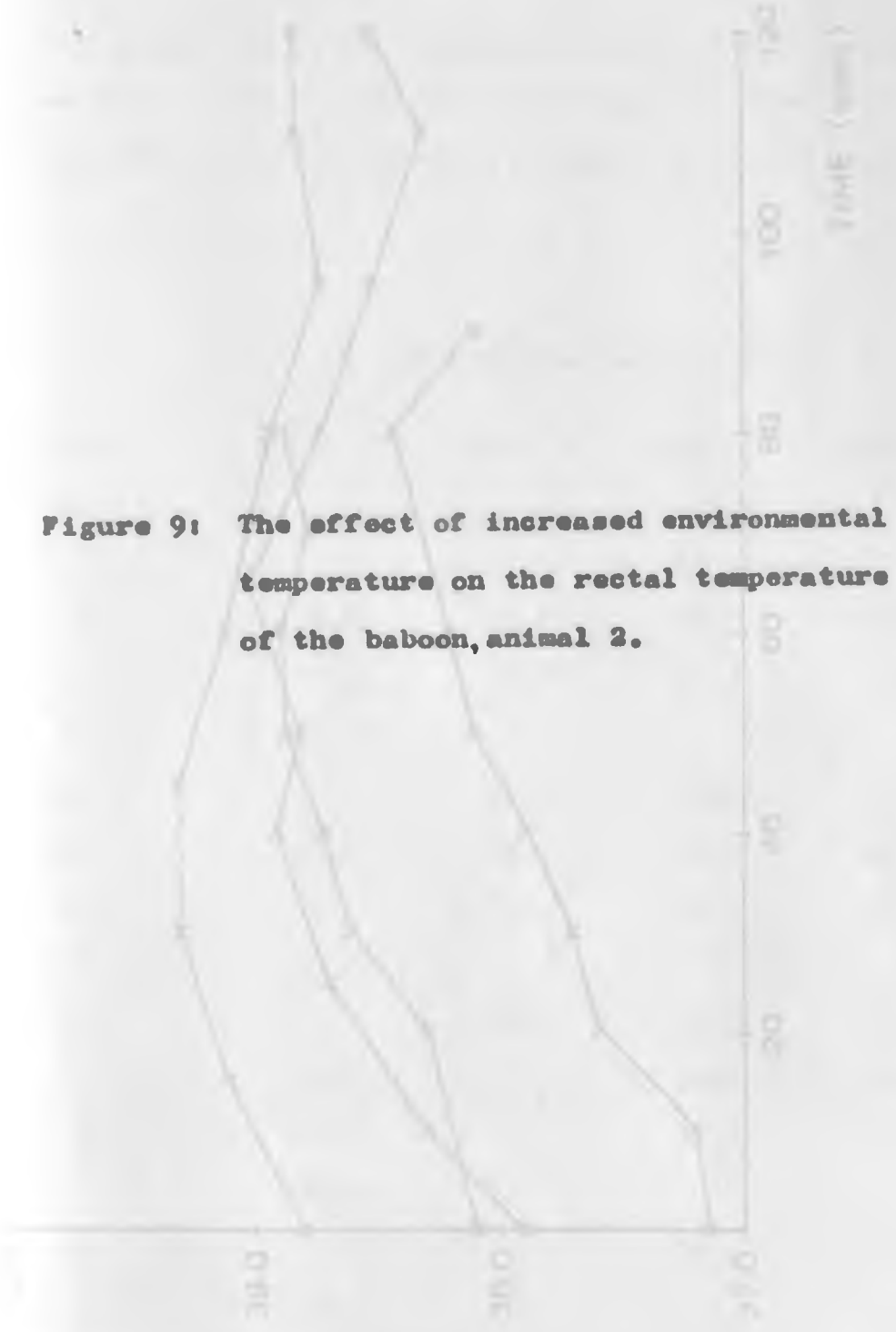


Figure 9: The effect of increased environmental temperature on the rectal temperature of the baboon, animal 2.

RECTAL TEMPERATURE (°C)
40.0
39.0
38.0
37.0
20
40

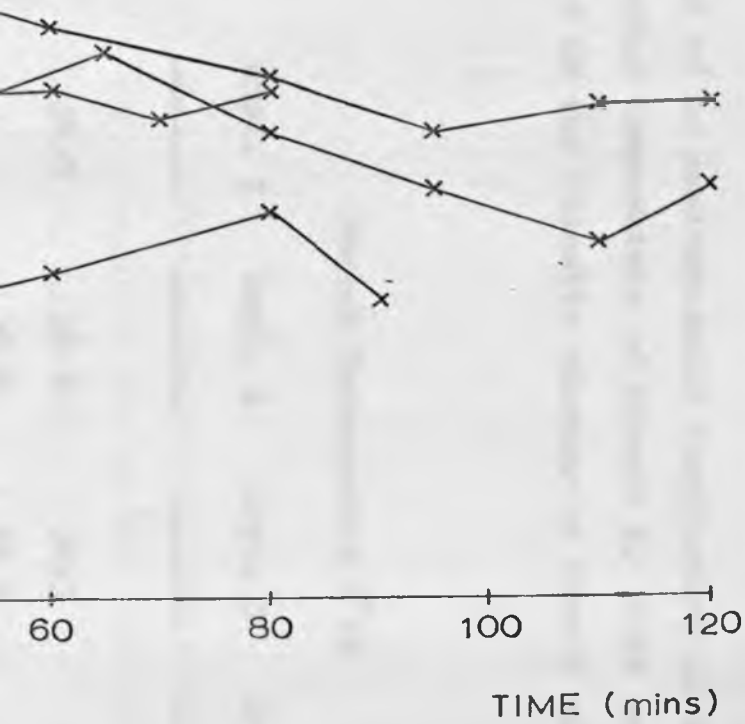

60
80
100
120
TIME (mins)

TABLE 8

The effect of an environmental temperature of 40°C on the rectal temperature of baboon 3. (The animal was placed in the climatic chamber at time 0 mins).

Time (mins)	Rectal Temperature (°C) Expt. 1	Expt. 2	Expt. 3	Expt. 4
0	38.5	38.4	38.3	38.1
10		38.6	38.4	38.0
15	38.2			
20		38.7	38.7	38.3
30	38.3	38.8	38.9	38.5
40		39.1	39.1	39.3
45	38.4			
50		39.3	39.4	39.6
60	39.0		39.6	39.9
70			39.4	40.1
75	39.6			
80			39.5	
90	39.9			
105	40.1			

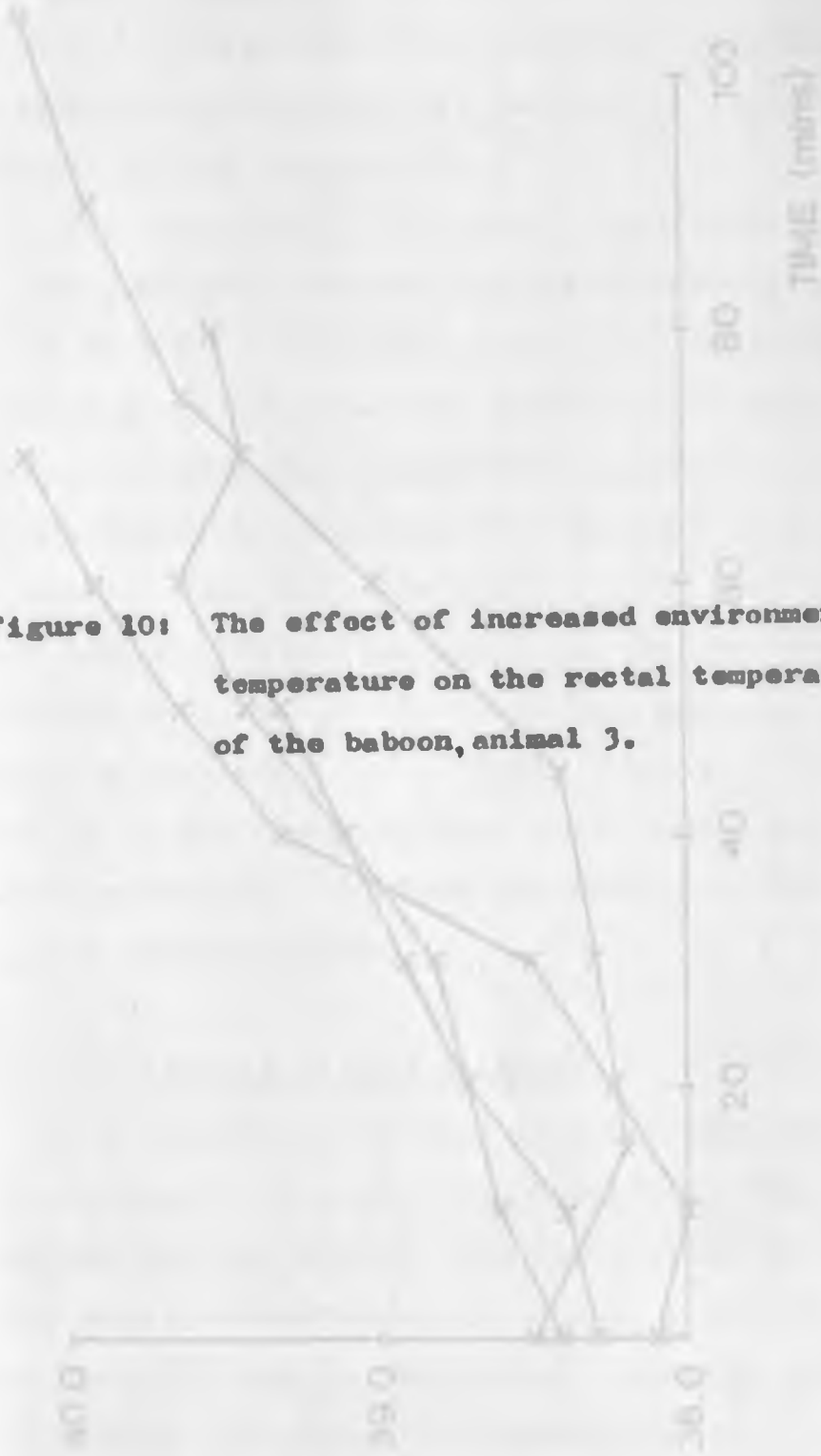
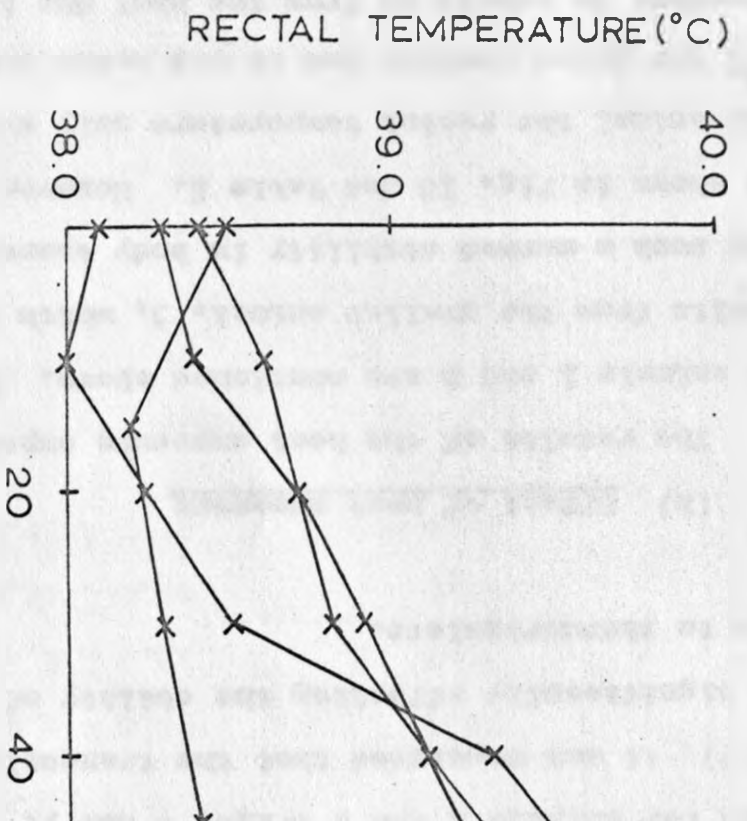
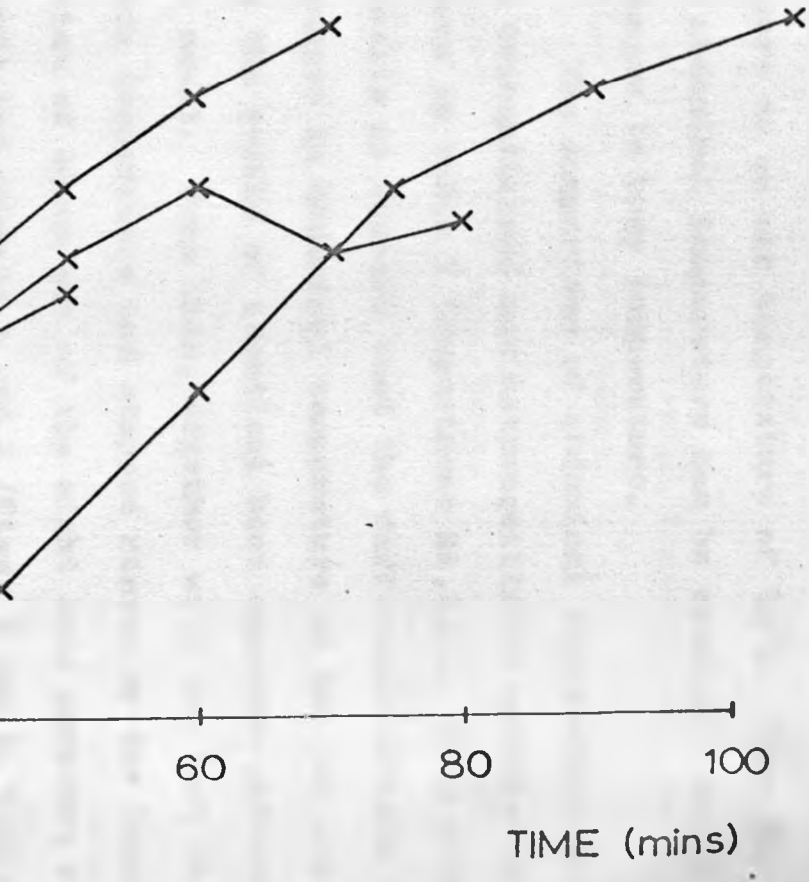


Figure 10: The effect of increased environmental temperature on the rectal temperature of the baboon, animal 3.


RECTAL TEMPERATURE (°C)
38.0
39.0
40.0
20
40


60
80
100
TIME (mins)

changes in rectal and abdominal temperature on exposure to an air temperature of 40°C. Thus changes in abdominal temperature can be used as a measure of changes in body temperature.

The comparison of abdominal temperature changes in tranquillised and untranquillised animals is also shown in Table 5 (Experiment 2A,B,C). From these results it appears that the difference between the changes in abdominal temperature in the two states, as the result of identical heat exposure patterns, is small. From this, together with the fact that body temperature had stopped rising by the termination of seven out of the eight heat exposure experiments for animals 1 and 2 (Figs. 8 and 9, Table 6 and 7), it was concluded that the tranquilliser was not significantly affecting the ability of the animals to thermoregulate.

(b) Effect of heat exposure

The results of the heat exposure experiments for animals 1 and 2 are mentioned above. The results from the smaller animal, 3, which did not show such a marked stability in body temperature, are shown in Fig. 10 and Table 8. However, even in this animal the rectal temperature only went above 40°C for brief periods and it was never felt necessary to remove it from the heat due to hyperthermia.

TABLE 9

The effect of environmental temperature on respiration rate in the baboon.

Animal No.	Environ. Temp. (°C)	Respiration Rate (no./min)
1	18.0	19.8 ± 2.3 S.D.
"	40.0	35.7 ± 10.5 S.D.
2	18.0	21.1 ± 5.7 S.D.
"	40.0	28.9 ± 6.1 S.D.
3	18.0	26.0 ± 2.9 S.D.
"	35.0	33.7 ± 4.4 S.D.
"	40.0	46.1 ± 10.7 S.D.
"	45.0	55.7 ± 17.5 S.D.

2. Respiration rate changes

The respiration rates of the three baboons at various environmental temperatures are shown in table 9.

For baboon 3 the increases were significant as follows:-

Between 18°C and 35°C increase significant at the 5.0% level.

Between 35°C and 40°C increase significant at the 0.1% level.

Between 40°C and 45°C increase significant at the 1.0% level.

For baboons 1 and 2 the increase in respiration rate from low ambient temperatures to 40°C was significant at the 0.1% and 1.0% levels respectively. The respiration rate was normally elevated within ten to fifteen minutes of commencement of exposure to the higher environmental temperature, after which it remained relatively stable as long as no further increase in temperature occurred. The maximum respiration rates recorded at 40°C were 66 times/min, 40 times/min, and 75 times/min in baboons 1, 2 and 3 respectively.

The correlation coefficient of respiration rate with rectal temperature for these animals is significant in baboon 1 at the 1.0% level (r=0.493) and in baboon 2 at the 0.1% level (r=0.616). In baboon 3 the correlation coefficient was 0.300,

TABLE 10

The effect of an environmental temperature of 35°C on cutaneous moisture loss in the baboon.

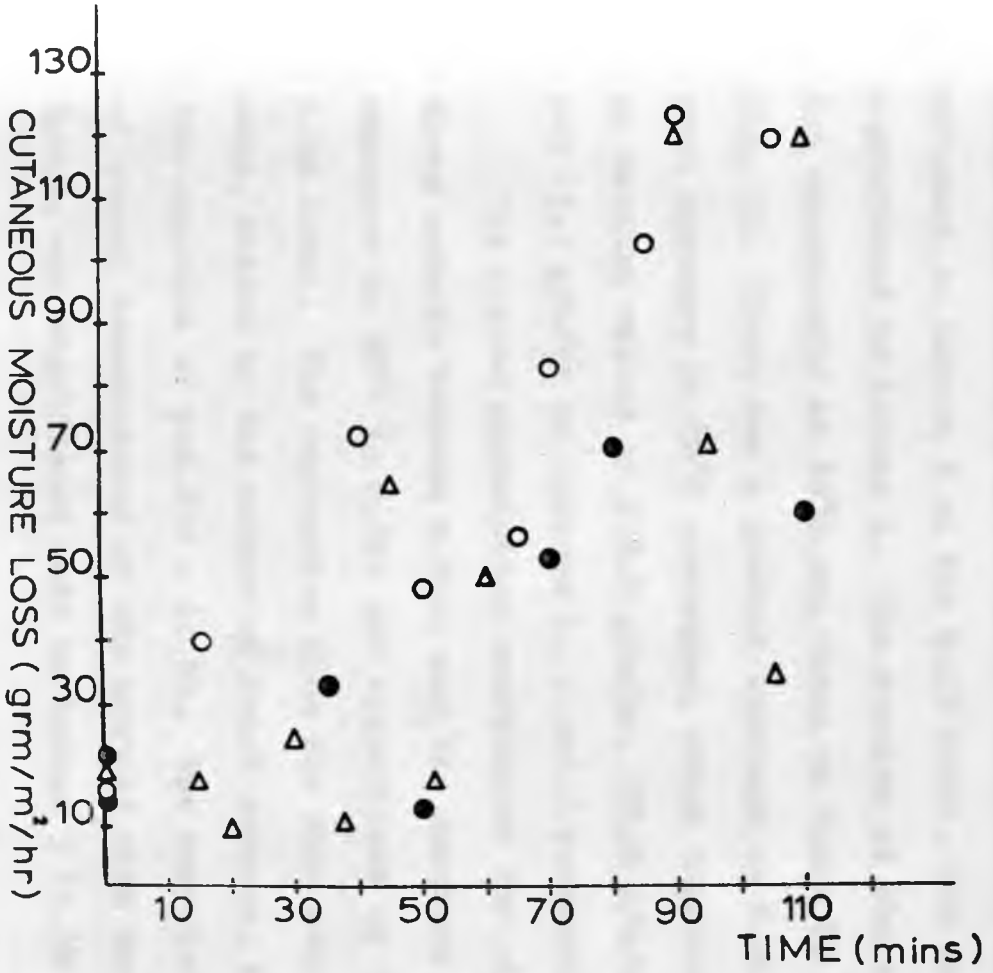
Animal No.	Environmental Temp. (°C)	C.M.L. ($g/m^2/hr$)
1	20.5	19.3
"	20.5	18.1
		18.7 (± 0.8 S.D.)
"	35.0	42.9
"	35.0	50.7
"	35.0	53.9
		49.2 (± 5.7 S.D.)
3	22.0	12.3
"	22.0	20.5
"	22.0	19.9
		16.0 (± 3.8 S.D.)
"	35.0	22.7
"	35.0	55.8
"	35.0	30.6
		36.4 (± 17.3 S.D.)

TABLE II

The effect of exposure to an environmental temperature of 40°C cutaneous moisture loss in the baboon. (The animal was placed in the climatic chamber at time 0 mins.)

Duration of Exposure (Mins)	C.M.L. ($g/m^2/hr$) Baboon I	Baboon 2	Baboon 3
15	17.1	39.4	
20	8.9		
25	22.7		
30	24.1		
35	30.6		32.1
38	10.4		
40		71.4	
45	63.5		
50		47.1	11.6
52	16.8		
60	48.6		
65		55.3	
70	90.3	81.1	50.5
80			69.2
85		101.0	
90	119.0	121.2	
95	69.6		
105		116.8	
110	117.0		58.7

Figure 11: The effect of an environmental temperature of 40°C on the Cutaneous Moisture Loss of the baboon.


CUTANEOUS MOISTURE LOSS (grm/m²/hr)
130
110
90
70
50
30
10
10
30
50
70
90
110
TIME (mins)

which is not significant.

3. Cutaneous moisture loss changes

Table 10 shows the C.M.L. recorded when baboons 1 and 3 were exposed to an environmental temperature of 35°C. The difference of this C.M.L. from the value recorded at a low ambient temperature is significant in baboon 3 at the 0.1% level, but is not significant in baboon 1. The results of the remaining experiments at 40°C are shown in Table 11 and fig. 11. There was a gradual increase in C.M.L. once exposure to 40°C commenced, which increased to maximum values of 119.0 g/m/hr, 121.2 g/m/hr, and 69.3 g/m/hr in baboons 1, 2 and 3 respectively.

The pooled correlation coefficient for all three animals between C.M.L. and the duration of exposure to 40°C is 0.692 and significant at the 0.1% level. The regression line for this relationship, fitted by the method of least squares, has the equation of $y=0.67x + 11.72$. The correlation of rectal temperatures of the animals with the C.M.L. was significant only in baboon 3 (1.0% level)

4. Pharmacological stimulation

(a) Intradermal

Another female baboon was used in this part of the investigation, in addition to animals 1, 2 and 3 used previously.

Adrenaline in concentrations from 10^{-7}g/cc to 10^{-3}g/cc failed to produce any sweat gland activity. Acetylcholine solutions did, however, have a considerable effect in each of the four animals. In two of them the lowest concentration used, 10^{-8}g/cc produced sweating over the injection weal. In the other two animals a concentration of 10^{-6}g/cc was the lowest effective concentration. In each animal the 10^{-3}g/cc solution caused sweating in the area adjacent to the weal, as well as over the weal itself. This was blocked if a 10^{-3}g/cc solution of Atropine was first injected into the same area. The other cholinergic drugs used, pilocarpine and carbacol, also stimulated the sweat glands, though their relative potency varied between animals.

(b) Systemic

Adrenaline was given at 5µg/kg and again failed to stimulate the sweat glands.

In two of the animals pilocarpine or carbacol given at a dosage of 5µg/kg produced very slight sweating, detected by the starch and iodine method, though this response was not sufficient to be detected by the ventilated capsule method.

These drugs always caused salivation in the animals and occasionally muscle spasms.

(c) Atropine and sweating due to heat exposure

Local injections of 0.1cc of a 10^{-3}g/cc

TABLE 12

The effect of atropine during exposure to 40°C on C.M.L., respiration rate and rectal temperature in the baboon. (The animal was placed in the climatic chamber at time 0 mins.) (Atropine in Experiment I (0.15 mg/kg) at 100 mins, in Experiment 2 (0.20 mg/kg) at 125 mins.)

