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THE EFFECTS OF AMBIENT TEMPERATURE ON POST-EXERCISE BLOOD PRESSURE

by

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A thesis submitted to the School of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Master of Science

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to myself

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ABSTRACT

Although the cardiovascular changes associated with dynamic exercise have been investigated, there still remain many unanswered questions with respect to blood pressure following exercise. This study examined the effect of temperature on systolic (SBP), diastolic blood pressure (DBP), heart rate (HR), oxygen consumption (VO_2), and skin temperature (Tsk) in 10 healthy, young males (23-28 years of age), during a 60-minute supine post-exercise recovery period. On separate occasions, 20 minutes of treadmill exercise at a heart rate of 140 beats per minute, preceded a 60-minute recovery period in a supine position conducted at either 18°C, 27°C, or 40°C. Results indicated that mean SBP was significantly more above baseline values at 18°C than at either 27°C or 40°C. However, no significant difference in SBP was observed between recovery at 27°C and 40°C. Also, no significant differences were observed among the three conditions for mean DBP. HR decreased progressively during recovery but remained above baseline values except at 18°C . No significant differences among recovery conditions were observed for VO_2. Mean skin temperatures were significantly different among the three recovery conditions except immediately upon entering the chamber. It was concluded that recovery at 18°C maintained post-exercise SBP above baseline values while 27°C and 40°C caused a decrease in post-exercise SBP with respect to baseline values. DBP and VO_2 during recovery were not influenced by temperature. HR decreased gradually post-exercise with the lowest HR observed during cold exposure. Therefore, for the conditions of this experiment temperature had a significant effect on SBP, HR and skin temperature all of which effectively appeared to be associated with peripheral vasodilation/constriction mechanisms.

INTRODUCTION

There are relatively few studies which have described the behaviour of post-exercise blood pressure in humans (Hannum and Kasch, 1981 and Bennett, Wilcox and MacDonald, 1984). Immediately following exercise, blood pressure decreases rapidly and then gradually returns to resting levels (Korner, 1952). However, some studies have also reported reductions in blood pressure below pre-exercise values for extended periods of time (Boyer and Kasch, 1970, Paulev, Jordal, Kristensen and Ladefoged, 1984, Choquette and Ferguson, 1973 and Fleg and Lakatta, 1986). In these studies, the average reduction of post-exercise systolic blood pressure below resting values was approximately 11 mmHg (Kaufman, Hughson and Schaman, 1987). The post-exercise reduction in blood pressure lasted for 90 to 120 minutes as a result of dynamic exercise (Hannum and Kasch, 1981).

A few studies have investigated the possible mechanism(s) which may be involved in the rapid decrease of the post-exercise blood pressure (Friedman et al, 1987, Kral et al, 1966 and Paulev et al, 1984). These include decreased catecholamines, decreased blood volume, baroreceptor resetting and vasodilator substances. However, several studies have attributed the reduction in blood pressure following exercise primarily to vasodilation and venous pooling (Fitzgerald, 1981).

The added physiological load imposed by either heat or cold stress on the body could also influence post-exercise blood pressure. The combination of heat stress and exercise would result

in reflexive vasodilation that persists following exercise, leading to a further decrease in post-exercise blood pressure (Kilgour et al, 1993). However, the application of a cold stress could cause the opposite reaction (Rowell, 1986). There would be peripheral vasoconstriction, which as a result would redirect blood to the central circulation and cause an increase in stroke volume resulting in the post-exercise blood pressure to remain above resting values. Therefore, various thermal conditions during exercise recovery may result in cardiovascular adjustments different than those observed under neutral temperatures.

Several studies have investigated the cardiovascular responses to exercise using different thermal stresses (Kruk et al, 1990, Harrison, 1986, Nadel et al, 1979, and Young, 1990). However, it appears that very few studies have investigated the behaviour of the post-exercise blood pressure response to various thermal stresses. Also most studies evaluated post-exercise blood pressure in a seated position. The aim of this study was to observe the effects of exercise and temperature during recovery to determine their influence on blood pressure response. This study was conducted to test the hypothesis that ambient temperature does have a significant effect on blood pressure following exercise. The purpose of this study was to determine (1) the influence of exercise on post-exercise blood pressure (2) the influence of ambient temperature on post-exercise blood pressure in young males while recovering in a supine position (3) if peripheral vasoconstriction/vasodilation mechanisms are involved.

METHODOLOGY

Subjects

Ten apparently healthy male subjects between the ages of 20 and 35 years old were recruited for this study. All subjects met the following criteria: answered "no" to all questions on the modified Par Q (Jette, Quennevillle and Sidney , 1991b), were non-smokers, had a resting blood pressure less than 140/90 mmHg, were not taking any medication which could alter the cardiovascular response to exercise, and had a normotensive blood pressure response to a moderate level (stage A of CAFT) of exercise (Jette, Landry and Sidney, 1991a). An informed written consent was obtained from each subject prior to participation in this study. Ethics approval was granted by the University of Ottawa's Ethics Committee.

Materials

Resting blood pressure and heart rate were measured using a standard sphygmomanometer and stethoscope. The modified Canadian Aerobic Fitness Test (CAFT) (Jette, Landry and Sidney, 1991a) was administered to ensure that subjects exhibited a normotensive response to exercise (change in BP following execise <45mmHg). Predicted maximal oxygen consumption was calculated from the results of the last stage performed of the CAFT (Jette et al, 1976). Dynamic exercise was performed on a Quinton Treadmill

(Model 18-54, Seattle, Washigton). The speed of the treadmill was set to elicit an exercising heart rate of 140 beats/min for each subject. A transmitting/receiving tachymeter watch (Polar electro Technology Sport Tester PE 3000: Nor Am Patient Care Product, Oakville, Ont.) was used to continuously measure heart rate during the exercise and recovery period. Skin temperatures were continuously measured using an automated Data Acquisition Control Unit (Hewlett Packard 3497A) and a Hewlett Packard 9000, PC-31 which recorded, calculated, and printed the corresponding temperatures. Each subject was fitted with the Finapres BP monitor 2300 (Ohmeda, Louisville, Colorado) finger cuff to continuously measure blood pressure and heart rate throughout the recovery periods. Hartmann and Bassenge (1989) studied the accuracy and reliability of the values obtained from the finapres models by comparing them with intra-arterial measurements in more than 300 patients. The values were in agreement. Subjects recovered in a controlled CAN-TROL environmental chamber (Environment Systems Limited, Toronto, Ont.) in the supine position on a rope cot.

Oxygen consumption was measured using a Tissot tank (Warren and Collins inc, Braintree, Ma.) and gas bags and an O_2 analyzer (S-3A1, Ametek, Pittsburgh, Pa) and a CO_2 analyzer (CD3A, Ametek, Pittsburgh, Pa.).

Procedure

The testing protocol was conducted over a period of four sessions. At the preliminary session, subjects were fully informed

of all testing procedures and thoroughly briefed on their involvement in the study. The subjects completed the modified Par Q (Jette, Quenneville and Sidney, 1991b), and an informed written consent. They were then seated for a period of five minutes, after which resting blood pressure and heart rate were recorded using a standard sphygmomanometer and stethoscope to ensure that subjects were normotensive. The subjects were then administered the Canadian Aerobic Fitness Test (Jette et al., 1991a). The blood pressure response to the first stage of the Canadian Aerobic Fitness Test (CAFT) was utilized to evaluate blood pressure behaviour to dynamic exercise (Jette et al., 1991a). Subjects who exhibited an exaggerated blood pressure response (SBP change >45mmHg) were not allowed to participate in the study.

Weight, height and skinfold measurements were recorded using the procedures outlined in the CAFT (CSTF, 1986).

Subjects were then initiated to the treadmill. They performed a progressive ten minute exercise session to determine the intensity (speed) required to achieve a heart rate of 140 beats/min. They were also familiarized with the controlled environmental chamber, the resting cot, the Polar Sport Tester, the Finapres BP 2300 and skin temperature thermistors.

All experimental sessions took place between 7:00 A.M. and 12:00 P.M. Subjects were requested to refrain from exercising, smoking and ingesting stimulants such as coffee and alcohol for 12 hours prior to testing.

During sessions 2,3, and 4, subjects were seated for a period

of 5 minutes, then supine for an additional five minutes, after which resting blood pressure and heart rate were recorded. These blood pressure measurements were used as the baseline values for the analysis. Next, surface skin temperature thermistors were placed on the mid-forehead, mid-tricep, the chest and over the rectus femoris muscle (Kilgour et al, 1993) and the Polar watch was fastened to their left wrist.

The subjects then performed a twenty minute run on the treadmill. The speed of the treadmill was progressively increased until they attained a heart rate of 140 beats/min (room temperature 22°C-24°C). After the twenty minute run the treadmill speed was gradually reduced to a walking speed for a one minute cool-down. During this period of walking the Finapres Bp monitor finger cuff was fitted on the second digit of the subject's left hand. Immediately following exercise, subjects were moved into the thermal chamber where they recovered in the supine position on a rope cot for 60 minutes. The arm was placed on the chest to maintain the finger and the cuff at the level of the heart throughout the recovery period. The transition following exercise into the chamber was completed within one minute. Subjects were then exposed at separate sessions, in a random manner, to one of the three different ambient temperatures: 18, 27 and 40 degrees celsius. After 30 minutes during the recovery, the Finapres BP monitor cuff was removed and fitted on the second digit of the subject's right hand to avoid possible finger necrosis. The transition was completed within one minute. Oxygen consumption was

measured at ten-minute intervals (Consolazio, Johnson and Pecora, 1963). Skin temperatures were monitored throughout the recovery periods. The weight of the subjects was recorded before and immediately following the recovery session.

Data Analysis

All data were entered and analyzed on a Leading Edge computer using Quattro 5.0 and SPSS 6.0 for windows statistics package. Descriptive analyses included : age, height, weight, skin fold measurements and predicted VO_{2max}.

The hemodynamic responses were compared at three different thermal stresses. The 27°C condition was utilized as the mid-point group in this study. A 3x13 design analysis of variance of the means (ANOVA), i.e. the means of 3 temperatures x 13 time measurements for each dependent variable (systolic blood pressure, diastolic blood pressure, heart rate and skin temperature) with single group repeated measures, were computed (Stevens, 1990). A 3x6 analysis of variance with repeated measures was used to analyze oxygen consumption. A "completely within" design was used to determine the main effects and interactions (Stevens, 1990). If a significant difference was observed, a post-hoc analysis (Tukey) was administered to determine the significant differences between the means.

RESULTS

The age and physical characteristics of the subjects are shown in Table 1.

Tables 2-4 and Figures 1-3 show the mean changes in blood pressures and heart rate during post-exercise recovery (0-60 minutes) at the three different temperatures (18°C, 27°C and 40°C). Results indicated that the ΔSBP at 27°C and 40°C conditions were lower than baseline values after the cessation of exercise and remained below baseline for the entire recovery period. However, at 18°C ΔSBP remained above baseline for 30 minutes. Once the cuff was transferred to the other finger ΔSBP fell below baseline for 10 minutes, then returned above baseline for the rest of the recovery period (Figure 1). Overall, there was a significant difference in the ΔSBP between recovery at 18°C to both 27 and 40°C. Post-hoc analysis showed time-dependent differences at the 10, 15, 25, 45, 50, 55, and 60 minute intervals of recovery between ΔSBP at 18°C and 40°C (Table 2). The post-hoc comparison between the 18°C and 27°C showed significant differences in ΔSBP change only at the 25, 50 and 55 minute intervals (Table 2). No significant differences in ΔSBP were observed at any time interval between 27°C and 40°C.

The ΔDBP response (Table 3 and Figure 2) displayed a pattern similar to that observed for ΔSBP during recovery at 18°C, 27°C and 40°C. ΔDBP was below baseline resting levels throughout the recovery period at 27°C and 40°C, whereas at 18°C, it fluctuated

between below and above baseline values.

The Δheart rate responses to 18°C, 27°C, and 40°C remained above baseline values throughout recovery, except at minutes 55 to 60 at 18°C when it fell below baseline values (Figure 3). Results indicated a significant differences among all conditions Δheart rate. Post-hoc analysis indicated that there was interaction among conditions over time. Significant differences were found among all three temperatures for Δheart rate. Post-hoc analysis indicated that at minutes 15, 20, 30, 35, 40, 45, 50 and 60 there were significant differences between 40°C and both 18°C and 27°C (Table 4). At 40°C, Δheart rate was significantly higher than at 18°C and 27°C. A significant difference was also found at the time intervals 0, 5, 10 and 25 minute between 40°C and 18°C (Table 4). At the 55 minute time interval there was a significant difference among all three conditions. ΔHeart rate at 40°C was significantly higher than at 18°C throughout recovery (Table 4). ΔHeart rate response within all three conditions show a similar linear decrease over time (Figure 3). Although decreasing linearly, the Δheart rate responses across all three conditions were distinct with 18°C having the lowest values and 40°C the highest values throughout recovery.

Table 5 and Figure 4 shows the VO_2 response during the recovery period at 18°C, 27°C and 40°C. All show a linear decrease over time within each condition. However, there were no significant differences among conditions over time (Table 5).

Tables 6-9 and Figure 5-8 shows the overall skin temperature

(Tsk) response during the recovery period. At 40°C, skin temperatures of the head (T_{he}), triceps (T_{tr}), thigh (T_{th}) and chest (T_{ch}) were much higher throughout recovery than those responses recorded at 18°C. Significant differences were found among the values at 18°C, 27°C and 40°C T_{he} (Table 6), T_{tr} (Table 7), T_{th} (Table 8) and T_{ch} (Table 9) temperatures throughout the recovery period, except immediately upon entering the chamber. At 40°C, T_{he} was significantly greater than at 27°C and 18°C. Moreover, at 27°C, T_{he} was significantly greater than at 18°C. T_{tr}, T_{th} and T_{ch} at 40°C were significantly greater than at 18°C.

Table 10 depicts pre-exercise and post-exercise values for SBP, DBP and HR at three recovery temperatures.

There were no significant differences among conditions for pre-exercise to post-exercise weight loss.

DISCUSSION

The main purpose of this study was to examine the influence of three different recovery temperatures on post-exercise blood pressure with subjects recovering in the supine position.

Although all subjects were exercised at room temperature, this study was unique because the subject's recovery period took place in a chamber at a predetermined temperature of either 18, 27 or 40°C. This allowed the effect of various temperatures on blood pressure recovery to be observed which is extremely important because the blood distribution during exercise uses the same reflex pathways as challenges to thermal homeostasis (Thoden et al 1994). Therefore, by separating the two we can properly observe the effect of temperature on post-exercise recovery without interference by any of the neuromuscular or noprioceptive events of exercise.

The observed reduction in systolic blood pressure from exercise varied from an average of 5 mmHg above resting, in a environment of 18°C, to 20 mmHg below baseline during recovery at 40°C. Several studies have investigated the possible cause(s) for this reduction in blood pressure following exercise. Fitzgerald (1981) noted a significant decrease in blood pressure following mild exercise for up to 4 to 10 hours, while recovering at room temperature in a seated position. He concluded this was a result of venous pooling in the extremities and persistent vasodilation in the muscles. Despite having subjects in a supine position, we

still observed a reduction in blood pressure throughout recovery. The observations of this study, are further supported by Hannum and Kasch (1981), Bennet and Wilcox (1984) and Wilcox et al. (1982). These authors also observed similar trends (below baseline values) in systolic blood pressure during their respective studies.

Twenty minutes of exercise at a heart rate of 140 beats per minute followed by a one hour supine recovery period at various temperatures elicited significant differences in systolic blood pressure and heart rate. ▲Systolic blood pressure (SBP) was significantly higher at 18°C in comparison to 27°C and 40°C. There were no significant differences in ▲SBP between 27°C and 40°C. ▲Heart rate was significantly different between 40°C and both at 27°C and 18°C but not between 18°C and 27°C. Results also indicated that there were significant differences among all three conditions for skin temperatures whereas no significant differences among recovery temperatures was observed for VO_2 and ▲DBP.

In this study, during recovery at 18°C, it was observed that ▲SBP was above baseline from 0-29 minutes, whereas ▲SBP decreased below baseline between minutes 30-45 before it increased again above baseline from 45-60 minutes. This significant change in SBP, when transferring the Finapres cuff from the left to the right finger Finapres, may have been due to the (1) Finapres exhibiting a temperature sensitivity (2) mechanical difficulties (3) differences in thermal responses in acral areas (4) exercise or pressure stimulus related responses in acral areas or fingers. In acral areas of the skin, during cold exposure there is a cold-

induced vasodilation that occurs in the fingers (Pergola et al, 1993). It is an initial vasoconstriction followed by alternating periods of vasodilation known as the "hunting reaction" . This is not observed in non-acral areas. Unfortunately, we did not measure finger temperature during this study. Although there is active vasodilation in non-acral tissues this is not the case for acral tissues (or fingers). Thoden et al, (1994) observed a continual decrease in all T_{sk} towards pre-exercise values except finger temperature, following exercise, throughout a 65 min recovery period. They hypothesised that this discrepancy may be a the result of some exercise related responses which may have had thermal effects. They postulated that factors other than vasodilators, such as a loss in vasoconstriction tone, were involved in adjusting acral skin blood flow (SBF), however they could not determine a specific cause. Therefore, we can speculate that the variability noted in our results of ASBP may be due to exercise related responses that disrupt the usual distribution of blood flow particularly in acral tissue.

The large standard deviations observed in ASBP may be the result of subject differences. These include: (1) Acclimatization. Subjects may have been warmer/cooler than usual due to the weather, which is related to the time of year the experiment took place. This could explain the variability of SBP and the high standard deviations of ΔSBP observed at each experimental temperature. (2) Exercise prior to session. Subjects may have cycled to the lab or performed some form of physical activity prior to the session

despite assurances to investigator. (3) Previous experimental exposure. None of the subjects indicated that they had been previously exposed for extended periods to such temperatures extremes or experimental conditions. (4) Fitness levels. Subjects fitness levels were only "predicted" using the CAFT. This may have contributed to the variability in SBP pre and post-exercise. (5) The variation in height, weight and % of body fat may also have contributed to mild variations in results.

A study which was performed by Thoden et al, (1994) may assist to explain some of the results observed in our study. They observed that during exercise oesophageal temperature (T_{es}) increased following 2-3 minutes of exercise and attributed this to blood flow to the active muscles and warming venous return, which was followed by a simultaneous increase in skin vasodilation , as indicated by T_{sk}. They determined that this exercise response may be carried over for an extended period of time following exercise. After exercise, they observed that T_{es} decreased rapidly within the first 5 min but remained above resting values throughout recovery despite the continuous fall of rectal temperature (T_{re}) and all skin temperatures (T_{sk}), except thigh temperature T_{th}, towards baseline values. They determined that this may be due to a transitional redistribution of blood flow. They also concluded that the elevated T_{th} suggests that there remains a good deal of metabolic heat in the legs after exercise. Therefore, during low ambient temperature the heat induced by exercise may actually weaken vasoconstriction by direct heat transfer from underlying muscle or SBF heat delivery to

the surface. This may explain the increase in T_{th} during the first five minutes of recovery in our results and its slow decrease during recovery. This may also explain some of the variability in ASBP throughout the recovery period within our study. Pergola et al. (1994), determined the reflex effect of increasing T_{sk} at rest on SBF during rest and concluded that it was due to a decrease in vasoconstriction activity and does not activate the vasodilation system. However, elevated internal temperature during exercise and increases in T_{sk} are the result of active vasodilation. Therefore, in our study we can postulate that internal temperature is elevated above pre exercise values throughout recovery (Thoden et al., 1993). Thus, during recovery at 40°C the reductions in blood pressure are mainly a result of increased active vasodilation, whereas at 18°C the lower T_{sk} may have altered the internal temperature threshold for cutaneous vasodilation to higher levels, thus allowing blood pressure to remain above resting values during recovery through a mild withdrawal of active vasodilation activity.

During our experimental conditions, peripheral vasodilation was indirectly examined by measuring changes in skin temperature. It was observed that there was a significant difference between skin temperatures and the different recovery conditions. Head (T_{he}), tricep (T_{tr}), thigh (T_{th}) and chest (T_{ch}) temperatures were significantly greater in recovery at 40°C when compared to temperatures at 18°C. Therefore, the decrease in blood pressure below baseline levels at 27°C and 40 °C may be attributed to a decrease in peripheral vascular resistance and persistent muscle

vasodilation. Pierpoli et al, (1994) observed a decrease in vascular resistance in the non-exercising limbs following maximal exercise. They also noted a significant reduction in total peripheral resistance (TPR) after exercise which persisted for one hour. This may have been the result of persistent vasodilation in both exercising and non-exercising limbs which may have contributed to the decrease in blood pressure observed in our study. Piepoli et al (1993) also observed that the peripheral vasodilation following exercise seen in the vascular beds of the non-exercising limbs is dependent on exercise intensity. They noted a reduction in the baroreflex sensitivity which suggests persistent sympathetic responses rather than vagal activation following exercise. They concluded that during the first 10 min of recovery the baroreflex plays little or no part in the fall in blood pressure. The vasodilator stimuli seems to overcome concurrent vasoconstrictor factors (Piepoli et al, 1993). Fleg and Lakatta (1974) also concluded that the decrease in blood pressure following exercise may be attributed to an excessive fall in vascular resistance, thereby accentuating venous pooling. Kellogg et al (1993) is also in agreement and suggested that the increase in skin blood flow during prolonged exercise at high T_{sk} is also a result of slow withdrawal of vasoconstriction activity.

During cold exposure, the body tries to conserve heat by redirecting blood flow to the central circulation which results in vasoconstriction of the peripheral blood vessels and subsequently, causes an increase in cardiac output and SBP (Raven, 1975). During

heat stress, two major adjustments occur: (1) active cutaneous vasodilation in non-acral skin and (2) sweating (Mack et al, 1994). Active cutaneous vasodilation occurs at a body core temperature threshold and results in a rapid increase in skin blood flow in proportion to the rise in T_{es} (Mack et al, 1994 and Johnson and Park, 1981). During exercise there is a delay in the onset of active cutaneous vasodilation (Kellogg et al, 1991) i.e. there is a shift in the T_{es} threshold for cutaneous vasodilation. However, the reductions in blood pressure below baseline throughout recovery at 27°C and 40°C observed in our study was believed to be due to the body's need to transfer excess heat into the environment away from the body (Sherwood, 1987) which was accomplished through peripheral vasodilation and the redirection of blood flow from the deep veins to the periphery (Rowell, 1986). This resulted in an elevated heart rate, cardiac output, skin temperature above baseline values (Rowell, 1986) and a decrease in systolic blood pressure following exercise (Rowell, 1983 and Kim, 1979). Thus, this support the greater decrease in post-exercise ▲SBP at 40°C than at 27°C, below baseline values, that was observed during recovery.

Other studies have observed significant reductions in diastolic blood pressure during a recovery period following dynamic exercise (Bennet et al., (1984), Wilcox et al., (1982) and Fitzgerald (1981). It is known that alterations in diastolic blood pressure are dependent upon peripheral resistance, which in turn is dependent on vessel radius and arterial vasculature (Pallatini, (1988). It is believed that during aerobic activity vascular

resistance is decreased due to local vasodilation in active tissue (Hannum and Kasch, 1981). Following exercise, vascular resistance is believed to be maintained at this lowered state thus resulting in the decreases in diastolic blood pressure recorded following exercise (Wilcox et al, 1982). Thus, the decreases in diastolic blood pressure observed during recovery at 27°c and 40°C in this study is believed to be a result of an increase in peripheral vasodilation. The fluctuation of DBP at 18°C could be a result of the body trying to preserve internal heat within the body due to the already lowered state of vascular resistance and the cold which results in vasoconstriction or in trying to maintain mean arterial blood pressure.

We still did not observe a significant decrease in diastolic blood pressure below baseline among all conditions. Thus, it may be concluded that an increase in peripheral vasodilation may cause a significant decrease in systolic blood pressure but not in diastolic blood pressure.

The present study noted that the diastolic blood pressure at 18°C remained above baseline for the first 30 and the last 10 minutes of recovery. As was the case for systolic blood pressure, the elevation of pressure above baseline is believed to be a result of a withdrawal of vasoconstriction due to the temperature change experienced by the body following a 20 minute exercise period.

