ACUTE THERMAL AND CARDIORESPIRATORY RESPONSES TO TRANQUILIZATION AND ELECTRO-ANESTHESIA IN SHEEP

Thesis for the Degree of Ph. D.
MICHIGAN STATE UNIVERSITY
WENDELL FEY HOFMAN
1970

This is to certify that the

thesis entitled

Acute Thermal and Cardiorespiratory Responses to Tranquilization and Electro-anesthesia in Sheep

presented by

Wendell Fey Hofman

has been accepted towards fulfillment of the requirements for

Ph. D. degree in Physiology

Earl D. Reigh
Major professor

Date Aug 11, 1970

O-169

ABSTRACT

ACUTE THERMAL AND CARDIORESPIRATORY RESPONSES TO TRANQUILIZATION AND ELECTRO-ANESTHESIA IN SHEEP

By

Wendell Fey Hofman

Acute thermal and cardiorespiratory responses were examined in closely shorn adult sheep administered propiopromazine HCl (1.0 mg/kg) and electro-anesthesia (700 c.p.s. A.C., bitemporal electrodes) together, and independently during a 330 minute whole body exposure to three environmental temperatures, 5°C., 25°C. and 35°C. Rectal (T_{re}) and 7 skin temperatures, oxygen consumption ($\dot{V}_{O_2}$), respiratory frequency (f), respiratory evaporative water loss (E), arterial blood pressure, heart rate, arterial pH, arterial carbon dioxide tension (P_{aCO_2}), bicarbonate concentration, and arterial hematocrits were measured. Values for alveolar ventilation ($\dot{V}_A$) and tidal volume (V_T) were computed. At the 5°C. exposure, T_{re}'s decreased in both the tranquilized (0.8°C.) and tranquilized-electro-anesthetized sheep (1.6°C.) after 2 hours of their respective treatments. The depression in T_{re} of these groups was associated with an inhibition of shivering which was related to a decreased $\dot{V}_{O_2}$, a suggested increase in net peripheral heat loss (ears and

face) and increased heat dissipation via E. Nevertheless, the sheep given electro-anesthesia alone showed no significant change in T_{re}. Little difference was recorded in T_{re}'s for tranquilized and/or electro-anesthetized sheep from control values at 25°C. However, an elevation in heat production (increased $\dot{V}_{O_2}$) was observed in the tranquilized and tranquilized-electro-anesthetized animals which overcame the increased net peripheral heat loss from vasoactive areas (i.e., ears and forelimbs). The elevation in T_{re} of the tranquilized (0.9°C.), tranquilized-electro-anesthetized (1.5°C.) and electro-anesthetized sheep (1.5°C.) from control measurements at the 35°C. exposure was attributed to a reduction in E attendant to depression of f. At all ambient temperatures, P_{aCO_2} was increased in tranquilized-electro-anesthetized sheep with smaller elevations observed when treatments were given separately. These elevations in P_{aCO_2} were related to decreases in arterial pH, f and $\dot{V}_A$. Arterial blood pressure was decreased in animals administered propiopromazine alone and with electro-anesthesia. Heart rate was increased in proportion to the fall in blood pressure. The animal administered electrical anesthesia alone exhibited an increase in blood pressure even though heart rate was increased only at the 35°C. exposure. Arterial hematocrits were increased in animals under electro-anesthesia but not in sheep given only propiopromazine.

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Wendell Fey Hofman

A THESIS

Submitted to
Michigan State University
in partial fulfillment of the requirements
for the degree of

DOCTOR OF PHILOSOPHY

Department of Physiology

1970

ACKNOWLEDGMENTS

I would like to express my sincere gratitude to Dr. G. D. Riegle, my major advisor, for his encouragement, guidance and help during my graduate program.

I would also like to thank Dr. T. Adams and Dr. S. R. Heisey for their advice and assistance with this research effort. My appreciation is also extended to Dr. J. E. Nellor and Dr. K. E. Moore for their suggestions and counsel.

I would like to express my appreciation to the entire staff of the Endocrine Research Unit for their support and help and for providing facilities to conduct this study.

I also wish to thank my wife, Karen, for her patience and sacrifices during my doctoral program.

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TABLE OF CONTENTS

LIST OF TABLES

INTRODUCTION

The application of trans-cranial electrical currents of various voltages and frequencies for the production of general anesthesia has been designated as "electrical anesthesia" or "electro-anesthesia." The impetus for research into electro-anesthesia is related to the advantages it affords over conventional chemical anesthetics. Electrical anesthesia has provided an efficacious technique for obtaining nearly instantaneous induction, reliable control and rapid recovery from anesthesia with few post-anesthetic complications. Nevertheless, like chemical anesthetics, electro-anesthesia is not without untoward physiological side effects. These include hyperthermia, hypertension tachycardia and muscle spasms. In order to ameliorate some of these effects and also to reduce electrical current requirements, concurrent drug therapy has been used.

In ruminants phenothiazine tranquilizers have been recommended as preanesthetic medications, but no studies have been undertaken to detach explicitly those physiological abberations associated with electro-anesthesia from those attributable to drug treatment.

This study describes some thermoregulatory and cardiorespiratory responses of sheep, acutely exposed to a range of environmental temperatures, when electrical anesthesia and a phenothizaine tranquilizer were administered independently and in combination.

LITERATURE REVIEW

Homeotherms are animals which utilize many physiological systems to maintain their deep body temperature within narrow limits. Mechanisms of thermoregulation in homeotherms have been widely reviewed (Hardy, 1961; von Euler, 1961; Hammel, 1968).

There have also been extensive reviews on both electro-anesthesia (Stephen, 1959; Smith et al., 1967; Herin, 1968) and substituted phenothiazine tranquilizers (Dundee, 1954; Dobbin et al., 1956; Lear, 1966). However, a brief historical background of these treatments is presented below.

Historical Review of Electro-Anesthesia

The studies of French professor Leduc (1902, 1903) and those with his associate (Rouxeau (1903a, 1903b, 1903c) were the first extensive investigations into the use of electrical currents solely for the purpose of anesthesia. They produced sleep-like states in dogs and rabbits by means of interrupted direct current. The current was dispensed through electrodes positioned on the back of the head (cathode) and over the kidney (anode). A wheel interrupter was used to produce a frequency of 100 to 200 cycles per

second with a pulse duration of 1 millisecond. A potential of 0 to 80 volts was supplied by large capacity storage batteries. Anesthesia was obtained with a frequency of 100 cycles per second and current intensities of 2 and 10 milliamperes in the rabbit and dog, respectively. A sudden high amperage input followed by a transient decrease in current intensity was utilized to induce anesthesia. The success attained in animal experiments, encouraged Leduc to attempt electro-anesthesia on himself. The experience was described as a "nightmare state" during which he noted a sensation of numbness, dimming of vision and loss of motor function.

The studies of Leduc and Rouxeau resulted in a reproducible technique for electrical anesthesia and served as a stimulus for further investigations in the succeeding years. Robinovitch (1906), a student of Leduc's, successfully obtained deep anesthesia in rabbits by use of an interrupted direct current similar to Leduc's. A few years later, Robinovitch (1914) wrote a chapter in Gwathmey's textbook Anesthesia relating a detailed description for the successful application of electro-anesthesia to humans.

Even though Leduc maintained that electro-anesthesia could be produced only by pulsatile direct current, van Harreveld and Kok (1934) successfully obtained anesthesia using 60 cycle per second, sinusoidal wave alternating current (300 milliamperes) delivered to dogs via bitemporal electrodes. A few years later, van Harreveld and co-workers

(1942) compared the anesthetic properties of different current forms in the dog. The anesthetic potential of constant direct current was regarded as less than that of pulsed direct or alternating currents.

Frostig and colleagues (1944) studied the effect of 60 cycle per second alternating current in dogs and humans. They described two types of electro-anesthesia in the dog: a narcotic type which resembled sleep, with slow deep breathing and bradycardia; a kinetic type characterized by general restlessness with accelerated respiratory and heart rates.

Knutson (1954) induced electro-anesthesia in 25 dogs for periods as long as 3 hours with a sinusoidal wave alternating current of 700 to 1500 cycles per second (25 to 80 milliamperes) applied through bitemporal electrodes. Knutson and co-workers (1956) administered the same current form to human subjects, but were unable to obtain successful anesthesia because of cardiovascular complications.

Anan'ev and co-workers (1957) used a rectangular wave alternating current at a frequency of 100 cycles per second (pulse duration of 1 to 1.4 milliseconds) applied in combination with a direct current component. They reported a total average current (A.C. plus D.C.) of 7 to 10 milliamperes was sufficient for general anesthesia in dogs, but higher current intensities facilitated muscle relaxation. The current was applied through electrodes positioned directly over the eye and on the mastoid region, over the

occiput. Smith et al. (1961), using a modification of Anan'ev's technique, successfully induced anesthesia in 200 dogs, 6 monkeys, and 1 chimpanzee.

Hardy and colleagues (1961) used 700 cycle per second alternating current to obtain anesthesia in dogs for periods as long as 8 hours. Histological examination of the brains of these animals revealed no gross microscopic injury. Herin (1964) was the first to use bitemporally positioned hypodermic needle electrodes to administer 700 cycle per second alternating current to dogs. Encephalographic patterns recorded from 20 animals immediately before and after electro-anesthesia were not noticeably different. Short (1965b) also employed subcutaneously placed hypodermic needles to deliver 700 cycle per second current (20-85 milliamperes) to over 100 animals including cattle, horses, sheep and pigs.

Basically, two types of anesthesia-producing current forms have developed, 700 to 1500 cycle per second alternating currents and 100 cycle per second pulsatile direct current. Each appears to have selective advantages over the other. Alternating current allows the use of hypodermic needle electrodes with negligible tissue damage or burning and minimal electrode iontophoresis. Advocates of direct current claim a more flexible current in meeting the anesthetic requirements of the animal as well as a smoother induction phase. Basically, however, the anesthesia and

concurrent side effects (hyperthermia, hypertension, somatic muscle hypertonicity) are qualitatively similar.

Historical Review of Phenothiazine Tranquilizers

According to a historical development of the phenothiazine tranquilizers by Lear (1966), the phenothiazine molecule was first synthesized by Bernthsen in 1883. A historical review by Jarvik (1967) relates that it was nearly 50 years later before phenothiazine was first used as an anthelmintic, urinary antiseptic and insecticide. In 1940, phenothiazine and some of its newly synthesized derivatives were tested for other pharmacological properties by French investigators (Lear, 1966). These investigators observed that substituted phenothiazines had potent antihistaminic properties and produced transient feelings of annihilation and lethargy. Laborit (1951) was the first to introduce promethazine as a potentiator of anesthesia. Dundee (1954) relates that in 1950 M. P. Charpentier synthesized chlorpromazine, a compound structurally resembling promethazine but with a more pronounced central depressant action. Laborit and co-workers (1952) reported that chlorpromazine also facilitated anesthesia and was capable of producing "artificial hibernation" without loss of consciousness.

Courvoisier and colleagues (1953) conducted the first comprehensive investigations on chlorpromazine. They

reported that chlorpromazine had a definite depressant action upon the central and autonomic nervous systems, enhanced effects of barbiturates and narcotics, depressed the hypothalamic thermoregulatory centers and the medullary chemoreceptor trigger zone and demonstrated some antifibrillatory and antishock potential. Currently, chlorpromazine and numerous closely related derivatives are employed clinically in human and veterinary medicine as preanesthetics, tranquilizers and antiemetics.

Cardiorespiratory and Biothermal Effects of Electro-Anesthesia

Cardiovasculature

Most cardiovascular abberations during electro-anesthesia have occurred during initial current application. Ivy and Barry (1932) observed that a rapid, high amperage induction produced transient bradycardia and hypotension. This was attributed to stimulation of the medullary cardio-inhibitory center. Subsequent current reduction resulted in tachycardia and an elevation in blood pressure which was ascribed to stimulation of the vasoconstrictor center. Vagotomy or atropine, together with rapid induction, diminish the bradycardia and hypotension concurrent with the rapid induction technique (Frostig et al., 1944). Nevertheless, several other investigators using a sudden induction procedure have reported only hypertension and tachycardia (Knutson, 1954; Hardy et al., 1961; Herin, 1963a; Photiades

et al., 1963; Price and Dornette, 1963). Herin (1969) found that heart rate may be depressed during induction if the time-course of current application is too rapid. The hypertension associated with "crash" induction has been prevented by adenergic blocking agents (Cameron and McIntyre, 1963). Hardy et al. (1961) attributed the hypertension to general stimulation of the sympathetic nervous system, since it was not observed in sympathectomized dogs. In contrast, studies utilizing a gradual induction technique have shown that blood pressure may either increase or decrease during induction (Anan'ev et al., 1961; Smith, 1964).

When anesthetizing currents are adjusted to maintenance levels, cardiac and blood pressure responses are variable. Increased systemic arterial blood pressure, bradycardia, and decreased cardiac output have been reported in electro-anesthetized dogs (Elkin and Vasko, 1966), but tachycardia and hypertension have been a more common observation (Knutson, 1954; Knutson et al., 1956; Hardy et al., 1961; Price and Dornett, 1963). Herin (1968) studied electro-anesthesia in dogs produced by various current wave forms and found an increase in pulse rate and blood pressure with all current forms tested.

Massion and Downs (1969) examined peripheral hemodynamics in an isolated forelimb preparation which was perfused with blood from a dog under electro-anesthesia. The vasoconstriction observed in the isolated limb was

attributed to catecholamine liberation in the intact anesthetized animal. In contrast, cross circulation studies with rabbits showed elevated arterial blood pressure only in the animals under electro-anesthesia (Volpitto et al., 1962). Nevertheless, elevated plasma catecholamimes have been reported and postulated to be a contributing factor to the hypertension observed in electro-anesthetized dogs (Hardy et al., 1961). Wood and co-workers (1964) found hypertension was abated with the placement of electrodes in certain locations (e.g., roof of the mouth and vertex of skull). The cardiovascular responses were probably dampened in that all dogs were premedicated with pentobarbital. Some cardiac arrhythmias have also been associated with electro-anesthesia (Frostig et al., 1944; Knutson, 1954; Hardy et al., 1961; Volpitto et al., 1962).

In dogs under electro-anesthesia Frostig and colleagues (1944) found that red blood cell counts were increased apparently due to splenic contraction. The studies of Martin (1966) showed that the hematocrit increased as current intensity increased. The hematocrit of splenectomized dogs did not increase under 700 cycle per second anesthesia (Powers and Wood, 1964). Herin (1968) showed an elevated hematocrit in dogs only with certain wave forms.

Respiration

Ventilatory responses to electro-anesthesia have not been extensively studied although changes in respiratory frequency have been used to gauge the rapidity of current application during induction and to evaluate anesthetic depth (Wulfsohn and McBride, 1966; Smith, 1963; Ramo Rao, 1966). Some investigators have reported increases in respiratory frequency during electro-anesthesia (Geddes, 1964; Himes, 1965), whereas Smith and co-workers (1964) reported a decrease in respiratory frequency, a small decrease in respiratory minute volume, and a doubling of tidal volume from preanesthetic measurements. In addition, arterial hemoglobin saturation remained above 91% and arterial P_{CO_2} rose significantly. On the other hand, a small decrease in arterial P_{CO_2}, with a slightly depressed respiratory frequency, was reported in dogs anesthetized with 700 cycle per second sine wave current (Herin, 1968). In squirrel monkeys, arterial P_{CO_2} increased and P_{O_2} decreased under electrical anesthesia (Larson and Sances, 1968).

Data reported by Shaikh and colleagues (1968) suggested hyperthermic polypnea was inhibited during electro-anesthesia in sheep premedicated with a substituted phenothiazine tranquilizer (propiopromazine).

A marked metabolic acidosis has been reported in dogs under electrical anesthesia even though P_{CO_2} was reduced due to mild hyperventilation by a mechanical respirator (Hardy et al., 1961). Similarly, Herin (1968) found

a mild acidosis, a decreased arterial P_{CO_2} and decreased arterial bicarbonate concentration in electro-anesthetized dogs with unassisted respiration. Nevertheless, Cutherbertson et al. (1965), showed that central (carbon dioxide) and peripherally medicated central (cyanide) respiratory reflexes in dogs were uneffected by electrical anesthesia.

Temperature Regulation

An elevation in the deep body temperature of dogs during electro-anesthesia has been reported by numerous investigators (Anan'ev et al., 1957; Smith et al., 1961; Geddes, 1964; Herin, 1964; McIntyre and Voloshin, 1964; Cuthbertson et al., 1965). The hyperthermia has been postulated to result from a temporary derangement of the hypothalamic temperature regulating center and increased somatic muscle activity during electro-anesthesia (Smith et al., 1961; Herin, 1969). In order to reduce current-induced muscle spasms, Herin (1964) administered a muscle relaxant (gallamine) to intubated dogs. However, 8 of 10 animals still had elevated colonic temperatures of 2 to 3°C. at the conclusion of electro-anesthesia. Gowing (1964), observed that intubated dogs, premedicated with gallamine and an anesthetic dose of a barbiturate, did not become hyperthermic under electro-anesthesia if respiratory rate was artificially increased above the spontaneous rate. Cuthbertson et al. (1965) associated the hyperthermia of electro-anesthesia to room temperature. Since elevations in deep body temperature

to 43.5°C. were observed in intubated dogs in "hot" weather but not at "cool" ambient temperatures, or when animals were paralyzed with succinyl choline. Intubation alone, resulting in limited respiratory evaporative cooling, has been suggested as a contributing factor to the hyperthermia associated with electrical anesthesia (Smith, 1964).

