THE EFFECTS OF ETHANOL WITH AND WITHOUT EXERCISE ON THE THERMAL BALANCE OF MAN IN COLD WATER

by

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Abstract

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Experiments involving genuine and placebo ethanol consumption, with and without exercise, were performed by ten healthy young men so that the effects of ethanol in hypothermia might be assessed. A maximal level of hypothermia was induced by immersing subjects in 10 ^{o}C water for a period of 45 minutes, (grand mean tympanic temperature equalled 34.4 ± 0.89 ^{o}C at the termination of cold exposure). Ethanol was given 60 minutes prior to immersions at a standardized dose capable of producing moderately high Blood Alcohol Concentration (BAC), (grand mean BAC equalled 81.6 ± 11.79 mg • 100 ml^{-1} at the time of immersion). During the first 20 minutes of immersions performed with exercise, subjects pedalled a submerged exercise bicycle fitted with an ergometric device adjusted to a one kg load in air, at a rate equal to 0.5 Hz.

The following physiological variables were studied: core cooling as assessed by rectal and tympanic temperatures, metabolic rate, skin temperatures at four sites, heart rate, blood pressure, BAC "decay" rate, and urine total volume and pH.

The following results were found:

1) prior to immersion, ethanol increased rectal cooling.
2) during cold-water immersion, ethanol without exercise reduced overall mean metabolic rate approximately 20%. This statistically significant reduction was of no physiological importance, since no ethanol-induced alterations of core cooling were found either with or without exercise. Exercise, either with or without ethanol however was found to increase tympanic cooling by approximately 75%, during the period

of its performance.

3) the BAC rate of decay was increased during cold exposure. However, this increase probably reflected haemodynamic alterations induced by hypothermia, rather than an actual increase in the rate of ethanol elimination in the cold.

4) core rewarming rates were not found to be significantly altered by either ethanol or exercise.

5) for all physiological variables studied, placebo conditions (three subjects only) did not yield results significantly different from those found under control type treatment.

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"in vino veritas"

Pliny

Introduction

Low molecular weight, monovalent alcohols exhibit several dose-dependant pharmacophysiological effects which modify the internal body temperatures of homeotherms (Harger and Forney, 1963; Majchrowicz, 1975). Ethanol, a systemic anesthetic (Root and Hofman, 1963; Bergersen et al., 1966; Kissin and Beglieter, 1971; Majchrowicz, 1975), acts as a typical central nervous system depressant, producing both a widespread neuronal narcosis and a marked hypnotic effect in the brain (Gradwhol, 1954; Goodman and Gilman, 1965; Goldstein et al., 1968; Kaye, 1970).

High doses of ethanol, producing Blood Alcohol Concentration (BAC) in excess of 500 mg • 100 ml^{-1}, (mgm%) can cause death due to respiratory failure, as a result of narcosis of the medulla (Haggard et al., 1940; Goodman and Gilman, 1965; Kaye, 1970; Kissin and Beglieter, 1971, 1972). BAC approximating 250 mgm% impairs hypothalamic function, thus producing sub-normal internal body temperatures (Goodman and Gilman, 1965; Kissin and Beglieter, 1971). Even in low dose (sufficient to produce BACs in excess of 40 mgm%) the drug has been found to cause cutaneous vasodilation, apparently resulting from narcosis of the constrictor nerves of peripheral blood vessels (Chushny, 1940; Harrison, 1952; Truitt et al., 1956; Kissin and Beglieter, 1972). This vasodilation results in an increased heat loss from the skin surface in cool air, and presumably would also increase the rate of cooling in conditions of more severe cold (Gaddum, 1949; Harrison, 1952; Goodman and Gilman, 1965). The frequent, fatal consequences of disregarding ethanol's ability to impair thermal balance in the cold are well documented. Medical literature abounds with reports of cases in which

persons have passed from a state of alcoholic narcosis into hypothermic narcosis as a result of becoming intoxicated in a cold exposure situation (Gradwhol, 1954; Keatinge, 1969; Strang, 1969; Kissin and Beglieter, 1973; Day and Morgan, 1974).

Similarly, there is little doubt that ethanol intoxication is either directly or indirectly responsible for a large percentage of the fatalities resulting from accidental immersions in water. According to statistics compiled by the United States National Safety Council in 1974, 33% of all "drowning" victims were found (on autopsy) to have BACs equal to or in excess of 100 mgm%. Thus, the clear elicitation of the pharmacophysiological effects of ethanol during cold exposure in general, and cold-water immersion in particular, are of considerable practical importance, as well as of theoretical interest.

Unfortunately, no studies to date have satisfactorily answered the fundamental question: to what extent does the ingestion of alcohol, in dosage sufficient to produce moderately high BAC (eg. 80 mgm%), alter the thermoregulatory processes and rate of entry into hypothermia of man immersed in cold-water? Perhaps the most thorough study on the effects of ethanol in cold exposure is that of Haight and Keatinge (1973) who utilized cold air (ie. 14.5 ^{o}C) as the environmental thermal stressor. These authors discovered that ethanol in low dose (BAC of approximately 40 mgm%) would produce accelerated core-cooling rates under hypothermia-inducing, exposure conditions only if hypoglycemia had been induced as a consequence of exhaustive exercise prior to the actual cold exposure. Since the thermal conductivity of water is many times that of air, a direct extrapolation of the results of these authors to a cold-water, hypothermia situation would seem tenuous. Accor-

ding to Chusny, (1940) a BAC approximating 40 mgm% is usually considered critical for the production of ethanol-induced, peripheral vasodilation. Thus, although BACs would likely be in excess of this critical level at the initiation of Haight and Keatinge's (1973) experiments, metabolic de-toxification would rapidly reduce BACs to levels below 40 mgm% as experiments proceeded. In addition to perhaps using too low an ethanol dose, these authors exercised their subjects to exhaustion which might be considered unusual, at least from the point of view of simulating a typical immersion incident.

A more recent study, that of Martin et al. (1977) found that no changes in core temperatures, skin temperatures, total ventilation rate, or end-tidal pCO_2 resulted when subjects were given ethanol at a dose capable of producing a mean BAC of 90 mgm%, prior to a cold-water immersion. These authors have stated that the immersion period of their study was too short to allow the prediction of ethanol's effects in immersions of longer duration and consequently is only of speculative value in predicting the drug's effects in immersions where a more severe degree of hypothermia would be induced. Additionally , these authors did not measure heat production, thus jeopardizing their general conclusions regarding ethanol's effects on heat balance.

Another study, that of Hobson and Collis (1977) found that a mean BAC of 50 mgm% actually resulted in a retardation of the core cooling rates of three of their four subjects immersed in 7.5 ^{o}C water. Unfortunately, in addition to using a low ethanol dose, their study involved a small sample size (ie. four subjects) making critical statistical assessment of treatment effects difficult. Furthermore, that study neglected to obtain measures of heat production or peripheral heat loss.

Finally, neither of these two latter studies incorporated an exercise condition in their experiments. Thus, these studies ignored the exercise effect, which Haight and Keatinge (1973) had shown was important in the manifestation of ethanol's low-dose effects on thermoregulation.

In addition to direct physiological effects of ethanol in hypothermia, many authors have reported that lessened anxiety and apprehension was evident in their subjects during immersions where ethanol had been given (Keatinge and Evans, 1960; Andersen et al., 1963; Hobson and Collis, 1977; Martin et al., 1977). Such a result suggests that ethanol perhaps acts to alter the psychological mood of subjects, and may therefore produce some alterations in the physiological responses of subjects to hypothermia.

Lastly, no study to date has investigated the effects that ethanol consumption (prior to hypothermia induction) might have on physiological function during the period of rewarming from hypothermia.

The present study was designed therefore to test the following null hypotheses:

1. ; That the consumption of ethanol prior to a hypothermia inducing, cold-water immersion would result in altered core cooling rates during such immersions.

1a. ; That the postulated ethanol-effect of hypothesis 1. would be potentiated in immersions where exercise was performed.

1b. ; That the postulated ethanol-effect of hypothesis 1. would be reflected by modification(s) of either physiological heat production and/or heat loss limiting mechanisms.

2. ; That the consumption of ethanol would result in some direct alteration of the cardio-vascular responses to hypothermia.

3. ; That an alteration of psychological mood, achieved through the performance of placebo ethanol immersions would result in some modification of the typical physiological responses to cold stress.

4. ; That the consumption of ethanol prior to hypothermia induction would result in some alteration of physiological responses during subsequent rewarming from hypothermia.

5. ; That the consumption of ethanol would result in some modification of "urinological" responses to cold stress.

6. ; That BAC decay would not be altered as a consequence of either hypothermia or exercise treatments.

In order to test the above hypotheses, experiments combining the effects of ethanol with and without moderate exercise were performed. Only moderate exercise was performed as a contrast to Haight and Keatinge's (1973) use of an exhaustive exercise regime, so as to simulate a level of exercise most pertinent to that of individuals immersed under accidental conditions.

Immersions were conducted in 10 ^{o}C water for a period of 45 minutes, in order that a maximal safe level of experimental hypothermia might be induced in the subjects. Both heat production, as assessed by O_2 consumption, and heat loss from two core (rectal and tympanic) sites and four peripheral (skin) sites were continually monitored throughout the course of experiments. Intermittent measures of blood pressure, and continual recordings of heart rate were obtained to determine ethanol's effects on peripheral circulation and cardiac function. BAC measures were made at selected times, in order that typical, mean BAC curves might be constructed and studied for possible hypothermia and exercise effects. Urine samples taken at selected times were subjected to simple

urinalysis to elucidate any effects of ethanol and cold stress on urine pH, glucose and protein concentrations, and total urine volume.

In order to test hypothesis 3., a series of placebo experiments were incorporated into the experimental design of this study.

Finally, all recordings were continued throughout the period of rewarming from hypothermia, in order that the drug's effects during that time period might be determined.

Literature Review

The literature regarding the specific physiological effects of both ethanol and hypothermia is exhaustive. Therefore no attempt will be made here to separately review these two topics. Instead a detailed review of the rather meager literature available on ethanol's effects in conjunction with hypothermia will be endeavored.[1]

Keatinge and Evans (1960) initiated the study of ethanol's effects in conjunction with hypothermia in man. These authors found that ethanol consumption 45 minutes prior to a 30 minute immersion in 15 ^{o}C water did not significantly alter the core-cooling rate (as determined by rectal temperatures) of the ten healthy men used in their study. Ethanol was shown to lower metabolic rates of immersees and reduce the usual rise in heart rate seen during an immersion. Additionally, the drug abolished ventricular extrasystoles which were evident in some subjects during control immersions. Ethanol ingestion was found to have no effect in lessening hyperventilation characteristically observed during the first few minutes of immersions. Mean blood flow through the finger tips of subjects was seen to decrease less rapidly during immersions following alcohol ingestion. However, a statistically significant decrease in blood flow at this site could not be established. These authors also reported that subjects felt both warmer and less apprehensive in immersions where alcohol had been consumed. BAC was not measured in the above study. However, by Widmark's method, it was

[1] There exist a number of excellent reviews of both ethanol and hypothermia's general physiological effects. It is suggested that a reader interested in ethanol's general principles should see Kissin and Beglieter (1971, 1972) and if interested in hypothermia, should refer to Keatinge (1969).

calculated that the mean BAC at the time of immersion would have approximated 110 mgm%, (see Appendix 1 for calculation). Keatinge and Evans (1960) concluded that alcohol ingestion was unlikely to accelerate the fall in core temperature and certainly did reduce the degree of discomfort seen in cold water induced hypothermia. Furthermore, these authors speculated that the drug might reduce some of the risks involved in sudden accidental immersion as a result of its apparently beneficial cardiac effects.

Anderson et al., (1963) examined the effects of alcohol ingestion on the thermal balance of sleeping men. In their study, two series of experiments were performed by each of their three subjects. Each subject performed a total of four experiments per series as follows: two control experiments where ethanol was not given, and two experiments where ethanol was given at a dose of either 1.0 ml ethanol • kg body weight^{-1} or 1.5 ml ethanol • kg body weight^{-1}. For both series, identical dietary restrictions were imposed from 12 o'clock of the experimental day. Two standardized light carbohydrate meals were given at four and six pm, and at eight pm subjects were stripped, "wired" and placed under heavy blankets in the experimental bed.

At nine pm, in series I experiments, the subjects were given ethanol at the appropriate dose and allowed to drink over a one hour period, while still covered by the blankets. At ten pm, the blankets were removed and subjects were exposed naked to an air temperature of 20 ^{o}C throughout the following eight hour experimental sleep period. During that cold exposure period, measurements of rectal and eight skin temperatures were made at 30 minute intervals. In addition to the temperature recordings, exhaust gasses collected from a respiratory hood

which subjects wore around their heads, were analysed to determine metabolic rates. Finally, at those times, records of bed movements and assessments of the subject's level of sleep were made.

At nine pm, in series II experiments, the blankets were removed and the subjects were again given ethanol at an appropriate dose, and were instructed to drink over the following hour, while exposed to an environmental air temperature of 15 ^{o}C. With the exception that recordings were taken every 15 rather that every 30 minutes, a data collection procedure identical to that used in series I experiments was then carried out.

For both experimental series, the authors were unable to find any difference in the rate of core-cooling, or any ethanol-induced vaso-dilatory effect as assessed by skin temperatures. Metabolic rate while intoxicated was not found to differ from control levels for any subject of series I experiments. However, a slight non-significant decrease in metabolic rate was observed in one subject given the higher ethanol dose under the greater cold stress of series II experiments. Under all experimental conditions, the consumption of ethanol was found to produce significantly greater levels of comfort, more sleep and less shivering in the subjects. Andersen et al., (1963) concluded that ethanol in moderate dose had no deleterious effects on heat balance during prolonged mild cold exposure. Furthermore, the only significant effect of the drug under the above experimental conditions was the elicitation of a marked hypnotic effect which resulted in greater subject comfort and increased sleep. Once again, BAC determinations were not made in the above study. However, it has been calculated again by Widmark's method, that the mean BAC at ten pm would have approximated 100 mgm%

when subjects were dosed at 1.0 ml ethanol • kg body weight^{-1}, and 155 mgm% when dosed at 1.5 ml ethanol • kg body weight^{-1}, in both experimental series. Eight hours later at the termination of the experiments, these levels would theorectically be 0 mgm% for the low dose and 30 mgm% for the higher dose, (see Appendix 2 for calculations). Although Andersen et al., (1963) considered such BACs as representative of a moderate dose, such levels might actually be considered relatively high. Certainly a BAC of 155 mgm% would be an adequate level to explain both the author's reports of vomiting in all subjects given the high ethanol dose, and the subjects reported complaints of "hang-over" at the termination of the experiments.

MacGregor et al., (1965) studied the effects of hypothermia on the rate of disappearance of ethanol from the arterial circulation of dogs given intravenous (IV) ethanol injections. All dogs used in the study were given an initial IV injection of d-tubocurarine, to induce respiratory paralysis and prevent shivering. The use of curarine necessitated artificial respiration with a positive pressure ventilator, by which ventilation rate was held constant at 20 inspirations • minute^{-1}. Control dogs were maintained in a normothermic condition (38 ^{o}C) while test dogs were cooled to a rectal temperature of 24 ^{o}C by immersion in an ice-water bath. Ethanol, at a dose of either 1.3 g ethanol • kg of body weight^{-1}, or 2.6 g ethanol • kg of body weight^{-1} was infused as a 15% solution in 5% glucose: distilled water, via a cannulated femoral vein, once hypothermia had been established. Blood samples for BAC determinations were drawn every five minutes over a one hour period from a cannulated femoral artery. The study showed that the rate of equilibration of ethanol in blood with total available body water space

was approximately doubled in hypothermic dogs. The rate of decrease of ethanol level in arterial blood was not significantly different between normo and hypothermic dogs and even a four-fold increase in tidal volume did not affect the rate of ethanol disappearance in the hypothermic dog. Finally, although the rate of ethanol clearance was not significantly different in hypothermia, cooled dogs exhibited absolute BAC in excess of those seen in normothermic animals. The authors concluded that the decreased rate of equilibration seen in hypothermia resulted from a general over-all decrease in circulation velocity and level of perfusion present in the peripheral tissues of hypothermic dogs. The observed result of no change in rate of ethanol clearance in hypothermia, in spite of an apparent dimminished availability of nicotinamide diphospho-nucleotide (NAD) the co-factor of alcohol dehydrogenase (ADH) the enzyme primarily responsible for ethanol oxidation in vivo, was shown to be due to an increased hepatic perfusion in hypothermia. That is, in hypothermic dogs an increased level of liver tissue perfusion was able to compensate for a decreased metabolic rate of de-toxification. The actual BAC achieved in experimental animals in the above study were extremely high, for example in hypothermic animals the low dose employed produced BAC in excess of 215 mgm%, while the higher dose resulted in BAC in excess of 410 mgm%, at the initiation of experimentation.

MacGregor et al., (1966) studied the effectiveness of anaesthetics in the reduction of core temperatures at which the onset of ventricular fibrillation began in dogs undergoing lethal ice-water immersions. In their study, dogs were "wired" for electro-cardiographic (ECG) recording and esophageal temperature, in addition to having a cannula

inserted into the right femoral vein. Through the cannula, they received a curarine injection prior to being placed on an artificial respirator. Four sets of experiments, each having six replicates were completed using the following anaesthetics: ether, ethanol, propanol and butanol. Ether was introduced through the respiratory apparatus, while the alcohols were given IV via the femoral cannula. The dogs were then immediately immersed in an ice-water bath, and recordings of esophageal temperature, ECG and blood samples for BAC determinations were taken at 15 minute intervals until the first onset of ventricular fibrillation or cardiac asystole occurred. As in the previous study by these authors (MacGregor et al., 1965) the direct infusion of alcohols resulted in extremely high BACs. For example, at the time of onset of ventricular fibrillation, ethanol BAC approximated 400 mgm%. The authors found that all four anaesthetics significantly increased the dogs' tolerance of hypothermia. Furthermore, the most effective alcohol in this regard was found to be ethanol, which allowed dogs to cool a further four degrees Centigrade prior to any initiation of ventricular fibrillation. These authors also reported that as a consequence of their experiments on dogs, ethanol at a BAC of 400 mgm% eliminated the danger of hypothermic-induced ventricular fibrillation usually prevalent in human cardiac surgery involving hypothermia. Ethanol infusion allowed surgeons to successfully cool two patients to 26 and 24.5 ^{o}C, in order to perform surgery requiring the induction of cardiac arrest.

Miller and Miller, (1967) studied the effects of ethanol and hypothermia on the survival time of newborn and adult guinea pigs subjected to asphyxiation. In ethanol trials, animals were injected intra-peritoneally (IP) with a dose of ethanol equivalent to 0.293 g · 100 g of

body weight^{-1}. Hypothermia was produced through immersion in an ice-water bath. Once the desired level of hypothermia (20 to 23 ^{o}C) had been achieved, animals were removed from the bath and were placed into an asphyxiation chamber through which a gas mixture consisting of 95% nitrogen and 5% carbon dioxide flowed. Subsequently the time of death was then recorded. These authors found that survival time during asphyxiation was increased over untreated controls in both ethanol and hypothermia treated animals. A synergistic effect was found when ethanol and hypothermia treatments were combined, such that the increase in survival time achieved was greater than that expected on the basis of either treatment alone. Hypothermia was observed to prolong survival time over ethanol treatment, and neonates were seen to exhibit a greater viability under all conditions than adults. The authors concluded that the beneficial effects of hypothermia were primarily due to: a reduction in general body metabolism (particularly a reduction in cerebral metabolism), a decreased rate of depletion of cardiac and cerebral glycogen, and a decreased level of blood acidosis. Ethanol was concluded to elicit its benefical effects through: decreased muscular activity, decreased cerebral oxygen uptake, and the production of an increase in cerebral perfusion as a result of vasodilation counteracting vasoconstriction and increased blood viscosity.

Haight and Keatinge, (1973) studied the ability of young men to thermoregulate when exposed to cold air, after a state of functional hypoglycemia had been induced in them by exercise and ethanol pre-treatment. In their study, 14 healthy young men exercised for a two hour period (at an exercise level proportional to 70% of each subjects' maximal oxygen uptake), prior to ingesting 28 ml of absolute ethanol.

After a 40 minute stabilization period had elapsed, subjects were exposed during a 30 minute period to an ambient air temperature of 14.5 °C. Measurements of rectal and esophageal temperatures were made throughout the experiments, along with measures of: oxygen consumption, blood glucose, plasma lactate, glycerol, β-hydroxybutyrate, and free fatty acid levels. In subjects who had received ethanol following exercise, both rectal and esophageal temperatures were seen to fall at a greater rate than that of subjects who had either not exercised, but had been given ethanol, or subjects who had not been given ethanol, but had exercised. Additionally, in ethanol-exercise treated subjects, a progressive and drastic fall in blood glucose levels was observed during the subsequent cold exposure period. Levels of plasma lactate, glycerol, β-hydroxybutyrate, and free fatty acids were seen to have increased over the levels characteristically observed under control conditions, where ethanol was not given, or exercise was not performed. Furthermore, ethanol-exercise treated subjects showed a reduction in metabolic rate and shivering levels when compared to controls. The administration of glucose (60.4 g) to ethanol-exercise treated subjects resulted in a restoration of a typical metabolic response to the cold stress, and also restored core-cooling rates to levels found under control conditions. To reiterate, neither ethanol nor exercise treatment alone was capable of producing a lowered blood glucose level, or any impairment of thermoregulation.

Haight and Keatinge, (1973) concluded that the impaired ability to thermoregulate resulted from ethanol's known ability to stop gluconeogenesis, thus producing an inhibition through a substrate deficiency, of the hypothalamic center responsible for thermoregulation. The role of

exercise in ethanol impairment was concluded to be primarily that of insuring that glucose reserves in the body were exhausted, thus allowing the ethanol-hypoglycemic effect to become critical during the subsequent cold exposure. BAC determinations were not made in the above study, however it has been calculated that the mean BAC achieved in that study would theoretically have approximated 40 mgm%, (see Appendix 3 for calculations). Such a BAC as that above is barely equivalent to the BAC generally considered critical to the production of ethanol-induced vasodilation of cutaneous blood vessels. However, since the cold exposure period began only 40 minutes after the beginning of drinking, the actual BAC would probably have still been increasing during cold exposure.

Hobson and Collis, (1977) reported that a mean BAC of 50 mgm% actually retarded the core cooling rate of three of their four subjects. In their study, two male and two female volunteers were given ethanol and were immersed in a cold-water tank until their rectal temperatures had dropped to 35 ^{o}C. The length of time taken by a subject to reach 35 ^{o}C in the ethanol trial was compared to the average time required for that same subject to reach 35 ^{o}C in five repeated trials without ethanol. These authors have cautioned readers that their results can not be considered as confirmatory evidence of a general retardation of core cooling under ethanol treatment, because of the limitations imposed by the small sample size employed in their study. Nevertheless, the results of that study clearly indicated that ethanol treatment did not result in an accelerated core cooling rate in any of the four subjects.

Martin et al., (1977) in a study involving eight male and five female healthy young subjects found that the ingestion of ethanol had no significant effects on the rate of core cooling or ventilatory responses

during a cold water immersion. In their study, immersions lasted for 20 minutes in 13 ^{o}C water. A 25 minute period was allowed to elapse after the cessation of ethanol consumption prior to the actual initiation of cold-water immersions. During immersions, changes in total expired volume, end-tidal pCO_2 level, ECG, and the rates of cooling at rectal, aural, and five skin sites were recorded under ethanol treatment. Mean BAC was found to equal 90 mgm% immediately prior to the immersion, and at the cessation of the cold exposure period was found to equal 99 mgm%. The authors suggested that the increase in mean BAC observed during the immersion period may in fact have been an artificial increase resulting from the utilization of subjects having widely different rates of ethanol absorption and/or elimination. These authors also reported that subjects felt less "uncomfortable" during immersions where ethanol had been given. Martin et al., (1977) concluded that ethanol ingestion did not increase body heat loss, or significantly alter ventilatory dynamics during mild cold-water-induced hypothermia.

Methods

I. Experimental Subjects

A call for male volunteers resulted in a small pool of potential experimental subjects from which it was possible to select a sub-group consisting of ten individuals who displayed a high level of conformity in regards to certain selection criteria. These selection criteria were as follows; age, 20 to 25 years ; weight, 68.0 to 75.0 kg ; average weekly alcoholic beverage consumption, not to exceed one liter of beer or wine. Additionally, all subjects used in this study were considered to be physically fit, on the basis of their past medical histories and their ability to pass a standardized cardio-pulmonary stress test. Some physical characteristics of the subjects are presented in Table I.

II. Immersion Scheduling

The main experimental subject group was segregated into three smaller sub-groups for the purposes of scheduling immersions. Since only one immersion per day was possible and a total of 60 immersions were originally planned, segregation into sub-groups was necessary so that each volunteer might perform his required number of immersions in the minimum time possible. A master schedule of immersions was consequently drawn up and is presented along with the actual schedule followed in Appendix 4.

III. General Experimental Design

As originally planned, all subjects were to perform a total of six immersions each. The experimental conditions of these immersions differed only in either the type of experimental drink consumed by the subjects prior to the immersion, and/or the level of exercise to be per-

Table 1. Physical characteristics of subjects used in study.

Subject	Age, years	Weight, kg	Height, cm	Skinfold thickness, mm at following skin sites, abdomen	left thigh	left biceps	sub-scapular
SC	20.0	70.0	185.5	7.2	9.1	8.0	8.2
JS	21.0	72.2	189.0	5.5	4.2	8.1	8.4
DP	20.0	71.8	172.7	6.6	5.0	7.2	11.5
HD	19.0	68.0	178.4	8.8	7.7	7.9	8.4
GP	22.0	68.5	175.3	17.3	13.4	14.3	17.6
TM	21.0	74.9	172.7	17.0	10.9	13.9	14.0
HW	21.0	73.2	181.0	8.9	6.0	6.5	10.4
JM	21.0	67.7	172.7	12.9	9.1	7.8	11.5
PC	25.0	74.7	173.0	13.8	7.4	7.7	11.4
RG	22.0	74.5	193.0	8.5	6.0	5.5	8.6
Mean	21.2	71.6	179.4	10.6	7.9	8.6	11.0
sd	1.62	2.85	7.55	4.26	2.84	2.77	3.00
Mean Range							
(mean + sd)	19.6	68.8	171.9	6.35	5.01	5.82	7.98
(mean - sd)	22.8	74.4	187.0	14.91	10.61	11.36	13.98

sd = standard deviation of the mean.

formed during the immersion. The six resulting immersion conditions were as follows:

A) Control, non-exercise immersion (control) - subject consumed the control type drink, was informed that it contained no ethanol and was instructed to not perform any voluntary exercise while immersed.

B) Control, exercise immersion (ex) - subject consumed the control type drink, was informed that it contained no ethanol and was instructed to perform a standardized level of voluntary exercise during the first 20 minutes of the immersion.

C) Alcohol, non-exercise immersion (al) - subject consumed the alcohol type drink, was informed that it contained ethanol and was instructed to not perform any voluntary exercise while immersed.

D) Alcohol, exercise immersion (alex) - subject consumed the alcohol type drink, was informed that it contained ethanol and was instructed to perform a standardized level of exercise during the first 20 minutes of the immersion.

E) Placebo, non-exercise immersion (pl) - subject consumed the placebo type drink, was informed that it contained ethanol and was instructed to not perform any voluntary exercise while immersed.

E) Placebo, exercise immersion (plex) - subject consumed the placebo type drink, was informed that it contained ethanol and was instructed to perform a standardized level of voluntary exercise during the first 20 minutes of immersion.

Placebo type immersions were performed only by the first sub-group cycled through the immersion schedule. Preliminary analysis of placebo core temperature curves indicated that placebo treatment did not alter cooling rate from that observed under control conditions. In view of

this result, and since all immersions involved some degree of risk and discomfort to the volunteers, all further placebo experiments were abandoned.

IV. Composition of Experimental Drinks

The composition of the three types of experimental drinks given to the subjects prior to the immersions were as follows;

A) Control type drink - distilled water, a volume corresponding to a dose of 1.15 ml · kg body weight^{-1} ; unsweetened orange juice, a volume equivalent to three times that of the volume of distilled water used ; glucose, 29.5 g dissolved in the drink.

B) Alcohol type drink - 95% ethanol : distilled water (v/v), a volume corresponding to a dose of 1.15 ml · kg body weight^{-1} ; unsweetened orange juice, a volume equivalent to three times that of the volume of 95% ethanol used ; 1.0 ml of artificial brandy extract.[1]

C) Placebo type drink - distilled water, a volume corresponding to a dose of 1.15 ml · kg body weight^{-1} ; unsweetened orange juice, a volume equivalent to three times that of the volume of distilled water used ; 1.0 ml artificial brandy extract ; glucose, 29.5 g dissolved in the drink.

All drinks were presented in a glass around which a cotton cloth had been wrapped. For the alcohol and placebo type drinks, this cloth was soaked in 95% ethanol; for the control drink, it was soaked in distilled water. This procedure was followed because according to Hobson, (1977) a subject must be able to at least smell alcohol if he is to be convinced that a placebo drink actually contains ethanol.[2]

1 The artificial brandy extract originally contained 40% (v/v) ethanol. This ethanol was boiled off, and the original volume of the extract was subsequently made up with distilled water.

Glucose was given in non-alcohol type drinks in an effort to provide caloric compensation for energy liberated as a result of ethanol oxidation during the immersion period of experiments. Appendix 5 presents the calculations involved in arriving at a standard figure of 29.5 gm of glucose.

V. Procedures Common to All Types of Immersions

The following is a brief out-line of procedures and conditions which were common to all types of immersions:

A) subjects were given explicit instructions regarding; diet, exercise, alcohol consumption and amount of sleep, to be followed on both the day prior to and the day of immersion. Appendix 6 presents a copy of these instructions and restrictions.

B) subjects arrived at the laboratory in a four hour post-absorptive condition.

C) on arrival, subjects produced a urine sample and then changed into a bathing suit.

D) subjects were then weighed and skinfold thicknesses at the following sites were taken; the lower abdomen (five cm below and lateral to the umbilicus), the left thigh (at the midpoint over the vastus externus muscle), sub-scapular (above the lower point of the scapula), and the left triceps (over the midpoint of the muscle).

E) subjects then retired to privacy and inserted a rectal thermocouple to a point 15 cm beyond the anus.

[2] Although ethanol can be absorbed from the respiratory tract, (Nicloux, 1900; Grehant, 1907; Lester et al., 1951) the actual amount of ethanol actually absorbed by this route is usually small. In this study, placebo treated subjects did not show BACs which were significantly differed from a BAC of 0 mgm%.

F) subjects then had ECG electrodes attached to the chest, and had insulated thermistors attached to the following skin sites; the sternum, (at the midpoint between the articulations of the fourth and fifth ribs) the left thigh, (over the midpoint of the vastus externus muscle) the left biceps, (over the midpoint of the muscle) and at a lumbar site, (two centimeters lateral to the articulation between the third and fourth lumbar vertebras).

G) a small thermocouple would then be placed in the right aural canal, and was adjusted by the subject until it lay in close apposition with the tympanic membrane, after which it was sealed in place with a wax plug.

H) the subject would then don a close-fitting face mask, connected by air lines to a continuous O_2 analyser. A pump insured that room air and the subject's expired breath were continually drawn from the face mask. A smaller pump drew off samples of air and breath which were then passed to the O_2 analyser.

I) all experiments followed an identical time course. A digital clock was started after all apparatus had been connected and was found to be operational. Recordings of heart rate and tympanic temperature were made continuously from this zero time point to the final termination of an experiment, some 180 minutes later. All other measurements were made at the times indicated in Table II, which presents a master chart of procedures common to all types of experiments.

VI. Standardization of Environmental Temperatures

All immersions took place in an especially constructed cold-water immersion tank, (Johnson et al., 1977). During immersions, subject either

Table II. Master chart of experimental procedures common to all types of immersions.

Experimental period	Subject activity during period	Time, minutes	Measurements recorded, Temp.	O_2 con.	B.P.	BAC	Urine
Stabilization	Subject sits	0.0	x	x		x	x
	quietly in the	5.0	x	x	x		
	laboratory	10.0	x	x			
		20.0	x	x			
Drinking	Subject removes	25.0	x				
	O_2 con face mask	30.0	x		x		
	and consumes the	35.0	x				
	appropriate drink	40.0	x				
Absorption	Subject finishes	45.0	x	x			
	drink and dons	55.0	x	x		x	
	O_2 con face mask	65.0	x	x			
		75.0	x	x			
		80.0	x	x	x		
		85.0	x	x		x	x
Immersion	Subject dons a	90.0	x	x			
	weight belt and	92.5	x	x			
	proceeds to the	95.0	x	x			
	cold water tank.	100.0	x	x			
	Once immersed the	105.0	x	x			
	subject follows	110.0	x	x			
	instructions re:	115.0	x	x		x	
	exercise to be	120.0	x	x			
	performed while	125.0	x	x			
	immersed.	130.0	x	x			
Rewarming	Subject exits the	135.0	x	x			
	cold water tank	140.0	x	x		x	
	proceeds to the	142.5	x	x			
	rewarm bath.	145.0	x	x			
		147.5	x	x			
		150.0	x	x			
		155.0	x	x	x		
		160.0	x	x		x	x
		165.0	x	x			
		170.0	x	x			
		175.0	x	x	x		
		180.0	x	x		x	

Temp = Tympanic, rectal, and four skin temperatures (^{o}C)
O_2 con. = Oxygen consumption (ml $minute^{-1}$)
B.P. = Blood Pressure
BAC = Blood Alcohol Concentration (as determined by breathalizer)
Urine = Urine sample for; total volume, pH, glucose and protein concentrations)

sat or exercised on a submerged exercise bicycle. Mean environmental temperatures during the experiment were as follows:

A) room air temperature - 22.6 ± 0.45 ^{o}C, throughout the pre-immersion period

B) cold water temperature - 10.0 ± 0.09 ^{o}C, at the initiation of immersions

C) rewarm bath temperatures - 25.6 ± 2.39 ^{o}C, at the initiation of rewarming, and increased during rewarming at a rate of 1.5 ^{o}C · minute^{-1} until a final temperature of 39.9 ± 0.55 ^{o}C had been reached.

Minor fluctuations in the above mean environmental temperatures were observed under the different experimental treatments. These fluctuations were found to be non-significant, and under all experimental treatments, mean environmental temperatures were found to be statistically equivalent, throughout the study. Appendix 7 presents a detailed analysis of mean environmental temperatures.

VII. Standardization of Posture and Exercise Level of Immersed Subjects

Upon entry into the cold water, subjects assumed a posture while sitting on the submerged bicycle, such that only their head and shoulders remained above water level. Throughout the immersions, subjects were closely monitored to insure that cold water could freely circulate around all body surfaces. Subjects were instructed to make no conscious effort to modify in any way their rate of shivering while immersed.

Additionally, every effort was made to insure that subjects performed a standardized level of activity while immersed. Prior to being submerged, the exercise bicycle's ergometer had been adjusted to a one kg load in air, and in immersions where subjects were to exercise, they

pedalled the bicycle at a rate of 0.5 Hz during the first 20 minutes of immersions. Occasionally, while under the influence of ethanol, subjects departed slightly from the above exercise regime. However with the exception of one subject who displayed a behavioral modification while intoxicated which was excessively deviant from that seen in his peers, the level of voluntary activity on the part of subjects undergoing ethanol trials did not significantly deviate from that seen in control experiments. Appendix 8 lists some observations made on the behaviour of the aforementioned deviant subject.

VIII. Blood Pressure Measurements

Measurements of blood pressure were made at the intervals indicated in Table II, throughout all experiments. Determinations were performed indirectly through the use of a standard sphygmomannometer. Sphygmomannometric determination was attempted on subjects while they were immersed in the cold water. Unfortunately, excessive shivering by the subjects made such determinations impossible and further attempts to obtain readings during the immersions were abandoned.

IX. Urine Sampling and Analysis

Each of the three urine samples obtained at the times indicated in Table II were analysed as follows: the total volume of urine excreted was recorded and a small sample of that volume was analysed by a simple "dip and read Combistix Strip" method, to determine the pH and the glucose and protein concentrations.

X. Determination of Correct Ethanol Dose to Achieve a BAC of 80 mgm%

From Widmark's method, it was determined that a dose corresponding to 0.90 ml of 95% ethanol : distilled water (v/v) $\cdot$ kg body weight^{-1}

should produce a BAC approximating 80 mgm%. Subsequent experiments using the experimentors as experimental subjects showed that the above dose was insufficient to produce consistantly the desired BAC. Further experimentation showed that a dose between 1.10 and 1.20 ml of 95% ethanol : distilled water (v/v) • kg body weight^{-1} would produce a BAC of about 80 mgm%, 40 minutes post ingestion. It was decided, therefore that all subjects would receive a standardized dose equivalent to 1.15 ml of 95% ethanol : distilled water (v/v) • kg body weight^{-1} throughout the study. Appendix 9 presents the calculations used in the determination of the original 0.90 ml dose.

XI. Method of Obtaining Breath Samples for Breathalizer Analysis

At the times indicated in Table II, genuine BAC determinations were made via a breathalizer in ethanol trials. Under placebo treatment, fake breathalizer readings were taken at the aforementioned times. Under control conditions, a single BAC determination was made prior to the cold water immersion.

The breathalizer was calibrated prior to and half-way through the study by officers of the Sannich Police Department, who routinely utilize an indentical unit. The breathalizer was operated in accordance with the manufacturer's instructions and only recommended certified reagents were used in BAC determinations.

Subjects were instructed in how to give breath samples prior to the commencement of the study. The procedure was as follows. The breathalizer was placed centrally in the laboratory and was not moved during the course of the study. Subjects exhaled through a length of surgical tubing. At the end of an expiration, the subject would signal to an experimentor stationed at the other end of the tubing, and both

the experimentor and the subject then trapped the breath in the tubing by simply placing their thumbs over the ends of the tubing. The experimentor next attached his end of the tubing to the intake port of the breathalizer. The subject exhaled once more into the tubing and his breath would then cycle through the tubing and into the sample chamber of the instrument. As the subject continued to exhale, surplus breath was vented out of the instrument's exhaust port. Just prior to the termination of his expiration, the subject would again signal to the experimentor who would then close the intake port of the instrument, thus trapping the last portion of the subject's exhaled breath in the sample chamber. In this manner, it was possible to sample a subject's breath for BAC determinations both while the subject was sitting prior to the immersion, and while he was actually submerged in the cold or rewarm tanks, without the necessity of either bringing the subject to the instrument or the instrument to the subject.

XII. Statistical Analysis

Access to the APL, Fortran, and Focal computer libraries of the University of Victoria and the Royal Jubilee Hospital, allowed the bulk of the required statistical analyses of this study to be completed by computerised methods.[3]

[3] The ISTAT (APL), and STAT (Focal) statistical packages were extensively utilized in the performation of the analyses.

Results

I. BAC

As expected, initial BACs of subjects (determined prior to immersions) in both control and placebo experiments were not found to vary from levels approximating 0.0 mgm%.

Table III presents the mean BAC and standard deviation of the mean found for each of the six times during which BAC determinations were made in al and alex trials.

From Table III, it can be seen that mean BACs achieved under alex conditions were consistantly lower than the mean levels found at identical times in al experiments. In an effort to determine if a treatment-induced difference existed between mean BACs taken at identical times, a series of two-factor anovas were performed for each of the time isolates of Table III. The results of this analysis appear in Table IV.

Table IV shows that a statistically significant difference between BAC due to treatment effects was only demonstrable for times 115 and 140 minutes. However, Table IV also indicates that at these times, significant differences in regards to BAC existed between subjects (people). Such a result suggests that BAC was subject to modification by inter-personal variation. Thus the inability to show an overall lower mean BAC under alex treatment, at all times during experiments may have in part been due to an inherent "masking" effect. That is, a high level of inter-personal variation inherent in the BAC data may have made a disproportionate contribution to the overall variance estimate of mean BAC at times other than 115 and 140 minutes.

Figure 1 presents the data of Table III in graphical form. From

Table III. Mean BAC and the standard deviations for the six times at which BAC determinations were made in al and alex trials

Time isolate, minutes	al, Mean BAC, mgm%	al, St. dev. mgm%	alex, Mean BAC, mgm%	alex, St. dev. mgm%
55	93.1	16.29	82.9	16.56
85	84.8	11.67	78.4	11.91
115	69.1	13.02	58.3	7.77
140	55.1	13.65	46.4	7.62
160	54.3	12.00	50.7	6.16
180	54.1	13.69	49.8	8.01

St. dev. = standard deviation of the mean

n = 10 for all times and both treatments

Table IV. Results of two-factor anova series performed on BAC for the time isolates of Table III.

Time isolate, minutes	Source of variation	f value	Probability value	Sig.
55	treatments	1.632	0.237	ns
	people	0.832	0.599	ns
85	treatments	3.277	0.108	**
	people	3.958	0.034	*
115	treatments	12.842	0.007	**
	people	4.594	0.023	*
140	treatments	9.806	0.014	*
	people	6.090	0.010	**
160	treatments	1.127	0.319	ns
	people	2.495	0.109	ns
180	treatments	1.027	0.340	ns
	people	2.058	0.164	ns

Sig. = significance

ns = not significant at 0.05% level ($P > 0.05$)

* = significant at 0.05% level ($P < 0.05$)

** = significant at 0.01% level ($P < 0.01$)

Figure 1. Experimental mean BAC curves for al and alex type experiments. (immer. = immersion)

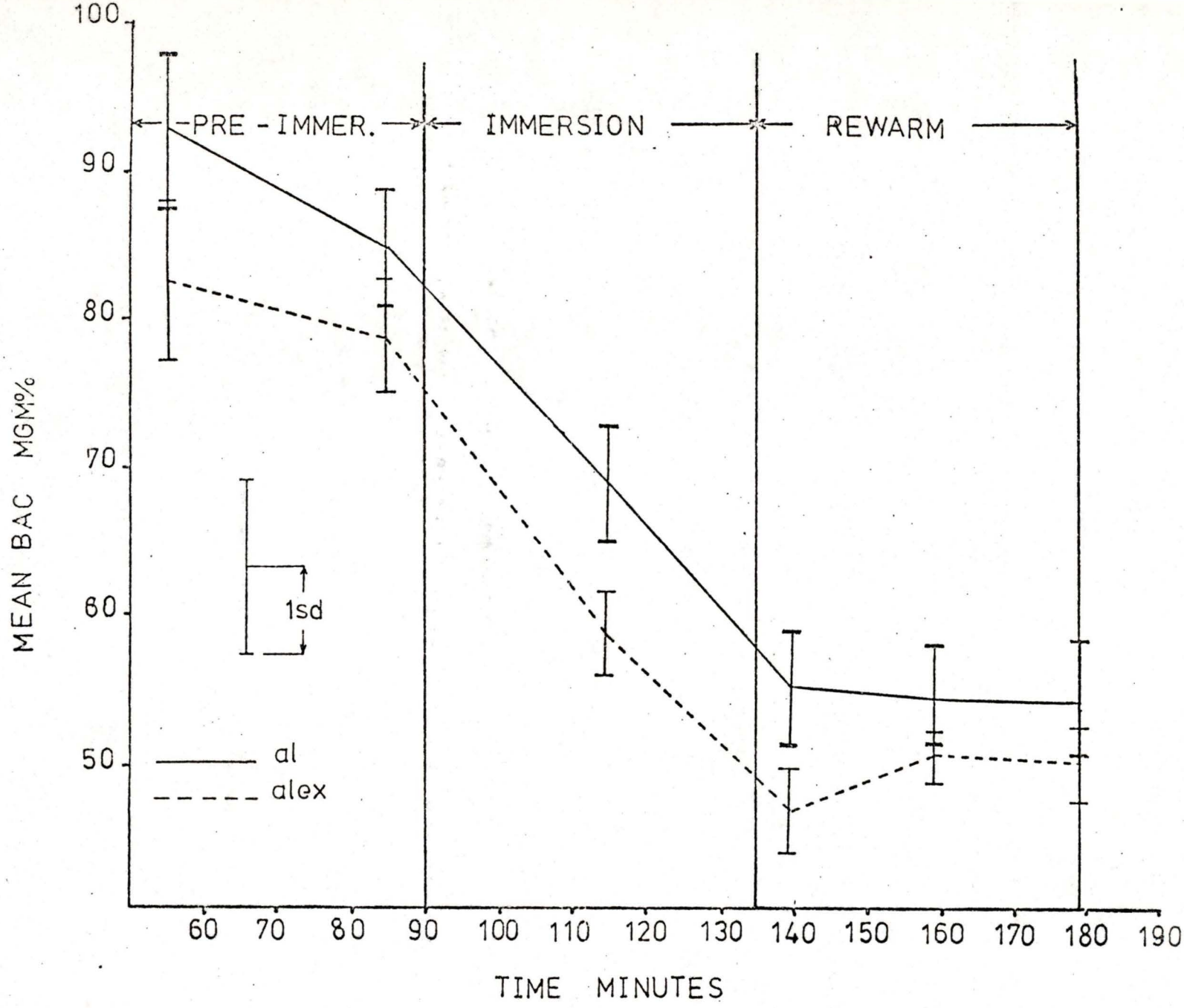

100
90
80
70
60
50
MEAN BAC MGM%
PRE - IMMER.
IMMERSION
REWARM
1sd
al
alex
60 70 80 90 100 110 120 130 140 150 160 170 180 190
TIME MINUTES

Figure 1, it is clear that mean BAC under both al and alex treatments followed similar courses. Both curves show that a "peak" BAC occurred at 55 minutes, indicating that ethanol absorption from the gut had ceased by this time. Since ethanol absorption had ceased, the BAC curves of Figure 1 are in fact actually BAC "decay" or oxidation curves. Figure 1 also shows that for both treatments, the overall BAC decay curve may be divided into three portions, each of which display differing slopes, and presumably, therefore, differing rates of ethanol oxidation. These three curve portions are seen to coincide with major alterations of experimental procedures common to both treatments. For example, BAC decay increased during the experimental immersion period as compared to the decay rate established during the pre-immersion period. Furthermore, both curves exhibit drastically altered slopes for BAC decay during the rewarming periods of experiments. Observations such as these are extremely unusual for BAC decay curves. Usually, BAC decay curves follow a constant linear decay equivalent to 16 mgm% $\cdot$ $hour^{-1}$, throughout the post-absorptive period, (Kalant, 1971). Utilizing the aforementioned standard decay rate, theoretically expected BAC decay curves were calculated for al and alex treatments, and are presented along with the observed experimental curves in Figure 2.

From Figure 2, it is obvious that for both treatments, the expected BAC decay curves differ markedly from the curves seen experimentally. In view of the information shown in Figures 1 and 2, experimental BAC decay rates were calculated. The results of these calculations appear in Table V.

The data of Table V were analysed by t-tests to determine if significant differences existed between the treatments decay rates. The results

Figure 2. Experimental and expected mean BAC curves for al and alex experiments. (act = actual, ie. experimentally determined, exp = expected, ie. calculated, immer. = immersion)

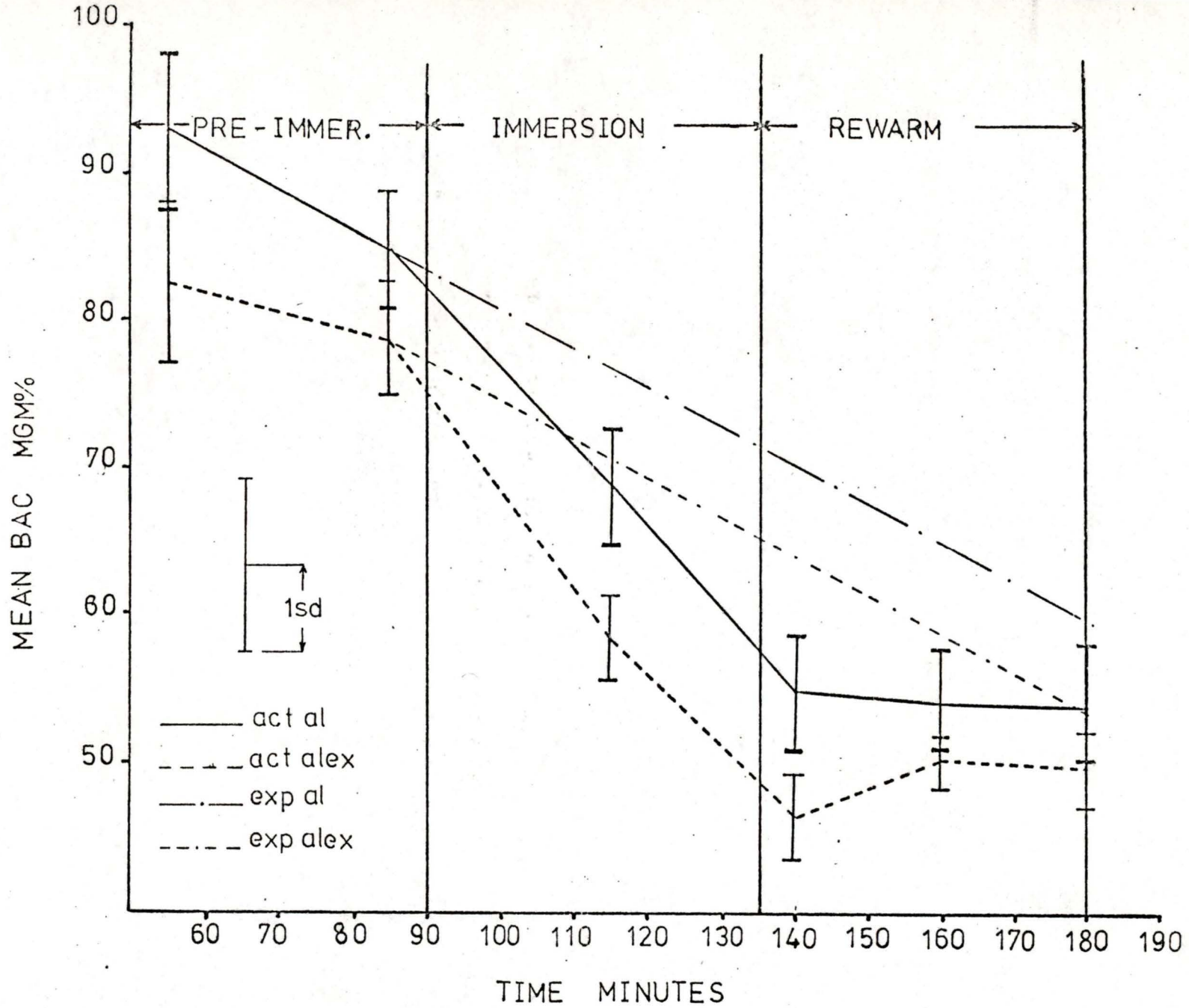

PRE-IMMER.
IMMERSION
REWARM
MEAN BAC MGM%
100
90
80
70
60
50
1sd
act al
act alex
exp al
exp alex
60 70 80 90 100 110 120 130 140 150 160 170 180 190
TIME MINUTES

Table V. Experimental BAC decay rates found under al and alex treatments, for various time periods (portions) of the experimental BAC curves of Fig. 1

Time period, minutes	al decay rate, mgm%·hour^{-1}	alex decay rate, mgm%·hour^{-1}
55 - 85	16.4 ± 12.60	8.7 ± 10.99
85 - 115	31.4 ± 11.05	40.4 ± 15.63
85 - 140	32.6 ± 8.93	35.1 ± 11.61
115 - 140	32.8 ± 15.78	28.8 ± 8.41
140 - 180	0.2 ± 24.38 †	10.7 ± 25.03 †

all rates given as mean ± 1 standard deviation of the mean

† actually rate of BAC increment not decay

of these t-tests are shown in Table VI.

The data of Table VI serve to confirm several of the observations made from the examination of the BAC decay curves shown in Figures 1 and 2. For example, it can be seen from Table VI that during identical time periods, no significant differences in mean BAC decay rates were found between the two treatments. Additionally, it is evident that a highly significant difference exists between the mean decay rates seen during the cold exposure portion of curves, and those of the rewarm portion. Experimental decay rates of 35.1 mgm% $\cdot$ hour^{-1} found in alex trials during cold exposure, and 32.6 mgm% $\cdot$ hour^{-1} found under al conditions during this time period, represent increases in decay rate proportional to 119 and 103% respectively over the expected standard decay rate of 16 mgm% $\cdot$ hour^{-1}. On the basis of typical BAC decay curves, it would be expected that BAC would continue to decrease in subjects undergoing rewarming. However, Table VI confirms that during rewarming, BAC decay curves actually appear to change direction, to produce a net BAC increase equivalent to 10.7 mgm% $\cdot$ hour^{-1} and 0.2 mgm% $\cdot$ hour^{-1}, respectively, under alex and al treatments. Finally, it is interesting to note that although in both treatments the final BACs seen at 180 minutes differed from expected mean BACs at that time, these differences were not significant. Thus, if BAC determinations had not been made throughout the cold exposure and rewarm periods, but had simply been made at times 85 and 180 minutes, one might erroneously conclude that the expected standard decay rate had been followed throughout the course of the experiments.

Table VI. Results of t-tests performed to determine if; a) mean BAC decay rates differed between al and alex treatments at various identical time periods, and b) mean BAC decay rates were equivalent during the cold-water immersion and rewarming periods for each treatment

Time period, minutes	Test conditions	df	t's	Significance
55 - 85	al versus alex	18	-1.456	ns
85 - 115	al versus alex	18	-1.490	ns
85 - 140	al versus alex	18	-0.531	ns
115 - 140	al versus alex	18	0.700	ns
140 - 180	al versus alex	18	-0.950	ns
85 - 180	al; cold-water immersion rate versus rewarming rate	18	3.861	***
85 - 180	alex; cold-water immersion rate versus rewarming rate	18	2.858	**

df = degrees of freedom

ns = not significant at 0.05% level ($P > 0.05$)

** = significant at 0.01% level ($P < 0.01$)

*** = significant at 0.001% level ($P < 0.001$)

II. Core Temperatures

Prior to reporting the results of the analyses of core (tympanic and rectal) temperatures, some preliminary discussion regarding the number of subjects used in the analyses is required.

Reference to Table I of the methods section shows that although several subjects displayed skinfold thicknesses at one or two skin sites which lay outside the overall established mean range of the group, only subjects GP and TM exhibited skinfold thicknesses which appeared to lie consistently outside the group's mean range at all sites. According to Keatinge (1969), skinfold thickness is considered to be an accurate measurement of subcutaneous fat. It has long been known that subcutaneous fat acts as an insulative barrier to surface heat loss in men undergoing cold water immersion. Therefore, greater levels of subcutaneous fat result in a slower rate of entry into hypothermia (Keatinge,1960; Cannon and Keatinge, 1960; Carlson et al., 1959; Hayward et al., 1975). If subjects GP and TM did indeed exhibit greater skinfold thicknesses, they would be expected to cool at a slower rate than their peers in the study.

In view of the above, it was considered critical to determine if subjects GP and TM did in fact have significantly greater skinfold thicknesses than their peers. In order to determine this, the main subject group was segregated into two sub-groups. Sub-group I consisted solely of subjects GP and TM, while the remaining subjects made up sub-group II. For both sub-groups, new means and standard deviations were calculated for body weight and the four skinfold thicknesses and are presented in Table VII.

Table VII. Means and standard deviations of the mean for, body weight and skinfold thicknesses at four skin sites calculated for subject subgroups I and II.

	Subgroup I, subjects GP and TM			Subgroup II, all remaining subjects		
Metric	Mean	sd	Mean range	Mean	sd	Mean range
Body weight, kg	71.7	4.49	67.2 - 76.2	71.5	2.72	68.8 - 74.2
Skinfold thickness, mm at;						
abdomen	17.1	0.18	17.0 - 17.3	9.0	2.91	6.1 - 11.9
lt. thigh	12.1	2.33	9.7 - 14.4	6.8	1.81	5.0 - 8.6
lt. triceps	13.6	0.92	12.7 - 14.5	7.3	0.89	6.5 - 8.2
sub-scapular	15.8	2.58	13.2 - 18.4	9.8	1.54	8.2 - 11.3

sd = standard deviation of the mean

mean range = mean ± standard deviation of the mean

lt. = left

The data of Table VII were analysed by single-factor anovas to determine if the apparent differences in skinfold thicknesses seen between sub-groups were significant. The results of these anovas are shown in Table VIII.

From Table VIII, it can be seen that although both sub-groups exhibited equivalent body weights, sub-group I had significantly higher skinfold thicknesses at all four skin sites compared to those of sub-group II. Subjects GP and TM were found to have an overall mean skinfold thickness of 14.6 mm, while their peers were found to have an overall mean skinfold thickness of 8.2 mm. According to Keatinge (1960), two groups of subjects displaying such differences in overall mean skinfold thicknesses would be expected to demonstrate a difference in mean rectal temperature equivalent to 0.6 ^{o}C at the time of exit from an immersion lasting 30 minutes in 15 ^{o}C water. Since immersions in this study lasted 45 minutes in 10 ^{o}C water, it would seem appropriate to double Keatinge's (1960) estimate of mean rectal temperature differences for the above two sub-groups of subjects. It would be expected therefore, that subjects GP and TM would exhibit mean rectal temperatures some 1.2 ^{o}C higher than that of their peers, at the time of exit from the cold water tank. Experimentally, these subjects were found to exhibit mean rectal temperatures which were 1.4 ^{o}C higher. This observed result is in close agreement with that expected on the basis of Keatinge's (1960) modified estimate.

The slower cooling rate evident for subjects GP and TM as a result of their increased levels of subcutaneous fat might act to raise the level of inter-personal variability inherent in the analyses of experimental samples in which these subjects were included. Furthermore,

Table VIII. Results of anovas performed to determine significance of apparent differences observed between subject subgroups I and II in the data of Table VII.

	Anova values, Subgroup I versus Subgroup II		
Metric	f value	Probability value	Significance
Body weight, kg	0.006	0.939	ns
Skinfold thickness, mm at;			
abdomen	14.205	0.006	**
lt. thigh	12.420	0.008	**
lt. triceps	78.145	0.000	***
sub-scapular	19.750	0.002	**

ns = not significant at 0.05% level ($P > 0.05$)

** = significant at 0.01% level ($P < 0.01$)

*** = significant at 0.001% level ($P < 0.001$)

any such increase in overall inter-personal variability might be expected to result in the "masking" of significant treatment effects on core cooling. For these reasons, it was decided that the analysis of core temperatures would be completed for two differing sample sizes as follows;

A) Tympanic temperature sample I (Ttym I) - sample size equalled ten subjects (subjects GP and TM were included in the sample)

B) Rectal temperature sample I (Trec I) - sample size equalled ten subjects (subjects GP and TM were included in the sample)

C) Tympanic temperature sample II (Ttym II) - sample size equalled eight subjects (subjects GP and TM were excluded from the sample)

D) Rectal temperature sample II (Trec II) - sample size equalled eight subjects (subjects GP and TM were excluded from the sample)

In addition to the unsuitability of subjects GP and TM as experimental subjects, subject PC was found to exhibit a deviant behavioural response to intoxication. This subject had considerable difficulty in performing the prerequisite level of exercise of alex trials. In view of this it was decided that this subject would also be excluded from the experimental sample, along with subjects GP and TM for the purpose of completing a third set of analyses on core temperatures. Thus in addition to the samples presented above, two additional samples, Ttym III and Trec III (each comprising seven subjects), were used in the analyses of core temperatures.

A. Ttym I analysis

Figure 3 presents a composite graph of mean tympanic temperatures observed at ten minute intervals under control, al, ex and alex treatments for the Ttym I sample.[1] For purposes of performing a preliminary analysis on the "raw score" data of Ttym I, only selected times of Figure 3 were used. These selected times coincided with some alteration of experimental procedures or conditions. Table IX lists these selected times and explains the alterations of experimental conditions which were initiated with them.

Figure 4 presents a composite graph of mean tympanic temperatures observed at the selected times of Table IX during control, al, ex and alex experiments for the Ttym I sample.[2]

Even a cursory examination of the original single treatment mean tympanic temperature graphs used to construct the composite graph of Figure 4, shows that all four treatments produced wide and often overlapping variances during the times selected for analysis. To determine

1 For Figure 3 and for all remaining figures dealing with composite graphs presented in the text proper of this thesis, no standard deviations of the means have been plotted. It was necessary to omit these variance estimates from composite graphs, since there was no feasible method available by which it would have been possible to indicate which standard deviation belonged to which individual treatment in them. However, for every composite graph cited in the text proper, the original single treatment graphs used to construct the composite graph in question will be presented in an appendix. Thus readers who wish to achieve some impression of the variances involved for the individual curves of composite graphs, will be instructed by a footnote as to which appendix they should search. For example, Appendix 10 presents the original single treatment graphs with their corresponding standard deviations plotted, which were used to produce the composite graph of Figure 3.

2 Appendix 11 presents the single treatment graphs used to construct the composite graph of Figure 4.

Figure 3. Composite graph of mean tympanic temperatures observed at ten minute intervals under control, al, ex and alex treatments for the Ttym I sample.

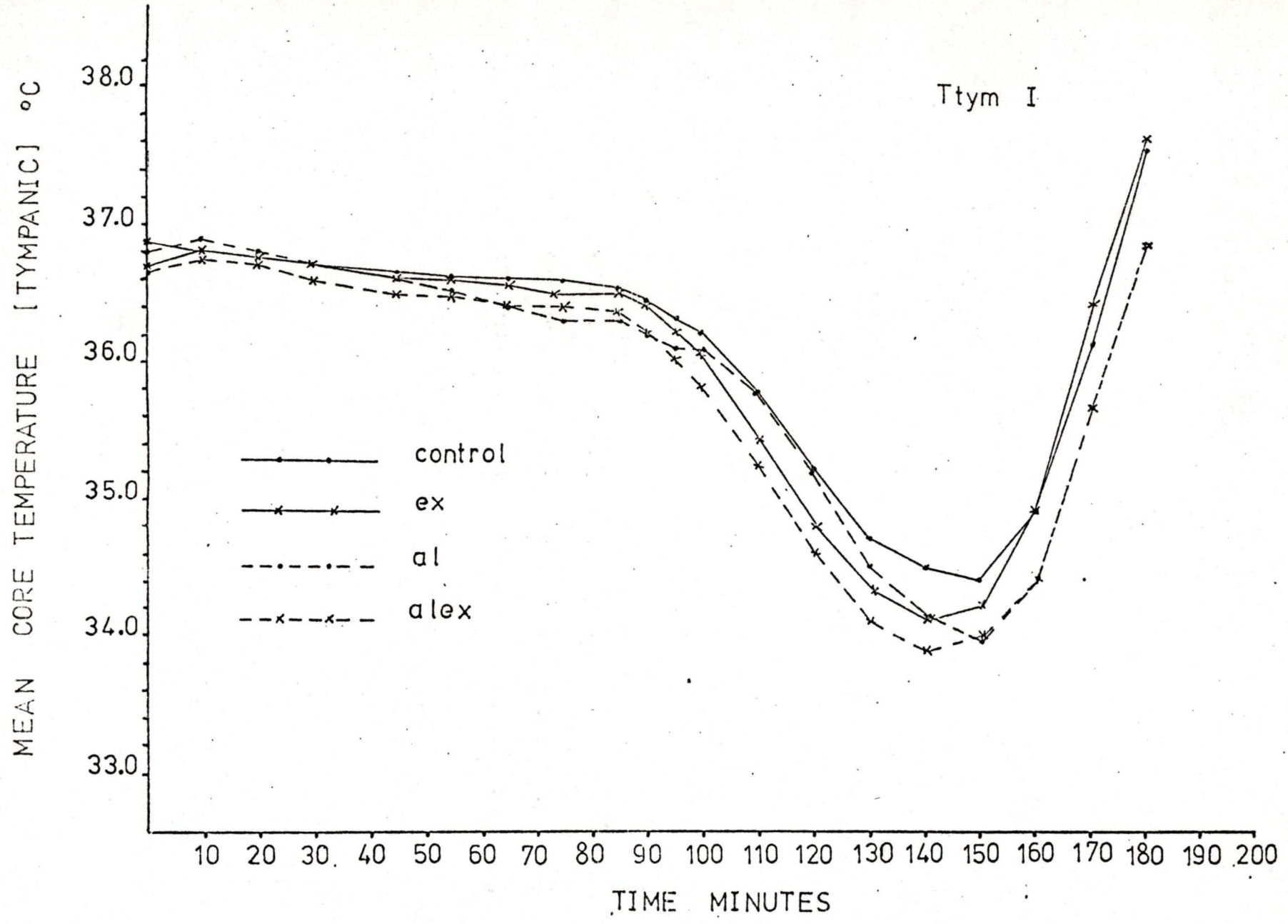
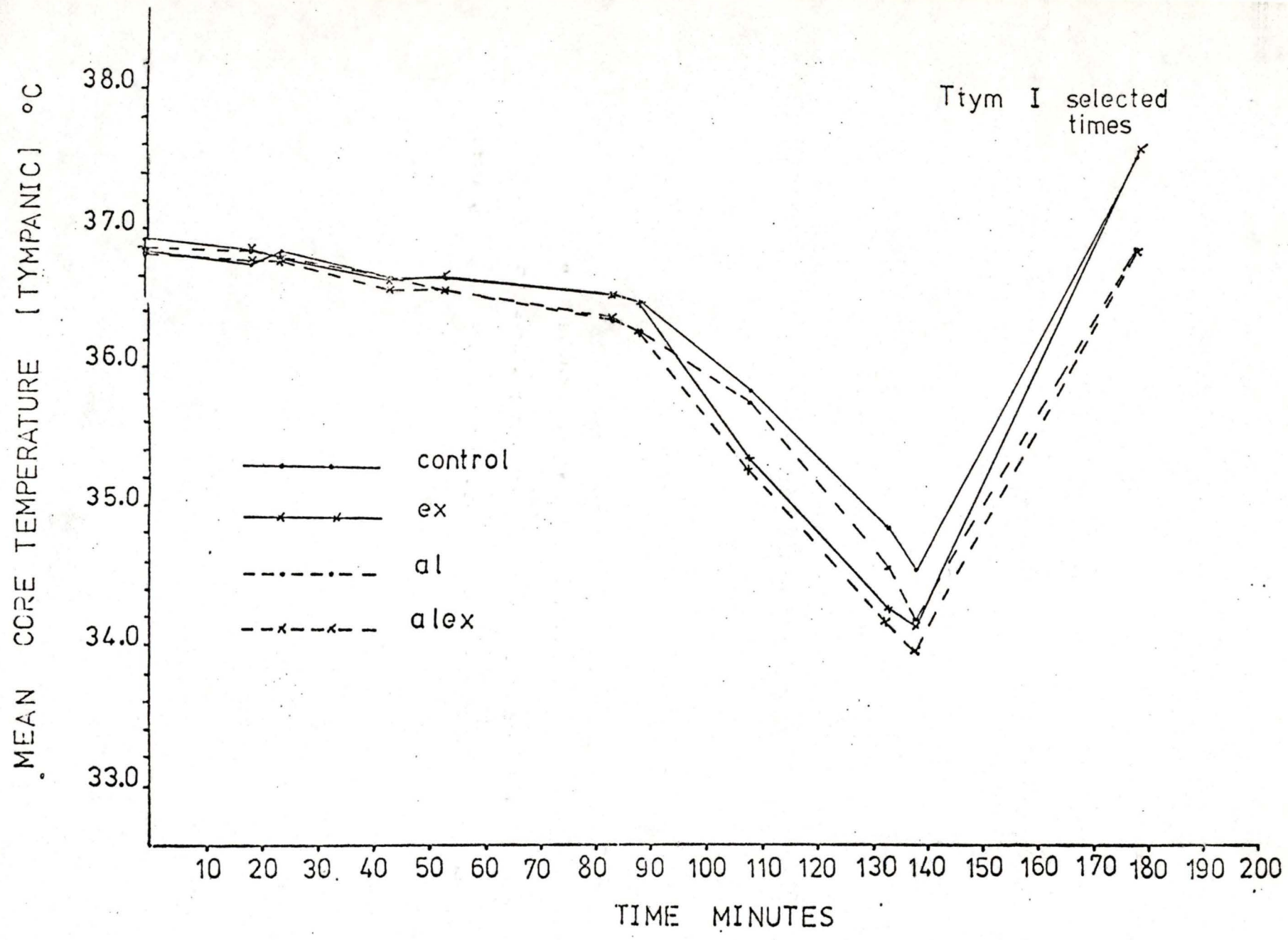

Ttym I
MEAN CORE TEMPERATURE [TYMPANIC] °C
38.0
37.0
36.0
35.0
34.0
33.0
control
ex
al
alex
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

Table IX. The selected times and the alterations of experimental conditions which occurred with them, used for the preliminary analysis of Ttym I "raw score" data

Selected times, minutes	Alteration of experimental condition which occurred
0	beginning of stabilization period
20	end of stabilization period
25	beginning of drinking period
45	end of drinking period
55	absorption period
85	end of absorption period
90	beginning of cold-water immersion period
110	end of exercise period of exercise type immersions
135	end of cold-water immersion period
140	beginning of rewarming period
180	end of rewarming period and termination of experiment

Figure 4. Composite graph of mean tympanic temperatures observed at the selected times of Table IX under control, al, ex and alex treatments for the Ttym I sample.