Kaufman et al., (1987) had subjects, who were 19-29 years of age, perform 5, 10 minute treadmill exercises with a 3 minute rest between each interval. The recovery period for these 2 studies was

60 minutes in a seated position. These authors also observed a significant decrease in diastolic blood pressure, however this effect was transient as the blood pressure showed no significant reduction by the end of the recovery period. These researchers speculated that the initial decrease was a result of a decreased stroke volume generated by an increase in peripheral body temperature, thus resulting in peripheral vasodilation. They also theorized that the decrease in diastolic blood pressure may be due to a time dependent clearance of vasodilation metabolites from skeletal muscle.

Further study into the effects of decreased room temperature on the subsequent elevation of core temperature within the body following exercise is warranted.

To observe the effect of temperature on heart rate during recovery, our study used 27°C as our mid-point and 18°C and 40°C representing below\above this mid-point, respectively. Throughout recovery at the different temperatures it was observed that a significant difference in heart rate responses occurred. For instance, heart rate was significantly higher at 40°C than at 18°C throughout recovery. This is in agreement with Rowell et al., (1969), who noted that an increase in Tsk temperature from 36.5 to 40.9 resulted in a very large increase in heart rate. Moreover, Raven et al., (1975) also noted a slight decrease in heart rate at ambient temperatures 20°C>ambient temp>10°C.

Kilgour et al., (1992) observed the heart rate response in a heat stress condition to be greater than that observed during a

control condition immediately following exercise and then throughout the recovery period. Floras et al. (1991), studying hypertensives, also found a significant difference in heart rate following exercise and concluded that it may be a result of arterial baroreceptor afferent discharge adaptation to the prolonged increase in systolic blood pressure evoked by exercise rather than by the exercise itself. However, this was not seen in normotensive subjects and was subsequently rejected. Floras et al., (1991) concluded that the normotensives ability to vasoconstrict in response to orthostatic stress was impaired after brief exhaustive exercise. It is not known if any studies have examined the added effect of cold or heat temperature during recovery on post-exercise blood pressure.

The present study shows that following a 20 min exercise on the treadmill in normotensive men, blood pressure fell below pre-exercised levels in all situations except in cold temperature recovery. It was hypothesized that the SBP would be maintained above resting levels during recovery in 18°C and below at 40°C. This was the case at 40°C however at 18°C it was above for the first 30 minutes and then below thereafter for min 35-45 then above for the remaining period of recovery.

Therefore, during dynamic exercise there is a decrease in vascular resistance and an increase in core temperature which results in cutaneous and skeletal muscle vasodilation (Wilcox et al, 1982). It is believed that the reduction in blood pressure following exercise may be a result of an increase in peripheral

vasodilation which decreases venous return, due to the lack of the muscle pump, while skeletal and cutaneous vasodilation persist. The baroreceptors induce vasoconstriction, however it does not overwhelm the vasodilator response to heat stress (Kellogg et al, 1990). In the cold the body attempts to conserve heat through vasoconstriction of the peripheral blood vessels and, thus causes an increase in central blood volume which results in an increase cardiac output and systolic blood pressure.

In conclusion, there is a significant effect of temperature on SBP, HR and T_{sk} temperatures during recovery. The reduction in post-exercise SBP below baseline values during recovery at (27°C) and (40°C) temperatures appears to be associated with the increase in peripheral vasodilation and persistent vasodilation in the muscles. However, at (18°C) SBP was maintained above baseline values during recovery which appears to be attributed to a withdrawal of active vasodilation.

REFERENCES

Bennett, T., Wilcox, G., and MacDonald, I.A. (1984). Post-exercise reduction of blood pressure in hypertensive men is not due to acute impairment of baroreflex function. Clin Sci;67:97-103.

Boyer, J.L. and Kasch, F.W. (1970). Exercise therapy in hypertensive men. JAMA;211:1668-1671.

Canadian Standardized Test of Fitness. (3rd ed.). Ottawa, Fitness and Amateur Sport, 1986.

Choquette, G. and Ferguson, R.J. (1973). Blood pressure reduction in borderline hypertensives following physical training. C.M.A.J;108:699-703.

Consolazio, Frank C., Johnson, Robert E., and Pecora, Louis J.. Physiological measurements of metabolic functions in man. McGraw-Hill Book Company Inc. U.S.A., 1963.

Fitzgerald, W. (1981). Labile hypertension and jogging: new diagnostic tool or spurious discovery ?. Br Med J;282:542-544.

Fleg, J.L., and Lakatta, E.G. (1986). Prevalence and significance of post-exercise hypotension in apparently healthy subjects. Am J Cardiol;57:1380-1384.

Floras, J.N. and Senn B.L. (1991). Absence of post exercise hypotension and sympathoinhibition in normal subjects: Additional evidence for increased sympathetic outflow in borderline hypertension. Can J Cardiol;7(6):253-258.

Friedman, D.B., Ordway, G.A., and Williams, S. (1987). Exercise induced functional desensitization of canine cardiac B-adrenergic receptors. Am Physiol Society;62(4):1721-1723.

Hannum, S.M., and Kasch, F.W. (1981). Acute postexercise blood pressure response of hypertensive and normotensive men. Scand J Sports Sci.;3(1):11-15.#3731

Hartmann, B. and Bassenge, E. (1989). Nitchinvasive, Kontonuierliche Messung des Fingerarteriendrucks mit dem Servo-Plethysmo-Manometer Finapres. Herz;14(4):251-259.

Jette, M., Campbell, J., Mongeon, J., and Routhier, R. (1976). The Canadian home fitness test as a predictor of aerobic capacity. Can Med Ass J;14:680-682.

Jette, M., Landry, F., and Sidney, K. (1991a). Blood pressure response to the Canadian Aerobic Fitness Test. Can J Public Health.

Jette, M., Quenneville, J., and Sidney, K. (1991b). Fitness testing and counselling in health promotion. Can J Sport Sci. 17: 194-198.

Johnson, J.M. and Park, M.K. (1981). Effect of upright exercise on threshold for cutaneous vasodilation and sweating. J Appl Physiol: respirat Environ Exercise Physiol; 50(4): 814-818.

Kaufman, F.L., Hughson, R.I., and Schaman, J.P. (1987). Effect of exercise on recovery blood pressure in normotensive and hypertensive subjects. Med Sci Sports Exerc;19(1):17-20.

Kellogg, D.L., JR, Johnson, J.M., and Kosiba, W.A. (1991). Control of internal temperature threshold for active cutaneous vasodilation by dynamic exercise. J Appl Physiol;71(6):2476-2482.

Kellogg, D.L., JR, Johnson, J.M., Kenney, W.L., Pergola, P.E., and Kosiba, W.A. (1993). Mechanisms of control of skin blood flow during prolonged exercise in humans. Am J Physiol 265 (Heart Circ Physiol 34): H562-H568.

Kilgour, R.D., Gariepy, P., and Rehel, R. (1993). Cardiovascular responses during recovery from exercise and thermal stress. Aviat Space Environ Med;64:224-229.

Kim, Y.D., Lake, C.R., Lees, D.E., Schutte, W.H., Bull, J.M., Weise, V., and Kopin, I.J. (1979). Hemodynamic and plasma catecholamine responses to hyperthermic cancer therapy in humans. AM J Physiol;30:375-384.

Korner, P.I.. (1952). The normal human blood pressure during and after exercise, with some related observations on changes in the heart rate and the blood flow in the limbs. Aust J Exp Biol Med Sci;30:375-384.

Kral, J., Chrastek, J., and Adamirova, J.. The hypotensive effect of physical activity in hypertensives. In Prevention of ischemic heart disease. Springfield, IL, Charles Thomas Publishers. 1966, p359-371.

Mack, G.W., Nose, H., Takamata, A., Okuno, T., and Morimoto, T. (1994). Influence of exercise intensity and plasma volume on active cutaneous vasodilation in humans. Med Sci Sports Exer;26(2):209-216.

Nadel, E.R., Cafarelli, E., Roberts, M.F., and Wenger, B. (1979). Circulatory regulation during exercise in different ambient temperatures. J Appl Physiol: Respirat Environ Physiol;46(3):430-437.

Palatini, P. (1988). Blood pressure behavior during physical activity. Sports Med;5:353-374.

Paulev, P., Jordal, R., Kristensen, O. and Ladefoged, J.(1984). Therapeutic effect of exercise on hypertension. Eur J Appl Physiol;53:180-185.

Pergola, P.E., Kellogg, D.L., JR., Johnson, J.M., Kosiba, W.A., and Solomon, D.E. (1993). Role of sympathetic nerves in the vascular effects of local temperature in human forearm skin. Am J Physiol 265 (Heart Cir Physiol 34):H785-H792.

Pergola, P.E., Kellogg, D.L., JR., Johnson, J.M., and Kosiba, W.A. (1994). Reflex control of active cutaneous vasoidlation by skin temperature in humans. Am J Physiol 266 (Heart Cir Physiol 35):H1979-H1984.

Piepoli, M., Coats, A.J.S., Adamopoulos, S., Bernardi, L., Feng, Y.H., Conway, J., and Sleight, P. (1993). Persistent peripheral vasodilation and sympathetic activity in hypotension after maximal exercise. J Appl Physiol;75(4): 1807-1814.

Piepoli, M., Isea, J.E., Pannarale, G., Adamopoulos, S., Sleight, P., and Coats, A.J.S.(1994). Load dependence of changes in forearm and peripheral vascular resistance after acute leg exercise in man. J Physiol;478(2):357-362.

Raven, P.B, Wilkerson, J.E., Horvath, S.M. and Bolduan, N.W. (1975). Thermal, metabolic, and cardiovascular responses to various degrees of cold stress. Can J Physiol Pharmacol;53:293-298.

Rowell, L.B., Brengelmann, G.L. and Murray, J.A. (1969). Cardiovascular responses to sustained high skin temperature in resting man. J Appl Physiol;27(5):673-680.

Rowell, L.B. (1983). Cardiovascular adjustments to thermal stress. In Handbook of physiology: the cardiovascular system, peripheral circulation and organ blood flow. section2(3), chapt. 27. American physiological society; Bethesda, MD, pp 967-1023.

Rowell, L.B.. Human circulation regulation during physical stress. Oxford University Press Inc.: New York, New York, 1986.

Sherwood, L. Human Physiology. West Publishing Company: U.S.A., 1989.

Stevens, James P., Intermediate statistics a modern approach. Lawrence Erlbaum associates Publishers. Hiildale, New Jersey, 1990.

Thoden, J. Kenny, G., Reardon, F., Jette, M., and Linvingstone, S. (1994) Disturbance of thermal homeostasis during post-exercise hyperthermia. J Appl Physiol;68: 170-176.

Wilcox, R.G., Bennett, T., Brown, A.M., and MacDonald, I.A. (1982). Is exercise good for high blood pressure. Br Med J;285:767-769.

Young, A.J. (1990). Energy substrate utilization during exercise in extreme environments. Ex Sports Sci Rev;18:65-117.

Table 1 : Age and physical Characteristics of the Subjects (n=10)

Variable	Units	Mean	Range	Standard Deviation
Age	years	25	28.0-23.0	+/- 5
Height	cm	178.1	182.6-161.9	+/- 7.3
Weight	kg	70.7	87.1-67.3	+/- 5.5
% Body Fat	%	10.77	12.6-9.2	+/- 4.2
Predicted VO_2max	ml/kg/min	54.0	58.4-45.2	+/- 3.2

TABLE 2 : Asystolic blood pressure (compared to resting values) during the 60 minute recovery at 18°C, 27°C and 40°C.

Time		18°C (1)	27°C (2)	40°C (3)
0 min	X(SD)	.5(15.9)	-8.7(8.9)	-5.8(8)
5 min	X(SD)	2.3(17.7)	-5.2(9.4)	-5.8(5.5)
10 min	X(SD)	6.8(-20)$^{(3)}$	-7.0(7.3)	-8.9(5.0)$^{(1)}$
15 min	X(SD)	7.1(24.6)$^{(3)}$	-9.1(8.4)	-11.0(7.7)$^{(1)}$
20 min	X(SD)	7.1(29.3)	-9.3(12.3)	-10.5(7.7)
25 min	X(SD)	9.4(4.6)$^{(2,3)}$	-10.7(10.9)$^{(1)}$	-12.8(8.1)$^{(1)}$
30 min	X(SD)	10.1(30.4)	-9.7(12.6)	-11.1(8.8)
35 min	X(SD)	-7.6(15.3)	-18.4(11.8)	-19.5(8.4)
40 min	X(SD)	-5.3(24.6)	-16.1(9.7)	-20.7(8.6)
45 min	X(SD)	-2.4(16.5)$^{(3)}$	-14.3(11.9)	-19.2(6.4)$^{(1)}$
50 min	X(SD)	5.7(21.7)$^{(2,3)}$	-12.5(12.8)$^{(1)}$	-19.0(9.9)$^{(1)}$
55 min	X(SD)	8.0(21.5)$^{(2,3)}$	-10.7(13.4)$^{(1)}$	-18.9(8.6)$^{(1)}$
60 min	X(SD)	6.2(17.8)$^{(3)}$	-9.2(14.4)	-12.6(7.8)$^{(1)}$

Note: Conditions with which a significant difference exist ($p<0.05$) are superscipted above the appropriate mean.

Table 3: Adiastolic blood pressure (compared to resting values) during the 60-minute recovery at 18°C, 27°C and 40°C

Time		18°C	27°C	40°C
0 min	X(SD)	-0.8 (12.5)	-6.5 (11.8)	-3.9 (12.5)
5 min	X(SD)	4.2 (11.8)	-1.8 (13.8)	-2.8 (12.1)
10 min	X(SD)	4.2 (11.2)	-1.9 (13.3)	-3.8 (11.7)
15 min	X(SD)	2.0 (10.2)	-3.5 (11.6)	-6.5 (12.7)
20 min	X(SD)	0.1 (21.5)	-5.0 (14.8)	-6.6 (12.7)
25 min	X(SD)	0.1 (21.5)	-6.1 (14.9)	-8.3 (12.7)
30 min	X(SD)	1.0 (16.9)	-4.7 (15.5)	-6.5 (14.5)
35 min	X(SD)	-10.5 (22.4)	-15.1 (17.9)	-15.8 (13.4)
40 min	X(SD)	-3.5 (25.2)	-9.8 (17.5)	-15.4 (12.1)
45 min	X(SD)	-4.3 (24.4)	-11.9 (14.1)	-13.5 (10.4)
50 min	X(SD)	0.4 (21.5)	-9.5 (14.2)	-14.1 (10.7)
55 min	X(SD)	1.2 (20.2)	-8.4 (15.6)	-14.6 (13.9)
60 min	X(SD)	5.0 (22.2)	-6.3 (15.1)	-11.0 (12.7)

Note: No significant differences found at the $p<0.05$ level of significance among conditions.

Table 4: Δheart rate (compared to resting values) during the 60-minute recovery at 18°C,27°C and 40°C.

Time		18°C (1)	27°C (2)	40°C (3)
0 min	X(SD)	25.3(6.6)[3]	27.2(8.8)	34.7(9.1)[1]
5 min	X(SD)	21.8(7.4)[3]	23.8(6.7)	29.8(6.8)[1]
10 min	X(SD)	16.5(8.0)[3]	19.2(7.3)	27.0(7.9)[1]
15 min	X(SD)	13.4(11.7)[3]	16.7(5.9)[3]	27.4(8.6)[1,2]
20 min	X(SD)	10.3(9.0)[3]	15.0(6.8)[3]	24.8(9.5)[1,2]
25 min	X(SD)	9.6(10.8)[3]	15.6(5.5)	24.2 (7.2)[1]
30 min	X(SD)	4.5(11.1)[3]	9.6(5.7)[3]	20.1(9.7)[1,2]
35 min	X(SD)	2.2(9.3)[3]	10.3(8.5)[3]	20.3(9.2)[1,2]
40 min	X(SD)	0.9(9.3)[3]	8.7(6.1)[3]	20.6(9.3)[1,2]
45 min	X(SD)	0.9(8.1)[3]	7.3(7.2)[3]	19.4(6.1)[1,2]
50 min	X(SD)	0.3(7.2)[3]	5.8(7.3)[3]	19.5(6.9)[1,2]
55 min	X(SD)	-5.0(11.5)[2,3]	7.4(4.6)[1,3]	18.4(7.6)[1,2]
60 min	X(SD)	-1.8(8.0)[3]	6.6 (8.7)[3]	18.4(11.5)

Note: Conditions with which a significant difference exist ($p<0.05$) are superscipted above the appropriate mean.

Table 5: Group means for O_2 during a 60-minute recovery at 18°C, 27°C and 40 °C.

Time		18°C (1)	27°C (2)	40°C (3)
1) 3 minute	X	.37	.34	.34
	SD	.11	.09	.14
1) 13 minute	X	.3	.30	.29
	SD	.08	.07	.09
1) 23 minute	X	.30	.27	.25
		.12	.05	.10
1) 33 minute	X	.28	.30	.24
	SD	.12	.10	.07
1) 43 minute	X	.32	.24	.26
	SD	.09	.05	.11
1) 53 minute	X	.31	.25	.29
	SD	.09	.07	.09

Note: No significant differences among conditions.

TABLE 6 : The mean head skin temperature during a 60 minute recovery at 18°C, 27°C and 40°C.

Time		18°C (1)	27°C (2)	40°C (3)
0 min	X	32.13	32.4	33.1
	SD	1.1	1.8	1.2
5 min	X	30.9	33.0	34.5
	SD	1.3	1.5	1.1
10 min	X	29.4	32.4	34.6
	SD	1.6	1.3	1.2
15 min	X	28.6	32.1	34.6
	SD	1.8	1.2	1.3
20 min	X	28.3	31.7	34.5
	SD	1.4	1.2	1.3
25 min	X	28.7	31.8	34.7
	SD	1.2	1.6	1.1
30 min	X	28.7	31.9	34.7
	SD	1.3	1.5	1.1
35 min	X	29.1	32.2	34.7
	SD	1.1	1.4	1.3
40 min	X	29.1	32.2	34.8
	SD	.9	1.3	1.5
45 min	X	29.3	32.4	34.9
	SD	.7	1.2	1.6
50 min	X	29.3	32.7	34.9
	SD	.7	1.2	1.4
55 min	X	29.4	32.8	34.9
	SD	.7	1.1	1.6
60 min	X	29.3	32.7	34.6
	SD	.7	1.0	1.7

Note: Significant differences found at the $p<0.05$ level of significance among all conditions except at time o

TABLE 7: The mean tricep skin temperature during a 60 minute recovery at 18°C, 27°C and 40°C.

Time		18°C (1)	27°C (2)	40°C (3)
0 min	X	32.61	33.6	34.7
	SD	2.0	1.3	.8
5 min	X	31.5	34.1	35.8
	SD	1.6	.9	1.0
10 min	X	30.7	33.9	35.9
	SD	1.6	.9	.7
15 min	X	29.9	33.8	36.2
	SD	1.8	.7	.5
20 min	X	29.4	33.7	36.4
	SD	1.9	.7	.5
25 min	X	29.2	33.7	36.4
	SD	1.8	.7	.5
30 min	X	28.9	33.6	36.5
	SD	1.8	.8	.5
35 min	X	28.4	33.5	36.5
	SD	1.6	.9	.7
40 min	X	28.2	33.5	36.7
	SD	1.8	.9	.5
45 min	X	28.1	33.5	36.9
	SD	1.7	1.1	.4
50 min	X	27.8	33.5	36.9
	SD	1.8	1.0	.4
55 min	X	27.8	33.4	37
	SD	1.9	1.1	.4
60 min	X	27.7	33.3	37
	SD	1.9	1.1	.4

Note: Significant differences found at the $p<0.05$ level of significance among all conditions except at time 0. Significant difference at time 0 between conditions 1 and 3 only.

TABLE 8: The mean thigh skin temperature during a 60 minute recovery at 18°C, 27°C and 40°C.

Time		18°C (1)	27°C (2)	40°C (3)
0 min	X	32.7	33.6	34.5
	SD	1.3	1.2	.9
5 min	X	32.3	34.5	36.3
	SD	2	.8	.4
10 min	X	31.9	33.9	36.4
	SD	1.4	1.4	.4
15 min	X	31.6	34.3	36.4
	SD	1.3	1.1	.4
20 min	X	31.2	34.0	36.3
	SD	1.3	1.0	.4
25 min	X	31	33.9	36.2
	SD	1.2	1.1	.4
30 min	X	30.9	34.1	36.1
	SD	1.2	.8	.4
35 min	X	30.7	33.9	36.1
	SD	1.1	.9	.3
40 min	X	30.5	33.8	36.1
	SD	1.1	.9	.3
45 min	X	30.4	33.7	36.1
	SD	1.1	1.1	.4
50 min	X	30.2	33.8	36.2
	SD	1.1	.9	.5
55 min	X	30.2	33.6	36.3
	SD	1.1	.9	.6
60 min	X	29.9	33.7	36.3
	SD	1.1	.9	.7

Note: Significant differences found at the $p<0.05$ level of significance among all conditions except at time 0.

TABLE 9: The mean chest skin temperature during a 60 minute recovery at 18°C, 27°C and 40°C

Time		18°C (1)	27°C (2)	40°C (3)
0 min	X	32.6	33.6	34.7
	SD	2.0	1.3	.8
5 min	X	31.5	34.1	35.7
	SD	1.6	.9	1.0
10 min	X	30.7	33.9	35.9
	SD	1.6	.9	.7
15 min	X	29.9	33.8	36.2
	SD	1.8	.7	.5
20 min	X	29.4	33.7	36.4
	SD	1.9	.7	.5
25 min	X	29.2	33.6	36.5
	SD	1.8	.7	.4
30 min	X	28.9	33.6	36.5
	SD	1.8	.8	.5
35 min	X	28.4	33.5	36.5
	SD	1.6	.9	.7
40 min	X	28.2	33.5	36.5
	SD	1.8	.9	.7
45 min	X	28.1	33.5	36.9
	SD	1.8	1.1	.4
50 min	X	27.9	33.5	37.3
	SD	1.8	1.0	.9
55 min	X	27.8	33.4	37
	SD	1.9	1.0	.4
60 min	X	27.7	33.3	37
	SD	1.9	1.1	.4

Note Significant differences found at the $p<0.05$ level of significance among all conditions except at time 0.

TABLE 10: Pre-exercise and post-exercise mean values for SBP, DBP and HR at three recovery temperatures.

