MAN IN COLD WATER:
HEART RATE AND ELECTROCARDIOGRAPHIC RESPONSES

by

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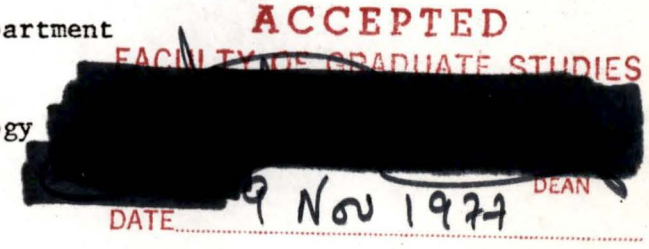
A THESIS SUBMITTED IN PARTIAL FULFILLMENT
OF THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF SCIENCE
in the Department
of
Biology



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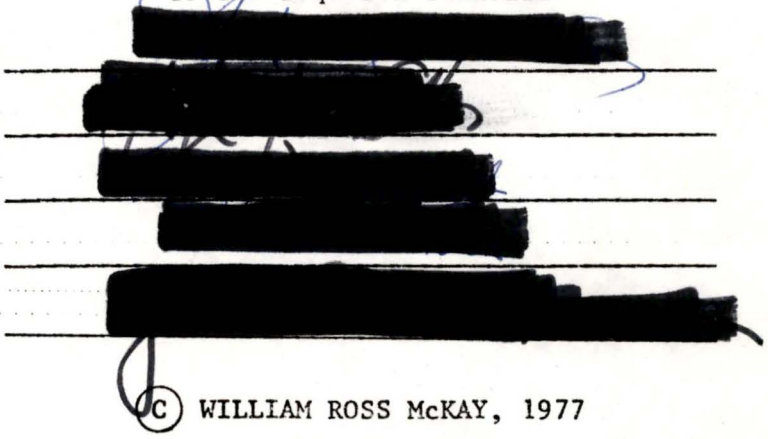

UNIVERSITY OF VICTORIA
August, 1977

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ABSTRACT

The heart rate and electrocardiographic (ECG) response to immersion in cold ocean water (4.6 - 18.2°C) was studied in conscious normal humans in relation to several variables. Individual responses were studied but most results were based on means of 12 (usually 6 male and 6 female) subjects. Continuous monitoring of heart rate, ECG, rectal temperature and oxygen consumption was carried out on subjects before and during cold immersion (until rectal temperature fell to 35.0°C), and subsequently during rewarming in a heated whirlpool bath.

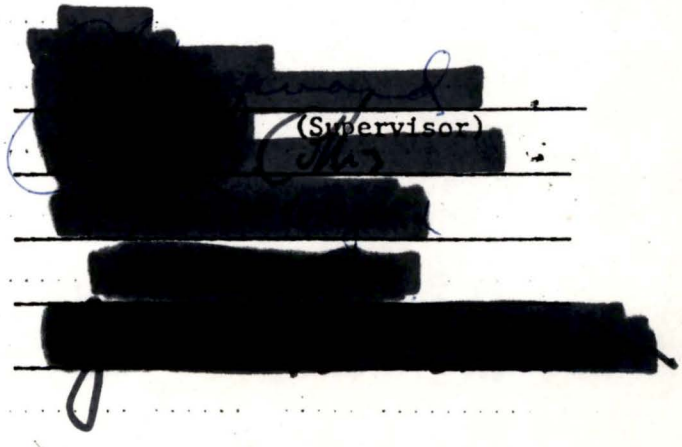
In immersions in water below 10.5°C where subjects held still, heart rate remained elevated (95 bpm at 4.6°C) or increased (82 bpm to 90 bpm at 10.5°C) as rectal temperature decreased while in the cold water. A positive correlation between heart rate and metabolic rate (oxygen consumption) was found, accounting for this increase. In the early rewarm period heart rate declined as rectal temperature continued to drop, then increased as subjects began to rewarm. In water at 18.2°C heart rate showed an overall increase of 5 bpm during the immersion. Although heart rate level was inversely related to ambient temperature, no significant differences were found in this relatively narrow range of temperatures. Heart rate level was directly related to the amount of thermal protection of clothing, but no significant differences were found. Sex and body size differences did not significantly affect heart rate response but due to confounding factors these relationships could not be accurately evaluated. Significant regressions of heart rate against rectal temperature showed a direct relationship in the first

22 minutes of cold immersion and the first 22 minutes of the rewarming phase but an inverse relationship between these periods. These relationships were complicated by the relationship of heart rate and metabolic rate, anxiety and muscular movement.

In immersions while subjects swam moderately, heart rate was significantly higher than while holding still (122 bpm active; 87 bpm still ($p < 0.01$)) but no significant difference was observed during the rewarming phase.

The intermittent ECG recordings of 33 subjects participating in 102 total immersions and cooling to an approximate rectal temperature of 34°C were analysed for significant changes. Quantitative analysis of ECG changes showed significant increases in the QTc interval and in the amplitude of the T wave with progressive hypothermia. PR and QRS time components were not increased significantly. Qualitatively, occasional atrial and ventricular extrasystoles and sinus arrhythmias were observed on immersion and with hypothermia. J point deflection, ST segment elevation and ST depression were only rarely seen.

No life-threatening cardiovascular changes were observed in this young, fit, healthy sample subjected to mild hypothermia. However, definite non-hypothermic and hypothermic stresses act upon subjects exposed to cold water. Their effect depends upon the age and health of those exposed and the severity of the exposure. These considerations are discussed.



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ACKNOWLEDGEMENT

To all the persons who assisted me in completing this thesis I would like to express my deep appreciation. In particular I thank my Supervisor, John Hayward, the members of my committee and my wife Susan for their encouragement and patience.

INTRODUCTION

The importance of adequate cardiac function in the survival of man accidentally immersed in cold water is self-evident: The failure of the heart to effectively pump blood either due to the initial stress of entry into cold water or due to subsequent hypothermia is equivalent with death. These two periods of potential cardiac distress are of interest in this study, the purpose of which is to examine the cardiovascular response to cold water immersion. There are three general situations in which the effects of cold exposure and body cooling have been studied previously: accidental, surgical and experimental. These studies have included both air and water environments.

Accidental hypothermia has been studied in both cold water immersion at sea (72, 99) and cold air (69, 96, 97, 25), and in most cases the subjects have been conscious. In the cold air, however, most subjects were above fifty years of age and were either alcoholic, diabetic or suffering from drug overdose. These reports as well as those on shipwreck victims do not contain physiological data on the initial exposure to cold, and reliable data commence only after subjects have reached a deep hypothermic state (body temperature $< 30^{\circ}$ C).

In surgical technology, hypothermia has been of interest as it enables reduction or cessation of tissue perfusion for long periods while surgical procedures are performed. (In accordance with van't Hoff's Law, the effect of cold on body tissues is to progressively reduce cellular metabolic processes (83)). Many studies of surgical hypothermia contain good cardiovascular data (45, 38, 88), but by their purpose do not apply to the accidental cold water immersion situation.

Deep hypothermia is of greatest interest in surgery and therefore the initial cooling stages from normal body temperature to 30°C are often not studied (e.g. 88). Anaesthetic agents mask responses such as shivering thermogenesis and the type of anaesthetic, depth of anaesthesia and rate of cooling vary greatly among these studies, complicating interpretation (80). In some studies patients had cardiovascular disease or congenital heart defects and therefore were abnormal (e.g. 52, 38). Gunton et al. (45), Rose et al. (88) and Emslie-Smith et al. (35) studied surgical patients free of cardiac problems but even so Rose et al. (88) concluded that uniform physiological responses could not be expected in the experimentally uncontrolled operating room setting.

Experimentally induced hypothermia has been administered to conscious man by exposure to air (23, 77, 85) and water (1, 13, 64). Conscious sheep (80), cats (84) and rats (5) have also been studied. Hypothermic anaesthetized experimental animals have been studied extensively including dogs (5, 50, 18, 37, 74), cats (55), rats (31, 5), hamsters (2), lambs (67), opossums (73) and isolated rabbit hearts (89). Although these have all contributed to the knowledge of cardiovascular responses to hypothermia, many of the results do not apply to the accidental cold water immersion situation.

Studies of conscious man often employed intermittent recording of parameters (e.g. 41, 64) which can lead to omission of changes in heart rate and electrocardiographic (ECG) wave form. Only a few subjects were observed in some studies (e.g. 13, 23, 86) while a significant level of hypothermia was not reached in others. The latter problem was due to

brevity of exposure (e.g. 23, 77), too great an ambient temperature (e.g. 41, 29), or thermal protection (20). Craig and Dvorak (30), studied the effects of exercising for one hour in relatively warm water ($\geq 24^{\circ}C$) as did Costill et al. (27) using 20 minute periods in water $\geq 17.4^{\circ}C$. Their experiments cannot be likened to prolonged swimming at lower water temperatures because hypothermia was minimal in both studies. Alexander (1), reported on the Dachau prison camp experiments which included continuous physiological monitoring of conscious humans in water between 2 and $12^{\circ}C$., but these subjects may have been physically weakened by prison life (72).

The use of experimental animals has been extensive, but one must be cautious in applying these results to the human (38, 88). Usually related to surgical hypothermia, these studies have mainly used anaesthesia and deep hypothermia again making comparison to accidental cold water immersion difficult.

The great variation in experimental populations and in methods of studying the response to hypothermia is evident in the varied conclusions drawn.

Progressive slowing of the heart rate with cooling, leading to sinus bradycardia is the common pattern in both deeply anaesthetized and conscious subjects with body temperatures less than about $32^{\circ}C$. (88, 18, 25). At body temperatures greater than this, it is well known that an increase in heat production through shivering accompanies exposure to cold stress in both mildly anaesthetized and conscious subjects (25, 77). Reports on what heart rate changes are concurrent, however, vary greatly. Heart rate increases initially remaining elevated but declining in anaesthetized dogs (50) and in conscious man in $5^{\circ}C$. water (1).

Conversely, Behnke and Yaglou (13) in water at 10°C. and Keatinge and Evans (64) in water at 5°C. found heart rate to increase initially, begin to decline and then increase well above control levels. At 15°C., however, Keatinge and Evans (64) found a gradual decline in rate to just below control levels.

In cold air at 5°C. Thauer (95) found no increase in either cardiac output or heart rate while Raven et al. (85) found a 78% increase in stroke volume and a slight increase in heart rate. With similar findings at 8°C., O'Hanlon and Horvath (77) attributed increased oxygen delivery to increases in both stroke volume and periferal extraction. It should be noted that in these cold air studies, the drop in rectal temperature was less than 1°C.

Rewarming of conscious subjects with significant hypothermia is characterized by an initial further drop in body temperature. Heart rate is elevated in the initial phases of rewarming, then declines below control levels and finally increases markedly once rectal temperature has stabilized (13). Although their monitoring ceased before a return to control body temperatures, heart rate likely would have continued to increase with increasing body temperatures as it does with anaesthetized subjects (74).

Electrocardiographic (ECG) changes associated with cold exposure and hypothermia are uniform with a few exceptions in both conscious (1, 96, 97) and anaesthetized (83, 52) subjects. With significant hypothermia the time components of the cardiac cycle become prolonged. The QT interval (electrical systole which includes depolarization and repolarization) is affected earliest and most, followed by the PR interval and QRS duration (83, 50).

Varied ECG configurational changes have been found. Inversion of the P wave after cooling to 25°C. (31) and ST segment elevation (83) are

reported. An upward deflection at the junction ("J") point between the QRS complex and the ST segment is found at body temperatures below 35°C., increasing in amplitude with decreasing temperature (78, 59, 35). T wave flattening and inversion occurs often at body temperatures below 32°C. (45, 74) but an increase in T amplitude has also been found in the same temperature range (89).

Rhythm disturbances vary in frequency and severity depending on levels of hypothermia and consciousness and on the presence or absence of cardiac disease. Due to progressive depression of higher centers of impulse formation (the Sino-Atrial node) arrhythmias often occur in the same sequence. Normal sinus rhythm is interrupted by a burst of sinus tachycardia upon initial cold exposure in mildly anaesthetized and conscious subjects (83, 64). Occasional atrial and ventricular premature contractions are reported at this time (64). Premature atrial contractions occur during early cooling (38) and are common at body temperatures below 30°C. (45). As previously mentioned, normal sinus rhythm slows with cooling becoming sinus bradycardia in deep hypothermia. Atrial fibrillation occurs at rectal temperatures below 30°C. in both conscious (1) and anaesthetized (45) subjects. Below 28°C. heart block, premature ventricular contractions and nodal rhythm may appear (1, 4, 69). Spontaneous ventricular fibrillation is a major cause of death below 30°C., affecting at least some subjects in most studies (83, 45, 35). In the Dachau experiments most deaths were attributed to cardiac asystole occurring at rectal temperatures below 27°C. (1).

When observations during rewarming were made, it is reported that changes in ECG time components, configuration and rhythm are reversible on return to control level body temperature or earlier (83, 13, 52, 74).

As outlined, these fragmented and often conflicting reports on the physiological responses to hypothermia reflect the need for more research. O'Hanlon and Horvath (77) point out that

> "few experiments have been designed to show quantitatively how different physiological systems interact during acute cold exposures. Absent are studies that show how these interactions develop and change over the course of time."

Specifically, there is little information on the response of conscious normal humans to immersion in cold water ($\angle 20^{\circ}C$) such as would be encountered in accidental immersion at sea. This dearth of literature is understandable considering the problems involved in such research: subjects are fearful of such an ordeal; they must be screened medically for safety; adequate cold water and rewarming methods must be available and technical problems of physiological monitoring must be solved. The Man Overboard Project at the University of Victoria, Canada, overcame these problems and began in 1971 to investigate the dangers of accidental cold water immersion and the value of certain prophylactic measures in minimizing these dangers.

One aspect of the project, the study of some cardiovascular parameters, is presented here. It attempts to answer the questions: what changes occur in the heart rate and electrocardiogram; how do these changes relate to other parameters; and at what point do the changes become life threatening? The subjects, conscious males and females were immersed in cold waters (range 4.6 - 18.2°C.) around Vancouver Island, B.C. and were rewarmed in a whirlpool bath. Rectal temperature, oxygen consumption, heart rate and electrocardiogram were continuously monitored while the effects of ambient temperature, activity, thermal protection, sex and body size were studied.

It is hoped that the findings presented here will elucidate some aspects of the cardiovascular response to accidental cold water immersion that claims many lives annually.

METHODS

The data for this study is part of the data bank of the Man Overboard Project. The series of immersions were carried out between August, 1971 and March, 1973 and are summarized in Table 1. All but one series were conducted in ocean waters around Vancouver Island from Canadian Government research vessels. The one other series (12) used tanks of fresh water.

The effect of different sea temperatures was studied in Series 1, 2 and 3, in which the subjects were as still as possible in the water. Moderate swimming (30 strokes per minute) was studied in Series 4 at the same sea temperature as Series 2. Within Series 2 sex and body size variables were also studied. The effects of thermal protection clothing were studied in Series 10. Electrocardiographic analyses were done in Series 12 and 13 and pertinent ECG data were obtained in all series.

The subjects, mainly students at the University of Victoria, were between 19 and 29 years old (the 3 senior scientists were 34, 35 and 45 years). Subjects were selected as average individuals in body size and fatness. Females were appromimately 3/4 the body size of males and had slightly greater amounts of subcutaneous fat as indicated by skinfold thickness. Screening included a physical examination and monitored ECG exercise test. An informed consent was signed by each subject. Table 2 summarizes subject statistics.

For this study an "immersion" is one subject's preparation, exposure to cold water and rewarming. A "series" is a group of immersions designed to study one or more effects of cold water exposure. A series may consist of all individual subjects or a few subjects repeatedly immersed with

TABLE 1 SUMMARY OF DATA COLLECTION PROGRAM

Series	Date	Location	Water Temp.[1]	Activity	Attire[2]	No. of Imm.	Subjects M	Subjects F
1	Jan. 5-6 1972	Drew Harbour	4.6 ± 0.1	Still	2	11	6	5
2	Aug. 16,20 1971	Pedder Bay	10.5 ± 0.1	Still	2	12	6	6
3	Aug. 17-19 1971	Departure Bay	18.2 ± 0.2	Still	2	12	6	6
4	Sept. 16-18 1971	Beechey Head	10.5 ± 0.1	Swimming (30 st. per min.)	2	12	6	6
5	Dec. 8-10 1971	Saanich Inlet	6.3 ± 0.2	Treading	1	12	6	6
6	Feb. 23-25 1972	Sherringham & Swiftsure	7.0 ± 0.3	Still or Treading	2	11	5	6
8	May 23-27 1972	Barkley So.	12.5 ± 0.2	Still or Swimming	2,3,4,5	20	5	0
9	Sept. 5-7 1972	San Juan Harbour	11.9 ± 0.4	Still or Sculling	2,6,7	12	3	0
10	Nov. 14-17 1972	Saanich Inlet	8.8 ± 0.2	Still	2,5,8	15	4	1
11	Jan. 1973	Strait of Juan de Fuca	7.0	Still	1	6	4	0
12	Mar. 1973	University of Victoria	10.5 ± 0.5	Still	1	8	3	5
13	Oct. 1972- Mar. 1973	Cadbord Bay & U.V.I.C.	9.5	Varied	2	4	3	1

1 Mean ± S.E., °C
2 See Table 4

TABLE 2. SUBJECT STATISTICS
(for series analysed only)

Series No.	Sex	No. of Subjects	Age (Years)[1]	Height (cm)	Weight (Kg)	Triceps Skinfold (mm)
1	M	6	30.0 ± 3.7	183.3 ± 1.8	83.7 ± 1.4	9.6 ± 0.9
	F	5	19.6 ± 0.4	163.1 ± 2.6	54.0 ± 1.2	10.6 ± 0.7
	TOT.	11	25.3 ± 2.6	174.1 ± 3.5	70.2 ± 4.8	10.0 ± 0.6
2	M	6	31.5 ± 3.0	179.1 ± 1.7	79.3 ± 1.9	8.9 ± 1.0
	F	6	22.2 ± 1.2	163.4 ± 2.1	56.6 ± 1.9	15.5 ± 1.6
	TOT.	12	26.8 ± 2.1	171.2 ± 2.7	67.9 ± 3.7	12.2 ± 1.4
3	M	6	31.5 ± 3.0	179.1 ± 1.7	79.3 ± 1.9	8.9 ± 1.0
	F	6	22.2 ± 1.2	163.4 ± 2.1	56.6 ± 1.9	15.5 ± 1.6
	TOT.	12	26.8 ± 2.1	171.2 ± 2.7	67.9 ± 3.7	12.2 ± 1.4
4	M	6	30.7 ± 3.5	180.8 ± 2.2	81.9 ± 1.9	9.0 ± 1.0
	F	6	22.5 ± 0.7	168.9 ± 1.9	60.6 ± 3.2	14.8 ± 1.6
	TOT.	12	26.6 ± 2.1	174.8 ± 2.3	71.2 ± 3.7	11.9 ± 1.3
10	ALL	5	32.0 ± 4.8	178.1 ± 3.5	74.4 ± 5.4	11.7 ± 1.8
ECG STUDIES: (Includes Several Series)						
(a) TIME COMPONENTS (SER. 1,6,12,13)	M	6				
	F	4				
	TOT.	10	26.0 ± 2.6	175.6 ± 3.2	71.5 ± 4.2	11.7 ± 1.1
(b) T WAVE AMPLITUDE (SER. 12,13)	M	3				
	F	4				
	TOT.	7	21.1 ± 0.3	173.3 ± 3.6	65.1 ± 4.6	11.6 ± 1.0
(c) DYSRYTHMIAS (SER. 1-6, 8-13)	M	14	25.9 ± 1.8	180.0 ± 1.9	77.2 ± 2.0	8.8 ± 0.5
	F	19	21.8 ± 0.7	166.1 ± 1.2	57.4 ± 1.3	13.7 ± 0.8
	TOT.	33	23.6 ± 0.9	172.0 ± 1.6	67.3 ± 1.2	11.2 ± 0.6

1. Mean ± S.E.

different treatments. "Hypothermia" here is defined as having a rectal temperature lower than control temperature. Subjects were removed from the cold water at or before a critical rectal temperature of 35.0°C. Significant hypothermia was considered to be a drop of $\geq$ 1.0°C. from pre-immersion value. "Cold" water is defined as water with a temperature of $<$ 20°C. The range of water temperatures used was 4.6 - 18.2°C. Table 3 outlines cold exposure in the series analysed and Table 4 lists abbreviations used in this paper.

EXPERIMENTAL FORMAT

Subject Preparation

Subjects were dressed in standard clothing consisting of light cotton pants, long sleeved cotton shirt, sox, briefs (2-piece bathing suits for females) and running shoes. In addition to this, a number of floatation-thermal protection devices were worn in various series (Table 1) and these are defined in Table 4. Monitoring devices were applied as follows: A rectal thermistor (Yellow Springs Instrument Co.) was inserted into the rectum 15 cm. ECG silver-silver chloride column gel electrodes (E.M. Co.) were attached following vigorous skin abrasion with isopropanol, then sealed with rubber surgical adhesive. A five thoracic electrode configuration (70) was used in all series except 2, 3 and 4 but lead II was almost exclusively monitored. In Series 2, 3 and 4 the CM_5 lead (17) was used (Fig. 1). A modified diving mask with one way air flow was placed on the subject and attached to a 30 meter umbilicus of polyvinyl tubing through which all lead wires travelled. Expired air was vacuum pumped through the umbilicus for analysis of oxygen consumption by a potentiometric oxygen analyser with graphic display (Beckman Instruments Ltd.; Hewlett-Packard Ltd.).

TABLE 3. EXPOSURE TO COLD

Series	No. of Subj.	No. of Imm.	Time in Cold Water (min.)	Decline in T Rectal (°C)
1	11	11	31 ± 2	2.7 ± 0.2
2	12	12	50 ± 2	3.0 ± 0.1
3	12	12	66 ± 2	2.2 ± 0.2
4	12	12	47 ± 3	3.1 ± 0.2
10	5	15	51 ± 2	3.0 ± 0.2
ECG (a) TIME COMPONENTS ANALYSIS	10	12	38 ± 3	3.0 ± 0.2
(b) T WAVE ANALYSIS	7	8	49 ± 3	2.5 ± 0.2

1. Mean ± S.E.

TABLE 4. ABBREVIATIONS

A. General

T_A	Ambient temperature
T_B	Body temperature
T_R	Rectal temperature
T_L	Laboratory temperature
T_T	Tub temperature
HR	Heart rate
BP	Blood pressure
ECG	Electrocardiogram
VO_2	Oxygen consumption
MR	Metabolic rate
bpm	beats per minute

B. Clothing and Buoyancy Devices Code

1.	STD.	= Standard clothing described in text
2.	M.O.T.	= STD. plus Ministry of Transport approved Kapok life vest
3.	1/8 NEO.	= 1/8" thick neoprene wet suit top
4.	¼ NEO.	= ¼" thick neoprene wet suit top
5.	FLO.	= Floater type jacket
6.	MOD. FLO.	= FLO. modified with leg straps
7.	B.B.	= MOD. FLO. plus beavertail and neckflaps
8.	UVIC SPECIAL	= MOD. FLO. with refinements

Air flow was measured by a gas meter (American Instrument Co.) and expired air temperature at the oxygen analyser was recorded on a Thermofax thermocouple recorder (Leeds-Northrup Co.). Rectal temperature and heart rate were recorded on a Beckman S411 Dynograph. ECG was monitored by a Hewlett-Packard model 141 oscilloscope with polaroid camera and a direct writing electrocardiograph (Sanborn Instrument Co.) recorded ECG tracings. Instrument calibration was done preceding each series and checked during the series.

Immersion Phases

Once prepared, subjects commenced the five phase immersion procedure:

I Rest Period

Baseline control recordings during 10 minutes of sitting quietly in the laboratory. The most stable 5 minute period was used as a control recording.

II Pre-immersion

Movement from laboratory to a ladder on the ship's hull and into the water. Mean time was 2 minutes.

III Cold Immersion

Subject oriented himself and either remained STILL with head and neck above water and hands on a life ring, swam at 30 strokes per minute (SWIMMING), or treaded water (TREADING). Time was 25 to 75 minutes, limited by a critical rectal temperature of 35.0°C and by subject's desire to stay in the water.

IV Post Immersion

Subject returned to laboratory and entered rewarm whirlpool bath. Mean time was 2 minutes.

V Rewarming

The water was warmed from 25°C. to 42°C. over approximately 10

minutes. The subject remained in bath until rectal temperature began to increase. Mean time 35 minutes.

Data Collection Protocol

Throughout all phases of the experiment continuous recordings of oxygen consumption, expired air temperature, heart rate and rectal temperature were made. Intermittent recordings of the ECG were made at approximately 10 minute intervals or when changes in waveform or rhythm were noted. Recordings of subjective observations and the subjects' comments were made. Ancillary data such as sea temperature, barometric pressure, laboratory temperature, outside air temperature wind, current and beaufort scale were recorded once for each immersion.

Continuous monitoring was a precautionary measure and technicians were trained in recognition of physiological limitation signs. Cardio-pulmonary resuscitation including defibrillation was available, but fortunately was not required in over 250 immersions. Subjects were free to curtail their involvement at any time.

Data Analysis

These objective and subjective data comprised a comprehensive data package for each immersion. Some data was not considered valid (such as heart rate and ECG recordings that were technically poor and/or intermittent) and were excluded. Statistical analysis was done both manually and by computer. Tests for significance of difference included student Newman Keul's and student t tests. Pearson correlation coefficient was used to test for significance of regression and correlations. When series mean values were plotted against time, the maximum time shown for any phase of the immersion was the mean phase time of all subjects in that series, to the closest minute. This eliminated the problem of different individual phase time lengths and was considered not to bias

the value of the dependent variable even though some subjects had a shorter phase time than the mean. In the analysis of ECG time components with respect to rectal temperature, measurements were lacking for some subjects at some rectal temperatures. A computer program interpolated values for these rectal temperatures based on available values for those subjects. Thus, values were available for 12 subjects at all rectal temperatures studied.

Figure 1 is a schematic diagram of the laboratory and instrumentation.

Fig. 1. Schematic Diagram of Experimental Operation.