Changes in deep body temperature have also been reported during electro-anesthesia in animals other than the dog. In sheep and cows pretreated with a phenothiazine tranquilizer (propiopromazine) Short (1964) found increases in deep body temperature of less than 1°F. in cows and 2 to 3°F. in sheep. Shiakh and associates (1968) examined some thermoregulatory changes during electro-anesthesia in sheep acutely exposed to a range of ambient temperatures. They reported significant increases in colonic temperatures of animals exposed to environmental temperatures of 18 to 23°C. and 27 to 29°C. The hyperthermia was attributed to inhibition of panting and increased somatic muscle tone. Their animals were also pretreated with propiopromazine prior to anesthesia.

There have been only a few studies on the effects of electro-anesthesia on peripheral skin temperatures. Cuthbertson and colleagues (1965), reported increases in paw skin temperatures of some animals, whereas others exhibited no change during electro-anesthesia. On the other hand, Wood et al. (1964) found reduced peripheral skin temperatures

in dogs given an anesthetic dose of pentobarbital and treated with anesthetizing currents.

Oxygen consumption ($\dot{V}_{O_2}$) measurements as an index to whole body metabolic rate or as an indirect assessment of the heat production attendant to increased somatic muscle tone during electro-anesthesia have received little attention. McIntyre and Voloshin (1964) reported increases in $\dot{V}_{O_2}$ of 20 to 100% in electro-anesthetized dogs over that for dogs under thiopentone anesthesia.

Estimates of respiratory evaporative water loss as a measure of heat loss during electro-anesthesia are totally lacking; the effects of electro-anesthesia on whole body temperature regulation are also unclear. No experiments have been conducted at controlled ambient temperatures with coincident evaluation of the biothermal (thermoregulatory) effects of concurrent drug therapy.

Cardiorespiratory and Biothermal Effects of Phenothiazine Tranquilizers

Chlorpromazine is generally considered the prototype phenothiazine tranquilizer and therefore has been more extensively studied than related substituted phenothiazines. It is tacitly assumed that the basic pharmacology of chlorpromazine is qualitatively similar to that of propiopromazine.

Cardiovasculature

Chlorpromazine has been reported to act directly on the heart and vasculature and indirectly through the central and autonomic nervous systems (Jarvik, 1967). In dogs, chlorpromazine-induced hypotension has been associated with a depression of centrally mediated pressor reflexes, as well as, a direct vasodilator action on peripheral vessels (Moyer et al., 1954). Courvoisier and co-workers (1953) found that chlorpromazine inhibited epinephrine-induced vasoconstriction in proportion to the dosage given. Chlorpromazine has also been reported to have a negative inotropic effect on cardiac muscle (Finkelstein et al., 1954; Courvoisier et al., 1953) and to decrease cardiac irritability (Melville, 1954).

Respiration

Studies on the respiratory responses to substituted phenothiazine administration are sparse and frequently conflicting. In dogs, anesthetized with pentobarbital Kissel and Yelnosky (1968) found no effect of chlorpromazine (0.2 to 4.0 mg/kg) on CO_2-stimulated respiration. Others have reported a therapeutic dose of chlorpromazine to have a respiratory stimulant action (Laborit, 1950; Reckless, 1954). Rabbits anesthetized with urethane exhibited a 20 to 40% increase in respiratory minute volume following 0.05-2 mg/kg chlorpromazine medication (Courvoisier et al., 1953). On the other hand, Lear (1959) found trifluprom azine (20 mg) given intravenously to humans produced a 25% decrease in

tidal volume. Similarly, Dobkins and co-workers (1956) reported that chlorpromazine (0.3-2 mg/kg) depressed both tidal volume and minute volume.

Higgins et al. (1964) noted that panting appeared to be less pronounced in propiopromazine treated dogs (2.2 mg/kg) during heat stress (37.8°C.) than in untreated animals. Similarly, Shiakh and colleagues (1968) observed a reduction in respiratory frequency following propiopromazine (1.1 mg/kg) treatment in sheep during exposure to various ambient temperatures. The initiation of panting in chlorpromazine treated (3 mg/kg) pigs was slower than non-treated animals during whole body heat exposure (Juszkiewicz and Jones, 1961).

Temperature Regulation

Many studies have examined the biothermal consequences of substituted phenothiazine treatment in animals. The hypothermic-producing capacity of these compounds is well documented (Courvoisier et al., 1953; Dundee et al., 1953; Ripstein et al., 1954; Higgins et al., 1964). During whole body cold exposure, peripheral vasodilation has been implicated as one avenue of increased heat loss in chlorpromazine treated animals (Decourt et al., 1953; Giaja, 1953; Kollias and Bullard, 1964).

Courvoisier et al. (1953) postulated that a depressed metabolic rate (e.g., $\dot{V}_{O_2}$) during tranquilizer medication contributed to whole body cooling. However, others suggested that metabolic depression was a secondary

effect of hypothermia (Giaja and Markovic-Giaja, 1954). The results of Kollias and Bullard (1964) in rats, suggested a direct relationship between $\dot{V}_{O_2}$ depression and the dosage of chlorpromazine given. At an ambient temperature of 23°C., the administration of a very high dose of chlorpromazine (25 mg/kg) decreased $\dot{V}_{O_2}$ coincident with an increased temperature and blood flow in the tail; shivering and piloerection were also diminished. With a lower dose of chlorpromazine (6.25 mg/kg), the rate of cooling was approximately half that of the higher level. In addition, shivering was not inhibited nor $\dot{V}_{O_2}$ depressed with low-dose treatment. Chlorpromazine has been reported to facilitate muscle relaxation by a selective depression of the gamma efferent system, which may diminish shivering thermogenesis during cold exposure (Henatsch and Ingrar, 1956).

At ambient temperatures above the thermoneutral zone, the biothermal responses to phenothiazine compounds are equivocal. Decourt et al (1953) reported a relative hypothermia in numerous different homeotherms given chlorpromazine at environmental temperatures greater than their internal body temperature. Although chlorpromazine has been demonstrated to increase survival rate in pigs exposed to ambient temperatures of 40°C. (Juszkiewicz and Jones, 1961) others have reported that chlorpromazine-treated animals become hyperthermic and survival rate is decreased (Binet and Decaud, 1954; Kollias and Bullard, 1964).

METHODS AND MATERIALS

Thermoregulatory and cardiorespiratory effects of propiopromazine and electrical anesthesia administered simultaneously and independently were evaluated in a series of experiments conducted during the interim of April 1, to November 22, 1969. For this study, care was taken to use a well-defined experimental subject, tested under controlled environmental exposure conditions.

Animals

Three non-pregnant, 2 year old purebred Dorset ewes (59 to 65 kg. body weight) were used in this study. Five months prior to the start of the experiments, the left common carotid artery of each animal was surgically exteriorized and sutured within a skin tube (Bone *et al.*, 1963). To insure uniform insulatory properties of wool, the sheep were closely shorn (less than 0.3 cm. of fleece remained) at weekly intervals. The animals were housed in individual pens (4 x 8 feet) in a building maintained at a temperature of 17°C. to 30°C. Each animal was fed a balanced ration of approximately 1500 kcal/day with water provided *ad libitum*. The animals were subjected to a training regime in a restraining device prior to the start of the trials (Figure 1).

Restraining Device

During the environmental chamber exposure the sheep were confined in a restraining device (22 x 42 x 56 inches) constructed of slotted angle steel (RS, Figure 1). The animals were supported by 2 plastic-coated canvas slings (SL, Figure 1) attached by ropes to a pulley system. During all trials, the animals were suspended so that their feet were just elevated off the floor of the restraining device in order to simulate the conditions of those trials where electro-anesthesia was used.

Environmental Chamber

All trials were conducted in an environmental chamber (3.5 x 5.5 x 7 feet) with an effective controlled temperature range of 5°C. to 50°C. $\pm$ 1°C. A thermistor temperature controller (Yellowsprings Instrument Co., Model 71) regulated the pumping of either a heated or cooled liquid through two convective heat exchangers which were located in front of fans inside the chamber (Figure 1).

Relative humidity of the chamber was controlled by a humidity controller (Hydrodynamic Inc., Model 15-3205) which regulated the cycling of chamber air through a humidifier (Kenmore, Model No. 758-72912) or dehumidifier (Coldspot, Model No. 106-637140). Major temperature differences within the chamber were minimized by an 8 inch oscillating fan.

Two sliding plexiglass windows (26 x 36 inches) on both sides of the chamber allowed access to the animals during the experiments.

Temperature Measurements

Rectal and 7 skin temperature measurements were obtained using copper-constantan thermocouples (36 s.w.g.) referenced to an ice bath and calibrated daily to the nearest 0.1°C. against a National Bureau of Standards thermometer. An automatic stepping relay allowed temperatures to be sequentially monitored on one channel of an adjustable range, adjustable zero strip chart recorder (Mosley, Model 7100B). Rectal temperature (T_{re}) was measured with a thermocouple fixed within a semi-rigid polethylene catheter and inserted 10 cm. into the lower colon. Skin temperatures were measured as follows:

a. Ear temperature (T_e) was monitored from a thermocouple taped to the dorsal side and approximate center of each ear. The mean skin temperature of the two ears was used in statistical analysis.

b. Face skin temperature (T_f) was measured from a thermocouple taped to the dorsal midline, approximately 5 cm. anterior to the eyes.

c. Forelimb skin temperatures were measured at 3 sites on the lateral surface of the right leg. The thermocouples were secured by a 1 $cm.^2$ piece of nylon

screening attached to an elastic band which encircled the extremity. Lower forelimb skin temperature (T_{ll}) was measured immediately distal to the pastern joint and mid-forelimb skin temperature (T_{ml}) was measured midway between the pastern and knee joints. Upper forelimb skin temperature (T_{ul}) was monitored from approximately 5 cm. proximal to the knee joint.

d. Trunk skin temperature (T_t) was measured with a thermocouple taped to the animal's left side, approximately 10 cm. lateral to the spinal column and 10 cm. posterior to the scapula.

Oxygen Consumption

Oxygen consumption ($\dot{V}_{O_2}$) was obtained by an open circuit technique. Air was drawn at a rate of 50 l/min through a cylindrical hood (15 inches diameter, 24 inches long), placed over the head of the animal (HD, Figure 1). $\dot{V}_{O_2}$ (STPD) was determined using an oxygen analyzer (Oxford Instrument Co.) which compared the partial pressure of oxygen in expired air with that of chamber air.

Respiratory Evaporative Water Loss

Respiratory evaporative water loss (E) was estimated with a relative humidity transducer (Hydrodynamics, Model No. 15-7012) by comparing the relative humidity of air entering and leaving the hood. A vacuum pump withdrew air

samples at the rate of 2.5 l/min. from the air flow system serving the oxygen analyzer; a reference air sample was drawn from within the chamber at an equal rate. Electrical potentials of 0 to 5.4 volts output (corresponding to 0 to 100% relative humidity) were metered on an adjustable range, adjustable zero strip chart recorder (Mosley, Model No. 7100B). Water evaporation was computed by:

$$Q_{HOH} = S\ (rh_o - rh_i)\ \dot{V} \qquad (1)$$

expressed as:

Q_{HOH} = water loss (gm./min)

S = grams of water in one liter of saturated air air (gm/l)

rh_o = relative humidity of chamber air (%)

rh_i = relative humidity of sample air (%)

$\dot{V}$ = air flow (l/min.).

Heat of vaporization was calculated from the formula described by Kleiber (1961):

$$\lambda = (Q_{HOH}) \cdot (K_1 - K_2 T_{re}) \qquad (2)$$

expressed as:

λ = heat of vaporization (kcal./min)

Q_{HOH} = water loss (gm./min)

K_1 = 0.5959 latent heat of vaporization at $0^{o}C.$ (kcal/gm.H_2O)

K_2 = 0.56×10^{-3} correction constant for temperature (kcal/gm.H_2O $^{o}C.$)

T_{re} = rectal temperature in $^{o}C.$

Respiratory Frequency

Respiratory rate was measured with a mercury-in-rubber strain gauge matched to a plethysmograph (Parks Electronics, Model No. 270) and monitored on one channel of a strip chart recorder (Mosley, Model No. 7100B). The strain gauge was attached to an elastic band which encircled the abdominal area offering maximal respiratory movement.

Blood Pressure and Heart Rate

Arterial blood pressure was measured by a catheter (.034 inch i.d.) inserted into the carotid loop (via 20 g. needle) and connected to a 4-way manifold valve, which served as a junction to a pressure transducer (Statham, Model No. P23A), an infusion pump (Harvard Apparatus Co., Model No. 1100) that flushed the catheter with heparinized saline (2 cc/hr.) and a blood sampling site. The blood pressure tracing was recorded with suitable amplification (Grass Preamplifier, Model No. 5PIK) on one channel of an adjustable range, adjustable zero strip chart recorder (Mosley, Model No. 7100B). Heart rate was obtained from the pressure pulse trace.

Arterial pH, Carbon Dioxide Tension and Bicarbonate Concentration

Arterial blood samples were collected anaerobically from the carotid loop catheter via the manifold stopcock. The samples were drawn into 5 cc. syringes (dead space filled with Na heparin; 1000 units/cc) immediately sealed with mercury-filled syringe caps and stored in ice water until the end of the experiment (4 hours). Arterial pH, P_{CO_2} and HCO_3^- concentration were determined by the Astrup technique using the nomogram (Radiometer, Code No. 984-200) developed by Siggaard-Andersen (1962). Anaerobic pH measurements were made using a micro-electrode chain attached to a Radiometer pH meter (Radiometer Co., Model 27). Equilibration of blood samples at known P_{CO_2}'s was accomplished using a Radiometer microtonometer (Radiometer Co. Model AMT-1). The micro-electrode chain and microtonometer were thermostatted to the animal's rectal temperature.

Alveolar Ventilation and Tidal Volume

Alveolar ventilation ($\dot{V}_A$) was computed (Rahn and Fenn, 1955) by assuming alveolar carbon dioxide tension (P_{ACO_2}) equal to arterial carbon dioxide tension (P_{aCO_2}), as:

$$\dot{V}_A = 0.760 \cdot \frac{T_{re}}{273} \cdot \frac{\dot{V}_{O_2}}{P_{aCO_2}} \qquad (3)$$

where:

$\dot{V}_A$ = alveolar ventilation (l BTPS/min)

T_{re} = rectal temperature (^{o}K)

$\dot{V}_{O_2}$ = oxygen consumption (ml STPD/min)

P_{aCO_2} = arterial carbon dioxide tension (mm.Hg.)

Tidal volume (V_T) was estimated by assuming a dead space (V_D) of 150 ml (Hales and Webster, 1966), according to the formula:

$$V_T = \frac{\dot{V}_A + V_D f}{f} \qquad (4)$$

where:

V_T = tidal volume (ml BTPS)

$\dot{V}_A$ = alveolar ventilation (ml BTPS/min)

V_D = dead space (ml BTPS)

f = respiratory frequency (breaths/min).

Electrical Anesthesia

A commercially available unit (Electronic Medical Instrument Co., Model No. 100-A) connected to an external oscillator (Hewlett Packard, Model No. 200AB) was used to produce electro-anesthesia. Bitemporal electrodes (18 gauge, 1.5 inch syringe needles) were inserted through the skin approximately 0.5 inches posterior to the base of the horns, immediately below the parietal crest, behind the coronoid process and deep enough ventrally to be in form contact with the skull. A local anesthetic (2% Lidocaine) was applied

to the electrode placement area prior to needle insertion. The electrodes were secured in place with adhesive tape. Electro-anesthesia was induced by presetting the oscillator at 700 cycles/second and gradually increasing the milliamperage from 0 to between 20 to 30 milliamperes. Generally, 2 to 5 minutes were required before satisfactory anesthetic depth was reached which was evaluated by the strength of a nociceptor reflex initiated by pinching or pricking the distal forelimb. The level of anesthesia was maintained by occasional adjustment of the current strength and frequency.

Experimental Procedures

A total of 60 trials were conducted at three ambient temperatures, 5°C., 25°C., and 35°C. Chamber relative humidity was controlled at 40% ± 5% for trials at 25°C. and 35°C. The limited cooling capacity of the chamber prevented maintenance of this humidity at the 5°C. experiments. Relative humidity for the 5°C. tests was maintained at 20% ± 5%. All tests lasted for 330 minutes or until T_{re} reached 41.8°C. Preliminary trials indicated that an approximate thermal "steady state" was achieved after a 90 minute chamber exposure at each ambient temperature.

At each exposure temperature, the animal was subjected to one of the following four experimental treatments:

a. Control or untreated trials: The animals were given a 1.5 cc intramuscular saline injection at 90 minutes.

b. Tranquilization trials (TQ): Animals were given propiopromazine HCl (1.0 mg/kg) intramuscularly at 90 minutes.

c. Tranquilization/Electro-Anesthesia trials (EA/TQ): Animals were given propiopromazine HCl (1.0 mg/kg) intramuscularly at 90 minutes followed by electro-anesthesia from 100 to 210 minutes.

d. Electro-Anesthesia trials (EA): Animals received only electro-anesthesia from 100 to 210 minutes.