MEAN PRE-EXERCISE VALUES

18°C			27°C			40°C		
SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR
125.0	65.8	59.6	124.2	62.2	60.4	119.6	64.6	55.8

MEAN POST-EXERCISE VALUES

	18°C			27°C			40°C		
TIME	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR
0	125.4	63.8	87.6	116.2	59.8	86.8	114.7	62.1	89.6
5	127.0	69.0	81.4	117.8	65.0	84.4	114.3	63.3	85.7
10	131.0	70.0	76.1	116.0	62.8	79.8	111.5	61.1	82.9
15	132.1	67.8	73.0	113.4	62.6	77.3	110.1	59.0	82.8
20	132.1	65.8	69.9	113.0	62.2	75.6	110.6	60.1	80.0
25	134.4	66.6	69.2	111.4	59.9	75.8	107.9	57.9	79.8
30	135.1	66.8	64.1	113.1	62.5	69.2	109.9	59.0	75.7
35	116.8	55.3	61.8	105.7	51.1	69.9	101.4	50.6	74.7
40	119.7	62.3	61.5	106.8	56.4	68.3	98.9	50.2	74.5
45	122.3	61.5	60.5	109.3	54.3	66.8	101.5	51.3	74.1
50	131.7	66.2	59.9	110.5	56.8	65.4	100.9	51.2	75.0
55	138.0	67.0	54.6	112.3	57.8	67.0	101.5	52.4	72.6
60	131.2	70.8	57.8	112.7	59.7	66.2	107.0	55.2	73.4

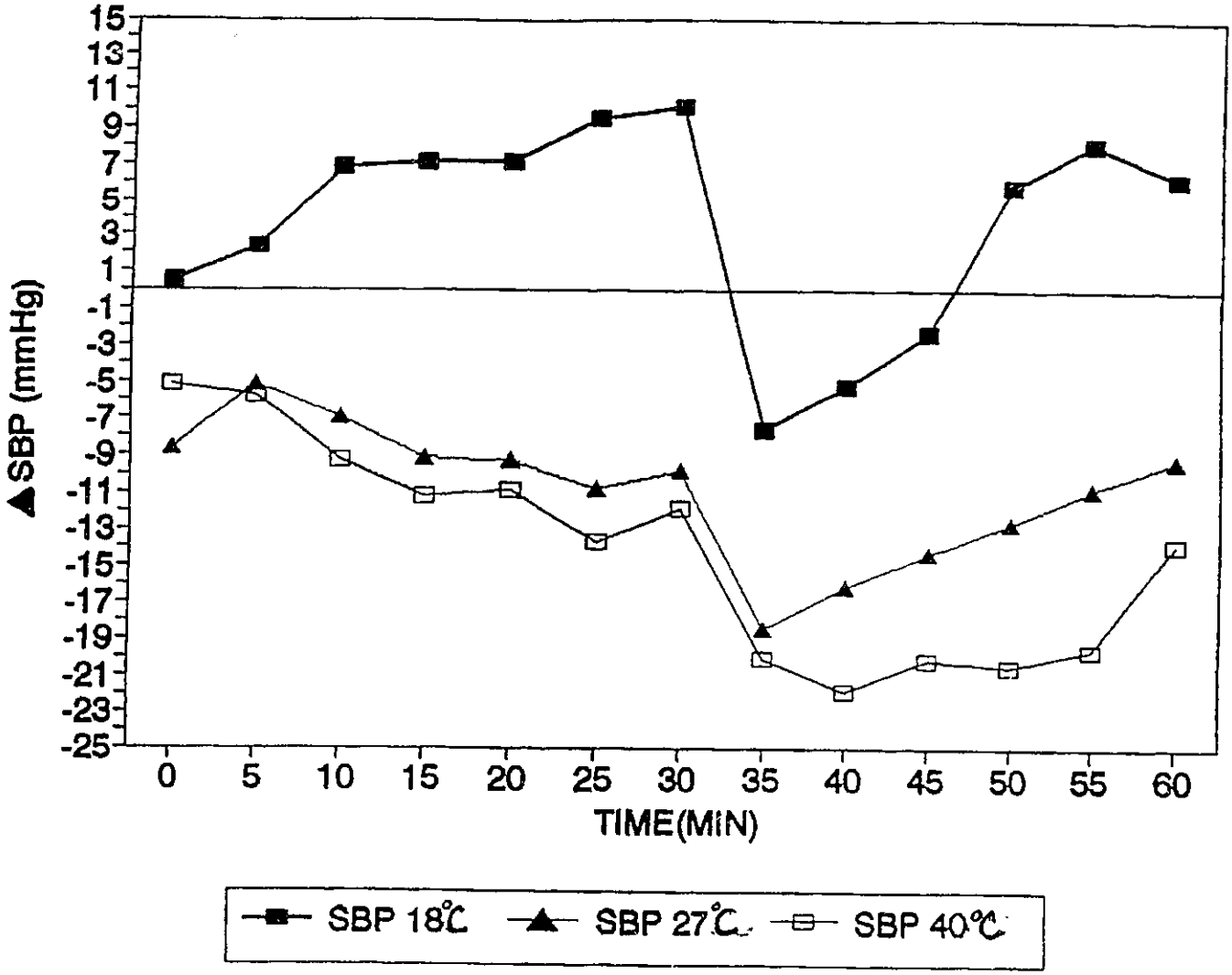

Fig 1: ▲SBP during post-exercise recovery to three experimental conditions.

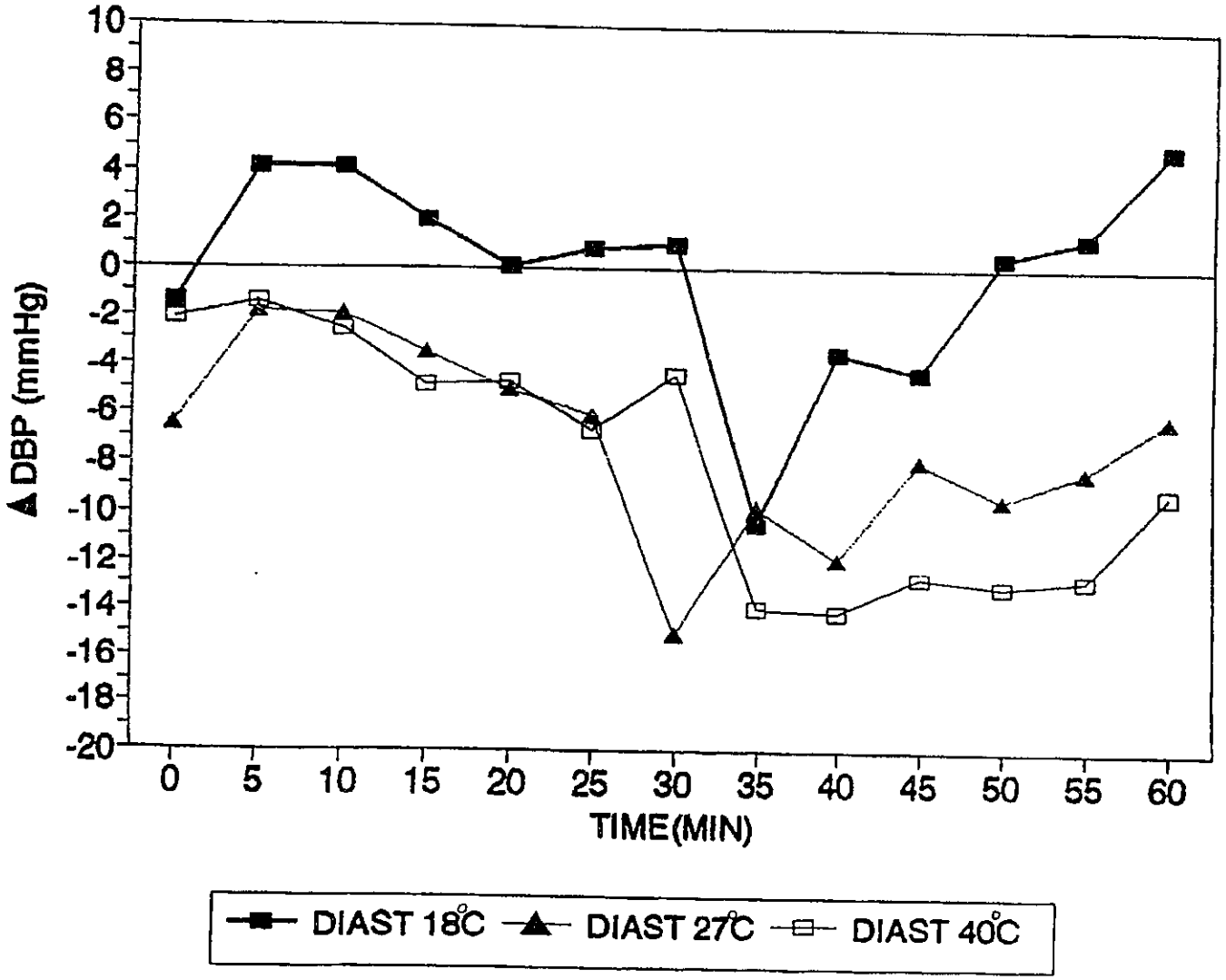

Fig 2: ▲DBP during post-exercise recovery to three experimental conditions.

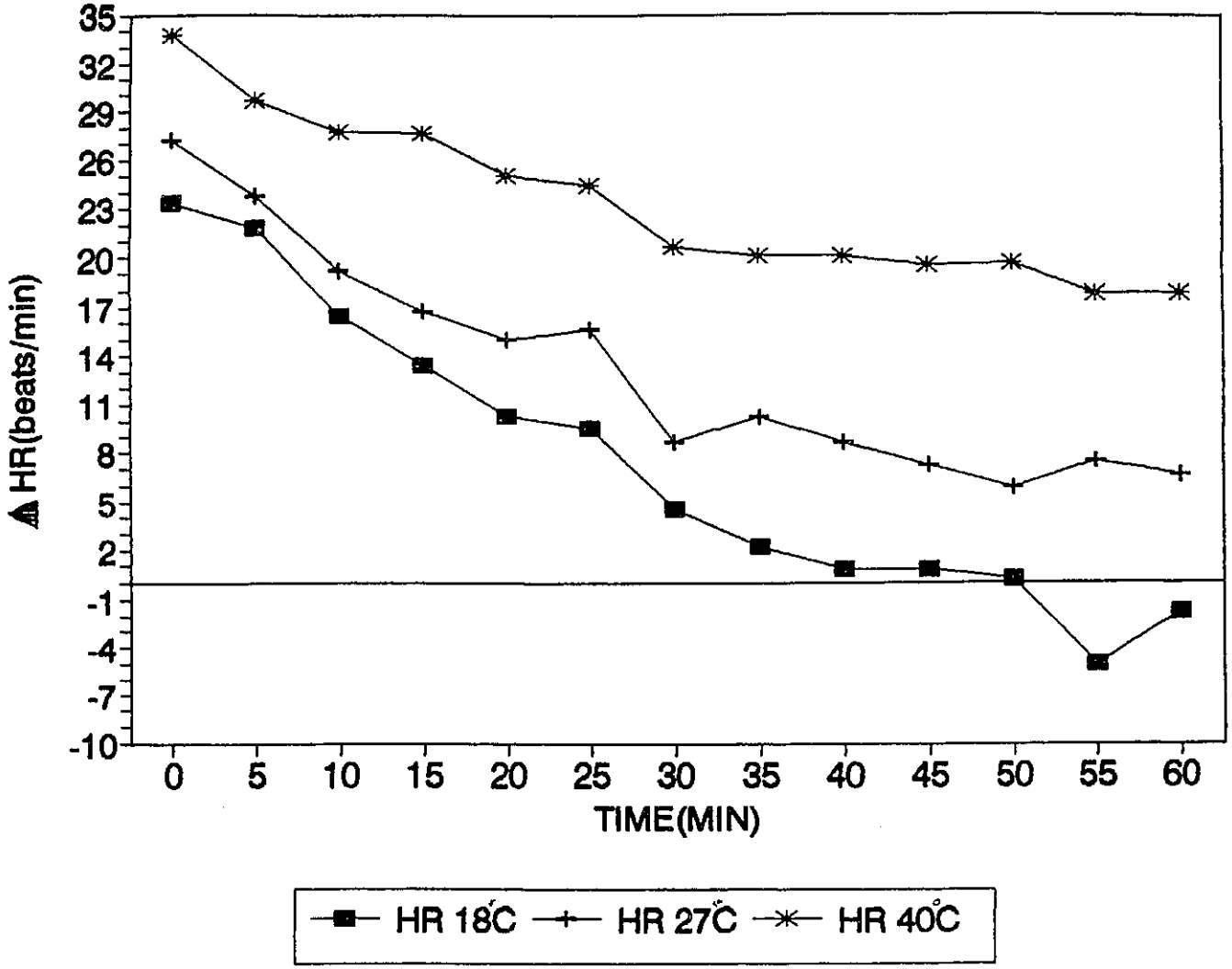

Fig 3: ΔHR during post-exercise recovery to three experimental conditions.

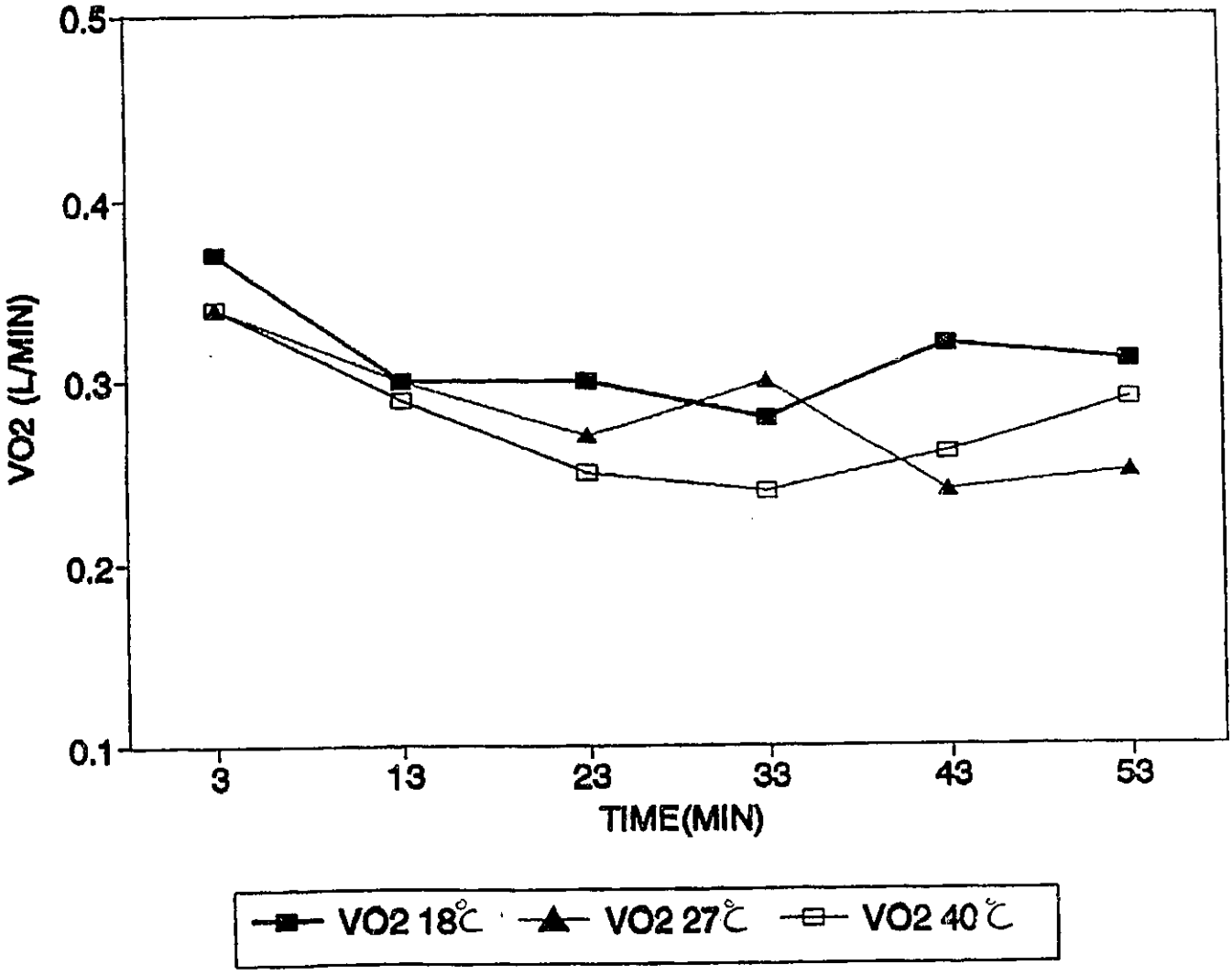

Fig 4: Mean VO_2 during post-exercise recovery to three experimental conditions.

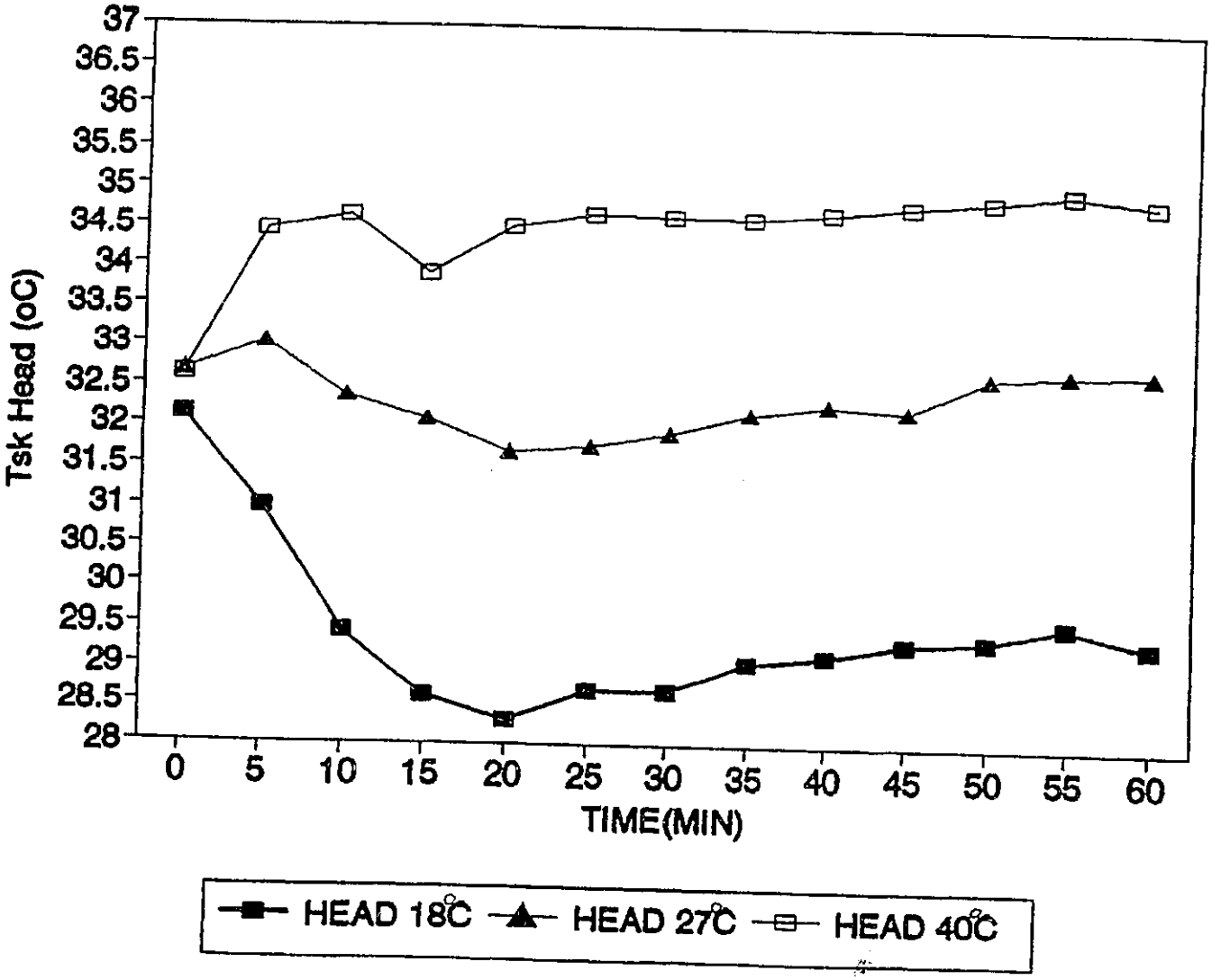

Fig 5: Mean skin temperature (Tsk) of the head during post-exercise recovery to three experimental conditions.

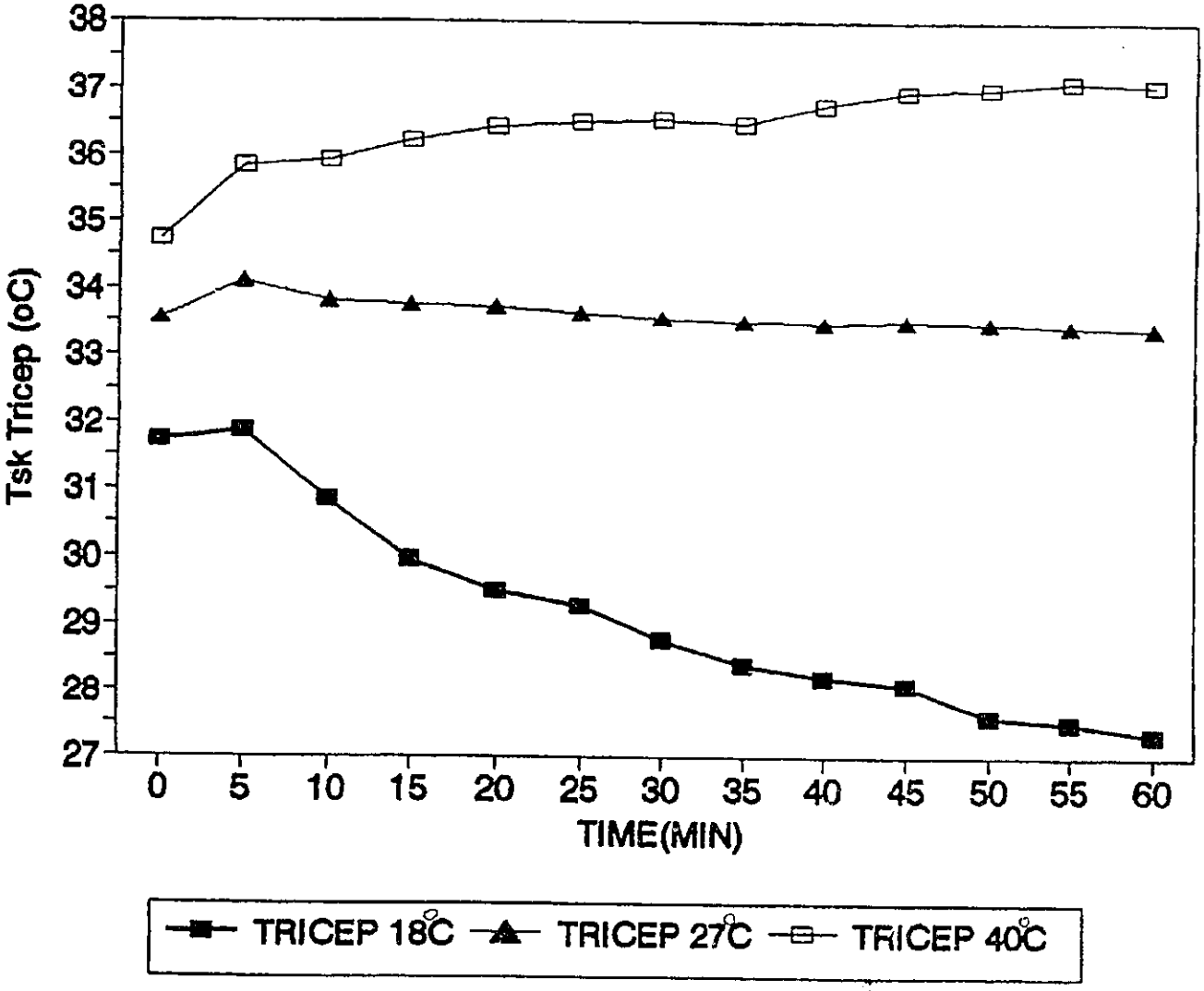

Fig 6: Mean skin temperature (Tsk) of the tricep during post-exercise recovery to three experimental conditions.