MEAN CORE TEMPERATURE [TYMPANIC] °C
38.0
37.0
36.0
35.0
34.0
33.0
Ttym I selected times
control
ex
al
alex
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

the major sources of this variability an overall three-factor anova was performed on the raw score data used to calculate the mean tympanic temperatures shown in Figure 4. The results of that three-factor anova appear in Table X.

Table X shows that for the raw score data of Ttym I, all main effects and all interactions between main effects were significant. Therefore, although treatment related differences in tympanic temperatures undoubtably existed, tympanic temperatures also differed at different times during the experiments, and among the ten subjects. Time as a main effect was removed from subsequent analysis by performing two-factor anovas for each "time isolate" of Table IX. These two-factor anovas were designed to determine the relative proportions of overall variance contributed by treatment effects and the effects of inter-personal variation, at any one selected time of the experiments. The results of the actual two-factor anova series performed for the time isolates of Table IX are given in Table XI.

From Table XI, it is clear that the only time isolate which exhibited significant treatment-related differences in regard to raw score data on tympanic temperatures was the 180 minute isolate. On the other hand, all time isolates tested showed that significant differences existed in the tympanic temperatures of the subjects. Such a result indicated that the level of inter-personal variability was inherently high in the raw score data and therefore that any further analysis of the raw score data of Ttym I would not allow a sufficiently critical examination of treatment effects to be accomplished. Consequently, all further analysis of raw score data was abandoned.

In an effort to remove as much inter-personal variability as possible

Table X. Results of overall three-factor anova performed on "raw score" data of Ttym I

Source of variation	f value	Probability value	Significance
Main effects:			
Treatments	25.569	0.000	***
Time	576.261	0.000	***
People	56.583	0.000	***
Interactions:			
Time x people	8.117	0.000	***
Time x Treatments	3.001	0.000	***
Treatments x people	9.057	0.000	***

*** = significant at 0.001% level ($P < 0.001$)

Table XI. Results of two-factor anova series performed on time isolates of Fig. 4

Time isolate, minutes	Source of variation	f value	Prob. value	Sig.
0	treatment	5.468	0.161	ns
	people	1.669	0.000	***
20	treatment	0.941	0.434	ns
	people	5.121	0.000	***
45	treatment	0.778	0.517	ns
	people	4.229	0.002	**
55	treatment	0.888	0.460	ns
	people	5.227	0.000	***
85	treatment	1.998	0.138	ns
	people	4.411	0.001	***
90	treatment	1.195	0.330	ns
	people	4.194	0.002	**
135	treatment	2.439	0.089	ns
	people	7.464	0.000	***
140	treatment	2.192	0.115	ns
	people	9.679	0.000	***
180	treatment	5.521	0.001	***
	people	10.093	0.000	***

Prob. = Probability Sig. = Significance

ns = not significant at 0.05% level ($P > 0.05$)

** = significant at 0.01% level ($P < 0.01$)

*** = significant at 0.001% level ($P < 0.001$)

from the tympanic temperature data, "net differences in tympanic temperatures" between the beginning and end of certain time blocks of the experiments were calculated for the subjects (eg. subject SC's tympanic temperature at time 0 minutes minus his tympanic temperature at time 20 minutes, etc. for all time blocks and all subjects of Ttym I). Table XII presents the time blocks for which the subject's net differences in tympanic temperatures were calculated.

The resulting net differences in tympanic temperatures represented rates of change in the tympanic temperature of each of the subjects under the four different treatments during various periods of the experiments. In order to determine if this manipulation of the data of Ttym I had produced any increased resolution of treatment effects as a result of the removal of a portion of the "masking effect" produced by high inter-personal variability, a three factor anova was performed on the new data. Table XIII presents the results of that overall three-factor anova.

When the results of Table XIII were compared to those of Table X, it was found that the treatments x people interaction was no longer significant. This result indicated that the level of inter-personal variability had been reduced by analysing net differences in tympanic temperature data and that further analysis of that data would probably allow a critical examination of treatment effects on tympanic temperature to be accomplished. Table XIII also showed that treatments as a main effect were non-significant during the overall period of the experiments. In order to determine if any significant treatment effects existed during certain time periods (time blocks) of the experiments, a series of two-factor anovas were performed for each of the time blocks

Table XII. Time span of experimental time blocks for which "net difference in temperatures" data were calculated

Time span, minutes	Experimental time block (ie experimental period)
0 - 20	stabilization period
25 - 45	drinking period
55 - 85	absorption period
90 - 110	exercise period of exercise type cold-water immersions
110 - 135	passive cold-water immersion period of all experiments
135 - 145	afterdrop period
140 - 180	rewarming period

Table XIII. Results of overall three-factor anova performed on "net difference in tympanic temperatures" data of Ttym I

Source of variation	f value	Probability value	Significance
Main effects:			
Treatments	1.317	0.273	ns
Time	903.985	0.000	***
People	3.253	0.023	*
Interactions:			
Time x people	8.153	0.000	***
Time x treatments	4.015	0.000	***
Treatments x people	0.060	0.922	ns

ns = not significant at 0.05% level ($P > 0.05$)

* = significant at 0.05% level ($P < 0.05$)

*** = significant at 0.001% level ($P < 0.001$)

of Table XII. The results of that anova series appears in Table XIV.

Table XIV shows that the 140 to 180 minute period contained significant treatment effects, and that the 90 to 135 minute time block almost showed significance for treatment effects (eg. P = 0.069). In view of these results it was decided to determine the significance of any variations between pairs of treatments for both the 90 to 135 and 140 to 180 minute periods of experiments. Two-factor anovas were employed in the analysis and the results of that anova series appears in Table XV.

Table XV shows that during the rewarming period of experiments, (140 to 180 minute time block) the rate of change in the subject's tympanic temperatures under ex treatment was significantly different from that found in subjects undergoing control, al, or alex treatments. Figure 4 shows that during the above period, both ex and alex treatments produced a decrease in mean tympanic temperatures of subjects compared to their mean temperatures found under control or al treatments. Thus, there was some suggestion that exercise per se, may have slowed the rate of rewarming. However, Figure 4 also shows that the slopes of mean tympanic temperature curves under all four treatments were similar. Furthermore, no significant differences in net differences in tympanic temperatures were found for either the control versus the alex, or the al versus the alex treatments in Table XV. The significant differences found under ex treatment during rewarming therefore, are thought to have resulted as a consequence of a lower starting tympanic temperature observed in ex trials at the initiation of rewarming, rather than as a result of any exercise induced alteration of the actual rate of rewarming.

Table XIV. Results of two-factor anova series performed on "net difference in tympanic temperatures" data for time blocks of Table XII.

Time block, minutes	Source of variation	f value	Probability value	Sig.
0 - 20	treatments	4.029	0.538	ns
	people	1.319	0.026	*
25 - 45	treatments	0.991	0.412	ns
	people	1.703	0.137	ns
55 - 85	treatments	2.686	0.126	ns
	people	1.255	0.305	ns
90 - 135	treatments	2.686	0.069	ns
	people	12.181	0.000	***
140 - 180	treatments	4.660	0.014	*
	people	7.364	0.000	***

Sig. = significance

ns = not significant at 0.05% level ($P > 0.05$)

* = significant at 0.05% level ($P < 0.05$)

*** = significant at 0.001% level ($P < 0.001$)

Table XV. Results of two-factor anova series performed on "net difference in tympanic temperatures" data to determine the significance of variation between treatments for the; 90 to 135, and 140 to 180 minute time blocks of Table XII

Test conditions	Source of var.	f-value	Prob. value	Sig.
A. for 90 to 135 minute time block:				
control vs al	treatments	0.298	0.600	ns
	people	4.451	0.025	*
control vs ex	treatments	15.044	0.005	**
	people	26.060	0.000	***
control vs alex	treatments	4.836	0.059	ns
	people	9.863	0.002	**
al vs ex	treatments	1.940	0.200	ns
	people	3.631	0.044	*
al vs alex	treatments	0.111	0.784	ns
	people	12.867	0.001	***
ex vs alex	treatments	1.471	0.260	ns
	people	3.678	0.042	*
B. for 140 to 180 minute time block:				
control vs al	treatments	0.088	0.290	ns
	people	3.236	0.075	ns
control vs ex	treatments	16.075	0.030	*
	people	8.952	0.005	**
control vs alex	treatments	0.032	0.224	ns
	people	7.041	0.004	**
al vs ex	treatments	6.158	0.038	*
	people	2.389	0.120	ns
al vs alex	treatments	0.174	0.688	ns
	people	2.673	0.093	ns
ex vs alex	treatments	21.306	0.002	**
	people	10.685	0.001	***

var. = variation, Prob. = probability, Sig. = significance,
vs = versus
ns = not significant at 0.05% level ($P > 0.05$)
* = significant at 0.05% level ($P < 0.05$)
** = significant at 0.01% level ($P < 0.01$)
*** = significant at 0.001% level ($P < 0.001$)

Table XV also shows that during the cold-water immersion period of experiments (90 to 135 minute time block) the only pair of treatments which were found to be significantly different regarding net change in tympanic temperatures were the control and ex treatments. However, the control and alex treatments were also found to almost show a significant difference at the 0.05% level ($P = 0.059$). Furthermore, Figure 4 shows that during the cold-water immersion period of experiments, both the ex and alex treatments exhibit decreased mean tympanic temperatures for the subjects, on comparison to the mean tympanic temperatures found under control and al treatments. These results suggested that exercise per se had produced an increase in tympanic cooling during the period of cold-water immersion.

In order to test if exercise had actually produced any increase in tympanic cooling during the 90 to 135 minute period, that time block was further subdivided into a 90 to 110 minute period, (the actual period during which exercise had been performed in ex and alex immersions) and a 110 to 135 minute period, (the period of passive cold exposure which was common to all types of immersions).

Figure 5 presents a graphical representation of the mean differences in tympanic temperatures found for all four treatments during the above mentioned time blocks of cold-water immersions. Table XVI, presents the results of paired t-tests used to determine if any significant differences existed between treatments during the two time periods.

It is clear from Figure 5 and from Table XVI that exercise performed during the al and alex trials did produce an increase in tympanic cooling during the 90 to 110 minute period of experiments. Furthermore, it was shown that the increased level of tympanic cooling found as a result

Figure 5. Graphical representation of mean differences in tympanic temperatures data of the Ttym I sample under control, al, ex and alex treatments for the 90 to 110 and 110 to 135 minute time blocks of Table XII.

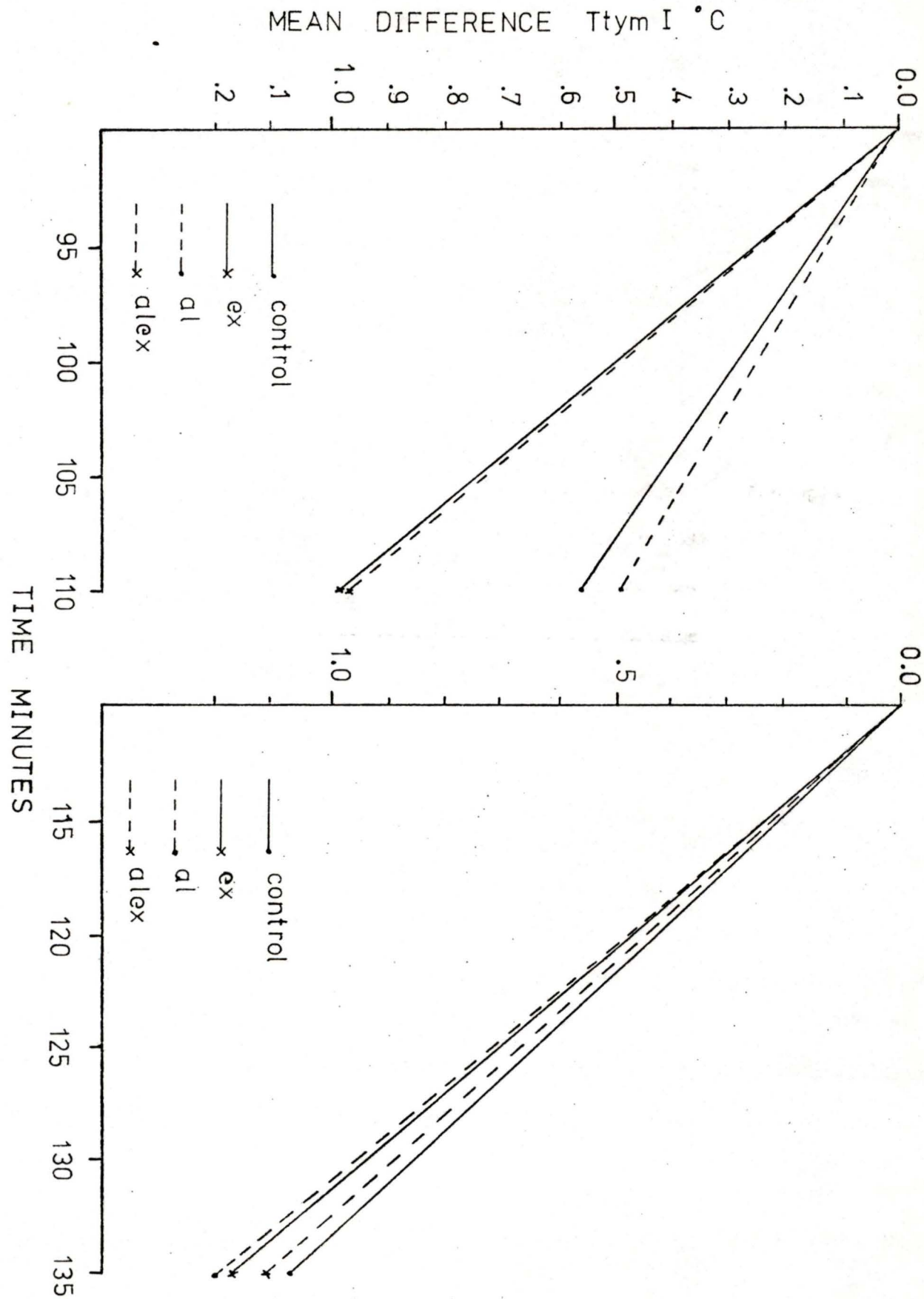

MEAN DIFFERENCE Ttym I °C
0.0
.1
.2
.3
.4
.5
.6
.7
.8
.9
1.0
control
ex
al
alex
95
100
105
110
TIME MINUTES
115
120
125
130
135

Table XVI. Results of t-tests performed on "net difference in tympanic temperatures" data of Ttym I, to determine significance of variation between pairs of treatments for the 90 to 110 and 110 to 135 minute time blocks of Table XII.

Time block, minutes	Test conditions	df	t's	Significance
90 to 110	control vs al	18	1.368	ns
	control vs ex	18	5.553	**
	control vs alex	18	4.222	**
	al vs ex	18	2.979	*
	al vs alex	18	5.712	**
	ex vs alex	18	0.109	ns
110 to 135	control vs al	18	1.295	ns
	control vs ex	18	0.099	ns
	control vs alex	18	0.706	ns
	al vs ex	18	1.086	ns
	al vs alex	18	1.393	ns
	ex vs alex	18	0.083	ns

df = degrees of freedom

ns = not significant at 0.05% level ($P > 0.05$)

* = significant at 0.05% level ($P < 0.05$)

** = significant at 0.01% level ($P < 0.01$)

of performing 20 minutes of exercise, was limited to the actual period of exercise performance, since during the passive exposure period (110 to 135 minutes) net differences in tympanic temperatures were not found to significantly differ amoung any of the experimental treatments.

In summary, the results of the analysis of Ttym I data showed that: a) the consumption of ethanol prior to an immersion performed either with or without exercise in no way altered the tympanic cooling of the subjects and b) that the performance of 20 minutes of exercise either with or without prior ethanol consumption produced a transitory increase in the tympanic cooling of the subjects.

B. Ttym II analysis

Following the exclusion of subjects GP and TM from the experimental sample, new means and standard deviations of tympanic temperatures were calculated for the selected times of Table IX. Figure 6 presents the resulting composite graph of mean tympanic temperatures of the Ttym II sample observed under control, al, ex and alex treatments.[3]

As in the previous analysis of tympanic temperature (Tym I), net differences in tympanic temperature were recalculated for Ttym II's eight subject sample. In order to determine if the removal of subjects GP and TM would result in any new conclusions regarding the treatment's effects on tympanic cooling, a series of two-factor anovas were performed on the data obtained for the time blocks of Table XII. The results of this analysis are shown in Table XVII.

Table XVII reveals that in addition to the 140 to 180 minute time block, the 90 to 135 minute time block contained statistically significant

3 Appendix 12 presents the original single treatment graphs used to construct the composite graph of Figure 6.

Figure 6. Composite graph of mean tympanic temperatures observed at the selected times of Table IX under control, al, ex and alex treatments for the Ttym II sample.

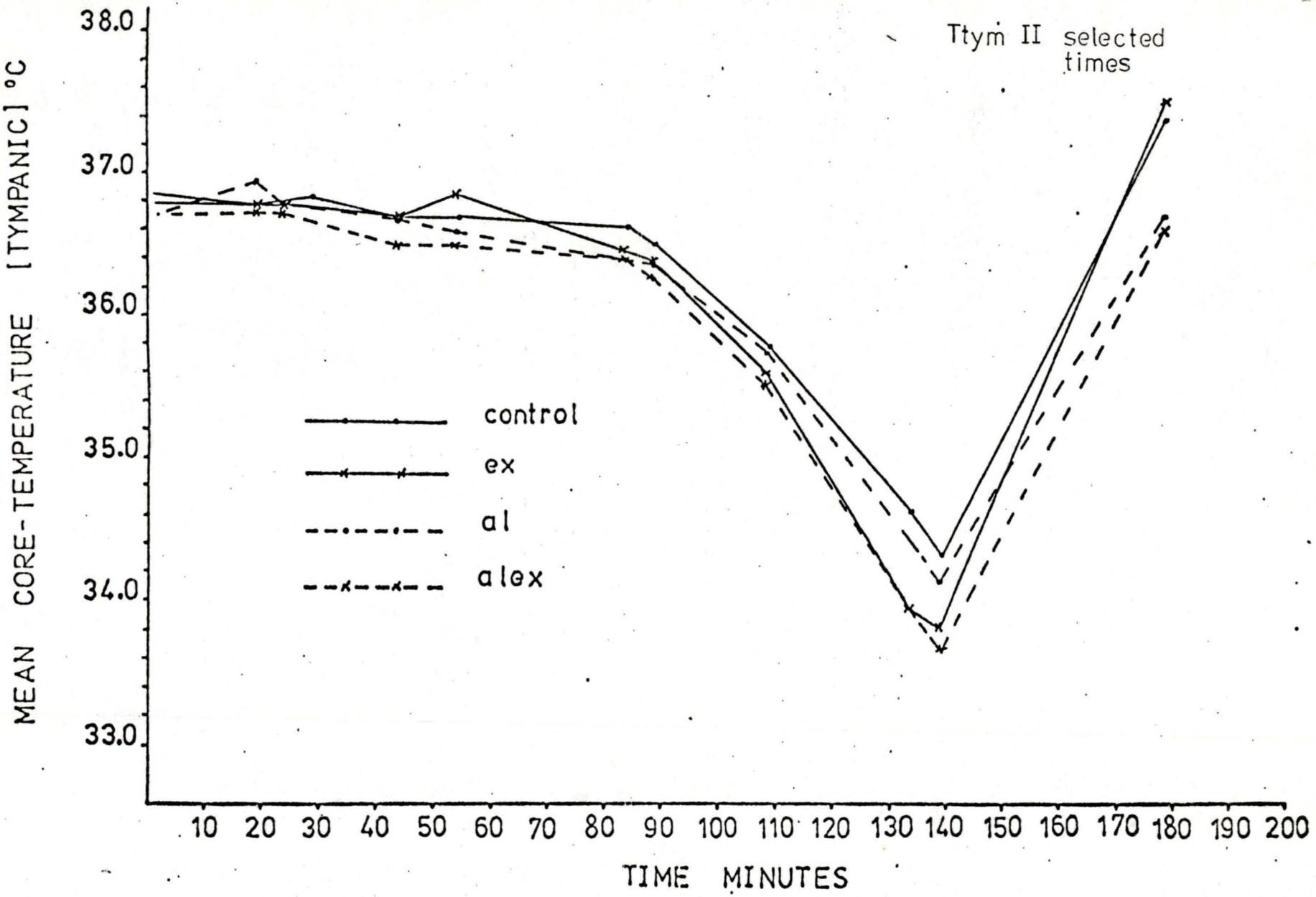

Ttym II selected times
MEAN CORE-TEMPERATURE [TYMPANIC] °C
38.0
37.0
36.0
35.0
34.0
33.0
control
ex
al
alex
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

Table XVII. Results of two-factor anova series performed on "net difference in tympanic temperatures" data of Ttym II for time blocks of Table XII.

Time block, minutes	Source of variation	f value	Probability value	Sig.
0 - 20	treatments	0.512	0.678	ns
	people	2.467	0.052	ns
25 - 45	treatments	2.167	0.122	ns
	people	2.671	0.037	*
55 - 85	treatments	1.317	0.295	ns
	people	1.369	0.269	ns
90 - 135	treatments	4.644	0.014	*
	people	8.296	0.000	***
140 - 180	treatments	5.902	0.005	**
	people	8.987	0.000	***

Sig. = significance

ns = not significant at 0.05% level ($P > 0.05$)

* = significant at 0.05% level ($P < 0.05$)

** = significant at 0.01% level ($P < 0.01$)

*** = significant at 0.001% level ($P < 0.001$)

differences in tympanic cooling due to treatment effects. Comparison of the results of Table XVII with those of Table XIV shows that, decreased inter-personal variability achieved by the exclusion of subjects GP and TM from the experimental sample, resulted in an increase of analytical "resolving power" in the assessment of treatment effects during the 90 to 135 minute block. As for Ttym I, an assessment of variation between pairs of treatments as determined by two-factor anovas was completed for the 90 to 135 and 140 to 180 minute time blocks of Ttym II. The results of these two-factor anova series are presented in Table XVIII.

Table XVIII shows that during the 90 to 135 minute block, net change in tympanic temperature was significantly different between alex and both control and al treatments. Additionally, Table XVIII shows that control and ex treatments differed significantly and that al and ex treatments almost showed a significant difference at the 0.05% level ($P = 0.058$). If the results of Table XVIII for the 90 to 135 minute time block are compared to those seen in Table XV for this time period, it is found that: a) the exclusion of subjects GP and TM resulted in a greater ability to resolve treatment effects on tympanic cooling in general, and b) in spite of this greater resolving ability, there still existed no significant differences in net tympanic temperatures between control and al or ex and alex treatments.

Similarly, during the 140 to 180 minute time period, although Table XVIII shows a greater resolution of the significance of treatment effects, the actual pattern of significance seen between treatments was identical to that observed in Table XV.

Once again the cold exposure period was subdivided into 90 to 110

Table XVIII. Results of two-factor anova series performed on "net difference in tympanic temperatures" data of Ttym II to determine the significance of variation between treatments for the 90 to 135, and 140 to 180 minute time blocks of Table XII

Test conditions	Source of var.	f-value	Prob. value	Sig.
A. for 90 to 135 minute time block:				
control vs al	treatments	0.023	0.884	ns
	people	2.861	0.113	ns
control vs ex	treatments	25.920	0.002	**
	people	23.400	0.001	***
control vs alex	treatments	6.419	0.044	*
	people	4.921	0.037	*
al vs ex	treatments	5.484	0.058	ns
	people	2.985	0.105	ns
al vs alex	treatments	5.966	0.050	*
	people	3.062	0.100	ns
ex vs alex	treatments	0.039	0.850	ns
	people	5.026	0.035	*
B. for 140 to 180 minute time block:				
control vs al	treatments	1.749	0.234	ns
	people	3.554	0.074	ns
control vs ex	treatments	17.389	0.006	**
	people	15.671	0.002	**
control vs alex	treatments	1.881	0.219	ns
	people	10.401	0.006	**
al vs ex	treatments	8.158	0.029	*
	people	2.767	0.121	ns
al vs alex	treatments	0.326	0.589	ns
	people	2.178	0.125	ns
ex vs alex	treatments	41.192	0.001	***
	people	18.410	0.001	***

var. = variation, Prob. = probability, Sig. = significance
vs = versus
ns = not significant at 0.05% level ($P > 0.05$)
* = significant at 0.05% level ($P < 0.05$)
** = significant at 0.01% level ($P < 0.01$)
*** = significant at 0.001% level ($P < 0.001$)

and 110 to 135 minute time blocks and these blocks were re-analysed for overall treatments significance using two-factor anovas. The results of this analysis appear in Table XIX.

Table XIX. Results of two-factor anovas performed on "net differences in tympanic temperatures" data of Ttym II to determine any overall treatments significance during the 90 to 110 and 110 to 135 minute time blocks

Time block, minutes	Source of var.	f value	Prob. value	Sig.
90 to 110	treatments	24.219	0.000	***
	people	6.622	0.000	***
110 to 135	treatments	0.140	0.935	ns
	people	7.080	0.000	***

var. = variation Prob. = probability Sig. = significance
ns = not significant at 0.05% level ($P > 0.05$)
*** = significant at 0.001% level ($P < 0.001$)

From Table XIX it is evident that although significant treatment induced differences existed during the 90 to 110 minute period, no such effects were found in the 110 to 135 minute block. Ttym II data for the 90 to 110 minute block were subjected to two-factor anovas to determine for which treatments significant differences existed. The results of this two-factor anova series are presented in Table XX.

Table XX shows that all treatments, with the exception of ex and alex treatments, were found to differ significantly from one and another. Table XX, unlike Table XVI, shows that the control and al treatments

Table XX. Results of two-factor anova series performed on "net difference in tympanic temperatures" data of Ttym II, to determine significance of variation between pairs of treatments for the 90 to 110 minute time block of Table XII.

Test conditions	Source of variation	f value	Prob. value	Sig.
control vs al	treatment	7.440	0.029	*
	people	9.100	0.005	**
control vs ex	treatment	19.496	0.003	**
	people	2.944	0.089	ns
control vs alex	treatment	28.493	0.001	***
	people	5.394	0.020	*
al vs ex	treatment	55.269	0.000	***
	people	3.881	0.047	*
al vs alex	treatment	31.752	0.001	***
	people	2.249	0.154	ns
ex vs alex	treatment	0.019	0.893	ns
	people	3.228	0.072	ns

Prob. = probability

Sig. = significance

vs = versus

ns = not significant at 0.05% level ($P > 0.05$)

* = significant at 0.05% level ($P < 0.05$)

** = significant at 0.01% level ($P < 0.01$)

*** = significant at 0.001% level ($P < 0.01$)

significantly differed during the 90 to 110 minute period. However, Figure 6 shows that although mean tympanic temperatures for control and al treatments were equivalent at 110 minutes, mean tympanic temperature under al treatment at 90 minutes actually started out at a lower temperature compared to that temperature found under control treatment. Thus, the significant difference observed in Table XX between these two treatments would appear to have resulted as a consequence of differing starting tympanic temperatures, rather than from any ethanol-induced modification of tympanic cooling during immersion.

In summary, it is evident that the exclusion of subjects GP and TM from the experimental sample of Ttym II resulted in an increase in the analytical ability to discriminate treatment effects on tympanic temperatures. However, no new conclusions regarding the general effects of either ethanol or exercise on tympanic cooling were deduced from the analysis of Ttym II data. The results of Ttym II analysis therefore verifies at more statistically critical levels the conclusions drawn from the analysis of Ttym I data, that; a) ethanol did not modify tympanic cooling and b) exercise depressed tympanic temperatures during the actual period of its performance.

C. Ttym III analysis

The net differences in tympanic temperatures calculated between the beginning and end of time blocks, used in the analyses of Ttym I and Ttym II samples actually represent crude measures of tympanic cooling rates. These crude rates could be expressed as total tympanic temperature change $^{o}C \cdot X$ minutes of a time block^{-1}. Consequently, it was decided that tympanic temperature cooling rates, expressed as $^{o}C \cdot hour^{-1}$, would be calculated for each individual, and would be analysed overall for

treatment effects in the Ttym III sample. Table XXI presents the time blocks used in the calculation of tympanic cooling rates of individuals of the Ttym III sample.

Table XXI shows that during the pre-drinking, drinking and absorption periods of experiments, identical time blocks to those used to calculate net tympanic temperature differences in Table XII, were used to calculate tympanic cooling rates of the Ttym III sample. It will be noticed from Table XXI that time blocks used during the immersion, afterdrop and rewarming periods of the experiments are not the same as those time blocks used during these periods in Table XII. It was necessary to sub-divide the tympanic cooling curve into portions by which, through forced linearity, meaningful slopes for tympanic cooling rate calculations could be achieved. For this reason the time blocks indicated in Table XXI, rather than those used in Table XII, were used in Ttym III analysis.

Table XXII presents for the time blocks of Table XXI the mean tympanic cooling rates observed under control, al, ex and alex experiments.

To determine if the mean cooling rates of Table XXII significantly differed amoung treatments, t-tests between pairs of treatments were performed for each of the time periods of Table XXII. The outcome of the t-test series are given in Table XXIII.

Table XXIII shows that during the drinking period of experiments, al treatment produced a higher mean rate of tympanic cooling than that seen under either control or ex treatments. Table XXII also shows that the alex mean cooling rate during the drinking period was also higher, although non-significantly so, than either control or ex cooling rates. On the basis of the results of Table XXIII, it was concluded that ethanol

Table XXI. Time span of experimental time blocks for which "mean cooling rate" data were calculated

Time span, minutes	Experimental time block (ie experimental period)
0 - 20	stabilization period
25 - 45	drinking period
55 - 85	absorption period
100 - 130	immersion period
135 - 145	afterdrop period
150 - 180	rewarming period

Table XXII. Mean tympanic cooling rates and their standard deviations calculated for the time blocks of Table XXI. for control, al, ex, and alex treatments (Ttym III)

Time block, minutes	Treatments, control, $^{o}C \cdot hr^{-1}$		al, $^{o}C \cdot hr^{-1}$		ex, $^{o}C \cdot hr^{-1}$		alex, $^{o}C \cdot hr^{-1}$	
	Mean	sd	Mean	sd	Mean	sd	Mean	sd
0 - 20	-0.04	0.671	-0.39	0.749	0.17	0.645	-0.26	1.074
25 - 45	0.13	0.293	0.64	0.113	0.17	0.293	0.39	0.449
55 - 85	0.11	0.157	0.34	0.276	0.26	0.190	0.20	0.258
100 - 130	3.56	1.555	3.50	1.273	4.03	1.690	3.97	1.157
135 - 145	2.83	1.951	3.09	1.879	1.46	1.141	1.80	1.510
150 - 180	-7.80	2.205	-8.40	2.581	-8.14	1.610	-8.24	2.534

hr = hour

sd = standard deviation of mean

Table XXIII. Results of t-test series performed on mean tympanic cooling rates, between treatment pairs for data of Table XXII. (Ttym III)

Time block, minutes	Test conditions	t's	Significance
0 - 20	control versus al	0.921	ns
	control versus ex	-0.597	ns
	control versus alex	0.460	ns
	al versus ex	-1.518	ns
	al versus alex	-0.263	ns
	ex versus alex	0.908	ns
25 - 45	control versus al	-4.299	***
	control versus ex	-0.256	ns
	control versus alex	-1.284	ns
	al versus ex	3.962	***
	al versus alex	1.429	ns
	ex versus alex	-1.086	ns
55 - 85	control versus al	-1.915	ns
	control versus ex	-1.286	ns
	control versus alex	-0.787	ns
	al versus ex	0.631	ns
	al versus alex	0.989	ns
	ex versus alex	0.493	ns
100 - 135	control versus al	0.079	ns
	control versus ex	-0.541	ns
	control versus alex	-0.560	ns
	al versus ex	-0.663	ns
	al versus alex	-0.723	ns
	ex versus alex	0.076	ns
135 - 145	control versus al	-0.254	ns
	control versus ex	1.604	ns
	control versus alex	1.105	ns
	al versus ex	1.961	ns
	al versus alex	1.416	ns
	ex versus alex	-0.475	ns
150 - 180	control versus al	0.468	ns
	control versus ex	0.330	ns
	control versus alex	0.347	ns
	al versus ex	-0.226	ns
	al versus alex	-0.117	ns
	ex versus alex	0.088	ns

ns = not significant at 0.05% level ($P > 0.05$)

*** = significant at 0.001% level ($P < 0.001$)

consumption accelerated the rate of mean tympanic cooling during the drinking period of al type experiments in the Ttym III sample.

D. Trec I analysis

Figure 7 presents a composite graph of mean rectal temperatures observed at ten minute intervals under control, al, ex and alex treatments.[4] As in previous tympanic temperature analysis (Tym I), rectal temperatures were analysed for treatment effects at the selected times indicated in Table IX. Figure 8 presents a composite graph of mean rectal temperatures observed at the above times under control, al, ex and alex treatments.[5]

"Raw score" rectal temperature data were analysed for the Trec I sample, and as in Ttym I, was found to be inadequate to determine critically the significance of treatment effects on rectal temperatures, because of high levels of inter-personal variability. Raw score Trec I analysis was therefore abandoned.

"Net change in rectal temperatures" between the beginning and the end of time blocks given in Table XII were calculated for the Trec I sample. As in the Ttym I analysis, this data was analysed by performing a series of two-factor anovas on each of the time blocks indicated in Table XII. The results of this analysis are presented in Table XXIV.

From Table XXIV it is clear that only the 55 to 85 minute time block contained significant treatment induced alterations in net change in rectal temperatures. In order to determine which treatments significantly differed during the 55 to 85 minute time block, two-factor anovas

4 Appendix 13 presents the single treatment graphs used to construct Figure 7.

5 Appendix 14 presents the single treatment graphs used to construct Figure 8.

Figure 7. Composite graph of mean rectal temperatures observed at ten minute intervals under control, al, ex and alex treatments for the Trec I sample.

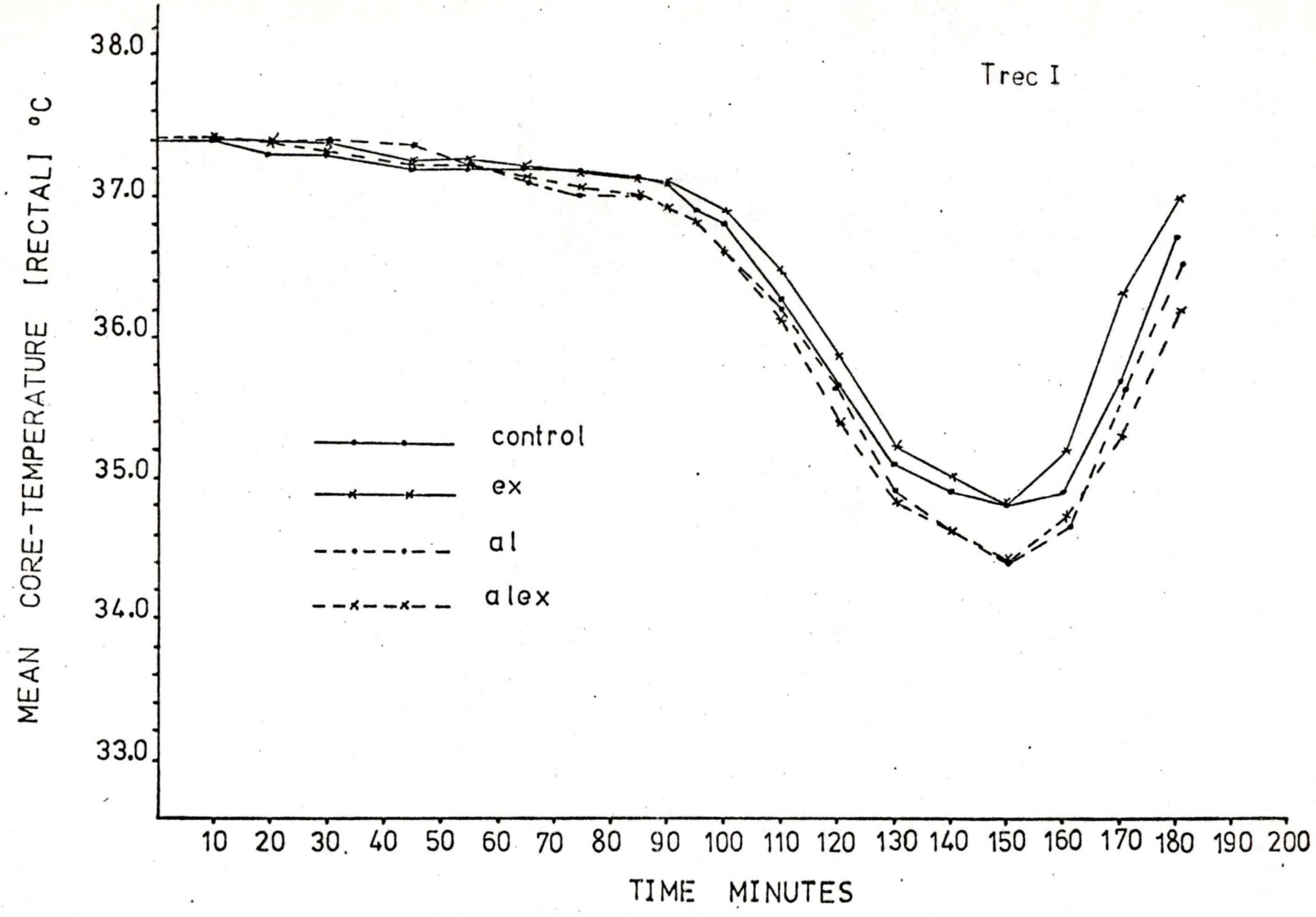

Trec I
MEAN CORE-TEMPERATURE [RECTAL] °C
38.0
37.0
36.0
35.0
34.0
33.0
control
ex
al
alex
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

Figure 8. Composite graph of mean rectal temperatures observed at the selected times of Table IX under control, al, ex and alex treatments for the Trec I sample.

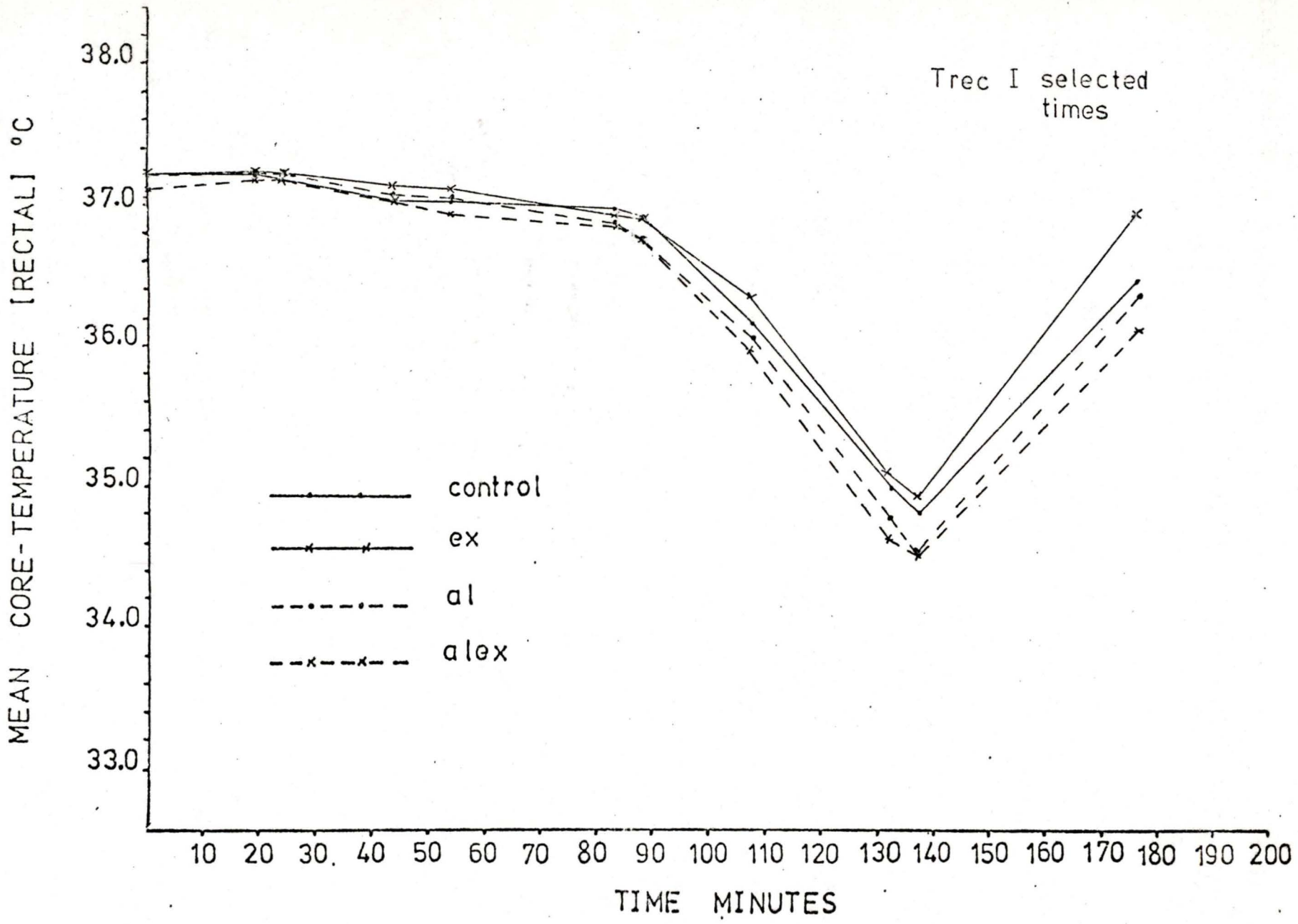

Trec I selected times
MEAN CORE-TEMPERATURE [RECTAL] °C
38.0
37.0
36.0
35.0
34.0
33.0
control
ex
al
alex
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

Table XXIV. Results of two-factor anova series performed on "net difference in rectal temperatures" data of Trec I for time blocks of Table XII.

Time block, minutes	Source of variation	f value	Probability value	Sig.
0 - 20	treatments	0.264	0.851	ns
	people	1.429	0.225	ns
25 - 45	treatments	0.804	0.503	ns
	people	1.993	0.080	ns
55 - 85	treatments	4.968	0.007	**
	people	1.244	0.311	ns
90 - 110	treatments	0.989	0.413	ns
	people	6.942	0.000	***
90 - 135	treatments	0.544	0.657	ns
	people	11.085	0.000	***
110 - 135	treatments	0.165	0.919	ns
	people	7.199	0.000	***
135 - 150	treatments	0.166	0.918	ns
	people	0.914	0.522	ns
140 - 180	treatments	0.515	0.676	ns
	people	5.927	0.000	***

Sig. = significance

ns = not significant at 0.05% level (P 0.05)

** = significant at 0.01% level (P < 0.01)

*** = significant at 0.001% level (P < 0.001_

were performed between treatment pairs. The results of the two-factor anova series are given in Table XXV.

During the 55 to 85 minute time block, no exercise was performed. Therefore, the control and ex, and al and alex treatments were actually replicates of each other during this time period. Figure 8 shows that during the absorption period of the experiments (i.e. 55 to 85 minutes), mean rectal temperatures appeared to be lower under both al and alex treatments than under control and ex treatments. Statistically however, and as Table XXV indicates, it was only possible to verify that rectal temperatures differed between al treatment and either control or ex treatments. Since al and alex are experimental replicates during the 55 to 85 minute period, rectal temperatures would be expected to be similar under both treatments. However, Table III shows that mean BACs were approximately ten mgm% lower in alex type experiments than in al experiments during the absorption period. It would appear that the failure to show significantly different net changes in rectal temperatures between alex and control or ex treatments in Table XXV indicates that mean BAC under alex treatment was at an insufficient level to elicit the effects of ethanol consumption on rectal cooling evident under al treatment.

In summary, the results of Trec I analysis showed that the consumption of ethanol produced some increase in rectal cooling during the absorption period of al type experiments.

E. Trec II analysis

Following the exclusion of subjects GP and TM from the experimental sample, new mean rectal temperatures were calculated for Trec II data. Figure 9 presents a composite graph of mean rectal temperatures seen

Table XXV. Results of two-factor anova series performed on "net difference in rectal temperatures" data of Trec I to determine the significance of variation between pairs of treatments for the 55 to 85 minute time block of Table XII.

Test conditions	Source of variation	f value	Prob. value	Sig.
control vs al	treatment	8.805	0.021	*
	people	0.804	0.624	ns
control vs ex	treatment	2.087	0.182	ns
	people	0.449	0.876	ns
control vs alex	treatment	2.939	0.121	ns
	people	0.837	0.603	ns
al vs ex	treatment	9.447	0.013	*
	people	3.981	0.026	*
al vs alex	treatment	3.992	0.077	ns
	people	1.000	0.500	ns
ex vs alex	treatment	0.000	1.000	ns
	people	0.927	0.544	ns

Sig. = significance

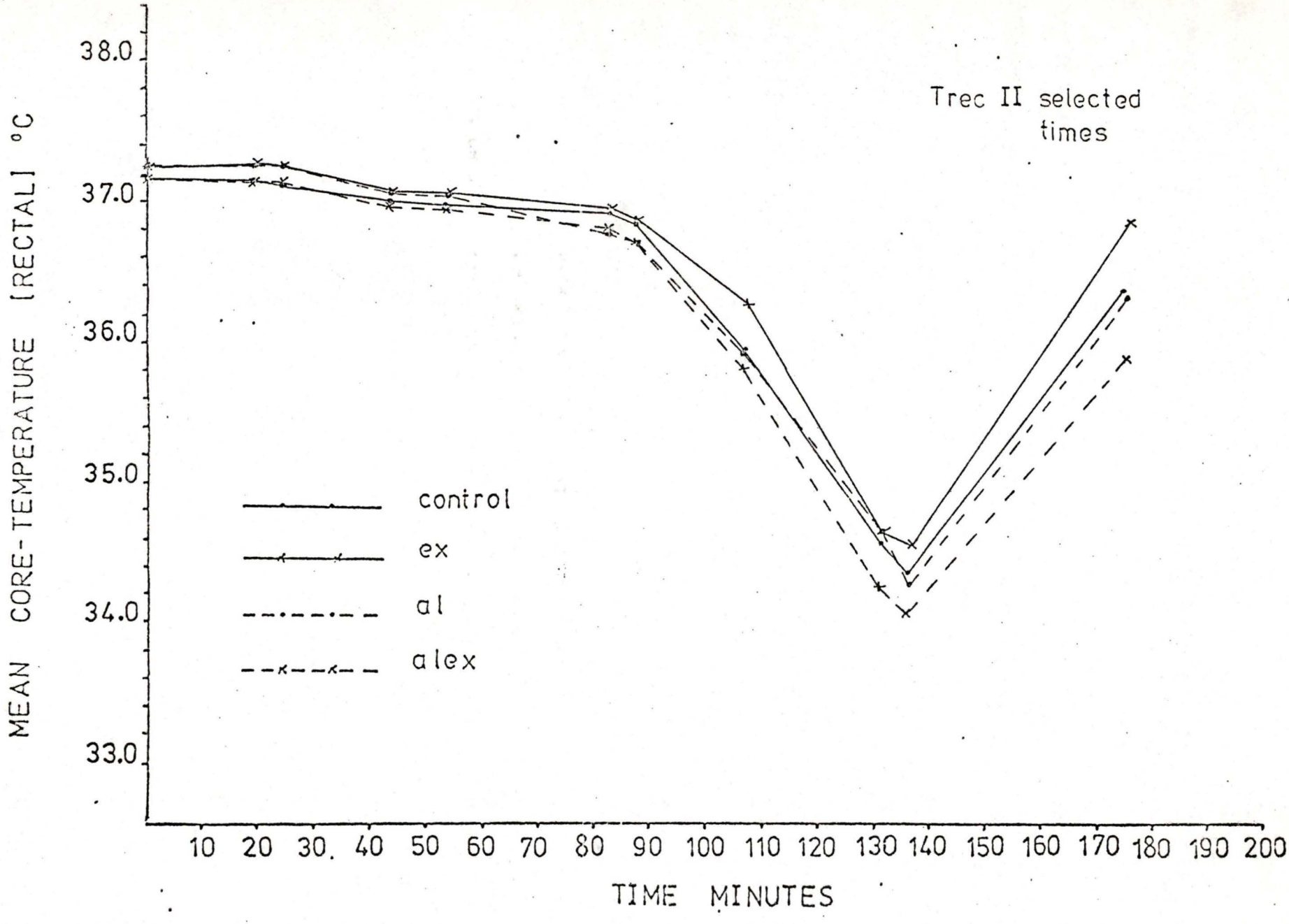
vs = versus

ns = not significant at 0.05% level ($P > 0.05$)

* = significant at 0.05% level ($P < 0.05$)

Prob. = probability

Figure 9. Composite graph of mean rectal temperatures observed at the selected times of Table IX under control, al, ex and alex treatments for the Trec II sample.


Trec II selected times
MEAN CORE-TEMPERATURE (RECTAL) °C
38.0
37.0
36.0
35.0
34.0
33.0
control
ex
al
alex
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

under control, al, ex and alex treatments for the selected times of Table IX.[6] As in the Ttym I analysis, net differences in rectal temperatures calculated for the time blocks of Table XII were subjected to two-factor anovas to determine if differences due to treatment effects existed. Table XXVI presents the results of this anova series.

From Table XXVI, it is clear that significant treatment induced alterations in the net change of rectal temperatures existed only during the 55 to 85 minute time block. Consequently, the absorption period was further analysed by two-factor anovas to decide between which pairs of treatments significant differences existed. The results of this analysis appear in Table XXVII.

Table XXVII shows, that rectal cooling under al treatment differed from that observed under either control or ex treatments. Furthermore, Figure 9 shows, that mean rectal temperatures under al trials were lower than those found under either control or ex experiments. However as in Table XXV, Table XXVII indicates that it is only possible to verify statistically that a reduced rectal temperature existed under al treatment.

Additionally, both Tables XXVI and XXVII display an improvement in the resolving ability of the analyses involving Trec II data over those analyses utilizing Trec I data. It will be noticed however that although Tables XXVI and XXVII exhibit an increased overall resolution of treatment effects, the actual patern of significance of treatment effects was not altered from that seen in Tables XXIV or XXV. Thus, the analysis of Trec II data serves to verify the conclusion of the Trec I analysis at a

6 Appendix 15 presents the single treatment graphs used to construct Figure 9.

Table XXVI. Results of two-factor anova series performed on "net difference in rectal temperatures" data of Trec II for time blocks of Table XII.

Time block, minutes	Source of variation	f value	Probability value	Sig.
0 - 20	treatments	0.193	0.900	ns
	people	1.541	0.208	ns
25 - 45	treatments	1.503	0.243	ns
	people	2.047	0.096	ns
55 - 85	treatments	4.642	0.012	*
	people	1.452	0.238	ns
90 - 110	treatments	1.057	0.388	ns
	people	4.140	0.005	**
90 - 135	treatments	0.842	0.489	ns
	people	6.191	0.001	***
110 - 135	treatments	0.284	0.836	ns
	people	4.610	0.005	**
135 - 150	treatments	0.231	0.874	ns
	people	0.858	0.544	ns
140 - 180	treatments	0.530	0.667	ns
	people	4.198	0.008	**

Sig. = significance

ns = not significant at 0.05% level ($P > 0.05$)

* = significant at 0.05% level ($P < 0.05$)

** = significant at 0.01% level ($P < 0.01$)

*** = significant at 0.001% level ($P < 0.001$)

Table XXVII. Results of two-factor anova series performed on "net difference in rectal temperatures" data of Trec II to determine significance of variation between pairs of treatments for the 55 to 85 minute time block of Table XII.

Test conditions	Source of variation	f value	Prob. value	Sig.
control vs al	treatment	9.704	0.012	*
	people	0.861	0.567	ns
control vs ex	treatment	1.595	0.247	ns
	people	0.367	0.895	ns
control vs alex	treatment	3.500	0.104	ns
	people	0.875	0.569	ns
al vs ex	treatment	12.600	0.009	**
	people	6.350	0.013	*
al vs alex	treatment	3.544	0.102	ns
	people	1.234	0.394	ns
ex vs alex	treatment	0.042	0.844	ns
	people	0.761	0.636	ns

Prob. = probability

Sig. = significance

vs = versus

ns = not significant at 0.05% level ($P > 0.05$)

* = significant at 0.05% level ($P < 0.05$)

** = significant at 0.01% level ($P < 0.01$)

statistically more critical level, i.e. that ethanol consumption reduced rectal cooling in the subjects during the absorption period of al type experiments.

F. Trec III analysis

As in the Ttym III sample, rectal cooling rates (as $^{o}C \cdot hour^{-1}$) of the Trec II sample were calculated for control, al, ex and alex treatments using the time blocks of Table XXI. Table XXVIII presents the mean rectal cooling rates calculated for the above time blocks and treatments.

As in the Ttym III analysis, the means of Table XXVIII were subjected to a series of t-tests to determine if mean rectal cooling rates differed between pairs of treatments within any particular time block. The results of this analysis appear in Table XXIX.

From Table XXIX it can be seen that a significant difference in rectal cooling rates was only found between the control and al treatments during the pre-drinking period of the experiments. The pre-drinking period represented a stabilization period common to all experiments, and therefore it would seem probable that the higher mean rectal cooling rate seen under control treatment resulted as an artifact of said stabilization.

Table XXIX also indicates that no significant differences existed in mean rectal cooling rate amoung any treatments during the absorption period of experiments. This result contradicts the conclusions drawn from the analysis of both Trec I and Trec II data during the 55 to 85 minute time period. For reasons which will be presented in the discussion section, it would appear that the lack of significant differences amoung treatments for Trec III data, resulted from a degree of expected analytical error.

Table XXVIII. Mean rectal cooling rates and their standard deviations calculated for the time blocks of Table XXI. for control, al, ex, and alex treatments (Trec III)

Time block, minutes	Treatments							
	control, $^{o}C \cdot hr^{-1}$		al, $^{o}C \cdot hr^{-1}$		ex, $^{o}C \cdot hr^{-1}$		alex, $^{o}C \cdot hr^{-1}$	
	Mean	sd	Mean	sd	Mean	sd	Mean	sd
0 - 20	0.23	0.311	-0.08	0.212	0.08	0.223	-0.04	0.669
25 - 45	0.51	0.641	0.34	0.207	0.26	0.207	0.47	0.596
55 - 85	-0.09	0.677	0.48	0.398	0.23	0.190	0.29	0.195
100 - 130	3.91	1.568	3.70	1.065	4.09	1.726	4.34	0.799
135 - 145	3.26	1.861	2.57	2.237	2.14	1.564	2.40	1.897
150 - 180	-5.66	1.804	-6.30	3.000	-6.16	2.540	-5.06	2.234

hr = hour

sd = standard deviation of the mean

Table XXIX. Results of t-test series performed on mean rectal cooling rates, between treatment pairs for data of Table XXVIII. (Trec III)

Time block, minutes	Test conditions	t's	Significance
0 - 20	control versus al	2.181	*
	control versus ex	1.055	ns
	control versus alex	1.045	ns
	al versus ex	-1.411	ns
	al versus alex	-0.164	ns
	ex versus alex	-0.492	ns
25 - 45	control versus al	0.667	ns
	control versus ex	0.981	ns
	control versus alex	0.121	ns
	al versus ex	0.723	ns
	al versus alex	-0.559	ns
	ex versus alex	-0.880	ns
55 - 85	control versus al	-1.955	ns
	control versus ex	-1.204	ns
	control versus alex	-1.427	ns
	al versus ex	1.561	ns
	al versus alex	1.195	ns
	ex versus alex	-0.332	ns
100 - 135	control versus al	0.293	ns
	control versus ex	-0.204	ns
	control versus alex	-0.646	ns
	al versus ex	-0.509	ns
	al versus alex	-1.272	ns
	ex versus alex	-0.348	ns
135 - 145	control versus al	-0.282	ns
	control versus ex	1.226	ns
	control versus alex	0.856	ns
	al versus ex	0.419	ns
	al versus alex	0.153	ns
	ex versus alex	-0.281	ns
150 - 180	control versus al	0.484	ns
	control versus ex	0.549	ns
	control versus alex	-0.553	ns
	al versus ex	-0.094	ns
	al versus alex	-0.878	ns
	ex versus alex	-0.860	ns

ns = not significant at 0.05% level ($P > 0.05$)

* = significant at 0.05% level ($P < 0.05$)

III. Metabolic Rate

Subjects' $\dot{O}_2$ consumption records for each experiment were corrected for STP, and used to calculate metabolic rate. Figure 10 presents a composite graph of mean metabolic rates observed at ten minute intervals for the ten subject experimental sample, under control, al, ex and alex treatments.[7] From Figure 10 it would appear that significant differences in mean metabolic rates existed amoung treatments during the immersion period of experiments. In order to determine if the apparent differences seen in Figure 10 were significant, a series of two-factor anovas were performed on the original "raw score" data used to calculate the means of Figure 10. Table XXX presents the results of this two-factor anova series.

From Table XXX it is evident that significant treatment-induced differences existed in the data on metabolic rate, and that these differences were limited to the actual cold water immersion period of experiments. In order to determine which treatments produced significantly different metabolic rates, further two-factor anovas were performed between pairs of treatments for all significant time isolates of Table XXX. Table XXXI gives the results of these anovas.

From Table XXXI and Figure 10, the following results regarding the metabolic rates of subjects under different treatments during the cold water immersion period of experiments were found:

a) throughout the entire 90 to 130 minute period, lower metabolic rates were seen under al treatment than under ex treatment.

b) between 90 and 120 minutes, metabolic rates were lower under al

7 Appendix 16 presents the original single treatment graphs used to construct Figure 10.

Figure 10. Composite graph of mean metabolic rates observed at ten minute intervals under control, al, ex and alex treatments for the ten subject sample.
(STAB = stabilization period of experiments,
DR = drinking period, ABSORPTION = absorption period,
IMMERSION = cold-water immersion period,
REWARM = rewarming period.)

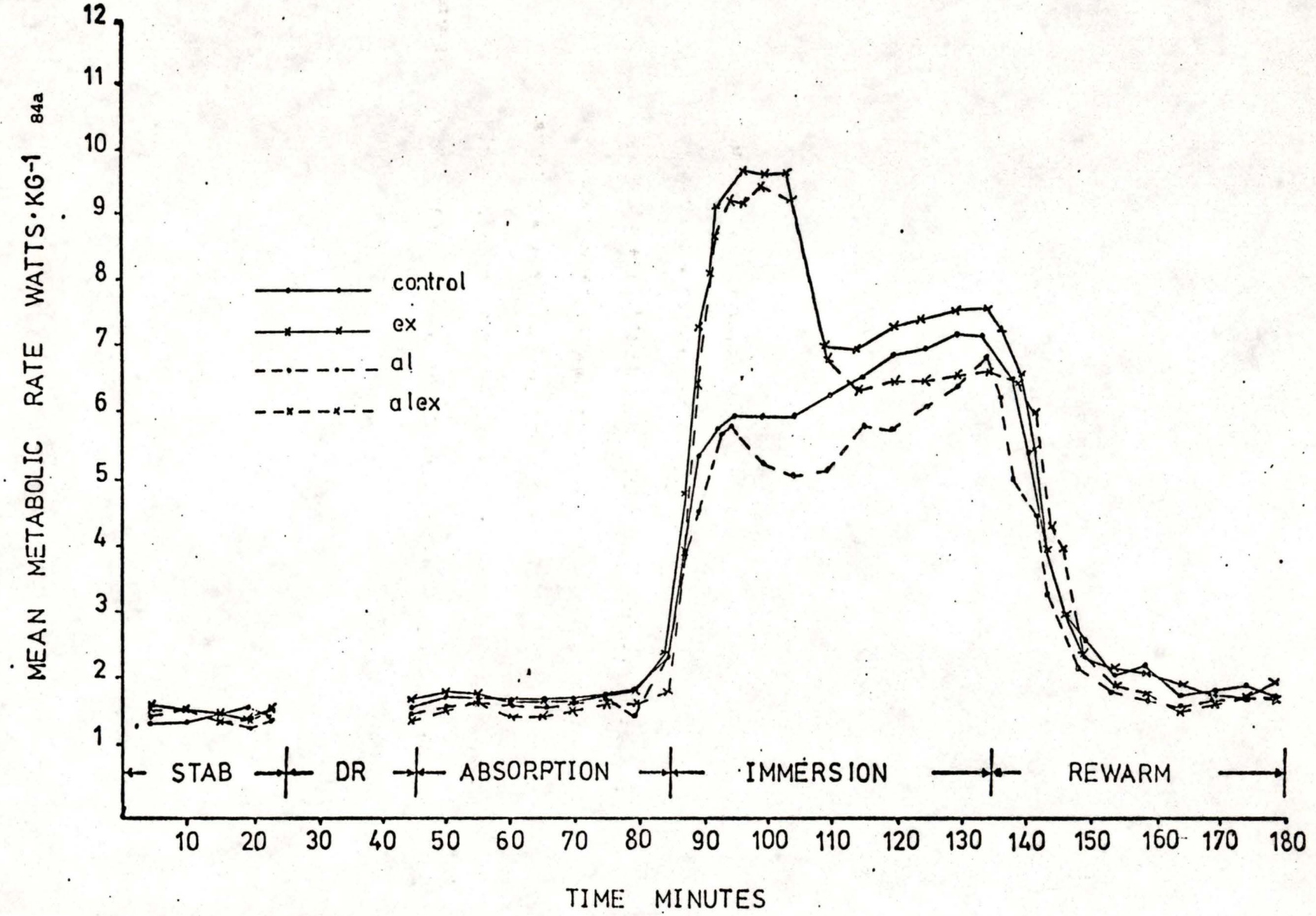

MEAN METABOLIC RATE WATTS·KG-1
84a
12
11
10
9
8
7
6
5
4
3
2
1
control
ex
al
alex
STAB
DR
ABSORPTION
IMMERSION
REWARM
10
20
30
40
50
60
70
80
90
100
110
120
130
140
150
160
170
180
TIME MINUTES

Table XXX. Results of two-factor anova series performed on "raw score" data of metabolic rate for various time isolates

Time isolate, minutes	Source of variation	f value	Prob. value	Significance
15	treatments	0.674	0.575	ns
	people	1.668	0.146	ns
85	treatments	1.932	0.148	ns
	people	1.921	0.092	ns
90	treatments	5.004	0.007	**
	people	2.630	0.025	*
100	treatments	90.223	0.000	***
	people	15.564	0.000	***
110	treatments	8.433	0.000	***
	people	12.976	0.000	***
115	treatments	7.743	0.001	***
	people	41.318	0.000	***
120	treatments	11.835	0.000	***
	people	36.543	0.000	***
130	treatments	10.556	0.000	***
	people	48.571	0.000	***
135	treatments	1.582	0.220	ns
	people	14.079	0.000	***
170	treatments	0.888	0.461	ns
	people	1.127	0.381	ns

Prob. = probability

ns = not significant at 0.05% level ($P > 0.05$)

* = significant at 0.05% level ($P < 0.05$)

** = significant at 0.01% level ($P < 0.01$)

*** = significant at 0.001% level ($P < 0.001$)

Table XXXI. Results of two-factor anova series performed on "raw score" data of metabolic rate, between pairs of treatments for the significant time isolates of Table XXX.