Tests on any sheep were separated by a minimum of 72 hours; no animal was in the same treatment group or at the same exposure temperature for two consecutive tests. Replicate trials were conducted at each exposure temperature and treatment, with the exception of the electro-anesthesia group (without prior tranquilization). Only one of the three experimental animals could be successfully anesthetized without tranquilization which limited this group to a program of duplicate trials on only one sheep at each ambient temperature.

Measurements of rectal temperature, 7 skin temperatures, blood pressure, heart rate, respiratory frequency and $\dot{V}_{O_2}$ were recorded at 10 minute intervals for the first and last 60 minutes of each trial and at 5 minute intervals during the intervening time period. Respiratory evaporative water loss was measured at 10 minute intervals throughout

the trial. Arterial blood samples were taken at 90, 150, 210, 240 and 300 minutes during each trial.

Statistical Analyses

Means, standard error of the means and statistical comparisons by Analysis of Variance with Student's t test (Appendix B) were computed on an Olivetti-Underwood Programma 101 computer. The TQ and EA/TQ treatment groups were statistically compared with the control group and with each other at corresponding time intervals.

A within group statistical comparison was computed for the EA treatment group. Means for different time intervals or periods were compared to pretreatment (90 minute) means.

Means that showed a probability of error of less than 5% ($p < 0.05$) were considered statistically different for all temperature, $\dot{V}_A$, V_T, P_{aCO_2}, pH, HCO_3^- and arterial hematocrit values. All other parameters ($\dot{V}_{O_2}$, f, E, blood pressure and heart rate) whose measurement error was greater than the above, were considered statistically different if the probability of error was less than 1% ($p < 0.01$).

RESULTS

Rectal and skin temperatures in any experimental group were not different when animals entered the chamber indicating the animals were housed at sufficiently uniform ambient temperatures to achieve similar initial "thermal states" for all trials (Tables 1-9). Additionally, all measured parameters reported for the tranquilized (TQ) and electro-anesthetized-tranquilized (EA/TQ) treatment groups during the "pretreatment" period (i.e., 90 minutes or 60 to 90 minutes) were not different from corresponding measurements for the control animals under the same exposure conditions. The electro-anesthesia (EA) treatment group was not statistically compared with control, TQ or EA/TQ groups at any time interval during the test period due to the unrepresentative composition and limited sample number comprising this group.

Rectal Temperatures

Mean rectal temperatures (T_{re}) for all groups are presented in Tables 1-3, and are plotted as a function of time in Figures 2-4. At 5°C. ambient temperature, T_{re} of the tranquilized animals was reduced from that of the control group one hour after propiopromazine medication. Deep

body temperature in this group did not show any additional alteration for the duration of the experiment. In the EA/TQ group, T_{re} was less than the control animals at the end of anesthesia (210 minutes) and throughout the "recovery" period. Although mean T_{re} of the EA/TQ group appeared to be lower than that of the TQ sheep, the difference was not significant. The EA treatment group showed no difference in mean T_{re} at any time interval from the pretreatment (90 minute) T_{re}.

At the 25°C. exposure temperature, mean T_{re} of the TQ animals was lower than that of the control group at 210 minutes but not different from their own pretreatment T_{re} at this time interval. Mean T_{re} in the EA/TQ group was not different from corresponding control measurements during or after anesthesia.

The TQ and EA/TQ animals at the 35°C. ambient temperature both exhibited a significant elevation in mean T_{re} over that of the control animals at 150, 210 and 240 minutes. The T_{re} of the TQ group continued to rise for 170 minutes after drug medication. The EA/TQ animals showed an even more rapid rate of rise in T_{re} than did the TQ group. At the end of electro-anesthesia, T_{re} of the EA/TQ animals was greater than that of the TQ group. Moreover, T_{re} of the EA/TQ animals continued to rise after the end of anesthesia forcing the termination of one trial at 260 minutes and two additional trials at 290 minutes. Although the EA treatment

group also exhibited a mean increase in T_{re} of 1.5°C. during anesthesia, it was not statistically different from the mean "pretreatment" (90 minute) T_{re}.

Ear Skin Temperatures

Mean ear skin temperatures (T_e) for each exposure temperature and experimental group are presented in Table 4. Mean T_e for the TQ group during the 5°C. exposure ranged from 1.6 to 4.8°C. higher than control group T_e. However, T_e's of the TQ group were different from the control animals only at one hour post-tranquilization. The EA/TQ animals T_e's were 4.6 to 7.6°C. higher than untreated animals with statistical differences noted at both time intervals during the "recovery" period (240 and 300 minutes). The T_e's for the EA/TQ group were not significantly higher than the TQ group. Mean T_e for the EA treatment group (T_a = 5°C.) was not different from pretreatment measurements.

At the 25°C. exposure temperature, mean T_e's of the TQ and EA/TQ treatment groups were elevated over that of control animals at all post-treatment time intervals. The EA/TQ group had a higher mean T_e than TQ animals at time intervals of 150, 240 and 300 minutes. The EA treatment group (T_a = 25°C.) also had an increased T_e from pretreatment values during electro-anesthesia, but not during the "recovery" period.

No differences in T_e were found in TQ animals at 35°C. T_a from control animals whereas, the EA/TQ group demonstrated a higher T_e than control animals only at 300 minutes. Even though mean T_e of the EA group increased progressively during anesthesia and the "recovery" period, it was not significantly different from pretreatment measurements.

Face Skin Temperatures

Mean face skin temperatures (T_f) for each treatment group and exposure temperature are presented according to time intervals in Table 5. At the 5°C. T_a, mean T_f of the TQ group was higher than that of control animals during the first and second hour post-medication, but not at subsequent time periods. The EA/TQ animals exhibited an increased T_f from both control and the TQ group, during and following electro-anesthesia. The EA group (without prior propiopromazine treatment) had a higher T_f than pretreatment (90 minute) values only at the end of anesthesia (210 minutes).

At ambient exposure temperatures of 25°C. and 35°C., both the TQ and EA/TQ animals had T_f only slightly higher than control animals. The T_f of the EA group was not measured at the 25 and 35°C. ambient temperatures.

Forelimb Skin Temperatures

Mean forelimb skin temperatures (T_{ll}, T_{ml}, T_{ul}) for each environmental temperature and treatment group are in reference to test time intervals in Tables 6-8. At the T_a of 5°C., mean T_{ll}, T_{ml} and T_{ul} for both TQ and EA/TQ treatment groups were not different from corresponding control values. The EA treatment group exhibited a significantly lower T_{ul} than the pretreatment (90 minute) mean during and after anesthesia whereas T_{ll} and T_{ml} were not different from pretreatment values.

At the 25°C. ambient temperature the TQ animals had a higher T_{ll} than control animals at all time intervals following tranquilizer medication. Nevertheless, T_{ml} and T_{ul} were higher than control means only at 300 minutes. Similarly, the EA/TQ animals had higher T_{ll} than the control group during anesthesia and the first half hour of the "recovery" period. However, T_{ml} was elevated over control measurements only at the termination of anesthesia (210 minutes) ant T_{ul} only at 300 minutes. The EA treatment group ($T_a = 25°C.$) demonstrated an increasein the T_{ll} and T_{ml} over pretreatment means at the end of anesthesia. The T_{ul} was not different from pretreatment values during electroanesthesia or the "recovery" period.

The TQ animals at the 35°C. exposure temperature had a higher mean T_{ll} and T_{ml} than control animals at time intervals of 240 and 300 minutes, whereas T_{ul} was significantly

greater only at 300 minutes. At this same exposure temperature, the EA/TQ treatment group exhibited an elevated T_{ll} at the end of anesthesia (210 minutes) and a higher T_{ml} than control animals during anesthesia and the "recovery" period. Mean T_{ul} of the EA/TQ group was greater than control animals at 210, 240 and 300 minutes. However, Tables 6, 7 and 8 indicate that mean increases in forelimb skin temperatures for the EA/TQ group were not different from the TQ animals. Forelimb skin temperatures for the EA group ($T_a = 35^{\circ}C.$) were not different from pretreatment means.

Trunk Temperatures

Mean trunk skin temperatures (T_t), at appropriate time intervals, are reported for all treatment groups in Table 9. Mean T_t for TQ and EA/TQ treatment animals were not different from control animals or each other at any time interval or exposure temperature. Similarly, mean T_t of the EA group was not significantly different at any time interval or exposure temperature from pretreatment (90 minute) means.

Oxygen Consumption

Mean oxygen consumption values ($\dot{V}_{O_2}$) for designated time periods are reported for each exposure temperature and treatment group in Tables 10 and 11. At the $5^{\circ}C.$ ambient temperature, $\dot{V}_{O_2}$ for the TQ group was not different from

control animals during the first and second hour of medication but was higher during the third hour. The EA/TQ group also exhibited a lower $\dot{V}_{O_2}$ during anesthesia than both control and TQ groups. Similarly, the EA group showed a reduction in $\dot{V}_{O_2}$ during anesthesia from pretreatment measurements.

At the 25°C. environmental temperature, $\dot{V}_{O_2}$ for the TQ animals was higher than that of control animals. The EA/TQ animals also exhibited an elevated $\dot{V}_{O_2}$ over control animals during the second 55 minutes of anesthesia and during the "recovery" period. The EA group ($T_a = 25^{\circ}C.$) increased $\dot{V}_{O_2}$ during anesthesia and the "recovery" period from pretreatment values.

At 35°C. T_a, both the TQ and EA/TQ groups had a lower $\dot{V}_{O_2}$ than control animals during the first hour of their respective treatments but not during the second hour. Also, both groups exhibited a significantly greater $\dot{V}_{O_2}$ than control animals during the "recovery" period. Oxygen consumption for the EA group was higher during anesthesia and the "recovery period than pretreatment values.

Respiratory Frequency

Respiratory frequency (f) means for different time periods, treatment groups and ambient temperatures are presented in Tables 12 and 13. Although the TQ group exhibited no difference in f from control animals at the 5°C. T_a, the EA/TQ group had a lower f during anesthesia than either

control or TQ animals during the same time intervals. However, during the "recovery" period, f for EA/TQ and TQ groups were not different from one another or control animals. The EA treatment animal ($T_a = 5^{\circ}C.$) demonstrated no significant change in f during anesthesia or the "recovery" period.

At the $25^{\circ}C.$ exposure temperature, f for both TQ and EA/TQ animals was lower than control animals for the duration of the experiment. However, f for the EA group was not different from pretreatment values at any time interval.

Changes in f for control, TQ and EA/TQ animals at the $35^{\circ}C.$ exposure temperature are plotted as a function of time in Figure 5. The f of TQ group was lower than that of control animals during both the first and second hour post-propiopromazine medication. The EA/TQ group exhibited a lower f than both control and TQ animals during the electro-anesthesia period. However, neither the TQ nor EA/TQ groups had respiratory frequencies significantly different from control animals during the "recovery" period. Even though the EA animals had a higher f during anesthesia than the pretreatment period, respiratory rate more than doubled following the conclusion of anesthesia.

Respiratory Evaporative Water Loss

Mean respiratory evaporative water loss (E) values at each environmental temperature for each treatment group at designated time intervals are reported in Tables 14 and 15. At 5°C. both the TQ and EA/TQ groups had a greater E than control animals at all time periods subsequent to their respective treatments. On the other hand, the EA group exhibited no difference in E during anesthesia from pretreatment measurements.

Although the E of the TQ group was not different from control animals at the 25°C. environmental temperature, the EA/TQ group had a reduced E during the first 55 minutes of anesthesia and an increased E during the "recovery" period compared to the control group. In contrast, the EA animal exhibited an increased E during anesthesia but not during the "recovery" period.

At 35°C. T_a, E for the TQ animal was reduced during the first hour of medication but not at subsequent time periods. The EA/TQ group had a lower E during anesthesia than both control and TQ animals at corresponding time periods. However, during the "recovery" period E was not significantly different from either control or TQ animals. In contrast, E for the EA group was increased during anesthesia compared to the pretreatment period.

Arterial pH, P_{CO_2} and HCO_3^-

Mean arterial pH, P_{CO_2} and HCO_3^- determinations for each treatment group and exposure conditions are reported in Tables 16, 17 and 18, respectively. At the 5°C. exposure, the TQ treatment group exhibited no significant change in arterial pH, P_{CO_2} or HCO_3^- from control animals even though arterial P_{CO_2} of the TQ animals increased progressively following propiopromazine treatment. An increased arterial P_{CO_2} and decreased pH was found in EA/TQ animals during and after anesthesia compared to the control animals. However, HCO_3^- concentration in the EA/TQ group did not vary from the control group. The EA group exhibited a rise in pH only during the "recovery" period (300 minutes) whereas ($T_a = 5°C.$) P_{aCO_2} and HCO_3^- were not different from pretreatment values at any time interval.

During whole body exposure to the 25°C. ambient temperature, the TQ animals showed no difference in arterial pH, P_{CO_2} and HCO_3^- concentration from control animals. However, the EA/TQ group at this exposure temperature exhibited an increase arterial P_{CO_2} with no difference in arterial pH from control animals during anesthesia. By the end of the "recovery" period, P_{aCO_2} values were approaching control measurements. Arterial HCO_3^- concentration in the EA/TQ animals varied little from control measurements. During anesthesia and the "recovery" period the EA group ($T_a = 25°C.$) had no significant change in arterial P_{CO_2} or pH from

pretreatment values. Arterial bicarbonate concentration during and after anesthesia in the EA animal was not different from pretreatment determinations.

The TQ animal at the 35°C. exposure, showed no difference in arterial pH, P_{CO_2} or HCO_3^- concentration from control animals. A significant increase in arterial P_{CO_2} and decrease in arterial pH from control measurements was found in the EA/TQ treatment group ($T_a = 35°C.$) at the end of 55 minutes of anesthesia. However, arterial pH and P_{CO_2} was not different from control animals at the termination of anesthesia or during the "recovery" period. Also, arterial bicarbonate concentration in the EA/TQ animal was not different from control animals at any sampling interval. The EA animal at 35°C. T_a demonstrated no statistical difference in P_{aCO_2}, HCO_3^- concentration or pH during and after anesthesia from pretreatment determinations.

Alveolar Ventilation and Tidal Volume

Mean values for alveolar ventilation ($\dot{V}_A$) and tidal volume (V_T) are presented according to time intervals in Tables 19 and 20, respectively, for each treatment group and exposure temperature. At the 5°C. T_a, the TQ animals exhibited no mean differences in either $\dot{V}_A$ or V_T from control animals. However, during anesthesia the EA/TQ animals demonstrated a reduced $\dot{V}_A$ from both the control and TQ group. The EA animals exhibited no difference in $\dot{V}_A$ or V_T during anesthesia from pretreatment measurements.

The TQ animals at the 25°C. T_a, showed a significant increase in V_T (210 and 300 minutes), whereas $\dot{V}_A$ was not significantly different from control animals at any time interval. However, the EA/TQ and EA groups showed no change in either $\dot{V}_A$ or V_T from respective control measurements.

At the 35°C. T_a, $\dot{V}_A$ for the TQ and EA/TQ group was not different from control animals even though V_T was increased in the TQ animals at 150 minutes and during anesthesia in the EA/TQ group. The EA animals showed no difference in either V_T or $\dot{V}_A$ during anesthesia or the "recovery" period from pretreatment measurements.

Arterial Blood Pressure and Heart Rate

Mean arterial blood pressure and heart rate responses for each exposure condition and treatment group are presented in Tables 21-24. With the T_a at 5°C., the TQ group had a lower mean arterial blood pressure and higher heart rate than control animals following propiopromazine medication. The EA/TQ animal also had a lower blood pressure than control animals at all time periods, and lower blood pressure than the TQ group during the second 55 minutes of anesthesia. Heart rate in the EA/TQ animal was higher than control animals during the second 55 minutes of anesthesia and higher than both control and TQ groups during the "recovery" period. In contrast, the EA animal has a higher arterial blood pressure during anesthesia than pretreatment values at

all environmental temperatures (i.e., 5°C., 25°C., 35°C.). During the "recovery" period, blood pressure in the EA animal was higher than pretreatment measurements only at the 5°C. exposure. Heart rate was accelerated over pretreatment means in the EA animals only during anesthesia at the 35°C. environmental temperature and during the "recovery" period at the 5 and 35°C. ambient temperatures.

At the T_a of 25°C., both the TQ and EA/TQ groups had a lower blood pressure than control animals during the time periods of 115-160 minutes and 165-210 minutes. Heart rate in the TQ group was increased over control animals at all time periods following medication, whereas heart rate in the EA/TQ group was increased over control and TQ animals during the second 55 minutes of anesthesia and during the "recovery" period.

With the 35°C. environmental temperature, mean arterial blood pressure in the TQ group was lower than control animals only during the first hour of medication. However, heart rate for the above group was faster than control animals at all time periods following treatment. Although blood pressure of the EA/TQ group was not different from either control or TQ animals at any time period, heart rate of this group was more rapid than control animals during the second 55 minutes of anesthesia, and faster than both control and TQ groups during the "recovery" period.

Arterial Hematocrits

Mean values for arterial hematocrits are recorded in Table 25, for each treatment group and exposure temperature at the time intervals corresponding to arterial blood sampling. The TQ group had no change in hematocrit from control animals at any exposure temperature. The EA/TQ animals showed a significant increase in hematocrit over control and TQ animals at the 5°C. T_a. Arterial hematocrit of the EA/TQ group was also higher than control animals at the 35°C. exposure. The hematocrits of the EA animal were not statistically different from the pretreatment determinations at any of the exposure temperatures.