Time (mins)	Resp. Rate (no/min)		Rectal Temp. (°C)		C.M.L. ($g/m^2/hr$)	
	Expt. I	Expt. 2	Expt. I	Expt. 2	Expt. I	Expt. 2
5	25		38.3			
10		30		39.3		
15					39.4	
20	21.5	30	38.7	39.3		
30		25		39.5		
35	25		38.9		71.4	12.5
40						
45	25	29	38.8	40.0		
55		32		40.1		
60	23		39.0			
65		30		40.0	81.1	13.7
75	31	37	38.7	39.9		
85		28		39.9	101.0	
90	25		38.5			
95	20	32	38.3	40.0		69.6
100	25		38.4			
105	32	37	38.4	40.0	12.5	
110	28.5		38.5			117.0
115	27		38.5			
120	28.5		38.7		11.1	
125	24	40	38.8	40.0		
130	23	40	38.9	39.9		
135		55		40.0	11.6	46.0
140				40.0		
145				40.1		
150		66		40.1		29.1

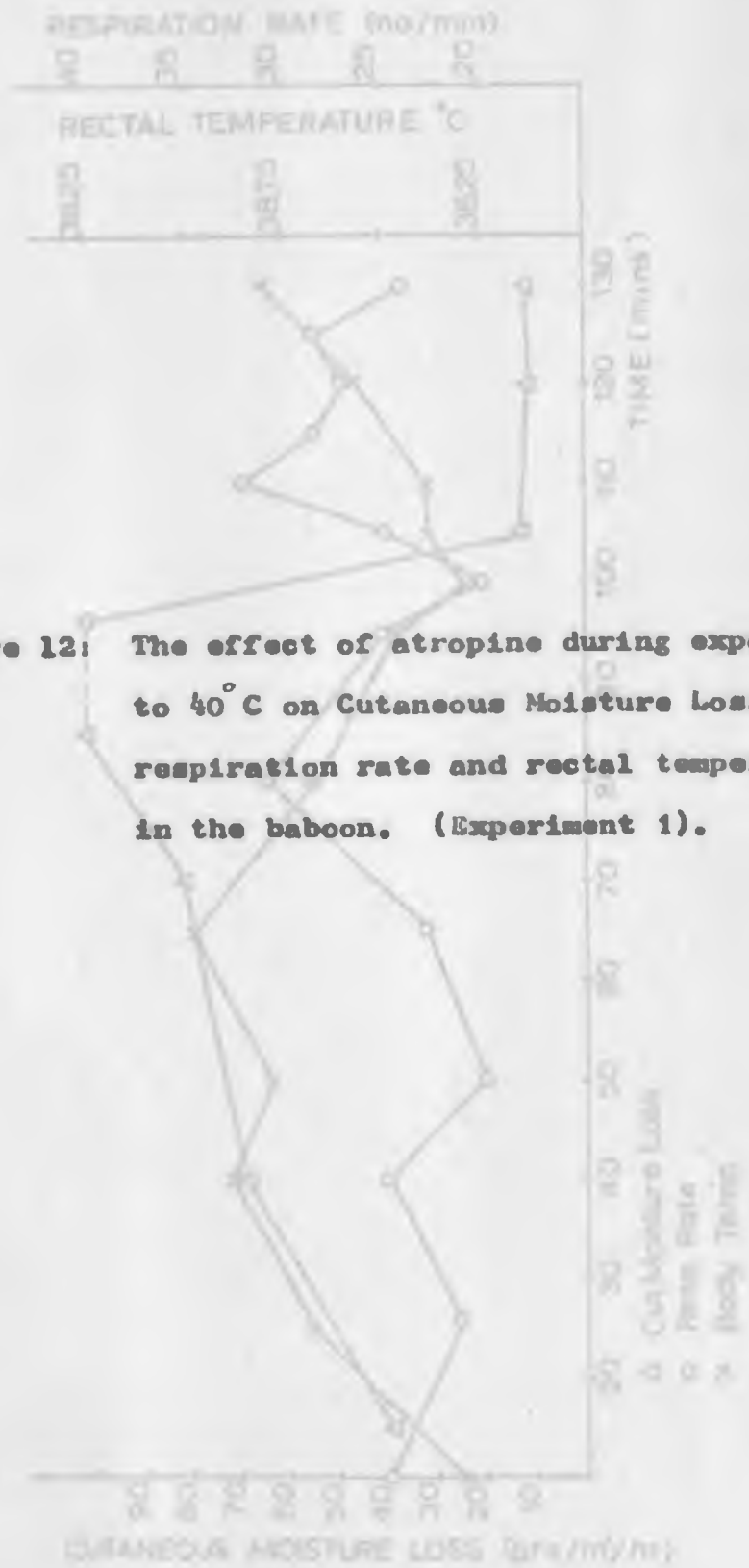


Figure 12: The effect of atropine during exposure to 40°C on Cutaneous Moisture Loss, respiration rate and rectal temperature in the baboon. (Experiment 1).

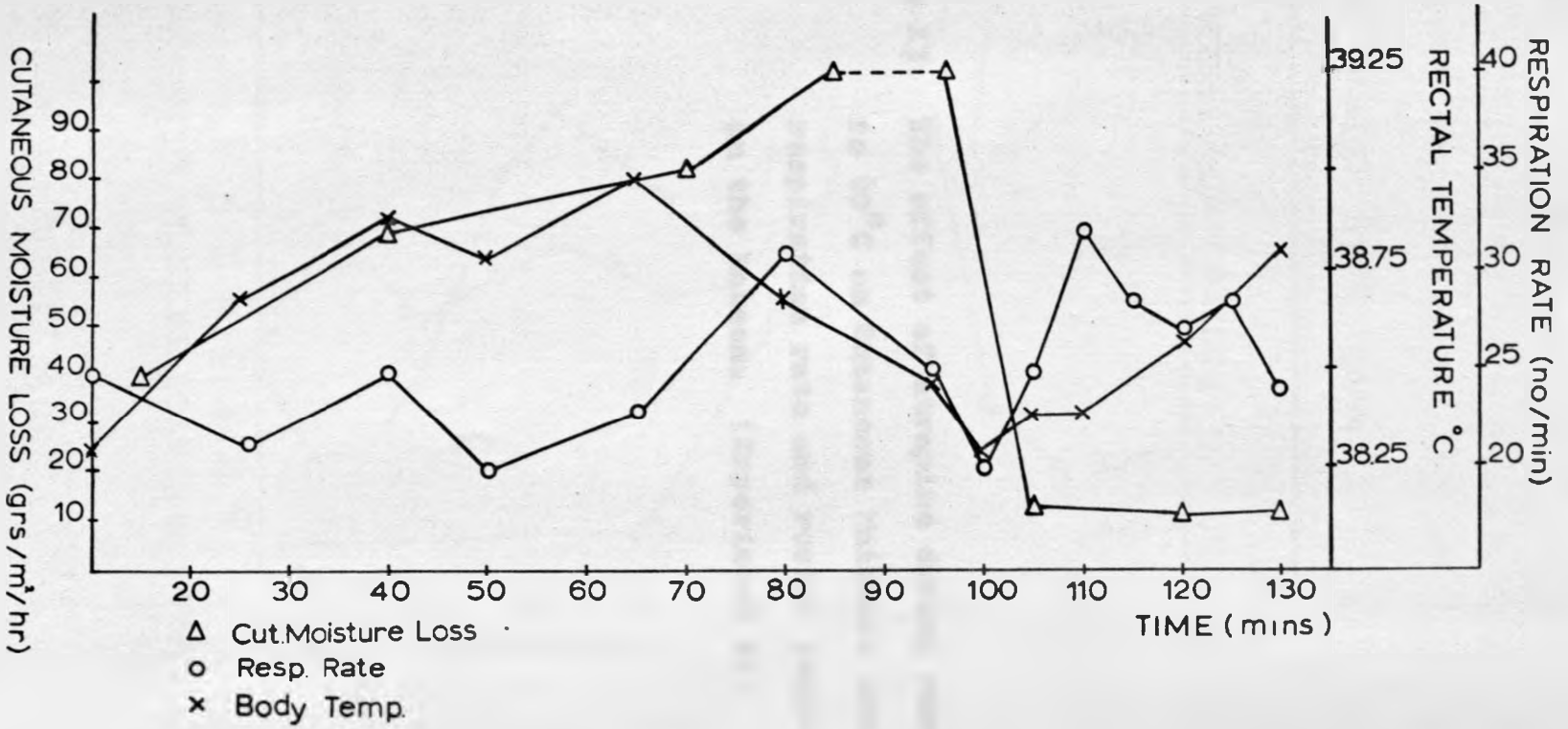
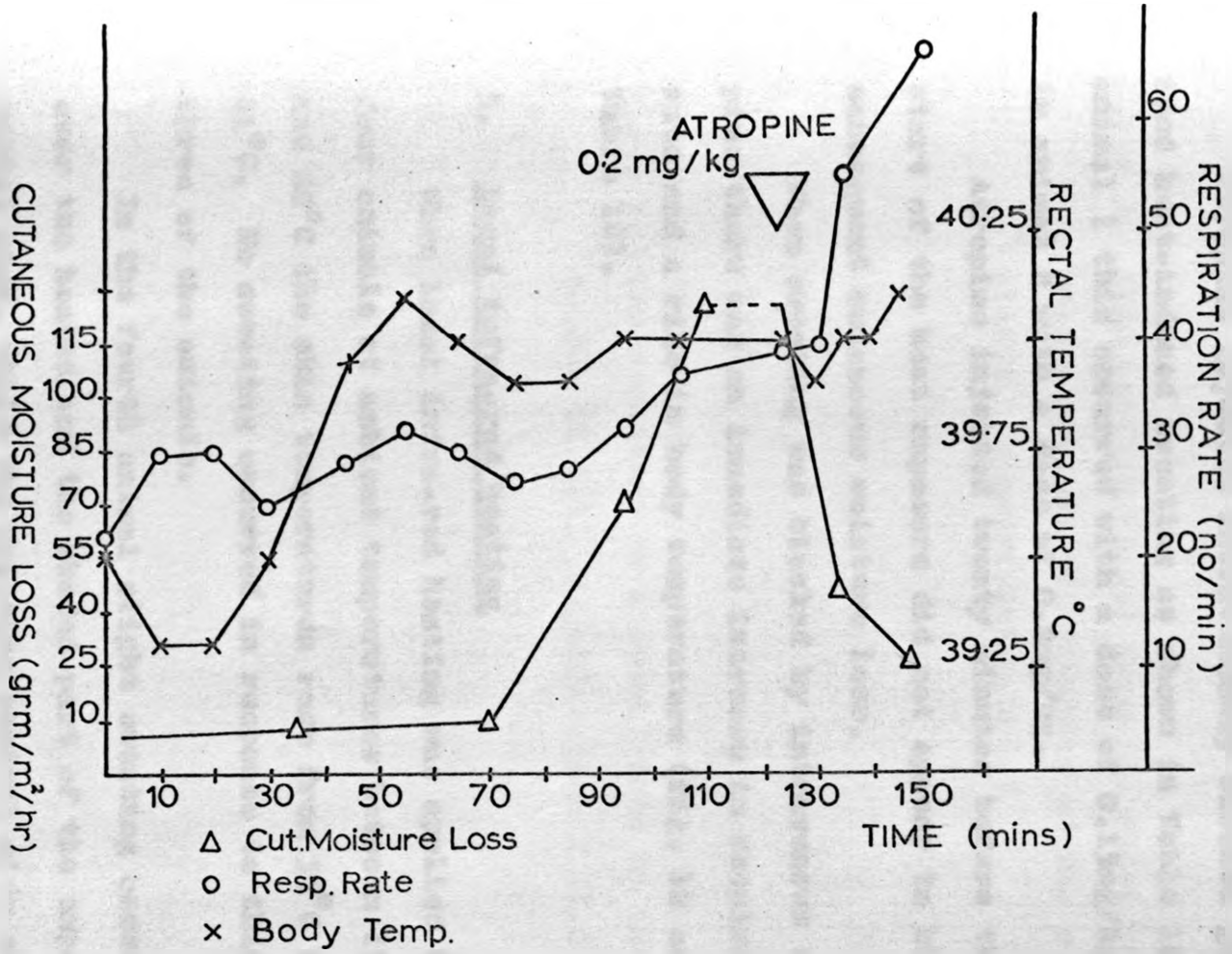

CUTANEOUS MOISTURE LOSS (grs/m²/hr)
90
80
70
60
50
40
30
20
10
20
30
40
50
60
70
80
90
100
110
120
130
TIME (mins)
39.25
38.75
38.25
RECTAL TEMPERATURE °C
40
35
30
25
20
RESPIRATION RATE (no/min)
Δ Cut.Moisture Loss
o Resp. Rate
× Body Temp.

Figure 13: The effect of atropine during exposure to 40°C on Cutaneous Moisture Loss, respiration rate and rectal temperature in the baboon. (Experiment 2).


ATROPINE
0·2 mg/kg
CUTANEOUS MOISTURE LOSS (grm/m²/hr)
115
100
85
70
55
40
25
10
RECTAL TEMPERATURE °C
40·25
39·75
39·25
RESPIRATION RATE (no/min)
60
50
40
30
20
10
10
30
50
70
90
110
130
150
TIME (mins)
Δ Cut.Moisture Loss
o Resp. Rate
× Body Temp.

solution of Atropine caused reduction or the complete blockage of any heat-induced sweat gland activity in the injection area.

Atropine injected intravenously blocked generalised heat-induced sweating as shown in Table 12. In animal 1 this occurred with a dose of 0.15mg/kg and in animal 2 with a dose of 0.2mg/kg.

Atropine injected twenty minutes before the start of the heat exposure did not appear to block subsequent cutaneous moisture loss.

When sweating was blocked by intravenous atropine there was an immediate increase in respiration rate and a rise in body temperature (fig. 12 and 13, Table 12).

5. Local infra-red heating

When local infra-red heating was applied to four animals at ambient temperatures between 17°C and 20°C the skin temperatures rose from 34°C to 41°C. No sweating occurred in response to this in three of the animals.

In the fourth animal slight sweating occurred over the heated and the shaded part of the experimental side, while the opposite control side showed no response.

6. Skin sample

On completion of the above work two small skin

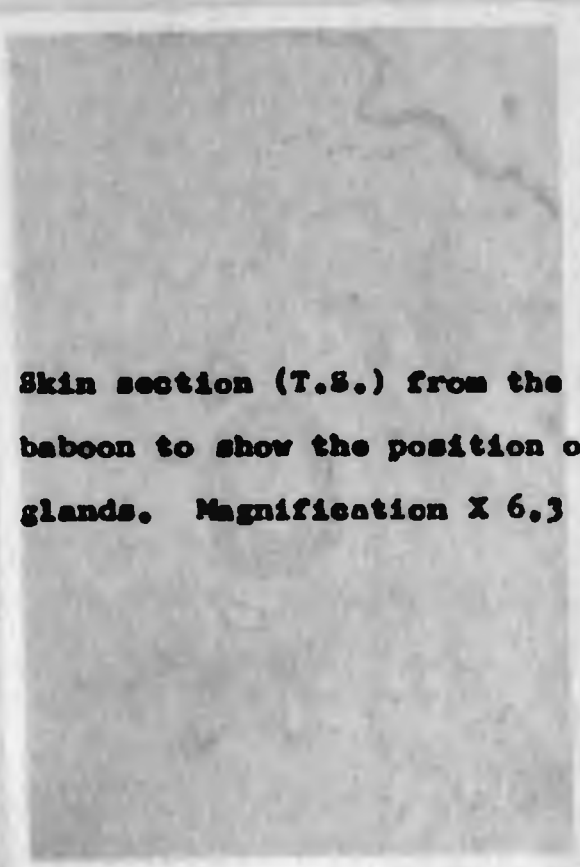
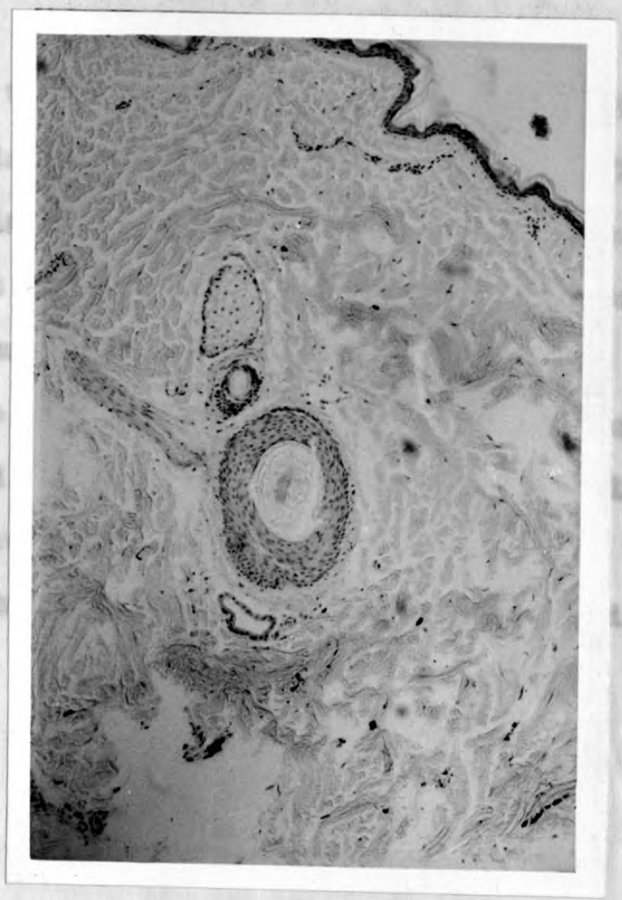
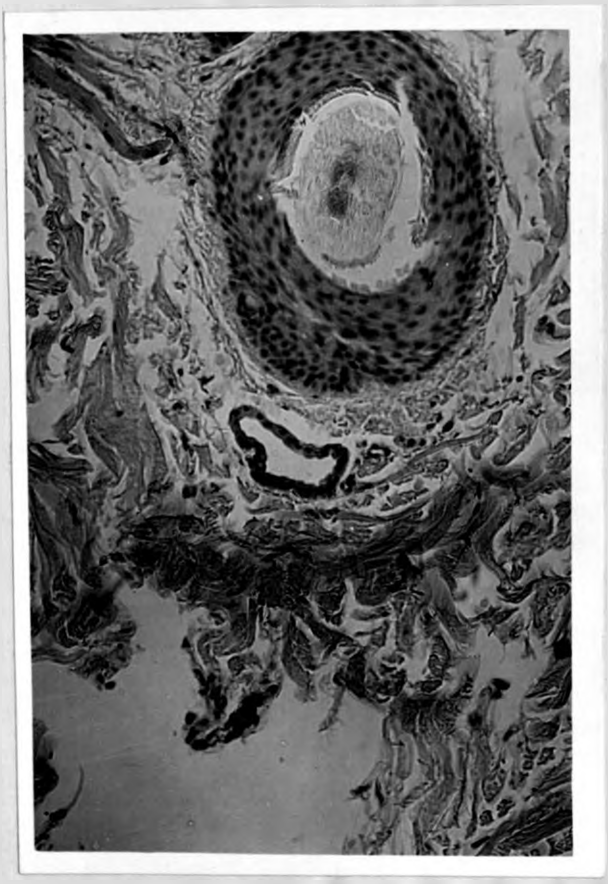


Plate 3: Skin section (T.S.) from the chest of the baboon to show the position of the sweat glands. Magnification X 6.3

Plate 4: The structure of a sweat gland in the baboon (T.S.). Magnification X 16.0





samples were taken from the experimental area. The sweat glands were observed to be sparsely distributed but in those present the lumen had no convulutions but was sack-like and lined with cuboidal epitheium. No myoepithelial cells were seen.

The sweat glands seen in plates 3 and 4 are situated adjacent to both a sebaceous gland and the pilocrector muscle of a hair follicle. The opening of the duct of the sweat gland was not located.

TABLE 13

The effect of an environmental temperature of 40°C on the rectal temperature of the chimpanzee. (The animal was placed in the climatic chamber at time 0 mins).

Duration of Exposure (mins)	Rectal Temperature (°C) Expt. 1	Expt. 2	Expt. 3
0	37.4	35.1	35.2
10	37.5	35.7	35.2
20	-	35.7	35.6
30	38.2	36.2	-
40	38.4	36.5	36.2
50	38.5	37.1	-
60	38.5	37.6	36.7
70	38.5	38.3	37.1
80	38.6	38.5	-
90	38.6	-	37.6
100	38.8	38.7	-
110	38.8	38.8	38.1
120	-	39.1	-
130	-	38.9	38.6
140	-	39.0	38.8
150	-	39.1	-
170	-	-	39.1
190	-	-	39.3
200	-	-	39.4
210	-	-	39.5

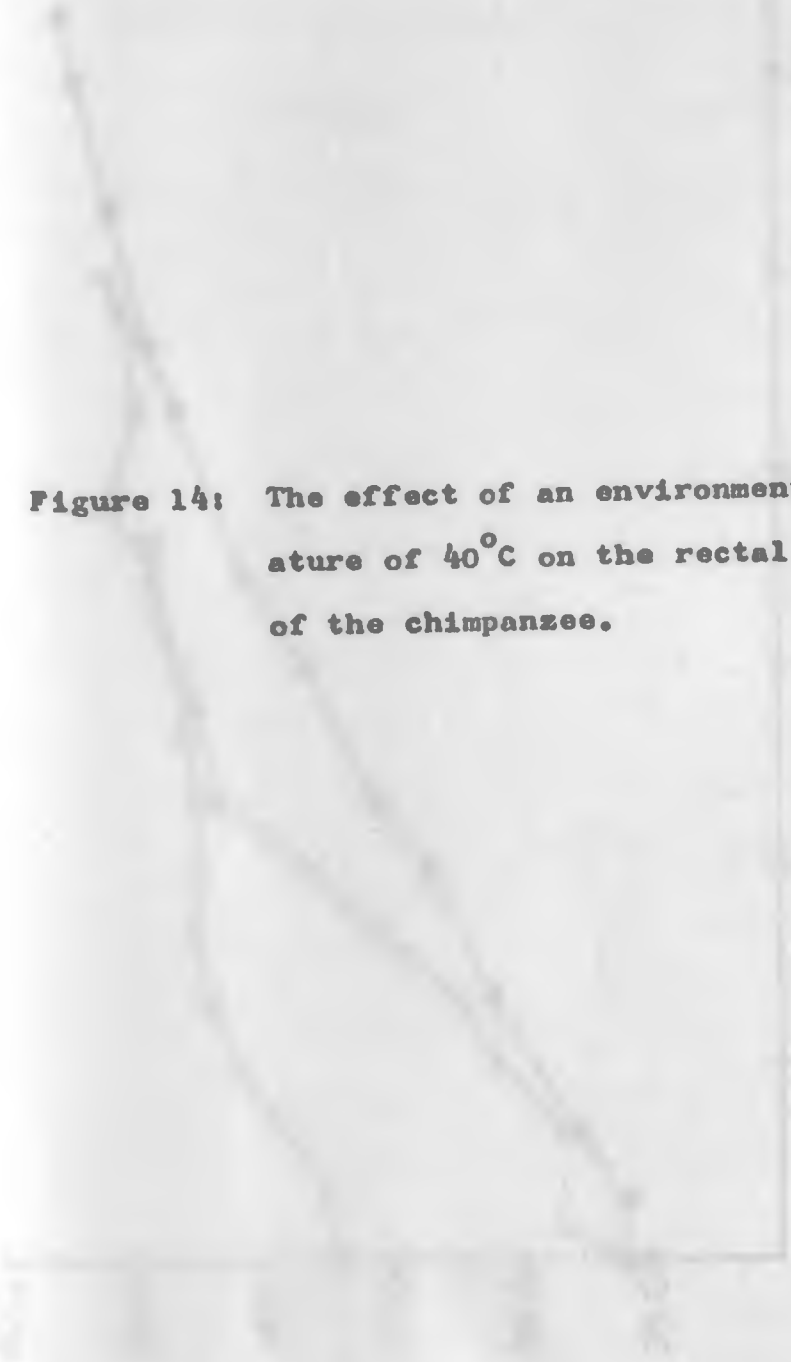
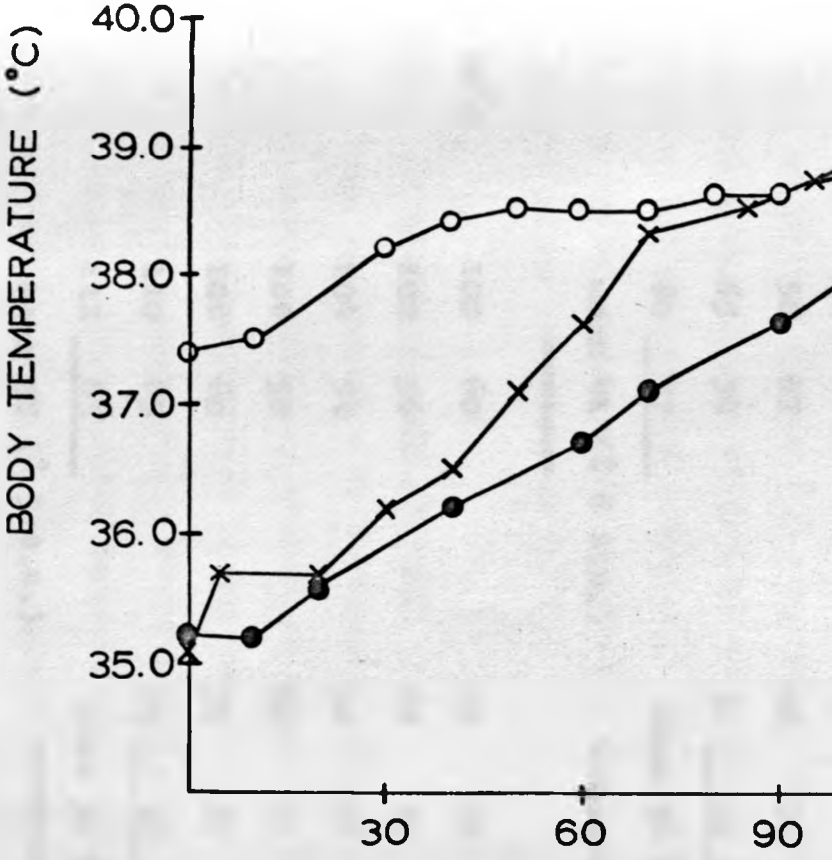


Figure 14: The effect of an environmental temperature of 40°C on the rectal temperature of the chimpanzee.