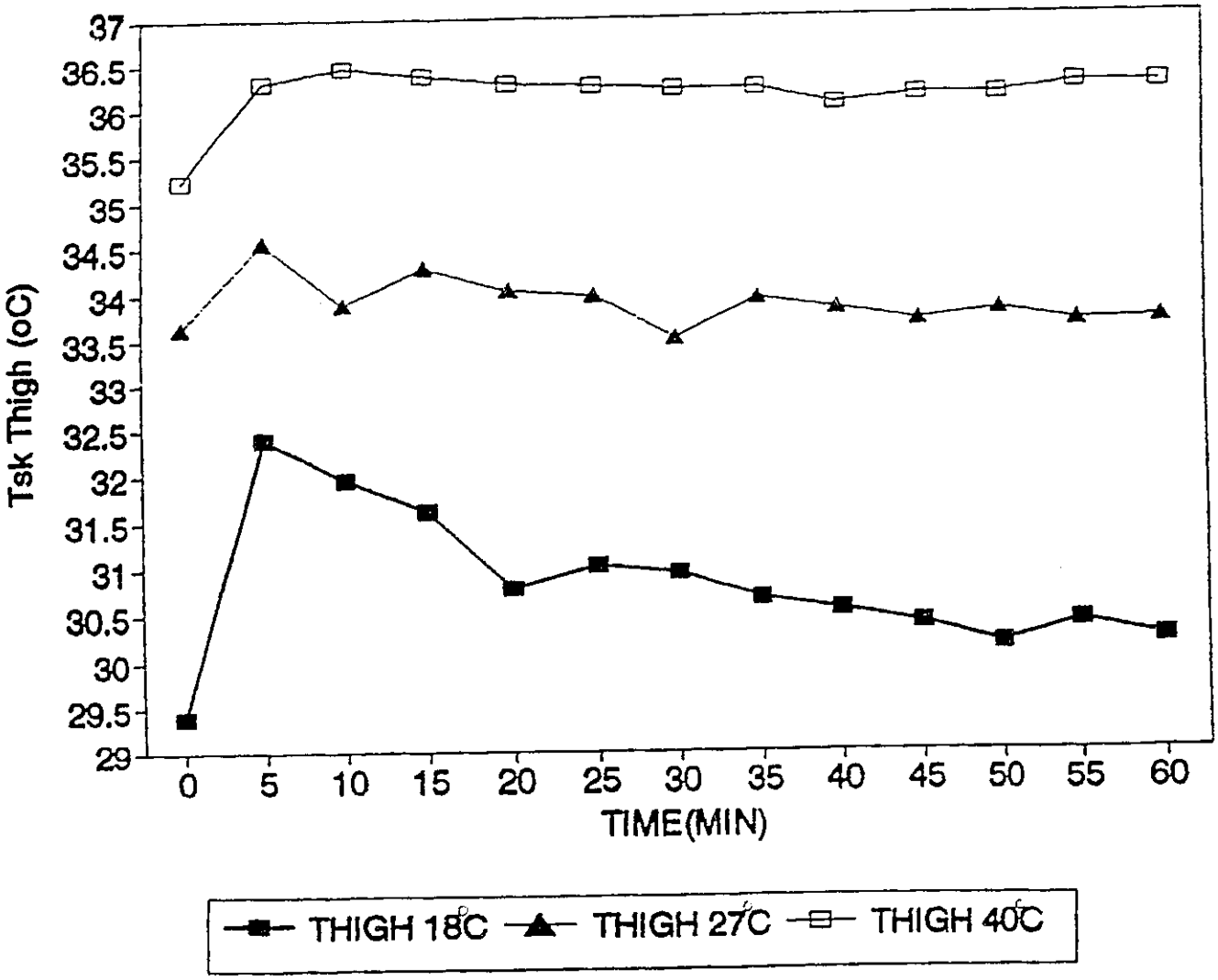

Fig 7: Mean skin temperature (Tsk) of the thigh during post-exercise recovery to three experimental conditions.

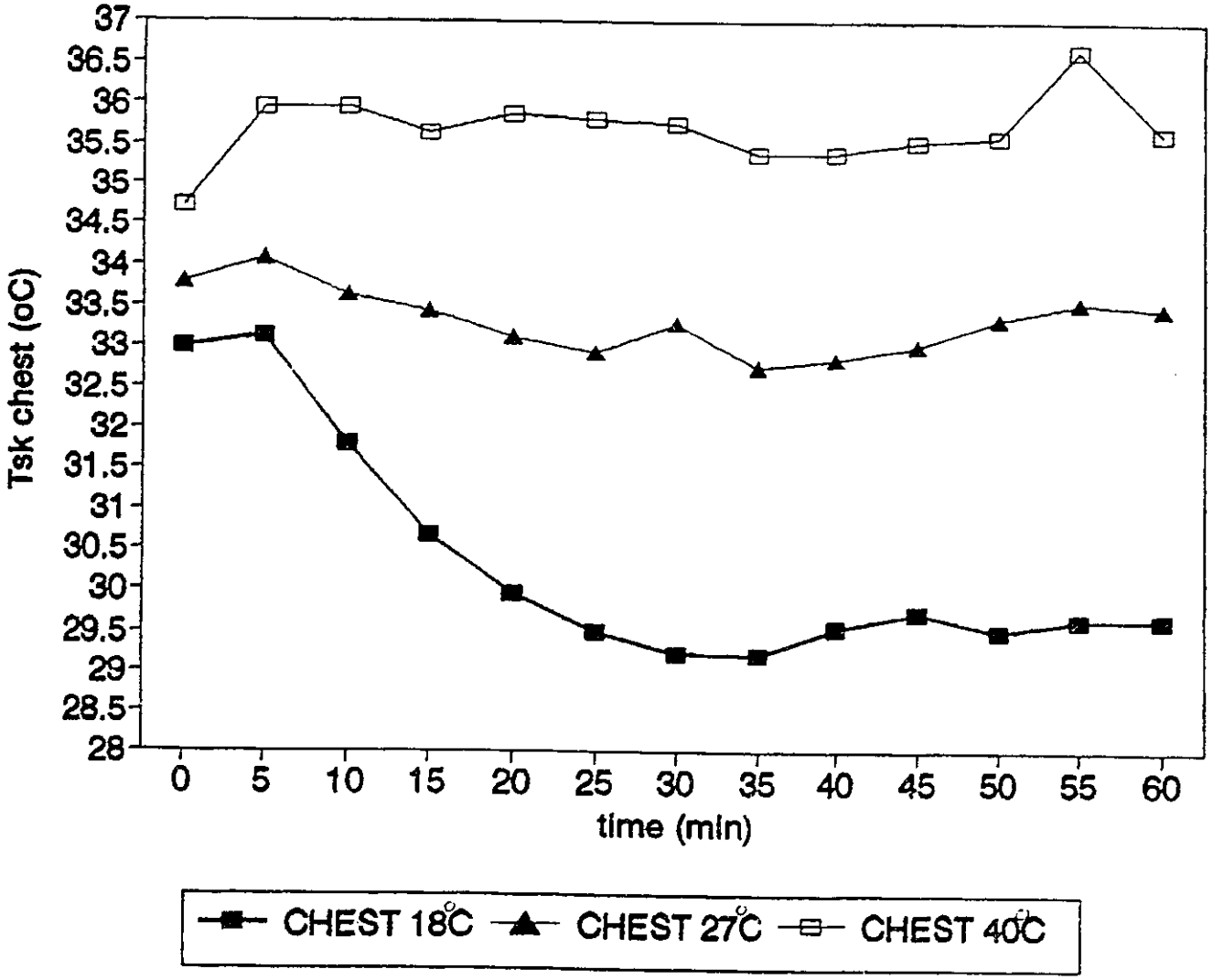

Fig 8: Mean skin temperature (Tsk) of the chest during post-exercise recovery to three experimental conditions.

REVIEW OF LITERATURE

INTRODUCTION

A series of physiological responses occur during exercise to meet the oxygen demands of the exercising muscles. The hemodynamic responses to exercise include an immediate increase in heart rate stroke volume and cardiac output, a gradual increase in blood pressure and a decrease in systemic vascular resistance. Following exercise, the exact nature of the mechanism(s) involved in the reduction in post-exercise blood pressure are not as well understood. The mechanisms which may be involved will be reviewed as well as the effect of temperature on the cardiovascular system.

HEMODYNAMIC RESPONSE DURING DYNAMIC EXERCISE

At the onset of exercise, several physiological changes occur to meet the energy demands placed on the body. An increase in cardiac output, heart rate, stroke volume, and systolic blood pressure is observed, as well as a slight decrease in total peripheral resistance and no change or a slight decrease in diastolic blood pressure (Palatini, 1988).

The primary function of the heart, regarding exercise, is to increase the blood flow to the arterial circulation (ASCM, 1988). Cardiac output is the main factor which determines the rate of O_2

delivery to the active skeletal muscle (ASCM, 1988).

Cardiac output

Cardiac output is determined by heart rate and stroke volume. At the onset of exercise, cardiac output increases rapidly and then more gradually until a plateau or steady state is reached. Cardiac output increases linearly with the increase in the oxygen (O_2) demands of the body, thus enabling the heart to increase its work load through an increase in blood flow to the exercising muscles (Rowell, 1986). Exercising at a continuous intensity will increase cardiac output until a steady state is reached in which the O_2 demands are met. Cardiac output during exercise may increase from approximately 5 l/min at rest, to 20 to 25 l/min (Davson and Eggleton, 1968).

There are two factors which limit cardiac output. They include heart rate and stroke volume.

Heart rate

Heart rate is one of the determinants of cardiac output. It is controlled by intrinsic factors of the heart, as well as extrinsic and hormonal factors (Jensen, 1980). The heart is innervated by the autonomic nervous system which includes both the sympathetic and parasympathetic nervous systems. Any change in heart rate is

determined primarily by the influence of the autonomic nervous system on the sinoatrial node (Sherwood, 1989). The sinoatrial node is influenced by the large number of receptor sites for parasympathetic and sympathetic nerves (Vander, 1980).

It is known that at rest, stimulation of the parasympathetic nerve or the vagus nerve causes heart rate to decrease while stimulation of the sympathetic nervous system causes heart rate to increase (Lamb, 1978). At the onset of exercise there is a reflex increase in heart rate. The initial increase in heart rate is due to the contraction of the muscles and joint capsules which their receptors transmit impulses to the spinal cord and then to the cardiac regulating center in the brain (Lamb, 1978), therefore, resulting in an inhibition of the vagus nerve and causing an increase in heart rate. Hence, the immediate response of heart rate to exercise can be attributed to a decrease in the parasympathetic activity or vagal withdrawal (Rowell,1986), while the later rise in heart rate has been associated with an increase in sympathetic nervous activity (Christensen, 1973).

Robinson et al, (1966) conducted a study to assess the mechanisms which regulate heart rate response to dynamic exercise. They studied the effect of parasympathetic blockage with atropine and sympathetic blockage by propranolol and observed that the immediate, rapid, increase in heart rate to exercise was absent with the administration of atropine. However, the later increase in heart rate still occurred once heart rate attained 100 beats/min and sympathetic nervous activity was stimulated. Conversely, the

blockage of the sympathetic activity with propranolol still resulted in the initial, rapid, increase in heart rate while the elevations thereafter, were much lower than previously observed (Robinson et al, 1966). From these results, it was postulated that the initial increase in heart rate was mediated through a release of vagal tone, although once heart rate attained or reached a rate of 100 b/min, there was sympathetic nervous system stimulation which was associated with a subsequent gradual increase in heart rate (Robinson et al, 1966). Escourrou et al, (1984) also postulated that the magnitude of the heart rate response was proportional to the stimulation of the sympathetic nervous system through a study using seven men during bicycle exercise. They observed an increase leakage of norepinephrine from the sympathetic nerve endings into the plasma as the severity of the exercise increased above 40% of VO_2 max. The Bannister and Griffiths (1972) study observed the same trend. They examined the levels of circulating catecholamines in response to various intensities of dynamic bicycle exercise and their results indicated that the amount of catecholamines secreted during exercise is directly proportional to the intensity of the exercise.

In brief, the initial rapid increase of heart rate at the onset of exercise is associated with a withdrawal of parasympathetic stimulation or vagal withdrawal. The later gradual increase in heart rate is due to an increase in sympathetic stimulation resulting in an increased catecholamine secretion. Thus, it is important to note the significance of the withdrawal of

vagus nerve stimulation on the heart rate, at low exercise levels, and the sympathetic stimulation in causing the subsequent gradual increases in heart rate to higher exercise levels to meet the demands of exercise (Rowell, 1986).

Stroke volume

Stroke volume is another factor affecting cardiac output. Stroke volume is primarily regulated by intrinsic factors of the heart. One of the intrinsic factors is the Frank-Starling mechanism which states that stroke volume is determined by the rate at which blood is returned to the heart through venous return (Patton et al, 1989). Therefore, an increase in venous return, causes an increase in left ventricular end diastolic volume, which results in an increase in the contraction of the myocardium via the length tension mechanism, hence resulting in an increase in stroke volume (Patton et al, 1989). The effect of the extrinsic factors such as sympathetic stimulation evoke an even greater increase in contractility of the heart. However, stroke volume unlike heart rate and cardiac output does not increase linearly with oxygen (O_2) uptake or workload. Stroke volume increases sharply at the onset of exercise, due to an increase in venous return, until 40 to 50% of VO_2 max is attained at which time it will increase only slightly thereafter (Astrand and Rodahl, 1986).

In brief, the increases in cardiac output and heart rate at

the onset of exercise are primarily due to the increased O_2 demands of the body and the initial vagal withdrawal. Further, increases in cardiac output and heart rate are linearly associated with the workload and increase in association to it until a steady state is reached and the oxygen demands are met. Stroke volume, however, increases rapidly at the onset until 40 to 50% of VO_2 max then stabilizing thereafter.

Total peripheral resistance

Total peripheral resistance is another important determinant of blood pressure. It is affected by the arterial vasculature, blood viscosity, and most importantly, vessel radius (Smith,1980,p20).

Vessel radius is determined by the balance between the vasodilators and vasoconstrictors. Vessel radius is under neural control of the sympathetic nervous system which when activated results in vasoconstriction. At the onset of exercise, sympathetic activity is not activated. However, as exercise progresses, sympathetic nervous activity is stimulated, hence causing vasoconstriction in non-working muscles (Astrand and Rodahl,1986). Therefore, it would seem that the vessels's resistance would increase during exercise. However, because vessels are also under local control the chemical factors released due to the increase in

muscle metabolism in active muscle results in local arterial vasodilation, thus, decreasing the vessels resistance and allowing vasodilation in the working muscles (Shepherd and Vanhoutte, 1979).

The slight decrease in total peripheral resistance during exercise is the result of the vasodilation of arterial vessels in active muscles, caused by the release of metabolites (lactic acid, potassium and phosphate) which overrides the sympathetic nervous activity, thus resulting in an increase in blood flow to the active muscle and a decrease in blood flow to the viscera and nonworking muscles (Patton et al,1989).

Blood pressure

The increase in sympathetic discharge throughout the body during exercise, which causes vasoconstriction of most of the blood vessels except those in the active muscles increases the arterial blood pressure during exercise. The increase in blood pressure during exercise provides the exercising muscle with the oxygen need (Palatini, 1988). During continuous moderate exercise blood pressure will increase usually only 20 to 40 mmHg because of the vasodilation occurring in the large muscle mass (Guyton, 1982).

During dynamic exercise, the blood flow to nonworking muscles is decreased due to vasoconstriction (Clausen, 1977). Although there is vasoconstriction in nonworking muscles, the vasodilation

of the blood vessels in the working muscles has a much greater effect and the end result is an overall decrease in vascular resistance. As a result, a decrease in vascular resistance would cause blood pressure to decrease if all else remained constant. However, the large increase in cardiac output outweighs the decrease in vascular resistance resulting in an increase in systolic blood pressure (Lamb,1978). Therefore, the increase in systolic blood pressure is mainly associated with an increase in cardiac output.

Diastolic blood pressure on the other hand decreases slightly or remains unchanged during exercise (Palatini, 1988).

In summary, at the onset of exercise heart rate, cardiac output and blood pressure increase rapidly to meet the oxygen demands of the working muscles. The magnitude of the increase is dependent on the type and intensity of the exercise. There is a decrease in total peripheral resistance resulting from the vasodilation in the working muscles. All physiological changes are the direct result of the oxygen demands of the body and the increase in blood flow needed to the active muscles for oxygen extraction.

ARTERIAL BARORECEPTORS

The baroreceptors located in the vessels leading to the brain (carotid-sinus) and in the major arterial trunk (aortic-arch) act

to regulate blood pressure via the stretch receptors in the arteries (Sherwood, 1989). An increase in blood pressure stretches the receptors in the arteries and stimulates the baroreceptors to increase their rate of firing (Selkurt, 1971). As a result, the cardiovascular centers are stimulated to inhibit sympathetic nervous activity and to increase parasympathetic activity in order to lower blood pressure.

Parasympathetic stimulation will cause bradycardia, a reduction in the strength of contraction of the heart, and induce compensatory dilation of the vascular system resulting in a decrease in blood pressure (Astrand and Rodahl, 1986). However, during exercise, blood pressure is increased, as well as heart rate (McRitchie et al, 1976). This would suggest that the baroreceptors are either reset or desensitized during exercise.

Studies have observed that surgical interruption of baroceptor nerves causes a dramatic increase in blood pressure in virtually all conditions of exercise (Brody, 1981). This appears to be consistent with the findings of several other investigators who believe that the baroreceptors are inhibited or "turned off" during exercise (Bristow et al, 1971 and McRitchie et al, 1976). Therefore, if the baroreceptors are normally "turned off" or inhibited during exercise the dramatic increases in blood pressure observed, are not influenced by the baroreceptors, thus indicating that they do not influence the cardiovascular responses during exercise (McRitchie et al, 1976). McRitchie et al, (1976) compared the cardiovascular response of denervated and intact arterial

baroreceptors in dogs to exercise. They observed in intact dogs an increase in cardiac output, heart rate, a small increase in stroke volume, a significant increase in aortic pressure and decreased vascular resistance during exercise. They found similar results in denervated dogs. Their results suggest that the arterial baroreceptors are turned off during exercise and do not play a significant role in the cardiovascular response. Bristow et al, (1971) found similar responses in humans. In agreement with McRitchie et al, (1976), Cunningham et al, (1972) observed a marked loss of baroreceptor sensitivity during bicycle exercise. They observed that a drug induced rise in arterial blood pressure caused a reflex reduction in heart rate, but as work load increased this bradycardia was abolished. Hence, they concluded that bicycling at moderate to severe intensities reduces the sensitivity of the baroreceptors to regulate blood pressure during exercise. The fact that heart rate increased as well as arterial pressure shows that there is a decrease in the reflex sensitivity of the baroreceptors. Bristow et al, (1971) artificially increased arterial blood pressure at rest and during exercise using phenylephrine, which does not affect heart rate directly, to determine whether there is a decrease in baroceptor sensitivity or if baroreceptors are possibly reset. At rest, they injected a bolus of phenylephrine which induced an increase in systolic blood pressure of approximately 20 mmHg. They observed that large doses of the stimulus (phenylephrine) were required to produce the same increase in blood pressure following exercise. Therefore, they

concluded that baroreflex sensitivity is reduced in proportion to the severity of exercise. In contrast, several other investigators have reported results contrary to those cited above as to the importance of the baroreceptors (Robinson et al, 1966 ; Undesser et al, 1985). Robinson et al, (1966) and Undesser et al, (1985) concluded that there is a resetting of baroreceptors. Undesser et al, (1985) studied the effect of the baroreflexes resetting on conscious rabbits. The investigators artificially raised arterial blood pressure using phenylephrine or angiotensin II in lieu of exercise. They used the renal sympathetic nerve activity as an index of the efferent limb on the baroreflex. This procedure elevated blood pressure for periods of one to sixty minutes. Upon returning to resting levels, they noted that the renal sympathetic nerve activity remained suppressed for up to 90 minutes. They also noted that an activation of the arterial baroreceptors for a period of 5 minutes or more will elicit an alteration of the baroreceptor setpoint. They concluded that the baroreceptors were reset rapidly and that they monitored sympathetic activity to limit the increase in blood pressure above safe levels (Bevegard and Shepherd, 1967).

The baroreflexes at rest respond to an increase in arterial blood pressure by reflex bradycardia and decrease in vascular resistance. However, during severe exercise, blood pressure is increased as well as heart rate. This would suggest that the arterial baroreceptors are reset at a higher level to allow blood pressure and heart rate to increase in order to meet the demands of

the body (Rowell, 1986). Baroreceptors are not completely "turned off" during exercise, since they limit the increase in blood pressure by further reducing vascular resistance throughout exercise, thereby ensuring that safe levels of blood pressure are not exceeded.

Physiology of heat exposure

Thermoregulatory responses to heat exposure on the body have been studied by several investigators (Rowell, 1974, Nadel, 1979 and Rowell, 1986). Changes in body temperature can affect cellular structures, impede enzyme systems and hinder physical and cardiovascular processes which take place in the body (Astrand, and Rodahl 1986). During heat exposure, the body's heat content increases (Astrand and Rodahl,1986) and thus causes several thermoregulatory responses. To maintain heat balance, during heat stress, the principal control mechanism is to manipulate the circulation of blood flow from the deep veins to the periphery, thus controlling heat transfer from the body core to the environment (Hellon et al, 1983). Cardiovascular and thermal changes occur to facilitate body temperature regulation.

THERMAL CHANGES TO HEAT EXPOSURE AT REST

Core temperature is measured as esophageal (Tes), rectal

(Tre), tympanic or right atrial blood (Tra) temperature (Rowell, 1983a). Between 97 and 100 °F, body temperature is subject to precise regulation (Sherwood, 1989). The skin temperature is, generally, lower than the core temperature and fluctuates between 68 and 104 °F without any adverse effects (Sherwood, 1989).

Exposure to heat, above 28°C, in the resting human causes the heat content of the body to increase. If the temperature in the surrounding area is lower than that of the skin, heat loss is promoted through radiation and convection (Astrand and Rodahl, 1986). The body, therefore, transfers excess heat into the environment away from the body. This is accomplished through the vasodilation of the skin vessels and the redirection of the blood from the deep veins to the periphery to increase the conductance of heat transfer through the skin (Rowell, 1986). Rowell et al, (1971) observed that direct body heating using water perfusion suits to maintain skin temperature at neutral temperature of 35°C for 30 min (control), then rapidly increasing temperature to 40.5°C for 43 to 50 minutes (heating period) caused an increase in core temperature. Core temperature increased, as measured by Tra temperature, from 36.72°C to 39.44°C. Wyss et al, (1975) also concluded that an increase in skin temperature (Tsk) from 37.5°C rapidly to 40°C increased Tra to approximately 38°C.

These small but important changes in core temperature determine the relative importance of the temperature regulating mechanism to maintain internal temperature within safe limits. This

is achieved by inducing thermoregulatory responses to facilitate the transfer of heat away from the body through vasodilation of the skin vessels.

BLOOD FLOW

During heat stress, the body adjusts the circulation by shifting blood flow from the deep veins to the peripheral veins in order to increase tissue conductance for heat dissipation (Astrand and Rodahl, 1986). There is, however, no increase in muscle blood flow during heat stress (Detry, 1972).

The study of Detry et al (1972) measured changes in forearm muscle blood flow due to heat stress by direct whole body heating. They used water perfused suits to elevate temperature to 47.5°C which they maintained for 30-45 min, and determined the clearance rate of 4-iodoantipyrine ^{125}I(^{125}IAP), which had been previously infused into the brachioradialis muscle, to determine blood flow to both arms. Their results concluded that during direct body heating, using ^{125}IAP (to determine blood flow), no increase in skeletal muscle blood flow occurred. However, an increase between 20 to 30 ml/min in forearm blood flow, during whole body heating, was observed as estimated by cardiac output. Johnson et al, (1976), also in agreement, found that locally increasing arm skin temperature to 42.5°C in 15 men was not associated with increased blood flow in the underlying muscles.

The increase in skin blood flow during heat stress at rest can be estimated by the increase in cardiac output (Rowell, 1983a).

Rowell (1983a) concluded that the increase in cardiac output observed during heat stress must be directed to the skin. As a result, there is a reduction of blood flow to renal and splanchnic areas which usually receive 65-70% of resting cardiac output (Rowell, 1983a). Hales et al (1979), used radioactive microspheres to measure the skin blood flow and distribution of cardiac output in awake heat stressed baboons. They measured the blood flow to most major organs in eight baboons. Their results showed that the fraction of cardiac output distributed to the skin increased from 3% to 14% and resulted in a compensatory decrease in splanchnic and renal blood flow. They concluded that only skin blood flow increased significantly when core temperature was elevated with no increase in cardiac output. Despite the differences seen in man, where both skin blood flow and cardiac output are increased, the baboon exhibits the same magnitude of splanchnic and renal vasoconstriction. Therefore, the baboon is considered a good indicator of changes in regional blood flow in man during heat stress. Johnson et al, (1976) also concluded that, during local heating, vasodilation in the skin vessels occur steadily resulting in increasing blood flow to the skin with increases in skin temperature of 42°C in 60 minutes.

In brief, during local or whole body heating the body dissipates heat through vasodilation of the skin vessels and increased conductance of the tissue. The increase in blood flow to

the skin during heat stress can be estimated by the increments in cardiac output. Throughout heat exposure at rest, there is no observed increase in muscle blood flow.