Shivering

At the cold exposure temperature (i.e., 5°C.) propiopromazine treatment inhibited observable shivering for approximately 25 minutes after medication, whereas, shivering in the EA/TQ animal was abated for an average of 50 minutes after treatment. The EA animal also showed no visible signs of shivering for an average of 25 minutes after initial current application.

Electro-Anesthesia Observations

The animals administered propiopromazine 10 minutes prior to electro-anesthesia (EA/TQ group) generally showed a 20 mm. Hg increase in blood pressure during initial current application. This was followed by a gradual fall and stabilization in blood pressure once the current was adjusted to "maintenance" levels. When the anesthesia was terminated, no appreciable change was detected in mean arterial blood pressure. The EA animal required from 5 to 10 more milliamperes of current to produce an anesthesia similar to that of the groups which received tranquilizer premedication. Muscle hypertonicity was not visibly observed to any appreciable extent during electro-anesthesia in animals receiving concurrent tranquilization.

TABLE 1

Effects of Tranquilizer (TQ) and Electro-anesthesia (EA) on Rectal Temperature (°C.) at 5°C.+

Treatment	No. of Trials	DURATION OF EXPOSURE (Mins.)					
		Pretreatment		Electro-anesthesia / Tranquilizer		Recovery	
		0	90	150	210	240	300
Control	6	39.3±0.1	39.4±0.2	39.4±0.3	39.4±0.2	39.4±0.2	39.4±0.3
TQ	6	39.2±0.3	39.5±0.3	38.9±0.2*	38.6±0.3*	38.6±0.4	38.6±0.3
EA/TQ	6	39.4±0.3	39.6±0.4	38.9±0.3	37.8±0.4*	37.6±0.4*	37.8±0.3*
EA	2	39.3±0.3	39.7±0.3	39.5±0.3	39.7±0.2	39.9±0.3	40.2±0.4

+ Mean values ± SEM.
* Significantly different from control ($p < 0.05$).

TABLE 2

Effects of Tranquilizer (TQ) and Electro-anesthesia (EA) on Rectal Temperature (°C.)+ at 25°C.

Treatment	No. of Trials	DURATION OF EXPOSURE (Mins.)					
		Pretreatment		Electro-anesthesia / Tranquilizer		Recovery	
		0	90	150	210	240	300
Control	6	39.4±0.2	39.8±0.3	40.0±0.4	40.0±0.2	40.0±0.2	39.9±0.4
TQ	6	39.0±0.4	39.4±0.3	39.2±0.3	39.1±0.4*	39.2±0.3	39.4±0.3
EA/TQ	6	39.4±0.2	39.9±0.2	39.9±0.3	39.5±0.3	39.6±0.4	39.8±0.2
EA	2	38.8±0.5	39.2±0.4	39.5±0.5	39.7±0.2	39.5±0.3	39.4±0.5

\+ Mean values ± SEM.
* Significantly different from control ($p < 0.05$)

TABLE 3

Effects of Tranquilizer (TQ) and Electro-anesthesia (EA) on Rectal Temperature+ (°C.) at 35°C.

Treatment	No. of Trials	DURATION OF EXPOSURE (Mins.) Pretreatment 0	Pretreatment 90	Electro-anesthesia Tranquilizer 150	Electro-anesthesia Tranquilizer 210	Recovery 240	Recovery 300
Control	6	39.4±0.3	40.0±0.4	40.1±0.1	39.9±0.2	39.9±0.2	39.9±0.4
TQ	6	39.5±0.3	40.1±0.3	40.6±0.2*	40.8±0.1*	40.9±0.3*	40.7±0.2
EA/TQ	5	39.1±0.2	39.7±0.2	40.7±0.2*	41.4±0.1**	41.6±0.1*	41.3±0.3*a
EA	2	39.1±0.1	39.7±0.3	40.4±0.2	41.2±0.7	41.2±0.7	---

\+ Mean values ± SEM.
* Significantly different from control ($p < 0.05$)
a n=2
** Significantly different from control and TQ animals ($p < 0.05$).

TABLE 4

TABLE 4

Effects of Tranquilizer (TQ) and Electro-anesthesia (EA) on Ear Skin Temperature (T_e)+ in °C.

Treatment	No. of Trials	Ambient Temp.(°C.)	DURATION OF EXPOSURE (Mins.) Pretreatment 0	Pretreatment 90	Electro-anesthesia / Tranquilizer 150	Electro-anesthesia / Tranquilizer 210	Recovery 240	Recovery 300
Control	6	5	14.8±0.5	8.1±0.5	8.1±0.5	7.9±0.6	8.0±0.5	7.5±0.5
TQ	6	5	15.3±0.4	7.8±0.2	9.7±0.5*	12.7±2.3	12.3±2.3	11.8±2.4
EA/TQ	6	5	15.1±0.3	8.2±0.4	12.7±3.0	13.7±3.1	15.2±3.1*	14.7±2.9*
EA	2	5	15.3±0.2	9.1±0.4	10.3±0.8	11.5±1.7	14.9±6.8	10.0±2.0
Control	6	25	24.4±0.9	27.5±1.4	27.7±1.3	27.1±1.0	27.1±0.9	28.4±1.5
TQ	6	25	24.4±1.0	27.4±1.1	32.3±0.3*	32.6±0.2*	32.4±0.3*	32.0±0.3*
EA/TQ	6	25	26.1±1.9	30.4±1.5	33.9±0.4**	33.8±0.6*	34.2±0.4**	33.9±0.6**
EA	2	25	22.6±1.7	28.8±3.4	31.7±1.0	29.7±2.7	28.7±3.3	27.2±2.8
Control	6	35	32.2±1.5	37.4±0.3	37.5±0.5	37.5±0.4	37.4±0.3	37.3±0.3
TQ	6	35	31.6±1.5	37.4±0.1	38.1±0.2	38.1±0.2	38.2±0.2	38.1±0.2
EA/TQ	5	35	30.0±1.5	36.6±0.3	37.8±0.4	37.9±0.5	38.0±0.4	38.6±0.3*a
EA	2	35	32.5±1.7	37.7±1.2	37.8±0.2	38.1±1.2	38.4±0.7	---

\+ Mean values ± SEM.
a n=2.
* Significantly different from control ($p < 0.05$).
** Significantly different from control and TQ animals ($p < 0.05$).

TABLE 5

Effects of Tranquilizer (TQ) and Electro-anesthesia (EA) on Face Skin Temperature (T_f)[+] in °C.

Treatment	No. of Trials	Ambient Temp.(°C.)	DURATION OF EXPOSURE (Mins.)					
			Pretreatment		Electro-anesthesia / Tranquilizer		Recovery	
			0	90	150	210	240	300
Control	6	5	20.9±0.5	16.4±1.0	16.5±0.9	16.2±0.9	16.5±1.0	16.3±1.1
TQ	6	5	20.9±0.5	16.3±0.8	20.8±1.5*	20.0±1.2*	19.0±1.4	19.2±1.3
EA/TQ	6	5	21.0±0.5	16.2±0.3	23.6±0.8*	21.6±0.5*	20.0±1.1*	19.8±0.9*
EA	2	5	19.3±1.0	16.2±0.4	19.7±1.8	19.1±0.3 p	17.4±1.1	17.6±1.1
Control	6	25	28.5±0.9	31.5±1.1	31.4±0.7	31.2±1.0	30.9±1.0	31.7±0.9
TQ	6	25	28.5±0.9	30.7±0.7	32.5±0.4	32.2±0.4	31.3±0.5	32.0±0.5
EA/TQ	6	25	28.9±1.4	30.7±0.8	32.6±0.1	32.4±0.3	32.0±0.3	30.9±0.7
Control	6	35	31.2±1.1	37.3±0.3	37.3±0.3	37.2±0.2	37.1±0.2	37.1±0.2
TQ	6	35	32.2±1.9	37.3±0.3	37.5±0.4	37.8±0.3	37.5±0.5	37.9±0.5
EA/TQ	5	35	31.5±1.8	36.7±0.2	37.6±0.2	37.5±0.3	37.6±0.3	37.2±0.7 a

\+ Mean values ± SEM.
a n=2.
* Significantly different from control ($p<0.05$).
p Significantly different from pretreatment value (90 min) ($p<0.05$) EA group only.

TABLE 6

Effects of Tranquilizer (TQ) and Electro-anesthesia (EA) on Lower Fore-limb Temperature (T_{ll})+ in °C.

Treatment	No. of Trials	Ambient Temp.(°C.)	DURATION OF EXPOSURE (Mins.) Pretreatment 0	Pretreatment 90	Electro-anesthesia / Tranquilizer 150	210	Recovery 240	300
Control	6	5	19.8±0.7	7.9±0.3	6.1±0.1	5.5±0.1	5.5±0.1	5.7±0.1
TQ	6	5	20.0±0.7	8.0±0.3	6.3±0.5	6.2±0.7	5.9±0.6	7.0±1.1
EA/TQ	6	5	20.1±0.6	7.9±0.2	5.8±0.1	5.4±0.1	5.8±0.1	6.6±0.7
EA	2	5	16.4±4.1	7.3±1.3	5.9±0.9	5.5±0.5	5.3±0.3	5.3±0.3
Control	6	25	28.8±0.8	26.4±0.3	26.7±0.7	27.3±1.3	27.5±1.2	27.6±1.1
TQ	6	25	26.2±1.8	25.9±1.1	31.1±1.1*	31.8±1.2*	33.1±1.0*	31.9±0.9*
EA/TQ	6	25	27.6±1.9	25.7±0.5	31.5±1.8*	32.1±1.3*	32.2±1.1*	31.2±1.4
EA	2	25	24.9±4.5	23.0±0.9	26.0±0.8	27.4±0.6p	26.5±1.1	25.4±0.9
Control	6	35	30.1±1.1	36.7±0.4	37.1±0.4	37.2±0.3	37.2±0.4	37.2±0.3
TQ	6	35	27.8±1.9	36.4±0.4	37.9±0.2	38.0±0.1	38.2±0.2*	38.2±0.1*
EA/TQ	5	35	28.6±1.5	35.6±0.5	38.1±0.3	38.3±0.3*	38.2±0.3	38.0±0.1a
EA	2	35	26.4±0.9	36.9±0.3	36.9±0.8	37.5±0.5	37.4±0.8	---

\+ Mean values ± SEM.
a n=2
* Significantly different from control ($p < 0.05$).
p Significantly different from pretreatment (90 min) values ($p < 0.05$) EA group only.

TABLE 7

Effects of Tranquilizer (TQ) and Electro-anesthesia (EA) on Mid-Forelimb Temperature (T_{ml})+ in °C.

Treatment	No. of Trials	Ambient Temp.(°C.)	DURATION OF EXPOSURE (Mins.) Pretreatment 0	Pretreatment 90	Electro-anesthesia / Tranquilizer 150	Electro-anesthesia / Tranquilizer 210	Recovery 240	Recovery 300
Control	6	5	19.6±0.7	9.2±0.3	7.9±0.2	7.1±0.3	7.1±0.3	6.8±0.4
TQ	6	5	19.6±0.4	8.5±0.3	7.1±0.4	7.1±0.4	7.0±0.3	8.9±1.7
EA/TQ	6	5	19.9±0.5	9.0±0.4	7.2±0.3	7.1±0.5	7.0±0.4	8.0±0.5
EA	2	5	17.5±2.9	7.9±1.1	6.9±1.2	6.5±0.6	6.2±0.5	6.7±0.2
Control	6	25	28.6±0.6	28.7±0.8	28.9±1.0	29.4±1.1	29.4±1.0	29.3±0.9
TQ	6	25	27.0±1.3	27.5±0.9	31.2±1.2	31.6±1.0	31.4±1.0	31.8±0.6*
EA/TQ	6	25	27.6±1.6	26.9±0.5	31.0±1.4	32.5±1.0*	31.8±1.0	30.4±1.3
EA	2	25	21.6±1.4	24.9±0.1	29.9±3.3	29.7±0.6P	28.4±0.5P	27.3±0.2P
Control	6	35	30.8±1.0	36.3±0.3	36.7±0.2	36.8±0.4	36.8±0.2	36.6±0.1
TQ	6	35	29.4±1.2	36.1±0.4	37.7±0.3	37.8±0.1	37.9±0.1*	37.9±0.2*
EA/TQ	5	35	29.2±1.0	35.8±0.5	37.9±0.1*	38.2±0.2*	37.9±0.3*	37.6±0.3*a
EA	2	35	27.5±0.7	37.1±0.2	36.8±0.3	37.4±0.2	37.3±0.4	—

\+ Mean values ± SEM.
a n=2.

* Significantly different from control ($p < 0.05$).
P Significantly different from pretreatment (90 min) values ($p < 0.05$) EA group only.

TABLE 8

Effects of Tranquilizer (TQ) and Electro-anesthesia (EA) on Upper Fore-limb Temperature (T_{ul})+ in °C.

Treatment	No. of Trials	Ambient Temp. (°C.)	DURATION OF EXPOSURE (Mins.) Pretreatment 0	Pretreatment 90	Electro-anesthesia Tranquilizer 150	Electro-anesthesia Tranquilizer 210	Recovery 240	Recovery 300
Control	6	5	27.0±0.6	19.3±0.8	17.9±0.8	17.2±0.9	16.9±1.0	16.4±1.1
TQ	6	5	26.7±0.5	19.4±0.8	18.9±1.8	19.1±2.0	19.1±1.5	18.0±1.4
EA/TQ	6	5	26.2±0.9	19.4±0.8	18.3±1.6	17.4±1.3	17.4±0.8	18.9±0.9
EA	2	5	22.9±1.2	17.2±0.4	14.8±0.5^{p}	13.3±0.1^{p}	13.1±0.6^{p}	---
Control	6	25	32.8±1.1	33.2±0.4	33.3±0.6	33.5±0.7	33.3±0.7	29.3±0.9
TQ	6	25	32.0±0.7	31.7±0.8	33.3±0.9	33.5±0.8	33.3±0.9	33.7±0.8*
EA/TQ	6	25	33.3±1.0	33.7±0.9	34.9±0.9	34.8±0.8	34.7±0.9	34.4±0.9*
EA	2	25	30.3±1.8	30.8±0.2	32.9±2.4	32.2±1.0	31.6±0.9	31.0±0.6
Control	6	35	35.6±0.5	38.1±0.2	38.2±0.2	38.1±0.1	38.2±0.1	38.0±0.1
TQ	6	35	34.9±0.7	37.8±0.2	38.5±0.2	38.5±0.2	38.6±0.1	38.5±0.1*
EA/TQ	5	35	34.6±0.7	37.7±0.4	38.7±0.5	39.1±0.4*	39.3±0.4*	39.1±0.1^{a}*
EA	2	35	32.4±0.3	37.6±0.4	37.1±0.5	37.7±0.6	37.8±0.6	---

\+ Mean values ± SEM.
a n=2

* Significantly different from control ($p<0.05$).
p Significantly different from pretreatment (90 min) values ($p<0.05$) EA group only.

TABLE 9

Effects of Tranquilizer (TQ) and Electro-anesthesia (EA) on Trunk Temperature (T_t)+ in °C.

Treatment	No. of Trials	Ambient Temp. (°C.)	DURATION OF EXPOSURE (Mins.) Pretreatment 0	90	Electro-anesthesia / Tranquilizer 150	210	Recovery 240	300
Control	6	5	21.3±1.1	18.4±1.0	18.4±1.0	18.4±1.0	18.4±0.9	18.2±1.0
TQ	6	5	21.9±1.1	17.8±1.1	19.1±1.1	18.7±1.0	18.3±0.9	18.0±0.9
EA/TQ	6	5	21.6±0.5	17.6±0.6	16.8±0.4	16.6±0.4	16.6±0.7	16.2±0.5
EA	2	5	22.6±1.0	21.0±0.9	20.6±1.3	20.2±2.0	22.1±0.6	22.4±0.7
Control	6	25	28.8±0.4	29.3±0.3	29.4±0.3	29.5±0.5	29.4±0.5	29.2±0.4
TQ	6	25	29.8±0.4	30.3±0.4	30.4±0.5	30.2±0.4	30.1±0.6	29.8±0.5
EA/TQ	6	25	29.8±0.8	30.7±0.7	30.6±0.8	30.3±0.7	30.3±0.6	30.5±0.7
EA	2	25	28.9±1.5	30.2±3.1	30.5±2.0	30.4±1.9	30.6±1.6	30.6±1.2
Control	6	35	35.3±0.5	36.7±0.3	36.6±0.3	37.0±0.4	37.0±0.4	36.5±0.3
TQ	6	35	34.6±0.8	36.7±0.2	37.3±0.4	36.9±0.3	36.9±0.4	36.9±0.4
EA/TQ	5	35	34.9±0.4	36.4±0.4	36.8±0.3	37.1±0.3	37.2±0.2	37.0±0.4[a]
EA	2	35	34.4±0.6	35.9±0.5	36.0±0.4	36.2±0.1	36.7±0.5	---

\+ Mean values ± SEM.
a n=2

TABLE 10

Effects of Tranquilizer (TQ) and Electro-anesthesia (EA) on Oxygen Consumption+

Treatment	No. of Trials	Ambient Temp.(°C.)	EXPOSURE INTERVALS (Mins.) Pretreatment 60-90 (n=42)	Electro-anesthesia Tranquilizer 115-160 (n=60)	160-210 (n=60)	Recovery 215-270 (n=72)
Control	6	5	281.2±13.8	301.8±14.0	312.8±16.8	296.3±11.7
TQ	6	5	272.1± 9.6	272.1± 8.3	289.7± 9.8	314.4± 8.4
EA/TQ	6	5	284.2± 7.4	204.9± 6.1**	214.1± 8.9**	265.3± 9.8
Control	6	25	207.9± 7.8	177.1± 6.8	178.9± 5.6	186.8± 5.4
TQ	6	25	188.2± 5.7	236.4± 8.5*	243.6± 8.7*	252.5±10.8*
EA/TQ	6	25	176.7± 6.2	181.8± 4.9	213.0± 5.7*	238.3± 3.9*
Control	6	35	254.0±10.5	276.9± 7.5	268.7± 7.8	249.1± 5.2
TQ	6	35	270.7± 6.9	243.7± 6.9*	274.0± 5.5	305.2± 5.0*
EA/TQ	5	35	256.0±11.2	234.7± 8.3*	284.0± 7.9	317.8±10.8*

\+ Mean values (ml/min,STPD) ± SEM.
* Significantly different from control ($p < 0.01$).
** Significantly different from control and TQ animals ($p < 0.01$).

