EFFECTS OF POSTURE AND STATIC EXERCISE ON HEAT DISSIPATING MECHANISMS IN HUMANS.

by

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THESIS SUBMITTED IN PARTIAL FULFILLMENT OF
THE REQUIREMENTS FOR THE DEGREE OF
DOCTOR OF PHILOSOPHY
in the School
of
Kinesiology.

SIMON FRASER UNIVERSITY

February 1990

APPROVAL

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Degree: Doctor of Philosophy

Title of Thesis:

EFFECTS OF POSTURE AND STATIC EXERCISE
ON HEAT DISSIPATING MECHANISMS IN HUMANS

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EFFECTS OF POSTURE AND STATIC EXERCISE ON HEAT DISSIPATING MECHANISMS IN HUMANS

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APRIL 5TH, 1990

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ABSTRACT

The activity of baroreceptors on convective and evaporative heat loss was investigated by requesting heat stressed subjects to recover idle or performing static exercise in either the supine or the upright posture.

Forearm skin blood flow ($\Delta F_{sk}BF$, $ml \cdot 100g^{-1} \cdot min^{-1}$) fell more ($P \leq 0.04$) in upright than in supine recovery. S_wr ($mg \cdot sec^{-1} \cdot m^{-2}$) changed less over a given range of T_{es} (^oC) and the point of its extinction was delayed ($P \leq 0.001$) in the upright compared with the supine position.

The forearm skin blood flow ($\Delta F_{sk}BF$) was higher ($P \leq 0.02$) during idle recovery than during static exercise recovery at any given T_{es}. The subjects sweated considerably more ($P \leq 0.03$) during the last 24 minutes of recovery with static exercise than while idle. For a given T_{es}, the T_{es}-S_wr relationship showed a trend ($P \leq 0.05$) of a higher slope during static exercise than while idle, whereas the cessation of the sweating response was similar.

A lower EDI ($ml \cdot kg^{-1}$, $P \leq 0.05$) and greater MAP (mm Hg, $P \leq 0.02$) and a concominant lower $F_{sk}BF$ and S_wr for a range of T_{es} measures in the upright versus the supine recovery position suggests baroreceptors may modify the thermoregulatory response. The lower $F_{sk}BF$ during recovery in the upright than in the supine position may be attributed to a skin vasoconstrictive reflex originating from the cardiopulmonary baroreceptors. Cardiopulmonary baroreflex action might also have provoked the S_wr changes observed between the upright and supine position.

Compared with the idle condition, there was a tendency for S_wr to be higher at a given T_{es} in the static exercise condition. Because it was found that the baroreceptors were unloaded in the upright compared with supine static exercise condition whereas S_wr was similar, it may be suggested that the higher S_wr during static exercise than during the idle recovery was not

caused by the unloading of the cardiopulmonary and arterial baroreceptors. It appears that the greater $S_w r$ during static exercise than that observed while idle may be ascribed to a reflex originating from skeletal muscle (myogenic reflex). An interaction of thermal, cardiac and myogenic reflexes is the most likely explanation of the observed F_{sk}BFresponse.

DEDICATION

TO MY PARENTS, WHO, ALTHOUGH OF HUMBLE ORIGIN AND POSSESSION,
NOURISHED ME WITH LIMITLESS MORAL SUPPORT AND AFFECTION.

QUOTATION

"THE HIGHER THE CONSIOUSNESS IN PERFORMING RESEARCH
THE HIGHER THE SKEPTICISM ABOUT THE WAY OF DISCUSSING
THE RESULTS"

ACKNOWLEDGMENTS

I wish to express my warm appreciation to professor V. Klissouras, who proselytized me to the research doctrine, and for the paternal assistance he offered to me in my preparatory endeavor to pursue graduate studies.

The completion of this thesis would not have been possible without the the support of many people.

I wish to express my genuine appreciation to Dr. J.M. Johnson for his kindness in reading my dissertation and his willingness to evaluate my dissertation defence.

I am indebted to my indefatigable senior supervisor, Dr. E.W. Banister, for his financial assistance, and his stern, but fair, treatment. I also owe special thanks to him for his trust in my capabilities during my initial rough period of initiation to the North American culture, language, and educational system.

I also wish to express my sincere gratitude to Dr. I.B. Mekjavić and Dr. O. Eiken for their "sweaty" friendship, constructive criticism, and academic assistance which was particularly crucial in the formulation and evalution, respectively, of the present piece of research.

Collection and analysis of data were expedited with the assistance of Gavril Morariu Dipl. Ing., Reo Audette, Vic Stobbs, Mel Frank, Cristine Moric, Sue Fairburn, Wendy Barker, and Maria Koskolou. I am thankful to all of them. I am, also, indebted to my lab mates Pat Sullivan M. Sc., Wendy Burke M.Sc, Mat White M.Sc., and Thanasis Passias for their cerebral and practical assistance whenever it was needed.

Moreover, my heartfelt thanks to Dr. Parveen Bawa for her assistance, friendship and costly encourangment to complete my graduate studies within the eighties. I am, also, grateful to all volunteers who participated in this study and Simon Fraser University for the financial support provided throughout my tenure as a graduate student.

Last and least, I wish to express my thanks to all the individuals and situations which, by abusing graduate students, enhanced my stamina and desire to conclude my studies quickly and sucessfully.

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1.0 INTRODUCTION

*Thermoregulation as a function of coincident thermal factors.*The set-point theory of human temperature regulation suggests that the body core temperature (T_c) of humans is maintained by the counteracting processes of metabolic heat production and heat loss which respond to core and skin temperature displacement. The threshold core temperature for the onset of shivering and sweating do not coincide, as was proposed by Cabanac and Massonnet (1977), but rather they establish a set-zone (Mekjavić and Bligh, 1989). The intensity of any thermoregulatory function may be plotted against core temperature and defined as the slope or gain of the relationship. The core threshold temperature for the onset of sweating and the rate of its increase is dependent upon the level of both core and skin temperature ($\bar{T}_{sk}$) (Hammel et al. 1963, Wyss et al. 1974, Johnson et al. 1984a) and their respective time derivatives (Wurster and McCook 1969, Libert et al. 1978). Thermal inputs from the skin and the core are well established determining factors eliciting thermoregulatory responses whereas the role of other possible factors invoking a thermoregulatory response remains to be established.

The effect of baroreflex on heat dissipating mechanisms. Numerous investigations have pointed out that the link between the sweat rate response (S_wr) is inadequately related to the thermal input originating from different central and peripheral sites of human body (Nielsen 1966, Saltin and Hermansen 1966, Smiles and Robinson 1971).

It is known that thermoregulatory function is closely related to function of the cardiovascular system (for ref. see Hales 1984). It has been shown, for instance, that forearm vascular conductance decreases more in response to a challenge to blood pressure regulation during heat stress than

under normothermic conditions (Johnson et al. 1973). However, information regarding the effect of changes in systemic arterial pressure and/or central blood volume on the sweating response remain sparse. The possible effect of baroreceptors on the sweating response (S_wr) during hyperthermia has only been considered indirectly in a few studies in subjects assuming different postures (Greenleaf et al. 1971, Kobayashi et al. 1980).

Greenleaf et al. (1971) found that in subjects performing submaximal moderate dynamic exercise in a warm environment, rectal temperature (T_{re}) was stabilised at a lower level when exercise was conducted in the supine position rather than in the upright posture. The lower T_{re} in the supine position was ascribed to an increase of non-evaporative heat loss due to elevated $\bar{T}_{sk}$ and skin blood flow as was shown previously by Stenberg et al. (1967). Total sweat loss was essentially the same in both postures. However, if the onset of sweating had occurred earlier and its increment had been slower in the upright position, for instance, compared with the supine position, total sweat loss would still be the same in both positions but due to quite different thermoregulatory processes.

Kobayashi et al. (1980) in a similar set of experiments as was performed by Greenleaf et al. (1971) found that the steady state T_{re} in the supine position was higher than in the upright position despite the fact that sweat, evaporated from the skin, was calculated to be 18% greater in the upright than in the supine position. It should be noted, however, that in the experiments of Kobayashi et al. (1980), the endogenous heat storage was probably higher (~10%) during exercise in the supine position than in the upright position since the absolute work rate was identical in both positions. It has been shown that exercise capacity is significantly higher in the upright than supine position (Åstrand and Saltin 1961, Eiken 1988) and that T_{re}

during exercise is better related to the relative rather than to the absolute work rate (Saltin and Hermansen 1966). Thus, it appears from the results of the original studies of Greenleaf et al. (1971) and Kobayashi et al. (1980) that evidence regarding a possible role of arterial baroreceptors in temperature regulation during exercise remains inconclusive .

The possible role of unloading cardiopulmonary and arterial baroreceptors on S_wr response has been investigated by Solack et al. (1985). These investigators exposed 6 healthy men to lower body sub-atmospheric pressure (LBSP) in the supine position after increasing their esophageal temperature (T_{es}) to a mean of 37.2 °C and at least doubling their S_wr by external heating through the use of a water-perfused suit at 38 °C. It was found that the S_wr-T_{es} slope decreased in 4 subjects and increased in 2 during LBSP. Because the S_wr-T_{es} slope was not re-established at its pre-LBSP level in the 4 subjects when LBSP was discontinued Solack et al. suggested that S_wr was not affected by the low-pressure baroreflex. However, it may be argued that results by Solack et al. (1985) do not conclusively exclude a possible effect on S_wr response of unloading low pressure baroreceptor first, because the S_wr-T_{es} range explored was very narrow and secondly, T_{es} was increasing throughout the LBSP application. This increase in T_{es} might have had a stimulatory thermoregulatory drive on S_wr masking any concomitant inhibitory input to sweating induced by unloading the cardiopulmonary and /or arterial baroreceptors.

Baroreflex stimulation by static exercise, and heat loss mechanisms.

In addition to the fact that identical combinations of peripheral and central temperatures produce widely differing S_wr responses during dynamic exercise (Saltin and Hermansen 1966), acute static arm exercise elicits marked whole body sweating even when warm venous blood from the

muscle is prevented from reaching the central thermoregulatory centers by occlusion of the circulation in the arms (Beaumont and Bullard 1966). The mechanism is unexplained but the possibility of baroreflex mediation might be considered, since muscular contraction increases MAP (Gillespie 1924, Helfant et al. 1971).

The increase in MAP during a static contraction appears to be caused mainly by a dramatic increase in HR and cardiac output since systemic vascular resistance remains unchanged or is slightly increased (for ref. see Lind 1970). The cardiac output increase during a MVC manoeuvre may amount to about 1 liter·min^{-1} (Lind 1983). It has also been shown that this excess cardiac output is not distributed to the kidney, splanchnic vascular bed or to inactive muscles (Lind et al. 1964, Lind et al. 1981). Moreover, during a static contraction, oxygen consumption increases disproportionately to cardiac output (Lind et al. 1964).

It is possible, therefore that the excess cardiac output may be used for thermoregulatory purposes. Kilbom and Brundin (1976) found that skin temperature increased during static contraction. Furthermore, a recent study showed that during static handgrip at a local skin temperature of 39 °C skin blood flow increased (Taylor et al. 1989).

Conversely, it has been found that static exercise in anesthetized animals is associated with vasoconstriction when carotid sinuses were kept at constant pressure and the vagi cut (Clement and Pannier 1980). Exercise also causes skin venoconstriction (Bevegård and Shepherd 1966, Seaman et al. 1973) which may gradually subside (Lorentsen 1975).

Thus, the literature on the effect of static exercise on skin blood flow and sweat rate and its contribution to body temperature regulation remains limited and rather inconclusive. It may be observed that cardiovascular

adjustments to static muscular exercise may serve the cause of temperature regulation rather more than the energetic requirement considering that a great amount of energy produced by isometrically contracting muscle is degraded to heat. While the effect of skin blood flow and sweat rate on temperature regulation during static exercise is quantifiable, an explanation of the underlying mechanism(s) poses a major problem.

The cardiovascular response to a static contraction is clearly elicited by a reflex action from the active muscles (myogenic reflex) although input from higher cerebral centers may be of some significance (for review see Lind 1983). Data support the action of afferents, in groups III and IV with unknown specific modalities, as effective in mobilising the exercise-induced cardiovascular response (McCloskey and Mitchell 1972). Jessen et al. (1983) recently demonstrated that there is neural afferent transmission of a temperature signal from skeletal muscle in the goat to the temperature regulating centers. This was attributed to sensitivity of afferent group III and IV fibres.

Thus, it seems conceivable that muscular exercise may provide input to the thermoregulatory centers directly either via muscle afferent stimulation and/or indirectly via baroreceptor stimulation.

Purpose of the current work. Despite extensive research into the control and regulation of sweating and skin blood flow, the contribution of a myogenic reflex, and a baroreflex *per se,* as well as their avenues of interaction (i.e. neuronal, humoral) with components of the thermoregulatory control system, remain unresolved and deserve further investigation. Furthermore, the interaction of arterial blood pressure and thermoregulatory effector mechanisms has significance in our understanding of basic exercise and environmental physiology and has practical relevance to

normotensive and hypertensive individuals exposed to extremes of ambient temperature.

Therefore, the present study investigated the effect of the baroreflex on heat dissipating mechanisms. After raising the internal body temperature with moderate dynamic exercise, the arterial blood pressure of 8 healthy subjects was manipulated first by requesting each subject to recover either in the upright or in the supine position. Secondly, the effect of the interaction of the baroreflex with the myogenic reflex on temperature regulation was studied on a separate occasion by imposing mild static exercise during the recovery phase in both the upright and supine positions.

The specific aims of this study were to investigate whether:

1. Posture modifies sweat rate and skin blood flow by effecting cardiopulmonary and arterial baroreceptors.
2. Increased muscular activity modifies sweat rate and skin blood flow.
3. Interaction of the baro- and myogenic reflexes affects sweat rate and skin blood flow.

2.0 METHODS

Subjects. Experiments were conducted on eight healthy males selected from volunteers in the age range of 22-29 yr. Their physical characteristics are given in Table 1. Sum of skinfolds was determined from measurements at the triceps, biceps, subscapular, suprailiac, thigh and calf sites (Ross and Marfell-Jones, 1983). Body surface area was estimated according to the height - weight formula of DuBois and DuBois (1916).

Table 1: Subjects physical characteristics, maximal aerobic power ($\dot{V}_{O_{2max}}$), length of esophageal probe insertion, and Maximal Voluntary Contraction (MVC) in the supine and upright position.

Subjects (initials)	AGE (year)	Weight (kg)	Height (cm)	Surface Area (m^2)	Sitting height (cm)	Esopha probe (cm)	Skinfold Sum (mm)	V_{O2max} (ml/kg $\cdot min^{-1}$)	MVC supine (N)	MVC upright (N)
C.W.	22	57.8	170.3	1.68	90.6	39	46.9	64.36	594.6	424.0
T.P.	25	68	171.5	1.8	93	40.1	43.75	54.12	1191.9	1106.6
P.S.	27	84	189.3	2.13	96.6	41.8	64.3	46.07	1149.7	1106.6
J.FC.	29	72.5	184.1	1.96	96.5	41.8	56.25	49.79	722.6	850.6
L.H.	22	65	171.4	1.76	93.2	40.2	32.5	57.85	765.3	850.6
A.RR.	23	76.5	184.1	2.01	97.2	42.1	43.25	67.45	1191.9	594.6
R.Q.	25	73.6	174.9	1.89	96.2	41.6	59	56.11	1618.7	1191.9
V.L.	22	64.5	184	1.88	95.8	41.4	44.25	55.97	765.3	679.9
Mean	24.4	70.24	178.7	1.89	94.89	41.0	48.77	56.46	1000.0	850.6
± S.E.	0.29	0.90	0.82	0.015	0.257	0.121	1.138	0.772	40.3	32.0

Protocol. Subjects were told to refrain from eating a large meal at least 3.5 hours prior to reporting to the laboratory. In a preliminary study before the trials, subjects bilateral maximal voluntary contraction force (MVC) of the biceps brachialis muscle was determined in both the upright and supine position by pulling a handle permanently attached by a chain to a

dynamometer. Elbows were flexed at a 135° angle and humerus was laterally adducted to the thorax in both positions. The subjects assumed a semisquat sitting position (110° -120° angle of the knee) with feet parallel and back straight as the MVC was attempted. The MVC in the supine position was determined as a subject's legs were bent at the knee at 110° -120° angle. In addition, each subject completed an incremental load test to volitional limit on a Uniwork Quinton-845 electrically braked ergometer in order to determine their maximal aerobic power.

Upon arrival in the laboratory for the experimental treatment subjects were requested to drink a cup of water to ensure euhydration. Sensors were attached to the subjects, who wore only shorts, in order to monitor the several physiological parameters described below. Once instrumented, they remained seated for 10 minutes in a thermoneutral environment to ensure that all sensors and data acquisition equipment were functioning properly and a resting value of each variable was recorded. Following the resting period, each subject exercised on a cycle ergometer for 25 minutes at a work rate equivalent to 50% of their maximal work rate, previously determined from the incremental load exercise test to exhaustion. Upon completion of the exercise period subjects were covered with a cotton sheet in order to ensure consistent skin temperature between experimental conditions,and rested until either cessation of induced sweating occurred or esophageal temperature approached pre-trial values. Physiological measurements were continued during this recovery period which lasted for at least 33 minutes. Each subject performed the aforementioned protocol on four separate experimental occasions. Each trial was conducted in a thermoneutral environment (22±0.6 °C, 42±8% RH) at the same time of day in order to avoid any diurnal influence on temperature changes. On the first two occasions subjects

recovered either in an upright or in a supine position. In the upright position, subjects were supported by either a stool or the cycle's seat. On two further occasions subjects performed low intensity sustained static bilateral elbow flexions during recovery in both positions. The intensity of the static exercise was equal to 160±6.1 N in both the supine and upright position and corresponded to 16%, and 19% of MVC respectively. These intensities were selected in order to ensure that the cardiovascular response to static exercise would reach steady state within a few minutes (Lind 1983), with minimal fatigue and blood flow occlusion in the contracting muscles (Barcroft and Millen 1939). During each static exercise trial subjects were repeatedly instructed to follow unrestricted respiration in order to prevent any pressor side effect due to Valsava manoeuvre.

Instruments and techniques.

Mean skin temperature ($\bar{T}_s$, °C). Mean skin temperature was measured using Thermonetics (San Diego,California) Heat Flux Transducers (model HA 13-18-10P(C)) with thermistors embedded in their measuring surface. Each transducer was positioned and taped on the skin surface. The transducers were calibrated using a copper encased water bath as previouly described by Mittleman (1987) and were attached on the arm, thigh, and beneath the scapula. An unweighted mean skin temperature was calculated from the individual sites monitored.

Internal *body temperature* was monitored in the *esophagus (T_{es}, °C).* An esophageal thermistor probe (Yellow Spring Instruments 701, Ohio) was inserted through a nostril to a depth proportional to a subject's sitting height, as shown in Table 1, (Mekjavić and Rempell, in press). All the thermistors,

were previously calibrated against a standard thermometer in a bath of stirred water over a wide range of temperatures (20-40 ±0.1 °C).

Sweat rate ($S_wr, mg \cdot sec^{-1} \cdot m^{-2}$). Sweat rate was monitored with a 3.33 cm^2 sweat capsule positioned on the skin surface of the forearm. Air at room temperature and a relative humidity of 20% was passed through the capsule at a rate of 531 $ml \cdot min^{-1}$ and sampled for temperature and water content on the exhaust side. By calculating the difference in water content of the air entering and exiting the capsule, a value for sweat secreted* in units of $mg \cdot sec^{-1} \cdot m^{-2}$ was determined. Water vapor content of air entering and leaving the capsule was measured with Shinyei (Japan) relative humidity sensors. The humidity sensors were calibrated prior to the study by circulating humidified air through a drierite column ($CaSo_4$) placed in series with the sensors. Drierite weight changes were measured with a Mettler (AE 163, Switzerland) scale in the range of 0 - 30 g to an accuracy of 0.001 g.

Arterial pressure (mm Hg). Arterial pressure was monitored at the brachial artery every 5 minutes during the recovery phase by an automatically inflated digital sphygmomanometer (UA-251, Japan), which was previously acoustically checked for accuracy and reliability. Although, sphygmomanometrical determination of DAP have been shown to be unreliable at high work intensities (Kaijser 1987) the work rate in the present study was relatively low, which, justifies the use of the above method. Mean arterial blood pressure (MAP) was calculated according to the equation: MAP = (DAP + 0.33(SAP - DAP)), where DAP and SAP correspond to diastolic and systolic arterial pressure, respectively.

*It should be noted that the method of measuring sweating used in the present study actually detects a forced evaporation of sweat and not the natural sweat evaporation.

Forearm skin blood flow ($F_{sk}BF, ml \cdot 100g^{-1} \cdot min^{-1}$). Skin blood flow was derived from continuous measurements of skin red blood cell velocity with a BPM 403A TSI laser-Doppler (LDF) blood perfusion monitor (Johnson et al. 1984b, Saumet et al. 1988). LDF calibration factor provided by the manifacturer is not universally accepted (Holloway and Watkins 1977, Kvietys et al. 1985, Shepherd et al. 1987) The laser-Doppler sensor was positioned on the forearm of the subjects.