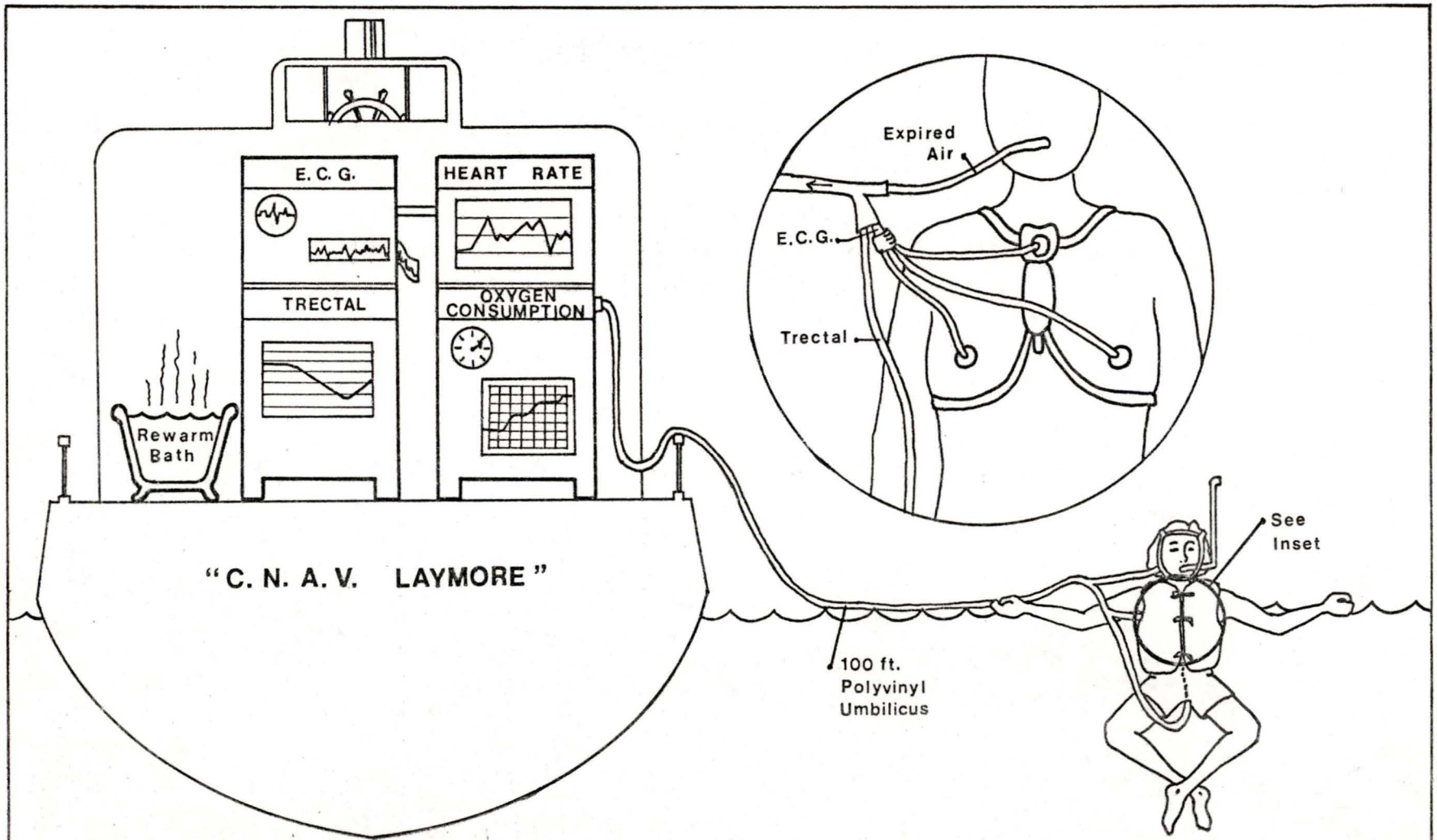


Figure I. Schematic diagram of experimental operation.

RESULTS

This section is presented in two sub-sections, the effect of cold water exposure and mild hypothermia on heart rate and on the electrocardiogram.

HEART RATE RESPONSE

Individual Records

It is useful to examine the individual subject response of heart rate because there are minute to minute variations that are lost in the averaging process and because the continuous recording through the whole immersion can be presented. The rectal temperature and heart rate records for one male and one female 26 year old subject who were immersed at 10.5°C in Series 2 are shown in Figures 2 to 5.

Using subject P.B. as an example (Fig. 2, 3), we note that in Phase I (the pre-immersion period) the rectal temperature is constant at 37.6°C and heart rate varies between 85 and 91 bpm. Phase II (between I and cold immersion) shows a rapid increase in heart rate peaking at 130 bpm and gradually decreasing for the first 25 minutes of immersion. Periodic fluctuations of about 10 bpm occur every 2 to 4 minutes during this overall decline and throughout the immersion and rewarming period. Seen in approximately half of the subjects are 5 bpm fluctuations in heart rate occuring about 8 times per minute. They are not coincident with but possibly influenced by respiration rate. Fig. 5 shows examples of these minor fluctuations which are not seen in the middle rewarming period when rectal temperature is at its lowest and shivering has subsided.

In Fig. 3, after 25 minutes the heart rate increases from 90 bpm to 105 bpm at the end of cold immersion. During this period shivering becomes progressively more intense and rectal temperature, which was stable for

Fig. 2. Exemplary Rectal Temperature Curve in One Female Subject (P.B., 26 Years Old) Immersed in 10.5°C Water While Holding Still.

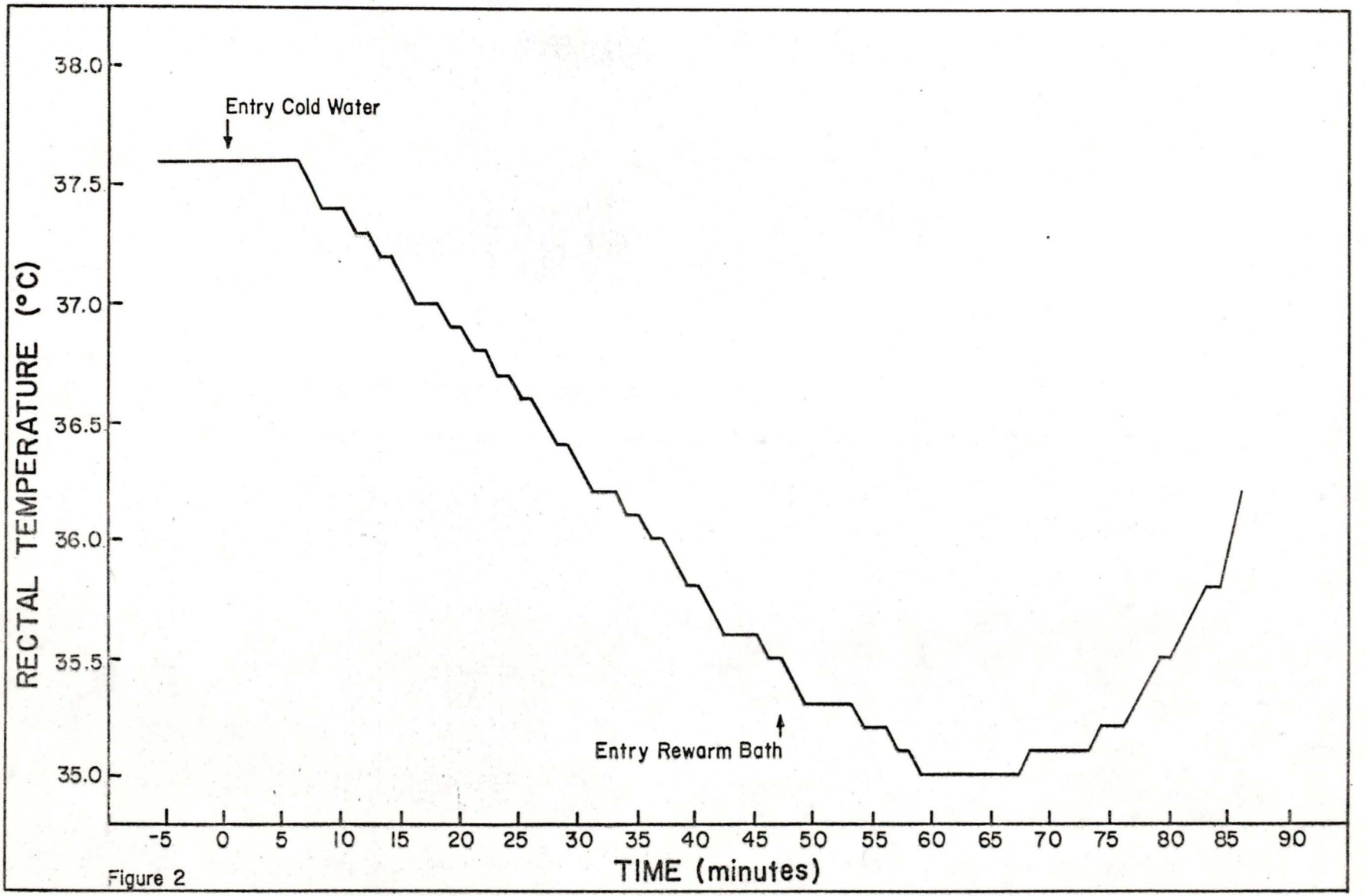

38.0
37.5
37.0
36.5
36.0
35.5
35.0
RECTAL TEMPERATURE (°C)
Entry Cold Water
Entry Rewarm Bath
-5
0
5
10
15
20
25
30
35
40
45
50
55
60
65
70
75
80
85
90
TIME (minutes)

Figure 2

Fig. 3. Exemplary Heart Rate Curve in One Female Subject (P.B., 26 Years Old) Immersed in 10.5°C Water While Holding Still.

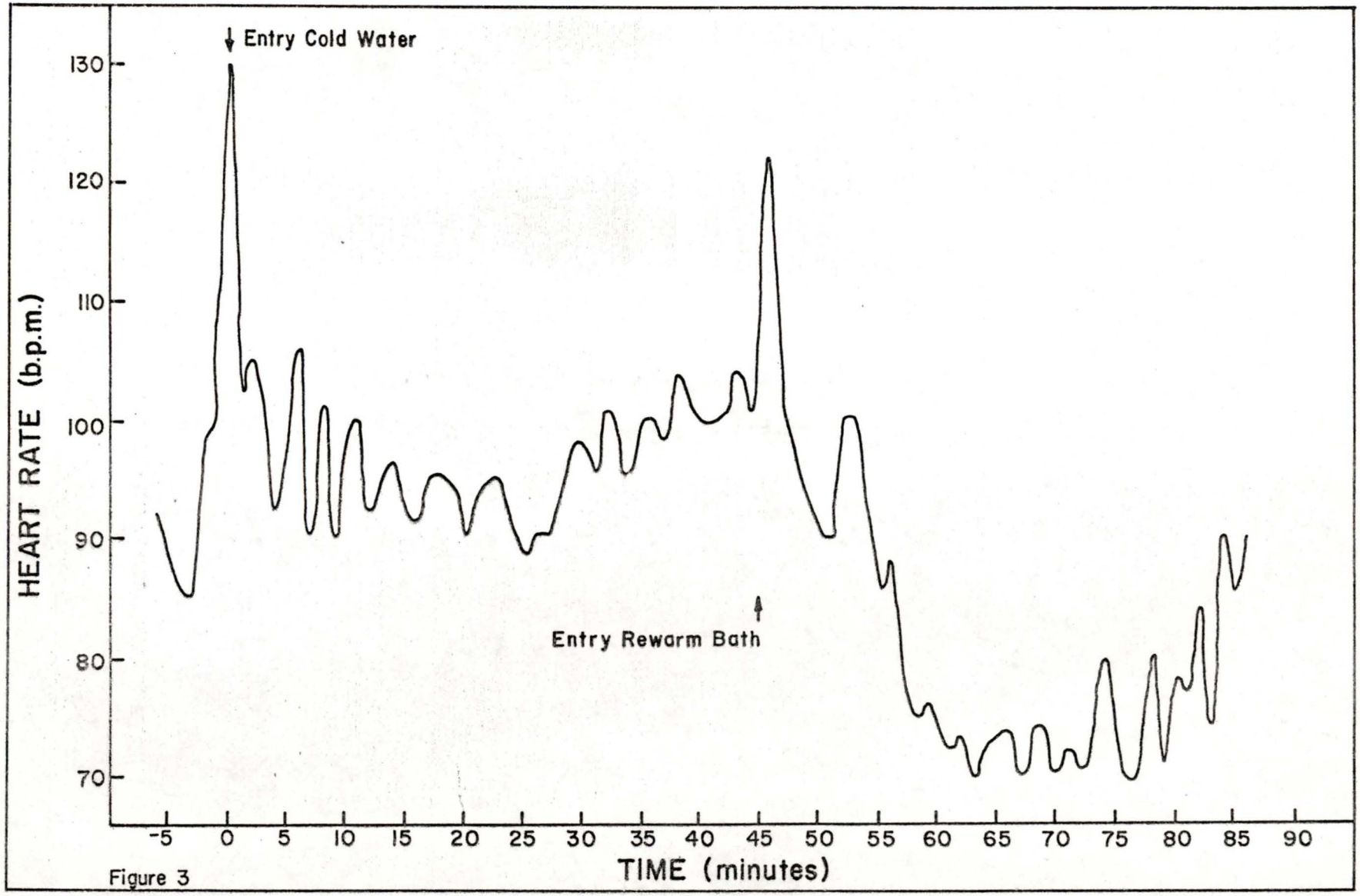

Entry Cold Water
Entry Rewarm Bath
HEART RATE (b.p.m.)
130
120
110
100
90
80
70
-5
0
5
10
15
20
25
30
35
40
45
50
55
60
65
70
75
80
85
90
TIME (minutes)

Figure 3

Fig. 4. Exemplary Heart Rate Curve in One Male Subject (P.W., 26 Years Old) Immersed in 10.5°C Water While Holding Still.

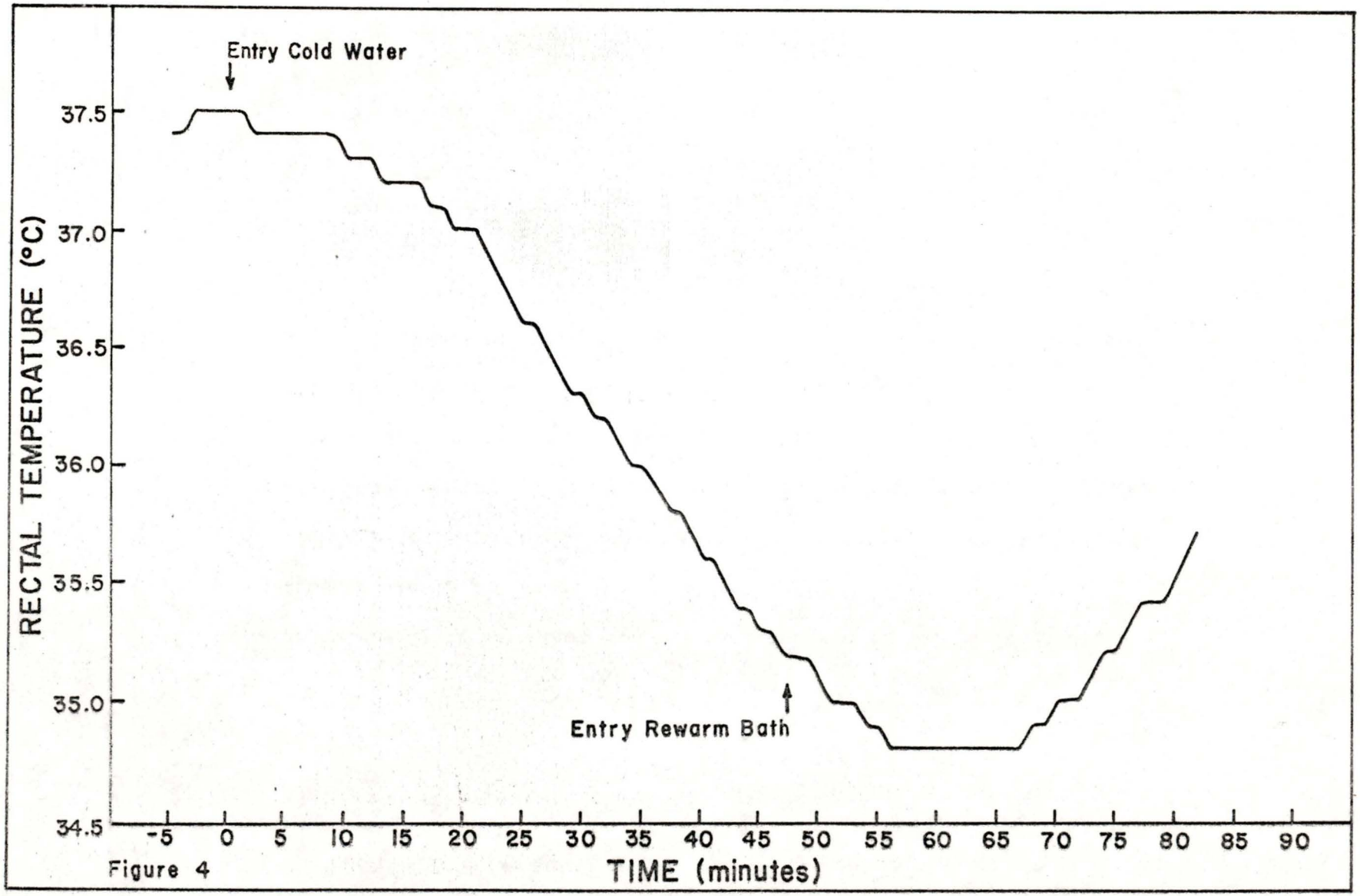

Entry Cold Water
Entry Rewarm Bath
RECTAL TEMPERATURE (°C)
37.5
37.0
36.5
36.0
35.5
35.0
34.5
-5 0 5 10 15 20 25 30 35 40 45 50 55 60 65 70 75 80 85 90
TIME (minutes)

Figure 4

Fig. 5. Exemplary Heart Rate Curve in One Male Subject (P.W., 26 Years Old) Immersed in 10.5°C Water While Holding Still.

Shown are three expanded segments of the continuous heart rate record: (A) Upon immersion heart rate reached 130 bpm and fluctuated considerably as subject became oriented in the water. (B) During cold immersion the heart rate showed 3 to 5 bpm fluctuations approximately 8 times per minute. These continued during the early rewarming period (C) but were often absent in the later rewarming period after cooling ceased.

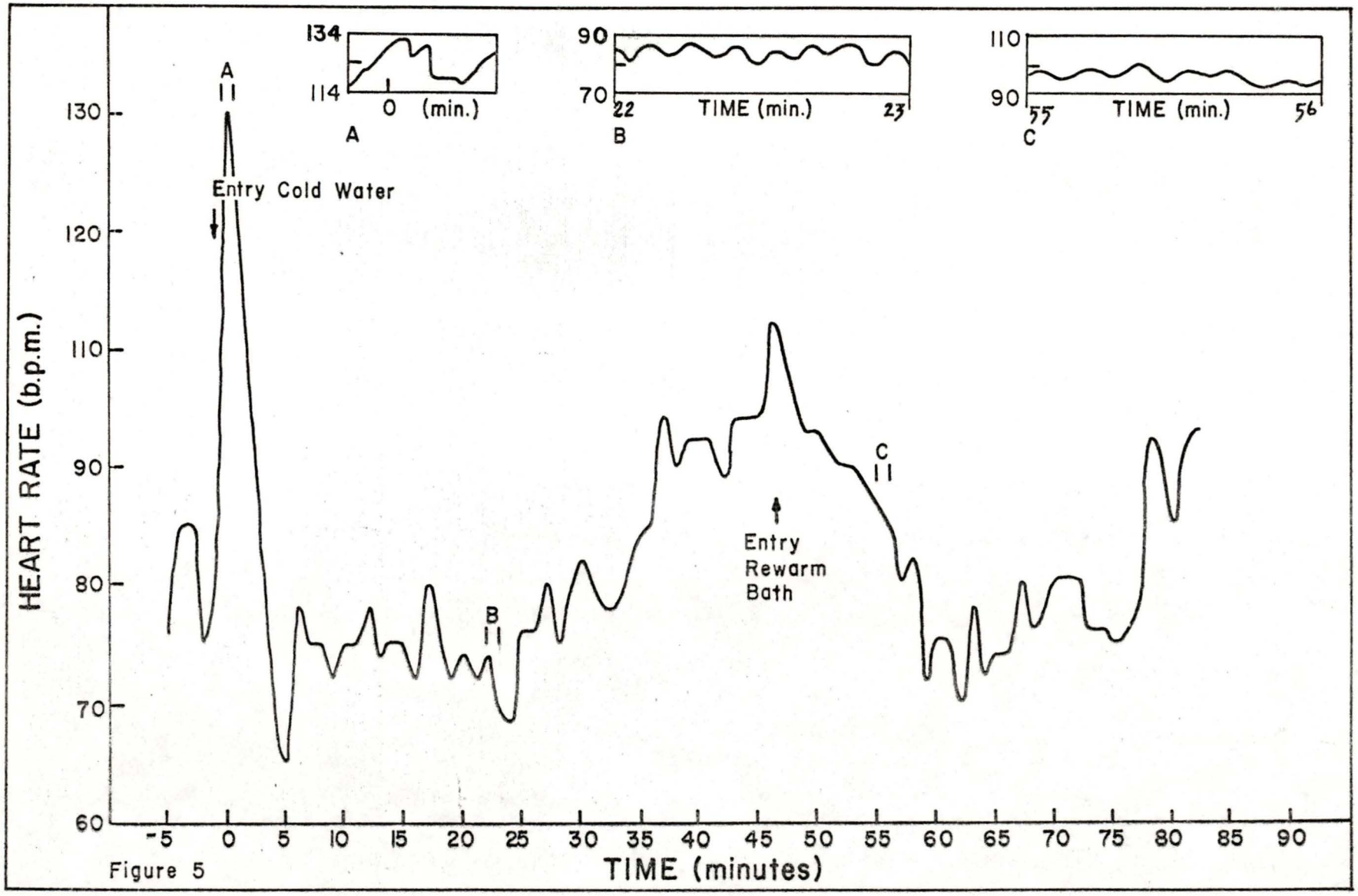

134
114
0 (min.)
A
90
70
22
TIME (min.)
23
B
110
90
55
TIME (min.)
56
C
A
Entry Cold Water
B
Entry
Rewarm
Bath
C
HEART RATE (b.p.m.)
130
120
110
90
80
70
60
-5
0
5
10
15
20
25
30
35
40
45
50
55
60
65
70
75
80
85
90
TIME (minutes)

Figure 5

6 minutes, has declined 1.0°C to 36.6°C by 25 minutes and to 35.6°C by the end of cold immersion.

Emersion from the cold water was accompanied by muscular activity and a rise in heart rate to 124 bpm was seen. This tachycardia subsided at 47 minutes when the subject was positioned in the rewarming bath (T_{H_2O}=21°C) and heart rate declined to 90 bpm at 50 minutes. An increase to 100 bpm occurred as the subject shivered violently and painfully from 51 to 53 minutes. Then it decreased steadily to 72 bpm at 61 minutes as shivering subsided and the subject felt comfortable. During the period after cold immersion to 59 minutes the rectal temperature continued to decline to 35.0°C, stabilized for 8 minutes and then steadily rose to 36.2°C at 86 minutes. The heart rate remained quite stable at 72 bpm for between 61 and 72 minutes and then increased steadily to 90 bpm at 86 minutes, this increase similar to but a few minutes behind the increase in rectal temperature. At the termination of Phase V (the rewarming period) the bath temperature was 40°C having been increased gradually from 21°C.

Comparing the two subjects shows the overall pattern to be similar. Subject P.B. had a decline in rectal temperature of 2.0°C from 37.6 to 35.6°C over 45 minutes with a low value at 35.0°C in the rewarming period. Subject P.W. declined 2.1°C in temperature from 37.4 to 35.3°C over 45 minutes and he had a low value of 34.8°C. His heart rate pattern is similar, showing 5 to 10 bpm variations every few minutes. The values were about 10 bpm lower than those of P.B. both in the cold and in the warm water, but he had been immersed once previously and seemed more relaxed. Sexual differences in response are studied later.

Mean Heart Rate Response

The mean heart rate response of 12 subjects (6 of each sex) immersed at 10.5°C in Series 2 is studied here and Figures 6 and 7 are plots of heart rate and rectal temperature. Values during cold immersion are taken only to 50 minutes which was the mean duration of this period. For continuity the intervals between pre-immersion and cold immersion and between cold immersion and rewarming are represented as 2 minutes which was the mean time of those periods. The arrow denotes entry into the rewarming bath.

In the resting period an 80 to 84 bpm heart rate was observed with a rectal temperature of 37.4°C. Upon immersion the heart rate peaked at 131 bpm, but it declined gradually, with intermittent fluctuations, to 79 bpm at 20 minutes. At that time the rectal temperature had declined 0.4°C to 37.0°C. Between 21 and 50 minutes the heart rate fluctuated but increased to 95 bpm coincident with an increase in shivering magnitude. The rectal temperature further decreased to 35.7°C by 50 minutes. Between 45 and 50 minutes there was an apparent decline in heart rate to 90 bpm which was an artifact attributable to a lower sample size in that interval.

Upon emersion from cold water the heart rate peaked at 110 bpm, declined to 83 bpm at 53 minutes and rose briefly to 85 bpm as an increase in shivering occurred. This brief shivering increase was widely variable among subjects from an intense painful shivering to a mild painless episode. Similarily, alterations in consciousness and orientation were noticed during rewarming, varying from none to feelings of euphoria, slurred speech and confusion.

Once shivering subsided the subjects felt more comfortable and regained

Fig. 6. Rectal Temperature of Subjects Immersed in 10.5°C Water While Holding Still.

The curve represents the mean of 12 subjects (6 males and 6 females). It shows a decline in rectal temperature of 1.7°C while in the cold water for 50 minutes and a subsequent decline of 1.2°C once out of the cold water and into the rewarming bath.

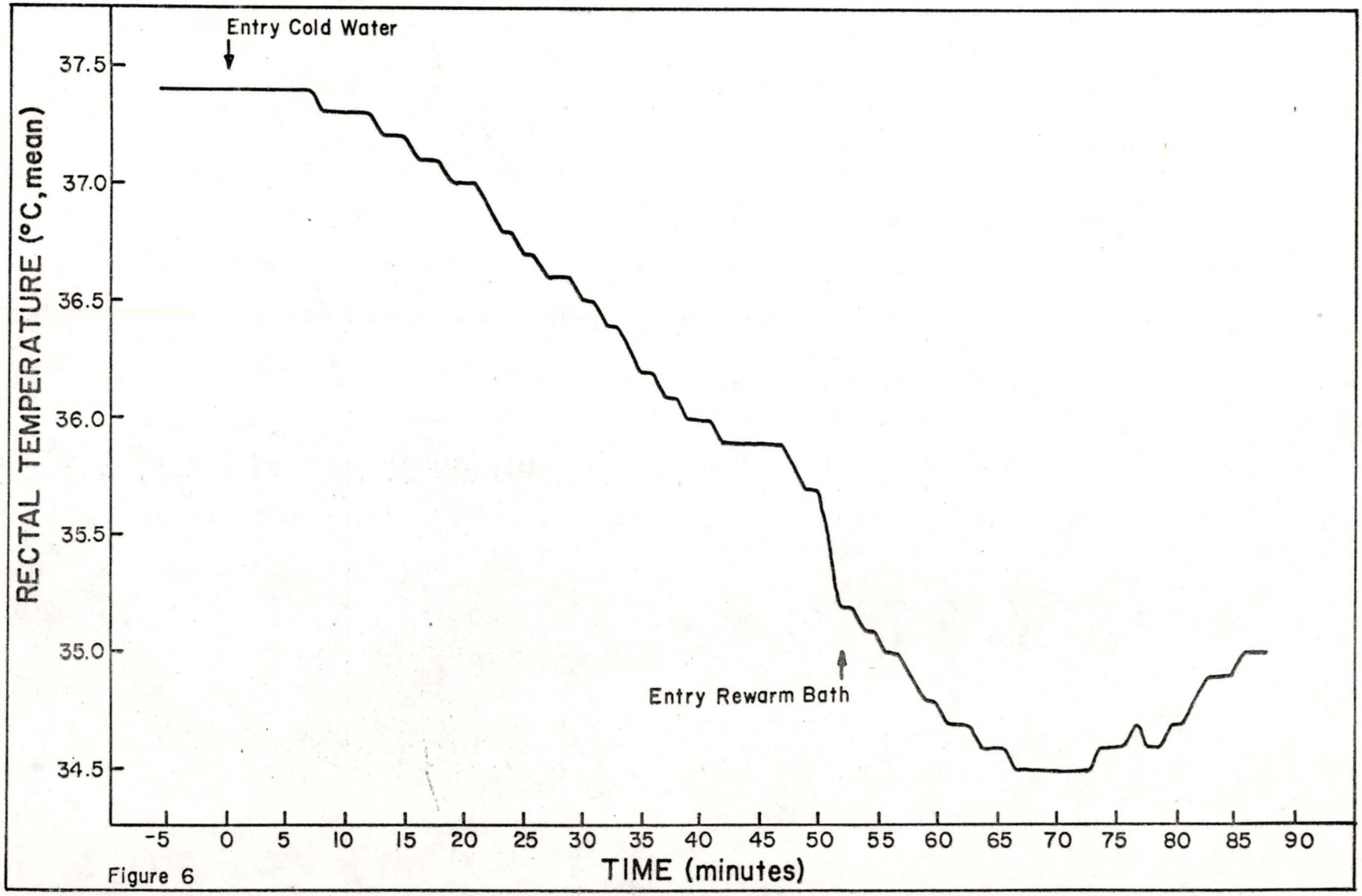

Entry Cold Water
Entry Rewarm Bath
RECTAL TEMPERATURE (°C, mean)
37.5
37.0
36.5
36.0
35.5
35.0
34.5
-5 0 5 10 15 20 25 30 35 40 45 50 55 60 65 70 75 80 85 90
TIME (minutes)

Figure 6

Fig. 7. Heart Rate of Subjects Immersed in 10.5°C Water While Holding Still. The curve represents the mean of 12 subjects (6 males and 6 females). It shows a tachycardia upon immersion then a decline in rate until 21 minutes and an increase in rate (coincident with shivering) until emersion from the cold water. In the rewarm bath the heart rate declined to a low value of 75 bpm interrupted at 6 minutes with a plateau coincident with a burst of shivering. It increased near the end of the rewarm period.