TABLE 11

Effects of Electro-anesthesia (EA) on Oxygen Consumption+

Treatment	No. of Trials	Ambient Temp.(°C.)	EXPOSURE INTERVALS (Mins.) Pretreatment 60-90 (n=10)	Electro-anesthesia 115-160 (n=20)	Electro-anesthesia 160-210 (n=20)	Recovery 215-270 (n=24)
EA	2	5	500.0±36.6	292.0±15.5*	352.8± 9.5*	451.4±25.0
EA	2	25	230.4± 8.4	356.5±12.5*	306.5± 7.8*	257.1± 6.6*
EA	2	35	305.4±16.4	421.3±11.5*	432.5±12.3*	398.8±10.2*

\+ Mean values (ml/min, STPD) ± SEM.
* Significantly different from pretreatment values (60-90 min) ($p < 0.01$).

TABLE 12

Effects of Tranquilizer (TQ) and Electro-anesthesia (EA) on Respiratory Frequency+

Treatment	No. of Trials	Ambient Temp.(°C.)	EXPOSURE INTERVALS (Mins.) Pretreatment 60-90 (n=42)	Electro-anesthesia Tranquilizer 115-160 (n=60)	165-210 (n=60)	Recovery 215-270 (n=72)
Control	6	5	17.4± 0.5	18.5±0.4	18.4±0.6	18.1±0.5
TQ	6	5	20.9± 1.1	19.3±0.6	18.6±0.4	19.2±0.5
EA/TQ	6	5	18.2± 0.6	15.4±0.3**	15.7±0.1**	18.0±0.3
Control	6	25	23.7± 1.3	27.5±2.0	24.9±1.5	25.2±1.1
TQ	6	25	20.0± 0.8	20.0±0.6*	19.4±0.5*	19.2±0.4*
EA/TQ	6	25	25.4± 1.4	18.2±0.4*	18.4±0.5*	19.2±0.6*
Control	6	35	107.5±10.4	156.9±5.9	162.0±6.0	160.9±6.4
TQ	6	35	92.8±10.2	72.9±4.6*	114.4±6.2*	156.8±6.6
EA/TQ	5	35	82.2± 8.7	30.7±1.9**	55.2±3.0**	139.2±6.6

\+ Mean values (breaths/min) ± SEM.
* Significantly different from control ($p < 0.01$).
** Significantly different from control and TQ animals ($p < 0.01$).

TABLE 13

Effects of Electro-anesthesia (EA) on Respiratory Frequency+

Treatment	No. of Trials	Ambient Temp.(°C.)	EXPOSURE INTERVALS (Mins.) Pretreatment 60-90 (n=10)	Electro-anesthesia 115-160 (n=20)	Electro-anesthesia 160-210 (n=20)	Recovery 215-270 (n=24)
EA	2	5	18.0±0.8	16.4± 0.5	17.3± 0.3	18.5± 0.4
EA	2	25	19.7±0.7	18.7± 0.9	18.3± 0.8	18.8± 0.8
EA	2	35	33.8±6.6	104.4±10.1*	142.1±10.8*	288.9±15.4*

\+ Mean values (breaths/min) ± SEM.
* Significantly different from pretreatment (60-90 min) value ($p < 0.01$).

TABLE 14

Effects of Tranquilizer (TQ) and Electro-anesthesia (EA) on Respiratory Evaporative Water Loss (E)+

Treatments	No. of Trials	Ambient Temp. (°C.)	EXPOSURE INTERVALS (Mins.) Pretreatment	Electro-anesthesia / Tranquilizer		Recovery
			60-90 (n=42	115-160 (n=60)	165-210 (n=60)	215-270 (n=72)
Control	6	5	50.7± 5.7	51.9±2.8	47.5± 2.9	48.7± 2.0
TQ	6	5	58.9± 4.1	73.3±3.0*	70.5± 2.1*	68.8± 2.1*
EA/TQ	6	5	67.2± 4.6	74.6±1.7*	73.4± 1.3*	67.0± 2.3*
Control	6	25	50.7± 3.5	47.5±4.4	37.7± 2.9	32.8± 2.3
TQ	6	25	39.6± 4.2	41.0±3.7	37.3± 2.5	34.0± 2.2
EA/TQ	6	25	40.2± 3.4	29.5±2.8*	37.1± 3.7	43.5± 2.4*
Control	6	35	152.7±13.4	194.2±7.9	192.6± 7.6	182.1± 7.8
TQ	6	35	149.0±15.4	124.2±8.7*	162.6±13.6	182.8±10.2
EA/TQ	5	35	131.2±12.8	61.4±3.8**	99.8± 6.1**	157.7± 7.7

\+ Mean values (cal/kg.bd.wt./hour) + SEM.
* Significantly different from control ($p < 0.01$).
** Significantly different from control and TQ animals ($p < 0.01$)

TABLE 15

Effects of Electro-anesthesia (EA) on Respiratory Evaporative Water Loss (E)+

Treatment	No. of Trials	Ambient Temp.(°C.)	EXPOSURE INTERVALS (Mins.) Pretreatment 60-90 (n=10)	Electro-anesthesia 115-160 (n=20)	Electro-anesthesia 165-210 (n=20)	Recovery 215-270 (n=24)
EA	2	5	75.8± 2.3	66.8±1.4	64.8± 1.8	63.7±2.1
EA	2	25	55.1± 6.0	110.9±9.2*	113.0±11.7*	73.5±5.3
EA	2	35	138.7±26.2	291.6±7.9*	300.0± 6.8*	---

\+ Mean values (cal/kg.bd. wt./hour)± SEM.
* Significantly different from pretreatment (60-90 min) value ($p < 0.01$).

TABLE 16

Effects of Tranquilizer (TQ) and Electro-anesthesia (EA) on Arterial pH[+].

Treatment	No. of Trials	Ambient Temp.(°C.)	Pretreatment	Electro-anesthesia / Tranquilizer		Recovery	
			90	150	210	240	300
Control	6	5	7.49±0.02	7.49±0.17	7.49±0.02	7.48±0.01	7.50±0.01
TQ	6	5	7.46±0.02	7.46±0.02	7.47±0.02	7.47±0.03	7.46±0.05
EA/TQ	6	5	7.47±0.01	7.43±0.03	7.42±0.02*	7.43±0.02*	7.46±0.02*
EA	2	5	7.41±0.01	7.37±0.02	7.39±0.01	7.44±0.01	7.45±0.01P
Control	6	25	7.50±0.04	7.51±0.04	7.52±0.04	7.50±0.03	7.52±0.02
TQ	6	25	7.49±0.04	7.49±0.05	7.48±0.05	7.47±0.05	7.47±0.04
EA/TQ	6	25	7.51±0.03	7.43±0.01	7.44±0.05	7.45±0.02	7.49±0.17
EA	2	25	7.47±0.09	7.46±0.09	7.48±0.04	7.49±0.10	7.50±0.07
Control	6	35	7.51±0.04	7.52±0.01	7.52±0.02	7.51±0.02	7.52±0.02
TQ	6	35	7.50±0.02	7.48±0.02	7.49±0.04	7.51±0.02	7.51±0.02
EA/TQ	5	35	7.50±0.02	7.47±0.02*	7.48±0.02	7.56±0.04	7.53±0.02[a]
EA	2	35	7.47±0.01	7.48±0.04	7.51±0.04	7.64±0.14	---

\+ Mean values ± SEM.
a n=2.
* Significantly different from control ($p < 0.05$).
p Significantly different from pretreatment (90 min) values ($p < 0.05$) EA group only.

TABLE 17

Effects of Tranquilizer (TQ) and Electro-anesthesia (EA) on Arterial P_{CO_2} + in mm Hg.

Treatment	No. of Trials	Ambient Temp.(°C.)	Pretreatment	Electro-anesthesia / Tranquilizer		Recovery	
			90	150	210	240	300
Control	6	5	39.7±1.8	40.2±1.1	40.2±1.6	40.3±0.7	39.8±1.0
TQ	6	5	40.4±2.0	41.4±2.3	43.5±2.5	44.9±2.5	46.8±5.4
EA/TQ	6	5	42.6±1.1	49.0±3.8*	49.7±2.6*	47.0±2.3*	44.9±1.9*
EA	2	5	44.7±0.5	49.0±3.0	47.9±1.6	42.2±0.8	40.6±0.9
Control	6	25	38.8±0.8	37.2±0.9	37.3±1.4	38.9±1.5	37.2±1.5
TQ	6	25	37.7±0.7	38.8±1.5	42.9±2.3	44.4±2.0	43.4±3.0
EA/TQ	6	25	38.8±1.7	48.1±1.9*	47.5±2.4*	43.2±2.5	42.8±2.3
EA	2	25	42.2±0.2	45.1±0.4	43.9±0.5	41.8±0.5	40.9±0.7
Control	6	35	36.3±1.7	36.2±1.2	35.8±1.1	35.6±1.5	36.0±1.1
TQ	6	35	37.1±1.5	38.7±2.9	37.1±2.9	34.6±0.8	33.7±1.6
EA/TQ	5	35	38.4±1.4	43.1±1.5*	39.8±1.8	32.8±3.0	33.6±5.1[a]
EA	2	35	36.9±0.5	35.5±2.0	29.8±5.3	21.6±9.9	—

\+ Mean values ± SEM.
a n=2.
* Significantly different from control ($p < 0.05$).

TABLE 18

Effects of Tranquilizer (TQ) and Electro-anesthesia (EA) on Arterial Bicarbonate + in meg/l.

Treatment	No. of Trials	Ambient Temp.(°C.)	Pretreatment	Electro-anesthesia Tranquilizer		Recovery	
			90	150	210	240	300
Control	6	5	29.4±1.5	29.7±1.1	29.6±1.1	29.3±1.1	29.9±0.5
TQ	6	5	27.8±1.1	28.6±0.8	30.1±0.9	30.9±1.0	31.0±1.0
EA/TQ	6	5	29.6±0.6	30.4±1.1	30.8±1.3	29.6±0.9	30.7±1.1
EA	2	5	27.0±0.5	26.9±0.3	28.1±0.7	27.8±0.3	26.8±0.2
Control	6	25	29.3±1.2	29.3±1.4	29.7±1.3	29.5±1.1	29.2±1.3
TQ	6	25	28.1±1.5	29.4±1.8	30.9±1.1	31.3±1.3	30.4±2.0
EA/TQ	6	25	29.8±1.0	30.9±1.2	31.0±1.4	29.4±1.4	31.4±1.5
EA	2	25	29.9±2.7	31.2±2.9	31.4±3.1	31.7±2.9	31.4±4.4
Control	6	35	28.2±1.2	28.7±1.3	28.6±1.4	28.6±1.3	28.3±1.3
TQ	6	35	28.0±1.1	27.9±1.5	27.1±1.7	26.9±1.5	26.3±1.4
EA/TQ	5	35	29.7±1.4	30.1±1.3	28.4±1.2	28.5±1.7	27.3±4.4[a]
EA	2	35	26.0±0.4	25.5±0.7	22.9±1.9	20.5±3.5	---

\+ Mean values (meg/l) ± SEM.
a n=2.

TABLE 19

Effects of Tranquilizer (TQ) and Electro-anesthesia (EA) on Alveolar Ventilation ($\dot{V}_A$)+

Treatment	No. of Trials	Ambient Temp.(°C.)	DURATION OF EXPOSURE (Mins.) Pretreatment	Electro-anesthesia / Tranquilizer			Recovery
			90	150	210	240	300
Control	6	5	6.4±0.7	6.5±0.7	6.7±1.0	6.3±0.8	7.3±1.3
TQ	6	5	5.9±0.7	6.4±0.8	6.2±0.8	6.2±0.7	6.0±0.9
EA/TQ	6	5	5.8±0.5	4.1±0.6**	3.9±0.7**	4.7±0.6	5.2±0.7
EA	2	5	10.2±2.8	6.5±0.6	6.3±0.3	8.6±0.6	8.4±0.4
Control	6	25	4.9±0.4	4.3±0.3	4.2±0.5	4.3±0.6	4.9±0.4
TQ	6	25	4.5±0.5	5.3±0.8	5.3±0.7	5.1±0.9	6.0±0.6
EA/TQ	6	25	4.2±0.4	3.5±0.5	4.4±0.5	4.8±0.2	4.8±0.6
EA	2	25	4.7±1.0	6.5±0.2	5.7±0.7	4.8±0.2	5.6±0.7
Control	6	35	6.3±0.9	6.5±0.9	5.9±0.5	6.3±0.5	6.0±0.5
TQ	6	35	6.5±0.6	5.4±0.9	7.0±1.0	7.5±0.5	8.3±0.7*
EA/TQ	5	35	5.8±0.9	5.1±0.7	7.0±0.6	7.8±1.0	9.9±2.6[a]
EA	2	35	6.6±1.7	10.4±0.2	12.9±4.1	21.6±11.9	---

\+ Mean values (liters/minute,BTPS) ± SEM.
a n=3
* Significantly different from control ($p < 0.05$).
** Significantly different from control & TQ animals ($p < 0.05$).

TABLE 20

Effects of Tranquilizer (TQ) and Electro-anesthesia (EA) on Tidal Volume (V_T)+

Treatment	No. of Trials	Ambient Temp.(°C.)	DURATION OF EXPOSURE (Mins.) Pretreatment	Electro-anesthesia / Tranquilizer		Recovery	
			90	150	210	240	300
Control	6	5	527± 34	489±29	540±61	501±44	597±69
TQ	6	5	458± 52	520±63	490±54	470±44	448±48
EA/TQ	6	5	483± 39	418±45	396±46	419±38	432±39
EA	2	5	454±261	509±34	534±56	623±25	658±67
Control	6	25	364± 34	330±35	340±41	335±31	329±29
TQ	6	25	369± 26	408±38	449±26*	415±31	478±44*
EA/TQ	6	25	337± 12	346±33	390±38	404±33	368±36
EA	2	25	393± 72	520±76	502±74	398± 9	459±39
Control	6	35	218± 19	190± 5	190± 7	189± 4	190± 5
TQ	6	35	283± 29	232±16*	216±12	204± 8	203± 8
EA/TQ	5	35	227± 27	290±22*	260±18*	204± 9	223±20[a]
EA	2	35	338±132	228±19	273±79	246±69	---

\+ Mean values (milliliters, BTPS) ± SEM.
a n=3
* Significantly different from control ($p < 0.05$).

TABLE 21

Effects of Tranquilizer (TQ) and Electro-anesthesia (EA) on Blood Pressure+

Treatment	No of Trials	Ambient Temp.(°C.)	EXPOSURE INTERVALS (Mins.) Pretreatment 60-90 (n=42)	Electro-anesthesia Tranquilizer 115-160 (n=60)	165-210 (n=60)	Recovery 215-270 (n=72)
Control	6	5	120.6± 1.1	120.0±0.9	120.2±0.9	117.7±0.9
TQ	6	5	122.5± 1.0	108.1±1.6*	109.7±1.6*	106.0±1.5*
EA/TQ	6	5	121.6± 0.9	103.9±2.0*	101.0±1.8**	104.7±2.3*
Control	6	25	114.3± 2.1	117.5±1.5	116.5±0.8	113.7±2.3
TQ	6	25	114.0± 1.2	103.6±1.2*	105.8±2.0*	111.4±1.8
EA/TQ	6	25	115.6± 1.5	97.7±1.7*	106.6±1.8*	109.1±2.3
Control	6	35	111.2± 1.4	111.6±0.7	112.7±1.0	111.1±0.9
TQ	6	35	108.9± 1.0	104.6±1.4*	108.3±1.8	108.3±1.4
EA/TQ	5	35	112.7± 1.4	109.0±2.2	116.2±2.0	112.5±2.2

+ Mean blood pressure (mm Hg) + SEM.
* Significantly different from control ($p < 0.01$).
** Significantly different from control and TQ animals ($p < 0.01$).