Heart rate (HR, beats·min^{-1}), Stroke volume (SV), Thoracic fluid index (TFI, Ohm) and *End diastolic index (EDI, ml·kg^{-1})* were measured from four pairs of silver / silver chloride electrodes (BOMED, California) attached laterally to the chest and the throat of the subjects as recommended by Sramek (1989). A constant current of 2.5 mA at 70 kHz was applied across the thorax through 4 electrodes. Voltage changes associated with ECG pulses and thoracic impedance variations were measured from the remaining electrodes. Electrodes were connected to an electrical bio-impedance monitor (NCCOM3-R7, BOMED, California). SV was calculated according to Sramek et al.'s equation (1983) by use of a micro-processor attached to the bio-impedance monitor . This technique is a modified approach based upon Kubicek's original method (Kubicek et al. 1966) of SV determination.As opposed to Kubicek formula, the Sramek's equation is not sensitive to movements nor to changes in blood resistivity (Quail et al. 1981, Shankar et al. 1985, Bernstein 1986). End diastolic index was the derivative of end diastolic volume divided by kg of body weight. End diastolic volume was calculated from the ratio of stroke volume to ejection fraction. Ejection fraction has been shown to be equal to 0.84 minus 0.64 of the pre-ejection period during the period of ventricular ejection time (Capan et al. 1987). TFI was an extrapolation of changes in thoracic electrical impedance.

Cardiac output ($l \cdot min^{-1}$) was derived from measurement of SV and HR. Cardiac output and stroke volume were calculated per kg of body weight and expressed as *cardiac (CI)* and *stroke index (SI)*, respectively.

*Vascular resistance.*Total peripheral resistance (*TPR*, *mm Hg·kg·min* $\cdot l^{-1}$) was computed from the ratio of MAP to CI. Skin vascular forearm conductance was calculated as $\Delta F_{sk}BF$ divided by MAP ($ml \cdot 100g^{-1} \cdot min^{-1} \cdot mm\ Hg^{-1}$).

Force generation *(N)* during static exercise was measured by a Daytronic model 300D (Ohio) dynamometer attached to a pulling handle. The strain gauge was previously calibrated with known weights to an accuracy of 0.1N.

Oxygen uptake ($L \cdot min^{-1}$) was continuously monitored. Mixed expired gas was analysed for oxygen and carbon dioxide content with an Applied Electrochemistry Oxygen Analyser (S-3A, California) and Godart Statham BV Capnograph (Model CG/58002, Holland), respectively. Analysers were calibrated with gases of known concentration before each experiment. Inspired volume was determined with an Alpha Technologies Ventilation Module (Model VMM110, California) which was calibrated prior to each experiment, with a syringe of 6.15 L capacity.

All analog signals, with the exception of sweat rate values, were collected on - line utilizing a Hewlett - Packard 3497A data acquisition system controlled by a Hewlett-Packard 9817 microcomputer. Humidity sensors were monitored with a separate Hewlett - Packard 3497A controlled by a Hewlett-Packard 85 computer.

3.0 ANALYSIS

Difference values. The $\bar{T}_{sk}$, T_{es}, S_{wr} and $F_{sk}BF$ variables were statistically compared at any point of time as the difference (Δ) between the first minute of recovery and the prevailing value (time = 2 to 33 min).

Two - way ANOVA design. Data from each dependent parameter were analyzed by a 2 factor (condition, position), 2 levels on each factor (idle - static exercise, upright - supine, respectively) analysis of variance (ANOVA) design with repeated measures (Dixon 1988) (Figure 1). The analysis was carried out for each minute value of the variables throughout 33 minutes, the latter being the common time all subjects sustained the different experimental conditions. Comparison of the overall column means (Fig. 1) i.e cell C vs. cell D addressed the first research question of whether changing posture, affected body temperature regulation. Similarly, comparison of the overall row means i.e. cell A vs. cell B tested whether recovery with static exercise affected thermoregulation in any different way than recovering while idle*. In statistical terms comparisons C vs. D and A vs. B are referred to as tests of significance for the 2 main effects of the research questions posed. In the present study these are the effect of condition and the effect of position on thermoregulation.

Interactions. In a two-way ANOVA a third research question may be explored. This question concerns the possible existence of an interaction. Interaction is the effect of two independent variables (e.g. condition and position) both operating together on a dependent variable (e.g. esophageal

* Throughout the text the term idle is used to define post exercise resting values during recovery after 25 min of moderate intensity cycling warm up in order to distinguish between resting values before the initiation of any experimental manipulation.

temperature, sweat rate, *etc.*). The interaction may be examined by first calculating the difference between the means in each experimental treatment across the rows of Fig. 1, and then comparing these differences i.e. ((I - II) vs. (III-IV)). A further method of examining the effect of interaction of position and condition acting together on the same dependent variable is to calculate the difference between cells I and III and compare it with the difference between cells II and IV e.g ((I-III) vs. (II-IV)). In general, the interpretation of any main effect depends on the presence or absence of significant interaction effects. If there is no interaction the main effects may be interpreted without qualification. Likewise, there is no complication in the interpretation of a significant main effect(s) when the interaction is significant but trivial enough to be considered inconsequential. In such a case, the interaction is named ordinal (Fig. 2). With a sizable and significant interaction, named disordinal, the meaning of significant main effects must be interpreted cautiously (Keppel 1982). To illustrate, consider the example presented in Fig. 2. The left panel indicates a typical example of ordinal significant interaction. The mean response (e.g. heart rate) in level 2(B) was consistently higher than the response in level 1(B) at both levels of the first factor. In addition, the difference of mean response between the level 2(B) and 1(B) condition at level 2 of the first factor was greater than the difference between level 2(B) and 1(B) condition at level 1 of the first factor. On the other hand, the right panel in Fig. 2 is a typical example of disordinal interaction. Overall, the mean response (e.g. thoracic fluid index) in level 2(A) is higher than the response in level 1(A) at the level 1 of

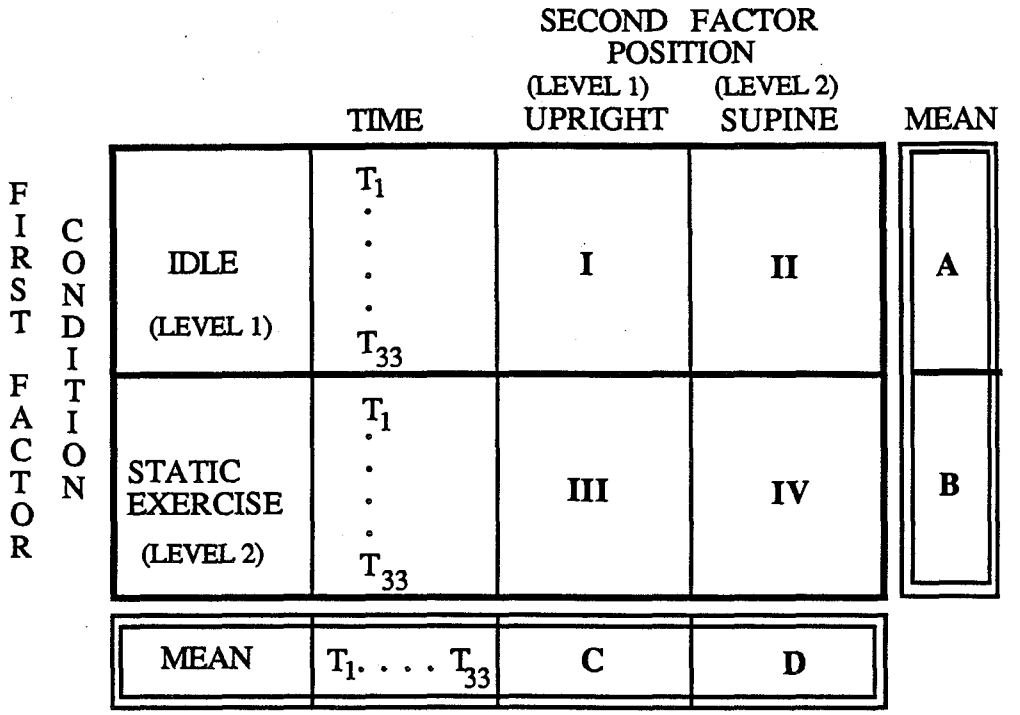


Fig. 1. Diagram showing a 2 factor (condition, position) by 2 level on each factor (idle, static exercise - upright, supine), ANOVA with repeated measures design used in the current investigation. Numbers I, II, III, and IV represent mean values of each experimental treatment in eight human subjects throughout 33 min of recovery. Letters A and B denote mean values for all subjects grouped regardless of position whereas letters C and D denote mean values for all subjects grouped across the second factor (position). See details in the text, p 13.

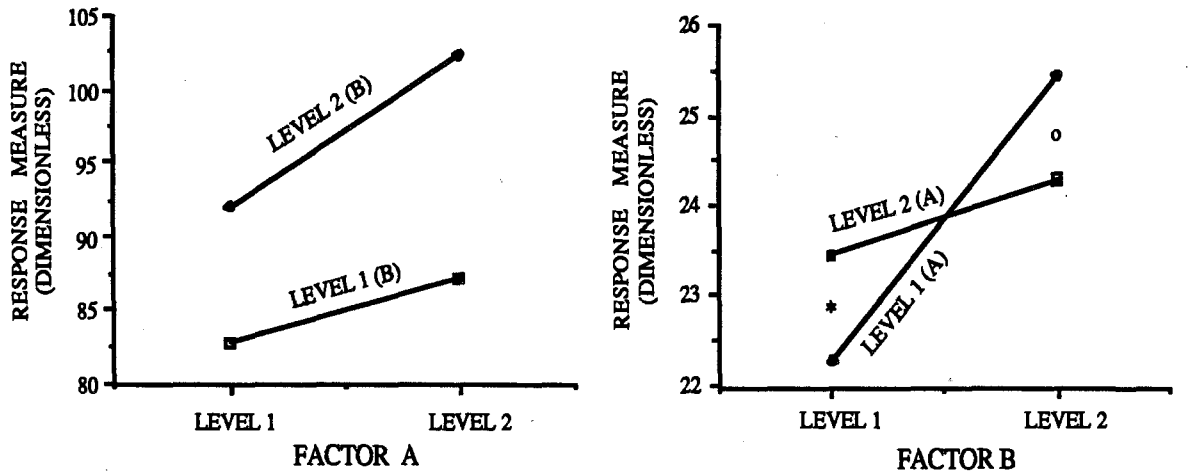


Fig. 2. Example of an ordinal (left panel) and of a disordinal interaction (right panel). (▣) symbol denotes level 1 of the second factor in the left panel and level 2 of the first factor in the right panel, whereas (●) represents level 2 and 1 of the second and first factor in the left and right panel, respectively. For details see pp. 13,14,16 in the text.

the second factor. The opposite, however, occurs between level 1(A) and 2(A) at the level 2 of the second factor.In this case, the significant main effect indicates that mean response of level 1 (Fig. 2, right panel, $*$) of the second factor is lower than the response at level 2 of the same factor (Fig. 2, right panel, $\circ$) but may be of little value considering the cross-over of the mean individual response values during the four individual experimental treatments.

Simple regression. Figure 3 presents the regression analysis procedure performed in every experimental condition in order to explore conjectural causal relationships. In particular, a simple regression analysis was executed (Feldman et al. 1986), by clustering individual raw data into an average relationship. $F_{sk}BF$ and T_{es} were individually as regressors, and $S_{w}r$ as a response, or on the other hand, T_{es} as the regressor and $F_{sk}BF$ as the response. In order to ensure linearity between the regressor and response the first three minutes of recovery were disregarded in all regression analyses. The coefficient of determination (r^2) and the level of significance were used initially as the sole criteria for evaluating the model's adequacy. Subsequently, an examination of the z score difference from unity between different slopes and intercepts was performed to determine the significance of these relationships (Kleinbaum and Kupper 1978).

Analysis of residuals. The values of single regression relationships were also evaluated by analysis of residuals and the test developed by Durbin-Watson (1971) was used to detect autocorrelation tendencies in the data. This test examined whether the error term of a response (e.g $S_{w}r$) was correlated and dependent on the error term of the regressor (e.g. T_{es}) since both had

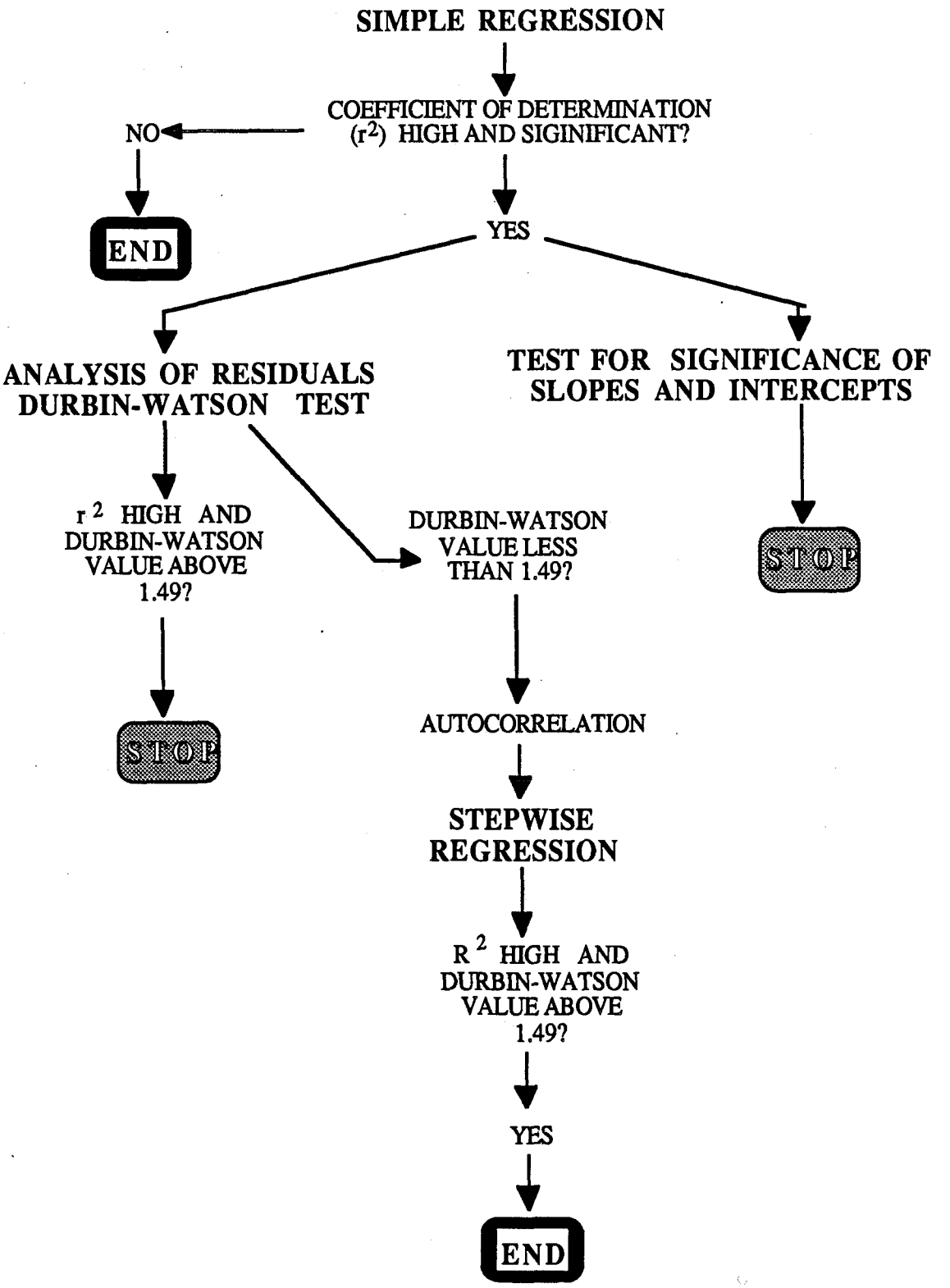


Fig. 3. Diagram indicating the regression analysis procedure followed in order to explore conjectural causal relationships between rate of sweating, forearm skin blood flow, and esophageal temperature. For details see text, pp.16, 18.

shown a natural sequential order throughout time (Montgomery and Peck 1982). No significant autocorrelation at $P \leq 0.05$ was considered to be present, if the Durbin-Watson value exceeded 1.49 (Montgomery and Peck 1982), for 30 measures (minutes in the present study) and for a single regressor. One of the major causes of autocorrelation in regression problems involving time series data, as in the present study, is failure to include other important regressors in the model (Montgomery and Peck 1982).

Stepwise regression. Autocorrelation, in any relationship explored, was treated with stepwise regression analysis (Efroymson 1960). In stepwise regression the response (e.g S_{wr}) was evaluated by adding new regressors (e.g. $F_{sk}BF$, T_{es}, DAP) gradually. At each step of a new regressor inclusion all regressors entered into the model previously were reassessed from their partial correlation with the response. If the correlation between one regressor and the response was inadequate then this regressor was dropped from the multiple linear regression model. The partial F-test, which is approximately an extrapolation of the t-test, was used to evaluate significance of the partial correlation between a regressor and the response. Evaluation of stepwise regression appropriateness relied on the Durbin-Watson test and the coefficient of determination (R^2) which was adjusted according to Ezekiel (1930) in order to optimize the selection of regressors taking into consideration the impact of adding regressors on the coefficient of determination.

Statistical level. The statistical level of significance in all tests performed was set at $P \leq 0.05$ except in the comparison of slopes and intecepts where significance was set at $P \leq 0.025$ in order to counteract an inflation error by performing 2 statistical tests (i.e. slope, intercept) on the same relationship.

4.0 RESULTS

The effect of posture

This section presents the results from post-exercise recovery in the upright and supine positions. Each recovery position comprises of data from both the idle and static exercise conditions.

Arterial pressure. Figure 4 (top) shows the time course of the mean arterial blood pressure (MAP) while subjects recovered from exercise, in the upright or supine position. Mean arterial pressure was moderately (98±3 vs. 92±4 mm Hg) but significantly ($P \leq 0.02$) higher in the upright than in the supine position. The same trend was observed for both the diastolic (DAP) and systolic arterial pressure (SAP) (Table 2). Unlike the diastolic pressure, which was significantly higher in the upright position ($P \leq 0.004$), the systolic pressure was not significantly affected by position ($P \leq 0.14$) .

Table 2: Mean and standard error (±)values in systolic and diastolic arterial pressure for 8 subjects during 33 minutes recovery in the upright or supine position after 25 min of exercising in a thermoneutral environment. Differences in diastolic pressure between positions are significant ($P \leq 0.004$).

TIME	SYSTOLIC PRESSURE (mm Hg)				DIASTOLIC PRESSURE (mm Hg)			
(min)	UPRIGHT	S.E.±	SUPINE	S.E.±	UPRIGHT	S.E.±	SUPINE	S.E.±
5	126.70	5.38	121.49	6.80	83.19	4.24	79.29	6.21
10	126.24	4.01	120.02	5.23	86.64	3.86	80.14	4.36
15	126.44	4.05	120.88	4.71	87.62	3.94	79.29	4.75
20	122.86	3.35	120.46	4.76	86.77	4.10	78.27	4.17
25	120.73	4.35	117.57	4.80	87.38	3.45	77.28	4.13
30	122.62	3.93	118.33	4.47	86.72	3.29	77.85	4.50
33	117.51	3.48	116.75	3.87	85.15	3.25	75.90	3.64
MEAN	123.30	4.08	119.36	4.95	86.21	3.73	78.29	4.54

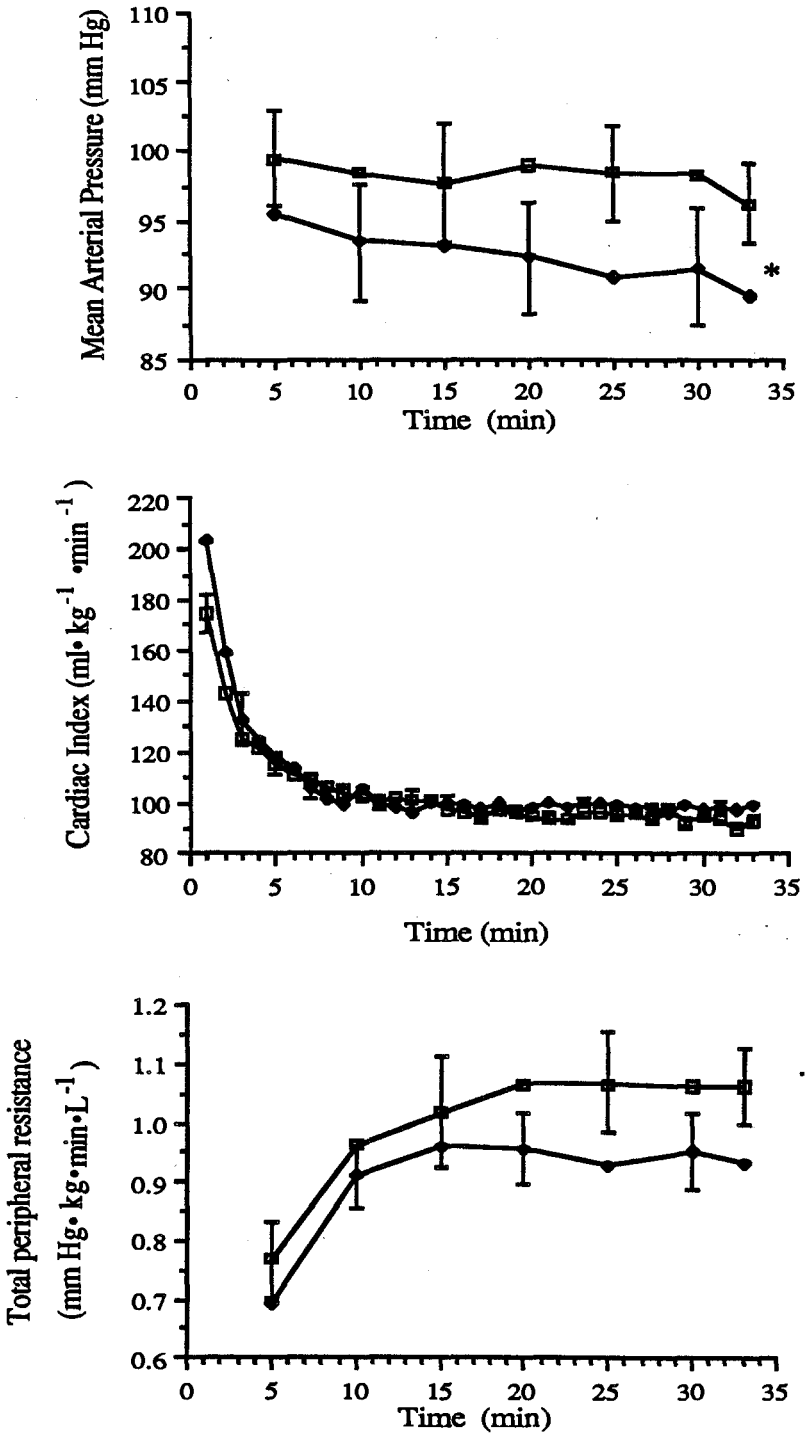


Fig. 4. Group changes (±SE) in mean arterial pressure (top), cardiac index (middle), and total peripheral resistance (bottom) in young males (n=8) during recovery in either the upright (-■-) or the supine (-◆-) position, after 25 minutes of body heating by performing moderate (50% $\dot{V}O_{2max}$) dynamic exercise in a thermoneutral ambient condition (22±0.6 °C, 42±8% rh). *P≤0.02.