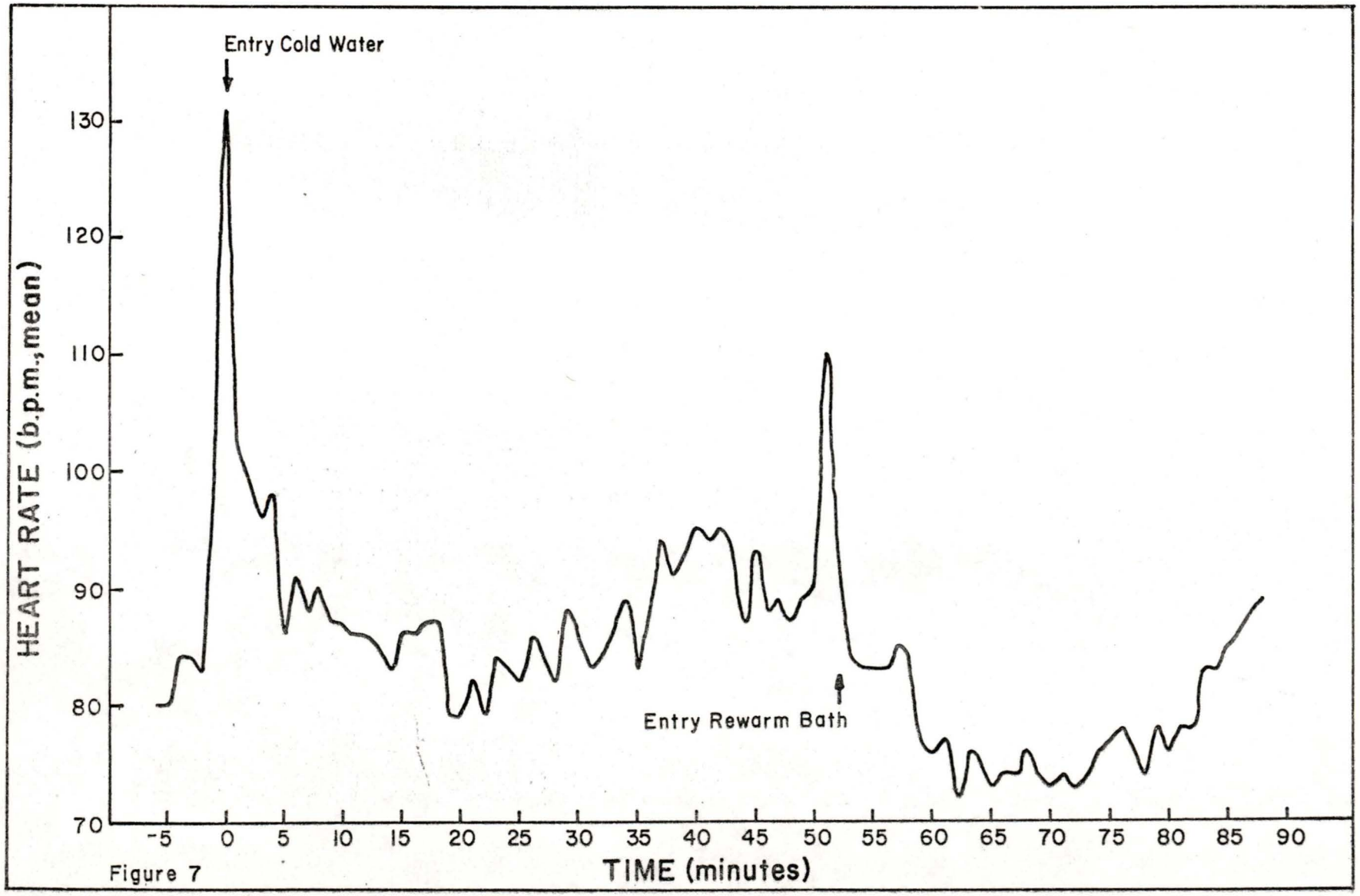

Entry Cold Water
Entry Rewarm Bath
HEART RATE (b.p.m., mean)
130
120
110
100
90
80
70
-5
0
5
10
15
20
25
30
35
40
45
50
55
60
65
70
75
80
85
90
TIME (minutes)

Figure 7

orientation. The heart rate declined to a relatively stable 74 bpm level by 62 minutes. During this plateau the second to second variations in heart rate described earlier seemed to be at a minimum or absent. Coinciding with this period (approximately 10 minutes) the rectal temperature, which had continued to decline a further 1.2°C during rewarming to 34.5°C, was at its lowest level. At 73 minutes both the rectal temperature and the heart rate increased steadily to 35.0°C and 89 bpm respectively at 88 minutes. Subjects who rewarmed without complication to within 1.5°C of their pre-immersion rectal temperature were removed from the bath feeling envigorated. If they remained in the bath longer they felt uncomfortably warm and somnolent.

In general, the pattern for heart rate and rectal temperature using the mean values closely follows the pattern described previously for the individual subjects.

Effect of Different Ambient Temperatures

In order to study the treatment effects of different cold water temperatures groups of 12 subjects were immersed in water temperatures of 4.6°C (Series 1), 10.5°C (Series 2) and 18.2°C (Series 3). The mean responses are shown in Figures 8 and 9 and Table 5 compares the results using the student t test ($p < 0.05$ unless otherwise noted).

At rest the heart rates were not significantly different ($p < 0.05$), but higher heart rates corresponded to the colder water temperature anticipated. They were similarly ordered upon immersion, Series 1 (4.6°C) achieving a maximum of 139 bpm. There were no significant differences.

Throughout the cold immersion which was 31 minutes (4.6°C), 50 minutes (10.5°C) and 66 minutes (18.2°C), the heart rate curves remained in the same order. There was a gradual decline over the first 20 minutes

Fig. 8. Comparison of Rectal Temperature Response to Immersion at Three Water Temperatures.

Each curve is the mean of 11 or 12 subjects (half of each sex) who were holding still in ocean water of the temperatures shown. The curves are lines of best fit through variates one minute apart. Significant differences occurred between the 18.2°C curve, the two others at 30 minutes ($p < 0.05$) and the 10.5°C curve at 50 minutes ($p < 0.01$). During rewarming the 18.2°C curve again was significantly different ($p < 0.05$) from both other curves at 6 minutes, the 10.5°C curve at 20 minutes and the 4.6°C curve at 30 minutes.

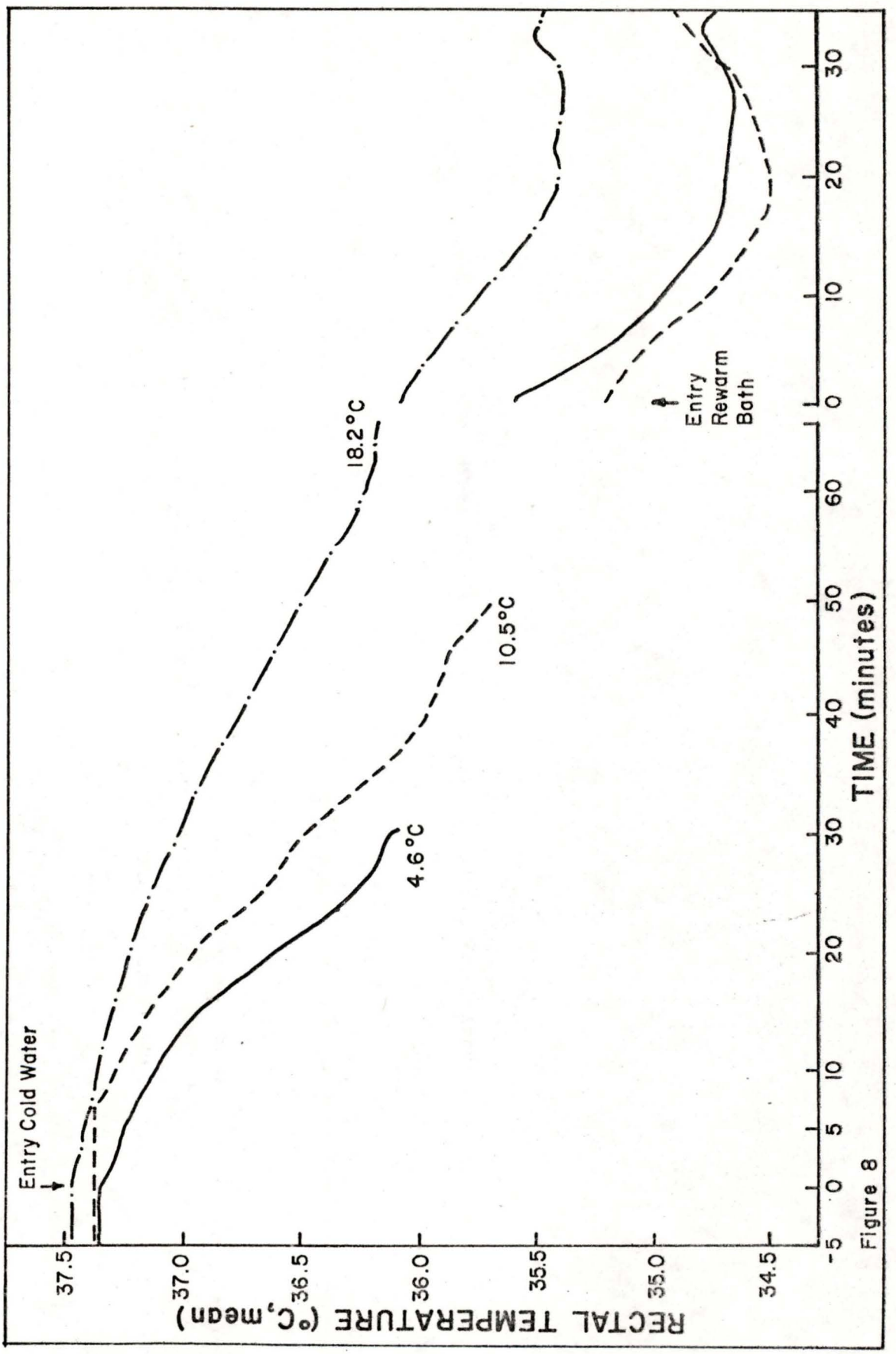

RECTAL TEMPERATURE (°C, mean)
37.5
37.0
36.5
36.0
35.5
35.0
34.5
-5
0
5
10
20
30
40
50
60
0
10
20
30
TIME (minutes)
Entry Cold Water
18.2°C
10.5°C
4.6°C
Entry
Rewarm
Bath

Figure 8

Fig. 9. Comparison of Heart Rate Response to Immersion at Three Water Temperatures.

The curves were plotted as for Fig. 8. Significant differences occurred between the 4.6°C curve and both other curves after 10 minutes of cold immersion ($p < 0.05$) and between all curves after 6 minutes of the rewarming period ($p < 0.01$).

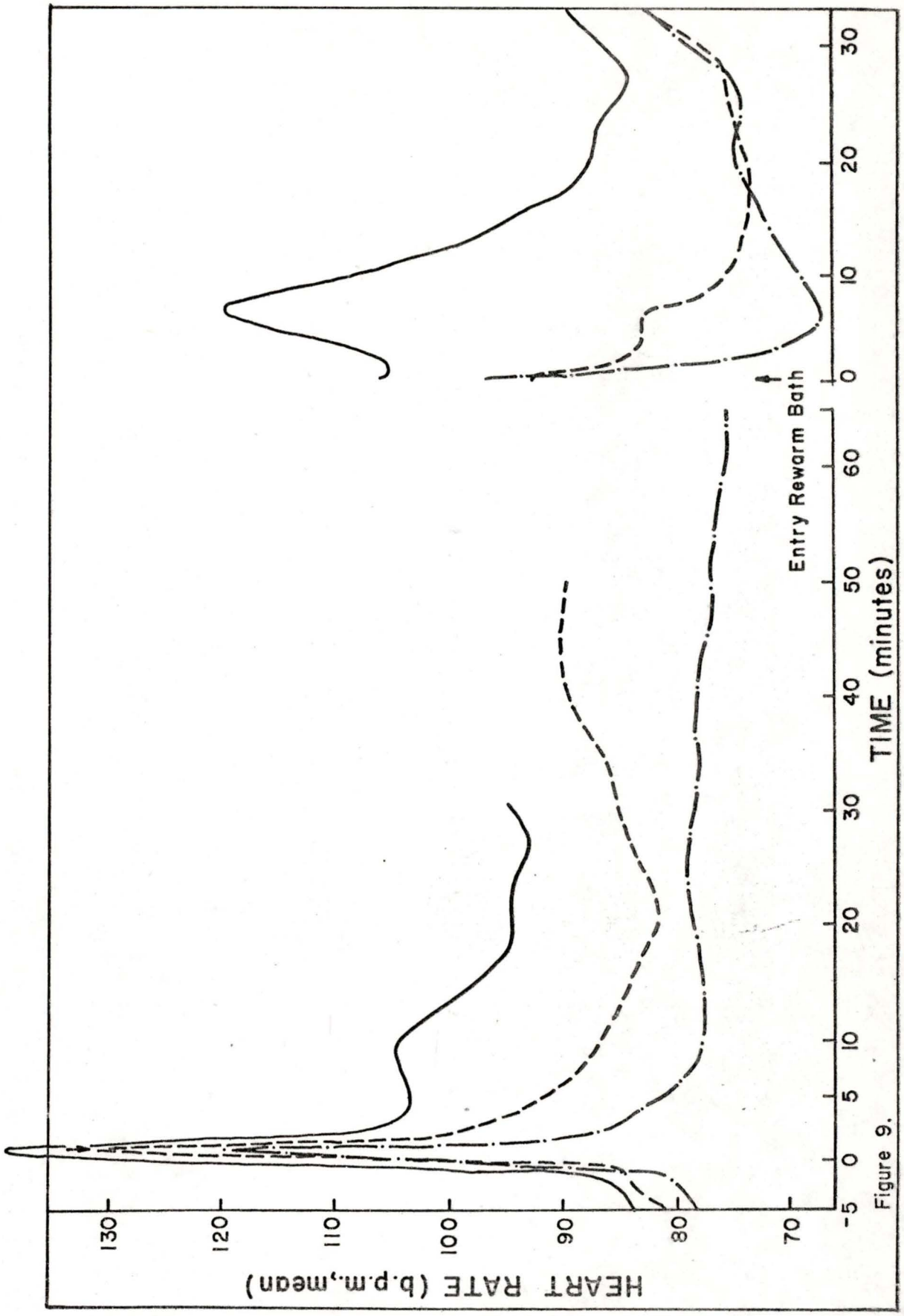

HEART RATE (b.p.m.,mean)
130
120
110
100
90
80
70
-5
0
5
10
20
30
40
50
60
TIME (minutes)
Entry Rewarm Bath
0
10
20
30

Figure 9.

TABLE 5. RECTAL TEMPERATURE AND HEART RATE AT THREE WATER TEMPERATURES

Parameter	Ser./T_A (°C[1])	Time In Cold Water (minutes)					Rewarming (minutes)		
		Control	0	10	30	50	6	20	30
Rectal Temp. (°C)[1]	1/4.6	37.4±0.3	37.4±0.3	37.2±0.3	36.2±0.5*	----	35.2±0.7*	34.7±1.0	34.8±1.0*
	2/10.5	37.4±0.2	37.4±0.2	37.3±0.2	36.5±0.4*	35.7±0.4**	35.0±0.4**	34.5±0.5*	34.7±0.6
	3/18.2	37.5±0.3	37.4±0.3	37.4±0.3	37.1±0.3	36.5±0.5	35.9±0.8	35.4±0.7	35.4±0.4
Heart Rate (bpm)[1]	1/4.6	85±16	139±11	108±18[+,*]	95±19	----	118±18[++,**]	88±17	87±13
	2/10.5	84±12	132±7	87±20	87±23	90±35	85±20*	74±14	78±14
	3/18.2	80±10	120±14	76±14	78±14	75±18	66±12	74±15	80±14

\+ $p < 0.05$ difference from Series 2

++ $p < 0.01$ difference from Series 2

* $p < 0.05$ difference from Series 3

** $p < 0.01$ difference from Series 3

1 mean ± S.D.

interrupted only in Series 1 by a slight increase from 104 to 106 bpm between 5 and 10 minutes. At 10 minutes this heart rate was significantly different from that of Series 2 (87 bpm) and from that of Series 3 (76 bpm). Figure 10 shows that by 30 minutes the heart rate in Series 1 (4.6°C) had risen from a low of 93 to 95 bpm. In Series 2 (10.5°C) it was 87 bpm and in Series 3 (18.2°C) the rate had declined below pre-immersion level to 78 bpm. None of these differences were significant although subjectively there was more shivering, anxiety and discomfort in subjects immersed at 4.6°C than those at 10.5°C or 18.2°C. At 50 minutes the rates were 90 bpm (Series 2) and 75 bpm (Series 3) which were not significantly different.

At the start of the rewarm period all 3 curves had elevated heart rates between 108 and 93 bpm. By 6 minutes of rewarming the Series 1 (4.6°C) curve showed a rapid increase in rate to 120 bpm coincident with a marked increase in shivering. In Series 2 (10.5°C) the rate had declined to 85 bpm but there was a plateau at 6 minutes during a less marked shivering period. The shivering and discomfort was much greater in Series 1 subjects than in Series 2 subjects even though the rectal temperature was greater (35.2°C) in Series 1. The relative ambient temperature change was greater, however, in Series 1 (20°C - 4.6°C = 15.4°C) than in Series 2 (20°C - 10.5°C = 9.5°C). In Series 3 the heart rate at 6 minutes was 66 bpm and there was no evidence of shivering. Significant differences in heart rate were found at 6 minutes between Series 1 and 2 ($p < 0.01$), 1 and 3 ($p < 0.01$) and 2 and 3 ($p < 0.05$).

After 6 minutes heart rate declined progressively in both Series 1 and 2. Series 2 reached 74 bpm at 20 minutes and Series 1 reached a minimum of 82 bpm at 25 minutes. Following the minimum rate levels there were increases to 87, 78 and 80 bpm in Series 1 to 3 respectively at 30

Fig. 10. Heart Rate After 30 Minutes in Cold Water at Three Ambient Temperatures.

The points represent the mean heart rates of 12 subjects (11 at 4.6°C) and the standard deviation of the mean is shown above and below each value.

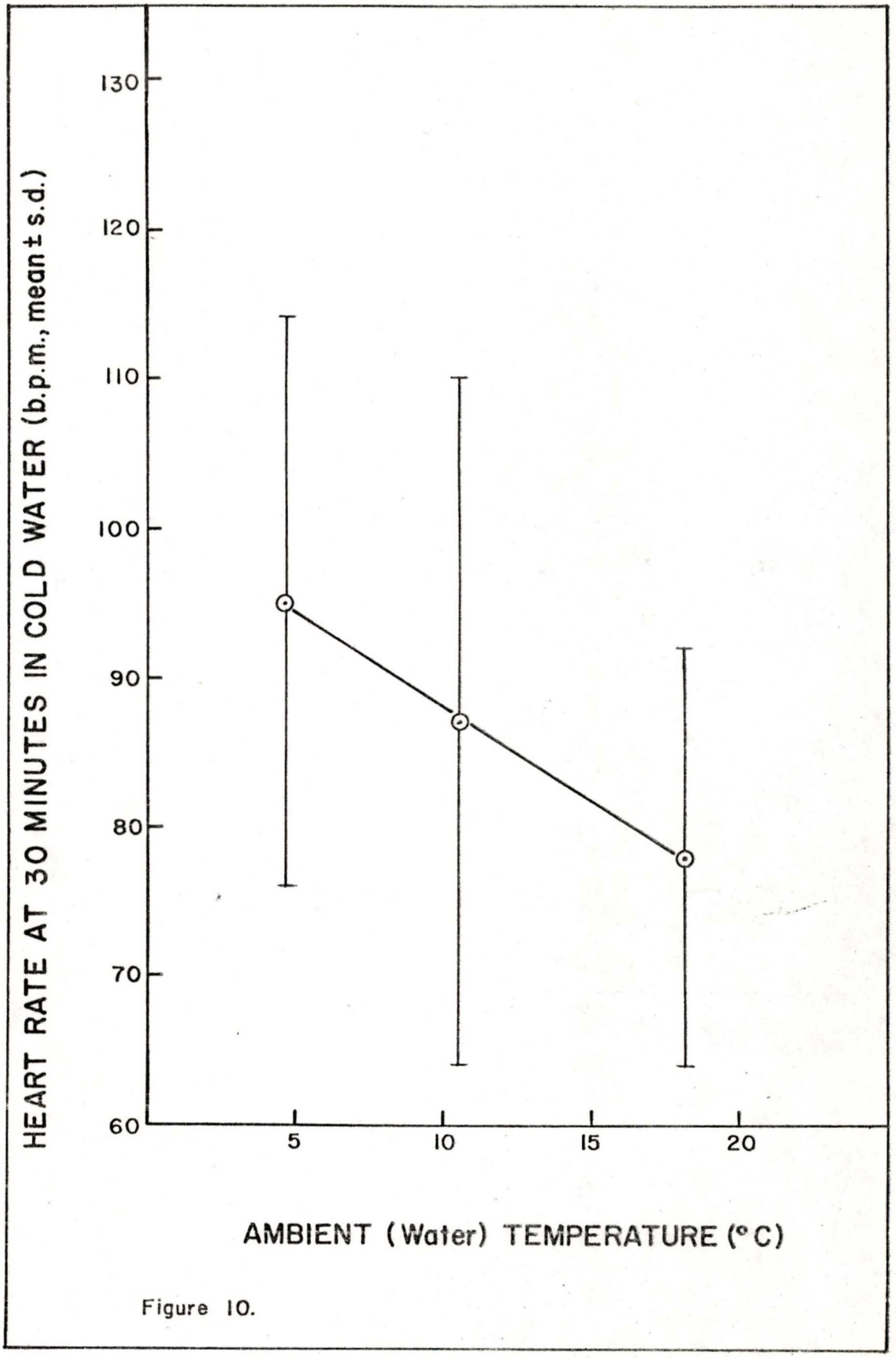

HEART RATE AT 30 MINUTES IN COLD WATER (b.p.m., mean ± s.d.)
130
120
110
100
90
80
70
60
5
10
15
20
AMBIENT (Water) TEMPERATURE (° C)

Figure 10.

minutes and none of these was significantly different.

The rectal temperature during rewarming followed the previously described pattern with low levels being reached at 20 minutes. The curve for Series 2 (10.5°C) which declined less rapidly than that for Series 1 (4.6°C) during the cold immersion was lower in the rewarming period because subjects stayed in longer and therefore cooled more. The values at 20 minutes were 34.7, 34.5 and 35.4°C for Series 1 to 3 respectively. Only Series 2 (10.5°C) and 3 (18.2°C) were significantly different.

Effect of Muscular Activity (Swimming)

The effect of muscular activity (gentle swimming at approximately 30 strokes per minute) was studied in Series 4 which included 12 subjects immersed in 10.5°C water. Comparison was made with Series 2 at the same temperature while subjects were "still."

Figures 11 and 12 are the mean rectal temperature and heart rate curves for this comparison and Table 6 includes comparison statistics. At rest and upon immersion there were no significant differences between the heart rates. There was a decline in both curves over the first 10 minutes, Series 2 declining from 132 to 87 bpm and Series 4 from 134 to 122 bpm, and as a result the curves became significantly separated ($p < 0.01$). After 20 minutes the centrally driven increase in metabolic rate is shown by an increase in heart rate in Series 2 such that at 40 minutes the curves are only different at the $p < 0.05$ level (Series 2: 95 bpm; Series 4: 120 bpm).

The heart rate curves during rewarming were of similar shape but the Series 4 (Active) curve was consistently above the Series 2 (Still) curve. At no time were they significantly different. Upon immersion in the rewarm bath the heart rate was 105 bpm in Series 4 and 96 bpm in

Fig. 11. Comparison of Rectal Temperature Response of Subjects Holding Still and Swimming in 10.5°C Water.

Each curve is the line of best fit through mean minute variates of 12 subjects (6 males and 6 females). See Methods for description of activities performed. Significant differences occurred at 10 minutes ($p < 0.01$) and at 40 minutes ($p < 0.05$) only.

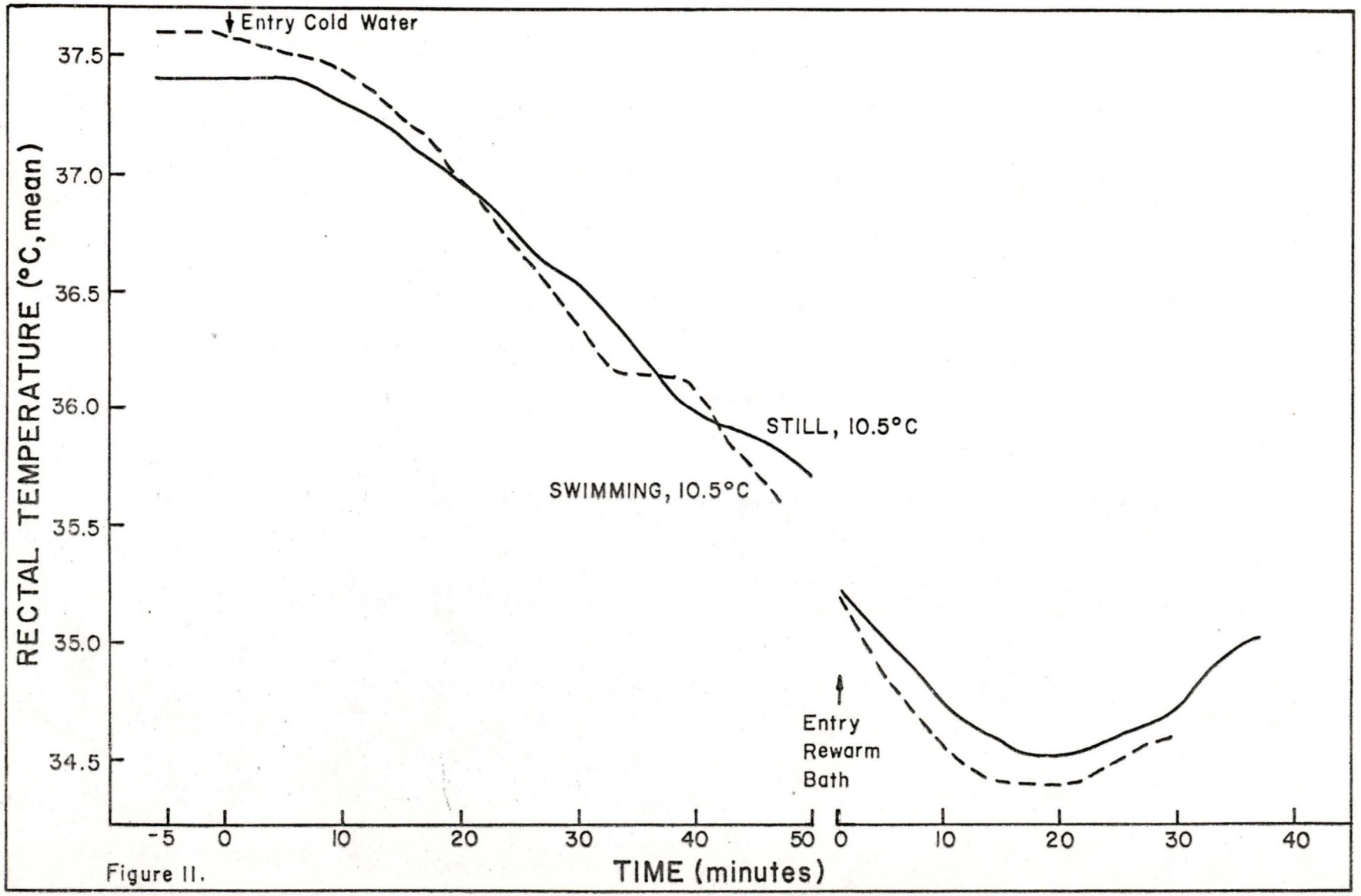

Entry Cold Water
37.5
37.0
36.5
36.0
35.5
35.0
34.5
RECTAL TEMPERATURE (°C, mean)
STILL, 10.5°C
SWIMMING, 10.5°C
Entry
Rewarm
Bath
-5
0
10
20
30
40
50
0
10
20
30
40
TIME (minutes)

Figure 11.