Time isolate, minutes	Test conditions	Source of var.	f value	Prob. value	Sig.
90	control vs al	treatment	1.846	0.207	ns
		people	3.718	0.032	*
	control vs ex	treatment	8.472	0.017	*
		people	3.409	0.041	*
	control vs alex	treatment	2.343	0.160	ns
		people	1.369	0.324	ns
	al vs ex	treatment	17.860	0.002	**
		people	3.629	0.034	*
	al vs alex	treatment	7.063	0.026	*
		people	1.471	0.287	ns
	ex vs alex	treatment	0.493	0.500	ns
		people	0.607	0.766	ns
95	control vs al	treatment	1.207	0.300	ns
		people	6.824	0.004	**
	control vs ex	treatment	301.198	0.000	***
		people	20.012	0.000	***
	control vs alex	treatment	46.752	0.000	***
		people	3.792	0.030	*
	al vs ex	treatment	196.911	0.000	***
		people	9.602	0.001	***
	al vs alex	treatment	62.088	0.000	***
		people	3.924	0.027	*
	ex vs alex	treatment	0.294	0.601	ns
		people	5.294	0.100	ns
100	control vs al	treatment	18.889	0.002	**
		people	24.813	0.000	***
	control vs ex	treatment	231.408	0.000	***
		people	18.036	0.000	***
	control vs alex	treatment	67.415	0.000	***
		people	6.146	0.006	**

	al vs ex	treatment	247.030	0.000	***
		people	10.837	0.001	***
	al vs alex	treatment	74.129	0.000	***
		people	3.556	0.036	*
	ex vs alex	treatment	0.203	0.659	ns
		people	9.071	0.002	**
105	control vs al	treatment	22.108	0.001	***
		people	21.169	0.000	***
	control vs ex	treatment	460.476	0.000	***
		people	35.671	0.000	***
	control vs alex	treatment	131.463	0.000	***
		people	11.868	0.001	***
	al vs ex	treatment	217.884	0.000	***
		people	8.910	0.002	**
	al vs alex	treatment	110.086	0.000	***
		people	4.836	0.014	*
	ex vs alex	treatment	2.092	0.182	ns
		people	12.037	0.001	***
110	control vs al	treatment	33.113	0.000	***
		people	29.200	0.000	***
	control vs ex	treatment	1.189	0.304	ns
		people	3.696	0.032	*
	control vs alex	treatment	2.138	0.178	ns
		people	12.681	0.000	***
	al vs ex	treatment	13.294	0.005	**
		people	3.731	0.031	ns
	al vs alex	treatment	34.270	0.000	***
		people	15.574	0.000	***
	ex vs alex	treatment	0.017	0.898	ns
		people	3.803	0.030	*
115	control vs al	treatment	18.760	0.002	**
		people	26.535	0.000	***
	control vs ex	treatment	0.735	0.413	ns
		people	14.871	0.000	***
	control vs alex	treatment	0.208	0.659	ns
		people	20.782	0.000	***
	al vs ex	treatment	18.465	0.002	**
		people	16.205	0.000	***
	al vs alex	treatment	14.189	0.004	**
		people	27.615	0.000	***
	ex vs alex	treatment	3.405	0.098	ns
		people	35.004	0.000	***

120	control vs al	treatment	18.872	0.002	**
		people	16.146	0.000	***
	control vs ex	treatment	0.839	0.384	ns
		people	18.993	0.000	***
	control vs alex	treatment	2.977	0.119	ns
		people	31.868	0.000	***
	al vs ex	treatment	17.314	0.002	**
		people	9.043	0.002	**
	al vs alex	treatment	13.353	0.005	**
		people	24.376	0.000	***
	ex vs alex	treatment	8.155	0.019	*
		people	29.565	0.000	***
130	control vs al	treatment	13.980	0.005	**
		people	25.748	0.000	***
	control vs ex	treatment	1.047	0.333	ns
		people	24.169	0.000	***
	control vs alex	treatment	18.194	0.002	**
		people	65.767	0.000	***
	al vs ex	treatment	17.756	0.002	**
		people	14.323	0.000	***
	al vs alex	treatment	0.701	0.424	ns
		people	26.172	0.000	***
	ex vs alex	treatment	12.727	0.006	**
		people	18.774	0.000	***

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var. = variation

Prob. = probability

Sig. = significance

vs = versus

ns = not significant at 0.05% level ($P > 0.05$)

* = significant at 0.05% level ($P < 0.05$)

** = significant at 0.01% level ($P < 0.01$)

*** = significant at 0.001% level ($P < 0.001$)

treatment than under alex treatment

c) between 100 and 130 minutes, metabolic rates were lower under al treatment than under control treatment

d) between 90 and 115 minutes, ex and alex treatments exhibited equivalent metabolic rates

e) between 120 and 130 minutes, metabolic rates were lower under alex treatment than under ex treatment

f) between 95 and 110 minutes, both control and al treatments exhibited metabolic rates which were lower than those seen under either ex or alex treatments

g) between 110 and 120 minutes, under both ex and alex treatments metabolic rates were seen to decrease to levels equivalent to those under control and al treatments

h) between 120 and 130 minutes, metabolic rates under alex treatment were seen to continue to decrease to a level lower than that seen under either control or ex treatments but equivalent to a level observed under al treatment.

In view of the above results, the overall percentage changes in metabolic rates, for significantly different treatment pairs, were calculated for various time periods of the cold water immersion. Table XXXII presents the results of these calculations.

From Table XXXII and the preceeding results of the analysis of metabolic rates, it is evident that: a) ethanol consumption prior to immersions in the absence of exercise reduced mean metabolic rate in subjects by approximately 20%, b) during the period of its performance, exercise successfully combatted ethanol's tendancy to reduce mean metabolic rate in subjects, and c) following the cessation of performing exercise, ethanol reduced mean metabolic rate by approximately 10%.

Table XXXII. Overall change as percent in mean metabolic rate calculated between pairs of significantly different treatments, for various time intervals during the cold-water immersion period of experiments

Time interval, minutes	Treatments compaired	Overall % change in mean meta. rate	Direction of change
90 - 135	al versus ex	28.61	dec. for al
95 - 105	con. versus ex	37.04	dec. for con.
95 - 105	al versus alex	43.88	dec. for al
100 - 135	con. versus al	18.22	dec. for al
120 - 135	ex versus alex	10.00	dec. for alex
130 - 135	con. versus alex	5.16	dec. for alex

meta. = metabolic

con. = control

dec. = decreased

IV. Skin Temperature

Figures 11, 12, 13 and 14 present composite graphs of mean skin temperatures observed at each of the four skin sites tested for control, al, ex and alex treatments.[8] From these figures, it can be seen that at each site, mean skin temperatures under all four treatments displayed a great deal of similarity during the course of the experiments. As a result of the apparent equivalence of mean skin temperatures at different skin sites, significant differences between the control treatment and the other three treatments, were tested for at selected times, at each skin site. The analysis was performed using t-tests, and the results appear as separate tables presented below, each table representing a different skin site.

Table XXXIII gives the results found for the sternum site.

Table XXXIII shows that at the sternum site, mean skin temperatures observed under al, ex and alex treatments did not significantly differ from those seen under control conditions.

Table XXXIV presents the results of the analysis for the left bicep skin site.

From Table XXXIV it is evident that at the left bicep site, no treatment induced alterations in mean skin temperatures existed.

Table XXXV presents the results of the analysis for the left thigh skin site.

Table XXXV shows that at the left thigh site, with the exception of times 90, 120 and 170 minutes, mean skin temperatures observed under al,

8 Appendices 17, 18, 19 and 20 present the original single treatment graphs used to construct Figures 11 through 14. The single treatment graphs do not have standard deviations plotted; however, Appendix 21 lists these missing standard deviations.

Figure 11. Composite graph of mean skin temperatures observed at ten minute intervals at the sternum site under control, al, ex and alex treatments for the ten subject sample.

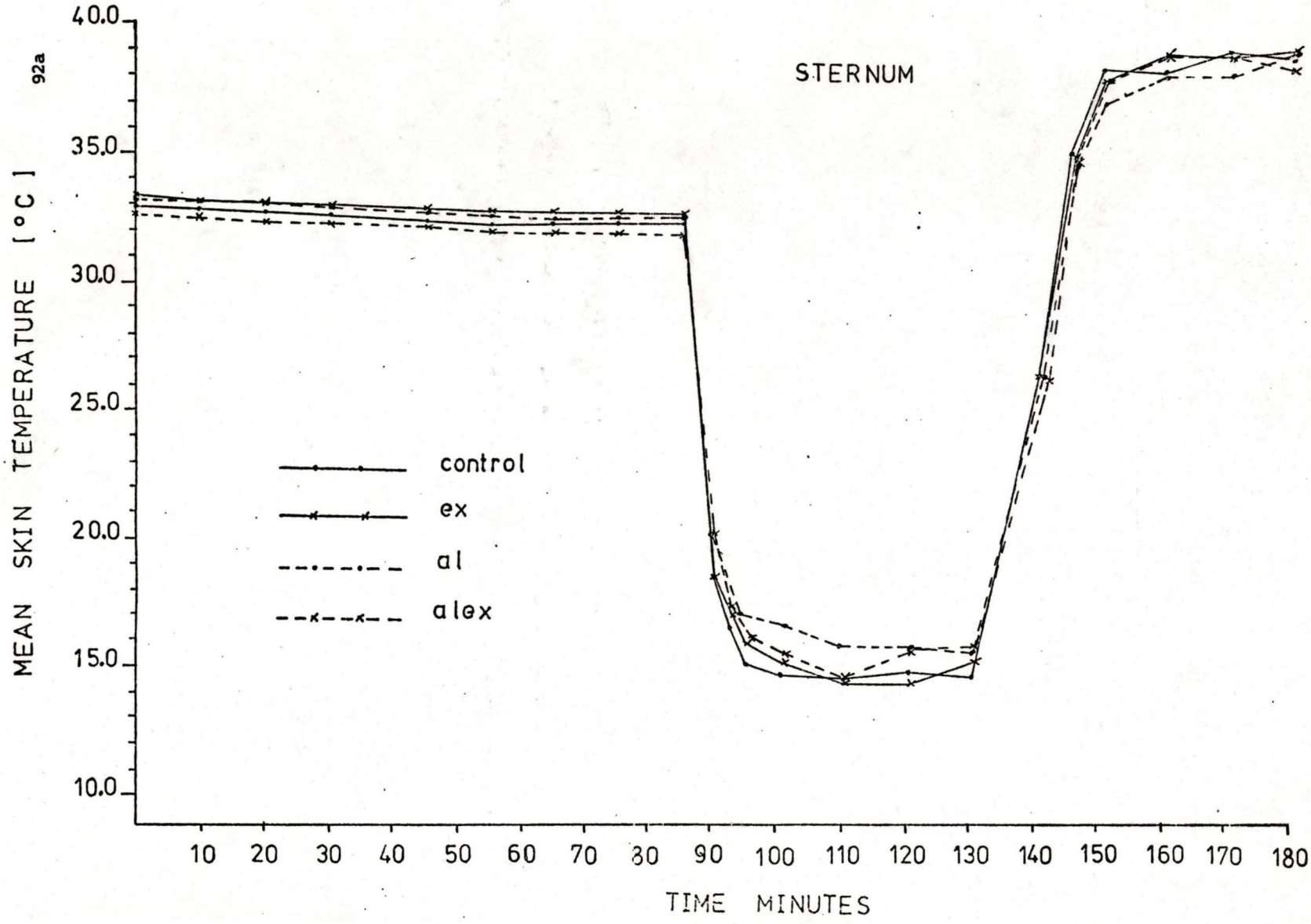

STERNUM
MEAN SKIN TEMPERATURE [°C]
92a
40.0
35.0
30.0
25.0
20.0
15.0
10.0
control
ex
al
alex
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES

Figure 12. Composite graph of mean skin temperatures observed at ten minute intervals at the left biceps site under control, al, ex and alex treatments for the ten subject sample.

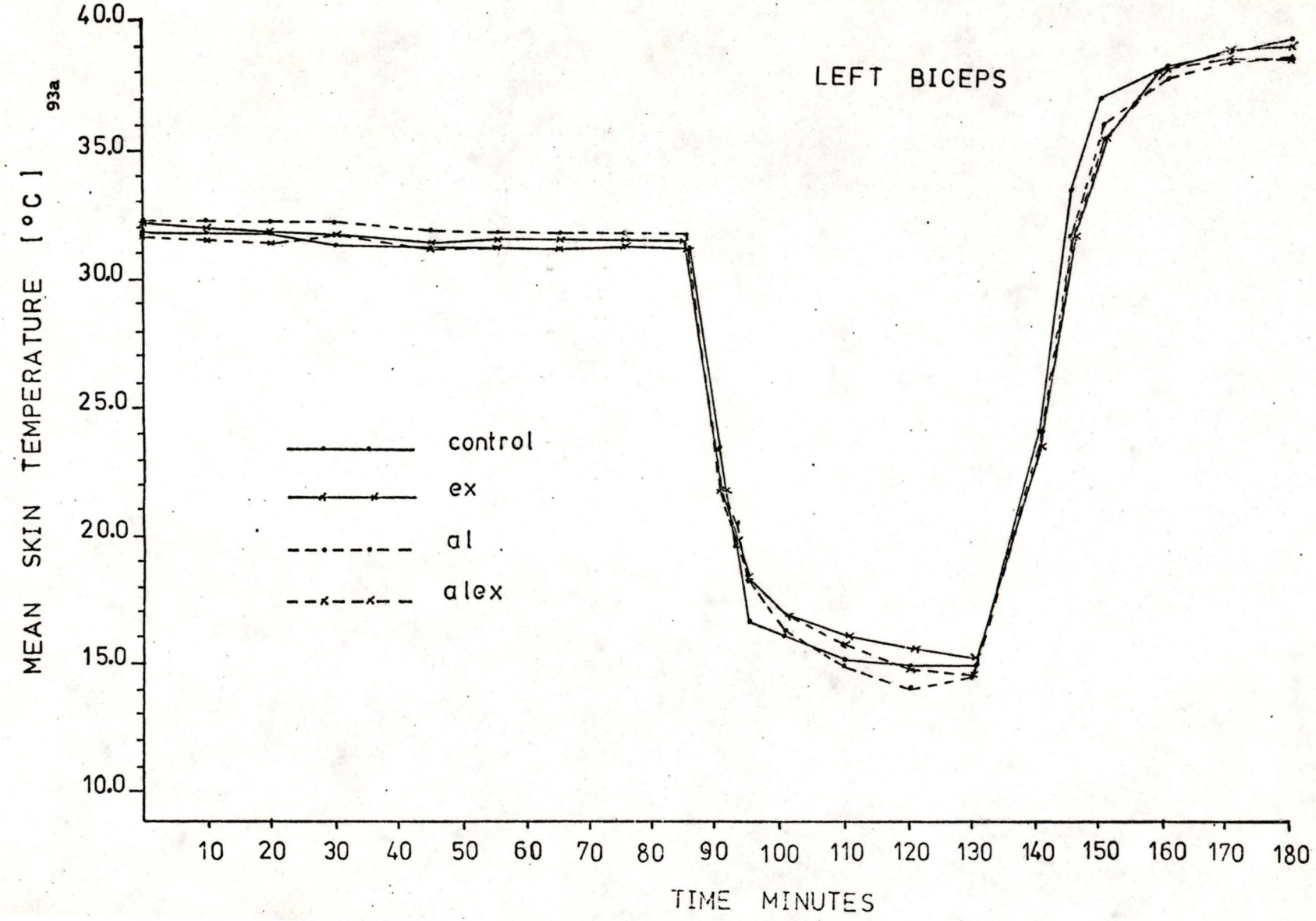

LEFT BICEPS
MEAN SKIN TEMPERATURE [°C] 93a
40.0
35.0
30.0
25.0
20.0
15.0
10.0
control
ex
al
alex
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES

Figure 13. Composite graph of mean skin temperatures observed at ten minute intervals at the left thigh site under control, al, ex and alex treatments for the ten subject sample.

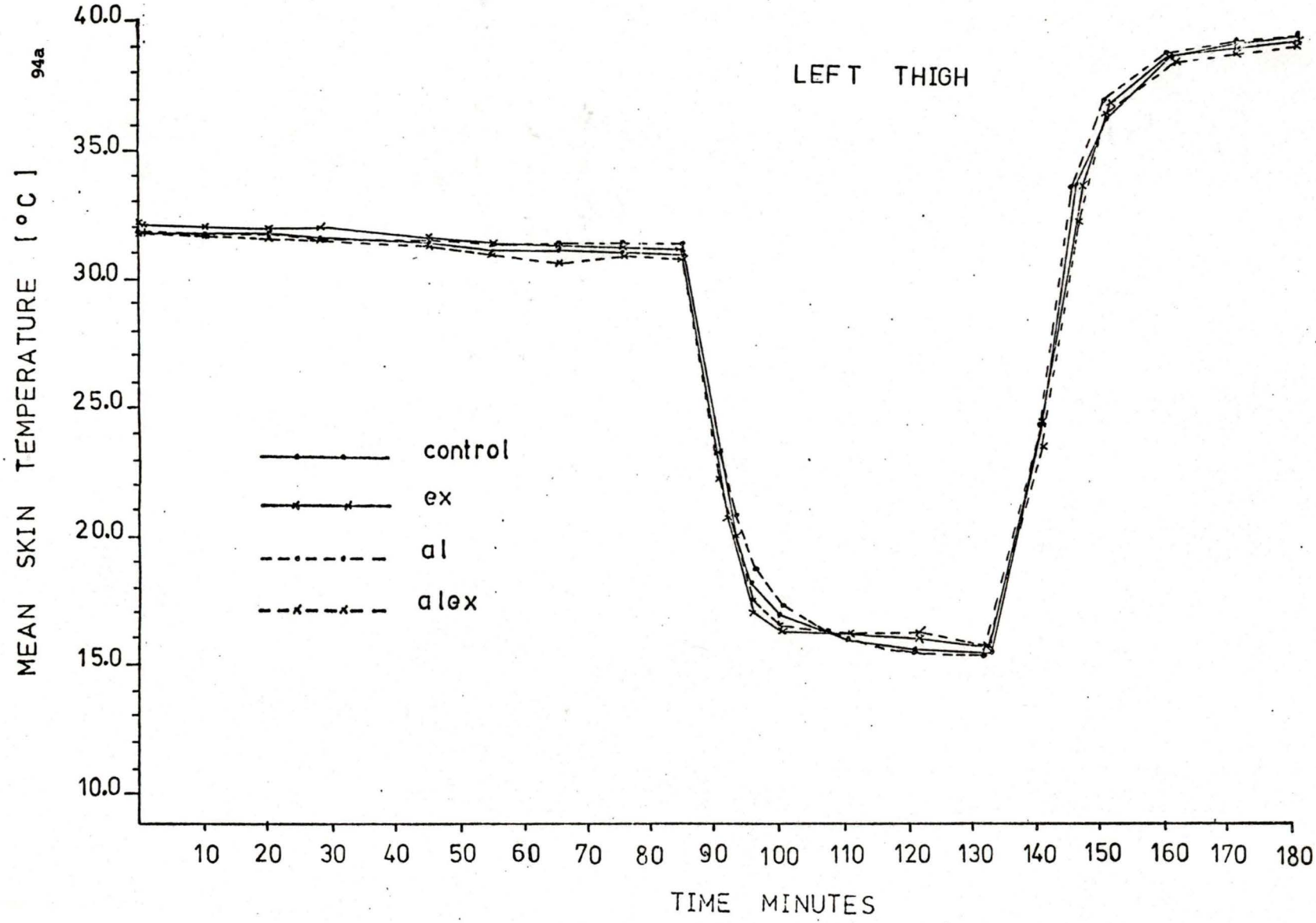

LEFT THIGH
MEAN SKIN TEMPERATURE [°C]
94a
40.0
35.0
30.0
25.0
20.0
15.0
10.0
control
ex
al
alex
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES

Figure 14. Composite graph of mean skin temperatures observed at ten minute intervals at the lumbar site under control, al, ex and alex treatments for the ten subject sample.

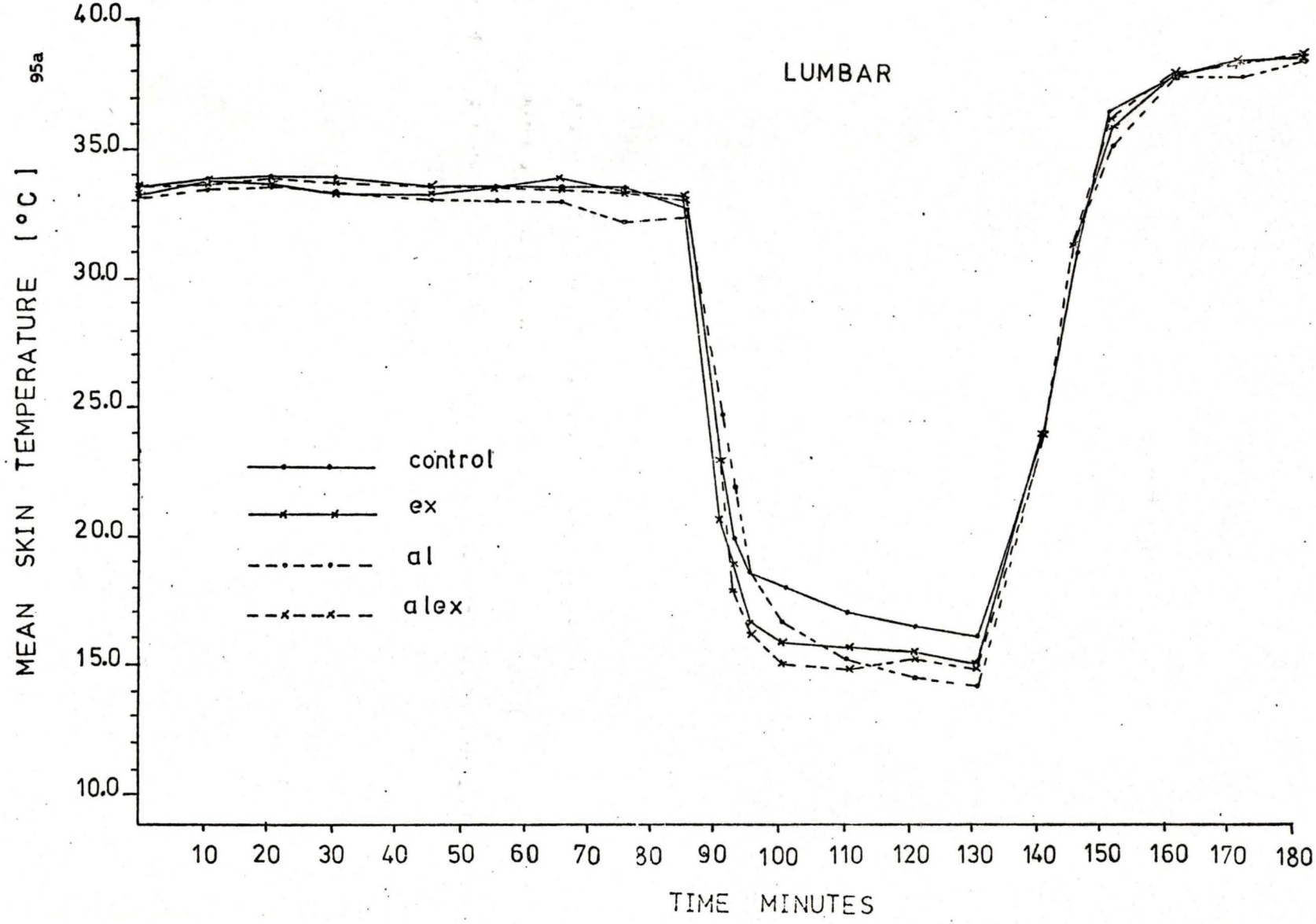

LUMBAR
MEAN SKIN TEMPERATURE [°C]
95a
40.0
35.0
30.0
25.0
20.0
15.0
10.0
control
ex
al
alex
10
20
30
40
50
60
70
80
90
100
110
120
130
140
150
160
170
180
TIME MINUTES

Table XXXIII. Results of t-tests performed between pairs of treatments on mean skin temperature data at sternum site for various times

Time isolate, minutes	Test conditions	Sternum site t's	Significance
20	control vs al	-0.787	ns
	control vs ex	-0.561	ns
	control vs alex	0.907	ns
85	control vs al	-0.180	ns
	control vs ex	-1.581	ns
	control vs alex	1.147	ns
90	control vs al	-0.891	ns
	control vs ex	-0.098	ns
	control vs alex	-1.430	ns
100	control vs al	-1.737	ns
	control vs ex	-0.428	ns
	control vs alex	-0.902	ns
110	control vs al	-1.234	ns
	control vs ex	0.184	ns
	control vs alex	0.122	ns
120	control vs al	0.139	ns
	control vs ex	-0.073	ns
	control vs alex	1.083	ns
130	control vs al	-0.958	ns
	control vs ex	-0.647	ns
	control vs alex	-1.307	ns
150	control vs al	1.999	ns
	control vs ex	1.456	ns
	control vs alex	1.493	ns
170	control vs al	2.009	ns
	control vs ex	0.542	ns
	control vs alex	0.541	ns

ns = not significant at 0.05% level ($P > 0.05$)

vs = versus

Table XXXIV. Results of t-tests performed between pairs of treatments on mean skin temperatures at left bicep site for various times

Time isolate, minutes	Test conditions	Left bicep site t's	Significance
20	control vs al	-1.974	ns
	control vs ex	-0.605	ns
	control vs alex	0.553	ns
85	control vs al	-1.398	ns
	control vs ex	-1.017	ns
	control vs alex	-0.023	ns
90	control vs al	0.289	ns
	control vs ex	1.679	ns
	control vs alex	1.826	ns
100	control vs al	-0.519	ns
	control vs ex	-1.657	ns
	control vs alex	-0.673	ns
110	control vs al	0.465	ns
	control vs ex	-1.780	ns
	control vs alex	-1.444	ns
120	control vs al	1.812	ns
	control vs ex	-1.399	ns
	control vs alex	0.209	ns
130	control vs al	0.367	ns
	control vs ex	-0.858	ns
	control vs alex	0.679	ns
150	control vs al	1.618	ns
	control vs ex	1.132	ns
	control vs alex	0.995	ns
170	control vs al	1.719	ns
	control vs ex	-0.775	ns
	control vs alex	0.464	ns

ns = not significant at 0.05% level ($P > 0.05$)

vs = versus

Table XXXV. Results of t-tests performed between pairs of treatments on mean skin temperatures at left thigh site for various times

Time isolate, minutes	Test conditions	Left thigh site t's	Significance
20	control vs al	0.001	ns
	control vs ex	-0.363	ns
	control vs alex	0.559	ns
85	control vs al	-0.937	ns
	control vs ex	2.282	*
	control vs alex	1.437	ns
90	control vs al	-0.483	ns
	control vs ex	1.785	ns
	control vs alex	1.130	ns
100	control vs al	0.803	ns
	control vs ex	-1.785	ns
	control vs alex	-1.130	ns
110	control vs al	0.808	ns
	control vs ex	-0.385	ns
	control vs alex	-1.066	ns
120	control vs al	1.006	ns
	control vs ex	-1.430	ns
	control vs alex	-2.638	*
130	control vs al	1.916	ns
	control vs ex	-0.304	ns
	control vs alex	-0.736	ns
150	control vs al	0.862	ns
	control vs ex	0.616	ns
	control vs alex	1.517	ns
170	control vs al	1.492	ns
	control vs ex	-0.361	ns
	control vs alex	3.562	**

ns = not significant at 0.05% level ($P > 0.05$)

* = significant at 0.05% level ($P < 0.05$)

** = significant at 0.01% level ($P < 0.01$)

vs = versus

ex and alex treatments did not differ from those seen under control conditions.

Table XXXVI presents the results of the analysis performed for the lumbar site.

From Table XXXVI it is clear that at the lumbar site, no treatment induced alterations in mean skin temperatures existed with the exception of times 100 and 170 minutes.

At no site was a consistent treatment related difference in mean skin temperature found during the immersion or rewarming periods of experiments. Therefore, it was concluded that treatments had no physiological effect on mean skin temperature.

Figures 11 through 14 also show that a high degree of similarity existed for skin temperature curves at different skin sites. In order to determine if mean skin temperatures at different skin sites actually covaried with one and another in respect to time, a series of correlations on the mean skin temperatures of identical treatments between different skin sites were performed. The results of these correlations appear in Table XXXVII.

Table XXXVII shows that mean skin temperatures did significantly covary between different sites under identical treatments.

Figure 15 presents a composite graph of mean environmental temperatures observed under control, al, ex and alex treatments.[9]

A comparison of Figures 11 through 14 with Figure 15 shows that mean skin temperatures apparently closely followed mean environmental temperatures. Table XXXVIII shows the results of a correlation performed

9 Appendix 22 presents the original single treatment graphs used to construct Figure 15. Once again, standard deviations have been ommitted from these graphs; however, Appendix 23 lists these missing standard deviations.

Table XXXVI. Results of t-tests performed between pairs of treatments on mean skin temperatures at lumbar site for various times

Time isolates, minutes	Test conditions	Lumbar site t's	Significance
20	control vs al	1.052	ns
	control vs ex	0.883	ns
	control vs alex	0.077	ns
85	control vs al	0.548	ns
	control vs ex	-0.793	ns
	control vs alex	-0.777	ns
90	control vs al	-1.289	ns
	control vs ex	2.089	ns
	control vs alex	-0.487	ns
100	control vs al	0.576	ns
	control vs ex	1.379	ns
	control vs alex	2.279	*
110	control vs al	1.324	ns
	control vs ex	0.477	ns
	control vs alex	1.669	ns
120	control vs al	-0.131	ns
	control vs ex	-0.082	ns
	control vs alex	-0.129	ns
130	control vs al	2.089	ns
	control vs ex	0.195	ns
	control vs alex	0.476	ns
150	control vs al	0.752	ns
	control vs ex	0.934	ns
	control vs alex	1.183	ns
170	control vs al	2.216	*
	control vs ex	0.132	ns
	control vs alex	0.845	ns

ns = not significant at 0.05% level ($P > 0.05$)

* = significant at 0.05% level ($P < 0.05$)

vs = versus

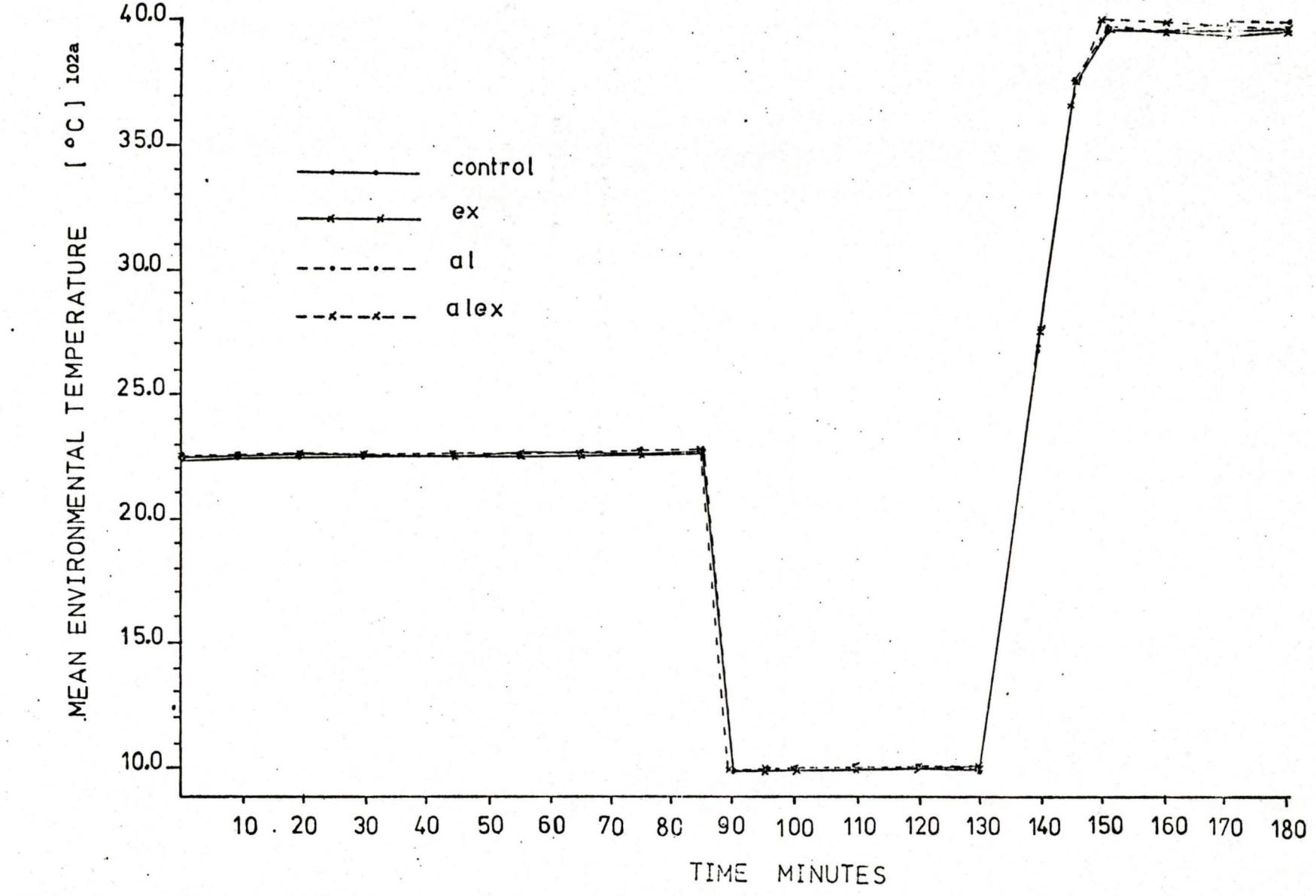
Table XXXVII. Results of correlation series performed for identical treatments but between different skin sites for mean skin temperatures

Sites correlated	Test conditions	r	Significance
Sternum & Lumbar	control vs control	0.990	***
	al vs al	0.986	***
	ex vs ex	0.996	***
	alex vs alex	0.989	***
Sternum & Lt. thigh	control vs control	0.989	***
	al vs al	0.999	***
	ex vs ex	0.883	***
	alex vs alex	0.997	***
Sternum & Lt. biceps	control vs control	0.986	***
	al vs al	0.992	***
	ex vs ex	0.992	***
	alex vs alex	0.996	***
Lt. biceps & lt. thigh	control vs control	0.999	***
	al vs al	0.998	***
	ex vs ex	0.998	***
	alex vs alex	0.998	***
Lumbar & Lt. biceps	control vs control	0.995	***
	al vs al	0.997	***
	ex vs ex	0.995	***
	alex vs alex	0.995	***
Lumbar & Lt. thigh	control vs control	0.995	***
	al vs al	0.994	***
	ex vs ex	0.995	***
	alex vs alex	0.991	***

Lt. = left

*** = significant at 0.001% level ($P < 0.001$)

Figure 15. Composite graph of mean environmental temperatures observed at ten minute intervals under control, al, ex and alex treatments for the ten subject sample.


MEAN ENVIRONMENTAL TEMPERATURE [°C] 102a
40.0
35.0
30.0
25.0
20.0
15.0
10.0
control
ex
al
alex
10
20
30
40
50
60
70
80
90
100
110
120
130
140
150
160
170
180
TIME MINUTES

between mean skin temperatures at the sternum site under control treatment, and mean environmental temperatures under control treatment.

Table XXXVIII. Results of a correlation performed between mean skin temperatures at the sternum skin site under control treatment, and mean environmental temperatures under control treatment

Correlates	r	Significance
sternum mean skin temperatures under control treatment		
versus	0.994	***
mean environmental temperatures under control treatment		

*** = significant at the 0.001% level ($P < 0.001$)

From Table XXXVIII it is evident that mean skin temperatures at the sternum site were significantly correlated to mean environmental temperatures. Since no significant differences in mean skin temperatures existed amoung the treatments at any skin site, and all four skin sites covaried with one and another under identical treatments, the results of Table XXXVIII may be considered as a general test of correlation between mean environmental temperatures and mean skin temperatures for all skin sites and treatments.

In summary, the results of the analysis of mean skin temperatures showed that: a) neither ethanol nor exercise altered mean skin tempera-

tures in any significant way, and b) skin temperatures of subjects were primarily determined by the environmental temperatures to which they were exposed.

V. Heart Rate

Figure 16 presents a composite graph of mean heart rates observed at ten minute intervals in the ten subject sample for control, al, ex and alex treatments.[10] From Figure 16 it can be seen that heart rate displayed considerable variation amoung treatments. In order to test if continuous treatment related differences in heart rate existed throughout experiments, the original "raw score" data used to calculate the means of Figure 16 was subjected to an overall three-factor anova. The results of this anova appear in Table XXXIX.

Table XXXIX shows that while treatments as a main effect were not significant, a significant time x treatments interaction did exist. Such results might be expected if significant treatment effects were not present throughout the entire experimental period but were restricted only to certain times during experiments. It was decided, therefore, that significant treatment differences would be tested for, in selected time isolates of Figure 16 which exhibited the greatest apparent variation between treatments. The results of this two-factor anova series appear in Table XL.

From Table XL it is evident that significant treatment related differences in heart rate were limited to the actual cold water immersion period of experiments. In order to determine which treatments differed during the significant time isolates of Table XL, further two-factor anovas were performed between pairs of treatments for those times. The results of this analysis appear in Table XLI.

10 Appendix 24 presents the original single treatment graphs used to construct Figure 16.

Figure 16. Composite graph of mean heart rates observed at ten minute intervals under control, al, ex and alex treatments for the ten subject sample.
(STAB = stabilization period of experiments,
DR = drinking period, ABSORPTION = absorption period,
IMMERSION = cold-water immersion period,
REWARM = rewarming period.)

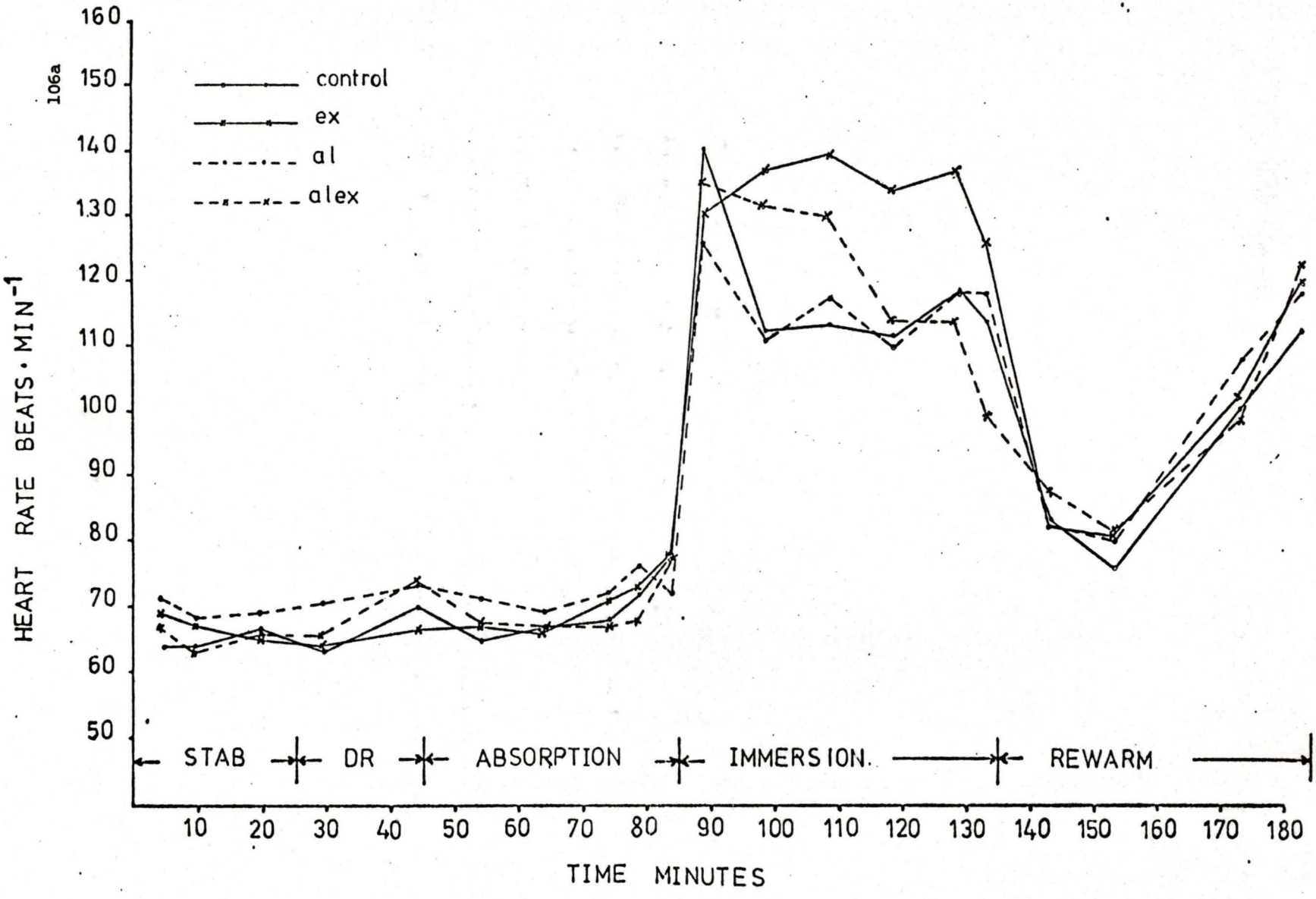

control
ex
al
alex
HEART RATE BEATS · MIN⁻¹
160
150
140
130
120
110
100
90
80
70
60
50
STAB
DR
ABSORPTION
IMMERSION.
REWARM
10
20
30
40
50
60
70
80
90
100
110
120
130
140
150
160
170
180
TIME MINUTES

Table XXXIX. Results of overall three-factor anova performed on "raw score" heart rate data

Source of variation	f value	Probability value	Significance
Main effects:			
Treatments	1.779	0.151	ns
Time	251.545	0.000	***
People	15.846	0.000	***
Interactions:			
Time x treatments	2.435	0.000	***
Time x people	1.828	0.000	***
Treatments x people	3.087	0.000	***

ns = not significant at 0.05% level ($P > 0.05$)

*** = significant at 0.001% level ($P < 0.001$)

Table XL. Results of two-factor anova series performed on "raw score" data of heart rate for various time isolates

Time isolate, minutes	Source of variation	f value	Probability value	Sig.
0	treatments	1.459	0.248	ns
	people	3.704	0.004	**
45	treatments	1.379	0.270	ns
	people	3.139	0.010	**
90	treatments	2.188	0.113	ns
	people	0.861	0.570	ns
100	treatments	6.372	0.002	**
	people	1.437	0.222	ns
110	treatments	3.752	0.023	*
	people	1.016	0.453	ns
120	treatments	3.744	0.023	*
	people	2.920	0.015	*
130	treatments	3.886	0.020	*
	people	4.069	0.002	**
135	treatments	1.758	0.179	ns
	people	4.996	0.001	***
145	treatments	0.499	0.686	ns
	people	2.351	0.042	*
155	treatments	0.753	0.530	ns
	people	2.403	0.038	*
185	treatments	1.566	0.221	ns
	people	1.455	0.215	ns

Sig. = significance

ns = not significant at 0.05% level ($P > 0.05$)

* = significant at 0.05% level ($P < 0.05$)

** = significant at 0.01% level ($P < 0.01$)

*** = significant at 0.001% level ($P < 0.001$)

Table XLI. Results of two-factor anova series performed between pairs of treatments on heart rate data for the significant time isolates of Table XL.

Time isolate, minutes	Test conditions	Source of var.	f value	Prob. value	Sig.
100	control vs al	treatment	0.024	0.881	ns
		people	1.046	0.474	ns
	control vs ex	treatment	5.769	0.040	*
		people	0.417	0.895	ns
	control vs alex	treatment	11.784	0.007	**
		people	3.307	0.045	*
	al vs ex	treatment	22.621	0.001	***
		people	2.390	0.105	ns
	al vs alex	treatment	9.960	0.012	*
		people	1.224	0.389	ns
	ex vs alex	treatment	0.474	0.508	ns
		people	1.281	0.359	ns
110	control vs al	treatment	0.114	0.743	ns
		people	0.721	0.683	ns
	control vs ex	treatment	9.878	0.012	*
		people	0.642	0.741	ns
	control vs alex	treatment	4.937	0.053	ns
		people	1.861	0.184	ns
	al vs ex	treatment	7.713	0.021	*
		people	1.316	0.344	ns
	al vs alex	treatment	1.134	0.315	ns
		people	0.343	0.937	ns
	ex vs alex	treatment	2.056	0.185	ns
		people	0.965	0.521	ns
120	control vs al	treatment	0.039	0.848	ns
		people	1.499	0.278	ns
	control vs ex	treatment	7.252	0.025	*
		people	1.640	0.236	ns
	control vs alex	treatment	0.252	0.628	ns
		people	6.833	0.004	**

	al vs ex	treatment	10.869	0.009	**
		people	1.458	0.292	ns
	al vs alex	treatment	0.257	0.624	ns
		people	1.377	0.321	ns
	ex vs alex	treatment	6.509	0.031	*
		people	1.682	0.225	ns
130	control vs al	treatment	0.001	0.979	ns
		people	3.250	0.047	*
	control vs ex	treatment	5.086	0.050	*
		people	1.145	0.422	ns
	control vs alex	treatment	0.417	0.535	ns
		people	7.652	0.003	**
	al vs ex	treatment	8.245	0.018	*
		people	1.723	0.215	ns
	al vs alex	treatment	0.194	0.670	ns
		people	2.968	0.060	ns
	ex vs alex	treatment	7.787	0.021	*
		people	1.319	0.343	ns

Prob. = probability

var. = variation

Sig. = significance

vs = versus

ns = not significant at 0.05% level (P > 0.05)

* = significant at 0.05% level (P < 0.05)

** = significant at 0.01% level (P < 0.01)

*** = significant at 0.001% level (P < 0.001)

From Table XLI and Figure 16, it can be seen that:

a) at 100 minutes, both ex and alex conditions produced equivalent heart rates which were significantly higher than those seen under control and al treatments

b) between 110 and 130 minutes, only the ex treatment produced heart rates which were significantly higher than those seen under control, al and alex treatments. Figure 16 shows that during this period, mean heart rates under alex treatment actually declined to levels equivalent to those of control and al conditions.

c) throughout the entire immersion period heart rates under control and al treatments were not found to differ significantly.

In summary, the results of the analysis of heart rates indicate that: a) following the termination of performing exercise, ethanol acted to lower heart rates which otherwise would have remained elevated as a consequence of exercise treatments and b) in the absence of exercise, ethanol was not seen to produce any significant alterations in heart rates whatsoever, throughout the cold exposure period.

VI. Blood Pressure

Figure 17 presents a composite graph of mean systolic, dystolic and pulse pressures observed in the ten subject sample under contol, al, ex and alex treatments.[11] In order to determine if any treatment related differences in mean blood pressures existed for the times of Figure 17, a series of t-tests were performed between the control and al, ex and alex treatments. The results of this series are given in Table XLII.

According to Table XLII, at 80 minutes mean systolic pressures under control and alex treatments differed. Figure 17 shows that at this time, mean systolic pressure under alex treatment was some eight mmHg below that seen for control conditions. Table XLII also shows that at this time control and al treatments did not differ in regards to mean systolic pressures. Furthermore, at 80 minutes, not only were the al and alex treatments simple replicates of one and another, since no exercise had yet been performed under alex treatment, but as Table III shows, mean BAC was approximately 10 mgm% higher under al than alex treatments. It would seem unlikely, therefore, that the lower mean systolic pressure seen under alex treatment actually represented a true ethanol-induced effect.

Having determined that no significant treatment-induced differences existed within any time isolate for mean blood pressure data, it was decided that treatment-induced differences in this data throughout time would be tested. This was accomplished by using t-tests to determine within identical treatments, any significant differences in

11 Appendix 25 presents the original single treatment graphs used to construct Figure 17.

Figure 17. Composite graph of mean; systolic, dystolic and pulse blood pressures observed at the selected times of Table XLII under control, al, ex and alex treatments for the ten subject sample.
(STAB = stabilization period of experiments,
DR = drinking period, ABSORPTION = absorption period,
IMMERSION = cold-water immersion period,
REWARM = rewarming period.)

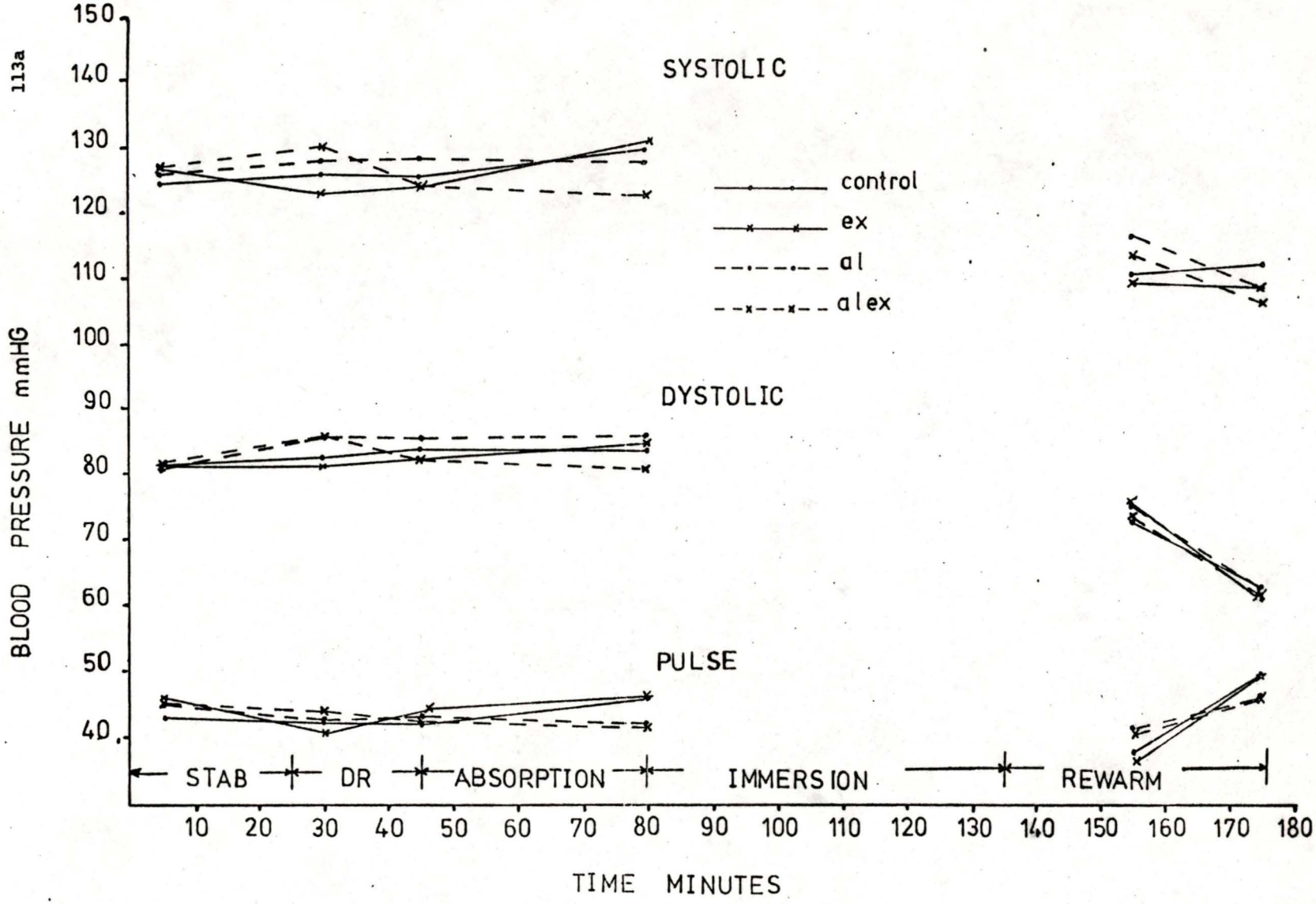

150
140
130
120
110
100
90
80
70
60
50
40
BLOOD PRESSURE mmHG
SYSTOLIC
DYSTOLIC
PULSE
control
ex
al
alex
STAB
DR
ABSORPTION
IMMERSION
REWARM
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES

Table XLII. Results of t-test series performed on mean blood pressure data between the control and al, ex, and alex treatments for various time isolates of experiments

Mean blood pressure	Time isolate, minutes	Test conditions	df	t's	Significance
Systolic	5	control vs al	18	-0.400	ns
		control vs ex	18	-0.524	ns
		control vs alex	18	-0.504	ns
	30	control vs al	18	-0.625	ns
		control vs ex	18	1.202	ns
		control vs alex	18	-1.236	ns
	45	control vs al	18	-0.567	ns
		control vs ex	18	0.582	ns
		control vs alex	18	1.139	ns
	80	control vs al	18	-0.586	ns
		control vs ex	18	-0.192	ns
		control vs alex	18	2.334	*
	155	control vs al	18	-1.368	ns
		control vs ex	18	0.323	ns
		control vs alex	18	-1.069	ns
	175	control vs al	16	1.285	ns
		control vs ex	16	1.227	ns
		control vs alex	16	2.033	ns
Dystolic	5	control vs al	18	0.282	ns
		control vs ex	18	0.194	ns
		control vs alex	18	0.002	ns
	30	control vs al	18	-1.636	ns
		control vs ex	18	0.517	ns
		control vs alex	18	-1.756	ns
	45	control vs al	18	-0.632	ns
		control vs ex	18	1.342	ns
		control vs alex	18	0.586	ns
	80	control vs al	18	-0.459	ns
		control vs ex	18	-0.162	ns
		control vs alex	18	0.925	ns

	155	control vs al	18	-0.328	ns
		control vs ex	18	-0.299	ns
		control vs alex	18	0.037	ns
	175	control vs al	16	0.114	ns
		control vs ex	16	0.508	ns
		control vs alex	16	0.375	ns
Pulse	5	control vs al	18	-0.673	ns
		control vs ex	18	-1.216	ns
		control vs alex	18	-0.675	ns
	30	control vs al	18	-0.071	ns
		control vs ex	18	0.816	ns
		control vs alex	18	-0.683	ns
	45	control vs al	18	-0.145	ns
		control vs ex	18	0.077	ns
		control vs alex	18	0.001	ns
	80	control vs al	18	1.375	ns
		control vs ex	18	-0.062	ns
		control vs alex	18	1.575	ns
	155	control vs al	18	-0.954	ns
		control vs ex	18	0.482	ns
		control vs alex	18	-1.058	ns
	175	control vs al	16	0.737	ns
		control vs ex	16	0.122	ns
		control vs alex	16	0.972	ns

---

vs = versus

ns = not significant at 0.05% level ($P > 0.05$)

* = significant at 0.05% level ($P < 0.05$)

mean blood pressures between time isolate pairs of Figure 17. The time isolates chosen for testing represented the beginning and end of an experimental time period (e.g. 5 to 80 minutes - pre-immersion period of experiments). The results of this t-test series appear in Table XLIII.

From Table XLIII and Figure 17 it can be seen that for:

a) All three mean blood pressures,

i) no treatment produced any significant alteration in mean blood pressure measures throughout the pre-immersion period (5 to 80 minutes) of experiments.

b) Mean systolic pressures,

i) for both determinations made during rewarming (155 to 175 minutes) mean systolic pressures under all treatments were found to be significantly reduced when compared to pressures seen immediately prior to cold water immersion (80 minutes).

ii) during the actual period of rewarming (155 to 175 minutes) mean systolic pressures were not found to change under either control or ex treatments. However under alex treatments, mean systolic pressure was found to show a further significant fall in pressure level. Under al treatment, no significant change in mean systolic pressure could be shown at the 0.05% probability level. However, significance was found at the 0.1% level.

c) Mean dystolic pressures,

i) lower mean dystolic pressures at 155 and 175 minutes than those at 80 minutes were found for all treatments tested.

ii) between 155 and 175 minutes, both ex and alex treatments displayed a significant decrease in mean dystolic pressures. No significant changes at the 0.05% level were found for either the al or

Table XLIII. Results of t-test series performed on mean blood pressure data, between results for different time isolates, within identical treatments

Mean blood pressure	Time isolates, minutes	Treatments compaired	df	t's	Significance
Systolic	5 - 80	con. to con.	18	-1.516	ns
		al to al	18	-0.452	ns
		ex to ex	18	-1.394	ns
		alex to alex	18	1.315	ns
	80 - 155	con. to con.	17	7.288	***
		al to al	17	2.437	*
		ex to ex	17	6.645	***
		alex to alex	17	3.255	**
	80 - 175	con. to con.	16	5.609	**
		al to al	16	5.506	**
		ex to ex	16	6.637	***
		alex to alex	16	5.486	**
	155 - 175	con. to con.	16	-0.699	ns
		al to al	16	1.767	†
		ex to ex	16	0.414	ns
		alex to alex	16	2.481	*
Dystolic	5 - 80	con. to con.	18	-0.944	ns
		al to al	18	-1.439	ns
		ex to ex	18	-1.618	ns
		alex to alex	18	0.229	ns
	80 - 155	con. to con.	17	3.666	**
		al to al	17	2.117	*
		ex to ex	17	3.694	**
		alex to alex	17	2.689	*
	80 - 175	con. to con.	17	4.260	**
		al to al	17	4.816	**
		ex to ex	17	5.019	**
		alex to alex	17	7.245	***
	155 - 175	con. to con.	16	1.973	†
		al to al	16	1.996	†
		ex to ex	16	2.745	*
		alex to alex	16	6.443	**

Pulse	5 - 80	con. to con.	18	-1.049	ns
		al to al	18	0.996	ns
		ex to ex	18	-0.219	ns
		alex to alex	18	1.259	ns
	80 - 155	con. to con.	17	3.011	**
		al to al	17	0.542	ns
		ex to ex	17	2.989	**
		alex to alex	17	0.645	ns
	80 - 175	con. to con.	17	-0.679	ns
		al to al	17	-1.022	ns
		ex to ex	17	-0.489	ns
		alex to alex	17	-1.309	ns
	155 - 175	con. to con.	16	-2.519	*
		al to al	16	-1.359	ns
		ex to ex	16	-2.951	**
		alex to alex	16	-1.419	ns

---

con. = control

ns = not significant at 0.05% level ($P > 0.05$)

* = significant at 0.05% level ($P < 0.05$)

** = significant at 0.01% level ($P < 0.01$)

*** = significant at 0.001% level ($P < 0.001$)

† = not significant at 0.05% level, but was significant at 0.1% level ($0.1 > P > 0.05$)

control treatments. However, a significant decrease in mean dystolic pressure was found for these treatments at the 0.1% probability level.

In general then, all treatments exhibited a progressive decrease in mean dystolic pressures during rewarming.

d) Mean pulse pressures

i) between 80 and 155 minutes, the control and ex conditions displayed a general decrease in mean pulse pressures. During this period both al and alex treatments produced no such significant alterations in mean pulse pressures.

ii) between 80 and 175 minutes, no treatment produced any significant change in mean pulse pressures.

iii) between 155 and 175 minutes, both al and alex treatments were found to show no significant changes in mean pulse pressures. Under control and ex treatments, mean pulse pressures were seen to significantly increase.

To summarize, the results of the analysis on blood pressures show that:

a) no treatment produced any changes in any mean blood pressure during the pre-immersion period

b) both mean systolic and mean dystolic pressures were seen to decrease approximately fourteen and eight mmHg, respectively, as a consequence of cold exposure. Ethanol consumption prior to cold water immersions,performed with or without exercise, produced a further decrease of approximately eight mmHg in mean systolic pressure during rewarming.

c) mean dystolic pressures were seen to show a significant decrease of approximately 12 mmHg under all treatments at the termination of

experiments as a consequence of rewarming

d) mean pulse pressures were found to fall between 80 and 155 minutes only under control and ex treatments. However, the decrease in mean pulse pressures produced under these treatments was transitory, since by 175 minutes mean pulse pressures were seen to have increased back to levels equivalent to those seen at 80 minutes. Thus throughout rewarming, ethanol consumption, with or without exercise, was not found to cause any prolonged alterations in mean pulse pressures.

VII. Urine

Table XLIV presents the means and standard deviations of urine pH and total volumes seen for the ten subject sample under control, al, ex and alex treatments.[12] The data of Table XLIV were graphed and appears as Figures 18 and 19. From Figures 18 and 19, it can be seen that within any single urine sample apparent treatment-related differences in mean pH and total volume existed. However, Table XLIV shows that for all three samples, mean variance under all treatments was extremely large, suggesting that perhaps the observed treatment-related differences seen in Figures 18 and 19 were not actually significant. Therefore, a series of t-tests between pairs of treatments were completed for both variables for each of the three urine samples. The results appear in Table XLV.

Table XLV shows that with the exception of mean total volumes seen under al and ex treatments in the third sample, no significant differences existed between treatments for either mean pH or total volume within any urine sample.

Figures 18 and 19 also show that mean pH continually decreased and mean total volume steadily increased under all treatments throughout the course of experiments. In order to test the significance of the aforementioned observations regarding mean pH decrease and mean total volume increase, a series of t-tests were performed between different urine samples for identical treatments. The results of this analysis appears in Table XLVI.

12 Mean levels of both urine glucose and protein were not found to significantly vary from a level of 0% under control, al, ex or alex treatments.

Figure 18. Composite graph of mean pH determined for the three urine samples obtained in control, al, ex and alex experiments.

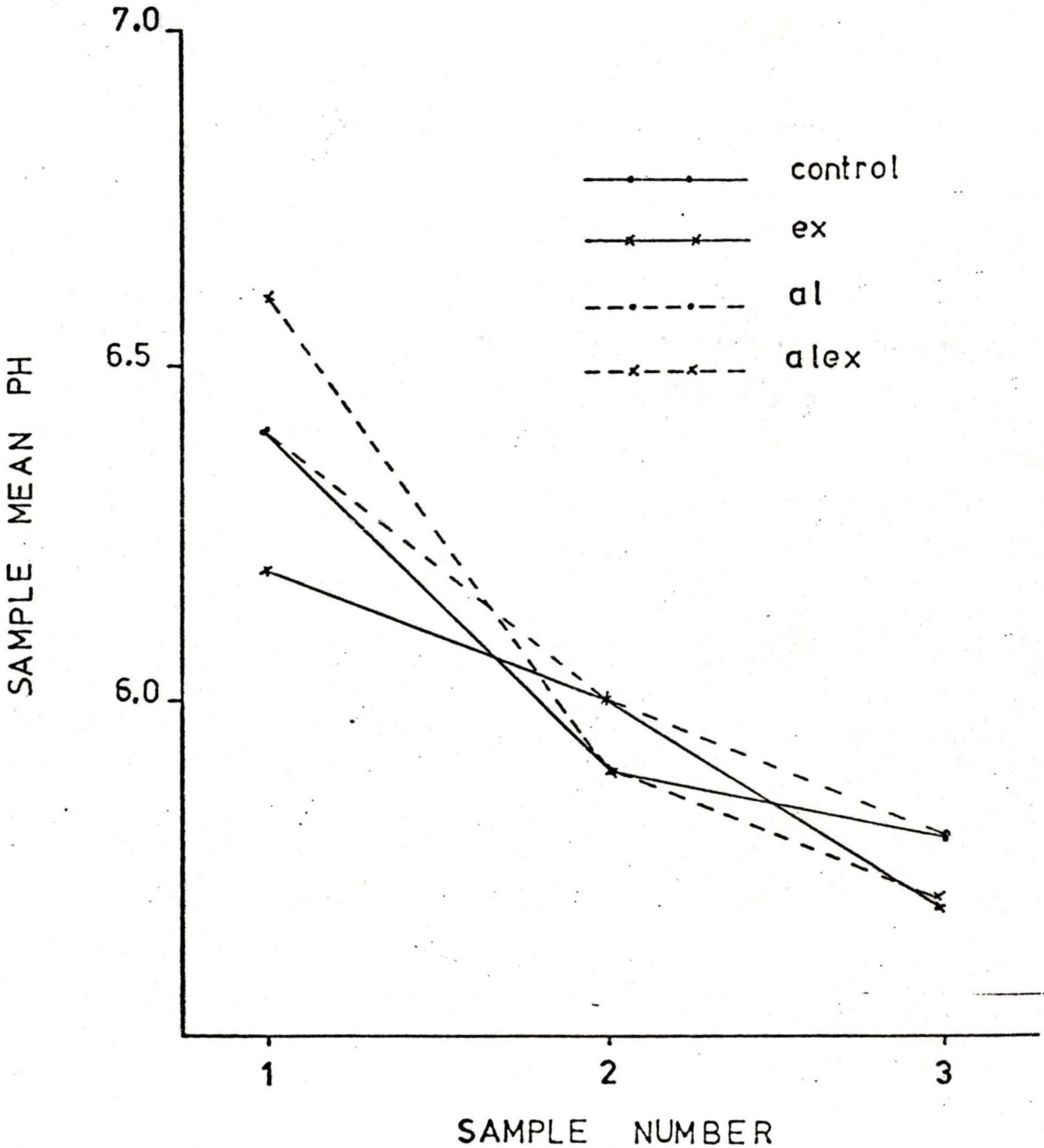

7.0
6.5
6.0
SAMPLE MEAN PH
control
ex
al
alex
1
2
3
SAMPLE NUMBER

Figure 19. Composite graph of mean total volumes of urine excreted determined for the three urine samples obtained in control, al, ex and alex experiments.