CARDIOVASCULAR ADJUSTMENTS TO HEAT EXPOSURE AT REST

Heat stress causes several cardiovascular changes. Exposure to extreme heat causes an immediate increase in heart rate and cardiac output when Tsk increases (Rowell, 1986). Mean arterial blood pressure is usually maintained; however, during the first 30 minutes it falls slightly by 10 to 15 mmHg but gradually rises back up to resting levels (Rowell, 1983a). This is a result of the high capacitance of the skin vessels, which vasodilate to increase heat transfer, causing a decrease in central blood volume thus reducing mean arterial pressure.

Rowell et al, (1969) studied the cardiovascular responses to sustained high skin temperature in resting men. They observed, during heat exposure, that cardiac output can be increased 7-10 ml/min primarily due to the increase in heart rate. Increasing skin temperature in water perfusion suits from 36.5 to 40.9°C, over a period of 40-53 min, produced a large increase in cardiac output, heart rate, a decrease in central venous pressure and total peripheral resistance, resulting in a decrease in right mean arterial pressure. The slight decrease in mean arterial pressure during body heating is believed to be caused by the decrease in

venous return and decreased cardiac filling pressure.

However, simultaneously there is an increase in sympathetic nerve activity associated with heat stress, resulting in an increase in contractility of the heart causing an increase in stroke volume despite the lowered filling pressure (Rowell, 1983b). This increase in stroke volume usually results in the gradual increase in blood pressure back to resting levels.

Kim et al, (1979) determined that in humans increased body temperature to 39.5°C and 41.5°C resulted in a parallel increase in heart rate and cardiac index. These increases were directly related to the increases in plasma norepinephrine levels (NE). The progressive rise of NE and splanchnic vascular resistance would suggest that heat stress increases sympathetic nervous activity (Escourrou et al, 1984). This increase in sympathetic outflow to the heart increases heart rate (Rowell, 1974) and induces vasoconstriction in visceral organs. Kim et al, (1979) also noted that as body temperature rose, mean arterial pressure decreased due to a reduction in total peripheral resistance.

During heat exposure there is a rise in skin temperature which is accompanied almost immediately by an increase in heart rate, cardiac output, and a decrease in both total peripheral resistance, splanchnic blood flow and a slight decrease in mean arterial pressure (Rowell, 1983).

Physiological responses to cold exposure

Thermal stress resulting from the environment evokes changes

in skin temperature (Tsk) preceding any change in core temperature (Tc). The changes of Tsk are sensed by the thermoreceptors in the skin. Therefore, a decrease in Tsk will increase the thermal gradient (Tc-Tsk) causing heat loss if the peripheral vessels of the skin do not vasoconstrict and cutaneous blood flow is not decreased. Any change in Tsk will elicit a reflex vasoconstriction of the peripheral vessels.

Thermal response to cold exposure

Exposure of man to cold causes several physiological responses. Cold exposure in the resting man causes the heat content of the body to decrease. The body will, therefore, try to conserve heat by vasoconstriction of skin blood vessels causing a decrease in tissue conductance thus, decreasing heat loss.

Vallerand and Jacobs (1989) observed that upon exposure to cold for a period of 2 hours at a temperature of 10°C caused a 3.1 + 0.2°C reduction in mean body temperature. Moreover, Hong and Nadel (1979) and Kruk et al (1990) also found that skin temperature fell during rest in a cold environment. Olschewski and Bruck (1988) reported that following exposure to 5 to 10°C air for 15 minutes caused Tc to decrease by 0.2°C. The decrease in mean body temperature causes the body to increase its heat production to

protect itself from hypothermia. A drop in body temperature will induce shivering to promote heat production.

Shivering consist of rhythmic, oscillating skeletal muscle contractions (Sherwood, 1989). This is a very effective way of increasing heat production in the body. Shivering can increase heat production 2 to 5 folds (Sherwood, 1989). Raven et al (1970) observed that immediately upon exposure to a cold environment of 5°C heat production increased markedly due to shivering.

In brief, upon exposure to cold, the body tries to conserve heat by redirecting blood flow to the deep veins away from the surface to prevent heat loss. In addition, to produce heat to maintain body temperature the body will induce shivering.

Cardiovascular responses to cold exposure

Several cardiovascular adjustments occur to accommodate the body to the cold. Exposure to cold at rest is accompanied by an increase in vascular resistance, causing a reduction in blood flow to the skin, increase in oxygen consumption, cardiac output, stroke volume and arterial blood pressure (Raven et al, 1975).

Heart rate response has been noted to be slightly lower or constant throughout cooling (Doubt, 1991) compared to a neutral temperature. Therminarias (1989) noted that heart rate was lower during cold exposure of -2°C in comparison to warmer temperatures. However, O'Hanlon and Horvath (1970) found no significant change in

heart rate during cold air exposure.

Raven et al (1975) noted that cardiac output and oxygen consumption increased with a decrease in mean body temperature. They postulated that the increase in cardiac output was primarily due to the increase in stroke volume. As reported by Raven et al, (1970), they also noted that the elevated stroke volume was a result of the accompanying increase in peripheral vascular resistance which possibly augmented venous return. They also observed an increase in blood pressure as a result of the increase in cardiac output. A similar response to 4.5-6.5°C and 10°C cold exposure was observed by Hanna et al (1975) and Wagner and Horvath, (1985), respectively. These increases are believed to be due, in part, to an elevation in circulating catecholamines (Wilkerson et al, 1974) and to the shivering response (O'Hanlon and Horvath, 1970).

The shivering response to cold exposure is believed to be one of the main causes of the increase in cardiac output. Shivering results in an increase in VO_2 to meet the oxygen demands (Nadel, 1977). The circulatory adjustments to cold exposure result in an increase in vascular resistance in the skin vessels causing an elevation in intrathoracic blood volume, resulting in an augmentation in stroke volume (Graham, 1988).

In brief, cold exposure causes an immediate vasoconstriction of the peripheral arteries, resulting in an increase in central blood volume. The elevation in central blood volume increases stroke volume and blood pressure. In addition, the body initiates

shivering to elevate its heat content, therefore, causing cardiac output to increase to meet the oxygen demands. The increase in cardiac output, stroke volume, vascular resistance and blood pressure are further stimulated by the increased circulation of catecholamines in responses to cold exposure. However, there is no notable rise in heart rate despite the oxygen demands and augmented catecholamine secretion. This is believed to be due to the influence of the baroreceptors to lower heart rate when blood pressure is increased.

As blood pressure increases in response to the cold exposure, the baroreceptors are activated to decrease blood pressure, thus causing a decline in heart rate. The decline in heart rate is a compensatory measure to limit the increases in blood pressure within safe limits while ensuring body temperature.

POST-EXERCISE BLOOD PRESSURE

After cessation of exercise, there is a decrease in blood pressure towards resting values. Some authors have observed that blood pressure behavior following exercise tends to decrease gradually towards pre-exercise values (Korner, 1952). In contrasts, others have observed rapid reductions in post-exercise blood pressure (Hannum and Kasch, 1981 and Bennett et al, 1984), while some have reported reductions below pre-exercise values for extended periods of time (Boyer and Kasch, 1970, Paulev et al, 1984, Choquette and Ferguson, 1973).

Several complex physiological mechanisms have been studied in order to determine the mechanism(s) responsible for the post-exercise reductions in blood pressure. They include, decrease in vascular resistance resulting in venous pooling, decrease catecholamines, resetting or impairment of baroreceptors and decreased blood volume. (Duncan et al, 1985, Undesser et al, 1985, Saltin et al, 1964, Kaufman et al, 1987).

MECHANISMS INVOLVED IN POST-EXERCISE BLOOD PRESSURE

VASCULAR RESISTANCE

During aerobic activity, vascular resistance is reduced due to local vasodilation of the vessels within the working muscles (Hannum and Kasch, 1981). This increased blood flow to the muscles is required to meet the oxygen demands placed on the body. Moreover, core body temperature is also elevated resulting in cutaneous vasodilation to cool the body (Wilcox et al, 1982). After exercise, the vascular resistance is believed to maintain this lowered state, thus, venous return is reduced due to the cessation of the muscle contractions. Moreover, cutaneous and skeletal vasodilation persists, thus leading to a fall in blood pressure. Wenger et al (1975), found a linear relationship between blood flow

and oesophageal temperature. They postulated that an increase in core temperature causes more vessels to dilate and thus causes an increase in blood flow. Therefore, following exercise, vasodilation would persist in the working muscles and cutaneous beds until core temperature is restored (Wenger et al, 1975). Fleg and Lakatta (1986) noted that after a graded exercise test, with recovery in the seated position, ten of the fifteen subjects experienced rapid reductions in blood pressure resulting in dizziness, nausea and yawning. The symptoms were alleviated by having the subjects lay down. They postulated that the reduction in post-exercise blood pressure may have been caused by an excessive fall in peripheral vascular resistance therefore accentuating venous pooling.

Fitzgerald (1981) observed a reduction in blood pressure below resting level, immediately following termination of low intensity exercise and which lasted between four to ten hours. The cause of this reduction was believed to be due to an increase in venous pooling in the extremities resulting from decreased vascular resistance and persistent vasodilation in the muscles.

Overton et al (1988) measured vascular resistance in rats by using indwelling arterial catheters to measure the regional blood flow to the iliac, superior mesenteric and renal arteries. The hypertensive rats exercised at 60 to 70% $\dot{V}O_2$ max. for 20 and 40 minutes. Although, a significant reduction in post-exercise blood pressure was observed, in contrast, they observed no marked pre to

post-exercise reduction in the regional vascular resistance. It was postulated that the mechanism responsible for this reduction in blood pressure was a decrease in cardiac output. Thus, they postulated that a decrease in vascular resistance would not seem to persist after exercise in rats.

In summary, during dynamic exercise there is a decrease in vascular resistance, an increase in core temperature resulting in cutaneous vasodilation and profound vasodilation in skeletal muscle (Wilcox et al, 1982). Post-exercise venous return is decreased due to the lack of muscle contractions, while skeletal and cutaneous vasodilation persist.

CATECHOLAMINES

During aerobic exercise, sympathetic nervous activity is increased and the parasympathetic activity is decreased. This results in an increased level of circulating catecholamines. Since sympathetic tone is increased by catecholamine concentration (Watson et al, 1980), it is conceivable that a reduction in the sympathetic stimulation would decrease sympathetic tone and, thus result in a decrease in blood pressure.

Floras and coworkers (1986) noted a reduction in sympathetic activity following 45 minutes of exercise at 70% heart rate reserve

in borderline hypertensives. The authors concluded that the sympathetic nerve activity in muscle was reduced from 28+ bursts/100 heart beats before to 15+4/100 heart beats after exercise. Floras et al (1991) repeated the same study but with normotensive individuals to determine if they displayed a similar decrease in systolic blood pressure and sympathetic nerve activity in muscle or both in comparison to hypertensives, following 45 minutes of sub-maximal exercise. Their results indicated that there was no decrease in muscle sympathetic nerve activity in normotensives. Hence, it was postulated that the reductions seen in borderlines hypertensives were probably due, in part, to a transient suppression of the augmentation of central sympathetic outflow.

In addition, Overton et al (1988), also found no evidence of a decrease or inhibition in sympathetic nerve activity in rats following exercise. They postulated that any reductions in mesenteric resistance would be the result of a decrease or inhibition in sympathetic nervous activity. The lack of change in pre- to post-renal mesenteric resistance following exercise would not have occurred had there been a reduction in sympathetic activity.

Hagberg et al (1984) also observed that a decrease in resting plasma catecholamines did not occur following a nine month training program in hypertensives and normotensive adolescents. The

adaptation to the sympathetic nervous system to training in hypertensives, which can be reflected by catecholamine response, was observed to be no different than that found in normotensives. Although the decrease in resting blood pressure cannot be attributed to a decrease in sympathetic neural activity it is still possible that adrenergic factors may be involved.

Prolonged exposure of cells or receptors to a high concentration of the same hormone tends to results in a decrease sensitivity or tolerance to that hormone. This is known as desensitization. Catecholamines are released in high concentration during exercise (Watson et al, 1980) and act on the adrenergic receptors. The increased stimulation of the receptors due to catecholamines results in the desensitization or tolerance of the receptors to catecholamines, thus resulting in reductions in blood pressure.

Further studies, by Friedman et al (1987), Duncan et al (1984) and Boone et al (1992), have examined the desensitization of the beta-adrenergic receptors in contrast to the decrease or inhibition of sympathetic nervous activity as a possible reason for the decrease in blood pressure.

Friedman et al (1987) tested this theory that desensitization of canine cardiac beta-adrenergic receptors occurs due to exercise. They utilized a chronotropic dose (CD 25) of isoproterenol that elevated the dogs heart rate by 25/beats/min at rest and again

after heart rate had returned to resting levels following a 60 minute bout of exercise. They noted that 12 out of 13 dogs exhibited less sensitivity to the chronotropic effect of cardiac beta-adrenergic receptor stimulation following exercise. They concluded that a 60 minute bout of exercise in dogs did result in a transient reduction in the chronotropic sensitivity of beta-adrenergic receptors stimulation for approximately 3 hours following exercise.

Duncan et al (1984) studied a similar beta-adrenergic blockade utilizing isoproterenol. However, they observed no change in the response of beta-receptors (CD25) to the exogenous administration of isoproterenol in their subjects. They concluded that it was unlikely that a decrease in the sensitivity of beta-receptors caused a reduction in blood pressure. Instead, they believe it was a decrease in sympathetic activity rather than a desensitization of the receptors.

Boone et al (1992) attempted to determine the effects and possibility of opioid receptor involvement in the reduction in post-exercise blood pressure. They examined the reversal of severe post-exercise reductions in blood pressure by using an antagonism, to opioid sensitive receptors (naloxone) in normotensive subjects. It would appear that naloxone augments the cardiovascular response during elevated sympathetic activity. However, exogenous opioids suppress the cardiovascular response. Once severe reductions in

post-exercise blood pressure were noted (after 30 minutes of sub-maximal exercise) the subjects were infused with naloxone. It reversed the reduction response from minute 15-27 during recovery. This would suggest that the post-exercise reduction in blood pressure response may be mediated through opioid sensitive receptors.

In brief, it is speculated that following exercise there is a desensitization of the adrenergic receptors to catecholamines, thus resulting in a reduction of the effects of the sympathetic nervous system on blood pressure. Therefore, a decrease in post-exercise blood pressure can be a result of the decreased sensitivity to sympathetic nervous system.

BARORECEPTORS

The baroreceptors are the most important short term regulators of blood pressure (Sherwood, 1989). Any change in blood pressure will trigger an immediate response of the baroreceptors. If there is a decrease in blood pressure there will be a decrease in the baroreceptor activity and vice versa (Sherwood, 1989). Baroreceptor resetting and desensitization after exercise has been studied as a possible cause in the reduction of post-exercise blood pressure.

Bennett et al (1984) studied the possibility of impairment of

the baroreceptors and found no evidence of this occurring in response to exercise. They measured the competence of the baroreflexes by lowering the lower body sub-atmospheric pressure (LBSP) 30 minutes before, 30 min and 60 min after exercise. They noted that the reduction of blood pressure following exercise was well maintained during LBSP. In addition, exposure to LBSP following exercise produced a greater increase in heart rate as well as in the forearm vascular resistance than before exercise. This shows that a resetting of the baroreflex mechanism and increased sensitivity occurs. Therefore, they concluded that the reduction in blood pressure was not caused by impairment of the baroreflex response but rather by a resetting of the baroreceptors.

As indicated previously , Undesser et al (1985) studied the baroreflex control in conscious rabbits. They concluded that the elevation of blood pressure during exercise may suppress the baroreflex response and for sometime thereafter. Therefore, the resetting of baroreceptor may provide an explanation for the reductions in blood pressure following exercise.

Sleight (1983) studied how much the variability of arterial pressure was influenced by the arterial baroreceptors. He measured intra-arterial blood pressure in sixty-one subjects for a period of 24hrs. The baroreflex sensitivity was measured throughout the day by injecting a pressor agent and measuring the relation between the pressure rise and reflex cardiac slowing. Their multivariate

analysis indicated that the pressure was mostly influenced by the baroreceptors. Therefore, the pressure rise in response to various stimuli throughout the day, including bicycle exercise was strongly determined by baroreceptor sensitivity.

The increase in blood pressure during exercise results in a resetting of the baroreceptors to a higher level, in order to meet the demands of exercise. Following exercise, the resetting of the baroreceptors at a higher levels allows a reduction in blood pressure to occur.

HYPOVOLEMIA

Hypovolemia or decreased blood volume is thought to be a possible factor in the decrease of post-exercise blood pressure.

The reduction in blood volume is usually attributed to excessive sweating or dehydration of the subject. However, a reduction in blood volume is generally not evident in most studies (Bruce et al, 1959). The decrease in blood volume, following moderate exercise, is generally too little to elicit any drastic changes in blood pressure (Saltin et al, 1964).

Greenleaf et al (1981) noted that there was an increase in plasma volume during aerobic exercise. Therefore, a decrease in blood volume would not seem to be a possible mechanism of post-

exercise reductions in blood pressure. Thus, a decrease in blood volume is not apparent in mild to moderate exercise and is not considered a possible cause of the reduction in post-exercise blood pressure toward baseline values.

Summary

Several possible mechanisms for the reduction of post-exercise arterial blood pressure have been suggested. These include: decreased vascular resistance due to vasodilation, decreased catecholamines, baroreceptors resetting or desensitization, and decreased blood volume. It has yet to be concluded which mechanism or a combination of the mechanisms will result in a reduction in blood pressure following exercise.

It has been observed that in most cases total peripheral resistance is decreased following exercise, as a result of local vasodilation in the working muscles which seems to persist after cessation of exercise. Yet others (Hagberg et al, 1983 and Hagberg et al 1984) have observed a marked increase in vascular resistance, in hypertensives as well as normotensives, immediately following exercise. It has been concluded that there is no decrease in catecholamine concentration following exercise, however, the possibility that the arterial receptors are desensitized has been

suggested (Floras, 1991, Overton et al, 1988, Friedman et al, 1987 and Duncan et al, 1984). Several investigators examined the desensitization of the beta-adrenergic receptors in contrast to the decrease or inhibition of sympathetic nerve activity as a possible reason for the decrease in blood pressure (Friedman et al (1987) and Boone et al,1992). Friedman et al (1987) reported that there is a desensitization of the receptors in dogs following exercise. However, Watson et al (1984) concluded that this did not occur in humans.

Several investigators (Bennett et al, 1984, Undesser et al, 1985 and Sleight 1983) studied the possibility of the involvement of baroreceptors in post-exercise blood pressure and noted that the baroreceptors are possibly reset following exercise.

In conclusion, the possible mechanism(s) in the reduction of blood pressure following exercise have yet to be fully elucidated.

REFERENCES

American College of Sports Medicine. Resource Manual for guidelines for exercise testing and prescription. Lea and Febiger: Philadelphia, PA, 1988.

Astrand, P., and Rodahl, K.. Textbook of work physiology: Physiological bases of exercise. McGraw-Hill Inc: U.S.A., 1986.

Bannister, E.W. and Griffiths, J. (1972). Blood pressure levels of adrenergic amines during exercise. J Appl Physiol;33(5):674-676.

Bennett, T., Wilcox, G., and MacDonald, I.A. (1984). Post-exercise reduction of blood pressure in hypertensive men is not due to acute impairment of baroreflex function. Clin Sci;67:97-103.

Bevegard, S.B., and Shepherd, J.T. (1967). Regulation of the circulation during exercise in man. Phys Rev;47:178-213.

Boone, J.B. et al (1992). Opioid receptor modulation of post exercise hypotension. Med Sci Sports Ex;24(10):1108-1113.

Boyer, J.L. and Kasch, F.W. (1970). Exercise therapy in hypertensive men. JAMA;211:1668-1671.

Bristow, J.D., Brown, E.B., Cunningham, D.J.C., Howson, M.G., Petersen, E.J., Pickering, T.G., and Sleight, P. (1971). Effect of bicycling on the baroreflex regulation of pulse interval. Circulation Res;28:582-591.

Brody, M.J., (1981). New developments in our knowledge of blood pressure. Federation Proc;40:2257-2261.

Bruce, R.A., Leonard, A., Cobb, A., Katsura, S., Morledge, J.H., Andrus, W., and Fuller, T.J. (1959). Exertional hypotension in cardiac patients. Circulation;19:543-551.

Christensen, N.J. and Brandsborg, O. (1973). The relationship between plasma catecholamine concentration and pulse rate during exercise and standing. Eur J Clin Invest;3:299-306.

Choquette, G. and Ferguson, R.J. (1973). Blood pressure reduction in borderline hypertensives following physical training. C.M.A.J;108:699-703.

Clausen, J.P. (1977). Effect of physical training on cardiovascular adjustments to exercise in man. Physiol Rev;57(4):779-815.

Cunningham, D.J.C., Petersen, E., Peto, R., Pickering, T.G., and Sleight, P. (1972). Comparison of the effect of different types of exercise on the baroreflex regulation of heart rate. Acta Physiol Scand;86:444-455.

Davson, H., and Eggleton, M.G. Starling and Lovatt Evans Principles of Human Physiology. Lea and Febiger: Philadelphia, U.S.A., 1968.

Detry, J.M., Brengelmann, G.L., Rowell, L.B., and Wyss, C. (1972). Skin and muscle components of forearm blood flow in directly heated resting man. J Appl Physiol;32(4):506-511.

Doubt, T.J. (1991). Physiology of exercise in the cold. Sports Med;11(6):367-381.

Duncan, J.J., Farr, J.E., Upton, J., Hagan, R.D., Oglesby, M.E., and Blair, S.N. (1985). The effects of aerobic exercise on plasma catecholamines and blood pressure in patients with mild essential hypertension. JAMA;254(18):2609-2613.

Escourrou, P., Johnson, G., and Rowell, L.B. (1984). Hypoxemia increases plasma catecholamine concentration in exercising humans. J Appl Physiol;57(5):1507-1511.

Fitzgerald, W. (1981). Labile hypertension and jogging: new diagnostic tool or spurious discovery ?. Br Med J;282:542-544.

Fleg, J.L., and Lakatta, E.G. (1986). Prevalence and significance of post-exercise hypotension in apparently healthy subjects. Am J Cardiol;57:1380-1384.

Floras, J.S., Aylward, P.E., Sinkey, C., and Mark, A.L. (1986). Postexercise decrease in blood pressure are accompanied by decrease in muscle sympathetic nerve activity. Clin Res;34(2).

Floras, J.N. and Senn B.L. (1991). Absence of post exercise hypotension and sympathoinhibition in normal subjects: Additional evidence for increased sympathetic outflow in borderline hypertension. Can J Cardiol;7(6):253-258.

Friedman, D.B., Ordway, G.A., and Williams, S. (1987). Exercise induced functional desensitization of canine cardiac B-adrenergic receptors. Am Physiol Society;62(4):1721-1723.

Graham, T.E. (1988). Thermal, metabolic, and cardiovascular changes in men and women during cold stress. Med Sci Sports Exerc;20(5):S185-S192.

Greenleaf, J.E., Sciaraffa, D., Shvartz, E., Keil, L.C. and Brock, P.J. (1981). Exercise training hypotension: implications for plasma volume, renin, and vasopressin. J Appl Physiol;51(2):298-305.

Guyton, A.. Basic human physiology. W.B. Saunders Company:U.S.A., 1982, p219-227.

Hagberg, J.M., Goldring, D., Ehsani, A.A., Heath, G.W., Hernandez, A., Schechtman, K., and Holloszy, J.O. (1983). Effect of exercise training on the blood pressure and hemodynamic features of hypertensive adolescents. Am J Cardiol;52:763-768.

Hagberg , J.M., Goldring, D., Heath, G.W., Ehsani, A.A., Hernandez, A., and Holloszy, J.O. (1984). Effect of exercise on plasma catecholamines and hemodynamics of adolescent hypertensives during rest, submaximal exercise and orthostatic stress. Clin Physiol;4:17-124.

Hales, J.R.S., Rowell, L.B. and King, R.B. (1979). Regional distribution of blood flow in awake heat-stressed baboons. Am J Physiol;237(6):H705-H712.

Hanna, J.N., Hill, P.M., and, Sinclair, J.D. (1975). Human cardiorespiratory responses to acute cold exposure. Clin Experim Pharmacol Physiol;2:229-238.