Fig. 12. Comparison of Heart Rate Response of Subjects Holding Still and Swimming in 10.5°C Water.

The curves were plotted as for Figure 11. They were significantly different from 2 minutes to the end of cold immersion ($p < 0.05$) but not during the rewarming period.

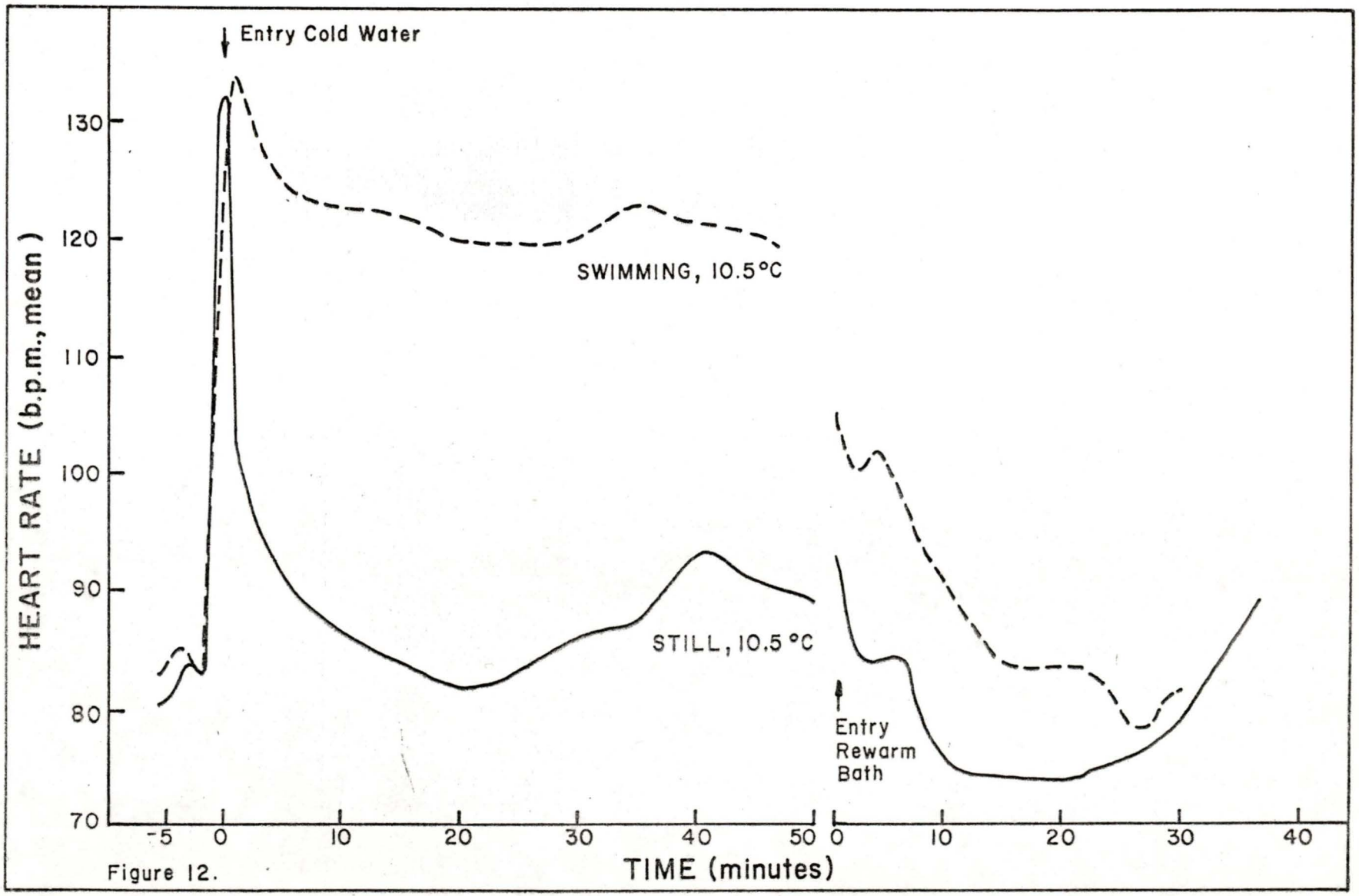

Entry Cold Water
SWIMMING, 10.5°C
STILL, 10.5 °C
Entry
Rewarm
Bath
HEART RATE (b.p.m., mean)
130
120
110
100
90
80
70
-5
0
10
20
30
40
50
0
10
20
30
40
TIME (minutes)

Figure 12.

TABLE 6. RECTAL TEMPERATURE AND HEART RATE WHILE STILL AND SWIMMING AT 10.5 C

Parameter	Series	Activity	Time In Cold Water (minutes)				Rewarming (minutes)		
			Control	9	10	47	4	20	30
Rectal Temp. °C[1]	2	Still	37.4±0.2	37.4±0.2	37.3±0.2	35.9±0.3	35.1±0.4	34.5±0.5	34.7±0.6
	4	Swim	37.6±0.4	37.5±0.3	37.4±0.4	35.6±0.3	34.9±0.4	34.4±0.2	34.6±0.5
Heart Rate bpm[1]	2	Still	84±12	132±7	87±20	95±9	83±12	74±14	78±14
	4	Swim	86±16	118±33	122±15**	120±11*	101±12	83±9	81±10

* $p < 0.05$ from Series 2

** $p < 0.01$ from Series 2

1 mean ± S.D.

Series 2. Then, following a slight decrease, a rise coincident with the usual shivering period occurred in both series at 5 - 7 minutes. Both curves then decreased gradually to a minimum (78 bpm in Series 4; 74 bpm in Series 2). After 21 minutes of rewarming the heart rate curve began to rise in Series 2 and it became almost coincident with the Series 4 curve which began to rise at 28 minutes.

The rectal temperature declined at a greater (but not significantly different) rate when subjects were swimming (dropping 2.0°C in 47 minutes) than when they were still (dropping 1.5°C in 47 minutes) showing that heat loss due to increased periferal blood flow exceeded thermogenesis in the swimming subjects.

The subjects in Series 2 remained in the water an average of 2 minutes longer and thus rectal temperature in the two series was equal (35.2°C) at the beginning of the rewarming period. The two curves showed an almost parallel decline, plateau and then increase during the rewarming period, with Series 4 reaching a minimum of 34.4°C, 0.1°C less than Series 2. The curves were not significantly different.

Relationship Between Heart Rate and Oxygen Consumption While Still and Swimming

It can be inferred from results above that the heart rate response to cold water immersion is influenced strongly by metabolic rate. To illustrate this observation heart rate and oxygen consumption (as the index of metabolic rate) were correlated in Series 2 (Still, at 10.5°C) for periods 1 - 20 minutes, 21 - 50 minutes and during the first 15 minutes of rewarming; and in Series 4 (Swimming at 10.5°C) for periods 15 - 50 minutes and during 1 - 15 minutes of rewarming. See Figures 13 and 14 and Table 7.

Fig. 13. Heart Rate and Oxygen Consumption of Subjects While Holding Still and While Swimming in 10.5°C Water.

The heart rate curve is a duplicate of Figure 12. The oxygen consumption curve also represents a line of best fit through mean minute variates of the same subjects. Similarities in the curves after 21 minutes in the cold water and in the rewarm period illustrates the correlation of the two parameters (Figure 14, Table 7).

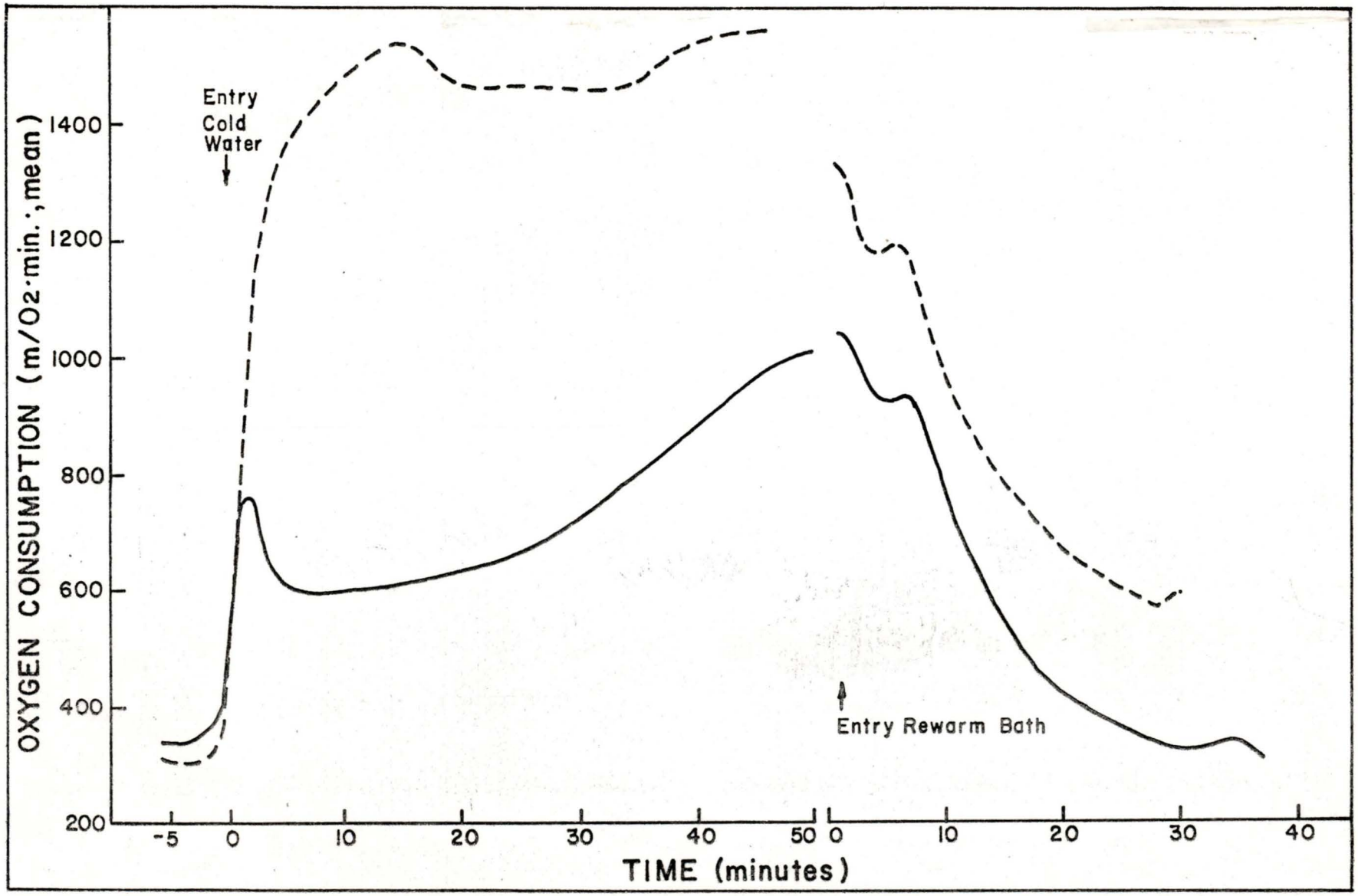

Entry
Cold
Water
Entry Rewarm Bath
OXYGEN CONSUMPTION (m/O2·min.·, mean)
1400
1200
1000
800
600
400
200
-5
0
10
20
30
40
50
0
10
20
30
40
TIME (minutes)

Figure 13.

Fig.13. (continued)

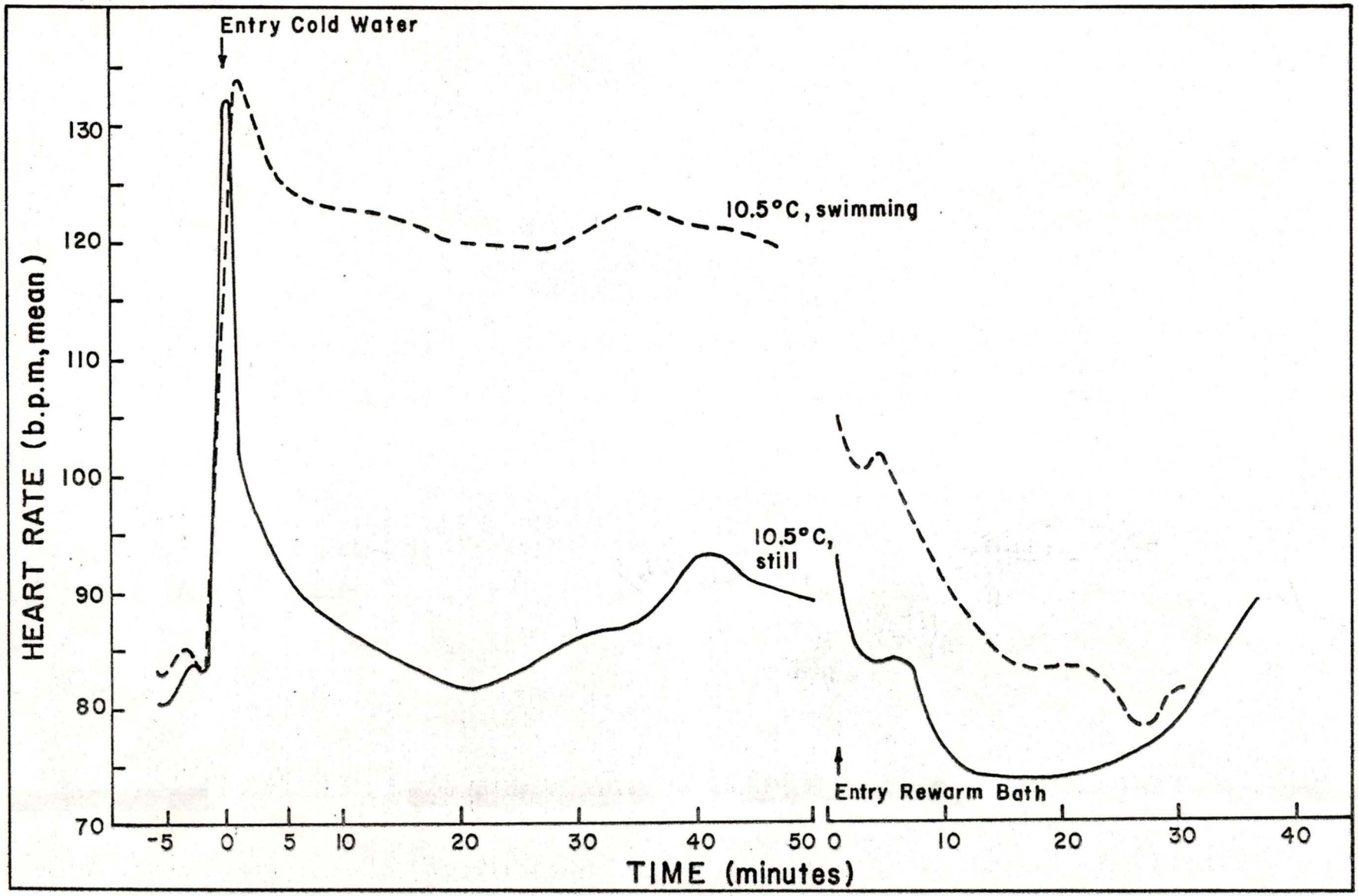

Entry Cold Water
10.5°C, swimming
10.5°C, still
Entry Rewarm Bath
HEART RATE (b.p.m., mean)
TIME (minutes)
130
120
110
100
90
80
70
-5
0
5
10
20
30
40
50
0
10
20
30
40

FIGURE 13

Fig. 14. Correlation of Heart Rate and Oxygen Consumption of Subjects While Holding Still and While Swimming in 10.5°C Water.

Pearson product-moment correlation coefficients were significant in still subjects between 1 and 20 minutes ($p < 0.05$), 21 and 50 minutes ($p < 0.01$) in cold water and between 1 and 15 minutes in the rewarm bath ($p < 0.01$). In swimming subjects the correlation was significant ($p < 0.01$) only in the rewarm bath (Table 7).

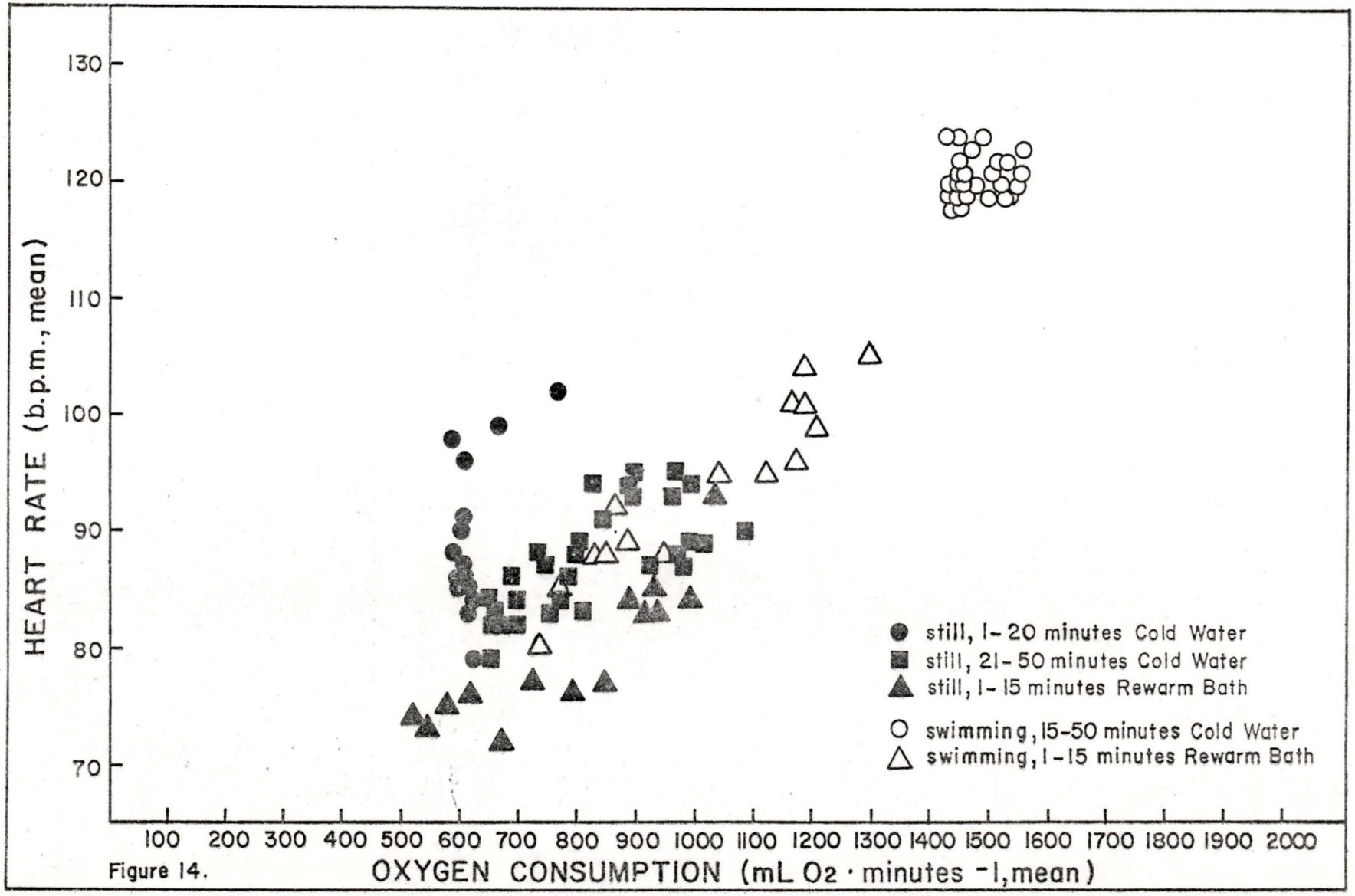

HEART RATE (b.p.m., mean)
130
120
110
100
90
80
70
100 200 300 400 500 600 700 800 900 1000 1100 1200 1300 1400 1500 1600 1700 1800 1900 2000
OXYGEN CONSUMPTION (mL O2 · minutes -1, mean)
still, 1-20 minutes Cold Water
still, 21-50 minutes Cold Water
still, 1-15 minutes Rewarm Bath
swimming, 15-50 minutes Cold Water
swimming, 1-15 minutes Rewarm Bath

Figure 14.

TABLE 7. CORRELATION OF HEART RATE AND OXYGEN CONSUMPTION WHILE STILL AND SWIMMING AT 10.5°C AND DURING REWARMING

Series	Activity	Period for Correl.	Minutes	r	Significance
2	Still	Cold	1 - 20	-0.449	$p < 0.05$
	Still	Cold	21 - 50	+0.714	$p < 0.01$
	Still	Rewarm	1 - 15	+0.880	$p < 0.01$
4	Swim	Cold	15 - 47	+0.235	NS
	Swim	Rewarm	1 - 15	+0.939	$p < 0.01$

In the first 20 minutes of Series 2 the metabolic rate increased rapidly, dropped to about 600 ml O_2/min. at 4 minutes and then climbed slightly to 629 at 20 minutes. This occurred while the heart rate dropped from 102 to 79 bpm. A Pearson product-moment correlation had a barely significant (at $p < 0.05$) coefficient "r" of - 0.449. This shows that as heart rate is declining metabolic rate is rising proportionately over the interval. From 21 - 50 minutes the correlation was indeed positive ($r = + .714$, significant at $p < 0.01$). During this time heart rate rose from 82 to 90 bpm and oxygen consumption from 654 to 1081 ml/min. In the first 15 minutes of the rewarming period the heart rate dropped from 93 to 74 bpm while the oxygen consumption dropped from 1030 to 520 ml/min. This was again significantly correlated ($r = + 0.880, p < 0.01$).

While actively swimming (Series 4), the heart rate and metabolic rate remained elevated and fairly constant after immersion in the water although heart rate dropped about 10 bpm in the first few minutes (Fig. 13). The correlation from 15 to 47 minutes, however, was not significant. During the rewarming period the two parameters were again significantly correlated ($r = + 0.939, p < 0.01$). Figure 14 plots the correlations for the five time periods described above.

Thermal Protection and Its Effect on Heart Rate

One objective of the Man Overboard Project was to evaluate the thermal protection of different buoyancy devices and to develop an optimum model. Accordingly, 5 subjects were immersed in ocean water with a mean temperature of 8.8°C while wearing standard clothing plus one of three buoyancy devices. A Ministry of Transport approved kapok life vest, a floater coat, and a modified floater coat with beaver tail ("UVIC SPECIAL") were worn on separate occasions. Their effect on heart rate and rectal temperature

during cold immersion is reported here (Figures 15 and 16; Table 8).

The heart rate curves were similar to each other but different from those of previous series. If the Series 2 and 10(a) heart rate curves are compared (both of these groups wore the M.O.T. life vest), it is noted that the absolute heart rate levels were about 10 bpm lower in Series 10(a) even though the water was almost 2.0°C colder. Also, the heart rate did not show the pattern of gradual decline for 20 minutes followed by an increase concurrent with increasing metabolic rate. Instead, the heart rate declined to resting level within 3 minutes of immersion and varied only 4 bpm thereafter. The heart rate at 40 minutes was 71 bpm, only 1 bpm greater than pre-immersion. The rectal temperature (T_R) was lower after 40 minutes (35.4°C) than in Series 2 (35.9°C), yet the heart rate was lower (71 bpm) than in Series 2 (95 bpm).

With added thermal protection (Series 10(b) and (c)), the heart rate curves showed similar patterns to those of Series 10(a), but the absolute rates became greater as the amount of thermal protection was increased. For example, at 23 minutes the mean rectal temperature had dropped 1.0°C to 36.4°C in Series 10(c) and the heart rate was 88 bpm while in Series 10(a) rectal temperature had dropped 1.4°C to 35.6°C and the heart rate was only 71 bpm. There was no significant difference ($p < 0.05$) between any two curves.

Sex Differences in Heart Rate Response

Male and female subjects in Series 2 were considered separately to investigate sex-related differences in heart rate response to cold exposure (Figures 17 and 18; Table 9). As expected, the patterns of the heart rate curves were similar to those described previously for all subjects, but the female heart rates were consistently greater. The only significantly greater value is at 40 minutes where 104 bpm is compared to 84 bpm ($p < 0.05$).

The trend of lower male heart rates shown here may be confounded by

Fig. 15. Comparison of Rectal Temperature Response of Subjects Wearing One of Three Buoyancy-Thermal Protection Devices Holding Still in 8.8°C Water. The curves are lines of best fit through mean variates of 5 subjects. The only significant difference is between the kapok vest and UVIC Special at 23 minutes ($p < .05$).