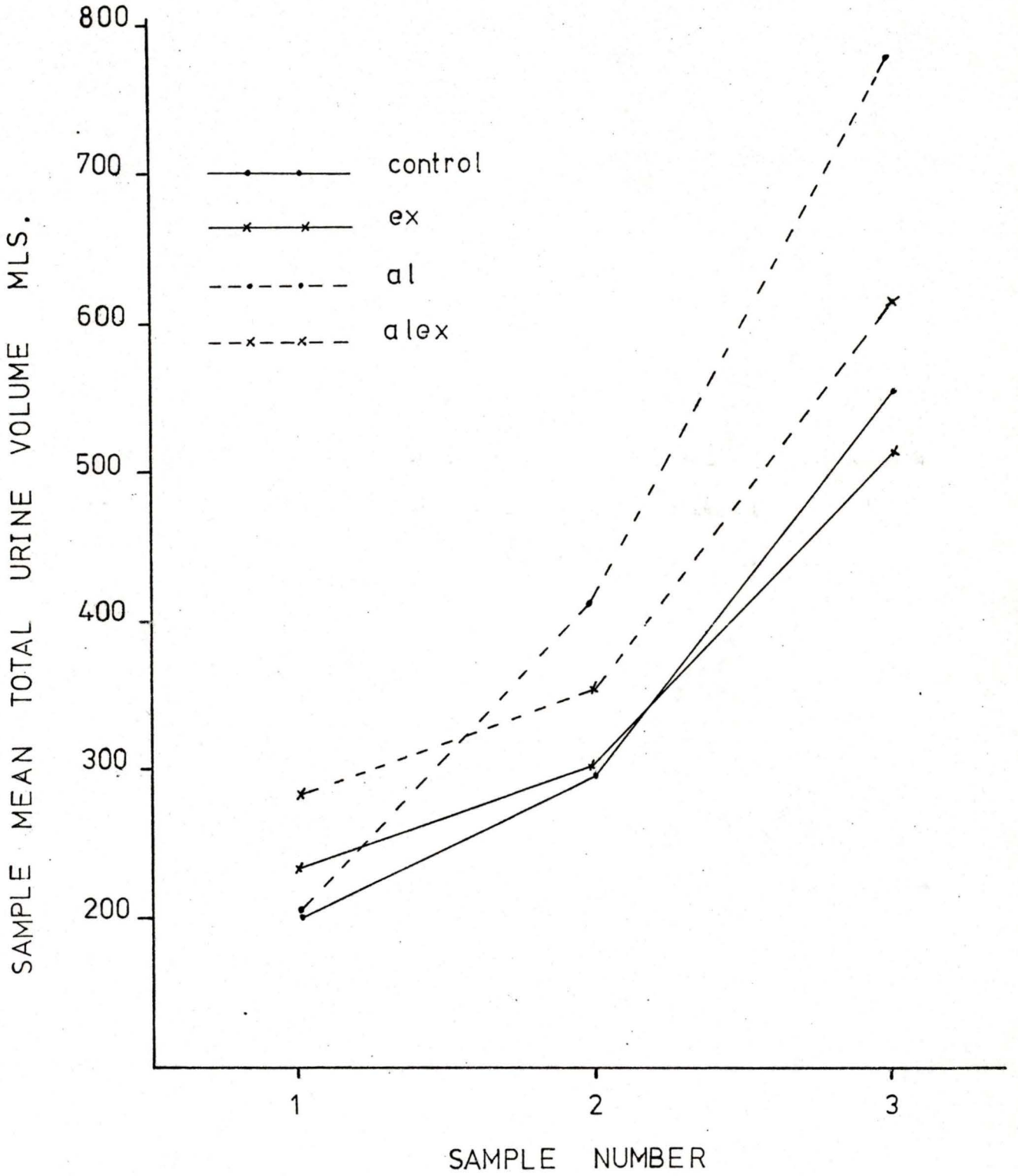

800
700
600
500
400
300
200
control
ex
al
alex
SAMPLE MEAN TOTAL URINE VOLUME MLS.
1
2
3
SAMPLE NUMBER

Table XLIV. Means and standard deviations determined for urine pH and total volume for the three urine samples taken in control, al, ex, and alex experiments

Sample number	Time of sample, minutes	Treatment	n	Mean	Standard deviation
A. Urine pH:					
1	0	control	8	6.4	0.74
		al	9	6.4	0.33
		ex	10	6.2	0.63
		alex	10	6.6	0.44
2	85	control	7	5.9	0.73
		al	9	6.0	0.71
		ex	8	6.0	0.46
		alex	8	5.9	0.42
3	160	control	10	5.8	0.42
		al	10	5.8	0.43
		ex	10	5.7	0.26
		alex	10	5.7	0.29
B. Urine total volume:					
1	0	control	8	201.0	76.4
		al	9	203.0	128.3
		ex	9	235.0	179.9
		alex	10	283.0	195.6
2	85	control	7	296.0	213.9
		al	9	414.0	191.2
		ex	8	303.0	181.4
		alex	8	354.0	215.7
3	160	control	10	553.0	231.6
		al	10	776.0	287.1
		ex	10	512.0	262.0
		alex	10	607.0	309.0

Table XLV. Results of t-test series performed between pairs of treatments on urine mean pH and urine mean total volume data, for the three urine samples of Table XLIV.

Sample number	Test conditions	df	t's	Significance
A. Urine mean pH:				
1	control vs al	15	0.000	ns
	control vs ex	15	0.602	ns
	control vs alex	15	-0.713	ns
	al vs ex	17	0.855	ns
	al vs alex	17	-1.111	ns
	ex vs alex	18	-1.657	ns
2	control vs al	15	-0.277	ns
	control vs ex	15	-0.321	ns
	control vs alex	15	0.000	ns
	al vs ex	12	0.001	ns
	al vs alex	12	0.287	ns
	ex vs alex	11	0.392	ns
3	control vs al	15	0.002	ns
	control vs ex	15	0.636	ns
	control vs alex	15	0.633	ns
	al vs ex	18	0.867	ns
	al vs alex	18	0.861	ns
	ex vs alex	18	0.000	
B. Urine mean total volume:				
1	control vs al	15	-0.041	ns
	control vs ex	15	1.413	ns
	control vs alex	15	-1.237	ns
	al vs ex	17	0.442	ns
	al vs alex	17	-1.041	ns
	ex vs alex	18	-0.571	ns
2	control vs al	15	-1.165	ns
	control vs ex	15	1.385	ns
	control vs alex	15	-0.462	ns
	al vs ex	17	1.263	ns
	al vs alex	17	-1.041	ns
	ex vs alex	11	-0.460	ns
3	control vs al	15	1.533	ns
	control vs ex	15	0.370	ns
	control vs alex	15	-0.370	ns
	al vs ex	18	2.146	ns
	al vs alex	18	1.267	ns
	ex vs alex	18	-0.741	ns

ns = not significant at 0.05% level (P > 0.05)

vs = versus

* = significant at 0.05% level (P < 0.05)

Table XLVI. Results of t-test series performed between different urine samples within identical treatments using data of Table XLIV.

Treatments tested	Test conditions	df	t's	Sig.
A. Urine mean pH:				
control to control	sam. 1 vs sam. 2	13	1.309	ns
	sam. 1 vs sam. 3	16	2.158	*
	sam. 2 vs sam. 3	15	0.357	ns
al to al	sam. 1 vs sam. 2	16	1.535	ns
	sam. 1 vs sam. 3	17	4.416	**
	sam. 2 vs sam. 3	17	0.802	ns
ex to ex	sam. 1 vs sam. 2	16	0.753	ns
	sam. 1 vs sam. 3	18	2.336	*
	sam. 2 vs sam. 3	16	1.747	ns
alex to alex	sam. 1 vs sam. 2	13	1.960	ns
	sam. 1 vs sam. 3	18	5.603	**
	sam. 2 vs sam. 3	13	1.156	ns
B. Urine mean total volume excreted:				
control vs control	sam. 1 vs sam. 2	13	-1.129	ns
	sam. 1 vs sam. 3	16	-4.565	**
	sam. 2 vs sam. 3	15	-2.121	ns
al to al	sam. 1 vs sam. 2	16	-2.749	*
	sam. 1 vs sam. 3	17	-5.710	**
	sam. 2 vs sam. 3	17	-3.194	**
ex to ex	sam. 1 vs sam. 2	16	-0.794	ns
	sam. 1 vs sam. 3	18	-2.752	*
	sam. 2 vs sam. 3	18	-2.071	ns
alex to alex	sam. 1 vs sam. 2	13	-0.642	ns
	sam. 1 vs sam. 3	18	-2.802	*
	sam. 2 vs sam. 3	13	-1.629	ns

Sig. = significance

sam. = sample

vs = versus

ns = not significant at 0.05% level ($P > 0.05$)

* = significant at 0.05% level ($P < 0.05$)

** = significant at 0.01% level ($P < 0.01$)

Table XLVI and Figures 18 and 19 show that for:

a) Mean pH, ◦

i) all treatments showed a non-significant decrease between the first and second samples.

ii) all treatments exhibited a further non-significant decrease between the second and third samples.

iii) all treatments displayed a significant decrease between the first and third samples.

b) Mean total volume,

i) control, alex and ex treatments showed a non-significant increase between the first and second samples.

ii) control, alex and ex treatments exhibited a further non-significant increase between the second and third samples.

iii) all treatments displayed a significant increase between the first and third samples.

iv) al treatment showed a significant increase between the first and second, and second and third samples.

Thus the results of the analysis on mean pH and total volume indicate that:

a) the consumption of ethanol or the performance of exercise did not significantly alter mean pH levels which were found to decrease in all treatments as a result of cold exposure

b) the performance of exercise did not alter the levels of urine mean total volume excreted, characteristically seen under all treatments to be increased as a result of cold exposure

c) the consumption of ethanol acted to increase further the total volumes of urine excreted as a consequence of cold water immersion.

VIII. Placebo Data

As previously mentioned in the methods section, placebo type immersions were completed only by subjects SC, DP and JS. In order to determine if placebo type treatments produced any significant physiological effects, t-test analysis was performed for each of the physiological variables studied. These t-tests determined significance by comparing the mean results seen under pl and plex treatments with the mean results for the above three subjects obtained under control and ex conditions. The results of the t-tests performed are presented as tables in the following sub-sections.

A. Mean Core Temperatures (Ttym and Trec)

Table XLVII presents the results of t-test analysis between placebo and control type treatments for mean core temperatures. This table shows that mean core temperatures under pl and plex treatments did not exhibit any significant differences from mean core temperatures seen under control and ex conditions for the time isolates tested.

B. Mean Metabolic Rates

Table XLVIII presents the results of the analysis between placebo and control type treatments for mean metabolic rates. From Table XLVIII no significant changes in mean metabolic rates were found for the times tested between either pl and control or plex and ex treatments.

C. Mean Heart Rates

Table XLIX presents the results of t-test analysis between placebo and control type conditions for mean heart rates. From Table XLIX it can be seen that mean heart rates at the times tested, did not significantly differ between either pl and control or plex and ex treatments.

Table XLVII. Results of t-test series performed on mean core temperatures between, pl and control and plex and ex treatments

Time isolates compared, minutes	Treatments tested			
	pl versus control		plex versus ex	
	t's	Significance	t's	Significance
A. Tympanic temperatures:				
0	0.211	ns	-0.329	ns
20	0.000	ns	0.000	ns
25	0.000	ns	0.474	ns
45	0.000	ns	0.200	ns
55	0.000	ns	0.000	ns
85	1.334	ns	0.203	ns
90	1.344	ns	0.802	ns
110	1.213	ns	-0.567	ns
135	0.599	ns	-0.944	ns
140	0.443	ns	-0.378	ns
160	0.444	ns	-0.533	ns
180	0.230	ns	-0.081	ns
B. Rectal temperatures:				
0	-0.606	ns	0.006	ns
20	-0.518	ns	-0.552	ns
25	-0.376	ns	-0.944	ns
45	-0.524	ns	-1.162	ns
55	0.004	ns	-1.161	ns
85	-0.507	ns	-0.866	ns
90	-0.507	ns	-1.252	ns
110	-0.008	ns	-0.539	ns
135	1.489	ns	-0.104	ns
140	1.214	ns	-0.236	ns
160	0.772	ns	-0.903	ns
180	0.073	ns	-0.301	ns

degrees of freedom for all tests = 4

ns = not significant at 0.05% level ($P > 0.05$)

Table XLVIII. Results of t-test series performed on mean metabolic rate data, between pl and control and plex and ex treatments

Treatments tested	Time isolates compared, minutes	df	t's	Significance
pl vs control	10	4	-0.744	ns
	20	4	-0.601	ns
	45	3	1.258	ns
	60	4	-0.449	ns
	80	4	-0.090	ns
	90	3	-0.051	ns
	100	4	-2.069	ns
	120	4	0.734	ns
	130	4	0.133	ns
	140	4	0.500	ns
	160	3	0.091	ns
	180	3	0.064	ns
plex vs ex	10	4	1.765	ns
	20	4	1.353	ns
	45	3	0.706	ns
	60	4	1.158	ns
	80	4	0.324	ns
	90	4	0.116	ns
	100	4	0.369	ns
	120	4	0.088	ns
	130	4	1.437	ns
	140	4	0.589	ns
	160	4	-0.825	ns
	180	3	-0.284	ns

vs = versus

ns = not significant at o.o5% level ($P > 0.05$)

Table XLIX. Results of t-test series performed on mean heart rate data, between pl and control and plex and ex treatments

Treatments tested	Time isolates compared minutes	t's	Significance
pl vs control	0	1.343	ns
	20	-1.222	ns
	45	0.337	ns
	65	2.372	ns
	80	-1.385	ns
	90	-0.762	ns
	110	0.635	ns
	120	-0.621	ns
	145	2.245	ns
	155	0.385	ns
	175	1.859	ns
	185	1.655	ns
plex vs ex	0	-0.433	ns
	20	-0.572	ns
	45	-0.522	ns
	65	-0.379	ns
	80	-1.093	ns
	90	0.900	ns
	110	0.005	ns
	120	-0.341	ns
	145	0.720	ns
	155	-0.729	ns
	175	0.214	ns
	185	0.137	ns

degrees of freedom for all tests = 4

vs = versus

ns = not significant at 0.05% level ($P > 0.05$)

D. Mean Blood Pressures

Table L presents the results of the analysis performed on mean systolic, dystolic and pulse pressures between placebo and control type conditions. This table shows that no difference in mean systolic, dystolic or pulse pressures were found between pl and control or plex and ex treatments.

E. Urine Mean pH and Mean Total Volume

Table LI gives the results of the analysis done on urine data between placebo and control treatments. From Table LI it is evident that mean pH and mean total volumes did not significantly differ under either pl and control or plex and ex conditions.

In summary, the results of the analysis performed between placebo and control treatments show that placebo treatment, with or without exercise, produced no significant alterations for any of the physiological variables studied.

Table L. Results of t-test series performed on data of mean blood pressures, between pl and control and plex and ex treatments

Mean blood pressure	Treatments tested	Time isolates compared minutes	t's	Significance
Systolic	pl vs control	5	-0.671	ns
		30	-0.319	ns
		45	-1.964	ns
		80	-0.761	ns
		175	1.343	ns
	plex vs ex	5	1.601	ns
		30	1.669	ns
		45	-1.342	ns
		80	0.744	ns
		155	-0.150	ns
		175	0.954	ns
Dystolic	pl vs control	5	0.728	ns
		30	1.105	ns
		45	0.905	ns
		80	1.059	ns
		175	1.358	ns
	plex vs ex	5	0.214	ns
		30	0.262	ns
		45	-1.342	ns
		80	1.406	ns
		155	-0.340	ns
		175	0.001	ns
Pulse	pl vs control	5	-0.926	ns
		30	-1.750	ns
		45	-1.367	ns
		80	-1.471	ns
		175	0.253	ns
	plex vs ex	5	1.984	ns
		30	1.273	ns
		45	-1.701	ns
		80	-1.423	ns
		155	0.119	ns
		175	1.572	ns

degrees of freedom for all tests = 4

vs = versus

ns = not significant at 0.05% level ($P > 0.05$)

Table LI. Results of t-test series performed on urine mean pH and mean total volume data, between pl and control and plex and ex treatments

Urine metric	Treatments tested	Urine sample number	t's	Significance
mean pH	pl vs control	1	-0.160	ns
		2	0.000	ns
		3	0.000	ns
	plex vs ex	1	1.548	ns
		2	0.392	ns
		3	0.000	ns
mean total volume	pl vs control	1	0.569	ns
		2	0.559	ns
		3	0.359	ns
	plex vs ex	1	-0.010	ns
		2	1.235	ns
		3	0.633	ns

degrees of freedom for all tests = 4

vs = versus

ns = not significant at 0.05% level ($P > 0.05$)

Discussion

I. BAC

A. Significance of the differences found for mean BACs between al and alex treatments at times 115 and 140 minutes.

At times 115 and 140 minutes, mean BACs under alex treatment were found to be lower than those found under al treatment. Such a result might indicate that the exercise performed under alex treatment was responsible for the observed lower BACs. However, the following results would indicate that most probably exercise had no effect in lowering mean BACs of alex trials.

From Figure 1, it was seen that throughout the entire course of the experiments, mean BACs were consistently lower under alex than under al treatment. Additionally, Table V showed that during the cold-water immersion period of experiments, mean BAC decay rates were equivalent between the above treatments. Finally, Table IV showed that the levels of inter-personal variability were significantly different between the two treatments at both 115 and 140 minutes.

It would seem highly probable therefore, that the statistically significant differences observed between mean BACs under al and alex treatments, at the aforementioned times resulted as a consequence of a decrease in the "masking effect" of inter-personal variability. Evidently, the decreased level of overall mean variation in BAC brought about by a reduction in the level of inter-personal variation at times 115 and 140 minutes, allowed the statistical significance of the original differences in "starting" mean BACs under al and alex treatments to finally become visible.

B. Significance of the differences observed between experimental and expected mean "ethanol decay rates" during the immersion and the rewarming periods of alcohol trials.

Prior to discussing the significance of the mean "ethanol decay rates" found in this study, a brief review of the general physiological mechanisms by which the concentration of alcohol in the body is first established and subsequently reduced is required.

Following the cessation of ethanol absorption from the gut, ethanol in the blood and body tissues and fluids equilibrate with each other within a matter of minutes, partitioning on the basis of relative water content, (Kissin and Beglieter, 1971). Body tissues and fluids having a high relative water content (eg. kidney and bile) would therefore have a higher absolute ethanol concentration than those having a lower relative water content (eg. muscle and blood), (Levy, 1935; Van Hecke et al., 1951). Once equilibration has occurred, the BAC will begin to fall as a result of the oxidative processes going on in the liver, and the excretion of ethanol in the breath and urine. Passive diffusion of ethanol from the body tissues and fluids into the blood insures that the blood-tissue equilibrium is maintained. Thus, ethanol would be continually drawn from the tissues and biological fluids into the blood at a rate proportional to the rate at which ethanol was eliminated from the body.

Alcohol dehydrogenase (ADH) the enzyme responsible for ethanol oxidation, becomes saturated with its substrate at BACs in excess of 10 mgm%, (Kalant, 1971). ADH, therefore works at a maximal rate throughout the majority of the oxidation period, and as a result, BAC is found to decrease at a constant rate producing a linear "decay" curve, (Marshall

and Fritz, 1951; Lundquist and Wolhters, 1958). Thus, according to Kalant, (1971) the rate of fall (ie. the "decay" rate) of a BAC curve is dependant on two criteria; a) the rate of efflux from the blood to all other body tissues and fluids, and b) the rate of alcohol elimination from the body, brought about through alcohol oxidation and excretion. In the post-absorptive condition, alcohol efflux from the blood to other body tissues and fluids does not normally occur, since an alcohol-blood-tissue equilibrium has been previously established. For this reason, the rate of decay of a BAC curve is generally considered as indicative of the "ethanol decay rate" (ie. the actual rate of ethanol elimination from the body), (Gradwhol, 1954).

During the cold exposure period of experiments involving al and alex treatments, subjects exhibited mean ethanol decay rates which were approximately double those which would have been expected under normothermic conditions where the standard decay rate approximates 16 mgm% $\cdot$ $hour^{-1}$. Such greatly accelerated decay rates as those seen under the hypothermic conditions of this study have also been observed under normothermic conditions, and have been explained in the literature on the basis of personal variations.

For example, Harger and Forney, (1963) reported variations in ethanol decay rates for their subjects which ranged from a high of 34 mgm% $\cdot$ $hour^{-1}$ to a low of 10 mgm% $\cdot$ $hour^{-1}$. In the present study, the mean overall ethanol decay rate during the pre-immersion period, under al and alex treatment was found to equal 12.6 mgm% $\cdot$ $hour^{-1}$. This pre-immersion rate agreed well with a typical ethanol decay rate of 13 to 16 mgm% $\cdot$ $hour^{-1}$ found for healthy young men in a normothermic environment by Coldwell and Smith, (1959). It is highly probable therefore that the increased

ethanol decay rate observed under hypothermic conditions in the subjects of the present study, resulted as a consequence of some cold-stress-induced modification of an otherwise normal ethanol decay rate.

The general effects of cold exposure on the rate of ethanol oxidation has been studied in various animals. Unfortunately, the results of these studies are somewhat contradictory, both within the same and amoung differing species. For example, Platnow et al., (1963) reported that the rate of ethanol oxidation was increased up to 12% in non-cold acclimated rats exposed to ambient air temperatures of 1.0 ^{o}C. However, Dybing, (1945) and Kalant et al., (1965) found that the rate of ethanol oxidation was decreased in hypothermic rats. Fuhman, (1946) and Jaulmes et al., (1956) found that ethanol oxidation rates were reduced in rabbits immersed in cool water over prolonged periods of time (i.e. 12 hours in 29 ^{o}C water). MacGregor et al., (1965) on the other hand, found no significant alteration in the ethanol oxidation rates of dogs which had been rendered hypothermic by ice-water baths. Unfortunately, the only studies involving man, ethanol and hypothermia in which actual experimental BAC determinations were performed are those of Martin et al., (1977) and Hobson and Collis, (1977). Martin et al., (1977) found that overall mean BAC actually increased in their subjects during a 20 minute immersion in 13.6 ^{o}C water. In that study, the overall mean BAC of the subjects was found to equal 90 mgm% immediately prior to the cold-water immersion, and 20 minutes later at the termination of immersions, overall mean BAC was found to equal 99 mgm%. However, it should be noted that the 9.0 mgm% difference in overall mean BAC found between pre and post-immersion determinations was not statistically significant. Hobson and Collis, (1977) like Martin et al., (1977) did not report the actual

ethanol decay rates of their subjects. However, their paper did report the BACs of each subject both immediately prior to the immersion, and at the termination of the immersion, along with the length of time each subject was immersed. Utilizing that data, it has been calculated that the overall mean ethanol decay rate of Hobson and Collis' (1977) subjects equalled 16.16 mgm% $\cdot$ hour^{-1}, (see Appendix 26 for statistics). Since the normothermic ethanol decay rate is usually assumed to equal 16.0 mgm% $\cdot$ hour^{-1}, (Kalant, 1971), Hobson and Collis' study would suggest that ethanol oxidation rates are not altered in man at core (rectal) temperatures of 35.0 ^{o}C.

Although ethanol decay rates were found to be greater during the cold exposure period of experiments than those expected, this does not necessarily indicate that the actual rate of ethanol elimination was accelerated as a result of cold-water immersion. Usually, any change in the rate of decay of a BAC curve is assumed as indicative of some concomitant alteration of the actual rate of ethanol elimination. However, it is clear from Kalant's (1971) criteria that such an assumption is valid only if the blood-tissue-ethanol equilibrium has not been altered. From Kalant's criteria, it is evident that the observed increased rates of ethanol decay seen under hypothermia in this study may have resulted from either: a) some modification of the rate of ethanol elimination, (i.e. some change in the rate of ethanol oxidation or excretion) or b) some alteration in the rate of ethanol efflux from the blood to other body tissues and fluids, (i.e. some disturbance of the ethanol equilibrium).

Let us consider the case for an altered rate of ethanol elimination. Assuming for the sake of argument that hypothermia will produce no major haemodynamic alterations, and thus will not disturb the ethanol-blood

tissue equilibrium established prior to cold-water immersions, an increase in the rate of ethanol oxidation might be expected as a result of hypothermia's effects on enzyme kinetics. The rate-limiting step in ethanol oxidation is the rate at which ethanol is converted to acetaldehyde through the action of ADH, (Sardesai, 1969). ADH in addition to becoming saturated with its substrate at low BACs, seems to be immune to cold impairment, as Theorell and Bonnichsen, (1951) have found that the in vitro activity of this enzyme is unchanged even when ambient temperatures are reduced to 20 ^{o}C. Therefore, one would not expect to find any significant decrease in ADH activity as a result of low body temperatures. Most enzymes display some increased activity under a general increase in metabolic rate, (Lehninger, 1976). Both hypothermia and physical exercise are known to increase metabolic rate. Therefore, an increase in ADH activity might be expected as a result of either hypothermic treatment, or the performance of physical exercise.

The actual effects of an increase in metabolic rate, on the rate of ethanol oxidation is unclear in the alcohol literature. Older literature as reviewed by Newman (1941) has stated that an increase in general metabolic rate and body temperature, elicited by either radiothermy or treatment with dinitrophenol, will result in an increase by as much as 100% in the rate of ethanol decay in man. However, it has been shown that such increases in ethanol decay rates under the above conditions are actually due to an increased loss of ethanol from the lung site as a consequence of hyperventilation, rather that from an increased rate of ADH activity as was first believed, (Newman and Tainter, 1936).

Similarly, the effects of physical exercise on the rate of ethanol decay is contradictory in the literature. For example, Newman, (1941)

reported that increased physical activity had no effect on the rate of ethanol oxidation. However, Hecksteden and Fehler, (1942) found increases ranging between 31 and 83% in the rate of ethanol oxidation as a result of their subjects performing strenuous exercise. More recent literature states that an increase in either general metabolic rate, or the performance of physical exercise has no effect on the rate of ethanol oxidation in either dogs, (Loomis, 1950) or in man, (Barnes et al., 1965; Pawan, 1968). In the present study, no increase in the rate of ethanol decay was found under al and alex treatments, during cold-water immersions. During the actual period of the performance of exercise (90 to 110 minutes), the mean metabolic rates of the subjects were observed to approximately double under alex treatment. Evidently then, even a doubling of mean metabolic rate elicited by exercise had no effect whatsoever on the rate of ethanol decay, since no changes in the rate of decay were found between al and alex treatments throughout the immersion periods. Additionally, it will be remembered that experimentally determined BACs were not significantly different at the time of exit from the rewarming bath, than BACs expected at that time which were based on a normal rate of ethanol oxidation. If an actual increase in the rate of ethanol elimination had occurred as a result of cold exposure, it would be highly unlikely that BACs would have equaled expected levels on exit from the rewarming bath. Thus there is little evidence supporting an increase in the level of ethanol oxidation as an etiological explanation of the observed increased rate of ethanol decay found in cold-water immersions.

Returning to Kalant's (1971) criteria for a fall in the BAC curve, the increased decay rate observed during cold exposure might have resulted

from some transitory disturbance of the blood-tissue-ethanol equilibrium established prior to immersions. Hypothermia per se produces a chain of haemodynamic effects which conceivably could explain both the increase in ethanol decay evident during cold exposure, and the apparent cessation of ethanol decay found during rewarming. Sudden cold-water immersion results in a rapid onset of cutaneous vasoconstriction, (Keatinge, 1969). Often as a result of this rapid peripheral vasoconstriction, both whole blood and blood plasma becomes trapped in the constricted capillaries, (Rodbard et al., 1951; D'Armato and Hegnauer, 1953). Prior to the immersions, ethanol would have been distributed throughout the peripheral tissues. Thus, the entrapment of blood, and more importantly, the decreased rate of peripheral perfusion which would occur during hypothermia would have resulted in the production of an alcohol-rich reservoir in the body. Analogically speaking, hypothermia would have produced an "alcohol holding-tank" in the body.

Peripheral vasoconstriction also results in a rapid increase in the central blood pressure, (Keatinge, 1969). This increased pressure is physiologically compensated for by increased levels of urine production and excretion, coupled with massive extra-vascular shifts of fluid, (Lennquist, 1972). Since ethanol is distributed in the body on the basis of relative water content, any increase in the level of diuresis would be expected to produce a concomitant increase in the level of ethanol eliminated via the urine. However, although cold diuresis is generally believed to result from a cold induced impairment of the pituitary's release of Anti-Diuretic Hormone (ADHor), (Keatinge, 1969), recent studies have placed this long held view in some doubt. Lennquist, (1972) found not only did the level of free water clearance actually decrease during

cold induced diuresis, but therapeutic doses of ADHor had no long term success in the cessation of the effect. Therefore, any increase in ethanol elimination mediated by an increased level of diuresis would not be expected to greatly modify the overall ethanol decay rate found during cold exposure.

The ability of hypothermia to produce a state of hypovolemia, and altered blood flows to various organs would probably be of more importance in the production of an increased ethanol decay rate. That hypothermia is capable of producing hypovolemia is well known, (Conley and Nickerson, 1945; Adolph et al., 1946; Keatinge, 1969; Harari et al., 1975; Zarins and Skinner, 1975). Furthermore, Eliot et al., (1949) have shown that haemoconcentration will still occur during hypothermia, even if a cessation of cold induced diuresis is achieved through Pitressin injections. It would appear therefore, that hypothermia-induced hypovolemia results primarily from a massive shift of fluid from the circulatory system proper out into the interstitial space.

Ethanol present in the blood would also move with the fluid out into the interstitial space, with the net effect of this transfer being the production of a second alcohol-rich reservoir in the body. Although the blood would remain in equilibrium with this interstitial-ethanol reservoir, the total water content and therefore, the actual ethanol concentration of the blood would be reduced. Thus, hypovolemia would be expected to produce an overall reduction in absolute BAC.

In addition to the above effects of hypovolemia, X-ray studies by Glasser et al., (1950) have shown that during acute cold exposure, there occurs in man an increase in blood flow to the brain, liver and lungs. As a consequence of the increased rate of perfusion of these organs, an

increase in the rate at which ethanol was "cleared" from these organs would also be expected. Thus, the elicitation by hypothermia of "abnormal" perfusion patterns producing a rapid decrease in the absolute ethanol concentrations of "target" organs, might also result in the erroneous conclusion that an acceleration in the ethanol decay rate of the body as a whole had been achieved.

During rewarming, the BAC curves under both al and alex treatments were found to change direction, so that BACs increased during that period of the experiments. There is some evidence to suggest that these increases were actually artificial, resulting as a consequence of a gradual reversal of the haemodynamic alterations produced by hypothermia.

During rewarming, the manifestations of hypothermic induced hypovolemia become evident. For example, there occurs an insufficient total blood volume to fill the rapidly dilating peripheral blood vessels. As a result, fluid previously held interstitially returns to the circulation proper, and with this fluid, the plasma content of the blood proportionately rises. Ethanol would once again follow the interstitial fluid, and the absolute BAC would be expected to also increase. Additionally, since rewarming restores normal blood flow to the periphery, the alcohol holding-tank previously established by hypothermia would in effect be "ruptured", allowing the introduction of ethanol-rich blood into the circulation. Mixing of this ethanol-rich peripheral blood with the ethanol-depleted core blood would probably be reflected by an increase in the overall BAC.

The effects of the production of an alcohol reservoir on the BAC decay curves of subjects under normothermia has been shown in a study performed by Carre and Tremolier's (1958). These authors found that

the large pools of ascitic fluid present in their subjects had abnormally slow blood-fluid-ethanol equilibration rates. It was determined that ethanol was actually still diffusing into these pools at times when the drug was diffusing out of the body's other tissues and fluids, into the blood. Eventually, as the BAC became low, these pools began to slowly discharge ethanol back into the circulation. This influx from the ascitic pools balanced the rate of ethanol efflux from all other tissues and fluids, resulting in the establishment of a "plateau" of indeterminate duration in the BAC decay curves. It would appear probable that the plateaus observed during the final few minutes of rewarming in the al and alex experiments of the present study have resulted as a consequence of a similar phenomena, namely the slow release of ethanol from peripheral and interstitial reservoirs established during hypothermia.

In conclusion, there is considerable evidence supporting the view that the unexpected ethanol decay rates found during cold-water immersion and rewarming periods of al and alex experiments resulted from hypothermia-induced haemodynamic alterations which disturbed the ethanol-blood-tissue equilibrium established prior to cold exposure. Conversely, there is little evidence to suggest that the above results regarding ethanol decay were caused by any alteration in the actual rate of ethanol elimination. Finally, it is interesting to note that the hypothermia-induced haemodynamic alterations produced no change in the level of total ethanol decay, since the experimentally determined end-point BACs were equivalent to the predicted BAC end-points based on a standard normothermic ethanol decay rate. Whether this latter result was simply coincidental, or mirrored the efficiency of the physiological mechanisms which insure maximal ethanol metabolism under adverse conditions, is unknown.

II. Core Temperatures

A. Tympanic temperature analysis

1. Significance of the results of Ttym I and Ttym II analyses

Significant differences in tympanic temperatures amoung the treatments could only be demonstrated in the data on net differences in tympanic temperatures, of Ttym I and Ttym II samples during the exercise periods of cold-water immersions (i.e. the 90 to 110 minute period). Further analysis of that time period showed that the observed differences found amoung the treatments were due to an increased level of cooling which was present in immersions where exercise had been performed. According to Keatinge, (1969) and Hayward et al., (1975a) exercise is known to increase cooling by increasing peripheral blood flow, thus increasing the rate of heat loss from the body surface. Since increased tympanic cooling only occurred under the exercise treatment of ex and alex experiments, it was concluded that exercise per se was responsible for the observed increase in cooling. The fact that tympanic temperatures under all treatments equilibrated on the cessation of performing exercise indicates that no prolonged impairment of core temperatures occurred as a result of performing exercise. Finally, aside from the above exercise effect, the results of Ttym I and Ttym II analyses showed general agreement with the results of other studies where ethanol had been given in moderate dose, and no exercise was performed during the cold exposure periods of experiments, (Keatinge and Evans, 1960; Andersen et al., 1963; Martin et al., 1977).

2. Significance of the results of Ttym III analysis

The results of the analysis performed on the Ttym III data showed

that the only significant differences found in mean tympanic cooling rates occurred during the drinking period (i.e. 25 to 45 minutes) of the experiments. During that time, tympanic mean cooling rate was found to be significantly increased under al type treatment. According to Goodman and Gilman, (1965) high doses of ethanol (e.g. sufficient to produce a BAC of 300 mgm%) result in a marked increase in mean cerebral blood flow, and a diminished cerebral vascular resistance. It might be expected therefore, that the observed increase in tympanic cooling rate under al treatment resulted as a consequence of cerebral vasodilation. However, in view of the reported high dose of ethanol required to elicit such an effect, this explanation would seem unlikely.

In addition to increasing cerebral vasodilation, high levels of ethanol (e.g. BACs of 250 to 300 mgm%) are known to produce a depression of hypothalamic function, (Kissin and Beglieter, 1972). Haight and Keatinge, (1973) have shown that low doses of ethanol will impair hypothalamic function, resulting in decreased body temperatures provided that blood glucose reserves are low in the body. Thus, the increase in mean tympanic cooling rate observed under al treatment, during the drinking period of experiments, may have resulted from some impairment by ethanol of the hypothalamic center responsible for the maintenance of internal body temperature.

However, several observations argue against such a possibility. First, since no exercise was performed under alex treatment during the drinking period of the experiments, the al and alex treatments represent simple experimental replicates of each other at this time. Therefore, if ethanol was producing some general effect on mean tympanic cooling rate during this time period, one would expect to find a similar mean

tympanic cooling rate under both al and alex treatments. However, Table XXIII showed that the alex mean tympanic cooling rate was not significantly different from the rates found under either control or ex treatments.

Second, since no exercise was performed during the drinking period of experiments, subjects under all four treatments would have had equivalent blood glucose levels. Thus, the prerequisite of a low blood glucose level to allow a low-dose ethanol impairment of hypothalamic function to occur, would have been lacking under al treatment.

Third, although BAC would be expected to be higher under al than under alex treatment during the drinking period, (since at 55 minutes, mean BAC under al treatment equaled 93 mgm%, and under alex treatment mean BAC equaled 83 mgm%) the actual level of ethanol present in the blood under al treatment fell far short of the cited level necessary to produce hypothalamic impairment in the absence of hypoglycemia.

Finally, the fact that the increased tympanic cooling rate of al treatment was limited to the drinking period of experiments indicates that no prolonged ethanol-induced effect on tympanic cooling was present even under al treatment. It would seem unlikely that once established hypothalamic impairment would not continue, as evidenced by some further increase in mean tympanic cooling rate under al and/or alex treatments, during the actual cold-water immersion period of the experiments. Thus, there is little evidence to support any ethanol-induced hypothalamic impairment as an etiological explanation of the observed increase in tympanic cooling found under al treatment during the drinking period of the experiments.

A more reasonable explanation is to be found from ethanol's known

ability to produce a variable degree of vasodilation of the facial blood vessels during the period of its ingestion, (Wolff, 1972). Since the increase in mean cooling rate was limited to both the drinking period, and the tympanic site, it would appear that ethanol's ability to produce a moderate and transitory degree of facial vasodilation was actually responsible for the observed alteration of tympanic cooling found under al treatment.

A result of more consequence was the lack of any detectable effect of exercise to alter mean tympanic cooling rates during the cold- water immersion periods of ex and alex type experiments. This result was contrary to the conclusions regarding exercise's effect drawn from the results obtained from the anova analyses performed on the data of both the Ttym I and Ttym II samples during the exercise periods of ex and alex type immersions.

In order to force linearity onto the cooling curves of the subjects, and thereby calculate slopes and mean cooling rates, the first ten minutes of results obtained during the cold-water immersion periods of all experiments were discarded for the Ttym III sample. Unfortunately, in ex and alex type immersions the discarded ten minutes represented one-half of the total exercise performance period. In contrast, the entire twenty minute exercise period was utilized in Ttym I and Ttym II analyses. Additionally Ttym III data was analyzed by t-tests while two-factor anovas were employed in the analyses of Ttym I and Ttym II data. According to Sokal and Rohlf, (1969), the t-test is only statistically equivalent to a single classification anova. Therefore the failure to determine the existence of an exercise effect in Ttym III data during the cold-water immersion periods of ex and alex type experiments, result-

ed primarily from the exclusion of a major portion of the pertinent exercise-related data, coupled with a lower statistical "power" of the t-tests employed in Ttym III analysis when compared to the two-factor anovas used in Ttym I and Ttym II analyses.

Thus, although the analysis of mean tympanic cooling rates yields an identical general conclusion to that drawn from the analyses of Ttym I and Ttym II data, (i.e. no ethanol effect on tympanic cooling during cold exposure) the analysis of Ttym III data was unable to distinguish the exercise effect on tympanic cooling which Ttym I and Ttym II analyses had shown was important.

B. Rectal temperature analysis

1. Significance of the results of Trec I and Trec II analyses

Significant treatment related differences in rectal temperatures were found during the absorption period (55 to 85 minutes) of the experiments. Further analysis of this time period determined that the differences in rectal temperatures were limited to experiments where ethanol had been ingested. The grand mean for rectal cooling in al and alex trials during the absorption period was found to equal 0.19 ± 0.255 ^{o}C, while under control and ex treatments during the same time period, the grand mean for rectal cooling equaled 0.08 ± 0.210 ^{o}C. Since no significant differences were found amoung the treatments for tympanic cooling, increased core cooling during the absorption period of experiments would appear to be limited to the rectal site. However, it should be noted that tympanic cooling under al and alex treatments was found to be approximately double that found under control and ex treatments (eg. grand mean for tympanic cooling under al and alex treatments during the absorption period equaled 0.15 ± 0.345 ^{o}C, under control and ex treat-

ments for the same time period, the grand mean for tympanic cooling was 0.08 ± 0.312 ^{o}C). These results suggest that during the absorption period of experiments involving ethanol, ethanol acted to increase core cooling at both sites, possibly as a result of some degree of ethanol induced narcotization of autonomic function. The lack of any statistical significance found for differences in tympanic temperatures amoung the treatments during the absorption period may have been due to some increased level of non-treatment related variance, which might have obscured ethanol's effects at the tympanic site.

2. Significance of the results of Trec III analysis

No significant differences in mean rectal cooling rates were found amoung the treatments throughout the experiments in the analysis of Trec III data. As in the t-test analysis performed of the Ttym III data, the failure of Trec III analysis to duplicate the results of the two-factor anova analyses performed on Trec I and Trec II data, probably resulted as a consequence of the low statistical power of the t-test employed. In all likelyhood, the results of Trec I and Trec II having been subjected to a greater critical analysis are more correct. It is thought therefore, that ethanol did produce some small increase in rectal cooling during the absorption period of al and alex experiments. However, this conclusion was not verified by the results of Trec III analysis.

C. General conclusions

Keatinge, (1973) has shown that in the absence of a level of exercise sufficient to induce functional hypoglycemia, moderate doses of ethanol have little or no effect on a subject's core cooling rate under severe cold stress. This observation has been indirectly confirmed by the work of others who have studied ethanol's effects on thermal balance

(Keatinge and Evans, 1960; Andersen et al., 1963; Hobson and Collis, 1977; Martin et al., 1977). The results of the present study would also agree with those of Keatinge (1973). That is, contrary to the popular opinion that ethanol's vasodilatory properties would prove detrimental to the maintenance of body temperature in severe cold stress, the results of this study indicate that the ingestion of ethanol (sufficient to produce a BAC of approximately 80 mgm%) will not accelerate the rate of a subject's entry into hypothermia induced through a cold-water immersion, even if a moderate level of exercise was performed by that immersee.

III. Metabolic rate

From the results, it was evident that in immersions where subjects performed a 20 minute period of exercise, mean metabolic rates were significantly increased (eg. a 37% increase in ex over control treatment, and a 44% increase in alex over al treatment). Exercise is known to increase metabolic rate through its ability to increase the level of chemical activity of muscle cells, (Chapman, 1968; Guyton, 1971). Since the avoidance of hypothermia is primarily dependent on the ability of an immersee to counteract heat loss to the environment through various heat producing and heat maintaining mechanisms, it might be postulated that any increase in mean metabolic rate achieved through the performance of exercise, would result in a slowed entry into hypothermia. However, reference to the results of Ttym I and Ttym II analysis show that in fact the opposite effect occurred during the actual exercise period of experiments. That is, during this period of ex and alex experiments, core cooling (tympanic) actually accelerated.

According to Keatinge, (1969) and Hayward et al., (1975a) exercise in water temperatures below 20 ^{o}C results in an accelerated cooling rate, in spite of substantial increases in metabolic heat production. An increase in core cooling occurs because exercise in addition to increasing metabolic rate, also has a deleterious effect on the circulatory gradient established through the constrictatory action of cold on peripheral blood vessels. Thus, exercise acts to increase blood flow to the body surface, therefore increasing net body conductance to the environment. In this respect, it is evident that exercise in cold water may actually be detrimental to the maintenance of man's thermal balance. It is not surprising therefore that in this study, exercise was found to

increase core cooling as well as increasing metabolic rate.

In addition to the above exercise effect, ethanol without exercise was found to exhibit a tendency to produce a reduction of the subjects' mean metabolic rates. Ethanol was shown to have decreased heat production by 18% in al type experiments, and by 10% in alex type experiments following the cessation of exercise performance.

A decreased pain and cold sense caused by a general narcotization of peripheral neurons under low to moderate doses of ethanol can bring about a decrease in body temperature as a result of an impairment of shivering and voluntary muscular activity, (Truitt et al., 1956; Root and Hofman, 1963; Kissin and Beglieter, 1971). Since according to both Keatinge, (1969) and Hayward, (1977) shivering represents the major mechanism by which immersed individuals can increase their net heat production, it is probable that the decreased mean metabolic rates seen in immersions involving ethanol resulted as a sonsequence of some ethanol-induced reduction in shivering. In the present study, the level of shivering "appeared" to be decreased in subjects undergoing alcohol type trial. Unfortunately, as no electromyographic recordings were performed in the present study, it can not be said with any certainty that shivering was actually depressed during ethanol type immersions. However, several other authors have reported that shivering during cold exposure was reduced in their subjects who had taken ethanol, (Keatinge and Evans, 1960; Andersen et al., 1963; Hobson and Collis, 1977; Martin et al., 1977).

In addition to its effects on shivering, ethanol is known to produce detrimental effects on normal intermediary metabolism, such as the cessation of gluconeogenesis and fat hydrolysis, which can result in the

rapid onset of carbohydrate substrate insufficiency and in some cases hyperlipidemia, (Forbes and Duncan, 1950; Frienkal et al., 1963; Kalant et al., 1963; Krebs et al., 1969; Lieber, 1976). These metabolic disturbances are dose-dependent, and usually large doses of ethanol (i.e. sufficient to produce a BAC in excess of 250 mgm%) are required to elicit them, (Sardesai, 1969). However, Haight and Keatinge, (1973) have shown the consequences of ethanol consumption prior to cold exposure when glucose reserves are low in the body. The resulting hypoglycemia produced under these circumstances will drastically reduce overall metabolic rate, and impair hypothalamic function, resulting in a dramatic increase of a subject's rate of entry into hypothermia.

In the present study, it is highly unlikely that the experimental exercise stress imposed was severe enough to have produced hypoglycemia in the subjects. Rather, it would appear that the decreased mean metabolic rates evident in immersions involving ethanol of the present study, resulted primarily from the drug's ability to impair shivering, as opposed to any direct ethanol effects on either intermediary metabolism, or hypothalamic function.

Any decrease in the level of heat production, if it occurred without some compensatory reduction in the level of heat loss, would be expected to produce an accelerated core-cooling rate in cold water. Yet reference to the analyses of core-cooling performed for this study, show that in spite of a significantly decreased level of heat production under ethanol treatment, subjects did not cool at rates in excess of those found under control treatments. Presented below are two plausible explanations tendered to account for the above contradictory result.

According to Keatinge, (1969) shivering, a form of involuntary

exercise, results in both increased heat production and increased heat loss. It is possible therefore, that under certain circumstances shivering would be detrimental to the maintenance of man's thermal balance during cold-water immersions. This view has been postulated by Hobson and Collis, (1977) who have stated that a reduction in the level of shivering achieved through ethanol may in fact be advantageous to immersees in situations where any thermogenic benefit provided by shivering is negated by an increased rate of heat loss resulting from shivering's effects on peripheral circulation. Unfortunately, this first explanation of ethanol's effects on heat production implies that subjects undergoing hypothermia-inducing exposure without ethanol treatment are engaged in some level of "non-productive shivering". Although such a possibility might be valid under certain conditions, it would seem a somewhat tenuous etiological explanation of the observed effect of ethanol on metabolic rate found in the present study.

The second and perhaps more reasonable explanation, relates to the actual efficiency of heat producing mechanisms in combating heat loss. Since ethanol did not significantly alter the rate of heat loss, it is probable that the statistically-significant reduction in the level of heat production, found under ethanol treatment for this study, was not representative of any concomitant physiologically-significant effect.[1] Hayward et al., (1975a) have shown that any increase in heat production is only approximately 30% efficient in combating the heat loss of subjects

1 Since mean skin temperatures were not found to significantly differ amoung any of the treatments throughout the course of the experiments, heat loss was concluded to have been equivalent under all four treatments.

under conditions similar to those used in this study. Assuming that mean metabolic rates observed in the absence of exercise under control treatments represent a maximal non-exercise response in regards to heat production for hypothermic subjects, then any reduction in mean metabolic rates found under ethanol treatments would represent some proportional reduction of the maximal 30% efficiency rate. Thus, the overall reductions of 18 and 10% found in mean metabolic rates under al and post-exercise alex treatments would be equivalent to an overall decrease in the maximal heat-production efficiency of only 5.4 and 3.0% respectively. It would seem improbable that such minor decreases in the overall efficiency of heat production to combat heat loss would be of any great physiological consequence regarding the establishment of core-cooling rates.

It is thought therefore, that the statistically-significant reduction in mean metabolic rates found in ethanol trials actually had little or no physiological effect insofar as any increase in the rate of entry into hypothermia was concerned. However, the ability of ethanol to reduce heat production when present at a BAC of approximately 80 mgm% would indicate that at a higher BAC the drug could be responsible for a physiologically-significant reduction of metabolic rate. Thus, some "risk" of increased core-cooling, as a result of an ethanol impairment of heat-producing mechanisms of the body, was implied by this study's results.

IV. Skin temperatures

Mean environmental temperatures were found to follow an identical course throughout all experiments. No significantly different mean skin temperatures were found amoung the treatments at either the sternum or the left biceps skin sites. Similarly, although some significant differences in mean skin temperatures were found amoung the treatments at certain time isolates of the mean skin temperature curves at both the lumbar and left thigh skin sites, no consistent treatment-induced differences in mean skin temperatures at either of the above sites were found to exist throughout the experiments. From the above, and further subsequent analyses performed on mean skin temperatures, it was concluded that no treatment-related differences existed in mean skin temperatures at any of the four sites tested.

Since at no site were mean skin temperatures of ethanol trials found to be significantly different from those found under control type experiments, it was concluded that ethanol with or without exercise did not produce any marked vasodilation of cutaneous blood vessels. This conclusion might be regarded with some surprise, in view of ethanol's known ability to produce marked cutaneous vasodilation under conditions of mild cold stress, even when the drug is present at low BAC, (Cushny, 1910; Cook and Brown, 1932; Gaddum, 1956; Kissin and Beglieter, 1971,1972). However, such a conclusion is not unprecedented under hypothermic conditions. For example, Andersen et al., (1963) who measured skin temperatures at eight sites after their subjects had been given relatively high levels of ethanol were unable to find any significant increase in heat loss from those skin sites when the subjects were exposed to ambient air temperatures of 15 and 20 ^{o}C. In the present study, environmental grand mean air

temperature equaled 22.5 °C prior to the cold exposure period of the experiments, and since the subjects were "dosed" at a level considerably lower than that used in the Andersen study, it would seem unlikely that the present study would find significant cutaneous vasodilation where Andersen did not. Additionally, Keatinge and Evans' (1960) study on ethanol's effects on men immersed in 15 °C water did not find any significant increase in the rate of mean blood flow through the index fingers of their subjects during ethanol trials. Furthermore, recent unpublished studies by Livingstone et al., (1977) involving ethanol and skin surface heat loss,in cool air (25 °C) as assessed by thermography and, in cool water (25 °C) as assessed by direct calorimetry,have shown that for healthy young men, the consumption of ethanol at a dose of 1.0 ml 100% ethanol · kg body weight^{-1} does not increase surface heat loss or produce any marked cutaneous vasodilation.

In fact, the long established vasodilatory action of ingested ethanol under normothermia, or mild cold stress, is itself presently in some doubt, as Fewings et al., (1966) have shown that ethanol, when injected into the forearms of men intravenously (IV), produces a rapid and prolonged vasoconstriction of the arterioles of both skin and musculature. This vasoconstriction was found to be unalterable by either sympathectomy or treatment with phenoxybenzamine. Conversely, these same authors reported that when ethanol was ingested, vasodilation of the skin arterioles with concomitant vasoconstriction of the arterioles of the musculature occurred.[2] These authors concluded that the vasoconstrictive action resulted from a direct effect of ethanol per se, on the smooth muscle

2 Such ethanol induced haemodynamic alterations as those cited above would be most conducive to the entrapment of whole blood in peripheral blood vessels, (see Discussion section I. BAC for further elaboration).

of arterioles, while the drug's vasodilatory effect represented some central reflex phenomena involving neural vasomotor regulatory centers.

Lastly, it is well known that under hypothermic conditions, the physiological "pressure" to vasoconstrict is extremely great, as a result of both peripheral nervous activity, and the constrictatory action of high levels of circulating catacholamines known to be present, (Johnson et al., 1977). Therefore, it was concluded that in the present study, ethanol was unable to elicit any significant cutaneous vasodilation in the face of an overwhelming pressure to vasoconstrict.

V. Heart rate

Under all treatments, the heart rates of the subjects increased dramatically on entry into the cold-water tank. On entry, subjects were also seen to hyperventilate during the first few minutes of the immersions. Such hyperventilation is actually characteristic of sudden cold-water immersions, (Hayward, 1975a). According to Goode, (1976) hyperventilation results from a neural reflex action (spill-over stimulus) of the skin temperature receptors affecting the respiratory center of the medulla. As the cutaneous receptors adapt to the lower skin temperatures, spill-over stimulation to the respiratory center ceases, and hyperventilation stops. Spill-over of excitatory stimulation is also known to occur between the respiratory and the cardiac centers located in the medulla, (Guyton, 1971). It was concluded therefore, that the dramatic increase in heart rates found on entry into the cold water probably resulted as a consequence of impulse spill-over from the respiratory to the heart-rate centers.

According to Goode, (1976) during hyperventilation, the heart rates of his subjects, immersed in 11 ^{o}C water, did not rise above a maximum of 120 beats $\cdot$ minute^{-1}. In the present study, the grand mean heart rate for all four treatments equaled 133 beats $\cdot$ minute^{-1}. Following the cessation of hyperventilation, mean heart rates were found to stabilize between 110 and 120 beats $\cdot$ minute^{-1} in both control and al type immersions. Mean heart rate was found to increase steadily during the extent of the exercise period of ex type immersions, while under alex treatment, during that time period, mean heart rate stabilized at approximately 135 beats $\cdot$ minute^{-1}. Following the cessation of performing exercise, mean heart rates decreased in both of the latter experiments. However, while

mean heart rates of alex trials dropped to levels equivalent to those seen under control and al treatments, mean heart rates of ex trials remained significantly elevated above the levels found under all other treatments, during the remainder of the cold-water immersion period.

Heart rate is known to decrease slowly with decreasing core temperatures under hypothermic conditions, (Keatinge, 1969; Nicolas et al., 1975; Goode, 1976). According to Keatinge, (1969) this induced bradycardia results from a cold narcosis of the pacemaker. In severe cases of hypothermia, death occasionally follows cardiac arrest resulting from a complete blockage of impulse conduction through the atrioventricular node, (Lloyd and Mitchell, 1974: Latsis and Leias-Sauss, 1975). However, usually death in hypothermia results from ventricular fibrillation elicited by an energy insufficiency of the cardiac muscle itself, (Fleming and Muir, 1957).

Early studies of ethanol's effects on heart rate under normothermia indicated that usually the ingestion of ethanol produces some increase in mean heart rate. This increase in heart rate, limited to the first 15 to 30 minutes of the actual drinking period, is often followed by a prolonged period of bradycardia, (Holmberg and Martens, 1958). It was later shown that the transitory period of tachycardia which occurs during ethanol consumption results as a side-effect of an increase in voluntary activity brought about by an ethanol-induced loss of psychological inhibition. No increase in mean heart rate has been reported during the drinking period of studies where excessive psychological stimulation was absent, (Gould et al., 1972).

Ethanol has also been found to block completely all impulse transmission in cardiac muscle, although the dose required to elicit cardiac

arrest by this method is approximately double that which will cause respiratory arrest, (e.g. the BAC required to produce cardiac arrest equals 1250 mgm%, to produce respiratory arrest it equals 550 mgm%), (Goodman and Gilman, 1965). Even in low dose however, the drug has been reported to elicit several cardiac effects. For example, ethanol has been found to reduce the increase in heart rate found during sudden cold-water immersions, and to offer some level of protection from both ventricular fibrillation and extrasystolic contraction in subjects throughout immersions, (Keatinge and Evans, 1960; MacGregor et al., 1966). The drug presumably produces these effects by decreasing the conductance of the excitable cells of the heart proper, thus increasing the refractory period of cardiac muscle.

The production of ethanol-induced bradycardia, was not evident in the present study in the absence of exercise performance, probably as BACs were too low to elicit any direct ethanol narcotization effect on cardiac muscle. The production of tachycardia through physical exercise is well known, and will not be discussed here. However, an attempt will be made to explain why bradycardia was only found under alex treatment following the cessation of performing exercise.

Ethanol is not metabolized by heart muscle, as it lacks a sufficient level of ADH to perform ethanol oxidation of any significant degree, (Larsen, 1959; Gailus and Verdy, 1970). Additionally, ethanol is known to stop gluconeogenesis in the liver, and residual glycogen reserves in the body would be rather rapidly depleted during the exercise period, (Saltin and Hermansen, 1967). In a post-exercise condition therefore, ethanol would represent the presence of a non-metabolizable substrate as far as cardiac muscle was concerned, at a time when cardiac glucose

reserves would be critically low. It is probable therefore, that during the post-exercise period of alex experiments, ethanol interfered with normal cardiac metabolism to such an extent that a significant level of bradycardia resulted. Furthermore, the results of this study would indicate that any ethanol related bradycardia produced in hypothermia would result as a secondary consequence of an exercise-induced metabolic stress imposed on the cardiac muscle, rather than as a primary result of a direct ethanol-induced narcosis of cardiac muscle.

VI. Blood pressure

Ethanol in low dose (i.e. sufficient to produce a BAC equivalent to 40 mgm%) has been reported to produce transient cutaneous vasodilation under normothermia or mild cold stress, (Docter and Perkins, 1960; Docter and Bernal, 1964; Gillespie, 1967). At moderate BAC (i.e. 100 mgm%) the drug is known to produce vasodilation of the gastric arteries during the ingestion period, (Ritchie, 1965). It might be expected that some concomitant increase in arterial blood pressure would also occur as a result of such vasodilation since presumably, some increase in pulse pressure would be achieved through an increase in venous return and cardiac stroke volume. However, a survey of the ethanol literature shows that even in cases where cutaneous vasodilation has been observed, low to moderate BACs produce no significant changes in arterial blood pressure, (Schnall and Wiener, 1958; Gould et al., 1971; Gould et al., 1972).

In the current study, no significant changes in mean systolic, dystolic or pulse pressures were found during the pre-immersion period of experiments whether ethanol had been ingested or not. Additionally, no cutaneous vasodilation as indirectly assessed by measures of mean skin temperatures was evident during any time period of any experiments involving ethanol.

According to Roth and Sheard, (1947) the presence of nicotine in the blood as a result of smoking will abolish the vasodilatory effect of consuming ethanol. Thus, it would appear that the degree of narcotization produced by ethanol on the constrictor neurons of cutaneous blood vessels is neither severe nor debilitating as even small doses of sympathomimetic drugs are able to abolish the vasodilatory effect. In the

present study, subjects sat attired in a bathing suit while exposed to an ambient air temperature of approximately 20 ^{o}C, throughout the pre-immersion period of the experiments. Evidently, the mild cold stress presented by this procedure was sufficient to produce a level of sympathetic-induced vasoconstriction of ample quantity to compensate for both the minor cold stress and any ethanol-induced vasodilation.

Shivering by subjects undergoing cold-water immersions made accurate sphygmomanometric determinations of blood pressure impossible during the entire cold-exposure period and the first ten minutes of the rewarming period. However, some speculation as to what most likely would have occurred regarding blood pressure during this "missing" time period is possible on the basis of previous studies. Severe cold stress is known to produce massive cutaneous vasoconstriction as a result of an increased peripheral sympathetic nervous activity, (Keatinge, 1969). In view of the ability of the subjects to compensate for even mild cold stress in the presence of ethanol, it would seem highly improbable that any significant ethanol-induced changes in mean blood pressures would have resulted under the greater cold stress imposed by cold-water immersion.

During the rewarming period, as the skin temperature of a subject rapidly increases, cutaneous blood vessels which were previously constricted dilate, (Keatinge, 1969). This dilation results in a fall in central blood pressure, as blood becomes trapped or "pools" in the peripheral venous bed, and thus decreases the total blood volume which returns to the heart. In addition to this pooling effect, total blood volume is further reduced as a consequence of the previous hypothermic treatment. It will be recalled that hypothermia causes an increase in central blood pressure during an immersion, as a result of peripheral vasoconstriction.

This increase in blood pressure is physiologically compensated for by an increased diuresis and occurrence of extra-vascular fluid shifts. Thus, during subsequent rewarming, a decrease in dystolic pressure is expected, as a result of the manifestation of hypothermia's hypovolemic effects. A similar decrease in systolic pressure would also be expected; however, this result was only found in immersions involving ethanol treatment. Since heart rates were equivalent under all treatments during rewarming, the lack of a decreased systolic pressure under control treatments, would suggest that an increased total blood volume was present in the arterial circulation of the subjects undergoing control trials. This result would also imply that ethanol treatment resulted in a net increase in the level of hypovolemia resulting as a consequence of hypothermia.

Ethanol might produce an increase in hypovolemia by several methods. Perhaps the most obvious being the drug's ability to increase cutaneous vasodilation, and therefore increase the pooling effect. However, although BACs were still high enough to elicit a dilatory effect, the results of mean skin temperature would indicate that actually no ethanol-induced vasodilation occurred during the rewarming period. A more feasible mechanism by which ethanol may have increased hypovolemia may be found in the data on urine mean total volume. Urine mean total volumes were found to be increased for the post-immersion samples as a result of an ethanol-induced diuresis. Presumably, ethanol's diuretic effect acted to increase hypovolemia indirectly by increasing the rate of water lost via the urine.

It was concluded therefore, that peripheral vasodilation was primarily responsible for the observed decrease of dystolic pressure found

under all treatments during the rewarming period. The observed decrease in systolic pressure found during rewarming under alcohol treatment resulted from a further increase in the level of hypothermia-induced diuresis. Finally, the increased pulse pressure seen during rewarming is thought to have resulted from the effects of a minor decrease in heart rate coupled with a decrease in peripheral resistance.

VII. Urine

Throughout the course of the experiments, no changes in the urinary excretion of either glucose, or total protein were found under any treatment. Presumably, this result indicated that no drastic impairment of kidney function occurred as a consequence of either ethanol ingestion, or cold-water immersion.

Similarly, throughout the course of al trials, the mean total volume of urine excreted was significantly and steadily increased. Additionally, it was found that under all experimental treatments, urine mean total volume, and urine mean pH were respectively increased, and decreased in urine sample III (i.e. the post-cold exposure sample).

According to Keatinge, (1969) an increased rate of both water and salt excretion occurs during cold exposure. This increase in excretion represents a physiological compensatory response to the increased blood volume brought about by vasoconstriction of peripheral blood vessels in the cold. There is some evidence that as body temperature falls, the rate of water and salt loss proportionately increases as a result of a direct cold impairment of renal tubular cells, (Moyer et al., 1957: Fisher et al., 1958). However, renal failure does not usually occur at core temperatures above 30 ^{o}C, (Keatinge, 1969).

In addition to the above cold diuretic effect, it has been known for some time that ethanol acts as a diuretic drug. The drug's diuretic effect is thought to be elicited through an ethanol-induced inhibition of the release of ADHor from the neurohypothesis of the pituitary gland, (Van Dyke and Ames, 1951). Additionally, ethanol diuresis begins only after a variable latent period, (Engleton, 1942). Usually, ethanol

diuresis is initiated coincidentally with the establishment of a "peak" BAC, (Engleton and Smith, 1946). It was concluded therefore, that the increase in total mean urine volume observed under all treatments for urine sample III, resulted primarily as a consequence of an expected degree of cold-induced diuresis. That this cold-induced diuresis was augmented to a considerable extent by the elicitation of a further ethanol-induced diuresis in alcohol experiments was evident from the results obtained under al treatment.

Hypothermia is known to impair the "clearance" of metabolic acids from the circulatory system, (Keatinge, 1969). Occasionally, acidosis occurs in hypothermic subjects as a result of an increase in the solubility of CO_2 which occurs at low temperatures, and the inability of subjects to efficiently "clear" excess lactic acid, produced as a by-product of shivering, from the bloodstream, (Henneman et al., 1958). However, according to Keatinge, (1969) although some transitory and occasionally severe acidosis has been found following the rapid rewarming of hypothermic subjects, acidosis does not usually result in immersees so long as the hypothermic state is maintained.

Ethanol oxidation produces a large surplus of hydrogen in the liver, (Sardesai, 1969). This excess hydrogen reduces pyruvate to lactate, thus ceasing gluconeogenesis in the liver, (Lieber, 1976). In the kidney, high levels of ethanol-produced lactic acid interfere with the excretion of uric acid, and in cases where BAC is in excess of 125 mgm%, hyperuricemia has been known to occur, (Lieber, 1976). The level of lactate contributed by ethanol is dependent on the amount of ethanol oxidized, (Sardesai, 1969). Therefore, under alcohol treatments, urine sample II (the post-drinking sample) would be expected to display a greater decrease

in urine mean pH when compared to that found for sample I (i.e. the pre-drinking sample) as sample II was taken at a time when the BAC curve was peaking, and therefore, ethanol oxidation would have been maximal during the time period between the collection of samples I and II. However, such a result was not found, alcohol treatment apparently had no effect in increasing the level of acidosis of the subjects. It was concluded therefore that the significant reductions of urine mean pH found under all treatments in the third urine sample were probably caused by an increase in the level of circulating lactic acid, produced in association with the rewarming process.

VIII. Placebo

That basic physiological functions are to varying degrees subject to both psychological and behavioral modification has been known for some time. For example, hunger, thirst, and pain sensations are all alterable through conscious effort, (Zimbardo, 1969), while Hayward et al., (1975b) have shown that simple behavioral modifications can approximately double the predicted survival time of cold-water immersees. Examples of the sub-conscious alteration of physiological processes are forth-coming from the medical literature on psychosomatic disease. Often, a great improvement in a psychosomatic condition results from the simple establishment of a good patient-physician interaction. Evidently, such "treatment" is able to alleviate physiological symptoms by reducing the level of psychological stress, (Urh and Miller, 1960). In view of the prevalence of psychophysiological effects, pharmacological studies often include a placebo control in an effort to differentiate between a drug's true pharmacophysiological effects and its possible psychophysiological effects.