Cardiac index. The time course of the mean decrease in CI as subjects recovered in either the upright or the supine position was similar (Fig. 4, middle). There was no significant difference, at any point, between the two experimental conditions ($P \leq 0.49$). At the 33rd minute of recovery CI values measured 92.99±2.41 $ml \cdot kg^{-1} \cdot min^{-1}$ in the upright and 99.32±2.26 $ml \cdot kg^{-1} \cdot min^{-1}$ in the supine position. The corresponding resting value in each position was 90.03±1.98 and 89.31±1.34 $ml \cdot kg^{-1} \cdot min^{-1}$, respectively.

Total peripheral resistance. Peripheral vasoresistance in the whole body increased during recovery in both positions reaching an asymptote within 10 minutes (Fig. 4, bottom). Total peripheral resistance tended to be higher during upright recovery than in the supine position ($P \leq 0.12$).

Heart rate. The group heart rate was significantly ($P \leq 0.004$) higher during recovery in the upright than in the supine position (Fig. 5, top). The initial upright and supine HR's were 133±2 and 129±2 $b \cdot min^{-1}$ and both declined exponentially to 89±2 and 76±2 $b \cdot min^{-1}$, respectively, by the 33rd minute of recovery. These values were still clearly above resting HR values for both the upright (71± 1 $b \cdot min^{-1}$) and for supine (69±1 $b \cdot min^{-1}$) positions.

Stroke index. Unlike the cardiac index, the SI value was significantly higher during supine recovery than in upright recovery ($P \leq 0.009$). The time course of the mean change in SI (Fig. 5, middle) is characterized by an exponential decrease to an asymptote during the first 15 minutes in both positions, followed by a slow rise in the supine position, which leads to final values of 1.30±0.04 and 1.05±0.04 $ml \cdot kg^{-1}$ in the supine and upright position, respectively. Stroke index at rest measured 1.27±0.02 and 1.30±0.01

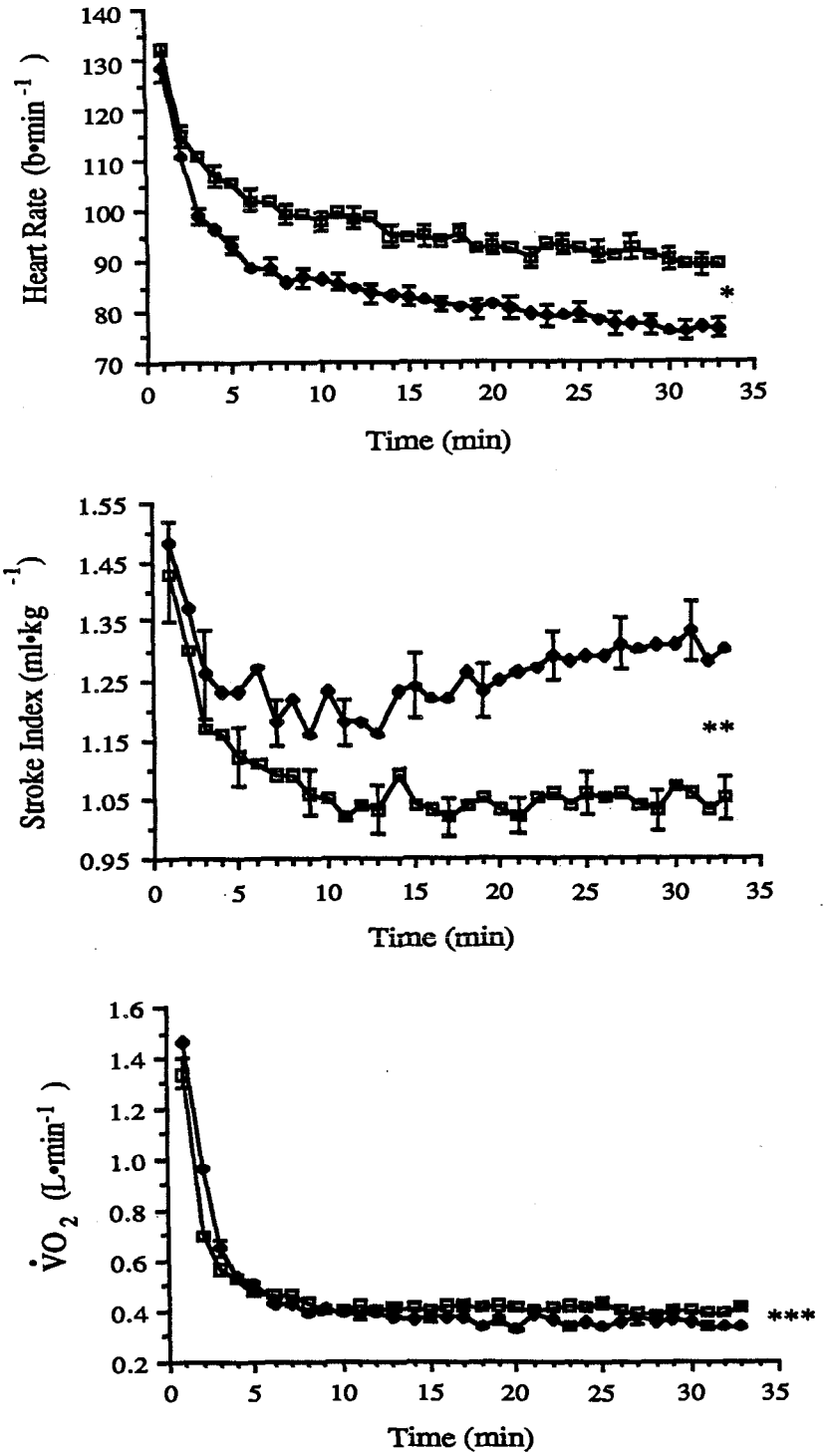


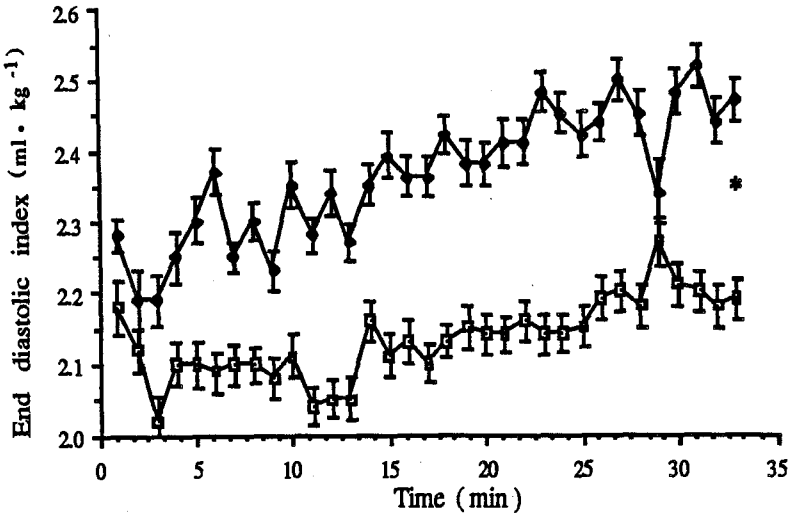
Fig. 5. Mean changes (±SE) in heart rate (top), stroke index (middle), and oxygen uptake ($\dot{V}O_2$, bottom) in groups of young males during recovery in either the upright (-■-) or the supine (-●-) position after 25 minutes of body heating by performing moderate (50% $\dot{V}O_{2max}$) dynamic exercise in a thermoneutral ambient condition (22±0.6 °C, 42±8% rh). *P≤0.004, **P≤0.009, ***P≤0.01 for only the last 24 minutes of recovery.

$ml \cdot kg^{-1}$ respectively in the upright and the supine conditions at rest.

Oxygen uptake. Upon cessation of the cycle exercise stimulus inducing initial sweating, the mean $\dot{V}O_2$, in both postural conditions, decreased dramatically within 5 minutes, finally attaining values slightly above resting $\dot{V}O_2$ (Fig. 5, bottom). After 33 minutes of recovery, oxygen uptake reached 0.41±0.01 $l \cdot min^{-1}$ and 0.34±0.02 $l \cdot min^{-1}$ in the upright and supine positions, respectively. At rest, prior to exercise these values were 0.40±0.01 and 0.36±0.01 $l \cdot min^{-1}$, respectively. During the 33 minutes of recovery oxygen uptake was not significantly different ($P \leq 0.21$) between the upright and supine position, but $\dot{V}O_2$ in the supine position became significantly higher in the upright position than in the supine posture ($P \leq 0.01$) during last 24 minutes of recovery.

End diastolic index.. Pre-trial resting end diastolic index values for upright and supine positions were 2.28 (±0.03) and 2.29 (±0.02), respectively.During the post-exercise recovery period EDI was higher ($P \leq 0.05$) in the supine than in the upright position (Fig. 6 top). In both positions EDI gradually increased during recovery. This incremental trend was more pronounced during recovery in the supine than in the upright position.

Thoracic fluid index. Mean TFI decreased during recovery in the supine position and always remained significantly lower ($P \leq 0.003$) than the corresponding value of the TFI in the upright position, the latter showing a minimal decrease upon cessation of dynamic exercise (Fig. 6, bottom).



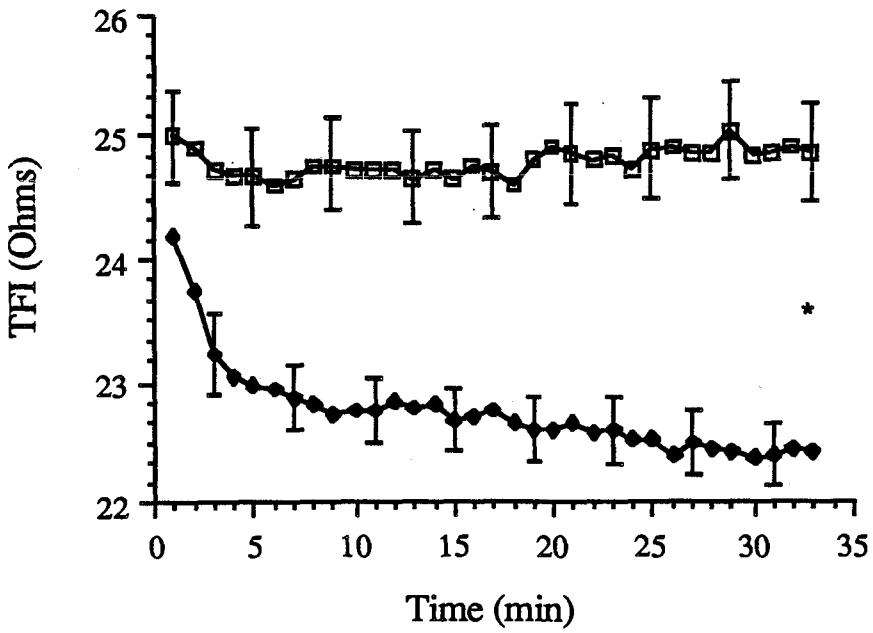


Fig. 6. Mean changes (±SE) in end diastolic index (top) and thoracic fluid index (TFI, bottom) in groups of young males (n=8) during recovery in either the upright (-■-) or the supine (-◆-) position after 25 minutes of body heating by performing moderate (50% $\dot{V}O_{2max}$) dynamic exercise in a thermoneutral ambient condition (22±0.6 °C, 42±8% rh). *P≤0.05 (top panel) amd *P≤0.003 (bottom panel).

Interactions.The effect of posture on arterial blood pressure should be interpreted considering that there was a significant interaction, in SAP ($P \leq 0.001$), DAP ($P \leq 0.05$), and MAP ($P \leq 0.02$), between position and condition. Figure 7 (A) shows the interaction which existed between different experimental treatments for the mean arterial pressure and discloses that the effect of body position during recovery on arterial blood pressure may almost be interpreted independently of the interaction.

It should be noted, moreover, that there was a significant interaction between position and condition in the heart rate variable ($P \leq 0.02$). The effect of position on HR was not obliterated by static exercise since the interaction appeared to be ordinal (Fig. 7, B).

There was also significant ($P \leq 0.02$) $\dot{V}O_2$ interaction between position and condition due to the fact that the oxygen uptake increase during recovery in the supine position with static exercise above that during recovery while idle, was disproportionally higher than the $\dot{V}O_2$ increase attained during recovery in the upright position with static exercise above that during upright recovery while idle (Fig. 7, C).

Figure 7 (D) shows that recovering in the upright position and simultaneously performing light static exercise, increases TFI much more than being upright and immobile during recovery. This disordinal interaction which exists between position and condition (Fig. 7, bottom) was significant ($P \leq 0.02$). However, the TFI response was always higher during upright recovery after 25 minutes of mild dynamic exercise regardless of whether passive or active (static) recovery was practiced.

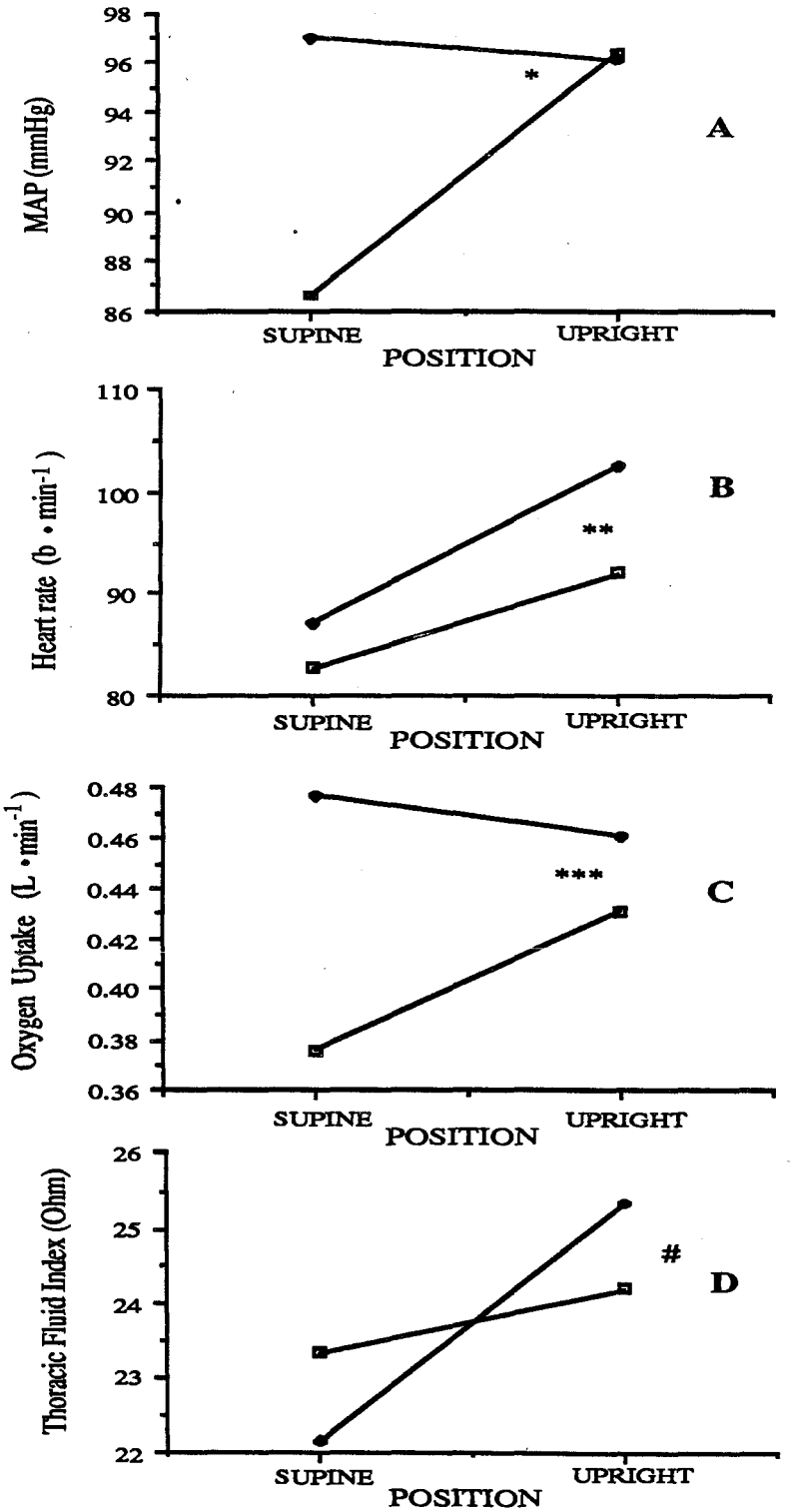


Fig. 7. Average temporal response in mean arterial blood pressure (A), heart rate (B), oxygen uptake (C), and thoracic fluid index (D) in 8 male subjects during recovery either performing static exercise (-◆-) or while idle (-■-) in the upright and the supine position after 25 minutes of body heating by performing moderate (50% V_{O2max}) dynamic exercise in a thermoneutral ambient condition (22±0.6°C, 42±8% rh).*P≤0.02, **P≤0.02, ***P≤0.02 ; #P≤0.02. All P values are referred to interaction

Esophageal temperature. The mean T_{es} at the onset of recovery in both the upright and supine position was almost identical, (37.48±0.09 and 37.49±0.29 °C, respectively). Group esophageal temperature decreased exponentially thereafter under both experimental conditions (Fig. 8, top). A large decrease in esophageal temperature occurred during the first 5 minutes of recovery (0.45 °C) and was followed by a small decline (0.2 °C) during the remaining 28 minutes of recovery. In particular, the final temperature after 33 minutes of recovery both in the upright and in the supine positions were 36.85±0.05 and 36.78±0.05 °C, respectively, while the corresponding resting values were 36.71±0.04 and 36.82 ±0.03 °C. The esophageal temperature in the upright position tended to be higher than in the supine recovery but the difference was not statistically significant (P≤0.20).

Skin temperature. Resting mean skin temperature for all subjects was 30.8±0.05 and 30.6±0.08 °C in the upright and the supine recovery positions, respectively. The group mean skin temperature ($\bar{T}_{sk}$) at the outset of recovery in the upright position was 32.5±0.37 °C and in the supine condition was 32±0.47 °C. $\bar{T}_{sk}$ increased for all subjects during the first 8 minutes regardless of the recovering position (Fig. 8, bottom). Group mean changes in skin temperature ($\Delta \bar{T}_{sk}$) were not significantly different (P≤0.07) between experimental conditions. However, statistical analysis of the last 24 minutes of $\Delta\bar{T}_{sk}$ recovery values revealed that ΔT_{sk} in the supine position was significantly higher than in the upright position (P≤0.02).