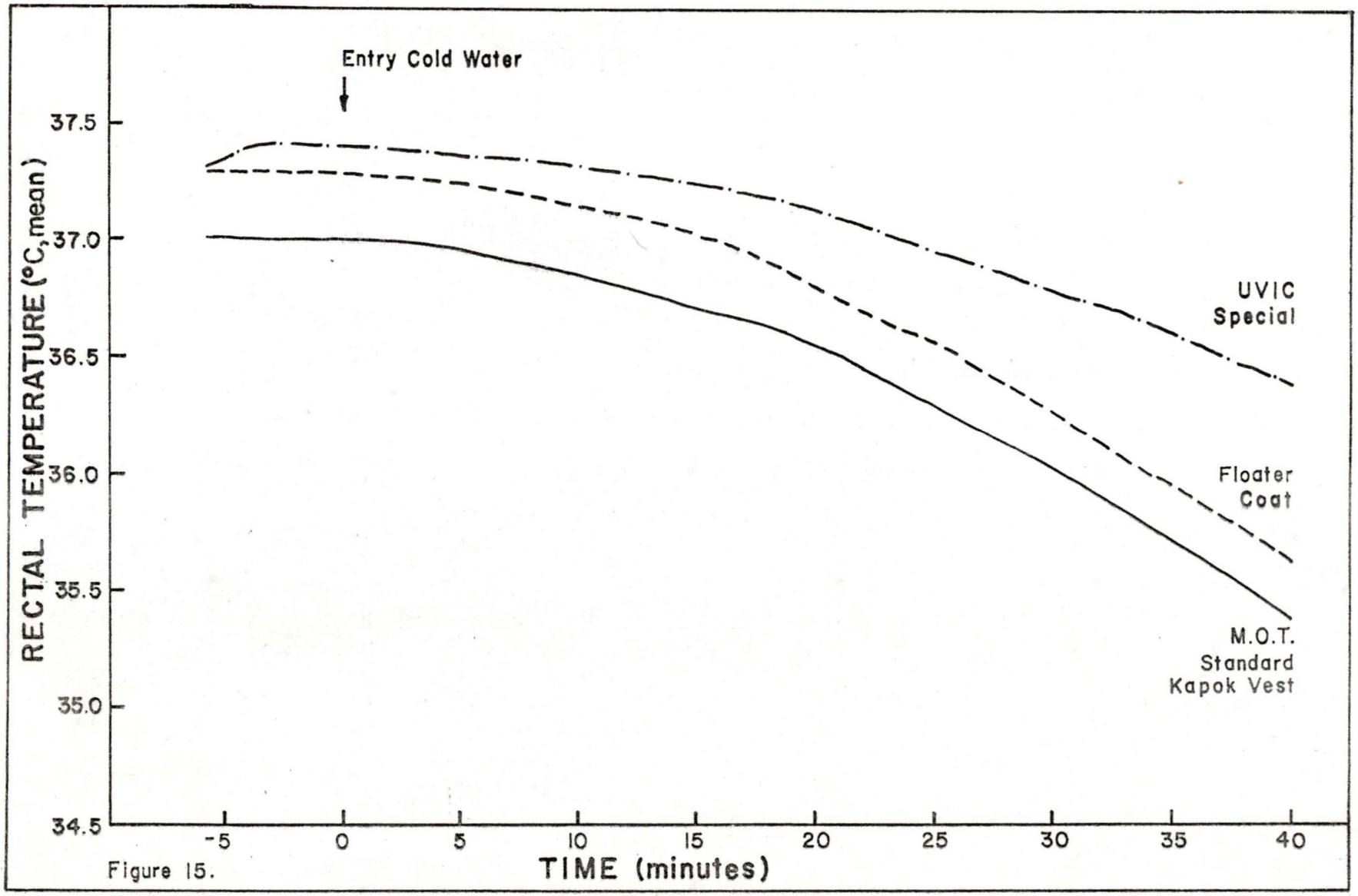

Entry Cold Water
RECTAL TEMPERATURE (°C, mean)
37.5
37.0
36.5
36.0
35.5
35.0
34.5
-5
0
5
10
15
20
25
30
35
40
TIME (minutes)
UVIC Special
Floater Coat
M.O.T. Standard Kapok Vest

Figure 15.

Fig. 16. Comparison of Heart Rate Response of Subjects Wearing One of Three Buoyancy-Thermal Protection Devices Holding Still in 8.8°C Water. The curves were plotted as in Figure 15. No significant differences were found.

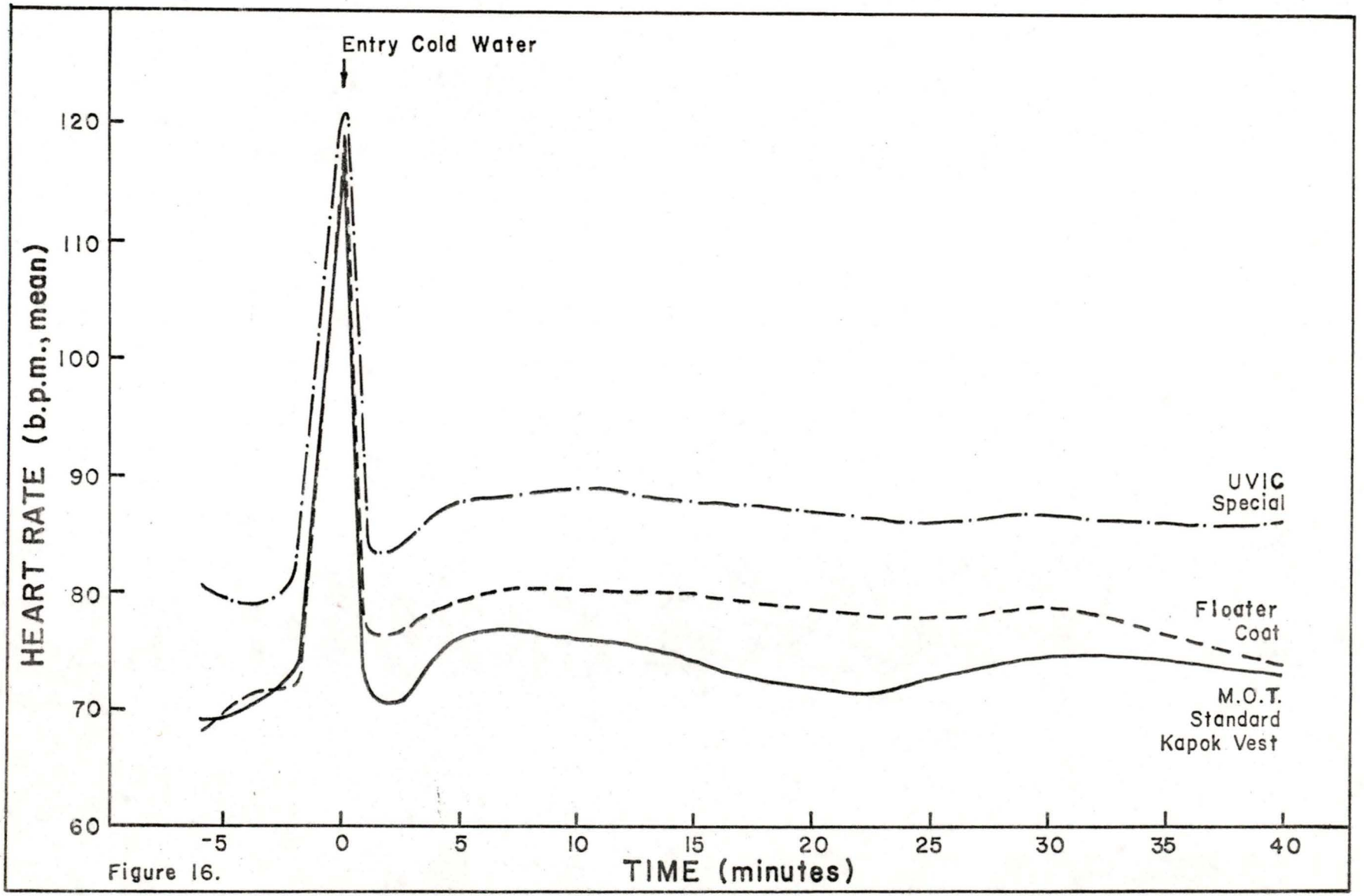

Entry Cold Water
HEART RATE (b.p.m., mean)
120
110
100
90
80
70
60
-5
0
5
10
15
20
25
30
35
40
TIME (minutes)
UVIC
Special
Floater
Coat
M.O.T.
Standard
Kapok Vest

Figure 16.

TABLE 8. RECTAL TEMPERATURE AND HEART RATE WITH VARIED THERMAL PROTECTION AT 8.8°C

Parameter	Attire[2] (Standard clothing +...)	Time in Cold Water (minutes)			
		Control	0	23	40
Rectal Temp. °C[1]	Kapok vest	37.0±0.4	37.0±0.4	36.4±0.3	35.6±0.2
	Floater Coat	37.3±0.3*	37.3±0.3	36.6±0.4	35.9±0.7
	UVIC SPECIAL	37.4±0.1	37.3±0.1	37.0±0.3*	36.4±0.6
Heart Rate[1]	Kapok Vest	70±11	118±10	69±16	71±9
	Floater Coat	72±15	117±18	76±20	76±13
	UVIC SPECIAL	78±18	121±16	85±14	88±10

1 Mean ± S.D.

2 See Table 4

* $p < 0.01$ difference from Kapok

Fig. 17. Comparison of Rectal Temperature Response of Male and Female Subjects Holding Still in 10.5°C Water.

Each curve is a line of best fit through mean variates of 6 subjects. The cooling rate of females (4°C per hour) was greater than that of males (1.6°C per hour) but no significant difference in rectal temperature was found.

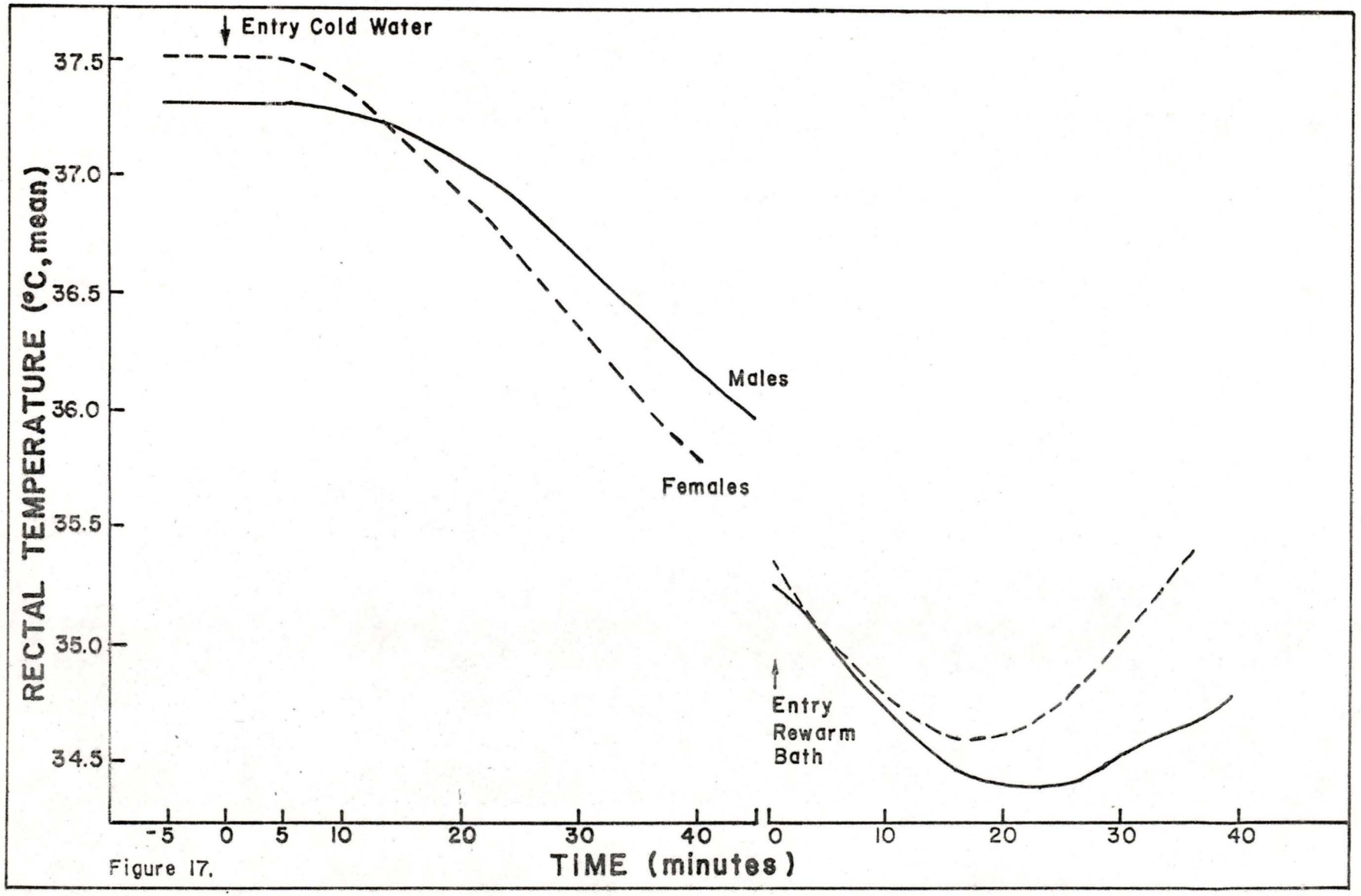

Entry Cold Water
37.5
37.0
36.5
36.0
35.5
35.0
34.5
RECTAL TEMPERATURE (°C, mean)
Males
Females
Entry
Rewarm
Bath
-5
0
5
10
20
30
40
0
10
20
30
40
TIME (minutes)

Figure 17.

Fig. 18. Comparison of Heart Rate Response of Male and Female Subjects Holding Still in 10.5°C Water.

Each curve is a line of best fit through mean variates of 6 subjects. The only significant difference in heart rate was at 10 minutes in the cold water ($p < 0.05$).

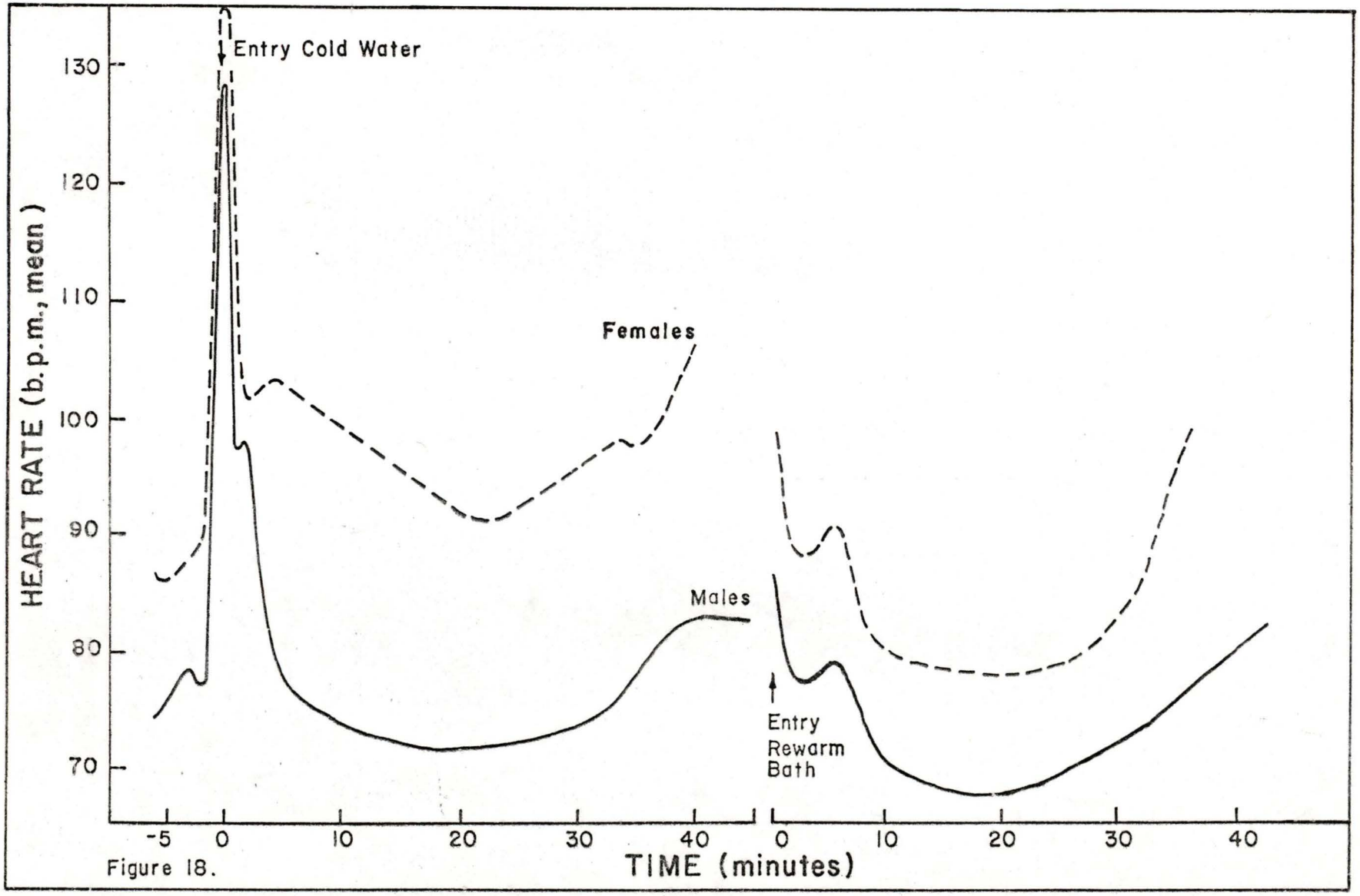

Entry Cold Water
Females
Males
Entry
Rewarm
Bath
HEART RATE (b.p.m., mean)
130
120
110
100
90
80
70
-5
0
10
20
30
40
0
10
20
30
40
TIME (minutes)

Figure 18.

TABLE 9. SEX DIFFERENCES EFFECT ON HEART RATE AND RECTAL TEMPERATURE AT 10.5°C

Parameter	Sex	Time In Cold Water (minutes)				Rewarming (minutes)		
		Control	0	10	40	6	20	35
Rectal Temp. °C [1]	Male	37.3	37.3	37.2	36.2	34.9	34.4	34.7
	Female	37.5	37.5	37.4	35.9	35.0	34.6	35.3
Heart Rate bpm [1]	Male	79	129	75	84	79	69	77
	Female	88	135	99*	108	91	79	97

* $\bar{p} < 0.05$ difference from males

1 Means of 6 subjects

the inclusion of four male researchers who had frequently been immersed. The mean age of the males was 31.5 ± 3.0 years compared to 22.2 ± 1.2 years for the females. The female group also included one subject (D.W.) known to have an unusually marked metabolic response to cold exposure.

The rectal temperature curves show that the women cooled at a greater rate than the men, dropping 0.7°C in the first 20 minutes. The males did so after 30 minutes. No significantly different cooling rate was found. In both groups the metabolic rate and heart rate began to increase after this 0.7°C decline in rectal temperature.

The rewarming heart rate curves were remarkably similar to each other with the female curve 8 - 10 bpm above the male curve. After 30 minutes in the rewarm bath the female curve increased more acutely than did the male curve. This is concommitant with a more acute rise in the female mean rectal temperature which increased 0.6°C from 34.7°C to 35.3°C between 25 and 35 minutes. In the same period the male rectal temperature increased only 0.3°C (from 34.4°to 34.7°C). There were no significant heart rate or rectal temperature differences between groups.

Effect of Body Size Differences

It was noted previously that mean cooling rate and heart rates were greater in females than in males and that the characteristic increase in metabolic rate and heart rate in the latter part of cold immersion occurred earlier in females. One major factor in these differences is likely the smaller body size in females whose mean body weight was 56.6 Kg compared to male mean weight of 79.3 Kg. Responses of 2 small groups of females (n = 2) from Series 2 were compared to eliminate the sex variable. Body size was crudely indexed as body weight. The two groups had mean weights of 51.8 Kg and 61.3 Kg and mean triceps skinfold thicknesses of 15.6 mm and 14.6 mm respectively.

It was recognized that with such a small sample size no statistical relationships could be concluded but trends were considered to be of value. One would expect that if sex differences were due mainly to body size differences, the lighter females with less insulative capacity and greater surface to volume ratio would respond as the whole female group did (that is, with greater cooling rate and greater heart rate during the cold immersion). Surprisingly the lighter females had a lesser cooling rate (0.4°C in 20 minutes and 1.6°C in 40 minutes) than the heavier group (0.8°C in 20 minutes and 2.2°C in 40 minutes). They did have a higher heart rate level which remained between 91 and 108 bpm (at 20 minutes the heart rate was 91 bpm, 7 bpm greater than that of the heavier group, but this was not significant). The higher levels probably reflected a sustained high metabolic rate from the beginning of immersion (before any cooling had occurred) enabling heat production to overcome greater heat losses due to size. This was not the usual pattern of heart rate increase following the metabolic rate after some cooling has taken place. Therefore this response differs from that of the female group as a whole (Figures 19 and 20; Table 10).

Heart Rate-Rectal Temperature Relationship

In all studies of hypothermia under general anaesthesia (52, 35) and in deeply hypothermic conscious patients (69, 97), bradycardia is progressive with decreasing body temperature. Although some subjects in this study have had bradycardia at lowered body temperatures, this finding has been rare and as cooling has progressed, the heart rates have actually increased.

To investigate the body temperature--heart rate relationship, regression analysis was done using data from Series 2 consisting of heart rate and

Fig. 19. Comparison of Rectal Temperature Response of Female Subjects of Two Body Weights Holding Still in 10.5°C Water.
Each curve is a line of best fit through mean variates of 2 subjects.
No significant difference was found.

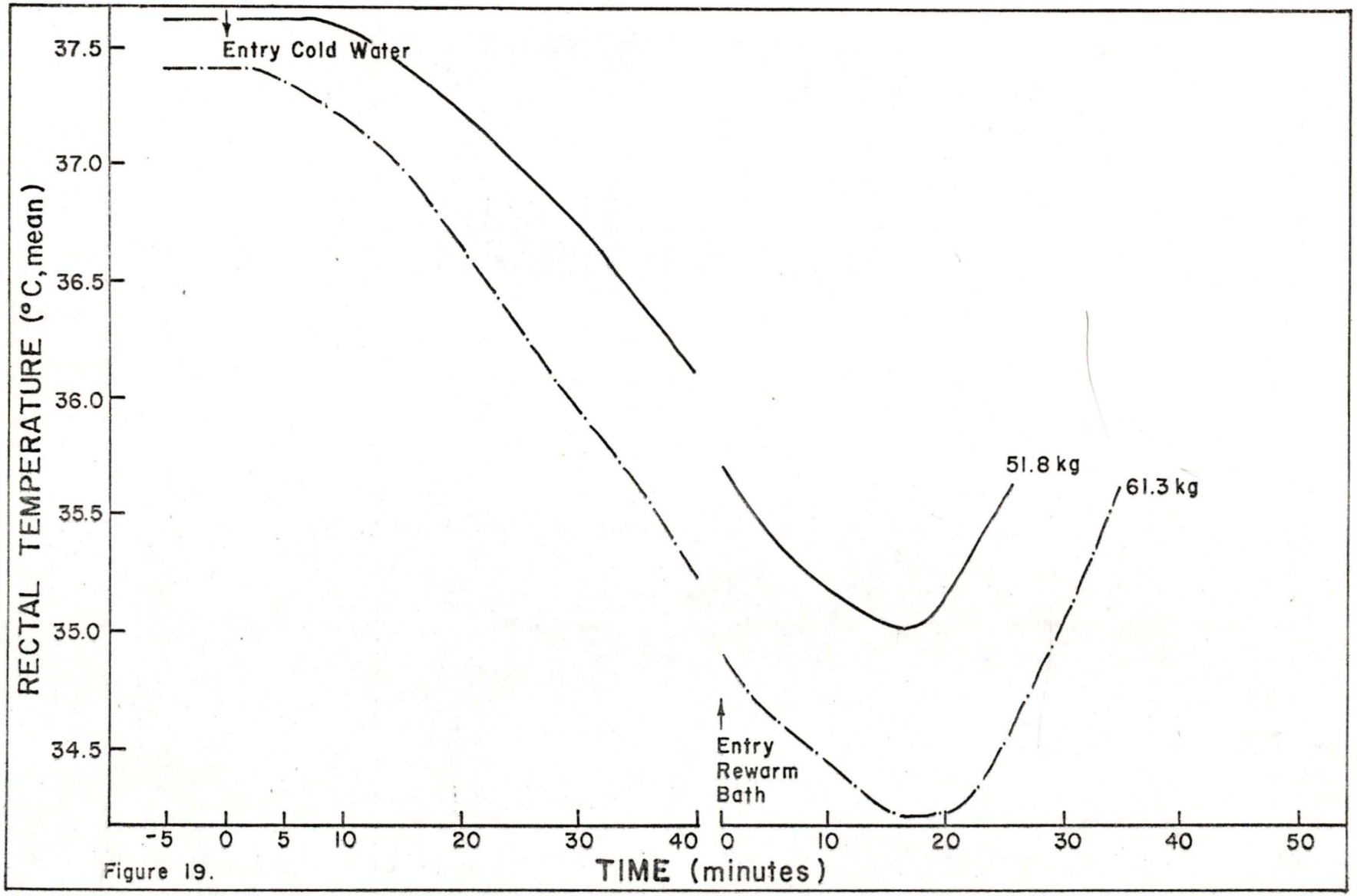

Entry Cold Water
51.8 kg
61.3 kg
Entry
Rewarm
Bath
RECTAL TEMPERATURE (°C, mean)
37.5
37.0
36.5
36.0
35.5
35.0
34.5
-5
0
5
10
20
30
40
0
10
20
30
40
50
TIME (minutes)

Figure 19.

Fig. 20. Comparison of Heart Rate Response of Female Subjects of Two Body Weights Holding Still in 10.5°C Water.

Each curve is a line of best fit through mean variates of 2 subjects. No significant difference between the 2 groups was found during cold immersion.

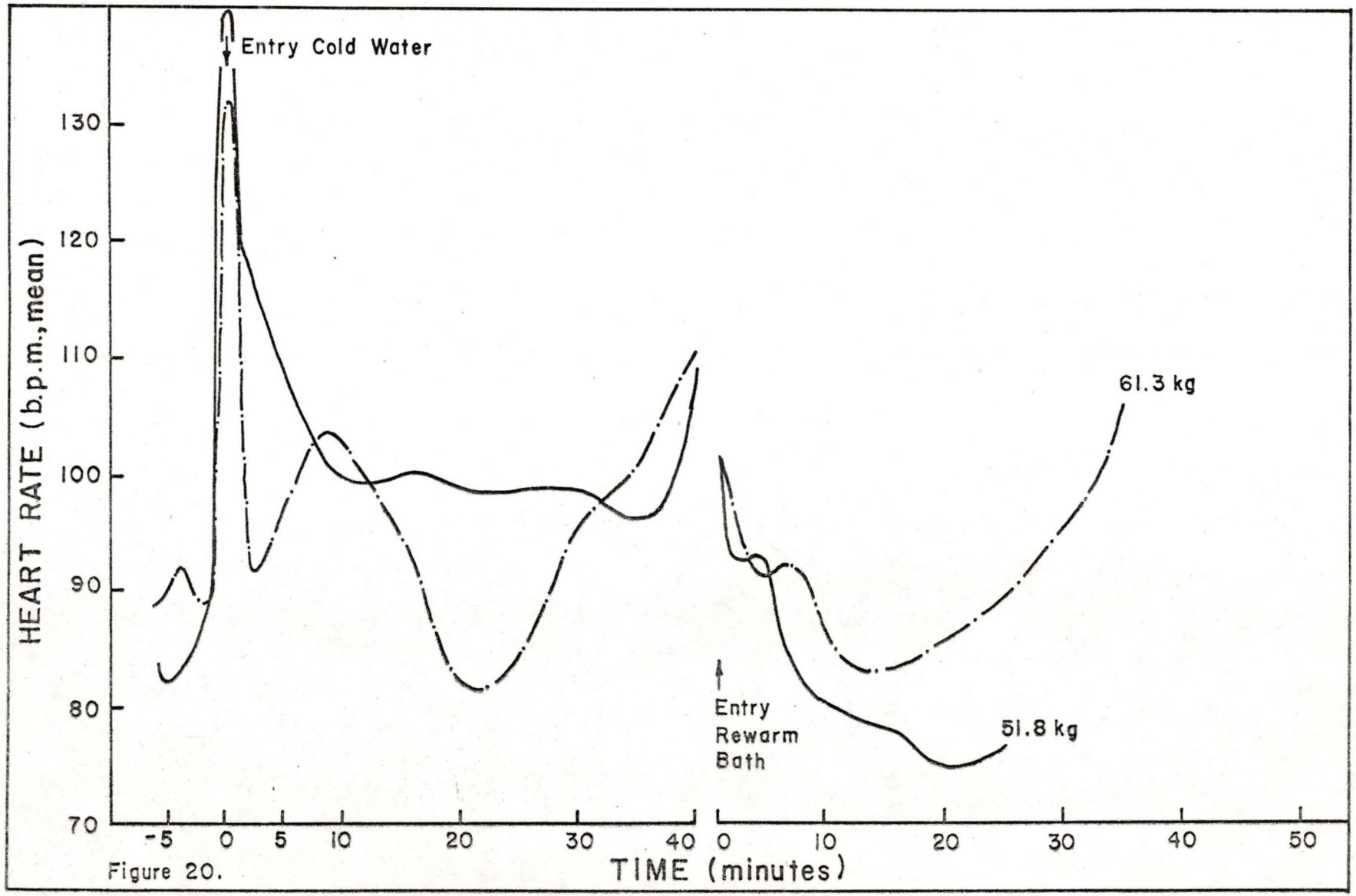

Entry Cold Water
HEART RATE (b.p.m., mean)
130
120
110
100
90
80
70
61.3 kg
51.8 kg
Entry
Rewarm
Bath
-5
0
5
10
20
30
40
0
10
20
30
40
50
TIME (minutes)

Figure 20.