In the present study, as in others, subjects undergoing cold-water immersions reported that they "felt" warmer and less anxious throughout the immersion if prior to its initiation, they had received ethanol. Such reports would indicate that as a result of consuming ethanol, the level of psychological stress was reduced in cold-water immersions. Such a reduction of psychological stress might be expected to produce some concomitant alteration of the typical physiological response to cold stress. The results of the placebo immersions performed in this study should therefore provide an estimate of the effectiveness of ethanol

in the production of significant psychophysiological alterations during cold stress.

The results for placebo treatment showed that for every physiological variable studied, placebo treatment acted as a simple experimental replicate of control treatment. Such a result might be considered somewhat unusual, as usually, some modification of physiological response occurs under placebo treatment, (Urh and Miller, 1960; Johnson and Solso, 1971). However, this result might be expected if either; a) the subjects did not believe that the placebo type drinks contained any ethanol, and therefore, no psychological alterations of mood insued as a result of placebo treatment, or b) simply no physiological alterations were accomplished in spite of placebo-induced alterations of psychological mood. Since all three subjects upon questioning stated that they believed they had actually consumed ethanol, albeit at a supposedly lower dose, in placebo trials, it would seem improbable that psychological alterations of mood did not result as a consequence of placebo treatment. Consequently it would appear that the level of physiological stress to which the subjects were exposed in cold-water immersions was adequate to overwhelm any modifying psychophysiological effects produced under placebo treatment. Furthermore, in view of the relatively minor physiological effects found to have been elicited by ethanol treatment <u>per se</u> during genuine alcohol trials, the lack of any significant changes in the physiological response to hypothermia found under placebo treatment may perhaps have been expected.

IX. Summary

A. General conclusions

Under the condition of this study, ethanol was found to produce the following significant effects:

1. Increased mean tympanic cooling rate during the drinking period of experiments, (under ethanol treatment, the grand mean for the tympanic cooling rate, during the drinking period equaled 0.5 ± 0.28 $^{o}C \cdot hour^{-1}$; under control treatment, the grand mean for the tympanic cooling rate, during the drinking period equaled 0.2 ± 0.29 $^{o}C \cdot hour^{-1}$).
2. Increased core cooling at the rectal site during the absorption period of experiments, (under ethanol treatment, the grand mean for total rectal cooling, during the absorption period equaled 0.2 ± 0.26 ^{o}C ; under control treatment, the grand mean for total rectal cooling, during the absorption period equaled 0.1 ± 0.21 ^{o}C).
3. Did not cause any alteration in core cooling during the actual period of cold-water immersion.
4. Did not cause any alteration in the rate of rewarming from hypothermia.
5. Produced a statistically-significant but physiologically-insignificant reduction (approximating 20%) in the mean metabolic rate found during the post-exercise period of immersions.
6. Did not alter mean skin temperatures, nor produce any measurable degree of cutaneous vasodilation (as assessed by mean skin temperatures) at any time during the experiments.
7. Produced bradycardia only in the post-exercise period of immersions where exercise had been performed.
8. Decreased systolic and stabilized pulse blood pressures during the final 20 minute period of rewarming.

9. Increased the mean total volume of urine excreted as a consequence of undergoing cold exposure.
10. On the basis of three subjects, produced no psychological effects capable of modifying any physiological response to cold stress.
11. Appeared to have produced a state of lessened anxiety and greater comfort for the volunteers during their subsequent immersions.

Exercise was found to have produced an approximate 75% increase in tympanic cooling during the actual period of its performance in cold-water immersions.

In view of the above results, the following conclusions were drawn regarding the original null hypotheses presented in the Introduction:

Hypothesis 1 : rejected; the consumption of ethanol prior to a hypothermia-inducing cold-water immersion did not produce any alterations in core-cooling rates during the immersions.

Hypothesis 1a : rejected; exercise did not potentiate any ethanol effect on core-cooling during the immersions, however a clear exercise-induced effect of increased tympanic cooling was found during the periods of exercise performance during the immersions.

Hypothesis 1b : rejected; ethanol did not produce any physiologically-significant effects on either heat-production or heat-loss mechanisms.

Hypothesis 2 : rejected; ethanol did not produce any direct effects on cardio-vascular function during hypothermia, however ethanol with exercise did produce significant bradycardia during the alcohol-exercise type immersions.

Hypothesis 3 : rejected; (on the basis of three subjects), ethanol's

ability to alter psychological mood did not produce any physiological alterations for any variable studied.

Hypothesis 4 : accepted; ethanol consumption prior to the inducement of hypothermia resulted in an alteration of typical peripheral circulatory dynamics during the subsequent periods of rewarming from hypothermia.

Hypothesis 5 : accepted; ethanol increased the level of diuresis normally found in immersion subjects, but had no effect on urine glucose and total protein concentrations, or on urine mean pH.

Hypothesis 6 : rejected; hypothermia increased the rate of BAC decay through the production of haemodynamic alterations; exercise however, had no effect on BAC decay rates.

B. Recommendations regarding ethanol and hypothermia

The results of this study indicate that ethanol will have no deleterious effects on the core-cooling rates of persons immersed in cold water, provided that; a) the drug is present in the blood in low to moderate concentration (i.e. at a BAC between 50 and 100 mgm%), and b) only a minor level of exercise is performed while immersed (i.e. a level insufficient to induce hypoglycemia). Ethanol's ability to reduce anxiety and increase the comfort of immersees might well serve to make sudden accidental cold-water immersions less traumatic. However, this does not signify that any endorsement of ethanol consumption by the boating public is in anyway implied, since the drug's well known effects on motor coordination and higher cerebral function would increase the probability of accidental immersion.

In summary, the risks involved in moderate ethanol consumption are insignificant when the drug is taken prior to the cold exposure, and the above conditions are met. However, it is not recommended that ethanol be consumed once immersed, nor that it be given as part of any general rescue treatment for hypothermia. In this study, ethanol was not found to significantly alter the rate at which normal core temperatures were re-established during the period of rewarming from hypothermia. However, during rewarming, a significant level of hypotension was found under alcohol treatment, indicating that ethanol had produced some cardio-vascular impairment. The actual degree of risk involved in such an impairment would seem to be minor, provided that the hypothermic subject is rapidly rewarmed, as is the case in experimental situations. Nevertheless, it is not desirable that ethanol be consumed during typical emergency rescue conditions, where inefficient rewarming of victims is common. This recommendation is based on the supposition that under inefficient rewarming, minor ethanol effects on thermal balance and/or cardio-vascular function may well be a critical factor in determing whether or not a hypothermic victim survives.

C. Further studies

Further haemodynamic studies would be of interest to determine to what extent the observed increases in the rate of BAC decay found in this study were in fact due to suspected hypothermia-induced haemodynamic alterations. Additionally, it would be interesting to investigate ethanol's effects under heat stress - a condition where a significant level of ethanol-induced cutaneous vasodilation might be found. Finally, the observed decrease in mean metabolic rate evident under ethanol treatment bears further examination, since it would be expected that at some critical

ethanol dose, the ability of an immersee to produce heat would be so impaired that his rate of entry into hypothermia would necessarily increase.

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Appendix 1. Widmark's hypothesis; general discussion of the hypothesis and use of the method to calculate an expected mean BAC for Keatinge and Evans' (1960) study

I. Widmark's Hypothesis - General Discussion

The Swedish pharmacologist, Erick Widmark first published his now famous hypothesis and method by which to calculate expected BACs for various time post-ethanol absorption in 1933. Since then his method has been used extensively in studies involving ethanol and man. The method's ability to closely estimate BAC has been well established, and is occasionally used to calculate BACs in situations where it is not feasable to obtain BAC by experimental methods. Basically, the hypothesis accounts for the various factors relating to an alcohol's absorption, distribution and elimination in the body, thus providing a method whereby an experimentor may calculate estimates of BAC for any time after the cessation of alcohol ingestion that he might desire.

Widmark's hypothesis states that BAC at any one time is primarily determined by; a) the actual level of alcohol present in the blood at that time, and b) the extent to which this level has been altered by alcohol elimination. The hypothesis is expressed algebraically below;

$$BAC = Ct = \{((A \cdot W^{-1}) \cdot 100) \cdot r^{-1} \} - \{ \beta \cdot t \}$$

The first portion of the equation ;

$$Ct = \{((A \cdot W^{-1}) \cdot 100) \cdot r^{-1} \}$$

is equivalent to the theorectical BAC which would result at the initiation of alcohol ingestion (or time 0), if absorption and distribution of ingested alcohol occured immediately without any concomitant oxidation of alcohol.

The algebric terms are equivalent to;

Ct = BAC at time t minutes (ie. the expected BAC)

A = the actual amount of alcohol ingested by the individual

W = the body weight of the individual

r = an experimentally determined constant (equivalent to 0.68), representing the ratio of the concentration of alcohol present in the body as a whole compared to the concentration of alcohol present in the blood itself, assuming alcohol absorption from the gut has ceased.

Unfortunately, the rate of alcohol absorption is variable amoung individuals, and within the same individual under different conditions. Widmark's method can not therefore be used with any accuracy so long as absorption is still in process. For this reason, experimentors allow a 20 to 40 minute period to elapse after the cessation of alcohol ingestion to insure that alcohol absorption has ceased, prior to making either experimental BAC determinations, or performing calculations involving Widmark's method. Additionally, the term r is subject to variation amoung individuals, and at best is considered only an approximation of any specific individual's true alcohol equilibration constant.

The second portion of the equation;

$$- \{ \beta \cdot t \}$$

is equivalent to the theorectical proportion of alcohol eliminated by all mechanisms from the body during the time interval t minutes.

The term β is an experimentally determined constant (equivalent to 0.265 mgm% $\cdot$ minute^{-1}) representing a typical level of oxidation in the liver. Once again as for the term r, β is variable amoung individuals and is considered only an approximation of any specific individual's rate of alcohol oxidation.

However in spite of the limitations presented above, Widmark's method has been found to yield reasonably accurate estimates of BAC in

situations where an experimentor knows only, an individual's body weight, and the amount of alcohol consumed by him.

II. Utilization of Widmark's method to calculate an approximate BAC for Keatinge and Evans' (1960) study.

A. From Keatinge and Evans' (1960) study;

amount of ethanol ingested = 75.0 ml 100% (v/v) ethanol

mean body weight of subjects = 150 lb

B. Conversion factors;

1.0 ml 100% (v/v) ethanol = 0.781 g 100% (v/v) ethanol

1.0 lb = 0.455 kg

C. Expected mean BAC of subjects at the initiation of the cold exposure in above study, (ie. 45 minutes after cessation of ingestion);

From

$$Ct = \{((A \cdot W^{-1}) \cdot 100) \cdot r^{-1}\} - \{\beta \cdot t\}$$

Where

Ct = BAC at t minutes

A = 58.35 g 100% (v/v) ethanol

W = 68.25 kg

r = 0.68

β = 0.265 mgm% $\cdot$ minute^{-1}

t = 45 minutes

Then

C45 = (125.7 mgm%) - (12.015 mgm%)

= 113.7 mgm%

Thus the mean BAC 45 minutes after the cessation of ethanol ingestion would be expected to equal approximately 110 mgm% under the conditions of Keatinge and Evans' (1960) study.

Appendix 2. Utilization of Widmark's method to calculate approximate mean BACs for Andersen et al's (1963) study

I. Utilization of Widmark's method to calculate approximate BACs for Andersen et al's (1963) study.

A. From Andersen et al's (1963) study;

ethanol doses given;

low dose = 1.0 ml 100% ethanol (v/v) · kg of body weight^{-1}

high dose = 1.5 ml 100% ethanol (v/v) kg of body weight^{-1}

mean body weight of subjects = 82.0 kg

B. Conversion factor;

1.0 ml 100% ethanol (v/v) = 0.781 g 100% ethanol (v/v)

C. Expected mean BACs under low and high doses for both experimental series of Andersen et al's (1963) study;

From

$$Ct = \{((A \cdot W^{-1}) \cdot 100) \cdot r^{-1}\} - \{\beta \cdot t\}$$

Where

variable		low dose	high dose
A	=	64.04 g	96.06 g
W	=	82.0 kg	82.0 kg
r	=	0.68	0.68
β	=	0.265 mgm% · minute^{-1}	0.265 mgm% · minute^{-1}
t	=	60.0 minutes	60.0 minutes

Then

C60	=	98.9 mgm%	156.3 mgm%
C480	=	0.0 mgm%	28.3 mgm%

Thus for both series, at the initiation of cold exposure, mean BAC under the low dose would have been expected to equal approximately 100 mgm%, and under the high dose BAC would have been expected to approximate 155 mgm%. At the termination of experiments some eight hours later, these BACs would have been expected to approximate 0 and 30 mgm% respectively for each dose.

Appendix 3. Utilization of Widmark's method to calculate approximate mean BACs for Haight and Keatinge's (1973) study

I. Utilization of Widmark's method to calculate approximate BACs for Haight and Keatinge's (1973) study.

A. From Haight and Keatinge's (1973) study;

amount of ethanol given = 30 ml 100% ethanol (v/v)

mean body weight of subjects = 152.5 lb

B. Conversion factors;

1.0 ml 100% ethanol (v/v) = 0.781 g 100% ethanol (v/v)

1.0 lb = 0.455 kg

C. Expected mean BACs at initiation of cold exposure (40 minutes post cessation of ingestion), and at termination of cold exposure (70 minutes post cessation of ingestion);

From

$$Ct = \{((A \cdot W^{-1}) \cdot 100) \cdot r^{-1} \} - \{ \beta \cdot t \}$$

Where

A = 23.43 g 100% ethanol (v/v)

W = 69.39 kg

r = 0.68

β = 0.265 mgm% $\cdot$ minute^{-1}

t = 40 minutes for initiation of cold exposure

70 minutes for termination of cold exposure

Then

C40 = 37.8 mgm% C70 = 29.8 mgm%

Thus expected mean BACs under the condition of Haight and Keatinge's (1973) study would have approximated 40 mgm% at the intiation of the cold exposure period, and 30 mgm% at the termination of the cold exposure period.

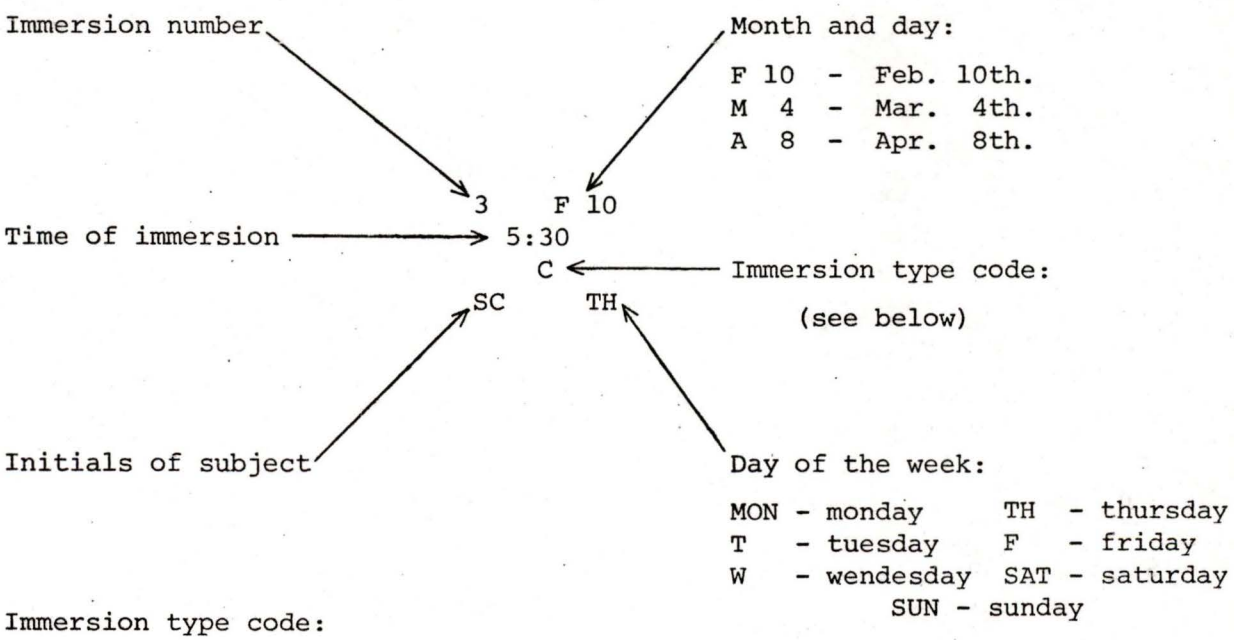
Appendix 4. Immersion scheduling

I. Scheduling

Schedule 1, presents the original master schedule which was to have been followed for the planned 60 immersions of the study.

Schedule 2, presents the master schedule which was actually followed during the 46 immersions performed in the study. Only 46 rather than the planned 60 immersions were completed since placebo type immersions were abandoned for all subjects except subjects SC, JS, and DP. Treatments were randomly assigned within each subject's block of immersions, and each subject only performed one control, al, ex, and alex type of immersion.

An explanation of the coding used within the cells of the two master schedules is given below:



Immersion type code:

A - pl (placebo non-exercise)
B - plex (placebo exercise)
C - al (alcohol non-exercise)
D - alex (alcohol exercise)
E - control (control non-exercise)
F - ex (control exercise)

Schedule I.

1 F 8 4:00 A JS T	4 F 11 4:30 B JS F	7 F 15 4:00 F JS T	10 F 18 4:30 D JS F	13 F 22 4:00 E JS T	16 F 25 4:30 C JS F
2 F 9 4:30 B DP W	5 F 12 1:00 A DP SAT	8 F 16 4:30 C DP W	11 F 19 1:00 F DP SAT	14 F 23 4:30 E DP W	17 F 26 1:00 D DP SAT
3 F 10 5:30 F SC TH	6 F 13 1:00 C SC SUN	9 F 17 5:30 B SC TH	12 F 20 1:00 A SC SUN	15 F 24 5:30 D SC TH	18 F 27 1:00 E SC SUN
19 M 1 4:00 E GP T	22 M 4 4:30 C GP F	25 M 8 4:00 D GP T	28 M 11 4:30 A GP F	31 M 15 4:00 B GP T	34 M 18 4:30 F GP F
20 M 2 4:30 A TM W	23 M 5 1:00 D TM SAT	26 M 9 4:30 C TM W	29 M 12 1:00 E TM SAT	32 M 16 4:30 B TM W	35 M 19 1:00 F TM SAT
21 M 3 5:30 F HD TH	24 M 6 1:00 A HD SUN	27 M 10 5:30 B HD TH	30 M 13 1:00 C HD SUN	33 M 17 5:30 E HD TH	36 M 20 1:00 D HD SUN
37 M 22 4:00 C JM T	41 M 26 1:00 F JM SAT	45 M 31 5:30 D JM TH	49 A 5 4:00 E JM T	53 A 9 1:00 A JM SAT	57 A 14 5:30 B JM TH
38 M 23 4:30 B HW W	42 M 27 1:00 F HW SUN	46 A 1 5:30 A HW F	50 A 6 4:30 E HW W	54 A 10 1:00 C HW SUN	58 A 15 5:30 D HW F
39 M 24 5:30 C RG TH	43 M 29 4:00 B RG T	47 A 2 1:00 A RG SAT	51 A 7 5:30 E RG TH	55 A 12 4:00 D RG T	59 A 16 1:00 F RG SAT
40 M 25 5:30 A PC F	44 M 30 4:30 F PC W	48 A 3 1:00 C PC SUN	52 A 8 5:30 A PC F	56 A 13 4:30 D PC W	60 A 17 1:00 B PC SUN

Schedule 2.

1 F 8 5:30 D JS T	11 F 19 1:00 B DP SAT	21 M 7 3:00 E SC MON	31 M 17 5:30 E MW TH	
2 F 9 4:30 E DP W	12 F 20 1:00 A SC SUN	22 M 8 5:30 D GP T	32 M 18 4:30 F PC F	
3 F 10 5:30 F SC TH	13 F 22 1:00 C JS T	23 M 9 5:30 D TM W	33 M 19 1:00 D HD SAT	41 M 29 5:00 C HD T
4 F 11 5:30 [illegible] JS F	14 F 23 4:30 F DP W	24 M 10 5:30 E JM TH	34 M 20 1:00 [illegible] RG SUN	42 M 30 4:30 C RG W
5 F 12 1:00 D DP SAT	15 F 24 5:30 C JS TH	25 M 11 3:00 A GP F	35 M 22 4:00 D MW T	43 M 31 5:30 F MW F
6 F 13 1:00 C SC SUN	16 F 26 1:00 A DP SAT	26 M 12 1:00 C TM SAT	36 M 23 4:30 C PC W	44 A 1 5:30 B PC F
7 F 15 5:30 A JS T	17 M 1 5:30 F GP F	27 M 13 1:00 C JM SUN	37 M 24 5:30 E HD TH	45 A 2 1:00 D JM SAT
8 F 16 4:30 C DP W	18 M 3 5:30 E TM TH	28 M 14 3:00 D SC MON	38 M 25 5:30 F RG F	46 A 3 1:00 D PC SUN
9 F 17 5:30 B JS TH	19 M 4 3:00 C GP F	29 M 15 5:00 F HD F	39 M 26 1:00 C MW SAT	
10 F 18 5:30 B SC F	20 M 5 1:00 F TM SAT	30 M 16 5:30 D RG W	40 M 28 1:30 F JM F	

Appendix 5. Calculation of the required amount of glucose to be added to placebo and control type drinks to achieve caloric compensation for ethanol of alcohol type drink

I. Determination of the required amount of glucose to provide caloric compensation for ethanol in placebo and control type drinks

According to Kalant (1971), the usual maximal rate of ethanol metabolism is 150 mg $\cdot$ kg^{-1} $\cdot$ $hour^{-1}$ in humans. Since the mean body weight of the subjects in the study was 71.6 kg, then a total of: (150 mg $\cdot$ kg^{-1} $\cdot$ $hour^{-1}$) $\cdot$ (71.6 kg) = 10734 mg $\cdot$ $hour^{-1}$ or, 10.734 g of ethanol would be metabolized by the subjects over a one hour period.

According to Goodman and Gilman (1965), the caloric equivalent of ethanol is 7.0 kcal $\cdot$ g^{-1}. Therfore:
(10.734 g $\cdot$ $hour^{-1}$) $\cdot$ (7.0 kcal $\cdot$ g^{-1}) = 75.138 kcal $\cdot$ $hour^{-1}$ or, a net caloric yield of 1.25 kcal $\cdot$ $minute^{-1}$, for ethanol metabolism.

Since drinking ceased at 45 minutes in all experiments, and the actual cold exposure period lasted from 90 to 135 minutes in all experiments, the subjects would have metabolized ethanol and produced heat at a rate of 1.25 kcal $\cdot$ $minute^{-1}$ over a 90 minute period in experiments. Thus the total expected caloric gain as a result of ethanol metabolism would approximate:
(1.25 kcal $\cdot$ $minute^{-1}$) $\cdot$ (90 minutes) = 112.5 kcal

According to Lenninger (1976), the caloric equivalent of glucose is 3.81 kcal $\cdot$ g^{-1}. Therefore to provide an equivalent amount of heat as a result of glucose metabolism:
(112.5 kcal) $\cdot$ (3.81 kcal g^{-1})$^{-1}$ = 29.5 g of glucose would be required. Thus assuming normal metabolic conditions, 29.5 g of glucose would have to be added to placebo and control type drinks in order to achieve caloric compensation for the amount of heat produced by the metabolism of ethanol in alcohol type drinks, during the 90 minute period of interest.

Appendix 6. Copy of instructions issued to all experimental subjects

INSTRUCTIONS FOR SUBJECTS:

Day prior to your immersion:

1. NO extended strenuous physical exercise
2. NO alcohol during the 24 hour period prior to your immersion
3. Minimum of 8 hours of sleep during night prior to immersion
4. Diet - Eat a normal well balanced diet

Day of your immersion:

1. Follow diet below;

   Breakfast - 1 bowl of cold cereal with milk
   1 egg and 2 slices of bacon
   2 slices of toast with jam

   Lunch - 1 bowl of thin soup (NO lunch if performing a week-end immersion)

2. NO heavily sugared drinks (eg. coffee, tea, soda pop ect.)
3. You must FAST from lunch to immersion time
4. Water is permitted *ad lib*
5. Void bladder 2½ hours prior to immersion time (arrive at lab with a full bladder)
6. Bring with you to lab:
   Bathing suit
   Towel
7. Try to arrive at lab at least 15 minutes prior to scheduled time
8. DO NOT bring spectators to the lab

Appendix 7. Results of statistical analysis performed on mean environmental temperatures observed under control, al, ex, and alex treatments

I. Statistical analysis performed for mean environmental temperatures under control, al, ex and alex treatments.

The significance of minor fluctuations of mean environmental temperatures observed amoung control, al, ex and alex treatments was determined by a series of t-tests completed between treatment pairs at various time isolates of the experiments. The results of these t-tests appear in Table I.

From Table I, it is evident that at the times tested, mean environmental temperatures between the different treatments did not significantly differ. It was concluded, therefore, that throughout the experimental time period, mean environmental temperatures were equivalent amoung all four treatments.

Table I. Results of t-test series performed on mean environmental temperatures, between treatment pairs for various times of control, al, ex, and alex experiments

Time isolate, minutes	Test conditions	t's	Significance
30	control versus al	-0.054	ns
	control versus ex	-0.304	ns
	control versus alex	0.577	ns
	al versus ex	-0.187	ns
	al versus alex	0.226	ns
	ex versus alex	0.369	ns
85	control versus al	-0.560	ns
	control versus ex	-0.647	ns
	control versus alex	-0.042	ns
	al versus ex	0.054	ns
	al versus alex	0.333	ns
	ex versus alex	0.326	ns
110	control versus al	1.597	ns
	control versus ex	0.219	ns
	control versus alex	0.923	ns
	al versus ex	-1.262	ns
	al versus alex	-0.767	ns
	ex versus alex	0.606	ns
140	control versus al	0.086	ns
	control versus ex	-0.845	ns
	control versus alex	1.521	ns
	al versus ex	-0.908	ns
	al versus alex	1.384	ns
	ex versus alex	2.838	ns
170	control versus al	-0.862	ns
	control versus ex	-1.385	ns
	control versus alex	-0.192	ns
	al versus ex	-0.430	ns
	al versus alex	0.637	ns
	ex versus alex	1.114	ns

ns = not significant at 0.01% level ($P > 0.01$)

Appendix 8. Some observations on the behavioural responses of subject PC while intoxicated under al treatment

I. Observations on subject PC's behavioural responses to cold water immersion while intoxicated under al treatment

1. At 20 minutes post ingestion, the subject fell off his chair.
2. The subject was placed back on his chair.
3. The subject fell off his chair for a second time in an uncontrollable laughing fit.
4. The subject was left on the floor until his laughter subsided (approximately 15 minutes).
5. The subject was placed back on his chair and remained quiet during the final ten minute period prior to his immersion.
6. The subject then stood up, put on a weight belt and got into the cold water tank.
7. While immersed, the subject talked, laughed and tried extremely hard to remain alert.
8. The subject reported he did not feel cold and that his time sense was disrupted (i.e. time seemed to pass very quickly).
9. The subject passed out six times during the immersion (the normal length of a "black-out" was approximately 40-50 seconds). During these black-outs, the subject required aid from the experimentors to avoid slipping under the water.
10. It was noted that as the subject cooled, the black-outs became both more frequent and of longer duration.
11. The subject talked continuously throughout the immersion,(it was noted that if he stopped talking, the likelyhood of him having a black-out increased).
12. The subject left the cold water at the 135 minute mark with only a minimum of aid from the experimentors.

13. At the rewarm bath the subject collapsed.
14. The subject was placed into the rewarm bath.
15. After one to two minutes in the rewarm bath the subject regained consciousness.
16. After two to three minutes in the rewarm bath the subject experienced a violent shivering bout lasting five minutes.
17. It was noted that the subject did not shiver while immersed in the cold water tank.
18. After his shivering bout in the rewarm bath, the subject became docile and drowsy.
19. The subject passed out once more in the rewarm bath.
20. However, at the termination of the experiment the subject appeared normal and rational but reported being exhausted.
21. Blood Alcohol Levels at various times throughout subject PC's al immersion.

time, minutes	BAC, mgm%
0	0
55	95
85	90
115	80
140	55
160	50
180	50

Appendix 9. Utilization of Widmark's method to calculate an estimate of the dose of 95% ethanol:distilled water (v/v) required to produce a BAC approximating 80 mgm% in subjects

I. Utilization of Widmark's method to calculate the estimated dose of 95% ethanol (v/v) required to achieve a BAC approximating 80 mgm%.

A. Mean weight of subjects = 72.0 kg

Desired time post consumption at which BAC should equal 80 mgm% = 60 minutes (note t = 60 minutes, i.e. 20 minute consumption period + 40 minute post consumption period).

B. From

$$Ct = \{((A \cdot W^{-1}) \cdot 100) \cdot r^{-1} \} - \{ B \cdot t \}$$

where A equals the amount of ethanol consumed.

Then rearranging for A

$$A = \{((Ct + (B \cdot t) \cdot 100^{-1}) \cdot r) \cdot W \}$$

Where

$Ct = 80$ mgm%

$B = 0.267$ mgm% $\cdot$ minutes^{-1}

$t = 60$ minutes

$r = 0.68$

$W = 72.0$ kg

Then

$A = 47.01$ gm 100% (v/v) ethanol

or

$$A = (47.01 \text{ gm}) \cdot (0.781 \text{ gm} \cdot \text{ml}^{-1})^{-1} = 60.20 \text{ ml } 100\% \text{ ethanol}$$

or

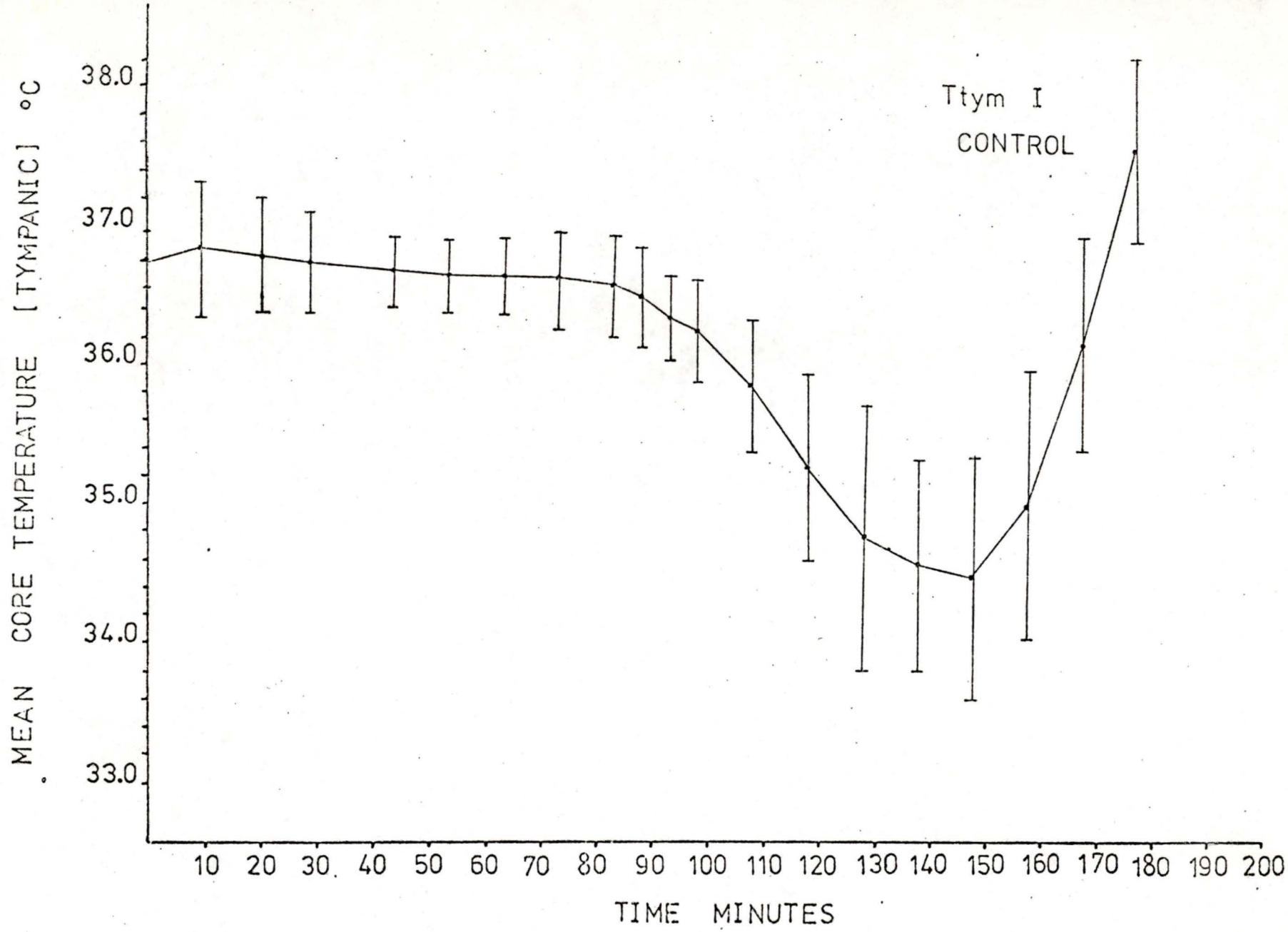
$$A = (60.20 \text{ ml}) + ((60.20 \text{ ml} \cdot 100^{-1}) \cdot 5) = 63.21 \text{ ml } 95\% \text{ ethanol}$$

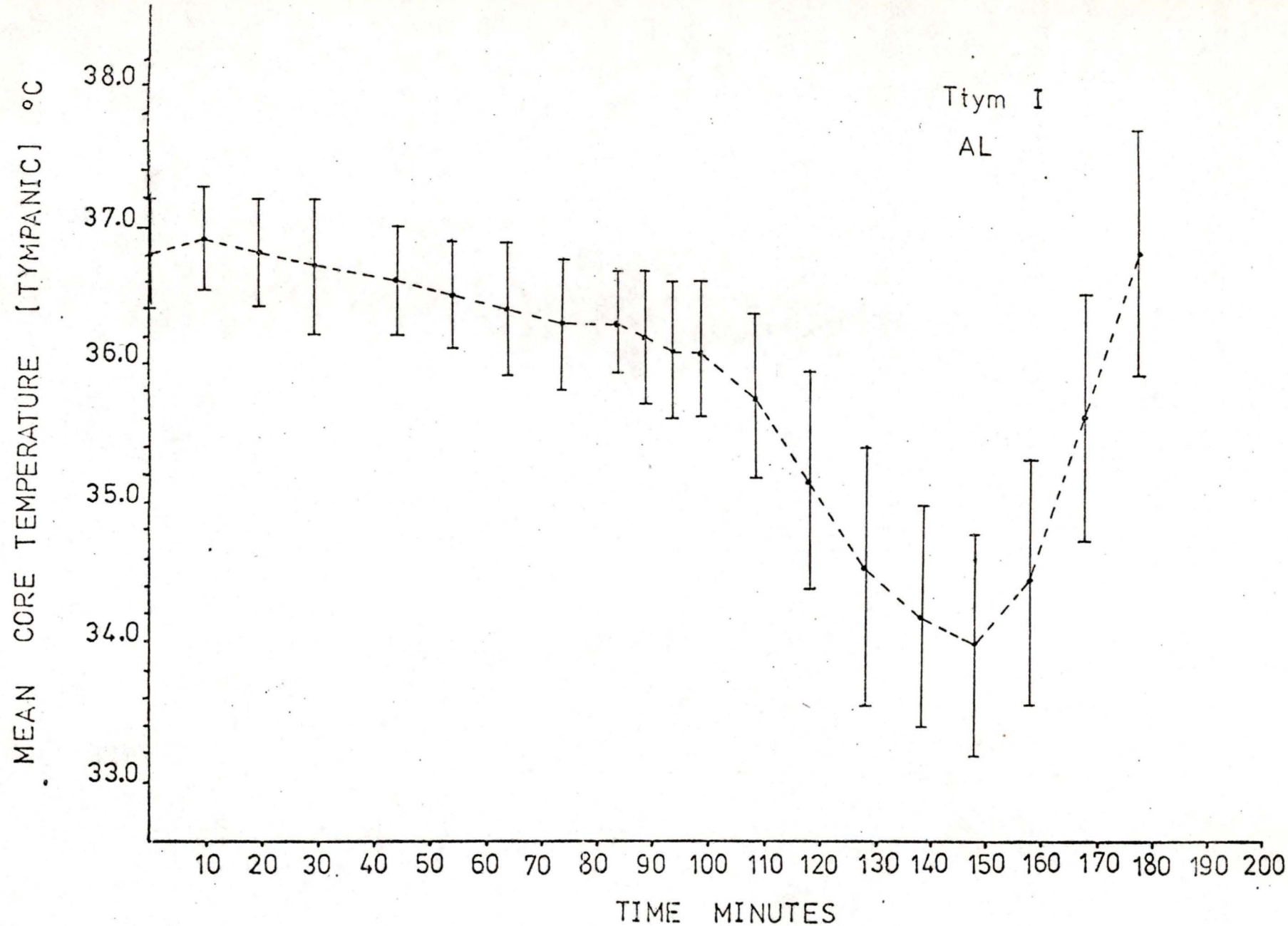
or a dose equivalent to

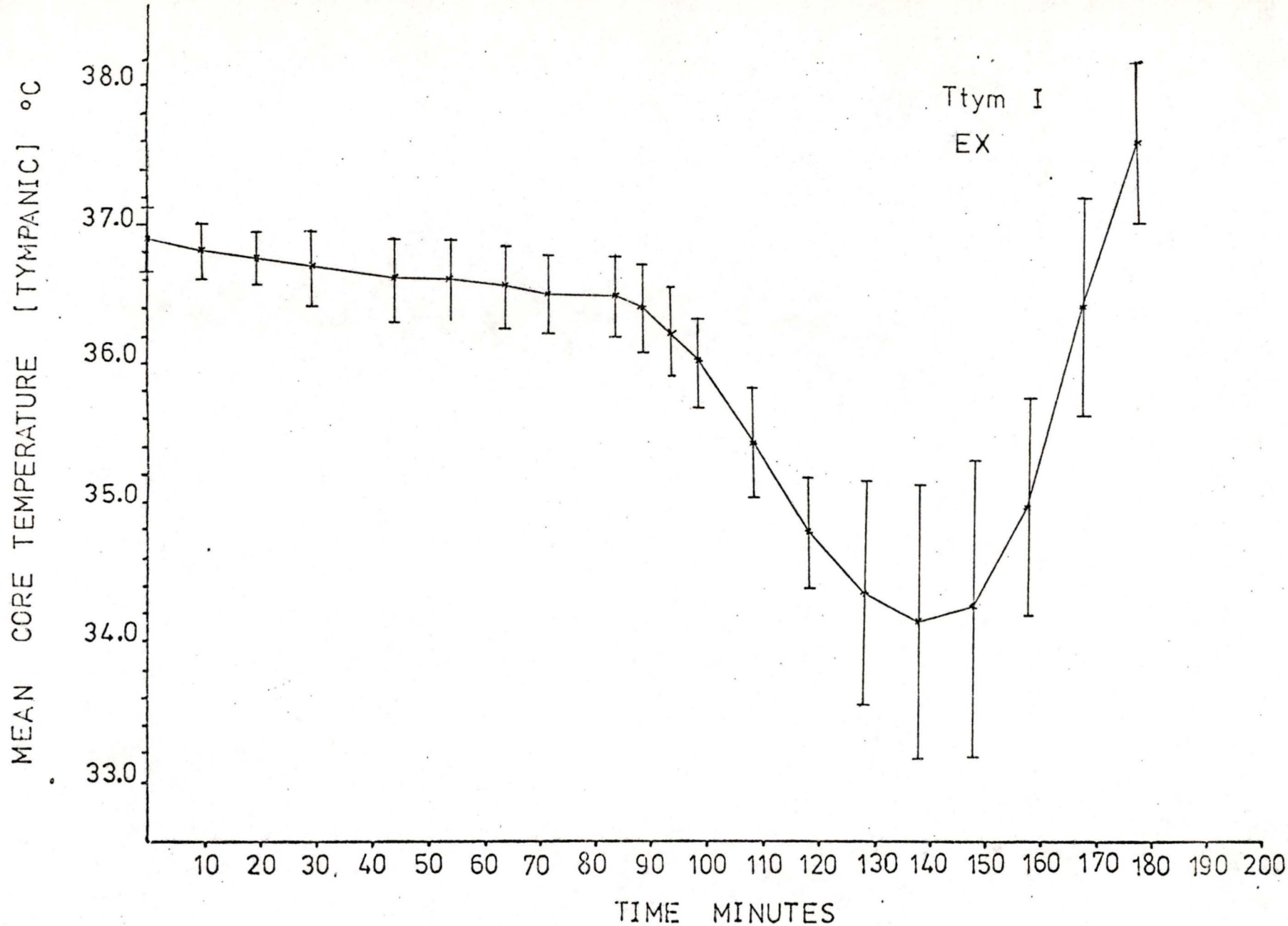
$$63.21 \text{ ml} \cdot 82.0 \text{ kg}^{-1} = 0.88 \text{ ml} \cdot \text{kg}^{-1}$$

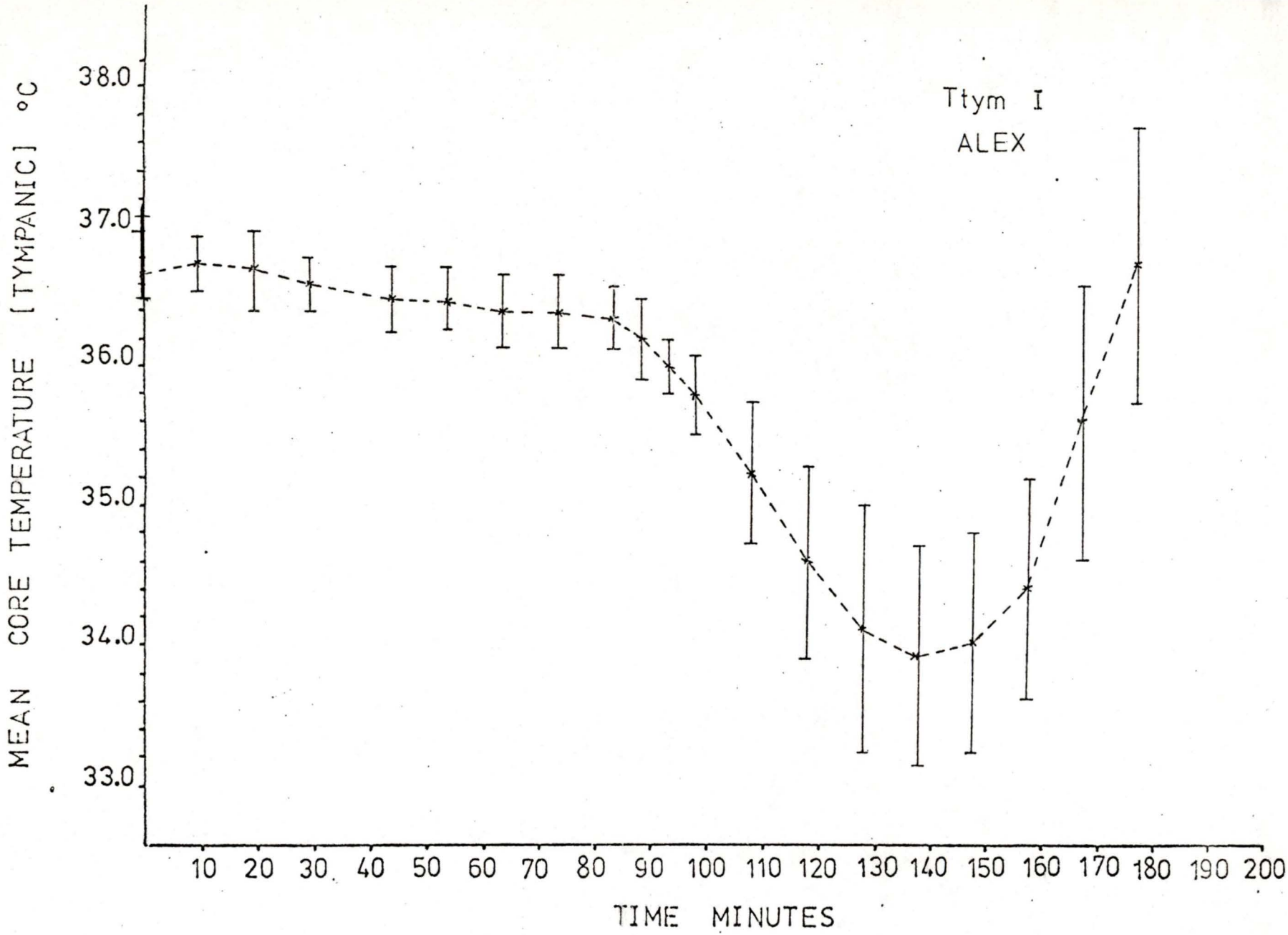
C. Therefore, a dose approximating 0.90 ml 95% ethanol · kg body weight^{-1} would be expected to produce a BAC of 80 mgm% 60 minutes after the beginning of drinking.

Appendix 10. Single treatment graphs used to construct the composite graph of Figure 3

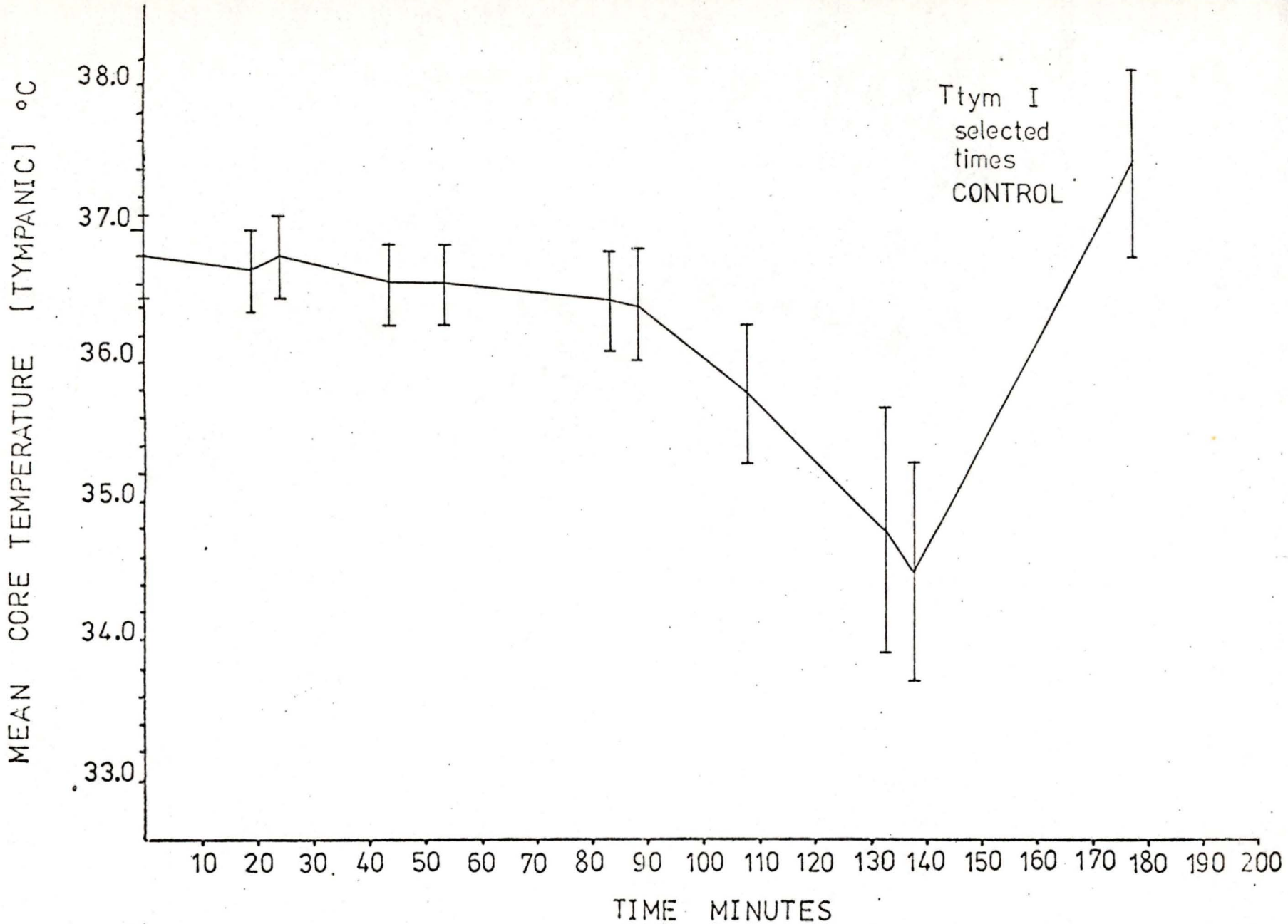

Ttym I
CONTROL
MEAN CORE TEMPERATURE [TYMPANIC] °C
38.0
37.0
36.0
35.0
34.0
33.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

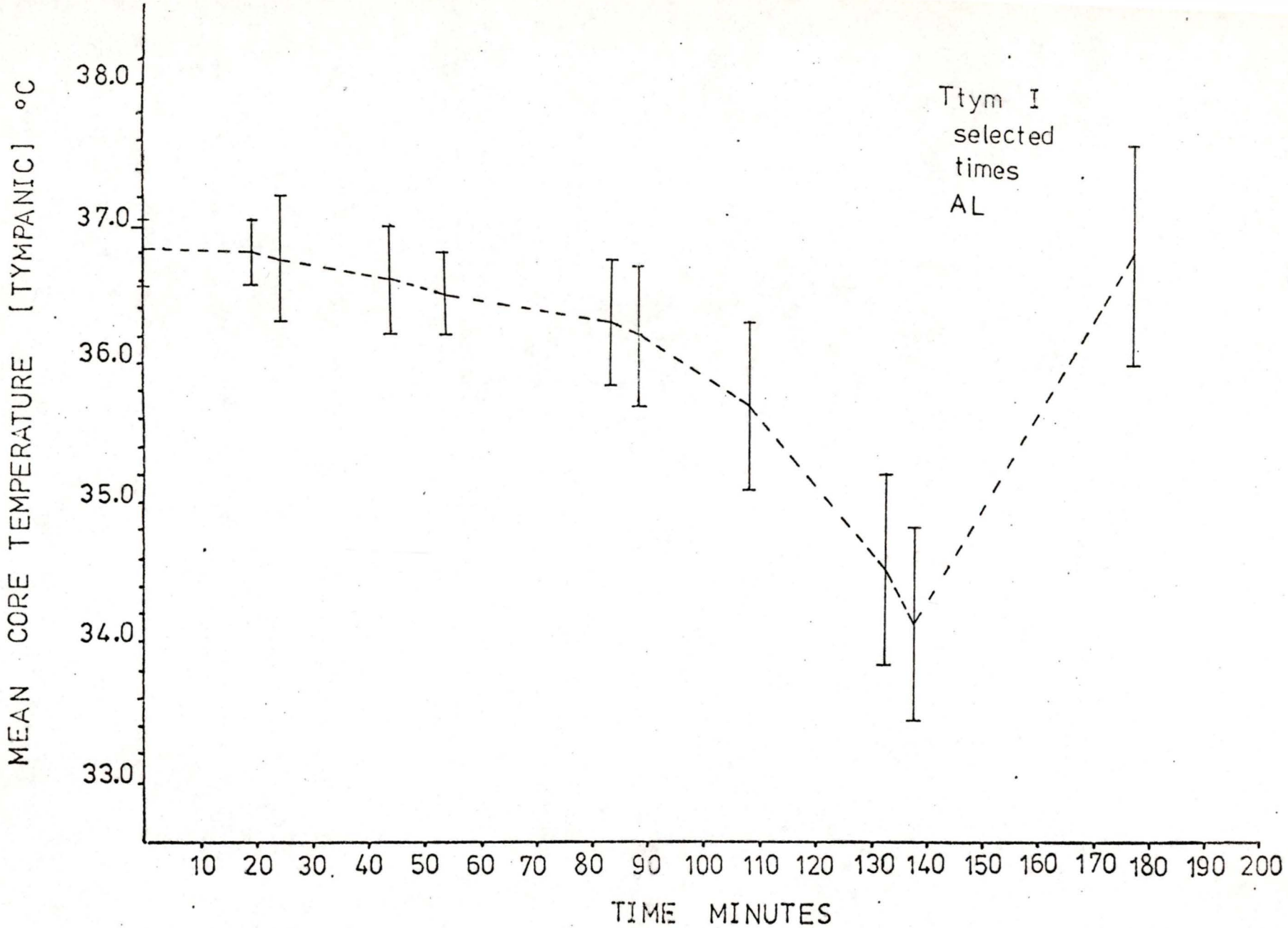

Ttym I
AL
MEAN CORE TEMPERATURE [TYMPANIC] °C
38.0
37.0
36.0
35.0
34.0
33.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

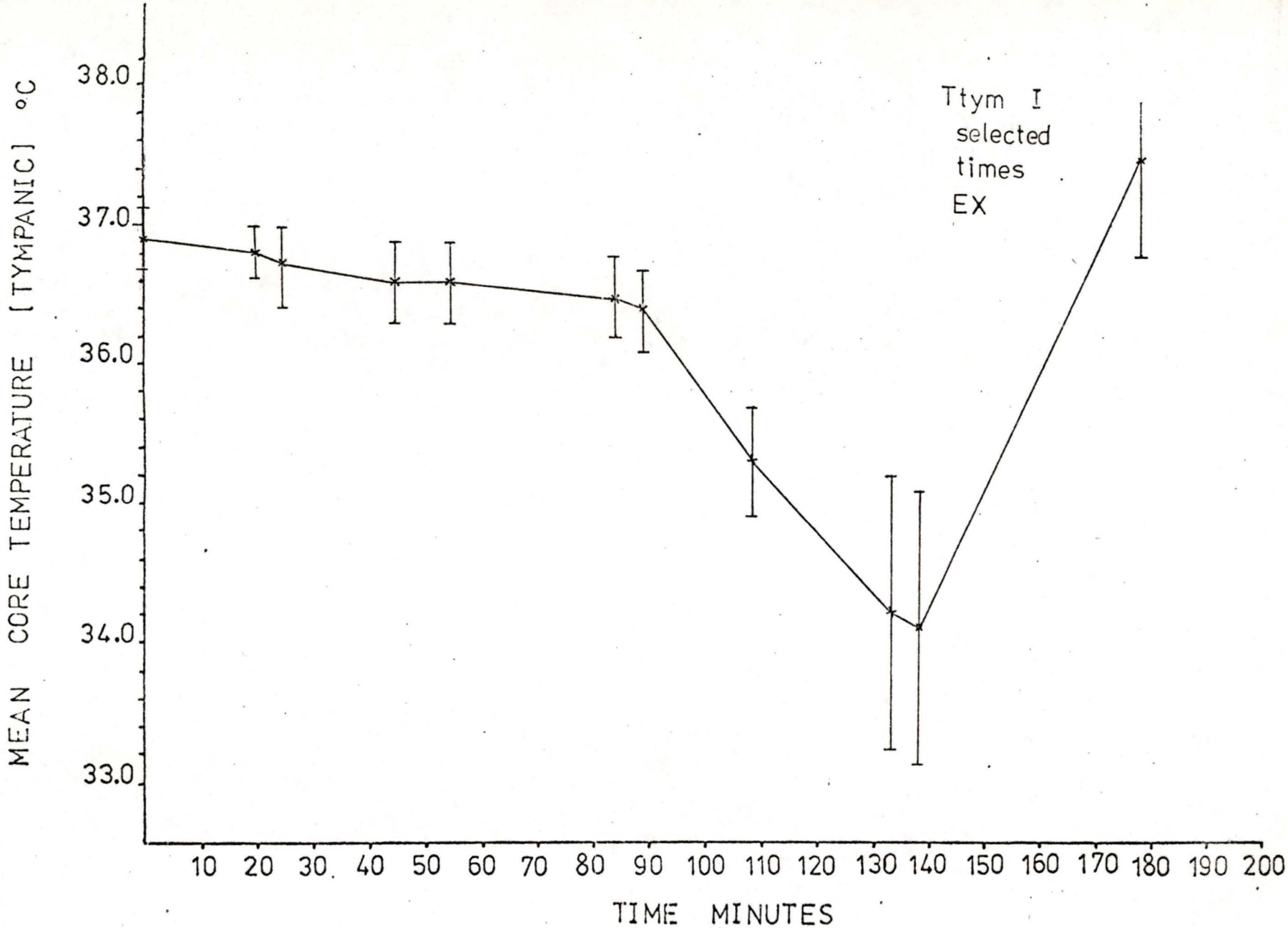

MEAN CORE TEMPERATURE [TYMPANIC] °C
38.0
37.0
36.0
35.0
34.0
33.0
Ttym I
EX
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

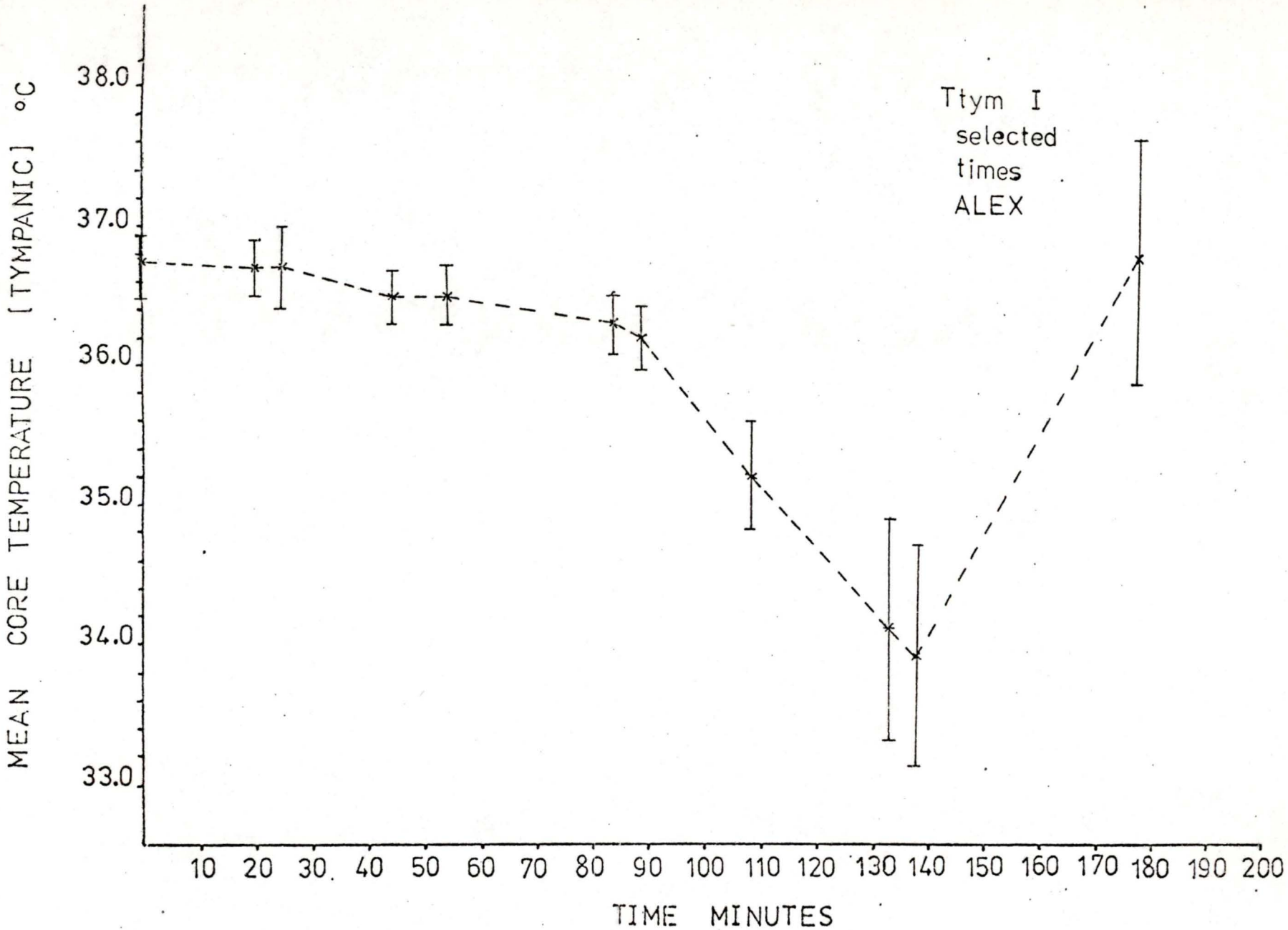

Ttym I
ALEX
MEAN CORE TEMPERATURE [TYMPANIC] °C
38.0
37.0
36.0
35.0
34.0
33.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

Appendix 11. Single treatment graphs used to construct the composite graph of Figure 4

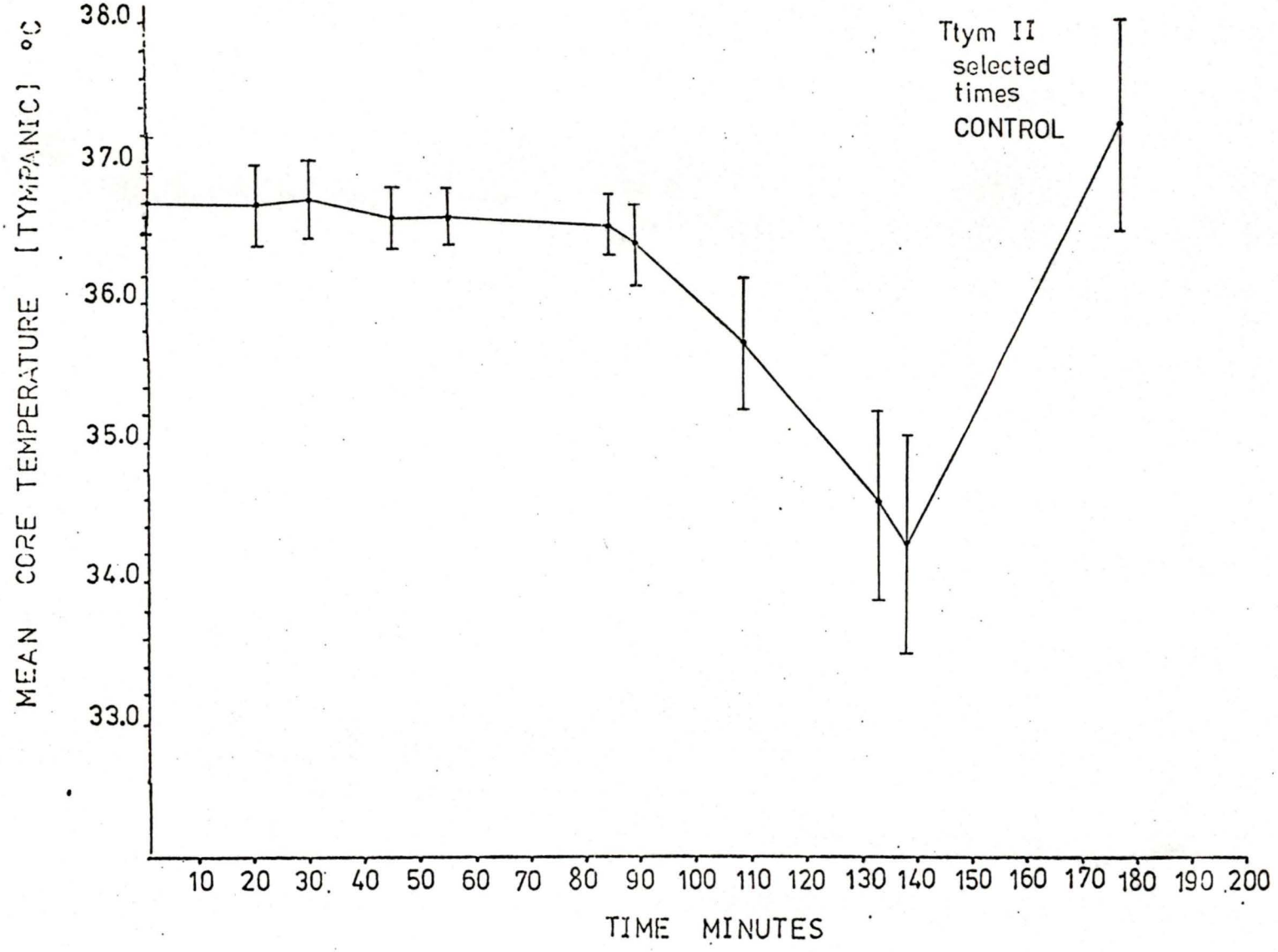

MEAN CORE TEMPERATURE [TYMPANIC] °C
38.0
37.0
36.0
35.0
34.0
33.0
Ttym I
selected
times
CONTROL
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