BODY TEMPERATURE (°C)
40.0
39.0
38.0
37.0
36.0
35.0
30
60
90

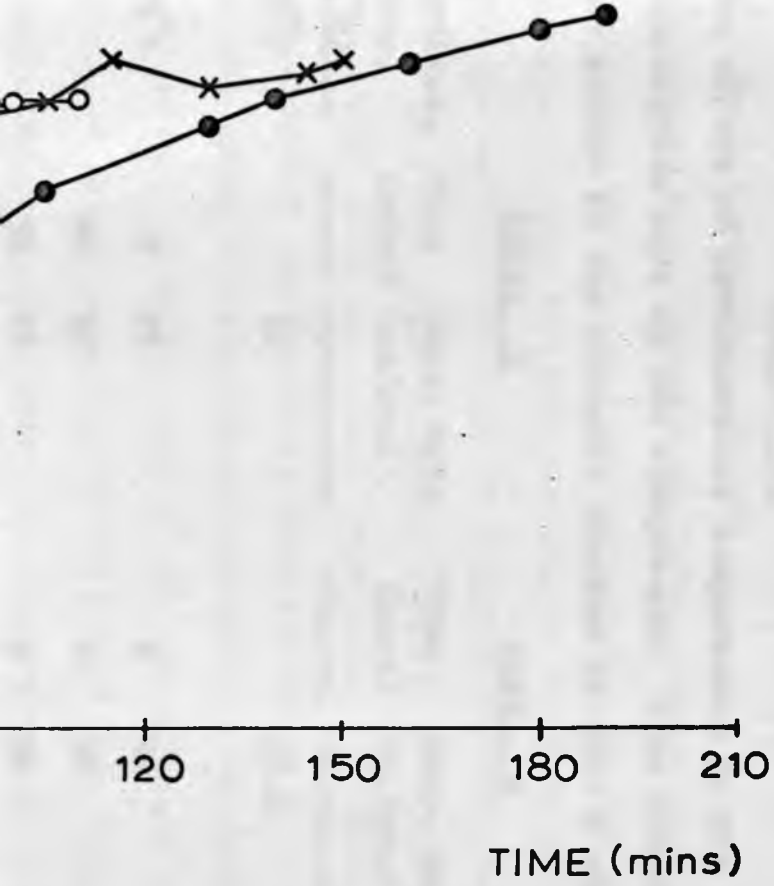

120
150
180
210
TIME (mins)

TABLE 14

The effect of environmental temperature on the respiration rate of the chimpanzee. (The animal was placed in the climatic chamber at time 0 mins).

	Expt. I		Expt. 2	
Environ. Temp. (°C)	Time (mins)	Resp. Rate (no/min)	Time (mins)	Resp. Rate (no/min)
18°C		30		29.5
35°C	5	37	2	43
	20	45	4	36
	35	46	6	36
	50	47	20	34
	65	34	30	29
	80	37	mean 36 (± 5 S.D.)	
	mean 41 (± 6 S.D.)			
40°C	100	60	37	34
	102	56	45	31
	104	54	50	38
	106	50	60	41
	108	60	70	37
	110	63	80	33
	112	65	mean 36 (± 4 S.D.)	
	mean 58 (± 5 S.D.)			

The effect of heat exposure on the chimpanzee

1. Body temperature changes

These were recorded in three experiments in which the animals were tranquillised. On two of these occasions the animals became slightly hypothermic between the time of tranquillisation and entry into the large climatic chamber (Fig. 14 and Table 13). For one animal this meant a fifteen minute exposure to an ambient temperature of 16°C and in the other a sixty minute exposure to an ambient temperature of 17°C. When those animals were exposed to an invironmental temperature of 40°C their rectal temperatures rose, stabilising in one individual at 39.1°C (after 135 minutes of exposure) while in the other it was beginning to stabilise at 39.4 (after 180 minutes of exposure.) The third animal had a rectal temperature of 37.4°C which rose to 38.6°C after 90 mins. at 40°C.

2. Respiration rate changes

The respiration rate changes on heat exposure of an untranquillised animal is shown in Table 14. In the first experiment there was a significant (0.1% level) increase in respiration rate on exposure to an environmental temperature of 35°C and a further increase (also significant at the 0.1% level) when this was increased to 40°C. In the second experiment the exposure to 35°C did not cause

TABLE 15

The relationship between the increase in rectal temperature, due to exposure to 40°C and respiration rate in the chimpanzee.

	Expt. 1		Expt. 2		Expt. 3	
Rectal Temp. (°C)	Resp. Rate (no/min)	Time (mins)	Resp. Rate (no/min)	Time (mins)	Resp. Rate (no/min)	Time (mins)
35.2	27	10				
35.6	27	20				
35.7			23	5		
35.7			21	20		
36.2	28	40	27	30		
36.5			27	40		
36.7	30	60				
37.1	32	70	35	50		
37.5					30	10
37.6	40	90	43	60		
38.1	39	105			46	30
38.3			60	70		
38.4					60	40
38.5			50	85	57	50
38.5					48	65
38.6	46	130			92	80
38.6					70	90
38.8	50	140	50	105	76	100
38.9			60	130	60	105
39.0			60	145		
39.1	60	160	60	150		
39.3	50	180				
39.4	46	190				

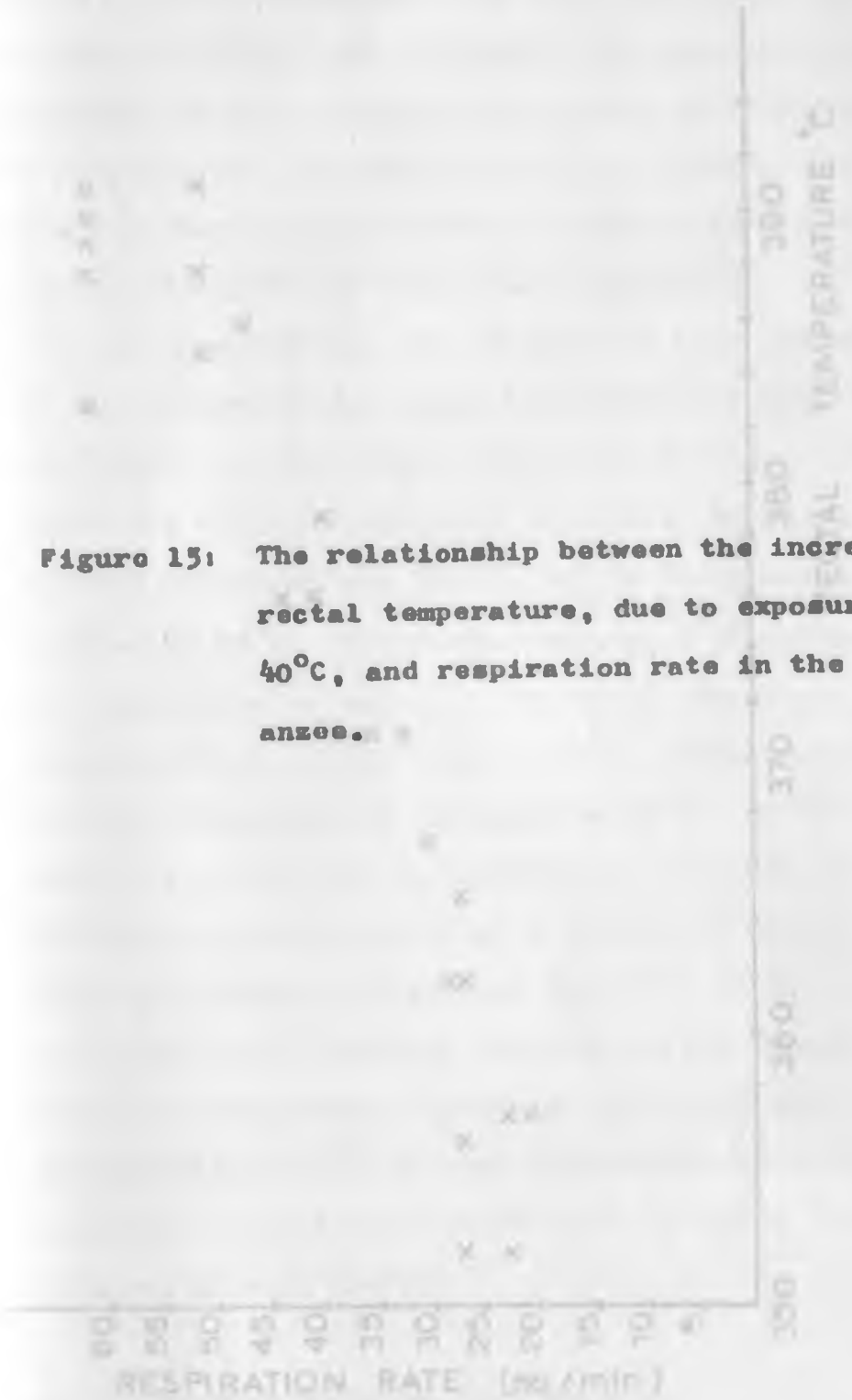
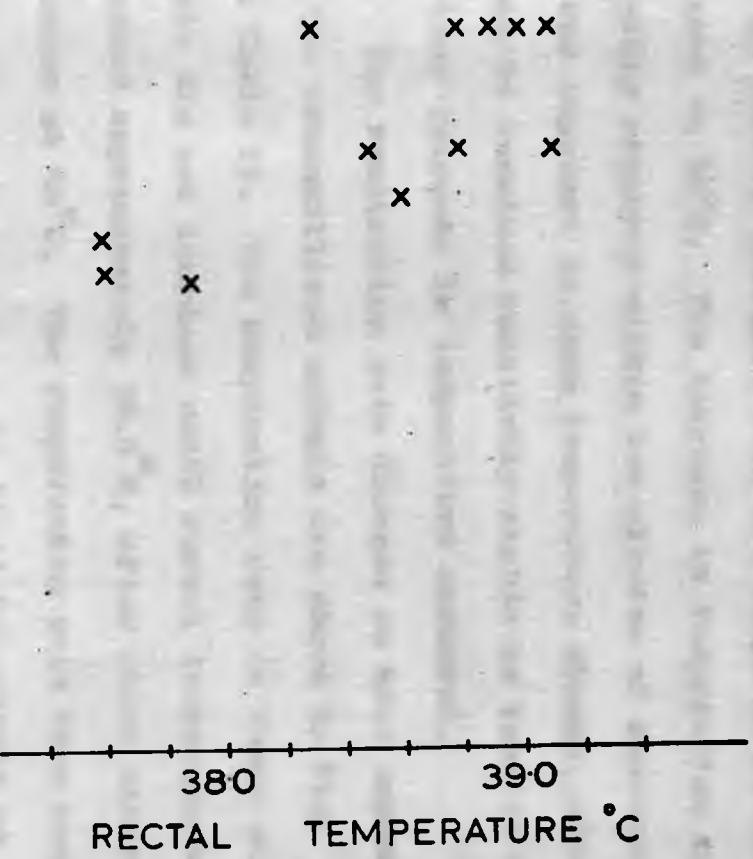


Figure 15: The relationship between the increase in rectal temperature, due to exposure to 40°C, and respiration rate in the chimpanzee.

RESPIRATION RATE (no/min)
60
55
50
45
40
35
30
25
20
15
10
5
35·0
36·0
37·0


38·0
39·0
RECTAL TEMPERATURE °C

a significant increase in respiration rate and there was no further increase when the temperature was increased to 40°C. The increase in respiration rates normally occurred within ten minutes of commencement of the exposure to each temperature change, after which it remained relatively stable as long as no further increase in temperature occurred.

The respiration rate changes on heat exposure of the tranquillised animals are shown in Fig. 15 and Table 15. The respiration rate in these experiments did not increase until rectal temperature had reached approximately 36.6°C, after about sixty minutes at 40°C. The respiration rate then rose to its maximum value about 60 times per minute as body temperature stabilised near 39.0°C. This was after 160 to 180 minutes of exposure to 40°C. In the third experiment the tranquillised animal had a maximum respiration rate of 92 times per minute after 90 minutes of exposure to 40°C. In both animals there was a gradual increase in the respiration rate up to these maximum values and this relationship of rectal temperature and respiration rate had a correlation coefficient of 0.628 (significant at 0.1% level).

3. Cutaneous moisture loss changes

The changes in C.M.L. on heat exposure are shown in figs. 16 and Table 16. The time which

TABLE 16

The relationship between the increase in rectal temperature due to exposure to 40°C and cutaneous moisture loss in the chimpanzee.

	Expt. I		Expt. 2	
Rectal Temp. (°C)	C.M.L. ($g/m^2/hr$)	Time (mins)	C.M.L. ($g/m^2/hr$)	Time (mins)
35.7	12.5	10		
35.9			22.2	30
36.4			23.6	45
36.8	20.5	45		
37.3			21.7	80
38.1			19.3	105
38.4	34.5	80		
38.7	61.2	100		
38.9			32.1	145
39.0	80.0	125		
39.1			34.5	160
39.4			35.7	190

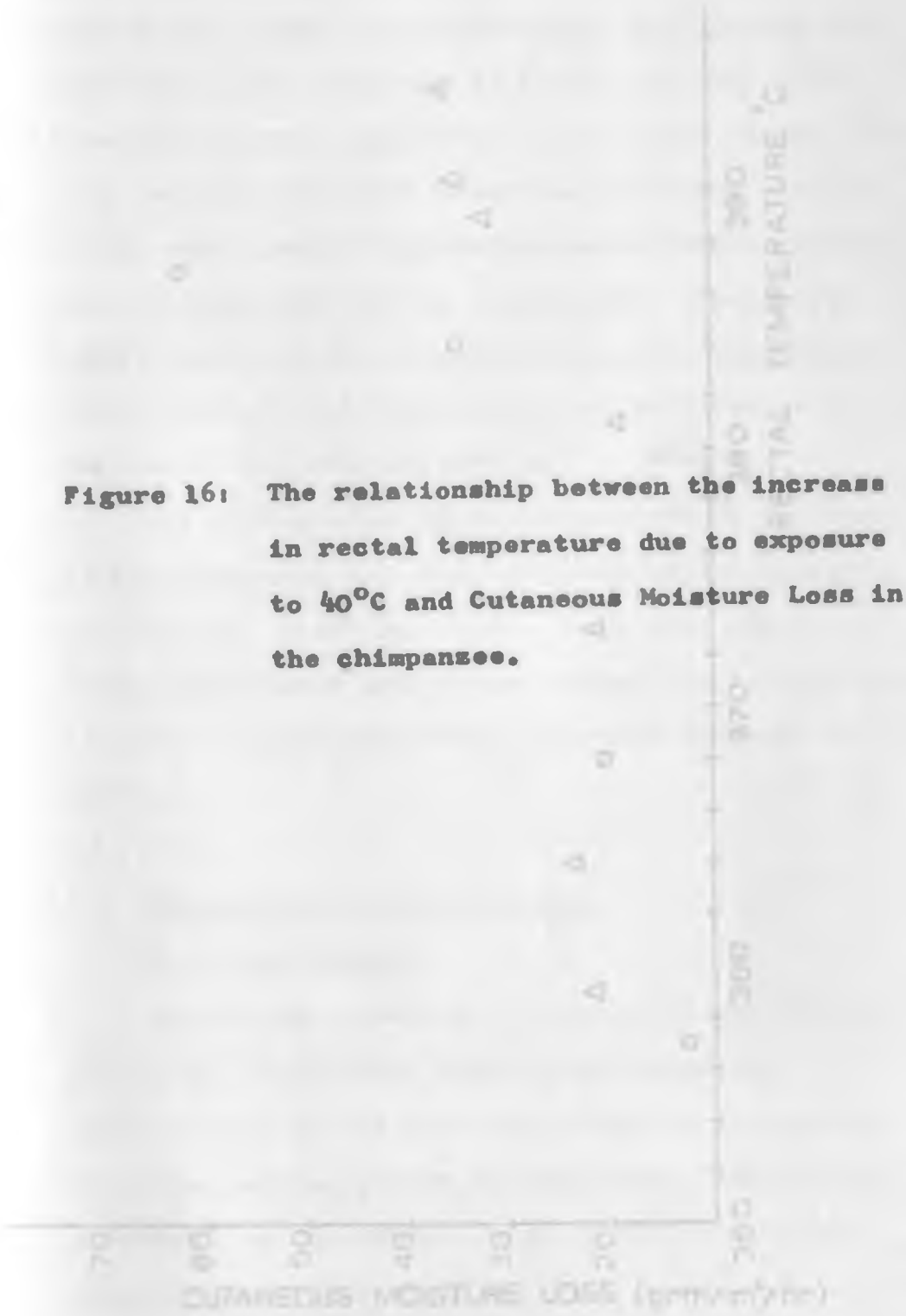
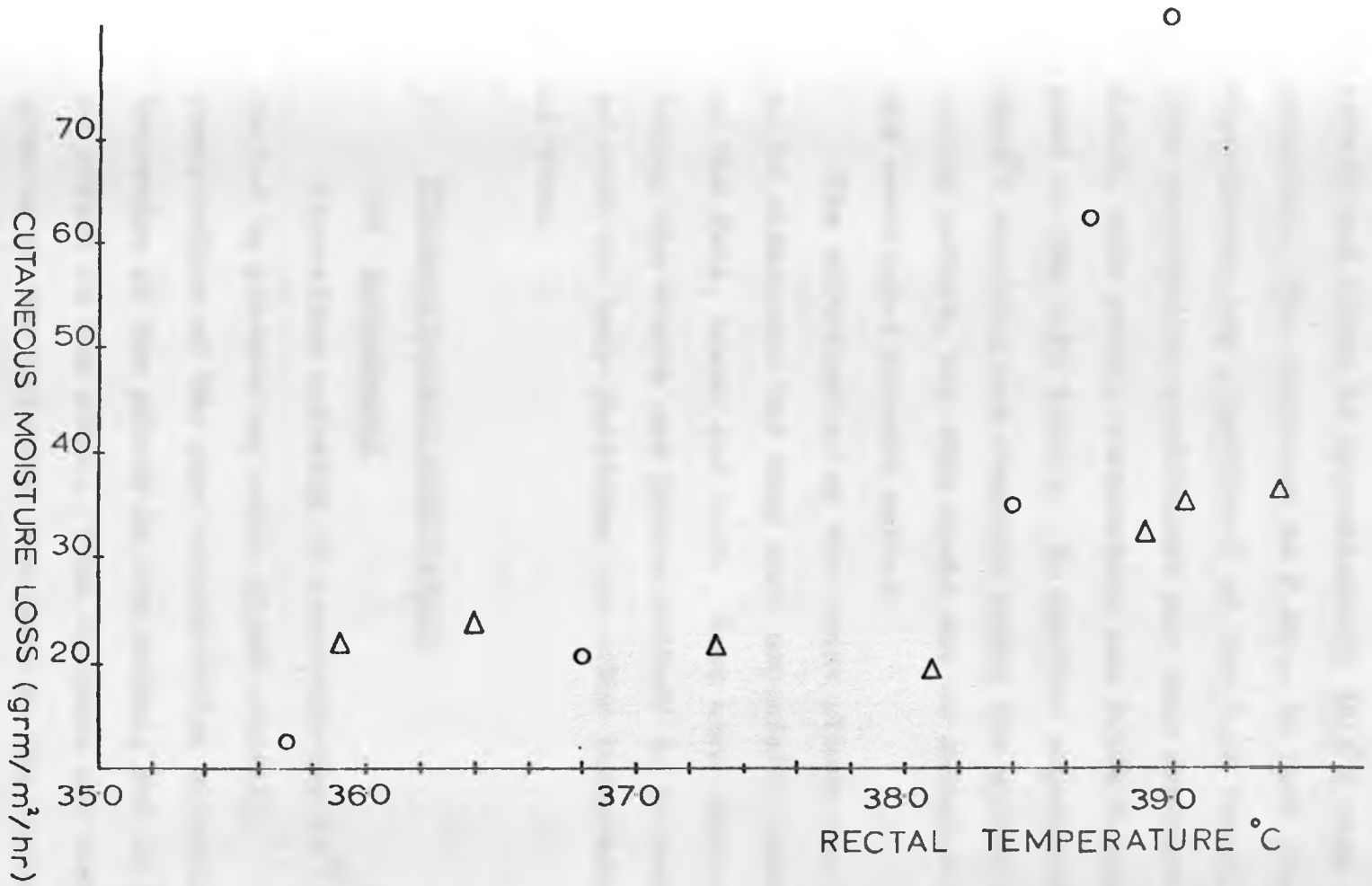


Figure 16: The relationship between the increase in rectal temperature due to exposure to 40°C and Cutaneous Moisture Loss in the chimpanzee.


CUTANEOUS MOISTURE LOSS (grm/m²/hr)
70
60
50
40
30
20
350
360
370
380
390
RECTAL TEMPERATURE °C

elapsed before the onset of sweating is shown in the table, but on both occasions the body temperatures had risen to approximately 38.3°C when this occurred. The increases in C.M.L. in both these experiments are significant at the 0.1% level. The mean correlation coefficient for this increase in C.M.L. with rectal temperature was 0.922 (significant at the 0.1% level). In another experiment at 40.0°C sweating was observed using the starch and iodine method, but this could not be detected by the ventilated capsule method.

The distribution of the sweat glands was found to be widespread but they were especially numerous on the face, hands and feet. Some were observed (using the starch and iodine method) to be associated with the hair follicles and other independent of them.

4. Pharmacological stimulation

(a) Intradermal

Adrenaline solution of concentration 10^{-3} g/cc failed to produce any sweat gland activity. Acetycholine of the same concentration stimulated secretion of the glands in one animal, but it had no effect in the other. This response was much greater if the injection was made in the region of the axilla.

TABLE 17

The effect of atropine during exposure to 40°C on C.M.L., respiration rate and rectal temperature in the chimpanzee. (The animal was put in the climatic chamber at time 0 mins - atropine (0.15 mg/kg) was given after 135 mins.)

Time (mins)	Resp. Rate (no/min)	Rectal Temp. (°C)	C.M.L. ($g/m^2/hr$)
5	23	35.7	
10			12.5
20	21	35.7	
30	27	36.2	
40	27	36.5	
45			20.5
50	35	37.1	
60	43	37.6	
70	60	38.3	
80	50	38.5	34.5
95	40	38.7	
100			61.2
105	50	38.8	
115	50	39.1	
125			80.0
130	60	38.9	
140			21.1
145	60	39.0	
150	60	39.1	

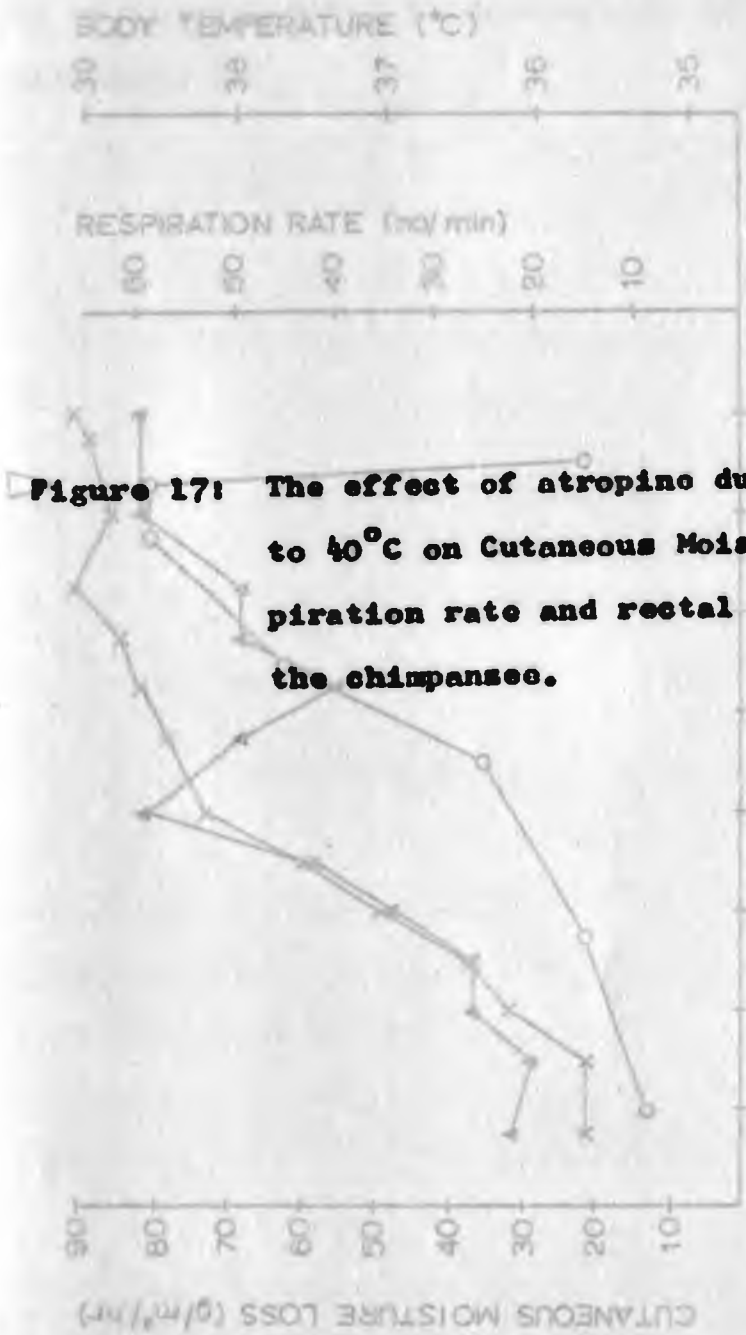
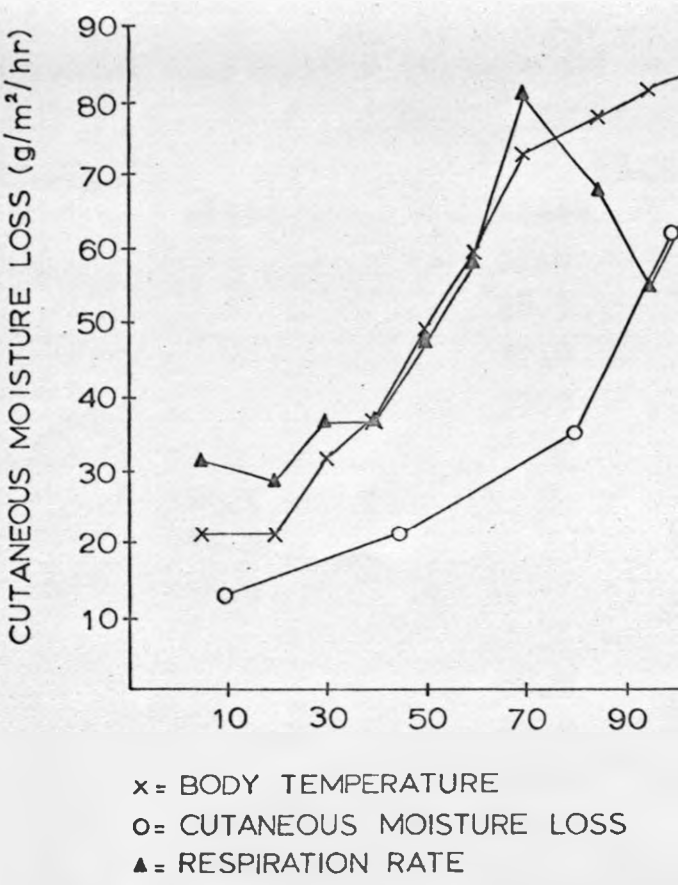


Figure 17: The effect of atropine during exposure to 40°C on Cutaneous Moisture Loss, respiration rate and rectal temperature in the chimpansee.