TABLE 22

Effects of Electro-anesthesia (EA) on Blood Pressure+

Treatment	No. of Trials	Ambient Temp.(°C.)	EXPOSURE INTERVALS (Mins.) Pretreatment 60-90 (n=10)	Electro-anesthesia 115-160 (n=20)	Electro-anesthesia 165-210 (n=20)	Recovery 215-270 (n=24)
EA	2	5	121.9±1.4	140.9±0.9*	141.5±1.0*	137.1±1.1*
EA	2	25	120.6±2.4	136.9±1.2*	136.2±0.7*	130.3±2.5
EA	2	35	110.7±0.7	135.0±1.3*	134.0±1.1*	113.6±1.7

\+ Mean values (mm Hg) ± SEM.
* Significantly different from pretreatment values (60-90 min) ($p < 0.01$).

TABLE 23

Effects of Tranquilizer (TQ) and Electro-anesthesia (EA) on Heart Rate+

Treatment	No. of Trials	Ambient Temp.(°C.)	EXPOSURE INTERVALS (Mins.) Pretreatment 60-90 (n=42)	Electro-anesthesia Tranquilizer 115-160 (n=60)	165-210 (n=60)	Recovery 215-270 (n=72)
Control	6	5	86.1±3.6	84.2±2.4	84.2±2.4	79.5±1.8
TQ	6	5	88.2±3.5	132.3±6.5*	141.4±5.6*	146.3±5.5*
EA/TQ	6	5	89.8±2.3	96.5±3.6	147.9±3.0*	179.9±4.6**
Control	6	25	67.0±2.3	63.2±1.1	61.2±0.9	65.1±0.9
TQ	6	25	62.6±1.8	79.4±3.7*	80.8±5.0*	92.5±4.6*
EA/TQ	6	25	69.7±1.3	67.9±1.2	100.1±2.8**	121.4±2.5**
Control	6	35	71.0±4.2	65.7±1.5	65.3±1.3	67.1±1.6
TQ	6	35	65.0±1.7	75.1±2.1*	80.2±2.2*	82.8±1.3*
EA/TQ	5	35	64.7±2.3	60.6±1.9	85.6±2.4*	108.4±3.5**

\+ Mean values (beats/min) ± SEM.
* Significantly different from control ($p < 0.01$).
** Significantly different from control and TQ animals ($p < 0.01$).

TABLE 24

Effects of Electro-anesthesia (EA) on Heart Rate+

Treatment	No. of Trials	Ambient Temp.(°C.)	EXPOSURE INTERVALS (Mins.) Pretreatment 60-90 (n=10)	Electro-anesthesia 115-160 (n=20)	Electro-anesthesia 165-210 (n=20)	Recovery 215-270 (n=20)
EA	2	5	94.2±9.5	81.1±1.5	96.0±2.9	132.8±10.4*
EA	2	25	70.3±1.3	74.7±1.7	71.7±0.8	75.6± 1.9
EA	2	35	69.9±2.0	90.1±1.4*	94.5±1.3*	114.6± 8.0*

\+ Mean values (beats/min) ± SEM.
* Significantly different from pretreatment (60-90 min) values ($p < 0.01$).

TABLE 25

Effects of Tranquilizer (TQ) and Electro-anesthesia (EA) on Arterial Hematocrit (%)+

Treatment	Ambient Temp.(°C.)	Pretreatment	Electro-anesthesia Tranquilizer		Recovery	
		90	150	210	240	300
Control	5	26.8±1.4(8)[a]	26.3±1.0(8)	24.7±0.9(8)	25.6±0.5(8)	24.3±0.7(6)
TQ	5	28.0±0.5(5)	28.5±1.0(6)	27.1±0.9(4)	27.4±0.5(6)	26.9±0.5(4)
EA/TQ	5	27.3±0.3(9)	31.3±1.5(10)*	31.1±1.2(10)**	33.0±0.5(10)**	31.6±1.0(10)**
EA	5	30.8±1.2(3)	34.0±1.5(3)	33.3±1.2(3)	29.3±0.9(3)	29.0±0.6(4)
Control	25	25.5±0.5(5)	25.4±1.1(6)	26.3±1.1(6)	27.0±1.1(6)	27.3±1.1(6)
TQ	25	25.7±1.2(6)	22.9±0.5(6)	23.3±0.5(6)	29.6±1.9(6)	31.6±2.4(5)
EA/TQ	25	25.3±0.5(8)	25.3±0.7(8)	29.4±0.8(8)	29.6±1.1(8)	26.6±1.2(4)
EA	25	29.8±0.9(4)	34.0±2.0(4)	34.3±1.9(4)	30.5±2.6(4)	31.3±2.5(4)
Control	35	24.6±0.7(6)	23.5±0.2(4)	23.1±0.5(6)	24.4±0.6(6)	24.3±0.1(4)
TQ	35	24.3±1.0(4)	23.4±0.4(4)	29.3±4.5(4)	26.3±2.6(4)	---
EA/TQ	35	23.9±0.2(5)	24.5±0.5(6)	27.4±1.3(6)*	25.4±1.5(5)	20.5±2.9
EA	35	31.5±0.9(2)	33.0±1.5(2)	33.0±1.5(2)	29.5±1.5(2)	28.5±1.0

\+ Mean values ± SEM.
a Number of samples
* Significantly different from control ($p < 0.05$).
** Significantly different from control and TQ animals ($p < 0.05$).

Figure 1. The sheep was restrained in the environmental chamber by an open and mobile restraining stanchion (RS) and canvas sling (SL) supported by a pulley system attached to angle iron at the top of the stanchion. Respiratory frequency was obtained using a mercury-in-rubber strain gauge (SG) encircling the abdomen. The animal's head was placed in a cylindrical, clear plastic hood (HD) through which an air flow (50 1/min.) was drawn and sampled by an O_2 analyzer and humidity transducer (HT). The carotid loop (CL) was catheterized with a 20 g. needle attached to polyethylene tubing for blood pressure (BP) and blood sampling (BS). Skin temperatures were measured at sites designated by t. Temperature within the chamber was sensed by a thermistor probe (TP) and regulated by a temperature controller (TC) which governed the pumping of heated (HP) or cooled liquid (CP) through two convective heat exchangers (CH). Fans (F) maintained an air flow across the CH's. Chamber air was circulated by a fan (CF) and cycled through a dehumidifier (DeHU) or humidifier (HU).

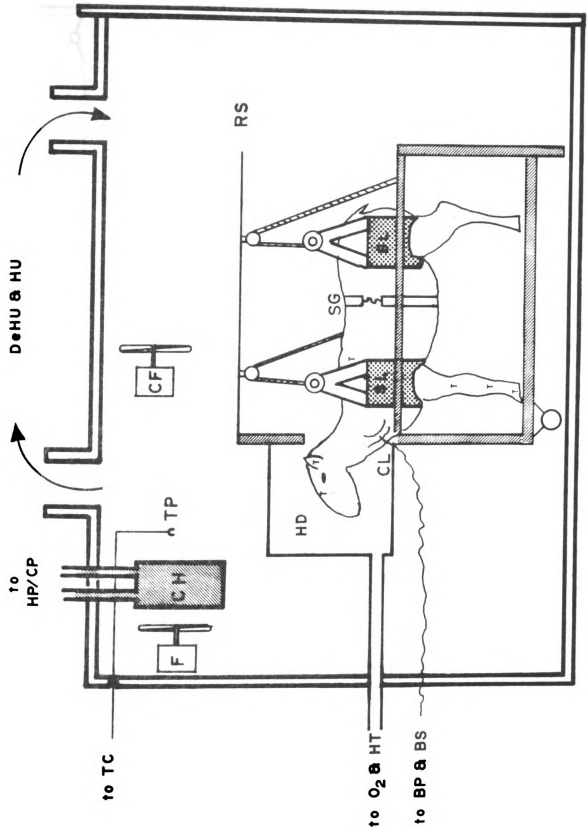

Figure 1

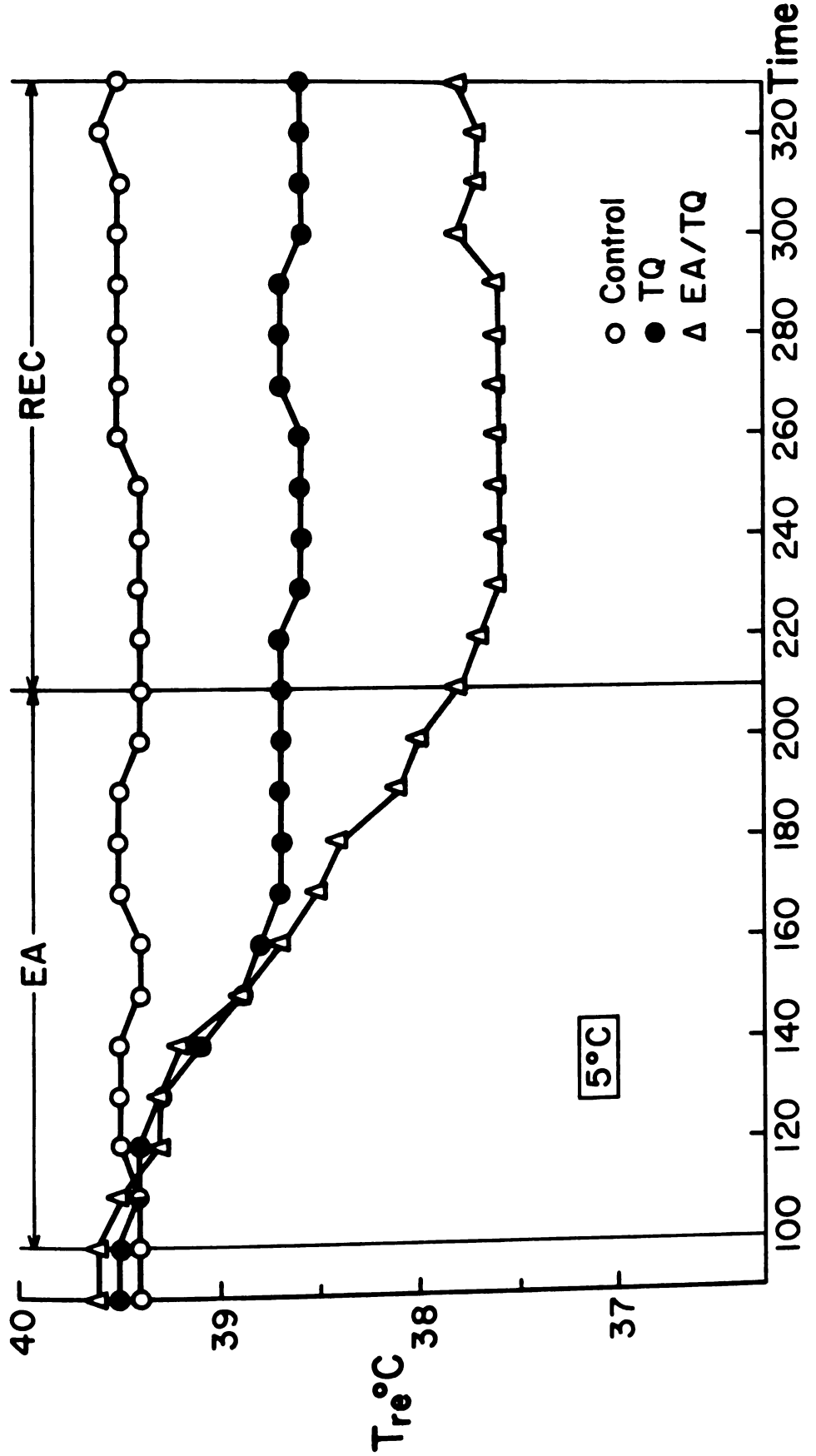

Figure 2. Rectal temperature (T_{re} in °C.) of control, tranquilized (TQ) and tranquilized-electro-anesthetized (EA/TQ) animals during exposure to the 5°C. ambient temperature as a function of time (min). The electro-anesthesia (EA) and "recovery" (REC) periods are indicated for the EA/TQ animals.

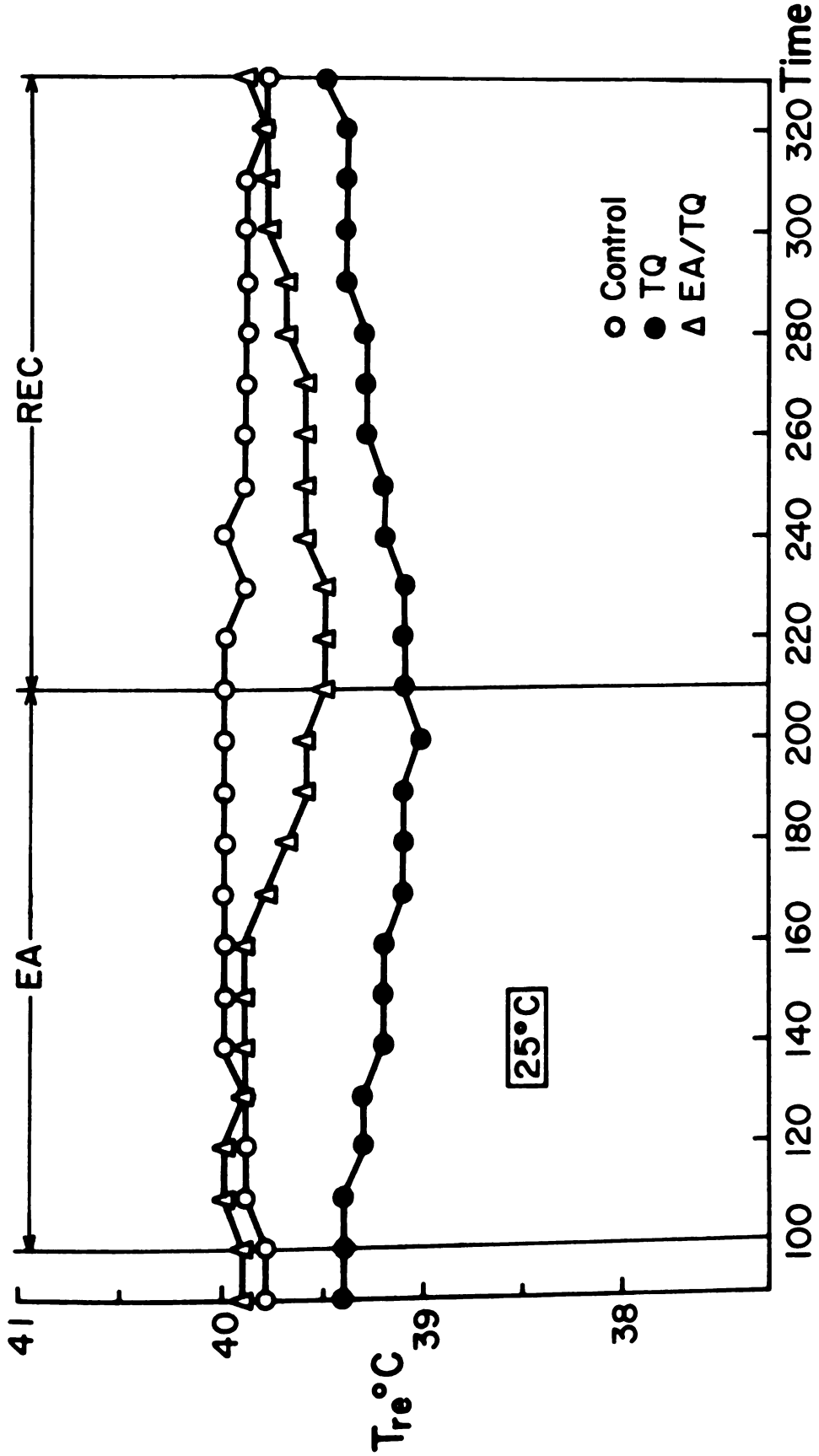

Figure 3. Rectal temperatures (T_{re} in °C.) of control, tranquilized (TQ) and tranquilized-electro-anesthetized (EA/TQ) animals during exposure to the 25°C. ambient temperature as a function of time (min.). The electro-anesthesia (EA) and "recovery" (REC) periods are indicated for the EA/TQ animals.