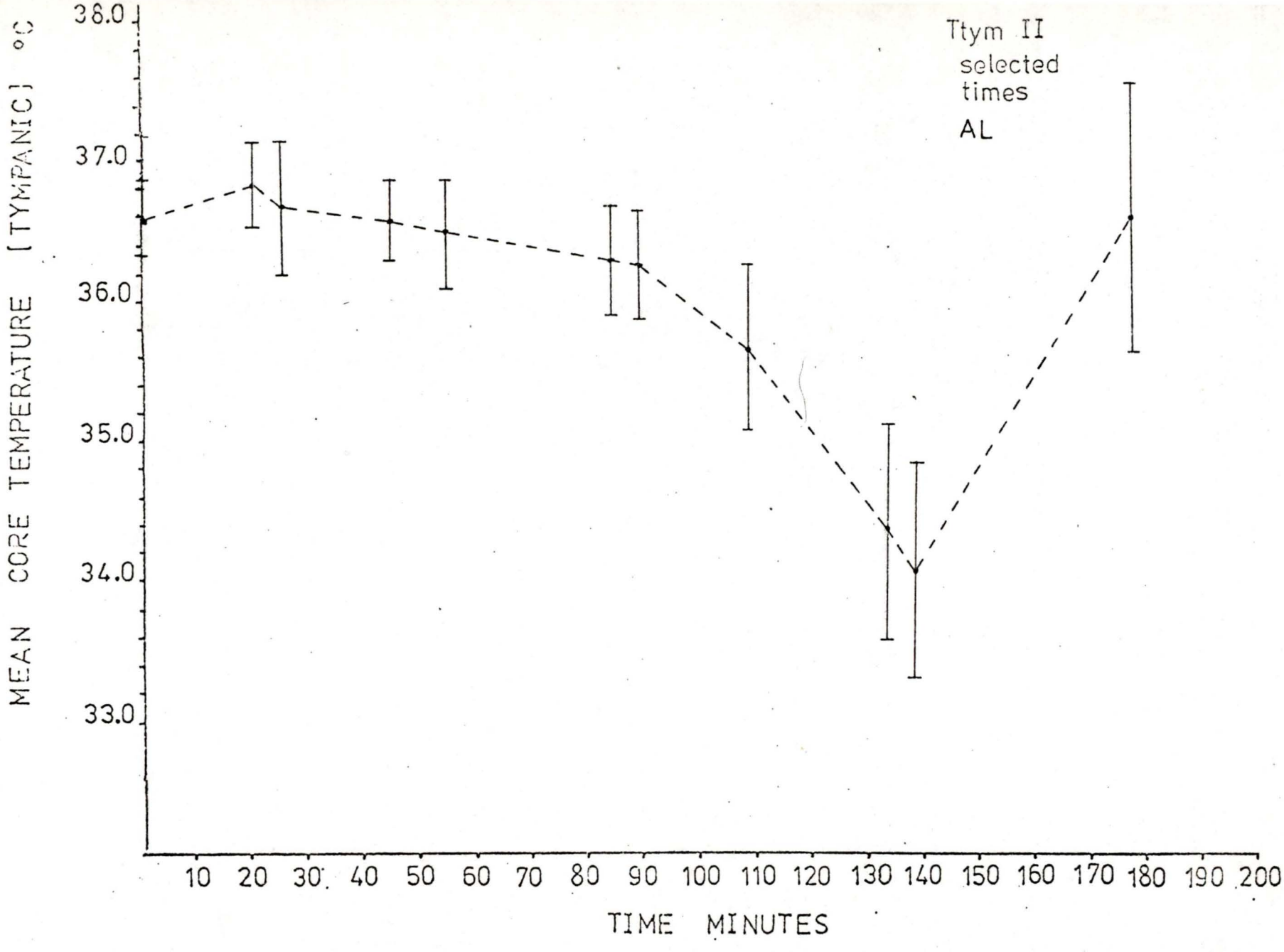

MEAN CORE TEMPERATURE [TYMPANIC] °C
38.0
37.0
36.0
35.0
34.0
33.0
Ttym I
selected
times
AL
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

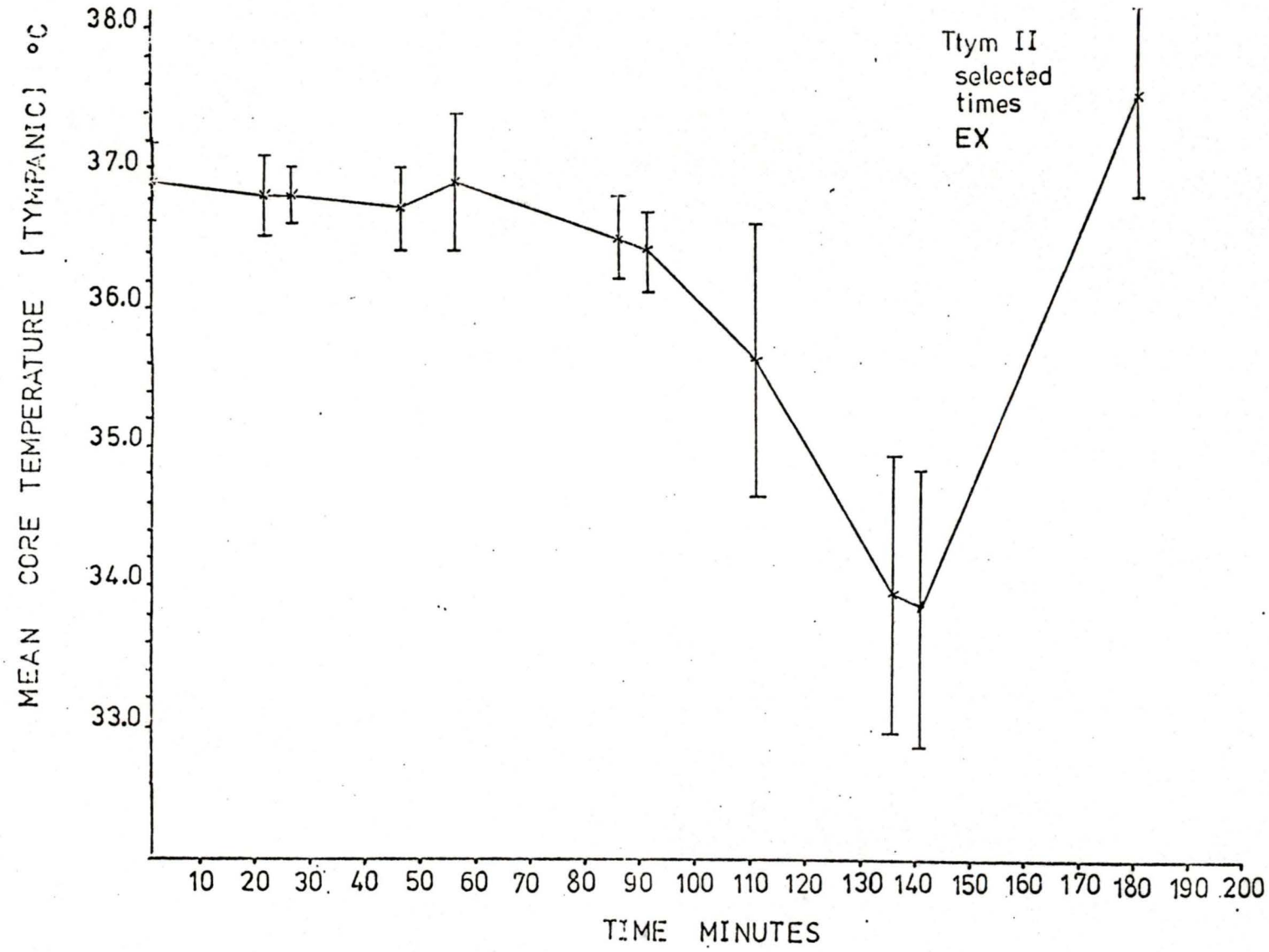

MEAN CORE TEMPERATURE [TYMPANIC] °C
38.0
37.0
36.0
35.0
34.0
33.0
Ttym I
selected
times
EX
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

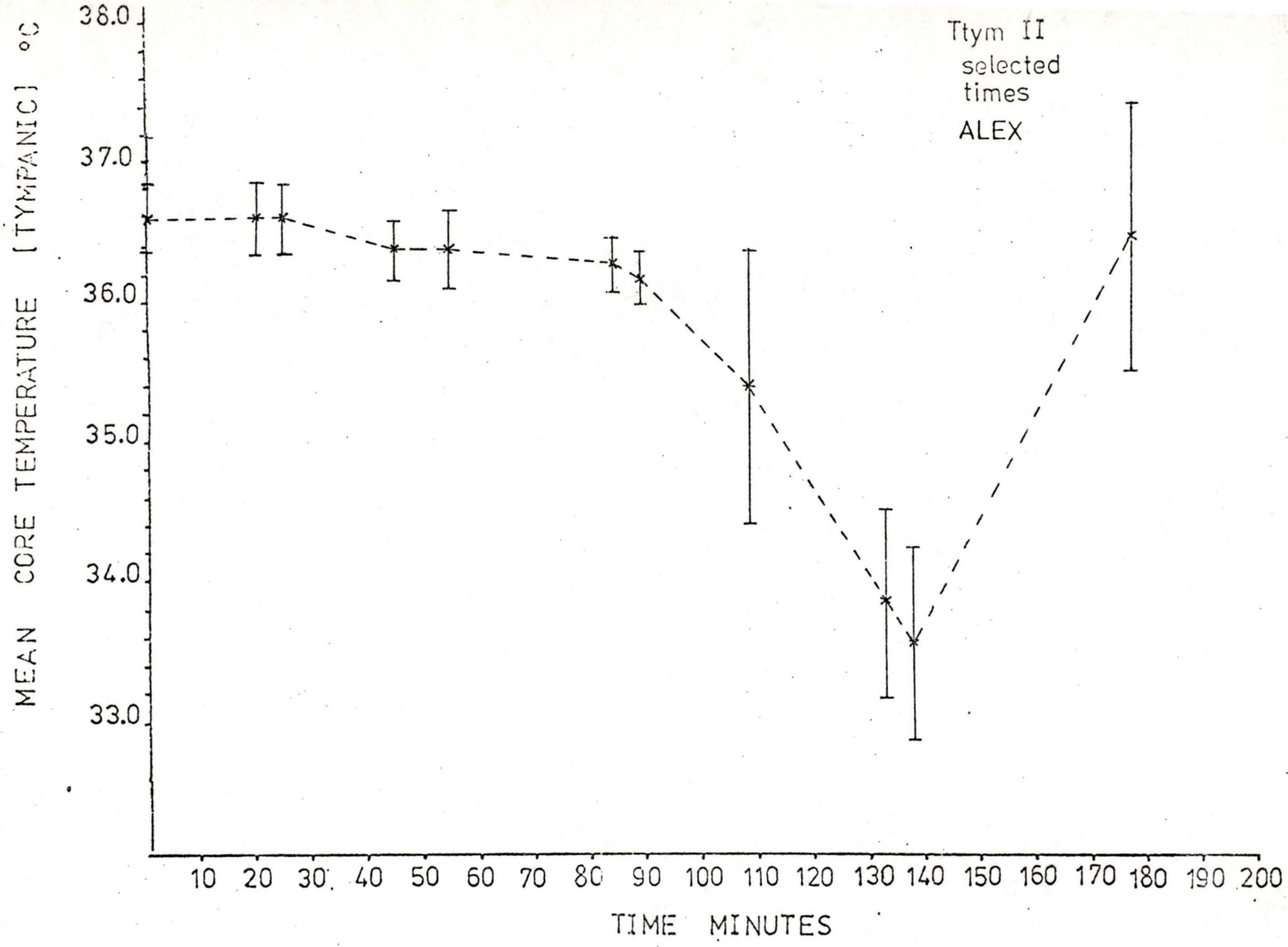

MEAN CORE TEMPERATURE [TYMPANIC] °C
38.0
37.0
36.0
35.0
34.0
33.0
Ttym I
selected
times
ALEX
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

Appendix 12. Single treatment graphs used to construct the composite graph of Figure 6

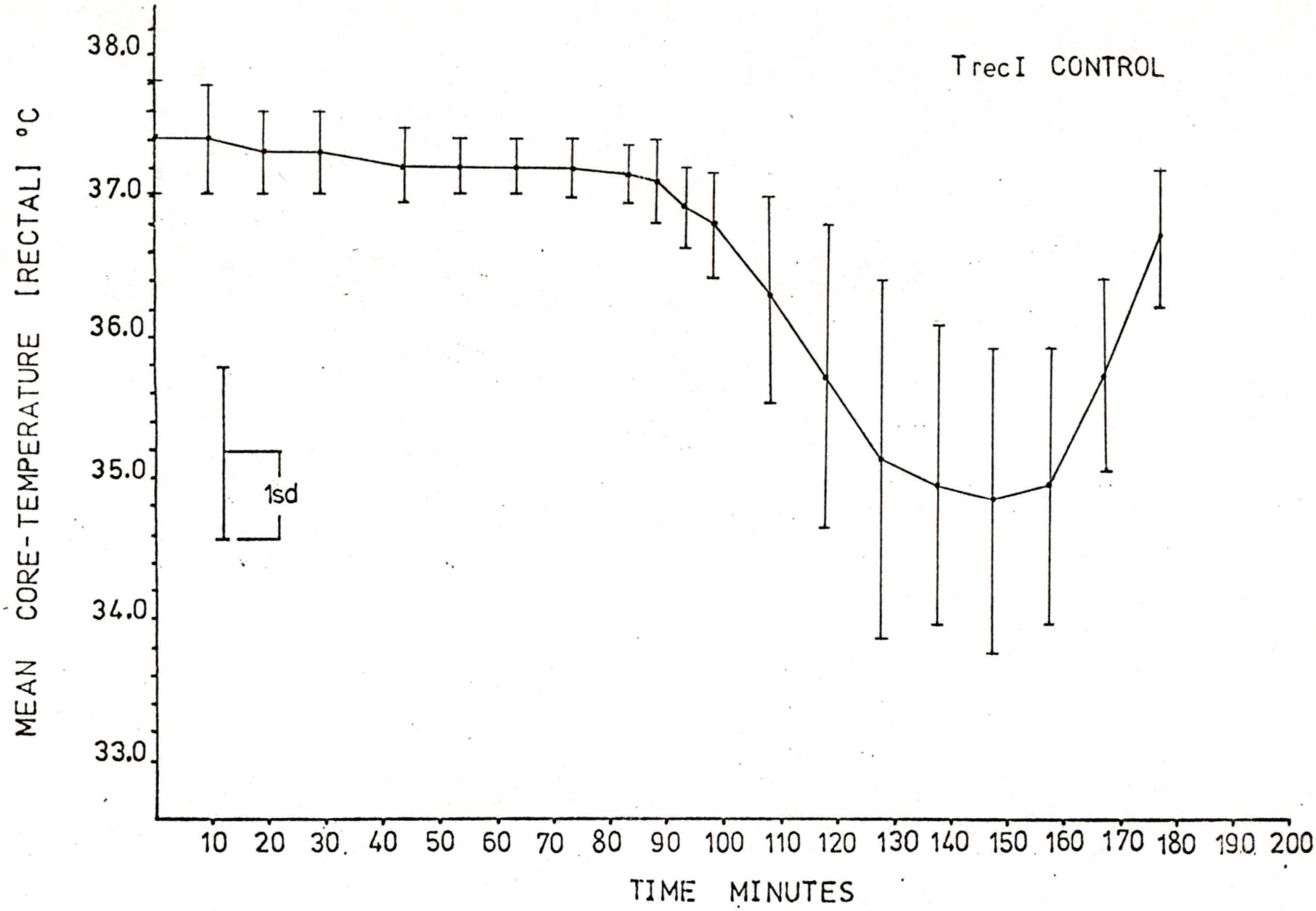

MEAN CORE TEMPERATURE [TYMPANIC] °C
38.0
37.0
36.0
35.0
34.0
33.0
Ttym II
selected
times
CONTROL
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

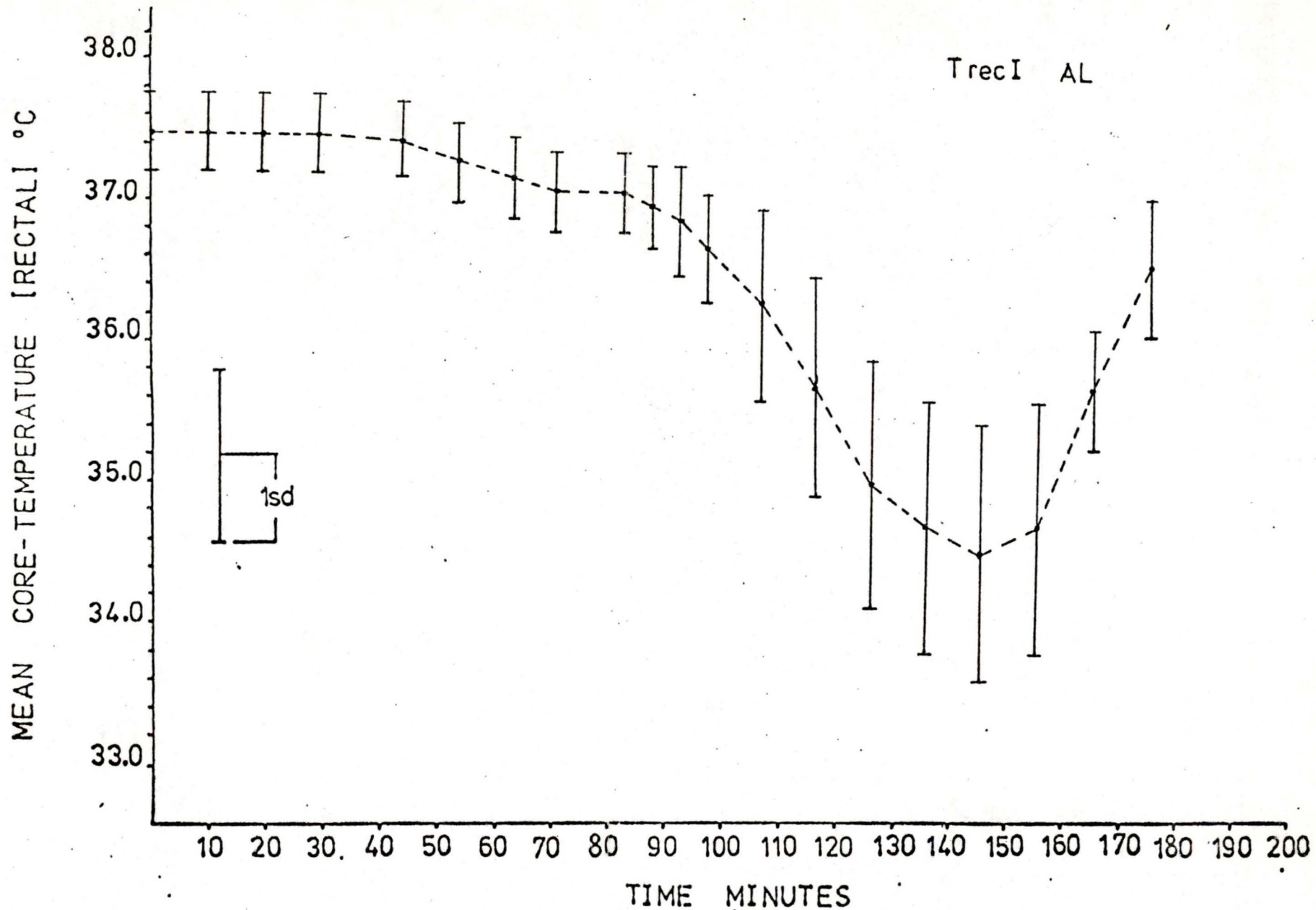

MEAN CORE TEMPERATURE (TYMPANIC) °C
38.0
37.0
36.0
35.0
34.0
33.0
Ttym II
selected
times
AL
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

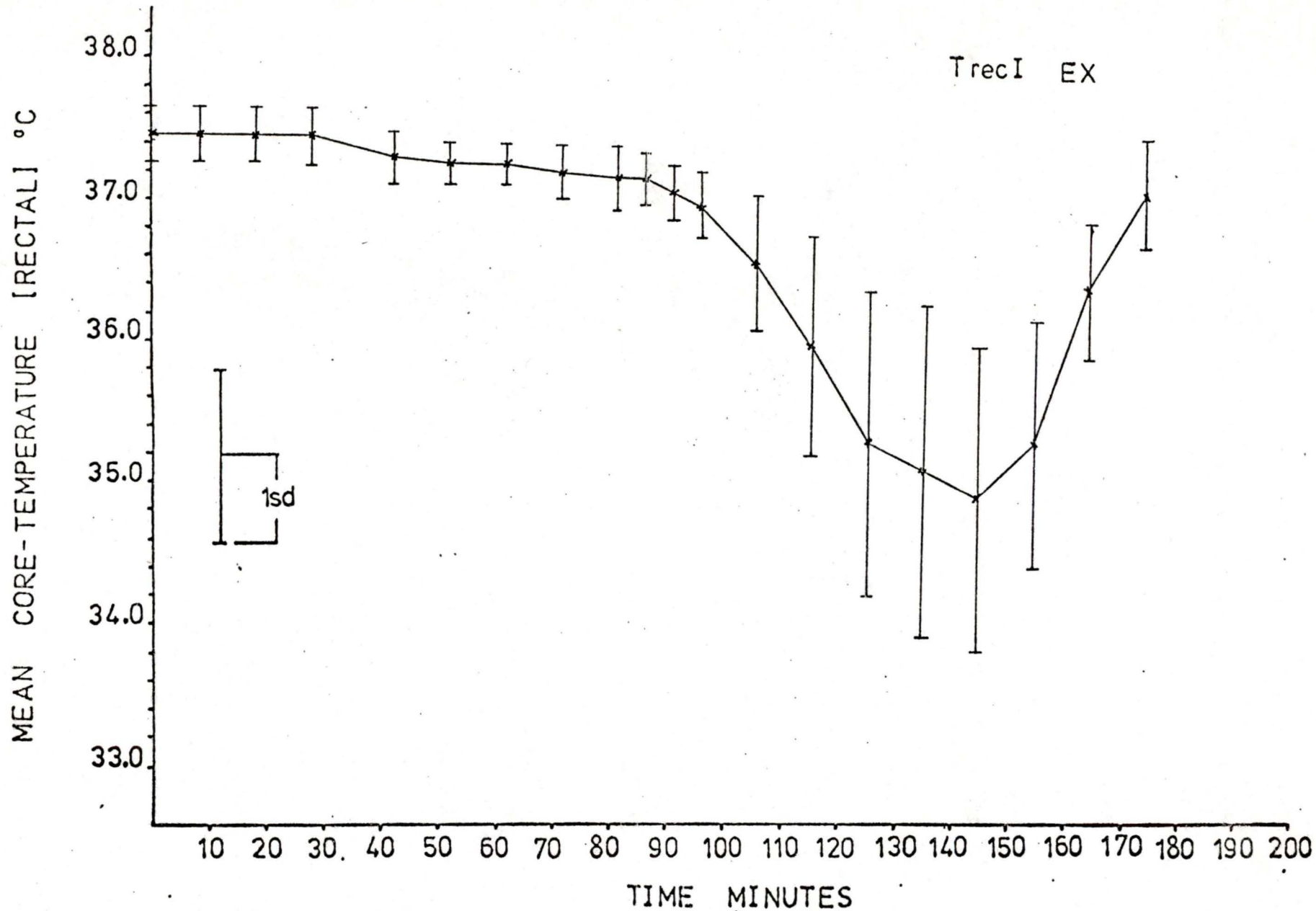

MEAN CORE TEMPERATURE [TYMPANIC] °C
38.0
37.0
36.0
35.0
34.0
33.0
Ttym II
selected
times
EX
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

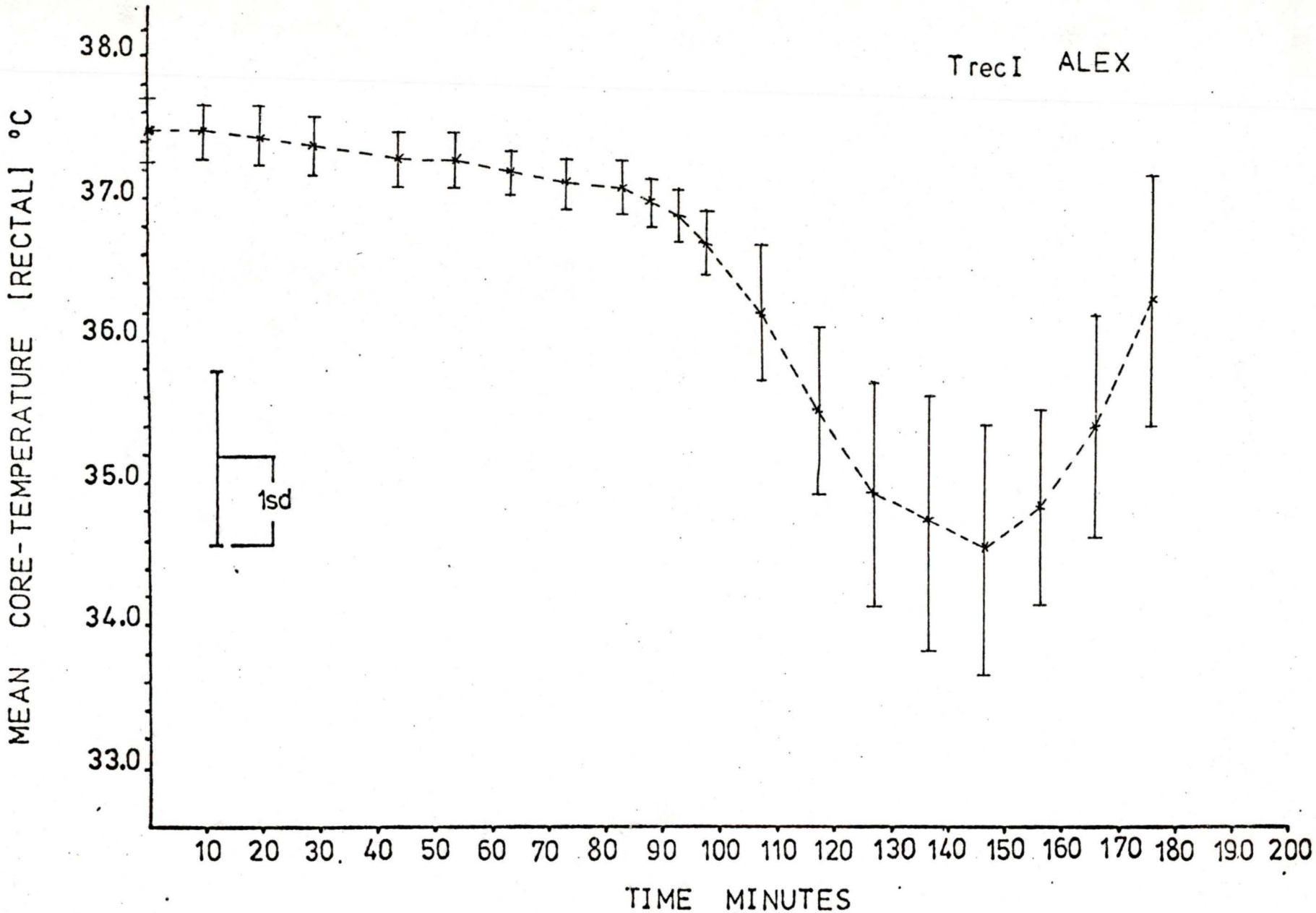

Ttym II
selected
times
ALEX
MEAN CORE TEMPERATURE (TYMPANIC) °C
38.0
37.0
36.0
35.0
34.0
33.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

Appendix 13. Single treatment graphs used to construct the composite graph of Figure 7

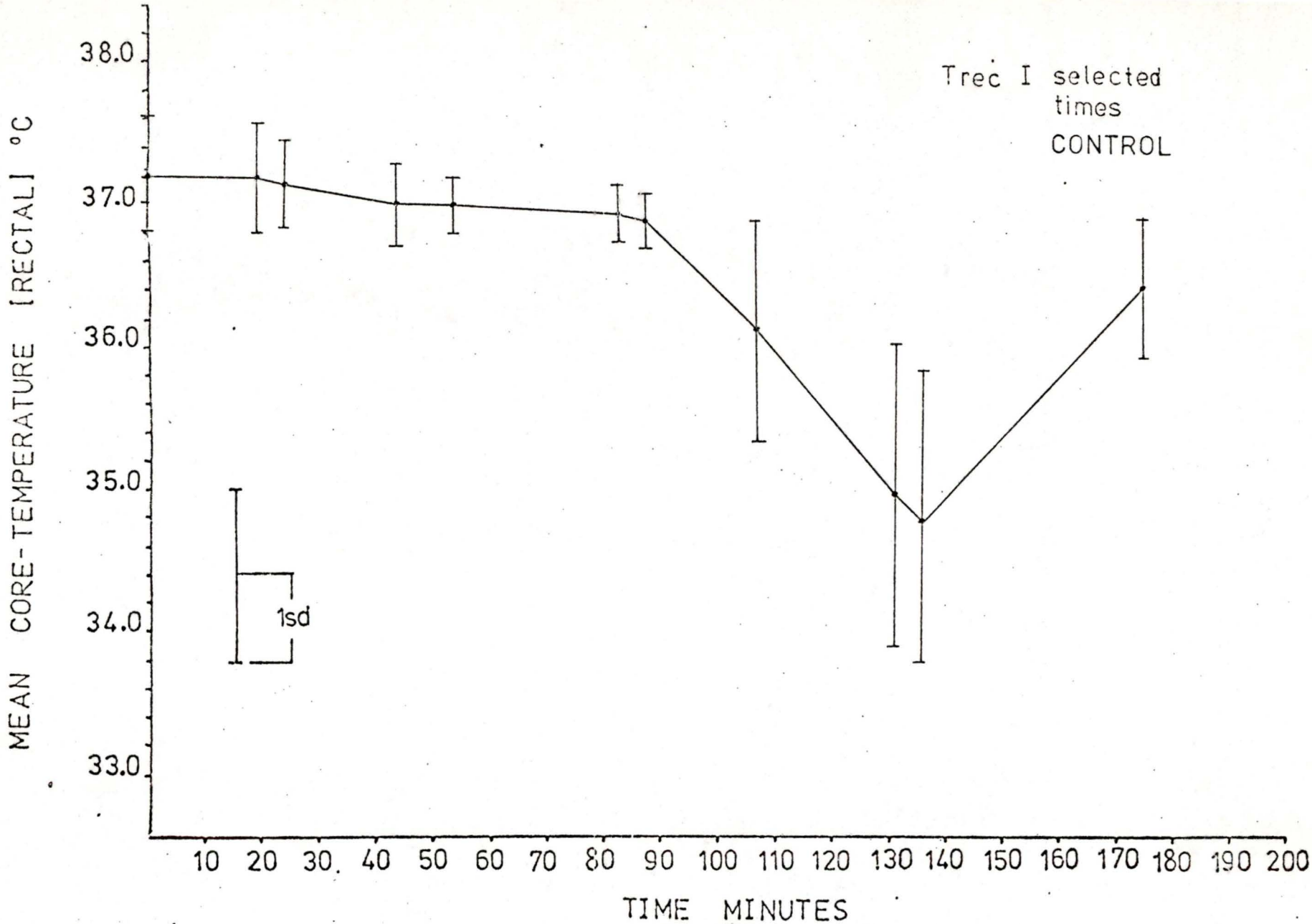

TrecI CONTROL
MEAN CORE-TEMPERATURE [RECTAL] °C
38.0
37.0
36.0
35.0
34.0
33.0
1sd
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

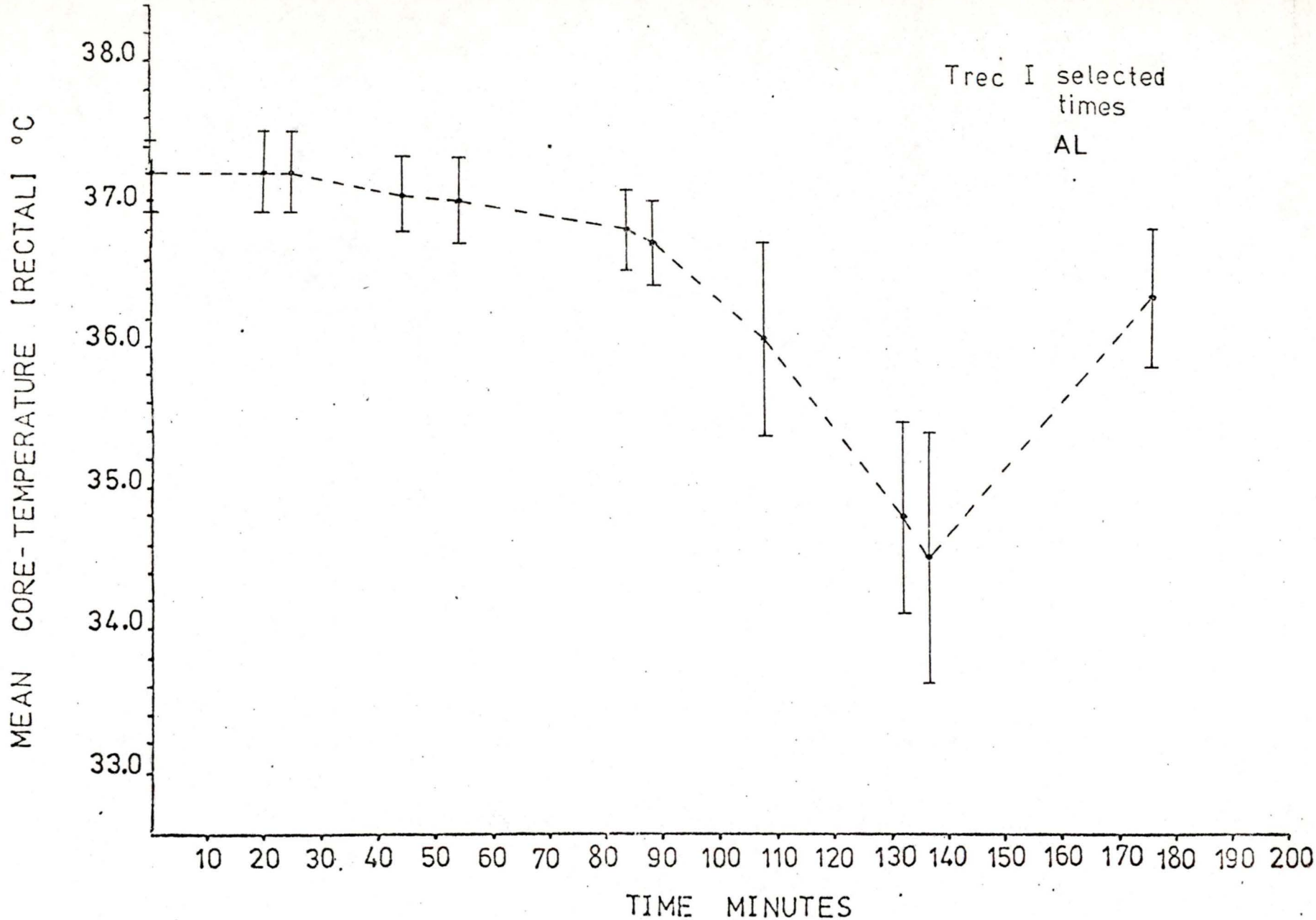

TrecI AL
MEAN CORE-TEMPERATURE [RECTAL] °C
38.0
37.0
36.0
35.0
34.0
33.0
1sd
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

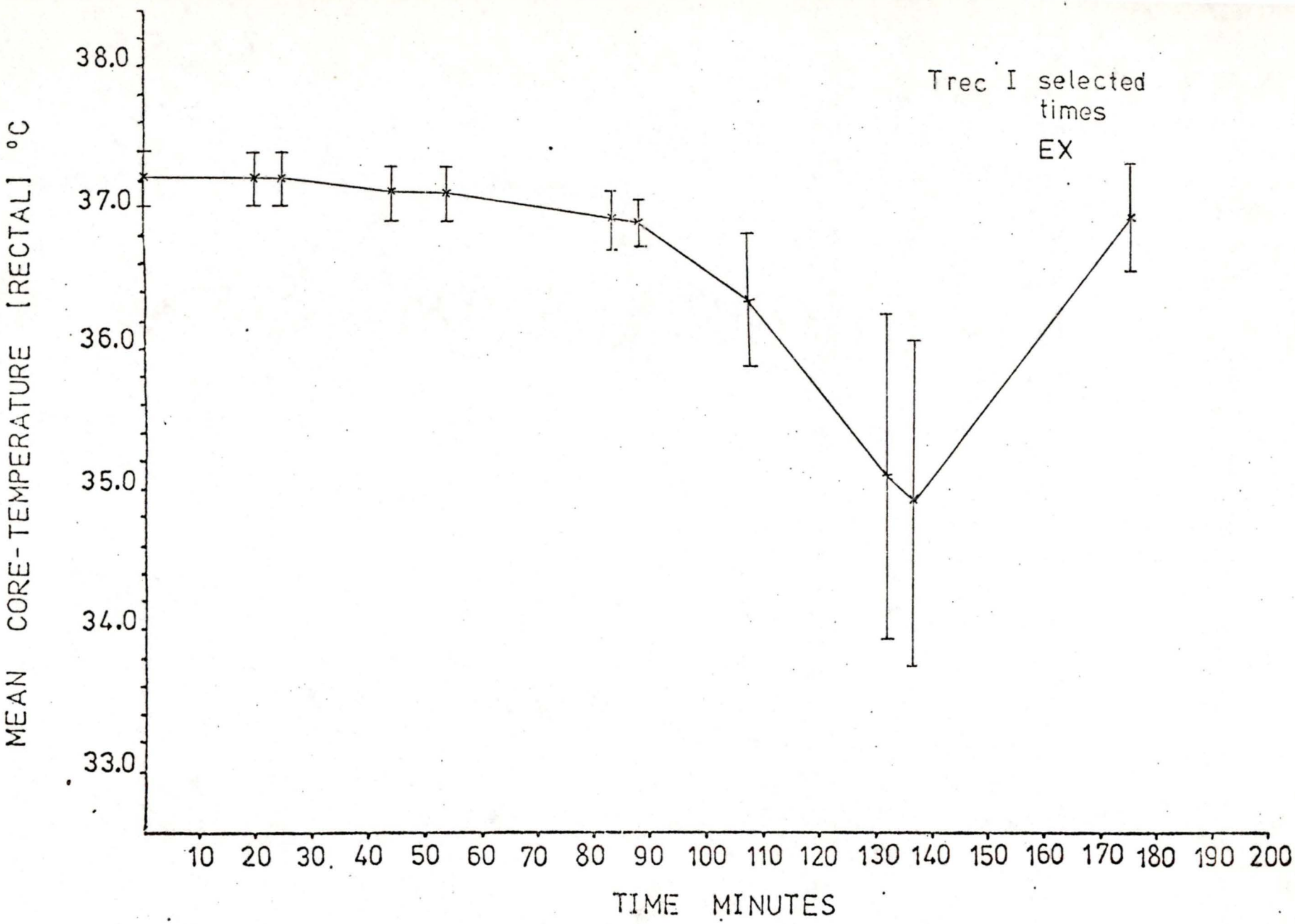

TrecI EX
MEAN CORE-TEMPERATURE [RECTAL] °C
38.0
37.0
36.0
35.0
34.0
33.0
1sd
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

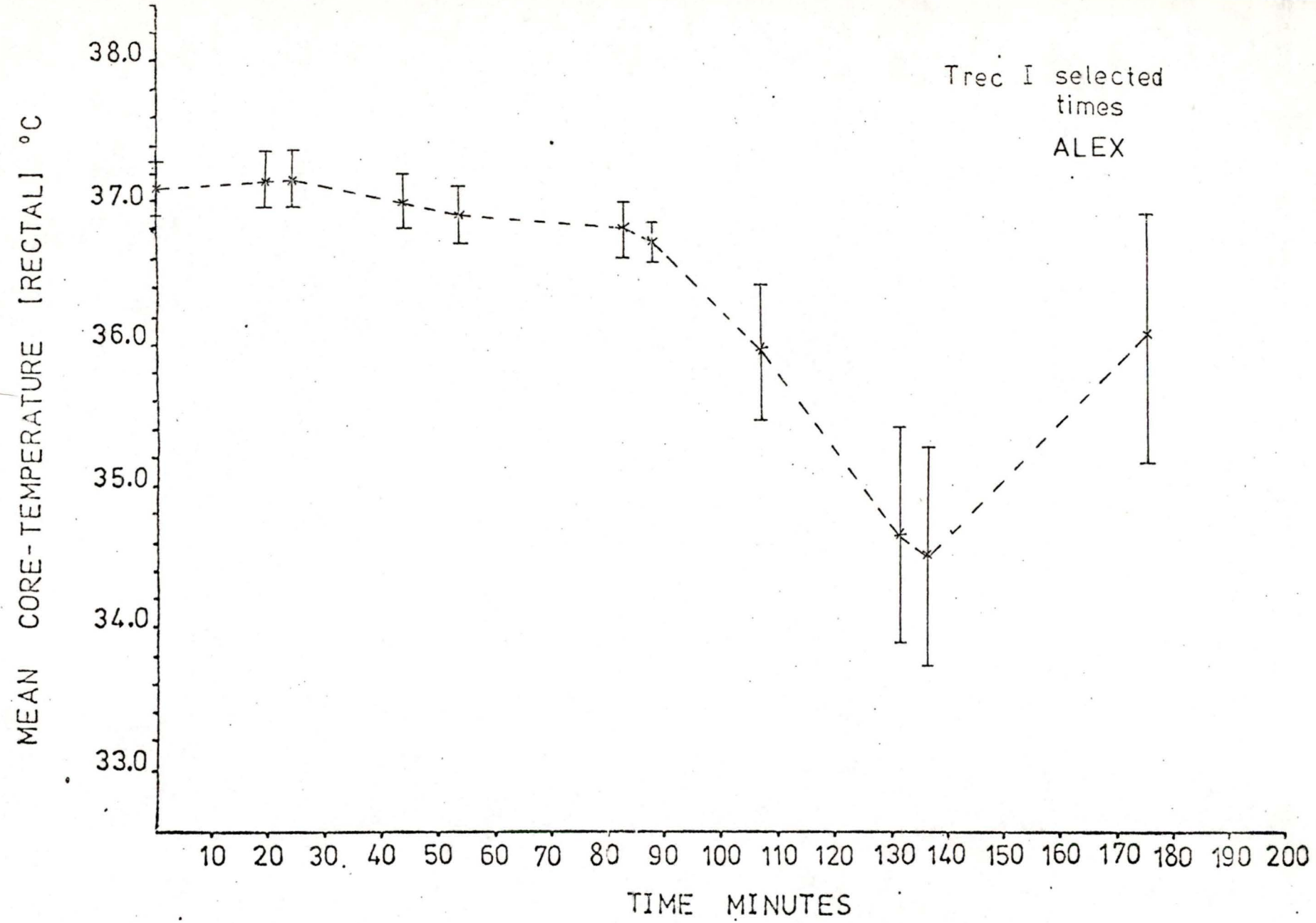

TrecI ALEX
MEAN CORE-TEMPERATURE [RECTAL] °C
38.0
37.0
36.0
35.0
34.0
33.0
1sd
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

Appendix 14. Single treatment graphs used to construct the composite graph of Figure 8

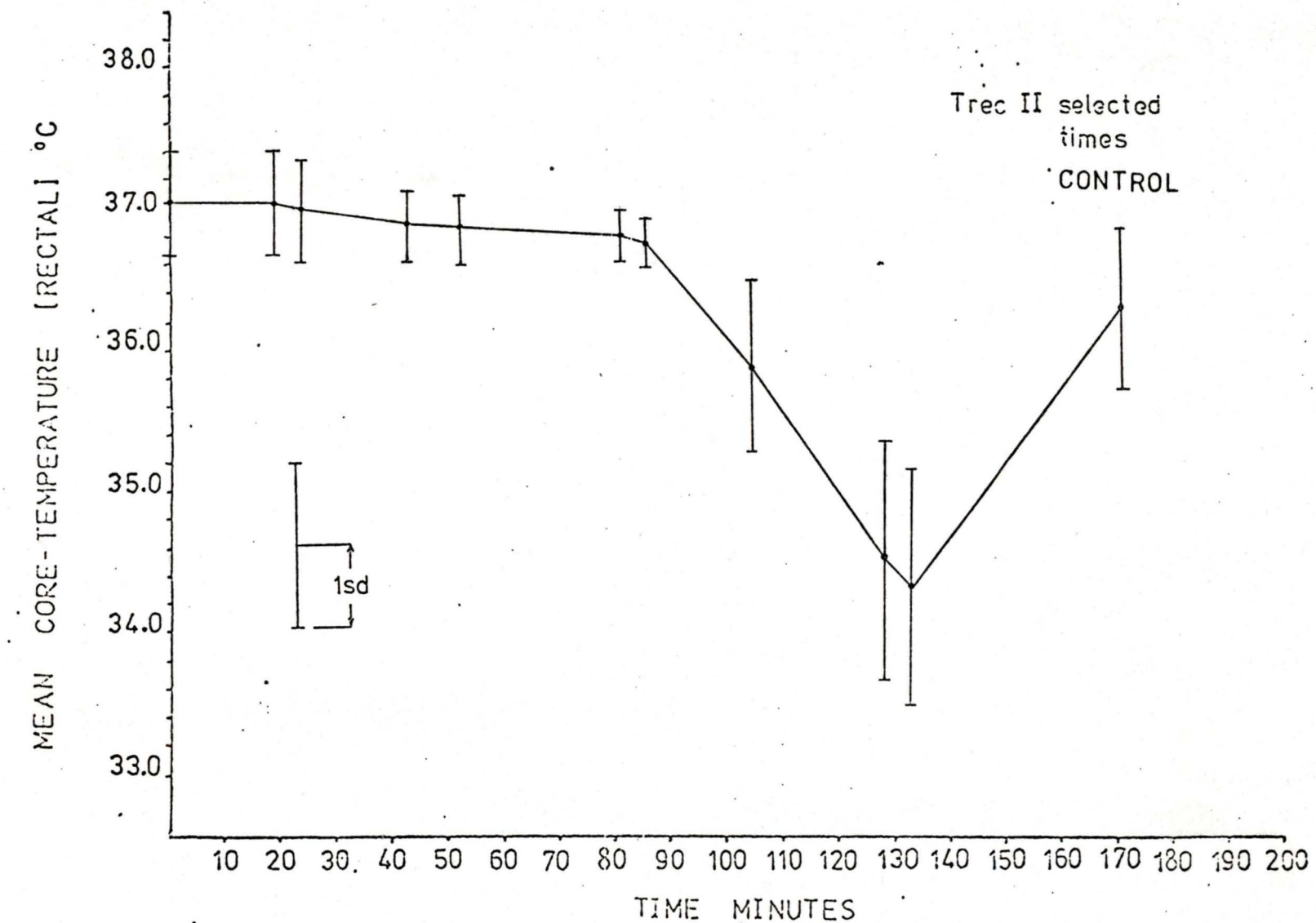

Trec I selected times
CONTROL
MEAN CORE-TEMPERATURE [RECTAL] °C
38.0
37.0
36.0
35.0
34.0
33.0
1sd
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

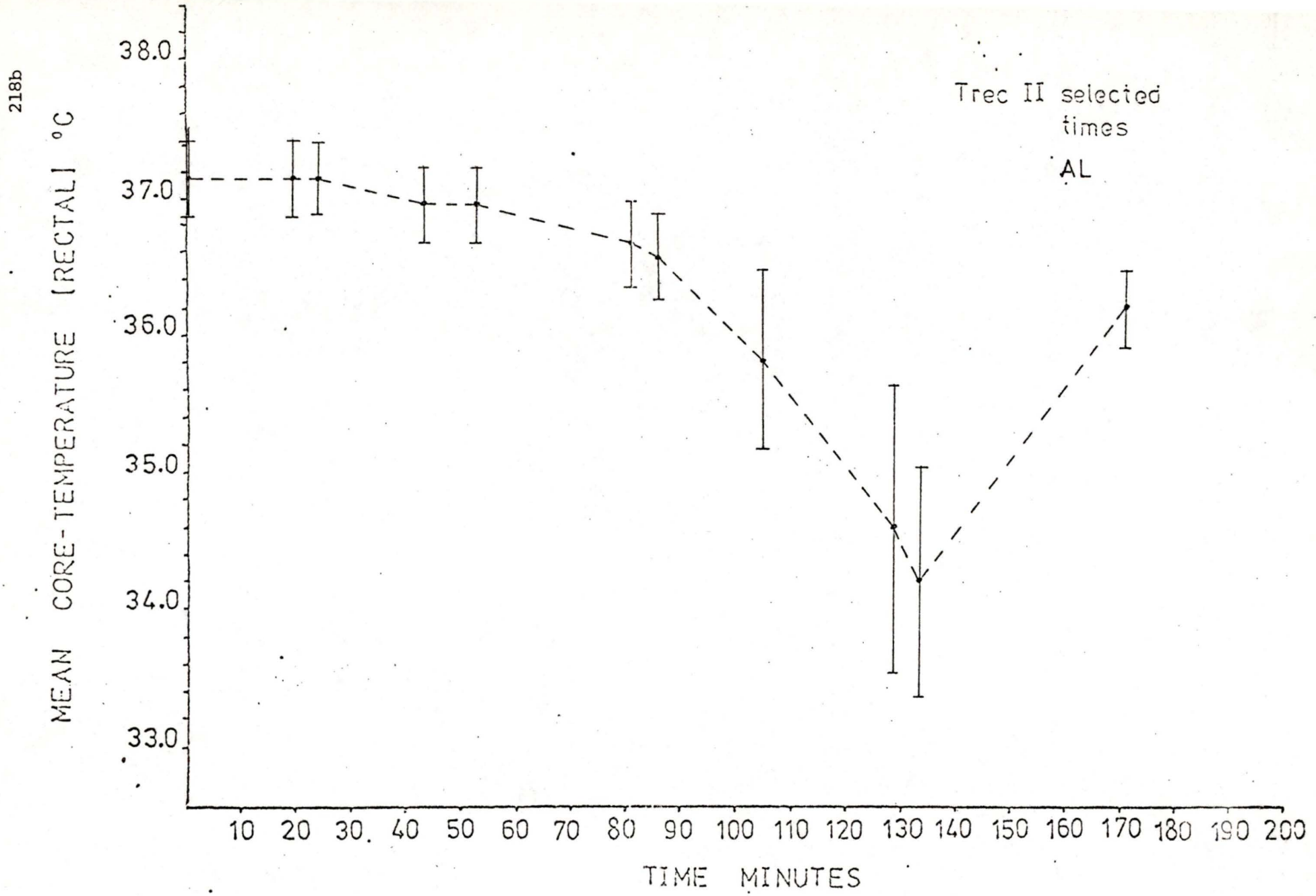

Trec I selected times
AL
MEAN CORE-TEMPERATURE [RECTAL] °C
38.0
37.0
36.0
35.0
34.0
33.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

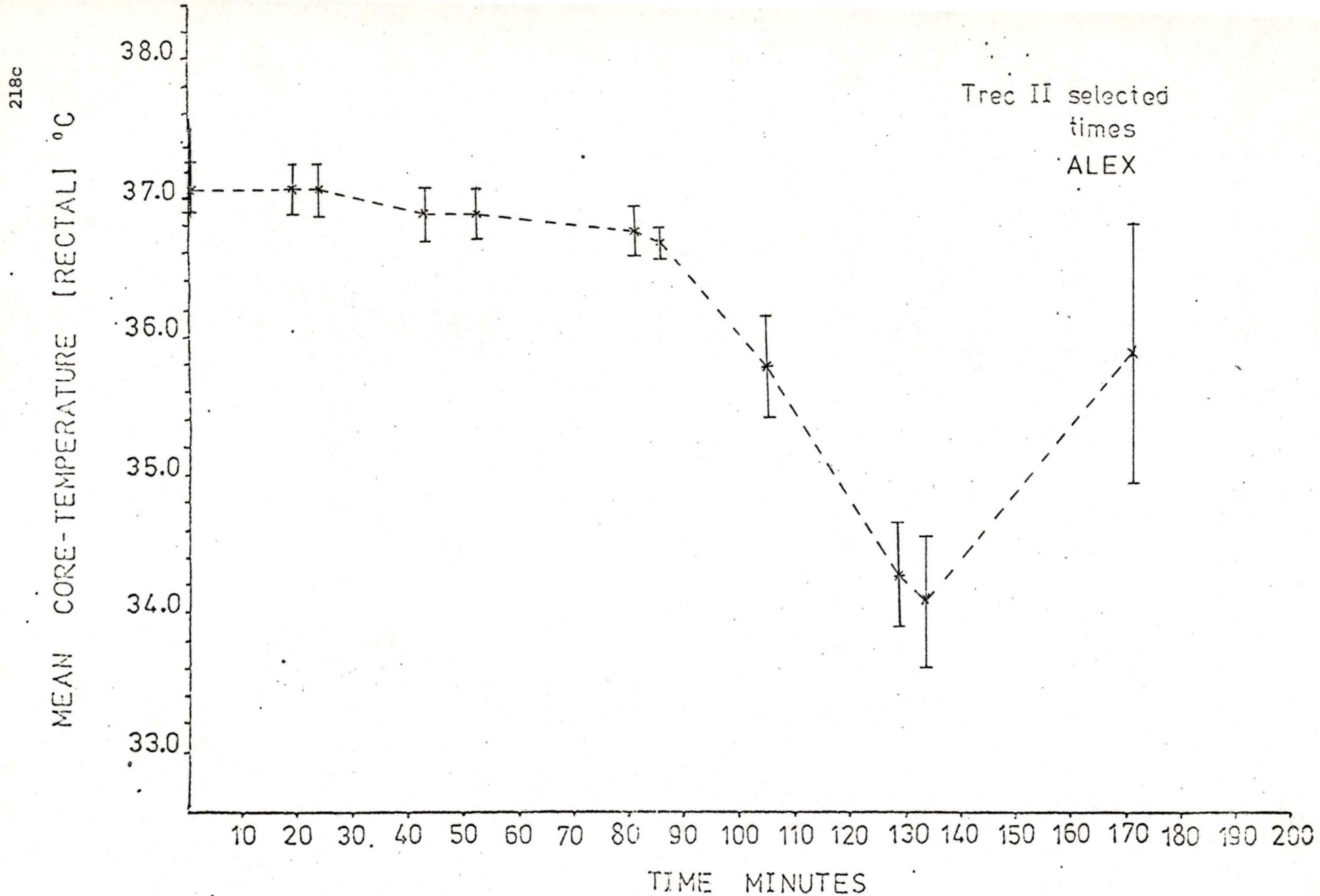

Trec I selected times
EX
MEAN CORE-TEMPERATURE [RECTAL] °C
38.0
37.0
36.0
35.0
34.0
33.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

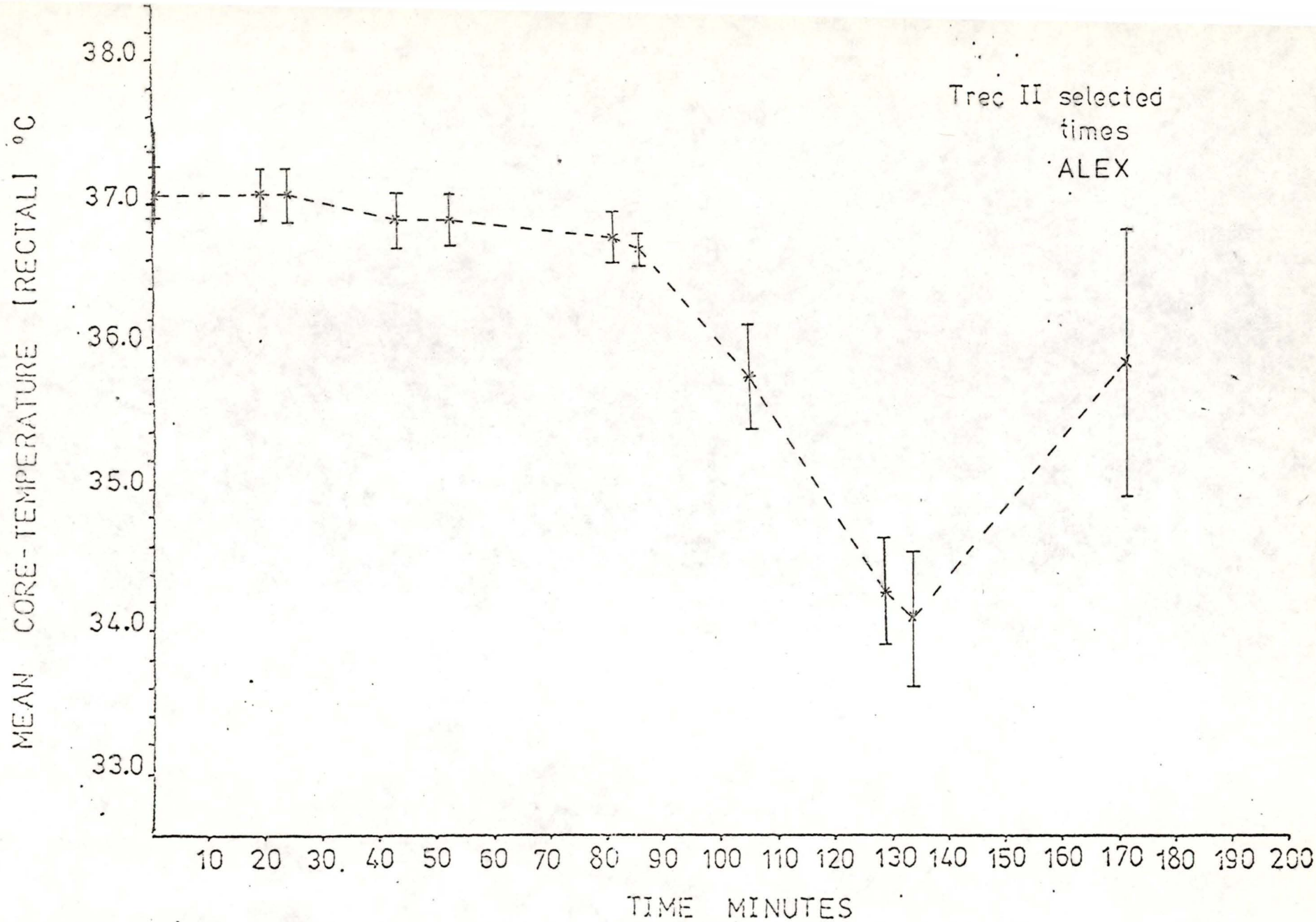

Trec I selected times
ALEX
MEAN CORE-TEMPERATURE [RECTAL] °C
38.0
37.0
36.0
35.0
34.0
33.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

Appendix 15. Single treatment graphs used to construct the composite graph of Figure 9

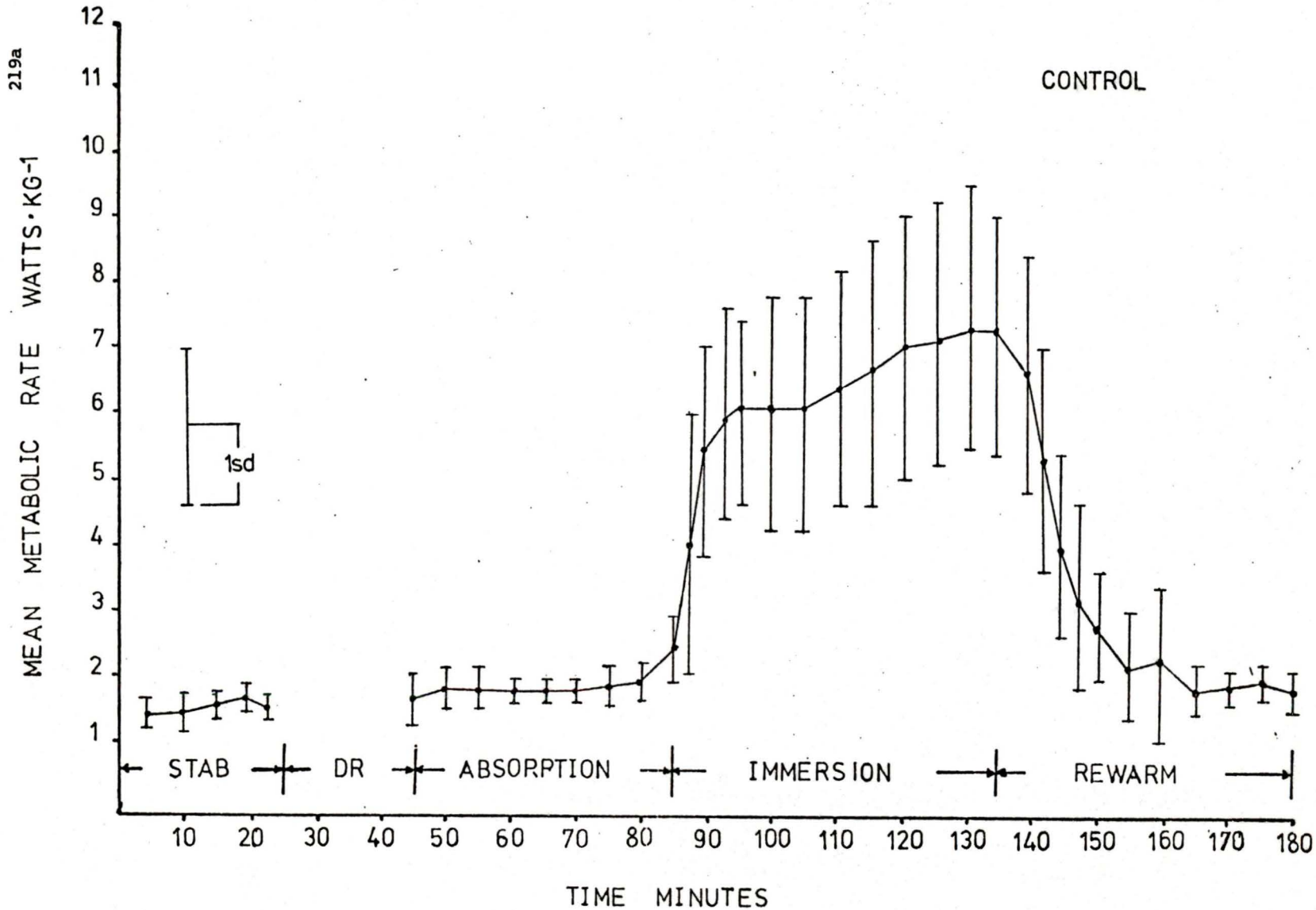

MEAN CORE-TEMPERATURE (RECTAL) °C
38.0
37.0
36.0
35.0
34.0
33.0
1sd
Trec II selected times
CONTROL
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

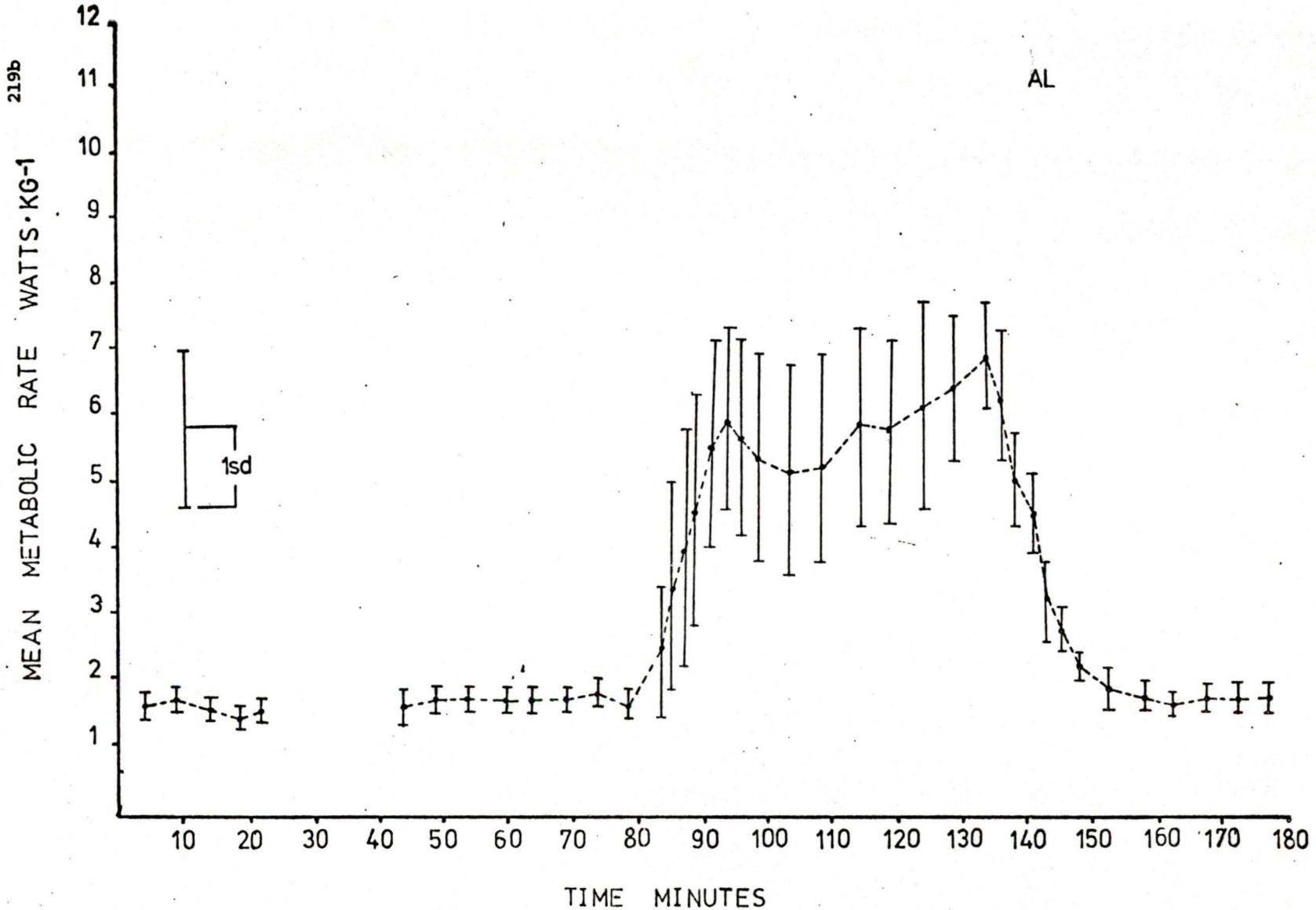

Trec II selected times
AL
MEAN CORE-TEMPERATURE (RECTAL) °C
38.0
37.0
36.0
35.0
34.0
33.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