Hannum, S.M., and Kasch, F.W. (1981). Acute postexercise blood pressure response of hypertensive and normotensive men. Scand J Sports Sci.;3(1):11-15.#3731

Harrison, M.H.(1986). Heat and exercise: Effects on blood volume. Sports Med;3:214-223.

Hellon et al. (1983). Thermoreceptors. In handbook of physiology: the cardiovascular system, peripheral circulation and organ blood flow, sect 2, vol III, chapt 18. American Physiol Society: Bethasda, MD, pp659-673.

Hong, S., and Nadel, E.R. (1979). Thermogenic control during exercise in a cold environment. J appl Physiol: Respirat Environ exercise Physiol;47(5):1084-1089.

Jensen, D. The principles of physiology. Appleton-Century-Crofts: U.S.A., 1980.

Jette, M., Campbell, J., Mongeon, J., and Routhier, R. (1976). The Canadian home fitness test as a predictor of aerobic capacity. Can Med Ass J;14:680-682.

Jette, M. (1983). Guide for anthropometric measurements of Canadian adults (1st ed.). Montreal: C.T. Management and Consultants Inc.

Jette, M., Landry, F., and Sidney, K. (1991a). Blood pressure response to the Canadian Aerobic Fitness Test. Can J Public Health.

Jette, M., Quenneville, J., and Sidney, K. (1991b). Fitness testing and counselling in health promotion. Can J Sport Sci.

Johnson, J.M., Brengelmann, G.L., and Rowell, L.B. (1976). Interactions between local and reflex influences on human forearm skin blood flow. J Appl Physiol;41(6):826-831.

Kaufman, F.L., Hughson, R.I., and Schaman, J.P. (1987). Effect of exercise on recovery blood pressure in normotensive and hypertensive subjects. Med Sci Sports Exerc;19(1):17-20.

Kilgour, R.D., Gariepy, P., and Rehel, R. (1993). Cardiovascular responses during recovery from exercise and thermal stress. Aviat Space Environ Med;64:224-229.

Kim, Y.D., Lake, C.R., Lees, D.E., Schuette, W.H., Bull, J.M., Weise, V., and Kopin, I.J. (1979). Hemodynamic and plasma catecholamine responses to hyperthermic cancer therapy in humans. Am J Physiol;237(5):H570-H574.

Korner, P.I.. (1952). The normal human blood pressure during and after exercise, with some related observations on changes in the heart rate and the blood flow in the limbs. Aust J Exp Biol Med Sci;30:375-384.

Kral, J., Chrastek, J., and Adamirova, J.. The hypotensive effect of physical activity in hypertensives. In Prevention of ischemic heart disease. Springfield, IL, Charles Thomas Publishers. 1966, p359-371.

Kruk, B., Pekkarinen, Harri, M., Manninen, and Hanninen, O. (1990). Thermoregulatory responses to exercise at low ambient temperature performed after precooling or preheating procedures. Eur J Appl Physiol;59:416-420.

Lamb, D. Physiology of exercise: responses and adaptations. MacMillan Publishing Co: U.S.A., 1978.

McRitchie, R.J., Vatner, S.F., Boettcher, D., Heyndrickx, G.R., Patrick, T.A., and Braunwald, E. (1976). Role of arterial baroreceptors in mediating cardiovascular response to exercise. Am J Physiol;230(1): 85-89.

Nadel, E.R.. Problems with temperature regulation during exercise. Academic press: New York, 1977.

Nadel, E.R., Cafarelli, E., Roberts, M.F., and Wenger, B. (1979). Circulatory regulation during exercise in different ambient temperatures. J Appl Physiol: Respirat Environ Physiol;46(3):430-437.

O'Hanlon, J.F., and Horvath, S.M. (1970). Changing physiological relationship in men under acute cold stress. Can J Physiol Pharmocol;48:1-10.

Olschewski, H., and Bruck, K. (1988). Thermoregulator, cardiovascular and muscular factors related to exercise after precooling. J Appl Physiol;64(2):803-811.

Overton, J.M., Joyner, M.J., and Tipton, C.M. (1988). Reductions in blood pressure after acute exercise by hypertensive rats. Am Physiol Society;62(2):748-752.

Palatini, P. (1988). Blood pressure behavior during physical activity. Sports Med;5:353-374.

Patton, H.D., Fuchs, A.F., Hille, B., Scher, A.M., and Stuner, R. Textbook of physiology. W.B. Saunders company: Philadelphia, Pa, 1989.

Paulev, P., Jordal, R., Kristensen, O. and Ladefoged, J.(1984). Therapeutic effect of exercise on hypertension. Eur J Appl Physiol;53:180-185.

Raven, P.B., Niki, I., Dahms, T.E., and Horvath, S.T. (1970). Compensatory Cardiovascular responses during an environmental cold stress, 5°C. J Appl Physiol;29(4):417-421.

Raven, P.B, Wilkerson, J.E., Horvath, S.M. and Bolduan, N.W. (1975). Thermal, metabolic, and cardiovascular responses to various degrees of cold stress. Can J Physiol Pharmacol;53:293-298.

Robinson, B.F., Epstein, S.E., Beiser, G.D., and Braunwald, E. (1966). Control of heart rate by the autonomic nervous system. Circulation Research;19:400-411.

Rowell, L.B., Marx, H.J., Bruce, R.A., Conn, R.D., and Kusumi, F. (1966). Reductions in cardiac output, central blood volume, and stroke volume with thermal stress in normal men during exercise. J Clin Invest;45(11):1801-1816.

Rowell, L.B., Brengelmann, G.L. and Murray, J.A. (1969). Cardiovascular responses to sustained high skin temperature in resting man. J Appl Physiol;27(5):673-680.

Rowell, L.B., Detry, J.M., Profant, G.R., and Wyss, C. (1971). Splanchnic vasoconstriction in hyperthermic man - role of falling blood pressure. J Appl Physiol;31(6):864-869.

Rowell, L.B. (1974). Human cardiovascular adjustments to exercise and thermal stress. Physiol Rev;54:75-159.

Rowell, L.B. (1983a). Cardiovascular adjustments to thermal stress. In Handbook of physiology: the cardiovascular system, peripheral circulation and organ blood flow. section 2(3), chapt 27. American physiological society; Bethesda, MD, pp 967-1023.

Rowell, L.B. (1983b). Cardiovascular aspects of human thermoregulation. Circ Res;52(4):367-379.

Rowell, L.B.. Human circulation regulation during physical stress. Oxford University Press Inc.: New York, New York, 1986.

Saltin, B. and Stenberg, J. (1964). Circulatory response to prolonged severe exercise. J Appl Physiol;19(5):833-838.

Seals, D.R. and Hagberg, J.M. (1992). The effect of exercise training on human hypertension: a review. Med Sci Sports Exerc;16(3):207-215.

Selkurt, E. Physiology. Little, Brown and Company: Boston, MA, 1971.

Shepherd, J.T., and Vanhoutte, P.M. The human cardiovasuclar system: facts and concepts. Raven press: New York, NY, 1979.

Sherwood, L.. Human physiology. West Publishing Company: U.A.S., 1989.

Sleight, P. (1983). the influence of arterial baroreceptors in man on the variability of blood pressure and plasma catecholamines in man. Chest;83(2) Suppl:320-322.

Smith, J.J.,and Kampine, J.P. Circulatory physiology: The essentials. Williams and Wilkins:Baltimore, Md, 1980.

Therminarias, A., Flore, P., Oddou-Chirpaz, M.F., Pellerie, E., and Quirion, A. (1989). Influence of cold on blood lactate response during incremental exercise. Eur J Appl Physiol;58:411-418.

Undesser, K.P., Jing-Yun, P., Lynn, P., and Bishop, S.V. (1985). Baroreflex control of sympathetic activity after elevations of pressure in conscious rabbits. Am J Physiol;248:H827-34.

Vallerand, A.L, and Jacobs, I. (1989). Rates of energy substrates utilization during cold exposure. Eur J Appl Physiol;58:873-878.

Vander, A.J., Sherman, J.H., and Luciano, D.S. Human physiology. McGraw-Hill book company: U.S.A., 1980.

Wagner, J.A., and Horvath, S.M. (1985). Cardiovascular reactions to cold exposures differ with age and gender. J Appl Physiol;58(1):187-192.

Watson, R.D.S., Hamilton, C.A., Jones, D.H., Reid, J.L., Stallard, T.J. and Littler, W.A. (1980). Sequential changes in plasma noradrenaline during bicycle exercise. Clin Sci;58:37-43.

Wenger, C.B., Roberts, M.F., Nadel, E.R., and Stolwijk, J.A. (1975) Thermoregulatory control of finger blood flow. J Appl Physiol;38(6):1078-1082

Wilcox, R.G., Bennett, T., Brown, A.M., and MacDonald, I.A. (1982). Is exercise good for high blood pressure. Br Med J;285:767-769.

Wilkerson, J.E., Raven, P.B., Bolduan, N., and Horvath, S.M. (1974). Adaptations in man's adrenal function in response to acute cold stress. J Appl Physiol;36:183-189.

Wyss, C.R., Brengelmann, G.L., Johnson, J.M., Rowell, L.B., and Silverstein, D. (1975). Altered control of skin blood flow at high skin and core temperatures. J Appl Physiol;38(5):839-845.

Young, A.J. (1990). Energy substrate utilization during exercise in extreme environments. Ex Sports Sci Rev;18:65-117.

APPENDIX A

MODIFIED PHYSICAL ACTIVITY READINESS QUESTIONNAIRE (PAR-Q)

Modified Physical Activity Readiness Questionnaire (PAR-Q)[1]

Date:
Family Name: Given Name:
Age:

1. Has a physician ever said you have heart trouble? Yes No
2. Do you frequently have pains in your heart and chest? Yes No
3. Do you often feel faint or have spells of severe dizziness? Yes No
4. Has a physician ever said that your blood pressure was too high? Yes No
5. Do you suffer from any respiratory tract problem such as chronic bronchitis, asthma or emphysema? Yes No
6. Have you ever had or are you now suffering from any nervous disorder? Yes No
7. Do you suffer from any bone or joint problem which either has been or may be irritated by an exercise session? Yes No
8. Do you know of a valid medical reason why you should not be involved in either a regular exercise program or an exercise testing session? Yes No
9. At present, are you taking medication for blood pressure? Yes No
If yes, please specify:

Reason:
Name:
Dosage:

10. At present, are you taking any other type of medication, whether are prescribed or "over the counter"? Yes No
If yes, please specify:

Reason:
Name:
Dosage:

Date: Signature:

[1] Jetté, M., Quenneville, & J., Sidney, K. (1991) Fitness testing and Counselling in Health Promotion. Canadian Journal of Sports Sciences, 17: 194-198.

APPENDIX B

THESIS RESEARCH CONSENT FORM

CONSENT FORM FOR THE EFFECTS OF AMBIENT TEMPERATURE ON POST-EXERCISE BLOOD PRESSURE

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CHAIR OF FHS-HREC: Dr. Marie-des-Anges Loyer
Faculty of Health Sciences
Human Research Ethics Committee
Room 2009, 451 Smyth Road
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Date: _____ Name of Volunteer: _____

The title of this study is the "Effects of ambient temperature on post-exercise blood pressure". The purpose of this study is to determine the influence of temperature on post-exercise blood pressure in apparently healthy males.

I understand that I will be asked to participate in a preliminary and three experimental sessions lasting from one hour to two hours. The study involves exercising on a treadmill for 20 minutes at a heart rate of 140 beats per minute, then recovering in a controlled environmental chamber at one of three different temperatures (18,27 or 40°C) presented at random. Every effort will be made to conduct the test in such a way as to minimize discomfort and risk. However, I understand that just as with other types of fitness tests there are potential risks. These include episodes of transient lightheadedness, fainting, chest discomfort, leg cramps, nausea, falling of the treadmill, and very rarely heart failure. It is understood that I can withdraw from the exercise at any time I may wish.

The confidentiality of my data will be maintained at all times. Only the principal investigator and the advisor will have access to the data. My name will not appear on any data sheet but will be identified by a coded number.

I AM FREE To WITHDRAW this consent and to discontinue my participation at any time without penalty or discrimination.

Signature of volunteer: _____ Date: _____

Signature of witness: _____

APPENDIX C

THESIS RESEARCH LETTER OF INFORMATION

LETTER OF INFORMATION

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Faculty of Health Sciences
Human Research Ethics Committee
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This project is submitted in partial fulfilment of the degree of master of science in movement studies.

The purpose of the study is to determine the influence of temperature on post-exercise blood pressure in apparently healthy males.

The study consists of a preliminary and three experimental sessions conducted over a period of four separate days. Prior to the preliminary and experimental sessions, you will be asked not to participate in any form of physical activity, drink caffeine containing beverages or smoke for 12 hours.

At the preliminary session you will be briefed in detail as to the nature of the study and your involvement. You will then be asked to complete the Physical Activity Readiness Questionnaire (Par-Q). Your resting blood pressure will be measured to ensure that you have normal blood pressure at rest (<140/90 mmHg) so that you may be included in the study. Your resting heart rate, height, weight and skinfold measurements will then be recorded. You will be administered the Canadian Aerobic Fitness Test (CAFT), a low-moderate intensity test, where you will step up and down a two-step staircase (20.3 cm). Each stage of the test lasts 3 minutes. If your heart rate does not exceed a predetermined level, you will be asked to perform a second and third stage of the (CAFT). Your blood pressure and heart rate will be measured before commencing the test, during the one minute interval between each stage and in the three minute recovery phase following the test.

You will then be initiated to the following apparatus:

treadmill that you will be using in the exercise protocol, Finapres blood pressure monitor (blood pressure, heart rate and mean arterial pressure monitor), skin temperature monitor and surface thermistors which will be positioned at the following locations: middle of the forehead, midway on the back of the arm, chest and on the right thigh, the oxygen consumption equipment and the mouth piece valve assembly and the controlled environmental chamber which will be used for this study. You will then be asked to exercise for 10 minutes on a motorized treadmill to determine the intensity needed to elicit a heart rate of 140 beats/minute This intensity will be utilized in the experimental sessions. The duration of this preliminary session will be of approximately one hour.

At the onset of each of the three experimental sessions, resting blood pressure and heart rate will be recorded following a 5 minute resting period in the sitting position and also following a 5 minute resting period laying down.

You will then perform a 20 minute moderate intensity treadmill exercise to elicit a heart rate of 140 beats/minute (which includes a five minute warm-up) at a room temperature of 22-24°C. Immediately following exercise, you will then proceed into the controlled environmental chamber where you will recover for 60 minutes laying down on a rope cot. You will be exposed in a random manner to one of three different temperatures (18, 27 or 40°C) during one of the three testing sessions. Throughout the recovery period, your blood pressure, heart rate and mean arterial pressure will be measured using a Finapres Blood Pressure Monitor. In addition, blood pressure will also be measured at set intervals using a standard sphygmomanometer (Blood pressure cuff). Skin temperature will be continuously monitored throughout recovery using surface skin thermistors. Respiratory gases will be collected using a mouth piece assembly valve at selected intervals for the purpose of measuring oxygen consumption. The total duration of each session will be two hours.

Every effort will be made to conduct the tests in such a way as to minimize discomfort and risk. However, You should understand that just as with other types of fitness tests there are potential risks. These include episodes of transient lightheadedness, fainting, chest discomfort, leg cramps, falling of the treadmill, nausea, and very rarely heart failure.

This study is designed to enhance knowledge with respect to the mechanism(s) involved in post-exercise blood pressure response to different temperatures.

Your privacy and anonymity will be protected in the following manner: Data sheets will not identify you by name but by a coded number. All data files will be destroyed upon completion of the study. Only myself and my advisor will have access to the data.

You can withdraw or refuse to participate in the study without fear of reprisals.

APPENDIX D

TESTING SHEETS

Data work sheet

Name:_____ date:_____

wt:_____ ht:_____ step/min:_____

room temperature:_____ Barometric pressure: _____

Calibrate Q-Plex: _____ Finapres cuff: _____

Calibrate finapres: _____ Temp of chamber: _____

Ensure all hoses, mouth piece, finapres, and skin temperature equipment is functioning: _____

pre-test

sitting: BP: ____/____/____ HR: _____

supine : BP: ____/____/____ HR: _____

Hook up thermistors: _____ Put on polar watch: _____
Exercise 20 minutes: _____ Transition time: _____

In chamber

5 min	BP: ____/____/____	HR: _____
10 min	BP: ____/____/____	HR: _____
15 min	BP: ____/____/____	HR: _____
20 min	BP: ____/____/____	HR: _____
25 min	BP: ____/____/____	HR: _____
30 min	BP: ____/____/____	HR: _____

Transition change finapres: _____

35 min	BP: ____/____/____	HR: _____
40 min	BP: ____/____/____	HR: _____
45 min	BP: ____/____/____	HR: _____
50 min	BP: ____/____/____	HR: _____
55 min	BP: ____/____/____	HR: _____
60 min	BP: ____/____/____	HR: _____

WT after test: _____ WT CHANGE: _____
END OF SESSION

COMMENTS?_____

INVESTIGATOR: _____ ASSISTANT: _____

DATA SHEET

NAME: _____ DATE: _____

WT: _____ HT: _____

AGE: _____ CODE: _____

Phone #: _____

Name of saving program: a) finapres: _____
b) thermistors: _____
c) Q-PLEX: _____

THERMISTORS USED:
a) head no.: _____ head
b) tricep no.: _____ back
c) chest no.: _____ abd
d) thigh no.: _____ thigh
e) room no.: _____ toe or finger

TEST COMPLETION SHEET

1) STEP TEST: _____ date: _____

2) Pre-treadmill exercise: _____ date: _____

3) Experimental test at hr 140 b/min

a) 40 oC _____ date: _____

b) 27 oC _____ date: _____

c) 18 oC _____ date: _____

CAFT DATA SHEET

NAME: _____ DATE: _____

AGE: _____ HT: _____

WT: _____ CODE: _____

PHONE #: _____

Remove shoes

1) STEP TEST

Pre-test
sitting: BP____/____/____ HR: _____

CAFT

29 b/min STAGE 3 BP: ____/____/____ HR: _____
26 b/min STAGE 4 BP: ____/____/____ HR: _____
STAGE 5 BP: ____/____/____ HR: _____

Post-test

time 2:30-3:00 BP: ____/____/____
3:00-3:15 HR: _____

Predicted VO2 max:
see formula:

2) Skin fold measurements

sites	trial 1	trial 2	trial 3	trial 4	trial 5
tricep					
bicep					
subscapular					
iliac					
calf					

Formula:

% of bodyfat: _____

Pre-treadmill exercise:

Put on polar watch
HR 140 b/min steps/min: _____
HR 150 b/min steps/min: _____

CON'T

OXYGEN CONSUMPTION

1) TEMPERATURE :
CHANGE TISSOT :
OXYGEN % :
CO2 % :
BREATHS/MIN :

2) TEMPERATURE oC :
CHANGE TISSOT mm:
OXYGEN % :
CO2 % :
BREATHS/MIN :

3) TEMPERATURE :
CHANGE TISSOT :
OXYGEN % :
CO2 % :
BREATHS/MIN :

4) TEMPERATURE oC :
CHANGE TISSOT mm:
OXYGEN % :
CO2 % :
BREATHS/MIN :

5) TEMPERATURE :
CHANGE TISSOT :
OXYGEN % :
CO2 % :
BREATHS/MIN :

6) TEMPERATURE oC :
CHANGE TISSOT mm:
OXYGEN % :
CO2 % :
BREATHS/MIN :

APPENDIX E

PREDICTED MAXIMUM AEROBIC CAPACITY EQUATIONS

Predicted Maximum Aerobic Capacity Equations

Stage Completed

5th MaxV02 = 42.5 + [16.6 * 2.0066] - [0.12 * weight (kg)] - [0.12 * HR] - [0.24 * Age]

6th MaxV02 = 42.5 + [16.6 * 2.3453] - [0.12 * weight (kg)] - [0.12 * HR] - [0.24 * Age]

7th MaxV02 = 42.5 + [16.6 * 2.7657] - [0.12 * weight (kg)] - [0.12 * HR] - [0.24 * Age]

From: Jetté, M., Campbell, J., Mongeon, J. and Routhier, R. The Canadian Home Fitness Test as a predictor of aerobic capacity, 1976.

APPENDIX F

VO2 EQUATIONS FROM GAS COLLECTION ANALYSIS

Calculation of $\dot{V}O_2$ From Gas Collection and Analysis

From the raw data recorded on the previous work sheet, $\dot{V}O_2$ can be calculated for any gas collection period using the following 2 step-by-step procedures. The Tissot tank method of gas collection will be illustrated here, but of course, other methods may be used to determine $\dot{V}_E$ (ATPS) and record in step 10 below.