Sweating. The response of S_wr during 33 minutes of recovery in the supine and upright position for 8 subjects is shown in Fig. 9 (top). The sweat rate declined somewhat exponentially throughout the period in

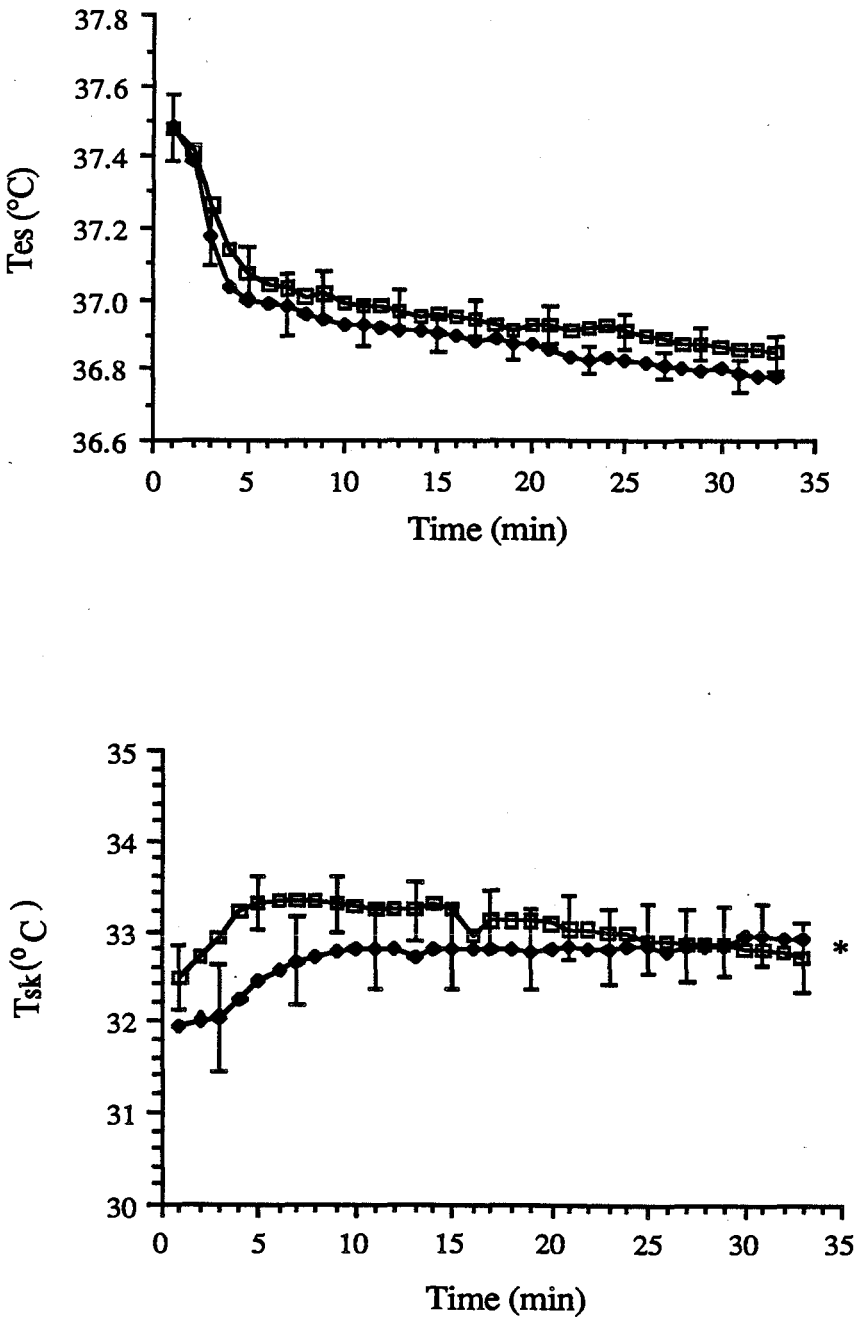


Fig. 8. Mean changes (±SE) in esophageal temperature (Tes, top) and mean skin temperature (Ts, bottom) in groups of young males (n=8) during recovery in either the upright (-■-) or the supine (-◆-) position after 25 minutes of body heating by performing moderate (50% $\dot{V}O_{2max}$) dynamic exercise in a thermoneutral ambient condition (22±0.6 °C, 42±8% rh). *P≤0.02 for the last 24 minutes of recovery in values expressed as difference (ΔTs).

both conditions. By the 33rd minute of recovery the difference in S_wr between the two positions increased from 15.38 at the onset to 32.26 mg·sec^{-1}·m^{-2}. By the end of the 33rd minute of recovery S_wr in the supine position was only moderately elevated (27.63±8.97 mg·sec^{-1}·m^{-2}) above the control pre-trial resting level (9.08 ±3.72 mg·sec^{-1}·m^{-2}) while S_wr during the upright recovery remained considerably elevated above the pre-trial resting condition (57.5±5.43 mg·sec^{-1}·m^{-2} vs. 11.41±4.05 mg·sec^{-1}·m^{-2} at rest, respectively). This pattern persisted until the cessation of the trial which occurred within 51.6 minutes in upright recovery and in 40.3 minutes in the supine position. The final S_wr values in the upright and supine recovery positions were 36.02±0.97 and 20.92±0.55 mg·sec^{-1}·m^{-2}, respectively.

Since there was an initial difference in S_wr of 15.38 mg·sec^{-1}·m^{-2} between the upright and supine position, all the values were expressed as a difference (ΔS_wr) throughout the time course of the 33 minutes by subtracting the current value at a given time point from the initial value. Statistical analysis of the delta sweat rate values (ΔS_wr) revealed an insignificant difference ($P \leq 0.19$) between either recovery position.

Forearm skin blood flow. Onset recovery $F_{sk}BF$ values were 6.84 ±1.37 and 7.87 ±1.22 ml·100g^{-1}·min^{-1} respectively in the supine and upright recovery, positions. Figure 9 (bottom) shows the changes in $\Delta F_{sk}BF$ i.e. the difference between $F_{sk}BF$ at the onset of recovery and the prevailing $F_{sk}BF$, which occurred during recovery in the supine and upright positions for 8 subjects. It is evident that $\Delta F_{sk}BF$ in the supine position decayed more slowly than in the upright position and from the 5th minute of recovery onwards, $\Delta F_{sk}BF$ in the supine condition was higher. These differences were statistically significant ($P \leq 0.04$). Forearm skin blood flow in the upright and supine positions amounted to 2.27±0.48

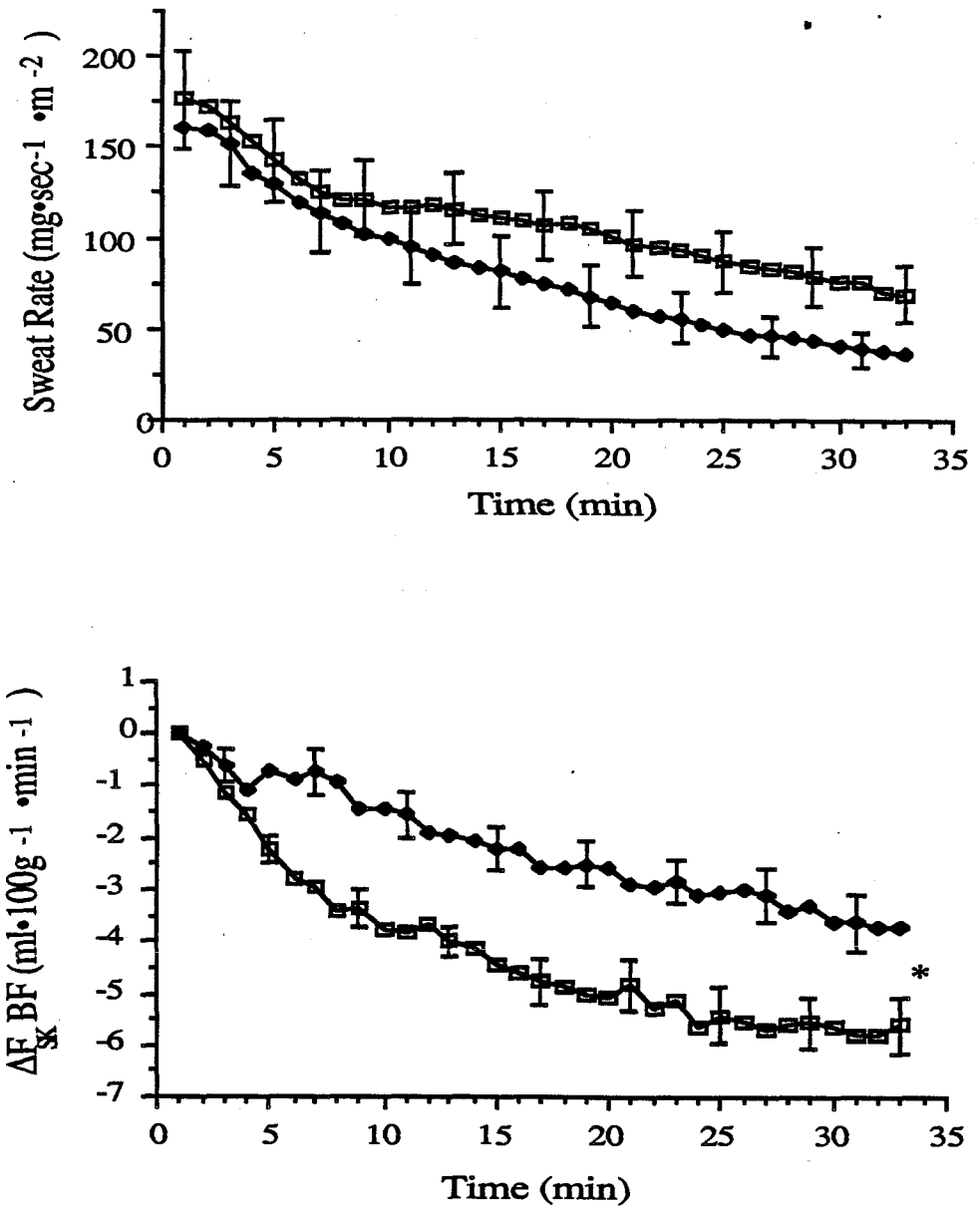


Fig. 9. Mean changes (±SE) in sweat rate (top), and difference of forearm skin blood flow (ΔFskBF, bottom) in groups of young males (n=8) during recovery in either the upright (-■-) or the supine (-●-) position after 25 minutes of body heating by performing moderate (50% $\dot{V}O_{2max}$) dynamic exercise in a thermoneutral ambient condition (22±0.6 °C, 42±8% rh). *P≤0.04.

and 3.06±1.01 ml·100g^{-1}·min^{-1}, respectively, by the 33rd minute of recovery. Pre-trial resting $F_{sk}BF$ was 1.49±0.72 and 1.10±0.68 ml·100g^{-1}·min^{-1}, respectively under these positions.

T_{es} - $S_w r$ relationship. A linear relationship existed between esophageal temperature and the rate of secretion of sweating in both experimental conditions (Fig. 10, top) with the characteristics shown in Table 3.

Table 3:Characteristics of simple regression analysis performed between $S_w r$ and T_{es} in the supine and upright positions. Both the slope and the intercept in supine position were significantly different ($P \leq 0.001$) from those in the upright position.

POSITION	INTERCEPT	SLOPE	DETERMINATION COEFFICIENT (r^2)
SUPINE	-15064.286	410.489	0.984
UPRIGHT	-11177.860	305.359	0.951

$F_{sk}BF$ - $S_w r$ relationship. Simple regression indicated a high association between skin blood flow and evaporation of sweat in both supine and upright recovery (Fig. 10,bottom). The main features of these linear relationships are shown in Table 4.

Table 4:Characteristics of simple regression analysis performed between $S_w r$ and $F_{sk}BF$ in the supine and upright positions. The slope in the supine position was greater than the slope in the upright position whereas the opposite was observed in the intercept. Both differences between position were significant ($P \leq 0.001$).

POSITION	INTERCEPT	SLOPE	DETERMINATION COEFFICIENT (r^2)
SUPINE	-65.987	30.079	0.984
UPRIGHT	53.294	13.047	0.903

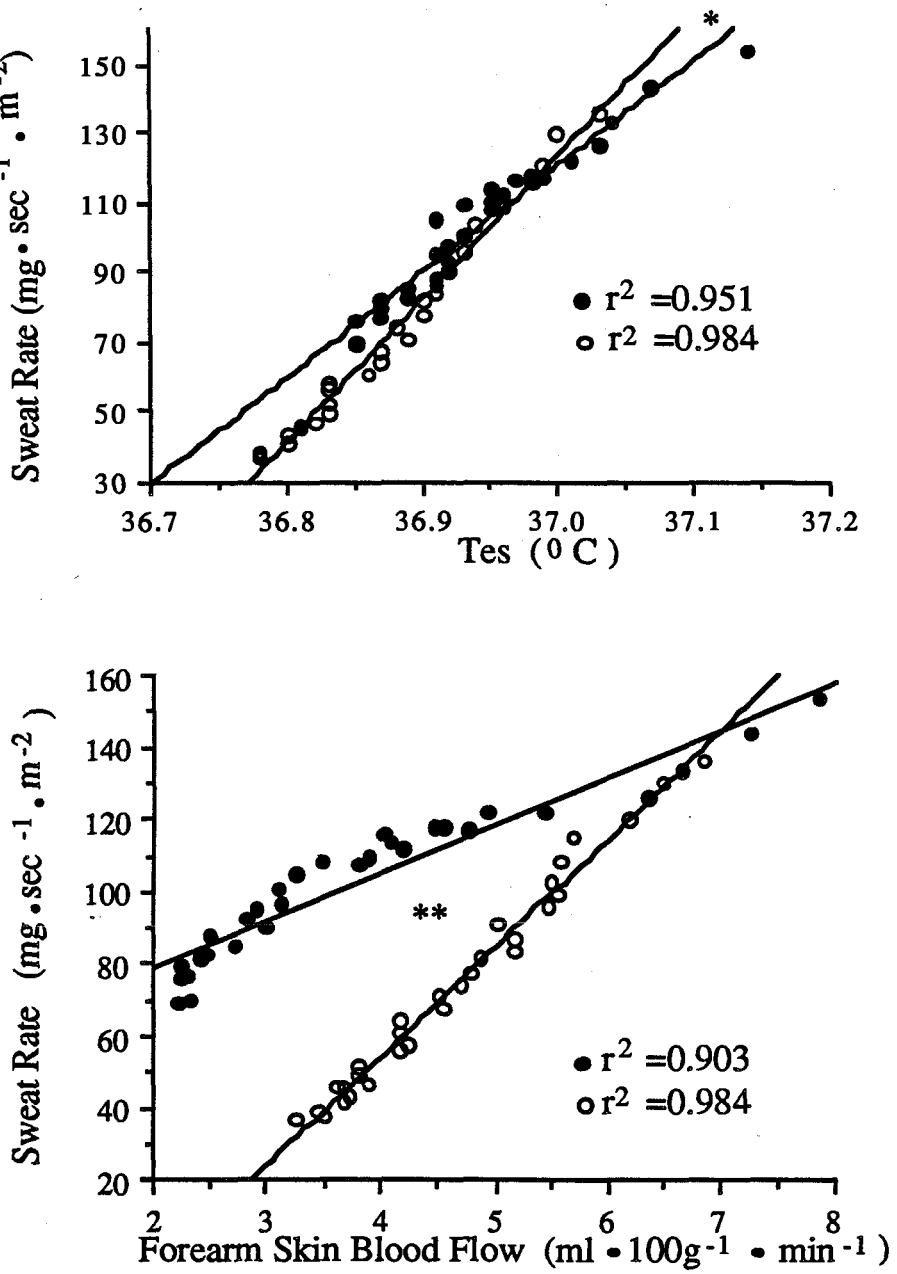


Fig. 10. Relationship between sweating rate and esophageal temperature (Tes, top), and sweating rate and forearm skin blood flow (bottom) in groups of young males (n=8) during 30 minutes of recovery in either the upright (●) or the supine (o) position after 25 minutes of internal body heating by performing moderate (50% $\dot{V}O_{2max}$) dynamic exercise in a thermoneutral ambient condition (22±0.6 °C, 42±8% rh). *P≤0.001, **P≤0.001. Level of significance refers to the comparison between the upright and the supine positions for both slope and intercept.

Simple regressions. As shown in Tables 3 and 4 98.4%, (r^2=0.984) of the $S_w r$ variance was predictable either from the esophageal temperature or skin blood flow values in the supine position. On the contrary, in upright recovery, only 95.1% (r^2=0.951) of the variance was predictable from the $S_w r$ on the basis of differences in a subject's T_{es}, and 90.3% (r^2=0.903) predictable on the basis of group differences in $F_{sk}BF$. However, analysis of residuals of all of the above simple regressions revealed that with the exception of $F_{sk}BF$ - $S_w r$ regression during supine recovery, a significant autocorrelation was present in the data.

Multiple regression of sweat rate. Predictability of $S_w r$ was not improved when stepwise regression analysis was performed accounting for both $F_{sk}BF$ and T_{es} during upright recovery. It was, however, found that the validity and accuracy of $S_w r$ prediction was improved by adding DAP as an extra regressor combined either with $F_{sk}BF$ or T_{es}. In particular, prediction of $S_w r$ was superior using a combination of $(333)T_{es}$ with (0.9)DAP. The coefficient of determination (R^2) in this case increased from 0.951 to 0.986 and the Dubin - Watson test value increased from 0.626 to 9.504 indicating complete elimination of the previous strong autocorrelation. Autocorrelation was not eliminated by adding $F_{sk}BF$ as an extra predictor in the regression between $S_w r$ and T_{es} during supine recovery. Thus, simple regression of $S_w r$ vs. $F_{sk}BF$ which was free of autocorrelation, was consolidated and retained as valid in this position.

The effect of static exercise

This section presents the results during post-exercise recovery in the idle and static exercise conditions. Each recovery condition comprises of data from both the supine and upright positions.

*Arterial pressure.*The main effect of different experimental conditions on MAP is shown in Fig. 11 (top). Mean arterial blood pressure was moderately (97±3 vs. 92±3 mm Hg) but significantly (P≤0.047) higher during static exercise than while idle in recovery, mainly due to equivalent changes in diastolic arterial pressure (Table 5). Systolic arterial pressure while idle in recovery tended to be lower than during the static exercise condition but the difference was statistically insignificant (P≤0.11).

Cardiac index.. Different experimental conditions (static exercise, idle) had no effect on CI (Fig. 11, middle) (P≤0.95). Cardiac index during both treatments decreased exponentially attaining values of 97.56±2.46 $ml \cdot kg^{-1} \cdot min^{-1}$ when a subject recovered idly and 97.74±2.32 $ml \cdot kg^{-1} \cdot min^{-1}$ during static exercise by the 33rd minute of recovery.These values were well above the resting pre-experimental values of 87.56±1.78,87.41±1.63 $ml \cdot kg^{-1} \cdot min^{-1}$ in both the idle and static exercise experimental conditions, respectively.

Table 5: Mean and S.E values in systolic and diastolic arterial pressure for 8 subjects during 33 minutes recovery during static exercise or while idle after 25 min. of exer cising in thermoneutral environment. Differences between conditions in diastolic pressure are significant (P≤0.03).

TIME	SYSTOLIC PRESSURE (mm Hg)				DIASTOLIC PRESSURE (mm Hg)			
(min)	IDLE	S.E.±	EXERCISE	S.E.±	IDLE	S.E.±	EXERCISE	S.E.±
5	125.26	4.45	122.93	5.64	77.54	4.05	84.94	4.25
10	119.44	3.08	126.83	4.53	80.45	3.61	86.33	3.15
15	120.88	2.45	126.44	5.00	81.25	3.52	85.67	3.55
20	119.49	2.62	123.83	3.92	79.28	3.58	85.75	3.29
25	116.11	3.21	122.19	4.51	79.78	2.94	84.87	3.24
30	116.99	3.01	123.96	3.90	78.57	2.93	86.00	3.58
33	114.76	2.71	119.50	3.34	77.77	2.53	83.27	3.42
MEAN	118.99	3.08	123.67	4.41	79.23	3.31	85.26	3.42

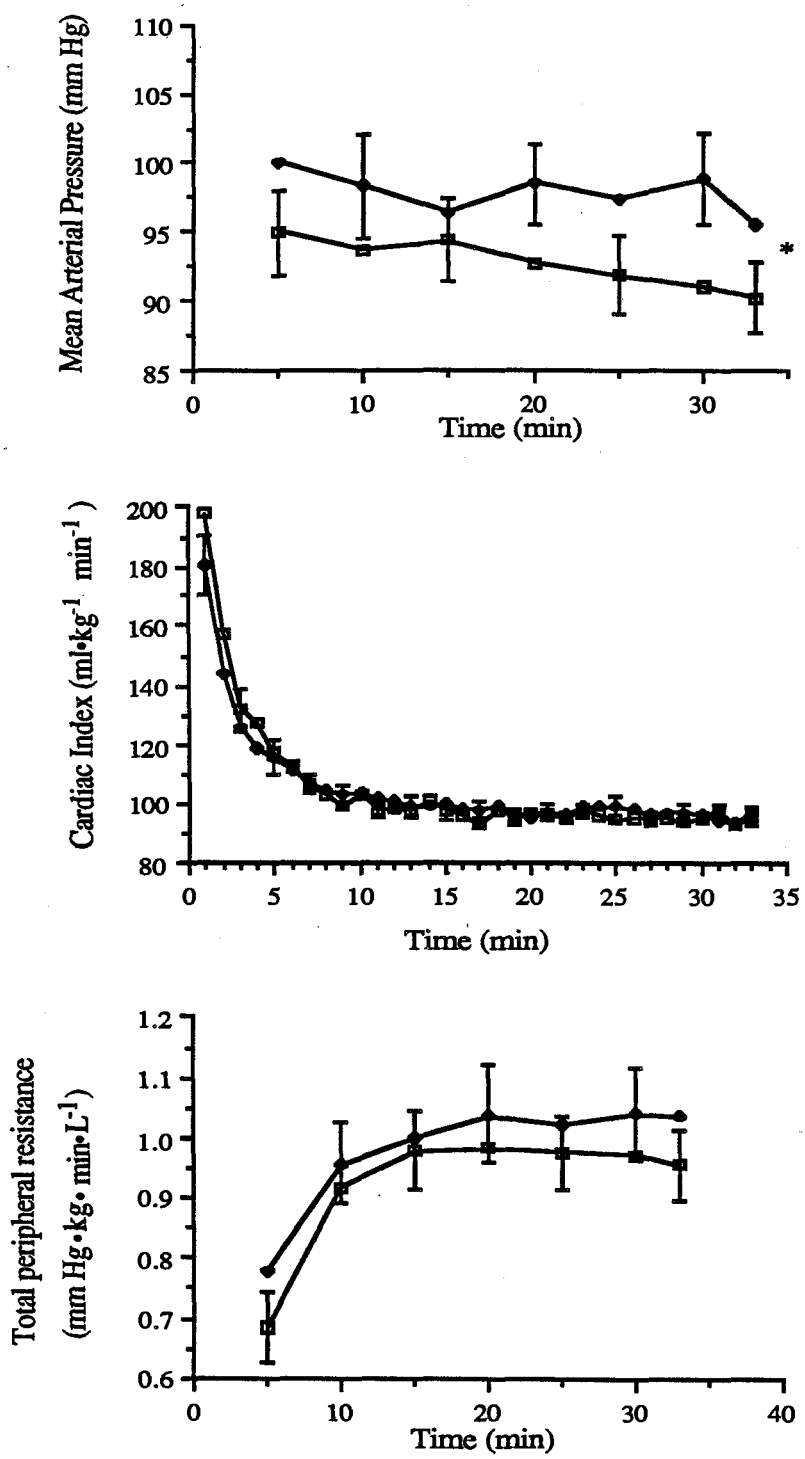


Fig. 11. Group changes (±SE) in mean arterial pressure (top), cardiac index (middle), and total peripheral resistance (bottom) in groups of young males (n=8) during recovery either with static exercise (-◆-, 16‡N) or while idle (-■-) after 25 minutes of body heating by performing moderate (50% V_{O2max}) dynamic exercise in a thermoneutral ambient condition (22±0.6 °C, 42±8% rh). *P≤0.01.