CUTANEOUS MOISTURE LOSS (g/m²/hr)
90
80
70
60
50
40
30
20
10
10
30
50
70
90
x = BODY TEMPERATURE
o = CUTANEOUS MOISTURE LOSS
▲ = RESPIRATION RATE

ATROPINE 0·15 mg/kg
RESPIRATION RATE (no/min)
60
50
40
30
20
10
BODY TEMPERATURE (°C)
39
38
37
36
35
110
130
150
TIME (hrs)

(b) Systemic

Atropine given at a dose of 0.15 mg/kg caused a decrease in cutaneous moisture loss during exposure to an environmental temperature of 40°C. (Fig. 17 and Table 17).

TABLE 18

The changes in rectal temperature through the day in the black and the white rhinoceros.

Date	Rectal Temperature (°C)					
	07.00 hrs		12.00 hrs.		18.00 hrs.	
	black	white	black	white	black	white
Feb.	36.9		37.8		38.4	
1971	37.2		38.3		38.4	
	37.3		37.8			
	37.3		37.9			
Dec.		36.5		37.1		37.5
1971		36.5		36.7		37.9
		36.9		37.4		37.4
April	37.3		38.8		38.0	
1972	37.0		37.8		38.0	
	37.2		38.0		38.2	
	37.2		38.2			
means	37.2(± 0.1S.D.)	36.6(± 0.2S.D.)	38.1(± 0.3S.D.)	37.1(± 0.3S.D.)	38.2(± 0.2S.D)	37.6(± 0.3S.D.)

The effect of climatic conditions through the day on the white rhinoceros and the black rhinoceros

1. Body temperature changes

Table 18 shows rectal temperatures recorded in the early morning after both species had been experiencing similar, cool environmental temperatures through the night. A comparison between the two species can, therefore, be made at this time. The black rhinoceros has the higher rectal temperature, the difference between the species being significant at the 0.1% level.

In considering changes in rectal temperature through the day, only days during which the animals were experiencing a reasonable heat load are considered, i.e. less than 40% cloud cover and an environmental temperature of, at least, 29°C at mid-day. The rectal temperature on such days recorded at mid-day and 18.00 hrs. in each species is also shown in Table 18.

The changes in body temperature are shown below with their degree of significance:

Black rhinoceros - difference from 08.00 to 12.00 hrs. signif. 0.1% level.

Black rhinoceros - difference from 12.00 to 18.00 hrs. not signif.

White rhinoceros - difference from 08.00 to 12.00 hrs. not signif.

White rhinoceros - difference from 12.00 to 18.00 hrs.

TABLE 19

The changes in respiration rate through the day in the black and the white rhinoceros.

Date	Respiration Rate (no/min) 07.00 hrs.		12.00 hrs.		18.00 hrs.	
	black	white	black	white	black	White
Feb.	28		96		20	
1971	23		86		44	
	20		80			
means	24 (± 4 S.D.)		87 (± 8 S.D.)		32 (± 17 S.D.)	
Dec.		24		26		23
1971		20		26		35
		22		30		
April	20		72		28	
1972	24		42		20	
	24		54		28	
	36		52			
means	26 (± 7 S.D.)	22 (± 2 S.D.)	55 (± 12 S.D.)	27 (± 2 S.D.)	25 (± 5 S.D.)	29 (± 8 S.D.)

TABLE 20

The changes in the respiration rate of the white rhinoceros through the afternoon.

Time (hrs.)	Resp. Rate (no/min)		Comments
	Animal I	Animal 2	
14.30	30	18	animals in the shade and lying down in the enclosure
14.45			animals left enclosure
15.00	50		animals in the direct sun while grazing
15.30	57.5		animal in the shade, but after grazing in the sun for 30 mins.
15.45		60	animal in the direct sun while grazing
16.00	57.5		animal in the direct sun while grazing
16.20	60	55	animal in the direct sun while grazing
18.00	23	30	animals back in the enclosure, solar radiation zero

signif. at 5.0% level.

2. Respiration rate changes

The respiration rates recorded at 07.00 hrs., 12.00 hrs. and 18.00 hrs. are shown in Table 19. The values recorded at 07.00 hrs. were considered as being the basal values for this parameter and these showed no significant difference between the black rhinoceros and the white rhinoceros.

The black rhinoceros always had an increase in respiration rate between 07.00 and 12.00 hrs. which was significant at the 0.1% level. This returned to close to the basal value by 18.00 hrs. This increase in the respiration rate was significantly greater (0.1% level) in the initial experiments during February, 1971, than it was during experiments in April 1972.

The white rhinoceros did not show any significant increase in respiration rate from 07.00 hrs. to 12.00 hrs. At 12.00 hrs. the animals were normally resting in their enclosure in the shade of a few small trees. The animals were followed during the afternoon as they grazed in the open and an increase in respiration rate was then apparent. (Table 20).

3. Cutaneous moisture loss changes

The changes in C.M.L. through the day are

TABLE 21

The changes in C.M.L. through the day in the black and the white rhinoceros.

C.M.L. ($g/m^2/hr$)

Date	07.00 hrs.		12.00 hrs.		18.00 hrs.	
	black	white	black	white	black	white
Feb. 1971	117.5		160.2		132.7	
	108.5		167.0		91.6	
	98.6		153.0			
means	108.2(±9.5S.D.)		160.6(±7.0S.D.)		112.2(±29.1S.D.)	
Dec. 1971		268.0		343.0		332.0
		280.0		345.0		320.6
		293.0		338.0		-
April 1972	407.0		480.4		397.8	
	218.8		362.6		298.3	
	253.9		348.8		266.2	
	269.2		405.9		-	
means	287.2(±82.6S.D.)	280.3(±12.5S.D.)	399.4(±59.2S.D.)	342.0(±3.6S.D.)	320.7(±68.9S.D.)	326.3(±8.0S.D.)

mean of last two experiments

284.3(± 59.0 S.D.)	374.8(± 51.9 S.D.)	323.0(± 48.8 S.D.)

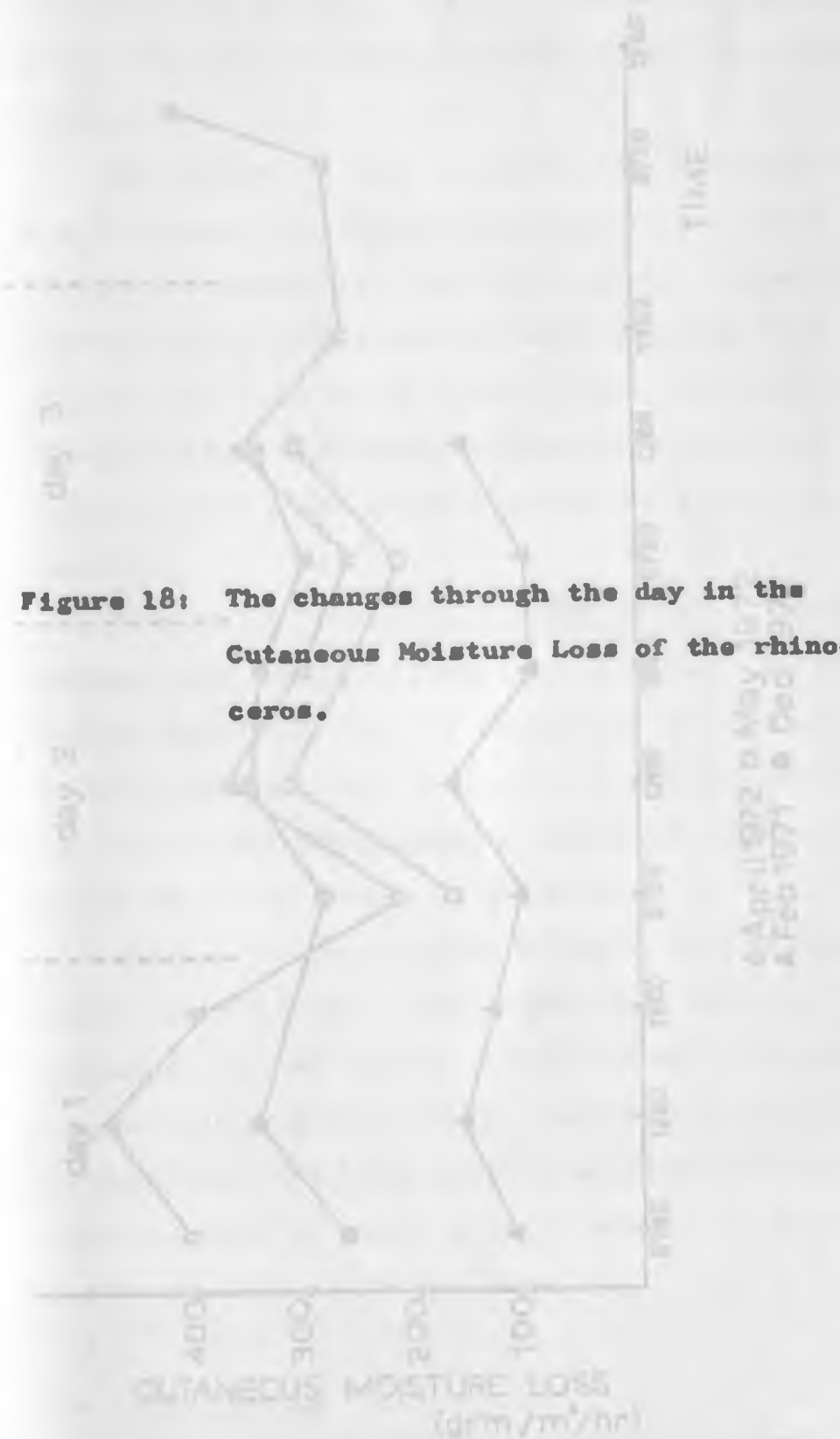
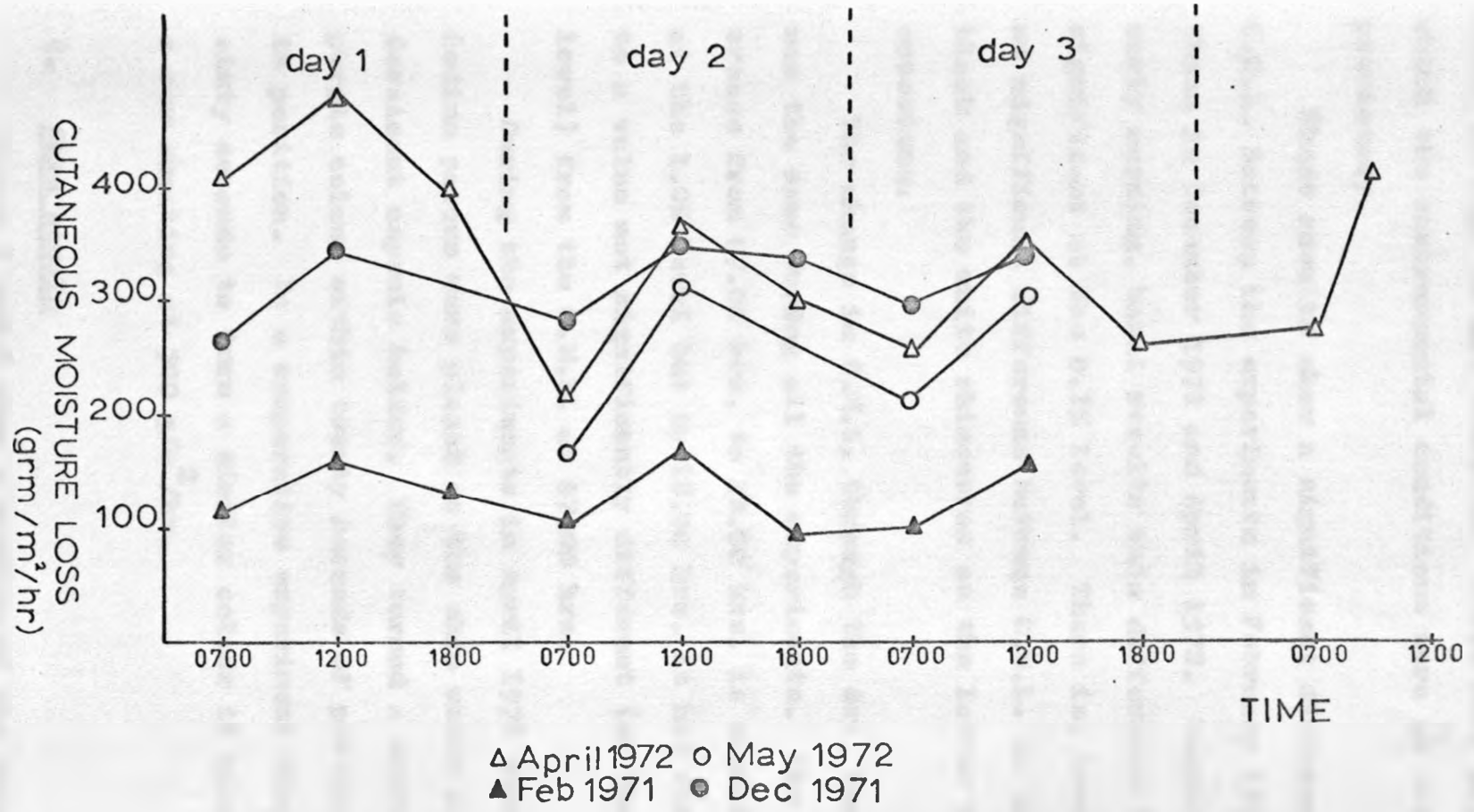


Figure 18: The changes through the day in the Cutaneous Moisture Loss of the rhinoceros.


day 1
day 2
day 3
CUTANEOUS MOISTURE LOSS (grm/m²/hr)
400
300
200
100
0700
1200
1800
0700
1200
1800
0700
1200
1800
0700
1200
TIME
△ April 1972 ○ May 1972
▲ Feb 1971 ● Dec 1971

shown in fig. 18 and Table 21. These were days on which the environmental conditions were as defined previously.

These results show a significant difference in C.M.L. between the experiments in February 1971 and those in December 1971 and April 1972. Comparing early morning, basal results this difference is significant at the 0.1% level. There is, however, no significant difference between C.M.L. in the black and the white rhinoceros on the latter two occasions.

The change in C.M.L. through the day, however, was the same during all the experiments. The increase from 07.00 hrs. to 12.00 hrs. is significant at the 1.0% level but by 18.00 hrs. it had returned to a value not significantly different (at the 5% level) from the C.M.L. at 07.00 hrs.

During the experiments in April 1972 starch iodine papers were placed on the skin under a dessicant capsule holder. They turned a uniform purple colour within twenty seconds of putting them in position. In a comparative experiment they took sixty seconds to turn a similar colour if placed on a cow sweating at 300 $g/m^2/hr$.

4. Skin sample

Plates 5 and 6 show a section of the skin taken from the back of the rhinoceros. Many large

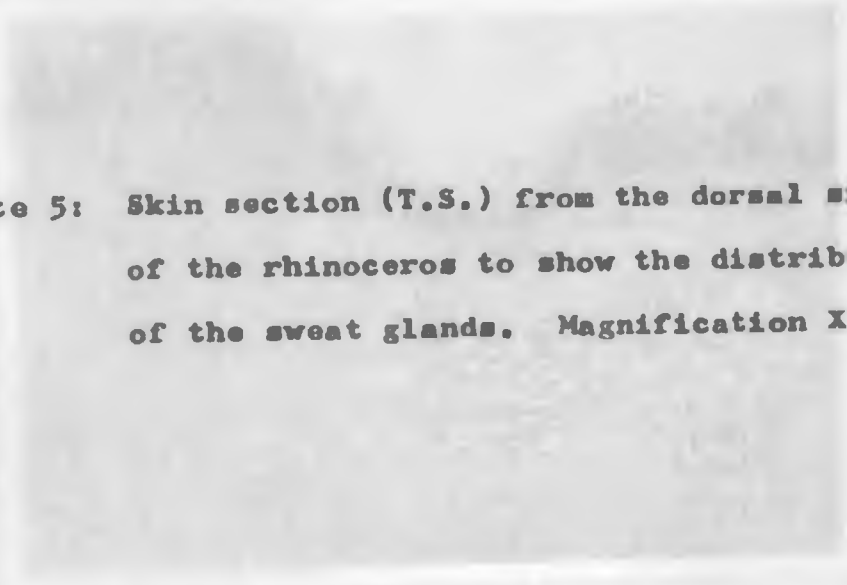
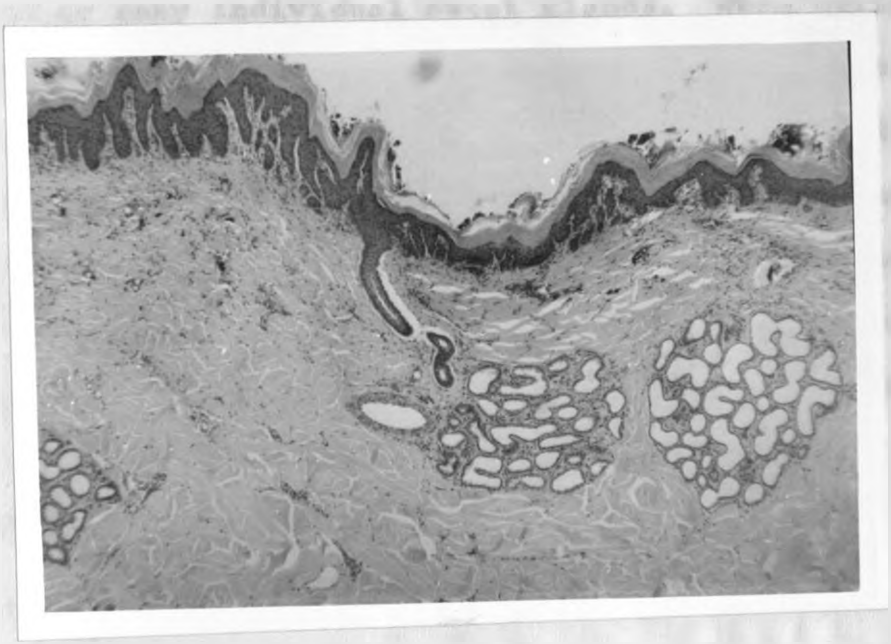
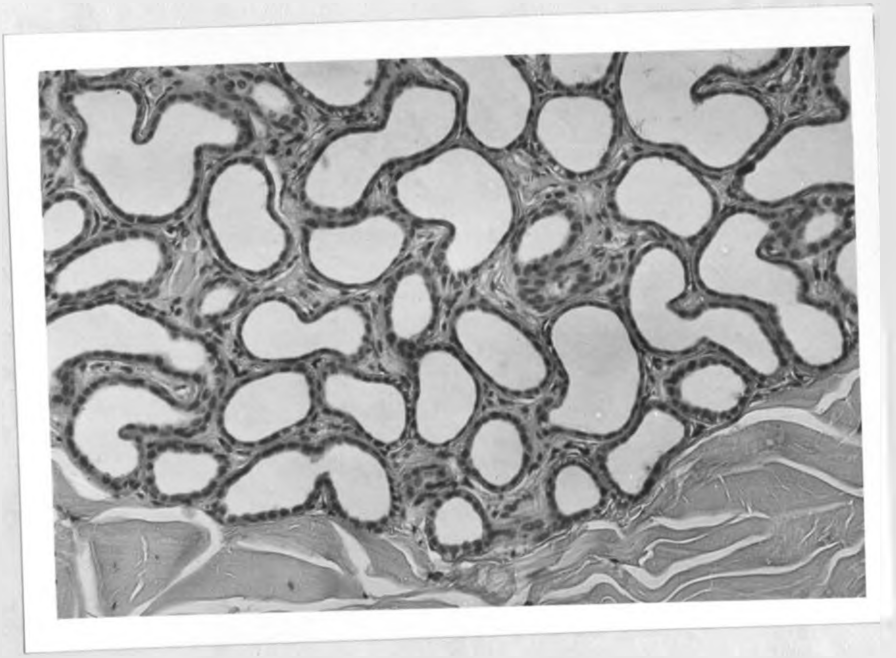


Plate 5: Skin section (T.S.) from the dorsal side of the rhinoceros to show the distribution of the sweat glands. Magnification X 3.5

Plate 6: The structure of a sweat gland in the rhinoceros (T.S.). Magnification X 16





sweat 'glands' can be seen in the dermal layer and each of these could be either one highly convuluted gland or many individual sweat glands. When serial sections were taken only one duct was found associated with each 'gland', and it is, therefore, likely that these consist of only a single gland in which the secretory tubule is very convuluted. The lumen of the tubule is surrounded by a layer of cuboidal epithelium and myoepithelial cells were seen to be distributed around the outside of this. No hair follicles were observed in these sections.

TABLE 22

The changes through the day in the rectal temperature of the elephant.

Date	Rectal Temperature (°C) 07.00 hrs.	12.00 hrs.	18.00 hrs.
February	35.8	37.4	38.4
1971	36.3	36.9	38.0
	35.4	37.3	38.2
			mean 38.2 (± 0.2 S.D.)
February	36.9	no heat load	
1972	36.9		
April	36.2	37.0	37.1
1972	36.4	37.2	37.2
	36.2	37.2	37.2
	36.0	37.2	
means	36.1 (± 0.4 S.D.)	37.2 (± 0.2 S.D.)	37.2 (± 0.1 S.D.)

TABLE 23

The charges through the day in the respiration rate of the elephant.

Date	Respiration Rate (no/min) 07.00 hrs.	12.00 hrs.	18.00 hrs.
February	7	11	8
1971	8	8	6
April	8	12	12
1972	4	12	11
	16	14	11
	12	-	-
mean	9 (± 4 S.D.)	11 (± 2 S.D.)	10 (± 3 S.D.)

Overall mean = 10 (± 3 S.D.)

The effect of climatic conditions through the day on the elephant

1. Body temperature changes

The recordings of rectal temperatures on days with environmental conditions as defined previously are shown in Table 22. Rectal temperature always increased from 07.00 hrs. to 12.00 hrs., this difference being significant at the 0.1% level. In experiments carried out in February 1971, rectal temperature showed a further increase from 12.00 hrs. to 18.00 hrs., this difference again being significant at the 0.1% level. In the experiments carried out in April 1972 there was no change from 12.00 hrs. to 18.00 hrs. There was no apparent difference in the weather, the routine or behaviour of the animals between these sets of experiments.

2. Respiration Rate changes

The respiration rates recorded in the elephant are shown in Table 23. There was no significant change through the day. It was usually quite difficult to see the respiratory movements in these animals, especially as they were often eating or drinking. Measurements were, however, taken outside periods of such activity.

3. Cutaneous moisture loss changes

The changes in C.M.L. recorded through the day

TABLE 24

The changes through the day in the C.M.L. of the elephant. (February and May 1971 - solar radiation loads relatively low).

Date	C.M.L. ($g/m^2/hr$) 07.00 hrs.	12.00 hrs.	18.00 hrs.
February	48.9	73.1	60.3
1971	61.1	75.6	64.1
	52.3	-	47.5
means	54.1 (± 6.3 S.D.)	74.4 (± 1.7 S.D.)	57.3 (± 8.7 S.D.)
	Overall mean = 64.8 (± 12.4 S.D.)		
February	175.0	-	171.0
1971	165.0	151.0	173.6
May	165.2	177.5	-
1972	162.3	202.0	190.0
means	166.8 (± 5.5 S.D.)	176.8 (± 25.5 S.D.)	178.2 (± 10.3 S.D.)
April	235.6	174.4	-
1972	208.8	194.3	203.5
	192.8	171.4	146.9
	221.8	186.7	-
means	214.6 (± 18.4 S.D.)	181.7 (± 10.7 S.D.)	175.2 (± 90.2 S.D.)
	Overall mean = 190.5 (± 21.1 S.D.)		