Simplified Method: $\dot{V}O_2 = \dot{V}_E$ (STPD) x "true O_2"

$\dot{V}CO_2 = \dot{V}O_2 \times R$

1. Barometric pressure _____ mmHg
2. Temperature of the collected gas _____ deg C
3. STPD correction factor _____ (from Table II-3)
4. Initial metre stick reading on Tissot _____ cm
5. Final stick reading _____ cm
6. Percent O_2 of collected gas _____ %
7. Percent CO_2 of collected gas _____ %
8. Subtract step 4 from step 5 _____ cm
9. Multiply step 8 by 1.332 (1 litre = 1.332 cm) = _____ litres
   Note: this depends on the size of the Tissot tank.
10. $\dot{V}_E$ (ATPS):
    Multiply step 9 by 2 (to convert to minute from 30 sec) = _____ $l \cdot min^{-1}$
11. $\dot{V}_E$ (STPD): Multiply step 10 by step 3 = _____ $l \cdot min^{-1}$
12. Use step 6 and step 7 with Figure II-1 nomogram to determine:
    "True O_2", _____ % and,
13. "R" _____
14. $\dot{V}O_2$: Multiply step 11 by step 12 and divide by 100 = _____ $l \cdot min^{-1}$
15. ~~Divide~~ Multiply step 14 by 1000 = _____ $ml \cdot min^{-1}$
16. $\dot{V}CO_2$: Multiply step 13 by step 14 = _____ $l \cdot min^{-1}$
17. $\dot{V}O_2$: Divide step 15 by body weight in kg = _____ $ml \cdot kg^{-1} \cdot min^{-1}$

Fitness Category _____ (from norms on page 2-12)

APPENDIX G RAW DATA

SBP18°C

	var00001	var00002	var00003	var00004	var00005	var00006	var00007	var00008	var00009
1	131.00	140.00	131.00	133.00	140.00	145.00	148.00	122.00	126.00
2	123.00	131.00	145.00	148.00	160.00	156.00	160.00	131.00	148.00
3	119.00	111.00	140.00	138.00	94.00	119.00	107.00	109.00	101.00
4	146.00	131.00	166.00	174.00	174.00	174.00	170.00	113.00	81.00
5	113.00	113.00	104.00	104.00	100.00	103.00	99.00	134.00	135.00
6	114.00	110.00	107.00	106.00	119.00	118.00	122.00	121.00	132.00
7	131.00	148.00	148.00	151.00	154.00	153.00	170.00	115.00	126.00
8	141.00	152.00	140.00	140.00	150.00	141.00	147.00	103.00	119.00
9	134.00	122.00	118.00	129.00	130.00	127.00	133.00	116.00	120.00
10	102.00	112.00	111.00	98.00	100.00	108.00	95.00	104.00	109.00

	var00010	var00011	var00012	var00013
1	136.00	122.00	129.00	133.00
2	123.00	138.00	133.00	132.00
3	120.00	121.00	122.00	123.00
4	106.00	186.00	196.00	171.00
5	138.00	138.00	138.00	132.00
6	132.00	132.00	126.00	136.00
7	120.00	122.00	124.00	121.00
8	116.00	128.00	170.00	117.00
9	116.00	131.00	126.00	134.00
10	116.00	99.00	116.00	113.00

SBP 27 °C

	var00001	var00002	var00003	var00004	var00005	var00006	var00007	var00008	var00009
1	119.00	116.00	111.00	108.00	110.00	103.00	116.00	106.00	103.00
2	114.00	112.00	110.00	104.00	106.00	106.00	108.00	112.00	100.00
3	118.00	112.00	113.00	113.00	113.00	110.00	108.00	101.00	102.00
4	110.00	124.00	123.00	111.00	120.00	119.00	129.00	109.00	116.00
5	129.00	137.00	133.00	138.00	134.00	126.00	127.00	119.00	124.00
6	118.00	115.00	107.00	110.00	112.00	108.00	106.00	106.00	111.00
7	111.00	111.00	119.00	109.00	95.00	99.00	98.00	95.00	99.00
8	121.00	132.00	131.00	125.00	130.00	123.00	131.00	122.00	115.00
9	105.00	110.00	106.00	106.00	104.00	105.00	102.00	85.00	94.00
10	117.00	109.00	107.00	110.00	106.00	115.00	106.00	102.00	104.00

	var00010	var00011	var00012	var00013
1	120.00	106.00	117.00	104.00
2	106.00	110.00	112.00	108.00
3	105.00	107.00	108.00	105.00
4	117.00	121.00	125.00	119.00
5	126.00	124.00	128.00	123.00
6	111.00	115.00	114.00	115.00
7	96.00	95.00	97.00	106.00
8	113.00	128.00	126.00	141.00
9	92.00	99.00	94.00	99.00
10	107.00	100.00	102.00	107.00

SBP 40℃

	var00001	var00002	var00003	var00004	var00005	var00006	var00007	var00008	var00009
1	105.00	105.00	102.00	93.00	91.00	94.00	92.00	108.00	105.00
2	128.00	124.00	121.00	114.00	109.00	106.00	117.00	85.00	89.00
3	111.00	113.00	102.00	113.00	113.00	114.00	117.00	92.00	99.00
4	133.00	128.00	133.00	127.00	130.00	128.00	126.00	120.00	124.00
5	104.00	104.00	103.00	108.00	106.00	105.00	104.00	102.00	87.00
6	118.00	120.00	122.00	126.00	128.00	126.00	130.00	118.00	110.00
7	112.00	114.00	109.00	101.00	105.00	106.00	102.00	94.00	94.00
8	125.00	117.00	109.00	113.00	118.00	107.00	108.00	93.00	86.00
9	99.00	104.00	102.00	100.00	103.00	100.00	103.00	102.00	99.00
10	112.00	114.00	112.00	106.00	103.00	93.00	100.00	100.00	96.00

	var00010	var00011	var00012	var00013
1	100.00	107.00	107.00	104.00
2	91.00	93.00	91.00	93.00
3	93.00	101.00	102.00	103.00
4	128.00	130.00	122.00	132.00
5	93.00	92.00	94.00	96.00
6	120.00	118.00	116.00	118.00
7	101.00	91.00	94.00	102.00
8	91.00	89.00	104.00	117.00
9	101.00	95.00	101.00	104.00
10	97.00	93.00	84.00	101.00

DBP & HR 18°C

	var00001	var00002	var00003	var00004	var00005	var00006	var00007	var00008	var00009
1	69.00	79.00	80.00	72.00	80.00	76.00	79.00	64.00	73.00
2	64.00	62.00	68.00	69.00	77.00	71.00	82.00	69.00	70.00
3	56.00	79.00	79.00	71.00	28.00	62.00	45.00	37.00	34.00
4	79.00	81.00	92.00	86.00	88.00	83.00	82.00	34.00	37.00
5	65.00	63.00	68.00	66.00	68.00	69.00	71.00	88.00	94.00
6	57.00	58.00	57.00	54.00	58.00	56.00	57.00	49.00	68.00
7	67.00	73.00	73.00	73.00	74.00	69.00	78.00	51.00	66.00
8	61.00	72.00	66.00	66.00	71.00	70.00	72.00	62.00	73.00
9	69.00	68.00	66.00	70.00	65.00	64.00	58.00	59.00	62.00
10	51.00	55.00	51.00	51.00	49.00	46.00	44.00	40.00	46.00

	var00010	var00011	var00012	var00013	var00014	var00015	var00016	var00017	var00018
1	72.00	72.00	76.00	81.00	84.00	77.00	72.00	70.00	67.00
2	64.00	71.00	72.00	76.00	98.00	84.00	73.00	68.00	65.00
3	45.00	49.00	53.00	54.00	76.00	71.00	64.00	72.00	56.00
4	33.00	53.00	50.00	53.00	90.00	86.00	82.00	78.00	74.00
5	96.00	101.00	101.00	99.00	93.00	83.00	75.00	74.00	68.00
6	68.00	68.00	66.00	81.00	87.00	82.00	79.00	65.00	75.00
7	60.00	64.00	68.00	67.00	92.00	85.00	82.00	77.00	79.00
8	69.00	76.00	71.00	79.00	79.00	77.00	72.00	63.00	65.00
9	65.00	65.00	65.00	70.00	88.00	82.00	78.00	79.00	71.00
10	43.00	43.00	48.00	48.00	89.00	87.00	84.00	84.00	79.00

	var00019	var00020	var00021	var00022	var00023	var00024	var00025	var00026
1	63.00	57.00	57.00	52.00	52.00	51.00	46.00	47.00
2	60.00	57.00	61.00	57.00	57.00	57.00	56.00	55.00
3	68.00	57.00	53.00	60.00	47.00	51.00	41.00	48.00
4	74.00	70.00	66.00	66.00	65.00	62.00	68.00	71.00
5	69.00	50.00	63.00	51.00	58.00	64.00	57.00	62.00
6	70.00	74.00	68.00	74.00	72.00	68.00	35.00	54.00
7	76.00	71.00	74.00	71.00	73.00	68.00	67.00	66.00
8	59.00	64.00	48.00	55.00	54.00	53.00	54.00	49.00
9	72.00	64.00	62.00	62.00	63.00	63.00	59.00	58.00
10	81.00	77.00	66.00	67.00	64.00	62.00	63.00	68.00

DBP & HR JIC

	var00001	var00002	var00003	var00004	var00005	var00006	var00007	var00008	var00009
1	64.00	66.00	68.00	66.00	68.00	60.00	67.00	63.00	64.00
2	46.00	67.00	63.00	64.00	66.00	68.00	73.00	57.00	59.00
3	64.00	72.00	74.00	68.00	69.00	66.00	69.00	59.00	57.00
4	73.00	74.00	77.00	75.00	73.00	59.00	71.00	63.00	63.00
5	72.00	75.00	73.00	66.00	70.00	68.00	73.00	68.00	68.00
6	62.00	64.00	61.00	60.00	59.00	55.00	57.00	31.00	38.00
7	59.00	63.00	59.00	61.00	61.00	60.00	56.00	56.00	59.00
8	56.00	62.00	66.00	58.00	53.00	55.00	58.00	46.00	45.00
9	55.00	62.00	60.00	59.00	57.00	57.00	53.00	30.00	67.00
10	47.00	45.00	47.00	49.00	46.00	51.00	48.00	38.00	44.00

	var00010	var00011	var00012	var00013	var00014	var00015	var00016	var00017	var00018
1	66.00	65.00	70.00	64.00	86.00	81.00	77.00	74.00	74.00
2	62.00	67.00	69.00	74.00	95.00	89.00	84.00	85.00	79.00
3	53.00	60.00	61.00	70.00	81.00	90.00	86.00	83.00	81.00
4	66.00	64.00	66.00	64.00	81.00	77.00	72.00	70.00	71.00
5	67.00	71.00	72.00	69.00	101.00	88.00	75.00	80.00	77.00
6	44.00	47.00	44.00	48.00	92.00	83.00	78.00	73.00	72.00
7	58.00	63.00	60.00	60.00	82.00	84.00	82.00	82.00	79.00
8	44.00	45.00	48.00	54.00	75.00	86.00	82.00	68.00	67.00
9	34.00	40.00	41.00	43.00	81.00	79.00	77.00	74.00	72.00
10	49.00	46.00	47.00	51.00	94.00	87.00	85.00	84.00	84.00

	var00019	var00020	var00021	var00022	var00023	var00024	var00025	var00026
1	71.00	67.00	70.00	66.00	67.00	60.00	63.00	60.00
2	82.00	82.00	72.00	77.00	74.00	72.00	72.00	73.00
3	81.00	78.00	79.00	77.00	75.00	74.00	75.00	75.00
4	65.00	57.00	66.00	58.00	60.00	54.00	57.00	62.00
5	78.00	64.00	62.00	64.00	63.00	59.00	68.00	54.00
6	75.00	69.00	68.00	68.00	68.00	67.00	72.00	71.00
7	82.00	65.00	78.00	73.00	73.00	76.00	68.00	71.00
8	67.00	63.00	62.00	58.00	54.00	53.00	57.00	58.00
9	74.00	69.00	70.00	67.00	61.00	67.00	67.00	59.00
10	83.00	78.00	72.00	75.00	73.00	72.00	71.00	79.00

DBP & HR 40°C

	var00001	var00002	var00003	var00004	var00005	var00006	var00007	var00008	var00009
1	58.00	64.00	65.00	60.00	64.00	63.00	62.00	61.00	63.00
2	71.00	69.00	68.00	64.00	61.00	58.00	72.00	46.00	47.00
3	65.00	63.00	63.00	63.00	63.00	60.00	65.00	41.00	51.00
4	61.00	60.00	59.00	54.00	56.00	52.00	53.00	49.00	51.00
5	56.00	56.00	55.00	56.00	55.00	54.00	54.00	54.00	41.00
6	65.00	65.00	63.00	59.00	58.00	57.00	53.00	51.00	55.00
7	67.00	66.00	62.00	63.00	65.00	60.00	64.00	48.00	39.00
8	54.00	58.00	56.00	54.00	54.00	54.00	54.00	51.00	49.00
9	54.00	58.00	58.00	55.00	53.00	53.00	55.00	45.00	48.00
10	70.00	74.00	62.00	62.00	72.00	68.00	58.00	60.00	58.00

	var00010	var00011	var00012	var00013	var00014	var00015	var00016	var00017	var00018
1	57.00	62.00	63.00	65.00	90.00	88.00	84.00	83.00	88.00
2	49.00	49.00	46.00	49.00	98.00	91.00	89.00	87.00	89.00
3	48.00	41.00	43.00	46.00	82.00	75.00	75.00	80.00	70.00
4	55.00	56.00	51.00	56.00	100.00	87.00	83.00	78.00	77.00
5	43.00	45.00	48.00	51.00	89.00	89.00	88.00	92.00	91.00
6	57.00	54.00	53.00	57.00	79.00	82.00	86.00	75.00	69.00
7	47.00	49.00	61.00	67.00	91.00	86.00	80.00	87.00	82.00
8	51.00	48.00	51.00	42.00	80.00	79.00	74.00	77.00	74.00
9	50.00	50.00	40.00	55.00	92.00	87.00	87.00	86.00	82.00
10	56.00	58.00	68.00	64.00	95.00	93.00	83.00	83.00	78.00

	var00019	var00020	var00021	var00022	var00023	var00024	var00025	var00026
1	77.00	85.00	73.00	74.00	73.00	71.00	68.00	68.00
2	84.00	87.00	85.00	89.00	82.00	81.00	78.00	87.00
3	71.00	57.00	73.00	70.00	57.00	63.00	64.00	68.00
4	77.00	72.00	72.00	78.00	84.00	79.00	78.00	74.00
5	91.00	86.00	82.00	86.00	80.00	93.00	85.00	88.00
6	69.00	66.00	62.00	69.00	68.00	66.00	60.00	69.00
7	87.00	80.00	79.00	77.00	80.00	78.00	82.00	68.00
8	80.00	74.00	74.00	68.00	70.00	70.00	68.00	54.00
9	81.00	75.00	77.00	66.00	78.00	72.00	73.00	80.00
10	81.00	75.00	70.00	68.00	69.00	77.00	70.00	78.00

subjects skin temperature at 18C

time	head	chest	tricep	thigh	room
0	32.12	33	31.71	29.38	
5	30.97	33.12	31.86	32.39	
10	29.41	31.8	30.84	31.95	
15	28.6	30.67	29.95	31.62	
20	28.29	29.95	29.49	30.78	
25	28.67	29.49	29.26	31.02	
30	28.67	29.22	28.77	30.95	
35	29.05	29.2	28.38	30.66	
40	29.13	29.53	28.2	30.54	
45	29.3	29.71	28.07	30.39	
50	29.34	29.5	27.58	30.16	
55	29.54	29.63	27.49	30.4	
60	29.28	29.63	27.33	30.22	

subjects skin temperature at 27C

time	head	chest	tricep	thigh	room
0	32.66	33.78	33.55	33.63	
5	33.02	34.08	34.1	34.54	
10	32.36	33.63	33.83	33.87	
15	32.07	33.43	33.76	34.26	
20	31.66	33.11	33.72	34.03	
25	31.75	32.91	33.64	33.96	
30	31.89	33.27	33.55	33.5	
35	32.15	32.73	33.51	33.92	
40	32.27	32.83	33.47	33.8	
45	32.2	33.01	33.48	33.66	
50	32.63	33.32	33.47	33.76	
55	32.68	33.53	33.42	33.64	
60	32.7	33.47	33.38	33.67	

subjects skin temperature at 40 C

time	head	chest	tricep	thigh	room
0	32.62	34.73	34.73	35.22	
5	34.45	35.96	35.84	36.29	
10	34.64	35.96	35.93	36.44	
15	33.89	35.65	36.22	36.36	
20	34.49	35.86	36.42	36.27	
25	34.65	35.8	36.49	36.24	
30	34.63	35.75	36.53	36.2	
35	34.6	35.37	36.47	36.2	
40	34.67	35.37	36.74	36.02	
45	34.77	35.52	36.93	36.13	
50	34.84	35.57	37	36.11	**
55	34.95	36.64	37.09	36.23	
60	34.82	35.62	37.06	36.23	

POST RECOVERY 18C ALL SUBJECTS

▲SYSTOLIC BLOOD PRESSURE

TIME(min)	0	5	10	15	20	25	30
SUBJECT							
1	19	28	19	21	28	33	36
2	5	13	27	30	42	38	42
3	-3	-9	18	16	-28	-3	-15
4	-18	-18	-28	-28	-32	-29	-33
5	10	-5	30	38	38	38	34
6	0	-4	-7	-8	5	4	8
7	13	24	12	12	22	13	19
8	1	18	18	21	24	23	40
9	12	0	4	7	8	5	11
10	-34	-24	-25	-38	-36	-28	-41

TIME(MIN)	35	40	45	50	55	60
SUBJECT						
1	10	14	24	10	17	21
2	13	30	5	10	15	14
3	-13	-21	-2	-1	0	1
4	2	3	6	6	6	0
5	-17	-55	-30	50	60	35
6	7	18	18	18	12	22
7	-25	-9	-12	0	-8	-11
8	-15	-4	-10	-8	-6	-9
9	-6	-2	-3	9	4	12
10	-32	-27	-20	-37	-20	-23

POST RECOVERY 27C ALL SUBJECTS ΔSYSTOLIC BP

TIME(MIN)	0	5	10	15	20	25	30
SUBJECTS							
1	3	0	-5	-8	-6	-13	0
2	-15	2	0	-1	3	5	0
3	-14	-20	-19	-19	-19	-22	-24
4	-18	-4	-5	-17	-8	-9	1
5	-9	-1	-5	0	-4	-12	-11
6	4	1	-7	-4	-2	-6	-8
7	-21	-21	-13	-23	-37	-33	-34
8	-3	8	7	1	6	-1	7
9	-13	-8	-12	-12	-14	-13	-16
10	-1	-9	-11	-8	-12	-3	-12

TIME(MIN)	35	40	45	50	55	60
SUBJECTS						
1	-10	-13	4	-10	1	-12
2	-9	-9	-10	0	2	9
3	-31	-30	-27	-25	-24	-27
4	-19	-12	-11	-7	-3	-9
5	-19	-14	-12	-14	-10	-15
6	-8	-3	-3	1	0	1
7	-37	-33	-36	-37	-35	-26
8	-2	-9	-11	4	2	17
9	-33	-24	-26	-19	-24	-19
10	-16	-14	-11	-18	-16	-11

ΔSYSTOLIC BP 40°C

TIME(MIN)	0	5	10	15	20	25	30
SUBJECTS							
1	-11	-11	-14	-23	-25	-22	-24
2	8	4	1	-6	-11	-14	-3
3	-1	1	-10	1	1	2	5
5	-5	-10	-5	-11	-8	-10	-12
6	-8	-8	-9	-4	-6	-7	-8
7	-10	-8	-13	-21	-17	-16	-20
8	5	-3	-11	-7	-2	-13	-12
9	-19	-14	-16	-18	-15	-18	-15
10	-6	-4	-6	-12	-15	-25	-18

TIME(MIN)	35	40	45	50	55	60
SUBJECTS						
1	-8	-11	-16	-9	-9	-12
2	-35	-31	-29	-27	-29	-27
3	-20	-13	-19	-11	-10	-9
5	-18	-14	-10	-8	-16	-6
6	-10	-25	-19	-20	-18	-16
7	-28	-28	-21	-31	-28	-20
8	-27	-34	-29	-31	-16	-3
9	-18	-19	-17	-23	-17	-14
10	-18	-22	-21	-25	-34	-17

	t1	t2	t3	t4	t5	t6	t7	t8	t9
1	17.00	27.00	28.00	20.00	28.00	24.00	27.00	12.00	21.00
2	-12.00	-14.00	-8.00	-7.00	1.00	-5.00	6.00	-7.00	-6.00
3	-26.00	-3.00	-3.00	-11.00	-54.00	-20.00	-37.00	-45.00	-48.00
4	3.00	5.00	.00	-2.00	.00	1.00	3.00	20.00	26.00
5	1.00	3.00	14.00	8.00	10.00	5.00	4.00	-44.00	-41.00
6	-1.00	.00	-1.00	-4.00	.00	-2.00	-1.00	-9.00	10.00
7	7.00	18.00	12.00	12.00	17.00	16.00	18.00	8.00	19.00
8	-11.00	-5.00	-5.00	-5.00	-4.00	-9.00	.00	-27.00	-12.00
9	11.00	10.00	8.00	12.00	7.00	6.00	.00	1.00	4.00
10	3.00	1.00	-3.00	-3.00	-4.00	-8.00	-10.00	-14.00	-8.00

	t10	t11	t12	t13	t14	t15	t16	t17	t18
1	20.00	20.00	24.00	29.00	4.00	6.00	8.00	6.00	8.00
2	-12.00	-5.00	-4.00	.00	-18.00	3.00	-1.00	.00	2.00
3	-37.00	-33.00	-29.00	-28.00	.00	.00	1.00	.00	-3.00
4	28.00	33.00	33.00	31.00	-4.00	-1.00	-6.00	-10.00	-6.00
5	-45.00	-25.00	-28.00	-25.00	-32.00	-34.00	-31.00	-30.00	-41.00
6	10.00	10.00	8.00	23.00	-3.00	1.00	-3.00	-1.00	-1.00
7	15.00	22.00	17.00	25.00	-4.00	2.00	6.00	-2.00	-7.00
8	-18.00	-14.00	-10.00	-11.00	6.00	14.00	16.00	10.00	11.00
9	7.00	7.00	7.00	12.00	1.00	8.00	6.00	5.00	3.00
10	-11.00	-11.00	-6.00	-6.00	-15.00	-17.00	-15.00	-13.00	-16.00

	t19	t20	t21	t22	t23	t24	t25	t26	t27
1	.00	7.00	3.00	4.00	6.00	5.00	10.00	4.00	-4.00
2	4.00	9.00	-7.00	-5.00	-2.00	3.00	5.00	10.00	11.00
3	-5.00	-5.00	-13.00	-13.00	-10.00	-11.00	-10.00	-10.00	1.00
4	-8.00	-3.00	-8.00	-8.00	-9.00	-5.00	-4.00	-7.00	-20.00
5	-45.00	-43.00	-59.00	-52.00	-46.00	-43.00	-46.00	-42.00	-29.00
6	-2.00	-6.00	-6.00	-3.00	-4.00	-1.00	-2.00	-2.00	-6.00
7	-5.00	-2.00	-14.00	-15.00	-16.00	-15.00	-12.00	-6.00	5.00
8	8.00	11.00	1.00	-1.00	-5.00	2.00	3.00	12.00	7.00
9	3.00	-1.00	-24.00	13.00	-20.00	-14.00	-13.00	-11.00	4.00
10	11.00	14.00	-24.00	-18.00	-13.00	-16.00	-15.00	-11.00	-8.00

ADBP cont
b:\dbpchg.sav

	t28	t29	t30	t31	t32	t33	t34	t35	t36
1	2.00	3.00	-2.00	2.00	1.00	.00	-1.00	1.00	-5.00
2	9.00	8.00	4.00	1.00	-2.00	12.00	-14.00	-13.00	-11.00
3	-3.00	-3.00	-3.00	-3.00	-6.00	-1.00	-25.00	-15.00	-18.00
4	-15.00	-15.00	-21.00	-23.00	-22.00	-25.00	-32.00	-26.00	-20.00
5	-30.00	-31.00	-36.00	-34.00	-38.00	-37.00	-41.00	-39.00	-35.00
6	-6.00	-7.00	-6.00	-7.00	-8.00	-8.00	-8.00	-21.00	-19.00
7	5.00	3.00	-1.00	-2.00	-3.00	-7.00	-9.00	-5.00	-3.00
8	6.00	2.00	3.00	5.00	.00	4.00	-12.00	-21.00	-13.00
9	8.00	6.00	4.00	4.00	4.00	4.00	1.00	-1.00	1.00
10	-4.00	-4.00	-7.00	-9.00	-9.00	-7.00	-17.00	-14.00	-12.00
11	.	.	.	.	.	.			

	t37	t38	t39	var00026
1	.00	1.00	3.00	.
2	-11.00	-14.00	-11.00	.
3	-25.00	-23.00	-20.00	.
4	-23.00	-30.00	-26.00	.
5	-34.00	-39.00	-34.00	.
6	-17.00	-14.00	-11.00	.
7	-6.00	-7.00	-3.00	.
8	-11.00	1.00	7.00	.
9	-2.00	1.00	-8.00	.
10	-12.0	-22.00	-7.00	.
11	.	.	.	.
12	.	.	.	.
13	.	.	.	.
14	.	.	.	.
15	.	.	.	.
16	.	.	.	.
17	.	.	.	.
18				
19	.	.	.	.
20				