TABLE 10. BODY WEIGHT EFFECT ON HEART RATE AND RECTAL TEMPERATURE AT 10.5°C

Parameter	Body Weight (Kg)	Time In Cold Water (minutes)	
		Control	20
Rectal Temp. °C [1]	51.8	37.6	37.2
	61.3	37.4	36.6
Heart Rate bpm [1]	51.8	85	91
	61.3	94*	84

* $p < 0.05$ difference from lighter weight

1 Mean of 2 subjects

rectal temperature values for 12 subjects immersed at 10.5°C.

Figure 21 is a graphic presentation of the regression lines. The heart rate values were taken as the mean of all mean minute values for a given mean rectal temperature (independent variable). That is, the mean rectal temperature was 37.4°C for 7 minutes and there were 7 mean heart rate values for 37.4°C. The mean of the mean values was taken as the heart rate value for regression against 37.4°C. It was felt that this method was more accurate than picking a single heart rate value from the several at a given temperature. Heart rate is so variable, the single value picked may be unrepresentative for the temperature.

Three time periods were regressed as described above:

(1) minutes 1 - 22 of cold immersion (positive sloping)

(2) minutes 23 - 45 of cold immersion (negative sloping)

(3) minutes 1 - 22 of rewarming (positive sloping)

A linear regression equation, 95% confidence limits and a Pearson product moment correlation coefficient were calculated for each regression line. Significance of these values was tested with the student t test (Table 11).

The first regression (Figure 21) showed a linear decline in heart rate from 94 to 79 bpm while the rectal temperature dropped 0.5°C from 37.4 to 36.9°C. The regression line, $Y = (-1102 + 32x)$ was highly significant ($p < 0.001$) and the correlation coefficient $r = 0.80$ was significant at $p < 0.01$. This seems equivalent to the trend in anaesthetic hypothermia but probably is not due solely to hypothermia. Decreased anxiety, reduced activity and the baroreceptor reflex play a role in this reduction, but probably not after the first 10 minutes. It is conceivable that some of the decrease in rate is due to hypothermia after 10 minutes in the cold water.

Fig. 21. Regression of Heart Rate on Rectal Temperature in Subjects Holding Still in 10.5°C Water.

Each regression line is a line of best fit through mean heart rate values at given rectal temperatures. Broken lines are 95% confidence limits. Each of the three linear regression coefficients (slopes) is significant ($p < 0.001$) as is each Pearson product-moment correlation coefficient ($p < 0.01$) (Table 11).

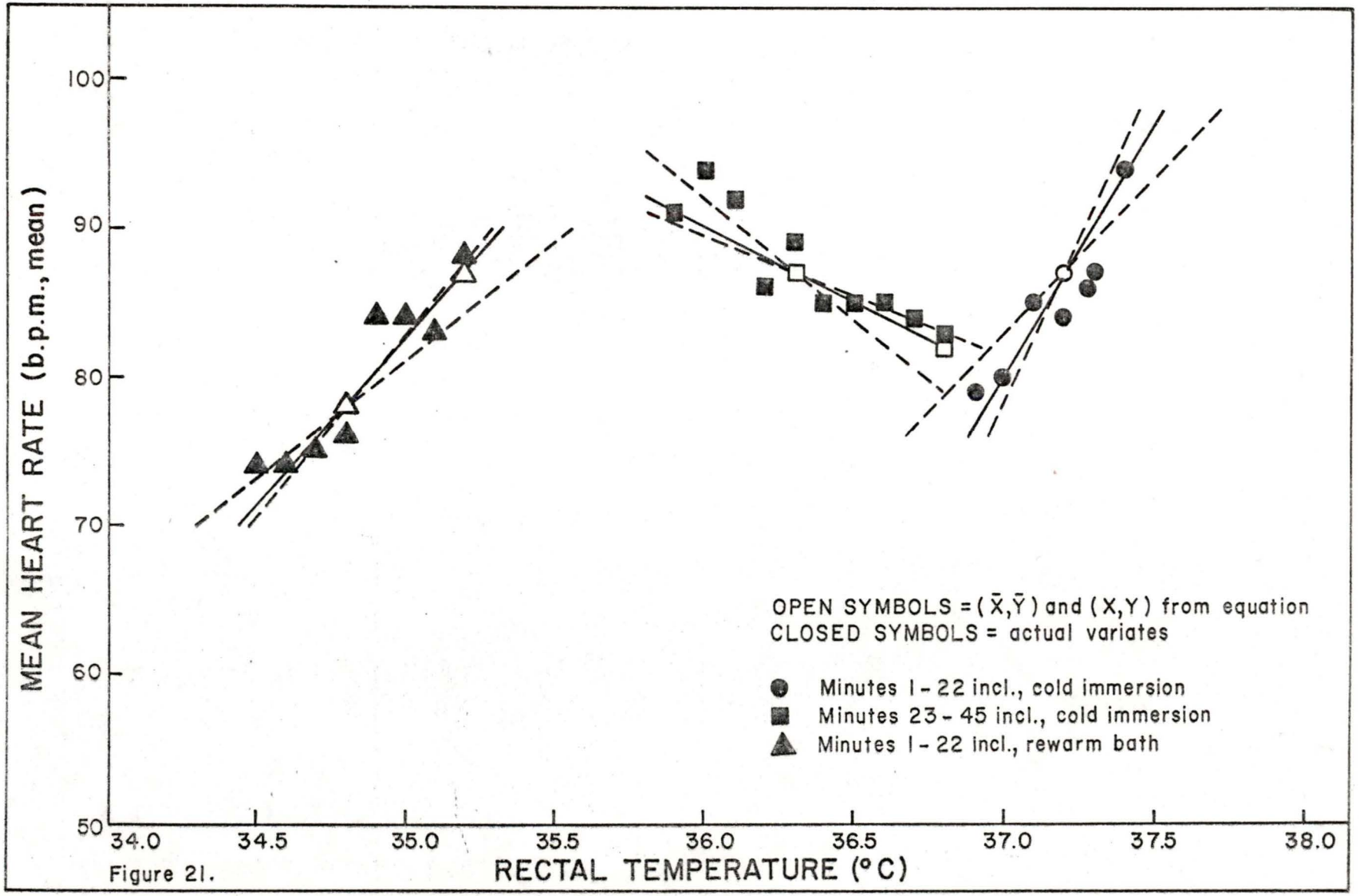

MEAN HEART RATE (b.p.m., mean)
100
90
80
70
60
50
34.0
34.5
35.0
35.5
36.0
36.5
37.0
37.5
38.0
RECTAL TEMPERATURE (°C)
OPEN SYMBOLS = ($\bar{X}$,$\bar{Y}$) and (X,Y) from equation
CLOSED SYMBOLS = actual variates
Minutes 1 - 22 incl., cold immersion
Minutes 23 - 45 incl., cold immersion
Minutes 1 - 22 incl., rewarm bath

Figure 21.

TABLE 11. REGRESSION AND CORRELATION OF HEART RATE AND RECTAL TEMPERATURE OF 12 SUBJECTS IMMERSED IN 10.5°C WATER

Value	Cold Water Immersion (minutes)		Rewarming (minutes)
	1 - 22	23 - 45	1 - 22
r	0.80**	-0.81**	0.91**
95% con. limit: L_1	20.8	-15.6	24.7
95% con. limit: L_2	43.1	- 7.9	16.0
Regression Eqn.	Y=(-1102)+(32)x	Y=(514)+(-12)x	Y=(-630)+(20)x
Slope $\pm$ S.D.	32$\pm$5***	-12$\pm$3***	20$\pm$2***

** $p < 0.01$ difference from 0

*** $p < 0.001$ difference from 0

The regression of minutes 23 to 45 shows a negatively sloping line as heart rate increased from 82 to 94 bpm while rectal temperature declined to 35.9°C. The equation, $Y = 514 - 12x$, was highly significant ($p < 0.001$) and the coefficient ($r = 0.81$) was significant ($p < 0.01$). The decline of heart rate is likely being overcome by a force not present in the anaesthetized patient, shivering thermogenesis. There may still be a cooling effect on the heart keeping rate in this period lower than a normothermic heart for the same metabolic rate.

During the rewarming period the regression line again had a positive slope ($Y = ^{-}630 + 20x$) along which heart rate declined from 88 to 74 bpm while rectal temperature fell to 34.5°C. The regression was highly significant ($p < 0.001$) and the coefficient ($r = 0.91$) was significant ($p < 0.01$). As seen before, the metabolic and heart rates were elevated for the first 5 or 10 minutes of rewarming until the thermogenic stimulus subsided and periferal receptor firing rate approached zero. In Series 2 at 10 minutes, the mean rectal temperature was 34.8°C, shivering had ceased and the mean heart rate was 76 bpm, 3 bpm lower than the last included heart rate in the first regression. It appears that once the metabolic rate falls off, the heart rate again begins to decline linearly with rectal temperature, at least until the tachycardia near the end of rewarming.

A similar pattern of regression curves was found in Series 1 data (4.6°C sea temperature) but this is not included here. These regressions are discussed here as there is no suitable section of the Discussion in which to include them.

ELECTROCARDIOGRAPHIC RESPONSE

Another important cardiovascular parameter is the electrocardiogram (ECG) which was monitored in most subjects according to the protocol in the Methods section. Technical problems did not permit use of all tracings in the analysis of ECG alterations but under each section below reference is made to the sample used.

It may be useful, as there is no specific ECG section in the discussion, to consider here the basis for the use of the ECG in this study.

The surface ECG is representative of the electrical activity of cardiac muscle fibres of three types: 1) pacemaker tissue (normally, the sino-atrial node in the right atrium; abnormally, an ectopic focus in atrium or ventricle), 2) conduction tissue (from sino-atrial node to the atrio-ventricular node, from atrio-ventricular node through the Bundle of His and then to the right and left bundle branches in the ventricles) and 3) ordinary myocardial tissue. The electrical activity may be altered by cold in several ways, most of which are shown by the ECG:

1. Slowed depolarization delaying both impulse formation and conduction (chronotropic and dromotropic effects). Manifestations are slowed heart rate, lengthened time components and configurational changes.

2. Slowed repolarization in which the active process of repolarization is delayed by depressed cellular metabolism. Manifestations are lengthened time components and configurational changes.

3. Increased irritability (bathmotropic effect) manifest by arrhythmias.

4. Electrolyte disturbances at both cellular and systemic levels reflected in configurational changes and arrhythmias.

The only parameter of cardiac function not estimable by the ECG is the inotropic factor (contractility or stroke volume). Alterations in ECG may be due to direct hypothermic effects on myocardial metabolism, relative ischemia or catecholamine release.

Although it is straightforward to document changes that occurred in individual or mean ECG parameters, interpretation is difficult. Any specific finding, for example a downward sloping ST segment or a prolonged conduction time, may be found in many physiological or pathological states. Some findings may simply be unmasked by the added stress of mild hypothermia without being directly caused by hypothermia. The exact mechanism of findings is often disagreed upon and remains unclear (e.g. junctional point deflection (52, 35, 59)). It seems therefore realistic to attempt a discussion of findings and to propose likely causative mechanisms within the study, rather than to conclude pathological states.

ECG Time Components

Three time components were measured from the ECG tracings of 10 subjects, 6 males and 4 females, participating in 12 separate immersions (1 male and 1 female had 2 immersions each). The mean drop in rectal temperature was $2.5 \pm 0.2^{\circ}C$. For other subject data see Tables 2 and 3. The PR interval (atrial depolarization), QRS interval (ventricular depolarization), and QTc interval (ventricular depolarization and repolarization corrected for heart rate) were manually measured at pre-immersion rectal temperature, then at decreasing temperature during immersion and rewarming. Values were obtained at 37.0, 36.5, 36.0, 35.5, 35.0 and $34.5^{\circ}C$ (see Methods). Mean values for each parameter were tested for significance of difference with the student t test. Table 12 summarizes these results which are plotted in Figure 22.

The pre-immersion value of the PR interval was 0.16 seconds. This increased to 0.18 seconds at a rectal temperature of $34.5^{\circ}C$, but still remained within the upper limit of normal (0.21 seconds (42)). It was

Fig. 22. ECG Time Component Changes with Declining Rectal Temperature. The mean of 12 values for PR, QRS and QTc intervals in seconds is plotted for each 0.5°C decline in rectal temperature from 37.0°C to 34.5°C. The student t test comparing values at 34.5 and 37.0°C shows no significant difference ($p < 0.05$) for PR and QRS but QTc interval is significantly different at $p < 0.01$.

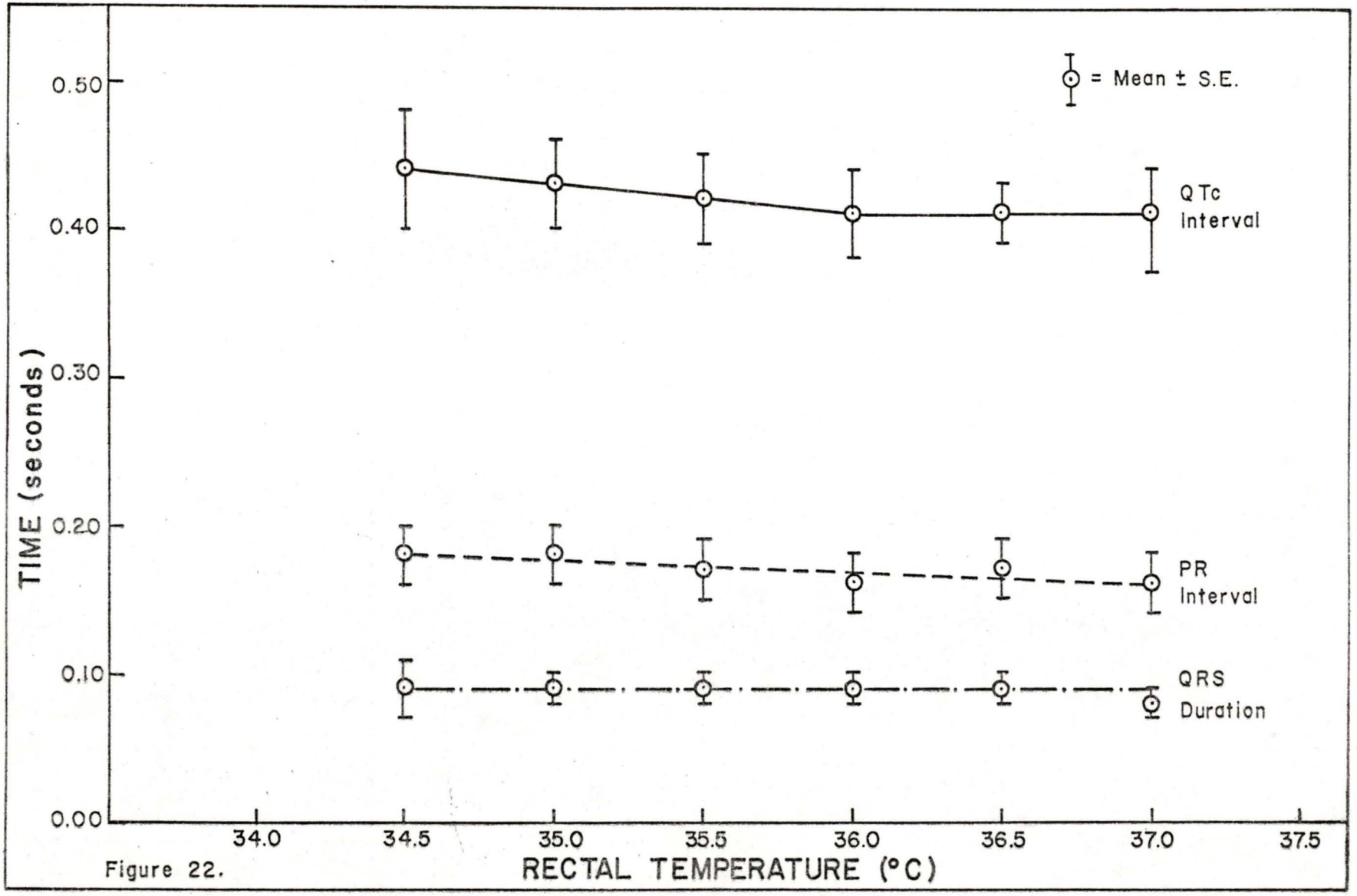

0.50
0.40
0.30
0.20
0.10
0.00
TIME (seconds)
= Mean ± S.E.
QTc Interval
PR Interval
QRS Duration
34.0
34.5
35.0
35.5
36.0
36.5
37.0
37.5
RECTAL TEMPERATURE (°C)

Figure 22.

TABLE 12. ECG TIME COMPONENTS (SECONDS ± S.E., MEAN OF 12 VALUES)

Parameter	37.0°C	34.5°C	Difference
PR Interval	0.16±.02	0.18±.02	NS
QRS Interval	0.08±.01	0.09±.02	NS
QTc Interval	0.41±.03	0.44±.04	$p < 0.05$

not significant at $p < 0.05$. The QRS interval was 0.08 seconds pre-immersion and 0.09 seconds at a rectal temperature of 34.5°C. This was not significant at $p < 0.05$ and was well within the normal limit of 0.12 seconds. The QTc interval was significantly increased at $p < 0.05$, rising from 0.41 seconds to 0.44 seconds with cooling to 34.5°C. This was above the upper limit of normal for normothermic patients (0.43 seconds) and is of potential pathological significance (42).

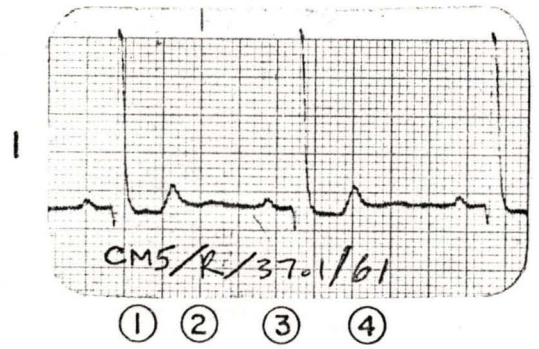
ECG Configurational Changes

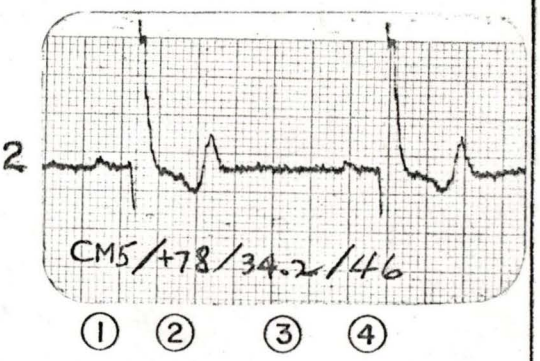
There is considerable variation in configuration of waves in the normal ECG but these limits are exceeded in pathological and certain physiological states such as hypothermia. To assess such changes in this study, ECG tracings from 33 subjects (14 males and 19 females) participating in 102 immersions were examined for such changes (see Methods, Tables 2, 3). The occurrence of changes is listed in Table 13.

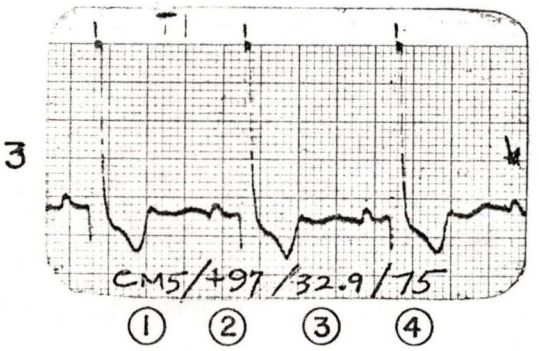
No significant P wave changes were found and no subjects showed prolonged QRS intervals. One subject (J.E.) had a depressed downward sloping ST segment at a rectal temperature of 32.9°C (Figure 23), suggestive of ischemia although no subjective evidence was found (63). Figure 24 shows several alterations in a single female subject, age 20 (L.T.). The QTc interval increased from 0.43 seconds at a rectal temperature of 37.0°C to 0.51 seconds at a rectal temperature of 33.9°C, then returned to 0.44 seconds as the rectal temperature became 35.1°C. The ST segment was elevated 1.0 mm. above the isoelectric line in the pre-immersion tracing and rose to 1.8 mm. above at a rectal temperature of 33.9°C. Later when rectal temperature increased to 35.1°C the ST segment was isoelectric. A similar pattern occurred in two other subjects. Although associated with acute myocardial infarction, ST elevation is found in anxious, young, athletic individuals and this is the likely cause here.

TABLE 13. ECG CONFIGURATIONAL CHANGES OBSERVED (NO. OF SUBJECTS)

Parameter	Drop in Rectal Temp. $< 0.5^{\circ}C$	Drop in Rectal Temp. $\geq 0.5^{\circ}C$
P wave changes	0	0
QRS changes	0	0
ST elevation	0	3
ST depression	0	1
J point deflection	0	2
T wave peaking	-	17/22 (77%)



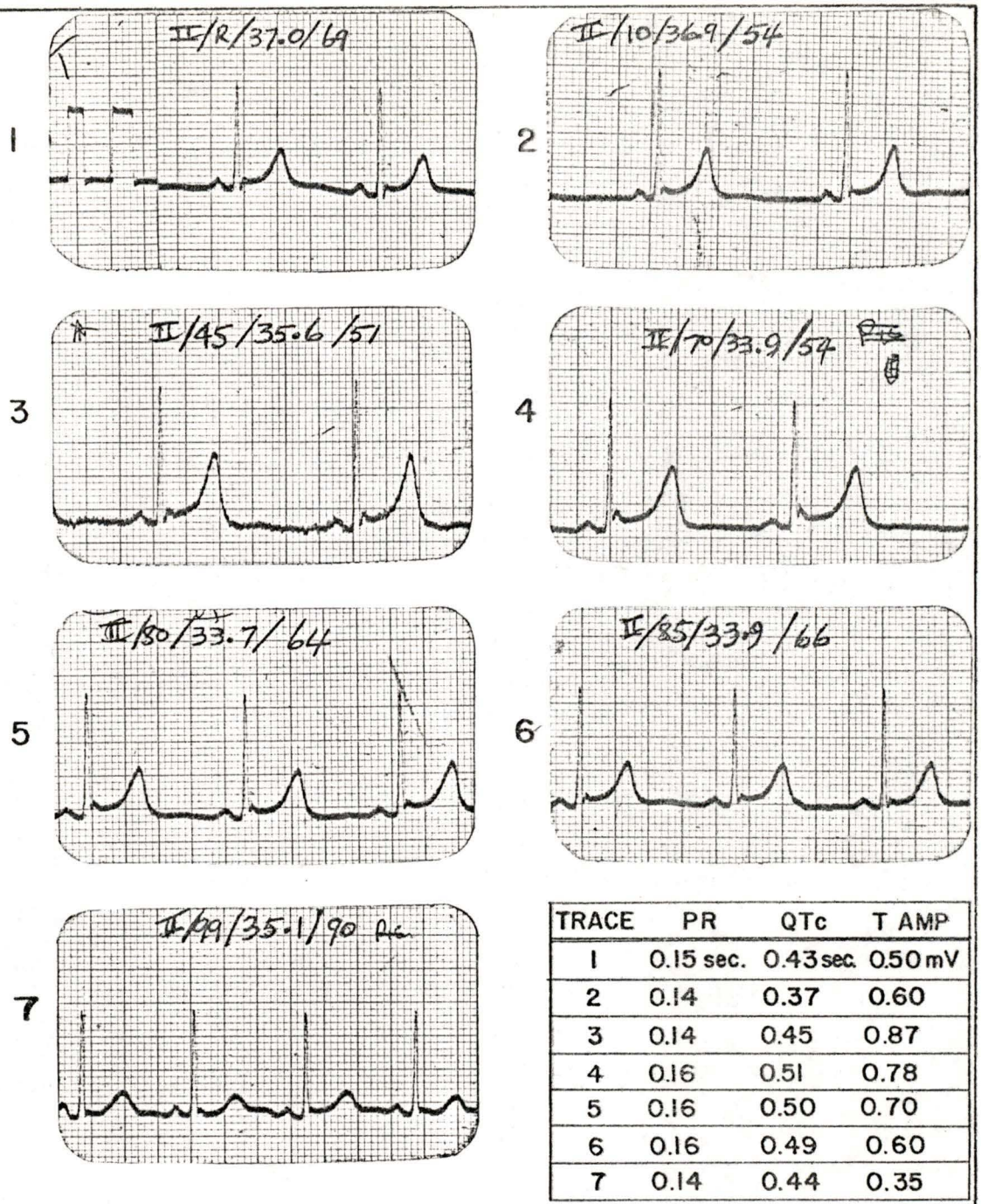




KEY TO ECG TRACING CODE
① Lead
② Time taken: "R" = Pre immersion rest "+5RE" = 5 minutes in rewarm period.
③ Rectal temperature (°C)
④ Heart rate (b.p.m.)

Figure 23.

Fig. 24. Configurational and Time Component Changes in Subject L.T. The small junctional point deflection (J wave) which is present at rest (1), is accentuated at a rectal temperature of 33.9°C (4), and becomes minimal as rectal temperature rewarms to 35.1°C (7). The ST segment is elevated 1.0 mm. in (1), 1.8 mm. in (4) and becomes isoelectric in (7). The phenomenon of increasing T wave amplitude during immersion is shown in the tracings and the table. It increases from 0.50 mV (1) to 0.87 mV in (3) but even though rectal temperature continues to fall in rewarming (tracings (4) and (5)), the T wave progressively gets smaller after shivering ceases (note shivering tremor in (3) that is absent in (4)). In tracing (7) the rectal temperature is 35.1°C yet the T wave amplitude is 0.35 mV, well below the pre-immersion value. Time components PR and QTc in seconds are shown in the table to be greatest at 33.9 and 33.7°C and to return to pre-immersion levels when rectal temperature becomes 35.1°C.



TRACE	PR	QTc	T AMP
1	0.15 sec.	0.43 sec.	0.50 mV
2	0.14	0.37	0.60
3	0.14	0.45	0.87
4	0.16	0.51	0.78
5	0.16	0.50	0.70
6	0.16	0.49	0.60
7	0.14	0.44	0.35

Figure 24.

The junctional point between the QRS and ST has been deflected upwards producing a "J wave" in many studies (52, 59) and at as high a rectal temperature as 35.3°C (35). There was a small deflection in Figure 24 in the pre-immersion tracing which was accentuated at a rectal temperature of 33.9°C. It became minimal when temperature rose again to 35.1°C. Emslie-Smith et al. (35) recommended that several leads be recorded to ensure visualization of the J wave and this need is shown in Figure 25 (subject M.C.). Leads II and V_5 showed an isoelectric point at pre-immersion. At 35.2°C lead V_5 showed a J wave whereas lead II did not. Although not shown in Figure 24, the tracings for that subject showed no J wave and lead II tracings did.