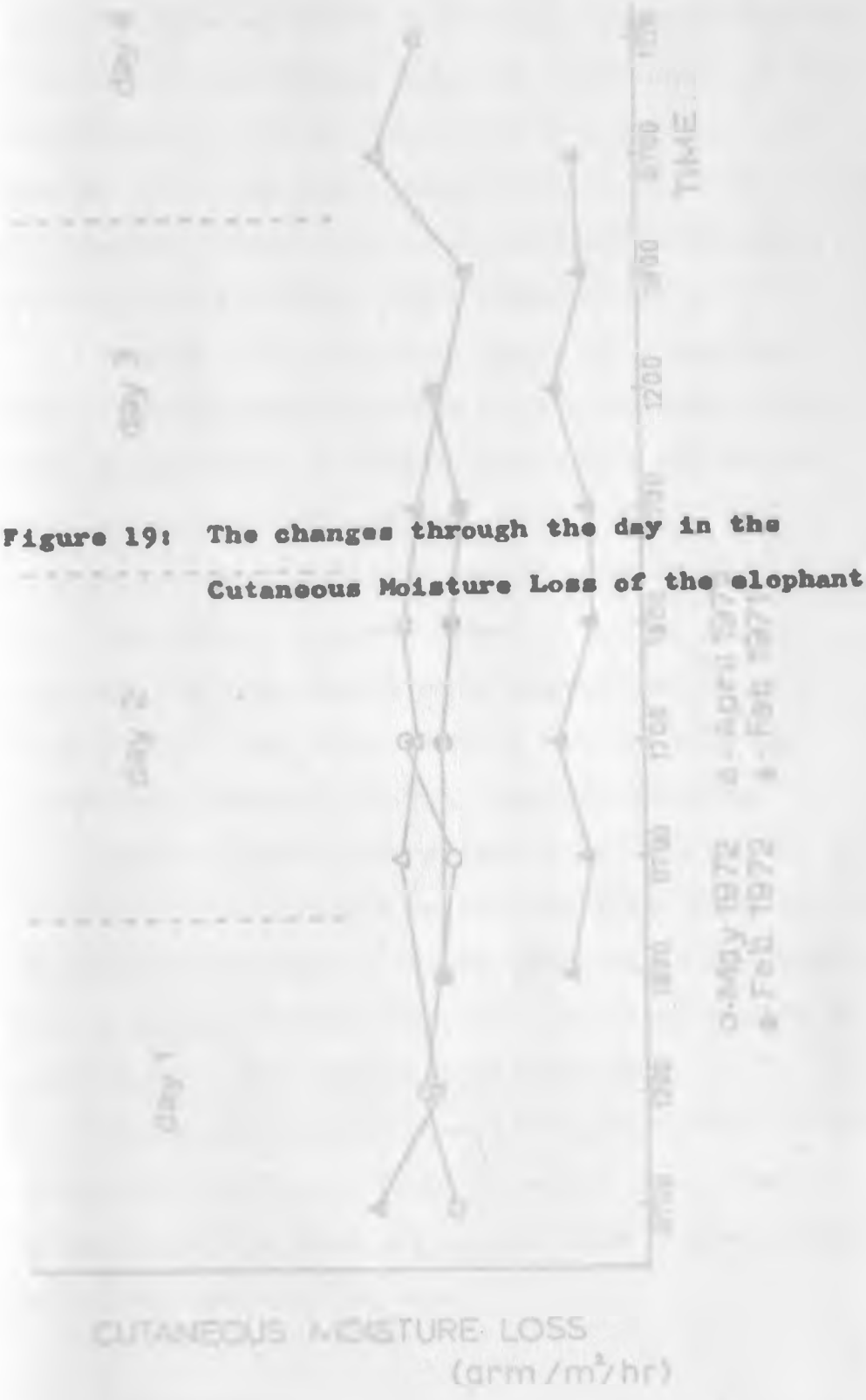
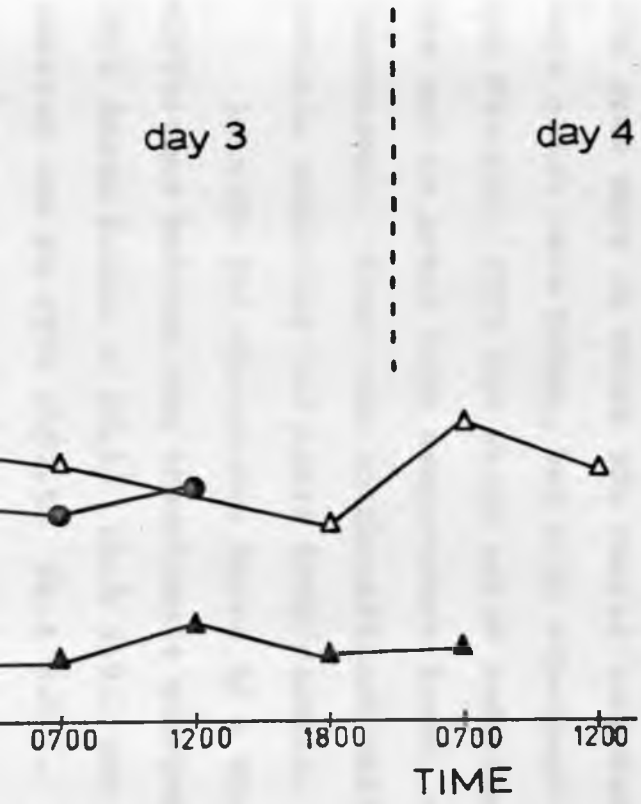


Figure 19: The changes through the day in the Cutaneous Moisture Loss of the elephant.

CUTANEOUS MOISTURE LOSS
(grm/m²/hr)
day 1
day 2
0700
1200
1800
0700
1200
1800
o-May 1972
Δ - April 1972
● - Feb. 1972
▲ - Feb 1971


day 3
day 4
0700
1200
1800
0700
1200
TIME

are shown in Table 24 and fig. 19. These include the same days on which the rectal temperature readings above were taken, but also experiments in May and February 1972 for which solar radiation load was not so great (air temperature lower, cloud cover increased). There was no significant difference between these and the April 1972 results.

As with the rhinoceros there is a distinct difference between the experiments carried out in 1971 (mean C.M.L. = 68.4$^{+}_{-}$ 14.4 S.D.) and those carried out in 1972 (190.5$^{+}_{-}$ 21.1 S.D.). This difference is significant at the 0.1% level.

The earlier results showed a slight change through the day, though only significant at the 5.0% level. The later results did not show any consistent change in C.M.L. through the day.

Starch iodine papers were also used on the elephant in an attempt to confirm these high cutaneous moisture losses. Again, the papers had turned purple within twenty seconds of placing them on the skin under a dessicant capsule holder.

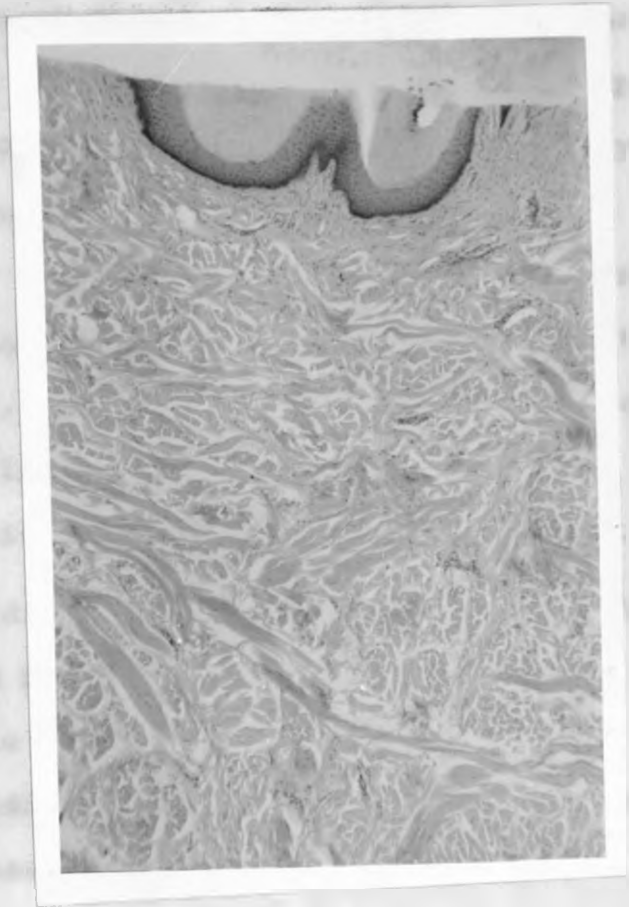
These papers were used in February 1971 to try to detect any C.M.L. from the ears. The results proved negative when the papers were placed against either side of the ear.

4. Skin sample

Plate 7 shows a section of the skin taken from

Plate 7: Skin section (T.S.) from the dorsal side of the elephant. Magnification X 3.5

Plate 8: Skin section (T.S.) from the inner side of the ear of the elephant. Magnification X 6.3



the back of the elephant. Most of the epidermis was removed before the section was taken so that only the lowest projections of the epidermal papillae are visible. No sweat glands were found in any of the sections taken.

Plate 8 shows a section of the skin taken from the inner side of the ear. Again, no sweat glands were found. This side of the ear has, however, many capillaries which can be seen between the epidermal papillae in the upper dermal layers.

Appendix 2 contains calculations of the heat inputs and heat outputs of the elephant in the experiments in February 1971.

Appendix 3 contains calculations concerning the increase in surface area of the elephant due to its ears.

TABLE 25

Changes through the day in the rectal temperature (Tr) of the zebra.

				Initial rise occurred to			Maximum occurred at		
Day	Zebra	Time (hrs.)	Tr (°C)	Time (hrs.)	Tr (°C)	% increase	Time (hrs.)	Tr (°C)	% increase
1	1	08.30	37.9	09.30	38.2	66	13.00	38.4	0.6
1	2	08.30	37.2	09.30	38.4	92	13.00	38.5	1.3
2	1	08.00	36.6	10.00	38.1	72	16.00	38.7	2.1
2	2	08.00	37.3	-	-	-	14.00	38.7	2.4
3	1	09.00	37.0	11.00	38.3	72	14.00	38.8	1.8
3	2	09.00	37.7	11.00	38.3	46	14.00	38.8	1.1
4	1	08.30	36.1	11.00	38.0	82	15.00	38.4	2.3
4	2	08.30	36.8	10.00	38.0	75	15.00	38.4	1.6

TABLE 26

The changes over four days of the respiration rate of the zebra.

	Respiration Rate (no/min)	
Time (hrs.)	Zebra I	Zebra 2
08.00	25	26
08.20	24	28
08.30	41	28
09.00	46	30
09.10	40	24
09.20	40	40
09.30	52	51
10.00	56	43
10.20	60	37
10.40	55	50
10.45	60	50
11.00	64, 48	46, 38
11.20	54	40
11.35	44	52
11.40	60	48
12.00	52	50
12.20	55	50
12.35	60	50
13.00	58, 60, 45	55, 44, 40
13.20	50	43
13.35	-	52
14.00	60, 56, 51	55, 50, 46
14.20	46	30
14.35	-	51
14.40	56	-
15.00	61, 60, 51	50, 48, 47
15.15	-	50
15.20	50	20
16.00	50, 47	45, 40
16.20		43

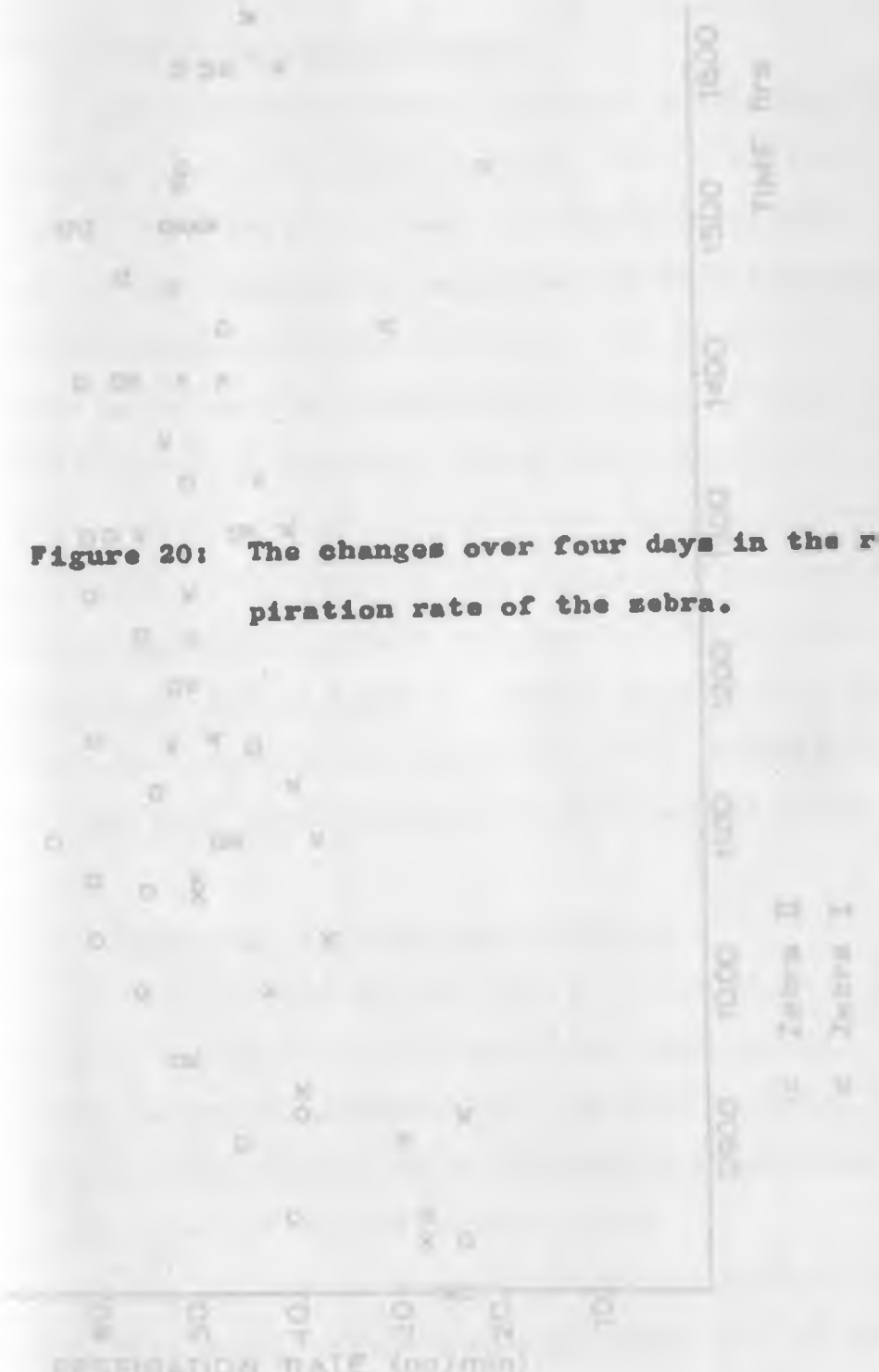
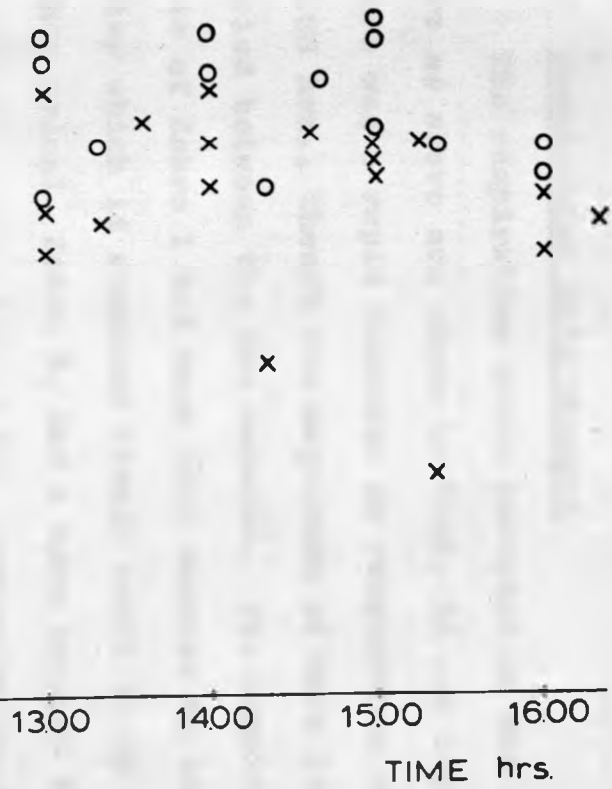


Figure 20: The changes over four days in the respiration rate of the zebra.

RESPIRATION RATE (no/min)
60
50
40
30
20
10
09.00
10.00
11.00
12.00
× Zebra II
o Zebra I


13.00
14.00
15.00
16.00
TIME hrs.

afternoon.

2. Respiration rate changes

The respiration rates recorded on the same four days as above are shown in Table 26 and fig. 20. There was a rapid increase in respiration after 09.00 hrs., though the magnitude of this increase varied between the two animals. The respiration rate of Zebra 1 had more than doubled by 10.00 hrs., after which it remained steady until 15.00 hrs. The other animal, Zebra 2, had a more gradual increase in respiration rate and the maximum value recorded was significantly lower (at the 0.1% level) than that recorded in Zebra 1. Both animals had a significant decrease in respiration rate (significant at the 0.1% level) between 15.00 hrs. and 16.00 hrs.

3. Cutaneous moisture loss changes

Figs. 21 and 22 and Tables 27 and 28 show the C.M.L. recorded on four days from each animal. Day 4 produced the highest C.M.L. in both animals, in Zebra 1 this value was significantly higher than the mean value of the other three days.

Fig. 23 shows the changes in mean C.M.L. for the four days. In both animals there was an increase up until 14.00 hrs., after which there was a gradual decline. The higher C.M.L. was recorded in Zebra 2. In comparison to Zebra 1 this animal had

TABLE 27

The changes over four days in the cutaneous moisture loss (C.M.L.) of Zebra I.

Time (hrs)	C.M.L. ($g/m^2/hr$)	Time (hrs)	C.M.L. ($g/m^2/hr$)
09.00	59.0	10.00	133.0
	73.0		135.0
	93.0		135.0
	114.0		214.0
mean	84.8 (± 24.0 S.D.)	mean	154.3 (± 39.8 S.D.)
11.00	172.0	12.00	172.0
	179.9		213.0
	192.0		258.0
	260.0		360.0
mean	200.7 (± 40.4 S.D.)		250.8 (± 80.6 S.D.)
13.00	168.0	14.00	191.0
	255.0		285.0
	276.0		298.0
	373.0		343.0
mean	268.0 (± 84.2 S.D.)	mean	279.3 (± 63.8 S.D.)
15.00	130.0		
	233.0		
	240.0		
	309.0		
mean	228.0 (± 73.8 S.D.)		

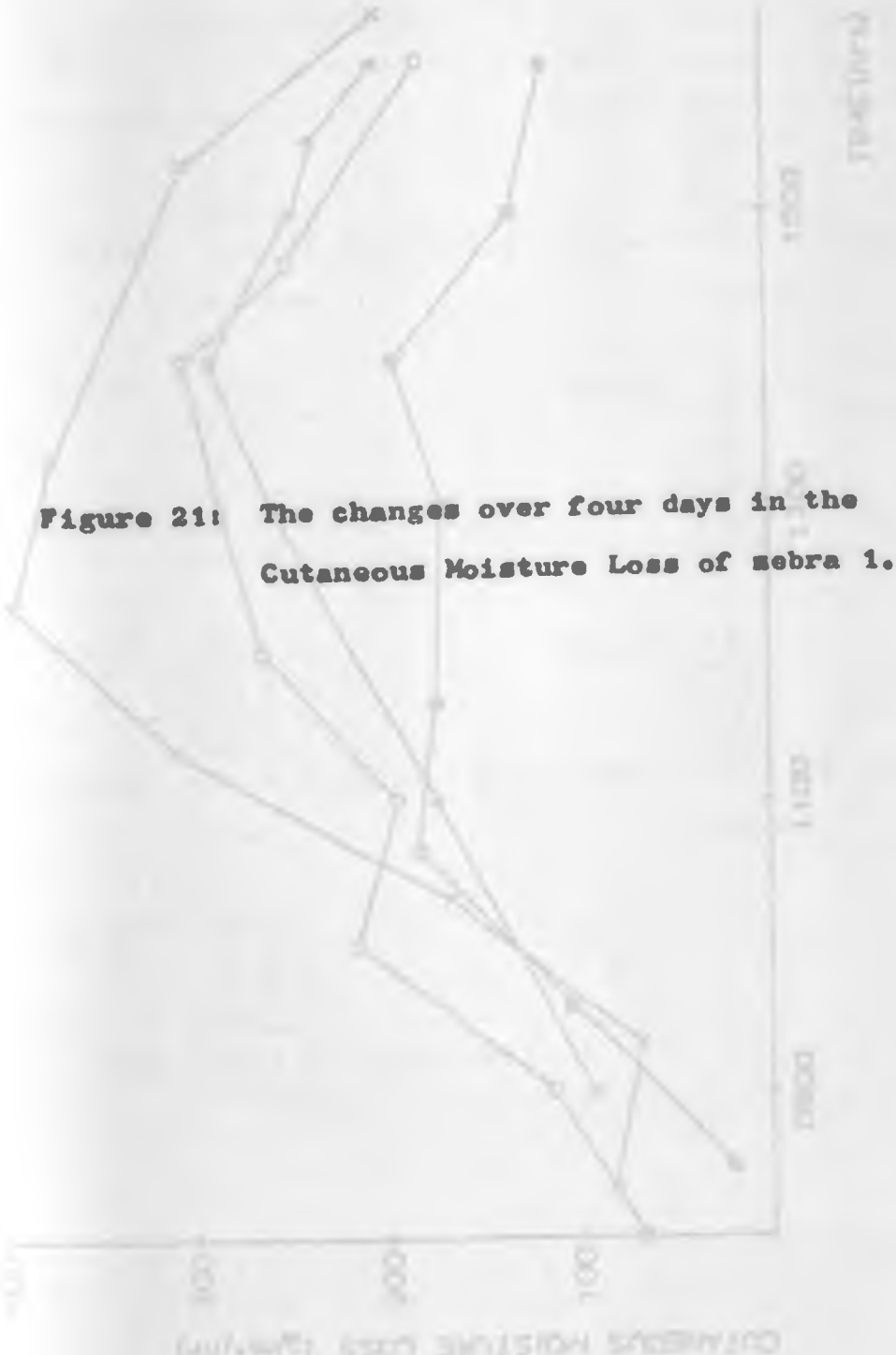


Figure 21: The changes over four days in the Cutaneous Moisture Loss of zebra 1.

400
300
200
100
CUTANEOUS MOISTURE LOSS (g/m²/hr)
09.00
11.00

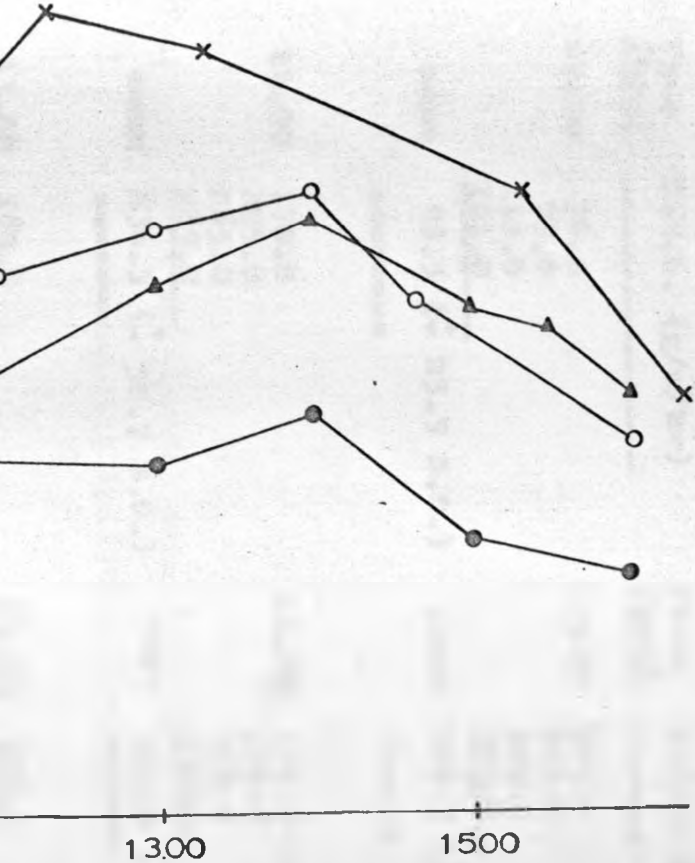

13.00
1500
TIME (hrs)

TABLE 28

The changes over four days in the cutaneous moisutre loss (C.M.L.) of Zebra 2.