Total peripheral resistance. Peripheral vascular resistance in the whole body increased exponentially during recovery reaching an asymptote within 10 minutes. Total peripheral resistance tended to be higher ($P \leq 0.18$) during static exercise than in the idle recovery condition (Fig. 11, bottom).

Heart rate. The group HR was significantly ($P \leq 0.001$) higher during static exercise recovery than recovering while idle, Fig. 12 (top). Upon cessation of the warm up exercise, HR decreased exponentially from an initial value of 131±2.56 and 130±2.48 $b \cdot min^{-1}$ respectively in the idle and static exercise recovery conditions to 78.63±1.87 and 86.94±2.14 $b \cdot min^{-1}$ after 33 minutes of recovery. The latter values were clearly higher than corresponding resting values measured prior to commencing the experiment (69±1.22 and 71±1.30 $b \cdot min^{-1}$, respectively).

Stroke index. Stroke index values were 1.29±0.03 and 1.28±0.02 $ml \cdot kg^{-1}$ in the upright and supine conditions respectively at rest, before any experimental manoeuvre.Like the cardiac index, group SI values were not significantly higher during the idle than during the static exercise recovery condition ($P \leq 0.07$). The time course of mean change in SI is characterized by an initial sharp decrease during the first 5 minute period followed by a slow rise in the idle condition and a continued decline to a lower asymptote in the static exercise condition (Fig. 12, middle) leading finally to 1.25±0.05 and 1.1±0.04 $ml \cdot kg^{-1}$ values, respectively, after 33 minutes of recovery.

Oxygen uptake. Mean $\dot{V}O_2$ recovery values also decreased sharply in both conditions, to a level slightly above resting (pre-experimental) $\dot{V}O_2$ values (Fig. 12, bottom). After 33 minutes of recovery, oxygen uptake attained values of 0.36±0.02 $l \cdot min^{-1}$ and 0.39±0.01 $l \cdot min^{-1}$ in idle and static exercise conditions, respectively while at rest, prior to the experiment, these values were 0.37±0.01 and 0.36±0.02 $l \cdot min^{-1}$. Oxygen uptake during static

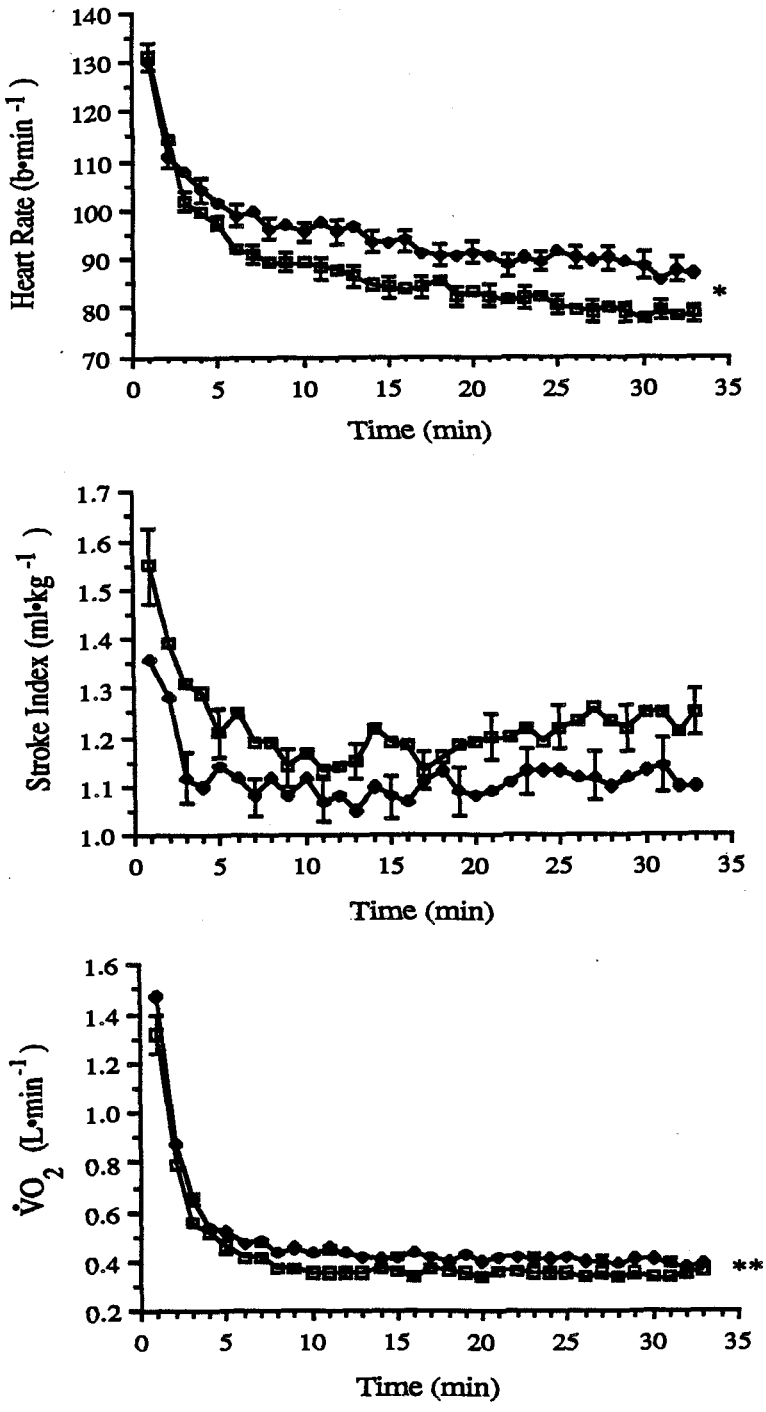


Fig. 12. Mean changes (±SE) in heart rate (top), stroke index (middle), and oxygen uptake ($\dot{V}O_2$, bottom) in groups of young males during recovery either with static exercise (-●-, 161N) or while idle (-■-) after 25 minutes of body heating by performing moderate (50% $\dot{V}O_{2max}$) dynamic exercise in a thermoneutral ambient condition (22±0.6 °C, 42±8% rh). *P≤0.001, **P≤0.01.

exercise was significantly higher (P≤0.01) by the 33rd min of recovery compared with the idle condition.

*End diastolic index.*Pre-trial resting end diastolic index values during idle and static exercise conditions were 2.39 (±0.03) and 2.12 (±0.02), respectively. During the post-exercise period EDI was higher (P≤0.06) during idle recovery than recovery with static exercise. In both conditions EDI was progressively increasing during recovery (Fig. 13).

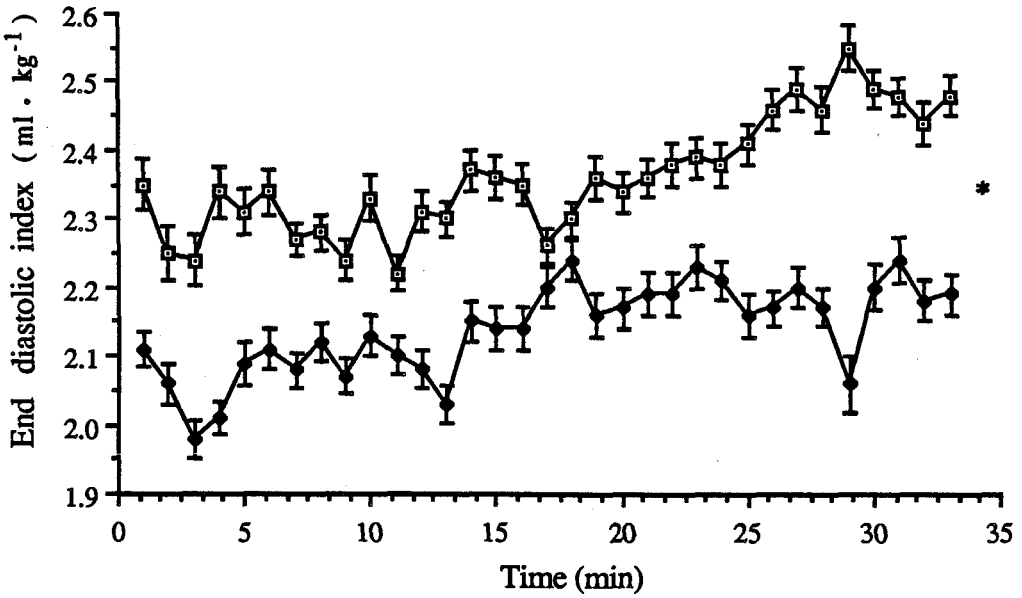


Fig. 13. Mean changes (±SE) in end diastolic index in groups of young males during recovery either with static exercise (-●-, 161N) or while idle (-■-) after 25 minutes of body heating by performing moderate (50% $\dot{V}O_{2max}$) dynamic exercise in a thermoneutral ambient condition (22±0.6 °C, 42±8% rh). *P≤0.06.

*Interactions.*There was a significant interaction (P≤0.01) of mean arterial blood pressure between subjects recovering both while idle and performing light static exercise (Fig. 14, top) which indicates that static exercise in the upright position induced no further increase in MAP than that caused by moving from the supine idle to the upright idle recovery position

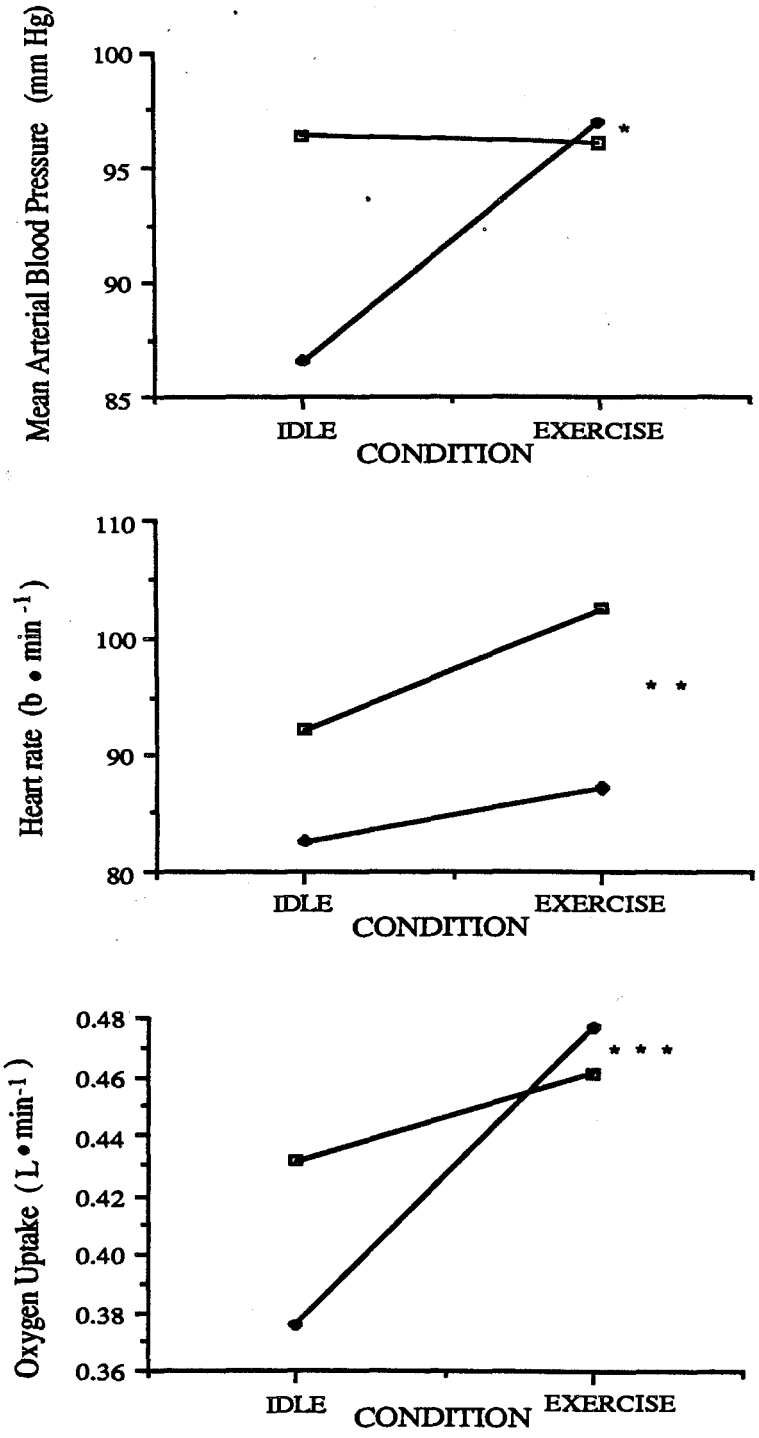


Fig. 14. Group responses in mean arterial pressure (top), heart rate (middle) and oxygen uptake (bottom) in a group of 8 male subjects during recovery either in the upright (-■-) or supine (-◆-) position while idle and during performing static exercise (161N), after 25 minutes of body heating by executing moderate (50% $\dot{V}O_{2max}$) dynamic exercise in a thermoneutral ambient condition (22±0.6 °C, 42±8% rh). *P≤0.02, **P≤0.02, ***P≤0.02. All P values are referred to the interaction between idle and static exercise recovery condition

did. Nevertheless, the effect of condition (i.e. idle or static exercise) on blood pressure may be interpreted independently of this interaction first because the interaction is close to ordinal and secondly because blood pressure is the independent variable in the present study.The interaction of experimental conditions on both SAP and DAP was significant ($P \leq 0.001$, and $P \leq 0.046$, respectively).

As shown in Fig. 14 (middle), the heart rate was disproportionally increased during recovery with static exercise while standing upright than while supine compared with upright and supine idle conditions, respectively. This ordinal interaction ($P \leq 0.02$) however, did not abrogate the main effect of the different experimental conditions (idle - static exercise) since heart rate was always higher during static exercise than during idle recovery regardless of the position involved.

It was also found that there was an ordinal interaction in $\dot{V}O_2$ between different individual experimental conditions (Fig. 14 bottom). Oxygen uptake, nevertheless, was overall higher during recovery with static exercise than while recovering idly.

Esophageal temperature. Mean rest esophageal temperature prior to the start of the experiments was 36.79±0.03 and 36.74±0.02 °C in the idle and static exercise conditions, respectively. Furthermore, the group mean T_{es} value in the 2 recovery modes at the start of recovery was slightly different attaining values of 37.54±0.11 and 37.42±0.07 °C, respectively. Esophageal temperature during the first 5 minutes decreased sharply in both idle and static exercise recovery (0.51 and 0.41 °C, respectively) and more gradually (0.25 and 0.19 °C, respectively) during the remaining 28 minutes of recovery. The esophageal temperature difference (ΔT_{es}) during static

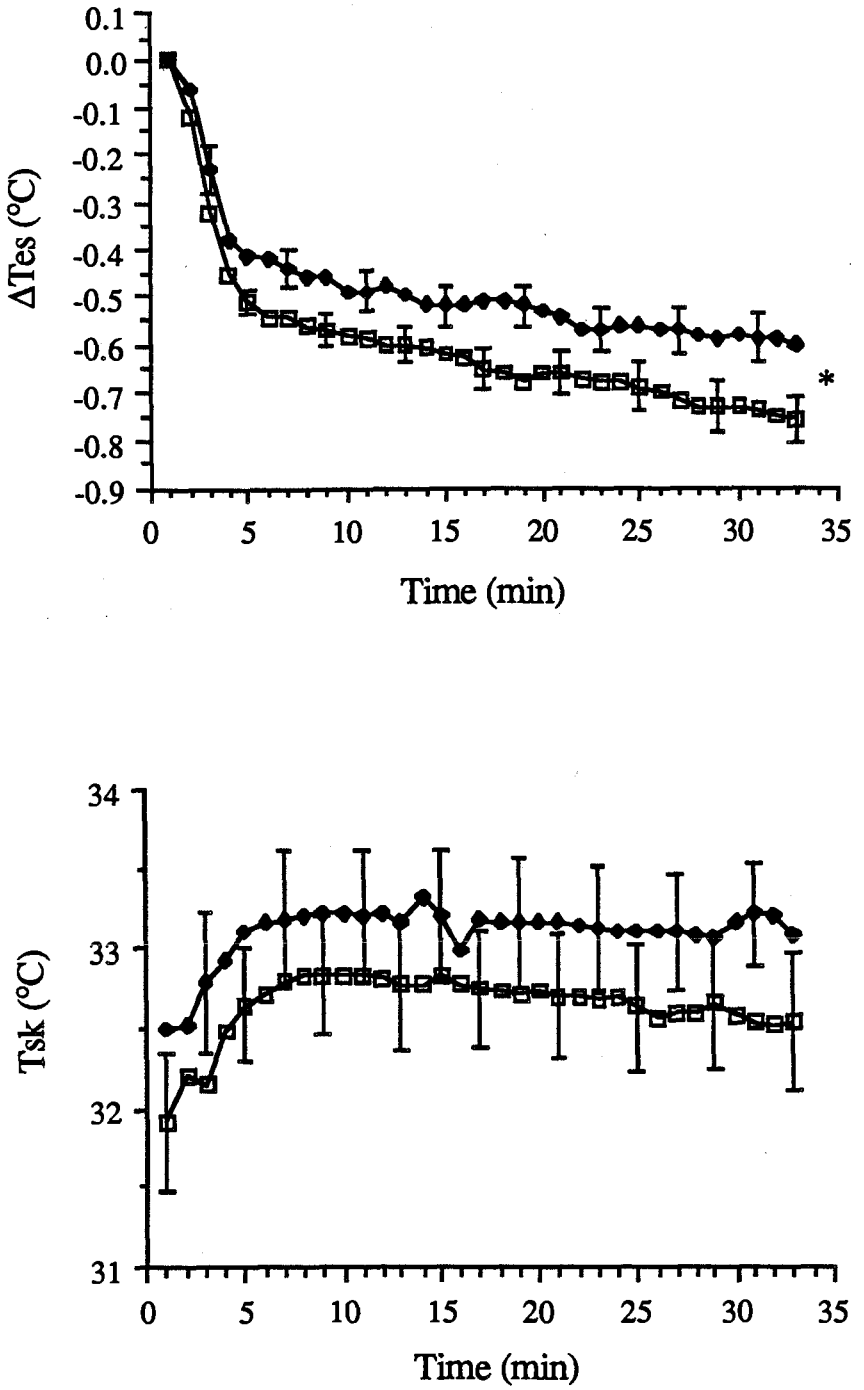


Fig. 15. Mean changes (±SE) in esophageal temperature differences (ΔTes, top) and mean skin temperature (Tsk, bottom) in groups of young males (n=8) during recovery either with static exercise (-◆-, 161N) or while idle (-■-) after 25 minutes of body heating by performing moderate (50% $\dot{V}O_{2max}$) dynamic exercise in a thermoneutral ambient condition (22±0.6 °C, 42±8% rh). *P≤0.04.

exercise recovery was significantly ($P \leq 0.04$) higher than during idle recovery as shown in Fig. 15 (top).

Skin temperature. Group $\bar{T}_{sk}$ at rest was 30.42±0.07 and 30.94±0.08 °C in the idle and static exercise recovery treatment, respectively. Mean skin temperature for all subjects at the onset of recovery in the idle and static exercise conditions were 31.90±0.43 and 32.51±0.41 °C, respectively. In both experimental conditions $\bar{T}_{sk}$ increased during the first 7 min of recovery subsequently attaining asymptotic values (Fig. 15, bottom) of 32.54 ±0.42 and 33.08±0.31 °C in the idle and static exercise treatments, respectively. Group mean skin temperatures differences ($\Delta\bar{T}_{sk}$) were not significantly different ($P \leq 0.42$) between experimental conditions.