A phenomenon of increasing T wave amplitude and peaking of the waves with cooling was observed in many subjects. This is exemplified in Figure 24. The T wave increased from 0.50 millivolts (mV) at a rectal temperature of 37.0°C to 0.87 mV at 35.6°C. After shivering ceased and as rectal temperature became stable in most subjects, the T wave abruptly became lower in amplitude. In Figure 24 it decreased progressively from 0.78 V at a rectal temperature of 33.9°C to 0.35 mV at 35.1°C. This was smaller than the pre-immersion T wave even though rectal temperature was well below pre-immersion level. In order to assure that the T wave amplitude was changing irrespective of the rest of the ECG, R wave amplitude, proportionate to the increase in T wave amplitude, was calculated and compared with actual R waves for the subject in Figure 24. The results in the table below show that the T wave was increasing proportionately more than the R wave.

Time (min.)	T amplitude (mV)	Proportionate R amp. (mV)	Actual R amp. (mV)
PRE	0.50	---	---
45	0.87	2.8	2.1
85	0.60	1.9	1.7

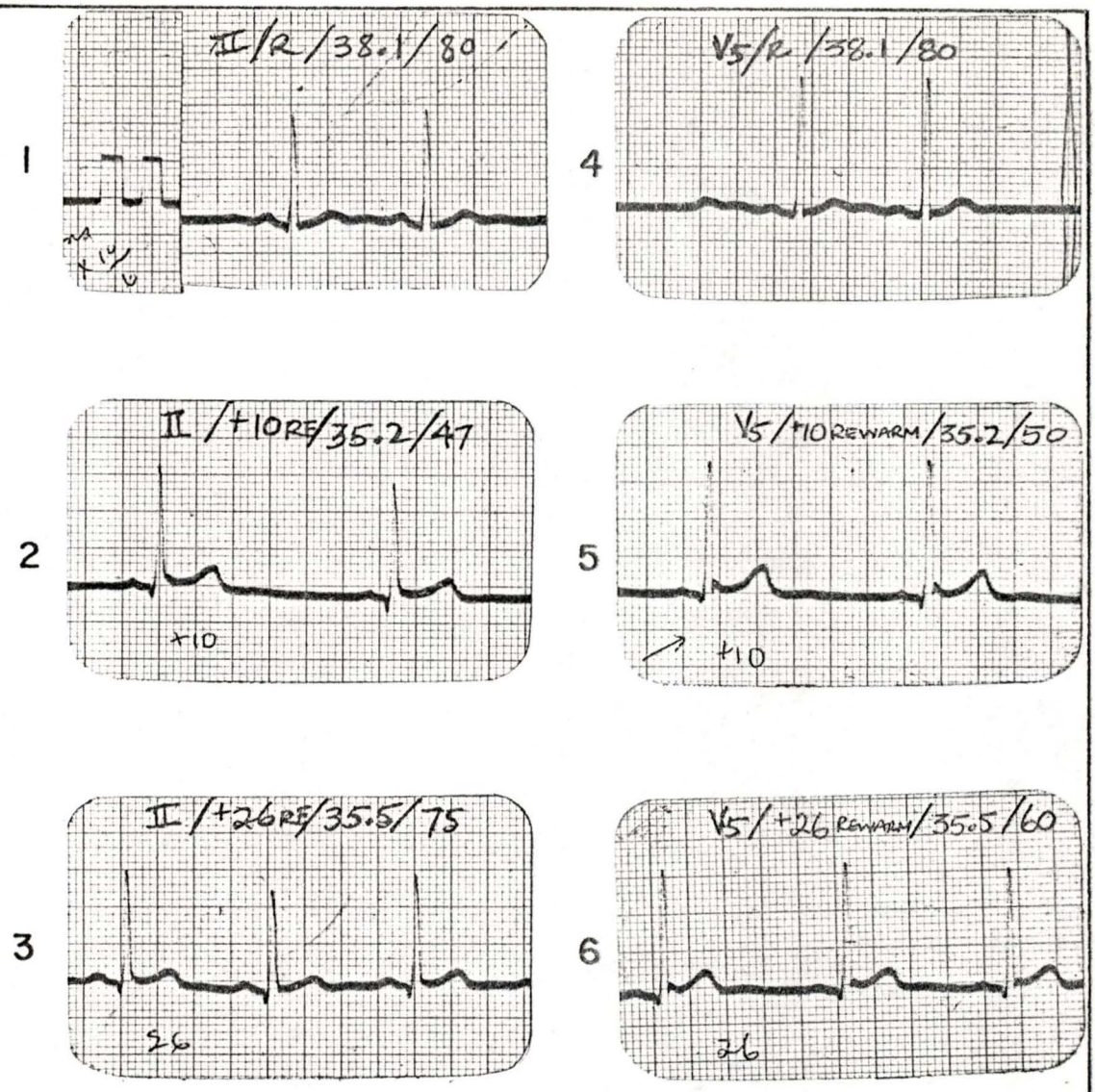

1
II/R/38.1/80
4
V5/R/38.1/80
2
II/+10RE/35.2/47
+10
5
V5/+10REWARM/35.2/50
+10
3
II/+26RE/35.5/75
26
6
V5/+26REWARM/35.5/60
26

Figure 25.

Using 8 subjects, the T wave phenomenon was quantified (Table 14). The mean wave amplitude was 0.40 mV at 37.0°C and increased to 0.72 mV at 35.2°C. This was significant at $p < 0.01$. Then at the same rectal temperature (35.2°C) as temperature was increasing from a lower level, the T wave amplitude was 0.40 mV or equal to the pre-immersion value.

ECG Arrhythmia

The ECG was monitored to see if any arrhythmias would occur during initial immersion or during mild hypothermia. Table 15 shows the incidence of observed arrhythmias in 33 subjects during 102 immersions.

Sinus tachycardia occurred in almost every subject upon immersion and is to be expected with the anxiety, activity and cold receptor stimulation. The mean maximum heart rate was less than 140 beats per minute. Tachycardia was sustained during Series 4 while subjects were swimming. Sinus bradycardia (heart rate less than 60 bpm) was found infrequently even during low body temperatures. It was seen in 5 subjects before their rectal temperature had declined 0.5°C and in 11 subjects after a drop in rectal temperature greater than 0.5°C. Sinus arrhythmia by definition must be a variation of at least 10% of the mean of the two extreme heart rates on an ECG tracing so many minor variations due to respiration and blood pressure regulation are not included. Seven cases were observed, 4 of which occurred after a rectal temperature decline of $\geq$ 0.5°C. An example is seen in subject R.J. (Figure 26).

Premature contractions (extrasystoles) occur before the sino-atrial node depolarization spreads throughout the myocardium. They result from delayed sino-atrial depolarization (e.g. from cooling) or from increased excitability of an ectopic pacemaker (e.g. from cooling or catecholamine stimulation). Atrial extrasystoles were seen in 1 subject before a

TABLE 14. T WAVE AMPLITUDE (mV±S.E., MEAN OF 8 SUBJECTS)

	37.0°C	35.2°C	Difference
Cooling	0.40±0.19	0.72±0.22	$p < 0.01$
Rewarming	0.40±0.19	0.40±0.16	NS

TABLE 15. ECG ARRHYTHMIAS OBSERVED
(NO. OF SUBJECTS)

Parameter	Drop in Rectal Temp. $< 0.5^{\circ}C$	Drop in Rectal Temp. $\geq 0.5^{\circ}C$
Sinus bradycardia	5	11
Sinus arrhythmia	3	4
Premature Atrial Contraction	1	2
Premature Ventricular Contraction	1	0
Bigeminy	0	1

Fig. 26. Sinus Arrhythmia.

At rest (1) this subject (R.J.) had normal sinus rhythm which occasionally became sinus arrhythmia after rectal temperature had declined 0.1°C (2) and remained so during further cooling and rewarming (3) and (4). Seven of 33 subjects showed similar patterns.

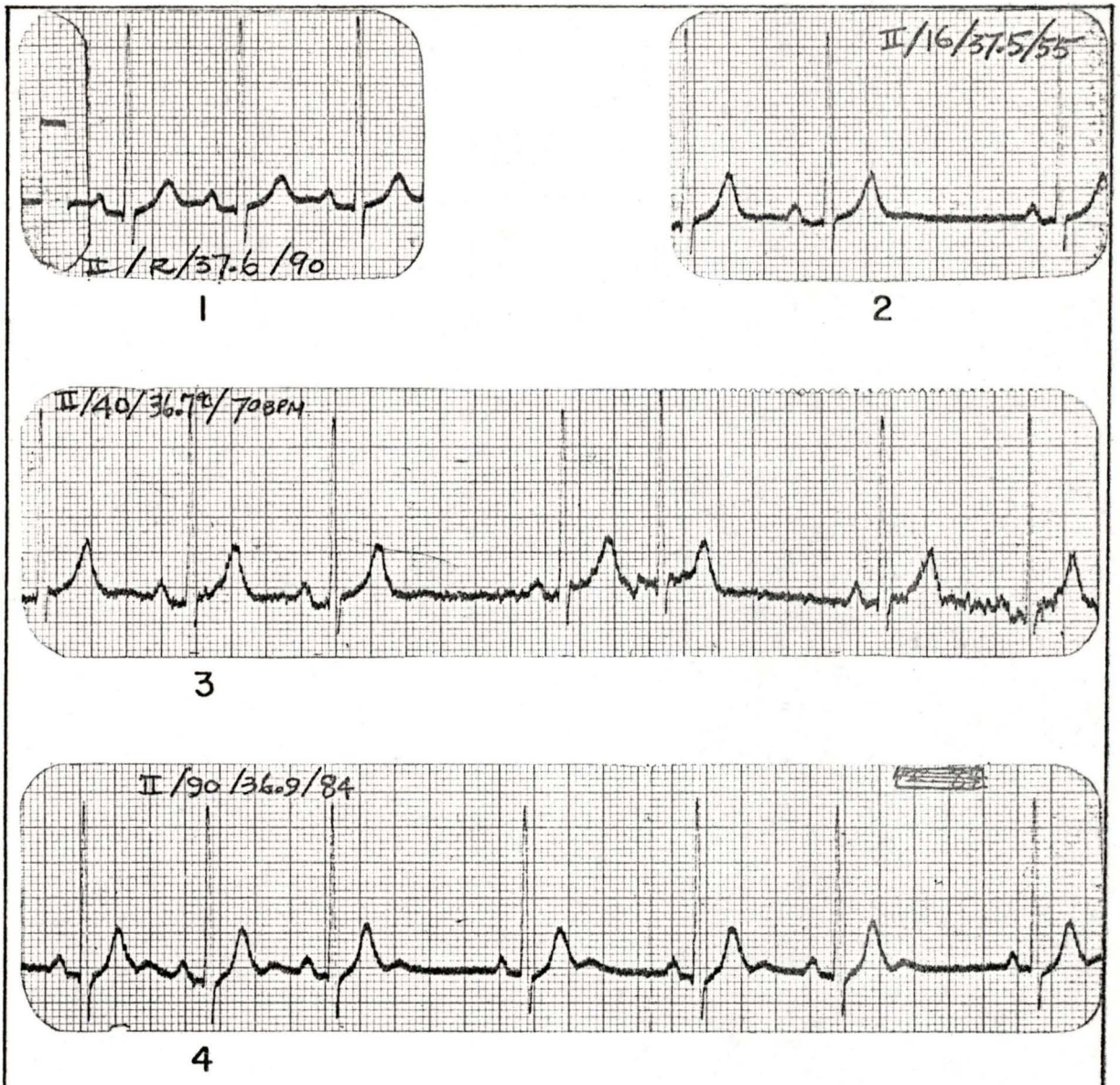
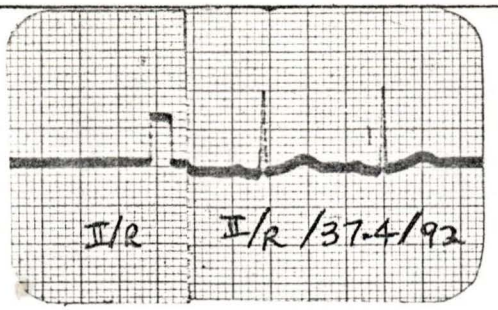


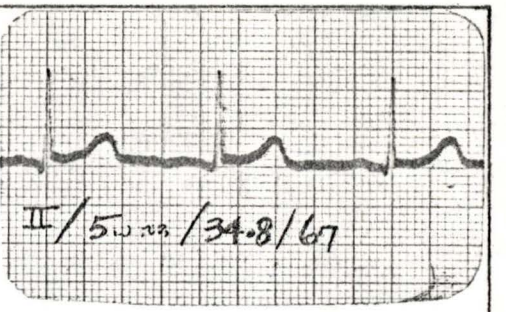
Figure 26.

significant drop in rectal temperature and in 2 others after rectal temperature declined $\geq 0.5^{\circ}C$ (Figure 27). No atrio-ventricular nodal extrasystoles were seen. Ventricular extrasystoles were observed in 2 female subjects both 22 years old (Figure 28 and 29). One had 2 isolated unifocal ventricular extrasystoles 9 beats apart in the first 30 seconds of immersion in $10.5^{\circ}C$ water. The second subject (Figure 29) had unifocal ventricular bigeminal rhythm (alternating normal and ectopic beats) after her rectal temperature had declined $0.7^{\circ}C$. The subject was asymptomatic and normal sinus rhythm resumed upon removal from the cold water.

No other arrhythmias were observed.



1



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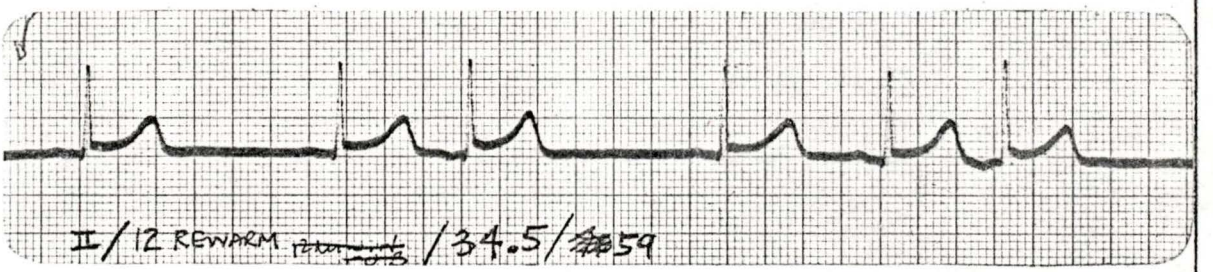

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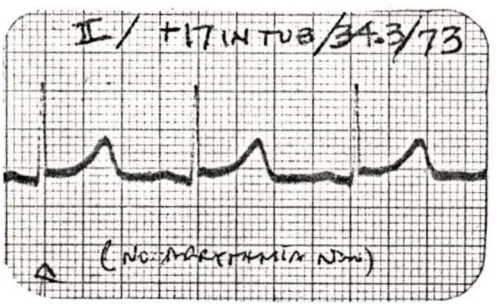

4

Figure 27.

Fig. 28. Premature Ventricular Contractions Upon Immersion. This subject (D.T.) showed 2 isolated unifocal P.V.C.'s 9 beats apart within 30 seconds of immersion in 10.5°C water. No other arrhythmia was seen in this subject.

Fig. 29. Unifocal Ventricular Bigeminal Rhythm in Mild Hypothermia. This rhythm was seen in only this subject (L.C.) after a decline in rectal temperature of 0.7°C from 37.8°C. Normal sinus rhythm followed removal from the cold water.

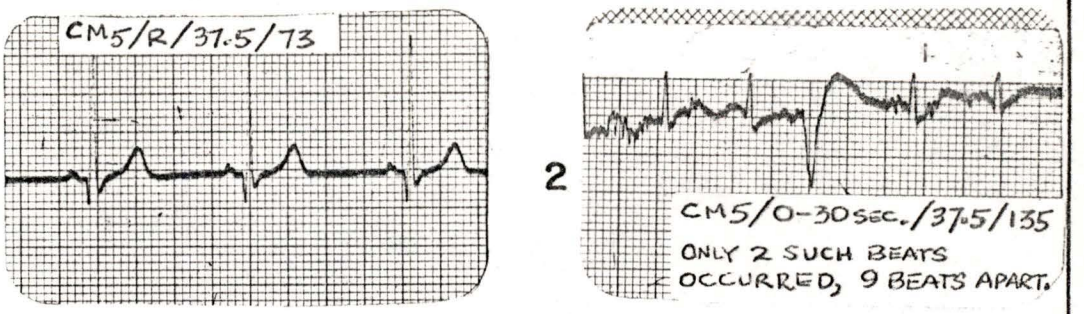

Figure 28.

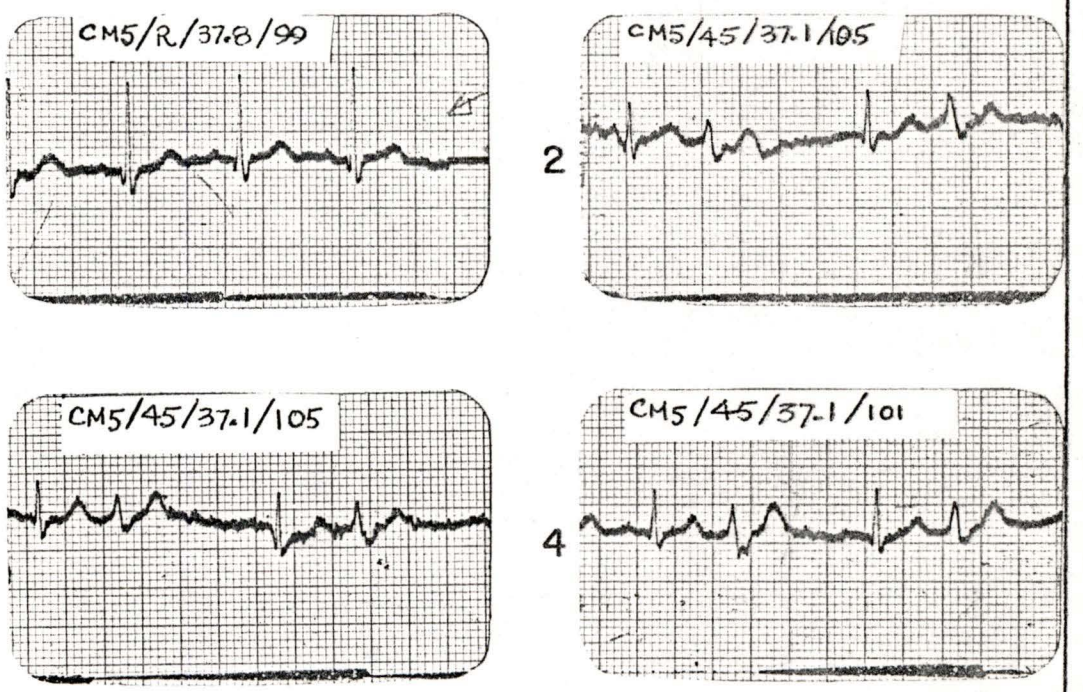

Figure 29.

DISCUSSION

HEART RATE

The Cooling Period

The progressive slowing of the heart rate with decreasing body temperature leading to sinus bradycardia is the common pattern seen in anaesthetized hypothermic subjects (31, 52, 88, 18) and in deeply hypothermic conscious subjects (96, 97, 25). This pattern was not found in conscious humans continuously monitored during cold water immersion and mild hypothermia.

This is understandable for one major reason: the central and periferal nervous system remains intact, not obtunded by anaesthetic or profound cooling. Anxiety, voluntary muscle movement and cutaneous cold receptors are able to stimulate sympathetic neurons to alter cardiac output and periferal resistance. Probably more important for survival, however, the periferal and central cold receptors stimulate hypothalamic vasoactive insulative reflexes and shivering thermogenesis. Shivering occurs until body temperature reaches about 32°C after which muscles become rigid (25). Hemingway (51) and Keatinge (65) conclude that there is little evidence for non-shivering thermogenesis in non-acclimatized man. The heart rate responses in conscious alert man, then, are a product of many more influences than in obtunded persons. Information on these responses has been scarce.

During the pre-immersion phase and the process of immersion, anxiety heightened in subjects immersed for the first time and undoubtedly this would happen in shipwreck victims. Muscular activity increased as subjects gained stability upon immersion and at that time a sensation of pain due

to vasoconstriction was felt by most subjects. The initial firing of cutaneous cold receptors tends to increase heart rate, respiratory rate and blood pressure (64, 23, 26). It is clear that all these factors account for tachycardia during the entry into cold water.

In the first 20 minutes of immersion the heart rate is influenced by the same factors. Anxiety and muscular activity decreased as subjects became oriented and attached to the life ring. Periferal vasoconstriction decreases the size of the vascular bed and raises blood pressure to initiate the baroreceptor reflex. Periferal cold receptors adapt or fatigue and decrease their firing rate after about 10 minutes (49). These changes act to gradually lower the heart rate from the initial peak even before cooling has occurred. After about 7 minutes the rectal temperature did begin to decline, giving the appearance of linearity with heart rate. This is probably an artifact of the factors mentioned above and has led to the common misconception that cooling of the sino-atrial node affects heart rate even in very early cooling such as reported in the Dachau experiments (1). The sino-atrial node is definitely slowed in a deeper hypothermia (14). This is due to cooling since atropine does not block this effect (82) but it is unlikely to occur with minimal cooling (83).

The metabolic rate was increasing in the first 20 minutes but the heart rate, which correlates with metabolic rate after 20 minutes was going in the opposite direction. This must be because it is at a sufficient level to give adequate cardiac output. Raven et al. (85) found a 78% increase in stroke volume but only a barely significant increase in heart rate in eleven subjects exposed to 5°C air. In a similar experiment O'Hanlon and Horvath (77) attributed increased oxygen consumption to either increased stroke volume or more efficient tissue oxygen extraction in subjects cooled less than 1.0°C.

A change occurred at about 20 minutes. Heart rate began to increase in correlation with metabolic rate ($r = 0.714$, significant at $p < 0.01$). This trend continued until the end of cold immersion as metabolic rate is driven by a declining core temperature in a vain attempt at thermoregulation. In more recent studies in this laboratory subjects clothed in bathing suits only have been studied in 10°C water while cooling to rectal temperatures of 34.0°C. Data from these experiments have shown that the correlation between oxygen consumption and heart rate continues for heart rates as high as 140 bpm at the end of cold immersion (49). There is nothing specific about 20 minutes. It simply signifies that sufficient cooling has occurred to stimulate central cold receptors. At both 10.5 and 4.6°C water temperatures this occurred after a rectal temperature decline of about 0.5°C. This compares favorably with Irving who is quoted in Hemingway (51) as finding that a decline in rectal temperature of 0 to 1.0°C is the threshold for shivering in men. Many subjects are stimulated to high levels of heat production as soon as they are immersed while others only respond after cooling much more than 0.5°C. One factor may be bad physical insulative capacity. Cannon and Keatinge (24) found a smaller metabolic response in fat than in thin subjects. This trend did not seem to apply to our subjects as was discussed earlier.

This general pattern of heart rate response to cold water immersion agrees with the findings of Behnke and Yaglou (13) and Keatinge and Evans (64). Probably the Dachau results (1) are similar in spite of the finding of a progressive fall in rate in 5.5°C water, because their intermittent recording could have missed the decline after immersion and the increase after threshold cooling.

Throughout the cold immersion 3 to 5 bpm variations in heart rate about 8 times per minute were seen. These did not coincide with respiration

and were probably due to central blood pressure regulation. Similarly the 5 to 10 bpm variations seen every 3 to 5 minutes were due to the same mechanism. The size of the vascular bed undoubtedly changes during vasoconstriction, subsequent changes in sympathetic tone and in muscle activity. Keatinge (65), and Budd and Warhaft (23) describe cold vasodilatation due to fatigue of constricting resistance vessels. This could be happening to a small degree but it is not as massive as they describe or the rectal temperature curve would reflect this.

The Rewarming Period

Upon removal from the water, muscular activity accounted for most of the tachycardia and once in the rewarm bath this began to subside. The uncomfortable few minutes of moderate to intense shivering is due to a combination of factors. Periferal cold receptors which have been adapted or fatigued may begin to fire aggressively with early skin rewarming (51) before skin temperature is near normal. The intense rewarm shivering in one subject reported by Behnke and Yaglou (13) occurred at a skin temperature of 21°C. The core temperature is continuing to decline during this period as renewed perfusion of cold tissues occurs (13). This may also contribute to increased metabolic drive.

After about 10 minutes in the bath, heart rate and metabolic rate declined and shivering ceased. The heart rate was positively correlated with metabolic rate. The metabolic stimulus is roughly the product of periferal and central inputs (46) and it seems likely that skin temperature has warmed sufficiently to stop periferal cold receptor firing making the net metabolic rate equal to the resting or maintenance rate (48). The regression of heart rate on rectal temperature at this time was highly significant and rectal temperature was about 34.5°C, down about 3°C from

normal. It is possible that hypothermia is affecting the sino-atrial node at this time.

There was a plateau of heart rate and rectal temperature after about 10 minutes in the bath. During this time the minute to minute heart rate fluctuations were often absent and occasionally euphoria consistent with hypotension and cyanotic extremities consistent with venous pooling were seen. The baroreceptor reflex functions at temperatures above 24°C (59, 82) so this cannot account for these findings. Perhaps there is a combination of decreased sympathetic tone, periferal vasodilatation due to an increased skin temperature and a so called vaso-vagal response of increased vagal tone. This last phenomenon is seen after vigorous exercise with an inadequate cool down period (34, 90). It begins with a progressive bradycardia followed by a systole and syncope, then reverses on assuming a supine posture or with external cardiac massage. Behnke and Yaglou (13) found blood pressures as low as 90/60 during the rewarming period. It therefore seems prudent to monitor subjective signs of hypotension, heart rate and ECG, and blood pressure during this period of rewarming, with adequate resuscitation available.

At about the time rectal temperature began to increase following the plateau there was a marked increase in heart rate lasting until termination of rewarming. This was unrelated to metabolic rate which is at pre-immersion levels and it is likely a baroreceptor reflex to the periferal vasodilatation.

Important Factors Altering Heart Rate

It is apparent that many factors influence the heart rate response of humans to cold water immersion. The more important factors were studied:

Immersion Per Se

The critical ambient temperature greater than which heat production increased above pre-immersion resting levels was 23.8°C (46). Immersion in water greater than this temperature may conceivably, however, have effects on heart rate through changes in the vascular volumes. That is, there may be an effect of immersion per se on heart rate. Keatinge and Evans (64) found no change in heart rate after immersion for 19 minutes at 35.0°C but found an increase at 37.8°C, presumably due to vasodilatation. Similarly, Craig and Dvorak (29) found no change in heart rate in subjects immersed at rest at 35.0°C for 1 hour. At temperatures above and below this they found increases and decreases respectively in the heart rate. For subjects at rest they concluded that the neutral ambient water temperature is between 35 and 35.5°C. In slightly cooler water (34.0 ± 0.5°C), Begin et al. (12) found no change in the heart rate or oxygen consumption during 4 hours of immersion. They found a 25% increase in cardiac output however, due to volume shifts to the pulmonary and intrathorathoracic vessels, thereby increasing stroke volume.

It seems that there is an ambient water temperature around 34 or 35°C at which no significant heart rate or oxygen consumption changes occur during immersion. This is good evidence that the effect of immersion per se on these variables is negligible.