Time (hrs)	C.M.L. ($g/m^2/hr$)	Time (hrs)	C.M.L. ($g/m^2/hr$)
09.00	76.0	10.00	124.0
	75.0		202.0
	110.0		209.0
	121.0		290.0
mean	95.5 (± 23.5 S.D.)	mean	206.3 (± 67.8 S.D.)
11.00	170.0	12.00	181.0
	225.0		224.0
	265.0		304.0
	285.0		335.0
mean	236.3 (± 50.7 S.D.)	mean	261.0 (± 70.9 S.D.)
13.00	180.0	14.00	225.0
	255.0		285.0
	361.0		355.0
	362.0		350.0
mean	289.5 (± 88.6 S.D.)	mean	303.7 (± 61.4 S.D.)
15.00	260.0		
	274.0		
	314.0		
	355.0		
mean	300.7 (± 42.8 S.D.)		

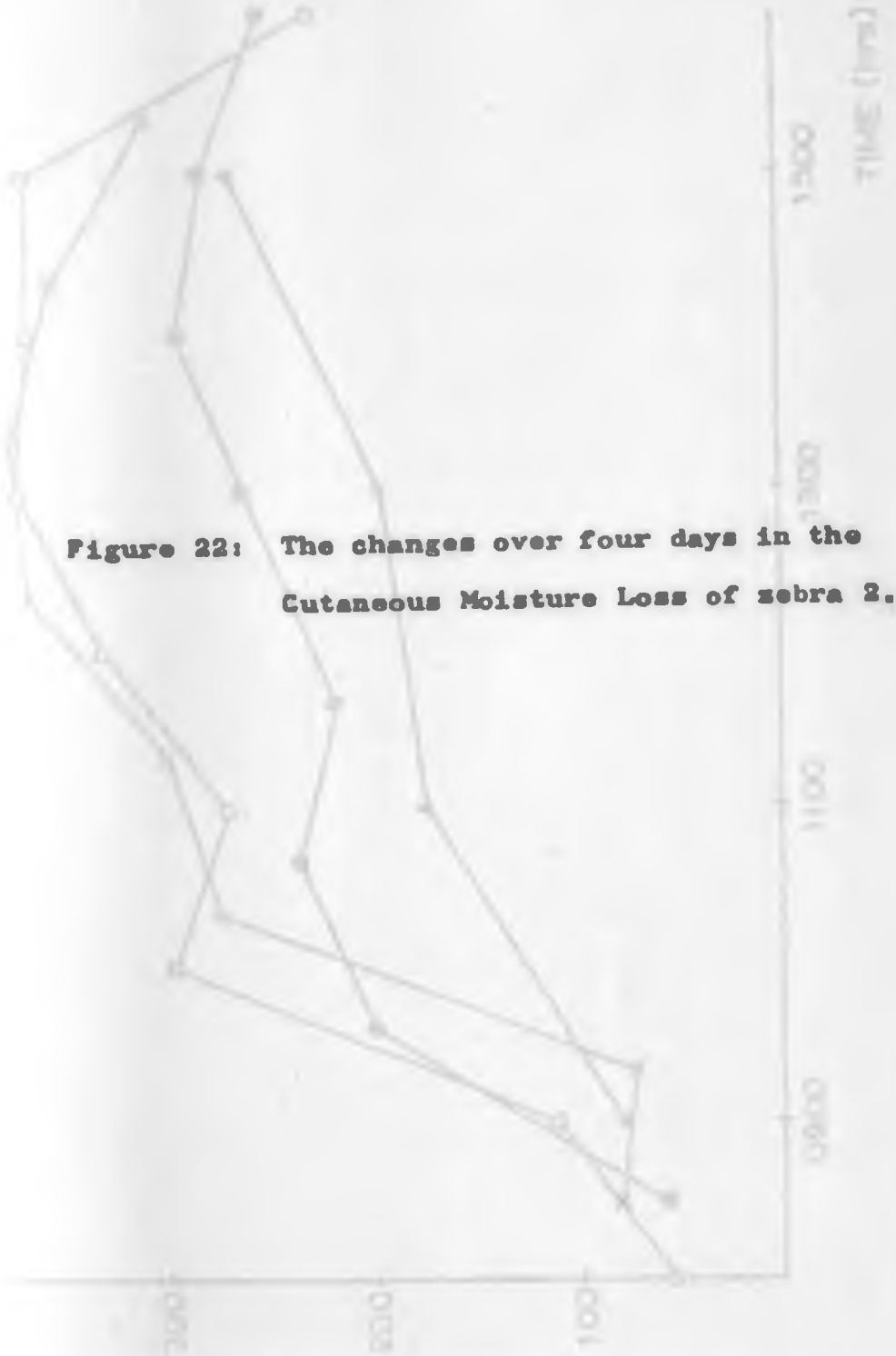
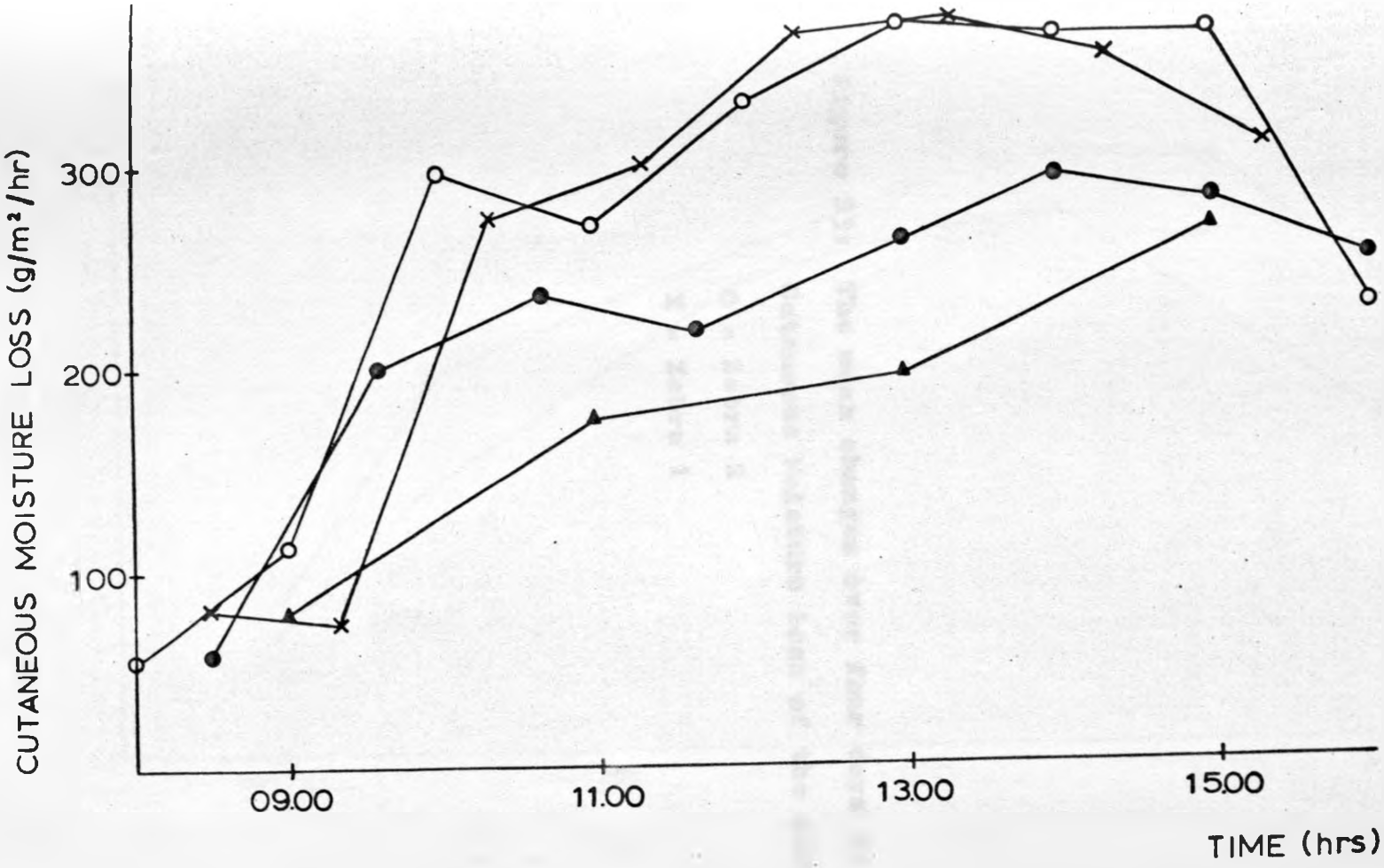


Figure 22: The changes over four days in the Cutaneous Moisture Loss of zebra 2.


CUTANEOUS MOISTURE LOSS (g/m²/hr)
300
200
100
09.00
11.00
13.00
15.00
TIME (hrs)

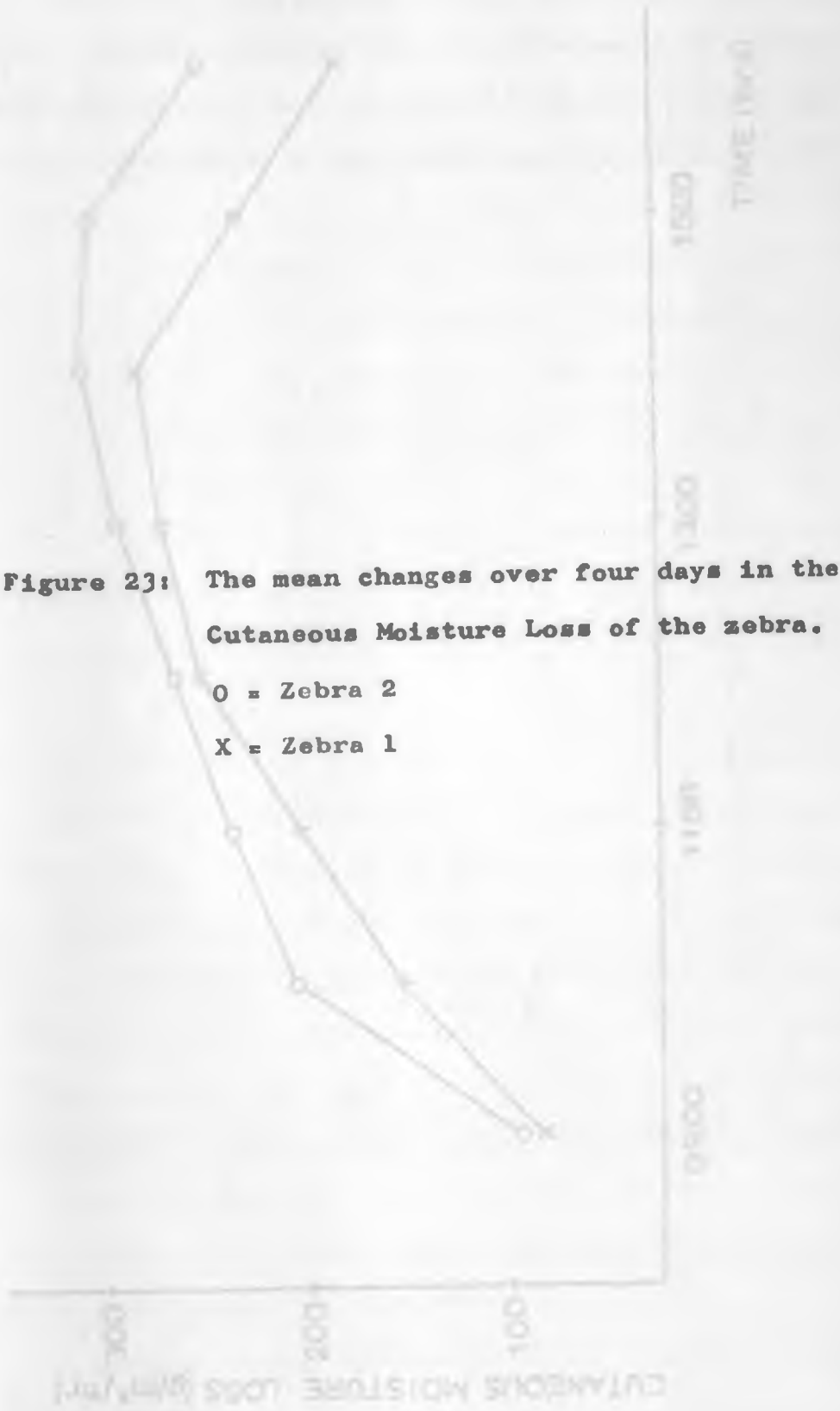
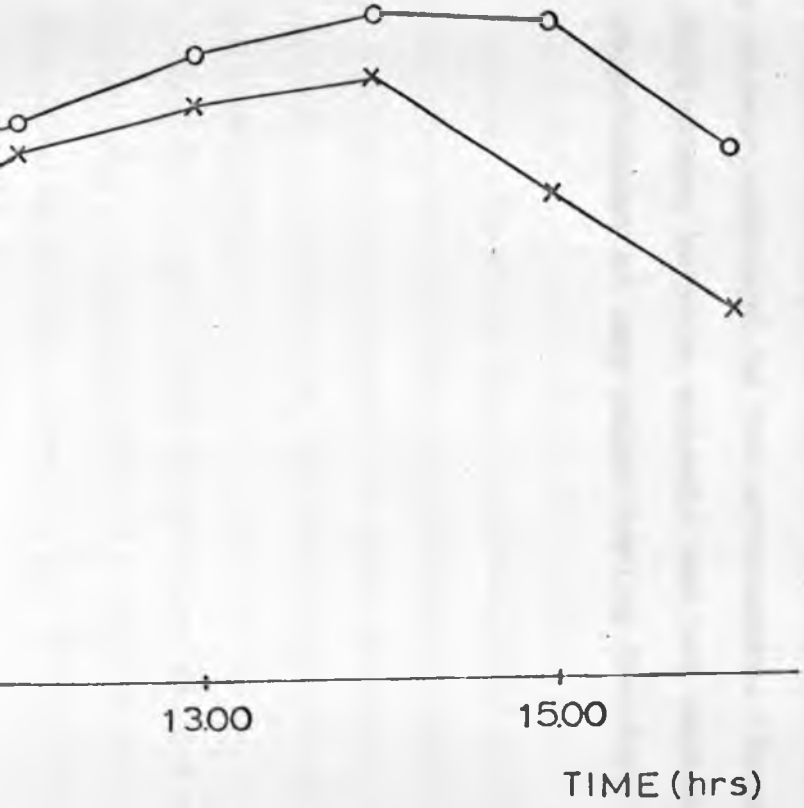


Figure 23: The mean changes over four days in the Cutaneous Moisture Loss of the zebra.

O = Zebra 2

X = Zebra 1

CUTANEOUS MOISTURE LOSS (g/m²/hr)
300
200
100
09.00
11.00


13.00
15.00
TIME (hrs)

a more rapid increase in C.M.L. during the mornings and a slower decrease in the afternoons. However, this difference between animals was not statistically significant at any point during the day.

Discussion of Materials and Methods

It is pertinent at this point to make some comment on the results obtained in relation to the materials or methods used rather than in relation to the overall objective of the study.

It was reported that the ventilated capsule results obtained in the small climatic chamber were such that it appeared in some cases as if there was an increase in the C.M.L. of the galago on heat exposure.

When the large climatic chamber became available for use, it was possible to have easy access to the animals as they were placed in a small open meshed cage with a relatively large opening panel. Any equipment could, therefore, be put in place, adjusted, or removed with the minimum of handling and without having to remove the animals from the heat. This meant that the ventilated capsule experiments could be repeated with better supervision, as well as allowing the use of the dessicant capsule method. The size of the chamber also allowed the thermocouple/wick system to be placed inside the chamber so that it was, therefore, at the same temperature as the air reaching it from the ventilated capsule.

Under these circumstances, the results showed conclusively that no increase in C.M.L. was occurring on heat exposure of the galagos. This

then posed the question of what were the causative factors of the original artefact.

It is possible that some slight differential heating or cooling was occurring between the tubes carrying the two streams of air to the thermocouple/wick system (Fig.1.) This could have happened inside or outside the chamber and would have differentially altered the relative humidity of the two streams of air. The relative humidity of the air stream is inversely proportional to the amount of evaporation that occurs at the wicks and, therefore, is also inversely proportional to the decrease in wet bulb temperature that results from the evaporation. As the output trace is dependent on the temperature difference between the pairs of thermocouple junctions which are recording this decrease in temperature, a differential heating or cooling such as is suggested would mean that the output obtained would fail to reflect the true skin air flow/ambient air flow difference in relative humidity. Since this value is determined by the amount of moisture present on the skin of the animal the results obtained were not an accurate measure of this and, therefore, failed to indicate correctly whether or not there were any changes in C.M.L.

The chances of this having occurred were made greater by the fact that the thermocouple/wick system was placed outside the small climatic chamber.

The two streams of air, therefore, had to pass out of the hot, controlled conditions of the chamber in to the cool, uncontrolled conditions of the laboratory. This was necessitated by the size of the chamber so the thermocouple/wick system was placed as close as possible to the exit of the air streams from the chamber in an attempt to minimise the risk.

The other disadvantage of the small climatic chamber was that it was not possible to see if the ventilated capsule was sitting properly on the animal, such that there was an air-tight seal between the rim of the capsule and the skin. Some difficulty had been experienced previously in maintaining this air-tight seal and, in order to overcome the problem, some sealing rubber had been placed round the outer edge of the rim. Even so it is possible that this was not sufficient and that the air was entering under the rim of the capsule. If this were the case, it would mean that a larger area of skin was being ventilated, the amount of moisture taken up would be greater and the C.M.L. loss would, therefore, appear to be high. While the capsule is in place the evaporation of any moisture under the rim is suppressed, whether it be insensible or sensible moisture. If the air-tight seal had broken as suggested above this moisture would have been carried into the capsule and would also have been reflected as an increase in C.M.L.

Whatever the reason(s), once the experiments were repeated in the large climatic chamber the results obtained showed no active increase in C.M.L., and this was confirmed by the dessicant capsule results.

The investigations carried out showed that the tranquilliser appeared to have little effect on the ability of the baboons to thermoregulate at high environmental temperatures. Ideally a similar investigation should have been carried out with the chimpanzees, instead of extrapolating the results from this work with the baboon. As the chimpanzees were obtained on the understanding that only observation experiments were to be carried out this was impossible. The tranquilliser was, therefore, used and any changes in rectal temperature monitored closely, so that in the event of hyperthermia (above 40.5°C) the animals could be removed from the chamber. These measurements were then used in drawing the conclusions concerning the interaction of the tranquilliser with the animal's ability to thermoregulate.

As has been mentioned, the tranquilliser did appear to have an effect on the ability of the chimpanzees to thermoregulate such that they were unable to prevent a fall in body temperature. The two days of the experiments were relatively cold and in one animal hypothermia occurred during a

period of fifty to sixty minutes during transportation, while in the other it occurred during a period of only ten to fifteen minutes while the tranquilliser was taking effect and basal measurements were being taken. Fortunately, this interaction did not appear to affect temperature regulation on heat exposure. In one experiment rectal temperature rose 1.2°C (37.4°C to 38.6°C) during a ninety minute exposure to 40.0°C, an increase similar to that observed in the baboon in similar circumstances. In the other two experiments rectal temperatures increased steadily from the low values, around 35.1°C, but never exceeded 39.4°C. In one experiment it was beginning to stabilise at this value after three hours at 40.0°C and in the other experiment it had already stabilised at 39.1°C after two and a quarter hours at 40.0°C. In the baboon experiments rectal temperatures of over 40.0°C were recorded during much shorter heat exposure periods. From a consideration of all these facts, it was concluded that it was unlikely that the tranquilliser would significantly alter the qualitative responses of either the baboon or the chimpanzee to heat exposure.

Robertshaw (personal communication) also found this to be true in work on stump-tailed macaque monkeys (Macaca speciosa), though the tranquilliser did have the effect of delaying the onset of the thermoregulatory responses to heat exposure.

In carrying out this study, the question arose of whether it was better to use the ventilated or the dessicant capsule method for the measurement of cutaneous moisture loss. The problem encountered initially in the ventilated capsule experiments with the galagos has already been discussed and the ways of overcoming it suggested.

In all ventilated capsule work, it is first necessary to find the critical flow rate (Fo) at which there is no difference in the skin temperature (Ts) between the inside and the outside of the capsule. If any other flow rate is used (F) a correction factor of $(Fo/F)^{k}$ has to be made to the measured C.M.L. (E), k being the linear coefficient of log E against log F. McLean (1963) states that these differences in Ts are usually small and that normally the 95% confidence limits for Fo are quite large, e.g. 1.11 litres/min to 2.70 litres/min if Fo=1.93 litres/min. This is so, unless the capsule is small in area and depth, when Fo becomes much more important. As the capsule used on the galagos was relatively small it was important to find Fo (or k in order to carry out the correction) to produce an accurate value of C.M.L. However, the difficulties arising from the size of the capsule and the problems in handling the animal meant that this was not possible. Therefore, this method was used only on a qualitative confirmatory basis.

In later experiments, this problem of determining Fo, the lack of quantitative reliability which resulted if it was not found, and a preference for the more straightforward method resulted in the use of the dessicant capsule. The ventilated capsule was then again used to obtain confirmatory results whenever possible. The distinct advantage of the ventilated capsule method is evident in experiments in which a continuous record of C.M.L. is required, e.g. in the response to the administration of a drug. In the present experiments the C.M.L. was normally low and so, consequently, the deflection from zero obtained on the milivolt recorder was also small. This meant that any changes in C.M.L. were often masked by the instability of the equipment, which resulted, mainly, from its increased sensitivity. Five sets of very small (48S.W.G.) thermocouples were used so that any variations in the differences between the wet bulb temperatures were magnified, regardless of whether these were experimental artefacts or natural variations. This was another contributory factor in the decision to use the dessicant capsule.

When the tranquilliser, phencyclidine hydrochloride, was used a dose of 1mg/kg given intramuscularly (i.m.) was normally sufficient to tranquillise the animal. It was then in a trance-like state of unconsciousness and could be handled

freely for two to three hours. Occasionally, the animals would attempt co-ordinated, forceful movements of the limbs within this time, and if this began to interfere with the experiment a further 0.25mg/kg of the tranquilliser was given. At least thirty six hours was allowed between consecutive experiments in an attempt to eliminate any such resistance to the effect of the tranquilliser.

There appeared to be no adverse side effects of the drug in the galago or the baboon, and these animals regained normal behaviour after four to eight hours. However, this was not the case with the chimpanzee. As the tranquillising effect of the drug began to wear off, the chimpanzee began to exhibit extreme agitation, rolling around on the floor of his cage and "screaming". This behaviour occasionally occurred in the untranquillised state but to a lesser degree, and it could be stopped by handling the animal. After tranquillisation, the animal could not be pacified in this way and it was necessary to administer a dose of 10µg/kg of acetyl promazine. It might be that the animal was experiencing hallucinations, a side effect known to be common in man (Martingdale, 1967). Subsequently, 10µg/kg of acetyl promazine was given intramuscularly on completion of each experiment to prevent these reactions occurring.

DISCUSSION

When discussing thermoregulation in the animals investigated, they are best divided into the two broad general categories of primate and non-primate species. Primate physiology is arousing considerable interest at present:

1. because primates are being used widely as a model for man in many physiological experiments.
2. with regard to the inter-relationships between primate species;
3. with regard to whether this information from extant species can be used to clarify man's evolution.

The three non-primate species have been considered in an attempt to fit the knowledge obtained into the more general picture of thermoregulation which is emerging in mammals.

This investigation has suffered throughout from the availability of insufficient numbers of animals from each of the species used. In an attempt to overcome this, experiments within each species have been replicated as often as possible, but the handling, management or associated problems have imposed a limit to this. Nevertheless, it is felt that sufficient information has been collected to make the more general qualitative conclusions valid, i.e. whether the animal sweats or pants. In considering

quantitative levels for the parameters a little more caution is necessary in making any conclusions until some confirmatory work on more individuals in each species is carried out.

The results obtained from the galago, however, need not be included in this generalisation. They were quite clear cut as in this prosimian species evaporative heat loss from the body only occurs through panting, i.e. from the upper respiratory tract. The increases in C.M.L. which did occur on heat exposure (up to 36.0 and 35.9g/m^2/hr.) are far too small to be considered as sweating. When the animal is experiencing a high heat load the minute volume of respired air is increased by panting and this increases the amount of evaporation which is taking place. In the galagos the respiration rate increased gradually from basal values of 40 to 80 times/minute at 18°C - 20°C up the maximum values of between 250 and 300 times/minute at 40°C.

Crawford (1962) found that when dogs were exposed to high temperatures there was an immediate increase in respiration rate up to 250 times/minute. This did not increase any further if the exposure was continued or the temperature increased. This frequency was found to be the resonant frequency of the respiratory tract and involved less expenditure of energy (and thus less heat production) to sustain it than other similar high respiratory rates.

However, the gradual increase in respiration rate in the galago indicates that, as in the majority of panting animals (Richards 1970), a similar principle of resonant frequency is not involved in the increase in respiration rate which occurs on heat exposure.

Montagna and Yun (1962) classified the skin of the greater galago as "primitive" on the basis of histological and histochemical examination. The lack of sweat gland activity observed in this study fits in with this observation. No sweat discharge could be stimulated using various concentrations of sympathetic and parasympathetic drugs, so, perhaps, these glands are merely "scent" glands as Montagna and Yun suggest. They found the glands to be sparsely distributed and apocrine; the histology carried out here would tend to confirm this. These glands could, however, be alternatively classified as epitrichial (Bligh 1967) as Montagna and Yun (1962) found them associated with hair follicles; in plate I the gland is seen in a similar position.