Sweating. The rates of sweating prior to any experimental manipulation were 11.63±1.43 and 8.86±1.43 $mg \cdot sec^{-1} \cdot m^{-2}$ in the static exercise and idle recovery treatments, respectively. Upon termination of the dynamic exercise warm up procedure, the group mean $S_w r$ declined progressively in both recovery conditions (Fig 16, top). The initial decay of sweat rate was greater in the idle condition than during static exercise. From the 10th minute onwards, $S_w r$ decay was parallel under both modes of treatments during recovery. After the 33rd minute, $S_w r$ during recovery with static exercise was still 81.51±14.63 $mg \cdot sec^{-1} \cdot m^{-2}$, while recovery in the idle condition was only 38.00±8.68 $mg \cdot sec^{-1} \cdot m^{-2}$. This trend persisted until the conclusion of the trials, which occurred at 51 minute for static exercise recovery and at 42 minute for idle recovery. Terminating values of $S_w r$ were equal to 31.5±2.86 and 25.42±2.94 $mg \cdot sec^{-1} \cdot m^{-2}$, respectively. During the final 24 minutes of recovery, the rate of sweating was always significantly higher ($P \leq 0.029$) for static exercise.

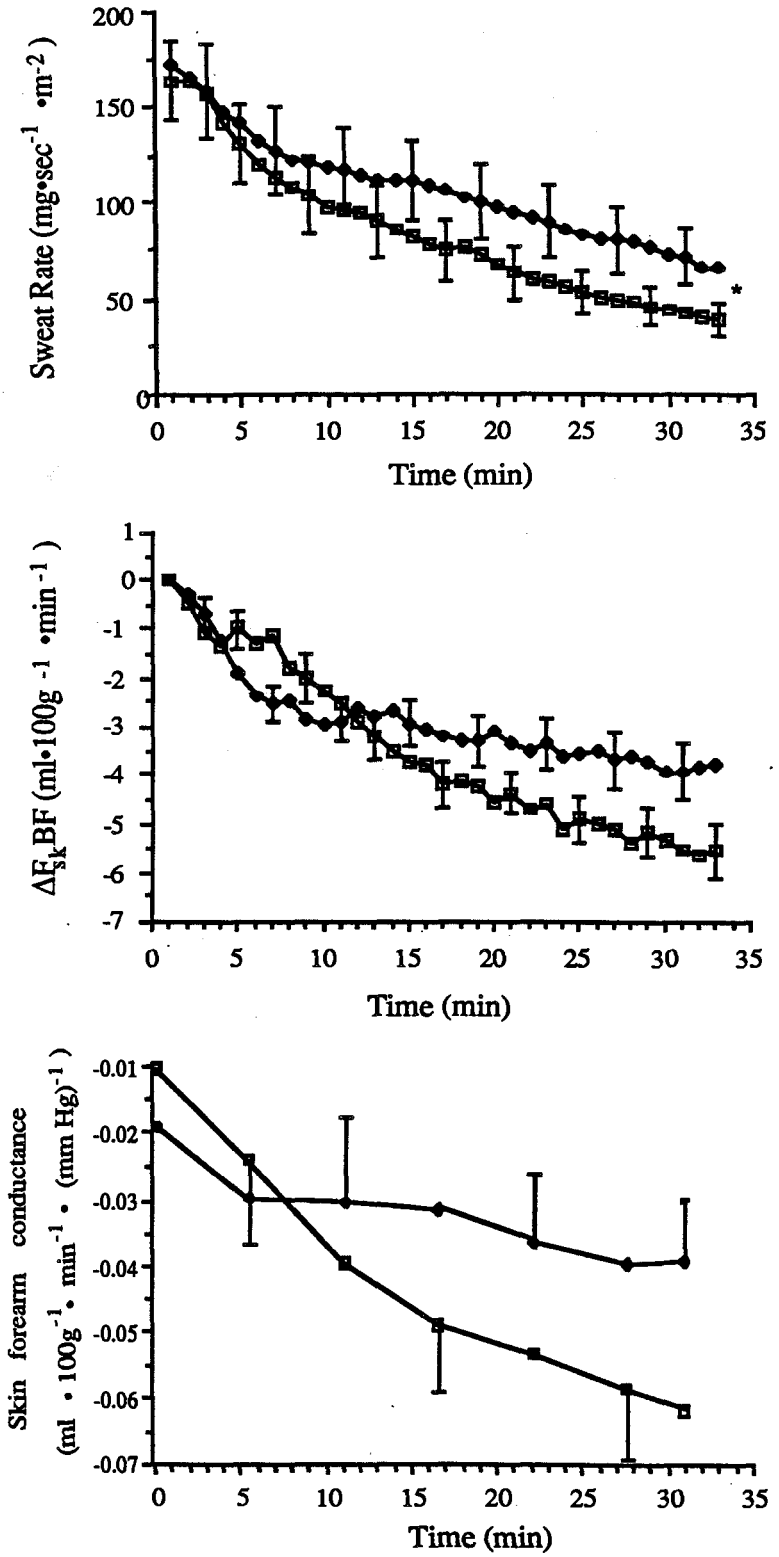


Fig. 16. Mean changes (±SE) in sweat rate (top), forearm skin blood flow difference ($\Delta F_{sk}BF$, middle) and skin forearm conductance (bottom) in groups of young males (n=8) during recovery either with static exercise (-◆-, 161N) or while idle (-■-) after 25 minutes of body heating by performing moderate (50% $\dot{V}O_{2max}$) dynamic exercise in a thermoneutral ambient condition (22±0.6 °C, 42±8% rh). *P≤0.03.

Forearm skin blood flow. Forearm skin blood flow prior to warm - up was equal to 1.36±0.42 and 1.26±0.38 ml·100g^{-1}·min^{-1} in the designated idle and static exercise groups respectively. At the start of recovery after exercise $F_{sk}BF$ in these groups was 7.97±1.31 and 6.81±1.73 ml·100g^{-1}·min^{-1}, respectively. There was an acute decrease in the $F_{sk}BF$ difference ($\Delta F_{sk}BF$) during recovering while idle and during static exercise (Fig 16, middle). However, $F_{sk}BF$ while idle declined to a lower (2.39±0.59), but insignificantly different value, from that during static exercise (3.03±0.89 ml·100g^{-1}·min^{-1}) by the 33rd minute of recovery.

Forearm skin conductance. Forearm skin conductance was insignificantly lower on the 33rd minute of idle recovery than static exercise recovery (Fig. 16, bottom).

T_{es} - $S_w r$ relationship. Both during idle and static exercise recovery, the rate of of sweating was linearly related to esophageal temperature (Fig. 17, top) with the constants shown in Table 6.

Table 6:Characteristics of simple regression analysis performed between $S_w r$ and T_{es} in the static exercise and idle recovery conditions.

CONDITION	INTERCEPT	SLOPE	DETERMINATION COEFFICIENT (r^2)
IDLE	-12755.346	347.760	0.966
EXERCISE	-13471.577	367.574	0.954

Statistical analysis showed that the difference between slopes in the idle and static exercise recovery conditions were significant at the 5% level but not at the hypothesized 2.5% level ($P \leq 0.049$). Similarly, the T_{es} intercept during idle recovery was not significantly different from the intercept

when static exercise recovery was undertaken ($P \leq 0.097$).

S_kBF - S_wr relationship. Figure 17 (bottom) shows a high degree of linearity between $F_{sk}BF$ and S_wr in both idle and exercise conditions, and Table 7 presents the characteristics of this linearity. As is evident, the slope for static exercise was slightly higher than idle recovery (20.03 vs. 18.06) but statistically insignificant ($P \leq 0.27$). Conversely, the intercept value during exercise was significantly different from that in idle recovery ($P \leq 0.001$).

Table 7:Characteristics of simple regression analysis performed between S_wr and $F_{sk}BF$ in the static exercise and idle recovery conditions. The difference in slope between conditions was not significant unlike the difference in intercepts ($P \leq 0.001$).

CONDITION	INTERCEPT	SLOPE	DETERMINATION COEFFICIENT (r^2)
IDLE	-5.444	18.065	0.986
EXERCISE	21.482	20.030	0.822

Evaluation of simple regressions. Despite high r^2 values shown in regression of S_wr against $F_{sk}BF$ and T_{es}, in both experimental conditions, a strong autocorrelation prevailed. In addition, the relationship between $F_{sk}BF$ and S_wr in the exercise condition (Fig. 17) is not linear despite the moderately high r^2 value (0.822).

Multiple regression of sweat rate. Simple regression, as it is described above, indicates that S_wr was ostensibly equally well predicted by changes in T_{es} or in $F_{sk}BF$. Both relationships, however, were subject to strong autocorrelation. Stepwise regression, considering both $F_{sk}BF$ and T_{es} as regressors improved, but did not totally eliminate autocorrelation effects, during recovery in the idle condition. In contrast, entering $F_{sk}BF$and oxygen

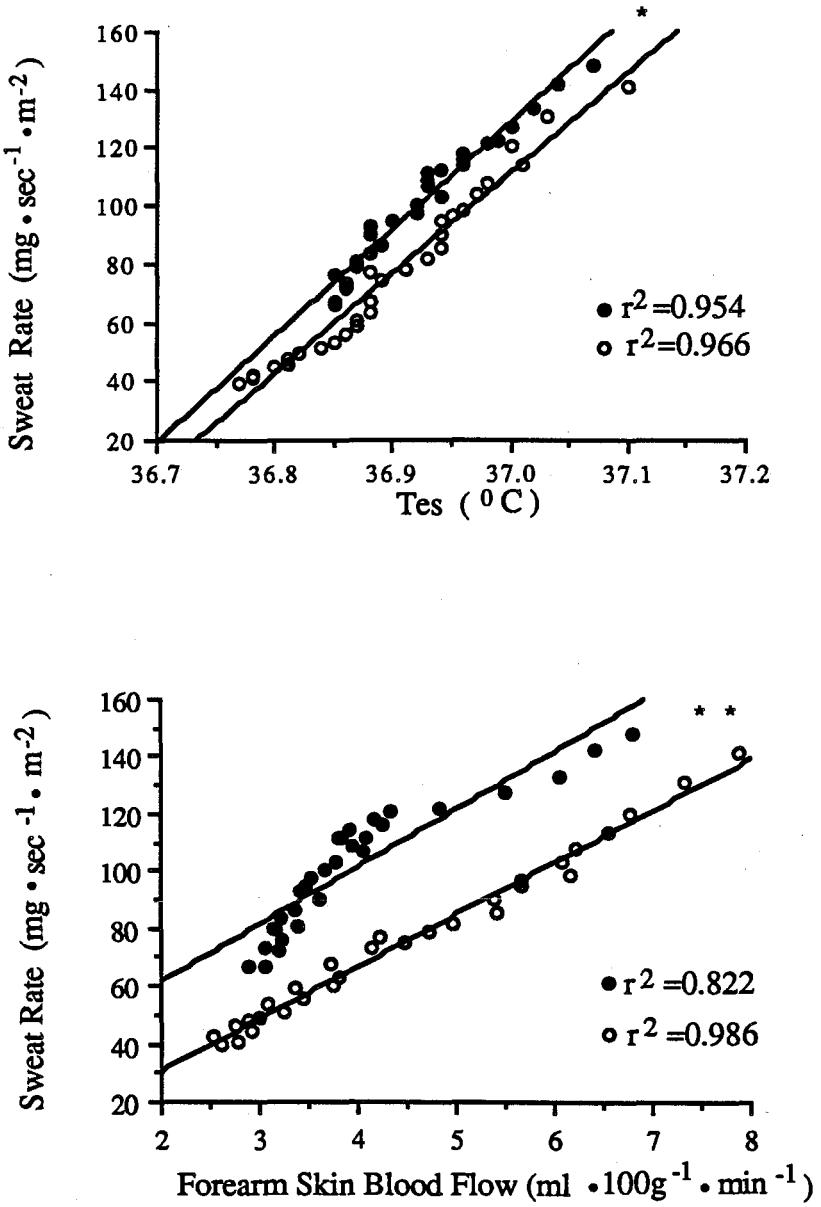


Fig. 17. Relationship between the sweating rate and esophageal temperature (Tes, top), and sweating rate and forearm skin blood flow (bottom) in groups of young males (n=8) during 30 minutes of recovery either with static exercise (● , 161N) or while idle (o) after 25 minutes of internal body heating by performing moderate (50% $\dot{V}O_{2max}$) dynamic exercise in a thermoneutral ambient condition (22±0.6 °C, 42±8% rh). *P≤0.05, **P≤0.001. Level of significance refers to comparison between the idle and static exercise recovery treatment for only the slope (*) and the intercept (**).

uptake as regressors into the regression model, eliminated autocorrelation and improved the R^2 value (0.988). During recovery while performing static exercise, however, $F_{sk}BF$ changes seemed not to be of any significant effect due to poor linearity and the autocorrelation which persisted no matter what other possible regressor was incorporated into the model. Conversely, entering the diastolic pressure as an extra regressor (2.25 DAP) the coefficient of determination and Dubin-Watson test value were both enhanced from 0.954 and 0.627 to 0.975 and 5.15, respectively, during recovery from thermal stress while exercising statically.

5.0 DISCUSSION

The following discussion of the results will focus on the contribution of high and low pressure baroreceptors and the effect of neuromuscular stimulus on the heat dissipating mechanisms. The former was achieved by changing the body position, whereas the latter was determined from observations made during mild static exercise either in the supine or upright position. The interpretation of the results depends on, and hence should be viewed first in the context of the cardiovascular responses to postural changes and static exercise.

The effect of posture

Cardiovascular response to postural changes.

The principal hemodynamic effect of altering central venous volume has been extensively studied mainly under conditions of lower body subatmospheric pressure (LBSP), water immersion, passive tilting or altered posture. In general, LBSP (at -50 mmHg), standing, and head up tilt at +70° induce an analogous central venous volume decrease (Wolthius et al. 1974). Water immersion up to the xiphoid notch and a recumbent posture induce a similar central venous volume increase (Rowell 1986).

Overall response. Total peripheral resistance tended to be higher (Fig. 4, bottom) in the upright sitting than in the supine position as has been previously shown by others (Brigden et al. 1950, Shvartz et al. 1983). By the 33rd minute* of supine recovery albeit after an acute exercise stimulus, heart rate, systolic and diastolic pressure decreased by 14.6%, 0.6% and 10.9% respectively while stroke index increased by 23.8% compared with upright recovery. These results, in general, are in accordance with the

* The 33rd minute of recovery was considered as a representive asymptotic value and frequent referall to this is made throughout the discussion.

literature. In particular, Thadani and Parker (1974) pointed out that when 10 male subjects changed from a sitting to a supine position mean heart rate, systolic and diastolic pressure decreased by 13.1%, 1.5% and 7.3% respectively, while stroke index increased by 42.8%.

Cardiac index. Despite a lower cardiac index observed by the 33rd minute of recovery in the upright than in the supine position, mean cardiac index was not statistically different between the supine and upright position in the present study (Fig. 4, middle) contrary to the majority of the current literature which reports cardiac index to be significantly lower in the sitting position than in the supine posture (Bevegård et al. 1960, Bevegård et al. 1963, Pentecost et al. 1963, Smith et al. 1970, Thadani and Parker 1974). Nevertheless, there is also some evidence to show that cardiac index may be insignificantly lower in the sitting position compared with the supine position at rest (Bevegård et al. 1966, Shvartz et al. 1983). Shvartz et al. (1983) ascribed such discrepancies in the literature to the fact that the cardiac index in the sitting position depends on the previous position and that it takes one hour before the full hemodynamic response to a position change may be accurately assessed. In addition, in the present study sitting subjects contracted their leg muscles *ad libitum* while recovering in the upright position, which may have contributed to a smaller decrease in stroke index than expected compared with the supine position value. Increased muscular activity in the upright position here is confirmed by a significantly elevated oxygen uptake (70 $ml \cdot min^{-1}$) in the same position than in the supine position. This finding is consistent with the results of Bevegård et al. 1960, and Bevegård et al. 1963.

Thermoregulatory response to postural changes.

Esophageal temperature. Upon cessation of the exercise-induced elevation of the core temperature, esophageal temperature decreased abruptly during the first five minutes in both upright and supine recovery (Fig. 8, top). This extensive decline in T_{es} was not caused by a dramatic enhancement of heat loss mechanisms (Fig. 9). It was probably caused by a diminution of heat production, tissue perfusion rate and blood flow redistribution accompanying recovery from dynamic exercise.

In particular, during dynamic exercise, T_{es} may reflect mainly active muscle temperature since muscle generates considerable amount of heat and receives almost 70% of cardiac output (Rowell 1974). Concomitantly, an elevated muscle perfusion rate due to exercise enhanced cardiac output could explain a dynamic rise in T_{es}. As soon as exercise stops, T_{es} should change as blood flow is redistributed and, also, reflect increasingly the thermal status of previously inactive, cool and vasoconstricted regions. Moreover, an abrupt decrease of T_{es} upon cessation of isotonic exercise is caused by a steep decrease of the rate the blood perfusing body tissues as shown by the cardiac output values (Fig. 4, middle). The above suggested sequence of events is supported by the work of Saltin and Hermansen (1966), who showed that during moderate isotonic exercise the T_{es} rise was similar to the rise of temperature in exercising muscle, while during recovery, muscle temperature was always higher than esophageal. Changes in the magnitude of cardiac output (Carlsten and Grimby 1958) and blood flow redistribution have also been suggested (Carlsten and Grimby 1958, Mekjavić and Bligh 1989) to affect T_{es}.

The initial acute decrease in T_{es} during recovery in either recovery position was not followed by a simultaneous steep decrease in either S_wr or forearm skin blood flow ($F_{sk}BF$).

The site of measuring the core temperature is still equivocal. T_{es} is similar to cardiac temperature in a steady state situation, contrary to transient periods of changing T_{es} during which cardiac temperature responds faster than T_{es} (Carlsten and Grimby 1958). Additionally, there is direct evidence from experiments on the monkey and in man that whole brain and hypothalamic temperatures during cooling preceded by inner passive heating, remain elevated during the first 5 minutes of cooling while T_{es} decreases immediately (Hayward and Baker 1968, Shiraki et al. 1988). This may explain the disassociation between T_{es}, as an input to central nervous system, and the overall thermal response in the initial phase of recovery observed in the present study.

Thus, during the first 5 minutes of recovery preceded by internal heating induced by moderate rythmical exercise, T_{es} is not a precise indicator of core thermal input to the regulatory system, because brain temperature may remain unchanged. Dynamic changes in the magnitude and distribution of the cardiac output and heat production (Fig. 5, bottom), remain therefore the chief candidates determining the T_{es} response during this period.

In the present study, there was a tendency for the temperature of the esophagus to be higher (0.1 °C) during recovery from exercise in the upright position than in the supine recovery position as it has also been shown previously (Cranston et al. 1954, Greenleaf et al. 1971) . This tendency might be ascribed either to modulation of heat dissipation mechanisms or/and to an alteration in heat production between positions (Bevegård et al. 1960, Bevegård et al. 1963).

Skin temperature. Mean skin temperature ($\bar{T}_{sk}$) in each recovery position, varied within the thermoneutral range implying that its contribution to the sweating response was probably negligible (Wurster et al. 1966, c.f. Nadel et al. 1971b, Wyss et al. 1974, McCaffrey et al. 1979). Furthermore, the difference in $\bar{T}_{sk}$ between positions was insignificant throughout the 33 minutes of the recovery period. In addition, the esophageal temperature difference between the upright and recumbent positions (0.1 °C) cannot be attributed to a greater heat dissipation via conduction and convection in the supine position than while sitting upright because under both treatments ambient and steady state absolute mean skin temperatures, which determine conductive - convective heat losses, were almost identical.

Mean skin temperature ($\bar{T}_{sk}$), during both upright and supine recovery from dynamic exercise, was characterized by an initial transient rise (Fig. 8, bottom) as has been shown previously by Robinson et al. (1965), and Greenleaf et al. (1971). This rise in $\bar{T}_{sk}$ in both upright and supine recovery occurred despite a concominant decrease in skin blood flow (Fig. 9, bottom) in both postures, as measured by the Laser-Doppler flowmeter technique. A recent study (Hirata et al.1989) showed that skin blood flow in both forearms and legs, also measured with the Laser-Doppler flowmeter technique, was decreased during recovery from prolonged (30 min) moderate exercise despite a concomitant elevation of $\bar{T}_{sk}$. It was pointed out that the transient $\bar{T}_{sk}$ increase during recovery from dynamic exercise was caused by the enhanced flow of warm venous blood returning from the feet and hands to the forearms and the legs (Hirata et al.1989). They reached this conclusion because the transient rise in leg and forearm skin temperature following dynamic exercise was prevented by wrist and ankle occlusion

(Hirata et al. 1989). The initial transient rise in $\bar{T}_{sk}$ following cessation of dynamic exercise might have also been caused by an early decline in sweat rate.

Forearm skin blood flow. Upon cessation of exercise, $F_{sk}BF$ decreased in both experimental conditions. The rate of $\Delta F_{sk}BF$ decline was greater in upright position than in the supine position (Fig. 10, bottom). The enhanced $F_{sk}BF$ in the supine position may be caused by the stimulation of cardiac volume receptors as is suggested by a higher end- diastolic index ($P \leq 0.05$) in the supine than in the upright position.

Blood flow decreases as a result of sympathetic vasoconstriction in the upright posture compared with the supine in almost all vascular networks (for ref. see Rowell 1986) including skin tissue (Amberson 1943, Johnson et al. 1974b). In the current study both the diastolic (Table 2) and the mean (Fig. 4, top) arterial pressure in the brachial artery were slightly higher in the upright than in the supine position during the recovery period from exercise. Taking into account the hydrostatic pressure components acting along the vessels in the upright position implies that DAP and MAP were lower at the sites of the arterial baroreceptors in the upright position. Although not without controversy (c.f. Sanders and Ferguson 1989), it has been shown that the level of diastolic and mean arterial pressure, not the systolic and pulse pressure values, determine arterial baroreflex neuroeffector responses in normal humans (Sanders and Ferguson 1989). Thus, unloading of arterial baroreceptors in the upright position in the present study, may have contributed to decrease the skin circulation in this posture (c.f. Beiser et al. 1970).