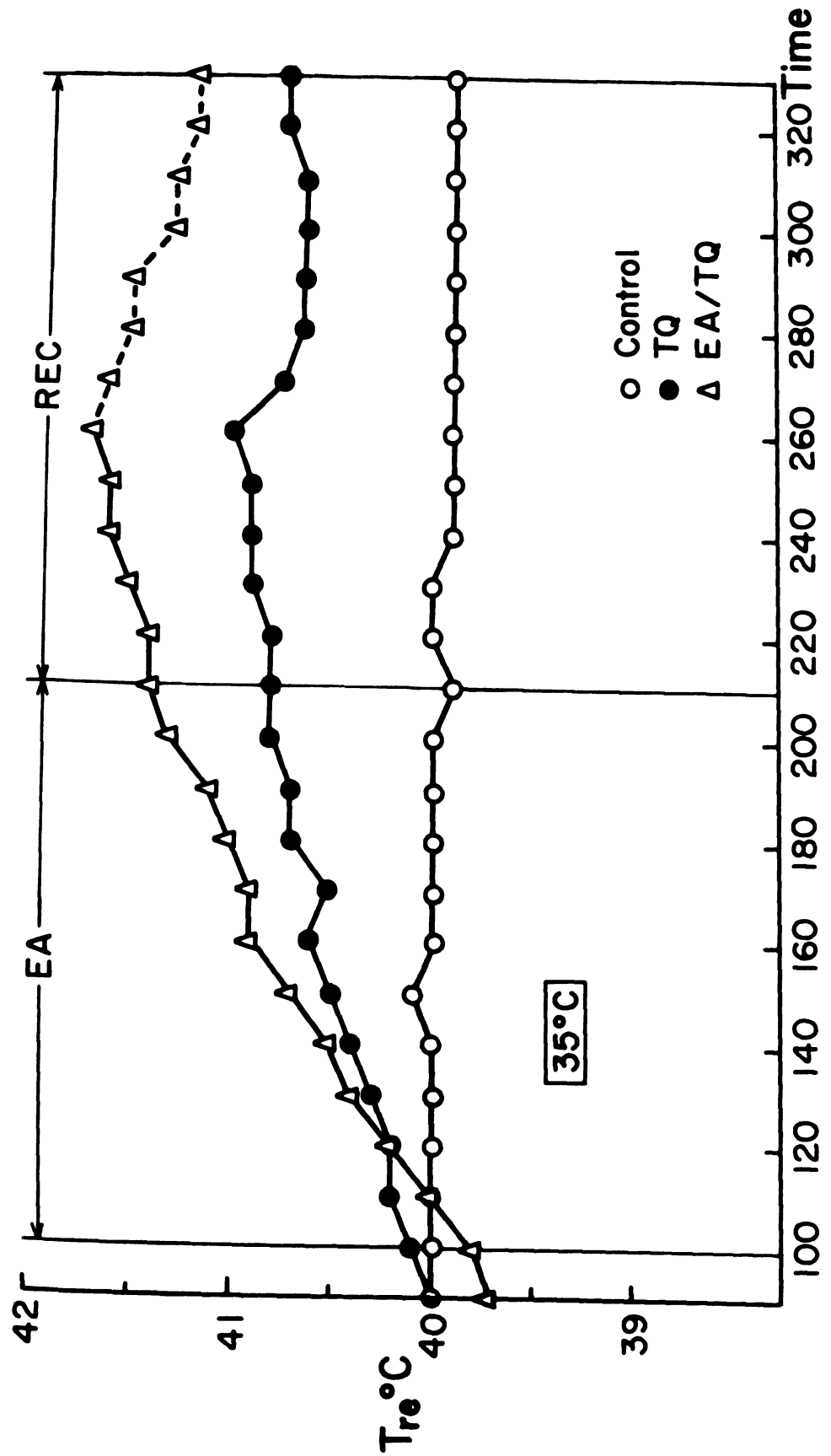

Figure 4. Rectal temperature (T_{re} in °C.) of control, tranquilized (TQ) and tranquilized-electro-anesthetized (EA/TQ) animals during exposure to the 35°C. ambient temperature as a function of time (min). The electro-anesthesia (EA) and "recovery" (REC) periods are indicated for the EA/TQ animals. Dotted line (—) denotes n=2.

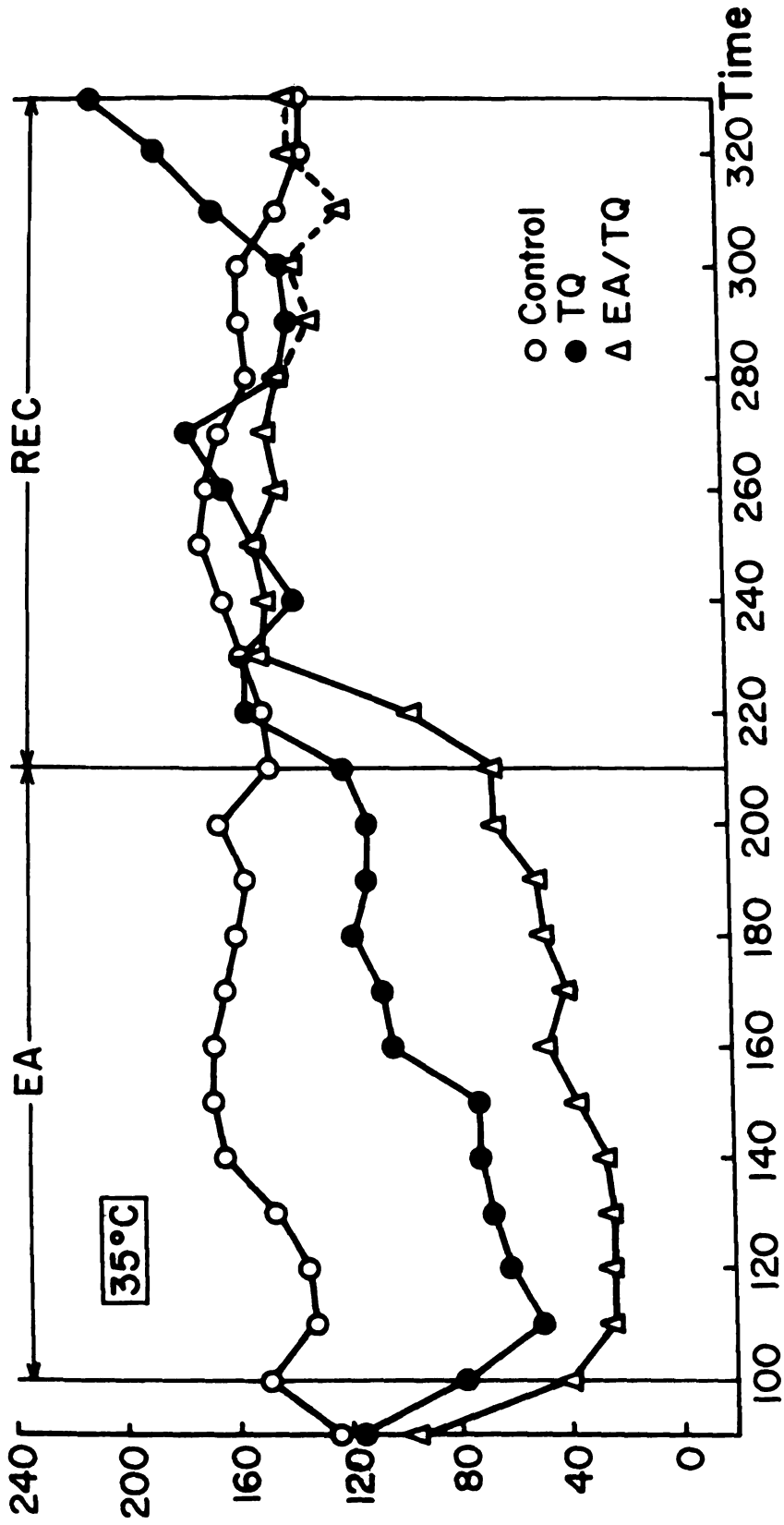

Figure 5. Respiratory frequency (f in breaths/min) of control, tranquilized (TQ) and tranquilized-electro-anesthetized (EA/TQ) animals during exposure to the 35°C. ambient temperature as a function of time (min). The electro-anesthesia (EA) and "recovery" (REC) periods are indicated for the EA/TQ animals. Dotted line (--) denotes $n=2$.

DISCUSSION

Whenever a homeotherm is exposed to a thermal stress, the animal must invoke certain biothermal (i.e., thermoregulatory) responses in order to maintain a nearly constant deep body temperature. The thermoregulatory adjustments involve both physical and chemical mechanisms to achieve an appropriate balance between heat production and heat dissipation, conservation or gain. Data reported here (Figures 2, 3 and 4) indicate that untreated sheep (control group) during whole body exposure to a cold (5°C.), neutral (25°C.), and hot (35°C.) thermal environment were capable of maintaining rectal temperature within the limits previously described for sheep (Lee, 1950; Kammlad, 1947; Knapp and Robinson, 1954). However, the thermoregulatory ability of these same animals was severely impaired by propiopromazine or electrical anesthesia administered separately or together.

During whole body cold exposure (5°C.), the initial rate of fall in the internal body temperature of the sheep administered electro-anesthesia following propiopromazine medication (EA/TQ), paralleled that of the sheep receiving the tranquilizer alone (TQ). Although the tranquilized sheep showed a plateau in T_{re} approximately 80 minutes after medication, T_{re} continued to fall in the EA/TQ animals

beyond the duration of anesthesia (Figure 2). A similar displacement of deep body temperatures from untreated animals, only in the reverse direction, was observed for these same treatment groups (TQ and EA/TQ) at the 35°C. T_a (Figure 4). Also, the T_{re}'s of the EA/TQ animals continued to rise after the termination of electro-anesthesia whereas, the TQ animals showed a plateau in T_{re} at 110 minutes after medication. The discontinuous fall in T_{re} of the tranquilized sheep suggests that propiopromazine alone may reduce the precision of body temperature control without necessarily shifting the "set point" of temperature regulation. This loss in precision of control allows deep body temperature to shift markedly when the animal is heat or cold stressed. Shifts in the "set point" of thermoregulatory control have been postulated by Hammel et al. (1963). Propiopromazine treatment coincident with electrical anesthesia thereby may account for a portion of the ensuing thermoregulatory impairment observed in these animals. Data reported in Table 1, support this hypothesis in that the animal electro-anesthetized without the tranquilizer at the 5°C. T_a, exhibited no appreciable net body heat loss as indicated by colonic temperature. On the other hand, this same animal demonstrated a progressive hyperthermia during electro-anesthesia at the 35°C. T_a (Table 3). Data reported in Figure 4 suggest that the initial rate of rise in T_{re} of the EA/TQ animals was more rapid than that observed when

these same animals were administered only propiopromazine or electro-anesthesia. It appears that during heat stress (i.e., $T_a = 35^{\circ}C.$), the thermal load evidenced in the EA/TQ animals may be a combined effect of both treatments.

The continuous fall in T_{re} at the $5^{\circ}C.$ T_a and elevation in T_{re} at the $35^{\circ}C.$ T_a observed in the EA/TQ sheep after the termination of anesthesia, suggests that thermoregulatory control is not completely restored upon current cessation. Also, at the $35^{\circ}C.$ T_a, the progressive elevation in T_{re} after electro-anesthesia, exhibited by the animal which was not premedicated with propiopromazine, suggests that these post-anesthetic changes in deep body temperature may be related to current induced thermoregulatory alterations and not necessarily with concurrent tranquilization. Even though the animals regained consciousness immediately after electro-anesthesia, they remained stunned for a variable period of time. This observation may be related to the apparent inability of these animals to restore thermal balance after anesthesia. This stunned appearance has also been observed in dogs after electro-anesthesia (Smith, 1963).

It would appear that the biothermal defense of deep body temperature is not as demanding during exposure to the $25^{\circ}C.$ ambient temperature since tranquilized and/or electro-anesthetized animals were able to maintain deep body temperature close to that of untreated animals. This may be accounted for by the near proximity of the $25^{\circ}C.$ exposure

temperature to the reported thermoneutral zone for adult shorn sheep (Blaxter et al., 1959). However, Shiakh et al. (1968) reported a significant elevation in the T_{re} of propiopromazine treated sheep at the end of 2 hours of electro-anesthesia during exposure to ambient temperatures of 18°C. and 27°C. which they assumed to be "thermoneutral" and "heat stressing" exposures. However, the animals used in the above study were unshorn and of widely varying body weights, making comparisons with the present study difficult.

The greatest biothermal effects of propiopromazine and electro-anesthesia were observed at T_a's 5°C. and 35°C. where the animals must rely upon "active" heat dissipation or conservation. The peripheral vasodilator actions of phenothiazine tranquilizers have been well documented (Courvoisier et al., 1953) and shown to facilitate heat exchange in superficial vessels of the rat (Kollias and Bullard, 1964). During cold exposure, the increased ear and face skin temperatures for the groups administered propiopromazine or electro-anesthesia suggest a reduction in peripheral vasomotor tone and thereby increased heat loss in these areas. In addition, Tables 4 and 5 relate that animals administered electro-anesthesia concurrent with propiomazine exhibited a greater heat loss in these areas than when the tranquilizer was given alone. Electro-anesthesia alone may facilitate decreased vasomotor tone in these areas (Tables 4 and 5). However, when propiopromazine

is administered with electro-anesthesia, the treatments appear to be additive in respect to vasomotor responses and heat loss. Presumably, this may have contributed to the greater reduction in T_{re} of the tranquilized-anesthetized group when compared to either treatment given independently. An analogous response in ear skin temperatures was found at the 25°C. exposure temperature. However, at the 35°C. T_a neither tranquilized or electro-anesthetized animals exhibited ear or face skin temperatures higher than control animals, suggesting that superficial ear and face skin vessels of the control animals were maximally vasodilated at this T_a.

Even though ear and face skin temperatures were elevated above those measured in untreated animals, the TQ and EA/TQ animals showed no significant increase in forelimb skin temperatures at T_a 5°C., whereas significant increases in forelimb skin temperatures were observed for these treatment groups at the 25°C. and 35°C. exposure temperatures (Tables 6, 7 and 8). This suggests that neither propiopromazine nor electro-anesthesia completely inhibits thermally-induced vasomotor responses in all superficial vasoactive vessels. Furthermore, these inconsistent vasomotor responses at different T_a's may be indicative of varied thermal thresholds in different peripheral vascular beds, and suggests some limited thermoregulatory capability during tranquilization and/or electro-anesthesia. Massion and Downs (1969),

reported that dogs under electro-anesthesia (at "room" temperature) consistently showed vasoconstriction in an isolated forelimb preparation. This is in direct contrast to the suggested vasodilation observed in this study with the sheep anesthetized at T_a's of 25°C. and 35°C., with and without concomitant tranquilization (Tables 6, 7 and 8). Others have reported changes in paw temperatures of electro-anesthetized dogs (at "room" temperatures) to be variable, either increasing or remaining at their initial temperature (Cuthbertson et al., 1965). These reported changes in paw temperatures may reflect some vasomotor adjustments to small non-measured changes in ambient temperature or they may suggest some thermoregulatory vasomotor responses during electro-anesthesia. The increases in forelimb skin temperatures of the TQ and EA/TQ animals at the 25°C. T_a may be related to the sympatholytic vasomotor actions of propiopromazine whereas the increases at the 35°C. T_a may also reflect the increased deep body temperature compared to untreated animals.

During cold stress, homeotherms must increase heat production and reduce heat dissipation in order to maintain a constant deep body temperature. Increased heat production is accompanied by increases in metabolic rate reflected by corresponding increases in $\dot{V}_{O_2}$. The large depression in oxygen consumption observed in the animals administered electro-anesthesia with propiopromazine compared to control and TQ animals at the 5°C. T_a (Table 10), suggests a decrease

in heat production which probably contributed to the lowered rectal temperature exhibited by the EA/TQ group (Figure 2; Table 1). This depressed heat production may be in part accounted for by a general suppression of shivering thermogenesis which lasted approximately 50 minutes after initial current application in the EA/TQ animals compared to 25 minutes in propiopromazine-treated sheep. This inhibition of shivering may be associated with tranquilizer medication since phenothiazine tranquilizers have been reported to reduce skeletal muscle tone (Henatsch and Ingvar, 1965). Similarly, chlorpromazine has been shown to decrease oxygen consumption and shivering in rats during whole body cold exposure (Kollias and Bullard, 1964). The longer suppression of shivering and greater fall in $\dot{V}_{O_2}$ observed in the EA/TQ group compared to the TQ sheep suggests an additive effect of electro-anesthesia on the tranquilizer-induced suppression of whole body metabolism. In addition, the lowered rectal temperatures in the tranquilized-anesthetized sheep at 5°C. T_a may also involve a Q_{10} effect[1] on whole body metabolism.

In contrast to the generally depressed $\dot{V}_{O_2}$ of the tranquilized sheep at 5°C., at the 25°C. T_a, $\dot{V}_{O_2}$ was

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[1]Since enzymatic reactions are temperature dependent, the higher the T_{re}, the faster the rate of metabolism, or vice versa as expressed by the Q_{10} factor.

significantly elevated over the control animals (Table 10). Figure 3 shows T_{re} of the TQ animals to be lower than that of the untreated animal at the 25°C. T_a. The elevated $\dot{V}_{O_2}$ may be associated with an increased heat production in order to sustain deep body temperature in face of the proportionately greater peripheral vascular heat loss (e.g., ear and forelimbs, Tables 4, 6, 7 and 8) than observed in control animals. Heat loss in the tranquilized sheep at T_a 25°C. appears to be associated with peripheral vasomotor changes rather than with a reduction in heat production. The EA/TQ animals at the 25°C. T_a also showed a slight elevation in $\dot{V}_{O_2}$ and presumably heat production when T_{re} began to fall during the second hour of anesthesia (Figure 3, Table 10). Oxygen consumption also remained elevated over control values after the termination of electro-anesthesia, and was accompanied by a progressive rise in T_{re}. This observation is similar to that of McIntyre and Voloshin (1964) who also reported increased $\dot{V}_{O_2}$ (20 to 100%) in the dog under electro-anesthesia compared to measurements under thiopentone anesthesia.