The greater galago is a nocturnal, aboreal animal found widely distributed in Africa, south of the Sahara (Haddow and Ellice 1964). The woodland environment it inhabits can be found at altitudes from sea level to 2,000 metres and due to the effect of altitude the species is thus

distributed over varying temperature zones. (Bearder and Doyle 1973). By its behaviour the galago avoids the high environmental heat load that it could experience over much of this area. They have been observed during the day to be sleeping on the branches of trees, where they were normally shielded from solar radiation, and any voluntary movement during the day only occurred in response to changes in temperature (Bearder and Doyle 1973). Alternatively, they sleep in hollow trees (Astley-Maberley 1967). They are active at night when any heat produced as a result of their activity can be easily dissipated. Besides this behavioural thermoregulation the animal is well adapted to overcome the cooler conditions due to its thick fur coat, while the hotter conditions it experiences are overcome by panting.

In the present study on the baboons their respiration rate also increased on heat exposure, though the magnitude varied between the three individuals. At an environmental temperature of 40°C there was an 80% increase in respiration rate in baboon 1 (19.8 to 35.7 times/min), a 33% increase in baboon 2 (21.1 to 28.9 times/min) and a 77% increase in baboon 3 (26 to 46 times/min). Funkhouser, Higgins, Adams and Snow (1967) exposed unanesthetised baboons to an air temperature of 45°C and this caused an average increase in respiration rate from

21.3 times/min (at 25°C) to 72.0 times/min (a 238% increase). This 5°C difference between the environmental temperature of 45°C used in their experiments and the 40°C used in the present work appears to make a considerable difference to the respiratory response of the animal to heat exposure. However, Newman, Cummings, Miller, and Wright (1970) reported that exposure to an environmental temperature of 43°C caused no increase in respiration rate in the baboons used in their study. Respiration rate increased only if sweating was blocked with scopalamine. An increase in respiration rate also occurred in the present study when sweating was blocked by atropine.

The increase in respiration rate on heat exposure was found to be correlated with rectal temperature in baboons 1 and 2. It is, therefore, probable that deep body temperature is a controlling factor for respiration rate. Thus, if sweating is blocked this causes an increase in deep body temperature which would then cause the increase in respiration rate observed.

Newman et al (1970) found that there was no substantial change in the water they collected from the head when sweating was blocked. From this data they concluded that the baboon obtains about 1/5th of its evaporative cooling from the respiratory tract.

The question then arises of what was the stimulus for the increased respiration rate which occurred in the present work. An increase in body temperature will raise the animal's metabolic rate and thus increase its oxygen consumption. This will stimulate the ventilation rate, but the Q_{10} for this increase would be expected to be between 2.0 and 3.0 due to the Vant Hoff effect. In the present work Q_{10} values for the increase in respiration rate were 9.1, 6.5 and 5.5 in baboons 1, 2 and 3 respectively. However, if there was a decrease in tidal volume this might offset the high Q_{10} values for the increase in respiration rate so that the increase in the ventilation of the lungs could still have a Q_{10} of 2.0 to 3.0. If there had been no change in tidal volume a fall in blood CO_2 would have occurred. This was not measured, but no signs of respiratory alkalosis, such as tetany, were seen. It is, therefore, likely that there was a decrease in tidal volume so that the increased respiration rate was not hyperventilation. Thus the animal was panting and the stimulus to respiration was more than merely a Q_{10} effect. If it had been possible it would have been interesting to:

1. measure tidal volume to confirm that it was panting;
2. partition the evaporative cooling into its respiratory and cutaneous components, and

thus find the importance of panting to the animal.

The basal values of respiration rate recorded at 18°C to 20°C in the present study had a mean value of 21.6 times/min. This is very similar to the mean of 24.8 times/min reported by Vice and Rodrigues (1965) in 80 tranquillised baboons at a similar temperature.

Punkhouser et al (1967) reported that "sweating was observed during exposure to heat" but no attempt was made to investigate it further. In the present study C.M.L. was found to have a mean basal value of 17.7 $g/m^2/hr$ at environmental temperatures between 18°C and 22°C. If these animals were then exposed to an environmental temperature of 40°C there was a gradual increase in C.M.L. The maximum value recorded was 121 $g/m^2/hr$ after 90 mins of the exposure to 40°C. It is noticeable that the smallest baboon (3) which had the highest respiration rate and the greatest increases in rectal temperature sweated the least. C.M.L. in this animal never exceeded $70g/m^2/hr$ and on one occasion reached only $60g/m^2/hr$ after 110 mins of exposure to 40°C. In all three animals droplets of sweat were seen or were detected by the starch and iodine method, which confirms that this is sweating and not merely an increase in insensible moisture loss.

This is further substantiated by the effect of

atropine on C.M.L. during heat exposure. When the atropine was injected intravenously it completely blocked sweating so that C.M.L. dropped to a pre-exposure level. At the same time there was a compensatory increase in respiration rate though this failed to prevent an increase in body temperature. This increase in respiration had an average Q_{10} of 10.2 in four experiments, i.e. it was not merely a result of increased body temperature/Oxygen consumption. This effect of atropine also proved that the sweat glands are cholinergic.

The mean correlation coefficient (for the three animals) between the duration of exposure to 40°C and C.M.L. was found to be significant, while that between rectal temperature and C.M.L. was only significant in baboon 3. From the data, it thus appears unlikely that the C.M.L. is as dependent as respiration rate on the deep body temperature of the animal.

The ability of an animal to thermoregulate efficiently is reflected in the stability of its body temperature on heat exposure. In the present work, body temperature of the baboons always increased on exposure to an environmental temperature of 40°C. In baboon 1 rectal temperature had normally stabilised at about 39.0°C, after a mean increase of 0.66°C, and in baboon 2 at about 39.4°C, after a mean increase of 0.57°C. However, baboon 3

always had a bigger increase in rectal temperature (mean increase = 1.45°C) and normally this had not stabilised, though the upper value recorded was never more than 40.1°C. Funkhouser et al (1967) reported the work of Slonim (1939) in which two baboons were able to tolerate exposure to 40°C without any change in body temperature. Also Newman et al (1970) found that body temperatures remained in equilibrium in their baboons during 150 minutes at 43°C. However, Funkhouser et al (1967) found that during 90 minutes at 45°C there was a steady rise in the body temperature of 4 untranquillised baboons from 38.2°C to 40.6°C.

It would appear from this limited collection of data that temperatures of 40°C to 43°C can be tolerated by the majority of individuals. However, the increase to 45°C imposes too much of a heat load on the baboon and efficient thermoregulation breaks down.

Histological examination in the present study suggests that the sweat glands present are eccrine glands, like those in man. Montagna and Yun (1962) found them to be numerous, though in the present study many sections had to be taken before a gland was located. After their histological and histochemical examination, Montagna and Yun classified these glands as "highly differentiated and having undergone as much evolutionary advance as those of

the gorilla and chimp". They also found a few apocrine glands, especially on the anterior chest and belly. These were not found in the present study, even though the skin samples were taken from the chest region.

The baboon (Papio spp.) is distributed through the whole of sub saharan Africa except for Central West Africa, though the yellow baboon used in the present study is restricted to East and Central Africa, (Napier and Napier 1967). Within this area it is normally found in open arid country (Maples 1972) in which it moves around during the day. It is, therefore, exposed to a much greater heat load than the galago, as well as having a much higher internal heat load due to this diurnal activity. To overcome this, the baboon has developed a sweating mechanism. This enables it to lose heat by evaporation of water from the skin surface, instead of restricting it to the upper respiratory tract. This increase in C.M.L. has not entirely superseded panting, though the thermoregulatory importance of panting does appear to be in some doubt.

The effect of the tranquilliser on the ability of the chimpansee to thermoregulate efficiently has already been discussed. If the animal is exposed to a high heat load it also dissipates excess heat from the body by both sweating and panting.

On exposure to 40°C the C.M.L. increased six-

fold in one animal (to 80.0g/m^2/hr) and doubled in the other (to 35.7g/m^2/hr). Whitford (1970 and personal communication) has confirmed this increase in C.M.L. as he found that the mean evaporative water loss at environmental temperatures of 37-38°C was more than double the value at 25°C. Some of this increase might have been due to an increase in respiration rate, a parameter he did not measure in his chimpanzees. However, he found that, on heat exposure, the rate of water loss from areas identified as sweat production areas was ten to twenty times greater than from other, non-sweating, areas. In this present work droplets of sweat were observed around the eyes, nose and mouth; on the palms and sides of the hands and feet; and around the scrotum. These are three of the eight areas that Whitford classifies as sweat production areas. This sweating was inhibited by atropine, i.e. the glands are cholinergic. The C.M.L. dropped from 80.0g/m^2/hr to 21.1g/m^2/hr when the drug was administered intravenously, but there was no significant change in rectal temperature or respiration rate in the ten minutes following this.

The respiration rate of the chimpanzees at ambient temperature (18-20°C) was normally about 30 times/min and on heat exposure this always increased. In three experiments at 40°C, two of which were with tranquillised chimpanzees, the

respiration rate stabilised at fifty to sixty times per minute. In another experiment with an untranquillised animal there was no change in respiration rate on exposure to 40°C, while in a fifth experiment with a tranquillised animal respiration rates of up to ninety times per minute were recorded on heat exposure.

These increases in respiration rate in the tranquillised animals had Q_{10} values of 7.6, 4.4 and 3.5. The possibility of this increased respiration rate being hyperventilation was increased by the use of phencyclidine hydrochloride as a tranquilliser. This drug is known to produce hallucinations in man (Martindale 1967) and these can often result in hysteria and hyperventilation. However, the animal never became agitated during the experiment and never showed any symptoms of alkalotic tetany, one of the consequences of hyperventilation. The conclusion in this animal was that again this increase in respiration rate was panting and the stimulus to respiration was more than merely a Q_{10} effect. It would be of interest to partition the evaporative water loss and measure tidal volume to confirm this.

The increases in both respiration rate and C.M.L. which occurred on heat exposure of the tranquillised chimpanzee were very closely correlated with rectal temperature. It is, therefore, probable

that the activity of these evaporative heat loss mechanisms is controlled by the animal's deep body temperature.

Through both these methods of evaporative water loss the chimpanzees were able to dissipate the heat load imposed upon them. However, Whitford (1971) reported that chimpanzees were unable to tolerate environmental temperatures of over 40°C. It would, therefore, appear that the temperature at which efficient thermoregulation in the chimpanzee breaks down must have been only slightly above the temperature used in the present work.

Under natural conditions these animals are distributed across the forest zone from Guinea to the forests of western Uganda and Tanzania (Kingdom 1971). In this environment, due to the foliage cover, the solar radiation input during the day is low (Napier and Napier 1967) so that in contrast to the baboons the chimpanzee never experiences any great heat load. For the same reason the heat loss from the environment during the night is also low and, consequently, the range of temperature that the chimpanzee experiences is normally narrow. Possibly as a result of this their ability to thermoregulate efficiently at environmental temperatures above 40°C appears to be less well developed than in the baboon.

When comparing thermoregulation between these

primate species a number of points become apparent:

1. The greater galago, a representative of the more primitive prosimian suborder, is able to thermoregulate efficiently and does so by panting.
2. Both the baboon and the chimpanzee have developed sweating as a heat loss mechanism. This appears to be used together with panting as an evaporative heat loss mechanism. The sweat glands in these animals are very similar on both a histological and histochemical basis to those of man. As in man, they are cholinergically controlled.
3. It appears that the C.M.L. in man is very much greater than that which occurs in any other primate. Newman (1970) reports that values as high as 1000 $g/m^2/hr$ have been recorded. As the density of the sweat glands in the skin of the baboon and the chimpanzee is not known and could not be measured, a comparison of the activity of individual glands cannot be made. The question of whether it is an increased number or an increased activity of the glands which is responsible for the high rates of C.M.L. in man must, therefore, be left open.
4. Having investigated thermoregulation in several primates, including the chimpanzee, the animal normally considered the closest relation to man,

the most interesting point which emerges is that man appears to be the only primate which has completely lost the ability to pant. The question then is - why? The only suggestion that the author can make is that it is possibly related to man's ability to speak. An attempt to hold an intelligent conversation in the mid-day African sun would be rather handicapped if the individuals concerned had to pant as well! However, the question of whether it was the development of speech or the lack of panting that occurred first is the important point in this respect. It could have been that speech only developed after hominids had ceased to pant.

In discussing the non-primate species the basic question which has to be answered is "how do these animals thermoregulate"? From this must follow a conclusion as to how effectively they are able to do this. The results obtained have posed some interesting questions concerning the evaporative water losses recorded from the rhinoceros and elephant. For this reason, these species will be considered together in the following discussion.

The published literature on the thermoregulatory physiology of all these animals is sparse or non-existent, probably as a result of the difficulty in obtaining experimental animals. This has also been a severe limiting factor in these current

experiments. However, it is hoped that this restricted look into a completely unknown field has proved to be of some use, if only to stimulate interest for a more comprehensive investigation.

Before discussing the general question of thermoregulation in these animals, the following points need a little clarifying:-

1. What increase in C.M.L. is there from early morning through to mid-day, i.e. do they sweat?
2. What is the explanation for the high insensible C.M.L. recorded?
3. Why is there a difference between the results of the initial experiments (February 1971) and those carried out in December 1971, February, April and May 1972?

Between 07.00 hrs. and mid-day an animal in the field is normally experiencing a gradually increasing ambient temperature and also a gradually increasing solar radiation load. The severity of the increases are likely to vary considerably, depending on weather conditions, season, etc., but they have to be compensated for through the animal's heat loss mechanisms to prevent a rise in body temperature. The difference in C.M.L. between 07.00 hrs. and mid-day will show how much reliance the animal places on sweating for this purpose. Measurements in the rhinoceros show a significant increase between the 07.00 hrs. and mid-day values of C.M.L. The mag-

nitude of this increase was not always constant, but this will be discussed in relation to question 3. It would, therefore, appear that the rhinoceros sweats (see page 142). In the elephant this is not so; although the initial experiments were a little inconclusive, further work has clearly shown that no significant variation in C.M.L. occurs through the day. In these later experiments cutaneous moisture loss from the elephant has been recorded at an average value of 190.5 $g/m^2/hr$. This is a very high value for insensible moisture loss. The rhinoceros was found to have an even higher insensible (07.00 hrs.) C.M.L., 284.3 $g/m^2/hr$, in experiments carried out at the same time. In the early experiments the insensible C.M.L. in the rhinoceros was also relatively high, 108.2 $g/m^2/hr$ and it is only the C.M.L. recorded in the early experiments on the elephant, 64.8 $g/m^2/hr$ which has a value similar to the insensible moisture loss of other species. These results pose the question of what is the explanation for these high insensible C.M.L. (question 2). It could be that these high insensible moisture losses are an artefact of the experimental method, as the dessicant capsule method used has often been found to produce high results (Maclean 1963). This could be explained by the fact that the skin underneath a dessicant capsule is exposed to air at zero humidity, a condition it never experiences under normal conditions.

It is possible that this could have been tending to "suck" moisture through the skin and so produce an apparently high rate of evaporative water loss, i.e. the skin has a high "coefficient of diffusion of water". In the rhinoceros and the elephant this coefficient is, perhaps, especially large thus producing these very high results. It could be checked by using the ventilated capsule method, in which the air passing over the skin is always at ambient humidity. However, this was not possible under field conditions.

If there was as much moisture present as these results suggested, it was thought that it should be possible to detect it by using starch-iodine papers. This has never been intended for use as a quantitative method, unless the individual marks from each sweat gland can be identified and counted. However, it was hoped that it might be of some help in clarifying the problem. When the papers were placed (underneath a dessicant capsule holder) on the skin of the rhinoceros or the elephant, they began to turn uniformly purple within 20 seconds. In similar experiments on a cow sweating at approximately 300 $g/m^2/hr$ the papers took at least 60 seconds to change colour. This shows the quantitative limitations of this method, as the rhinoceros and elephant definitely were not sweating at 900 $g/m^2/hr$! However, it does prove that there is a lot of moist-

ure coming through the skin of these animals even when the dessicant in the capsule is not present.

In recently published work, Young (1972) has found that the elephant can lose up to 560 $g/m^2/hr$ of moisture through the skin, even though there are no sweat glands present. Young states that this flow of moisture ("sweat") from the blood vessels through the skin tissue to the skin surface is enhanced by a large number of capillaries in the dermis. The distribution of moisture onto the skin from these capillaries is then "hastened" by a "chemical" which surrounds the vessels, though exactly what this chemical is, or how it functions is not stated.

As there are no sweat glands in the skin of the elephant, the C.M.L. must be controlled by the skin/air vapour pressure gradient. At 07.00 hrs. the sweat glands in the rhinoceros are unlikely to be active (see below). Therefore, the high basal level of C.M.L. in this animal is also insensible moisture loss, and thus also dependent on the vapour pressure gradient. Assuming that the vapour pressure (V.P.) of the skin is equal to the saturated V.P. at skin temperature, the C.M.L. in both animals would be expected to decrease during the night as the animal's skin temperature fell, ambient V.P. increased, and thus the V.P. gradient decreased. However, if the level of evaporative cooling should decrease during the night, the increase in convection, conduction,

and radiation that always occurs in the cooler night-time conditions would normally supplement the animal's heat loss sufficiently to dissipate the animal's metabolic heat load, plus any heat stored during the day.

This then leaves the question of the discrepancy between the initial and later experiments to be discussed. A subjective assessment of vasomotor tone at 07.00 hrs. in the prominent blood vessels on the back of the ears of the elephant suggested that there was always cutaneous vasoconstriction at this time. It is unlikely, therefore, that the evaporative heat loss mechanisms in the rhinoceros were active at 07.00 hrs., as it was in the same temperature conditions as the elephant. Measurements of C.M.L. at 07.00 hrs. would thus simply reflect insensible moisture loss. The mean insensible C.M.L. in the elephant showed a threefold difference from 64.8 $g/m^2/hr$ to 190.5 $g/m^2/hr$ between the initial experiments in February 1971 and the later ones in 1972. On the same occasions the rhinoceros was recorded as having an insensible C.M.L. of 108.2 $g/m^2/hr$ and 284.3 $g/m^2/hr$ respectively.

In an attempt to explain this difference in the values of C.M.L. obtained a comparison should be made of the experimental conditions during the two sets of experiments.

The management of the animals were exactly the

same, though the animals were between twelve to fifteen months older when the second experiments were carried out. The air temperature was a little higher (approx. 1-2°C) when the lower results were obtained. The experimental methods used were the same, although the actual capsules used were different. However, measurements made at the Athi River field station to compare the "old" (used in February 1971) and "new" (used later) dessicant capsules, showed no difference between them. Subsequently the same "new" capsules were used on the zebra and produced values for insensible moisture loss comparable to other species (40-60 $g/m^2/hr$). On both occasions water was available ad libitum, but the first set of experiments was carried out in the middle of a prolonged drought period. The moisture content of the vegetation was, therefore, probably low so that the animals might have been relatively dehydrated at the time of these early experiments. Dehydration has been found to decrease the insensible water loss from non-sweating animals (Finch personal communication), and it is possible that this is the explanation for the lower insensible C.M.L. in the first set of experiments.

Otherwise, it can only be suggested that the difference in C.M.L. in the two sets of experiments might be linked with the age difference of the animals. It would seem, however, that a younger animal

would benefit more from a high C.M.L. since it has a larger surface area to body weight ratio than an older animal. The effect of this is that in the younger animal the incoming radiation through the skin surface has only a relatively small body mass to heat up and unless this excess heat can be removed, e.g. by a high C.M.L., the animal is in danger of becoming hyperthermic.

Some of the results obtained show that this difference in insensible C.M.L. between the earlier and later experiments is reflected in the other physiological responses of the animal to the increased environmental temperature. In the rhinoceros the mean respiration rate at mid-day in the earlier experiments, when C.M.L. was low, was 87.3 times/minute. In the later experiments, when C.M.L. was high, respiration rate was significantly lower and had a mean value of 55.0 times/minute. The elephant on both occasions showed an increase in rectal temperature up until 12.00 hrs. from 36.1°C to 37.2°C. When C.M.L. was high there was never any further increase but when it was low, in the earlier experiments, it increased by a further 1.0°C to 38.2°C at 18.00 hrs.

The general question of the mechanisms for body temperature regulation in relation to the results obtained can now be discussed. The rhinoceros showed an increase through the day of 52.4

$g/m^2/hr$ in the first experiments and 90.5 $g/m^2/hr$ in the second. Early morning, basal C.M.L. was high on both occasions, 108.2 and 284.3 $g/m^2/hr$ respectively, and it is possible these increases in C.M.L. recorded through the day are merely an increase in this insensible moisture loss. However, the presence of the large, numerous, well developed sweat glands and the magnitude of these C.M.L. differences, especially in relation to the elephant, which possesses no sweat glands, would tend to indicate that this is an active secretion of moisture on to the skin, i.e. the animal is sweating.

The rhinoceros would, therefore, appear to be like many other of the larger East African mammals, e.g. waterbuck (Taylor, Spinage and Lynman 1969), eland (Taylor and Lynman 1969), impala (Maloiy and Hopcraft 1971) and buffalo (Robertshaw and Taylor 1969), as it relies on both an increase in evaporative water loss from the skin (sweating) and from the respiration tract (panting) in order to help dissipate any heat load imposed upon it. However, unlike these other species, the rhinoceros also appears to have a high insensible C.M.L. This must also help in the heat dissipation of the animal as the mean insensible C.M.L. of 108.2 $g/m^2/hr$ recorded in the first experiments and the 284.3 $g/m^2/hr$ recorded in the second experiments would result in a heat loss of 1399.6 $kcal/m^2$ and 3684.4 $kcal/m^2$

respectively if it continued over a twenty four hour period.

The white rhinoceros was observed to be sweating in the shade at mid-day, while the respiration rate did not increase until the animal was exposed to a solar radiation load in the afternoon. It would thus appear that sweating is the major method of evaporative heat loss and that panting is used to supplement this in more severe conditions.

The body temperature variation through the day was 1.0°C in both the black and white species. This can be compared to other species, Bligh and Harthorn (1969) Schmidt-Nielsen (1957), Table 29, in which rectal temperature increases are considerably greater. Even though the environmental temperature change that some of these animals were experiencing was somewhat more than in the present work it would appear that the rhinoceros is able to maintain a relatively stable body temperature. However, Allbrook, Harthorn, Luck and Wright (1958) found that the rectal temperature of white rhinoceros increased from 34.5°C at sunrise to 37.5 at sunset. This latter value is very similar to the mean rectal temperature of 37.6°C found at 18.00 hrs. (one hour before sunset) in this work, though the increase in rectal temperature that they recorded through the day was considerably greater. Allbrook *et al* (1958) also identified large numerous sweat glands in the skin of

the white rhinoceros but found no evidence of sweating during the day using the quinizarin-sodium carbonate-starch method for detection of moisture loss. They did not report any changes in respiration rate nor any of the environmental conditions during these experiments. It can only be presumed that the conditions were insufficient to initiate any thermoregulatory response but were adequate to cause a rise in rectal temperature. The relatively labile body temperature and lack of C.M.L. observed could have been due to dehydration of the animals. Finch (personal communication) and Taylor (1969) observed a decreased C.M.L. in dehydrated oryx and eland and consequently a large diurnal variation in body temperature.

Under natural conditions the rhinoceros is sparsely distributed through East and South Africa. Within these areas it is often found in dry bush country where the solar radiation load is high (Dorst and Dandelot 1970). The ability of these animals to both sweat and pant would thus be of thermoregulatory benefit in this habitat.

An attempt to explain thermoregulation in the elephant presents rather a problem. In the earlier experiments body temperature increased through the day by a maximum value of 2.1°C (07.00 hrs. to 18.00 hrs.); in the later experiments the maximal increase was 1.1°C. Benedict and Lee (1936) study-

ing a 400 kg elephant, found a maximum daily variation of 1.6°C using the temperature of urine samples as an indicator of body temperature. However, this work was done in the Nutritional Laboratory of the Carnegie Institute of Washington in Boston, where environmental temperature only varied between 9°C and 13°C during the day. When Buss and Walker (1965) shot and measured body temperature in nine elephants here in East Africa, and then measured fecal temperatures of twenty more, they found variations in body temperature of 96.8°F to 98.2°F (36.0°C to 36.8°C). They concluded from this that "the relatively stable body temperature reflects the efficiency of the thermoregulatory mechanisims" as, presumably, this data was collected at various times through the day, so that the elephants concerned would have been experiencing varying solar radiation loads. Young (1972) also noted their ability to thermoregulate when he found that the rectal temperature of elephants in the Kruger National Park, South Africa, seldom varied by more than 1% from an average value of 36.4°C. However, he makes no mention of the circumstances or method of measurement of this. The stability of rectal temperature in the present work can also be used as confirmatory evidence for the ability of the elephant to thermoregulate, especially if it is again compared to rectal temperature changes in other species,

TABLE 29

The changes in rectal temperature through the day in
(from Bligh and Harthorn 1969).

		Rectal Temperature °C		
Animal		min.	max.	variation
Camel	1	35.8	39.1	3.3
	2	35.7	38.25	2.55
	3	35.1	37.7	2.6
Giraffe		37.75	39.1	1.35
Buffalo	1	36.9	40.1	3.2
	2	37.6	40.1	2.5
Eland	1	38.4	39.8	1.4
	2	38.3	39.85	1.55
Oryx		36.6	40.0	3.4

a number of East African mammals

Environmental Temperature °C		
min.	max.	variation
21.0	31.0	10.0
21.0	32.5	11.5
21.0	35.5	14.5
10.5	21.5	11.0
10.0	24.5	14.5
10.0	24.5	14.5
17.0	26.5	9.5
14.5	24.0	9.5
12.0	25.5	13.5

Table 29.