However, Johnson et al. (1974b) showed that the application of LBSP in humans induced a $F_{sk}BF$ vasoconstrictor response which arose mainly from nonarterial baroreceptors in the cardiopulmonary region.

When $F_{sk}BF$ is expressed for a given range of esophageal temperature (Fig. 18) the slope of $F_{sk}BF$-T_{es} relationship was reduced in the supine and the cessation of $F_{sk}BF$ was delayed compared with the upright posture. Others have also found that upright position delays the $F_{sk}BF$ onset compared with the supine posture (Roberts and Wenger 1980, Johnson and Park 1981). Johnson et al. (1974a) also found a reduced $F_{sk}BF$ at a given T_{es} in the upright position as compared to supine rest.

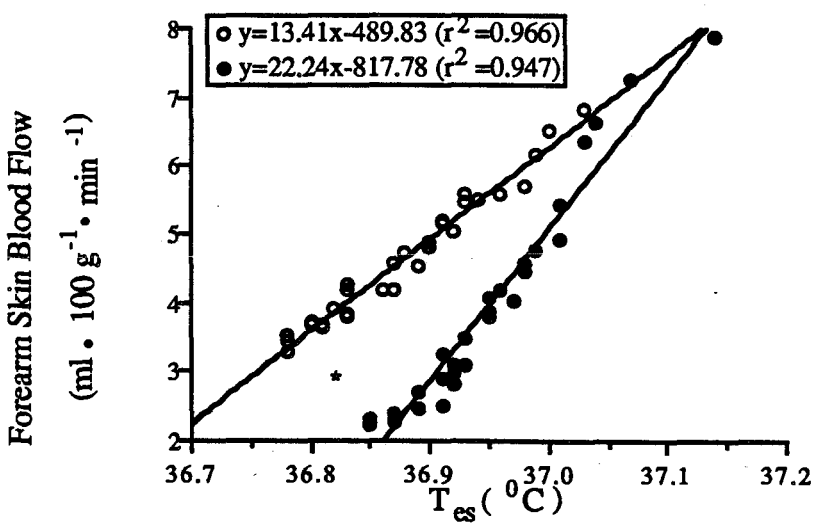


Fig. 18. Relationship between forearm skin blood flow and esophageal temperature (Tes) in groups of young males (n=8) during 30 minutes of recovery in either the upright (•) or the supine (o) position after 25 minutes of internal body heating by performing moderate (50% $\dot{V}O_{2max}$) dynamic exercise in a thermoneutral ambient condition (22±0.6 °C, 42±8% rh). *P≤0.001. Level of significance refers to comparison between the upright and supine positions for both slope and intercept.

Figure 18 shows that at higher internal temperatures the vasoconstrictive effect of an erect over a supine posture on forearm skin tissue is modified and disappears. The difference in $F_{sk}BF$ between the upright and supine positions also seems amplified at a low esophageal

temperature. There is firm evidence, however, that skin vasoconstriction due to standing or LBSP actually becomes more evident during heat stress because the absolute decrease in $F_{sk}BF$ becomes greater at an enhanced blood flow (Mosley 1969, Johnson et al. 1973). Reasons for these points of divergence between the current investigation and previous studies performed by Mosley (1969) and Johnson et al. (1973) on the resultant $F_{sk}BF$ may be the effects of interaction between thermal and cardiac receptors. A number of reflexes are important in the regulation of skin blood flow. These reflexes mainly center upon cardiovascular and thermal factors (for review see Johnson 1986). The exact mechanism(s) which caused these contradicting results are not known in the current investigation. Some speculation is, however, offered in the following sections.

In the absence of any significant effect of non-thermal factors on human temperature regulation, the relationship between the response of a thermoregulatory effector to changes in skin and core temperature (T_c) should always be indentical. A typical response of a heat loss effector to increasing core temperature at a constant skin temperature is represented by line A in Fig. 19 (top). Thus for the combination of core and skin temperature, the response will be characterized by a threshold core temperature at which the response is initiated, and a gain of the response (denoted by the slope of the relationship). Increasing the level of steady state skin temperature will normally result in a parallel shift of the effector response for similar change in core temperature, as indicated by line B; reducing skin temperature will result in a shift represented by line C, as Wenger et al. (1975) have shown. It is evident therefore that the core threshold for the effector response is determined by both skin and core temperature. Moreover, the rate at which T_c is altered through the indicated

core temperature range will alter the gain of the response. Starting with the control condition depicted by line A, increasing or decreasing T_c ($^oC \cdot min^{-1}$) whereas $\bar{T}_{sk}$ remains unaltered,may yield responses represented by lines E and D, respectively. The above discussion emphasizes the need for controlling T_c and $\bar{T}_{sk}$ when investigating the nature of the thermoregulatory effector responses. This is especially critical when the effect of non-thermal factors on the responses are being examined under varying experimental conditions.

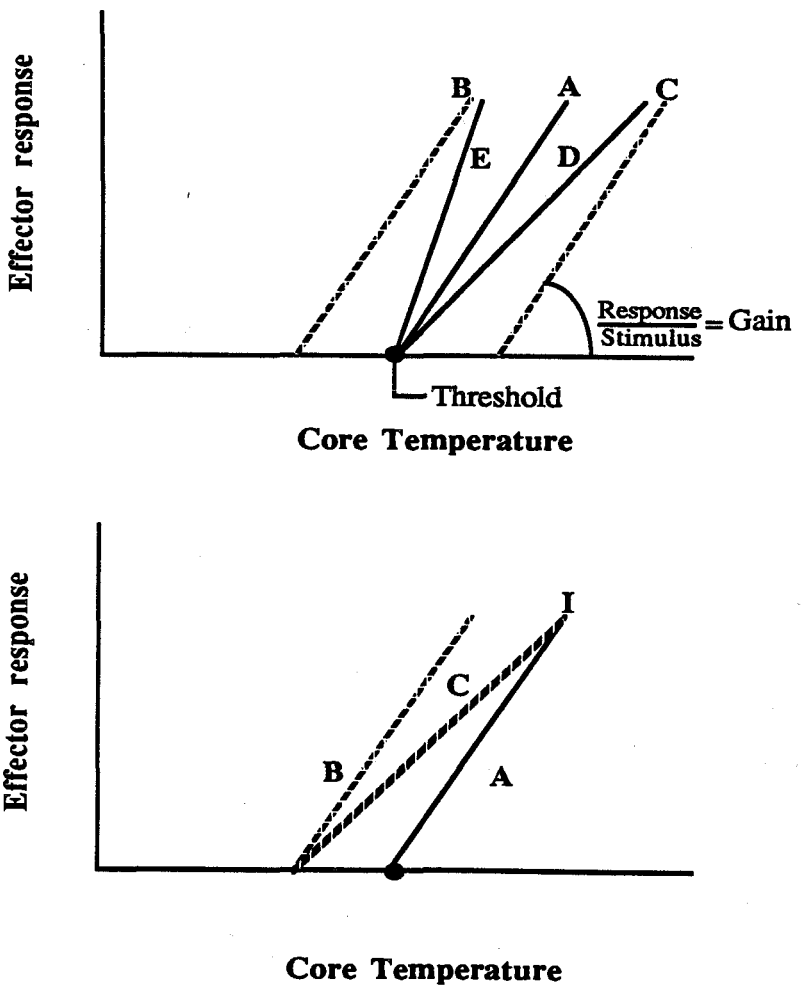


Fig. 19. Schematic diagram illustrating an idealized heat loss effector response to changes in thermal (top) and non-termal factors (bottom). See text for explanation.

In the present study, the relationship between $F_{sk}BF$ and T_{es} was examined during recovery from exercise in the upright and supine postures. Since the response of $\bar{T}_{sk}$ and T_{es} were similar in these two experimental conditions, the discrepancy observed in the $F_{sk}BF$-T_{es} response between the two conditions (Fig. 18), most probably arises due to the influence of a non-thermal factor in the sensor to effector pathway. The shift of the core threshold temperature to lower T_{es} levels and the decrease in the gain of the response during supine recovery is difficult to explain solely on the basis of an excitatory or inhibitory input synaptically converging on the sensor to effector pathway. Assuming that under a given experimental condition, the net effect of the non-thermal factor(s) was to provide an increase in the excitatory input synaptically converging on sensor-to-effector pathway, then the stimulus - response relationship represented by A in Fig. 19 (bottom) would be shifted to B, and the effect of non-thermal factor would be characterized by an earlier onset of the heat loss response with a greater magnitude of the response. This example assumes that the effect of the non-thermal factor during supine recovery is constant. The relationship derived in Fig. 18 was obtained during cooling of T_{es}. Thus, it would appear that at the intercept of lines A and C, at the onset of the postural change (I in Fig. 19 bottom), the magnitude of the effect of the unspecified non-thermal factor(s) was similar with cooling of the core in different postures. However, the effect of the non-thermal factor in the supine position (represented by line C in Fig. 19 bottom) appears to be either a progressive increase in the excitatory drive or a progressive reduction in inhibition.

Results from the present investigation also support the above speculative interpretation. During supine recovery, a leftwards shift in

the esophageal threshold temperature for skin vasodilation was observed* (Fig. 18) compared with the upright $F_{sk}BF$-T_{es} threshold. Since end diastolic index (EDI Fig. 6, top) and thoracic fluid index (TFI Fig. 6, bottom) remained unchanged throughout the entire upright recovery period, it was assumed that the slope of $F_{sk}BF$-T_{es} relationship was determined from a consant perturbation detected by cardiopulmonary receptors and introduced into the thermoregulatory system. In contrast, during supine recovery EDI (Fig. 6, top) and TFI (Fig. 6, bottom) never attained a steady state value, implying a non constant perturbation into the regulatory system finally affecting the slope of $F_{sk}BF$-T_{es} relationship. Had such changes in central venous pressure not occurred, the slope of $F_{sk}BF$-T_{es} relationship in the supine recovery position should have been parallel to $F_{sk}BF$-T_{es} slope during upright recovery and should not rotate counter-clockwise as observed according to the analysis shown in Fig. 18.

The variable non-thermal factor converging onto the temperature regulatory system assumed in the current study during supine recovery, may be caused by fluid shift between the intra- and extravascular compartments. Plasma volume measurements were not performed in the present study but plasma volume increase during the supine recovery period is suggested by the fact that stroke index (Fig.5, middle) and end diastolic index (Fig. 6, top) never attained steady state values. In addition, upon cessation of the upright cycle exercise ergometry warmup in the current study, any hemoconcentration produced (Bock et al. 1927, Thompson et al. 1928, Diaz et al. 1979, Senay et al. 1980) was probably slowly reversed during supine

* Distinct threshold values were never observed in this study due to a slow thermoregulatory recovery process. Discussion on threshold is based by extrapolation of intercept values acquired from the regression analysis.

recovery (Harrison et al. 1975, Greenleaf et al. 1977) while it remained the same during upright recovery.

The explanation regarding factors influencing the slope and the onset of the core temperature thermal response relationship proposed here, questions the claim of Hammel (1968) and Nadel et al.(1971a) that threshold shifts are caused by central events while slope changes in the $F_{sk}BF$-T_{es} relationship may only occur due to a peripheral cause.

It should be noted that a rightward shift of the $F_{sk}BF$-T_{es} relationship during supine recovery could also be caused by a nonconstant stimulus from the hypothalamic osmoreceptors influencing the operating value of core temperature since fluid shifts are associated with changes in the osmotic environment of the hypothalamus (Senay 1979). It should be emphasized in addition that the above explanation of changes in the threshold and slope of the $F_{sk}BF$-T_{es} relationship takes no account of any modification in the response of an effector caused by local factors in the periphery. This may be a crucial point since the relationship among active vasodilation, sweating and forearm flow is still not well understood.

There has been evidence that skin active vasodilation and sweat gland activity are functionally, somewhat, linked (Fox and Hilton 1958, Brengelman et al. 1981). If the latter notion was true, Figure 10 (bottom) shows that unloading the baroreceptors during recovery in the upright position enhances skin vasoconstrictor tone than rather decreases active skin vasodilation flow since for a given value of sweat rate $F_{sk}BF$ was higher in the supine than upright recovery position.

Sweating. Figure 11 (top) indicates that in the supine compared with the upright recovery position the onset of sweating is delayed to a higher internal temperature and the S_wr-T_{es} slope is increased for this situation. An interesting and original finding observed in the present investigation was that during a baroreflex manipulation by postural change, S_wr response within a given range of internal temperature, is linked more to cardiovascular parameters. In particular, the influence of increased cardiac filling pressure on sweat rate was emphasized by the fact that by the 33rd minute of supine recovery S_wr was 23.6% above its resting value while T_{es} had attained a value 20% below resting. In contrast, by the 33rd minute of upright recovery S_wr was 45% above the resting value, while at the same time T_{es} was 32% above the esophageal temperature at rest.

During upright recovery, it was found that diastolic pressure and temperature of the esophagus were the main variables from which sweat rate response was influenced. There is no reason to accept a cause and effect relationship between sweating and diastolic pressure. It is plausible, however, that diastolic pressure will be associated with changes observed in end-diastolic index in the present study. As was previously mentioned, orthostasis may be associated with hypovolemia and in the present study end-diastolic index was higher in the supine than in the upright position ($P \leq 0.05$). The mechanism by which a transition from supine to an upright position decreases body sweat rate may involve the unloading of cardiopulmonary baro-receptors, which are known to have afferent neural projections to the hypothalamus (for ref. see Gauer et al. 1970). In addition, it was recently indicated that slices of hypothalamic temperature-sensitive neurons lack any strong functional specificity and respond to various experimental situations (Boulant and Dean 1986). Thus, alteration in the activity of cardiac receptors

accompanying changes in CVP and vascular resistance, induced by assuming different postures, could modify the activity of temperature sensitive hypothalamic neurons which are involved in the central control of sweating. Alternatively, reduced cardiac filling as in the upright posture could have an indirect central and/or peripheral effect on sweat rate by releasing arginine vasopressin (AVP) from the posterior hypophysis which is neuronally connected with the hypothalamus. This latter speculation will be analysed later in this section of the discussion.

If the upright posture is considered the control condition as Groedel (c.f. Dietlen 1910) first stated and Gauer and Thron (1965) later advocated vigorously on the basis that modern man spends more time erect than supine, recumbency progressively causes significant hypervolemia. The present results showed that S_wr in the supine position is ceased earlier and this agrees with Fortney et al. (1981) who showed that hypervolemia tends to delay the onset of sweating on the forearm. Recovery in the upright position decreased the change in sweat rate for a given change in esophageal temperature (Fig. 10, top) which is also in accord with the results of Fortney et al. (1981), who showed that hypovolemia decreases the slope of S_wr-T_{es} relationship in the forearm.

Solack et al. (1985) also found a reduced S_wr-T_{es} slope subsequent to the onset of LBSP. The S_wr-T_{es} slope decrease when LBSP was applied, was interpreted by Solack et al. (1985) as a change neither in the origin nor the slope of S_wr-T_{es} contours. These investigators were the first to suggest that the observed reduced central venous pressure upon LBSP application initiated a time - dependent response due to hormonal action rather than a neural reflex. Subsequently, however, any hormonal effect on the observed reduced S_wr-T_{es} relationship was discarded by speculation (Solack et al.

1985). It was finally suggested that a reduction in central venous pressure might induce a hypothalamic osmotic gradient which would provide a mechanism of S_wr inhibition during LBSP. Such speculation is insufficient to explain the similar S_wr result observed by reducing venous pressure in the present study. Despite moderate isotonic exercise increasing osmolality, which returns toward control levels during recovery (Harrison et al. 1975); passive tilt at 40° (Bie et al. 1986), and inflation of an antigravity suit had no effect on plasma osmolality (Kravik et al. 1986, Geelen et al. 1989), compared with adoption of a supine and upright position, respectively.

Possible humoral effects. The present study does not distinguish between a direct effect of loading cardiopulmonary and/or arterial baroreceptors and an indirect effect of humoral agent release, secondary to such baroreceptor stimulation, which may affect S_wr or T_{es} during supine recovery from dynamic exercise. Inhibition of release of arginine vasopressin (AVP), renin-angiotensin-aldosterone, and norepinephrine (NE) may be associated with recumbency compared with standing erect. All these humoral agents could exert a strong effect either centrally or peripherally on the sweating response as observed in the present study. These neuro-humoral effects have remained unstudied in this investigation.

Several investigators (Segar and Moore 1968, Robertson and Athar 1976, Davies et al. 1976,1977) have found that passive or active orthostasis elevates AVP, while several other groups (Goldsmith et al. 1982-1984, Leimbach et al. 1984, Bie et al. 1986) have found no correlation between CVP and plasma AVP in humans.

In the present study AVP was not measured but it appears likely that a recovery in the upright position had no effect on plasma AVP because Goldsmith et al. (1982) stated that AVP is only elevated when orthostasis or

LBSP is long or severe enough to cause hypotension which was not the case in the present study. Therefore, in the present investigation, if plasma AVP changes occurred, they would have a minimal, if any, effect on sweating because AVP does not influence sweat gland secretion (Sato 1977) and on the other hand may elicit ambiguous effects on body temperature (Kasting et al. 1980, Lipton and Glyn 1980) when is administered centrally.

Although plasma aldosterone is elevated in erect humans (Sassard et al. 1976) and aldosterone reduces sweat rate at the gland level (Dobson and Slegers 1971), it cannot account for the reduced S_wr-T_{es} slope (Fig. 11, top), since aldosterone requires six hours in order to become effective on the sweat gland (Sato and Dobson 1970).

Orthostasis in humans is associated with a significant increase in plasma concentration of angiotensin II (Sancho et al. 1976) and NE (Vlachakis 1979, Péronnet et al. 1989), which each may have a central inhibitory effect on sweating (Bligh et al. 1971, Lin et al. 1980a,b, Owen et al. 1989) producing a smaller S_wr-T_{es} slope in the upright position than in the supine, as it was observed in the present study.

However, because the present study explored the cessation and the decay of sweating for a given range of esophageal temperatures rather than the onset and the rise of S_wr-T_{es} relationship, the decay of sweating might be faster in the upright position than in the supine position due to an inhibitory effect of the sympathomimetic hormones (ANG II, NE).This possibility further suggests that the S_wr-T_{es} relationship between upright and supine position observed during cooling in the present study will be reversed during a heating procedure in the same positions.

An alternative explanation is that an elevated S_wr enhances $F_{Sk}BF$ (Fox and Hilton 1958, Brengelman et al. 1981), while vasoconstriction in

turn may be associated with inhibition of sweating (Elizondo 1973). Although the present study cannot solidly refute the existence of these factors, it appears that inhibition of sweating during upright recovery was not caused by any of them. First, skin ischemia may cause local inhibition of sweating by suppressing acetylcholine secretion (Elizondo 1973). It is doubtful, however, that skin vasoconstriction shown during upright recovery, in the present study, might have caused tissue ischemia since sweating has been reported to be unaffected during arterial occlusion (Beaumont and Bullard 1966).

In summary, it may be argued that compared with the supine position, sympathetic arousal in the upright position induced blood volume and unidentified humoral changes which caused a reduced $F_{Sk}BF$ and an earlier sweating at a reduced rate. In both recovery positions T_{es} was well related with the S_wr. Doubt about the significance of T_{es} as a valid measure of core temperature influencing the thermal response in some experimental circumstances also arises.

The effect of mild static exercise

Cardiovascular response to static exercise.

Overall response . The cardiovascular response to mild static exercise compared with the idle condition following dynamic exercise consisted of a rise in blood pressure (Fig. 11, top) which was associated with a parallel increase in systemic vascular resistance (Fig. 11, bottom), as previously observed by many investigators (for ref. see Lind 1983). By the 33rd minute of recovery, heart rate, mean arterial pressure, and total peripheral resistance were respectively 11%, 6%, and 8.5% higher during static

exercise than during idle recovery. However, despite a significantly higher HR value during recovery with static exercise than during idle recovery, as previously has been found (for ref. see Lind 1983), the cardiac index was similar in both experimental conditions in contrast with other reports (Helfant et al. 1971, Perez-Gonzales et al. 1981). Cardiac index remained unaltered because there was a tendency ($P \leq 0.07$) in the stroke index (SI) to be higher while idle than during recovery with static exercise. Stroke index by the 33rd minute of recovery was 13.6% higher during idle than during static exercise recovery .

Stroke index. The discrepancy between the current investigation and similar reports in the literature of stroke index response to mild static exercise might be attributed to the different experimental design. In particular, dynamic exercise, which in the present study was used as means of internal heating, elevates mean arterial pressure and internal body temperature both leading to hemoconcentration (for ref. see Harrison 1985). These changes may be expected to be slowly reversed during recovery. Removal of metabolic by-products of muscle activity by the systemic circulation will also tend to decrease a pressor response which, in turn, will restore vascular compartment fluids. The faster esophageal temperature decreases, the more rapidly such hemoconcentration would be reversed. Consequently, cardiac filling would be enhanced stimulating cardiac baroreceptors further reducing MAP by concomitantly enhancing stroke index and attenuating heart rate. Evidence of these proposed mechanisms is indicated in Fig. 11 (top), Fig. 12, (middle), and Fig. 13.

Mean arterial pressure. It appears that the pressor response to static exercise was attenuated in the present study, because there was an interaction

between static exercise and posture evident in the recorded MAP (Fig. 14, top).