AHR

b:\hrchg.sav

	groupe	t1	t2	t3	t4	t5	t6	t7	t8
1	1.00	17.00	37.00	32.00	30.00	27.00	23.00	17.00	17.00
2	1.00	34.00	20.00	9.00	4.00	1.00	-4.00	-7.00	-3.00
3	1.00	32.00	27.00	20.00	28.00	12.00	24.00	13.00	9.00
4	1.00	25.00	15.00	7.00	6.00	.00	1.00	-18.00	-5.00
5	1.00	18.00	14.00	10.00	6.00	2.00	2.00	-2.00	-6.00
6	1.00	19.00	14.00	11.00	-3.00	7.00	2.00	6.00	.00
7	1.00	19.00	17.00	12.00	3.00	5.00	-1.00	4.00	-12.00
8	1.00	32.00	25.00	22.00	17.00	19.00	16.00	11.00	14.00
9	1.00	28.00	22.00	18.00	19.00	11.00	12.00	4.00	2.00
10	1.00	29.00	27.00	24.00	24.00	19.00	21.00	17.00	6.00
11	2.00	38.00	33.00	29.00	26.00	26.00	23.00	19.00	22.00
12	2.00	23.00	17.00	12.00	13.00	7.00	10.00	10.00	.00
13	2.00	33.00	29.00	24.00	22.00	23.00	17.00	9.00	18.00
14	2.00	37.00	24.00	11.00	16.00	13.00	14.00	.00	-2.00
15	2.00	28.00	19.00	14.00	9.00	8.00	11.00	5.00	4.00
16	2.00	26.00	28.00	26.00	26.00	23.00	26.00	9.00	22.00
17	2.00	23.00	34.00	30.00	16.00	15.00	15.00	11.00	10.00
18	2.00	9.00	18.00	14.00	11.00	9.00	9.00	6.00	7.00
19	2.00	21.00	19.00	17.00	14.00	12.00	14.00	9.00	10.00
20	2.00	34.00	17.00	15.00	14.00	14.00	17.00	18.00	12.00
21	3.00	42.00	40.00	36.00	35.00	40.00	29.00	37.00	25.00
22	3.00	46.00	39.00	37.00	35.00	37.00	32.00	35.00	33.00
23	3.00	42.00	35.00	35.00	40.00	30.00	31.00	17.00	33.00
24	3.00	42.00	30.00	20.00	25.00	22.00	21.00	15.00	22.00
25	3.00	36.00	23.00	19.00	14.00	13.00	13.00	8.00	8.00
26	3.00	25.00	25.00	24.00	28.00	27.00	27.00	22.00	18.00
27	3.00	23.00	26.00	30.00	19.00	13.00	13.00	10.00	6.00
28	3.00	35.00	30.00	24.00	31.00	26.00	31.00	24.00	23.00
29	3.00	20.00	19.00	14.00	17.00	14.00	20.00	14.00	14.00
30	3.00	36.00	31.00	31.00	30.00	26.00	25.00	19.00	21.00

	t9	t10	t11	t12	t13
1	12.00	12.00	11.00	6.00	7.00
2	-7.00	-7.00	-7.00	-8.00	-9.00
3	6.00	3.00	7.00	-3.00	4.00
4	-17.00	-10.00	-4.00	-11.00	-6.00
5	-6.00	-7.00	-10.00	-4.00	-1.00
6	6.00	4.00	.00	-33.00	-14.00
7	-5.00	-6.00	-7.00	-6.00	-11.00
8	11.00	13.00	8.00	7.00	6.00
9	2.00	3.00	3.00	-1.00	-2.00
10	7.00	4.00	2.00	3.00	8.00
11	18.00	19.00	12.00	15.00	12.00
12	5.00	2.00	.00	.00	1.00
13	10.00	12.00	6.00	9.00	14.00
14	.00	-1.00	-5.00	4.00	-10.00
15	4.00	4.00	3.00	8.00	7.00
16	17.00	17.00	20.00	12.00	15.00
17	6.00	2.00	1.00	5.00	6.00
18	5.00	4.00	2.00	3.00	3.00
19	7.00	1.00	7.00	7.00	-1.00
20	15.00	13.00	12.00	11.00	19.00
21	26.00	25.00	23.00	20.00	20.00
22	37.00	30.00	29.00	26.00	35.00
23	30.00	17.00	23.00	24.00	28.00
24	25.00	18.00	18.00	24.00	24.00
25	14.00	20.00	15.00	14.00	10.00
26	22.00	16.00	29.00	21.00	24.00
27	13.00	12.00	10.00	4.00	13.00
28	21.00	24.00	22.00	26.00	12.00
29	8.00	10.00	10.00	8.00	-6.00
30	10.00	22.00	16.00	17.00	24.00

	groupe	t1	t2	t3	t4	t5	t6	t7	t8
1	1.00	34.70	32.86	30.52	29.55	26.67	29.24	29.27	28.75
2	1.00	30.11	30.58	30.61	29.93	29.48	29.41	28.49	28.82
3	1.00	29.03	28.91	29.02	26.98	27.15	26.57	25.99	26.13
4	1.00	31.85	31.11	30.33	29.21	29.84	28.89	29.20	28.52
5	1.00	33.55	30.61	29.14	28.74	28.46	27.39	27.69	26.09
6	1.00	32.76	30.36	28.78	28.07	27.52	27.04	26.67	27.02
7	1.00	35.40	34.43	33.34	32.48	31.82	31.06	29.51	28.83
8	1.00	33.44	33.11	32.90	31.97	31.07	30.81	30.46	30.64
9	1.00	33.83	32.28	31.90	32.13	32.12	31.66	31.44	30.30
10	1.00	31.44	30.81	30.42	30.04	30.08	29.83	30.61	29.00
11	2.00	35.62	35.29	34.22	34.04	33.81	33.54	33.51	33.34
12	2.00	32.26	33.28	33.66	33.71	33.28	33.43	33.50	33.38
13	2.00	32.49	34.17	34.01	33.77	33.72	33.76	33.63	33.25
14	2.00	34.04	34.81	34.73	34.47	34.42	33.91	34.40	34.69
15	2.00	34.72	34.71	34.28	34.12	34.06	34.29	33.76	33.90
16	2.00	32.38	32.75	33.43	33.76	33.92	33.71	33.66	33.67
17	2.00	33.41	33.53	32.22	32.44	32.18	32.01	31.64	31.61
18	2.00	34.22	35.04	35.04	34.64	34.82	34.58	34.67	34.47
19	2.00	34.52	34.73	34.36	34.21	33.78	34.02	33.47	33.79
20	2.00	31.89	32.71	32.72	32.98	33.21	33.21	33.30	33.09
21	3.00	35.81	36.42	36.35	36.36	36.50	36.62	36.79	36.83
22	3.00	35.01	36.70	36.61	36.62	36.73	36.56	36.65	36.55
23	3.00	34.27	33.86	34.88	35.62	35.93	36.17	36.17	34.74
24	3.00	34.99	35.02	35.65	35.99	36.02	36.23	36.10	36.12
25	3.00	35.09	36.69	36.61	36.62	36.77	36.56	36.66	36.55
26	3.00	34.49	36.28	36.19	36.51	37.21	36.99	37.29	37.19
27	3.00	34.93	36.09	36.20	35.95	35.78	35.85	36.33	36.83
28	3.00	35.52	36.57	36.68	36.89	36.97	37.07	37.08	37.33
29	3.00	34.70	35.68	35.74	35.78	35.88	36.23	36.03	36.04
30	3.00	32.82	34.30	35.12	35.60	36.06	36.43	35.79	36.42
31	.	.	.	.	.	.	.	.	.

	t9	t10	t11	t12	t13
1	28.70	28.63	28.54	28.51	28.25
2	28.53	27.90	27.08	27.25	27.28
3	25.79	25.83	25.64	25.54	25.37
4	28.12	27.84	27.47	27.20	26.76
5	25.32	25.41	25.01	24.93	24.60
6	27.19	27.24	27.24	27.17	27.03
7	28.44	28.24	28.61	28.82	28.91
8	30.68	30.68	30.50	30.35	30.23
9	30.68	30.57	30.30	30.68	30.57
10	28.77	28.56	28.34	27.93	27.64
11	33.27	33.26	33.31	33.19	33.21
12	33.30	33.34	33.27	33.06	32.85
13	32.49	31.95	32.33	32.36	32.13
14	34.81	34.88	34.94	34.94	34.93
15	33.85	33.85	33.79	33.79	33.81
16	34.02	34.18	34.29	34.33	34.51
17	31.47	31.64	31.49	31.26	31.24
18	34.58	34.66	34.55	34.53	34.45
19	33.94	34.03	33.72	33.77	33.50
20	33.11	33.05	33.01	32.97	33.19
21	37.05	37.23	37.30	37.36	37.22
22	36.51	36.53	36.49	36.56	36.55
23	35.81	36.61	36.74	36.83	36.83
24	36.23	36.59	36.55	36.60	36.65
25	36.50	36.53	36.48	36.53	36.55
26	37.23	37.23	37.29	37.40	37.24
27	36.99	37.18	37.40	37.55	37.45
28	37.36	37.46	37.49	37.62	37.69
29	36.35	36.51	36.65	36.68	36.70
30	36.89	37.09	37.21	37.28	37.39
31					

	groupe	t1	t2	t3	t4	t5	t6	t7	t8
1	1.00	33.63	33.09	30.59	29.21	28.06	28.88	29.28	29.40
2	1.00	32.55	32.11	31.18	30.80	30.16	30.24	30.07	30.34
3	1.00	31.95	30.38	29.17	28.73	28.36	28.06	27.95	28.42
4	1.00	32.96	29.48	28.19	26.73	26.52	26.13	26.69	27.83
5	1.00	31.85	29.29	25.90	24.54	25.58	28.54	26.79	27.04
6	1.00	32.28	30.50	29.21	28.70	28.33	28.60	30.02	30.72
7	1.00	33.54	32.26	31.04	30.44	29.45	29.90	29.66	29.62
8	1.00	31.21	31.35	30.42	29.17	28.38	27.85	28.00	28.61
9	1.00	31.12	30.05	29.54	29.56	29.54	29.57	29.55	29.49
10	1.00	30.18	30.54	28.87	28.13	28.53	28.79	28.76	29.07
11	2.00	30.85	31.49	30.31	29.73	29.07	29.25	29.95	30.71
12	2.00	33.52	33.79	33.27	33.47	33.20	35.08	35.17	35.22
13	2.00	36.52	36.56	35.01	33.73	33.04	32.86	33.10	32.29
14	2.00	32.29	33.65	33.05	32.68	32.14	32.13	32.09	32.37
15	2.00	33.00	92.92	31.90	31.71	31.09	31.27	31.15	31.58
16	2.00	30.95	32.27	31.59	31.68	31.10	30.87	30.66	30.69
17	2.00	32.62	33.18	32.62	32.15	31.59	31.22	31.14	31.37
18	2.00	31.26	32.34	32.02	31.73	31.63	31.53	31.51	31.43
19	2.00	31.99	32.43	32.71	32.84	32.77	32.88	32.74	32.74
20	2.00	30.68	31.57	31.14	31.04	31.02	30.45	31.25	33.32
21	3.00	33.08	33.41	33.05	33.25	32.89	33.66	34.92	35.56
22	3.00	35.37	35.40	35.12	34.98	35.20	35.04	35.48	35.45
23	3.00	32.79	33.86	35.37	35.53	35.25	35.10	34.95	34.87
24	3.00	34.98	35.23	35.40	35.35	35.20	34.99	34.95	34.84
25	3.00	32.95	35.63	35.63	35.35	35.56	35.47	35.48	35.04
26	3.00	32.60	35.50	35.67	36.01	35.49	35.52	35.05	35.18
27	3.00	31.89	33.18	33.29	33.22	32.82	32.99	32.76	32.51
28	3.00	31.83	32.88	32.54	32.22	32.35	32.58	32.53	32.12
29	3.00	33.12	35.43	35.54	35.72	35.74	35.89	35.81	35.86
30	3.00	32.43	34.58	34.70	34.07	34.89	35.23	35.16	35.30

	t9	t10	t11	t12	t13
1	29.32	29.59	29.69	30.07	29.98
2	30.07	30.04	29.88	29.97	29.58
3	28.55	29.07	29.26	29.28	29.10
4	28.14	28.40	28.38	28.49	28.33
5	27.40	27.98	28.14	28.29	28.02
6	30.44	30.43	30.46	30.53	30.33
7	29.84	29.72	29.81	29.99	29.89
8	29.10	29.28	29.33	29.31	29.18
9	29.36	29.22	29.15	29.12	29.10
10	29.07	29.30	29.23	28.95	29.15
11	30.97	31.78	32.26	32.38	32.49
12	34.91	35.14	35.00	34.76	34.28
13	32.02	31.47	33.72	34.27	34.30
14	32.42	32.51	32.56	32.61	32.61
15	31.47	31.40	31.24	31.50	31.48
16	30.83	31.56	31.76	32.30	32.77
17	31.64	32.11	32.22	32.25	32.31
18	31.52	31.34	31.20	31.22	31.14
19	32.51	32.80	32.56	32.79	32.46
20	33.60	33.62	33.79	33.66	33.29
21	35.83	36.04	36.16	36.20	36.11
22	36.50	36.64	35.63	35.63	32.95
23	34.84	34.98	34.75	34.78	34.69
24	34.85	34.80	34.70	34.56	34.59
25	35.21	34.98	35.12	35.40	35.36
26	35.24	35.53	35.73	35.96	35.98
27	32.59	32.56	33.26	32.89	33.13
28	31.61	31.92	31.75	31.56	31.15
29	35.95	36.05	36.10	36.16	36.20
30	35.62	35.96	35.54	36.24	35.47

	groupe	t1	t2	t3	t4	t5	t6	t7	t8
1	1.00	34.70	32.86	30.52	29.55	26.67	29.24	29.27	28.75
2	1.00	30.11	30.58	30.61	29.93	29.48	29.41	28.49	28.82
3	1.00	29.03	28.91	29.02	26.98	27.15	26.57	25.99	26.13
4	1.00	31.85	31.11	30.33	29.21	29.84	28.89	29.20	28.52
5	1.00	33.55	30.61	29.14	28.74	28.46	27.39	27.69	26.09
6	1.00	32.76	30.36	28.78	28.07	27.52	27.04	26.67	27.02
7	1.00	35.40	34.43	33.34	32.48	31.82	31.06	29.51	28.83
8	1.00	33.44	33.11	32.90	31.97	31.07	30.81	30.46	30.64
9	1.00	33.83	32.28	31.90	32.13	32.12	31.66	31.44	30.30
10	1.00	31.44	30.81	30.42	30.04	30.08	29.83	30.61	29.00
11	2.00	35.62	35.29	34.22	34.04	33.81	33.54	33.51	33.34
12	2.00	32.26	33.28	33.66	33.71	33.28	33.43	33.50	33.38
13	2.00	32.49	34.17	34.01	33.77	33.72	33.76	33.[illegible]	33.25
14	2.00	34.04	34.81	34.73	34.47	34.42	33.91	34.40	34.69
15	2.00	34.72	34.71	34.28	34.12	34.06	34.29	33.75	33.90
16	2.00	32.38	32.75	33.43	33.76	33.92	33.71	33.66	33.67
17	2.00	33.41	33.53	32.22	32.44	32.18	32.01	31.64	31.61
18	2.00	34.22	35.04	35.04	34.64	34.82	34.58	34.67	34.47
19	2.00	34.52	34.73	34.36	34.21	33.78	34.02	33.47	33.79
20	2.00	31.89	32.71	32.72	32.98	33.21	33.21	33.30	33.09
21	3.00	35.81	36.42	36.35	36.36	36.50	36.62	36.79	36.83
22	3.00	35.01	36.70	36.61	36.62	36.73	36.56	36.65	36.55
23	3.00	34.27	33.86	34.88	35.62	35.93	36.17	36.17	34.74
24	3.00	34.99	35.02	35.65	35.99	36.02	36.23	36.10	36.12
25	3.00	35.09	36.69	36.61	36.62	36.77	36.56	36.66	36.55
26	3.00	34.49	36.28	36.19	36.51	37.21	36.99	37.29	37.19
27	3.00	34.93	36.09	35.20	35.95	35.78	35.85	36.33	36.83
28	3.00	35.52	36.57	36.68	36.89	36.97	37.07	37.08	37.33
29	3.00	34.70	35.68	35.74	35.78	35.88	36.23	36.03	36.04
30	3.00	32.82	34.30	35.12	35.60	36.06	36.43	35.79	36.42
31	.	.	.	.	.	.	.	.	.

	t9	t10	t11	t12	t13
1	28.70	28.63	28.54	28.51	28.25
2	28.53	27.90	27.08	27.25	27.28
3	25.79	25.83	25.64	25.54	25.37
4	28.12	27.84	27.47	27.20	26.76
5	25.32	25.41	25.01	24.93	24.60
6	27.19	27.24	27.24	27.17	27.03
7	28.44	28.24	28.61	28.82	28.91
8	30.68	30.68	30.50	30.35	30.23
9	30.68	30.57	30.30	30.68	30.57
10	28.77	28.56	28.34	27.93	27.64
11	33.27	33.26	33.31	33.19	33.21
12	33.30	33.34	33.27	33.06	32.85
13	32.49	31.95	32.33	32.36	32.13
14	34.81	34.88	34.94	34.94	34.93
15	33.85	33.85	33.79	33.79	33.81
16	34.02	34.18	34.29	34.33	34.51
17	31.47	31.64	31.49	31.26	31.24
18	34.58	34.66	34.55	34.53	34.45
19	33.94	34.03	33.72	33.77	33.50
20	33.11	33.05	33.01	32.97	33.19
21	37.05	37.23	37.30	37.36	37.22
22	36.51	36.53	36.49	36.56	36.55
23	35.81	36.61	36.74	36.83	36.83
24	36.23	36.59	36.55	36.60	36.65
25	36.50	36.53	36.48	36.53	36.55
26	37.23	37.23	37.29	37.40	37.24
27	36.99	37.18	37.40	37.55	37.45
28	37.36	37.46	37.49	37.62	37.69
29	36.35	36.51	36.65	36.68	36.70
30	36.89	37.09	37.21	37.28	37.39
31	.	.	.	.	.

	groupe	t1	t2	t3	t4	t5	t6	t7	t8
1	1.00	32.85	33.54	32.87	32.61	32.20	31.82	31.77	31.40
2	1.00	32.67	32.25	31.81	31.63	30.94	30.32	30.22	30.25
3	1.00	30.96	28.18	30.66	30.71	30.63	30.54	30.43	30.25
4	1.00	34.22	33.87	32.99	32.73	32.26	31.92	31.79	31.38
5	1.00	32.19	31.17	30.41	30.21	29.93	30.17	30.36	30.12
6	1.00	34.39	34.84	33.58	32.85	32.25	32.17	31.87	31.87
7	1.00	31.14	31.77	31.30	31.22	30.95	30.85	30.60	30.64
8	1.00	31.26	30.03	29.27	29.05	28.37	28.54	28.42	28.23
9	1.00	33.45	33.79	33.08	32.53	31.88	32.00	32.20	31.39
10	1.00	33.90	33.52	32.75	32.49	32.22	31.95	31.88	31.72
11	2.00	33.63	34.42	33.90	34.06	33.52	33.46	33.60	33.55
12	2.00	33.90	35.08	35.13	35.20	34.97	35.08	35.02	35.01
13	2.00	30.73	32.57	31.54	31.49	31.40	31.01	32.15	31.49
14	2.00	33.97	35.19	34.97	34.82	34.58	34.55	34.40	34.34
15	2.00	33.80	34.58	31.09	34.25	34.06	33.96	33.72	33.61
16	2.00	34.12	34.57	34.13	34.11	34.00	34.01	34.06	33.99
17	2.00	34.32	34.77	34.54	34.55	34.16	34.05	33.91	33.86
18	2.00	32.65	34.16	33.92	34.17	34.21	34.39	34.48	34.33
19	2.00	34.19	34.78	34.54	34.86	34.45	34.37	34.49	34.34
20	2.00	35.04	35.32	34.97	35.09	34.95	34.80	34.74	34.68
21	3.00	34.96	35.77	35.67	35.81	35.63	35.67	35.64	35.53
22	3.00	33.69	36.61	36.76	36.68	36.63	36.61	36.50	36.40
23	3.00	33.51	35.67	36.27	36.25	36.02	36.11	36.05	36.10
24	3.00	33.68	36.45	36.25	36.23	36.07	36.15	36.02	36.20
25	3.00	33.68	36.60	36.75	36.70	36.63	36.60	36.50	36.40
26	3.00	34.43	36.43	36.42	36.08	35.79	35.55	35.17	35.53
27	3.00	35.79	36.43	36.58	36.59	36.50	36.50	36.42	36.29
28	3.00	35.49	36.58	36.61	36.59	36.56	36.53	36.34	36.38
29	3.00	34.83	35.83	35.92	35.99	35.96	36.13	36.01	35.93
30	3.00	35.32	36.65	36.82	36.85	36.71	36.53	36.21	36.27

	t9	t10	t11	t12	t13
1	31.39	31.09	30.80	31.09	30.89
2	30.13	29.89	29.69	29.29	28.92
3	30.04	30.02	29.93	29.83	29.78
4	31.34	31.17	31.00	30.90	30.72
5	29.76	29.58	29.35	29.27	29.05
6	31.63	31.48	31.33	31.30	31.12
7	30.37	30.36	30.16	30.14	30.04
8	28.06	27.93	27.83	27.85	27.80
9	30.97	30.66	30.55	30.29	30.07
10	31.72	31.78	31.65	31.55	31.41
11	33.36	33.39	33.30	33.16	33.29
12	34.88	34.90	34.88	34.63	34.65
13	31.50	30.91	31.46	31.32	31.40
14	34.17	34.14	34.09	34.03	33.98
15	33.49	33.38	33.37	33.35	33.37
16	33.89	33.81	33.68	33.43	33.47
17	33.83	33.84	33.82	33.72	33.73
18	34.37	34.35	34.32	34.25	34.23
19	34.05	34.17	34.14	34.11	34.10
20	34.53	34.56	34.58	34.45	34.54
21	35.45	35.56	35.54	35.55	35.47
22	36.45	36.42	36.43	36.43	36.42
23	36.14	36.30	36.49	36.65	36.72
24	36.46	36.52	36.74	36.76	36.85
25	36.45	36.41	36.43	36.41	36.42
26	35.83	36.04	36.43	36.74	36.92
27	36.19	35.50	35.09	34.94	34.80
28	36.44	36.39	36.44	36.35	36.34
29	35.99	36.01	36.07	36.26	36.28
30	36.30	36.57	36.68	36.75	36.75

	groupe	t1	t2	t3	t4	t5	t6
1	1.00	.47	.42	.55	.46	.42	.39
2	1.00	.45	.27	.24	.14	.25	.22
3	1.00	.22	.21	.21	.19	.15	.17
4	1.00	.34	.30	.37	.41	.39	.45
5	1.00	.49	.34	.24	.26	.26	.30
6	1.00	.50	.35	.33	.33	.40	.36
7	1.00	.41	.30	.37	.28	.36	.30
8	1.00	.19	.20	.20	.23	.24	.37
9	1.00	.27	.20	.14	.10	.36	.24
10	1.00	.33	.36	.37	.38	.34	.35
11	2.00	.45	.45	.34	.50	.31	.42
12	2.00	.30	.19	.17	.15	.16	.19
13	2.00	.32	.32	.25	.22	.24	.24
14	2.00	.34	.31	.33	.30	.25	.24
15	2.00	.21	.24	.23	.24	.26	.25
16	2.00	.41	.35	.28	.38	.25	.30
17	2.00	.46	.28	.29	.33	.21	.20
18	2.00	.32	.28	.26	.21	.31	.21
19	2.00	.21	.23	.26	.28	.24	.28
20	2.00	.34	.31	.33	.23	.20	.20
21	3.00	.65	.44	.43	.34	.44	.23
22	3.00	.30	.27	.17	.19	.30	.19
23	3.00	.31	.37	.27	.20	.29	.37
24	3.00	.48	.40	.40	.40	.46	.41
25	3.00	.29	.20	.14	.22	.14	.13
26	3.00	.25	.21	.20	.25	.25	.27
27	3.00	.36	.30	.21	.23	.24	.35
28	3.00	.15	.14	.19	.19	.12	.37
29	3.00	.24	.30	.22	.19	.23	.27
30	3.00	.38	.25	.31	.20	.17	.31

b:\co2all.sav

	var00001	var00002	var00003	var00004	var00005	var00006	var00007
1	1.00	.49	.46	.58	.43	.43	.32
2	1.00	.63	.30	.23	.13	.23	.20
3	1.00	.15	.16	.16	.18	.12	.11
4	1.00	.28	.25	.33	.34	.35	.35
5	1.00	.45	.35	.23	.23	.22	.25
6	1.00	.20	.22	.20	.23	.22	.37
7	1.00	.38	.28	.34	.24	.30	.24
8	1.00	.20	.15	.20	.19	.11	.14
9	1.00	.24	.19	.15	.11	.34	.23
10	1.00	.37	.38	.38	.37	.30	.29
11	2.00	.67	.61	.41	.57	.39	.39
12	2.00	.34	.23	.19	.17	.19	.20
13	2.00	.27	.32	.24	.19	.20	.21
14	2.00	.32	.34	.31	.28	.24	.24
15	2.00	.17	.21	.20	.20	.22	.21
16	2.00	.42	.36	.26	.35	.25	.29
17	2.00	.48	.32	.35	.34	.22	.22
18	2.00	.27	.26	.23	.19	.27	.19
19	2.00	.22	.25	.22	.27	.24	.26
20	2.00	.36	.33	.35	.23	.19	.18
21	3.00	.88	.68	.52	.40	.64	.27
22	3.00	.37	.37	.19	.21	.36	.21
23	3.00	.25	.31	.24	.19	.26	.34
24	3.00	.54	.40	.40	.38	.42	.37
25	3.00	.24	.18	.13	.21	.13	.12
26	3.00	.28	.23	.21	.25	.26	.26
27	3.00	.36	.29	.20	.26	.23	.31
28	3.00	.63	.39	.38	.35	.45	.45
29	3.00	.19	.26	.21	.19	.23	.27
30	3.00	.45	.29	.36	.21	.19	.29