The African elephant is found distributed in woodland, grassland, sub-desert, and many intermediate ecological zones throughout much of tropical and sub-tropical Africa (Sale, personal communication) (Sykes 1971), and is thus often exposed to high air temperatures and also very high solar radiation loads (Appendix 2). In order to maintain its relatively stable rectal temperature it must control the amount of heat gained and/or lost from its body. However, the data presented here does not give any one obvious answer as to how they do this. It would seem to be due to a number of factors, as follows:-

1. Non-evaporative heat loss, i.e. convection, conduction, and radiation. The most important of these is the re-radiation of heat from the animals skin (See Appendix 2), but all are dependant on either the skin temperature or the skin/air temperature gradient.

At mid-day skin temperatures went up to 43°C, so creating a net flow of heat into the animal from the environment.

However, at other times during the day, when skin temperature was less than deep body temperature, there was a net flow of heat out of the body.

The skin temperature of the animal is dependant on the animals heat load and also the cutaneous vaso-motor tone (C.V.M.T.) in the skin. By altering

this C.V.M.T. the elephant is able to vary skin temperature and thus have some control over non-evaporative heat loss.

2. Evaporative heat loss:- C.M.L. appears to remain constant but high through the day, and must therefore be of assistance in dissipating heat from the animal. In the present work the mean C.M.L. in the early experiments was 64.8 $g/m^2/hr$ and in the later experiments 190.5 $g/m^2/hr$. This would account for a heat loss of 830.0 $kcal/m^2$ and 2,4688 $kcal/m^2$ respectively if it continued over a twenty-four hour period. Young (1972) found values as high as 540 $g/m^2/hr$ in the Kruger National Park in South Africa, where environmental temperatures were as high as 41°C, some 10°C higher than the present work.

As elephants do not have any sweat glands it might be expected that C.M.L. is, therefore, dependent on the vapour pressure gradient between the skin and the air - as insensible moisture loss normally is. It has already been mentioned (page 138) that the insensible C.M.L. in the elephant would be expected to fall at night. During the day an increased skin temperature (i.e. increased skin V.P.) and a decreased ambient V.P. would be expected to cause an increase in C.M.L. at 12.00 hrs. This, however, did not always occur (Table 24) and thus it would appear that changes in the V.P. gradient do not have a marked effect on insensible C.M.L. in

the elephant.

If this is the case, any alteration in C.V.M.T. that the animal might bring about would have little effect on C.M.L. Thus the elephant would appear to have no means of controlling this method of heat loss.

In the present work respiration rate did not alter significantly during the day and thus evaporative heat loss from the respiratory tract (panting) is not used to help regulate heat loss in the elephant.

3. The ear. The surface area of an adult elephant is increased by about 23.0% due to the ears (Appendix 3), so enhancing both evaporative and non-evaporative heat loss. In yearling elephants this increase is significantly greater (Appendix 3) and this might help the smaller animals to overcome the relatively greater heat stress (as discussed on page 140) that they experience. The ears are orientated vertically so that the direct solar radiation input at noon is minimal, the heat lost/heat gained ratio should be maximal, and the ears are of the greatest possible benefit to the animal. It was found, however, that the back of the ears was relatively cool at mid-day, some 5-6^{o}C cooler than the rest of the skin. This means that the skin/air temperature gradient is only half of that found elsewhere over the body surface and thus non-evaporative

heat loss is relatively low.

Buss and Estes (1971) found a positive correlation of ear flapping and environmental temperature, from which they suggest that this has some thermoregulatory advantage. Ear flapping would have little effect on non-evaporative heat losses from the ear, convective losses being of minimal importance (Appendix 2). However, it would be an advantage if sweating was taking place, as suggested by Sykes (1971), or if insensible C.M.L. was high, as suggested by Young (1972). In the present study no sweat glands were found to be present in the ear and the diffusion of moisture through the skin of the ear did not appear to be any greater than over the rest of the body. So that calculations made after the experiments in February 1971 (Appendix 2) show that heat lost from the ears (306 kcal/hr) was 19.3% of the total heat loss. This is not even in proportion to the surface area due to the ears, some 24.1%, and only accounts for 14.5% of the calculated heat load at the time (2252 kcal/hr). Even so, Young (1972) considers them to function in a manner similar to the radiator of a motor car. He mentions the work of Luck and Wright (1965) who found that the blood that flows through the ear is cooled by 9°C before flowing back into the body. Although Buss (personal communication) classifies the movement of blood through the ear as "significant" the effect that this

cooled blood would have in cooling the large body mass of an elephant would appear to be somewhat limited. If, however, the venous drainage from the ear surrounded the arterial supply to the brain and thus acted as a counter-current heat exchanger, it would cool the brain and thus be of considerable advantage to the animal. By this means body temperature could be labile yet the temperature of the brain, which is the most thermosensitive organ, could be kept more or less constant.

It has already been mentioned that the much quoted phenomena of elephants being "wet behind the ears" was not observed, unless, as both Buss (personal communication) and Young (1972) also mention, the elephant sprayed the ear with moisture with its trunk. In this case, heat loss from the ears would be markedly increased.

By altering the C.V.M.T. of the blood vessels on the inner side of the ear the elephant makes the best use of its ears as structures for losing heat from the body. As the blood vessels are very close to the surface of the inner side of the ear it is possible to see the state of C.V.M.T. A visual assessment of this showed that there was cutaneous vasodilation at 18.00 but not at 07.00 hrs. or 12.00 hrs. This caused the skin temperature of the inside of the ear to be higher than the rest of the body at 18.00 hrs. Due to this the loss of heat from the

ears is relatively more important at the cooler part of the day, when heat can be lost easily through non-evaporative means. Thus through conduction, convection, and radiation - especially re-radiation to the cool night sky - the elephant is able to lose heat that it has had to store during the day, plus its metabolic heat load.

The significance of the ears in the cooling of the elephant, therefore, appears to depend on:

(i) Any evaporative cooling that occurs through behaviour; i.e. water or saliva spreading (see below).

(ii) C.V.M.T. especially in relation to increased heat loss in the cooler part of the day due to vasodilation.

Otherwise the ears only appear to be important in increasing the surface area of the animal and possibly through localized cooling of the brain.

4. Behavioural thermoregulation. Even the casual observer will have noticed that elephants seek the shade during the heat of the day. In the Kruger National Park this can result in a drop of up to 4.5°C in the air temperature to which the elephant is exposed (Young 1972), as well as decreasing the direct solar radiation input to the animal. Also, they will often cover themselves with mud or water if the opportunity arises. The elephant will even draw up with its trunk water present in the mouth or

oesophagus in order to spray itself on very hot days. Glover, (personal communication), and Young (1972) classify this water as stomach contents, which presumably, must have been regugitated to make them available. It is possible, however, that this water is in fact saliva, and that this behaviour is similar to saliva spreading that has been observed in rats, Hainsworth (1968) and monotremes (Dawson 1972). This water is particularly used to wet the backs of the ears (Young 1972).

The animal used in the present work was one of four orphaned elephants at Tsavo and these animals always went to the water when returned to their pens at mid-day. They always drank large amounts of water but on the hotter days they also splashed it over themselves, which would increase their evaporative heat loss.

Behaviour is, therefore, very important in the thermoregulation of the elephant, probably more in lowering the heat load that it is experiencing than in increasing heat loss. As solar radiation constituted approximately 91% of the heat load on the experimental animal used here (Appendix 2), any decrease in this would have a significant effect on its heat balance. However, as the elephant possesses no sweat gland with which to control C.M.L. the increase in evaporative heat loss, as a result of spraying or bathing in hot weather, must also be very

important.

By a combination of these four factors the elephant was able to maintain a relatively stable body temperature in the environmental conditions during the experiments. It's ability to thermoregulate in more severe environmental conditions would, however, appear to be rather limited. If it was exposed to a high air temperature, a high ambient humidity, and an unavoidable solar radiation load, it is unlikely it would be able to maintain a stable body temperature. Although such conditions would also present a heat stress to any animal, other East African mammals would rely on a controlled increase in evaporative water loss to overcome it. In the elephant this could not happen. However, the large body size of the elephant means that the heat required to increase its rectal temperature is fairly considerable. This would tend to limit the increase in rectal temperature in these circumstances.

An attempt was made to extend the calculations mentioned in (1) and (2) above to try and work out the relative importance of each of these factors to the elephant's thermoregulation. However, it proved impossible to calculate a heat input/heat output balance for the animal. Finch (1972) was able to obtain this balance because the animals were measured every hour through the day while restricted in a crush. In the present experiments the animals were

measured at 07.00 hrs., 12.00 hrs. and 18.00 hrs. and were free to move around in between. During this time they were likely to have been in the shade, or near different types of radiative surfaces so that no true integrated value of heat input or output could be obtained. However, it was not possible to improve on this arrangement, as permission could not be obtained to pen the animals up through the day. It must, therefore, suffice to calculate the heat outputs without expecting them to balance with the heat inputs. In so doing, it is then possible to obtain an idea of the relative importance of the various methods of heat dissipation, including the ears. However, in the present experiments the elephant was able to maintain a relatively stable body temperature and must, therefore, have been able to balance heat input and output in practice.

In the zebra the increase in rectal temperature during the day varied between 0.6°C and 2.4°C, the maximum temperatures recorded on each of the four days being between 38.4°C and 38.8°C. These increases in rectal temperature are somewhat less than most of the increases in deep body temperature reported by Bligh and Harthorn (1969) in several other wild African ungulates experiencing similar environmental temperature changes (Table 29). Also, they are less than the increase in rectal temperature of the donkey on heat exposure as reported by Maloiy

(1971) whose animals had increases in rectal temperatures of between 3-4°C. Although the environmental temperature (40°C) in these climatic chamber experiments (Maloiy 1971) was considerably higher than in the present field work (maximum 31°C) the animals were not exposed to the high solar radiation load, which is the biggest heat stress factor in the field. It is interesting to note that most of the increase in rectal temperature in the present experiments occurred before 11.00 hrs. The respiration rate during this time had normally increased to the maximum value recorded, while C.M.L. was still gradually increasing. It would therefore appear that the increase in respiration rate was unable to prevent an increase in body temperature. Only when C.M.L. had risen considerably did the rectal temperature begin to stabilise. However, even with a high C.M.L. around mid-day, rectal temperature rose slightly until mid-afternoon, when the heat load, rectal temperature, and sweating rate all started to fall.

The pattern of respiration rate through the day were similar in the two animals. However, Zebra 1 consistently had a higher respiration rate (55.2 times/min) than Zebra 2 (48.9 times/min), this difference being significant at the 0.1% level. Since respiratory evaporative heat loss probably represents a small proportion of the total heat loss

the higher respiration rate in Zebra 1 is unlikely to have been of any major thermoregulatory benefit to the animal. This is supported by the fact that there was no significant difference in C.M.L. between the two animals.

From the present, rather limited, results it appears that the increase in respiration rate on heat exposure is fairly consistent. It is the level of C.M.L. which varies depending on the severity of the heat load. This is different from the rhinoceros in which C.M.L. remained high during a period of partial shading while respiration rates only increased when it was exposed to a higher solar radiation load.

Although no other work on thermoregulation in the zebra has been published, a comparison can be made to other members of the genus, Equus. The values for evaporative water losses reported here, 388 $g/m^2/hr$ and 367 $g/m^2/hr$ in the two zebras, are similar to the value of 360 $g/m^2/hr$ reported in the donkey exposed to air temperatures of 35-40°C in the desert (Schmidt-Nielsen, Schmidt-Nielsen, Jarnum and Houpt, 1957). It is, however, a little greater than both the values reported in the burro under similar conditions, 250-270 $g/m^2/hr$ (Bullard, Dill & Yough 1971) and the value for "moderate sweating" in the horse, 300 $g/m^2/hr$ (Nakayos, Takagi, Arimura and Ueno, 1957). Work in a climatic chamber has produced evaporative water losses very much lower than

all these values; Allen and Bligh (1969) found an evaporative water loss of 166 $g/m^2/hr$ and Maloiy (1971) a value of 130-140 $g/m^2/hr$. It would thus appear that the C.M.L. in the zebra is not significantly different from other equines which are experiencing similar environmental conditions. However, this is always augmented by panting, a phenomena which does not always occur in other equines.

As the Zebra inhabits open grassy plains and well-grassed woodlands in Eastern and Southern Africa (Dorst and Danolelot 1970) they will invariably be experiencing a high solar radiation load and a high air temperature. In these circumstances, the ability to lose heat through both sweating and panting would appear to be a considerable thermoregulatory advantage.

In considering each animal individually most of the points mentioned in the introduction have been clarified. It is unfortunate that the experiments on the elephant, the rhinoceros, and the zebra had to be confined to their gross thermoregulatory responses to solar radiation loads. A comparison of the control of the sweat glands that were found would have been interesting and would have allowed a much better placement of the animals into the general picture of mammalian thermoregulation. It must, therefore, suffice to classify the elephant as unique in respect of its thermoregulation, as

it neither sweats nor pants. The rhinoceros and the zebra, as they both pant and sweat in order to thermoregulate, are typical of the majority of large terrestrial mammals.

The more controlled conditions of the climatic chamber allowed a much more systematic investigation of thermoregulation in the three primate species to be carried out. As a result of this, a number of conclusions concerning the thermoregulatory inter-relationships of these species have already been made (page 133).

APPENDIX I

The programme overleaf is in Fortran IV for use on an I.C.L. 1900 computer. It will give a series of tables with wet bulb temperature across the top for every 0.1°C, from 10°C to 20°C. Wet/dry bulb temperature difference is given down the side of each table, also tabulated for each 0.1°C difference.

A millivolt difference (M.V.D.) was obtained from the thermocouple pair(s). This was then converted into a wet bulb temperature difference between the thermocouples, as a previous calibration of M.V.D. and temperature difference had been made.

Two values corresponding to the two wet bulb temperatures at the dry bulb temperature of the reading could then be read off these output tables. The difference of these two values was then the cutaneous moisture loss at the time of the reading.

To change wet bulb temperature range the cards numbered 21, 28, 31 and 38 should be changed to give the range required.

For atmospheric pressure other than 630mm Hg (the pressure during the present experiments) card 19 should be changed.

If a different size ventilated capsule and/or flow rate is used the factor in card 44 should be changed.

GENERAL LISTING (XRLP) 17/08/72

```
      LIST (LP)
      PROGRAM(S44A)
      INPUT 2 = CR7
      OUTPUT 3= LP7
      END
       MASTER S44A
     0 DIMENSION VPUA(300),VPUB(300),TD(210),SLP(300),
     I           TW(11),SWR(11,210),TTLA(10),TTLB(10)
C
     1 FORMAT      (12F4.2)
     2 FORMAT(10A8)
     5 FORMAT (5(10(10X,F10.1,10X,10F9.2)/),1H1)
     7 FORMAT ((12X,10A8,12)/(12X,10A8))
     9 FORMAT ((110X,5HPAGE I2,1H-,I1)/((25X,3HTW=,10F9.1)//))
C
       READ (2,1)  (VPUA(I),I=70,300,10)
       READ (2,1)  (VPUB(I),I=70,300,10)
        READ(2,2)TTLA,TTLB
       CON = 0.0065*(13.0/5.5)*4.7
C FOR OTHER ALTITUDES CHANGE THE 13.0 TO APPROPRIATE B DIFFERENCE
       DO 12 I = 100,200,10
       DELA=(VPUA(I+10)-VPUA(I))*0.1
       DELB=(VPUB(I+10)-VPUB(I))*0.1
       IP8=I+8
       DO 12 J=I,IP8
        VPUA(J+1)=VPUA(J)+DELA
    12 VPUB(J+1)=VPUB(J)+DELB
       DO 13 I = 100,209
    13 SLP(I)=(VPUA(I)-VPUB(I))*0.01
C
       DO 17 J = 100,200,10
       M=J/10
       DO 14 I=2,11
       NI=J+(I-2)
       FI=NI
       TW(I)=FI*0.1
       SWR(I,1)=VPUA(NI)
       DO 14 K=1,209
       SWR(I,K+1)=SWR(I,K)-SLP(NI)
       FK=K-1
       TD(K)=FK*0.1
       SWR(1,K)=TD(K)
       HOLD=SWR(I,K)+TD(K)*CON
    14 SWR(I,K)=(HOLD*4702.0)/(TW(I)+TD(K)+273.0)
       WRITE (3,7) TTLA,M,TTLB
       DO 17 MPAGE=1,4
       KHI=50*MPAGE
       KLO=KHI-49
       WRITE (3,9) M,MPAGE,(TW(I),I=2,11)
    17 WRITE (3,5) ((SWR(I,K),I=1,11),K=KLO,KHI)
       STOP
       END
       FINISH
075108040861092109851052112411991279136414541549
164917551866198421092240237825242677283830083186
024602980354041404770544061406900769085309421037
113612421353147015941724186220072160232024892667
```

SECTION

TABLE OF SWEATING RATES CORRECTED FOR ALTITUDE,
WET BULB TEMPS ACROSS TOP, WET-DRY TEMP DIFFERENCE DOWN THE LEFT, IN CENTIGRADE

APPENDIX 2

Heat balance of the elephant

The following calculations are based on Porter and Gates (1969), in which the animal is treated as a cylinder in order to calculate the total heat load. The ears have been considered as vertical plates held permanently extended away from the body of the elephant. In this state they would be of maximum benefit to the animal if they are to function as structures for increasing heat loss.

The probable reason for the failure of input and output to balance has been discussed previously.

Appendix of Elephant Heat Balance

	Tues.	Wed.
a = absorbtion coefficient for skin surface	= 0.925	0.925
S = radiation direct from the sun = (total solar radiation - radiation from the sky).		
s = radiation from the sky	= 0.50	0.41
r = radiation reflected from the ground	= 0.31	0.31
(S + s) = total solar radiation	= 1.46	1.50
Ta = air temperature in ^{o}A	= 304.7	304.7
Tg = ground temperature in ^{o}A	= 326.0	332.0
e = Vapour pressure in millibars	= 19.4	28.4

$$Qabs = \frac{a(2S/\pi) + as + ar(S + s) + Ra + Rg}{2}$$

To calculate Qabs

direct radiation from the sun $= a(2S/\pi)$

Tuesday $= 0.925\ (2(\frac{1.46 - 0.50}{\pi}))$

$= 0.561\ cal/cm^2/min$

Wednesday $= 0.925\ (2(\frac{1.50 - 0.41}{\pi}))$

$= 0.642\ cal/cm^2/min$

mean $= 0.601\ cal/cm^2/min$

radiation from the sky $= a \times s$

Tuesday $= 0.825(0.5) = 0.413\ cal/cm^2/min$

Wednesday $= 0.825(0.41) = 0.338 cal/cm^2/min$

mean $= 0.376\ cal/cm^2/min$

radiation from the ground $= a \times r \times (S + s)$

Tuesday $= 0.825 \times 0.31 \times 1.46$

$= 0.373\ cal/cm^2/min$

Wednesday $= 0.825 \times 0.31 \times 1.50$

$= 0.384\ cal/cm^2/min$

mean $= 0.379\ cal/cm^2/min$

long wave radiation from the air $= \bar{\sigma}\ Ta^4\ (0.44 + 0.08\ \sqrt{e})$

Tuesday $= 0.550\ cal/cm^2/min$

Wednesday $= 0.602\ cal/cm^2/min$

mean $= 0.576\ cal/cm^2/min$

long wave radiation from the ground $= \bar{\sigma}\ Tg^4$

Tuesday = 0.919 cal/cm^2/min

Wednesday = 0.975 cal/cm^2/min

mean = 0.947 cal/cm^2/min

mean Qabs = 2.879 cal/cm^2/min

average radiation absorbed by the animal = ½ Qabs

= 1.439 cal/cm^2/min

Total mean heat load from the sun on the animal

(S.A. = surface area = 2.27m^2)

= ½ Qabs x S.A. x 60 cal/hr

= 1.439 x 2.27 x 600 kcal/hr

= 1,959.9 kcal/hr

Metabolic Heat Load (W = weight of the animal = 300kg)

= $70/W^{0.73}$ kcal/24hr

= 189.0 kcal/hr

Total mean Heat Load = 2,148.9 kcal/hr

Solar Radiation as % of Total Heat Load = 91.2%

	Tues.	Wed.
Ts = skin temperature in ^oA	311.0	313.0
Δt = skin/air temperature gradient ^oC	6.3	8.9
h = conductance in cal/m^2/hr/ ^oC t	2187.4	2386.1
C.M.L. = cutaneous moisture loss g/m^2/hr	73.1	75.6

	Tues.	Wed.
L.H. = latent heat of evaporation of water cal/g	540	
ΔTr = change in rectal temperature ^{o}C	0.6	1.6
ΔTs = change in skin temperature ^{o}C	7.0	10.0
D = time taken for ΔTr and ΔTs (hrs)	5.33	5.25

Heat lost by radiation = σ Ts^4 x S.A.

Tuesday = 1035.2 kcal/hr

Wednesday = 1059.0 kcal/hr

mean = 1047.1 kcal/hr

Heat lost by convection = h x S.A. xΔt

Tuesday = 2187.4 x 2.27 x 6.3

= 31.3 kcal/hr

Wednesday = 2386.1 x 2.27 x 8.9

= 48.2 kcal/hr

mean = 39.8 kcal/hr

Evaporative Heat Loss = C.M.L. x L.H. x S.A.

Tuesday = 73.1 x 540 x 2.27

= 89.7 kcal/hr

Wednesday = 75.6 x 540 x 2.27

= 92.6 kcal/hr

mean = 91.2 kcal/hr

Heat Stored = $\frac{0.83 \times W \times (4\Delta Tr + \Delta Ts)}{5 \times D}$

Tuesday = $\frac{0.83 \times 300 \times (2.4 + 7.0)}{5 \times 5.33}$

= 87.8 kcal/hr

Wednesday $= \frac{0.83 \times 300 \times (6.4 \times 100)}{5 \times 5.25}$

= 155.5 kcal/hr

mean = 121.6 kcal/hr

mean Total Heat Loss from the body

= 1299.7 kcal/hr

Ears

	Tues.		Wed.	
	front	back	front	back
Ts ^{o}C	38.0	32.0	40.0	36.0
h	2589.7	-	2824.8	2364.6
S.A. of ear	$0.3116m^2$			
Δt	6.3 approx.	0	8.9	4.9

Heat loss from the front of the ears.

Radiation = $\bar{6}Ts^4$ x S.A.

Tuesday = 142.1 kcal/hr

Wednesday = 144.7 kcal/hr

mean = 143.4 kcal/hr

Convection = h x S.A. x Δt

Tuesday = 5.1 kcal/hr

Wednesday = 7.8 kcal/hr

mean = 6.4 kcal/hr

Insensible = C.M.L. x S.A. x L.H.

Tuesday = 12.3 kcal/hr

Wednesday = 12.7 kcal/hr

mean = 12.5 kcal/hr

Total Heat Loss from the front of the ears (F)

= 162.6 kcal/hr

Heat loss from the back of the ears.

Radiation = $\bar{6}Ts^4$ x S.A.

Tuesday = 131.4 kcal/hr

Wednesday = 136.6 kcal/hr

mean = 134.0 kcal/hr

Convection = h x S.A. x t

Tuesday $\triangleq$ 0, as Ts $\triangleq$ T air

Wednesday = 3.61 kcal/hr

mean = 1.8 kcal/hr

Insensible = C.M.L. x S.A. x L.H.

Tuesday = 12.3 kcal

Wednesday = 12.7 kcal

mean = 12.5

Total Heat Loss from the back of the ears (B)

= 148.3 kcal/hr

Total Heat Loss from ears (F + B)

= 310.9 kcal/hr

Total Heat Loss from body and ears = 1,610.6 kcal/hr

% heat loss due to ears = 19.3%

Total heat load = 2,148.9 kcal/hr

Total heat loss as % of Total Heat Load = 74.9%

Total heat loss from ears as % of Total Heat Load = 14.5%

Area of ears = 0.623 m^2

Area of body = 2.270 m^2

Total S.A. = 2.582 m^2

% of S.A. due to ears = 24.12

APPENDIX 3

Calculations of the area of the ear in relation to the total surface area of the elephant.

Data below was taken from photographs kindly lent for use by Mr. T. Corfield, Tsavo Research Centre, Voi, Kenya.

All pictures used were taken at right angles to the animal while the ears were flat against its side.

Adult (areas in cm^2 measured from photograph)

Body area (π.d.l.)	Total area of ears	Increased area due to ear %
96.5	18.0	18.65
24.4	7.2	29.50
33.0	9.1	27.57
69.7	14.2	21.37
32.8	11.0	33.53
48.4	8.2	16.94
32.5	5.6	17.22
35.4	6.9	19.49
37.7	8.5	22.54
32.6	8.3	25.46
33.2	9.0	27.10
75.7	21.9	28.92
53.5	12.6	23.55
40.0	11.2	28.00
38.5	7.4	19.22

Body area (π.d.l.)	Total area of ears	Increased area due to ear %
19.8	4.2	21.21
22.6	5.9	26.10
130.5	22.6	17.31
31.1	6.9	22.18
113.5	21.3	18.76

Yearling (1 and 2 areas in cm^2 from photograph)

(3 actual area m^2 of experimental animal)

	Area of body	Total area of ears	% increase due to ears
1.	16.0	4.3	26.90
2.	4.0	1.3	32.51
3.	2.27	0.62	27.32

mean for adult = 23.23%

mean for yearling = 28.91%

dif. = 5.68% = signif. 2% level

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