Interaction of cardiopulmonary, carotid, thermal, and somatic reflexes might have caused the lack of MAP response beyond the orthostatic reaction during static exercise in the upright recovery position (Fig. 14, top). An investigation directly relevant to the present study (Freyschuss 1970) showed, inexplicably, that a static muscle contraction (70% MVC) during 60° head-up tilt did not significantly affect the amplitude of the blood pressure response beyond the orthostatic reaction compared with the supine position either while idle or with static exercise of identical (70% MVC) intensity .

In the present study, individual analysis of experimental groups* showed that static exercise during recovery in the upright position did not increase the MAP, because the total peripheral resistance remained unaltered compared with upright idle recovery since the CI between the respective two experimental conditions was almost identical ($P \leq 0.90$). The expected myogenic vasoconstrictive response was not observed during upright recovery with static exercise compared with upright recovery while idle, probably because of an inhibitory influence of thermal reflexes acting on the sympathetic tone of peripheral vessels (for review see Rowell 1983). This suggestion is supported by the fact that esophageal temperature during recovery with static exercise in the upright position was significantly higher ($P \leq 0.05$) than T_{es} in the same position while idle. On the contrary, variation in T_{es} was similar between supine recovery while idle and supine recovery with static exercise and with supine and upright positions while idle, where a

* These individual experimental groups were: upright idle, supine idle, upright static exercise, and supine static exercise. See also Fig. 2 cells I, II, III, IV, respectively

vasoconstrictive response and consequent elevation of MAP occurred from the somatic and gravitational stress exerted. The effect of elevated esophageal temperature is probably further shown in Fig. 14 (middle) where the heart rate increase was disproportionally higher during static exercise in the upright position than during static exercise in the supine position compared with recovery while idle in the upright and supine position, respectively. An increase in body temperature has been previously shown to produce a direct rise in heart rate (Jose et al. 1970, Geladas and Banister 1988). The disproportionate difference between the heart rate during recovery with static exercise in the upright position and the supine position compared with the heart rate response during their respective idle conditions, might be also due to an inhibitory effect of the arterial baroreflex on the cardiac frequency during recovery in the supine position with static exercise where MAP is elevated. It has been shown that elevation of MAP induces predominantly parasympathetic withdrawal (Glick and Braunwald 1965, Robinson et al. 1966) and heart rate decrease. This latter, however, mechanism suggested is highly controversial (for references see Mancia and Mark 1983).

Stimulation of mesenteric 'stretch' receptors might have contributed to a higher vasoconstrictive response (Andrews et al. 1972) to myogenic reflex during static exercise in the supine position since thoracic fluid index was lower ($P \leq 0.002$) in this position than during static exercise in the upright position.

In summary, during recovery with static exercise, the MAP did not increase more than the initial pressor response to upright recovery while idle or static recovery in the supine position, both, relative to idle supine recovery. The total peripheral resistance did not respond to the myogenic input induced by the static exercise stimulus in the upright position probably

because of an inhibitory influence initiated by a thermal reflex. The participation of a cardiogenic reflex on total peripheral resistance unresponsiveness is questioned. The concomitant disproportionate rise in the heart rate, above the resting values, during static exercise in the upright recovery compared with recovering with static exercise while supine, may be attributed to an excitatory thermal input in combination with a removal of the inhibitory carotid reflex.

Thermoregulatory response to static exercise.

T_{es}, T_{sk}, and oxygen uptake. Mean skin temperature attained a steady state value within 5 minutes of recovery in both conditions and it was insignificantly higher (0.54 °C) during recovery with static exercise than while idle (Fig. 15, bottom). Thus, the peripheral temperature was similar and within the thermoneutral range (32.5 to 33 °C) in both experimental conditions.

Conversely, esophageal temperature was significantly higher ($P \leq 0.05$) in the static exercise recovery condition than in the idle recovery condition (Fig.15, top). This difference was probably caused by a significantly ($P \leq 0.01$) higher oxygen uptake during recovery with static exercise than while idle. Specifically, oxygen uptake during static exercise was 65 ml•min^{-1} higher, equivalent to a 0.17 °C elevation of the internal temperature above that observed in idle condition by the 33rd minute of recovery*. Indeed, T_{es} measured during recovery with static exercise was

* In this calculation, it was assumed that 1 liter of oxygen is equivalent to 4.82 kcal and all of the metabolic energy produced during isometric exercise is degraded to heat. Mean group body weight and specific heat of tissue were equal to 70.24 kg, Table 1, and 0.80 kcal · °C^{-1} · kg^{-1}, respectively (c.f. Geladas and Banister 1988). Mean oxygen uptake values for both conditions were considered in the calculations.

0.16 °C higher than recovery while idle by the 33rd minute of recovery. The elevated esophageal temperature during recovery with static exercise seemed to enhance conductive - convective and evaporative heat losses (Fig. 16).

Forearm skin blood flow. Clement and Pannier (1980) showed in anesthetized dogs with vagal input abolished and a constant pressure maintained in the carotid sinuses, that skin blood flow was significantly decreased during static muscular contraction. Similarly, it was found, in the same type of vagotomized animals, that a somatopressor reflex from the muscle induced a skin vasodilatory response which was converted to a constrictor response when baroreflex activity was abolished (Nutter and Wickliffe 1981). In a study using humans, static hand contraction elicited either a slight increase in skin sympathetic nerve activity or no change (Delius et al. 1972).

Based on the above studies, it seems that during static exercise sympathetic skin nerve activity is minimally enhanced whereas baroreflex activity increases $F_{Sk}BF$. It was recently pointed out that a 30% MVC of static handgrip in humans considerably increased the blood flow in skin at 39°C (Taylor et al. 1989). These authors intentionally discounted any vasodilatory effect of the baroreflex since static exercise performed in normothermic subjects with thermoneutral skin temperature does not enhance skin conductance(Taylor et al. 1989). Rather they suggested that the elevation of arterial pressure observed would have some local effect on the skin vessels which would be susceptible to dilation due to a thermally induced high distensibility (Taylor et al. 1989).

Thus, it might be expected that $\Delta F_{sk}BF$ during recovery with static exercise, where MAP is elevated, may be higher than recovery while idle under hyperthermic conditions. It was also expected that during

normothermia $\Delta F_{sk}BF$ would tend to be similar during recovery both with exercise and while idle.Skin blood flow difference in the forearm ($\Delta F_{sk}BF$) tended initially to be lower during static exercise recovery than during idle recovery whereas the relationship was reversed from the 10th minute onwards (Fig. 16, middle). The initial acute drop in $F_{sk}BF$ observed in the present study (Fig. 16, middle) during static exercise while recovering from heat strain may be ascribed to a skin venoconstriction which has also previously been observed to occur in the initial phase of static exercise (Freyschuss 1970, Seaman et al. 1973) and gradually subsides (Lorentsen 1975). Figure 16 (bottom) indicated that when a higher mean arterial pressure was taken into account, skin forearm conductance tended to be still higher during recovery with static exercise than while idle.However, Fig. 20 shows that a tendency for a higher $F_{sk}BF$ during static exercise recovery than in idle recovery was mainly affected by a higher T_{es} in the former type of recovery.

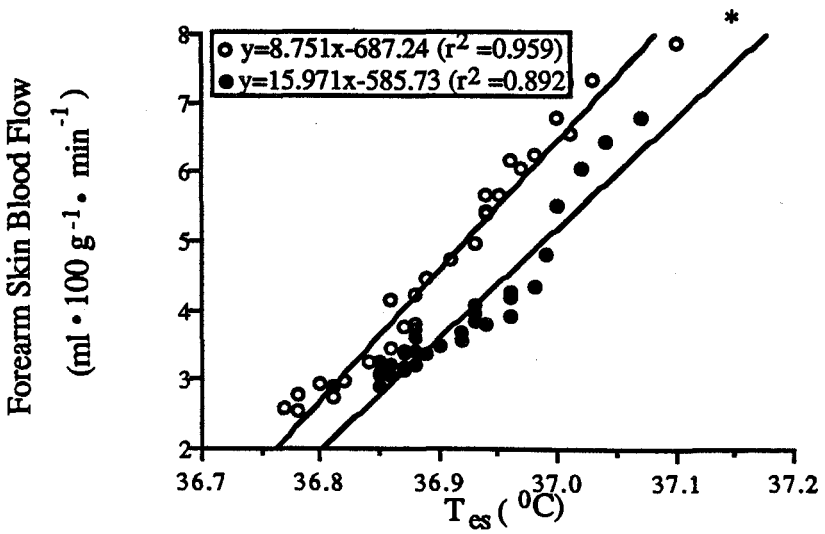


Fig. 20. Relationship between esophageal temperature (Tes) and forearm skin blood flow and in groups of young males (n=8) during 30 minutes of recovery either with static exercise (●) or while idle (o) after 25 minutes of internal body heating by performing moderate (50% maximal $\dot{V}O_2$) dynamic exercise in a thermoneutral ambient condition (22±0.6 °C, 42±8% rh). *P≤0.016. Level of significance refers to the comparison of idle and static exercise during recovery from the relative hyperthermia for only the slope (*).

It may be seen (Fig. 20) also, that during recovery with static exercise the rate of rise in forearm skin blood flow is temperature dependent with a threshold approximate of 36.95 °C. This is probably due to the fact that arterioles are more distensible at high body temperatures and arterial pressure was higher during the static exercise than idle condition.

The results of the present study suggest that there is a vasoconstrictive effect of myogenic origin during static exercise which might be inhibited by a thermal reflex. When the effect of thermal reflex on vasomotor tone is considered, the myogenic excitatory vasoconstrictive reflex on peripheral vessels prevails over a contradictory inhibitory effect originating from a systemic baroreflex as discussed previously. The role of cardiac reflex is ambiguous because end-diastolic index tended to be lower ($P \leq 0.07$) during recovery with static exercise than while idle.

Sweating. The present study investigated whether baroreflex and muscular input produced by static exercise modified heat dissipating mechanisms. It was found (Fig. 17, top) that during static exercise while recovering from thermal stress sweat rate was remarkably enhanced for a given esophageal temperature.

When comparing upright and supine static exercise recovery conditions it was found that MAP in the brachial artery was similar in both postures. This in turn means that the baroreceptors were unloaded in the upright comparised with the supine static exercise condition. Therefore, and because S_wr was similar in both exercise positions it seems likely that during static exercise S_wr was determined by factors other than arterial and/or cardiopulmonary baroreceptor activity.

It appears, therefore, that the static exercise *per se* enhances sweat rate. There is indirect evidence supporting the suggestion that the higher

sweat rate present during recovery with static exercise than while idle might have been due to a neuromuscular stimulus. Autocorrelation in the T_{es}-S_wr relationship was removed when diastolic pressure was added as a contributing factor to the regression of sweat rate on T_{es} in the static exercise condition. However, a cause and effect relationship between diastolic pressure and sweat rate should not exist, because any important influence of a cardiovascular change has been already disregarded from the above discussion. It may be suggested, nevertheless, that as muscular contraction elicits a myogenic reflex which increases the arterial pressure, the same muscular event evokes, in parallel, a reflex which leads to a higher sweat rate during recovery with static exercise than while idle.

In order to verify the possible modulatory effect of direct muscular input to the thermoregulatory center, two of the subjects of the present study

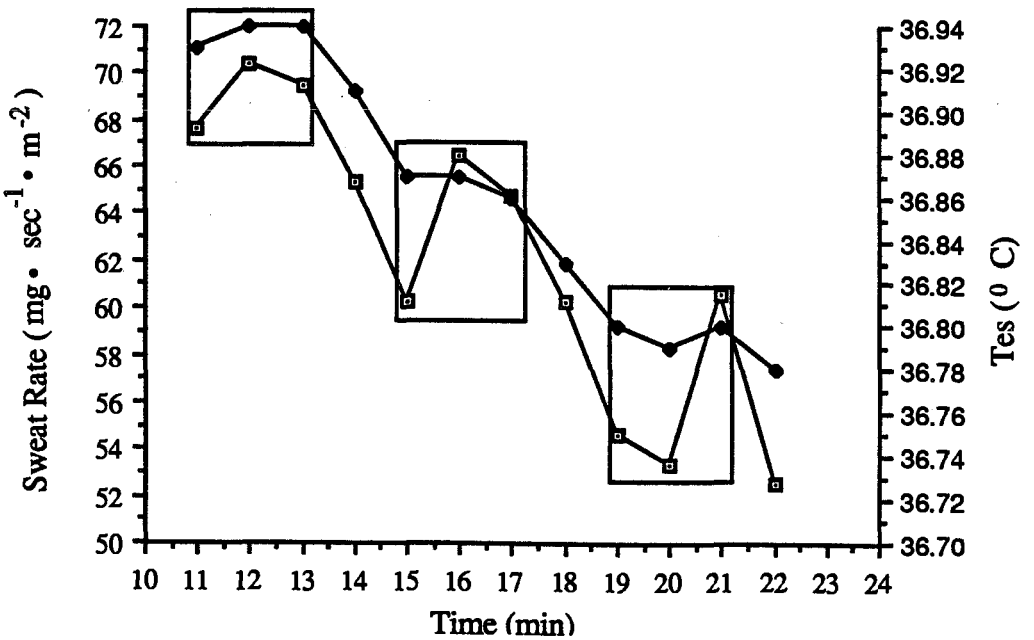


Fig. 21. Composite data from an experiment with one representative subject. Individual changes in esophageal temperature (Tes) sweat rate ( ) during recovery with intermittent static exercise (161N) after 25 minutes of body heating resulting from moderate (50% maximal $\dot{V}O_2$) dynamic exercise in a thermoneutral ambient condition (22±0.6 °C, 42±8% rh). Square inserts represent bouts of 2 minutes static contraction.

were requested to perform intermittent static exercise (161N) during recovery following 25 minutes of internal body heating by dynamic exercise. Two minute periods of static exercise were alternated with 2 minutes of idleness during 33 minutes of recovery from heat stress. Both subjects responded in the same fashion. Fig. 21 shows the sweat rate and esophageal temperature response from the 10th to the 22nd minute of recovery in one of these subjects, during which the response was more pronounced. It appears here that the sweat rate response to the exercise stimulus is much more pronounced than is that of T_{es}. Static exercise serves to enhance sweating rate remarkably followed by a sharp decline in the rate during the idle interval. The decay of T_{es} is considerably slowed during static exercise intervals. These dependent effects of static exercise are thus obviously out of phase since increased sweating should be the logical response to an elevated T_{es}. The opposite is observed, and the two parameters (S_wr and T_{es}) seem disassociated. However, the static exercise-induced impediment of T_{es} decay may have, in turn, affected the sweating response.

A direct thermal input from the exercising muscle might have caused enhanced sweating during static exercise although the size of muscular stimulus was quite small. Jessen et al. (1983) found in goats that deep thermosensitive elements in the legs feed the temperature regulating system. In addition, there is firm evidence that one portion of type III and IV afferent fibers in skeletal muscle are temperature sensitive, while another portion evokes cardiovascular responses (for review see Mitchell and Schmidt 1983). Muscle temperature during recovery following dynamic exercise is higher than esophageal temperature (Saltin and Hermansen 1966). Based on these concepts, it would be expected that static exercise would increase muscle temperature further, above esophageal temperature, during recovery,

driving the sweat rate at a higher level than during recovering idly, particularly if circulatory occlusion occurred due to static exercise.

Similarly, Smiles and Robinson (1971) suggested that a stimulus of neuromuscular origin is an essential part of the thermoregulatory system in man during exercise. A number of other studies (Robinson et al.1965, Saltin et al.1970), nevertheless, found a weak relationship between muscle temperature and sweating measured from weight loss during and after dynamic exercise. Finally, a long standing hypothesis, based on histological evidence, suggesting that thermoreceptors may exist in the deeper skin plexuses which are sensitive to an elevation of temperature as warm venous blood return from the exercising muscle (Bazett 1951).

Possible humoral effects. The higher sweat rate response with static exercise than while idle during recovery might be attributed partially to non-thermal factors. Evidence adduced for this is that, by the 33rd minute of recovery with static exercise sweat rate was 51.2% above the pre-trial resting value whereas T_{es} at this time was only 24.25% above the pre-trial resting. In contrast, by the 33rd minute of recovery while idle the $S_{w}r$ had attained a value 22.05% above the resting value, whereas T_{es} was 10% below the pre-trial resting value.

Research on hormonal changes induced by static exercise is sparse and contradictory. It may be assumed that, in the current investigation an increase in plasma catecholamines was probably neglible due to the low intensity of static exercise sustained (c.f. McDermott et al. 1974, Few et al. 1975, Watson et al. 1979), since catecholamines increase only when static exercise induces fatigue (Kozlowski et al. 1973).

Although a single injection of arginine vasopressin in the intracerebroventricle kept the rectal temperature in rabbits high (Lipton

and Glyn 1980) for 2 hours of recovery from hyperthermia, the inital increase in AVP caused by static exercise dramatically subsides from the 3rd minute onward (Nazar et al.1989). Adrenocorticotropine hormone (ACTH) may also be secreted during static exercise (Few et al. 1975) or may not (Nazar et al. 1989). Either way ACTH decreased rectal temperature in rabbits when administered centrally (Lipton and Glyn 1980). Vasoactive intestinal peptide (VIP) -immunoreactive nerves have been identified around the eccrine sweat glands (Vaalasti et al. 1985). In addition, VIP evoked profound eccrine sweat secretion *in vitro* (Sato and Sato 1987), whereas centrally infused VIP increased rectal temperature in rabbits (Lipton and Glyn 1980). Of course, the effect of static exercise on VIP response is completely unknown.

Based on existing knowledge, therefore, it appears that the differences in sweat rate between recovery with static exercise and idle recovery observed in the present study cannot be caused solely by humoral agents, the secretion of which may have been initiated by static contraction. The matter, therefore, must remain, somewhat equivocal due to lack of adequate evidence.

In summary, Static exercise compared with idle recovery increased MAP by increasing total peripheral resistance. The increase of MAP during static exercise was lower than expected mainly because static exercise in the upright position did not elicit any pressor response beyond the orthostatic arterial pressure reaction. This may be due to some interaction between thermal, cardiac and baroreflexes. In addition, static exercise during recovery from thermal stress compared with idle recovery modified the heat dissipating mechanisms. Changes in heat loss through convection and

conduction between the experimental conditions was not identified during the 33 minutes of recovery period probably because of competing myogenic vasoconstrictive and thermal vasodilating effect respectively which originate from the higher esophageal temperature observed during recovery with static exercise. Evaporative heat loss was enhanced during recovery with static exercise than while recovering idly. This sweat rate increase was not associated with any cardiovascular event accompanying static exercise. The higher slope in the T_{es}-$S_w r$ relationship observed during static exercise in recovery from heat stress than while idle during recovery, may be mainly attributed to a reflex of neuromuscular origin, secondary to a thermogenic metabolic effect. Humoral factors might have contributed to a high sweating during recovery with static exercise but there is lack of adequate information.

6.0 SUMMARY

The aim of the present study was to investigate the separate or combined effects of posture and static exercise on heat dissipating mechanisms in humans.

It was demonstrated that the cardiovascular response induced by postural changes was similar with the cardiovascular response induced by static exercise. Both posture and static exercise modulated the heat loss mechanisms of sweating and forearm skin blood flow. The manner in which the modulation is effected appears to depend on the nature of afferent information.

Specifically, at elevated body temperature the cardiovascular adjustment to gravitational stress and static exercise may arise from similar changes in end-diastolic volume, diastolic pressure, and peripheral resistance.When postural changes from supine to upright were initiated in subjects, withdrawal of cardiopulmonary and/or arterial baroreflex probably modulated both forearm skin blood flow and rate of sweat secretion. When static exercise was performed, forearm skin blood flow was influenced by an interaction between cardiopulmonary, thermal and myogenic reflexes. Secretion of sweat was affected from thermal reflexes originating not only from the esophageal temperature. It was assumed that muscular contraction might also be important.

Assuming that active skin vasodilation is linked somewhat to sweating as others have suggested, the results of the present study show that an unspecified input associated with the upright position and static exercise conditions modulated mainly skin vasoconstriction tone, since forearm skin

blood flow was lower for a given value of sweat rate and esophageal temperature in the above two conditions when compared with the supine and idle conditions, respectively.

Among the four experimental conditions, there was a tendency for selective activation of either the sudomotor or the skin vasoconstrictor neural system with concomitant suppression of the activity of the other system. Similar results were obtained by Bini et al. (1980) during the recording of multiunit sympathetic activity of human skin nerve fascicles innervating the sudomotor and the vasoconstriction system in subjects exposed to different ambient temperatures. The underlying mechanism(s) of such phenomena remain entirely unknown but the teleological appeal of uniform homeostasis between overlapping regulatory systems such as the thermoregulation and the cardiovascular is apparent.

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GLOSSARY:

T_{es} : Esophageal temperature

$\bar{T}_{sk}$: Skin temperature

T_c : Core temperature

T_{re} : Rectal temperature

S_{wr} : Sweat rate

LBSP : Low body subatmospheric pressure

$\dot{V}O_{2max}$: Maximum oxygen consumption

MAP : Mean arterial pressure

DAP : Diastolic arterial pressure

SAP : Systolic arterial pressure

CI : Cardiac index

SI : Stroke index

HR : Heart rate

MVC : Maximum voluntary contraction

$F_{Sk}BF$: Forearm skin blood flow

$\Delta F_{Sk}BF$: Relative Forearm skin blood flow

CVP : Central venous pressure

TFI : Thoracic fluid index

EDI : End diastolic index