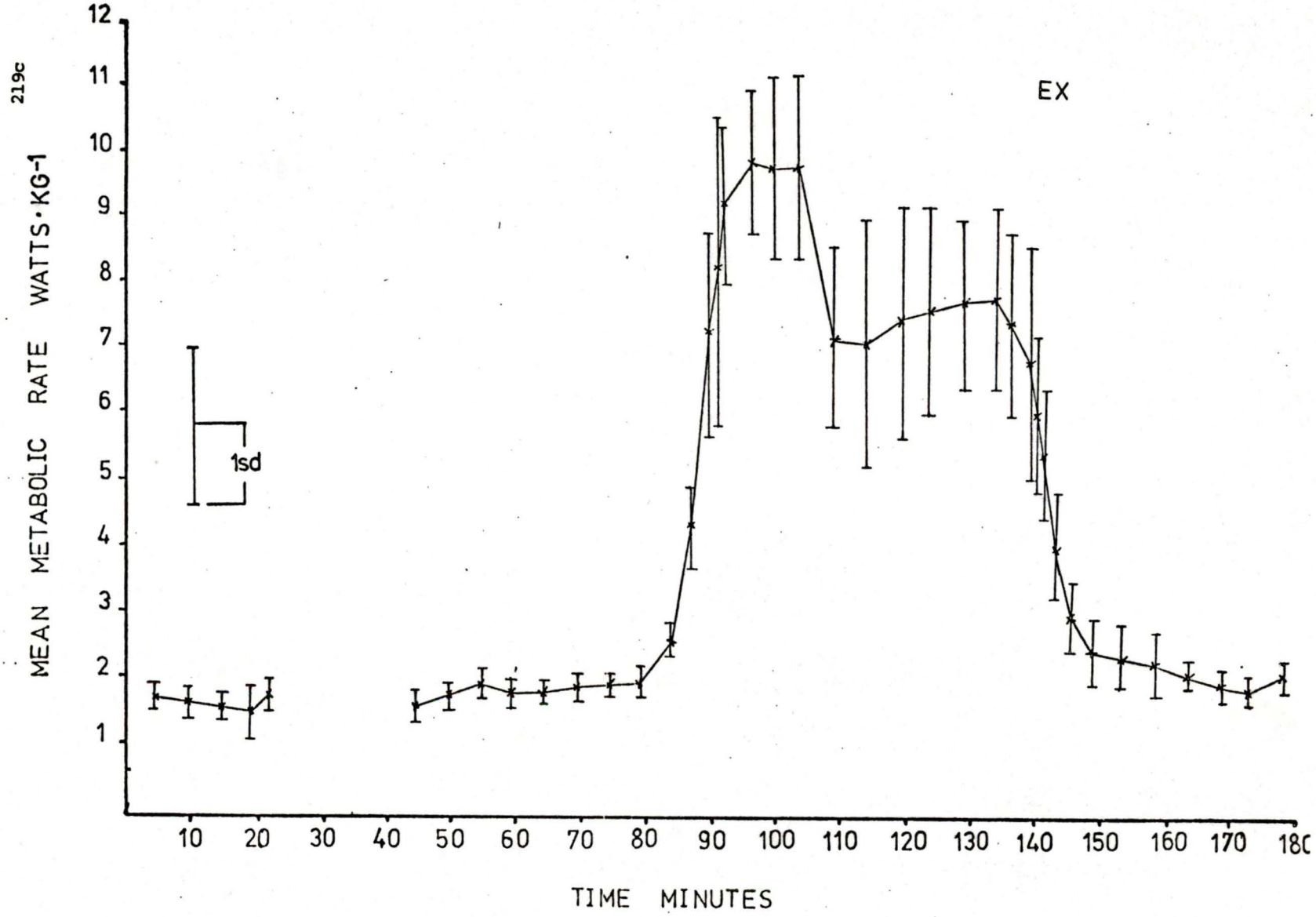

Trec II selected
times
ALEX
MEAN CORE-TEMPERATURE [RECTAL] °C
38.0
37.0
36.0
35.0
34.0
33.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

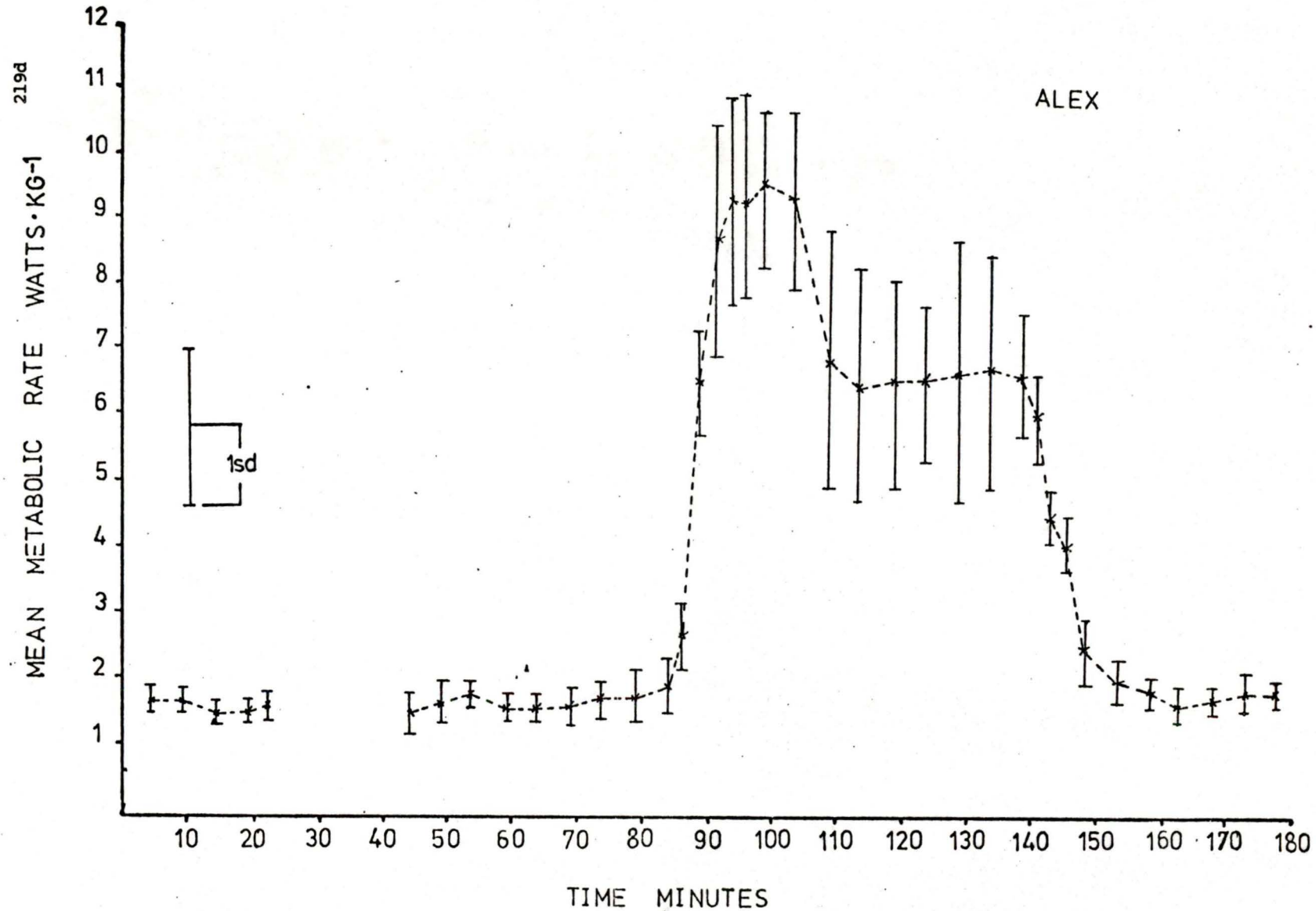

Trec II selected times
ALEX
MEAN CORE-TEMPERATURE (RECTAL) °C
38.0
37.0
36.0
35.0
34.0
33.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200
TIME MINUTES

Appendix 16. Single treatment graphs used to construct the composite graph of Figure 10

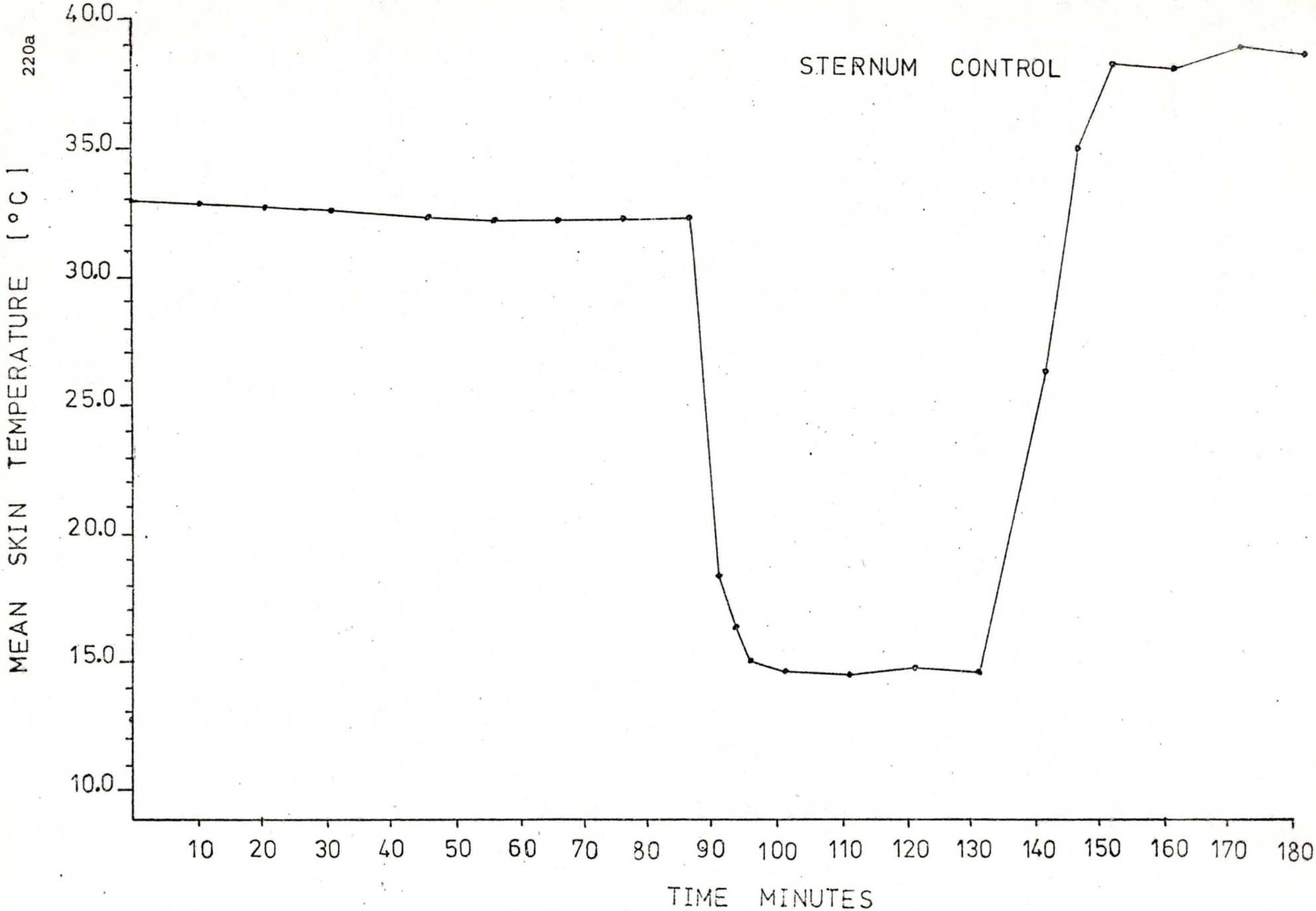

CONTROL
MEAN METABOLIC RATE WATTS·KG-1
12
11
10
9
8
7
6
5
4
3
2
1
1sd
STAB
DR
ABSORPTION
IMMERSION
REWARM
10
20
30
40
50
60
70
80
90
100
110
120
130
140
150
160
170
180
TIME MINUTES

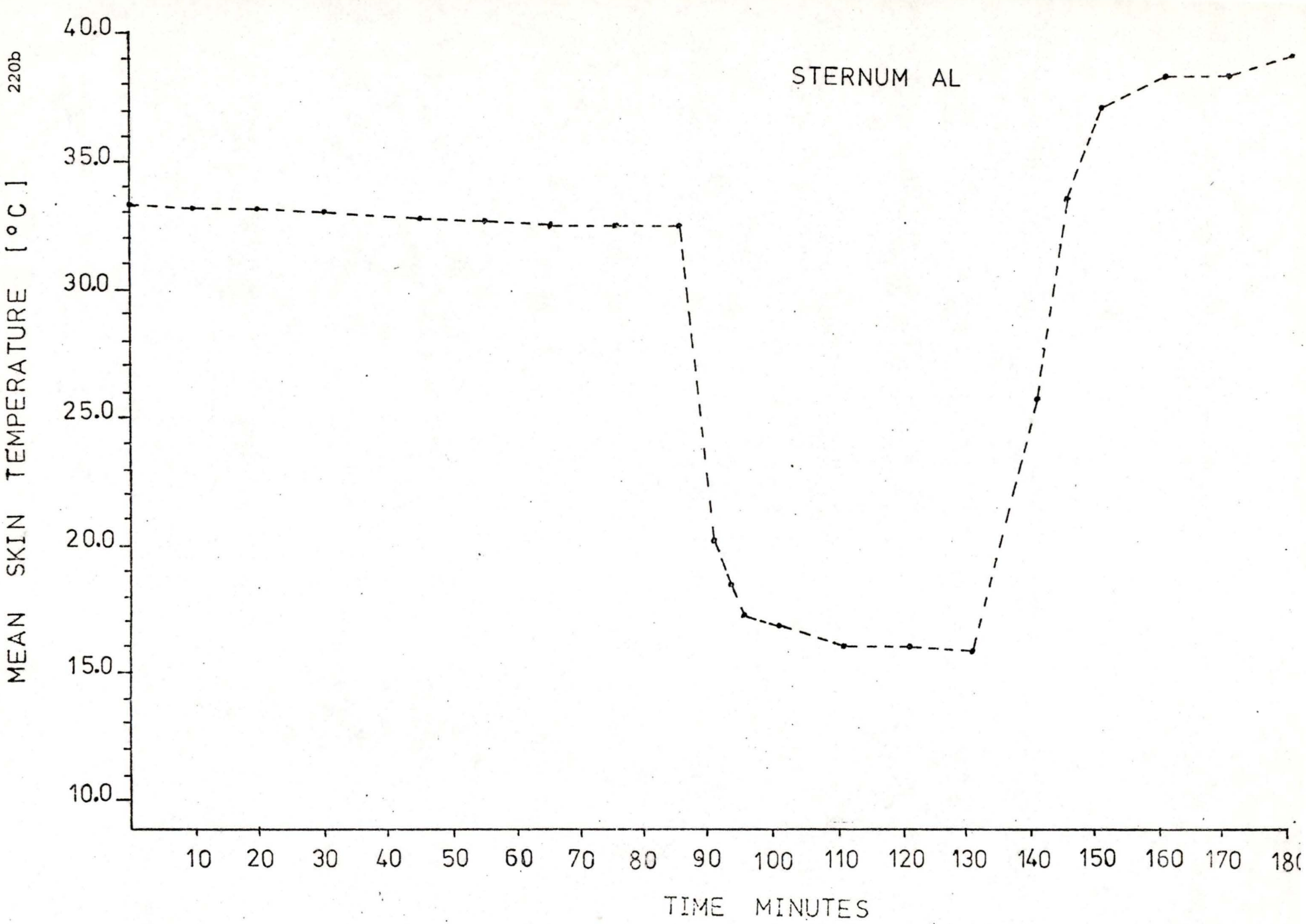

MEAN METABOLIC RATE WATTS·KG-1
219b
AL
1sd
12
11
10
9
8
7
6
5
4
3
2
1
10
20
30
40
50
60
70
80
90
100
110
120
130
140
150
160
170
180
TIME MINUTES

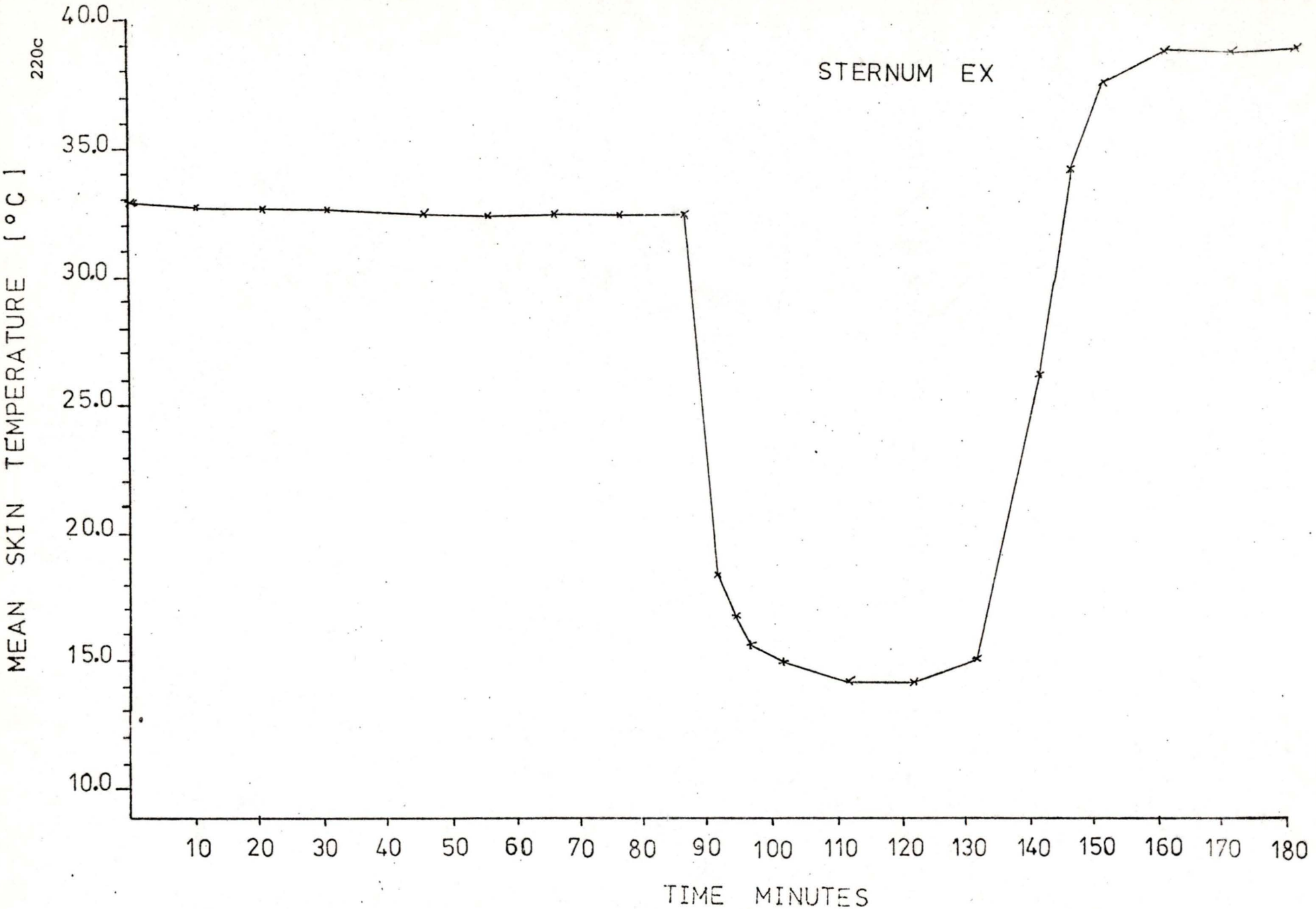

MEAN METABOLIC RATE WATTS·KG-1
219c
EX
1sd
12
11
10
9
8
7
6
5
4
3
2
1
10
20
30
40
50
60
70
80
90
100
110
120
130
140
150
160
170
180
TIME MINUTES

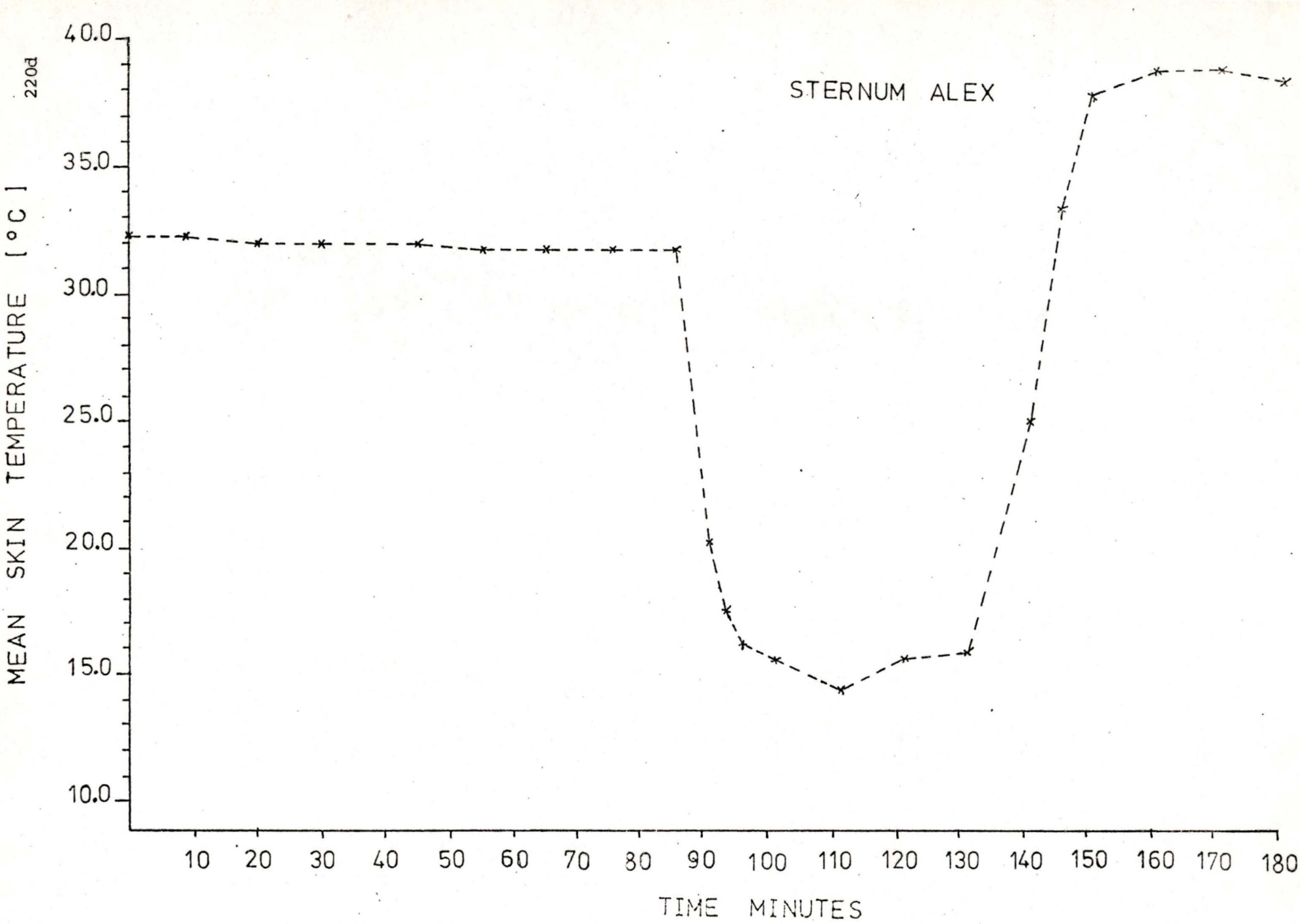

MEAN METABOLIC RATE WATTS·KG-1
219a
ALEX
1sd
12
11
10
9
8
7
6
5
4
3
2
1
10
20
30
40
50
60
70
80
90
100
110
120
130
140
150
160
170
180
TIME MINUTES

Appendix 17. Single treatment graphs used to construct the composite graph of Figure 11

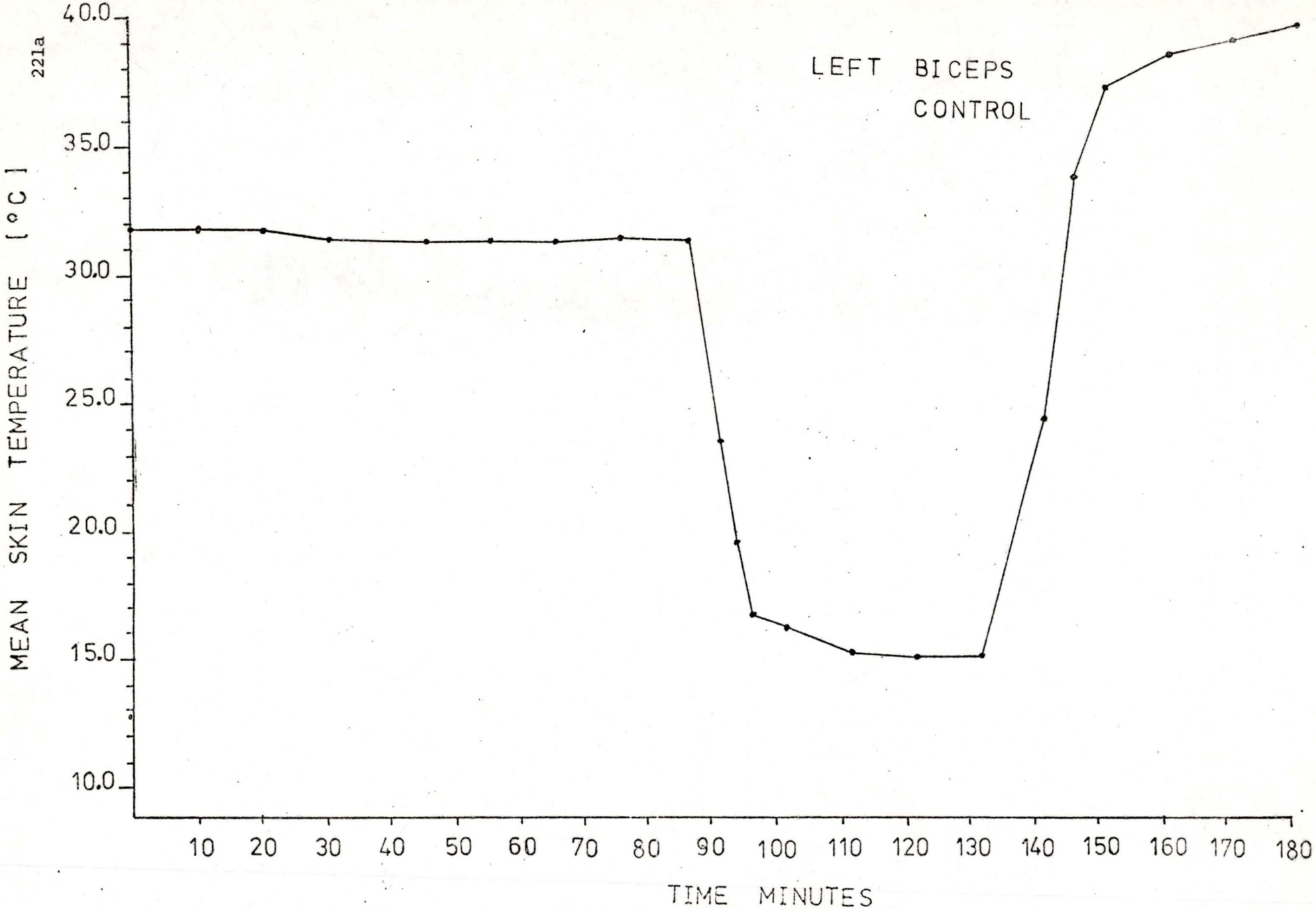

STERNUM CONTROL
MEAN SKIN TEMPERATURE [°C]
40.0
35.0
30.0
25.0
20.0
15.0
10.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES

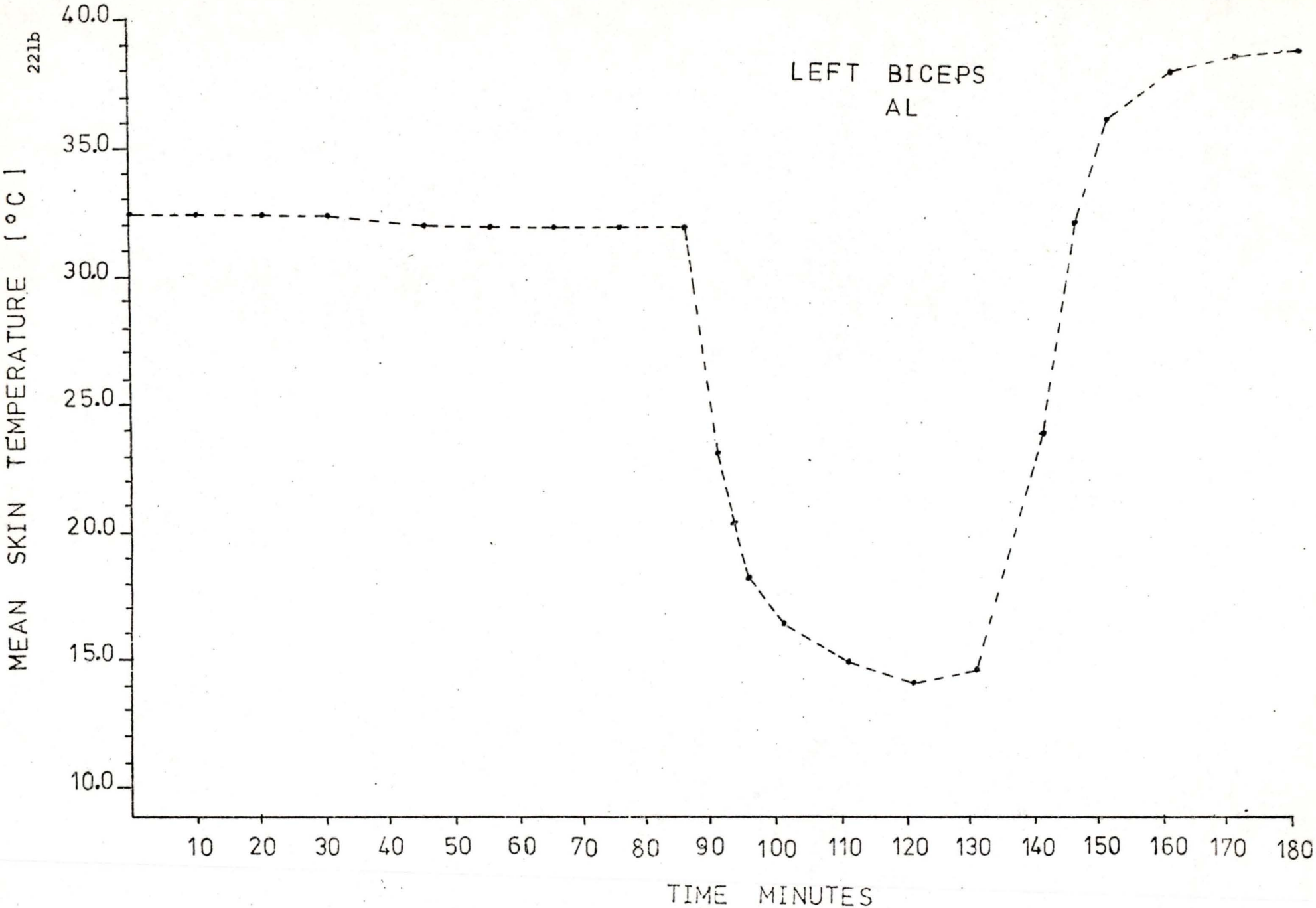

220b
STERNUM AL
MEAN SKIN TEMPERATURE [°C]
40.0
35.0
30.0
25.0
20.0
15.0
10.0
10
20
30
40
50
60
70
80
90
100
110
120
130
140
150
160
170
180
TIME MINUTES

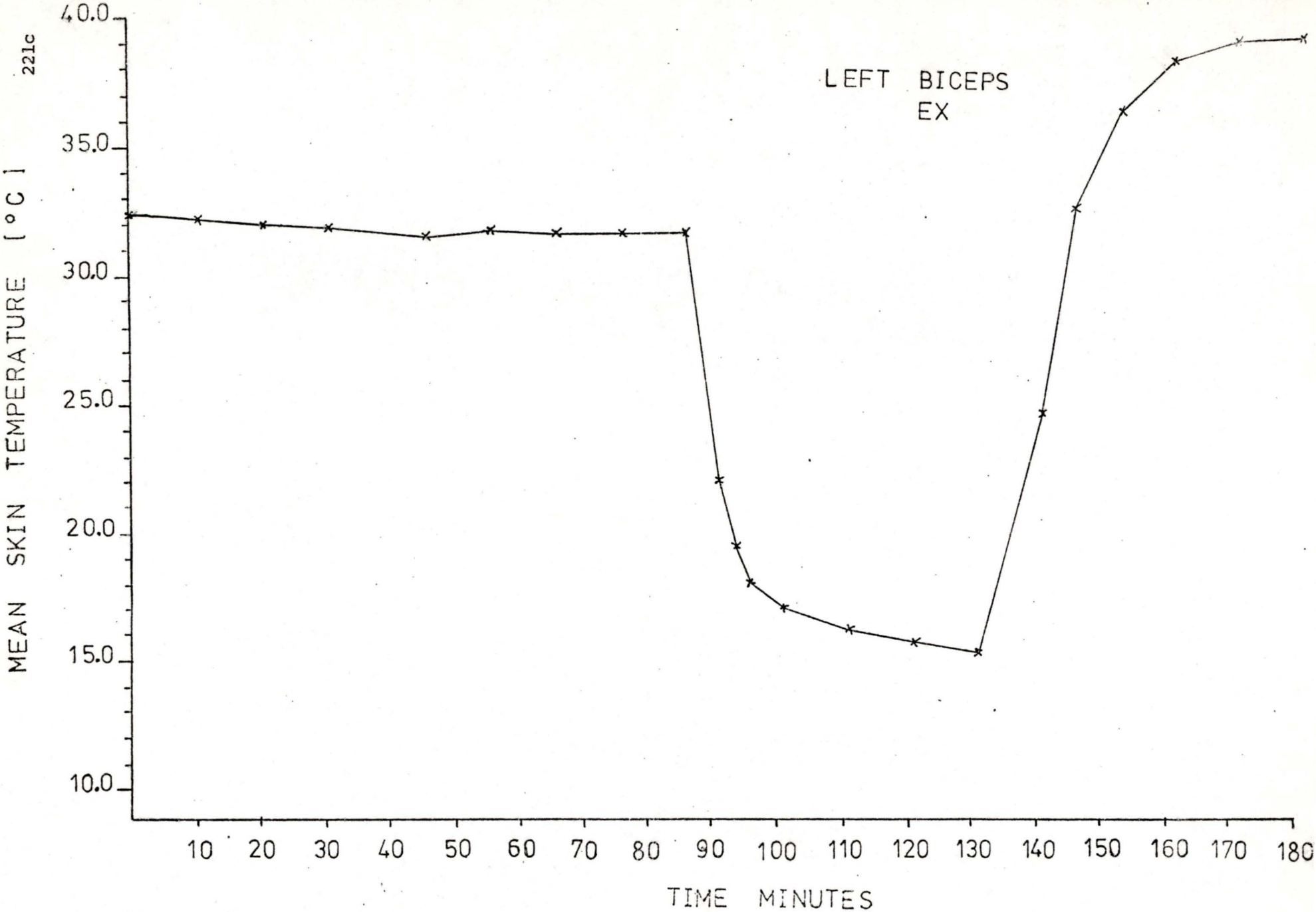

STERNUM EX
220c
MEAN SKIN TEMPERATURE [°C]
40.0
35.0
30.0
25.0
20.0
15.0
10.0
10
20
30
40
50
60
70
80
90
100
110
120
130
140
150
160
170
180
TIME MINUTES

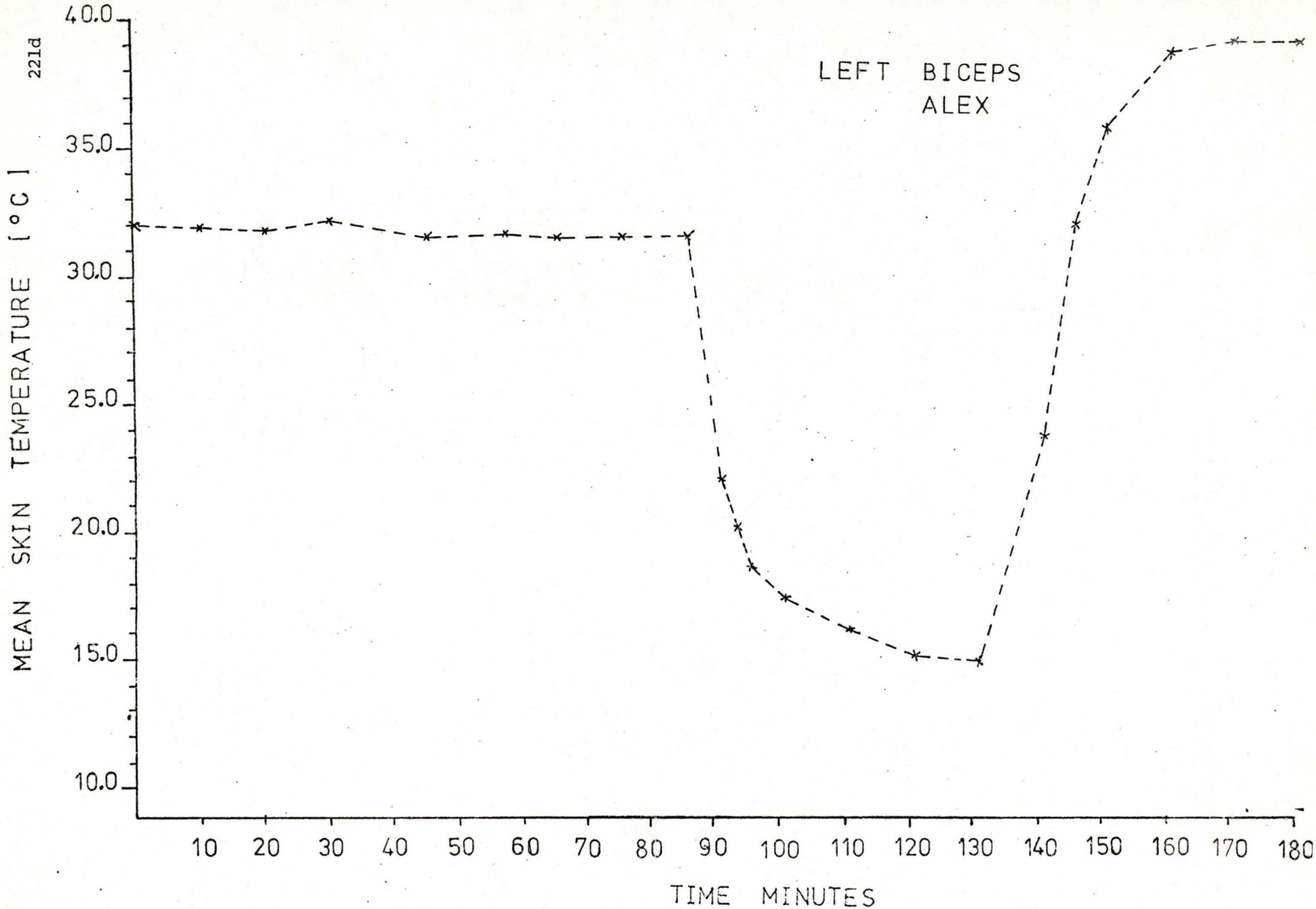

STERNUM ALEX
MEAN SKIN TEMPERATURE [°C] 220d
40.0
35.0
30.0
25.0
20.0
15.0
10.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES

Appendix 18. Single treatment graphs used to construct the composite graph of Figure 12

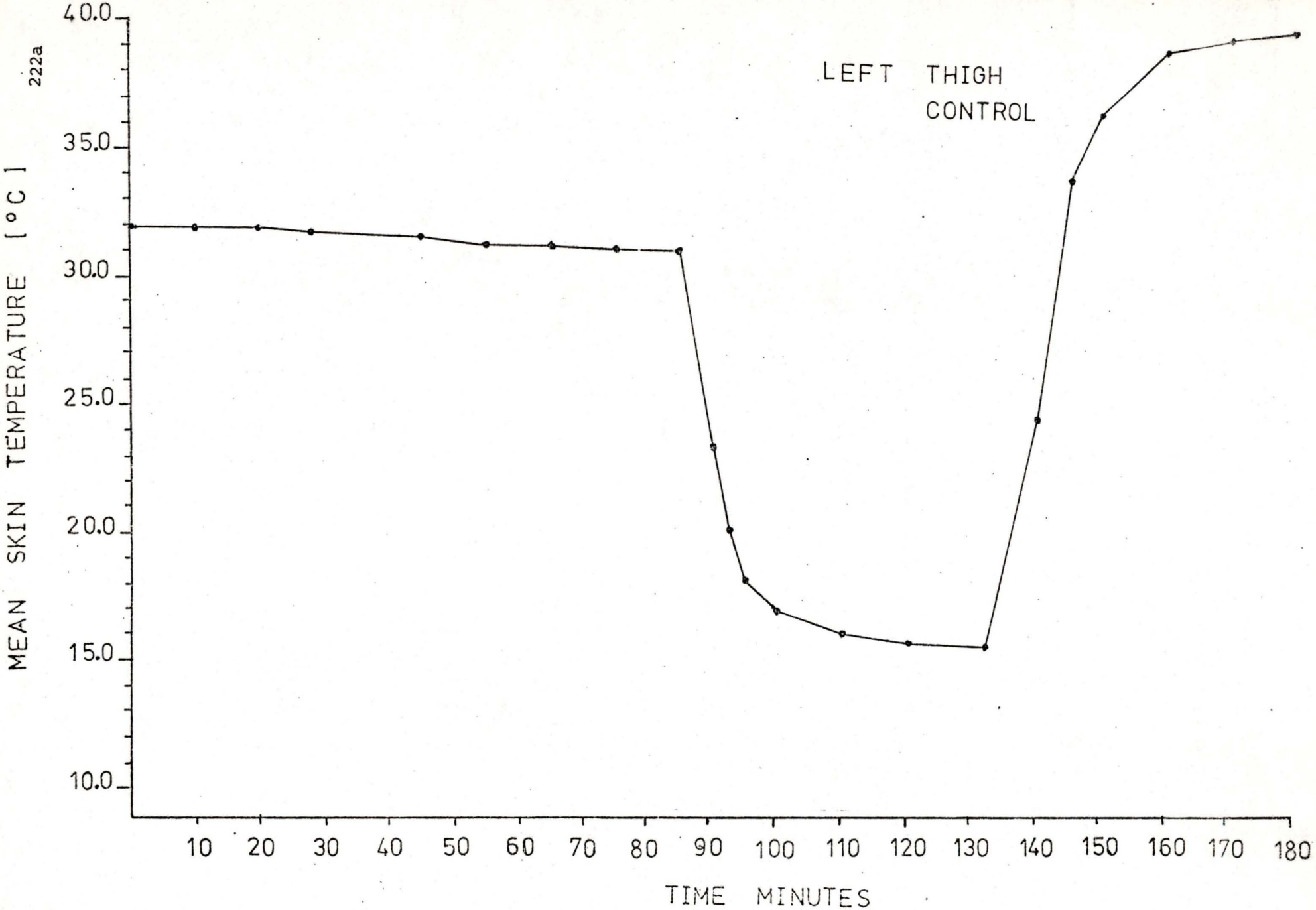

LEFT BICEPS
CONTROL
MEAN SKIN TEMPERATURE [°C]
40.0
35.0
30.0
25.0
20.0
15.0
10.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES

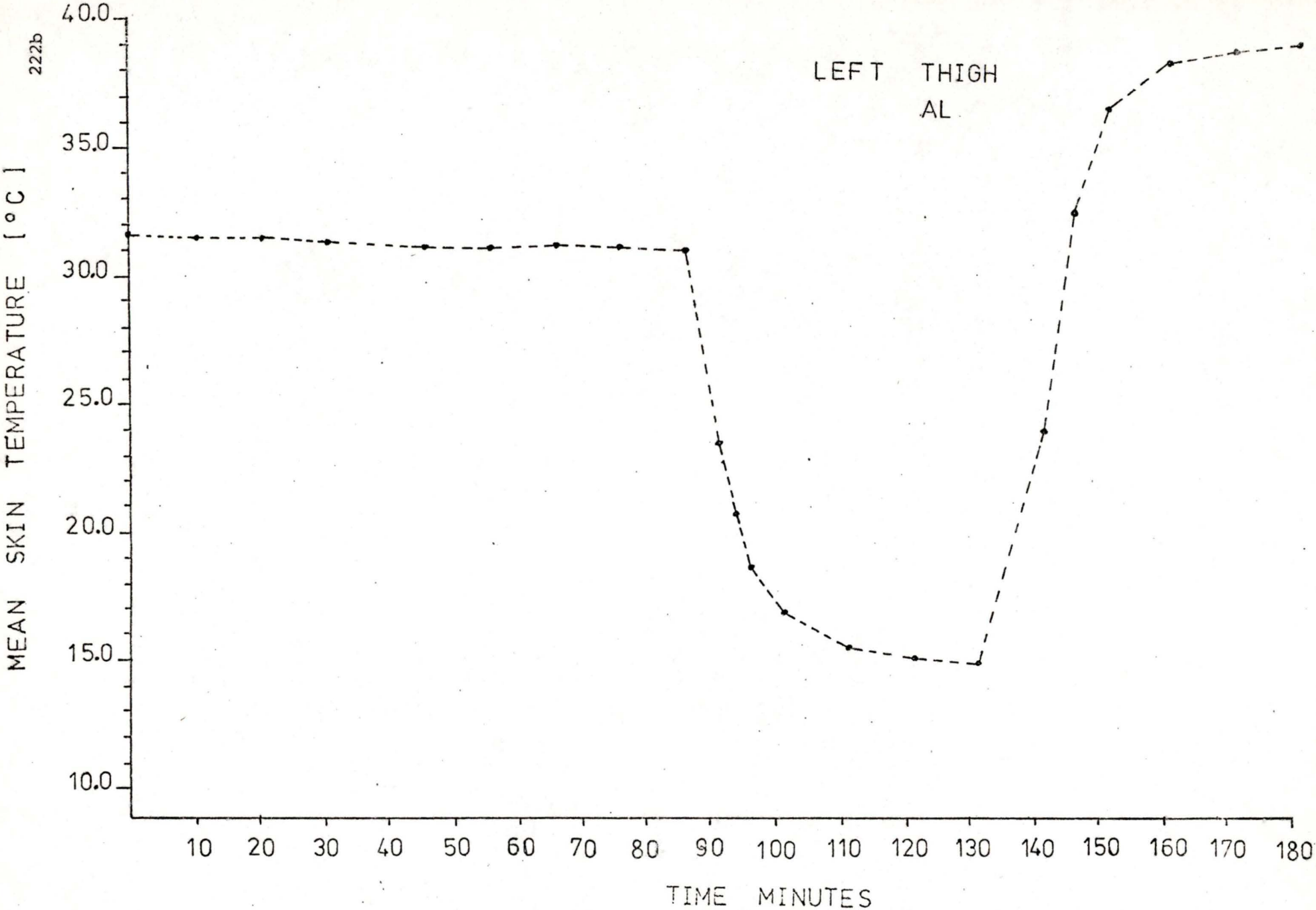

221b
LEFT BICEPS
AL
MEAN SKIN TEMPERATURE [°C]
40.0
35.0
30.0
25.0
20.0
15.0
10.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES

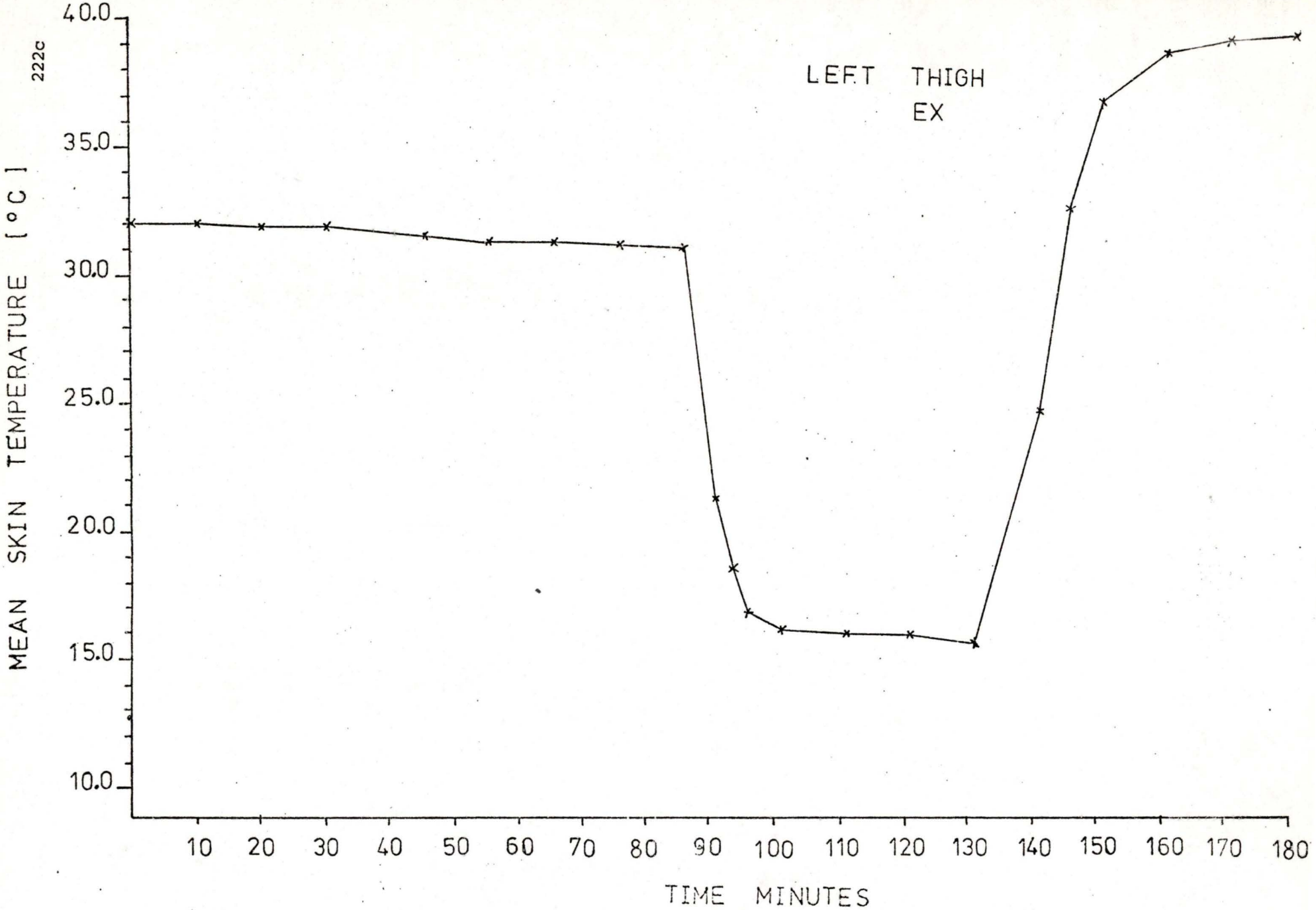

221c
LEFT BICEPS
EX
MEAN SKIN TEMPERATURE [°C]
40.0
35.0
30.0
25.0
20.0
15.0
10.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES

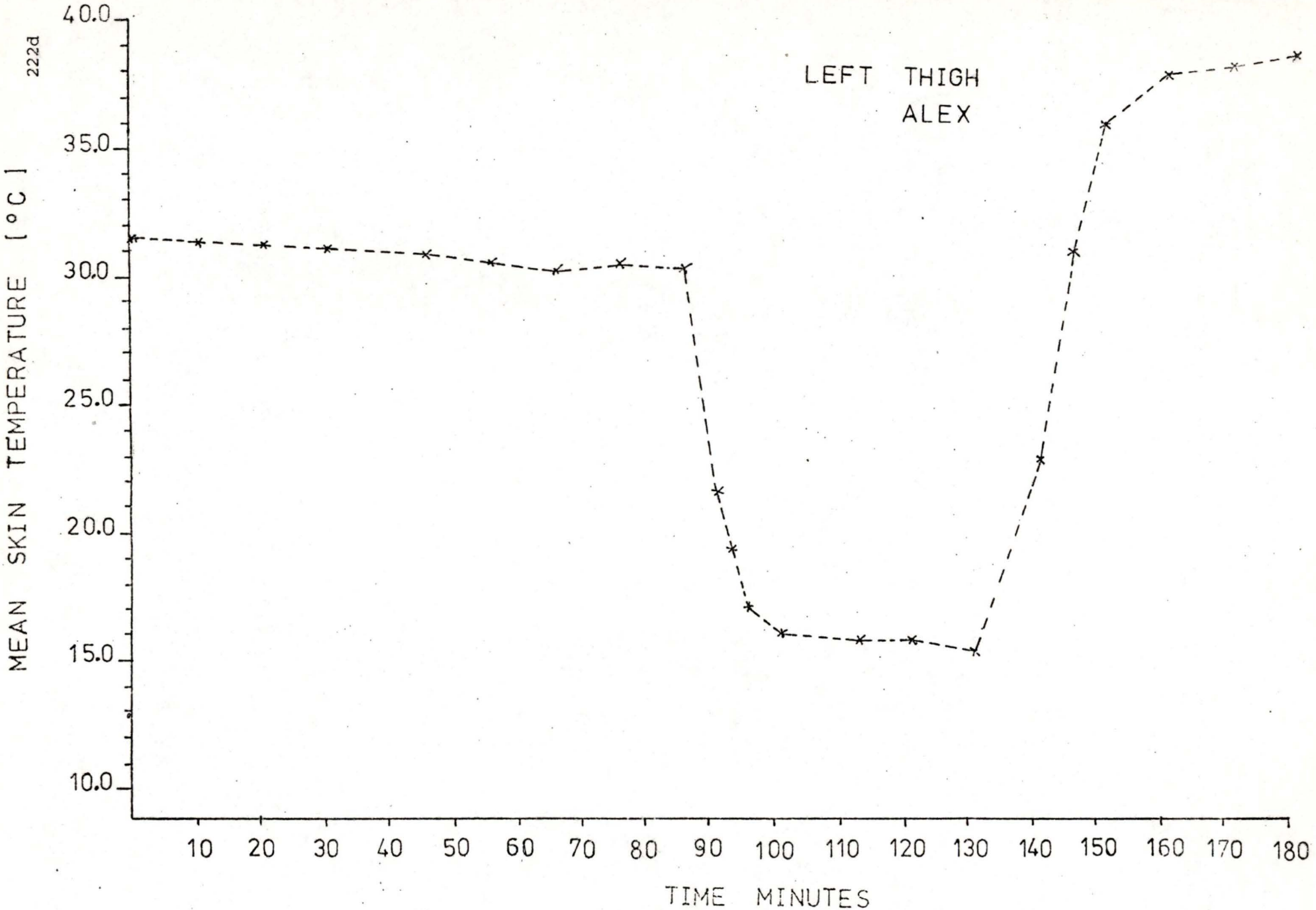

221d
LEFT BICEPS
ALEX
MEAN SKIN TEMPERATURE [°C]
40.0
35.0
30.0
25.0
20.0
15.0
10.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES

Appendix 19. Single treatment graphs used to construct the composite graph of Figure 13

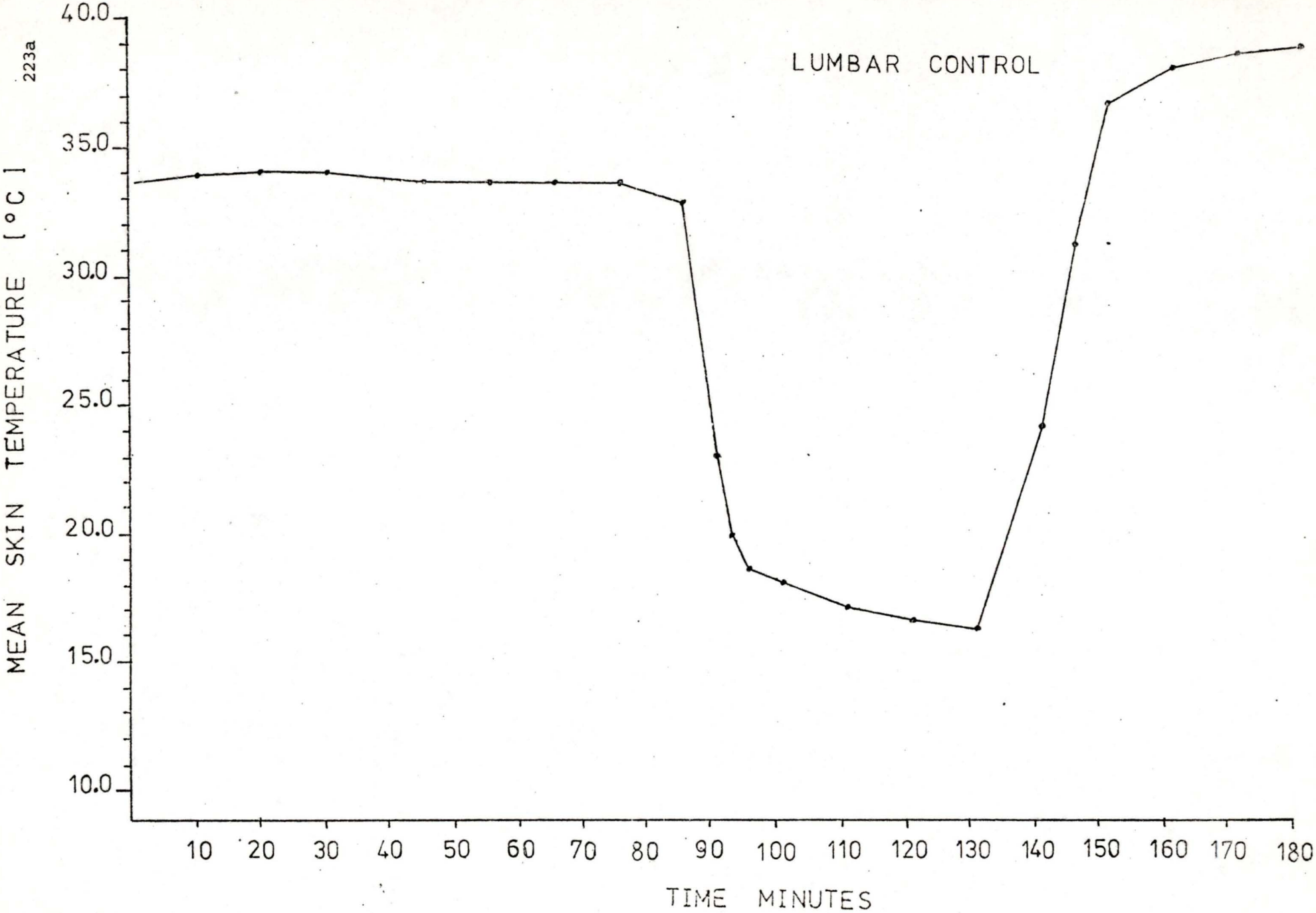

LEFT THIGH
CONTROL
MEAN SKIN TEMPERATURE [°C]
40.0
35.0
30.0
25.0
20.0
15.0
10.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES

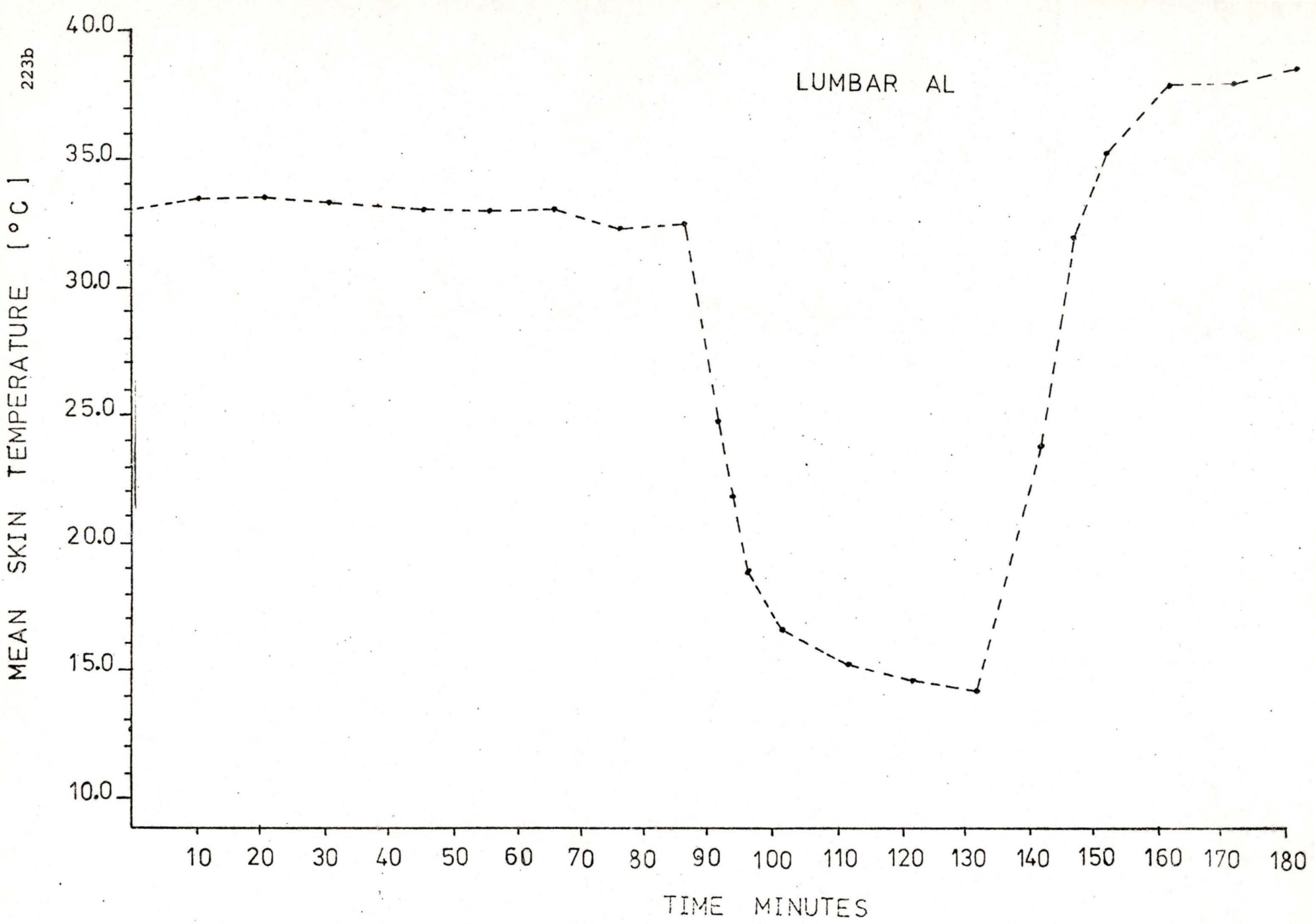

222b
LEFT THIGH
AL
MEAN SKIN TEMPERATURE [°C]
40.0
35.0
30.0
25.0
20.0
15.0
10.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES

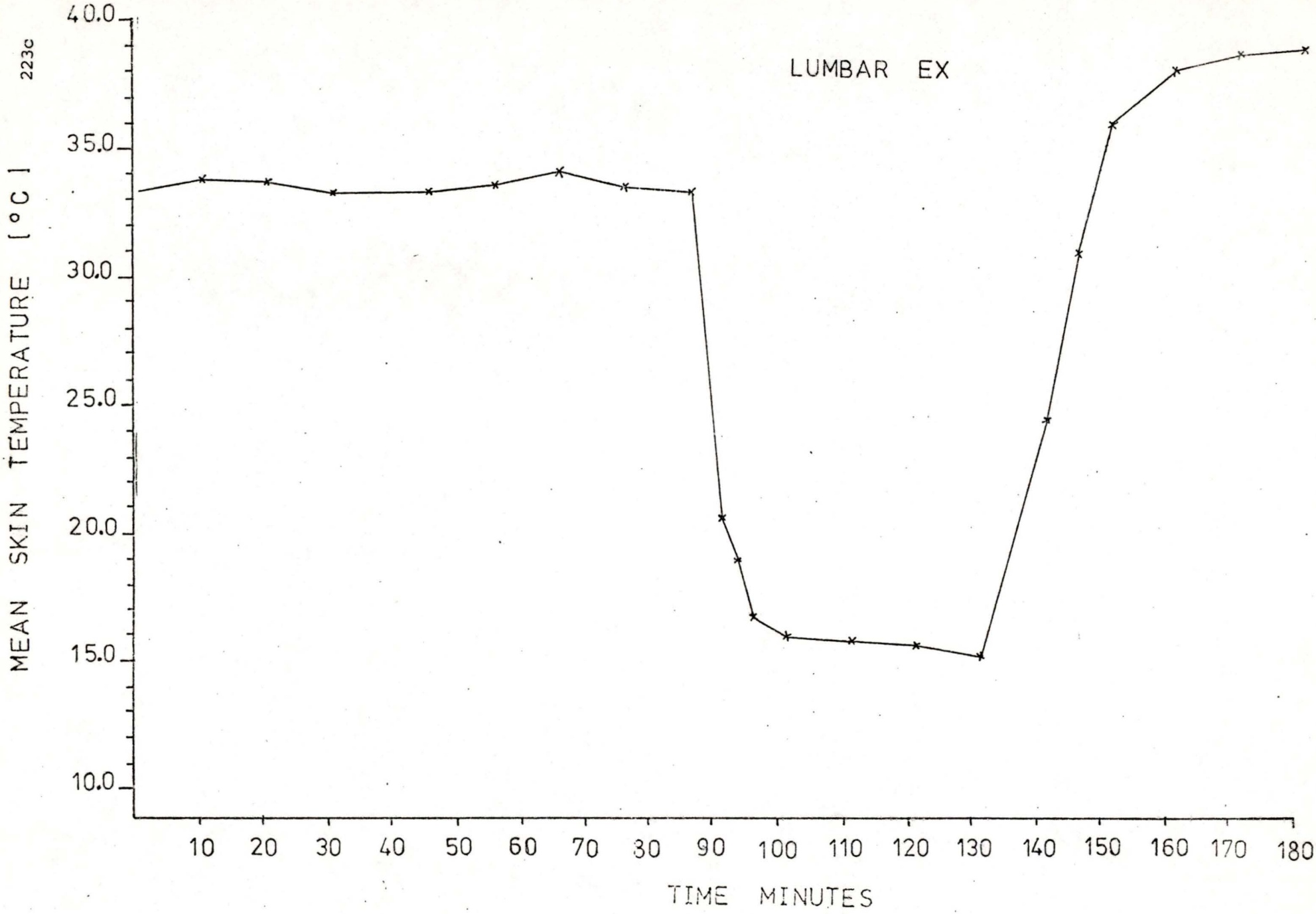

222c
LEFT THIGH
EX
MEAN SKIN TEMPERATURE [°C]
40.0
35.0
30.0
25.0
20.0
15.0
10.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES

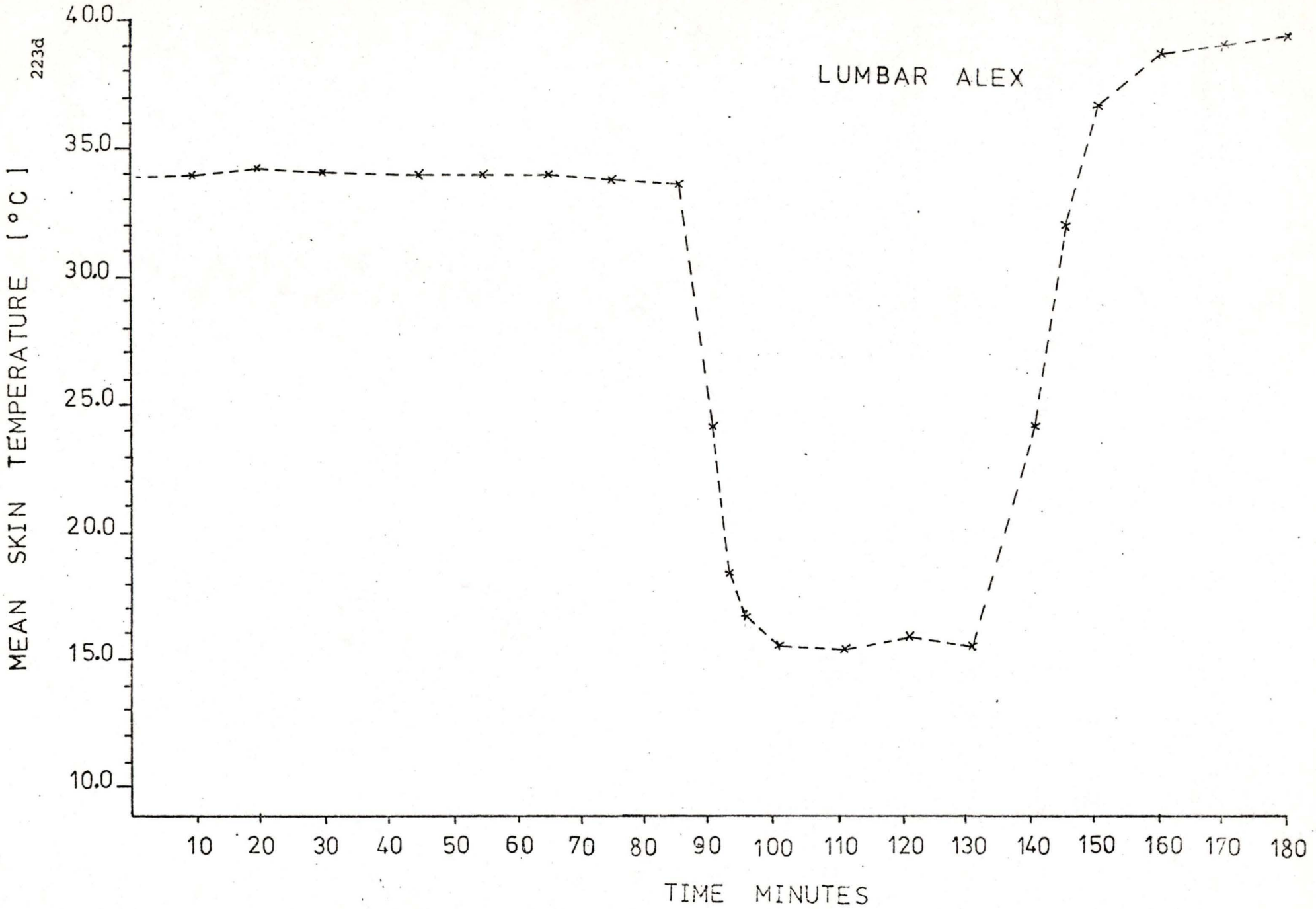

222d
LEFT THIGH
ALEX
MEAN SKIN TEMPERATURE [°C]
40.0
35.0
30.0
25.0
20.0
15.0
10.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES

Appendix 20. Single treatment graphs used to construct the composite graph of Figure 14


LUMBAR CONTROL
MEAN SKIN TEMPERATURE [°C]
40.0
35.0
30.0
25.0
20.0
15.0
10.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES


223b
LUMBAR AL
MEAN SKIN TEMPERATURE [°C]
40.0
35.0
30.0
25.0
20.0
15.0
10.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES


LUMBAR EX
223c
MEAN SKIN TEMPERATURE [°C]
40.0
35.0
30.0
25.0
20.0
15.0
10.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES


LUMBAR ALEX
MEAN SKIN TEMPERATURE [°C]
223d
40.0
35.0
30.0
25.0
20.0
15.0
10.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES

Appendix 21. Lists of means and standard deviations of the means observed at various times for skin temperatures at; sternum, left biceps, left thigh, and lumbar skin sites during control, al, ex, and alex experiments

List 1. Means and standard deviations observed at; sternum, left biceps, left thigh, and lumbar skin sites under control treatment

Time, minutes	Statistic	Skin sites, sternum	lt. biceps	lt. thigh	lumbar
0	mean	32.71	31.74	31.47	33.56
	sd	0.859	0.430	0.673	0.913
10	mean	32.61	31.76	31.46	33.85
	sd	0.895	0.483	0.650	0.715
30	mean	32.38	31.32	31.23	33.91
	sd	0.812	0.605	0.815	0.999
45	mean	32.14	31.21	31.11	33.63
	sd	0.855	0.705	0.756	1.267
55	mean	32.00	31.21	30.85	33.56
	sd	0.835	0.719	0.848	1.339
65	mean	32.04	31.09	30.73	33.57
	sd	0.897	0.989	0.891	1.381
75	mean	32.10	31.23	30.66	33.63
	sd	0.750	0.877	0.911	1.366
85	mean	32.17	31.09	30.56	32.80
	sd	0.760	1.103	0.841	1.211
90	mean	18.36	23.35	22.98	23.08
	sd	4.165	2.028	1.687	1.377
95	mean	15.02	16.66	17.78	18.66
	sd	2.761	1.365	0.882	1.172
100	mean	14.67	16.23	16.61	17.18
	sd	2.418	1.003	0.628	1.226
110	mean	14.58	15.16	15.75	16.23
	sd	2.119	1.035	0.509	1.245
120	mean	14.86	14.98	15.44	15.69
	sd	2.147	1.211	0.643	1.115
130	mean	14.65	15.01	15.34	15.44
	sd	2.211	1.221	0.726	1.097
140	mean	26.40	24.23	24.20	24.26
	sd	2.572	1.669	2.042	2.049
145	mean	34.94	33.46	33.36	32.83
	sd	1.384	1.744	1.949	1.467
150	mean	38.33	36.95	36.85	36.68
	sd	1.079	1.287	1.296	0.829
160	mean	38.63	38.26	38.37	38.06
	sd	0.833	0.381	0.308	0.945
170	mean	38.92	38.77	38.84	38.69
	sd	0.807	0.421	0.245	0.603
180	mean	38.64	39.28	39.08	39.00
	sd	1.312	0.498	0.413	0.621

lt. = left n = 10 for all above means

sd = standard deviation of the mean

List 2. Means and standard deviations observed at; sternum, left biceps, left thigh, and lumbar skin sites under al treatment

Time, minutes	Statistic	Skin sites, sternum	lt. biceps	lt. thigh	lumbar
0	mean	33.46	32.37	31.48	33.09
	sd	1.715	0.799	0.676	0.803
10	mean	31.43	32.38	32.90	33.48
	sd	1.115	0.679	0.935	0.826
30	mean	32.70	32.25	31.23	33.32
	sd	1.137	0.999	0.684	1.163
45	mean	32.48	31.89	31.08	33.22
	sd	1.124	1.096	0.719	1.215
55	mean	32.44	31.72	30.97	33.04
	sd	0.974	0.951	0.701	1.246
65	mean	32.24	31.67	31.09	33.07
	sd	1.126	0.873	0.919	1.480
75	mean	32.23	31.72	31.15	32.39
	sd	1.043	0.970	1.074	1.321
85	mean	32.24	31.72	30.92	32.50
	sd	1.001	0.903	0.878	1.238
90	mean	20.04	23.10	23.39	24.83
	sd	4.211	1.831	2.091	4.067
95	mean	17.07	18.32	18.84	18.97
	sd	3.350	1.159	1.530	2.241
100	mean	16.71	16.45	16.94	16.79
	sd	2.818	0.886	0.994	1.755
110	mean	15.89	14.95	15.53	15.36
	sd	2.603	0.985	0.701	1.664
120	mean	15.80	14.16	15.14	14.75
	sd	2.404	0.763	0.689	1.293
130	mean	15.68	14.66	14.85	14.43
	sd	2.582	1.777	0.598	1.066
140	mean	25.47	23.78	23.87	23.92
	sd	2.304	2.483	1.999	1.818
145	mean	33.16	32.04	32.42	31.88
	sd	2.104	1.420	1.511	1.612
150	mean	36.77	36.03	36.38	36.22
	sd	1.552	1.256	1.136	1.749
160	mean	37.95	37.90	38.08	37.64
	sd	0.366	0.469	0.447	0.563
170	mean	38.03	38.46	38.57	38.04
	sd	1.144	0.393	0.512	0.705
180	mean	38.76	38.68	38.86	38.63
	sd	0.545	0.485	0.887	0.281

lt. = left n = 10 for all above means
sd. = standard deviation of the mean

List 3. Means and standard deviations observed at; sternum, left biceps, left thigh, and lumbar skin sites under ex treatment

Time, minutes	Statistic	Skin sites, sternum	lt. biceps	lt. thigh	lumbar
0	mean	33.12	32.20	31.76	33.22
	sd	0.759	0.864	0.552	0.954
10	mean	32.87	31.95	31.70	33.74
	sd	0.777	0.888	0.772	1.257
30	mean	32.75	31.68	31.60	33.24
	sd	0.726	0.811	0.776	1.177
45	mean	32.55	31.44	31.29	33.30
	sd	0.703	0.845	0.746	1.183
55	mean	32.46	31.55	31.03	33.54
	sd	0.619	0.917	0.804	1.355
65	mean	32.48	31.51	31.01	34.07
	sd	0.636	0.913	0.681	1.574
75	mean	32.52	31.45	30.94	33.46
	sd	0.533	1.097	0.647	1.202
85	mean	32.61	31.59	30.84	33.23
	sd	0.511	1.097	0.762	1.214
90	mean	18.54	22.02	21.06	20.64
	sd	4.052	1.469	2.058	3.426
95	mean	15.79	18.02	16.75	16.78
	sd	2.901	1.246	1.142	2.566
100	mean	15.14	17.06	16.01	16.06
	sd	2.496	1.226	0.858	2.255
110	mean	14.41	16.32	15.86	15.90
	sd	2.013	1.594	0.747	1.797
120	mean	14.93	15.71	15.87	15.74
	sd	2.161	1.121	0.701	1.582
130	mean	15.33	15.44	15.53	15.34
	sd	2.485	1.012	0.747	1.199
140	mean	26.03	24.68	24.50	24.51
	sd	2.009	1.460	1.254	1.381
145	mean	34.13	32.49	32.29	31.93
	sd	2.703	2.021	1.992	2.202
150	mean	37.46	36.31	36.50	36.02
	sd	1.505	1.241	1.246	2.075
160	mean	38.79	38.32	38.40	38.13
	sd	0.751	0.674	0.726	1.014
170	mean	38.68	38.96	38.92	38.65
	sd	1.959	0.657	0.705	0.746
180	mean	38.83	39.20	39.01	38.90
	sd	1.120	0.459	0.463	0.454

lt. = left n = 10 for all above means

sd = standard deviation of the mean

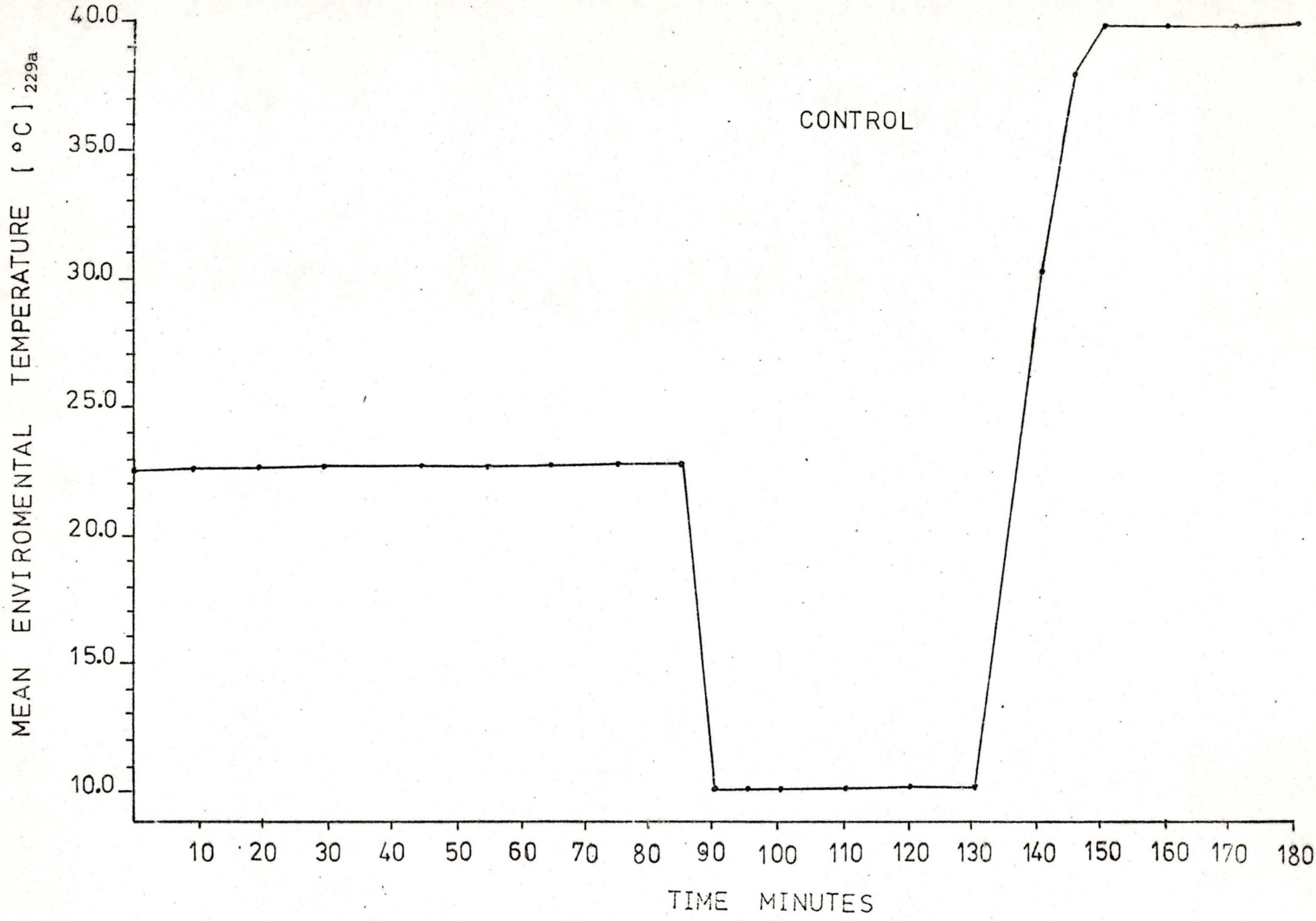
List 4. Means and standard deviations observed at; sternum, left biceps, left thigh, and lumbar skin sites under alex treatment

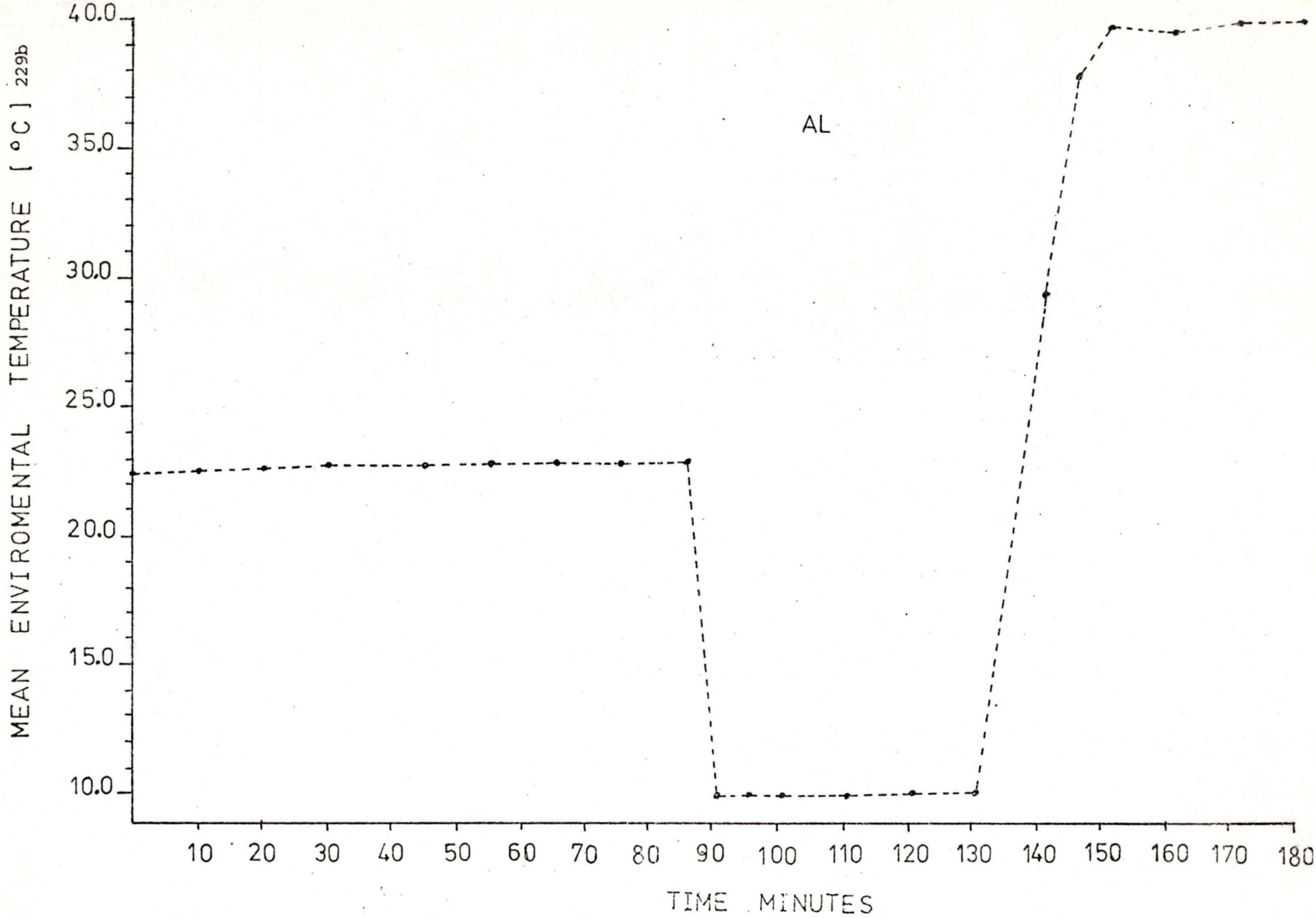
Time, minutes	Statistic	Skin sites, sternum	lt. biceps	lt. thigh	lumbar
0	mean	32.30	31.61	31.47	33.57
	sd	1.375	1.004	0.688	1.002
10	mean	32.25	31.52	31.33	33.69
	sd	1.313	1.005	0.710	0.978
30	mean	31.95	31.14	30.97	33.80
	sd	1.217	1.002	0.631	0.930
45	mean	31.90	31.10	30.84	33.65
	sd	1.062	0.951	0.582	1.099
55	mean	31.75	31.28	30.50	33.61
	sd	1.304	1.118	0.585	1.242
65	mean	31.66	31.07	30.26	33.55
	sd	1.133	0.803	0.378	1.237
75	mean	31.63	31.11	30.45	33.44
	sd	1.077	0.943	1.077	1.106
85	mean	31.60	31.10	30.31	33.23
	sd	1.146	0.764	0.767	1.263
90	mean	20.34	21.24	21.68	23.81
	sd	1.487	3.037	2.307	4.359
95	mean	16.27	17.76	17.23	16.42
	sd	2.419	1.965	1.394	3.095
100	mean	15.58	16.68	16.23	15.22
	sd	2.079	1.645	1.103	2.432
110	mean	14.46	15.93	16.03	15.08
	sd	2.288	1.331	0.657	1.787
120	mean	15.70	14.87	16.08	15.61
	sd	2.063	1.138	0.418	1.606
130	mean	15.89	14.69	15.64	15.16
	sd	2.028	0.857	0.536	1.505
140	mean	25.00	23.42	23.09	23.89
	sd	2.556	2.327	1.717	2.152
145	mean	33.33	31.56	31.03	31.61
	sd	2.514	2.037	1.699	1.609
150	mean	37.69	36.34	36.96	36.14
	sd	1.674	1.449	1.327	1.182
160	mean	38.68	38.30	37.96	38.14
	sd	0.744	0.719	0.479	0.740
170	mean	38.75	38.67	38.23	38.50
	sd	0.579	0.543	0.489	0.377
180	mean	38.30	38.66	38.68	38.87
	sd	1.373	0.868	0.547	0.530

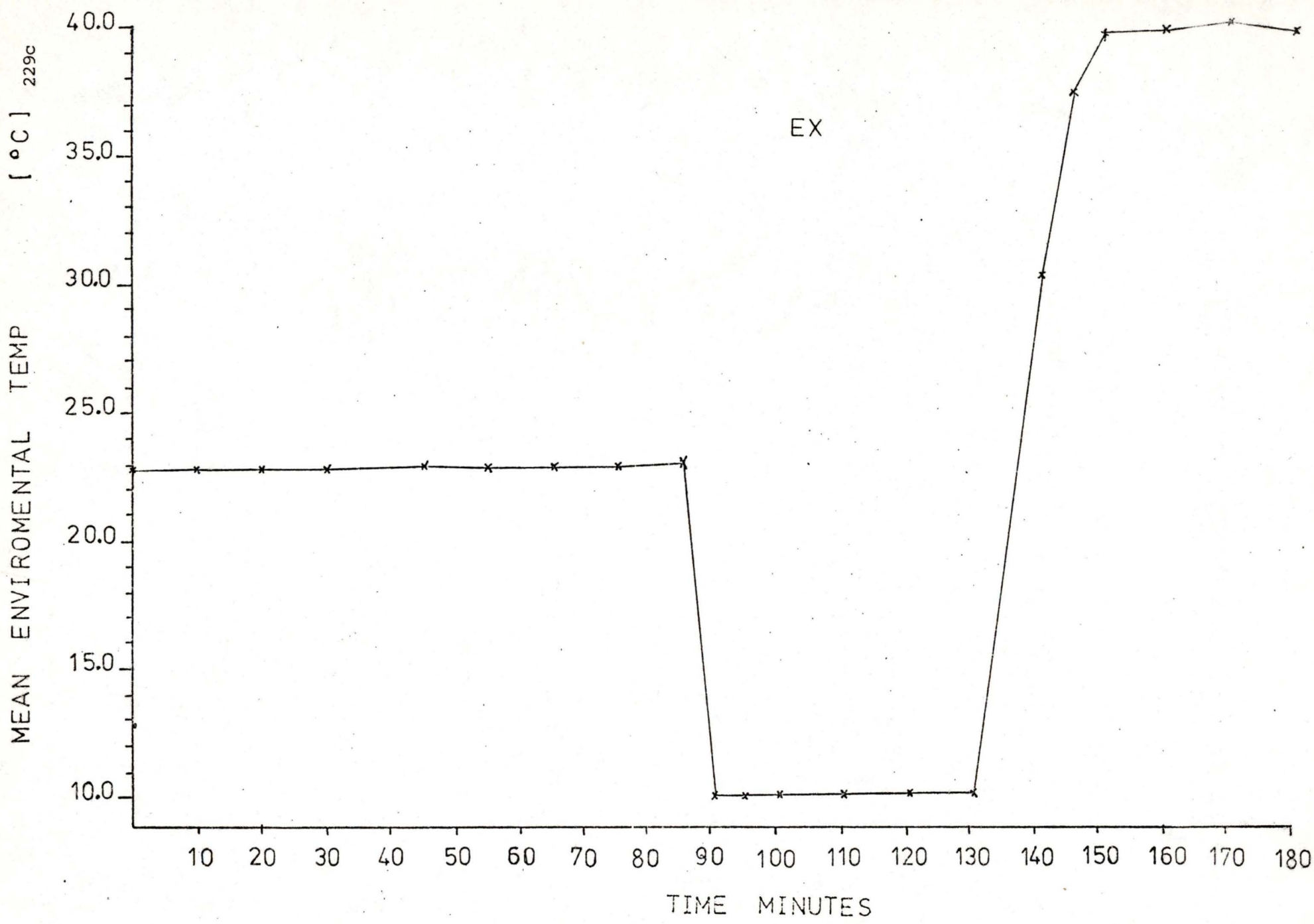
lt. = left n = 10 for all above means

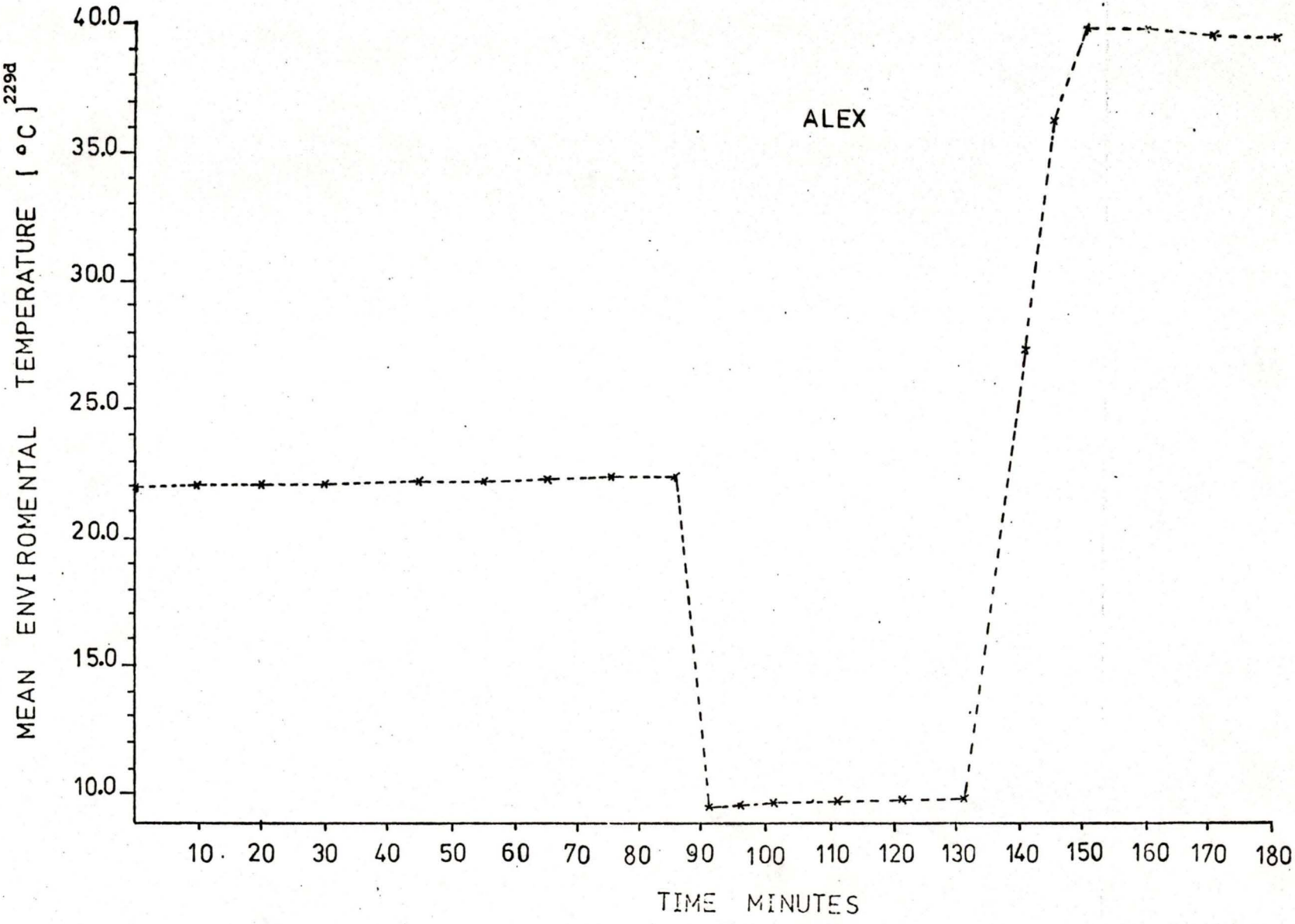
sd. = standard deviation of the mean

Appendix 22. Single treatment graphs used to construct the composite graph of Figure 15


CONTROL
MEAN ENVIROMENTAL TEMPERATURE [°C] 229a
40.0
35.0
30.0
25.0
20.0
15.0
10.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES


MEAN ENVIROMENTAL TEMPERATURE [°C] 229b
40.0
35.0
30.0
25.0
20.0
15.0
10.0
AL
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES


MEAN ENVIROMENTAL TEMP [°C] 229c
40.0
35.0
30.0
25.0
20.0
15.0
10.0
EX
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES


ALEX
MEAN ENVIROMENTAL TEMPERATURE [°C] 229d
40.0
35.0
30.0
25.0
20.0
15.0
10.0
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES

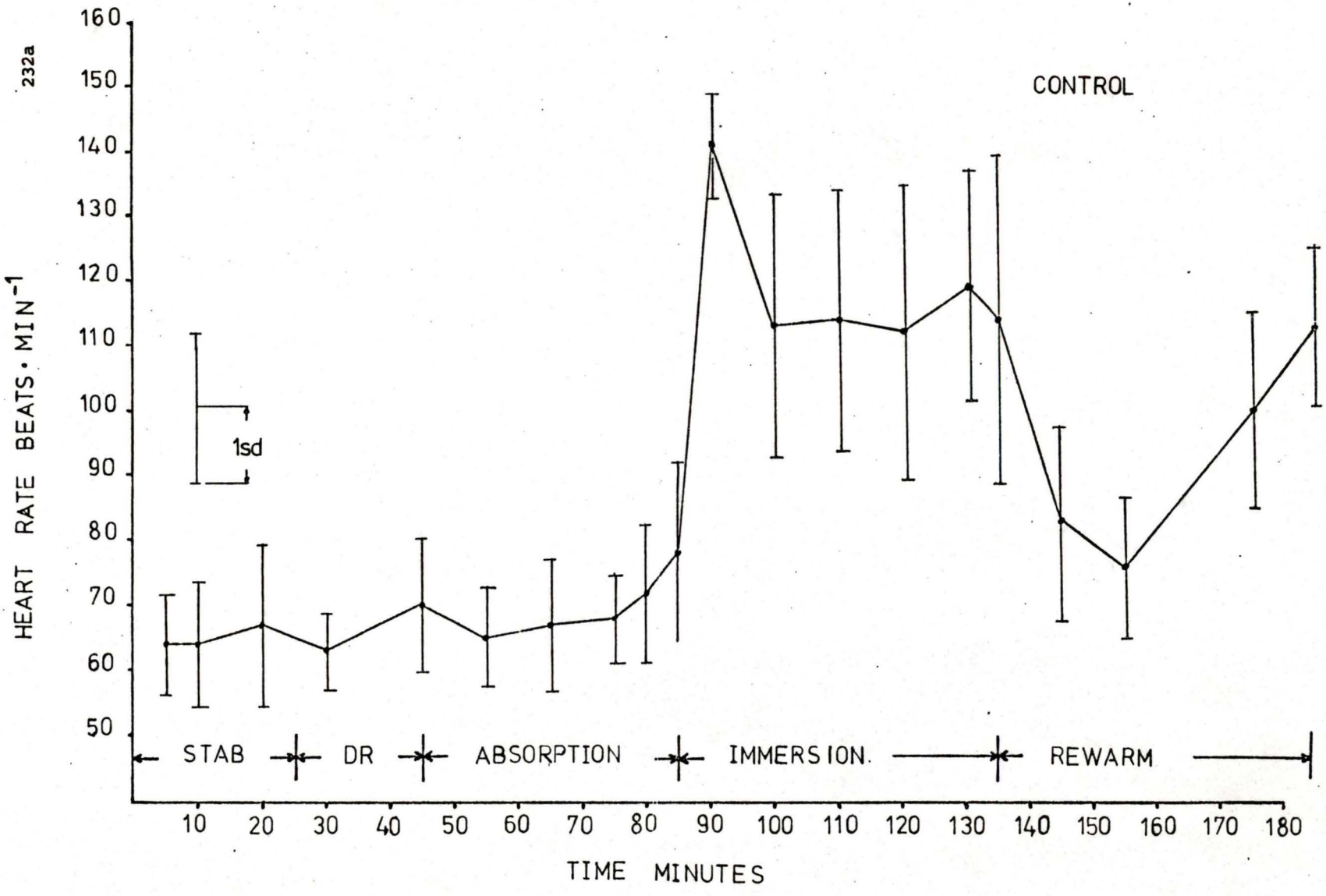
Appendix 23. List of means and standard deviations of the means observed at various times for environmental temperatures during; control, al, ex, and alex experiments

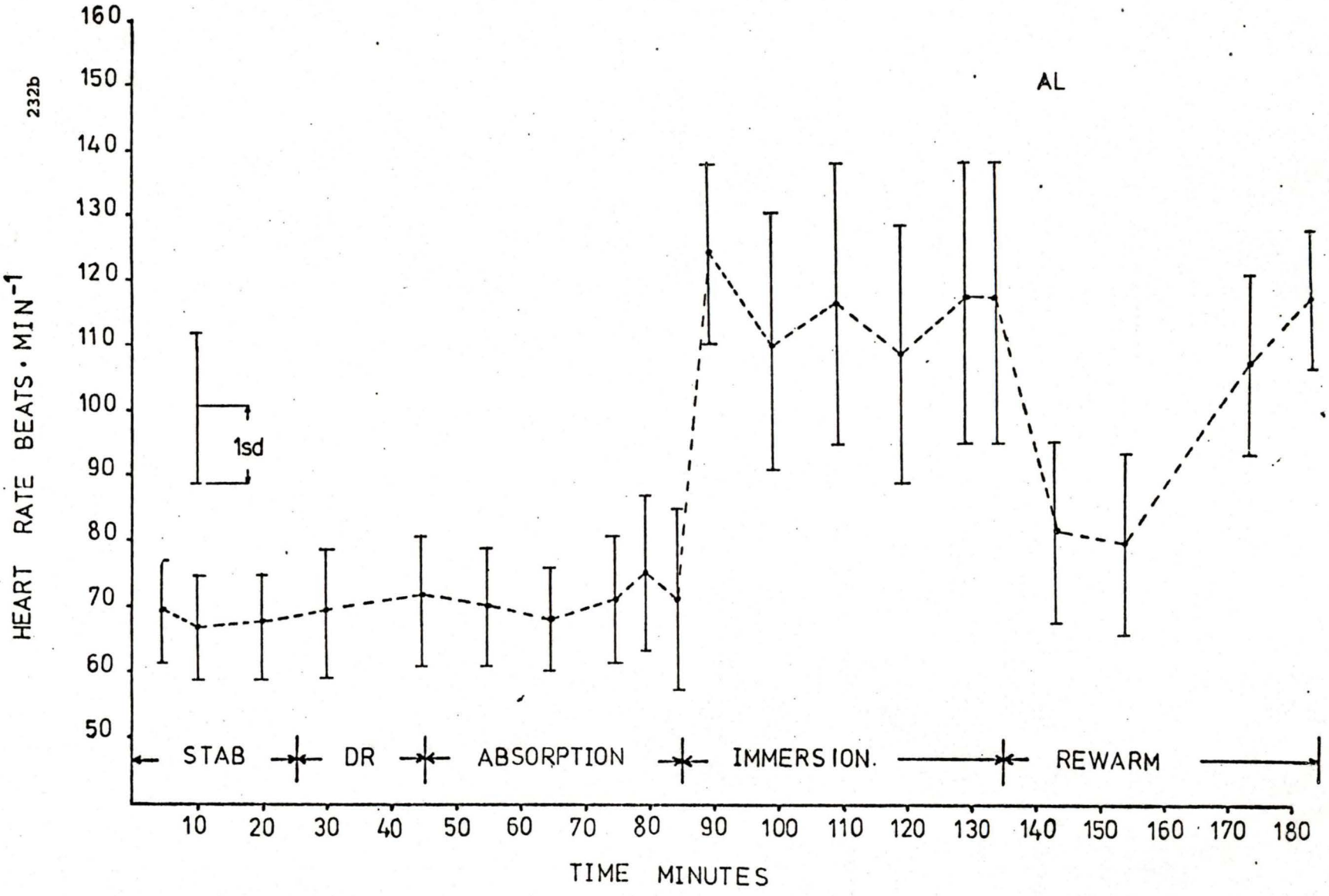
List 1. Means and standard deviations of environmental temperatures observed for control, al, ex and alex treatments

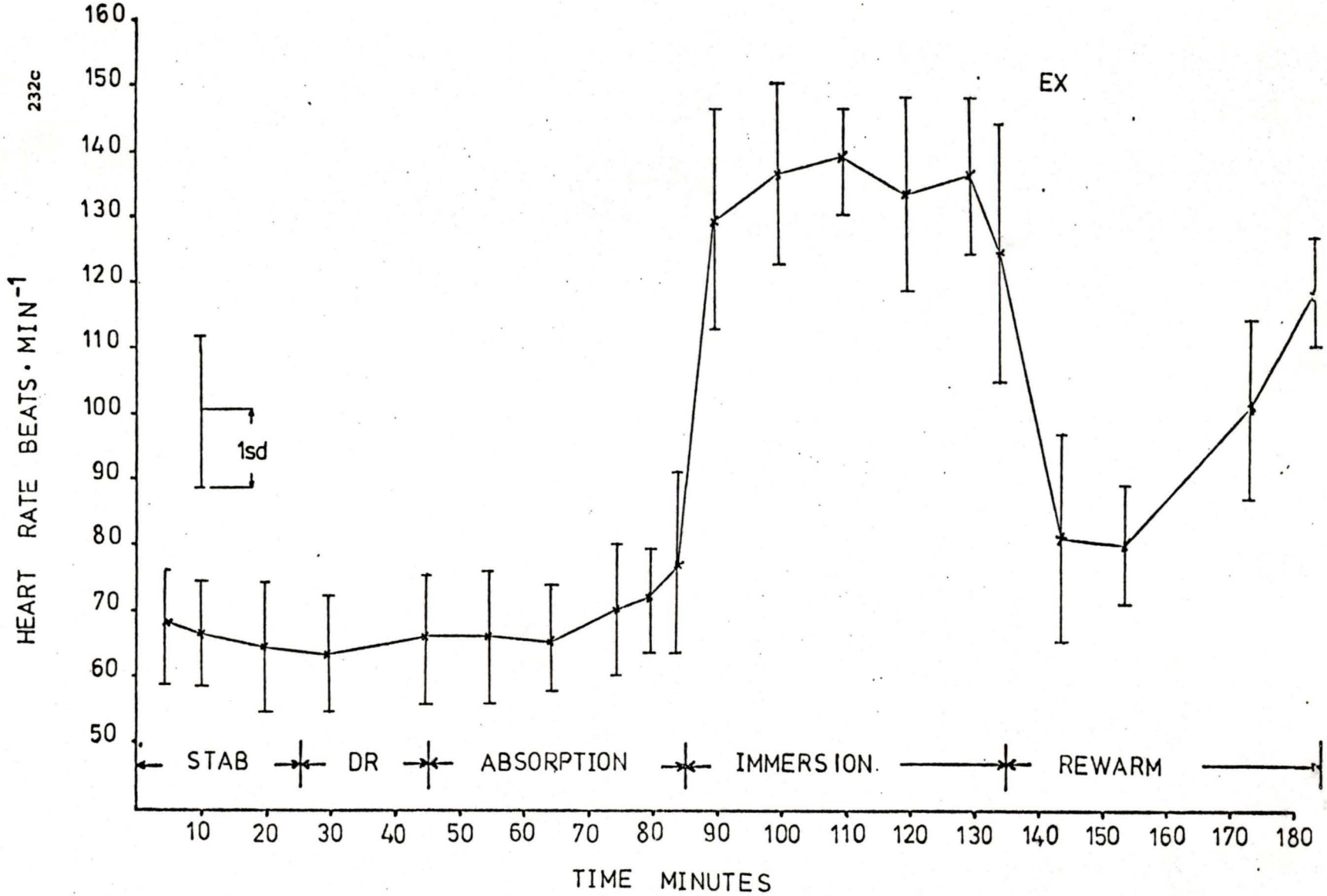
Time, minutes	Statistic	Treatments, control	al	ex	alex
0	mean	22.37	22.53	22.19	22.45
	sd	0.435	0.462	0.321	0.792
10	mean	22.44	22.59	22.43	22.48
	sd	0.395	0.378	0.440	0.760
30	mean	22.58	22.63	22.59	22.52
	sd	0.278	0.439	0.515	0.835
45	mean	22.63	22.73	22.61	22.57
	sd	0.312	0.347	0.517	0.787
55	mean	22.61	22.68	22.72	22.63
	sd	0.256	0.358	0.442	0.741
65	mean	22.68	22.74	22.75	22.70
	sd	0.270	0.357	0.570	0.766
75	mean	22.72	22.80	22.80	22.73
	sd	0.305	0.326	0.546	0.698
85	mean	22.77	22.86	22.87	22.78
	sd	0.287	0.334	0.487	0.702
90	mean	10.06	10.06	10.00	9.99
	sd	0.069	0.123	0.071	0.105
95	mean	10.14	10.09	10.04	10.06
	sd	0.096	0.119	0.097	0.107
100	mean	10.17	10.13	10.09	10.13
	sd	0.095	0.116	0.119	0.133
110	mean	10.21	10.20	10.13	10.17
	sd	0.087	0.115	0.132	0.106
120	mean	10.28	10.26	10.19	10.24
	sd	0.079	0.107	0.128	0.107
130	mean	10.32	10.30	10.24	10.30
	sd	0.079	0.105	0.126	0.122
140	mean	29.58	30.37	29.47	27.69
	sd	2.762	1.052	2.064	2.794
145	mean	37.83	37.39	37.83	36.48
	sd	2.296	2.372	1.849	2.495
150	mean	39.84	39.79	39.82	40.11
	sd	0.781	0.673	0.839	1.083
160	mean	39.82	39.87	39.63	40.07
	sd	0.597	0.343	0.332	0.439
170	mean	39.73	40.13	39.97	39.77
	sd	0.335	0.849	0.814	0.568
180	mean	39.84	39.90	40.09	39.68
	sd	0.226	0.611	0.706	0.653

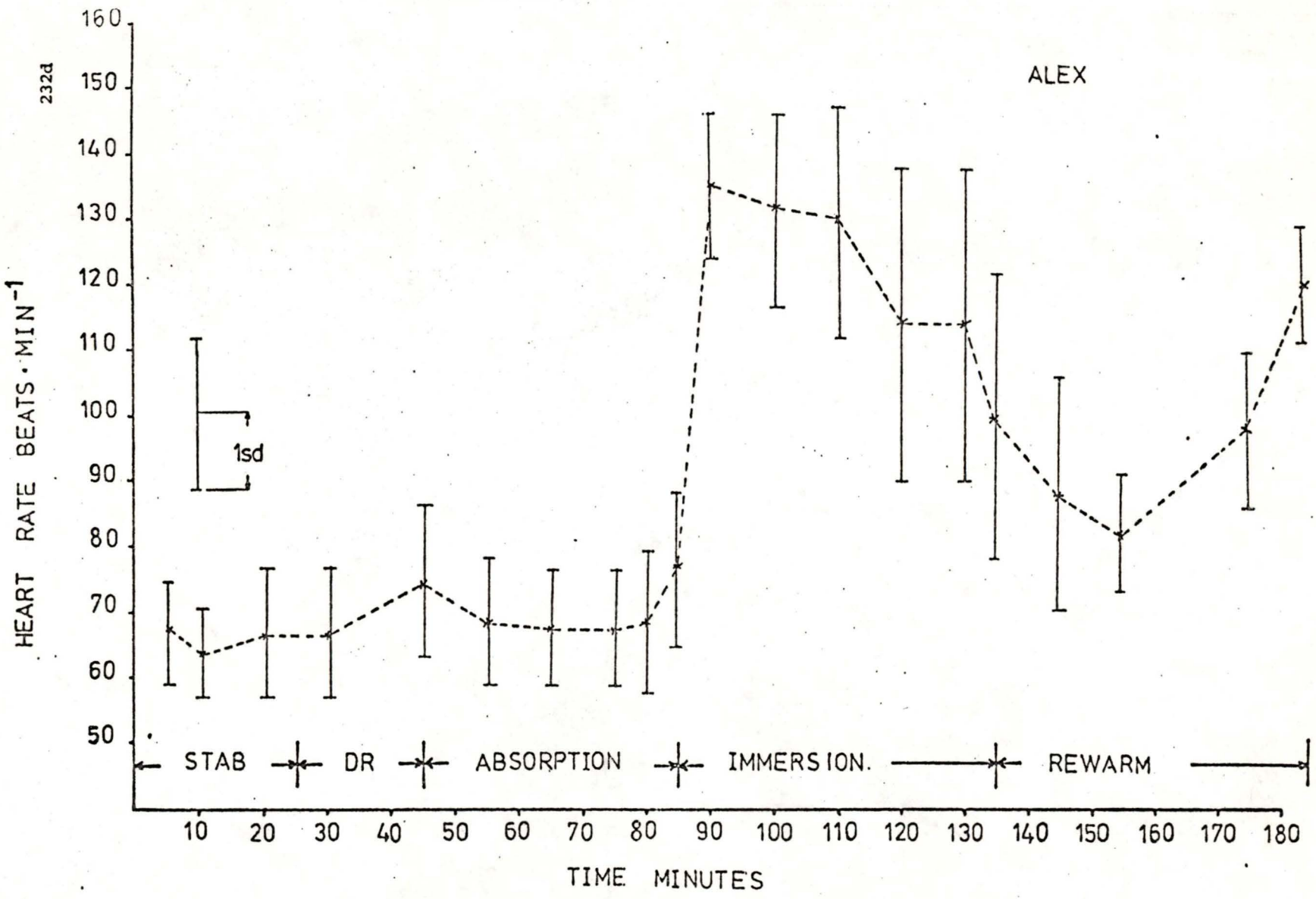
sd = standard deviation of the mean n = 10 for all above means

Appendix 24. Single treatment graphs used to construct the composite graph of Figure 16

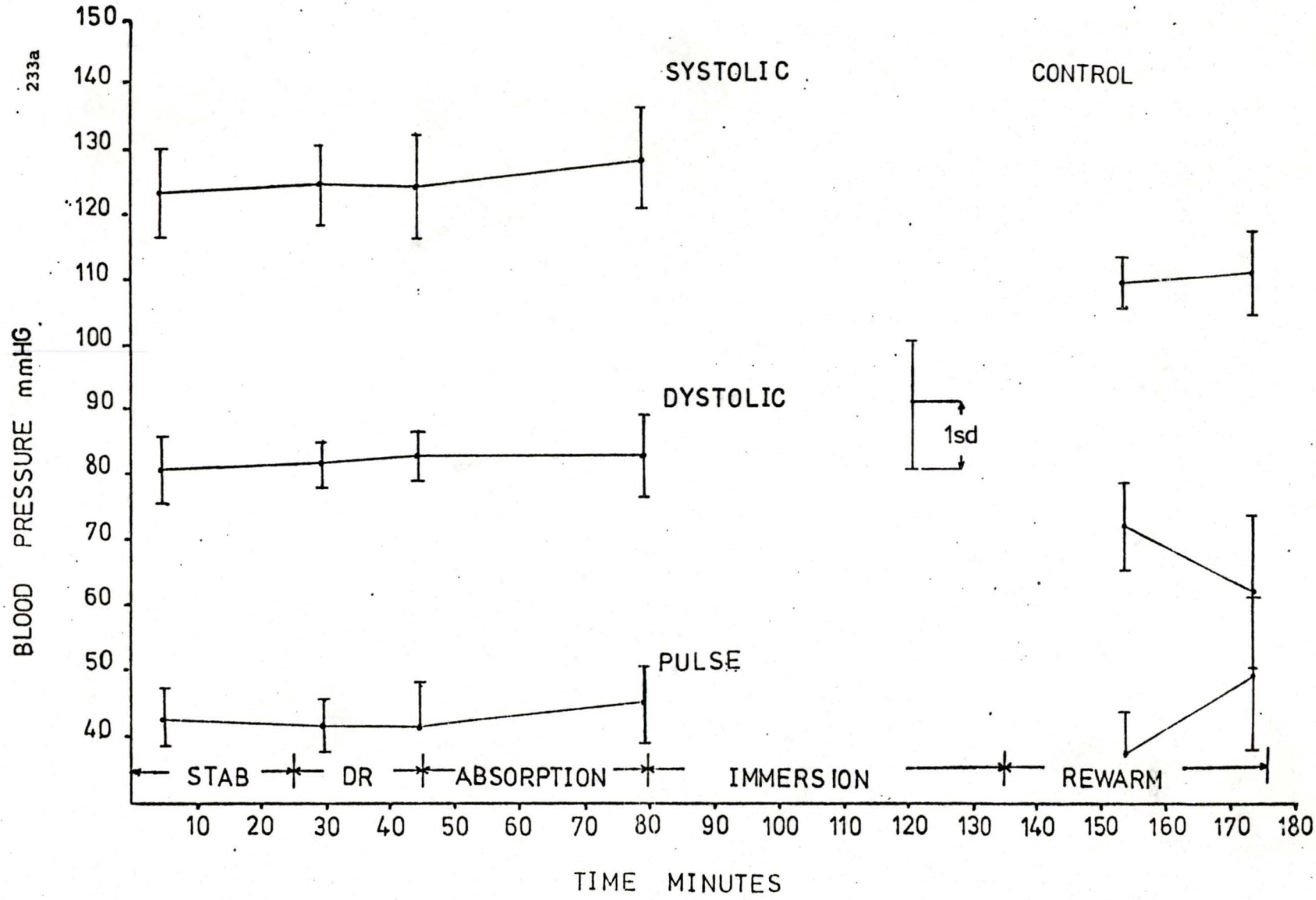

232a
CONTROL
HEART RATE BEATS · MIN⁻¹
160
150
140
130
120
110
100
90
80
70
60
50
1sd
STAB
DR
ABSORPTION
IMMERSION
REWARM
10
20
30
40
50
60
70
80
90
100
110
120
130
140
150
160
170
180
TIME MINUTES

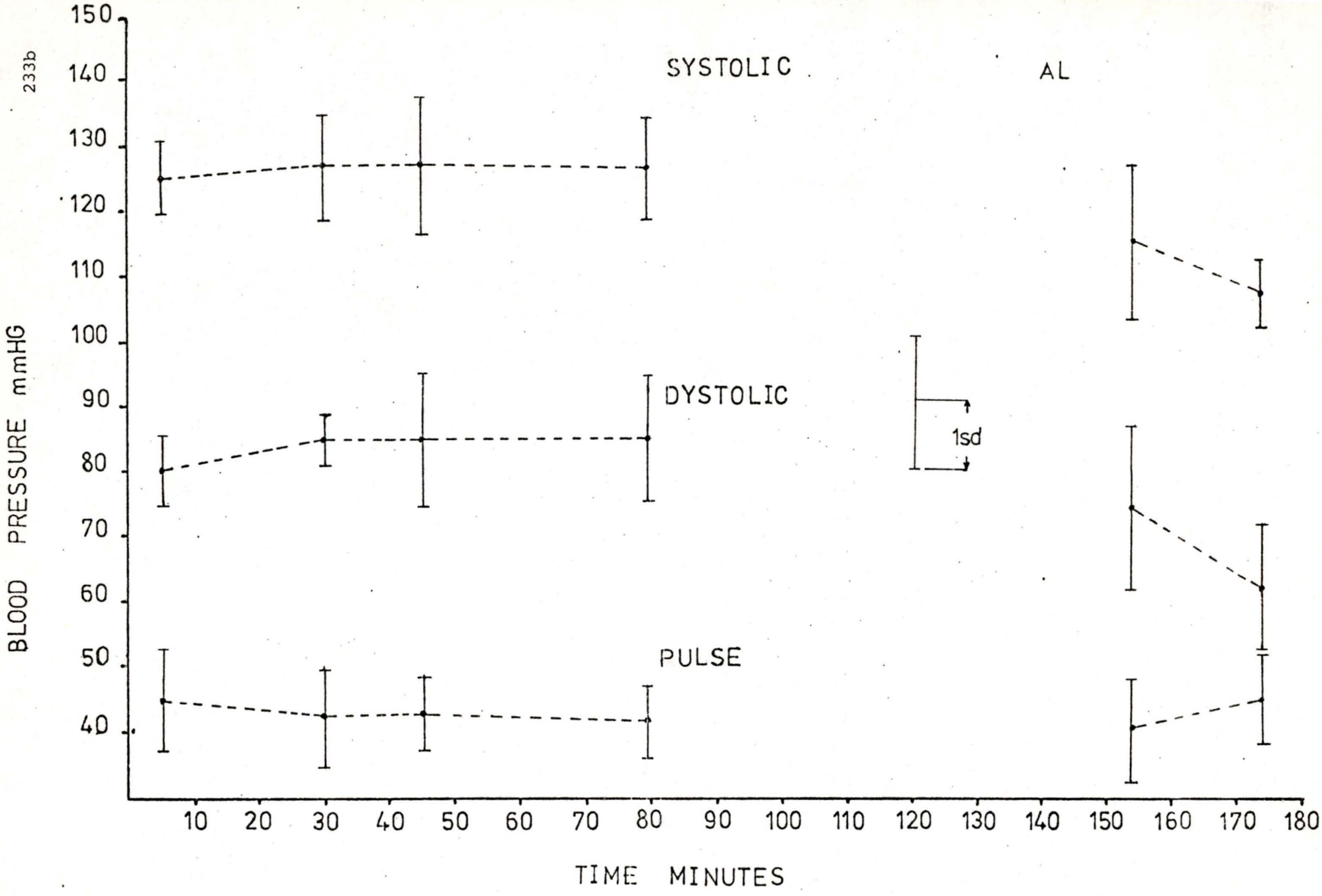

232b
AL
HEART RATE BEATS · MIN⁻¹
160
150
140
130
120
110
100
90
80
70
60
50
1sd
STAB
DR
ABSORPTION
IMMERSION.
REWARM
10
20
30
40
50
60
70
80
90
100
110
120
130
140
150
160
170
180
TIME MINUTES

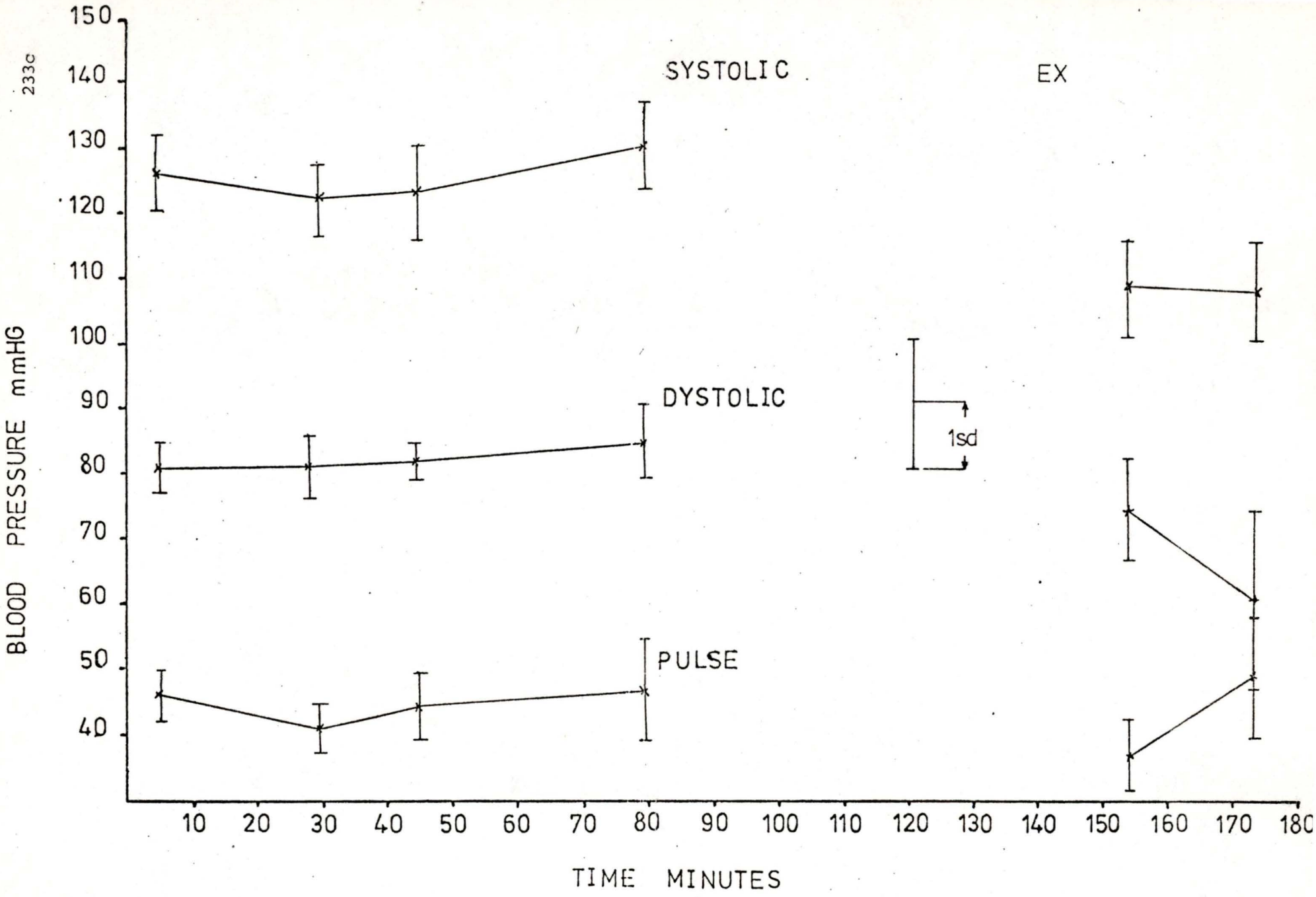

232c
HEART RATE BEATS • MIN⁻¹
160
150
140
130
120
110
100
90
80
70
60
50
EX
1sd
STAB
DR
ABSORPTION
IMMERSION.
REWARM
10
20
30
40
50
60
70
80
90
100
110
120
130
140
150
160
170
180
TIME MINUTES

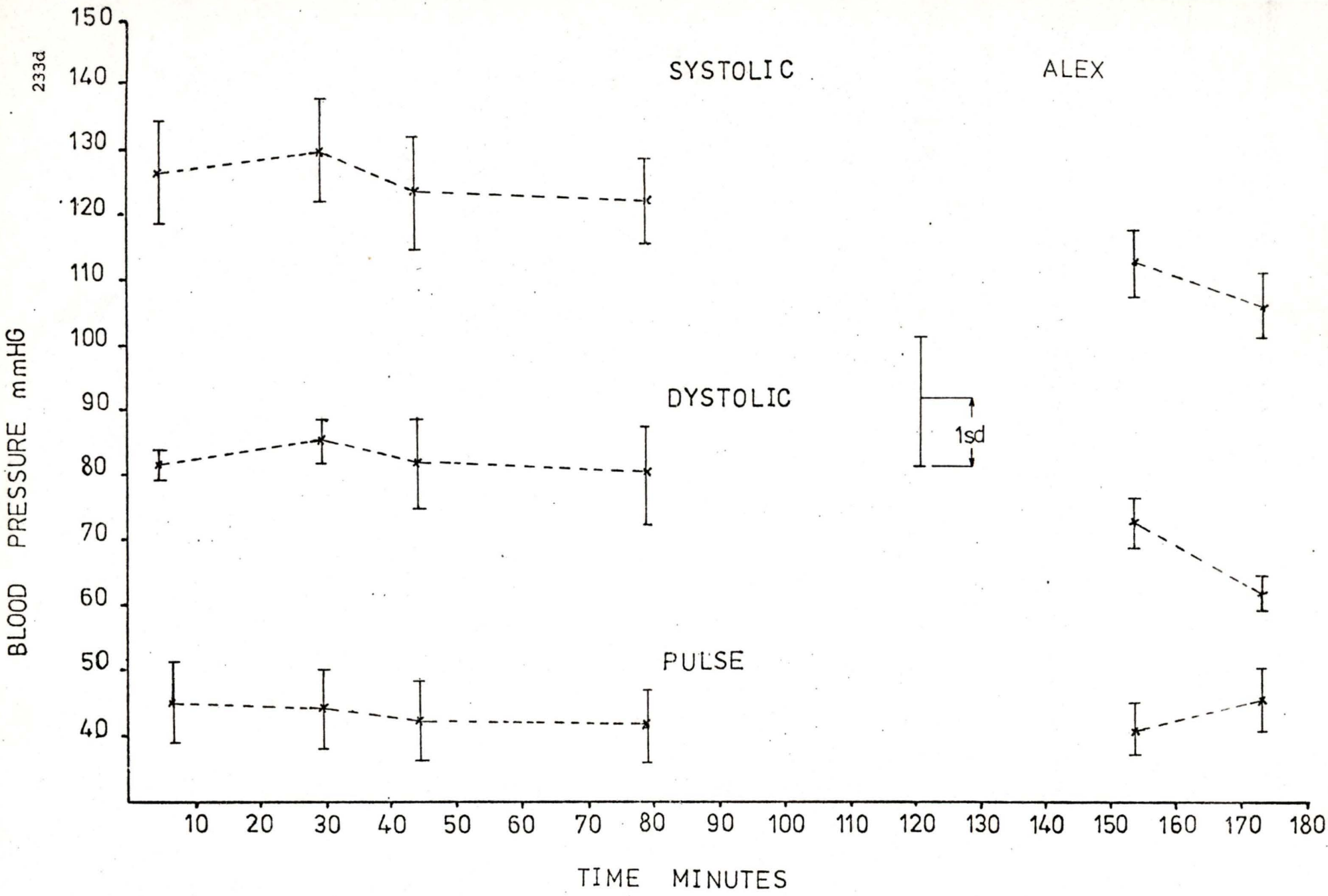

232d
ALEX
HEART RATE BEATS · MIN⁻¹
160
150
140
130
120
110
100
90
80
70
60
50
1sd
STAB
DR
ABSORPTION
IMMERSION.
REWARM
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES

Appendix 25. Single treatment graphs used to construct the composite graph of Figure 17


SYSTOLIC
CONTROL
DYSTOLIC
1sd
PULSE
BLOOD PRESSURE mmHG
150
140
130
120
110
100
90
80
70
60
50
40
STAB
DR
ABSORPTION
IMMERSION
REWARM
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES


233b
SYSTOLIC
AL
DYSTOLIC
1sd
PULSE
BLOOD PRESSURE mmHG
150
140
130
120
110
100
90
80
70
60
50
40
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
TIME MINUTES


233c
SYSTOLIC
EX
DYSTOLIC
1sd
PULSE
BLOOD PRESSURE mmHG
150
140
130
120
110
100
90
80
70
60
50
40
10
20
30
40
50
60
70
80
90
100
110
120
130
140
150
160
170
180
TIME MINUTES


233d
SYSTOLIC
ALEX
DYSTOLIC
1sd
PULSE
BLOOD PRESSURE mmHG
150
140
130
120
110
100
90
80
70
60
50
40
10
20
30
40
50
60
70
80
90
100
110
120
130
140
150
160
170
180
TIME MINUTES

Appendix 26. Mean BAC decay rates calculated for Hobson and Collis' (1977) study

I. Mean BAC decay rates calculated for Hobson and Collis' (1977) study.

From Hobson and Collis (1977)

Subject number	Time immersed, minutes	BAC, mgm% pre-immersion	BAC, mgm% post-immersion	BAC decay rate, $mgm\% \cdot hour^{-1}$
1	75	54.0	37.0	13.5
2	66	51.0	38.0	11.8
3	37	51.0	40.0	24.3
4	52	52.0	39.0	15.0
Mean	57.5	53.0	38.5	16.2
Standard deviation	16.62	18.3	1.29	5.55

VITA

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Degrees, Diplomas, Etc., Awarded, with Dates and Names of Institutions:

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Publications:

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THE EFFECTS OF ETHANOL WITH AND WITHOUT EXERCISE ON THE THERMAL BALANCE OF MAN IN COLD WATER

Author: GLYN R. FOX

AUGUST 1st 1978