At the 35°C. exposure temperature, the TQ and EA/TQ groups showed closely paralleled metabolic responses as evidenced by $\dot{V}_{O_2}$ measurements. The depressed $\dot{V}_{O_2}$ during the first hour of their respective treatments may be indicative of a generalized reduction in heat production occurring simultaneously with the observed rise in deep body temperature (Figure 4, Table 10) or the reduced $\dot{V}_{O_2}$ may reflect a

general inhibition of thermal polypnea observed initially in both treatment groups (Figure 5). The subsequent elevation in $\dot{V}_{O_2}$ during the "recovery" period was probably associated with an observed resumption of panting. A Q_{10} effect may also be a significant factor contributing to increased heat production in view of the elevated deep body temperature during the "recovery" period (Figure 4). Whittlow and Findlay (1968) reported that in cattle, only a portion of the increased heat production during heat stress is accounted for by the muscular activity of panting and that the major portion is attributable to the Q_{10} effect of increased rectal temperature.

In summary, it appears that increases in heat production dependent upon shivering are reduced by both propiopromazine and electro-anesthesia, most severely when both are administered concurrently. However, elevated heat production not dependent upon shivering does not appear to be appreciably altered by either or both treatments. Some investigators have postulated that increased heat production is coupled to the enhanced general somatic muscle tone commonly observed in electro-anesthetized subjects (Geddes et al., 1964; Geddes, 1965; Herin, 1964). However, muscle hypertonicity did not appear to be particularly prominent in this study except in one pilot study where a rapid induction technique was tested. Moreover, an elevation in $\dot{V}_{O_2}$ due to increased muscle tone was not apparent. Smith et al. (1966)

have attributed inadequate muscle relaxation during electro-anesthesia to improper placement of electrodes. Muscle tremors were reduced by Wulfsohn and McBride (1962) by varying the current frequency. Respiratory evaporative water loss (E) has been reported to be a significant factor in maintaining heat balance in sheep and may be the principle method of evaporative cooling (Brook and Short, 1960; Brockway et al., 1965). However, the effectiveness of respiratory evaporation as a cooling mechanism depends upon the respiratory frequency and tidal volume, ambient temperature and relative humidity, varying directly with respiratory frequency and tidal volume and inversely with relative humidity. Also respiratory evaporative water loss is an effective heat dissipating mechanism only at ambient temperatures below the deep body temperature of the subject.

Data reported in Table 14, suggest an increased respiratory evaporative heat loss during cold exposure ($5^{o}C.$) for animals administered propiopromazine alone or coincident with electro-anesthesia compared to the untreated animals. Since the sheep administered only electro-anesthesia (without tranquilization) showed a decrease in E (Table 15) from pretreatment values, the elevated E observed in the TQ and EA/TQ treatment groups may be related to propiopromazine medication. Moreover, this enhanced respiratory heat loss may have been a contributing factor to the fall in T_{re} observed in these treatment groups (Figure 2). The increased

E for the EA/TQ group was accompanied by a significant reduction in respiratory frequency (Tables 12 and 14), implying that tidal volume was increased even though the calculated V_T at both time intervals during anesthesia were slightly lower than control animals (Table 20). Although the computed V_T for the TQ group was slightly greater than control animals only at 150 minutes, V_T was probably increased in the TQ group since the increased E (Table 14) and unaltered f (Table 12) suggests an undetected increase in V_T following propiopromazine treatment.

At the 25°C. T_a, both the TQ and EA/TQ animals exhibited a significant reduction in f (Table 12) and a small elevation in V_T (Table 20). The increase in V_T was greater for the TQ animals than the EA/TQ group reflected by a correspondingly greater E for the TQ group than the EA/TQ animals during the first hour of their treatments. The decreased respiratory heat loss in the EA/TQ group was compensated by increased peripheral heat loss (i.e., ears and forelimbs) so that T_{re} remained essentially unchanged. The significant increase in E (Table 15) observed in the EA animal (T_a = 25°C.) appears to be related to an increased V_T since respiratory frequency was not different from pretreatment values (Table 13).

The inhibition of hyperthermic polypnea both by propiopromazine and/or electro-anesthesia at the 35°C. T_a probably contributed more than any other single factor to

the elevation in rectal temperature observed in these treatment groups (Figure 5). Even though V_T apparently increased, the decrease in f severely compromised respiratory evaporative cooling as evidenced by the large decrease in E for both TQ and EA/TQ groups when compared to control animals (Figure 11). Furthermore, the depression in respiratory frequency for the EA/TQ group was twice that observed for the TQ group (Table 12). This was closely paralleled by nearly equivalent reductions in E for these two groups (Table 14). This may account, in part, for the greater rise in T_{re} observed in the EA/TQ sheep than in the TQ animals. Even though the animal administered electro-anesthesia without tranquilizer showed a significant increase in respiratory frequency and reduced V_T during anesthesia from pretreatment values, it appears respiratory frequency was dampened since it doubled after the termination of anesthesia (Table 13). A similar increase in respiratory frequency with a 50% reduction in V_T occurred after cessation of current application in the EA/TQ group. It appears, therefore, that both propiopromazine and electro-anesthesia reduce respiratory evaporative cooling during heat stress by the depression of hyperthermic polypnea. Moreover, this depression is greater when both propiopromazine and electro-anesthesia are administered simultaneously than when either is given independently. These results are in agreement with Shiakh et al. (1968) who reported a significant reduction in

respiratory frequency in sheep administered propiopromazine alone or coincident with electro-anesthesia. They also observed large increases in respiratory rate following the conclusion of electro-anesthesia. Higgins et al. (1964) also observed a decrease in panting in propiopromazine-treated dogs during heat stress.

The general depression in respiratory frequency, uniformly observed in the EA/TQ treatment group independent of exposure temperature, may be related to a general decrease in $\dot{V}_A$ and increased arterial P_{CO_2} particularly during the first hour of anesthesia. The decreased $\dot{V}_A$ appears to be more pronounced at the 5°C. T_a than at other exposure temperatures. This may be attendant to a Q_{10} effect on ventilatory control mechanisms in view of the lower T_{re} of the EA/TQ group compared to TQ and untreated animals. Although the TQ animals also showed a significantly reduced f at T_a's 25°C. and 35°C., V_T was generally increased so that $\dot{V}_A$ and P_{CO_2} were not appreciably changed.

The increased arterial P_{CO_2} in the EA/TQ group at all T_a's was accompanied by a nearly proportionate decrease in arterial pH. This observation is similar to that of Larson and Sances (1968) who reported a decreased arterial pH in squirrel monkeys under electro-anesthesia. The results of Herin (1968) suggested that the observed arterial P_{CO_2} changes in dogs may be associated with the current wave form applied. He found a decrease in P_{CO_2} with square,

pulse triangular and saw tooth wave forms with a slight increase during sine wave anesthesia. However, arterial pH was uniformly decreased with all wave forms applied. Hardy and co-workers (1961) also found a decrease in arterial pH even though the dog was hyperventilated by a mechanical respirator. Therefore, it appears that the fall in arterial pH may have both respiratory and metabolic components, however, arterial HCO_3^- concentration did not vary appreciably with either tranquilization or electro-anesthesia administered alone or together (Table 18). During the "recovery" period at the 35°C. exposure temperature, both the EA/TQ and EA groups showed decreased arterial P_{CO_2} and slightly increased $\dot{V}_A$ from control values indicating that a mild hyperventilation occurred when panting resumed.

$\dot{V}_A$ in control animals was greater at the thermally stressing exposure temperatures (e.g., 5 and 35°C.) than at the neutral exposure (25°C.) even though arterial P_{CO_2} decreased as a function of T_a. The increased $\dot{V}_A$ and P_{aCO_2} during cold exposure relative to the thermoneutral exposure may represent an increased oxygen demand and carbon dioxide production during shivering. However, the increase in $\dot{V}_A$ and reduced P_{aCO_2} at the 35°C. T_a, compared to the 25°C. T_a, may indicate that during panting the sheep is hyperventilating and not exchanging only dead space air as a cooling mechanism. These observations are similar to those reported by Heisey et al. (1970) for the heat-stressed goat.

The cardiac responses to electrical anesthesia are appreciably different when propiopromazine is administered as a preanesthetic. The animal which was given only electro-anesthesia showed a significant increase in arterial blood pressure at all ambient temperatures and an increase in heart rate at the 35°C. ambient temperature (Tables 22 and 24). This suggests that the blood pressure elevation particularly at the T_a's of 5 and 25°C. may be associated with an increase in total peripheral vascular resistance resulting from generalized vasoconstriction. Hypertension has been a common observation in animals under electro-anesthesia (Knutson et al., 1956; Hardy et al., 1961; Herin, 1963). Herin (1968) who examined cardiovascular effects during application of five different current wave forms in dogs, observed a uniform increase in arterial blood pressure and heart rate with all current forms tested. In contrast, the sheep which were premedicated with propiopromazine prior to electro-anesthesia showed a significant reduction in arterial blood pressure at all exposure temperatures (Table 21). Since sheep which were treated with the tranquilizer alone also showed a reduction in arterial blood pressure from untreated animals, it appears the hypotension observed during electro-anesthesia is associated with concurrent propiopromazine medication. Cuthbertsen et al. (1965) also observed that hypertension was reduced in electro-anesthetized animals given prior doses of tranquilizers. However, the present

study suggests that when electro-anesthesia is administered in conjunction with propiopromazine, the fall in arterial blood pressure is greater than when the tranquilizer is given alone (Tables 21 and 22). This suggests that electro-anesthesia enhances the reported vasodilator properties of the tranquilizer and is in conflict with the hypertensive response observed when electro-anesthesia is given without prior medication. The decrease in arterial blood pressure for both TQ and EA/TQ treatment groups from control animals was more pronounced at 5°C. and 25°C. exposure temperatures than at the 35°C. T_a. However, the arterial blood pressure in the control sheep was also decreased as exposure temperature increased, presumably reflecting vasodilatory heat dissipation. This decrease in arterial blood pressure of the sheep at the 35°C. T_a, probably minimized vascular sympatholytic actions of propiopromazine at this temperature.

In general, heart rate increased in proportion to the reduction in blood pressure in both TQ and EA/TQ animals (Tables 21 and 23). This suggests that baroceptor reflexes were not appreciably altered by either propiopromazine or electro-anesthesia. However, the fall in blood pressure was not completely compensated by the reflex tachycardia.

Since a rapid induction procedure was not employed to induce electro-anesthesia in this study, the reported sudden increase in blood pressure and heart rate concurrent with initial current application (Knutson, 1954; Hardy et al.,

1969; Herin, 1963) was not observed. A progressive increase in blood pressure and heart rate, however, was observed in this study as current strength was increased. With anesthetic current at "maintenance" levels, animals premedicated with propiopromazine had blood pressure levels below that observed during the pretreatment phase. In contrast, the animal receiving only electro-anesthesia showed an elevation in blood pressure lasting throughout the anesthesia period. These studies suggest that tranquilizer medication is beneficial if a gradual electrical anesthesia induction technique is employed since it reduces excitement and generally lowers the current strength required to obtain anesthesia.

An elevation in arterial hematocrit was observed in animals administered electro-anesthesia independent of prior medication and exposure temperature (Table 25). Frostig et al. (1944) have attributed this to contraction of the spleen in electro-anesthetized dogs. Others have reported that the elevation in hematocrit is a function of current intensity (Power and Wood, 1964) or wave form (Herin, 1968). In the present study data suggest a splenic contraction during electrical anesthesia but not during propiopromazine medication in the sheep.

SUMMARY AND CONCLUSIONS

Biothermal and cardiorespiratory responses in three closely shorn adult sheep (of similar breed, sex and weight) administered propiopromazine HCl (1.0 mg/kg) and electro-anesthesia (700 c.p.s. A.C., bitemporal electrodes) individually and concurrently were investigated during a 330 minute whole body exposure to three environmental temperatures: $5^{o}C.$, $25^{o}C.$ and $35^{o}C.$ Each animal was exposed twice untreated, twice tranquilized and twice tranquilized and electro-anesthetized (110 minutes) to each of the 3 ambient temperatures. One of the three animals was twice electro-anesthetized (110 minutes) without prior tranquilization at each of the exposure temperatures. Rectal and 7 skin temperatures, oxygen consumption, respiratory frequency, evaporative water loss, arterial blood pressure and heart rate were monitored and recorded at 5 or 10 minute intervals during the entire exposure period. Measurements of arterial carbon dioxide tension (P_{aCO_2}), pH, bicarbonate concentration and hematocrits were obtained from serial blood samples drawn from the carotid artery at designated time intervals. Alveolar ventilation ($\dot{V}_A$) and tidal volume (V_T) were computed for different time intervals.

During the 5°C. exposure, rectal temperatures decreased in both tranquilized (0.8°C.) and tranquilized-electro-anesthetized animals (1.6°C.) from control animals after 2 hours of their respective treatments. However, the sheep administered electro-anesthesia without prior tranquilization did not show any appreciable change in deep body temperature after 2 hours of anesthesia. The depression in internal body temperature in the animals receiving propiopromazine was associated with increased heat loss via superficial vessels of the ear and face, which was greater when propiopromazine and electro-anesthesia were given simultaneously than individually. In addition, shivering was more severely inhibited when tranquilization and electro-anesthesia were administered concurrently than independently. The depression in oxygen consumption in the above groups was interpreted as decreased heat production coincident with the suppression of shivering. Also, an increased heat loss via respiratory surface evaporation was observed in both tranquilized and tranquilized-anesthetized animals ($T_a = 5°C.$) even though respiratory frequency was unchanged or decreased, respectively, from control animals, indirectly suggestive of an increased V_T. Alveolar ventilation was reduced in the tranquilized-anesthetized animal at this exposure temperature.

Little difference was exhibited in rectal temperatures of tranquilized and/or electro-anesthetized animals at the 25°C. environmental temperature when compared to

untreated or pretreatment measurements. Neither electrical anesthesia or propiopromazine administration appears to interfere with heat production not dependent upon shivering (evidenced by $\dot{V}_{O_2}$ measurements), invoked in these animals to overcome the increased peripheral heat dissipation (e.g., ears and forelimbs). A decrease in respiratory frequency, with a small increase in V_T, was observed in tranquilized and tranquilized-electro-anesthesia animals compared to control animals.

At the 35°C. ambient temperature, the elevation in deep body temperature from untreated animals observed in tranquilized (0.9°C.), tranquilized-electro-anesthetized (1.5°C.) and electro-anesthetized animals (1.5°C.) was attributed to a reduction in respiratory evaporative cooling. Respiratory evaporative water loss was most severely affected when both treatments were administered simultaneously and attendant to a dampening of hyperthermic polypnea. Increased somatic muscle tone, commonly reported during electrical anesthesia, was not considered an important factor contributing to the increase in total body heat content since it was not observed, and rectal temperature continued to rise after anesthesia. These results indicated impairment of both heat loss and conservation mechanisms in tranquilized and electro-anesthetized animals, the effects being additive when treatments are combined at thermally stressing ambient temperatures. Nevertheless, sheep appear to retain

some limited thermoregulatory capability as evidenced by increases in $\dot{V}_{O_2}$ attendant to a lowered rectal temperature and some vasomotor changes in forelimbs, when treated with propiopromazine and/or electro-anesthesia.

Arterial P_{CO_2} increased during electrical anesthesia in animals pretreated with propiopromazine at all exposure temperatures. This was related to the decrease in arterial pH and fall in respiratory frequency observed in these animals. Smaller elevations in P_{aCO_2} (and decreases in pH) were observed when propiopromazine and electrical anesthesia were administered independently.

Arterial blood pressure was reduced in animals given propiopromazine alone and with electro-anesthesia. The reduction in blood pressure was greater when treatments were administered in combination than independently and was more pronounced during the 5°C. and 25°C. environmental temperatures than during the 35°C. exposure. Increases in heart rate were generally inversely related to the reduction in blood pressure and in proportion to the magnitude of the fall in blood pressure. In contrast, the animal administered electro-anesthesia without prior medication exhibited an increase in blood pressure, even though its heart rate was accelerated only at the 35°C. exposure temperature.

Arterial hemotocrits were increased during the application of electrical anesthesia but unchanged from control animals when only propiopromazine was administered.

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APPENDICES

APPENDIX A

FREQUENTLY USED SYMBOLS

T_a	Ambient Temperature
T_{re}	Rectal Temperature
T_e	Ear Skin Temperature
T_f	Face Skin Temperature
T_{ll}	Lower Forelimb Skin Temperature
T_{ml}	Mid-Forelimb Skin Temperature
T_{ul}	Upper Forelimb Skin Temperature
$\dot{V}_{O_2}$	Oxygen Consumption
P_{aCO_2}	Arterial Carbon Dioxide Tension
f	Respiratory Frequency
$\dot{V}_A$	Alveolar Ventilation
V_T	Tidal Volume

APPENDIX B

STATISTICAL EQUATIONS

Grand mean $\bar{X} = \frac{\Sigma \bar{x}}{n}$

where: $\Sigma \bar{x}$ = sum of individual means

n = number in sample

Standard error of mean

$$S_{\bar{x}} = \sqrt{\frac{\Sigma \bar{x}^2 - \frac{(\Sigma \bar{x})^2}{n}}{n\ (n-1)}}$$

$$t = \frac{\bar{X}_1 - \bar{X}_2}{\sqrt{\frac{1}{n_1} + \frac{1}{n_2}} \cdot \sqrt{\frac{SS_{x_1} + SS_{x_2}}{n_1 + n_2 - 2}}}$$

where: $$SS_x = \frac{\Sigma \bar{x}^2 - (\Sigma \bar{x})^2}{n}$$