Ambient Temperature

Exposure to ambient temperature below the critical temperature on the temperature metabolism curve is known to result in an increase in metabolic rate and such increase is greater for lower ambient temperatures (4, 40). The temperature metabolism curve for this study was described elsewhere (46) and it exemplifies the typical homeotherm pattern. Since heart rate levels seem most strongly related to metabolic rate we expected to see

higher heart rate levels at cooler temperatures. The heart rate curves at ocean temperatures of 4.6°C, 10.5°C and 18.2°C showed the trend of higher heart rates in colder water but these differences were not significant. The labile nature of the heart rate in this experimental situation due to anxiety, previous immersion, amount of motion, wave height and weather tends to bring the heart rates closer together at various times. These factors would need to be controlled for a more meaningful assessment.

The range of ambient temperatures used is narrow and perhaps a comparison of heart rates at 10.5°C and 24°C or more would show significant differences. In Series 2, the mean heart rate at 40 minutes of exposure was 95 bpm and Craig and Dvorak (29) studying 10 males (mean age 26 years) report mean heart rates of 59 bpm in 32°C water after 40 minutes of exposure. Lack of individual variates prevents a comparison of these values statistically but they differ by 36 bpm and 41 bpm respectively from Series 2, likely to be significant.

The pattern of the response at different water temperatures also varied. The rise in heart rate after cooling occurred later at 10.5°C than at 4.6°C, likely because cooling was slower and the hypothalamic metabolic stimulus was activated later. Similarly Keatinge and Evans (64) found an increase in heart rate after 19 minutes in water at 7.0°C. This occurred only after 40 minutes at 10.0°C (13). At 15.0°C Keatinge and Evans (64) found a slight decrease in heart rate from control levels as we did in subjects immersed at 18.2°C, even though they all shivered.

It seems that heart rate levels in cold immersion are mainly dependent upon metabolic rate. With a warmer ambient temperature cooling is slower and the increased metabolic demand can be met temporarily by increasing

arterio-venous oxygen difference and stroke volume (86). Apparently when a threshold of heat production is reached the heart rate increases, but at temperatures greater than 15.0°C this does not occur in the first hour or so of exposure.

Thermal Protection

The value of increasing thermal protection and decreasing heat loss is obvious, particularly when it is combined with a buoyancy device such as the UVIC THERMOFLOAT.[1] The heart rate data comparing a kapok life vest, a floater coat, and a THERMOFLOAT-like coat were somewhat confusing. The heart rates for the kapok vest were lower than in Series 2 (10.5°C) even though the water was over 2°C colder and the cooling rate was greater. These results may be artifactual due to a smaller sample size, use of 3 frequently immersed subjects and reduced anxiety because the ship was in dock. However, even with the same subjects, the heart rate values were greater with greater thermal protection and slower cooling rate. This appears paradoxical according to the relationship between heart rate and metabolic rate. At first it was thought that the insulation may have enabled periferal receptors to continue to fire at a greater rate than in less well insulated skin, thereby increasing metabolic rate and heart rate. When the metabolic rates for the three groups were plotted (Fig. 30) however, they did not differ consistently or significantly. It seems more likely that in the confined jacket with little cold water convection the trunkal skin remained warm enough to have less vasoconstriction. If so, the baroreceptor reflex may have made the heart rate greater than with

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[1]The UVIC SPECIAL described in Table 4 was subsequently modified and is now marketed as the UVIC THERMOFLOAT JACKET, a buoyant life preserver with thermal protection and other safety features.

Fig. 30. Oxygen Consumption with Different Thermal Protection.

Oxygen consumption as an index of metabolic rate during immersion at 8.8°C. Each line shows the mean of the same 5 subjects in three different types of buoyancy-thermal protection device: a kapok vest, a floater coat and the UVIC Special.

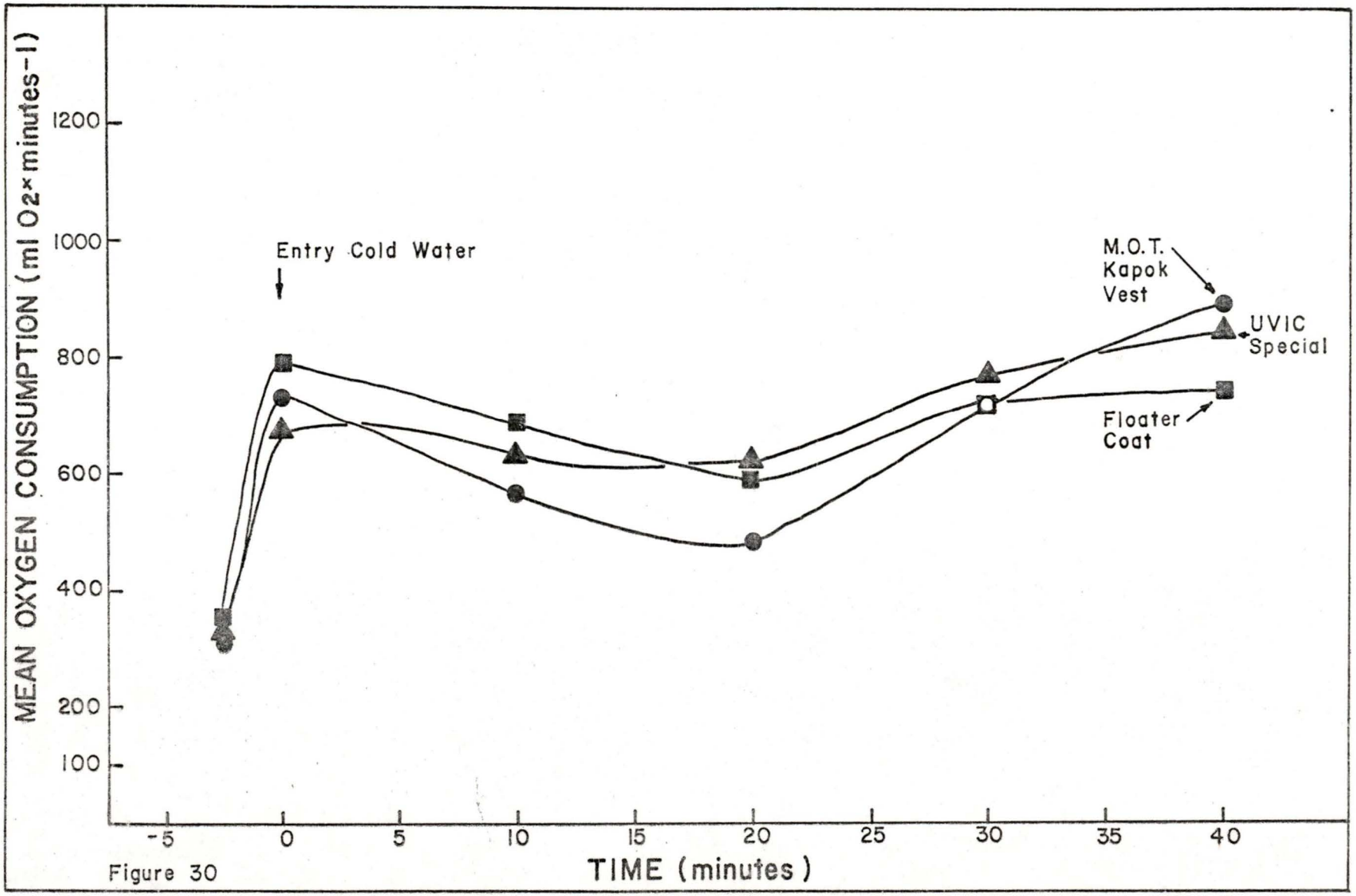
MEAN OXYGEN CONSUMPTION (ml O2 × minutes-1)
1200
1000
800
600
400
200
100
Entry Cold Water
M.O.T.
Kapok
Vest
UVIC
Special
Floater
Coat
-5
0
5
10
15
20
25
30
35
40
TIME (minutes)

Figure 30

less thermal protection. That is, this increase in heart rate was irrespective of metabolic rate.

Exercise

Exercise at thermoneutral ambient temperatures is well known to increase heart rate in meeting increased oxygen requirements in the skeletal muscles. There is no doubt that the same effect will be seen in water of lower ambient temperatures but we may expect added metabolic demands such as shivering so heart rate may be greater than that for a given activity in warm water. Costill (27) found that sub maximal and maximal work in water between 17.4 - 33.1°C did not require any increased oxygen consumption or heart rate to balance increased heat loss. In this study at 10.5°C, however, Hayward et al. (46) have shown that heat production in subjects swimming at 30 strokes per minute falls behind heat loss and shivering results. Evidence of this added metabolic demand can be seen if we compare our heart rates and oxygen consumption values in Series 2 (10.5°C) with those of Craig and Dvorak at 26°C (30). Their subjects cycled under water with a "heavy" workload and lifted arm weights for one hour. If that workload is considered roughly equivalent to the one in this study, the difference in metabolic rate and heart rate would be due to shivering and adrenergic stimulation. In Series 2 the mean oxygen consumption (1575 ml O_2/min. at 45 minutes of immersion) was about 1.7 times theirs (920 ml O_2/min. at 60 minutes of immersion) and the mean increase in heart rate from resting levels (36 bpm) was 1.7 times theirs (20 bpm) at the same times. Activity in cold water seems to require a higher metabolic rate and a higher heart rate than activity in warmer water.

In comparing still subjects to swimming subjects at 10.5°C there are three factors to consider. As above, exercise per se will increase the

heart rate. Secondly, cooling rate will be greater because of increased periferal blood flow, increased skin exposure and more convection of water through the clothing (24, 46). Finally, there will be a greater circulatory volume. The first and last factors will obviously increase the heart rate in exercising subjects. If the greater cooling rate acts as before we would expect to see a superimposed increasing heart rate occurring earlier in the active group. This was not seen (Fig. 12) and must indicate that both shivering and exercise oxygen requirements were being met by the already rapid heart rate. Certainly the rates were well below predicted maximal heart rates for the group (7). During the rewarming period the heart rates for the active group were greater than the still group, reflecting their lower rectal temperature and greater heat production in this period.

The correlation of heart rate and oxygen consumption is widely used to estimate the latter from the former in fitness evaluation (7). Their lack of correlation in swimming subjects in this study is likely because sympathetic tone and blood pressure varied during the cold immersion producing heart rate fluctuations unrelated to oxygen consumption.

The importance of exercise during cold water immersion is not in the heart rate response <u>per se</u>, it is in the finding that cooling rate is accelerated 35% by activity (46). The heart rate simply reflects increased metabolic demands and an increased circulatory volume.

Sex and Body Size

No conclusive results were obtained when comparing male and female groups in Series 2, because of a possible bias with older, more experienced, male subjects. It seems probably that with larger, unbiased samples a similar pattern of consistently higher heart rates in the female group

would be found. There has been a close relationship between heart rate and metabolic rate once about 0.5°C of cooling occurs. Females have a greater surface to volume ratio on the basis of body size (40), they cooled more than males in the same time and therefore females would be expected to have higher metabolic and heart rates.

The comparison of body size with the sex variable eliminated was also invalidated by small and biased samples as presented in the results of that comparison. The major factor in sex-related differences in response is almost certainly body size and more work is required to clarify this relationship.

EVALUATION OF COLD STRESS

The stresses involved in cold water immersion did not result in serious cardiovascular consequences in the young healthy population in this study. Most data were obtained in as close to a real "man overboard" situation as possible and are useful in evaluating possible dangers to the individual in such a position. Some limitations must be kept in mind. The population was young (mean age about 26 years), most subjects were physically fit and all were healthy on the basis of a physical examination and exercise stress test. A few subjects were immersed repeatedly and this may produce an acclimatization effect (64, 4). Accidents at sea often occur at nighttime and in storm conditions which may considerably alter responses through enhanced sympathetic nervous activity. Finally, the subjects were immersed for a relatively shorter period of time (usually less than 60 minutes) than that required for most rescue craft to retrieve shipwreck victims. Accordingly, the lowest rectal temperature recorded here was 32.9°C and most subjects declined about 3.0°C from normal rectal temperatures.

Cause of Death

The exact cause of death in cold water immersion in man is not clear, nor is there a known lethal mean body temperature at which it occurs (4). The same authors upon reviewing the literature note that temperatures approaching 0°C are compatible with resuscitation if tissue perfusion is adequate. This points to the most common cause of death, Central Nervous System anoxia due to cardio-respiratory failure. They estimate that half of humans will die before rectal temperature has reached 27°C. This disagrees with Molnar (72) who estimated from military reports of shipwreck that 8% will die between temperatures of 29.5°C and 32.2°C, 31% by 26.6°C and 88% by 23.9°C. Roughly in the same temperature range, the Dachau prison experiments (1, 72) found ventricular fibrillation or asystole to be the most common causes of death, usually by temperatures of 24 - 25.7°C. Prec et al. (83) also found ventricular fibrillation to cause death in most dogs but two dogs died with temperatures of 34 and 35°C and heart rates greater than 90 bpm. They had massive decreases in periferal resistance and blood pressure followed by respiratory arrest. Keatinge and Evans (64) postulated that death need not be due to hypothermia but can occur upon immersion, triggering ventricular fibrillation. It seems, therefore, that cardiac arrhythmias, especially ventricular fibrillation and asystole are the frequent cause of death in cold water immersion either as a result of initial exposure or of cooling the myocardium below 32°C.

Stresses in This Study

Let us evaluate in more detail the stresses experienced by subjects in the present study. Cardiovascular stresses of sudden cold water immersion can be classified into two broad categories: Non-Hypothermic, including

anxiety and immediate responses to cold exposure; and Hypothermic, including altered metabolism, increased myocardial irritability, acid-base and electrolyte changes, and vasomotor-blood pressure problems.

Non-Hypothermic Stresses

The non-hypothermic stresses were mild. Anxiety accompanies the apprehension of any painful experience and humans encounter sympathetic storms regularly in their lives. Premature atrial and ventricular contractions are experienced in a large portion of the general population in a given 24 hour period (98). They may be precipitated by ethanol or coffee ingestion, exercise or emotional distress (81). Single infrequent extrasystoles are usually benign but at rates of greater than five per minute, coupled, occurring on T wave, or from more than one ventricular focus they indicate a very irritable myocardium. Ventricular tachycardia or fibrillation can rapidly follow. During the pre-immersion period mean heart rates did not exceed 90 bpm and no extrasystoles were observed.

The heart rates upon immersion (mean 135 bpm, maximum 150 bpm) were not themselves dangerous compared with heart rates achieved in older persons by climbing stairs, bicycling or walking briskly. They were well below predicted maximal heart rates for all humans (7). There are, however, other factors increasing the risks of these rates. There is a sudden increase in systemic blood pressure as cardiac output and periferal resistance both increase. Significant catecholamine release occurs (60) which can sensitize the myocardium. Both of these effects occur much more rapidly than during exercise and even then they may precipitate stroke or arrhythmia in individuals with hypertension or atherosclerosis.

In the first two minutes of immersion at 15.0°C Keatinge and Evans (64) reported frequent premature ventricular contractions (PVC's) which were

multifocal in one subject. They also found ocasional extrasystoles within 15 minutes of immersions at 25 and 35°C. Similarly, but in only one subject, we observed 2 unifocal PVC's separated by 9 beats in the first minute of immersion. It seems that these were innocent occurrences perhaps attributable to anxiety. Keatinge however, considers that arrhythmias, elevated venous filling pressure and hyperventilation is evidence that occasional individuals may develop ventricular fibrillation upon immersion in cold water (65).

Hypothermic Stresses

Altered Metabolism

During the hypothermic period the heart is affected by the direct action of cooling and by more generalized metabolic changes in the body. The magnitude of some of these changes is insignificant in mild (not ∠ 30°C) hypothermia simply because the body is functioning almost as it does normally. For example, shivering thermogenesis shown here is not unlike mild exercise and it results in an increased heart rate and cardiac output, heat production and blood pressure (31). The myocardial oxygen consumption is undoubtedly increased and in a person with sufficiently compromised coronary vessels, it would be possible to get angina pectoris as in exercise.

It is useful to briefly consider some of the changes in cardiac function during deep or profound hypothermia. Myocardial oxygen consumption is decreased during cooling. Coronary perfusion increases progressively due to this and to cold induced dilatation (16, 15). Contractility is improved although the velocity is decreased, prolonging systole. Diastole is also increased giving an increased stroke volume, but with the progressively decreasing heart rate the net effect is a decreased cardiac output (82).

Myocardial tissue injury has been shown to occur by MacLean et al. (69). They found elevated myocardial and skeletal muscle enzyme levels in the blood of hypothermic patients at rectal temperatures as high as 34.9°C. These levels were found in accidentally hypothermic patients who had other conditions or injuries that make interpretation difficult (e.g. bruises, myocardial infarction) (42). This is likely not a major concern in the present subjects although some tissue injury likely occurred in subjects who cooled below 34.0°C. There were no ECG changes suggestive of myocardial infarction but the ECG did reflect some of the metabolic changes of deep hypothermia. Bradycardia due directly to cooling was mentioned earlier. Cooling also slows conduction pathways (31, 44), but no abnormal prolongation of PR interval or of QRS duration was found. There was a significantly increased QT_c interval which is roughly equivalent to the time of depolarization plus repolarization of the ventricles. The QRS duration was normal, indicating that repolarization was most affected, in accordance with the findings of Prec et al. (83) and Ruskin and Decherd (89). This is likely due to slower sodium-potassium pump activity (82). A prolonged QT_c can be due to hypocalcemia (42), but this is not the case in hypothermia. Some ST-T segment changes, which usually occur later than QT_c changes (83) were observed. Although the marked ST depression in Fig. 23 is highly suggestive of ischemia, this subject was very active athletically and there was no clinical evidence of ischemia. His rectal temperature was 32.9°C and one would expect improved coronary perfusion (16, 15). The likely explanation is altered repolarization due to depressed metabolism at the cell membrane level, rather than ischemia. Acute myocardial infarction can cause ST segment

elevation, but a more likely cause in the three subjects who developed this finding is physiological ST elevation. This is common in young, athletic or anxious persons (87). The J point deflection was seen in two subjects. This wave has been attributed to acidotic myocardial injury (78), atrial repolarization (91), myocardial hypoxia or conduction defect (35), but the exact cause is obscure. More recently, Kernohan (66) suggested that it is due to imbalanced electrical activity at the end of depolarization due to myocardial cooling. Johansson (59) implicates alteration of the relationship between depolarization and repolarization. The suggestion of Emslie-Smith et al. (35) that more than one lead must be monitored to find the J wave was borne out in our results (Fig. 25). Since we usually monitored one lead, the prevalence of this wave in mild hypothermia cannot be assessed.

Acid/Base and Electrolyte Changes

The marked increase in T wave amplitude resembled the pattern of hyperkalemia particularly as it was progressive (42). Acidosis is a recognized complication of hypothermia (78, 59, 82, 68) due to hypoventilation and anaerobic metabolism. It seems possible that acidosis from shivering could displace sufficient intracellular potassium ions to give the hyperkalemic changes in the ECG. A serum potassium level of greater than 7.0 meq. per 1. (normal 3.5-5.0) can give peaked tall T waves (42). The cardiac complications of hyperkalemia (fibrillation or asystole), cannot be reliably predicted from T wave amplitude or serum levels and these are thus poor warning signs (42). The reversion of the T wave below pre-immersion height was rapid after shivering subsided in the rewarming tub. This suggests an abrupt change in either serum or local potassium levels (63) perhaps as pH returns to normal, allowing potassium to shift

intracellularly. Renal excretion of potassium, which rapidly follows hyperkalemia (9) also plays a role here. Cupples has shown increased potassium excretion during cold water immersion in this laboratory (32). Water immersion per se does not affect excretion of potassium. Epstein and Saruta (36) found no change in serum potassium in men after six hours of immersion in 34.0°C water.

Myocardial Irritability

Evidence of increased myocardial irritability was rarely seen during the hypothermic period. One subject had frequent atrial extrasystoles and two others had isolated ones, but these are considered benign arrhythmias (42). Ventricular bigeminy was seen momentarily in one subject after 0.7°C of cooling. This unifocal extrasystole also was without complication and would be unlikely to progress to a serious arrhythmia. The three most dangerous arrhythmias, ventricular tachycardia, fibrillation and asystole were not seen. One reason for the absence of gross signs of irritability is that the ventricular refractory period is prolonged in hypothermia (82). Of concern is the finding of hyperkalemic T wave changes, because this electrolyte disturbance is associated with the fatal arrythmias.

Hypotension in Rewarming

These were discussed as a stress with the rewarming period.

In general the stresses of the cold water immersions studied were of no consequence to the safety of the subjects. As mentioned above, however, these results were not obtained in an actual shipwreck situation, the subjects were young and healthy, and the hypothermia was mild. In a shipwreck non-hypothermic stresses are likely to be greater. The hypothermia is likely to be deeper depending upon rescue time and rewarming techniques. Many persons thrown overboard each year are in the middle age or older

population which has a statistically higher rate of hypertension, systemic atherosclerosis and coronary disease. Keatinge (65) points out that such individuals are more prone to stroke, myocardial infarction or fatal arrhythmias. These can be fatal immediately or lead to drowning. Gunton (45) notes that in hypothermic surgery, patients who remained in sinus rhythm tended to be younger than those who developed atrial fibrillation. Keatinge further recommends repeated, slow immersion in cold water under supervision for those at risk of accidental immersion, in the belief that habituation will decrease the stresses involved.

CONCLUSIONS

The pattern of the heart rate and electrocardiographic response to cold water immersion, mild hypothermia and rewarming in conscious humans was described and discussed. Treatment effects studied were ambient temperature, exercise, thermal protection, sex and body size. The following conclusions can be drawn:

1. The heart rate response in conscious man exposed to cold water differed from that in anaesthetized and deeply hypothermic semi-conscious subjects because of intact central and periferal nervous function.

2. The cold induced thermogenic response is accompanied by an increased heart rate when ambient temperature is below 10.5°C. At 18.2°C this is not so.

3. The stresses of cold water immersion in the sample studied do not result in any side effects. The ECG findings do not show any significant arrhythmias. However, indirect ECG evidence of increased serum potassium levels was found during cooling and this disturbance can lead to fatal arrhythmias.

Some evidence of hypotension and venous stasis is seen during the rewarming period after shivering subsides.

4. An increased risk of significant cardiovascular effects could be expected for persons outside the limitations of this study. That is, anxious persons with cardiovascular disease, no life jacket or thermal protection, who are thrown overboard in bad weather for a prolonged time would be at the highest risk.

5. Many aspects of the cardiovascular response to cold water immersion still require investigation. Some suggestions are:

a) ECG leads II and V_5 atleast should be monitored.

b) Biochemical monitoring should include:

(i) urinary catecholamines

(ii) serum potassium levels

(iii) arterial blood gases and pH.

c) Blood pressure (indirect) should be monitored, particularly during the rewarming period.

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APPENDIX

Minute Heart Rate Variates

Fig. 31. Heart Rates at Different Water Temperatures and Activities. Shown are plots of the minute variates of heart rate of 12 subjects immersed while Still (water temperatures of 4.6°C, 10.5°C and 18.2°C) and Swimming (water temperature 10.5°C). The variates are means of 11 or 12 subjects in Series 1, 2, 3 and 4 although not all the same subjects were used in each series.

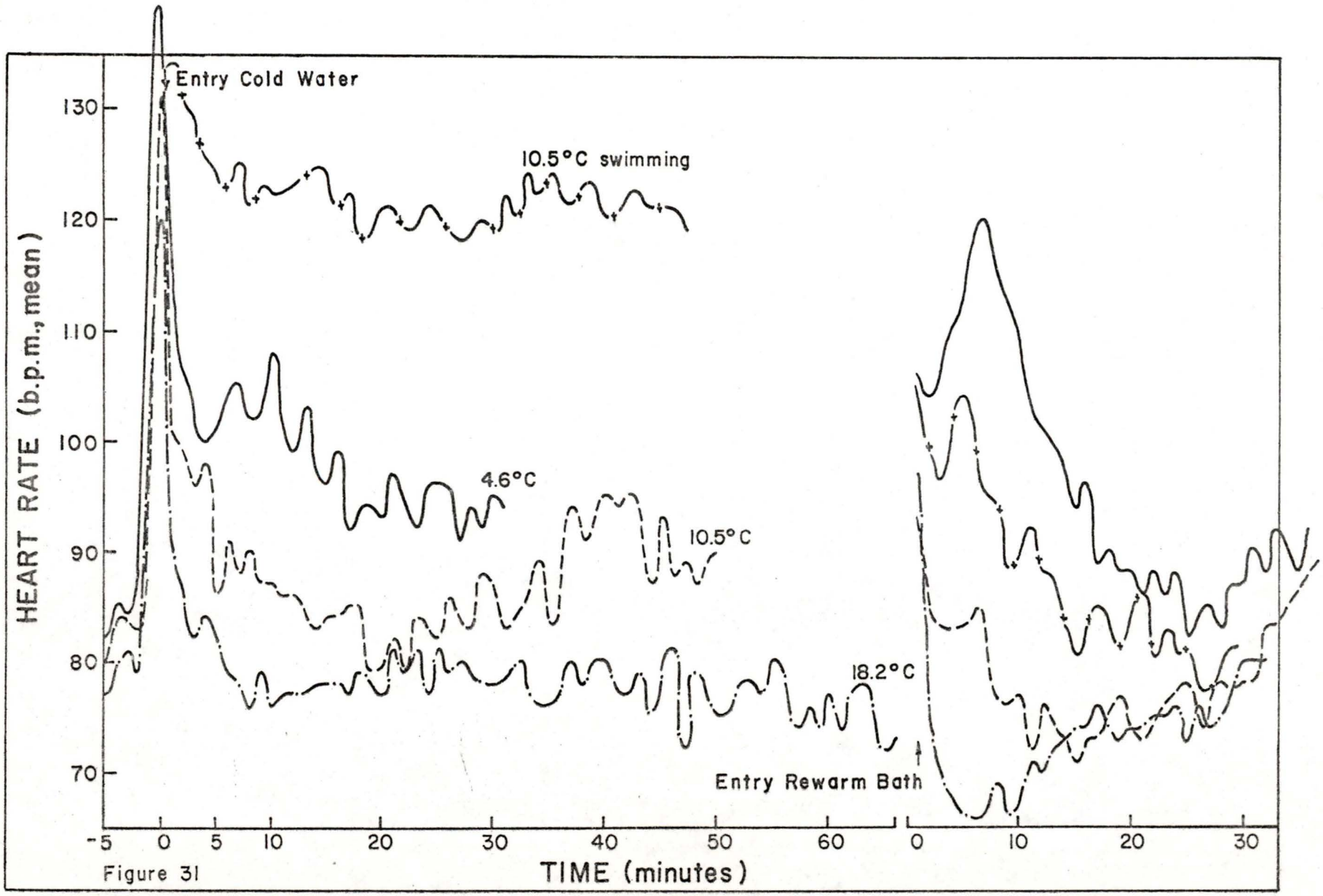
Entry Cold Water
10.5°C swimming
4.6°C
10.5°C
18.2°C
Entry Rewarm Bath
HEART RATE (b.p.m., mean)
130
120
110
100
90
80
70
-5
0
5
10
20
30
40
50
60
0
10
20
30
TIME (minutes)

Figure 31

VITA

Surname: McKay Given Names: William Ross

Place of Birth: Nelson, B.C. Date of Birth: April 6, 1948

Educational Institutions Attended, with Dates of Entering and Leaving:

NOTRE DAME UNIVERSITY, NELSON	1966	to	1967
UNIVERSITY OF VICTORIA, VICTORIA	1967	to	1977
UNIVERSITY OF BRITISH COLUMBIA, VANCOUVER	1974	to	PRESENT

Degrees, Diplomas, Etc., Awarded, with Dates and Names of Institutions:

B.Sc.	1972	University of Victoria, B.C.

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